01 - SECTION 1 Disorders of the Alimentary Tract

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Disorders of the Gastrointestinal System PART 10 Section 1 Disorders of the Alimentary Tract William L. Hasler, Chung Owyang

Approach to the Patient

with Gastrointestinal

Disease ANATOMIC CONSIDERATIONS The gastrointestinal (GI) tract extends from the mouth to the anus and is composed of organs with distinct functions. Sphincters assist in gut compartmentalization to separate the organs. Distinct gut wall layers contribute to regional activities. The mucosa regulates fluid and nutrient transfer but is also a barrier to prevent entry of some luminal contents. Smooth muscle and enteric nerves mediate pro pulsion. Many organs possess a serosal layer to provide a supportive foundation and permit external input. The gut interacts with many other systems. Pancreaticobiliary conduits deliver bile and enzymes into the duodenum. Lymphatic channels assist in gut immune activities. Intrinsic nerves control propulsion and fluid regulation. Extrinsic neural input provides volitional or involuntary control specific for each gut region. FUNCTIONS OF THE GI TRACT The GI tract serves two main functions—assimilating nutrients and eliminating waste. In the mouth, food is processed, mixed with salivary amylase, and delivered to the pharynx. Aborally propagating esopha geal contractions propel the ingested bolus into the stomach in coor dination with relaxation of the upper and lower esophageal sphincters. Basal tone in the lower esophageal sphincter prevents gastroesophageal reflux. The stomach triturates and mixes food with pepsin and acid. The proximal stomach serves a storage function by relaxing to accommo date the meal. Phasic contractions in the distal stomach propel food residue against the pylorus, where it is ground and thrust proximally for further mixing prior to emptying into the duodenum. Most nutrient absorption occurs in the small intestine. Mucosal villi provide maximal surface area and possess specialized enzymes and transporters. Triturated food from the stomach mixes with pancreatic secretions and bile in the duodenum. Pancreatic juice contains both enzymes for digestion and bicarbonate to optimize luminal pH for enzyme activation. Bile secreted by the liver and stored in the gallbladder is needed for lipid digestion. Most nutri ents and minerals

are rapidly absorbed in the proximal intestine, while bile acids and vitamin B12 are absorbed in the ileum. Bile contains byproducts of erythrocyte degradation, toxins, medica tions, and cholesterol that are emptied into the colon along with indigestible residue. The ileocecal junction is a sphincter that limits coloileal reflux of colonic microbes. The colon prepares waste for evacuation by to-and-fro contractions that dehydrate the stool. As a consequence, daily luminal volumes decrease from 1000–1500 mL in the ileum to 100–200 mL that are expelled from the rectum. Esophageal transit takes seconds, stomach and small intestine emptying times range from minutes to a few hours, but colon propagation requires >1 day in most individuals. A dense colonic microbiome ferments undigested carbohydrates and shortchain fatty acids and modulates immune activity. The colon terminates in the anus, which possesses volitional and involuntary controls to retain stool in the rectum until it can be released in a convenient setting.

EXTRINSIC MODULATION OF GUT FUNCTION GI function is modified by influences outside the gut. Unlike other systems, the gut is in continuity with the outside environment. Several mechanisms protect against injury from foods, medications, toxins, and microbes. Mucosal immune pathways include epithelial and lamina propria lymphocytes and plasma cells and lymph node chains to prevent noxious agents from entering the circulation. Antimicrobial peptides secreted by Paneth cells defend against pathogens. Drugs and toxins absorbed into the portal venous circulation are filtered and detoxified in the liver. Many GI reflexes involve extrinsic vagus or splanchnic nerve pathways. The brain-gut axis alters function in gut regions not under volitional regulation. Stress disrupts normal gut motor, secretory, and sensory activities. OVERVIEW OF GI DISEASES GI diseases range in severity from conditions that produce mild symp toms and no long-term morbidity to those with intractable symptoms or adverse outcomes. Diseases may be localized to one gut organ or exhibit diffuse involvement at many sites. ■ ■CLASSIFICATION OF GI DISEASES GI diseases are manifestations of alterations in nutrient and fluid assimilation, transit and sensation, immune function, and vascular supply. Neoplastic degeneration may affect any GI site. Genetic factors underlie some disorders. Impaired Digestion and Absorption Diseases of the stomach, small intestine, biliary tree, and pancreas can disrupt digestion and absorption. The most common maldigestion syndrome, lactase defi ciency, produces gas and diarrhea after ingesting dairy products but has no adverse outcomes. Intestinal sucrase-isomaltase deficiency produces similar symptoms after consuming sucrose. Disorders that affect digestion and/or absorption more diffusely, including celiac disease, bacterial overgrowth, infectious enteritis, Crohn’s ileitis, and radiation enteritis produce anemia, dehydration, electrolyte disorders, or malnutrition. Gastric acid hypersecretory conditions impair diges tion by damaging the intestinal mucosa, impairing pancreatic enzyme activation, and accelerating transit. Benign or neoplastic biliary obstruction impairs fat digestion. Impaired pancreatic enzyme release in chronic pancreatitis or pancreatic cancer impairs digestion and leads to malnutrition. Altered Secretion Some GI diseases result from alteration of gut secretion. Gastric acid hypersecretion occurs in gastrinoma, G-cell hyperplasia, retained antrum, and some patients with duodenal ulcers. Gastric acid is reduced in atrophic gastritis and pernicious anemia. Intestinal or colonic hypersecretion and/or impaired absorption causes diarrhea with acute bacterial or viral infection, chronic Giardia or cryptosporidia infections, small-intestinal bacterial overgrowth, bile salt diarrhea, microscopic colitis, diabetic diarrhea, and endocrine neoplasias with tumor overproduction of secretagogue transmitters such as vasoactive intestinal polypeptide. Altered Gut Transit Mechanical obstruction can block transit of gut contents. Esophageal occlusion most often is due to stricture (from acid exposure or eosinophilic esophagitis) or neoplasm. Gastric obstruction develops from ulcer disease or cancer. Small-intestinal obstruction commonly results from adhesions but also occurs with Crohn’s disease, radiation- or

drug-induced strictures, and malig nancy. The most common cause of colonic obstruction is colon cancer, although inflammatory strictures develop with inflammatory bowel disease (IBD), after diverticulitis, or with some medications. Delayed propulsion can develop in any gut region. Achalasia is characterized by impaired esophageal body peristalsis and incomplete lower esophageal sphincter relaxation. Gastroparesis is the delay in gastric emptying resulting from impaired antral motility or spastic

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