Finding the Balance

Simple Acid-Base Interpretation

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Physicians practising in the emergency setting commonly encounter acid-base disturbances. While some are relatively simple and easy to interpret, others can be rather complicating and challenging.

How is pH balance maintained?

Many of our bodily functions are concerned with maintaining a low concentration of hydrogen ions (H^+). Under normal circumstances, the H^+ concentration of our extracellular fluid is approximately 35 mEq/L to 45 mEq/L. This defines a narrow pH range of approximately 7.35

Jack's case

Jack, 28, has a history of alcoholism. He presents with delirium and hypotension. His arterial blood gas shows:

- PaO_2: 88 mmHg
- $PaCO_2$: 24 mmHg
- HCO_3^-: 7 mEq/L
- pH : 7.11

His biochemistry profile shows:

- Na^+: 143 mEq/L
- Cl^-: 105 mEq/L
- HCO_3^-: 7 mEq/L
- Glucose: 8.4 mmol/L
- Urea: 5.5 mmol/L
- Osmolarity: 350 mmol/L

PaO_2: Partial pressure of oxygen in arterial blood
$PaCO_2$: Partial pressure of carbon dioxide in arterial blood
HCO_3^-: Bicarbonate
Na^+: Sodium
Cl^-: Chloride

What is this patient's acid-base problem?

Table 1
Rules of acid-base interpretation

Rule	Formula	Response	Purpose
#1 Metabolic acidosis	$1.2(\Delta \downarrow HCO_3^-) = \Delta \downarrow PCO_2)$	Rapid	Used to predict the appropriate respiratory response to metabolic acidosis
#2 Metabolic alkalosis	$0.6(\Delta \uparrow HCO_3^-) = (\Delta \uparrow PCO_2)$	Rapid	Used to predict the appropriate respiratory response to metabolic alkalosis
#3 Respiratory acidosis & alkalosis	$0.4(\Delta PCO_{2)} = \Delta HCO_3^-$	Slow	Used to predict the proper eventual metabolic response to a respiratory acidosis or alkalosis
#4 Anion gap and base deficit	$\Delta \uparrow AG = \Delta \downarrow HCO_3^-$		With a pure AG metabolic acidosis, the increase in AG should equal the decrease in HCO_3^- (BD)

HCO_3^-: Bicarbonate concentration (mEq/L)
PCO_2: Partial pressure of carbon dioxide
AG: Anion gap (mEq/L)
BD: Base deficit

to 7.45, which is essential for all cellular functions. An arterial pH of < 7.35 is called acidosis or acidemia, while an arterial pH of > 7.45 is called alkalosis or alkalemia. The normal pH is maintained by complex interactions between extracellular and intracellular buffers, as well as by the kidneys and the lungs.

Two types of acid are produced daily from normal metabolism. Carbon dioxide (CO_2) is the end-product of aerobic energy production and combines with water to form weak carbonic acid (H_2CO_3), which, aided by the enzyme carbonic anhydrase, readily dissociates into H^+ and bicarbonate (HCO_3). The H^+ generated this way must be buffered, if only temporarily. In the lungs, this reaction reverses and CO_2 is regenerated and excreted by ventilation. During protein and lipid catabolism, fixed acids, such as sulfates and phosphates, are produced and must be buffered until they can be excreted by the kidneys. In renal failure, these (fixed) acids can accumulate, leading to acidosis. Increased production of other endogenous acids, such as ketones and lactate, may also cause acidosis, while the ingestion of toxins, such as salicylates or methanol, can cause severe acidemia.

The major buffer system of the extracellular fluid is the pair of HCO_3^- and H_2CO_3. Most H_2CO_3 exists in the blood in the form of CO_2, so this system is often referred to as the HCO_3/CO_2 buffer pair. The pH of arterial blood can be calculated from a known concentration of HCO_3^- and partial pressure of CO_2 by the Henderson-Hasselbach equation [$pH = 6.1 + \log (HCO_3^-)/0.03(PCO_2)$].

The kidneys perform three major functions to prevent acidosis. First, almost all of the HCO_3^- filtered by the glomeruli is reabsorbed by the proximal tubular cells. Second, fixed acids are excreted. Finally, new HCO_3^- is synthesized to replenish what

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was depleted in the buffering of these fixed acids. In response to alkalosis, the kidneys will increase HCO_3^- excretion by decreasing its synthesis and reabsorption.

In the lungs, hyperventilation and increased CO_2 excretion occur in response to acidemia, while hypoventilation and increased arterial PCO_2 attempt to compensate for alkalemia.

What are the must-know rules and formulas?

There are four useful rules, derived from the Henderson-Hasselbach equation, for interpreting any acid-base problem (Table 1). Respiratory compensation occurs very quickly, while renal compensatory mechanisms take place over many hours.

There are also formulas used for calculating the anion gap (AG) and osmolar gap (OG) (Table 2).

Three questions need to be asked in the case of any patient with a metabolic acidosis:

1. What is the AG? Calculating the AG using Formula #1 will assist in sorting through the various causes of acidosis (Table 3).
2. Does the increase in the AG equal the base deficit (BD)? If the AG exceeds the BD, there is likely to be a metabolic alkalosis in addition to the AG acidosis.
3. What is the OG? Calculating the OG can help you differentiate between different exogenous acids.

Table 2
Anion and osmolar gaps

Formula	Normal value
Formula #1	
$AG = [Na^+] - [(HCO_3^-) + (Cl^-)]$	10
Formula #2	
$OG = osmo_{(m)} - [2(Na^+) + glu + urea]$	-10 to +10

AG: Anion gaps
OG: Osmolar gaps
Na^+: Sodium (mEq/L)
HCO_3: Bicarbonate (mEq/L)
Cl^-: Chloride (mEq/L)
$Osmo_{(m)}$: Measured osmolarity (mmol/L)
Glu: Glucose (mmol/L)
Urea: (mmol/L)

More on Jack

In Jack's case, it is important to first look at the HCO_3^- concentration from the arterial blood gas (ABG). This patient has a metabolic acidosis, with a BD of about 19 (the normal value is 26 in this particular hospital). Rule #1 can be applied to predict the appropriate respiratory response to this degree of metabolic acidosis. The rule says that for a BD of 19, the $PaCO_2$ should decrease by 23 mmHg, and this should happen very quickly. But his ABG reveals a $PaCO_2$ of 24 mmHg, and not 17, as it should be. So we can assume there is an additional respiratory acidosis of some cause. If his $PaCO_2$ had been 17, his pH would have been over 7.3, instead of the very dangerous 7.11 he currently shows.

With any metabolic acidosis, remember the three questions to be asked:

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1. What is his AG? Using Formula #1, his AG is calculated at 31 mEq/L. Assuming a normal AG of up to 10 mEq/L, he still has 21 mEq/L of something else contributing to his metabolic acidosis.
2. Does the increase in AG equal the BD? It is close enough. His deficit is 19. There is no additional metabolic abnormality.
3. What is his OG? Using Formula #2, the OG is calculated at about 50 mmol/L, and this indicates some form of alcohol in his system.

There may be causes other than alcohol for Jack's elevated AG, but these can be ruled out by measuring his lactic acid and ketone levels, and assessing his renal function. While other exogenous substances, such as acetylsalicylic acid or cyanide, may be considered, an elevated AG with an elevated OG represents a toxic alcohol ingestion until proven otherwise. The patient's ethanol, methanol, and ethylene glycol levels should be measured.

The rules and formulas discussed in this article can be used to solve almost any of the common acid-base problems. Above all, remember the "golden rule" of acid-base interpretation—always look at the patient. Patients may be very ill, despite numbers that are not far from normal values. Alternatively, the body's ability to adapt is impressive, and patients may have very abnormal values, but compensate quite readily. Dx

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Table 3
Causes of metabolic acidosis

Increased anion gap (H^+ gain)

- Decreased fixed acid excretion
  - Acute renal failure
  - Chronic renal failure
- Increased endogenous acid production
  - Lactic acidosis
  - Ketoacidosis
- Exogenous acid ingestion
  - Normal osmolar gap (ASA, CN)
  - Increased osmolar gap (toxic alcohols, such as methanol or ethylene glycol)

Hyperchloremic/normal anion gap (HCO_3^- loss)

- Gastrointestinal tract
  - Diarrhea
- Renal
  - Diuretics
  - Renal tubular acidosis

H^+: Hydrogen
ASA: Acetylsalicylic acid
CN: Cyanide
HCO_3^-: Bicarbonate

Take-home message

- Most acid-base problems can be solved using the formulas and rules in Tables 1 and 2.
- Three questions need to be asked for each patient showing signs of acid-base disturbances:
  1. What is the AG?
  2. Does the increase in AG equal the BD?
  3. What is the OG?
- Remember, always look at the patients.

Suggested Readings

1. Williamson JC: Acid-base disorders: Classification and management strategies. Am Fam Physician 1995; 52(2):584-90.
2. Fall PJ: A stepwise approach to acid-base disorders. Practical patient evaluation for metabolic acidosis and other conditions. Postgrad Med 2000; 107(3):249-58.
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4. Swenson ER: Metabolic acidosis. Respir Care 2001; 46(4):342-53.
5. Ishihara K, Szerlip HM: Anion gap acidosis. Semin Nephrol 1998; 18(1):83-97.

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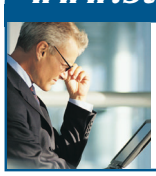
www.gasnet.org/acid-base

www.stacommunications.com



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Frequently Asked Questions

1. What substances will cause the OG to be elevated?

Sodium and its attendant anion, urea, and various sugars and alcohols will increase the osmolarity. Since Na^+, urea, and glucose are factors in the calculated osmolarity, they do not affect the OG.

2. Why does the addition of acid to the body increase the AG?

The body must maintain electroneutrality between anion and cations. Essentially, when you add an acid to the body (or produce too much), the cation (H^+) brings along its anion, contributing to the gap.

3. Why does the AG equal the BD in most cases of metabolic acidosis?

Sodium is the major cation in the body, while Cl^- and HCO_3^- are the major anions. When you add an acid to the body (or produce more), the H^+ must be buffered by HCO_3^-, decreasing the latter, and hence increasing the AG.

4. Why does HCO_3^- loss cause a metabolic acidosis with a normal AG?

As HCO_3^- is lost, Cl^- is conserved to maintain electroneutrality, with no impact on the other anions that make up the AG.

5. When might the AG exceed the BD?

An additional metabolic alkalosis could lead to this paradoxical situation. Vomiting of acid-rich gastric contents will cause a metabolic alkalosis with an initially higher than normal HCO_3^-. The change in HCO_3^- (BD) will be the same, but appear to be less than the increase in AG.