Understanding acid-base balance

Find out how to interpret values and steady a disturbed equilibrium in an acutely ill patient.

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Many critical illnesses can upset a patient's acid-base balance, and a disturbance in acid-base equilibrium may indicate other underlying diseases or organ damage. Accurately interpreting acid-base balance requires simultaneous measurements of arterial pH and plasma electrolytes, as well as knowledge of compensatory physiologic mechanisms.

In this article, we'll review normal acid-base physiology, acid-base disturbances, and lab techniques and mathematical calculations used to identify the cause of acid-base derangements. Lastly, we'll discuss potential treatments for acid-base disturbances.

What's normal?

A normal range for arterial pH is 7.35 to 7.45. Acidosis is a pH less than 7.35; alkalosis is a pH greater than 7.45. Because pH is measured in terms of hydrogen (H^+) ion concentration, an increase in H^+ ion concentration decreases pH and vice versa. Changes in H^+ ion concentration can be stabilized through several buffering systems: bicarbonate-carbonic acid, proteins, hemoglobin, and phosphates.

Acidosis, therefore, can be described as a physiologic condition caused by the body's inability to buffer excess H^+ ions. At the other end, alkalosis results from a deficiency in H^+ ion concentration. Acidemia and alkalemia refer to the process of acidosis or alkalosis, respectively, occurring in arterial blood.

Body acids are formed as end products of cellular metabolism. Under normal physiologic conditions, a person generates 50 to 100 mEq/day of acid from metabolism of carbohydrates, proteins, and fats. In addition, the body loses base in the stool. In order to maintain acid-base homeostasis, acid production must balance the neutralization or excretion. The lungs and kidneys are the main regulators of acid-base homeostasis. The lungs release CO_2, an end product of carbonic acid (H_2CO_3). The renal tubules, with the regulation of bicarbonate (HCO_3^-), excrete other acids produced from the metabolism of proteins, carbohydrates, and fats.[1]

Compensating for changes

The body has three compensatory mechanisms to handle changes in serum pH:

• **Physiologic buffers,** consisting of a weak acid (which can easily be broken down) and its base salt or of a weak base and its acid salt. These buffers are the bicarbonate-carbonic acid buffering system, intracellular protein buffers, and phosphate buffers in the bone.

• **Pulmonary compensation,** in which changes in ventilation work to change the partial pressure of arterial carbon dioxide ($Paco_2$) and drive the pH toward the normal range. A drop in pH, for example, results in increased ventilation to blow off excess CO_2. An increase in pH decreases ventilatory effort, which increases $Paco_2$ and lowers the pH back toward normal.

• **Renal compensation,** which kicks in when the other mechanisms have been ineffective, generally after about 6 hours of sustained acidosis or alkalosis. While respira-

tory compensation occurs almost immediately, renal mechanisms can take hours to days to make a difference. In acidosis the kidneys excrete H^+ in urine and retain HCO_3^-. In alkalosis, the kidneys excrete bicarbonate and retain H^+ in the form of organic acids, resulting in near-normalization of pH.[2,3] Lastly, bone may also serve as a buffer because it contains a large reservoir of bicarbonate and phosphate and can buffer a significant acute acid load. Patients who have low albumin levels and bone density due to malnutrition or chronic disease, and anemic patients, have an ineffective buffering capability.[4]

Common acid-base upsets

Generally, if your patient has changes in acid-base homeostasis, you'd look for the cause first before intervening to normalize the pH. But because some acid-base disturbances have a limited number of causes, you can systematically eliminate some potential causes.

Start by looking at the patient's arterial blood gas analysis. Many disorders are mild and don't require treatment, and in some cases, too-hasty treatment can do more harm than the imbalance itself. Also, critically ill patients may have more than one acid-base imbalance simultaneously.

The most common acid-base derangements can be divided into four categories: metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis. Let's look at each and how you'd respond.

Metabolic acidosis

Metabolic acidosis is an increase in the amount of absolute body acid, either from excess production of acids or excessive loss of bicarbonate, sodium, and potassium. Causes of metabolic acidosis include lactic acidosis, diabetic ketoacidosis, and loss of bicarbonate through severe diarrhea or bicarbonate wasting through the kidneys or gastrointestinal (GI) tract.

In general, the kidneys attempt to preserve sodium by exchanging it for excreted H^+ or potassium. In the presence of an H^+ load, H^+ ions move from the extracellular fluid into the intracellular fluid.[2] For this process to occur, potassium moves outside the cell into the extracellular fluid to maintain electroneutrality. In severe acidosis, significant overall depletion of total body potassium stores can occur despite serum hyperkalemia. This is why I.V. potassium is given to patients with diabetic ketoacidosis early in treatment, despite the often-elevated serum potassium level.[5,6] External and internal potassium balances are regulated to maintain an extracellular fluid concentration of 3.5 to 5.5 mEq/L and a total body content of about 50 mEq/kg (40 mEq/kg in females).[6]

Metabolic alkalosis

Metabolic alkalosis occurs when HCO_3^- is increased, usually as the result of excessive loss of metabolic acids. Causes of metabolic alkalosis include diuretics, secretory adenoma of the colon, emesis, hyperaldosteronism, Cushing's syndrome, and exogenous steroids.

Some causes of metabolic alkalosis respond to treatment with 0.9% sodium chloride solution. If the patient's urine chloride concentration is less than 15 mmol/L, his metabolic alkalosis is saline-responsive; urine chloride levels above 25 mmol/L indicate nonsaline-responsive metabolic alkalosis.[2,7] The mechanisms resulting in saline-responsive metabolic alkalosis include GI loss, diuresis, or renal compensation from hypercapnia. Nonsaline responsive metabolic alkalosis results from mineralocorticoid excess or potassium depletion.

Fluid administration is the foundation for treatment for saline-responsive metabolic alkalosis.[8] In cases of extreme alkalosis, the patient may be given dilute hydrochloric acid. Saline-resistant alkalosis is treated by addressing the underlying etiology.

Respiratory acidosis

In respiratory acidosis, the patient's pH is less than 7.35 and his $PaCO_2$ is above 45 mm Hg (the upper limit of normal). Alveolar hypoventilation is the only mechanism that causes hypercarbia, or a $PaCO_2$ above the upper limit of normal. The amount of alveolar ventilation necessary to maintain normal $PaCO_2$ varies depending upon CO_2 produced.

The relationship between $PaCO_2$ and plasma HCO_3^- determines arterial pH. Generally, acute increases in $PaCO_2$ are accompanied by only minimal changes in serum HCO_3^-. However, over a period of 1 to 3 days, renal conservation of HCO_3^- results in an increase in pH.[9]

Chronic respiratory acidosis occurs secondary to a chronic reduction in alveolar ventilation—for example, in chronic lung diseases such as chronic obstructive pulmonary disease (COPD). Acute respiratory acidosis is caused by an acute change in alveolar ventilation; respiratory depression from acute opioid ingestion is one cause. Treatment for respiratory acidosis is largely supportive, but if opioid ingestion is suspected, I.V. naloxone may be given as an antidote.

Respiratory alkalosis

Common in critical care, respiratory alkalosis occurs when $PaCO_2$ is reduced, causing an increase in pH. The most common cause of respiratory alkalosis is increased alveolar ventilation, which can happen in hyperventilation, mechanical overventilation, hepatic

disease, pregnancy, and septicemia.

Determining appropriate compensatory changes in HCO_3^- is key to determining if the patient also has a concomitant metabolic disorder. In chronic respiratory alkalosis, the compensatory mechanisms result in mild reduction in plasma HCO_3^- levels to maintain a near normal or normal pH. This causes a mixed acid-base disorder, which will be discussed later.

Treatment of respiratory alkalosis is directed at discovering and correcting the underlying etiology. For example, if a patient is hyperventilating from anxiety, have him breathe into a paper bag. In mechanically ventilated patients with mechanical overventilation, reducing the minute ventilation or tidal volume will increase $PaCO_2$ and reduce pH. Monitor the patient closely, because a rapid reduction of $PaCO_2$ in a patient with chronic respiratory alkalosis may cause acute metabolic acidosis.[10]

Mixed acid-base imbalances

When a patient has two or three acid-base imbalances simultaneously, he's said to have a mixed acid-base imbalance.[7] Examples include:
• respiratory alkalosis or acidosis that shrouds a metabolic acidosis or alkalosis
• metabolic alkalosis or acidosis that shrouds another metabolic alkalosis acidosis.

Combined respiratory and metabolic imbalances may occur when the respiratory system compensates inappropriately for metabolic imbalances. Look at the difference between the patient's observed $PaCO_2$ and the calculated changes in $PaCO_2$, or the observed or expected change in HCO_3^-.[2,11] If the observed $PaCO_2$ is higher than the calculated $PaCO_2$, the patient has respiratory acidosis with a mixed metabolic disturbance. If the observed $PaCO_2$ is lower than the calculated $PaCO_2$, the patient has respiratory alkalosis mixed with a metabolic imbalance. Generally, the $PaCO_2$ should be similar to the two last digits of the patient's pH. For example, if the patient's pH is 7.25, you'd expect his $PaCO_2$ to be about 25 mm Hg.[1]

Mixed metabolic acidosis and alkalosis can be identified by calculating the anion gap.[11] The anion gap is an approximate measure of the additional amount of acid in the body; the HCO_3^- should decrease by about an amount equaling the increase in the anion gap. If the HCO_3^- is higher than the calculated increase of the anion gap, a chief metabolic alkalosis is mixed with the metabolic acidosis. Conversely, if the HCO_3^- is lower than the increase of the

anion gap, then a non-anion gap metabolic acidosis is considered to be present and is worsening the anion gap acidosis. For more examples, see *Comparing acid-base imbalances*.

Caring for the critically ill

Acid-base imbalances are common in critically ill patients. By understanding the basic physiology of acid-base balance and what can go wrong, you can help your patient get back in balance. ∎

Comparing acid-base imbalances

Condition	pH	$PaCO_2$	HCO_3^-
Pure respiratory alkalosis	High	Low	Normal
Pure respiratory acidosis	Low	High	Normal
Pure metabolic alkalosis	High	Normal	High
Pure metabolic acidosis	Low	Normal	Low
Metabolic alkalosis with partial respiratory compensation	High	High	High
Metabolic acidosis with partial respiratory compensation	Low	Low	Low

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