

Electrolytes

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Introduction

Electrolytes are essential for basic life functioning, such as maintaining electrical neutrality in cells and generating and conducting action potentials in the nerves and muscles. Significant electrolytes include sodium, potassium, chloride, magnesium, calcium, phosphate, and bicarbonates. Electrolytes come from our food and fluids.

These electrolytes can be imbalanced, leading to high or low levels. High or low levels of electrolytes disrupt normal bodily functions and can lead to life-threatening complications. This article reviews the basic physiology of electrolytes and their abnormalities, and the consequences of electrolyte imbalance.

Sodium

Sodium, an osmotically active cation, is one of the essential electrolytes in the extracellular fluid. It is responsible for maintaining the extracellular fluid volume and regulating the membrane potential of cells. Sodium is exchanged along with potassium across cell membranes as part of active transport.[1]

Sodium regulation occurs in the kidneys. The proximal tubule is where the majority of sodium reabsorption takes place. In the distal convoluted tubule, sodium undergoes reabsorption. Sodium transport occurs via sodium-chloride symporters, controlled by the hormone aldosterone.[2]

Among the electrolyte disorders, hyponatremia is the most frequent. Hyponatremia is diagnosed when the serum sodium level is less than 135 mmol/L. Hyponatremia has neurological manifestations.[3] Patients may present with headaches, confusion, nausea, and delirium. Hypernatremia occurs when serum sodium levels are greater than 145 mmol/L. Symptoms of hypernatremia include tachypnea, sleeping difficulty, and restlessness. Rapid sodium corrections can have severe consequences like cerebral edema and osmotic demyelination syndrome (ODS). Other factors like chronic alcohol misuse disorder and malnutrition also play a role in the development of ODS.[4]

Potassium

Potassium is mainly an intracellular ion. The sodium-potassium adenosine triphosphatase pump is primarily responsible for regulating the homeostasis between sodium and potassium, which pumps out sodium in exchange for potassium, which moves into the cells. In the kidneys, the filtration of potassium takes place at the glomerulus. Potassium reabsorption occurs at the proximal convoluted tubule and thick ascending loop of Henle.[5] Potassium secretion occurs at the distal convoluted tubule. Aldosterone increases potassium secretion.[6] Potassium channels and potassium-chloride cotransporters at the apical tubular membrane also secrete potassium.[5]

Potassium derangements may result in cardiac arrhythmias. Hypokalemia occurs when serum potassium levels are under 3.6 mmol/L. The features of hypokalemia include weakness, fatigue, and muscle twitching. Hypokalemic paralysis is generalized body weakness that can be either familial or sporadic.[7] Hyperkalemia occurs when the serum potassium levels are above 5.5

mmol/L, which can result in arrhythmias. Muscle cramps, muscle weakness, rhabdomyolysis, and myoglobinuria may be presenting signs and symptoms of hyperkalemia.[8]

Calcium

Calcium has a significant physiological role in the body. It is involved in skeletal mineralization, contraction of muscles, the transmission of nerve impulses, blood clotting, and secretion of hormones. The diet is the predominant source of calcium. Calcium is a predominately extracellular cation. Calcium absorption in the intestine is primarily controlled by the hormonally active form of vitamin D, which is 1,25-dihydroxy vitamin D3. Parathyroid hormone also regulates calcium secretion in the distal tubule of the kidneys.[9] Calcitonin acts on bone cells to decrease calcium levels in the blood.

Hypocalcemia diagnosis requires checking the serum albumin level to correct for total calcium. Hypocalcemia is diagnosed when the corrected serum total calcium levels are less than 8.8 mg/dL, as in vitamin D deficiency or hypoparathyroidism. Checking serum calcium levels is a recommended test in post-thyroidectomy patients.[10] Hypercalcemia is when corrected serum total calcium levels exceed 10.7 mg/dL, as seen with primary hyperparathyroidism. Humoral hypercalcemia presents in malignancy, primarily due to PTHrP secretion.[11]

Bicarbonate

The acid-base status of the blood drives bicarbonate levels. The kidneys predominantly regulate bicarbonate concentration and maintain the acid-base balance. Kidneys reabsorb the filtered bicarbonate and generate new bicarbonate by net acid excretion, which occurs through the excretion of titrable acid and ammonia. Diarrhea usually results in bicarbonate loss, causing an imbalance in acid-base regulation.[12] Many kidney-related disorders can result in imbalanced bicarbonate metabolism leading to excess bicarbonate in the body.[13]

Magnesium

Magnesium is an intracellular cation. Magnesium is mainly involved in adenosine triphosphate (ATP) metabolism, proper functioning of muscles, neurological functioning, and neurotransmitter release. When muscles contract, calcium re-uptake by the calcium-activated ATPase of the sarcoplasmic reticulum is brought about by magnesium.[14] Hypomagnesemia occurs when the serum magnesium levels are less than 1.46 mg/dL. Alcohol use disorder, gastrointestinal conditions, and excessive renal loss may result in hypomagnesemia. It commonly presents with ventricular arrhythmias, which include torsades de pointes. Hypomagnesemia may also result from the use of certain medications, such as omeprazole.[15]

Chloride

Chloride is an anion found predominantly in the extracellular fluid. The kidneys predominantly regulate serum chloride levels. Most chloride, filtered by the glomerulus, is reabsorbed by both proximal and distal tubules (majorly by proximal tubule) by both active and passive transport. [16]

Hyperchloremia can occur due to gastrointestinal bicarbonate loss. Hypochloremia presents in gastrointestinal losses like vomiting or excess water gain like congestive heart failure.

Phosphorus

Phosphorus is an extracellular fluid cation. Eighty-five percent of the total body phosphorus is in the bones and teeth in the form of hydroxyapatite; the soft tissues contain the remaining 15%. Phosphate plays a crucial role in metabolic pathways. It is a component of many metabolic intermediates and, most importantly, of ATP and nucleotides. Vitamin D3, PTH, and calcitonin

regulate phosphate simultaneously with calcium. The kidneys are the primary avenue of phosphorus excretion.

Phosphate imbalance is most commonly due to one of three processes: impaired dietary intake, gastrointestinal disorders, and deranged renal excretion.[17]

Specimen Collection

A blood specimen for electrolytes uses lithium heparin tubes, plus the standard phlebotomy equipment and personnel, as with any blood draw.[18]

Procedures

Blood is collected in lithium heparin tubes and then goes to the laboratory to evaluate serum electrolytes.[18] The collection tubes should not be left for an extended period as cell lysis can occur, causing the intracellular electrolytes and other contents to come out in the serum.

Indications

Indications to order serum electrolyte panels are numerous. Some indications are:

- Routine blood investigations
- Routine monitoring of hospitalized patients on medications, receiving fluid therapy, undergoing dietary changes, or being treated for ongoing illnesses.
- Any illness that can cause electrolyte derangements, such as malnutrition, gastrointestinal disorders, cardiac disorders, kidney dysfunction, endocrine disorders, circulatory disorders, lung disorders, and acid-base imbalance[19]
- Arrhythmias
- Cardiac arrest
- Use of diuretics or any medications that can interfere with fluid and electrolyte homeostasis

Potential Diagnosis

Measurement of electrolytes will help clinicians in the diagnosis of a medical condition, the effectiveness of treatment, and the potential side effect of medications. Examples include:

A patient with heart failure receiving diuretics needs a workup for sodium, potassium, bicarbonate, and magnesium, as diuretics can exert adverse effects on electrolyte balance.[20]

A patient that presents with weakness needs a basic electrolyte workup, as an electrolyte imbalance, especially in sodium and potassium levels, can lead to generalized weakness.

A patient with gastroesophageal reflux disease on long-term proton pump inhibitor therapy should be monitored for hypomagnesemia.

Normal and Critical Findings

Laboratory Values

Serum Sodium

- Normal range: 135 to 145 mmol/L
- Mild to moderate hyponatremia: 125 to 135 mmol/L

- Severe hyponatremia: less than 125 mmol/L
- Mild to moderate hyponatremia: 145 to 160 mmol/L
- Severe hyponatremia: greater than 160 mmol/L

Serum Potassium

- Normal range: 3.6 to 5.5 mmol/L
- Mild hypokalemia: less than 3.6 mmol/L
- Moderate hypokalemia: less than 2.5 mmol/L
- Severe hypokalemia: less than greater than 2.5 mmol/L
- Mild hyperkalemia: 5 to 5.5 mmol/L
- Moderate hyperkalemia: 5.5 to 6.5 mmol/L
- Severe hyperkalemia: 6.5 to 7 mmol/L

Serum Calcium

- Normal range: 8.8 to 10.7 mg/dL
- Hypocalcemia: less than 8.8 mg/dL
- Mild to moderate hypercalcemia: greater than 10.7 to 11.5 mg/dL
- Severe hypercalcemia: greater than 11.5 mg/dL

Serum Magnesium

- Normal range: 1.46 to 2.68 mg/dL
- Hypomagnesemia: less than 1.46 mg/dL
- Hypermagnesemia: greater than 2.68 mg/dL

Bicarbonate

- Normal range: 23 to 30 mmol/L
- It increases or decreases depending on the acid-base status.

Phosphorus

- Normal range: 3.4 to 4.5 mg/dL
- Hypophosphatemia: less than 2.5 mg/dL
- Hyperphosphatemia: greater than 4.5 mg/dL

Interfering Factors

Factors such as total protein content, hormones, and total body volume status can biochemically influence electrolyte levels. Hypomagnesemia can lead to hypocalcemia due to its effects on parathyroid hormone activity. Intravenous insulin administration is associated with a spurious decrease in potassium levels as insulin shifts potassium intracellularly.^[21]

Most serum calcium is bound to proteins; albumin-bound calcium comprises about 80%.

Therefore, a patient with hypoalbuminemia, as seen in liver cirrhosis or nephrotic syndrome, will

demonstrate artificially abnormal serum calcium levels.[22]

Complications

Hyponatremia, hypernatremia, and hypomagnesemia can lead to neurological consequences such as seizures.

Hypokalemia and hyperkalemia, as well as hypocalcemia, may cause cardiac arrhythmias.[23]

Bicarbonate imbalance can lead to metabolic acidosis or alkalosis.

Some consequences of potassium, calcium, and magnesium abnormalities are fatigue, lethargy, and muscle weakness.

Patient Safety and Education

Patients should be counseled to take all medications exactly as prescribed to avoid any potential adverse effect of electrolyte imbalance. They should also call for immediate medical help if experiencing generalized weakness, muscle aches, or altered mental status.

Clinical Significance

Some of the common causes of electrolyte disorders seen in clinical practices are:

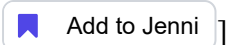
- Hyponatremia: low dietary sodium intake, primary polydipsia, syndrome of inappropriate antidiuretic hormone secretion (SIADH), heart failure, cirrhosis, adrenal insufficiency, prolonged hyperglycemia, and severe dyslipidemia.
- Hypernatremia: unreplaced fluid loss via the skin or gastrointestinal tract, osmotic diuresis, or hypertonic saline administration.
- Hypokalemia: hyperaldosteronism or the use of loop diuretics.
- Hyperkalemia: metabolic acidosis, insulin deficiency, hypoaldosteronism, prolonged beta-blocker use, or acute or chronic kidney disease.
- Hypercalcemia: malignancy, hyperparathyroidism, or chronic granulomatous diseases such as tuberculosis or sarcoidosis.[24]
- Hypocalcemia: acute pancreatitis, iatrogenic parathyroid dysfunction, resistance to parathyroid hormone, hypomagnesemia, or sepsis.
- Hypermagnesemia: increased oral magnesium intake.
- Hypomagnesemia: increased renal losses with diuretics, alcohol use disorder, or gastrointestinal losses.[25]
- Bicarbonate level: increases in primary metabolic alkalosis or compensation to primary respiratory acidosis and decreases in primary metabolic acidosis or compensation to primary respiratory alkalosis.
- Hyperchloremia: excessive normal saline infusion.
- Hypochloremia: increased gastrointestinal or renal losses.
- Hypophosphatemia: refeeding syndrome, vitamin D deficiency, or hyperparathyroidism.[26]
- Hyperphosphatemia: hyperparathyroidism or chronic kidney disease.

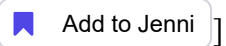
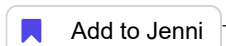
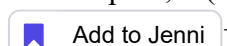
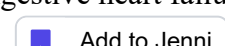
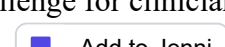
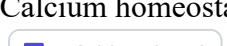
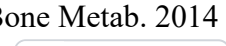
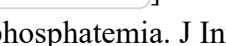
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References

1. Ferrannini E. Sodium-Glucose Co-transporters and Their Inhibition: Clinical Physiology. *Cell Metab.* 2017 Jul 05;26(1):27-38. [PubMed: 28506519] [Add to Jenni](#)]
2. Palmer LG, Schnermann J. Integrated control of Na transport along the nephron. *Clin J Am Soc Nephrol.* 2015 Apr 07;10(4):676-87. [PMC free article: PMC4386267] [PubMed: 25098598] [Add to Jenni](#)]
3. Buffington MA, Abreo K. Hyponatremia: A Review. *J Intensive Care Med.* 2016 May;31(4):223-36. [PubMed: 25592330] [Add to Jenni](#)]
4. Ambati R, Kho LK, Prentice D, Thompson A. Osmotic demyelination syndrome: novel risk factors and proposed pathophysiology. *Intern Med J.* 2023 Jul;53(7):1154-1162. [PubMed: 35717664] [Add to Jenni](#)]
5. Gumz ML, Rabinowitz L, Wingo CS. An Integrated View of Potassium Homeostasis. *N Engl J Med.* 2015 Jul 02;373(1):60-72. [PMC free article: PMC5675534] [PubMed: 26132942] [Add to Jenni](#)]
6. Ellison DH, Terker AS, Gamba G. Potassium and Its Discontents: New Insight, New Treatments. *J Am Soc Nephrol.* 2016 Apr;27(4):981-9. [PMC free article: PMC4814195] [PubMed: 26510885] [Add to Jenni](#)]
7. Stedwell RE, Allen KM, Binder LS. Hypokalemic paralyses: a review of the etiologies, pathophysiology, presentation, and therapy. *Am J Emerg Med.* 1992 Mar;10(2):143-8. [PubMed: 1586409] [Add to Jenni](#)]
8. Viera AJ, Wouk N. Potassium Disorders: Hypokalemia and Hyperkalemia. *Am Fam Physician.* 2015 Sep 15;92(6):487-95. [PubMed: 26371733] [Add to Jenni](#)]
9. Veldurthy V, Wei R, Oz L, Dhawan P, Jeon YH, Christakos S. Vitamin D, calcium homeostasis and aging. *Bone Res.* 2016;4:16041. [PMC free article: PMC5068478] [PubMed: 27790378] [Add to Jenni](#)]
10. Cooper MS, Gittoes NJ. Diagnosis and management of hypocalcaemia. *BMJ.* 2008 Jun 07;336(7656):1298-302. [PMC free article: PMC2413335] [PubMed: 18535072] [Add to Jenni](#)]
11. Turner JJO. Hypercalcaemia - presentation and management . *Clin Med (Lond).* 2017 Jun;17(3):270-273. [PMC free article: PMC6297576] [PubMed: 28572230] [Add to Jenni](#)]
12. Hamm LL, Nakhoul N, Hering-Smith KS. Acid-Base Homeostasis. *Clin J Am Soc Nephrol.* 2015 Dec 07;10(12):2232-42. [PMC free article: PMC4670772] [PubMed: 26597304] [Add to Jenni](#)]
13. Kraut JA, Madias NE. Adverse Effects of the Metabolic Acidosis of Chronic Kidney Disease. *Adv Chronic Kidney Dis.* 2017 Sep;24(5):289-297. [PubMed: 29031355] [Add to Jenni](#)]
14. Jahnhen-Dechent W, Ketteler M. Magnesium basics. *Clin Kidney J.* 2012 Feb;5(Suppl 1):i3-i14. [PMC free article: PMC4455825] [PubMed: 26069819] [Add to Jenni](#)]
15. Hansen BA, Bruserud Ø. Hypomagnesemia as a potentially life-threatening adverse effect of omeprazole. *Oxf Med Case Reports.* 2016 Jul;2016(7):147-9. [PMC free article: PMC4962887] [PubMed: 27471598] [Add to Jenni](#)]
16. Morrison G. Serum Chloride. In: Walker HK, Hall WD, Hurst JW, editors. *Clinical Methods: The History, Physical, and Laboratory Examinations.* 3rd ed. Butterworths;

Boston: 1990. [PubMed: 21250151] 

17. Berkelhammer C, Bear RA. A clinical approach to common electrolyte problems: 3. Hypophosphatemia. *Can Med Assoc J*. 1984 Jan 01;130(1):17-23. [PMC free article: PMC1875686] [PubMed: 6418367] 
18. Toffaletti J, Ernst P, Hunt P, Abrams B. Dry electrolyte-balanced heparinized syringes evaluated for determining ionized calcium and other electrolytes in whole blood. *Clin Chem*. 1991 Oct;37(10 Pt 1):1730-3. [PubMed: 1914173] 
19. Raza M, Kumar S, Ejaz M, Azim D, Azizullah S, Hussain A. Electrolyte Imbalance in Children With Severe Acute Malnutrition at a Tertiary Care Hospital in Pakistan: A Cross-Sectional Study. *Cureus*. 2020 Sep 19;12(9):e10541. [PMC free article: PMC7574973] [PubMed: 33094080] 
20. Cody RJ, Pickworth KK. Approaches to diuretic therapy and electrolyte imbalance in congestive heart failure. *Cardiol Clin*. 1994 Feb;12(1):37-50. [PubMed: 8181024] 
21. Liamis G, Liberopoulos E, Barkas F, Elisaf M. Spurious electrolyte disorders: a diagnostic challenge for clinicians. *Am J Nephrol*. 2013;38(1):50-7. [PubMed: 23817179] 
22. Boden SD, Kaplan FS. Calcium homeostasis. *Orthop Clin North Am*. 1990 Jan;21(1):31-42. [PubMed: 2404236] 
23. Hoppe LK, Muhlack DC, Koenig W, Carr PR, Brenner H, Schöttker B. Association of Abnormal Serum Potassium Levels with Arrhythmias and Cardiovascular Mortality: a Systematic Review and Meta-Analysis of Observational Studies. *Cardiovasc Drugs Ther*. 2018 Apr;32(2):197-212. [PubMed: 29679302] 
24. Negri AL, Rosa Diez G, Del Valle E, Piulats E, Greloni G, Quevedo A, Varela F, Diehl M, Bevione P. Hypercalcemia secondary to granulomatous disease caused by the injection of methacrylate: a case series. *Clin Cases Miner Bone Metab*. 2014 Jan;11(1):44-8. [PMC free article: PMC4064440] [PubMed: 25002879] 
25. Agus ZS. Mechanisms and causes of hypomagnesemia. *Curr Opin Nephrol Hypertens*. 2016 Jul;25(4):301-7. [PubMed: 27219040] 
26. Marinella MA. Refeeding syndrome and hypophosphatemia. *J Intensive Care Med*. 2005 May-Jun;20(3):155-9. [PubMed: 15888903] 

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