Gastric Motility
Physiology and
Surgical Intervention

Jack W. Rostas III, MD, Tam T. Mai, MD, William O. Richards, MD*

The stomach plays a critical role in digestion, as a site of significant processing of meals and distribution of chyme to the small intestine. Gastric motility requires extensive integration of neural and hormonal regulatory input, rendering proper function vulnerable to a host of pathologic processes. Disordered gastric function can manifest as a spectrum of symptoms, ranging from inconvenient to completely debilitating and potentially life threatening. Whereas symptomatic gastric dysmotility is managed non-operatively in the majority of cases, surgical intervention is required for patients with severe symptoms refractory to medical therapy. Therefore, the foregut surgeon must be thoroughly familiar with the current diagnostic and management techniques available for deranged gastric motility.

NORMAL GASTRIC MOTILITY

Background

The traditional anatomic structures of the stomach are the fundus, corpus (body), antrum, and pylorus. These anatomically distinct regions do not correlate with the functional regions of the stomach.1 In general, the proximal stomach serves as a temporary reservoir for meals, while the distal stomach churns and mixes food with digestive juices. Once the distal stomach has processed the solid food to an appropriate size and consistency, the pylorus regulates its outflow into the duodenum. The proximal reservoir consists of the fundus and proximal one-third of the corpus, the distal pump consists of the distal two-thirds of the corpus and antrum, and the pyloric sphincter comprises the final gate to the small bowel2 (Fig. 1).

Gastric smooth muscle activity is modulated by myogenic, neural, and hormonal influences. Intrinsic myogenic contraction forms the fundamental basis of gastric

KEYWORDS

- Gastric motility
- Physiology
- Delayed gastric emptying
- Gastroparesis
- Dumping syndrome
- Gastric electrical stimulation

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motility, and occurs in the absence of any other influence. Neural regulation emanates primarily from the intrinsic gastric myenteric plexus, with further contributions from extrinsic parasympathetic (vagal) and sympathetic (splanchnic) input. Hormonal influences play a significant role in the regulation of gastric motility. The list of hormones known to modulate gastric motility is extensive.

Gastric peristalsis occurs primarily in the distal stomach and is regulated by the gastric slow wave, a 3-cycle-per-minute depolarization of the smooth muscle cell membrane. The gastric slow waves are paced by the interstitial cells of Cajal (ICC), specialized cells located primarily along the mid-portion of the greater curvature of the stomach. The ICC provide the coordination and propagation of electrical activity within the gastric smooth muscle cells. The propagation of the slow wave is slightly faster in the greater curvature as compared with the lesser curvature, such that the signals synchronize on reaching the pylorus.

Fasting Gastric Motility

Fasting gastric motility comprises the migrating motor complex (MMC), which serves to clear indigested debris from the lumen of the stomach and intestine. During this period the proximal stomach undergoes tonic contraction, while the gastric slow wave modulates the coordinated peristalsis of the distal stomach. The MMC consists of a 90- to 120-minute cycle with 4 distinct phases. Phase I comprises a 40- to 60-minute period of inactivity. Phase II is heralded by the progressive but irregular increase in the magnitude of the peristaltic wave over a period of 30 to 50 minutes. Phase III consists of high-amplitude, regular contractions at 3 cycles per minute over a 5- to 10-minute period, which performs the task of clearing luminal contents. The pylorus is open for the duration of this phase to allow emptying. Phase IV marks the rapid return to baseline from the contractions during phase III.

Postprandial Gastric Motility

Five to 10 minutes after the ingestion of food the MMC gives way to the fed state of gastric muscle activity. The proximal stomach stretches to accommodate the contents of a meal and allow mixing of gastric contents with pepsin and hydrochloric acid to initiate digestion. Relaxation of the proximal gastric smooth muscle occurs in response to swallowing, a reflex termed “receptive relaxation.” Similarly, expansion of the proximal stomach occurs in response to increases in gastric volume, a process referred to as “gastric accommodation.” These processes occur via stimulatory vagal input, as well as intrinsic and vasovagal reflexes in response to stretch. The overall
### Table 1
Hormonal influences of gastric motility

<table>
<thead>
<tr>
<th>Stimulatory Hormone</th>
<th>Stimulation</th>
<th>Site of Secretion</th>
<th>Other Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrin</td>
<td>Gastric pH &gt;3, vagus nerve, antral distention, protein, calcium, alcohol</td>
<td>Gastric antrum and duodenum (G cells)</td>
<td>Inhibits small bowel and colonic motility</td>
</tr>
<tr>
<td>Ghrelin</td>
<td>Fasting</td>
<td>Gastric fundus</td>
<td>Stimulates hunger</td>
</tr>
<tr>
<td>Motilin</td>
<td>Acid and vagus nerve</td>
<td>Stomach and duodenum (M cells)</td>
<td>Induces phase 3 MMC duodenal peristalsis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Inhibitory Hormone</th>
<th>Stimulation</th>
<th>Site of Secretion</th>
<th>Other Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholecystokinin</td>
<td>Increased fat and protein in small bowel</td>
<td>Duodenum and jejunum</td>
<td>Gastric relaxation, sensation of fullness</td>
</tr>
<tr>
<td>Glucagon</td>
<td>Hypoglycemia, amino acids, β-adrenergic stimulation</td>
<td>Pancreas (α cells)</td>
<td>Slows MMC</td>
</tr>
<tr>
<td>Glucagon-like peptide 1</td>
<td>Increased glucose, fatty acids, and amino acids</td>
<td>Distal small intestine and colon</td>
<td>Decreases appetite</td>
</tr>
<tr>
<td>Peptide YY</td>
<td>Increased glucose, fatty acids, and amino acids</td>
<td>Terminal ileum and colon</td>
<td>Decreases appetite</td>
</tr>
<tr>
<td>Secretin</td>
<td>Acid, lipid, or bile in duodenal lumen</td>
<td>Duodenum (D cells)</td>
<td>Acts indirectly via inhibiting gastrin release</td>
</tr>
<tr>
<td>Somatostatin</td>
<td>Acidification of duodenal lumen</td>
<td>Gastric antrum (D cells)</td>
<td>Induces fasting small intestinal activity</td>
</tr>
</tbody>
</table>

Data from Refs. 5–10
result is expansion of the proximal stomach to provide temporary storage for the contents of a meal, without an increase in intragastric pressure.\(^2\)

In the presence of food, the myenteric plexus releases hormonal signals to stimulate the gastric membrane potential to undergo an absolute increase in magnitude.\(^4\) On reaching threshold potential, an action potential results and contraction occurs in the distal stomach. Neurotransmitters from extrinsic neurons modulate the amplitude of action potentials in a dose-dependent manner. Acetylcholine, released from the vagus nerve, functions as an excitatory neurotransmitter, while the inhibitory neurotransmitters norepinephrine, nitric oxide, and vasoactive intestinal peptide are withheld from release from splanchnic neurons.\(^1\)

Peristalsis begins at the mid-stomach at the site of the gastric pacemaker and progresses along the body toward the pylorus, mobilizing and crushing food into a particulate consistency to facilitate its passage distally. Initially, contractions of irregular magnitude and frequency originate in the distal stomach. This pattern is similar to phase II of the MMC, with the exception that only about half of the gastric slow-wave potentials reach threshold for contraction. During each contraction, luminal contents lag behind the progression of the peristaltic wave, due to frictional forces against the gastric wall. Larger particles are forced retrograde to be exposed to these frictional forces repeatedly until adequately reduced in size. This effect is more pronounced with the more solid component of the chyme mixture.
The distal most portion of the stomach is the pylorus, a thick muscular ring that serves to regulate bidirectional passage of material between the antrum and duodenum. The peristaltic wave leads to a narrowing of the pylorus at the leading edge of the admixture, allowing only liquids and particulate matter 1 to 2 mm in size to funnel appropriately out of the stomach. The pylorus remains closed for most of the duration of the fed state, synchronized with the most intense antral contractions to facilitate churning of food. Opening occurs intermittently in conjunction with relatively minor antral contractions, to allow passage of processed gastric contents.

**Gastric Emptying**

Strict regulation of gastric motility ensures the appropriate delivery of gastric contents into the duodenum to allow for optimal absorption. This process requires passage of gastric contents at both the appropriate rate and composition. The regulation of gastric emptying begins with the accommodation of the proximal stomach. This expansion in response to a food bolus allows timely flow to the distal stomach for processing and distribution. Abnormally reduced compliance of the proximal stomach results in increased intragastric pressure and accelerated emptying. Normal transit allows gastric contents to pass from the proximal to the distal stomach for processing and, in conjunction with the pylorus, delivery to the small bowel.

The composition of the gastric contents affects the rate of gastric emptying. Liquid emptying occurs more rapidly than that of solids, and is completed first when both are present. However, the emptying time of liquids increases with the relative proportion of the solid component. Solid emptying initially occurs slowly to allow for mixing and processing of gastric contents, and increases progressively as smaller particles become available for emptying. Solid-phase gastric emptying, as measured by the technetium-99-labeled scrambled egg study, classically demonstrates a 10- to 20-minute lag phase corresponding to the grinding of food, which is followed by a linear emptying of the food into the duodenum. Gastric emptying of liquids does not show the lag phase, as liquids exit the pylorus via first-order kinetics, directly proportional to the volume present (Fig. 3). This exit translates into a normal gastric emptying time of approximately 120 minutes, or a $T_{1/2}$ of 60 to 90 minutes, after an average, mixed solid/liquid meal.

The gastric emptying rate is primarily governed by caloric content, to allow for optimal absorption in the small intestine. The rate of gastric emptying is tightly regulated to distribute 1 to 4 kilocalories per minute to the proximal small bowel. Consequently, fats empty slower than either carbohydrates or proteins. Cholecystokinin (CCK) plays a pivotal role in this process via inhibition of gastric emptying. CCK is released from the small intestine in the presence of intraluminal fat and protein. Other characteristics of the gastric contents that determine emptying rate include anxiety, fear, depression, and intense exercise. Decreased temperature of the luminal contents also delays emptying, whereas the converse is true for increased temperature. Hypertonic or hypotonic contents exit more slowly than isotonic solutions.

Similar to the stomach, the duodenum contains an intrinsically regulated pacing system. However, the duodenal slow wave frequency is regulated at 12 cycles per minute. Therefore, the gastric peristaltic wave approaches the gastroduodenal junction and synchronizes with only about one-fourth of the duodenal contractions. This frequent, independent peristalsis ensures clearance of the duodenal lumen for efficient reception of gastric contents.

The gastrointestinal (GI) tract distal to the stomach also has multiple processes in place to regulate the flow of gastric contents. The duodenum and colon partially regulate their own inflow when distended, via a reflex arc that results in decreased fundal
The duodenum also directly contracts to slow filling in response to stretch. The presence of high concentrations of glucose, lipids, or protein in the lumen of the ileum slows gastric emptying. This reflex, termed the “ileal brake,” is largely induced by the release of peptide YY from the ileum. A similar process occurs in the duodenum in response to lipid and protein. These components of GI regulation serve to further optimize the flow rate of nutrients into the distal bowel to maximize the absorptive capacity of the intestine.

DISORDERS OF GASTRIC MOTILITY

The complex mechanisms influencing gastric motility allow a wide number of pathologic processes to interfere with normal transit. Disorders of gastric motility fall under the Rome III consensus criteria of functional dyspepsia, in the subcategory of postprandial distress syndrome. These criteria represents a spectrum of dysfunction ranging from abnormally slow transit, termed delayed gastric emptying (DGE), to abnormally rapid gastric emptying, commonly referred to as “dumping syndrome.” Although divergent in origin, both rapid and delayed gastric emptying can produce remarkably similar symptoms. Varying degrees of nausea, vomiting, and abdominal pain can be the predominant symptoms in both extremes of gastric motility disorders, although the presence of diarrhea is more likely to occur in rapid emptying. To further complicate matters, both of these divergent disorders can result from diabetes or vagal nerve dysfunction. These similarities emphasize the importance of obtaining a precise history and physical examination during evaluation. Correctly distinguishing these two processes is critical in ensuring the proper application of what is often quite divergent therapy.

DGE is typified by diabetic gastroparesis, while rapid emptying is best exemplified by the classic postsurgical “dumping syndrome.” The presence of any related symptoms merits a complete investigation, to establish the proper diagnosis and its underlying cause. Fortunately, these disorders overwhelmingly stem from benign processes, such as diabetes and postsurgical alterations. However, the etiology must be firmly
established to ensure that a more concerning disease, such as a systemic autoimmune disorder or even occult malignancy, is not the culprit. Diagnosis of disordered gastric motility involves pursuing the appropriate imaging study based on clinical suspicion. Once the diagnosis of a motility disorder is established, initial management is exclusively nonoperative and is usually successful. Operative intervention is reserved for the most severe and medically refractory cases.

**Delayed Gastric Emptying**

DGE is defined as abnormally slow gastric transit in the absence of physical obstruction. The estimated prevalence of DGE is 4% of the population, 80% of which are women. The 3 most common causes, in descending order, are medications/drugs, diabetes, and postsurgical. Medications and other drugs that commonly cause DGE are listed in Box 1.

The second most common cause of DGE is diabetes related. DGE can be found in 20% to 50% of diabetic patients, and is usually associated with later stages of the disease. Although the pathophysiology of diabetic gastroparesis is not fully understood, there is certainly a multifactorial influence. Evidence suggests that both hyperglycemia and hyperinsulinemia suppress phase III of the MMC and result in an increase in pyloric contractility. Both mechanisms contribute to DGE, as well as reduced gastric clearance in the fasting state with potential bezoar formation.

Hyperglycemia has also been shown to result in a direct myopathy of the gastric antrum.

The third most common cause of DGE is postvagotomy gastroparesis. This disease entity encompasses vagotomy during an acid-reducing procedure, as well as inadvertent injury to the vagus or its gastric branches. Truncal vagotomy results in a 5% incidence of postoperative DGE, even when a concomitant drainage procedure is performed. When performed correctly, highly selective vagotomy should not induce

<table>
<thead>
<tr>
<th>Box 1</th>
<th>Medications and drugs that delay gastric emptying</th>
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<tr>
<td>Alcohol</td>
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<tr>
<td>Aluminum hydroxide antacids</td>
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<tr>
<td>Muscarinic cholinergic receptor antagonist</td>
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<tr>
<td>β-Agonists</td>
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<tr>
<td>Calcium channel blockers</td>
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<tr>
<td>Diphenhydramine</td>
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<td>Glucagon</td>
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<tr>
<td>Dopamine agonists</td>
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<tr>
<td>Lithium</td>
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<td>Ondansetron</td>
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<td>Opioid analgesics</td>
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<td>Phenothiazines</td>
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<tr>
<td>Tobacco/smoking</td>
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<tr>
<td>Tricyclic antidepressants</td>
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</table>

Data from Refs.11,21–23
DGE as the antral/pyloric innervation is left intact. Loss of vagal input eliminates a critical postprandial stimulus to the enteric nervous system, leading to reduced peristalsis of the distal stomach and resulting in DGE from a reduced ability to empty solids. Vagotomy may also lead to loss of phase II of the MMC in the fasting state. Fortunately, fewer than 1% of these patients experience persistent, disabling symptoms. Other causes of DGE are listed in Box 2. Of note, infections implicated in DGE include Helicobacter pylori, Epstein-Barr virus (EBV), and cytomegalovirus (CMV). Fortunately, DGE from an infectious source is often self-limiting.

**Diagnosis**

Patients with DGE often suffer from nausea, vomiting, bloating, early satiety, abdominal pain, and discomfort. These symptoms are made worse with the ingestion of meals proportionally higher in solid content. Weight fluctuations are another common complaint. Although these symptoms are nonspecific, abdominal pain, bloating, and fullness best correlate with DGE. Physical examination findings may include a rotund, tender, and possibly tympanic upper abdomen. Laboratory findings may show hypokalemia and a contraction alkalosis from poor intake and persistent vomiting.

The complex and often contradictory symptoms of DGE necessitate confirmatory imaging to establish a definitive diagnosis. The 4-hour radionuclide colloid scintigraphy gastric emptying study (GES) is the gold standard for diagnosis. This test can be performed using a radiolabeled liquid or solid meal, although the solid-based test is preferred because the liquid-based scan can be normal in advanced disease states. The solid-meal GES is usually administered as an isotope-labeled, low-fat, scrambled-egg meal. Greater than 60% retention at 2 hours or greater than 10% retention at 4 hours confirms the diagnosis of DGE.

Any suggestion of outlet obstruction should be initially ruled out with endoscopic evaluation. The presence of retained items in the stomach, especially phyto-bezoars,

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**Box 2**

**Causes of gastroparesis**

- Medications and other drugs (listed in Box 1)
- Diabetes
- Surgery-related, vagotomy, duodenectomy, postgastrectomy
- Metabolic (hyperglycemia, hypokalemia, hypermagnesemia)
- Hypopituitarism, hypoadrenalism, hypothyroidism
- Chronic renal failure
- Portal hypertension
- Intra-abdominal malignancy
- Infectious: *H pylori*, EBV, CMV
- Autoimmune: Systemic sclerosis, systemic lupus erythematosus
- Myotonic dystrophies
- Central nervous system disorders (Parkinson disease, multiple sclerosis)
- Peripheral nervous system disorders (amyloid neuropathy, Guillain-Barré, primary dysautonomia)
- Psychiatric disorders (anorexia nervosa, rumination syndrome)

*Data from Refs.* 5,20,23,26,27
indicates a high probability of DGE. A fluoroscopic GES can assess gastric emptying and evaluate potential outlet obstruction. Although not directly useful in the assessment of DGE, a barium upper GI series can be essential to rule out mechanical obstruction. On very rare occasions, diagnostic laparotomy may be required to correctly distinguish a partial distal small bowel obstruction from a small bowel pseudo-obstruction with a component of DGE.

Many other imaging modalities are available, each having inherent advantages and disadvantages. Real-time ultrasonography can be used to calculate gastric volumes after the ingestion of a liquid. Although this test is better for patients who should not be exposed to radiation, it is somewhat operator-dependent. Contrasted MRI can be used with sequential axial scanning, and has the advantage of measuring multiple parameters simultaneously. Although supine positioning is an obvious disadvantage, this test has been found to correlate well with the gold standard scintigraphic GES. Single-photon computed tomography can be obtained after intravenous administration of [99Tc]pertechnetate. This isotope accumulates in the gastric wall and provides a 3-dimensional image of the stomach to measure real-time gastric volumes.

Finally, patients can be given radiopaque markers to swallow. A follow-up abdominal radiograph obtained 6 hours after swallowing should demonstrate absence of markers from the stomach. Although this test is simple and inexpensive, it cannot be directly correlated to the emptying of digestible material. Other options for assessment include the satiety test, which measures the amount of a liquid ingested until the patient reports feeling full. Although this parameter is reduced in DGE, the applicability of this test tends to be very subjective. GI manometry uses an intraluminal catheter to measure gastric pressures in real time. A 4- to 5-hour fasting period is initially assessed, followed by a 2-hour postprandial period. Manometry can help distinguish specific causes of DGE. For example, autonomic neuropathy would demonstrate normal fasting pattern due to inherent myogenic contraction, with the absence of conversion to the fed state. Conversely, DGE derived from a myopathy would demonstrate abnormal contractions of the gastric musculature.

Multiple other studies may have application in the assessment of DGE, but have not undergone full clinical validation. The barostat system uses a balloon in the stomach to measure pressure and volume. This study has been shown to demonstrate gastric emptying effectively, but produces nonphysiologic gastric accommodation that limits its clinical usefulness. An indirect assessment of gastric emptying using breath testing shows early promise. A 13C-labeled substrate is ingested, which is metabolized in the intestine and eventually exhaled as 13CO2. The rate of 13CO2 exhalation is proportional to the gastric emptying rate. Another method uses a radiotelemetry capsule, which constantly transmits a pH measurement. Gastric emptying time is the period from ingestion of the capsule until a neutral pH is obtained, signifying passage into the proximal duodenum. This test has been shown to correlate with the findings of a simultaneously performed GES.

Noninvasive techniques for measuring gastric motility in early stages of development show much promise for accurate assessment of the underlying electrical activity of the stomach. Electrogastrography (EGG) measures gastric potentials transcutaneously to assess for disordered motility. Tachygastria is defined as greater than 4 cycles per minute, and bradygastria is the presence of less than 2 cycles per minute. While promising as a logical correlate to electrocardiography and the heart, cutaneous EGG cannot accurately predict the underlying abnormalities of gastric electrical activity and has been virtually abandoned by clinicians.
The inherent limitations of EGG have led to the development of magnetogastrography (MGG). MGG uses a superconducting magnetometer to measure surface current density of the magnetic fields overlying the abdominal wall generated by the electrical activity of the gastric smooth muscle. Because magnetic fields are not attenuated by the intervening tissues of the abdominal wall, a more accurate assessment of the underlying gastric electrical activity can be obtained. This information is translated to frequency, direction, amplitude, and velocity of the gastric slow wave. MGG shows great promise to accurately measure the underlying electromagnetic fields generated by the gastric smooth muscle. Studies using this technology suggest that differences in the underlying gastric electrical activity can be linked to DGE (Fig. 4).

**Initial management**

Initial management of DGE includes optimal medical therapy for any predisposing conditions (ie, glucose control in diabetics). Small, frequent meals are advocated to reduce symptoms. High-residue diets should also be avoided because of the risk of accumulation and bezoar formation. Empiric acid-reducing therapy can be administered, but often will only treat concomitant gastroesophageal reflux without altering the underlying motility disorder. Prokinetic and antiemetic medications are the mainstay of pharmacotherapy. Prokinetic agents include metoclopramide, erythromycin, and domperidone. Antiemetic agents include prochlorperazine, promethazine, and 5-hydroxytryptamine receptor agonist (ie, odansetron). Other options include endoscopic intrapyloric botulinum toxin injection. Though purported to alleviate DGE caused by aberrant contractions at the gastric outlet, results have been disappointing.

**Surgical management**

Operative management of DGE is reserved for severe and persistent symptoms refractory to medical management. Multiple options have been described, ranging from percutaneous gastrostomy to near total gastrectomy. Unfortunately, all traditional methods suffer from poor symptomatic relief and high recurrence rates, along with substantial morbidity associated with more drastic measures. A gastrostomy is an

![Fig. 4. Magnetogastrography (MGG). Illustration demonstrating a simulated surface current density recorded over the stomach by a superconducting magnetometer. (From Bradshaw L, Cheng L, Richards W, et al. Surface current density mapping for identification of gastric slow wave propagation. IEEE Trans Biomed Eng 2010. Available at: http://ieeexplore.ieee.org/lpdocs/epic03/wrapper.htm?arnumber=4895313; with permission.)](image)
appealing option secondary to its relative ease of placement and low morbidity. A gastrostomy tube can be placed percutaneously with endoscopic or fluoroscopic assistance, or surgically via open or laparoscopic methods. A jejunostomy can be placed using all of these same methods, or via a new or previously placed gastrostomy. Both function to provide enteral access for nutrition, which may be critical in cases of DGE resulting in severe malnutrition and weight loss. In these cases, enteral access is preferred over the multitude of complications arising from long-term intravenous nutrition. Furthermore, a gastrostomy or jejunostomy also provides a mechanism to periodically release pressure from the upper GI tract to treat episodes of symptomatic distention resulting from gastroparesis.

The ultimate intervention for refractory DGE is subtotal or complete gastrectomy. This procedure should only be considered for the management of the most severe cases of DGE with no other alternative treatment. While an extensive procedure in the best of circumstances, completion gastrectomy will often need to be performed in a poor surgical candidate with a history of systemic illness or multiple previous abdominal operations. Reconstruction with a Roux-en-Y gastrojejunostomy is usually preferred. The best long-term improvements have been seen in patients undergoing completion gastrectomy for symptoms related to previous partial gastrectomy or vagotomy.

**Gastric neurostimulation**

The overall poor results from conventional therapy, along with a greater understanding of gastric electrical physiology, have led to the development of gastric electrical stimulation devices. The first and only device to have received approval from the Food and Drug Administration (FDA) is the Enterra gastric neurostimulator (Medtronic, Minneapolis, MN). This device uses a low-energy, high-frequency (12 cycles per minute), and short-duration pulse to stimulate the gastric enteric nervous system. During laparotomy or laparoscopy, paired electrodes are placed approximately 1 to 2 cm apart near the native pacing zone located on the greater curvature of the stomach. These leads are tunneled to a pocket within the anterior abdominal wall containing the pulse generator. Upper endoscopy can be considered after placement rule out any violation of the gastric lumen during placement. Potential complications include pocket infection, erosion of the pulse generator, intestinal obstruction, and gastric lead breakage, dislodgment, or perforation.

In a subjective study via questionnaire, Enterra therapy is consistently found to improve symptoms of DGE in over half of the patients studied, with more improvement reported in those patients suffering from diabetic than from idiopathic gastroparesis. Interestingly, these studies have also demonstrated that gastric neurostimulation can result in improvement in nausea and vomiting more readily than abdominal pain. Indications for it use are also being expanded to post-operative DGE, with good preliminary results. Improvements of these devices focus on less invasive placement techniques, using the same basic mechanism. Methods currently in development include cutaneous or percutaneous endoscopically placed electrodes. A significant drawback of the Enterra neurostimulator is the lack of evidence to show that gastric emptying is altered in any way with this form of therapy.

A similar device, the Tantalus II (MetaCure, Kfar-Saba, Israel) system, is approved for use only in Europe, with current FDA approval for use within clinical trials. This device synchronizes to gastric slow waves and delivers electrical signals that serve to modulate gastric contractility. High-energy, low-frequency, and long-duration pulses stimulate the stomach at a rate slightly above the slow-wave rate of 3 cycles per minute. The device is placed in the same manner as the Enterra system, except
3 pairs of electrodes are placed in the gastric wall and are connected to the pulse generator. Lead pairs are placed at the fundus and the anterior and posterior antral wall, with each pair being placed 2 cm apart perpendicular to the long axis of the stomach. Potential complications are also similar to those of the Enterra device. Unlike the Enterra device, Tantalus therapy has been found to pace the gastric musculature and accelerate gastric emptying of solids. Of interest, the Tantalus system is also being explored as a treatment of type 2 diabetes mellitus via reduction in weight and blood glucose levels.

**Rapid Gastric Transit**

Rapid gastric transit (RGT) describes a spectrum of symptoms resulting from accelerated flow of gastric contents into the small bowel. The critical mechanism in this disorder is the transit of unprocessed or poorly processed hyperosmolar gastric contents into the proximal small bowel, not necessarily the speed of gastric emptying. RGT can result from any functional impairment of the pyloric sphincter allowing hyperosmolar contents to abruptly enter the small bowel. RGT is best described by “dumping syndrome,” the classically described postprandial symptoms following pyloroplasty or gastrectomy.

Dumping syndrome is categorized into two phases according to distinct symptoms. Early dumping occurs 15 to 30 minutes after eating when hyperosmolar luminal contents swiftly enter the proximal intestine; this leads to a sudden fluid shift into the intestine, resulting in nausea and vomiting, diarrhea, diaphoresis, hypotension, and possible syncope. Furthermore, the hyperosmolar luminal contents trigger serotonin release from the argentaffin cells of the small intestine. Serotonin release results in massive peripheral and mesenteric vasodilation, further contributing to the hypotension-inducing fluid shifts of early-phase dumping. Many other hormone levels are elevated in association with the dumping syndrome, including neurotensin, pancreatic polypeptide, enteroglucagon, peptide YY, insulin, glucagon, and glucagon-like peptide. Late dumping, although variably present, results from the swift uptake of glucose and other sugars from the small bowel. This resultant hyperglycemia stimulates a reactive increase in insulin, along with rebound hypoglycemia and hypokalemia. The hypoglycemia that occurs with dumping typically manifests 45 to 60 minutes after the meal.

**Etiology**

Consequences of gastrectomy were traditionally the leading causes of RGT and dumping syndrome. Partial gastrectomy has been shown to result in dumping syndrome in 15% to 20% of cases, and in 6% to 14% of patients after truncal vagotomy and pyloroplasty. Gastric resection with Roux-en-Y reconstruction results in some form of dumping symptoms in up to 70% of patients postoperatively, but most symptoms resolve with conservative intervention in the follow-up period. This ability to recover normal function depends on the regeneration of the phase III MMC from within the jejunal limb. Multiple procedures were devised to provide for the best functional results after gastrectomy. Pylorus-preserving gastrectomy has consistently been shown to provide the lowest incidence of dumping syndrome. When resection of the distal stomach is required, distal gastrectomy with Roux-en-Y reconstruction has been shown to result in a reduced incidence of dumping syndrome as compared with Billroth I reconstruction.

Anatomic alterations imposed by bariatric surgery constitute an increasing proportion of surgical causes of RGT. Symptomatic dumping syndrome was found in 0.3% of restrictive (decreased gastric capacity) procedures and 14.6% of combined restrictive
and malabsorptive (decreased intestinal absorptive length) procedures.53 Alternatively, vertical sleeve gastrectomy virtually eliminates any risk of postoperative dumping syndrome.54–56 Other sources of RGT include any source of autonomic dysfunction, such as large-fiber neuropathy, neuropathy related to diabetes mellitus, amyloidosis, or idiopathic neuropathy.19 Of interest, RGT occurs more often in early-stage diabetes than at any other time in the course of the disease.57 Zollinger-Ellison syndrome and peptic ulcer disease are rare causes of RGT.29

Other rare causes of RGT include increased antral peristalsis or reduced pyloric resistance.19 Furthermore, reduced gastric compliance can result in increased intragastric pressure and contributes to accelerated emptying. Causes of decreased compliance include gastric resection, vagotomy, post-fundoplication, or rumination syndrome.1,4 Interruption of vagal efferents can lead to chronic gastric atony, with loss of receptive relaxation with increased intragastric pressure in response to a food bolus. This process results in bloating and early satiety with rapid emptying of liquids.22 Tube feedings represent an iatrogenic source of RGT from reduced compliance, as the process of receptive relaxation initiated during swallowing is completely bypassed.

**Diagnosis**
Suspicion of RGT can be made on clinical grounds in patients demonstrating the aforementioned postprandial symptoms.4 Formerly a barium GES was used to help confirm the diagnosis. A modified oral glucose challenge can also be employed, in which a 50-gram solution of glucose is administered the morning after a fast. Early evidence of a significant fluid shift (increased pulse rate >10 beats/min or hematocrit >3%) or late hypoglycemia (<60 mg/dL) confirms the diagnosis.50 Alternatively, radionuclide colloid scintigraphy using an isotope-labeled 2% scrambled-egg meal can be used, with a greater than 50% transit of luminal contents at 1 hour establishing the diagnosis.29

**Initial management**
Initial management of RGT includes dietary modification, minimizing liquids and any particularly hyperosmolar intake. Patients are encouraged to avoid liquids with or 2 hours after a meal. Small, high-protein, low-carbohydrate meals are encouraged, spreading out intake over 6 meals a day.50 Lying down after meals can further help delay emptying time, as well as counteract any symptoms arising from hypovolemia.50 The α-glucosidase inhibitor acarbose can be used to slow carbohydrate digestion and absorption, helping to prevent late reactive hyperglycemia.4,50 Administration of the long-acting somatostatin analogue, octreotide acetate, has been shown to be highly effective in the treatment of medically refractory dumping syndrome. Octreotide functions by promoting the fasting state of intestinal motility along with inhibition of vasoactive hormone release.4,9,50 One of the authors (W.O.R.) has successfully treated a small number of patients with severe dumping for over 10 years with small doses (10–50 µg subcutaneously) of octreotide acetate before meals.9

**Surgical management**
Operative intervention may be necessary for the most severe and refractory cases of RGT. Results from these “rescue operations” are unpredictable and often poor.50 In addition, the morbidity of what is often a repeat operation must be considered. These facts emphasize attention to detail and patience during the initial trial of dietary modification and medical therapy, noting the success of the administration of octreotide acetate in severe cases.
The first surgical option is restoration of normal anatomy. Direct pyloric reconstruction may be of benefit for refractory RGT after vagotomy and pyroplasty. In the few patients who develop disabling dumping syndrome after Roux-en-Y gastric bypass, failure of dietary and medical therapy to correct severe reactive hypoglycemia necessitates surgical revision to restore the original anatomy. The use of laparoscopy during reversal has been found to be feasible and safe. Contraindications are previous vagotomy and pyloric obstruction.

A second surgical option includes the use of a reversed intestinal segment as a pyloric bypass. This method, although effective, suffers from a high rate of obstruction necessitating yet another operative intervention. The third option for surgical management of RGT is modification of the anastomosis created during a previous gastric resection. Although a Billroth II construction could be converted to a Billroth I to take advantage of a lower rate of dumping syndrome, results have not shown particular success. Roux-en-Y construction often leads to a delay in gastric emptying (Roux stasis syndrome), providing a versatile method for the treatment of RGE. As with any Roux-en-Y bypass, the significant risk of marginal ulceration merits a concomitant vagotomy and hemigastrectomy. A novel therapeutic approach involves the reduction of the diameter of the anastomotic channel to reduce gastric transit. Other methods currently in development and undergoing clinical validation include the endoscopic use of fibrin glue, suturing, and argon plasma coagulation.

SUMMARY

Disordered gastric motility is primarily managed with dietary modification followed by appropriate pharmacotherapy. Traditional surgical interventions for the most severe cases tended to be formidable, rarely definitive, and fraught with complications. The increase in the number of bariatric procedures and predisposing conditions such as diabetes will continue to produce a significant number of these refractory cases. Improvements in minimally invasive procedures will provide options for earlier and more effective intervention, as well as opportunities for management in those formerly deemed poor surgical candidates.

REFERENCES


