

monitoring of cold-induced diuresis and can provide an ancillary approach for temperature monitoring. Dehydration is encountered commonly with chronic hypothermia, and most patients benefit from an intravenous or intraosseous crystalloid bolus. Normal saline is preferable to lactated Ringer's solution, as the liver in hypothermic patients inefficiently metabolizes lactate. The placement of a pulmonary artery catheter can cause perforation of the less compliant pulmonary artery. Insertion of a central venous catheter deeply into the cold right atrium should be avoided since this procedure, similar to transvenous pacing, can precipitate refractory arrhythmias.

Arterial blood gases should *not* be corrected for temperature (**Chap. 55**). An uncorrected pH of 7.42 and a P_{CO_2} of 40 mmHg reflect appropriate alveolar ventilation and acid-base balance at any core temperature. Acid-base imbalances should be corrected gradually, since the bicarbonate buffering system is inefficient. A common error is overzealous hyperventilation in the setting of depressed CO_2 production. When the P_{CO_2} decreases by 10 mmHg at 28°C (82°F), it doubles the pH increase of 0.08 that occurs at 37°C (99°F).

The severity of anemia may be underestimated because the hematocrit increases 2% for each 1°C drop in temperature. White blood cell sequestration and bone marrow suppression are common, potentially masking an infection. Although hypokalemia is more common in chronic hypothermia, hyperkalemia also occurs; the expected electrocardiographic changes are often obscured by hypothermia. Patients with renal insufficiency, metabolic acidosis, or rhabdomyolysis are at greatest risk for electrolyte disturbances.

Coagulopathies are common because cold inhibits the enzymatic reactions required for activation of the intrinsic cascade. In addition, thromboxane B_2 production by platelets is temperature dependent, and platelet function is impaired. The administration of platelets and fresh-frozen plasma is therefore not effective. Coagulation studies can be deceptively normal and contrast with the observed *in vivo* coagulopathy. This contradiction occurs because all coagulation tests are routinely performed at 37°C (99°F), and the enzymes are thus rewarmed.