clinical investigations in critical care

Sodium Bicarbonate for the Treatment of Lactic Acidosis*

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Lactic acidosis often challenges the intensivist and is associated with a strikingly high mortality. Treatment involves discerning and correcting its underlying cause, ensuring adequate oxygen delivery to tissues, reducing oxygen demand through sedation and mechanical ventilation, and (most controversially) attempting to alkalinize the blood with IV sodium bicarbonate. Here we review the literature to answer the following questions: Is a low pH bad? Can sodium bicarbonate raise the pH *in vivo*? Does increasing the blood pH with sodium bicarbonate have any salutary effects? Does sodium bicarbonate have negative side effects? We find that the oft-cited rationale for bicarbonate use, that it might ameliorate the hemodynamic depression of metabolic acidemia, has been disproved convincingly. Further, given the lack of evidence supporting its use, we cannot condone bicarbonate administration for patients with lactic acidosis, regardless of the degree of acidemia. *(CHEST 2000; 117:260–267)*

Key words: acid-base; acidosis; alkalinizing therapy; bicarbonate; lactic acidosis; sodium bicarbonate

Abbreviations: DCA = dichloroacetate; DKA = diabetic ketoacidosis; SID = strong ion difference

L actic acidosis, defined as a lactate concentration > 5 mmol/L and a pH < 7.35, commonly complicates critical illness. Its causes are legion, including sepsis, cardiogenic shock, severe hypoxemia, hepatic failure, and intoxication. Many of these share reduced delivery of oxygen to cells or impaired use of oxygen in mitochondria, yet some are based in more complex derangements. For example, the lactic acidosis of sepsis is poorly understood but probably cannot be explained simply by tissue hypoxia, at least at the level of the whole body. Treatment of lactic acidosis involves discerning and correcting its underlying cause, ensuring adequate oxygen delivery to tissues, reducing oxygen demand through sedation and mechanical ventilation, and (in some ICUs) attempting to alkalinize the blood with IV sodium bicarbonate. Even in the face of maximal supportive therapy, lactic acidosis is associated with a mortality of 60 to 90%.[1–4]

Although the use of sodium bicarbonate for the treatment of metabolic acidosis has been debated most heavily in the past 15 years, its use was questioned as far back as 1934.[5] Nevertheless, it is still considered standard therapy and recommended in many textbooks and review articles,[6–8] despite the lack of relevant clinical data supporting its effectiveness. Here we review the animal and human studies that bear on this complex, yet common, clinical conundrum.

We will not address the use of sodium bicarbonate in the bicarbonate-losing metabolic acidoses, such as those caused by diarrhea or renal tubular acidosis, in which replacement of lost bicarbonate is widely accepted. Although we touch on the use of bicarbonate in diabetic ketoacidosis (DKA) and some other forms of metabolic acidosis, our discussion and conclusions largely relate to the single most common cause of severe lactic acidosis in the modern ICU, that caused by severe sepsis.[4] We find little evidence that sodium bicarbonate is detrimental in these conditions, but its use fails the most basic criteria expected of any drug–efficacy.

Those who continue to advocate the use of sodium bicarbonate for lactic acidosis generally use the following chain of reasoning (explicitly or implicitly):

1. A low pH, in and of itself, is harmful (most notably by impairing cardiovascular function).
2. Sodium bicarbonate can increase the pH when infused IV.
3. Raising the pH with sodium bicarbonate improves cardiovascular function or some other relevant outcome.
4. Any adverse effects of sodium bicarbonate are outweighed by its benefits.

We address below each of these points in turn.

Is a Low pH Bad?

Proteins, which underlie the function and structure of human cells, contain areas of both positive and negative charge and are thereby sensitive to the pH of the surrounding milieu. One can conceive of innumerable ways in which excess acid could impair protein function and, by extension, the function of the whole body. Yet it is overly simplistic to assume that the clinician's window on acid-base state, the arterial blood pH, reflects accurately the pH at a (likely more important) cellular level. For example, a 50% reduction in blood flow to a tissue causes the arterial-venous Pco_2 difference to double (as long as CO_2 production and excretion remain constant) as predicted by the Fick principle. This will substantially raise the tissue Pco_2 and lower the local intracellular pH. At the same time, neither the arterial pH nor Pco_2 changes at all, both failing completely to reveal the tissue acidosis. Further clouding the value of the arterial blood pH, there may be different acid-base states in different cells of a single organ, or within different organs of a single patient. Mitochondrial pH may be even more crucial than cellular (cytoplasmic) pH, as these organelles are the site of energy production. In experiments involving isolated rat hepatocytes, the mitochondrial membrane pH gradient did not change when extracellular pH was lowered from 7.40 to 6.9.[9]

Because adults with acidosis generally also have sepsis, hypoxemia, intoxication, or hypoperfusion, discerning the physiologic effects of low pH from those of endotoxemia, hypoxemia, and so on is a challenging task. In isolated animal heart muscle preparations in which the pH of the perfusate is lowered, acidosis generally reduces contractile function,[10] sometimes severely.[11] Human ventricular muscle excised during open-heart surgery displays only modestly reduced contractility in the face of severe acidosis.[12] The cardiac depressant effect may be caused by inorganic phosphate-mediated impairment of actin-myosin crossbridge cycling, disruption of energy production,[11,13] interference with calcium delivery to myofilaments, or decreased sensitivity of contractile proteins to calcium.[14] In whole-animal preparations, the effects of acidosis are more difficult to discern, because of competing effects of acidosis on contractility, heart rate, vascular tone, and the adrenal and sympathetic systems. Various investigators have controlled heart rate, preload, and afterload, finding that acidosis caused contractility to remain constant,[15] decrease marginally,[16] or transiently rise and then fall.[17]

The increasing experience with permissive hypercapnia for patients with ARDS or status asthmaticus, in which hypercapnia and acidemia are tolerated to avoid alveolar overdistention, has changed many clinicians' perspective about the adverse impact of acidemia. In sedated and ventilated patients with ARDS, rapid intentional hypoventilation (pH falling from 7.40 to 7.26 in 30 to 60 min) lowered systemic vascular resistance while cardiac output rose. Mean systemic arterial pressure and pulmonary vascular resistance were unchanged.[18] Further, in many studies of patients undergoing permissive hypercapnia, a pH of well below 7.2 was tolerated well,[18–25] as it is in young patients with DKA,[26] children with "supercarbia,"[27] and those with grand mal seizures.[28–30] The feared consequences of acidemia, projected from the experience with patients having lactic acidosis (and, usually, concomitant sepsis), failed to materialize. In normal subjects who rebreathed carbon dioxide, QT dispersion was increased, which could signal a risk of arrhythmia.[24] With data now available for many patients permissively hypoventilated, the systemic hemodynamic effects are quite small even as the pH falls to 7.15, with the typical patient experiencing no change or small increases in cardiac output and BP. Rhythm disturbances have not been a problem. Patients whose pH levels fall far below 7.0 are fewer in number, so firm conclusions cannot be drawn, but they similarly tolerate their acidemia. The current practice of permissive hypercapnia does not generally include an attempt to alkalinize the blood to compensate for respiratory acidosis.

Cardiac contractile response to catecholamines is also impaired by acidosis, perhaps mediated by a decline in β-receptors on the cell surface.[13,31,32] Other potentially detrimental cardiovascular effects of acidosis include reduced resuscitability from induced ventricular fibrillation, which has been shown in rats but not in dogs or pigs,[33–35] impaired load tolerance of the right ventricle,[36] and altered renal blood flow (both increased and decreased, depending on the degree of acidemia).[37] Diaphragmatic contractility is reduced also by respiratory acidosis,[38] but apparently not by metabolic acidosis.[39]

Paradoxically, acidosis may have protective effects in critical illness. A low pH has been shown to delay

the onset of cell death in isolated hepatocytes exposed to anoxia[9] and to chemical hypoxia.[40] Correcting the pH took away the protective effect and accelerated cell death. In addition, acidosis during reperfusion limits myocardial infarct size.[41,42]

In summary, although a very low pH has negative inotropic effects in isolated hearts, the whole-body response in patients is much less clearly detrimental. Although clinical shock and metabolic acidosis often coincide, the striking discordance between the clinical course and outcome of patients with (usually septic) lactic acidosis compared with those who have DKA or ventilatory failure suggests that the low pH itself does not crucially underpin the hemodynamic collapse of these ill patients. Independent of the acidemia, the lactate ion may be significant, because lactate buffered to a pH of 7.4 can cause decreased cardiac contractility in animal models.[43]

Can Sodium Bicarbonate Raise the pH *In Vivo*?

It seems straightforward that adding a base to acidic blood will raise the pH–the reality is more complex. First, bicarbonate is not one of the independent determinants of the blood pH. Rather, these include the difference between the total concentrations of strong cations and anions (the strong ion difference, [SID]); the total concentration of weak acids; and the $Paco_2$.[44] Administration of exogenous sodium bicarbonate increases the SID (which tends to raise the pH) because sodium is a strong cation and bicarbonate is not a strong ion at all, but at the same time it elevates the $Paco_2$ (which tends to lower pH). In patients with lactic acidosis receiving mechanical ventilation, a modest infusion of sodium bicarbonate (2 mmol/kg for 15 min IV) boosted $Paco_2$ from 35 to 40 mm Hg.[2]

Second, bicarbonate administration may engender metabolic reactions that may themselves alter (SID), the total concentration of weak acids, or $Paco_2$. For example, in animals and humans, bicarbonate infusion can augment the production of lactic acid, a strong anion.[45–50] Mechanisms to explain this remain speculative, but include a shift in the oxyhemoglobin-saturation relationship[51]; enhanced anaerobic glycolysis, perhaps mediated by the pH-sensitive, rate-limiting enzyme phosphofructokinase; and changes in hepatic blood flow or lactate uptake.[52]

In animal models of lactic acidosis, sodium bicarbonate does not predictably raise the arterial pH. In some studies, pH remained constant or fell.[46,47] Most whole-animal studies, however, have shown that pH can be raised and even normalized.[16,48,49,53–55] Most relevant to clinical practice, in two studies of patients with lactic acidosis receiving mechanical ventilation, IV infusion of sodium bicarbonate in dosages of 2 mmol/kg for 15 min or 1 mmol/kg for 1 to 2 min raised the pH only 0.14 and 0.05 units, respectively.[2,56]

Yet, the body has multiple compartments separated by membranes of differing permeabilities and systems of active transport. Even when sodium bicarbonate added to the central veins reliably elevates the arterial pH, its effects on the cerebrospinal fluid and intracellular spaces may not be concordant. This could happen because carbon dioxide, produced when bicarbonate reacts with metabolic acids, diffuses readily across cell membranes and the blood-brain barrier, whereas bicarbonate cannot. Alternatively, as discussed above, bicarbonate may provoke reactions within cells, vessels, or organs that lower the local SID or raise the local Pco_2.

Sodium bicarbonate lowered cerebrospinal fluid pH in dogs with DKA,[57] dogs being resuscitated after a ventricular fibrillation arrest,[58] and two patients with DKA, in whom it was associated with a decrease in mental status.[59] Intracellular pH has been measured in cells or animals using nuclear magnetic resonance spectroscopy, pH-sensitive fluorescent dyes, intramuscular electrodes, and the distribution of carbon 14-labeled dimethadione (Table 1). The results are inconsistent, with intracellular pH rising in one study,[55] not changing in six studies,[11,34,49,53,60,61] falling in nine studies,[46,47,62–68] and either rising or falling depending on the buffer used in two investigations.[69,70] In various studies, intracellular pH has been shown to fall with bicarbonate in RBCs,[46] muscle,[68] liver,[47] lymphocytes,[66] and brain.[63] We are aware of only one human study involving determination by magnetic resonance spectroscopy of intracellular pH in the brain.[65] When these normal volunteers were given sodium bicarbonate IV, brain pH fell significantly.

In summary, sodium bicarbonate can raise the blood pH when given IV. In contrast, this therapy fails to augment reliably the intracellular pH. Indeed, intracellular pH falls in most animal models and in most organs studied, but the effect is variable.

Does Increasing the Blood pH With Sodium Bicarbonate Have any Salutary Effects?

The most direct question to pose regarding sodium bicarbonate therapy is whether it improves the problems that prompt its use. Namely, does it correct hemodynamics, "buy time" for other interventions, or improve outcome?

In isolated rat or rabbit hearts perfused with acidic solutions, bicarbonate fails to augment ventricular

Table 1—*Results of Studies of the Effect of Bicarbonate on Intracellular pH* *

Source	Subject	Acidosis	Method of Measuring Intracellular pH	Serum pH	Intracellular pH
Beech et al.[55]	Rat	DKA, shock	^{31}P NMR	↑	↑ (heart)
Rhee et al.[49]	Dog	Hypoxic lactic	^{31}P NMR	↔	↔ (heart)
Beech et al.[53]	Rat	Hypotensive lactic	C2 NMR	↑	↔ (muscle)
Bollaert et al.[60]	Rat	Septic (LPS)	^{31}P NMR	↑	↔ (muscle)
Shapiro[11]	Rat heart	Acidic perfusate	^{31}P NMR	↑	↔ (heart)
Thompson et al.[61]	Rat	None	^{31}P NMR	↑	↔ or ↓ depending on route (IV or IP)
Kette et al.[34]	Pig	Cardiac arrest	Electrode	↑	↔ (heart)
Arieff et al.[46]	Dog	Phenformin lactic	^{14}C DMO	↔	↓ (liver, RBC)
Graf et al.[47]	Dog	Hypoxic lactic	^{14}C DMO	↔	↓ (liver)
Bersin and Arieff[62]	Dog	Hypoxic lactic	^{14}C DMO	↓	↓ (liver)
Shapiro et al.[63]	Rat	NH$_4$Cl, hypercapnic	^{31}P NMR	↑	↓ (brain)
Shapiro et al.[64]	Rat	NH$_4$Cl	^{31}P NMR	↑	↓ (liver)
Arieff et al.[68]	Animal	Phenformin lactic	Not stated	↑	↓ (liver and muscle)
Nakashima et al.[65]	Human	None	^{31}P NMR		↓ (brain)
Bjerneroth et al.[66]	Lymphocytes	Acidic buffer	Fluorescent dye	↑	↓
Ritter et al.[67]	Platelets	Acidic buffer	Fluorescent dye		↓
Levraut et al.[70]	Rat hepatocytes	Acidic buffer	Fluorescent dye		↑ or ↓ depending on buffer
Goldsmith et al.[69]	Leukocytes	Acidic buffer	Fluorescent dye		↑ or ↓ depending on buffer

*NH$_4$Cl = ammonium chloride; NMR = nuclear magnetic resonance; DMO = dimethyloxazolidine; IP = intraperitoneal; LPS = lipopolysaccharide; ↑ = increase; ↓ = decrease; ↔ = no change.

contractility.[11,71] In whole animals (including various models of metabolic acidosis), the effects of bicarbonate on ventricular function are more difficult to tease out from its impact on systemic vessels. Further, one must take care not to interpret these studies too simplistically. For example, a fall in BP is not necessarily detrimental (if cardiac output rises). Nevertheless, these studies uniformly fail to reveal any hemodynamic benefit for sodium bicarbonate when compared with iso-osmolar saline solution.[16,45–49,53,55,62,64,72–74] When it has been measured, cardiac output either does not change[48,49,74] or falls.[11,46,47] Right ventricular contractility[72,74] and hepatic blood flow[46,47] fall. Perhaps the most careful study of left ventricular function involved L-lactic acid infusion in anesthetized, ventilated, β-blocked, and atrially paced dogs. Before and after bicarbonate infusion, the left ventricular pressure-volume relationship was determined with a ventricular Millar catheter and three orthogonal pairs of ultrasonic crystals imbedded in the ventricular walls.[16] Although sodium bicarbonate increased the arterial pH and did not increase lactate concentrations, mean arterial pressure fell, and cardiac output and ventricular contractility (slope of the end-systolic pressure-volume relationship) did not change. The hemodynamic effects were indistinguishable from those of saline solution.

There have been two studies of the hemodynamic impact of sodium bicarbonate in human lactic acidosis.[2,56] In both studies, patients were receiving mechanical ventilation, had a mean blood lactate between 7 mmol/L and 8 mmol/L, and were receiving continuous infusions of vasoactive drugs (except one patient in one study). Although sodium bicarbonate raised pH and serum bicarbonate concentrations, it did not improve hemodynamics or catecholamine responsiveness. Specifically, bicarbonate was indistinguishable from saline with regard to heart rate, central venous pressure, pulmonary artery pressure, mixed venous oxyhemoglobin saturation, systemic oxygen delivery, oxygen consumption, arterial BP, pulmonary artery occlusion (wedge) pressure, and cardiac output.[2,56] These findings suggest that the commonly observed hemodynamic response to bicarbonate administration in patients treated with vasoactive drug infusions may simply be one of preload augmentation (rather than enhanced catecholamine responsiveness). When the most severely acidemic (pH range 6.9 to 7.2) subset of patients was analyzed separately, these negative findings persisted.[2] This result does not support the practice of some physicians who withhold bicarbonate from patients with mild acidemia but feel compelled to give it to those with acidemia of greater magnitude. Indeed, if there are negative effects of bicarbonate infusion, there are reasons to expect that this subset of patients will suffer disproportionately (*ie*, develop more profound paradoxical intracellular acidosis).

Outcome is difficult to measure because animal models have a nearly 100% mortality and human trials have generally not been designed to detect differences in survival or other (nonhemodynamic) measures of outcome. In both prospective and retrospective studies of patients with DKA treated with or without sodium bicarbonate, there were no dif-

ferences in the neurologic status, incidence of hypo-kalemia or hypoglycemia, or rate of correction of acidemia,[75,76] but there was a suggestion of delayed clearance of ketones and lactate in patients given bicarbonate.[77,78] Dichloroacetate (DCA) infusion, which, like sodium bicarbonate infusion, effectively raises serum pH in critically ill patients with lactic acidosis (but lowers lactate concentrations), also has no apparent hemodynamic benefit and does not improve survival.[3]

The only study that has shown any positive effect on outcome is in the setting of canine ventricular fibrillation.[79] Dogs resuscitated from prolonged arrest who were given bicarbonate had improved return of the circulation, less neurologic deficit, and greater survival to 24 h. On the other hand, several other studies in both humans and animals do not support these data. A study in dogs showed no effect of respiratory or metabolic acidosis on defibrillation threshold.[35] Sodium bicarbonate had no detectable impact on myocardial cell pH or resuscitability from ventricular fibrillation in pigs in one study[34] and worsened coronary perfusion pressure and resuscitability in yet another.[80] Its use was associated with poorer outcomes in a retrospective study of human cardiopulmonary arrest.[81]

In summary, no controlled study has shown improved hemodynamics attributable to sodium bicarbonate infusion, regardless of the effect on pH, and many show worsening of some hemodynamic variable. It is significant that such negative findings include two studies in critically ill humans receiving infused catecholamines, the subset of patients who might be expected to benefit most dramatically.

Does Sodium Bicarbonate Have Negative Side Effects?

The most obvious side effects of sodium bicarbonate are the fluid and sodium load. This can cause hypervolemia, hyperosmolarity, and hypernatremia.[82] Sodium bicarbonate given as a rapid IV bolus can cause a transient fall in mean arterial pressure and a transient rise in intracranial pressure[83] that is probably related to its hypertonicity, and this is alleviated when given as a slow IV infusion. Sodium bicarbonate has been shown in three studies to lower PaO_2 from 5 to 15 mm Hg in both acidemic animals and nonacidemic patients with congestive heart failure.[57,84] The mechanism for this is unclear, but the authors speculated that there might be worsening of intrapulmonary shunt.

When normal human volunteers were made acidemic with acetazolamide and then corrected with sodium bicarbonate, the acute correction of the pH

caused increased hemoglobin affinity for oxygen that worsened oxygen delivery. This effect lasted about 8 h.[51] This was thought to be caused by the immediate nature of the Bohr effect and the delayed nature of the 2,3-diphosphoglycerate effect on hemoglobin-oxygen affinity.

Lactate concentrations increased with sodium bicarbonate infusion (compared with control subjects) in animal studies of hypoxic lactic acidosis,[45,47,49] phenformin-induced lactic acidosis,[46,68] hemorrhagic shock,[48] and DKA.[55,57] This finding has also been reported in cases of chronic lactic acidosis associated with malignancies.[85] It is possible that lactate rises in these settings because of impaired oxygen delivery to tissues. Even if enhanced lactate production does not signal cellular hypoxia, bicarbonate-induced hyperlactatemia may be detrimental inasmuch as lactate itself has potentially detrimental actions, as discussed earlier.

Serum ionized calcium concentration is reduced by sodium bicarbonate infusion. In a randomized, controlled study of ICU patients with lactic acidosis, sodium bicarbonate lowered ionized calcium from 0.95 to 0.87 mmol/L.[2] In an animal study of cardiac arrest, sodium bicarbonate decreased ionized calcium, although this had no apparent detrimental effects.[79] Because left ventricular contractility has been shown to vary directly with ionized calcium concentration,[86] any beneficial effects of pH correction may be masked by hypocalcemic ventricular depression.

A single study in patients with lactic acidosis treated with sodium bicarbonate failed to reveal any significant changes in venous lactate concentration, hemoglobin affinity for oxygen, total body oxygen consumption, oxygen extraction ratio, transcutaneous oxygen pressure, serum sodium concentration, or osmolality.[56] However, the dose of bicarbonate (1 mmol/kg) was small. Another study of three patients treated with sodium bicarbonate (mean dose, 90 mEq) during cardiac arrest revealed a rise in osmolality from 308 to 343 mosm/kg.[58]

In summary, many potentially detrimental effects of bicarbonate administration have been identified, but their clinical relevance has not been established.

Other Alkalinizing Therapies: Carbicarb, Dichloroacetate, Tris-Hydroxymethyl Aminomethane, and Dialysis

Carbicarb is an equimolar mixture of sodium carbonate and sodium bicarbonate. Compared with sodium bicarbonate, Carbicarb raises the SID (thereby the pH) far more[45,48,49,54,62] and boosts the PCO_2 far less[48,49,64] when given IV to animals with

metabolic acidosis. To the extent that the failure of sodium bicarbonate to effect hemodynamic improvement is caused by the generation of carbon dioxide, Carbicarb might be superior. Carbicarb more consistently increases intracellular pH,[49,62,64] and although it improved hemodynamics in two studies,[11,49] it did not in three others.[48,62,64]

DCA is a compound that probably works by stimulating the pyruvate dehydrogenase complex, the rate-limiting enzyme that regulates the entry of pyruvate into the tricarboxylic acid cycle, thus promoting the clearance of accumulated lactate. In addition, DCA increases myocardial glucose utilization and contractility.[87] Although several animal and clinical trials showed that DCA could raise pH and bicarbonate concentration while lowering lactate concentrations, with little apparent toxicity,[52,88,89] a large, multicenter, placebo-controlled trial in patients with lactic acidosis failed to confirm improved hemodynamics or outcome.[3] This drug is not available commercially. Tris-hydroxymethyl aminomethane is a weak alkali that penetrates cells easily. Its potential to raise blood and intracellular pH without producing carbon dioxide has been confirmed in animal models of metabolic acidosis. Tris-hydroxymethyl aminomethane also improved myocardial contraction and relaxation in an isolated rabbit heart preparation.[71] Although tris-hydroxymethyl aminomethane is commercially available, complications of hyperkalemia, hypoglycemia, extravasation-related necrosis, and neonatal hepatic necrosis are likely to limit its use.

There have been many case studies that report the use of dialysis to control the volume and sodium loads that accompany sodium bicarbonate infusion. In most of these reports, sodium bicarbonate alone did not improve the pH or lactate concentrations, but bicarbonate-buffered peritoneal dialysis did. Perhaps importantly, peritoneal dialysis was very effective at removing lactate. The returned dialysate contained between 2.6 mEq/L and 14 mEq/L of lactate in one study,[90] and the calculated lactate clearance by peritoneal dialysis averaged 21 mL/min in another.[91] The impact of bicarbonate infusion plus dialysis on cardiovascular function and outcome has not been studied systematically, nor has the relevance of the bicarbonate component of this potential treatment been examined.

Conclusion

Sodium bicarbonate is clearly effective in raising the arterial pH in critically ill patients with lactic acidosis. The impact on intracellular pH is unknown in such patients, but extrapolation from extensive animal studies suggests that it is negative. Despite the correction of arterial acidemia, sodium bicarbonate, like DCA, has no favorable cardiovascular effects, even for patients with severe acidemia and receiving continuous infusions of catecholamines. Although hemodynamic improvement is not the only mechanism by which bicarbonate might be beneficial, animal studies have failed to yield alternatives. Even theoretical arguments in favor of sodium bicarbonate administration rely on a naïve representation of acid-base physiology, ignoring the complex compartmentalization of pH, the second-level effects of bicarbonate infusion, the impact of carbon dioxide generation, or the negative consequences of hyperlactatemia. We believe most clinicians who continue to use bicarbonate for patients with severe lactic acidosis do so largely because of their inclination to action: How can I "fail" to give bicarbonate when no alternative therapy is available and the mortality of this condition is so high?

The oft-cited rationale for bicarbonate use, that it might ameliorate the hemodynamic depression of metabolic acidemia, has been disproved convincingly. Any future role for bicarbonate in these patients depends on the formulation of new hypotheses of efficacy followed by animal and clinical studies to seek to confirm any proposed benefit. Given the current lack of evidence supporting its use, we cannot condone bicarbonate administration for patients with lactic acidosis. We extend this to those with pH < 7.2 on vasoactive drugs, inasmuch as bicarbonate has no measurable beneficial effects even in these sickest patients. Indeed, we do not give or advise bicarbonate infusion regardless of the pH.

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