system is usually the dominant concern. **ECG changes** begin with peaking of the T waves. As the potassium level increases, an increased P-R interval, flattening of the P wave, and widening of the QRS complex occur; this eventually can progress to ventricular fibrillation. Asystole also may occur. Some patients have paresthesias, weakness, and tingling, but cardiac toxicity usually precedes these clinical symptoms.

Diagnosis

The etiology of hyperkalemia is often readily apparent. Spurious hyperkalemia is common in children, so a repeat potassium level is often appropriate. If there is a significant elevation of the white blood cells or platelets, the repeat sample should be from plasma that is evaluated promptly. The history initially should focus on potassium intake, risk factors for transcellular shifts of potassium, medications that cause hyperkalemia, and the presence of signs of renal insufficiency, such as oliguria or an abnormal urinalysis. Initial laboratory evaluation should include serum creatinine and assessment of acid-base status. Many causes of hyperkalemia, such as renal insufficiency and aldosterone insufficiency or resistance, cause a metabolic acidosis. Cell destruction, as seen in rhabdomyolysis or tumor lysis syndrome, can cause concomitant hyperphosphatemia, hyperuricemia, and an elevated serum lactate dehydrogenase.

Treatment

The plasma potassium level, the ECG, and the risk of the problem worsening determine the aggressiveness of the therapeutic approach. A high serum potassium level with ECG changes requires more vigorous treatment. An additional source of concern is a patient with increasing plasma potassium despite minimal intake. This situation can occur if there is cellular release of potassium (tumor lysis syndrome), especially in the setting of diminished excretion (renal failure).

The first action in a child with a concerning elevation of plasma potassium is to stop all sources of additional potassium (oral and IV). If the potassium level is greater than 6.5 mEq/L, an ECG should be obtained to help assess the urgency of the situation. Therapy of hyperkalemia has two basic goals:

1. Prevent life-threatening arrhythmias.
2. Remove potassium from the body (Table 36-3).

Table 36-3 Treatment of Hyperkalemia

Rapidly decrease the risk of life-threatening arrhythmias
Shift potassium intracellularly
Sodium bicarbonate administration (IV)
Insulin and glucose (IV)
β-Agonist (albuterol via nebulizer)
Cardiac membrane stabilization
IV calcium
Remove potassium from the body
Loop diuretic (IV or PO)
Sodium polystyrene (PO or rectal)
Dialysis

IV, Intravenous; *PO,* oral.

Treatments that acutely prevent arrhythmias all work quickly (within minutes), but do not remove potassium from the body.

Long-term management of hyperkalemia includes reducing intake via dietary changes and eliminating or reducing medications that cause hyperkalemia. Some patients require medications, such as sodium polystyrene sulfonate and loop or thiazide diuretics, to increase potassium losses. The disorders due to a deficiency in aldosterone respond to replacement therapy with fludrocortisone, a mineralocorticoid.

Chapter 37

ACID-BASE DISORDERS

Close regulation of pH is necessary for cellular enzymes and other metabolic processes, which function optimally at a normal pH (7.35 to 7.45). Chronic, mild derangements in acid-base status may interfere with normal growth and development, whereas acute, severe changes in pH can be fatal. Control of acid-base balance depends on the kidneys, the lungs, and intracellular and extracellular buffers.

The lungs and the kidneys maintain a normal acid-base balance. Carbon dioxide (CO_2) generated during normal metabolism is a weak acid. The lungs prevent an increase in the partial pressure of CO_2 (P_{CO_2}) in the blood by excreting the CO_2. Production of CO_2 varies depending on the body's metabolic needs. The rapid pulmonary response to changes in CO_2 concentration occurs via central sensing of the P_{CO_2} and a subsequent increase or decrease in ventilation to maintain a normal P_{CO_2} (35 to 45 mm Hg).

The kidneys excrete endogenous acids. An adult normally produces about 1 to 2 mEq/kg/day of hydrogen ions, whereas a child produces 2 to 3 mEq/kg/day. The hydrogen ions from endogenous acid production are neutralized by bicarbonate, potentially causing the bicarbonate concentration to fall. The kidneys regenerate this bicarbonate by secreting hydrogen ions, maintaining the serum bicarbonate concentration in the normal range (20 to 28 mEq/L).

CLINICAL ASSESSMENT OF ACID-BASE DISORDERS

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Acidemia
Alkalemia

Acidemia is a pH below normal (<7.35), and **alkalemia** is a pH above normal (>7.45). **Acidosis** is a pathologic process that causes an increase in the hydrogen ion concentration, and **alkalosis** is a pathologic process that causes a decrease in the hydrogen ion concentration. A **simple acid-base disorder** is a single primary disturbance. During a simple metabolic disorder, there