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Strategies for interpreting arterial blood gases

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Abstract: This article provides a step-by-step guide for nurses to interpret arterial blood gas (ABG) results, focusing on five key components: SaO_2 , PaO_2 , pH, PaCO_2 , and HCO_3^- . It explains how to assess a patient's oxygenation status, compensation levels, and determine if there is an acid-base disturbance and if that disturbance is respiratory or metabolic.

Keywords: arterial blood gases, ABG, compensation in ABG analysis, pH, acid-base balance, SaO_2 , PaO_2 , PaCO_2 , HCO_3^- ; metabolic acidosis, metabolic alkalosis, respiratory acidosis, respiratory alkalosis

Case scenario: Introduction

At 2322, DP arrived in the ED complaining of shortness of breath. DP, a male, age 67, had a history of chronic obstructive pulmonary disease (COPD). He sat on the edge of his chair, leaning forward, with both hands on his knees.

Physical findings included a barrel-shaped chest, slightly cyanotic nail beds with a slow capillary refill time, and digital clubbing. His breath sounds were distant, with bilateral basilar inspiratory crackles. His vital signs were: a tympanic temperature of 101.8 °F (38.8 °C), heart rate of 94

beats/minute, respiratory rate of 22 breaths per minute, BP of 155/92 mm Hg, an SpO_2 of 87% on room air, and a productive cough with sputum that had changed from the usual clear color to a deep yellow, indicating an exacerbation of his COPD likely due to an infection. This was his second ED visit for a COPD exacerbation in 4 months. Soon after arrival, a blood sample for arterial blood gas (ABG) was drawn and analyzed. The results included these key components: pH 7.33 (normal, 7.35–7.45), PaCO_2 68 mm Hg (normal, 35–45), HCO_3^- 34

mEq/L (normal, 22-26), PaO₂ 55 mm Hg (normal, 80-100), and SaO₂ 84% on room air (normal, 95-100).

Five key components

ABGs have five key components to assess: percentage of hemoglobin saturated with oxygen in arterial blood (SaO₂), partial pressure of oxygen dissolved in arterial blood (PaO₂), arterial blood acidity or alkalinity (pH), partial pressure of carbon dioxide dissolved in arterial blood (PaCO₂), and concentration of bicarbonate ions in arterial blood (HCO₃⁻) (see *Normal ABGs in adults*).

SaO₂ and PaO₂: Oxygenation

Oxygen is transported in the blood as oxyhemoglobin and oxygen molecules. Oxyhemoglobin (oxygen bound to hemoglobin molecules in red blood cells [RBCs]) accounts for about 98% of the oxygen in arterial blood and is measured as SaO₂. A normal oxyhemoglobin saturation should be greater than 95%; if the value drops to below 92% in otherwise healthy patients or below 92% to 88% in patients with COPD, the patient should be assessed and supplemental oxygen administered.

The remaining 2% of oxygen in arterial blood travels as dissolved oxygen molecules.¹ This is measured as PaO₂ and is related to the patient's SaO₂: As oxygen dissolves into the blood, it also combines with hemoglobin in the RBCs. With a higher PaO₂, hemoglobin quickly takes up oxygen molecules until the hemoglobin is saturated. At that point, the SaO₂ is 100%.

More oxygen can still dissolve into the blood. As such, the PaO₂ can climb higher than normal (80-100 mm Hg). For example, in a young, healthy person with no lung disease breathing 100% oxygen for a short period, the PaO₂ could reach about 600 mm Hg.

An S-shaped oxyhemoglobin dissociation curve graphically shows the relationship between PaO₂ and SaO₂ (see *Oxyhemoglobin dissociation curve*). Changes in certain parameters in the body will cause a shift of the S-shaped curve to the left or right. A shift to the left, which indicates hemoglobin's increased affinity for oxygen (inhibiting oxygen release to the cells), can be caused by increased pH, decreased temperature, or decreased PaCO₂. A shift to the right, which indicates hemoglobin's decreased affinity for oxygen and easier movement of oxygen into cells, can be caused by decreased pH, increased temperature, and increased PaCO₂. The SaO₂ is dependent on the PaO₂; oxygen has to first dissolve in the blood before it can bind to hemoglobin.²

If the patient is hypoxemic, the low oxygen content in the blood will be reflected in low PaO₂ and SaO₂ values. Normal oxygen values are defined as a PaO₂ of 80 to 100 mm Hg. Situations that reduce oxygenation include COPD, severe asthma attacks, pulmonary embolism or edema, and severe infections such as sepsis and bilateral pneumonia.³

Mild hypoxemia is defined as a PaO₂ of 60 to 79 mm Hg; moderate hypoxemia, 40 to 59 mm Hg; and severe hypoxemia, less than 40 mm Hg. Prolonged or severe hypoxemia leads to tissue hypoxia and anaerobic metabolism, altering the patient's acid-base status. Administering supplemental oxygen to a patient who is hypoxemic or hypoxic may prevent significant changes in acid-base status.

pH: Acidic or basic?

The acidity or alkalinity of a solution is measured by its pH: the greater the concentration of hydrogen ions, the greater the acidity and the lower the pH; conversely, the lower the concentration of hydrogen ions, the greater the alkalinity and the higher the pH.

The normal range for pH is narrow (7.35 to 7.45); below 6.8 or above 7.8, the body's metabolic processes fail, and the patient dies.

With ABGs, the pH is affected by the PaCO₂ (involving the pulmonary system) and the HCO₃⁻ (involving the renal system). When the pH changes due to an issue with one system, the other usually attempts to correct the pH. In almost all ABGs, the pH is nearly or completely acidic or alkaline, depending on whether it is within normal limits (absolute normal is 7.40). The relationship between pH, PaCO₂, and HCO₃⁻ is the patient's acid-base status.⁴

PaCO₂: The respiratory parameter

PaCO₂ is a measure of the partial pressure that dissolved carbon dioxide exerts in arterial blood and is directly related to the amount of carbon dioxide produced by the cells. The lungs regulate the PaCO₂ and can be used to determine if an acid-base disturbance is respiratory in origin. This value is inversely related to the alveolar ventilation rate. For example, a patient with bradypnea retains carbon dioxide, and a patient with tachypnea exhales more carbon dioxide.

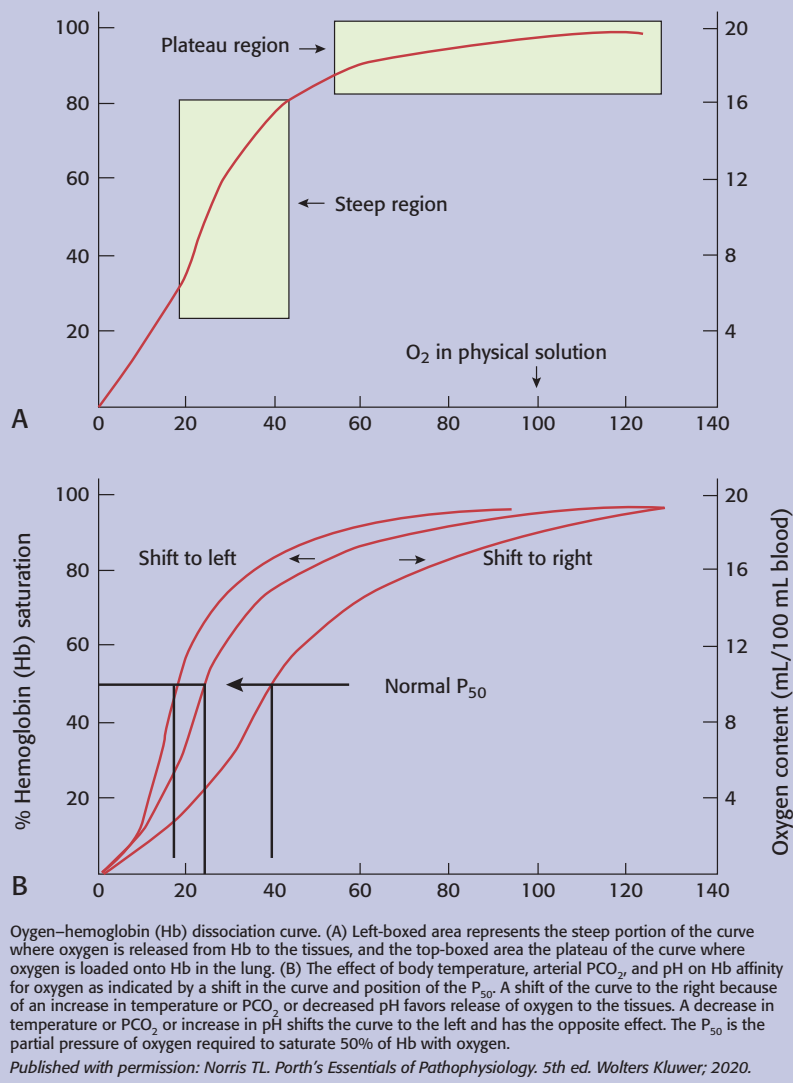
Respiratory disorders that affect ventilation, like COPD, pulmonary fibrosis, or neuromuscular disease, will affect the lungs' ability to remove PaCO₂. The ABG will reflect this by a higher PaCO₂.³

Increased ventilation reduces PaCO₂; decreased ventilation raises PaCO₂. A PaCO₂ level below 35 mm

Normal ABGs in adults

ABG component	Normal range
pH	7.35-7.45
PaO ₂	80-100 mm Hg
PaCO ₂	35-45 mm Hg
HCO ₃ ⁻	22-26 mEq/L
SaO ₂	95%-100%

Oxyhemoglobin dissociation curve



Hg causes respiratory alkalosis; a level above 45 mm Hg causes respiratory acidosis. The body can adjust the level of PaCO₂ within minutes by increasing or decreasing the respiratory rate or the tidal volume (the volume of air inhaled and exhaled in one breath).

PaCO₂ should be considered an acidic parameter because more hydrogen ions are produced as it increases. Conversely, decreases in CO₂ will decrease hydrogen ions. When considered on its own, increases in PaCO₂ lead to an acid state; decreases lead to an alkaline state.

HCO₃⁻: The renal (metabolic) parameter

The bicarbonate ion (HCO₃⁻) is regulated by the kidneys and serves as the metabolic component of the ABG analysis. As part of the body's buffering system, the kidneys retain or excrete the alkalotic bicarbonate ion as needed.

The HCO₃⁻ value can be used to determine if the source of an acid-base disturbance is respiratory or metabolic. An HCO₃⁻ level below 22 mEq/L indicates metabolic acidosis; above 26 mEq/L indicates metabolic alkalosis. Some references use a normal range of

21 to 27 mEq/L.⁵ Unlike the respiratory system, which can quickly adjust PaCO₂ levels, the renal system needs several hours (normal renal function) to days (impaired renal function) to alter HCO₃⁻ levels.

In contrast to PaCO₂, HCO₃⁻ should be considered an alkaline parameter. Increases in bicarbonate reduce hydrogen ions; decreases in bicarbonate increase hydrogen ions. Thus, HCO₃⁻ increases lead to an alkaline state and decreases lead to an acid state.

Acid-base imbalance: Causes

Acute causes of changes in acid-base balance include oversaturation and head trauma (resulting in respiratory acidosis), anxiety and anemia (resulting in respiratory alkalosis), starvation and diabetic ketoacidosis (resulting in metabolic acidosis), and vomiting and prolonged nasogastric tube suctioning (resulting in metabolic alkalosis).

Compensation

Compensation is the body's attempt to maintain a normal pH level. The respiratory system controls the carbon dioxide level, and the renal system controls the bicarbonate level. The body uses these two systems to oppose each other to maintain a normal pH. For example, if one system changes in the acidic direction (lower pH and higher hydrogen ions), the other will compensate in the alkalotic direction to shift the pH higher (and decrease hydrogen ions).

A patient who is rapidly breathing exhales too much carbon dioxide, reducing their PaCO₂ and increasing the pH of arterial blood. The body tries to compensate for this alkalosis by excreting more bicarbonate from the kidneys (getting rid of a base or an alkaline substance), which makes arterial blood more acidic.

There are three levels of compensation. First, an uncompensated status indicates that the respiratory or renal system has not attempted to

compensate for the changing pH. Second, a partially compensated status indicates that the opposing body system is attempting to compensate but has not changed enough to bring the pH back to normal limits. Lastly, a fully compensated status consists of pH within normal limits and values for the respiratory and metabolic components outside their normal ranges but in opposite directions.⁶

In an acute respiratory acidosis, for every 10 mm Hg increase in PaCO₂, the HCO₃⁻ will increase 1 mEq/L as it begins to compensate (generally still in an uncompensated state). In a chronic respiratory acidosis, for every 10 mm Hg increase in PaCO₂, the HCO₃⁻ will increase 5 mEq/L to reach full compensation. The kidneys will rarely (if ever) fully compensate for respiratory acidosis (pH will stay a little acidic and not be 7.40).^{6,7}

A systematic approach

Suppose a patient's ABG results are as follows: pH 7.52; PaCO₂ 30 mm Hg; HCO₃⁻ 24 mEq/L; PaO₂ 89 mm Hg; and SaO₂ 96%. The pH is elevated, the PaCO₂ is low, and the remaining values are within normal limits. These values should be assessed in the following steps:

Step 1: Examine the PaO₂ and the SaO₂ levels to determine if hypoxemia exists and intervene if necessary. Both values are within normal limits in the example, so the patient is not hypoxemic. Continue to monitor the patient's oxygenation status.

Step 2: Examine the pH and determine if it is nearing or is acidic or alkaline and make a note using the correct label. A pH between 7.35 and 7.39 is considered normal and partially acidic; a pH between 7.41 and 7.45 is considered normal but partially alkalotic. In the example, the pH of 7.52 indicates a clear alkalosis.

Step 3: Examine the PaCO₂ and determine if it indicates acidosis or

alkalosis. In this example, the PaCO₂ is low and outside normal limits, so the respiratory component indicates alkalosis.

Step 4: Examine the HCO₃⁻ and determine if it indicates acidosis or alkalosis. In the example, this metabolic component is within normal limits.

Step 5: Identify the origin of the acid-base disturbance as respiratory or metabolic. In this example, the low PaCO₂ matches the high pH, indicating respiratory alkalosis.

Step 6: Determine whether the patient is in compensation. Is the pH within normal limits (but nearly acid or alkaline) and both parameters are outside normal limits but in opposite directions (one clearly acidic, the other clearly alkaline)? If so, the patient is fully compensated. If not, reassess the value that didn't match the pH, (the HCO₃⁻ in the example). If it is within normal limits, the ABG is uncompensated. In the example, the patient would be partially compensated if this value had been outside the normal limits on the acidic side and the pH was still outside normal limits.

Step 7: Combine the analysis from the steps above. Regarding the example at the beginning of this section, the patient has an

uncompensated respiratory alkalosis with normal oxygenation.

Case scenario: ABG application

DP's ABGs were: pH 7.33, PaCO₂ 68 mm Hg, HCO₃⁻ 34 mEq/L, PaO₂ 55 mm Hg, and SaO₂ 84%. This indicates partially compensated respiratory acidosis with moderate hypoxemia or "acute-on-chronic respiratory acidosis."³ With his history of COPD and a PaCO₂ of 68 mm Hg his expected HCO₃⁻ in a stable, fully compensated state would be around 39 mEq/L.

As previously mentioned, in chronic respiratory acidosis, for every 10 mm Hg increase in PaCO₂, the HCO₃⁻ will increase 5 mEq/L to reach full compensation. His HCO₃⁻ is 34 mEq/L, corresponding to a predicted PaCO₂ of about 60 mm Hg if he was at a usual baseline in his pulmonary condition. With this exacerbation, he appears to have pneumonia, which will interfere with effective alveolar ventilation due to inflammation, increased sputum, and bronchospasm. In addition, his metabolic rate will be higher. Decreased alveolar ventilation plus a higher metabolic rate will contribute to the retention of more CO₂ in the blood and a

Suggested reading/links

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Additional practice cases

Case 1: The patient's ABG values are pH 7.32, PaCO₂ 31 mm Hg, HCO₃⁻ 19 mEq/L, PaO₂ 78 mm Hg, and SaO₂ 89%.

Step 1: The PaO₂ and SaO₂ indicate mild hypoxemia. Administer supplemental oxygen and continue to monitor the patient's oxygenation status.

Step 2: The pH indicates acidosis and is outside the normal range.

Step 3: The PaCO₂ indicates alkalosis in the respiratory component of the ABG.

Step 4: The HCO₃⁻ indicates acidosis in the metabolic component of the ABG.

Step 5: This patient is in acidosis because the pH is below normal. The origin of the acidosis is metabolic because the HCO₃⁻ value matches the acid-base status of the pH.

Step 6: The PaCO₂ is not within normal limits, and neither is the pH, so the patient is partially compensated. Note that the pH is outside normal range and the PaCO₂ and HCO₃⁻ are also outside their normal ranges (respectively) and are moving in opposite directions. This is the hallmark of a partially compensated ABG.

Step 7: The patient has a partially compensated metabolic acidosis with mild hypoxemia.

Case 2: The patient's ABG values are pH 7.36, PaCO₂ 29 mm Hg, HCO₃⁻ 20 mEq/L, PaO₂ 108 mm Hg, and SaO₂ 99%.

Step 1: The PaO₂ and SaO₂ indicate no hypoxemia.

Step 2: The pH is leaning toward an acidosis but is within the normal range.

Step 3: The PaCO₂ indicates alkalosis in the respiratory component of the ABG.

Step 4: The HCO₃⁻ indicates acidosis in the metabolic component of the ABG.

Step 5: The patient is in acidosis because the pH is on the low side of the normal range. The origin of the acidosis is metabolic because the HCO₃⁻ matches the acid-base status of the pH.

Step 6: The PaCO₂ is not within normal limits, and the pH is, so the patient is fully compensated. Notice both the PaCO₂ and the HCO₃⁻ are outside their normal limits and in opposite directions.

Step 7: The patient is in a fully compensated metabolic acidosis with normal oxygenation. Notice that the PaCO₂ is low. This is due to hyperventilation.

Case 3: The patient's ABG values are pH 7.37, PaCO₂ 58 mm Hg, HCO₃⁻ 33 mEq/L, PaO₂ 65 mm Hg, and SaO₂ 87%.

Step 1: The PaO₂ and SaO₂ indicate mild hypoxemia. Administer oxygen and continue to monitor the patient's oxygenation status.

Step 2: The pH is leaning toward an acidosis but is within the normal range.

Step 3: The PaCO₂ indicates acidosis in the respiratory component of the ABG.

Step 4: The HCO₃⁻ indicates alkalosis in the metabolic component of the ABG.

Step 5: The patient is in acidosis because the pH is on the low side of the normal range.

The origin of the acidosis is respiratory because the PaCO₂ matches the acid-base status of the pH.

Step 6: The HCO₃⁻ is not within normal limits, but the pH is (and both the PaCO₂ and the HCO₃⁻ are out of their normal limits in opposite directions), so the patient is fully compensated.

Step 7: The patient is in fully compensated respiratory acidosis with mild hypoxemia. This is a typical ABG for a stable patient with COPD and is a commonly seen acid-base disturbance.

Case 4: The patient's ABG values are pH 7.37, PaCO₂ 44 mm Hg, HCO₃⁻ 23 mEq/L, PaO₂ 81 mm Hg, and SaO₂ 92%.

Step 1 The PaO₂ and SaO₂ are a bit low but are acceptable. No hypoxemia is present.

Step 2 The pH is leaning toward an acidosis but is within normal range.

Step 3 The PaCO₂ is within normal range.

Step 4 The HCO₃⁻ is within normal range.

End analysis. This is a normal ABG. No compensation needed.

Case 5: The patient's ABG values are pH 7.20, PaCO₂ 61 mm Hg, HCO₃⁻ 19 mEq/L, PaO₂ 58 mm Hg, and SaO₂ 84%.

Step 1: The PaO₂ and SaO₂ indicate moderate hypoxemia. Administer oxygen and continue to monitor the patient's oxygenation status.

Step 2: The pH indicates acidosis.

Step 3: The PaCO₂ indicates acidosis in the respiratory component of the ABG.

Step 4: The HCO₃⁻ indicates acidosis in the metabolic component of the ABG.

Step 5: The patient is in acidosis because the pH is on the low side of the normal range. The origin of the acidosis is both respiratory and metabolic because the PaCO₂ and the HCO₃⁻ match the acid-base status of the pH.

Step 6: The pH, PaCO₂, and the HCO₃⁻ are not within normal limits. All three are acidic, so no compensation is occurring.

Step 7: The patient is in a combined metabolic and respiratory acidosis with moderate hypoxemia. Consider the severity of the acid-base situation along with the hypoxemia. Can this patient continue in this situation, or could this be impending respiratory failure? If so, the patient may need more support than supplemental oxygen, such as continuous positive airway pressure therapy, bilevel positive airway pressure therapy, or invasive mechanical ventilation.

decrease in oxygen. So, this ABG makes sense.

After a 4-day admission and treatment of his pneumonia, his final ABG before discharge was: pH 7.37, PaCO₂ 59 mm Hg, HCO₃⁻ 34 mEq/L, PaO₂ 66 mm Hg, and SaO₂ 88% on room air. This indicates a fully compensated respiratory acidosis with mild hypoxemia and is an acceptable ABG for him.

Conclusion

ABGs provide valuable insights into the patient's condition and the effects of the healthcare team's efforts to affect the patient's cardiopulmonary health at the time of the sample. However, interpretation of an ABG is a fairly complicated process that can be challenging to understand. This

step-by-step approach provides a practical, understandable way to address ABG interpretation. Finally, the best way to gain skill and accuracy in interpreting ABGs is to practice working through many examples, looking at all the components. ■

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