AACN CCRN Review

Renal

Presenter: Carol Rauen, RN, MS, PCCN, CCRN, CCNS, CEN
I. Introduction  
AACN 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  
   - BUN  
   - Creatinine  
   - Serum electrolytes  
   - Hgb and Hct  
   - Serum albumin  
   - Serum osmolality  

2. Urine Assessment  
   - Volume and concentration  
   - Urinalysis (see table 1)  
   - 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: >100 diseases can cause RF  
  - Glomerular disease
A. Terms
- Azotemia—nitrogenous waste products in the bloodstream
- Uremic syndrome—systemic and laboratory manifestations of ESRD
- Renal replacement therapy—treatment options

B. Stages of Renal Failure
- Diminished renal reserve
- Renal insufficiency
- 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 One
<table>
<thead>
<tr>
<th>Category</th>
<th>Pre-Renal</th>
<th>Post-Renal</th>
<th>Renal</th>
</tr>
</thead>
<tbody>
<tr>
<td>The problem is not actually with the kidneys but with perfusion (blood flow) to the kidneys</td>
<td>Volume: Dehydration</td>
<td>Ischemia: hypovolemic shock, cardiogenic shock, septic shock, hypoxemia, low cardiac output, heart failure, severe hypertension</td>
<td>Hemodynamic instability, multisystem organ failure, trauma</td>
</tr>
<tr>
<td>The problem is not actually with the kidneys, but after the kidneys</td>
<td>Urethral: stricture, prostatic hypertrophy</td>
<td>Urethral: fibrosis, calculi, blood clots</td>
<td>Bladder: neurogenic problems, neoplasms/cancer, obstruction</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Trauma</td>
</tr>
<tr>
<td>The problem is in the kidney itself effecting function. Kidney diseases</td>
<td>Glomerulus: acute glomerulonephritis, acute cortical necrosis, hepatorenal syndrome</td>
<td>Tubule: acute tubular necrosis, acute pyelonephritis</td>
<td>Nephrotoxins: heavy metals, antibiotics, radiographic contrast media, anesthetics</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Pigments: hemoglobin, myoglobin</td>
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<td></td>
<td></td>
<td></td>
<td>Trauma, intravenous hemolysis, rhabdomyolysis</td>
</tr>
</tbody>
</table>

C. Differentiating Pre-Renal from Renal Diagnosis for ATN

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Pre-renal (hypoperfusion)</th>
<th>Renal (tissue damage)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinary sodium</td>
<td>&lt;20mEq/L</td>
<td>&gt;20 mEq/L</td>
</tr>
<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>
</tbody>
</table>

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**  
  o Fluid loss  
  o Goal: maintain adequate fluid balance and regulate electrolytes  
  o Alteration in electrolytes

• **Recovery Phase**  
  o Goal is supportive care  
  o Prevent further insults  
  o Assessment of renal function  
  o Keep patient well hydrated and free from infection  
  o 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>
<thead>
<tr>
<th>Patient Problem</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| Extracellular volume overload | Restrict NaCl and H₂O diuretics  
<pre><code>                          | Dialysis                                      |
</code></pre>
<p>| Hyponatremia               | Restrict oral H₂O                                |
|                            | Restrict hypotonic IV solutions                   |
| Hyperkalemia               | Restrict K intake                                  |
|                            | Dialysis                                        |
|                            | K binding resins                                 |
|                            | Glucose/Insulin                                  |</p>
<table>
<thead>
<tr>
<th>Condition</th>
<th>Treatment 1</th>
<th>Treatment 2</th>
<th>Treatment 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>Na Bicarb</td>
<td>Dialysis</td>
<td></td>
</tr>
<tr>
<td>Hyperphosphatemia</td>
<td>Restrict PHO₄</td>
<td>Dialysis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Phosphate-binding agents</td>
<td></td>
<td></td>
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<tr>
<td>Hypocalcemia</td>
<td>Calcium carbonate</td>
<td>Calcium gluconate</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Phosphate-binding agents</td>
<td>Dialysis</td>
<td></td>
</tr>
<tr>
<td>Hypermagnesemia</td>
<td>D/C Mg-containing antacids</td>
<td>Dialysis</td>
<td></td>
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<tr>
<td>Nutrition</td>
<td>High protein</td>
<td></td>
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<tr>
<td></td>
<td>Enteral or parental nutrition</td>
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<tr>
<td>Drug dosage</td>
<td>Adjust doses around GFR</td>
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<tr>
<td></td>
<td>Avoid NSAIDs, ACE I, dye, nephrotoxic abx</td>
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</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 semipermeable 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 semipermeable 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 the case 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 patient’s blood and 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 medications
- 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 is only changed periodically, should problems arise.

Procedure: Approximately 2 liters of sterile dialysate are 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 (the entire process is called an exchange). This process is done repeatedly during a 24-hour 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; 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 or two venous cannulation sites are required.

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); 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 those with hemodialysis, and are 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 essential components 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(Na) + BG + BUN
   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\(_4^{=}\)), and phosphate (PO\(_4^{-}\)).

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 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>
</tbody>
</table>
Calcium (Ca\(^{++}\))
- Extracellular: T 8.5–10.5 mg/dL
- 4.0–5.0 mg/dL
- I 0–2 mg/dL

Magnesium (Mg\(^{++}\))
- Intracellular: 1.5–2.5 mEq/L
- 30–40 mEq/L

Phosphate (PO_4^{−})
- Intracellular: 2.5–4.5 mg/dL
- 1.7–2.6 mEq/L
- 100 mEq/L

Chloride (Cl\(−\))
- Extracellular: 96–109 mEq/L
- 1–4 mEq/L

Bicarbonate (HCO_3^{−})
- Extracellular: 22–26 mEq/L
- 4–10 mEq/L

<table>
<thead>
<tr>
<th>C. Sodium: Na(^+) 135–146 mEq/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. <strong>Function</strong>: 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.</td>
</tr>
</tbody>
</table>

| 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**: Include muscle weakness, headache, fatigue, apathy, malaise, orthostatic hypotension, poor skin turgor, weight 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 adrenocortical secretions, diabetes insipidus, strict fluid restrictions, hypothalamic dysfunction, osmotic diuretics, hypercalcemia or hypokalemia, excessive IV infusion of sodium chloride solutions, pregnancy |
**Signs and Symptoms:** Include 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 H\(_2\)O deficit (L) = \( \frac{(.6 \times \text{kg}) \times \text{Na} - 140}{140} \)

Example: 70 kg patient with Na of 160

\( (.6 \times 70) \times \frac{160 - 140}{140} = 42 \times 0.14 = 5.88 \text{ L} \) H\(_2\)O 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

**Hypernatremia**
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:** 10 mL 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
- 5-10 U regular insulin
- 50 mL bolus 50% dextrose
- Albuterol 10–20 mg inhalation or intravenous (beta₂ adrenergic agent—stimulates B₂ 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 1 g will lower the K⁺ 1mEq with oral administration, and 0.5 mEq 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

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

**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**
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 Ca²⁺ = Total Ca²⁺ + 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 Ca²⁺ 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 Ca²⁺ and therefore plays a role in muscle contraction and vasodynamics.

Calcium Regulation: Ca²⁺ 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 Ca²⁺ into and out of the bone, GI tract and kidney
- Vit D is necessary for PHT assistance in Ca²⁺ regulation
- Calcitriol (hormone) stimulates absorption and reabsorption of Ca²⁺
- 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 Ca²⁺ <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), hyperparathyroidism, hypomagnesemia, liver failure, sepsis, pancreatitis, burns, diarrhea, diuretics, malabsorption syndromes, vitamin D deficiency, medications (radiographic contrast, NaHCO₃, protamine, aminoglycosides), inadequate dietary intake of Ca²⁺, hypothyroidism, metabolic bone disease, hyperphosphatemia (including rapid infusion of PHO₄⁻), 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 hyper-reflexia, tetany, seizures, laryngospasm 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 (CN VII) 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 vitamin D, aluminum hydroxide gel for hyperphosphatemia, Mg for hypomagnesemia, monitor patient 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 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, hyper-reflexia, vertigo, seizures, confusion, hallucinations, depression up to coma, increased SVR and hypertension, nausea and vomiting

**Treatment:** IV administration of Mg⁺⁺ with close monitoring: 1–4 g MgSO₄ over 2 minutes to 6 hours (depending on severity of depletion). Common side effects of MgSO₄ administration are flushed feeling or sweating, bradycardia, hypotension and IV site burning

<table>
<thead>
<tr>
<th><strong>Clinical Pearl</strong></th>
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<td>When low Mg⁺⁺ and low K⁺ are both present the patient will be unresponsive to KCl therapy until the hypomagnesaemia is treated</td>
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**Hypermagnesemia: Mg⁺⁺ >2.5mEq/L**

**Cause:** Excess Mg⁺⁺ intake (MgSO₄, 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

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**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