

[Test Your Knowledge: Potassium Homeostasis](#)

A recent Core Curriculum by [Palmer and Clegg](#) published in *AJKD* reviews the regulatory aspects of normal K⁺ physiology as a preface to highlighting common disorders in K⁺ homeostasis and their treatment. Test your knowledge on this topic with the quiz below.

1. A 56-year-old male with chronic lymphocytic leukemia (CLL) presents to the emergency room with fatigue and complaints of body aches. A laboratory data review reveals WBC count of 677 X10⁹/L and Hemoglobin of 8 g/dl with total platelets of 150 X10⁹/L. The patient has normal kidney function with serum creatinine of 0.7 mg/dl but is noted to have a **serum** potassium level of 4.0 meq/L and a **plasma** potassium level of 5.6 meq/L. The patient is not on any medications. He is initiated on normal saline infusion at 75 cc/hr. What is the cause of the elevated plasma potassium?
 - A. Pseudohyperkalemia
 - B. Shifting of potassium out of the cells
 - C. Resolving acute kidney injury
 - D. Reverse pseudohyperkalemia
2. A 67-year-old cardiologist with CKD Stage 3 (eGFR 45 ml/min) is visiting you for a regular clinic visit. He is currently on metoprolol, albuterol, spironolactone, and takes occasional ibuprofen. He has long standing hypertension and diabetes mellitus type 2. Which one the following **DOES NOT** increase his risk of hyperkalemia in CKD?
 - A. Metoprolol
 - B. Albuterol
 - C. Chronic diabetes mellitus
 - D. NSAID use
 - E. Spironolactone
3. An 18-year-old male has persistent hyperkalemia over the last few years. He has several direct family members that also developed hyperkalemia at a young age as well. His physical exam reveals a blood pressure of 160/80 mmHg and heart rate of 78 bpm. On exam, he has a short stature and some dental abnormalities. He has some degree of intellectual challenges during school. His lab data reveals a serum potassium of 5.7 meq/L and metabolic acidosis. What is the most likely cause of his hyperkalemia?

- A. Bartter's syndrome
 - B. Pseudohypoaldosteronism type 2 (Gordon syndrome)
 - C. Gitelman's syndrome
 - D. Pseudohypoaldosteronism type 1
4. A 67-year-old female with metastatic cervical cancer develops sudden onset of mild acute kidney injury (serum creatinine 1.3 mg/dl from baseline of 1.0 mg/dl), hyperkalemia, and hyponatremia. A kidney sonogram reveals moderate bilateral hydronephrosis. What is the predominant mechanism of hyperkalemia in hydronephrosis?
- A. Shifting of potassium out of the cells
 - B. Proximal renal tubular acidosis
 - C. Hyporeninemic hypoaldosteronism
 - D. Impaired glomerular filtration

- Quiz prepared by [Kenar Jhaveri](#), AJKDBlog Contributor. Follow him [@kdjhaveri](#).

To view [Palmer and Clegg \(FREE\)](#), please visit [AJKD.org](#).

Title: [Physiology and Pathophysiology of Potassium Homeostasis: Core Curriculum 2019](#)

Authors: Biff F. Palmer and Deborah J. Clegg

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[Answers to Test Your Knowledge: Potassium Homeostasis](#)

1. **D. Reverse pseudohyperkalemia**

The most common electrolyte disorder encountered in CLL patients is pseudohyperkalemia. Leakage of potassium from the intracellular compartment at the time of collection due to hemolysis, cell fragility or heparin-induced damage can lead to spurious elevation of measured potassium levels, which is known as pseudohyperkalemia. This is also seen in myeloproliferative disorders and thrombocytosis. As a result, the potassium concentration is generally higher in serum compared with plasma from platelets during clotting.

Reverse pseudohyperkalemia refers to a false elevation in plasma potassium with normal serum levels and is the correct answer here. This is most commonly seen in hematologic malignancy, and is due to heparin-induced cell membrane damage during processing. It may also result from mechanical damage to cells during transport of specimens to the laboratory.

2. **B. Albuterol**

A spectrum of abnormalities in the renin-angiotensin aldosterone system have been reported in patients with diabetes mellitus. These include hyporeninemic hypoaldosteronism and normal renin release alongside reduced capacity for aldosterone release. Hypoaldosteronism and concomitant use of angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, or aldosterone blockers, markedly increases the risk for hyperkalemia.

Beta blockers leads to potassium shifting out of the cell and can add to the risk of hyperkalemia in CKD. NSAIDs and beta blockers also lead to inhibition of renin release from juxtaglomerular cells.

Albuterol is a beta agonist and allows for shifting of potassium into the cells and is used in treatment of acute hyperkalemia. Hence, the correct answer is albuterol.

3. **B. Pseudohypoaldosteronism type 2 (Gordon syndrome)**

Hyperkalemia and metabolic acidosis are key features of familial hyperkalemic hypertension (pseudohypoaldosteronism type II, Gordon syndrome). Mutations in the WNK4 and WNK1 kinases are most commonly the cause for this autosomal

dominant form of hypertension. While an increase in plasma K⁺ concentration normally stimulates aldosterone release from the adrenal gland, plasma aldosterone levels are generally low in this disorder. These patients usually have short stature, club feet and stiff joints as the patient presented in the case.

Pseudohypoaldosteronism type I is characterized by hyperkalemia, metabolic acidosis, and renal salt wasting and is due to mineralocorticoid resistance. Hypertension is not a common finding in this disease. Homozygous mutations in the 3 subunits of ENaC are responsible for the autosomal recessive form of the disease. There is an autosomal dominant form of the disease that arises as a result of mutations in the mineralocorticoid receptor.

The above presentation is not the phenotype of Bartter's or Gitelman's syndrome, as patients tend to be hypokalemic and would be expected to have a normal or slightly low blood pressure.

4. C. Hyporeninemic hypoaldosteronism

In urinary obstruction, the earliest defects occur in the distal nephron and accounts for the development of hyperkalemia with only a mild decrease in GFR. Impaired renin release causing hyporeninemic hypoaldosteronism often coexists in obstruction leading to a hyperkalemic renal tubular acidosis (Type IV) in such cases.

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