GASTROINTESTINAL SYSTEM

Objectives:
1. Review the anatomy, physiology and pathophysiology of the GI tract.
2. Identify alternative procedures for providing nutrition.
3. List common diagnostic studies and invasive procedures related to assessments of the GI tract.
4. Discuss causes, pathophysiology, assessment, interventions, treatment and nursing care of the following:
   a. GI bleeding
   b. Pancreatitis
   c. Bowel obstruction
   d. Crohn’s disease
   e. Ulcerative colitis
   f. Hepatitis
   g. Cirrhosis
   h. Pancreatitis
   i. Gall bladder disease

Readings:

Please use the following outline, along with your readings, to complete the course objectives.

NORMAL GASTROINTESTINAL (GI) SYSTEM

I. INNERVATION
   A. Parasympathetic = excitatory
   B. Sympathetic = inhibitory
   C. Enteric = intrinsic “gut brain”; movement and secretion of GI tract
II. CIRCULATION
A. 25-30% cardiac output to gut
B. can be diverted during stress or exercise

III. PRIMARY FUNCTIONS
A. Providing body with continual supply of nutrients, electrolytes, and water
B. Appetite center in hypothalamus stimulated by empty stomach, decrease in body temperature, hypoglycemia, habit; sight, smell, taste of food
C. Digestion & Absorption
1. Mouth: mechanical and chemical digestion; saliva lubricates and softens food mass; amylase hydrolyzes starch to maltose
2. Stomach:
   a) Food: storage and mixing with gastric juices and mucin; chemical and mechanical changes
   b) Absorption: small amounts of water, glucose, alcohol, and water
   c) Secretions: pepsin (chief cells); HCl, water and intrinsic factor (parietal cells); intrinsic factor promotes absorption of B12 (extrinsic factor)
3. Small Intestine
   a) Most absorption occurs here
   b) Functional units: villi
      (1) contain goblet cells that secrete mucus and absorptive cells that absorb digested food stuffs
      (2) increase surface area of digestion
4. Large Intestine
   a) Divisions: cecum (with appendix), colon, ascending, transverse, descending, rectum, anus
   b) Most important function: absorption of water & electrolytes
5. Rectum and Anus: elimination via defecation: reflex action, voluntary and involuntary control; feces in rectum stimulates sensory nerve endings
6. Liver: portal circulatory system
   a) Functions: manufacture, storage, transformation, excretion of substances involved in metabolism
   b) Liver Function Tests: Elevated enzymes: Alkaline Phosphatase (obstructive jaundice); SGOT(liver damage); SGPT (liver damage)
7. **Biliary Tract**: Gallbladder and duct system: concentrate and store bile (holds 45 ml); bile salts needed for fat emulsification

8. **Pancreas**:
   1. Exocrine: secretes pancreatic enzymes into duodenum
   2. Endocrine: islets of Langerhans: beta cells secrete insulin; alpha cells secrete glucagon

**PROBLEMS WITH THE GI SYSTEM**

I. **MALNUTRITION**
   A. **Types**:
      1. **Primary**: result of poor eating habits
      2. **Secondary**: defect in digestion, ingestion, absorption, or metabolism
   B. **Treatment**:
      1. **Total Parenteral Nutrition (TPN)**: requires placement of a venous access device with tip in superior vena cava; increased risk if line infection; monitor blood glucose levels; monitor electrolyte levels, especially magnesium; monitor I&O, daily weights
      2. **Tube Feedings**:
         a) requires placement of naso- or oro- gastric feeding tube (short term) or gastric tube (long term; PEG (percutaneous endoscopic gastrostomy tube); keep patient’s head of bed elevated at least 30 degrees, can still aspirate
         b) monitor tolerance (check residual levels of feeding in the stomach routinely); blood glucose levels; I&O, daily weights

II. **GASTROESOPHAGEAL REFLUX DISEASE (GERD)**
   A. **Syndrome** produced by conditions resulting in reflux of gastric secretions into esophagus
   B. **Predisposing causes**: hiatal hernia, incompetent lower esophageal sphincter (LES), decreased esophageal clearance, decreased gastric emptying
   C. **Clinical manifestations**: heartburn (pyrosis), occurs following the ingestion of foods that decrease the LES pressure:
      1. **Decrease LES pressure**: fatty foods, chocolate, peppermint, ETOH, nicotine, tea, coffee, anticholinergics, Theophylline, Valium, morphine, beta and calcium channel blockers
      2. **Increase LES pressure**: Urecholine, Reglan
III. GI BLEEDING
A. Upper GI Bleeding: arterial (bright red, profuse); venous ("coffee ground", brown; has been partially digested); melena - tarry stools from a slow UGI bleed
B. Peptic Ulcer Disease: full-thickness erosion in mucosa of esophagus, stomach, or duodenum; up to 80% caused by Helicobacter pylori, and 20% caused by non-steroidal anti-inflammatory drugs (NSAIDS)

IV. INFLAMMATORY BOWEL DISEASE
A. Ulcerative Colitis: inflammation and ulceration of colon and rectum; peak ages 15-40; more women; over 10 years, increased risk for colon cancer
   1. acute fulminating crisis or mild to severe acute exacerbations
   2. major symptoms- bloody diarrhea and abdominal pain
   3. treatment: bowel rest, anti-inflammatory drugs, antibiotics, pain control, parental nutrition, surgery in 15-20%
B. Crohn’s Disease: chronic, non-specific inflammatory disorder; unknown cause; ages 15-30 years mainly; slightly more women
   1. any part of GI tract: usually terminal ileum, jejunum, colon
   2. major symptoms - abdominal pain, diarrhea, usually non-bloody
   3. Complications: intestinal scarring and strictures, impaired absorption
   4. Treatment: anti-inflammatory drugs, low residue diet, surgery at least once in course of disease; new: IV infusion of Infliximab (Remicade)

V. INTESTINAL OBSTRUCTION
A. Types:
   1. Mechanical: most common obstruction 90% (adhesions 50%, hernias 15%, neoplasms 15%); most in small intestine, in ileum
   2. Non-mechanical:
      a) neuromuscular: paralytic (non-dynamic) ileus most common and occurs to some degree post surgery; other causes include inflammatory reactions, acute pancreatitis or appendicitis, electrolyte abnormalities, thoracic/lumbar spinal fusions
      b) Vascular - interference with the blood supply to portion of the bowel by emboli or atherosclerosis
B. Pathophysiology:
1. fluid, gas, intestinal contents accumulate proximal to the obstruction causing distention, increasing pressure in bowel lumen, increasing capillary permeability with fluid and electrolytes extravasation into peritoneal cavity
2. edema, congestion, possible rupture, necrosis can occur
3. fluid retention in intestine and peritoneal cavity can lead to severe decrease in circulating blood volume which can lead to hypotension and hypovolemic shock

C. Clinical Manifestations: depends on location of bowel affected: nausea, vomiting, abdominal pain, distention, absence of flatus, obstipation, high pitched bowel sounds over area of obstruction
   1. obstruction high in small intestine: usually rapid onset with projectile vomiting of bile-containing emesis and pain relieved with vomiting.
   2. obstruction lower in small intestine: more gradual onset; orange-brown, fecal smelling emesis; persistent colicky abdominal pain

D. Treatment:
   1. intestinal decompression by removing gas and fluid with nasogastric tube placed to suction
   2. correction and maintenance of fluid and electrolyte balance
   3. relief or removal of obstruction: may need surgery

VI. HEPATITIS
A. Types:
   1. Hepatitis A: RNA virus; transmission oral-fecal route; doesn’t lead to chronic hepatitis or cirrhosis
   2. Hepatitis B: DNA virus; blood borne; parenteral, permucosal, sexual and perinatal transmission; can progress to cirrhosis, chronic hepatitis, liver cancer and death
   3. Hepatitis C: RNA virus; often progresses to chronic hepatitis, cirrhosis, and death
   4. Hepatitis D: RNA virus, need coinfection with Hepatitis B; may progress to cirrhosis and chronic hepatitis; high mortality rate
   5. Hepatitis E: RNA virus; transmitted through fecal contamination of water, primarily in developing countries, rare in U.S.; acute infection that doesn't progress to chronic hepatitis with 10% mortality rate in pregnant women
   6. Hepatitis G: RNA virus; transmitted through blood and sexually;
B. **Clinical manifestations:** fatigue, mild fever, muscle and joint aches, nausea, vomiting, diarrhea, abdominal discomfort, jaundice, color changes in urine and stool

C. **Treatment:** rest, good nutrition, proper precautions (enteric or blood borne pathogen) to keep infection from spreading; caution with over the counter meds since hepatic function slows; avoidance of alcohol 6-12 mos. or longer; instruct that cannot donate blood with any of parenterally transmitted forms of hepatitis; hepatitis A infections should be reported to the Health Department

VII. **CIRRHOSIS:** chronic progressive disease characterized by extensive degeneration and destruction of liver parenchymal cells; ETOH abuse most common cause

A. **Complications:** portal hypertension and esophageal varices; peripheral edema and ascites; hepatic encephalopathy from increased serum ammonia; hepatorenal syndrome (functional renal failure)

B. **Treatment:** rest (recovery of liver cells and decrease sodium retention); sodium restriction and diuretics (reduce ascites); paracentesis shunt (drain ascites fluid); for esophageal varices, avoid ASA, irritating foods, control coughing, may need sclerotherapy or ligation of varices; dietary protein restriction (reduce ammonia formation); Lactulose (reduce ammonia in blood)

VIII. **ACUTE PANCREATITIS:** acute inflammatory process; mild edema to severe hemorrhagic necrosis; primary etiology: biliary tract disease, alcoholism

A. **Clinical manifestations:** Abdominal pain (often severe and may be accompanied by dyspnea, flushing and cyanosis); nausea, vomiting, slight fever, leukocytosis, tachycardia, hypotension, jaundice; possible decreased or absent bowel tones, ileus, distention, respiratory crackles, ecchymoses; serum amylase over 200

B. **Treatment:** pain relief; shock prevention; NPO (decrease pancreatic enzymes); correct fluid & electrolyte imbalances, prevention/treatment of infection, removal of precipitating cause

IX. **GALLBLADDER DISEASE:** cholelithiasis (stones); cholecystitis (inflammation); more females (multiparous, over 40 years old)

A. **Clinical manifestations:** indigestion with moderate to severe pain, fever, jaundice, restlessness, diaphoresis, nausea, vomiting

B. **Treatment:** surgery (open or laparascopic cholecystectomy)