12

ELECTROLYTES, OTHER MINERALS, AND TRACE ELEMENTS

Lingtak-Neander Chan

OBJECTIVES

After completing this chapter, the reader should be able to

- Describe the homeostatic mechanisms involved in sodium and water balance, hyponatremia, and hypernatremia
- Describe the physiology of intracellular and extracellular potassium regulation as well as the signs and symptoms of hypokalemia and hyperkalemia
- List common causes of serum chloride abnormalities
- List common conditions resulting in serum magnesium abnormalities and describe signs and symptoms of hypomagnesemia and hypermagnesemia
- Describe the metabolic and physiologic relationships among the metabolism of calcium, phosphate, parathyroid hormone, and vitamin D
- List common conditions resulting in serum calcium abnormalities and describe signs and symptoms of hypocalcemia and hypercalcemia
- List common conditions resulting in altered copper, zinc, manganese, and chromium homeostasis and describe the signs and symptoms associated with their clinical deficiencies
- Interpret the results of laboratory tests used to assess sodium, potassium, chloride, calcium, phosphate, magnesium, copper, zinc, manganese, and chromium (in the context of a clinical case description, including history and physical examination)

Serum or plasma electrolyte concentrations are among the most commonly used laboratory tests by clinicians for assessment of a patient's health status, clinical conditions and disease progression. The purpose of this chapter is to present the physiological basis of the need to assess serum concentrations of common electrolytes and minerals. The interpretation of these laboratory results and the clinical significance of abnormal results are addressed.

Serum sodium, potassium, chloride, and total carbon dioxide content (often referred to *as serum bicarbonate concentration*) are among the most commonly monitored electrolytes in clinical practice. Magnesium, calcium, and phosphate are also monitored as determined by the patient's disease states and clinical indication. The homeostasis of calcium and phosphate is frequently discussed in the context of the endocrine system because of the effects of vitamin D and parathyroid hormone (PTH) on the regulation of these minerals. Serum total carbon dioxide content, often measured in conjunction with electrolytes, is discussed in Chapter 13 because of its significance for the assessment of acid–base status. Listed in **Table 12-1** are the current dietary reference intake for electrolytes, minerals, and trace elements.

TABLE 12-1. Recommended Dietary Reference Intake of Electrolytes and Minerals for Healthy Adults According to the Dietary Guidelines 2015–2020

NUTRIENT	DIETARY REFERENCE INTAKE[a]	
	Male	Female
Sodium	2300 mg (100 mEq)	
Potassium	4700 mg (~120 mEq)	
Chloride	Varies with potassium and sodium intakes	
Magnesium	19–30 yo: 400 mg	19–30 yo: 310 mg
	31+ yo: 420 mg	31+ yo: 320 mg
Calcium	19–70 yo: 1000 mg	19–50 yo: 1000 mg
	71+ yo: 1200 mg	51+ yo: 1200 mg
Phosphorus	700 mg	
Copper	900 mcg	
Zinc	11 mg	8 mg
Manganese	2.3 mg	1.8 mg
Chromium[b]	19–50 yo: 35 mcg	19–50 yo: 25 mcg
	51+ yo: 30 mcg	51+ yo: 20 mcg

yo = years old.

[a]According to the Recommendations from 2015–2020 Dietary Guidelines for Americans. http://health.gov/dietaryguidelines/2015/ (accessed 2016 Feb 8).

[b]Adequate intakes according to Institute of Medicine (U.S.) Food and Nutrition Board. Dietary Reference Intakes. Washington, DC: National Academies Press (U.S.); 2001.

ELECTROLYTES

The traditional units, International System (SI) units, and their conversion factors for electrolytes, minerals, and trace elements discussed in this chapter are listed in **Table 12-2**. Although the normal ranges of serum concentrations for each of the electrolytes are listed below, clinicians should always confirm with the institutional clinical laboratory department for their institutional reference range due to the variance introduced by equipment, analytical technique, and quality assurance data.

Sodium

Normal range: 135–145 mEq/L (135–145 mmol/L)

Sodium is the most abundant cation in the extracellular fluid and is the major regulating factor for bodily fluid and water balance. Extracellular (i.e., intravascular and interstitial) and intracellular sodium contents are closely affected by body fluid status. Thus, an accurate interpretation of serum sodium concentration must include an understanding of body water homeostasis and the interrelationship between the regulation of sodium and water.[1]

Physiology

Sodium is essential for maintaining the optimal transmembrane electric potential for action potential and neuromuscular functioning as well as regulating serum osmolality and water balance. Serum osmolality is an estimate of the water-solute ratio in the vascular fluid. It can be measured in the laboratory or estimated using the following equation:

$$\begin{aligned}&\text{Estimated serum osmolality (mOsm/kg)}\\&= (2 \times \text{serum [Na]}) + [\text{glucose, in mg/dL}]/18\\&\quad + [\text{blood urea nitrogen, in mg/dL}]/2.8\end{aligned}$$

The normal range of serum osmolality is 285–295 mOsm/kg. The measured osmolality should not exceed the estimated value by more than 10 mOsm/kg. A difference of 10 mOsm/kg or more is considered an increased osmolal gap, which may suggest the presence of other unmeasured solutes (e.g., organic solvents, alcohol) and is useful to providing assessments in clinical toxicology. Decreased serum osmolality usually suggests a water excess, whereas increased serum osmolality suggests a water deficit. Although serum osmolality may be helpful in assessing water status, especially the intravascular volume, it should not be the primary and only parameter in assessing fluid status. The results also should be interpreted in the context of the ability of the solute to cross cellular membranes (e.g., uremia causing hyperosmolality without relative intracellular depletion) and the patient's symptoms and signs of disease. **Figure 12-1** summarizes the inter-relationship and regulation between water and sodium.

Changes in body water and plasma volume can directly or indirectly affect the serum sodium concentration. For example, as the result of changes in effective circulating volume, baroreceptors and osmoreceptors will respond accordingly to restore an isovolemic state of the body. Baroreceptors are located in the carotid sinus, aortic arch, cardiac atria, hypothalamus, and the juxtaglomerular apparatus in the kidney. Stimulation of these receptors will promote urinary loss of water and sodium. Osmoreceptors are present primarily in the hypothalamus. The three major effectors in response to the stimulation of the osmoreceptors include vasopressin or antidiuretic hormone (ADH), the renin-angiotensin-aldosterone system, and natriuretic peptides. The resultant renal effects from these three distinct pathways collectively regulate the homeostasis of water and sodium.

The kidneys are the primary organ responsible for the retention and excretion of body sodium and water. The glomeruli receive and filter about 180 L of plasma fluid daily. On average, fewer than 2 L of water and between 0.1–40 g of sodium are excreted in the urine, depending on the fluid status of the individual. Although almost 100% of the plasma sodium is filtered by the glomeruli, <1% is excreted in the urine under normal circumstances. The proximal tubule and the loop of Henle collectively account for up to 90% of sodium reabsorbed from the kidneys.

The homeostatic mechanism for water and sodium also involves the equilibrium among intravascular, interstitial, and intracellular fluids.[3] Net movement of water occurs from areas of low osmolality to areas of high osmolality. This effect can be readily observed in patients with a low serum osmolality due to a deficit of serum sodium or excess of plasma water. In patients with hyponatremia, water moves from the plasma to the higher osmolality in the interstitial space.[3] In the presence of high hydrostatic and oncotic pressure gaps across capillary walls, the net effect is excessive interstitial water accumulation and edema formation.[2,3]

Antidiuretic hormone (vasopressin). ADH, also known as *arginine vasopressin*, is a nine amino acid peptide hormone that regulates renal handling of free water. By altering the amount of water reabsorbed by the kidney, ADH has an indirect but pivotal effect in changing or maintaining serum sodium concentration. ADH is secreted by the magnocellular neurons in the supraoptic and paraventricular nuclei of the hypothalamus, where both osmoreceptors and baroreceptors are present to detect fluid changes in the vasculature.

TABLE 12-2. Conversion Factors to SI Units

NUTRIENT	TRADITIONAL UNITS	CONVERSION FACTORS TO SI UNITS	SI UNITS
Sodium	mEq/L	1	mmol/L
Potassium	mEq/L	1	mmol/L
Chloride	mEq/L	1	mmol/L
Magnesium	mEq/L	0.5	mmol/L
Calcium	mg/dL	0.25	mmol/L
Phosphate	mg/dL	0.3229	mmol/L
Copper	mcg/dL	0.1574	µmol/L
Zinc	mcg/dL	0.153	µmol/L
Manganese	mcg/L	18.2	nmol/L
Chromium	mcg/L	19.2	nmol/L