Hyponatremia Review Questions – Handout

I. ADH Physiology & Mechanisms

1. What is the primary trigger for ADH release in primary adrenal insufficiency?

2. Which medications can bind to the V2 receptor (normally activated by ADH)?

3. Where is ADH synthesized? (Hint: paraventricular neurons of the hypothalamus as pre-pro-AVP)

4. Where is ADH stored before release? (Hint: posterior pituitary)

5. Once ADH binds to its receptor and increases AQP channels, what must be present for water reabsorption to occur? (Hint: osmotic gradient)

6. Name non-osmotic stimuli for ADH release.

7. Why can't we routinely measure ADH in clinical practice, and which molecule serves as its surrogate?

8. What is the major determinant of plasma osmolality?

II. Diuretics & Hyponatremia

1. Which diuretic is more likely to cause hyponatremia — loop or thiazide?

2. Which diuretic can be used to treat hyponatremia — loop or thiazide?

3. True or False: Thiazide-induced hyponatremia is only due to volume depletion.

4. Can ESRD or anephric patients develop hyponatremia? If so, how?

III. Electrolytes & Endocrine

1. Does hyperkalemia occur with primary or secondary adrenal insufficiency, and why?

2. True or False: A single mechanism explains hyponatremia in primary adrenal insufficiency.

IV. IV Fluids & Sodium Content

1. How much NaCl ("salt") is in 1 liter of 0.9% normal saline?

2. How much sodium (Na) is in 1 liter of 0.9% normal saline?

V. SIADH & Pharmacologic Therapy

1. What is the most important treatment for SIADH? (Give specific fluid restriction target)

2. How does tolvaptan treat hyponatremia?

3. In which situations should tolvaptan not be given?

4. True or False: It is safe to administer oral salt tablets to patients with peripheral, pulmonary, or both types of edema.