

Final Exam concept review

Endotracheal tube

-Ensure patent

-Suction as needed

*(prior to suction) Hyperoxygenate: failure to do so can cause desaturation, leading to arrhythmias due to temporary hypoxemia.

-Monitor lip line to make sure the tube is not moved or displaced.

-Provide oral care q2 hours to prevent pneumonia.

-Aspiration precaution: keep HOB elevated

Ventilator care

-Ensure vent tubing connected to the patient, to the ventilator

-Assess breath sounds bilaterally (if tubing slips will go into right bronchial first)

-Low-pressure alarms are due to a low exhaled volume (didn't exhale enough air)

*no pressure, no resistance

*check for disconnection, cuff leak, or tube displacement

*a patient may desat or decline in condition.

-High-pressure alarms are due to an obstruction in the circuit

*blocked or kinked tubing.

*check for excess secretions/coughing

Chest tube care

-A disposable 3-chamber system most often used:

*1st chamber: drainage collection

*2nd chamber: water seal

**contains 2cm of water

**continuous bubbling in the water seal chamber indicates a leak

**tidaling is a fluctuation in the water level and occurs with respiration.

*3rd chamber: suction control (can be wet or dry)

-Diagnostic testing:

- *Chest X-ray and computed tomography scan: provide initial identification of a PE.
- *Ventilation-perfusion scan: show circulation of air and blood in the lungs and can detect PE.
- *Pulmonary angiography: gold standard to detect PE. Invasive and costly.

-Tx:

-Anticoagulants (heparin, enoxaparin, warfarin, & fondaparinux)

*used to prevent clots from getting larger or additional clots from forming

-Direct factor Xa inhibitor (rivaroxaban)

*inhibits the production of thrombin

-Thrombolytic therapy (alteplase, reteplase, and tenecteplase)

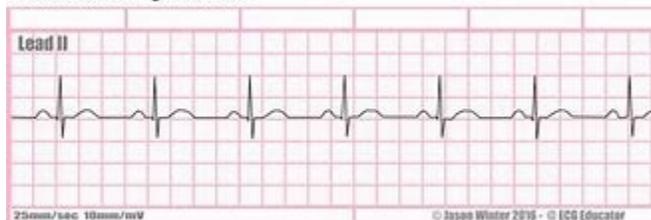
*used to dissolve blood clots and restore pulmonary blood flow

Dysrhythmias

“Sinus”= p wave present. Consistent R-R intervals. PR interval 0.12-0.2 sec. (Each small box is 0.04 sec. One larger box is 0.2 sec. So the PR interval should be between 3-5 small boxes.)

Sinus rhythm: P wave is present in front of every QRS complex. R-R intervals are equally spaced. Normal PR interval. **HR is between 60-100bpm**. For most strips, they are 6-sec strips. Count the number of R waves and multiply by 10 to get the beats per minute.

Normal Sinus Rhythm (NSR)

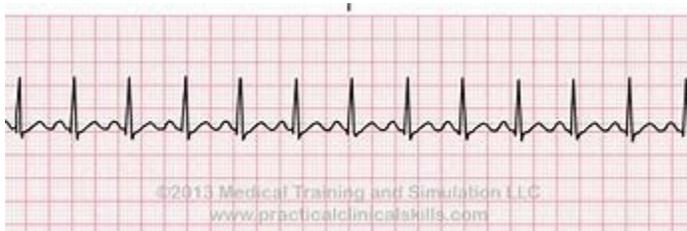


Sinus bradycardia: P wave is present in front of the QRS complex. R-R intervals are equally spaced. Normal PR interval. **HR is less than 60 bpm**.

Sinus Bradycardia

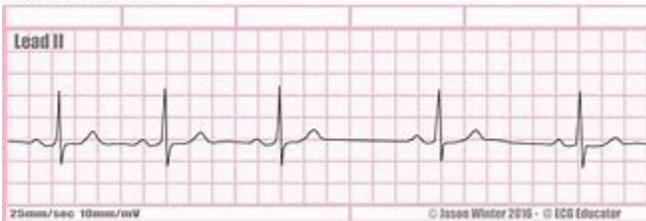


Sinus tachycardia: P wave, R-R, and PR intervals all normal. **HR is greater than 100.**



Sinus arrhythmia: P wave, PR intervals all normal. Heart rate typically 60-100. **R-R intervals vary**

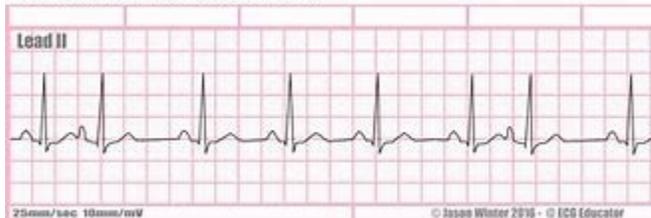
Sinus Arrhythmia



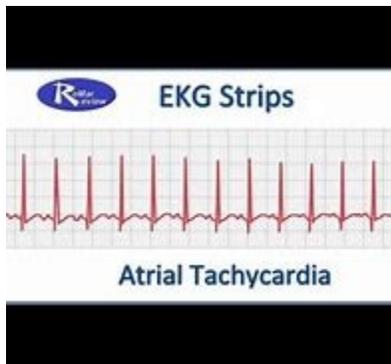
“Atrial” rhythms= originating in the atria. These rhythms affect the P wave

PACs- premature atrial contractions= happening before it should be. The one offset PQRS has a P wave that looks different. R-R interval varies with this complex but all other R-R intervals are equal

Premature Atrial Contraction (PAC)



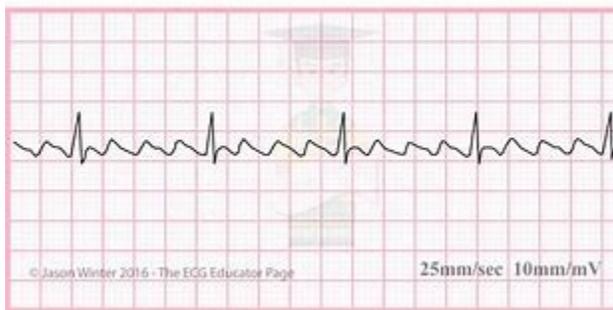
Atrial Tachycardia- Consistent R-R interval. **HR 150-250.** P wave is flattened or hidden inside the T wave because of the rapid rhythm.



Atrial Flutter- Atrial rate is rapid (greater than 100). Meaning there are multiple p waves between each QRS complex. P waves are well defined and have a “sawtooth” appearance. PR interval is hard to measure. R-R interval consistent.

“Regularly irregular”

Atrial Flutter



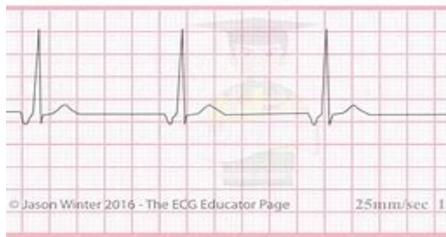
Atrial fibrillation- “Irregularly Irregular”. Atrial and ventricular activity is irregular and chaotic. Unable to predict PR interval, p waves, or R-R intervals.



“Junctional” rhythms- P wave hidden in preceding T wave, can be inverted, or come after QRS complex. R-R interval consistent.

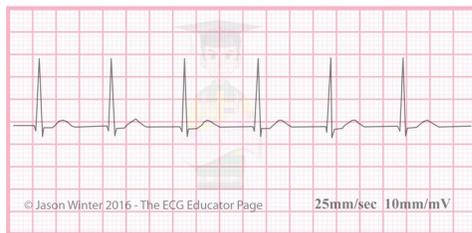
Junctional rhythm: rate 40-60bpm

Junctional Rhythm

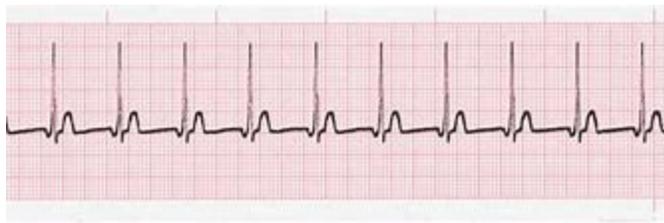


Accelerated Junctional Rhythm: rate 60-100bpm

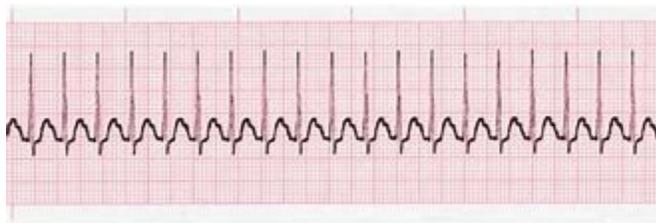
Accelerated Junctional Rhythm



Junctional tachycardia: rate 100-180bpm



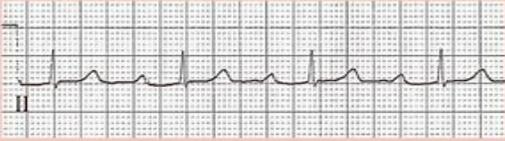
Supraventricular Tachycardia: Rate >150bpm. Too fast to see the p wave. R-R interval is regular. Hard to accurately identify the rhythm so it is given the descriptive identification of SVT.



Heart Blocks- conduction trouble at the AV node.

First degree: PR interval greater than 0.20sec and constant. P wave present. R-R interval consistent. This heart block is fairly common.

First Degree Heart Block



Second degree:

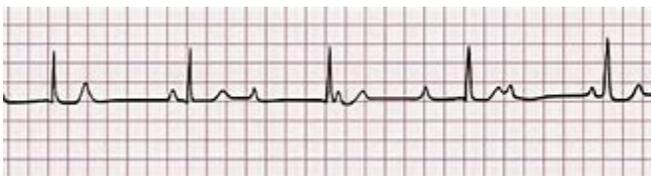
Type 1: (Wenckebach) "Going, Going, Gone". PR interval progressively gets longer until one P wave is NOT followed by the QRS complex.



Type 2: PR intervals are all the same size. Random p waves drop the QRS.

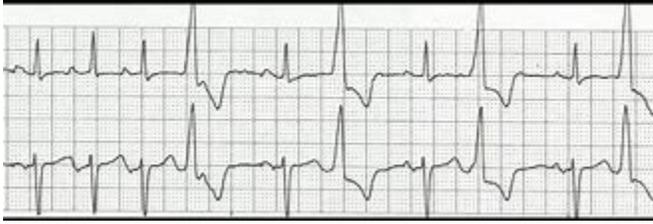


Third-degree (complete): More P waves than QRS. No PR interval. P waves have no relationship to QRS complexes.

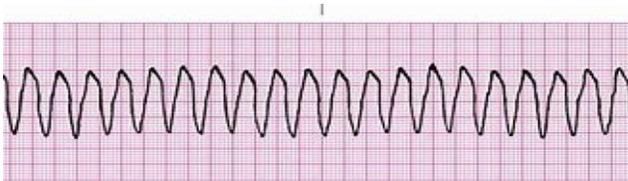


Ventricular Rhythms- more serious.

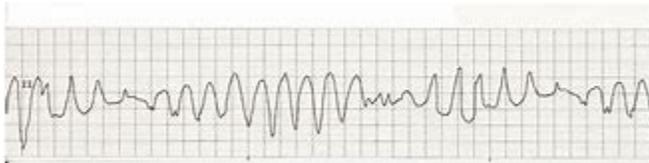
PVCs: Very common. Wide QRS out of the normal sequence. Identify if they are Unifocal or multifocal and how often they are occurring. If falling on a T wave it is called the "R on T phenomenon". 2 PVCs in a row = couplet. Bigeminy= every other beat. trigeminy= every 3rd beat.



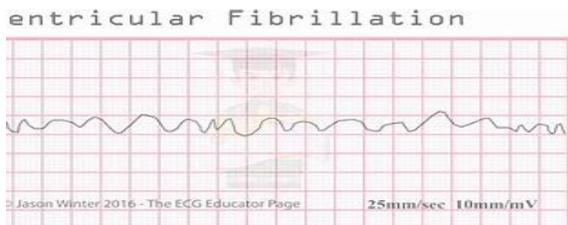
Ventricular Tachycardia: Run of PVCs. Rate of 150-250 bpm. Uniform, wide, measurable complexes.



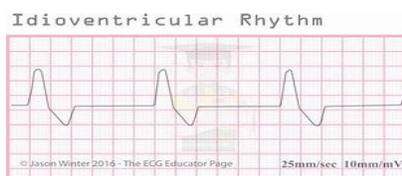
Torsade de Pointes: Polymorphic V-tach



Ventricular fibrillation: Easy to recognize. No measurable waves or complexes.



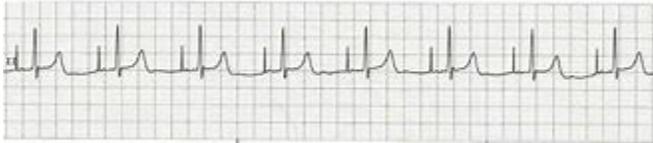
Idioventricular rhythm (Last chance): rate less than 40 bpm. No p wave. Wide and bizarre QRS



Asystole: flatline

Pulseless Electrical Activity: activity seen on EKG no pulse is produced

Pacemaker: detectable by "capture spike". Can be before the p wave, before QRS, or both.



MI

-Occurs because of the abrupt stoppage of blood flow through a coronary artery from a thrombus caused by platelet aggregation.

-Risk factors:

- *male gender or postmenopausal women
- *ethnic background
- *sedentary lifestyle
- *hypertension
- *tobacco use
- *hyperlipidemia
- *obesity
- *excessive alcohol consumption
- *metabolic disorders (diabetes mellitus: glucose can cause atherosclerosis, hyperthyroidism)
- *stress
- *methamphetamine or cocaine use

-S/sx:

- *anxiety, feeling of impending doom
- *nausea, dizziness
- *chest pain (can radiate and is described as crushing or aching pressure)
- *pallor, and cool, clammy skin
- *tachycardia and heart palpitations (ST elevation)
- *tachypnea and shortness of breath
- *diaphoresis

-Labs & Diagnostic

- *Myoglobin: the earliest marker of injury to cardiac or skeletal muscle (levels no longer evident after 24 hours)
- *Creatine kinase-MB (CKMB): Peaks around 24 hours after onset of chest pain (levels no longer evident after 3 days).
- *Troponin I or T: any positive value indicates damage to cardiac tissue

>Troponin I: levels no longer evident after 7 to 10 days.

>Troponin T: levels no longer evident after 10 to 14 days.

*ECG: recording of the electrical activity of the heart over time.

*Cardiac catheterization: an invasive diagnostic procedure used to evaluate the presence and degree of coronary artery blockage.

**can have a stent or balloon placement, or CABG

-Tx:

*MONA: morphine, oxygen, nitroglycerin, aspirin

*Medications

**Vasodilators (nitroglycerin): prevents coronary artery vasospasm and reduces preload and afterload, decreasing myocardial oxygen demand.

**Analgesics (morphine): used to treat moderate to severe pain.

**Beta-blockers (metoprolol): decrease infarct size and improve short- and long-term survival rates.

**Thrombolytic agents (alteplase & reteplase): used to break up blood clots.

**Antiplatelet agents (aspirin & clopidogrel): prevent platelets from forming together.

**Anticoagulants (heparin & enoxaparin): used to prevent clots from becoming larger or other clots from forming.

CAD

-Nonmodifiable risk factors include age, race/ethnicity, gender, family history.

-Reducing modifiable risk factors (cholesterol abnormalities, tobacco use, hypertension, and diabetes, obesity, sedentary lifestyle)

-A diet low in saturated fat and high in soluble fiber

-Regular, moderate physical activity

-Using medications to help control cholesterol, hypertension, diabetes.

-controlling psychosocial risk factors

Transfusion

-Consent form required prior to any type of blood product given.

-Verification requires 2 RNs.

*verify patient, ID number, ABO group, RH compatibility, and expiration date on blood bag against patient's armband and blood bank bracelet.

-Must administer within 30 minutes of receiving from the blood bank.

*Packed red blood cells (PRBCs) within 4 hours

*Fresh frozen plasma (FFP) over 30-60 minutes

*Platelets within 15-30 minutes (as fast as the patient can tolerate)

-Use Y blood tubing with filter and prime with 0.9% NS only.

-Vitals with temp and assessment prior to starting transfusion.

*monitor vitals q15 minutes (for the first hour) during infusion.

-Reactions occur during the first 15-30 minutes:

*acute hemolytic (due to incompatible blood)

s/sx: low-back pain, chills, fever, anxiety, impending sense of doom

tx: stop the infusion; initiate infusion of 0.9% NS using new tubing, administer Benadryl.

*febrile (recipient antibodies reacting with white cell antigens or fragments)

s/sx: chills, temperature increase, flushing, tachycardia

tx: stop infusion; administer antipyretic

*bacterial (contaminated blood)

s/sx: wheezing, dyspnea, chest tightness, cyanosis, hypertension, shock

tx: stop infusion; administer antibiotic

ARF

-a condition in which the kidneys suddenly can't filter waste from the blood

-S/sx:

*decreased urine output

*swelling due to fluid retention

*nausea *fatigue *shortness of breath

-Tx: involves treating the underlying cause.

-medication:

*diuretic (bumetanide, furosemide) to treat fluid retention

-dialysis:

Disseminated intravascular coagulation (DIC)

-a life-threatening coagulopathy in which clotting and anticlotting mechanisms occur at the same time.

*at risk for both internal and external bleeding, as well as damage to organs resulting from ischemia caused by micro clots.

-Secondary to other complications

*septicemia *cardiopulmonary arrest *cancer

*trauma (hemorrhage, burns, crush injuries)

*obstetric complications (toxemia, amniotic fluid embolus, placental abruption)

-Expected findings:

*unusual spontaneous bleeding from the gums and nose (epistaxis)

*oozing, trickling, or flow of blood from incisions or lacerations

*petechiae or ecchymoses *hematuria

*excessive bleeding from venipuncture, injection sites, or slight traumas

*tachycardia, hypotension, and diaphoresis *respiratory distress

*organ failure secondary to microemboli

-Dx:

*decreased hemoglobin *decreased platelet levels (thrombocytopenia)

*decreased fibrinogen levels *increased prothrombin time

*increased partial thromboplastin *increased thrombin time

*increased D-dimer

-Nursing care:

*initially focus on assessing for and correcting the underlying causes

*focus then turns to preventing organ damage secondary to microemboli and replacing the blood's clotting components

*avoid use of NSAIDs

*implement bleeding precautions (avoid use of needles)

*Medications:

anticoagulants (heparin) used to decrease micro clots from forming and using up clotting factors.

Fluid volume: Hypervolemia, hypovolemia

<p>Hypovolemia- occurs when the loss of ECF volume exceeds the intake of fluid. Occurs when water and electrolytes are lost in the same proportion as they exist in normal body fluids, thus the ratio of serum electrolytes to water remains the same. FVD should not be confused with dehydration, which refers to the loss of water alone, with increased serum sodium levels.</p> <p><u>Causes:</u> abnormal fluid losses (V/D, GI suctioning, sweating), decreased intake (nausea or lack of access to fluids), 3rd space fluid shifts (edema formation in burns, ascites with liver dysfunction)</p>	<p>Hypervolemia- refers to the isotonic expansion of ECF caused by abnormal retention of water and sodium in the same proportions in which they normally exist in the ECF. R/T to simple fluid overload or diminished function of the homeostatic mechanisms responsible for regulating fluid balance.</p> <p>Contributing factors can include heart failure, renal failure, and cirrhosis of the liver, excessive amounts of the table or other sodium salts.</p> <p>Excessive administration of sodium-containing fluids in a patient impaired regulatory mechanisms may predispose as well.</p>
<p>S/S: acute weight loss decreased skin turgor oliguria concentrated urine capillary filling time prolonged Low CVP decreased BP and increased pulse Flattened neck veins dizziness weakness thirst confusion muscle cramps sunken eyes nausea increased temperature cool, clammy, pale skin</p>	<p>S/S: acute weight gain peripheral edema and ascites JVD crackles elevated CVP shortness of breath increased BP bounding pulse increased RR increased urine output cough</p>
<p>Labs: elevated hemoglobin, hematocrit, serum urine osmolality, and specific gravity, BUN, creatinine. decreased urine sodium.</p>	<p>Labs: decreased hemoglobin and hematocrit, serum and urine osmolality, urine sodium, and specific gravity</p>
<p>Nursing management:</p>	<p>Nursing management:</p>

<ul style="list-style-type: none"> -Isotonic electrolyte solutions (lactated ringers, 0.9% sodium chloride) 1ST CHOICE -Monitor I&O (every 8 hours), daily weights vital signs, CVP, LOC, breath sounds, skin color, skin, and tongue turgor. -Monitor for fluid overload -If oliguric: consider prerenal azotemia vs VTN -Volumes of fluid are administered at specific rates and intervals while monitoring their body's response. -Prevent hypovolemia -Correct hypovolemia- offer small volumes of oral rehydration solutions 	<ul style="list-style-type: none"> -Diuretics: generally thiazide diuretics are prescribed for mild to moderate hypervolemia and loop diuretics for severe hypervolemia. -Dialysis: when pharmacological agents aren't effective -Nutritional therapy: a sodium-restricted diet -Measure I&O: gain of 1 kg -Daily weights -Assess breath sounds -Monitor degree of edema in dependent parts of the body
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c. Isotonic solutions → The concentration of solutes is the same both inside and outside the cell

0.9% normal saline & lactated ringers

ABG's

-reports the status of oxygenation and acid-base balance of the blood.

pH (acid) 7.35 - 7.45 (alkalosis)

PaCO₂ (alkalosis) 35 - 45 (acidosis)

HCO₃ (acidosis) 22 - 26 (alkalosis)

PaO₂ 80 - 100

-Respiratory acidosis

(initial) decreased pH, elevated or normal HCO₃, elevated PaCO₂

(compensation) increased renal acid excretion (increased PaCO₂) and increased serum HCO₃ (>26mEq/L)

-Respiratory alkalosis

(initial) increased pH, decreased or normal HCO₃, decreased PaCO₂

(compensation) decreased renal acid excretion (decreased PaCO₂) and decreased serum HCO₃ (<21 mEq/L)

-Metabolic acidosis

(initial) decreased pH, decreased HCO₃, decreased or normal PaCO₂

(compensation) hyperventilation with resulting decreased PaCO₂ (>45 mm Hg), decreased HCO₃

-Metabolic alkalosis

(initial) increased pH, increased HCO₃, increased or normal PaCO₂

(compensation) hypoventilation with resulting increased PaCO₂ (<35 mm Hg), increased HCO₃

Electrolyte imbalance

Sodium

Electrolyte	Functions	Sources & Losses	Regulation
<p>Sodium (Na⁺)</p> <ul style="list-style-type: none">• Chief electrolyte of ECF• Normal serum sodium: 135-145 mEq/L	<ul style="list-style-type: none">• Regulates extracellular fluid volume; Na⁺ loss or gain accompanied by a loss or gain of water• Affects serum osmolality• Role in muscle contraction and transmission of nerve impulses• Regulation of acid-base balance as sodium bicarbonate	<ul style="list-style-type: none">• Normally enters the body through the GI tract from dietary sources, such as salt added to processed foods, sodium preservatives added to processed foods• Lost from the gastrointestinal tract, kidneys, and skin	<ul style="list-style-type: none">• Transported out of the cell by the sodium-potassium pump• Regulated by the renin-angiotensin-aldosterone system• Elimination and reabsorption regulated by the kidneys• Sodium concentrations affected by salt and water intake

Hyponatremia

- Serum sodium <135 mEq/L
- Causes: loss of sodium-containing fluids (draining wounds, V/D, primary adrenal insufficiency), water excess in relation to the amount of sodium (dilutional hyponatremia such as renal failure, SIADH, psychiatric disorders), or combination of both
- Manifestations: poor skin turgor, dry mucosa, headache, decreased salivation, decreased blood pressure, nausea, abdominal cramping, neurologic changes, CONFUSION
- Medical management: water restriction, sodium replacement
- Level should not increase more than 8-12 mEq/L in the 1st 24 hrs
- Nursing management: assessment and prevention, dietary sodium and fluid intake identify and monitor at-risk patients, effects of medications (diuretics, lithium)
 - Hypertonic saline
 - Used in emergency settings when the patient requires 4-6 mEq/L increase in serum sodium as soon as possible
 - Example à 3% saline
 - Administer slowly because it may cause volume overload and pulmonary edema

Hypernatremia

- • Serum sodium >145 mEq/L
- • Causes: inadequate water intake, excess water loss, or rarely, sodium gain
- • Manifestations: thirst; elevated temperature; dry, swollen tongue; sticky mucosa; neurologic symptoms; restlessness; weakness
- • Note: thirst may be impaired in the elderly or the ill
- • Medical management: Depends on the underlying cause
- -If water deficit à fluid replacement (oral or IV)
- -If sodium excess à sodium-free IV fluids (D5W), diuretics, and restrict dietary sodium
- -Serum sodium should not decrease by more than 8-15 mEq/L in an 8 hr period à too fast and the result is cerebral edema & neurologic complications (risk is greatest if hypernatremia developed over several days or longer)
- • Nursing management: assessment and prevention, assess for OTC sources of sodium, offer and encourage fluids to meet patient needs, provide sufficient water with tube feedings
-

5. Hypokalemia/Hyperkalemia

Hypokalemia

- Below-normal serum potassium (<3.5 mEq/L), may occur with normal potassium levels with alkalosis due to shift of serum potassium into cells
- Causes: GI losses, medications, alterations of acid-base balance, hyperaldosteronism, poor dietary intake
- Manifestations: fatigue, anorexia, nausea, vomiting, dysrhythmias, muscle weakness and cramps, paresthesias, glucose intolerance, decreased muscle strength, DTRs
- Medical management: increased dietary potassium, potassium replacement, IV for severe deficit
- Nursing management: assessment, severe hypokalemia is life-threatening, monitor ECG and ABGs, dietary potassium, nursing care related to IV potassium administration

- **Potassium deficit**

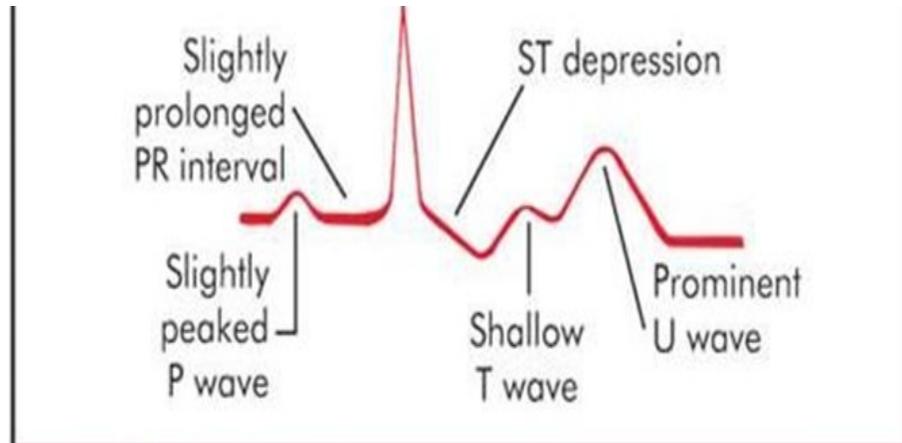
- ● Potassium deficit varies directly with the severity of hypokalemia
- • A potassium deficit of 200-400 mEq is required to lower the serum potassium concentration by 1 mEq/L
- • However, these estimates are only an approximation of the amount of potassium replacement required to normalize the serum potassium concentration, and careful monitoring is required

- **Potassium Replacement**

- • Potassium chloride
- – Preferred
- – Raises serum potassium concentration at a faster rate
- – Oral can be given in the crystalline form (salt substitutes), as a liquid, or in a slow-release tablet or capsule
- – IV route → pain and phlebitis during infusion, primarily occurs at rate >10mEq/hr
- • Must use an infusion pump
- • Increase intake of potassium-rich foods
- – Highest → dried figs, molasses, seaweed
- – Very High → dried fruits, nuts, avocados, wheat germ, lima beans
- – High → spinach tomatoes, broccoli, beets, carrots, cauliflower, potatoes, bananas, cantaloupes, kiwis, oranges, mangos, ground beef, steak, pork, veal, lamb
- • If continued potassium losses → rate of potassium administration must be increased by the rate of potassium loss to produce the desired rate of potassium repletion

- **IV Potassium Safety Alert**

- ● IV KCl must always be diluted and never given in concentrated amounts
- • Never give KCl via IV push or as a bolus
- • Invert IV bags containing KCl several times to ensure even distribution in the bag
- • Do not add KCl to hanging IV bag to prevent giving a bolus dose
-
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- Patients with hypokalemia may also have hypomagnesemia d/t concurrent loss w/ diarrhea or diuretic therapy
- • Such patients can be refractory to potassium replacement alone; thus measurement of serum magnesium should be considered



Potassium

Electrolyte

Potassium (K⁺)
 •Major cation of ICF
 •Normal serum potassium: 3.5-5.0

Functions

- Controls intracellular osmolality
- Regulator of cellular enzyme activity
- Role in the transmission of electrical impulses in nerve, heart, skeletal, intestinal, and lung tissue; protein and carbohydrate metabolism; and cellular building
- Regulation of acid-base balance by cellular exchange with H⁺

Sources & Losses

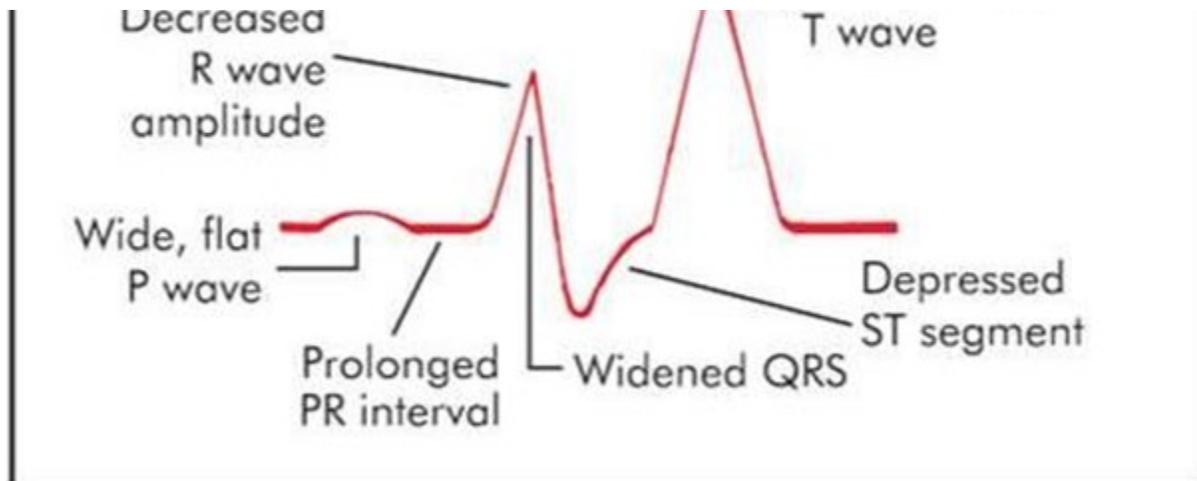
- Adequate quantities via a well-balanced diet
- Leading food sources: fruits and vegetables, dried peas and beans, whole grains, milk, meats
- Lost via kidneys, stool, sweat, emesis
- Gastrointestinal (GI) secretions contain potassium in large quantities, so can be lost through vomitus

Regulation

- Regulated by aldosterone
- Eliminated by the kidneys (no effective method of conserving potassium)
- Additional regulation via transcellular shift between the ICF and ECF compartments

Hyperkalemia

- Serum potassium >5.0 mEq/L
- **Causes:** usually treatment-related, impaired renal function, hypoaldosteronism, tissue trauma, acidosis
- **Manifestations:** cardiac changes and dysrhythmias, muscle weakness with potential respiratory impairment, paresthesias, anxiety, GI manifestations
- **Medical management:** monitor ECG, limitation of dietary potassium, cation-exchange resin (Kayexalate), IV sodium bicarbonate, IV calcium gluconate, regular insulin, and hypertonic dextrose IV, b-2 agonists, dialysis
- **Nursing management:** assessment of serum potassium levels, mix IVs containing K⁺ well, monitor medication effects, dietary potassium restriction/dietary teaching for patients at risk
- Hemolysis of blood specimen or drawing of blood above IV site may result in the false laboratory result
- Salt substitutes, medications may contain potassium
- Potassium-sparing diuretics may cause elevation of potassium
- Should not be used in patients with renal dysfunction
- **Treatment of Hyperkalemia**
 - **Antagonism of membrane action potentials**
 - Calcium
 - Calcium gluconate vs calcium carbonate → calcium chloride contains 3x the concentration of elemental calcium however calcium gluconate is generally preferred d/t calcium chloride causing local irritation at the injection site
 - Drive extracellular potassium into cells
 - Insulin & glucose
 - Sodium bicarbonate (primarily if metabolic acidosis)
 - Beta 2 adrenergic agonists (Albuterol)
 - Removal of potassium from the body
 - Loop or thiazide diuretics (if no severe renal impairment)
 - Gastrointestinal cation exchange (ie - patiromer, Kayexalate)
 - Dialysis, preferably hemodialysis if severe
 - Digoxin like drugs and B-adrenergic blockers (e.g., propranolol) can impair the entry of potassium into cells, resulting in a higher ECF potassium concentration
 - • Several drugs, such as heparin, potassium-sparing diuretics, angiotensin II receptor blockers (e.g., losartan), and angiotensin-converting enzyme (ACE) inhibitors (e.g., lisinopril) can contribute to hyperkalemia by reducing the kidney's ability to excrete potassium
 - Because patients on digoxin therapy have an increased risk of toxicity if their serum potassium level is low, monitor the patient for digitalis toxicity
 - • Digoxin toxicity occurs as plasma concentration rises above 2.0, however, signs of toxicity may occur at levels below 1.3-1.5 in the presence of hypokalemia
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6. Hypocalcemia/Hypercalcemia

- Hypocalcemia
 - Contributing factors: hypoparathyroidism, malabsorption, pancreatitis, alkalosis, massive transfusion of citrated blood, renal failure, medications, other
 - s/s: tetany, circumoral numbness, paresthesias, hyperactive DTRs, Trousseau's sign, Chvostek's sign, seizures, respiratory symptoms of dyspnea and laryngospasm, abnormal clotting, anxiety
 - Nursing management: assessment, severe hypocalcemia is life-threatening, weight-bearing exercises to decrease bone calcium loss, patient teaching related to diet and medications, and nursing care related to IV calcium administration
- Hypercalcemia
 - Contributing Factors: malignancy and hyperparathyroidism, bone loss related to immobility
 - s/s: muscle weakness, incoordination, anorexia, constipation, nausea and vomiting, abdominal and bone pain, polyuria, thirst, ECG changes, dysrhythmias
 - Nursing Management: assessment, hypercalcemic crisis has high mortality, encourage ambulation, fluids of 3 to 4 L/d, provide fluids containing sodium unless contraindicated, fiber for constipation, ensure safety

7. Hypomagnesemia/Hpermagnesemia

- Hypomagnesemia
 - Contributing Factors: alcoholism, GI losses, enteral or parenteral feeding deficient in magnesium, medications, rapid administration of citrated blood; contributing causes include diabetic ketoacidosis, sepsis, burns, hypothermia
 - s/s: neuromuscular irritability, muscle weakness, tremors, athetoid movements, ECG changes and dysrhythmias, alterations in mood and level of consciousness

- Nursing Management: assessment, ensure safety, patient teaching related to diet, medications, alcohol use, and nursing care related to IV magnesium sulfate
- Hypermagnesemia
 - Contributing Factors: renal failure, diabetic ketoacidosis, excessive administration of magnesium
 - s/s: flushing, lowered BP, nausea, vomiting, hypoactive reflexes, drowsiness, muscle weakness, depressed respirations, ECG changes, dysrhythmias
 - Nursing Management: assessment, do not administer medications containing magnesium, patient teaching regarding magnesium-containing OTC medications

8. Hypophosphatemia/Hyperphosphatemia

Electrolyte	Functions	Sources & Losses	Regulation
Phosphate (PO ₄ ⁻) <ul style="list-style-type: none"> • Major ICF anion; a buffer anion in both ICF and ECF • Normal serum phosphate level: 2.5–4.5 mg/dL 	<ul style="list-style-type: none"> • Role in acid-base balance as a hydrogen buffer • Promotes energy storage; carbohydrate, protein, and fat metabolism • Bone and teeth formation • Regulation of hormone and coenzyme activity • Role in muscle and red blood cell function 	<ul style="list-style-type: none"> • Enters body via the gastrointestinal tract • Sources include all animal products (meat, poultry, eggs, milk, bread, ready-to-eat cereal) • Absorption is diminished by concurrent ingestion of calcium, magnesium, and aluminum 	<ul style="list-style-type: none"> • Eliminated by kidneys • Regulation by parathyroid hormone and by activated vitamin D • Phosphate and calcium are inversely proportional; an increase in one results in a decrease in the other

Hypophosphatemia

- •Serum level below 2.5 mg/DL
- •Causes: alcoholism, refeeding of patients after starvation, pain, heatstroke, respiratory alkalosis, hyperventilation, diabetic ketoacidosis, hepatic encephalopathy, major burns, hyperparathyroidism, low magnesium, low potassium, diarrhea, vitamin D deficiency, use of diuretic and antacids
- •Manifestations: neurologic symptoms, confusion, muscle weakness, tissue hypoxia, muscle and bone pain, increased susceptibility to infection
- •Medical management: oral or IV phosphorus replacement
- •Nursing management: assessment, encourage foods high in phosphorus, gradually introduce calories for malnourished patients receiving parenteral nutrition

Hyperphosphatemia

- Serum level above 4.5 mg/DL
- **Causes:** renal failure, excess phosphorus, excess vitamin D, acidosis, hypoparathyroidism, chemotherapy
- **Manifestations:** few symptoms; soft-tissue calcifications, symptoms occur due to associated hypocalcemia
- **Medical management:** treat underlying disorder, vitamin-D preparations, calcium-binding antacids, phosphate-binding gels or antacids, loop diuretics, NS IV, dialysis
- **Nursing management:** assessment, avoid high-phosphorus foods; patient teaching related to diet, phosphate-containing substances, signs of hypocalcemia

Shock

-Classifications of shock

-Hypovolemic: shock state resulting from decreased intravascular volume due to fluid loss

-Cardiogenic: shock state resulting from failure of the heart to pump effectively due to a cardiac factor.

-Distributive: shock state resulting from widespread vasodilation and increased capillary permeability.

*Septic: circulatory shock state resulting from overwhelming infection causing relative hypovolemia.

*Neurogenic: shock state resulting from loss of sympathetic tone causing relative hypovolemia

*Anaphylactic: circulatory shock state resulting from severe allergic reaction producing overwhelming systemic vasodilation, relative hypovolemia.

-Obstructive: shock state resulting from physical obstruction impeding the filling or outflow of blood resulting in reduced cardiac output.

-Vital signs are key indicators of hemodynamic status

-Clinical findings in Stages of Shock

-Blood pressure

(compensatory) normal (progressive) systolic <90 mmHg; MAP <65 mmHg; requires fluids resuscitation to support blood pressure (irreversible) requires mechanical or pharmacologic support

-Heart Rate (compensatory) >100 (progressive) >150 (irreversible) erratic or asystole

-Respiratory status (compensatory) >20 breaths/min PaCO₂, <32 mmHg (progressive) rapid, shallow respirations; crackles PaCO₂ <80 mmHg PaCO₂ >45 mmHg (irreversible) requires intubation and mechanical ventilation and oxygenation.

-Skin (compensatory) cold, clammy (progressive) mottled, petechiae (irreversible) jaundice

-Urinary tract (compensatory) decreased (progressive) <0.5 mL/kg/h (irreversible) anuric, requires dialysis

-Mentation (compensation) confusion &/or agitation (progressive) lethargy (irreversible) unconscious

-Acid-base balance (compensatory) respiratory alkalosis (progressive) metabolic acidosis (irreversible) profound acidosis

-Fluid replacement in Shock

-Crystalloids (0.9% sodium chloride)

(advantage) widely available, inexpensive (disadvantage) requires large volume of infusion; can cause hypernatremia, pulmonary edema, abdominal compartment syndrome

-Lactated Ringer's

(advantage) lactated ion that helps buffer metabolic acidosis (disadvantage) requires large volume of infusion' can cause metabolic acidosis, pulmonary edema, abdominal compartment syndrome

-Colloids (Albumin 5%, 25%)

(advantage) rapidly expands plasma volume of crystalloid (or colloid equivalent) (disadvantage) expensive; requires human donors; limited supply; can cause heart failure

Cast care

1. Immobilization Devices (Indications, Nursing management)

-Indications:

- to prevent further injury
- reduce pain

- promote healing/circulation
- correct a deformity

-Nursing Management:

- Neurovascular assessment

*essential throughout immobilization

*assessments are performed every 1 hour for the 1st 24 hrs and 1-4 hrs thereafter

*Includes:

-Pain: assess pain level, location, and frequency; assess pain using a 0-10 pain scale, and have the client describe the pain; immobilization, ice, and elevation of the extremity with the use of analgesics should relieve most of the pain.

-Sensation: assess for numbness or tingling of the extremity; loss of sensation can indicate nerve damage.

-Skin temperature: check the temperature of the affected extremity; extremity should be warm, not cool, to touch; cool skin can indicate decreased arterial perfusion.

-Capillary refill: press nail beds of affected extremity until blanching occurs; blood return should be within 3 seconds; prolonged refill indicates decreased arterial perfusion; nail beds that are cyanotic can indicate venous congestion.

-Pulses: should be palpable and strong; pulses should be equal to the unaffected extremity; edema can make it difficult to palpate pulses, so Doppler ultrasonography might be required.

-Movement: client should be able to move affected extremity in active motion.

Cast

-Nursing Management:

-monitor neurovascular status every 1 hr for the first 24 hrs and assess pain

-apply ice for 24-48 hrs

-handle a plaster cast with the palms, not fingertips, until the cast is dry to prevent denting the cast

-avoid setting the cast on hard surfaces or sharp edges

-prior to casting, the area is cleaned and dried, tubular cotton web roll is placed over the affected area to maintain skin integrity; the casting material is then applied

-after cast application, position the client so that warm, dry air circulates around and under the cast (support the casted area without pressure under or directly on the cast) for faster drying and to prevent pressure from changing the shape of the cast. Use gloves to touch the cast until it is completely dry.

-elevate the cast above the level of the heart during the first 24-48 hrs to prevent edema of the affected extremity; use a cloth-covered pillow instead of plastic while the cast is drying

-ensure the cast is not too tight; there should be room for one finger between the skin and cast

-document the presence of drainage and report sudden increase in drainage; circling drainage on the cast is an unreliable indicator of drainage amount and can increase client anxiety

-older adult clients have an increased risk for impaired skin integrity d/t loss of elasticity of the skin and decreased sensation (comorbidities).

Fractures

1. **Fractures** (indications, nursing management, complications)

-**Indications:** (Risk Factors)

-Osteoporosis

*excessive exercising and weight loss from eating and malnutrition can lead to osteoporosis.

*women who do not use estrogen replacement therapy after menopause lose estrogen and are unable to form strong new bone.

*clients on long-term corticosteroid therapy lose calcium from their bones d/t direct inhibition of osteoblast function, inhibition of GI calcium absorption, and enhancement of bone resorption (prednisone weakens bones)

-Falls

-Motor vehicle crashes

-substance abuse disorder

-disease (bone cancer, Paget's disease)

-contact sports and hazardous recreational activities (football, skiing)

-Physical abuse

-Lactose intolerance

-Age, as bone becomes less dense w/ advancing age

-**Manifestations:**

-crepitus: a grating sound created by the rubbing of bone fragments

-deformity: internal rotation of extremity, shortened extremity, visible bone with open fracture

-muscle spasms: d/t pulling forces of the bone when not aligned

-edema: swelling from trauma

-ecchymosis (bruise): bleeding into underlying soft tissues from trauma

-**Nursing Management:**

-provide emergency care at the time of injury

-maintain ABCs.

-monitor VS and neurologic status because injury to vital organs can occur d/t bone fragments (fractures of pelvis or ribs)

-**Stabilize the injured area**, including the joints above and below the fracture, by using a splint and **avoiding unnecessary movement**.

-maintain proper alignment of the affected extremity

-assess for bleeding and apply pressure, if needed

-cover open wounds with a sterile dressing

-remove clothing and jewelry near the injury or on the affected extremity

-keep the client warm

-assess pain frequently and follow pain management protocols, both pharmacological and non-pharmacological

- initiate and continue neurovascular checks at least every hour; immediately report any change in status to the provider
- prepare the client for any immobilization procedure appropriate for the fracture.

-Complications:

- Compartment Syndrome
- Venous thromboembolism (DVT, PE)
- Avascular necrosis
- Fat embolism
- Osteomyelitis
- Failure of fracture to heal

Closed (simple) fracture

- does not break through the skin surface

Open (compound) fracture

- disrupts the skin integrity, causing an open wound and tissue injury with a risk of infection

Displaced fracture

- has bone fragments that are not in alignment

Non-displaced fracture

- has bone fragments that remain in alignment

Pathological (spontaneous) fracture

- occurs to bone that is weak from a disease process, such as bone cancer or osteoporosis

Compression fracture

- occurs from a loading force pressing on callus bone; this condition is common in older adult clients who have osteoporosis

Amputation: Above-knee and below-knee

Above Knee (AKA)

Below Knee(BKA)

- removal of a body part
- can be elective d/t complications of PVD, atherosclerosis, osteomyelitis, or malignant tumor

- cardiopulmonary complications r/t co-morbid conditions
- DVT
- infection
- phantom limb pain
- Stump hematoma
- need for re-amputation (revision)
- flexion contraction

Osteomyelitis

Osteomyelitis

-Infection of the bone that begins as an inflammation within the bone secondary to penetration by infectious organisms (virus, bacteria, or fungi) following trauma or surgical repair of a fracture.

-Manifestations:

- bone pain that is constant, pulsating, localized, and worse w/ movement
- erythema and edema at the site of infection
- fever: older adults might not have an elevated temperature
- leukocytosis and possible elevated sedimentation rate (ESR)
- many of these manifestations will disappear if the infection becomes chronic

-Nursing management:

- administer antibiotics as prescribed to maintain a constant blood level
- administer analgesics as needed
- conduct neurovascular assessment if debridement is done
- if the wound is left open to heal, standard precautions are adequate, and clean technique can be used during dressing changes.

Acute kidney injury

-the sudden cessation of renal function that occurs when blood flow to the kidneys is significantly compromised.

-Risk factors:

- *trauma
- *renal vascular obstruction
- *shock
- *decreased cardiac output causing decreased renal perfusion
- *sepsis
- *hypovolemia
- *peripheral vascular resistance
- *liver failure
- *acute nephrotoxins
- *infection, vasculitis, acute glomerulonephritis
- *stone, tumor, bladder atony
- *spinal cord disease or injury

-S/sx:

- *waste buildup *decreased urine output
- *fluid overload (dependent and generalized edema) *dysrhythmia (hyperkalemia)
- *crackles, decreased oxygenation, shortness of breath
- *scant to normal or excessive urine output *lethargy, muscle twitching, seizures

-Dx:

- *elevated creatinine and BUN *decreased hematocrit
- *Urine specific gravity varies (elevated in prerenal, diluted in intrarenal)
- *Urinalysis: presence of sediment (RBC, casts)
- *X-ray of the pelvis, or kidneys, urethra, and bladder (KUB) to detect calculi and hydronephrosis
- *Ultrasound to detect an obstruction in the urinary tract
- *Kidney biopsy to detect immunological disease or determine kidney function

-Tx:

- *Medications: diuretics (furosemide, mannitol, ethacrynic acid) to promote increased infiltration of blood by the kidney.
- *for AKI caused by medication nephrotoxicity, administer calcium channel blocker to prevent the movement of calcium into the kidney cells.
- *sodium polystyrene sulfonate replaces sodium with potassium in the intestinal tract to promote potassium excretion.

Hemodialysis

-shunts blood from the body through a dialyzer and back into circulation; requires vascular access.

-Indications:

- *renal insufficiency *acute renal injury *chronic kidney disease
- *drug overdose *persistent hyperkalemia
- *hypervolemia that does not respond to diuretics

-Manifestations:

- *fluid volume changes, electrolyte and pH imbalances, and nitrogenous wastes.
- *hemodialysis is based on symptoms, not the glomerular filtration rate

*Manifestations include fluid overload, neurological changes, bleeding, and uremia (cognitive impairment, pruritus, vomiting)

Peritoneal dialysis

-involves instillation of hypertonic dialysate solution into the peritoneal cavity and subsequent dwell times. Drain the dialysate solution that includes the waste products; peritoneum serves as the filtration membrane.

*client should have intact peritoneal membrane, without adhesions from infection or multiple surgeries.

-Indications: Peritoneal dialysis treats clients requiring dialysis who:

*are unable to tolerate anticoagulants

*have difficulty with vascular access

*have chronic infections or are unstable

*have chronic diseases, such as diabetes mellitus, heart failure, or severe hypertension.

Pancreatitis

-An autodigestion of the pancreas by pancreatic digestive enzymes that activate prematurely before reaching the intestines.

-Risk factors:

*biliary tract disease

*alcohol use

*increased age

*gastrointestinal surgery

*metabolic disturbances

*trauma

*kidney failure or transplant

*genetic predisposition

*penetrating ulcer

*medication toxicity

*viral infections

*cigarette smoking

-Expected findings:

*sudden onset of severe, boring pain

*nausea & vomiting

*weight loss

*generalized jaundice

*ascites

*decreased or absent bowel sounds

*seepage of blood-stained exudates into tissue

*warm, moist fruity breath

*tetany due to hypocalcemia

-Dx:

*serum amylase & lipase increase

*WBC increased due to infection

*Platelets decreased

*serum calcium & magnesium increased

*serum liver enzymes and bilirubin increased *serum glucose increased

*erythrocyte sedimentation rate elevated.

-Tx:

-Opioid analgesics (morphine) to relieve acute pain.

-antibiotics (Imipenem) for the infection

-histamine receptor antagonists (ranitidine) decreases gastric acid secretion

-proton pump inhibitors (omeprazole) to decrease gastric acid secretion.

-pancreatic enzymes (pancrelipase) aid with digestion of fats and proteins when taken with meals and snacks.

Chronic kidney disease

-a progressive, irreversible kidney disease

-Risk factors:

*acute kidney injury

*diabetes mellitus

*chronic glomerulonephritis

*nephrotoxic medications (gentamicin, NSAIDs) or chemicals

*hypertension, especially in African American clients

*Autoimmune disorders (systemic lupus erythematosus)

*polycystic kidney disease

*pyelonephrosis

*renal artery stenosis

*recurrent severe infections

-S/sx:

*nausea, fatigue, lethargy, involuntary movement of legs, depression, intractable hiccups

*slurred speech, tremors or jerky movements, ataxia, seizures, coma

*fluid overload, hypertension, dysrhythmias, heart failure, orthostatic hypotension

-Dx:

*urinalysis: hematuria, proteinuria, and decreased in specific gravity

*serum creatinine: increased

*BUN: increased

*Serum electrolytes: decreased sodium (dilutional) and calcium; increased potassium, phosphorus, and magnesium

*CBC: decreased hemoglobin and hematocrit from anemia secondary to the loss of erythropoietin

-Tx:

*medications:

Digoxin: increases contractility of the myocardium and promotes cardiac output

Sodium polystyrene: increases elimination of serum potassium

Epoetin alfa: stimulates production of red blood cells; given for anemia

Ferrous sulfate: an iron supplement to prevent severe iron deficiency

Calcium carbonate: taken with meals to bind phosphate in food and stop phosphate absorption.

Furosemide: a loop-diuretic administered to excrete excess fluids

Acute kidney disease

-the sudden cessation of renal function that occurs when blood flow to the kidneys is significantly compromised.

-Risk factors:

*trauma

*renal vascular obstruction

*shock

*decreased cardiac output causing decreased renal perfusion

*sepsis

*hypovolemia

*peripheral vascular resistance

*liver failure

*acute nephrotoxins

*infection, vasculitis, acute glomerulonephritis

*stone, tumor, bladder atony

*spinal cord disease or injury

-S/sx:

*waste buildup

*decreased urine output

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*dysrhythmia (hyperkalemia)

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*sodium polystyrene sulfonate replaces sodium with potassium in the intestinal tract to promote potassium excretion.

ICP

Intracranial Pressure(ICP) (level, indications, nursing management)

-pressure exerted by the volume of the intracranial contents within the cranial vault

-normal ICP pressure is 10-15 mmHg

-persistent elevation of ICP extinguishes cerebral circulation, which will result in brain death if not treated urgently

*bleeding, edema and CSF take up space

-Manifestations:

-(Early)

-changes in LOC

-any change in condition (restlessness, increasing drowsiness, increased respiratory effort, purposeless movements)

-pupillary changes and impaired ocular movements

-weakness in one extremity or one side

-Headache: constant, increasing in intensity, or aggravated by movement or straining

-(Late)

-respiratory and vasomotor changes

-VS: increase in systolic blood pressure, widening of pulse pressure, and slowing of the heart rate; pulse may fluctuate rapidly from tachycardia to bradycardia; temperature increase

*Cushing's triad: bradycardia, hypertension, bradypnea

-projectile vomiting -further deterioration of LOC; stupor to coma

-hemiplegia, decortication, decerebration, or flaccidity

-respiratory pattern alterations including Cheyne-Stokes breathing and arrest

-loss of brainstem reflexes: pupil, gag, corneal, and swallowing

ICP monitoring

-a device inserted into the cranial cavity that records pressure and is connected to a monitor that shows a picture of the pressure waveforms

*monitoring ICP facilitates continual assessment and is more precise than vague manifestations

*insertion procedure is performed by a neurosurgeon in the OR, ER, or ICU

-Indications;

-early identification and treatment of increased ICP

-manifestations of increased ICP: severe HA, deteriorating LOC, restlessness, irritability, dilated or pinpoint pupils, slowness to react, altered respirations (Cheyne-Stokes), deterioration in motor function, and abnormal posturing

-Nursing Management:

-follow strict surgical aseptic technique

-perform sterile dressing changes per facility protocol

-keep drainage systems closed

-limit monitoring to 3-5 days

-irrigate the system only as needed to maintain patency

-elevate HOB (decreases pressure)

-monitor temperature

-avoid coughing, sneezing, vomiting, straining, seizures

-give antipyretics, antiemetics

-avoid restless activity

-monitor and report any condition changes

Osmotic diuretic

-Mannitol is an osmotic diuretic used to treat cerebral edema. When used for increased ICP, the medication draws fluid from the brain into the blood.

-Nursing considerations:

*administer IV to treat acute cerebral edema

*insert indwelling urinary catheter to monitor fluid and renal status. Monitor urine osmolality

*Monitor serum electrolytes and osmolality closely.

CPP

CPP (cerebral perfusion pressure)(pressure inside blood vessels)

-Closely linked to ICP

Level 70- 100

Monitoring CPP=MAP (mean arterial pressure) -ICP

*a CPP of less than 50 results in permanent neurologic damage

Burns

Burns: (full-thickness, deep partial-thickness, partial-thickness, Superficial burns)

- 1st degree-superficial
 - Area involved- the epidermis
 - Appearance
 - Pink to red
 - No blisters
 - Minimal edema
 - No eschar
 - Sensation/healing
 - painful/tender
 - Sensitive to heat
 - Heals within 3-6 days
 - No scarring
 - Examples
 - Sunburn
 - Flash burn (sudden intense heat)
- 2nd-degree type 1-superficial partial thickness
 - Area involved- damage to the entire epidermis and some parts of the dermis
 - Appearance
 - Pink to red
 - Blisters
 - Mild to moderate edema
 - No eschar
 - sensation/healing
 - Painful
 - Heals within 2-3 weeks
 - No scarring, but minor pigment changes
 - Examples
 - Flash flames and scalds

- Brief contact with a hot object
- 2nd-degree type 2- deep partial-thickness
 - Area involved- damage to the entire epidermis and deep into the dermis
 - Appearance
 - Red to white
 - Blisters rare
 - Moderate edema
 - Eschar soft and dry
 - sensation/healing
 - Painful and sensitive to touch
 - Heals within 2-6 weeks
 - Scarring likely, possible grafting
 - examples
 - Flame and scalds
 - Grease, tar, or chemical burns
 - Prolonged exposure to hot objects
- 3rd degree-full thickness burns
 - Area involved- entire epidermis and dermis
 - Appearance
 - Red, black, brown, yellow, or white
 - No blisters
 - Severe edema
 - Eschar hard and inelastic
 - sensation/healing
 - Sensation minimal or absent
 - Heals within weeks to months
 - Scarring
 - Grafting
 - Examples
 - Scalds
 - Grease, tar, or electrical burns
 - Prolonged exposure to hot objects
- 4th degree- deep full-thickness
 - Areas involved- damage to all layers of skin, extends to muscles, tendons, and bones
 - Appearance
 - Black
 - No blisters
 - No edema
 - Eschar hard and inelastic

- Sensation/healing
 - No pain
 - Heals within weeks to months
 - Scarring
 - Grafting
- examples
 - High voltage of prolonged electrical burns
 - flames

S/S with nursing actions

- Parkland formula
- What will be given as medication
- What fluids given
- Wound Care
 - Clean/Debride (using scissors & forceps) during a regular shower or w/ pt in bed
 - Extensive debridement is done in OR
 - Daily shower & dressing change done daily
 - Utilize PPE & sterile gloves when applying ointments & sterile dressings
 - Autograft & Allograft
 - Newer biosynthetic options are now available
- Administration of medications
- Nutritional Support
 - Basal metabolic rate is 40-100x higher than normal w/ burn injury
 - Maintain NPO until bowel sounds are heard, slowly advance diet
 - Diet high in protein, carbs, fats, & vitamins
 - 5,000 calories daily
 - Monitor I&Os, calorie intake, & daily weight
 - Failure to supply adequate calories & protein leads to malnutrition & delayed healing
- Emotional Support
 - Encourage patient to help with care and look at burns
 - In an acute phase, the patient may not help. This is their way of coping
 - Encourage patient to make decisions in patient's care
 - Assist client through stages of grief
 - Referrals for social workers, psychiatry, counselor, spiritual advisor
- Physical Mobility
 - Deep breathing, turning, and proper positioning
 - Specialty beds

- PROM & AROM started on the day of admission
- Assist with ambulation as early as possible
- Splints or functional devices
- OT & PT

Labs (ABGs, fluid volume, potassium, H&H)

- Elevated Hemoglobin is commonly caused by hemoconcentration resulting from fluid loss (hypovolemia). The fluid loss is total plasma water. The fluid leaves the vascular system due to the loss of the vascular permeability, which leaves the RBC in the vascular system. Which falsely makes the H&H appear high. “Up to half of the total plasma water can be lost from the vascular compartment within 2 to 3 hours after a 40% TBSA burn.⁶ Intravascular hypovolemia and an Elevated Hemoglobin is commonly caused by hemoconcentration resulting from fluid loss (hypovolemia). The fluid loss is total plasma water. The fluid leaves the vascular system due to the loss of the vascular permeability, which leaves the RBC in the vascular system. Which falsely makes the H&H appear high. “Up to half of the total plasma water can be lost from the vascular compartment within 2 to 3 hours after a 40% TBSA burn.⁶ Intravascular hypovolemia and a

Treatments including fluids, how to monitor each system, surgical procedures

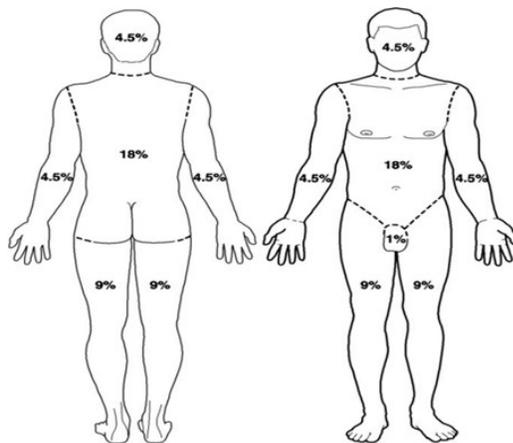
- Escharotomy
 - Incision through the eschar
 - Relieves pressure & improves circulation
- Fasciotomy
 - Incision through the eschar & fascia
 - Relieves pressure when escharotomy does not & improves circulation
- Skin coverings
 - Biologic skin coverings
 - Autograft: patient’s own skin
 - Allograft: cadaver skin
 - Xenograft: skin from animals
 - Synthetic skin coverings
 - Biosynthetic dressings
 - Wound grafts

Prioritize care

- Secure airway
- Support circulation & organ perfusion
- Manage pain
- Prevent infection
- Maintain body temp
- Provide emotional support

Rule of nines (what it is and do a calculation of it)

- A quick method to approximate the extent of burns by dividing the body into multiples of nine. The sum equals the TBSA
- Head: 4.5%, arms: 4.5, trunk: 18%, legs: 9%, genitals: 1%



Psychosocial aspects

- Provide support
- Prevent scars and contractures
- Resume activities such as work, family, and social roles

Fire safety

- Know Race (rescue, activate, contain, extinguish/evacuate)

Parkland Baxter formula

- $4 \text{ ml} \times \text{kg} \times \% \text{TBSA} = \text{total volume for 1st 24 hours}$
- Then give $\frac{1}{2}$ of total in first 8 hours
- Then give $\frac{1}{4}$ of total in the second 8 hours
- Then give $\frac{1}{4}$ of total in the third 8 hours

Diabetes insipidus

Diabetes Insipidus (DI)

-Results from a deficiency of ADH

(3)Types of DI

-Primary: a lack of ADH production or release; caused by defects in the hypothalamus or pituitary gland

-Secondary: a lack of ADH production or release; caused by infection, tumors in or near the hypothalamus or pituitary gland, head trauma, or brain surgery.

-Nephrogenic: inherited; renal tubules do not react to ADH

-Drug-induced: Lithium carbonate or demeclocycline can alter the way the kidneys respond to ADH.

Risk Factors

- head injury
- tumor or lesion (pressing on gland)
- irritation near or around pituitary gland
- surgery to pituitary gland
- infection
- Lithium carbonate or demeclocycline

Signs/Symptoms

- Polyuria (excessive urination)
- Polydipsia (excessive thirst)
- Nocturia
- Fatigue
- Dehydration
- *Physical assessment:
 - sunken eyes
 - tachycardia
 - hypotension

-loss or absence of skin turgor

-dry mucous membranes

-weak, poor peripheral pulses

-decreased cognition

Specific Gravity

-decreased urine specific gravity <1.005

-decreased urine pH

-decreased osmolality <200 mOsm/L

-decreased urine sodium

*as urine volume increases,
urine osmolality decreases

-decreased urine potassium

Diagnostics

-Water deprivation test (ADH stimulation test)

*withhold fluids for 8 to 12 hours or until 3-5% of the body weight is lost

*Patient continues to excrete large volumes of urine

**low specific gravity

**increasing serum osmolality & elevated serum sodium levels

Medications

Vasopressin (DDAVP)

-Synthetic vasopressin (ADH)

*longer duration of action, fewer side effects

*administered intranasally, orally, or parenterally

*results in increased water absorption from kidneys and decreased urine output

Thiazides

-used to treat Nephrogenic form of DI

Nursing Management

-Monitor vitals, urinary output, central venous pressure, I&O, labs (potassium, sodium, BUN, creatinine), specific gravity osmolarity

-assess skin turgor and mucous membranes

-IV therapy: hydration

-weigh daily (assess fluid loss/gain)

-maintain prescribed diet

-provide skin & mouth care

-encourage client to drink when thirsty

-monitor for medication side effects

myxedema

Myxedema Coma

-rare but serious disorder results from persistently low thyroid production

Risk Factors

*life-threatening condition that occurs when hypothyroidism is untreated or when a stressor (acute illness, surgery, chemotherapy, discontinuing thyroid replacement therapy, or use of sedatives/opioids) affects a client who has hypothyroidism

Signs/Symptoms

-respiratory failure

-hypoglycemia

-hypothermia

-hyponatremia

-coma

-hypotension

-dysrhythmias

-bradycardia

Nursing Management

-maintain patent airway with O2 administration

-provide continuous ECG monitoring

-initiate aspiration precautions

-assess the client's temperature hourly, keep client warm

-monitor blood pressure, ABGs, and electrolytes frequently

-monitor changes in mental status

Medications

- Levothyroxine

*replace thyroid hormone by administering large doses of levothyroxine IV bolus.

IV Fluids

*replace fluids with 0.9% sodium chloride IV

Intravenous Glucose

*treat hypoglycemia with glucose

Corticosteroids

*administer corticosteroids until coexisting adrenal insufficiency is ruled out.

Pericarditis

Commonly follows a respiratory infection

Can be due to an MI

Sitting or leaning forward relieves pain

Can lead to valve dysfunction

Compartment syndrome

Compartment syndrome (5 P's)

-Usually affects extremities and occurs when the pressure within 1 or more of the muscle compartments (an area covered with an elastic tissue called fascia) of the extremity compromises circulation, resulting in an ischemia-edema cycle.

-Manifestations:

-Pain: increased pain unrelieved with elevation or by pain medication; intense pain when passively moved.

-Paresthesia: or numbness, burning, and tingling are early manifestations

-Paralysis: motor weakness, or inability to move the extremity indicates major nerve damage are late manifestations.

- Pallor: the color of tissue is pale (pallor), and nails beds are cyanotic
- Pulselessness: is a late manifestation of compartment syndrome
 - *if palpated muscles are hard and swollen from edema
 - *if untreated-tissue necrosis can result; neuromuscular damage occurs within 4-6 hr.
- surgical treatment is a fasciotomy
- a surgical incision is made through the subcutaneous tissue and fascia of the affected compartment to relieve the pressure and restore circulation.
- after the fasciotomy, the open wound requires sterile packings and dressing until secondary close occurs; skin grafts might be necessary; negative pressure wound therapy can be used to reduce edema

-Nursing management:

- Prevention include the following:
 - *assess neurovascular status frequently
 - *notify the provider when compartment syndrome is suspected
 - *the provider will cut the cast on one side (univalve) or both side (bivalve)
 - *loosen the constrictive dressing or cut the bandage or tape

Addison's (adrenocortical crisis)

- cause: damage/dysfunction of adrenal cortex
- low production of mineralocorticoids and glucocorticoids
- low aldosterone and cortisol
- s/sx

*Hyperpigmentation	*weight loss	*craving for salt
*weakness, fatigue	*N/V/D or constipation	*abd pain
*dizzy w/ ortho hypotension	*dehydration	*low hyponatremia, hypoglycemia
- *increase hypercalcemia, hyperkalemia
- Acute adrenal insufficiency (Addisonian Crisis)
 - *rapid onset, medical emergency
 - *prognosis poor-if not quickly & adequately treated
- s/sx:

*shock *severe HA, confusion, restlessness *Pallor

*cyanosis *hypotension *fever

*N/V/D *abd pain *

*exposure to cold, infection, dec in salt & circulatory collapse, shock and death if untreated

*stress, surgery, dehydration can precipitate crisis

-Physical findings:

*Hyperpigmentation

-Dx:

Labs:

*Hyponatremia (crave salt) *hyperkalemia

*Hypoglycemia

*increase potassium and calcium *increase BUN and Creatinine

*decrease sodium *normal to low glucose

*decrease serum cortisol *ECG: dysrhythmias d/t electrolyte imbalance

*XR, CT, MRI to determine adrenal insufficiency caused by tumor or adrenal atrophy

-Nursing management:

*monitor electrolytes, glucose, serum cortisol

*cardiac/heart monitoring (due to electrolyte changes)

*admin insulin and dextrose *admin Ca and sodium polystyrene sulfonate

*if acidosis--sodium bicarb *fluids 0.9%

*continue to monitor vs, I&O, electrolytes, skin care

Esophageal varices

Pneumothorax

-Presence of air or gas in the pleural space that causes lung collapse

*tension pneumothorax

*spontaneous pneumothorax

-risk factors:

*blunt chest trauma

*penetrating chest wounds

*closed/occluded chest tube

*COPD

-s/sx:

*anxiety

*pleuritic pain

*signs of respiratory distress (tachypnea, tachycardia, hypoxia, cyanosis, dyspnea, & use of accessory muscles)

*tracheal deviation

*reduced or absent breath sounds

*asymmetrical chest wall movement

*dull percussion (hemothorax)

*hyperresonance on percussion d/t trapped air (pneumothorax)

Dx:

*ABGs (hypoxemia PaO₂ <80)

*CXR (confirm dx.)

*thoracentesis (surgical perforation of chest wall and pleural space w/large bore needle)

-Nursing care:

*administer O₂

*check AGs, SaO₂, CBC, CXR results

*auscultate heart and lung sounds, monitor vs every 4 hours

*document ventilator settings hourly

*monitor chest tube drainage

*position client to maximize ventilation (High-Fowler's)

*administer medications as prescribed

*encourage clients to deep breathe to promote lung expansion.

Traumatic brain injury (TBI)

Glascow Coma Scale (GCS)

-Concentrates on neurologic function and is useful to determine the LOC and monitor responses to treatment.

-reported as a number that allows providers to immediately determine if neurologic changes have occurred.

-Considerations:

*Eye opening (E): best eye response

4= eye opening occurs spontaneously

3= eye opening occurs to sound

2=eye opening occurs secondary to pain

1= eye opening does not occur

*Verbal (V): best verbal response

5= conversation is coherent and oriented

4= conversations is incoherent and oriented

3= words are spoken but inappropriately

2= sounds are made, but no words

1= vocalization does not occur

*Motor (M): best motor response

6= commands are followed

5= local reaction to pain occurs

4= general withdrawal to pain

3= decorticate posture (adduction of arms, flexion of elbows & wrist)

2= decerebrate posture (abduction of arms, extension of elbows & wrist)

1= motor response does not occur

-Interpretation of findings:

-best possible score--15

-score <8 -- associated w/ severe head injury and coma

*8= intubate (score 8 or less requires intubation)

-score 9-12--indicates moderate head injury

-score >13--associated with minor head trauma

Diabetes Mellitus (DKA, HHS, hypoglycemia)

Diabetic Ketoacidosis (DKA) vs Hyperglycemic Hyperosmolar State (HHS)

-DKA (diabetic ketoacidosis): uncontrolled hyperglycemia, metabolic acidosis, dehydration, electrolyte loss, & accumulation of ketones in the blood & urine. rapid onset.

-HHS (hyperglycemic hyperosmolar state: profound hyperglycemia, hyperosmolarity that leads to dehydration, and an absence of ketosis. gradual onset.

Signs/Symptoms

-DKA:

- polyuria
- polydipsia (excess thirst)
- polyphagia
- weight loss
- GI effects (nausea, vomiting, abdominal pain)
- blurred vision, headache, weakness
- orthostatic hypotension
- acetone breath (fruity odor)
- Kussmaul respirations
- metabolic acidosis
- mental status changes
- reversible paralysis
- seizures, myoclonic jerking
- marked fatigue
- weak, rapid pulse

-HHS:

- polyuria
- polydipsia
- polyphagia
- weight loss
- blurred vision, headache, weakness
- orthostatic hypotension
- mental status changes
- seizures, myoclonic jerking
- reversible paralysis
- dry mucous membranes
- poor skin turgor
- tachycardia
- hemiparesis (stroke)

Diagnostic

-DKA:

- Serum glucose greater than 300mg/dl
- Serum electrolytes: Na⁺ (low, normal or high); K⁺ (initial levels depend on how long DKA existed prior to treatment: then decrease with treatment)
- BUN & Creatinine: increased secondary to dehydration (BUN > 30mg/dl; Cr > 1.5mg/dl)
- Ketones present in blood and urine
- Serum osmolarity increased (high)

-Serum pH (ABGs): metabolic acidosis with respiratory compensation (Kussmaul respirations), pH less than 7.3

-HHS:

-Serum glucose: greater than 600mg/dl

-Serum electrolytes: Na⁺ (normal to low); K⁺ (normal to high as a result of dehydration; must monitor for decrease when treatment started)

-Serum BUN & Creatinine: increased secondary to dehydration (BUN >30mg/dl; Cr >1.5mg/dl)

-Ketones: absent in blood and urine

-Serum osmolarity: greater than 320 mOsm/L

-Serum pH (ABG): absence of acidosis, pH greater than 7.4

Rehydration Rule 0.9% vs 0.45%

-DKA:/-HHS:

-Isotonic solutions (0.9% NaCl)

*Hypotonic: 0.45% NaCl

-Rapid infusion for first 1-3 hours

*0.5-1 liter per hour

-Rehydrate up to 6-10 Liters of IV fluid

*BS <250 -- Dextrose 5% (prevents hypoglycemia)

Electrolyte restoration (know when K⁺ is given)

-DKA:/HHS:

-Insulin therapy will shift potassium into cells causing hypokalemia.

*provide potassium replacement in IV fluids

Insulin administration (know what type of insulin, route)

-DKA:/HHS:

-acidosis reversed with insulin

*inhibits fat breakdown--ends ketone production and acid buildup

-Regular insulin in units (IV) until it is appropriate to administer SQ insulin

*cannot stop insulin drip until SQ insulin therapy has been initiated (will see overlap of IV insulin and SQ insulin).

*hourly glucose monitoring

Traction

Traction

-uses a pulling force to promote and maintain alignment of the injured area

-traction prescriptions should include:

*type of traction

*amount of weight

*whether traction can be removed for nursing care

-Nursing Management:

-assess the neurovascular status of the affected body part every hour for 24 hrs and every 4 hrs after that

-Maintain body alignment and realign if the client seems uncomfortable or reports pain

-Avoid lifting or removing weights

-Ensure that weights hang freely and are not resting on the floor.

-If the weights are accidentally displaced, replace with weights; if the problem is not corrected, notify the provider

-Ensure that pulley ropes are free of knots, fraying, loosening, and improper positioning at least every 8 to 12 hrs

-Notify the provider if the client experiences severe pain from muscle spasm unrelieved with medications or repositioning; move the client in halo traction as a unit without applying pressure to the rods; this will prevent the loosening of pins and pain

-Routinely monitor skin integrity and document

-Use heat/massage as prescribed to treat muscle spasms

-Use therapeutic touch and relaxation techniques

-Pin Care:

-pin care is done frequently throughout immobilization (skeletal traction and external fixation methods) to prevent and to monitor for manifestations of infection

*drainage and redness (color, amount, odor)

*loosening of pins

*tenting of skin at pin site (skin rising up pin)

-pin care protocols (chlorhexidine) are based on provider preference and facility policy; a primary concept of pin care is that one cotton swab is designated for each pin to avoid cross-contamination

-pin care is provided usually once a shift, 1 to 2 times a day, or per facility protocol.

Meningitis

MODS

20. MODS: failure of 2 or more organ systems in an acutely ill patient such that homeostasis cannot be maintained without intervention

a. s/s

-Respiratory: severe dyspnea, tachypnea

*PaCO₂/FIO₂ ratio <200

*Bilateral fluffy infiltrates on CXR

*Refractory hypoxemia

-Cardiovascular: Myocardial depression

*Massive vasodilation

*Decreases SVR, BP, MAP

*Increase HR

*Biventricular failure

-Central Nervous System: Acute change in neurologic status- confusion, disorientation, delirium

*fever

*seizures

*failure to wean, prolonged rehabilitation

-Endocrine System: Hyperglycemia

-Renal system: Pre-renal

*BUN/Cr rate >20:1

Intrarenal:

*BUN/Cr rate <10:1

-Gastrointestinal system: hypoperfusion - decrease peristalsis, paralytic ileus

*GI bleeding

-Hepatic system: Bilirubin >2, increased LFTs

*Hepatic encephalopathy

-Hematologic system: coagulation (increased PT & PTT, decreased platelet count)

*increased d-dimer

b. Nursing Management

-Prevention remains the top priority

-early detection & documentation of initial signs of infection are essential

*primary nursing interventions are aimed at supporting the patient and monitoring organ perfusion until primary organ insults are halted

*providing information and support to family members is a critical role of the nurse - health care team must address end-of-life decisions to ensure that supportive therapies are congruent with the patient's wishes.

*patients who survive MODS must be informed about the goals of rehabilitation and expectations for progress toward these goals because the massive loss of skeletal muscle mass makes rehabilitation a long, slow process

Compartment syndrome

Heat stroke

Signs and symptoms

- Temperature > 104

- Lack of perspiration
- Hypotension
- Tachycardia
- AMS
- Abnormal potassium and sodium levels
- Management:
 - Cool sheets/towels
 - Cold bath
 - Ice
 - Cooling blanket
 - Environmental controls
 - Cold bath (if able)
 - Rectal thermometer for CORE TEMP
 - Monitor airway, VS, ECG, mental status, urinary output, and labs
- GOAL: REDUCE TEMP

Emergency/Triage (ESI level, mass casualty)

Be able to prioritize patients

-5 levels of triage system:

*Resuscitation- Level 1 (ex. cardiac arrest)	15 min
*Emergent- Level 2 (ex. CP w/cardiac hx)	30 min
*Urgent- Level 3 (ex. abdominal pain)	60 min
*Less Urgent- level 4 (ex. laceration)	120 min
*Nonurgent- level 5 (ex. simple rash)	120 min

Examples:

- Level 5: no resources. This patient will need an eye exam and will be discharged to home with prescriptions and an appointment to follow up with an ophthalmologist.
- Level 1:suspected medication overdose. Respirations are 10 and her spo2 on room air is 86%. Requires immediate life-saving intervention. The patient's respiratory rate, oxygen saturation, and inability to protect her own airway indicate the need for immediate endotracheal intubation.
- Level 3: patient requires an x-ray of his right arm and sutures to his elbow. 2 or more resources needed.

- Level 2: high risk. Suspected DVT due to onset shortness of breath, respiratory distress, and history of birth control pill administration.
- Level 4: one resource needed. Urinalysis and urine culture. Most likely has a UTI and will need oral medications.

Definition	ESI-1	ESI-2	ESI-3	ESI-4	ESI-5
Stability of vital functions (ABCs)	Unstable	Threatened	Stable	Stable	Stable
Life threat or organ threat	Obvious	Likely but not always obvious	Unlikely but possible	No	No
How soon patient should be seen by HCP	Immediately	Within 10 min	Up to 1 hr	Could be delayed	Could be delayed
Expected resource intensity	<ul style="list-style-type: none"> • High resource intensity • Staff at bedside continuously • Often mobilization of team response 	<ul style="list-style-type: none"> • High resource intensity • Multiple, often complex diagnostic studies • Frequent consultation • Continuous monitoring 	<ul style="list-style-type: none"> • Medium to high intensity • Multiple diagnostic studies • Complex procedures 	<ul style="list-style-type: none"> • Low resource intensity • One simple diagnostic study 	<ul style="list-style-type: none"> • Low resource intensity • Examination only
Examples	Cardiac arrest, intubated trauma patient, overdose w/ bradypnea, severe respiratory distress	Chest pain probably resulting from ischemia, multiple trauma unless responsive	Abdominal pain or gynecological disorders unless in severe distress, hip fracture in older patient	Closed extremity trauma, simple laceration, cystitis	Cold symptoms, minor burn, recheck (e.g. wound), prescription refill

Know how to color code triage patients and actions for those colors

- RED-immediate
 - Life-threatening injuries but survivable w/ minimal intervention
 - Can progress rapidly if treatment is delayed
 - **First** priority
 - Example: sucking chest wound, airway obstruction secondary to mechanical cause, shock, hemothorax, tension pneumothorax, asphyxia, unstable chest or abdominal wounds, incomplete amputations, open fractures of long bones, 2nd and 3rd-degree burns of 15%-40% of total body surface area, incomplete/stabilize fractures
- YELLOW- delayed
 - Injuries are significant and require medical care but can wait hours without threat to life or limb
 - **Second** priority-commonly non-ambulatory with certain exceptions
 - Example: wounds without evidence of significant hemorrhage, soft tissue injuries, maxillofacial wounds without airway compromise, vascular injuries with adequate collateral circulation, genitourinary tract disruption,

fractures requiring open reduction, debridement, external fixation, most **eye**, and central nervous system injuries

- GREEN-minimal
 - Injuries are minor and treatment can be delayed hours to days
 - Move away from the main triage area
 - **Third** priority- typically on their feet, not always
 - Example: upper extremity fractures, minor burns, sprains, small lacerations without significant bleeding, behavioral disorders or psychological disturbances
- BLACK- Expectant
 - Injuries are extensive and unlikely to survive even with definitive care
 - Separate from others but do not abandon
 - Comfort care
 - **Last** priority
 - Example: unresponsive, penetrating head wounds, high spinal cord injuries, wounds involving multiple anatomic sites and organs, 2nd and 3rd-degree burns in excess of 60% of body surface areas, seizures or vomiting within 24 hours after radiation exposure, profound shock with multiple injuries, agonal respirations, no pulse, no blood pressure, pupils fixed and dilated