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Cardiovascular Complications of Large Hiatal Hernias: Expanding the Indications for Robotic Surgical Anatomic and Physiologic Repair: A Review

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Abstract

Background: Historically, the pathophysiology of Hiatal Hernias (HH) has not been fully understood. As a result, the surgical therapy of HH has focused primarily on gastrointestinal symptoms and Gastroesophageal Reflux (GERD). This treatment strategy has been associated with poor relief of symptoms and poor long-term outcomes. In fact, until recently, most patients with HH have been watched and referred for surgery as a last resort. Recent experience has shown that a large (giant) Hiatal Hernia (GHH) is a common problem known to impact adjacent organs such as the hearts and lungs. Those referred for surgical repair often complain of dyspnea, which is erroneously attributed to pulmonary compression or aspiration, but has been shown to be from tamponade caused from compression of the heart by herniated abdominal contents. This article reviews the present understanding of GHH, the cardiac complications which result from GHH, and the most advanced robotic minimally invasive surgical approach to the anatomic and physiologic repair of GHH. **Methods:** In a prospective cohort study, we evaluated patients undergoing RRHH with at least a 2-year follow-up. All patients undergoing elective (RRHH) were identified preoperatively and enrolled prospectively in this study. Preoperative characteristics, medical comorbidities, and clinical information were all recorded prospectively and recorded into a secure surgical outcomes database. All patients received the previously validated Gastroesophageal Reflux Disease-Health-Related Quality of Life (GERD-HRQL) questionnaire preoperatively and at postoperative time points of 1 month, 1 year, and 2 years. Patients routinely had a barium swallow postoperatively before discharge but did not undergo a bar-

ium swallow, an endoscopy, or a CT scan study at the 1-month time point unless indicated by symptoms. At 6 months, 1 year, and yearly intervals thereafter, all patients received an endoscopy study to ascertain the presence of a recurrence, regardless of symptoms. Recurrence was defined as over 2 cm or 10% of the stomach above the diaphragm detected by CT, esophagogram or endoscopy. In addition, an extensive search was conducted using Pub Med in order to extract references to the cardiovascular complications of HH. **Results:** 423 patients underwent RRHH. With a long-term follow-up, there was a significant decrease in the Median Symptom Severity Score from 42.0 preoperatively, to 3.0 postoperatively. Recurrence was seen in 5 patients (5/423) for a recurrence rate of 1.1%. **Conclusion:** This experience has been the basis of two important realizations: 1) all patients with GHH have at least some degrees of clinically relevant compression of the inferior vena cava and the left atrium which causes tamponade and cardiogenic dyspnea which completely resolves after successful surgical repair; and 2) primary care providers and gastroenterologists who usually treat patients for GHH repair rarely recognize cardiac compression and tamponade as the cause of the shortness of breath and gradual increase in dyspnea on exertion and progressive fatigability in these patients. This article reviews the present understanding of GHH, the cardiac complications which result from GHH and the most advanced robotic minimally invasive surgical approach to the anatomic and physiologic repair of GHH.

Keywords

Hiatal Hernia, GERD, Paraesophageal Hernia, Robotic Surgery, Laparoscopic Repair, Nissen, Belsey, Gastroesophageal Valvuloplasty

1. Introduction

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 the "blind" men in the Indian parable. In that story, a group of blind men who had never come across an elephant before were tasked with describing the elephant by touching it. These "blind" men examined the different parts of the elephant by virtue of their incomplete knowledge, and were unable to recognize the "whole elephant". Indeed, this has been the case with HHs. Patients with a HH are usually seen by primary care providers, gastroenterologists, pulmonologists, cardiologists, otolaryngologists, and surgeons who, as in the case of the "blind" men, have failed to recognize the overarching pathophysiology of HH's. Recently 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 on understanding the pathophysiology, the extra-gastrointestinal complications, and consequently the expanded surgical indications for the repair of Hiatal

Hernias. Two significant realizations have changed the understanding of Hiatal Hernias. First, Hiatal Hernias are now seen as a gastrointestinal pathologic process that is more than GERD (Gastroesophageal Reflux). Second, a Hiatal Hernia is identified as a common condition which by virtue of its anatomic location has significant cardiovascular complications. These factors have erased the borders between specialty "silos" and brought new insights into the treatment of this important condition.

It is estimated that Hiatal Hernias (HHs) affect approximately 20% of the population. The incidence of HHs is 37% in patients with Morbid Obesity, as defined by BMI > 43 kg/m^2. Historically, the term Giant Hiatal Hernia has been used to describe a HH where greater than 30% of the stomach is above the hiatus as seen on imaging chest radiographs. Presently, 37% of HHs are defined as "Large" or "Giant" and are usually diagnosed by imaging studies or are classified as such when the HH is greater than 4 cm on endoscopy [1].

2. Classification of HH

Classically, hiatal hernias have been classified in two ways:

1) Types I-IV

This classification is of historic interest and dates back to the early part of the twentieth century, the first use off chest radiographs, and is based on the position of the gastroesophageal junction relative to the diaphragm [2] [3].

Type I, A Sliding HH. This 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" Paraesophageal (PEH) HH, occurs when the stomach migrates into the chest and "rolls" over the esophagus with the gastroesophageal junction still laying down in 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 [2] [3].

This classification which dates back to 1927, does not have 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 stomach, retroperitoneal and intra-abdominal structures which have migrated into the chest. 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 [4].

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 splaying 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, wider splaying of the posterior portion of the crural limbs with migration of the retroperitoneal fatty structures above the diaphragm 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".

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 2 cm 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 2 cm of the esophagus has been pulled out, the GE Valve becomes incompetent, resulting in reflux (**Figure 1**). Therefore, GERD requires a hiatal defect of at least 2 cm. 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 [5].

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

Figure 1. Lateral view of the GEV. 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.

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 [6] [7].

2.1. Symptoms

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 [8] [9] [10]. 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 [11] [12].

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 [13]. 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 [14]. 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 (Figure 2). 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 patients with a HH 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 [15]. However, in these patients spirometry has not correlated with to the level of functional compromise [16] [17]. 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%) [18]. 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 usually 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.
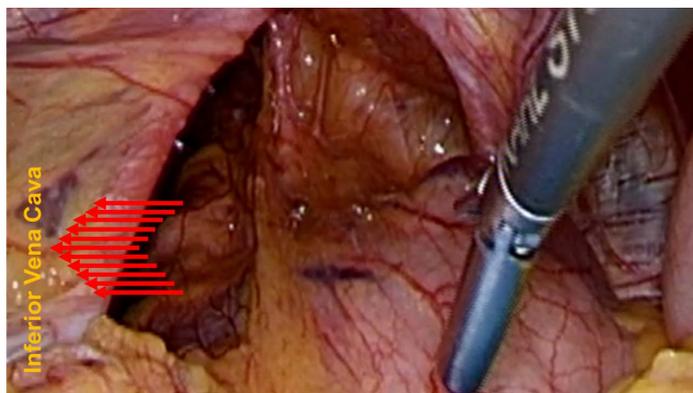
Figure 2. Intraoperative photograph of a large Hiatal Defect. Extension into the right chest results in compression (red arrows) of the Inferior Vena Cava.

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 [19]. Chau *et al.* demonstrated a large hiatal hernia as the cause of chest pain in patients that presented to emergency department with acute angina [18]. A hiatal hernia can cause pulmonary edema and cardiac failure through pulmonary venous obstruction [20] [21]. 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 [22]. 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 [23]. 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 [24] [25] [26] [27]. 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 [22]. 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 [28]. Previous animal studies have confirmed a relationship among CS compression and impaired myocardial blood flow, increased ventricular blood volume, decreased ventricular distensibility, and diastolic dysfunc-

tion [29] [30] [31]. These may represent further mechanisms for 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 [32]. 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 fatigibiliy. 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 the case of large HHs which extend posteriorly into the right chest, the Inferior Vena Cava (IVC) and the hepatic veins are compressed (**Figure 3**). 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 [33]. This results in poor right atrial filling and a "tamponade" physiology, and lower extremity edema. This is yet another cause of poor filling in diastole in patients with large HHs. Furthermore, with pressure on the hepatic veins the patients can present with ascites [34].

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
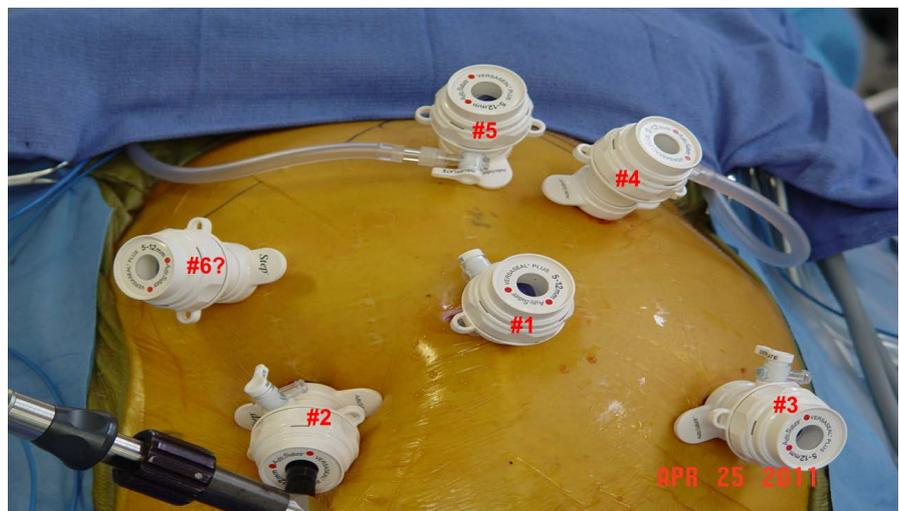
Figure 3. Port Placement.

men with HH and 19-fold higher in women with HH compared to the frequency of AF reported in the general population [35]. Atrial arrythmias 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.

Gastric Symptoms: The strangulation of the stomach in the esophageal hiatus results in discomfort and pain in the subxiphoid region. Gastric volvulus is a rare presentation of HHs.

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. Typically, anemia resolves in more than 90% of patients following the hernia repair [36] [37] [38] [39] [40].

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.

2.2. Diagnosis

Although most patients with HHs are symptomatic, in the majority of patients with non-gastrointestinal symptoms, a HH is not recognized as the cause. In these patients a HH is 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 [41].

Barium swallow is probably the best diagnostic study and gives information about the amount of the herniated stomach and the direction of herniation [42]. [43] A computed tomography scan of the chest and the abdomen because it may provide additional information on the type and location of the hernia.

Upper endoscopy is useful for visualization of the esophageal and gastric mucosa, detection of Barrett's esophagus, erosive esophagitis, and Cameron's ulcers. Further-

more, 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 - 4 cm 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 - 4 cm) 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 [44] [45]. In fact, studies 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 [46] [47].

2.3. 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 [48] [49].

The surgical treatment strategy in patients with Stage I symptoms with HHs in the range of 0 - 2 cm, 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 2 cm, 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 [50]. 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 [51] [52]. 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 repairs 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 [53] [54].

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 [55]. 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 have continued to advise 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 [56].

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 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 [57]. 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 [58]. 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 [59] [60].

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 - 3 cm, 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 [61] [62].

The advent of laparoscopy introduced an alternative to open procedures. However, laparoscopy has been hampered by the shortcomings of two-dimensional visualization and un-wristed 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 [63]. In fact, a randomized controlled trial from Watson *et al.* demonstrated similar outcomes between suture and mesh repair [64].

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 $30 \ kg/m^2$ were 4.2 times more likely to have a hiatal hernia than those with a BMI lower than $25 \ kg/m^2$ [65]. However, a 10-year retrospective review of laparoscopic repair of HHs identified obesity as a risk factor for long-term adverse outcomes [66]. In other studies, obesity has also been shown to increase the failure rate of antireflux surgery [67] [68]. Because of the increased risk of surgical failure in this challenging population, a sleeve gastrectomy or gastric bypass has been recommended [65]. 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 > $40 \ kg/m^2$, alone, or 35 - $40 \ kg/m^2$, 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 facili-

tated 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 Gastroesophgaeal 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".

2.4. Surgical Technique

Anesthesia Management:

In patients with large HHs, many times the pleural space is entered during the robotic dissection. This is especially true in elderly female patients. In order to perform a complete dissection of the hernia sac, and return all the peritoneal contents into the abdomen, it is imperative to have full exposure of the entire mediastinum. Entry into the pleural space results in loss of pneumo-peritoneum, a ten-

sion pneumothorax, downward pressure on the diaphragm, and loss of exposure at the hiatus. Consequently, in order to have full control of the exposure and to complete a perfect robotic dissection, it I important to have a mitigation plan in place. We prefer to use a double lumen endotracheal tube in patients with large hiatal defects. In case of pleural entry, the lumen of the tube to the ipsilateral lung is clamped, thereby isolating the ipsilateral lung. This maneuver creates a large space in the chest, thereby "buying" more time before the CO_2 pressure can result in "tension" and tamponade physiology. The pleural entry is closed with robotically applied clips and a member of the surgical team places a small chest tube through the 9th interspace anteriorly. After placement of the chest tube thoracestomy and evacuation of the CO_2, the ipsilateral lung is re-inflated. This strategy allows the surgeon to continue with the dissection with perfect exposure and without interruption. In cases where the pleural space must be entered and closure of the pleura is not possible, the tube thoracostomy evacuates the CO_2 and facilitates an excellent exposure of the surgical field. We use two laparoscopic insufflators in order to maintain the pneumoperitoneum at a pressure of 15 mmHg.

Port Placement:

The patient is placed in the lithotomy position. The surgeon stands between the legs. Two Laparoscopic CO_2 insufflators are used. We prefer to accurately place laparoscopic ports and introduce the robotic arms through these ports. This strategy diversifies the options for the surgeon in the event of adhesions, unexpected complications, and if the surgeon elects to use conventional laparoscopy for the repair and reconstruction phase of the procedure. We prefer to use the Visiport Instrument (Medtronic, Norwalk Conn., USA) for initial port entry into the peritoneum (Figure 3). Port #1 (Camera Port) is placed inferior to the umbilicus. A small curvilinear incision is made under the umbilicus. A Kocker clamp is used to grasp the frenulum of the umbilicus and to elevate the anterior abdominal wall. Upward traction on the clamp provides the countertraction which is necessary for safe peritoneal entry under direction videoendoscopic guidance using the visiport instrument. Alternatively, a Veress Needle is introduced inferior to the umbilical frenulum and upon entry into the peritoneum a characteristic popping sensation is felt. Saline is introduced through the needle, and an unobstructed free peritoneal position of the needle is verified by the "hanging drop method" where the saline flows freely into the peritoneal cavity with elevation of the abdominal wall. A 10 - 12 Versiport trocar (Covidien/Medtronic Inc., Norwalk, Conn.) is introduced using the Veress Needle. A 0 degree Endoeye videoendoscope (Olympus Inc.) is used. Pneumoperitoneum is created using CO_2 gas to a maximum pressure of 15 mmHg. The table is placed in a steep Reverse Trendelenberg position. Under direct videoendoscopic guidance 5 to 6 other ports are placed. We prefer to use the 10 - 12 Versiport trocar (Medtronic Inc., Norwalk, Conn.) for all ports. These ports do not require reducer caps. An additional design advantage of these ports is that the port sites do not have to be closed. The peritoneal entry site is only 4 mm and is virtually pain free. The use of the Versiports allows for the placement of extra ports as needed, especially in pa-

tients with a high BMI or very large hiatal defects which may extend far above the diaphragm. Furthermore, the capless design of these ports enables rapid instrument change without loss of pneumoperitoneum. Port #2 is placed in the right paraumbilical region at the mammary line. An Endo-Paddle Retract retractor (Medtronic Inc., Norwalk, Conn.) is placed through Port #2 and fixed to the table using a self-retaining system (Mediflex, Velmed Inc., Wexford, PA). The advantage of the Endopladdle retract device is that it is used to exert constant fixed upward traction onto the apex of the esophageal hiatus, and thereby, facilitates visualization and instrument maneuverability within the hiatal opening. Port #3 is placed halfway between the costal arch and the umbilicus as laterally on the right side of the abdomen as possible. This port will carry the left robotic arm. Using the videoendoscope the left and right limbs of the right crus are identified. Port #4 is placed in the subcostal region halfway between the umbilicus and the xiphoid just to the left of the midline. This port is aligned with the right limb of the right crus of the diaphragm. Port #5 is placed in the subcostal region two finger-breaths to the left and caudad to Port #4. Port #5 is aligned with the left limb of the right crus of the diaphragm. The Laparoscopic insufflator is disconnected from Port #1 and attached to port #4. A second insufflator is attached to Port #5. The use of two high flow insufflators facilitates rapid extra corporeal knot placement while preserving pneumoperitoneum and exposure of the esophageal hiatus. Port #6 is placed halfway between the costal arch and the umbilicus as laterally on the left side of the abdomen as possible. This port will carry the right robotic arm. At times a 7th port is needed to retract the contents of the hiatal defect. In such an instance Port #7 is placed in the mammary line halfway between pots #1 and #6.

Positioning and Introduction of the Robot:

The surgical robot (daVinci, Intuitive Surgical, Sunnyvale, Ca.) is docked using "side docking" technique (Figure 4). A 30 degree down-viewing robotic binocular camera is used, and it is introduced through Port #1. The right Robotic

Figure 4. Side docking of the robot.

arm with a hook cautery instrument is introduced through Port #3. The left Robotic arm with a Debakey grasper instrument is introduced through Port #2. The entire dissection uses electrocautery and meticulous hemostasis. It is important not to use vessel sealing or other dissecting devices. The use of the hook cautery allows the surgeon to dissect along anatomic planes. Two assistants are used. A paddle retractor (Endo-paddle Retract, (Medtronic, Norwalk, Conn USA) is introduced by the Assistant #1 through Port #6. This is used to retract the tissues in a caudal direction at different points in the dissection. Assistant #2 introduces two Endo-Kittner instruments through Ports # 4 and #5. The Endo-Kittner instruments are used to place lateral and upward traction on the limbs of the esophageal crus. This maneuver opens the space inside the hiatus further and allows the surgeon to have optimal exposure.

The Operation is divided into 7 Steps:

Step 1. Dissection of the Right Side of the Hiatal Defect: The lesser omentum overlying the caudate lobe of the liver is opened. This allows for entry into the lesser sac and visualization of the right limb of the esophageal crus (RL) The vessels that cross over the caudate lobe and RL are dissected and elevated by the surgeon, clipped using Hem-o-lock Clips (Telleflex Inc., Morrisville, NC, USA) which are introduced through Port #4 by Assistant #2, and divided. This gives full visualization of RL. It is imperative to open the peritoneum overlying the RL. The space between the peritoneum and the muscle of RL needs to be entered. This is a natural and relatively avascular plane. Dissection in this plane allows for mobilization of the peritoneal sack and the contents of the hiatal defect with virtually no blood loss and perfect exposure. The Endo-Kittner which is introduced through Port # 5 and manned by Assistant #2, is placed at the 11 o'clock position of RL and used to retract RL laterally. Next the Endopaddle retractor manned by Assistant #1 and introduced through Port #6 is placed at the 7 o'clock position of the esophageal crus and used to sweep the tissues in a caudal and leftward direction. These maneuvers allow the surgeon to grasp the peritoneum and dissect in the avascular plane between the pleura and the hiatal sac. (If the pleura is entered, the anesthesiologist clamps the ipsilateral lung (right), clips are placed to close the pleural opening, and after the completion of the dissection, a 24 French Chest tube is placed through an anterior thoracostomy. It is important to dissect the right side of the hiatal defect first. The dissection is then carried inferiorly until the posterior "V" formation between the RL and the left limb of the esophageal crus (LL) is identified. The LL is deeper than RL and is covered with fatty tissue. It is important to dissect the fatty tissue which overlies the LL until the muscle fibers are visualized. At this point the esophagus is elevated with the grasper in the left robotic hand, and the posterior aspect of the esophagus is separated from the crural "V" and the aorta. This maneuver allows for the identification and preservation of the Right (Posterior) Vagal Nerve.

Step 2. Dissection of the Arch of the Esophageal Hiatus: Assistant #2 introduces an Endo-Kittner through Port # 4. This Endo-Kittner is used to retract the right limb of the esophageal crus (RL) laterally. The surgeon uses a sweeping

maneuver with the hook cautery to separate the adventitial tissue and some blood vessels from the 11 O'clock to 2 o'clock position of the hiatus. The anterior vagus nerve is deep to these tissues and is not in danger of injury.

Step 3. Dissection of the Left Side of the Hiatal Defect:

The Endopaddle retractor is positioned at the 3 o'clock position and used to retract the tissues at the hiatus laterally to the right of the patient and in a caudal direction. The LL is identified and the tissues overlying the LL are dissected away until the muscle is visualized. The key to the hiatal dissection is to use the limbs of diaphragmatic crus as a landmark. The dissection of the LL is then carried inferiorly and laterally to the right of the patient until the "V" with the RL is identified. If the left pleura is entered, the same strategy as with the right pleural entry is utilized: the left lung is deflated, the pleural defect is closed with clips, a chest tube is placed through an anterior thoracostomy, and the exposure of the hiatus and pneumoperitoneum is maintained.

Step 4. Encircling the Esophagus:

It is important to resist the temptation of encircling the esophagus above the crural opening. In patients with large hiatal hernias, the only constant anatomic landmark is the muscle of the crus. Therefore, in order to prevent injury to the aorta or the esophagus, the esophagus must be encircled at the crus. The Endopaddle retractor is used to sweep the tissues at the hiatus to the left of the patient and caudally and the "V" formation between the RL and LL is identified. The grasper in the left robotic arm is placed behind the esophagus and used to follow the muscle of LL in an oblique sweeping motion from a cuadad to cephalad direction and toward the patient's left shoulder. Assistant #2 passes a vessel loop through Port # 4, the vessel loop is retracted around the esophagus and a Hem-o-clip is used to attach the two limbs of the vessel loop together. The excess vessel loop is cut and removed. Next, Assistant #2 introduces a laparoscopic grasper through Port #4, the vessel loop just above the Hem-o-clip is grasped and the esophagus is retracted laterally to the left of the patient.

Step 5. Completion of the Mediastinal Dissection:

In order to repair the hiatus in an anatomic fashion at a later point in the procedure, the esophagus needs to be dissected free from the mediastinal tissues. This dissection should be carried posterior to the pericardium, to the level of the inferior pulmonary vein. Complete dissection and mobilization of the esophagus facilitates a tension free primary repair and places at least 2cm of esophagus below the hiatal reconstruction. Assistant #2 retracts the esophagus laterally to the left and then to the right, thereby facilitating exposure of the periesophageal mediastinal tissues. Esophageal dissection is continued laterally and superiorly at least to the level of the inferior pulmonary vein. All vascular and adventitial connections to the esophagus are divided such that the vessel loop can be moved freely up onto the distal esophagus. In addition the periesophageal fat pad and migrated retroperitoneal fatty tissue is dissected away from the esophagus. Frequently retroperitoneal fat, and at times lesser sac fatty tissue, that migrates between the posterior vagus nerve and the esophagus on the right side of the hiatal defect or the

lesser curve aspect of the GE junction. In addition, fatty tissue from the retroperitoneum can migrate behind and to the left of the esophagus at the greater curve aspect of the GE junction. The retroperitoneal fatty herniation results in kinking and twisting of the esophagus and will need to be dissected away. At the end of the dissection, the esophagus and the vagus nerve should be the only tissues that remain within the encircling vessel loop.

Step 6. Anatomic and Physiologic Repair of the Esophageal Hiatus:

The strategy is to recreate the normal anatomy of the hiatus and thereby recreate the normal gastroesophageal antireflux barrier. This step can be carried out with the use of the robot or by conventional laparoscopy. We prefer conventional laparoscopy for this step. In our experience laparoscopic suturing with extracorporeal knot tying technique is more rapid and facilitates more accurate knot placement under tension. The crucial role of the robot and its significant differential advantage to laparoscopy is in the dissection of the hernia sac, and full mobilization of the esophagus. In order to accomplish full esophageal mobilization to the level of the inferior pulmonary veins, many times the pleura needs to be entered and the esophagus needs to be dissected away from the inferior pulmonary ligament. This level of accurate and extensive dissection cannot be accomplished by laparoscopy. However, as the repair phase of the procedure is confined to the hiatus, laparoscopic or robotic repair are equivalent and are dictated by the surgeon's preference.

The antireflux mechanism is the result of the intussusception of the esophagus into the stomach by 2 cm anteriorly spanning approximately 240 degrees of the circumference of the gastroesophageal junction from the 8 0'clock position on RL to 4 0'clock position on LL. The intussusception forms a horseshoe shaped valve which opens and closes like a trap door. The intussusception of the esophagus enters at an acute angle and the esophagogastric junction is angulated posteriorly. This complex three-dimensional relationship is held in place and is suspended onto the esophageal hiatus.

Step 6a—Posterior Crural Closure: Posterior crural closure is accomplished by reapproximating the RL and LL with two or three sutures. We prefer the Endostitch Instrument (Medtronic Inc. Norwalk, Conn., USA) with O Ethibond suture. The Endostitch Instrument is an ideal suturing device for laparoscopy as it facilitates one-handed suturing thereby allowing the surgeon's left hand to provide appropriate exposure. Furthermore, when approximating the RL and LL of the right crus posteriorly, the straight needle of the Endostitch Instrument passes in a tangential plain anterior to the aorta and carries a lower risk of inadvertent aortic injury which usually is the result of deep suture placement with a curved needle. The curved needle used with a laparoscopic needle driver can pass deeper than intended and can engage the anterior wall of the aorta.

The Endopaddle retractor is placed on the medial aspect of the esophagus and used to retract the esophagus laterally and to the left. The maneuver exposes the "V" shaped posterior junction of the RL and LL of the right crus. A 1 cm squared absorbable pledget cut from Vicryl mesh (Ethicon, Inc., Sommervile, NJ, USA) is

passed through Port #5. The Endostitch with O Ethibond is passed through Port #4. Intracorporeally the pledget is loaded onto the needle. The needle is passed through LL, a second pledget is loaded intracorporeally onto the needle, and the needle is passed through RL. Next, intracorporeally the needle is passed through a third vicryl pledget which is introduced with the grasper in the surgeon's left hand. The Endostich carrying the suture is withdrawn out of the entry Port #4, and extracorporeal knots are placed using a long external knot pusher. The suture is cut above the knot. This technique is repeated for all the posterior crural sutures.

Step 6b—Suspension of the Esophagus onto the Esophageal Crus: The camera is moved to Port #7. In a similar manner an O Ethibond suture on the Endostitch device is introduced through Port #4. Intracorporeally the pledget is loaded onto the needle, the needle is passed through LL at the 4 O'clock position, then through the lateral wall of the esophagus just above the GE junction at the greater curve, a second Vicryl pledget is loaded as described, and the suture is tied using extracorporeal technique. This fixes the left lateral aspect of the esophagus to the esophageal hiatus and recreates the normal attachment of the phreno-esophageal ligament. Next, an O Ethibond suture on the Endostitch device is introduced through Port #4. Intracorporeally the pledget is loaded onto the needle, the needle is passed through the medial wall of the esophagus just above the GE junction at the lesser curve, through RL at the 8 O'clock position, then, a second Vicryl pledget is loaded as described, and the suture is tied using extracorporeal technique. This fixes the right medial aspect of the esophagus to the esophageal hiatus and recreates the normal attachment of the phreno-esophageal ligament.

Step 6c—Anterior Crural Closure: In a similar manner to the posterior crural closure, 0 Etibond sutures on the Endostitch instrument with intracorporeally loaded pledgets of vicryl mesh are used to reapproximate the anterior portion of the crural arch. The anterior crural closure allows for the formation of an acute angle at the Gastroesophageal junction and recreates one of the important features of the normal Antireflux Barrier. The sutures are passed through Port #4, a Vicryl pledget is loaded on the suture intracorporeally and the suture is passed through the LL, a second pledget is loaded intracorporeally onto the needle, and the needle is passed through LL at the crural arch. A third Vicryl pledget is loaded intracorporeally onto the suture and the suture is tied using extracorporeal technique as outlined previously. Usually one to two anteriorly placed sutures are required. The crural closure is sized based on the passage of a 60 French Bougie into the distal esophagus.

Step 6d—Creation of the Normal Gastroesophageal Valve: Following crural closure, the normal gastroesophageal valve is re-created. The intussusception of the esophagus into the stomach is accomplished for the anterior 240-degrees (from RL to LL of the right crus) of the 360-degree circumference of the esophagogastric junction. The esophagus is marked 2 cm above the esophagogastric junction (EG) at the 4 o'clock position lateral to Left Vagus nerve(E1), at the 8 O'clock position

just anterior to the Right Vagus nerve (E3) and halfway in between at approximately the 11 o'clock position (E2). The stomach is marked 2 cm below the GE junction at the greater curvature (G1), the Lesser curvature (G3) and at a point halfway between G1 and G3 (G2) (Figure 5).

The Endostitch instrument with 0 Ethibond is introduced through Port #4. The first suture (G3 to E3, Lesser Curve) passes from G3 to E3 and through the diaphragm at the right crural limb, RL at 8 O'clock position. A vicryl pledget is introduced with a grasper through Port #5, and the suture is passed through the pledget. The suture is withdrawn through port #4. The suture is tied using extracorporeal knot tying technique.

The second suture (G1 to E1, Greater Curve) is passed in a similar manner from G1 to E1 and through the diaphragm at the left crural limb, LL at 4 O'clock position. A vicryl pledget is introduced with a grasper through Port #5, and the suture is passed through the pledget. This suture is withdrawn from Port #4 and tied using a knot-pusher and extracorporeal knots.

The third Suture (G2 to E2, midpoint) is introduced in the same manner from G2 to E2 and through the diaphragm at the midpoint of the crural arch. This suture is withdrawn from Port #4 and tied using a knot-pusher and extracorporeal knots.

Placement of the Valvuloplasty sutures results in the intussusception of the esophagus into the stomach by 2cm for approximately 240 degrees and recreates the normal gastroesophageal valve (Figure 6).

At this point the newly created Gastroesophageal Valve is graded based on the
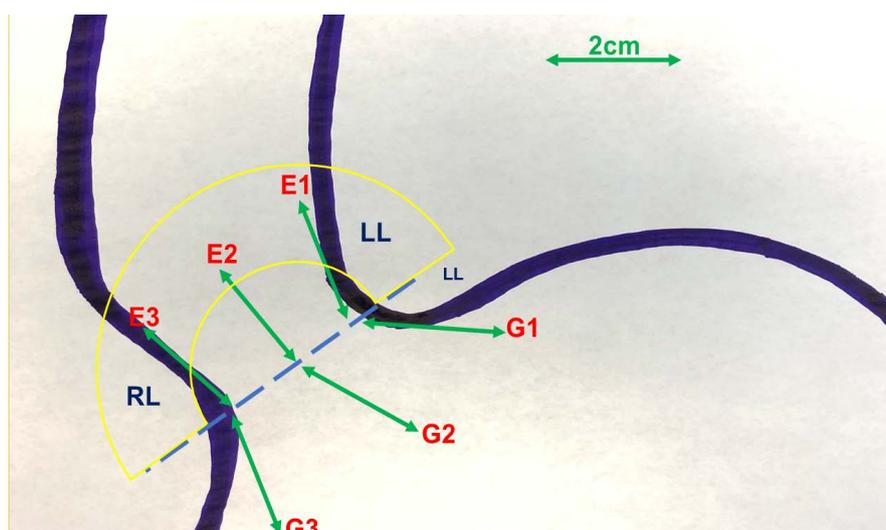
Figure 5. Creation of the Normal Gastroesophageal Valve: The intussusception of the esophagus into the stomach is accomplished for the anterior 240-degrees (from RL to LL of the right crus) of the 360-degree circumference of the esophagogastric junction. The esophagus is marked 2 cm above the esophagogastric junction (EG) at the 4 o'clock position lateral to Left Vagus nerve(E1), at the 8 O'clock position just anterior to the Right (posterior) Vagus nerve (E3) and halfway in between at approximately the 11 o'clock po-sition (E2). The stomach is marked 2 cm below the GE junction at the greater curvature (G1), the Lesser curvature (G3) and at a point halfway between G1 and G3 (G2).

Figure 6. Retroflexed endoscopic view of the newly created Gastro-esophageal Valve Placement of the Valvuloplasty sutures results in the intussusception of the esophagus into the stomach by 2 cm for approximately 240 degrees and recreates the normal gastroesophageal valve.

Hill I-IV grading system using intraoperative endoscopy. Only a Grade I Valve is acceptable. Any deviations which would necessitate a Grade less than Grade I need to be corrected at this time and before removal of the ports.

Step 7. Evacuation of CO_2, and Port Closure: Only the camera port needs to be closed. This trocar site is closed using a laparoscopic suture passer and 0 Vicryl (Ethicon Endo-Surgery). CO_2 is evacuated from the highest trocar by placing the patient in a steep Reverse Trendelenburg position. The other Versiport trocars are removed and the tissues are allowed to close around the introducer sheath. Subcutaneous tissues are closed with 00 Vicryl and the skin is closed with staples.

3. Results

Evaluating the success of Robotic Anatomic and Physiologic Repair of large HHs (RRHH) requires long-term follow-up.

In a prospective cohort study, we evaluated patients undergoing RRHH with at least a 2-year follow-up. All patients undergoing elective (RRHH) were identified preoperatively and enrolled prospectively in this study. Exclusion criteria included previous repair of HH, previous fundoplication, esophageal surgery for a malignant disease process, any subject unwilling to provide informed consent, or any individual who was unwilling to undergo the required follow-up studies.

Preoperative characteristics, medical comorbidities, and clinical information were all recorded prospectively by trained research personnel and recorded into a secure surgical outcomes database.

Postoperatively, patients typically were started on a full liquid diet and advanced quickly to a soft diet for 2 weeks postoperatively. A registered dietitian assisted with teaching in all patients before discharge. The patients were followed by surgical clinic visits, clinic visits with their gastroenterologist, and telephone consultation by specially trained Nurse Practitioners. In addition, the patients were followed by their local gastroenterologist by at least semi-annual clinic visits and endoscopy.

All patients received the previously validated Gastroesophageal Reflux Disease-Health-Related Quality of Life (GERD-HRQL) questionnaire preoperatively and at postoperative time points of 1 month and 1 year and 2 years. The questionnaire consists of 10 questions with a maximum score of 50 (6 questions relate to gastroesophageal reflux disease, 2 questions relate to swallowing, 1 question relates to bloating, and 1 for medication use). A greater score indicates a worse symptom severity. Patient satisfaction with their current condition was determined at each time point. These questionnaires were administered by trained personnel during scheduled clinic visits.

Patients routinely had a barium swallow postoperatively before discharge but did not undergo a barium swallow, an endoscopy, or a CT scan study at the 1-month time point unless indicated by symptoms. At 6 months, 1 year, and yearly intervals thereafter, all patients received an endoscopy study to ascertain the presence of a recurrence, regardless of symptoms.

Recurrence was defined as over 2 cm or 10% of the stomach above the diaphragm detected by CT, esophagogram or endoscopy. It is important to point out that due to the intussusception of the esophagus into the stomach by 2 cm in the process of creating the GE Valve, there will always be 2cm of stomach above the GE junction for 240 degrees of the circumference. Any stomach above the diaphragm, however, represents a recurrence. In order to decrease the chance of bias, the studies were interpreted by the referring gastroenterologists, and independent radiologists who were reminded of the study parameters and definitions but were blinded to the rest of the clinical data.

423 patients underwent RRHH. With a long term follow up, there was a significant decrease in the Median Symptom Severity Score from 42 preoperatively to 3, postoperatively. Recurrence was seen in 5 patients (5/423) for a recurrence rate of 1.1%.

4. Conclusions

For the first half of the twentieth century, Hiatal Hernias (HHs) were repaired "Anatomically". The anatomic repair of hiatal hernias was not successful in relieving the symptoms in patients with hiatal hernias and reflux disease. Therefore, with a 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 1970s, investigators proposed that hiatal hernias were irrelevant, and the answer was in the relief of GERD. From the 1950s until recently, Hiatal hernia surgery evolved from "Anatomic Repair" to "Physiologic Restoration". 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) A 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) Changes in the definition of symptomatic hiatal hernias;

6) Possibility of complex anatomic reconstruction using minimally invasive techniques which has been brought about from the advances in intraoperative three-dimensional visualization and greater instrument dexterity provided by the robotic platform.

In addition, the indication for the repair of symptomatic HH has evolved from gastrointestinal symptoms to the more insidious respiratory and cardiovascular symptoms which result from cardiac tamponade.

For several reasons, the syndrome of cardiac tamponade from HH creates a natural laboratory for cognitive errors in diagnosis. First, neither the literature nor national guidelines provide the base rate of tamponade expected for the typical patient with HH. Uncertainty about whether this problem is a "zebra or a horse" distorts Bayesian reasoning, which makes both the patient and provider normalize or dismiss tamponade symptoms from a HH or causes the misdiagnosis of acute/chronic pulmonary disease. Referral to specialists often is unable to resolve the dilemma because of the silo mentality that characterizes these two specialties. Finally, the diagnosis is difficult to confirm a priori. Signs of classic tamponade on echocardiography (diastolic collapse of the right ventricle and/or right atrium, exaggerated respiratory variability in mitral inflow velocity, and inferior vena cava plethora) are not usually seen with focal extrinsic cardiac compression of the left atrium or inferior vena cava by a GHH. Instead, echo images are often misinterpreted as pulmonary hypertension or hypovolemia. A disease with an unknown incidence that occurs mainly in complex patients and cuts across specialties that are traditionally siloed from each other, and does not have a clear method for diagnosis, will inevitably lead to underdiagnosis by overburdened providers. On the other hand, immediate resolution in tamponade and the associated symptoms after GHH repair makes the diagnosis easy to confirm in retrospect.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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