

Answers to Potassium Self Assessment Questions

1. D. Note the inadequate pCO₂ response, indicating a superimposed respiratory acidosis. A is wrong: a nongap acidosis is due to retention of normal body acid (e.g. HCl). B would have lowered the serum Na
2. E. hyperkalemia suppresses ammoniogenesis, leading to retention of acid. Correct the K, and you correct the ammonia problem, correcting the acidosis. As a diabetic, she probably has hyporeninemic hypoaldosteronism. Hyperglycemia and pseudohyperkalemia are potential causes of hyperkalemia, but the serum glucose is likely normal (note no decreased serum sodium) and you can never assume pseudohyperkalemia unless the patient has NO reasons to have a high K.
3. C. All the others are good hyperkalemia treatments. Digoxin might be dangerous in the setting of hyper (and hypo-)kalemia.
4. D. K lost in vomiting via renal bicarbonate diuresis. A consumes extra K in unrestrained cell growth. B causes hyperkalemia. D shifts K.
5. C. See the lecture
6. C. Na and Cl will be excreted in the urine in ongoing diuretic use, despite volume depletion. The urine K would be high, even if little K is ingested, because of the diuretic.
7. E. See the alkalosis lecture, and use of urine chloride. The big difference here is that vomiting leads to chloride depletion (and low urine CL) while hyperaldosteronism causes Na retention, mild volume expansion, and Cl excretion (with high urine CL).

Both lead to alkalemia, and the urine anion gap (ammonium excretion) would not be different, given similar serum K and pH. (*Side note: What would ammoniogenesis be in this case? Alkalemia suppresses ammoniogenesis, but hypokalemia stimulates it. Net is probably no change...*)