TABLE 66-7 RESPIRATORY ACID-BASE DISORDERS

I. Alkalosis
- A. Central nervous system stimulation
  1. Pain
  2. Anxiety, psychosis
  3. Fever
  4. Cerebrovascular accident
  5. Meningitis, encephalitis
  6. Tumor
  7. Trauma
- B. Hypoxemia or tissue hypoxia
  1. High altitude
  2. Pneumonia, pulmonary edema
  3. Aspiration
  4. Severe anemia
- C. Drugs or hormones
  1. Pregnancy, progesterone
  2. Salicylates
  3. Cardiac failure
- D. Stimulation of chest receptors
  1. Hemothorax
  2. Flail chest
  3. Cardiac failure
  4. Pulmonary embolism
- E. Miscellaneous
  1. Septicemia
  2. Hepatic failure
  3. Mechanical hyperventilation
  4. Heat exposure
  5. Recovery from metabolic acidosis

II. Acidosis
- A. Central
  1. Drugs (anesthetics, morphine, sedatives)
  2. Stroke
  3. Infection
- B. Airway
  1. Obstruction
  2. Asthma
- C. Parenchyma
  1. Emphysema
  2. Pneumoconiosis
  3. Bronchitis
  4. Adult respiratory distress syndrome
  5. Barotrauma
- D. Neuromuscular
  1. Poliomyelitis
  2. Kyphoscoliosis
  3. Myasthenia
  4. Muscular dystrophies
- E. Miscellaneous
  1. Obesity
  2. Hypoventilation
  3. Permissive hypercapnia

inhalational burn, or toxin injury. Chronic hypercapnia and respiratory acidosis occur in end-stage obstructive lung disease. Restrictive disorders involving both the chest wall and the lungs can cause respiratory acidosis because the high metabolic cost of respiration causes ventilatory muscle fatigue. Advanced stages of intrapulmonary and extrapulmonary restrictive defects present as chronic respiratory acidosis.

The diagnosis of respiratory acidosis requires the measurement of $Paco_2$ and arterial pH. A detailed history and physical examination often indicate the cause. Pulmonary function studies **(Chap. 306e)**, including spirometry, diffusion capacity for carbon monoxide, lung volumes, and arterial $Paco_2$ and O_2 saturation, usually make it possible to determine if respiratory acidosis is secondary to lung disease. The workup for nonpulmonary causes should include a detailed drug history, measurement of hematocrit, and assessment of upper airway, chest wall, pleura, and neuromuscular function.

TREATMENT RESPIRATORY ACIDOSIS

The management of respiratory acidosis depends on its severity and rate of onset. Acute respiratory acidosis can be life-threatening, and measures to reverse the underlying cause should be undertaken simultaneously with restoration of adequate alveolar ventilation. This may necessitate tracheal intubation and assisted mechanical ventilation. Oxygen administration should be titrated carefully in patients with severe obstructive pulmonary disease and chronic CO_2 retention who are breathing spontaneously **(Chap. 314)**. When oxygen is used injudiciously, these patients may experience progression of the respiratory acidosis. Aggressive and rapid correction of hypercapnia should be avoided, because the falling $Paco_2$ may provoke the same complications noted with acute respiratory alkalosis (i.e., cardiac arrhythmias, reduced cerebral perfusion, and seizures). The $Paco_2$ should be lowered gradually in chronic respiratory acidosis, aiming to restore the $Paco_2$ to baseline levels and to provide sufficient Cl^- and K^+ to enhance the renal excretion of HCO_3^-.

Chronic respiratory acidosis is frequently difficult to correct, but measures aimed at improving lung function **(Chap. 314)** can help some patients and forestall further deterioration in most.

RESPIRATORY ALKALOSIS

Alveolar hyperventilation decreases $Paco_2$ and increases the $HCO_3^-/Paco_2$ ratio, thus increasing pH (Table 66-7). Nonbicarbonate cellular buffers respond by consuming HCO_3^-. Hypocapnia develops when a sufficiently strong ventilatory stimulus causes CO_2 output in the lungs to exceed its metabolic production by tissues. Plasma pH and $[HCO_3^-]$ appear to vary proportionately with $Paco_2$ over a range from 40–15 mmHg. The relationship between arterial $[H^+]$ concentration and $Paco_2$ is ~0.7 mmol/L per mmHg (or 0.01 pH unit/mmHg), and that for plasma $[HCO_3^-]$ is 0.2 mmol/L per mmHg. Hypocapnia sustained for >2–6 h is further compensated by a decrease in renal ammonium and titratable acid excretion and a reduction in filtered HCO_3^- reabsorption. Full renal adaptation to respiratory alkalosis may take several days and requires normal volume status and renal function. The kidneys appear to respond directly to the lowered $Paco_2$ rather than to alkalosis per se. In chronic respiratory alkalosis a 1-mmHg decrease in $Paco_2$ causes a 0.4-to 0.5-mmol/L drop in $[HCO_3^-]$ and a 0.3-mmol/L decrease (or 0.003 increase in pH) in $[H^+]$.

The effects of respiratory alkalosis vary according to duration and severity but are primarily those of the underlying disease. Reduced cerebral blood flow as a consequence of a rapid decline in $Paco_2$ may cause dizziness, mental confusion, and seizures, even in the absence of hypoxemia. The cardiovascular effects of acute hypocapnia in the conscious human are generally minimal, but in the anesthetized or mechanically ventilated patient, cardiac output and blood pressure may fall because of the depressant effects of anesthesia and positive-pressure ventilation on heart rate, systemic resistance, and venous return. Cardiac arrhythmias may occur in patients with heart disease as a result of changes in oxygen unloading by blood from a left shift in the hemoglobin-oxygen dissociation curve (Bohr effect). Acute respiratory alkalosis causes intracellular shifts of Na^+, K^+, and PO_4^{2-} and reduces free $[Ca^{2+}]$ by increasing the protein-bound fraction. Hypocapnia-induced hypokalemia is usually minor.

Chronic respiratory alkalosis is the most common acid-base disturbance in critically ill patients and, when severe, portends a poor prognosis. Many cardiopulmonary disorders manifest respiratory alkalosis