in patients with edema and hypertension because of possible aggravation of these conditions.

METABOLIC ALKALOSIS

Metabolic alkalosis is established by an elevated arterial pH, an increase in the serum [HCO_3^-], and an increase in $Paco_2$ as a result of compensatory alveolar hypoventilation (Table 55-1). It is often accompanied by hypochloremia and hypokalemia. The elevation in arterial pH establishes the diagnosis because pH is decreased in respiratory acidosis, even though both have an elevated $Paco_2$. Metabolic alkalosis frequently occurs as a mixed acid-base disorder in association with either respiratory acidosis, respiratory alkalosis, or metabolic acidosis.

■ ETIOLOGY AND PATHOGENESIS

Metabolic alkalosis occurs as a result of net gain of [HCO_3^-] or loss of nonvolatile acid (usually HCl by vomiting) from the extracellular fluid. When vomiting causes loss of HCl from the stomach, HCO_3^- secretion cannot be initiated in the small bowel, and thus, HCO_3^- is retained in the extracellular fluid. Thus, vomiting or nasogastric suction is an example of the *generation stage* of metabolic alkalosis, in which the loss of acid typically causes alkalosis. Upon cessation of vomiting, the *maintenance stage* ensues because secondary factors prevent the kidneys from excreting HCO_3^- appropriately.

Maintenance of metabolic alkalosis, therefore, represents a failure of the kidneys to eliminate excess HCO_3^- from the extracellular compartment. The kidneys will retain, rather than excrete, the excess alkali and maintain the alkalosis if (1) volume deficiency, chloride deficiency, and K^+ deficiency exist in combination with a reduced GFR (associated with a low urine [Cl^-]) or (2) hypokalemia exists because of autonomous hyperaldosteronism (normal urine [Cl^-]). In the first example, saline-responsive metabolic alkalosis is corrected by extracellular fluid volume (ECFV) restoration