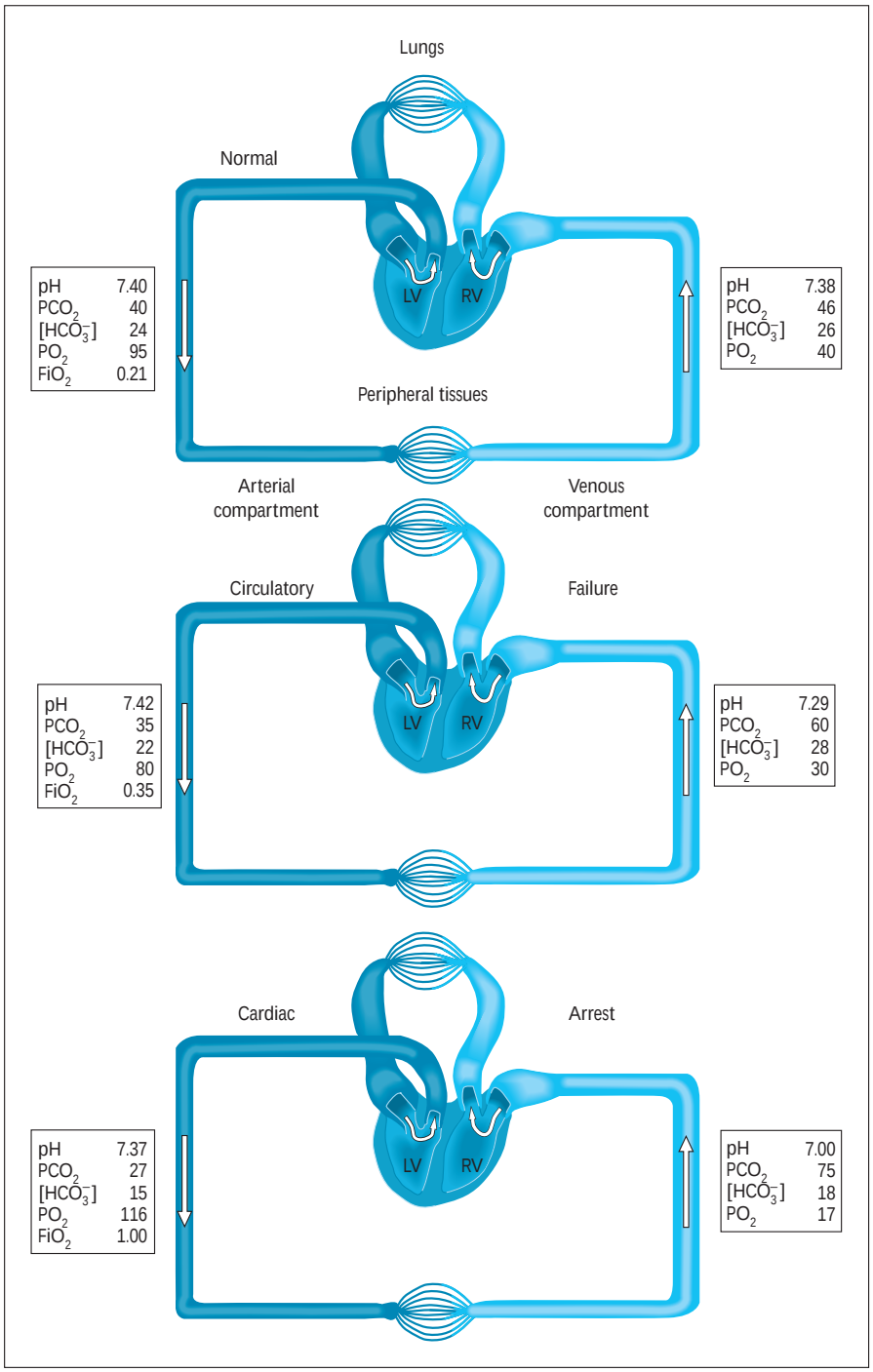

FIGURE 6-14

Pseudorespiratory alkalosis. This entity develops in patients with profound depression of cardiac function and pulmonary perfusion but relative preservation of alveolar ventilation. Patients include those with advanced circulatory failure and those undergoing cardiopulmonary resuscitation. The severely reduced pulmonary blood flow limits the amount of carbon dioxide delivered to the lungs for excretion, thereby increasing the venous carbon dioxide tension (PCO_2). In contrast, the increased ventilation-to-perfusion ratio causes a larger than normal removal of carbon dioxide per unit of blood traversing the pulmonary circulation, thereby giving rise to arterial hypocapnia [12,13]. Note a progressive widening of the arteriovenous difference in pH and PCO_2 in the two settings of cardiac dysfunction. The hypobicarbonatemia in the setting of cardiac arrest represents a complicating element of lactic acidosis. Despite the presence of arterial hypocapnia, pseudorespiratory alkalosis represents a special case of respiratory acidosis, as absolute carbon dioxide excretion is decreased and body carbon dioxide balance is positive. Furthermore, the extreme oxygen deprivation prevailing in the tissues might be completely disguised by the reasonably preserved arterial oxygen values. Appropriate monitoring of acid-base composition and oxygenation in patients with advanced cardiac dysfunction requires mixed (or central) venous blood sampling in addition to arterial blood sampling. Management of pseudorespiratory alkalosis must be directed at optimizing systemic hemodynamics [1,13].

Metabolic Acidosis

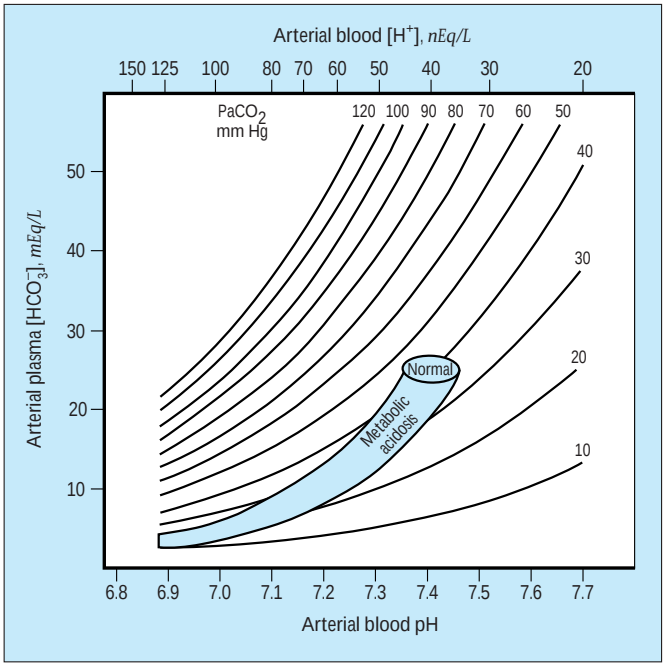

FIGURE 6-15

Ninety-five percent confidence intervals for metabolic acidosis. Metabolic acidosis is the acid-base disturbance initiated by a decrease in plasma bicarbonate concentration ($[HCO_3^-]$). The resultant acidemia stimulates alveolar ventilation and leads to the secondary hypocapnia characteristic of the disorder. Extensive observations in humans encompassing a wide range of stable metabolic acidosis indicate a roughly linear relationship between the steady-state decrease in plasma bicarbonate concentration and the associated decrement in arterial carbon dioxide tension ($PaCO_2$). The slope of the steady state $\Delta PaCO_2$ versus $\Delta[HCO_3^-]$ relationship has been estimated as approximately 1.2 mm Hg per mEq/L decrease in plasma bicarbonate concentration. Such empiric observations have been used for construction of 95% confidence intervals for graded degrees of metabolic acidosis, represented by the area in color in the acid-base template. The black ellipse near the center of the figure indicates the normal range for the acid-base parameters [3]. Assuming a steady state is present, values falling within the area in color are consistent with but not diagnostic of simple metabolic acidosis. Acid-base values falling outside the area in color denote the presence of a mixed acid-base disturbance [4]. $[H^+]$—hydrogen ion concentration.

SIGNS AND SYMPTOMS OF METABOLIC ACIDOSIS

Respiratory System	Cardiovascular System	Metabolism	Central Nervous System	Skeleton
Hyperventilation	Impairment of cardiac contractility, arteriolar dilation, venoconstriction, and centralization of blood volume	Increased metabolic demands	Impaired metabolism	Osteomalacia
Respiratory distress and dyspnea	Reductions in cardiac output, arterial blood pressure, and hepatic and renal blood flow	Insulin resistance	Inhibition of cell volume regulation	Fractures
Decreased strength of respiratory muscles and promotion of muscle fatigue	Sensitization to reentrant arrhythmias and reduction in threshold for ventricular fibrillation	Inhibition of anaerobic glycolysis	Progressive obtundation	
	Increased sympathetic discharge but attenuation of cardiovascular responsiveness to catecholamines	Reduction in adenosine triphosphate synthesis	Coma	
		Hyperkalemia		
		Increased protein degradation		

FIGURE 6-16

Signs and symptoms of metabolic acidosis. Among the various clinical manifestations, particularly pernicious are the effects of severe acidemia (blood pH < 7.20) on the cardiovascular system. Reductions in cardiac output, arterial blood pressure, and hepatic and renal blood flow can occur and life-threatening arrhythmias can develop. Chronic acidemia, as it occurs in untreated renal tubular acidosis and uremic acidosis, can cause calcium dissolution from the bone mineral and consequent skeletal abnormalities.

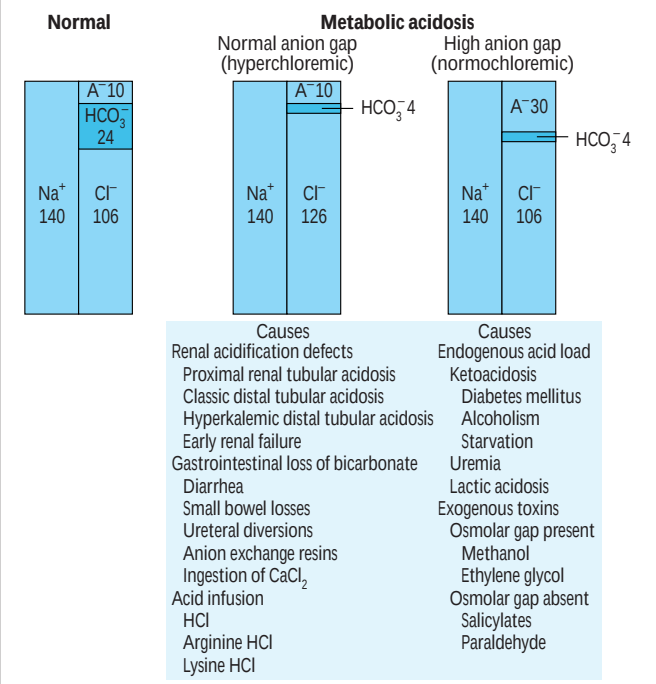

FIGURE 6-17

Causes of metabolic acidosis tabulated according to the prevailing pattern of plasma electrolyte composition. Assessment of the plasma unmeasured anion concentration (anion gap) is a very useful first step in approaching the differential diagnosis of unexplained metabolic acidosis. The plasma anion gap is calculated as the difference between the sodium concentration and the sum of chloride and bicarbonate concentrations. Under normal circumstances, the plasma anion gap is primarily composed of the net negative charges of plasma proteins, predominantly albumin, with a smaller contribution from many other organic and inorganic anions. The normal value of the plasma anion gap is 12 ± 4 (mean ± 2 SD) mEq/L, where SD is the standard deviation. However, recent introduction of ion-specific electrodes has shifted the normal anion gap to the range of about 6 ± 3 mEq/L. In one pattern of metabolic acidosis, the decrease in bicarbonate concentration is offset by an increase in the concentration of chloride, with the plasma anion gap remaining normal. In the other pattern, the decrease in bicarbonate is balanced by an increase in the concentration of unmeasured anions (*ie*, anions not measured routinely), with the plasma chloride concentration remaining normal.

Lactic acidosis

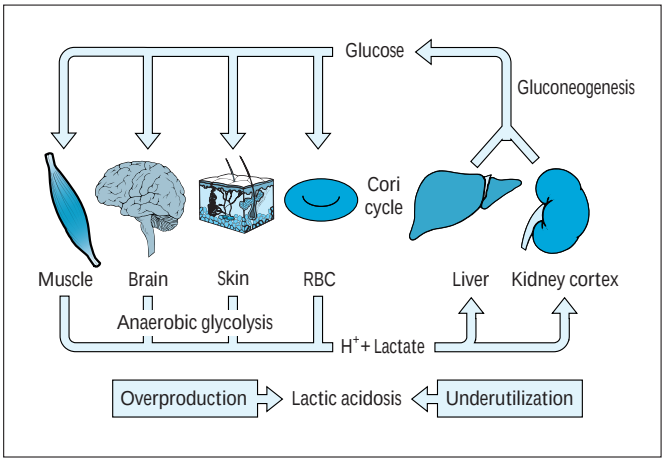

FIGURE 6-18

Lactate-producing and lactate-consuming tissues under basal conditions and pathogenesis of lactic acidosis. Although all tissues produce lactate during the course of glycolysis, those listed contribute substantial quantities of lactate to the extracellular fluid under normal aerobic conditions. In turn, lactate is extracted by the liver and to a lesser degree by the renal cortex and primarily is reconverted to glucose by way of gluconeogenesis (a smaller portion of lactate is oxidized to carbon dioxide and water). This cyclical relationship between glucose and lactate is known as the *Cori cycle*. The basal turnover rate of lactate in humans is enormous, on the order of 15 to 25 mEq/kg/d. Precise equivalence between lactate production and its use ensures the stability of plasma lactate concentration, normally ranging from 1 to 2 mEq/L. Hydrogen ions (H^+) released during lactate generation are quantitatively consumed during the use of lactate such that acid-base balance remains undisturbed. Accumulation of lactate in the circulation, and consequent lactic acidosis, is generated whenever the rate of production of lactate is higher than the rate of utilization. The pathogenesis of this imbalance reflects overproduction of lactate, underutilization, or both. Most cases of persistent lactic acidosis actually involve both overproduction and underutilization of lactate. During hypoxia, almost all tissues can release lactate into the circulation; indeed, even the liver can be converted from the premier consumer of lactate to a net producer [1,14].