Microcourse: Blood Gas Testing and Interpretation for Medical-Surgical Nurses

Introduction

Arterial blood gas (ABG) measurements are used to evaluate oxygenation, ventilation, and acid-base balance in critically and chronically ill patients. In this course, learners will review the basics of acid-base balance; risk and error prevention during the testing procedure; and methods for quick interpretation of ABGs.

Rationale for Testing

The American Association for Respiratory Care (AARC) published clinical practice guidelines recommending arterial blood gas analysis, for the evaluation of oxygenation, ventilation, and acid-base status, to assess patient response to therapeutic interventions, and to monitor progression and severity of cardiopulmonary disease processes (Davis et al., 2013).

A physician may order an ABG test when a patient has symptoms that portend imminent physiological decline, such as oxygen desaturation unresponsive to supplemental oxygen, deteriorating vital signs, increased effort of breathing, or altered mental status. In addition, ABGs may be ordered as a series, to guide treatment decisions for patients on ventilators or supplemental oxygen.

Acid-Base Balance and the Bicarbonate Buffer

Acidity in a solution increases when hydrogen ions (H^+) are added to the solution, and is expressed as pH (potential of hydrogen) on a 14-point scale, with 7 being neutral, values under 7 being more acidic, and values over 7 being more basic (i.e., alkaline).

A strong acid is a compound that completely dissociates in an aqueous solution, releasing hydrogen ions. A strong base is a compound that completely dissociates in solution, releasing hydroxide ions (OH^-). A chemical buffer is a weak acid or a weak base that can donate or absorb a hydrogen ion (also called a proton) when a stronger acid or base is added to solution. The presence of a buffer allows a solution to maintain a relatively constant pH when a strong acid or strong base is added.

When a strong base is added to a buffer solution, the weak acid buffer will donate H^+ ions to the strong base, which results in a greater concentration of weak base in the solution. On the other hand, when a strong acid is added to a weak base buffer in solution, the buffer will absorb the excess hydrogen ions, producing a higher concentration of weak acid. The net pH will remain relatively unchanged (Betts, 2013).

Cell metabolism constantly produces acid, yet, to survive, the body must maintain a slightly basic blood pH, between 7.35 to 7.45. This is made possible by the presence of several buffer systems in the body. Inside the cells, phosphates and proteins buffer some acid, while, outside of the cells, ammonia and bicarbonate do the same. Ammonia is a major buffer in the kidneys, while bicarbonate is the primary buffer in the blood (Betts, 2013).

Bicarbonate is a weak base depicted as the bicarbonate ion, $HCO3^-$. It is able to accept a hydrogen ion when acid is added to the blood, to form the weak carbonic acid, or H2CO3. When blood pH is at the middle of its normal range, at 7.4, the bicarbonate to carbonic acid ratio is about 20:1, which allows the buffer to neutralize the relatively larger proportion of acid waste produced by metabolism. (Betts, 2013)

Regulation of Acid and Base by the Lungs and Kidneys

Below is the balanced chemical equation that represents the bicarbonate-carbonic acid buffering system at equilibrium. This system allows blood pH to remain within the narrow range of 7.35 to 7.45.

$$\mathbf{CO2 + H2O <-> H2CO3 <-> HCO3^{--} + H^+}$$

When acid (H^+) is added to the system, as shown on the right side of the equation, the bicarbonate will accept the hydrogen ion, producing the weak carbonic acid (H2C03) (Hopkins, 2022).

Metabolism produces more acid than base, so if this were a closed system, the bicarbonate would eventually be exhausted, and the blood would become too acidic to sustain life. However, carbonic acid quickly breaks down into carbon dioxide (CO2) and water, as shown on the left side of the equation. CO2 easily diffuses out of the blood and into the lungs, where it can be exhaled. This reduces the net amount of acid in the blood. (Hopkins, 2022).

The lungs, by controlling the rate of ventilation, can “hold onto” (hypoventilate) or “blow off” (hyperventilate) carbon dioxide (CO2), thus increasing or decreasing the acidity of the blood, compensating for changes in blood pH caused by metabolism. The lungs exert their effects quickly, within seconds to minutes (Hopkins, 2022).

The kidneys regulate acid primarily by reabsorbing bicarbonate and secreting hydrogen ions. If there is a problem with ventilation, such as hypoventilation caused by sedation, CO2 builds up in the blood, increasing the acidity of the blood. This occurs when the CO2 combines with water, producing carbonic acid, which then quickly dissociates into bicarbonate and hydrogen ions. The excess hydrogen ions increase acidity. In the kidneys, the hydrogen ions combine with phosphates and ammonia, and are excreted, while the bicarbonate is reabsorbed, restoring the bicarbonate to carbonic acid ratio to 20:1. The kidneys can also do the opposite---retain hydrogen ions and excrete bicarbonate---if the blood becomes too alkaline (as can occur with some drugs, for example). Unlike with the lungs, regulation of acid-base by the kidneys generally occurs over hours to days, rather than within minutes (Hopkins, 2022).

Measuring ABGs

When the lungs and/or kidneys are unable to compensate for changes in blood pH, an acid-base disorder develops. When an acid-base disorder is suspected, the physician may order an arterial blood gas test, which reports pH, PaO2 (partial pressure of oxygen), PaCO2 (partial pressure of carbon dioxide), and bicarbonate. Saturation of oxygen (SaO2), either measured or

calculated, is also reported. Measuring arterial blood gases can help the physician diagnose acid-base imbalance, as well as problems with ventilation and oxygenation.

Minimizing Risk During the Testing Procedure

Though rare, ABG testing carries risks, such as bleeding, infection, and nerve damage. Practitioners drawing ABGs should be trained in the procedure, and well-versed in evidence-based guidelines, their facility policies, and their state-granted scopes of practice. The RN may or may not be responsible for the blood draw, but it is helpful to understand the procedure in case assistance is needed (World Health Organization, 2010).

ABGs can be drawn from an arterial catheter or by puncture using a needle attached to a syringe. The World Health Organization (WHO) has published a guideline describing the appropriate arterial puncture process in step-by-step detail (World Health Organization, 2010).

When drawing blood via arterial puncture, a specialized syringe should be used, rather than a vacutainer. ABG syringes are pre-heparinized and designed to minimize air exposure which can alter blood gas values (World Health Organization, 2010).

WHO recommends the radial artery for puncture, although the femoral, ulnar, or brachial arteries are sometimes used. The radial artery is the best choice as it is easily palpated and accessed, with less risk of damaging nerve structure. It is also easy to assess the radial artery for adequate collateral circulation, by performing the Allen test. The Allen test can determine whether the ulnar artery can adequately supply blood to the hand in case of injury to the radial artery. A step-by-step description of the Allen test can be found in the WHO guidelines, in Annex I (World Health Organization, 2010).

To minimize risk and improve the patient experience, the guidelines stress the importance of explaining the procedure and obtaining informed consent. Lidocaine has been found to reduce pain at the site, and may be used first, if appropriate and available. Explaining the procedure and reducing pain can help reduce anxiety in the patient. Anxiety can lead to arteriospasm, making it difficult to get blood (World Health Organization, 2010).

A full history should be obtained prior to the test, with emphasis on circulatory problems, bleeding or anticoagulant therapy, and history of fainting. The patient should be placed supine for the test, in case of fainting (World Health Organization, 2010).

Supplies should be at the bedside in advance, to minimize interruptions and stress to the patient. Supplies should include:

- gloves
- alcohol wipes to clean the site prior to puncture
- pre-heparinized syringe
- needles of the appropriate size and length (20, 23, and 25 gauge, of different lengths, depending on the site) ---use of a larger gauge is less likely to cause lysis of the

specimen. Needles should be equipped with safety covers so that they do not need to be manually recapped.
- a bandage for covering the site, with gauze to allow for applying pressure to site after puncture
- a bag or container of crushed ice for transport, unless testing is done at point of care

The radial artery should be located using the Allen test. Once the artery is located, anatomic landmarks should be noted, so that the site can be found again without palpating. If it is necessary to palpate the site after cleaning, sterile gloves should be worn. PPE should be worn (gown and face shield) if exposure to blood is a possibility (World Health Organization, 2010).

The site should be disinfected with 70% alcohol for 30 seconds, then let dry. Once cleaned, the puncture site should not be palpated, unless sterile gloves are worn (World Health Organization, 2010).

Pre-assembled syringe and needle kits may be used, or, if unavailable, the syringe and needle may be assembled at the bedside. The syringe plunger should be pulled to the fill level required by the laboratory (World Health Organization, 2010).

The syringe is held like a dart at a 45-degree angle. The pulse is located again with the index finger, and to avoid contamination, the needle is inserted about 1 cm distal to the index finger palpating the pulse. Inform the patient prior to puncturing the skin (World Health Organization, 2010).

The needle should be advanced until there is blood flash, and the syringe allowed to fill, without pulling on the plunger. When the syringe is filled to the appropriate level, the needle is withdrawn and recapped using the safety-cap mechanism (World Health Organization, 2010).

After the needle is withdrawn, a pressure dressing should be held firmly at the site for 2-3 minutes. If bleeding continues, pressure may need to be applied for 5 minutes or more. Patients, with high blood pressure, bleeding disorders, or on anticoagulants, may experience prolonged bleeding. (World Health Organization, 2010)

The needle with safety cap is removed from the syringe. Air bubbles should be expelled from the syringe and the syringe capped. The syringe should be rolled gently in the hands to mix the sample and heparin. The syringe should be labeled as per facility policy and placed in ice for prompt transport (World Health Organization, 2010).

WHO states that most sampling errors are due to the following: the presence of air in the sample, an improper quantity or mixing of heparin in the syringe after blood draw, and delays in specimen transport to the lab. (World Health Organization, 2010)

Interpreting ABGs

Laboratories vary on the units of measurement and normal ranges used for lab values. Below

are typical normal ranges of ABG parameters:

- **pH** (potential of hydrogen) (7.35-7.45)
- **PaO2** (partial pressure of arterial oxygen) (75-100 mmHg)
- **PaCO2** (partial pressure of arterial carbon dioxide) (35-45 mmHg)
- **$HCO3^-$**(bicarbonate) (22-26 meq/L)
- **SaO2** (saturation of arterial oxygen) (95-100%)

Evaluating Oxygenation and Ventilation
ABG interpretation is the act of evaluating oxygenation, ventilation, and acid-base status, based on the above parameters and their relationships to each other.

Oxygenation is assessed by looking at PaO2 and SaO2. PaO2 is the concentration of oxygen dissolved in the blood, while saturation of oxygen (SaO2) refers to the percentage of hemoglobin (Hb) bound with oxygen. In an ABG report, the SaO2 may be calculated by a blood gas machine algorithm, which compares the fraction of hemoglobin bound to oxygen (O2Hb) to the fraction of deoxygenated hemoglobin (HHb). Modern blood gas analyzers are often equipped with a co-oximeter, which can directly measure oxygen saturation, and can also detect levels of dysfunctional hemoglobin states, such as methemoglobinemia and carbon monoxide poisoning. (To Co-Ox or Not to Co-Ox, n.d.)

Hypoxemia is the state of inadequate oxygen (PaO2) in the blood, while hypoxia is the state of inadequate oxygen exchange from the blood to the tissues. A low PaO2 indicates hypoxemia, but one can have a normal PaO2 and still have hypoxia. If there is poor oxygen saturation, as with dysfunctional hemoglobin states, the patient may be hypoxic without having hypoxemia (To Co-Ox or Not to Co-Ox, n.d.)

Evaluating Acid-Base Balance
Problems with ventilation may be indicated if there is hypoxemia. If there is poor ventilation due to sedation or chest injury, for example, PaO2 may be low. However, problems with ventilation may also be reflected in the PaCO2.

Prolonged problems in ventilation can affect acid-base balance because hypoventilation increases concentration of CO2 and acid in the blood, while hyperventilation decreases CO2 and acid in the blood. Acid-base imbalance caused by a problem with ventilation is called a respiratory acid-base disorder (mediconepal, 2020).

The rate of ventilation and the concentration of blood CO2 can also reflect a metabolic problem. In this case, the ventilation rate changes are not caused by a problem with the lungs or with breathing, but rather, in response to an acid-base imbalance caused by a metabolic acid-base disorder (mediconepal, 2020).

Understanding the relationships between the respiratory and metabolic components of the ABG report can help one pinpoint the cause of the acid-base disorder.

Is the Primary Disorder Respiratory, Metabolic, or Both?

The first step is to identify whether there is acidosis or alkalosis. Remember, normal blood pH is 7.4, and the normal range is 7.35 to 7.45. Acidemia means that blood pH is less than 7.35; alkalemia means blood pH is greater than 7.45. On the other hand, acidosis (or alkalosis) means that there is a process in the body causing a trend toward too much acid or base. One can thus have a normal pH and still have an acidosis or alkalosis. This can happen when there is compensation by the lungs or kidneys. For example, if the lungs are successfully compensating for acidosis caused by a metabolic problem, the pH may be within the normal range; the lungs have been able to "blow off" the excess CO2 and bring the pH closer to normal. However, in this case one will find the pH to be below 7.4, i.e., closer to the acidotic side of the range. Therefore, it is important to note where the pH is in relation to 7.4. (mediconepal, 2020).

After noting the pH, one must determine if there is a primary respiratory or metabolic acid-base disorder. Although this is often complex, a common mnemonic, called "ROME" is often used to do a preliminary analysis.

"ROME" is an acronym formed from the words, "Respiratory, Opposite; Metabolic, Equal". One can use ROME to help one determine if the pH is related to a primary respiratory or metabolic issue (mediconepal, 2020).

Primary Respiratory Disorder

Ventilation problems can cause acid-base derangements, so one should start by looking for a respiratory cause. "Respiratory, Opposite" is a trigger to compare the direction of the pH to that of PaCO2. If there is a respiratory problem, the pH and PaCO2 will go in opposite directions. For example, if there is a pH of 7.30, this is "going downward" toward increasing acidity. Next, looking at the PaCO2, if it is moving in the opposite direction from the pH, then it is "going upward"; that is, blood CO2 is increasing (mediconepal, 2020). Retaining CO2 increases blood acidity (and decreases pH). If the PaCO2 is high and the pH is below 7.4, it is likely that this is a primary respiratory acidosis. This could happen, for example, in an overly sedated patient with slow respirations who is hypoventilating and retaining CO2.

On the other hand, if a patient is hyperventilating due to anxiety, for example, there will be a decrease in CO2 as it is "blown off." If the pH is alkalotic at 7.47, the pH is high, and moving in the direction opposite to the CO2. This suggests that the imbalance is a primary respiratory alkalosis (mediconepal, 2020).

Respiratory, Opposite: pH↓ PaCO2↑--- Respiratory Acidosis
pH↑ PaCO2↓--- Respiratory Alkalosis

Primary Metabolic Disorder

If the pH and PaCO2 are moving in the same direction, then this is not a primary respiratory disorder. At this point, one looks at the bicarbonate ($HCO3^-$). In a metabolic disorder, the $HCO3^-$

and pH will be moving in the same direction, as depicted in the mnemonic, “Metabolic, Equal” (mediconepal, 2020).

To illustrate, a patient with diarrhea will eliminate $HCO3^-$, and if severe, could become acidotic due to the loss of bicarbonate buffer. The patient will have a low pH and a low $HCO3^-$, i.e., both of their values are moving in the same direction, downwards.

On the other hand, for example, a patient may take an excess of antacids containing sodium bicarbonate. The pH then rises, as does the bicarbonate level. Since both values are going in the same direction, a primary metabolic disorder is suspected.

Metabolic, Equal: pH↓ $HCO3^-$↓ --- Metabolic Acidosis
pH↑ $HCO3^-$↑ --- Metabolic Alkalosis

Combined Disorders
In combined disorders there can be both a respiratory disorder and one or more metabolic disorders, at the same time. For example, a patient could have retained CO2 due to hypoventilation caused by CNS depression, with concurrent loss of HCO3-, due to an intestinal fistula, resulting in combined respiratory acidosis and metabolic acidosis. Another example is the patient who is hyperventilating, due to anxiety and pain, causing respiratory alkalosis, but who is also vomiting and losing HCl, causing metabolic alkalosis. In combined primary disorders, one will see the HCO3- and the PaCO2 moving in opposite directions, with the pH moving in the opposite direction to the PaCO2. (Pompey & Abraham-Settles, 2019)

pH↓ PaCO2↑ $HCO3^-$↓ --- Combined Respiratory and Metabolic Acidosis
pH↑ PaCO2↓ $HCO3^-$↑ --- Combined Respiratory and Metabolic Alkalosis

Of course, this mnemonic is a simplification and determining the cause of an acid-base disorder is often more complicated. There can be mixed acidotic and alkalotic processes which are harder to parse out. Obviously, one cannot have a mixed respiratory acidosis and respiratory alkalosis because the lungs regulate pH through the single mechanism of exhaling CO2.

However, one may have a metabolic acidosis and a metabolic alkalosis at the same time, as various metabolic processes or toxins increase acids or bases in the blood. This could happen, for instance, when a patient has metabolic acidosis, secondary to accumulation of lactic acid (which occurs in sepsis), mixed with a metabolic alkalosis, secondary to vomiting and subsequent loss of stomach HCl. The pH will depend on whether the acidotic or the alkalotic process is predominant, and whether there is compensation (Types of Disturbances – EClinpath, n.d.).

Compensated Disorders
A compensated disorder is apparent when the PaCO2 and $HCO3^-$ are abnormal, but the pH is within the normal range of 7.35-7.45. To figure out which system is compensating for which, one starts with the pH, which, in compensated states, is within the normal range of 7.35-7.45.

An important point to remember is that compensation rarely brings pH back to the median of 7.40. (Pompey & Abraham-Settles, 2019) In a compensated disorder, if the normal pH is >7.4, it is a compensated alkalosis, and if it is <7.4 it is a compensated acidosis.

Next, in compensated states, one will see that the PaCO2 and $HCO3^-$ are abnormal and moving in the same direction. So, if one has a pH of 7.36, a PaCO2 of 46, and an $HCO3^-$ of 26, this is a compensated respiratory acidosis. The compensation is being performed by the kidneys, which is reflected by the high bicarbonate.

pH NL (but <7.40) PaCO2↑ HCO3-↑ --- Compensated Respiratory Acidosis

In comparison, using the same abnormal values as in the previous example, with a PaCO2 of 46 and $HCO3^-$ of 26, the diagnosis will be different if the pH is on the alkalotic side of normal (>7.40 and <7.45). In this case, the lungs are compensating for a metabolic alkalosis, as shown by the high level of CO2 with the more alkalotic pH.

pH NL (but >7.40) PaCO2↑ HCO3-↑ --- Compensated Metabolic Alkalosis

Compensated respiratory alkalosis and compensated metabolic acidosis are illustrated below.
pH NL (but >7.40) PaCO2↓ HCO3-↓ --- Compensated Respiratory Alkalosis
pH NL (but <7.40) PaCO2↓ HCO3-↓ --- Compensated Metabolic Acidosis

Partially Compensated Disorders
A partially compensated disorder will have an abnormal pH, and the PaCO2 and $HCO3^-$ will also be abnormal, with both moving in the same direction. The acid-base derangement will fall on the side that reflects the primary disorder. (Pompey & Abraham- Settles, 2019) So, one might have a pH of 7.30, high CO2, and high $HCO3^-$, as follows:

pH↓ PaCO2↑ $HCO3^-$↑ --- Partially Compensated Respiratory Acidosis

If pH and PaCO2 are low, but HCO3- is normal, serial ABGs can show whether compensation is in process. A second ABG might show pH of 7.33 and HCO3- rising, and a third pH may fall into the low normal range, at 7.35. At this point, the respiratory acidosis is compensated.

Uncompensated Disorders
We already know how to interpret an uncompensated primary disorder. An uncompensated disorder will occur when the respiratory or renal system is unable to correct the deficit. For example, respiratory compensation has a limit of approximately 15 mmHg in a healthy adult, and this limit is lower in patients who are older or have comorbidities. Indeed, hyperventilation is limited by respiratory muscle exhaustion, while hypoventilation is limited by the need to maintain a respiratory rate that sustains oxygenation. The kidneys also may be constrained in their ability to compensate for an imbalance. For example, in the case of a respiratory acidosis due to hypoventilation secondary to CNS depression, the kidneys would normally compensate by increasing $HCO3^-$ to buffer the respiratory acidosis. However, if there is a concurrent

disorder, say, lactic acidosis due to sepsis, or ketoacidosis due to uncontrolled diabetes, then the $HCO3^-$ may be overwhelmed because it is already buffering the metabolic acids. $HCO3^-$ may also be insufficient due to concurrent issues, such as GI losses, and kidney disease. In this case, the combined respiratory and metabolic acidosis is not compensated, reflected in a pH below 7.35. (Metabolic Acidosis Treatment & Management, 2021)

Narrowing Ventilation Issues

Remember, ABG interpretation evaluates oxygenation, ventilation, and acid-base disorders. If there is hypoxemia, the cause may be narrowed down by looking for ventilation issues. This is done by calculating the alveolar(A) to arterial(a) oxygen gradient. Oxygen diffuses from an area of greater to lesser concentration, or "down" the pressure gradient. The pressure is normally slightly higher in the alveoli, based on physiologic factors related to age. Thus, the normal alveolar to arterial (A-a) gradient for the patient can be calculated using the age, like this:

A-a gradient = (Age + 10) / 4

One can then compare the expected gradient to the actual gradient calculated from the blood gas results, using the alveolar gas equation, which follows:

PAO2 = (Patm - PH2O) FiO2 - PaCO2/RQ

Note that the PAO2 in the above equation, contains the capital "A," and stands for the partial pressure of oxygen in the alveoli. PAO2 cannot be directly measured, but is calculated, as above. Patm is the barometric pressure at sea level and PH2O is the partial pressure of water in air (about 45mmHg). FiO2 is the fraction of inspired oxygen, which at room air is 21%. FiO2 may differ from 21% if the patient is receiving supplemental oxygen, and this value should be documented at the time of blood draw. RQ is the ratio of CO2 produced to O2 consumed, during basal metabolism. RQ is generally a standard number except in cases of hypoalbuminemia (Hantzidiamantis & Amaro, 2021). Because many of these numbers are constant, the equation can be simplified, as below. The value of PAO2 is thus easy to calculate if the FiO2 and PaCO2 are known (A-a Gradient, n.d.).

PAO2 = (FiO2 x 713) - PaCO2 / 0.8

Narrowing Metabolic Issues

Blood gas analyzers may also measure electrolytes that are used to determine the anion gap (AG). The anion gap is the difference between the number of negative charged anions to positive charged cations in the blood. If there is a metabolic acidosis, the anion gap can be calculated, to help pinpoint the cause, using the values of sodium (Na+), chloride (Cl-) and bicarbonate, as follows:

$AG = Na^+ – (Cl^- + HCO3^-)$

The normal serum anion gap is typically 4 to 12mmol/L, depending on lab standards. The anion gap widens as bicarbonate is used up to correct acidosis. An elevated AG (>30 mmol/L) suggests metabolic acidosis. A low anion gap is rare. A high anion gap acidosis can be caused by organ failure, sepsis, lactic acidosis, diabetic ketoacidosis, and toxic ingestions. Diagnosis of a metabolic acidosis with a high anion gap can be aided by using the mnemonic, GOLDMARK, representing glycols, oxyproline, L-lactate, D-lactate, methanol, aspirin, renal failure, and ketoacidosis. A metabolic acidosis with a normal anion gap is called hyperchloremic acidosis and is caused by the loss of $HCO3^-$ in exchange for chloride, as can happen with diarrhea or renal tubular acidosis (Anion Gap Acidosis: How Do I Calculate It? How Do I Interpret It?, n.d.).

Key Considerations

- ABGs are used to evaluate oxygenation, ventilation, and acid-base imbalance in critically and chronically ill patients.
- Clinicians performing this test should practice in accordance with evidence -based guidelines, with the aim of preventing adverse events and sampling errors.
- Patients should be educated on the procedure and why it is needed and should provide informed consent.
- As it can be challenging to work with multiple variables under stressful conditions, a systematic approach for interpreting ABGs is recommended, and use of mnemonics such as ROME and GOLDMARK may be helpful.

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