Table VII-3-1. Acid-Base Effects on Potassium Balance

	Acute Alkalosis	Chronic Alkalosis	Acute Acidosis	Chronic Acidosis
Plasma (K^+)	↓ Hypokalemia ↓		↑ Hyperkalemia ↑	
Intracellular (K^+)	↑	↓	↓	↓
Renal K^+ excretion	↑	↑	↓	↑
K^+ balance	Negative	Negative	Positive	Negative

Chapter Summary

In the proximal tubule, two-thirds of the water and electrolytes are reabsorbed, along with almost all the organic molecules filtered. An exception is urea. Because equal amounts of solutes and fluid are reabsorbed, the fluid remains isotonic.

The loop of Henle, acting as a countercurrent multiplier, creates an osmotic gradient in the medullary interstitium, with the tip reaching a maximum of 1200 mOsm/L. This value determines the maximum osmolarity of the urine. Because the descending limb is permeable to water, tubular osmolarity increases in this limb. Because the ascending limb is impermeable to water and sodium chloride is reabsorbed, osmolarity decreases in this limb, and the tubular fluid leaves hypotonic.

Net acid secretion into the filtrate occurs in the distal tubule/collecting duct. This excreted acid is buffered with either phosphate or ammonia. The bicarbonate secreted to the interstitium represents a net gain to body stores.

Metabolic alkalosis causes hypokalemia and negative potassium balance.

Acute and chronic metabolic acidosis both cause hyperkalemia; they differ in that the positive potassium balance in the first 24 hours becomes negative in the chronic condition.