Gastrointestinal

Presenter: Carol Rauen, RN, MS, PCCN, CCRN, CCNS, CEN
I. Introduction
A. AACN Blueprint: 6%
   - Acute Abdominal Trauma
   - Acute GI Hemorrhage
   - Bowel Infarction/Obstruction/Perforation (e.g. mesenteric ischemia, adhesions)
   - GI Surgeries
   - Hepatic Failure/Coma (e.g. portal hypertension, cirrhosis, esophageal varicies)
   - 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 (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 – 4.1 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, 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 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. It 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 $\rightarrow$ Coma
     - Agitation $\rightarrow$ 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 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 and 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**
  - SandS 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
  - SandS of Dehydration
  - NandV
- **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 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

### Amylase

<table>
<thead>
<tr>
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<th>Serum: 27 – 131 U/L</th>
<th>P type: 30 – 55%</th>
<th>S type: 45 – 70%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine</td>
<td>1 – 17 U/hr (need 24 hr urine)</td>
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The enzyme amylase comes from the pancreas, 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, Cimetadine, Steroids, Salicylates, Furosemide, Thiazides, Estrogens)

- Clinical Presentation
  - Pain
  - Low Grade Fever
  - NandV
  - 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

- Ranson’s Criteria
  - On Admission
    - Age > 55yr
    - WBC > 16,000

  - Elevated Amylase
  - Elevated Lipase
  - Elevated LFTs
  - Elevated WBC
  - Decreased H/H
  - ? Increased H/H

  - During Initial 48 hr
    - HCT Dec > 10%
    - BUN > 5
    - Ca < 8
    - PaO2 < 60mmHg
    - Base Def > 4mEq/L
    - Fluid Seq. > 6L

- Necrotizing Pancreatitis
  Cullen’s Sign:
    - Bluish Discoloration Umbilical
  Grey Turner’s Sign:
    - Bluish Discoloration Flanks
• 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

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 Bbd
  • Hyperactive Bowel Sounds
  • Hypovolemia
  • Shock

• Assessment
  • H and H
  • Coags and 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 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 → Stomach and Duodenum
- Billroth II: Vagotomy, Antrectomy, Anastomosis → Stomach and Jejunum
- Whipple: Removal of the Distal 3rd 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 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

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 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
  • 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 SandS)
  • 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
  • ABX
  • Fluids
  • Tx of Sepsis
  • Tx of MODS

VI. 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

VII. 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 Hallow 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 Tx Concerns
  • Infection/Sepsis
  • Hemodynamic Status
  • Organ Function
  • ARDS, ATN, MODS

VIII. Summary
Renal Part 1

Presenter: Carol Rauen, RN, MS, PCCN, CCRN, CCNS, CEN
I. Introduction
AACN-CCRN Blueprint 6%
- Acute Renal Failure
- Chronic Renal Failure
- Life-Threatening Electrolyte Imbalance

II. Renal physiology

Major Functions of the Kidney
1. Excretion of Metabolic Wastes
2. Urine Formation
3. Acid-Base Balance Regulation
4. Electrolyte Regulation
5. Fluid Regulation
6. Blood Pressure Regulation
7. Erythropoietin Secretion/Anemia Regulation

Renal Assessment
1. Blood Work
   - Blood Urea Nitrogen
   - Creatinine
   - Serum Electrolytes
   - Hgb and Hct
   - Serum Albumin
   - Serum Osmolality

2. Urine Assessment
   - Volume and Concentration
   - Urinalysis (see table)
   - Renal Clearance Studies

3. Other Tests
   - KUB X-ray
   - Renal Arteriography
   - IVP
   - CT
   - Ultrasound
   - Biopsy

III. Chronic Renal Failure:
Acute renal failure affects many body systems. Chronic renal failure affects EVERY body system. Chronic renal failure (CRF) is a permanent, irreversible condition in which the kidneys cease to remove metabolic wastes and excessive water from the blood. (ESRF, ESRD, CRD, CKD)
- Etiology - more than 100 different diseases can cause RF
  - Glomerular Disease
  - Tubular Diseases
  - Vascular Kidney Diseases
  - Urinary Tract Disease
  - Infection (kidney)
  - Systemic Vascular Diseases
  - Metabolic Diseases
  - Connective Tissue Diseases

A. Terms
  1. Azotemia – Nitrogenous Waste Products in the Bloodstream
  2. Uremic Syndrome – Systemic and Laboratory Manifestations of ESRD
  3. Renal Replacement Therapy – Treatment Options

B. Stages of Renal Failure
  1. Diminished Renal Reserve
  2. Renal Insufficiency
  3. End Stage Renal Disease (ESRD) – Affects every system in the body

C. Treatment: Renal Replacement Therapies
  - Medications
  - Hemodialysis
  - Peritoneal Dialysis
  - Renal Transplant

IV. Acute Renal Failure:

A. Pathophysiology: a sudden deterioration in renal function usually associated with the loss of the kidney’s ability to concentrated urine, as well as the retention and accumulation of nitrogen wastes.
  - Decreased Glomerular Filtration Rate
  - Interstitial Inflammatory Changes
  - Tubular Lumen Obstruction
  - Oliguric, < 400 mL/day
  - Non-Oliguric, Large Amount of Dilute Urine

B. Common Etiologies
  - Severe Hypotension (all forms of shock)
  - Heart Failure
  - Dehydration
  - Nephrotoxic Agents
  - Complication of Infection
  - Severe Hypertension
<table>
<thead>
<tr>
<th>Category</th>
<th>Cause/Conditions</th>
</tr>
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<tbody>
<tr>
<td><strong>Pre Renal</strong></td>
<td><strong>Volume: Dehydration</strong></td>
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<tr>
<td></td>
<td>The problem is not actually with the kidneys but with perfusion (blood flow) to the kidneys</td>
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<td>Ischemia: hypovolemic shock, cardiogenic shock, septic shock, hypoxemia, low cardiac output, heart failure, severe hypertension</td>
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<td>Hemodynamic instability, multisystem organ failure, trauma</td>
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<td><strong>Post Renal</strong></td>
<td><strong>Urethral: Stricture, Prostatic Hypertrophy</strong></td>
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<tr>
<td></td>
<td>The problem is not actually with the kidneys but after the kidneys.</td>
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<td></td>
<td>Urethral: fibrosis, calculi, blood clots</td>
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<td>Bladder: neurogenic problems, neoplasms/cancer, obstruction</td>
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<tr>
<td><strong>Renal</strong></td>
<td><strong>Trauma</strong></td>
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<td></td>
<td>The problem is in the kidney itself effecting function. Kidney diseases</td>
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<td></td>
<td>Glomerulus: acute glomerulonephritis, acute cortical necrosis, hepatorenal syndrome</td>
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<td>Tubule: acute tubular necrosis, acute pyelonephritis</td>
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<td>Nephrotoxins: heavy metals, antibiotics, radiographic contrast media, anesthetics</td>
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<td></td>
<td>Pigments: hemoglobin, myoglobin</td>
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<td></td>
<td>Trauma, intravenous hemolysis, rhabdomyolysis</td>
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</tbody>
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C. Differentiating Pre-Renal From Renal Diagnosis for ATN

<table>
<thead>
<tr>
<th>Assessment</th>
<th><strong>Pre-Renal (Hypoperfusion)</strong></th>
<th><strong>Renal (Tissue Damage)</strong></th>
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<tbody>
<tr>
<td>Urinary Sodium</td>
<td>&lt; 20mEq/L</td>
<td>&gt; 20 mEq/L</td>
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<tr>
<td>BUN:Creatinine Ratio</td>
<td>&gt; 20:1</td>
<td>10-20:1 (normal)</td>
</tr>
<tr>
<td>Responds (increase in UO) to volume or diuretics</td>
<td>Positive Response</td>
<td>No Response</td>
</tr>
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D. Phases of ARF

- **Onset Phase**
  - BUN and Creatinine Rising
  - Urine Output Dropping
  - Diuretics Still Working
  - Acidosis Beginning

- **Oliguric Phase**
  - Alteration in Electrolyte Balance
  - Potential for Infection
  - Alteration in A-B Balance
  - Alteration in Nutrition Status
  - Uremic Syndrome
  - Alteration in Pulmonary Status
  - Alteration in GI Function

- **Diuretic Phase**
  - Fluid Loss
- Goal is to maintain adequate fluid balance and regulate electrolytes
- Alteration in Electrolytes

**Recovery Phase**
- Goal is Supportive Care
- Prevent Further Insults
- Assessment of Renal Function
- Keep patient well hydrated and free from infection
- Prevent Further Insults

**E. Systemic Response to Acute Failure**
- Hypertension
- Tachycardia
- Decreased UO
- Lethargy
- Pulmonary Edema
- Depends on Type
- Very Similar to Chronic RF

**F. Nursing Care Needs**
- Ensure Hydration
- Fluid Challenges
- Diuretics
- Monitor Fluid Status
- Weigh Daily and I and O
- Monitor Electrolyte Imbalance
- Support Renal Function

**G. Treatment Options/Alternatives**
- Drug Therapy
- Diet Therapy
- Renal Replacement Therapies (CVVH, Hemodialysis, Peritoneal Dialysis)
- Renal Transplant

**H. Support Therapy for ATN**

<table>
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<tr>
<th>Pt Problem</th>
<th>Treatment</th>
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<tr>
<td>Extracellular Volume Overload</td>
<td>Restrict NaCl and H$_2$O</td>
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<tr>
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<td>Diuretics</td>
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<td>Dialysis</td>
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<tr>
<td>Hyponatremia</td>
<td>Restrict Oral H$_2$O</td>
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<td>Restrict Hypotonic IV Solutions</td>
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<td>Hyperkalemia</td>
<td>Restrict K intake</td>
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<td>K Binding Resins</td>
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<td>Eliminate K Supplements</td>
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<td>Dialysis</td>
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<td>Metabolic Acidosis</td>
<td>NaBicarb</td>
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<td>Dialysis</td>
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<td>Hyperphosphatemia</td>
<td>Restrict PHO$_4$</td>
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<td>Phosphate Binding Agents</td>
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<td>Hypocalcemia</td>
<td>Calcium Carbonate</td>
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<td>Calcium Gluconate</td>
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<td>Dialysis</td>
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<td>Hypermagnesemia</td>
<td>D/C Mg Containing Antacids</td>
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<tr>
<td></td>
<td>Dialysis</td>
</tr>
<tr>
<td>Nutrition</td>
<td>High Protein</td>
</tr>
<tr>
<td></td>
<td>Enteral or Parental Nutrition</td>
</tr>
<tr>
<td>Drug Dosage</td>
<td>Adjust Doses Around GFR</td>
</tr>
<tr>
<td></td>
<td>Avoid NSAIDS, ACE I, Dye, Nephrotoxic Abx</td>
</tr>
</tbody>
</table>

V. Renal Replacement Therapies

Goal – to remove body waste and fluids in the presence of acute or chronic renal failure

A. Terms –
- **Diffusion**: movement of particles from an area of greater to an area of lesser concentration. During dialysis diffusion results in the movement of urea, creatinine, and uric acid from the patient’s blood in the dialysate.

- **Osmosis**: the movement of water across a semi-permeable membrane from an area of lesser to an area of greater concentration (osmolality) of particles. During dialysis osmosis results in extra fluid from the patient being removed.

- **Ultrafiltration**: the movement of fluid across a semi-permeable membrane as a result of an artificially created pressure gradient. More efficient than osmosis for the removal of water.

- **Dialysis**: involves the movement of fluid and particles across a semipermeable membrane. It is a treatment that can help restore fluid and electrolyte balance, control acid-base balance, and remove waste and toxic material from the body. It can sustain life successfully in both acute and chronic situations where substitution for or augmentation of normal renal function is needed.

B. Insurance Coverage – in 1972 the Congress enacted legislation that provides for people with ESRD to receive Medicare regardless of age. This is not true in all countries.

Hemodialysis

Goal – involves shunting the patient’s blood from the body through a dialyzer in which diffusion and ultrafiltration occur and then back into the patient’s circulation. Requires access to the pt’s blood, a mechanism to transport the blood to and from the dialyzer (where exchange of fluid, electrolytes, and waste products occur). HD can be used in the treatment of acute and chronic renal failure

Access – five different types of access can be used
- Arteriovenous Fistula
- Arteriovenous Graft
- External Arteriovenous Shunt
- Femoral Vein Catheterization
- Subclavian Vein Catheterization
Contraindications - Causes rapid fluid shifts
- Labile Cardiovascular States
- Recent MI
- Hypotension

Complications
- Hypotension
- Air Embolism
- Arrhythmias
- Infection
- Disequilibrium Syndrome - Rapid shifts in osmolality between cerebral spinal fluid and blood can lead to cerebral edema
- Coagulopathies - Heparin used during dialysis to prevent clotting of blood outside of body

Chronic Care Needs –
- Patients are typically hemodialyzed 2-3 times a week for 2-4 hours
- Require many medication
- Encounter multiple acute and chronic health risks as a result of the renal failure and dialysis
- Have dietary and fluid restrictions
- Safety concerns regarding access sites
- Assessment requirements for access sites

Peritoneal Dialysis
Goal – The goal is the same as above but a machine is not used to perform the “cleaning of the blood.” The dialyzing fluid is instilled into the peritoneal cavity, and the peritoneum becomes the dialyzing membrane. PD is used for acute and chronic renal failure and can be done in the hospital or at home.

Access – an abdominal catheter is inserted into the peritoneal space. In chronic use this catheter remains in place permanently and only changed periodically should problems arise.

Procedure – Approximately 2 liters of sterile dialysate is instilled into the peritoneal cavity and allowed to dwell for a period of time. During this time osmosis and diffusion of particles takes place. The catheter is then reopened and the fluid is drained from the patient (entire process is called an exchange). This process is done repeated during a 24 hr period.

Contraindications
- Peritonitis
- Abdominal Surgery
- Abdominal Adhesions
- Pregnancy

Complications
- Peritonitis
- Respiratory Distress
**Chronic Care Needs** – PD can be done independently at home and the individual can lead a fairly normal schedule. Not as many risks as HD. Most common problem is infection of abdominal catheter.

- Continuous ambulatory peritoneal dialysis (CAPD) – 4–5 exchanges are done a day.
- Continuous cyclic peritoneal dialysis (CCPD) – exchanges are done with the use of a machine to control the infusion, dwell and drain times and patients can set up before going to sleep and have their PD occur automatically while they sleep. They are completely independent the rest of the day.

**Continuous Renal Replacement Therapy**

**Goal** - CRRT provides continuous ultrafiltration of extracellular fluid and clearance of uremic toxins. Only done in the critical care setting.

**Access** – Arterial and venous cannulation sites are required or two venous cannulation.

**Procedure** – the blood leaves the patient and flow through a hemofilter where the ultrafiltration takes place and removal of water and waste (collected into standard urine bag) and then the blood is returned to the patient via the venous access. The flow gradient to move the blood through the filter is the patient’s own blood pressure. There are several types of processes that are used in the critical care setting for CRRT. Not necessary to learn this year. It will be covered in your acute care course next fall.

**Contraindications:**
- Inability to tolerate extracorporeal circulation
- Hypercoagulability
- Inability to tolerate anti-coagulation therapy (heparin)
- Fluid, electrolyte and acid-base shifts are less severe than with hemodialysis and usually better tolerated

**Complications**
- Fluid Imbalance - Hypo/Hypervolemia (Depends on ultrafiltration rate and intravascular volume requirements)
- Electrolyte Imbalance - Hypokalemia, Hyponatremia,
- Hypocalcemia, and Hypomagnesaemia
- Metabolic Acidosis - Bicarbonate readily removed
- Drug removal - Potential for removing most drugs
- Hemorrhage - Heparin used as blood leaves body to prevent coagulation
- Thrombosis/Infection
- Hypo/Hyperthermia

**VI. Renal Transplantation**

**VII. Summary**
Electrolyte Disturbances

I. Introduction
Fluid and electrolyte monitoring are an essential component of patient assessment. These factors regulate most physiological functions and the acid-base balance.

II. Physiologic fluid balance

A. Total Body Water – 60% of body weight (approximately 40L)
   1. Intracellular – 67% of total body H2O
      a. Primarily made up of intracellular electrolytes
   2. Extracellular – 33% of total body H2O
      a. Plasma Water – 8%, Water, proteins and lipids
      b. Interstitial Fluid and Lymph – 20%, Fluid bathing the cells
      c. Transcellular Fluid – 7%, Pleural, pericardial, peritoneal, synovial and fluids in secretions (GI, respiratory, salivary)

B. Osmolarity – the concentration of particles within a solution
   1. Plasma osmolarity avg. 290 ± 5 mOsm/kg
      Na is the primary regulator of extracellular osmolarity
      K is the primary regulator of intracellular osmolarity
   2. Calculated osmolarity = \(2(\text{Na}) + \text{BG} + \text{BUN} \over 18 \) 2.8

III. Electrolyte balance

A. Physiology:
Electrolytes are particles or solutes found throughout the body in fluids. They carry an electrical charge and are essential for fluid and acid base balance within the body. The cations (positively charged ions) are sodium (Na\(^+\)), potassium (K\(^+\)), magnesium (Mg\(^{++}\)), and calcium (Ca\(^{++}\)). The anions (negatively charged ions) are chloride (Cl\(^-\)), bicarbonate (HCO\(_3\)^-), sulfate (SO\(_4\)^{2-}\), and phosphate (PO\(_4\)^{3-}\).

The four major functions of electrolytes are:
1. Regulate Acid Base Balance
2. Maintain Fluid Balance and Osmolarity
3. Distribute the Body Fluid and H\(_2\)O Between the Compartments
4. Promote Neuromuscular Function/Irritability

B. Distribution:
Electrolytes are found in the intracellular and extracellular fluid. They are concentrated in one of these two compartments and exert osmotic properties within that compartment. Electrolytes help to maintain total body fluid balance and also help to regulate fluid movement in and out of the cell. For example K\(^+\) is the major intracellular ion and Na\(^+\) is the major extracellular ion and they each play a significant role in maintaining homeostasis within each of their compartments. Each electrolyte serves a unique physiologic function and concentrations above or below the “normal” range can affect homeostasis or specific organ function detrimentally.
### Electrolyte or Compound

<table>
<thead>
<tr>
<th>ELECTROLYTE or COMPOUND</th>
<th>PRIMARY COMPARTMENT</th>
<th>EXTRACELLULAR CONCENTRATION (plasma or intravascular)</th>
<th>INTRACELLULAR CONCENTRATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (Na⁺)</td>
<td>Extracellular</td>
<td>135 – 146 mEq/L</td>
<td>10 – 15 mEq/L</td>
</tr>
<tr>
<td>Potassium (K⁺)</td>
<td>Intracellular</td>
<td>3.5 – 5.5 mEq/L</td>
<td>140 - 150 mEq/L</td>
</tr>
<tr>
<td>Calcium (Ca++)</td>
<td>Extracellular</td>
<td>T 8.5 – 10.5 mg/dL, I 4.0 – 5.0 mg/dL</td>
<td>0 - 2 mg/dL</td>
</tr>
<tr>
<td>Magnesium (Mg++)</td>
<td>Intracellular</td>
<td>1.5 – 2.5 mEq/L</td>
<td>30 – 40 mEq/L</td>
</tr>
<tr>
<td>Phosphate (PHO₄⁻)</td>
<td>Intracellular</td>
<td>2.5 – 4.5 mg/dL, 1.7 – 2.6 mEq/L</td>
<td>100 mEq/L</td>
</tr>
<tr>
<td>Chloride (Cl⁻)</td>
<td>Extracellular</td>
<td>96 – 109 mEq/L</td>
<td>1 – 4 mEq/L</td>
</tr>
<tr>
<td>Bicarbonate (HCO₃⁻) or Serum C0₂</td>
<td>Extracellular</td>
<td>22 – 26 mEq/L</td>
<td>4 – 10 mEq/L</td>
</tr>
</tbody>
</table>

### C. Sodium: Na⁺ 135 – 146 mEq/L

1. **Function:** Sodium is the major extracellular cation. Its osmotic properties make it very important in both fluid and acid-base balance within the body. There is a close relationship between water and sodium. Sodium is also essential for physiologic activities, the active and passive transport mechanism across the cell membrane and intracellular metabolism.

2. **Hyponatremia:** Na⁺ < 135 mEq/L

   **Causes** - Fluid Excess or Sodium Deficit:
   - thiazide diuretics, decreased Na⁺ dietary intake, vomiting, diarrhea, SIADH, adrenal insufficiency, NG suctioning, profuse diaphoresis, draining fistulas, over hydration, congested heart failure, renal failure, salt-losing nephritis, liver failure, hyperglycemia (osmotic diuresis)

   **Signs and Symptoms:** muscle weakness, headache, fatigue, apathy, malaise, orthostatic hypotension, poor skin turgor, wt. loss, nausea, anorexia, vomiting, decreased CVP, abdominal cramps, seizures, respiratory distress, confusion up to coma.

   **Treatment:** oral or IV replacement of sodium. 0.9% Sodium Chloride or Lactated Ringer’s solutions. Hypertonic Saline can be used for emergency situations.

   **Clinical Pearl**
   
   Hyponatremia is the most frequent electrolyte imbalance seen in hospitalized patients.

3. **Hypernatremia:** Na⁺ > 146 mEq/L

   **Cause:** Fluid Deficit or Sodium Excess:
   - excess dietary intake, mineral corticoids, excessive adrencorticord secretions, diabetes insipidus, strict fluid restrictions, hypothalamic dysfunction, osmotic diuretics, hypercalcemia or hypokalemia, excessive IV infusion of sodium chloride solutions, pregnancy.

   **Signs and Symptoms:** muscle weakness, restlessness, tachycardia, low urine output, orthostatic hypotension, dry mucous membranes, flushed skin, irritability, lethargy, seizures, dyspnea, dehydration, confusion to coma.
**Treatment:** replace volume and treat underlining cause.

Free H₂O deficit (L) = \(0.6 \times \text{kg} \times \frac{\text{Na} - 140}{140}\)

Example: 70kg patient with Na of 160

\((0.6 \times 70) \times \frac{160 - 140}{140} = 42 \times 0.14 = 5.88\) L H₂O deficit
Sodium

Hyponatremia
Fluid Excess
Sodium Deficit

Neuro:
Headache,
Fatigue, Apathy,
Seizures,
Confusion →
Coma

Pulm: Resp
Distress

CV: Orthostatic
Hypotension,
Drop CVP

GI: Anorexia, Wt
Loss, N/V, Abd
Cramps

Mus/Sk: Muscle
Weakness

Hyponatremia
Fluid Deficit
Sodium Excess

Neuro:
Restlessness,
Irritability,
Lethargy,
Seizures,
Confusion →
Coma

Pulm: Dyspnea,

CV: Tachycardia,
Orthostatic
Hypotension,
Dry Mucous
Membranes,
Dehydration,
Flushed Skin

GU: Low Urine

Mus/Sk: Muscle
Weakness

Common
Electrolyte Changes
K Opposite Direction
Cl Same Direction
D. Potassium: $K^+ = 3.5 – 5.5 \text{ mEq/L}$

1. **Function:** Major intracellular cation contributes to cell homeostasis and function by maintaining its osmolarity and electro neutrality. Potassium plays a principle role in electrical conductivity by influencing neuromuscular transmission of nerve impulses and cardiac muscle contractility. Also helps to maintain acid-base balance and normal kidney function.

2. **Hypokalemia:** $K^+ < 3.5 \text{ mEq/L}$
   
   **Cause:** Decreased Intake, Increase Loss or Shift of K into Cells:
   starvation, dehydration, massive fluid infusion lacking in $K^+$, decreased dietary intake, vomiting, diarrhea, corticosteroids therapy, draining fistula, diuretics, some antibiotics, laxative overuse, NG suctioning, hypernatremia, metabolic alkalosis (relative hypokalemia), aldosteronism.

   **Signs and Symptoms:** ECG Changes – depressed ST segments, flat or inverted T waves, presence of U waves, dysrhythmias, cardiac arrest, dilute urine, anorexia, nausea, vomiting, ileus, lethargy, mental depression, paralysis, confusion, muscle weakness, respiratory arrest, can precipitate digitalis toxicity.

   **Treatment:** Oral or Parenteral Replacement of $K^+$

3. **Hyperkalemia:** $K^+ > 5.5 \text{ mEq/L}$
   
   **Cause:** Excess Intake, Decreased Loss, Shift of K out of Cells: movement of K out of the cells (acidosis, sepsis, fever, trauma, hyperglycemia, rhabdomyolysis, catecholamines, insulin deficiency, tissue necrosis), excessive dietary intake, renal failure (decreased excretion), Addison’s disease (adrenal insufficiency), large volume of stored blood products, potassium sparing diuretics, medications that promote $K^+$ retention (ACE inhibitors, beta blockers, NSAIDS, heparin), hyperosmolar states, excessive potassium administration.

   **Signs and Symptoms:** ECG changes – tall, peaked, tented T waves, flattened or absent P waves, widening QRS, asystole, alteration of depolarization/repolarization of cardiac muscle, oliguria, nausea, vomiting, diarrhea, calf pain, numbness or paresthesia, hyporeflexia up to flaccid paralysis.

   **Treatment:** Three-Part Therapy
   
   1. **Cardiac Protect:** 10ml of Calcium Chloride or Calcium Gluconate slow IV push. Renders the myocardium less excitable by decreasing the effects of excess extracellular $K^+$.
   
   2. **Shift $K^+$ into the Cell:**
      - 1 amp Sodium Bicarbonate
      - 5-10U Regular Insulin
      - 50ml Bolus 50% Dextrose
      - Albuterol 10 – 20mg inhalation or intravenous (beta$_2$ adrenergic agent – stimulates B$_2$ receptor in the pancreas to release more insulin).

   3. **Removal of $K^+$:**
      - Loop Diuretic
      - Sodium Polystyrene Sulfonate (Kayexalate) a cation exchange resin given orally or by retention enema. Oral administration is more effective. Each 1gm will lower the $K^+$ 1mEq with orally administration, and 0.5mEq with rectal administration. Sorbitol prevents constipation.
      - Dialysis can also be utilized to remove K+ from the body.
Potassium

**Hypokalemia**
- Decrease Intake
- Increased Loss
- Shift of K into Cells

**Neuro:**
- Lethargy,
- Decreased Reflexes,
- Confusion,
- Depression

**CV:** Drop BP,
- Dysrhythmias,
- Cardiac Arrest

**GI:** Anorexia,
- N/V, Distension
- Ileus

**GU:** Dilute,
- Urine, Water
- Loss, Thirst

**Mus/Sk:** Weak,
- Flaccid, Resp
- Arrest

**Hyperkalemia**
- Excess Intake
- Decreased Loss
- Shift K out of Cells

**Neuro:**
- Numbness,
- Paresthesias,
- Hyporeflexia

**CV:** Conduction
- Disturbances,
- V-Fib, Asystole

**GI:** N/V/D

**GU:** Oliguria,
- Anuria

**Mus/Sk:** Early →
- Irritability
- Late →
- Weakness
- Flaccid Paralysis

Common Electrolyte Changes
Na Opposite Direction
E. Calcium  Total 8.5 – 10.5 mg/dL,

**Ionized (biologically active) 4.0 – 5.0 mg/dL**
Total = 45% ionized + 40% protein bound + 15% complexed
Corrected Ca\(^{++}\) = Total Ca\(^{++}\) + 0.8(4.0 – serum albumin)

**Function:** Calcium is necessary for many physiologic and metabolic processes. The transmission of nerve impulses, and cardiac muscle contractility are calcium dependent. Because Ca\(^{++}\) lines the pores of the cell membrane it plays an important role with action potential and pacemaker function. Calcium is needed for activation of the clotting mechanisms and in teeth and bone formation. Vascular smooth muscle is affected by Ca\(^{++}\) and therefore it plays a role in muscle contraction and vasodynamics.

**Calcium Regulation:** Ca\(^{++}\) Homeostasis is Maintained by Organ Regulation and Hormonal Control.
- Organ Regulation: Bone, Intestinal and Kidney
- Parathyroid glands secrete parathyroid hormone (PHT) which regulates movement of Ca\(^{++}\) into and out of the bone, GI tract and kidney
- Vit D is necessary for PHT assistance in Ca\(^{++}\) regulation
- Calcitriol (hormone) stimulates absorption and reabsorption of Ca\(^{++}\)
- Calcitonin (thyroid hormone) is secreted in hypercalcemia to inhibit bone reabsorption and increase renal excretion.
- Acid-Base Regulation. Alkalosis = Hypocalcemia, Acidosis = Hypercalcemia
- Hyperphosphatemia = Hypocalcemia
- Hypomagnesemia = Hypocalcemia

**Hypocalcemia:** Total Ca\(^{++}\) < 8.5 mg/dL
Ionized < 4.0 mg/dL

**Cause:** Excess Loss, Inadequate Intake, Decreased Ionized Ca, Decreased GI/Bone Absorption, Movement of Ca into Cell (Alkalosis): Alkalosis, renal disease, large transfusions of PRBC (citrate), hypoparathyroidism, hypomagnesemia, liver failure, sepsis, pancreatitis, burns, diarrhea, diuretics, malabsorption syndromes, Vit D deficiency, medications (radiographic contrast, NaHCO\(_3\), protamine, aminoglycosides), inadequate dietary intake of Ca\(^{++}\), Hypothyroidism, metabolic bone disease, hyperphosphatemia (including rapid infusion of PHO\(_4\)^–), elevated calcitonin, alcoholism, post op thyroid, parathyroid or radical neck surgeries.

**Signs and Symptoms:** ECG Changes – prolonged ST segment, torsades de pointes, catecholamine insensitivity, and bradycardia. Osteoporosis, paresthesia, numbness, tingling, muscle weakness, twitching and/or hyperreflexia, tetany, seizures, larynogospasm and bronchospasm, bruising/bleeding.

**Chvostek’s Sign** – Twitching of the lip and/or muscles on the side of the face simulated from tapping the facial nerve (CNVII) on that same side.

**Trousseau’s Sign** – Palmar flexion of the hand simulated from inflating a blood pressure cuff (3 minutes) on that arm. The cuff induces ulnar nerve ischemia.
**Treatment:** Oral or IV replacement of Ca\(^{++}\) (calcium gluconate or calcium chloride), administer Vit. D, aluminum hydroxide gel for Hyperphosphatemia, Mg for Hypomagnesemia, monitor pt carefully.

<table>
<thead>
<tr>
<th>Clinical Pearl</th>
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<tbody>
<tr>
<td>Calcium chloride has more Ca(^{++}) than calcium gluconate but is also more irritating to the vein</td>
</tr>
</tbody>
</table>

4. **Hypercalcemia:**  
   - **Total Ca\(^{++}\)> 10.5 mg/dL**  
   - **Ionized > 5.0 mg/dL**

**Cause:** Excess Intake, Loss from Bones, Increased mobilization from Bones, Movement of Ca out of Cell (Acidosis):  
   - Metastatic carcinoma (breast, bone, multiple myeloma, osteolytic metastases) and hyperparathyroidism account for 80% of all hypercalcemia. Also acidosis, immobilization, thiazide diuretics, renal failure, tuberculosis, sarcoidosis, excessive dietary intake, steroid therapy, Grave’s disease (hyperthyroidism).

**Signs and Symptoms:** ECG Changes – shortening of the ST and QT segments, heart blocks. Muscle weakness, hypotonia, hyporeflexia, seizures, confusion up to coma, anorexia, nausea, vomiting, constipation, peptic ulcer, renal failure flank and leg pain, fatigue.

**Treatment:** Volume expansion with normal saline, loop diuretics or corticosteroids, calcitonin and/or mithramycin (prevent bone reabsorption), treat underlying cause.
**Calcium**

**Hypocalcemia**
- Excess Loss
- Inadequate Intake
- Decreased Ionized
  - GI/Bone Absorption
- Alkalosis

**Hypercalcemia**
- Excess Intake
- Loss from Bones
- ↑Mobilization from Bones
- Acidosis

**Neuro:** Tingling →
  - Convulsions,
  - Hyperreflexia

**Pulm:**
- Laryngospasm,
- Bronchospasm

**CV:**
- Dysrhythmias,
- Cardiac Arrest,
- Bruising, Bleeding

**GI:**
- Inc Peristalsis,
- N/V/D

**Mus/Sk:**
- Osteoporosis →
  - Fractures, Abnormal
  - Deposits of Ca in
  - Body Tissues,
  - Muscle Spasm,
  - Tetany

**Common**
- Electrolyte Changes
- Mg Same Direction
- PH04 Opposite Direction

**Neuro:** Dec Reflexes,
  - Lethargy → Coma,
  - Seizures

**CV:**
- Depressed
  - Activity,
  - Dysrhythmias,
  - Cardiac Arrest

**GI:**
- Dec GI Tract
  - Motility, N/V,
  - Constipation

**GU:**
- Kidney Stones,
  - Flank Pain

**Mus/Sk:**
- Muscle
  - Fatigue, Hypotonia,
  - Bone Pain,
  - Osteoporosis,
  - Fractures
F. Magnesium: Mg\(^{++}\) = 1.5 – 2.5 mEq/L

**Function:** Magnesium is essential for the production and use of energy, all ATP reactions involve Mg\(^{++}\). The Na\(^+\)/K\(^+\) ATPase pump is dependent on Mg\(^{++}\), therefore making it an important component in the action potential and depolarization and repolarization of the cardiac muscle. Mg\(^{++}\) appears to play a role in membrane stabilization decreasing the likelihood of cardiac cell irritability or ectopy. It also has vasodilating effects, and it influences the release of neurotransmitters at the neuromuscular junction by stabilizing the nerve axon.

**Hypomagnesemia:** Mg\(^{++}\) < 1.5 mEq/L

**Cause:** Excess Loss, Decreased Intake, Impaired Absorption, Movement of Mg into the Cell (Alkalosis): Excessive diuretic therapy, starvation, malabsorption, medications (digitalis, cyclosporine, cisplatin), endocrine disorders (DKA, HHNK, hyperaldosteronism, hyperthyroidism), chronic alcoholism, pancreatitis, alkalosis, vomiting, NG suctioning, citrate-chelation, decreased intake (enteral or parenteral).

**Signs and Symptoms:** (very similar to hypocalcemia) ECG Changes – flat or inverted T waves, ST segment depression, prolonged QT interval, supraventricular and/or ventricular ectopy including torsades de pointes and Vfib. Chvostek’s and Trousseau’s signs, hyperreflexia, vertigo, seizures, confusion, hallucinations, depression up to coma, increased SVR and hypertension, nausea and vomiting.

**Treatment:** IV administration of Mg\(^{++}\) with close monitoring. 1–4g Mg\(SO_4\) over 2 minutes to 6 hr (depending on severity of depletion). Common side effects of Mg\(SO_4\) administration are flushed feeling or sweating, bradycardia, hypotension and IV site burning.

<table>
<thead>
<tr>
<th>Clinical Pearl</th>
</tr>
</thead>
<tbody>
<tr>
<td>When low Mg(^{++}) and low K(^+) are both present the patient will be unresponsive to KCL therapy until the hypomagnesaemia is treated.</td>
</tr>
</tbody>
</table>

**Hypermagnesemia:** Mg\(^{++}\) > 2.5 mEq/L

**Cause:** Excess Mg\(^{++}\) intake (Mg\(SO_4\), laxatives, antacids), Renal Insufficiency or Failure, Movement of Mg out of Cell (Acidosis)

**Signs and Symptoms:** ECG Changes – peaked T waves, shortened QT interval, prolonged PR and QRS intervals, bradycardia, heart blocks, asystole. Hyporeflexia, respiratory depression to apnea, lethargy to coma, seizure, hypotension, hypocalcaemia, hyperkalemia, flushed/warm skin.

**Treatment:** Volume administration, diuretics, decrease Mg\(^{++}\) intake, IV insulin and glucose will drive Mg\(^{++}\) back into cell, treat acidosis, hemodialysis or CAPD with Mg-free dialysate.

V. **Summary**

<table>
<thead>
<tr>
<th>Na</th>
<th>Cl</th>
<th>Glucose</th>
</tr>
</thead>
<tbody>
<tr>
<td>K</td>
<td>CO2</td>
<td></td>
</tr>
</tbody>
</table>
Magnesium

Hypomagnesemia
Excess Loss
Decreased Intake
Impaired Absorption
Alkalosis

Hypermagnesemia
Excess Intake
Renal Insufficiency/Failure
Acidosis

Neuro: Agitation,
Depression,
Confusion,
Convulsions,
Paresthesias,
Ataxia,
Hyperreflexia,
Vertigo, Seizures

CV: Dysrhythmias,
Tachycardia,
Hypertension, Inc
SVR

GI: N/V

Mus/Sk: Cramps,
Spasticity, Tetany

Common Electrolyte Changes
Ca Same Direction

Neuro:
Hyporeflexia,
Lethargy → Coma

Pulm: Resp
Depression,
Apnea

CV: Dysrhythmias,
Hypotension,
Flushed/Warm
Skin

Mus/Sk: Muscle
Fatigue,
Hypotonia, Bone
Pain,
Osteoporosis,
Fractures