Session 5

Gastrointestinal & Renal

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Gastrointestinal

I. INTRODUCTION

PCCN Test Plan

*Endocrine, Heme, GI, & Renal: 18%*

a. Functional GI Disorders (e.g., obstruction, ileus, diabetic gastroparesis, gastroesophageal reflux, irritable bowel syndrome)

b. GI Bleed
   - Lower
   - Upper

c. GI Infections

d. Hepatic Failure

e. Ischemic Bowel

f. Malnutrition (e.g., failure to thrive, malabsorption disorders)

g. Pancreatitis

**Structures/Function/Digestion**

a. Mouth

b. Esophagus

c. Stomach

d. Small Intestine

e. Pancreas

f. Gallbladder

g. Liver

h. Spleen

i. Portal Circulation

j. Mesentery Circulation

k. Large Intestine

l. Digestive Hormones

m. Digestive Enzymes

**Assessment**

a. Inspection

b. Auscultation

c. Palpation

d. Percussion
II. THE HEPATIC SYSTEM

Liver Function

a. Metabolic Factory & Waste Disposal Plant  
b. Carbohydrate, Fat & Protein Metabolism  
c. Production of Bile Salts  
d. Production of Clotting Factors  
e. Bilirubin Metabolism  
f. Detoxification: Nutrients, Drugs, Toxins, Bacteria, Everything  
g. Vitamin & Mineral Storage:  
h. Blood Reservoir: 10% of Total Blood Volume

Liver Function Tests

<table>
<thead>
<tr>
<th>Test</th>
<th>Results</th>
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</table>
| 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  
                             | • Pre-Albumin: 17 – 40 mg/dL |
| Serum Ammonia               | • 19 – 60 mcg/dL                                                        |
| 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 |
| Coagulation Studies            | • Indirectly reflect liver function  
                              | o PT  
                              | o PTT  
                              | o INR  
                              | o Bleeding Time  
                              | o ACT |
| Hepatic Enzymes             | • ALP: 42 – 136U/L  
                             | • GGT  
                             | o Men: 0–85 U/L  
                             | o Women: 0-70 U/L  
                             | • AST  
                             | o Men: 15-40 U/L  
                             | o Women: 13-35U/L  
                             | • ALT  
                             | o Men: 10-55U/L  
                             | o Women: 7-30U/L |
Liver Dysfunction & Failure

Pathophysiology
a. Liver Tissue (cells) are Destroyed and Replaced with Fibrotic Tissue
b. Functions are Altered
c. Organ Changes Shape
d. Vascular Flow is Obstructed
e. Portal Hypertension

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.
a. Causes
   • Alcoholic, Laennec’s Portal, or Fatty
   • Post Necrotic: Toxic, Nodular, or Post Hepatic
   • Biliary: Cholangitic or Obstructive

Hepatitis
Widespread Inflammation of Liver Cells
a. Causes
   • Primary Viral – Most Common
   • Hepatotoxins - Toxic or Drugs
   • Secondary Viral, Low Mortality

Clinical Presentation of Liver Dysfunction

Hepatic Encephalopathy
The liver is unable to perform its detoxification function and toxins build up. Primarily ammonia causing altered LOC, behavior and motor abilities.
a. Clinical Presentation
   • Confusion → Coma
   • Agitation → Unsafe Behavior
   • Asterixis: Flap like Tremor of Hands
   • Apraxia: Inability to Perform Purposeful Acts
   • Elevated Ammonia
b. Common Treatment Modalities
   • Limit Protein Intake
   • Limit Hepatotoxic Drugs
   • Lactulose & Neomycin
   • Safe Environment
Malnutrition
The liver is unable to perform its function of carbohydrate, protein and fat metabolism. This leads to malnutrition.
   a. Clinical Presentation
   b. Common Treatment Modalities
      • Need to tx the Cause of Liver Failure
      • Parenteral Nutrition
      • Limit Protein Intake
      • Restrict Fluids

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.
   a. Clinical Presentation
      • Bleeding Tendencies
      • Nonspecific Bleeding
   b. Common Treatment Modalities
      • Monitor Coagulation Studies & Platelet Ct
      • Decrease Bleeding and Bruising Risk
      • Administer Blood Products

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.
   a. Clinical Presentation
      • Caput Medusae: dilated cutaneous veins radiating from the umbilical
      • Spider angiomas commonly seen in Cirrhosis
      • Upper GI Bleeding
   b. Common Treatment Modalities
      • Surgical Shunting
      • TIPSS - Transjugular Intrahepatic Portosystemic Stent Shunt
      • Treat Bleeding
      • Treat Cause

Hepatorenal Syndrome
A form of pre-renal failure caused by the liver dysfunction. Mortality of liver failure is very high once renal failure develops.
   a. Clinical Presentation
      • S&S of Renal Dysfunction
b. Common Treatment Modalities
   - Maintain Adequate Renal Perfusion
   - Restrict Fluids
   - Restrict Nephrotoxic Agents
   - Continuous Renal Replacement Therapies

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.

a. Clinical Presentation
   - Inc. Abdominal Girth
   - Hypotension and Tachycardia
   - Dyspnea, Orthopnea, Tachypnea
   - S&S of Dehydration
   - N&V

b. Common Treatment Modalities
   - Restrict PO Fluid
   - Diuretics (if tolerated hemodynamically)
   - Restrict Na
   - Respiratory Support
   - Paracentesis
   - Peritoneovenous Shunt Surgery

Infection
One of the functions of the liver cells (Kuppfer 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.

a. Clinical Presentation
   - Poor Wound Healing
   - Increased Risk of Infection

b. Common Treatment Modalities
   - Heightened Prevention Measures
   - Abx Therapy – w Caution

II. THE PANCREAS

Function

a. Endocrine Functions
   • Synthesis & Release of Hormones:
     o Glycogen
     o Insulin
     o Gastrin
b. Exocrine Functions
   • Pancreatic Enzymes Break Down Protein, Starch & Fat. > 2L/day
   • Bicarbonate Raise pH

c. PNS, Gastrin & Hormones Regulate Secretions

Pancreatic Enzymes

a. Trypsin: Aids in Protein Digestion
b. Amylase: Aids in Carbohydrate Digestion
c. Lipase: Aids in Fat Digestion

Acute Pancreatitis

Pathophysiology

a. Auto Digestion
   • Tissue Damage
   • Fat Necrosis
   • Vascular Damage & Hemorrhage
   • Increased Capillary Permeability
   • Hypotension
b. Forms/Types
   • Edematous
   • Hemorrhagic
c. Classifications
   • Acute Pancreatitis
   • Recurrent Acute
   • Recurrent Chronic
   • Chronic Pancreatitis

Cause (blocked enzyme release)

a. Alcoholism
b. Biliary Stones
c. Hyperlipidemia
d. Abd Trauma
e. Infection (bacterial or viral)
f. Shock
g. Drugs (Most Common: Cyclosporine, Acetaminophen, Cimetidine, Steroids, Salicylates, Furosemide, Thiazides, Estrogens)

**Clinical Presentation**
a. Pain  
b. Low Grade Fever  
c. N&V  
d. Distended/Tender/Rigid Abd  
e. Guarding with Rebound Tenderness  
f. Jaundice  
g. Hypoactive Bowel Sounds  
h. Steatorrhea: bulky, pale, foul-smelling stools  
i. ? Ascites  
j. Hypovolemic Shock  
k. In Necrotizing Pancreatitis  
  • Cullen’s Sign:  
    o Bluish Discoloration Umbilical  
  • Grey Turner’s Sign:  
    o Bluish Discoloration Flanks

**Labs**  
Underlined labs are the MOST diagnostic  
a. Hypocalcemia (classic sign)  
b. Low Ca, Mg, K  
c. Hyperglycemia  
d. Hyperbilirubinemia  
e. Hypertriglyceridemia  
f. Increased BUN & Creatinine  
g. Elevated Amylase  
h. Elevated Lipase  
i. Elevated LFTs  
j. Elevated WBC  
k. Decreased H/H  
l. ? Increased H/H

**Treatment Options**  
a. Fluid Resuscitation  
b. Rest the Pancreas: NPO, NGT  
c. Pain Management  
d. Monitor & Replace Electrolytes  
e. Tx Multisystem  
f. Nutritional Support  
g. Surgery
IV. ACUTE GI HEMORRHAGE

Lower GI Bleeding

Not Typically Life Threatening
a. Causes
   - Diverticulitis
   - Angiodysplasia
   - Cancer
   - Hemorrhoids
   - Inflammatory Bowel Disease (Ulcerative Colitis; Crohn's Disease)
   - Bowel Infarction

Upper GI Bleeding

a. Causes
   - Peptic Ulcer Disease: Duodenal, Gastric and Stomal ulcers account for 50% bleeding episodes
   - Gastritis or Esophagitis
   - Esophageal Varices
   - Mallory -Weiss Syndrome
b. Clinical Presentation
   - Hematemesis
   - Melena
   - PUD
   - Distended & Tender Abd
   - Hyperactive Bowel Sounds
   - Hypovolemia
   - Shock
c. Assessment
   - H & H
   - Coags & Platelets
   - Hemoconcentration
   - Elevated BUN
   - LFTs
   - Endoscopy
   - Angiography
   - Raionuclide Scans
d. Treatment
   - NG Decompression/Lavage – Room Temp vs Iced
   - Fluid Resuscitation
   - Blood Product Admin
   - Endoscopic Sclerotherapy
Session 5: Gastrointestinal & Renal

- Pharmacology
- 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
  - Portal Caval Shunt

III. DISORDERS OF THE BOWEL

Bowel Infarction

a. Etiology
   - Embolic or Thrombotic Occlusion
   - Typically from the Superior Mesenteric Artery
b. Clinical Presentation
   - Severe Epigastric Pain
   - Rebound Tenderness
   - Guarding & Rigidity
   - Stimulated Sympathetic Response from Pain
c. Treatment Options
   - Angiography to Identify/Confirm Occlusion
   - Surgery to Remove Occlusion & Dead Bowel

Bowel Obstruction

a. Etiology
   - Internal Lumen Obstruction ex. Tumor
   - External Lumen Obstruction ex. Adhesions
   - Emboli: no blood flow
   - Paralytic Ileus
b. Clinical Presentation
   • Complete vs Partial
   • Distended Edematous Bowel
   • Fluid and Electrolytes Leaking from Bowel
   • Elevated WBC
   • Fever
   • Small Intestine
     o Acute Pain w Sudden Onset
     o N & V (movement on both ends)
     o Wave-Like Hyperactive High Pitched Bowel Sounds
     o May Have Some Gas or Feces
     o Distention (mild)
   • Large Intestine
     o Slow Onset Pain Progression Mild → Severe, Lower Abd
     o No N & V (nothing moving)
     o No Stool
     o Low Pitched Bowel Sounds
     o Distention (large amount)
   • Treatment Options
     o Diagnosis Obstruction by Hx, X-Ray, CT, Upper or Lower Barium Radiology Tests
     o Pain Management
     o IV Fluids
     o Decompress w NG, Rectal or Intestinal Tube
     o Abx
     o NPO and Time (rest the bowel)
     o Surgery

Perforation/Peritonitis

a. Etiology
   • Gastric/Intestinal Contents Leak into Peritoneal Cavity
   • Ulcer Perforation
   • Diverticular Rupture
   • Trauma
   • Bowel Infarction
b. Clinical Presentation
   • Infection/Sepsis (all the S&S)
   • Sudden Onset of Severe Pain
   • Rigid Abdomen w Rebound Tenderness
   • Hypoactive Bowel Sounds → No Bowel Sounds
c. Treatment Options
   • Surgery to Repair Cause & Clean Up
   • ABX
   • Fluids
   • Tx of Sepsis
   • Tx of MODS

Functional GI Disorders

a. Ileus
b. Diabetic Gastroparesis
c. Gastroesophageal Reflux
d. Irritable Bowel Syndrome
Renal

I. INTRODUCTION

PCCN Test Plan

*Endocrine, Hematology, Renal & GI: 18%*

- a. Acute Renal Failure
- b. Chronic Renal Failure
- c. Contrast-Induced Nephropathy
- d. End-Stage Renal Disease (ESRD)
- e. Electrolyte Imbalances
- f. Medication-Induced Renal Failure
- g. Nephritic Syndrome

II. RENAL PHYSIOLOGY

Major Functions of the Kidney

- a. Excretion of Metabolic Wastes
- b. Urine Formation
- c. Acid-Base Balance Regulation
- d. Electrolyte Regulation
- e. Fluid Regulation
- f. Blood Pressure Regulation
- g. Erythropoietin Secretion/Anemia Regulation

Renal Assessment

- a. Blood Work
  - Blood Urea Nitrogen
  - Creatinine
  - Serum Electrolytes
  - Hgb & Hct
  - Serum Albumin
  - Serum Osmolality
b. Urine Assessment
   • Volume & Concentration
   • Urinalysis
   • Renal Clearance Studies

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

d. Nephritic Syndrome

III. END-STAGE RENAL DISEASE (ESRD)

a. Acute renal failure affects many body systems.

b. Chronic renal failure affects EVERY body system.

c. 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).

d. 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

Terms

a. Azotemia – Nitrogenous Waste Products in the Bloodstream

b. Uremic Syndrome – Systemic and Laboratory Manifestations of ESRD

c. Renal Replacement Therapy – Treatment Options

Stages of Renal Failure

a. Diminished Renal Reserve

b. Renal Insufficiency

c. End Stage Renal Disease (ESRD) – Affects every system in the body
**Treatment**
Renal Replacement Therapies

a. Medications  
b. Hemodialysis  
c. Peritoneal Dialysis  
d. Renal Transplant

**IV. ACUTE RENAL FAILURE**

**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.

a. Decreased Glomerular Filtration Rate  
b. Interstitial Inflammatory Changes  
c. Tubular Lumen Obstruction  
d. Oliguric < 400 mL/day  
e. Non-Oliguric, Large Amt of Dilute Urine

**Common Etiologies**

a. Severe Hypotension (all forms of shock)  
b. Heart Failure  
c. Dehydration  
d. Nephrotoxic Agents
e. Complication of Infection
f. Severe Hypertension

<table>
<thead>
<tr>
<th>Etiologies of Acute Renal Failure</th>
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<tbody>
<tr>
<td><strong>Category</strong></td>
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<tr>
<td><strong>Pre Renal</strong></td>
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<tr>
<td><strong>Post Renal</strong></td>
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**Phases of ARF**

a. Onset Phase
   - BUN & Creatinine Rising
   - Urine Output Dropping
   - Diuretics Still Working
   - Acidosis Beginning
   - Oliguric Phase
   - Alteration in Electrolyte Balance
   - Potential for Infection
   - Alteration in acid-base Balance
   - Alteration in Nutrition Status
   - Uremic Syndrome
   - Alteration in Pulmonary Status
   - Alteration in GI Function

b. Diuretic Phase
   - Fluid Loss
   - Goal is to Maintain Adequate Fluid Balance and Regulate Electrolytes
• Alteration in Electrolytes
c. Recovery Phase
  • Goal is Supportive Care
  • Prevent Further Insults
  • Assessment of Renal Function
  • Keep Patient Well Hydrated and Free From Infection
  • Prevent Further Insults

Systemic Response to Acute Failure

a. Hypertension
b. Tachycardia
c. Decreased UO
d. Lethargy
e. Pulmonary Edema
f. Depends on Type
g. Very Similar to Chronic RF

Nursing Care Needs

a. Ensure Hydration
b. Fluid Challenges
c. Diuretics
d. Monitor Fluid Status
e. Weigh Daily & I & O
f. Monitor Electrolyte Imbalance
g. Support Renal Function

Treatment Options/Alternatives

a. Drug Therapy
b. Diet Therapy
c. Renal Replacement Therapies (Hemodialysis, Peritoneal Dialysis)
d. Renal Transplant
### Support Therapy for Renal Failure

<table>
<thead>
<tr>
<th>Pt Problem</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| Extracellular Volume Overload | • Restrict NaCl and H₂O  
|                              | • Diuretics                                             
|                              | • Dialysis                                              |
| Hyponatremia                | • Restrict Oral H₂O  
|                              | • Restrict Hypotonic IV Solutions                       |
| Hyperkalemia                | • Restrict K intake  
|                              | • Dialysis                                              
|                              | • K Binding Resins                                      
|                              | • Glucose/Insulin                                        
|                              | • Eliminate K Supplements                                
|                              | • NaBicarb                                              
|                              | • Ca Gluconate                                           |
| Metabolic Acidosis          | • Na Bicarb                                             
|                              | • Dialysis                                              |
| Hyperphosphatemia           | • Restrict PHO₄                                         
|                              | • Dialysis                                              
|                              | • Phosphate Binding Agents                              |
| Hypocalcemia                | • Calcium Carbonate                                     
|                              | • Calcium Gluconate                                     
|                              | • Phosphate Binding Agents                              
|                              | • Dialysis                                              |
| Hypermagnesemia             | • D/C Mg Containing Antacids                            
|                              | • Dialysis                                              |
| Nutrition                   | • High Protein                                          
|                              | • Enteral or Parental Nutrition                         |
| Drug Dosage                 | • Adjust Doses Around GFR                               
|                              | • Avoid NSAIDS, ACE I, Dye, Nephrotoxic Abx             |

### V. RENAL REPLACEMENT THERAPIES

**Goal**  
To remove body waste and fluids in the presence of acute or chronic renal failure

**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.
Access
Five different types of access can be used
a. Arteriovenous Fistula
b. Arteriovenous Graft
c. External Arteriovenous Shunt
d. Femoral Vein Catheterization
e. Subclavian Vein Catheterization

Contraindications
Causes rapid fluid shifts
a. Labile Cardiovascular States
b. Recent MI
c. Hypotension

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

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

Peritoneal Dialysis (PD)

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.
Procedure
A 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 fluid is drained from the patient. This process is done repeatedly during a 24 hr period or just during the night.

Contraindications
a. Peritonitis
b. Abdominal Surgery
c. Abdominal Adhesions
d. Pregnancy

Complications
a. Peritonitis
b. Respiratory Distress

Chronic Care Needs
Not as many risks as HD. Most common problem is infection of catheter.

Fluids & Electrolytes

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

Total Body Water
60% of body weight (approximately 40L)

a. Intracellular – 67% of total body H₂O
   - Primarily made up of intracellular electrolytes
b. Extracellular – 33% of total body H₂O
   - Plasma Water – 8%, Water, proteins and lipids
   - Interstitial Fluid & Lymph – 20%, Fluid bathing the cells
   - Transcellular Fluid – 7%, Pleural, pericardial, peritoneal, synovial and fluids in secretions (GI, respiratory, salivary)
**Osmolarity**

The concentration of particles within a solution

a. Plasma osmolarity avg. 290 ± 5 mOsm/kg
b. Na⁺ is the primary regulator of extracellular osmolarity
c. K⁺ is the primary regulator of intracellular osmolarity
d. Calculated osmolarity = \(2(\text{Na}^+) + \frac{\text{BG} + \text{BUN}}{18} \times 2.8\)

**IV Fluids**
The most common IV solution used in Med/Surg is D5.45NS with 20mEq KCL because it is most “like” normal fluid in the human body. Typically at 125ml/hr – 3L a day

a. Isotonic Fluids
   - Normal Saline & Lactated Ringers
   - 275 -295 mOsm/L
   - Volume Expanders
   - Tend to stay in intravascular space
b. Hypotonic Fluids
   - .45% NS or less
   - Less than 275mOsm/L
   - Severe Dehydration with Dry Tissues
   - Leak out of vascular space into tissues
c. Hypertonic Fluids
   - 3% NS and above
   - D5WLR
   - D5 .9%NS
   - Greater than 290 mOsm/L
   - Volume Expanders
   - Stay in intravascular space
   - PULL fluid from interstitial space and tissues

**III. ELECTROLYTE BALANCE**

**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 (Mg²⁺), and calcium (Ca²⁺). The anions (negatively charged ions) are chloride (Cl⁻), bicarbonate (HCO₃⁻), sulfate (SO₄²⁻), and phosphate (PO₄³⁻).
The four major functions of electrolytes

a. Regulate Acid Base Balance
b. Maintain Fluid Balance and Osmolarity
c. Distribute the Body Fluid and H₂O between the Compartments
d. Promote Neuromuscular Function/Irritability

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.

<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 (PHO₄³⁻)</td>
<td>Intracellular</td>
<td>2.5 – 4.5 mg/dL I 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 CO₂</td>
<td>Extracellular</td>
<td>22 – 26 mEq/L</td>
<td>4 – 10 mEq/L</td>
</tr>
</tbody>
</table>
## Sodium

<table>
<thead>
<tr>
<th></th>
<th>Hyponatremia</th>
<th>Hypernatremia</th>
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</thead>
<tbody>
<tr>
<td><strong>Fluid Excess</strong></td>
<td>Headache</td>
<td>Restlessness</td>
</tr>
<tr>
<td></td>
<td>Fatigue</td>
<td>Irritability</td>
</tr>
<tr>
<td></td>
<td>Apathy</td>
<td>Lethargy</td>
</tr>
<tr>
<td></td>
<td>Seizures</td>
<td>Seizures</td>
</tr>
<tr>
<td></td>
<td>Confusion → Coma</td>
<td>Confusion → Coma</td>
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<tr>
<td><strong>Fluid Deficit</strong></td>
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<tr>
<td><strong>Sodium Deficit</strong></td>
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<tr>
<td><strong>Sodium Excess</strong></td>
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</tr>
</tbody>
</table>

### Neurological
- Headache
- Fatigue
- Apathy
- Seizures
- Confusion → Coma
- Restlessness
- Irritability
- Lethargy
- Seizures
- Confusion → Coma

### Pulmonary
- Respiratory Distress
- Dyspnea

### Cardiovascular
- Orthostatic Hypotension
- Drop in CVP
- Tachycardia
- Orthostatic Hypotension
- Dry Mucous Membranes
- Dehydration
- Flushed Skin

### GI
- Anorexia
- Wt Loss
- N/V
- Abd Cramps

### GU
- Low Urine Output

### Muscular/Skeletal
- Muscle Weakness
- Muscle Weakness
# Potassium

<table>
<thead>
<tr>
<th>Potassium</th>
<th>Hypokalemia</th>
<th>Hyperkalemia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypokalemia</strong></td>
<td><em>Decreased Intake</em></td>
<td><em>Excess Intake</em></td>
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<tr>
<td></td>
<td><em>Increased Loss</em></td>
<td><em>Decreased Loss</em></td>
</tr>
<tr>
<td></td>
<td><em>Shift of K⁺ into Cells</em></td>
<td><em>Shift K⁺ out of Cells</em></td>
</tr>
<tr>
<td>Neurological</td>
<td>• Lethargy</td>
<td>• Numbness</td>
</tr>
<tr>
<td></td>
<td>• Decreased Reflexes</td>
<td>• Paresthesias</td>
</tr>
<tr>
<td></td>
<td>• Confusion</td>
<td>• Hyporeflexia</td>
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<tr>
<td></td>
<td>• Depression</td>
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</tr>
<tr>
<td>Cardiovascular</td>
<td>• Drop BP</td>
<td>• Conduction Disturbances</td>
</tr>
<tr>
<td></td>
<td>• Dysrhythmias</td>
<td>• V-Fib</td>
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<tr>
<td></td>
<td>• Cardiac Arrest</td>
<td>• Asystole</td>
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<tr>
<td>GI</td>
<td>• Anorexia</td>
<td>• N/V/D</td>
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<tr>
<td></td>
<td>• N/V</td>
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<td>• Distension Ileus</td>
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<tr>
<td>GU</td>
<td>• Dilute Urine</td>
<td>• Oliguria</td>
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<tr>
<td></td>
<td>• Water Loss</td>
<td>• Anuria</td>
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<tr>
<td></td>
<td>• Thirst</td>
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<tr>
<td>Muscular/Skeletal</td>
<td>• Weak</td>
<td>• Early → Irritability</td>
</tr>
<tr>
<td></td>
<td>• Flaccid</td>
<td>• Late → Weakness</td>
</tr>
<tr>
<td></td>
<td>• Respiratory Arrest</td>
<td>• Flaccid Paralysis</td>
</tr>
<tr>
<td>EKG Changes</td>
<td>• Depressed ST segments</td>
<td>• Tall, peaked, tented T waves</td>
</tr>
<tr>
<td></td>
<td>• Flat or inverted T wave,</td>
<td>• Flattened or absent P waves</td>
</tr>
<tr>
<td></td>
<td>• Presence of U waves</td>
<td>• Widening QRS</td>
</tr>
<tr>
<td></td>
<td>• Dysrhythmias, ventricular</td>
<td>• Asystole</td>
</tr>
<tr>
<td></td>
<td>• Cardiac arrest</td>
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</tbody>
</table>

**HyperKalemia Treatment**

Three-Part Therapy

a. 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⁺.

b. Shift K⁺ into the Cell:
   - 1 amp Sodium Bicarbonate
   - 5-10U Regular Insulin
   - 50ml Bolus 50% Dextrose
   - Albuterol 10 – 20mg inhalation or intravenous (beta₂ adrenergic agent – stimulates B₂ receptor in the pancreas to release more insulin).
c. 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 oral administration, and 0.5mEq with rectal administration.
    - Sorbitol prevents constipation.
  - Dialysis can also be utilized to remove $K^+$ from the body

## Calcium

<table>
<thead>
<tr>
<th>Hypocalcemia</th>
<th>Hypercalcemia</th>
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<tbody>
<tr>
<td><em>Excess Loss</em></td>
<td><em>Excess Intake</em></td>
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<tr>
<td><em>Inadequate Intake</em></td>
<td><em>Loss from Bones</em></td>
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<tr>
<td><em>Decreased Ionized</em></td>
<td><em>Mobilization from Bones</em></td>
</tr>
<tr>
<td><em>GI/Bone Absorption</em></td>
<td><em>Acidosis</em></td>
</tr>
</tbody>
</table>

### Neurological
- Tingling $\rightarrow$ Convulsions
- Hyperreflexia
- Dec Reflexes
- Lethargy $\rightarrow$ Coma
- Seizures

### Pulmonary
- Laryngospasm
- Bronchospasm

### Cardiovascular
- Dysrhythmias
- Cardiac Arrest
- Bruising
- Bleeding
- Depressed Activity
- Dysrhythmias
- Cardiac Arrest

### GI
- Increased Peristalsis
- $N/V/D$
- Decreased GI Tract Motility
- $N/V$
- Constipation

### GU
- Kidney Stones
- Flank Pain

### Muscular/Skeletal
- Osteoporosis $\rightarrow$ Fractures
- Abnormal Deposits of Ca in Body Tissues
- Muscle Spasm
- Tetany
- Muscle Fatigue
- Hypotonia
- Bone Pain
- Osteoporosis
- Fractures

### ECG Changes
- Prolonged ST segment
- Prolonged QT interval, torsades de pointes
- Decreased HR
- Short ST/QT
- Heart Blocks
## Magnesium

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<thead>
<tr>
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<td><em>Decreased Intake</em></td>
<td><em>Renal Insufficiency/Failure</em></td>
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<td><em>Impaired Absorption</em></td>
<td><em>Acidosis</em></td>
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Session 5: Gastrointestinal & Renal