LECTURE 13

GASTROINTESTINAL PATHOPHYSIOLOGY

Read through the introductory material in your textbook (Chapter 40), and skim read Chapters 41, 42, and 43 prior to beginning this lecture material. Be sure to refresh your memory on the anatomy and physiology of the GI tract and its accessory organs. You may omit the Oral Cavity from your review.

INTRODUCTION

The gastrointestinal (GI) system is specialized to process ingested particulate or liquid nutrients by reducing them to an "absorbable" size (**digestion**). This means that large, polymeric macromolecules (starches, lipids, proteins, and nucleic acids) must be reduced to their monomeric molecular subunits (e.g. – proteins to amino acids) for ease of transport across cell membranes of the GI mucosal lining. Four basic processes are necessary to complete this nutrient degradation: (1) **mechanical degradation** (i.e.- chewing and churning); (2) **liquefaction** via secretion of GI accessory glands (i.e.- salivary, hepatic, pancreatic secretions, etc.); (3) **enzymatic hydrolysis** (i.e.- amylases break starch molecules into monosaccharide sugar subunits); (4) **absorption** via extensive mucosal membrane surface of lower GI tract.

The entire GI tract is described as a "regionally specialized tube" with each region being specialized to carry out one or more of the above processes of digestion. For example, the mouth is specialized for mechanical degradation by chewing, for liquification by salivary secretions, and for limited enzymatic hydrolysis via salivary amylases. Although water and some drugs are absorbed across the oral mucosa (e.g.- nitroglycerin for angina), there is no specialization in the mouth for nutrient absorption.

Since absorbed nutrients enter hepatic portal circulation and require a sequential passage of materials through the specialized regions of the GI tract, interference with either of these activities can have both GI and cardiovascular repercussions. Certain types of bowel obstructions (e.g.- volvulus and/or adhesions) can interfere with material movement and can impair circulation to regions of the bowel. Liver damage can impair lipid digestion and cause cardiovascular portal hypertension. So, as you work through the lecture material, think of potential impact on related organ systems.

PATHOPHYSIOLOGY

Oral or Buccal Cavity pathophysiology falls within the domain of dentistry/oral medicine and will not be included here. As you read through the lecture, look up each of the topics highlighted in **blue** in your textbook to broaden your knowledge in each of these areas.

Esophagus and Stomach

The esophagus is a muscular tube connecting the laryngopharynx and the stomach cardia. The muscular component of the esophagus consists of two smooth muscle layers within the wall, one with longitudinally oriented fibers and another with circular fibers. Coordinated contractions of these two muscle layers produce peristaltic waves that move food material toward the stomach. However, when a peristaltic wave begins at the pharyngeal end of the esophagus, the cardia end must stretch to accommodate the shortening. A congenitally short esophagus or one damaged by alcohol abuse can not contract appropriately and peristaltic waves can pull a portion of the stomach cardia through the hiatus of the diaphragm, a hiatal hernia. With hiatal hernias, stomach digestive fluids are trapped in the herniated portion and are continually exposed to the esophageal lining. The esophagus does not have the extensive mucus secretion that protects the stomach lining and is subject to enzymatic erosion with resultant inflammation (esophagitis).

Although the stomach is well protected by copius mucus secretions, the stomach lining is subject to inflammation (gastritis) from a variety of sources ranging from microbial infection to alcohol abuse. A common cause of gastritis relates to excessive acid ingestion, particularly salicylic acid (asprin).

Chronic inflammation of the stomach lining can lead to ulceration, but ulcers are more common in either the duodenum or esophagus. Previously, these ulcerations of the esophagus, stomach and duodenum were thought to be the result of excessive stomach acid secretion, which exceeded the protective capacity of the mucus. Now, many of these ulcers are known to have a bacterial cause. Without intervention, ulcerations can erode completely through the GI tract wall (**perforation**) allowing septic contents into the body cavity (**coelom**).

REVIEW QUESTIONS:

What is peritonitis and how does it relate to ulcerations?

What is the risk of chronic drinkers taking asprin to treat their persistent hangovers?

Small Intestine

Unlike the stomach, the small intestine does not have a protective mucus layer and is susceptible to ulceration, especially in the duodenal region. However, certain foods, drugs, alcohol, or microbial agents can inflame any region of the small intestine

resulting in enteritis. Such inflammatory responses can be problematic. Inflammation of the intestine results in an increase in motility, causing material to move through this portion of the GI tract too rapidly for proper absorption (diarrhea). Since much of the water from intestinal contents is absorbed in the small intestine, failure of this water absorption can lead to dehydration and pH imbalances.

Since the intestine is suspended and attached to the dorsal body wall by sheets of mesentery, the small intestine is in contact with the abdominal body wall. Any weakness in this wall (e.g.- the umbilicus and inguinal canals) can allow a loop of the intestine to protrude through the weakened abdominal wall (hernia). Herniation can cause bowel obstruction and can occlude the blood supply to the herniated protion resulting in death of a portion of the intestine.

Abdominal surgery can cause problems as the surgical incision heals and forms scar tissue. If a loop of the intestine contacts the healing area, it can become attached by the forming scar tissue (adhesion). Adhesions of the small intestine can cause the unattached, motile portions on each side of the adhesion to twist and turn with normal motility movements. Such movements can cause the intestine to twist and "knot" upon itself (volvulus). A volvulus not only obstructs the bowel but can twist the mesenteric blood vessels (**strangulation**) and result in death of intestinal tissue.

Accessory Glands: Liver and Pancreas

The liver is an accessory gland of the GI tract and empties its secretion (**bile**) via the gall bladder and common bile duct into the duodenum. Bile contains bile salts to emulsify lipid materials and enhance their hydrolysis as well as bile pigments (**bilirubin and biliverdin**), the wastes that result from hemoglobin breakdown. These waste pigments normally are emptied into the small intestine for elimination with the feces and are responsible for the dark fecal coloration of normal stools.

Inflammation of the liver (hepatitis) can occur with alcohol abuse, hepatotoxic drugs, or microorganisms. Once inflamed, the liver responds like any other organ with swelling. However, swelling of the liver results in compression obstruction of the internal bile ducts preventing bile flow along them. Similarly, crystalline stones can occur in the gallbladder and obstruct the cystic or common bile duct. In any case, bile duct obstruction (**obstructive jaundice**) prevents bile from entering the duodenum as it normally would and results in "clay colored" feces from the lack of bile pigments. Instead of elimination via the feces, the blocked bile spills over into circulating blood and accumulates in light tissues giving them a yellow coloration **("yellow" jaundice).** In this case, the bile pigments are excreted by the kidneys producing an amber or darker "coffee" urine.

Although the liver has remarkable regenerative capability, chronic hepatitis can cause irreversible liver damage. In this case, normal functional liver cells are replaced by connective scar tissue, which performs none of the normal liver functions. This development of scar tissue in the liver is termed "cirrhosis" and has a number of

unfortunate consequences. The most significant of these is an increased resistance to blood flow through the liver. Under increased resistance, blood backs up in the hepatic portal vein (portal hypertension) and related tributaries.

Review hepatic portal circulation including the hepatic portal vein and its tributaries (p. 821).

As a result of portal hypertension, the spleen enlarges (splenomegaly) and varices (varicose veins) appear in the esophagus (esophageal varices). With cirrhosis, it is not liver failure that is the major cause of death, but ruptured esophageal varices. A ruptured esophageal varix results in GI bleeding and is not immediately apparent. Dangerously high blood loss can occur before the bleeding can be stopped.

Similar to hepatitis, the pancreas can also become inflamed (pancreatitis) for the same reasons. With the resultant swelling, pancreatic ducts become compressed and blocked preventing the normal flow of pancreatic secretions. Recall that pancreatic secretions contain digestive enzymes and their retention in the pancreas can result in digestion of pancreatic tissue. Such destruction of the pancreas is a major complication of chronic pancreatitis.

REVIEW QUESTIONS:

1. Based upon liver functions, explain why blood loss can be excessive with a ruptured esophageal varix.

2. What might be the possible causes of RUQ pain in an individual describing the symptom of clay colored feces.

Large Intestine: Colon and Rectum

In addition to being shorter in length than the small intestine, the colon shows a number of other specializations for function. The primary function of the colon is described as "feces formation" and is a function of water absorption. Fecal material that contains too much water is described as "unformed". That colon function involves water absorption is evident by the fact that colon regions of the intestine are best developed in terrestrial animals where water conservation is of primary importance. Except for water and certain vitamins, there is very little absorption in the colon. In fact, most water absorption occurs in the small intestine leaving the colon the function of final water recovery before undigested residues are eliminated. The first part of the colon, the cecum, bears the vermiform appendix whose function remains obscure. However, it commonly becomes inflamed (appendicitis) and requires surgical removal.

As with any other region of the GI tract, the colon is susceptible to inflammation from a variety of causes. Colon inflammation is categorically termed colitis and can appear with varying degrees of severity. The increased motility resulting from inflammation can lead to diarrhea and eventual dehydration.

Structurally, the colon shows "outpocketings" all along its length termed haustra. These haustra slow the passage of material through the short colon to facilitate water absorption. In some individuals, these haustra become enlarged to form diverticula. Possessing diverticula is termed diverticulosis. Should diverticula become inflamed, the condition is termed diverticulitis.

The distal most length of the colon is S-shaped and termed the sigmoid colon, which terminates with a highly vascularized region, the rectum. These colon regions store fecal material prior to elimination and the heavily vascularized rectum serves as the final site for water absorption. Clinically, the rectum, with its heavy vascular supply, can be utilized as an alternative route for administration of medication to unconscious or uncooperative clients. Blood congestion in the venous vessels of the rectum cause varicosities termed hemorrhoids.

REVIEW QUESTIONS:

1. What would be an anticipated consequence of surgical removal of a portion of the small or large intestine? Why?

2. What pH imbalance might occur with chronic vomiting? Why?

3. What pH imbalance might occur with chronic diarrhea? Why?

DISCUSSION QUESTIONS: (Post answers to the "Patho Discussion Group")

1. From your knowledge of liver function, predict the signs and symptoms of cirrhosis.

2. What pH compensatory mechanisms might one see with the pH imbalances resulting from chronic vomiting? chronic diarrhea?