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High Resolution Manometry: An Atlas of Esophageal Motility Disorders and Findings of GERD using Esophageal Pressure Topography

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INTRODUCTION

High resolution manometry, in and of itself, is an adaptation of conventional manometric hardware that basically employs an increased number of pressure sensors spaced closely together. The data generated by HRM would therefore be displayed as a tracing format similar to what would be utilized for conventional manometric interpretation. The real advance in terms of manometry is primarily focused on the analysis techniques that were derived to optimize the information from high-resolution manometry. In order to better visualize the data, Clouse and Staino incorporated a process of interpolation or averaging between sensors to display the information in the form of seamless isobaric color regions on esophageal pressure topography plots (EPT) (1) (Figure 1). The EPT or “Clouse Plots” have the capacity to convert manometric information into distinct patterns that illustrate the physiology of contractile coordination and the mechanics associated with bolus transit. Additionally, Geoff Hebbard’s group also deserve special mention in any discussion on HRM as they were responsible for developing one of the first analysis software packages focused on EPT. This software was subsequently utilized to define subtle peristaltic defects important in bolus transit that were missed with conventional manometry (2).

Although this technique was reserved primarily to research centers, it has now become widely available and currently is moving into mainstream clinical practice. Fortunately, there has been a substantial amount of work that have adapted the concepts of the classification scheme utilized in conventional manometry into a new classification for EPT that incorporates the added detail and accuracy of this new analysis technique. This work has resulted in a new classification scheme and the development of new measurements derived specifically for EPT that focus heavily on pattern recognition of contractile and bolus pressurization patterns. Thus, EPT is more akin to an imaging technique as opposed to a data output stream displayed over time.

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The goal of the current review is to provide an Atlas of esophageal motility disorders focused on dysphagia and gastroesophageal reflux disease. Although there is some overlap between esophageal motor disorders associated with dysphagia and the defects and esophageal motor function that would predispose the patient to more severe GERD, the following review will be organized to differentiate the distinct pathophysiologic components of the two disease groups. The section on dysphagia will provide a description of disorders associated with EGJ outflow obstruction and disorders of contractility associated with premature spastic contractions, hypercontractility and impaired bolus clearance. In contrast, the section on GERD will focus on a description of an incompetent EGJ and antireflux barrier. A section on impaired bolus transit will also be included in the GERD section to highlight the important aspect of impaired clearance in the pathogenesis of GERD.

Dysphagia

Dysphagia will typically result secondary to either an obstruction to flow or any inability to adequately propel the bolus in the antegrade direction into the stomach. Thus, an Atlas focused on describing disorders associated with the symptom of dysphagia should highlight examples of EGJ outflow obstruction and abnormalities of contraction associated with impaired antegrade flow.

EGJ obstruction—The measurement of the pressure changes through the esophago-gastric junction during swallowing is much more complex than previously understood. The EGJ is a complex anatomical zone that maintains a closed state at baseline via a delicate interplay between neurogenic, myogenic and mechanical properties. During swallowing the lower esophageal sphincter normally relaxes to allow bolus transit through the EGJ. However, other factors also impede flow by resisting opening during bolus transit. The hiatal canal is usually the narrowest diameter through the EGJ(3) and thus, this will be the primary determinant of flow rate through the EGJ. Additionally, there are other mechanical properties of the esophageal wall which will also resist opening secondary to elastic properties of the EGJ. Therefore, the pressure signal through the EGJ during swallowing can be altered by dysfunction of LES relaxation and a reduced opening diameter related to a mechanical obstruction (stricture, eosinophilic esophagitis, tumor, LES hypertrophy)(4) or abnormal anatomy (hiatus hernia)(5).

The 2 measurements that are important in defining EGJ outflow obstruction are the integrated relaxation pressure (IRP) and the intra-bolus pressurization pattern (6, 7). The IRP is a complex metric as it involves accurately localizing the margins of the EGJ, demarcating the time window following deglutitive upper sphincter relaxation within which to anticipate EGJ relaxation to occur, applying an e-sleeve measurement [axial EGJ domain] within that 10 second time box (Figure 2). The IRP is presented as the mean value of the four seconds during which the e-sleeve value was least. Patients that exhibit an abnormal IRP and/or an elevated intra-bolus pressurization have evidence of EGJ outflow obstruction, however, the specific cause cannot be discriminated purely by manometric evaluation of the EGJ alone. Thus the distinction of achalasia from other disorders associated with EGJ outflow obstruction will require further evaluation of the pathognomonic contractile and pressure patterns in addition to a detailed description of the EGJ anatomy (Figure 3).

Achalasia: Achalasia is defined by an increased IRP and absent peristalsis. This can be further subtyped into 3 specific groups based on the characteristics that are associated with absent peristalsis. Type I achalasia is associated with 100% failed peristalsis and no evidence of contractile activity or pressurization greater than 30 mmHg (Figure 4A). Type II achalasia is also associated with no normal peristalsis and no significant contractile activity but distinguished from type I based on the presence of panesophageal pressurization greater

than 30 mmHg (Figure 4B). Type III achalasia manifests absent peristalsis in the context of preserved fragments of contraction or premature spastic contractions greater than equal to 20% of the swallows studied space (Figure 4C). The clinical relevance of this subtype classification has been assessed in 3 studies(8-10). These studies support that Type II has the best prognosis, while Type I is worse than Type II but much better than Type III. Type III patients will still manifest spastic contractions within the body of the esophagus and will require treatment above the EGJ focused on reducing the spastic contractions.

EGJ outflow obstruction: EGJ outflow obstruction is defined by an abnormal IRP and/or elevated intra-bolus pressure in the context of some instances of intact peristalsis or weak peristalsis with small breaks such that the criteria for achalasia are not met (4). This may be related to a mechanical obstruction secondary to a stricture (Figure 5A), or an anatomical defect such as a hernia (Figure 5B). Additionally, this pattern can be seen in patients with post surgical complications at the EGJ (fundoplication) or the proximal stomach (lap band).

Spastic Disorders—The major pathologic features of spastic disorders of the esophagus are premature contractions defined by reduced latency and rapid contractions with normal latency. The three measurements important in defining spastic disorders are noted in Table 1 and described in Figures 6 and 7. Before the appropriate measurements can be made, two important landmarks must be established: Onset of swallowing and the contractile deceleration point (CDP)(11). Once these landmarks are established, measurements of CFV and Distal Latency can be made. The latency interval is a measurement that defines the zone of normal deglutitive inhibition through the smooth muscle esophagus (12). Contractions where the contractile deceleration point occurs within 4.5 seconds of the initiation of the swallow defined by the UES are likely associated with impaired inhibition in the smooth muscle esophagus and bolus entrapment (Figure 7)(13). Rapid contractions are defined by contractile velocity greater than 9 cm/s, however, this particular parameter must be evaluated in the context of latency (11). Swallows with rapid contraction and normal latency do not exhibit impaired inhibition in the smooth muscle esophagus and thus, are different than the classic spastic disorders that are associated with the corkscrew esophagus or rosary bead esophagus on fluoroscopy. The different phenotypes of spastic disorders are illustrated in Figure 8 (Pandolfino et al – *In Press- Gastroenterology 2011*). The clinical implications of rapid contractions with normal latency is unclear, however, rapid contractions associated with reduced Distal Latency is a distinct pathophysiologic entity never encountered in asymptomatic control populations.

Hypercontractile Disorders—The classic hypercontractile disorder was originally described as nutcracker esophagus due to the high peristaltic amplitudes noted on conventional manometry(14). Current definitions are based on the measurement of mean contractile amplitudes 3 and 8 cm above the LES (15) and over the years some experts have called for a more stringent definition to better define this pathophysiologic clinical entity (16). With this call to better define hypercontractile disorders, the EPT classification of hypertensive contractile disorders has also sought to separate out clinically significant phenotypes of hypercontractility. A new metric to define hypercontractility, the Distal Contractile Integral (DCI), was devised to summarize the vigor of the distal esophageal contraction measured for the segment spanning from the proximal to distal pressure troughs (Figure 9). The DCI can be conceptualized as the volume of the pressure from P to D, thereby being sensitive to the length of that span, as well as the amplitude and duration of the contraction at each locus along the way (17). To exclude the effects of intrabolus pressure in the DCI computation, the first 20 mmHg is ignored (17, 18). Consequently, if a swallow was not associated with any recorded pressure >20 mmHg in the P to D span, the DCI for that swallow would be zero. However, keep in mind that the DCI was devised

primarily to identify swallows of excessive contractile vigor making the upper rather than the lower limit of normal the more relevant limit. The upper limit of normal defined by the 95th percentile in a normal population is 5,000 mmHg-s-cm whereas defined by the value never encountered in a normal population, it is 8,000 mmHg-s-cm (18, 19).

Weak and Failed Peristalsis—The importance of Weak and Failed peristalsis in the pathogenesis of dysphagia is unclear and therefore, these motor disorders will be more fully discussed in the section on GERD. However, two specific disorders do deserve special mention. Absent peristalsis is characterized by failed peristalsis with 100% of swallows. This disorder is uniformly associated with poor bolus transit and this motor pattern is associated with type I achalasia (Figure 4A) and the scleroderma pattern if the patient has a hypotensive LES. Occasionally, absent peristalsis can be found in the context of a normal LES pressure and intact deglutitive EGJ relaxation. These patients will also be predisposed to poor esophageal clearance and will therefore, be susceptible to more severe GERD and possibly post-fundoplication dysphagia(20). A distinct form of weak peristalsis associated with a defect in the transition zone or the proximal trough can be associated with dysphagia and poor proximal bolus clearance (2, 21, 22). These patients may be susceptible to pill esophagitis and proximal regurgitation (see section on impaired esophageal clearance).

Gastroesophageal Reflux

The primary determinants of GERD severity are a dysfunctional antireflux barrier and impaired esophageal clearance. The antireflux barrier prevents reflux of gastric contents into the esophagus, while peristalsis helps to clear the refluxate in order to reduce exposure to the noxious components of the gastric juice. The primary mechanisms of reflux have focused on three dominant mechanisms: 1) transient LES relaxations (tLESRs), without anatomic abnormality, 2) LES hypotension, again without anatomic abnormality, or 3) anatomic distortion of the EGJ inclusive of (but not limited to) hiatus hernia. Once the gastroesophageal refluxate enters the esophagus, peristalsis functions to clear the esophagus of the refluxate. Defects in the integrity of the peristaltic wave will lead to impaired bolus transit and prolonged esophageal acid exposure. This is a critical component of the pathogenesis of GERD and prolonged exposure times are associated with more severe GERD.

Antireflux barrier—Disruption of the antireflux barrier can be related to a hypotensive LES (< 10 mmHg)(23), an abnormal gastroesophageal flap valve(24), radial disruption of the crural canal(25) and hiatus hernia. These defects are not mutually exclusive and the defects are cumulative in terms of their effect on disrupting the antireflux barrier. High-resolution manometry and EPT allow one to accurately assess the intrinsic LES and the crural component contribution to the EGJ high-pressure zone (Figure 10). The crural component can be assessed by measuring the inspiratory augmentation during baseline recordings and recent data suggests that this measurement may be a predictor of GERD (26).

In addition, the separation between the intrinsic LES and crural diaphragm can be measured simultaneously during HRM studies to document the presence of hiatus hernia (27). In addition, localization of the respiratory inversion point can provide some insight into the level of crural disruption (Figure 10).

Impaired esophageal clearance—Patients with weak or failed peristalsis will have an impaired ability to clear the refluxate after gastroesophageal reflux occurs. The degree of impairment is likely a function of the number of failed peristaltic events and whether the peristaltic contraction is intact or associated with breaks in the peristaltic wavefront (Figure 11). Patients with absent peristalsis have the most severe impairment in esophageal

clearance and thus, are likely to have the most severe GERD. If this is associated with a hypotensive LES, a diagnosis of scleroderma must be considered, however, this pattern is not pathognomonic for this disease and can be seen in the context of severe GERD (Figure 12). Another esophageal motor defect associated with GERD is ineffective esophageal motility. Classically, this entity was defined as a peristaltic amplitude less than 30 mmHg in the conventional recording sites located in the distal esophagus 3 and 8 cm above the LES(28). In HRM, this definition has changed by leveraging the spatial resolution of HRM to measure the actual defect lengths in the contractile wavefronts. Recent work by Roman et al have defined that defects greater than 5 cm are uniformly associated with impaired bolus transit (29). Although confirmatory studies are needed, it is likely reasonable to equate distal defects in the contractile wavefront with swallows associated with IEM.

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Figure 1.

The distinction between high-resolution manometry (HRM) and esophageal pressure topography (EPT). The high resolution manometry catheter consists of 21 to 36 closely spaced pressure sensors. The position of pressure sensors in the esophagus is indicated in the anatomical drawing on the left panel. In the middle panel, the data from HRM recordings are displayed as tracings for each pressure sensor similar to conventional manometry. EPT is illustrated in the panel on the right. EPT is distinct from HRM in that it is a purely a data analysis method where as HRM is a data acquisition method. The Clouse Plots (EPT plots) are derived from the data acquired from HRM and are displayed using a color code to describe peristaltic amplitude in a space time continuum. A technique of interpolating between the recording sensors allows one to obtain a seamless representation of the pressure activity through the entire swallow. Note the tremendously enhanced detail provided by the Clouse plot, especially in the area of the esophago-gastric junction.

Figure 2.

Concomitant esophageal pressure topography (EPT) and fluoroscopy during esophageal emptying illustrating the transition from peristaltic transport to ampullary emptying. The fluoroscopic images in the windows are synchronized with the EPT plot. The white and blue dots indicate areas of intrabolar pressure and the onset of luminal closure respectively. The second image (at about time 8 s) is near the contractile deceleration point (CDP), evident both by the transition of the fluoroscopic image to ampullary conformation and slowing of the luminal closure front. The dashed rectangles within the deglutitive relaxation window indicate the time fragments used to compute the integrated relaxation pressure (IRP). The distal border of the esophago-gastric junction is indicated by black line on EPT and by white arrows on barium swallow. Note that the pressure signal through the two boxes which demarcate the IRP measurement are a manifestation of the compartmentalized intrabolar pressure between the EGJ and the contractile wavefront. Thus, this measurement provides a measure of the pressure gradient across the EGJ and it therefore, is not a one dimensional measure of LES relaxation.

Figure 3.

Diagnosis and treatment algorithm for patients with dysphagia/chest pain and negative structural work-up. Esophageal pressure topography (EPT) study allows an assessment of distal outflow pressure. An abnormal integrated relaxation pressure (IRP) and/or abnormal intra-bolus pressure (IBP) defines that an esophago-gastric junction (EGJ) outflow obstruction is present and further diagnosis requires an analysis of contractile activity. In conjunction with absent peristalsis, achalasia is diagnosed. The treatment will then focus on reducing EGJ outflow obstruction. When esophageal peristalsis is preserved (normal, weak or hypertensive), the pattern is distinguished from achalasia and is defined as EGJ outflow obstruction. Further evaluations are then required to ascertain the cause of obstruction.

Figure 4.

Achalasia subtypes. All three subtypes are characterized by impaired EGJ relaxation (IRP >15 mmHg) and absent contractile activity. In type I (Panel A) there is negligible pressurization in the esophageal body, evident by the absence of any area circumscribed by the 30 mmHg isobaric contour (black line). In type II (Panel B) pan-esophageal pressurization occurs evident by the banding pattern of the 30 mmHg isobaric contour spanning from the upper esophageal sphincter (UES) to the esophagogastric junction (EGJ). This represents elevated intrabolus pressure and is associated with contraction of the longitudinal muscle on the muscularis propria. Type III achalasia (Panel C) is characterized by spastic contractions with or without periods of compartmentalized pressurization.

Figure 5.

Examples of esophago-gastric junction (EGJ) outflow obstruction. In Panel A, EGJ outflow obstruction occurs in a context of stricture. It is characterized by an elevated IRP and a distal compartmentalization. In Panel B, EGJ outflow obstruction occurs in a context of hiatal hernia. The hernia is evidenced on Clouse plots by the separation between the lower esophageal sphincter (LES) and the crural diaphragm (CD). In this case, IRP is restricted to the LES (black box, IRP_{LES}) and CD (black dashed boxes, IRP_{CD}) separately. The CD component of the basal EGJ pressure profile is quite pronounced. A compartmentalized pressurization occurred between the contractile wave and the CD. This reflects the greater resistance through the CD evidenced by elevated IRP_{CD} (16.6 mmHg).

Figure 6.

A normal swallow in a Clouse plot. Before and after the swallow, 2 high pressure zones are visualized: the upper esophageal sphincter (UES) and the esophago-gastric junction (EGJ). The highlighted black line is the 30-mmHg isobaric contour circumscribing areas on the plot with intraluminal pressure greater than 30 mmHg. The peristaltic esophageal contraction is characterized by 2 troughs, one proximal (P) and one distal (D). The contractile deceleration point (CDP) represents the inflexion point in the contractile front propagation. It is localized on Clouse plots by fitting 2 tangential lines to the initial and terminal portions of the 30-mmHg isobaric contours and noting intersection of the lines (white dot). On fluoroscopic image it corresponds to the transition to ampullary conformation and slowing of the luminal closure front. The contractile front velocity (CFV) corresponds to the slope of the tangent line to the initial portion of the contraction (between P and the CDP). The distal latency (DL) is measured from the onset of swallow (dashed vertical line) to the CDP.

Figure 7.

The concept of reduced distal latency in spasm as described by Behar and Biancani (Behar J, Biancani P. Pathogenesis of simultaneous esophageal contractions in patients with motility disorders. *Gastroenterology*. 1993;105(1):111-8). The latency of propagation for normal controls (black circles) and a patient with spasm (orange circles) adapted from Behar and Biancani (left panel). The latency interval was measured using conventional manometry by as the time from onset of contraction at sensor 21 to onset of contraction at sensor 1. The latency interval was determined to be a marker of the inhibitory ganglionic integrity, suggesting that patients with spasm had evidence of reduced latency and premature contraction. In the right panels, the latency interval plots from the conventional manometry study are superimposed on EPT tracings of a swallow with normal latency (top) and short latency (bottom). In each case, the time and sensor position scales are adjusted to approximate those of the conventional manometry tracing. *With permission: The Northwestern University HRM Database*

Figure 8.

Examples of premature and rapid esophageal contractions. Premature contraction is defined by a distal latency (DL, measured from onset of swallow to the contractile deceleration point) < 4.5 s. Rapid contraction is defined by a contractile front velocity (CFV, measured from the proximal trough to the CDP) > 9 cm/s. The contraction on Panel A is rapid and premature and this represents a manometric description of the “corkscrew” or “rosary bead” esophagus. Note that this contraction is associated with a large proximal defect. The contraction on Panel B is premature with a normal CFV and occurred in a context of an abnormal esophagogastric junction (EGJ) relaxation. This pattern is extremely rare and is typically associated with evidence of obstruction at the EGJ. The contraction on Panel C is rapid with a normal DL and is associated with a large proximal defect. This pattern is found in asymptomatic controls and is associated with poor bolus transit related to the defect in the contractile wavefront.

Figure 9.

Hypertensive peristalsis. Distal contractile integral (DCI) is the metric used to define the vigor of the contraction. It is the product of the amplitude $\times$ duration $\times$ length of the contraction between the proximal and the distal trough. The contraction exhibited in Panel A is normal. A hypertensive contraction defined by a DCI $> 5,000$ mmHg-s-cm is presented in Panel B. This contraction occurs in a context of normal propagation and normal esophago-gastric (EGJ) relaxation. Nutcracker esophagus is defined by a mean DCI of 10 swallows greater than 5,000 mmHg-s-cm in a context of normal propagation and normal EGJ relaxation. Panel C illustrates an extremely phenotype of hypertensive contraction characterized by a DCI $> 8,000$ mmHg-s-cm and repetitive prolonged contractions evoking the action of the jackhammer. This extreme phenotype is named jackhammer contraction and it is never found in asymptomatic controls.

Figure 10.

Esophago-gastric junction (EGJ) morphology characterized in Clouse plots. The two main EGJ components are lower esophageal sphincter (LES) and crural diaphragm (CD), which cannot be independently quantified when superimposed, classified as type I EGJ (Panel A). The respiratory inversion point (RIP), shown by the horizontal dashed line, lies at the proximal margin of the EGJ. During the expiration (E) EGJ pressure increases whereas it decreases during inspiration (I). In case of hypotensive EGJ only the diaphragmatic component is identified (Panel B). Panels C and D correspond to type III EGJ defined as LES-CD separation > 2 cm. A type III EGJ is the manometric criterion for hiatal hernia. The RIP is within the EGJ at the proximal margin of the CD. Two subtypes were discernible, IIIa (Panel C) and IIIb (Panel D), with the distinction being that the respiratory inversion point was proximal to the CD with IIIa and proximal to the LES in IIIb.

Figure 11.

Varying degrees of peristaltic integrity in high resolution manometry combined with impedance. In each panel, the black line represents the 20 mmHg isobaric contour. Impedance data are displayed by overlaid pink colorization with the pink shading indicative of areas on the topography plots with retained bolus. The swallow in Panel A is intact (no disruption in the 20 mmHg IBC) and associated with complete bolus transit. Panel B illustrates a failed swallow (absence of 20 mmHg integrity in the distal two thirds of the esophagus) associated with bolus retention in the esophagus. Swallows with small break in the 20 mmHg isobaric contour are illustrated in Panels C and D. The transition zone break is responsible for bolus escape in Panel C. In Panel D the distal break is associated with bolus retention in the distal esophagus and corresponds to what has been previously defined as ineffective esophageal motility.

Figure 12. Scleroderma. The typical pattern is characterized by hypotensive lower esophageal sphincter pressure and absence of contraction in the distal two thirds of the esophagus.

Table 1

Important measures in assessing contractile propagation

CDP (time, position) <i>Contractile Deceleration Point</i>	The inflection point along the 30 mmHg isobaric contour where propagation velocity slows demarcating the tubular esophagus from the phrenic ampulla
CFV (cm/s) <i>Contractile Front Velocity</i>	Slope of the tangent approximating the 30 mmHg isobaric contour between P and the CDP
DL (s) <i>Distal Latency</i>	Interval between UES relaxation and the CDP