

Blood Gases - Interpretation and Clinical Utility

Arterial blood gas analysis provides information regarding the patient's acid-base status, ventilation and oxygenation. Venous blood gases provide information regarding the patient's acid-base status and ability to ventilate. Blood gas instruments measure the pH (H^+ concentration), the partial pressure of carbon dioxide (PCO_2) and the partial pressure of oxygen (PO_2). With this information the machine can calculate bicarbonate (HCO_3^-) levels and base excess (BE) and the percent hemoglobin saturation with oxygen (SO_2)

The BE and HCO_3^- serve as measures of the metabolic component of acid-base balance, while the PCO_2 evaluates ventilation and represents the respiratory component of the patient's acid-base balance. The patient's oxygenation can be evaluated with arterial partial pressure oxygen measurements (P_aO_2) and additional calculations derived from this value

INTERPRETATION

Rule 1: What is the pH of the blood? Acidemia technically exists if the blood $pH < 7.35$ and alkalemia is present if the blood $pH > 7.45$, clinically we should become concerned with a $pH < 7.20$ or a $pH > 7.55$

Rule 2: Is there a respiratory component? *Hypoventilation* is characterized by increases in the PCO_2 as the CO_2 is retained within the blood ($PaCO_2 > 45$ mm Hg). CO_2 represents a volatile acid so this leads to respiratory acidosis. *Hyperventilation* is characterized by decreases in PCO_2 as the CO_2 is blown off from the alveoli. This leads to a respiratory alkalosis ($PaCO_2 < 35$ mm Hg).

Rule 3: Is there a metabolic component? The metabolic contribution to the acid base balance can be assessed with the Base excess (BE). The BE takes into account all of the body's buffer systems, including bicarbonate, to predict the quantity of acid or alkali required to return the extracellular fluid compartment to neutrality (pH 7.4) while the $PaCO_2$ is held constant at 40 mmHg. Its relevance stems from standardizing for the effects of the respiratory contribution; hence, BE is representative of all the metabolic acid-base disturbances. Normally, BE is 0 ± 4 mEq/L. More negative values represent a metabolic acidosis ($BE < -4$), while more positive values ($BE > +4$) represent a metabolic alkalosis. Alternatively, the HCO_3^- can be satisfactorily used to estimate the metabolic component of an acid-base disturbance. Normally, HCO_3^- concentration is 20-24 mEq/L, with $HCO_3^- < 20$ mEq/L representing a metabolic acidosis and $HCO_3^- > 24$ mEq/L representing a metabolic alkalosis.

Rule 4: Determine which component is the primary disorder: The primary disorder is the component that has changed in the same manner as the pH. For example, if the pH < 7.35 and the HCO₃⁻ is < 20mEq/L (or BE < -4), the primary disorder is metabolic [acidosis].

If the pH is < 7.35 and the pCO₂ is > 45mmHg, the primary disorder is respiratory [acidosis].

If both the HCO₃⁻ and pCO₂ have changed in the manner of the pH, a mixed acid-base disturbance exists. An example of a mixed disorder would be a hit by car cat patient with lactic acidosis from shock (pH < 7.35 and the HCO₃⁻ < 20mEq/L (or BE < -4) and, concurrent hypoventilation (PCO₂ > 45mmHg) from a pneumothorax preventing proper lung expansion. Both metabolic and respiratory acidosis in this patient will contribute to pronounced acidemia (pH < <7.35), as there are no compensatory responses and, furthermore, both processes contribute to the acidemia.

Rule 5: Is there a compensatory response? Simple acid base disorders are due to any of the four primary acid-base disturbances, metabolic or respiratory in origin, with an anticipated compensatory change. Figure 1 summarizes the four primary acid-base disorders and their compensatory changes.

| CONDITIONS | | PRIMARY DISORDER | COMPENSATION |
|------------|---------------------------------------|-----------------------|---------------------------------------|
| ↓pH | ↓ HCO ₃ ⁻ (↓BE) | Metabolic Acidosis | ↓PCO ₂ |
| ↑pH | ↑ HCO ₃ ⁻ (↑BE) | Metabolic Alkalosis | ↑PCO ₂ |
| ↓pH | ↑PCO ₂ | Respiratory Acidosis | ↑ HCO ₃ ⁻ (↑BE) |
| ↑pH | ↓PCO ₂ | Respiratory Alkalosis | ↓ HCO ₃ ⁻ (↓BE) |

Figure 1: ***The four primary acid-base disorders and their compensatory changes.*** The primary disorder leads to a change in pH, while compensatory changes attempt to bring the pH back to neutral. Compensatory changes in pCO₂ and HCO₃⁻ parallel each other, as shown by the direction of the arrows in any given row.

The most common acid-base disturbance encountered in small animals is metabolic acidosis. If metabolic acidosis is the primary disturbance, it will be represented by a pH < 7.35, a BE < -4 or HCO₃⁻ < 20 mEq/L, and a compensatory decrease in the PCO₂ (< 35mmHg) in an attempt to blow off excess acid load.

The most common causes of each acid-base disorder and treatment are:

1.) Metabolic acidosis: Diabetic ketoacidosis, renal insufficiency, lactic acid production (shock, sepsis, pancreatitis, hypoxemia), exogenous toxins (ethylene glycol, salicylic acid), diarrhea

Metabolic acidosis should be initially addressed with IV fluid support to re-establish tissue perfusion and help restore appropriate oxygen delivery to the tissues. Sodium bicarbonate should only be considered in severe cases of metabolic acidosis (pH <7.1, $\text{HCO}_3^- < 12\text{mEq/L}$) after verifying that perfusion is adequate, underlying causes for the persistent metabolic acidosis have been addressed, and the patient is ventilating appropriately.

2.) Respiratory acidosis (hypoventilation): Common causes of respiratory depression include those affecting the neurologic control of respiration (e.g. anesthesia, sedation, CNS disease), those affecting breathing mechanics (e.g. diaphragmatic hernia, pleural space disease), and those affecting the proper flow of air through the airways (e.g. upper or lower airway obstruction) or the alveoli.

Severe respiratory acidosis may require intubation and positive pressure ventilation if it persists despite reversal of any drug that may depress neurologic control of respiration, including, inhalant anesthesia. It is usually accompanied by hypoxemia, so oxygen supplementation is concurrently administered. Sodium bicarbonate is NOT indicated for respiratory acidosis.

3.) Metabolic alkalosis: Underlying causes of metabolic alkalosis are: Upper GI obstruction, potassium depleting diuretic therapy, hyperadrenocorticism, exogenous steroid therapy, and previous bicarbonate therapy.

0.9% NaCl, which is an acidifying crystalloid, should be administered in this rare condition.

4.) Respiratory alkalosis (hyperventilation): Common causes of respiratory alkalosis include pain and anxiety, hypoxemia, pulmonary disease, and overzealous manual or mechanical ventilation.

Remember that patients will also commonly hyperventilate as a compensation for a metabolic acidotic state (DKA, Renal disease).

Rule 6: Evaluate Oxygenation

With an arterial sample, we can also assess oxygenation. Anytime a low PaO_2 ($<80\text{mm Hg}$) is obtained from an arterial sample in a patient breathing room air (an inspired oxygen fraction of 21% or an $\text{FIO}_2 = 0.21$), the Alveolar gas equation should be calculated to determine the Alveolar-arterial (A-a) oxygen gradient. This gradient provides a measure of the adequacy of oxygen transfer across the alveolar membrane into the pulmonary capillaries perfusing these alveoli (i.e. oxygen loading into the blood). It does so by accounting for the effects of altitude, inspired oxygen percentage, and ventilation on the patient's oxygenation. Serial calculations of the Alveolar-arterial oxygen gradient allows for objective estimates of pulmonary function over time.

Most pulmonary diseases will alter the ventilation to perfusion ratio (i.e. V/Q mismatch) of individual alveoli leading to a reduction in oxygen loading into the blood and a corresponding lower PaO_2 . V/Q mismatches lead to an increase in the A-a gradient. Normal values for the A-a gradient are 5-15 mm Hg. Figure 2 shows a simplified, clinically useful calculation, used to obtain the A-a gradient. Figure 3 depicts the complete Alveolar Gas Equation with all the parameters that play a make up the calculation.

| | |
|------------------------------------|---|
| Alveolar Gas Equation (Simplified) | $\text{PAO}_2 = 150 - 1.2(\text{PaCO}_2)$ |
| A-a gradient | $\text{PAO}_2 \text{ (Calculated)} - \text{PaO}_2 \text{ (Measured)} = \text{A-a gradient}$ |

Figure 2: Alveolar Gas equation useful for clinical purposes if the patient is breathing room air ($\text{FIO}_2 = 0.21$) and resides at or near sea level ($\text{P}_b = 760 \text{ mm Hg}$).

PAO_2 is the alveolar partial pressures of oxygen. This value is calculated from the PaCO_2 measurements.

The PaO_2 , measured from the arterial blood gas sample, is then subtracted from the PAO_2 to obtain the A-a gradient. PaCO_2 represents the arterial partial pressures of carbon dioxide while PaO_2 represents the arterial partial pressures of oxygen.

| | |
|-----------------------|--|
| Alveolar Gas Equation | $P_{A}O_2 = FIO_2 (P_B - 50) - 1.2(PaCO_2)$ |
| A-a gradient | $P_{A}O_2 \text{ (Calculated)} - PaO_2 \text{ (Measured)} = \text{A-a gradient}$ |

Figure 3: **Calculations used to quantify pulmonary gas exchange efficacy in the face of hypoxemia (PaO₂ of < 80 mm Hg) at room air.** *FIO₂ is the fraction of oxygen in inspired air (0.21 at room air), P_B is the atmospheric pressure (760mm Hg at sea level) and 50 represents the water vapor pressure in mm Hg, which is subtracted as only dry alveolar gas pressures are measured. The factor 1.2 represents the respiratory quotient or the ratio of O₂ uptake to CO₂ exhaled. P_AO₂ and PaO₂ are the alveolar and arterial partial pressures of oxygen, respectively, while the PaCO₂ represents the arterial partial pressures of carbon dioxide.*

Clinically, a normal A-a gradient (5-15 mm Hg) excludes pulmonary disease and suggests that the arterial hypoxemia (PaO₂ of < 80 mm Hg) is due to hypoventilation (PaCO₂ > 45 mm Hg), decreased inspired oxygen (FIO₂ < 0.21, possibly with a closed anesthesia pop-off valve) or due to low barometric pressures (as seen with in very high altitudes). Patients with a gradient > 25 mm Hg should be considered to have a degree of V/Q mismatch from pulmonary parenchymal disease, although cardiovascular pathology can also affect this value. Serial A-a determinations can be utilized to assess proper response to oxygen therapy and determine the need for additional diagnostics (e.g. Chest radiographs) and/or additional interventions (e.g. mechanical ventilation).

Summary:

Blood gases help assess three vital physiologic processes in the critically ill veterinary patient: acid-base balance, ventilation and oxygenation. Initial blood gas analysis helps diagnose underlying disease processes as well as guide therapeutic interventions. Serial measurements can be utilized to assess proper response to therapy. Blood gas analysis takes a step-by-step approach and practice. Blood gas data should always be integrated in light of the full clinical and laboratory information.

Addendum:

Please use the reference ranges found on your point of care analyzer or laboratory of use. The reference values given in this document are meant to illustrate concepts more than serve as diagnostic or treatment cut-offs.

Additional Resources:

1. DiBartola, S. P. (2006). Introduction to acid-base disorders. Fluid, electrolyte, and acid-base disorders. S. P. DiBartola. St. Louis (MO) Elsevier: 229–251.
2. Bateman SW. Making sense of blood gas results. *Vet Clin North Am Small Anim Pract* 2008; 38(3):543-557.
3. Davis H. Arterial and Venous Blood Gases. In: Wingfield WE, Raffe MR, eds. *The Veterinary ICU Book*. Jackson, WY: Teton NewMedia; 2002:258-259
4. Camps-Palau MA, Marks SL, Cornick JL. Small animal oxygen therapy. *Compend Educ Pract Vet* 1999; 21(7):1-10.