and triamterene. The benefits of the RAS inhibitors in ameliorating hyperfiltration and progression of CKD often favor their cautious and judicious use with very close monitoring of plasma potassium concentration. Coadministration of potassium-lowering agents such as patiromer may allow for the use of RAS inhibitors with reduced risk of hyperkalemia.

Certain causes of CKD can be associated with earlier and more severe disruption of potassium secretory mechanisms in the distal nephron, out of proportion to the decline in GFR. These include conditions associated with hyporeninemic hypoaldosteronism, such as diabetes, and renal diseases that preferentially affect the distal nephron, such as obstructive uropathy and sickle cell nephropathy.

Hypokalemia is not common in CKD and usually reflects markedly reduced dietary potassium intake, especially in association with excessive diuretic therapy or concurrent GI losses. The use of potassium supplements and potassium-sparing diuretics may be risky in patients with impaired renal function and needs to be monitored closely.

Metabolic Acidosis Metabolic acidosis is a common disturbance in CKD. The majority of patients can still acidify the urine, but they produce less ammonia and, therefore, cannot excrete the quantity of protons required to maintain acid-base balance in most diets. Hyperkalemia, if present, further depresses ammonia production. The combination of hyperkalemia and hyperchloremic metabolic acidosis is often present, even at earlier stages of CKD, in patients with diabetic nephropathy or in those with predominant tubulointerstitial disease or obstructive uropathy. With further declining GFR, the total urinary net daily acid excretion may be severely limited to less than 30–40 mmol, and the accumulation of anions of retained organic acids can then lead to an anion-gap metabolic acidosis. Thus, the non-anion-gap metabolic acidosis seen in earlier stages of CKD may be complicated by the addition of an anion-gap metabolic acidosis as CKD progresses. In most patients, the metabolic acidosis is mild; the pH is rarely <7.32 and can usually be corrected with oral sodium bicarbonate supplementation. Studies have suggested that even modest degrees of metabolic acidosis