# High-Resolution Manometry and Esophageal Pressure Topography

## Filling the Gaps of Convention Manometry

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**KEYWORDS**

• Esophageal motility disorders • High-resolution manometry • Achalasia
• Distal esophageal spasm

**KEY POINTS**

- Diagnostic schemes for conventional manometry and esophageal pressure topography (EPT) rely on measurements of key variables and descriptions of patterns of contractile activity. However, the enhanced assessment of esophageal motility and sphincter function available with EPT has led to the further characterization of clinically relevant phenotypes.
- Differentiation of achalasia into subtypes provides a method to predict the response to treatment.
- A diagnosis of diffuse esophageal spasm represents a unique clinical phenotype when defined by premature esophageal contraction (measured via distal latency) instead of when defined by rapid contraction (measured by contractile front velocity and/or wave progression) alone.
- Defining hypercontractile esophagus with a single swallow with a significantly elevated distal contractile integral, as opposed to using a mean value more than a predetermined 95th percentile, may define a more specific clinical syndrome characterized by chest pain and/or dysphagia.
- EPT correlates of the conventional manometric diagnosis of ineffective esophageal motility include weak and frequent-failed peristalsis; however, the clinical significance of these diagnoses is not completely understood.

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## INTRODUCTION

In 2001, based on a review of the literature to date, Spechler and Castell[1] proposed a classification scheme for esophageal motility disorders incorporating defined conventional manometry (CM) criteria. This description was the state-of-the-art description of manometry at the time. However, the investigators recognized that the clinical significance of any observed manometric findings may be limited because the abnormalities were often reported to occur with poor correlation to symptoms, and therapeutic corrections of manometric findings often did not lead to improvement in symptoms.[2]

A few years after that review, high-resolution manometry (HRM) and esophageal pressure topography (EPT) started to appear on the scene, both in research and clinical practice. HRM is comprised of multiple, closely spaced pressure sensors (usually 1 cm apart) that record pressure without significant gaps in data along the length of the esophagus. This data can be modified using interpolation to generate EPT plots that are color coded, spatiotemporal representations of pressure recordings in the esophagus (Clouse plots). This technology lends itself to an objective assessment of EPT metrics that have been integrated into a new classification scheme for esophageal motility disorders, referred to as the Chicago classification scheme.[3] As clinical and research experience grows with HRM, the Chicago classification scheme has been intermittently updated in an attempt to improve its representation of clinically relevant phenotypes.[4–7] The goal of this review is to compare conventional and HRM classification schemes for esophageal motility disorders and to illustrate how these new clinical phenotypes on EPT have evolved from previous definitions used by Spechler and Castell for CM.

## METHODOLOGY: CM AND HRM

The procedure for both types of manometry begins with the placement of the manometry catheter transnasally until the distal pressure sensors cross the esophagogastric junction (EGJ) and enter the stomach. The comparative measurements made with CM and HRM are displayed in **Table 1**.

In CM, a pull-through technique is used to determine the position of the lower esophageal sphincter (LES) pressure by identifying the pressure inversion point and a high-pressure zone. The pressure sensor is then left positioned in the LES, and the basal pressure is recorded over at least 2 minutes with minimal swallowing. Once the baseline recording is complete, LES relaxation is measured during at least 5 wet (5 mL water) swallows with the pressure sensor maintained at the position where the middle of the LES high-pressure zone was recorded. Peristaltic function is typically assessed with pressure sensors spaced anywhere from 3 to 5 cm apart, with a repositioning of the pressure sensors into the body or by simultaneous pressure recording at the LES using a sleeve or single sensor.

In HRM, the distal end of the catheter is passed into the gastric compartment below the LES and hiatal canal, and no pull through is required because the catheter can provide recording from the stomach through the esophagus into the oropharynx. During an HRM study, EPT plots, also known as Clouse plots, are generated by computer software during 10 wet (5 mL water) swallows, and there is no need to perform different steps in the evaluation because all variables can be assessed during the single swallows.[8,9]

Analysis of an EPT study is performed using a stepwise approach that focuses on an algorithm-based scheme that first defines patients based on EGJ relaxation pressures

**Table 1**
**Comparison of CM and HRM metrics**

| Esophageal Motility Characteristic | CM Measurement | HRM Measurement |
|---|---|---|
| LES relaxation | | |
| | LES relaxation with swallow | IRP |
| Normal[a] | Complete (<8 mm Hg more than gastric pressure) | <15 mm Hg |
| Peristaltic propagation | | |
| | Wave progression between pressure sensors 8 and 3 cm above the LES | CFV |
| Normal[a] | 2–8 cm/s (UES to LES) | <9 cm/s |
| | *(no corresponding CM metric)* | DL |
| Normal[a] | | ≥4.5 s |
| Contractile vigor | | |
| | Mean distal wave maximum amplitude of pressure sensors 8 and 3 cm above the LES | DCI |
| Normal[a] | 30–180 mm Hg | 450–5000 mmHg-s-cm |

*Abbreviations:* CFV, contractile front velocity; DCI, distal contractile integral; DL, distal latency; IRP, integrated relaxation pressure; LES, lower esophageal sphincter; UES, upper esophageal sphincter.
[a] Normal values as stated in Refs.[1,6]

and subsequently uses individual swallow patterns defined by EPT metrics to further subclassify patients into specific categories.

- Step 1: Assessment of EGJ pressure morphology at baseline
  - The first step of the analysis process focuses on describing the pressure morphology of the EGJ to determine whether a hiatus hernia is present and where the pressure inversion point is located because this can have dramatic effects on the measures of EGJ function. The baseline end-expiratory pressure and inspiratory augmentation are recorded to assess the integrity of the crural diaphragm as an extrinsic sphincter.
- Step 2: Assessing EGJ relaxation and bolus pressure dynamics through the EGJ
  - Patients are defined as having normal or abnormal EGJ relaxation using the integrated relaxation pressure (IRP). The IRP is the lowest mean EGJ pressure for 4 contiguous or noncontiguous seconds during the deglutitive period. As demonstrated in **Fig. 1**, the IRP has replaced the conventional measures of nadir or end-expiratory LES relaxation pressure on CM because EPT evaluation made it quite clear that the pressure measured through the EGJ during swallowing was heavily reliant on intrabolus pressure and was not a pure measure of LES relaxation.[10,11]
- Step 3: Assess integrity of the peristaltic wave
  - Once the IRP is measured, esophageal peristaltic integrity is characterized to determine if the peristaltic activity is intact, failed, or associated with small (2–5 cm) or large (>5 cm) peristaltic breaks in the 20-mm Hg isobaric contour. This step is performed before any other measurements are made because the subsequent measurements depend on the presence of intact or preserved peristaltic integrity in the distal esophagus. This metric is similar to using

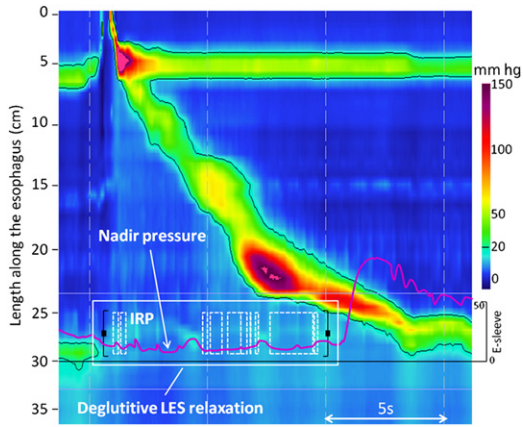


**Fig. 1.** Assessment of EGJ relaxation. Nadir LES pressure (CM line tracing, *purple*) and IRP (*dotted white boxes* indicating lowest LES pressure segments over 4 noncontiguous seconds) are demonstrated in a normal swallow. IRP 4.8 mm Hg. Nadir LES pressure 0.3 mm Hg more than gastric pressure. The nadir pressure is likely a measurement of intragastric pressure.

a 30-mm Hg threshold at 3 and 8 cm above the proximal border of the LES to define effective swallows.[12] However, the isobaric contour tool provides a more complete assessment of the swallow as demonstrated in **Fig. 2**.

- Step 4: Determine the location of the contractile deceleration point (CDP)
  - The CDP is defined as the inflection point along the contractile wavefront defined by the 30-mm Hg isobaric contour tool where the greatest deceleration occurs and the function of the esophagus converts from a stripping wave to a compartmentalized ampulla to promote emptying of the remaining bolus (**Fig. 3**). This landmark is in close proximity to the proximal border of the LES

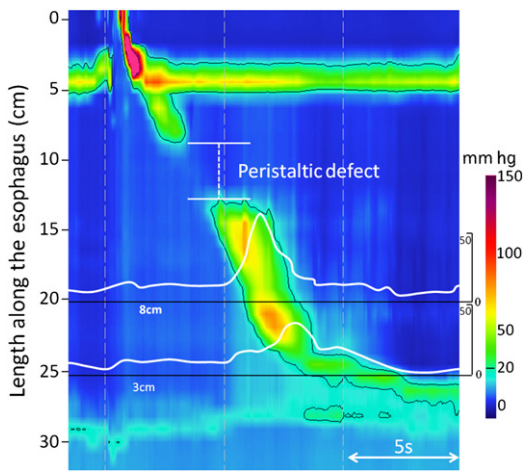


**Fig. 2.** Peristaltic integrity. A Clouse plot of a swallow with a large (5.1 cm axial length) peristaltic defect in the 20-mm Hg isobaric contour is displayed. CM line tracings at 3 and 8 cm would not normally detect this defect in the transition zone. Black lines indicate the CM recording sites with their position from the LES (eg, 3 cm, as labeled).

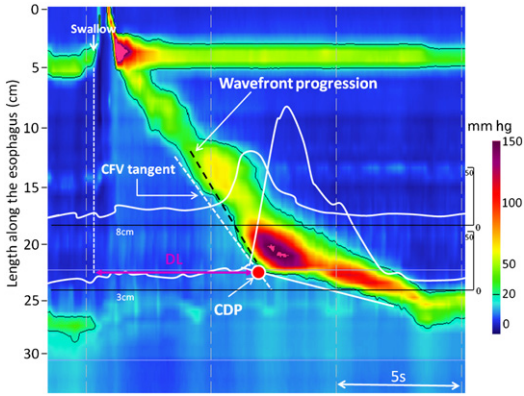


**Fig. 3.** Propagation of peristalsis. The EPT metrics of CDP, distal latency (DL), contractile front velocity (CFV), and the CM wavefront progression are displayed on a normal swallow. The CDP (*red circle*) is located at the intersection of the CFV tangent (*white dashed line*) and the velocity tangent of the terminal segment of esophageal peristalsis (*solid white line*), which correlates with emptying of the esophageal ampulla. The DL (*purple arrow*) is defined as the time from the initiation of the swallow to the CDP and measures 7 seconds in the swallow. The wavefront progression (*black dashed line*) is determined from CM line tracings (measuring 5.0 cm/s) and is comparable to the CFV (3.4 cm/s) in EPT.

during maximal shortening and is usually associated with maximal concurrent axial contraction of the esophageal body.[13] The CDP should be localized within the third contractile segment defined by Clouse, and there is no method or measure on CM that localizes the CDP.[8]

- Step 5: Assess propagation
  - Propagation and timing of peristalsis is defined by assessing the distal latency to determine whether the swallow is premature and possibly associated with impaired inhibitory function of the esophageal body. It is defined as the interval between upper esophageal relaxation and the CDP, as demonstrated in **Fig. 3**. There is no correlate to this metric in CM.
  - Velocity of the stripping wave is determined by an assessment of the contractile front velocity (CFV). It is defined as the slope of the tangent approximating the 30-mm Hg isobaric contour between the proximal pressure trough and the contractile deceleration point. This measurement is akin to the measurement of velocity using the pressure sensor located 3 and 8 cm above the proximal aspect of the LES on CM. It is interesting that the 3-cm point used on CM closely approximates the CDP; thus, this measure has good correlation with CFV.
- Step 6: Measure contractile vigor
  - Contractile vigor has been revised to objectively measure all of the contractile activity within the domain of the distal smooth muscle esophagus below the transition zone. The transition zone is typically localized approximately 6 cm below the lower border of the Upper esophageal sphincter (UES) and represents the first pressure trough between segments 1 and 2 on the Clouse plot. The metric used to quantify the contractile activity between the transition zone and proximal aspect of the EGJ is termed the *distal contractile integral* (DCI), and it uses the space time domain of the second and third contractile segments to provide a single number that quantifies contractile vigor (**Fig. 4**). The DCI is

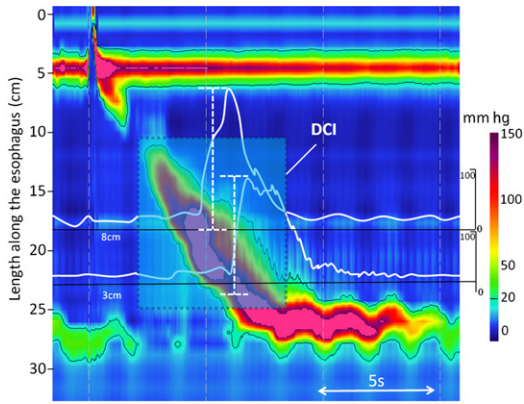


**Fig. 4.** Contractile vigor. The DCI is the software-generated sum of the esophageal body contractile activity from the transition zone to the distal pressure trough (area within the transparent box). CM assessment of contractile vigor is depicted by the mean of the peak wave amplitudes at 3 and 8 cm from the LES (*white dashed lines*). In the hypertensive swallow, the DCI is 7195 mmHg-s-cm and the mean peak amplitude is 214 mm Hg.

used in place of measuring the mean value of the highest wave amplitude at 3 and 8 cm above the proximal aspect of the LES on CM. The EPT plots also allow a qualitative assessment of the contraction that helps define focal contractile abnormalities and disorders associated with LES after contraction.

- Step 7: Determine whether abnormal pressure patterns are present
  - Abnormal intrabolus pressure is a sign of abnormal mechanics of bolus transit related to either an outflow obstruction in the distal esophagus/EGJ or a poorly compliant esophageal wall. It is measured using the isobaric contour tool that is referenced to atmospheric pressure to identify pressurization patterns. These patterns can be compartmentalized between a propagating peristaltic wavefront and the EGJ or between the 2 sphincters (panesophageal pressurization). There is no correlate for this measure on CM; however, astute clinicians can assess the initial ramp pressure on tracings (**Fig. 5**) or identify isobaric pressure patterns on tracings (**Fig. 6**B).

## EPT CORRELATES OF CM DEFINITIONS

### *Disorders with Abnormal LES Relaxation*

Abnormal LES relaxation is the hallmark of achalasia, the best defined of the esophageal motility disorders and the one with the most effective therapies.[1,14] Classically, achalasia also demonstrates a lack of esophageal peristalsis on manometry, although other manometric findings and subclassifications have been described and proposed.

#### *Conventional criteria*

The proposed criteria for a diagnosis of classic achalasia by CM criteria included incomplete relaxation of the LES (defined as a mean LES relaxation pressure during swallowing more than 8 mm Hg above gastric pressure) and aperistalsis of the esophageal body (either simultaneous contractions with amplitudes less than 40 mm Hg or no apparent esophageal contraction).[1] *Atypical disorders of LES relaxation* are also described that exclude a diagnosis of classic achalasia with some preserved peristalsis and/or esophageal contractions with amplitudes greater than 40 mm Hg, the latter situation often being referred to as *vigorous achalasia*.

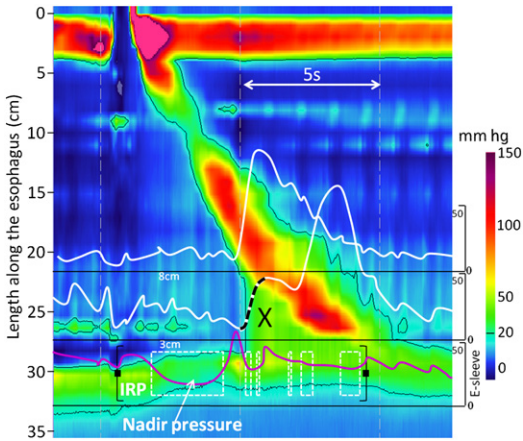


**Fig. 5.** Compartmentalized pressure. Compartmentalized intrabolus pressure (X) visualized on EPT can also be implicated via assessment of ramp pressure (*black dashed* portion of line tracing at 3 cm from the LES) on CM line tracings. This Clouse plot is an example of a swallow characteristic of EGJ outflow obstruction with abnormal EGJ relaxation (IRP 25.6 mm Hg; nadir LES pressure 28.9) and evidence of intact peristalsis.

### *Achalasia phenotypes*

The classification of achalasia has evolved with the updated revisions of the Chicago classification to reflect different achalasia subtypes that have demonstrated varying symptom profiles and responses to different treatment modalities. The achalasia subtypes are all associated with abnormal EGJ relaxation and are categorized based on the pattern of esophageal body contraction and pressurization: type I, absent peristalsis; type II, achalasia with panesophageal pressurization in 20% or more of swallows; and type III, spastic achalasia (no normal peristalsis and premature contractions in 20% or more of swallows).[6,7] Representative Clouse plots with overlying CM line tracings are displayed in **Fig. 6**. This subclassification of achalasia distinguishes separate clinical phenotypes that are helpful in predicting the response to therapy, and this scheme is supported by 3 separate retrospective studies.[15–17]

The authors' initial study analyzed 99 patients with newly diagnosed achalasia with EPT who underwent balloon dilation, Heller myotomy, and/or Botox injection.[15] Another study analyzed 246 patients with achalasia, 230 with CM, and 16 with EPT, and followed patients after undergoing Heller-Dor myotomy.[16] A third study analyzed 51 patients with EPT, 45 of which underwent pneumatic dilation.[17] In each study, pretreatment symptom assessment suggested that chest pain may be more common in patients with type III (spastic) achalasia. The response to treatment was consistent across all 3 studies, with type II patients having the best and type III patients having the worst response to treatment. The study assessing the response to dilation, myotomy, and/or Botox even suggested that type I patients may have a better response to myotomy (compared with dilation or Botox injection) as the initial treatment.[15] Although prospective treatment trials are needed for further evaluation, these initial studies suggest that achalasia subtypes represent unique clinical phenotypes and may have predictive benefits in treatment planning for patients with achalasia.

### *EGJ outflow obstruction*

EPT analysis also demonstrates a population of patients with abnormal EGJ relaxation with remaining peristaltic activity that fails to meet criteria for a diagnosis of achalasia,

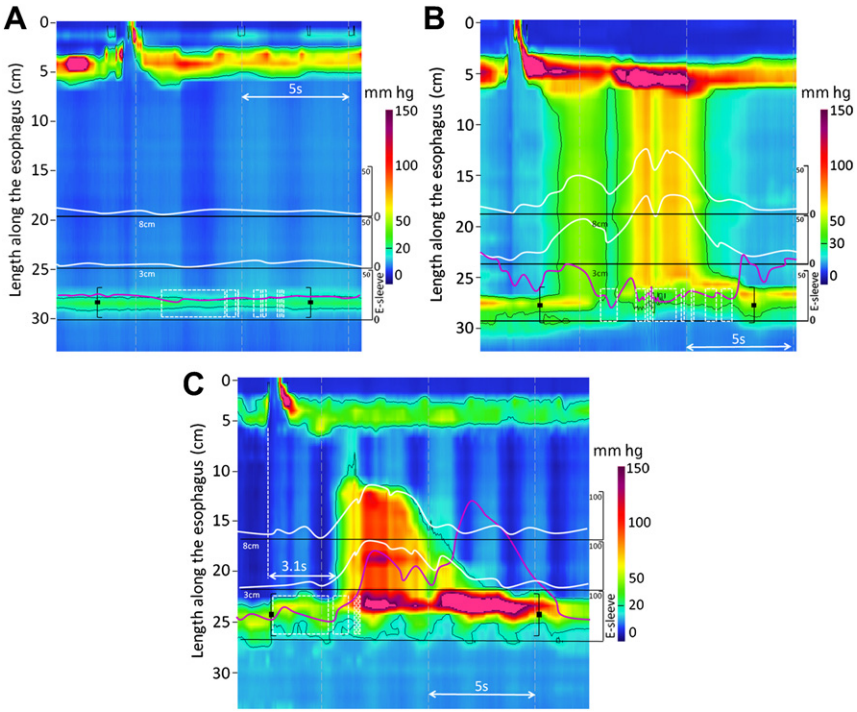


**Fig. 6.** Achalasia phenotypes. Once abnormal EGJ relaxation is determined ([*A*] IRP 17.6 mm Hg, nadir LES pressure 23.3 mm Hg; [*B*] IRP 26.5 mm Hg, nadir LES pressure 25.3 mm Hg; [*C*] IRP 46.5 mm Hg, nadir LES pressure 42.3 mm Hg), these disorders can be further classified based on their esophageal body contractility patterns. Type I (*A*) classic achalasia is characterized by absent peristalsis. Type II (*B*) achalasia with esophageal compartmentalization demonstrates pressurization spanning the length of the esophagus without intact peristalsis. Panesophageal pressurization can be identified with CM by noting the isobaric pressure tracings, as seen here. Type III (*C*), spastic achalasia, can demonstrate fragments of distal peristalsis and/or premature esophageal contractions, as demonstrated here with a shorter-than-normal latency (*white arrow*) of 3.1 seconds. Elevated wave amplitudes are also present on the CM line tracings, which has previously prompted labeling as vigorous achalasia.

similar to those with *atypical disorders of LES relaxation* in the conventional criteria. This pattern was termed *functional obstruction* in the early Chicago classification schemes.[4–6] However, further analysis of patients with these EPT findings displayed similar characteristics, including an elevated intrabolus pressure, as patients with a known mechanical obstruction, such as postfundoplication. Thus, this entity is now categorized as EGJ outflow obstruction.[7,18] A representative swallow is displayed in **Fig. 5**. Again, these manometric findings seem to reflect a clinical phenotype that frequently presents with dysphagia and/or chest pain, may respond poorly to balloon dilation or Botox injection overall, and may have a favorable outcome in response to treatment with myotomy.[19] Although additional study is needed to further characterize this group, it is possible that patients with this manometric profile may represent undetected inflammatory or infiltrating malignant disorders or may be a variant or earlier form of achalasia. Given this heterogeneous differential diagnosis, it may be helpful if these findings are correlated with either endoscopic ultrasound or other imaging modalities.

### *Diffuse Esophageal Spasm*

Diffuse esophageal spasm (DES) is often implicated as the cause of noncardiac chest pain or dysphagia; however, the manometric criteria for the diagnosis of DES has frequently been questioned. Although differences in the requirements for repetitive, spontaneous, high-amplitude, or rapid contractions have varied in the previous literature, simultaneous esophageal contractions are nearly universally described as manometric criteria for DES and have been proposed to be the essential criteria in the diagnosis of DES.

#### *Conventional criteria*

Spechler and Castel[1] proposed that a diagnosis of DES should require (1) simultaneous contractions with more than 10% of wet swallows and (2) a mean simultaneous contraction amplitude of more than 30 mm Hg.[1] They reported that other common, but not required, features may include spontaneous contractions, repetitive contractions, multiple peaked contractions, and intermittent normal peristalsis. They also stated that if the LES pressure is abnormal, the disorder is better classified as an atypical disorder of LES relaxation.

#### *Distal esophageal spasm*

*Simultaneous contractions* were interpreted in the early versions of the Chicago classification as rapid contractions, which were defined by a CFV of more than 8 mm/s.[3,5,6] The CFV, however, has been demonstrated to be susceptible to regional variability in contractile velocity within the swallow and, thus, is a nonspecific finding of unknown significance.[20] The distal latency (DL), however, seems to be a more reliable measure of premature contractions that likely represents a clinical phenotype defined by dysphagia and chest pain.

A study that analyzed 1070 consecutive interpretable EPT studies found 24 patients that exhibited premature contraction (defined by DL <4.5 s) and 67 patients that were found to have rapid contractions alone (defined as CFV >9 m/s) but normal DL.[20] A review of medical records revealed that all 24 of the patients with premature contractions had a dominant symptom of dysphagia or chest pain and were diagnosed and managed as DES (6 patients) or spastic achalasia (18 patients). The 67 patients with rapid contractions with normal latency had a more heterogeneous dominant symptom (56% dysphagia, 34% gastrointestinal reflux disease [GERD], and 10% other) and were ultimately diagnosed and managed with an array of manometric diagnoses (14 normal, 39 weak, 5 hypertensive, 7 EGJ outflow obstruction, and another 2 patients had rapid contraction with normal latency that could potentially have been described as weak peristalsis given the large breaks in the 20-mm Hg isobaric contour plot). Thus, the current version of the Chicago criteria requires 20% or more of the swallows to have a reduced DL (**Fig. 7**A), defined as less than 4.5 s, to meet the criteria for a diagnosis of DES.[7] Patients with 20% or more of the swallows with a rapid CFV (>9 cm/s) but with normal DL (see **Fig. 7**B) are categorized as rapid contraction, which is a diagnosis without a known clinical significance.

Although further study of treatment outcomes using DL as the diagnostic criterion for DES is needed for additional support for the use of DL, this study suggests that the diagnosis of DES based on an abnormal DL defines a more distinct clinical phenotype and, in agreement with Spechler and Castell,[1] is likely an uncommon disorder.

### *Esophageal Hypercontraction*

Another disorder frequently associated with noncardiac chest pain and dysphagia is nutcracker esophagus, a disorder usually defined by an elevated intensity of

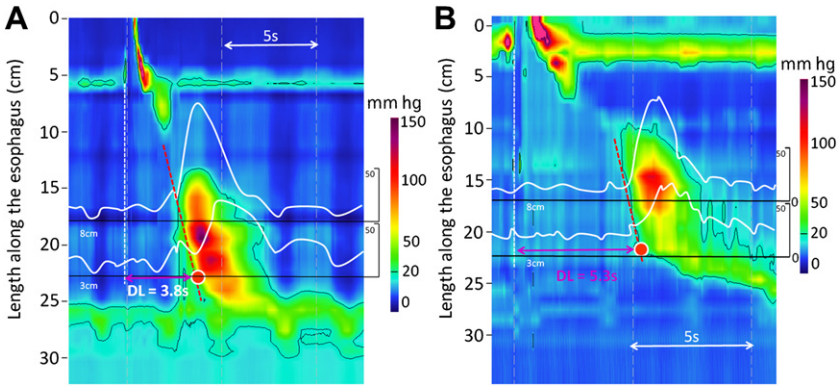


**Fig. 7.** Distal esophageal spasm. (*A*) DES, when defined by premature contractions (DL <4 seconds), is nearly uniformly associated with chest pain or dysphagia. (*A*) DL 3.8 seconds; CFV 12.1 cm/s. (*B*) Rapid contraction (CFV >9 cm/s) with normal latency (DL 5.3 seconds, CFV 14.1 cm/s) is associated with various clinical symptoms as well as in normal control. The EGJ relaxation is normal in both panels ([*A*] IRP 12.3 mm Hg, [*B*] IRP 0 mm Hg). DL (*purple arrow*). CDP (*red circle*). CFV (*red dashed line*).

esophageal peristaltic contractions. Nutcracker esophagus is the primary disorder of esophageal hypercontractility described by Spechler and Castell[1] and the early versions of the Chicago criteria. However, further evaluation with EPT has again refined this spectrum of esophageal hypercontractile disorders to distinguish borderline motor function from a primary abnormality of peristalsis.

### *Conventional criteria*

Previous studies on nutcracker esophagus with CM have generally defined the disorder by a distal wave amplitude of more than 2 standard deviations more than the normal. However, normal values and the location of high amplitude contractions (eg, diffuse or segmental) have varied. Thus, Spechler and Castell[1] proposed that the diagnostic criteria for nutcracker esophagus be focused on a mean distal esophageal peristaltic wave amplitude more than 180 mm Hg, measured as the average amplitude of the 10 swallows at recording sites 3 and 8 cm above the LES.[1] Increased contraction duration was an inconsistently described characteristic of nutcracker esophagus and, thus, was not required for manometric diagnosis.

### *Phenotypes of hypercontractile disorders*

Although the elevated wave amplitude has persistently been a part of the diagnostic criteria of nutcracker esophagus, its occurrence is not always associated with the characteristic symptoms of dysphagia and chest pain.[21] EPT uses the metric of DCI to measure peristaltic contractile vigor, which accounts for both contractile intensity (akin to wave amplitude) and duration. EPT analysis of normal subjects (N = 75) and patients (N = 400) defined a mean normal (95th percentile) DCI value of less than 5000 mmHg-s-cm, although there was substantial heterogeneity in the group of patients with a mean DCI of 5000 to 8000 mmHg-s-cm (a group classified as *hypertensive peristalsis* or *nutcracker esophagus*).[4] In addition, a mean DCI more than 8000 mmHg-s-cm (defined in the early Chicago classifications as *spastic nutcracker esophagus*) was a rare finding, seemed to exhibit a distinct pattern with repetitive high-amplitude contractions (**Fig. 8**), and was universally associated with dysphagia and/or chest pain.[4–6]

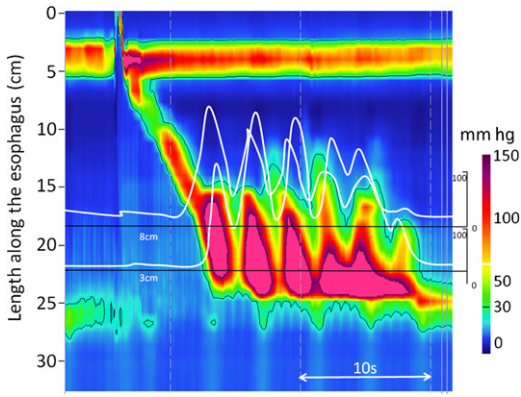


**Fig. 8.** Jackhammer esophagus. Clouse plot with overlying CM line tracings of a hypertensive swallow with significantly elevated DCI (15,025 mmHg-s-cm), mean wave amplitude (260 mm Hg), and displaying multi-peaked esophageal body contractions.

Further evaluation of EPT studies of 72 asymptomatic controls and 1070 patients led to the refinement of this classification.[22] The examination of individual swallows, as opposed to the *mean* DCI, found that within the control group, subjects often had individual swallows with DCIs more than 5000 mmHg-s-cm (median DCI 2073 mmHg-s-cm, 5th–95th percentile 757–5946); the highest single DCI value seen in the control group was 7732 mmHg-s-cm. On the other hand, 44 patients (4.1%) were found to have at least one swallow with a DCI more than 8000 mmHg-s-cm, the majority (75%) of whom presented with dysphagia and generally had a positive response to a variety of treatments (including antireflux, anticholinergic, and endoscopic Botox injection). Classifying patients based on a *single* swallow with a DCI more than 8000 mmHg-s-cm, a group whose *mean* DCI had an interquartile range of approximately 3900 to 8700, had a high proportion of patients that presented with symptoms of chest pain and dysphagia, thus deterring the use of the previous arbitrarily set use of the *mean* DCI.

Multi-peaked contractions were frequently seen in the patients with a DCI more than 8000 mmHg-s-cm (36 out of 44, 86%); thus, the term *jackhammer esophagus* was coined to describe this pattern. The patients that displayed multi-peaked hypertensive contractions were more likely to have a normal IRP than patients who were hypertensive without multi-peaked contractions. Although the presence or absence of these multi-peaked contractions did not seem to have an association with the symptom profile or to have an association with the response to treatment, this was a retrospective report of an uncontrolled data set with a wide variety of treatments. Further systematic treatment trials classifying patients with hypertensive (DCI >8000 mmHg-s-cm) peristaltic contractions based on the presence or absence of multi-peaked, jackhammerlike contractions may lend additional insight on the clinical phenotype of this manometric pattern.

Based on this study, the current version of the Chicago classification describes the diagnostic criteria for determining hypercontractile (jackhammer) esophagus based on at least one swallow with a DCI more than 8000 mmHg-s-cm (with or without multi-peaked contractions), whereas hypertensive peristalsis (nutcracker esophagus) is defined as a mean DCI more than 5000 mmHg-s-cm but not meeting criteria for hypercontractile esophagus.[7]

### *Hypocontractile Disorders: Ineffective Esophageal Motility*

Spechler and Castell[1] classified a group of disorders with hypocontractile characteristics as ineffective esophageal motility disorders (IEM). These disorders had previously often been referred to as *scleroderma esophagus* and *nonspecific esophageal motility disorders*, although many other disorders other than scleroderma (eg, GERD) may demonstrate similar motility patterns.

#### *Conventional criteria*

The CM diagnostic criterion for IEM proposed in the 2001 review was ineffective wet swallows in 30% or more of the swallows. Ineffective swallows could be demonstrated manometrically by any combination of (1) distal esophageal peristaltic wave amplitude of 30 mm Hg or less, (2) simultaneous contractions with amplitudes of 30 mm Hg or less, (3) failed peristalsis (the peristaltic wave does not traverse the length of the distal esophagus), and/or (4) absent peristalsis. These criteria were based on previous findings that similar esophageal CM metrics were associated with impaired bolus transport or ineffective esophageal acid clearance.[1,23] An additional study using impedance-manometry to assess esophageal bolus clearance refined the criteria of IEM, demonstrating that a cutoff of 50% or more of the ineffective swallows improved identification of patients with abnormal esophageal bolus transport and had a trend toward representation of a group of patients more likely to demonstrate dysphagia and/or heartburn.[24,25]

#### *EPT: weak and failed peristalsis*

EPT allows one to characterize the contractile activity of the entire esophagus as opposed to separated axial measurements as used in studies using CM. Thus, EPT offers improved detection of breaks in the peristaltic wavefront (**Fig. 9**). Studies using EPT and intraluminal impedance have demonstrated that peristaltic breaks of more than 2 cm in the 20-mm Hg isobaric contour plot may be associated with impaired bolus transport.[26,27] The Chicago criteria describes hypocontractile disorders as *weak peristalsis* and *frequent failed peristalsis*. *Weak peristalsis* is defined by >30% of swallows with small (2–5 cm) peristaltic defects or >20% of swallows with large

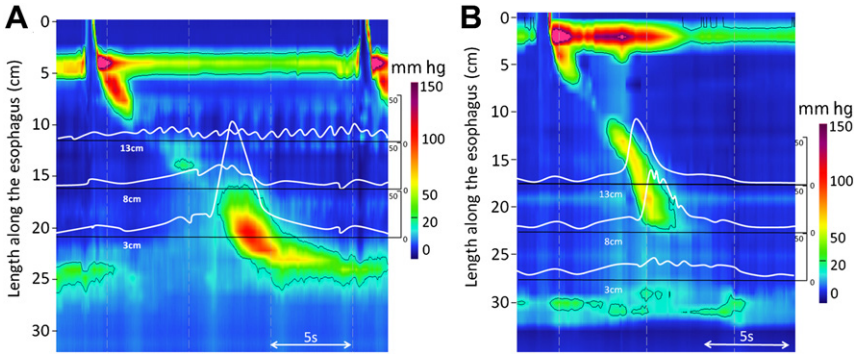


**Fig. 9.** Ineffective esophageal motility. EPT correlates of IEM included failed (not pictured) and weak (*A, B*) peristalsis. Axial separation of CM pressure sensors limits the assessment of esophageal peristaltic integrity as demonstrated with the Clouse plots with overlying CM line tracings from recording sites at 3, 8, and 13 cm from the LES of a swallow with a large transition zone peristaltic defect (*A*) and another swallow with both proximal and distal defects (*B*). Of note, the DCIs of swallows in (*A*) and (*B*) are 820 and 316 mmHg-s-cm, respectively.

(>5 cm) peristaltic defects.[7] Frequent failed peristalsis is defined by >30%, but <100% of swallows with failed peristalsis.[7] A comparison of EPT characteristics of 75 normal controls and 113 patients with nonobstructive dysphagia demonstrated that weak peristalsis with small and large peristaltic breaks (but not failed peristalsis) were all seen more commonly in the patients than the controls.[27] However, the peristaltic breaks were only seen in approximately one-third of the patients and were also occasionally present in the normal controls.

Although hypocontractile peristalsis could be defined adequately using weak peristalsis and frequent failed peristalsis on EPT, many clinicians and investigators were hesitant to adopt this new terminology and continued to use IEM. This idea was the impetus for a recent study that sought to correlate the CM diagnosis of IEM (a diagnosis not included in the Chicago classification) with EPT findings. They compared individual swallows and manometric classifications (based on a complete 10-swallow study) in terms of CM line tracings (taken at 3 and 8 cm from the LES) and EPT.[12] IEM was defined by the updated CM criteria (>50% ineffective swallows), and EPT studies were analyzed according to the Chicago classification diagnoses of weak peristalsis and frequent-failed peristalsis (defined earlier). EPT abnormalities (individual swallows with peristaltic break or failed swallow; classification of weak or frequent failed peristalsis) were found in more than 25% of swallows deemed normal by CM and more than 35% of studies classified as normal. By removing comparison with EPT studies with proximal pressure trough (transition zone) defects, the agreement in manometric characterization between the two methods increased appreciably. The addition of a CM line tracing at 13 cm from the LES considerably increased the ability of CM line tracings to detect the presence of a transition zone defect (see **Fig. 9**A), which is a defect whose size may have an association with symptoms such as heartburn or dysphagia.[28]

The same study described earlier also suggests that DCI had a strong correlation with mean wave amplitudes at 3 and 8 cm from the LES.[12] Low DCI, a measure not previously included in the Chicago classification, was shown to be a strong predictor of ineffective (DCI <450 mmHg-s-cm) or failed (DCI <50 mmHg-s-cm) peristalsis and, thus, could potentially be incorporated into automated manometry diagnostic software and possibly future revisions of the Chicago classification.

As expected, the additional data of peristaltic integrity generated by EPT are able to increase the characterization of disorders of esophageal hypocontraction, although a correlation could be made between the CM diagnosis of IEM and the combined EPT classifications of weak peristalsis with small or large defects and frequent-failed peristalsis. Nonetheless, the clinical significance of any of these disorders or findings is not completely clear, and further manometric pattern assessment and clinical treatment trials may offer additional insight into their contribution to heartburn and/or dysphagia.

## SUMMARY

Increased clinical experience with EPT has helped identify specific patterns that seem to distinguish clinically relevant phenotypes within the classical description of esophageal motility disorders using CM. The Chicago criteria has attempted to bridge the gap between the previous diagnostic experience with CM and the new technology and has primarily focused on using the enhanced information available with HRM to better define abnormal motor function. The objective metrics of esophageal peristaltic and sphincter function available with EPT analysis also facilitate the use of a diagnostic scheme that uses an algorithm-based diagnostic model that can be incorporated into analytic software programs.

Ultimately, as is initially apparent with achalasia subtype designations, it is hoped that further characterization of clinical phenotypes represented by manometric patterns will offer an improved ability to tailor therapy to specific clinical entities and enhance our ability to care for patients with esophageal motility disorders.

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