Communication from the ASGE Technology Committee

TECHNOLOGY STATUS EVALUATION REPORT

ASGE American Society for Gastrointestinal Endoscopy

Techniques and devices for the endoscopic treatment of gastroparesis (with video)

Mansour A. Parsi, MD, MPH, FASGE,[1] Pichamol Jirapinyo, MD, MPH,[2] Barham K. Abu Dayyeh, MD, MPH, FASGE,[3] Manoop S. Bhutani, MD, FASGE,[4] Vinay Chandrasekhara, MD,[3] Kumar Krishnan, MD,[5] Nikhil A. Kumta, MD, MS,[6] Joshua Melson, MD, MPH, FASGE,[7] Rahul Pannala, MD, MPH, FASGE,[8] Guru Trikudanathan, MBBS,[9] Arvind J. Trindade, MD,[10] Amrita Sethi, MD, FASGE,[11] Rabindra R. Watson, MD,[12] John T. Maple, DO, FASGE, previous Committee Chair (2016-2019),[13] David R. Lichtenstein, MD, FASGE, ASGE Technology Committee Chair[14]

Background and Aims: Gastroparesis is a symptomatic chronic disorder of the stomach characterized by delayed gastric emptying in the absence of mechanical obstruction. Several endoscopic treatment modalities have been described that aim to improve gastric emptying and/or symptoms associated with gastroparesis refractory to dietary and pharmacologic management.

Methods: In this report we review devices and techniques for endoscopic treatment of gastroparesis, the evidence regarding their efficacy and safety, and the financial considerations for their use.

Results: Endoscopic modalities for treatment of gastroparesis can be broadly categorized into pyloric, nonpyloric, and nutritional therapies. Pyloric therapies such as botulinum toxin injection, stent placement, pyloroplasty, and pyloromyotomy specifically focus on pylorospasm as a therapeutic target. These interventions aim to reduce the pressure gradient across the pyloric sphincter, with a resultant improvement in gastric emptying. Nonpyloric therapies, such as venting gastrostomy and gastric electrical stimulation, are intended to improve symptoms. Nutritional therapies, such as feeding tube placement, aim to provide nutritional support.

Conclusions: Several endoscopic interventions have shown utility in improving the quality of life and symptoms of select patients with refractory gastroparesis. Methods to identify which patients are best suited for a specific treatment are not well established. Endoscopic pyloromyotomy is a relatively recent development that may prove to be the preferred pyloric-directed intervention, although additional and longer-term outcomes are needed. (Gastrointest Endosc 2020;92:483-91.)

The American Society for Gastrointestinal Endoscopy (ASGE) Technology Committee provides reviews of existing, new, or emerging endoscopic technologies that have an effect on the practice of GI endoscopy. Evidence-based methods are used, with a MEDLINE literature search to identify pertinent clinical studies on the topic and a MAUDE (U.S. Food and Drug Administration Center for Devices and Radiological Health) database search to identify the reported adverse events of a given technology. Both are supplemented by accessing the "related articles" feature of PubMed and by scrutinizing pertinent references cited by the identified studies. Controlled clinical trials are emphasized, but in many cases data from randomized controlled trials are lacking. In such cases, large case series, preliminary clinical studies, and expert opinions are used. Technical data are gathered from traditional and Web-based publications, proprietary publications, and informal communications with pertinent vendors. Technology Status Evaluation Reports are drafted by 1 or 2 members of the ASGE Technology Committee, reviewed and edited by the committee as a whole, and approved by the Governing Board of the ASGE. When financial guidance is indicated, the most recent coding data and list prices at the time of publication are provided. For this review the MEDLINE database was searched through August 2018 for articles related to endoscopic treatment of gastroparesis by using keywords

This video can be viewed directly from the GIE website or by using the QR code and your mobile device. Download a free QR code scanner by searching "QR Scanner" in your mobile device's app store.

such as "endoscopy" or "endoscopic" combined with "gastroparesis" and "stomach motility," among others. Technology Status Evaluation Reports are scientific reviews provided solely for educational and informational purposes. Technology Status Evaluation Reports are not rules and should not be construed as establishing a legal standard of care or as encouraging, advocating, requiring, or discouraging any particular treatment or payment for such treatment.

Gastroparesis is a symptomatic chronic disorder of the stomach characterized by delayed gastric emptying in the absence of mechanical obstruction.[1,2] A population-based study of Olmsted County, Minnesota suggested an estimated prevalence of 9.6 and 37.8 per 100,000 in men and women, respectively.[3] Although multiple conditions have been associated with gastroparesis, the most common causes are idiopathic, diabetic, and postsurgical.[2,4] Patients with gastroparesis may report a combination of symptoms including nausea, vomiting, early satiety, belching, bloating, or upper abdominal discomfort.

Management of symptomatic gastroparesis is challenging for both patients and clinicians. A thoughtful approach is required that ensures a proper diagnosis, considers the etiology and associated medical conditions, and uses a comprehensive treatment strategy, including dietary and pharmacologic interventions.[4] Several endoscopic treatment modalities have been described that aim to improve gastric emptying and/or symptoms associated with gastroparesis in patients refractory to medical management. These endoscopic techniques and related devices are reviewed in this technology report.

MECHANISM OF ACTION

Current understanding of the underlying pathophysiologic mechanisms for gastroparesis is incomplete and continues to evolve. However, it is generally acknowledged that a diverse set of etiologies leads to a common clinical presentation.[5] Loss of interstitial cells of Cajal, neuropathy, and myopathy are believed to play a central role in the pathophysiology of gastroparesis.[6] These structural abnormalities may be associated with varied states of compromised function, including impaired gastric accommodation, antral hypomotility, pylorospasm, duodenal dysmotility, autonomic dysfunction, and/or visceral hypersensitivity.[7]

Endoscopic treatment modalities for gastroparesis can be broadly categorized into pyloric, nonpyloric, and nutritional therapies.[6] Pyloric therapies such as botulinum toxin injection, stent placement, pyloroplasty, and pyloromyotomy specifically focus on pylorospasm as a therapeutic target. These interventions aim to reduce the pressure gradient across the pyloric sphincter, with a resultant improvement in gastric emptying. Nonpyloric therapies such as venting gastrostomy and gastric electrical stimulation (GES) are intended to improve symptoms. Nutritional therapies, such as feeding tube placement, aim to provide nutritional support.

PYLORIC THERAPIES

Identification of patients who are best suited for pyloric therapies is difficult. Manometry of the pylorus is invasive, requires expertise for performance and interpretation, and lacks standardized control data.[6,8,9] Impedance planimetry with the use of an endoscopic functional luminal imaging probe (EndoFLIP; Crospon Inc, Galway, Ireland) is a technology that has been used to measure tissue distensibility. Its utility in detection of patients with pylorospasm remains poorly studied.[5] Currently there is no consensus to standardize patient selection for various forms of pyloric therapy.

Intrapyloric botulinum toxin injection

Botulinum toxin A (BTA) is a protein produced by the bacterium *Clostridium botulinum*.[10] When injected into striated or smooth muscle, the toxin blocks neuromuscular conduction, leading to temporary paralysis of the affected muscle.[11,12] This effect has been exploited clinically to treat various disorders including gastroparesis.[13] In the United States BTA is available in powder form in 50- or 100-U vials.[14] The powder is usually diluted in 5 mL normal saline solution, yielding a solution containing 10 or 20 U/mL. Before intrapyloric botulinum toxin injection, a diagnostic upper endoscopy is performed to exclude structural abnormalities that could cause delayed gastric emptying.[15] Aliquots of 1 to 2 mL BTA solution (10-40 U) are injected into each of the 4 quadrants of the pylorus using a 23- or 25-gauge injection needle.[14] Total doses of 80 to 200 U have been reported.[14,15] Patients are discharged home after routine postsedation criteria are met and can eat a light meal later on the same day.[14]

Initial open-label studies of intrapyloric BTA injections in patients with gastroparesis reported symptomatic improvement in 90% to 100% of patients lasting several months.[11,15-17] However, 2 randomized controlled trials (RCTs) with 23 and 32 patients, respectively, did not find any differences in symptoms or gastric emptying time after intrapyloric injections of BTA compared with placebo.[18,19] Weaknesses of these RCTs include the small size and heterogeneity of the patient populations.[6] A 2013 clinical guideline from the American College of Gastroenterology did not recommend intrapyloric BTA injection as a treatment for gastroparesis.[4] However, some nonrandomized, open-label studies have suggested a role for intrapyloric BTA injection in selected patient groups with gastroparesis.[20,21]

Transpyloric stent placement

Covered self-expandable metallic stents placed across the pylorus have been used for treatment of refractory gastroparesis.[22,23] The stents used in published reports are marketed for use in the esophagus. During upper endoscopy a long guidewire is advanced distal to the second portion of the duodenum under endoscopic and/or fluoroscopic guidance. Reports have described the use of both standard and through-the-scope esophageal stent delivery systems. Stent deployment is performed under endoscopic visualization, with or without fluoroscopy, such that the proximal flared end is in the antrum and the distal flared end is in the duodenum proximal to the ampulla.[23] Anchoring of the stent to the wall of the antrum to potentially decrease the rate of migration has been reported using through-the-scope or over-the-scope clips or endoscopic suturing.[23]

Transpyloric placement of a covered self-expandable metallic stent was initially reported in a case series of 3 patients with refractory gastroparesis.[22] All patients had improvement in gastric emptying parameters and significant symptomatic benefit during a mean follow-up time of 4.5 months. A subsequent retrospective analysis from the same institution described 30 patients with refractory gastroparesis (16 idiopathic, 8 diabetic, 6 postsurgical) who were treated with transpyloric stent placement. The authors reported symptomatic improvement in 75% of the patients during a mean follow-up of 5 months.[23] However, stent migration was very frequent, occurring in 59% of all patients and in 48% of patients whose stents were anchored to the stomach wall with endoscopic suturing.[23] Given the high frequency of stent migration, the optimal stent dwell time and whether benefits persist after stent migration or removal are unknown.

Pyloric balloon dilation

Pyloric dilation using a 20-mm through-the-scope balloon under endoscopic visualization has been described as a treatment for gastroparesis in a single prospective series of 10 refractory gastroparesis patients with low fasting pyloric compliance as measured with impedance planimetry.[24] In this trial, the transpyloric balloon was inflated to 20 mm for a duration of 1 minute, with 2 subsequent balloon inflations in the same endoscopic session for a total of 3 dilations. The dilations resulted in an increase in pyloric compliance when measured 10 days later and improvement in symptoms and quality of life.[24]

Peroral pyloromyotomy

Peroral pyloromyotomy (POP), also known as gastric peroral endoscopic myotomy (POEM), uses a technique similar to esophageal POEM with minor modifications for use in the gastric antrum.[6] The distal end of a gastroscope is fitted with a transparent cap to facilitate submucosal access and tunnel creation. A routine diagnostic upper endoscopy using carbon dioxide insufflation is performed, and any fluid and debris in the stomach is suctioned. Subsequently, the antral mucosa is injected with a lifting agent at a site 3 to 7 cm proximal to the pylorus, typically along the lesser or greater curve. When an endoscopic submucosal dissection knife is used, a 1.5- to 4-cm transverse or longitudinal incision is made into the lifted mucosa. Once the mucosal incision is complete, the cap-fitted endoscope is advanced into the submucosal space.[25] Submucosal tunneling is performed using repeated injections and dissection using an endoscopic submucosal dissection knife. The tunnel is extended just beyond the pyloric muscle. Once exposed, the inner circular fibers of the pyloric muscle are divided, and subsequently the myotomy is extended a short distance (1-2 cm) proximally into the gastric antrum. After the myotomy is completed and hemostasis is ensured, the endoscope is removed from the submucosal tunnel and the mucosotomy site is closed with endoscopic clips or suturing.[25] A demonstration of POP technique is shown in Video 1 (available online at www.giejournal.org). Devices used for POP are similar to those used for esophageal POEM and have been described in detail in an American Society for Gastrointestinal Endoscopy technology report.[26]

After POP, most reports describe hospital admission for overnight observation.[25] Patients are typically maintained nil per os until a water-soluble contrast upper GI series is obtained and excludes a leak. A clear liquid diet is then initiated and advanced toward a low residue diet as tolerated by the patient. Proton pump inhibitor therapy is frequently used to assist in healing of the mucosotomy. Some centers have used intraprocedural and postprocedural (eg, 3-5 days) broad-spectrum antibiotics.[27]

A single-center prospective study evaluated 100 consecutive patients with refractory gastroparesis of various etiologies (56 idiopathic, 21 diabetic, 19 postsurgical, and 4 other) who were treated with POP.[28] Most patients (67%) had undergone prior endoscopic or surgical interventions for gastroparesis. The mean Gastroparesis Cardinal Symptom Index at 90 days postprocedure was 2.4 $\pm$ 1.2, as compared with a mean Gastroparesis Cardinal Symptom Index of 3.8 $\pm$ 0.9 before POP (absolute difference, 1.4; $P < .001$). In 53 patients who underwent both a pre-POP and 90-days post-POP 4-hour solid-phase gastric emptying study, the mean percentage of retention at 4 hours improved from 39.9% to 16.3% ($P < .001$).[28] In another prospective study, 20 patients with refractory gastroparesis (10 diabetic and 10 nondiabetic) underwent POP.[29] Compared with baseline values, POP significantly improved symptoms, quality of life, and gastric emptying at 3 months.[29] Other studies have reported similar results with improvement in symptoms and a reduction in the number of emergency department visits and hospitalizations among gastroparesis patients who underwent POP.[27,30,31] Clinical studies evaluating POP that have been published to date are summarized in Table 1.

A retrospective study of 60 matched patients compared the outcomes of POP (n = 30) with laparoscopic

TABLE 1. Characteristics of peroral pyloromyotomy studies

Reference	Study design	Country	No. of patients	Age (y)	Technical Success (%)	Procedural time (min)	Follow-up time (mo)	Clinical response (%)	Preprocedure GCSI score	Postprocedure GCSI score
Shlomovitz 2015[55]	Retrospective	USA	7	51 ± 11.7	100	90-120	6.5	86	—	—
Khashab 2017[27]	Retrospective	USA	30	47 ± 13	100	72 ± 42	6	85	—	—
Rodriguez 2017[56]	Retrospective	USA	47	43.7 ± 14.8	100	41.2 ± 28.5	3	—	4.6 ± .9	3.3 ± 1.4
Gonzalez 2017[53]	Retrospective	France	29	52.8 ± 17.7	100	47 ± 21.2	3 and 6	79 (3 mo) 69 (6 mo)	3.3 ± .9	1.0 ± 1.2 (3 mo) 1.1 ± .9 (6 mo)
Dacha 2017[57]	Retrospective	USA	16	44.8 ± 14.8	100	49.7 ± 22.1	1, 6, and 12	81	3.40 ± .50	1.48 ± .95 (1 mo) 1.36 ± .9 (6 mo) 1.46 ± 1.4 (12 mo)
Malik 2018[58]	Retrospective	USA	13	45.7 ± 10.3	100	119 ± 23	3	72.7%	2.1 ± 0.8	1.9 ± 1.0
Mekaroonkamol 2019[30]	Retrospective	USA	30	47.0 ± 15.7	100	48.3 ± 16.5	1, 6, 12, and 18	80 (1 mo) 71.4 (6 mo) 57.1 (12 mo) 66.7 (18 mo)	3.5 ± .6	1.8 ± 1.0 (1 mo) 1.9 ± 1.2 (6 mo) 2.6 ± 1.5 (12 mo) 2.1 ± 1.3 (18 mo)
Jacques 2018[29]	Prospective cohort	France	20	—	100	56.5 ± 13.7	1 and 3	90	3.5 ± 1.0	1.8 (1 mo) 1.3 (3 mo)
Rodriguez 2018[28]	Prospective cohort	USA	100	45 ± 14.6	100	33.8 ± 21.6	3	—	3.8 ± .86	2.4 ± 1.2
Xu 2018[59]	Retrospective	China	16	63.5 ± 15.8	100	45.25 ± 12.96	14.5	81.25	2.7 ± .6	.7 ± .1
Kahaleh 2018[31]	Retrospective	France USA	33	52 (21-85)	100	77.6 (37-255)	11.5	85	3.3	.8
Landreneau 2019[32]	Retrospective	USA	30	44.1 ± 13.5	100	33.9 ± 18.8	3	—	4.0 ± .8	2.4 ± 1.5

GCSI, Gastroparesis Cardinal Symptom Index; *mo*, months; —, not reported.

pyloroplasty (n = 30) for the treatment of refractory gastroparesis.[32] The authors reported equal efficacy with regard to improvement in the Gastroparesis Cardinal Symptom Index score at 30 and 90 days as well as in postprocedure scintigraphic gastric emptying. Length of hospital stay, operative time, estimated blood loss, and adverse events were increased in patients undergoing laparoscopic pyloroplasty.

NONPYLORIC THERAPIES

Venting PEG

PEG tubes have been used for gastric decompression ("venting") and symptom relief in patients with refractory gastroparesis.[4] Various techniques for PEG placement have been described in detail elsewhere.[33] Placement of a PEG with a concomitant jejunal extension tube allows gastric venting and jejunal feeding.

Data regarding the clinical benefits of venting PEG placement are limited to a single small series of 8 female patients with refractory idiopathic gastroparesis.[34] In all patients, 20F PEG tubes were placed endoscopically using a standard pull technique. Patients were instructed to relieve symptoms by aspirating gastric contents with a 60-mL syringe as needed. Over a mean follow-up of 29 months (range, 8-41), the authors observed significant improvements in patient symptom scores as well as weight gain (mean of 4.5 kg at 1 year) and successful cessation of prokinetic agents in all patients. Venting PEG tubes were removed in 3 patients during follow-up, of whom 1 remained symptom free and 2 experienced some return of gastroparesis symptoms.

Endoscopic GES

The mechanism of action for GES in gastroparesis remains poorly understood. An effect on gastric accommodation resulting in symptomatic benefit has been suggested.[35] Although currently nearly all GES devices are inserted surgically, endoscopic implantation of electrodes has been reported as a proof of concept.

In a prospective study, 20 consecutive patients with refractory gastroparesis underwent endoscopic placement of temporary electrodes either through a pre-existing PEG tube (n = 14) or the esophagus (n = 6).[36] The electrodes were connected to an external generator. Stimulation led to significant improvement in a composite symptom score with a mean time to improvement (>50% reduction in vomiting frequency) of approximately 2.5 days.[36] In a double-blinded RCT, 58 gastroparesis patients underwent endoscopic placement of temporary gastric electrodes and were randomized to stimulation or sham with crossover after 4 days.[37] Although overall treatment effects were not significant, differences in favor of stimulation were suggested in a per-protocol analysis. This trial was hindered by small sample size, frequent electrode dislodgement (22%), and a greater than expected placebo effect from electrode placement alone. A miniature GES with a generator placed endoscopically into the gastric submucosa has been developed but has not been studied in humans.[38]

ENDOSCOPIC NUTRITIONAL THERAPIES

Temporary endoscopic feeding tubes

Enteral nutritional support may become necessary in patients with severe gastroparesis who are unable to sustain adequate oral calorie intake.[4] Enteral nutrition is preferably delivered via postpyloric feeding, bypassing the dysfunctional stomach.[4] Nasoduodenal or nasojejunal feeding tubes are often placed before more invasive forms of access as a trial to ensure postpyloric feeding will be tolerated.[2,4] Various endoscopic techniques for placement of nasoduodenal/nasojejunal feeding tubes have been described.[39,40]

In an RCT, 73 patients in an intensive care unit with poor gastroduodenal motility as evidenced by large gastric residual volumes were randomized to receive enteral nutrition via an endoscopically placed nasojejunal tube (n = 34) or a nasogastric tube (n = 39).[41] Enteral nutrition delivered via a nasojejunal tube was associated with a significant reduction in gastric residual volumes and improved tolerance of enteral nutrition. A clinical guideline from the American College of Gastroenterology advocates nasojejunal rather than nasogastric feeding in patients with gastroparesis.[4]

Long-term endoscopic feeding tubes

Although used frequently for short-term feeding, nasoenteric tubes have disadvantages that limit their use for long-term management. In patients who tolerate postpyloric feeding via nasoenteric tubes, PEG with a jejunal extension tube (PEG-J) or direct percutaneous endoscopic jejunostomy (DPEJ) offers a long-term solution for postpyloric feeding. Although PEG-J tubes allow gastric venting in addition to jejunal feeding, the jejunal extension tube can migrate back into the stomach, and thus DPEJ tubes may be more reliable.[4,42]

DPEJ placement technique is similar to pull-type PEG placement. An enteroscope or a pediatric colonoscope is advanced beyond the ligament of Treitz. Endoscopic transillumination and finger indentation are used to select a proper site. The 21-gauge needle used for local anesthesia is advanced through the anterior abdominal wall at the site of maximal transillumination directly into the jejunum and is captured with a snare. This stabilizes the jejunal loop for subsequent advancement of the trocar alongside the smaller needle. Once trocar access is achieved, a standard pull-type gastrostomy tube is deployed.[43] Although similar to PEG placement, DPEJ placement is technically more challenging; a large cohort study of 307 consecutive attempts at DPEJ placement reported a technical success rate of 68%.[44,45] Use of balloon-assisted enteroscopy has been suggested to yield higher DPEJ placement success rates in 2 small case series of 10 and 11 patients.[46,47]

For patients with gastroparesis who are unable to maintain adequate nutrition with oral intake, placement of a feeding jejunostomy may decrease symptoms and reduce hospitalizations.[2,48] No studies compare the effectiveness of DPEJ to PEG-J for nutritional treatment of gastroparesis. However, PEG-J is associated with higher rate of tube dysfunction. In a retrospective study of 105 patients, DPEJ (n = 56) was associated with significantly lower rate of endoscopic reintervention and longer feeding tube patency compared with PEG-J (n = 49).[42] DPEJ is considered to be preferable if durable small-bowel feeding is required.[6,42,43]

EASE OF USE AND LIMITATIONS

Pyloric therapies

Pyloric BTA injection and balloon dilation use standard endoscopic techniques and do not require additional training. Transpyloric covered self-expandable metallic stent placement requires proficiency in endoluminal stent placement and knowledge of design, deployment characteristics, and potential risks associated with various types of stents. Performance of POP requires special training and equipment. No published studies assess the learning curve for performance of POP; however, the learning curve for technical proficiency with esophageal POEM has varied from 13 to 100 cases.[49-52]

Nonpyloric and nutritional therapies

Endoscopic pacing of the stomach is not widely available, and nearly all studies have been performed by a few expert investigators, typically in the setting of a

research protocol. Technically, endoscopic placement of nasoenteric feeding tubes is straightforward and may be performed with or without the use of fluoroscopy. Placement under fluoroscopic guidance allows immediate adjustment if the final position of the tube is not satisfactory and obviates the need for postprocedure radiologic confirmation of the tube position. Endoscopic placement of a PEG-J is technically easier than DPEJ and associated with higher rate of success.[45] However, a higher rate of tube dislodgement is associated with PEG-J.[42,43]

SAFETY

Pyloric therapies

BTA injection of the pylorus appears to be safe. No adverse events have been described with pyloric BTA injection, although the cumulative number of patients in the available literature is limited. In 2 small RCTs comparing intrapyloric injections of BTA with placebo, no differences occurred in adverse events between the study groups.[18,19] Data on safety of transpyloric stent placement and pyloric balloon dilatation are limited because of the number and size of available studies. Transpyloric stent placement is frequently associated with stent migration despite anchoring attempts; however, stent retrieval has been routinely possible in cases of proximal stent migration into the stomach, and no adverse events or need for intervention has been reported with distal stent migration. Reported adverse events associated with POP include tension capnoperitoneum, ulcer formation, bleeding, abscess formation, and perforation.[27,29,53] Asymptomatic capnoperitoneum is common after POP and typically requires no intervention.

Nonpyloric and nutritional therapies

In a study of 58 patients with gastroparesis who underwent endoscopic placement of orogastric temporary electrodes to facilitate GES, the only observed adverse event was electrode dislodgement, occurring in 13 patients (22%).[37] Possible adverse events associated with PEG and DPEJ tubes are peristomal infection, leakage, bleeding, buried bumper syndrome, peritonitis, tube blockage, tube displacement, and inadvertent removal.[54] Potential adverse events associated with nasoenteric tubes are blockage, dislodgment, sinusitis, gastroesophageal reflux, esophagitis, and esophageal stricture.[54]

FINANCIAL CONSIDERATIONS

Endoscopic devices for the treatment of gastroparesis and their costs are summarized in Table 2. Relevant current procedural technology codes for endoscopic BTA injection include 43236 (EGD with submucosal injection of any substance) with the Healthcare Common Procedure Coding System level II code J0585 (botulinum toxin injection). Code 43266 (EGD with enteral stent placement; including pre- and postdilation and guidewire passage) is used for pyloric stent placement, and codes 43245 or 43249 are used for EGD with balloon dilation.

Currently, there is no specific current procedural technology code for POP. Most centers report using 43235 (EGD), 43236 (EGD with submucosal injection of any substance), or 43999 (unlisted procedure, stomach). Any reimbursement, however, is variable and payer dependent. When reporting unlisted or "miscellaneous" codes, supporting documentation should be included with each claim. The information should detail the nature, extent, and need for the procedure and the time, effort, and equipment necessary to provide the procedure. Additional items to include are the complexity of symptoms, final diagnosis, pertinent patient findings, diagnostic and therapeutic procedures, concurrent problems, and follow-up care. It is helpful in such a cover letter to compare the work relative value units (RVU) or total RVU for the procedure to an existing code of similar intraservice time and intensity. Payers also often find it helpful to

TABLE 2. Commonly used devices for endoscopic treatment of gastroparesis

Device*	Price†
Pyloric therapies	
Intrapyloric botulinum toxin injection	
Botulinum powder vial	500
Injection needle	30-40
Transpyloric stent placement	
Self-expandable metallic enteral stent	1600-2600
Pyloric balloon dilation	
Dilating balloon	140-180
Peroral pyloromyotomy	
Injection needle	30-40
Submucosal lifting agent	80
Various endoscopic knives	490-770
Hemostatic forceps	220
Distal attachment	20
Endoscopic clip	100-230
Endoscopic suturing system	850
Tissue helix	185
Suture cinch	65
Suture	50
Nonpyloric therapies	
PEG tube kit	80-140
Endoscopic nutritional therapies	
Nasojejunal tube	90
Jejunal tube	140

*Most listed devices are available from multiple manufacturers.
†Approximate cost per item in U.S. dollars.

review copies of paid invoices. For enteral feeding procedures, relevant current procedural technology codes include 43246 (EGD with PEG placement), 43752 (nasogastric or nasoduodenal tube placement) and 44372 (DPEJ) or 44373 (small-intestinal endoscopy with conversion of percutaneous gastrostomy tube to percutaneous jejunostomy tube).

AREAS FOR FUTURE RESEARCH

Further development and evaluation of diagnostic tools that identify pathophysiologic mechanisms in gastroparesis are needed. Studies to identify subgroups of patients that respond better to various treatment options would be useful. This would permit tailored treatment where possible. Further studies are needed to assess the durability and long-term safety of endoscopic treatments.

SUMMARY

Gastroparesis is a chronic, debilitating disorder, often with a poor response to dietary modifications and pharmacologic intervention. Various endoscopic interventions have shown utility in improving the quality of life and symptoms of select patients with refractory gastroparesis. The best timing and selection of endoscopic interventions in the overall care plan for these patients is not well established. POP is a relatively recent development that may prove to be the preferred pyloric-directed intervention, although additional and longer-term outcomes are needed.

ACKNOWLEDGMENT

Dr. Parsi's time to develop this manuscript was supported by The Louisiana Board of Regents Endowed Chair for Eminent Scholars and C. Thorpe Ray, MD endowed chair in Medicine.

REFERENCES

1. Parkman HP, Hasler WL, Fisher RS, et al. American Gastroenterological Association medical position statement: diagnosis and treatment of gastroparesis. Gastroenterology 2004;127:1589-91.
2. Parkman HP, Hasler WL, Fisher RS, et al. American Gastroenterological Association technical review on the diagnosis and treatment of gastroparesis. Gastroenterology 2004;127:1592-622.
3. Jung HK, Choung RS, Locke GR, et al. The incidence, prevalence, and outcomes of patients with gastroparesis in Olmsted County, Minnesota, from 1996 to 2006. Gastroenterology 2009;136:1225-33.
4. Camilleri M, Parkman HP, Shafi MA, et al. Clinical guideline: management of gastroparesis. Am J Gastroenterol 2013;108:18-37; quiz 38.
5. Ahuja NK, Clarke JO. Pyloric therapies for gastroparesis. Curr Treat Options Gastroenterol 2017;15:230-40.
6. Su A, Conklin JL, Sedarat A. Endoscopic therapies for gastroparesis. Curr Gastroenterol Rep 2018;20:25.
7. Nguyen LA, Snape WJ. Clinical presentation and pathophysiology of gastroparesis. Gastroenterol Clin North Am 2015;44:21-30.
8. Desipio J, Friedenberg FK, Korimilli A, et al. High-resolution solid-state manometry of the antropyloroduodenal region. Neurogastroenterol Motil 2007;19:188-95.
9. Waseem S, Moshiree B, Draganov PV. Gastroparesis: current diagnostic challenges and management considerations. World J Gastroenterol 2009;15:25-37.
10. Davletov B, Bajohrs M, Binz T. Beyond BOTOX: advantages and limitations of individual botulinum neurotoxins. Trends Neurosci 2005;28:446-52.
11. Wiesel PH, Schneider R, Dorta G, et al. Botulinum toxin for refractory postoperative pyloric spasm. Endoscopy 1997;29:132.
12. DeSantis ER, Huang S. Botulinum toxin type A for treatment of refractory gastroparesis. Am J Health Syst Pharm 2007;64:2237-40.
13. Jankovic J. Botulinum toxin in clinical practice. J Neurol Neurosurg Psychiatry 2004;75:951-7.
14. Ukleja A, Tandon K, Shah K, et al. Endoscopic Botox injections in therapy of refractory gastroparesis. World J Gastrointest Endosc 2015;7:790-8.
15. Miller LS, Szych GA, Kantor SB, et al. Treatment of idiopathic gastroparesis with injection of botulinum toxin into the pyloric sphincter muscle. Am J Gastroenterol 2002;97:1653-60.
16. Lacy BE, Zayat EN, Crowell MD, et al. Botulinum toxin for the treatment of gastroparesis: a preliminary report. Am J Gastroenterol 2002;97:1548-52.
17. Ezzeddine D, Jit R, Katz N, et al. Pyloric injection of botulinum toxin for treatment of diabetic gastroparesis. Gastrointest Endosc 2002;55:920-3.
18. Arts J, Holvoet L, Caenepeel P, et al. Clinical trial: a randomized-controlled crossover study of intrapyloric injection of botulinum toxin in gastroparesis. Aliment Pharmacol Ther 2007;26:1251-8.
19. Friedenberg FK, Palit A, Parkman HP, et al. Botulinum toxin A for the treatment of delayed gastric emptying. Am J Gastroenterol 2008;103:416-23.
20. Coleski R, Anderson MA, Hasler WL. Factors associated with symptom response to pyloric injection of botulinum toxin in a large series of gastroparesis patients. Dig Dis Sci 2009;54:2634-42.
21. Wellington J, Scott B, Kundu S, et al. Effect of endoscopic pyloric therapies for patients with nausea and vomiting and functional obstructive gastroparesis. Auton Neurosci 2017;202:56-61.
22. Clarke JO, Sharaiha RZ, Kord Valeshabad A, et al. Through-the-scope transpyloric stent placement improves symptoms and gastric emptying in patients with gastroparesis. Endoscopy 2013;45(Suppl 2 UCTN):E189-90.
23. Khashab MA, Besharati S, Ngamruengphong S, et al. Refractory gastroparesis can be successfully managed with endoscopic transpyloric stent placement and fixation (with video). Gastrointest Endosc 2015;82:1106-9.
24. Gourcerol G, Tissier F, Melchior C, et al. Impaired fasting pyloric compliance in gastroparesis and the therapeutic response to pyloric dilatation. Aliment Pharmacol Ther 2015;41:360-7.
25. Allemang MT, Strong AT, Haskins IN, et al. How I do it: per-oral pyloromyotomy (POP). J Gastrointest Surg 2017;21:1963-8.
26. Pannala R, Abu Dayyeh BK, Aslanian HR, et al. Per-oral endoscopic myotomy (with video). Gastrointest Endosc 2016;83:1051-60.
27. Khashab MA, Ngamruengphong S, Carr-Locke D, et al. Gastric per-oral endoscopic myotomy for refractory gastroparesis: results from the first multicenter study on endoscopic pyloromyotomy (with video). Gastrointest Endosc 2017;85:123-8.
28. Rodriguez J, Strong AT Haskins IN, et al. Per-oral pyloromyotomy (POP) for medically refractory gastroparesis: short term results from the first 100 patients at a high volume center. Ann Surg 2018;268:421-30.
29. Jacques J, Pagnon L, Hure F, et al. Peroral endoscopic pyloromyotomy is efficacious and safe for refractory gastroparesis: prospective trial with assessment of pyloric function [abstract]. Endoscopy 2018;87(Suppl):AB50.

30. Mekaroonkamol P, Dacha S, Wang L, et al. Gastric peroral endoscopic pyloromyotomy reduces symptoms, increases quality of life, and reduces health care use for patients with gastroparesis. Clin Gastroenterol Hepatol 2019;17:82-9.
31. Kahaleh M, Gonzalez JM, Xu MM, et al. Gastric peroral endoscopic myotomy for the treatment of refractory gastroparesis: a multicenter international experience. Endoscopy 2018;50:1053-8.
32. Landreneau JP, Strong AT, El-Hayek K, et al. Laparoscopic pyloroplasty versus endoscopic per-oral pyloromyotomy for the treatment of gastroparesis. Surg Endosc 2019;33:773-81.
33. Kwon RS, Banerjee S, Desilets D, et al. Enteral nutrition access devices. Gastrointest Endosc 2010;72:236-48.
34. Kim CH, Nelson DK. Venting percutaneous gastrostomy in the treatment of refractory idiopathic gastroparesis. Gastrointest Endosc 1998;47:67-70.
35. Soffer EE. Gastric electrical stimulation for gastroparesis. J Neurogastroenterol Motil 2012;18:131-7.
36. Ayinala S, Batista O, Goyal A, et al. Temporary gastric electrical stimulation with orally or PEG-placed electrodes in patients with drug refractory gastroparesis. Gastrointest Endosc 2005;61:455-61.
37. Abell TL, Johnson WD, Kedar A, et al. A double-masked, randomized, placebo-controlled trial of temporary endoscopic mucosal gastric electrical stimulation for gastroparesis. Gastrointest Endosc 2011;74:496-503.
38. Hajer J, Novák M. Development of an autonomous endoscopically implantable submucosal microdevice capable of neurostimulation in the gastrointestinal tract. Gastroenterol Res Pract 2017;2017:8098067.
39. Wiggins TF, DeLegge MH. Evaluation of a new technique for endoscopic nasojejunal feeding-tube placement. Gastrointest Endosc 2006;63:590-5.
40. Qin H, Lu XY, Zhao Q, et al. Evaluation of a new method for placing nasojejunal feeding tubes. World J Gastroenterol 2012;18:5295-9.
41. Davies AR, Froomes PR, French CJ, et al. Randomized comparison of nasojejunal and nasogastric feeding in critically ill patients. Crit Care Med 2002;30:586-90.
42. Fan AC, Baron TH, Rumalla A, et al. Comparison of direct percutaneous endoscopic jejunostomy and PEG with jejunal extension. Gastrointest Endosc 2002;56:890-4.
43. DeLegge MH. Small bowel endoscopic enteral access. Gastrointest Endosc Clin North Am 2007;17:663-86.
44. Delegge MH. Endoscopic options for enteral feeding. Gastroenterol Hepatol 2007;3:690-2.
45. Maple JT, Petersen BT, Baron TH, et al. Direct percutaneous endoscopic jejunostomy: outcomes in 307 consecutive attempts. Am J Gastroenterol 2005;100:2681-8.
46. Despott EJ, Gabe S, Tripoli E, et al. Enteral access by double balloon enteroscopy: an alternative method of direct percutaneous endoscopic jejunostomy placement. Dig Dis Sci 2011;56:494-8.
47. Aktas H, Mensink PB, Kuipers EJ, et al. Single-balloon enteroscopy-assisted direct percutaneous endoscopic jejunostomy. Endoscopy 2012;44:210-2.
48. Fontana RJ, Barnett JL. Jejunostomy tube placement in refractory diabetic gastroparesis: a retrospective review. Am J Gastroenterol 1996;91:2174-8.
49. El Zein M, Kumbhari V, Ngamruengphong S, et al. Learning curve for peroral endoscopic myotomy. Endosc Int Open 2016;4:E577-82.
50. Liu Z, Zhang X, Zhang W, et al. Comprehensive evaluation of the learning curve for peroral endoscopic myotomy. Clin Gastroenterol Hepatol 2018;16:1420.
51. Lv H, Zhao N, Zheng Z, et al. Analysis of the learning curve for peroral endoscopic myotomy for esophageal achalasia: Single-center, two-operator experience. Dig Endosc 2017;29:299-306.
52. Patel KS, Calixte R, Modayil RJ, et al. The light at the end of the tunnel: a single-operator learning curve analysis for per oral endoscopic myotomy. Gastrointest Endosc 2015;81:1181-7.
53. Gonzalez JM, Benezech A, Vitton V, et al. G-POEM with antropyloromyotomy for the treatment of refractory gastroparesis: midterm follow-up and factors predicting outcome. Aliment Pharmacol Ther 2017;46:364-70.
54. Pearce CB, Duncan HD. Enteral feeding. Nasogastric, nasojejunal, percutaneous endoscopic gastrostomy, or jejunostomy: its indications and limitations. Postgrad Med J 2002;78:198-204.
55. Shlomovitz E, Pescarus R, Cassera MA, et al. Early human experience with per-oral endoscopic pyloromyotomy (POP). Surg Endosc 2015;29:543-51.
56. Rodriguez JH, Haskins IN, Strong AT, et al. Per oral endoscopic pyloromyotomy for refractory gastroparesis: initial results from a single institution. Surg Endosc 2017;31:5381-8.
57. Dacha S, Mekaroonkamol P, Li L, et al. Outcomes and quality-of-life assessment after gastric per-oral endoscopic pyloromyotomy (with video). Gastrointest Endosc 2017;86:282-9.
58. Malik Z, Kataria R, Modayil R, et al. Gastric per oral endoscopic myotomy (G-POEM) for the treatment of refractory gastroparesis: early experience. Dig Dis Sci 2018;63:2405-12.
59. Xu J, Chen T, Elkholy S, et al. Gastric peroral endoscopic myotomy (G-POEM) as a treatment for refractory gastroparesis: long-term outcomes. Can J Gastroenterol Hepatol 2018;2018:6409698.

Abbreviations: BTA, botulinum toxin A; DPEJ, direct percutaneous endoscopic jejunostomy; GES, gastric electrical stimulation; POEM, peroral endoscopic myotomy; POP, peroral pyloromyotomy; RCT, randomized controlled trial.

DISCLOSURE: *The following authors disclosed financial relationships: P. Jirapinyo: Consultant for GI Dynamics and Endogastric Solutions; research support from GI Dynamics, Apollo Endosurgery, and Fractyl. B. Abu Dayyeh: Consultant for Metamodix, BFKW, DyaMx, and Boston Scientific; research support from Apollo Endosurgery, USGI, Spatz Medical, GI Dynamics, Cairn Diagnostics, Aspire Bariatrics, and Medtronic; speaker for Johnson and Johnson, Endogastric Solutions, and Olympus. M. Bhutani: Research support from Silenseed Inc, Galera Inc, Oncosil Inc, and Augmenix Inc; food, beverage, or travel compensation from Boston Scientific, Augmentix, and Conmed Corporation. V. Chandrasekhara: Advisory board for Interpace Diagnostics; shareholder in Nevakar, Inc. K. Krishnan: Consultant for Olympus Medical. N. Kumta: Consultant for Boston Scientific, Olympus Corporation of the Americas, Gyrus ACMI, Inc, and Apollo Endosurgery US Inc. D. Lichtenstein: Consultant for Allergan Inc, Augmenix, Gyrus ACMI, Inc, and Olympus Corporation of the Americas; speaker for Aries Pharmaceutical, Gyrus ACMI, Inc, and Olympus Corporation of the Americas; tuition compensation from Erbe USA Inc. J. Melson: Research support from Boston Scientific; stock options in Virgo Imaging. R. Pannala: Consultant for HCL Technologies; travel compensation from Boston Scientific; stockholder in AbbVie. G. Trikudanathan: Speaker and honorarium from Boston Scientific; advisory board for Abbvie. A. Trindade: Consultant for Olympus Corporation of the Americas and Pentax of America, Inc; food and beverage compensation from Boston Scientific; research support from NinePoint Medical, Inc. A. Sethi: Consultant for Olympus America, Boston Scientific, and Fujifilm. R. Watson: Consultant for Apollo Endosurgery, Boston Scientific, Medtronic, and Neptune Medical Inc; speaker for Apollo Endosurgery and Boston Scientific.*

0016-5107/$36.00
https://doi.org/10.1016/j.gie.2020.03.3857
Received March 30, 2020. Accepted March 30, 2020.

Current affiliations: Section for Gastroenterology and Hepatology, Tulane University Health Sciences Center, New Orleans, Louisiana, USA (1), Division of Gastroenterology, Hepatology and Endoscopy, Brigham and Women's Hospital, Boston, Massachusetts, USA (2), Department of Gastroenterology and Hepatology, Mayo Clinic, Rochester, Minnesota, USA (3),

Department of Gastroenterology Hepatology and Nutrition, Division of Internal Medicine, The University of Texas MD Anderson Cancer Center, Houston, Texas, USA (4), Division of Gastroenterology, Department of Internal Medicine, Harvard Medical School and Massachusetts General Hospital, Boston, Massachusetts, USA (5), Division of Gastroenterology, Mount Sinai Hospital, New York, New York, USA (6), Division of Digestive Diseases, Department of Internal Medicine, Rush University Medical Center, Chicago, Illinois, USA (7), Department of Gastroenterology and Hepatology, Mayo Clinic, Scottsdale, Arizona, USA (8), Department of Gastroenterology, Hepatology and Nutrition, University of Minnesota, Minneapolis, Minnesota, USA (9), Department of Gastroenterology, Zucker School of Medicine at Hofstra/Northwell, Long Island Jewish Medical Center, New Hyde Park, New York, USA (10), New York-Presbyterian Medical Center/Columbia University Medical Center, New York, New York, USA (11), Department of Gastroenterology, Interventional Endoscopy Services, California Pacific Medical Center, San Francisco, California, USA (12), Division of Digestive Diseases and Nutrition, University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma, USA (13), Division of Gastroenterology, Boston Medical Center, Boston University School of Medicine, Boston, Massachusetts, USA (14).