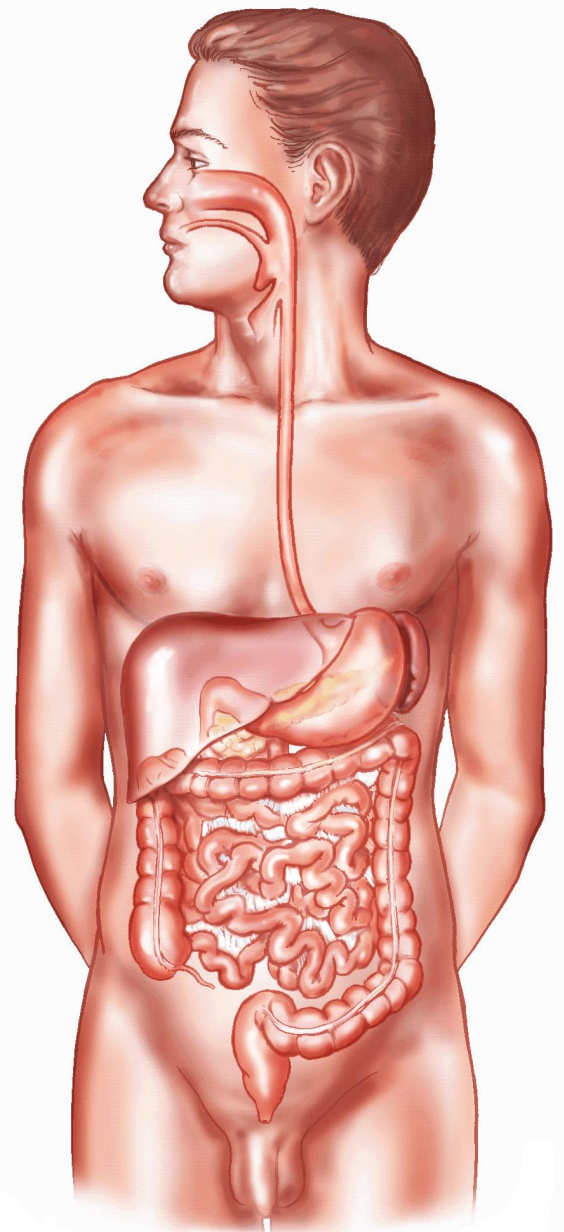
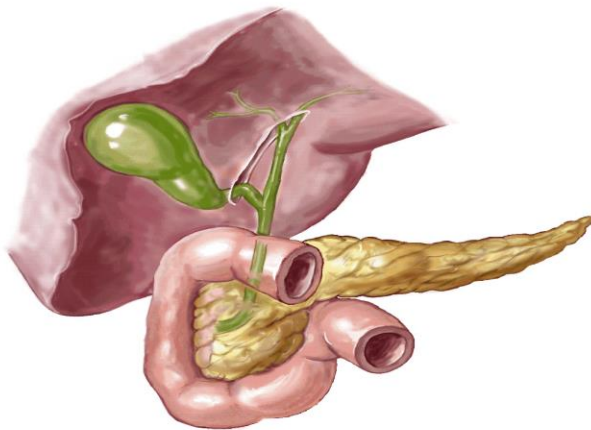
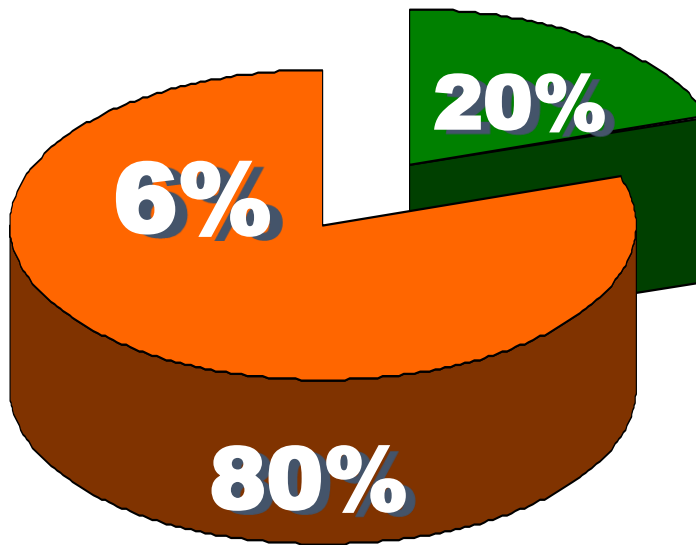


# Gastrointestinal



# Gastrointestinal

## I. Introduction

### A. AACN – CCRN/CCRN-E Blueprint: 6%

- Acute Abdominal Trauma
- Acute GI Hemorrhage
- Bowel Infarction/Obstruction/
- GI Surgeries
- Hepatic Failure/Coma (e.g. portal hypertension, cirrhosis, esophageal varicies)
- Malnutrition and Malabsorption
- Pancreatitis

#### Structures/Function/Digestion

- Mouth
- Esophagus
- Stomach
- Small Intestine
- Pancreas
- Gallbladder
- Liver
- Spleen
- Portal Circulation
- Mesentery Circulation
- Large Intestine
- Digestive Hormones
- Digestive Enzymes

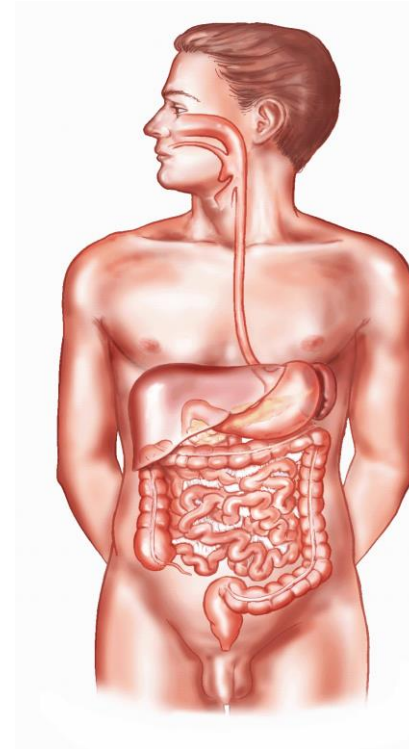
### B. Assessment

- Inspection
- Auscultation
- Percussion
- Palpation

## II. The Hepatic System

### A. Liver Function

- Metabolic Factory & Waste Disposal Plant
- Carbohydrate, Fat & Protein Metabolism
- Production of Bile Salts



- Production of Clotting Factors
- Bilirubin Metabolism
- Detoxification: Nutrients, Drugs, Toxins, Bacteria, Everything
- Vitamin & Mineral Storage:
- Blood Reservoir: 10% of Total Blood Volume

Any time the liver is not functioning normally (hepatitis, cirrhosis, shock liver, hepatic cancer, biliary duct obstruction, cholecystitis, ect...) many, if not all, of these essential functions are impaired. Therefore liver dysfunction will impact multiple bodily functions from wound healing, coagulation, substrate metabolism to level of consciousness.

## B. Liver Function Tests

1. **Serum Proteins:**      **Total Protein: 6.0 – 8.0 g/dL**  
    **Serum Albumin: 3.5 – 5.0 g/dL**  
    **Serum Globulins: 2.6 – 4.1g/dL**

Serum proteins include albumin and globulins. There are five globulins and five different types of gamma globulins. The globulin proteins play a major role in the immune system. Therefore liver dysfunction will impact the immune system. Changes in the total protein may reflect changes in albumin or the globulins. Increased globulins are seen in chronic inflammatory diseases, neoplastic diseases and biliary obstruction.

**Serum Albumin:** Albumin is synthesized by the liver. It is essential in maintaining vascular fluid volume by regulating the serum colloid osmotic pressure. It also transports and circulates many substances in the bloodstream (drugs, lipids, and hormones), functions as a buffer in the acid-base balance and impacts serum Ca<sup>+</sup> levels.

**Hypoalbuminemia:** Liver failure is a common reason for low albumin because albumin is synthesized by the liver. Other reasons include cirrhosis, AIDS, renal dysfunction, nephritic syndrome, severe burns, ulcerative colitis, prolonged immobilization, medications (penicillin, aspirin and Vit. C), CHF and inadequate protein intake (with severe malnutrition – late).

**Hyperalbuminemia:** Not reflective of liver function.  
Reflective of a dehydrated state from vomiting or diarrhea.  
Heparin administration may also elevate the albumin level.

**Pre-Albumin:** 17 – 40 mg/dL

Albumin has a half-life of up to 24 days while pre albumin has a half-life of only 2 – 4 days. It is a more sensitive measure in nutrition assessment when looking at liver dysfunction and changes in catabolism. **Decreased levels** are seen in protein-wasting diseases, malnutrition, inflammation, cancer and cirrhosis. **Elevated levels** are seen in end-stage renal disease, Hodgkin's disease and with steroid, and NSAID administration.

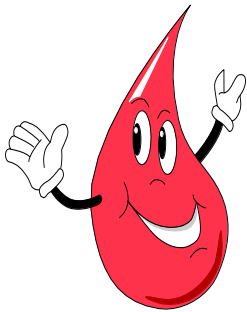
**2. Serum Ammonia:** 19 – 60 mcg/dL

Ammonia (NH<sub>3</sub>) is a waste by product of protein metabolism. The liver converts NH<sub>3</sub> to urea, which is then excreted by the kidneys. In liver damage/failure the conversion of NH<sub>3</sub> to urea does not take place and NH<sub>3</sub> levels build in the blood. Other reasons for elevated ammonia levels include Reye's syndrome, cor pulmonale, CHF and in patients with a portal-caval shunt. Elevations in Ammonia will lead to hepatic encephalopathy and coma. Treatment options include limiting the protein intake and the administration of an ammonia detoxicant (lactulose). Potassium levels should be monitored closely when the NH<sub>3</sub> levels are high. The body is less able to handle NH<sub>3</sub> during hypokalemic states.

**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**

Bilirubin is the waste product that is formed when red blood cells are broken down by the reticuloendothelial system. Bilirubin cannot be excreted in its lipid-soluble form so it is brought to the liver by albumin where it is conjugated and made water-soluble. Old terms for conjugated and unconjugated bilirubin were direct and indirect bilirubin. The conjugated Bilirubin is carried with the bile salts through the common bile duct to the intestines. In the intestines the bacteria convert the Bilirubin to urobilinogen. The majority of urobilinogen is excreted in the stool with a very small amount in the

urine. Measuring urine and fecal levels of urobilinogen are some times necessary.



**Total Bilirubin:**

Measurement of both types. If normal no need to evaluate any further. Will increase and decrease for the reasons listed below

**Unconjugated Bilirubin:**

An elevated unconjugated (indirect) Bilirubin reflects increased levels of RBC waste products in the circulation. Conditions that cause hemolysis will cause an increase: sickle cell disease, autoimmune diseases, hemorrhage, medications, a transfusion reaction, pernicious anemia, malaria, sepsis, and physical stress. The other reason for an elevation is liver dysfunction and the inability to convert to bilirubin. Crigler-Najjar syndrome is the deficiency of the enzyme glucuronyl transferase that is necessary to conjugate bilirubin in the liver. Gilbert's syndrome is associated with decrease in enzyme activity.

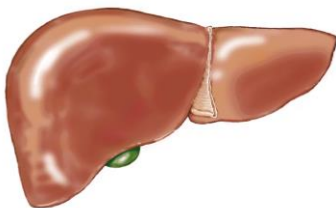
**Conjugated Bilirubin:**

Normally the circulating conjugated (direct) bilirubin is very small because once the bilirubin is conjugated it is transported to the intestines, converted and excreted. The most common reason to see an elevation is with bile flow obstructive problems. Gallstones, obstructive jaundice, cancer, hepatitis, cirrhosis, and infectious mononucleosis will all cause an elevation as will a variety of drugs.

**4. Coagulation Studies**

PT, PTT, INR, Bleeding Time, ACT all indirectly reflect liver function.

**5. Hepatic Enzymes:**



<b>ALP</b>	<b>42 – 136U/L</b>
<b>GGT</b>	<b>Men: 0–85 U/L Women: 0-70 U/L</b>
<b>AST</b>	<b>Men: 15-40 U/L Women: 13-35U/L</b>
<b>ALT</b>	<b>Men: 10-55U/L Women: 7-30U/L</b>

Enzyme elevation occurs in hepatic dysfunction for the same reason it occurs in cardiac ischemia. The enzymes, which are typically intracellular, are allowed to leak out when the cell is ischemic or damaged.

**Alkaline Phosphatase: ALP            42 – 136 U/L**

Alkaline Phosphatase (ALP) is an enzyme produced mainly in the liver and bone and to a lesser degree in the kidneys, intestines, and placenta. It is helpful in evaluating liver and bone disorders. Isoenzymes can be measured to distinguish between liver and bone problems but are rarely done. The liver pathologies that will elevate the ALP are tissue damage, bile flow obstruction, spasm of the sphincter of Oddi, bile duct stones, pancreatic cancer, and certain drugs. ALP levels are commonly done as a cancer-screening test. A decrease in ALP is seen with hypothyroidism, celiac disease, CF, chronic nephritis, scurvy and malnutrition.

**Gamma Glutamyl Transferase:**

**GGT:Men: 0–85 U/L Women: 0-70 U/L**

Gamma Glutamyl Transferase (GGT) is an enzyme used in amino acid transport and found mainly in the liver and kidney and to a lesser degree in the spleen, prostate gland and heart. If the ALP is elevated the GGT can be evaluated to determine if the elevation is from the liver or bone. Increases in the GGT are seen in liver disease, hepatic cancer, alcohol abuse, biliary obstruction, infectious mononucleosis, acute cholecystitis, acute pancreatitis and hepatitis. Elevations can also be seen in CHF and on the fourth day after a MI.

**Aspartate Aminotransferase:**

**AST Men: 15-40 U/L Women: 13-35 U/L**

Aspartate Aminotransferase (AST), formerly named Serum Glutamic Oxaloacetic Transaminase (SGOT), is an enzyme that is important in energy transformation. It is found in the liver, heart and skeletal muscle. The AST will rise in liver necrosis before many other signs of failure (even jaundice). It will rise in hepatitis, shock, cirrhosis, Reye's syndrome, MI and skeletal muscle damage or trauma. Many drugs will increase the AST. The AST:ALT ratio is useful in differentiating various types of liver disorders.

### *Clinical Pearl*

When the **AST** > **ALT** cirrhosis and metastatic cancer may be present in the liver.  
When the **AST** < **ALT** hepatitis, nonmalignant obstruction may be present in the liver.

#### **Alanine Aminotransferase (ALT)**

**ALT Men: 10-55 U/L Women: 7-30 U/L**

Alanine Aminotransferase (ALT), formerly named Serum Glutamic Pyruvic Transaminase (SGPT), is an enzyme that is important in energy transformation. It is found in the liver, heart and skeletal muscle.

#### **C. Liver Dysfunction & 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, regulate coagulation and nutrition.

- Causes
  - Alcoholic, Laennec's Portal, or Fatty
  - Post Necrotic: Toxic, Nodular, or Post Hepatic
  - 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 anti-virus antibodies
- Presence of specific immunoglobins
- Evidence of liver damage/failure from LFTs

**Hepatitis A:** Enteral (oral-fecal) transmission with an incubation period of 2-12wks. 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 & 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. It is 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 & 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 & Neomycin
    - Safe Environment
  
- b. **Malnutrition:** 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 tx 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 Vit. K. These are needed to maintain the ability to clot. Platelet aggregation and adhesion are also effected by liver dysfunction.
  - Clinical Presentation
    - Bleeding Tendencies
    - Nonspecific Bleeding
  - Common Treatment Modalities
    - Monitor Coagulation Studies & Platelet Ct
    - 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

- Caput Medusae: dilated cutaneous veins radiating from the umbilical (spider angiomas) commonly seen in Cirrhosis
    - Upper GI Bleeding
  - Common Treatment Modalities
    - Surgical Shunting
    - TIPSS - Transjugular Intrahepatic Portosystemic Stent Shunt
    - Treat Bleeding
    - 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
    - S&S of Renal Dysfunction
  - Common Treatment Modalities
    - Maintain Adequate Renal Perfusion
    - Restrict Fluids
    - Restrict Nephrotoxic Agents
    - 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
    - Inc. Abdominal Girth
    - Hypotension and Tachycardia
    - Dyspnea, Orthopnea, Tachypnea
    - S&S of Dehydration
    - N&V
  - Common Treatment Modalities
    - Restrict PO Fluid
    - Diuretics (if tolerated hemodynamically)
    - Restrict Na
    - Respiratory Support
    - Paracentesis

## ■ Peritoneovenous Shunt Surgery

- g. **Infection:** one of the functions of the liver cells (Kupffer 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
    - Poor Wound Healing
    - Increased Risk of Infection
  - Common Treatment Modalities
    - Heightened Prevention Measures
    - Abx Therapy – w Caution

### III. The Pancreas

#### A. Function

- Endocrine Functions
  - Synthesis & Release of Hormones: Glycogen, Insulin, Gastrin
- Exocrine Functions
  - Pancreatic Enzymes Break Down Protein, Starch & Fat. > 2L/day
  - Bicarbonate Raise pH
- PNS, Gastrin & Hormones Regulate Secretions

#### B. Pancreatic Enzymes

- Trypsin: Aids in Protein Digestion
- Amylase: Aids in Carbohydrate Digestion
- Lipase: Aids in Fat Digestion

**Amylase: Serum: 27 – 131 U/L**  
**P type: 30 – 55%**  
**S type: 45 – 70%**  
**Urine: 1 – 17 U/hr** (need 24 hr urine)

The enzyme amylase comes from the pancreas, the salivary glands. It is necessary to convert starch to sugar. Amylase has 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 & 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&V
- Distended/Tender/Rigid Abd
- Guarding with Rebound Tenderness
- Jaundice
- Hypoactive Bowel Sounds
- Steatorrhea: bulky, pale, foul-smelling stools
- ? Ascites
- Hypovolemic Shock

**Necrotizing Pancreatitis**

Cullen's Sign: Bluish Discoloration umbilical  
 Grey Turner's Sign: Bluish Discoloration Flank

❑ Labs (MOST diagnostic underlined)

- |                                      |                           |
|--------------------------------------|---------------------------|
| ■ <u>Hypocalcemia</u> (classic sign) | ■ <u>Elevated Amylase</u> |
| ■ Low Ca, Mg, K                      | ■ <u>Elevated Lipase</u>  |
| ■ <u>Hyperglycemia</u>               | ■ Elevated LFTs           |
| ■ Hyperbilirubinemia                 | ■ <u>Elevated WBC</u>     |
| ■ Hypertriglyceridemia               | ■ Decreased H/H           |
| ■ Increased BUN & Creatinine         | ■ ? Increased H/H         |

❑ Ranson's Criteria

- |                              |                                      |
|------------------------------|--------------------------------------|
| ■ <b><u>On Admission</u></b> | ■ <b><u>During Initial 48 hr</u></b> |
| ■ Age > 55yr                 | ■ HCT Dec > 10%                      |
| ■ WBC > 16,000               | ■ BUN > 5                            |
| ■ Glucose > 200              | ■ Ca < 8                             |
| ■ LDH > 350                  | ■ PaO <sub>2</sub> < 60mmHg          |
| ■ AST > 250                  | ■ Base Def > 4mEq/L                  |
|                              | ■ Fluid Seq. > 6L                    |

- ❑ Treatment Options
  - Fluid Resuscitation
  - Rest the Pancreas: NPO, NGT
  - Pain Management
  - Monitor & Replace Electrolytes
  - Tx Multisystem
  - Nutritional Support
  - Surgery

## 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 & Tender Bbd
  - Hyperactive Bowel Sounds
- Hypovolemia
- Shock

- ❑ Assessment

- H & H
- Coags & Platelets
- Hemoconcentration
- Elevated BUN
- LFTs
- Endoscopy
- Angiography
- Raionuclide 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 & 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 → Stomach and Duodenum
  - ✓ Billroth II: Vagotomy, Antrectomy, Anastomosis → Stomach and Jejunum
  - ✓ Whipple: Removal of the Distal 3<sup>rd</sup> of Stomach, Entire duodenum, Head of Pancreas, Gastrojejunotomy
  - ✓ Colon Resection
  
- Bleeding Esophageal Varices
  - ✓ TIPSS: Transjugular Intrahepatic Portosystemic Stent Shunt
  - ✓ 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 & Rigidity
- Stimulated Sympathetic Response from Pain

- ❑ Treatment Options
  - Angiography to Identify/Confirm Occlusion
  - Surgery to Remove Occlusion & Dead Bowel

**B. Bowel Obstruction**

- ❑ Etiology
  - Internal Lumen Obstruction ex. Tumor
  - External Lumen Obstruction ex. Adhesions
  - Emboli: no blood flow
  - Paralytic Ileus

Terms

Strangulated: Obstruction with diminished blood flow

Incarcerated, Volvulus, Herniated: Intestinal loops over itself creating a closed off section.

- ❑ Clinical Presentation
  - Complete vs Partial
  - Distended Edematous Bowel
  - Fluid and Electrolytes Leaking from Bowel
  - Elevated WBC
  - Fever
  - Small Intestine
    - ✓ Acute Pain w Sudden Onset
    - ✓ N & 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 & 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
- Abx
- 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&S)
- Sudden Onset of Severe Pain
- Rigid Abdomen w Rebound Tenderness
- Hypoactive Bowel Sounds → No Bowel Sounds

Treatment Options

- Surgery to Repair Cause & Clean Up
- ABX
- Fluids
- Tx of Sepsis
- Tx of MODS

**VI. Malnutrition – Intake is inadequate to meet current demands of the body.**

Causes

- Decreased Intake
- Increased Losses
- Increased Needs (Increased Metabolic Demands)

Clinical Presentation

- Nausea
- Vomiting
- Diarrhea
- Anorexia
- Inability to Consume Food
- Inability to Digest Nutrients
- Chronic Diseases

Assessment

- Weight Loss (20% considered malnutrition)
- Muscle Wasting
- Peripheral Edema
- Ascites
- Poor Wound Healing
- Increased Infections
- MODS
- Agitation/Irritability
- Fatigue/Apathy

- Treatment
  - Oral Feedings
  - Enteral Feedings
    - ✓ Nasogastric
    - ✓ Nasoduodenal
    - ✓ Nasodjejunal
    - ✓ PEG - Percutaneous Endoscopic Gastrostomy
    - ✓ PEJ - Percutaneous Endoscopic Jejunostomy
  - Parenteral Feedings
  - Treatment of Primary Cause of Malnutrition

## VII. GI Surgeries

### A. Types

- Ex lap with Lysis of Adhesions
- Colon Resection
- Colostomy vs Ileostomy
- Esophago-Gastrectomy
- Gastric Bypass
- Splenectomy
- Appendectomy

### B. Care Concerns

- Infection - Leaks
- Sepsis
- Third Spacing/Hypovolemia
- Bleeding
- Electrolyte Imbalance
- Nutrition
- Immobility
- Pain
- Potential for Respiratory Compromise

## VIII. Abdominal Trauma

### A. Mechanism of Injury

- Blunt Trauma
  - MVC
  - Falls
  - Assaults
  - Crush

- Sports
- Penetrating Trauma
  - GSW
  - Stabbings
  - 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(ACS): increased abdominal compartment pressures due to shock/ischemia/hypoperfusion. Bladder pressures are measured to reflect abd compartment pressures. Normal bladder pressure is 0-5mmHg. Intra-abdominal Hypertension (IAH) is sustained /repeated pressures of > 12mmHg

- Grade I 12-15 mmHg
- Grade II 16-20 mmHg
- Grade III 21-25mmHg
- Grade IV  $\geq$  25mmHg
- APP = MAP – IAH goal to maintain  $\geq$  60mmHg  
Abdominal perfusion pressure

#### **D. Treatment**

- Fluid Resuscitation
- Diagnose Problem
- Plug Holes and/or Repair Lacerations
- Support Damaged Organ(s)
- Remove Damaged Tissue/Organ(s)
- Post Tx Concerns
  - ✓ Infection/Sepsis
  - ✓ Hemodynamic Status
  - ✓ Organ Function
  - ✓ ALI, ATN, MODS

### **IX. Gastro-Esophageal Reflux – Self review**

- Etiology
  - Inappropriate relaxation of the lower esophageal sphincter (LES) – actual cause is unknown or gastric volume or intraabdominal pressure is increased.
  - Gastric and Duodenal Contents Moves Back into Distal Esophagus
  - Frequent Episodes Cause Esophageal Inflammation, Hyperemia and Erosion
  - Barrett’s Epithelium – Changes to The Tissue
- Clinical Presentation (20min → 2hr Post Eating)
  - Heartburn – Frequently Confused with Cardiac Pain
  - Regurgitation
  - Reflex Hypersecretion (mostly water)
  - Belching or Flatulence
  - Dysphagia or Odynophgia (difficulty to painful swallowing)
  - Nocturnal Cough, Wheezing, Hoarseness
- Treatment Options
  - Medications

- ✓ Antacids
- ✓ Histamine Receptor Antagonists
- ✓ Proton Pump Inhibitors
- ✓ Prokinetic Drugs (inappropriate for long term use secondary to side effects)
  
- Life Style Changes
  - ✓ Loss Weight
  - ✓ Avoid Over Eating
  - ✓ Maintain Upright Position Post Eating
  - ✓ Restrict use of Constrictive clothing
  - ✓ Restrict Heavy Lifting
  - ✓ Sleep on an Incline
  
- Diet
  - ✓ Small Frequent Meals
  - ✓ Avoid Foods that Increase Incidence of Reflux (fatty foods, cola, coffee, tea, chocolate, onions, tomato-based products, alcohol, spicy foods)
  - ✓ Adequate Protein Intake
  - ✓ Avoid Eating 2hr Before Bedtime
  
- Surgery (limited to severe cases)
  - ✓ Fundoplication: wrapping and suturing of the gastric fundus around the esophagus

## **X. Summary**