

# CMC Review Course NTI 2009 New Orleans

## Renal / Electrolytes / Ischemic Stroke

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A Ship in the  
Harbor is Safe .....

But that is not what  
ships are built for.

Let us begin our adventure!

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## 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

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## 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

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## 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

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## 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

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## 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*

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## 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 used if hemodynamically unstable (adequate circulating volume is priority)
  - Loop diuretics or dialysis rarely needed

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## 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

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## 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.

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## Hypokalemia: Causes

- K<sup>+</sup> less than 3.5 mEq / L (total body deficit of 5-10%)
- Causes:
  - Poor K<sup>+</sup> intake
  - Increased GI loss (not usually cause of symptomatic imbalance)
  - Increased renal loss
    - Renal tubular acidosis
    - Diuretics
    - Excess mineral or glucocorticoids
    - **Low magnesium**
    - Certain antibiotics
- Extracellular to intracellular shifts
  - Alkalosis (potassium exchanged for hydrogen ions)
  - Insulin
  - Treatment of DKA or HHNK
  - Beta adrenergic agonists
- Note: Does not reflect total body potassium - Correct with caution
- *Caution with hypokalemia in presence of acidosis.*

Urinary K<sup>+</sup>: High with renal loss; low with other causes

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## Hypokalemia: Signs and Symptoms

- Symptoms occur when K<sup>+</sup> < 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 (N&V, cramping)
- 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

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## 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
- Most any arrhythmia
- Severe hypokalemia
  - Increased PR interval
  - Increased QRS interval

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## Hypokalemia: Treatment

- Treat cause
- Correct alkalosis
- Correct hypomagnesemia
- Increased potassium intake (dietary or supplement) if potassium  $\geq 3.0$  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<sup>+</sup> 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.<sup>14</sup>

## 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 antiinflammatories, Heparin)

### ■ Increased Intake

- Salt substitutes
- Supplements
- High dose penicillin with K<sup>+</sup>
- Lactated ringers
- Transfusion of banked blood

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## Hyperkalemia: Causes

### ■ Cellular disruption with leak of intracellular K<sup>+</sup>

- 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

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## 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

Parathesias

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

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## 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

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## Hyperkalemia: Treatment

- Kayexalate is an exchange resin
  - Exchange sodium for K<sup>+</sup> and moves K<sup>+</sup> out via the GI tract
  - Can be given orally or as retention enema
- Oral dose is administered in sorbital
  - Sorbital orally acts as osmotic laxative
- Retention enema is administered in dextrose

- Loop diuretics if functioning kidneys
- Dialysis if renal dysfunction

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## Calcium

- Less than 50% of dietary intake is absorbed.
- The majority of the body's calcium is in the bone.
- Serum level regulated by parathyroid levels and vitamin D.
  - Also influenced by serum phosphate levels (inverse relationship), albumin levels, and blood pH.
  - Calcium in bone can be exchanged to maintain extracellular levels.
- There are 3 types of serum calcium:
  - > 40% of calcium is protein bound (mostly albumin)
  - 10% is chelated (non-ionized) with substances such as citrate or phosphate
  - 50% is ionized (free to leave the extracellular fluid and participate in intracellular function)

Important for several key processes:

- ✓ **Muscle contraction**
- ✓ **Transmission of nervous system impulses**
- ✓ Hormone secretion
- ✓ Blood clotting and wound healing
- ✓ Cellular function

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## Hypocalcemia

- Calcium < 8.8 mg / dL or ionized calcium < 4.65 mg / dL.

**Common disorder in critical care.**

**Generally asymptomatic if development is slow or if ionized calcium remains normal.**

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## Hypocalcemia: Causes

Decreased calcium intake or absorption	Increased calcium excretion	Impaired ability to mobilize calcium from bone stores	Increased calcium binding; Increased calcium chelation (decreased ionized calcium)
Low dietary intake Hypomagnesemia Renal failure Vitamin D deficiency Liver disease Steroid therapy Cushing's disease	Diuretic therapy Chronic Diarrhea Hyperphosphatemia (phosphate elimination is impaired in renal failure)	Inadequate levels of parathyroid hormone (Decreased magnesium inhibits parathyroid release)	Alkalosis Acute Pancreatitis Drugs Cimetidine Heparin Theophylline Aminoglycosides Citrate anticoagulation used in bank blood

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## Hypocalcemia: Signs and Symptoms

Most common due to neuromuscular irritability.

- Parathesias (common)
- Hyperreflexia
- Tetany (spasms of face, hands, and feet)
- **Chvostek's sign**
  - Tapping of face over facial nerve located below the temple
  - Positive sign results in spasm of lip, nose or face.
- **Trousseau's sign**
  - Inflate blood pressure above systolic BP and hold for 3 minutes
  - Positive sign results in contraction of fingers or hand.
- Stridor / wheezing / bronchospasm
- For severe deficit: laryngeal spasm, change in mental status, seizures
- Chronic: dry skin and hair and brittle nails; bone pain and risk of fracture

■ Cardiovascular effects:

- Decreased contractility
- Hypotension
- **Prolonged QT (ST segment hugging baseline for extended period)**
- Torsades de pointes
- Bradycardia / heart block
- **Digitalis insensitivity**
- Heart failure
- Cardiac arrest

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## Hypocalcemia: Treatment

- Goal: Low-normal range
- High calcium, low phosphorous diet
- Vitamin D supplements if deficiency
- Phosphate binding antacids \*
- Magnesium for hypomagnesemia
- Correct alkalosis (increases ionized Ca<sup>++</sup>)
- Thiazide diuretics (increase tubular calcium reabsorption)
- IV calcium chloride or calcium gluconate

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## IV Calcium Administration

- Calcium gluconate
  - Give 10 ml
  - 10 ml contains 4.5 mEq of calcium
- Calcium chloride
  - Give 3-4 ml
  - 10 ml contains 13.6 mEq of calcium

√Administer no faster than 1 ml per minute

√May cause sloughing or necrosis (central vein preferred)

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## Hypercalcemia

- Calcium > 10.4 mg / dL or ionized calcium > 5.26 mg / dL
- Causes:
  - Increased calcium intake (supplement or antacids)
  - Increased calcium absorption (hypophosphatemia, excessive vitamin D)
  - Increased mobilization of calcium from the bone (Vitamin D excess, immobility, hyperparathyroidism, thyroidtoxicosis, neoplasms)
  - Acidosis (increased ionized calcium)
  - Decreased calcium excretion (thiazide diuretics)

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## Hypercalcemia: Signs and Symptoms

- Hypophosphatemia
- Signs and symptoms related to dehydration
- Gastrointestinal symptoms (slowing of GI tract)
- Bone and flank pain / osteoporosis / pathological fractures
- Muscular symptoms: Hypotonicity / weakness / fatigue
- Neurological symptoms: Decreased mentation, agitation, comma, seizures.
- Calcium salts form at high levels
  - Pruritis from skin deposits.
  - Renal calculi and potential kidney injury
  - Deposits on the aorta, cardiac valves, and coronary arteries.

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## Hypercalcemia: Signs and Symptoms

- Cardiac symptoms:
  - hypertension (may be offset by co-existing dehydration)
  - cardiac ischemia
  - shortened QT segments
  - arrhythmias (conduction abnormalities)
  - ***digitalis toxicity***
- Life threatening signs and symptoms are rare unless calcium levels reach > 14 mg/dL.

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## Hypercalcemia: Treatment

- Primary Treatment: Rehydration with 0.9 NS
- Decrease calcium absorption
  - Low calcium, high phosphorous diet
  - Glucocorticoids
- Increase calcium excretion
  - Fluids (0.9NS)
  - Loop diuretics
  - Dialysis if renal failure or life threatening
  - Inhibit bone resorption (calcitonin, mithramycin, biphosphonates)
- Prevent cardiac effects
  - Calcium Channel Blockers
- Prevent renal calculi
  - Acidify urine

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## Low Magnesium: < 1.5 mEq/dL

- |  |  |
|--|--|
| <ul style="list-style-type: none"> <li>■ Common disorder in hospitalized patients</li> <li>■ Most common causes renal and GI loss                             <ul style="list-style-type: none"> <li>– Transcellular shift after hypothermia</li> </ul> </li> <li>■ Low magnesium = increased digitalis effect</li> <li>■ EKG: Prolonged QT and Torsades de pointes</li> <li>■ <b><i>Neuromuscular irritability and decreased ability to relax neuromuscular tone</i></b></li> </ul> | <ul style="list-style-type: none"> <li>■ <b>May induce hypokalemia and hypocalcemia</b></li> <li>■ Signs and symptoms overlap with hypokalemia and hypocalcemia (often concurrent)</li> <li>■ Oral magnesium can cause diarrhea and further lower magnesium levels</li> <li>■ IV 1 to 2 grams over 10 – 60 minutes                             <ul style="list-style-type: none"> <li>– More rapidly if life threatening</li> <li>– Decrease if kidney injury present</li> </ul> </li> </ul> |
|--|--|

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## High Magnesium: > 2.5 mEq/L

- Uncommon disorder outside kidney dysfunction
- S & S
  - Hyporeflexia
  - Hypotension
  - Heart block/bradycardia
  - Muscle weakness
  - Change in mental status
  - Lethargy/coma
  - Cardiopulmonary arrest

Treat with fluids and diuretics if normal renal function.

Dialysis if renal failure.

Clinical application: SE of IV magnesium administration is hypotension.

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## Low Phosphorous: < 2.5 to 3.0 mg/dL

- Easily lost from RBCs and skeletal muscle but levels well preserved in cardiac muscle
- Various neuromuscular and central nervous system effects related to depleted intracellular stores
- Enteral replacement preferred if not life threatening
- Parenteral sodium phosphate or potassium phosphate if severe
  - Dose .6mg/kg/hr to .9mg/kg/hr
  - Observe for signs of hypocalcemia
  - Replace with caution since predominantly intracellular

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## High Phosphorous: > 4.5 mg/dL

### ■ Causes:

- Renal dysfunction
- Laxatives with phosphate
- Hypocalcemia
- Increased cellular release

### ■ Same clinical signs as hypocalcemia

#### **Treat hypocalcemia**

**Aluminum antacids bind with phosphate**

**Acetazolamide to increase urinary excretion**

**Dialysis if due to renal failure**

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## Nephron

### ■ Functional unit of kidney

#### ■ Glomerulus

- 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

- Proximal convoluted tubule
- Loop of Henle
- Distal convoluted tubule
- Collecting tubule

#### ■ 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

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## 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
  - MAP 80-180
- Constriction of the afferent arterioles decreases glomerular pressure and filtration.
- Constriction of the efferent arterioles increases glomerular pressure and filtration.
- Both the afferent and efferent arterioles are innervated by the sympathetic nervous system. They are also sensitive to vasoactive hormones such as angiotensin II.
- *Clinical Application*
- In shock, afferent arterioles constrict (SNS stimulation) and glomerular filtration and urine output can fall to near zero.

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## 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

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## Diagnostic Parameters

<b>Urine Volume</b>	<ul style="list-style-type: none"> <li>✓Less specific</li> <li>✓Reflects kidney perfusion</li> </ul>
<b>Urine Specific Gravity / Osmolality</b>	<ul style="list-style-type: none"> <li>✓Inability to concentrate is early sign of renal dysfunction</li> <li>✓Concentrating ability = tubular functioning</li> </ul>
<b>BUN</b>	<ul style="list-style-type: none"> <li>✓Not most specific indicator</li> <li>✓Variations exist in urea load</li> <li>✓BUN rises in disproportion to renal function with volume depletion</li> </ul>

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## Diagnostic Parameters

<b>Serum Creatinine</b>	<ul style="list-style-type: none"> <li>✓Specific for renal function</li> <li>✓Rise may not be evident until 50% GFR is lost</li> <li>✓Creatinine needs to stabilize before an accurate assessment of renal function can be made</li> <li>✓In total loss creatinine will rise 1-2 mg/dL per day and stabilize at 12-15 mg/dL</li> </ul>
<b>Creatinine Clearance</b>	<ul style="list-style-type: none"> <li>✓ As GFR falls, creatinine excretion is increased and the rise in serum creatinine is less.</li> <li>✓ GFR can be overestimated (limitation of creatinine clearance)</li> </ul>
<b>Glomerular Filtration</b>	<ul style="list-style-type: none"> <li>✓Glomerular filtration determined by estimation of creatinine clearance.</li> <li>✓GFR is usually estimated with the Cockcroft-Gault equation: (140-age) x weight (kg) / plasma creatinine x 72 (value multiplied by 0.85 in females)</li> </ul>

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## 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

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## Stages of Acute Kidney Injury

Stage	Creatinine Criteria	Urine Output Criteria
1	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%	Less than 0.5 ml/kg per hour for more than 6 hours.
2	Increase in serum creatinine to more than 200% to 300%.	Less than 0.5 ml/kg per hour for more than 12 hours.
3	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.	Less than 0.3 ml/kg per hour for 24 hours or anuria for 12 hours.

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## Etiology

### ■ Outside Hospital

- Glomerular nephritis
- Vasculitis
- Obstructive Uropathy

### ■ Inside Hospital

- Renal hypoperfusion
- Drug toxicity
- Combination of hypoperfusion and drug effect

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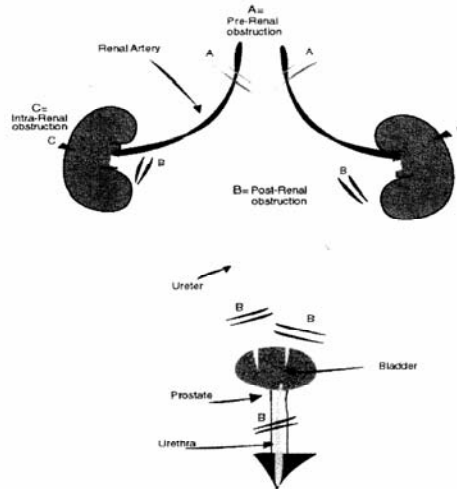
## Signs and Symptoms

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

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## Classifications of Acute Kidney Injury

- Prerenal
- Intrarenal
- Postrenal



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## 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.*

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## Diagnostic Parameters for Prerenal AKI

- Positive response to a fluid challenge is diagnostic of prerenal 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.

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## 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

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## 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)

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## Nephrotoxic Agents

- Aminoglycosides
- Amphotericin B
- Chemotherapy agents
- Cyclosporine
- ACE inhibitors
  - If hypotension is contributing factor
- 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

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## 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

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## 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.

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## 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

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## Glomerularnephritis (Cortical)

- Causes
  - Subacute bacterial endocarditis
  - Post streptococcal infection
  - Systemic lupus erythematosus
  - Drug induced vasculitis
  - Malignant hypertension
- Diagnostic Parameters
  - Urinalysis will have RBC casts, protein, and leukocytes
  - BUN to creatinine ratio 10:1 and elevated
- 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
- Treatment
  - Immunosuppressant medications
  - Plasmapheresis

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## 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

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## 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.

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## 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

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## 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

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## Electrolyte Abnormalities in AKI

- Hyperkalemia – most common with oliguric AKI
- Hyperphosphatemia
- 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.

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## 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.

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## 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

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## Treatment of Established Oliguric Kidney Injury

- |  |   |
|--|---|
| <ul style="list-style-type: none"> <li>■ Modify dose of drugs metabolized or excreted from kidney                     <ul style="list-style-type: none"> <li>▪ Base dose adjustment on an assumed GFR of zero.</li> </ul> </li> <li>■ Limit fluid intake to avoid congestion</li> <li>■ Manage electrolytes                     <ul style="list-style-type: none"> <li>▪ Restrict potassium, phosphate, and magnesium intake.</li> <li>▪ Assess for hyponatremia</li> </ul> </li> <li>■ Prevent complications                     <ul style="list-style-type: none"> <li>– Infection-related complications are the most common cause of death                             <ul style="list-style-type: none"> <li>▪ Nosocomial pneumonia.</li> <li>▪ IV catheter infections.</li> <li>▪ Intra abdominal sepsis.</li> </ul> </li> <li>– Hemorrhage                             <ul style="list-style-type: none"> <li>▪ Uremic toxins inhibit platelets and factor VIII.</li> <li>▪ Factor VIII may need to be replaced.</li> <li>▪ Arginine vasopressin can also increase levels of factor VIII.</li> </ul> </li> </ul> </li> </ul> | <ul style="list-style-type: none"> <li>■ Nutrition                     <ul style="list-style-type: none"> <li>– Sufficient fat and carbohydrate calories to prevent protein wasting</li> <li>– Limit protein if not on dialysis</li> <li>– Folate and pyridoxine are lost through dialysis</li> </ul> </li> <li>■ Avoid corticosteroids (except for interstitial nephritis and some types of renal vasculitis).                     <ul style="list-style-type: none"> <li>– Catabolic effect</li> <li>– Adversely affects immune function</li> </ul> </li> </ul> |
|--|---|

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## 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

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## 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

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## 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.

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## 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

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## 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.

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## Renal Replacement Therapy: CEBT

### Hemofiltration

- CVVH – Continuous veno-venous hemafiltration
- 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

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## 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

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## 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.

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## 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.

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Therapy	Principles	Replacement Solution	Dialysate Solution
SCUF	Ultrafiltration Adsorption	No	No
CVVHD	Ultrafiltration Adsorption Diffusion	No	Yes
CVVH	Ultrafiltration Adsorption Convection	Yes	No
CVVHDF	Ultrafiltration Adsorption Diffusion Convection	Yes	Yes

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## 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

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## 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

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## 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

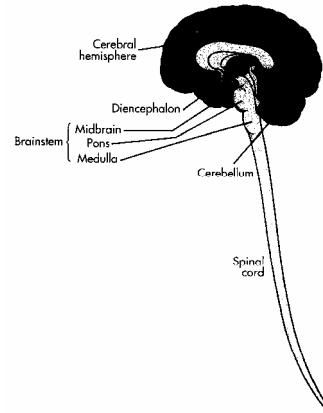
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## Neuro Anatomy

- Cerebral hemispheres
- Diencephalon
  - Thalamus
  - Hypothalamus
  - Limbic system
- Brainstem
  - Midbrain
  - Pons
  - Medulla
- Cerebellum



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## Cerebral Hemisphere Key Functions

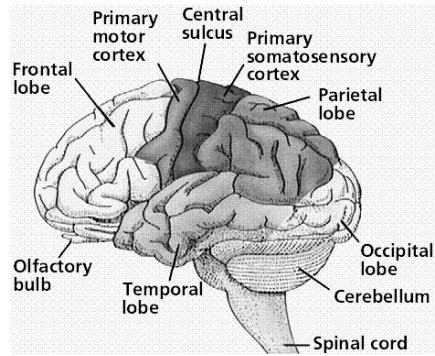
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|--|--|
| <ul style="list-style-type: none"><li>■ Left<ul style="list-style-type: none"><li>– Analysis</li><li>– Problem solving</li><li>– Language</li><li>– Mathematics</li><li>– Abstract reasoning</li></ul></li></ul> | <ul style="list-style-type: none"><li>■ Right<ul style="list-style-type: none"><li>– Spatial relationships</li><li>– Non verbal communication</li><li>– Music</li><li>– Artistic ability</li></ul></li></ul> |
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Corpus Callosum

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## Cerebral Lobes: Key Functions

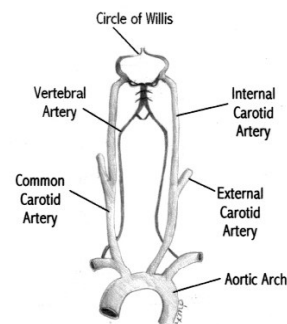
- **Frontal Lobe:** Voluntary motor function, intellectual function, personality
- **Temporal:** Memory function and emotion
- **Parietal:** Sensory function, object recognition and position sense, body awareness and image
- **Occipital:** Visual reception



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## Cerebral Circulation

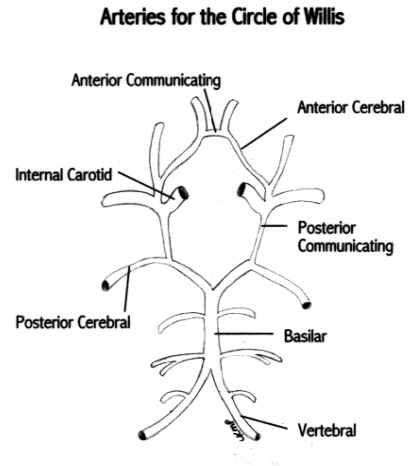
- Two internal common carotid arteries anteriorly
  - Provide the major blood supply to the brain
  - Arise from the common carotids
  - Supply optic nerves, retina, and the majority of the cerebral hemispheres
  - Divides into: Anterior cerebral artery and middle cerebral artery
- Two vertebral arteries posteriorly
  - Arise from right and left subclavian arteries.
  - Merge to form basilar artery; basilar artery divides into two posterior cerebral arteries.
  - Supplies the cervical cord, brainstem, medulla, cerebellum, caudal part of diencephalons, medial and posterior temporal lobes, and the occipital lobes



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# Cerebral Circulation

- Circle of Willis - an anatomical ring of vessels joining the carotid artery system and the vertebrobasilar system.
  - Posterior communicating artery.
  - Posterior cerebral artery.
  - Anterior communicating artery.
  - Anterior cerebral artery.



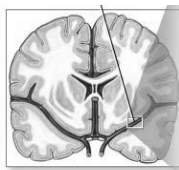
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# Anatomy Application



## Middle Cerebral Artery

Most common intracerebral vessel affected by stroke



### Symptoms of TIA

Factor	Carotid	Vertebrobasilar
Loss of vision	✓	✓
Weakness	✓	✓
Numbness or tingling	✓	✓
Slurred speech	✓	✓
Language difficulty	✓	
Vertigo		✓
Ataxia, imbalance		✓
Double vision		✓

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## Cerebral Blood Flow

- Normal: 750 ml / minute (arterial blood)
- Brain takes up 2% of total body weight and uses 20% of total body oxygen consumption
- Auto regulation
  - Capacity to maintain constant CBF despite systemic BP
    - MAP 50 – 150 has no effect on CBF due to auto regulation
    - MAP <50 or > 150 ↪ failure of auto regulation

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## Cerebral Blood Flow

- When auto regulation fails cerebral blood flow alters with changes in systemic pressure
  - Hypertension = increased ICP
  - Hypotension = hypoperfusion and ischemia

**Autoregulation is altered in an injured brain.  
There is oligemia (reduced blood flow)  
the first 24 hours after injury.**

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## Cerebral Perfusion Pressure (CPP)

- Pressure at which the brain is perfused
- ***Cerebral blood flow is impacted by CPP***
- $CPP = MAP - ICP$
- Normal: 80–100 mmHg
- Acceptable: 60-150 mmHg
- Injured brain needs a CPP > 70 mmHg

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## Conditions with Increased Risk for Stroke

- Previous TIA
- Vascular bed disease
- Migraine HA
- Sleep apnea
- Hypercoagulability
- Sickle cell disease
- Atrial fibrillation
- Dilated myopathy
- Extensive MI
- Valvular heart disease (endocarditis)
- Cardiac surgical procedures
- Congenital heart defects

Risk factors for CVA similar for those for CAD: Hypertension most important risk factor!

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## Prevention of Stroke

- Antiplatelet therapy
  - Aspirin
  - Clopidogrel
- Carotid endarterectomy
  - **Surgical removal of plaque for stroke prevention**
  - **Indications**
    - **Symptomatic disease with hemodynamically significant stenosis**
    - **Asymptomatic disease with critical stenosis**
  - **Surgical considerations and techniques**
    - **Aspirin**
    - **Clamping of carotid**
- Carotid stenting in select patients
- HRT (not indicated)

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### Carotid Endarterectomy Post Operative Care

- Assessment of neurological status
  - Vagus nerve and hypoglossal nerve function are tested after fully awake
  - Cranial nerve damage is potential complication
- Management of blood pressure
  - Avoid both hypertension and hypotension
  - Temporary auto regulation can be lost on operative side
- Assessment of wound
  - Assess for hematoma
  - Maintain adequate airway if hematoma present
  - Surgical evacuation required for hematoma

### Carotid Stenting

- Treatment option for high risk surgical patients
- Potential advantages
- Potential disadvantages
- Clopidogrel and aspirin for 48 hours pre procedure and 4 weeks post procedure
  - Aspirin indefinitely

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## Ischemic Stroke

- Sudden, severe disruption of the cerebral circulation with a subsequent loss of neurologic function caused by thrombus or embolism
  
- Ischemic strokes account for 75% of all strokes

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## Definitions

- TIA – Transient Ischemic Attack
  - Episode of neurologic impairment
  - Focal cerebral ischemia
  - Resolves within 24 hours
- RIND – Reversible Ischemic Neurologic Deficit
  - Focal cerebral ischemia
  - Lasts longer than 24 hours
  - Usually resolves 1-3 days may take 3-4 weeks

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## Causes of Ischemic Stroke

- Thrombosis
  - Night time stroke (sluggish blood flow)
- Progressing thrombotic stroke
  - Focal Neurologic impairment
  - Thrombus in artery serving the brain
  - Exhibits a stepwise worsening over minutes, hours or days after initial presentation
- Complete thrombotic stroke
  - Focal Neurologic impairment
  - Thrombus in artery serving the brain
  - Deficit persists > 3 weeks
- Embolism
  - Air or fat embolism
  - Embolic Clot

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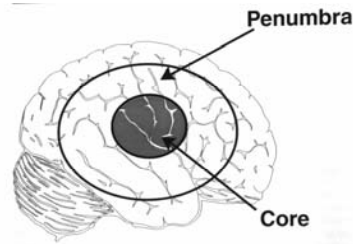
## Stroke Classifications

- Lacunar stroke
  - Special subset of stroke: small cavities called lacunes
  - Often seen in hypertensive patients with small vessel disease
  - Well localized infarcts with characteristic neurologic abnormalities
    - Pure motor or pure sensory hemiplegia and dysarthria with clumsy hand syndrome
- Cryptogenic strokes
- Watershed or border zone infarct
- Unusual causes

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## ISCHEMIC CASCADE

- Primary cell death
  - Within 4-5 minutes
  
- Secondary cell death
  - Compromised cells
  - Penumbra
  - Potential for recovery within 3 hours
  
- Inflammation and immune response
  - Impair ability to dissolve clot
  - Results in cerebral edema
    - ***Increased risk for intracerebral hemorrhage***



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## Key Signs and Symptoms

- Numbness
- Confusion
- Slurred speech / difficulty speaking
- Sudden visual problems
- Loss of balance / coordination
- Severe dizziness
- Sudden severe HA



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## Treating Ischemic Stroke

- Maintain cerebral perfusion
  - Maintain adequate MAP
  - Fibrinolytics
    - Activase (alteplase)
      - Total dose = .9 mg/kg
      - 10% of dose over 1<sup>st</sup> minutes; 90% over next 60 minutes
    - **Within 3 hours of symptom onset**
  - Platelet aggregation inhibitors
    - Within 48 hours of stroke
  - Anticoagulants
    - Hold for 1<sup>st</sup> 24 hours after fibrinolytics
    - Avoid in large completed thrombotic stroke

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## Fibrinolytic Specifics

- National Guidelines
  - Door to doctor
    - 10 minutes
  - Door to CT
    - 15 minutes
  - CT to Read
    - 45 minutes
  - Door to Needle
    - 90 minutes
- Baseline CT excludes hemorrhage or > 1/3 MCA territory
- National Institutes of Health Stroke Scale Score
  - > 4 with at least 2 from motor

*The higher the score the more severe the stroke*

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## Fibrinolytic Specifics

- **Contraindications**
  - Rapid improving deficit
  - BP > 185/ 110
  - Seizure at stroke onset
  - Recent surgery
  - Bleeding disorder / anticoagulants
  - Stroke or head trauma within 3 months
  - Recent noncompressible arterial puncture
  - Glucose < 50 or > 400

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## Additional Treatment Options

- **Intraarterial thrombolysis**
  - **Delivers directly to lesion (middle cerebral artery)**
  - **Can extend window of opportunity**
- **Mechanical thrombolysis** (lasers, retrieval devices, therapeutic ultrasound, etc)
- **Hypothermia**
  - Extends window
  - Improves penumbral survival
  - Techniques
    - Surface
    - Intravenous
    - Radiant catheter
- **Volume expansion and drug induced hypertension**
  - Complication of intracerebral edema and hemorrhage

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## Common Changes That Occur With Stroke

- Pain or Numbness
- Fatigue
- Ignoring Affected Side
- Sensory Deprivation
- Visual Field Problems
- Rapid Mood Changes
  - Reflex Crying
- Bowel / Bladder Problems
- Depression



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## Changes With Right Brain Strokes

- Left CVA
  - Right Sided Paralysis
  - Hesitancy
  - Aphasia
- Right CVA
  - Left Sided Paralysis
  - Impulsiveness
  - Spatial / Perception Deficits

Monitor and treat complications:

- ✓ ROM and physical therapy
- ✓ Monitor for postural imbalances
- ✓ Adequate nutrition / aspiration prevention
- ✓ Monitor for speech / swallowing difficulties
- ✓ Therapy, therapy, therapy

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## Types of Intracranial Bleeds

- Subdural hematoma
- Epidural hematoma
- Intracerebral hematoma
- Subarachnoid Hemorrhage
  - Usually caused by intracranial aneurysms
- Intracerebral Bleed
  - Called hemorrhagic stroke when caused by aneurysm, AV malformation, vascular tumor, or rupture of a vessel due to hypertension

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