

Drug-Induced Hyperkalemia (K >5.5mEq/L)

K⁺ Gastrointestinal absorption is complete, resulting in daily excess intake of about 1 mEq/kg/d (60-100 mEq). This excess is excreted through the kidneys (90%) and the gut (10%), Therefore, K homeostasis is maintained predominantly through the regulation of renal excretion. Drug-induced hyperkalemia most often occurs from impaired renal potassium excretion.

Potassium administration alone, however, rarely causes hyperkalemia **in the absence** of an underlying defect in potassium homeostasis. Hyperkalemia may be mild (5 to 5.5 mEq/L), moderate (5.5 to 6 mEq/L), or severe (>6 mEq/L),

Excretion is decreased by the following:

Absence of aldosterone

Low sodium delivery to the distal tubule

Low urine flow

- **Renal dysfunction**

Medication	Mechanism	Average amount of change in Potassium level	Reported Percentage of Patients Who Develop Hyperkalemia from the Drug
Potassium Supp.	Potassium ingestion	Varies on supp.	3-24
Beta blockers	↓ beta-2 driven K ⁺ uptake	0.5-1 mEq/L	1-5
Digoxin	↓ Na ⁺ -K ⁺ -ATPase activity	0.2-0.5 mEq/L	2-15
Potassium-sparing drugs	Impaired renal excretion	0.8 mEq/L	2-19
NSAIDs	↓RBF, ↓GFR, Aldosterone antagonism	0.5-1.7 mEq/L	10-46
ACE inhibitors & Angiotensin-II blockers	↓Aldosterone synthesis, ↓RBF, ↓GFR	0.8mEq/L (ARB probably similar to ACEI)	10-38 2-7
Trimethoprim & Pentamidine	Block Na ⁺ channels in principal cells	0.5-1 mEq/L	6-21
Cyclosporine Tacrolimus	↓Aldosterone synthesis, ↓ Na ⁺ -K ⁺ -ATPase activity,	0.5-2.5 mEq/L	5-24 11-44 15-53
Heparin	↓Aldosterone synthesis	0.2-1.7 mEq/L	8-17