CRENSHAW COMMUNITY HOSPITAL

Emergency Department

Interpretation of Arterial Blood Gases

First Things First

Step 1: Properly obtaining ABG sample

- Values measured directly: PaCO2, PaO2, pH
- Values calculated: HCO3-
- PaO2 and PaCO2 must be corrected for temperature variations from the 37 degrees of the analyzer. Increased temperature decreases gas solubility and falsely increases the gas tensions. The opposite is true for lowering temperature.
- Sampling syringes must be specifically for obtaining ABG specimen.
- Samples should be free of air bubbles that allow equilibration of gases between the bubble and the blood sample, thereby lowering the measured gas tensions.
- Room-temperature samples should be analyzed within 15 minutes.
- Iced samples can be analyzed up to an hour after acquisition.

Step 2: Oxygenation and PaO2

- PaO2 is directly measured and can be used to assess oxygen exchange through a few relationships.
    - Normal PaO2 values = 80-100 mmHg
    - Severe Hypoxemia is PaO2 < 50 mmHg
- **PaO2/FiO2**
    - The PaO2 rises with increasing FiO2. Inadequate or decreased oxygen exchange decreases the ratio.
    - Normal PaO2/FiO2 is >400 mmHg
    - Approximate PaO2 by multiplying FiO2 by 5 (eg, FiO2 = 21%, then PaO2 = 100 mmHg)
- **A-a Gradient**
    - PaO2 is dependent on alveolar oxygen (PAO2), which is influenced by the FiO2, barometric pressure (high altitude), PaCO2 increase (respiratory depression), and the gradient between alveolar and arterial oxygen tension, which can be increased by ventilation and perfusion mismatch.
    - A-a = (Pb-PH2O) x FiO2 – (PaCO2/0.8)
    - Normal is < 10 mmHg

Step 3: Ventilation and PCO2

- Normal values for PaCO2 are usually 35-45 mmHg. The PaCO2 is directly measured and is used to estimate CO2 exchange.
- VD/VT = PaCO2 – PECO2/PaCO2:

- Normal values for the dead space to tidal volume ratio are 20-40%. As dead space increases, there is less equilibration between arterial and alveolar CO2 tensions and this ratio increases. However, this information is not readily available without sampling the exhaled volume and measuring CO2.

Step 4: Acid–base disorders

- Respiratory disorders are due to hypercarbia or hypocarbia. A shift in the following equation leads to an increase or decrease in the number of hydrogen ions, changing the pH.
  - H2O + CO2 < -> H+CO+ < -> [HCO3-] + H+
- Metabolic disorders are related to decreased losses, or increased ingestions or production of acids or bases. Metabolic disorders are reflected by a change in [HCO3-]

Step 4A: pH elevated or decreased

- pH is measured directly using an optical absorbance technique.
  - Normal values 7.35-7.45
  - Acidemia: pH < 7.35
  - Acidosis: processes leading to acidemia
  - Alkalemia: pH values >7.45

Step 4B: PaCO2 elevated for respiratory acidosis

- Failure to breathe off CO2 (poor ventilation) leads to acidemia. Compensation for increased CO2 is increased renal reabsorption of bicarbonate.
- pH will decrease 0.08 for every 10 mmHg the PaCO2 increases above 40 mmHg.
- Acute respiratory acidosis: [HCO3-] increases 1 mEq/L for every 10-mmHg rise of PaCO2 above 40 mmHg
- Chronic respiratory acidosis: [HCO3-] increases 4 mEq/L for every 10-mmHg rise of PaCO2 above 40 mmHg

Step 4C: Decreased PaCO2 for respiratory alkalosis

- An increase in ventilation rate or volume decreases CO2 and shifts the above equation to the left, decreasing the concentration of hydrogen ions, and alkalemia. Compensation is achieved by decreased renal bicarbonate absorption.
- pH increases 0.08 for every 10-mmHg decrease in PaCO2 below 40 mmHg.
- Acute respiratory alkalosis: [HCO3-] decreases 2 mEq/l for every 10-mmHg drop in CO2 below 40 mmHg
- Chronic respiratory alkalosis: [HCO3-] decreases 5 mEq/L for every 10-mmHg drop in CO2 below 40 mmHg

Step 4D: Low pH and decreased PaCO2 for metabolic acidosis

- Decreased bicarbonate or excess acid load leads to metabolic acidosis. Compensation for the decrease in pH is achieved by respiratory CO2 elimination, and renal bicarbonate reabsorption. The expected PaCO2 can be derived three ways.
  - PaCO2 = last two digits of the pH
  - PaCO2 = 15 + [HCO3-]
  - PaCO2 = 1.5[HCO3-] + 8 +/- 2
- **Anion Gap**

- The AG can provide information as to whether the acidosis is due to increased acid accumulation or bicarbonate loss. The AG is attributed to unmeasured anions and cations.
    - AG = Na+ - [Cl- + HCO3-]. Normal AG = 3-11 mEq/L
- Plasma proteins provide a major buffering capacity; therefore, hypoalbuminemia can alter the AG, necessitating correction.
- Corrected AG = AG + 2.5(4 - Serum Albumin)

- **High Anion Gap**
    - An increase of hydrogen ions in the ECF from increased production or decreased renal secretion. Common causes on the differential can be remembered by the mnemonic MUDPILES:
        - **M**ethanol, **U**remia, **D**iabetic ketoacidosis (starvation/EtOH ketosis), **P**araldehyde, **I**soniazid**/I**ron, **L**actic acidosis, **E**thylene glycol (antifreeze), **S**alicylates
- **Normal Anion Gap**
    - A normal AG due to acidosis is attributed to bicarbonate loss or the accumulation of hydrogen ions by increased production or ingestion. Bicarbonate can be lost from both renal and extrarenal sources. Chloride is often retained in response to the bicarbonate loss. Normal AG acidosis is also referred to as hyperchloremic metabolic acidosis.
    - Differential: large GI losses, type II RTA, dilutional with large volumes of saline
- **Low Anion Gap**
- Laboratory error is the most common cause, followed by hypoalbuminemia, since albumin constitutes up to 80% of the unmeasured anions. Other causes include halogen (iodine, bromide) intoxication, lithium excess, paraproteinemias, and polymyxin B therapy.

Step 4E: Elevated pH and increased PaCO2 for metabolic alkalosis

- Increases in bicarbonate or hydrogen ion loss define metabolic alkalosis.
- Increased alkali: citrate, acetate, antacids
- Hydrogen ion loss: emesis, aggressive GI suctioning, diuretics, volume depletion
- Compensation occurs with the retention of PaCO2.
- The expected PaCO2 can be derived from:
- PaCO2 = 0.7[HCO3-] + 20 +/- 2

ABG Interpretation

2 Contact Hours

This peer reviewed course is applicable for the following professions:

Advanced Registered Nurse Practitioner (ARNP), Certified Registered Nurse Anesthetist (CRNA), Clinical Nurse Specialist (CNS), Licensed Practical Nurse (LPN), Licensed Vocational Nurses (LVN), Nursing Student, Registered Nurse (RN), Respiratory Therapist (RT)

This course will be updated or discontinued on or before Friday, November 5, 2021

Outcomes

The purpose of this course will be to provide an overview of arterial blood gas interpretation, raise awareness and understanding of the various aspects of arterial blood gases, and to provide a comfort level with the care of the patient by increasing the knowledge base.

Objectives

After completion of this course, the learner will:

- List the steps in obtaining an arterial blood gas sample
- Compare and contrast metabolic acidosis, metabolic alkalosis, respiratory

acidosis and respiratory alkalosis
- Discuss the causes of metabolic acidosis and alkalosis
- Discuss the causes of respiratory acidosis and alkalosis
- Identify the role of the arterial blood gas in a trauma assessment

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Last Updated: 10/29/2019

To earn of certificate of completion you have one of two options:

1. Take test and pass with a score of at least 80%
2. Reflect on practice impact by completing self-reflection, self-assessment and course evaluation.

   (NOTE: Some approval agencies and organizations require you to take a test and self reflection is NOT an option.)

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Introduction

Understanding the significance of the findings for arterial blood gases (ABG) is the first step in the interpretation of them. Without this understanding, the nurse cannot be expected to realize the implication of the results.

Adult students demonstrate various methods of learning to enhance their knowledge base. Finding the best education method for the individual is the first step to success in clinical competence.

A study conducted in Illinois at Freeport Health Network Memorial Hospital and Swedish American Hospital demonstrated that a computer-based module helped nurses to learn ABG interpretation.[1]

Short educational modules for nurses in arterial blood gas analysis can have significant impact on improving the knowledge of the nurse. A recent study showed that 98% of nurses believed that this training was useful.[2]

Whatever the underlying cause for the acid-base disturbance, one must gain knowledge for interpretation of the ABG to establish the best course of treatment. Therefore, the healthcare provider will determine the limitations of therapy based on the results of the ABG.

Another recent analysis showed that teaching a stepwise approach to evaluating arterial blood gases and using case studies, along with tables and figures, could enhance the ability of the nurse to interpret arterial blood gases.[3]

ABG Sampling

Arterial blood gas sampling is a procedure that involves the direct puncture of an artery. It is associated with a low incidence of complication and is used to determine gas exchange levels in the blood and assesses renal, metabolic and respiratory function.

Indications for ABG include:

- Determining the partial pressure of respiratory gases that are involved in ventilation and oxygenation
- Evaluating arterial respiratory gases during diagnostic workups
- Monitoring acid-bases status
- Monitoring for metabolic, respiratory, and mixed acid-base disorders
- Evaluating the effectiveness of mechanical ventilation in a patient with respiratory failure
- Getting a blood sample when venous sampling is not feasible

Absolute contraindications to ABG include:

- Local infection
- Distorted anatomy
- Abnormal Allen test – at the radial site (use a different site)
- Severe peripheral vascular disease in the limb being tested
- Arteriovenous fistulas
- Vascular grafts

Relative contraindications to ABG include:

- Severe coagulopathy
- Currently taking blood thinners (warfarin, heparin, direct thrombin inhibitors, or factor X inhibitors)
- Use of thrombolytic agents

Factors that make ABG difficult:

- Uncooperative patient
- Pulses that cannot be identified easily

- Difficult to position the patient
- Obesity – because subcutaneous fat over access areas obscures landmarks
- Vascular disease leading to rigidity in vessel walls
- Poor distal perfusion – heart failure, hypovolemia, vasopressor therapy

Sources of errors include:

- Air bubbles can increase PaO_2 and lower $PaCO_2$
- Heparin may lower $PaCO_2$
- Gas may diffuse through a plastic syringe
- Acid-base balance may be inaccurate in arterial blood in those with reduced cardiac output (CPR/circulatory failure).

ABG reflect the physiologic state of the patient at the time the test was done. The radial artery is typically the preferred site because it has collateral circulation and is accessible. When the radial artery is not feasible, the femoral or brachial artery can be used. The femoral artery is a deeper artery, and there is greater risk of damage to adjacent structures. It is close to the femoral vein and nerve. When the femoral artery is sampled it requires monitoring and is often only done in an inpatient setting.

The brachial artery is also a deep artery and is more difficult to identify. There are multiple problems with the brachial artery. It is a small caliber vessel. It does not have good collateral circulation, and attaining hemostasis is more difficult.

Repeated punctures increase the risk of artery laceration, inadvertent venous sampling, hematoma, and scarring. When frequent sampling is needed, the use of an indwelling arterial catheter may be beneficial.

When it is difficult to identify sampling sites such as those who have weak pulses, distorted anatomic landmarks or when a deep vascular artery is being accessed, the use of ultrasound-guided ABG sampling may be done. This allows more accurate placement of the

needle and reduced the risk of damage to the surrounding structures.

- If there is a lack of pulsatile flow or the blood is very dark, a vein has likely been punctured
- When no blood is obtained, pulling the needle back may help as the needle may have gone through the artery
- If air bubbles are not removed the partial pressure of oxygen may be increased
- For those with a lot of soft tissue or extra skin over the puncture site the nondominant hand can be used to smooth the skin
- An ultrasound may be used to find the femoral or brachial artery
- If no arterial blood flow occurs when the needle is in the body, the pulse should be found again, and the needle should be repositioned and redirected to the pulse
- When there is poor distal perfusion, the plunger may be pulled back to get a blood sample, but this increases the risk of getting a venous sample. This occurs in those with advanced heart failure, hypovolemia or those on vasopressors
- Do not puncture the femoral or brachial artery if there is poor perfusion distally

Allen Test

Prior to getting a sample of blood from the radial artery, collateral circulation should be assessed. This is commonly done with the Allen or modified Allen test.

The modified Allen test is used to assure ulnar artery collateral circulation and palmar arch patency. It is unknown if it can predict ischemic complications with radial artery occlusion.[4]

In the modified Allen test, the patient holds the hand high and clenches the fist while the clinician compressing the radial and ulnar arteries. The hand is lowered, the fist is opened and pressure is removed off the ulnar artery. Within 5-15 seconds the color should return to

the hand. This suggests that the ulnar artery and the superficial palmar arch are patent. If it takes more than 15 seconds the test is abnormal.

The Allen test (from which the modified Allen test evolved) is performed identically, except the Allen test is done two times. One time the pressure is released from the radial artery and once from the ulnar artery.

The patient should not overextend the hand or spread the fingers wide as it may lead to false-normal results.

Much debate has been held regarding the necessity to obtain an Allen Test prior to obtaining an ABG. Many believe the Allen Test is a standard of care and as such, is written into policy at facilities across the country.

Because the definition of an abnormal Allen Test is difficult to describe, criteria for determining abnormality is challenging. In a study led by Jarvis, the conclusion was that the Allen Test was only accurate about 80% of the time.[5] A more recent study looked at the Allen test and its ability to determine if there is adequate collateral circulation in the palm. The study concluded that the Allen test is not a valid test as a screening tool for collateral circulation of the hand. It also is not able to predict ischemia to the hand after an arterial blood gas measurement. It was concluded that there is not adequate evidence to support its use before arterial puncture.[6] Even though the Allen Test is controversial, hospital policy must be adhered to at all times.

Performing ABGs

Technique

- Determine the site to be sampled
- The site is prepped in a sterile fashion
- Consider local analgesia before to arterial puncture as it reduces pain without

negatively impacting the procedure

- Use an ABG kit
- Palpate the artery with the nondominant hand
- Puncture the artery with the needle at a 45-degree angle relative to the skin
- The syringe should fill on its own – get 2-3 mL of blood
- Hold pressure on the site for 5-10 minutes

Prior to performing ABG, the patient should be educated about the procedure including the risks and benefits. The patient should let the health care provider know if there is new/worsening pain, reduced movement, numbness/tingling in the limb or active bleeding after the procedure is performed.

To get a sample from the radial artery, the patient should lie supine with the forearm supinated on a hard surface. The wrist is extended 20-30 degrees; a small roll may be put under the wrist to make the radial artery more superficial. If a sample is to be taken from the femoral artery the patient is supine with the leg in a neutral position. If blood is taken from the brachial artery, place the arm on a firm surface and the shoulder slightly abducted with the forearm supinated and the elbow extended.

When performing an ABG sampling, the provider should wear gloves and eye protection. The site should be cleaned with an antiseptic solution. The non-dominate hand locates the arterial pulse with the second and third fingers with both fingers proximal to the desired puncture site.

The needle is inserted at a 45-degree angle aiming at the artery with the needle bevel facing upwards. When the needle is angled it reduces vessel trauma and allows the muscle fibers to seal the puncture site after the puncture.

When the blood starts filling the syringe, remove the nondominant hand. After 2-3 ml is obtained the needle is removed and gauze is placed over the site with the nondominant hand to hold pressure for five minutes. For those at risk for bleeding, pressure may need to be held for longer periods of time. Afterward, an adhesive dressing should be placed over the puncture site.

The excess air should be removed from the syringe, the syringe capped and placed in ice while it is awaiting analysis. No air bubbles should be present as this may underestimate the $PaCo_2$ and overestimate the PaO_2.

The nurse must monitor for complications. Active profuse bleeding from the puncture site suggests that there is vessel laceration. Compartment syndrome may result from an expanding hematoma that compromising circulation. Compartment syndrome is suggested by the six P's: pain, pallor, paresthesia, paralysis, poikilothermia, and pulselessness. Ischemia from a thrombus, vasospasm or arterial occlusion presents as pulselessness, color change and distal coldness. A nerve injury may present with paresis and persistent pain. Infection presents with fever and local erythema.

ABG Interpretation

When interpreting the ABG results, one must first know the five major components of the ABG to be addressed: oxygen saturation (SaO_2), partial pressure of oxygen (PaO_2), acidity or alkalinity (pH), partial pressure of carbon dioxide ($PaCO_2$), and bicarbonate ions concentration (HCO_3).

- Acidemia – arterial pH less than 7.35
- Acidosis – lowering of the extracellular fluid pH caused by an elevated PCO_2 or a reduced HCO_3

- Metabolic acidosis – reduction in pH and serum HCO_3
- Respiratory acidosis – reduction in pH with an elevation of the arterial PCO_2
- Alkalemia – arterial pH above 7.45
- Alkalosis – elevation of the extracellular fluid pH caused by an fall in PCO_2 or a rise in HCO_3
- Metabolic alkalosis - elevation in pH and serum HCO_3
- Respiratory alkalosis – elevation of the pH with a reduction in the arterial PCO_2
- Mixed acid-base disorder – more than one acid-base disorder at the same time
- Anion gap = (Na) - (Cl + HCO_3)
    - Normal range is 8-16 mEq/L

The four main acid-base disorders are metabolic alkalosis, metabolic acidosis, respiratory alkalosis and respiratory acidosis. Respiratory alkalosis and acidosis may be classified as acute or chronic as it takes up to five days for the renal system to compensate for respiratory disorders.

The acid-base balance of the blood is maintained by two areas of the body, which are the lungs and the kidneys. The lower pH represents acidosis, and the higher pH represents alkalosis with the normal range of pH from 7.35-7.45.

The PaO_2 evaluates the oxygen in plasma and has a normal range of 80-95 mm Hg. The PaO_2 does not measure the amount of oxygen attached to the hemoglobin. SaO_2 measures the amount of oxygen attached to the hemoglobin. The normal range is 95-99% and generally should be above 90%.

$PaCO_2$ evaluates the ventilation component. The normal range is 35-45 mmHg. However, the value is inversely related to ventilation. For example, decreased ventilation has a higher value, and increased ventilation has a lower value. Therefore, hyperventilation causes alkalosis because the patient is blowing off carbon dioxide, and hypoventilation causes acidosis because the patient is retaining carbon dioxide. The body adjusts for these conditions by changing the respiratory rate.[6]

HCO_3 is regulated by the kidneys and evaluates the metabolic component. The normal range is 22–26 mEq/L. Below 22 mEq/L is considered to be acidosis and above 26 mEq/L is alkalosis. The body can adjust to the abnormalities in the HCO_3 levels but not as quickly as it can to the abnormal $PaCO_2$ levels. Several days could be required to make the necessary adjustments to bring the HCO_3 levels to a normal range.[7]

TEST	NORMAL VALUE
pH	7.35–7.45
HCO_3	22–26 mEq/L
$PaCO_2$	35–45 mm Hg
PaO_2	80–95 mm Hg
SaO_2	95–99%

Four conditions are evaluated based on the ABG: respiratory acidosis, respiratory alkalosis, metabolic acidosis, and metabolic alkalosis. As we explore these conditions, the potential causes, the ABG values, and the compensatory mechanisms, we will gain a better understanding of what is happening within the body.

Respiratory Acidosis

Respiratory acidosis is a condition that happens when the lungs are not able to eliminate enough of the carbon dioxide made by the body. The body excretes the extra hydrogen in the urine and exchanges it for bicarbonate ions. When this happens HCO_3 rises to restore the body to a normal pH. Until the pH returns to normal, the $PaCO_2$ may stay elevated.

Any situation that can cause the patient to develop a depressed respiratory status can cause this medical condition. Examples of these situations could be hypoventilation, asphyxia, central nervous system depression, chronic obstructive pulmonary disease, infection, and drug-induced respiratory depression (Table 9).

The ABG values one would see with respiratory acidosis would be: pH < 7.35; $PaCO_2$ > 45 mmHg; and HCO_3 > 26 mEq/L if compensating.

In acute respiratory acidosis, to compensate the HCO_3 increases approximately 1 mEq/L for each 10 mmHg in $PaCO_2$. In chronic respiratory acidosis (after 3-5 days), the HCO_3 will increase up to 5 mEq/L per 10 mmHg of $PaCO_2$. If there is a mild-to-moderate chronic respiratory acidosis, suggested by a $PaCO_2$ less than 70 mmHg, the pH may be in the low-normal range or slightly reduced. If the pH is significantly acidic in chronic acidosis, there is typically a co-existent metabolic acidosis or an acute respiratory acidosis. If the pH is 7.40 or more than there is likely a co-existent acute respiratory alkalosis or a metabolic alkalosis.

pH	Decreased
$PaCO_2$	Increased
HCO_3	Normal (increased if compensating)

Respiratory Alkalosis

Respiratory alkalosis is a compensatory mechanism of the body aimed to increase the excretion of HCO_3 and retention of the hydrogen ions. Respiratory alkalosis lowers the HCO_3 and restores pH to normal. Conditions that cause the respiratory system to be overstimulated can be extenuating factors in respiratory alkalosis such as hyperventilation (see Table 9). In addition, respiratory alkalosis can be seen in those who are critically ill, such as those who are on ventilators or those with lung or heart disease.[7]

The ABG values one would see with respiratory alkalosis would be: pH > 7.45; $PaCO_2$ < 35 mm Hg; and HCO_3 < 22 mEq/L if compensating.

In acute respiratory alkalosis, the compensation is to lower the serum HCO_3 by 2 mEq/L for every 10 mmHg reduction in $PaCO_2$. In chronic respiratory alkalosis (after 3–5 days) the serum HCO_3 falls about 4–5 mEq/L for every 10 mmHg reduction in $PaCO_2$.[9]

pH	Increased
$PaCO_2$	Decreased
HCO_3	Normal (decreased if compensating)

Metabolic Acidosis

When a patient is demonstrating metabolic acidosis, his or her body is pulling the HCO_3 into the cells as a buffer and therefore depletes the plasma level. The body begins compensating by increasing the ventilation, and thus, renal retention of the HCO_3 takes place.

When patients present with the following conditions, one must consider the patient could be a candidate for metabolic acidosis: HCO_3 loss from diarrhea, shock, renal tubular acidosis, drug intoxication, salicylate poisoning, renal failure, diabetic ketoacidosis, and circulatory failure producing lactic acid.

ABG values one would see with metabolic acidosis would be: pH < 7.35; HCO_3 < 22 mEq/L; and $PaCO_2$ < 35 mm Hg if compensating.

Respiratory compensation for metabolic acidosis causes a reduction in the arterial $PaCO_2$ by about 1.2 mmHg for every 1 mEq/L reduction in the serum HCO_3. If compensation does not occur, there is likely underlying neurologic or respiratory disease.[8]

Calculation of the serum anion gap should be determined in metabolic acidosis. In metabolic acidosis, the anion gap may be high, normal or combined. Determining the anion gap will help determine the cause of the metabolic acidosis.

pH	Decreased
$PaCO_2$	Normal (decreased if compensating)
HCO_3	Decreased

One generally considers the ABG to be a test for respiratory conditions; however, a study of ABGs in Brazil to test patients for metabolic acidosis in relation to sepsis and shock was conducted. The study revealed a group who were not able to clear their inorganic ions had a higher morbidity rate, whereas those who were able to correct their acidosis survived.[9]

The severely septic patient who developed acute renal failure upon arrival to the intensive care unit (ICU) had a battery of tests, including ABG. Results of the ABG revealed: a pH of 7.32, $PaCO_2$ of 45 mmHg, and an HCO_3 of 21mEq/L. Without treatment metabolic acidosis will become progressively worse; steps need to be taken to bring the patient into a compensatory mode to recovery.

Treatment of metabolic acidosis is variable depending on the cause and whether it is acute or chronic. In severe metabolic acidosis, sodium bicarbonate is sometimes used.

Metabolic Alkalosis

With metabolic alkalosis, one will see an increased level of HCO_3. This could be caused by several factors such as too much bicarbonate during a code, excess hydrogen loss during vomiting or suctioning, potassium loss from diuretics or steroids, or excessive alkali ingestion. The kidneys will increase the HCO_3 excretion trying to conserve the hydrogen, and the respiratory system will compensate by decreasing the ventilation and conserving the CO_2 and raising the $PaCO_2$. Patients with normal kidney function are able to excrete excess bicarbonate in the urine so if metabolic alkalosis is maintained there is an inability to excrete bicarbonate in the urine.

ABG values one would see with metabolic alkalosis would be: pH > 7.45; HCO_3 > 26 mEq/L and $PaCO_2$ > 45 Hg if compensating.

Respiratory compensation of metabolic alkalosis typically raises the $PaCO_2$ approximately 0.7 mmHg for every 1 mEq/L increase in HCO_3. The arterial $PaCO_2$ rarely goes above 55 mmHg.[9]

pH	Increased
$PaCO_2$	Normal (increased if compensating)
HCO_3	Increased

Compensation

Acid-base disorders are typically associated with a compensatory response that lessens the change in the HCO_3/ $PaCO_2$ ratio and consequently, in pH. For example, if there is a metabolic acidosis (a reduction in the serum HCO_3) there should be respiratory compensation by moving the $PaCO_2$ in the same direction as the serum HCO_3 (falling). The respiratory compensation lessens the change in the ratio of the serum HCO_3 to $PaCO_2$ and consequently the pH. Respiratory compensation is a rapid adjustment. In metabolic acidosis the respiratory compensation starts within 30 minutes and is done in 12-24 hours.

> If the pH is in the normal range in the face of an abnormal PCO_2 & HCO_3, compensation has taken place.

A respiratory acid-base disorder leads to compensation in two phases: immediate and delayed. The immediate change is a small change in serum HCO_3 in the same direction as the $PaCO_2$. If the respiratory condition persists then the kidneys produce larger changes in the HCO_3. This is meant to stabilize the pH.

In respiratory alkalosis, urinary HCO_3 and hydrogen ion secretion are reduced and in respiratory acidosis, hydrogen ion secretion and HCO_3 are increased to compensate. Renal compensation takes longer than respiratory compensation; it takes three to five days to complete compensation.

A glance at the four disorders demonstrates what happens with the pH, what the initiating event causing the disorder, and the compensatory effect will be shown in the following table (Table 8). It is important to remember that compensating effects are seen in chronic conditions.

Disorder	pH	Initiating Event	Compensating Effect
Respiratory Acidosis	↓	↑ $PaCO_2$	↑ HCO_3
Respiratory Alkalosis	↑	↓ $PaCO_2$	↓ HCO_3
Metabolic Acidosis	↓	↓ HCO_3	↓ $PaCO_2$
Metabolic Alkalosis	↑	↑ HCO_3	↑ $PaCO_2$

Trauma

Studies have shown that along with other indicators such as the Glasgow Coma Scale (GCS), the ABG results can serve as a strong indicator of a patient's mortality during the hospital course. A recent study showed that acid-base disturbances were predictors of death in

major trauma patients.[10]

Addressing the GCS of each trauma patient arriving in the ED is an important step in the assessment process. Using the information included in the following Glasgow Coma Scale, the nurse can assess eye opening, motor response, and verbal response.

Eyes Open	Spontaneous	4
	To verbal command	3
	To pain	2
	No response	1
Best Motor Response to verbal command	Obeys	6
Best Motor Response to painful stimulus	Localizes pain	5
	Flexion-withdrawal	4
	Flexion-abnormal	3
	Extension	2
	No response	1

Best Verbal Response	Oriented and converses	5
	Disoriented and converses	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1

Patients arriving in the Emergency Department (ED) post-trauma receive a head to toe trauma assessment, including the GCS. When there is head trauma, a GCS of < 8 indicates a severe head injury and generally has a poor outcome. The low GCS coupled with a strong ion gap is a strong predictor of hospital mortality.[11] Therefore, vascular injury must be assessed along with other areas of assessment and fluid resuscitation initiated to prevent further decline.

However, a patient can arrive with a GCS of 15, indicating he or she has spontaneous eye opening, obeys verbal commands and he or she is oriented and conversing. This patient can be suffering from respiratory distress caused by a traumatic injury to the lungs; therefore, when used alone the GCS may be a poor indicator of the patient's condition.

Case Study 1

A 50-year-old female arrives in the ED via ambulance. She was the driver of a vehicle that ran head-on into the median underpass on the interstate. She was wearing a seat belt but hit the steering wheel before the airbag deployed. When she arrived in the emergency department she had a GCS of 15 and was anxious. At initial inspection she had bruising to her chest from the seat belt with no visible head injury noted. Her vital signs were within

normal limits. Shortly after arriving in the ED she began complaining of needing to have a bowel movement and difficulty breathing. Her oxygen saturation dropped to 88% on room air. An ABG was obtained with the following results: pH 7.32, $PaCO_2$ 47 mmHg, and HCO_3 28 mEq/L. Vital signs have now made a slight change from being within normal limits to blood pressure 120/70 mm Hg, HR 102, and respiratory rate 30.

Understanding the patient's blood gas reveals a respiratory acidosis; preparations begin to treat the cause when the results of the x-ray reveal a left side pneumothorax.

Causes of Respiratory Acidosis

- COPD
- Severe asthma
- Obstructive sleep apnea
- Chest wall disorders – kyphoscoliosis, flail chest, ankylosing spondylitis
- Obesity
- Sedative/narcotic overdose
- Neuromuscular disease – myasthenia gravis, ALS, Guillain-Barré Syndrome
- CNS depression – encephalitis, trauma

Causes of Respiratory Alkalosis

- Hyperventilation
- Fever
- Anxiety
- Hypoxemia
- Pregnancy

Causes of Metabolic Acidosis

- Increased acid production – generally increased anion gap
  - Ketoacidosis
  - Lactic acidosis
  - Ingestions – aspirin, methanol, ethylene glycol
- Loss of bicarbonate – generally normal anion gap
  - Diarrhea
  - Intestinal tube drainage
  - Carbonic anhydrase inhibitor
  - Renal tubular acidosis Type 2
- Decreased renal acid secretion
  - Chronic kidney disease
  - Renal tubular acidosis Type 1 and 4

Causes of Metabolic Alkalosis

- Renal hydrogen loss
  - Diuretics
  - Primary mineralocorticoids excess
- Gastrointestinal hydrogen loss
  - Chronic diarrhea
  - Vomiting
  - NG suctioning
- Contraction alkalosis
  - Diuresis
  - Sweat loss in cystic fibrosis
  - Vomiting/NG suction in achlorhydria
- Intracellular shift of hydrogen

- Low serum potassium

- Alkali administration

ABG Cheat Sheet[13, 14]

	pH	$PaCO_2$	HCO_3	
Metabolic Acidosis	< 7.35	35–45	< 22	
Metabolic Alkalosis	> 7.45	35–45	>26	
Respiratory Acidosis	< 7.35	> 45	22–26	
Respiratory Alkalosis	> 7.45	< 35	22–26	
Fully Compensated Metabolic Acidosis	7.35–7.45	<35	<22	pH usually < 7.4
Fully Compensated Metabolic Alkalosis	7.35–7.45	>45	>26	pH usually > 7.4
Fully Compensated Respiratory Acidosis	7.35–7.45	>45	>26	pH usually < 7.4
Fully Compensated Respiratory Alkalosis	7.35–7.45	<35	<22	pH usually > 7.4

Case Study 2

A 45-year-old female presents to the emergency department with severe diarrhea for the last two days. She has the following ABG.

- Arterial pH - 7.25
- HCO_3 – 12 mEq/L
- $PaCO_2$ - 26 mmHg

The pH is low therefore, the patient has acidemia. The low HCO_3 suggests metabolic acidosis. The HCO_3 is 12 mEq/L below the normal (which is 24 mEq/L). This should (and did) lead to respiratory compensation with a 14 mmHg fall in $PaCO_2$ (the normal $PaCO_2$ is 40 mmHg). Respiratory compensation for metabolic acidosis is when the arterial $PaCO_2$ falls about 1.2 mmHg per 1 mEq/L reduction in the serum HCO_3 concentration.

This patient has a partially compensated metabolic acidosis (the pH is not in the normal range – so it is only partially compensated). If the $PaCO_2$ was significantly higher (above 26 mmHg) than expected there would be a concurrent respiratory acidosis (e.g., an obtunded patient).

If the $PaCO_2$ was significantly lower than expected (below 26 mmHg) than a concurrent respiratory alkalosis may be present. Respiratory alkalosis with metabolic acidosis is often seen in salicylate intoxication or septic shock.

The patient is noted to have a normal anion gap, which is consistent with a metabolic acidosis caused by diarrhea.

Conclusion

Regardless of the condition of the patient, an important aspect of the ABG is to take a

systematic approach to interpretation of the ABG and determine between the differential diagnoses. Know the patient history and begin treatment as soon as feasible to ensure the best possible outcomes. Placing the patient at the top of the pyramid is the absolute most significant step in the process.

Select one of the following methods to complete this course.

	OR	
Pass an exam testing your knowledge of the course material.		Describe how this course will impact your practice. (No Test)

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