

# Determinants of Plasma Acid-Base Balance

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Acid-base abnormalities are extremely common in modern ICUs. Although most cases are mild and self-limiting, there are certain circumstances in which acid-base derangements are dangerous. Such is the case when the disorders are extreme (eg, pH <7.0 or >7.7); especially when the acid-base derangement develops quickly. Such severe abnormalities can be the direct cause of organ dysfunction. Clinical manifestations can include cerebral edema, seizures, decreased myocardial contractility, pulmonary vasoconstriction, and systemic vasodilation to name a few. Furthermore, even less extreme derangements may produce harm because of the patient's response to the abnormality. For example, a spontaneously breathing patient who has metabolic acidosis attempts to compensate by increasing minute ventilation. The workload that is imposed by increasing minute ventilation can lead to respiratory muscle fatigue with respiratory failure or diversion of blood flow from vital organs to the respiratory muscles which results in organ injury. Acidemia is associated with increased adrenergic tone and, on this basis, can promote the development of cardiac dysrhythmias in critically ill patients, or increase myocardial oxygen demand in patients who have myocardial ischemia. In such cases, it may be prudent to treat the underlying disorder and to provide symptomatic treatment for the acid-base disorder. Accordingly, it is important to understand the causes of acid-base disorders and the limitations of various treatment strategies. Finally, emerging evidence suggests that changes in acid-base variables influence immune effector cell function [1,2]. Thus, avoiding acid-base derangements may prove to be important in the management of critically ill patients for their own sake and

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for the sake of standardizing study protocols that attempt to manipulate immunologic responses (eg, anticytokine therapies).

### Classification and quantification of acid-base disorders

To diagnose, treat, and it is hoped that, to avoid acid-base disorders it is paramount that the critical care clinician recognize acid-base disturbances and understand how and why they occur. To understand acid-base physiology it is important to agree on how to describe and measure it. Since Sørensen first introduced the pH notation, we have used the pH scale to quantify acid-base balance. The pH scale has a tremendous advantage because it lends itself to colorimetric and electrometric techniques. There also is some physiologic relevance to the logarithmic pH scale [3]. pH is a confusing variable, however. It is a nonlinear transformation of  $H^+$  concentration—the logarithm of its reciprocal. Strictly speaking, pH only can be thought of as a dimensionless representation of  $H^+$  concentration and is not, itself, a concentration. pH actually is the logarithmic measure of the volume that is required to contain 1 Eq of  $H^+$ . In blood plasma at pH 7.4, this volume is roughly 25 million liters [4].

Regardless of how we express the concentration of  $H^+$ , either directly or as the pH, it generally is accepted that changes in blood  $H^+$  concentration occur as the result of changes in volatile ( $PCO_2$ ) and nonvolatile acids (eg, hydrochloric, sulfuric, lactic). Clinically, changes in volatile acid are referred to as “respiratory” and changes in nonvolatile acids are referred to as “metabolic.” There are three major methods of quantifying (describing) acid-base disorders; each differs only in assessment of this latter, “metabolic” component. These three methods quantify the metabolic component by using (1)  $HCO_3^-$  (in the context of  $PCO_2$ ); (2) the standard base excess (SBE); or (3) the strong ion difference (SID). Although there has been significant debate about the accuracy and usefulness of each method compared with the others, all three yield virtually identical results when used to quantify the acid-base status of a given blood sample [5,6]. The only differences between these three approaches are conceptual (ie, in how they approach the understanding of the mechanism) [7–9].

#### *Beyond Henderson and Hasselbalch*

Since Hasselbalch adapted the Henderson equation to the pH notation of Sørensen, the following equation has been used to understand the relationship between respiratory and metabolic acid-base variables:

$$pH = pK + \log \left[ \frac{HCO_3^-}{(0.03 \times PCO_2)} \right]$$

This is the Henderson-Hasselbalch (HH) equation and it is important to realize what this equation tells us. An increase in  $PCO_2$  will result in a decrease in pH

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