How to Interpret Acid Base

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A proper understanding of the terms acidosis, alkalosis, acidemia, and alkalemia is necessary to differentiate simple from mixed acid base disorders. Acidosis and alkalosis refer to the pathophysiologic processes that cause net accumulation of acid or alkali in the body, whereas acidemia and alkalemia refer specifically to the pH of extracellular fluid. In acidemia, the extracellular fluid pH is less than normal and the $[H^+]$ is higher than normal. In alkalemia, the extracellular fluid pH is higher than normal and the $[H^+]$ is lower than normal. Due to the effectiveness of compensatory mechanisms, animals can have acidosis or alkalosis but not acidemia or alkalemia. For example, a dog with chronic respiratory alkalosis may have a blood pH that is within the normal range. Such a patient has alkalosis, but does not have alkalemia.

The primary acid base disorders are divided into metabolic and respiratory disturbances: metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis. The Henderson-Hasselbach equation in its clinically relevant form emphasizes the relationship between the metabolic and respiratory systems in determining extracellular fluid pH:

$$pH = pK_a + \log([HCO_3^-]/.03 pCO_2)$$
$$pH = 6.1 + \log([HCO_3^-]/.03 pCO_2)$$

Traditionally, the kidneys have been considered responsible for regulation of the metabolic component (blood bicarbonate concentration, $[HCO_3^-]$) and the lungs for regulation of the respiratory component (partial pressure of CO_2, $[pCO_2]$). In this form, the Henderson-Hasselbach equation makes it clear that the pH of extracellular fluid is determined by the *ratio* of the bicarbonate concentration and pCO_2.

Each primary (metabolic or respiratory) acid base disturbance is accompanied by a secondary (opposing) response in the other system (respiratory or metabolic). Blood pH is returned nearly, but not completely, to normal, and overcompensation does not occur. Understanding these principles is central to recognition of mixed acid base disturbances. A simple acid base disorder is defined as a primary disturbance and the expected adaptive (or secondary) response in the opposite system. A mixed acid base disturbance results from two (or rarely three) separate primary disturbances present simultaneously in the same individual. For example, the independent occurrence of metabolic acidosis, metabolic alkalosis, and a primary respiratory disturbance is referred to as a "triple disorder." Four disorders cannot coexist in one individual because hyperventilation (respiratory alkalosis) and hypoventilation (respiratory acidosis) cannot occur at the same time in one patient. The key to interpreting mixed acid base disturbances is to know the expected adaptive (compensatory) responses. Unfortunately, the magnitude of expected compensation in a given clinical situation is cannot be known with certainty, and data in dogs have been derived mainly from experiments using normal animals. These compensatory rules should be applied to cats with extreme caution because values have been derived from a limited number of normal cats with experimentally induced acid-base disorders. The expected compensatory responses for dogs are shown in Table 1.

Table 1: Compensatory responses for acid base disturbances in dogs

Primary disorder	Compensatory response
Metabolic acidosis	0.7-1.2 mm ↓ pCO2 per 1.0 mEq/L ↓ HCO3-
Metabolic alkalosis	0.7 mm ↑ pCO2 per 1.0 mEq/L ↑ HCO3-
Acute respiratory acidosis	0.15 mEq/L ↑ HCO3- per 1.0 mm ↑ pCO2
Chronic respiratory acidosis	0.35 mEq/L ↑ HCO3- per 1.0 mm ↑ pCO2
Acute respiratory alkalosis	0.25 mEq/L ↓ HCO3- per 1.0 mm ↓ pCO2
Chronic respiratory alkalosis	0.55 mEq/L ↓ HCO3- per 1.0 mm ↓ pCO2

Note that there is only one guideline for respiratory compensation in metabolic disorders, but two guidelines for metabolic compensation in respiratory disorders. The reason for the difference is that when a metabolic acid load titrates bicarbonate, the sole response is hyperventilation to lower pCO_2. Respiratory compensation for metabolic disorders normally is complete within 48 hours. When H_2CO_3 is formed from CO_2 and H_2O in response to respiratory acidosis, however, the resultant $[H^+]$ initially are buffered by combination with hemoglobin in red cells and intracellular phosphates and proteins in other cells. This response results in the initial smaller increase in blood bicarbonate concentration observed (0.15 mEq/L increase in HCO_3^- per 1.0 mm increase in pCO_2). Over the next 2 to 6 days, the kidneys increase their generation of bicarbonate. This response accounts an additional increase in blood bicarbonate concentration, and necessitates use of a second compensatory guideline (0.35 mEq/L increase in HCO_3^- per 1.0 mm

increase in pCO_2). Note also that when speaking of responses to acid base disturbances, the term "acute" refers to a process that has been present for less than 48 hours whereas the term "chronic" refers to a process that has been present for more than 48 hours.

When attempting to interpret blood gas data and decide whether or not an acid base disorder is present, the clinician should strive to answer 4 questions:

1.   Is an acid base disturbance present?
2.   What is the primary disturbance?
3.   Is the secondary (adaptive) response as expected?
4.   What underlying disease process is responsible for the acid base disturbance?

The application of these principles is best illustrated by considering several cases. First, consider arterial blood gas results from a dog presented for evaluation of acute renal failure: pH 7.27, HCO_3^- 12 mEq/L, pCO_2 27 mmHg. For the purposes of these case illustrations, consider normal values as: pH 7.39, HCO_3^- 22 mEq/L, and pCO_2 37 mmHg. Is an acid base disturbance present? Yes, this is obvious from the pH. Of what general type is the acid base disturbance? Clearly, it is an acidosis, and the patient is acidemic (pH 7.27 < 7.39). Is the acidosis metabolic or respiratory? The pCO_2 is low, and thus the primary disturbance can't be respiratory acidosis. The HCO_3^- concentration is low, and thus the primary disturbance must be metabolic acidosis. To assess the possibility of a mixed disturbance, it must be determined whether or not the secondary (adaptive) response is as expected. The observed HCO_3^- is 10 mEq/L *lower* than normal (22-12). A normal dog can lower its pCO_2 0.7-1.2 mmHg for every 1.0 mEq/L decrement in HCO_3^-, and we can use 1.0 mmHg as average figure. Therefore, the expected pCO_2 is 37-10 or 27 mmHg. The observed pCO_2 is 27 mmHg, and the conclusion is that the adaptive response is appropriate. This patient has a simple metabolic acidosis with appropriate respiratory compensation.

Next, consider arterial blood gas results from a dog sick for 1 week with coughing and dyspnea: pH 7.33, HCO_3^- 29 mEq/L, pCO_2 57 mmHg. Is an acid base disturbance present? Yes, although less obvious than the previous case, this can be concluded from the pH. Of what general type is the acid base disturbance? Again, it is an acidosis and the patient is slightly acidemic (pH 7.33 < 7.39). Is the acidosis metabolic or respiratory? The HCO_3^- is high and thus it can't be metabolic acidosis. The pCO_2 is high and therefore it must be respiratory acidosis. To assess the possibility of a mixed disturbance, it must be determined whether or not the secondary (adaptive) response as expected. The observed pCO_2 is 20 mmHg *higher* than normal (57-37). A normal dog can increase its HCO_3^- 0.35 mEq/L for every 1.0 mmHg increment in pCO_2 in a chronic disturbance (i.e. one that has been present more than 48 hours). Therefore, the expected HCO_3^- is 22+7 or 29 mEq/L. The observed HCO_3^- is 29 mEq/L, and the conclusion is that the adaptive response is appropriate. This patient has a simple respiratory acidosis with appropriate metabolic compensation. Even in simple disturbances, calculated compensatory pCO_2 and HCO_3^- values usually won't match the observed values because calculations are based on "average" values. Thus, a mixed disturbance should not be diagnosed unless the calculated value is > 2 to 3 mmHg (pCO_2) or mEq/L (HCO_3^-) different from the observed value. Now, let's consider some more difficult examples.

Consider arterial blood gas results from a seriously ill dog: pH 7.05, HCO_3^- 12 mEq/L, pCO_2 44 mmHg. Is an acid base disturbance present? Absolutely, just look at the pH. Of what general type? Clearly, a severe acidosis (pH 7.05 << 7.39). Is the acidosis metabolic or respiratory? The pCO_2 is high and thus it could be respiratory acidosis, but the HCO_3^- is low as expected for metabolic acidosis. Is the secondary (adaptive) response as expected? Clearly it is not. If this disorder were a simple metabolic acidosis, the pCO_2 would be low in response. If it were a simple respiratory acidosis, the HCO_3^- would be high in response. The inescapable conclusion is that this disturbance represents a mixed metabolic and respiratory acidosis. The extremely low pH alerts you to the likelihood of a mixed disorder in which both independent primary disturbances are dragging the pH in the same direction.

Consider arterial blood gas results from a dog with sudden onset of gastric dilatation and volvulus: pH 7.38, HCO_3^- 12 mEq/L, pCO_2 21 mmHg. Is an acid base disturbance present? If so, it's not obvious from the pH. The pCO_2 is low and thus it could be respiratory alkalosis, but the HCO_3^- also is low and thus it could be metabolic acidosis. Given these confusing findings, it is best to consider both possibilities. If it is a primary metabolic acidosis, there has been a 10 mEq/L decrement in HCO_3^- (22-12) and the expected pCO_2 would be 27 mmHg (37-10). The observed pCO_2 = 21 mmHg. If it is a primary acute respiratory alkalosis, there has been a 16 mmHg decrement in pCO_2 (37-21) and the expected HCO_3^- would be 18 mEq/L (22-4). The observed HCO_3^- is 12 mEq/L. Neither is as expected. Thus, the conclusion is that this is a mixed metabolic acidosis and respiratory alkalosis. Is mixed metabolic acidosis and respiratory alkalosis compatible with acute gastric dilatation-volvulus? Yes, there may be primary metabolic acidosis due to shock and decreased tissue perfusion and primary respiratory alkalosis due to hyperventilation induced by pain or septicemia. The normal (and confusing) pH is a consequence of a mixed disturbance with independent disturbances that are pulling the pH in opposite directions, resulting in a normal value of 7.38. If this dog had been sick with some other disease for 1 week or more, these results could have represented a simple chronic respiratory alkalosis. The 16 mmHg decrement in pCO_2 (37-21) predicts a HCO_3^- of 13.2 mEq/L (22-8.8) which is < 2 mEq/L different from the observed HCO_3^- of 12 mEq/L. Thus, a conclusion of simple chronic respiratory alkalosis with appropriate metabolic compensation would be justified in such a situation.

References

deMorais HAS and Leisewitz AL: Mixed Acid-base disturbances. In DiBartola SP: Fluid, Electrolyte, and Acid Base Disorders in Small Animal Practice (ed 4), Elsevier, St. Louis, 2012, pp 302-315.

DiBartola SP: Introduction to Acid-Base Disorders. In DiBartola SP: Fluid, Electrolyte, and Acid Base Disorders in Small Animal Practice (ed 4), Elsevier, St. Louis, 2012, pp 231-252.

DiBartola SP: Metabolic Acid-Base Disorders. In DiBartola SP: Fluid, Electrolyte, and Acid Base Disorders in Small Animal Practice (ed 4), Elsevier, St. Louis, 2012, pp 253-286.