The effects of acidosis on cardiovascular function

LYNETTE A. JEFFERS, CRNA
Cleveland, Ohio

The author provides a review of the effects of acidosis on the cardiovascular system and considerations for the anesthetist dealing with acid-base imbalances.

Acidosis has long been thought to depress cardiac performance and reduce myocardial responsiveness to catecholamines. The normal arterial blood pH is 7.35 to 7.45 and any significant deviation from the norm can cause serious life-threatening disturbances throughout the body. The development of acidosis results from either a loss of base or gain of strong acid in the extracellular fluid. Small changes in plasma pH reflect large changes in hydrogen ion concentration and can affect vital areas such as myocardial function, peripheral vascular changes and cellular responses to endogenous and exogenous chemical compounds. This article will review the effects of acidosis on the function of the cardiovascular system.

Many aspects of cell metabolism are acutely pH-dependent, and translocation of substances across cellular membranes may be affected in a number of ways by pH changes both intracellularly and extracellularly. The concentration of hydrogen ions (H+) in extracellular fluid is small relative to other ions such as sodium, potassium or chloride. Yet small changes in plasma H+ may cause large alterations in organ function. The reduction of cardiac contractility associated with an acidosis is determined by the fall of pH in the intracellular fluid, which affects the function of many organelles within the cardiac cell. The effects of H+ may be direct or indirect. Direct refers to the ability of H+ to alter intracellular biochemical processes such as membrane excitability and energy production. Indirect effects refer to alterations in blood flow and influences on the endocrine system. Indirect actions may have more profound effects than direct actions.

pH and catecholamines

Acidosis is a strong stimulant of the sympathoadrenal system, evoking an increase in sympathetic activity. Circulating catecholamines are released from the adrenal glands and from nerve endings as a direct effect of increased H+ ion concentration. This stimulatory effect is a protective mechanism against the negative inotropic effects of acidosis on the myocardium. The direct depressive effect of acidosis on the contractile strength of the heart muscle is counterbalanced by the positive inotropic stimulus resulting from increased catecholamine release. This stimulatory effect of catecholamines is sufficient to offset the direct negative inotropic effects of moderate acidosis, but does not overcome the effects of more severe acidosis.

The immediate effects of the stimulation of the sympathoadrenal system are valuable, but tachycardia is induced, thus increasing myocardial work and oxygen demand. This can be especially
The catecholamine-releasing effect of acidosis is complicated by the fact that the pharmacologic activity of catecholamines is lessened by acidosis. In a lowered pH, catecholamines become chemically inactive and ineffective. This has clinical importance in that acidosis, resulting from a cardiac arrest, must often be corrected first to permit exogenously administered catecholamines to be therapeutically effective.

**Effects of pH on the vascularity**

Acidosis has been shown to have profound effects on the vascularity resulting in peripheral vasodilation, increased pulmonary vascular resistance and increased intravascular clotting. The direct effect of acidosis on most systemic vascular beds is to relax smooth muscle, thereby causing vasodilation. Vasodilation, in turn, influences cardiac contractility by altering preload and afterload. As previously stated, the catecholamine response to acidosis promotes vascular constriction, but the direct vasodilating effect of acidosis on the vascularity may reduce the vasoconstrictor effect of catecholamines. Ultimately, vascular changes depend upon the vascular reactivity to catecholamines influenced by the patient's age, state of health and state of vascular tone. In severe acidosis, the peripheral vessels become non-responsive to catecholamines with resulting dilation. This depression of the circulation has been shown to increase intravascular clotting due to sludging of blood, which perpetuates the spiraling effects of shock.

In contrast to its effect on the vascular beds, acidosis is a pulmonary vasoconstrictor with alkalosis having the opposite effect. The mechanism for constriction is unclear, but studies have shown that induced metabolic acidosis results in a large reduction of total systemic resistance with pulmonary vascular resistance greatly increased.

The pharmacological treatment of hypotension is facilitated by a restoration of a normal pH. The rationale behind the administration of sodium bicarbonate prior to injection of epinephrine in the treatment of cardiac arrest is also based on this relationship, since the primary action of epinephrine is to increase vascular resistance rather than beta-adrenergic stimulation of the heart.

**Effects of pH on the heart**

Acidosis can alter the contractility of the heart in two different forms. First, the heart is affected by systemic acidosis. Second, an acidosis develops locally in the myocardium during ischemia. The former is extracellular and the latter is intracellular. The reduction of contractility associated with an acidosis is determined by the fall of pH intracellularly, which affects the organelles within the cardiac cell. The normal pH of cardiac muscle is between 6.9 and 7.2.

The dominant mechanism for the reduction of contractility is competitive inhibition of the slow calcium current by hydrogen ions. The slow calcium current initiates cardiac contraction and contributes to the action potential. The major effect of acidosis on the action potential of ventricular muscle is to cause a lengthening owing to inhibition of potassium exchange and as a consequence of a reduced calcium current. The reduced contractility is dependent upon the severity of the acidosis. Beta-adrenergic agents are powerful stimulants of the myocardial slow channel current. Patients receiving beta-blockers may be more susceptible to the myocardial effects of acidosis.

The decrease in cardiac muscle tension at any given external pH is always greater in respiratory acidosis than in metabolic acidosis. A respiratory acidosis causes a rapid fall in contractility because carbon dioxide diffuses rapidly into a cell, whereas in a metabolic acidosis, the flux of bicarbonate (HCO₃⁻) and hydrogen (H⁺) ions into the cell is smaller over the same period of time. There is a much greater permeability of the cell membrane to CO₂.

Another important factor is that acidosis is commonly associated with an increased plasma potassium concentration, which reduces contractility and may also cause incoordinated ventricular contraction by slowing the spread of excitation across the ventricles.

The chronotropic response to acidosis varies depending upon the balance between sympathetic and parasympathetic tone. Acidosis does enhance the duration of asystole secondary to increased vagal tone. Arrhythmias commonly accompany acid-base imbalances. Hypercapnia mimics the effects of epinephrine administration, producing atrial, nodal and ventricular extrasystoles.

Sympathetic response is an important factor in attenuating the depression of acidosis on cardiac function. Acidosis depresses cardiac performance and, in addition, reduces myocardial responsiveness to catecholamines. Prime emphasis is placed on the need to correct acidosis before improved cardiac function and responsiveness to sympathomimetics can be achieved.
Considerations for the anesthetist

Treatment of metabolic acidosis involves removal of the cause of the accumulation of acids in the circulation. The intravenous administration of sodium bicarbonate is indicated if metabolic acidosis is associated with myocardial depression or cardiac dysrrhythmias. A commonly used formula to calculate the dose of sodium bicarbonate is based on the deviation of the plasma concentration of bicarbonate ions from the normal value of 24 mEq/L, the percent of body mass that is extracellular fluid (approximately 20%), and the ideal body weight. The best approach is to administer about one-half of the calculated dose of sodium bicarbonate, followed by a repeat measurement of the arterial pH to evaluate the impact of therapy.8

\[
\text{Sodium bicarbonate} = \text{body weight} \times \text{plasma bicarbonate ion concentration} \times \text{extracellular fluid volume as a fraction of body mass (0.2)}.
\]

In conclusion, acidosis has multiple effects on the cardiovascular system and its functions. An understanding of these effects is of primary importance to the anesthetist who must deal with a patient who presents with an acid-base imbalance.

REFERENCES

AUTHOR
Lynette A. Jeffers, CRNA, received her diploma in nursing from Fairview General Hospital School of Nursing, Cleveland, Ohio. She is a 1984 graduate of the Mt. Sinai School of Nurse Anesthesia, Cleveland, Ohio. She is completing her BA in nurse anesthesia at Ursuline College, Pepper Pike, Ohio. Currently, she is a staff anesthetist and clinical instructor at Mt. Sinai Medical Center, Cleveland, Ohio. This article was written when she was a senior nurse anesthesia student.