

The role of endoscopy in gastroduodenal obstruction and gastroparesis

This is one of a series of statements discussing the use of GI endoscopy in common clinical situations. The Standards of Practice Committee of the American Society for Gastrointestinal Endoscopy (ASGE) prepared this text. In preparing this guideline, a search of the medical literature was performed by using PubMed. Additional references were obtained from the bibliographies of the identified articles and from recommendations of expert consultants. Guidelines for appropriate use of endoscopy are based on a critical review of the available data and expert consensus at the time the guidelines are drafted. Further controlled clinical studies may be needed to clarify aspects of this guideline. This guideline may be revised as necessary to account for changes in technology, new data, or other aspects of clinical practice. The recommendations are based on reviewed studies and are graded on the strength of the supporting evidence (Table 1).¹ The strength of individual recommendations is based both on the aggregate evidence quality and an assessment of the anticipated benefits and harms. Weaker recommendations are indicated by phrases such as “We suggest . . .,” whereas stronger recommendations are typically stated as “We recommend . . .”

This guideline is intended to be an educational device to provide information that may assist endoscopists in providing care to patients. This guideline is not a rule and should not be construed as establishing a legal standard of care or as encouraging, advocating, requiring, or discouraging any particular treatment. Clinical decisions in any particular case involve a complex analysis of the patient's condition and available courses of action. Therefore, clinical considerations may lead an endoscopist to take a course of action that varies from these guidelines.

Enteral obstruction and delayed gastric emptying can result from a variety of benign and malignant conditions. Endoscopy is an important tool in the evaluation of these patients and can identify, localize, or exclude structural causes. Moreover, various endoscopic procedures may be used to treat the underlying etiology or alleviate symptoms.

This document describes the role of endoscopy in known and suspected obstruction of the proximal GI tract. A discussion of special considerations in a pediatric population is also included.

ETIOLOGY AND PRESENTATION

Gastric outlet obstruction (GOO) is caused by mechanical gastroduodenal obstruction or motility disorders and can be divided into 3 major categories: benign mechanical, malignant mechanical, and motility disorders (Table 2). Peptic ulcer disease with or without secondary stricture is the most common cause of benign mechanical GOO, although the recent decline in peptic ulcer disease has decreased the incidence of clinically evident peptic strictures.² Malignant mechanical GOO usually results from cancer affecting the distal stomach or proximal duodenum. Gastric and pancreatic cancers are the most common malignant mechanical causes of GOO.³

The most common gastric motility disorder is gastroparesis, often resulting from long-standing diabetes, although gastroparesis may also be idiopathic, viral, or related to medications.⁴⁻⁶ Surgical procedures that intentionally or unintentionally disrupt the vagus nerve (eg, procedures for peptic ulcer disease, bariatric procedures, esophagectomy, fundoplication) may also result in gastroparesis. Several solid and hematologic malignancies may induce gastroparesis and small-bowel dysmotility through a paraneoplastic process or secondary infiltrative diseases (eg, amyloidosis, carcinomatosis).^{7,8}

Patients with GOO may present with nausea and vomiting, weight loss, abdominal bloating, early satiety, and/or abdominal discomfort. Because of shared clinical features, it is often difficult to distinguish motility disorders from mechanical obstruction or functional dyspepsia based solely on symptoms.^{9,10} Nevertheless, initial evaluation should include a detailed history and careful physical examination. Vomiting soon after a meal suggests an upper anatomic abnormality, whereas symptoms delayed for several hours after meals characterize gastroparesis or a more distal obstruction.¹¹ Vomiting will frequently relieve symptoms from a proximal obstructive cause. GOO may not be clinically evident until high-grade obstruction occurs because of the ability of the stomach to distend significantly to accommodate contents. Patients with GOO may demonstrate a succussion splash on physical examination.

Table 1. GRADE System for rating the quality of evidence for guidelines

Quality of evidence	Definition	Symbol
High quality	Further research is very unlikely to change our confidence in the estimate of effect	⊕⊕⊕⊕
Moderate quality	Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate	⊕⊕⊕○
Low quality	Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate	⊕⊕○○
Very low quality	Any estimate of effect is very uncertain	⊕○○○

Adapted from Guyatt et al.¹

Table 2. Differential diagnosis of gastric outlet obstruction

Mechanical	Motility disorders
Benign	
Peptic ulcer disease	Gastroparesis
Crohn's disease	Postsurgical gastroparesis
NSAID-related stricture	Medication-associated dysmotility
Anastomotic stricture	Systemic disease-associated (eg, scleroderma, amyloidosis)
Postradiation stricture	Intestinal pseudo-obstruction
Foreign body or bezoar	Paraneoplastic syndrome
Gallstone (Bouveret syndrome)	
Benign polyps (eg, antral polyps, inflammatory, hyperplastic, inflammatory pseudotumor, hamartoma, Peutz-Jeghers syndrome)	
Eosinophilic gastroenteritis	
Extrinsic compression (eg, annular pancreas, chronic pancreatitis with/without pseudocyst)	
Malignant	
Gastroduodenal cancer, gastric lymphoma (eg, MALT lymphoma), pancreatic cancer, cystic neoplasm of the pancreas, gallbladder and bile duct cancer, carcinoid, retroperitoneal lymphadenopathy (eg, metastatic tumor, lymphoma), retroperitoneal sarcoma, leiomyosarcoma, GI stromal tumor	
Children	
Hypertrophic pyloric stenosis, duodenal or pyloric atresia, antral and duodenal webs, gastroduodenal duplication, gastroduodenal intussusception and gastric volvulus, heterotopic pancreatic tissue in the gastric antrum, diaphragmatic herniation, malrotation and peritoneal fibrous bands, congenital anomalies of the pancreatobiliary system, foreign body, peptic ulcer disease, eosinophilic GI disease, chronic granulomatous disease, Crohn's disease, lymphoproliferative disease	

MALT, Mucosa-associated lymphoid tissue; NSAID, nonsteroidal anti-inflammatory drug.

EVALUATION

Most patients with signs or symptoms of gastroduodenal obstruction or dysmotility will require structural evaluation with EGD and/or radiographic studies. If complete intestinal obstruction or perforation is suspected, initial evaluation with radiographic studies should be performed

before endoscopy. CT is the preferred radiologic test for suspected intestinal obstruction.¹²⁻¹⁴ Because oral barium contrast may interfere with subsequent endoscopy, its use should be minimized or avoided if endoscopy is anticipated. Furthermore, high osmolar water-soluble contrast agents can cause severe bronchial irritation and pulmonary edema when inadvertently aspirated in the setting of

obstruction and thus should be used with extreme caution.¹⁵ Endoscopic examination after gastric decompression can usually identify the nature and the precise level of obstruction, but the degree of the stenosis often does not correlate with symptoms. Endoscopy also offers the capability of tissue sampling and endoscopic therapy, where indicated.

When structural abnormalities have been excluded, GI motility can be evaluated by using scintigraphy, radiographic contrast techniques, breath testing, electrogastrography, or gastroduodenal manometry. A comprehensive technical review of the diagnosis of gastroparesis was published in 2004.¹⁶ Gastroduodenal manometry can be performed to differentiate intestinal myopathy from enteric or extrinsic neuropathy, but the availability of this test is limited and may not influence therapy.¹⁷⁻¹⁹ A wireless pH and motility capsule has been developed that can assist with assessing GI motility,^{20,21} although its clinical utility remains to be defined.²²

TREATMENT

Benign mechanical obstruction

Treatment options for benign mechanical obstruction include balloon dilation, self-expandable metal stent (SEMS) placement, and surgery. GOO related to peptic ulcer disease can be treated with balloon dilation.²³⁻²⁶ Although technical success with immediate symptom improvement is common, multiple dilations are often required.²³ Perforation rates with balloon dilation in benign peptic strictures range from 3% to 7%, with higher rates corresponding to larger balloon diameter of more than 15 mm.^{23,24,27,28} Balloon dilation can also be effective in the treatment of caustic-induced GOO or post-endoscopic submucosal dissection stricture at the pylorus.^{29,30}

Once adequate dilation is achieved, a durable response is seen in 70% to 80% of patients.²³⁻²⁵ Treatment of *Helicobacter pylori*, when present, elimination of nonsteroidal anti-inflammatory drugs, and concurrent use of antisecretory therapy may improve sustained response.³¹ The efficacy of proton pump inhibitor therapy may be attenuated in the setting of GOO because of a failure to reach the jejunum for absorption and premature activation in the acidic environment of stomach.³¹ Recurrent stricture after endoscopic dilation may require surgical treatment. In one study, the need for more than 2 endoscopic dilations for symptoms was a significant predictor for the need for surgical treatment.³² Although there have been case reports of SEMS placement for the treatment of benign stenosis of the pylorus, the experience with these devices in this patient population is very limited.^{26,33,34}

Malignant mechanical obstruction

General considerations. Treatment options for malignant GOO include surgical resection, surgical bypass, endoscopic stenting, and palliative decompressive gas-

trostomy with or without feeding tube placement. Surgery is the preferred strategy for those patients who are potential candidates for curative resection. Diagnostic laparoscopy or exploratory laparotomy may be beneficial to assess the extent of disease with intent to perform surgical bypass as deemed necessary. Endoscopic placement of an SEMS should be considered provided there is no evidence of obstruction distal to the site of planned stent deployment. In patients with multiple sites of obstruction, palliative decompressive gastrostomy can be considered with jejunal feeding tube placement or total parenteral nutrition (TPN).

Endoscopic SEMS placement. SEMS are composed of metal alloys designed to be constrainable on a delivery catheter, yet resume their desired shape once the constraint is removed. Although some can be delivered through the endoscope, others have a larger delivery system that requires placement alongside the endoscope and/or with fluoroscopic guidance. Some SEMSs are covered by a membrane to help prevent tumor ingrowth. A detailed discussion of enteral stents is available in another ASGE document.³⁵

Technical and clinical success of endoscopically placed SEMSs. Technical success is defined as the successful deployment of the stent at the desired anatomic location, whereas relief of obstructive symptoms and/or improvement of oral intake define clinical effectiveness. Attempts to place an SEMS may fail because of the inability to pass the guidewire beyond the level of the obstruction or other anatomic difficulties. Clinical improvement is commonly assessed by the Gastric Outlet Obstruction Score,³⁶ quality of life, and performance status.³⁷

Case series of SEMS placement for gastroduodenal obstruction have found high technical and clinical success rates in patients with malignant GOO.^{3,38-45} It is important to note that such studies are often composed of heterogeneous patient populations with various malignancies treated with an assortment of stents, making uniform conclusions about efficacy difficult. A systematic review of 32 case series summarized the technical success and clinical effectiveness of SEMSs.³ The mean survival time was 12 weeks (range 1-184 weeks). The technical success rate of endoscopic placement of SEMSs was 97%³ and ranged from 91% to 100% in prospective studies.³⁸⁻⁴⁵ Clinical success was 89% overall, ranging from 63% to 95%.^{3,38-45} Such discrepancies between technical success and clinical success are seen uniformly across prospective studies and may be attributed to underlying GI dysmotility with or without neural involvement by tumor, distal obstruction secondary to peritoneal carcinomatosis, or general deconditioning and anorexia caused by advanced malignancy.^{38,39,46} In the systematic review, the mean time to resuming oral intake after SEMS placement was 4 days, and 48% were able to resume a full diet, 39% were tolerating soft solids, and 13% were on liquids only.³ Therefore,

Table 3. Adverse events of endoscopically placed self-expandable metal stents

Bleeding
Perforation
Peritonitis
Sepsis
Aspiration
Pain
Biliary obstruction
Pancreatitis
Cholangitis
Stent migration
Stent dysfunction
Tumor ingrowth
Tumor overgrowth
Food impaction

patients undergoing SEMS placement need to be informed of likely limitations on oral intake, including the avoidance of foods that may result in stent occlusion. In addition, although SEMS placement may significantly improve obstructive symptoms, improvement in quality of life and performance status is not consistently demonstrated in this patient population.^{41-43,47}

Contraindications and complications of enteral SEMSs. Contraindications to SEMS placement include those conditions that generally preclude endoscopic procedures (eg, severe cardiopulmonary disease, perforated viscus).

Complications of enteral stents are listed in Table 3 and include severe complications (eg, perforation and bleeding) in approximately 1% of cases. Nonsevere complications (eg, stent malfunction, pain, and occlusion of the ampullary orifice leading to pancreatitis and/or cholangitis) are fairly common, occurring in approximately one fourth of cases.^{3,36,40} Stent malfunction caused by tumor ingrowth, food impaction, or stent migration is the most commonly reported complication (17%) and is typically managed by insertion of additional stents and/or clearance of the food impaction. Stent migration within 8 weeks of placement was significantly more common with covered SEMSs (currently not available in the United States) compared with uncovered SEMSs (28% vs 3%; $P = .009$).⁴⁵ Repositioning or removal of distally migrated stents can be attempted when recognized early.^{35,43} Placement of an additional SEMS is usually effective if repositioning fails. Completely migrated stents can cause intestinal obstruction requiring surgical intervention.^{39,45}

Approach to the patient with combined enteral and biliary obstructions. Patients with malignant gastroduodenal obstruction commonly present with or experience the development of coincident biliary obstruction. In a systematic review of 243 patients, 61% of patients receiving a duodenal stent also required a biliary stent.³ Biliary stent placement was performed before duodenal stenting in 41% or at the time of duodenal stenting in 18%, with an additional 2% undergoing stenting afterward. In most cases, duodenal stents do not appear to obstruct bile flow even when covered stents bridging the ampulla are used.⁴⁸ Although successful deployment of biliary stents through the interstices of the duodenal stent has been reported,^{49,50} this approach is technically more difficult, and in most cases percutaneous, transhepatic placement is needed. For this reason, biliary SEMSs (not plastic stents) should be considered before duodenal SEMSs in patients with known or impending biliary obstruction and GOO.

Percutaneous decompressive gastrostomy. In poor surgical and SEMS candidates with malignant gastroduodenal obstruction, peritoneal carcinomatosis, and/or diffuse bowel strictures caused by metastatic lesions, decompressive gastrostomy either by percutaneous endoscopic gastrostomy (PEG) or percutaneous radiologic gastrostomy (PRG) methods may be beneficial. PEG with jejunal extension allows decompression in addition to access for enteral nutrition.⁵¹ Decompressive PEG or PRG was reported to be of significant clinical benefit with high rates of symptom relief (approximately 90%) and avoidance of nasogastric tube decompression.^{52,53} In a study of 370 patients, PRG was reported to have a higher 30-day complication rate than PEG (23% vs 11%, $P = .038$), including infections and inadvertent tube removal.⁵⁴ Ascites is considered a relative contraindication to percutaneous gastrostomy placement.⁵⁵ However, paracentesis before gastrostomy placement may facilitate the successful placement of PRG with low complication rates.^{56,57}

Comparative studies of endoscopic and surgical palliation of malignant GOO. The optimal modality for palliation of malignant GOO has been a focus of debate. In a systematic review, patients treated with enteral stents were more likely to tolerate oral intake (odds ratio 2.62; 95% CI, 1.17-5.86; $P = .02$) and to resume oral intake more quickly (mean difference 7 days) than patients treated with gastrojejunostomy.⁵⁸ Furthermore, patients receiving enteral stents had a shorter hospital stay (mean difference 12 days). There were no significant differences in mortality, complication rates, or overall survival. In a retrospective study of 95 patients, those undergoing SEMS placement had a more rapid development of late (>7 days) complications including recurrent obstructive symptoms and need for reintervention during 3 months of follow-up, indicating a more durable effect of gastrojejunostomy.⁵⁹ Three prospective, randomized studies comparing SEMS and surgery have been reported.^{47,60,61} One study has shown improvement in quality-of-life score with SEMS but

none with surgical bypass,⁴⁷ whereas another did not show a difference between the groups.⁶¹ All 3 studies showed comparable rates of technical success and mortality, and longer hospital stay with surgery.^{47,60,61} SEMS placement was associated with more rapid improvement in symptoms.^{60,61} In the recent largest randomized study with longer follow-up, late complications (ie, recurrent obstruction and need for reintervention) were more common with an SEMS than with gastrojejunostomy, confirming the results of the previous retrospective study suggesting the benefit of surgical gastrojejunostomy in patients with longer life expectancy.^{59,61}

Multiple studies have compared the cost of endoscopic stenting with those of gastrojejunostomy for palliation and have uniformly found that an endoscopic approach was more cost-effective.⁶¹⁻⁶⁴ A decision-analytic model comparing open gastrojejunostomy, laparoscopic gastrojejunostomy, and endoscopic stenting for malignant gastroduodenal obstruction showed that SEMS placement was the most cost-effective strategy and was associated with the lowest rate of complications and the highest success rate over a 1-month period.⁶⁵ Therefore, although surgical palliation offers more durable results than SEMS placement, SEMS placement would be a more appropriate option for those patients with poor performance status and/or a short life expectancy. Ultimately, the palliative approach chosen should depend on local expertise and the patient's prognosis and preferences.

Motility disorders

Medical therapies. Whenever possible, medications that delay gastric emptying or slow intestinal transit (eg, narcotics, anticholinergics, calcium channel blockers) should be discontinued in patients with dysmotility of the upper GI tract. Glycemic control should be optimized in diabetic patients because hyperglycemia may delay gastric emptying and reduce antral contractility independent of the presence or absence of diabetic neuropathy.^{66,67} Dietary measures that may reduce symptoms include consumption of small, frequent meals that are low in fat and low residue. In severe cases, ingestion of calories in a liquid rather than a solid form may be beneficial. Prokinetic and antiemetic medications may be used to increase gastric contractility, promote gastric emptying, and reduce symptoms overall. Metoclopramide and domperidone act as dopamine receptor antagonists in the stomach to improve gastric emptying and block emetic pathways in the brainstem. However, domperidone is not approved by the U.S. Food and Drug Administration and is only available in the United States as a compounded drug. Metoclopramide, unlike domperidone, crosses the blood-brain barrier resulting in side effects (eg, fatigue, drowsiness, irritability, acute dystonic reactions) that may limit clinical use. Infrequently, metoclopramide may produce Parkinson-like symptoms or tardive dyskinesia that may not resolve with discontinuation of the medication and have led to a black-

box warning from the U.S. Food and Drug Administration recommending that its continuous use not exceed 3 months. The macrolide antibiotics, including erythromycin, azithromycin, and clarithromycin, act as motilin-receptor agonists to stimulate gastric motility. Although erythromycin is a potent stimulant of gastric emptying, side effects are common with oral use (eg, nausea, vomiting, abdominal cramping, diarrhea). Furthermore, tachyphylaxis often will limit long-term efficacy.

Endoscopic therapies. When gastroduodenal dysmotility is associated with weight loss, recurrent episodes of dehydration, or electrolyte disturbances, supplemental nutrition via enteral or parenteral routes should be considered. In patients with isolated gastric dysmotility, postpyloric enteral nutrition is preferable to TPN, given the costs and potential side effects (eg, infection, vascular thrombosis, steatohepatitis) associated with TPN. A detailed review of the treatment of gastroparesis, including timing and indications for enteral nutrition supplementations⁴ and a guideline for the role of endoscopy in enteral feeding⁵⁵ have previously been published. PEG may also be used to facilitate gastric decompression in selected individuals.⁶⁸⁻⁷¹

Botulinum toxin is a neurotoxin that irreversibly binds to cholinergic receptors and impairs acetylcholine release.⁷² Botulinum toxin has been evaluated for the treatment of gastroparesis and is typically injected in a radial pattern at or within 2 cm of the pylorus, with a total dose of 100 to 200 units. Numerous uncontrolled studies have shown symptom reduction in patients with gastroparesis treated with pyloric botulinum toxin injection.⁷³⁻⁷⁷ However, 2 placebo-controlled trials involving a small number of patients (55 total) showed no significant benefit.^{76,78} If there are benefits from endoscopic botulinum toxin injection, they may depend on the dose used and patient selection. In a retrospective cohort study of 179 patients, doses of 200 units were beneficial in a significantly greater proportion of patients than doses of 100 units (77% vs 54%, $P = .02$).⁷⁷ In this same study, multivariate analysis showed that female sex, age younger than 50 years, and etiology other than diabetes or surgical vagal nerve manipulation were associated with an improved response to therapy. The reported duration of benefit from pyloric botulinum toxin ranges from 1 to 5 months, and repeated injections may be associated with the return of clinical response in a subset of patients.^{77,79}

In patients with gastroduodenal dysmotility and symptoms refractory to medical or botulinum toxin therapy, placement of decompressive gastrostomy can be effective.^{69,71} In a small ($N = 8$) series of women with idiopathic gastroparesis, placement of a venting gastrostomy was associated with significant improvement in symptoms and weight gain that was sustained at 3 years.⁶⁹ There are no published studies of endoscopic dilation of the pylorus using balloons or bougienage dilators in patients with gastroparesis.

Gastric pacing. Gastric pacing using electrical stimulation delivered via electrodes implanted in the peritoneal side of the anterior stomach wall has been used in the treatment of gastroparesis refractory to medical or endoscopic therapies. Leads are typically inserted surgically, although there has been a reported case series (N = 20) of temporary gastric pacing using an endoscopic technique.⁸⁰ An open-label, multicenter study of 38 patients showed a decrease in nausea and vomiting, as well as weight gain in 35 patients treated with gastric pacing,⁸¹ but sham stimulation–controlled studies have produced lesser clinical responses.^{81,82} Complications associated with these devices occur in as many as one fourth of patients and include infection, lead dislodgment, and wire breakage. The relatively high rate of complications led the U.S. Food and Drug Administration to limit the use of the device to humanitarian indications and to centers in which the local institutional review board has approved its use. Contraindications to device placement include diffuse motility disorders (eg, amyloidosis, scleroderma), previous gastric resections, and the presence of other neurostimulating or pacing (including cardiac) devices.

Surgical therapies. There are a variety of surgical interventions that have been performed for the treatment of severe, refractory gastroparesis including pyloroplasty, complete or partial gastrectomy, or feeding jejunostomy, although there are no randomized trials.⁸³ In a retrospective study of 26 patients with diabetic gastroparesis who had undergone surgical jejunostomy placement, 83% reported improved overall health, although only 39% reported symptom improvement.⁸⁴ In a large (N = 81) retrospective study, 80% of patients with postsurgical gastroparesis who had undergone near-total gastrectomy with Roux-en-Y reconstruction reported long-term symptom relief.⁸⁵ In contrast, a second study reported symptom improvement in only 43% of 62 patients who had undergone the same surgery for severe postvagotomy gastroparesis.⁸⁶

SPECIAL CONSIDERATIONS FOR THE PEDIATRIC POPULATION

GOO in early infancy often results from congenital defects of the upper GI tract (Table 2). Hypertrophic pyloric stenosis, the most common cause of GOO in children, typically presents in early infancy. Diagnosis is directed by the clinical picture and radiologic evaluation. Clinical features include those typical of upper intestinal obstruction (eg, vomiting), although a history of polyhydramnios during pregnancy may signify the presence of in utero obstruction before delivery. Plain abdominal x-rays may show the absence of gas beyond the stomach or the typical “double-bubble” of duodenal atresia; the second air fluid level is from a distended proximal duodenum and a markedly distended gastric cavity. An upper GI contrast study is typically the next investigation performed, al-

though abdominal US or CT may be necessary for determination of the source of obstruction before surgery. Hypertrophic pyloric stenosis is diagnosed as a palpable pyloric mass confirmed with transabdominal US or an upper GI contrast study.⁸⁷ Endoscopy is not indicated in the management of pyloric stenosis; however, high-resolution EUS may be useful in imaging the pyloric mass in equivocal cases,⁸⁸ and pneumatic balloon dilation has been used successfully in cases presenting outside of infancy.^{89,90}

It is important to note that obstructing lesions in the gastric cavity, such as an antral web and a pedunculated mass, may be missed with contrast studies. Upper endoscopy is diagnostic in such cases and may also be therapeutic (eg, in the setting of an antral web⁹¹). Endoscopy is also essential for the diagnosis of mucosal inflammation causing pyloric obstruction, such as with eosinophilic gastropathy.⁹² Although the paucity of data precludes any specific recommendations regarding endoscopy in the management of GOO in children, it is advisable that children without a clear diagnosis despite radiologic investigation for GOO symptoms undergo a diagnostic endoscopy to exclude structural abnormality.

Motility disorders have also been reported in children. Delayed gastric emptying is most commonly reported to occur after viral infections, although it may also result from eosinophilic gastropathy.⁹³ Gastroparesis is not a significant feature in pediatric diabetic patients; however, idiopathic functional GOO has been described in children.⁹⁴ Endoscopy is indicated for children with evidence to suggest gastric emptying delay, gastroparesis, or functional GOO to examine for mucosal pathology. Although gastroduodenal motility has been used to guide therapy in children with GI motility abnormalities, this procedure is still considered investigational and is not widely available.⁹⁵ Most medical and surgical options described in adults for gastroparesis have also been used in children. For example, the management of idiopathic functional GOO in children has involved gastric outlet surgery, and pneumatic balloon dilation has also been described.^{96,97}

Recommendations

1. We recommend endoscopy for the evaluation of patients with suspected gastroduodenal obstruction. ⊕⊕⊕⊕
2. We recommend SEMS placement for the treatment of malignant gastroduodenal obstruction in those patients with poor performance status and/or short life expectancy. ⊕⊕⊕○ For other patients with malignant gastroduodenal obstruction, surgical gastrojejunostomy may offer a more durable result. The palliative approach chosen should depend on local expertise and the patient’s prognosis and preferences.
3. We recommend endoscopic biliary SEMS placement before enteral SEMS placement for malignant gastroduode-

- nal obstruction in the setting of established or impending biliary obstruction, when technically possible. ⊕⊕⊕○
4. We suggest palliative decompressive gastrostomy when malignant gastroduodenal obstruction is not amenable to surgical bypass or SEMS placement. ⊕⊕○○
 5. We suggest endoscopic balloon dilation for the management of benign GOO. ⊕⊕○○
 6. We recommend optimization of medical and dietary measures (eg, improved glycemic control) before endoscopic interventions for the management of gastroduodenal dysmotility. ⊕⊕⊕○
 7. We recommend enteral nutrition for severe and refractory gastroparesis because it is associated with fewer complications and lower cost compared with parenteral nutrition. ⊕⊕⊕○
 8. There are insufficient data to make a recommendation regarding the role of botulinum toxin in the treatment of gastroparesis.
 9. We recommend endoscopy for the evaluation of infants and children with suspected gastroduodenal obstruction when radiologic studies are inconclusive or unrevealing or when endoscopic therapy is indicated. ⊕⊕○○

DISCLOSURE

Dr Harrison served as a consultant for Fujinon, Inc. Dr Decker served as a consultant for Facet Biotechnology. Dr Fanelli received honoraria from Ethicon, served as a consultant for RII Biologics, and is the owner/governor of New Wave Surgical Corp. Dr Jain served as a researcher for BARRX Medical, Inc. No other financial relationships relevant to this publication were disclosed.

Abbreviation: GOO, gastric outlet obstruction; PEG, percutaneous endoscopic gastrostomy; PRG, percutaneous radiologic gastrostomy; SEMS, self-expandable metal stent; TPN, total parenteral nutrition.

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