Stewart Acid-Base: A Simplified Bedside Approach

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Let me invite you to try something that will be new to many. You are faced with an intubated and ventilated patient with known cirrhosis who is transferred from the emergency room for a laparotomy. The patient has had saline resuscitation. Plasma chemistries obtained in the emergency room show the following: sodium, 133 mmol/L; chloride, 110 mmol/L; albumin, 22 g/L; and lactate, 5 mmol/L. An arterial blood gas reveals the following: pH, 7.20; Pco_2, 40 mm Hg; and bicarbonate, 15 mmol/L. Now consider the following questions:

1. What would be the acid-base consequence of further resuscitation with saline or plasmalyte?
2. What would be the acid-base consequence of further resuscitation with 5% albumin?
3. Is the lactate the primary cause of this patient's metabolic acidosis?

To help assess and manage this type of complex clinical case, I will describe what I call a simplified Stewart approach, which combines the base excess[1] and Stewart approach[2,3] to acid-base disorders. What I describe is the latest iteration of an approach we first described over 10 years ago[4] and is built on the efforts of others.[5–9] I use this method routinely and find it helpful in managing patients during perioperative care, including those who have had cardiac surgery or liver transplantation. Although experts in clinical chemistry may argue with some of what I describe, I hope to show that this approach has clinical utility during patient care.

Let me further suggest a challenge: for the next 10 patients in your care requiring arterial blood gas analysis, try the simplified Stewart approach detailed below and summarized in the following equation and see whether it enhances your understanding of the patient's acid-base condition:

$$\text{Base-excess} = [\text{Na} - \text{Cl} - 35] + [1 - \text{lactate}] + [0.25 \times (42 - \text{albumin})] + \text{other ions.}$$

The simplified Stewart approach has several steps and associated principles.

1. BASE EXCESS IS A GOOD OVERALL MEASURE OF METABOLIC ACID-BASE STATUS

The standard base-excess calculation[10,11] has been inconsistently used for acid-base analysis in the United States, but it is widely used in the rest of the world. Base excess was developed in the 1960s by Siggaard-Andersen[10] in Denmark and later refined to the plasma or standard base excess for clinical use.[1,11] To derive the base excess, Siggaard-Andersen conducted in vitro studies that equilibrated blood with a carbon dioxide (CO_2) partial pressure of 40 mm Hg, effectively removing any respiratory abnormality.[11] He then quantified the amount of strong, fully dissociated acid (hydrochloric acid [HCl]) or base (sodium hydroxide [NaOH]) in millimoles required to return a liter of blood to pH 7.40. This quantity is the base excess in millimoles per liter and is considered negative if NaOH must be used (acidosis) and positive if HCl is needed (alkalosis). As an alternative, some use the base deficit, which has the opposite sign of the base excess, so that as acidosis worsens, and the base deficit is an increasing positive number rather than the more negative number seen with base excess. Because of this difference, the equations that follow would have to be rearranged to accommodate base deficit.

The standard (or plasma) base excess allows for altered buffering in extravascular extracellular fluid and is routinely calculated by blood gas machines using the Van Slyke equation.[1,10,11] The reference normal value for standard base excess is 0 mmol/L, and the normal range is −3 to 3 mmol/L, with increasingly negative values indicating metabolic acidosis and positive values indicating metabolic alkalosis. Because base excess is defined as the amount of strong univalent acid (HCl) or base (NaOH) required to titrate 1 L of blood back to pH 7.40, 1 mmol/L = 1 meq/L. Importantly, unlike bicarbonate, no metabolic base-excess changes are expected with acute respiratory changes.[12] Furthermore, many clinicians are unaware that base excess can be corrected for chronic respiratory changes, approximately 0.4 mmol/L for every 1-mm Hg chronic change in carbon dioxide partial pressure.[12]

2. THE PRINCIPAL STEWART METABOLIC FACTOR IS THE PLASMA STRONG-ION DIFFERENCE

In the Stewart approach, the 3 independent controllers of acid-base status in body fluids are the partial pressure of CO_2, the strong-ion difference (SID), and the total amount of weak acids. Strong ions are those that are completely dissociated in a solution, in this case plasma. The measured SID is the sum of the plasma cations that are both routinely measured in clinical chemistry and completely dissociated (sodium, potassium, calcium, and magnesium) minus the anions that are routinely measured and

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completely dissociated (chloride and lactate). One way to visualize the SID is a Gamblegram,[5,13] developed by U.S. physiologist James Gamble (Fig. 1). Assuming electroneutrality, the Gamblegram demonstrates that the ions that fill the SID between the strong cations and anions are primarily bicarbonate (a factor thus dependent on the SID) and the total amount of weak acids, including albumin, which is the most important weak acid (reference anionic effect of 10 meq/L). A reduced SID suggests a lower bicarbonate level and the presence of an acidosis. The strong ions can be thought of as squeezing out the bicarbonate. For those who use bicarbonate-centered approaches to assessing acid-base disorders,[14,15] the presence of an acidosis is indicated by the decreased bicarbonate level suggested by the smaller SID. If the SID is increased, an increased bicarbonate level can be inferred, and an alkalosis is present. At the bedside, the most important strong ions for calculating the change in the SID in the simplified Stewart approach are sodium, chloride, and lactate.

3. WEAK ACIDS ARE ALSO IMPORTANT FOR METABOLIC ACID-BASE CHANGES

Weak acids are partly dissociated acids,[2,15] and therefore, by definition, not strong ions. Importantly, in a given fluid and at a given point in time, the SID does not influence the total weak acid concentration and, similarly, the total weak acid concentration does not influence the SID. Changes in bicarbonate and pH are secondary to changes in either the SID or the total amount of weak acids or both. Mechanistically, weak acids play a role opposite to the SID in determining the metabolic side of acid-base disorders. Acidosis is caused by a decrease in the SID but an increase in the total weak acid concentration, whereas the converse is true for alkalosis.[9]

The principal weak acids routinely measured in clinical chemistry in plasma are albumin and phosphate[7,16]—albumin usually being the more important quantitatively. In critically ill patients with hypoalbuminemia and renal failure with severe hyperphosphatemia, phosphate becomes dominant. In the Stewart approach, the total amount of weak acid(s) present is an independent contributor to acid-base status and has a reciprocal relationship with bicarbonate concentration. The bicarbonate concentration will decrease with an increase in the total weak acid concentration and vice versa. Dissociated weak acids form anions, which can be represented in a Gamblegram.[5] (Fig. 1) Albumin in plasma has an overall negative charge due to the dissociation of hydrogen ions from the histidine residues[16] and therefore it sits in the anions column and has a charge effect that can be estimated in milliequivalents per liter. It can be seen from the Gamblegram that, if the total amount of weak acid increases and SID is unchanged, bicarbonate will be squeezed out, resulting in an acidosis. The most important and frequent weak acid change in surgical and critical care patients is a decrease in the plasma albumin concentration, which causes a metabolic alkalosis.[4,7] Therefore, in critically ill patients, there can often be a decreased SID causing acidosis and a decreased weak acid concentration and producing less metabolic alkalosis, as in our example. (Table 1 and Fig. 1)

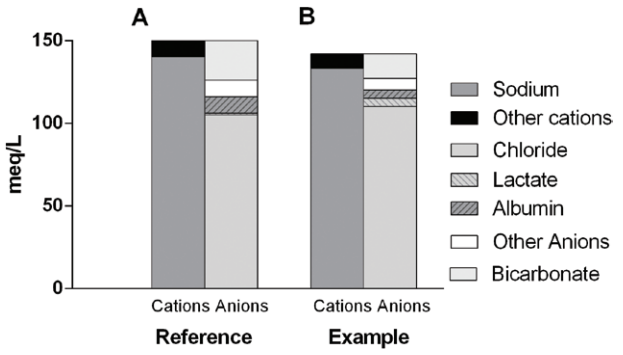

Figure 1. Gamblegrams of ions in plasma with paired columns of cations and anions. A, Reference values for the simplified Stewart approach based on the medians of the reference ranges. B, The plasma chemistry for the example (Table 1) for a patient with cirrhosis, sepsis, and saline resuscitation.

4. CHANGE IN BASE EXCESS IS DETERMINED BY CHANGES IN SID AND THE AMOUNT OF WEAK ACID

In addition to normal blood gas values (pH 7.40, P_{CO_2} 40 mm Hg, and bicarbonate 24 mmol/L), the other important reference values are sodium 140 meq/L, chloride 105 meq/L, lactate 1 meq/L, and albumin 42 g/L. Milliequivalents are units of electrical charge, and milliequivalents per liter can be used to unify the concentrations of plasma chemistry constituents. A milliequivalent is the amount of substance it takes to combine with 1 mmol of hydrogen ions; therefore, for univalent ions including Na, K, Cl, lactate, and bicarbonate, milliequivalents per liter can be directly substituted for millimoles per liter, whereas divalent ions such as calcium are in a 2:1 ratio of milliequivalents per liter to millimoles per liter. Accurately estimating the electrical charge of albumin is complex,[16] but a pragmatic approximation is 0.25 × albumin concentration in grams per liter.[13,17] Furthermore, because base excess is derived from millimoles per liter of HCl, changes in millimoles per liter of base excess are in a 1-to-1 ratio with milliequivalents per liter changes in the determinants of base excess. Of note, chloride reference ranges vary between assays by approximately 2 meq/L, and 105 mmol/L is now more frequently the median reference compared with 103 mmol/L.[4,13]

5. THE DIFFERENCE BETWEEN SODIUM AND CHLORIDE ION CONCENTRATIONS IS THE PREDOMINANT SID

The principal element of the plasma SID is the sodium – chloride (Na-Cl) difference. Using reference values, the normal Na-Cl difference is 140 – 105 = 35 meq/L.

$$\text{Na} - \text{Cl Base-excess effect } (\text{meq/L}) = \text{measured Na} - \text{measured Cl} - 35. \quad (1)$$

For every 1 meq/L change in the Na-Cl difference, the base excess will change by 1 meq/L: in the negative direction for a decrease in the SID, and in the positive direction for an increase in the SID. In hyponatremic patients, a normal chloride concentration (relative hyperchloremia) will result in a decreased SID and a metabolic acidosis. Conversely, hypernatremic patients will have an increased SID and a metabolic alkalosis with a chloride concentration in the reference range. Although it is possible

Table 1. Example of Applying the Simplified Stewart Approach

An intubated and ventilated patient with known cirrhosis is transferred from the emergency room for a laparotomy. The patient has had saline resuscitation.

Plasma chemistry: sodium, 133 mmol/L; chloride, 110 mmol/L; albumin, 22 g/L; and lactate, 5 mmol/L

Arterial blood gas: pH, 7.20; P_{CO_2}, 40 mm Hg; bicarbonate, 15 mmol/L; standard base excess, −11.5 mmol/L.

1. The sodium chloride base-excess effect = 133 − 110 − 35 = −12 meq/L (Equation 1)
2. Lactate base-excess effect = 1 − 5 = −4 meq/L (Equation 2)
3. Albumin base-excess effect = 0.25 × (42 − 22) = +5.5 meq/L (Equation 3)
4. Given base excess is −11.5 meq/L, the other ions (OI) base-excess effect = −1 meq/L (Equation 5)

If we use a bicarbonate-based approach, this patient has an acidemia and decreased bicarbonate level suggesting a metabolic acidosis with inadequate respiratory compensation (expected P_{CO_2} approximately 30.5 mm Hg: 1.5 × bicarbonate + 8)[15] and therefore a mixed disorder with increased lactate. The base excess alone tells us that there is a quantitatively important metabolic acidosis (expected P_{CO_2} approximately 29.5 mm Hg: 40 + base excess)[12] and therefore a mixed disorder, with an elevated lactate.

The simplified Stewart approach tells us this information, and also, quantitatively, that much of the change in base excess is secondary to a relative hyperchloremic metabolic acidosis partly offset by decreased albumin and aggravated by lactic acidosis. It also tells us that other ions do not currently play a major role. This finding is consistent with saline resuscitation in a patient with cirrhosis and an abdominal surgical problem. In addition to surgery and increased ventilation, a switch to lower chloride fluids such as Plasmalyte would be expected to widen the Na-Cl difference and decrease the acidosis. If Plasmalyte administration (Na 140 mmol/L and Cl 98 mmol/L) were to increase sodium by 1 meq/L and decrease chloride by 3 meq/L, the base excess would be expected to improve by 4 meq/L. Furthermore, because albumin is a weak acid, administering albumin will increase the total weak acid concentration and will increase the metabolic acidosis. This effect is in addition to adverse clinical chemistry effects of the crystalloid carrier for albumin that may increase the chloride concentration and that may sustain or worsen the Na-Cl base-excess effect by further decreasing the strong-ion difference. Finally from a Stewart perspective, sodium bicarbonate may be seen as chloride-free sodium that will increase the Na-Cl difference.

to divide this effect into a sodium (water excess) effect and separate chloride effect,[7,18] it is simpler to consider the Na-Cl difference[19] first to assess the overall acid-base state and then evaluate the individual electrolyte concentrations to analyze current and future fluid, electrolyte, and osmolality changes.

6. THE ANION LACTATE IS THE OTHER CLINICALLY IMPORTANT PLASMA STRONG ION

Apart from chloride, lactate is the other strong anion that is important in clinical acid-base changes. Because the principal cation normally associated with lactate, sodium, is accounted for in the Na-Cl difference (Equation 1), the effect of lactate on base excess may be estimated as follows:

$$\text{Lactate base-excess effect } (\text{meq/L}) = 1 - \text{measured lactate}. \quad (2)$$

It can be seen that as plasma lactate concentration increases, Equation 2 produces a more negative base excess and thus an acidosis. If lactate is not routinely measured, it would be considered in step 8 (below).

7. ALBUMIN IS THE PRINCIPAL WEAK ACID

The principal weak acid in plasma is albumin. The work by Figge[20] has been central to understanding the effective charge of albumin in plasma. As noted previously, a simple way to calculate the effective ionic concentration of albumin is (meq/L) = 0.25 × albumin concentration in grams per liter. Therefore, the ionic concentration corresponding to a normal albumin level is (42 g/L) = 0.25 × 42 g/L = 10.5 meq/L. The acid-base effect of albumin changes is calculated from the difference between this reference value and the ionic concentration corresponding to the patient's albumin level.

$$\text{Albumin base-excess effect, meq/L} = 0.25 \times (42 - \text{measured albumin}). \quad (3)$$

Therefore, for every 10 g/L decrease in plasma albumin, the base excess will increase by 2.5 meq/L, making the patient more alkalotic.

8. CONSIDER OTHER CHANGES IN STRONG IONS AND WEAK ACIDS

Other plasma constituents, both measured and unmeasured—both strong ions and weak acids—will effect metabolic acid-base changes, and therefore base-excess changes, and are uncommon in healthy people but are common in those with organ dysfunction, such as kidney or liver impairment.[21,22] Other ions include measured and unmeasured cations and anions.[13,23] Measured cations include potassium, calcium, and magnesium as well as unmeasured cations from proteins, lithium, or aluminum.[13] Other anions that are frequently more important than cations include phosphate, which is often measured in clinical practice, and anions that are likely to be present but not routinely unmeasured in clinical chemistry, such as sulfate and acetate, and then a multitude of currently unknown ions.[23]

9. PUTTING IT ALL TOGETHER

Changes in base excess are associated with changes in Na, Cl, albumin, lactate, and other strong ions and weak acids. To estimate the overall effect, the base-excess effects from Equations 1, 2, and 3 are combined.

$$\text{Base-excess} = \text{Na-Cl effect} + \text{lactate effect} + \text{albumin effect} + \text{OI effect}. \quad (4)$$

Substituting

$$\text{Base-excess} = [\text{Na} - \text{Cl} - 35] + [1 - \text{lactate}] + [0.25 \times (42 - \text{albumin})] + \text{OI}. \quad (5)$$

Equation 5 can also be solved for other ions:

$$\text{OI} = \text{Base-excess} - [\text{Na} - \text{Cl} - 35] - [1 - \text{lactate}] - [0.25 \times (42 - \text{albumin})]. \quad (6)$$

The importance of other ions is that if the Na-Cl, lactate, and albumin effects do not explain observed changes in the base excess, then one or more other factors must be present. In critically ill patients with acidosis, other ions, such

as phosphate and sulfate, commonly have diagnostic and prognostic importance,[13,23] as does an absence of unmeasured ions.

Equation 5 provides a simplified quantitative way to evaluate the acid-base contributions of the major measured plasma constituents in the Stewart approach. A worked example for a critically ill patient is shown in Table 1 and Figure 1B.

This simplified Stewart approach not only provides insights into the patient's current status and how it developed (i.e., sepsis, cirrhosis, and saline resuscitation) but it also helps the clinician anticipate the acid-base effects of future fluid and other therapies (Table 1).

STEWART IN CONTEXT

A recent review in the *New England Journal of Medicine*[15] describes several elements, and some of the complexity, of the Stewart approach to acid-base disorders. The Stewart approach is named after the Canadian physiologist, Peter Stewart, who argued that the bicarbonate-based approach to acid-base disorders failed to account for the complexity of multiple, interacting chemical systems.[2,24] To describe acid-base status, Stewart created 6 simultaneous equations that combined the chemical laws of mass action, conservation of mass, and electrical neutrality.[3,17] This complex approach (including a fourth-order polynomial) is detailed in the rereleased Stewart book[3] and summarized in an excellent critique by Morgan.[17]

Stewart used more general views of acidifying chemicals, especially chloride,[9,25] and derived an approach that integrates clinical plasma chemistry with quantifying clinical acid-base (patho)physiology. The fundamental, and most controversial, difference between Stewart and the bicarbonate-centered models of acid-base is the underlying proposal that the concentrations of hydrogen ions (therefore pH) and bicarbonate ions are not independent determinants of acid-base status, but the result of changes in other systems.[17] The Henderson-Hasselbalch equation for carbonic acid is still important for the Stewart approach, and bicarbonate has a role in describing acid-base status. But Stewart argued that the bicarbonate concentration did not cause acid-base status. Unfortunately, in part because of the intimate relationship between all the factors in a body fluid such as plasma, no one has yet developed an experiment to unequivocally demonstrate the role of bicarbonate as either a dependent (Stewart) or an independent (bicarbonate centered) factor in plasma acid-base status. Pure proponents of either approach thus typically start with differing fundamental positions on bicarbonate. (it is either dependent or independent).[9]

In contrast to bicarbonate-centered approaches, in the Stewart approach, bicarbonate and hydrogen ion concentrations are dependent on the combined SID, total weak acid concentration, and partial pressure of carbon dioxide. However, even if we use the Stewart approach to describe the underlying physiology, bicarbonate can be used to make acid-base diagnoses with the only difference that bicarbonate is a marker, but not a mechanism. In the simplified Stewart approach described here, pH and Pco_2 are treated as they would be with bicarbonate-centered approaches.[15] Furthermore, as with all clinical acid-base analysis, a key element is the deviation from reference values: pH, 7.40; Pco_2, 40 mm Hg; and bicarbonate, 24 mmol/L at 37°C.[2,10,15] One problem for clinical application of the original detailed Stewart approach is mathematical complexity[2,13,24] that many find daunting. Some of the other, albeit simpler, Stewart-based approaches are still too complex for quick bedside use without a calculator.[7,18] So the hybrid approach described here combines routine plasma chemistry with complex acid-base chemistry to provide a quantitative approach with simple arithmetic that highlights the most important features to diagnose and manage complex problems of the patients.

CONCLUSIONS

What I have described is an alternative approach to deciphering the acid-base status of patients with complex problems that quantitatively integrates plasma chemistry and acid-base at the bedside. In the same way that a pulse oximeter in the operating room allows estimation of dangerous changes in arterial blood gasses, this simplified Stewart approach combines several aspects of patient physiology and often provides helpful information. This approach provides direct insights into how changes in plasma chemistry associated with diseases such as renal or hepatic impairment—and how therapies such as normal saline—influence changes in acid-base status. Furthermore, this approach allows the clinician to anticipate the effects of clinical fluid choices, such as switching from saline to lactated Ringer's solution or giving 4% albumin, and the administered volumes. Compare and contrast this approach with the process one would normally use in analyzing patients, their situations, and what to do next, particularly with fluid and electrolyte therapy. If this simplified Stewart approach helps, then you now have another tool for deciphering metabolic disturbances. If not, then you at least have given Stewart a go.

DISCLOSURES

Name: David A. Story, MBBS, MD, BMedSci, FANZCA.
Contribution: This author prepared the manuscript.
Attestation: David A. Story approved the final manuscript.
This manuscript was handled by: Avery Tung, MD.

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monitoring can be a reliable and safe data transmission system in the OR. The prototype has been developed from a common work between anesthesiologists of the Military Teaching Hospital of Brest and engineers of the Civilian-Military French National Graduate Engineering Institute (ENSTA Bretagne) since September 2015. Continuous exchanges have allowed engineers to find step solutions to physicians' challenges, such as resistance to EIM or autonomy. First results are encouraging, and bluetooth ECG monitoring seems to be accurate and reliable. For example, bradycardia and tachycardia are detected by bluetooth ECG monitoring as quickly as the wired one. Our ongoing study answers to Hofer and Cannesson's editorial,[3] which asks for more research into wireless monitoring in anesthesiology.

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In Response

We thank Ariès et al[1] for their interest in our recent article.[2] We stated in our Discussion, "wireless technology in the critical care setting is generally safe and reliable."[2] Our point was that if implementing a wired solution is practicable, then this is preferable, given that there are sources of interference that may be difficult to identify and impossible to avoid completely. We mentioned several sources of potential interference in our discussion: "cordless phones, Wi-Fi adapters, wireless baby monitors,"[2] but this list was not intended to be comprehensive. Certainly, there are many other sources of electromagnetic interference in the Bluetooth bandwidth; this only serves to reinforce our conclusion.

We agree with the editorial by Hofer and Cannesson[3] that additional research is needed to better understand the risks and benefits of wireless monitoring.

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Stewart Versus Traditional Approach to Acid-Base Disorders

To the Editor:

The title of the article "Stewart Acid-Base: A Simplified Bedside Approach" by Story[1] is a paradox. There is nothing simple about the "Stewart Approach." For years, Stewart advocates have tried, unsuccessfully, to demonstrate that this approach offers a unique mechanistic or pathophysiologic insight into acid-base physiology.

Undoubtedly, the Stewart methodology (and the "base excess" approach) can be used to diagnose metabolic acid-base disorders, but it has no advantage over the classic physiologic methodology advanced by Schwartz and Relman[2] and Narins and Emmett.[3] The "Stewart Approach" is a more complicated and less-intuitive framework for diagnosing and understanding acid-base physiology/pathophysiology. Dr Story describes an intubated cirrhotic patient (who had received generous intravenous saline expansion) with the following laboratory results: Na: 133; Cl: 110; lactate: 5 (all mmol/L); albumin: 22 g/L; arterial blood gas—pH 7.20; Pco_2 40; HCO_3 15. Potassium and venous HCO_3 (or total CO_2)

were not reported. The latter can be assumed to be about 16 to 17 mEq/L. Dr Story then uses the Stewart framework to reach his acid-base diagnostic conclusions (with which I agree). I will use the traditional approach to analyze and interpret the patient's disorder:

1. pH = 7.20, this defines acidemia. The HCO_3 is reduced—therefore, this patient has metabolic acidosis.
2. Metabolic acidosis should generate hyperventilation and reduce the Pco_2. How low should it be? The "Winter's equation [$Pco_2 = 1.5 (HCO_3) + 8$] or other rules [$Pco_2 = HCO_3 + 15$] can be used. They indicate that the HCO_3 should be about 30 mm Hg. But the Pco_2 is too high at 40! Therefore, this patient also has respiratory acidosis.
3. Whenever a diagnosis of metabolic acidosis is established, determine whether it is an anion gap (AG) acidosis, a hyperchloremic acidosis, or a combination of the 2. The AG is calculated as AG = Na − (Cl + HCO_3). In this case, AG = 133 − (110 + 16) = 7 mEq/L. However, an additional step is required when the albumin concentration is reduced. Add 3 mEq/L for each 1 gm% reduction below the normal albumin concentration. Therefore, the "corrected AG" is about 13 mEq/L.

The reduction in HCO_3 concentration (or delta HCO_3), which is about 8 mEq/L (from a normal baseline of about 25), should be similar to the elevation in the AG, the increase in Cl, or a combination of the 2. In this case, the AG is increased by about 3 mEq/L (normal baseline = about 10), but the HCO_3 is reduced by about 8 mEq/L. Therefore, this patient has both AG and hyperchloremic acidosis. The AG acidosis is a lactic acidosis (lactate = 5 mEq/L). The hyperchloremic acidosis was at least partially generated by extra-cellular fluid expansion with normal saline. Large-volume saline expansion invariably generates a hyperchloremic metabolic acidosis because of dilution of extra-cellular fluid HCO_3 and other base buffers. The classic traditional approach generates the correct acid-base diagnoses: hyperchloremic (probably NaCl expansion) and AG (lactic) metabolic acidosis and respiratory acidosis. However, it is a simpler, more straightforward and easier to understand methodology than that of Stewart.

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In Response

I thank Professor Emmett for his response[1] to my Open Mind piece.[2] He uses the bicarbonate approach to correctly conclude that my example patient has "…hyperchloremic (probably NaCl expansion) and anion gap (lactic) metabolic acidosis and respiratory acidosis." I entirely agree with this. However, the simplified Stewart approach then provides an extended quantitative assessment of this problem: severe metabolic acidosis (base excess −11.5 mEq/L) predominantly due to a relative hyperchloremic effect (−12 mEq/L) that is deceptively severe as the result of both increased chloride (110 mmol/L) and decreased sodium (133 mmol/L) concentrations. The acid-base effect of lactate is relatively minor (−4 mEq/L). One point of convergence is that his calculation to correct the anion gap for decreased albumin in the bicarbonate approach is similar to that for the albumin effect on base excess in the simplified Stewart approach (answer about +6 mEq/L for the example patient).

As a critical care anesthetist, I find this quantitative simplified Stewart approach useful in managing patients at the bedside, particularly in working out the effects of fluid therapy. From this approach, the question: "Does my patient have hyperchloremic acidosis?" is answered with one easy estimate: Is Na-Cl-35 <0? Furthermore, the question, "What will a liter of 4% albumin do?" can be quantitatively estimated.

Our perceptions about acid-base are derived from training, experience, and clinical practice needs. Professor Emmett concludes that the bicarbonate-based approach is "…simpler, more straightforward and easier to understand…." As someone who has used Stewart for more than 15 years, I find the reverse to be true. Stewart becomes intuitive if one stops thinking about bicarbonate and instead focuses on the other routinely measured components of plasma chemistry. If I give a patient 30 mL/kg saline,[3] their chloride goes up and they get an acidosis due to the chloride. I do not need to think much about bicarbonate. Again, I encourage clinicians to compare what I have proposed with what they do now, using blood gasses in the operating room, intensive care unit, emergency department, or the internal medicine clinic. If what they do (including the bicarbonate approach) is adequate for their needs, that is fine.

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