Anatomy and Physiology of the Stomach

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Among the viscera, the stomach is among the earliest to have been described by priests, physicians, and anatomists and to have been studied functionally by alchemists, chemists, and physiologists [1–3]. The ancient Egyptians recognized the gross anatomy and the infirmity of the stomach; at the time of burial, it was preserved separately in one of the four so-called “canopic” jars (protected by the jackal god-son of Horus, Tuamutef). Hippocrates called digestion “pepsis,” likening it to cooking, and proposing that the heat of the stomach was responsible for the breakdown of food [1].

A scientifically motivated understanding of gastric structure and function can be traced to 1547, when Andreas Vesalius, in his De Humani Corporis Fabrica, provided anatomically correct descriptions of the human stomach and intestines. In 1648, observations of animal digestion led J.B. van Helmont to postulate that different kinds of acids might play a role in digestion, calling them ferments [2]. In the 1780s, Lazzaro Spallanzani published his Dissertationi de Fisica Animale e Vegetale. This and his subsequent observations were works of extraordinary breadth and dedication to providing empirical distinction among fermentation (a chemical process of dissolution), digestion (the chemical process of dissolution produced by vital organs), and trituration (the mechanical process of foodstuff disintegration). Spallanzani had experimental subjects, including himself, swallow enclosed receptacles (linen bags or perforated metal tubes). He observed that, over time, the contents disappeared from the receptacle and postulated the involvement of acid [3,4]. In 1823, Prout, Tiedemann, and Gmelin each independently identified the acid in the stomach as hydrochloric acid [4]. International excitement and acclaim followed the publication in 1833, by the American army surgeon William Beaumont, of his Observations on the Gastric Juice and the Physiology of Digestion. Taking advantage of the opportunity to study human digestion through the portal of a gastro-cutaneous fistula in the...
young fur trapper, Alexis St. Martin, Beaumont persuasively confirmed the hypothesis that proper digestion requires the secretion of hydrochloric acid, observed evidence for an additional factor that permits putrefaction (pepsin?), and recognized changes in mucosal color and gastric motility in response to emotional disturbances or ingestion of strong spirits [5]. Beaumont is also generally credited with recognizing that secretion of digestive agents implies that the stomach has mechanisms for protecting itself from the damaging effects of its secretions [5], a physiologic principle not experimentally defined until the early 1960s in the work of Charles Code [6] and Horace Davenport [7].

Anatomy of the stomach

Landmarks

Topographically, the stomach has five regions (Fig. 1): (1) the cardia and gastroesophageal (GE) junction, (2) the fundus, (3) the corpus, (4) the antrum, and (5) the pylorus. The fundus and corpus harbor acid-secreting glands, whereas the antrum harbors alkaline-secreting surface epithelium and endocrine, gastrin-secreting G-cells. Viewed through a laparotomy incision or a laparoscope (Fig. 2), the GE junction is recognized at the sharp angle between the rounded dome of the fundus and the straight esophageal tube. The pylorus has no easily visualized landmarks, but is easily palpated as a ring of muscle separating the stomach and duodenum. Viewing the stomach externally, the junction between the acid-secreting corpus and the non-acid secreting antrum is identified on the lesser curvature by the incisura angularis.

Viewed endoscopically, the GE junction is easily distinguished by the transition between the flat, pale, stratified epithelium of the esophagus and

Fig. 1. Topography of the stomach.
the lush, pink, glandular epithelium of the upper stomach (Fig. 3A, B). The junction between the acid-secreting corpus and the non-acid secreting antrum is also relatively easily distinguished by the rugal pattern: those of the antrum are linear and aligned with the long axis of the organ, whereas those of the corpus are convoluted and oriented obliquely (Fig. 3C). The

Fig. 2. Laparoscopic view of the stomach. (A) Anterior view. (B) GE junction, left crura, and anterior vagus. (C) Posterior vagus.
pylorus is also easily visualized, outlined by the underlying ring of muscularis (Fig. 3D).

In the elderly, the non-acid secreting mucosa of the antrum may migrate cephalad, replacing acid-secreting mucosa in association with up to a 30% decrease in functional acid-secreting capacity [8–10]. Loss of oxyntic mucosa is likely due to the presence of chronic gastritis [8,10], increasing the area of gastrin-secreting mucosa and also altering the region of decreased resistance where gastric ulcers tend to arise (within 2 to 3 cm of the corpus/antrum junction) [11]. These are important considerations in choosing the boundaries of distal gastric resection for peptic ulcer disease.

Anatomic relationships

At the GE junction, the anatomic relationships include the diaphragm and crura (Figs. 4 and 5). Laterally, the cardiac notch signals a cardiac fat pad that must be released to expose the left crura. At the level of the fundus and proximal corpus, which are oriented vertically, the spleen is lateral and the lateral segment of the left lobe is medial and anterior (see Figs. 4 and 5).
Posteriorly and medially lies the abdominal aorta, after being transmitted from the thorax through the diaphragm. Importantly, if the left lobe must be mobilized to expose the GE junction or proxima lesser curvature, the triangular ligament of the left hepatic lobe is incised, but not so far to the left as to injure the branch of the left inferior phrenic vein that passes in front of the esophageal hiatus toward the inferior vena cava, anteriorly and to the right.

The incisura signals the junction of the distal corpus and antrum (see Fig. 3), which are oriented horizontally. At this level, the aorta passes directly posterior to the body of the pancreas, which is in turn directly posterior to the gastric antrum. The transverse colon hangs interiorly, and the splenic flexure lies laterally to the left. The fundus of the gallbladder hangs superior to the pylorus and duodenal bulb, and the common bile duct passes posterior to the duodenal bulb on its way into the head of the pancreas, ultimately to empty on the medial wall of the duodenum.

The greater omentum is suspended from the greater curvature of the stomach, and has largely avascular attachments to the hepatic flexure, transverse segment, and splenic flexure of the colon. The lesser omentum

Fig. 4. CT images of the stomach—transverse sections. (A) Relationships of the cardia and fundus. (B) Relationships in the proximal corpus. (C) Relationships in the distal corpus, at the level of the celiac axis and the splenic artery. (D) Relationships of the antrum and pyloris. In this panel, the patient has a duodenal perforation in the duodenal bulb.
hangs between the lesser curvature of the stomach and a plane roughly connecting the falciform ligament. A portion of the lesser omentum, the pars flaccida, lies loosely near the lesser curvature and is a guidepost in morbid-obesity operations.

**Arterial blood supply**

The stomach is richly vascularized, with contributions from five major sources (Fig. 6): (1) the left gastric artery, a branch of the celiac axis, which supplies the cephalad portion of the lesser curvature; (2) the right gastric artery, a branch of the common hepatic artery, which supplies the caudal portion of the lesser curvature; (3) the right gastroepiploic artery, a branch of the gastroduodenal artery, which supplies the antrum and lower corpus; (4) the left gastroepiploic artery, a branch of the splenic artery, which
supplies the upper corpus; and (5) a series of short gastric arteries passing to the fundus and cephalad portion of the corpus from the splenic hilum, and thus ultimately from the splenic artery. An inconstant branch to the pylorus has been also described, often as a branch of the gastroduodenal artery. On the lesser curvature, the left gastric artery does not always trace directly back from the lesser curvature to the celiac axis; in some cases it dips behind the body the pancreas before ascending posteriorly. On the greater curvature, there is a small bare area between the entrances of the right and left gastroepiploic into the gastric wall. This bare area serves as a useful landmark in identifying the proximal extent of the gastric antrum, corresponding to the incisura on the lesser curvature.

Fig. 6. Magnetic resonance arteriography (MRA) images of gastric vascular anatomy. (Upper) Maximum intensity projection (MIP) showing all branches of the celiac axis, with organs subtracted. (Lower) Three-dimensional projections to enhance vessels around the stomach, with attenuation of branches of hepatic artery and superior mesenteric artery. (Courtesy of Matthew Barish, MD, Department of Radiology, Brigham and Women’s Hospital.)
**Innervation**

The vagus nerves descend laterally along the esophagus; at the diaphragm they form the anterior and posterior vagal trunks (Fig. 7). At the level of the diaphragm, the anterior vagus is composed variably of one or two, and occasionally three, trunks adherent to the muscularis of the esophagus (Fig. 8) [12]. At the level of the GE junction, small branches pass through the anterior leaflet of the lesser omentum toward the liver and gallbladder; at this point, the vagal trunk becomes the anterior nerve of Latarjet. At this level, the posterior vagus is usually, but not always, a single trunk, passing the left side of the esophagus, bowing away from the lesser curvature. At the GE junction, small branches diverge to the right and posteriorly, and a sizeable branch is often observed angling sharply to the left to curl around the cardia (see Fig. 2C). In ulcer surgery, failure to recognize this latter branch, the so-called “criminal” nerve of Grassi, is thought to be responsible for some cases of incomplete vagotomy and subsequent recurrence of symptoms.

**Lymphatic drainage**

Lymphatic drainage pathways run in close proximity to the arterial supply (Fig. 9) [13]. A superior or left gastric group of nodes (between 10 and 20) lie along the cephalad lesser curvature and the left gastric artery. A suprapyloric group of nodes (3 to 6) lies along the lesser curvature and right gastric artery. The pancreaticosplenic group of nodes (3 to 5) drain the greater curvature along the fundus and upper corpus. Between 6 and 12 nodes lie along the right gastroepiploic artery. An additional subpyloric

![Fig. 7. Schematic illustration of the vagus and its branches as they descend along the greater curvature of the stomach.](image-url)
group of nodes (6 to 8) are identified at the pylorus and junction of the right gastroepiploic artery and gastroduodenal artery. Interconnections are numerous. For the purposes of staging of gastric carcinoma, 16 nodal stations have been distinguished, according to the Japanese Research Society for the study of Gastric Cancer (JRSGC). These stations are outlined in Table 1 [13], along with their designations as local (R1), regional (R2) or distal-regional (R3) spread.

Fig. 8. Variations in anatomy of the anterior and posterior branches of the vagus nerves in the region of the GE junction and diaphragm. Incidence (%) of each anatomic group is indicated. (Adapted from Jackson RG. Anatomic Record 1949;103:1, 6; with permission.)

Fig. 9. Regional lymphatic drainage sites of the stomach, classified according to the Japanese Research Society for the Study of Gastric Cancer (JRSGC). (From Jpn J Surg 1981;11:127–39; with permission.)
Table 1
Stations of nodal spread in gastric cancer, classified according to the system of the Japanese Research Society of study of Gastric Cancer

<table>
<thead>
<tr>
<th>Station</th>
<th>Location</th>
<th>Antrum</th>
<th>Corpus/fundus</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Right cardia</td>
<td>R2</td>
<td>R1</td>
</tr>
<tr>
<td>2</td>
<td>Left cardia</td>
<td>R2</td>
<td>R1</td>
</tr>
<tr>
<td>3</td>
<td>Lesser curve</td>
<td>R1</td>
<td>R2</td>
</tr>
<tr>
<td>4</td>
<td>Greater curve</td>
<td>R1</td>
<td>R2</td>
</tr>
<tr>
<td>5</td>
<td>Suprapyloric Right gastric a.</td>
<td>R1</td>
<td>R2</td>
</tr>
<tr>
<td>6</td>
<td>Infracyloric</td>
<td>R1</td>
<td>R2</td>
</tr>
<tr>
<td>7</td>
<td>Left gastric a.</td>
<td>R1</td>
<td>R1</td>
</tr>
<tr>
<td>8</td>
<td>Common hepatic a.</td>
<td>R2</td>
<td>R2</td>
</tr>
<tr>
<td>9</td>
<td>Celiac axis</td>
<td>R3</td>
<td>R3</td>
</tr>
<tr>
<td>10</td>
<td>Splenic hilum</td>
<td>R3</td>
<td>R1</td>
</tr>
<tr>
<td>11</td>
<td>Splenic a.</td>
<td>R3</td>
<td>R1</td>
</tr>
<tr>
<td>12</td>
<td>Hepatoduodenal Ligament</td>
<td>R2</td>
<td>R1</td>
</tr>
<tr>
<td>13</td>
<td>Pancreas head</td>
<td>R2</td>
<td>R2</td>
</tr>
<tr>
<td>14</td>
<td>Root of SMA</td>
<td>R3</td>
<td>R3</td>
</tr>
<tr>
<td>15</td>
<td>Middle Colic a.</td>
<td>R3</td>
<td>R3</td>
</tr>
<tr>
<td>16</td>
<td>Para-aortic</td>
<td>R3</td>
<td>R3</td>
</tr>
</tbody>
</table>


Functional anatomy and physiology

The gastric mucosa

Functionally, the gastric mucosa is divided into acid-secreting and non-acid secreting regions. Acid- and pepsinogen-secreting mucosa is found in the corpus and fundus. The acid secreting unit of the mucosa is the gastric gland, schematically illustrated in Fig. 10. At the base of the gastric gland lie the pepsinogen-secreting chief cells. The middle of the gastric gland is populated largely with the HCl-secreting parietal cells. Toward the lumen, at the neck, parietal cells are still present, but give way to mucus neck cells and then, near the opening, the mucosa is largely populated with surface epithelial cells. Intercalated between parietal cells and smaller immature cells are enterochromaffin-like (ECL) cells expressing histidine decarboxylase, the enzyme that is essential in production of the paracrine agonist, histamine. The key features of the cell biology of acid secretion [14,15] are illustrated in Fig. 11, including its basis in ion transport (Fig. 11A), and in intracellular signaling stimulated by locally active neurohumoral agonists (Fig. 11B).

Neuroendocrine regulation of acid secretion

Three neurohumoral pathways figure prominently in the stimulation of acid secretion by the gastric mucosa [16]. These include: (1) acetylcholine, which is released by the vagus nerve; (2) histamine, released locally by ECL cells; and (3) gastrin, released by the gastric antrum and carried through the circulation to act on ECL cells and the parietal cells. As emphasized in
Fig. 11 B, full function of each pathway relies on robustness of the others. Thus, blockade of histamine H₂ receptors by drugs such as cimetidine attenuates secretory responses to cholinergic agonists, and interruption of vagal efferents attenuates responses to histamine [15,17,18].

A key feature of the antral mucosa is the presence of gastrin-secreting G cells and somatostatin-secreting D cells. Only recently has it been appreciated that acidity of the gastric lumen activates the secretion of somatostatin, which in turn inhibits secretion of gastrin. The converse is true: alkaline pH reduces somatostatin secretion, which in turn permits circulating gastrin levels to rise. As discussed below, this relationship is a cornerstone in the physiology of meal-stimulated acid secretion. It is important to note that gastrin receptors (classified as gastrin/cholecystokinin type B receptor [CCKB]) on the parietal cell are largely trophic; that is, they stimulate growth and development of parietal cells [18,19]. In experimental systems looking at parietal cell function in isolation, gastrin is not a strong agonist of acid secretion. The power of gastrin as a secretory agonist lies in its stimulation of histamine release by the ECL cell [17,18]. Fig. 12 summarizes pharmacologic and surgical approaches to inhibition of acid secretion, based on the physiologic concepts outlined in Fig. 11 B.

Three endogenous classes of inhibitory neurohumoral signals are somatostatin, epidermal growth factor and transforming growth factor alpha (EGF/TGFα), and prostaglandins of the E and I series. Somatostatin indirectly regulates acid secretion through its effects on gastrin secretion and independent suppression of histamine release from the ECL cell. It remains unclear whether somatostatin directly alters parietal cell responses to secretory stimulation by cholinergic agonists or histamine. Inhibition of acid
secretion by EGF/TGFα occurs within the parietal cell, through modulation of intracellular tyrosine kinase pathways that have downstream regulatory influences on signaling pathways discussed above [14]. Prostaglandin E₂ has effects at several levels, including release of histamine and suppression of intracellular signaling pathways in the parietal cells that are activated by cholinergic agonists and histamine [20,21]. Thus, acid secretion may be inhibited physiologically, by endogenous neurohumoral agents that act at the level of the brain and central nervous system (CNS), at the level of the histamine-secreting ECL cell, and at the level of the parietal cell. Thus far, none of these endogenous inhibitory pathways has provided a basis for clinical interventions in controlling acid secretion.

**Alkaline secretion by gastric mucosa**

The non-acid secreting mucosa of the gastric antrum and pylorus is characterized by the presence of relatively simple glands populated by mucus- and HCO₃⁻-secreting surface epithelium. The surface epithelium, in
both the antrum and the corpus/fundus regions, is the basis of the “mucosal barrier.” The mechanisms thought to protect the mucosa from back-flux of $H^+$ from the lumen are illustrated in Fig. 13.

**Gastric digestion and contributions to downstream absorption**

The stomach contributes to digestion of solid food by mixing chyme with acid and pepsin (pepsinogen autoactivated in the presence of luminal acid), which helps break down protein to simple peptides that will be absorbed or broken down further by intestinal peptidases. Subpopulations of parietal
cells also secrete intrinsic factor, an essential cofactor in the absorption of vitamin B₁₂ downstream in the terminal ileum. Gastric acid itself enables absorption of specific metals and nonmetal cations, including Ca²⁺, Fe³⁺, and other trace metals. At low pH, Ca²⁺ is more fully released from binding bases, and is thus more available for absorption in the duodenum. Similarly, Fe²⁺ auto-oxidizes in the presence of luminal acid, placing it in a form more easily absorbed in the small intestine.

**Gastric motility**

The stomach has three layers of muscularis: an inner circular layer, a middle longitudinal layer, and an outer but incomplete oblique layer. Motor functions in the stomach are segregated by region. The fundus relaxes as fluids and solids enter the esophagus, a response known as receptive relaxation, and further as food actually enters the fundus, a process known as adaptive relaxation [22,23]. This response allows the liquid to pool in the fundic pouch while the solid components of the meal remain in the mainstream of flow toward the pylorus.

On the greater curvature, in muscularis of the upper corpus, lies the primary electrical pacemaker of the stomach. Superimposed on the basic electrical rhythm of the pacemaker, the corpus and antrum engage in

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![Diagram of mucosal blood flow](image-url)
a coordinated propulsion of the luminal contents toward the pylorus. The pylorus itself acts as a sieve, remaining open in anticipation of the wave of peristalsis. As the wave advances, small particles pass through the pyloric sphincter; when the wave hits, the pylorus closes, thereby acting as a barricade. The chyme, propelled with increasing velocity against the pyloric sphincter, is thus broken up by enzymatic digestion in combination with mechanical disruption.

**Satiety**

The role of the stomach in regulating food intake has become an increasingly important theme, especially with increasing numbers of procedures for bariatric surgery. In this regard, the recently described hormone ghrelin has assumed central importance. Ghrelin is an appetite-stimulating hormone that is released by gastric mucosal to the portal circulation when the stomach is empty, passing to the central circulation to stimulate appetite centers in the hypothalamus; circulating levels of ghrelin fall precipitously as soon as the stomach begins to fill. In bariatric surgical procedures that create small pouches that distend quickly, baseline and premeal peaks of ghrelin are suppressed, suggesting that blunting of ghrelin responses may contribute to suppression of appetite after bariatric surgery. By no means is ghrelin the dominant signal for control of satiety (Fig. 14) [24], but its effects must be understood in the context of other neural and hormonal inputs to satiety centers in the pituitary.

*An integrated view of gastric function in response to a meal*

When chyme, containing both liquid and solid components, enters the stomach, the process of true digestion begins, distinguished from mastication upstream in the oral cavity and absorption that occurs downstream in the intestine. Between meals, gastric secretion in the average adult is relatively low, producing an average of 4 mEq/hr (~25 mL of pure gastric juice). The sensation if hunger is mediated by a multidimensional process, including conditioned behaviors [25] and release of key hormones such as ghrelin. The sight and smell of food initiates the vagally-mediated, Pavlovian response, which not only activates salivation in the oral cavity but also initiates the cephalic phase of acid secretion in the stomach. In addition, gastrin-releasing peptide (GRP) is released by vagal inputs to antral G-cells, thereby activating early release of gastrin in anticipation of the passage of the meal to the stomach. About 15% of the total quantity of acid that is secreted in response to a meal [16] is attributed to the cephalic phase. The capacity to secrete acid given only the sight, smell, and chewing of food leads to a reasonably reliable method for monitoring the completeness of vagotomy in postoperative patients. In one study, Bradshaw and Thirlby [26] used such sham-feeding protocols to identify patients with unexpectedly
robust vagal responses to a meal, thereby identifying those patients as candidates for additional antisecretory therapy after vagotomy.

In addition to stimulation of acid secretion, the cephalic phase of vagal stimulation also prepares the gastric fundus to relax in anticipation of the flow of chyme into the stomach [22]. The predigestive phase of acid secretion is mediated by cholinergic efferents, but the process of receptive relaxation is mediated by noncholinergic vagal fibers, involving capsaicin-sensitive fibers that elaborate calcitonin gene-related peptide (CGRP) and nitric oxide (NO) as neurotransmitters [23].

Food entering the gastric lumen initially segregates into solid components that, by and large, stay within the mainstream, and liquid components that are diverted to the expanding gastric fundus (adaptive relaxation). Distension of the gastric antrum and increases in pressure stimulate peristalsis and churning within the mainstream of the gastric lumen, a process known as trituration. The admixture of acid, activation of pepsinogen, and the increasingly accessible protein components of the chyme leads to rapid breakdown into smaller peptides. Expansion of the intragastric space, increase in luminal pressure, the appearance of small peptides, and the rapid buffering of luminal
acid all lead to suppression of somatostatin secretion and enhancement of gastrin release, which in turn activates local release of histamine from ECL cells. Local vagally mediated reflexes enhance parietal cell responsiveness to histamine. This gastric phase of acid secretion is responsible for about 75% of the total secretory response [16]. In normal subjects, the integrated secretory response to a steak meal is about 90 to 100 mEq over 3.5 hours, equivalent to approximately 650 to 700 cc of gastric juice [16].

Importantly, during this period, trituration of chyme leads to its pulverization and accumulation of small fragments that will pass through the sieve created by the pylorus just before the advancing wave of peristalsis. As the lumen contracts, sequestered liquid from the fundus starts to pass into the mainstream and facilitates a more thorough mixing of the remnants of the chyme with pepsins.

Potentially important physiologic disturbances of mixing and motility occur in response to vagotomy, which is usually accompanied by loss of the pyloric gating function. These consequences include: (1) early emptying of liquids, caused by loss of receptive and adaptive relaxation, which leads to bloating and gas pain even in the absence of pyloric obstruction [27,28]; (2) rapid emptying of hyperosmotic or inadequately digested chyme into the intestine, caused by bypass of loss of pyloric function, which leads to early and late dumping syndromes [27,29]; and (3) bile backwash and overgrowth of bacteria in the normally clean (<100 cfu/ml) gastric lumen caused by loss of gastric acidity, which leads to disturbances in mucosal proliferation and growth and perhaps malignant transformation [30,31]. These predictable disturbances should be monitored and taken into account in assessment of the efficacy and risks of emerging bariatric procedures.

**Evolving areas of interest in gastric physiology**

Despite intense interest over 200 years, there remains no satisfactory answer to the question posed by William Beaumont [32]: why does the stomach not digest itself? Whereas the last 200 years of inquiry have been directed at understanding conditions and mechanisms of acid secretion, new interventions and procedures may be expected to challenge our understanding of the mucosal resistance to the damaging effects of luminal acid and other hostile luminal conditions. Over the years, several paradoxes have been presented by experimental work in each of the putative dimensions of gastroprotection. For example, considerable interest attended observations that physical properties of gastric mucus are altered by ambient pH [33]. Extracted from the interface of the gastric mucosa and lumen, mucin resists bulk flow of acid but not diffusion of protons [34]. Gastric mucin is a complex structure, characterized by cysteine-rich clusters and noncovalent binding of protein, lipid, and carbohydrate components [35,36]. Mucin components are also thought to play a role in mucosal proliferation, growth, and renewal [37], identifying them as putative growth
factors that might contribute to healing of chronic peptic ulcers [38] and pathogenesis of malignancy [39].

Equally intriguing are recent observations that the interaction between food and bacteria in the upper gastrointestinal (GI) tract profoundly influence mucosal function, even under physiologic conditions. Recent studies have suggested that dietary nitrate \( \text{NO}_3^- \), found in many meats and foodstuffs, are rapidly reduced to nitrite \( \text{NO}_2^- \) by nitrate reductase systems of commensal bacteria that reside in the oropharynx [40,41]. These nitrites are converted by gastric acid to nitric oxide [42], a highly consequential and biologically active agent that influences gastric mucosal function, motility, and blood flow. Depending on the circumstances, NO and its breakdown products may be considered helpful or harmful to mucosal function and integrity [43–45]. In recent studies, it has been suggested that therapeutic manipulations affecting function of the foregut (oral cavity to duodenum) may alter mucosal function, growth, and barrier function at the gastro-esophageal junction, an area increasingly recognized for its susceptibility to metaplasia and malignant transformation [46]. These considerations emphasize that surgeons should be at the forefront of investigation into biochemical and physiological stresses that might be caused by emerging pharmacologic interventions and invasive procedures (surgical, minimally invasive, or endoscopic) of the stomach and GE junction.

References


