

Consensus Statement of Society of Abdominal Radiology Disease-Focused Panel on Barium Esophagography in Gastroesophageal Reflux Disease

Marc S. Levine¹
 Laura R. Carucci²
 David J. DiSantis³
 David M. Einstein⁴
 Mary T. Hawn⁵
 Bonnie Martin-Harris⁶
 David A. Katzka⁷
 Desiree E. Morgan⁸
 Stephen E. Rubesin¹
 Francis J. Scholz⁹
 Mary Ann Turner²
 Ellen L. Wolf¹⁰
 Cheri L. Canon⁸

Keywords: barium esophagography, Barrett esophagus, esophageal adenocarcinoma, esophagram, gastroesophageal reflux disease, reflux disease, reflux esophagitis

DOI:10.2214/AJR.16.16323

Received February 22, 2016; accepted after revision April 29, 2016.

M. S. Levine and S. E. Rubesin are consultants for Bracco Diagnostics.

¹Department of Radiology, Perelman School of Medicine, Hospital of the University of Pennsylvania, 3400 Spruce St, Philadelphia, PA 19104. Address correspondence to M. S. Levine (marc.levine@uphs.upenn.edu).

²Department of Radiology, Virginia Commonwealth University Medical Center, Richmond, VA.

³Department of Radiology, Mayo Clinic, Jacksonville, FL.

⁴Imaging Institute, Cleveland Clinic, Cleveland, OH.

⁵Department of Surgery, Stanford University Medical Center, Stanford, CA.

⁶Department of Otolaryngology, Medical University of South Carolina, Charleston, SC.

⁷Department of Medicine, Mayo Clinic, Rochester, MN.

⁸Department of Radiology, University of Alabama at Birmingham School of Medicine, Birmingham, AL.

⁹Department of Radiology, Lahey Hospital and Medical Center, Burlington, MA.

¹⁰Department of Radiology, Montefiore Medical Center, Bronx, NY.

AJR 2016; 207:1–7

0361–803X/16/2075–1

© American Roentgen Ray Society

OBJECTIVE. The Society of Abdominal Radiology established a panel to prepare a consensus statement on the role of barium esophagography in gastroesophageal reflux disease (GERD), as well as recommended techniques for performing the fluoroscopic examination and the gamut of findings associated with this condition.

CONCLUSION. Because it is an inexpensive, noninvasive, and widely available study that requires no sedation, barium esophagography may be performed as the initial test for GERD or in conjunction with other tests such as endoscopy.

Gastroesophageal reflux disease (GERD) is likely the most common disease affecting the gastrointestinal tract. GERD has a frequency of 10–20% in Western countries [1], and heartburn occurs in at least 5% of adult Americans [2]. Accurate diagnosis of GERD enables affected individuals to benefit from a variety of treatment options, ranging from lifestyle changes to medication or antireflux surgery.

Patients with GERD may undergo a variety of diagnostic tests, including fiberoptic endoscopic examination of swallowing or nasopharyngolaryngoscopy to assess for functional or structural abnormalities in the pharynx or larynx; endoscopy to assess for reflux esophagitis and complications, such as peptic strictures and Barrett esophagus; esophageal manometry to assess for dysmotility; and 24-hour esophageal impedance–pH monitoring to determine the degree of acid and nonacid reflux.

In current medical practice, endoscopy has become a widely used diagnostic test for GERD, whereas barium studies have been underutilized in the workup of this condition. The decreasing role of esophagography is related to a variety of factors, including a growing shortage of experienced teachers in gastrointestinal fluoroscopy; the common perception of barium radiology as a labor-intensive, time-consuming, and technically demanding modality; and an endoscopy-first mentality of gastroenterologists that undervalues the usefulness of barium studies. In practice guidelines from various gastroenter-

ologic and endoscopic organizations, barium esophagography either is not recommended or not even mentioned as an option for the workup of GERD [3–5].

Despite this literature, barium esophagography has been recognized as a global examination that can simultaneously evaluate swallowing function, esophageal motility, gastroesophageal reflux (GER), and morphologic abnormalities in the pharynx and esophagus [6]. It is also a noninvasive and inexpensive procedure that does not require sedation and is widely available. The barium study, therefore, is a useful test for detecting GERD and its complications, distinguishing GERD from other pathologic conditions involving the esophagus, facilitating selection of additional diagnostic tests, and guiding decisions about medical, endoscopic, or surgical treatment of these patients.

The Society of Abdominal Radiology recently established a disease-focused panel on GERD whose mission is to advance the concept of a multidisciplinary approach to GERD that recognizes the important role of barium esophagography in conjunction with endoscopy and other diagnostic tests. The panel is composed of 10 gastrointestinal and abdominal radiologists, one gastroenterologist, one gastrointestinal surgeon, and one speech language pathologist. After review of the scientific literature and discussion among the panelists, our panel has developed a consensus statement on the role of barium esophagography for GERD, recommended techniques for performing

the fluoroscopic examination, and the gamut of radiographic findings associated with this condition.

Clinical Issues

Patients with GERD often present with classic symptoms of heartburn and regurgitation. Heartburn is usually characterized by intermittent substernal burning that markedly improves or resolves on treatment with proton pump inhibitors [7]. When substernal discomfort is particularly pronounced, it can mimic pain of cardiac origin [8]. Less frequently, substernal pain may be caused by eosinophilic esophagitis or esophageal motor disorders, such as diffuse esophageal spasm or achalasia masquerading as GERD [9].

Many patients with GERD present with recurrent episodes of regurgitation characterized by a bitter or sour taste in the mouth and the sensation of fluid moving up and down in the chest [7]. Regurgitation often is exacerbated by lying down, so this symptom is especially common at night. When patients with GERD have intractable nocturnal regurgitation as their predominant complaint, an antireflux procedure may be required for long-term clinical relief.

Other patients with GERD present with dysphagia because of esophageal sensitivity to refluxed acid in the esophagus or marked esophageal dysmotility associated with reflux esophagitis [7]. Dysphagia in these patients may also be caused by esophageal narrowing from peptic strictures, lower esophageal rings, Barrett esophagus, or even adenocarcinomas arising in Barrett esophagus. When dysphagia is present, the clinical history is extremely helpful for differentiating benign from malignant causes; benign strictures cause long-standing dysphagia and little or no weight loss, whereas malignant strictures are characterized by recent onset of progressive dysphagia and greater weight loss.

Some patients with GERD experience epigastric pain or dyspepsia that is erroneously attributed to peptic ulcer disease or other causes [10]. Other patients have extraesophageal symptoms such as a globus sensation, chronic cough, laryngitis, hoarseness, asthma, or water brash (profound salivation) secondary to daytime or nocturnal reflux of acid into the pharynx with subsequent aspiration of acid into the larynx or airway [11, 12]. Findings of GERD may also be detected unexpectedly in patients who undergo barium studies for other reasons.

Technique of Examination

When barium esophagography is performed for GERD, a pharyngogram routinely should be included to detect associated abnormalities in the pharynx and cervical esophagus. Thus, the study technically should be called a "pharyngoesophagram," though the shorter term "esophagram" is used here for the sake of brevity.

Single-contrast esophagography has been an unreliable technique for detecting reflux esophagitis, with an overall sensitivity of only 50–75% [13–16]. In contrast, double-contrast esophagography has a sensitivity of nearly 90% for detecting this condition [14, 16, 17]. A major advantage of a double-contrast technique is its ability to detect superficial ulcers or mucosal edema and inflammation in the absence of ulcers. Nevertheless, false-negative studies occur because of excessive intraluminal barium that obscures mucosal disease, and false-positive studies occur because of air bubbles and undissolved effervescent agent that mimic the findings of esophagitis. An optimal double-contrast technique therefore is required for these examinations.

We recommend performing the barium study as a multiphasic examination that includes dynamic evaluation and spot images of the pharynx and cervical esophagus to detect associated abnormalities in these structures, including upright double-contrast views of the esophagus with high-density barium to detect reflux esophagitis and esophageal tumors; double-contrast views of the cardia and fundus to detect tumors and other abnormalities in the cardiac region; prone single-contrast views of the esophagus with low-density barium to detect esophageal narrowing from strictures, rings, or other causes; assessment of esophageal motility to detect GERD-related esophageal dysmotility; and assessment of GER. Strategies for optimizing these various components of the barium study are suggested in the following sections.

Pharyngogram

Nocturnal reflux of acid into the pharynx may cause swallowing dysfunction with decreased epiglottic tilt, pharyngeal paresis, laryngeal penetration, or tracheal aspiration. Lateral and frontal views of the pharynx and cervical esophagus may be obtained by dynamic imaging with digital recordings or rapid sequence imaging as the patient swallows high- and low-density barium to assess swallowing function. If marked swallowing

dysfunction or aspiration is observed, a more careful pharyngeal examination can be performed as a modified barium swallow in conjunction with a speech language pathologist, using standardized protocols with barium agents of varying viscosity [18]. Depending on the findings, various compensatory maneuvers may be attempted to improve swallowing function and prevent or minimize aspiration [19]. Lateral and frontal spot images of the pharynx and cervical esophagus may also be obtained during suspended respiration and phonation to detect and document structural abnormalities of the pharynx and cervical esophagus (e.g., Zenker diverticulum and cervical esophageal webs) associated with GERD [20, 21].

Double-Contrast Esophagram

After ingesting an effervescent agent, the patient should continuously swallow high-density barium while in the upright left posterior oblique position for double-contrast views of the esophagus. Continuous swallowing of high-density barium suppresses esophageal peristalsis, enabling the esophagus to remain distended. If the patient takes only intermittent swallows of barium, each new episode of peristalsis collapses the esophagus, limiting the window of opportunity for obtaining double-contrast radiographs with adequate esophageal distention.

Emphasis should be placed on obtaining double-contrast views of the lower thoracic esophagus in patients with reflux symptoms, because this is the usual site of involvement by reflux esophagitis. Not infrequently, excessive intraluminal barium produces a white sheen that obscures mucosal disease from reflux esophagitis, also known as flow artifact [22]. Double-contrast views therefore should be obtained after the barium coating has thinned to minimize flow artifact and improve detection of reflux esophagitis.

Collapsed views of the esophagus (i.e., mucosal relief views) may also be obtained to show thickened or irregular folds from reflux esophagitis.

Examination of Gastric Cardia

After double-contrast views of the esophagus have been obtained, the patient should be placed in a recumbent right-side-down lateral position for a double-contrast view of the gastric cardia, which typically is characterized by three or four stellate folds radiating to a central point at the gastroesophageal junction, also known as the cardiac rosette

[23]. Because dysphagia often is referred proximally to the thoracic inlet or even the throat, the cardia should be evaluated in all patients with dysphagia, regardless of its subjective localization [24]. Early tumors at the cardia may be recognized by distortion or obliteration of the cardiac rosette, with subtle areas of nodularity, ulceration, or mass effect [25, 26]. At the same time, the cardia is a dynamic structure with a varying appearance, depending on whether the lower esophageal sphincter (LES) is open or closed. In some patients, the cardia may invaginate into the fundus when the LES is closed, producing an unusually prominent or nodular cardiac rosette, but this finding should vanish when the LES is open [27]. A questionable finding at the cardia therefore can be further evaluated by having the patient swallow additional barium to determine whether this apparent abnormality persists when the LES is open.

Evaluation of Esophageal Motility

Esophageal motility should be assessed as the patient takes separate single swallows of low-density barium in a prone right anterior oblique position. Normal motility is thought to be present when a primary peristaltic wave is seen as an inverted V stripping the barium column as it traverses the entire length of the esophagus. Esophageal dysmotility classically is thought to be present when peristalsis is abnormal on two or more of five swallows [28], though our panel agreed that two to three swallows are usually adequate for evaluating motility. It is important to recognize that primary peristalsis often is disrupted as a transient finding at the level of the aortic arch in older patients because of weakened peristalsis at the junction of the striated and smooth muscle portions of the esophagus [29]. This finding should not be mistaken for esophageal dysmotility.

Single-Contrast Esophagram

Esophageal distensibility is optimally evaluated when the patient continuously swallows low-density barium in the prone right anterior oblique position. This is important for visualizing distal esophageal rings and strictures that are easily missed on double-contrast views because of inadequate distention of this region when the patient is upright [30, 31]. Conversely, overdistention of the distal esophagus can lead to overlap between the distal esophagus and an adjacent hiatal hernia, preventing visu-

alization of the distal esophagus in profile and obscuring rings or ringlike strictures at or near the gastroesophageal junction [32]. This overlap phenomenon sometimes can be eliminated by having the patient swallow barium more slowly.

Evaluation of Gastroesophageal Reflux

GER is assessed by placing the patient in a supine position to pool barium in the gastric fundus. The cardia is located on the posteromedial wall of the fundus, so the patient is slowly turned into a supine right posterior oblique position to assess for GER as barium flows past the cardia. In some patients with an incompetent LES, barium refluxes spontaneously into the esophagus without the need for provocative maneuvers. Depending on how much the sphincter is compromised, patients can have occasional, intermittent, frequent, or continuous reflux of barium from the stomach. When GER is observed, a low-magnification image should be obtained to document the height and width of the barium column in the esophagus.

If spontaneous GER is not observed, the fluoroscopist may perform a straight leg-raising or Valsalva maneuver to raise intraabdominal pressure, increasing the sensitivity for GER. If GER still is not observed, the fluoroscopist may perform a water-siphon test by having the patient sip water in a supine right posterior oblique position [27]. When ingested water traverses the gastroesophageal junction and the LES opens, there normally is no GER or only a wisp of reflux into the distal esophagus (i.e., physiologic GER). In contrast, a large-volume reflux of barium into the esophagus almost always indicates pathologic GER. The water-siphon test has been shown to markedly increase the sensitivity of esophagography for pathologic GER, while simultaneously decreasing the specificity because of physiologic GER [33]. Some investigators, therefore, think that the water-siphon test is not useful for detecting GER [34]. However, our experience is that pathologic GER can be distinguished from physiologic GER in most patients, so we think the water-siphon test is a valuable adjunct to assess for GER on barium studies.

When GER is detected at fluoroscopy, patients should be asked to swallow their saliva to assess whether refluxed barium is cleared rapidly from the esophagus by esophageal peristalsis or remains within the esophagus for a prolonged period because of esophageal dysmotility.

Abnormalities Caused by Gastroesophageal Reflux Disease

Esophageal Dysmotility

Patients with GERD often have esophageal dysmotility, manifested as intermittently weakened or absent peristalsis in the mid and lower thoracic esophagus, typically in the absence of nonperistaltic contractions (also known as tertiary contractions) [35]. This common form of esophageal dysmotility should be distinguished from age-related dysmotility, in which intermittently weakened or absent peristalsis is almost always associated with nonperistaltic contractions of varying severity [29]. The presence or absence of nonperistaltic contractions therefore is a useful feature for differentiating age-related esophageal dysmotility from GERD as the cause of this motor dysfunction [35]. Rarely, GERD may be manifested as esophageal aperistalsis, with complete absence of a primary stripping wave in the esophagus [36].

Gastroesophageal Reflux

It is important not only to establish whether GER is present at fluoroscopy but also to assess the volume, level, frequency, and duration of GER episodes. Patients with high-volume reflux may have a large amount of refluxed barium distending the esophagus to the thoracic inlet, whereas patients with low-volume reflux may have only wisps of barium refluxing into the distal esophagus. GER to or above the thoracic inlet is particularly worrisome, because this type of reflux is more likely to be associated with nocturnal reflux of acid into the pharynx or larynx [11, 12].

It is also important to assess the duration of reflux episodes, because patients with prolonged reflux are at higher risk for injury. The classic example is patients with esophageal involvement by scleroderma, in which peristalsis is absent in the smooth muscle portion of the esophagus below the aortic arch, leading to poor clearance of refluxed acid from the esophagus [29]. As a result, these patients are at greater risk for developing reflux esophagitis and Barrett esophagus. In one study, 37% of patients with scleroderma and reflux symptoms had Barrett esophagus at endoscopy [37].

Hiatal Hernias

The vast majority of gastric hernias are hiatal hernias in which the gastroesophageal junction and proximal stomach herniate through the esophageal hiatus of the diaphragm into the lower thorax. The frequency

of hiatal hernias increases with age; 60% of older adults in the United States are found to have hiatal hernias on barium studies [38]. Although hiatal hernias may predispose to GERD, small hernias per se have doubtful clinical importance.

Because the gastroesophageal junction is demarcated by a mucosal junction ring (i.e., a B ring), hiatal hernias can be diagnosed on prone single-contrast views of the esophagus when a B ring is located more than 2 cm above the diaphragm [39]. In contrast, physiologic hiatal hernias (in which a B ring is located 2 cm or less above the diaphragm) result from contraction of longitudinal muscle in the esophageal wall and upward retraction of the esophagus by cricoid cartilage elevation during swallowing. Even when a B ring is not visualized, a hiatal hernia often can be recognized by the presence of gastric rugae within the hernia.

Single-contrast images with ingestion of barium in the prone right anterior oblique position are ideal for showing small sliding hiatal hernias that often are reduced when the patient is upright. Conversely, large hiatal hernias that persist in the upright position may indicate fixed longitudinal esophageal shortening, a finding that has ramifications for antireflux surgery [40]. Less frequently, barium studies may reveal paraesophageal hernias that are more likely to require surgery because of the risk of strangulation and infarction [39].

Reflux Esophagitis

Reflux esophagitis is usually manifested on double-contrast studies as a finely nodular or granular appearance in the distal esophagus caused by edema and inflammation of the mucosa. The granularity is characterized by poorly defined radiolucent elevations extending proximally from the gastroesophageal junction as a continuous area of disease [10]. It is the single most frequent sign of reflux esophagitis on double-contrast esophagrams, with a specificity and positive predictive value of about 90% [41]. Nevertheless, this granularity is sometimes obscured by flow artifact in the distal esophagus [22], so double-contrast spot images should be obtained after the barium coating has thinned, to improve detection of reflux esophagitis.

Some patients with reflux esophagitis may have multiple tiny ulcers visualized as punctate or linear barium collections at or near the gastroesophageal junction [10]. Progressive disease may lead to more extensive ul-

ceration in the distal thoracic esophagus. However, it would be extremely unusual for the ulcers in reflux esophagitis to be confined to the upper or midesophagus with distal esophageal sparing, so this finding should always suggest another cause of disease. Less commonly, reflux esophagitis may be manifested as a single dominant ulcer at or abutting the gastroesophageal junction [42]. Such ulcers tend to be located on the posterior wall of the distal esophagus because of nocturnal reflux of acid that pools on the posterior wall of the esophagus when patients sleep in the supine position [42].

Some patients with reflux esophagitis may have thickened longitudinal folds secondary to submucosal edema and inflammation [10], whereas others may have a single prominent fold that arises at the gastroesophageal junction and extends into the distal esophagus as a smooth polypoid protuberance known as an inflammatory esophagogastric polyp [43,44]. This benign lesion can almost always be differentiated from a true neoplasm by its characteristic appearance and location on barium studies without the need for endoscopy.

Scarring From Reflux Esophagitis

Reflux esophagitis is the most common cause of scarring in the distal esophagus. Reflux-induced (i.e., peptic) strictures typically are manifested as smooth tapered areas of concentric narrowing in the distal esophagus, almost always above a hiatal hernia [10]. Less commonly, peptic strictures may cause eccentric narrowing and small wide-mouthed sacculations secondary to focal outpouching of the wall between areas of fibrosis [10]. Still other peptic strictures may lead to the development of esophageal intramural pseudodiverticula, consisting of dilated excretory ducts of deep mucous glands in the esophagus. These structures typically appear on barium studies as tiny flask-shaped outpouchings from the esophageal wall [45]. When viewed *en face*, the pseudodiverticula can be confused with tiny ulcers, but when viewed in profile, they often seem to be floating just outside the lumen, whereas true ulcers almost always communicate directly with the lumen.

Although peptic strictures usually range from 1 to 3 cm in length, as many as 40% appear on barium studies as ringlike areas of narrowing less than 1 cm in length [46]. Such strictures may be difficult to differentiate from lower esophageal rings that cause dysphagia (i.e., Schatzki rings). However,

Schatzki rings are characterized by smooth symmetric ringlike constrictions at the gastroesophageal junction that are 2–4 mm in height [47], whereas ringlike peptic strictures are more asymmetric and almost always are greater than 4 mm in height [46]. Regardless of the cause, endoscopic dilation of the narrowed segment usually is required for relief of dysphagia.

In contrast, unusually long strictures in the distal esophagus should suggest reflux disease variants associated with severe scarring from reflux esophagitis [48]. Possible causes include Zollinger-Ellison disease, in which the refluxate contains an unusually high concentration of acid [49]; alkaline reflux esophagitis, in which bile refluxes into the esophagus after partial or total gastrectomy [50]; nasogastric intubation, in which acid refluxes around an indwelling nasogastric tube [51]; and esophageal involvement by scleroderma, in which absent peristalsis leads to poor clearance of refluxed acid from the esophagus [48]. Eosinophilic esophagitis is also characterized by long strictures or even diffuse esophageal narrowing, producing a so-called small-caliber esophagus [52]. All of these conditions therefore should be included in the differential diagnosis for unusually long distal esophageal strictures.

Scarring from reflux esophagitis can also lead to longitudinal shortening of the esophagus and the development of fixed transverse folds in the region of a peptic stricture, with multiple horizontal collections of barium trapped between the folds, producing a stepladder appearance [53]. In contrast, the feline esophagus is a transient phenomenon associated with GER that is characterized by thin closely spaced folds extending all the way across the esophagus [54], whereas fixed transverse folds due to scarring from chronic reflux disease are further apart, extend only part way across the esophagus, and are seen as a persistent finding associated with peptic strictures [53].

Barrett Esophagus and Esophageal Adenocarcinoma

Barrett esophagus is an acquired condition characterized by columnar (intestinal) metaplasia of the distal esophagus secondary to chronic GER and reflux esophagitis [10]. Barrett esophagus is more common than previously recognized, with an overall frequency of about 10% in patients with reflux esophagitis and 40% in patients with peptic strictures [10]. Barrett esophagus is impor-

tant because it is a premalignant condition associated with an increased risk of developing esophageal adenocarcinoma through a sequence of progressively severe epithelial dysplasia [55]. Published guidelines therefore recommend endoscopic surveillance of patients with Barrett esophagus for early detection and treatment of dysplasia before these individuals develop esophageal adenocarcinoma [56].

Barrett esophagus may be classified as short-segment (≤ 3 cm in length) or long-segment (> 3 cm in length) types [10]. The classic radiographic findings of long-segment Barrett esophagus consist of a high esophageal stricture or ulcer at a considerable distance from the gastroesophageal junction [10]. In the presence of a hiatal hernia and GER, a high stricture or ulcer is strongly suggestive of Barrett esophagus [10]. A distinctive reticular pattern of the mucosa has also been described as a specific sign of Barrett esophagus, particularly if it is adjacent to the distal aspect of a high stricture [57]. However, these classic findings of Barrett esophagus are found in only 5–10% of all patients with this condition [10]. Other more common findings, such as reflux esophagitis and peptic strictures, often are present in patients with uncomplicated GERD who do not have Barrett esophagus [10]. Thus, those findings that are more specific for Barrett esophagus (i.e., a high stricture or ulcer or a reticular mucosal pattern) are not sensitive, and those findings that are more sensitive (i.e., reflux esophagitis or peptic strictures) are less specific. As a result, many investigators think that double-contrast esophagography is a poor screening examination for Barrett esophagus and that endoscopy is required to establish this diagnosis.

Gilchrist et al. [58] have shown, however, that the findings on double-contrast studies can be used to classify patients as being at a high, moderate, or low risk for long-segment Barrett esophagus. Patients at high risk because of a high stricture or ulcer or a reticular mucosal pattern are almost always found to have Barrett esophagus, so endoscopic biopsy specimens should be obtained for a definitive diagnosis. A larger group of patients is found to be at moderate risk for Barrett esophagus because of reflux esophagitis or peptic strictures; the decision for endoscopy in this group should be based on the age and health of the patients (i.e., whether they are reasonable candidates for surveillance). However, most patients are found to be at

low risk for Barrett esophagus because of the absence of esophagitis or strictures. The frequency of Barrett esophagus is so low in this group that these patients can be treated empirically for their reflux symptoms without the need for endoscopy. Thus, the findings on double-contrast esophagography can be used to assess the risk of Barrett esophagus and the relative need for endoscopy in patients with reflux symptoms [58].

Pharyngeal and Cervical Esophageal Abnormalities

GERD that leads to nocturnal reflux of acid into the pharynx may result in swallowing abnormalities, such as decreased or delayed epiglottic tilt, pharyngeal paresis, laryngeal penetration, or tracheal aspiration secondary to chronic acid-induced inflammation of the pharynx or larynx. GERD may also cause cricopharyngeal dysfunction, most likely as a compensatory mechanism to prevent reflux of acid into the pharynx [59]. In such cases, barium studies typically reveal a smooth extrinsic indentation on the posterior aspect of the pharyngoesophageal junction secondary to incomplete cricopharyngeal opening, sometimes resulting in pharyngeal dysphagia. In some patients, cricopharyngeal dysfunction may contribute to the development of a Zenker diverticulum, manifested as a midline outpouching from the posterior wall of the pharyngoesophageal junction directly above a prominent cricopharyngeus [21]. Finally, GERD may be associated with the formation of cervical esophageal webs, another cause of dysphagia in these patients [20]. The value of pharyngography for evaluating GERD therefore cannot be overstated.

Relationship Between Radiographic Findings and Treatment of Gastroesophageal Reflux Disease

When patients with intractable reflux symptoms have high-volume GER to the thoracic inlet, these individuals may require a surgical antireflux procedure (e.g., Nissen fundoplication) for amelioration of symptoms. When planning this type of surgery, radiographic assessment of an underlying hiatal hernia is paramount. If a hernia is observed when the patient is in the recumbent position but not in the upright position (i.e., the hernia reduces in the upright position), these individuals are candidates for typical antireflux surgery (i.e., partial or total fundoplication). If patients have a large fixed hiatal hernia that fails to reduce in the upright position,

however, these individuals have a shortened esophagus that could prevent reduction of the hernia at surgery [40]. Such patients may require an esophageal lengthening procedure such as Collis gastroplasty, in which the surgeon fashions a gastric tube that functionally restores the distal esophagus when the hernia is reduced [40]. Thus, the radiographic findings not only can affect the decision between medical versus surgical treatment of patients with intractable GERD but also determine the type of surgery that is performed.

It is important to assess esophageal motility on barium studies, because esophageal dysmotility may impair clearance of refluxed acid from the esophagus, leading to more severe injury. Moreover, some surgeons think that normal esophageal motility is a prerequisite for fundoplication, because emptying of solids may be impaired at the site of the fundoplication wrap if esophageal motility is abnormal [60]. Conversely, other patients with GERD and esophageal dysmotility may have improved or even normal esophageal motor function after fundoplication because of decreased GER [61].

In patients with GERD, dysphagia may be caused by a host of abnormalities, ranging from reflux esophagitis to peptic strictures, Barrett esophagus, or even esophageal adenocarcinoma. In patients with GER or reflux esophagitis, acid-suppression therapy with proton pump inhibitors may ameliorate dysphagia secondary to esophageal sensitivity to refluxed acid or esophageal dysmotility. When dysphagia is caused by a distal esophageal stricture that has a benign appearance on barium esophagography, these strictures virtually always are found to be benign [46], so relief of dysphagia usually can be obtained by an endoscopic dilation procedure. If, however, the barium study reveals an equivocal or suspicious stricture, multiple biopsy specimens are required to differentiate a peptic stricture from Barrett carcinoma. Radiographic assessment of the morphologic features of a benign stricture can also assist the endoscopist in choosing the appropriate dilation technique and in determining the likelihood that multiple dilations will be required. Thus, the findings on barium studies have a major role in guiding subsequent selection of diagnostic or therapeutic procedures in these patients.

Conclusion

Our panel thinks that barium esophagography is a useful test for GERD, not only for

detecting a host of morphologic and functional abnormalities associated with this disease but also for facilitating other diagnostic tests, such as endoscopy, and for helping to guide medical versus surgical management. Because it is an inexpensive, noninvasive, and widely available study that requires no sedation, esophagography may be performed as the initial diagnostic test in patients with known or suspected GERD or in conjunction with other tests, such as endoscopy, 24-hour esophageal impedance-pH monitoring, and manometry.

References

- Dent J, El-Serag HB, Wallander MA, Johansson S. Epidemiology of gastro-oesophageal reflux disease: a systematic review. *Gut* 2005; 54:710–717
- Camilleri M, Dubois D, Coulie B, et al. Prevalence and socioeconomic impact of upper gastrointestinal disorders in the United States: results of the US Upper Gastrointestinal Study. *Clin Gastroenterol Hepatol* 2005; 3:543–552
- DeVault KR, Castell DO. Updated guidelines for the diagnosis and treatment of gastroesophageal reflux disease. *Am J Gastroenterol* 2005; 100:190–200
- Kahrilas PJ, Shaheen NJ, Vaezi MF, et al. American Gastroenterological Association medical position statement on the management of gastroesophageal reflux disease. *Gastroenterology* 2008; 135:1383–1391
- Katz PO, Gerson LB, Vela MF. Guidelines for the diagnosis and management of gastroesophageal reflux disease. *Am J Gastroenterol* 2013; 108:308–328
- Levine MS, Rubesin SE, Laufer I. Barium esophagography: a study for all seasons. *Clin Gastroenterol Hepatol* 2008; 6:11–25
- Kahrilas PJ. Regurgitation in patients with gastroesophageal reflux disease. *Gastroenterol Hepatol (N Y)* 2013; 9:37–39
- Lam HG, Dekker W, Kan G, Breedijk M, Smout AJ. Acute noncardiac chest pain in a coronary care unit. *Gastroenterology* 1992; 102:453–460
- Patterson DR. Diffuse esophageal spasm in patients with undiagnosed chest pain. *J Clin Gastroenterol* 1982; 4:415–417
- Levine MS. Gastroesophageal reflux disease. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*, 4th ed. Philadelphia, PA: Elsevier, 2015:291–311
- Vaezi MF, Hicks DM, Abelson TI, Richter JE. Laryngeal signs and symptoms and gastroesophageal reflux disease (GERD): a critical assessment of cause and effect association. *Clin Gastroenterol Hepatol* 2003; 1:333–344
- Hom C, Vaezi MF. Extra-esophageal manifestations of gastroesophageal reflux disease: diagnosis and treatment. *Drugs* 2013; 73:1281–1295
- Ott DJ, Gelfand DW, Wu WC. Reflux esophagitis: radiographic and endoscopic correlation. *Radiology* 1979; 130:583–588
- Koehler RE, Weyman PJ, Oakley HF. Single- and double-contrast techniques in esophagitis. *AJR* 1980; 135:15–19
- Ott DJ, Wu WC, Gelfand DW. Reflux esophagitis revisited: prospective analysis of radiologic accuracy. *Gastrointest Radiol* 1981; 6:1–7
- Creteur V, Thoeni RF, Federle MP, et al. The role of single and double-contrast radiography in the diagnosis of reflux esophagitis. *Radiology* 1983; 147:71–75
- Graziani L, De Nigris E, Pesaresi A, Baldelli S, Dini L, Montesi A. Reflux esophagitis: radiologic-endoscopic correlation in 39 symptomatic cases. *Gastrointest Radiol* 1983; 8:1–6
- Martin-Harris B, Brodsky MB, Michel Y, et al. MBS measurement tool for swallow impairment: MBSImp—establishing a standard. *Dysphagia* 2008; 23:392–405
- Dodds WJ, Logemann JA, Stewart ET. Radiologic assessment of abnormal oral and pharyngeal phases of swallowing. *AJR* 1990; 154:965–974
- Gordon AR, Levine MS, Redfern RO, Rubesin SE, Laufer I. Cervical esophageal webs: association with gastroesophageal reflux. *Abdom Imaging* 2001; 26:574–577
- Rubesin SE. Structural abnormalities of the pharynx. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*, 4th ed. Philadelphia, PA: Elsevier, 2015:237–265
- Maglinte DD, Lappas JC, Chernish SM, Anger RT, Miller RE. Flow artifacts in double-contrast esophagography. *Radiology* 1985; 157:535–536
- Herlinger H, Grossman R, Laufer I, Kressel HY, Ochs RH. The gastric cardia in double-contrast study: its dynamic image. *AJR* 1980; 135:21–29
- Levine MS, Rubesin SE. Radiologic investigation of dysphagia. *AJR* 1990; 154:1157–1163
- Freeny PC, Marks WM. Adenocarcinoma of the gastroesophageal junction: barium and CT examination. *AJR* 1982; 138:1077–1084
- Levine MS, Laufer I, Thompson JJ. Carcinoma of the gastric cardia in young people. *AJR* 1983; 140:69–72
- Levine MS. Examination of the esophagus, stomach, and duodenum: techniques and normal anatomy. In: Levine MS, Ramchandani P, Rubesin SE, eds. *Practical fluoroscopy of the GI and GU tracts*. Cambridge, UK: Cambridge University Press, 2012:23–39
- Ott DJ, Chen YM, Hewson EG, et al. Esophageal motility: assessment with synchronous video tape fluoroscopy and manometry. *Radiology* 1989; 173:419–422
- Ott DJ, Levine MS. Motility disorders of the esophagus. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*, 4th ed. Philadelphia, PA: Elsevier, 2015:279–290
- Chen YM, Ott DJ, Gelfand DW, Munitz HA. Multiphasic examination of the esophagogastric region for strictures, rings, and hiatal hernia: evaluation of the individual techniques. *Gastrointest Radiol* 1985; 10:311–316
- Ott DJ, Chen YM, Wu WC, Gelfand DW, Munitz HA. Radiographic and endoscopic sensitivity in detecting lower esophageal mucosal ring. *AJR* 1986; 147:261–265
- Hsu WC, Levine MS, Rubesin SE. Overlap phenomenon: a potential pitfall in the radiographic detection of lower esophageal rings. *AJR* 2003; 180:745–747
- Thompson JK, Koehler RE, Richter JE. Detection of gastroesophageal reflux: value of barium studies compared with 24-hr pH monitoring. *AJR* 1994; 162:621–626
- Blumhagen JD, Christie DL. Gastroesophageal reflux in children: evaluation of the water siphon test. *Radiology* 1979; 131:345–349
- Campbell C, Levine MS, Rubesin SE, Laufer I, Redfern G, Katzka DA. Association between esophageal dysmotility and gastroesophageal reflux on barium studies. *Eur J Radiol* 2006; 59:88–92
- Simeone JF, Burrell M, Toffler R, Smith GJ. Aperistalsis and esophagitis. *Radiology* 1977; 123:9–14
- Recht MP, Levine MS, Katzka DA, Reynolds JC, Saul SH. Barrett's esophagus in scleroderma: increased prevalence and radiographic findings. *Gastrointest Radiol* 1988; 13:1–5
- Ellis H. Diaphragmatic hernia: a diagnostic challenge. *Postgrad Med J* 1986; 62:325–327
- Levine MS. Abnormalities of the gastroesophageal junction. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*, 4th ed. Philadelphia, PA: Elsevier, 2015:438–448
- Canon CL, Morgan DE, Einstein DM, Herts BR, Hawn MT, Johnson LF. Surgical approach to gastroesophageal reflux disease: what the radiologist needs to know. *RadioGraphics* 2005; 25:1485–1499
- Dibble C, Levine MS, Rubesin SE, Laufer I, Katzka DA. Detection of reflux esophagitis on double-contrast esophagrams and endoscopy using the histologic findings as the gold standard. *Abdom Imaging* 2004; 29:421–425
- Hu C, Levine MS, Laufer I. Solitary ulcers in reflux esophagitis: radiographic findings. *Abdom Imaging* 1997; 22:5–7
- Blesman MH, Banner MP, Johnson RC, DeFord JW. The inflammatory esophagogastric polyp and fold. *Radiology* 1978; 128:589–593
- Styles RA, Gibb SP, Tarshis A, Silverman ML, Scholz FJ. Esophagogastric polyps: radiographic and endoscopic findings. *Radiology* 1985;

Consensus Statement on Barium Esophagography in GERD

- 154:307–311
45. Levine MS, Moolten DN, Herlinger H, Laufer I. Esophageal intramural pseudodiverticulosis: a re-evaluation. *AJR* 1986; 147:1165–1170
46. Gupta S, Levine MS, Rubesin SE, Katzka DA, Laufer I. Usefulness of barium studies for differentiating benign and malignant strictures of the esophagus. *AJR* 2003; 180:737–744
47. Ott DJ, Gelfand DW, Wu WC, Castell DO. Esophagogastric junction and its rings. *AJR* 1984; 142:281–287
48. Luedtke P, Levine MS, Rubesin SE, Weinstein DS, Laufer I. Radiologic diagnosis of benign esophageal strictures: a pattern approach. *RadioGraphics* 2003; 23:897–909
49. Agha FP. Esophageal involvement in Zollinger-Ellison syndrome. *AJR* 1985; 144:721–725
50. Levine MS, Fisher AR, Rubesin SE, Laufer I, Herlinger H, Rosato EF. Complications after total gastrectomy and esophagojejunostomy: radiologic evaluation. *AJR* 1991; 157:1189–1194
51. Waldman I, Berlin L. Strictures of the esophagus due to nasogastric intubation. *AJR* 1965; 94:321–324
52. White SB, Levine MS, Rubesin SE, Spencer GS, Katzka DA, Laufer I. The small-caliber esophagus: radiographic sign of idiopathic eosinophilic esophagitis. *Radiology* 2010; 256:127–134
53. Levine MS, Goldstein HM. Fixed transverse folds in the esophagus: a sign of reflux esophagitis. *AJR* 1984; 143:275–278
54. Samadi F, Levine MS, Rubesin SE, Katzka DA, Laufer I. Feline esophagus and gastroesophageal reflux. *AJR* 2010; 194:972–976
55. Levine MS, Halvorsen RA. Carcinoma of the esophagus. In: Gore RM, Levine MS, eds. *Textbook of gastrointestinal radiology*, 4th ed. Philadelphia, PA: Elsevier, 2015:366–393
56. Shaheen NJ, Falk GW, Iyer PG, Gerson LB. ACG clinical guideline: diagnosis and management of Barrett's esophagus. *Am J Gastroenterol* 2016; 111:30–50
57. Levine MS, Kressel HY, Caroline DF, Laufer I, Herlinger H, Thompson JJ. Barrett esophagus: reticular pattern of the mucosa. *Radiology* 1983; 147:663–667
58. Gilchrist AM, Levine MS, Carr RF, et al. Barrett's esophagus: diagnosis by double-contrast esophagography. *AJR* 1988; 150:97–102
59. Brady AP, Stevenson GW, Somers S, Hough DM, Di Giandomenico E. Premature contraction of the cricopharyngeus: a new sign of gastroesophageal reflux disease. *Abdom Imaging* 1995; 20:225–229
60. Hunter JG, Trus TL, Branum GD, Waring JP, Wood WC. A physiologic approach to laparoscopic fundoplication for gastroesophageal reflux disease. *Ann Surg* 1996; 223:673–685; discussion, 685–687
61. Ravi N, Al-Sarraf N, Moran T, et al. Acid normalization and improved esophageal motility after Nissen fundoplication: equivalent outcomes in patients with normal and ineffective esophageal motility. *Am J Surg* 2005; 190:445–450