

# LECTURE 2

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## FLUID IMBALANCES

### Review of Lecture 1

In Lecture 1, we considered the three basic fluid compartments of the body and ways their components can be interchanged among the three compartments. These fluid compartments are interchangeable to greater or lesser degrees depending upon the permeability nature of the partition(s) that separate them (e.g.- capillary wall or cell membrane). Physical or chemical changes in one compartment can sometimes appear in the other two. In order to understand this principle, we must pay close attention to the barriers that separate these three fluid compartments as well as the mechanisms by which fluids and small dissolved materials cross these partitions. For example, water moves along osmotic gradients and can easily cross both the cell membrane and capillary wall. Thus, changes in osmolarity in one compartment can cause water to shift into or out of the other interconnected compartments. In contrast, albumin protein is too large to cross either capillary pores or cell membranes so that it remains confined to the intravascular plasma fluid compartment. However, being osmotically active, changes in albumin concentration can change the osmolarity of blood plasma and cause fluid shifts into or out of this and other compartments.

### REVIEW QUESTIONS:

1. Where is albumin produced in a healthy human?
2. Why is isotonic saline frequently used to replace lost blood volume if usable donor blood is not available? Why not use water, which is much more economical?
3. Sodium is osmotically active. Considering the location of most sodium in the body, describe the fluid shifts that would result if sodium concentration were to increase or decrease.
4. How would the situation above change if potassium were the ion changing in concentration?

### Fluid Imbalances: Hyper- and Hypovolemia

In a normal human, fluid balance is achieved primarily by balancing fluid intake with output. Considering the ingestion of food and beverages, the **average water intake is 2000 ml/day**. This means that average output should also be 2000ml. They must be equal for normal fluid balance. If output is more than intake, hypovolemia and possible dehydration results. If input is greater than output, hypervolemia with possible edema results. So normal water balance is going to require about 2 L/day turnover. What are the mechanisms of control over this normal fluid balance? The hormones **ADH and Aldosterone** are the major controllers of water/fluid balance.

### REVIEW QUESTIONS:

1. What is the mechanism of ADH release?
2. How does ADH affect water balance via the kidneys?
3. What is the mechanism of aldosterone release?
4. How does aldosterone (a cortical steroid hormone) affect fluid balance via the kidneys?
5. Which hormone above affects the renal output of water only?
6. What is diabetes insipidus?
7. How might head trauma, alcohol, and general anesthesia impact water/fluid balance?

Normal water and solute concentrations in body fluids is referred to as an isotonic/isosmotic state. In this normal situation, fluids and their dissolved solutes are balanced among the fluid compartments and water does not shift significantly from one to another. However, if water is selectively lost from a fluid compartment (usually blood), this tends to increase the concentration of solutes remaining and to cause water from other compartments to shift into the vascular compartment while stimulating thirst as a corrective measure to replace lost water. Lost water lowers blood volume and pressure to decrease renal output via low glomerular filtration and increased ADH activity, both of which act to conserve fluids. Thus, in dehydration, the need is to both acquire and retain water. You acquire it by drinking it, and you retain it by not urinating it. Conversely, if fluid balance is shifted toward the excess side, thirst should decrease and urine output should increase until normal isosmotic balance is again restored. What would be the role of ADH in hypervolemia? Could it cause it? Could it correct it? Explain!?

We will be considering clients that have hyper- or hypovolemia problems. Hypervolemia simply means too much fluid volume, not always just too much water. If it should occur as a result of too much water, ADH mechanisms serve to correct it. However, if just the volume is expanded and there is no disruption of water concentration, the correction mechanism will differ. Basically, the expanded volume increases blood pressure and results in increased glomerular filtration in nephrons of the kidney causing increased urine output until the volume imbalance is corrected. However, in this instance, the increased urine volume being eliminated contains less water per unit of volume than would be expected if ADH were the controlling agent.

How would the above situation change if hypovolemia were the case? What differences would be seen in ADH activity?

### REVIEW QUESTIONS:

1. What are diuretics and their general mechanisms of action?
2. Why might diuretics be given to a hypertensive individual?
3. Why are diabetics always thirsty and experience polyuria?
4. Compare and contrast diabetes mellitus and diabetes insipidus.

Read the Textbook section on fluid balance (Chapter 10 through page 199) and examine the Table below. From the table, with both hyper- and hypovolemia, take each “cause” and each “clinical manifestation” and be sure you can explain the underlying physiology associated with each situation mentioned in the table. The example following the table below illustrates the approach you should take as you work through the table entries.

### WATER IMBALANCES

CONDITION	CAUSE	CLINICAL MANIFESTATIONS
Hypovolemia	Extracellular and/or intracellular fluid loss due to: diminished intake; diabetes mellitus/insipidus; burns or wound draining; diaphoresis; diarrhea or vomiting.	Weight loss; hypotension; tachycardia; thirst; skin tenting; <u>increases in</u> : hematocrit, temperature without infection, serum albumin, BUN, electrolytes, and creatinine; increased urine concentration
Hypervolemia	Extracellular fluid excess due to: increased intake/iatrogenic; renal failure; liver failure	Weight gain; hypertension, bradycardia; edema; <u>decreased</u> hematocrit and other hematology lab values

What might cause a hypervolemic state? Excessive I.V. administration of isotonic solution serves as a good clinical application.

Does this disrupt the fluid and electrolyte balance?

Well, yes and no! Since you gave “excess” fluid via I.V., the fluid volume would be expanded yielding a hypervolemic fluid imbalance. However, since the fluid given was isotonic (same solute concentration as blood and body fluids), electrolytes were given in the same proportion as they occur in normal blood. So, the actual concentration of electrolytes would remain unchanged. Essentially, the patient experiences an isotonic hypervolemia in which plasma volume increases while plasma composition remains unchanged. **NOTICE!** Only plasma composition remains unchanged. If whole blood composition is considered, a drop in hematocrit would be seen initially because the I.V. contained only saline and no formed blood elements. Thus, blood would be diluted and exhibit a lowered hematocrit.

What signs and symptoms might be seen in this situation and why?

- Systemic hypertension as a result of expanded blood volume.
- “Bounding” pulse due to increased stroke volume from the hypervolemic increase in venous return.
- Bradycardia as the baroreceptor reflex attempts to slow the heart to lower pressure.
- Rapid weight gain since 60-80% of body weight is fluid weight and excess fluids were just administered.
- Lowered hematocrit since saline was given instead of whole blood.
- Increased renal output due to the hypertensive increase in glomerular filtration.
- Peripheral edema since capillary filtration exceeds reabsorption in hypertensive situations.
- In people with poor cardiovascular compliance, moist “rales” would be heard upon chest auscultation from the resultant pulmonary hypertension and pulmonary edema.

Notice the approach taken in the above example! Both the causes and the clinical manifestations were not just listed, but were explained in the context of the underlying physiological disruptions associated with the disorder. **This is the approach you want to take as you work through the table. Avoid trying to memorize situations!!** There are too many in this course. Practice thinking through the underlying physiological reasons at every opportunity.

### **DISCUSSION QUESTIONS: (Post answers to the “Patho Discussion Group”)**

1. What is hypovolemic “shock” and explain the signs and symptoms by the procedure used in the previous example?

2. Compare and contrast “hemorrhagic” and “anaphylactic” shock.