Pressure Natriuresis

Short-term (seconds to minutes) control of blood pressure is largely regulated by the nervous system. This finely tunes very rapid changes in blood pressure, like when an individual stands up. However, long-term (days to years) control of blood pressure is closely related to the regulation of body fluid volume. Extracellular fluid volume is a balance between the intake and output of sodium and water. This is primarily controlled by hormonal and neural systems that affect the kidney as well as intrinsic mechanisms within the kidney itself. The hormonal and neural segments of blood pressure control will be discussed other labs.

The intrinsic mechanisms within the kidney that play an important role in extracellular fluid volume (blood volume) are called pressure diuresis and pressure natriuresis. Pressure diuresis is the effect of an increase in arterial pressure to increase excretion of fluid. The same increase in arterial pressure also dramatically increases excretion of sodium (pressure natriuresis). So if an individual increases their salt and water intake dramatically, this would increase extracellular fluid volume and blood volume, which increases mean circulatory filling pressure and arterial pressure. However, even slight increases in arterial pressure can robustly increase the excretion of sodium and fluid, causing a decrease of the extracellular fluid volume, blood volume, and arterial pressure back to normal.

Protocol 1:

1. Run for 10 minutes to establish a baseline.
2. Fix left and right afferent arteriolar conductances at normal levels. Increase renal perfusion pressure from 90 to 100 mmHg.
3. Run for 30 minutes to observe changes in renal excretion of sodium and water. Run for 6 hours to see an effect on plasma volume, arterial pressure, and urine output.

Questions:

1. What happened to urine output of sodium and fluid? Under normal physiological conditions, why doesn’t a slight increase of arterial pressure (~10 mmHg) normally have profound increases in urine output?
2. As in the simulation, predict what would happen to arterial pressure if pressure natriuresis kept increasing urine output.

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Protocol 2:

1. Run for 10 minutes to establish a baseline.
2. Give your patient an extreme renal artery stenosis in each kidney.
3. Run for 6 hours to observe effects on renal excretion, blood volume, and arterial pressure.

Questions:

1. What happened to the excretion of water and sodium? What other factors may be playing a role besides pressure natriuresis? What happened to angiotensin II and aldosterone concentrations?
2. If you restart the simulation and run for 24 hours, what happens to urine output and why?
3. What happened to total sodium mass (total amount of sodium in the body)? After stenosis, and impairing the ability to excrete sodium, why doesn’t the concentration of sodium increase?