Hypertension (HT)

1. Definition—based on measured systolic/diastolic pressures
2. Incidence: roughly one third of the U.S. population
3. Classified into two broad categories: essential (primary) & secondary
4. Primary HT—most common in the population, but etiology not clearly understood in all cases
5. Secondary HT—symptom of another disease, e.g. renal dysfunction
6. Basic issue is increases in CO and/or TPR due to renal, hormonal, neurogenic factors (possibly hereditary), and dietary sodium intake—variables affected are CO (via volume) and peripheral vascular resistance
7. Sustained HT requires increased blood volume—primacy of kidneys
8. Deleterious effect of HT on various organs: heart, kidneys, circulatory system
9. Fundamental renal-body fluid system for long-term control of arterial pressure—pressure diuresis/natriuresis
10. Graphical analysis of sodium intake/output matching; "equilibrium point"
11. Two primary determinants of pressure regulation over the long-term: 1) direction/extent of shifting of renal output curve, 2) level of sodium/water intake
12. BP = CO * TPR, but only ΔCO can influence equilibrium point
13. If TPR increases, but renal fxn stays normal, then CO will decrease to compensate and BP will remain constant
14. But if specifically RENAL resistance changes, that will affect volume via a rightward shift in the renal output curve
15. In most cases, increase in TPR are secondary to volume-induced increases in CO
16. Some experimental results showing how volume both initiates HT and also indirectly causes persistant increase in TPR
17. RAA system acts to fine-tune pressure diuresis/natriuresis, especially in the face of variations in salt intake
18. RAA dysfunction can cause HT by shifting the renal output curve

What is required for sustained hypertension?

Why does a simple increase in total peripheral resistance (TPR) alone not normally produce an increase in blood pressure for more than just a day or two?

Define pressure diuresis/natriuresis.

What the renal output (aka renal function) curve repreent?

What are the two major determinants in the renal-body fluid control of arterial blood pressure?

Sometimes, the immune response to bacterial infections results in large, nasty globs of bacterial-antibody complexes getting stuck in the glomerular capillary beds, thus effectively plugging the fenestrations (pores) through which water and solutes are filtered. What would you predict would happen to the renal output curve as a result, and what would that imply about the kidney's ability to match salt & water output to their intake?

What is the "equilibrium point?" What would renal disease do to that point? What would an increase in total peripheral resistance do to that point? What would a constriction to the afferent arteriole do? What would an increase in salt intake do?

If there is an increase in total peripheral resistance, but no change in renal vascular resistance, what would happen to the equilibrium point?

How does an increase in cardiac output (due to say, and increase in total circulating volume), lead to an increase in total peripheral resistance?

Why is an increase in salt (Na^+) intake more likely to elevate the arterial pressure than an increase in water intake? IOW, why does salt increase ECF volume far more than does water?

In the experiment on volume-loading hypertension we discussed in lecture (slide #16) why did arterial pressure rise upon infusion of 0.9% saline? (Hint: think of the two determinants of long-term pressure regulation). Was the equilibrium point in the renal output curve changing in this experiment?

Experiments have shown that direct injection of norepinephrine into the renal arteries can cause permanent damage to the kidneys. How might that affect the renal function curve, and what implications might that have for persons who regularly undergo extreme stress? (Recall that stress causes increases n sympathetic activity) Would you expect such persons to be prone, or not prone to hypertension?

On the graph in slide #22 three curves showing renal output are shown—one for "normal" subjects and the other two for subjects with either salt-sensitive or salt-insenitive essential hypertension. In the normal subjects, increasing to a high intake of salt shifts the equilibrium point (E) to a pressure level only very slightly past the normal point (D). Why do you think this is? (Hint: compare against what is likely occurring in the essential hypertension curves (points A & B) to the right)

What appears to be the major role of the renin-angiotensin-aldosterone system in the overall control of ECF volume and arterial blood pressure?

What are the direct and indirect effects of angiotensin II in regards to renal handling of salt and water?