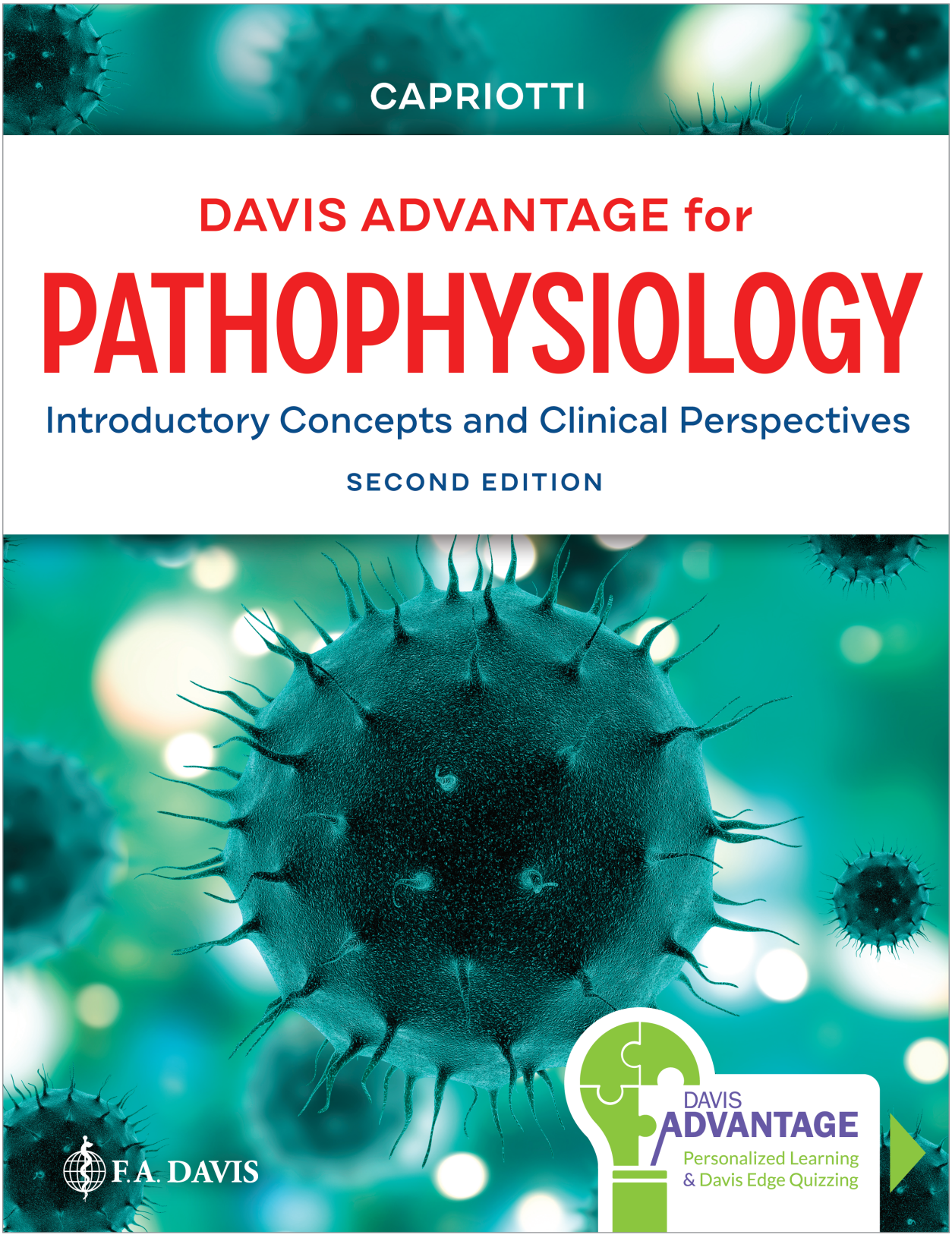
Sneak Preview!

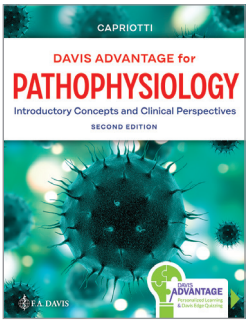


YOUR GUIDE TO

Davis Advantage for PATHOPHYSIOLOGY

SECOND EDITION

Building Confidence & Knowledge for Class and Exams



\+

\+

LEARNING **APPLYING** **ASSESSING**

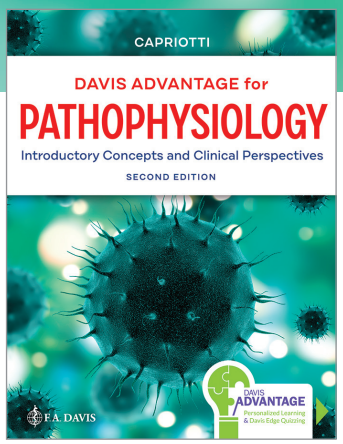
Your journey to success begins here!

Davis Advantage for **Pathophysiology** combines your textbook with an interactive, personalized learning and quizzing experience that makes this challenging, but must-know content easier to master.

CALL ON US

Learn more.
Schedule a walkthrough.
Request preview access.

800.323.3555 (US) or
800.665.1148 (CAN)
Hello@FADavis.com
www.FADavis.com/Patho



LEARNING

Connecting concepts from the written word to the real world

Pathophysiology, 2nd Edition helps you make the connections between the pathophysiologic processes you're learning about in class and the conditions you'll encounter in clinical settings.

STEP #1 Build a solid foundation.

Learning Objectives

Upon completion of this chapter, the student will be able to:

- List the four primary types of acid–base disturbances.
- Name and describe the three primary buffer systems in the body.
- Explain how the lungs and kidneys compensate for acid–base disturbances.
- Describe how pH abnormalities may cause alterations in electrolyte levels.
- Interpret arterial blood gas values to identify acid–base disturbances.

Learning Outcomes at the beginning of each chapter guide your reading and highlight the information you'll need to know.

CLINICAL CONCEPT

The two important premises to understand are:

1. **Hyperventilation reduces CO_2**, diminishing H^+ and raising pH, thus compensating for acidosis.
2. **Hypoventilation causes retention of CO_2**, which increases H^+ ion levels and decreases pH, thus compensating for alkalosis.

Clinical Concept boxes explain how key information applies to clinical practice.

ALERT! Respiratory acidosis can occur with severe asthma despite the patient having tachypnea. The breathing rate is increased, but the breaths are very shallow and do not eliminate CO_2. CO_2 accumulates, causing increased production of acids in the bloodstream.

Alerts, highlighted in red, warn you of potential problems or complications that can arise in patients based on the underlying pathophysiology.

Making the Connections

Disorder and Pathophysiology

Signs and Symptoms	Physical Assessment Findings	Diagnostic Testing	Treatment
Respiratory Acidosis | Lungs are not ventilating; retaining too much Co_2, creating too much H^+. Commonly due to COPD, severe asthma, or any cause of reduced ventilation.			
Dyspnea. Respiratory distress. Patient may be lethargic, stuporous, or comatose.	Diminished respiratory rate. Cyanosis. Clubbing if chronic hypoxia.	Uncompensated: blood pH less than 7.35. Pco_2 greater than 45 mm Hg. Po_2: low. Urine: acidic.	Treat the lung disorder for better ventilation. Bronchodilation. Antibiotics if pneumonia. Intubation and mechanical ventilation if needed.
Respiratory Alkalosis | Lungs are hyperventilating; losing too much CO_2 creates too little H^+ in the blood. Commonly due to hyperventilation secondary to anxiety or shallow respirations in asthma.			
Hyperventilation. Anxiety. Palpitations. Paresthesia. Patient may have pain.	High respiratory rate. Tachycardia.	Uncompensated: blood pH greater than 7.45. Pco_2 less than 35 mm Hg. Urine: basic.	Slow the breathing rate; CO_2 rebreather. Patient may need sedative.

Making the Connections boxes demonstrate the relationships between a disease state, the diagnosis, and nursing care.

Chapter Summary

- An acid is defined as any compound that donates hydrogen ions (H^+) in solution.
- A base is a compound that accepts H^+ ions in solution.
- When H^+ ions predominate in a solution, the solution is acidic. When basic ions predominate in a solution, the solution is alkaline.
- Buffers resist changes in pH by donating or accepting H^+ ions as needed.
- Three main buffer systems that exist in the body are protein, phosphate, and carbonic acid–bicarbonate system.
- The lungs and the kidneys regulate the body's acid–base balance through use of the carbonic acid–bicarbonate buffer system.
- Blood pH, partial pressure of oxygen (Po_2), partial pressure of carbon dioxide (Pco_2), and bicarbonate ion concentration (HCO_3^-) are the values indicated by an ABG.

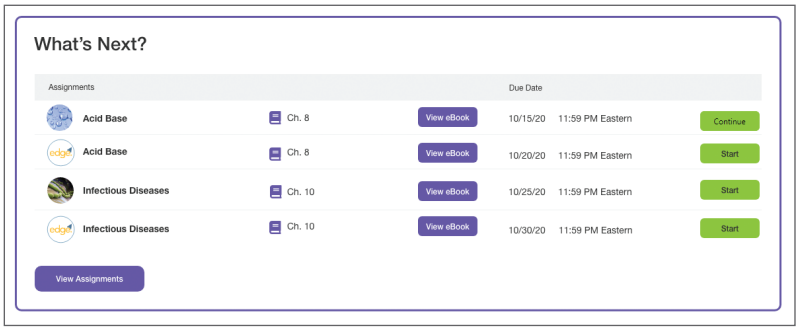
Chapter Summaries make it easy for you to review the most important concepts.

APPLYING

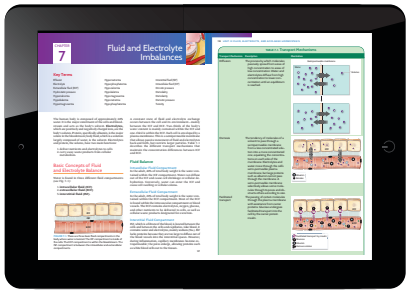
A personalized learning experience tailored to your needs

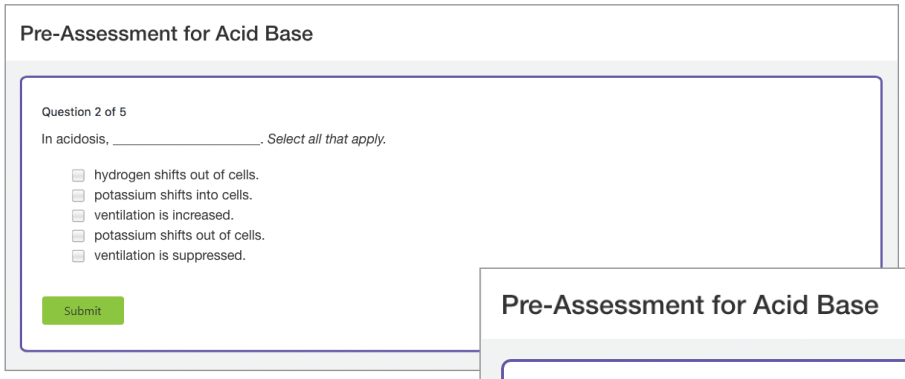
Put a study plan to work that's based on your strengths and weaknesses. Engaging, interactive content helps you make the connections and develop your understanding of key Pathophysiology topics.

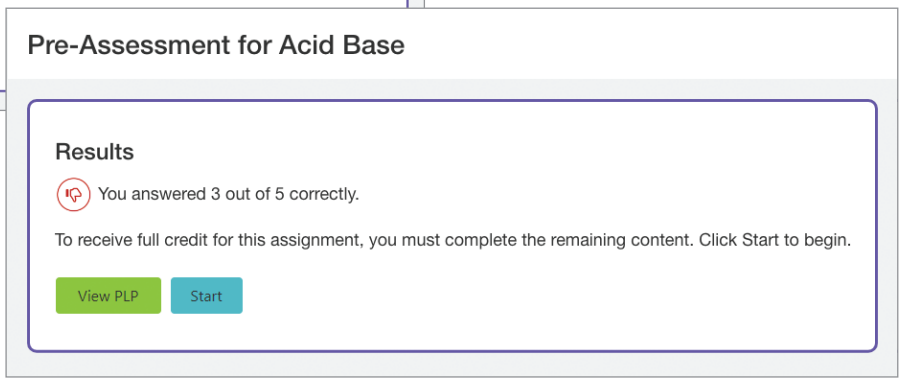
STEP #2 Make the connections and apply your knowledge.



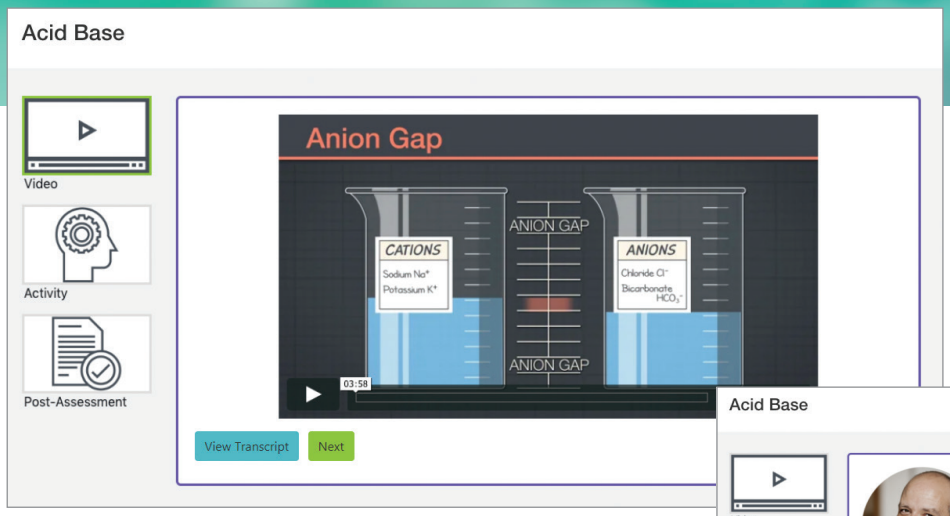
Assignments in Davis Advantage are mapped to a specific chapter in your book. Begin by reading from your printed text or click the ebook button to be taken to the chapter in your **FREE integrated ebook.**



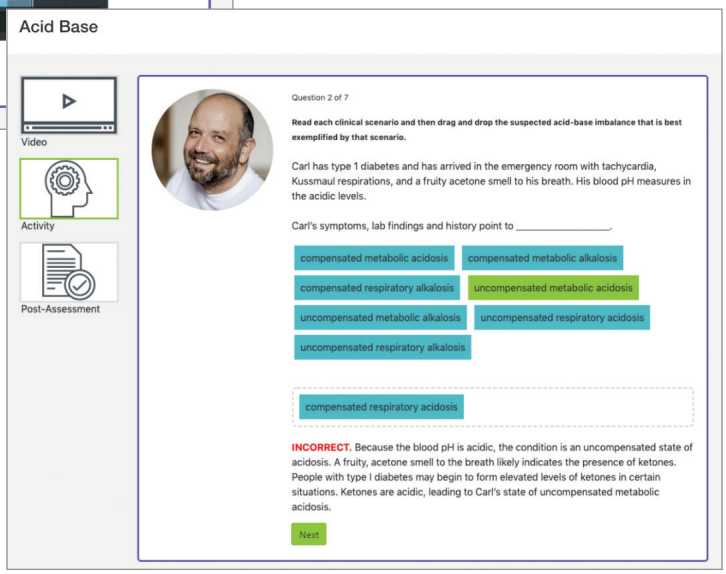




Following your reading, take the **Pre-Assessment** quiz to evaluate your understanding of the content. You'll receive **immediate feedback** that identifies your strengths and weaknesses.

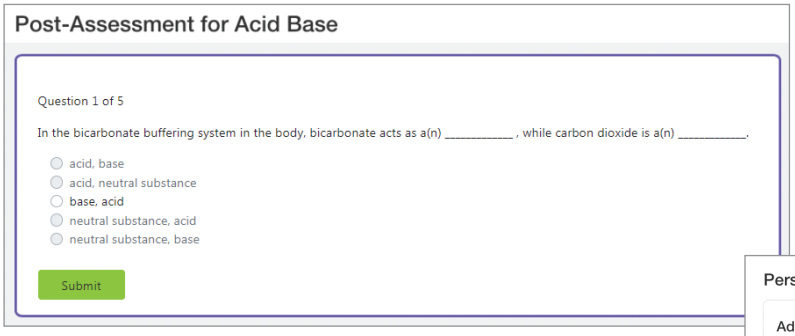


Animated mini-lecture videos make key concepts easier to understand.

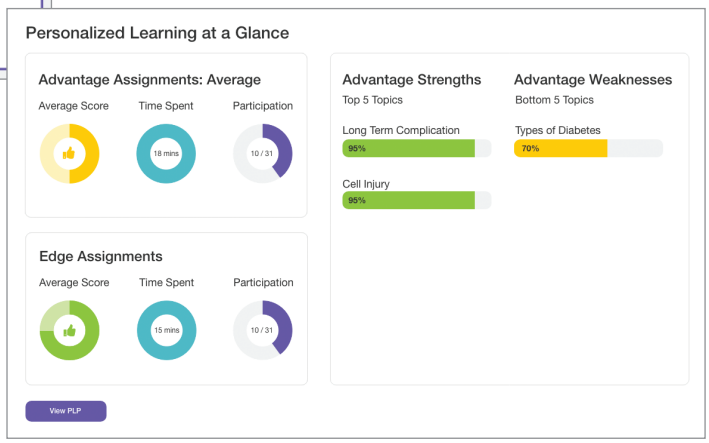


Interactive learning activities let you apply your knowledge to make the connections.

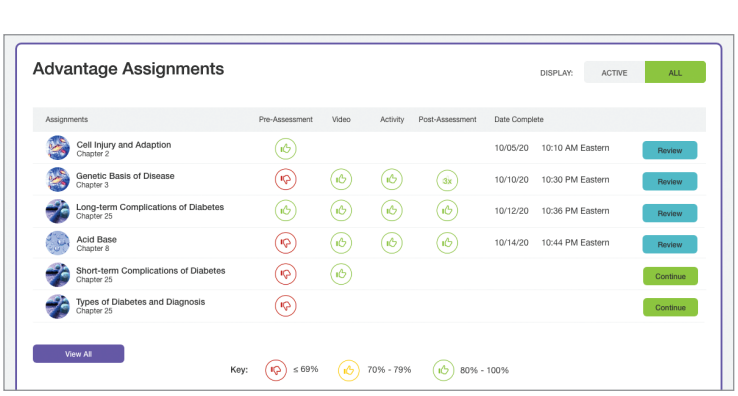
After working through the video and activity, a **Post-Assessment** quiz tests your mastery. The results feed into your **Personalized Learning Plan**, where your instructor is able to view them.



Comprehensive rationales for correct and incorrect responses help you understand why your answers are right or wrong.



Your dashboard provides a snapshot of your **performance at a glance** as you work through your assignments.



Your **Personalized Learning Plan** is mapped to your needs and tracks your progress by topic to identify the specific areas that require additional study.

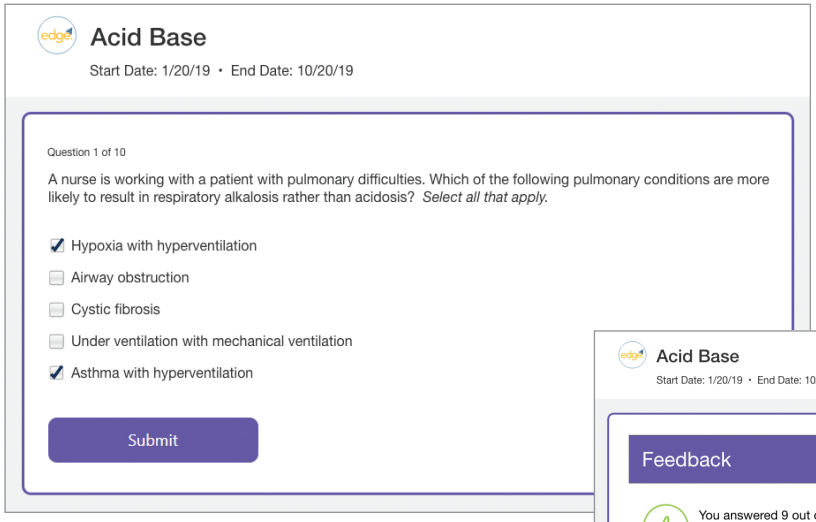
Online content subject to change upon publication.

ASSESSING

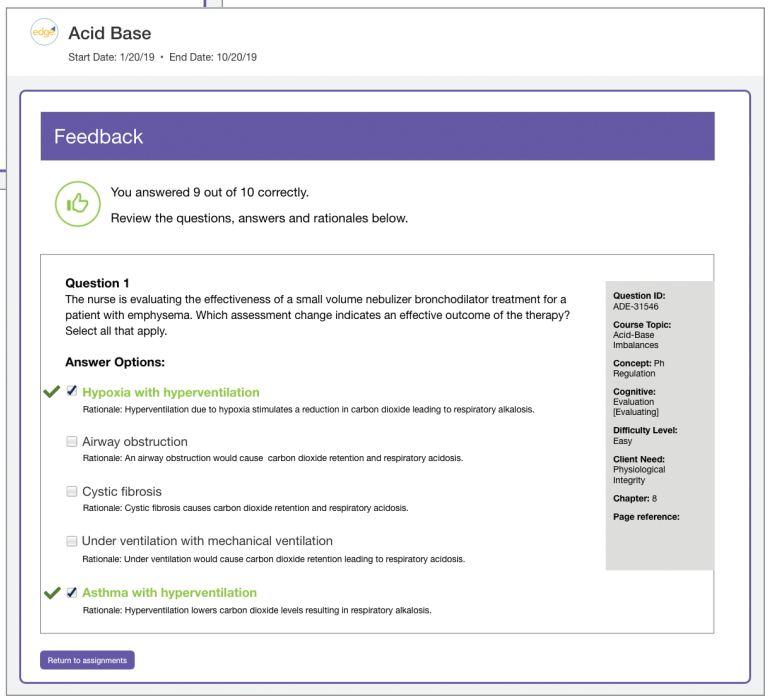
Building mastery and improving test scores with Personalized Quizzing

Q&A provides the additional practice you need to master Pathophysiology content and improve your scores on classroom exams.

STEP #3 Study smarter, not harder with Davis Edge quizzing.



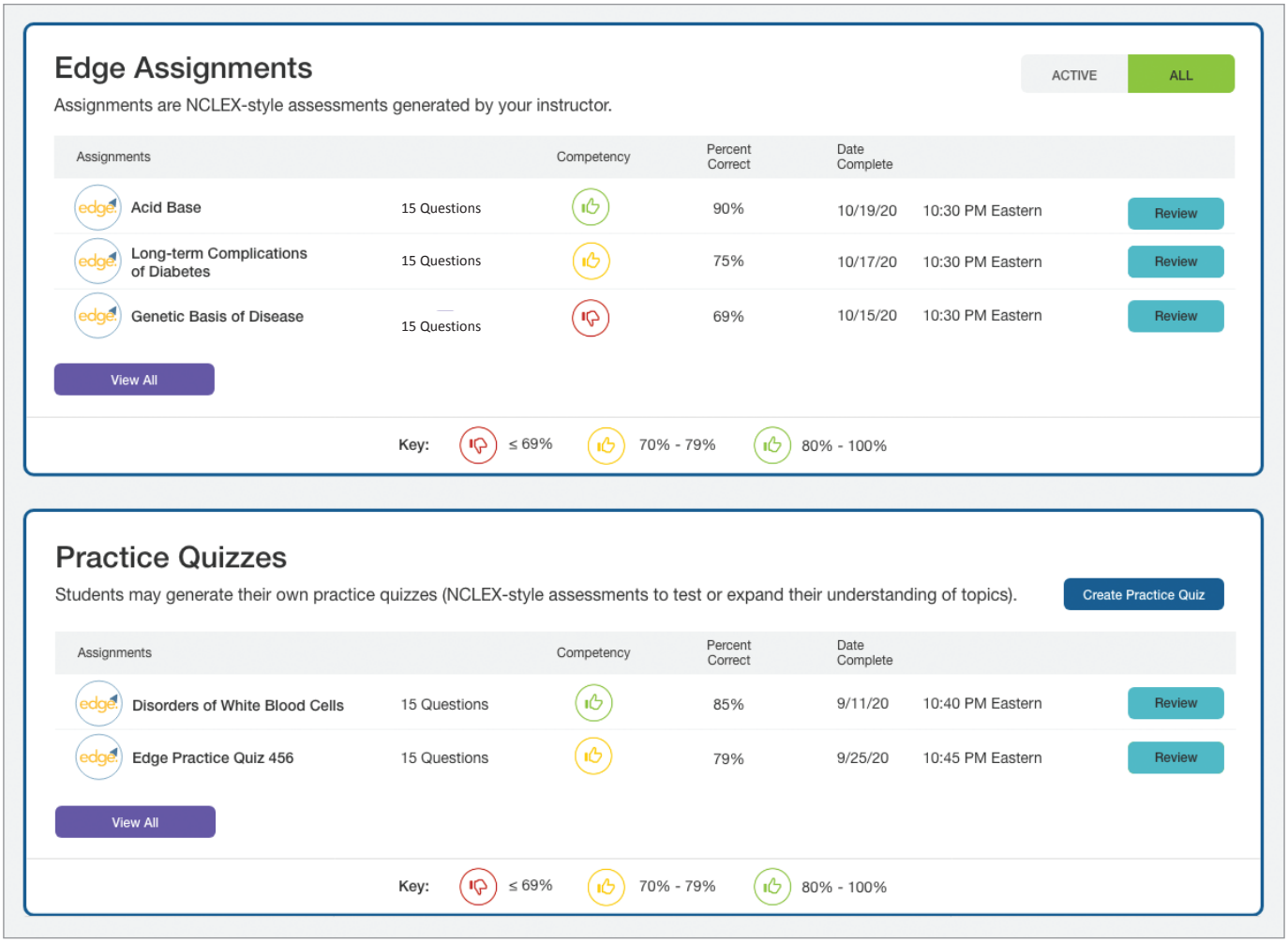
Questions assess your understanding and challenge you to think critically. Practice with standard format questions like **multiple choice,** as well as more difficult format types such as **select all that apply**.



Immediate feedback with **comprehensive rationales** explains why your responses are **correct** or **incorrect**. Page-specific references direct you to the relevant content in your *Pathophysiology* text, while **Test-Taking Tips** improve your test-taking skills.

Online content subject to change upon publication.

As you complete the **Davis Edge assignments** created by your instructor, your **Personalized Learning Plan** identifies your strengths and weaknesses topic by topic.



Create your own **practice quizzes** to focus on topic areas where you are struggling, or use as a study tool to review for an upcoming exam.

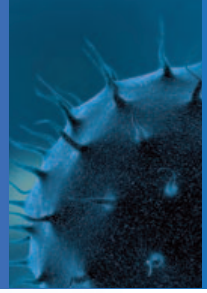
GET STARTED TODAY!

Use the access code on the inside front cover to unlock **Davis Advantage** for **Pathophysiology**!

Table of Contents

CHAPTER

8 Acid–Base Imbalances



Learning Objectives

Upon completion of this chapter, the student will be able to:

- List the four primary types of acid–base disturbances.
- Name and describe the three primary buffer systems in the body.
- Explain how the lungs and kidneys compensate for acid–base disturbances.
- Describe how pH abnormalities may cause alterations in electrolyte levels.
- Interpret arterial blood gas values to identify acid–base disturbances.

Key Terms

Acid
Acidic
Acidosis
Acidemia
Alkaline
Alkalosis
Alkalemia
Anion gap (AG)
Arterial blood gas (ABG)
Base
Basic
Blood pH
Buffer
$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$
Carbonic acid–bicarbonate system
Compensation
Metabolic acidosis
Metabolic alkalosis
Nonvolatile acid
Saturation of hemoglobin with oxygen (SaO_2)
Partial pressure of carbon dioxide (PCO_2)
Partial pressure of oxygen (PO_2)
pH
Pulse oximetry
Respiratory acidosis
Respiratory alkalosis
Volatile acid

Every second, a multitude of physiologic and biochemical reactions occur within the body. As cells require sufficient oxygen and nutrients to function normally, they also require a suitable acid–base environment. The proteins within the body contain many acidic and basic groups; thus any alteration in pH disrupts protein structure and function. To prevent such changes in pH, the body employs **buffer** systems. The body utilizes three buffer systems: proteins, phosphates, and the carbonic acid–bicarbonate system. Although all of these systems are important, the majority of this chapter focuses on the carbonic acid–bicarbonate buffer system.

Basic Concepts of Acid–Base Balance

Knowing the chemistry of acids, bases, and buffers provides a means for understanding disturbances in the body.

Acids, Bases, and Buffers

An **acid** is any compound that donates hydrogen ions (H^+) in solution. When H^+ ions predominate in a solution, the solution is **acidic**. In the body, acids are present in two forms: volatile and nonvolatile. When CO_2, a volatile gas, combines with water, the **volatile acid**, carbonic acid (H_2CO_3), forms. Carbonic anhydrase, an enzyme present in large amounts in erythrocytes, helps to catalyze this reaction. H_2CO_3 can also dissociate into CO_2 and H_2O, with the CO_2 then being exhaled by the lungs. Other acids are not converted to CO_2 and thus are referred to as **nonvolatile** (or fixed) acids.

A **base** is a compound that accepts H^+ ions in solution. When basic ions predominate in a solution, the solution is **alkaline** or **basic**.

Metabolism and Acid–Base Levels

Various compounds in the body are acidic or basic. For example, cellular metabolism of fats and carbohydrates

produces large quantities of CO_2. The CO_2 combines with H_2O in the bloodstream, forming the volatile acid, carbonic acid (H_2CO_3). Acidic products also form during hypoxic states when pyruvate converts to lactic acid during anaerobic metabolism. Metabolism of positively charged amino acids and hydrolysis of phosphates also produce acidic compounds. Most basic compounds form from the metabolism of negatively charged amino acids. By using buffers, the body counteracts potential pH changes brought on by these metabolic products.

pH Values

The H^+ ion is a very strong acid. In body fluids, however, the concentration of H^+ ions compared with other ions is extremely low. Because the hydrogen ion concentration is so small, it is expressed in terms of **pH**. The values for pH are calculated as the negative logarithm (p) of the H^+ ion concentration in mEq/L. For example, a pH value of 4 indicates that the H^+ concentration of a solution is 10^{-4} (0.00001 mEq/L). pH values and H^+ ion concentration are inversely related. A lower pH value indicates a higher concentration of H^+ ions and a more acidic solution, whereas a higher pH value represents a lower concentration of H^+ ions and a more alkaline solution (see Fig. 8-1).

Buffers

The normal range for **blood pH** is slightly basic at **7.35 to 7.45**. Deviations outside this normal range affect cellular function profoundly and are potentially life threatening. To prevent such large swings in blood pH, three buffer systems (protein, phosphate, and carbonic acid–bicarbonate) absorb excess H^+ ions or donate H^+ ions as needed.

Protein Buffering System

Because of their structure, almost all proteins can serve as functional buffers. Taken together, the proteins serve as the largest buffering system in the body. The amino and carboxyl groups found on amino acids enable proteins to absorb or donate H^+ ions as needed to maintain physiologic pH. One of the primary proteins that carries out this function is hemoglobin.

Phosphate Buffering System

Phosphates play a key role in regulating pH in the intracellular environment. Phosphates (PO_4^-) can take on an acidic form, dihydrogen phosphate, or a basic form, hydrogen phosphate, to buffer pH changes.

Carbonic Acid–Bicarbonate System

The buffering system most commonly discussed is the **carbonic acid–bicarbonate system**. Carbon dioxide, carbonic acid, hydrogen ions, and bicarbonate ions (HCO_3^-) all play a role in this buffering system. When CO_2 combines with water, H_2CO_3 (carbonic acid) is formed. The H_2CO_3 then dissociates, yielding H^+ (a strong acid) and HCO_3^- (a weak base). The chemical reaction of H_2CO_3 formation and dissociation is the following:

$$\mathbf{CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-}$$

The equation moves in both directions. When CO_2 levels are elevated, the equation *moves toward the right,* forming more H^+ and HCO_3^- ions. Likewise, when H^+ ion levels are elevated, the equation *moves toward the left*, as H^+ ions are converted to CO_2 and the CO_2 is exhaled. The carbonic acid–bicarbonate buffering system plays a significant role in the body, as two organs, the lungs and kidneys, use this buffering system to compensate for alterations in physiologic pH. Because of this system, arterial blood gases (ABGs) values include arterial carbon dioxide and bicarbonate levels, along with other factors (see Box 8-1).

Renal and Respiratory Compensations for Acid–Base Disturbances

As mentioned, the lungs and kidneys utilize the carbonic acid–bicarbonate buffering system to adjust any pH disturbances. Both organs relate to the environment in such a way that excretion or retention of acidic and basic compounds occurs in order to regulate blood pH. When the lungs and kidneys attempt to adjust pH disturbances, the process is called **compensation** (see Fig. 8-2). The lungs respond to acid–base disturbances within minutes, with the response reaching maximal levels by 24 hours. The response, though, cannot be maintained indefinitely. The kidneys require hours to a day to compensate; however, the response can be maintained for much longer.

Respiratory Compensation for Acid–Base Disturbances

Under normal conditions, the lungs correct pH imbalances by increasing or decreasing ventilation as needed. An increase in ventilation decreases CO_2. As CO_2, is exhaled, H^+ ion concentration falls (raising pH) by *moving the buffer equation toward the left*:

$$\mathbf{CO_2 + H_2O \Leftarrow H_2CO_3 \Leftarrow H^+ \text{ and } HCO_3^-}$$

Decreased ventilation retains CO_2. The retention of CO_2 *moves the buffer equation toward the right,* resulting in an elevation of H^+ ion level and a decrease in pH (see Fig. 8-3).

$$\mathbf{CO_2 + H_2O \Rightarrow H_2CO_3 \Rightarrow H^+ \text{ and } HCO_3^-}$$

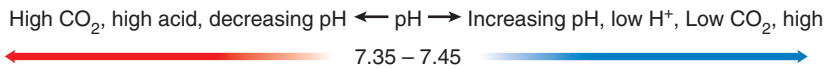


FIGURE 8-1. pH and relationship to H^+ and CO_2.

BOX 8-1. The Carbonic Acid–Bicarbonate Chemical Reaction

This chemical reaction within the bloodstream can go back and forth according to the amount of acids or bases or CO_2 in the bloodstream.

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ \text{ and } HCO_3^-$$

Example 1: If there is excess CO_2 in the bloodstream, the equation moves toward the right, which shows that more acid (H+), which is a very strong acid, is created. The bloodstream becomes high in acid in a condition called acidemia (also called acidosis). [*HCO_3^- is a weak base and cannot neutralize H^+*]

$$CO_2 + H_2O \rightarrow H_2CO_3 \rightarrow H^+ \text{ and } HCO_3^-$$

Example 2: If there is an excess of H^+ in the bloodstream, the equation moves toward the left, which shows that more CO_2 is created and CO_2 is exhaled vigorously by the lungs. This reduction of H^+ then makes the blood alkalemic (also called alkalotic).

$$CO_2 + H_2O \leftarrow H_2CO_3 \leftarrow H^+ \text{ and } HCO_3^-$$

Because of the link between ventilation, CO_2, and pH, carbon dioxide levels in the bloodstream are kept in a narrow range. The normal range for **partial pressure of carbon dioxide (Pco_2)** is **35 to 45 mm Hg.** Sometimes the Pco_2, specifically within the arterial blood, is written as $PaCO_2$. Chemoreceptors in the brain closely monitor H^+ ion levels and send signals to the respiratory center in the medulla to adjust ventilation, and subsequently carbon dioxide levels, as needed. When the blood pH is too low (acidic), the ventilation rate increases, causing exhalation of CO_2, which in turn reduces acid in the blood and raises pH. The opposite occurs when blood pH is too high (alkaline). Ventilation is suppressed, increasing CO_2 levels in the bloodstream, which creates H^+ and lowers the pH (see Table 8-1).

CLINICAL CONCEPT

The two important premises to understand are:

1. **Hyperventilation reduces CO_2**, diminishing H^+ and raising pH.
2. **Hypoventilation causes retention of CO_2**, which increases H^+ ion levels and decreases pH.

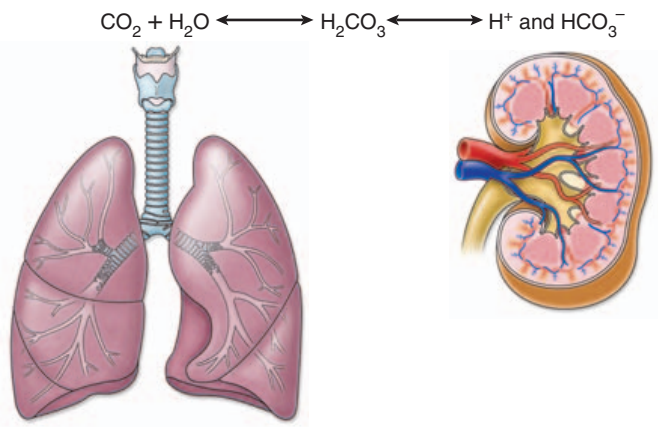


FIGURE 8-2. Acid–base balance via the lungs and kidneys.

Renal Compensation for Acid–Base Disturbances

The kidneys compensate for acid–base disturbances by regulating the excretion or reabsorption of two factors of the carbonic acid–bicarbonate system: H^+ and HCO_3^-. In conditions in which pH is too low, or acidic, the kidneys excrete more acid (H^+) and reabsorb more base (HCO_3^-). These actions lessen the amount of acid in the blood while adding more base (HCO_3^-), thereby raising blood pH and compensating for acidosis. Likewise, in conditions in which pH is too high, the kidneys reabsorb more H^+ and excrete more HCO_3, thereby lowering the pH, compensating for alkalosis. The kidneys' compensation is slow and may take days to reach maximal effectiveness. Therefore medical interventions are commonly necessary to facilitate balancing the bloodstream's pH (see Table 8-2).

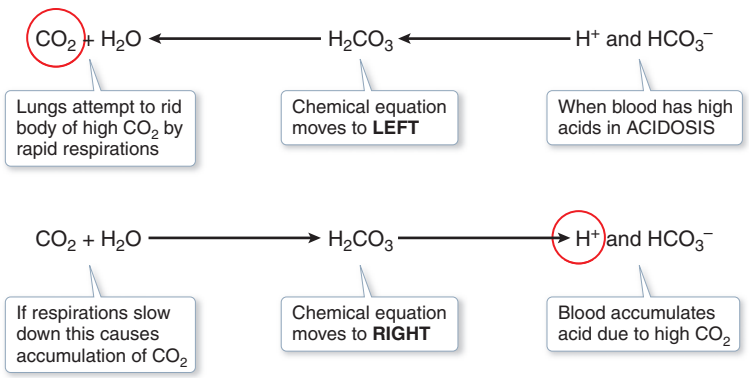


FIGURE 8-3. Respiratory compensation for acid–base disturbances.

TABLE 8-1. Respiratory Compensation for Acid–Base Imbalances and Medical Intervention

The lungs attempt to correct metabolic acid–base disturbances when they occur but are often insufficient to complete compensation. This is when medical intervention is necessary.

Condition	Blood pH	Respiratory Compensation	Medical Intervention
Metabolic acidosis *Excess H+ or lack of base is in the bloodstream due to toxicity or illness*	<7.35	Respirations increase in depth and rate to blow off CO_2	Treatment to resolve the origin of the acid–base disturbance (e.g., insulin in DKA). Sodium bicarbonate administration may be necessary.
Metabolic alkalosis *Excess base or lack of acids in blood due to toxicity or illness*	>7.45	Respirations slow down to increase CO_2 retention	Treatment to resolve the origin of the acid–base disturbance (e.g., in vomiting, administer antiemetic medication). Acetazolamide administration increases HCO_3– excretion.

TABLE 8-2. Renal Compensation in Acid–Base Disturbances and Medical Intervention

The kidneys attempt to correct acid–base disturbances when they occur but are often too slow to reach complete compensation. This is when medical intervention is necessary.

Condition	Blood pH	Renal Compensation	Necessary Medical Intervention
Respiratory acidosis *Excess CO_2 has accumulated, which is generating H^+*	<7.35	Kidneys attempt to excrete H^+ and conserve HCO_3–	Patient may need assistance with ridding body of CO_2: • Requires intubation and mechanical ventilation • Can also administer sodium bicarbonate ($NaHCO_3$)
Respiratory alkalosis *Lungs blow off too much CO_2, creating less H^+ in blood*	>7.45	Kidneys attempt to retain H^+ and excrete HCO_3–	Patient may need assistance with retaining H^+ and conserving HCO_3– • Patient can use a CO_2 rebreather mask to increase CO_2

CLINICAL CONCEPT

The lungs and kidneys are two organs that to work to maintain acid–base balance. Therefore if ventilation is suboptimal or if renal dysfunction occurs, acid–base imbalance can occur.

Arterial Blood Gases

In studying acid–base disturbances and compensation by the lungs and kidneys, **arterial blood gas (ABG)** levels are one of the most informative sets of values to consider. Analysis of ABGs helps determine the presence of acid–base imbalances within the bloodstream and whether the cause is respiratory or metabolic in nature. ABGs measure oxygenation, acidity, and alkalinity of the arterial blood. Blood pH, **partial pressure of oxygen (Po_2)** , Pco_2, and HCO_3 are the values indicated by an ABG. Sometimes the Po_2, specifically in the arterial blood, is written as PaO_2. The blood sample is commonly obtained from the radial or femoral artery. Alternatively, an indwelling arterial catheter is sometimes used for patients who require frequent ABG measurement. In addition to the values mentioned, **saturation of hemoglobin with oxygen (SaO_2)** may be reported. This measurement is obtained through a procedure called **pulse oximetry**. A pulse oximeter is a noninvasive sensor placed on the patient's finger. Normal ABG values are the following:

- **Blood pH:** 7.35 to 7.45
- **Pco_2:** 35 to 45 mm Hg
- **Po_2:** 90 to 100 mm Hg
- **HCO_3^-:** 22 to 26 mEq/L
- **SaO_2:** 95% to 100%.

CLINICAL CONCEPT

When measuring ABGs, low Po_2 is classified as *hypoxia* or *hypoxemia,* elevated Pco_2 is termed *hypercapnia,* and diminished Pco_2 is termed *hypocapnia*.

Arterial Blood Gases and Acid–Base Disorders

The ability to interpret ABGs affords an understanding of the etiology behind acid–base disorders. The normal range for blood pH is 7.35 to 7.45. If blood pH is lower

than 7.35, the bloodstream is acidic and the condition is termed **acidemia** (also called **acidosis**). If the blood pH is greater than 7.45, the bloodstream is basic and the condition is termed **alkalemia** (also called **alkalosis**). In addition to these classifications of acidosis and alkalosis, acid–base disturbances are categorized as respiratory or metabolic, based on the origin of the disturbance.

Respiratory Acid–Base Disturbances

Respiratory acidosis and alkalosis are marked by abnormalities in carbon dioxide levels, leading to the acid–base disturbance. For example, in **respiratory acidosis**, retention of CO_2 causes a reduction in pH. Often, respiratory acidosis arises with compromised gas exchange in the lungs, as may occur with chronic obstructive pulmonary disease (COPD), infection, foreign body obstruction, and asthma. In **respiratory alkalosis**, lower-than-normal CO_2 levels reduce H^+ ion levels and increase pH. Respiratory alkalosis occurs with hyperventilation.

Metabolic Acid–Base Disturbances

Metabolic acid–base disturbances involve an origin other than the pulmonary system and abnormal CO_2 levels. Metabolic acid–base disturbances may manifest for a variety of reasons, including toxicity, diabetes, renal failure, and excessive gastrointestinal (GI) losses. **Metabolic acidosis** may result from increased production of acids other than CO_2, as occurs in diabetic ketoacidosis (DKA), or from the excessive loss of a base, such as bicarbonate through, for example, prolonged diarrhea. **Metabolic alkalosis** develops from excess base, such as retention of sodium bicarbonate or from loss of H^+ ions, as may result from prolonged vomiting.

When interpreting ABGs, a clinician needs to determine if the patient is enduring an acid–base imbalance and, if so, the source of the imbalance, whether respiratory or metabolic.

ABG values are not the whole story, however. They must be interpreted in relation to the patient's vital signs, history, and physical examination. When analyzing ABG results in conjunction with all other indicators, use questions in a step-by-step process (see Box 8-2). Remember to evaluate the whole clinical picture, including Po_2 and SaO_2.

Anion Gap

In addition to ABGs, another piece of information useful in determining the cause of an acid–base imbalance is the anion gap. The term **anion gap (AG)** represents the concentration of the *unmeasured* anions (negatively charged ions) in the bloodstream when comparing the

BOX 8-2. Steps for Interpreting Basic Arterial Blood Gas Disturbances

When analyzing ABG lab results, remember that the patient's clinical condition must be taken into consideration. ABG lab results should not be analyzed alone apart from the patient condition. For example, ask if the patient is having difficulty breathing or is breathing rapidly. Look at the respiratory rate. Does the patient have a renal or gastrointestinal disorder? Is it possible that the patient has drug or chemical toxicity or diabetic ketoacidosis? Or lactic acidosis?

Step 1. Begin by asking if the blood pH is acidic, basic, or within the normal range.

- pH <7.35 indicates acidosis
- pH >7.45 indicates alkalosis
- pH 7.35–7.45
  - Normal if PCO_2 and HCO_3– are normal
  - Normal but compensations are occurring if PCO_2 and HCO_3– are abnormal

Step 2. Identify the PCO_2 level. Is it high, low, or normal?

- Normal = 35–45 mm Hg
- High >45 mm Hg
- Low <35 mm Hg

Step 3. Determine if the acid–base disturbance is respiratory or metabolic.

Comparing the pH and PCO_2 levels enables you to determine if the respiratory system is the origin of the acid–base imbalance or if the acid–base imbalance is metabolic in origin. Examine both the pH and the PCO_2 levels and use these rules:

- If ***pH and PCO_2 are moving in opposite directions***, meaning if the PCO_2 is high and the pH is low, then the PCO_2 levels are contributing to the acid–base disturbance and thus it is ***respiratory in nature***. If the PCO_2 is low and the pH is high, the PCO_2 levels are contributing to the acid–base disturbance and thus it is ***respiratory in nature***.
- If ***PCO_2 is normal or is moving in the same direction as pH, meaning if the PCO_2 is normal or high and the pH is high,*** the condition is ***metabolic in nature*** (i.e., not due to respiratory involvement). If the PCO_2 is normal or low and pH is low, the condition is ***metabolic in nature*** and is not due to respiratory involvement.

Examples of Step 3:

Respiratory acidosis: pH <7.35 and PCO_2 >45 mm Hg

- pH is low = acidosis, PCO_2 is high
- pH and CO_2 are moving in opposite directions. The high CO_2 is contributing to the acidic pH; thus the acid–base imbalance is respiratory in origin.

Respiratory alkalosis: pH >7.45 and PCO_2 <35 mm Hg

- pH is high = alkalosis, PCO_2 is low
- pH and CO_2 are moving in opposite directions. The low CO_2 is contributing to the basic pH; thus the acid–base imbalance is respiratory in origin.

Continued

BOX 8-2. Steps for Interpreting Basic Arterial Blood Gas Disturbances–cont'd

Metabolic acidosis: pH <7.35 and PCO_2 <35 mm Hg or normal

- pH is low = acidosis, PCO_2 is low
- pH and CO_2 are moving in the same direction. The pH is low (acidic) and CO_2 is low; thus CO_2 is not causing the acidic pH so the disturbance is metabolic in origin.

Metabolic alkalosis: pH >7.45 and PCO_2 >45 mm Hg

- pH is high = alkalosis, PCO_2 is high
- pH and CO_2 are moving in the same direction. The pH is high (basic) and CO_2 is high; thus CO_2 is not causing the alkaline pH, so the disturbance is metabolic in origin.

Step 4. Determine if the condition is a compensated or uncompensated condition.

Uncompensated: pH will be abnormal

- Use the previous steps to determine if the condition is respiratory or metabolic in nature.

Compensated: pH is normal or nearing normal

- Use the previous steps to determine if PCO_2 is contributing to the original acid–base disturbance. If so, a compensated respiratory condition is present. If not, the disorder is metabolic in nature, and PCO_2 is compensating.

COMPENSATED RESPIRATORY ACIDOSIS

pH = initially low, elevating to normal, PCO_2 >45 mm Hg, HCO_3– (>26 mEq/L)

- pH = initially low, PCO_2 = high
- pH and PCO_2 are initially in opposite direction, so it is respiratory in nature
- High bicarbonate compensating for high CO_2 and acidic pH

COMPENSATED RESPIRATORY ALKALOSIS

pH = initially high, lowering to normal, PCO_2 <35 mm Hg, HCO_3– (<22 mEq/L)

- pH = initially high, PCO_2 = low
- pH and PCO_2 are initially in opposite direction, so it is respiratory in nature
- Low HCO_3– compensating for low CO_2 and basic pH

COMPENSATED METABOLIC ACIDOSIS

pH = initially low, elevating to normal, PCO_2 <35 mm Hg, HCO_3– (<22 mEq/L)

- pH = initially low, PCO_2 = low
- pH and PCO_2 are initially in same direction, so it is metabolic in nature
- Original acidity of blood not due to CO_2, low CO_2 is compensating for low base (i.e., acidic pH).

COMPENSATED METABOLIC ALKALOSIS

pH = initially high or lowering to normal, PCO_2 >45 mm Hg, HCO_3– (>26mEq/L)

- pH = initially high, PCO_2 = high
- pH and PCO_2 are initially in same direction, so it is metabolic in nature
- Original alkalinity of blood not due to high CO_2, high CO_2 is compensating for alkalinity of the blood

CASE STUDY 1

Patient A is enduring an asthma attack and is brought into the emergency department. The patient's vital signs are: Temp: 98.4°F, Pulse: 110 beats/min, Resp rate: 24 shallow breaths/min, BP: 136/86 mm Hg.

ABGs are:

- Blood pH: 7.30
- P_{CO_2}: 58 mm Hg
- P_{O_2}: 88 mm Hg
- HCO_3^-: 28 mEq/L
- SaO_2: 88%.

Step 1. Does the blood pH show an acidotic, alkalotic, or normal bloodstream?

In this problem, the blood is acidic at pH 7.30, which is less than 7.35; therefore the condition is acidosis.

Step 2. What is the P_{CO_2}?

A P_{CO_2} of 58 mm Hg is elevated beyond the normal range of 35–45 mm Hg.

Step 3. Is the acid-base imbalance caused by a respiratory or metabolic source?

pH (low) and PCO_2 (high) are moving in opposite directions. The high PCO_2 is causing the low pH, indicating it is a respiratory disturbance and therefore respiratory acidosis.

The high P_{CO_2} indicates a ventilation problem, and the P_{O_2} and SaO_2 are also low, further confirming a lung problem.

Step 4. Is this a compensated or uncompensated problem?

The pH is abnormal; therefore the condition is uncompensated. The body is attempting to compensate by reabsorption of HCO_3^- at the kidney. HCO3- is slightly elevated at 28 mEq/L vs. the normal range of 22–26 mEq/L.

Result

Because of the low blood pH and high P_{CO_2}, this is **uncompensated respiratory acidosis.** The kidney is attempting to compensate through the reabsorption of HCO_3^-.

CASE STUDY 2

Patient B is unconscious and brought into the emergency department because of suspected drug toxicity. Vital signs include: Temp: 97.8°F, Pulse: 90 beats/min, Resp rate: 12 breaths/ min, BP: 100/70 mm Hg.

The patient's ABGs are:

- Blood pH: 7.29
- P_{CO_2}: 32 mm Hg
- P_{O_2}: 95 mm Hg
- HCO_3^-: 13 mEq/L
- SaO_2: 98%.

Using the previous step-by-step process:

Step 1. Does the blood pH show an acidotic, alkalotic, or normal bloodstream?

In this problem, the pH is less than 7.35; therefore the condition is acidosis.

Step 2. What is the P_{CO_2}?

The P_{CO_2} is 32 mm Hg, which is low. This indicates the lungs are eliminating CO_2 excessively.

BOX 8-2. Steps for Interpreting Basic Arterial Blood Gas Disturbances–cont'd

Step 3. Is the acid–base imbalance caused by a respiratory or metabolic source?

As pH (low) and PCO_2 (low) are moving in the same direction, PCO_2 is not contributing to the acid–base imbalance. The acid–base disturbance is metabolic in nature. Also, because the Po_2 and SaO_2 are normal, the lungs are functioning well.

Step 4. Is this a compensated or uncompensated problem?

The pH is abnormal, so the condition is uncompensated. The lungs in this case are trying to compensate for the low pH in the bloodstream by exhaling CO_2, but the compensation is inadequate.

Result

Because of the low blood pH and low Pco_2, the condition is uncompensated metabolic acidosis. The lungs attempt to compensate for the acidosis by increasing ventilation to decrease CO_2. In cases of metabolic acidosis, a further step would be to calculate the anion gap to help narrow the list of possible causes.

CASE STUDY 3

Patient C is having an anxiety attack and comes to the emergency department. Vital signs are as follows: Temp: 98.1°F, Pulse: 121 beats/min, Resp rate: 28 breaths/min, and BP: 138/88 mm Hg.

The patient's ABGs are:

- Blood pH: 7.58 mm Hg
- Pco_2: 28 mm Hg
- Po_2: 93 mm Hg
- HCO_3^-: 22 mEq/L
- SaO_2: 92%.

Step 1. Does the blood pH show an acidotic, alkalotic, or normal bloodstream?

The pH is greater than 7.45; therefore the condition is alkalosis.

Step 2. What is the Pco_2?

The Pco_2 is 28 mm Hg, which is low. The lungs are hyperventilating, exhaling CO_2.

Step 3. Is the acid–base imbalance caused by a respiratory or metabolic source?

As pH (high) and PCO_2 (low) are moving in opposite directions, a respiratory condition is occurring. The Po_2 and SaO_2 are on the low side, additionally indicating a pulmonary problem.

Step 4. Is this a compensated or uncompensated problem?

The pH is abnormal; therefore this is uncompensated. The hyperventilation is causing a reduction in CO_2, elevating pH. The kidneys are attempting to compensate by excreting HCO_3^-.

Result

Because of the high blood pH, low Pco_2, and low HCO_3^-, this is uncompensated respiratory alkalosis.

CASE STUDY 4

Patient D has endured 3 days of nausea and vomiting caused by a virus. Vital signs are as follows: Temp: 101.1°F, Pulse: 98 beats/min, Resp rate: 12 breaths/min, BP: 90/60 mm Hg.

The patient's ABGs are:

- Blood pH: 7.61 mm Hg
- Pco_2: 49 mm Hg
- Po_2: 99 mm Hg
- HCO_3^-: 49 mEq/L
- SaO_2: 99%.

Step 1. Does the blood pH show an acidotic, alkalotic, or normal bloodstream?

A pH of 7.61 indicates alkalosis.

Step 2. What is the Pco_2?

The Pco_2 is high.

Step 3. Is the acid–base imbalance caused by a respiratory or metabolic source?

In this case, pH (high) and PCO_2 (high) are moving in the same direction, thus indicating a metabolic acid–base disturbance. The high CO_2 is not causing the alkaline pH. Also, the Po_2 and SaO_2 are normal, indicating normal lung function. Therefore the lungs are not causing the alkalosis.

Step 4. Is this a compensated or uncompensated problem?

The pH is abnormal, so this is uncompensated. The body is attempting to retain acids in the bloodstream by slow respirations (which increases CO_2) and neutralize the elevated bicarbonate levels.

Result

Because of a high blood pH and high Pco_2, this is an uncompensated metabolic alkalosis. The lungs are attempting to compensate by breathing slowly but cannot accomplish full compensation.

CASE STUDY 5

Patient E presents to the emergency department in a coma with no history. The ABGs are:

- Blood pH: 7.37
- Pco_2: 47 mm Hg
- Po_2: 85 mm Hg
- HCO_3^-: 28 mEq/L
- SaO_2: 87%.

Step 1. Does the blood pH show an acidotic, alkalotic, or normal bloodstream?

In this problem, the blood pH is between 7.35 and 7.45, which is within the normal range. However, both PCO_2 and HCO_3^- values are abnormal, indicating some type of compensation.

Step 2. What is the Pco_2?

The Pco_2 is 47 mm Hg. This is a high Pco_2, which means the lungs are hypoventilating and retaining CO_2.

Continued

BOX 8-2. Steps for Interpreting Basic Arterial Blood Gas Disturbances—cont'd

Step 3. Is the acid–base imbalance caused by a respiratory or metabolic source?

pH is normal, but PCO_2 is high, as is HCO_3^-, so further analysis is required. Compensation for an acid–base disturbance is likely occurring. The low P_{O_2} and SaO_2, coupled with the high PCO_2, indicate a pulmonary problem is likely. The elevated bicarbonate levels are indicative of a compensation to address the higher-than-normal PCO_2 due to respiratory problems.

Step 4. Is this a compensated or uncompensated problem?

The blood pH is normal, whereas PCO_2 and HCO_3^- are abnormal, indicating a compensated condition. The kidneys are trying to reabsorb enough HCO_3^- to neutralize the H^+ caused by high P_{CO_2}.

Result

Because of the normal pH, high P_{CO_2}, and high HCO_3^-, this is compensated respiratory acidosis.

measured cations (positively charged ions), [Na^+] and [K^+], and *measured* anions, [Cl^-] and [HCO_3^-], of the blood. The AG is calculated by summing the measured cations, [Na^+] and [K^+], and subtracting the measured anions, [Cl^-] and [HCO_3^-]. To maintain electrochemical balance, the body's total number of cations should equal the total number of anions. Normally, though, a small number of unmeasured free anions exist in the bloodstream. These unmeasured anions comprise the normal AG between measured cations and anions (see Fig. 8-4). The major unmeasured anions include negatively charged plasma proteins (albumin), sulfates, and phosphates. The calculation of anion gap only applies to cases of metabolic acidosis.

Anion Gap Range

The normal range for the AG is 8 to 16 mEq/L, although laboratories may slightly differ in their reference range. The AG can be high, normal, or low. A normal AG has a small number of unmeasured anions in the bloodstream (see Fig. 8-4). A low AG is uncommon but can be caused by decreased unmeasured anions (usually low protein, especially hypoalbuminemia), increased unmeasured cations, or laboratory error. A high AG has a value between 16 and 20 mEq/L and may occur in specific instances of metabolic acidosis, such as DKA.

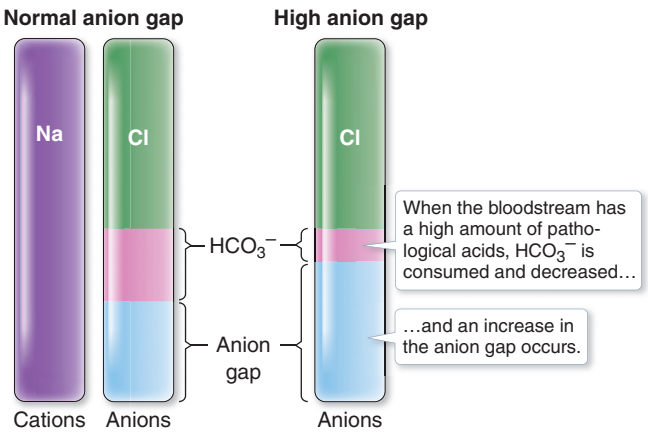


FIGURE 8-4. The normal anion gap versus high anion gap within the bloodstream. The bloodstream normally has a certain level of unbound anions, called the normal anion gap. When the bloodstream has an increased amount of acids, as in diabetic ketoacidosis, the anion gap is increased.

CLINICAL CONCEPT

The AG equation given by [$Na^+ + K^+$] – [$Cl^- + HCO_3^-$] works on the assumption that a patient has a normal albumin level. Low serum albumin reduces the accuracy of the AG calculation.

Increased Anion Gap in Metabolic Acidosis

In certain instances of metabolic acidosis, the AG increases. The increase occurs when large amounts of unmeasured acids, such as ketones in DKA, are added to the blood. Ketones dissociate into H^+ ions and keto-anions. Bicarbonate, a measured anion, buffers the H^+ ions. As this buffering occurs, the amount of measured anions (bicarbonate) in the blood decreases, whereas the unmeasured anions (keto-anions) increases. The AG, indicative of unmeasured anion, thus increases. The AG becomes important to clinicians in differentiating underlying causes of metabolic acidosis, as some causes of metabolic acidosis, such as GI loss of bicarbonate, do not present with an elevated AG.

CLINICAL CONCEPT

Calculating the AG is clinically useful, as it helps differentiate types of metabolic acidosis disease states.

Metabolic acidosis with an elevated AG is found in the following conditions:

- Lactic acidosis
- Ketoacidosis
- Renal failure

- Overdose of acetylsalicylic acid (ASA), also known as aspirin
- Ingestion of methanol or ethylene glycol

Metabolic acidosis with a normal AG is found in the following conditions:

- GI loss of HCO_3^-
- Increased renal HCO_3^- loss
- Hypoaldosteronism
- Ingestion of ammonium chloride
- Hyperalimentation

Acid–Base Disturbances and Electrolytes

Changes in pH can influence the movement of ions between the intracellular fluid (ICF) and extracellular fluid (ECF), and vice versa, and changes in electrolyte levels can influence the pH state. Ion movement is driven by the electrochemical gradient. Changes in this gradient, due to changes in one or more ions, can profoundly affect the movement of other ions. Two of the ions most affected by alterations in pH levels are K^+ and Ca^{++}. Many of the systemic signs and symptoms of acid–base disturbances are not simply due to pH changes, but rather the impact of H^+ ion concentration on electrolyte movement.

Relationship Between H+ and K+

Both K^+ and H^+ ions are positively charged, and both ions move freely between the ICF and ECF. As such, these ions are often exchanged for one another, and changes in H^+ concentration can affect the movement of K^+ ions and vice versa. In the case of acid–base disturbances, shifts in potassium are more pronounced in acidosis than alkalosis and are also greater in metabolic acidosis than respiratory acidosis

Acidosis and Hyperkalemia. In the intracellular and extracellular electrolyte environment, excess H^+ in the bloodstream causes ion movements. H^+ ions move into the cells, and K^+ ions move out of the cells into the bloodstream. The bloodstream thus becomes high in K^+, causing *hyperkalemia*. However, the total body level of K^+ is unchanged. The bloodstream appears as though it is hyperkalemic. As soon as the state of acidosis resolves, the K^+ levels re-equilibrate between the intracellular and extracellular environment.

Also, in acidosis, the kidney, which usually excretes K^+, is inundated with H^+ and selectively excretes H^+ in lieu of K^+. This causes K^+ to accumulate in the bloodstream because the kidney is not excreting it. Therefore although the blood may seem to have high potassium levels, the body content of K^+ is not changed. However, high levels of K^+ retained in the blood (hyperkalemia) are a serious complication of acidosis because this has effects on cardiac tissue. Hyperkalemia can cause dysrhythmias, or in severe cases, cardiac arrest. As soon as the acidosis condition is resolved, K^+ is once again excreted by the kidney and blood levels equilibrate back to normal.

CLINICAL CONCEPT

Intravenous (IV) fluid replacement with K^+ may be needed in some states of acidosis due to the shift of K^+ from the intracellular to the extracellular space and potential K^+ ion loss in the urine.

Alkalosis and Hypokalemia. As acidosis may lead to hyperkalemia, alkalosis is linked to hypokalemia, as K^+ ions shift into the cells from the plasma. This movement of potassium from the ECF to the ICF lowers serum potassium, resulting in *hypokalemia*. Also, in alkalosis, transport mechanisms in the kidneys result in additional K^+ loss in the urine.

The Effect of Potassium Levels on pH. Because of the interrelatedness of H^+ and K^+ ion movements, changes in K^+ ion levels can lead to acid–base disturbances. Hypokalemia may cause alkalosis, as H^+ shifts into cells to compensate for lower-than-normal K^+ levels, whereas hyperkalemia may cause acidosis as H^+ ions enter the bloodstream.

The Effect of pH on Calcium Levels. Calcium ion levels are also affected by pH disturbances. Calcium is transported in the blood in a free, ionized form or attached to the plasma protein albumin. The binding and transport of calcium by albumin is a reversible process influenced by H^+ ion concentration. In acidosis, H^+ ions compete with Ca^{++} ions for binding sites on albumin. Thus in acidosis, free, ionized forms of calcium increase, leading to hypercalcemia. In alkalosis, with fewer H^+ ions to compete for binding sites on albumin, free, ionized calcium levels decrease as more calcium binds to albumin. Alkalosis is thus associated with hypocalcemia.

pH, Electrolyte Levels, and Cellular Functioning. Electrolyte disturbances due to changes in pH can have a profound impact on several cellular processes, particularly in excitable cells such as neurons and muscle cells, including the cells of the heart. Potassium, in particular, affects the resting membrane potential of myocardial cells.

In acidosis, which may result in hyperkalemia, the increased K^+ ion levels cause the resting membrane potential of cells to become more positive, making them hyperexcitable. In hypokalemia associated with alkalosis, reduced K^+ ion levels cause the resting membrane potential to become less positive and the cells less excitable. Changes in the resting membrane potential affect the functioning of the heart, neurons, and muscles. The heart's functionality can be compromised to the point that severe arrhythmias, and even cardiac arrest, develop.

pH-induced changes in albumin binding affinity for calcium and the subsequent changes in free, ionized calcium also negatively affect cells. Hypercalcemia,

which can develop in acidosis, increases the threshold for depolarization, making cells less excitable. In hypocalcemia due to alkalosis, lower-than-normal Ca^{++} levels increase the excitability of cells. The link between pH, K^+, and Ca^+ levels and cell excitability provides yet another example of the critical importance of maintaining these factors within a narrow range.

Pathophysiologic Concepts Regarding Acid–Base Imbalances

There are four states of acid–base imbalance in the bloodstream:

1. Respiratory acidosis
2. Respiratory alkalosis
3. Metabolic acidosis
4. Metabolic alkalosis

Each has a different etiology, clinical presentation, compensatory mechanism, and treatment.

Respiratory Acidosis

Respiratory acidosis occurs when the body accumulates too much CO_2 and cannot exhale it sufficiently. The development of respiratory acidosis indicates inadequate exchange of carbon dioxide within the lungs, leading to an elevation in CO_2 known as *hypercapnia*. Hypercapnia pushes the carbonic acid–bicarbonate buffer equation to the right, producing more H^+ and HCO_3^-:

$$\mathbf{CO_2 + H_2O \Rightarrow H_2CO_3^- \Rightarrow H^+ \text{ and } HCO_3^-}$$

As CO_2 converts into H^+ ions, pH levels fall. The shift in pH due to elevated CO_2 can occur rapidly or over an extended period. The hallmark of respiratory acidosis is a pH below 7.35 and a P_{CO_2} above 45 mm Hg.

Epidemiology

The incidence of respiratory acidosis is different for its varied etiologies.

Etiology

Box 8-3 lists common causes of respiratory acidosis.

Pathophysiology

Respiratory acidosis develops when the lungs are unable to remove sufficient CO_2, causing it to accumulate in the bloodstream. When CO_2 is high, the $\mathbf{CO_2 + H_2O \rightarrow H_2CO_3 \rightarrow H^+ \text{ and } HCO_3^-}$ shifts to the right, creating increased acid (H^+). Higher H^+ ion levels reduce the pH. In respiratory acidosis, insufficient CO_2 elimination leads to CO_2 levels rising above 45 mm Hg.

Chemoreceptors in Chronic Respiratory Acidosis

In chronic respiratory acidosis, as may occur with COPD, the respiratory center in the medulla becomes insensitive to high CO_2 levels. Normally when CO_2 rises in the bloodstream, the chemoreceptors in the medulla stimulate increased ventilation to rid the body of CO_2. However, in long-term COPD, high CO_2 levels do not stimulate the medulla and respirations as expected. Patients with long-term COPD live with hypercapnia and precariously balanced blood pH values because of this adaptation.

BOX 8-3. Causes of Respiratory Acidosis

PULMONARY

- Chronic obstructive lung disease, such as asthma, emphysema, and bronchiectasis
- Pulmonary edema
- Pneumonia
- Airway obstruction, such as laryngospasm, bronchospasm, and aspiration
- Underventilation by mechanical ventilation
- Hypoventilation secondary to obesity, postoperative pain, abdominal distention, or use of abdominal binders
- Excessive fatigue or weakness of rib cage muscles
- Cystic fibrosis

NONPULMONARY

- Overdosage of anesthetic, sedatives, and narcotics
- Neuromuscular disorders, such as Guillain–Barré, myasthenia gravis, and advanced multiple sclerosis
- Severe spinal deformities
- Central nervous system depression related to cerebral infarct, meningitis, or trauma
- Cardiopulmonary arrest

CLINICAL CONCEPT

Patients with long-term COPD retain CO_2, which increases susceptibility to respiratory acidosis.

Clinical Presentation

Patients in respiratory acidosis complain of anxiety, restlessness, headache, lethargy, fatigue, shortness of breath, rapid breathing, and cough. Advanced respiratory acidosis leads to confusion, somnolence, and possible coma. The effects of excess CO_2 are commonly referred to as "carbon dioxide narcosis."

Physical Examination Findings

The thoracic examination of patients with respiratory acidosis usually reveals obstructive lung disease with compromised air exchange. The signs include diffuse wheezing, hyperinflation of the lungs, barrel-shaped chest in emphysema, decreased breath sounds, hyperresonance on percussion, and prolonged expiration. Rhonchi may also be heard. Cyanosis and clubbing may

indicate the presence of chronic hypoxia. Confusion, disorientation, somnolence, or stupor can be present with high levels of P_{CO_2}.

ALERT! Respiratory acidosis can occur with severe asthma despite the patient having tachypnea. The breathing rate is increased, but the breaths are very shallow and do not eliminate CO_2. CO_2 accumulates, causing increased production of acids in the bloodstream.

Compensatory Mechanisms and Values

As discussed, the kidneys are the primary means of compensation when an acid–base imbalance is respiratory in nature. In respiratory acidosis, respiratory compensation is incapable of totally counteracting the pH disturbance. In acute respiratory acidosis, the kidneys attempt to compensate by reabsorbing HCO_3^- and excreting H^+. ABG values for *uncompensated respiratory acidosis* reveal a pH less than 7.35 and a CO_2 level greater than 45 mm Hg. If the kidneys can successfully compensate for the pH abnormality by reabsorbing additional bicarbonate (HCO_{3^-}), then pH value normalizes. ABG values for *compensated respiratory acidosis* reveal a pH that is normal, CO_2 level greater than 45 mm Hg.

Treatment

Treatment of respiratory acidosis centers on improving gas exchange. Oxygenation of blood may be maintained by administering oxygen. Bronchodilation is attempted via oral and parenteral adrenergic agents. If the compromised lung function is due to a pulmonary infection, treatment of the infection is required. If gas exchange does not improve, endotracheal intubation with mechanical ventilation is necessary.

Respiratory Alkalosis

Respiratory alkalosis occurs when CO_2 levels in the blood are low, often due to hyperventilation. Causes of hyperventilation include stress and anxiety, drug toxicity, and head injuries. The reduction in CO_2 resulting from hyperventilation lowers H^+ ion levels and elevates pH, shifting the carbonic acid–bicarbonate buffer equation toward the left:

$$\mathbf{CO_2 + H_2O \Leftarrow H_2CO_3^- \Leftarrow H^+ \text{ and } HCO_3^-.}$$

Etiology

Respiratory alkalosis is a common acid–base abnormality observed in critically ill patients. It is also common in patients with hyperventilation due to anxiety. Box 8-4 lists common causes of respiratory alkalosis.

Pathophysiology

Hyperventilation, with a subsequent reduction in CO_2 and H^+ ion levels, causes respiratory alkalosis. Many of the signs and symptoms present in persons with respiratory alkalosis relate to the ion disturbances that may develop with hypocapnia, such as hypocalcemia and hypokalemia.

BOX 8-4. Causes of Respiratory Alkalosis

PULMONARY
- Pneumonia
- Pulmonary edema
- Pulmonary embolus
- Asthma
- Lung disease with shortness of breath (asthma, pneumonia, acute respiratory distress syndrome [ARDS], fibrosis, pulmonary embolism)
- Hypoxia with hyperventilation
- Overventilation by mechanical ventilation

NONPULMONARY
- Anxiety
- Pain
- Liver disease
- Fever/infection/sepsis
- Central nervous system disorders (tumors, cerebrovascular accidents)
- Salicylate intoxication
- Alcohol intoxication

Clinical Presentation

In respiratory alkalosis, tingling of extremities (paresthesia), muscle cramps, tetany, dizziness and/or syncope, confusion, anxiety, seizures, and coma may occur. Cardiac symptoms include palpitations, dysrhythmias, and hypotension. Many patients with hyperventilation due to anxiety feel as though they are enduring a cardiac problem.

Physical Assessment Findings

Many patients enduring hyperventilation appear anxious and are frequently tachycardic. With acute hyperventilation, obvious chest wall movements and use of intercostal muscles to breathe are visible. Hypocalcemia may elicit muscle spasms, as well as Chvostek's and Trousseau's signs. Underlying pulmonary disease may be present with signs such as crackles and rhonchi. If the patient is hypoxic, cyanosis may be apparent. The patient may have focal neurological signs or a depressed level of consciousness. Cardiac rhythm disturbances often occur.

Compensatory Mechanisms and Values

Because the lungs cannot adequately compensate for the acid–base disturbance, as indicated by the low CO_2, the kidneys carry out the majority of compensation by reabsorbing H^+ into the bloodstream and excreting HCO_3^-. The compensation can take hours to days to accomplish, so medical intervention is needed. In *uncompensated respiratory alkalosis,* blood pH is above

7.45 with a CO_2 level lower than 35 mm Hg. If the kidneys compensate successfully through absorption of H^+ and excretion of HCO_{3^-} ions, ABG values reveal a pH that is normalizing or decreasing toward normal and CO_2 less than 35 mm Hg.

Treatment

Treatment of respiratory alkalosis lies in identifying the underlying trigger that has produced hyperventilation. Pain management or sedation may be required to slow and control the respiratory rate. One common treatment for respiratory alkalosis involves patients breathing into a paper bag. This allows for rebreathing of exhaled CO_2 to bring CO_2 levels back up to a normal range. A CO_2 rebreather, available in the clinical setting, is a type of breathing apparatus that recycles the exhaled CO_2 and adds O_2 to compensate for the oxygen consumed by the user.

CLINICAL CONCEPT

Hyperventilation is the most common cause of respiratory alkalosis. Simply rebreathing into a paper bag can replace lost CO_2.

Metabolic Acidosis

Metabolic acidosis is due to an excess of acid not related to CO_2. The primary findings are a pH below 7.35 but with normal or lower-than-normal CO_2 levels, indicating that CO_2 is not driving the reduction in pH. One of the primary causes of metabolic acidosis is a metabolic condition that leads to acidic end products, such as ketones or lactic acid. Alternatively, excessive bicarbonate loss due to kidney or GI tract disorders can reduce pH levels.

Metabolic acidosis is mainly divided into processes associated with a normal (8 to 16 mEq/L) or an elevated AG (greater than 16 mEq/L) (see the section on the AG). Addition of acids, such as ketones, increases the anion gap, whereas loss of a base, such as bicarbonate, does not alter it.

Epidemiology

Morbidity and mortality in metabolic acidosis are dependent on the underlying condition. If severe forms of metabolic acidosis go untreated, death may result.

Etiology

Box 8-5 contains a list of common causes of metabolic acidosis.

Pathophysiology

Metabolic acidosis is a condition characterized by an arterial pH lower than 7.35 in the absence of an elevated PCO_2. Three primary mechanisms may lead to the condition of metabolic acidosis:

1. Increased level of acids in the bloodstream
2. Decreased excretion of acids
3. Loss of base from the bloodstream

Increased production of acids that occurs under certain metabolic conditions may lead to metabolic acidosis. Diabetic ketoacidosis (DKA) is one of the most common causes of metabolic acidosis. In DKA an accumulation of keto-acids leads to widespread metabolic acidosis. In another example, when widespread ischemia is present, cells with the capacity to rely on anaerobic metabolism produce lactic acid. The accumulation of lactic acid leads to the development of lactic acidosis, which is a type of metabolic acidosis. Alternatively, toxic ingestion or medication overdoses with acidic substances can cause metabolic acidosis. An example is aspirin toxicity, which causes ASA accumulation in the bloodstream. Metabolic acidosis may also develop when there is a reduction in the ability of the kidneys to excrete H^+ or to reabsorb HCO_{3^-}. For example, prolonged diarrhea, in which intestinal contents, including bicarbonate, are lost, can also result in metabolic acidosis.

As previously discussed, acid–base imbalances alter electrolyte levels. In metabolic acidosis, the kidneys excrete H^+ in lieu of K^+. Thus K^+ accumulates in the bloodstream. Hyperkalemia develops and potentially disrupts the functioning of the heart. Arrhythmias, peaked T waves, QRS widening, and ventricular fibrillation may manifest. Tachycardia is the most common cardiovascular effect seen with mild metabolic acidosis, as hypotension from decreased contractility of the heart may develop. Serum calcium levels elevate in metabolic acidosis due to reduced binding of Ca^{++} to albumin. Hypercalcemia causes muscle weakness, confusion, and lethargy.

Clinical Presentation

The signs and symptoms of metabolic acidosis are widespread and are often due to abnormal serum

BOX 8-5. Causes of Metabolic Acidosis

INCREASED NONCARBONIC ACIDS

- Diabetic ketoacidosis
- Lactic acidosis
- Alcoholic ketoacidosis
- Uremic acidosis
- Ingestion of toxic substances (antifreeze, aspirin)
- Intestinal, biliary, or pancreatic fistulas
- Hypocalcemia, hypokalemia, or hypomagnesemia

BICARBONATE LOSS

- Prolonged diarrhea
- Renal tubular acidosis
- Interstitial renal disease
- Ureterosigmoid loop
- Ingestion of acetazolamide or ammonium chloride

potassium and calcium levels. The patient complains of respiratory distress as the lungs attempt to compensate for the acidosis. Neurological symptoms include headache, drowsiness, confusion, seizures, neuromuscular fatigue, twitching, and coma. GI symptoms such as nausea, vomiting, and anorexia are common. Cardiovascular symptoms present as hypotension, dysrhythmias, and decreased cardiac contractility.

Physical Assessment Findings

The patient is tachypneic and in respiratory distress. Cardiovascular signs may include weak pulses, tachycardia, hypotension, and arrhythmia. The patient may have GI pain and vomiting. Excessive vomiting can lead to dehydration. Signs of dehydration may include tachycardia, dry mucous membranes, and delayed capillary refill. Patients with DKA may present with fruity odor to their breath due to ketone production. Metabolic acidosis can also cause confusion, lethargy, and possibly coma or seizures.

Compensatory Mechanisms and Values

Unlike respiratory acidosis, in which the kidneys attempt to excrete excess acids, in metabolic acidosis, the lungs, along with the kidneys, attempt to compensate. In metabolic acidosis, the high H^+ ion levels stimulate chemoreceptors, which in turn stimulate the respiratory center to increase the respiratory rate. Elimination of CO_2 pulls H^+ ions out of the bloodstream, increasing the blood pH. This is evident in the carbonic-bicarbonate equation as it moves to the left:

$$\mathbf{CO_2 + H_2O \leftarrow H_2CO_3 \leftarrow H + and\ HCO_3^-}$$

Deep, rapid breathing due to metabolic acidosis is referred to as Kussmaul's breathing. Kussmaul's breathing is particularly common in DKA. The lungs ventilate rapidly to attempt to rid the body of CO_2 and, consequently, decrease H^+ levels. In addition, to compensate for metabolic acidosis, the kidneys, if healthy, reabsorb HCO_3^- and excrete H^+. Compensatory mechanisms require hours to days to remedy the condition, often necessitating medical intervention. The hallmark of *uncompensated metabolic acidosis* is pH less than 7.35 with normal to low CO_2 levels, indicating CO_2 is not the reason for the acidic pH. HCO_3^- values will also be lower than normal (<22 mEq/L).

A higher-than-normal AG is due to excess acids, as may occur in DKA or ingestion of acidic compounds. A normal AG indicates the reduced pH is due to loss of basic compounds, such as bicarbonate.

In *compensated metabolic acidosis,* pH is normal or rising toward normal. PCO_2 is lower than normal (<35 mm Hg) as CO_2 is exhaled to compensate for the acidic pH. The kidneys reabsorb more HCO_3^- and excrete H^+ ions to compensate.

Treatment

All types of metabolic acidosis require treating the underlying cause. For example, if the cause is DKA, insulin is needed. If the metabolic acidosis is caused by kidney failure, in which the kidneys cannot effectively remove H^+ ions from the blood, hemodialysis is required. Correcting the underlying disorders and restoring electrolyte and fluid balance are critical. IV sodium bicarbonate may be utilized in severe cases of metabolic acidosis when pH is lower than 7.20. Caution is needed, as excessive use of sodium bicarbonate may produce a rebound metabolic alkalosis.

Metabolic Alkalosis

Metabolic alkalosis is a blood pH greater than 7.45 with a normal or higher-than-normal CO_2 level. It is caused by excessive loss of acids unrelated to CO_2 or an increase in bicarbonate levels, such as with retention of sodium bicarbonate. Loss of acids can take many forms, including intracellular shift of H^+ ions from the plasma, as occurs with hypokalemia, or loss of H^+ through the GI tract, as occurs with severe vomiting. The use of certain diuretics can result in the loss of H^+ ions by the kidneys, resulting in alkalosis. Administration of excess sodium bicarbonate and volume depletion are the primary reasons for bicarbonate excess, which also results in metabolic acidosis.

Epidemiology

Metabolic alkalosis is an acid–base disturbance that commonly occurs in hospitalized patients. The more elevated the pH beyond the normal range, the higher the morality rate.

Etiology

Box 8-6 lists common causes of metabolic alkalosis.

Pathophysiology

The most common cause of metabolic alkalosis is depletion of H^+ ions. Loss of H^+ ions occurs primarily through the kidneys and GI tract. Gastric secretions contain large amounts of hydrochloric acid (HCl). Any process that depletes gastric fluid, such as severe vomiting or GI tract suctioning, can result in metabolic alkalosis. Development of metabolic alkalosis due to gastric loss of H^+ ions is a concern for individuals suffering from bulimia, who frequently induce vomiting.

BOX 8-6. Causes of Metabolic Alkalosis

- Bicarbonate ingestion
- Excess IV sodium bicarbonate
- Potassium-wasting diuretics
- Loss of gastric fluids from vomiting, gastric suctioning, diarrhea, or binge–purge syndrome
- Cushing's syndrome
- Primary hyperaldosteronism
- Secondary hyperaldosteronism

The kidneys may contribute to metabolic alkalosis if they are unable to retain adequate H^+ or excrete HCO_3^- at the necessary level.

As observed with the other acid–base disturbances, electrolyte imbalances may play a role in pH abnormalities and vice versa. Metabolic alkalosis may develop in response to hypokalemia. Hypokalemia may develop with the use of certain diuretics or in Cushing's syndrome, in which elevated amounts of aldosterone increase K^+ ion excretion by the kidneys. With the loss of potassium ions, H^+ ions shift into the intracellular space, depleting H^+ ion levels in the bloodstream. Metabolic alkalosis also commonly occurs in cardiac resuscitation. Administration of large amounts of sodium bicarbonate are needed to neutralize the lactic acidosis that forms in cardiac arrest. Excessive amounts of sodium bicarbonate in the bloodstream can exceed the capacity of the kidneys to excrete the bicarbonate. Finally, metabolic alkalosis can lead to hypokalemia and hypocalcemia. These electrolyte disturbances account for some of the signs and symptoms associated with metabolic alkalosis.

Clinical Presentation

The symptoms of metabolic alkalosis are widespread, affecting the neurological, cardiovascular, GI, and musculoskeletal systems primarily through alteration in ion levels. Patients may present with confusion, dizziness, agitation, weakness, vomiting, diarrhea, and possibly seizures.

Physical Assessment Findings

The physical signs of metabolic alkalosis are nonspecific and multisystemic. Hypokalemia due to metabolic alkalosis can cause muscular weakness, myalgia, muscle spasms, and cardiac arrhythmias. Hypocalcemia may also develop and present as tetany, Chvostek's sign, and Trousseau's sign. Fluid volume status can also change. Evaluation of this status includes assessment of orthostatic changes in blood pressure and heart rate, mucous membranes, presence or absence of edema, skin turgor, weight change, and urine output. In patients with metabolic alkalosis who are suffering from bulimia, erosion of the teeth enamel and dental caries may be present.

Compensatory Mechanisms and Values

Similar to metabolic acidosis, both the lungs and kidneys attempt to compensate in states of metabolic alkalosis. Chemoreceptors detect the higher-than-normal pH of the blood and induce a reduction in ventilation. By slowing the breathing rate, the lungs retain CO_2, thereby raising H^+ content of the blood and lowering pH. The kidneys compensate by reabsorbing H^+ into the bloodstream and excreting HCO_3^-. This can take days to reach the point of adequate compensation; therefore medical intervention is necessary. In *uncompensated metabolic alkalosis* pH is greater than 7.45 with normal-to-high CO_2 levels, indicating CO_2 is not causing the elevation in pH. Elevated HCO_3^- values are present (>26 mEq/L). pH will be normal or reducing toward normal in *compensated metabolic alkalosis,* with an elevated PCO_2 (>45 mm Hg). CO_2 retention by slow ventilation of the lungs helps reduce pH levels.

Treatment

Treatment of metabolic alkalosis includes electrolyte and fluid replacement. Potassium-sparing diuretics may be administered if the cause of alkalosis is diuretic use. Acetazolamide, which reduces HCO_3^- reabsorption in the kidneys, may also be used to treat conditions of moderate to severe metabolic or respiratory alkalosis.

Mixed Disorders

Clinicians must also be aware that more than one type of acid–base disturbance can be present at any given time. These mixed acid–base disorders manifest when more than one underlying condition disrupts pH. Analysis of ABG values, AG, and patient presentation are critical in determining the course of the acid–base disturbance and the underlying causes of the coexisting disturbances.

Chapter Summary

- An acid is defined as any compound that donates hydrogen ions (H^+) in solution.
- A base is a compound that accepts H^+ ions in solution.
- When H^+ ions predominate in a solution, the solution is acidic. When basic ions predominate in a solution, the solution is alkaline.
- Buffers resist changes in pH by donating or accepting H^+ ions as needed.
- Three main buffer systems that exist in the body are protein, phosphate, and carbonic acid–bicarbonate system.
- The lungs and the kidneys regulate the body's acid–base balance through use of the carbonic acid–bicarbonate buffer system.
- Blood pH, partial pressure of oxygen (P_{O_2}), partial pressure of carbon dioxide (P_{CO_2}), and bicarbonate ion concentration (HCO_3^-) are the values indicated by an ABG.

- The normal pH of blood is 7.35 to 7.45. A pH level lower than 7.35 is acidemia (also called acidosis); a level greater than 7.45 is alkalemia (also called alkalosis).
- A chemical buffering system used by the body is the carbonic acid–bicarbonate system: $CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+$ and HCO_3^-. Both the lungs and kidneys utilize this system to compensate for acid–base disturbances.
- During hypoventilation the lungs retain CO_2; during hyperventilation, the lungs blow off CO_2.
- The greater the amount of CO_2 in the body, the greater the formation of H^+ ions.
- Four possible acid–base disturbances occur in the body: respiratory acidosis, respiratory alkalosis, metabolic acidosis, and metabolic alkalosis.
- Hypoventilation, which causes P_{CO_2} greater than 45 mm Hg, results in respiratory acidosis; hyperventilation, which causes P_{CO_2} less than 35 mm Hg, results in respiratory alkalosis.
- An excess of acid or a loss of HCO_3^- in the blood causes metabolic acidosis. In uncompensated metabolic acidosis, HCO_3^- will be lower than 22 mEq/L and the pH will be lower than 7.35.
- Metabolic alkalosis is caused by excessive loss of acids, shift of H^+ ions into the intracellular space, or increase in bicarbonate in the bloodstream. In uncompensated forms, the blood pH will be greater than 7.45 and the bicarbonate concentration greater than 26 mEq/L.
- Acid–base imbalances affect serum electrolyte levels. Acidosis is generally associated with hyperkalemia, and alkalosis with hypokalemia. Due to altered albumin binding affinity for Ca^{++} with pH changes, acidosis may cause hypercalcemia and alkalosis may cause hypocalcemia.

Making the Connections

Disorder and Pathophysiology

Signs and Symptoms	Physical Assessment Findings	Diagnostic Testing	Treatment
Respiratory Acidosis | Lungs are not ventilating; retaining too much CO_2, creating too much H^+. Commonly due to COPD, severe asthma, or any cause of reduced ventilation.			
Dyspnea. Respiratory distress. Patient may be lethargic, stuporous, or comatose.	Diminished respiratory rate. Cyanosis. Clubbing if chronic hypoxia.	Uncompensated: blood pH less than 7.35. P_{CO_2} greater than 45 mm Hg. P_{O_2}: low. Urine: acidic.	Treat the lung disorder for better ventilation. Bronchodilation. Antibiotics if pneumonia. Intubation and mechanical ventilation if needed.
Respiratory Alkalosis | Lungs are hyperventilating; losing too much CO_2 creates too little H^+ in the blood. Commonly due to hyperventilation secondary to anxiety or shallow respirations in asthma.			
Hyperventilation. Anxiety. Palpitations. Paresthesia. Patient may have pain.	High respiratory rate. Tachycardia.	Uncompensated: blood pH greater than 7.45. P_{CO_2} less than 35 mm Hg. Urine: basic.	Slow the breathing rate; CO_2 rebreather. Patient may need sedative.
Metabolic Acidosis | Excessive acid in the bloodstream (e.g., ketoacids or lactic acid) or excessive loss of HCO_3^- (e.g., GI tract loss). Commonly due to DKA, lactic acidosis, drug toxicity, or GI loss of excessive HCO_3^- as in diarrheal illness.			
Symptoms according to etiology of disorder: respiratory distress, headache, drowsiness, confusion, seizures, fatigue. GI symptoms of nausea, vomiting, and anorexia are common.	Tachycardia. Hypotension, weak pulses. Dehydration signs may be present: dry mucous membranes, poor skin turgor, and delayed capillary refill. Patients with DKA may present with fruity odor to their breath. Metabolic acidosis can also cause confusion, lethargy, and possibly coma or seizures.	Uncompensated: blood pH less than 7.35. P_{CO_2} normal or slightly low. Serum K^+: high. Urine: acidic. Electrocardiogram (ECG) changes caused by hyperkalemia: arrhythmias, peaked T waves, QRS widening, and ventricular fibrillation possible.	Sodium bicarbonate IV. Treat etiologic disorder (for example, if DKA, treat diabetes).

Continued

Making the Connections–cont'd

Signs and Symptoms	Physical Assessment Findings	Diagnostic Testing	Treatment
Metabolic Alkalosis | Excessive base in the bloodstream (such as toxic ingestion) or lack of sufficient acid in the bloodstream caused by high loss of H^+ (such as loss of HCl with excessive vomiting).			
Symptoms according to etiology of disorder; often related to decreased calcium ionization resulting from low H^+ level. Low Ca^{++} levels cause tetany, irritability, disorientation, and seizures. Prolonged vomiting may be the cause.	Chvostek's sign. Trousseau's sign. Hypotension or hypertension may be present. Patients with bulimia often have erosions of teeth enamel and dental caries.	Uncompensated: blood pH greater than 7.45. P_{CO_2} normal or slightly high. Urine: basic. Serum ionized Ca^{++} low. ECG may show dysrhythmias.	IV acetazolamide.

Bibliography

Barrett, K. E., Barman, S. M., Boitano, S., & Brooks, H. L. (2015). *Ganong's review of medical physiology* (25th ed.). New York, NY: McGraw-Hill.

Hamilton, P. K. et al. (2017). Understanding acid-base disorders. *The Ulster Medical Journal*, *86*(3), 161–166.

Hamm, L. L., Nakhoul, N., & Hering-Smith, K. S. (2015). Acid-base homeostasis. *Clinical Journal of the American Society of Nephrology*, *10*(12), 2232–2242.

Harwani, S., Baumhover, N., Berry, C., Denison, D., Langraff, T., & Mathis, D. (2009). *CCRN certification for adult, pediatric and neonatal critical care nurses.* New York, NY: Kaplan Publishing.

Jones, M. B. (2010). Basic interpretation of metabolic acidosis. *Critical Care Nurse*, *30*(5), 63–69.

Mæhle, K. et al. (2014). Metabolic alkalosis is the most common acid–base disorder in ICU patients. *Critical Care, 18*(2), 420.

McPhee, S. J., & Papdakis, M. A. (2018). *Current medical diagnosis and treatment*. New York, NY: Lange/McGraw-Hill.

Myall, K., Sidney, J., & Marsh, A. (2011). Mind the gap! An unusual metabolic acidosis. *Lancet*, *377*(9764), 526.

Palmer, B. (2012). Evaluation and treatment of respiratory alkalosis. *American Journal of Kidney Diseases*, *60*(5), 834–838.

Schiavo, A., Renis, M., Polverino, M., Iannuzzi, A., & Polverino, F. (2016). Acid–base balance, serum electrolytes and need for non-invasive ventilation in patients with hypercapnic acute exacerbation of chronic obstructive pulmonary disease admitted to an internal medicine ward. *Multidisciplinary Respiratory Medicine*, *11*, 23.

Davis Advantage for Pathophysiology Topics

TRADITIONAL VIEW

DAVIS ADVANTAGE PATHOPHYSIOLOGY

Cellular Processes and Stress Response
Cell Injury and Adaptations
Stress Response
Genetic Basis of Disease
Pain – NEW!

Fluid, Electrolyte, and Acid-Base Homeostasis
Fluid and Fluid Imbalances
Electrolyte Imbalances
Acid-Base Imbalances

Inflammation and Infection
Inflammation and Wound Healing
Infectious Diseases
Hypersensitivities and Autoimmune Disorders
Immunodeficiency and HIV

Hematologic Disorders
Disorders of White Blood Cells
Disorders of Red Blood Cells
Disorders of Platelets, Hemostasis, and Coagulation

Arterial and Ischemic Heart Disease and Conduction Disorders
Hyperlipidemia and Hypertension
Atherosclerosis and Other Arterial Diseases
Ischemic Heart Disease
Conduction Disorders and Other MI Complications

Heart Failure and Valvular Disease
Cardiac Function and Assessment
Left and Right Heart Failure
Heart Valve Disease

Pulmonary Disorders
Upper and Lower Respiratory Tract Disorders
Obstructive Pulmonary Disorders
Restrictive and Vascular Pulmonary Disorders

Renal and Urologic Disorders
Basic Pathologies of Renal Disorders
Renal Disorders
Urologic Disorders

Endocrine Disorders
Basic Pathologies of Endocrine Disorders
Pituitary and Thyroid Disorders
Parathyroid and Adrenal Disorders

Diabetes Mellitus and Metabolic Syndrome
Types of Diabetes and Diagnosis
Short-term Complications of Diabetes
Long-term Complications of Diabetes

Gastrointestinal Disorders
Disorders of Esophagus, Stomach, and Small Intestine
Disorders of the Large Intestine
Disorders of the Liver
Disorders of Gallbladder, Pancreas, Bile Duct

Neurological Disorders
Cerebrovascular Disorders
Chronic and Neurodegenerative Disorders
Brain and Spinal Cord Injury

Musculoskeletal Disorders
Musculoskeletal Trauma and Complications
Degenerative Musculoskeletal Disorders
Musculoskeletal Infection and Inflammation

Integumentary and Sensory Disorders – NEW!
Skin Disorders – NEW!
Burns – NEW!
Eye and Ear Disorders – NEW!

Reproductive Disorders – NEW!
Disorders of the Female Reproductive System and Breasts – NEW!
Disorders of the Male Reproductive System – NEW!

Cancer and Multiple Organ Failure and Death
Cancer
SIRS and Organ Dysfunction
Shock

Davis Advantage for Pathophysiology Topics

CONCEPT VIEW

DAVIS ADVANTAGE PATHOPHYSIOLOGY

Cellular Regulation
Cell Injury and Adaptations
Stress Response
Genetic Basis of Disease
Cancer

Comfort – NEW!
Pain – NEW!

Digestion
Disorders of Esophagus, Stomach, and Small Intestine
Disorders of the Large Intestine
Disorders of the Liver
Disorders of Gallbladder, Pancreas, Bile Duct

Fluid and Electrolyte Imbalance
Fluid and Fluid Imbalances
Electrolyte Imbalances

Female Reproduction – NEW!
Disorders of the Female Reproductive System and Breasts – NEW!

Hematologic Regulation
Disorders of White Blood Cells
Disorders of Red Blood Cells
Disorders of Platelets, Hemostasis, and Coagulation

Immunity
Hypersensitivities and Autoimmune Disorders
Immunodeficiency and HIV

Infection
Infectious Diseases

Inflammation
Inflammation and Wound Healing
SIRS and Organ Dysfunction

Male Reproduction – NEW!
Disorders of the Male Reproductive System – NEW!

Metabolism
Basic Pathologies of Endocrine Disorders
Pituitary and Thyroid Disorders
Parathyroid and Adrenal Disorders
Types of Diabetes and Diagnosis
Short-term Complications of Diabetes
Long-term Complications of Diabetes

Mobility
Musculoskeletal Trauma and Complications
Degenerative Musculoskeletal Disorders
Musculoskeletal Infection and Inflammation

Neurological Disorders
Cerebrovascular Disorders
Chronic and Neurodegenerative Disorders
Brain and Spinal Cord Injury

Oxygenation
Upper and Lower Respiratory Tract Disorders
Obstructive Pulmonary Disorders
Restrictive and Vascular Pulmonary Disorders

Perfusion
Hyperlipidemia and Hypertension
Atherosclerosis and Other Arterial Diseases
Ischemic Heart Disease
Conduction Disorders and Other MI Complications
Cardiac Function and Assessment
Left and Right Heart Failure
Heart Valve Disease
Shock

pH Regulation
Acid-Base Imbalances

Sensory Perception – NEW!
Eye and Ear Disorders – NEW!

Skin Integrity – NEW!
Skin Disorders – NEW!
Burns – NEW!

Urinary Elimination
Basic Pathologies of Renal Disorders
Renal Disorders
Urologic Disorders

PERSONALIZED TEACHING PLAN

TOPIC: Acid-Base Imbalances

Relationships: Acid-Base Disorders and Lab Values

DIRECTIONS

- Divide students into groups of 3. Have them work together to complete the following table.
- The first person of the group will indicate whether the pH of the disorder is acidic, alkaline, or normal. The next student will indicate if PCO_2 values are likely high, normal, or low, and the third student will indicate whether bicarbonate values are high, normal, or low.
- A student handout is provided and answers are shown below.

Disorder	pH	PCO_2	Bicarbonate
Uncompensated Respiratory Acidosis	**Low**	**High**	**Normal**
Uncompensated Respiratory Alkalosis	**High**	**Low**	**Normal**
Uncompensated Metabolic Acidosis	**Low**	**Normal**	**Low**
Uncompensated Metabolic Alkalosis	**High**	**Normal**	**High**
Compensated Respiratory Acidosis	**Normal or increasing towards normal**	**High**	**High (compensating for CO2 issue)**
Compensated Respiratory Alkalosis	**Normal or decreasing towards normal**	**Low**	**Low (compensating for CO2 issue)**
Compensated Metabolic Acidosis	**Normal or increasing towards normal**	**Low (compensating for low base)**	**Low**
Compensated Metabolic Alkalosis	**Normal or decreasing towards normal**	**High (compensating for high base)**	**High**

TIPS

This activity can be utilized in a number of ways, either in the group format suggested, as an entire class, or even as an individual student assignment. Remind students to follow the steps outlined at the end of the Acid-Base Imbalance chapter when completing this assignment.

GOALS

This activity stimulates discussion about acid-base disorders and helps students create a way to understand the laboratory values associated with these conditions.

ONLINE APPLICATION

- Assign the table as an individual assignment.
- Many approaches to acid-base imbalances have been developed. Create a discussion board and challenge students to find an on-line resource on how to learn acid-base disorders. Choose the top 3 and have the class vote on which one they like best.

Exchange of Ideas: Patient Scenario

DIRECTIONS

- Divide students into groups of 4. Assign each group a disorder from the list below. (Some groups will likely have the same topic.) Groups should not tell one another which disorder they have been assigned.
- Each group then creates a very brief patient case scenario that contains enough relevant information for others to identify the condition. Include patient data, clinical manifestations, and laboratory values as appropriate.
- Groups then exchange their patient scenario with other groups. Each group attempts to identify the other group's patient scenario disease.
- Disorders to assign:
    - Respiratory acidosis
    - Respiratory alkalosis
    - Metabolic acidosis
    - Metabolic alkalosis

TIPS

Emphasize to students that this activity is designed to help them see the "big picture." It is one thing to study and memorize laboratory values associated with acid-base disorders. It is another challenge to recognize and identify when and how a patient may present with these disorders.

GOALS

This activity helps students make the connection between laboratory values, pathophysiology, and clinical context.

ONLINE APPLICATION

- Divide students into groups and have them create a patient scenario as outlined above. Have each group post their scenario in a discussion board and have the other students in the class guess the condition presented.
- For an individual assignment, assign an acid-base disorder to each student and have each student create a patient scenario.

Case Study: Metabolic Acidosis

DIRECTIONS

- Divide students into groups of 3 to 4 students.
- Ask students to review and answer the case study questions found on the student handout.
- Answers are below.

Case Study

Daryl is an 18-year-old male who was diagnosed with type 1 diabetes 10 years ago. He is brought to the emergency department by his parents. Daryl seems anxious and confused. He is pale and hyperventilating. He has a racing heart rate and hypotension.

Blood glucose measurements indicate a serum glucose of 350 mg/dL. Daryl explains that he ate "some junk food" with friends and forgot to take his insulin.

Arterial blood gases are as follows:
pH = 7.25
PaO_2 = 100 mm Hg
$PaCO_2$ = 22 mm Hg

Serum values reveal hyperkalemia, reduced bicarbonate, and acetone.

1. What acid-base abnormality does Daryl display?

Metabolic acidosis as evidenced by the low pH and low CO_2.

2. Why is Daryl's PCO_2 value lower than normal?

In metabolic acidosis, the lungs compensate through hyperventilation to reduce CO_2 and, by extension, H+ ion levels.

3. Why is Daryl hyperventilating?

The acidic pH stimulates the chemoreceptors in the medulla oblongata to increase respiration as a compensation for the reduced pH.

4. Why is Daryl's serum bicarbonate reduced?

Bicarbonate is being utilized to buffer all the excess H+ ions.

5. Why is Daryl experiencing hyperkalemia?

In acidosis, particularly metabolic acidosis, elevations in H+ ions cause H+ ions to shift from the plasma into cells, which results in K+ shifting out of the cells into the plasma leading to hyperkalemia.

6. What is the significance of the presence of acetone in the serum sample?

The presence of acetone in the blood samples indicates Daryl is producing ketones, which are acidic.

7. What medical intervention may help Daryl's condition?

Daryl needs insulin to lower blood glucose back to normal and prevent further ketone formation. Because he has presented with hypotension, fluid replacement may also be necessary.

TIPS

Although students should take time to work on this case in small groups, bringing the entire class together to discuss and review answers will help make certain everyone has learned the concepts.

GOALS

This case helps students apply acid-base disorder knowledge to a "real world" scenario.

ONLINE APPLICATION

- Assign the case study as an individual assignment or as an assignment for groups to complete.
- Create a "daily check-in" by posting 1 to 2 questions of the case for a certain number of days. Require students to "check-in" and respond for 1 to 2 of the days.

RELATED QUIZZING ASSIGNMENTS

To help students explore the full depth of this topic, the following are available to assign:

- Pre-set Edge assignment (about 15 questions):
  Acid-Base Imbalances
- Quick or custom assignment from the full topic question bank:
  Acid-Base Imbalances

ADDITIONAL RESOURCES

PowerPoint slides for Chapter 8, *Acid-Base Imbalances*
Image Bank for Chapter 8, *Acid-Base Imbalances*
Test Bank for Chapter 8, *Acid-Base Imbalances*

Student Handout for *Relationships: Acid-Base Disorders and Lab Values*

DIRECTIONS: *After being divided into groups, work with your group to complete the following table. The first student indicates whether the pH of the disorder is acidic, alkaline, or normal. The next student indicates if PCO2 values are likely high, normal, or low, and the third student indicate whether bicarbonate values are high, normal, or low.*

Disorder	pH	PCO_2	Bicarbonate
Uncompensated Respiratory Acidosis			
Uncompensated Respiratory Alkalosis			
Uncompensated Metabolic Acidosis			
Uncompensated Metabolic Alkalosis			
Compensated Respiratory Acidosis			
Compensated Respiratory Alkalosis			
Compensated Metabolic Acidosis			
Compensated Metabolic Alkalosis			

Student Handout for Case Study: Metabolic Acidosis

DIRECTIONS: *After being divided into groups, read the following case study and answer each of the questions as directed by your instructor. Be prepared to discuss your answers with the class.*

Case Study

Daryl is an 18-year-old male, who was diagnosed with type 1 diabetes 10 years ago. He is brought to the emergency department by his parents. Daryl seems anxious and confused. He is pale and hyperventilating. He has a racing heart rate and hypotension.

Blood glucose measurements indicate a serum glucose of 350 mg/dL. Daryl explains that he ate "some junk food" with friends and forgot to take his insulin.

Arterial blood gases are as follows:

pH= 7.25
PaO_2= 100 mm Hg
$PaCO_2$ = 22 mm Hg

Serum values reveal hyperkalemia, reduced bicarbonate, and acetone.

Questions

1. What acid-base abnormality does Daryl display?

2. Why is Daryl's PCO_2 value lower than normal?

3. Why is Daryl hyperventilating?

4. Why is Daryl's serum bicarbonate reduced?

5. Why is Daryl experiencing hyperkalemia?

6. What is the significance of the presence of acetone in the serum sample?

7. What medical intervention may help Daryl's condition?