

# HOSPITAL PHYSICIAN®

## NEPHROLOGY BOARD REVIEW MANUAL

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## Metabolic Acidosis

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### Metabolic Acidosis

Metabolic acidosis is characterized by an abnormal decrease in plasma bicarbonate ( $\text{HCO}_3^-$ ) concentration and a decrease in arterial pH, which produce acidemia and compensatory hyperventilation that results in a reduced  $\text{PCO}_2$ . Thus, in a pure metabolic acidosis, measurements of  $\text{HCO}_3^-$  concentration, pH, and  $\text{PCO}_2$  all move in the same direction. Measurements of arterial pH distinguish metabolic acidosis that is induced by decreased  $\text{HCO}_3^-$  concentration from metabolic acidosis that results from a compensatory response to respiratory alkalosis. A plasma  $\text{HCO}_3^-$  concentration less than 10 mEq/L is a definite indicator of metabolic acidosis, because renal compensation to respiratory alkalosis does not produce such a marked decrease in  $\text{HCO}_3^-$  concentration. Therefore, all hypobicarbonatemia is abnormal and all noncompensatory hypobicarbonatemia reflects a metabolic acidosis.

Physicians often misdiagnose or fail to recognize acid-base disorders, particularly metabolic acidosis. A survey conducted at a university teaching hospital showed that only 40% of the test samples of arterial blood gases analyzed by physicians were correctly interpreted.<sup>1</sup> Another survey showed that incorrect acid-base interpretations led to errors in patient management in one third of the arterial blood gas samples interpreted.<sup>2</sup> These surveys point to the need for a better understanding of the mechanisms of acid-base disorders.

Metabolic acidosis may be induced by 2 basic mechanisms. First, an inability of the kidney to excrete hydrogen ion ( $\text{H}^+$ ) from dietary sources may result in a slowly developing acidosis. Second, increased generation of  $\text{H}^+$  secondary to  $\text{H}^+$  addition or  $\text{HCO}_3^-$  loss may result in rapidly developing acidosis and may be due to derangement in gut function, metabolic derangements, or exogenous toxins (eg, methanol, ethylene glycol).

This review presents a structured approach to the evaluation of patients with metabolic acidosis. Brief reviews of body handling of acids and the anion gap (AG) are followed by 3 patient scenarios that explore the major causes and treatments of metabolic acidosis.

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#### BODY HANDLING OF ACIDS

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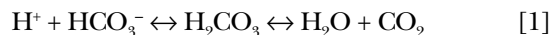
$\text{H}^+$  homeostasis is a dynamic process that involves  $\text{H}^+$  production by metabolism of energy sources, buffering mechanisms that prevent significant fluctuations in the free proton concentration, respiratory compensation, and renal adaptation or correction (eg, disposal).

#### PROTON BUFFERING

Buffering refers to the ability of a solution containing a weak or poorly dissociated acid and its anion (ie, conjugate base) to resist a change in pH upon addition of a strong acid or base. In the body fluids, free  $\text{H}^+$  concentration is extremely low compared with other ions. In fact, free  $\text{H}^+$  concentration is approximately 40 nEq/L, which is one millionth the concentration of ions such as sodium, potassium, and chloride. Maintaining a stable  $\text{H}^+$  concentration is important because minute changes in  $\text{H}^+$  concentration produce significant effects on cellular enzymatic activity<sup>3,4</sup> and thus critically impair tissue and organ performance. Buffering occurs in the extracellular fluid (ECF), intracellular fluid (ICF), and bone.

#### Extracellular Buffering

In the ECF,  $\text{HCO}_3^-$  plays a vital role in the initial buffering of an acid load, which occurs within minutes after addition of  $\text{H}^+$  and accounts for approximately 40% of the body's total buffering capacity.  $\text{HCO}_3^-$  combines with  $\text{H}^+$  according to the following equation:



Because the concentration of carbonic acid is very low relative to the concentrations of dissolved  $\text{HCO}_3^-$  and carbon dioxide, the law of mass action can be used to calculate  $\text{H}^+$  concentration as follows:

$$[\text{H}^+] = \frac{K'_a \times (0.03) (\text{PCO}_2)}{[\text{HCO}_3^-]} \quad [2]$$

where  $K'_a$  is the dissociation constant for the reaction and