

MEDICAL PHYSIOLOGY

Renal Conference - Quiz 7A

February 13, 2001

1. Diuretics such as furosemide are often given to increase salt and water excretion and thereby reduce blood pressure. Why wasn't furosemide prescribed for this patient?

Furosemide inhibits K reabsorption and indirectly stimulates K secretion which would only aggravate the patient's hypokalemia.

2. This patient was hypertensive. Did the low level of renin in the blood contribute to the hypertension? Explain.

No, rather the hypertension contributed to the low level of renin. High blood pressure and ECF volume expansion inhibit renin secretion.

3. How does the Na flux through the ENaC channel affect potassium secretion?

The increase in cell concentration of Na stimulates Na-K-ATPase and thus increases K entry into the cell across the basolateral membrane raising the chemical gradient that drives K movement from the cell into the tubular fluid. The increased Na flux through the ENaC channel also depolarizes the apical membrane, reducing the electrical gradient opposing the K flux from the cell to the lumen.

4. A rise in blood volume acting through atrial volume receptors will DECREASE sympathetic nerve activity to the kidney resulting in a FALL in salt reabsorption by the tubules.

5. The patient exhibited abnormalities in the EKG including a flattened T wave. What caused this flattening?

The hypokalemia caused the gK of ventricular muscle to fall. This slows the repolarization of ventricular cells which is brought about by K efflux from the cells. The slower repolarization causes the T wave to flatten.

MEDICAL PHYSIOLOGY

Renal Conference - Quiz 7B

February 15, 2001

1. What does spironolactone do and why didn't it reduce blood pressure in this patient?

Spironolactone competes with aldosterone for attachment to mineralocorticoid receptors in principal cells and blocks the effect of aldosterone. In a patient with hyperaldosteronism, the drug would increase salt and water excretion and thus lower blood pressure. It did not work in this patient because her aldosterone level was so low there was no effect to block.

2. To what extent is angiotensin II contributing to the hypertension in this patient? Explain.

Angiotensin II levels were probably very low because renin secretion was inhibited by the expansion of the ECF vol. Thus enalapril, an ACE inhibitor had little effect on blood pressure. In other words the hypertension contributed to low AII levels.

3. How does the magnitude of the Na flux through the ENaC channel regulate Na-K-ATPase?

An increase in the flux of Na through the ENaC channel increases the Na concentration in the cell and this is a powerful stimulant of the Na-K-ATPase.

4. The patient in this conference exhibited a low concentration of aldosterone in the blood. Was this a factor contributing to the hypokalemia? Explain.

Aldosterone stimulates K secretion, thus a low concentration of aldosterone should reduce K excretion and compensate to some extent for the hypokalemia. Actually the hypokalemia contributes to the inhibition of aldosterone secretion in this patient.

5. Why was amiloride an effective treatment for the hypertension and the hypokalemia?

Amiloride directly blocks the ENaC channel which is the source of all the patient's problems. By blocking the channel, Na reabsorption and K secretion are reduced; ECF vol falls reducing the hypertension and K is retained repairing the hypokalemia