Arterial and Venous Blood Gases: Indications, Interpretations, and Clinical Applications

Ricardo Irizarry, DVM
Adam Reiss, DVM, DACVECC
Southern Oregon Veterinary Specialty Center
Medford, Oregon

Abstract: *Blood gas analysis is frequently requested as part of the point-of-care testing for emergency or critical care patients presenting with metabolic or respiratory abnormalities. With the advent of portable units, information regarding a patient's acid–base, ventilation, and oxygenation status can be rapidly obtained. This article provides essential information on arterial and venous blood gas analysis with the goal of helping clinicians integrate such data in their case management.*

At a Glance

Metabolic derangements and respiratory distress are common presenting problems in emergency medicine.[1] A focused physical examination and emergency intervention should precede any diagnostic testing if the clinical condition of the patient dictates such urgent care. After the patient is stabilized, a history should be taken and the patient's hydration, ventilation, and oxygenation status assessed. The patient's electrolyte levels and acid–base status (pH) should also be determined.

Indications

Blood gas analysis can help assess underlying disease processes and the severity of illness and can guide emergency interventions (e.g., IV fluid administration, oxygen therapy, electrolyte supplementation, positive-pressure ventilation).[2]

Arterial blood gases primarily provide information regarding oxygenation (i.e., oxygen loading from the lungs into the blood), ventilation (i.e., carbon dioxide (CO_2) off-loading from the blood into the lungs), and acid–base status. Venous blood gases can provide information on acid–base status and ventilation (i.e., venous partial pressure of CO_2 ($PvCO_2$)).[3,4] In adequately perfused patients, the $PvCO_2$ is normally 4 to 6 mm Hg higher than the arterial partial pressure of CO_2 ($PaCO_2$); the difference can be greater in severely hypoperfused patients.[5]

Arterial samples are particularly useful in assessing the patient's oxygenation and ventilation status. For example, the oxygenation status can be evaluated by measuring the arterial partial pressure of oxygen (PaO_2) and using this value in additional calculations, as described in step 5 below.[6,a] Arterial samples are usually collected from the dorsal pedal artery, femoral artery, or, in anesthetized patients, sublingual artery. Step-by-step instructions for sample collection techniques can be found elsewhere.[7,8]

Analytes

Point-of-care blood gas analyzers directly measure the pH, partial pressure of oxygen (PO_2), and partial pressure of CO_2 (PCO_2). These measured values are then used to derive the percentage of hemoglobin saturated with oxygen (SO_2), bicarbonate (HCO_3^-) concentration, total CO_2 (TCO_2) concentration, and base excess of the extracellular fluid (BEecf). The SO_2 is usually determined by the PO_2 from the oxygen dissociation curve. The HCO_3^-

[a]More information about calculating additional oxygenation parameters is available in the companion article, "Beyond Blood Gases: Making Use of Additional Oxygenation Parameters and Plasma Electrolytes in the Emergency Room."

BOX 1

Typical Analytes Provided by Point-of-Care Blood Gas Analyzers

- Venous
  - pH
  - P_{CO_2}
  - TCO_2[a]
  - HCO_3^-[a]
  - BEecf[a]
- Arterial
  - pH
  - P_{CO_2}
  - P_{O_2}
  - TCO_2[a]
  - HCO_3^-[a]
  - BEecf[a]
  - SO_2[a]

[a]Calculated value.

QuickNotes

The most common acid–base disturbance encountered in small animals is metabolic acidosis, which is represented by a lower pH, a negative BEecf or lower HCO_3^- concentration, and a compensatory decrease in the P_{CO_2} in an attempt to blow off excess acid load.

concentration, TCO_2 concentration, and BEecf are also derived from formulas and nomograms. **BOX 1** lists some of the analytes typically reported by point-of-care analyzers.

The BEecf, HCO_3^- concentration, and TCO_2 concentration all serve as measures of the metabolic component of the patient's acid–base status, whereas P_{CO_2} evaluates ventilation and represents the respiratory component of the acid–base status.[9] Oxygenation, as calculated from the Pa_{O_2}, is also part of the respiratory component.

Step-By-Step Approach to Arterial and Venous Blood Gas Analysis

Step 1: pH

The blood pH represents the overall balance of all the acid (acidotic) and base (alkalotic) processes in the body.[7,10] It is determined by the ratio between the metabolic (HCO_3^-) and respiratory (P_{CO_2}) components of the acid–base balance.[11]

In general, *acidemia* is defined as a blood pH below 7.35 and *alkalemia* as a blood pH above 7.45 (7.4 is neutral).[10] Based on the Henderson–Hasselbalch equation, the pH can be defined by the ratio of the HCO_3^- concentration ($[HCO_3^-]$) to the dissolved CO_2 concentration ($[\alpha P_{CO_2}]$) in the extracellular fluid[12]:

$$pH \approx \frac{[HCO_3^-]\ (metabolic)}{[\alpha P_{CO_2}]\ (respiratory)}$$

In this equation, α is the solubility coefficient for CO_2, and it equals 0.03.

A good rule is that pH generally changes in the same direction as the primary disorder.[12]

Step 2: Evaluate the Respiratory Component

P_{CO_2} provides information regarding ventilation, or the respiratory component of acid–base balance.[13] *Alveolar ventilation* is defined as the volume of gas per unit time that reaches the alveoli, where gas exchange with pulmonary blood occurs.[14]

Hypoventilation is characterized by increases in P_{CO_2} (>45 mm Hg) as CO_2 is retained in the blood. CO_2 is a volatile acid, so retention of CO_2 leads to respiratory acidosis.[15] In most instances, respiratory acidosis is caused by some aspect of ventilatory failure, whereby normal amounts of CO_2 produced by tissue metabolism cannot be properly excreted by alveolar minute ventilation.[13] Common causes of hypoventilation include those affecting neurologic control of respiration (e.g., anesthesia, sedation), breathing mechanics (e.g., diaphragmatic hernia, pleural space disease), or proper flow of air through the airways (e.g., upper or lower airway obstruction) or the alveoli.[15]

Hyperventilation is characterized by decreases in P_{CO_2} as the CO_2 is blown off from the alveoli, which leads to respiratory alkalosis (P_{CO_2} <35 mm Hg).[16] Causes of hyperventilation include hypoxemia, pulmonary disease, pain, anxiety, and overzealous manual or mechanical ventilation. Hyperventilation may also develop as a compensation for metabolic acidosis.[16]

Although oxygenation may not directly affect the acid–base balance, it should be assessed in critically ill patients.

Step 3: Evaluate the Metabolic Component

The metabolic contribution to the acid–base balance can be assessed with the HCO_3^- concentration and the BEecf.[12,17] Typical reference ranges for HCO_3^- are 19 to 23 mEq/L in dogs and 17 to 21 mEq/L in cats.[18] Values less than these ranges indicate metabolic acidosis, whereas values greater than the ranges indicate metabolic alkalosis.

As mentioned above, the HCO_3^- concentration is calculated from the pH and P_{CO_2}; thus, it is not independent of respiratory activity.[19] In an attempt to isolate the metabolic component from respiratory influences, the concept

of BEecf was developed.[17] The BEecf takes into account all of the body's buffer systems, including HCO_3^-, 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 mm Hg.[10] By standardizing for the effects of the respiratory component, the BEecf is representative of all the metabolic acid–base disturbances in a patient.[17] Normally, the BEecf is 0 ± 4 mEq/L.[12] Lower values (BEecf <–4) indicate metabolic acidosis, whereas higher values (BEecf >+4) indicate metabolic alkalosis.

Metabolic acidosis can be caused by increases in the generation of hydrogen ions (H^+) from endogenous (e.g., lactate, ketones) or exogenous acids (e.g., ethylene glycol, salicylates) and by the inability of the kidneys to excrete H^+ from dietary protein (renal failure). These increases in H^+ in the body are buffered by decreases in HCO_3^-, producing a lowered HCO_3^-:Pco_2 ratio and, subsequently, a lowered pH. In addition, metabolic acidosis can be caused by a direct loss of bicarbonate (HCO_3^-) through the gastrointestinal tract (diarrhea) or kidneys (renal tubular acidosis) or, less commonly, by the aggressive use of intravenous fluids that contain no bicarbonate or bicarbonate precursors (e.g., saline).[12] Metabolic alkalosis can occur from a loss of H^+ (vomiting of stomach contents) or from a gain of HCO_3^- (e.g., sodium bicarbonate administration, hypochloremic alkalosis caused by the use of loop diuretics).[20]

Step 4: Evaluate the Compensatory Response

Simple acid–base disorders are caused by the four primary acid–base disturbances, metabolic or respiratory in origin, with an anticipated compensatory change[9] (**TABLE 1**). The primary disorder leads to a change in pH, while compensatory changes attempt to normalize the HCO_3^-:Pco_2 ratio and bring the pH back to neutral. Compensatory changes in Pco_2 and HCO_3^- parallel each other, as shown by the direction of the arrows in each row in **TABLE 1**.

Typically, pH changes arising from one component (e.g., metabolic) are opposed by changes in the other component (e.g., respiratory) to maintain the proper ratio of metabolic to respiratory contribution to the overall pH.[10,21] For example, with metabolic acidosis, the HCO_3^- concentration decreases, thereby lowering the HCO_3^-:Pco_2 ratio and resulting in acidemia (pH <7.35).[12] In most

TABLE 1 The Four Primary Acid–Base Disorders and Their Compensatory Changes[a]

Conditions	Primary Disorder	Compensation
↓pH and ↓HCO_3^- (↓BEecf)	Metabolic acidosis	↓Pco_2
↑pH and ↑HCO_3^- (↑BEecf)	Metabolic alkalosis	↑Pco_2
↓pH and ↑Pco_2	Respiratory acidosis	↑HCO_3^- (↑BEecf)
↑pH and ↓Pco_2	Respiratory alkalosis	↓HCO_3^- (↓BEecf)

[a]Rose BD, Post TW. Introduction to simple and mixed acid–base disorders. In: *Clinical Physiology of Acid–Base and Electrolyte Disorders*. 5th ed. New York: McGraw-Hill Book Co; 2001:541.

TABLE 2 Sample Arterial Blood Gas Report From a Patient With Acute Respiratory Failure

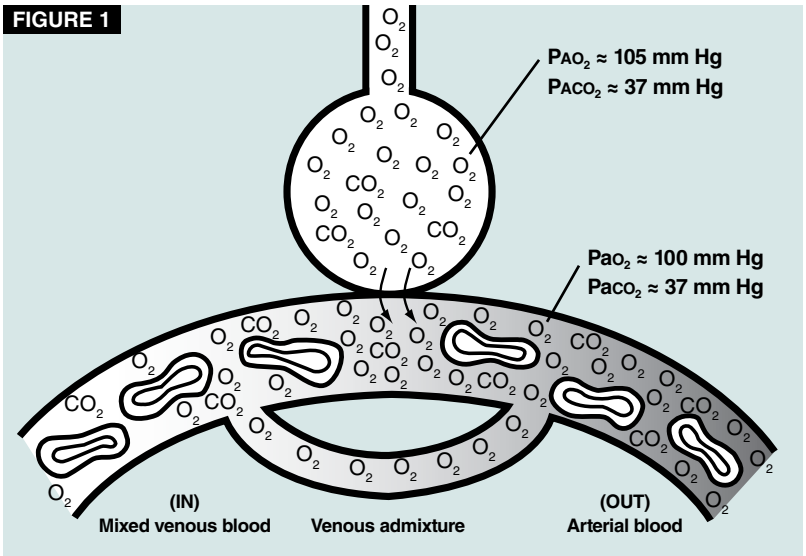
Analyte	Value	Reference Range
pH	7.22	7.35–7.45
$Paco_2$	65 mm Hg	36–40 mm Hg
Pao_2	45 mm Hg	90–100 mm Hg
HCO_3^-	26 mEq/L	20–24 mEq/L
BEecf	+4 mEq/L	–4 to +4 mEq/L

TABLE 3 Summary of Compensatory Responses in Dogs With Metabolic and Respiratory Acid–Base Disorders[21]

Primary Disorder	Expected Compensation
Metabolic acidosis: ↓HCO_3^- (↓BEecf)	↓Pco_2 of 0.7 mm Hg per 1.0 mEq/L decrease in [HCO_3^-] (±3)
Metabolic alkalosis: ↑HCO_3^- (↑BEecf)	↑Pco_2 of 0.7 mm Hg per 1.0 mEq/L increase in [HCO_3^-] (±3)
Acute respiratory acidosis: ↑Pco_2	↑[HCO_3^-] of 0.15 mEq/L per 1.0 mm Hg increase in Pco_2 (±2)
Chronic respiratory acidosis: ↑Pco_2	↑[HCO_3^-] of 0.35 mEq/L per 1.0 mm Hg increase in Pco_2 (±2)
Acute respiratory alkalosis: ↓Pco_2	↓[HCO_3^-] of 0.25 mEq/L per 1.0 mm Hg decrease in Pco_2 (±2)
Chronic respiratory alkalosis: ↓Pco_2	↓[HCO_3^-] of 0.55 mEq/L per 1.0 mm Hg decrease in Pco_2 (±2)

[HCO_3^-] = bicarbonate concentration

FIGURE 1



Cross-section of the alveoli–pulmonary circulation interface showing oxygen (O_2) and carbon dioxide (CO_2) pressures. The depicted P_{AO_2} is obtained from the alveolar gas equation by using normal reference values (e.g., P_{AO_2} = 150 – [1.2 × 38 mm Hg] ≈ 105 mm Hg). The Pa_{O_2} is slightly lower than the P_{AO_2} because of physiologic venous admixture (Box 2). With pulmonary disease, the degree of admixture may increase, thus increasing the oxygen pressure gradient between the alveoli and the systemic arterial circulation (i.e., the alveolar–arterial gradient). (Modified with permission from Martin L, ed. *All You Really Need to Know to Interpret Arterial Blood Gases.* 2nd ed. Baltimore: Lippincott Williams & Wilkins; 1999:70.) P_{ACO_2} = alveolar partial pressures of CO_2; Pa_{CO_2} = arterial partial pressures of CO_2; P_{AO_2} = alveolar partial pressures of oxygen; Pa_{O_2} = arterial partial pressures of oxygen.

QuickNotes

Oxygenation can be assessed by Pa_{O_2} measurements obtained from an arterial sample.

instances, the body compensates by decreasing the P_{CO_2} or hyperventilating in an attempt to maintain the ratio (i.e., $\downarrow HCO_3^-$:$\downarrow P_{CO_2}$). In other words, the respiratory component compensates for the metabolic acidosis in an attempt to raise the pH to neutral. Physiologic compensation rarely completely resolves the primary acid–base abnormality and never leads to overcompensation. Therefore, the pH typically deviates from neutral even after adequate compensation, although it can be within the reference range in patients with mild acid–base disorders.[22]

For example, in **TABLE 2**, the patient is in acute respiratory failure, with primary respiratory acidosis (Pa_{CO_2} = 65 mm Hg), severe hypoxemia (Pa_{O_2} = 45 mm Hg), and HCO_3^- accumulation (HCO_3^- = 26 mEq/L). The pH indicates acidemia, and the Pa_{CO_2} indicates hypercapnia; therefore, the system responsible for the acidemia in this patient is the respiratory system. In this case, hypoventilation (represented by increased Pa_{CO_2}) decreases the HCO_3^-:P_{CO_2} ratio, thus lowering the pH. Given the mild increase in HCO_3^-, there appears to be a mild metabolic compensation, but this increase is more likely attributable to respiratory influences on the HCO_3^- concentration. The BEecf suggests a trend toward metabolic compensation that has not yet been truly achieved. However, if this condition persists for longer than 48 hours, the kidneys will have enough time to retain HCO_3^- in an attempt to compensate for what would then be considered chronic respiratory acidosis, resulting in notable increases in the HCO_3^- and BEecf values (e.g., 38 mEq/L and +10 mEq/L, respectively).[13] Again, note that physiologic compensation for a primary acid–base disturbance is almost never able to return pH to neutral.

Metabolic acidosis is the most common acid–base disturbance in small animals.[1] If metabolic acidosis is the primary disturbance, it will be represented by a lower pH, a negative BEecf or lower HCO_3^- concentration, and a compensatory decrease in the P_{CO_2} in an attempt to blow off excess acid load. The adequacy of the compensatory response can be quantified with the use of formulas that predict the expected response to the primary disturbance (**TABLE 3**).[21] These responses have not been objectively evaluated in cats, but in most cases, cats' responses are assumed to be similar to those of dogs.[17] Acute responses last less than 2 days, whereas chronic responses may take 2 to 5 days to reach maximal effect.

By quantifying the degree of compensatory changes and comparing with the expected (calculated) values, clinicians can assess whether the patient's values are within or outside a defined margin of error. If they are within the margin, the patient has a primary disturbance and is compensating adequately; if they are outside it, the patient likely has multiple primary acid–base disorders (a mixed acid–base disorder).[17] For example, inappropriate respiratory compensation (P_{CO_2}) for metabolic acidosis ($\downarrow HCO_3^-$) is diagnosed by comparing the measured P_{CO_2} with the expected (i.e., calculated) changes in P_{CO_2} predicted for each mEq/L decrease in HCO_3^- (first row in **TABLE 3**). When the measured P_{CO_2} is lower than expected, primary respiratory alkalosis is complicating the metabolic acidosis. An example of a patient with such a mixed disturbance would be a hyperventilating dog with renal failure. Pain, fear, and excitement can cause hyperventilation (decreased P_{CO_2}) in excess of the calculated compensation for metabolic acidosis, which, in this patient, is a result of the uremic acidosis asso-

ciated with renal failure. Conversely, when the measured P_{CO_2} is higher than expected, primary respiratory acidosis is complicating the metabolic acidosis. An example would be a trauma patient (e.g., hit by a car) with lactic acidosis from shock and hypoventilation (increased P_{CO_2}) from pneumothorax preventing proper lung expansion.

In summary, a simple acid–base disturbance should be suspected when the patient meets expected compensation values, and a mixed disturbance should be suspected when compensation does not fall within the expected values.[23] In addition, a mixed disturbance should be suspected when the pH is within the reference range but P_{CO_2} and HCO_3^- values are not or when P_{CO_2} and HCO_3^- concentrations change in opposite, not parallel, directions.[6]

Step 5: Evaluate Oxygenation

Hypoxemia refers to a reduction of oxygen in the arterial blood, indicated by P_{aO_2} values below 80 mm Hg.[6] The presence of hypoxemia can be life-threatening, and a P_{aO_2} value below 60 mm Hg warrants immediate therapeutic intervention.[4] Any time a low P_{aO_2} is obtained from a patient breathing room air, the alveolar gas equation should be used to determine the alveolar–arterial (A–a) oxygen gradient (see below).[6] Normal values for the A–a gradient are 5 to 15 mm Hg. By accounting for the effects of altitude, fraction of inspired oxygen (F_{IO_2}), and ventilation on the patient's oxygenation, the A–a oxygen gradient provides a measure of the adequacy of oxygen transfer across the alveolar membrane into the pulmonary capillaries perfusing the alveoli (i.e., oxygen loading into the blood).[24,25] Serial calculations of the A–a oxygen gradient allow for objective estimates of pulmonary function over time.[24,26]

Most pulmonary diseases alter the ventilation:perfusion ratio (i.e., V/Q mismatch) of individual alveoli, which leads to a reduction in oxygen loading into the blood and a corresponding lower P_{aO_2} (**FIGURE 1** and **BOX 2**).[26] V/Q mismatches lead to an increase in the A–a gradient. The calculations used to quantify pulmonary gas exchange efficacy in the presence of hypoxemia (P_{aO_2} <80 mm Hg) at room air and obtain the A–a gradient are as follows[4,24]:

Alveolar gas equation: Alveolar partial pressure of oxygen (P_{AO_2}) = $[F_{IO_2} \times (P_B - 47)] - (1.2 \times P_{aCO_2})$

A–a Gradient = P_{AO_2} (calculated) – P_{aO_2} (measured)

BOX 2

Ventilation:Perfusion Ratios: Shunts and Venous Admixture

Venous admixture occurs when blood passes through the pulmonary capillaries without being properly oxygenated by the alveoli (**FIGURE 1**). Depending on the severity of the admixture, it can be considered a shunt (i.e., ventilation:perfusion [V/Q] ratio = 0) or cause a decreased V/Q ratio (V/Q ratio <1 but not 0).[a] The ideal V/Q ratio is 1.[26]

A shunt occurs when zero to very minimal ventilation occurs in alveoli that are still adequately perfused by pulmonary capillaries. Shunts can be physiologic or anatomic.[a] In the former, there is a severe physiologic redistribution of pulmonary blood flow away from collapsed or markedly infiltrated alveoli (as seen with lobar atelectasis and with severe pneumonia, pulmonary contusions, or pulmonary edema). In the latter, there is an anatomic diversion of blood away from the pulmonary circulation (as seen with right to left shunting from a ventricular septal defect or a right to left patent ductus arteriosus).[24]

In terms of its effect on oxygenation, a shunt differs from venous admixture that occurs from decreased V/Q ratios by being poorly responsive to oxygen supplementation.[6] Because shunted blood receives no air from the alveoli, increasing the fraction of inspired oxygen (F_{IO_2}) does not typically improve oxygenation. In contrast, oxygen supplementation may improve hypoxemia in patients with venous admixture because the low-V/Q lung areas are still exchanging some alveolar air. Ultimately, patients that do not adequately respond to oxygen supplementation may need mechanical ventilation in an attempt to reopen and convert collapsed alveoli into more functional gas exchange units.[26]

[a]Martin L. P_{aO_2} and the alveolar-arterial P_{O_2} difference. In: Martin L, ed. *All You Really Need to Know to Interpret Arterial Blood Gases.* Baltimore: Lippincott Williams & Wilkins; 1999:55-57.

In these equations, P_B is atmospheric pressure (760 mm Hg at sea level), and 47 is the water vapor pressure in mm Hg (which is subtracted because only dry alveolar gas pressures are measured). The factor 1.2 represents the respiratory quotient, or the ratio of oxygen uptake to CO_2 exhaled. **FIGURE 1** helps illustrate the concept.

The following equation is a simplified version of the alveolar gas equation that can be used for patients breathing room air (F_{IO_2} = 0.21) at sea level (P_B = 760 mm Hg)[24]:

$P_{AO_2} = 150 - (1.2 \times P_{aCO_2})$

Clinically, a normal A–a gradient (5 to 15 mm Hg) excludes pulmonary disease and suggests that arterial hypoxemia (P_{aO_2} <80 mm Hg) is due to hypoventilation (increased P_{aCO_2}) or decreased inspired oxygen.[26] Patients with a gradient above 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.[17,26]

Conclusion

Blood gas analysis helps assess three vital physiologic processes in critically ill veterinary patients: acid–base status, ventilation, and oxygenation. Initial blood gas analysis helps diagnose underlying disease processes and guide therapeutic interventions. Serial measurements can be used to assess proper response to therapy. Blood gas analysis requires a step-by-step approach and practice. Blood gas data should always be interpreted in light of full clinical and laboratory information. C

QuickNotes

Ventilation can be assessed by P_{CO_2} measurements from an arterial sample (P_{aCO_2}) or P_{vCO_2} from a central venous sample in patients with adequate perfusion.

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1. A pH of 7.40 means the
- **a.** HCO_3^- concentration is within normal limits.
- **b.** P_{CO_2} is within normal limits.
- **c.** patient does not have an acid–base disorder.
- **d.** HCO_3^-:P_{CO_2} ratio is normal.

2. A simple metabolic acidosis is characterized by a low pH and a
- **a.** decreased BEecf, HCO_3^- concentration, and P_{CO_2}.
- **b.** normal BEecf and HCO_3^- concentration and increased P_{CO_2}.
- **c.** decreased T_{CO_2}.
- **d.** normal BEecf and HCO_3^- concentration and decreased P_{CO_2}.

3. Hypoventilation ($\uparrow P_{CO_2}$) is potentially dangerous because it
- **a.** correlates with a faster respiratory rate and an increased respiratory effort.
- **b.** causes an increase in the A–a gradient.
- **c.** by itself causes the pH and the P_{aO_2} to decrease.
- **d.** is unresponsive to oxygen supplementation.

4. The BEecf is the most effective parameter to measure the metabolic component of acid–base disorders because
- **a.** it is the only useful parameter when calculating the amount of sodium bicarbonate to administer to severely acidemic patients.
- **b.** it is a direct measurement, not a calculation.
- **c.** it standardizes for the respiratory contribution (P_{CO_2} of 40 mm Hg) to the acid–base balance.
- **d.** a negative value always indicates a metabolic acidosis, not metabolic compensation for a respiratory alkalosis.

5. Simple acid–base disturbances are typically characterized by which of the following?
- **a.** an abnormal pH with P_{CO_2} and HCO_3^- concentration values changing in opposite directions (e.g., $\downarrow HCO_3^-$ and $\uparrow P_{CO_2}$)
- **b.** an acid–base disorder (metabolic or respiratory in origin) and no apparent change in the opposing system
- **c.** a neutral pH (7.4) with abnormal P_{CO_2} or HCO_3^- concentration values
- **d.** an acid–base disorder (metabolic or respiratory in origin) and a quantifiable parallel compensation in the opposing system (e.g., $\downarrow HCO_3^-$ and $\uparrow P_{CO_2}$)

6. Jack, an adult Labrador retriever, presented with a 2-day history of weakness, excessive thirst, and urination. On examination, he was found to be 8% to 10% dehydrated. Please explain Jack's acid–base status from his initial venous blood gas results:

Venous blood gas results:

Results		Interpretation	Reference Value*
pH	7.012	Acidemia	7.4
P_{CO_2} mm Hg	24.2	Respiratory alkalosis	38
HCO_3^- mEq/L	5.4	Metabolic acidosis	22
BEecf mEq/L	-26.2	Metabolic acidosis	0

*midpoint of range

The expected respiratory compensation for this metabolic acidosis (within a margin of variance of ±3 mm Hg) can be calculated as follows:

Expected P_{CO_2} decrease from midpoint reference value = Δ HCO_3^- (decrease in HCO_3^- concentration from a midpoint reference value associated with the metabolic acidosis) × 0.7 (expected mm Hg decrease in P_{CO_2} for each 1.0 mEq/L decrement in HCO_3^-)

In this case: (22 – 5.4) × 0.7
16.6 × 0.7 = **11.6 mm Hg**

Expected P_{CO_2} = P_{CO_2} midpoint reference range – Expected change in P_{CO_2}

In this case:
38 mm Hg – 11.6 = **26.4 mm Hg**

Therefore, Jack's expected P_{CO_2} is 26.4 mm Hg, and his measured P_{CO_2} is 24.2 mm Hg.

After reviewing Jack's measured and expected P_{CO_2} values, we can conclude that Jack's respiratory compensation for his metabolic acidosis is

a. adequate (within the ±3 margin of variance).
b. inadequate (outside the ±3 margin of variance).
c. acute.
d. chronic.

7. Bonbon, a shih tzu puppy, presented with a 2-day history of vomiting and anorexia. On physical examination, a foreign body was palpated in the cranial abdomen. Please explain Bonbon's acid–base status from her initial venous blood gas results:

Venous blood gas results:

Results		Interpretation	Reference Value*
pH	7.5	Alkalemia	7.4
P_{CO_2} mm Hg	52.1	Respiratory alkalosis	38
HCO_3^- mEq/L	37.3	Metabolic alkalosis	22
BEecf mEq/L	+12.0	Metabolic alkalosis	0

*midpoint of range

Bonbon's metabolic alkalosis could be explained by

a. an upper GI obstruction with loss of gastric juices in the vomitus.
b. pain-associated hypoventilation.
c. compensation for the primary respiratory acidosis.
d. lactic acidosis from shock.

8. Please explain the acid–base status of Lucy, a geriatric dog with a 3-day history of progressive increased respiratory effort. On presentation, Lucy's temperature was 103.6°F (39.8°C), her respiratory rate was 42 breaths/min, and her heart rate was 140 bpm. Her physical examination revealed inspiratory stridor on auscultation.

Arterial blood gas results (F_{IO_2} = 21%, at sea level):

Results		Interpretation	Reference Value*
pH	7.2	Acidemia	7.4
P_{CO_2} mm Hg	62.1	Hypoxemia	95
P_{CO_2} mm Hg	67.6	Respiratory acidosis	38
HCO_3^- mEq/L	30.4	Metabolic alkalosis	22
BEecf mEq/L	+12.4	Metabolic alkalosis	0

*midpoint of range

The expected metabolic compensation for this chronic respiratory acidosis (within a margin of variance of ± 2 mEq/L) can be calculated as follows:

Expected HCO_3^- increase from midpoint reference value = Δ P_{CO_2} (increase in P_{CO_2} from midpoint reference range associated with the respiratory acidosis) × 0.35 (expected mEq/L increase in HCO_3^- for each 1.0 mm Hg increment in P_{CO_2}):

In this case: (67.6 – 38) × 0.35
29.6 × 0.35 = **10.4 mEq/L**

Expected HCO_3^- = HCO_3^- midpoint reference range + Expected increase in HCO_3^-

In this case:
22 + 10.4 = **32.4 mEq/L**

Therefore, Lucy's expected HCO_3^- is 32.4 mEq/L, and her measured HCO_3^- is 30.4 mEq/L.

Lucy's acid–base status can be described as

a. a mixed acid–base disorder.
b. respiratory acidosis with adequate metabolic compensation (within the ±2 margin of variance).
c. metabolic alkalosis with respiratory compensation.
d. respiratory acidosis with no compensation (outside the ±2 margin of variance).

9. Please explain Lucy's oxygenation status from her arterial blood gas results. The sample was collected before oxygen supplementation was instituted.

Arterial blood gas results (F_{IO_2} = 21%):

Results		Interpretation	Reference Value*
P_{CO_2} mm Hg	62.1	Hypoxemia	95
P_{CO_2} mm Hg	67.6	Respiratory acidosis	38

*midpoint of range

The A–a gradient calculation is:
$P_{AO_2} = 150 – 1.2 (P_{aCO_2})$
$P_{AO_2} = 150 – 1.2 (67.6)$
$P_{AO_2} = 68.9$

$A–a = P_{AO_2} – P_{aO_2}$
A–a = 68.9 – 62.1 = 6.8

Lucy's respiratory evaluation reveals

a. hypoxemia secondary to ventilation–perfusion abnormalities and hypoventilation.
b. hypoxemia secondary to hypoventilation only.
c. hypoxemia secondary to ventilation–perfusion abnormalities only.
d. hypoxemia secondary to pulmonary disease.

10. Bob is a young adult mixed-breed dog that presented after being hit by a car. Physical examination revealed extensive abrasions on his thorax. His temperature was 102.8°F (39.4°C), his respiratory rate was 62 breaths/min, and his heart rate was 140 bpm. Please explain Bob's oxygenation status from the arterial gas data collected on presentation:

Arterial blood gas results (F_{IO_2} = 21%):

Results		*Interpretation*	Reference Value*
pH	7.48	Alkalemia	7.4
P_{CO_2} mm Hg	63	Hypoxemia	95
P_{CO_2} mm Hg	25.9	Respiratory alkalosis	38
HCO_3^- mEq/L	18.8	Metabolic acidosis	22
BEecf mEq/L	-4.8	Metabolic acidosis	0

*midpoint of range

The A–a gradient is:
$P_{AO_2} = 150 – 1.2 (P_{aCO_2})$
$P_{AO_2} = 150 – 1.2 (25.9)$
$P_{AO_2} = 119$

$A–a = P_{AO_2} – P_{aO_2}$
A–a = 119 – 63 = 56

Bob's respiratory evaluation reveals

a. hypoxemia secondary to ventilation–perfusion abnormalities and hypoventilation.
b. hypoxemia secondary to hypoventilation only.
c. hypoxemia secondary to ventilation–perfusion abnormalities only.
d. hypoxemia secondary to decreased inspired oxygen.