Metabolic Acidosis in Cardiac Surgical Patients

Metabolic acidosis is a common finding in the perioperative period among cardiac surgical patients. Although mild metabolic acidosis is common, severe or persistent metabolic acidosis is a nonspecific sign of tissue ischemia or hypoperfusion and associated with an unfavorable prognosis (1-3). In the absence of pre-existing acid-base disorders, acquired metabolic acidosis during cardiac surgery is almost always caused by excessive acid production that overwhelms the buffering capacity of the body and exceeds the ability of the kidneys and lung to eliminate the excess acid. The detection of metabolic acidosis should prompt a search for sites of tissue ischemia and initiation of medical interventions to improve tissue perfusion. Despite the frequency of this problem, considerable controversy exists regarding the physiologic consequences of acute metabolic acidosis and the role of sodium bicarbonate for the treatment of this problem.

The Physiologic Consequences of Metabolic Acidosis

Several of the physiologic consequences of metabolic acidosis are well established. Metabolic acidosis causes a decrease in the pH within the central nervous system that increases respiratory drive to increase minute ventilation and decrease arterial $P_aCO_2$. The normal respiratory response to metabolic acidosis is an arterial $P_aCO_2$ (mm Hg) = 1.5 [$HCO_3^-$] ± 2, but the effects of metabolic acidosis on respiratory drive may be masked by general anesthesia, controlled mechanical ventilation, or cardiopulmonary bypass. Metabolic acidosis also causes a right shift of the oxygen-hemoglobin dissociation curve to favor the unloading of oxygen to tissues.

The precise cardiovascular consequences of metabolic acidosis in the clinical setting are more difficult to establish because it is not possible to separate the independent cardiovascular actions of acidosis from the cardiovascular effects of the underlying conditions that produce acidosis. Laboratory investigations suggest that severe metabolic acidosis with pH values less than 7.2 cause the release of endogenous catecholamines, direct myocardial depression, a decreased responsiveness of the myocardium to catecholamines, arterial vasodilation, and even bradycardia at extremely low pH values (4). Based on these laboratory findings, a pH value of 7.2 is commonly considered the clinical threshold for treatment with alkalinizing agents such as sodium bicarbonate. Because the cardiovascular actions of metabolic acidosis depend upon many factors, the precise cardiovascular responses in among individual patients may vary.
over time depending upon pre-existing conditions, ongoing drug therapy, and endogenous sympathoadrenal function.

**Metabolic Acidosis and the Cardiopulmonary Bypass (CPB) Pump Prime**

The rapid expansion of plasma volume with crystalloid solution upon the initiation of CPB produces may contribute to metabolic acidosis (5-9). The metabolic acidosis caused by the CPB prime is associated with a decrease in the pH, a decrease in $[HCO_3^-]$, a decrease in the base excess, an increase in $[Cl^-]$, and a decreased or normal anion gap ([Na$^+$]-[$Cl^-$]-[HCO$_3^-$]). Explanations for the acute non-anion gap acidosis caused by CPB include dilution of the plasma bicarbonate pool, hyperchloremia caused by the administration of a chloride solution at supra-physiologic concentrations (normal saline), hemodilution-induced decrease in plasma protein concentration (that act as weak acids), or the addition of unmeasured anions in the CPB prime solution (lactate, gluconate, or acetate). Acute non-anion gap metabolic acidosis caused by acute hemodilution has also been reported in response to rapid volume expansion with normal saline and acute normovolemic hemodilution with 5% albumin or 6% hydroxyethyl starch (10-11). The metabolic acidosis caused by acute plasma dilution accompanying the onset of CPB can be attenuated by priming the CPB circuit with solutions containing physiologic concentrations of bicarbonate or with solutions containing lactate to balance the concentration of chloride. The use of balanced crystalloid solutions containing lactate can increase plasma lactate concentrations during CPB.

**Lactic Acidosis**

Lactate is a metabolic product of glycolysis and accumulates when the body must generate ATP in the absence of oxygen. Lactate is buffered by bicarbonate and is mostly oxidized in the liver, but can also be cleared to a lesser extent by renal excretion or gluconeogenesis. Normal arterial concentrations of lactate are less than 1.5 mM per liter. Plasma concentrations of lactate in the range of 4-5 mM per liter or greater will produce lactic acidosis with an accompanying decrease in serum bicarbonate associated with an increased anion gap. The most common cause of lactic acidosis (type A) is overproduction as a consequence of hypoperfusion, hypoxemia, circulatory failure, malperfusion, severe anemia, hypotension, sepsis, or carbon monoxide poisoning (12). The reperfusion of ischemic tissue or a period of circulatory arrest during CPB will reliably produce lactic acidosis in cardiac surgical patients. Lactic acidosis can also occur in the absence of hypoperfusion or hypoxia (type B) as a consequence of liver disease, diabetes mellitus, hypoglycemia, renal failure, malignancies, certain drugs, thiamine deficiency, metabolic toxins, and hereditary defects. Epinephrine increases glycolysis and inhibits the oxidation of pyruvate and can contribute to increased blood lactate levels. Metformin has been implicated in lactic acidosis through its action on inhibiting oxidative metabolism, gluconeogenesis, and the gastrointestinal absorption of glucose (13). Lactic acidosis is also a nonspecific sign of nitroprusside toxicity in cardiac surgical patients. The administration of buffered electrolyte solutions that contain lactate such as Lactated Ringer’s solution or Hartmann’s solution will also increase arterial lactate concentrations. As a consequence, lactic acidosis is a relatively common finding among cardiac surgical patients with
multiple potential etiologies and typically generates a common diagnostic and treatment dilemma in the perioperative management of patients undergoing cardiac operations.

The Treatment of Lactic Acidosis

The definitive treatment of lactic acidosis caused by tissue ischemia, hypoperfusion, shock, or reperfusion should be the identification of the source of lactic acidosis and therapeutic interventions to improve perfusion and oxygen delivery to ischemic tissue. Restoration of tissue perfusion will decrease the rate of lactic acid production and restoration of hepatic and renal function will increase the rate of lactic acid oxidation and excretion. Despite appropriate medical and surgical therapy, the source of lactic acidosis cannot always be identified, normal hepatic and renal function may be impaired, and patients may develop severe metabolic acidosis that may compromise cardiovascular function. For this reason, immediate therapy to attenuate the deleterious effects of severe acidosis is often justified during the course of treatment. The administration of sodium bicarbonate is commonly prescribed as a supportive measure. Although sodium bicarbonate is commonly administered in the clinical setting for this purpose and is effective at increasing arterial pH and plasma bicarbonate concentration, considerable controversy exists regarding its efficacy in the setting of lactic acidosis (14-15). The administration of sodium bicarbonate is associated with an increase arterial CO₂, decrease plasma ionized Ca²⁺, and hypernatremia. Alkalinization of the plasma may also decrease intracellular pH, increase lactic acid production, and produce a leftward shift in the oxygen-hemoglobin dissociation curve that may decrease unloading of oxygen to tissues. Sodium bicarbonate solutions are very hypertonic compared to serum and the accumulation of solute will lead to hypernatremia or volume overload. Clinical studies have yet to demonstrate that sodium bicarbonate administration for severe metabolic acidosis improves survival (16-17). As an alternative to sodium bicarbonate, hemodialysis or ultrafiltration has also been reported as an effective means of clearing plasma lactate and may even mask lactic acidosis in critically ill patients (18).

References: