

# An approach to complex acid-base problems

## *Keeping it simple*

Anthony M. Herd, MD, CCFP, CCFP(EM)

### ABSTRACT

**OBJECTIVE** To review rules and formulas for solving even the most complex acid-base problems.

**SOURCES OF INFORMATION** MEDLINE was searched from January 1966 to December 2003. The search was limited to English-language review articles involving human subjects. Nine relevant review papers were found and provide the background. As this information is well established and widely accepted, it is not judged for strength of evidence, as is standard practice.

**MAIN MESSAGE** An understanding of the body's responses to acidemia or alkalemia can be gained through a set of four rules and two formulas that can be used to interpret almost any acid-base problems. Physicians should, however, remember the "golden rule" of acid-base interpretation: always look at a patient's clinical condition.

**CONCLUSION** Physicians practising in acute care settings commonly encounter acid-base disturbances. While some of these are relatively simple and easy to interpret, some are more complex. Even complex cases can be resolved using the four rules and two formulas.

### RÉSUMÉ

**OBJECTIF** Passer en revue les règles et formules qui permettent de résoudre même les problèmes acido-basiques les plus complexes.

**SOURCES DE L'INFORMATION** Une recherche limitée aux articles de revue de langue anglaise portant sur des sujets humains a été effectuée dans MEDLINE entre janvier 1966 et décembre 2003. Les neuf articles pertinents repérés ont servi de base au présent travail. Comme cette information est bien fondée et généralement reconnue, sa valeur probante n'a pas été estimée, contrairement à la coutume.

**PRINCIPAL MESSAGE** L'utilisation de quatre règles et deux formules pour interpréter la plupart des problèmes acido-basiques permet de mieux comprendre les réponses de l'organisme à l'acidose ou à l'alcalose. Le médecin doit toutefois se rappeler la règle d'or de l'interprétation acido-basique: toujours tenir compte de la condition clinique du patient.

**CONCLUSION** Le médecin qui pratique dans un contexte de soins aigus est confronté à des déséquilibres acido-basiques. Si certains cas sont relativement simples et faciles d'interprétation, d'autres sont plus complexes. Ces derniers cas peuvent toutefois être résolus à l'aide des quatre règles et deux formules.

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Cet article a fait l'objet d'une évaluation externe.

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## Case 1

A 19-year-old woman came into the local emergency department with nausea and epigastric pain of several hours' duration. She recently found out she is unexpectedly pregnant and is quite upset with her unsupportive partner.

Her vital signs reveal moderate tachycardia and tachypnea, but she is normotensive. On examination, she is distressed, her chest is clear on auscultation, and her abdomen is soft with mild epigastric tenderness. An arterial blood gas (ABG) analysis has the following results:  $\text{PaO}_2 = 93$  mm Hg,  $\text{PaCO}_2 = 25$  mm Hg,  $\text{HCO}_3^- = 25$  mEq/L,  $\text{pH} = 7.57$ . What is the acid-base disturbance?

A few hours later she begins to vomit blood-streaked emesis and looks ill. A repeat ABG has the following results:  $\text{PaCO}_2 = 29$  mm Hg,  $\text{HCO}_3^- = 29$  mEq/L,  $\text{pH} = 7.62$ . What is the acid-base disturbance now?

Much later she gets drowsy and hypotensive. Repeat examination of her vital signs now shows marked hypotension. A third ABG reveals the following:  $\text{PaCO}_2 = 31$  mm Hg,  $\text{HCO}_3^- = 9$  mEq/L,  $\text{pH} = 7.12$ . What is the acid-base disturbance now, and what is a possible diagnosis?

Physicians practising in acute care settings commonly encounter patients with acid-base disturbances. While some of these are relatively simple and easy to interpret, some, such as the case described above, can be complicated. Keep in mind that, as cases get more intricate, they also become more interesting and challenging.

This article reviews rules and formulas for solving even the most complex acid-base problems. It describes how to use these rules and formulas to work through cases. By the end of the article,

*Dr Herd practises in the Department of Emergency Medicine at the Health Sciences Centre in Winnipeg, Man, and teaches in the Section of Family Medicine in the Faculty of Medicine at the University of Manitoba.*

readers should be able to answer the three questions posed in the case above and resolve almost any acid-base question, no matter how complex.

## Sources of information

A computer-assisted search of the scientific literature (MEDLINE) from January 1966 to December 2003 was carried out using National Center for Biotechnology Information PubMed search engines. Search terms included "acid-base analysis," "acid-base disorders," "arterial blood gas," and "metabolic acidosis." Given the nature of the topic, searches were limited to English-language review articles involving human subjects. More than 200 papers were initially found and their abstracts, where available, were reviewed. From these papers, nine relevant review articles were selected to provide background information.<sup>1-9</sup> As this information is well established and widely accepted, it is not judged for strength of evidence, as is standard practice.

## Acid-base interpretation

An in-depth knowledge of acid-base physiology is not essential for solving acid-base problems. For those who wish, a detailed discussion of it can be found in Costanzo.<sup>10</sup> An understanding of renal and pulmonary responses to acidemia or alkalemia can be gained through a set of rules and formulas (Table 1) that can be used to interpret acid-base abnormalities. Reading the following cases, you will see that you can solve acid-base problems without paying

**Table 1. Rules and formulas for interpretation of acid-base disturbances: The golden rule is "Look at the patient."**

RULE OR FORMULA	MATHEMATICAL FORMULA	RESPONSE
Rule 1. Metabolic acidosis	$1.2(\Delta\downarrow\text{HCO}_3^-) = \Delta\downarrow\text{PCO}_2$	Rapid
Rule 2. Metabolic alkalosis	$0.6(\Delta\uparrow\text{HCO}_3^-) = \Delta\uparrow\text{PCO}_2$	Rapid
Rule 3. Respiratory acidosis and alkalosis	$0.4(\Delta\text{PCO}_2) = \Delta\text{HCO}_3^-$	Slow
Rule 4. Anion gap	$\Delta\uparrow\text{anion gap} = \Delta\downarrow\text{HCO}_3^-$	
Formula 1. Anion gap	$\text{Anion gap} = [\text{Na}^+] - ([\text{HCO}_3^-] + [\text{Cl}^-])$	
Formula 2. Osmolar gap	$\text{Osmolar gap} = \text{osmo}_m - [2(\text{Na}^+) + \text{glucose} + \text{urea}]$	

much attention to pH values. Above all, remember the “golden rule” of acid-base interpretation: always look at the patient. Some patients could be really ill, despite results that are not much different from normal values. And, conversely, patients who are relatively stable despite abnormal arterial blood gas (ABG) results might still need close observation, but only conservative therapy.

### Rule 1. Metabolic acidosis

In pure metabolic acidosis, every 1 mEq/L decrease in serum bicarbonate should lead to a compensatory decrease of 1.2 mm Hg in  $P_{CO_2}$ . This change in  $CO_2$  should occur very quickly, and anything greater or less than this predicted change should lead you to consider an accompanying respiratory acid-base derangement. Rule 1 can be mathematically represented as  $1.2(\Delta \downarrow HCO_3^-) = \Delta \downarrow P_{CO_2}$ .

### Case 2

A 56-year-old man with a history of diabetes mellitus and alcoholism presents after having eaten no food and taken no insulin for the last 3 days and drinking “lots” of alcohol. He is hypotensive, tachycardic, and markedly tachypneic (respiratory rate 36). He smells strongly of acetone and is dehydrated, and clinical findings are consistent with left lower lobe pneumonia. Results of ABG testing are:  $P_{aO_2}$  = 68 mm Hg,  $P_{aCO_2}$  = 17 mm Hg,  $HCO_3^-$  = 6 mEq/L, and pH = 7.30. What is the acid-base abnormality?

Using rule 1, the answer is that he has metabolic acidosis with an appropriate respiratory response. His bicarbonate level is low, likely because of a combination of prolonged alcohol intake, starvation, and ketone production (the acetone on his breath). To attempt to correct his pH, he hyperventilates and exhales his  $P_{CO_2}$  down to 17, almost as low as it can go. His pH approaches normal but does not quite get there. Renal and respiratory compensatory mechanisms can almost, but never completely, correct an abnormal pH level.

What would this patient's acid-base profile be if his respiratory rate were 14 breaths/min and his  $P_{aCO_2}$  were 29 mm Hg? What might his pH be? The answer is that he would have metabolic acidosis and concomitant respiratory acidosis. Although his  $P_{aCO_2}$  is low at 29, it is not as low as it should be. He is actually hypoventilating relative to his needs, possibly due to pneumonia and fatigue. His pH is in fact 6.99, potentially life threatening. He not only needs standard therapy with fluids and insulin for ketoacidosis, but might very well require ventilatory support. If his  $P_{aCO_2}$  were to rise to 40, still a “normal” value, his pH would plummet to 6.75, and he would die.

### Rule 2. Metabolic alkalosis

With pure metabolic alkalosis, every 1 mEq/L increase in serum bicarbonate should result in a compensatory rise in  $P_{CO_2}$  of 0.6 mm Hg. This increase in  $CO_2$  should occur very quickly, and again, anything greater or less than this should lead you to think of an associated respiratory abnormality. This rule can be mathematically represented as  $0.6(\Delta \uparrow HCO_3^-) = \Delta \uparrow P_{CO_2}$ .

### Case 3

A 44-year-old woman presents with 24 hours of unremitting emesis. She is dehydrated and hypotensive. Tests of her ABG show the following values:  $P_{aO_2}$  = 104 mm Hg,  $P_{aCO_2}$  = 46 mm Hg,  $HCO_3^-$  = 34 mEq/L, and pH = 7.49. What is the acid-base disturbance here?

Using rule 2, we see that she has metabolic alkalosis from the continuous vomiting and loss of acid-rich gastric contents. In response to the increase in bicarbonate (she has a base excess of 10 mEq/L), her body appropriately allows her  $CO_2$  to rise in an attempt to return the pH to normal. Although the metabolic abnormality has evolved over an entire day, the respiratory response can occur and change quickly. What would the acid-base profile look like if she were anxious and hyperventilating?

What would her acid-base status be if she became obtunded and began hypoventilating?

If she were to hyperventilate and drive her  $\text{PaCO}_2$  down to, say, 26 mm Hg, her pH would be something like 7.67. If she hypoventilated and allowed her  $\text{CO}_2$  to rise too much to, say, 56 mm Hg, her pH would be 7.42. Although this value is healthier-looking than before, her clinical situation has clearly worsened (the golden rule). A normal pH value usually points to a double acid-base abnormality. Compensatory mechanisms can never quite normalize the pH.

### Rule 3. Respiratory acidosis or alkalosis

For pure respiratory acidosis, every 1-mm Hg increase in  $\text{PaCO}_2$  should cause the bicarbonate level to rise by 0.4 mEq/L. Conversely, with pure respiratory alkalosis, every 1-mm Hg decrease in  $\text{PaCO}_2$  should result in the  $\text{HCO}_3^-$  falling by 0.4 mEq/L. Any change in  $\text{HCO}_3^-$  greater or less than predicted *might* point to an accompanying metabolic abnormality. The situation can get confusing, however, because the kidneys take up to 24 hours to respond fully to respiratory acidosis or alkalosis. Rule 3 can be mathematically represented as  $0.4(\Delta\text{PaCO}_2) = \Delta\text{HCO}_3^-$ .

### Case 4

Two patients, both in respiratory distress, present to your hospital when you have only a single ventilator available. Each has a history of emphysema and each wants “everything possible done.” Results of ABG testing of the first patient show  $\text{PaO}_2 = 68$  mm Hg,  $\text{PaCO}_2 = 58$  mm Hg, and  $\text{HCO}_3^- = 22$  mEq/L. Results for the second patient are  $\text{PaO}_2 = 59$  mm Hg,  $\text{PaCO}_2 = 75$  mm Hg, and  $\text{HCO}_3^- = 38$  mEq/L. All else being equal and based purely on ABG analysis, which patient should get the ventilator?

The first patient clearly has respiratory acidosis: his  $\text{PaCO}_2$  is 18 mm Hg greater than normal. Using rule 3, an appropriate renal response would

be to conserve  $\text{HCO}_3^-$  and increase it to about 31 mEq/L. Since his system has not yet done this, we can assume this is *acute* respiratory acidosis. Note how we can arrive at this conclusion without even knowing his pH (which, incidentally, at 7.18 supports our judgment). The second patient has an even greater respiratory acidosis: his  $\text{PaCO}_2$  is 35 mm Hg higher than normal. Rule 3 tells us that his predicted  $\text{HCO}_3^-$  should be 38, exactly as his ABG reveals. Thus he is showing an entirely appropriate metabolic response to *chronic* hypercapnia. Again, we have reached this conclusion without knowing his pH (which, in fact, is 7.34). Hence, the first patient needs the ventilator more than the second. You must, of course, follow the golden rule and base your decision on clinical grounds.

### Formula 1. The anion gap

The anion gap is equal to the difference between  $\text{Na}^+$  concentration and the sum of the concentrations of  $\text{Cl}^-$  and  $\text{HCO}_3^-$ . It can be mathematically represented as  $\text{anion gap} = [\text{Na}^+] - ([\text{HCO}_3^-] + [\text{Cl}^-])$ .

The normal value for the anion gap is less than 10 mEq/L, and under usual circumstances, it is composed of sulfate, phosphate, citrate, and certain plasma proteins. It might become greater with decreased excretion of fixed acids, addition of lactate or ketones, or ingestion of certain toxins.

### Case 5

A 41-year-old man with a history of delirium tremens presents having had multiple seizures. He has been drinking heavily and has neglected to take his anticonvulsant medication for 4 days. He is drowsy but tremulous with fever, tachycardia, and hypertension. Results of ABG tests are  $\text{PaO}_2 = 62$  mm Hg,  $\text{PaCO}_2 = 3$  mm Hg,  $\text{HCO}_3^- = 13$  mEq/L, and  $\text{pH} = 7.17$ . What is this acid-base disturbance?

He has metabolic acidosis with a base deficit of 11 mEq/L. Using rule 1, you can see that he also has respiratory acidosis. If he were ventilating

appropriately, his  $P_{aCO_2}$  would be 27 and his pH would be 7.30. As his ABG results show, however, his  $P_{aCO_2}$  is actually much higher and his pH much lower. Although his  $CO_2$  is in the normal range, he looks very sick (the golden rule).

Laboratory values reveal the following:  $Na^+ = 146$  mEq/L,  $HCO_3^- = 12$  mEq/L, and  $Cl^- = 112$  mEq/L. What is his anion gap? Using formula 1, we see that his anion gap is 22 (normal value is up to 10). What this means is that he still has to account for about 12 mEq/L of some unidentified anion in his extracellular fluid compartment. **Table 2** shows causes of anion gap acidosis.

**Table 2. Causes of metabolic acidosis**

INCREASE IN ANION GAP ( $H^+$ GAIN)
Decreased fixed acid excretion (eg, renal failure)
Increased endogenous acid production
• Lactic acidosis
• Ketoacidosis
Exogenous acid ingestion
• Acetylsalicylic acid
• Cyanide
• Toxic alcohols (methanol, ethylene glycol)
HYPERCHLOREMIC OR NORMAL ANION GAP ( $HCO_3^-$ LOSS)
Gastrointestinal tract (eg, diarrhea)
Renal system (eg, diuretics, renal tubular acidosis)

## Case 6

A 74-year-old man, taking diuretics for heart failure, presents with weakness and diarrhea of 2 days' duration. On examination he is markedly dehydrated and hypotensive. His ABG results are:  $P_{aCO_2} = 22$  mm Hg,  $HCO_3^- = 10$ , and pH = 7.33. Serum biochemical analysis shows the following:  $Na^+ = 130$  mEq/L,  $HCO_3^- = 9$  mEq/L, and  $Cl^- = 116$  mEq/L. What is his acid-base problem, and what is his anion gap?

This patient has metabolic acidosis with a base deficit of 15 mEq/L. Using rule 1, we can see that his  $P_{CO_2}$  should be about 22 mm Hg, so his ventilatory response to the acidemia is appropriate. Using

formula 1, we calculate his anion gap at 5 mEq/L. Metabolic acidosis with a normal anion gap arises from bicarbonate loss. **Table 2** lists causes of hyperchloremic metabolic acidosis. The probable causes of his  $HCO_3^-$  depletion are diarrhea and chronic diuretic use.

## Rule 4. The base deficit and the anion gap

Metabolic acidosis resulting from any acid gain will be associated with an increase in unmeasured anions (anion gap). Addition of  $H^+$  will also deplete  $HCO_3^-$  as it is buffered. Because each of the acids listed in **Table 2** dissociates into a single hydrogen ion and its respective anion, every 1-mEq rise in the anion gap should be accompanied by a 1-mEq fall in bicarbonate. A higher-than-expected  $HCO_3^-$  should lead you to consider an additional metabolic alkalosis. This rule can be represented as  $\Delta \uparrow$  anion gap =  $\Delta \downarrow HCO_3^-$ .

## Case 7

A 56-year-old man presents with anorexia and unremitting emesis for 4 days. Results of ABG testing are  $HCO_3^- = 18$  mEq/L,  $P_{aCO_2} = 30$  mm Hg, and pH = 7.40. His biochemistry profile reveals the following:  $Na^+ = 130$  mEq/L,  $HCO_3^- = 18$  mEq/L, and  $Cl^- = 89$ . What is his acid-base disturbance, and what is his anion gap?

This man has metabolic acidosis with a base deficit of about 7 mEq/L. Rule 1 reveals satisfactory respiratory compensation for the acidemia. Using formula 1, we can calculate his anion gap at 23 mEq/L. His base deficit is smaller than his anion gap. How can this be? The answer is quite simple. If he had a preceding metabolic alkalosis from 4 days of vomiting acid-rich gastric contents, he might have started with a higher  $HCO_3^-$  concentration before starvation ketoacidosis set in. **Table 3** lists common causes of metabolic alkalosis.

Note his "dead-on" normal pH of 7.4, despite the elevated anion gap acidosis, compensatory



**Table 3. Causes of metabolic alkalosis**

Volume contraction
• Dehydration
• Overdiuresis
Loss of hydrochloride
• Vomiting
• Gastric suction
• Taking excessive antacids
Hypokalemia
Hypercapnia (acute correction)
Excessive resuscitation (Ringer's lactate solution)
Excessive transfusion (citrated blood)
Hypoalbuminemia
Hypoparathyroidism
Excretion of nonresorbable anions (eg, $\text{Po}_4^{2-}$ )

respiratory alkalosis, and initial metabolic alkalosis. A perfectly normal pH strongly suggests a triple acid-base disturbance.

## Formula 2. The osmolar gap

The osmolar gap is equal to the difference between the *measured* serum osmolarity ( $\text{osmo}_m$ ) and the *calculated* serum osmolarity ( $\text{osmo}_c$ ). It can be mathematically represented by the following formula:  $\text{osmolar gap} = \text{osmo}_m - \text{osmo}_c$ .

The main osmotically active substances in serum are sodium chloride (NaCl), urea, and glucose (the osmolarity of NaCl is twice that of glucose or urea). Thus, the calculated serum osmolarity can be derived as:  $\text{osmo}_c = 2(\text{Na}^+) + \text{glucose} + \text{urea}$ .

Substances that increase the  $\text{osmo}_m$  and, hence, the osmolar gap are sugars other than glucose (which figures in the calculation), and all alcohols. Toxic alcohols also increase the anion gap (Table 2). As a general rule, an anion gap metabolic acidosis with an elevated osmolar gap is due to a toxic alcohol until proven otherwise.

## Complex metabolic acidosis

With any complex metabolic acidosis, you can work toward a specific diagnosis if you ask the following three questions. First, what is the anion gap

(formula 1)? Second, does the anion gap equal the base deficit (rule 4)? Third, what is the osmolar gap (formula 2)? This will allow you to sort through the many causes listed in Table 2. And, of course, the golden rule is still important.

## Case 8

A 33-year-old woman with a long history of alcoholism presents unresponsive with hypotension, tachycardia, and tachypnea. Her ABG results are  $\text{PaCO}_2 = 23$ ,  $\text{HCO}_3^- = 7$  mEq/L, and  $\text{pH} = 7.12$ . Her venous biochemistry profile shows the following:  $\text{Na}^+ = 142$ ,  $\text{Cl}^- = 105$ ,  $\text{HCO}_3^- = 6$ , glucose = 8.4, urea = 5.5, osmolarity = 349, and ethanol = 22 mmol/L. How do you work through this woman's acid-base abnormality?

This patient has marked metabolic acidosis with a base deficit of 20 mEq/L. Rule 1 indicates that she also has an accompanying respiratory acidosis. Her  $\text{PCO}_2$  should be 16 mm Hg, but it is 23 mm Hg.

Formula 1 shows her anion gap to be 31 mEq/L. As a normal anion gap is up to 10 mEq/L, her anion gap is about 21 mEq/L, which approximates her base deficit, satisfying rule 4. We are unlikely to see any additional metabolic problem. Formula 2 allows us to calculate her osmolar gap; it is markedly elevated at 50 mmol/L. Her ethanol level accounts for only 22 mmol/L of the osmolar gap. Thus, we should strongly suspect that a toxic alcohol is causing her anion gap metabolic acidosis. Additional laboratory tests ultimately show that her lactate and ketone levels are only mildly elevated, but her methanol level is 26 mmol/L. She needs aggressive therapy with hemodialysis or she will die.

## Answer to case 1

The 19-year-old woman came to your local emergency department with nausea and epigastric pain. An initial ABG analysis showed the following:  $\text{PaO}_2 = 93$  mm Hg,  $\text{PaCO}_2 = 25$  mm Hg,  $\text{HCO}_3^- = 25$  mEq/L,  $\text{pH} = 7.57$ . What is the acid-base disturbance?

She has respiratory alkalosis: her  $\text{PaCO}_2$  is 15 mm Hg lower than normal. Rule 3 tells us that

a proper response by her kidneys would result in a  $\text{HCO}_3^-$  concentration of about 20 mEq/L. She has not yet had sufficient time for this to happen, so we can say she has acute respiratory alkalosis (note her high pH). A few hours later, repeat ABG tests show the following:  $\text{PaCO}_2 = 29$  mm Hg,  $\text{HCO}_3^- = 29$  mEq/L, pH = 7.62. What is the acid-base disturbance now?


She has now developed metabolic alkalosis from the vomiting. Note that her  $\text{HCO}_3^-$  has risen to 29 instead of decreasing, and her pH is even higher. Rule 2 tells us that if her  $\text{PaCO}_2$  were to rise to 43, as it should, her pH would be much closer to normal. She still has some respiratory alkalosis. Much later, when she appears more ill, a third ABG test reveals the following:  $\text{PaCO}_2 = 31$  mm Hg and pH = 7.12. What is the acid-base disturbance now, and what is a possible diagnosis?

She has now developed metabolic acidosis. Her bicarbonate has plummeted from 29 to 9 mEq/L. **Table 2** lists possible causes. When you calculate her anion gap as very high and her osmolar gap as very low, the list of possibilities is narrowed.

Finally, rule 1 tells us that her ventilatory response should be to drive her  $\text{PaCO}_2$  down to 22 mm Hg, but it is actually higher. So she has associated respiratory acidosis, and this is shown by the very low pH.

She eventually admits to having taken a substantial overdose of acetylsalicylic acid (readers will likely have already recognized the characteristic profile of ASA toxicity). Salicylates commonly cause initial respiratory alkalosis, due to direct central nervous system stimulation, followed by metabolic acidosis due to direct toxic effects.

## Conclusion

Using the four rules and two formulas for acid-base problem interpretation, we have worked through several cases and demonstrated their usefulness. Even when cases are complex, they can be solved readily using the rules and formulas. 

## Competing interests

None declared

### EDITOR'S KEY POINTS

- Acid-base disturbances can be conveniently understood using four rules and two formulas.  
Rule 1. Metabolic acidosis:  $1.2(\Delta\downarrow\text{HCO}_3^-) = \Delta\downarrow\text{PCO}_2$ ; Rule 2. Metabolic alkalosis:  $0.6(\Delta\uparrow\text{HCO}_3^-) = \Delta\uparrow\text{PCO}_2$ ; Rule 3. Respiratory acidosis and alkalosis:  $0.4(\Delta\text{PCO}_2) = \Delta\text{HCO}_3^-$ ; Rule 4. Anion gap:  $\Delta\uparrow\text{anion gap} = \Delta\downarrow\text{HCO}_3^-$ ; Formula 1. Anion gap:  $\text{anion gap} = [\text{Na}^+] - ([\text{HCO}_3^-] + [\text{Cl}^-])$ ; Formula 2. Osmolar gap:  $\text{osmolar gap} = \text{measured osmolality} - \text{calculated osmolality}$
- Solve most problems by asking: What is the anion gap? (formula 1); does the anion gap equal the base deficit? (rule 4); and what is the osmolar gap? (formula 2).
- Remember the "golden rule" of acid-base interpretation: always look at a patient's condition.

### POINTS DE REPÈRE DU RÉDACTEUR

- On peut facilement interpréter les déséquilibres acido-basiques en recourant à quatre règles et deux formules.  
Règle 1. Acidose métabolique :  $1,2 (\Delta\downarrow\text{HCO}_3^-) = \Delta\downarrow\text{PCO}_2$ ; Règle 2. Alcalose métabolique :  $0,6 (\Delta\uparrow\text{HCO}_3^-) = \Delta\uparrow\text{PCO}_2$ ; Règle 3. Acidose et alcalose respiratoire :  $0,4 (\Delta\text{PCO}_2) = \Delta\text{HCO}_3^-$ ; Règle 4. Trou anionique :  $\text{trou anionique} = \Delta\uparrow\text{HCO}_3^-$ ;  
Formule 1. Trou anionique =  $[\text{Na}^+] / ([\text{HCO}_3^-] + [\text{Cl}^-])$   
Formule 2. Trou osmolaire =  $\text{osmolalité mesurée} - \text{osmolalité calculée}$ .
- On peut résoudre la plupart des problèmes en se demandant : quel est le trou anionique? (formule 1); le trou anionique est-il égal au déficit en bases (règle 4) ; et quel est le trou osmolaire ? (formule 2).
- Se souvenir de la «règle d'or» de l'interprétation acido-basique : toujours tenir compte de l'état du patient.

**Correspondence to:** Dr Anthony M. Herd, GE-207, Health Sciences Centre, 820 Sherbrook St, Winnipeg, MB R3A 1R9; telephone (204) 787-2934; fax (204) 787-5134; e-mail [aherd@hsc.mb.ca](mailto:aherd@hsc.mb.ca)

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