ABCs OF BLOOD GAS EVALUATION

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Key Points

- Blood gas evaluation should be done in a consistent and systematic fashion every time.
- Metabolic acidosis, metabolic alkalosis, respiratory acidosis, respiratory alkalosis and mixed acid base disorders may be present.
- The PaO_2, 5 x FiO_2, A-a gradient, 120 Rule, and PaO_2: FiO_2 ratio can all be used to assess oxygenation.

Blood gas analysis is used to evaluate acid base, oxygenation, and ventilation. Arterial blood gases are necessary to assess oxygenation parameters and can be used to assess ventilation and acid base status. Venous blood gases are adequate to assess acid base status and are also important in the post arrest patient. Blood gas samples should be collected anaerobically in a heparinized syringe and ideally should be run immediately. Samples capped and stored on ice show little change in oxygen and acid base if run within 4-6 hours. At room temperature, PaO_2 shows significant changes within 12 minutes and acid-base is significantly different after 30 minutes. Delays in measurement can decrease sample oxygen content and increase CO_2 partial pressure due to metabolism by blood cells. Air bubbles mixed with the sample can increase O_2 content and decrease CO_2 content as the gases attempt to equilibrate across the fluid-air interface.

Analytes Measured on the Blood Gas

The ***pH*** gives a measure of alkalinity or acidity. It is determined by the ratio between the HCO_3^- and $PaCO_2$ and is calculated by the equation: $pH = pK + \log [(HCO_3^-)/ 0.03(PaCO_2)]$. Normal arterial pH = 7.35-7.45; pH $\leq$ 7.35 indicates acidemia, pH $\geq$ 7.45 indicates alkalemia.

PaO_2 is the partial pressure of oxygen dissolved in the blood, expressed in mmHg.

$PaCO_2$ is the partial pressure of carbon dioxide dissolved in the blood, expressed in mm Hg. Hyperventilation is defined as a $PaCO_2$ < 35 mmHg (hypocapnia) and hypoventilation is defined as $PaCO_2$ > 45 mmHg (hypercapnia).

HCO_3^- or bicarbonate concentration is one of the parameters used to assess the metabolic component of acid-base balance.

Base excess (BE) is derived from pH, Hb, body temperature, and pCO_2. BE quantifies the metabolic portion and essentially tells us how much acid or base is needed to titrate patient to pH=7.4 if the pCO_2 is held steady at 40 mmHg. Normal BE is -4 to +4 mmol/L. Negative BE (<-4 mmol/L), or base deficit, indicates metabolic acidosis. Positive BE (>4 mmol/L) indicates metabolic alkalosis.

Balance of respiratory and metabolic systems

Acid-base status is dependent upon a balance between the respiratory or metabolic defect, buffering systems, and compensatory mechanisms designed to limit excursions in the pH. Blood proteins and bicarbonate act as the primary buffering systems in the intracellular and extracellular compartments. Compensatory mechanisms are based on the fact that carbon dioxide and bicarbonate concentrations are linked by the equation:

$$\mathbf{CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-}$$

such that a change in CO_2 leads to a change in HCO_3^-, and vice versa. Blood buffering systems (such as phosphates, sulfates) are immediately effective in minimizing minor pH changes. Respiratory compensation occurs over minutes to hours after onset of metabolic derangements. Ventilation is either increased (to decrease CO_2) as compensation for metabolic acidosis or ventilation is decreased (to increase CO_2) as compensation for metabolic alkalosis. Renal compensation, in response to primary respiratory derangements, occurs over several days, as the mechanisms to increase or decrease bicarbonate take longer to upregulate.

Classic Blood Gas Analysis

When analyzing blood gas data, it is best to evaluate the parameters in a set order every time to ensure consistent and complete evaluation.

1) Identify whether the sample is arterial or venous.
2) Is the pH normal, acidemic, or alkalemic? A normal pH does not mean the animal is normal. If the pH is in the normal range, look at which side of normal it is: the acid side (7.35-7.4) or the alkaline side (7.4-7.45)?
3) Evaluate the respiratory component. What is the $PaCO_2$? Is it normal or is the patient hypercapnic or hypocapnic? How does the $PaCO_2$ compare to the pH – are they changed in the same or opposite directions (ie, both acidic, both alkaline)?
4) Evaluate the metabolic component. What is the BE? What is the HCO_3^-? How do they compare to the pH – are they changed in the same or opposite direction?
5) Determine the primary disorder(s). The body never over compensates. The pH and the primary disturbance should both be in the same direction (acidic or alkaline).

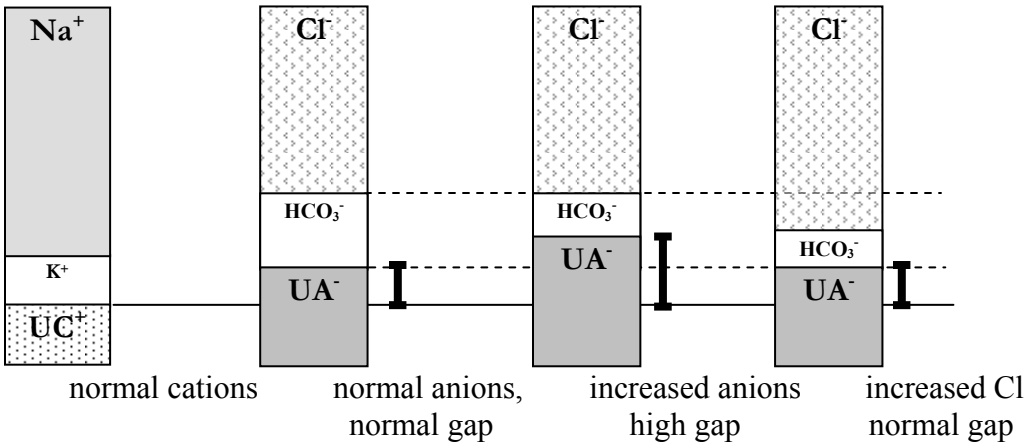
Types of Acid Base Abnormalities

There are four primary acid-base disturbances that occur: metabolic acidosis and alkalosis, and respiratory acidosis and alkalosis.

Metabolic acidosis is the most common acid base abnormality in critically ill small animals. On blood gas, metabolic acidosis is denoted by a low pH, low bicarb, a large negative BE and a compensatory decrease in $PaCO_2$. Anion gap can help differentiate causes of metabolic acidosis. Anion gap is calculated as: $AG = (Na^+ + K^+) – (Cl^- + HCO_3^-)$ or unmeasured anions (UA) minus unmeasured cations (UC).

Increased anion gap, or normochloremic, metabolic acidosis is the most common form of metabolic acidosis. The increase in anion gap is due to presence of additional unmeasured anions. The top five causes of increased gap metabolic acidosis in dogs and cats include: lactic acidosis, ketoacidosis, uremic acids (sulfates, phosphates, etc), ethylene glycol, and salicylate toxicity. Nongap, or hyperchloremic, metabolic acidosis occurs secondary to loss of bicarbonate with excess of chloride. Examples include acute small intestinal diarrhea, renal tubular acidosis, or administration of large quantities of chloride (eg, 0.9% NaCl).

Metabolic alkalosis is seen as an increase in HCO_3^-, large positive BE, and a compensatory increase in $PaCO_2$. The most common causes of metabolic alkalosis are proximal duodenal or pyloric obstruction, severe vomiting, diuretic therapy (furosemide), and administration of bicarbonate solution. It is the hypochloremia, more than loss of H^+, that leads to the metabolic alkalosis. Hypoalbuminemia can also contribute to metabolic alkalosis.



Respiratory acidosis occurs with an increased PaCO2 (hypercapnia) and a compensatory increase in HCO_3^-. Common causes of respiratory acidosis include severe upper airway obstruction (laryngeal paralysis, tracheal collapse, foreign body), pulmonary diseases including asthma or pulmonary fibrosis, anesthetics and analgesics that decrease respiratory drive, neuromuscular diseases such as cervical spinal lesions, diffuse lower motor neuron disease, severe hypokalemia causing muscle weakness, or pleural space or thoracic cage injuries.

Respiratory alkalosis is noted as a decrease in $PaCO_2$ (hypocapnia) and a compensatory decrease in HCO_3^-. The amount of compensation depends on whether the respiratory alkalosis is acute or chronic, since the metabolic compensatory mechanisms take some time to upregulate. Respiratory alkalosis can be seen secondary to peripheral chemoreceptor stimulation from hypoxemia or respiratory disease or direct stimulation of the respiratory center (sepsis, certain drugs, hyperthermia). Pain, anxiety, and excitement can all lead to respiratory alkalosis as many patients, especially dogs, increase respiratory rate which can increase ventilation.

Mixed acid base scenarios, or multiple primary events, commonly occur. Diagnosis can be difficult and it is important to look at all the parameters. If compensation calculations are not compatible with one primary disorder, if the respiratory and metabolic parameters are both changed in the same direction (acidic, alkaline), if the pH is changed in the opposite direction from what is expected for a primary disorder (eg, the lactate is high but the pH is alkaline), or when pCO_2 and HCO_{3-} change in opposite directions from what is predicted, a mixed acid base disorder is likely present.

Evaluating Oxygenation Parameters

Hypoxemia is defined as a low P_aO_2. There are five causes for hypoxemia: 1) low fraction of inspired oxygen, 2) hypoventilation, 3) venous admixture (low V/Q or no V/Q) Venous admixture (V/Q mismatch) occurs when there is disproportional ventilation and perfusion. This is the most common cause of hypoxemia in animals. 4) diffusion impairment – rarely a cause of hypoxemia in veterinary patients and 5) right to left shunt.

The most direct assessment of oxygenation is the PaO_2. However, to get a better idea of lung function, you can use the blood gas information to calculate other indices. These indices include the 5 x FiO_2, A-a gradient, 120 Rule, and PaO_2: FiO_2 ratio.

PaO_2 and 5 x FiO_2: PaO_2 is a measure of dissolved oxygen in the blood. It is obtained by blood gas analysis. In most animals, PaO_2 is usually 90-100 mmHg. A $PaO_2 < 80$ mmHg is considered hypoxemic and $PaO_2 \leq 60$ mmHg is the minimum number to prior to instituting

oxygen therapy if on room air, or mechanical ventilation, if already receiving oxygen supplementation. In a normal patient, the measured PaO_2 should equal 5 x FiO_2. For example, a patient breathing 40% O_2 should have a PaO_2 of 5 x 40 = 200 mmHg. A PaO_2 less than 4 to 5 times the FiO_2 suggests poor lung function or hypoventilation.

Alveolar-arterial equation (A-a gradient): Venous admixture is the most common cause of hypoxemia. The alveolar-arterial equation (is one way to evaluate for presence of significant venous admixture. The complete A-a gradient equation is: $P_AO_2 - P_aO_2 = [FiO_2 (P_B-P_{H20}) - PaCO_2/R] - P_aO_2$, which can be simplified to: A-a = 150 – 1.1($PaCO_2$) – PaO_2

where P_AO_2 = Alveolar partial pressure of oxygen, P_aO_2 = arterial partial pressure of oxygen, as measured on the blood gas, FiO_2 = fraction of inspired oxygen. This equation is best used if on room air, so FiO_2 = 0.21 (21% oxygen), P_B = barometric pressure (760 mmHg at sea level), P_{H20} = water vapor pressure at 37°C (47 mmHg), $PaCO_2$ = arterial partial pressure of CO_2 as measured on blood gas analysis, R = respiratory quotient (accounts for the inverse relationship between level of alveolar ventilation and $PaCO_2$) which is 0.8-0.9. The simplified equation can only be used at sea level when breathing room air. A normal A-a is 5-10 mmHg. An A-a greater than 15 mmHg indicates venous admixture with the higher number suggesting worse admixture.

The "120 rule" is an even more abridged version of the A-a equation. To use this rule, the patient MUST be at sea level, breathing room air (21% O_2). This equation assumes alveolar nitrogen and water vapor pressure do not vary much and oxygen and carbon dioxide are related by the respiratory quotient. In a normal patient, the PaO_2 and $PaCO_2$, when added together, should equal 140 +/- 10. If they do not add to $\geq$ 120, the patient has venous admixture. The lower the number, the worse the admixture.

PaO_2/FiO_2 ratio is another measure of hypoxemia. This allows comparison across time of severity of hypoxemia on patients breathing various oxygen concentrations. To use this ratio, you must have the PaO_2 from the blood gas and the FiO_2 at the time the blood gas was drawn. Normal PaO_2:FiO_2 = 100 mmHg/0.21 ≈ 500. The lower the ratio, the worse the disease process. PaO_2:FiO_2 < 300 is consistent with ALI (acute lung injury). PaO_2:FiO_2 < 200 is consistent with ARDS (acute respiratory distress syndrome).

Case examples will be used to demonstrate acid base and oxygenation assessments.