

Respiratory Concepts

The difference between a successful person and others is not a lack of strength, not a lack of knowledge, but rather a lack of will.

—Vince Lombardi

RESPIRATORY TEST BLUEPRINT

Respiratory 15% of total test

22 questions

- Acute pulmonary embolus
- ARDS
- Acute respiratory failure
- Acute respiratory infection (e.g., pneumonia)
- Aspiration
- Chronic conditions (e.g., COPD, asthma, bronchitis, emphysema)
- Failure to wean from mechanical ventilation
- Pleural space abnormalities (e.g., pneumothorax, hemothorax, empyema, pleural effusions)
- Pulmonary fibrosis
- Pulmonary hypertension
- Status asthmaticus
- Thoracic surgery

- Thoracic trauma (e.g., fractured rib, lung contusion, tracheal perforation)
- Transfusion-related acute lung injury (TRALI)^{*}

^{*}This topic is covered in the Hematology/Immunology Concepts chapter of this book.

RESPIRATORY TESTABLE NURSING ACTIONS

- Interpret blood gas results
- Recognize indications for and manage patients requiring:
 - Modes of mechanical ventilation
 - Noninvasive positive pressure ventilation (e.g., BiPAP, CPAP, high-flow nasal cannula)
 - Oxygen therapy delivery devices
 - Prevention of complications related to mechanical ventilation (ventilator bundle)
 - Prone positioning
 - Pulmonary therapeutic interventions related to mechanical ventilation: airway clearance, extubation, intubation, weaning
 - Therapeutic gases (e.g., oxygen, nitric oxide, heliox, CO₂)
 - Thoracentesis
 - Tracheostomy

***Note:** Although the test blueprint refers to these topics as respiratory concepts, the terms *respiratory* and *pulmonary* are

synonymous and are used interchangeably throughout this book.

Plan on spending approximately 22 hours studying respiratory concepts, since there will be 22 questions related to respiratory topics. The first half of this chapter reviews pulmonary physiological concepts that may appear on the exam. These concepts are the basis of the specific respiratory disorders and treatments that are covered in the second half of this chapter.

Pulmonary Assessment and Physiology

Ventilation

- Ventilation is the movement of air in (from the atmosphere) and out (from the body) to maintain appropriate concentrations of O₂ and CO₂.
- Central control (brain stem): primary control
 - Senses blood pH, decrease in pH → ventilation is stimulated
 - A decrease in pH = acidosis, which results in an increase in the rate and/or depth of breathing
- Peripheral control (PaO₂ “sensors” in aortic arch): secondary control
 - Senses PaO₂ of blood, decrease in PaO₂ → ventilation stimulated
 - Decrease in PaO₂ = hypoxemia, which results in an increase in the rate and/or depth of breathing
 - Chronic PaCO₂ retainers rely on mild hypoxemia for ventilator drive. If the PaO₂ is corrected to normal, this may result in a decreased drive to breathe (ventilate).
- What is the clinical indicator of ventilation? How do you know that your patient is ventilating normally?
 - You need to know the **PaCO₂** (NOT the PaO₂).
- What is minute ventilation?
 - Tidal volume (V_t) × respiratory rate (RR)—easily seen on the ventilator of a patient who requires mechanical ventilation
 - Normal ventilation is ~ 4 L/minute.
 - An increase in minute ventilation = an increase in **work of breathing**
- What is the primary muscle of ventilation?
 - Diaphragm
 - Anything that affects the “health” of the diaphragm (deconditioning, hypoxemia, acidosis, hypophosphatemia) will adversely affect ventilation.
- What is the position for optimal ventilation?
 - Upright sitting position

- Supine position is NOT good for ventilation; if a patient is in respiratory distress, the worst position for the patient is flat on his or her back!

Dead Space Ventilation

- Volume of air that does not participate in gas exchange
 - Anatomic dead space: ~ 2 mL/kg of V_t
 - We all have this; it is normal.
 - No gas exchange at level of nose down to alveoli
 - Alveolar dead space: pathologic, non-perfused alveoli, PE
 - Physiological dead space = anatomic dead space + alveolar dead space
- ☆ A **pulmonary embolus** results in increased alveolar dead space! A clot in the pulmonary circulation (a pulmonary embolus): no blood flow past alveoli in that area of the pulmonary circulation (Figure 4-1).

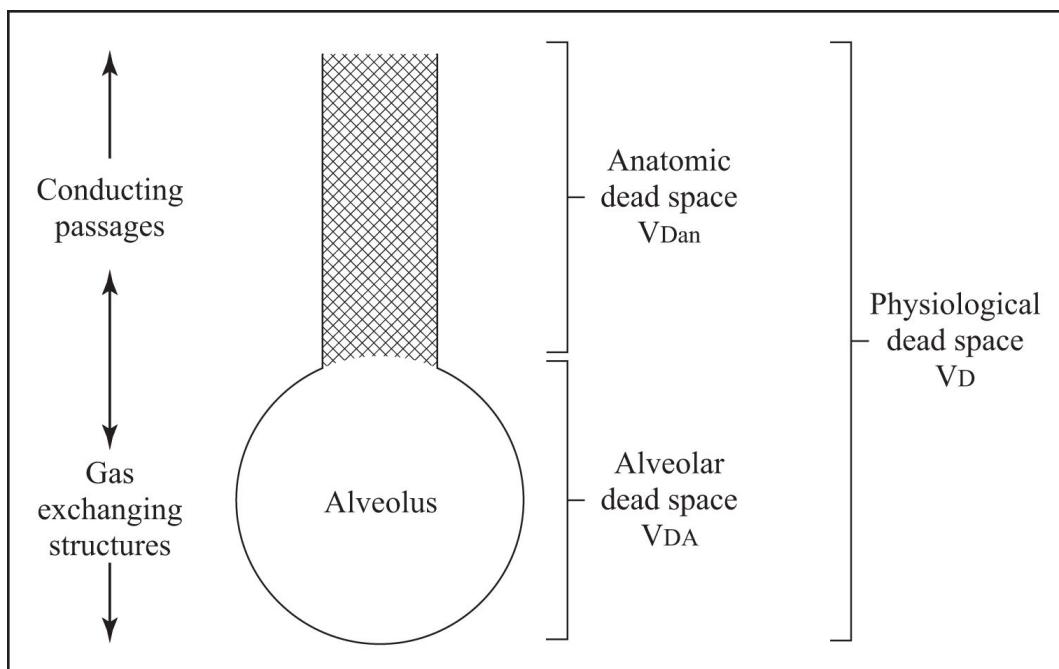


Figure 4-1. Dead space ventilation

Pulmonary Perfusion

The main function of the pulmonary system is gas exchange. For gas exchange to occur normally, there needs to be ventilation. However, movement of air alone is not enough for normal gas exchange. There needs to be perfusion, movement of blood past alveoli.

- Pulmonary perfusion is movement of blood through pulmonary capillaries.
- Any decrease in blood flow past alveoli (e.g., pulmonary embolus, low cardiac output states) will affect the ventilation/perfusion ratio and gas exchange.
- Normal ventilation/perfusion ratio:

$$\frac{4 \text{ L ventilation/min (V)}}{5 \text{ L perfusion/min (Q)}}$$

Ideal lung unit = 0.8 ratio, normal V/Q ratio

Any problem that alters ventilation (V) or perfusion (Q) can result in abnormal gas exchange if compensatory mechanisms are not successful. For example, even though it is not a pulmonary problem, a low cardiac output can result in poor gas exchange.

- You will not be expected to calculate V/Q ratios for the exam. However, you will need to know that the pulmonary problems (discussed in this chapter) will result in abnormal V/Q ratios, from mild to extreme, depending upon the extent of the problem.

EFFECT OF GRAVITY ON PULMONARY PERFUSION

- In the upright position, most pulmonary blood is in the lower lung lobes (see A in Figure 4-2). When lying supine, most pulmonary blood is posterior (see B in Figure 4-2). Rarely are ALL lung units perfused, but an example would be vigorous exercise (as in C in Figure 4-2).

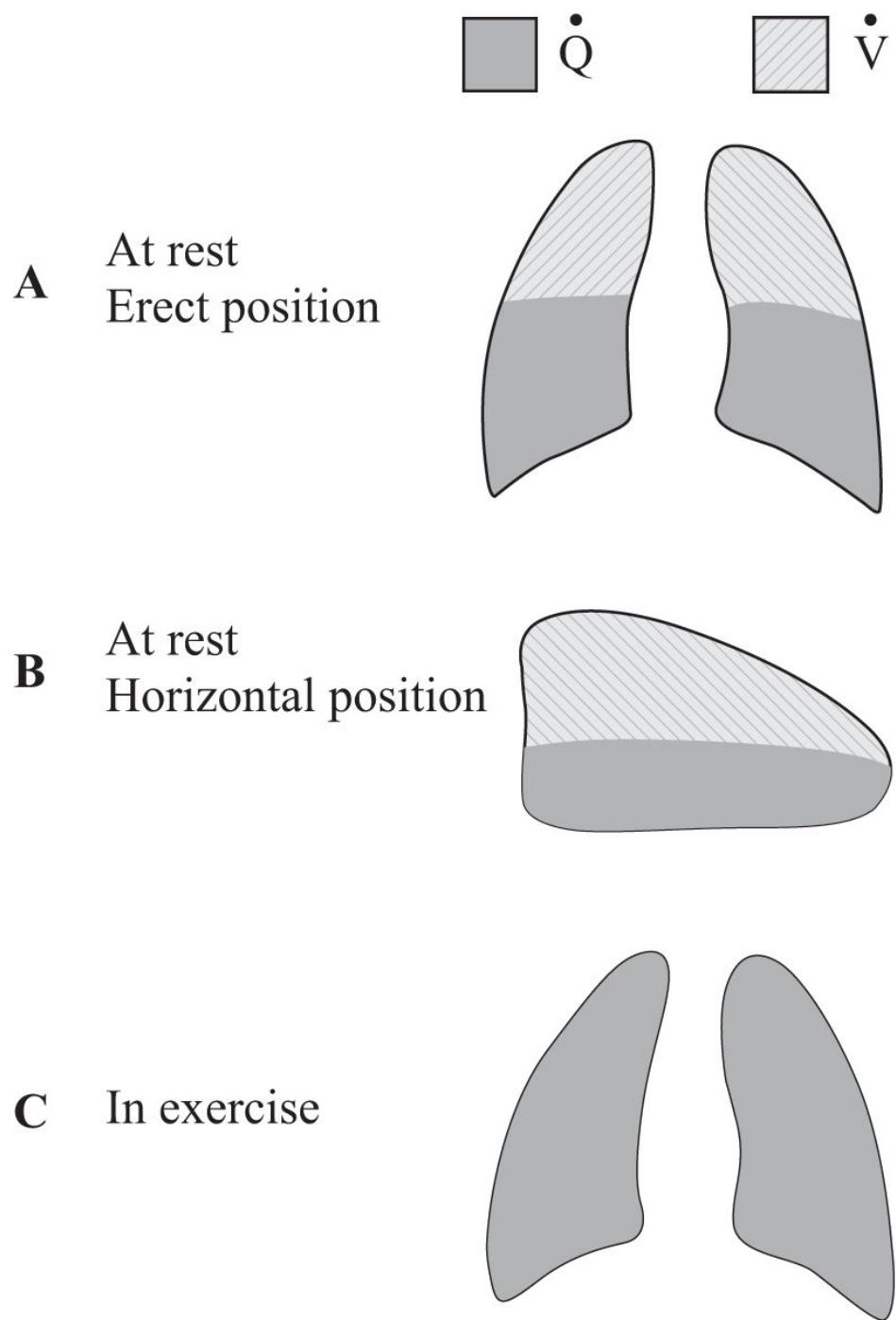


Figure 4-2. Perfused lung units

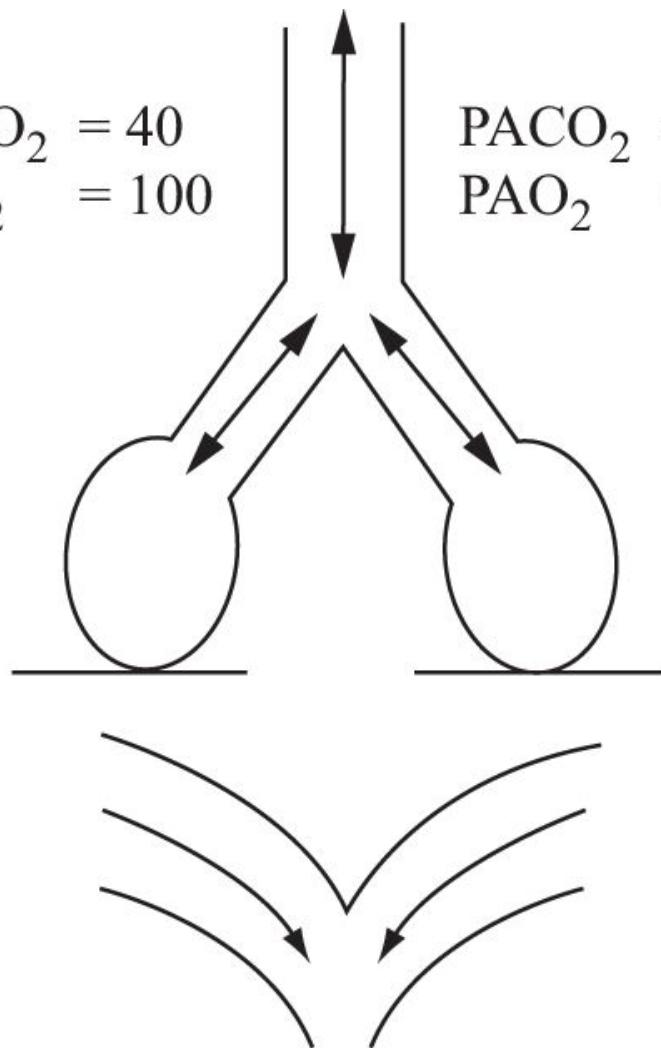
- What are the clinical implications?
- ☆ You want the “good” lung down.

- Large right lung pneumonia: if the patient is turned to the right (“bad” lung), more blood goes to the right, and the patient may become hypoxemic.
 - This patient should not be turned to the right side.
- ☆ Perfusion of under-perfused anterior chest alveoli explains the improved oxygenation seen during PRONE positioning for severe hypoxemia.

V/Q Ratio

☆ Normal V/Q ratio

- When there are no problems with either ventilation or perfusion, the patient will have normal gas exchange on room air (Figure 4-3).



Systemic artery

$\text{PaCO}_2 = 40$

$\text{PaO}_2 = 100$

$\text{SaO}_2 = 99\%$

Figure 4-3. Normal V/Q ratio, FiO_2
0.21

- Abnormal V/Q ratio

- When there is a problem with ventilation or perfusion, there is a V/Q mismatch.
- The patient will develop hypoxemia on room air. However, providing oxygen will generally correct the hypoxemia until the etiology can be determined and addressed (Figure 4-4).

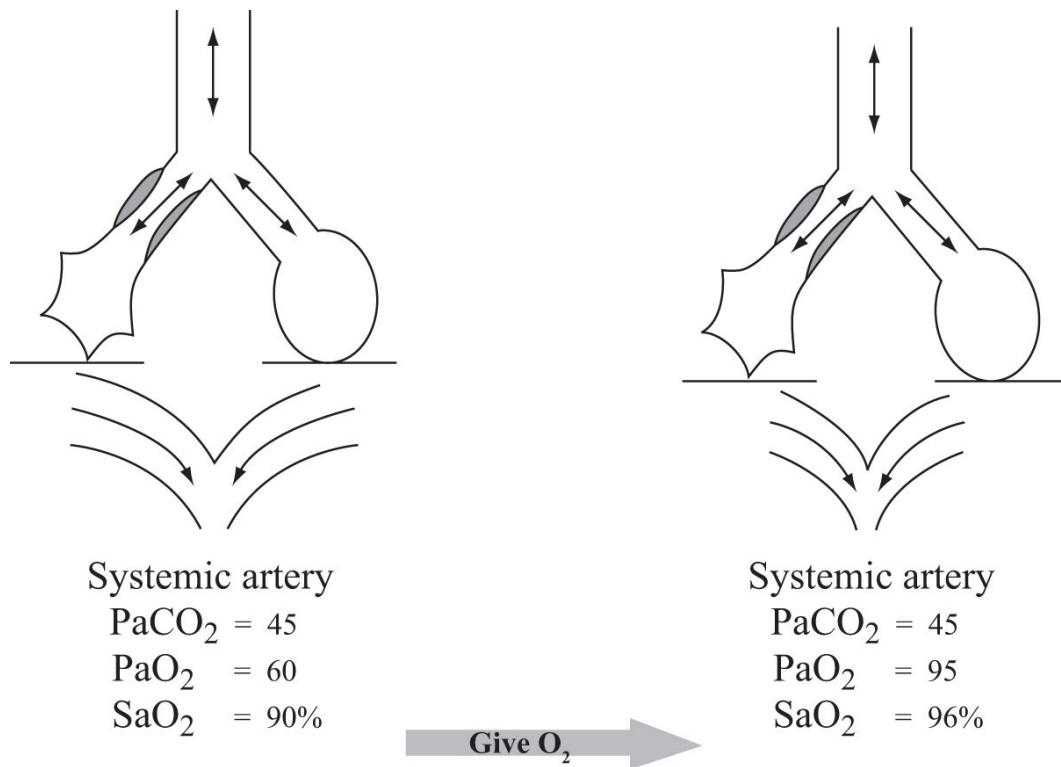


Figure 4-4. V/Q mismatch (i.e., pneumonia, pulmonary embolism)

- Treatment of V/Q mismatch
 - Give O₂
 - Identify and treat the underlying problem
- ★ Shunt
 - An extreme V/Q mismatch; even providing 100% FiO₂ will NOT correct the hypoxemia (Figure 4-5).

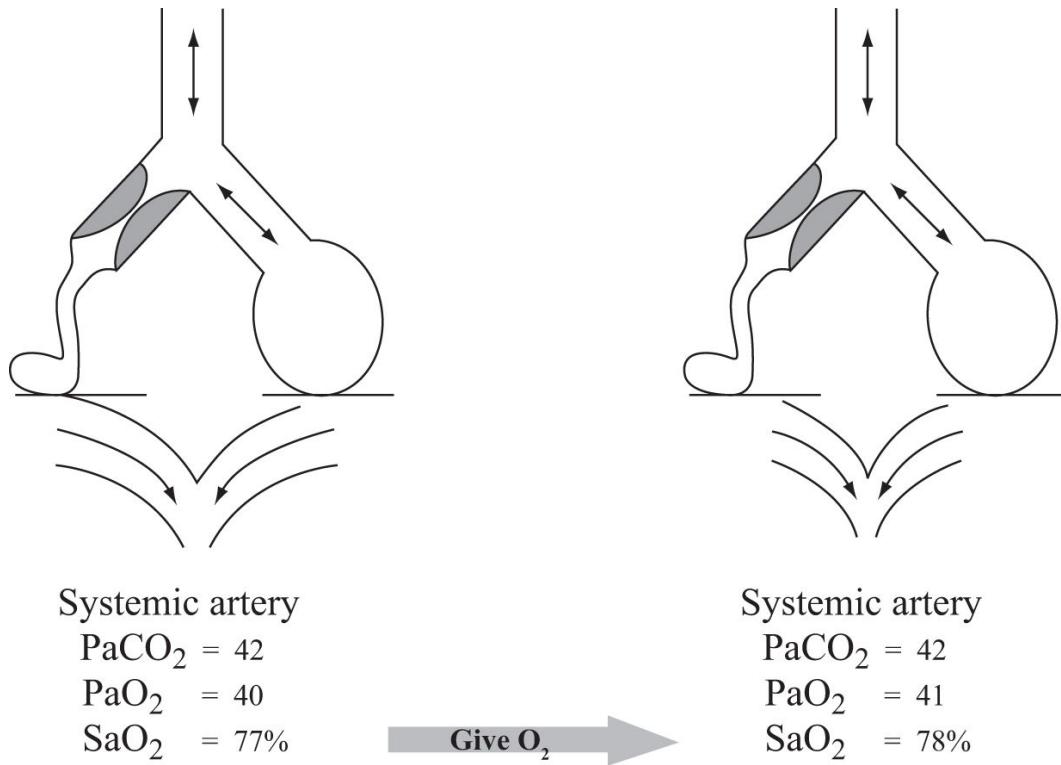


Figure 4-5. Shunt (i.e., acute respiratory distress syndrome (ARDS))

- A shunt is movement of blood from the right side of the heart to the left side of the heart without getting oxygenated; venous blood moves to the arterial side.
- Normal physiological shunt: Thebesian veins of the heart empty into the left atrium. This is why the normal oxygen saturation on room air is 95% to 99%; it cannot be 100% on room air due to this shunt. (With supplemental oxygen, 100% saturation can be achieved.)
- Anatomic shunt: two examples are a ventricular septal defect and an atrial septal defect.
- ☆ Pathologic shunt: **ARDS!** Blood goes through the lungs but does NOT get oxygenated, resulting in **refractory hypoxemia**.
 - Treatment of a shunt: administer **oxygen, AND**
 - **Positive end-expiratory pressure (PEEP)**
 - Prevents expiratory pressure from returning to zero; by keeping expiratory pressure **POSITIVE**, it . . .
 - ↓ Surface tension of the alveoli, preventing atelectasis
 - ↑ Alveolar recruitment

- ↑ Driving pressure, extends time of gas transfer, allows for a ↓ in FiO_2 (Figure 4-6)

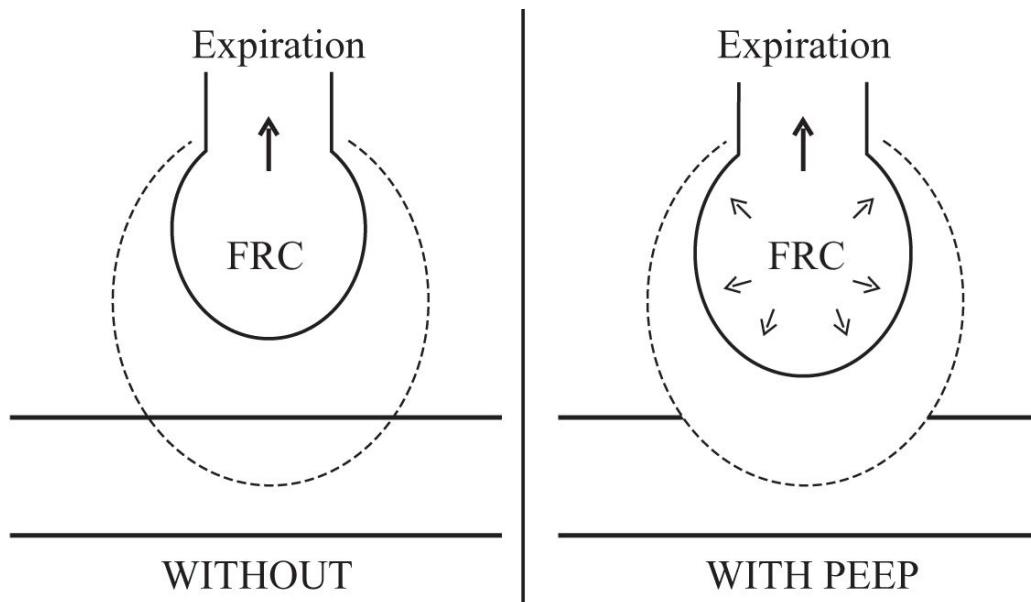


Figure 4-6. Visual representation of PEEP

- With the addition of PEEP to the airway (provided in cm water pressure, i.e., 10 cm, 15 cm), hypoxemia will be addressed and the FiO_2 may be decreased from 100%.

Assessment of Oxygenation in the Critically III

Adequate oxygenation is the delivery of O_2 to meet tissue demands at the **cellular** level. In order to achieve this, each of the following needs to occur:

- Adequate ventilation
- Transfer of O_2 across alveolar-capillary membrane
- Presence of hemoglobin to carry O_2
- Adequate cardiac output to deliver O_2 to the tissue bed
- Release of O_2 from the hemoglobin molecule
- Ability of cells to utilize O_2

At the cellular level, sufficient oxygen is needed for the production of adenosine triphosphate (ATP), which is needed for cell energy and life. See Figure 4-7 to see how oxygen is needed for aerobic metabolism, along with the production of sufficient ATP for cell life. Without sufficient oxygen at the cellular level, lactic acid is produced (LACTIC ACIDOSIS), which is the evidence of anaerobic metabolism, organ failure, and eventual cell death.

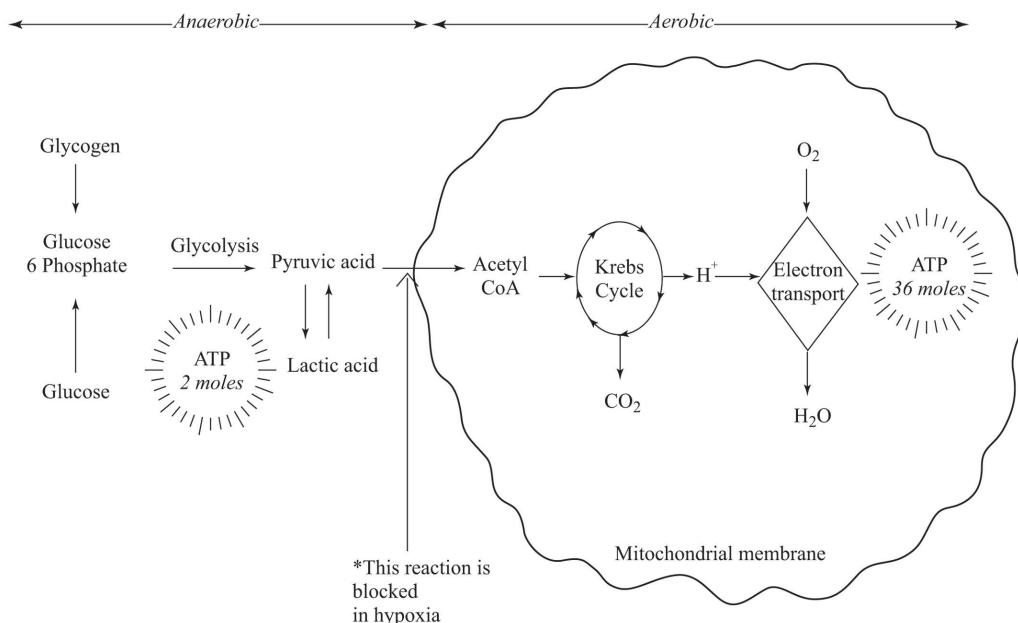


Figure 4-7. Importance of oxygen at the cellular level

It is NOT sufficient to examine only PaO_2 and SaO_2 ! For example, a patient with sepsis/septic shock may have a normal PaO_2 , SaO_2 , and hemoglobin; clear lungs; and adequate ventilation and oxygen delivery, yet a lactate level of 10. This is lactic acidosis. Why? Oxygen utilization is affected by sepsis/septic shock and results in anaerobic metabolism at the cellular level. Table 4-1 lists the clinical indicators of oxygenation in the critically ill.

Table 4-1. Indicators of Oxygenation

Parameter	Normal	How It's Calculated/Measured	Clinical Relevance
Arterial oxygen (PaO_2)	80-100 mmHg on room air	Directly measured	Less than 80 mmHg = hypoxemia (classified as mild, moderate, or severe)
Saturation of arterial oxygen (SaO_2)	95-99% on room air	Directly measured	Direct relationship with PaO_2 ; amount of hemoglobin combined with O_2
Mixed venous oxygen saturation (SvO_2)	60-75%	Direct measurement (pulmonary artery)	Most sensitive indicator of oxygenation at the cellular level
Oxygen content (CaO_2)	15-20 mL/100 mL blood	$\text{CaO}_2 = (\text{Hgb} \times 1.39 \times \text{SaO}_2) + (\text{PaO}_2 \times 0.003)$	Severe anemia may result in hypoxia
Oxygen delivery (DO_2)	900-1,100 mL/min	$\text{CaO}_2 \times \text{CO} \times 10$	Pump problems (heart) will decrease DO_2
Oxygen consumption, utilization (VO_2)	250-350 mL/min	$(\text{SaO}_2 - \text{SvO}_2) \times \text{Hgb} \times 13.9 \times \text{CO}$	Low with septic shock
Alveolar-arterial (A-a) gradient	< 10 mmHg	PAO_2 minus PaO_2 $(\% \text{FiO}_2 \times 715) - \text{PaCO}_2$ $(0.8 - \text{PaO}_2)$	Calculates the difference between the alveolar oxygen and the arterial oxygen Indicates whether the gas transfer is normal and, if not, how bad the V/Q mismatch or shunt is; just remember what normal is for the exam

★ For the exam:

► Do not memorize the formulas.

- Remember that the assessment of oxygenation is more than just examining the PaO_2 or SaO_2 .
- Consider the effects of severe anemia, low cardiac output, and an inability to utilize oxygen even when delivery is adequate (e.g., sepsis).

Most critically ill patients have continuous monitoring of oxygen saturation (SaO_2) that is noninvasively measured with the use of a bedside pulse oximetry (SpO_2). Although the normal SaO_2 on room air is 95% to 99%, the goal for most critically ill patients is to maintain the SpO_2 at 90% or greater, usually with supplemental oxygen. Note from the curve depicted in Figure 4-8 that when the SaO_2 is less than 90%, the PaO_2 is less than 60 mmHg. When the PaO_2 is less than 60 mmHg, cells begin to have difficulty maintaining aerobic metabolism (without compensation, i.e., an increase in heart rate or oxygen delivery).

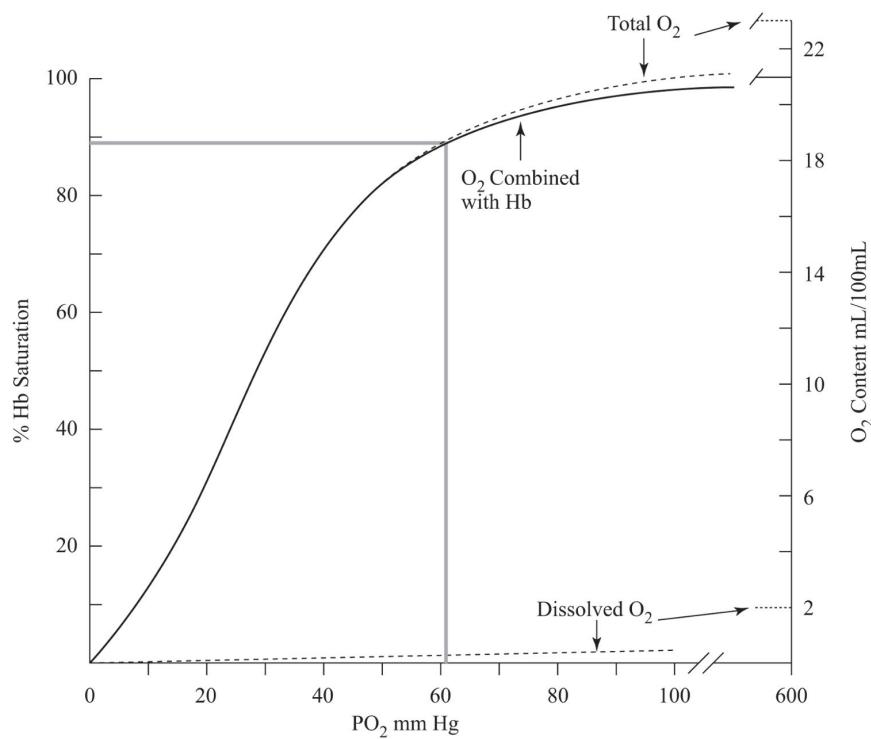


Figure 4-8. $\text{PaO}_2/\text{SaO}_2$ relationship

For the Adult CCRN exam, you will need to understand a high-level concept: the **oxyhemoglobin-dissociation curve**. Certain clinical conditions make hemoglobin “hold on” to oxygen molecules (the curve shifts to the LEFT). Other conditions allow hemoglobin to “release” the oxygen more easily to the tissue (the curve shifts to the RIGHT). See Table 4-2 for conditions that shift the curve to the left and right and Figure 4-9 for an illustration of the oxyhemoglobin-dissociation curve.

Table 4-2. Clinical Conditions That Cause a Shift of the Oxyhemoglobin-Dissociation Curve

Shift to the Left	Shift to the Right
Alkalosis (low H ⁺) Low PaCO ₂ Hypothermia Low 2,3-DPG	Acidosis (high H ⁺) High PaCO ₂ Fever High 2,3-DPG
Bad for tissues; SaO ₂ is high but O ₂ is stuck to Hgb	Good for tissues; SaO ₂ is low but O ₂ is easily released to the tissues

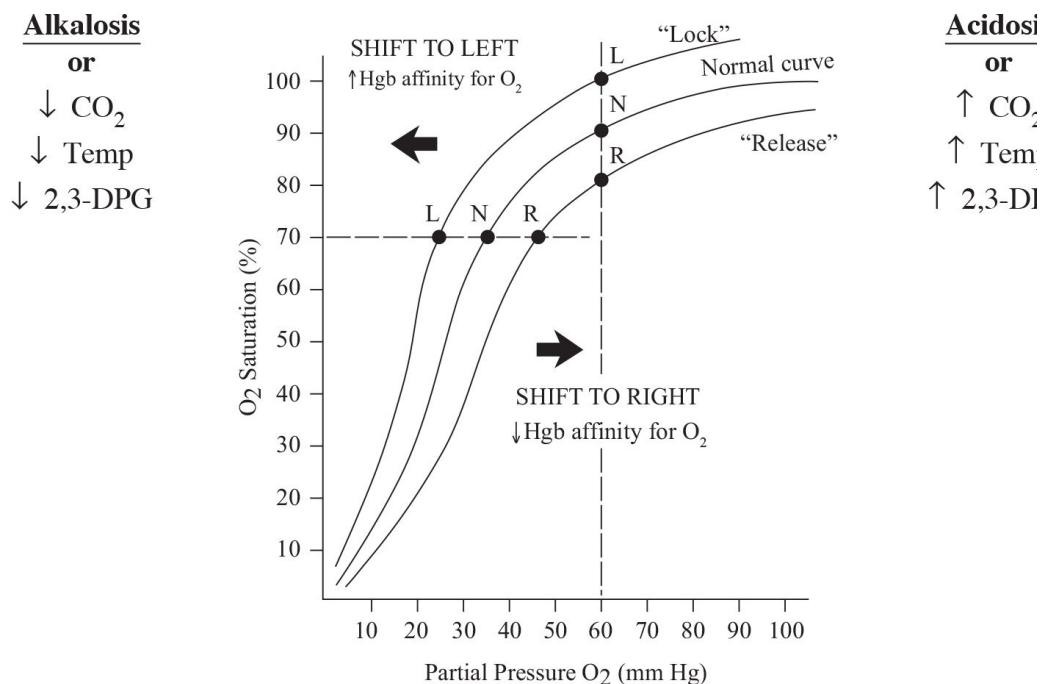


Figure 4-9. Oxyhemoglobin-dissociation curve

- Conditions that cause a shift to the left result in a higher SaO₂, but the tissues do not get needed O₂ as readily.
- Conditions that cause a shift to the right result in a somewhat lower SaO₂, but the tissues receive O₂ more readily.

2,3-Diphosphoglycerate (2,3-DPG)

- What is 2,3-DPG? It is an organic phosphate, found in RBCs, that has the ability to alter the affinity of Hgb for oxygen.
 - Decreased 2,3-DPG results in hemoglobin holding on to O₂ (Table 4-3).
 - Increased 2,3-DPG results in hemoglobin more readily releasing O₂ (Table 4-3).

Table 4-3. Effect of 2,3-DPG on Hgb Affinity for Oxygen

Decreased 2,3-DPG	Increased 2,3-DPG
Multiple blood transfusions of banked blood	Chronic hypoxemia (e.g., prolonged time spent at high altitudes or chronic HF)
Hypophosphatemia	Anemia
Hypothyroidism	Hyperthyroidism
Result: Less O ₂ is available to tissues	Result: More O ₂ is available to tissues

Carbon Monoxide Poisoning

- Carbon monoxide (CO) has a greater affinity for hemoglobin than oxygen—approximately 230 times greater (Figure 4-10)!

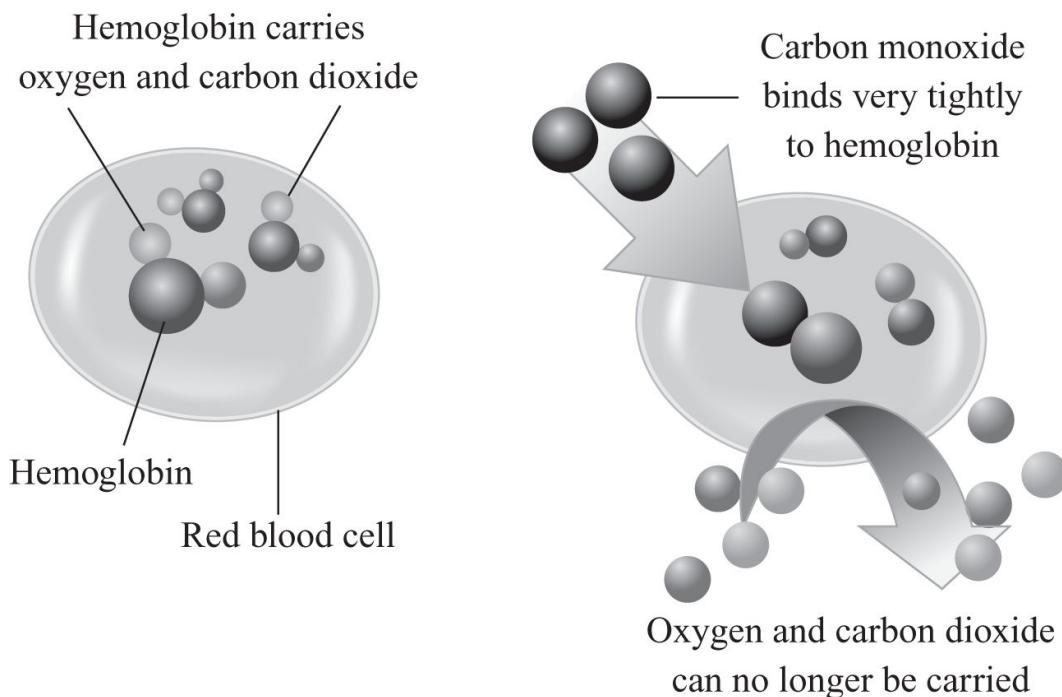


Figure 4-10. Carbon monoxide's effects on hemoglobin

- In the presence of CO, oxygen cannot be carried → tissue hypoxia.
- Do NOT use pulse oximetry to monitor the oxygenation status for a patient with CO poisoning. The pulse oximeter cannot differentiate between CO and O₂. Therefore, an SpO₂ of 95%, in the presence of CO poisoning, only means that the hemoglobin is saturated with a total of 95% molecules.
 - If the CO level of the blood is 40%, the maximum amount of O₂ that can be carried by hemoglobin is 60%.

Carboxyhemoglobin (COHb) levels and associated clinical presentation:

- 0–5% = Normal
- < 15% = Often in smokers, truck drivers

- 15–40% = Headache, some confusion
- 40–60% = Loss of consciousness, Cheyne-Stokes respiration
- 50–70% = Mortality > 50%

☆ Treatment

- 100% FiO_2 until symptoms resolve and carboxyhemoglobin level is < 10%
- Hyperbaric oxygen chamber if available, generally within 30 minutes

Lung Compliance

Think of compliance as the degree of elasticity of tissue. Therefore, a decrease in compliance increases resistance or stiffness.

- **Static compliance:** measurement of the elastic properties of the **lung**

Tidal volume ÷ plateau pressure (minus PEEP)

- Note that an increase in plateau pressure will decrease compliance.

- **Dynamic compliance:** measurement of the elastic properties of the **airways**

Tidal volume ÷ peak inspiratory pressure (minus PEEP)

- Note that an increase in peak inspiratory pressure will decrease compliance.
- Normal for both is ~ 45–50 mL/cm H₂O.
- Patients with pulmonary problems that mainly involve the airways (e.g., asthma) have a decrease in dynamic compliance, but their static compliance remains normal (Figure 4-11).
- Patients with pulmonary problems that mainly involve the lungs (e.g., pneumonia, ARDS) have a decrease in static compliance, but their dynamic compliance may also decrease as the lung pressures may transmit up to the airways (Figure 4-11).

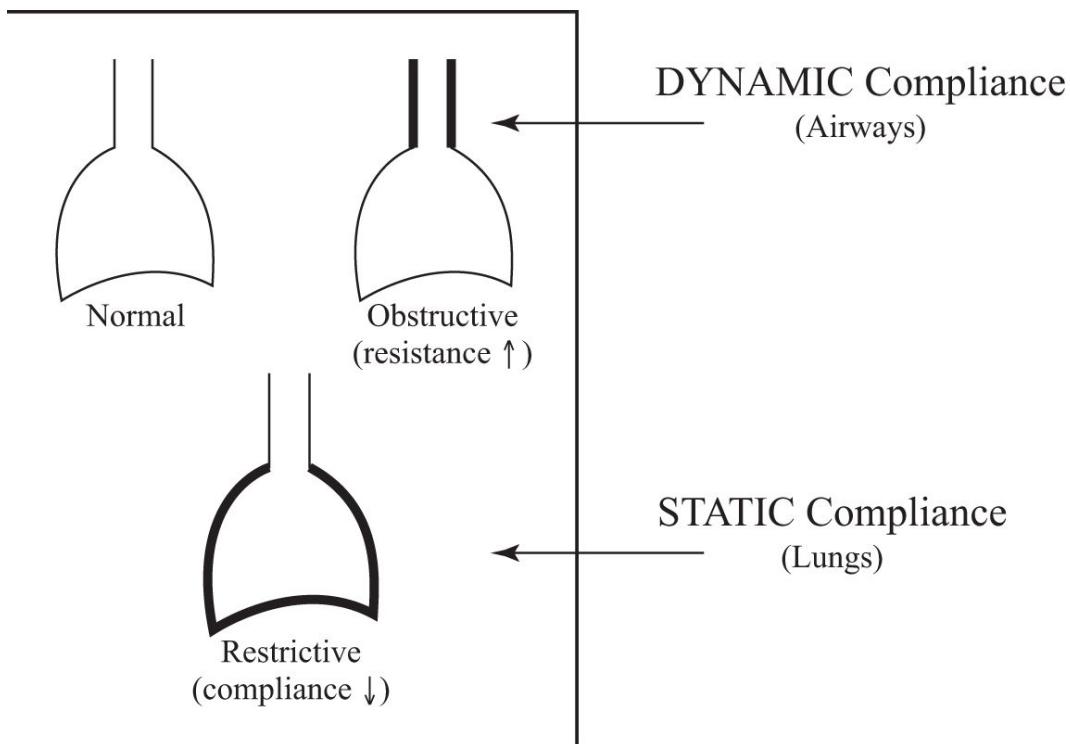


Figure 4-11. Dynamic vs. static compliance

TIP

★ For the exam, remember that decreased compliance increases the work of breathing.

- Status asthmaticus
 - Static compliance (lungs) would be normal.
 - Dynamic compliance would be low.
- ARDS
 - Static compliance would be low.
 - Dynamic compliance would also be low.

Acid-Base Interpretation

ABGs WILL be tested on the Adult CCRN exam. You need to understand the 4 major acid-base abnormalities as well as states of compensation—uncompensated, partial compensation, and full compensation. Not only will you need to know how to interpret the ABG, but you will also need to know what the clinical implications are for the patient and the treatment, if there are any. How did you do on the ABG question in the pretest of this book? A succinct review of acid-base interpretation is given in Table 4–4. Practice ABGs are also provided later in this section (with answers at the end of this chapter) so that you can practice as needed.

Table 4-4. Normal ABG Parameters

Parameter	Normal Range	Absolute Normal
pH	7.35–7.45	7.40
pCO ₂	35–45 mmHg	40 mmHg
HCO ₃	22–26 mmol/L	24 mmol/L
BE	–2 to +2	0
PO ₂	80–100 mmHg	
SaO ₂	95–99% (on room air)	

The information that follows will focus on acid-base interpretation, not oxygenation, which is covered in other areas of this book.

General Points Related to Acid-Base Balance

- Remember that the pH represents the hydrogen ion (H^+) concentration of the blood.
- Due to the Henderson-Hasselbalch equation, when the H^+ concentration is increased, the pH decreases, and when the H^+ concentration is decreased, the pH increases. There is an inverse relationship between the H^+ and the pH, so don't get confused!
- Think of $PaCO_2$ as an acid. When it increases, there is acidosis. When it decreases, there is alkalosis.
 - The $PaCO_2$ is controlled by the lungs. It is the **respiratory** parameter.
 - The lungs can change the $PaCO_2$ within minutes (rapid change).
- Think of HCO_3 as a base. When it is greater than normal, alkalosis may be present. When it is less than normal, acidosis may be present.
 - The HCO_3 is controlled by the kidneys. It is the **metabolic** parameter.
 - The kidneys alter the HCO_3 over hours to days (slow change).

Table 4-5. Four Primary Acid-Base Disorders and Expected Compensatory Change

Imbalance	pH*	Primary Change	Compensatory Change
Respiratory acidosis	< 7.35	↑ $PaCO_2$	↑ HCO_3
Metabolic acidosis**	< 7.35	↓ HCO_3	↓ $PaCO_2$
Respiratory alkalosis	> 7.45	↓ $PaCO_2$	↓ HCO_3
Metabolic alkalosis	> 7.45	↑ HCO_3	↑ $PaCO_2$

*In the presence of FULL compensation, the pH will enter the normal range.

**Metabolic acidosis may also be evaluated by the anion gap (see below) and the venous CO_2 (which will be lower than normal in the presence of metabolic acidosis).

Anion Gap

The anion gap is the difference between positive and negative anions. In most instances of metabolic acidosis, there is an increase in the anion gap. In several types of metabolic acidosis, though, the anion gap remains normal.

You will NOT need to calculate the anion gap for this exam, but you should know what is normal:

$$(\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-)$$

- Normal is 5–15 mEq/L.
- The anion gap is helpful in determining the cause of and/or response to treatment for metabolic acidosis (Table 4-6). For instance, if a patient with DKA presents with an anion gap of 25 mEq/L, one would expect the anion gap to decrease gradually as the patient responds positively to treatment. Since electrolytes are assessed frequently, the acidosis can be monitored by monitoring the anion gap without getting frequent ABGs.

Table 4-6. Problems Associated with the Anion Gap

Problems Associated with an Increase in the Anion Gap (think “Kussmaul”)	Problems Associated with a Normal Anion Gap
Ketoacidosis	Saline infusion (hyperchloremic acidosis)
Uremia	TPN
Salicylate intoxication	Diarrhea
Methanol toxicity	Acute renal failure, sometimes chronic
Alcoholic ketosis	
Unmeasured osmoles: ethylene glycol, paraldehyde	Note that there are fewer problems associated with a normal anion gap metabolic acidosis than those associated with a high anion gap acidosis.
Lactic acidosis: shock, hypoxemia	

NOTE

You will not need to know how to calculate the anion gap for this exam.

Acid-Base Compensation

- Compensation is the body's way of attempting to return the pH to normal (7.35–7.45).
 - Uncompensated
 - Partial compensation
 - Full compensation: **rare** in critically ill, suspect a mixed disorder if present
 - Review Tables 4-7, 4-8, 4-9, and 4-10

Table 4-7. Examples of Compensation for Respiratory Acidosis

Uncompensated	Partial Compensation	Full Compensation
pH 7.30	pH 7.32	pH 7.35
PaCO ₂ 50	PaCO ₂ 50	PaCO ₂ 50
HCO ₃ 24	HCO ₃ 29	HCO ₃ 31

Table 4-8. Examples of Compensation for Respiratory Alkalosis

Uncompensated	Partial Compensation	Full Compensation
pH 7.55	pH 7.50	pH 7.45
PaCO ₂ 25	PaCO ₂ 25	PaCO ₂ 25
HCO ₃ 22	HCO ₃ 18	HCO ₃ 16

Table 4-9. Examples of Compensation for Metabolic Acidosis

Uncompensated	Partial Compensation	Full Compensation
pH 7.30	pH 7.32	pH 7.35
PaCO ₂ 35	PaCO ₂ 33	PaCO ₂ 30
HCO ₃ 16	HCO ₃ 16	HCO ₃ 16

Table 4-10. Examples of Compensation for Metabolic Alkalosis

Uncompensated	Partial Compensation	Full Compensation
pH 7.50	pH 7.47	pH 7.45
PaCO ₂ 45	PaCO ₂ 48	PaCO ₂ 50
HCO ₃ 32	HCO ₃ 32	HCO ₃ 32

COMBINED ACID-BASE DISORDERS—SELDOM SEEN ON THE EXAM

Occurs when 2 single disorders are present simultaneously to produce the **same** abnormality.

- Combined respiratory and metabolic acidosis:

pH 7.21

PaCO₂ 50

HCO_3 12

- Combined respiratory and metabolic alkalosis:

pH 7.59

PaCO_2 30

HCO_3 33

MIXED ACID-BASE DISORDERS—SELDOM SEEN ON THE EXAM

- Simple acid-base disorders result from a single process, such as metabolic acidosis.
- In many critically ill patients, multiple acid-base disturbances exist concurrently and result in complex, mixed acid-base disorders.
- For example, a patient with septic shock may present with respiratory alkalosis **and** metabolic acidosis.
- Complex formulas can be applied to determine whether the compensating parameter (PaCO_2 or HCO_3) has compensated more than predicted for the primary problem, indicating that a mixed disorder is present. You will not need to know these formulas for this exam.

SYSTEMATIC ASSESSMENT OF ACID-BASE

1. Evaluate the pH → determine whether it is normal, acidotic, or alkalotic.
2. Evaluate respiratory parameters and then renal parameters → determine which, if either, is abnormal.
3. Determine the state of compensation.
4. Evaluate for a mixed disorder.
5. Assess oxygenation (PaO_2 , SaO_2).

Practice ABGs

Directions: Interpret each of the following ABGs. Identify the primary problem and the type of compensation. The answers can be found on page 108.

ABG	Interpretation
1. pH 7.48 PaCO ₂ 32 HCO ₃ 24	1. _____
2. pH 7.32 PaCO ₂ 48 HCO ₃ 25	2. _____
3. pH 7.30 PaCO ₂ 38 HCO ₃ 18	3. _____
4. pH 7.28 PaCO ₂ 60 HCO ₃ 29	4. _____
5. pH 7.49 PaCO ₂ 40 HCO ₃ 30	5. _____
6. pH 7.28 PaCO ₂ 70 HCO ₃ 33	6. _____
7. pH 7.50 PaCO ₂ 49 HCO ₃ 38	7. _____
8. pH 7.31 PaCO ₂ 32 HCO ₃ 15	8. _____
9. pH 7.30 PaCO ₂ 50 HCO ₃ 25	9. _____
10. pH 7.48 PaCO ₂ 40 HCO ₃ 30	10. _____
11. pH 7.38 PaCO ₂ 80 HCO ₃ 47	11. _____

Respiratory Disorders

About 14 respiratory topics are included in the Adult CCRN test blueprint. However, some topics are more likely to be included on the exam than others. It is highly recommended that you study ARDS, status asthmaticus, pneumonia, and pulmonary embolism in order to do well on the respiratory questions. The physiological concepts that were reviewed previously in this chapter are the foundation for all of the respiratory disorders. Specific respiratory disorders that are likely to be seen on this exam are reviewed in the following sections.

Acute Respiratory Failure

- Acute respiratory failure is defined as a **rapidly** occurring inability of the lungs to maintain adequate oxygenation of the blood with or without impairment of carbon dioxide (CO_2) elimination.

Specifically, the ABG demonstrates:

- PaO_2 of 60 mmHg or less, with or without an elevation of PaCO_2 to 50 mmHg or more with $\text{pH} < 7.30$
- As seen from the definition, the primary problem may be one of hypoxemia (Type 1) or hypercarbia (Type 2) or both (Type 3).
- **Type 1 (Hypoxemic)**
 - ARDS
 - Asthma
 - Atelectasis
 - Interstitial fibrosis
 - Pneumonia
 - Pulmonary edema (heart failure)
 - Pulmonary embolism (massive)
 - Smoke inhalation
- **Type 2 (Hypercapnic)**
 - CNS depression due to oversedation
 - COPD (acute exacerbation)
 - Head trauma
 - Musculoskeletal disorders or trauma
 - Sleep apnea
 - Status asthmaticus
- **Type 3**

- ARDS (late)
- COPD (late, acute exacerbation)
- Status asthmaticus (late)
- Clinical signs/symptoms of acute **hypoxemic** respiratory failure
 - Pulmonary: tachypnea, adventitious breath sounds, accessory muscle use
 - Cardiac: tachyarrhythmias (initial), bradyarrhythmias (late), hypertension or hypotension, cyanosis (central, e.g., lips, earlobes)
 - Neurological: anxiety, agitation
- Clinical signs/symptoms of acute **hypercapnic** respiratory failure
 - Pulmonary: shallow breathing, bradypnea, lungs may be clear or there may be adventitious breath sounds
 - Neurological: progressive decreased level of consciousness (lethargic, obtunded, stuporous, unresponsive)

Prompt identification and treatment may prevent a catastrophic outcome! The etiology of the signs/symptoms may not be the primary focus initially.

Treatment of Acute Respiratory Failure

- Maintain airway and improve ventilation.
 - Positioning (upright)
 - Suctioning
 - Bronchodilator therapy for wheezing
 - Noninvasive ventilation
 - Intubation, mechanical ventilation if needed
 - Repeat ABGs as needed
- Optimize oxygenation.
 - Adjust FiO_2 to keep $\text{SaO}_2 \sim > 0.90$
 - Decrease FiO_2 to 0.50 or less ASAP
 - Do not allow hypoxemia to occur to “prevent O_2 toxicity”
 - Use PEEP/CPAP as needed
 - Use pulse oximetry to monitor response to therapy
- Optimize circulation, cardiac output.
 - Manage hypotension
 - Address cardiac arrhythmias
- Identify the etiology, target treatment accordingly.
- Provide emotional support.

Use of Noninvasive Ventilation for the Management of Acute Respiratory Failure

When used for an appropriate patient, noninvasive ventilation (NIV) has been shown to decrease morbidity and mortality. There are 2 main types of NIV. Generally, though, the exam does not cover the details related to the type of NIV. More importantly, understand those who would NOT benefit from this therapy. Occasionally, a patient may initially be a good candidate for NIV but then, due to a change in condition, the patient should be intubated with an endotracheal tube.

- CPAP—Continuous positive airway pressure
 - Indicated for patients with hypoxemic respiratory failure who have increased work of breathing (e.g., cardiogenic pulmonary edema)
 - Settings include FiO_2 and 1 pressure setting in $\text{cm H}_2\text{O}$ pressure.
- BiPAP—Bilevel positive airway pressure
 - Indicated for patients with hypoxemic and/or hypercapnic respiratory failure
 - Settings include FiO_2 and 2 pressure settings: the inspiratory positive airway pressure (IPAP) and the expiratory positive airway pressure (EPAP).
 - IPAP assists ventilation and EPAP assists oxygenation.

Advantages of NIV

- Buys time for medical treatment to take effect
- Reduces the work of breathing (WOB)
- Decreases preload and afterload
- Improves oxygenation
- Improves ventilation (BiPAP)
- Reduces atelectasis
- Prevents intubation and resultant risks

☆ Contraindications for NIV

- Hemodynamic instability or life-threatening arrhythmias
- Copious secretions
- High risk of aspiration
- Impaired mental status (unable to protect airway)
- Suspected pneumothorax
- Inability to cooperate
- Life-threatening refractory hypoxemia ($\text{PaO}_2 < 60$ with $\text{FiO}_2 1.00$)

Use of High-Flow Nasal Cannula (HFNC) Oxygen

HFNC oxygen therapy, which has long been used for pediatric and infant populations, is now used for select adult populations in the treatment of acute respiratory failure and post-extubation.

- HFNC oxygen delivery systems are able to deliver FiO_2 (up to 100%) of heated and humidified gas at flow rates up to 60 L/minute via a nasal cannula.
- Advantages of HFNC therapy
 - Able to provide high FiO_2 (up to 100%)
 - Heated and humidified oxygen may improve secretion clearance and decrease airway inflammation
 - Able to meet high inspiratory flow demands of tachypneic patients
 - Seems to promote alveolar recruitment and increase FRC
 - Decreases dead space ventilation
 - More comfortable than CPAP or BiPAP masks, allows access to the mouth without removal of a mask
- Limitations of HFNC oxygen therapy
 - Unable to deliver higher airway pressures (PEEP or CPAP), and the low levels of airway pressure provided are variable when mouth breathing
 - Provides limited pressure support for a patient with hypercapnic respiratory failure
- Indications: community-acquired pneumonia; cardiogenic pulmonary edema when NIV is not tolerated; preoxygenation

prior to intubation; post-extubation (even in low-risk patients); for a patient who refuses intubation (DNI) but accepts alternate treatment measures

- HFNC oxygen therapy may also be used in conjunction with NIV post-extubation to prevent re-intubation.
- Nursing implications
 - Monitor for deteriorating oxygenation/ventilation (the patient may require NIV or intubation and mechanical ventilation)
 - Assess for nasal skin irritation

Chronic Obstructive Pulmonary Disease (COPD): Acute Exacerbation

- COPD includes emphysema, asthma, and bronchitis (more details on status asthmaticus are in the next section).
- In general, with each type of COPD, it is easier for air to enter the pulmonary system than to exit it; inspiration is easier than exhalation.
- Physiological consequences of COPD include:
 - Dynamic hyperinflation occurs due to too much air in the lungs.
 - Air trapping and auto-PEEP are common.
 - Expiratory flow rates are LOW.
 - An acute exacerbation results in a V/Q mismatch due to a problem with ventilation and an increase in the PaCO_2 .
 - The patient may have chronic CO_2 retention; if so, the patient will have partial or complete compensation and high HCO_3 on the ABG.
 - COPD may result in right ventricular enlargement (cor pulmonale) and elevated CVP.
- Signs of an acute exacerbation of COPD include:
 - Worsening dyspnea
 - Increase in sputum purulence
 - Increase in sputum volume
 - Hypercapnia, hypoxemia
- Management of an acute exacerbation of COPD includes:

- Titrate FiO_2 to $\text{PaO}_2 > 60 \text{ mmHg}$ or $\text{SaO}_2 > 90\%$ with care not to overcorrect hypoxemia and decrease respiratory drive
 - Must address **severe** hypoxemia; do not withhold oxygen only because hypoventilation may occur . . . cells still need oxygen
- Bronchodilator therapy
 - Inhaled short-acting beta-agonist (SABA), e.g., albuterol
 - Inhaled anticholinergic
- Monitor the level of consciousness for decreased responsiveness (clinical sign of worsening hypercapnia).
- Corticosteroid therapy
- Antibiotic therapy (when pneumonia is thought to be the trigger)
- Proceed with mechanical ventilatory support if needed (noninvasive or invasive).
 - Multiple studies have shown that noninvasive ventilation (NIV) is beneficial for patients with an acute exacerbation of COPD.

Status Asthmaticus

- Status asthmaticus is airway hyper-reactivity that produces severe airway narrowing that is refractory to aggressive bronchodilator therapy, which may result in respiratory failure. Status asthmaticus can be fatal as evidenced by the pathophysiology diagram in Figure 4-12.

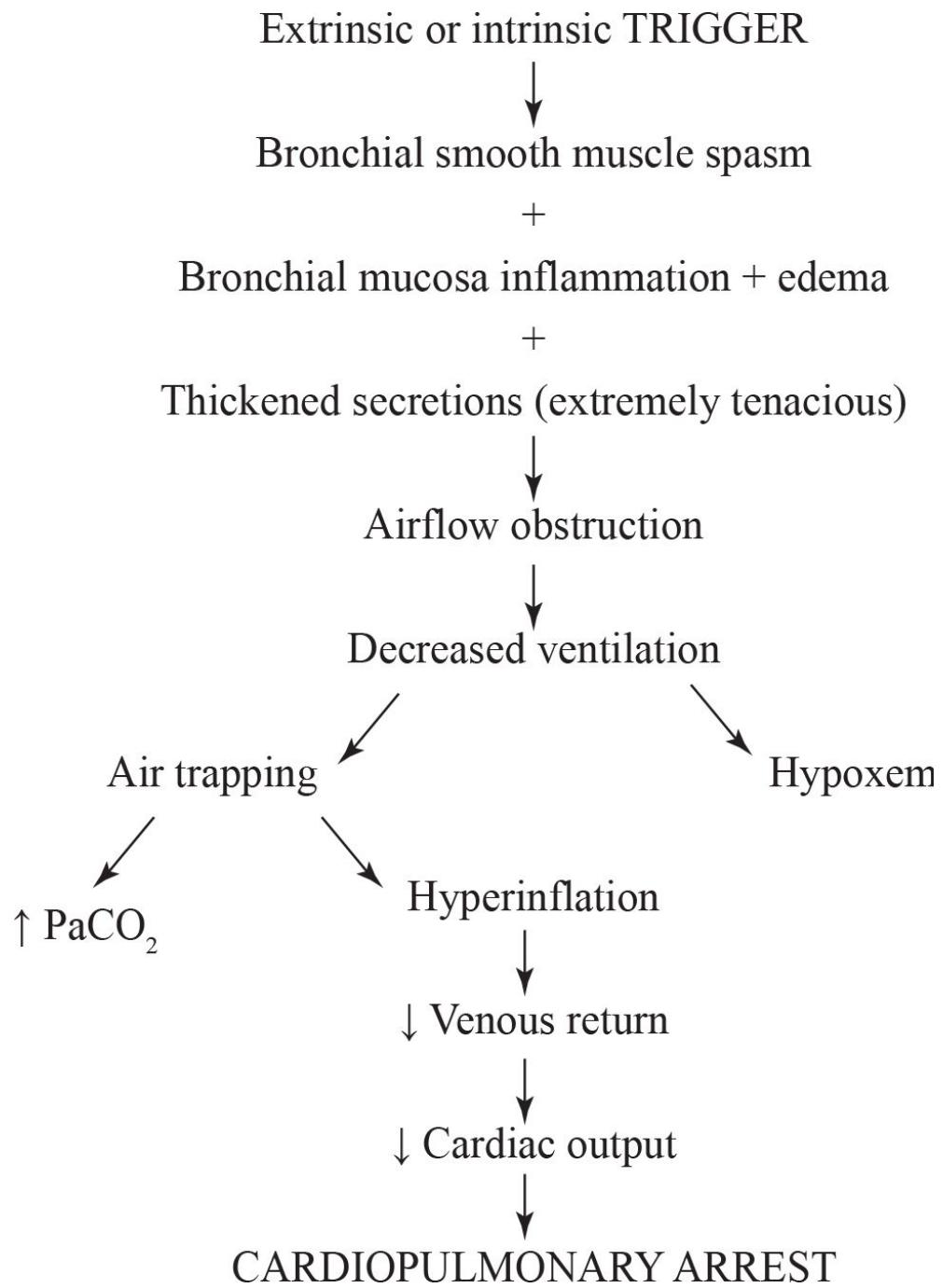


Figure 4-12. Pathophysiology of status asthmaticus

Clinical Presentation of Status Asthmaticus

- Dyspnea, tachypnea
- Cough, chest tightness
- Accessory muscle use
- Wheezing → decreased breath sounds → absent breath sounds . . . ominous sign!
- V/Q mismatch
- Chest X-ray may have flattened diaphragm (sign of air trapping).
- Tachycardia
- Pulsus paradoxus ≥ 15 mmHg (severe is > 18 mmHg)
- Anxiety → ↓ LOC
- May have elevated WBC, eosinophils
- Peak flow rate $< 80\%$ of predicted, $< 50\%$ is severe
- History of previous intubations (higher mortality)
- Table 4-11 describes ABG changes.

Table 4-11. ABG Progression in Status Asthmaticus (on Room Air)

Stage 1	Normal PaO ₂ , respiratory alkalosis (↓ PaCO ₂)
Stage 2	Mild hypoxemia, respiratory alkalosis (↓ PaCO ₂)
Stage 3	Worsening hypoxemia, normalization of pH and PaCO ₂
Stage 4	Severe hypoxemia, respiratory acidosis

Management of Status Asthmaticus

- Measure presenting peak flow rate (PFR).
 - Admit the patient to the hospital if the PFR is 50–70%.
 - Admit the patient to the ICU if the PFR is < 50%.
- Bronchodilator: short-acting beta-2 agonists, e.g., albuterol (Ventolin)
- Anticholinergics, e.g., ipratropium (Atrovent)
- Corticosteroids (systemic)
- O₂, pulse oximetry
- Hydration to prevent thickened secretions
- Avoid sedation agents.
- Intubation, mechanical ventilation if any of the following ominous signs occur:
 - Respiratory acidosis
 - Severe hypoxemia
 - Silent chest
 - Change in the level of consciousness (LOC)
- If the patient is intubated and sedated on mechanical ventilation, avoid paralytics because paralytics combined with steroids increase incidences of neuropathy.
 - ☆ Ventilator management for status asthmaticus
 - Use low rate to increase exhalation time.
 - Use low tidal volumes to prevent auto-PEEP.
 - Increase inspiration/expiration (I/E) ratio, often greater than 1:3–4, to allow time for optimal exhalation and to prevent auto-PEEP.

Pulmonary Embolism

- A pulmonary embolism (PE) is a partial or complete obstruction of the pulmonary capillary bed by a blood clot or another substance such as fat, air, amniotic fluid, or a foreign material, with a disruption of blood flow to an area of the lung.
 - Massive: > 50% occlusion
 - Submassive: < 50% occlusion
 - 80–90% result from DVT
- Although a pulmonary embolism may be the result of a variety of causes, venous thromboembolism (VTE) is the primary cause. VTE and a fat embolism are the 2 types of embolism that are most likely to be covered on the Adult CCRN exam. Refer to Table 4-12 for the risk factors for VTE and Figure 4-13 for the pathophysiology of PE.

Table 4-12. Risk Factors for Venous Thromboembolism (VTE)

Strong	Moderate	Weak
Fracture (hip or leg) Hip or knee replacement Major trauma Spinal cord injury	Arthroscopic knee surgery Central venous lines Chemotherapy HF or respiratory failure Hormone replacement therapy Malignancy Oral contraceptives Stroke Pregnancy, postpartum Previous VTE	Bed rest > 3 days Prolonged sitting Increasing age Laparoscopic surgery Obesity Pregnancy, antepartum Varicose veins

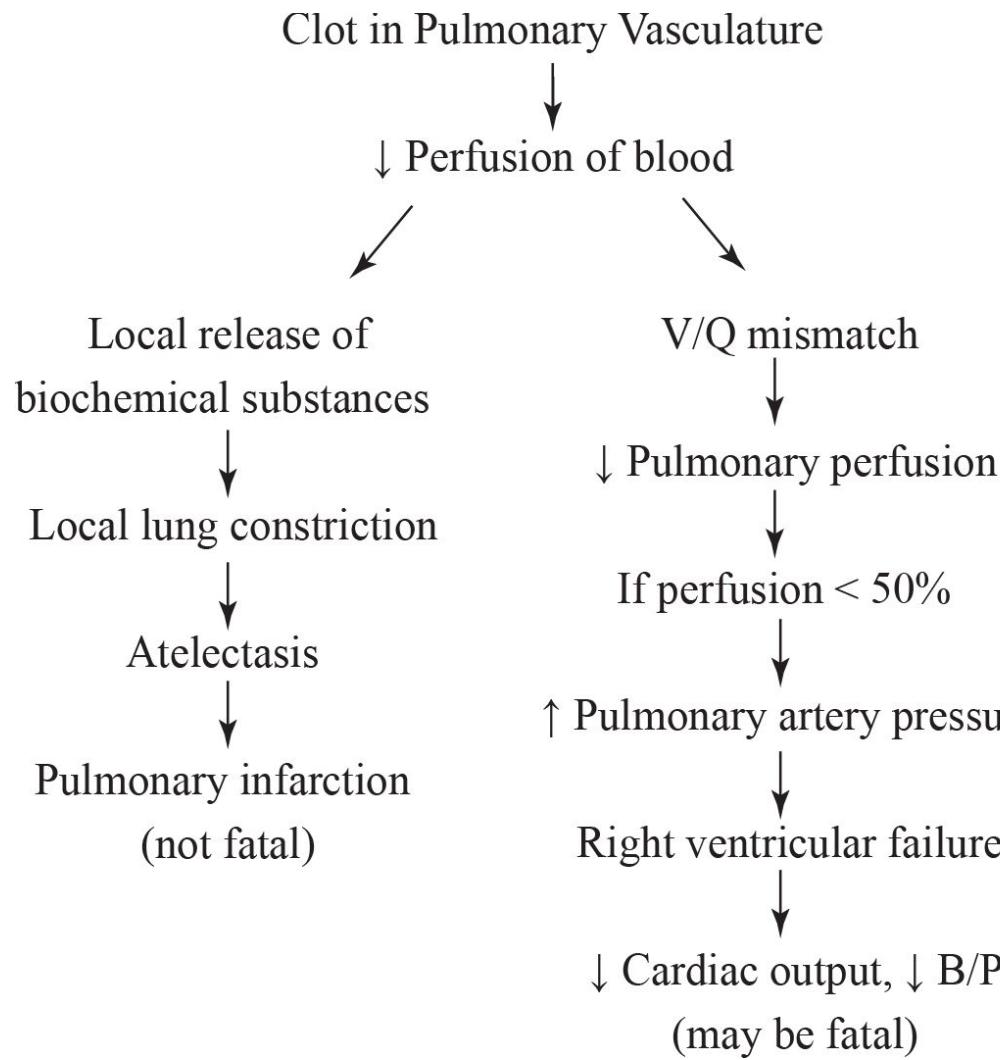


Figure 4-13. Pathophysiology of pulmonary embolism

Refer to Table 4-13 for the signs and symptoms of a PE.

Table 4-13. Signs and Symptoms of a PE

Most PEs	Massive PEs
Dyspnea, tachypnea	Hypoxemia
Tachycardia, chest pain	Hypotension
Right-sided S3 or S4 heart sounds	EKG changes—RBBB, right axis deviation on the ECG, tall peaked P-waves in lead II, RV strain pattern, ST elevation in V1 and V2
Anxiety, apprehension	Cardiopulmonary arrest—PEA
Cough, hemoptysis, crackles	Elevated BNP (due to right ventricular wall stress)
Syncope	
Petechiae (fat emboli)	
Low-grade fever	
Respiratory alkalosis	
Positive D-dimer	

Types of PE

- **Venous thromboembolism—DVT**
- **Fat emboli—long bone, pelvic fractures**
- Air emboli—surgery, IV lines
- Catheter embolization
- RA/LA or RV embolus—AFib/flutter (left atrial leading to stroke is more common)
- Amniotic fluid embolism (rare)—amniocentesis, abruptio placenta, or abortion
- Tumor emboli—malignancy causes an increase in thrombin
- Septic emboli—bacterial/viral

Diagnosis of PE

- Pulmonary angiography—gold standard!
- V/Q scan: “high” probability, “low” probability, not definitive
- High-speed CT scan
- D-dimer: good rule-out test; if positive, it means that a clot is present in the body; therefore, if symptoms ARE due to a PE, expect the D-dimer to be positive
- Venous Doppler (helps with source)

TIP

★ A PE will increase alveolar dead space!

Note: ~ 2/3 of PEs never get diagnosed!

Table 4-14 lists mechanical and pharmacological agents that are used to **prevent** PEs.

Table 4-14. Prevention of PEs

Mechanical	Pharmacological
Graduated compression stockings (GCS) and/or intermittent pneumatic compression (IPC) Use continuously except while ambulating!	Low-molecular-weight heparin (LMWH): enoxaparin (Lovenox) DAILY Low-dose unfractionated heparin: t.i.d. rivaroxaban (Xarelto): DAILY apixaban (Eliquis): b.i.d.

Treatment of PE

- Maintain adequate airway, ventilation, and oxygenation
- Fluids!
- Anticoagulation
 - Heparin (80 units/kg IVP and then 18 units/kg/hr drip)
 - Low-molecular-weight heparin (1 mg/kg q 12 hrs)
 - Coumadin **on the first treatment day** if able
 - The patient may require long-term anticoagulation.

NOTE

You will not need to know drug doses for this exam.

- Fibrinolytic therapy: **for all patients with hemodynamic compromise with a low risk for bleeding**
- Maintain cardiac output (inotropes, fluids)
- Analgesics for a patient who experiences pain

Pulmonary Hypertension

Pulmonary hypertension (PH) is included on the test blueprint. However, you are more likely to see questions that cover status asthmaticus, PE, pneumonia, and ARDS than questions that cover PH. Keep this in mind when studying.

- PH is defined as a MEAN pulmonary artery pressure that is greater than 25 mmHg at rest and a PAOP that is less than 16 mmHg at rest with secondary right heart failure.
 - The normal mean pulmonary artery pressure is ~ 20 mmHg. Since the RV normally pumps into a low-pressure system, the wall of the RV is thin compared to that of the LV. Pulmonary hypertension results in cor pulmonale and right ventricular failure.
- 5 groups of PH, as defined by the World Health Organization (WHO)
 - Group 1—Pulmonary arterial hypertension (PAH); sporadic and hereditary due to localized small pulmonary muscular arterioles (i.e., collagen vascular diseases, drug/toxin induced)
 - Group 2—Pulmonary hypertension (PH) due to left heart disease, such as LVF or valvular (mitral, aortic) disease
 - Group 3—PH due to lung diseases or hypoxemia
 - Group 4—PH due to chronic thromboembolic problems
 - Group 5—PH that has unclear factors or is multifactorial (e.g., sarcoidosis)

Signs and Symptoms of Pulmonary Hypertension

- Exertional dyspnea, lethargy, and fatigue due to an inability to increase cardiac output with activity
- Progression to RV failure, chest pain, syncope with exertion, and peripheral edema
- Passive hepatic congestion may cause anorexia and ABD pain
- Ortner's syndrome—cough, hemoptysis, and hoarseness
- Systolic ejection murmur, increased intensity of pulmonic component of S2 heart sound, diastolic pulmonic regurgitation murmur, right-sided murmurs, and gallops are augmented with inspiration.
- RV hypertrophy, elevated JVD, hepatomegaly, ascites, and pleural effusion

Treatment of Pulmonary Hypertension

- Treat the underlying cause as able.
- Each “group” has specific treatments based on the cause.
- All regimens should consider diuretics, oxygen, anticoagulants, digoxin, and exercise training.
- Use dilators, which include calcium channel blockers or phosphodiesterase-5 inhibitors, e.g., sildenafil (Viagra), tadalafil (Cialis), or treprostинil (Remodulin).
- For patients who are refractory to all medical interventions—lung transplantation (bilateral or heart-lung transplant) or possible atrial septostomy (right-to-left shunt)

Pneumonia

Pneumonia is an acute inflammation of the lung parenchyma (caused by an infectious agent) that can lead to alveolar consolidation.

Causative agents include:

1. Bacterial
2. Viral
3. Fungal
4. Parasitic

Pneumonia may also be classified according to where it developed:

- Community-acquired pneumonia (CAP)
 - Outside the hospital
 - Common pathogens: *Streptococcus pneumoniae*, *Legionella pneumophila*, *Klebsiella pneumoniae*, *Haemophilus influenzae*, *Staphylococcus aureus*, *Mycoplasma pneumoniae*, *Pseudomonas aeruginosa*
- Hospital-acquired pneumonia (HAP)
 - Acute care
 - Long-term care
 - Nursing home
- ☆ Ventilator-associated pneumonia (VAP); now referred to as ventilator-associated event (VAE)

- By definition, develops 48 hours or more after admission to the hospital
- Common pathogens: *P. aeruginosa*, *Escherichia coli*, *K. pneumoniae*, *Acinetobacter baumannii*, *Staphylococcus aureus* (especially diabetes and head trauma), MRSA
- HAP has higher mortality than CAP.

Risk Factors for Pneumonia

Multiple factors will increase the risk, including:

- Age
- Preexisting pulmonary disease
- Smoking
- ↓ level of consciousness (LOC)
- Artificial airways
- Chronic illness
- Malnutrition
- Immunocompromised
- Increased secretions
- Atelectasis
- Immobility
- Depressed cough or gag reflexes
- Concurrent antibiotic therapy
- Aspiration
- Organisms spread from another site (gut, wound) to the lungs.
- Multiple organ dysfunction syndrome (MODS)

Signs and Symptoms of Pneumonia

- Chills, diaphoresis, fever, malaise
- Tachycardia, chest pain
- Confusion (especially for the elderly)
- Productive cough
- Use of accessory muscles
- Dehydration
- Over area of consolidation on the chest:
 - ↑ Tactile fremitus
 - Dull to percussion
 - Bronchial breath sounds or diminished breath sounds
 - Bronchophony (louder/clearer)
 - Egophony (“e” to “a”)
 - Whispered pectoriloquy (whisper heard better with a stethoscope)

Diagnosis of Pneumonia

- CXR: consolidation or diffuse patchy infiltrates
- Sputum culture with Gram stain
- Blood cultures
- WBC: high but may be normal or low in immunocompromised or elderly people
- WBC differential: increased bands $> 10\%$
- ABGs: hypoxemia
- Thoracentesis for effusions

Treatment of Pneumonia

- Optimize oxygenation and ventilation.
 - Titrate FiO₂
- ☆ Positioning—GOOD lung DOWN
 - Bronchial hygiene, chest physiotherapy
 - Prone positioning for severe hypoxemia
 - Noninvasive ventilation or intubation/mechanical ventilation as needed
 - Bronchoscopy (with lavage, if needed)
 - Mobilize, clear secretions
- Identify organism
 - Sputum culture and sensitivity (C&S)
 - Blood cultures
- Antibiotic therapy
 - Empiric therapy: choice of agent is based on the likely causative organism (as determined by a patient assessment and the types of pneumonia seen in the community and in the institution) and whether that organism may be resistant to therapy
 - Timing: first dose within 4 hours if the patient first presents to the Emergency Department (and is later admitted to the hospital); the first antibiotic dose should be given in the Emergency Department; note that if the patient has sepsis, the antibiotic timing differs (see Chapter 5 of this book)
 - Organism-specific therapy: as soon as the results of the C&S are available

- System support
 - Hydration
 - Fever management
 - Glucose control
 - Nutrition
- General preventative measures
 - Smoking cessation
 - Pneumonia vaccine for those who are 65 and older
 - Flu vaccine

Prevention of Hospital-Acquired Pneumonia

- Practice hand hygiene.
- Keep HOB elevated 30 degrees or greater.
- Prevent bacterial translocation from GI tract: use the gut, feed patient.
- Practice oral hygiene!
- Provide education on common institution pathogens and the rates of nosocomial pneumonia.
- Use evidence-based confirmation of feeding tube placement.
 - Confirm with an X-ray prior to using for feeding.
 - Mark the exit site with an indelible marker for future reference.
 - Assess patency every 4 hours.
 - Observe for a change in the length of the external portion of the feeding tube (as determined by movement of the marked portion of the tube).
 - Review routine chest and abdominal X-ray reports to look for notations about tube location.
 - Observe changes in volume of aspirate from feeding tube.
 - If pH strips are available, measure the pH of feeding tube aspirates if feedings are interrupted for more than a few hours.
 - Observe the appearance of feeding tube aspirates if feedings are interrupted for more than a few hours.
 - Obtain an X-ray to confirm tube position if there is doubt about the tube's location.

Prevention of Ventilator-Associated Pneumonia

Prevention involves all of the interventions for preventing hospital-acquired pneumonia, plus:

- Drain accumulated condensate from tubing.
- Prevent backflow of tubing condensate into endotracheal tube (ETT).
- Change ventilator tubing only when it is contaminated.
- Mobilize the patient.
- Utilize aseptic technique for ETT, tracheostomy suctioning.
- Adhere to mouth care protocol, chlorhexidine mouth rinse.
- Brush teeth to remove plaque.
- Keep ETT cuff inflated.
- Perform subglottic suctioning prior to cuff deflation.
- Perform routine oropharyngeal suctioning.

Aspiration

Aspiration is the inhalation of toxic substances into the lung, with an injury to the lung that is the result of the chemical, mechanical, and/or bacterial characteristics of the aspirate.

- Oropharyngeal is most common!
- May or may not involve an infection
- May be acute or chronic: micro or massive
- Table 4-15 shows management techniques.

Table 4-15. Emergent Management of Aspiration

Witnessed Aspiration	All Aspirations
Place patient in slight Trendelenburg and turned to the right side to aid drainage Suction mouth and pharyngeal areas Bronchoscopy for large particles	O ₂ , titrate up as needed Intubation/mechanical ventilation as needed Monitor for the onset of noncardiogenic pulmonary edema (ARDS) or pneumonia Monitor for ↓ BP

- ★ Due to the anatomy of the right mainstem bronchus (shorter, wider, and with less of an angle), most aspirations occur in the **RIGHT** lung. Although aspirations may occur in both lungs, they seldom are isolated solely to the left lung.

Etiology of Aspirations

- Altered level of consciousness
- Drug or alcohol abuse
- Depressed gag, cough, or swallowing reflexes
- Presence of feeding tubes (all types)
- Improper patient positioning
- Presence of artificial airways
- Ileus or gastric distension
- History of dysphagia, GERD, esophageal strictures, ↓ GI motility
- Increased secretions

Signs and Symptoms of Aspirations

- Acute respiratory distress
- Presence of gastric contents in oropharynx
- Tachycardia
- Hypoxemia
- Crackles
- Copious secretions due to alveolar edema
- Hypotension (massive fluid shifts may occur)

Acute Respiratory Distress Syndrome (ARDS) and Acute Lung Injury (ALI)

ARDS and ALI are syndromes caused by a variety of acute conditions that trigger an inflammatory response, resulting in an increase in the permeability of the pulmonary capillary membrane, which allows a transudation of proteinaceous fluid into the interstitial and alveolar spaces. They may also be referred to as “noncardiogenic pulmonary edema.” Damage to Type II alveolar cells is one of the pathological consequences. Since these are the cells that are responsible for the production of surfactant, massive atelectasis occurs.

TIP

★ This exam always includes several questions on this topic! Study accordingly.

ALI is similar to ARDS in that both involve a shunt, which results in hypoxemia. The degree of the shunt is represented by the PaO_2 to FiO_2 ratio, as described in Table 4-16. Note that you will not be expected to differentiate between ALI and ARDS on this exam.

- All of the criteria in Table 4-16 must be present for a diagnosis of ARDS or ALI. The pulmonary edema is not due to heart failure. Hypoxemia is REFRactory, meaning that the FiO_2 is increased to the maximum of 100% and hypoxemia is still present.
- Since a shunt is present, PEEP needs to be provided in order to increase alveolar recruitment and treat the refractory hypoxemia.

Table 4-16. Differentiation of ARDS and ALI

ARDS	ALI
Acute onset with precipitating event Bilateral infiltrates consistent with pulmonary edema $\text{PaO}_2/\text{FiO}_2$ ratio ≤ 200 mmHg, regardless of the level of PEEP $\text{PAOP}^* \leq 18$ mmHg	Acute onset with precipitating event Bilateral infiltrates consistent with pulmonary edema $\text{PaO}_2/\text{FiO}_2$ ratio between 201 and 300 mmHg, regardless of the level of PEEP $\text{PAOP}^* \leq 18$ mmHg

*PAOP = pulmonary artery occlusion pressure

NOTE

PEEP treats a shunt by preventing alveolar collapse; it does not necessarily decrease pulmonary shunting.

PaO₂/FiO₂ Examples

- The patient is receiving 50% FiO₂ and PaO₂ is 90:

$$90 \div 0.50 = 180$$

- The patient is receiving 30% FiO₂ and PaO₂ is 110:

$$110 \div 0.30 = 367$$

- The patient is receiving room air and PaO₂ is 62:

$$62 \div 0.21 = 295$$

- The patient is on 100% FiO₂ and PaO₂ is 95:

$$95 \div 1.00 = 95$$

Remember: It is not only the PaO₂/FiO₂ ratio that is considered when diagnosing ALI or ARDS; the other 3 factors also need to be present.

Surfactant

- Phospholipid/lipoprotein produced by Type II alveolar cells
- Stabilizes alveoli, "keeps them open"
- Increases lung compliance
- Eases work of breathing
- Therefore, with ARDS (destruction of Type II alveolar cells):
 - Massive atelectasis, alveolar collapse
 - Decreased compliance
 - Increased work of breathing
 - Decreased functional residual capacity (FRC)

Figure 4-14 and Tables 4-17 and 4-18 show the pathophysiology, etiology, and signs and symptoms of ARDS and ALI.

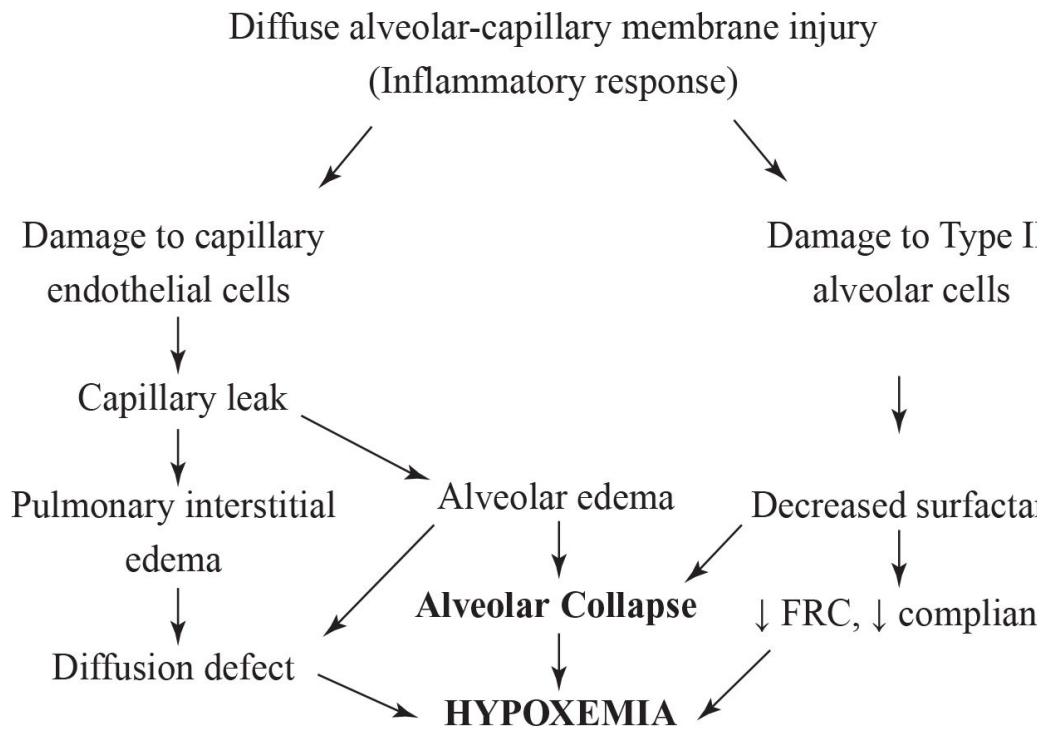


Figure 4-14. Pathophysiology of ARDS/ALI

Table 4-17. Etiology of ARDS/ALI

“Direct” Injury	“Indirect” Injury
Aspiration	Sepsis
Pneumonia	Shock
Pulmonary contusion	Head injury
Fat/air embolism	Non-thoracic trauma
O ₂ toxicity	Blood transfusion
Inhalation injury	Pancreatitis
Drowning	Burns
Transthoracic radiation	Heart bypass
	DIC

Table 4-18. Signs and Symptoms of ARDS/ALI

Early	Late
Tachycardia	Tachycardia, episodes of bradycardia
Apprehension, restlessness	Agitation
Mild dyspnea	Extreme dyspnea
Respiratory alkalosis	Respiratory and metabolic acidosis
Few crackles	Crackles, wheezes
Chest X-ray → isolated infiltrate or “ground-glass” appearance	Chest X-ray → whiteout/bilateral infiltrates
PaO ₂ on room air ~ 60 mmHg	PaO ₂ on room air ~ 30 mmHg, refractory hypoxemia despite ↑ FiO ₂

Treatment of ARDS/ALI

- ☆ Pulmonary stabilization strategies
 - Intubation with mechanical ventilation
 - PEEP, usually 15 cm H₂O or greater; monitor for barotrauma and ↓ cardiac output; treat hypotension, but do NOT discontinue PEEP
 - Note: Disconnection of the ventilator circuit (and PEEP) will result in alveolar derecruitment and hypoxemia that may not be readily corrected.
 - Limit plateau pressure to 30 cm H₂O or less.
 - Limit tidal volume (V_t) to 5–6 mL/kg → “permissive hypercapnia” to prevent volutrauma.
 - A low V_t will cause a rise in the PaCO₂ and a drop in the pH; however, patients tend to tolerate a pH as low as 7.2.
- Cardiovascular stabilization
 - Support the BP (fluids, vasopressors, especially when ARDS is due to septic shock).
 - Monitor for and treat arrhythmias.
- Prone positioning: helps deliver blood flow to underperfused lung units, thereby improving ventilation/perfusion; keeps alveolar lung units open, thus improving gas exchange and preventing further injury
 - Use extreme caution to avoid misplacement or loss of airway.
 - Prevent a pressure injury.
- Monitor acid-base balance.
- DVT and stress ulcer prophylaxis
- Analgesia, sedation
- Nutritional support
- Nitric oxide, prone positioning may provide improvement in oxygenation.
- Coordinate the interdisciplinary team—PT, OT, and dietitian.
- Prevent, identify organ failure.
- Provide emotional support (to the patient and the patient’s family).
- Monitor for complications.

- **No steroids** (except for select patients with COVID-19 pneumonia)

Complications of ARDS/ALI

The mortality from ARDS is still around 30%, although patients do not die from hypoxemia. Instead, they die from multiple organ dysfunction syndrome (MODS) and other complications, as listed below:

- Secondary infections
- Pulmonary embolus
- Ileus
- Skin breakdown
- Malnutrition
- Barotrauma: pneumothorax, subcutaneous emphysema

Pneumothorax

Pneumothorax (Figures 4-15 and 4-16 and Table 4-19) is included in the Adult CCRN test blueprint. A simple, unilateral pneumothorax is generally not life-threatening, unless it occurs in a patient with end-stage chronic lung disease. A tension pneumothorax, however, may be life-threatening. Therefore, you must know the difference between the two types. The test makers also expect you to have an understanding of chest tube management.

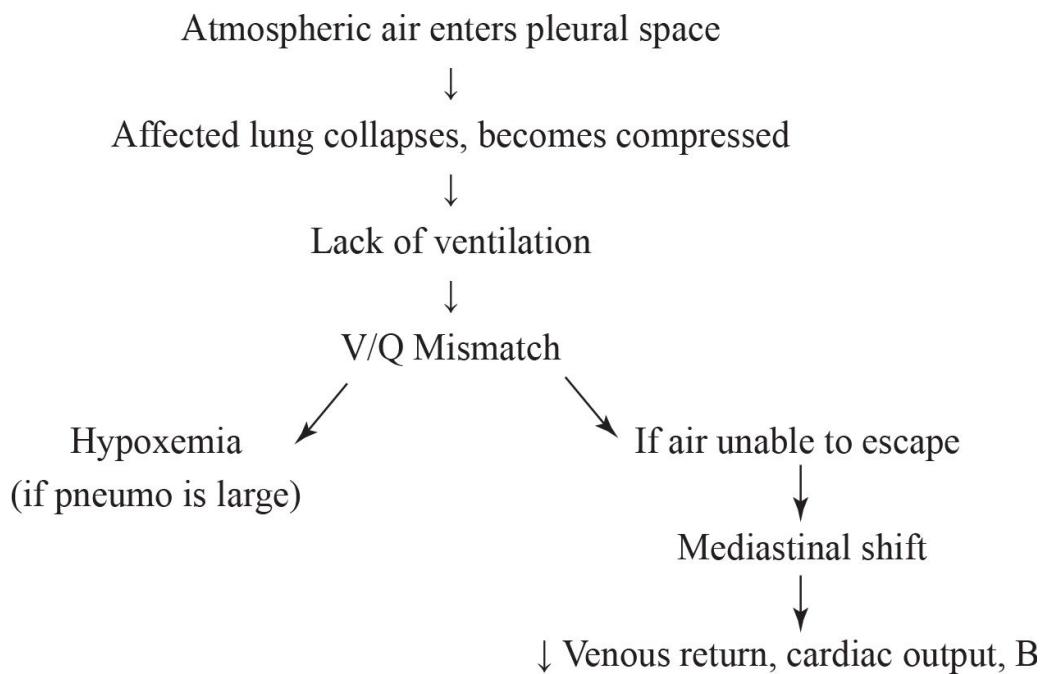


Figure 4-15. Pathophysiology of a pneumothorax

Types of Pneumothorax

- Spontaneous
- Traumatic
 - Open (penetrating chest trauma)
 - Closed (blunt chest trauma)
 - Iatrogenic (due to therapeutic or diagnostic procedures) (Figure 4-16)

☆ Tension

- Air is unable to exit → mediastinal shift
- Life-threatening

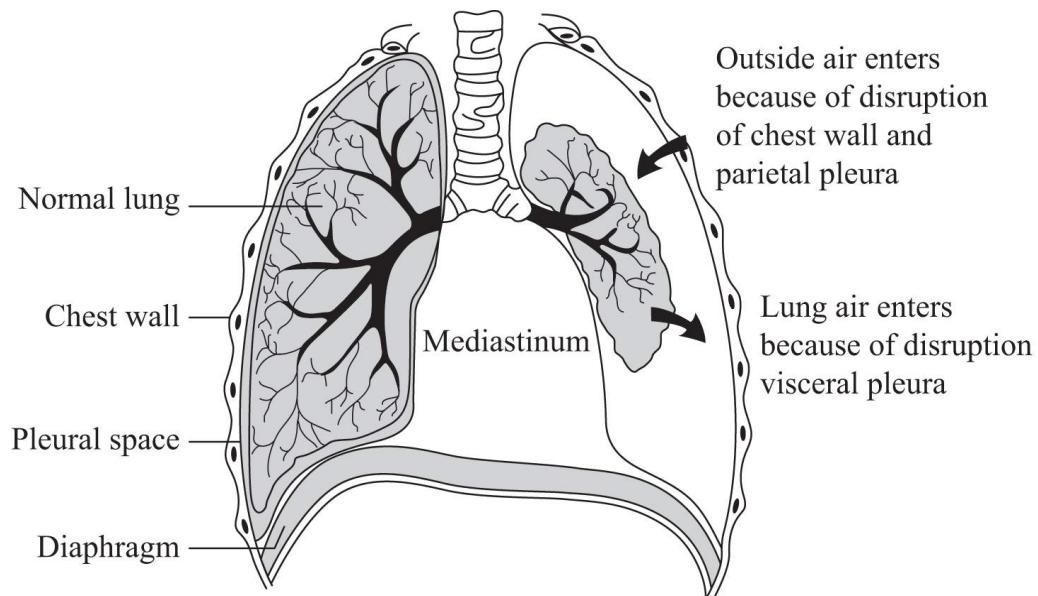
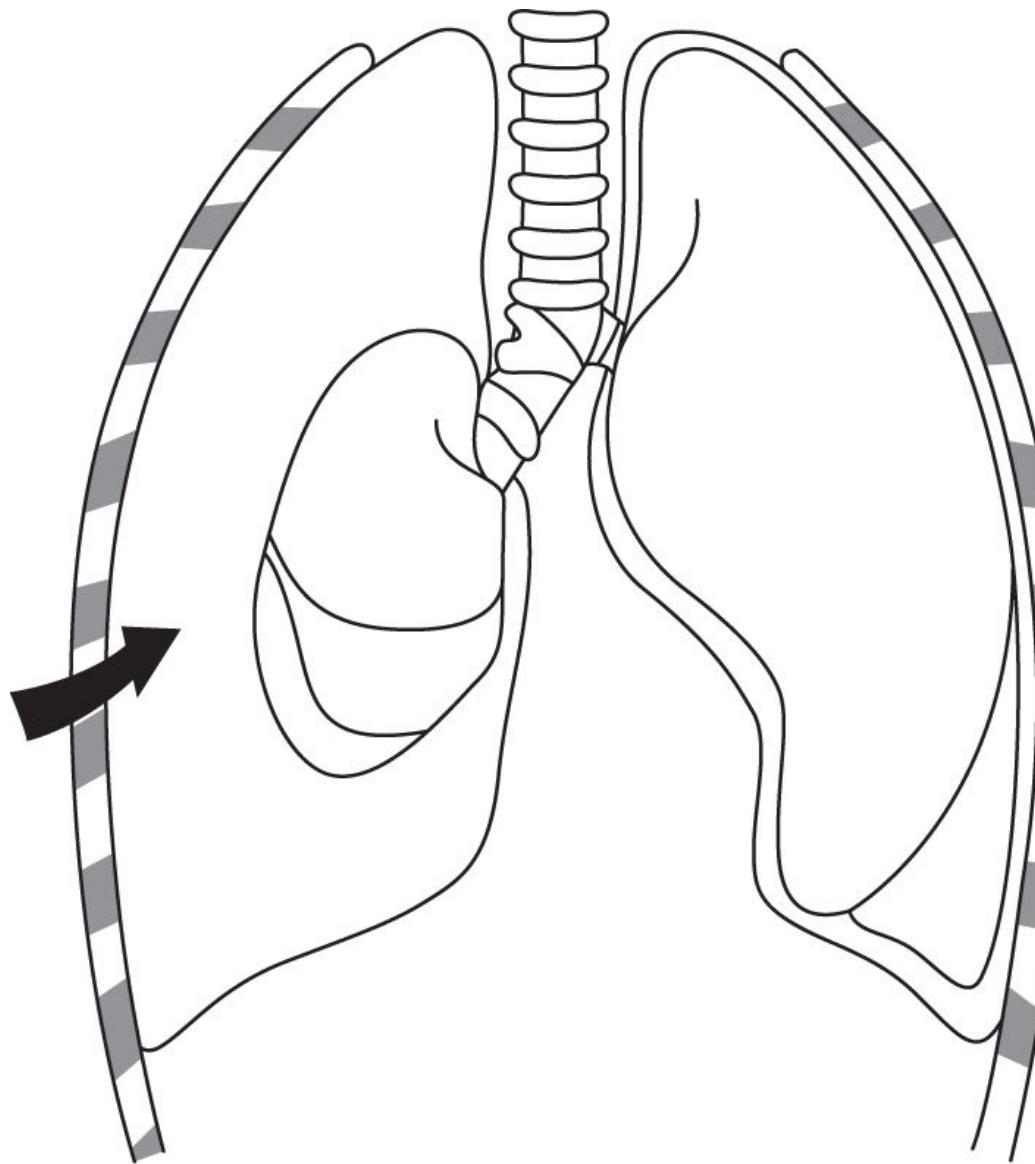


Figure 4-16. Pneumothorax (no mediastinal shift)

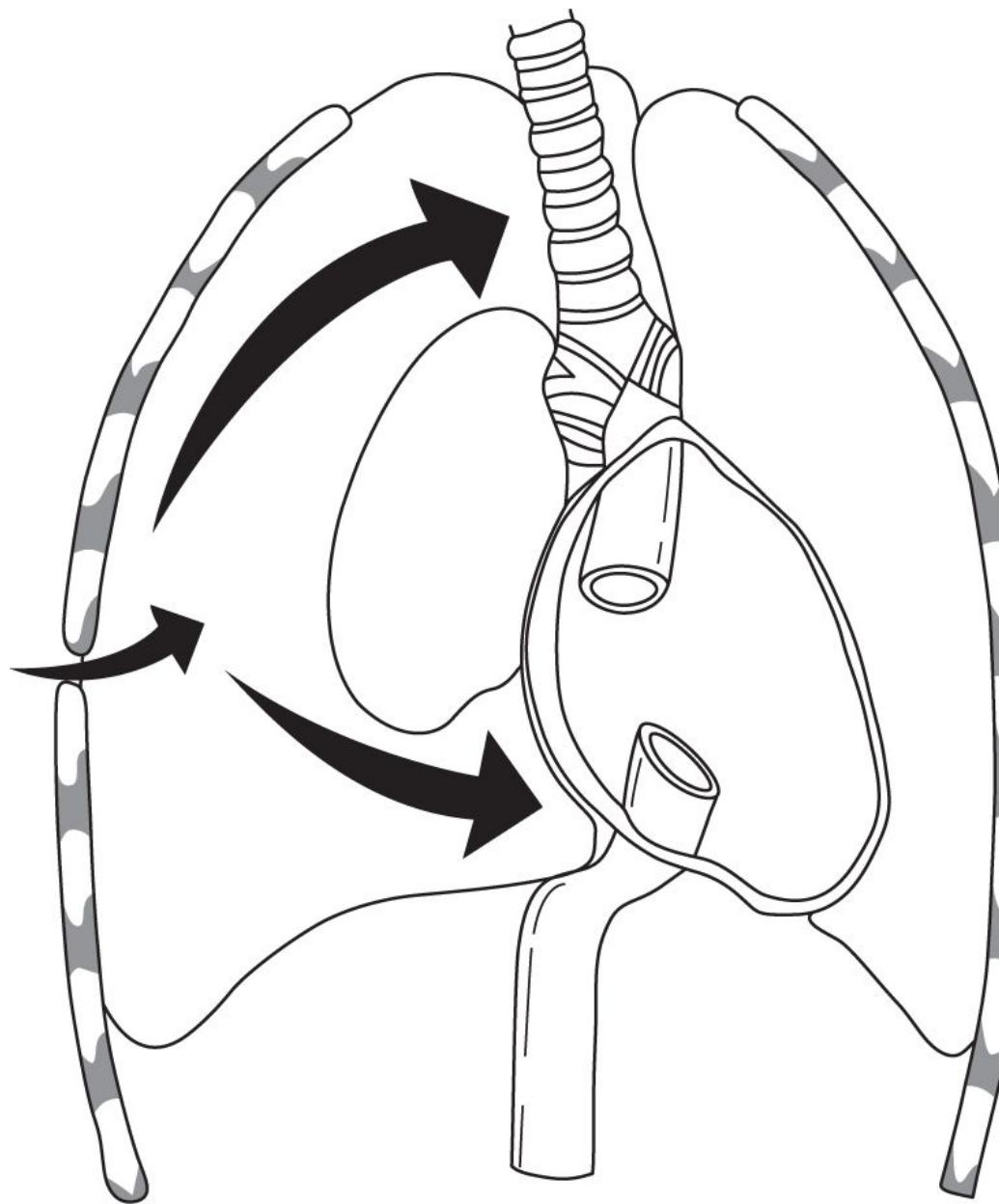
Table 4-19. Signs and Symptoms of a Pneumothorax

Spontaneous or Traumatic	Tension
<p>Depends upon the size of the pneumothorax and depends upon the underlying lung disease (if any)</p> <ul style="list-style-type: none"> • Dyspnea, tachypnea • Chest pain (not all cases) • Unequal chest excursion • Tracheal deviation (if present) toward the affected side • Hypoxemia (if large) • Decreased or absent breath sounds on the affected side • Mediastinum remains midline, no shift (see Figure 4-17) 	<p>Similar to a traumatic pneumothorax, EXCEPT:</p> <ul style="list-style-type: none"> • Tracheal deviation away from the affected side (see Figure 4-18) • Tachycardia • Distended neck veins • Mediastinal shift • HYPOTENSION • Life-threatening!



Simple Right Pneumothorax

Figure 4-17. Simple right pneumothorax (no tension)



Right Tension Pneumothorax

Figure 4-18. Right tension pneumothorax, mediastinal shift

Hemothorax

- Usually due to trauma; presents as lung collapse, with blood in the pleural space or in the mediastinal space

- Dullness to percussion
- Absent breath sounds on the affected side
- Tracheal deviation toward the unaffected side

Treatment for a Pneumothorax

- Pneumothorax > 20%
 - Chest tube: reestablish negative pleural pressure
 - Supplemental O₂
 - Treat pain, if needed.
- Pneumothorax < 20%
 - O₂
 - Monitor for lung reexpansion.
 - If there is a pneumothorax plus an underlying lung disease, the patient may need a chest tube.

☆ Chest Tube Assessment and Management

- Close assessment of the patient's respiratory status, which should improve after chest tube insertion
- Pain assessment, treatment
- Entry site—dressing assessment
- Tubing—no dependent loops!
- Drainage collection chamber
 - Keep lower than chest
- Water seal chamber
 - Tidaling with deep inspiration is normal.
 - Air leak
 - Bubbling in water seal chamber is not normal.
 - May be present postoperatively; if there has not been a leak and now there is bubbling, notify the physician.
 - Avoid high airway pressures with chest tubes in place in order to avoid an air leak.
- Suction control chamber: gauge or water level determines the amount of suction, NOT the wall suction source.
- Clamp only when changing the system, with inadvertent disconnection, or with a physician order.
 - Clamping cuts off the negative pressure water seal chamber; expanded lung may re-collapse.

Mechanical Ventilation

Endotracheal Tube Placement

- Confirmation of correct placement is done immediately after intubation.
 - Waveform capnography is most accurate.
 - End-tidal CO₂ detector
 - Auscultation
- Cuff inflation to 20 cm H₂O pressure
- Obtain a chest radiograph for placement confirmation; should be **3–5 cm above the carina**.
- Assess and document tube placement at the level of the teeth or gum line for an ongoing assessment of correct placement.
- If the tube migrates down, it most often migrates to the right lung due to the anatomy of the mainstem bronchi (right is shorter/wider, with less of an angle, than the left).
- Get ABGs within 20 to 30 minutes of intubation to assess acid-base status.
- The endotracheal tube:
 - Is narrow and increases airway resistance, similar to breathing through a straw
 - Is longer than a tracheostomy tube; therefore, it has a greater degree of dead space ventilation than a tracheostomy tube

Ventilator Management

- Ventilator breaths may be delivered at a set volume (most common for adults) or at a set pressure.
- The main focus of the exam is how the ventilator settings may differ depending upon the patient's primary problem (e.g., ARDS or asthma); nontraditional modes of ventilation are not covered on this exam.

Ventilator Modes

- Assist-control (AC) mode
 - The patient receives the **set tidal volume** at the set breath rate; the patient also receives the **set tidal volume** for each breath triggered by the patient's spontaneous effort made above the set breath rate.
 - For example, if the AC is set at a rate of 12 breaths/minute, at a tidal volume of 700 mL, and the patient's total rate per minute is 20, the tidal volume of the 8 extra breaths initiated by the patient is 700 mL because the extra breaths are sensed by the machine and the set tidal volume (700 mL) is given.
 - All breaths are machine breaths.
 - Provides full ventilatory support
 - Not used as a weaning mode unless it is being alternated with periods of spontaneous breathing (reducing the AC rate does nothing if the patient is spontaneously breathing)

- Can result in overventilation and/or hyperinflation of the lungs at higher spontaneous breathing rates
- Synchronized intermittent mandatory ventilation (SIMV) mode
 - The patient receives the set tidal volume at the set breath rate, and all breaths above the set rate are spontaneous breaths at the patient's own tidal volume.
 - For example, if the SIMV is set at a rate of 12 breaths/min, at a tidal volume of 700 mL, and the patient's total rate per minute is 20, the tidal volume of the 8 extra breaths will vary from breath to breath because the breaths that are spontaneously initiated by the patient are at the patient's own tidal volume.
 - All machine breaths are synchronized with the patient's breathing effort.
 - Provides full or partial ventilatory support
 - Reducing the SIMV rate will allow the patient to assume more of the work of breathing.
 - Spontaneous breaths may be pressure supported.

Ventilator Settings

Positive End-Expiratory Pressure (PEEP)

- Positive pressure is applied to the airways at the end of exhalation.
- Increases lung volume at the end of exhalation (FRC), creating more surface area for gas exchange; increases alveolar recruitment
- Can be applied to patients via artificial airways, full face mask, nasal mask, and nasal prongs (neonates)

- Think oxygenation!

Continuous Positive Airway Pressure (CPAP)

- CPAP is PEEP applied to a spontaneously breathing patient.
- The patient does not receive machine breaths.
- The patient assumes all of the work of breathing.
- Usually the last step in the weaning process
- All breaths may be pressure supported.
- The patient may experience fatigue if left on CPAP for an extended period of time.

Pressure Support Ventilation (PSV)

- The patient receives an increase in airway pressure during inspiration to augment (boost) the spontaneous tidal volume.
- Patient-triggered mode (if the patient is paralyzed and/or sedated, PSV will not be triggered on)
- Rate, tidal volume, inspiratory flow rate, and inspiratory time are determined by the patient's effort.
- You cannot use PSV with assist-control mode.
- PSV is frequently used during weaning to **reduce the work of breathing** and to overcome imposed work of ETT and ventilator circuit.

Breath Rate

- The breath rate is determined by the PaCO_2 , generally 12–16 breaths/minute on full ventilator support.

Tidal Volume (V_t)

- The tidal volume (V_t) is determined by the patient's ideal body weight and medical problem.
- Generally, the V_t is 6–8 mL/kg.
- For ARDS, in order to prevent "volutrauma," it is 5–6 mL/kg.

Fraction of Inspired Oxygen (FiO₂)

- The fraction of inspired oxygen (FiO₂) is generally set at 100% on intubation and is then adjusted down according to the PaO₂; the goal is to decrease it to 50% or less as soon as you are able to.

Ventilator Alarms

- Alarms are set for patient safety.

Cause of High-Pressure Limit Alarms

- Agitation
- Coughing
- Secretions
- Aspiration
- Kinked/occluded ETT or ventilator circuit
- Bronchospasm or mucosal edema
- Decreasing lung compliance (ARDS)
- Pneumothorax

Cause of Low-Pressure Limit Alarms

- Ventilator circuit disconnection or leak
- Inadequate tidal volume
- Cuff leak
- Chest tube leak

If You Are Unable to Troubleshoot an Alarm, What Should You Do?

- Disconnect the ventilator from the airway, and ventilate with a bag/valve device.
 - A bag/valve device should be at the bedside of every patient who is receiving mechanical ventilation, with the mask available as well, in case there is an issue with the artificial airway.
 - As noted in the section on ARDS, disconnecting the ventilator circuit (and PEEP) for a patient who requires high PEEP will result in alveolar derecruitment and hypoxemia, which may not be readily corrected.
- ☆ Refer to Table 4-20 for the different ventilator setting guidelines used for ARDS and asthma.

Table 4-20. Ventilator Setting Guidelines Used for ARDS and Asthma

ARDS	Asthma
Plateau (static) pressure (< 30 cm) Low tidal volume (5-6 mL/kg) High PEEP (15-20 cm)	Provide short inspiratory time and long expiratory time <ul style="list-style-type: none">• Low breath rate• Low V_t• High peak flow rate Monitor for auto-PEEP

Weaning from Mechanical Ventilation

Criteria for weaning from mechanical ventilation and initiating a spontaneous breathing trial:

- Original reason for intubation is being resolved.
- Resting minute ventilation (ideally < 10 L/min)

- Spontaneous tidal volume (ideally > 5 mL/kg)
- Negative inspiratory force (NIF) (ideally > -25 cm H₂O)
- Rapid shallow breathing index (respiratory rate/Vt) (ideally < 105 breaths/min/L)
- Vital capacity (ideally above 10 mL/kg body weight)
- ABGs/oxygenation acceptable with FiO₂ 50% or less

Criteria for stopping the spontaneous breathing trial:

- Respiratory rate (> 35 breaths/minute)
- Respiratory rate (< 8 breaths/minute)
- SpO₂ ($< 88\%$)
- Respiratory distress
- Mental status change
- Acute cardiac arrhythmia
- Acute hypotension

Now that you have reviewed the key respiratory concepts, go to the Practice Questions. Answer the questions, and then check your answers. Continue to review the information until you answer 80% of the practice questions correctly.

Practice Questions

1. Which of the following developments would indicate that a patient, who is receiving noninvasive ventilation, may require intubation with an endotracheal tube?
 - (A) a need for an increase in FiO_2 from 0.30 to 0.40
 - (B) a dry cough and a fever
 - (C) a change in mental status with difficulty to arouse
 - (D) positive blood cultures
2. A patient was discharged from the medical unit 7 days ago following an acute ischemic stroke and is now anxious and complaining of severe shortness of breath. The patient demonstrates tachycardia, tachypnea, hypotension, an SpO_2 of 88% on 3 L/nasal cannula, and a temperature of 37.9°C. Breath sounds are present, clear, and equal bilaterally. A chest X-ray is clear. ABGs reveal respiratory alkalosis. Based on this history and assessment, which of the following problems exists and which interventions should the nurse anticipate?
 - (A) increased dead space ventilation; fibrinolytic therapy
 - (B) decreased dynamic compliance; bronchodilators
 - (C) infection; antibiotics
 - (D) shunt; PEEP therapy

3. A patient who is receiving mechanical ventilation has a peak inspiratory pressure of 70 cm H₂O and a plateau pressure of 35 cm H₂O. What intervention would be most beneficial for this patient?
- (A) Increase the FiO₂.
(B) Administer morphine.
(C) Obtain stat ABGs.
(D) Administer a bronchodilator.
4. Which of the following is an appropriate intervention for a patient with status asthmaticus who is on a ventilator?
- (A) Decrease the peak flow rate.
(B) Increase the tidal volume.
(C) Check for auto-PEEP.
(D) Increase the breath rate.
5. Which of the following interventions has been demonstrated to decrease ventilator-associated pneumonia?
- (A) Provide sedation.
(B) Maintain the head of the bed at 20° or greater.
(C) Adhere to a mouth care protocol.
(D) Deflate the endotracheal tube cuff prior to suctioning.
6. A 49-year-old male, who weighs 70 kg, is admitted to the ICU with smoke inhalation and ARDS. He is receiving mechanical ventilation with the following ventilator settings: FiO₂ = 0.80, assist-control mode = 10 breaths/minute, Vt = 400 mL, and

PEEP = 15 cm of pressure. Arterial blood gases are as follows: pH 7.39, PaCO₂ 42, PaO₂ 96, HCO₃ 22, and O₂ sat 98%.

Which of the following interventions is appropriate?

- (A) Decrease the FiO₂.
- (B) Increase the tidal volume.
- (C) Decrease the PEEP.
- (D) Do not make any changes.

7. Which of the following statements about lung compliance is TRUE?

- (A) An increase in the peak inspiratory pressure will decrease static compliance.
- (B) A decrease in compliance increases the work of breathing.
- (C) Static compliance is decreased with an asthma exacerbation.
- (D) The plateau pressure is used to calculate dynamic compliance.

8. Each of the following is a clinical feature of ARDS EXCEPT:

- (A) bilateral chest infiltrates on an X-ray.
- (B) PAOP > 18 cm H₂O.
- (C) refractory hypoxemia.
- (D) tachypnea.

9. A patient has mild tachypnea, a productive cough, bronchial breath sounds in the right mid and lower lobes, dullness to percussion over the right lower chest, and an SpO₂ of 92% on

0.40 FiO₂. Which of the following interventions would be appropriate for this patient?

- (A) Contact the physician for an order for a bronchodilator.
- (B) Use noninvasive ventilation.
- (C) Avoid turning the patient to the right side.
- (D) Maintain the patient in a supine position.

10. Drug therapy that is most likely to be prescribed for a patient with status asthmaticus includes which of the following drug categories?

- (A) bronchodilators and anticoagulants
- (B) corticosteroids and diuretics
- (C) antibiotics and expectorants
- (D) bronchodilators and corticosteroids

11. The following interventions may improve oxygenation EXCEPT:

- (A) increasing the FiO₂.
- (B) giving PRBCs.
- (C) giving fluids or pressors to increase a low BP.
- (D) increasing the ventilator breath rate.

12. While monitoring a patient post-thoracotomy, the nurse suspects that there is hypoventilation. Which characteristic best defines hypoventilation?

- (A) $\text{PaCO}_2 > 45 \text{ mmHg}$
- (B) respiratory rate < 12
- (C) $\text{pH} < 7.35$
- (D) $\text{PaO}_2 < 60 \text{ mmHg}$

13. A patient who was admitted with a carboxyhemoglobin (COHb) level of 45% is lethargic and complains of a headache. The patient is receiving 100% O_2 per a face mask and is noted to have an SpO_2 of 100%. Which of the following is indicated?
- (A) Administer hydrocodone with acetaminophen.
 - (B) Continue the FiO_2 of 1.00.
 - (C) Decrease the FiO_2 .
 - (D) Intubate and initiate mechanical ventilation.

For questions 14 and 15

A 38-year-old female is admitted with respiratory failure secondary to viral pneumonitis. She is receiving mechanical ventilation and suddenly becomes restless, tachypneic, tachycardic, and hypotensive. The high-pressure ventilator alarm is continuous, and pulse oximetry (SpO_2) decreases to 0.83. Breath sounds are diminished on the right with tracheal deviation to the left.

14. Based on this information, what condition is likely developing?

- (A) ARDS (acute respiratory distress syndrome)
 - (B) hemothorax
 - (C) tension pneumothorax
 - (D) pulmonary embolism
15. Treatment for the patient described in this scenario would likely include which of the following?
- (A) morphine and furosemide
 - (B) addition of PEEP
 - (C) vasopressors
 - (D) chest tube

For questions 16 and 17

A 63-year-old male is admitted with acute respiratory distress. Symptoms include marked shortness of breath and circumoral cyanosis. He is awake and complains of shortness of breath. He has a history of COPD (chronic obstructive pulmonary disease). Blood gases reveal the following information:

pH	7.22
PaCO ₂	62
PaO ₂	54
SaO ₂	81%
HCO ₃	25

FiO_2 30%

16. Based on this information, what condition is likely developing?

- (A) congestive heart failure
- (B) ARDS
- (C) acute respiratory failure
- (D) pulmonary emboli

17. What would be the priority treatment indicated at this time?

- (A) Increase the FiO_2 .
 - (B) Intubate and initiate mechanical ventilation.
 - (C) Use postural drainage treatment.
 - (D) Administer a bronchodilator.
-

18. Which of the following may be an effect of mechanical ventilation and PEEP (positive end-expiratory pressure)?

- (A) atelectasis
- (B) oxygen toxicity
- (C) ARDS
- (D) reduced cardiac output

19. Which of the following is NOT a clinical finding seen in pneumonia?

- (A) a chest X-ray with an area of consolidation
 - (B) hypoxemia refractory to O₂ administration with a need for PEEP
 - (C) normal WBCs with increased immature neutrophils (bands)
 - (D) bronchial breath sounds, diminished breath sounds, or crackles on auscultation
20. Which of the following would be the earliest sign of hypoventilation?
- (A) respiratory rate of 20 breaths/minute
 - (B) anxiety
 - (C) decreased level of consciousness
 - (D) SpO₂ of 85%
21. A 70 kg patient with ARDS is intubated and mechanically ventilated. The patient is on a continuous vecuronium infusion to maintain a twitch of "1." The peak inspiratory pressure is 55 cm H₂O, and the plateau pressure is 50 cm H₂O. The PaO₂ is 60. The physician orders the following ventilator settings: assist-control mode = 12 breaths/minute, tidal volume 700 mL, FiO₂ 1.00, and PEEP 15. The nurse knows that:

- (A) continuous positive airway pressure (CPAP) is an appropriate setting for a patient who is receiving vecuronium.
 - (B) a tidal volume of 700 mL is inappropriate for this patient.
 - (C) the PEEP should be decreased to 5 cm H₂O pressure to improve oxygenation.
 - (D) the plateau pressure is appropriate for this patient.
22. When caring for a postoperative patient with a chest tube, care of the patient needs to include:
- (A) loop tubing right above the collection chamber.
 - (B) placing the collection chamber on the IV pole when turning the patient.
 - (C) clamping the chest tube during patient transport.
 - (D) reporting sudden bubbling in the negative pressure chamber.
23. A patient with an acute exacerbation of COPD is minimally responsive, tachypneic, and tachycardic. The ABG results include pH 7.20, PaCO₂ 68, and PaO₂ 65. The nurse anticipates that the next intervention will be:
- (A) initiating noninvasive ventilation.
 - (B) endotracheal intubation.
 - (C) beginning low-flow oxygen per a nasal cannula.
 - (D) administering sodium bicarbonate to correct acidosis.

24. A patient with a known history of asthma is admitted with asthma exacerbation. The critical care nurse needs to be aware of the potential clinical signs that may indicate a need for intubation and initiation of mechanical ventilation. Which of the following would indicate a possible need for intubation?
- (A) difficulty to arouse
 - (B) respiratory alkalosis
 - (C) bilateral wheezing
 - (D) SaO_2 92%
25. Which of the following factors would DECREASE the release of oxygen from hemoglobin at the tissue level?
- (A) a temperature of 39°C
 - (B) increased levels of 2,3-DPG
 - (C) arterial pH of 7.30
 - (D) a massive transfusion of stored, banked blood
26. A trauma patient with multiple long bone fractures suddenly develops agitation, tachypnea, tachycardia, and mild hypoxemia. Her lungs are clear, and a petechial rash is noted on her upper body. Which of the following is suspected?
- (A) acute respiratory distress syndrome
 - (B) fat embolism
 - (C) deep vein thrombosis
 - (D) delirium

27. A patient's arterial blood gas (ABG) is as follows: pH 7.32, PaCO_2 33, and HCO_3 16. Based on this ABG, what might be this patient's problem?

- (A) anxiety
- (B) shock
- (C) drug overdose
- (D) electrolyte imbalance

Answer Key

1. **C**

2. **A**

3. **D**

4. **C**

5. **C**

6. **A**

7. **B**

8. **B**

9. **C**

10. **D**

11. **D**

12. **A**

13. **B**

14. **C**

15. **D**

16. **C**

17. **A**

18. **D**

19. **B**

20. **C**

21. **B**

22. **D**

23. **B**

24. **A**

25. **D**

26. **B**

27. **B**

Answers and Explanations

1. **(C)** Noninvasive ventilation is not safe for a patient who is unable to protect his or her airway. A depressed level of consciousness increases the risk of airway obstruction or aspiration. Choice (A) can be provided with NIV. Choices (B) and (D) do not put a patient who is receiving NIV at a greater risk for a poor outcome.
2. **(A)** This scenario describes a patient who is experiencing a pulmonary embolism (PE). Signs of hypotension and hypoxemia indicate that it is a massive PE. The PE increases dead space ventilation due to the drop in pulmonary perfusion. Fibrinolytic therapy (tissue plasminogen activator) is indicated for a massive PE. For any of the other choices to be correct, the patient would have to have abnormal breath sounds.
3. **(D)** This clinical picture is one of asthma. Asthma is an airway problem that results in bronchospasm, which increases airway pressure as evidenced by an elevated inspiratory pressure while the lung pressure itself (the plateau pressure) remains normal. Symptoms would be relieved with a bronchodilator. The other choices would not relieve these symptoms.
4. **(C)** A patient with asthma has difficulty with expiration. Therefore, with each breath air may get trapped, resulting in elevation of the end-expiratory pressure without PEEP being set on the ventilator. This may decrease cardiac output because

it decreases venous return. The other interventions would all increase the air trapping.

5. **(C)** Mouth care decreases bacterial colonization in the mouth, which decreases the possibility of microaspiration of bacteria in lower airways. The other 3 choices have NOT been found to decrease incidences of ventilator-associated pneumonia. Sedation should be minimized. The head of the bed should be greater than 30°. The ETT cuff should not be deflated prior to suctioning but, instead, prior to ETT removal. (At that time, the mouth and the subglottic space should be well suctioned.)
6. **(A)** The goal is to get the FiO_2 down to 50% or less ASAP for a patient who requires high FiO_2 . This patient's oxygenation status would allow for a decrease in the FiO_2 . During acute ARDS, the tidal volume needs to be low to prevent volutrauma, and the PEEP needs to be maintained to limit atelectasis.
7. **(B)** Decreased compliance increases "stiffness" and therefore requires more work to ventilate. Choice (A) is not correct because an increase in the peak inspiratory pressure will decrease dynamic compliance. Choice (C) is not correct because asthma decreases dynamic (airway) compliance, not static (lung) compliance. Choice (D) is not correct because dynamic compliance is calculated using the peak inspiratory pressure.
8. **(B)** If the PAOP is high, the pulmonary edema is most likely cardiac in origin, whereas in ARDS, the pulmonary edema is noncardiogenic in origin. The other 3 choices (bilateral infiltrates, refractory hypoxemia, and tachypnea) are all present in ARDS.

9. **(C)** This clinical scenario is one of right-sided pneumonia. Therefore, the right side is the “bad” side. Due to the effects of gravity on perfusion, the patient generally does better with the “good” lung DOWN. With the “bad” lung down, there is an increased risk of worsening hypoxemia. Choice (A) is not correct because the patient is not wheezing. Therefore, there is no indication for a bronchodilator. Choice (B) is not correct because this condition does not warrant NIV. Choice (D) is not correct because the best position for a patient with a pulmonary problem is sitting upright, not lying flat.
10. **(D)** A patient with asthma needs a bronchodilator (to treat the bronchospasm caused by smooth muscle contraction around the airways) and steroids (to treat the airway inflammation). There is no indication for anticoagulants, diuretics, or antibiotics to treat asthma.
11. **(D)** An increase in the ventilator breath rate will influence the PaCO_2 (ventilation), not oxygenation. The other 3 choices will improve oxygenation at the tissue level. An increase in the FiO_2 will provide more oxygen at the alveolar-capillary membrane. A blood transfusion will increase oxygen content by increasing hemoglobin, which is the carrier of oxygen. Increasing a low blood pressure will increase oxygen delivery.
12. **(A)** The best clinical indicator of effective ventilation is the PaCO_2 . Although a breath rate less than 12 may indicate hypoventilation, it is not as sensitive an indicator as is PaCO_2 . In addition, the breath rate does not include the other determinant of effective ventilation (depth of ventilation or tidal volume). Although a low pH may indicate hypoventilation, it may

also indicate metabolic acidosis. Hypoxemia (PaO_2 less than 80 mmHg on room air) may be present during hyperventilation.

13. **(B)** If the patient has a COHb level of 45%, the best SaO_2 achievable is 55% since saturation of hemoglobin cannot be greater than 100% total. The patient needs 100% FiO_2 until COHb levels have returned to normal and symptoms (alteration in the level of consciousness and headaches) are relieved. Opiates may depress the level of consciousness and mask the patient's response to FiO_2 . A decrease in the FiO_2 would decrease the driving forces of oxygen and prolong abnormal COHb levels. Intubation would not help oxygenation since the same FiO_2 can be provided with a face mask. Intubation would be indicated for a ventilation problem, but the patient does not have signs of hypoventilation.
14. **(C)** This clinical scenario describes a pneumothorax. As a result of hypotension and tracheal deviation to the opposite side of the pneumothorax, the pneumothorax is a life-threatening tension pneumothorax with symptoms that are the result of a mediastinal shift. If ARDS was the problem, the patient would have bilateral crackles. If a hemothorax was the problem, there would generally be a history of trauma, not an infection. A mediastinal shift (tracheal deviation to the opposite side and hypotension) is not usually present with a hemothorax. If the problem was a PE, the breath sounds would be equal bilaterally.
15. **(D)** The treatment for a tension pneumothorax is a chest tube (generally after emergent needle decompression). None of the other choices is beneficial for treating a tension pneumothorax.

16. **(C)** Not enough information is provided to select any condition other than acute respiratory failure. Acute respiratory failure is an ABG diagnosis, and this ABG meets the requirements of acute respiratory failure. Do NOT read into the questions.
17. **(A)** This patient has severe hypoxemia that needs to be addressed first in order to prevent all of the associated effects of the severe hypoxemia. Although the patient is not ventilating normally, the elevated PaCO_2 is less of a problem, as indicated by the patient's mental status. The level of consciousness is not depressed. There is no indication in the scenario for choices (C) or (D).
18. **(D)** Initiation of mechanical ventilation (even without PEEP) that delivers positive pressure breaths results in a mild increase in the intrathoracic pressure with a resultant decrease in venous return. PEEP increases the intrathoracic pressure even more and causes a decrease in venous return. Mechanical ventilation and/or PEEP do not cause any of the other 3 choices.
19. **(B)** Pneumonia results in a V/Q mismatch, not a shunt. Therefore, the administration of oxygen will correct the hypoxemia. PEEP is not required as it is for a shunt (ARDS). The worse the pneumonia, the greater the FiO_2 requirement will be. The other 3 choices are possible clinical indicators of pneumonia.
20. **(C)** As ventilation decreases, the PaCO_2 rises and affects the brain by decreasing the level of consciousness from lethargy to an obtunded state, to a stupor, to a comatose state. Although the patient MAY have hypoventilation with a respiratory rate of 20 breaths/minute, it would not be considered a typical sign.

Anxiety and SpO₂ are more likely signs of hypoxemia than early signs of hypoventilation.

21. **(B)** Since the patient has ARDS, low tidal volumes (5–6 mL/kg) are indicated (permissive hypercapnia) in order to prevent volutrauma. Since the patient weighs 70 kg, a V_t of 700 mL is too high. CPAP requires that the patient has a spontaneous breathing effort. Therefore, CPAP is not appropriate for this patient. A patient with ARDS requires high PEEP to prevent atelectasis and hypoxemia. Therefore, a decrease in PEEP would worsen the hypoxemia. The plateau pressure should be kept at 30 cm H₂O or less. Therefore, this patient's plateau pressure is too high, and a decrease in the V_t may help decrease the plateau pressure.
22. **(D)** Sudden bubbling in the negative pressure chamber is an indication of a possible air leak; this should be reported to the physician. All of the other choices should be avoided when caring for a patient with a chest tube. Loops will increase pressure in the system. The collection chamber needs to be kept below the level of the chest at all times. Clamping the chest tube cuts off the negative pressure provided by the negative pressure chamber and could result in lung re-collapse.
23. **(B)** Due to the patient's decreased level of consciousness, the need for improved ventilation is a priority. NIV would address the hypoventilation, but it would not be considered safe for a patient with minimal responsiveness. Providing oxygen alone would not address the main problem (hypoventilation), which in this case is worse than the problem of hypoxemia. This patient

has respiratory acidosis and should not be treated with sodium bicarbonate.

24. **(A)** A depressed level of consciousness may be an indication of hypoventilation (respiratory acidosis) and a precursor to respiratory arrest for a patient with asthma exacerbation. Respiratory alkalosis and bilateral wheezing are common early manifestations during an asthma exacerbation. Mild hypoxemia should be treated with an increase in FiO_2 but is not considered ominous unless it is severe.
25. **(D)** Banked blood does not have normal 2,3-DPG. Low levels of 2,3-DPG shift the oxyhemoglobin-dissociation curve up to the left, which will decrease hemoglobin's release of oxygen at the tissue level. The remaining 3 choices—a fever, increased levels of 2,3-DPG, and acidosis—all shift the curve to the right, which increases the release of oxygen from hemoglobin.
26. **(B)** This clinical scenario is one of a pulmonary embolism. With the history of long bone fractures and the development of petechiae, a fat embolism is most likely. ARDS does not present with clear lungs. DVT does not always result in a PE. If it did, though, the PE would be due to a blood clot and petechiae would not be present. Delirium may result in agitation but not the other symptoms.
27. **(B)** The low pH indicates acidosis. The low bicarbonate indicates that the acidosis is metabolic, and the low PaCO_2 indicates that hyperventilation has started in order to compensate. The only option that causes metabolic acidosis is shock. Anxiety generally causes respiratory alkalosis. An opiate

drug overdose results in respiratory acidosis. An electrolyte imbalance (low chloride) causes metabolic alkalosis.

Answers to Practice ABGs

1. Respiratory alkalosis, no compensation
2. Respiratory acidosis, no compensation
3. Metabolic acidosis, no compensation
4. Respiratory acidosis, partial compensation
5. Metabolic alkalosis, no compensation
6. Respiratory acidosis, partial compensation
7. Metabolic alkalosis, partial compensation
8. Metabolic acidosis, partial compensation
9. Respiratory acidosis, no compensation
10. Metabolic alkalosis, no compensation
11. Mixed disorder: respiratory acidosis and metabolic alkalosis

