LECTURE 3

FLUIDS AND ELECTROLYTES

Review of Lecture 2

By this time, you should be comfortable with the three fluid compartments of the body and the partitions that separate them and control their compositions. The roles of hydrostatic and osmotic pressures in the shifting of fluids from one compartment to another should be "second nature" to you. You should have also practiced working through the basic physiology underlying a number of fluid imbalances and you should be capable of assessing a client experiencing a fluid imbalance.

Now it is important to realize that fluids of the body do not exist independently. They have a number of chemical or cellular elements dissolved or suspended within them. Electrolytes, capable of conducting electrical current, exist dissolved in the fluid compartments of the body. It is because of these dissolved electrolytes and their capacity to conduct current, that it is possible to measure brain activity (EEG) as well as heart (EKG) and other muscle (EMG) by placing electrodes on the body surface. However, electrolytes also serve major physiological functions which are in danger of failing should an electrolyte imbalance occur.

Continue your text reading through Chapter 10 before proceeding.

Normal Electrolyte Functions

By chemical definition, electrolytes are charged particles (ions). These particles exist dissolved in the various fluid compartments of the body (intravascular, interstitial, and intracellular) and perform a variety of functions in the total physiology of the human body. The electrolytes of importance at this point in the course are: (1) Sodium; (2) Potassium; (3) Calcium; (4) Hydrogen; and (5) Bicarbonate. Since hydrogen and bicarbonate ions are primarily involved in pH balance, their discussion will be delayed until that point. Sodium, potassium and calcium will be considered here. Recall from the prior discussion of fluid composition that sodium is primarily an extracellular ion, that potassium occurs primarily intracellularly, and that calcium performs a variety of functions. The important role of calcium in this discussion is its control of membrane permeability. Being a positive ion, calcium that associates with the plasma membrane

and serves to repel other positive ions (like charge repulsions) to help control the membrane's permeability to positive ions.

Recall from your background study of excitable cells, such as nerve and muscle, that sodium and potassium are essential for the action potentials conducted along the surface membranes of these cell types. Sodium, an extracellular ion, enters an excitable cell during the depolarization phase of the action potential. Whereas potassium, an intracellular ion, leaves an excitable cell during the repolarization phase of the action potential. All cells, including excitable cells in the "resting" state (not conducting an action potential), are polarized such that the interior is negative relative to the exterior. This difference in net charge across a cell's membrane is due in part to the intracellular proteins and body pH. Since normal body pH is slightly alkaline (7.4), proteins lose hydrogen ions (behave like acids) and show a net negative charge. Since most proteins are intracellular, when body pH is normal the positively charged intracellular potassium ions are offset by the negatively charged intracellular proteins to give the cell's interior a net negative charge compared to the outside where proteins are deficient.

REVIEW QUESTIONS:

1. How do excitable cells differ from other cells of the body regarding their response to stimulation?

2. Refamiliarize yourself with the four phases of an action potential: (1) depolarization; (2) repolarization; (3) hyperpolarization; and (4) return to resting state.

3. What are the ion movements associated with each phase of the action potential?

4. What would you predict might occur with an excitable cell if the normal locations of sodium and potassium were to be reversed?

Electrolyte Disorders / Assessment

People who have disturbances in either sodium, potassium, or calcium are probably going to show signs and symptoms of these disturbances in organ systems whose normal functions depend upon action potentials, particularly neuromuscular systems. Consequently, clients showing lethargy and muscle weakness or those with increased irritability may have an electrolyte imbalance. The assessment problem then becomes one of identifying the electrolyte(s) involved and whether they are abnormally high (hyper-) or low (hypo-).

Electrolyte imbalances may be "primary" or "seconday" in origin. A primary electrolyte imbalance usually affects only one electrolyte and typically involves an abnormality in either the intake or output of the ion of interest. For example, a high salt diet can result in hypernatremia while some diuretics "waste" potassium and can cause hypokalemia. Since electrolytes are assayed in blood samples, you are seeing the

intravascular concentrations expressed on a lab report. Because of capillary pore permeability, these changes probably also appear in the interstitial fluid as well and can, in some instances, affect intracellular concentrations. Consequently, you must know where each of the three electrolytes occur normally so you can predict the causes and effects of their changing concentrations. Since electrolytes are osmotically active, they can cause fluid shifts as well.

REVIEW QUESTIONS:

1. Why would hyperkalemia cause cardiac arrest?

2. How does an EKG change with increasing potassium concentrations?

A **seconday electrolyte imbalance** is one resulting from an abnormality in some other physiological function. Secondary imbalances usually affect **more than one electrolyte** and are common with fluid imbalances since they occur as a result of concentration or dilution of body fluids. For example, in renal failure the kidneys fail to output urine and can result in increased concentrations of all electrolytes normally excreted in the urine. Secondary imbalances are usually detected by looking first for fluid imbalances. Skin turgor ("tenting" in dehydration and "pitting" in edema) is a good fluid balance indicator as are sudden weight changes, blood pressure abnormalities, and peripheral or pulmonary edema. Fluid imbalances often appear on lab reports as changes in hematocrit (percentage of formed elements in blood). A high hematocrit suggests a possible concentration of blood maybe due to dehydration while a low hematocrit can appear with fluid overloads.

The table below covers electrolyte imbalances that involve Na, K, and Ca. Work through the table as you did in previous exercises being certain that you can explain the basic physiology underlying each **cause** and each **clinical manifestation** appearing in the table.

You should take each one of the imbalances and be sure that you can explain the underlying physiological abnormality underlying each cause and each clinical manifestation. This is an excellent review of basic physiology!!

ELECTROLYTE IMBALANCES

SODIUM IMBALANCES (PRIMARY AND SECONDARY)

CONDITION	CAUSE	CLINICAL MANIFESTATIONS
Hyponatremia	Decreased intake and adrenal insufficiency (1^0); inappropriate ADH; diaphoresis with water replacement; diuretic therapy	Cellular swelling with cerebral edema leading to headache, stupor and coma; muscle weakness; decreased thirst; edema if secondary to hypervolemia;
Hypernatremia	Increased intake or renal failure	Cellular shrinking with increased CNS

	(1^0); water deprivation; decreased ADH secretion; increased aldosterone; liver failure; hypothalamic lesion	irritability; increased thirst; hypotension with oliguria if secondary to hypovolemia

POTASSIUM IMBALANCES (PRIMARY AND SECONDARY)

CONDITION	CAUSE	CLINICAL MANIFESTATIONS
Hypokalemia	Decreased intake, adrenal cortex hyperfunction and diuretic therapy (1^0); alkalosis; vomiting/gastric suction	Cardiac arhythmia (lower T and appearance of U wave due to slow repolarization) and muscle weakness;
Hyperkalemia	Increased intake or renal failure and hypoaldosteronism (1^0); acidosis; RBC hemolysis;	Cardiac depression (shallow, wide QRS with elevated T due to exaggerated repolarization); paresthesia and/or paralysis

CALCIUM IMBALANCES (PRIMARY AND SECONDARY)

CONDITION	CAUSE	CLINICAL MANIFESTATIONS
Hypocalcemia	Decreased intake (1^0), vit. D deficiency, hypoparathyroid; hypoalbuminemia; alcohol abuse or liver failure	Increased neuromuscular activity (possible convulsions); skeletal muscle tetany;
Hypercalcemia	Increased intake (1^0); immobility; hyperparathyroidism; bone malignancies; renal failure	Decreased neuromuscular activity (stupor to coma); renal calculi; increased fracture risk

DISCUSSION QUESTIONS: (Post answers to the "Patho Discussion Group")

1. Explain why hyponatremia could cause cerebral edema.

2. How could it be treated in order to get the most rapid results?