AACN CCRN Review

Gastrointestinal

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I. Introduction
A. AACN Blueprint: 6%
   - Acute abdominal trauma
   - Acute GI hemorrhage
   - Bowel infarction/obstruction/perforation (eg, mesenteric ischemia, adhesions)
   - GI surgeries
   - Hepatic failure/coma (eg, portal hypertension, cirrhosis, esophageal varices)
   - Malnutrition and malabsorption
   - Pancreatitis

B. Structures/Function/Digestion
   - Mouth
   - Esophagus
   - Stomach
   - Small intestine
   - Pancreas
   - Gallbladder
   - Liver
   - Spleen
   - Portal circulation
   - Mesentery circulation
   - Large intestine
   - Digestive hormones
   - Digestive enzymes

C. Assessment
   - Inspection
   - Auscultation
   - Palpation
   - Percussion

II. The Hepatic System
A. Liver Function
   - Metabolic factory and waste disposal plant
   - Carbohydrate, fat, and protein metabolism
   - Production of bile salts
   - Production of clotting factors
   - Bilirubin metabolism
   - Detoxification: nutrients, drugs, toxins, bacteria, everything
   - Vitamin and mineral storage
   - Blood reservoir: 10% of total blood volume

Any time the liver is not functioning normally (eg, hepatitis, cirrhosis, shock liver, hepatic cancer, biliary duct obstruction, cholecystitis, etc.) many, if not all, of these essential functions are impaired. Therefore, liver dysfunction will impact multiple bodily functions, from wound healing, coagulation, and substrate metabolism to level of consciousness
**B. Liver Function Tests (LFTs) (Normal Values)**

1. **Serum proteins**
   - Total protein: 6.0–8.0 g/dL
   - Serum albumin: 3.5–5.0 g/dL
   - Serum globulins: 2.6–4.1 g/dL

2. **Serum ammonia** 19–60 mcg/dL

3. **Bilirubin**
   - Total bilirubin: 0.1–1.2 mg/dL
   - Unconjugated bilirubin: 0.1–1.0 mg/dL
   - Conjugated bilirubin: 0.1–0.2 mg/dL

4. **Coagulation studies**
   - PT, PTT, INR, bleeding time, ACT all indirectly reflect liver function

5. **Hepatic enzymes**
   - ALP: 42–136 U/L
   - GGT: Men: 0–85 U/L; women: 0–70 U/L
   - AST: Men: 15–40 U/L; women: 13–35 U/L
   - ALT: Men: 10–55 U/L; women: 7–30 U/L

**C. Liver Dysfunction and Failure**

1. **Pathophysiology**
   - Liver tissue (cells) are destroyed and replaced with fibrotic tissue
   - Functions are altered
   - Organ changes shape
   - Vascular flow is obstructed
   - Portal hypertension

2. **Cirrhosis**: A chronic progressive liver disease where diffuse fibrotic bands of connective tissue distort the liver’s normal architecture and functional ability. The liver loses its ability to regulate fluids, metabolize waste, and regulate coagulation and nutrition
   - Causes
     - Alcoholic, Laennec's portal, or fatty
     - Postnecrotic: toxic, nodular, or posthepatic
     - Biliary: cholangitic or obstructive

3. **Hepatitis**: Widespread Inflammation of Liver Cells
   - Causes
     - Primary Viral—Most Common
     - Hepatotoxins—Toxic or Drugs
     - Secondary Viral, Low Mortality

**Hepatitis Tests**

**Serologic Tests for Hepatitis**
- Presence of virus RNA or DNA
- Presence of virus antigen(s)
- Presence of antivirus antibodies
- Presence of specific immunoglobulins
- Evidence of liver damage/failure from LFTs
**Hepatitis A:** Enteral (oral–fecal) transmission with an incubation period of 2–12 wks. Jaundice is an early symptom. The infection is usually acute and self-limiting. Vaccine available

**Tests:** Anti-HAV-IgM, Anti-HAV-IgG. IgM denotes acute phase of infection, IgG denotes recovery, past infection or immunity.

**Hepatitis B:** Parenteral (IV and sexual) transmission with an incubation period of 6–24 weeks. There are acute and chronic stages to this disease and it is the leading cause of liver carcinoma

**Tests:** HBV-DNA, HBsAg, anti-HBs, HBeAg, HbcAg, anti-HBc-IgM, Anti-HBc. HBsAg is the earliest indicator of HBV infection and is typically present for the first 12 weeks. If followed by the anti-HBs antibody indicating recovery or immunity, HBeAg appears during infection and is present in the chronic carrier state. Anti-HBe denotes recovery. The anti-HBc-IgM indicates acute infection and the anti-HBc indicates that the individual has been infected and this serum maker may be present for several years. There is a vaccine available

**Hepatitis C:** Parenteral (IV and sexual) transmission with an incubation period of 2–26 weeks. Cirrhosis due to HCV is the most common reason for liver transplantation

**Tests:** HCV-RNA, anti-HCV, ALT, liver biopsy. One half of HCV infected patients will become chronic carriers. High incidence of cirrhosis and liver cancer from HCV. No vaccine available

4. **Clinical Presentation of Liver Dysfunction**

**A. Hepatic Encephalopathy:** The liver is unable to perform its detoxification function and toxins build up. Primarily ammonia causing altered LOC, behavior and motor abilities.

- Clinical presentation
  - Confusion → coma
  - Agitation → unsafe behavior
  - Asterixis–flap-like tremor of hands
  - Apraxia–inability to perform purposeful acts
  - Elevated ammonia

- Common treatment modalities
  - Limit protein intact
  - Limit hepatotoxic drugs
  - Lactulose and neomycin
  - Safe environment

**B. Malnutrition/malabsorption:** The liver is unable to perform its function of carbohydrate, protein, and fat metabolism. This leads to malnutrition

- Clinical presentation
- Common treatment modalities
  - Need to treat the cause of liver failure
  - Parenteral nutrition
  - Limit protein intake
  - Restrict fluids

**C. Coagulopathy:** The liver is unable to synthesize fibrinogen, prothrombin and factors V, VII, IX, X, XI, XIII, fibrinolytic factors, and vitamin K. These are needed to maintain the ability to clot. Platelet aggregation and adhesion are also affected by liver dysfunction

- Clinical presentation
  - Bleeding tendencies
  - Nonspecific bleeding
- Common treatment modalities
- Monitor coagulation studies and platelet count
• Decrease bleeding and bruising risk
• Administer blood products

D. Portal Hypertension: Increased pressure in the portal vein occurs secondary to flow obstruction from inflammation, bands, or fibrotic hepatic tissue. This retrograde pressure leads to formation of varices in the esophagus, stomach and rectal vault

• Clinical Presentation
  o Caput medusae: dilated cutaneous veins radiating from the umbilical (spider angiomas) commonly seen in cirrhosis
• Upper GI bleeding - Common treatment modalities
  o Surgical shunting
  o Transjugular intrahepatic portosystemic stent shunt (TIPSS)
  o Treat bleeding
  o Treat cause

E. Hepatorenal Syndrome: A form of pre-renal failure caused by the liver dysfunction. Mortality of liver failure is very high once renal failure develops

• Clinical presentation
  o S and S of renal dysfunction
• Common treatment modalities
  o Maintain adequate renal perfusion
  o Restrict fluids
  o Restrict nephrotoxic agents
  o Continuous renal replacement therapies

F. Ascites: Fluid accumulation in the peritoneal space secondary to decreased production of albumin, decreased systemic oncotic pressure, increased hepatic lymph production, and increased capillary permeability. The fluid accumulation impacts the respiratory (diaphragm) and cardiac (hemodynamic) systems primarily, as well as comfort and body image

• Clinical presentation
  o Increase in abdominal girth
  o Hypotension and tachycardia
  o Dyspnea, orthopnea, tachypnea
  o S and S of dehydration
  o N and V
• Common treatment modalities
  o Restrict PO fluid
  o Diuretics (if tolerated hemodynamically)
  o Restrict Na
  o Respiratory support
  o Paracentesis
  o Peritoneovenous shunt surgery

G. Infection: One of the functions of the liver cells (Kupfer cells) is to clean the blood of bacteria. With liver failure this function is not provided and bacteria builds up (primarily gram negative bugs) in the systemic circulation increasing the risk of infection

• Clinical presentation
  o Poor wound healing
  o Increased risk of infection
• Common treatment modalities
  o Heightened prevention measures
III. The Pancreas

A. Function

- Endocrine functions
  - Synthesis and release of hormones: glycogen, insulin, gastrin
- Exocrine functions
  - Pancreatic enzymes break down protein, starch, and fat. >2L/day
  - Bicarbonate raise pH
- PNS, gastrin, and hormones regulate secretions

B. Pancreatic Enzymes

- Trypsin—aids in protein digestion
- Amylase—aids in carbohydrate digestion
- Lipase—aids in fat digestion

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<tr>
<th>Enzyme</th>
<th>Normal Serum Levels</th>
<th>Normal Urine Levels</th>
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<tr>
<td>Amylase</td>
<td>27–131 U/L</td>
<td>1–17 U/hr (need 24-hour urine)</td>
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<tr>
<td></td>
<td>P type: 30%–55%</td>
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<tr>
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<td>S type: 45%–70%</td>
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The enzyme amylase comes from the pancreas and the salivary glands. It is necessary to convert starch to sugar. Amylase had two isoenzymes: P type from the pancreas and S type from the salivary glands. In addition to serum levels, amylase levels can also be measured in urine, ascitic fluid, pleural effusion and saliva. Serum amylase is frequently ordered to assess acute abdominal pain and identify pancreatitis.

**Elevated Amylase:**
Acute and chronic pancreatitis, obstruction of pancreatic duct, acute cholecystitis, pancreatic cancer, alcoholism, medications that cause spasm at the sphincter of Oddi, biliary tract disease, thiazide diuretic, diagnostic dyes, DKA, renal failure, BPH, burns and trauma to the pancreas. Parotitis and mumps will cause an elevation of the total amylase from the S type increase.

**Lipase:** 20–180 U/L
Lipase is a pancreatic enzyme that is secreted into the duodenum to aid in the digestion of fat. Lipase breaks down fat into glycerol and fatty acids. Lipase only comes from the pancreas and therefore is specific to identify pancreatic disorders. Lipase elevations will occur with pancreatic cancer, acute and chronic pancreatitis, obstructions of the pancreatic duct, injury or trauma to the pancreas, acute cholecystitis and acute renal failure. Lipase will rise with amylase in pancreatic disorders but the serum lipase elevation occurs later in the course and remains elevated longer (up to 14 days after acute attack, amylase only 3 days).
C. Acute Pancreatitis

- **Pathophysiology**
  - Auto digestion
    - Tissue damage
    - Fat necrosis
    - Vascular damage and hemorrhage
    - Increased capillary permeability
    - Hypotension
  - Forms/types
    - Edematous
    - Hemorrhagic
  - Classifications
    - Acute pancreatitis
    - Recurrent acute
    - Recurrent chronic
    - Chronic pancreatitis

- **Cause** (blocked enzyme release)
  - Alcoholism
  - Biliary stones
  - Hyperlipidemia
  - Abd trauma
  - Infection (bacterial or viral)
  - Shock
  - Drugs (most common: cyclosporine, acetaminophen, cimetidine, steroids, salicylates, furosemide, thiazides, estrogens)

- **Clinical Presentation**
  - Pain
  - Low-grade fever
  - N and V
  - Distended/tender/rigid abd
  - Guarding with rebound tenderness
  - Jaundice
  - Hypoactive bowel sounds
  - Steatorrhea: bulky, pale, foul-smelling stools
  - ? ascites
  - Hypovolemic shock

- **Labs** (MOST diagnostic underlined)
  - Hypocalcemia (classic sign)
  - Low Ca, Mg, K
  - Hyperglycemia
  - Hyperbilirubinemia
  - Hypertriglyceridemia
  - Increased BUN and creatinine
  - Elevated amylase
  - Elevated lipase
  - Elevated LFTs
  - Elevated WBC
  - Decreased H/H
  - ? increased H/H
Ranson’s Criteria

- **On admission**
  - Age >55yr
  - WBC >16,000
  - Glucose >200
  - LDH >350
  - AST >250

- **Treatment Options**
  - Fluid resuscitation
  - Rest the pancreas: NPO, NGT
  - Pain management
  - Monitor and replace electrolytes
  - Tx multisystem
  - Nutritional support
  - Surgery

- **During initial 48 hr**
  - HCT dec >10%
  - BUN >5
  - Ca <8
  - PaO2 <60 mmHg
  - Base def >4mEq/L
  - Fluid seq >6L

IV. Gastrointestinal Bleeding

A. **Lower GI Bleeding**: Not Typically Life Threatening

- **Causes**
  - Diverticulitis
  - Angiodysplasia
  - Cancer
  - Hemorrhoids
  - Inflammatory bowel disease (ulcerative colitis; Crohn's disease)
  - Bowel infarction

B. **Upper GI Bleeding**

- **Causes**
  - Peptic ulcer disease: duodenal, gastric, and stomal ulcers account for 50% bleeding episodes
  - Gastritis or esophagitis
  - Esophageal varices
  - Mallory-Weiss syndrome

- **Clinical Presentation**
  - Hematemesis
  - Melona
  - PUD
  - Distended and tender abdomen
  - Hyperactive bowel sounds
  - Hypovolemia
  - Shock

- **Assessment**
  - H and H
  - Coags and platelets
  - Hemoconcentration
  - Elevated BUN
  - LFTs
  - Endoscopy
  - Angiography
  - Radionuclide scans

- **Treatment**
  - NG decompression/lavage—room temp vs iced
  - Fluid resuscitation
  - Blood product admin
  - Endoscopic sclerotherapy
  - Pharmacology
  - H₂ blockers, antacids, proton pump inhibitors
  - Sucralfate
  - Vasopressin: constricts splanchnic inflow to reduce portal pressure
- Somatostatin and octreotide: vasoconstricts splanchnic vessels to decrease blood flow
  - **Surgery**
    - Vagotomy and Pyloroplasty
    - Oversew Ulcer or Tear
    - Total and Subtotal Gastric Resection
    - Billroth I: Vagotomy, Antrectomy, Anastomosis \( \rightarrow \) Stomach and Duodenum
    - Billroth II: Vagotomy, Antrectomy, Anastomosis \( \rightarrow \) Stomach and Jejunum
    - Whipple: Removal of the Distal 3\(^{rd}\) of Stomach, Entire duodenum, Head of Pancreas, Gastrojejunostomy
    - Colon Resection
  - **Bleeding Esophageal Varices**
    - Transjugular Intrahepatic Portosystemic Stent Shunt (TIPSS)
    - Beta blocker—decreases pressure
    - Blakemore tube
    - Portal caval shunt

V. Disorders of the Bowel
A. Bowel Infarction
  - **Etiology**
    - Embolic or thrombotic occlusion
    - Typically from the superior mesenteric artery
  - **Clinical presentation**
    - Severe epigastric pain
    - Rebound tenderness
    - Guarding and rigidity
    - Stimulated sympathetic response from pain
  - **Treatment Options**
    - Angiography to identify/confirm occlusion
    - Surgery to remove occlusion and dead bowel

B. Bowel Obstruction
  - **Etiology**
    - Internal lumen obstruction ex. tumor
    - External lumen obstruction ex. adhesions
    - Emboli: no blood flow
    - Paralytic ileus

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<td>Strangulated: Obstruction with diminished blood flow</td>
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<td>Incarcerated, Volvulus, Herniated: Intestinal loops over itself creating a closed off section</td>
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Clinical presentation
- Complete vs partial
- Distended edematous bowel
- Fluid and electrolytes leaking from bowel
- Elevated WBC
- Fever
- Small intestine
  - Acute pain with sudden onset
  - N and V (movement on both ends)
  - Wave-like hyperactive high-pitched bowel sounds
  - May have some gas or feces
  - Distention (mild)
- Large intestine
  - Slow onset pain progression mild → severe, lower abd
  - No N and V (nothing moving)
  - No stool
  - Low-pitched bowel sounds
  - Distention (large amount)

Treatment Options
- Diagnosis obstruction by Hx, x-ray, CT, upper or lower barium radiology tests
- Pain management
- IV fluids
- Decompress w NG, rectal or intestinal tube
- Antibiotics
- NPO and time (rest the bowel)
- Surgery

C. Perforation/Peritonitis

Etiology
- Gastric/intestinal contents leak into peritoneal cavity
- Ulcer perforation
- Diverticular rupture
- Trauma
- Bowel infarction

Clinical presentation
- Infection/sepsis (all the S and S)
- Sudden onset of severe pain
- Rigid abdomen w rebound tenderness
- Hypoactive bowel sounds → no bowel sounds

Treatment options
- Surgery to repair cause and clean-up
- Antibiotics
- Fluids
- Tx of sepsis
- Tx of MODS

VI. GI Surgeries

A. Types
- Ex lap with Lysis of adhesions
- Colon resection
- Colostomy vs ileostomy
• Esophagogastrectomy
• Gastric bypass
• Splenectomy
• Appendectomy

B. Care Concerns
• Infection—leaks
• Sepsis
• Third spacing/hypovolemia
• Bleeding
• Electrolyte imbalance
• Nutrition
• Immobility
• Pain
• Potential for respiratory compromise

VII. Abdominal Trauma
A. Mechanism of Injury
• Blunt trauma
  o MVC
  o Falls
  o Assaults
  o Crush
  o Sports
• Penetrating trauma
  o GSW
  o Stabbings
  o Impalements

B. Types of Injuries
• Organ contusions
• Organ laceration
• Spleen common site of injury
• Solid organs vs hollow organs
• Crush w tissue damage
• Vascular injury
• Hypoperfusion
• Hemorrhage

C. Assessment
• Abd exam
• Pain/tenderness
• Firmness
• Discoloration
• Bowel sounds
• Abd sonogram
• CT
• Diagnostic peritoneal lavage
• Labs
• X-ray
• Cullen’s sign—hemorrhagic patches (bruising) around the umbilicus (pancreatitis, GI hemorrhage, ruptured ectopic pregnancy)
• Grey Turner’s sign—bruising around the flank area (hemorrhagic pancreatitis, retroperitoneal bleeding)
• Kehr’s sign—left shoulder pain from irritation to the diaphragm from blood as a result of splenic rupture. Best elicited with pt lying flat or in Trendelenburg’s position
• Abdominal compartment syndrome

D. Treatment
• Fluid resuscitation
• Diagnose problem
• Plug holes and/or repair lacerations
• Support damaged organ(s)
• Remove damaged tissue/organ(s)
• Post-treatment concerns
  o Infection/sepsis
  o Hemodynamic status
  o Organ function
  o ARDS, ATN, MODS

VIII. Summary