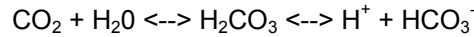


A practical guide to the solution of acid-base problems

Understanding of the carbonic acid equation is the central to understanding acid-base problems. Bicarbonate is the principal extracellular buffer.



$$\text{H}^+ = 24 \left(\frac{\text{CO}_2}{\text{HCO}_3} \right)$$

pH must be converted to H^+ (nEq/L). $\text{pH} - 7.40 = 40 \text{ nEq/L}$

Rearrangement of the carbonic acid equation results in the Henderson equation as shown above. From this equation it can be seen that changes in hydrogen ion concentration (pH) can occur only by changing the ratio of carbon dioxide to bicarbonate. This means there are only four primary acid-base disturbances. If only one of these primary acid-base disturbances is present, it is a simple disorder. If more than one primary disturbance is present simultaneously, it is known as a mixed disorder. By looking at the carbonic acid equation, the effect of changes in carbon dioxide and bicarbonate can be easily understood.

Changes in carbon dioxide are known as respiratory disturbances:

1. If carbon dioxide increases (respiratory acidosis) this is synonymous with the addition of acid (hydrogen ion). From the carbonic acid equation, increased CO_2 results in driving the reaction to the right, which results in increased hydrogen ion and increased bicarbonate.
2. Decrease in the substrate CO_2 (respiratory alkalosis) will drive the equation to the left, resulting in decreased hydrogen ion and decreased bicarbonate.

Also from the equation one can see what happens in metabolic disturbances which are caused by primary changes in plasma bicarbonate concentration (metabolic acid-base disorders).

1. Decrease in the substrate bicarbonate drives the equation to the right, resulting in decreased carbon dioxide and increased hydrogen ion.
2. Increased bicarbonate substrate drives the equation to the left, resulting in increased CO_2 and decreased hydrogen ion. These changes are summarized in the following table.

Since pH must be carefully guarded, compensatory mechanisms exist, which in the presence of an acid-base disturbances, attempt to bring pH back toward normal. The secondary response to metabolic disorders involves changes in the rate of ventilation in the lung, resulting in changes in the rate of CO_2 excretion. Therefore, hyperventilation occurs in response to metabolic acidosis to eliminate acid by the body by eliminating increased amounts of carbon dioxide. Likewise, hypoventilation occurs in response to metabolic alkalosis in order to retain acid in the form of carbon dioxide. Changes in the rate of ventilation can occur within seconds to minutes; therefore, there is no difference in terms of the secondary (compensatory) response between an acute or chronic metabolic disorder. In contrast, in respiratory disturbances the kidney must intervene to attempt to bring pH back toward normal by altering plasma bicarbonate concentration. With respiratory acidosis, the kidney enhances the rate of acid secretion into the urine. Increased acid secretion into the urine results in net addition of bicarbonate to the blood. Thus, in respiratory acidosis the kidney increases acid excretion and thereby raises plasma bicarbonate concentration. In contrast, in respiratory alkalosis, the kidney decreases its rate of acid excretion allowing for a decrease in plasma bicarbonate concentration. This process is limited; such that renal compensation cannot increase plasma bicarbonate concentration above approximately 32 mEq per liter in chronic respiratory acidosis and cannot decrease bicarbonate concentration to below about 16 mEq per liter in chronic respiratory alkalosis. There is a marked difference between acute and chronic respiratory disorders, because the kidney is rather slow to make these changes in plasma bicarbonate concentrations (2 to 3 days). There is, therefore, very limited secondary response to acute respiratory disorders.

	pH	CO ₂	HCO ₃	2° Responses
RAc	< 7.4	> 40	> 24	increased renal acid excretion (increased HCO ₃ , rarely > 32 mEq/L)
R Alk	> 7.4	< 40	< 24	Decreased renal acid excretion (decreased HCO ₃ , rarely <16 mEq/L)
MAc	< 7.4	< 40	< 24	Hyperventilate
M Alk	> 7.4	> 40	> 24	Hypoventilate (pCO ₂ usually < 55 mmHg)

A stepwise approach to assessing an acid-base problems.

1. The most useful information comes from the clinical description of the patient by history and physical examination. Based on this description what acid-base disorder is likely to be present? This should always be the initial step.
2. Look at the pH. Is it acidemic, normal, or alkaline?
3. When you have made the decision about pH look at the carbon dioxide concentration. Is it < 40, 40, or > 40? This will allow you to determine if the pH change is due to respiratory or metabolic disturbance
4. Look at the plasma bicarbonate concentration. Is it < 24, 24, or > 24?
5. Look at the plasma anion gap. This is particularly of used in assessing metabolic acidosis.
6. Look at the plasma chloride concentration, and look at it in relation to the plasma bicarbonate concentration. Note that normal anion gap metabolic acidosis is associated with hyperchloremia, a normal anion gap, and a low plasma bicarbonate. In contrast, increased anion gap metabolic acidosis has a normal plasma chloride, a reduced plasma bicarbonate, and increased unmeasured anions (increased plasma anion gap). In metabolic alkalosis, particularly those associated with chloride depletion, such as nausea and vomiting or diuretics, the plasma chloride concentration is low in association with an increased plasma bicarbonate concentration.

Golden rules of determining if an acid-base disorder is a simple or mixed disorder:

1. In a simple acid-base disturbance the plasma bicarbonate and CO₂ concentrations change in the same direction. If they don't, it is a mixed disorder.
2. In a simple acid-base disorder, the appropriate secondary response must be present. If they are not present, it is a mixed disorder.
3. In a simple acid-base disorder, the secondary response never fully corrects the pH. They bring pH back toward normal. If they fully correct the pH or overshoot, it is a mixed acid-base disorder.

Clinical description in pointing toward specific acid-base disorders:

1. Tachypnea suggests hyperventilation and, therefore, respiratory alkalosis.
2. Obstruction to airway flow or inability to breathe, such as oversedation, suggest impaired gas exchange by the lung; therefore, increased carbon dioxide concentration which is respiratory acidosis.
3. Nausea and vomiting, chloride depletion metabolic alkalosis
4. Diuretics, chloride depletion metabolic alkalosis
5. Diarrhea results in direct bicarbonate loss from the gut, resulting in normal anion gap metabolic acidosis.
6. Chronic renal insufficiency when mild to moderate typically results in normal anion gap metabolic acidosis. More advanced renal failure results in the retention of acid anions, such as phosphate and urates, and has an increased anion gap metabolic acidosis.
7. Type I diabetic, off insulin, suggests ketoacidosis, which is a cause of increased anion gap metabolic acidosis.
8. Circulatory shock from any cause will tend to result in anaerobic metabolism leading to lactic acidosis with an increased plasma anion gap.