

FIGURE 6-33

Changes in urine acid-base composition during development, maintenance, and correction of vomiting-induced metabolic alkalosis. During acid removal from the stomach as well as early in the phase after vomiting (maintenance), an alkaline urine is excreted as acid excretion is suppressed, and bicarbonate excretion (in the company of sodium and, especially potassium; see Fig. 6-32) is increased, with the net acid excretion being negative (net alkali excretion). This acid-base profile moderates the steady-state level of the resulting alkalosis. In the steady state (late maintenance phase), as all filtered bicarbonate is reclaimed the pH of urine becomes acidic, and the net acid excretion returns to baseline. Provision of sodium chloride (NaCl) and potassium chloride (KCl) in the correction phase alkalizes the urine and suppresses the net acid excretion, as bicarbonaturia in the company of exogenous cations (sodium and potassium) supervenes [22,23]. HCO_3^- —bicarbonate ion.

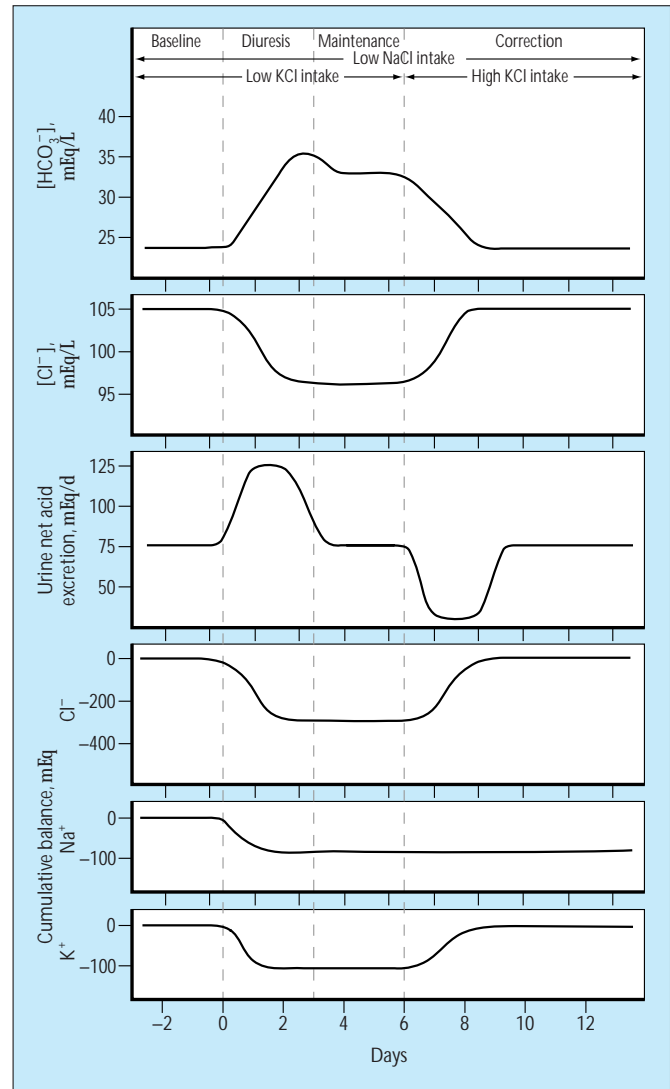
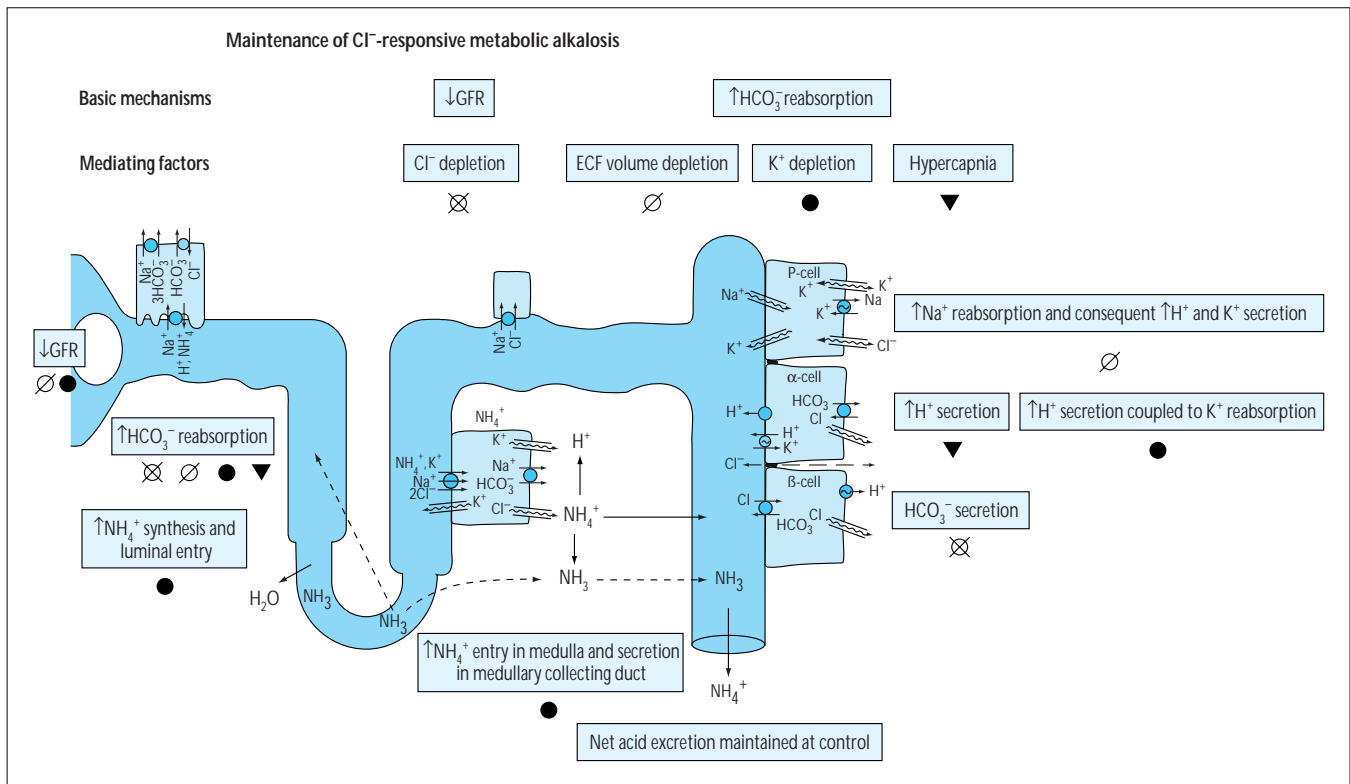


FIGURE 6-34

Changes in plasma anionic pattern, net acid excretion, and body electrolyte balance during development, maintenance, and correction of diuretic-induced metabolic alkalosis. Administration of a loop diuretic, such as furosemide, increases urine net acid excretion (largely in the form of ammonium) as well as the renal losses of chloride (Cl^-), sodium (Na^+), and potassium (K^+). The resulting hyperbicarbonatemia reflects both loss of excess ammonium chloride in the urine and an element of contraction (consequent to diuretic-induced sodium chloride [NaCl] losses) that limits the space of distribution of bicarbonate. During the phase after diuresis (maintenance), and as long as the low-chloride diet is continued, a new steady state is attained in which the plasma bicarbonate concentration ($[\text{HCO}_3^-]$) remains elevated, urine net acid excretion returns to baseline, and renal excretion of electrolytes matches intake. Addition of potassium chloride (KCl) in the correction phase repairs the chloride and potassium deficits, suppresses net acid excretion, and normalizes the plasma bicarbonate and chloride concentration ($[\text{Cl}^-]$) levels [23,24]. If extracellular fluid volume has become subnormal following diuresis, administration of NaCl is also required for repair of the metabolic alkalosis.

**FIGURE 6-35**

Maintenance of chloride-responsive metabolic alkalosis. Increased renal bicarbonate reabsorption frequently coupled with a reduced glomerular filtration rate are the basic mechanisms that maintain chloride-responsive metabolic alkalosis. These mechanisms have been ascribed to three mediating factors: chloride depletion itself, extracellular fluid (ECF) volume depletion, and potassium depletion. Assigning particular roles to

each of these factors is a vexing task. Notwithstanding, here depicted is our current understanding of the participation of each of these factors in the nephronal processes that maintain chloride-responsive metabolic alkalosis [22–24]. In addition to these factors, the secondary hypercapnia of metabolic alkalosis contributes importantly to the maintenance of the prevailing hyperbicarbonatemia [25].

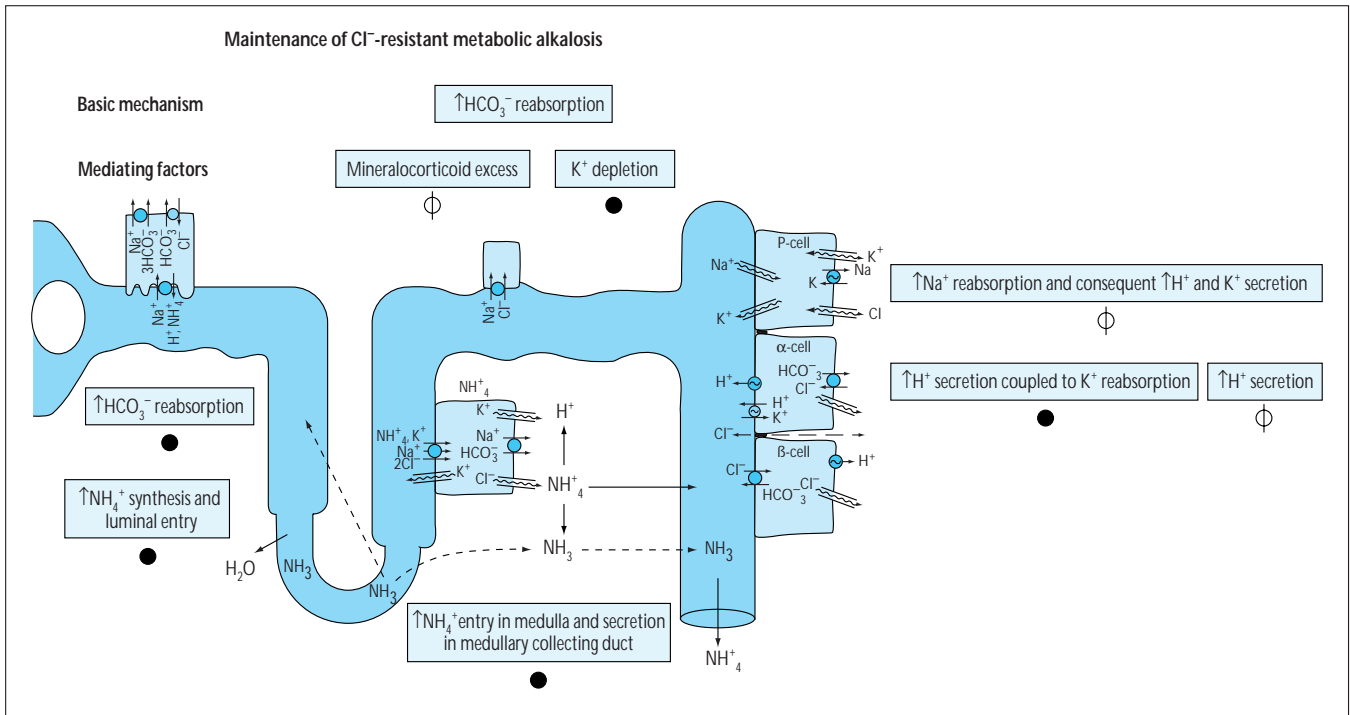


FIGURE 6-36

Maintenance of chloride-resistant metabolic alkalosis. Increased renal bicarbonate reabsorption is the sole basic mechanism that maintains chloride-resistant metabolic alkalosis. As its name implies, factors independent of chloride intake mediate the height-

ened bicarbonate reabsorption and include mineralocorticoid excess and potassium depletion. The participation of these factors in the nephronal processes that maintain chloride-resistant metabolic alkalosis is depicted [22–24, 26].

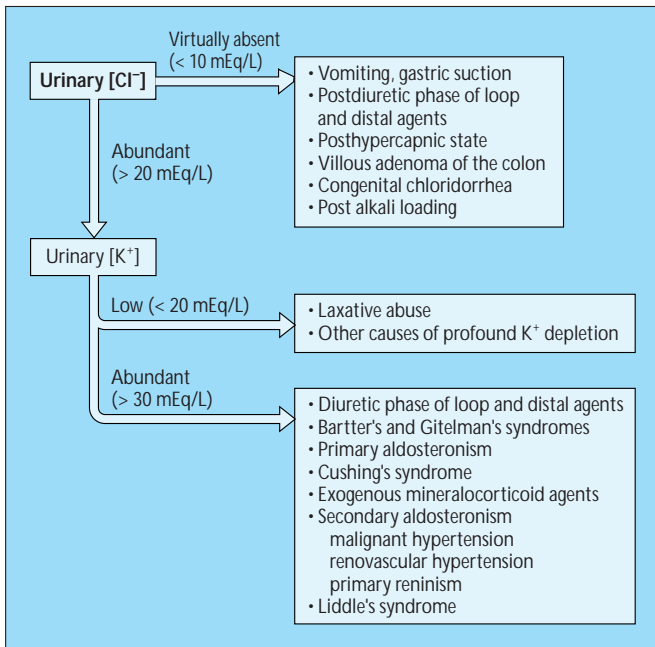


FIGURE 6-37

Urinary composition in the diagnostic evaluation of metabolic alkalosis. Assessing the urinary composition can be an important aid in the diagnostic evaluation of metabolic alkalosis. Measurement of urinary chloride ion concentration ([Cl⁻]) can help distinguish between chloride-responsive and chloride-resistant metabolic alkalosis. The virtual absence of chloride (urine [Cl⁻] < 10 mEq/L) indicates significant chloride depletion. Note, however, that this test loses its diagnostic significance if performed within several hours of administration of chloruretic diuretics, because these agents promote urinary chloride excretion. Measurement of urinary potassium ion concentration ([K⁺]) provides further diagnostic differentiation. With the exception of the diuretic phase of chloruretic agents, abundance of both urinary chloride and potassium signifies a state of mineralocorticoid excess [22].