Published in final edited form as:
J Clin Gastroenterol. 2008 ; 42(5): 627–635. doi:10.1097/MCG.0b013e31815ea291.

Esophageal Motility Disorders in Terms of Pressure Topography:

The Chicago Classification

Peter J. Kahrilas, MD, **Sudip K. Ghosh, PhD**, and **John E. Pandolfino, MD**
Department of Medicine, The Feinberg School of Medicine, Northwestern University, Chicago, IL

Abstract

Two recent advances have revolutionized the performance of clinical esophageal manometry; the introduction of practical high resolution manometry (HRM) systems and the development of sophisticated algorithms to display the expanded manometric dataset as pressure topography plots. We utilized a large clinical experience of 400 consecutive patients and 75 control subjects to develop a systematic approach to analyzing esophageal motility using HRM and pressure topography plots. The resultant classification scheme has been named as the Chicago Classification of esophageal motility. Two strengths of pressure topography plots compared with conventional manometric recordings were the ability to (1) delineate the spatial limits, vigor, and integrity of individual contractile segments along the esophagus and (2) to distinguish between loci of compartmentalized intraesophageal pressurization and rapidly propagated contractions. Making these distinctions objectified the identification of distal esophageal spasm, vigorous achalasia, functional obstruction, and nutcracker esophagus subtypes. Applying these distinctions made the diagnosis of spastic disorders quite rare: spasm in 1.5% of patients, vigorous achalasia in 1.5%, and a newly defined entity, spastic nutcracker, in 1.5%. Ultimately, further clinical experience will be the judge, but it is our expectation that pressure topography analysis of HRM data, along with its well-defined functional implications, will prove valuable in the clinical management of esophageal motility disorders.

Keywords

high resolution manometry; nutcracker esophagus; distal esophageal spasm

---

Two recent advances have revolutionized the performance of clinical esophageal manometry: the introduction of practical high resolution manometry (HRM) systems and the development of sophisticated algorithms to display the expanded manometric dataset as pressure topography plots. These developments permit the visualization of esophageal motility as a spatial continuum along the length of the esophagus with isobaric conditions among sensors indicated by color continuums. In conjunction with a multitude of closely spaced manometric sensors, this essentially eliminates the problem of movement-related artifact, which was always a weakness of conventional manometric systems.

In the context of esophageal motility, highly resolved pressure topography plots facilitate localizing and tracking focal areas of high pressure. Thus, sphincter relaxation can be accurately quantified as the greatest residual pressure within the spatial domain of the esophagogastric junction (EGJ) despite the fact that the sphincter may exhibit axial motion of up to 9 cm in some circumstances during profound longitudinal muscle contraction.[1] Similarly,

Reprints: Peter J. Kahrilas, MD, Northwestern University, Feinberg School of Medicine, Department of Medicine, Division of Gastroenterology, 676 St Clair St, Suite 1400, Chicago, Illinois 60611-2951 (p-kahrilas@northwestern.edu).

The authors declare no conflict of interest.

peristaltic contractions can be imaged and quantified in terms of their segmental constituents rather than at arbitrary distances relative to the lower esophageal sphincter (LES).[2,3] Figure 1 depicts the typical pressure topography of both sphincters and the entire length of intervening esophagus during a swallow. The relative timing of sphincter relaxation and segmental contraction and the position and length of the transition zone are readily demonstrated. Basically, HRM has the potential to much more precisely define the contractile characteristics of the esophagus and its sphincters.

With the adoption of HRM and pressure topography display methodology, there is a need to reconsider the classification of esophageal motility that was developed for conventional manometric systems. Conventional metrics simply do not apply to the color topography plots. Some clinicians have reacted to this shortcoming by transforming the unfamiliar pressure topography displays back to conventional line tracings and then applying a conventional analysis. Although a practical solution, that is, in essence, dumbing down the technology, abandoning whatever incremental gain may be achieved from the topographic plots. The alternative approach is to build a classification scheme that parallels conventional manometric classifications but enhances them on the basis of the strengths of the enriched technology. Toward that goal, we recently performed a comprehensive characterization of esophageal HRM data in 75 normal subjects and 400 patients using new analysis paradigms unique to pressure topography interpretation.[4–8] In the process, we developed a systematic, pressure topography oriented classification of motor disorders of the distal esophagus. It is our hope that this scheme along with the analytic process described herein will aid in the recognition and treatment of clinically relevant abnormalities of esophageal function.

HIGH RESOLUTION MANOMETRY

The dataset used to develop the Chicago Classification of esophageal motility was obtained using a consistent manometric and analytic protocol. A solidstate HRM assembly with 36 solid-state sensors spaced at 1-cm intervals was used (Sierra Scientific Instruments Inc., Los Angeles, CA). The response characteristics of this device, calibration procedure, and poststudy thermal correction algorithm have been described in detail elsewhere.[9] Each sensor is circumferentially sensitive and zeroed to atmospheric pressure. The HRM assembly was passed transnasally and positioned to record from the hypopharynx to the stomach with about 5 intragastric sensors. The manometric protocol included a 5-minute period to assess basal sphincter pressure and ten 5-mL water swallows.

Manometric data were analyzed using both ManoView analysis software (Sierra Scientific Instruments Inc., Los Angeles, CA) and custom programs written in Matlab (The MathWorks Inc., Natick, MA). Although pressure topography plots can be generated using ManoView, Matlab was also used because of greater flexibility in analysis and in customizing isobaric contour plots. However, it is important to note that all of the analysis paradigms described here can be performed or approximated using the current version of ManoView software.[8] Similarly, although some of the numerical cutoffs defining normality may change with the use of different devices, the principles of analysis described here generalize to any HRM system.

THE ALGORITHM OF ANALYSIS USING PRESSURE TOPOGRAPHY PARAMETERS

Recognizing that esophageal bolus transport is effected by the interaction of resistance through the EGJ, intrabolus pressure and esophageal closure pressure behind the bolus,[10] we applied a stepwise HRM analysis algorithm that focused on each of these 3 key functional variables. Patients were first characterized by the presence or absence of impaired deglutitive EGJ relaxation. This is essential because the pressure topography within the distal esophagus is greatly altered by impaired deglutitive relaxation.

Deglutitive EGJ relaxation was analyzed within defined temporal and spatial limits (Fig. 1). In most instances the spatial limits spanned from at least 2 cm above the proximal aspect of the EGJ at rest to its most distal aspect. Within the EGJ relaxation window, instantaneous maximal EGJ pressure was then ascertained for each instant; in essence, a sleeve type measurement. The duration of relaxation corresponding to a particular threshold EGJ relaxation pressure was determined as the cumulative time within the postdeglutitive relaxation period during which EGJ pressure was at or below that pressure. Plots were then constructed detailing the duration of EGJ relaxation with incrementally increasing relaxation pressure cutoffs (Fig. 2A) or the marginal EGJ relaxation pressure associated with incrementally increasing the specified relaxation duration (Fig. 2B). Note that Figure 2B is simply an X-Y transposition of Figure 2A. The plot depicted in Figure 2B was used to calculate the integrated relaxation pressure (IRP), defined as the minimal average pressure during a 3 or 4-second relaxation period. Thus, the IRP would be the integral of the curve in Figure 2B cut off at 3 or 4 seconds divided by 3 or 4 seconds, respectively. Alternatively, the 3-second nadir eSleeve (ManoView) relaxation, defined as the lowest mean relaxation pressure for a 3-second contiguous period, approximates the 3-second IRP with the caveat being that the 3-second nadir eSleeve value needs to be a contiguous period of time whereas the 3-second IRP does not.

Extensive analysis of several candidate measures of deglutitive EGJ relaxation concluded that the best 3 were the 3-second nadir eSleeve, 3-second IRP, and 4-second IRP, all being substantially better than nadir pressure or non–sleeve-type measures.[7] In the key clinical test of differentiating achalasia patients from nonachalasia, all 3 measures performed in the range of 95% sensitivity and 95% specificity. In fact, the 4-second IRP was marginally better than the 3-second IRP, which was marginally better than the 3-second nadir eSleeve. The advantage of the IRP is that the relaxation period quantified need not be contiguous, making it much less vulnerable to crural diaphragm artifact. Having said that, the current version of ManoView software calculates only the 3-second nadir eSleeve so, as a practical matter, this was used in analyzing the 400 patient series. Impaired EGJ relaxation was defined as ≥15mm Hg on the basis of this value exceeding the 95th percentile encountered in 75 control subjects.

Following the analysis of deglutitive EGJ relaxation, a swallow was further categorized by the characteristics of the distal esophageal contraction. This analysis was facilitated by the generation of isobaric contour plots of the distal esophageal segment and EGJ at a 30-mm Hg threshold pressure. Under circumstances of normal deglutitive EGJ relaxation, the 30-mm Hg pressure threshold provided a reliable means of differentiating intrabolus pressure from closure pressure and, thus, the timing of the onset of the peristaltic contraction. Pressurization front velocity (PFV) was calculated from the 30-mm Hg isobaric contour plots by marking the distal temporal margin of the transition zone and the superior margin of the EGJ on the 30-mm Hg isobaric contour and then calculating the slope between the two, expressed in cm/s (Fig. 3). From an analysis of 75 normal subjects, we determined the 95th percentile of normal for mean PFV to be 4.5 cm/s.[5]

A normal PFV depends upon there being both an intact distal peristaltic contraction and normal EGJ relaxation. In instances of peristaltic defects, the 30-mm Hg isobaric contour is discontinuous and in instances of impaired EGJ relaxation, it becomes very rapid because of the elevated intrabolus pressure associated with outflow obstruction. Each swallow was thus characterized as normal (intact 30-mm Hg isobaric contour and a PFV <8 cm/s), failed (complete failure of contraction with no pressure domain above 30mm Hg), hypotensive (≥2 cm defect in the 30-mm Hg isobaric contour), or rapidly conducted (PFV ≥8 cm/s) (Fig. 4). Swallows with a rapid PFV were further characterized on the basis of the distinction between it being caused by impaired EGJ relaxation with compartmentalized esophageal pressurization or a rapidly conducted contraction (spasm) (Fig. 5). The simplest way to differentiate between these two possibilities is to draw a second isobaric contour of higher pressure than the first and clearly exceeding EGJ pressure (50mm Hg in Fig. 5). In instances in which the 2 isobaric contours are nearly parallel, the rapid PFV is attributable to a contraction but if they diverge it is attributable to compartmentalized esophageal pressurization. Alternatively, if the isobaric contour remains vertical even when scaled all the way up to EGJ pressure, this represents panesophageal pressurization, a condition in which the entire esophageal lumen is pressurized between the two sphincters.

Once swallows were characterized by the integrity of deglutitive EGJ relaxation and normality of the PFV, the distal esophageal contraction was characterized for the vigor of contraction using a newly developed measure, the distal contractile integral (DCI). The DCI quantified the length, vigor, and persistence of postdeglutitive pressurization in the distal esophageal segment, expressed as mm Hg s cm[9] (Fig. 6). Using data from the 75 control subjects, a DCI value greater than 5000mm Hg s cm was considered elevated.

ESOPHAGEAL MOTILITY CLASSIFICATION USING PRESSURE TOPOGRAPHY PARAMETERS

After each patient's swallows were analyzed and categorized, their overall motility pattern was classified using a scheme adapted to topographic metrics from conventional manometric criteria[11] as detailed in Table 1. Patients with normal EGJ pressure, normal EGJ relaxation, normal PFV, and a DCI <5000 mm Hg s cm were reported as normal whereas the array of potential abnormalities is detailed below. Note that, in contrast to most conventional classification schemes, there is no category of nonspecific esophageal motility disorders. This is intentional because, as a practical matter, all manometric findings are nonspecific; manometry only describes esophageal function and there is always more than one diagnosis associated with a particular functional pattern.[12,13] Even the most specific pattern, achalasia, is seen as a result of outflow obstruction or achalasia. Hence, in the Chicago Classification, the functional abnormalities encountered are described in specific mechanical terms with the intent that these will then be interpreted within the clinical context of the patient.

Aperistalsis and Peristaltic Dysfunction

Patients with failed peristalsis in all test swallows were classified as aperistalsis. If this was associated with a hypotensive EGJ (<10mm Hg), this constituted a scleroderma pattern. Of 29 patients with aperistalsis (7% of the patient series), 14 exhibited a scleroderma pattern whereas the remaining 15 had normal EGJ pressure. Six of the scleroderma pattern patients were treated patients with achalasia whereas 4 had a confirmed diagnosis of scleroderma and 4 had only GERD symptoms. Patients with aperistalsis and normal EGJ pressure comprised partially treated patients with achalasia (5), postfundoplication patients with dysphagia (3), GERD (3) and undefined dysphagia (4). Patients with failed peristalsis or hypotensive peristalsis in ≥30% but <70% of test swallows were classified as mild peristaltic dysfunction whereas those with ≥70% of swallows with these patterns had severe peristaltic dysfunction. These lesser degrees of peristaltic dysfunction were relatively common, seen in 73 patients (18% of the patient series); severe in 28 patients and mild in 45.

Hypertensive Peristalsis and Rapidly Propagated Pressurization

Patients with a PFV <8 cm/s in >90% of swallows and a DCI greater than 5000mm Hg s cm were considered to have hypertensive peristalsis and this was further stratified on the basis of the magnitude of the DCI and the locus of the hypercontractile segment. Patients with a PFV >8 cm/s in ≥20% of swallows were classified as having rapidly propagated pressurization.

These patients were further classified as spasm or compartmentalized esophageal pressurization.

Thirty-seven patients had normal deglutitive EGJ relaxation and a DCI >5000mm Hg s cm thereby defining hypertensive peristalsis (9% of the patient series). However, there was substantial heterogeneity among this group. LES function was variable with 7 patients also exhibiting a hypertensive LES. One particularly interesting subgroup, defined by a higher threshold DCI (>8000mm Hg s cm), exhibited repetitive high amplitude contractions and was uniformly associated with dysphagia or chest pain (Fig. 7). This spastic nutcracker pattern was found in 12 patients. An additional 6 subjects with a DCI >5000 had a locus of hypertensive peristalsis isolated within only one of the distal esophageal contractile segments that would likely have gone undetected using conventional methods and criteria (Fig. 8). Three of these patients had a segmental focus of hypertensive peristalsis limited to the LES after-contraction and were thus classified as nutcracker LES.

Once the distinction was made between a rapid PFV attributable to compartmentalized esophageal pressurization and that attributable to a rapidly conducted contraction, only 6 patients met criteria for distal esophageal spasm (1.5% of the patient series), making this a very rare diagnosis (Fig. 5).

Achalasia

A diagnosis of achalasia requires both aperistalsis and impaired deglutitive EGJ relaxation. In its most obvious form this occurs in the setting of esophageal dilatation with negligible pressurization within the esophagus (Fig. 9). However, despite there being no peristalsis, there can be substantial pressurization within the esophagus. In fact, a very common pattern encountered is of pan-esophageal pressurization (Fig. 10, left). The other, much less common pattern is of vigorous achalasia in which there is a spastic contraction within the distal esophageal segment (Fig. 10, right). In a series of 73 consecutive patients with achalasia (18% of the patient series), 40 had aperistalsis, 29 had pan-esophageal pressurization, and 4 had vigorous achalasia.

Functional Obstruction

Incomplete deglutitive EGJ relaxation in the setting of preserved peristalsis is categorized as functional obstruction. This has several potential etiologies including mechanical obstruction attributable to fundoplication or paraesophageal hernia, variant achalasia, and some cases of eosinophilic esophagitis.[8] We subdivide cases of functional obstruction into mild and severe on the basis of the magnitude of elevated intrabolus pressure recorded in the distal esophagus. In mild cases the distal esophageal intrabolus pressure ranging from 15 to 30mm Hg whereas in severe cases it exceeded 30mm Hg. Severe cases exhibited a pattern of compartmentalized intraesophageal pressurization and sometimes even panesophageal pressurization as in Figure 11. Of the 37 patients with functional obstruction (11% of the patient series), the etiologies were postfundoplication (15), peptic stricture (5), eosinophilic esophagitis (3), and no defined pathology presumed to be variant achalasia (14).

CONCLUSIONS

Solid-state HRM capable of simultaneously monitoring the entire axial pressure profile from the pharynx to the proximal stomach along with sophisticated topographic plotting algorithms represents an unquestionable evolution in esophageal manometry. Along with this new methodology come challenges and one of those was in devising a scheme to apply HRM to the clinical evaluation of patients in such a way as to take maximal advantage of the technology. This review describes the Chicago Classification of esophageal motility disorders, the result of a systematic analysis of pressure topography patterns in 75 control subjects and 400 consecutive patients. Although it is unlikely that this scheme will be the last word on the subject, the classification described should serve as a foundation for further refinement as new developments arise.

In the process of analyzing these 475 studies, we found 2 major strengths of pressure topography plots compared with conventional manometric recordings: (1) the ability to easily delineate the spatial limits and contractile characteristics of individual contractile segments along the esophagus and (2) the ability to easily distinguish between loci of compartmentalized esophageal pressurization and rapidly propagated esophageal contractions. Making these distinctions was of great utility in identifying distal esophageal spasm, vigorous achalasia, functional obstruction, and subtypes of nutcracker esophagus. Using these restrictive definitions, we found the spastic motility disorders to be quite rare: (1) DES was found in 6 (1.5%) patients, (2) vigorous achalasia in 6 (1.5%), and a newly defined entity, spastic nutcracker, in 6 (1.5%). This in contrast to 69 patients (17.4% of the patient series) being encountered with typical achalasia.

One question that always arises as one contemplates revising a classification scheme is, why bother? In this case, the answer gets to the root of why one performs esophageal manometry in the first place. The optimal clinical use of esophageal manometry is to detect clinically relevant abnormalities of esophageal function: peristaltic defects that may impair bolus transit, spastic disorders that may cause dysphagia and pain, and most importantly, disorders of sphincter relaxation that underlie dysphagia. High resolution manometry with pressure topography analysis facilitates a much more complete understanding of these functional abnormalities and, as evident in Table 1, is much more specific in their identification. Therapeutic interventions are, after all, directed at functional abnormalities and you need to identify them before you can target them.

In summary, we have utilized a large clinical experience of 400 consecutive patients and 75 control subjects to develop a systematic approach to classifying esophageal motility using HRM and pressure topography plots. The resultant scheme (Table 1) is consistent with conventional classifications with the caveats that: (1) hypercontractile conditions are more specifically defined, (2) distinctions are made between “simultaneous contractions” attributable to rapidly propagated contractions and those attributable to compartmentalized esophageal pressurization, and (3) there is no “nonspecific esophageal motor disorder” classification. Ultimately, further clinical experience will be the judge, but it is our expectation that pressure topography analysis of HRM data, along with its well-defined functional implications, will prove valuable in the clinical management of esophageal motility disorders.

Acknowledgments

Supported by R01 DC00646 (PJK) from the Public Health Service.

References

1. Pandolfino JE, Zhang Q, Ghosh SK, et al. tLESRs and reflux: mechanistic analysis using concurrent fluoroscopy and high-resolution manometry. Gastroenterology 2006;131:1725–1733. [PubMed: 17087957]
2. Clouse RE, Staiano A. Topography of the esophageal peristaltic pressure wave. Am J Physiol 1991;261:G677–G684. [PubMed: 1928353]
3. Clouse RE, Staiano A, Alrakawi A, et al. Application of topographical methods to clinical esophageal manometry. Am J Gastroenterol 2000;95:2720–2730. [PubMed: 11051340]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

4. Pandolfino JE, Ghosh SK, Zhang Q, et al. Quantifying EGJ morphology and relaxation with high-resolution manometry: a study of 75 asymptomatic volunteers. Am J Physiol (Gastrointest Liver Physiol) 2006;290:G1033–G1040. [PubMed: 16455788]
5. Ghosh SK, Pandolfino JE, Zhang Q, et al. Quantifying esophageal peristalsis with high-resolution manometry: a study of 75 asymptomatic volunteers. Am J Physiol (Gastrointest Liver Physiol) 2006;290:G988–G997. [PubMed: 16410365]
6. Ghosh SK, Pandolfino JE, Zhang Q, et al. Deglutitive upper esophageal sphincter relaxation: a study of 75 volunteer subjects using solid-state high-resolution manometry. Am J Physiol (Gastrointest Liver Physiol) 2006;291:G525–G531. [PubMed: 16645162]
7. Ghosh SK, Pandolfino JE, Rice J, et al. Impaired deglutitive EGJ relaxation in clinical esophageal manometry: a quantitiative analysis of 400 patients and 75 controls. Am J Physiol 2007;293:G878–G885.
8. Pandolfino JE, Ghosh SK, Rice J, et al. Classifying esophageal motility by pressure topography characteristics: a study of 400 patients and 75 controls. Am J Gastroenterol. 2007 [Epub ahead of print, Sept 27].
9. Pandolfino JE, El-Serag HB, Zhang Q, et al. Obesity: a challenge to esophagogastric junction integrity. Gastroenterology 2006;130:639–649. [PubMed: 16530504]
10. Massey BT, Dodds WJ, Hogan WJ, et al. Abnormal esophageal motility. An analysis of concurrent radiographic and manometric findings. Gastroenterology 1991;101:344–354. [PubMed: 2065909]
11. Spechler SJ, Castell DO. Classification of oesophageal motility abnormalities. Gut 2001;49:145–151. [PubMed: 11413123]
12. Kahrilas PJ, Clouse RE, Hogan WJ. American Gastroenterological Association technical review on the clinical use of esophageal manometry. Gastroenterology 1994;107:1865–1884. [PubMed: 7958705]
13. Pandolfino JE, Kahrilas PJ. AGA technical review on the clinical use of esophageal manometry. Gastroenterology 2005;128:209–224. [PubMed: 15633138]

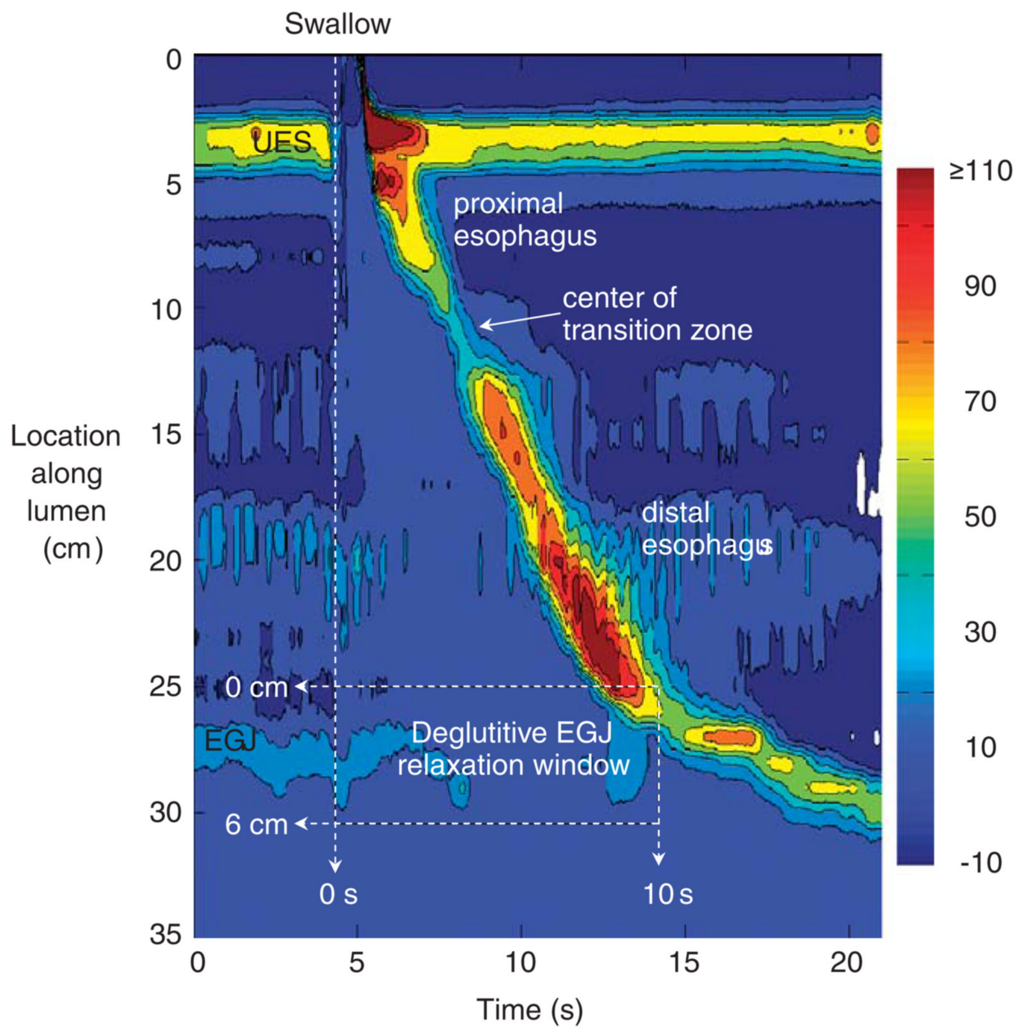


FIGURE 1.
Typical pressure topography of a swallow spanning the entire esophagus from the pharynx (locations 0 to 2 cm) to stomach (locations 32 to 35 cm) of a normal subject with normal peristalsis and normal EGJ relaxation. Note that the transition zone demarcating the end of the proximal esophageal segment (striated muscle) and the beginning of the distal esophageal segment (smooth muscle) is readily identified as the pressure minimum between the sphincters. The classification scheme described herein encompasses the distal esophagus and the EGJ. The onset of the deglutitive relaxation window is defined by the onset of upper sphincter relaxation whereas the offset is 10 seconds later. The spatial domain within which EGJ relaxation is assessed is user defined, spanning at least 6 cm, depending on the extent of EGJ shortening after the swallow.

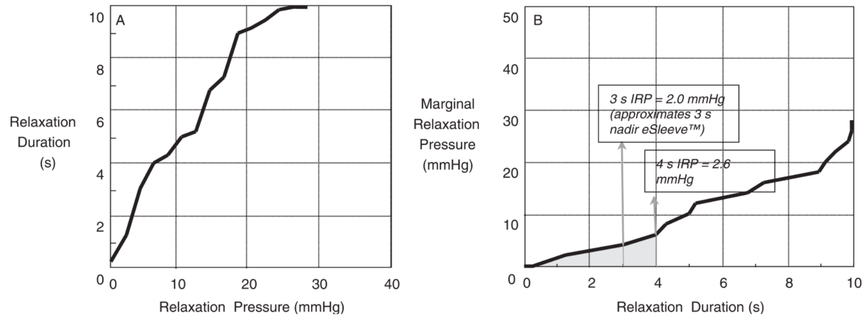


FIGURE 2.
Methodology for calculating deglutitive EGJ relaxation. A, The cumulative duration of EGJ relaxation within the relaxation window detailed in Figure 1 as the threshold relaxation pressure cutoff was increased; for example, for a relaxation pressure cutoff of 10mm Hg, the period of relaxation was about 5 seconds. B, X-y transposition of Panel A illustrating the marginal relaxation pressure as the specified duration of relaxation is increased from 0 to 10 seconds. This plot was used to calculate the 3 and 4 seconds IRP values (indicated), which are the integrals of the curve (shaded) divided by 3 or 4 seconds, respectively. The 3-second nadir eSleeve measure of deglutitive relaxation is quantitatively similar to both the 3 and 4 seconds IRP values but has the requirement that the relaxation period analyzed be contiguous leaving it subject to crural diaphragm artifact in individuals with rapid respiration.

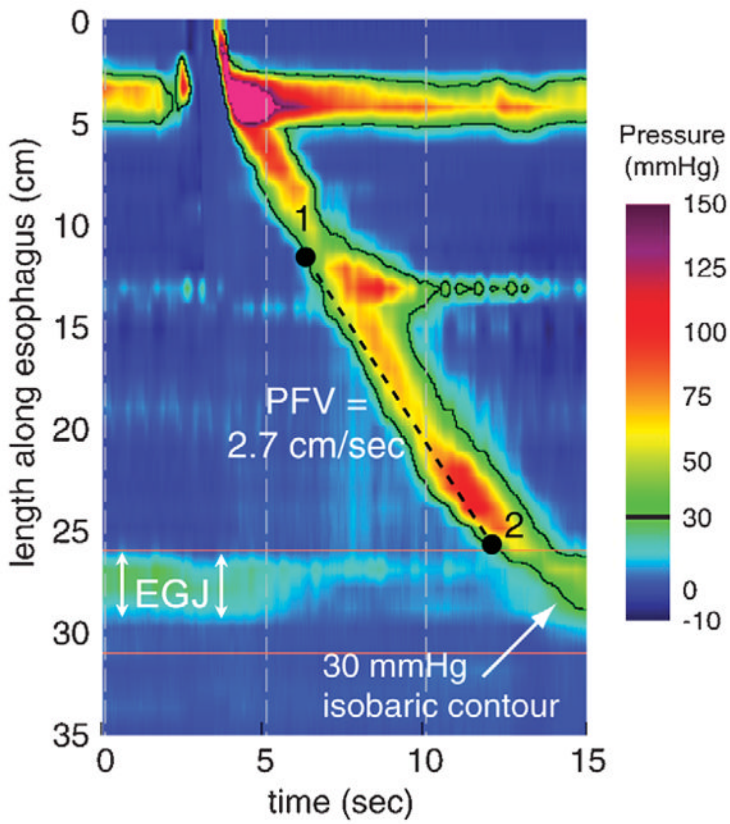


FIGURE 3.
Derivation of the pressurization front velocity (PFV) from 30-mm Hg isobaric contour plots. The heavy black line delineates the pressure domain ≥30mm Hg. To calculate the PFV, the distal temporal margin of the transition zone (point 1) and the proximal margin of the EGJ on the 30-mm Hg isobaric contour (point 2) were localized by hand. The slope of the line connecting the 2 points was the PFV, expressed in cm/s.

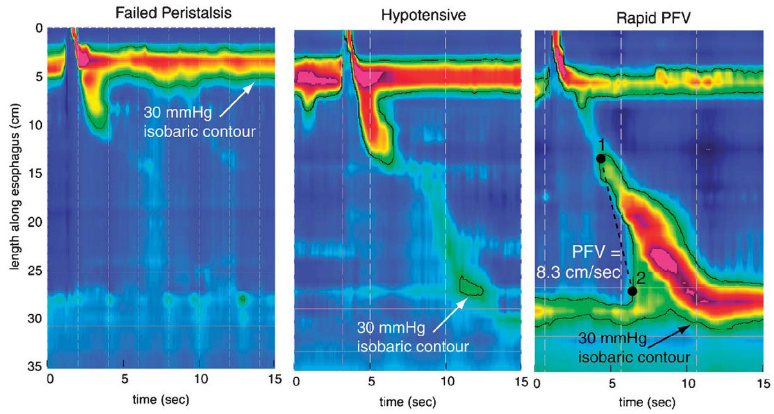
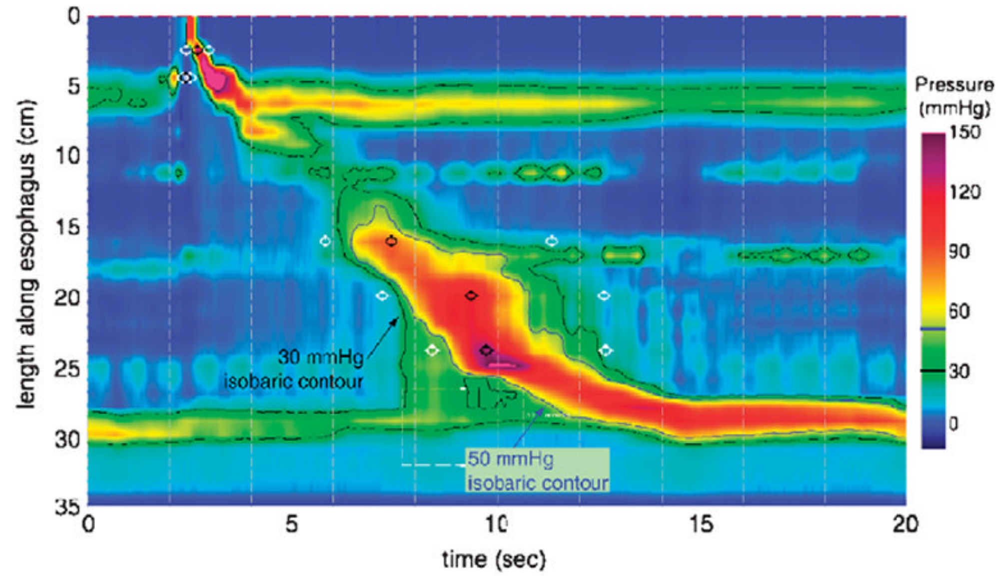


FIGURE 4.

Three circumstances of abnormal PFV: failed peristalsis, hypotensive peristalsis, and a rapid PFV. In the instance of failed peristalsis, no pressure domain exists within the distal esophageal segment greater than the 30-mm Hg isobaric contour whereas with the hypotensive contraction the 30-mm Hg isobaric contour is incomplete (compare to Fig. 2). Note that the example of the rapid PFV is attributable to compartmentalized esophageal pressurization in the setting of obviously impaired deglutitive EGJ relaxation (the EGJ pressure is never <30mm Hg).



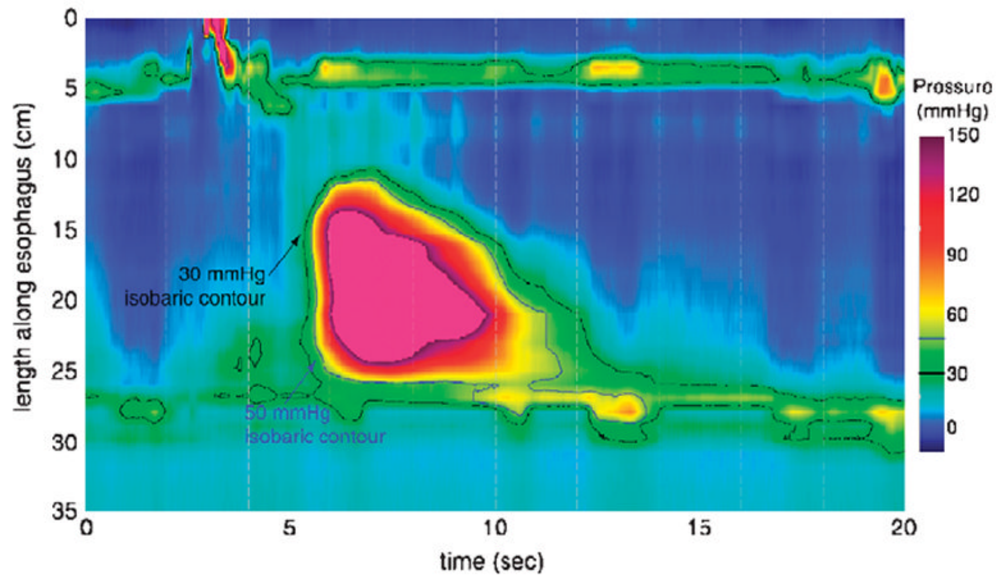


FIGURE 5.

Differentiating a rapid PFV attributable to compartmentalized esophageal pressurization (top) from a rapidly propagated contraction (bottom). The upper panel illustrates a swallow with functional obstruction at the EGJ. Note that the 30-mm Hg isobaric contour domain (black) deviates quickly from the propagating contractile wavefront highlighted by the 50-mm Hg isobaric contour line. The PFV of the 30-mm Hg isobaric contour domain is 8.2 cm/s and would fit criteria for a rapid contraction. However, the pressurization front velocity of the 50-mm Hg isobaric contour would be normal. In contrast, the lower panel represents a swallow with rapid PFV attributable to spasm. The 30 and 50-mm Hg isobaric contours parallel each other indicating that no compartmentalized esophageal pressurization has occurred. The entire distal esophagus is contracting simultaneously.

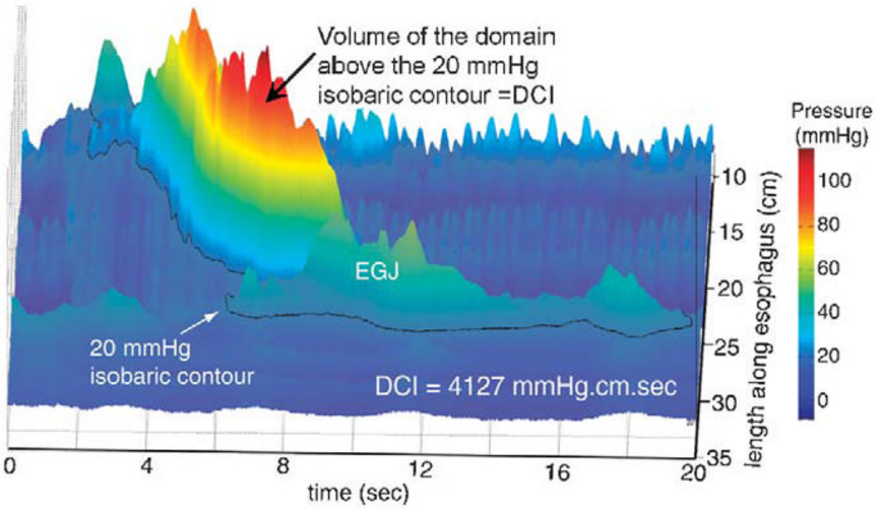


FIGURE 6.
Derivation of the DCI. Conceptually, if the isobaric contour plot of distal esophageal contraction is envisioned as a 3-dimensional solid, the footprint of the solid is time multiplied by length of the distal esophageal segment (cm) and the height of the solid is pressure. The distal contractile integral is the volume of that solid spanning from 20mm Hg at the base to its peak, expressed as mm Hg s cm.

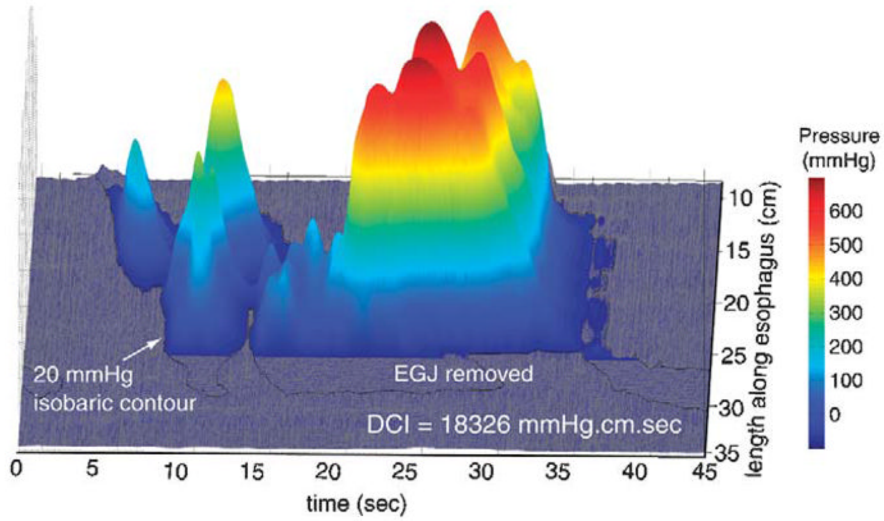


FIGURE 7.

Distal contractile integral of a patient with an extreme example of spastic nutcracker. Note the PFV is normal (3.0 cm/s). The contraction has a spastic component that occurs after the wavefront propagates to the EGJ. Typical of patients with a PFV>8000mm Hg s cm, this patient had chest pain and dysphagia.

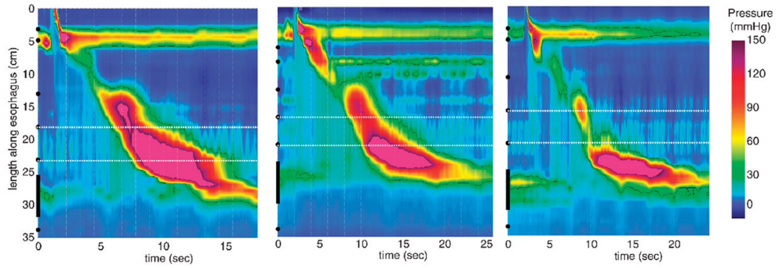


FIGURE 8.
Heterogeneity of hypertensive peristalsis. The left panel represents nutcracker esophagus defined a distal contractile integral greater than 5000mm Hg s cm and a normal PFV. The dashed lines represent conventional measurement points of 3 and 8 cm above the LES and this patient would fulfill conventional criteria for nutcracker esophagus. The center panel represents another patient with a distal contractile integral greater than 5000mm Hg s cm, however, this patient would have been missed by conventional measurement as the hypercontractile focus is limited to a short segment in the distal esophagus. The right panel illustrates a patient with nutcracker LES; the hypertensive contraction is limited to the sphincter segment after contraction.

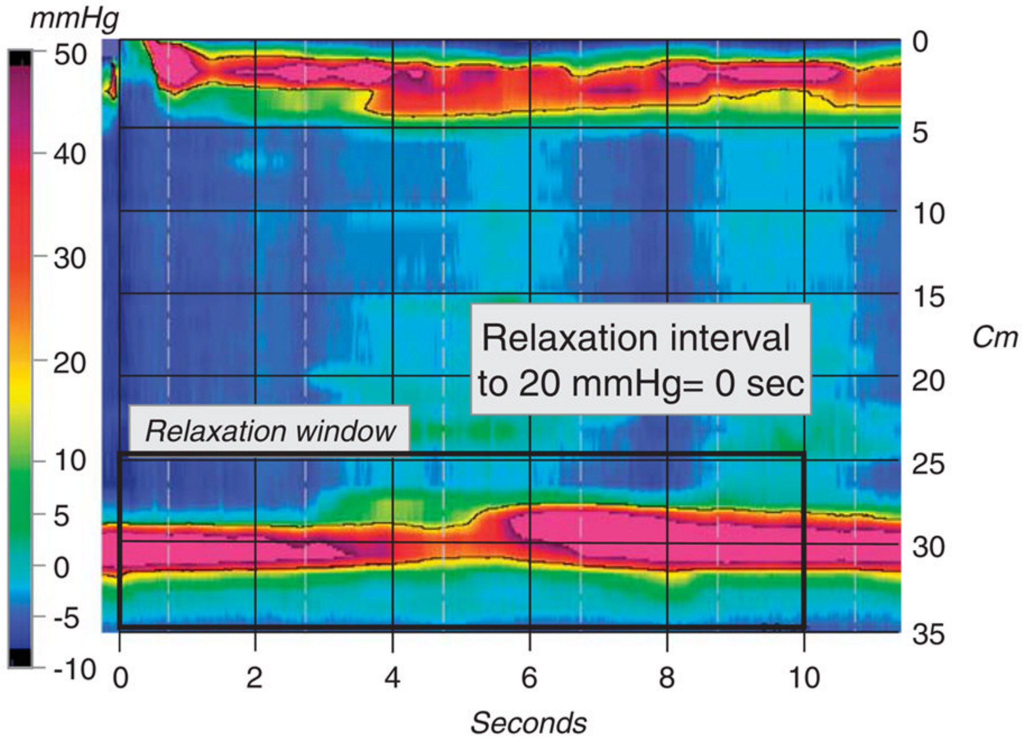


FIGURE 9.
Typical advanced achalasia with a nonrelaxing LES and minimal pressure activity within the dilated esophageal lumen.

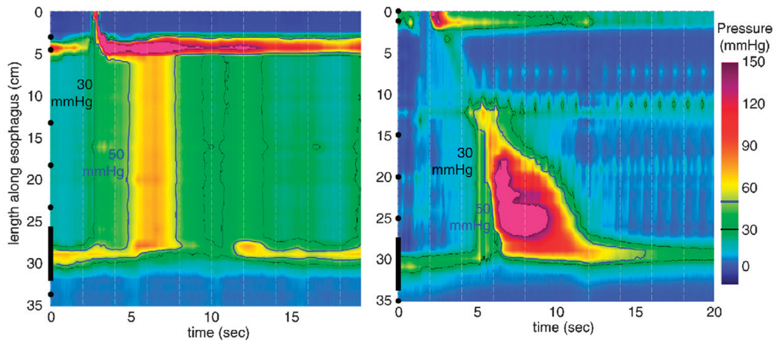


FIGURE 10.
The distinction between achalasia associated with pan-esophageal pressurization (left) and vigorous achalasia (right). In each case, the black line indicates the 30-mm Hg isobaric pressure contour and both examples have an abnormal eSleeve 3-second nadir LES relaxation measurement.

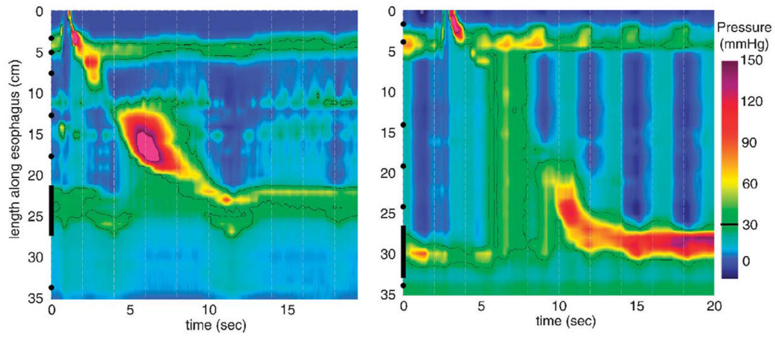


FIGURE 11.
Pressure topography plots illustrating mild functional obstruction in a postfundoplication patient (left) and severe functional obstruction in a patient with eosinophilic esophagitis (right). Because the intrabolus pressure never exceeds 30mm Hg, the PFV is normal in the patient with mild functional obstruction. However, in the patient with severe functional obstruction, the PFV is >8 cm/s and proceeded by a period of pan-esophageal pressurization.

TABLE 1

Esophageal Motility Classification on the Basis of Pressure Topography Criteria

The Chicago Classification of Esophageal Motility

Normal

- PFV <8 cm/s in >90% of swallows
- DCI <5000mm Hgs cm
- Normal EGJ pressure (10–35mm Hg) and deglutitive relaxation (eSleeve 3-s nadir <15mm Hg)

Peristaltic dysfunction

- Mild: ≥3 and <7 swallows with either failed peristalsis or a ≥2-cm defect in the 30-mm Hg isobaric contour of the distal esophageal segment
- Severe: ≥7 swallows with either failed peristalsis or a ≥2-cm defect in the 30mm Hg isobaric contour of the distal esophageal segment

Aperistalsis

- No continuous pressure domain above an isobaric contour of 30mm Hg in the distal esophageal segment in any swallow
  - Scleroderma pattern: no continuous pressure domain above an isobaric contour of 30mm Hg in the distal esophageal segment in any swallow and a mean LES pressure <10mm Hg

Hypertensive peristalsis

- PFV <8 cm/s in >90% of swallows
- Mean DCI: >5000mm Hgs cm
  - Nutcracker: mean DCI>5000 and <8000mm Hgs cm
  - Segmental Nutcracker: mean DCI >5000 with only one segmental focus of hypertensive contraction (>180mm Hg)
  - Spastic Nutcracker: mean DCI > 8000mm Hgs cm
  - Nutcracker LES: mean DCI >5000mm Hgs cm with the focus of hypertensive contraction (>180mm Hg) limited to the LES after-contraction.

Rapidly propagated pressurization

- PFV>8 cm/s in ≥20% of swallows
  - Spasm (increased PFV attributable to rapid contractile wavefront)
  - Compartmentalized pressurization (increased PFV attributable to distal compartmentalized esophageal pressurization)

Abnormal LES tone (end expiratory)

- Hypotensive: mean <10mm Hg with normal peristaltic function
- Hypertensive: mean >35mm Hg with normal peristaltic function and EGJ relaxation

Achalasia

- Impaired deglutitive EGJ relaxation
- Aperistalsis
  - Classic: aperistalsis or pan-esophageal pressurization with no identifiable segmental contractile activity with all swallows
  - Vigorous: with distal spasm

Functional obstruction

- Impaired deglutitive EGJ relaxation
  - Mild: PFV <8 cm/s in >90% of swallows with a mild elevation (15 to 30mm Hg) of distal esophageal pressurization
  - Severe: PFV >8 cm/s in ≥20% of swallows with compartmentalized pressurization