Physiology of Gastrointestinal Disorders

Representative types of gastrointestinal malfunction that have special physiologic bases or consequences.

Disorders of Swallowing and of the Esophagus

Paralysis of the Swallowing Mechanism:

CAUSES:

- Damage to the $5^{th,}$ 9^{th} or 10^{th} cerebral nerve can cause paralysis of significant portions of the swallowing mechanism.
- Diseases, such as *poliomyelitis or encephalitis, can prevent* normal swallowing by damaging the swallowing center in the brain stem.
- Paralysis of the swallowing muscles, as occurs in *muscle dystrophy* or in failure of neuromuscular transmission in *myasthenia gravis or botulism, can also prevent* normal swallowing.
- when patients are under deep anesthesia, anesthetic blocks the reflex mechanism, causing vomiting & choking

CONSEQUENCES:

- Complete abrogation of the swallowing act
- Failure of the glottis to close so that food passes into the lungs instead of the esophagus,
- Failure of the soft palate and uvula to close the posterior nares so that food refluxes into the nose during swallowing.

Achalasia and Megaesophagus.

Achalasia is a condition in which the lower esophageal sphincter fails to relax during swallowing. As a result, food swallowed into the esophagus then fails to pass from the esophagus into the stomach.

CAUSES: Damage in the neural network of the myenteric plexus in the lower two thirds of the esophagus.

CONSEQUENCE:

When achalasia becomes severe, the esophagus often cannot empty the swallowed food into the stomach for many hours, Over months and years, the esophagus becomes tremendously enlarged (M**egaesophagus) ,** often becomes putridly infected during the long periods of esophageal stasis.

The infection may also cause ulceration of the esophageal mucosa, sometimes leading to severe substernal pain or even rupture and death.

TREATMENT: Considerable benefit can be achieved by stretching the lower end of the esophagus by means of a balloon inflated on the end of a swallowed esophageal tube. Antispasmotic drugs (anticholinergic drugs that relax smooth muscle) can also be helpful.

Disorders of the Stomach

Gastritis—Inflammation of the Gastric Mucosa.

➢Common in the middle to later years of adult life. Gastritis inflammation may be only superficial and therefore not very harmful, or it can penetrate deeply into the gastric mucosa, sometimes causing almost complete atrophy of the gastric mucosa.

➢In a few cases, gastritis can be acute and severe (gastric ulcers), with ulcerative excoriation of the stomach mucosa by the stomach's own peptic secretions.

CAUSES

➢Caused by chronic bacterial infection of the gastric mucosa. Can be treated successfully by an intensive regimen of antibacterial therapy.

➢Certain ingested irritant substances (excesses of *alcohol or aspirin)* can be especially damaging to the protective gastric mucosal barrier often leading to severe acute or chronic gastritis.

➢Chronic gastritis, the mucosa atrophy will result in little or no gastric gland digestive secretion (*achlorhydria and, occasionally, to pernicious anemia)*.

➢Autoimmune reactions against the gastric mucosa, can also lead to gastric atrophy.

Peptic Ulcer

A peptic ulcer is an excoriated area of stomach or intestinal mucosa caused principally by the digestive action of gastric juice or upper small intestinal secretions.

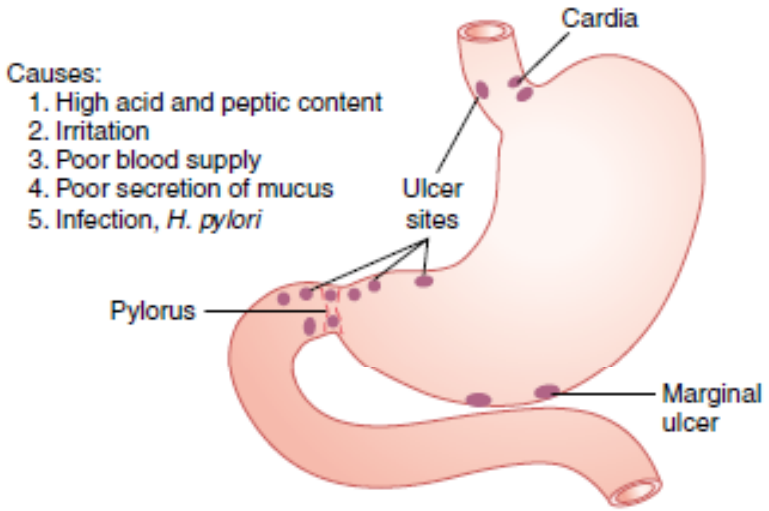


Figure 66-1 Peptic ulcer. *H. pylori, Helicobacter pylori.*

The mucosa protection is provided by large amounts of bicarbonate ions which inactivates pepsin and thus prevent mucosal damage.

1. Pancreatic secretion
2. Secretions of the large Brunner's glands in the initial region of the duodenum
3. Bile coming from the liver.

- Stimulation of gastric factors causing excess secretion of acid and pepsin by the gastric mucosa (psychic disturbances)

- Diminished gastroduodenal inhibition can cause peptic ulcers

CAUSES:

1. Bacterial Infection by *Helicobacter pylori Breaks Down the* Gastroduodenal Mucosal Barrier and Stimulates Gastric Acid Secretion (physical capability to burrow through the barrier and by releasing ammonium that liquefies the barrier).

2. Psychic disturbances Stimulates Gastric Acid Secretion, *Smoking,* presumably because of increased nervous stimulation of the stomach secretory glands

4. Alcohol, Aspirin and other nonsteroidal anti-inflammatory drugs have a strong propensity for breaking down this barrier.

TREATMENT

(1) Antibiotics
(2) Administration of an acid suppressant drug, especially *ranitidine, an antihistaminic* that blocks the stimulatory effect of histamine on gastric gland histamine2 receptors, thus reducing gastric acid secretion by 70 to 80 %.

Old treatment

1. Removal of as much as four fifths of the stomach, thus reducing stomach acid–peptic juices enough to cure most patients.
2. To cut the two vagus nerves that supply parasympathetic stimulation to the gastric glands.

(Basal stomach secretion returned after a few months and in many patients the ulcer also returned.)

Disorders of the Large Intestine

Constipation: Often associated with large quantities of dry, hard feces in the descending colon that accumulate because of over absorption of fluid.

CAUSES

Any pathology of the intestines that obstructs movement of intestinal contents, such as tumors, adhesions that constrict the intestines, spasm of a small segment of the sigmoid colon, or ulcers, can cause constipation.

A frequent functional cause of constipation is irregular bowel habits that have developed through a lifetime of inhibition of the normal defecation reflexes.

Megacolon (Hirschsprung's Disease).

➢Occasionally, constipation is so severe that bowel movements occur only once every several days or sometimes only once a week. This allows tremendous quantities of fecal matter to accumulate in the colon, causing the colon sometimes to distend to a diameter of 3 to 4 inches.

CAUSE: lack of or deficiency of ganglion cells in the myenteric plexus in a segment of the Sigmoid colon. As a consequence, neither defecation *reflexes* nor strong peristaltic motility can occur in this area of the large intestine.

Diarrhea: Diarrhea results from rapid movement of fecal matter through the large intestine.

CAUSES

Enteritis—Inflammation of the Intestinal Tract usually caused either by a virus or by bacteria, most common in the large intestine and the distal end of the ileum (Cholera).

TREATMENT:
Antibiotics and intravenous solutions.

Psychogenic Diarrhea.

Diarrhea that accompanies periods of nervous tension, such as during examination time caused by excessive stimulation of the parasympathetic nervous system, which greatly excites both
(1) motility and (2) excess secretion of mucus in the distal colon. These two effects added together can cause marked diarrhea.

Ulcerative Colitis. extensive areas of the walls of the large intestine become inflamed and ulcerated resulting from an allergic or immune destructive effect, but it also could result from chronic bacterial infection.

Vomiting

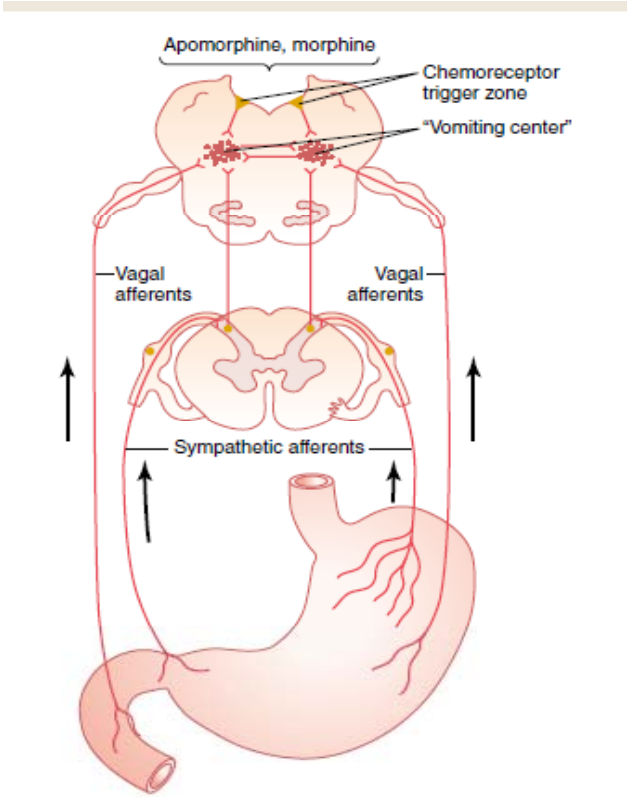
➢Vomiting is the process by which the upper GI tract rids itself of its contents when almost any part of the upper tract becomes excessively irritated , overdistended, or even overexcitable.

➢Excessive distention or irritation of the duodenum provides an especially strong stimulus for vomiting.The sensory signals that initiate vomiting originate mainly from the pharynx, esophagus, stomach, and upper portions of the small intestines.

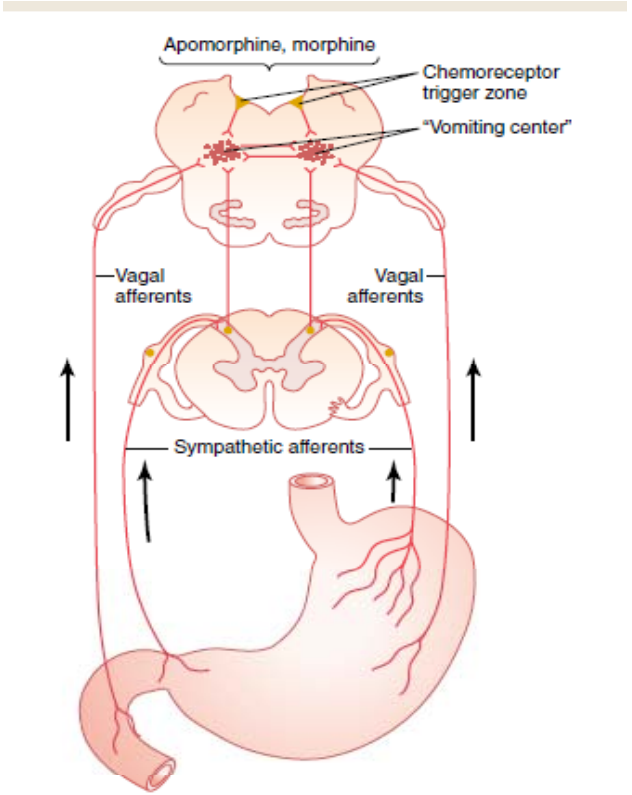
➢The nerve impulses are transmitted, by both vagal and sympathetic afferent nerve fibers to multiple distributed nuclei in the brain stem that all together are called the "vomiting center."

➢*Motor impulses that cause the actual* vomiting are transmitted from the vomiting center by way of

a. fifth, seventh, ninth, tenth, and twelfth cranial nerves to the upper GI tract
b. vagal and sympathetic nerves to the lower tract
c. spinal nerves to the diaphragm and abdominal muscles.



“Chemoreceptor Trigger Zone” in the Brain Medulla for Initiation of Vomiting by Drugs or by Motion Sickness.



➢Aside from irritative stimuli , vomiting can also be caused by nervous signals arising in areas of the brain.

➢A small area located bilaterally on the floor of the fourth ventricle called the *chemoreceptor trigger zone for vomiting.*

➢Electrical stimulation of this area can initiate vomiting; but, more important, administration of certain drugs, including apomorphine, morphine, and some digitalis derivatives, can directly stimulate this chemoreceptor trigger zone and initiate vomiting.

➢Destruction of this area blocks this type of vomiting but does not block vomiting resulting from irritative stimuli.

➢Rapidly changing direction or rhythm of motion of the body can cause vomiting. The motion stimulates receptors in the vestibular labyrinth of the inner ear, and from here impulses are transmitted mainly by way of the brain stem *vestibular nuclei into the cerebellum,* then to the chemoreceptor trigger zone, and finally to the vomiting center to cause vomiting.

Antiperistalsis, the Prelude to Vomiting.

- *Antiperistalsis (upward movement)* begins to occur often many minutes before vomiting appears.

- May begin in the ileum, traveling back up the intestine at a rate of 2 to 3 cm/sec; pushing a large share of the lower small intestine contents all the way back to the duodenum and stomach within 3 to 5 minutes.

- As the upper portions of the GI tract, especially the duodenum, become overly distended, this distention becomes the exciting factor that initiates the actual vomiting act.

Vomiting Act.

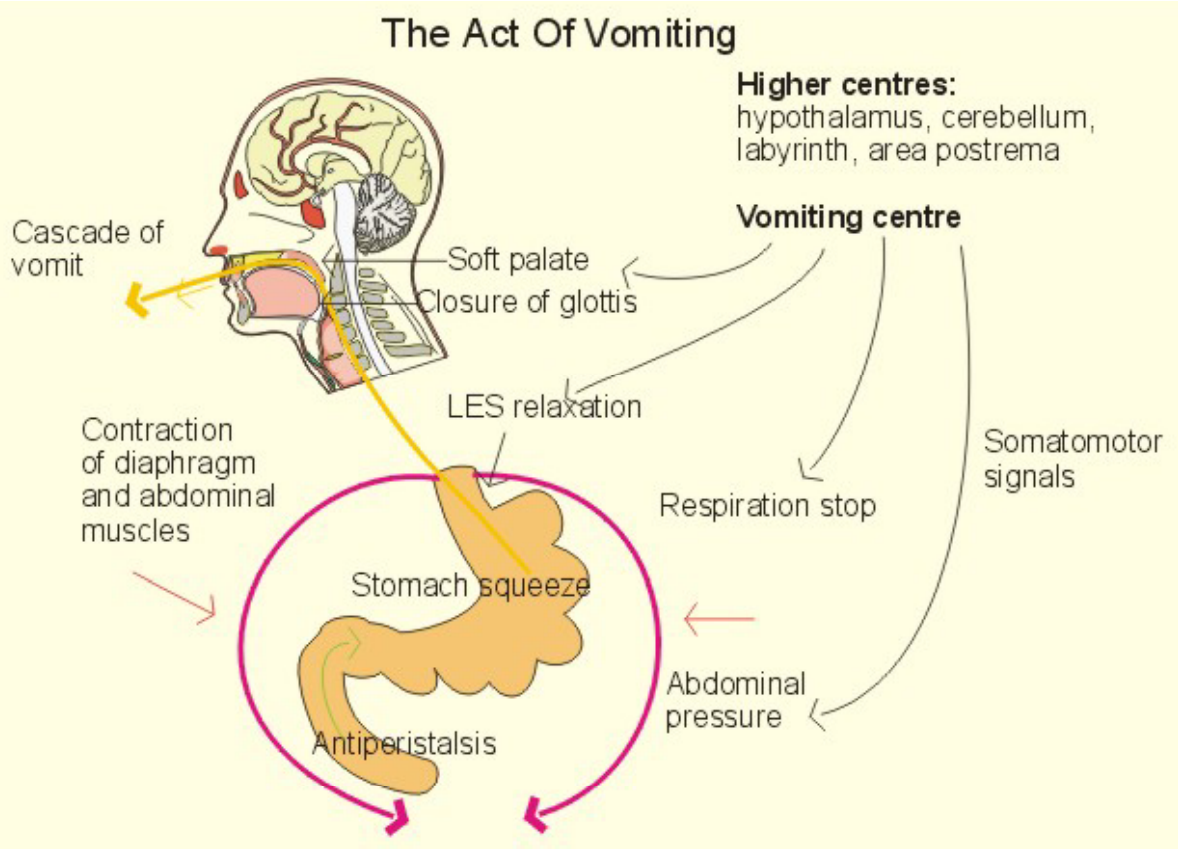
Effects of vomiting center activation

(1) a deep breath, (2) raising of the hyoid bone and larynx to pull the upper esophageal sphincter open, (3) closing of the glottis to prevent vomitus flow into the lungs, and (4) lifting of the soft palate to close the posterior nares.

(5) A strong downward contraction of the diaphragm along with simultaneous contraction of all the abdominal wall muscles.

(6) This squeezes the stomach between the diaphragm and the abdominal muscles, building the intragastric pressure to a high level.

(7) The lower esophageal sphincter relaxes completely, allowing expulsion of the gastric contents upward through the esophagus.



Nausea

Nausea is the conscious recognition of subconscious excitation in an area of the medulla closely associated with or part of the vomiting center, and it can be caused by

(1) Irritative impulses coming from the GI tract
(2) Impulses that originate in the lower brain associated with motion sickness, or
(3) Impulses from the cerebral cortex to initiate vomiting.

Vomiting occasionally occurs without the prodromal sensation of nausea, indicating that only certain portions of the vomiting center are associated with the sensation of nausea.

lactose malabsorption

- Cause: 1. lactase deficiency.

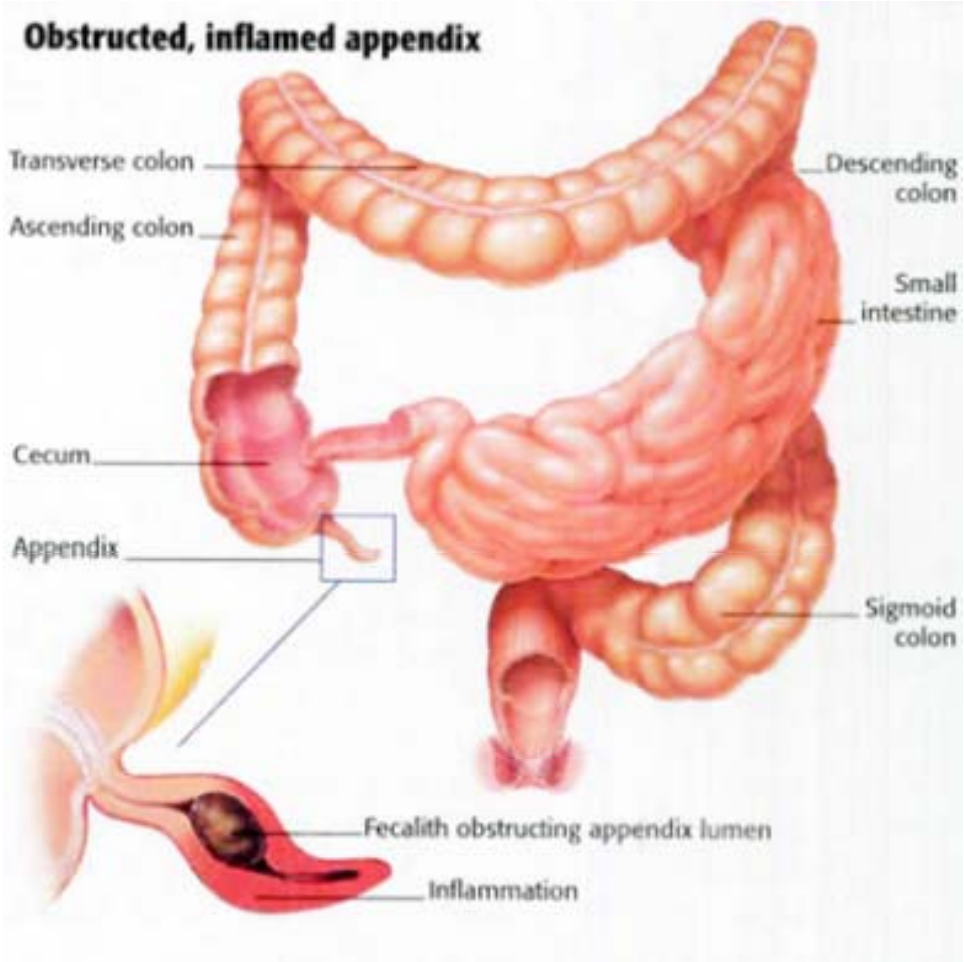
  2. secondary lactase deficiency occurs in association with small-intestinal mucosal disease with abnormalities in both structure and function of other brush border enzymes and transport processes. Secondary lactase deficiency is often seen in celiac sprue

- Symptoms: Most individuals with primary lactase deficiency do not have symptoms. Some individuals with lactose malabsorption develop symptoms such as diarrhea, abdominal pain, cramps, and/or flatus.

- Treatment: strict lactose-free diet

Appendicitis

- **A blind sac attached to the ceacum and has no known function.**
- When appendix becomes inflamed
- If it ruptures, bacteria from appendix can spread to peritoneal cavity.
- Symptoms- pain, rebound tenderness, fever, nausea, and vomiting
- Treatment - appendectomy



Paralysis of Defecation in Spinal Cord Injuries

➢Defecation is normally initiated by accumulating feces in the rectum, which causes a spinal cord–mediated *defecation reflex* ***Parasympathetic defecation reflex*** *passing from the rectum* to the *conus medullaris of the spinal cord and then back* to the descending colon, sigmoid, rectum, and anus.

➢When the spinal cord is injured somewhere between the conus medullaris and the brain, the voluntary portion of the defecation act is blocked while the basic cord reflex for defecation is still intact.

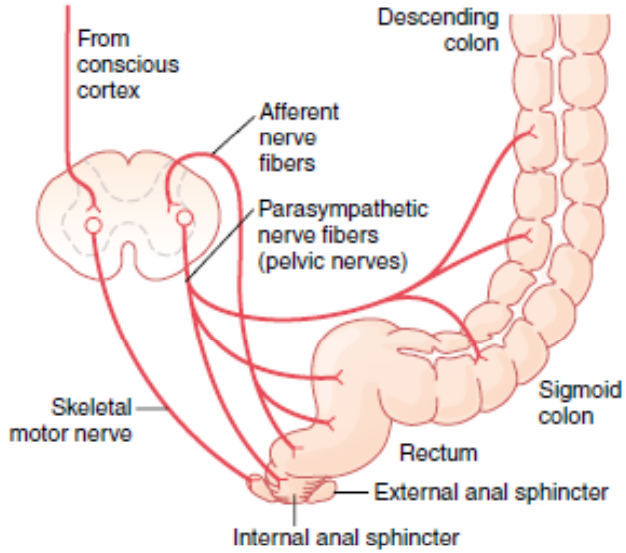


Figure 63-6 Afferent and efferent pathways of the parasympathetic mechanism for enhancing the defecation reflex.