Chicago Classification Version 4.0 and Its Impact on Current Clinical Practice

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Abstract: High-resolution manometry (HRM) has revolutionized esophageal motility testing, and the evolving Chicago Classification has been critical in codifying HRM metrics and definitions of old and new motility disorders. The latest Chicago Classification (version 4.0) is the result of a working group of 52 members (10 women) from 20 countries. Two critical new elements are the expansion of the normal database from 75 to 469 healthy volunteers and the recommendation of ancillary function tests (timed barium esophagram, functional lumen imaging planimetry, and/or impedance) to help with inconclusive HRM metrics, especially in cases of suspected achalasia, esophagogastric junction outflow obstruction (EGJOO), and ineffective esophageal motility (IEM). Important changes relevant to clinical practice include (1) refinement of the diagnosis criteria for EGJOO, which now require elevated integrated relaxation pressure in an upright position along with primary symptoms of dysphagia/noncardiac chest pain and obstruction at the esophagogastric junction; (2) exclusion of mechanical obstruction in cases of suspected distal esophageal spasm and hypercontractile esophagus; and (3) a shift to a more restrictive metric (>70% ineffective peristalsis) for a diagnosis of IEM. In addition, the working group urged caution in using treatments such as pneumatic dilation or myotomy, which can irreversibly destroy lower esophageal sphincter competency and peristalsis, as the natural history of EGJOO/hypercontractile esophagus is poorly understood and spontaneous symptom resolution is common. Future versions should address the routine use of impedance with HRM, the role of HRM in pharyngeal/upper esophageal sphincter diseases, and the need for better criteria to determine which subsets of spastic disorders warrant aggressive treatment, as is done with achalasia.

Keywords
High-resolution manometry, Chicago Classification, achalasia, esophagogastric junction outflow obstruction, diffuse esophageal spasm, hypercontractile esophagus, ineffective esophageal motility, absent contractility

It has been 30 years since high-resolution manometry (HRM) revolutionized the field of esophageal motility testing,[1] vastly improving diagnostic accuracy for certain esophageal motility diseases (achalasia), enabling the identification of new manometric abnormalities (esophagogastric junction outflow obstruction [EGJOO]), and making esophageal motility testing easier to perform and interpret. Along with

HRM came the need for a new classification system, coined the Chicago Classification in recognition of the important work led by Drs Peter Kahrilas and John Pandolfino at Northwestern University. This system has been refined over time based on the results of worldwide clinical studies. The first 3 iterations of the Chicago Classification have been cited more than 2000 times,[2] and the new version, 4.0, was published online in 2020.[3] This article examines the history of HRM and the Chicago Classification, the important diagnostic updates in the latest version, and the clinical areas that still need to be addressed.

Historical Perspective

High-Resolution Manometry

Older manometry techniques recorded esophageal peristalsis using 5-mm to 8-mm widely spaced water-perfusion channels in an esophageal motility catheter. Two significant advances in the 1990s—an increase in pressure sensors along the catheter and the use of spatiotemporal plots for data display—led to what is now recognized as HRM.

HRM was conceived and later developed by Dr Ray Clouse at Washington University in St Louis, Missouri. Dr Clouse decided that there was hidden information in the esophagus between the widely spaced recording ports of the conventional manometry catheter, leading health care providers (HCPs) to possibly miss important information for assessing peristalsis and lower esophageal sphincter (LES) function. Over 10 years of experiments,[4] Clouse and colleagues developed a 21-lumen water-perfusion catheter, digitized the information to give a smooth topographic map of esophageal peristalsis, and assigned colors to amplitude levels in spatiotemporal contour plots (an idea from his university training in architecture).[1,5,6] As technology progressed toward solid-state pressure sensors, Clouse found a key collaborator in Dr Thomas Park, who formed a new company, Sierra Scientific, Inc, to advance the field. Together, they developed a solid-state catheter with 36 high-fidelity circumferential sensors,[7] new software programs, and an electronic sleeve[8] that more accurately measured postswallow residual pressures. With Dr Clouse's untimely death from cancer, HRM technology moved from St Louis to Chicago and beyond. In honor of his contributions, the revolutionizing color pressure topography plots will always be known as Clouse plots.

Chicago Classification

The move to Chicago was heralded with a pivotal decision by Drs Kahrilas and Pandolfino to develop a new classification system, not limited to their own institution, but through collaboration with motility experts throughout the world via the American Neurogastroenterology and Motility Society and the European Society of Neurogastroenterology and Motility. They evolved the concept of a living document, which would be regularly updated as clinical and scientific data dictated to coincide with the anticipated 3-year cycle of Digestive Disease Week (DDW) being hosted in Chicago. The first major version of the Chicago Classification was published in 2009 after the inaugural meeting of the International HRM Working Group in San Diego in 2008.[9] The next major update followed from a meeting of the International HRM Workshop Group in Ascona, Switzerland in 2011 and was endorsed by numerous international motility and gastroenterology societies.[10] Later, an expanded International HRM Working Group met in Chicago in conjunction with DDW 2014 to formulate the Chicago Classification version 3.0.[11] Chicago Classification version 4.0 (CCv4.0) was delayed by the COVID-19 pandemic but was published online in 2020.[3] The working group was composed of 52 members (10 women) selected by 6 international motility societies, representative of 20 countries. The initiative was a 2-year process with 3 international meetings and multiple Web conferences. In addition to expert consensus, one of the group's main objectives was to use formally validated methodologies to determine both the appropriateness of statements and the level of supportive evidence for each statement. The RAND/University of California Los Angeles Appropriateness Method was used, with 2 rounds of independent electronic voting to determine the appropriateness of each statement.[12] Statements with greater than or equal to 85% agreement were considered strong recommendations, whereas statements

Table 1. Key Points About the Chicago Classification Version 4.0 Working Group, Expanded Database, and New Definitions

• 52 members (10 women) from 20 countries
• New normal database: 469 healthy volunteers (55% women, median age of 28 years) – EGJOO: supine, 25 (5.3%); upright, 2 (1.1%) – DES: 19 (4.1%) – Hypercontractile (jackhammer) esophagus: 1 (0.2%) – IEM: 71 (15.1%); more restrictive diagnosis of IEM, 47 (10%) – Absent contractility: 1 (0.2%)
• New definitions for EGJOO and IEM
• Eliminated fragmented peristalsis

DES, distal esophageal spasm; EGJOO, esophagogastric junction outflow obstruction; IEM, ineffective esophageal motility.

with 80% to 85% agreement were considered conditional recommendations. Moreover, statements that met criteria for inclusion in the final document underwent further independent evaluation to assess the level of supportive evidence using the Grading of Recommendations Assessment, Development, and Evaluation process.[13]

What Makes Chicago Classification Version 4.0 Different?

The definition of normality is critical for any classification system after the manometric criteria have been defined. For the first 3 versions of the Chicago Classification, normal values were primarily obtained from 75 healthy controls studied in Chicago.[14] This group consisted of 47% women with an age range of 19 to 48 years. Version 4.0 (Table 1) has expanded the normal database to 469 healthy volunteers (55% women, median age of 28 years, age range of 18-79 years) acquired from 15 countries across 4 continents using the 3 available commercial HRM systems.[15] Three-quarters had a normal HRM pattern and none had achalasia. Ineffective esophageal motility (IEM) was the most frequent diagnosis (15.1%), followed by EGJOO (5.3%). The supine position reduced the portion with IEM to 7.9% and the upright position reduced the portion with EGJOO to 2 volunteers (1.1%). Expanding the normal database also impacted the diagnosis of hypercontractile esophagus (HE), increasing the distal contractile integral (DCI) from greater than 5000 to greater than 8000 mm Hg-s-cm, as well as requiring the presence of 20% or more hypercontractile supine swallows to define abnormality.[16]

Although not specifically stated, the metrics used in the Chicago Classification hoped to allow HRM to define all features related to esophageal pump vigor of contractions, peristalsis, and esophagogastric junction (EGJ) compliance. It was hoped the new measures of distal latency (DL)[17] would replace the old terminology of simultaneous contractions and correlate more closely with bolus clearance by impedance studies, and integrated relaxation pressure (IRP)[18] would be an improvement on LES relaxation and better correlate with EGJ compliance. This has not been the case, especially with achalasia and EGJOO, where the median IRP of less than 15 mm Hg misses the diagnosis of achalasia in up to 20% of patients,[19] and an IRP of greater than 15 mm Hg overdiagnoses EGJOO in the supine position in more than 50% of patients.[20] Therefore, the CCv4.0 now encourages supportive testing with timed barium esophagram (TBE) combined with a 13-mm barium tablet[21] and/or endoluminal functional lumen imaging planimetry (FLIP)[22] in patients with an inconclusive diagnosis of achalasia/EGJOO with dysphagia as the presenting symptom.[3]

Table 2. Disorders of Esophagogastric Junction Outflow

Achalasia
• Type I: Abnormal IRP and absent contractility (100% failed peristalsis)
• Type II: Abnormal IRP and absent contractility with panesophageal pressurization in 20% or more swallows
• Type III: Abnormal IRP and evidence of spasm (20% or more swallows with premature contraction) with no peristalsis
• Cutoff of spasm in 20% of swallows is arbitrary; confidence for Type III achalasia is increased with higher number of premature swallows
• Inconclusive diagnosis of achalasia is best resolved with a TBE with a 13-mm barium tablet and/or FLIP in patients with dysphagia
• Opioids are associated with Type III achalasia, and patients should be studied off opioid medication, if possible. Timing of discontinuation is based on drug half-life
EGJOO
• Manometric diagnosis of EGJOO is always considered clinically inconclusive
• Manometric diagnosis requires an elevated median IRP in both the supine and upright positions, increased intrabolus pressure, and evidence of peristalsis
• Clinically relevant symptoms of EGJOO include dysphagia (usually solid foods) and/or noncardiac chest pain
• Definitive diagnosis of EGJOO requires supportive evidence of obstruction by TBE with a barium tablet and/or FLIP

Bold text indicates important new criteria in Chicago Classification version 4.0.

EGJOO, esophagogastric junction outflow obstruction; FLIP, functional lumen imaging planimetry; IRP, integrated relaxation pressure; TBE, timed barium esophagram.

Disorders of Esophagogastric Junction Outflow

Achalasia

As shown in Table 2, the basic definitions of the 3 types of achalasia have not changed in CCv4.0. Abnormal median IRP is the first key measurement, with a threshold of 15 mm Hg in the supine position using the Medtronic system and 22 mm Hg for the Laborie/Diversatek system.[16] The major improvement in CCv4.0 is the recognition that these absolute cutoff values do not always accurately measure outflow obstruction and the compliance of the

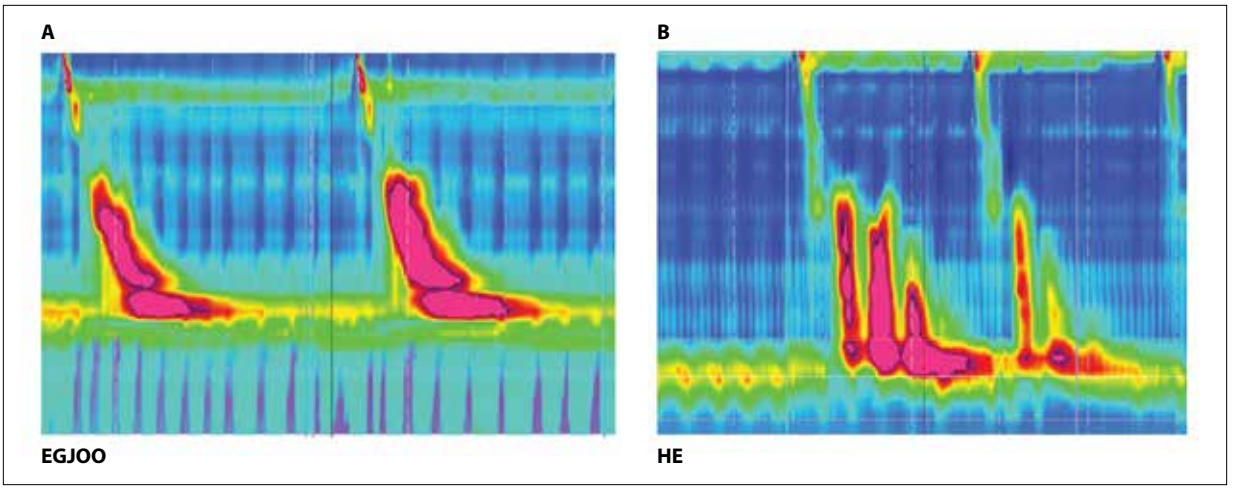

Figure. High-resolution manometry images (**A:** EGJOO; **B:** HE) from the University of South Florida's Swallowing Center. The vertical axes are esophageal length (cm), horizontal axes are time (s), and colors increasing from blue to red represent pressures (intrathoracic chest pressure) in mm Hg.

EGJOO, esophagogastric junction outflow obstruction; HE, hypercontractile (jackhammer) esophagus.

EGJ. Rather, high values with aperistalsis give us confidence in a diagnosis of achalasia, but values of less than 15 mm Hg can still be seen in up to 20% to 25% of patients with achalasia and dysphagia, both in the naive or treated state.[19] For these inconclusive cases, a TBE with a barium tablet or FLIP is critical for making the diagnosis. Both measure distensibility (compliance) of the EGJ with the TBE column at 5 minutes greater than 2 cm correlating closely with a FLIP distensibility index greater than 2.9 mm^2/mm Hg.[23] For this reason, many HCPs routinely use HRM first, followed by a TBE in patients with suspected achalasia. A repeat TBE after treatment is then performed to assess improvement in esophageal emptying.[24]

A relatively new observation is that Type III achalasia and EGJOO are significantly more likely to be associated with the use of opioids compared with achalasia Types I and II.[25,26] Studies suggest that 11% to 13% of Type III achalasia and 13% to 37% of EGJOO may be due to activation of μ and κ receptors by opiates, which impair LES relaxation. The most common narcotics are oxycodone, hydrocodone, and tramadol. It is suggested, but not known, that studying patients off their opioids for variable times based on drug half-life will reduce this potential confusion. Although an interesting observation, there are no good data that patients on opioids with Type III achalasia/EGJOO and dysphagia and abnormal TBE/FLIP will not do well with traditional therapies if the opioids cannot be discontinued.

Esophagogastric Junction Outflow Obstruction

A critical update in CCv4.0 is the clarification and rigorous definition of EGJOO (Table 2).[3] Following the introduction of EGJOO as a motility disorder, nearly 10% of patients undergoing HRM were identified to have this pattern (Figure).[27] An unknown subset of these patients present with a variant of achalasia, but the vast majority of presentations are related to benign conditions (eg, peptic strictures, large hiatal hernia, eosinophilic esophagitis, tight Nissen fundoplication), opioid use, subtle cancers, or artifacts of the pressure measurements. All these presentations usually have dysphagia for solid food and, less frequently, noncardiac chest pain as the predominant symptoms.

When a diagnosis of EGJOO is suspected after 10 swallows in the supine position, the patient should be positioned upright and asked to provide at least 5 swallows. An upright median IRP of greater than 12 mm Hg (Medtronic) or greater than 15 mm Hg (Laborie/Diversatek) is defined as abnormal[20] and rarely seen in healthy patients.[15] The reason for the decrease in IRP from supine to an upright position may be a catheter impingement artifact.[27] In patients with hiatal hernia, catheter angulation of 1 to 2 sensors located in the EGJ segment by the hernia sac can result in erroneous elevation of the IRP. Sitting the patient upright is the easiest way to resolve this issue. Other provocative tests to assess for outflow obstruction during HRM include rapid drink challenge with subsequent esophageal pressurization, solid test meal with replication of symptoms, or pharmacologic provocation with amyl nitrate.[27]

As my colleague and I have previously suggested,[28] an elevated IRP should be evaluated for other mechanical causes of obstruction using upper endoscopy with biopsies, barium esophagram, or endoscopic ultrasound

(especially in patients with significant weight loss), while also reviewing history of opioid use. If no etiologies are found, then a functional obstruction (sometimes called Type IV achalasia) must be confirmed with an abnormal TBE with barium pill or FLIP. In a retrospective study of TBE by Blonski and colleagues,[21] only 27% of patients had an abnormal test, which was similar to the 30% reported by Triggs and colleagues.[20]

Based on the published data, the key question is whether idiopathic EGJOO is a manometric curiosity or an obstructive disease that warrants treatment. Proposed treatments in case reports or series include muscle relaxants, bougie dilation, botulinum toxin (BTX), pneumatic dilation, or surgical myotomy, with improvements (usually determined only by symptom assessment) ranging from 35% to 100%.[28] Importantly, no treatment with observation alone may result in spontaneous resolution over 6 months to 2 years in 15% to 72% of patients.[28] One study, however, did find that symptoms were likely to persist if dysphagia was the predominant symptom and the IRP was very high (>32 mm Hg).[29] Thus, members of the CCv4.0 Working Group suggest avoiding invasive, irreversible treatments on the LES, especially pneumatic dilation or myotomy. If symptoms are severe, then first consideration should be given to BTX injection, as it is safe and reversible.[27] Porter and Gyawali[30] describe the beneficial results of a single injection lasting up to 1.5 years in 55% of patients. However, my colleagues and I did this early in our experience with EGJOO and saw 2 patients evolving to Type III achalasia.[31] We currently treat healthy patients with EGJOO, severe dysphagia, and an abnormal TBE with pneumatic dilation and recently reported a symptomatic response rate of 67% and marked improvement in esophageal emptying.[32] Patients have been doing well for up to 7 years, and none have returned with achalasia, raising the possibility that more aggressive improvement of EGJ compliance may prevent the loss of peristalsis over time.

Disorders of Peristalsis

Distal Esophageal Spasm

Distal esophageal spasm (DES) is a rare and elusive motility disorder due to partial loss of inhibitory innervation via nitric oxide, producing premature, rapid, or simultaneous contractions.[33,34] As shown in Table 3, CCv4.0 has not changed the primary manometric criteria of (1) greater than or equal to 20% premature contractions and (2) DCI of greater than 450 mm Hg-s-cm.[3] For an unexplained reason, some normal peristalsis being present is no longer required.[11] This omission could cause some confusion with Type III achalasia with a normal IRP unless careful assessment of EGJ compliance is performed. The presence of premature contractions with a DCI of less than 450 mm Hg-s-cm is inconclusive for the manometric diagnosis of DES. In this scenario, these manometric changes are often part of the gastroesophageal reflux disease (GERD) spectrum. The additional observation of abnormal inhibition defined by the persistence of peristalsis in the

Table 3. Disorders of Peristalsis

DES
• Manometric diagnosis of DES is defined as the presence of at least 20% premature contractions (distal latency <4.5 s) and DCI >450 mm Hg-s-cm
• Some normal peristalsis is no longer required
• A diagnosis of DES requires clinically relevant symptoms (dysphagia and/or noncardiac chest pain) and manometric criteria
HE
• Manometric diagnosis of HE is defined as 20% or more hypercontractile, supine swallows (DCI >8000 mm Hg-s-cm)
• Must have clinically relevant symptoms (dysphagia/ noncardiac chest pain)
• A diagnosis of HE can only be made when criteria for achalasia/DES are not met and mechanical obstruction has been excluded
IEM
• A diagnosis of IEM requires more than 70% ineffective swallows (DCI >100 mm Hg-s-cm but <450 mm Hg-s-cm) or at least 50% failed peristalsis (DCI <100 mm Hg-s-cm)
• The presence of 50% to 70% ineffective swallows is inconclusive for a diagnosis of IEM. Supportive testing is required, including **– Poor bolus transit on impedance or barium esophagram** **– Lack of contraction reserve on multiple rapid swallows**
• The term fragmented peristalsis should no longer be used
Absent Contractility
• A diagnosis of absent contractility is defined as normal median IRP in the supine and upright positions and 100% failed peristalsis

Bold text indicates important new criteria in Chicago Classification version 4.0.

DCI, distal contractile integral; DES, distal esophageal spasm; HE, hypercontractile (jackhammer) esophagus; IEM, ineffective esophageal motility; IRP, integrated relaxation pressure.

distal esophagus during multiple rapid swallows supports a diagnosis of DES.[3] Importantly, to prevent confusion with normal variations, GERD, or neuropathic entities such as diabetes mellitus, CCv4.0 now requires the clinically relevant symptoms of dysphagia and/or noncardiac chest pain to make a definitive diagnosis of DES.

Hypercontractile Esophagus

HE is the HRM version of the old nutcracker esophagus first described by Benjamin and colleagues.[35] The manometric criteria (Table 3) are identical for both versions 3.0 and 4.0 of the Chicago Classification. That is, a manometric diagnosis of HE is defined as 20% or more hypercontractile swallows in the supine position (DCI >8000 mm Hg-s-cm). Esophageal hypercontractility can either be limited to the esophageal body or include the LES. A variant form with prominent, high-amplitude, repetitive contractions has been given the colorful name of jackhammer esophagus (Figure).

HE is a rare condition that ranges from 1.5% to 3% of manometric diagnoses in motility centers.[36] The pathophysiology is the result of excessive cholinergic drive with temporary asynchrony of circular and longitudinal muscle contractions.[37,38] HE can sometimes be associated with EGJOO as well as other causes of mechanical obstruction, including GERD, obstructing hiatal hernia, eosinophilic esophagitis, and gastric laparoscopic bands.[39] Opioid use also has been associated with HE.[25] CCv4.0 recognizes this potential causality and emphasizes the importance of appropriately addressing these diseases before treatment directed only at the hypercontracting esophagus, such as smooth muscle relaxants, BTX, or long surgical myotomy.

As with DES, diagnosis of HE requires that patients have symptoms of dysphagia and/or noncardiac chest pain. Noncardiac chest pain is not associated with any specific manometric criteria, but several studies have reported dysphagia associated with DCI of the hypercontractile swallows and with intrabolus pressure.[39,40] The jackhammer subgroup is typically associated with higher DCI values and greater symptom severity.[39]

Given the heterogenicity of hypercontractile patterns, the CCv4.0 Working Group advocates for a cautious approach in treating contractile vigor as an endpoint[3] because its relationship to symptoms, especially chest pain, and natural history are poorly understood. Relevant to these points are 2 important observations. A French randomized study of BTX in patients with HE demonstrated that symptom improvement after BTX was not superior to sham and that symptoms and manometric patterns may improve spontaneously over time.[41] Specifically, 3 of 10 patients who did not receive any treatment had resolution of their HE, including 1 with jackhammer esophagus, over 3 to 12 months. This could be due to a placebo effect or to spontaneous resolution. Similarly, in a Mayo Clinic retrospective study[29] of HRM scans, which included 40 patients with HE, 72% of patients had resolution of their symptoms over 2.8 years of follow-up. Interestingly, predictors of continued symptoms included dysphagia as the main symptom and a DCI of greater than 32,132 mm Hg-s-cm. These studies need to be appropriately weighed when considering treating refractory EGJOO and HE with peroral endoscopic myotomy surgery.[42]

Ineffective Esophageal Motility

A major improvement in CCv4.0 is the change in the diagnostic criteria for IEM, which was driven by the finding that 71 healthy volunteers (15.1%) in the expanded database met this criteria.[16] Therefore, a definitive diagnosis of IEM was made more restrictive, now requiring greater than 70% ineffective swallows (DCI >100 mm Hg-s-cm but <450 mm Hg-s-cm) or greater than or equal to 50% failed swallows (DCI <100 mm Hg-s-cm).[3] With that, the presence of 50% to 70% ineffective swallows will be inconclusive for a definitive diagnosis of IEM and will require confirmatory testing to strengthen confidence in these cases. Supportive testing for the diagnosis of IEM may include poor bolus transit on impedance or barium esophagram or lack of contraction reserve on multiple rapid swallows. Fragmented peristalsis (rarely seen in practice) was eliminated as a distinct esophageal motility disorder and absorbed into the definition of IEM.[3]

On the surface, these changes may seem like minor fine-tuning, but in clinical practice, they serve as important new guidance given that IEM is the most commonly reported esophageal motility disorder in patients (20%-58%).[43] GERD is observed in nearly half of patients with IEM, and patients with Barrett esophagus have a strong predilection for IEM. In addition, IEM has been noted as a frequent abnormality in patients with diabetes mellitus with autonomic dysfunction, alcoholism with neuropathy, and collagen vascular diseases. However, it can be seen in healthy patients, even up to 10% with the more rigid diagnostic criteria, and many patients have minimal or no symptoms of dysphagia.[44] The finding of IEM becomes clinically important, especially in patients with GERD, when dysphagia is the dominant symptom and antireflux surgery is planned. Patients with IEM have an increased risk of postfundoplication dysphagia if a 360-degree Nissen fundoplication is performed,[45] and IEM is a contraindication for magnetic sphincter augmentation.[46] Reserving the diagnosis of IEM for the more severe form with additional evidence of poor bolus clearance and impaired peristaltic reserve will expand the

potential for antireflux surgery without increasing the chance for troubling dysphagia.

Absent Contractility

As shown in Table 3, the criteria for the diagnosis of absent contractility (previously known as absent peristalsis) were not revised in CCv4.0.[3] A definitive diagnosis will require a normal median IRP in the supine and upright positions and 100% failed contractions. In this context, a borderline high IRP should prompt consideration of Type I achalasia, and supportive testing with TBE or FLIP should be performed if dysphagia is the dominant symptom.

Similar to IEM, absent contractility is not a specific diagnosis and has been reported in many diseases with a neuropathic or myopathic etiology. However, in clinical practice, this motility pattern is most suggestive of a collagen vascular disease, and although sometimes termed scleroderma esophagus, this pattern has been reported in all the collagen vascular diseases, including healthy patients with isolated Raynaud's phenomenon.[47] Esophageal disease is observed in up to 90% of patients with either limited or diffuse forms of scleroderma.[47] In a recent prospective study of 200 patients with scleroderma, absent contractility was reported in 56% of patients, followed by normal motility in 26%, and IEM in 10%. Interestingly, the classic scleroderma esophagus motility pattern with low LES pressure and absent contractility was only observed in 33% of patients.[48]

Future Directions

CCv4.0 highlights areas ripe for future research. Although many issues need to be addressed, there are several areas that may be most relevant to clinical practice. First, future iterations of the Chicago Classification must incorporate impedance topography to better assess intrabolus pressure and bolus flow.[49] This may be most relevant to the perplexing diagnosis of DES, where at least 20% premature contractions (DL <4.5 s) makes the diagnosis. However, catheters combining HRM and impedance routinely find these abnormal contractions associated with normal bolus clearance. Thus, like the HRM metric of IRP, a DL of less than 4.5 seconds needs to be supported by impaired bolus clearance before abnormality can be assured. Another area needing attention is the use of HRM for pharyngeal and upper esophageal sphincter (UES) function in health and disease.[50] This may be more challenging because the striated muscle can be markedly affected by psychological factors. Here, the modified barium swallow will need to be included to ensure the relevance of HRM metrics with bolus flow and UES relaxation.

Although CCv4.0 has refined the diagnosis of EGJOO and HE, it leaves HCPs with the conundrum of who to treat and how to treat. Telling patients that their symptoms of dysphagia and chest pain are likely to improve spontaneously will be met with resistance and added confusion like the old nutcracker esophagus dilemma. Natural history and treatment outcome studies, preferably with a randomized control design, are greatly needed. Furthermore, the roles of TBE and FLIP in defining who to treat or just follow need refinement. Future iterations may propose manometric criteria for therapy selection, such as the role of peroral endoscopic myotomy for spastic disorders and risk stratification and tailoring of fundoplication to prevent postfundoplication dysphagia.

Lastly, there remains a vast unknown between achalasia (and its 3 phenotypes) and normal, with 5 manometric patterns representing heterogenous diseases whose natural histories are unknown and treatments remain uncertain. It is hoped that future versions of the Chicago Classification will provide guidance on these areas as well.

Summary

The Chicago Classification has been critical in codifying HRM metrics and definitions of old and new motility disorders. The latest version (4.0) has 2 critical new elements: (1) expansion of the normal database from 75 to 469 healthy volunteers and (2) the recommendation of ancillary function tests (TBE, FLIP, and/or impedance) to help with inconclusive HRM metrics, especially in cases of suspected achalasia, EGJOO, and IEM. Important changes for the HCP include (1) refinement of the diagnosis of EGJOO, (2) exclusion of mechanical obstruction in cases of suspected DES and HE, and (3) a shift to a more restrictive metric (>70% ineffective peristalsis) for a diagnosis of IEM. Additionally, the working group urged caution in using treatments such as pneumatic dilation or surgical myotomy, which can irreversibly destroy LES competency and peristalsis, as the natural history of EGJOO/HE, unlike that of classic achalasia Types I to III, is poorly understood and spontaneous symptom resolution is common.

Disclosures

Dr Richter serves as a consultant for Medtronic. He is a coauthor of the Chicago Classification versions 3.0 and 4.0.

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