Study guide for volume regulation

Lect. 1. Intro , body fluid compartments

1. Intracellular and extracellular spaces
2. ECF = interstitial + plasma compartments
3. Fluid distribution between ICF, plasma & interstitium
4. Losses from/gains to the body are via the ECF
5. Key solutes within each compartment determine that compartment's osmolality, and osmolality determines if and to what extent fluid will shift between compartments
6. Osmolality and the Gibbs-Donnan equilibrium—role of proteins
7. Starling forces—hydrostatic & oncotic pressures and edema; role of lymphatics
8. Regulation of interstitial vs plasma compartment volumes—short-term vs long-term regulation
9. ECF osmolality—regulated through control of water retention vs absorption (ADH)
10. ECF volume—regulated through control of sodium retention vs absorption (physical factors/renin-angiotensin-aldosterone (RAA) system, ANP

What is the major stimulus for activation of physiological mechanisms whose role it is to regulate ECF volume (i.e. changes in what parameter are being sensed)? Are these mechanisms active over the short-term, long-term, or both?

What does the effective circulating volume (ECV) refer to? Which of the three body fluid compartments contains the ECV?

Under normal conditions, do changes in the environment have a direct or indirect effect on fluid composition/volume inside cells? If indirect, how are such changes mediated?

What are the sources of ECF fluid loss? Gain? With regard to fluid loss, which sources of loss are "automatic" (i.e. uncontrolled) and which ones are under physiological control?

What are the general signs (i.e. symptoms) of changing volume in the two sub-compartments which make up the ECF? Which sign suggests a change in one sub-compartment vs the other?

What is osmosis? What causes it? What is a solute? Which ones predominate in the ECF/ICF?

What is the difference between osmotic pressure and oncotic pressure? Which compartments should we be thinking about when we're talking about these respective two parameters? What is the primary determinant of osmotic pressure? Which one determines oncotic pressure?

What are Starling forces? What causes them? Which one favors filtration (i.e. movement of fluid from the vascular space to the interstitial space), and which one favors absorption (i.e. the reverse of filtration)? How do each of these Starling forces act the way they do?

Use your understanding of Starling forces to explain why edema may result from heart failure, or liver failure.

How do the lymphatics prevent edema from occurring all the time in the normal state?

How is ECF osmolality regulated? If this regulation is disrupted, what could be the consequences for the intracellular space?

How is ECF volume regulated?

Lect 2. Renal handling of sodium and water

1. Sodium as the major determinant of ECF volume; kidney is major site of physiological control of ECF sodium.
2. Nephron as functional unit of the kidney
3. Nephron functional anatomy—tubular system and associated blood vessels; general sites of sodium absorption within the nephron
4. Glomerular function—bulk filtration from glomerular capillaries
5. Tubular function—selective reabsorption from peritubular capillaries
6. Proximal tubule—site of majority of sodium/water reabsorption (60-70%)—importance of basolateral Na^+-K^+ ATPase pump
7. Glomerular-tubular balance—GFR directly correlates with reabsorption
8. Loop of Henle—accounts for 25% of sodium/water reabsorption
9. Distal tubule & collecting duct—sites of hormonal control
10. Principal & intercalated epithelial cells
11. Juxtaglomerular apparatus/juxtaglomerular feedback—local constrictive vs dilatory effects on afferent/efferent arterioles & systemic effects via renin-angiotensin-aldosterone system
12. Effects of aldosterone on sodium vs potassium
13. Water balance: intake = output; this matching achieved via sensing of $[Na^+]_{ECF}$ (ECF osmolality)
14. Hypothalamic osmoreceptors/ADH release/ADH stimulated water reabsorption in medullary collecting duct
15. Loop of Henle & Vasa recta: countercurrent multiplication & exchange

How is a stable ECF volume maintained?

What is unique about the routing of blood through the renal vascular system?

Where in the nephron is the blood filtered? What is the major driving force for this filtration process? What is filtered into the nephron, what is left behind in the blood?

After leaving the glomerular capillaries where does the blood go next, and what is the net process occurring there? (hint: use your understanding of the Starling forces when thinking about this)

Glucose is freely-filtered at the glomerulus, yet normally is never present in the urine. What has happened to all that glucose that was filtered?

If more potassium is seen to be in the urine than was filtered by the glomerulus, then how did it get there? What is going on with that potassium?

What is the general relationship between the amount of a substance that is filtered, the amount reabsorbed, the amount secreted, and the amount excreted?

In the proximal tubule, what are the two cellular mechanisms whereby sodium is reabsorbed? What is the key protein that drives these mechanisms?

What is glomerular-tubular balance? How is it achieved? In which capillary bed (glomerular vs peritubular) is the hydrostatic pressure higher? Why is it higher? How 'bout oncotic pressure? How do those differences explain the net processes (filtration vs absorption) occuring in those two vascular regions?

Under normal conditions, the loop of Henle reabsorbs roughly 25% of reabsorbed water and sodium. Does this normally change , or does it tend to remain constant? If it changed, name one key feature of normal kidney function that could be adversely affected (hint: think about what allows us to survive in a terrestrial environment where drinking water might be occasionally scarce).

What cells in the distal tubular system (distal convoluted tubule & cortical collecting duct) are mainly concerned with sodium reabsorption? What hormone affects this process, and what is its effect on potassium?

What is the relationship between juxtaglomerular feedback and aldosterone? What is renin? What stimulates it, where is it released, and what does it do?

Besides directly stimulating sodium reabsorption in the proximal tubule and aldosterone release from the adrenal gland, what other significant effect does angiotensin II have?

If aldosterone levels are high, what are the effects on sodium and potassium?