Physiology of Gastrointestinal Disorders

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Disorders of Swallowing and the Esophagus

Paralysis of the Swallowing Mechanism.

- Damage to 5^{th}, 9^{th}, 10^{th} cerebral nerves
- Poliomyelitis /encephalitis prevent swallowing by damaging swallowing center in BS.
- Muscle dystrophy/ myasthenia gravis/botulism: Paralysis of muscles

Disorders of Swallowing and the Esophagus

- Swallowing paralysis:

1. Failure of glottis to close → food passes into lungs
2. Failure of soft palate and uvula to close posterior nares →food refluxes into nose

- Deep anesthesia > paralysis of swallowing mechanism> patients may choke to death on their own vomitus.

Achalasia and Megaesophagus

- Achalasia> lower esophageal sphincter fails to relax during swallowing> food fails to pass from esophagus to stomach.
- Damage in **myenteric plexus** in lower 2/3 of esophagus> loss of receptive relaxation
- Esophagus is enlarged >esophageal stasis> infection> ulceration >substernal pain /rupture and death.
- Rx: balloon inflated on end of swallowed esophageal tube & Antispasmodic drugs

Vomiting

- **Vomiting: ejecting** contents of upper GIT
- **Causes:** irritation/ overdistended/ overexcitation. Excessive distention or irritation of D > strong stimulus for vomiting.

Nerve impulses are transmitted by **vagal and sympathetic afferent nerve fibers** to multiple nuclei in BS, especially the **area postrema=vomiting center** →motor impulses:

✓ Fifth, seventh, ninth, tenth & twelfth cranial nerves > upper GI
✓ Vagal and sympathetic nerves to lower GI
✓ Spinal nerves to diaphragm and abdominal muscles.

❖ **Chemoreceptor trigger zone** in brain medulla for initiation of vomiting by **drugs** (apomorphine, morphine, and digitalis) or by **motion sickness** (through receptors in vestibular labyrinth-inner ear →BS vestibular nuclei →cerebellum→ chemoreceptor trigger zone→ vomiting center

Vomiting Act.

Once vomiting center has been sufficiently stimulated and vomiting act has been instituted:

(1)Deep breath

(2)Raising of hyoid bone and larynx to pull the UES open

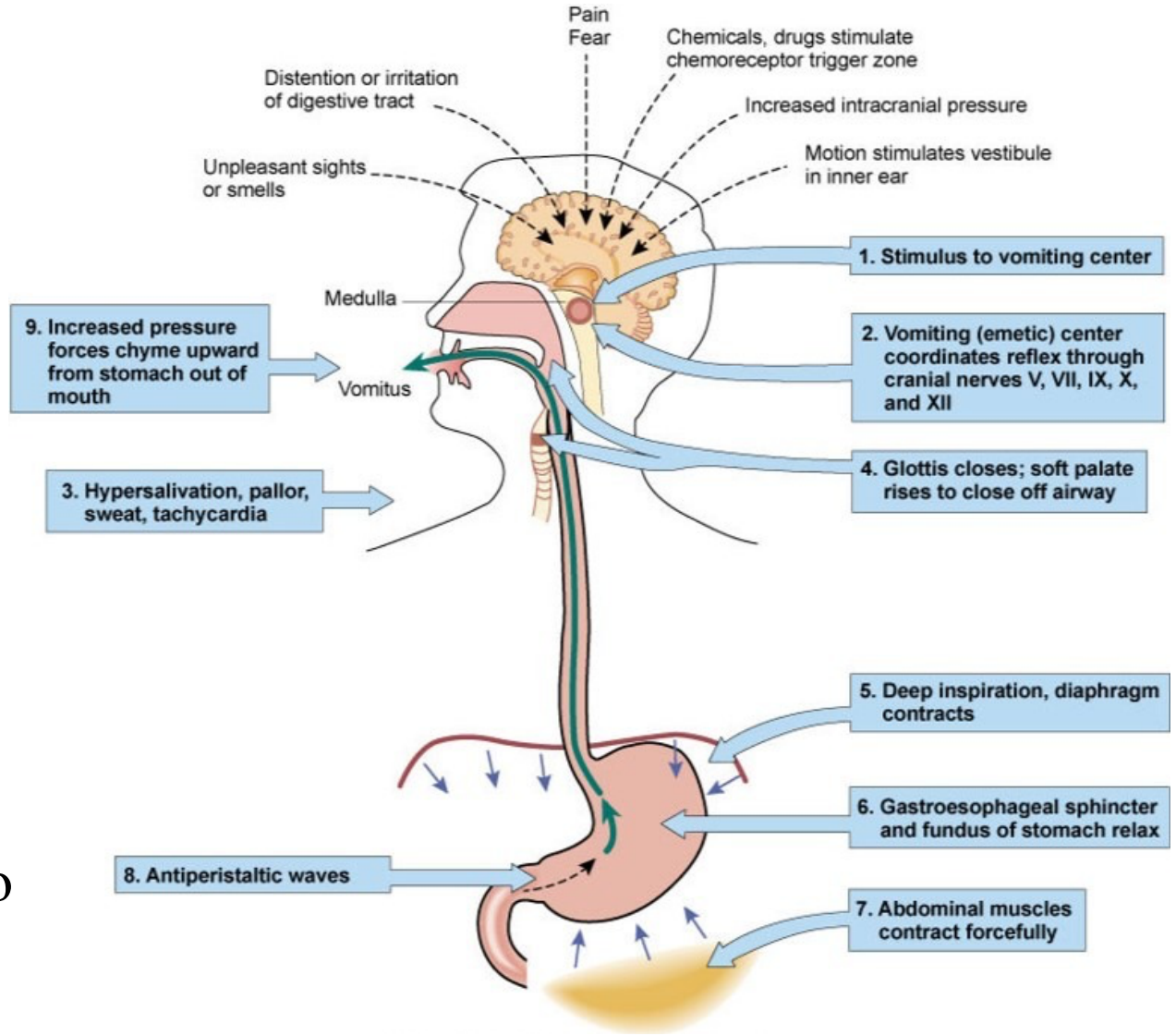
(3) Closing of glottis to prevent vomitus flow into lungs

(4)Lifting of soft palate to close posterior nares.

(5)Downward contraction of diaphragm & all abdominal wall muscles > squeezing stomach between diaphragm and abdominal muscles> building the intragastric pressure to a high level.

(6)LES relaxes > expulsion of gastric contents upward through esophagus.

sensory signals originate from pharynx, esophagus, stomach, and upper SI.



Nausea

- Prodrome of vomiting.
- excitation in an area of medulla closely associated with vomiting center.
- Causes:

✓ Irritative impulses from GI
✓ Impulses from lower brain associated with motion sickness
✓ Impulses from cerebral cortex to initiate vomiting.

❖ Vomiting occasionally occurs without nausea

Disorders of the Stomach/ Gastritis

- **Gastritis**—Inflammation of gastric Mucosa
- Mild-moderate chronic gastritis is common in population
- Superficial/deep
- Long-standing > atrophy of gastric mucosa.
- Acute and severe gastritis> ulceration by stomach's peptic secretions.

Disorders of the Stomach/ Gastritis

- Gastritis is mostly caused by **chronic bacterial infection** /less-ingested irritant substances (alcohol, smoking or aspirin)

- Chronic gastritis →gastric atrophy & loss of stomach secretions (achlorhydria-no HCl secretion, usually no pepsin & pernicious anemia-no intrinsic factor, no B12 absorpttion)

Peptic Ulcer

- Excoriation of stomach /intestinal mucosa
- Causes: digestive action of gastric juice/upper SI secretions.
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- The most frequent site is within a few cm of **pylorus**.
- Frequently occur along lesser curvature
- Rarely in the lower end of the esophagus
- Marginal ulcer >occurs wherever a surgical opening

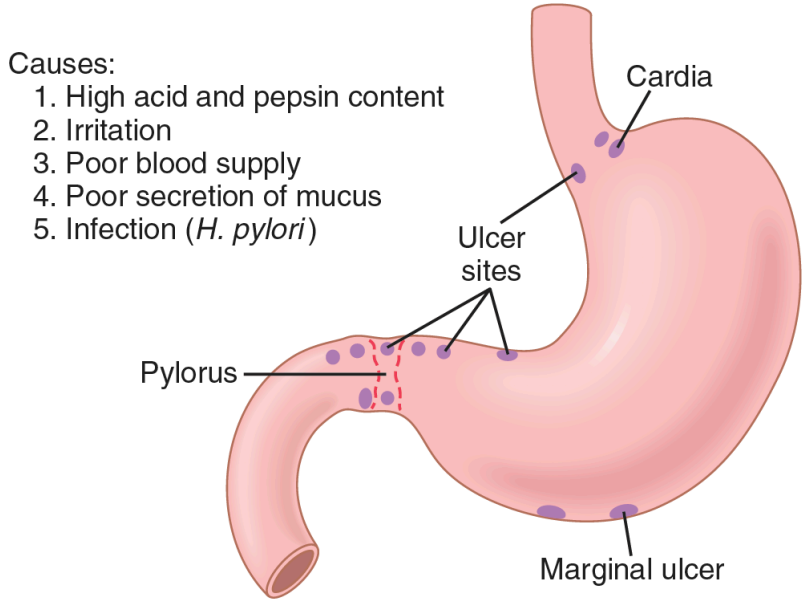

Figure 67-1. Peptic ulcer.

Cause of Peptic Ulceration

- Imbalance between rate of secretion of gastric juice & degree of protection (mucosal barrier & neutralization of gastric acid by duodenal juices)

- **H. pylori** breaks gastroduodenal mucosal barrier and stimulates gastric acid secretion.
- 75% of persons with peptic ulcers > chronic infection of terminal portions of gastric mucosa & initial portions of D mucosa by H. pylori.
- Bacterium penetrates mucosal barrier & releasing **ammonium** that liquefies barrier and stimulates secretion of HCl leading to peptic ulceration.
- Rx: antibiotics & acid suppressant drug/ surgery

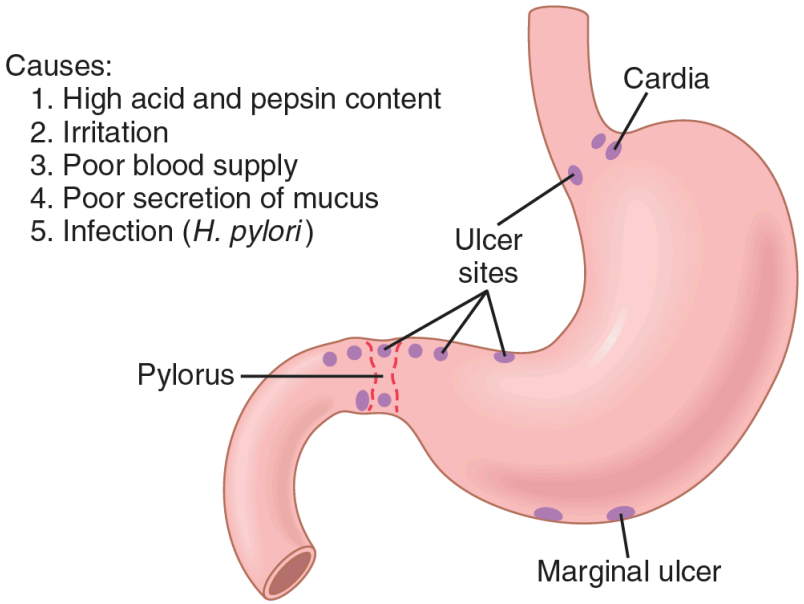

Figure 67-1. Peptic ulcer.

Pancreatic Failure

- Pancreas fails to secrete pancreatic juice into SI.
- Causes: pancreatitis /gallstone at the papilla of Vater/ malignancy.
- Loss of pancreatic juice > up to 60 % of fat may not be absorbed, 1/3-1/2 proteins and carbohydrates.
- Copious, fatty feces are excreted.

Pancreatitis

- Acute /chronic
- The **most common cause of pancreatitis is drinking excess alcohol**
- The **2nd most common cause is blockage of papilla of vater by gallstone**→pancreatic enzymes accumulate in ducts & acini → trypsinogen accumulates → overcomes trypsin inhibitor → trypsinogen activated to form trypsin → trypsin activates more trypsinogen, chymotrypsinogen & carboxypolypeptidase → enzymes digest pancreas

Malabsorption by the small intestinal mucosa/Sprue

- Causes: removal of large portions of small intestine

- **Nontropical** = idiopathic sprue/celiac disease/ gluten enteropathy

✓ Mild: Damage to microvilli

✓ Severe : Damage to villi

- **Tropical sprue**

✓ In tropics

✓ Inflammation of intestinal mucosa resulting from unidentified infectious agents.

Malabsorption

- **Fat** absorption is more impaired than other digestive products.

- Fat appears in stools in form of **salts of fatty acids** (steatorrhea)

- Impaired absorption of proteins, carbohydrates, calcium, vitamin K, folic acid, and vitamin B12 also occurs→

(1) Severe nutritional deficiency, which often results in wasting of the body
(2) Osteomalacia (demineralization of the bones because of lack of calcium)
(3) Inadequate blood coagulation caused by lack of vitamin K
(4) Macrocytic anemia of the pernicious anemia type, resulting from diminished vitamin B12 and folic acid absorption.

Disorders of LI

- **Constipation:** slow movement of feces through LI

- Large quantities, dry, hard feces in descending colon
- causes: excess absorption of fluid /insufficient fluid intake/ Obstruction (tumors/adhesions/ ulcers)

✓Spasm of sigmoid colon → constipation followed by excessive colonic secretions → diarrhea (alternating constipation & diarrhea.

- Inhibition of natural defecation reflexes/ laxatives overuse→**atonic colon**

Megacolon (Hirschsprung's Disease)

- Severe constipation
- Fecal accumulation in colon> excessive colon distention (megacolon)
- Lack of **ganglion cells in myenteric plexus in sigmoid colon** →No defecation reflexes & weak peristalsis
- Sigmoid becomes small and spastic
- Feces accumulate in ascending, transverse, and descending colons.

❖ Diarrhea

- Rapid movement of fecal matter through LI.
- **Causes: (**virus/bacteria)→↑rate of secretion & motility > washing infectious agent

❖ **Psychogenic Diarrhea**.

✓ Accompanies nervous tension

✓ Excessive stimulation of the parasympathetic nervous system (↑ mucus secretion & motility)

Appendicitis

- Inflammation of appendix
- Very common between 10 and 30 years of age.
- Causes: Bacterial/viral infection/blockage of connection between appendix and large intestine by feces, foreign body or tumor.
- If not treated immediately, the appendix may rupture and the inflammation will spread to the whole body, leading to severe complications, sometimes even death.

❖ Ulcerative Colitis

- Inflamed and ulcerated large intestine→↑ motility & secretions→diarrhea
- Cause is unknown- allergic/immune destructive effect/ chronic bacterial infection
- Hereditary tendency for susceptibility to ulcerative colitis.

❖ Paralysis of Defecation in Persons With Spinal Cord Injuries

- Spinal cord injury between conus medullaris and brain→ block voluntary defecation

- Cord reflex for defecation is still intact-enema to excite action of this cord reflex to cause defecation.

Gastrointestinal Obstruction

Causes of obstruction:

1. Cancer
2. Fibrotic constriction resulting from ulceration/ adhesions
3. Spasm of gut
4. Paralysis of gut.

<u>Consequences of obstruction depend on the point of obstruction.</u>

- ✓ Obstruction at pylorus→persistent vomiting (H+→**metabolic alkalosis**).
- ✓ Obstruction beyond stomach→antiperistaltic reflux from SI causes intestinal juices flow backward into stomach→ large amounts of water and electrolytes are lost >**little change in acid-base balance occurs.**
- ✓ Obstruction distally in large intestine → constipation, later distention of intestine → severe vomiting
- ✓ Prolonged obstruction of LI can cause rupture of intestine or dehydration and circulatory shock resulting from the severe vomiting.

Gases in the Gastrointestinal Tract (Flatus)

Sources: swallowed / gut bacterial action/ diffusion from blood to GIT.

Stomach gases: N2 & O2 from swallowed air-expelled by belching.
SI- small amounts of gas passes from stomach
LI-bacterial action generates most of gases.

Certain foods cause greater expulsion of flatus through anus—beans, cabbage, onion, cauliflower …etc. Suitable medium for gas-forming bacteria

Amount of gases entering or forming in LI/ day 7 -10 L
Average amount expelled through anus is about 0.6 L.
The remainder is normally **absorbed** into blood through the intestinal mucosa and expelled through the lungs.