

HOSPITAL PHYSICIAN®

EMERGENCY MEDICINE BOARD REVIEW MANUAL

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The *Hospital Physician Emergency Medicine Board Review Manual* is a peer-reviewed study guide for residents and practicing physicians preparing for board examinations in emergency medicine. Each quarterly manual reviews a topic essential to the current practice of emergency medicine.

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Management of Electrolyte Emergencies

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Management of Electrolyte Emergencies

N. Ewen Wang, MD

INTRODUCTION

Electrolyte panels are frequently ordered and often show results outside of normal ranges. Although most electrolyte abnormalities do not require specific treatment, some are emergent. Emergency medicine physicians should be familiar with common electrolyte imbalances as well as when and how to manage them.

DISORDERS OF SODIUM IMBALANCE

HOMEOSTASIS OF BODY WATER AND SODIUM

The kidneys have evolved to conserve water and salt in order to maintain a “private ocean” bathing the body cells. As the most abundant extracellular cation, plasma sodium is the major determinant of osmotic forces in the extracellular fluid (ECF). Thus, sodium regulation must be considered in conjunction with body water regulation. Antidiuretic hormone (ADH) and aldosterone enable the kidney to conserve water by concentrating urine. The healthy kidney is also able to excrete large volumes of excess water in order to maintain a constant plasma osmolality despite dietary variations.

ADH is the main regulator of water homeostasis. ADH enhances water permeability in the kidney’s collecting duct, increasing water reabsorption. ADH is secreted in response to hypovolemia and high plasma osmolality. ADH and aldosterone will maintain intravascular volume, even at the expense of electrolyte balance. The renin-angiotensin system is the main regulator of sodium homeostasis. Renin is produced in the kidney in response to decreased intravascular volume and via angiotensin stimulates adrenal production of aldosterone. Aldosterone increases sodium resorption and potassium excretion by the kidney. Hypothalamic cells regulate thirst in response to hyperosmolality and body fluid volume deficit.

Because of the complex interrelationship between sodium and water homeostasis, sodium disturbances are linked to water imbalances. Changes in total body sodium and water are usually proportionate and do not

cause either hyponatremia or hypernatremia.¹ Sodium imbalances require severe and disproportionate loss or gain of total body sodium or total body water (TBW).

Symptoms of hypernatremia and hyponatremia result primarily from compartmental fluid shifts. Both disorders cause similar pictures of altered level of consciousness, coma, and seizures. The severity of the symptoms depends on the rapidity and the degree of the imbalance. Patients at the extremes of age have more severe symptoms at any given sodium level.

HYPONATREMIA

Hyponatremia, defined as a plasma sodium concentration less than 130 mEq/L, is the most common electrolyte disturbance seen in the hospital population.² Although most patients with hyponatremia are stable and do not require emergent therapy, acute, severe hyponatremia and its treatment can result in serious morbidity or death. Hyponatremia can cause cerebral edema secondary to the movement of water from the hypotonic extracellular space into the intracellular space, resulting in increased intracerebral pressure and decreased cerebral blood flow. Hyponatremia is a leading cause of afebrile seizures in infants.³

Etiology

Low or high plasma sodium concentration can occur in different states of hydration, depending on the ratio of TBW to total body sodium.

Factitious hyponatremia. Low or high plasma sodium concentration can be the result of how plasma sodium is measured. Pseudohyponatremia (isotonic hyponatremia; plasma osmolality, 280–295) can be due to a blood draw error or an excess of a nonosmotic substance in the ECF (eg, hyperlipidemia, hyperproteinemia). Errors occur because some laboratory techniques for measuring sodium concentration consider the entire plasma volume as plasma water, resulting in a false increase in the ECF volume.

Redistributive hyponatremia occurs when there is an increase of osmotic particles in the ECF (hypertonic hyponatremia; plasma osmolality > 295). This form of hyponatremia can occur with hyperglycemia or when hyperosmolar substances, such as mannitol, are administered.