Published in final edited form as:

Surgery. 2010 January ; 147(1): 57–64. doi:10.1016/j.surg.2009.05.011.

Unique features of esophagogastric junction pressure topography in hiatus hernia patients with dysphagia

John E. Pandolfino, MD, MSCI, Monika A. Kwiatek, PhD, Kim Ho, BA, John R. Scherer, MD, and Peter J. Kahrilas, MD

Department of Medicine, Division of Gastroenterology, Feinberg School of Medicine, Northwestern University, Chicago, IL

Abstract

Background—Our aim was to assess pressure dynamics within the esophagogastric junction (EGJ) in sliding hiatus hernia (HH) during normal peristalsis and to compare the pressure profiles of HH patients with gastroesophageal reflux disease (GERD) symptoms (HH-GERD) to HH patients with dysphagia (HH-dysphagia).

Methods—High-resolution manometry studies in 230 consecutive patients and 68 controls were reviewed. HH patients were defined by a ≥ 1.5 cm separation between the lower esophageal sphincter (LES) and crural diaphragm (CD) on pressure topography plots. The HH population was further culled to eliminate those patients with motor disorders or stricture. The study groups were composed of 18 HH patients with only reflux symptoms and 10 HH patients with only dysphagia. Analysis of the pressure dynamics within the EGJ was performed at rest and after swallowing to independently quantify the LES and CD contributions to residual EGJ pressure, as well as the magnitude and genesis of distal esophageal intrabolus pressure (IBP). Differences among study groups were analyzed with analysis of variance.

Results—After swallows, HH-dysphagia patients had greater residual CD pressure (9 mmHg; standard deviation [SD], 4) and IBP pressure (19 mmHg; SD, 4) compared to HH-GERD patients (5 mmHg; SD, 2; and 12 mmHg; SD, 2, respectively; $P < .001$) or normal subjects (NA; 11 mmHg; SD, 3; $P < .001$).

Conclusion—Sliding HH alters the pressure dynamics through the EGJ and can lead to a functional obstruction. Patients with HH and dysphagia have greater pressures through the CD compared to HH patients with GERD symptoms, supporting the hypothesis that sliding HH in and of itself may be responsible for dysphagia.

The pathophysiologic consequences of sliding hiatus hernia (HH) mainly pertain to reflux disease and compromise of the antireflux barrier.¹ HH is associated with a decreased pressure at the esophagogastric junction (EGJ) and a decrease in length of the functional EGJ. These changes are attributable to axial separation of the lower esophageal sphincter (LES) and crural diaphragm (CD) and possibly to a loss of the abdominal segment of the LES.² Furthermore, reflux disease patients with HH may exhibit dilatation of the crural aperture resulting in increased EGJ compliance and wider opening diameter during relaxation.³ In combination, these defects compromise the CD as a component of the antireflux barrier and likely increase the volume of refluxed fluid during reflux events.

Although the HH defects predisposing to reflux should also reduce resistance to antegrade flow through the EGJ after swallowing, HH has also been implicated as a cause of dysphagia. Kaul et al⁴ used fluoroscopy and manometry to study patients with HH and dysphagia but without significant esophagitis or motility abnormalities. Fluoroscopy revealed that hiatal flow obstruction, manifest by hold-up of swallowed contrast and retrograde escape, was more common in HH patients with dysphagia than those without dysphagia. The authors speculated that this finding was due to compression of the herniated stomach within the hiatal canal, and they correlated these findings with a double-hump axial pressure profile found on pull-through manometry.⁴

Studies that are more recent suggest additional pathophysiologic aberrations associated with HH that could impair esophagogastric bolus transit. Using synchronized fluoroscopy and manometry through the EGJ, Lin et al⁵ concluded that emptying of the distal esophagus was more a function of the elastic recoil of phrenoesophageal attachments restoring resting esophageal length after peristalsis-associated shortening than it was of the peristaltic contraction per se. Both peristalsis-associated shortening and the subsequent recoil are reduced with HH, suggesting that HH might compromise this function.⁶ Furthermore, even in the absence of HH, the sustained CD contraction associated with a Müller maneuver can compromise esophageal emptying by outlasting the time-locked peristaltic contraction until supradiaphragmatic intrabolus pressure (IBP) exceeds esophageal (or LES) closure pressure resulting in retrograde flow.⁵

Although the physiologic evidence detailed above suggests mechanisms whereby HH may impede esophageal emptying, there are minimal clinical data substantiating an association between sliding HH and dysphagia. This lack of data is in part due to inconsistencies in defining sliding HH⁷ and in part due to the multitude of potential confounding variables, such as esophagitis, peptic stricture, peristaltic dysfunction, and Schatzki ring. Recently, high-resolution esophageal pressure topography has become available as a practical method to perform a detailed characterization of LES, CD, and IBP pressure dynamics within the EGJ after swallowing.

The aim of this study was to determine whether or not the detailed characterization of these intra-EGJ pressure dynamics in well-characterized HH patients revealed a characteristic pattern associated with dysphagia.

METHODS

Subjects

High-resolution manometry (HRM) studies performed on 230 consecutive patients (90 males; age range, 19–94 years) between January 2008 and July 2008 at Northwestern Memorial Hospital were reviewed retrospectively to identify patients with manometric evidence of HH using the criterion of ≥ 1.5 -cm separation between the LES and the CD during the baseline recording. Endoscopic records of HH subjects were retrieved to identify and exclude patients with potential inflammatory or structural etiologies for dysphagia. Patients with type II, III, or IV HH were excluded, because these paraesophageal hernias are known to be associated with mechanical obstruction and our aim was to investigate the role of smaller type I (sliding) HH. Given our focus on the relationship between pressure dynamics within the EGJ during swallowing and dysphagia, patients with peristaltic weakness or peristaltic dysfunction were also excluded because they were potentially confounding variables. In addition, we included a comparator group of 68 asymptomatic controls (33 males; age range, 19–57 years) without HH. The study protocol was approved by the Northwestern University Institutional Review Board.

HRM protocol

All subjects underwent a standard HRM lab protocol including a brief interview, physical exam, anthropometric measurements, and questionnaire completion. The HRM procedure was also standardized. A solid-state HRM assembly with 36 pressure sensors spaced at 1-cm intervals (optical density [OD], 4.2 mm) was used (ManoScan; Sierra Scientific Instruments Inc., Los Angeles, CA). The recording characteristics of this device have been described previously.⁸ The transducers were calibrated at 0 and 100 mmHg using externally applied pressure before the study. The HRM assembly was placed transnasally, and the manometric catheter positioned to record from the hypopharynx to the stomach with about 5 intragastric sensors. Studies were performed with the patients in a supine position after at least a 6-hour fast. The HRM protocol included a 3-minute period to assess basal EGJ pressure and 10 water swallows of 5 mL each.

HRM analysis

All HRM analysis was performed using the ManoView software package (Sierra Scientific Instruments Inc.) applied to the data tracings viewed in the color pressure topography mode of the HRM assembly. The proximal border of the EGJ was defined by a pressure increase ≥ 2 mmHg/cm pressure relative to intraesophageal pressure, and the distal border of the EGJ was defined by a pressure increase ≥ 2 mmHg/cm relative to intragastric pressure. When there was a double-peaked EGJ pressure profile on the isobaric contour plot during inspiration, the proximal peak was defined as the LES and the distal as the CD. The LES-CD separation was defined as the distance separating the peaks at inspiration.

Given the 1-cm spacing and the 2.5-mm length of the pressure sensor-recording segment, a double peak signal on the isobaric contour correlates with a minimal detectable separation of approximately 1.5 cm. Two investigators independently measured LES-CD separation for each patient directly from the color isobaric contour plots using the ManoView SmartMouse tool (Sierra Scientific Instruments Inc.). End-expiratory EGJ pressure was measured during the 3-minute baseline recording using the eSleeve tool (Sierra Scientific Instruments Inc.) spanning the entire EGJ. End-expiratory LES and CD measurements were made in the HH subjects by restricting the eSleeve (Sierra Scientific Instruments Inc.) domain to a 2-cm span across each of these elements, respectively.

Patients with HH were then further analyzed to exclude those with distal peristaltic weakness or dysfunction. These analyses were performed using the following methods: (1) the isobaric contour tool set at 30 mmHg to ascertain whether the peristaltic wavefront was intact; (2) the contractile front velocity (CFV) to ascertain normal propagation velocity; and (3) the distal contractile amplitude to identify hypertensive contractions.⁹ Each swallow was characterized as follows: (1) *normal* (intact isobaric contour, CFV < 8 cm/s, mean contractile amplitude < 180 mmHg); (2) *hypotensive* (> 3 cm break in the 30 mmHg isobaric contour between the distal segment and the EGJ); (3) *absent peristalsis* (complete failure of contraction); (4) *hypertensive* (CFV < 8 cm/s and mean contractile amplitude > 180 mmHg); (5) *spastic* (rapidly propagated contraction with CFV ≥ 8 cm/s); (6) *increased IBP* (> 15 mmHg compartmentalized pressure between the EGJ and peristaltic wavefront); or (7) *panesophageal pressurization* (simultaneous esophageal pressurization to > 30 mmHg extending from the upper esophageal sphincter to the EGJ). Patients were required to have 7 intact, normally propagated peristaltic contractions to be included in the analysis. Thus, the 7 swallows could be a combination of normal, hypotensive, and increased IBP (Fig 1). Patients with distal esophageal spasm were also excluded.

The pressure profile within the EGJ was then further analyzed using the ManoView integrated relaxation pressure (IRP) tool (Sierra Scientific Instruments Inc.).¹⁰ The default settings on the

IRP tool establish a $6\text{ cm} \times 10\text{ s}$ domain after the swallow and calculate the lowest mean eSleeve pressure for 4 contiguous or noncontiguous seconds of relaxation within that window; however, in the setting of HH, the IRP could conceivably be indicative of either LES relaxation or CD relaxation. Thus, the default IRP setting spanning both the LES and the CD components was reported as IRP_{EGJ} . To measure the deglutitive relaxation pressure through the LES and CD separately, the IRP boundaries were adjusted to a 2-cm domain, capturing each EGJ element independently (Fig 2).

Separate measurements were then made of IRP_{LES} and IRP_{CD} . All IRP measurements through the EGJ were referenced to concurrent intragastric pressure. Additionally, IBP was measured proximal to the EGJ using a new ManoView tool (Sierra Scientific Instruments Inc.). This measurement (IBP_{IRP}) averages the IBP 1-cm proximal to the EGJ during the 4-second period used to calculate the IRP, regardless of whether the IRP measure had been applied to the EGJ, LES, or CD. Thus, IBP_{IRP} provides a measurement of the IBP during the 4-second period after a swallow when the obstructive pressure through the EGJ (attributable to both the LES and CD) is at its lowest (Fig 2). All IBP measurements were referenced to atmospheric pressure.

Clinical variables

At entry, patients completed a validated symptom questionnaire focused on GERD (Reflux Disease Questionnaire [RDQ]; AstraZeneca R&D, Mölndal, Sweden). In addition, medical records were reviewed by an investigator blinded to the manometric analysis to determine the dominant symptoms at presentation based on the specialist referral note before HRM. Endoscopic images and reports were also reviewed. Patients were categorized as *HH-GERD* if their primary referral indication was for symptoms of GERD based on their most recent clinic note and their RDQ response data confirmed GERD symptoms. Patients were categorized as *HH-dysphagia* if the primary referral indication was dysphagia and they had a normal RDQ score.

Statistical analysis

The manometric parameters and clinical variables obtained from the medical records were summarized using mean and standard deviation (SD). Analysis of variance was used to compare the manometric parameters among the 3 study groups (HH-GERD, HH-dysphagia, Controls). Chi-square analysis was used to compare categorical variables among the 2 patient groups and controls.

RESULTS

Of the 230 patients referred for HRM, 64 met our manometric criteria of HH. A total of 2 patients were excluded because of peptic stricture, and 34 patients were excluded because of the predefined criteria for peristaltic weakness or peristaltic dysfunction. Of the remaining 28 patients with intact peristalsis, 10 had a primary complaint of dysphagia and normal RDQ score (HH-dysphagia), and 18 had a primary complaint of heartburn or regurgitation along with an abnormal RDQ score (HH-GERD). Patients in the HH-dysphagia group were significantly older and had significantly lower RDQ scores (Table I).

EGJ pressure measurements during the interdeglutitive period

Table II summarizes and compares the EGJ pressure characteristics of the control subjects, HH-GERD patients, and HH-dysphagia patients during the interdeglutitive period. Patients with HH-GERD had numerically lower basal EGJ pressures compared to normal subjects ($P = .07$) whereas HH-dysphagia patients had similar EGJ pressures compared to controls. Average EGJ pressure of the HH groups was slightly different than the measurements of either

EGJ component because there was variability in the localization of maximal EGJ pressure among HH patients.

Specifically, the LES component of the EGJ high-pressure zone was greater than the CD component in 10 of 18 HH-GERD patients and 5 of 10 HH-dysphagia patients. The HH-GERD patients had numerically greater LES-CD separation compared to the HH-dysphagia patients (Table II). The difference, however, was slight, and there was substantial overlap between groups (HH-GERD group: range, 1.5–4.5 cm; HH-dysphagia group: range, 1.5–3.0 cm).

Pressure measurements through the EGJ during deglutitive relaxation

Table III summarizes the pressure dynamics through the EGJ after test swallows. Note that, when considered as a single group, the HH patients had similar IRP_{EGJ} values to the normal subjects; when considered independently; however, the HH-dysphagia group had significantly increased values of IRP_{EGJ} . Furthermore, the magnitude of residual CD relaxation pressure within the EGJ was significantly different between HH groups; on average, IRP_{CD} was significantly greater among the HH-dysphagia patients (9 mmHg; SD, 4) than among the HH-GERD patients (5 mmHg; SD, 2). Supporting the physiologic significance of the increased IRP_{CD} , the reduced CD relaxation pressure was paralleled by increased mean IBP_{IRP} among the HH-dysphagia patients (19 mmHg; SD, 4) compared to either the HH-GERD patients (12 mmHg; SD, 2) or normal subjects (11 mmHg; SD, 3; Fig 3). The increased IBP attributable to residual CD relaxation pressure is also clearly evident in the example illustrated in Fig 2, C.

The data in Tables II and III also emphasize a significant limitation in the interpretation of deglutitive EGJ relaxation pressure in the setting of a small sliding HH. A 6-cm sleeve device (or the eSleeve equivalent; Sierra Scientific Instruments Inc.) straddles multiple physiologic domains, detecting the greatest residual pressure among them. Indicative of this, 5 of the 10 HH-dysphagia patients had IRP_{EGJ} values greater than 15 mmHg, the range consistent with a diagnosis of achalasia.¹⁰ Thus, these individuals would be diagnosed potentially as *achalasia* if they were aperistaltic or a *variant of achalasia* if they had some degree of peristalsis (Fig 4). The abnormal IRP_{EGJ} in these 5 patients, however, was a manifestation of the increased IBP just proximal to the CD, because the IRP_{LES} and IRP_{CD} values separately were both within normal limits.

DISCUSSION

The aim of this study was to determine whether a detailed analysis of intra-EGJ pressure dynamics in well-characterized Type I (sliding) HH patients revealed a characteristic pattern associated with dysphagia. The major finding was that sliding HH is a heterogeneous condition such that HH patients with dysphagia and without reflux symptoms exhibit functional EGJ obstruction localized to the CD. This condition was associated with abnormally high deglutitive EGJ relaxation pressure and increased esophageal IBP of sufficient magnitude that it might be confused with achalasia in some cases. These changes were not seen in HH patients with reflux symptomatology.

Furthermore, our findings suggest that manometric indices of deglutitive EGJ relaxation in HH patients are not always indicative of LES relaxation, as the maximal residual pressure within the EGJ can be either at the CD or immediately proximal to this area in the form of an IBP or hernia sac pressure. Thus, at any 1 instant, a 6-cm value as measured with the eSleeve tool (Sierra Scientific Instruments Inc.) may be attributable to the LES, CD, IBP, or even intragastric pressure. Consequently, impaired deglutitive EGJ relaxation in HH patients has at least 2 potential causes: (1) achalasia with impaired intrinsic LES relaxation; or (2) a “tight” hiatal canal (Fig 4). These entities can be discerned with high-resolution esophageal pressure

topography, but only if care is taken to appropriately restrict the eSleeve analysis domain to each area of interest.

Although the mechanical obstruction associated with type II or III paraesophageal hernia is well known, there has been little scrutiny of the role of sliding HH in dysphagia. We recently analyzed 400 consecutive patients with HRM and described a subset that exhibited a pattern of EGJ obstruction associated with an abnormal EGJ relaxation pressure, intact peristalsis, and increased IBP.⁹ After excluding pathology at the EGJ (such as eosinophilic esophagitis, disrupted fundoplication, or stricture), we were left with a group of 14 (3.5%) with idiopathic functional EGJ obstruction. Although some of these patients may have had evolving achalasia, others had HH with normal LES relaxation and increased IBP. We hypothesized that these abnormalities could be the consequence of functional obstruction at the diaphragmatic hiatus attributable to a relatively normal-sized hiatal aperture surrounding the relatively thick-walled proximal stomach. Findings from this study support our previous hypothesis. After excluding patients with GERD symptoms, stricture, or compromised peristalsis, we concluded that sliding HH, in and of itself, can cause functional obstruction at the EGJ.

Hiatus hernia patients with dysphagia exhibited greater deglutitive CD relaxation pressure and IBP compared to HH-GERD patients or normal subjects. These data are consistent with a previous report from Kaul et al⁴ in which they suggested CD impingement on the herniated stomach manifests as a “double hump” axial pressure profile during a pull-through of the EGJ and a numerical difference in the amplitude of the distal (CD) hump between patients with and without dysphagia. Our results are in line with these findings; however, we were able to leverage the temporal and spatial resolution of high-resolution pressure topography plotting to analyze the pressure dynamics within the EGJ during swallowing and provide further insight into possible mechanisms. We confirmed that the distal hump of the axial pressure profiles localized at the CD and that increased IBP extended to this area in the HH-dysphagia patients, signifying that it was the site of obstruction. These findings were observed during normal LES relaxation, and they potentially represent a decrease in opening diameters of the CD in the context of intact peristalsis. This pattern is analogous to a patient with a slipped Nissen fundoplication, and studies have shown that these changes lead to retrograde escape and impaired bolus transit after the time-locked peristaltic sequence terminates.^{11,12}

In conclusion, our results suggest that HH can result in altered pressure dynamics within the EGJ, leading to functional EGJ obstruction in a subset of HH patients. HH patients with dysphagia but without GERD symptoms exhibited a relative obstruction at the CD. This observation supports the hypothesis that sliding HH, in and of itself, could be responsible for dysphagia. Furthermore, this analysis emphasizes that not all HH patients are the same. By assessing individual pressure components within the EGJ, one can theoretically identify clinically meaningful HH subtypes that could potentially help define treatment. Patients presenting with increased EGJ relaxation pressure in the context of a small type I HH require careful analysis of the various components of the EGJ before making a diagnosis of achalasia and before surgical myotomy or dilation are considered. Although it is intriguing to further speculate that the HH-dysphagia group could benefit from surgical intervention aimed at reducing the obstruction at the CD, prospective randomized studies are still required before this approach can be supported.

Acknowledgments

Supported in part by research grants from the from the Public Health Service (R01 DC00646 to P.J.K., J.E.P.).

REFERENCES

1. Mittal RK, Balaban DH. The esophagogastric junction. *N Engl J Med* 1997;336:924–32. [PubMed: 9070474]
2. Kahrilas PJ. The role of hiatus hernia in GERD. *Yale J Biol Med* 1999;72:101–11. [PubMed: 10780571]
3. Pandolfino JE, Shi G, Trueworth B, Kahrilas PJ. Esophagogastric junction opening during relaxation distinguishes nonhernia reflux patients, hernia patients, and normal subjects. *Gastroenterology* 2003;125:1018–24. [PubMed: 14517784]
4. Kaul BK, DeMeester TR, Oka M, Ball CS, Stein HJ, Kim CB, et al. The cause of dysphagia in uncomplicated sliding hiatal hernia and its relief by hiatal herniorrhaphy. A roentgenographic, manometric, and clinical study. *Ann Surg* 1990;211:406–10. [PubMed: 2322035]
5. Lin S, Brasseur JG, Pouderoux P, Kahrilas PJ. The phrenic ampulla: distal esophagus or potential hiatal hernia? *Am J Physiol* 1995;268:G320–7. [PubMed: 7864129]
6. Kahrilas PJ, Wu S, Lin S, Pouderoux P. Attenuation of esophageal shortening during peristalsis with hiatus hernia. *Gastroenterology* 1995;109:1818–25. [PubMed: 7498646]
7. Kahrilas PJ, Kim HC, Pandolfino JE. Approaches to the diagnosis and grading of hiatal hernia. *Best Pract Res Clin Gastroenterol* 2008;22:601–16. [PubMed: 18656819]
8. Pandolfino JE, El-Serag HB, Zhang Q, Shah N, Ghosh SK, Kahrilas PJ. Obesity: a challenge to esophagogastric junction integrity. *Gastroenterology* 2006;130:639–49. [PubMed: 16530504]
9. Pandolfino JE, Ghosh SK, Rice J, Clarke JO, Kwiatek MA, Kahrilas PJ. Classifying esophageal motility by pressure topography characteristics: a study of 400 patients and 75 controls. *Am J Gastroenterol* 2008;103:27–37. [PubMed: 17900331]
10. Ghosh SK, Pandolfino JE, Rice J, Clarke JO, Kwiatek M, Kahrilas PJ. Impaired deglutitive EGJ relaxation in clinical esophageal manometry: a quantitative analysis of 400 patients and 75 controls. *Am J Physiol Gastrointest Liver Physiol* 2007;293:G878–85. [PubMed: 17690172]
11. Ghosh SK, Kahrilas PJ, Lodhia N, Pandolfino JE. Utilizing intraluminal pressure differences to predict esophageal bolus flow dynamics. *Am J Physiol Gastrointest Liver Physiol* 2007;293:G1023–8. [PubMed: 17855763]
12. Pandolfino JE, Ghosh SK, Lodhia N, Kahrilas PJ. Utilizing intraluminal pressure gradients to predict esophageal clearance: a validation study. *Am J Gastroenterol* 2008;103:1898–905. [PubMed: 18637086]

Fig 1.

Methodology for characterizing the distal esophageal contraction. A total of 3 examples are shown: (A) normal, (B) hypertensive, and (C) increased intrabolus pressure (IBP). The color pressure scale for all 3 is to the left. Swallows were characterized by the integrity of the 30 mmHg isobaric contour (*black line*), the contractile front velocity (CFV) (*black dashed line*), and the mean distal contractile amplitude (*white box*). A contraction was characterized as intact only if the 30 mmHg isobaric contour did not exhibit a defect ≥ 3 cm in length between the distal contraction and the esophagogastric junction (EGJ) and the CFV was < 8 cm/s. Intact swallows were further characterized as *normal* if the mean distal contractile amplitudes was < 180 mmHg, *hypertensive* if the mean distal contractile amplitudes was > 180 mmHg, and *increased IBP* if the swallow was associated with an IBP measurement of > 15 mmHg.

Fig 2.

Methodology for analyzing esophagogastric junction (EGJ) pressure components in a (A) normal control, (B) hiatus hernia (HH) patient with gastroesophageal reflux disease (GERD) symptoms (HH-GERD), and (C) HH patients with dysphagia (HH-dysphagia). The pressure scale for each plot is on the left and positioned along the manometry catheter (cm) to the right. The *red line* indicates the time during the baseline recording corresponding to the single spatial pressure variation plot on the right of each pressure topography plot. The pressure profile within the EGJ was then further analyzed using the ManoView integrated relaxation pressure (IRP) tool ((Sierra Scientific Instruments Inc) encompassing (A) the entire EGJ (IRP_{EGJ}) (purple boxes) or restricted to (B, C) the lower esophageal sphincter (LES) (IRP_{LES}) (orange boxes) and crural diaphragm (CD) (black boxes) separately. The red boxes just above the IRP boxes indicate the time and location used to quantify the simultaneous IBP measurements in each case. In (A), there is overlap of the LES and CD, and the spatial pressure variation plot illustrates a single pressure peak before the swallow. The IRP_{EGJ} for this swallow is 2 mmHg and the IBP_{EGJ} is 9 mmHg. In the HH-GERD patient (B), the IRP_{LES} is 1 mmHg, whereas the IRP_{CD} is 4 mmHg and the IBP is 14 mmHg, suggesting minimal pressurization through the EGJ. In contrast, in the HH-dysphagia patient (C), the CD component of the basal EGJ pressure profile is more pronounced (distal hump), and the IBP_{IRP} (22 mmHg) is substantially greater than that of the HH-GERD patient. This finding reflects the greater resistance through the CD evidenced by an increased IRP_{CD} (11 mmHg).

Fig 3.

Comparison of the IRP_{CD} (*solid circles*) and the IBP_{IRP} (*open circles*) values of hiatus hernia (HH) patients with gastroesophageal reflux disease symptoms (HH-GERD) patients and HH patients with dysphagia (HH-dysphagia). HH-dysphagia patients had significantly greater mean IRP_{CD} ($P = .005$) and IBP_{IRP} ($P < .001$) values compared to HH-GERD patients.

Fig 4.

Example of a hiatus hernia (HH) patient with abnormal deglutitive esophagogastric junction (EGJ) relaxation (increased IRP_{EGJ}) attributable to distinct abnormalities within the EGJ. The swallows are presented as landscape plots to better illustrate the pressure gradients spanning the esophagus and proximal stomach. This patient has an obstructive sliding HH; lower esophageal sphincter (LES) relaxation is normal (IRP_{LES}), which is depicted in the oval, and the increased IRP_{EGJ} (17 mmHg) is attributable to increased intrabolus pressure (IBP) within the hernia, which is localized by the rectangle.

Table I

Demographic profile of the study population

	<i>Controls n = 68</i>	<i>HH-GERD n = 18</i>	<i>HH-dysphagia n = 10</i>
M/F	33/35	8/10	4/6
Age, y	37.4 (5.2)	49.4 (11.5)	69.5 (10.8) ^{*,†}
BMI, kg/m ²	NA	28.4 (5.6)	23.7 (7.9)
RDQ	NA	2.3 (1.5)	0.8 (0.5) [*]

All values are mean (SD) except M/F ratio.

HH-GERD, Hiatus hernia (HH) patients with gastroesophageal disease (GERD) symptoms; *HH-dysphagia*, HH patients with dysphagia; *M*, males; *F*, females; *BMI*, body mass index; *RDQ*, Reflux Disease Questionnaire.

^{*} $P < .05$ vs HH-GERD group.

[†] $P < .05$ vs normal subjects.

Table II

High-resolution manometric variables on the esophagogastric junction during the interdeglutitive period

	Normal subjects	HH-GERD	HH-dysphagia
EGJ pressure, mmHg	16.0 (8.2)	11.0 (5.7)	16.0 (7.7)
LES pressure, mmHg	NA	8.0 (6.9)	14.0 (10)
CD pressure, mmHg	NA	6.0 (4.0)	8.0 (4.9)
LES-CD separation, cm	<1	2.7 (0.8) [†]	2.1 (0.6) ^{*,†}

All values are mean (SD).

HH-GERD, Hiatus hernia patients with gastroesophageal disease symptoms; *HH-dysphagia*, hiatus hernia patients with dysphagia; *EGJ*, esophagogastric junction; *LES*, lower esophageal sphincter; *CD*, crural diaphragm; *NA*, not applicable.

* $P < .05$ vs HH-GERD group.

[†] $P < .05$ vs normal subjects.

Table III

High-resolution manometric pressure measurements of the esophagogastric junction during the deglutitive period

	Normal subjects	All HH	HH-GERD	HH-dysphagia
IRP _{EGJ} , mmHg	9.0 (3.2)	9.0 (5.5)	6.0 (3.2)	13.0 (5.1) ^{*,†}
IRP _{LES} , mmHg	NA	5.0 (4.3)	4.0 (3.0)	7.0 (5.5) [*]
IRP _{CD} , mmHg	NA	7.0 (3.9)	5.0 (1.7)	9.0 (3.8) [*]
Intragastric pressure, mmHg	5.0 (2.5)	9.0 (2.8) [†]	9.0 (3.0) [†]	10.0 (2.1) [†]
IBP _{IRP} , mmHg	11.0 (2.9)	15.0 (5.1) [†]	12.0 (2.0)	19.0 (4.3) ^{*,†}

All values are mean (SD) except M/F ratio

HH, Hiatus hernia; HH-GERD, HH patients with gastroesophageal reflux disease (GERD) symptoms; HH-dysphagia, HH patients with dysphagia; IRP, integrated relaxation pressure tool; EGJ, esophagogastric junction; LES, lower esophageal sphincter; CD, crural diaphragm; IBP, intrabolus pressure.

^{*} $P < .05$ vs HH-GERD group.

[†] $P < .05$ vs normal subjects.