MEDICAL PROCEDURE: ELECTROLYTES

SODIUM

Normally, serum sodium levels range from 135 to 145 mEq/L (SI, 135 to 145 mmol/L)

The serum sodium test measures serum levels of sodium, the major extra-cellular cation. Sodium affects body water distribution, maintains osmotic pressure of extracellular fluid, and helps promote neuromuscular function; it also helps maintain acid-base balance and influences chloride and potassium levels. Sodium is absorbed by the kidneys; a small amount is lost through the skin.

Hypernatremia	**Hyponatremia**
Causes	**Causes**
Hypernatremia with hypovolemia (decreased TBW and Na; relatively greater decrease in TBW)	**Hyponatremia with hypovolemia** (decreased TBW and Na; relatively greater decrease in Na)
Extrarenal losses	Extrarenal losses
GI: Vomiting, diarrhea	GI: Vomiting, diarrhea
Skin: Burns, excessive sweating	Third-space losses: Pancreatitis, peritonitis, small-bowel obstruction, rhabdomyolysis, burns
Renal losses	Renal losses
Intrinsic renal disease	Diuretics
Loop diuretics	Mineralocorticoid deficiency
Osmotic diuresis	Osmotic diuresis
(glucose, urea, mannitol)	(glucose, urea, mannitol)
Hypernatremia with euvolemia (decreased TBW; near-normal total body Na)	Salt-losing nephropathies
Extrarenal losses	**Hyponatremia with euvolemia** (increased TBW; near-normal total body Na)
Respiratory: Tachypnea	Diuretics
Skin: Fever, excessive sweating	Glucocorticoid deficiency
Renal losses	Hypothyroidism
Central diabetes insipidus	Primary polydipsia
Nephrogenic diabetes insipidus	States that increase release of ADH (postoperative opioids, pain, emotional stress)
Other	Syndrome of inappropriate ADH secretion
Inability to access water	**Hyponatremia with hypervolemia** (increased total body Na; relatively greater increase in TBW)
Primary hypodipsia	Extrarenal disorders
Reset osmostat	Cirrhosis
Hypernatremia with hypervolemia (increased Na; normal or increased TBW)	Heart failure
Hypertonic fluid administration (hypertonic saline, $NaHCO_3$, total parenteral nutrition)	Renal disorders
Mineralocorticoid excess	Acute renal failure
Adrenal tumors secreting deoxycorticosterone	Chronic renal failure
Congenital adrenal hyperplasia (caused by 11-hydroxylase defect)	Nephrotic syndrome
Iatrogenic	
TBW = total body water.	

Serum sodium levels are evaluated in relation to the amount of water in the body, which is affected by the cellular mechanics of sodium (decreased sodium levels promote water excretion, and increased levels promote retention). For example, hyponatremia refers to a decreased level of sodium in relation to the body's water level.

Sodium imbalance can result from a loss or gain of sodium or from a change in water volume.

Sodium test interference

- Most diuretics, lithium, chlorpropamide, and vasopressin suppress serum sodium levels.
- Corticosteroids and antihypertensives (such as methyldopa, hydralazine, and reserpine) elevate serum sodium levels

POTASSIUM

Normally, serum potassium levels range from 3.5 to 5 mEq/L (SI, 3.5 to 5 mmol/L).

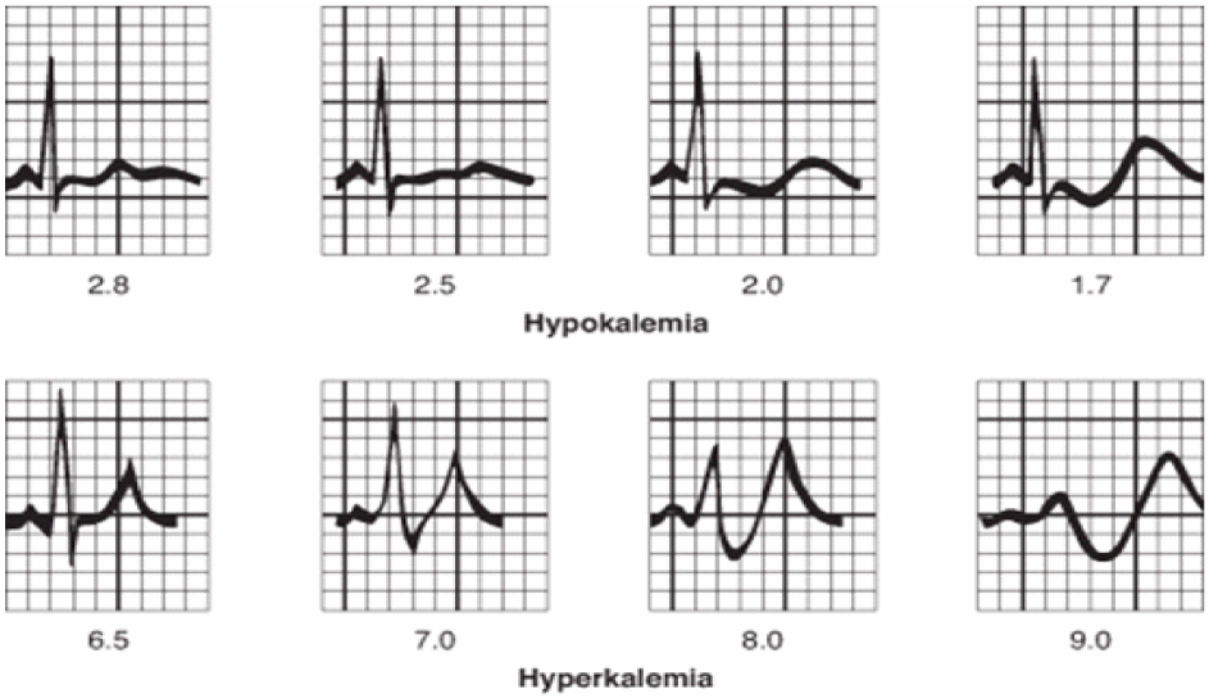
Potassium is the major intracellular cation. The intracellular concentration of potassium is 150 to 160 mEq/L (SI, 150 to 160 mmol/L); evaluation of serum potassium measures the extracellular levels of this electrolyte.

Potassium is important in maintaining cellular electrical neutrality. The sodium-potassium active transport pump maintains the ratio of intracellular to extracellular potassium that determines the resting membrane potential necessary for nerve impulse transmission. Disturbances in this ratio alter cardiac rhythms, transmission and conduction of nerve impulses, and muscle contraction.

Cardiac effects of hypokalemia are usually minimal until plasma K levels are < 3 mEq/L. Hypokalemia produces sagging of the ST segment, depression of the T wave, and elevation of the U wave. With marked hypokalemia, the T wave becomes progressively smaller and the U wave becomes increasingly larger. Sometimes, a flat or positive T wave merges with a positive U wave, which may be confused with QT prolongation (see Fig). Hypokalemia may produce premature ventricular and atrial contractions, ventricular and atrial tachyarrhythmias, and 2nd- or 3rd-degree atrioventricular block. Such arrhythmias become more severe with increasingly severe hypokalemia; eventually, ventricular fibrillation may occur. Patients with significant preexisting heart disease and/or those receiving digoxin are at risk of cardiac conduction abnormalities

Initial ECG changes in hyperkalemia occur with K > 5.5 mEq/L, characterized by shortening of the QT interval and tall, symmetric, peaked T waves. K > 6.5 mEq/L produces nodal and ventricular arrhythmias, widening of the QRS complex, PR interval

ECG patterns in hypokalemia and hyperkalemia.



prolongation, and disappearance of the P wave. Finally, the QRS complex degenerates into a sine wave pattern, and ventricular fibrillation or asystole ensues.malities even from mild hypokalemia.

Potassium test interference

The following factors may cause elevated potassium levels:

- repeated fist clenching before venipuncture
- excessive or rapid potassium infusion, spironolactone or penicillin G potassium therapy, or renal toxicity from administration of amphotericin B, methicillin, or tetracycline.

Magnesium is absorbed by the small intestine and is excreted in the urine and feces.

PHOSPHATE

Normally, serum phosphate levels in adults range from 2.7 to 4.5 mg/dl (SI, 0.87 to 1.45 mmol/L). In children, the normal range is 4.5 to 6.7 mg/dl (SI, 1.45 to 1.78 mmol/L).Phosphate are essential in the storage and utilization of energy, calcium regulation, red blood cell function, acid-base balance, bone formation, and metabolism of carbohydrates, protein, and fat. Tests for phosphate measure serum levels of phosphate,

Hyperkalemia	Hypokalemia
Causes • Increased potassium intake • Shift in the concentration from intracellular to extracellular fluid • Decreased renal excretion • Infusion of stored whole blood • Penicillin G • Replacement potassium • Acidosis • Insulin deficiency • Burns • Crushing injuries • Diabetic ketoacidosis • Extensive surgery • Myocardial infarction • Renal failure	Causes • diabetic ketoacidosis and insulin administration without potassium supplements • GI and renal disorders • vomiting • diarrhea • gastric suctioning • diuretics • excessive aldosterone secretion • excessive licorice ingestion.

MAGNESIUM

Normally, serum magnesium levels range from 1.3 to 2.1 mg/dl (SI, 0.65 to 1.05 mmol/L).

Magnesium is a commonly overlooked electrolyte vital to neuromuscular function. Magnesium activates many essential enzymes and affects the metabolism of nucleic acids and proteins. It also helps transport sodium and potassium across cell membranes and, through its effects on the secretion of parathyroid hormone, influences intracellular calcium levels.

Magnesium measured to evaluate electrolyte status and assess neuromuscular or renal function. The serum magnesium test, a quantitative analysis, measures serum levels of magnesium, which is (after potassium) the most abundant intracellular cation.

Most magnesium is found in bone and in intracellular fluid; a small amount is found in extracellular fluid.

the dominant cellular anions.The GIT absorbs a considerable amount of phosphate from dietary source, but adequate levels of Vitamin D are necessary for their absorption. The kidneys regulate phosphate excretion and retention. Because calcium and phosphates interact in a reciprocal relationship, urinary excretion of phosphate increases or decreases in inverse proportion to serum calcium levels.Abnormal phosphate levels result more commonly from improper excretion than from abnormal ingestion or absorption from dietary sources.

Hypophosphatemia may result from:

- malnutrition,
- malabsorption syndromes,
- hyperparathyroidism,
- renal tubular acidosis,
- treatment of diabetic acidosis.

Hypomagnesemia		Hypermagnesemia
Cause		• Hypermagnesemia that is not caused by magnesium administration or ingestion most commonly occurs in renal failure, when the kidneys excrete inadequate amounts of magnesium. • Adrenal insufficiency (Addison's disease) can also elevate serum magnesium levels.
Alcoholism	Due to both inadequate intake and excessive renal excretion	
Gl losses	Chronic diabetes	
	Steatorrhea	
Pregnancy-related	Pre-eclampsia/eclampsia (see Abnormalities of Pregnancy: Preeclampsia and Eclampsia)	
	Lactation (increased Mg requirements)	
Primary renal losses	Rare disorder(s). Inappropriately high urinary Mg excretion without apparent cause (eg, Gitelman's syndrome)	
Secondary renal losses	Loop and thiazide diuretics	
	Hypercalcemia	
	After removal of parathyroid tumor	
	Diabetic ketoacidosis	
	Hypersecretion of aldosterone, thyroid hormones, or ADH	
	Nephrotoxins (amphotericin B SOME TRADE NAMES ABELCET AMBISOME AMPHOCIN AMPHOTEC cisplastin, cyclosporine SOME TRADE NAMES NEORAL SANDIMMUNE aminoglycosides)	

In children, low phosphate levels can suppress normal growth.

Hyperphosphatemia may result from;
- skeletal disease,
- healing fractures,
- hypoparathyroidism,
- acromegaly,
- diabetic acidosis,
- high intestinal obstruction,
- renal failure.

Elevated phosphate levels are rarely clinically significant; however, if prolonged, they can alter bone metabolism by causing abnormal calcium phosphate deposits.

CALCIUM

Normal calcium values are as follows:
- *total calcium:* 8.2 to 10.2 mg/dl (SI, 2.05 to 2.54 mmol/L) in adults; 8.6 to 11.2 mg/dl (SI, 2.15 to 2.79 mmol/L) in children
- *ionized calcium:* 4.65 to 5.28 mg/dl (SI, 1.1 to 61.32 mmol/L).

Total calcium measurement is the most commonly performed test for evaluation of serum calcium levels. Approximately 1% of the total calcium in the body circulates in the blood. Of this, about 50% is bound to plasma proteins and 40% is ionized, or free.Evaluation of serum calcium levels measures the total amount of calcium circulating in the blood. Evaluation of ionized calcium levels measures the fraction of serum calcium that's in the ionized form, which is the most physiologically active form of serum calcium. The other 99% of the calcium in the body is stored in the bones and teeth.Many laboratories don't have the equipment to measure ionized calcium levels. Because of this, serum albumin should be measured at the same time serum calcium is measured because the serum calcium level decreases 0.8 mg/dl for every 1-g decrease in the serum albumin level. The measured serum calcium is then adjusted upward by the amount of decrease in serum albumin. Ionized calcium is estimated to be approximately half of the adjusted calcium value.Hypercalcemia may occur in patients with;

- hyperparathyroidism and parathyroid tumors (because of oversecretion of parathyroid hormone),
- Paget's disease of the bone,
- multiple myeloma,
- metastatic carcinoma,
- multiple fractures,
- Prolonged immobilization.
- Elevated serum calcium levels may also result from inadequate excretion of calcium, such as in adrenal insufficiency and renal disease
- From excessive calcium ingestion;
- From overuse of antacids such as calcium carbonate.

Calcium test interference

The following factors may alter calcium test results:

- excessive ingestion of vitamin D or its derivatives (dihydrotachysterol, calcitrol)
- use of androgens, calciferol-activated calcium salts, progestins-estrogens, or thiazide diuretics, which may elevate levels
- chronic laxative use
- excessive transfusions of citrated blood
- administration of acetazolamide, corticosteroids, or mithramycin.Hypocalcemia may result from;
- hypoparathyroidism,
- total parathyroidectomy,
- malabsorption.
- Cushing's syndrome,
- renal failure,
- acute pancreatitis,
- peritonitis.

CHLORIDE

Normal serum chloride levels range from 100 to 108 mEq/L (SI, 100 to 108 mmol/L)Interacting with sodium, chloride helps maintain the osmotic pressure of blood and therefore helps regulate blood volume and arterial pressure. Chloride levels also affect acid-base balance. Serum concentrations of chloride are regulated by aldosterone secondarily to regulation of sodium. Chloride is absorbed from the intestines and is excreted primarily by the kidneys.Chloride levels relate inversely to those of bicarbonate and thus reflect acid-base balance. Excessive loss of gastric juices or of other secretions containing chloride nay cause hypochloremic metabolic alkalosis; excessive chloride retention or ingestion may lead to hyperchloremic metabolic acidosis.The serum chloride test, a quantitative analysis, measures serum levels of chloride, the major extracellular fluid anion.

The lowdown on levels

Hyperchloremia may result from;

- bicarbonate loss caused by diarrhea,
- severe dehydration,
- complete renal shutdown,
- head injury (producing neurogenic hyperventilation),
- primary aldosteronism.Low chloride levels (hypochloremia) are usually associated with low sodium and potassium levels. Possible underlying causes include prolonged vomiting, gastric suctioning, intestinal fistula, chronic renal failure, and Addison's disease. Heart failure or edema resulting in excess extracellular fluid can cause dilutional hypochloremia.

Chloride test interference

- Ammonium chloride, cholestyramine, or excessive I.V. infusion of sodium chloride may elevate serum chloride levels.
- Thiazide diuretics, ethacrynic acid, furosemide, or bicarbonates, or prolonged I.V. infusion of dextrose 5% in water may decrease serum chloride levels.

ANION GAP

Normally, the anion gap ranges from 8 to 14 mEq/L (SI, 8 to 14 mmol/L). A normal anion gap doesn't rule out metabolic acidosis.The anion gap reflects anion-cation balance in the serum and helps distinguish types of metabolic acidosis without expensive, time-consuming measurement of all serum electrolytes. The anion gap test uses serum levels of routinely measured electrolytes – sodium, chloride, and bicarbonate – for a quick calculation based on a simple physical principle: Total concentrations of cations and anions are normally equal, thereby maintaining electrical neutrality in serum.Because sodium accounts for more than 90% of circulating cations, whereas chloride and bicarbonate

together account for 85% of the counterbalancing anions, the gap between measured cation and anion levels represents those anions not routinely measured, including sulfate, phosphates, proteins, and organic acids, such as ketone bodies and lactic acid.Normal anion gap acidosisWhen acidosis results from loss of bicarbonate in the urine or other body fluids, renal reabsorption of sodium promotes retention of chloride, and the anion gap remains unchanged. Thus, metabolic acidosis associated with excessive chloride levels is known as normal anion gap acidosis.High anion gap acidosisWhen acidosis results from accumulation of metabolic acids, as with lactic acidosis, the anion gap increases above 14 mEq/L (14 mmol/L) with the increase being in unmeasured anions. Metabolic acidosis caused by such accumulation is known as high anion gap acidosis. Because the anion gap determines only total anion-cation balance, it doesn't necessarily reflect abnormal values for individual electrolytes. Further investigation and diagnostic tests are usually necessary to determine the specific cause of metabolic acidosis.Understanding anion gap and metabolic acidosisMetabolic acidosis with a normal anion gap (8 to 14 mEq/L [SI, 8 to 14 μmol/L]) occurs with bicarbonate loss, such as from:

- hypokalemic acidosis associated with renal tubular acidosis, diarrhea, or ureteral diversions
- hyperkalemic acidosis caused by acidifying agents (for example, ammonium chloride or hydrochloric acid), hydronephrosis, or sickle cell nephropathy.Metabolic acidosis with an increased anion gap (above 14 mEq/L [SI, greater than 14 mmol/L]) occurs with accumulation of organic acids, sulfates, or phosphate, such as from:
- renal failure
- ketoacidosis associated with starvation, diabetes mellitus, or alcohol abuse
- lactic acidosis
- ingestion of toxins, such as salicylates, methanol, ethylene glycol (antifreeze), and paraldehyde.

A decreased anion gap (less than 8 mEq/L) is rare but may occur in hypermagnesemia and in paraproteinemic states, such as multiple myeloma and Waldenström's macroglobulinemia.

Anion gap test interference

A range of medication and other substances can alter the anion gap.

Increases the gap

- Ammonium chloride
- Antihypertensives
- Bicarbonates
- Corticosteroids
- Ethacrynic acid
- furosemide
- Prolonged infusion of dextrose 5% in water
- Salicylates
- Thiazide diuretics

Decreases the gap

- Chlorothiazide diuretics
- Chlorpropamide
- Cortisome
- Diuretics
- Excessive ingestion of alkalis or licorice
- Lithium
- Vasopressin