

observation for clinical signs of uremia and determination of the blood urea nitrogen (BUN) and creatinine (uremic acidosis); (5) inspection of the urine for oxalate crystals (ethylene glycol ingestion); and (6) recognition of the numerous clinical settings in which lactate levels may be increased (hypotension, shock, cardiac failure, leukemia, cancer, and drug or toxin ingestion).

Lactic Acidosis An increase in plasma L-lactate may be secondary to poor tissue perfusion (type A)—circulatory insufficiency (shock, cardiac failure), severe anemia, mitochondrial enzyme defects, and inhibitors (carbon monoxide, cyanide)—or to aerobic disorders (type B)—malignancies, nucleoside analogue reverse transcriptase inhibitors in HIV, diabetes mellitus, renal or hepatic failure, thiamine deficiency, severe infections (cholera, malaria), seizures, or drugs/toxins (biguanides, ethanol, and the toxic alcohols: ethylene glycol, methanol, or propylene glycol). Unrecognized bowel ischemia or infarction in a patient with severe atherosclerosis or cardiac decompensation receiving vasopressors is a common cause of lactic acidosis in elderly patients. Pyroglutamic acidemia may occur in critically ill patients receiving acetaminophen, which causes depletion of glutathione and accumulation of 5-oxoproline. D-Lactic acid acidosis, which may be associated with jejunoileal bypass, short bowel syndrome, or intestinal obstruction, is due to formation of D-lactate by gut bacteria.

APPROACH TO THE PATIENT

L-Lactic Acid Acidosis

The overarching goal of treatment is to correct the underlying condition that disrupts lactate metabolism; tissue perfusion should be restored when inadequate, but vasoconstrictors should be avoided, or used cautiously, because they may worsen tissue perfusion. Alkali therapy is generally advocated for acute, severe acidemia (pH <7.00) to improve cardiovascular function. However, NaHCO₃ therapy may paradoxically depress cardiac performance