the degree of ketosis (see below). Excretion of ketoacids obligates the excretion of cations, such as Na^+ and K^+, contributing to volume depletion and Cl^- retention. In some circumstances, a mixed non-AG–high-AG acidosis may occur simultaneously and is recognized when the ΔHCO_3^- exceeds the ΔAG. It should be noted that bicarbonate therapy is rarely necessary in DKA except with extreme acidemia (pH <7.00) or if the patient is in shock. If administered, $NaHCO_3$ should be administered in only limited amounts because of the risk for cerebral edema. Patients with DKA are typically volume depleted and require fluid resuscitation with isotonic saline. Volume overexpansion should be avoided, however, because overly aggressive saline administration may cause hyperchloremic acidosis during or following treatment of DKA. Regular insulin should be administered IV as an initial bolus of 0.1 U/kg followed by an infusion of 0.1 U/kg/h until the AG returns to normal; see **Chap. 403** for more detail.

ALCOHOLIC KETOACIDOSIS (AKA) AKA is usually associated with chronic alcoholism, binge drinking, vomiting, abdominal pain, poor nutrition, and volume depletion. The glucose concentration is variable, and acidosis may be severe because of elevated ketones, predominantly β-hydroxybutyrate. The presence of a high-AG acidosis, in the absence of hyperglycemia, in a patient with chronic alcoholism suggests the diagnosis of AKA. Mixed acid-base disorders are common in AKA. Hypoperfusion may enhance lactic acid production (mixed high-AG acidosis), chronic respiratory alkalosis may accompany liver disease (mixed high-AG acidosis and respiratory alkalosis), and metabolic alkalosis can result from vomiting (mixed high-AG acidosis and metabolic alkalosis: ΔAG exceeds ΔHCO_3^-). As the circulation is restored by administration of IV fluids, the preferential accumulation of β-hydroxybutyrate is then shifted to acetoacetate. This explains the common clinical observation of an increasingly positive nitroprusside reaction (ketones) as the circulation is restored. The nitroprusside reaction can detect acetoacetic acid but not β-hydroxybutyrate, so that the degree of ketosis and ketonuria can not only change with therapy,