Quality indicators for upper GI endoscopy

EGD is a minimally invasive, generally safe, and well-tolerated procedure to diagnose and treat disorders of the esophagus, stomach, and duodenum. The rate of EGDs performed in adults is rising across age groups, with database analyses estimating 7.5 million EGDs performed in adults in the United States in 2019, an increase from 6.1 million EGDs in 2013.[1,2] The expanding list of accepted indications for EGD include evaluation and/or management of dysphagia, odynophagia, gastroesophageal reflux symptoms, upper abdominal symptoms, and GI bleeding; screening, surveillance, and endoscopic management of preneoplastic conditions; and newer treatment modalities in endobariatrics and third-space endoscopy. At the same time, EGD is used without an appropriate indication in 5% to 49% of cases, highlighting a clinical challenge and priority area.[3] Potential risks of EGD include bleeding, infection, perforation (≤.30 per 10,000 EGDs performed), emergency department visits and/or hospital admission, and death (.11 per 10,000 EGDs performed).[2]

Delivery of high-quality care in EGD is essential. Generally, a high-quality EGD is one that is clearly indicated, during which relevant diagnoses are established or excluded, any therapy that is provided is appropriate and effective, and harm is minimized to the greatest extent possible.[4-6] Quality indicators are a method to assess performance of quality in EGD and can be divided into 3 categories: structural measures, assessing characteristics of the entire healthcare environment (eg, availability and maintenance of endoscopy equipment at a hospital); process measures, assessing performance during the delivery of care (eg, proportion of patients who receive endoscopic treatment to ulcers with high-risk stigmata of bleeding); and outcome measures, assessing the results of the care that is provided (eg, proportion of patients recommended to undergo treatment and assessment for eradication in the case of endoscopically diagnosed *Helicobacter pylori* infection). By developing quality indicators with performance targets, measuring performance on these benchmarks, and implementing interventions to improve performance, the quality of care delivered at the endoscopist-, practice-, and field-level can be maximized. This document presents quality indicators with performance targets in EGD that, to the greatest extent possible, integrate new data relevant to existing quality indicators and introduces new indicators as appropriate based on interval progress in the field.

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METHODS

This work represents the third iteration of the American Society for Gastrointestinal Endoscopy and American College of Gastroenterology quality indicators document pertaining to EGD. The first version of this document was published by the American Society for Gastrointestinal Endoscopy and American College of Gastroenterology Task Force on Quality in Endoscopy in 2006[7,8] and was revised in 2015.[5,6] This current revision integrates new data relevant to existing quality indicators and introduces new indicators as appropriate based on interval progress in the field. This document focuses on quality indicators unique to EGD (Table 1). The indicators that are common to all GI endoscopic procedures are presented in detail in a separate article[9] and, for completeness, are also listed in Table 2. These common indicators are addressed herein only insofar as the discussion needs to be modified specifically to relate to EGD.

As in preceding versions, we prioritized indicators that have wide-ranging clinical implications, are associated with variation in practice and outcomes, and have been validated in clinical studies. Of note, niche areas in EGD, including endobariatrics and third-space endoscopy, were not within the scope of this quality indicator document. When supportive data were absent, indicators of particular clinical importance were chosen by expert consensus. Substantial progress has been made in the last decade in our ability to measure performance; however, feasibility and efficiency challenges remain. Nevertheless, the task force elected to include a limited number of highly relevant but not yet easily measurable indicators to promote their eventual adoption.

As in previous documents, quality indicators are divided into 3 time periods: preprocedure, intraprocedure, and postprocedure. Additionally, each quality indicator is classified as an outcome or process measure. Although outcome measures are generally considered more impactful toward improving quality of care, some can be difficult or impossible to measure in routine clinical practice because of the need for large amounts of data and/or long-term follow-up

TABLE 1. Quality indicators for EGD

	Quality indicator	Performance target (%)	Type of measure	Level of evidence
Preprocedure				
1	Frequency with which endoscopy is performed for an indication that is included in a published standard list of appropriate indications and the indication is documented	>95	Process	1C+
2	Frequency of EGD performed within 24 hours for patients admitted to or under observation in hospital for upper GI bleeding	>80	Process	1C
Intraprocedure				
3*	Frequency of photodocumentation of the esophagus, gastroesophageal junction, gastric cardia/fundus, corpus, incisura, antrum/pylorus, second portion of duodenum, and detected lesions in patients undergoing EGD	>90	Process	3
4	Frequency of obtaining a total of 6 biopsy samples (or more) obtained from at least 2 levels (proximal/mid and distal) of the esophagus in the absence of an endoscopically evident etiology for dysphagia in patients reporting dysphagia	>90	Process	2B
5	Frequency of endoscopic reference score documentation when eosinophilic esophagitis is suspected or established	>95	Process	2C
6*	Frequency of Los Angeles classification documentation when erosive esophagitis is present	>98	Process	2C
7	Frequency with which the locations of the squamocolumnar junction, gastroesophageal junction, and diaphragmatic hiatus (if there is a hiatal hernia present) are recorded for patients with endoscopically suspected columnar metaplasia in the tubular esophagus	>95	Process	1C+
8*	Frequency with which the presence of at least 1 cm of endoscopically evident columnar mucosa is documented while obtaining biopsy samples to evaluate for BE	>95	Process	1C
9*	Frequency with which the extent of suspected or confirmed BE is documented using the Prague criteria in cases of suspected or confirmed BE	>95	Process	1C+
10	Frequency with which high-definition white-light endoscopy (with dye-based or virtual chromoendoscopy) is used for performing surveillance endoscopy in patients with BE	>90	Process	1B
11	Frequency of systematic 4-quadrant biopsy sampling every 2 cm taken throughout the extent of the endoscopically involved segment of BE in patients with known BE undergoing surveillance endoscopy	>90	Process	1C
12	Frequency with which biopsy samples or endoscopic resection is obtained from visible lesions and processed separately from the systematic biopsy samples in a patient with known BE with a visible lesion identified on surveillance endoscopy	>90	Process	3
13*	Frequency with which, during EGD examination revealing peptic ulcers, at least 1 of the following stigmata is noted: active bleeding, nonbleeding visible vessels (pigmented protuberance), adherent clot, flat spot, or clean based	>98	Process	1A
14*	Frequency of endoscopic treatment delivered to ulcers with active spurting or oozing or with nonbleeding visible vessels	>90	Process	1A
15	Frequency of a second treatment modality delivered (eg, coagulation, clips, argon plasma) when epinephrine injection is used to treat actively bleeding or nonbleeding visible vessels in patients with bleeding peptic ulcers	>98	Process	1A
16*	Frequency with which achievement of primary hemostasis in cases of attempted hemostasis of nonvariceal upper GI bleeding lesion is documented	>90	Outcome	2A
17	Frequency with which gastric biopsy sampling is done or follow-up endoscopy is planned to exclude malignancy in patients with gastric ulcers	>80	Process	2C
18*	Frequency of systematic biopsy sampling of the gastric corpus, antrum, and incisura in patients with known GPMCs, patients at high-risk for gastric cancer, or patients with an endoscopic appearance concerning for GPMCs	>90	Process	2C
19	Frequency with which high-definition white-light endoscopy and virtual chromoendoscopy is used in patients with known GPMCs, patients at high-risk for gastric cancer, or patients with an endoscopic appearance concerning for GPMCs	>90	Process	2C
20	Frequency with which gastric polyps (without the typical appearance of a fundic gland polyp) >10 mm in size undergo biopsy sampling or are resected	>80	Process	2C
21	Frequency with which ≥4 duodenal biopsy samples (including 1 from the bulb) are obtained in patients with suspected celiac disease	>98	Process	1C

(*continued on the next page*)

TABLE 1. Continued

	Quality indicator	Performance target (%)	Type of measure	Level of evidence
Postprocedure				
22	Frequency of repeat endoscopy recommendation after a course of acid suppression in cases of Los Angeles grade C or D erosive esophagitis	>90	Process	2B
23	Frequency of acid suppression therapy recommendation for patients who underwent dilation for peptic esophageal strictures and do not have allergy or other contraindication to these medications	>98	Process	2B
24	Frequency with which follow-up surveillance endoscopy is recommended no sooner than 3 years if systematic surveillance biopsy sampling was performed in a patient known to have nondysplastic BE without prior history of dysplasia	>80	Process	2C
25	Frequency of achieving complete eradication of intestinal metaplasia within 18 months of initial endoscopic treatment in patients with BE and dysplasia or intramucosal carcinoma undergoing endoscopic eradication therapy	>75	Outcome	1C+
26*	Frequency of administering high-dose proton pump inhibitor therapy (continuous or intermittently for 3 days) after successful endoscopic hemostatic therapy of a bleeding ulcer in patients without allergy or contraindication to the medication	>95	Process	1A
27	Frequency with which plans to test for *Helicobacter pylori* infection are documented in patients with GPMCs, peptic ulcer disease, and other *H pylori*–associated conditions	>95	Process	2C
28	Frequency with which plans to treat and assess eradication of *H pylori* infection are documented in patients with endoscopically diagnosed *H pylori*	>95	Outcome	2A
29	Frequency that the GPMC surveillance plan is documented in patients with known GPMCs	>90	Process	2C

Recommendations are graded from 1A to 3 based on methodologic strength supporting the evidence and clarity of benefit. Grade 1A implies a strong recommendation that can be applied to most clinical setting, grade 1B implies a strong recommendation that is likely to be applied to most practice settings, and grade 1C+ implies a strong recommendation that can apply to most practice settings in most situations. Grade 1C implies an intermediate-strength recommendation that may change when stronger evidence is available. Grade 2A implies an intermediate-strength recommendation in which the best action may differ depending on circumstances or patients' or societal values. Grade 2B implies a weak recommendation in which alternative approaches may be better under some circumstances. Grade 2C implies a weak recommendation in which alternative approaches are likely to be better under some circumstances. Grade 3 implies a weak recommendation likely to change as data become available.
BE, Barrett's esophagus; *GPMC*, gastric premalignant condition.
*Indicates a priority indicator.

and because their measurement or interpretation may be confounded by other factors. In such cases, process indicators are provided as surrogate measures of high-quality endoscopic practice. The relative value of a process indicator hinges on the evidence that supports its association with a clinically relevant outcome, and such process measures were emphasized. The measures in this document pertain directly to endoscopic care. Of course, the quality of care delivered to patients is influenced by additional factors, including those related to the facilities in which endoscopy is performed. These structural measures are covered in a separate article dedicated to unit-level quality.[10]

For this revision, the task force critically appraised existing quality indicators and determined whether to maintain or remove them by consensus based on several factors, including ongoing relevance and strength of evidence. Additionally, new quality indicators were proposed by task force members and, if appropriate, adopted by consensus based on similar considerations. For each indicator, relevant articles were identified by the authors through a systematic search of PubMed (U.S. National Library of Medicine, National Institutes of Health) from January 2014, which was the date of the last update of this document, through May 2023. The search strategies for each indicator included a combination of subject headings (MeSH in PubMed) and pertinent key words. English language restrictions were applied. To identify additional articles, the authors reviewed PubMed's "similar articles" and manually searched reference lists of relevant articles. Search strategies for the selected indicators were facilitated by health science librarians with expertise in systematic review. Based on literature review, the strength of recommendation for each indicator was evaluated according to a previously used framework (Table 3). Within this framework, the strength of each quality indicator was divided across a spectrum from "1A," denoting a strong quality indicator that can be applied to most clinical settings, to "3," denoting a weak quality indicator because of absence of evidence requiring reliance on expert opinion only. The strength of recommendation grade for each indicator was established by consensus of the authors.

The process measures included in this document are attached to a performance target; therefore, each measure is considered a quality indicator. The task force selected performance targets based on published benchmarking data, informed by literature review. In the absence of available data, when expert consensus considered the failure to perform a given quality indicator a "never event," such as failure to monitor vital signs during sedation, the performance target was expressed as >98%, because only in exceptional circumstances would the quality indicator not be fulfilled.

It is important to emphasize that the included quality indicators and associated performance targets do not

TABLE 2. Quality indicators common to all endoscopic procedures with associated performance targets

Quality indicator	Grade of recommendation	Measure type	Performance target (%)
Preprocedure			
1. Frequency with which endoscopy is performed for an indication that is included in a published standard list of appropriate indications and the indication is documented (priority indicator)	1C+	Process	>95
2. Frequency with which informed consent is obtained and documented	3	Process	>98
3. Frequency with which preprocedure history and directed physical examination are performed and documented	3	Process	>98
4. Frequency with which a sedation plan that includes risk for sedation-related adverse events is documented before sedation is initiated	3	Process	>98
5. Frequency with which prophylactic antibiotics are administered for appropriate indications (priority indicator)	Varies	Process	>98
6. Frequency with which management of antithrombotic therapy is formulated and documented before the procedure (priority indicator)	3	Process	>95
7. Frequency with which a team pause is performed and documented	3	Process	>98
8. Frequency with which endoscopy is performed or supervised by an individual who is fully trained and appropriately credentialed to perform that particular procedure	3	Process	>98
Intraprocedure			
9. Frequency with which photodocumentation is performed	3	Process	>90
10. Frequency with which patient monitoring during sedation is performed and documented	3	Process	>98
11. Frequency with which procedure interruption and premature termination because of sedation-related issues is documented	3	Process	>98
12. Frequency with which endoscopic specimen verification is performed and documented	3	Process	>98
Postprocedure			
13. Frequency with which discharge from the endoscopy unit according to predetermined discharge criteria is documented	3	Process	>98
14. Frequency with which patient instructions are provided	3	Process	>98
15. Frequency with which endoscopic findings, pathology results, and follow-up recommendations are communicated to the patient and appropriate providers	3	Process	>98
16. Frequency with which a complete procedure report is created	3	Process	>98
17. Frequency with which adverse events are documented (priority indicator)	3	Process	>98
18. Frequency with which adverse events occur	Varies	Outcome	N/A
19. Frequency with which patient satisfaction data are collected	N/A	Outcome	N/A

Recommendations are graded from 1A to 3 based on methodologic strength supporting the evidence and clarity of benefit. Grade 1A implies a strong recommendation that can be applied to most clinical setting, grade 1B implies a strong recommendation that is likely to be applied to most practice settings, and grade 1C+ implies a strong recommendation that can apply to most practice settings in most situations. Grade 1C implies an intermediate-strength recommendation that may change when stronger evidence is available. Grade 2A implies an intermediate-strength recommendation in which best action may differ depending on circumstances or patients' or societal values. Grade 2B implies a weak recommendation in which alternative approaches may be better under some circumstances. Grade 2C implies a weak recommendation in which alternative approaches are likely to be better under some circumstances. Grade 3 implies a weak recommendation likely to change as data become available.
N/A, Not applicable.

necessarily reflect the standard of care, credentialing requirements, or training standards, and it is a misapplication to use any of the quality indicators in this document as such. Rather, the quality indicators are designed and intended to serve as a framework for quality improvement efforts. To guide continual improvement for endoscopists and units in varying phases of their quality journey, the task force again recognized a subset of indicators deemed "priority indicators," based on their clinical relevance and importance, evidence that performance varies significantly in clinical practice, and feasibility of measurement (a function of the number of procedures needed to obtain an accurate measurement with narrow confidence intervals [CIs] and the ease of measurement). We believe that quality improvement efforts should initially focus on priority indicators and then progress to include other indicators once it is ascertained that endoscopists are performing above recommended thresholds, either at baseline or after corrective interventions.

PREPROCEDURE QUALITY INDICATORS

The preprocedure period begins at the time of first contact between the patient and members of the endoscopy team and ends at the time of administration of sedation

TABLE 3. Grades of recommendation

Grade	Clarity of benefit	Methodologic strength supporting evidence	Implications
1A	Clear	Randomized trials without important limitations	Strong recommendation, can be applied to most clinical settings
1B	Clear	Randomized trials with important limitations (inconsistent results, nonfatal methodologic flaws)	Strong recommendation, likely to apply to most practice settings
1C+	Clear	Overwhelming evidence from observational studies	Strong recommendation, can apply to most practice settings in most situations
1C	Clear	Observational studies	Intermediate-strength recommendation, may change when stronger evidence is available
2A	Unclear	Randomized trials without important limitations	Intermediate-strength recommendation, best action may differ depending on circumstances or patients' or societal values
2B	Unclear	Randomized trials with important limitations (inconsistent results, nonfatal methodologic flaws)	Weak recommendation, alternative approaches may be better under some circumstances
2C	Unclear	Observational studies	Very weak recommendation, alternative approaches are likely to be better under some circumstances
3	Unclear	Expert opinion only	Weak recommendation, likely to change as data become available

Adapted from Guyatt G, Sinclair J, Cook D, et al. Moving from evidence to action. Grading recommendations—a qualitative approach. In: Guyatt G, Rennie D, editors. Users' guides to the medical literature. Chicago, IL: AMA Press; 2002. p. 599-608.

(or insertion of the endoscope in unsedated procedures). Preprocedure quality indicators common to all endoscopic procedures are detailed in a separate article[9] and encompass appropriate indication, informed consent, preprocedure history and physical examination, sedation plan, management of prophylactic antibiotics and antithrombotic therapy, and team pause (Table 2).

1. *Frequency with which endoscopy is performed for an indication that is included in a published standard list of appropriate indications and the indication is documented*
   Strength of recommendation: 1C+
   Performance target: >95%
   Type of measure: Process
   Table 4 lists general indications for EGD evaluation and/or management of upper gastroenterologic conditions as well as a list of indications for which EGD is generally not indicated. For every EGD, an appropriate indication should be documented. When a procedure is performed for a nonstandard reason, the rationale for the procedure should be justified in the documentation. In general, endoscopy is indicated when the information gained or the therapy provided will improve patient outcomes and the benefits clearly outweigh any potential harms. Studies have shown that when EGD is done for appropriate indications, significantly more clinically relevant diagnoses are made.[11,12]

2. *Frequency of EGD performed within 24 hours for patients admitted to or under observation in the hospital for upper GI bleeding (UGIB)*
   Strength of recommendation: 1C
   Performance target: >80%
   Type of measure: Process

Discussion. EGD is recommended to be performed within 24 hours of presentation for patients admitted or under observation with overt UGIB. This recommendation excludes patients who are considered low risk for a hospital-based intervention (Glasgow-Blatchford score $\leq$1) and/or may be discharged early from the emergency department. The recommendation to perform EGD within 24 hours stems from the potential economic benefit driven by reduced length of stay and a mortality benefit and reduction in need for surgery seen in several observational studies.[13] Observational studies suggest that endoscopy within 1 day of admission is associated with a shorter length of stay, with a possible risk reduction of requiring surgery,[14,15] and mortality.[16-18] In a large, nationwide, retrospective cohort study of ulcer bleeding, there was an increased mortality risk in high-risk patients (American Society of Anesthesiologists score of III to IV) with very early (<12 hours) or very late (>36 hours) endoscopy.[19] EGD within 6 to 24 hours had a mortality benefit compared with both urgent EGD (<6 hours) and delayed EGD (24-48 hours) in a territory-wide cohort study of 6474 patients with UGIB.[20] Finally, urgent endoscopy within 6 hours of presentation has not been shown to be beneficial, as a large randomized controlled trial (RCT) compared outcomes of urgent EGD in acute UGIB within 6 hours with within 6 to 24 hours of presentation in high-risk patients with UGIB (Glasgow-Blatchford score $\geq$12), with no difference seen in 30-day mortality or further bleeding.[21]

INTRAPROCEDURE QUALITY INDICATORS

The intraprocedure period extends from the administration of sedation, or insertion of the endoscope when no

TABLE 4. Indications for upper GI endoscopy

EGD is generally indicated for evaluating and/or managing
Upper abdominal symptoms, which persist despite an appropriate trial of therapy
Upper abdominal symptoms associated with other symptoms or signs suggesting serious organic disease (eg, anorexia and weight loss) or in patients aged >45 y
Dysphagia or odynophagia or food impaction
Esophageal reflux symptoms (heartburn, regurgitation, noncardiac chest pain), which are persistent or recurrent despite appropriate therapy
Esophageal physiology using impedance planimetry
Placement of wireless pH capsule for esophageal or extraesophageal reflux symptoms in absence of severe erosive reflux disease
Screening for Barrett's esophagus
Persistent vomiting of unknown cause
Other diseases in which the presence of upper GI pathology might modify other planned management, eg, patients with a history of ulcer or GI bleeding scheduled for organ transplantation, long-term anticoagulation, or chronic nonsteroidal anti-inflammatory drug therapy for arthritis and those with cancer of the head and neck
Familial adenomatous polyposis syndromes
For confirmation and specific histologic diagnosis of radiologically demonstrated lesions • Suspected neoplastic lesion • Gastric or esophageal ulcer • Upper tract stricture or obstruction
GI bleeding • In patients with active or recent bleeding • For presumed chronic blood loss and for iron deficiency anemia when the clinical situation suggests an upper GI source or when colonoscopy result is negative
When sampling of tissue or fluid is indicated
In patients with suspected portal hypertension to document or treat esophageal varices
To assess acute injury after caustic ingestion
Treatment of bleeding lesions such as ulcers, tumors, and vascular abnormalities (eg, electrocoagulation, heater probe, laser photocoagulation, or injection therapy)
Banding or sclerotherapy of varices
Removal of foreign bodies
Removal of selected polypoid lesions
Placement of feeding or drainage tubes (peroral, PEG, or percutaneous endoscopic jejunostomy)
Management of stenotic lesions (eg, with transendoscopic balloon dilators or dilation systems by using guidewires)
Management of achalasia (eg, endoscopic myotomy, botulinum toxin, balloon dilation)
Palliative treatment of stenosing neoplasms (eg, laser, multipolar electrocoagulation, stent placement)
Endoscopic therapy for intestinal metaplasia
Management of diverticular lesions (eg, endoscopic myotomy)
Endoscopic bariatric therapy
Intraoperative evaluation of anatomic reconstructions typical of modern foregut surgery (eg, evaluation of anastomotic leak and patency, fundoplication formation, pouch configuration during bariatric surgery)
Management of operative adverse events (eg, dilation of anastomotic strictures, stent placement of anastomotic disruption, fistula, or leak in selected circumstances)
Sequential or periodic EGD may be indicated • Surveillance for malignancy in patients with premalignant conditions (ie, Barrett's esophagus, gastric intestinal metaplasia) • Assessment of disease activity in eosinophilic esophagitis or eosinophilic GI diseases • Assessment of mucosal healing and screening for Barrett's esophagus in patients with severe erosive esophagitis
EGD is generally not indicated for evaluating
Symptoms that are considered functional in origin (exceptions include endoscopic examination done once to rule out organic disease, especially if symptoms are unresponsive to therapy)
Metastatic adenocarcinoma of unknown primary site when the results will not alter management

(*continued on the next page*)

TABLE 4. Continued

Radiographic findings of • Asymptomatic or uncomplicated sliding hiatal hernia • Uncomplicated duodenal ulcer that has responded to therapy • Deformed duodenal bulb when symptoms are absent or respond adequately to ulcer therapy
Sequential or periodic EGD is generally not indicated for • Surveillance of healed benign disease such as esophagitis or gastric or duodenal ulcer • Surveillance during repeated dilations of benign strictures unless there is a change in status

sedation is given, until the endoscope is removed. This period includes all technical aspects of the endoscopy and cognitive decision-making required for successful and safe completion of the procedure. Intraprocedure quality indicators pertinent to all endoscopic procedures are detailed in a separate article[9] and mainly regard patient monitoring during sedation and anesthesia and specimen verification (Table 2). The intraprocedure quality indicators in EGD regard photodocumentation and indicators specific to esophageal biopsy sampling for dysphagia, documentation of Los Angeles (LA) classification in erosive esophagitis, screening and surveillance for Barrett's esophagus (BE), evaluation and management of peptic ulcers and UGIB, surveillance for gastric premalignant conditions, evaluation and management of gastric polyps, and evaluation for celiac disease.

3. *Frequency of photodocumentation of the esophagus, gastroesophageal junction (GEJ), gastric cardia/fundus, corpus, incisura, antrum/pylorus, second portion of the duodenum, and detected lesions in patients undergoing EGD (Priority Indicator)*
   Strength of recommendation: 3
   Performance target: >90%
   Type of measure: Process

Discussion. EGD is generally undertaken with the intent to inspect the esophagus beginning at the upper esophageal sphincter, stomach (including a retroflexed view of the gastric cardia), and up to the second portion of the duodenum (if applicable based on patient's anatomy). Intubation of the second portion of the duodenum is defined as passage of the endoscope tip to a point distal to the ampulla. Although there is little evidence to support the practice of photodocumentation in EGD, it is intuitive that this practice is a surrogate metric for mucosal cleaning, mucosal inspection including endoscopist blind spots, and a complete endoscopic examination and serves as a reference in serial examinations and lesions referred for endoscopic resection.[22] Photodocumentation is a well-established key quality indicator in colonoscopy and in more recent years is recommended in EGD by organizations worldwide including the European Society of Gastrointestinal Endoscopy guidelines as well as the World Endoscopy Organization in EGD.[23-25] Systematic photodocumentation of the following segments and landmarks is recommended to signify a complete examination: esophagus, antegrade view of the GEJ, retroflexed view of the gastric cardia/fundus, antegrade view of the gastric antrum/pylorus, antegrade view of the gastric incisura, and second portion of the duodenum (Fig. 1). The GEJ is defined as either top of the gastric folds or distal end of the lower esophageal palisade vessels where the veins merge with the submucosal venous system. Regions with a higher incidence of gastric cancers recommend up to 22 images of the stomach to optimize early diagnosis. A quality improvement study demonstrated a significant increase in the rate of photodocumentation by segment following a simple training program.[26,27]

4. *Frequency of obtaining a total of 6 biopsy samples (or more) taken from at least 2 levels (proximal/mid and distal) of the esophagus in the absence of an endoscopically evident etiology for dysphagia in patients reporting dysphagia*
   Strength of recommendation: 2B
   Performance target: >90%
   Type of measure: Process

Discussion. Eosinophilic esophagitis (EoE), an immune-mediated condition characterized by eosinophilic infiltrate of the esophageal mucosa, is rising in incidence. The current diagnostic standard for EoE is the presence of $\geq$15 eosinophils per high-powered field on esophageal biopsy samples. However, practice patterns vary in evaluation and management of EoE. Involvement of the esophageal epithelium and lamina propria in active and inactive EoE is uneven from the proximal to distal esophagus.[28] Further, studies suggest that to morphologically exclude a diagnosis of reflux esophagitis as the cause of intraepithelial eosinophilia, distal esophageal biopsy samples should be accompanied by more proximal biopsy samples. However, in a cross-sectional survey study of 240 gastroenterologists, only 67% of respondents reported obtaining biopsy samples from 2 levels of the esophagus.[29] In terms of the optimal number of biopsy samples obtained, the diagnostic sensitivity of EoE increases with more biopsy samples obtained. In a study of children, diagnostic sensitivity was 73% for a total of 1, 84% for a total of 2, 97% for a total of 3, and 100% for a total of 6 biopsy samples obtained. Thus, specimens should be submitted from at least 2 regions including the

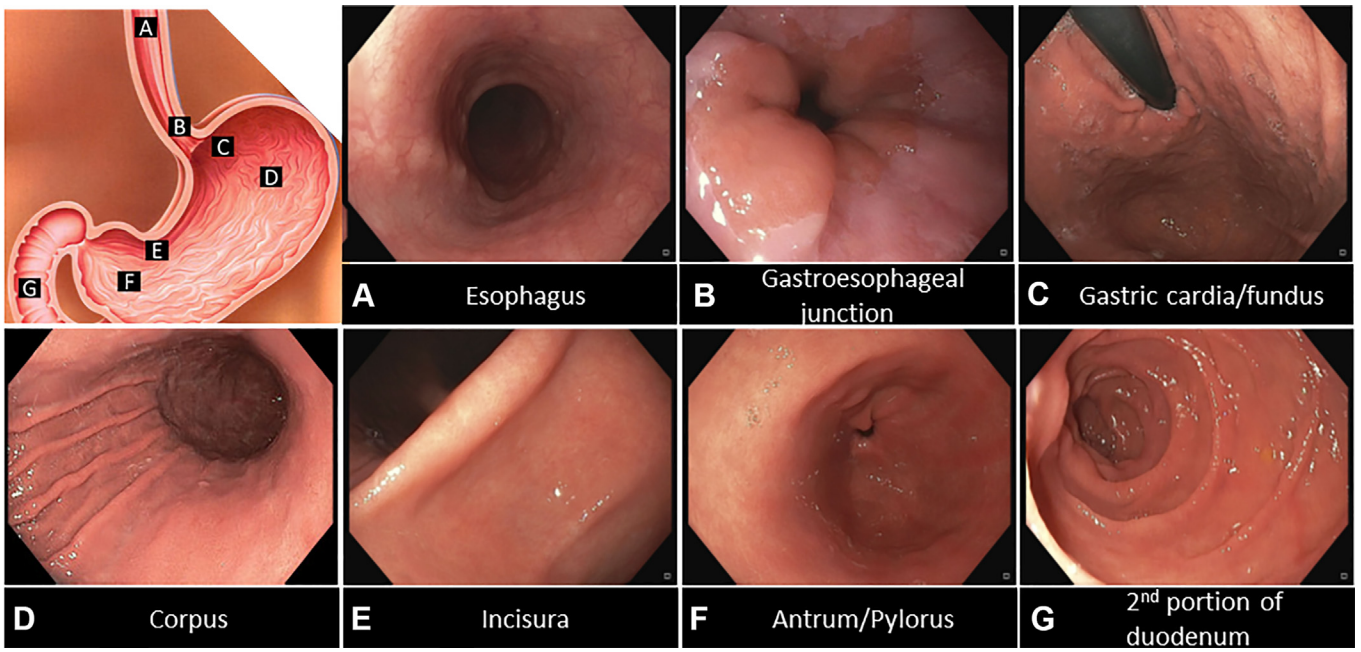

Figure 1. Landmarks for photodocumentation during EGD. **A,** Esophagus; **B,** gastroesophageal junction; **C,** gastric cardia/fundus; **D,** corpus; **E,** incisura; **F,** antrum/pylorus; **G,** second portion of duodenum. (Images courtesy of Center for Esophageal Diseases at University of California San Diego.)

mid and/or proximal esophagus in addition to the distal esophagus for a total of at least 6 biopsy fragments.[30,31]

5. *Frequency of endoscopic reference score documentation when EoE is suspected or established*
   Strength of recommendation: 2C
   Performance target: >95%
   Type of measure: Process

Discussion. Characteristic endoscopic features of active EoE include inflammatory findings such as exudate, loss of vascularity and furrowing, and fibrostenotic findings such as rings and stricture. Multiple studies, including RCTs, demonstrate improvement in endoscopic features after treatment of EoE. However, global endoscopic assessment of EoE activity can vary.[32] The endoscopic reference score (EREFS) is a validated grading system for EoE that grades the degree of *E*dema (loss of vascular markings; grade 0 or 1), *R*ings (trachealization; grade 0-3), *E*xudate (white plaques; grade 0-2), *F*urrows (vertical lines; grade 0-2), and *S*tricture (grade 0 or 1). The EREFS has good interobserver agreement among endoscopists and trainees.[32-34] Further, the EREFS score correlates with histologic activity and symptom response with treatment and is used as a reliable and responsive outcome measure of EoE endoscopic activity.[35,36] In addition, EREFS is used to characterize EoE patients as having inflammatory, fibrostenotic, or both inflammatory and fibrostenotic phenotypes. Therefore, in patients with established EoE and in those with suspected EoE, the EREFS score should be documented in the endoscopic report.

6. *Frequency of LA classification documentation when erosive esophagitis is present (Priority Indicator)*
   Strength of recommendation: 2C
   Performance target: >98%
   Type of measure: Process

Discussion. Mucosal breaks in the distal esophagus can represent a spectrum of disease from normal mucosa to erosive esophagitis. A greater severity of findings is associated with a greater likelihood of pathologic acid exposure in the esophagus and an increased risk for BE. The LA classification system is the most widely used and validated scoring system for erosive esophagitis (Fig. 2).[37] LA grade A esophagitis is not sufficient for a definitive diagnosis of GERD because it cannot always be reliably distinguished from normal and can be seen in 5% to 7.5% of healthy subjects.[37-41] On the other hand, LA grades B, C, or D esophagitis is diagnostic of GERD and does not require further reflux monitoring to confirm a diagnosis of GERD.[42] LA grades B and C esophagitis demonstrate similar acid exposure times on reflux monitoring and symptom response with proton pump inhibitors (PPIs).[39,42,43] LA grade D is associated with the highest acid exposure time on reflux monitoring,[44] and a greater proportion of patients with LA grades C or D esophagitis have been identified to have BE on subsequent endoscopy compared with patients with LA grades A or B.[45] Therefore, documentation of LA classification when erosive esophagitis is encountered has important implication on risk stratification, diagnosis, and management.

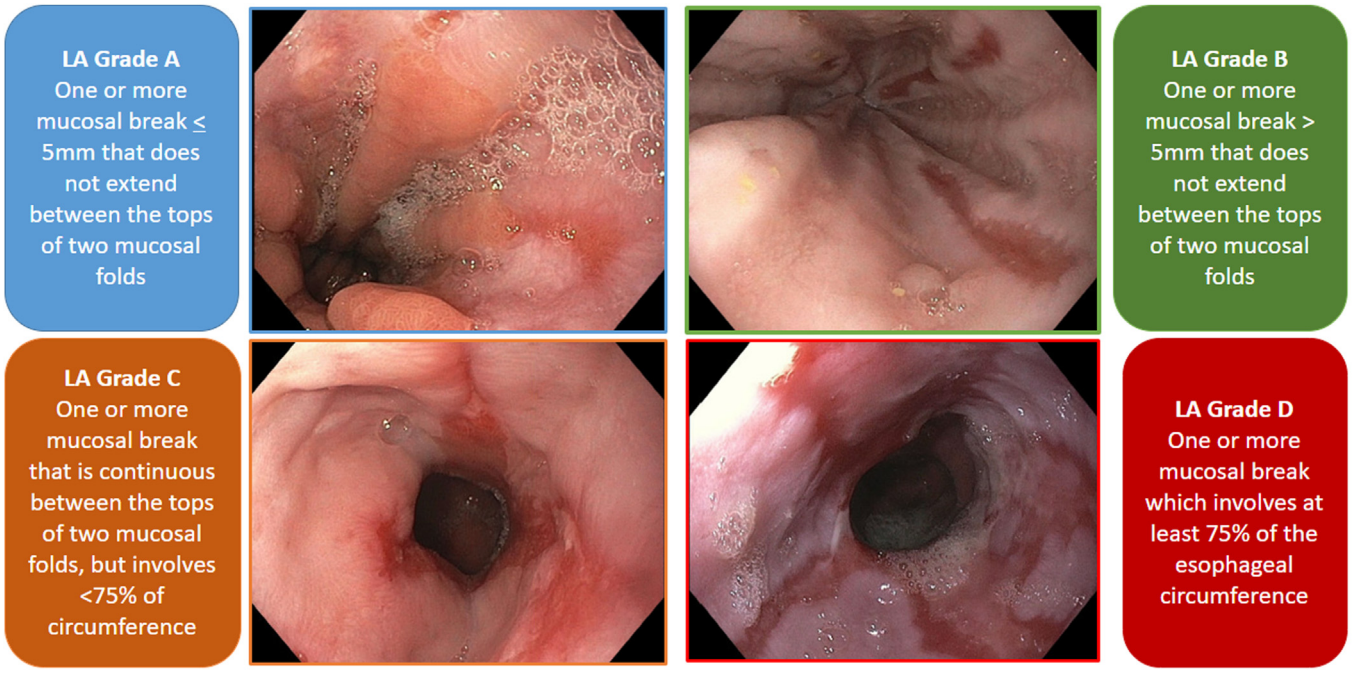

Figure 2. Los Angeles classification scheme for erosive esophagitis. *LA*, Los Angeles.

7. *Frequency with which the locations of the squamocolumnar junction (SCJ), GEJ, and diaphragmatic hiatus (if a hiatal hernia is present) are recorded for patients with endoscopically suspected columnar metaplasia in the tubular esophagus*
   Strength of recommendation: 1C+
   Performance target: >95%
   Type of measure: Process

Discussion. It is crucial to document the locations of the SCJ, GEJ, and diaphragmatic hiatus, particularly in cases where columnar metaplasia is suspected in the tubular esophagus. The SCJ is the morphologic junction of the esophageal squamous mucosa and columnar mucosa, and location is determined in the forward view. The GEJ is the anatomic junction between the tubular esophagus and the proximal stomach, determined by the proximal extent of the gastric folds. The location of the GEJ and SCJ may not coincide endoscopically in cases of columnar metaplasia. The location of the diaphragmatic hiatus is determined by the indentation or the pinch of the crural diaphragm. In a healthy state, the diaphragmatic hiatus will be at the level of the GEJ but may be at the level of the stomach in the setting of a hiatal hernia. Accurate documentation of these landmarks provides valuable information for diagnostic and management purposes for several reasons. First, localization of pathologic changes aids in determining the extent and severity of the disease and in guiding targeted biopsy sampling and treatment strategies.[46-48] Further, clear documentation of the SCJ and GEJ helps differentiate between various conditions involving the esophagus and stomach, such as reflux disease, esophagitis, hiatal hernia, and BE, each of which requires specific management approaches. Additionally, precise recording of the SCJ, GEJ, and diaphragmatic hiatus facilitates consistent surveillance over time, enabling accurate monitoring of disease progression and guiding subsequent interventions.[48] Finally, documenting the locations of these anatomic landmarks enhances consistency, accuracy, and reliability of clinical reports; facilitates effective communication among healthcare providers; allows for easy comparison of findings between endoscopies; and aids in interdisciplinary collaboration.

8. *Frequency with which the presence of at least 1 cm of endoscopically evident columnar mucosa is documented while obtaining biopsy samples to evaluate for BE (Priority Indicator)*
   Strength of recommendation: 1C
   Performance target: >95%
   Type of measure: Process

Discussion. Documenting at least 1 cm of endoscopically evident columnar mucosa is one of the criteria to diagnose a patient with BE (the other being demonstration of intestinal metaplasia [IM] from the esophageal tissue). An irregular Z-line, characterized by the presence of columnar tongues extending <1 cm proximal to the GEJ, has been observed in approximately 10% to 15% of

individuals undergoing upper endoscopy, with about 44% harboring IM.[49,50] However, the risk of progression to high-grade dysplasia (HGD) or esophageal adenocarcinoma (EAC) is exceedingly low for those with a normal or irregular Z-line measuring <1 cm. In a multicenter cohort study involving 1791 patients undergoing BE surveillance, followed for a median of 5.9 years, none of 167 patients with an irregular Z-line (<1 cm) developed HGD or EAC. Interestingly, follow-up biopsy samples in individuals with an irregular Z-line revealed no evidence of IM in 53% of cases.[50] Similarly, in a population-based cohort study of 86 patients with IM of the GEJ over a median follow-up period of 8 years, none progressed to HGD or EAC.[51] In a more recent prospective, single-center study of 166 patients with an irregular Z-line, 39% had IM, 8.8% developed nondysplastic BE, and 1.9% developed low-grade dysplasia (LGD), with no HGD or EAC cases.[52]

Finally, the interobserver agreement, even among expert endoscopists, on diagnosing an irregular Z-line or columnar mucosa <1 cm is extremely poor.[48] These data make it fairly convincing that the risk of disease progression in those with an irregular Z-line is negligible. Thus, regular surveillance would not result in the best use of resources. Therefore, it is imperative to establish clear documentation of at least 1 cm of endoscopically evident columnar mucosa for individuals to enter into BE surveillance. This distinction has significant implications regarding direct and indirect costs and the quality of life for patients and the overall healthcare system.

9. *Frequency with which the extent of suspected or confirmed BE is documented using the Prague criteria in cases of suspected or confirmed BE (Priority Indicator)*
   Strength of recommendation: 1C+
   Performance target: >95%
   Type of measure: Process

Discussion. Twenty-nine experts from 14 countries created the Prague C and M classification to provide a standardized approach to the endoscopic grading of BE.[48] This classification uses the "C" value to represent the "circumferential extent" and the "M" value to indicate the "maximal extent" of BE above the GEJ in centimeters. The Prague classification demonstrated excellent reliability for both circumferential (reliability coefficient, .94; 95% CI, .91-.97) and maximal (reliability coefficient, .93; 95% CI, .89-.96) extent of BE. Its utility has been validated among gastroenterology trainees and community clinicians as well.[53,54]

Several studies have established the utility of this classification in disease progression and risk stratification. The Prague classification system also enables consistent reporting of BE extent in research studies and clinical trials worldwide. This standardization allows for better comparability of results, facilitates meta-analyses and systematic reviews, and contributes to evidence-based decision-making. The Prague classification provides a common language for communication between healthcare providers, endoscopists, and pathologists, facilitating accurate transmission of information. The clear documentation of BE extent using the Prague criteria allows for easier follow-up and monitoring of patients over time, aiding in the evaluation of disease progression and response to treatment.

10. *Frequency with which high-definition white-light endoscopy (HDWLE; with dye-based or virtual chromoendoscopy) is used for performing surveillance endoscopy in patients with BE*
    Strength of recommendation: 1B
    Performance target: >90%
    Type of measure: Process

Discussion. Endoscopic surveillance involves careful inspection of the BE mucosa to detect visible lesions harboring dysplasia or neoplasia, followed by endoscopic therapy to prevent or treat EAC in appropriately selected cases. Endoscopically, most dysplasia is subtle in appearance and patchy in distribution, making detection challenging. Hence, careful inspection of the mucosa using high-definition WLE, after cleansing the mucosa to remove adherent mucus, saliva, or food, is critical. Indeed, longer BE inspection times have been associated with a higher likelihood of identifying visible lesions harboring dysplasia or neoplasia.[55,56] Approximately 1 min/cm of circumferential BE has been suggested as a guide during BE inspection. Given these challenges, dysplasia or neoplasia can be missed in a substantial proportion of patients,[57] and hence strategies to improve dysplasia detection have been studied.

Technologies to improve characterization of the BE mucosa involve spraying the mucosa with dyes (methylene blue, indigo carmine) to enhance the surface characteristics. Spraying the mucosa with acetic acid leads to whitening of the mucosa, which reverses more rapidly in dysplastic mucosa, allowing its identification. Several "virtual" chromoendoscopy technologies integrated into the endoscope platform, such as narrow-band imaging (NBI), flexible spectral imaging color enhancement, and i-SCAN, are also available. These technologies use either optical filters to modify the spectrum of illumination, enhancing the mucosal and vascular pattern, or achieve the same result with postimage acquisition processing. These techniques do not require dye spraying, can be engaged with the press of a button on the endoscope, and are hence convenient to use. Inspecting the mucosa with virtual chromoendoscopy further increases mucosal inspection time, potentially facilitating detection of visible abnormalities.

Classification systems to identify dysplasia have been developed for several of these techniques, and their accuracy compared with WLE have been studied. Performance thresholds for the adoption of these technologies have been defined (American Society for Gastrointestinal Endoscopy Preservation and Incorporation of Valuable Endoscopic Innovations criteria), and systematic reviews and

meta-analyses have demonstrated that NBI and acetic acid chromoendoscopy (in the hands of expert endoscopists) meet these criteria.[58] Evidence of the value of NBI in comparison with high-definition WLE has been presented in 2 randomized trials.[59,60] One trial reported higher dysplasia detection rates with NBI-targeted biopsy sampling, whereas the other trial reported similar findings with NBI needing fewer biopsy samples. Finally, a systematic review and meta-analysis reported[61] that use of either dye-based or virtual chromoendoscopy techniques increased the diagnostic yield of dysplasia and cancer by 34% to 35% (in comparison with biopsy sampling with WLE). Hence, several gastroenterological societies now recommend the use of high-definition WLE with (virtual) chromoendoscopy with high-definition monitors for the inspection of BE mucosa before obtaining biopsy samples.

11. *Frequency of systematic 4-quadrant biopsy sampling every 2 cm taken throughout the extent of the endoscopically involved segment of BE in patients with known BE undergoing surveillance endoscopy*
    Strength of recommendation: 1C
    Performance target: >90%
    Type of measure: Process

Discussion. Multiple gastroenterological societies recommend endoscopic surveillance in patients with BE using the Seattle protocol (4-quadrant biopsy sampling every 2 cm in those without dysplasia and every 1 cm in those with dysplasia electing to undergo surveillance), with biopsy samples at each level placed in a separate jar to enable subsequent identification of focal lesions harboring dysplasia. In patients with noncircumferential BE, quadrantic biopsy sampling every 2 cm from the tongues of columnar mucosa can be obtained, with a minimum of 4 biopsy samples obtained. The use of a systematic biopsy sampling approach has been shown to improve dysplasia detection compared with random biopsy sampling.[62-64]

A systematic review and meta-analysis assessed the effect of compliance with the Seattle protocol on the detection of dysplasia.[65] Compliance with this metric increased the detection of dysplasia by almost 2-fold (relative risk [RR], 1.9; 95% CI, 1.36-2.64; $I^2 = 45\%$) and was impactful for the detection of both LGD and HGD/EAC, raising their odds of detection by 2-fold as well. Another systematic review and meta-analysis found that lower EAC-related and all-cause mortality were associated with regular surveillance (RR, .60 [95% CI, .50-.71]; hazard ratio, .75 [95% CI, .59-.94]) compared with no or infrequent surveillance.[66] This systematic review and meta-analysis also showed lower EAC-related and all-cause mortality among patients with surveillance-detected EAC versus symptom-detected EAC (RR, .73 [95% CI, .57-.94]; hazard ratio, .59 [95% CI, .45-.76]). Surveillance was also associated with detection of EAC at an earlier stage. However, it was observed that the adjustment for lead and length time bias substantially attenuated this benefit.

Unfortunately, compliance with the Seattle protocol has been shown to be suboptimal. One study from a national pathology database reported compliance with guidelines to be only 51%, which further decreased with increasing BE segment length.[67] Additionally, reduced compliance was associated with decreased odds of detecting dysplasia. Another study reported higher compliance rates overall (82%-87%) but reaffirmed increasing BE segment length as the most significant predictor of noncompliance. Additionally, performance of surveillance by nongastroenterologists was also a predictor of noncompliance.[68] This is especially concerning given the strong association of longer segment length with a higher prevalence of dysplasia and higher risk of progression.[69]

12. *Frequency with which biopsy sampling or endoscopic resection is obtained from visible lesions and processed separately from the systematic biopsy sampling in a patient with known BE with a visible lesion identified on surveillance endoscopy*
    Strength of recommendation: 3
    Performance target: >90%
    Type of measure: Process

Discussion. Current recommendations for endoscopic surveillance in BE include careful systematic inspection of the BE mucosa after cleaning the mucosa, with the goal of identifying visible abnormalities, which may harbor dysplasia and adenocarcinoma. Use of the Paris classification of endoscopic visible lesions enables standardization of reporting terminology (similar to the Prague classification).[70] If the lesion does not undergo complete resection at that time, it is recommended that tissue samples from these abnormalities be placed in separate biopsy sample jars with a notation of the location (eg, nodule biopsy sample at 34 cm from the incisors and at the 3 o'clock position or flat hypervascular lesion at 36 cm from the incisors at the 6 o'clock position) to enable subsequent localization and treatment as determined by the histopathology report. This should precede acquisition of biopsy samples every 1 to 2 cm in a 4-quadrant fashion. Studies have reported detection of dysplasia in both "targeted" biopsy samples and those taken "randomly" for the Seattle protocol, with some reporting greater rates of dysplasia detection by Seattle biopsy sampling than in targeted biopsy sampling.[71] Whether this reflects missed endoscopically visible lesions or true flat dysplasia is difficult to determine. Sampling of visible abnormalities can be done by biopsy sampling or endoscopic resection; if subsequent endoscopic resection is anticipated, biopsy samples from these lesions should be carefully targeted and kept to the minimum to avoid biopsy sampling–induced fibrosis. The choice likely depends on available expertise and prior history of confirmed dysplasia (which should prompt consideration of endoscopic resection).

The prevalence of visible abnormalities sampled separately ("targeted" biopsy sampling) in routine surveillance is not well described in the literature. In a population-based study reporting the prevalence of HGD or EAC in 1066 patients undergoing their first surveillance endoscopy, targeted biopsy samples were taken in 54 patients (5%).[71] Of these 54 patients, 26 had nondysplastic BE, 9 had LGD, 9 had HGD, and 10 had EAC. Additionally, these yielded 8% to 50% of all LGD, HGD, and EAC diagnoses, suggesting the continued utility of Seattle protocol biopsy sampling in detecting prevalent dysplasia. A randomized trial comparing HDWLE with NBI reported targeted biopsy sampling in 17% of patients in the HDWLE arm and 5% of patients in the NBI arm.[60] However, it should be noted that this trial was enriched with patients with LGD and HGD, and hence these numbers may not be representative of the general BE surveillance population. Another retrospective study that compared dysplasia yield with and without acetic acid chromoendoscopy in a surveillance population not enriched with dysplastic BE reported a targeted biopsy sampling rate of 2% in the conventional HDWLE group compared with 12.5% in the acetic acid group.[72]

13. *Frequency with which, during EGD examination revealing peptic ulcers, at least 1 of the following stigmata is noted: active bleeding, nonbleeding visible vessels (pigmented protuberance), adherent clot, flat spot, or clean based (Priority Indicator)*
    Strength of recommendation: 1A
    Performance target: >98%
    Type of measure: Process

Discussion. Ulcers seen during an EGD should be classified using the Forrest classification to differentiate between low- and high-risk stigmata for further bleeding.[73] The Forrest classification is defined as follows: FIa, spurting hemorrhage; FIb, oozing hemorrhage; FIIa, nonbleeding visible vessel; FIIb, adherent clot; FIIc, flat pigmented spot; FIII, clean based.[74] In a systematic review of recurrent bleeding risk after endoscopic intervention for bleeding peptic ulcers, presence of active bleeding at the initial endoscopy (F1a or F1b) was a risk factor for further bleeding.[75] Conversely, ulcers with a flat pigmented spot (FIIc) or clean base (FIII) are at a low risk for recurrent bleeding and do not require endoscopic therapy.[76] A post-hoc analysis of data from an RCT comparing intravenous esomeprazole with placebo in ulcer bleeding demonstrated that recurrent bleeding rates after successful endoscopic hemostasis were 22.5% for FIa ulcers, 17.7% for FIIb ulcers, 11.3% for FIIa ulcers, and 4.9% for FIb ulcers.[77] Given that the classification of bleeding ulcers has a clear impact on recurrent bleeding risk and dictates the need for endoscopic therapy and postintervention PPI dose, the Forrest classification should be used in procedure documentation.

14. *Frequency of endoscopic treatment delivered to ulcers with active spurting or oozing or with nonbleeding visible vessels (Priority Indicator)*
    Strength of recommendation: 1A
    Performance target: >90%
    Type of measure: Process

Discussion. Endoscopic hemostatic therapy is recommended for ulcers with active spurting, active oozing, and nonbleeding visible vessels. A meta-analysis of 19 RCTs showed significant benefit in reducing further bleeding for endoscopic therapy compared with no endoscopic therapy in patients with both active ulcer bleeding (RR, .29; 95% CI, .20-.43) and nonbleeding visible vessels (RR, .49; 95% CI, .40-.59).[78] Prior studies have typically grouped spurting and oozing bleeding into 1 category, although data suggest that further bleeding after endoscopic intervention is significantly higher in active spurting ulcers compared with oozing, the latter of which may have a lower recurrent bleeding rate than nonbleeding visible vessels.[77] American College of Gastroenterology guidelines on the management of UGIB could not reach a recommendation for the need for endoscopic hemostatic therapy in ulcers with adherent clots because of a lack of available data.[13]

Options for endoscopic therapy with moderate-quality data include bipolar coagulation, heater probe, and sclerosant therapy. A meta-analysis of 15 RCTs showed that thermal contact therapy with a heater probe or bipolar electrocautery was associated with a reduced risk of further bleeding compared with no endoscopic therapy (RR, .44; 95% CI, .36-.54).[78] In a meta-analysis of 3 RCTs, absolute alcohol injection as a sclerosant was superior to no endoscopic therapy for further bleeding (RR, .56; 95% CI, .38-.83).[78] Evidence for the use of clips is not as strong, because no direct RCT comparisons have been done of clips with no therapy in ulcer bleeding. However, clips have been shown to be superior to epinephrine monotherapy in 2 RCTs in decreasing further bleeding (RR, .20; 95% CI, .07-.56).[13] Other options for treatment with lower quality evidence include argon plasma coagulation, soft monopolar coagulation, hemostatic powder spray with TC-325, and over-the-scope clips (OTSCs).[13] In an RCT of patients with high-risk ulcers, soft monopolar forceps coagulation significantly reduced recurrent bleeding when compared with the use of hemostatic clips.[79] Recent RCTs have shown that OTSCs may be a viable option for initial monotherapy compared with standard hemostatic therapy. In an RCT of patients hospitalized with high-risk ulcers, use of OTSCs versus standard endoscopic treatment (contact thermal therapy or clips with or without epinephrine) was associated with a reduced risk of 30-day further bleeding (3.2% vs 14.6%; risk difference, 11.4%; 95% CI, 3.3-20.0).[80] In another multicenter RCT of patients with UGIB with high risk of recurrent bleeding, use of OTSCs versus standard therapy (use of at least 2 through-the-

scope clips or hemostasis with a thermal therapy) was associated with higher rate of clinical success and lower rate of persistent bleeding.[81]

Compliance with guidelines recommending endoscopic treatment for high-risk stigmata may be suboptimal. In an audit of consecutive patients admitted across 21 Canadian hospitals with nonvariceal UGIB, only 65% of patients with high-risk stigmata received endoscopic intervention.[82]

15. *Frequency of a second treatment modality delivered (eg, coagulation, clips, argon plasma) when epinephrine injection is used to treat actively bleeding or nonbleeding visible vessels in patients with bleeding peptic ulcers*
    Strength of recommendation: 1A
    Performance target: >98%
    Type of measure: Process

Discussion. Epinephrine should only be used as part of a combination therapy with another endoscopic modality when treating high-risk ulcers. In a meta-analysis of 4 RCTs, epinephrine injection as the monotherapy was inferior in reducing further bleeding compared with standard monotherapies such as bipolar electrocoagulation and clips.[13] In a meta-analysis of 7 RCTs, epinephrine injection with a second modality was more effective in reducing further bleeding compared with epinephrine injection alone (RR, .34; 95% CI, .23-.50).[78] When evaluating specific modalities, a meta-analysis of 3 trials found that the combination of hemostatic clips and injection therapy was associated with reduced recurrent bleeding compared with injection therapy alone (number needed to treat, 10; risk difference, −.10; 95% CI, −.18 to −.03).[83] A meta-analysis of 2 small RCTs evaluated the benefit of adding an epinephrine injection before bipolar electrocoagulation compared with bipolar monotherapy and found a reduction in risk of further bleeding (RR, .35; 95% CI, .18-.71).[78] Finally, a network meta-analysis demonstrated that the combination of epinephrine injection plus mechanical therapy with clips significantly reduced recurrent bleeding compared with epinephrine injection alone (odds ratio, .19; 95% CI, .07-.52).[84] The findings of studies uniformly indicate that although epinephrine can be used successfully to achieve hemostasis initially, it should only be used in combination with a second treatment modality to optimize a reduced risk of recurrent bleeding.

16. *Frequency with which achievement of primary hemostasis in cases of attempted hemostasis of nonvariceal UGIB lesion is documented (Priority Indicator)*
    Strength of recommendation: 2A
    Performance target: >90%
    Type of measure: Outcome

Discussion. The frequency with which achieving primary hemostasis in patients undergoing therapeutic endoscopy for UGIB is documented was a process measure in the prior iteration of this document, with the goal of benchmarking endoscopist competence. The 2021 American College of Gastroenterology clinical guideline on the management of ulcer bleeding provides recommendations focused primarily on the outcome of further bleeding (including both persistent and recurrent bleeding) and did not provide recommendations for documentation of hemostasis or specific targets for how often primary hemostasis should be achieved.[13] Regardless, obtaining initial hemostasis in cases of active ulcer bleeding is an important endpoint, because failure to achieve initial hemostasis during the index endoscopy is associated with increased mortality, recurrent bleeding, and hospital costs.[85]

Clinical data support high rates of initial hemostasis during EGD. In a recent RCT of OTSCs versus standard treatment (thermal therapy or clips with epinephrine) in patients with high-risk, nonvariceal UGIB, the primary endpoint of successful initial hemostasis was achieved in 92% of the OTSC group and 73% in the standard group.[81] In a meta-analysis of RCTs comparing hemostatic spray (TC-325) versus standard therapy in nonvariceal UGIB, the pooled initial hemostasis rate was 91% in the topical spray group and 79% in the standard arm.[86]

Several variables may influence rates of hemostasis and include type of bleeding lesion seen (spurting vs oozing lesion vs visible vessel), modality of endoscopic therapy chosen (OTSCs, hemostatic spray, thermal therapies, clips), timing of endoscopy, administration of high-dose PPIs, and patient-level variables such as use of antiplatelet and anticoagulant medications. Ultimately, further RCTs are needed to determine the optimal treatment modality for ulcer bleeding, both in terms of initial hemostasis and reducing further bleeding.

The current quality indicator focuses on documentation of hemostasis, not the achievement of hemostasis. Although achievement of hemostasis would be theoretically preferable as a quality indicator, wide differences in patient mix and lesion characteristics between endoscopists make it difficult to establish a generic benchmark for attaining hemostasis.

17. *Frequency with which gastric biopsy sampling is done or follow-up endoscopy is planned to exclude malignancy in patients with gastric ulcers.*
    Strength of recommendation: 2C
    Performance target: >80%
    Type of measure: Process

Discussion. A minority of gastric ulcers are malignant, but endoscopic features alone are not sufficient to exclude malignancy. Performing biopsy sampling of gastric ulcers either at the initial endoscopy or at the follow-up endoscopy is highly accurate in identifying or excluding malignancy.[87-95] The exact number of biopsy specimens to obtain is unclear but should be representative of the

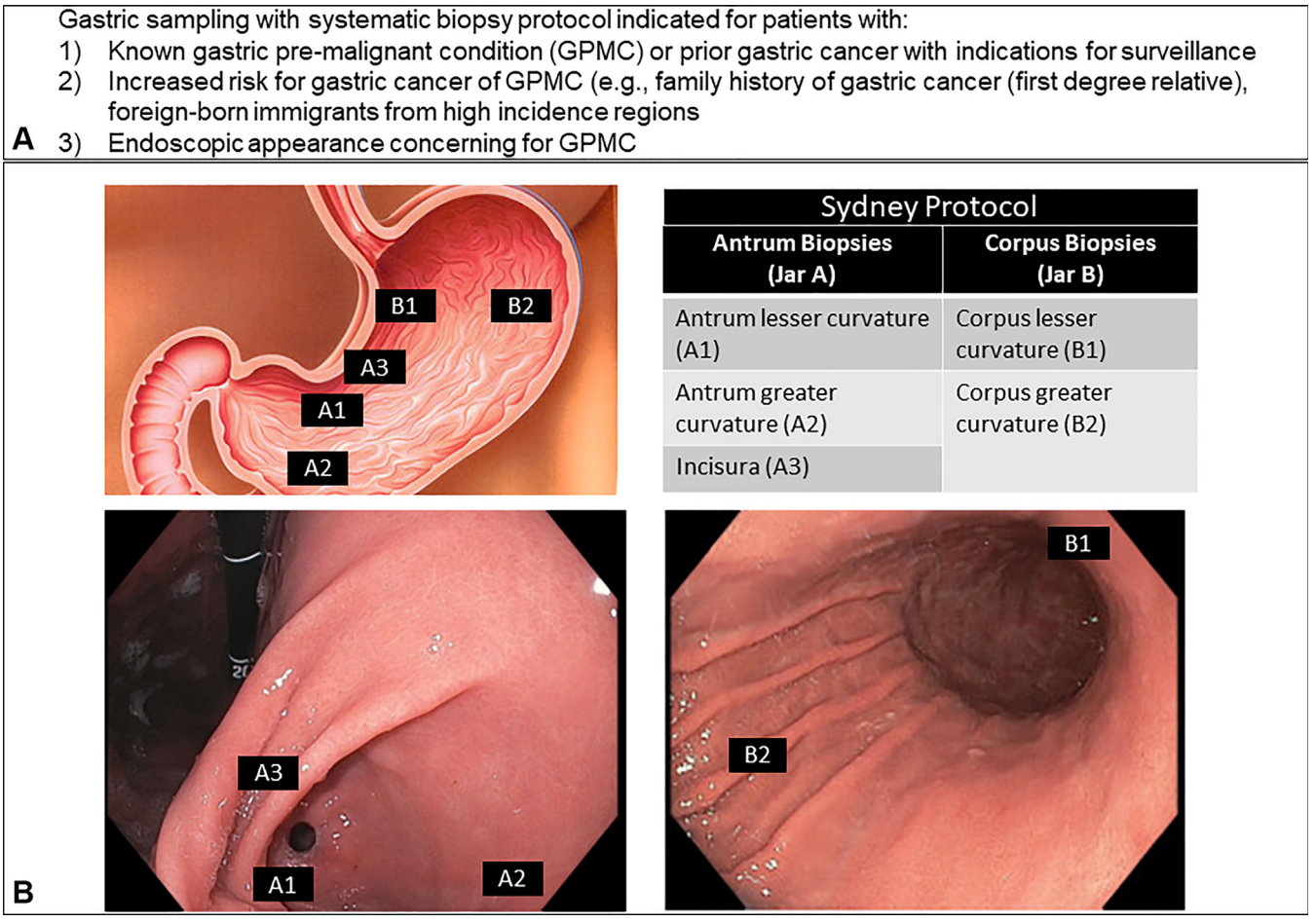

Figure 3. Systematic gastric biopsy sampling protocol for gastric premalignant conditions. **A,** Indications for systematic gastric biopsy sampling. **B,** Sydney protocol.

lesion. In cases when the gastric ulcer is bleeding or has high-risk stigmata for bleeding, biopsy sampling can be deferred to a subsequent endoscopy. Gastric biopsy samples may not be needed if there are patient factors in which the risk of gastric cancer is considered negligible (eg, a young healthy patient with small superficial ulcers in the setting of heavy nonsteroidal anti-inflammatory drug use).

18. *Frequency of systematic biopsy sampling of the gastric corpus, antrum, and incisura in patients with known gastric premalignant conditions (GPMCs), patients at high risk for gastric cancer, or patients with an endoscopic appearance concerning for GPMCs (Priority Indicator)*
    Strength of recommendation: 2C
    Performance target: >90%
    Type of measure: Process

Discussion. Gastric sampling with a systematic biopsy sampling protocol is indicated for 3 groups of patients: patients with known GPMCs or prior gastric cancer who have indications for surveillance; individuals at increased risk for gastric cancer or GPMCs, for example, those with a family history of gastric cancer (first-degree relative) or foreign-born immigrants from high-incidence regions; and patients with an endoscopic appearance concerning for GPMCs (Fig. 3A). The updated Sydney system is the recognized standard for gastric biopsy sampling in patients with suspected or known GPMCs.[96] The Sydney biopsy sampling protocol requires 1 to 2 biopsy samples from each of 5 sites: antrum greater and lesser curvatures, incisura, and corpus greater and lesser curvatures. At minimum, 2 separate containers should be used, 1 for the antrum and incisura and 1 for the corpus (Fig. 3B). The use of image-enhanced endoscopy (IEE) (eg, NBI) in these groups of patients will help focus biopsy sampling within the framework of the Sydney protocol. Additional targeted biopsy specimens of mucosal abnormalities should be placed in a separate container.

A high-quality gastric pathology report for patients with known or suspected GPMCs who have undergone biopsy sampling using the Sydney protocol should include *H pylori* status, severity of atrophy and gastric IM (GIM) (mild, moderate, severe), subtype GIM (complete,

incomplete), and anatomic extent of GPMCs. Limited GIM is confined to the antrum and incisura, whereas extensive GIM also involves the corpus. The severity refers to the proportion of GIM (or atrophy) in individual biopsy samples in each compartment (antrum, incisura, corpus). The GIM subtype and anatomic extent and presence of *severe* atrophy or GIM are the principal histologic features for the recommendation of GPMC surveillance endoscopy.

The Sydney system of biopsy sampling serves as the basis for pathology staging systems such as the Operative Link for Gastritis Assessment and Operative Link on Gastric Intestinal Metaplasia Assessment (OLGIM) systems to assess the extent and severity of gastric atrophy and IM. The Operative Link for Gastritis Assessment and OLGIM systems stage gastric antral and corpus atrophy or GIM based on the presence of none (score 0), mild (score I), moderate (score II), or severe (score III) atrophy or IM in the corpus and antrum (including the incisura angularis).[97,98] The presence and severity of atrophy or IM at the 2 locations is used to generate a cancer risk stage. The European literature underscores that the Sydney biopsy sampling system combined with this staging system correlates with the risk of progression to gastric cancer (eg, stages III and IV).[97,98]

Few studies have examined the outcomes based on the endoscopy biopsy sampling rate. In 1 European multicenter study, the endoscopy biopsy sampling rate was shown to correlate with GPMCs and cancer detection. The odds ratios for the moderate, high, and very-high endoscopy biopsy sampling rate groups of detecting GPMCs were 1.6 (95% CI, 1.3-1.9), 2.0 (95% CI, 1.7-2.4), and 2.5 (95% CI, 2.1-2.9), respectively, compared with the low endoscopy biopsy sampling rate group ($P < .001$). This association was confirmed with the same thresholds in a validation cohort. Endoscopists with higher endoscopy biopsy sampling rates had a lower risk of missed cancer compared with those in the lower endoscopy biopsy sampling rate group (odds ratio, .44; 95% CI, .20-1.00).[99]

19. *Frequency with which HDWLE and virtual chromoendoscopy are used in patients with known GPMCs, at high risk for gastric cancer, or with an endoscopic appearance concerning for GPMCs*
    Strength of recommendation: 2C
    Performance target: >90%
    Type of measure: Process

Discussion. In patients undergoing upper endoscopy for evaluation of GPMCs, we recommend the use of HDWLE and virtual chromoendoscopy, also labeled as IEE. This recommendation is primarily based on the extensive literature of early gastric cancer diagnosis in East Asia and Europe. Specific studies are emerging in Europe and North America.[100-103]

GPMCs have identifiable endoscopic features that are associated with high sensitivity and specificity when HDWLE and IEE (eg, NBI) are used. Areas with GIM typically appear mildly nodular with ridged or "tubulovillous" mucosal patterns, with the characteristic "light blue crest" sign (on NBI with near-focus endoscopy) (Fig. 4).[100,104] Usual endoscopic features of atrophic gastritis include pale appearance of the gastric mucosa, increased visibility of the vasculature because of thinning of the gastric mucosa, and loss of gastric folds.[105] Artificial intelligence protocols and systems are anticipated; however, data on the efficacy of these systems to increase detection or classification of GPMCs are lacking in U.S. populations.

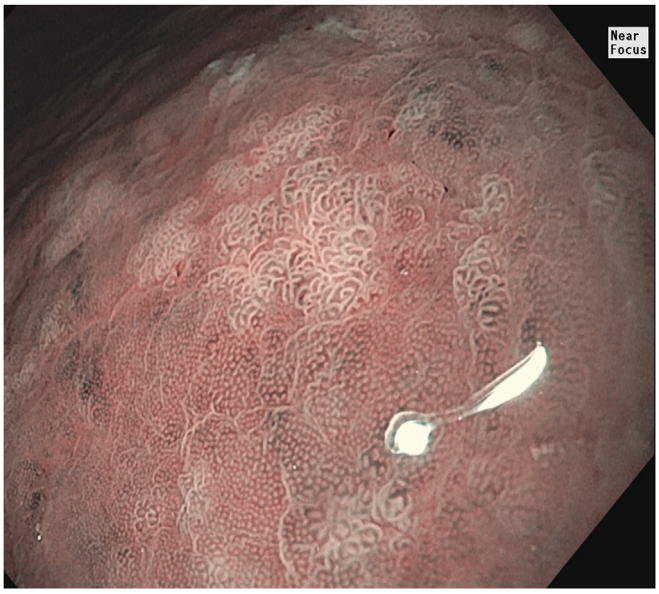

Figure 4. Patchy gastric intestinal metaplasia (GIM) with near-focus imaging and narrow-band imaging (NBI). The patchy multifocal aspect of GIM is demonstrated. In the inferior area, normal glandular structures are arranged in a regular honeycomb pattern. In the central area, the tubular white glandular structures of gastric metaplasia are observed. The light blue crest is a thin white or blue line located at the borders of the tubular glands. The light blue crest is often white with NBI and is specific for GIM. The NBI examination with near focus facilitates targeted biopsy sampling within the framework of the Sydney system biopsy sampling protocol. (Photo courtesy of Dan Li, Department of Gastroenterology, Kaiser Permanente Medical Center, Santa Clara, Calif, USA.)

20. *Frequency with which gastric polyps (without the typical appearance of a fundic gland polyp) >10 mm in size are biopsy sampled or resected*
    Strength of recommendation: 2C
    Performance target: >80%
    Type of measure: Process

Discussion. Most gastric polyps are benign, with low malignant potential. Evidence to guide management of gastric polyps is limited. Hyperplastic and adenomatous polyps should be resected when possible, because biopsy sampling alone may not detect HGD or cancer.[106,107] All adenomatous gastric polyps should be resected. The risk of malignant potential with hyperplastic polyps increases

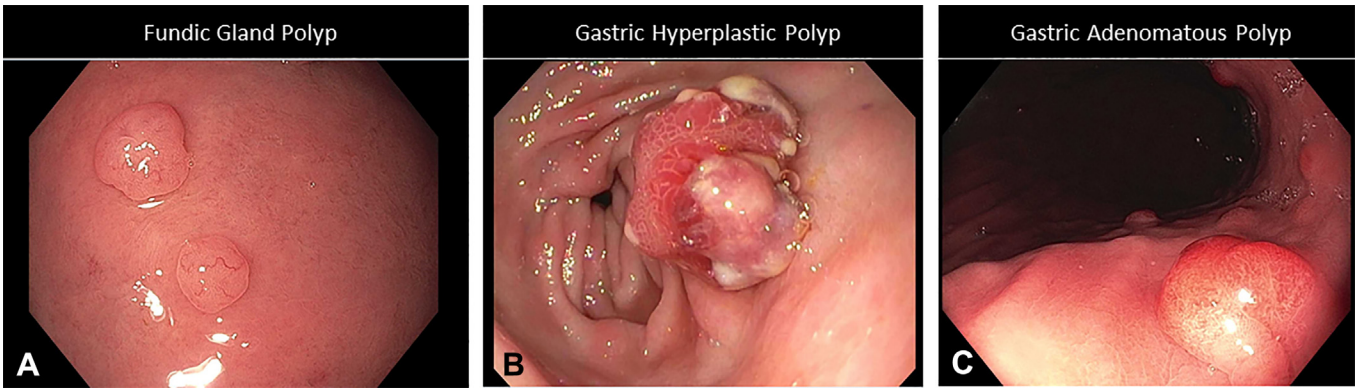

Figure 5. Endoscopic characteristics of gastric polyps. **A,** Fundic gland polyps, which are typically small, numerous, and located in the fundus and body of the stomach. They often have a translucent appearance with lacy vessels and can be sessile or semipedunculated. **B,** Gastric hyperplastic polyps can be smooth and rounded or have an ulcerated surface, as in this picture. They are typically sessile or semipedunculated and typically occur in the antrum. **C,** Gastric adenomatous polyps may be polypoid or flat and are usually small and solitary. Most gastric adenomas occur in the antrum but can occur in the body or cardia.

with size. A retrospective cohort study by Joao et al[108] demonstrated that among 195 gastric hyperplastic polyps removed, the median size of polyps with neoplasia was 17 mm (10-50 mm), and in multivariate analysis, size >25 mm was significantly associated with neoplasia (odds ratio, 84; 95% CI, 7.4-954). Because of the low risk of neoplasia in small hyperplastic polyps, only those polyps >.5 cm typically require resection.[108-110] Patients with gastric adenomas and hyperplastic polyps have an increased prevalence of GPMCs[111,112] and should undergo a detailed visual inspection (HDWLE and IEE) in combination with Sydney protocol biopsy sampling for GPMCs and *H pylori* status. Patients with familial adenomatous polyposis have an increased risk for upper GI cancers, and these patients often have multiple fundic gland polyps.[111-114] At the time of initial diagnosis, samples of representative polyps should be taken. Polyps >1 cm without the typical appearance of fundic gland polyps (slightly hyperemic, sessile, and with a smooth surface contour) should undergo biopsy sampling or be completely resected (Fig. 5).[115-122]

21. *Frequency with which ≥4 duodenal biopsy samples (including 1 from the bulb) are obtained in patients with suspected celiac disease*
    Strength of recommendation: 1C
    Performance target: >98%
    Type of measure: Process

Discussion. In patients with clinically suspected celiac disease, small-intestine biopsy specimens are instrumental for the diagnosis. Similarly, biopsy samples elucidate the response to therapy. Celiac disease histologic abnormalities are often patchy.[123] In patients in whom celiac disease is suspected, multiple biopsy specimens should be taken to maximize accuracy of the diagnosis. The minimum standard is 4 biopsy samples, including 1 from the duodenal bulb (at the 9 o'clock or 12 o'clock position), which can be accomplished with 2 passes of the biopsy forceps. Optimally in a detailed examination, 4 biopsy samples are obtained distal to the bulb and 1 to 2 from the duodenal bulb (at the 9 o'clock or 12 o'clock positions), with the sets of biopsy specimens placed in 2 separate jars.[124-126] Biopsy sampling of the duodenal bulb may improve the diagnostic yield by detecting the most severe villous atrophy within the duodenum.[127,128] Endoscopists may consider 1 biopsy bite per pass to optimize orientation.[129]

POSTPROCEDURE QUALITY INDICATORS

The postprocedure period extends from the time the endoscope is removed until subsequent follow-up. Postprocedure activities common to all endoscopic procedures include procedure documentation; postprocedure monitoring; providing instructions to the patient; recognizing, documenting, and rescuing patients from adverse events; pathology follow-up; communication with referring physicians; and assessing patient satisfaction. The following provides details on postprocedure quality indicators relevant to EGD.

22. *Frequency of repeat endoscopy recommendation after a course of acid suppression in cases of LA grade C or D erosive esophagitis*
    Strength of recommendation: 2B
    Performance target: >90%
    Type of measure: Process

Discussion. Lack of healing of erosive esophagitis with 8 weeks of antisecretory therapy can be seen in up to 20% of patients, with rates of nonhealing in up 30% of more severe (LA grades C or D) esophagitis. Further, in a prospective study of 172 patients with erosive esophagitis, BE was confirmed in 17.4% of those with LA grade C or D esophagitis.[45,130] Thus, LA grade C or D esophagitis signifies severe erosive reflux disease that is more refractory to acid

TABLE 5. Potent acid suppressive agents with U.S. Food and Drug Administration approval

Agent	U.S. Food and Drug Administration–approved indications and dosages
Pantoprazole	Healing of EE: 40 mg daily for up to 8 wk Maintenance of healed EE: 40 mg daily Pathologic hypersecretory conditions: 40 mg daily
Omeprazole*	Healing of EE: 20 mg daily for 4-8 wk Maintenance of healed EE: 20 mg daily GERD, symptomatic (nonerosive): 20 mg daily for up to 4 wk Gastric ulcer, short-term treatment of benign: 40 mg daily for 4-8 wk Duodenal ulcer (short-term treatment): 20 mg daily Pathologic hypersecretory conditions: 60 mg twice daily Frequent heartburn: over-the-counter treatment: 20 mg daily for 14 days
Lansoprazole*	Healing of EE: 30 mg daily for up to 8 wk Maintenance of healed EE: 15 mg daily GERD, symptomatic (nonerosive): 15 mg daily for up to 8 wk Gastric ulcer, short-term treatment of benign: 30 mg daily for up to 8 wk Gastric ulcer, healing of NSAID-associated: 30 mg daily for up to 8 wk Gastric ulcer, risk reduction of NSAID associated: 15 mg daily for up to 12 wk Duodenal ulcer (short-term treatment): 15 mg daily for 4 wk Duodenal ulcer (maintenance of healed): 15 mg daily Pathologic hypersecretory conditions: 60 mg twice daily Frequent heartburn: over-the-counter treatment: 15 mg daily for 14 days
Esomeprazole*	Healing of EE: 20 mg or 40 mg daily 4-8 wk Maintenance of healed EE: 20 mg daily GERD, symptomatic (nonerosive): 20 mg daily for 4 wk Gastric ulcer, risk reduction of NSAID-associated: 20 mg or 40 mg daily for up to 6 mo Pathologic hypersecretory conditions: 40 mg twice daily Frequent heartburn: over-the-counter treatment: 22.3 mg daily for 14 days
Rabeprazole*	Duodenal ulcer (short-term treatment): 20 mg daily for 4 wk GERD, healing of erosive or ulcerative: 20 mg daily for 4-8 wk GERD, maintenance of healing of erosive or ulcerative: 20 mg daily GERD, symptomatic (nonerosive): 20 mg daily for 4 wk Pathologic hypersecretory conditions: 60 mg daily
Dexlansoprazole	Healing of EE: 60 mg daily up to 8 wk Maintenance of healed EE: 30 mg daily GERD, symptomatic (nonerosive): 30 mg daily for 4 wk
Vonoprazan*	Healing of EE: 20 mg daily Maintenance of healed EE: 10 mg daily

EE, erosive esophagitis; *NSAID*, nonsteroidal anti-inflammatory drug.
*U.S. Food and Drug Administration approval as part of a *Helicobaster pylori* eradication therapy.

suppression and at increased risk of BE, and repeat EGD after potent acid suppression in the form of double-dose PPIs or potassium competitive acid blocker (Table 5)[131] for at least 8 weeks is recommended.[47,132]

23. *Frequency of acid suppression therapy recommendation for patients who underwent dilation for peptic esophageal strictures and do not have allergy or other contraindication to these medications*
    Strength of recommendation: 2B
    Performance target: >98%
    Type of measure: Process

Discussion. Peptic strictures result from chronic inflammation from gastroesophageal reflux, and management hinges on disruption of the stricture and potent gastric acid suppression. A randomized double-blind trial of patients with peptic stricture identified a significantly higher risk of persistent stricture and need for redilation in patients managed with H2 receptor antagonists (ranitidine 150 mg twice daily) compared with PPIs (omeprazole 20 mg daily).[130,133,134] The prevalence of peptic strictures has decreased since the introduction of PPI therapy. Therefore, acid suppression, preferably with PPIs or another potent acid suppressive agent, is recommended in patients with peptic strictures to prevent stricture recurrence and promote healing of esophagitis.

24. *Frequency with which follow-up surveillance endoscopy is recommended; no sooner than 3 years if systematic surveillance biopsy sampling was performed in a patient known to have nondysplastic BE without a prior history of dysplasia*
    Strength of recommendation: 2C
    Performance target: >80%
    Type of measure: Process

Discussion. Endoscopic surveillance appears to be modestly effective in reducing EAC-related mortality.[60] Recommended surveillance intervals are based on the rate of progression to EAC, and given the low rate of progression in those with nondysplastic BE (.3/100 patient-years),[135] surveillance has been recommended every 3 to 5 years. Progression risk has also been strongly correlated with increasing BE segment length.[69] Given the lower risk of progression in those with short-segment BE (<3 cm),[127] guidelines have suggested that those with short-segment BE could undergo surveillance every 5 years.[47,136]

Unfortunately, surveillance endoscopy appears to be done more frequently than recommended in guidelines in a substantial portion of patients. In an analysis from the GI Quality Improvement Consortium database, 30% of patients with nondysplastic BE were recommended to undergo surveillance endoscopy at shorter intervals (1-2 years).[137] Younger age and increasing BE length were associated with shorter recommended surveillance intervals. Overuse of surveillance has also been reported by other single-center and multicenter studies from academic and Veteran Affairs medical centers with rates ranging from 38% to 65%.[138-140] Several factors such as patient expectations and fear of cancer may drive overuse of surveillance. Financial incentives and fee-for-service models may also be a factor. Involvement in a malpractice suit has been associated with overuse of BE surveillance.[141,142] Notably, studies have also reported underuse of surveillance endoscopy in the Veteran Affairs system with 50% of BE patients not undergoing surveillance endoscopy after the initial diagnosis.[143]

Consequences of surveillance overuse include a higher chance of adverse events (although rare) from sedation and procedure-related issues. Additionally, costs (both direct [procedure related] and indirect [related to loss of work for both the patient and caregivers]) also need to be considered, particularly in light of studies showing cost-effectiveness of surveillance endoscopy in those without dysplasia at 3- to 5-year intervals but generally not at shorter intervals.[144] Recommendation of appropriate surveillance intervals (≥3 years in those without dysplasia) has been suggested as a quality metric by the American Gastroenterological Association[145] and was included as one of the gastroenterology metrics to reduce overuse of healthcare services in the Choosing Wisely campaign by the American Board of Internal Medicine foundation.[146]

25. *Frequency of achieving complete eradication of IM within 18 months of initial endoscopic treatment in patients with BE and dysplasia or intramucosal carcinoma undergoing endoscopic eradication therapy*
    Strength of recommendation: 1C+
    Performance target: >75%
    Type of measure: Outcome

Discussion. A primary aim of endoscopic eradication therapy for BE is complete eradication of IM given the associated risk reduction of progression to EAC. In a multicenter RCT at 9 European sites of 136 patients with LGD comparing endoscopic surveillance with endoscopic eradication therapy, 88.2% had complete eradication of IM with ablation.[147] In the AIM Dysplasia trial, a multicenter sham-controlled RCT of 127 patients, complete eradication of IM was seen within 12 months of the first treatment in 77.4% of patients undergoing radiofrequency ablation, and when stratified by grade of dysplasia, 74% of those with HGD and 81% of those with LGD achieved complete eradication of IM.[148] In terms of durability, complete eradication of IM was observed in 93% at 2 years, 91% at 3 years,[149] and 90% at 5 years.[150] In the EURO-II European multicenter study, combined EMR and ablation achieved 87% complete eradication of IM among patients with HGD or EAC.[151] Additional prospective studies using multimodal endoscopic eradication therapy reported complete eradication of IM rates ranging from 83% to 90%[152,153]; database registry studies from the United Kingdom and the United States also reported similar results.[154,155] The TREAT-BE quality indicators developed for endoscopic eradication therapy for dysplastic BE, published in 2017, specified an 18-month time period to achieve complete eradication of IM.[156,157]

26. *Frequency of administering high-dose PPI therapy (continuous or intermittently for 3 days) after successful endoscopic hemostatic therapy of a bleeding ulcer in patients without allergy or contraindication to the medication (Priority Indicator)*
    Strength of recommendation: 1A
    Performance target: >95%
    Type of measure: Process

Discussion. High-dose PPI therapy, defined as ≥80 mg of pantoprazole or equivalent daily for ≥3 days administered either intermittently or by continuous infusion, should be given after successful endoscopic intervention of a high-risk ulcer. In a systematic review and meta-analysis of 7 RCTs comparing high-dose PPI therapy with placebo or no therapy after successful endoscopic therapy, the risk of further bleeding was significantly reduced by high-dose PPI therapy (RR, .43; 95% CI, .33-.56), as was the risk of mortality (RR, .41; 95% CI, .22-.79), compared with placebo or no therapy.[13] In a meta-analysis of 9 RCTs comparing high-dose PPI therapy with H2 receptor antagonists after successful endoscopic therapy, there was a significant reduction in further bleeding with PPI use compared with H2 receptor antagonists (RR, .56; 95% CI, .41-.77). The magnitude of benefit was identical with a high-dose bolus followed by continuous infusion of PPIs (80-mg bolus, 8-mg/h infusion) and intermittent PPIs with average total daily doses of 80 to 160 mg.[158] Finally, Laine and McQuaid[78] compared the risks of further bleeding with a high-dose PPI bolus followed by a continuous infusion of intravenous PPI therapy versus less-intensive PPI regimens after successful

endoscopic hemostatic therapy. Further bleeding was not significantly different in a meta-analysis comparing bolus-continuous infusion versus less-intensive regimens (RR, 1.12; 95% CI, .86-1.47).[78] In another meta-analysis of RCTs comparing intermittent PPIs with bolus plus continuous-infusion PPIs after endoscopic therapy for high-risk ulcers, the risk ratios for 30-day recurrent bleeding were similar.[159] Despite uncertainty about optimal dosing of intermittent (oral or intravenous) high-dose PPI therapy, high-dose PPI therapy is recommended (either continuously or intermittently) after endoscopic therapy of high-risk ulcers given the high-quality data supporting its use when compared with placebo or no treatment.

27. *Frequency with which plans to test for* H pylori *infection are documented in patients with GPMCs, peptic ulcer disease, and other* H pylori–associated conditions
Strength of recommendation: 2C
Performance target: >95%
Type of measure: Process

Discussion. All patients with a positive test of active infection with *H pylori* should be offered treatment; thus, the central issue is which patients should be tested for the infection. Plans to test for *H pylori* should be documented in patients with peptic ulcer disease, GPMCs, and other *H pylori*–associated conditions. *H pylori* infection affects approximately one-third of the U.S. population, with variation among racial, ethnic, immigrant, and socioeconomic groups. *H pylori* has a high attributable risk for peptic ulcer disease, GPMCs, and gastric adenocarcinoma. *H pylori* eradication therapy facilitates peptic ulcer healing and prevention of recurrence.[160-162]

H pylori is the most important known risk factor for gastric cancer, which initiates and perpetuates the carcinogenesis cascade of chronic gastritis, multifocal atrophy, GIM, dysplasia, and adenocarcinoma. Eradication of *H pylori* demonstrates benefits for the reduction of the risk of GPMC progression to gastric cancer[161,163,164] as well as the risk of metachronous lesions in the remnant stomach among patients with early gastric cancer after endoscopic resection.[165-168]

28. *Frequency with which plans to treat and assess eradication of* H pylori *infection are documented in patients with endoscopically diagnosed* H pylori
Strength of recommendation: 2A
Performance target: >95%
Type of measure: Outcome

Discussion. Patients diagnosed endoscopically with *H pylori* require documentation of the plan to treat the infection and to test for successful eradication. Because a positive *H pylori* finding occurs after the endoscopy report is finalized, plans to treat the infection and test for successful eradication will not be part of the endoscopy report but should be part of the medical record. A standardized phrase for the follow-up of a positive test could be placed in the endoscopy report, with subsequent documentation in the medical records once pathology results are available with a plan to treat and test for eradication if positive for *H pylori*. Post-treatment testing is performed at least 1 to 2 months after the completion of therapy.[160,167] The assessment of eradication is indicated for all patients who are treated for *H pylori* infection. Studies suggest poor performance in the United States, particularly in the inpatient setting, with respect to testing (eg, peptic ulcer disease) and confirmation of eradication. In a large Veteran Affairs cohort from an integrated health system, only 23.9% of patients were retested.[169] The lack of post-treatment testing and delays in retreatment have been linked to an increased risk for UGIB.[170] Specific interventions may be productive.[171] Although beyond the scope of this recommendation, management is further compounded by poor provider compliance with guideline-directed *H pylori* eradication regimens, antibiotic resistance, and appropriate patient education.[172]

29. *Frequency that the GPMC surveillance plan is documented in patients with known GPMCs*
Strength of recommendation: 2C
Performance target: >90%
Type of measure: Process

Discussion. In patients with established GPMCs, we recommend the documentation of the endoscopic surveillance plan including the recommendation of surveillance versus no surveillance based on clinical and histologic risk factors for progression to gastric adenocarcinoma. Endoscopic surveillance at 3-year intervals is recommended for individuals with GIM who are considered at higher risk for progression to gastric cancer. High-risk individuals are those with GIM and at least one of the following clinical or histologic factors. The clinical factors include a family history of gastric cancer (first-degree relative), being a foreign-born immigrant from a high-incidence nation, and being from a high gastric cancer incidence U.S. population (eg, East Asian, Hispanic, Black, Native American). High-risk GIM histology encompasses incomplete GIM (versus complete GIM) and extensive GIM (involvement of the antrum/incisura and the corpus). We note a less-common scenario of individuals with severe-grade atrophic gastritis on biopsies of the antrum or corpus, wherein surveillance would also be indicated.[163,173]

We suggest the documentation of the plan for *no surveillance* for individuals with low-risk GIM or atrophy, including individuals without high-risk clinical factors (as listed above) and with low-risk histology (complete-type GIM [without a component of incomplete GIM] and GIM of limited anatomic extent). Complete GIM confined to the antrum and/or incisura is the common scenario.

Optimization of surveillance intervals requires further study in the United States. In general, in individuals with non-neoplastic GPMC who are deemed high risk, we

recommend endoscopic surveillance every 3 years.[174,175] Patients with multiple risk factors may be considered for shorter intervals, with patient–physician shared decision-making. For example, an individual with incomplete GIM on the index endoscopy and with a family history of gastric cancer may be considered for a 2-year interval.[176]

Although no data exist comparing the duration of surveillance intervals among different risk groups, there are sufficient data in the form of large observational studies to understand the natural history of GIM among these groups. For example, based on data from the Gastric Cancer Epidemiology and Molecular Genetics Programme cohort, individuals with OLGIM stage II progressed to early gastric neoplasia in a median of 50.7 months (range, 28.5-73.3) and those with OLGIM stages III and IV progressed in a median of 22.7 months (range, 12.7-44.8).[90,173,176]

DISCUSSION

In this document, we reviewed the updated quality indicators for EGD. Common across all EGDs is the new quality indicator addressing thorough photodocumentation. Because EGD is used to evaluate and manage distinct upper GI conditions, most quality indicators relate to a specific presentation or condition including erosive esophagitis, BE, EoE, UGIB, gastric ulcers, gastric polyps, GPMCs, and celiac disease. This document includes new quality indicators as well as quality indicators retained from the prior iteration. Some quality indicators from the prior iteration were not retained because compliance with the indicators was high in endoscopic practice and thus less useful to measure. These include providing prophylactic antibiotics before PEG tube placement, variceal ligation as the first modality of endoscopic treatment for esophageal varices, and acid suppressive therapy after the endoscopic diagnosis of peptic ulcer disease. Although not included as quality indicators, these practices remain integral to quality endoscopy. Further, quality indicators related to antibiotics in patients with portal hypertension and cirrhosis presenting with UGIB and to vasoactive drugs for suspected variceal bleeding were not included in this document because they relate to medical management and are not specific to endoscopy.

Several areas were not ready for inclusion as quality indicators but are ripe for investigation and future consideration. One of these areas is documentation of procedure time, whether the collective time spent during EGD or the specific time spent inspecting premalignant conditions such as during surveillance for BE or GIM. In particular, initial data suggest a correlation between inspection time of the BE segment and dysplasia detection, representing an important area for ongoing research and consideration as a quality indicator in the future. Expanding on the concept of adenoma detection rate in screening colonoscopy, there is potential for measurement of the neoplasia detection rate during surveillance of BE or the GIM detection rate during GIM screening. We anticipate future quality indicator documents in EGD to address the role of deep learning and artificial intelligence as well as quality in advanced endoscopy such as endobariatrics and third-space endoscopy.

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Abbreviations: BE, Barrett's esophagus; CI, confidence interval; EAC, esophageal adenocarcinoma; EoE, eosinophilic esophagitis; EREFS, endoscopic reference score; GEJ, gastroesophageal junction; GIM, gastric intestinal metaplasia; GPMC, gastric premalignant condition; HDWLE, high-definition white-light endoscopy; HGD, high-grade dysplasia; IEE, image-enhanced endoscopy; IM, intestinal metaplasia; LA, Los Angeles; LGD, low-grade dysplasia; NBI, narrow-band imaging; OLGIM, Operative Link on Gastric Intestinal Metaplasia Assessment; OTSC, over-the-scope clip; PPI, proton pump inhibitor; RCT, randomized controlled trial; RR, relative risk; SCJ, squamocolumnar junction; UGIB, upper GI bleeding.

REFERENCES

1. Peery AF, Crockett SD, Murphy CC, et al. Burden and cost of gastrointestinal, liver, and pancreatic diseases in the United States: update 2021. Gastroenterology 2022;162:621-44.
2. Peery AF, Crockett SD, Murphy CC, et al. Burden and cost of gastrointestinal, liver, and pancreatic diseases in the United States: update 2018. Gastroenterology 2019;156:254-72.
3. Zullo A, Manta R, De Francesco V, et al. Diagnostic yield of upper endoscopy according to appropriateness: a systematic review. Dig Liver Dis 2019;51:335-9.
4. Chassin MR, Galvin RW. The urgent need to improve health care quality. Institute of Medicine National roundtable on health care quality. JAMA 1998;280:1000-5.
5. Park WG, Shaheen NJ, Cohen J, et al. Quality indicators for EGD. Gastrointest Endosc 2015;81:17-30.
6. Park WG, Shaheen NJ, Cohen J, et al. Quality indicators for EGD. Am J Gastroenterol 2015;110:60-71.
7. Faigel DO, Pike IM, Baron TH, et al. Quality indicators for gastrointestinal endoscopic procedures: an introduction. Gastrointest Endosc 2006;63(4 Suppl):S3-9.
8. Faigel DO, Pike IM, Baron TH, et al. Quality indicators for gastrointestinal endoscopic procedures: an introduction. Am J Gastroenterol 2006;101:866-72.
9. Elmunzer BJ, Anderson MA, Mishra G, et al. Quality indicators common to all GI endoscopic procedures. Gastrointest Endosc 2024;100:382-94.
10. ASGE Endoscopy Unit Quality Indicator Taskforce; Day LW, Cohen J, Greenwald D, et al. Quality indicators for gastrointestinal endoscopy units. VideoGIE 2017;2:119-40.
11. Buri L, Hassan C, Bersani G, et al. Appropriateness guidelines and predictive rules to select patients for upper endoscopy: a nationwide multicenter study. Am J Gastroenterol 2010;105:1327-37.
12. Hassan C, Bersani G, Buri L, et al. Appropriateness of upper-GI endoscopy: an Italian survey on behalf of the Italian Society of Digestive Endoscopy. Gastrointest Endosc 2007;65:767-74.
13. Laine L, Barkun AN, Saltzman JR, et al. ACG clinical guideline: upper gastrointestinal and ulcer bleeding. Am J Gastroenterol 2021;116:899-917.
14. Cooper GS, Chak A, Connors AF Jr, et al. The effectiveness of early endoscopy for upper gastrointestinal hemorrhage: a community-based analysis. Med Care 1998;36:462-74.
15. Cooper GS, Kou TD, Wong RC. Use and impact of early endoscopy in elderly patients with peptic ulcer hemorrhage: a population-based analysis. Gastrointest Endosc 2009;70:229-35.
16. Garg SK, Anugwom C, Campbell J, et al. Early esophagogastroduodenoscopy is associated with better outcomes in upper gastrointestinal bleeding: a nationwide study. Endosc Int Open 2017;5:E376-86.
17. Wysocki JD, Srivastav S, Winstead NS. A nationwide analysis of risk factors for mortality and time to endoscopy in upper gastrointestinal haemorrhage. Aliment Pharmacol Ther 2012;36:30-6.
18. Jeong N, Kim KS, Jung YS, et al. Delayed endoscopy is associated with increased mortality in upper gastrointestinal hemorrhage. Am J Emerg Med 2019;37:277-80.
19. Laursen SB, Leontiadis GI, Stanley AJ, et al. Relationship between timing of endoscopy and mortality in patients with peptic ulcer bleeding: a nationwide cohort study. Gastrointest Endosc 2017;85:936-44.
20. Guo CLT, Wong SH, Lau LHS, et al. Timing of endoscopy for acute upper gastrointestinal bleeding: a territory-wide cohort study. Gut 2022;71:1544-50.
21. Lau JYW, Yu Y, Tang RSY, et al. Timing of endoscopy for acute upper gastrointestinal bleeding. N Engl J Med 2020;382:1299-308.
22. Sharma P. Top 10 tips for performing a high-quality upper endoscopy (with videos). Gastrointest Endosc 2021;94:989-94.
23. Bisschops R, Rutter MD, Areia M, et al. Overcoming the barriers to dissemination and implementation of quality measures for gastrointestinal endoscopy: European Society of Gastrointestinal Endoscopy (ESGE) and United European Gastroenterology (UEG) position statement. United Eur Gastroenterol J 2021;9:120-6.
24. Emura F, Sharma P, Arantes V, et al. Principles and practice to facilitate complete photodocumentation of the upper gastrointestinal tract: World Endoscopy Organization position statement. Dig Endosc 2020;32:168-79.
25. Valori R, Cortas G, de Lange T, et al. Performance measures for endoscopy services: a European Society of Gastrointestinal Endoscopy (ESGE) quality improvement initiative. United Eur Gastroenterol J 2019;7:21-44.
26. Cordova H, Sanchez-Montes C, Delgado-Guillena PG, et al. Quality indicators for esophagogastroduodenoscopy: a comparative study of outcomes after an improvement programme in a tertiary hospital [Spanish]. Gastroenterol Hepatol 2017;40:587-94.
27. Yang LS, Thompson AJ, Taylor ACF, et al. Quality of upper GI endoscopy: a prospective cohort study on impact of endoscopist education. Gastrointest Endosc 2022;96:467-75.
28. Hiremath G, Sun L, Collins MH, et al. Esophageal epithelium and lamina propria are unevenly involved in eosinophilic esophagitis. Clin Gastroenterol Hepatol 2023;21:2807-16.
29. Eluri S, Iglesia EGA, Massaro M, et al. Practice patterns and adherence to clinical guidelines for diagnosis and management of eosinophilic esophagitis among gastroenterologists. Dis Esophagus. Epub 2020 May 7.
30. Nielsen JA, Lager DJ, Lewin M, et al. The optimal number of biopsy fragments to establish a morphologic diagnosis of eosinophilic esophagitis. Am J Gastroenterol 2014;109:515-20.

31. Shah A, Kagalwalla AF, Gonsalves N, et al. Histopathologic variability in children with eosinophilic esophagitis. Am J Gastroenterol 2009;104:716-21.
32. Schoepfer AM, Hirano I, Coslovsky M, et al. Variation in endoscopic activity assessment and endoscopy score validation in adults with eosinophilic esophagitis. Clin Gastroenterol Hepatol 2019;17:1477-88.
33. Hirano I, Moy N, Heckman MG, et al. Endoscopic assessment of the oesophageal features of eosinophilic oesophagitis: validation of a novel classification and grading system. Gut 2013;62:489-95.
34. van Rhijn BD, Warners MJ, Curvers WL, et al. Evaluating the endoscopic reference score for eosinophilic esophagitis: moderate to substantial intra- and interobserver reliability. Endoscopy 2014;46:1049-55.
35. Ma C, Bredenoord AJ, Dellon ES, et al. Reliability and responsiveness of endoscopic disease activity assessment in eosinophilic esophagitis. Gastrointest Endosc 2022;95:1126-37.
36. Wechsler JB, Bolton SM, Amsden K, et al. Eosinophilic esophagitis reference score accurately identifies disease activity and treatment effects in children. Clin Gastroenterol Hepatol 2018;16:1056-63.
37. Lundell LR, Dent J, Bennett JR, et al. Endoscopic assessment of oesophagitis: clinical and functional correlates and further validation of the Los Angeles classification. Gut 1999;45:172-80.
38. Akdamar K, Ertan A, Agrawal NM, et al. Upper gastrointestinal endoscopy in normal asymptomatic volunteers. Gastrointest Endosc 1986;32:78-80.
39. Rusu RI, Fox MR, Tucker E, et al. Validation of the Lyon classification for GORD diagnosis: acid exposure time assessed by prolonged wireless pH monitoring in healthy controls and patients with erosive oesophagitis. Gut 2021;70:2230-7.
40. Takashima T, Iwakiri R, Sakata Y, et al. Endoscopic reflux esophagitis and *Helicobacter pylori* infection in young healthy Japanese volunteers. Digestion 2012;86:55-8.
41. Zagari RM, Fuccio L, Wallander MA, et al. Gastro-oesophageal reflux symptoms, oesophagitis and Barrett's oesophagus in the general population: the Loiano-Monghidoro study. Gut 2008;57:1354-9.
42. Visaggi P, Del Corso G, Gyawali CP, et al. Ambulatory pH-impedance findings confirm that grade B esophagitis provides objective diagnosis of gastroesophageal reflux disease. Am J Gastroenterol 2023;118:794-801.
43. Armstrong D, Bennett JR, Blum AL, et al. The endoscopic assessment of esophagitis: a progress report on observer agreement. Gastroenterology 1996;111:85-92.
44. Bredenoord AJ, Hemmink GJ, Smout AJ. Relationship between gastro-oesophageal reflux pattern and severity of mucosal damage. Neurogastroenterol Motil 2009;21:807-12.
45. Hanna S, Rastogi A, Weston AP, et al. Detection of Barrett's esophagus after endoscopic healing of erosive esophagitis. Am J Gastroenterol 2006;101:1416-20.
46. Levine DS, Haggitt RC, Blount PL, et al. An endoscopic biopsy protocol can differentiate high-grade dysplasia from early adenocarcinoma in Barrett's esophagus. Gastroenterology 1993;105:40-50.
47. Shaheen NJ, Falk GW, Iyer PG, et al. Diagnosis and management of Barrett's esophagus: an updated ACG guideline. Am J Gastroenterol 2022;117:559-87.
48. Sharma P, Dent J, Armstrong D, et al. The development and validation of an endoscopic grading system for Barrett's esophagus: the Prague C & M criteria. Gastroenterology 2006;131:1392-9.
49. Dickman R, Levi Z, Vilkin A, et al. Predictors of specialized intestinal metaplasia in patients with an incidental irregular Z line. Eur J Gastroenterol Hepatol 2010;22:135-8.
50. Thota PN, Vennalaganti P, Vennelaganti S, et al. Low risk of high-grade dysplasia or esophageal adenocarcinoma among patients with Barrett's esophagus less than 1 cm (irregular Z line) within 5 years of index endoscopy. Gastroenterology 2017;152:987-92.
51. Jung KW, Talley NJ, Romero Y, et al. Epidemiology and natural history of intestinal metaplasia of the gastroesophageal junction and Barrett's esophagus: a population-based study. Am J Gastroenterol 2011;106:1447-55; quiz 1456.
52. Itskoviz D, Levi Z, Boltin D, et al. Risk of neoplastic progression among patients with an irregular Z line on long-term follow-up. Dig Dis Sci 2018;63:1513-7.
53. Alvarez Herrero L, Curvers WL, van Vilsteren FG, et al. Validation of the Prague C&M classification of Barrett's esophagus in clinical practice. Endoscopy 2013;45:876-82.
54. Vahabzadeh B, Seetharam AB, Cook MB, et al. Validation of the Prague C & M criteria for the endoscopic grading of Barrett's esophagus by gastroenterology trainees: a multicenter study. Gastrointest Endosc 2012;75:236-41.
55. Gupta N, Gaddam S, Wani SB, et al. Longer inspection time is associated with increased detection of high-grade dysplasia and esophageal adenocarcinoma in Barrett's esophagus. Gastrointest Endosc 2012;76:531-8.
56. Vithayathil M, Modolell I, Ortiz-Fernandez-Sordo J, et al. The effect of procedural time on dysplasia detection rate during endoscopic surveillance of Barrett's esophagus. Endoscopy 2023;55:491-8.
57. Visrodia K, Singh S, Krishnamoorthi R, et al. Magnitude of missed esophageal adenocarcinoma after Barrett's esophagus diagnosis: a systematic review and meta-analysis. Gastroenterology 2016;150:599-607.
58. ASGE Technology Committee; Thosani N, Abu Dayyeh BK, Sharma P, et al. ASGE Technology Committee systematic review and meta-analysis assessing the ASGE Preservation and Incorporation of Valuable Endoscopic Innovations thresholds for adopting real-time imaging-assisted endoscopic targeted biopsy during endoscopic surveillance of Barrett's esophagus. Gastrointest Endosc 2016;83:684-98.
59. Wolfsen HC, Crook JE, Krishna M, et al. Prospective, controlled tandem endoscopy study of narrow band imaging for dysplasia detection in Barrett's esophagus. Gastroenterology 2008;135:24-31.
60. Sharma P, Hawes RH, Bansal A, et al. Standard endoscopy with random biopsies versus narrow band imaging targeted biopsies in Barrett's oesophagus: a prospective, international, randomised controlled trial. Gut 2013;62:15-21.
61. Qumseya BJ, Wang H, Badie N, et al. Advanced imaging technologies increase detection of dysplasia and neoplasia in patients with Barrett's esophagus: a meta-analysis and systematic review. Clin Gastroenterol Hepatol 2013;11:1562-70.
62. Fitzgerald RC, Saeed IT, Khoo D, et al. Rigorous surveillance protocol increases detection of curable cancers associated with Barrett's esophagus. Dig Dis Sci 2001;46:1892-8.
63. Abela JE, Going JJ, Mackenzie JF, et al. Systematic four-quadrant biopsy detects Barrett's dysplasia in more patients than nonsystematic biopsy. Am J Gastroenterol 2008;103:850-5.
64. Nachiappan A, Ragunath K, Card T, et al. Diagnosing dysplasia in Barrett's oesophagus still requires Seattle protocol biopsy in the era of modern video endoscopy: results from a tertiary centre Barrett's dysplasia database. Scand J Gastroenterol 2020;55:9-13.
65. Beaufort I, Akkerman E, van Munster S, et al. Effect of biopsy protocol adherence vs non-adherence on dysplasia detection rates in Barrett's esophagus surveillance endoscopies: a systematic review and meta-analysis. Endosc Int Open 2023;11:E221-9.
66. Codipilly DC, Chandar AK, Singh S, et al. The effect of endoscopic surveillance in patients with Barrett's esophagus: a systematic review and meta-analysis. Gastroenterology 2018;154:2068-86.
67. Abrams JA, Kapel RC, Lindberg GM, et al. Adherence to biopsy guidelines for Barrett's esophagus surveillance in the community setting in the United States. Clin Gastroenterol Hepatol 2009;7:736-42; quiz 710.
68. Wani S, Williams JL, Komanduri S, et al. Endoscopists systematically undersample patients with long-segment Barrett's esophagus: an analysis of biopsy sampling practices from a quality improvement registry. Gastrointest Endosc 2019;90:732-41.
69. Krishnamoorthi R, Singh S, Ragunathan K, et al. Factors associated with progression of barrett's esophagus: a systematic review and meta-analysis. Clin Gastroenterol Hepatol 2018;16:1046-55.
70. The Paris endoscopic classification of superficial neoplastic lesions: esophagus, stomach, and colon: November 30 to December 1, 2002. Gastrointest Endosc 2003;58(6 Suppl):S3-43.

71. Dhaliwal L, Codipilly DC, Gandhi P, et al. Neoplasia detection rate in Barrett's esophagus and its impact on missed dysplasia: results from a large population-based database. Clin Gastroenterol Hepatol 2021;19:922-9.
72. Tholoor S, Bhattacharyya R, Tsagkournis O, et al. Acetic acid chromoendoscopy in Barrett's esophagus surveillance is superior to the standardized random biopsy protocol: results from a large cohort study (with video). Gastrointest Endosc 2014;80:417-24.
73. Gralnek IM, Stanley AJ, Morris AJ, et al. Endoscopic diagnosis and management of nonvariceal upper gastrointestinal hemorrhage (NVUGIH): European Society of Gastrointestinal Endoscopy (ESGE) guideline—update 2021. Endoscopy 2021;53:300-32.
74. Forrest JA, Finlayson ND, Shearman DJ. Endoscopy in gastrointestinal bleeding. Lancet 1974;2:394-7.
75. Elmunzer BJ, Young SD, Inadomi JM, et al. Systematic review of the predictors of recurrent hemorrhage after endoscopic hemostatic therapy for bleeding peptic ulcers. Am J Gastroenterol 2008;103:2625-32; quiz 2633.
76. Hsu PI, Lai KH, Lin XZ, et al. When to discharge patients with bleeding peptic ulcers: a prospective study of residual risk of rebleeding. Gastrointest Endosc 1996;44:382-7.
77. Jensen DM, Eklund S, Persson T, et al. Reassessment of rebleeding risk of Forrest IB (oozing) peptic ulcer bleeding in a large international randomized trial. Am J Gastroenterol 2017;112:441-6.
78. Laine L, McQuaid KR. Endoscopic therapy for bleeding ulcers: an evidence-based approach based on meta-analyses of randomized controlled trials. Clin Gastroenterol Hepatol 2009;7:33-47; quiz 1-2.
79. Toka B, Eminler AT, Karacaer C, et al. Comparison of monopolar hemostatic forceps with soft coagulation versus hemoclip for peptic ulcer bleeding: a randomized trial (with video). Gastrointest Endosc 2019;89:792-802.
80. Lau JYW, Li R, Tan CH, et al. Comparison of over-the-scope clips to standard endoscopic treatment as the initial treatment in patients with bleeding from a nonvariceal upper gastrointestinal cause: a randomized controlled trial. Ann Intern Med 2023;176:455-62.
81. Meier B, Wannhoff A, Denzer U, et al. Over-the-scope-clips versus standard treatment in high-risk patients with acute non-variceal upper gastrointestinal bleeding: a randomised controlled trial (STING-2). Gut 2022;71:1251-8.
82. Lu Y, Barkun AN, Martel M, et al. Adherence to guidelines: a national audit of the management of acute upper gastrointestinal bleeding. The REASON registry. Can J Gastroenterol Hepatol 2014;28:495-501.
83. Baracat F, Moura E, Bernardo W, et al. Endoscopic hemostasis for peptic ulcer bleeding: systematic review and meta-analyses of randomized controlled trials. Surg Endosc 2016;30:2155-68.
84. Shi K, Shen Z, Zhu G, et al. Systematic review with network meta-analysis: dual therapy for high-risk bleeding peptic ulcers. BMC Gastroenterol 2017;17:55.
85. Roy A, Kim M, Hawes R, et al. The clinical and cost implications of failed endoscopic hemostasis in gastroduodenal ulcer bleeding. United Eur Gastroenterol J 2017;5:359-64.
86. Deliwala SS, Chandan S, Mohan BP, et al. Hemostatic spray (TC-325) vs. standard endoscopic therapy for non-variceal gastrointestinal bleeding: a meta-analysis of randomized controlled trials. Endosc Int Open 2023;11:E288-95.
87. Yang LS, Hartley I, Thompson AJ, et al. Evaluation of endoscopic practices and outcomes in follow-up of gastric ulcers. J Clin Gastroenterol 2022;56:412-8.
88. Esmadi M, Ahmad DS, Hammad HT. Endoscopic surveillance for gastric ulcers. South Med J 2014;107:289-91.
89. Gielisse EA, Kuyvenhoven JP. Follow-up endoscopy for benign-appearing gastric ulcers has no additive value in detecting malignancy: It is time to individualise surveillance endoscopy. Gastric Cancer 2015;18:803-9.
90. Lee YB, Han J, Cho JH, et al. Clinical outcomes of endoscopic surveillance for gastric ulcers in populations with a high prevalence of gastric cancer. Turk J Gastroenterol 2016;27:421-7.
91. Pimenta-Melo AR, Monteiro-Soares M, Libanio D, et al. Missing rate for gastric cancer during upper gastrointestinal endoscopy: a systematic review and meta-analysis. Eur J Gastroenterol Hepatol 2016;28:1041-9.
92. Saini SD, Eisen G, Mattek N, et al. Utilization of upper endoscopy for surveillance of gastric ulcers in the United States. Am J Gastroenterol 2008;103:1920-5.
93. Yeh JM, Ho W, Hur C. Cost-effectiveness of endoscopic surveillance of gastric ulcers to improve survival. Gastrointest Endosc 2010;72:33-43.
94. Hopper AN, Stephens MR, Lewis WG, et al. Relative value of repeat gastric ulcer surveillance gastroscopy in diagnosing gastric cancer. Gastric Cancer 2006;9:217-22.
95. Selinger CP, Cochrane R, Thanaraj S, et al. Gastric ulcers: malignancy yield and risk stratification for follow-up endoscopy. Endosc Int Open 2016;4:E709-14.
96. Dixon MF, Genta RM, Yardley JH, et al. Classification and grading of gastritis. The updated Sydney System. International Workshop on the Histopathology of Gastritis, Houston 1994. Am J Surg Pathol 1996;20:1161-81.
97. Capelle LG, de Vries AC, Haringsma J, et al. The staging of gastritis with the OLGA system by using intestinal metaplasia as an accurate alternative for atrophic gastritis. Gastrointest Endosc 2010;71:1150-8.
98. Rugge M, Genta RM, Fassan M, et al. OLGA gastritis staging for the prediction of gastric cancer risk: a long-term follow-up study of 7436 patients. Am J Gastroenterol 2018;113:1621-8.
99. Januszewicz W, Wieszczy P, Bialek A, et al. Endoscopist biopsy rate as a quality indicator for outpatient gastroscopy: a multicenter cohort study with validation. Gastrointest Endosc 2019;89:1141-9.
100. Buxbaum JL, Hormozdi D, Dinis-Ribeiro M, et al. Narrow-band imaging versus white light versus mapping biopsy for gastric intestinal metaplasia: a prospective blinded trial. Gastrointest Endosc 2017;86:857-65.
101. Ezoe Y, Muto M, Uedo N, et al. Magnifying narrowband imaging is more accurate than conventional white-light imaging in diagnosis of gastric mucosal cancer. Gastroenterology 2011;141:2017-25.
102. Zhang Q, Wang F, Chen ZY, et al. Comparison of the diagnostic efficacy of white light endoscopy and magnifying endoscopy with narrow band imaging for early gastric cancer: a meta-analysis. Gastric Cancer 2016;19:543-52.
103. Zhou F, Wu L, Huang M, et al. The accuracy of magnifying narrow band imaging (ME-NBI) in distinguishing between cancerous and noncancerous gastric lesions: a meta-analysis. Medicine 2018;97:e9780.
104. Dinis-Ribeiro M, da Costa-Pereira A, Lopes C, et al. Magnification chromoendoscopy for the diagnosis of gastric intestinal metaplasia and dysplasia. Gastrointest Endosc 2003;57:498-504.
105. Fukuta N, Ida K, Kato T, et al. Endoscopic diagnosis of gastric intestinal metaplasia: a prospective multicenter study. Dig Endosc 2013;25:526-34.
106. Ahn JY, Son DH, Choi KD, et al. Neoplasms arising in large gastric hyperplastic polyps: endoscopic and pathologic features. Gastrointest Endosc 2014;80:1005-13.
107. Hoffman A, Atreya R, Rath T, et al. Current endoscopic resection techniques for gastrointestinal lesions: endoscopic mucosal resection, submucosal dissection, and full-thickness resection. Visc Med 2021;37:358-71.
108. Joao M, Areia M, Alves S, et al. Gastric hyperplastic polyps: a benign entity? Analysis of recurrence and neoplastic transformation in a cohort study. GE Port J Gastroenterol 2021;28:328-35.
109. Forte E, Petit B, Walter T, et al. Risk of neoplastic change in large gastric hyperplastic polyps and recurrence after endoscopic resection. Endoscopy 2020;52:444-53.
110. Lam SK, Lau GKK. Idiopathic multitudinous fundic gland polyposis: a new disease and a management dilemma. JGH Open 2021;5:525-7.
111. Abraham SC, Montgomery EA, Singh VK, et al. Gastric adenomas: intestinal-type and gastric-type adenomas differ in the risk of adenocarcinoma and presence of background mucosal pathology. Am J Surg Pathol 2002;26:1276-85.
112. Abraham SC, Singh VK, Yardley JH, et al. Hyperplastic polyps of the stomach: associations with histologic patterns of gastritis and gastric atrophy. Am J Surg Pathol 2001;25:500-7.

113. Mankaney GN, Cruise M, Sarvepalli S, et al. Identifying factors associated with detection of sessile gastric polyps in patients with familial adenomatous polyposis. Endosc Int Open 2022;10:E1080-7.
114. Wang KK, Kim K, Bancila L, et al. Novel endoscopic polypectomy surveillance technique for fundic gland polyps in familial adenomatous polyposis can improve early detection of dysplasia and gastric cancer. Am J Gastroenterol 2022;117:1246-54.
115. Draganov PV, Wang AY, Othman MO, et al. AGA Institute clinical practice update: endoscopic submucosal dissection in the United States. Clin Gastroenterol Hepatol 2019;17:16-25.
116. Liu X, Li Y, Zhou X, et al. Endoscopic treatment of cardia lesions: the effects on gastroesophageal reflux disease. Ann Palliat Med 2021;10:6669-77.
117. Qin S, Xu Y. Progress in clinical diagnosis and treatment for gastric polyps. Zhong Nan Da Xue Xue Bao Yi Xue Ban 2020;45:74-8.
118. Reggiani Bonetti L, Manta R, Manno M, et al. Optimal processing of ESD specimens to avoid pathological artifacts. Tech Coloproctol 2018;22:857-66.
119. Zagari RM, Frazzoni L, Fuccio L, et al. Adherence to European Society of Gastrointestinal Endoscopy quality performance measures for upper and lower gastrointestinal endoscopy: a nationwide survey from the Italian Society of Digestive Endoscopy. Front Med 2022;9:868449.
120. Olmez S, Sayar S, Saritas B, et al. Evaluation of patients with gastric polyps. North Clin Istanb 2018;5:41-6.
121. Salomao M, Luna AM, Sepulveda JL, et al. Mutational analysis by next generation sequencing of gastric type dysplasia occurring in hyperplastic polyps of the stomach: mutations in gastric hyperplastic polyps. Exp Mol Pathol 2015;99:468-73.
122. Vatansever S, Akpinar Z, Alper E, et al. Gastric polyps and polypoid lesions: retrospective analysis of 36650 endoscopic procedures in 29940 patients. Turk J Gastroenterol 2015;26:117-22.
123. Hopper AD, Cross SS, Sanders DS. Patchy villous atrophy in adult patients with suspected gluten-sensitive enteropathy: Is a multiple duodenal biopsy strategy appropriate? Endoscopy 2008;40:219-24.
124. Lebwohl B, Kapel RC, Neugut AI, et al. Adherence to biopsy guidelines increases celiac disease diagnosis. Gastrointest Endosc 2011;74:103-9.
125. Pais WP, Duerksen DR, Pettigrew NM, et al. How many duodenal biopsy specimens are required to make a diagnosis of celiac disease? Gastrointest Endosc 2008;67:1082-7.
126. Rubio-Tapia A, Hill ID, Semrad C, et al. American College of Gastroenterology guidelines update: diagnosis and management of celiac disease. Am J Gastroenterol 2023;118:59-76.
127. Evans KE, Aziz I, Cross SS, et al. A prospective study of duodenal bulb biopsy in newly diagnosed and established adult celiac disease. Am J Gastroenterol 2011;106:1837-742.
128. Kurien M, Evans KE, Hopper AD, et al. Duodenal bulb biopsies for diagnosing adult celiac disease: Is there an optimal biopsy site? Gastrointest Endosc 2012;75:1190-6.
129. Latorre M, Lagana SM, Freedberg DE, et al. Endoscopic biopsy technique in the diagnosis of celiac disease: One bite or two? Gastrointest Endosc 2015;81:1228-33.
130. Lightdale CJ, Schmitt C, Hwang C, et al. A multicenter, randomized, double-blind, 8-week comparative trial of low-dose esomeprazole (20 mg) and standard-dose omeprazole (20 mg) in patients with erosive esophagitis. Dig Dis Sci 2006;51:852-7.
131. Laine L, DeVault K, Katz P, et al. Vonoprazan versus lansoprazole for healing and maintenance of healing of erosive esophagitis: a randomized trial. Gastroenterology 2023;164:61-71.
132. Katz PO, Dunbar KB, Schnoll-Sussman FH, et al. ACG clinical guideline for the diagnosis and management of gastroesophageal reflux disease. Am J Gastroenterol 2022;117:27-56.
133. Smith PM, Kerr GD, Cockel R, et al. A comparison of omeprazole and ranitidine in the prevention of recurrence of benign esophageal stricture. Restore Investigator Group. Gastroenterology 1994;107:1312-8.
134. Desai M, Hamade N, Sharma P. Management of peptic strictures. Am J Gastroenterol 2020;115:967-70.
135. Desai TK, Krishnan K, Samala N, et al. The incidence of oesophageal adenocarcinoma in non-dysplastic Barrett's oesophagus: a meta-analysis. Gut 2012;61:970-6.
136. Fitzgerald RC, di Pietro M, Ragunath K, et al. British Society of Gastroenterology guidelines on the diagnosis and management of Barrett's oesophagus. Gut 2014;63:7-42.
137. Wani S, Williams JL, Komanduri S, et al. Over-utilization of repeat upper endoscopy in patients with non-dysplastic Barrett's esophagus: a quality registry study. Am J Gastroenterol 2019;114:1256-64.
138. Tavakkoli A, Appelman HD, Beer DG, et al. Use of appropriate surveillance for patients with nondysplastic Barrett's esophagus. Clin Gastroenterol Hepatol 2018;16:862-9.
139. Faqih A, Broman KK, Huang LC, et al. Frequency of endoscopic surveillance for Barrett's esophagus is influenced by health insurance status: results from a population-based analysis. Dis Esophagus 2017;30:1-8.
140. Crockett SD, Lipkus IM, Bright SD, et al. Overutilization of endoscopic surveillance in nondysplastic Barrett's esophagus: a multicenter study. Gastrointest Endosc 2012;75:23-31.
141. Rubenstein JH, Pohl H, Adams MA, et al. Overuse of repeat upper endoscopy in the Veterans Health Administration: a retrospective analysis. Am J Gastroenterol 2017;112:1678-85.
142. Rubenstein JH, Saini SD, Kuhn L, et al. Influence of malpractice history on the practice of screening and surveillance for Barrett's esophagus. Am J Gastroenterol 2008;103:842-9.
143. El-Serag HB, Duan Z, Hinojosa-Lindsey M, et al. Practice patterns of surveillance endoscopy in a Veterans Affairs database of 29,504 patients with Barrett's esophagus. Gastrointest Endosc 2012;76:743-55.
144. Rubenstein JH, Inadomi JM. Cost-effectiveness of screening, surveillance, and endoscopic eradication therapies for managing the burden of esophageal adenocarcinoma. Gastrointest Endosc Clin North Am 2021;31:77-90.
145. Sharma P, Katzka DA, Gupta N, et al. Quality indicators for the management of Barrett's esophagus, dysplasia, and esophageal adenocarcinoma: international consensus recommendations from the American Gastroenterological Association Symposium. Gastroenterology 2015;149:1599-606.
146. Naik AD, Hinojosa-Lindsey M, Arney J, et al. Choosing wisely and the perceived drivers of endoscopy use. Clin Gastroenterol Hepatol 2013;11:753-5.
147. Phoa KN, van Vilsteren FG, Weusten BL, et al. Radiofrequency ablation vs endoscopic surveillance for patients with Barrett esophagus and low-grade dysplasia: a randomized clinical trial. JAMA 2014;311:1209-17.
148. Shaheen NJ, Sharma P, Overholt BF, et al. Radiofrequency ablation in Barrett's esophagus with dysplasia. N Engl J Med 28 2009;360:2277-88.
149. Shaheen NJ, Overholt BF, Sampliner RE, et al. Durability of radiofrequency ablation in Barrett's esophagus with dysplasia. Gastroenterology 2011;141:460-8.
150. Cotton CC, Wolf WA, Overholt BF, et al. Late recurrence of Barrett's esophagus after complete eradication of intestinal metaplasia is rare: final report from Ablation in Intestinal Metaplasia Containing Dysplasia Trial. Gastroenterology 2017;153:681-8.
151. Phoa KN, Pouw RE, Bisschops R, et al. Multimodality endoscopic eradication for neoplastic Barrett oesophagus: results of a European multicentre study (EURO-II). Gut 2016;65:555-62.
152. Guarner-Argente C, Buoncristiano T, Furth EE, et al. Long-term outcomes of patients with Barrett's esophagus and high-grade dysplasia or early cancer treated with endoluminal therapies with intention to complete eradication. Gastrointest Endosc 2013;77:190-9.
153. Phoa KN, Pouw RE, van Vilsteren FGI, et al. Remission of Barrett's esophagus with early neoplasia 5 years after radiofrequency ablation with endoscopic resection: a Netherlands cohort study. Gastroenterology 2013;145:96-104.
154. Haidry RJ, Butt MA, Dunn JM, et al. Improvement over time in outcomes for patients undergoing endoscopic therapy for Barrett's oesophagus-related neoplasia: 6-year experience from the first 500 patients treated in the UK patient registry. Gut 2015;64:1192-9.
155. Pasricha S, Bulsiewicz WJ, Hathorn KE, et al. Durability and predictors of successful radiofrequency ablation for Barrett's esophagus. Clin Gastroenterol Hepatol 2014;12:1840-7.
156. Wani S, Muthusamy VR, Shaheen NJ, et al. Development of quality indicators for endoscopic eradication therapies in Barrett's esophagus:

the TREAT-BE (Treatment with Resection and Endoscopic Ablation Techniques for Barrett's Esophagus) Consortium. Gastrointest Endosc 2017;86:1-17.
157. Wani S, Muthusamy VR, Shaheen NJ, et al. Development of quality indicators for endoscopic eradication therapies in Barrett's esophagus: the TREAT-BE (Treatment With Resection and Endoscopic Ablation Techniques for Barrett's Esophagus) Consortium. Am J Gastroenterol 2017;112:1032-48.
158. Zhang YS, Li Q, He BS, et al. Proton pump inhibitors therapy vs H2 receptor antagonists therapy for upper gastrointestinal bleeding after endoscopy: a meta-analysis. World J Gastroenterol 2015;21:6341-51.
159. Sachar H, Vaidya K, Laine L. Intermittent vs continuous proton pump inhibitor therapy for high-risk bleeding ulcers: a systematic review and meta-analysis. JAMA Intern Med 2014;174:1755-62.
160. Chey WD, Leontiadis GI, Howden CW, et al. ACG clinical guideline: treatment of *Helicobacter pylori* Infection. Am J Gastroenterol 2017;112:212-39.
161. Ford AC, Yuan Y, Forman D, et al. *Helicobacter pylori* eradication for the prevention of gastric neoplasia. Cochrane Database Syst Rev 2020;7:CD005583.
162. Malfertheiner P, Megraud F, O'Morain CA, et al. Management of *Helicobacter pylori* infection—the Maastricht V/Florence Consensus Report. Gut 2017;66:6-30.
163. Gawron AJ, Shah SC, Altayar O, et al. AGA technical review on gastric intestinal metaplasia—natural history and clinical outcomes. Gastroenterology 2020;158:705-31.
164. Mera RM, Bravo LE, Camargo MC, et al. Dynamics of *Helicobacter pylori* infection as a determinant of progression of gastric precancerous lesions: 16-year follow-up of an eradication trial. Gut 2018;67:1239-46.
165. Choi IJ, Kook MC, Kim YI, et al. *Helicobacter pylori* therapy for the prevention of metachronous gastric cancer. N Engl J Med 22 2018;378:1085-95.
166. Choi JM, Kim SG, Choi J, et al. Effects of *Helicobacter pylori* eradication for metachronous gastric cancer prevention: a randomized controlled trial. Gastrointest Endosc 2018;88:475-85.
167. Crowe SE. *Helicobacter pylori* infection. N Engl J Med 2019;380:1158-65.
168. Khan MY, Aslam A, Mihali AB, et al. Effectiveness of *Helicobacter pylori* eradication in preventing metachronous gastric cancer and preneoplastic lesions. A systematic review and meta-analysis. Eur J Gastroenterol Hepatol 2020;32:686-94.
169. Kumar S, Metz DC, Kaplan DE, et al. Low rates of retesting for eradication of *Helicobacter pylori* infection after treatment in the Veterans Health Administration. Clin Gastroenterol Hepatol 2021;19:305-13.
170. Guo CG, Cheung KS, Zhang F, et al. Delay in retreatment of *Helicobacter pylori* infection increases risk of upper gastrointestinal bleeding. Clin Gastroenterol Hepatol 2021;19:314-22.
171. Heffley JD, Zubarik R. A standardized protocol improves testing rates for *Helicobacter pylori* among inpatients with peptic ulcer disease. Helicobacter 2021;26:e12800.
172. Shah S, Cappell K, Sedgley R, et al. Diagnosis and treatment patterns among patients with newly diagnosed *Helicobacter pylori* infection in the United States 2016-2019. Sci Rep 2023;13:1375.
173. Li D, Bautista MC, Jiang SF, et al. Risks and predictors of gastric adenocarcinoma in patients with gastric intestinal metaplasia and dysplasia: a population-based study. Am J Gastroenterol 2016;111:1104-13.
174. den Hollander WJ, Holster IL, den Hoed CM, et al. Surveillance of premalignant gastric lesions: a multicentre prospective cohort study from low incidence regions. Gut 2019;68:585-93.
175. Lee JWJ, Zhu F, Srivastava S, et al. Severity of gastric intestinal metaplasia predicts the risk of gastric cancer: a prospective multicentre cohort study (GCEP). Gut 2022;71:854-63.
176. Reddy KM, Chang JI, Shi JM, et al. Risk of gastric cancer among patients with intestinal metaplasia of the stomach in a US integrated health care system. Clin Gastroenterol Hepatol 2016;14:1420-5.