ASMBS position statement on the rationale for performance of upper gastrointestinal endoscopy before and after metabolic and bariatric surgery

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Abstract

The following position statement is issued by the American Society for Metabolic and Bariatric Surgery in response to inquiries made to the Society by patients, physicians, society members, hospitals, health insurance payors, the media, and others regarding the need and possible strategies for screening endoscopic examination before metabolic and bariatric surgery (MBS), as well as the rationale, indications, and strategies for postoperative surveillance for mucosal abnormalities, including gastroesophageal reflux disease and associated esophageal mucosal injuries (erosive esophagitis and Barrett’s esophagus) that may develop in the long term after MBS, specifically for patients undergoing sleeve gastrectomy or Roux-en-Y gastric bypass. The general principles described here may also apply to procedures such as biliopancreatic diversion (BPD) and BPD with duodenal switch (DS); however, the paucity of procedure-specific literature for BPD and DS limits the value of this statement to those procedures. In addition, children with obesity undergoing MBS may have unique considerations and are not specifically addressed in this position statement. This recommendation is based on current clinical knowledge, expert opinion, and published peer-reviewed scientific evidence available at this time. The statement is not intended to be and should not be construed as stating or establishing a local, regional, or national standard of care. The statement will be revised in the future as additional evidence becomes available. (Surg Obes Relat Dis 2021;17:837–847.) © 2021 American Society for Bariatric Surgery. Published by Elsevier Inc. All rights reserved.

Keywords: GERD; Barrett’s esophagus; Bariatric surgery; Sleeve gastrectomy; Gastric bypass; Obesity; Endoscopy; Screening
The 2 most common bariatric surgical procedures currently performed in the United States are sleeve gastrectomy (SG) and Roux-en-Y gastric bypass (RYGB) [1,2]. Both procedures have been shown to have an excellent safety profile, particularly when performed in accredited centers. Moreover, both procedures provide meaningful and relatively similar weight loss and remission of obesity-associated comorbidities, at levels that are far superior to those of nonsurgical therapy [3–7]. Both procedures are also associated with significant reductions in premature deaths from cardiovascular diseases, deaths related to type 2 diabetes (T2D), and deaths associated with multiple cancers [3–6,8].

However there is evidence that SG is associated with worsening gastroesophageal reflux disease (GERD) in some patients with preexisting GERD, as well as a higher rate of de novo GERD, when compared to RYGB [9–16]. In some published studies, 25% to 40% of patients who undergo SG may develop de novo GERD, with about 30% having endoscopic evidence of erosive esophagitis [17–23]. Although prospective and better-designed studies are required for confirmation, it has been observed that some SG patients may develop findings that could represent de novo Barrett’s esophagus 3 or more years after SG, a phenomenon which has not been reported after RYGB [24,25].

In this statement, we will review current definitions of GERD and Barrett’s esophagus and summarize the peer-reviewed scientific literature for the frequency of GERD, Barrett’s esophagus, and other upper gastrointestinal conditions, as diagnosed using upper esophagogastroduodenoscopy (EGD) before and after metabolic and bariatric surgery (MBS).

The statement has been divided into the following 4 sections:

1. A review of the literature on the value of preoperative EGD in patients seeking bariatric surgery;
2. A review of the definitions and standards for evaluation, screening, and surveillance of GERD and Barrett’s esophagus in the general population;
3. A review of the literature on GERD, erosive esophagitis, and Barrett’s esophagus before and after SG and RYGB, with an emphasis on objective testing; and
4. Recommendations for upper gastrointestinal endoscopy before and after MBS.

Methods

For Sections 1 and 3, we performed an electronic Ovid Medline literature search for articles on the role of upper gastrointestinal endoscopy before and after MBS, with a focus on GERD and Barrett’s esophagus, that were published between January 1975 and January 2021. Key terms searched for were “endoscopy,” “bariatric surgery,” “weight loss surgery,” “sleeve,” “bypass,” “preoperative,” “postoperative,” “Gastroesophageal reflux,” “esophagitis,” “sleeve gastrectomy,” “gastric bypass,” “Barret’s esophagus,” and/or “Intestinal Metaplasia.” We excluded all articles that published abstracts only or were case reports, letters and comments, and animal or in vitro studies. Articles were also excluded if there was no description of endoscopic findings, there were fewer than 10 patients, the follow-up was less than 6 months, or they were published in a language other than English. After this initial screening, a full-text copy of each article was obtained for review. References within the selected articles were checked manually to supplement the electronic search for additional relevant articles. Selected studies could be of any design. When different articles reporting on overlapping populations were identified, the most recent article with the largest study population was selected for review. Each selected article was searched to extract data related to the research design used, population studied, treatment described, and outcome measures. Only articles in which the actual number of patients could be extracted were included.

The data collected were recorded in a database (Microsoft Excel, Microsoft Corporation). Information extracted from eligible studies included study data (year of publication, country or countries of origin, and design) and all available patient demographic and biometric data. Outcomes measured were any EGD findings; the presence of, details of, and definitions for GERD and Barrett’s esophagus before and after bariatric surgery; bariatric surgical technique; and follow-up period. For all outcome measures, we preferentially recorded the number of patients evaluated as the denominator wherever possible. The overall rate of EGD findings before bariatric surgery and rates of GERD, esophagitis, and Barrett’s esophagus before and after MBS were estimated using weighted averages of the sample prevalence in each study, with weights equal to the number of patients. There were no statistical analyses, as there were no cohorts without an intervention for comparison.

For Section 2, we reviewed the current standards and society guidelines for EGD indications in the general population and the currently accepted definitions of GERD and Barrett’s esophagus.

Section 1: review of the literature on the value of preoperative EGD in patients seeking bariatric surgery

There were 28 studies identified that evaluated the role of endoscopy prior to bariatric procedures in both symptomatic and asymptomatic patients (Table 1). A total of 12,385 patients are represented, with a mean age of 43.9 years, 68.5% female, and a mean body mass index (BMI) of 45.9 kg/m² (range 40.6–50.1 kg/m²). The average proportions of patients with GERD, hiatal hernia, erosive
esophagitis, and Barrett’s esophagus before MBS were 27.3%, 21%, 16.4%, and 2.7%, respectively. It is important to note that definitions for all conditions varied across studies. Also, continuous pH monitoring tests were positive in 52.5% of all patients (with and without symptoms), and patients had at least 1 of numerous other findings on endoscopy, including esophageal papilloma and polyps, epiphrenic diverticula, Schatzki ring, esophageal and gastric adenocarcinoma, gastritis, gastric submucosal lipoma, celiac disease, gastric polyps and ulcers, *Helicobacter pylori* infection, gastrointestinal stromal tumors, gastric carcinoid, duodenal ulcer, erosions and polyps, and gastric and duodenal neuroendocrine tumor. The rate at which malignancy was found in preoperative endoscopy in these studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Patients, n</th>
<th>Study design</th>
<th>Mean age, years</th>
<th>Female, %</th>
<th>Mean BMI, kg/m²</th>
<th>GERD, %</th>
<th>Hiatal hernia, %</th>
<th>Esophagitis, %</th>
<th>Barrett’s, %</th>
<th>Positive pH test, %</th>
<th>Other findings, %</th>
</tr>
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<td>Azagury, 2006 [29]</td>
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<td>R</td>
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<td>8.1</td>
<td>0</td>
<td>3.7</td>
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<td>P</td>
<td>37</td>
<td>69.3</td>
<td>45.1</td>
<td>54.7</td>
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<td>69</td>
<td>P</td>
<td>43.4</td>
<td>62.3</td>
<td>47.6</td>
<td>17.4</td>
<td>29.0</td>
<td>11.6</td>
<td>1.4</td>
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<td>P</td>
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<td>41</td>
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<td>77.9</td>
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<td>R</td>
<td>44.3</td>
<td>76.5</td>
<td>46.5</td>
<td>-</td>
<td>21.8</td>
<td>16.6</td>
<td>1.3</td>
<td>-</td>
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<td>Gomez, 2014 [31]</td>
<td>232</td>
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<td>25.4</td>
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<td>47.4</td>
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<td>10.3</td>
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<td>34.9</td>
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<td>48</td>
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<td>10.8</td>
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<td>-</td>
<td>-</td>
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<td>R</td>
<td>43.8</td>
<td>64.7</td>
<td>50.1</td>
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<td>-</td>
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<td>Genco, 2017 [19]</td>
<td>110</td>
<td>R</td>
<td>-</td>
<td>-</td>
<td>45.8</td>
<td>33.6</td>
<td>14.5</td>
<td>24.5</td>
<td>0</td>
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<td>Mandeville, 2017 [33]</td>
<td>100</td>
<td>R</td>
<td>41.4</td>
<td>61</td>
<td>40.6</td>
<td>17</td>
<td>36.1</td>
<td>53</td>
<td>0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Heimgartner, 2017 [43]</td>
<td>100</td>
<td>R</td>
<td>40</td>
<td>68</td>
<td>44.9</td>
<td>54</td>
<td>11.0</td>
<td>36</td>
<td>6</td>
<td>52</td>
<td>-</td>
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<tr>
<td>Schlottmann, 2018 [44]</td>
<td>193</td>
<td>R</td>
<td>46</td>
<td>63.7</td>
<td>44.5</td>
<td>-</td>
<td>23.8</td>
<td>16.6</td>
<td>1.6</td>
<td>-</td>
<td>-</td>
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<td>Schneider, 2018 [45]</td>
<td>1190</td>
<td>R</td>
<td>42.2</td>
<td>71.3</td>
<td>44.4</td>
<td>-</td>
<td>4.6</td>
<td>19.2</td>
<td>.3</td>
<td>-</td>
<td>32.5</td>
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<tr>
<td>Saarinen, 2018 [32]</td>
<td>1275</td>
<td>R</td>
<td>48.5</td>
<td>72.6</td>
<td>46.1</td>
<td>17.4</td>
<td>15.5</td>
<td>6.9</td>
<td>3.7</td>
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<td>1.6</td>
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<td>D’Silva, 2018 [46]</td>
<td>675</td>
<td>R</td>
<td>45</td>
<td>56.7</td>
<td>43.9</td>
<td>-</td>
<td>52.4</td>
<td>16.9</td>
<td>1.8</td>
<td>-</td>
<td>22.5</td>
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<tr>
<td>Abou Hussein, 2018 [35]</td>
<td>1278</td>
<td>R</td>
<td>41.3</td>
<td>61</td>
<td>43.7</td>
<td>-</td>
<td>23.8</td>
<td>20.3</td>
<td>3</td>
<td>-</td>
<td>42.7</td>
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<tr>
<td>Mazzini et al., 2019 [47]</td>
<td>93</td>
<td>P</td>
<td>37</td>
<td>80.6</td>
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<td>53.8</td>
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<td>R</td>
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<td>17.4</td>
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<td>-</td>
<td>51</td>
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<tr>
<td>Chang, 2020 [49]</td>
<td>631</td>
<td>R</td>
<td>44</td>
<td>72.4</td>
<td>46</td>
<td>-</td>
<td>27.1</td>
<td>26.5</td>
<td>4.6</td>
<td>-</td>
<td>4.9</td>
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<tr>
<td>Moulla, 2020 [50]</td>
<td>636</td>
<td>R</td>
<td>47.8</td>
<td>66.4</td>
<td>50.2</td>
<td>-</td>
<td>32.5</td>
<td>17.8</td>
<td>11.8</td>
<td>-</td>
<td>17.9</td>
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<tr>
<td>Ozeki, 2020 [34]</td>
<td>260</td>
<td>R</td>
<td>54</td>
<td>25</td>
<td>44.9</td>
<td>49.2</td>
<td>25.8</td>
<td>20.8</td>
<td>7.3</td>
<td>-</td>
<td>65</td>
</tr>
<tr>
<td>Makiewicz, 2020 [51]</td>
<td>1000</td>
<td>R</td>
<td>47.2</td>
<td>78.9</td>
<td>48.6</td>
<td>-</td>
<td>23.1</td>
<td>9.5</td>
<td>3.1</td>
<td>-</td>
<td>95.2</td>
</tr>
<tr>
<td>Bhambr, 2020 [52]</td>
<td>211</td>
<td>P</td>
<td>-</td>
<td>73.9</td>
<td>46.2</td>
<td>38.9</td>
<td>24.6</td>
<td>36.9</td>
<td>0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>12,385</td>
<td>Weighted averages</td>
<td>43.9 (34.6–51)</td>
<td>68.5% (25.4–82)</td>
<td>45.9 (40.6–50.1)</td>
<td>27.3% (8.1–64)</td>
<td>21% (4.6–52.4)</td>
<td>16.4% (5–52.7)</td>
<td>2.7% (0–11.8)</td>
<td>52.5% (35.5–67.4)</td>
<td>29.5% (1.6–97.1)</td>
</tr>
</tbody>
</table>

BMI = body mass index; GERD = gastroesophageal reflux disease; R = retrospective; P = prospective.
motility status, determined by manometry and relation between preoperative LES and esophageal body (SG) often have manometric abnormalities, a clear cor-
novo GERD after laparoscopic sleeve gastrectomy be determined before MBS. Although patients with de
evaluation, whether standard or high resolution, remains to
sphincter (LES) and esophageal body manometric eval-
in assisting the decision-making process before MBS.
particularly in patients without esophageal mucosal injury,
continuous ambulatory pH monitoring in addition to EGD,
controlled studies are needed to define the possible role of
GERD is defined as symptoms and/or esophageal mucosal injury that are caused by abnormal distal esopha-
geal exposure to acid [55], and a diagnosis of GERD is
not based on the finding of abnormal distal esophageal exposure to acid on continuous ambulatory pH monitoring
alone in the absence of symptoms or esophageal mucosal injury. Also, since obesity is a known risk factor for
GERD, the clinical significance of abnormal distal esophageal exposure to acid in asymptomatic patients is not yet
defined. It is conceivable that esophageal acid exposure may be a modifiable variable in at least some patients
once obesity is treated effectively. Prospective and
controlled studies are needed to define the possible role of
continuous ambulatory pH monitoring in addition to EGD,
particularly in patients without esophageal mucosal injury,
in assisting the decision-making process before MBS.
Similarly, the role of a preoperative lower esophageal sphincter (LES) and esophageal body manometric eval-
uation, whether standard or high resolution, remains to
be determined before MBS. Although patients with de
novo GERD after laparoscopic sleeve gastrectomy (SG) often have manometric abnormalities, a clear cor-
relation between preoperative LES and esophageal body
motility status, determined by manometry and
postoperative GERD outcomes, has yet to be demon-
strated [23,26,27].
Interestingly, in 25 of the 28 studies presented in Table 1,
the authors make recommendations for the use of EGD in
the pre-MBS evaluation. Seven authors [28–33,34] recom-
ended selective use of EGD before bariatric surgery based
on symptoms and other factors. The other 18 authors
[35,36–52] suggest an EGD be routinely done before surgery.
There are 2 published meta-analyses on pre-MBS EGD
[53,54]. Bennett et al. [54] examined 48 studies with a to-
tal of 12,261 patients. The proportion of preoperative
EGDs resulting in a change in surgical management was
7.8%, while medical management changes occurred in
27.5%. The authors concluded that in the asymptomatic
patient, a preoperative EGD should be considered optional. Parikh et al. [53] analyzed 28 studies with
6,616 patients. Patients were divided into 2 groups. Group
1 findings were classified as those not significantly chang-
ing management: mild/moderate duodenitis, Los Angeles
Grade A or B esophagitis, mild or moderate gastritis, H.
pylori infection, and hiatal hernia <2 cm. Group 2 find-
ings were those that caused a delay, change, or cancella-
tion of the planned procedure, such as severe duodenitis,
Los Angeles Grade C or D esophagitis, gastric varices, hi-
atral hernia >2 cm, or neoplasm. The majority (92.4%) of
the patients had a normal EGD or findings that did not
change clinical management. The approximate prevalence
of Barrett’s esophagus and esophageal carcinoma was .1%
and .08%, respectively. The authors concluded that preop-
erative EGD should be considered in selected patients,
only based on symptoms and the type of procedure planned [53].
In summary, we found that in all patients seeking MBS,
27.3% had a diagnosis of GERD and, for those both with
or without gastrointestinal symptoms, the average propor-
tions of patients with hiatal hernia, erosive esophagitis,
and BE were 21%, 16.4%, and 2.7%, respectively, with
34.6% of patients having at least 1 abnormal finding on
endoscopy. In patients seeking MBS without gastrointes-
tinal or GERD symptoms, a hiatal hernia, any erosive
esophagitis, and BE were detected in 17%, 16.9%, and
.7%, respectively.

Table 2
Studies evaluating the role of endoscopy before bariatric procedures in asymptomatic patients

<table>
<thead>
<tr>
<th>Study</th>
<th>Asymptomatic patients, n</th>
<th>Hiatal hernia, %</th>
<th>Esophagitis, %</th>
<th>Barrett’s, %</th>
<th>Positive pH test, %</th>
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<tr>
<td>Azagury, 2006 [29]</td>
<td>319</td>
<td>17</td>
<td>6.6</td>
<td>1.3</td>
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<td>Madalosso, 2008 [28]</td>
<td>34</td>
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<td>23.5</td>
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<td>44.1</td>
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<td>136</td>
<td>18</td>
<td>13.2</td>
<td>0</td>
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<tr>
<td>Mazzini, 2019 [47]</td>
<td>43</td>
<td>-</td>
<td>27.9</td>
<td>0</td>
<td>18.6</td>
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<tr>
<td>Bhambr, 2020 [52]</td>
<td>129</td>
<td>-</td>
<td>32.6</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Total n and weighted averages (range)</td>
<td>729</td>
<td>17% (13–18)</td>
<td>16.9% (6.6–39.1)</td>
<td>.7% (0–1.3)</td>
<td>39.3% (18.6–59.1)</td>
</tr>
</tbody>
</table>
Section 2: review of the definitions and standards for evaluation, screening, and surveillance of GERD and Barrett’s esophagus in the general population

GERD

GERD is a common disease and is estimated to affect 20% of the general population. GERD is defined by the presence of symptoms (heartburn, regurgitation, dysphagia, and others) and/or esophageal mucosal injury (erosive esophagitis, ulcers, stricture, and Barrett’s esophagus) caused by the abnormal reflux of gastric contents into the distal esophagus [55]. It is essential to underscore that the diagnosis of GERD based solely on symptoms, with or without an empiric trial of proton pump inhibitors (PPI), has low sensitivity and specificity, even if made by specialists (70% and 67%, respectively), when compared to objective testing [56]. The recommended algorithm for GERD diagnosis, the use of objective testing, and the testing modality may vary depending on the clinical situation [55].

In 2017, the Lyon GERD consensus meeting [57] reviewed the evidence regarding GERD diagnosis in the general population. Since clinical evaluation by symptoms and empiric PPI test yields low sensitivity and specificity [56], the consensus provided recommendations for the use and interpretation of objective testing. In summary, the absence of esophagitis on EGD does not rule out GERD in symptomatic patients and should be followed by a 24-hour or 48-hour pH test or pH-impedance testing when surgical therapy for GERD is being considered. Findings defined as conclusive of GERD in patients with GERD symptoms are EGD findings of esophagitis Los Angeles Classification grades C and D and/or a 24-hour or 48-hour pH test or pH-impedance testing with total acid exposure time greater than 6%. A total acid exposure time lower than 4% refutes the diagnosis of GERD. Borderline findings that were not conclusive of GERD included esophagitis Los Angeles Classification grades A and B and total acid exposure times between 4% and 6%.

Standard indications for screening EGD

For the general population, the Clinical Guidelines Committee of the American College of Physicians [58] currently recommends that screening EGD should not be routinely performed for heartburn symptoms in isolation for women of any age or for men aged <50 years. They recommend that EGD be performed in patients with symptoms that include heartburn associated with “alarm symptoms” (dysphagia, bleeding, vomiting, weight loss, anemia); persistent GERD symptoms despite twice-daily use of PPI therapy after 4 to 8 weeks; and for men aged ≥50 years with chronic GERD symptoms (>5 years) and additional risk factors for esophageal adenocarcinoma (nocturnal GERD, history of hiatal hernia, BMI > 35 kg/m², intra-abdominal fat distribution, and tobacco use).

In addition to the recommendation for patients with GERD symptoms, a combined statement from the American College of Gastroenterology (ACG) and the Canadian Association of Gastroenterology [59] recommends that patients experiencing symptoms of dyspepsia (defined as predominant epigastric pain lasting at least 1 month that can be associated with any other upper gastrointestinal symptom, such as epigastric fullness, nausea, vomiting, or heartburn, provided epigastric pain is the patient’s primary concern) should undergo EGD when they are aged >60 to exclude upper gastrointestinal neoplasia. These standard indications for EGD should also apply to patients seeking MBS and are summarized in Table 3.

Barrett’s esophagus

Barrett’s esophagus is accepted to be a complication of GERD in predisposed patients and is found in about 15% of patients with chronic GERD, or about 1% to 2% of the general population [60–62]. The 2016 clinical guidelines of the ACG [61] recommend that the diagnosis of Barrett’s esophagus should be made when there is an extension of salmon-colored mucosa into the tubular esophagus for at least 1 cm proximal to the gastroesophageal junction gastro-esophageal junction (GEJ), defined as the top of the rugal folds, with biopsy confirmation of intestinal metaplasia (IM; goblet cells) in simple columnar-type epithelium. It is essential to underscore that the definition of Barrett’s esophagus in the current ACG Guidelines does not include salmon-colored mucosa segments less than 1 cm above the GEJ, with or without IM. Furthermore, segments of salmon-colored mucosa that do not harbor IM—independent of the length of these segments above the GEJ—are not considered Barrett’s esophagus.

Endoscopists should follow standards for biopsy in patients suspected of having Barrett’s esophagus to accurately assess the status in an individual patient. Those standards include the following:

1. Biopsies should only be obtained for segments of salmon-colored mucosa of ≥1 cm.
2. At least 8 random biopsies should be obtained to maximize the yield of IM on histology, although in patients with short (1–2 cm) segments of suspected Barrett’s esophagus in whom 8 biopsies are unattainable, at least 4 biopsies per centimeter of circumferential Barrett’s esophagus, and 1 biopsy per centimeter in tongue of Barrett’s esophagus, should be taken.
3. The extent of metaplastic change, including circumferential and maximal segment lengths, should be described using the Prague classification [63].
Table 3: Standard indications for upper gastrointestinal endoscopy in the general population

1. Patients with symptoms that include heartburn associated with “alarm symptoms” (dysphagia, bleeding, vomiting, weight loss, anemia)
2. Persistent GERD symptoms despite twice-daily use of proton-pump inhibitor therapy after 4–8 wk
3. Men aged ≥50 yr with chronic GERD symptoms (>5 years) and additional risk factors for esophageal adenocarcinoma (nocturnal GERD, history of hiatal hernia, body mass index >35 kg/m², intra-abdominal fat distribution, and tobacco use)
4. Patients aged >60 yr experiencing symptoms of dyspepsia

Data are from references Shaheen et al. [58,61], Bolckmans et al. [68], and ASGE Standards Of Practice Committee et al. [77]. GERD = gastro-esophageal reflux disease; ASGE = American Society for Gastrointestinal Endoscopy.

4. In patients with suspected Barrett’s esophagus found to demonstrate no IM on histology, a repeat endoscopy should be considered in 1–2 years to further evaluate for Barrett’s esophagus.

Surveillance of Barrett’s esophagus to detect progression to dysplasia or early esophageal adenocarcinoma should only be done after adequate counseling regarding the risks and benefits of surveillance. Adequate counseling to choose which patient may benefit and desire surveillance is important, as 93% of patients with Barrett’s esophagus will die from causes other than esophageal adenocarcinoma, most commonly from cardiac disease (35%), followed by pulmonary disease (20%) and other malignancies (16%) [61]. If surveillance is chosen, EGD is recommended to be repeated in patients with an established diagnosis of nondysplastic Barrett’s esophagus at intervals not to exceed 3 to 5 years.

Notably, the currently estimated cancer risk in Barrett’s esophagus is low for all patients except those with high-grade dysplasia. However, the risk varies depending on the segment length and presence or severity of dysplasia:

1. For nondysplastic Barrett’s esophagus of any length, the risk of cancer progression is .35% (.2%–.5%) per year.
2. For nondysplastic short-segment Barrett’s esophagus <3 cm, the annual risk of progression to cancer is .19% per year.
3. For Barrett’s esophagus with low-grade dysplasia, the annual risk of progression to cancer is .7% per year.
4. For Barrett’s esophagus with high-grade dysplasia, the annual risk of progression to cancer is 7% per year [61,64–66].

Section 3: review of the literature on GERD, erosive esophagitis, and Barrett’s esophagus after MBS using objective testing

There is a body of evidence showing that RYGB improves GERD outcomes and, to date, there are no reports of de novo Barrett’s esophagus after RYGB [15,24–26]. In addition, the 2 series where objective endoscopic and 24-hour pH monitoring were used before and after RYGB both demonstrated significant improvements in GERD by objective measurements [15,67]. Madalosso et al. [15] showed that 3 years after RYGB in 53 patients, the mean composite score on 24-hour pH monitoring decreased from 28.6 to 1.2 (normal <14.76), and erosive esophagitis resolved or improved in 83.4% of patients. Similarly, Rebecchi et al. [67] showed that 5 years after RYGB in 72 patients with complete follow-up (37% with abnormal acid exposure in 24-hr multichannel intraluminal impedance-pH monitoring pre-operatively), esophageal acid exposure measured again by 24-hour pH-impedance monitoring was normalized in the group of patients with abnormal preoperative pH tests. Of note, while the authors detected an increased prevalence of weakly acid reflux after RYGB (the clinical significance of which is currently unknown), they reported a decrease in “macroscopic” endoscopic esophagitis (definition and esophagitis grade not reported), and no patients developed esophageal intestinal metaplasia at 60 months of follow-up.

A summary of 11 studies in which endoscopic findings were noted before and after SG has been reported [68]. These 11 studies [22,23,69–76] are included in Table 4. There were cumulatively 1192 patients at baseline and 1004 patients at follow-up. The follow-up period ranged from 1 year to more than 10 years. Two studies [23,69] reportedly excluded patients with GERD symptoms or abnormal EGD findings at baseline. In the remaining 9 studies, at baseline, erosive esophagitis was found in 17.4% (range, 7%–36%), and hiatal hernia at baseline was reported in 7 studies, with an average proportion of hiatal hernia of 17.7% (range, 6.1%–31%). After SG, all but 1 study [71] reported an increase in the rates of esophagitis. At follow-up, esophagitis was found in 36.3% of patients (range, 8%–67%). Eight studies [23,26,69,72–76] reported the rates of hiatal hernia at follow-up. Hiatal hernia was found in 22.7% of patients (range, 5.3%–60%). When analyzing the 2 studies that excluded patients with symptoms or objective findings of GERD at baseline, there were 253 patients at follow-up, and the de novo rates of esophagitis and hiatal hernia were 25.3% and 11.1%, respectively. The rates of PPI use were inconsistently reported across all the studies, and PPI use might have influenced the results, leading to underestimations of the rates of esophagitis, especially after surgery.

Four case series (3 from Europe and 1 from Chile) have reported rates of GERD, erosive esophagitis, and de novo Barrett’s esophagus after SG [22,23,69,70] (Table 5). GERD symptoms at follow-up were found in 67.4% of patients (range, 57%–76%) when aggregating data from the 4 series, while erosive esophagitis was found in 38.4% (range, 16%–59.8%) and Barrett’s esophagus was
Table 4  
Studies evaluating the GERD outcomes after primary SG using objective testing

<table>
<thead>
<tr>
<th>Study design</th>
<th>Study population</th>
<th>Sleeve technique</th>
<th>N at baseline</th>
<th>Baseline EE, %</th>
<th>Baseline HH, %</th>
<th>Follow-up period</th>
<th>No. with EGD at follow-up</th>
<th>Follow-up EE, %</th>
<th>Follow-up HH, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>Primary SG</td>
<td>4 cm pylorus</td>
<td>86</td>
<td>16.7</td>
<td>6.1</td>
<td>1 year</td>
<td>66</td>
<td>67</td>
<td>27.3</td>
</tr>
<tr>
<td>P</td>
<td>Primary SG; no large hiatal hernia, gastric surgery</td>
<td>36 Fr bougie</td>
<td>71</td>
<td>12.3</td>
<td>11.3</td>
<td>2 yr</td>
<td>65</td>
<td>8</td>
<td>-</td>
</tr>
<tr>
<td>P</td>
<td>Primary SG</td>
<td>4–5 cm pylorus</td>
<td>35</td>
<td>18.8</td>
<td>25</td>
<td>1 year</td>
<td>32</td>
<td>25</td>
<td>34.4</td>
</tr>
<tr>
<td>R</td>
<td>No GERD symptoms, esophagitis, BE, or hiatal hernias</td>
<td>6 cm pylorus 42–48 Fr bougie</td>
<td>97</td>
<td>0 (excluded)</td>
<td>0 (excluded)</td>
<td>≥10 yr</td>
<td>44</td>
<td>16</td>
<td>39</td>
</tr>
<tr>
<td>P</td>
<td>Primary SG</td>
<td>6 cm pylorus</td>
<td>144</td>
<td>36.1</td>
<td>19.4</td>
<td>mean 5.5 yr (range 3.4–7.4)</td>
<td>144</td>
<td>60</td>
<td>-</td>
</tr>
<tr>
<td>P</td>
<td>Primary SG</td>
<td>4–6 cm pylorus</td>
<td>109</td>
<td>20.1</td>
<td>22</td>
<td>1.5 yr</td>
<td>109</td>
<td>30</td>
<td>34.8</td>
</tr>
<tr>
<td>P</td>
<td>Primary SG</td>
<td>3 cm pylorus</td>
<td>63</td>
<td>14.3</td>
<td>-</td>
<td>1 year</td>
<td>63</td>
<td>44.4</td>
<td>19</td>
</tr>
<tr>
<td>R</td>
<td>Primary SG with ≥5 y FU; no GERD symptoms, esophagitis, HH, BE</td>
<td>34 Fr bougie</td>
<td>315</td>
<td>0 (excluded)</td>
<td>0 (excluded)</td>
<td>≥5 yr (mean 7.1±2.3)</td>
<td>209</td>
<td>27</td>
<td>5.3</td>
</tr>
<tr>
<td>P</td>
<td>Primary SG with ≥5 y FU; EGD baseline no BE</td>
<td>Not mentioned</td>
<td>90</td>
<td>10</td>
<td>-</td>
<td>≥5 yr (mean 6.5±1)</td>
<td>90</td>
<td>40</td>
<td>-</td>
</tr>
<tr>
<td>R</td>
<td>Primary SG</td>
<td>6 cm pylorus</td>
<td>147</td>
<td>7</td>
<td>16</td>
<td>1 year</td>
<td>147</td>
<td>29</td>
<td>22</td>
</tr>
<tr>
<td>P</td>
<td>Primary SG regardless of GERD symptoms</td>
<td>4 cm pylorus 32 Fr bougie</td>
<td>35</td>
<td>11</td>
<td>31</td>
<td>1 year</td>
<td>35</td>
<td>54.1</td>
<td>60</td>
</tr>
</tbody>
</table>

Total and weighted averages (range)  

1192  
17.4% (7%–36.1%)  
17.7% (6.1%–31%)  
1004  
36.3% (8%–67%)  
22.7% (5.3%–60%)  
25.3%  
11.1%

GERD = gastroesophageal reflux disease; SG = sleeve gastrectomy; EE = erosive esophagitis; HH = hiatal hernia; R = retrospective; P = prospective; EGD = esophagogastroduodenoscopy; SG = sleeve gastrectomy; FU = follow-up.  
* Calculated from the studies without esophagitis and hiatal hernia at baseline. Adapted from Bolckmans et al. [68].
identified overall in 10.7% (range, 4.8%–18.8%). It is noteworthy that all reported cases of de novo Barrett’s esophagus after SG were nondysplastic and had short segments (≤3 cm). Of the 4 case series, 2 included patients with GERD symptoms at baseline (20% and 41%) [22,70]. However, all 4 studies reported that none of the patients had Barrett’s esophagus at the pre-MBS, baseline EGD. Of the 4 studies, 3 also specified the proportions of patients with and without GERD symptoms [22,69,70]. Of 49 patients with de novo Barrett’s esophagus after SG, 16.7% were asymptomatic (weighted average of the proportions).

There are significant limitations in all 4 case series [22,23,69,70] that should be underscored and considered while interpreting the finding of an overall 10.7% incidence of de novo Barrett’s esophagus after SG. As stated, the ACG recommends that only segments of salmon-colored mucosa of ≥1 cm above the GEJ with IM be considered as Barrett’s esophagus [61]; however, 2 of the studies did not state whether the ACG definition [23,69] was followed and, per the described biopsy protocol, may have included cases that did not fit that criteria. The other 2 series [22,70], while stating they utilized the ACG definition, failed to document circumferential and maximum extent of salmon-colored mucosa above the GEJ, as is recommended.

Most importantly, all 4 series lack a comparison group of patients not exposed to SG and receiving either other surgical treatment options or usual care. Thus, it is unknown whether the 10.7% rate of de novo Barrett’s esophagus after SG would have been reproduced if a similar population were followed without SG and therefore remained with obesity as a refluxogenic factor for a comparable period. Additional important unanswered questions include the following:

1. Would the weight loss and metabolic benefits of SG outweigh the possible risk associated with de novo or persistent GERD and Barrett’s esophagus after SG?
2. Can technical modifications be made to SG that would reliably decrease the reported rate of GERD and Barrett’s esophagus?
3. Were all SG procedures in the 4 studies done following current technical standards?
4. What are the roles of weight regain or weight failure, which were noted in many patients in these observational studies?

With these important limitations in mind, the 10.7% rate of de novo Barrett’s esophagus identified in these 4 studies is comparable to the American Society for Gastrointestinal Endoscopy recommendation that any population at an estimated 10% risk for Barrett’s esophagus should undergo endoscopic screening [77]. However, while better-designed studies are needed to confirm or refute the evidence presented in this review, until better data are available, it may
be advisable for clinicians to offer screening for Barrett’s esophagus in SG patients 3 or more years post-SG, irrespective of GERD symptoms, in addition to offering screening for those with the standard indications as presented in Section 2.

**Recommendations for upper gastrointestinal endoscopy before and after bariatric surgery**

**Upper gastrointestinal endoscopy before bariatric surgery**

In addition to the currently accepted guidelines that apply to the general population (Table 3), and based on the data presented, we recommend that

1. All patients seeking MBS should be interviewed to determine the presence of gastrointestinal symptoms, including GERD symptoms. It is important to underscore that clinical evaluation by symptoms alone does not reliably diagnose or rule out GERD or other upper gastrointestinal abnormalities that may need treatment before bariatric surgery, may indicate the need for an adjunctive procedure (e.g., hiatal hernia repair), and/or may affect GERD outcomes after surgery. This information should be taken into account when counseling patients about preoperative EGD, surgical options, and GERD outcomes. Although there might be potential attrition of patients by requiring EGD before MBS, the current scientific evidence suggests that preoperative EGD, even in asymptomatic patients, may guide treatment of modifiable conditions before MBS (duodenal ulcer, *H. pylori* infection, others), lead to identification of associated anatomical abnormalities that could be treated during the bariatric operation (e.g., hiatal hernia), assist in tailoring the choice of surgical approach, or guide a decision not to proceed with MBS altogether.

2. Given the current evidence of GERD outcomes following RYGB and SG, patients with conclusive and objective evidence of preoperative GERD (EGD finding of esophagitis Los Angeles Classification grades C and D and/or a 24-hr or 48-hr pH test or pH-impedance testing with total acid exposure time greater than 6%) or severe GERD symptoms are better served by current techniques of RYGB rather than SG. However, the effect on GERD is not the only outcome of importance in the decision to offer one procedure over another and there may be other compelling reasons to consider offering a SG to a patient, even with existing GERD. Thus, the surgeon, multidisciplinary team, and patient, may reasonably decide to proceed with SG rather than RYGB, once the risks of persisting GERD are discussed.

**Upper gastrointestinal endoscopy after bariatric surgery**

In addition to the currently accepted guidelines for EGD indications that apply to the general population (Table 3), and based on the preliminary and significant limited evidence as described regarding the possibility of development of de novo Barrett’s esophagus after SG, even in asymptomatic patients, it is conditionally recommended that, until better-designed studies either confirm or refute the evidence presented, clinicians should consider a screening EGD for all patients with gastrointestinal symptoms, including GERD symptoms, after MBS and in patients 3 or more years after SG, irrespective of symptoms. It may also be reasonable to offer further EGD screening every 5 years afterward if the index screening EGD is normal.

**Summary**

1. Clinical evaluation by symptoms alone does not reliably diagnose or rule out GERD, and upper gastrointestinal abnormalities are found in a significant proportion of patients undergoing EGD before bariatric surgery, even in asymptomatic patients. While some of these findings do not modify medical or surgical management, routine preoperative EGD is justifiable and should be done at the surgeon’s discretion.

2. Patients with conclusive and objective evidence of preoperative GERD (EGD finding of esophagitis Los Angeles Classification grades C and D and/or a 24-hr or 48-hr pH test or pH-impedance testing with total acid exposure time greater than 6%) or severe GERD symptoms are better served by current techniques of RYGB rather than SG. However, the effect on GERD is not the only outcome of importance in the decision to offer one procedure over another and there may be other compelling reasons to consider offering a SG to a patient, even with existing GERD. Thus, the surgeon, multidisciplinary team, and patient, may reasonably decide to proceed with SG rather than RYGB, once the risks of persisting GERD are discussed.

3. There have been reports of de novo Barrett’s esophagus after SG, but not after RYGB. All cases reported to date after SG were nondysplastic and involved only short-segment Barrett’s esophagus. Long-term clinical implications in the setting of bariatric surgery are not yet known and require further well-designed studies using widely accepted criteria to confirm Barrett’s esophagus.

4. After bariatric surgery, screening with EGD should be considered for all patients with gastrointestinal symptoms, including GERD symptoms. It is reasonable to perform EGD on patients ≥3 years after SG, irrespective of GERD symptoms, to rule out Barrett’s esophagus. More long-term surveillance every 5 years after that would be reasonable even if the index screening EGD is normal and is compatible with clinicians exercising an abundance of caution until better-designed and longer-term studies are available.
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