Anatomy and Physiology of Renal System

- Size and shape of each kidney
  - 10-12 cm long
  - 5-6 cm wide
  - 2.5 cm in depth
- Surrounded by mass of fatty connective tissue for protection
- Blood vessels, nerves, and ureters connected via the hilum
- Renal artery branches into 5 smaller arteries that enter hilus of kidney; smaller branches give rise to afferent arterioles
Renal Anatomy

- **Cortex**
  - Cortical area
  - Juxtamedullary area (next to medulla)
  - Contains:
    - Glomeruli
    - Proximal tubules
    - Cortical loops of Henle
    - Distal tubules
    - Cortical collecting ducts

- **Medulla**
  - Contains renal pyramids
    - Medullary loops of Henle
    - Medullary portions of collecting ducts
      - Join to form calyces which further join to become the conduit for urine to enter the ureter

Renal Anatomy

[Diagram of renal anatomy showing the gland and its components]
Renal Anatomy

Nephron

- Functional unit of kidney
- Consists of glomerulus and a tubular structure
- 85% of nephrons originate in superficial part of cortex (cortical nephrons)
- 15% of nephrons originate deeper in the cortex (juxtamedullary nephrons)
  - Longer, thinner loops of Henle
  - Responsible for urine concentration
Nephron: Capillary System

- Glomerular high pressure capillary system between afferent and efferent arterioles
- Peritubular capillary system; a low system originating from the efferent arterioles
  - Surround loop of Henle

- Medullary nephrons have an additional capillary system, the vas recta, consisting of long straight capillaries following the long loops of Henle

Glomerulus of Nephron

- Tuft of capillaries emerging from afferent arterioles
- Contained within Bowman’s capsule
- Blood flows out of glomerular capillaries via the efferent arterioles
- Space within Bowman’s capsule for the filtrate: Bowman’s space
- Basement membrane of the glomerular capillary membrane determines permeability; permeable to water but not to plasma proteins
Tubular Structure of Nephron

- **Proximal convoluted tubule**
  - Majority of all reabsorptive and secretory processes
- **Loop of Henle**
  - Descending limb (thin walled)
  - Ascending limb (thick walled)
  - Impermeable to water
  - Solutes are reabsorbed
  - Loop diuretics work here
  - Filtrate is diluted to allow for excretion of free water
- **Distal convoluted tubule**
  - Thiazide diuretics work here to inhibit sodium reabsorption
  - Aldosterone works on late distal tubule and cortical collecting tubule (below)
- **Collecting tubule or duct**
  - Several distal tubules drain into the collecting tubule
  - Single layer of epithelial cells
  - No further electrolyte absorption or secretion
  - Cortical collecting tubule
    - Aldosterone works here
  - Medullary collecting tubule
    - ADH (vasopressin) works here
    - Responsible for determining concentration and acidity of urine

Glomerular Filtration

- Filtration of protein free plasma through the glomerular capillaries into Bowman’s space.
- The capillary filtration pressure is approximately 60 mm Hg. (Higher pressure than other capillary beds)
- Glomerular filtration produces 125 mL of filtrate each minute.
- Autoregulation.
Definitions

• Azotemia: Accumulation of nitrogenous wastes in the blood
• Renal insufficiency: Reduction in glomerular filtration to 20 to 50% of normal
• Oliguria: U.O. < 400 ml / 24 hours
• Anuria: U.O. < 50 ml / 24 hours

Diagnostic Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine Volume</td>
<td>✓ Less specific&lt;br&gt;✓ Reflects kidney perfusion</td>
</tr>
<tr>
<td>Urine Specific Gravity / Osmolality</td>
<td>✓ Inability to concentrate is early sign of renal dysfunction&lt;br&gt; ✓ Concentrating ability = tubular functioning</td>
</tr>
<tr>
<td>BUN</td>
<td>✓ Not most specific indicator&lt;br&gt;✓ Variations exist in urea load&lt;br&gt; ✓ BUN rises in disproportion to renal function with volume depletion</td>
</tr>
</tbody>
</table>
Diagnostic Parameters

<table>
<thead>
<tr>
<th>Serum Creatinine</th>
</tr>
</thead>
<tbody>
<tr>
<td>✓ Specific for renal function</td>
</tr>
<tr>
<td>✓ Rise may not be evident until 50% GFR is lost</td>
</tr>
<tr>
<td>✓ Creatinine needs to stabilize before an accurate assessment of renal function can be made</td>
</tr>
<tr>
<td>✓ In total loss creatinine will rise 1-2 mg/dL per day and stabilize at 12-15 mg/dL</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Creatinine Clearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>✓ As GFR falls, creatinine excretion is increased and the rise in serum creatinine is less.</td>
</tr>
<tr>
<td>✓ GFR can be overestimated (limitation of creatinine clearance)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Glomerular Filtration</th>
</tr>
</thead>
<tbody>
<tr>
<td>✓ Glomerular filtration determined by estimation of creatinine clearance.</td>
</tr>
<tr>
<td>✓ GFR is usually estimated with the Cockroft-Gault equation: (140-age) x weight (kg) / plasma creatinine x 72 (value multiplied by 0.85 in females)</td>
</tr>
</tbody>
</table>

Acute Kidney Injury

- A sudden loss of the kidneys' ability to excrete wastes, concentrate urine, and conserve electrolytes.
- The definition of acute injury includes one or more of the following that occurs abruptly (within 48 hours):
  - An absolute increase in serum creatinine of more than or equal to 0.3 mg/dL.
  - A percentage increase in serum creatinine of more than or equal to 50%.
  - A reduction in urine output of less than 0.5 ml/kg per hour for more than six hours.
- Occurs in approximately 20% of critically ill patients.
- Mortality ranges from 28% to 90%.
- Variations in statistics exist due to differences in past definitions for acute renal failure.
### Stages of Acute Kidney Injury

<table>
<thead>
<tr>
<th>Stage</th>
<th>Creatinine Criteria</th>
<th>Urine Output Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Increase in serum creatinine of more than or equal to 0.3 mg/dL or increase to more than or equal to 150% to 200%</td>
<td>Less than 0.5 ml/kg per hour for more than 6 hours.</td>
</tr>
<tr>
<td>2</td>
<td>Increase in serum creatinine to more than 200% to 300%.</td>
<td>Less than 0.5 ml/kg per hour for more than 12 hours.</td>
</tr>
<tr>
<td>3</td>
<td>Increase in serum creatinine to more than 300% or serum creatinine of more than or equal to 4.0 mg/dL with an acute increase of at least 0.5 mg/dL.</td>
<td>Less than 0.3 ml/kg per hour for 24 hours or anuria for 12 hours.</td>
</tr>
</tbody>
</table>

### Etiology

- **Outside Hospital**
  - Glomerular nephritis
  - Vasculitis
  - Obstructive Uropathy

- **Inside Hospital**
  - Renal hypoperfusion
  - Drug toxicity
  - Combination of hypoperfusion and drug effect
Signs and Symptoms

- Fatigue
- Confusion
- Twitching or weakness related to metabolic acidosis
- Dry skin
- Edema
- Pallor
- Uremic frost/pruritis
- Flank pain
- Infection

Classifications of Acute Kidney Injury

- Prerenal
- Intrarenal
- Postrenal
## Prerenal AKI

**Causes**
- Decreased intravascular volume
- Decreased cardiac output
- Vasodilation during sepsis
- Bilateral renal vascular obstruction
- Hepatorenal syndrome

**Treatment**

*The treatment of pre-renal AKI is aimed at the rapid reversal of the underlying cause of renal hypoperfusion in order to restore adequate renal perfusion.*

## Diagnostic Parameters for Prerenal AKI

- Positive response to a fluid challenge is diagnostic of pre-renal AKI.
- Oliguria.
- Urinary Sodium < 20 mEq/L.
- Concentrated urine: Urine specific gravity > 1.0150 and urine osmolality > 500 mOsm/L.
- BUN: Creatinine Ratio > 10:1 (usually closer to 20:1). Increased proximal tubular reabsorption of BUN.
- Fractional excretion of sodium (FENa) < 1%
- Fractional excretion of urea (FEurea) < 35%
- Urine Protein 0 or minimal.
- Urine Sediment: Normal or minimally abnormal; hyaline casts, finely granular casts.
Intrarenal AKI: Classifications

- Tubular: Acute Tubular Necrosis (most common cause)
- Glomerular: Glomerulonephritis and small vessel vasculitis.
- Intersitial: Interstitial Nephritis.
- Vascular: Athroembolic disease, large vessel vasculitis.

Acute Tubular Necrosis (Medullary): Etiology

- Nephrotoxic agents
- Prolonged ischemic injury
- Hemolysis or rhabdomyolysis
- Endotoxin release in sepsis
- Hypercalcemia
- Any cause of prerenal AKI that is prolonged  (clinical challenge)
Nephrotoxic Agents

- Aminoglycosides
- Amphotericin B
- Chemotherapy agents
- Cyclosporine
- ACE inhibitors
- NSAIDS
- Contrast agents

Contrast Agents

- Risk reduced by pretreatment with oral n-acetylcysteine
  - 24 to 48 hours before contrast exposure
  - 600 mg BID
- Risk reduced by adequate pre-procedure hydration with 0.9NS or sodium bicarbonate drip
  - 154 mEq of sodium bicarbonate/L at 3 ml/kg for 1 hour prior to procedure
  - Followed by 1 ml/kg/hr for 6 hours post procedure
  - Some evidence that sodium bicarbonate is superior to sodium chloride

ATN Pathophysiology

- Destruction of the tubular epithelial layer of cells
- Often reversible if treatment is promptly initiated.
  - If the tubular basement membrane is damaged from prolonged injury and ischemia, it cannot be regenerated.
- Oliguria develops when tubules become obstructed due to tissue swelling or cellular debris.
- A reabsorption (into circulation) of urine filtrate can occur through damaged tubular epithelium.
- Damaged tubular cells can leak ATP and potassium, and calcium can leak into the cell.
- Scar tissue can form over necrotic areas
ATN Diagnostic Parameters

- Urine Sodium > 20 mEq/L.
- Urine osmolality < 400 mOsmol/L (loss of tubular concentrating ability).
- Bun:Creatinine Ratio 10:1.
- Fractional excretion of sodium (FENa) > 2-3%.
- Fractional excretion of urea (FEurea) > 50%.
- Minimal to moderate proteinuria.
- Urine Sediment: Muddy brown casts, tubular casts, renal epithelial cells.

ATN: Treatment

- Optimizing volume status for prevention
- Avoid all nephrotoxic agents; avoid or dose adjust all medications requiring renal clearance
- A loop diuretic can be used to correct volume overload if the patient is still responsive to diuretics
  - Diuretics are controversial in the treatment of acute kidney injury
- Dopamine, fenoldopam, and mannitol are not indicated
- Treatment is supportive
  - Managing fluid and acid/base balance, electrolytes, and hematologic abnormalities
Glomerular Nephritis (Cortical)

- **Causes**
  - Subacute bacterial endocarditis
  - Post streptococcal infection
  - Systemic lupus erythematosus
  - Drug-induced vasculitis
  - Malignant hypertension

- **Pathophysiology**
  - Cortical involvement from the above causes renal capillary swelling.
  - Edema and cellular debris obstruct the glomeruli, resulting in a decrease the GFR and oliguria.

- **Diagnostic Parameters**
  - Urinalysis will have RBC casts, protein, and leukocytes.
  - BUN to creatinine ratio 10:1 and elevated.

- **Treatment**
  - Immunosuppressant medications.
  - Plasmapheresis.

Interstitial Nephritis (Cortical)

- **Causes**
  - Drug-induced: Allergic nephritis.
    - Common but often unrecognized allergic event in the interstitium of the kidney.
    - Usually in response to a specific drug.
    - May have associated fever, rash, eosinophilia.
  - Bacterial, viral, and other infections.
  - Immune and neoplastic disorders.

- **Diagnostic Parameters**
  - WBC casts with eosinophils.
  - BUN to creatinine ratio 10:1 and elevated.

- **Treatment**
  - Remove the drug that is the causative agent.
  - Steroids may be used.
Postrenal AKI

- **Classifications**
  - Mechanical
    - Urinary calculi
    - Tumor
    - Prostatic hypertrophy
    - Fibrosis
    - Blood clot
    - Retroperitoneal hemorrhage
  - Functional
    - Neurogenic bladder
    - Ganglionic-blocking agents

- **Pathophysiology**
  - Obstruction can increase renal interstitial pressure causing an increased opposing force to GFR.

- **Signs and Symptoms**
  - Abrupt decrease in urine output
  - Urinalysis may show hematuria.

Stages of AKI

**Onset Phase**
- Hours to days
- Renal blood flow and glomerular filtration fall
- Urine output falls
- BUN: Creatinine – Normal or slight increase

**Oliguric / Anuric (Maintenance)**
- 8-14 days
- Decreased GFR
- Urine output < 15 ml/hr (400 cc/24 hours)
- BUN and creatinine increased
- Metabolic acidosis
- Increased potassium
- Water gain with hypertension, dilutional hyponatremia, and pulmonary congestion
- Uremia can develop: Neuromuscular irritability, seizures, coma, death
- High mortality rate
Stages of AKI

Diuretic Phase
- 3 to 4 weeks after onset
- Can last 1-2 weeks
- BUN and creatinine begin to decrease
  - Diuresis may occur before BUN and creatinine fall
- Urine output may exceed 3L/24 Hr: 150-200% of normal
  - Osmotic diuresis from elevated BUN
  - Tubules cannot yet concentrate
  - Fluid losses can jeopardize adequate circulating volume
- Uremic symptoms may not completely resolve because tubular function is not yet normal

Recovery Phase
- Recovery is shorter in non-oliguric renal failure
- Begins with stabilization of laboratory values
- Several months to one year
- BUN: Creatinine almost normal; residual dysfunction may remain
- Urine output returns to normal

Electrolyte Abnormalities in AKI

- Hyperkalemia – most common with oliguric AKI.
- Hyperphosmatemia.
- Hypermagnesemia.
- Hypocalcemia.
- Acidemia
  - The kidneys excrete acid.
  - Oral sodium bicarbonate is typically used to treat.
  - Negative hemodynamic effects have been associated with IV sodium bicarbonate bolus dosing
  - The treatment for severe metabolic acidosis remains controversial.
Uremic Syndrome

- Seen in both acute kidney injury and in chronic renal failure.
- All organs can be affected
- Signs and symptoms can include: nausea, vomiting, pruritis, bleeding, encephalopathy, and pericarditis
- Symptoms not related solely to elevated BUN or creatinine.
- Uremic symptoms warrants aggressive treatment with some type of dialysis therapy.

Treatment of Early Oliguric Kidney Injury

- Eliminate all contributing pre-renal factors
- Rule out postrenal obstructive causes
- A loop diuretic can be used to correct volume overload if the patient is still responsive to it. (Diuretics are controversial)
- Dopamine, fenoldopam, and mannitol are not indicated
- Avoid all nephrotoxic agents; avoid or dose adjust all medications requiring renal clearance.
- Initiate some form of extra corporeal blood therapy early
- Provide meticulous supportive care
- Avoid complications
  - Infection
  - Fluid, electrolyte, and acid/base imbalances
  - Hematologic abnormalities
  - Drug toxicity from drugs metabolized or excreted from the kidney
Treatment of Established Oliguric Kidney Injury

- Modify dose of drugs metabolized or excreted from kidney.
  - Base dose adjustment on an assumed GFR of zero.
- Limit fluid intake to avoid congestion.
- Manage electrolytes
  - Restrict potassium, phosphate, and magnesium intake.
  - Assess for hypotension.
- Prevent complications
  - Infection-related complications are the most common cause of death
    - Nosocomial pneumonia.
    - IV catheter infections.
    - Intra abdominal sepsis.
  - Hemorrhage
    - Uremic toxins inhibit platelets and factor VIII.
    - Factor VIII may need to be replaced.
    - Arginine vasopressin can also increase levels of factor VIII.
- Nutrition
  - Sufficient fat and carbohydrate calories to prevent protein wasting
  - Limit protein if not on dialysis
  - Folate and pyridoxine are lost through dialysis

Avoid corticosteroids (except for interstitial nephritis and some types of renal vasculitis).
  - Catabolic effect
  - Adversely affects immune function

Nursing Considerations

- Maintain skin integrity (uremic effects - high risk for breakdown)
- Prevent infection (infection is major cause of mortality)
  - BUN > 80 to 100 mg/dL is associated with a high risk of infection.
- Nutrition
  - Patients can have accelerated protein catabolism
  - BUN> 100mg/dL - despite routine dialysis).
  - Need higher protein intake.
- Maintain fluid restriction.
- Replace water soluble vitamins
- Monitoring of electrolytes, serum protein, albumin, hematocrit, and BUN and creatinine.
  - Low serum protein and albumin levels have an immunosuppressive effect
Renal Replacement Therapy: Intermittent Hemodialysis

- Central venous access (emergency)
- Arteriovenous grafts or fistulas (chronic)
- Anticoagulation is generally required; non-heparin dialysis is also an option
- Blood pumped through an artificial kidney on one side of the dialysis membrane, while the dialysate (electrolyte) solution flows the opposite direction
- Combines adsorption, diffusion, osmosis, and ultrafiltration
  - Remove fluid and maximal amount of solute (electrolytes, metabolic products, drugs, and toxins)
  - Maximum removal allows for intermittent sessions
- Requires more hemodynamic stability than hemofiltration
- Hypotension is the most common problem
- SLEDD is alternative form of delivery

Dialysis Equilibrium Syndrome

- From shifts in extracellular compartment
- Nausea, vomiting, confusion, seizures, coma.
- Most common in first dialysis session with high BUN.
- Treatment.
  - Decreased dialysis time.
  - Decreased dialysis flow rates.
  - Dialyzer with smaller surface area.
  - Sodium chloride, dextrose, mannitol.
Renal Replacement Therapy: Peritoneal Dialysis

- Slow form of dialysis - exchange of fluids and solutes between the peritoneal cavity and peritoneal capillaries
- Utilizes diffusion
- Less efficient than hemodialysis
- No need for vascular access
- No significant hemodynamic effects
- 1 to 3 L of solution with dwell time of 30 to 40 minutes
- Osmotic gradient for fluid removal
  - Hyperosmolar glucose concentrations
- Complications
  - Abdominal distention and increased work of breathing
  - Pleural effusion
  - Hyperglycemia
  - Peritonitis

Renal Replacement Therapy: CEBT

Ultrafiltration

- SCUF – Slow continuous ultrafiltration
- Fluid moves through a semipermeable membrane via a pressure gradient (higher pressure gradient creates more fluid removal)
- Results are primarily fluid removal
- Hemofiltration, hemodialysis, and hemodiafiltration all use ultrafiltration as a component of therapy
- Adsorption is another principle involved in all 4 therapies
  - Clinging of positively charged molecules to the negatively charged membrane of the filter.
  - Filter can become clogged with molecules. The removal of these molecules from systemic circulation is a benefit of CEBT therapy.
Renal Replacement Therapy: CEBT

**Hemofiltration**
- CVVH – Continuous veno-venous hemofiltration
- Uses convection to remove solutes
  - Process of solute removal by solvent drag
  - More fluid through semi permeable membrane = more solute removed.
  - Replacement solution is used to create solvent drag
  - Faster rate of replacement solution = more solvent drag
- Convection removes medium and large molecules
- Solute removal is slow so the process must be continuous
- Fluid removal still exceeds solute removal
- Less likely than hemodialysis to produce hypotension
- Some medications are cleared via hemofiltration and require a dose adjustment
  - Dose adjusted based on an assumed creatinine clearance of approximately 14 ml/minute

Renal Replacement Therapy: CEBT

**Hemodialysis**
- CVVHD – Continuous veno-venous hemodialysis
- Uses dialysate solution to create selective diffusion of electrolytes
  - Excellent technique for the removal of small particles
- Hemodialysis removes both solutes and fluid
- Often used on patients who are chronic dialysis
- Provides more hemodynamic stability than intermittent hemodialysis
- Allows fluid overloaded critically ill patients to receive a higher caloric intake
Renal Replacement Therapy: CEBT

**Hemodiafiltration**
- CVVHDF – Continuous veno-venous hemodiafiltration.
- Uses both hemodialysis and hemofiltration
- Allows for the removal of small, medium, and large molecules.

**CEBT**
- A venous to venous connection with a double lumen venous catheter
- Jugular, subclavian, and femoral veins can be used.
- Venous-only access avoids the risk of limb ischemia associated with arterial access.
- Extra corporeal pump is used to create flow through the system.
- Filtration is ineffective when MAP fall below 60mmHg.
- Equipment
  - Blood filter, blood pumps, circuit tubing, dialysate and replacement infusion tubing, anticoagulant tubing, and a collection bag.
<table>
<thead>
<tr>
<th>Therapy</th>
<th>Principles</th>
<th>Replacement Solution</th>
<th>Dialysate Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCUF</td>
<td>Ultrafiltration Adsorption</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>CVVHD</td>
<td>Ultrafiltration Adsorption Diffusion</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>CVVH</td>
<td>Ultrafiltration Adsorption Convection</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>CVVHDF</td>
<td>Ultrafiltration Adsorption Diffusion Convection</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>
### Criteria for Intermittent Dialysis

- Volume overload in presence of oliguria or anuria
- Uncontrolled hyperkalemia, hyperphosphatemia, hypermagnesemia
- Life threatening acidosis
- Life threatening drug overdoses or toxicity requiring dialysis
- Symptomatic uremia
  - Nausea and vomiting
  - Bleeding
  - Pericarditis
  - Seizures, coma
- BUN 80-100 mg/dL
- Creatinine 10 mg/dL

### Criteria / Candidates for CEBT

- Hemodynamically unstable patients with criteria for intermittent hemodialysis
- Patients with increased ICP who need dialysis
- Critically ill patients with early signs of AKI
- Nontraditional indications:
  - Hyperthermia
  - Rhabdomyolysis
  - Systemic inflammatory response syndrome,
  - Fluid management in the hemodynamically unstable patient without renal failure
Nursing Considerations in CEBT

- Hypothermia
  - Warming blankets
  - Use of warmers as part of the ECBT equipment

- Coagulation
  - Clotting versus clogging
  - Heparin most commonly used
  - Alternative is to use a technique that includes the use of a replacement solution. Use of replacement solution results in continuous dilution of the hematocrit.

- Cardiac arrhythmias
  - Fluid and electrolyte imbalance
  - Equipment can cause ECG artifact that mimics cardiac arrhythmias
  - Equipment should be temporarily stopped so the patient’s rhythm can be reassessed

Electrolytes: Overview

- Abnormalities often occur in groups and symptoms can be from mixed disorders
- Treatment is focused on immediate crisis and underlying cause
- Suspect electrolyte abnormalities:
  - Renal disease
  - Endocrine disease
  - Acute change in mental status
  - Ventricular arrhythmias
Sodium

- Dominant extracellular cation
- Primary determinant of serum osmolality
- Sodium is closely related to water (regulates ECF)
- Factors to consider when assessing sodium abnormalities:
  - Serum and urine osmolality
  - Intravascular volume status / presence of edema
  - Serum albumin, lipids, and glucose
  - Medications and IV fluids
  - Renal function

Hyponatremia:
Sodium < 135 mEq/L

- S&S related to rapidness of onset and severity
  - Signs and symptoms can also be related to fluid balance.
  - Primary effects are CNS related.
- Cognitive and motor function changes @ < 125 mEq/L
- Permanent changes at levels < 110 mEq/L
- 50% mortality when < 105 mEq/L

- Muscle cramps
- Twitching / tremors
- Muscle weakness
- N&V / abdominal cramps
- Headache
- Irritability / personality changes
- Confusion
- Lethargy progressing to coma
- Seizures
Hypotonic Hyponatremia

- Most common form of hyponatremia
- Results in intracellular hypoosmolar state (creates S&S)
- Occurs as a result of excess free water in relation to sodium
- Patients can be:
  - Hypovolemic
  - Isovolemic *most common form
    - SIADH
  - Hypervolemic

Treatment for Hyponatremia

- Sodium should be corrected to an initial level of 120 to 130 mEq/L
  - Over 12 to 24 hours
  - No more than 8-12mEq / 24 hours unless life threatening symptoms
- Correct at rate proportional to development (slower rate for chronic hyponatremia)
- Free water restriction for levels > 125 to 130 mEq/L

Caution: Osmotic demyelinating syndrome
### Hypernatremia

**Definition:** Sodium greater than 145 mEq/L with a serum osmolality > 295 mOsm/kg. *Most cases of hypernatremia involve a hyperosmolar state.*

- Rarely occurs in patients with:
  - Normal ADH secretion
  - Thirst mechanism
  - Ability to consume free water

- Almost always causes cellular dehydration.

**Treatment**

- Correct underlying cause
- Decrease 0.5 to 1.0 mEq/L per hour
  - Replacement of free water with D5W or 0.2 or .45 NS
  - Normal saline may be used if hemodynamically unstable (adequate circulating volume is priority)
  - Loop diuretics or dialysis rarely needed

*Caution – Cerebral Edema*

### Hypernatremia

**Causes**
- Conditions with limited ability to consume free water (ICU setting)
- Hypertonic tube feedings
- Dehydration (burns, tachypnea, hyperthermia)
- Osmotic diuretics with excessive free water clearance.
- Diabetes mellitus
- Diabetes insipidus

**Signs and Symptoms**
- Thirst (early symptom)
- Urine output decreases and urine osmolality increases due to renal water conservation
- Dry mouth and skin
- Increased body temperature
- Muscle weakness
- Headache
- Irritability and agitation
- Seizures
- Coma
Potassium

- 95% or > of potassium is intra cellular
- Majority of potassium contained in muscle
  - Declines with age due to decrease in muscle mass
- Dietary intake is the major source / kidneys responsible for excretion
- Ratio of extracellular to intracellular important for electrical membrane potentials
- Major body systems impacted by abnormalities:
  - GI
  - Neuromuscular
  - Cardiac

Nerve impulse and muscular function transmission dependent on potassium.

Hypokalemia: Causes

- K+ less than 3.5 mEq / L (total body deficit of 5-10%)
- Causes:
  - Poor K+ intake
  - Increased GI loss (not usually cause of symptomatic imbalance)
  - Increased renal loss
    - Renal tubular acidosis
    - Diuretics
    - Excess mineral or glucocorticoids (aldosterone)
    - Low magnesium
    - Certain antibiotics

- Extracellular to intracellular shifts
  - Alkalosis (potassium exchanged for hydrogen ions)
    - also causes increased renal loss
  - Insulin
  - Treatment of DKA or HHNK
    - Insulin
  - Beta adrenergic agonists

- Note: Does not reflect total body potassium - Correct with caution

- Caution with hypokalemia in presence of acidosis.
Hypokalemia: Signs and Symptoms

- Symptoms occur when $K^+ < 3.0$ mEq/L
- Severity dependent on:
  - Rapidness of onset
  - Systemic pH
  - Calcium level
- S&S related to altered membrane potentials and impaired muscle contractility
  - Increase in resting membrane potential of neuronal and muscular cells
  - Reduces excitability
- GI
  - Orthostatic hypotension
  - Parasthesias, weakness, fatigue and muscle cramps
    - Lower extremities are typically impacted first
  - Respiratory muscle weakness, dyspnea, paralysis and arrest ($< 2.5$ mEq/L)
- Enhanced digitalis effect
- Severe hypokalemia can result in rhabdomyolysis

Hypokalemia: ECG Changes

- Mild hypokalemia: delays ventricular repolarization
  - ST depression, inverted T wave
  - Heightened U waves, prolonged QT interval
- Lowered threshold for ventricular fibrillation and reentrant tachycardias
- Any arrhythmia
- Severe hypokalemia
  - Increased PR interval
  - Increased QRS interval
Hypokalemia: Treatment

- Treat cause
- Correct alkalosis
- Correct hypomagnesemia
- Increased potassium intake (dietary or supplement) if potassium ≥ 3.0 mEq/L
  - Foods high in potassium: orange juice, bananas, raisins, milk, green vegetables
  - Oral supplements up to 40 mEq can be used safely several times per day.
- Add potassium to maintenance IV fluid
- IV potassium bolus for severe deficiency (less than 3.0 mEq /L if on digoxin, symptoms related to hypokalemia, or less than 2.5 mEq /L without symptoms)
  - Non glucose solution
  - Safe dosage: 10 mEq / 100 cc over 1 hour
  - May give 20 mEq over 1 hour if K+ is < 3.5 mEq /L (higher doses if life threatening)
  - Concentration should not exceed 10 mEq per 100 ml via peripheral line or 20 mEq per 100 ml if central line

Note: Replace cautiously in those with impaired ability to excrete.

Hyperkalemia:

K+ greater than 5.0 mEq / L

- Rarely occurs in healthy people
- Impaired potassium management:
  Renal Disease
  Diabetics

- Decreased Excretion
  - Renal disease
    - Decreased renal perfusion
    - Sickle cell disease
  - Decreased aldosterone
    - Addison’s
    - Diabetes
    - Drugs inhibiting aldosterone (aldactone, ACE-I, ARBs, Non steroidal antinflammatories, Heparin)

- Increased Intake
  - Salt substitutes
  - Supplements
  - High dose penicillin with K+ (higher doses if life threatening)
  - Lactated ringers
  - Transfusion of banked blood
### Hyperkalemia: Causes

- **Cellular disruption with leak of intracellular K+**
  - Crush injuries
  - Rhabdomyolysis
  - Hemolysis (blood transfusion reaction)
  - Early burns
  - Trauma
  - Large hematoma
  - Severe catabolic state
  - Lysis of tumor cells (chemotherapy)

- **Intracellular to extracellular shift**
  - Metabolic acidosis
  - Hypertonic glucose with insulin deficiency
  - Hyperosmolality
  - Digitalis toxicity
  - Depolarizing neuromuscular blocking agents
  - Beta blockers

### Hyperkalemia: Signs and Symptoms

**Symptoms when K+ > 6.0 mEq/L**
- Skeletal muscle effects when K+ > 7.0 mEq/L
- Neuromuscular effects complicated by acidosis, low sodium, low calcium, high magnesium
- Parathesia
- Lower extremity weakness
- Hypotension

**EKG Changes**
- Tall narrow peaked T waves
- Wide QRS
- Prolonged PR and flattened to absent P wave
- Dysrhythmias
  - √ Bradycardia / heart block
  - √ Sine wave pattern
  - √ Asystole
Hyperkalemia: Treatment

- Level > 6.0 mEq/L should be treated. Urgency based on clinical manifestations.
- Limit K+ intake
- Volume expansion
- K+ > 6.5 or dysrhythmias
  - Stabilize cardiac membrane with calcium chloride
    - Not if digitalis toxic
  - Shift potassium into cell
    - 50% Dextrose and insulin (50 ml and 10 units)
    - High dose inhaled beta agonists (synergistic)
    - Sodium bicarbonate to correct acidosis

Hyperkalemia: Treatment

- Kayexalate is an exchange resin
  - Exchange sodium for K+ and moves K+ out via the GI tract
  - Can be given orally or as retention enema
- Oral dose is administered in sorbitol
  - Sorbitol orally acts as osmotic laxative
- Retention enema is administered in dextrose
  - Sorbitol can cause intestinal necrosis when given by enema
- Loop diuretics if functioning kidneys
- Dialysis if renal dysfunction
QUESTIONS??

Thanks for Attending
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You may contact us at www.cardionursing.com
The moment you stop learning ..... You stop leading.