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

Sachin Wani, MD,[1,*] V. Raman Muthusamy, MD,[2,*] Nicholas J. Shaheen, MD, MPH,[3] Rena Yadlapati, MD,[4] Robert Wilson, BA,[1] Julian A. Abrams, MD,[5] Jacques Bergman, MD, PhD,[6] Amitabh Chak, MD,[7] Kenneth Chang, MD,[8] Ananya Das, MD,[9] John Dumot, MD,[7] Steven A. Edmundowicz, MD,[1] Glenn Eisen, MD,[10] Gary W. Falk, MD,[11] M. Brian Fennerty, MD,[12] Lauren Gerson, MD, MPH,[13] Gregory G. Ginsberg, MD,[11] David Grande, BA,[4] Matt Hall, PhD,[1] Ben Harnke, MLIS,[1] John Inadomi, MD,[14] Janusz Jankowski, MD,[15] Charles J. Lightdale, MD,[5] Jitin Makker, MD,[2] Robert D. Odze, MD,[16] Oliver Pech, MD,[17] Richard E. Sampliner, MD,[18] Stuart Spechler, MD,[19] George Triadafilopoulos, MD,[20] Michael B. Wallace, MD,[21] Kenneth Wang, MD,[22] Irving Waxman, MD,[23] Srinadh Komanduri, MD, MS[4]

This document was reviewed and approved by the governing boards of the American Society for Gastrointestinal Endoscopy and the American College of Gastroenterology. It appears simultaneously in *Gastrointestinal Endoscopy* and the *American Journal of Gastroenterology*.

Barrett's esophagus (BE) is the only identifiable precursor to esophageal adenocarcinoma (EAC), a malignancy that is associated with an increasing incidence and a dismal 5-year survival rate of 15% to 20%.[1-3] BE is characterized by the replacement of normal squamous epithelium of the distal esophagus with metaplastic intestinal-type columnar epithelium.[4,5] The presumed step-wise progression of BE to invasive EAC through the histopathologic stages of low-grade dysplasia (LGD), high-grade dysplasia (HGD), and intramucosal EAC provides opportunities to halt the progression and decrease the incidence of BE-related EAC.[6-10] Endoscopic eradication therapy (EET) in BE patients at increased risk of progression to invasive EAC (intramucosal EAC, HGD, and LGD) is a strategy that has been investigated extensively for cancer prevention, with the ultimate goal of reducing morbidity and mortality.

The effectiveness and safety of EET in eradicating BE-related neoplasia and maintaining remission, as demonstrated in randomized controlled trials, large observational studies, and population-based studies, has revolutionized the management of these patients and avoids the morbidity and mortality associated with esophagectomy.[11-21] Population-based studies report comparable outcomes after EET and esophagectomy in the management of HGD and mucosal EAC.[22] In addition, this practice is now endorsed by multiple recent GI society guidelines and consensus documents.[4,23-25]

EET is used increasingly not only at academic and tertiary-care centers but also among community practices.[15] Although available data support the increasing use of EET in patients with BE-related neoplasia, quality indicators in the field of EET are not well-defined. There is currently a lack of guidance with regard to quality indicators for EET such as the role of an expert pathologist, advanced endoscopic imaging, benchmark rates of complete eradication of intestinal metaplasia (CE-IM), and the number of treatment sessions necessary to achieve this endpoint in clinical practice. In addition, significant variability in endoscopic practices and lack of concordance with published guidelines is well-described both at a tertiary-care and community levels.[26] Although recent guidelines,[4,5,24] consensus documents,[25] and the American Society for Gastrointestinal Endoscopy (ASGE)/American College of Gastroenterology (ACG) Task Force on Quality in Endoscopy documents[27-29] provide excellent direction as to best practices for care of patients with BE, there is a need for formal development of quality indicators for EET in the management of patients with BE-related neoplasia.

The objective of this study was to use a methodologically rigorous process to develop valid quality indicators for EET in the management of patients with BE-related neoplasia. Defining quality indicators has the potential to optimize the management of BE-related neoplasia by increasing high-quality care.

*Drs Wani and Muthusamy contributed equally to this article.

0016-5107/$36.00
http://dx.doi.org/10.1016/j.gie.2017.03.010

METHODS

RAND/University of California, Los Angeles Appropriateness Method

The RAND/University of California, Los Angeles Appropriateness Methodology[30] (RAM) was used to develop quality indicators for EET in patients with BE-related neoplasia. In the RAM, the concept of appropriateness refers to the relative weight of the benefits and harms of an intervention. An appropriate indicator is one in which the expected health benefit exceeds the expected negative consequences by a sufficiently wide margin exclusive of costs.[30] This is a modified Delphi method[25] that, unlike the original Delphi, provides panelists with the opportunity to discuss their judgments between rating rounds in a face-to-face meeting to discuss their answers, similar to the method of the National Institute of Health Consensus Conferences. This methodology is applicable when randomized controlled trials are not available or cannot provide evidence at a level of detail sufficient to apply to the wide range of patients seen in everyday clinical practice.[30] It is a well-described methodology for the development of quality indicators and has been applied across a broad range of disease processes and procedures within gastroenterology (upper endoscopy, colonoscopy, GERD, esophageal manometry) and non-GI conditions (vascular interventions, orthopedic surgeries, surgical oncology, among others).[31-41]

Study design and methodology

The study design used to develop quality indicators for EET in BE-related neoplasia is highlighted in this section and Figure 1.

Recruitment of the expert panel. An international multidisciplinary panel of experts (gastroenterologists, a pathologists, epidemiologist, RAM methodologist, and a statistician) was recruited. The main selection criteria in the nomination process included a history of peer-reviewed publications in the field of BE and EET as well as diversity of geography and practice setting. RAM experts suggest that expert panels can be of any size that permits sufficient diversity (a minimum of 7) while ensuring that all have a chance to participate.[30]

Round 0 meeting: face-to-face meeting to discuss study objectives and methodology. This was a face-to-face meeting of invited expert panelists (Digestive Disease Week, May 16-19, 2015, Washington, DC). During this meeting, the panel was oriented to RAM and discussed the study objectives, population, diagnostic parameters, and management strategies. This facilitated the rating process and improved the efficiency of the panel process by building confidence in the methodology and creating a positive environment for future work.[30]

Compilation of potential quality metrics. After review of available guidelines, consensus documents, and relevant published literature, panel members were randomly assigned to 1 of 3 working groups that developed potential quality indicators in the before, during, and post–procedure categories. To do this, panel members provided potential quality indicators, conference calls were held to discuss and vet the proposed quality indicators, and a list was created of potential quality indicators for initial ranking.

Round 1: Initial ranking of potential quality metrics. All panel members independently ranked the potential quality indicators generated by the 3 working groups. The list of potential quality indicators was sent as a link to a REDCap database (Appendix 1, available online at www.giejournal.org) with specific instructions for ranking via e-mail (Appendix 2). Instructions highlighted that the purpose of the proposed quality indicators was to assist practitioners with quality improvement. Panel members were instructed that the indicators were intended to be measured and reported at the practice level and need not apply to a specific patient but rather to the overall care of patients with BE. An indicator was considered appropriate and/or valid if adherence to the indicator was deemed critical to providing quality care to patients with BE exclusive of cost or feasibility. As per the RAM protocol, it was emphasized that the panel members should not consider cost implications or the feasibility of implementing the indicator in their rankings. The indicator should have applied to the average patient presenting to the average physician at an average facility. Where appropriate, panelists suggested a benchmark threshold for satisfying specific metrics. Each indicator was ranked on a 9-point interval scale in which a score of 1 to 3 was signified as inappropriate, 4 to 6 was of uncertain appropriateness, and 7 to 9 was deemed appropriate. The panelists also had the opportunity to provide comments regarding each proposed quality indicator and suggest modifications.

Search strategy and systematic review of literature. A medical librarian performed a comprehensive literature search of Ovid Medline (Ovid MEDLINE in-process and other non-indexed citations, Ovid MEDLINE) Daily and Ovid MEDLINE 1946 to present), Embase (via Embase.com), and the Cochrane Database of Systematic Reviews/Cochrane Register of controlled trials (via Wiley Online Library). Publication dates were limited to 1990 through August 12, 2015, and the search was limited to English language articles. Medline records were excluded from the Embase search results before export to an EndNote Library (Clarivate Analytics, Philadelphia, Penn). The primary concept of BE along with 30 other dimensions of interventions and outcomes, their associated synonyms, and MeSH/Emtree controlled vocabulary were searched. The 30 dimensions were "ORed" together to create a single large set that was then "ANDed" with the BE set. The full search strategy for Ovid Medline can be found in the online

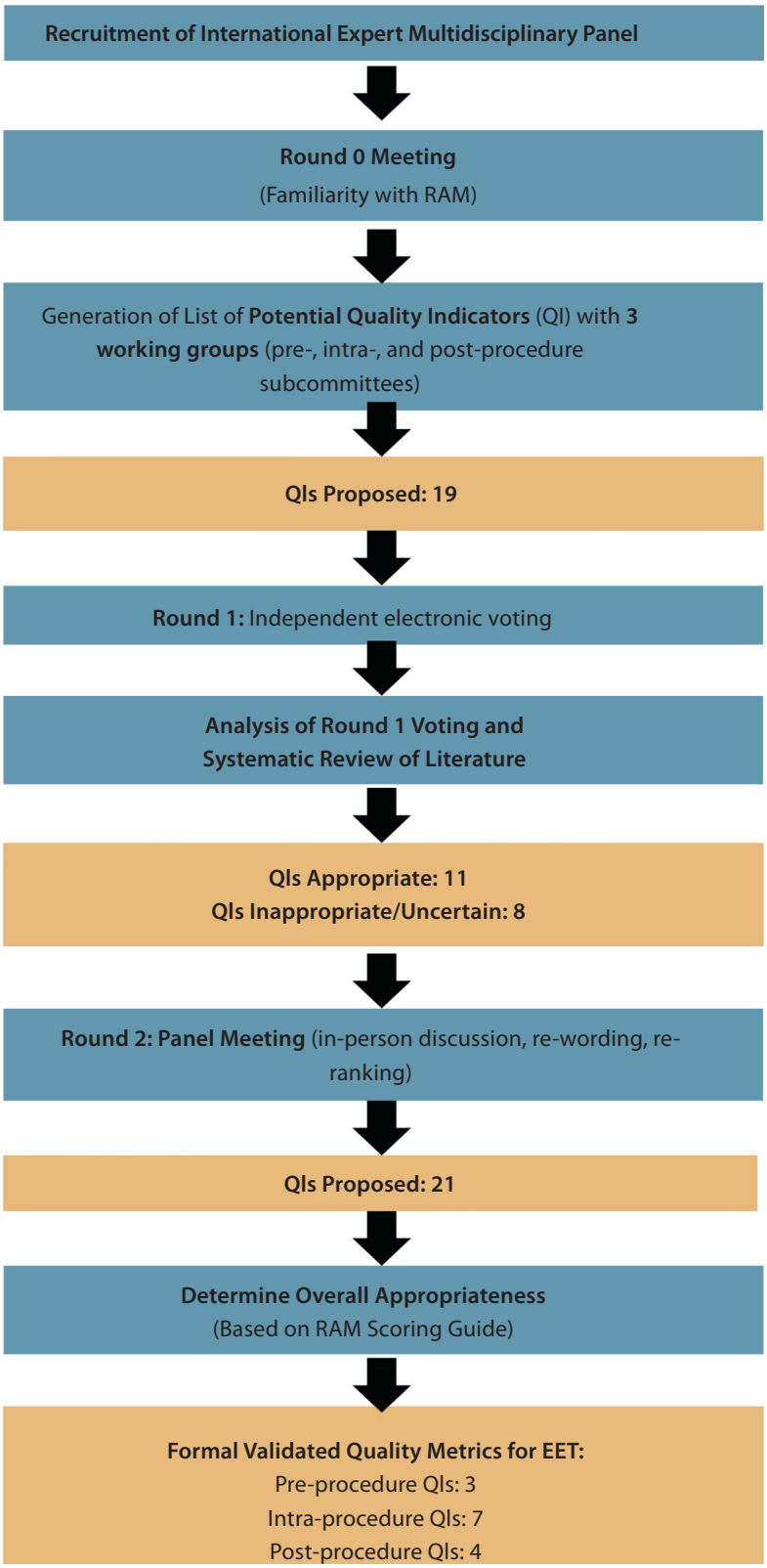

Figure 1. Study design used to develop quality indicators for endoscopic eradication therapy in Barrett's esophagus–related neoplasia.

supplementary material (Appendix 3). The results of the search were classified as pertinent to before, during, and post–procedure workgroups.

Round 2 meeting: Discussion of potential quality indicators, rewording, and changing rankings. This face-to-face meeting among the expert panel members was conducted on November 13, 2015 in Chicago, Illinois. Before this meeting, a folder with individual summary results of Round 1 rankings with overall aggregated ranking results was provided to the panelists. In addition, a summary of the systematic review and details regarding the RAM were provided (operational definitions of levels of appropriateness and methods to assess levels of disagreement). This meeting involved presentations by an expert RAM methodologist and the chief statistician, followed by a detailed discussion among all expert panel members with regard to each proposed quality indicator. Based on the systematic review for each indicator, panelists discussed the available evidence and areas of disagreement followed by rewording and redefining the numerator and denominator when applicable. New quality indicators could be proposed during this meeting. Next, panel members independently re-ranked each of the proposed quality indicators for their perceived levels of appropriateness and provided a threshold for each indicator that reflected the rate at which physicians should fulfill the quality indicator in clinical practice (Appendix 4, available online at www.giejournal.org). No attempt was made to force the panel to consensus. All panel members completed an after-meeting questionnaire (Appendix 5, available online at www.giejournal.org) that addressed questions related to Round 1 (addressing ease of task and consistency), the literature review (addressing objectivity and comprehensiveness), the Round 2 face-to-face meeting (addressing group discussion and impact of feedback on ratings), and their overall experiences as panel members (addressing comparison of personal ratings to overall appropriateness and ability of the group to lead to an official set of recommendations for quality indicators in EET).

Statistical analysis

Rounds 1 and 2 responses for each question were summarized and provided to the participants. At the end of Round 1, panelists were provided a histogram for each question, with their individual rating and distribution of all responses across the 9 categories including (1) the median response, (2) measures of spread of the responses, and (3) 3 measures of appropriateness. The measures of spread included the count of responses in each 3-point region (1-3, 4-6, and 7-9), and the mean absolute deviation from the median. Appropriateness of a metric (typically classified as appropriate, uncertain, or inappropriate) was based on (1) the median rating and (2) whether or not panelists agreed or not, as measured by the amount of dispersion of the ratings. Because there is no consensus on the best approach to measuring dispersion, and different methods can result in different conclusions, the participants were provided 3 different versions: BIOMED classical, *P* value, and interpercentile range adjusted for symmetry (IPRAS). The BIOMED classical definition is an extension of the RAND classical definition beyond 9 panel members and defines for different panel sizes the maximum number of responses that are allowed to fall outside the 3-point region that contains the median, in order to conclude agreement. The *P* value method definition of agreement is the result of a binomial hypothesis test that 80% of the ratings are within the 3-point region containing the median. Finally, the IPRAS is based on the nonparametric measure of spread, the interpercentile range (IPR; the difference between the 70th and 30th percentiles). After adjusting for the lack of symmetry in the responses, if the resulting IPRAS is less than the IPR, there is disagreement. A measure was considered appropriate if the metric met the definition of appropriateness by using all defined statistical methods. All statistical analyses were performed by using SAS v. 9.4 (SAS Institute, Cary, NC).

RESULTS

A total of 33 international experts were invited, of whom 27 (81.8%) participated in Round 1 and 19 (57.5%) participated in both Rounds 1 and 2. At the end of Round 1, 11 of 19 proposed quality indicators were ranked as appropriate and 8 as uncertain. At the end of Round 2, 14 of 21 quality indicators were ranked as appropriate, 5 as uncertain, and 2 as inappropriate. Tables 1 to 3 highlight the results of Round 2 ranking along with suggested benchmarks for each appropriate and valid measure. The valid quality indicators were categorized into the pre-procedure (n = 3), intra-procedure (n = 7), and post-procedure (n = 4) quality indicator categories.

Appropriate pre-procedure quality indicators

The pre-procedure period includes all contact between the endoscopist with the patient before administration of sedation. The following measures were deemed appropriate pre-procedure quality indicators.

For patients in whom a diagnosis of dysplasia has been made, the rate at which the reading is made by a GI pathologist or confirmed by a second pathologist before EET is begun.

Type of measure: Process

Performance target: 90%

TABLE 1. Appropriate quality indicators from the RAND/University of California, Los Angeles Appropriateness Method measures with median score, number of experts in each category range, and a suggested threshold benchmark with range

	Quality indicator	Median score	No. of experts 1-3 range	No. of experts 4-6 range	No. of experts 7-9 range	MAD-M score	Threshold
Pre-procedure	The rate at which the reading is made by a GI pathologist or confirmed by a second pathologist before EET is begun for patients in whom a diagnosis of dysplasia has been made.	9	1	1	17	0.84	90 (75, 100)
	Centers in which EET is performed should have available HD-WLE and expertise in mucosal ablation and EMR techniques.	8	0	1	18	0.79	N/A
	The rate at which documentation of a discussion of the risks, benefits, and alternatives to EET is obtained from the patient prior to treatment.	9	0	0	19	0.16	>98 (85, 100)
Intra-procedure	The rate at which landmarks and length of BE is documented (eg, Prague grading system) in patients with BE before EET.	8	1	1	17	1.1	90 (75, 100)
	The rate at which the presence or absence of visible lesions is reported in patients with BE referred for EET.	8	0	2	17	0.79	90 (60, 100)
	The rate at which the BE segment is inspected by using HD-WLE.	9	0	2	17	0.63	95 (0, 100)
	The rate at which complete endoscopic resection (en bloc resection or piecemeal) is performed in patients with BE with visible lesions.	9	0	0	19	0.42	90 (80, 100)
	The rate at which a defined interval for subsequent EET is documented for patients undergoing EET who have not yet achieved complete eradication of intestinal metaplasia.	8	1	2	16	1.1	90 (0, 100)
	The rate at which complete eradication of dysplasia is achieved by 18 months in patients with BE-related dysplasia or intramucosal cancer referred for EET.	9	0	0	19	0.42	80 (70, 95)
	The rate at which complete eradication of intestinal metaplasia is achieved by 18 months in patients with BE-related dysplasia and intramucosal cancer referred for EET.	9	0	2	17	0.89	70 (50, 80)
Post-procedure	The rate at which a recommendation is documented for endoscopic surveillance at a defined interval for patients who achieve complete eradication of intestinal metaplasia.	8	0	1	18	0.68	90 (50, 100)
	The rate at which biopsies of any visible mucosal abnormalities are performed during endoscopic surveillance after EET.	8	0	3	16	1.21	95 (50, 100)
	The rate at which an anti-reflux regimen is recommended after EET.	8	1	2	16	1.21	90 (50, 100)
	The rate at which adverse events are being tracked and documented in individuals after EET.	8	2	0	17	1.21	90 (50, 100)

BIOMED, *P* value, and interpercentile range adjusted for symmetry (IPRAS) values were determined appropriate in all cases.
MAD-M, Mean absolute deviation from the median; *EET*, endoscopic eradication therapy; *HD-WLE*, high-definition white light endoscopy; *N/A*, not applicable; *BE*, Barrett's esophagus.

Evidence summary: The presence and grade of dysplasia remains the best predictor of the risk of developing malignancy in patients with BE.[42] However, there can be significant heterogeneity in the pathologic interpretation of dysplasia, with substantial rates of overdiagnosis of LGD and HGD reported.[43-46] The histologic features of LGD overlap in some regard with regenerative changes. As a result, significant interobserver variability exists among pathologists regarding the interpretation of LGD. Agreement is better when HGD and/or cancer versus nondysplastic BE is evaluated.[6,47-51] Interobserver agreement is marginally better among specialized GI pathologists compared with general pathologists.[52,53]

The Vienna classification[54] was developed to improve interobserver variability in grading dysplasia among pathologists. It is composed of 5 categories: (1) negative for neoplasia and/or dysplasia, (2) indefinite for neoplasia and/or dysplasia, (3) noninvasive low-grade neoplasia (low-grade adenoma and/or dysplasia), (4) noninvasive high-grade neoplasia (high-grade adenoma and/or dysplasia, noninvasive carcinoma, and suspicion of invasive carcinoma), and (5) invasive neoplasia (intramucosal carcinoma, submucosal carcinoma or beyond).[54] Additional techniques and/or variables that have been investigated include the use of immunohistochemistry and obtaining larger specimens via

TABLE 2. Appropriate quality indicators with numerator, denominator, type of measure (process/outcome), and suggested median threshold benchmark with range

	Metric	Numerator	Denominator	Type	Threshold
Pre-procedure	The rate at which the reading is made by a GI pathologist or confirmed by a second pathologist before EET is begun for patients in whom a diagnosis of dysplasia has been made.	No. of patients whose dysplasia diagnosis is made by a GI pathologist or a second pathologist before EET is begun.	All patients who receive EET for treatment of dysplasia.	Process	90 (75, 100)
	Centers in which EET is performed should have available HD-WLE and expertise in mucosal ablation and EMR techniques.	N/A	N/A	Process	N/A
	The rate at which documentation of a discussion of the risks, benefits, and alternatives to EET is obtained from the patient prior to treatment.	No. of patients who undergo EET with clear documentation of risks, benefits, and EET alternatives.	All patients who receive EET.	Process	>98 (85, 100)
Intra-procedure	The rate at which landmarks and length of BE is documented (eg, Prague grading system) in patients with BE before EET.	No. of patients who undergo EET after documented landmarks and length of BE are established.	All patients who receive EET.	Process	90 (75, 100)
	The rate at which the presence or absence of visible lesions is reported in patients with BE referred for EET.	The no. of times endoscopists specifically state that either there were no lesions seen on EGD or describe lesions if they exist.	All endoscopies in patients with BE being considered for EET.	Process	90 (60, 100)
	The rate at which the BE segment is inspected by using HD-WLE.	No. of times specific documentation that HD-WLE examination was performed	All endoscopies in patients with BE being considered for EET.	Process	95 (0, 100)
	The rate at which complete endoscopic resection (en bloc resection or piecemeal) is performed in patients with BE with visible lesions.	No. of times a report reads that complete mucosal resection was performed in patients with visible lesions.	All patients with BE with visible lesions being considered for EET.	Process	90 (80, 100)
	The rate at which a defined interval for subsequent EET is documented for patients undergoing EET who have not yet achieved CE-IM.	All patients undergoing EET who have not yet achieved CE-IM who have a documented EET interval recommendation.	All patients undergoing EET who have not yet achieved CE-IM.	Process	90 (0, 100)
	The rate at which CE-N is achieved by 18 months in patients with BE-related dysplasia or intramucosal cancer referred for EET.	Patients who are referred for EET for treatment of BE-related dysplasia or intramucosal cancer who achieve CE-D within 18 months.	All patients who are referred for EET for treatment of BE-related dysplasia or intramucosal cancer.	Outcome	80 (70, 95)
	The rate at which CE-IM is achieved by 18 months in patients with BE-related dysplasia and intramucosal cancer referred for EET.	Patients who are referred for EET for treatment of BE-related dysplasia or intramucosal cancer who achieve CE-IM within 18 months.	All patients who are referred for EET for treatment of BE-related dysplasia or intramucosal cancer.	Outcome	70 (50, 80)

(*continued on the next page*)

EMR.[55,56] Importantly, the number of pathologists who agree on the presence of dysplasia appears to correlate with an increased risk of progression to cancer.[44,45,57] Given these data, the panel strongly agreed that a second pathologist (preferably a GI pathologist) confirm all diagnoses of BE-related dysplasia before EET is initiated.

Centers where EET is performed should have available high-definition white light endoscopy

TABLE 2. Continued

	Metric	Numerator	Denominator	Type	Threshold
Post-procedure	The rate at which a recommendation is documented for endoscopic surveillance at a defined interval for patients who achieve CE-IM.	No. of patients with CE-IM who have a documented surveillance interval recommendation.	No. of patients who achieve CE-IM.	Process	90 (50, 100)
	The rate at which biopsies of any visible mucosal abnormalities are performed during endoscopic surveillance after EET.	No. of surveillance procedures with biopsies of visible abnormalities.	No. of surveillance procedures with visible abnormalities present.	Process	95 (50, 100)
	The rate at which an anti-reflux regimen is recommended after EET.	Patients who are recommended for an anti-reflux regimen after EET.	All patients who have received EET.	Process	90 (50, 100)
	The rate at which adverse events are being tracked and documented in individuals after EET.	Adverse events that are tracked and documented.	All endoscopic procedures involving EET.	Process	90 (50, 100)

EET, endoscopic eradication therapy; *HD-WLE*, high-definition white light endoscopy; *N/A*, not applicable; *BE*, Barrett's esophagus; *CE-IM*, complete eradication of intestinal metaplasia; *CE-N*, complete eradication of neoplasia; *CE-D*, complete eradication of dysplasia.

(HD-WLE) and expertise in mucosal ablation and EMR techniques.

Type of measure: Process

Performance target: N/A

Evidence summary: Endoscopists performing EET should have access to the appropriate equipment needed to optimize dysplasia detection, including high-definition endoscopes as suggested by recent guidelines.[4] In addition, the capability to perform both ablative techniques and EMR are essential, given the high frequency of focal lesions and nodular disease in patients referred for EET. Hence, it has been proposed that centers performing endoscopic ablation have the capability to perform EMR.[4,24]

The rate at which documentation of a discussion of the risks, benefits, and alternatives to EET is obtained from the patient before a course of treatment is begun.

Type of measure: Process

Performance target: >98%

Evidence summary: Before EET is performed, it is essential that the informed consent process include discussions of the risk of progression to cancer and/or dysplasia (if not already present), appropriate surveillance and treatment options, risks and/or benefits of each management strategy, and a summary of the frequency and duration of follow-up.[11] It has been recommended that this discussion be held during an outpatient clinic visit.[24] However, given the distance many patients travel to undergo EET at expert and/or referral centers, the optimal method of obtaining informed consent is uncertain. Potential options include a dedicated outpatient consultation, a pre-procedure telephone call, or a detailed discussion before the planned EET procedure. All panelists felt this was an important step before EET is begun.

Appropriate intra-procedure quality indicators

The intra-procedure period extends from the administration of sedation to the removal of the endoscope. This period includes all the technical aspects of the procedure including completion of the examination and therapeutic maneuvers (process measures) along with outcomes measures related to therapeutic maneuvers. The following were considered appropriate intra-procedure quality indicators.

The rate at which landmarks and length of BE is documented by using the Prague criteria[58] in patients with BE before EET.

Type of measure: Process

Performance target: 90%

Evidence summary: Current guidelines support the use of the Prague criteria in describing the extent of metaplastic change along with the location of the diaphragmatic hiatus, gastroesophageal junction, and squamocolumnar junction in the endoscopy report.[4,5,24] The Prague C & M criteria were developed by the International Working Group for Classification of Oesophagitis by using validated, explicit, consensus-driven criteria.[58] This includes assessment of the circumferential and maximal extent of the visualized columnar-lined esophagus as well as endoscopic landmarks such as a diaphragmatic hiatal pinch and the proximal extent of the gastric folds. Reliability coefficients for a BE segment $\geq$1 cm were high (0.72), whereas that for a BE segment <1 cm was low (0.22). These criteria have been validated in multiple studies among experts and trainees and among Western and Asian endoscopists.[59-62] A recent study, currently in abstract form, showed that the before-EET BE extent as measured by the Prague criteria[58] is associated with the rate of achieving CE-IM, and this was true for both the circumferential and maximal parameters.[63] Because this system does

TABLE 3. Quality indicators ranked as inappropriate or uncertain using the RAND/University of California, Los Angeles Appropriateness Method measures

	Quality indicator	Type	Median score	No. of experts 1-3 range	No. of experts 4-6 range	No. of experts 7-9 range	MAD-M score	BIOMED	*P* value	IPRAS	Threshold
Pre-procedure	The rate at which EET is performed by trained endoscopists (determined by volume of cases per year).	Process	5	6	9	4	1.79	Uncertain	Uncertain	Uncertain	90 (0, 100)
Intra-procedure	The rate at which the BE segment is inspected by using optical chromoendoscopy.	Process	5	5	7	7	1.94	Uncertain	Uncertain	Uncertain	75 (0, 100)
	The rate at which appropriate and recommended ablation steps are implemented during RFA.	Process	4	9	6	4	2.26	Uncertain	Uncertain	Uncertain	75 (0, 100)
Post-procedure	The rate at which the first endoscopy after CE-IM with biopsies is performed within 6 months after completion of CE-IM.	Process	5	9	7	3	2.37	Uncertain	Uncertain	Uncertain	50 (0, 90)
	The rate at which the exact distance from the incisors and endoscopic appearance of the new squamocolumnar junction, obtained by using HD-WLE and optical chromoendoscopy after CE-IM, is reported.	Process	3	11	6	2	1.58	Uncertain	Inappropriate	Inappropriate	60 (0, 90)
	The rate at which symptoms of gastroesophageal reflux are documented after EET.	Outcome	3	10	5	4	2.42	Uncertain	Inappropriate	Inappropriate	50 (0, 90)
	The rate at which recurrence of intestinal metaplasia is tracked and documented in patients who achieve CE-IM.	Outcome	8	1	6	12	1.58	Uncertain	Appropriate	Appropriate	80 (50, 100)

MAD-M, Mean absolute deviation from the median; *IPRAS*, interpercentile range adjusted for symmetry; *EET*, endoscopic eradication therapy; *BE*, Barrett's esophagus; *RFA*, radio frequency ablation; *CE-IM*, complete eradication of intestinal metaplasia; *HD-WLE*, high-definition white light endoscopy.

not account for columnar-lined epithelium (islands) that are not continuous with the squamocolumnar junction, it may underestimate the true maximal extent of BE and potentially the highest grade of dysplasia.[64,65] The panelists acknowledged that improved patient outcomes (eg, higher rates of CE-IM) from using endoscopic classification systems for BE such as the Prague criteria[58] have not been established by formal investigation, and patients with any extent of intestinal metaplasia currently are managed similarly.[5]

The rate at which the presence or absence of visible lesions is reported in patients with BE referred for EET.

Type of measure: Process

Performance target: 90%

Evidence summary: Visible lesions within the BE segment should be described clearly and include nodularity, ulceration, plaques, areas of depression, strictures, and areas of mucosal discoloration, no matter how subtle when present and regardless of how they are detected (HD-WLE or optical chromoendoscopy).[66] The Paris classification[67] provides a uniform grading system for visible lesions identified during upper endoscopy. Visible lesions are described as follows: protruded lesions, 0-Ip (pedunculated) 0-Is (sessile) and flat lesions, 0-IIa (superficially elevated), 0-IIb (flat), 0-IIc (superficially depressed), and 0-III (excavated).[67] Lesions classified as 0-Is, 0-IIc, and 0-III are most likely to harbor invasive cancer, whereas 0-IIa and 0-IIb are unlikely to contain invasive cancer. In a study that included 344 patients with BE with 380 neoplastic lesions who were referred for EET, type IIa (37%) and IIb (28%) were most commonly described. The mean kappa value for interobserver agreement was 0.86 and for intraobserver agreement was 0.89 by using still images.[68] A retrospective study showed that most endoscopically resected early BE neoplasia are type 0-II, and submucosal infiltration is more often encountered in type 0-I and 0-IIc lesions.[69] However, unlike the Japanese classification for early gastric cancer, use of the Paris classification[67] has not been evaluated in BE-related neoplasia as a prognostic tool. The British Society of Gastroenterology guidelines recommend describing all visible lesion morphology by using the Paris classification because it provides an indication of the likelihood of invasive cancer and aids communication between clinicians, although presently it cannot be used to predict prognosis.[24] The panelists acknowledged that there was a lack of data demonstrating improved patient outcomes with use of the Paris classification in describing visible lesions.

The rate at which the BE segment is inspected by using HD-WLE.

Type of measure: Process

Performance target: 95%

Evidence summary: The use of HD-WLE should be considered as the standard of care and the first critical step in the evaluation of patients with BE undergoing surveillance or being considered for EET. Although there are no data from randomized controlled trials comparing HD-WLE to standard definition white light endoscopy, this recommendation has been endorsed by guidelines and consensus documents.[4,5,24] Indirect evidence from 3 prospective trials and a single retrospective study suggests that HD-WLE is more sensitive than standard definition white light endoscopy in the detection of BE-related neoplasia.[70-73]

The rate at which endoscopic resection (defined as en bloc resection or piecemeal) is performed in patients with BE with visible lesions.

Type of measure: Process

Performance target: 90%

Evidence summary: The role of EMR as a diagnostic and/or staging and therapeutic tool in the management of BE-related neoplasia is well-described. Several studies have demonstrated that EMR results in a change in the histopathologic diagnosis of patients with BE with neoplasia referred for EET.[74-77] Results from a multicenter cohort study showed that EMR resulted in a change in diagnosis in 30% of patients with BE with early neoplasia.[78] In addition, provision of a larger specimen results in an improvement in interobserver agreement among pathologists, compared with biopsy specimens.[56,79] Consistent with published guidelines,[4,5,24,80] all panelists agreed that EMR should be performed in patients with nodularity within the BE segment as the initial diagnostic (to determine the T-stage and/or grade of dysplasia) and therapeutic maneuver.

Among patients undergoing EET who have not yet achieved CE-IM, the rate at which a defined interval for subsequent EET is documented.

Type of measure: Process

Performance target: 90%

Evidence summary: Although there are no data comparing outcomes (CE-IM, recurrence rates, progression rates to cancer) in patients in whom a defined interval for subsequent EET is documented, the panelists agreed that this was an important aspect in the management of BE-related neoplasia in patients undergoing EET. Caution was exercised against evaluating the rate at which patients undergo EET at specified time intervals (every 2-3 months) to account for variable clinical practices and noncompliant patients. A study evaluating national practice patterns showed that in both academic and community practices, patients are treated with radiofrequency ablation (RFA) at approximately 3-month intervals.[81]

The rate at which complete eradication of neoplasia is achieved by 18 months in patients with BE-related dysplasia or intramucosal cancer referred for EET.

Type of measure: Outcome

Performance target: 80%

The rate at which CE-IM is achieved by 18 months in patients with BE-related dysplasia and intramucosal cancer referred for EET.

Type of measure: Outcome

Performance target: 70%

Evidence summary: Several studies (including data from randomized controlled trials) have demonstrated the following: (1) Patients with BE-related HGD, intramucosal cancer (T1a) and true LGD are ideal candidates for EET, and (2) current therapies available (EMR and RFA) are effective, with a favorable safety profile.[21] The goal of

EET is complete eradication of dysplasia (CE-D) and CE-IM; CE-D alone is not an optimal endpoint for EET given the risk of metachronous neoplasia (seen in up to 30% of cases after EMR).[16]

Two randomized controlled trials have shown that RFA achieves a high rate of CE-D and CE-IM and reduces the risk of progression to cancer.[13,82] A recent multicenter randomized controlled trial randomized 136 patients with a confirmed diagnosis of LGD to either RFA or surveillance (control). During a 3-year follow-up since randomization, ablation reduced the risk of progression to HGD or EAC by 25% (ablation 1.5% vs controls 26.5%; 95% confidence interval [CI], 14.1-35.9; $P < .001$) and risk of progression to EAC by 7.4% (1.5% vs 8.8%; 95% CI, 0-14.7; $P = .03$). CE-D and CE-IM rates were significantly higher in the ablation group (CE-D 92.6% vs 27.9%, CE-IM 88.2% vs 0; $P < .001$).[82] Results from the AIM dysplasia trial demonstrated a significantly higher rate of CE-D and CE-IM in patients undergoing ablation compared with those who received a sham procedure (LGD: CE-D 90.5% vs 22.7%, HGD: CE-D 81% vs 19%; $P < .001$; CE-IM over all 77.4% vs 2.3%; $P < .001$). Patients in the ablation group had less disease progression (3.6% vs 16.3%; $P = .03$) and fewer cancers (1.2% vs 9.3%; $P = .04$).[13] Durability data from this study showed that at 3 years, CE-D and CE-IM were noted in 98% and 91% of patients, respectively, allowing for maintenance RFA and >85% and >75%, respectively, without maintenance RFA. The rate of progression to EAC was 0.55%/patient-years and to any neoplasia was 1.37%/patient-years.[83] At 5 years, CE-D and CE-IM were noted in 99% and 90% of patients, respectively.[84] A European multicenter study (EURO-II) that reported long-term outcomes of combined EMR and RFA for patients with BE with HGD and/or EAC showed that CE-D was achieved in 121 of 132 patients (92%) and CE-IM in 115 of 132 patients (87%), based on an intention-to-treat analysis.[85] The Amsterdam group also reported long-term data on 54 patients enrolled in 4 consecutive cohort studies and showed CE-D and CE-IM rates of 90% at 5 years of follow-up.[18] A single-center retrospective study that assessed outcomes associated with multiple-modality EET showed CE-D and CE-IM rates of 95% and 83%, respectively.[86] Data from the UK patient registry compared CE-D and CE-IM rates between 2 time intervals (2008-2010 and 2011-2013) and reported improvement in both CE-D and CE-IM rates between the 2 time periods, from 77% and 56% to 92% and 83%, respectively ($P < .0001$).[87] Results from the US RFA Registry, which included 5521 patients, showed that CE-IM was achieved in 85% of patients.[88] Finally, a systematic review and meta-analysis (18 studies, 3802 patients) published in 2013 assessed the efficacy and durability of RFA for patients with dysplastic and nondysplastic BE and showed a CE-D rate of 91% (95% CI, 87%-95%) and CE-IM rate of 78% (95% CI, 70%-86%).[89]

In addition to an extensive discussion regarding these published data, the panel members drafted these quality indicators after also accounting for patient noncompliance and those referred to surgery. The decision to specify an 18-month time period to achieve CE-D and CE-IM was made to make these quality indicators more specific and after accounting for the median number of sessions required to achieve CE-IM.

Appropriate post-procedure quality indicators

The post-procedure period, for the purpose of this document, extends from the time of CE-IM to subsequent follow-up. This includes surveillance endoscopy and biopsy strategies and the documentation and tracking of adverse events. The following measures were considered appropriate post-procedure quality indicators.

Among patients who achieve CE-IM, the rate at which a recommendation is documented for endoscopic surveillance at a defined interval.

Type of measure: Process

Threshold: 90%

Evidence summary: The reported rates for CE-IM after a combination of EMR and RFA ranged between 72% and 97%.[18,19,83,88,90-96] Despite the effectiveness of EET, recurrence rates of 0% to 15% for dysplasia and 5% to 39.5% for intestinal metaplasia are reported,[16,17,19,20,83,90-92,97-101] raising concern regarding the durability of EET. Therefore, current guidelines recommend endoscopic surveillance with biopsies after EET. Although there are no data to validate specific surveillance intervals, expert opinion suggests an initial endoscopic examination at 3 to 6 months after CE-IM is achieved, followed by similar surveillance intervals based on pretreatment histology. Consistent with currently available data and society guidelines, all panelists agreed that endoscopic surveillance was essential after EET was completed for BE-related neoplasia. However, given the paucity of evidence directing the intervals between these surveillance examinations, no quality indicator could be developed for interval length.

During endoscopic surveillance after EET, the rate at which biopsies of any visible mucosal abnormalities are performed.

Type of measure: Process

Threshold: 95%

Evidence summary: As reviewed under metric *Appropriate post-procedure quality indicators (Among patients who achieve CE-IM, the rate at which a recommendation is documented for endoscopic surveillance at a defined interval),* the panel agreed that endoscopic surveillance after EET is essential. Consistent with the most recent ACG guidelines for BE, the panel uniformly agreed that for the confirmation of persistent or recurrent neoplasia or metaplasia, histology is required.[4] This recommendation is consistent with the prerequisite of histology in the United States for diagnosis of intestinal metaplasia during any endoscopy. There is currently no standardized technique for

surveillance biopsies after EET. Although previous studies have reported sampling of the new squamocolumnar junction along with implementation of the Seattle protocol for sampling every 1 to 2 cm in 4-quadrant fashion for the entire length of pretreatment BE segment, this remains an expert opinion.[102-104] Other data suggest that there is no additional yield from biopsies >2 cm from the gastroesophageal junction in the absence of a visible lesion in the neosquamous epithelium and that surveillance biopsies of normal squamous neosquamous tissue beyond 2 cm from the gastroesophageal junction are unnecessary.[99] As such, the panel agreed that surveillance biopsies are needed and should at the very least target visible mucosal abnormalities, but the panel otherwise declined to endorse a specific surveillance endoscopy strategy.

The rate at which an anti-reflux regimen is recommended after EET.

Type of measure: Process

Threshold: 90%

Evidence summary: The role of uncontrolled gastroesophageal reflux in the progression of BE to EAC is established.[3,105-107] Nearly 25% of patients with BE did not have normalization of intraesophageal pH when they were studied with pH monitoring.[107-109] Before EET, the clinical importance of ongoing reflux was debated, because most patients achieved effective symptom control with proton pump inhibitor therapy. However, it has been reported that uncontrolled reflux is associated with persistence of intestinal metaplasia after RFA.[110] A recent study demonstrated that hiatal hernia size >4 cm was a significant predictor of recurrence of BE with HGD or intramucosal carcinoma,[111] and another study found that effective intraesophageal pH control was associated with improved RFA outcomes.[112] Together, these data suggest that uncontrolled reflux may be an important determinant of recurrent intestinal metaplasia after EET and emphasize the importance of maintaining an effective anti-reflux regimen after EET. The most recent ACG guidelines recommend continued medical anti-reflux therapy, with a goal of minimizing the frequency of reflux symptoms (<1 time per week) and achieving the absence of esophagitis on endoscopy.[4] Although there are no data to suggest a role for anti-reflux surgery, expert opinion suggests selective use of fundoplication for patients with persistent or refractory symptoms that are clearly related to gastroesophageal reflux. Based on these data and guidelines, the majority of the panel agreed that recommending an anti-reflux regimen is important after completion of EET.

The rate at which adverse events are being tracked and documented in individuals post EET.

Type of measure: Process

Threshold: 90%

Evidence summary: Despite the relative safety of EET for BE, significant adverse events can occur and must be recognized. A recent systematic review and meta-analysis of 37 published manuscripts found a pooled rate of adverse events for EET of 8.8% (95% CI, 6.5%-11.9%).[21] The overall rate for stricture formation was 6%, and the rate of perforation was 0.6%. The majority of adverse events occurred in patients who had EMR. The addition of EMR to RFA for completion of EET resulted in a 4.4-fold increase in the incidence of adverse events over RFA alone. The meta-analysis was limited by the heterogeneity of the studies, whereby it was difficult to separate adverse events that could be directly attributed to RFA or EMR. Based on this systematic review and meta-analysis, along with previously reported rates of adverse events, the panel strongly supported the practice of actively tracking and documenting adverse events occurring after completion of EET. The panel debated the setting of benchmark rates of adverse events (for instance, an post-procedure stricture rate of $\leq$8%). Although such an outcome quality indicator measure would be highly desirable, the heterogeneity of reported outcomes data on adverse events coupled with the wide diversity of patient populations receiving EET made it impossible to solidly ground such measures in data. Because an inappropriately benchmarked quality indicator could have perverse consequences, including "cherry-picking" of patients at low risk for adverse events, the panel chose to focus for now on the tracking and documentation of these events, as opposed to the proportion with them, in the hope that in the future this process measure might be replaced by an outcome measure adequately supported by data.

Inappropriate or uncertain quality indicators

The quality indicators found to be inappropriate or uncertain after final ranking was completed are depicted in Table 3.

After-round 2 survey results. Results from the after-Round 2 survey completed by 16 of the panel members are highlighted in Table 4. With regard to the Round 1 ranking, 93.8% of the panelists felt that the instructions were clear, and 75% found the task easy. The majority of the panelists felt that the literature review was objective (81.3%) and informative (87.5%). All respondents agreed that the moderator functioned effectively as a group leader and that the discussion was informative. The discussion during the Round 2 face-to-face meeting influenced the rankings for 81.2% of panelists. Finally, the vast majority of panelists felt that their own ratings reflected the overall appropriateness (87.5%) and that this panel process can lead to an official set of recommendations for quality indicators in EET (81.3%).

DISCUSSION

Several advances in the field of EET have resulted in a paradigm shift in the management of patients with BE-related neoplasia. However, although available data support the increasing use of EET in patients with BE-related neoplasia, quality indicators for EET are lacking. Defining

TABLE 4. After-study questionnaire results

Question	Not at all/little	Somewhat	Pretty much/very much
Round 1	n (%)	n (%)	n (%)
How easy did you find the task?	0	4 (25)	12 (75)
How onerous did you find the task?	12 (75)	1 (6.3)	3 (18.8)
How clear were the instructions?	0	1 (6.3)	15 (93.8)
How inconsistent do you believe they were?	12 (75)	3 (18.8)	1 (6.3)
How much did it influence your first round ratings? (due to effects of fatigue, memory, different times to rate, format of instrument, etc)	6 (37.6)	9 (56.3)	1 (6.3)
How useful did you find the online REDCap rating system?	0	3 (18.8)	13 (81.2)
Literature review			
How completely did you read it?	2 (12.5)	5 (31.3)	9 (56.3)
How objective was it?	0	3 (18.8)	13 (81.3)
How informative was it?	0	2 (12.5)	14 (87.5)
How much did it influence your second round ratings?	1 (6.3)	7 (43.8)	8 (50.1)
Panel meeting (Round 2)			
How many hours did you spend reading it?			
How well did the moderator function as a group leader?	0	0	16 (100)
How informative was the discussion?	0	0	16 (100)
How argumentative was the discussion?	5 (31.3)	8 (50)	3 (18.8)
How much did the feedback from the first round ratings influence your second round ratings?	2 (12.5)	5 (31.3)	9 (56.3)
How much did the discussion influence your second round ratings?	0	3 (18.8)	13 (81.2)
Overall experience			
How well do you believe your own ratings reflect the appropriateness of quality metrics for EET?	0	2 (12.5)	14 (87.5)
How well do you believe the panel's ratings will reflect the appropriateness of quality metrics for EET?	0	3 (18.8)	13 (81.3)
How much do you believe that this panel process can lead to an official set of recommendations for quality metrics in EET?	0	3 (18.8)	13 (81.3)

EET, Endoscopic eradication therapy.

quality indicators may help to ensure the delivery of high-quality care. In this era of value-based and quality-based healthcare, the development of quality indicators that benchmark performance is critical. The quality of health care can be measured by comparing the performance of an individual or a group of individuals with an ideal or benchmark.[113] Quality indicators often are reported as ratios between the incidence of correct performance and the opportunity for correct performance or as the proportion of interventions that achieve a predefined goal.[114] Quality indicators are held to a higher standard compared with published guidelines, and nonadherence to a quality indicator reflects suboptimal care.[31] In this current health care landscape, the Department of Health and Human Services aims to reform health care delivery through increased use of incentives to foster higher value care with a goal of linking 90% of all Medicare fee-for-service payments to quality or value by 2018.[115,116] By using reporting systems such as the Merit-based Incentive Payment System (the latest transformation of the Physician Quality Reporting System), physicians will be required to track and report their performances during endoscopy, and reimbursement will be linked to reporting and performance on defined quality indicators. The National Quality Forum has established a framework for several high-quality measures. These key components include the following: important to measure and report—evidence based, scientific acceptability—reliability and validity, feasibility, usability, and comparison to related or competing measures.[117] Quality indicators need to be precisely defined with a clear description of the numerator and denominator along with the performance benchmark. In addition, any initiative that generates quality indicators should not only focus on process

measures but also on outcomes measures. Finally, gastroenterologists and national gastroenterology societies need to drive the development of quality indicators rather than accept potentially flawed indicators introduced by administrative or governmental agencies.[27,115] The development of quality indicators for EET by using a formal validated methodology is reported in this document.

Quality is a key focus for gastroenterology, driven by a desire to promote best practice among gastroenterologists and to foster evidence-based care. The development of quality indicators for EET has several important implications. Compliance with this group of quality indicators has the potential to improve quality of care, reduce variability in health care, and ultimately improve patient outcomes. Establishing quality indicators is also in line with the current health care landscape as it transitions from a volume-based to a value-based and quality-based system. The current study methodology does not provide the level of evidence and strength of recommendation. However, it should be recognized that the goal of this document is not to promote or create specific practice guidelines but rather to provide baseline quality indicators by which patients, physicians, payers, and institutions may assess the quality of care related to EET for patients with BE-related neoplasia. The strengths of this initiative include (1) the use of a formal, well-described methodology that included an international panel of experts to develop appropriate and valid quality indicators in EET, (2) the development of well-defined quality indicators with inclusion and exclusion criteria for the numerator and denominator for the indicator, (3) the definition of threshold benchmarks for clinical practice, and (4) the development of outcomes measures that are tied to outcomes of interest such as complete eradication rates and adverse events.

As the field continues to expand and evolve, quality indicators undoubtedly will need to be refined and updated. Future studies need to assess the impact of implementation of these quality indicators on relevant patient outcomes (CE-IM, progression to cancer, adverse events, and mortality). The next steps will include addressing challenges in the process of measurement and evaluation. The feasibility of incorporating these quality indicators into national repositories and linking them to public reporting and reimbursement will need to be evaluated. The GI Quality Improvement Consortium, Ltd, a joint initiative of the ACG and the ASGE, established a data repository and benchmarking tool. This registry has an expanding colonoscopy and EGD database and may provide the infrastructure required for such an endeavor. Future work also needs to address the issues of implementation costs, measurement fatigue, and unintended consequences of implementation of quality indicators.[115,118,119]

This process identified several areas of future research related to quality indicators that were considered important but not ranked as appropriate for various reasons—lack of high-quality data being the most common reason. Performance of EET by trained endoscopists (determined by volume of case per year) was considered as a quality indicator. However, limited data exist on the extent of training necessary to perform EET adequately,[120-122] and a formal determination of what constitutes competency in EET by using a validated competency assessment tool is needed. It is clear that practitioners of EET need to (1) have expertise in careful inspection of BE by using HD-WLE and optical chromoendoscopy, (2) be able to recognize visible lesions within the BE segment, (3) be trained in the performance of EMR and ablative techniques, and (4) be equipped for the management of adverse events (perforations, bleeding, strictures, and recurrence). Future studies need to clarify the role of advanced imaging techniques in guiding EET. Further research is required to assess the impact of using standardized grading systems for BE (Prague criteria[58]) and Paris classification[67] for visible lesions on patient outcomes. The role of gastroesophageal reflux monitoring and pH control needs clarification. Finally, future prospective studies that use standardized definitions for study endpoints and focus on recurrence as the primary outcome are required. These studies will provide important data on the annual recurrence risk and predictors of recurrence, which can help generate evidence-based recommendations regarding surveillance endoscopic and biopsy protocols after CE-IM.

In conclusion, this physician-led initiative identified and formally validated quality indicators for EET in patients with BE-related neoplasia. The ultimate purpose of gathering data on these quality indicators was to identify performance gaps that allow for targeted improvement efforts in delivering quality endoscopy care to patients. These quality indicators may be assessed by individuals, practices, and payers. These quality indicators and benchmark targets also may be incorporated into the training curriculum of new endoscopists. Compliance with these quality indicators should improve the quality of management of patients with BE-related neoplasia.

DISCLOSURE

This study was supported by a grant from the University of Colorado, Department of Medicine Outstanding Early Scholars Program to S. Wani. No writing assistance was provided for this manuscript. No industry funding supported any part of this work. The following authors disclosed financial relationships relevant to this publication: Dr Abrams, consultant and research support from C2 Therapeutics, consultant for GIE Solutions; Dr Bergman, research support from NinePoint Medical, C2 Therapeutics, Erbe Medical, GIE Solutions, Medtronic, Cernostics, Olympus Endoscopy, Fujifilm Endoscopy, Pentax Endoscopy, Boston Scientific, Fractyl Medical, consultancy and paid lectures, Boston

Scientific, consultant for WATS3D; Dr Gerson, consultant for CDX Diagnostics; Dr Jankowski, educational grant from A2, speakers bureau, Takeda; Dr Komanduri, consultant for Medtronic, Boston Scientific, Cook Medical; Dr Lightdale, consultant for Boston Scientific, consultant, grant support from C2 Therapeutics, royalties from Cook Medical, consultant, grant support from NinePoint Medical; Dr Muthusamy, consultant, research support, Medtronic, consultant for Boston Scientific; Dr Pech, honoraria from Falk Foundation, Boston Scientific, Creo Medica; Dr Shaheen, grant funding from Medtronic, CSA Medical, C2 Therapeutics; Boston Scientific; Dr Spechler, consultant for Ironwood Pharmaceuticals; consultant for Interpace Diagnostics, consultant for Takeda Pharmaceuticals; Dr Wang, Research for C2 Therapeutics; Dr Wani, consultant for Medtronic, Boston Scientific, Dr Waxman, consultant for Cook Medical, Olympus, Medtronic. All other authors disclosed no financial relationships relevant to this publication.

Abbreviations: ACG, American College of Gastroenterology; ASGE, American Society for Gastrointestinal Endoscopy; BE, Barrett's esophagus; CE-D, complete eradication of dysplasia; CE-IM, complete eradication of intestinal metaplasia; EAC, esophageal adenocarcinoma; EET, endoscopic eradication therapy; HD-WLE, high-definition white light endoscopy; HGD, high-grade dysplasia; IPR, interpercentile range; IPRAS, interpercentile range adjusted for symmetry; LGD, low-grade dysplasia; RAM, RAND/University of California, Los Angeles Appropriateness Method; RFA, radiofrequency ablation.

REFERENCES

1. Pohl H, Welch HG. The role of overdiagnosis and reclassification in the marked increase of esophageal adenocarcinoma incidence. J Natl Cancer Inst 2005;97:142-6.
2. Lepage C, Rachet B, Jooste V, et al. Continuing rapid increase in esophageal adenocarcinoma in England and Wales. Am J Gastroenterol 2008;103:2694-9.
3. Hur C, Miller M, Kong CY, et al. Trends in esophageal adenocarcinoma incidence and mortality. Cancer 2013;119:1149-58.
4. Shaheen NJ, Falk GW, Iyer PG, et al. ACG clinical guideline: diagnosis and management of Barrett's esophagus. Am J Gastroenterol 2016;111:30-50; quiz 51.
5. Spechler SJ, Sharma P, Souza RF, et al. American Gastroenterological Association technical review on the management of Barrett's esophagus. Gastroenterology 2011;140:e18-52; quiz e13.
6. Wani S, Falk GW, Post J, et al. Risk factors for progression of low-grade dysplasia in patients with Barrett's esophagus. Gastroenterology 2011;141:1179-86;1186. e1.
7. Wani S. Population-based estimates of cancer and mortality in Barrett's esophagus: implications for the future. Clin Gastroenterol Hepatol 2011;9:723-4.
8. Rastogi A, Puli S, El-Serag HB, et al. Incidence of esophageal adenocarcinoma in patients with Barrett's esophagus and high-grade dysplasia: a meta-analysis. Gastrointest Endosc 2008;67:394-8.
9. Hvid-Jensen F, Pedersen L, Drewes AM, et al. Incidence of adenocarcinoma among patients with Barrett's esophagus. N Engl J Med 2011;365:1375-83.
10. Bhat S, Coleman HG, Yousef F, et al. Risk of malignant progression in Barrett's esophagus patients: results from a large population-based study. J Natl Cancer Inst 2011;103:1049-57.
11. Wani S, Sharma P. Challenges with endoscopic therapy for Barrett's esophagus. Gastroenterol Clin North Am 2015;44:355-72.
12. Wani S, Early D, Edmundowicz S, et al. Management of high-grade dysplasia and intramucosal adenocarcinoma in Barrett's esophagus. Clin Gastroenterol Hepatol 2012;10:704-11.
13. Shaheen NJ, Sharma P, Overholt BF, et al. Radiofrequency ablation in Barrett's esophagus with dysplasia. N Engl J Med 2009;360:2277-88.
14. 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.
15. Wolf WA, Pasricha S, Cotton C, et al. Incidence of esophageal adenocarcinoma and causes of mortality after radiofrequency ablation of Barrett's esophagus. Gastroenterology 2015;149:1752-61.e1.
16. Pech O, Behrens A, May A, et al. Long-term results and risk factor analysis for recurrence after curative endoscopic therapy in 349 patients with high-grade intraepithelial neoplasia and mucosal adenocarcinoma in Barrett's oesophagus. Gut 2008;57:1200-6.
17. Orman ES, Li N, Shaheen NJ. Efficacy and durability of radiofrequency ablation for Barrett's esophagus: systematic review and meta-analysis. Clin Gastroenterol Hepatol 2013;11:1245-55.
18. Phoa KN, Pouw RE, van Vilsteren FG, 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.
19. van Vilsteren FG, Pouw RE, Seewald S, et al. Stepwise radical endoscopic resection versus radiofrequency ablation for Barrett's oesophagus with high-grade dysplasia or early cancer: a multicentre randomised trial. Gut 2011;60:765-73.
20. Gupta M, Iyer PG, Lutzke L, et al. Recurrence of esophageal intestinal metaplasia after endoscopic mucosal resection and radiofrequency ablation of Barrett's esophagus: results from a US Multicenter Consortium. Gastroenterology 2013;145:79-86.e1.
21. Qumseya BJ, Wani S, Desai M, et al. Adverse events after radiofrequency ablation in patients with Barrett's esophagus—a systematic review and meta-analysis. Clin Gastroenterol Hepatol 2016.
22. Wani S, Drahos J, Cook MB, et al. Comparison of endoscopic therapies and surgical resection in patients with early esophageal cancer: a population-based study. Gastrointest Endosc 2014;79:224-32.e1.
23. American Gastroenterological Association; Spechler SJ, Sharma P, Souza RF, et al. American Gastroenterological Association medical position statement on the management of Barrett's esophagus. Gastroenterology 2011;140:1084-91.
24. 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.
25. Bennett C, Vakil N, Bergman J, et al. Consensus statements for management of Barrett's dysplasia and early-stage esophageal adenocarcinoma, based on a Delphi process. Gastroenterology 2012;143:336-46.
26. 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.
27. Cohen J, Pike IM. Defining and measuring quality in endoscopy. Am J Gastroenterol 2015;110:46-7.
28. Rizk MK, Sawhney MS, Cohen J, et al. Quality indicators common to all GI endoscopic procedures. Gastrointest Endosc 2015;81:3-16.
29. Park WG, Shaheen NJ, Cohen J, et al. Quality indicators for EGD. Gastrointest Endosc 2015;81:17-30.
30. Fitch K, Aguilar MD, Burnand B, et al. The RAND/UCLA appropriateness method user's manual. Santa Monica (Calif): RAND 2001.
31. Yadlapati R, Gawron AJ, Keswani RN, et al. Identification of quality measures for performance of and interpretation of data from esophageal manometry. Clin Gastroenterol Hepatol 2016;14:526-34.e1.

32. Yadlapati R, Gawron AJ, Bilimoria K, et al. Development of quality measures for the care of patients with gastroesophageal reflux disease. Clin Gastroenterol Hepatol 2015;13:874-83.e2.
33. Shekelle PG, Kahan JP, Bernstein SJ, et al. The reproducibility of a method to identify the overuse and underuse of medical procedures. N Engl J Med 1998;338:1888-95.
34. Halverson AL, Sellers MM, Bilimoria KY, et al. Identification of process measures to reduce postoperative readmission. J Gastrointest Surg 2014;18:1407-15.
35. Bilimoria KY, Bentrem DJ, Lillemoe KD, et al. Assessment of pancreatic cancer care in the United States based on formally developed quality indicators. J Natl Cancer Inst 2009;101:848-59.
36. Bilimoria KY, Raval MV, Bentrem DJ, et al. National assessment of melanoma care using formally developed quality indicators. J Clin Oncol 2009;27:5445-51.
37. Aguilar MD, Fitch K, Lazaro P, et al. The appropriateness of use of percutaneous transluminal coronary angioplasty in Spain. Int J Cardiol 2001;78:213-21; discussion 221-3.
38. Bernstein SJ, Lazaro P, Fitch K, et al. Effect of specialty and nationality on panel judgments of the appropriateness of coronary revascularization: a pilot study. Med Care 2001;39:513-20.
39. Lawson EH, Gibbons MM, Ko CY, et al. The appropriateness method has acceptable reliability and validity for assessing overuse and underuse of surgical procedures. J Clin Epidemiol 2012;65:1133-43.
40. Maggard MA, McGory ML, Shekelle PG, et al. Quality indicators in bariatric surgery: improving quality of care. Surg Obes Relat Dis 2006;2:423-9; discussion 429-30.
41. McGory ML, Shekelle PG, Ko CY. Development of quality indicators for patients undergoing colorectal cancer surgery. J Natl Cancer Inst 2006;98:1623-33.
42. Montgomery E, Goldblum JR, Greenson JK, et al. Dysplasia as a predictive marker for invasive carcinoma in Barrett esophagus: a follow-up study based on 138 cases from a diagnostic variability study. Hum Pathol 2001;32:379-88.
43. McKenna BJ, Appelman HD. Dysplasia of the gut: the diagnosis is harder than it seems. J Clin Gastroenterol 2002;34:111-6.
44. Curvers WL, ten Kate FJ, Krishnadath KK, et al. Low-grade dysplasia in Barrett's esophagus: overdiagnosed and underestimated. Am J Gastroenterol 2010;105:1523-30.
45. Duits LC, Phoa KN, Curvers WL, et al. Barrett's oesophagus patients with low-grade dysplasia can be accurately risk-stratified after histological review by an expert pathology panel. Gut 2015;64:700-6.
46. Sangle NA, Taylor SL, Emond MJ, et al. Overdiagnosis of high-grade dysplasia in Barrett's esophagus: a multicenter, international study. Mod Pathol 2015;28:758-65.
47. Montgomery E, Bronner MP, Goldblum JR, et al. Reproducibility of the diagnosis of dysplasia in Barrett esophagus: a reaffirmation. Hum Pathol 2001;32:368-78.
48. Reid BJ, Haggitt RC, Rubin CE, et al. Observer variation in the diagnosis of dysplasia in Barrett's esophagus. Hum Pathol 1988;19:166-78.
49. Ormsby AH, Petras RE, Henricks WH, et al. Observer variation in the diagnosis of superficial oesophageal adenocarcinoma. Gut 2002;51:671-6.
50. Kerkhof M, van Dekken H, Steyerberg EW, et al. Grading of dysplasia in Barrett's oesophagus: substantial interobserver variation between general and gastrointestinal pathologists. Histopathology 2007;50:920-7.
51. Downs-Kelly E, Mendelin JE, Bennett AE, et al. Poor interobserver agreement in the distinction of high-grade dysplasia and adenocarcinoma in pretreatment Barrett's esophagus biopsies. Am J Gastroenterol 2008;103:2333-40; quiz 2341.
52. Pech O, Vieth M, Schmitz D, et al. Conclusions from the histological diagnosis of low-grade intraepithelial neoplasia in Barrett's oesophagus. Scand J Gastroenterol 2007;42:682-8.
53. Alikhan M, Rex D, Khan A, et al. Variable pathologic interpretation of columnar lined esophagus by general pathologists in community practice. Gastrointest Endosc 1999;50:23-6.
54. Schlemper RJ, Riddell RH, Kato Y, et al. The Vienna classification of gastrointestinal epithelial neoplasia. Gut 2000;47:251-5.
55. Kaye PV, Haider SA, Ilyas M, et al. Barrett's dysplasia and the Vienna classification: reproducibility, prediction of progression and impact of consensus reporting and p53 immunohistochemistry. Histopathology 2009;54:699-712.
56. Wani S, Mathur SC, Curvers WL, et al. Greater interobserver agreement by endoscopic mucosal resection than biopsy samples in Barrett's dysplasia. Clin Gastroenterol Hepatol 2010;8:783-8.
57. Skacel M, Petras RE, Gramlich TL, et al. The diagnosis of low-grade dysplasia in Barrett's esophagus and its implications for disease progression. Am J Gastroenterol 2000;95:3383-7.
58. 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.
59. Lee YC, Cook MB, Bhatia S, et al. Interobserver reliability in the endoscopic diagnosis and grading of Barrett's esophagus: an Asian multinational study. Endoscopy 2010;42:699-704.
60. Chang CY, Lee YC, Lee CT, et al. The application of Prague C and M criteria in the diagnosis of Barrett's esophagus in an ethnic Chinese population. Am J Gastroenterol 2009;104:13-20.
61. Vahabzadeh B, Seetharam AB, Cook MB, et al. Validation of the Prague C & M criteria for the endoscopic grading of Barrett's esophagus among gastroenterology trainees: a multicenter study [abstract]. Gastrointest Endosc 2010;71:AB156.
62. 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.
63. Konda VJ, Repici A, Gupta N, et al. The Prague criteria predict response to successful endoscopic eradication therapy for Barrett's esophagus with dysplasia or early cancer: results from an international, multi-center consortium. Gastroenterology 2015;148:S214-5.
64. Epstein J, Canto MI, Ji Shin E, et al. The Prague classification underestimates the true maximal extent and dysplasia grade of Barrett's esophagus. Gastroenterology 2013;144:S693.
65. Hundal R, Blakely P, Wong CK. Enhanced characterization of Barrett's esophagus islands through a revision of the Prague criteria. Gastroenterology 2015;148:S349.
66. Wani S, Rubenstein JH, Vieth M, et al. Diagnosis and management of low-grade dysplasia in Barrett's esophagus: clinical practice updates expert review from the Clinical Guidelines Committee of the American Gastroenterological Association. Gastroenterology 2016;151:822-35.
67. Paris Workshop on Columnar Metaplasia in the Esophagus and the Esophagogastric Junction, Paris, France, December 11-12 2004. Endoscopy 2005;37:879-920.
68. Pech O, Gossner L, Manner H, et al. Prospective evaluation of the macroscopic types and location of early Barrett's neoplasia in 380 lesions. Endoscopy 2007;39:588-93.
69. Peters FP, Brakenhoff KPM, Curvers WL, et al. Histologic evaluation of resection specimens obtained at 293 endoscopic resections in Barrett's esophagus. Gastrointest Endosc 2008;67:604-9.
70. 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.
71. Kara MA, Peters FP, Rosmolen WD, et al. High-resolution endoscopy plus chromoendoscopy or narrow-band imaging in Barrett's esophagus: a prospective randomized crossover study. Endoscopy 2005;37:929-36.
72. Curvers W, Baak L, Kiesslich R, et al. Chromoendoscopy and narrow-band imaging compared with high-resolution magnification endoscopy in Barrett's esophagus. Gastroenterology 2008;134:670-9.
73. Sami SS, Subramanian V, Butt WM, et al. High definition versus standard definition white light endoscopy for detecting

dysplasia in patients with Barrett's esophagus. Dis Esophagus. Epub 2014 Sep 10.
74. Larghi A, Lightdale CJ, Memeo L, et al. EUS followed by EMR for staging of high-grade dysplasia and early cancer in Barrett's esophagus. Gastrointest Endosc 2005;62:16-23.
75. Peters FP, Brakenhoff KP, Curvers WL, et al. Histologic evaluation of resection specimens obtained at 293 endoscopic resections in Barrett's esophagus. Gastrointest Endosc 2008;67:604-9.
76. Moss A, Bourke MJ, Hourigan LF, et al. Endoscopic resection for Barrett's high-grade dysplasia and early esophageal adenocarcinoma: an essential staging procedure with long-term therapeutic benefit. Am J Gastroenterol 2010;105:1276-83.
77. Chennat J, Konda VJ, Ross AS, et al. Complete Barrett's eradication endoscopic mucosal resection: an effective treatment modality for high-grade dysplasia and intramucosal carcinoma—an American single-center experience. Am J Gastroenterol 2009;104:2684-92.
78. Wani S, Abrams J, Edmundowicz SA, et al. Endoscopic mucosal resection results in change of histologic diagnosis in Barrett's esophagus patients with visible and flat neoplasia: a multicenter cohort study. Dig Dis Sci 2013;58:1703-9.
79. Mino-Kenudson M, Hull MJ, Brown I, et al. EMR for Barrett's esophagus–related superficial neoplasms offers better diagnostic reproducibility than mucosal biopsy. Gastrointest Endosc 2007;66:660-6; quiz 767, 769.
80. ASGE Standards of Practice Committee; Evans JA, Early DS, Fukami N, et al. The role of endoscopy in Barrett's esophagus and other premalignant conditions of the esophagus. Gastrointest Endosc 2012;76:1087-94.
81. Pasricha S, Bulsiewicz WJ, Infantolino A, et al. National practice patterns in the timing of radiofrequency ablation (RFA) for Barrett's esophagus: results from the U.S. RFA registry. Gastroenterology 2013;144:S580.
82. Phoa KN, Van Vilsteren FGI, Weusten BLAM, 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.
83. Shaheen NJ, Overholt BF, Sampliner RE, et al. Durability of radiofrequency ablation in Barrett's esophagus with dysplasia. Gastroenterology 2011;141:460-8.
84. Wolf WA, Overholt BF, Li N, et al. Durability of radiofrequency ablation (RFA) in Barrett's esophagus with dysplasia: the AIM dysplasia trial at five years. Gastroenterology 2014;146: S-131.
85. Phoa KN, Pouw RE, Bisschops R, et al. Multimodality endoscopic eradication for neoplastic Barrett oesophagus: results of an European multicentre study (EURO-II). Gut 2015;65:555.
86. 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.
87. 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.
88. 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.e1.
89. Orman ES, Li N, Shaheen NJ. Efficacy and durability of radiofrequency ablation for Barrett's Esophagus: systematic review and meta-analysis. Clin Gastroenterol Hepatol 2013;11:1245-55.
90. Gondrie JJ, Pouw RE, Sondermeijer CM, et al. Effective treatment of early Barrett's neoplasia with stepwise circumferential and focal ablation using the HALO system. Endoscopy 2008;40:370-9.
91. Pouw RE, Wirths K, Eisendrath P, et al. Efficacy of radiofrequency ablation combined with endoscopic resection for Barrett's esophagus with early neoplasia. Clin Gastroenterol Hepatol 2010;8:23-9.
92. Fleischer DE, Overholt BF, Sharma VK, et al. Endoscopic radiofrequency ablation for Barrett's esophagus: 5-year outcomes from a prospective multicenter trial. Endoscopy 2010;42:781-9.
93. Agoston AT, Dulai PS, Rothstein RI, et al. Predictors of durable treatment response after radiofrequency ablation for Barrett's esophagus associated intramucosal adenocarcinoma. Lab Invest 2014;94:162A.
94. Chandra S, Gorospe EC, Leggett CL, et al. Durability of radiofrequency ablation for Barrett's esophagus: a single center 10-year experience. Gastroenterology 2013;144:S685.
95. Haidry RJ, Banks MR, Butt MA, et al. Long term follow up after successful radiofrequency ablation for Barrett's related neoplasia is essential to diagnose recurrent disease: data from the United Kingdom patient registry [abstract]. Gastrointest Endosc 2014;79:AB393-4.
96. Haidry RJ, Lipman G, Butt MA, et al. Six year disease durability outcomes on patients treated with endoscopic therapy for Barrett's related neoplasia from the UK registry. Gastroenterology 2015;148:S16.
97. Vaccaro BJ, Gonzalez S, Poneros JM, et al. Detection of intestinal metaplasia after successful eradication of Barrett's Esophagus with radiofrequency ablation. Dig Dis Sci 2011;56:1996-2000.
98. Haidry RJ, Banks M, Gupta A, et al. Recurrence after successful radiofrequency ablation for Barrett's related neoplasia is more likely in males: data from the United Kingdom patient registry. Gut 2014;63:A113-4.
99. Cotton CC, Wolf WA, Pasricha S, et al. Recurrent intestinal metaplasia after radiofrequency ablation for Barrett's esophagus: endoscopic findings and anatomic location. Gastrointest Endosc 2015;81:1362-9.
100. Orman ES, Kim HP, Bulsiewicz WJ, et al. Intestinal metaplasia recurs infrequently in patients successfully treated for Barrett's esophagus with radiofrequency ablation. Am J Gastroenterol 2013;108:187-95; quiz 196.
101. Prasad GA, Dunagan KT, Tian J, et al. Recurrence of intestinal metaplasia following radiofrequency ablation: rates and predictors [abstract]. Gastrointest Endosc 2011;73:AB145-6.
102. Phoa KN, Pouw RE, Bisschops R, et al. Multimodality endoscopic eradication for neoplastic Barrett oesophagus: results of an European multicentre study (EURO-II). Gut. Epub 2015 Mar 2.
103. Krishnamoorthi R, Ragunathan K, Crews NR, et al. Risk of recurrence of Barrett's esophagus after successful endoscopic therapy: a systematic review and meta-analysis [abstract]. Gastrointest Endosc 2015;81:AB505-6.
104. Adler DG, Lieb JG, Cohen J, et al. Corrigendum: quality indicators for ERCP. Am J Gastroenterol 2015;110:608.
105. Anand O, Wani S, Sharma P. Gastroesophageal reflux disease and Barrett's esophagus. Endoscopy 2008;40:126-30.
106. Bennett C, Moayyedi P, Corley DA, et al. BOB CAT: a large-scale review and Delphi consensus for management of Barrett's esophagus with no dysplasia, indefinite for, or low-grade dysplasia. Am J Gastroenterol 2015;110:662-82; quiz 683.
107. Vaezi MF, Richter JE. Role of acid and duodenogastroesophageal reflux in gastroesophageal reflux disease. Gastroenterology 1996;111:1192-9.
108. Gharahkhani P, Tung J, Hinds D, et al. Chronic gastroesophageal reflux disease shares genetic background with esophageal adenocarcinoma and Barrett's esophagus. Hum Mol Genet 2016;25:828-35.
109. Cook MB, Corley DA, Murray LJ, et al. Gastroesophageal reflux in relation to adenocarcinomas of the esophagus: a pooled analysis from the Barrett's and Esophageal Adenocarcinoma Consortium (BEACON). PLoS One 2014;9:e103508.
110. Krishnan K, Pandolfino JE, Kahrilas PJ, et al. Increased risk for persistent intestinal metaplasia in patients with Barrett's esophagus and

uncontrolled reflux exposure before radiofrequency ablation. Gastroenterology 2012;143:576-81.
111. Yasuda K, Choi SE, Nishioka NS, et al. Incidence and predictors of adenocarcinoma following endoscopic ablation of Barrett's esophagus. Dig Dis Sci 2014;59:1560-6.
112. Akiyama J, Marcus SN, Triadafilopoulos G. Effective intra-esophageal acid control is associated with improved radiofrequency ablation outcomes in Barrett's esophagus. Dig Dis Sci 2012;57:2625-32.
113. Wani S, Wallace MB, Cohen J, et al. Quality indicators for EUS. Gastrointest Endosc 2015;81:67-80.
114. Petersen BT. Quality assurance for endoscopists. Best Pract Res Clin Gastroenterol 2011;25:349-60.
115. Rubenstein JH, Lieberman D, Fennerty B, et al. Measuring the quality of Barrett's esophagus management with measures that are high quality. Gastroenterology 2015;149:1298-301.
116. Burwell SM. Setting value-based payment goals—HHS efforts to improve U.S. health care. N Engl J Med 2015;372:897-9.
117. National Quality Forum. Available at: www.qualityforum.org/docs/measure_evaluation_criteria.aspx. Accessed May 15, 2015.
118. McGlynn EA, Schneider EC, Kerr EA. Reimagining quality measurement. N Engl J Med 2014;371:2150-3.
119. Cassel CK, Conway PH, Delbanco SF, et al. Getting more performance from performance measurement. N Engl J Med 2014;371:2145-7.
120. Pasricha S, Cotton C, Hathorn KE, et al. Effects of the learning curve on efficacy of radiofrequency ablation for Barrett's esophagus. Gastroenterology 2015;149:890-6.e2.
121. van Vilsteren FG, Pouw RE, Herrero LA, et al. Learning to perform endoscopic resection of esophageal neoplasia is associated with significant complications even within a structured training program. Endoscopy 2012;44:4-12.
122. Fudman DI, Lightdale CJ, Poneros JM, et al. Positive correlation between endoscopist radiofrequency ablation volume and response rates in Barrett's esophagus. Gastrointest Endosc 2014;80:71-7.

Received March 6, 2017. Accepted March 6, 2017.

Current affiliations: University of Colorado Anschutz Medical Center, Aurora, Colorado (1), University of California in Los Angeles, Los Angeles, California (2), University of North Carolina, Chapel Hill, North Carolina (3), Northwestern University, Chicago, Illinois (4), Columbia University Medical Center, New York, New York (5), Academic Medical Center, Amsterdam, The Netherlands (6), University Hospitals, Cleveland, Ohio (7), University of California in Irvine, Irvine, California (8), Arizona Center for Digestive Health, Gilbert, Arizona (9), The Oregon Clinic, Portland, Oregon (10), University of Pennsylvania Perelman School of Medicine, Philadelphia, Pennsylvania (11), Oregon Health and Sciences University, Portland, Oregon (12), California Pacific Medical Center, San Francisco, California (13), University of Washington, Seattle, Washington (14), UCLan Medical School, England, United Kingdom (15), Brigham and Women's Hospital, Boston, Massachusetts (16), St. John of God Hospital, Regensburg, Germany (17), University of Arizona, Tucson, Arizona (18), Dallas Veterans Affairs Medical Center, Dallas, Texas (19), Stanford University, Palo Alto, California (20), Mayo Clinic, Jacksonville, Florida (21), Mayo Clinic, Rochester, Minnesota (22), University of Chicago, Chicago, Illinois, USA (23).

Presented at Digestive Disease Week, May 21-24, 2016, San Diego, CA (Gastrointest Endosc 2016;83:AB129).

Reprint requests: Sachin Wani, MD, Associate Professor of Medicine, Division of Gastroenterology and Hepatology, University of Colorado Anschutz Medical Center, Mail Stop F735, 1635 Aurora Court, Rm 2.031, Aurora, CO 80045. Fax: 720-848-2749. E-mail: sachinwani10@yahoo.com.

APPENDIX 2. RANKING INSTRUCTIONS PROVIDED TO ALL PANEL MEMBERS

Dear Colleagues,

Thank you for participating in the Quality Measures in Endoscopic Eradication Therapies In Barrett's Esophagus Consensus Document—The TREAT-BE (Treatment With Resection And Endoscopic Ablation Techniques For Barrett's Esophagus) Consortium development process.

The aim of this initiative is to systematically evaluate and enumerate quality measures for endoscopic eradication therapies (EETs) in patients with Barrett's esophagus (BE) by using validated and formal methodology. The ultimate objective is to improve quality of care delivered to patients with BE.

The following link will take you to an online list of quality measures. These are measures that have been shortlisted by the before-, during-, and post-procedure EET quality metrics subcommittees. You will be prompted to rate the proposed quality measures according to your perceived appropriateness and necessity in clinical practice. Please use the following instructions when ranking the proposed quality measures:

1. The purpose of these measures is to assist practitioners with quality improvement. All measures are intended to be calculated and reported at the practice level and need not have a direct benefit to an individual patient. Practices will be able to compare themselves with one another in hopes that practices will feed back their own data to institute quality improvement initiatives when appropriate.
2. The measures do not necessarily have to apply to any one specific patient, but rather, they may pertain to the overall care of patients with BE.
3. A measure is considered valid if compliance with this measure is critical to providing quality care to patients with BE, exclusive of costs or feasibility. Do not consider cost implications or the feasibility of implementing the measure in your rankings.
4. Base your rankings on your own personal judgment and not what you believe other experts or the panel might say.
5. Consider these measures for the average patient presenting to the average physician at an average hospital.
6. For suggested quality measures that you feel are appropriate, please suggest a threshold percentage as a benchmark.
7. Please complete the online questionnaire by August 15.
8. We will contact you to revise your rankings if the rankings do not follow the instructions above. If you feel a measure is unreasonable, not useful, or dangerous, please rank it a 1 instead of leaving it blank.
9. Once all of your responses are received, we will analyze the rankings and present the blinded, aggregate results at the expert panel meeting on Friday, November 13.

Thank you, and please feel free to contact us with any questions.

APPENDIX 3. SEARCH STRATEGY FOR SYSTEMATIC REVIEW

The asterisk * is a truncation symbol that returns any variation that follows the root word. For example, *injur** will return *injury, injuries, injured, etc*. Adjacency is indicated by 'adjN' where 'N' is the amount of word separation allowed. In Ovid Medline 'adj3' allows up to a 2-word separation (N-1 word separation). 'tw' is the Ovid text word tag and searches the title and abstract field, whereas 'kf' searches the author-supplied keyword field. Finally, '/' indicates a medical subject heading (MeSH) with 'exp' indicating that all narrower vocabulary terms were searched as well.

Search performed on August 12, 2015

Database(s): Ovid MEDLINE(R) In-Process and Other Non-Indexed Citations, Ovid MEDLINE(R) Daily and Ovid MEDLINE(R) 1946 to Present

SEARCH STRATEGY: Ovid Medline

#	Searches	Annotations
1	barrett*.tw,kf. or exp barrett esophagus/	Barrett's esophagus
2	limit 1 to (english language and yr="1990 -Current")	Limits applied
3	(((catheter or electric* or radiofrequenc* or radio frequenc* or RF or surgical or technique* or thermal or RFA or laser*) adj2 ablation*) or electrocautery).tw,kf. or exp catheter ablation/	radiofrequency ablation
4	(cryotherap* or ((cold or cryogenic or hypothermal or cryoballon) adj2 (therap* or surg* or ablation*)) or cryotherm* or cryotreatment* or cryosurg* or cryo-surg* or cryoablation*).tw,kf. or exp cryotherapy/ or exp cryosurgery/	cryotherapy
5	(endoscop* or oesophagoscop* or esophagoscop* or oesophagogastroduodenoscop* or esophagogastroduodenoscop*).tw,kf. or exp endoscopy/ or esophagoscopy/ or exp endoscopes/	endoscopy
6	((laser adj2 (microscop* or cytometr* or endomicroscop*)) or cslm).tw,kf.	confocal laser endomicroscopy
7	(high resolution adj3 (endoscop* or oesophagoscop* or esophagoscop* or oesophagogastroduodenoscop* or esophagogastroduodenoscop*)).tw,kf.	high resolution endoscopy
8	(((endoscop* or oesophagoscop* or esophagoscop* or oesophagogastroduodenoscop* or esophagogastroduodenoscop*) adj3 (muc* adj2 resection*)) or (endoscopic adj2 mucosectom*)).tw,kf.	endoscopic mucosal resection
9	((endoscop* or oesophagoscop* or Esophagoscop* or oesophagogastroduodenoscop* or esophagogastroduodenoscop* or angioscop* or arthroscop* or bronchoscop* or colposcop* or culdoscop* or cystoscop* or colonoscop* or sigmoidoscop* or enteroscop* or duodenoscop* or gastroscop* or proctoscop* or fetoscop* or hysteroscop* or laparoscop* or laryngoscop* or mediastinoscop* or neuroendoscop* or thoracoscop* or ureteroscop*) adj3 submuc* adj3 dissection*).tw,kf.	endoscopic submucosal dilation
10	((Endosonograph* or (endoscop* or oesophagoscop* or Esophagoscop* or oesophagogastroduodenoscop* or esophagogastroduodenoscop* or angioscop* or arthroscop* or bronchoscop* or colposcop* or culdoscop* or cystoscop* or colonoscop* or sigmoidoscop* or enteroscop* or duodenoscop* or gastroscop* or proctoscop* or fetoscop* or hysteroscop* or laparoscop* or laryngoscop* or mediastinoscop* or neuroendoscop* or thoracoscop* or ureteroscop*)) adj2 (ultrasound* or ultrasongraph* or echo* or ultrasonic)).tw,kf. or Endosonography/	endoscopic ultrasound/ultrasonography
11	((endoscop* or oesophagoscop* or Esophagoscop* or oesophagogastroduodenoscop* or esophagogastroduodenoscop* or angioscop* or arthroscop* or bronchoscop* or colposcop* or culdoscop* or cystoscop* or colonoscop* or sigmoidoscop* or enteroscop* or duodenoscop* or gastroscop* or proctoscop* or fetoscop* or hysteroscop* or laparoscop* or laryngoscop* or mediastinoscop* or neuroendoscop* or thoracoscop* or ureteroscop*) adj2 eradicat* adj2 (therap* or treatment*)).tw,kf.	endoscopic eradication therapies
12	((clinical or patient-relevant or rehabilitation or treatment or short term or long term or recovery) adj3 (effectiveness* or outcome* or efficac* or effectiveness)).tw,kf. or Treatment Outcome/	treatment outcomes
13	(Recurren* or Recrudescence* or Relapse*).tw,kf. or Recurrence/	recurrence
14	(Durab* or perdur* or long lasting).tw,kf.	durability
15	((Complete adj2 eradicat*) or ((disease or progression or event or barrett*) adj2 free)).tw,kf. or Disease-Free Survival/	complete eradication
16	((adverse or undesirable or injurious) adj2 effects).tw,kf. or ae.fs.	adverse effects
17	(progression* or course* or development or evolution or exacerbation*).tw,kf. or exp disease progression/	progression
18	(strictur* or constriction* or stenos* or resteno*).tw,kf. or "Esophageal Stenosis"/ or "Constriction, Pathologic"/	stricture
19	(perforation* or puncture* or hole* or tear* or fissure or rupture* or rip or rips or ripping or ripped or dissection* or clip*).tw,kf. or exp Esophageal Perforation/ or Rupture/ or Rupture, Spontaneous/	perforation

(*continued on the next page*)

SEARCH STRATEGY. Continued

#	Searches	Annotations
20	(bleed* or h?emorrhag* or (blood adj2 loss*) or h?ematoma* or h?ematocel* h?ematur* or extrava* or purpura* or ecchymo* or epistax* or exsanguination* or h?ematemesis or melena* or h?emarthros* or h?emobilia* or h?emoperitoneum* or hemoptys* or h?emothorax* or h?emopneumothorax* or aneurysm* or aneurism*).tw,kf. or exp "hemorrhage"/ or exp "Aneurysm, Ruptured"/ or "Cerebral Hemorrhage, Traumatic"/	bleeding
21	(surviva* or morbidit* or mortalit* or ((case* or outcome*) adj2 fatal*) or death rate* or incidenc* or prevalence* or cause of death).tw,kf. or exp Morbidity/ or exp Mortality/ or exp Survival Analysis/ or Survival Rate/	survival/morbidity/mortality
22	(narrow band imaging* or narrowband imaging*).tw,kf. or Narrow Band Imaging/	narrow band imaging
23	Low grade dysplasia*.tw,kf.	low-grade dysplasia
24	High grade dysplasia*.tw,kf.	high-grade dysplasia
25	Biops*.tw,kf. or exp Biopsy/	biopsy/biopsies
26	surveillance.tw,kf. or exp health surveys/	surveillance
27	((oesophag* or esophag*) adj2 (adenocarcinoma* or malignant adenoma* or granular cell carcinoma* or tubular carcinoma* or cribriform carcinoma*)).tw,kf. or ((exp esophagus/ or barrett esophagus/) and exp adenocarcinoma/)	esophageal adenocarcinoma
28	((observer or interobserver or inter-observer or intraobserver or intra-observer) adj2 agreement*).tw,kf.	interobserver/intraobserver agreement
29	((observer or interobserver or inter-observer or intraobserver or intra-observer) adj2 (variat* or variab* or bias*)).tw,kf. or Observer Variation/	interobserver variability
30	(Pathol* or histopath* or telepathol*).tw,kf. or exp Pathology/ or pa.fs.	pathologist/pathology
31	(Paris adj2 (critera* or classification*)).tw,kf.	Paris Classification
32	(Prague adj2 (criteria* or classification*)).tw,kf.	Prague Criteria/Classification
33	3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27 or 28 or 29 or 30 or 31 or 32	All dimensions or'd
34	2 and 33	BE + all dimensions