TABLE 27-6 COMPENSATION IN ACID-BASE DISORDERS

DISORDER	COMPENSATORY CHANGES
Acute respiratory acidosis	For every 10 mm Hg rise in P_{CO_2}, the HCO_3^- increases by 1 mEq/L
Chronic respiratory acidosis	For every 10 mm Hg rise in P_{CO_2}, the HCO_3^- increases by 3.5 mEq/L
Acute respiratory alkalosis	For every 10 mm Hg fall in P_{CO_2}, the HCO_3^- decreases by 2 mEq/L
Chronic respiratory alkalosis	For every 10 mm Hg decrease in P_{CO_2}, the HCO_3^- decreases by 5 mEq/L
Metabolic acidosis	1.2 mm Hg decrease in P_{CO_2} for each 1 mEq/L fall in HCO_3^- $P_{CO_2} = HCO_3^- + 15$ P_{CO_2} resembles last two digits of pH
Metabolic alkalosis	P_{CO_2} increases by 0.7 mm Hg for each mEq/L HCO_3^-

HCO_3^- concentration falls by 2 mEq/L for every 10 mm Hg decrease in P_{CO_2}. Table 27-6 provides the expected compensatory responses for acid-base disorders.

In chronic respiratory alkalosis, the renal HCO_3^- reabsorptive capacity decreases, and there is a transient HCO_3^- diuresis. This process takes 2 to 3 days to fully manifest. After the new steady state is achieved, the HCO_3^- concentration has decreased by 5 mEq/L for each 10 mm Hg fall in the P_{CO_2}. A higher or lower value for the plasma HCO_3^- concentration suggests an additional metabolic disorder.

To defend ECF volume in the setting of increased urinary loss of $NaHCO_3$, the kidney retains NaCl. These changes are reflected in the serum electrolyte levels of patients with chronic respiratory alkalosis, in whom the Cl^- level is typically increased with respect to the serum Na^+ concentration. Another characteristic finding is an increase of 3 to 5 mEq/L in the serum anion gap. The increased gap results from the greater fixed negative charge on serum albumin and an increase in the serum lactate concentration. Lactate production is increased due to a stimulatory effect of high pH on phosphofructokinase, the rate-limiting step in the glycolytic pathway.

Treatment

Primary respiratory alkalosis is treated by correcting the underlying cause. A patient with anxiety-hyperventilation syndrome should be treated by providing reassurance. Rebreathing into a paper bag or any other closed system causes the P_{CO_2} to increase with each breath taken and leads to partial correction of hypocapnia and improvement of symptoms. In the rare case when there is no response to conservative management, sedatives can be used. In mechanically ventilated patients, the P_{CO_2} can be increased by raising the inspired carbon dioxide tension or by increasing the dead space of the ventilator circuit.

Correction of respiratory alkalosis may prove helpful in correcting arrhythmias in patients with underlying coronary disease. In contrast, caution is warranted in raising the P_{CO_2} in patients with brain injury because cerebral perfusion may increase and worsen intracranial pressure. Respiratory alkalosis frequently develops as a complication of hypoxia. Administration of oxygen or return to lower altitudes can reverse the respiratory alkalosis that develops in this setting.

RESPIRATORY ACIDOSIS

Definition

Respiratory acidosis develops as a result of ineffective alveolar ventilation. This acid-base disorder, which is also called *primary hypercapnia*, should be differentiated from secondary hypercapnia, which develops as a compensatory mechanism in the setting of primary metabolic alkalosis. Primary hypercapnia is clinically recognized by P_{aCO_2} levels greater than 45 mm Hg on arterial blood gas analysis. However, P_{aCO_2} levels of less than 45 mm Hg may still indicate respiratory acidosis if a primary metabolic acidosis is not adequately compensated by alveolar ventilation.

The development of respiratory acidosis is usually multifactorial. Major causes of carbon dioxide retention include diseases of or malfunction in any element of the respiratory system, including the central and peripheral nervous systems, respiratory muscles, thoracic cage, pleural space, airways, and lung parenchyma. Six factors should be considered in the differential diagnosis of acute and chronic respiratory acidosis: inhibition of the medullary respiratory center, disorders of the chest wall and respiratory muscles, airway obstruction, disorders affecting gas exchange across the pulmonary capillary, increased carbon dioxide production, and mechanical ventilation.

Clinical Presentation

Hypercapnic encephalopathy is a clinical syndrome that usually starts with irritability, headache, mental cloudiness, apathy, confusion, anxiety, and restlessness. It can progress to asterixis, transient psychosis, delirium, somnolence, and coma. Papilledema and other manifestations of increased intracranial pressure that are collectively named *pseudotumor cerebri* are occasionally observed in patients with acute or chronic hypercapnia. The increase in intracranial pressure is caused in part due by cerebral vasodilation resulting from acidemia.

Acute respiratory acidosis is typically much more symptomatic than acute metabolic acidosis because carbon dioxide diffuses and equilibrates across the blood-brain barrier much more rapidly than does HCO_3^-, resulting in a more rapid fall in cerebral spinal fluid and cerebral interstitial pH. Severe hypercapnia also can lead to decreased myocardial contractility, arrhythmias, and peripheral vasodilatation, particularly when the blood pH falls to less than 7.1.

Diagnosis

The diagnosis of primary respiratory acidosis is based on the finding of acidemia and hypercapnia on arterial blood gas analysis. Changes in the serum chemistries can aid in the diagnosis of respiratory acidosis.

Acute hypercapnia is associated with a shift of HCO_3^- out of red blood cells in exchange for Cl^-, a process called *red cell HCO_3^--Cl^- shift*. Acutely, the plasma HCO_3^- concentration increases by 1 mmol/L for each 10 mm Hg of elevation in the P_{aCO_2}. After 24 to 48 hours of hypercapnia, proximal tubular cells increase H^+ secretion, resulting in accelerated HCO_3^- reabsorption. The retention of $NaHCO_3$ leads to slight expansion of the ECF compartment and increases renal excretion of NaCl to return the volume level to normal. The net effect is increased