Gastric Motility Physiology and Surgical Intervention

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KEYWORDS

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

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 nonoperatively 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 bowel[2] (**Fig. 1**[3]).

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

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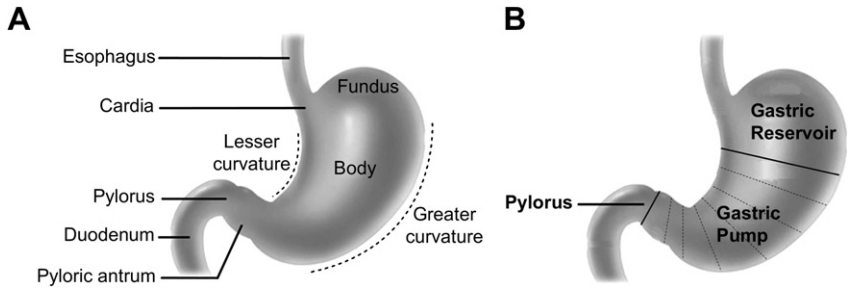

Fig. 1. Anatomic and functional regions of the stomach. (*A*) Anatomic regions. (*B*) Functional regions. (*Adapted from* Mercer DW, Liu TH, Castaneda A. Anatomy and physiology of the stomach. In: Zuidema GD, Yeo CJ. Shackelford's surgery of the alimentary tract. 5th edition, vol. 2. Philadelphia: Saunders; 2002. p. 3. Copyright Elsevier; with permission.)

motility, and occurs in the absence of any other influence.[2] Neural regulation emanates primarily from the intrinsic gastric myenteric plexus, with further contributions from extrinsic parasympathetic (vagal) and sympathetic (splanchnic) input.[4] Hormonal influences play a significant role in the regulation of gastric motility. The list of hormones known to modulate gastric motility is extensive (**Table 1**[5–10]).

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.[4] 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.[4] 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.[11]

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.[2] During this period the proximal stomach undergoes tonic contraction, while the gastric slow wave modulates the coordinated peristalsis of the distal stomach.[4] 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 (**Fig. 2**[12]).

Postprandial Gastric Motility

Five to 10 minutes after the ingestion of food the MMC gives way to the fed state of gastric muscle activity.[2,4] 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