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

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 (CO₂) 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 CO₂ (PvCO₂)).3,4 In adequately perfused patients, the PvCO₂ is normally 4 to 6 mm Hg higher than the arterial partial pressure of CO₂ (PaCO₂); 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 (PaO₂) and using this value in additional calculations, as described in step 5 below.6 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 (PpO₂), and partial pressure of CO₂ (PaCO₂). These measured values are then used to derive the percentage of hemoglobin saturated with oxygen (SO₂), bicarbonate (HCO₃⁻) concentration, total CO₂ (TCO₂) concentration, and base excess of the extracellular fluid (BEecf). The SO₂ is usually determined by the PpO₂ from the oxygen dissociation curve. The HCO₃⁻

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 care management.

At a Glance
- Indications
- Analytes
- Step-By-Step Approach to Arterial and Venous Blood Gas Analysis
- The Four Primary Acid–Base Disorders and Their Compensatory Changes
- Sample Arterial Blood Gas Report From a Patient With Acute Respiratory Failure
- Summary of Compensatory Responses in Dogs With Metabolic and Respiratory Acid–Base Disorders

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“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.”

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concentration, TCO₂ 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, HCO₃⁻ concentration, and TCO₂ concentration all serve as measures of the metabolic component of the patient’s acid–base status, whereas Pco₂ evaluates ventilation and represents the respiratory component of the acid–base status. Oxygenation, as calculated from the PaO₂, 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. It is determined by the ratio between the metabolic (HCO₃⁻) and respiratory (Pco₂) components of the acid–base balance.

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). Based on the Henderson–Hasselbalch equation, the pH can be defined by the ratio of the HCO₃⁻ concentration ([HCO₃⁻]) to the dissolved CO₂ concentration ([αPco₂]) in the extracellular fluid:

$$\text{pH} = \frac{[\text{HCO}_3^-]}{[\alpha\text{Pco}_2]}$$

In this equation, α is the solubility coefficient for CO₂, and it equals 0.03.

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

Step 2: Evaluate the Respiratory Component

Pco₂ provides information regarding ventilation, or the respiratory component of acid–base balance. Alveolar ventilation is defined as the volume of gas per unit time that reaches the alveoli, where gas exchange with pulmonary blood occurs.

Hyperventilation is characterized by decreases in Pco₂ as the CO₂ is blown off from the alveoli, which leads to respiratory alkalosis (Pco₂ < 35 mm Hg). 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.

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Step 3: Evaluate the Metabolic Component

The metabolic contribution to the acid–base balance can be assessed with the HCO₃⁻ concentration and the BEecf. Typical reference ranges for HCO₃⁻ are 19 to 23 mEq/L in dogs and 17 to 21 mEq/L in cats. Values less than these ranges indicate metabolic acidosis, whereas values greater than the ranges indicate metabolic alkalosis.

As mentioned above, the HCO₃⁻ concentration is calculated from the pH and Pco₂; thus, it is not independent of respiratory activity.

In an attempt to isolate the metabolic component from respiratory influences, the concept...
of BEecf was developed. The BEecf takes into account all of the body's buffer systems, including HCO3−, to predict the quantity of acid or alkali required to return the extracellular fluid compartment to neutrality (pH = 7.4) while the Paco2 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 HCO3−, producing a lowered HCO3−:Paco2 ratio and, subsequently, a lowered pH. In addition, metabolic acidosis can be caused by a direct loss of bicarbonate (HCO3−) 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).[13] Metabolic alkalosis can occur from a loss of H+ (vomiting of stomach contents) or from a gain of HCO3− (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 (TABLE 1). The primary disorder leads to a change in pH, while compensatory changes attempt to normalize the HCO3−:Paco2 ratio and bring the pH back to neutral. Compensatory changes in Paco2 and HCO3− 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 HCO3− concentration decreases, thereby lowering the HCO3−:Paco2 ratio and resulting in acidemia (pH < 7.35).[12] In most

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**TABLE 1** The Four Primary Acid–Base Disorders and Their Compensatory Changes

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Primary Disorder</th>
<th>Compensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓pH and ↓HCO3− (↓BEecf)</td>
<td>Metabolic acidosis</td>
<td>↓Paco2</td>
</tr>
<tr>
<td>↑pH and ↑HCO3− (↑BEecf)</td>
<td>Metabolic alkalosis</td>
<td>↑Paco2</td>
</tr>
<tr>
<td>↓pH and ↑Paco2</td>
<td>Respiratory acidosis</td>
<td>↑HCO3− (↑BEecf)</td>
</tr>
<tr>
<td>↑pH and ↓Paco2</td>
<td>Respiratory alkalosis</td>
<td>↓HCO3− (↓BEecf)</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Analyte</th>
<th>Value</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.22</td>
<td>7.35–7.45</td>
</tr>
<tr>
<td>Paco2</td>
<td>65 mm Hg</td>
<td>36–40 mm Hg</td>
</tr>
<tr>
<td>Pao2</td>
<td>45 mm Hg</td>
<td>90–100 mm Hg</td>
</tr>
<tr>
<td>HCO3−</td>
<td>26 mEq/L</td>
<td>20–24 mEq/L</td>
</tr>
<tr>
<td>BEecf</td>
<td>+4 mEq/L</td>
<td>–4 to +4 mEq/L</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Primary Disorder</th>
<th>Expected Compensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis: ↓HCO3− (↓BEecf)</td>
<td>↓Paco2 of 0.7 mm Hg per 1.0 mEq/L decrease in [HCO3−] (±3)</td>
</tr>
<tr>
<td>Metabolic alkalosis: ↑HCO3− (↑BEecf)</td>
<td>↑Paco2 of 0.7 mm Hg per 1.0 mEq/L increase in [HCO3−] (±3)</td>
</tr>
<tr>
<td>Acute respiratory acidosis: ↑Paco2</td>
<td>↑[HCO3−] of 0.15 mEq/L per 1.0 mm Hg increase in Paco2 (±2)</td>
</tr>
<tr>
<td>Chronic respiratory acidosis: ↑Paco2</td>
<td>↑[HCO3−] of 0.35 mEq/L per 1.0 mm Hg increase in Paco2 (±2)</td>
</tr>
<tr>
<td>Acute respiratory alkalosis: ↓Paco2</td>
<td>↓[HCO3−] of 0.25 mEq/L per 1.0 mm Hg decrease in Paco2 (±2)</td>
</tr>
<tr>
<td>Chronic respiratory alkalosis: ↓Paco2</td>
<td>↓[HCO3−] of 0.55 mEq/L per 1.0 mm Hg decrease in Paco2 (±2)</td>
</tr>
</tbody>
</table>

[HCO3−] = bicarbonate concentration
Cross-section of the alveoli–pulmonary circulation interface showing oxygen (O₂) and carbon dioxide (CO₂) pressures. The depicted Pao₂ is obtained from the alveolar gas equation by using normal reference values (e.g., Pao₂ = 150 – [1.2 × 38 mm Hg] = 105 mm Hg). The Pao₂ is slightly lower than the Pao₂ 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.) Paco₂ = alveolar partial pressures of CO₂; Paco₂ = arterial partial pressures of CO₂; Pao₂ = arterial partial pressures of oxygen.

QuickNotes

Oxygenation can be assessed by Pao₂ measurements obtained from an arterial sample.
Associated with renal failure. Conversely, when the measured PCO₂ 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 Pco₂) 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. In addition, a mixed disturbance should be suspected when the pH is within the reference range but PCO₂ and HCO₃⁻ values are not or when PCO₂ and HCO₃⁻ concentrations change in opposite, not parallel, directions.

Step 5: Evaluate Oxygenation

Hypoxemia refers to a reduction in oxygen in the arterial blood, indicated by Pao₂ values below 80 mm Hg. The presence of hypoxemia can be life-threatening, and a Pao₂ value below 60 mm Hg warrants immediate therapeutic intervention. Any time a low Pao₂ 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). Normal values for the A–a gradient are 5 to 15 mm Hg. By accounting for the effects of altitude, fraction of inspired oxygen (Fio₂), 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). Serial calculations of the A–a oxygen gradient allow for objective estimates of pulmonary function over time.

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 Pao₂ (FIGURE 1 and BOX 2). 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 (Pao₂ <80 mm Hg) at room air and obtain the A–a gradient are as follows:

$$\text{A–a Gradient} = \text{Pao}_2 \text{ (calculated)} - \text{Pao}_2 \text{ (measured)}$$

In these equations, Pn 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 CO₂ 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 (Fio₂ = 0.21) at sea level (Pn = 760 mm Hg):

$$\text{Pao}_2 = 150 - (1.2 \times \text{Paco}_2)$$

Clinically, a normal A–a gradient (5 to 15 mm Hg) excludes pulmonary disease and suggests that arterial hypoxemia (Pao₂ <80 mm Hg) is due to hypoventilation (increased Paco₂) or decreased inspired oxygen. 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.

**Conclusion**

**References**


**QuickNotes**

Ventilation can be assessed by PCO₂ measurements from an arterial sample (PaCO₂) or PcO₂ from a central venous sample in patients with adequate perfusion.

**CE TEST**

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1. A pH of 7.40 means the a. HCO₃⁻ concentration is within normal limits.
   b. PCO₂ is within normal limits.
   c. patient does not have an acid–base disorder.
   d. HCO₃⁻:PCO₂ ratio is normal.

2. A simple metabolic acidosis is characterized by a low pH and a a. decreased BE, HCO₃⁻ concentration, and PCO₂.
   b. normal BE and HCO₃⁻ concentration and increased PCO₂.
   c. decreased TCO₂.
   d. normal BE and HCO₃⁻ concentration and decreased PCO₂.

3. Hypoventilation (↑PCO₂) 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 Pao₂ 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 acidic patients.
   b. it is a direct measurement, not a calculation.
   c. it standardizes for the respiratory contribution (PCO₂ 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 PCO₂ and HCO₃⁻ concentration values changing in opposite directions (e.g., ↓HCO₃⁻ and ↑PCO₂)
   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 PCO₂ or HCO₃⁻ concentration values
   d. an acid–base disorder (metabolic or respiratory in origin) and a quantifiable parallel compensation in the opposing system (e.g., ↓HCO₃⁻ and ↑PCO₂)
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:

<table>
<thead>
<tr>
<th>Results</th>
<th>Interpretation</th>
<th>Reference Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.012</td>
<td>Acidemia 7.4</td>
</tr>
<tr>
<td>Pco2 mm Hg</td>
<td>24.2</td>
<td>Respiratory alkalosis 38</td>
</tr>
<tr>
<td>HCO3- mEq/L</td>
<td>5.4</td>
<td>Metabolic acidosis 22</td>
</tr>
<tr>
<td>BEecf mEq/L</td>
<td>-12.6</td>
<td>Metabolic acidosis 0</td>
</tr>
</tbody>
</table>

*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 Pco2** = Pco2 midpoint reference range – Expected change in Pco2

In this case:

38 mm Hg – 11.6 = 26.4 mm Hg

Therefore, Jack’s expected Pco2 is 26.4 mm Hg, and his measured Pco2 is 24.2 mm Hg.

After reviewing Jack’s measured and expected Pco2 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:

<table>
<thead>
<tr>
<th>Results</th>
<th>Interpretation</th>
<th>Reference Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.1</td>
<td>Alkalemia 7.4</td>
</tr>
<tr>
<td>Pco2 mm Hg</td>
<td>52.1</td>
<td>Respiratory alkalosis 38</td>
</tr>
<tr>
<td>HCO3- mEq/L</td>
<td>37.3</td>
<td>Metabolic acidosis 22</td>
</tr>
<tr>
<td>BEecf mEq/L</td>
<td>+12.0</td>
<td>Metabolic acidosis 0</td>
</tr>
</tbody>
</table>

*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 (FiO2 = 21%, at sea level):

<table>
<thead>
<tr>
<th>Results</th>
<th>Interpretation</th>
<th>Reference Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.4</td>
<td>Alkalemia 7.4</td>
</tr>
<tr>
<td>Pco2 mm Hg</td>
<td>62.1</td>
<td>Hypoxemia 95</td>
</tr>
<tr>
<td>Pco2 mm Hg</td>
<td>67.6</td>
<td>Respiratory alkalosis 38</td>
</tr>
<tr>
<td>HCO3- mEq/L</td>
<td>30.4</td>
<td>Metabolic alkalosis 22</td>
</tr>
<tr>
<td>BEecf mEq/L</td>
<td>+21.6</td>
<td>Metabolic acidosis 0</td>
</tr>
</tbody>
</table>

*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 HCO3-** increase from midpoint reference value = ∆ Pco2 (increase in Pco2 from midpoint reference range associated with the respiratory acidosis) × 0.7 (expected mm Hg decrement in HCO3-)

In this case: (67.6 – 38) × 0.7 = 23.9 mm Hg

Therefore, Lucy’s expected HCO3- is 32.4 mEq/L, and her measured HCO3- 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 (FiO2 = 21%):

<table>
<thead>
<tr>
<th>Results</th>
<th>Interpretation</th>
<th>Reference Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pco2 mm Hg</td>
<td>62.1</td>
<td>Hypoxemia 95</td>
</tr>
<tr>
<td>Pco2 mm Hg</td>
<td>67.6</td>
<td>Respiratory alkalosis 38</td>
</tr>
</tbody>
</table>

*midpoint of range

The a–a gradient calculation is:

PaO2 = 150 – 1.2 (Paco2)
PaO2 = 150 – 1.2 (67.6)
PaO2 = 86.9

A–a = PaO2 – Pao2
A–a = 86.9 – 62.1 = 6.8

Lucy’s respiratory evaluation reveals:

- a. hypoxemia secondary to ventilation-perfusion abnormalities and hyperventilation.
- 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 blood gas data collected on presentation:

Arterial blood gas results (FiO2 = 21%):

<table>
<thead>
<tr>
<th>Results</th>
<th>Interpretation</th>
<th>Reference Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.48</td>
<td>Alkalemia 7.4</td>
</tr>
<tr>
<td>Pco2 mm Hg</td>
<td>63</td>
<td>Hypoxemia 95</td>
</tr>
<tr>
<td>Pco2 mm Hg</td>
<td>25.9</td>
<td>Respiratory alkalosis 38</td>
</tr>
<tr>
<td>HCO3- mEq/L</td>
<td>18.8</td>
<td>Metabolic acidosis 22</td>
</tr>
<tr>
<td>BEecf mEq/L</td>
<td>-4.8</td>
<td>Metabolic acidosis 0</td>
</tr>
</tbody>
</table>

*midpoint of range

The a–a gradient is:

PaO2 = 150 – 1.2 (Paco2)
PaO2 = 150 – 1.2 (25.9)
PaO2 = 119

A–a = PaO2 – Pao2
A–a = 119 – 63 = 56

Bob’s respiratory evaluation reveals:

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