Fluid, Electrolyte and Acid-Base Balance

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Fluid Balance

General Concepts Intake = Output = Fluid Balance

Sensible losses ; **Urination .Defecation .Wound drainage**

Insensible losses ; **Evaporation from skin .Respiratory loss from lungs**

Extracellular 20% of body weight

Two types

Fluid Compartments ; **Intracellular 40% of body weight .**

Extracellular 20% of body weight

INTERSTITIAL (between)- INTRAVASCULAR (inside)

Age-Related Fluid Changes; **Full-term baby - 80%**
Lean Adult Male - 60%- Aged client - 40%

- **Fluid and Electrolyte Transport**; **PASSIVE TRANSPORT SYSTEMS** (**Diffusion- Filtration- Osmosis**) **ACTIVE TRANSPORT SYSTEM Pumping Requires energy expenditure**

- **Diffusion Molecules move across a biological membrane from an area of higher to an area of lower concentration**

Membrane types ;Permeable -Semi-permeabl- Impermeable

- **Filtration** Movement of solute and solvent across a membrane caused by **hydrostatic (water pushing) pressure** Occurs at the capillary level If normal pressure gradient changes (as occurs with right-sided heart failure) edema results from "third spacing"

- **Osmosis** Movement of solvent from an area of lower solute concentration to one of higher concentration Occurs through a semipermeable membrane using **osmotic (water pulling) pressure**

- **Active** Transport System Solutes can be moved against a concentration gradient Also called "**pumping**" **Dependent on the presence of ATP**

- **Fluid Types;** Isotonic- Hypotonic- Hypertonic

•**Isotonic Solution**; No fluid shift because solutions are equally concentrated **Normal saline solution** (0.9% NaCl)

•**Hypotonic Solution**; Lower solute concentration Fluid shifts from hypotonic solution into the more concentrated solution to create a balance (cells swell) **Half-normal saline solution** (0.45% NaCl)

•**Hypertonic Solution**; Higher solute concentration Fluid is drawn into the hypertonic solution to create a balance (cells shrink) **5% dextrose in normal saline** (D5/0.9% NaCl)

•**Regulatory Mechanisms**; Baroreceptor reflex Volume receptors Renin-angiotensin-aldosterone mechanism Antidiuretic hormone

•**Baroreceptor Reflex** Respond to a fall in arterial blood pressure Located in the atrial walls, vena cava, aortic arch and carotid sinus Constricts afferent arterioles of the kidney resulting in retention of fluid

- **Volume Receptors** Respond to fluid excess in the atria and great vessels Stimulation of these receptors creates a strong renal response that increases

- **Renin-Angiotensin-Aldosterone** Renin Enzyme secreted by kidneys when arterial pressure or volume drops Interacts with angiotensinogen to form angiotensin I (vasoconstrictor)

- Angiotensin I is converted in lungs to angiotensin II using ACE (angiotensin converting enzyme) Produces vasoconstriction to elevate blood pressure Stimulates adrenal cortex to secrete aldosterone

- **Aldosterone** Mineralocorticoid that controls Na^+ and K^+ blood levels Increases Cl^- and HCO_3^- concentrations and fluid volume

•Aldosterone Negative Feedback Mechanism;

• ECF & Na+ levels drop secretion of ACTH by the anterior pituitary release of aldosterone by the adrenal cortex fluid and Na+ retention

•Antidiuretic Hormone Also called vasopressin Released by posterior pituitary when there is a need to restore intravascular fluid volume.

• Release is triggered by osmoreceptors in the thirst center of the hypothalamus

•Fluid volume excess decreased ADH Fluid volume deficit increased ADH

•Fluid Imbalances; Dehydration- Hypovolemia -Hypervolemia -Water intoxication

•Dehydration; Loss of body fluids increased concentration of solutes in the blood and a rise in serum Na+ levels- Fluid shifts out of cells into the blood to restore balance -Cells shrink from fluid loss and can no longer function properly

•Clients at Risk Confused Comatose Bedridden Infants Elderly Enterally fed

•**What Do You See?** Irritability Confusion Dizziness Weakness Extreme thirst urine output- Fever- Dry skin/mucous membranes -Sunken eyes -Poor skin turgor Tachycardia

•**What Do We Do?** Fluid Replacement - oral or IV over 48 hrs. Monitor symptoms and vital signs Maintain I&O Maintain IV access-- Daily weights Skin and mouth care

•**Hypovolemia** Isotonic fluid loss from the extracellular space Can progress to hypovolemic shock Caused by: Excessive fluid loss (hemorrhage)- Decreased fluid intake- Third space fluid shifting

•**What Do You See?** Mental status deterioration- Thirst- Tachycardia- Delayed capillary refill -Orthostatic hypotension- Urine output < 30 ml/hr Cool, pale extremities Weight loss

•**What Do We Do?** Fluid replacement- Albumin replacement- Blood transfusions for hemorrhage- Dopamine to maintain BP MAST trousers for severe shock .
•**Assess for fluid overload with treatment**

•**Hypervolemia;** Excess fluid in the **extracellular compartment** as a result of fluid or sodium retention, excessive intake, or renal failure.
• Occurs when compensatory mechanisms fail to restore fluid balance Leads to CHF and pulmonary edema

•**What Do You See?** Tachypnea- Dyspnea- Crackles Rapid, bounding pulse Hypertension- S_3 gallop- Increased CVP, pulmonary artery pressure and pulmonary artery wedge pressure (Swan-Ganz) -Acute weight gain Edema

•**Edema** Fluid is forced into tissues by the hydrostatic pressure- First seen in dependent areas.
• **Anasarca** - severe generalized edema- Pitting edema- Pulmonary edema

•What Do We Do? Fluid and Na+ restriction- Diuretics -Monitor vital signs -Hourly I&O Breath sounds- Monitor ABGs and give O_2 as ordered.
• Maintain IV access Skin & mouth care -Daily weights

•Water Intoxication; Hypotonic extracellular fluid shifts into cells to attempt to restore balance Cells swell Causes: SIADH- Rapid infusion of hypotonic solution Excessive tap- water NG irrigation or enemas- Psychogenic polydipsia

•What Do You See? Signs and symptoms of increased intracranial pressure Early: change in LOC, N/V, muscle weakness, twitching, cramping Late: bradycardia, widened pulse pressure, seizures, coma

•What Do We Do? Prevention is the best treatment-- Assess neuro status Monitor I&O and vital signs- Fluid restrictions IV access- Daily weights- Monitor serum Na+- Seizure precautions

•Electrolytes

•Electrolytes Charged particles in solution Cations (+) Anions (-) -Integral part of metabolic and cellular processes

•Positive or Negative? Cations (+) Sodium Potassium Calcium Magnesium Anions (-) Chloride Bicarbonate Phosphate Sulfate

•Major Cations; ELLULAEXTRACR SODIUM (Na+) INTRACELLULAR POTASSIUM (K+)

•Electrolyte Imbalances; Hyponatremia/ hypernatremia Hypokalemia/ Hyperkalemia Hypomagnesemia/ Hypermagnesemia Hypocalcemia/ Hypercalcemia Hypophosphatemia/ Hyperphosphatemia Hypochloremia/ Hyperchloremia

•Sodium Major extracellular cation Attracts fluid and helps preserve fluid volume Combines with chloride and bicarbonate to help regulate acid-base balance Normal range of serum sodium 135 - 145 mEq/L

•**Sodium and Water;** If sodium intake suddenly increases, extracellular fluid concentration also rises Increased serum Na+ increases thirst and the release of **ADH**, which triggers kidneys to retain water.
• **Aldosterone** also has a function in water and sodium conservation when serum Na+ levels are low

•**Sodium-Potassium Pump;** Sodium (abundant outside cells) tries to get into cells Potassium (abundant inside cells) tries to get out of cells Sodium-potassium -pump maintains normal concentrations -Pump uses **ATP**, magnesium and an enzyme to maintain sodium-potassium concentrations.
• **Pump** prevents cell swelling and creates an electrical charge allowing neuromuscular impulse transmission

•**Hyponatremia** Serum Na+ level < 135 mEq/L- Deficiency in Na+ related to amount of body fluid= **Several types;** Dilutional- Depletional- Hypovolemic- Hypervolemic Isovolemic

•**What Do You See?** Primarily neurologic symptoms Headache, N/V, muscle twitching, altered mental status, stupor, seizures, coma- **Hypovolemia** - poor skin turgor, tachycardia, decreased BP, orthostatic hypotension **Hypervolemia** - edema, hypertension, weight gain, bounding tachycardia

•**What Do We Do? MILD CASE** Restrict fluid intake for hyper/isovolemic hyponatremia IV fluids and/or increased po Na+ intake for hypovolemic hyponatremia- **SEVERE CASE** Infuse hypertonic NaCl solution (3% or 5% NaCl) Furosemide to remove excess fluid- Monitor client in ICU

•**Hypernatremia** Excess Na+ relative to body water Occurs **less often than hyponatremia** **Thirst** is the body's main defense- **When hypernatremia occurs**, fluid shifts outside the cells May be caused by water deficit or over-ingestion of Na+- Also may result from diabetes insipidus

•**What Do You See?** Think S-A-L-T- Skin flushed- Agitation- Low grade fever -Thirst Neurological symptoms Signs of hypovolemia

•**What Do We Do?** Correct underlying disorder Gradual fluid replacement Monitor

•**Potassium Major intracellular** cation. Untreated changes in K+ levels can lead to serious neuromuscular and cardiac problems -Normal K+ levels = 3.5 - 5 mEq/L

•**Balancing Potassium** Most K+ ingested is excreted by the kidneys.
• Three other influential factors in K+ balance : Na+/K+ pump- Renal regulation- pH level

•**Sodium/Potassium Pump** Uses ATP to pump potassium into cells Pumps sodium out of cells Creates a balance

•**Renal Regulation** Increased K+ levels increased K+ loss in urine Aldosterone secretion causes Na+ reabsorption and K+ excretion

•pH Potassium ions and hydrogen ions exchange freely across cell membranes Acidosis hyperkalemia (K+ moves out of cells) Alkalosis hypokalemia (K+ moves into cells)

•Hypokalemia Serum K+ < 3.5 mEq/L. Can be caused by GI losses, diarrhea, insufficient intake, non-K+ sparing diuretics (thiazide, furosemide)

•What Do You See? Think S-U-C-T-I-O-N Skeletal muscle weakness- U wave (EKG changes)- Constipation/ ileus- Toxicity of digitalis glycosides- Irregular, weak pulse- Orthostatic hypotension- Numbness (paresthesias)

•What Do We Do? Increase dietary K+- Oral KCl supplements- IV K+ replacement Change to K+-sparing diuretic -Monitor EKG changes

•IV K+ Replacement Mix well when adding to an IV solution bag Concentrations should not exceed 40-60 mEq/L Rates usually 10-20 mEq/hr -NEVER GIVE IV PUSH POTASSIUM

•Hyperkalemia Serum K+ > 5 mEq/L- Less common than hypokalemia-
• Caused by altered kidney function, increased intake (salt substitutes), blood transfusions, meds (K+-sparing diuretics), cell death (trauma)

•What Do You See? Irritability- Paresthesia- Muscle weakness (especially legs)- EKG changes (tented T wave)- Irregular pulse- Hypotension- Nausea, abdominal cramps, diarrhea

•What Do We Do? Mild Loop diuretics (Lasix)- Dietary restriction- Moderate Kayexalate- Emergency 10% calcium gluconate for cardiac effects- Sodium bicarbonate for acidosis

•Magnesium Helps produce ATP Role in protein synthesis & carbohydrate metabolism Helps cardiovascular system function (vasodilation)- Regulates muscle contractions

•**Hypomagnesemia** Serum Mg++ level < 1.5 mEq/L- Caused by poor dietary intake, poor GI absorption, excessive GI/urinary losses. **High risk clients** Chronic alcoholism Malabsorption -GI/urinary system disorders- Sepsis- Burns. Wounds needing debridement

•**What Do You See?** CNS Altered –LOC- Confusion- Hallucinations

•**What Do You See?** Neuromuscular- Muscle weakness- Leg/foot cramps -Hyper DTRs Tetany- Chvostek's & Trousseau's signs

•**What Do You See?** Cardiovascular- Tachycardia- Hypertension- EKG changes

•**What Do You See?** Gastrointestinal- Dysphagia- Anorexia -Nausea/vomiting

- **What Do We Do?** Mild Dietary replacement -**Severe** IV or IM magnesium sulfate Monitor Neuro status Cardiac status Safety

- **Mag Sulfate Infusion** Use infusion pump - no faster than 150 mg/min- Monitor vital signs for hypotension and respiratory distress- Monitor serum Mg++ level q6h Cardiac monitoring- **Calcium gluconate as an antidote for overdosage**

- **Hypermagnesemia** Serum Mg++ level > 2.5 mEq/L- Not common -Renal dysfunction is most common cause -Renal failure- Addison's disease -Adrenocortical insufficiency- Untreated DKA

- **What Do You See?** Decreased neuromuscular activity- Hypoactive DTRs Generalized weakness- Occasionally nausea/vomiting

•**What Do We Do?** Increased fluids if renal function normal-loop diuretic if no response to fluids- Calcium gluconate for toxicity- Mechanical ventilation for respiratory depression- Hemodialysis (Mg++-free dialysate)

•**Calcium:** 99% in bones, 1% in serum and soft tissue (measured by serum Ca++) Works with phosphorus to form bones and teeth- Role in cell membrane permeability -Affects cardiac muscle contraction -Participates in blood clotting

•**Calcium Regulation Affected** by body stores of Ca++ and by dietary intake & Vitamin D intake- **Parathyroid hormone** draws Ca++ from bones increasing low serum levels (Parathyroid pulls) With high Ca++ levels, **calcitonin** is released by the thyroid to inhibit calcium loss from bone (Calcitonin keeps)

•**Hypocalcemia** Serum calcium < 8.5 mg/dl- Ionized calcium level < 4.5 mg/Dl- **Caused by** inadequate intake, malabsorption, pancreatitis, thyroid or parathyroid surgery, loop diuretics, low magnesium levels

•**What Do You See?** Neuromuscular Anxiety, confusion, irritability, muscle twitching, paresthesias (mouth, fingers, toes), tetany- Fractures- Diarrhea Diminished response to digoxin -EKG changes

•**What Do We Do?** Calcium gluconate for postop thyroid or parathyroid client- Cardiac monitoring- Oral or IV calcium replacement

•**Hypercalcemia** Serum calcium > 10.5 mg/dl- Ionized calcium > 5.1 mg/dl -**Two major causes** Cancer- Hyperparathyroidism

•What Do You See? Fatigue, confusion, lethargy, coma- Muscle weakness, hyporeflexia- Bradycardia cardiac arrest- Anorexia, nausea/vomiting, decreased bowel sounds, constipation- Polyuria, renal calculi, renal failure

•What Do We Do? If asymptomatic, treat underlying cause- Hydrate the patient to encourage diuresis Loop diuretics -Corticosteroids

•Phosphorus The primary anion in the intracellular fluid Crucial to cell membrane integrity, muscle function, neurologic function and metabolism of carbs, fats and protein Functions in ATP formation, phagocytosis, platelet function and formation of bones and teeth

•**Hypophosphatemia** Serum phosphorus < 2.5 mg/dl -Can lead to organ system failure **Caused by** respiratory alkalosis (hyperventilation), insulin release, malabsorption, diuretics, DKA, elevated parathyroid hormone levels, extensive burns

•**What Do You See? Musculoskeletal** muscle weakness- respiratory muscle failure osteomalacia- pathological fractures. **CNS** confusion, anxiety, seizures, coma. **Cardiac** hypotension- decreased cardiac output. **Hematologic** hemolytic anemia- easy bruising infection risk

•**What Do We Do? MILD/MODERATE** Dietary interventions -Oral supplements. **SEVERE** IV replacement- using potassium phosphate or sodium phosphate

•**Hyperphosphatemia** Serum phosphorus > 4.5 mg/dl- **Caused by** impaired kidney function, cell damage, hypoparathyroidism, respiratory acidosis, DKA, increased dietary intake

•What Do You See? Think C-H-E-M-O; Cardiac irregularities- Hyperreflexia- Eating poorly Muscle weakness -Oliguria

•What Do We Do? Low-phosphorus diet Decrease- absorption with antacids that bind phosphorus- Treat underlying cause of respiratory acidosis or DKA -IV saline for severe hyperphosphatemia in patients with good kidney function

•Chloride Major extracellular anion -Sodium and chloride maintain water balance.
• Secreted in the stomach as hydrochloric acid- Aids carbon dioxide transport in blood

•Hypochloremia Serum chloride < 96 mEq/L- Caused by decreased intake or decreased absorption, metabolic alkalosis, and loop, osmotic or thiazide diuretics

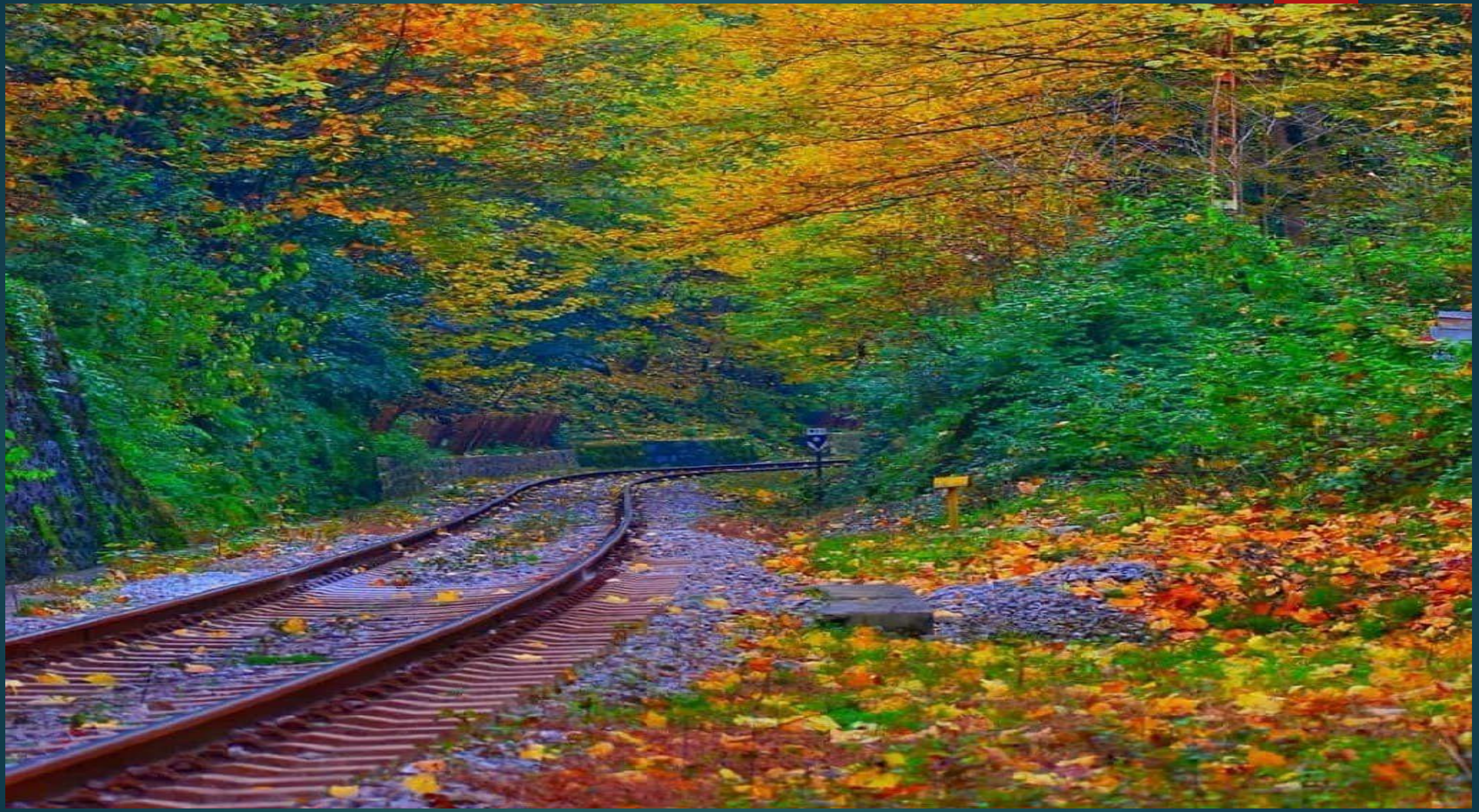
•What Do You See? Agitation, irritability Hyperactive DTRs, tetany Muscle- cramps, hypertonicity Shallow, slow respirations- Seizures, coma Arrhythmias

- **What Do We Do?** Treat underlying cause- Oral or IV replacement in a sodium chloride or potassium chloride solution

- **Hyperchloremia** Serum chloride > 106 mEq/L- Rarely occurs alone- **Caused by** dehydration, renal failure, respiratory alkalosis, salicylate toxicity, hyperparathyroidism, hyperaldosteronism, hypernatremia

- **What Do You See?** Metabolic Acidosis- Decreased LOC- Kussmaul's respirations Weakness- Hypernatremia- Agitation- Tachycardia, dyspnea, tachypnea, HTN Edema

- **What Do We Do?** Correct underlying cause- Restore fluid, electrolyte and acid-base balance- IV Lactated Ringer's solution to correct acidosis



- Acid-Base Balance

- Acid-Base Basics Balance depends on regulation of free hydrogen ions Concentration of hydrogen ions is measured in pH Arterial blood gases are the major diagnostic tool for evaluating acid-base balance

- Arterial Blood Gases pH 7.35 - 7.45 $PaCO_2$ 35 - 45 mmHg HCO_3 22-26 mEq/L

- Acidosis pH < 7.35 Caused by accumulation of acids or by a loss of bases

- Alkalosis pH > 7.45 Occurs when bases accumulate or acids are lost

- Regulatory Systems Three systems come into play when pH rises or falls Chemical buffers Respiratory system Kidneys

•Chemical Buffers Immediate acting Combine with offending acid or base to neutralize harmful effects until another system takes over Bicarb buffer - mainly responsible for buffering blood and interstitial fluid Phosphate buffer - effective in renal tubules Protein buffers - most plentiful - hemoglobin

•Respiratory System Lungs regulate blood levels of CO_2 $CO_2 + H_2O$ = Carbonic acid High CO_2 = slower breathing (hold on to carbonic acid and lower pH) Low CO_2 = faster breathing (blow off carbonic acid and raise pH) Twice as effective as chemical buffers, but effects are temporary

•Kidneys Reabsorb or excrete excess acids or bases into urine Produce bicarbonate Adjustments by the kidneys take hours to days to accomplish Bicarbonate levels and pH levels increase or decrease together

- Arterial Blood Gases (ABG) Uses blood from an arterial puncture Three test results relate to acid-base balance pH $PaCO_2$ HCO_3

- Interpreting ABGs Step 1 - check the pH Step 2 - What is the CO_2? Step 3 - Watch the bicarb Step 4 - Look for compensation Step 5 - What is the PaO_2 and SaO_2?

- Step 1 - Check the pH pH < 7.35 = acidosis pH > 7.45 = alkalosis Move on to Step 2

- Step 2 - What is the CO_2? $PaCO_2$ gives info about the respiratory component of acid-base balance If abnormal, does the change correspond with change in pH? High pH expects low $PaCO_2$ (hypocapnia) Low pH expects high $PaCO_2$ (hypercapnia)

- Step 3 – Watch the Bicarb Provides info regarding metabolic aspect of acid-base balance If pH is high, bicarb expected to be high (metabolic alkalosis) If pH is low, bicarb expected to be low (metabolic acidosis)

- Step 4 – Look for Compensation If a change is seen in BOTH $PaCO_2$ and bicarbonate, the body is trying to compensate Compensation occurs as opposites, (Example: for metabolic acidosis, compensation shows respiratory alkalosis)

- Step 5 – What is the PaO_2 and SaO_2 PaO_2 reflects ability to pickup O_2 from lungs SaO_2 less than 95% is inadequate oxygenation Low PaO_2 indicates hypoxemia

- Acid-Base Imbalances Respiratory Acidosis Respiratory Alkalosis Metabolic Acidosis Metabolic Alkalosis

•Respiratory Acidosis Any compromise in breathing can result in respiratory acidosis Hypoventilation carbon dioxide buildup and drop in pH Can result from neuromuscular trouble, depression of the brain's respiratory center, lung disease or airway obstruction

•Clients At Risk Post op abdominal surgery Mechanical ventilation Analgesics or sedation

•What Do You See? Apprehension, restlessness Confusion, tremors Decreased DTRs Diaphoresis Dyspnea, tachycardia N/V, warm flushed skin

•ABG Results Uncompensated pH < 7.35 $PaCO_2$ >45 HCO_3 Normal Compensated pH Normal $PaCO_2$ >45 HCO_3 > 26

- **What Do We Do? Correct underlying cause Bronchodilators Supplemental oxygen Treat hyperkalemia Antibiotics for infection Chest PT to remove secretions Remove foreign body obstruction**

- **Respiratory Alkalosis Most commonly results from hyperventilation caused by pain, salicylate poisoning, use of nicotine or aminophylline, hypermetabolic states or acute hypoxia (overstimulates the respiratory center)**

- **What Do You See? Anxiety, restlessness Diaphoresis Dyspnea (rate and depth) EKG changes Hyperreflexia, paresthesias Tachycardia Tetany**

- **ABG Results Uncompensated pH > 7.45 $PaCO_2$ < 35 HCO_3 Normal Compensated pH Normal $PaCO_2$ < 35 HCO_3 < 22**

•What Do We Do? Correct underlying disorder Oxygen therapy for hypoxemia Sedatives or antianxiety agents Paper bag breathing for hyperventilation

•Metabolic Acidosis Characterized by gain of acid or loss of bicarb Associated with ketone bodies Diabetes mellitus, alcoholism, starvation, hyperthyroidism Other causes Lactic acidosis secondary to shock, heart failure, pulmonary disease, hepatic disease, seizures, strenuous exercise

•What Do You See? Confusion, dull headache Decreased DTRs S/S hyperkalemia (abdominal cramps, diarrhea, muscle weakness, EKG changes) Hypotension, Kussmaul's respirations Lethargy, warm & dry skin

- **ABG Results Uncompensated pH < 7.35 $PaCO_2$ Normal HCO_3 < 22 Compensated pH Normal $PaCO_2$ < 35 HCO_3 < 22**

- **What Do We Do? Regular insulin to reverse DKA IV bicarb to correct acidosis Fluid replacement Dialysis for drug toxicity Antidiarrheals**

- **Metabolic Alkalosis Commonly associated with hypokalemia from diuretic use, hypochloremia and hypocalcemia Also caused by excessive vomiting, NG suction, Cushing's disease, kidney disease or drugs containing baking soda**

- **What Do You See? Anorexia Apathy Confusion Cyanosis Hypotension Loss of reflexes Muscle twitching Nausea Paresthesia Polyuria Vomiting Weakness**

- **ABG Results Uncompensated pH > 7.45 $PaCO_2$ Normal HCO_3 >26 Compensated pH Normal $PaCO_2$ > 45 HCO_3 > 26**

- **What Do We Do? IV ammonium chloride D/C thiazide diuretics and NG suctioning Antiemetics**

- **IV Therapy Crystalloids – volume expander Isotonic (D5W, 0.9% NaCl or Lactated Ringers) Hypotonic (0.45% NaCl) Hypertonic (D5/0.9% NaCl, D5/0.45% NaCl) Colloids – plasma expander (draw fluid into the bloodstream) Albumin Plasma protein Dextran**

- **Total Parenteral Nutrition Highly concentrated Hypertonic solution Used for clients with high caloric and nutritional needs Solution contains electrolytes, vitamins, acetate, micronutrients and amino acids Lipid emulsions given in addition**

The End
(Whew!!!!!!)