

# Caring for patients in respiratory failure

Even if you don't work in an ICU, you're likely to encounter patients in respiratory failure.

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**RESPIRATORY FAILURE** is one of the most common reasons for admission to the intensive care unit (ICU) and a common comorbidity in patients admitted for acute care. What's more, it's the leading cause of death from pneumonia and chronic obstructive pulmonary disease (COPD) in the United States. This article briefly reviews the physiologic components of respiration, differentiates the main types of respiratory failure, and discusses medical treatment and nursing care for patients with respiratory failure.

## Physiologic components of ventilation and respiration

The lung is highly elastic. Lung inflation results from the partial pressure of inhaled gases and the diffusion-pressure gradient of these gases across the alveolar-capillary membrane. The lungs play a passive role in breathing, but ventilation requires muscular effort. When the diaphragm contracts, the thoracic cavity enlarges, causing the lungs to inflate. During forced inspiration when a large volume of air is inspired, external intercostal muscles act as a second set of inspiratory muscles.

Accessory muscles in the neck and chest are the last group of inspiratory muscles, used only for deep and heavy breathing, such as during intense exercise or respiratory failure. During expiration, the diaphragm relaxes, decreasing thoracic cavity size and causing the

lungs to deflate. With normal breathing, expiration is purely passive. But with exercise or forced expiration, expiratory muscles (including the abdominal wall and internal intercostal muscles) become active. These important muscles are necessary for coughing.



## LEARNING OBJECTIVES

1. Discuss the physiology of ventilation and respiration.
2. Differentiate the types of respiratory failure.
3. Describe the treatment of respiratory failure.

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Respiration—the process of exchanging oxygen ( $O_2$ ) and carbon dioxide ( $CO_2$ )—involves ventilation, oxygenation, and gas transport; the ventilation/perfusion (V/Q) relationship; and control of breathing. Respiration is regulated by chemical and neural control systems, including the brainstem, peripheral and central chemoreceptors, and mechanoreceptors in skeletal muscle and joints. (See *Control of breathing*.)

A dynamic process, ventilation is affected by the respiratory rate (RR) and tidal volume—the amount of air inhaled and exhaled with each breath. Pulmonary ventilation refers to the total volume of air inspired or expired per minute.

Not all inspired air participates in gas exchange. Alveolar ventilation—the volume of air entering alveoli taking part in gas exchange—is the most important variable in gas exchange. Air that distributes to the conducting airways is deemed dead space or wasted air because it's not involved in gas exchange. (See *Oxygenation and gas transport*.)

Ultimately, effective ventilation is measured by the partial pressure of  $CO_2$  in arterial blood ( $Paco_2$ ). All expired  $CO_2$  comes from alveolar gas. During normal breathing, the breathing rate or depth adjusts to maintain a steady  $Paco_2$  between 35 and 45 mm Hg. Hyperventilation manifests as a low  $Paco_2$ ; hypoventilation, as a high



PaCO<sub>2</sub>. During exercise or certain disease states, increasing breathing depth is far more effective than increasing the RR in improving alveolar ventilation.

### Lung recoil and compliance

The lungs, airways, and vascular trees are embedded in elastic tissue. To inflate, the lung must stretch to overcome these elastic components. *Elastic recoil*—the lung's ability to return to its original shape after stretching from inhalation—relates inversely to *compliance*. Lung compliance indirectly reflects lung stiffness or resistance to stretch. A stiff lung, as in pulmonary fibrosis, is less compliant than a normal lung.

With reduced compliance, more work is required to produce a normal tidal volume. With extremely high compliance, as in emphysema where there is loss of alveolar and elastic tissue, the lungs inflate extremely easily. Someone with emphysema must expend a lot of effort to get air out of the lungs because they don't recoil back to their normal position during expiration. In both pulmonary fibrosis and emphysema, inadequate lung ventilation leads to hypercapnic respiratory failure.

### Respiratory failure

Respiratory failure occurs when one of the gas-exchange functions—oxygenation or CO<sub>2</sub> elimination—fails. A wide range of conditions can lead to acute respiratory failure, including drug overdose, respiratory infection, and exacerbation of chronic respiratory or cardiac disease.

Respiratory failure may be acute or chronic. In *acute* failure, life-threatening derangements in arterial blood gases (ABGs) and acid-base status occur, and patients may need immediate intubation. Respiratory failure also may be classified as hypoxemic or hypercapnic.

## Control of breathing

Breathing is controlled by carefully integrated neurologic, chemical, and mechanical processes. The neurologic respiratory center in the brain's medulla is sensitive to carbon dioxide (CO<sub>2</sub>) and hydrogen-ion concentrations in body fluids. It can compensate to changing conditions within seconds to minutes.

- *Acidemia* (decreased blood pH) or elevated partial pressure of arterial CO<sub>2</sub> stimulates the respiratory center, which increases respiratory rate or depth. This, in turn, reduces the CO<sub>2</sub> level and increases blood pH.
- *Alkalemia* (increased blood pH) inhibits the respiratory center, resulting in slower or more shallow breathing, CO<sub>2</sub> retention, and reduced blood pH.

Usually, the lungs can compensate for respiratory and metabolic imbalances. But when lung dysfunction is severe or the metabolic imbalance is prolonged, the lungs can't fully compensate and renal compensation is required. However, renal compensation is slow, taking hours to days.

Interruption of or damage to respiratory structures or respiratory control mechanism ultimately affects the ability to exchange gases effectively and can lead to such disorders as chronic obstructive pulmonary disease, interstitial lung disease, and respiratory failure.

Clinical indicators of acute respiratory failure include:

- partial pressure of arterial oxygen (PaO<sub>2</sub>) below 60 mm Hg, or arterial oxygen saturation as measured by pulse oximetry (SpO<sub>2</sub>) below 91% on room air
- PaCO<sub>2</sub> above 50 mm Hg and pH below 7.35
- PaO<sub>2</sub> decrease or PaCO<sub>2</sub> increase of 10 mm Hg from baseline in patients with chronic lung disease (who tend to have higher PaCO<sub>2</sub> and lower PaO<sub>2</sub> baseline values than other patients).

In contrast, *chronic* respiratory failure is a long-term condition that develops over time, such as with COPD. Manifestations of chronic respiratory failure are less dramatic and less apparent than those of acute failure.

### Three main types of respiratory failure

The most common type of respiratory failure is *type 1*, or hypoxemic respiratory failure (failure to exchange oxygen), indicated by a PaO<sub>2</sub> value below 60 mm Hg with a normal or low PaCO<sub>2</sub> value. In ICU patients, the most common causes of type 1 respiratory failure are V/Q mismatching and shunts.

COPD exacerbation is a classic example of V/Q mismatching. Shunting, which occurs in virtually all acute lung diseases, involves alveolar collapse or fluid-filled alveoli. Examples of type 1 respiratory failure include pulmonary edema (both cardiogenic and noncardiogenic), pneumonia, influenza, and pulmonary hemorrhage. (See *Ventilation and perfusion: A critical relationship*.)

*Type 2*, or hypercapnic, respiratory failure, is defined as failure to exchange or remove CO<sub>2</sub>, indicated by PaCO<sub>2</sub> above 50 mm Hg. Patients with type 2 respiratory failure who are breathing room air commonly have hypoxemia. Blood pH depends on the bicarbonate level, which is influenced by hypercapnia duration. Any disease that affects alveolar ventilation can result in type 2 respiratory failure. Common causes include severe airway disorders (such as COPD), drug overdose, chest-wall abnormalities, and neuromuscular disease.

*Type 3* respiratory failure (also called perioperative respiratory failure) is a subtype of type 1 and results from lung or alveolar atelectasis. General anesthesia can cause collapse of dependent lung

## Oxygenation and gas transport

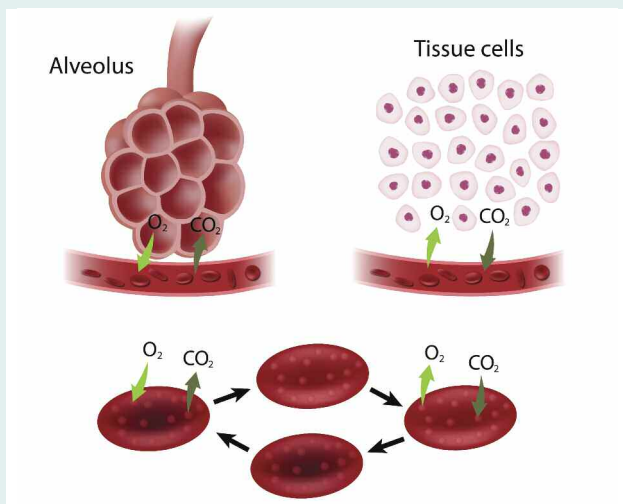
A gas consists of individual molecules in constant random motion. These molecules bombard the walls of any vessel containing them, exerting pressure against the wall. The pressure exerted is proportional to gas temperature and concentration.

### How gas exchange occurs

Movement of gases—oxygen ( $O_2$ ) and carbon dioxide ( $CO_2$ )—into and out of the alveoli and cells occurs by simple diffusion, defined as passive movement of molecules from an area of higher concentration to one of lower concentration. The distance across the capillary wall and the lubricating plasma layer measures about 1 micron—roughly 100 times smaller than a single human hair. This short distance allows quick, effective exchange of  $O_2$  and  $CO_2$  across the alveolar-capillary membrane.

Hemoglobin (Hgb) transports  $O_2$  from the lungs to the tissues. Every 100 mL of blood that passes through the tissues delivers about 4 mL of  $O_2$ . Where  $O_2$  pressure is high, oxygen combines loosely with the heme portion of hemoglobin in the lung, forming oxyhemoglobin. When oxyhemoglobin reaches the tissues, where  $O_2$  pressure is low, hemoglobin releases  $O_2$ .

The illustration below shows the basic process of gas exchange.



The oxyhemoglobin curve depicts  $O_2$  binding to Hgb in the lung and  $O_2$  unloading in the tissues. Body temperature, blood acidity, and underlying conditions, such as anemia or chronic hypoxia, may influence the efficiency of this process.

### Factors affecting gas diffusion across the respiratory membrane

The following factors affect the rate of gas diffusion across the respiratory (alveolar-capillary) membrane.

- **Membrane thickness.** The diffusion rate is inversely proportional to the thickness of the respiratory membrane. Such conditions as pulmonary edema, excessive sputum, and fibrosis thicken the membrane, lengthening the distance required to cross the membrane.
- **Surface area.** The lung's large surface area is influenced by alveolar size and inflation. Surface area is directly proportional to diffusion; a large surface area favors diffusion. Clinical conditions that reduce surface area include atelectasis and such procedures as lobectomy.
- **Gas solubility and molecular weight.** Each gas has an inherent solubility and molecular weight. The diffusion rate of a gas is directly proportional to its solubility and inversely proportional to its molecular weight. Although  $CO_2$  weighs more than  $O_2$ , it's more soluble and diffuses 20 times faster. Damage to the respiratory membrane impairs  $O_2$  diffusion capacity but may not affect  $CO_2$  diffusion. Therefore, in disease states, the partial pressure of  $O_2$  in arterial blood ( $PaO_2$ ) decreases before the partial pressure of  $CO_2$  in arterial blood ( $PaCO_2$ ) changes. So patients with pulmonary diseases that interfere with diffusion develop problems related to hypoxemia before they start retaining  $CO_2$ .
- **Driving pressure.** In the lung, driving pressure is the gradient between  $PaO_2$  or  $PaCO_2$  in the alveoli and the pressure of these gases in the blood. In the tissues, driving pressure is the difference between  $PaO_2$  and  $PaCO_2$  in the capillaries compared to pressures in tissue cells. In the alveolus,  $O_2$  driving pressure is 64 mm Hg;  $CO_2$  driving pressure is only 5 mm Hg. Because  $CO_2$  is more soluble, it doesn't need a high driving pressure. Administering supplemental  $O_2$  increases driving pressure.

alveoli. Patients most at risk for type 3 respiratory failure are those with chronic lung conditions, excessive airway secretions, obesity, immobility, and tobacco use, as well as those who've had surgery involving the upper abdomen. Type 3 respiratory failure also may occur in patients experiencing shock, from hypoperfusion of respiratory muscles. Normally, less than 5% of total cardiac output flows to respiratory muscles. But in pulmonary edema, lactic acidosis, and anemia (conditions that commonly arise during shock),

up to 40% of cardiac output may flow to the respiratory muscles.

### Signs and symptoms of respiratory failure

Patients with impending respiratory failure typically develop shortness of breath and mental-status changes, which may present as anxiety, tachypnea, and decreased  $SpO_2$  despite increasing amounts of supplemental oxygen.

Acute respiratory failure may cause tachycardia and tachypnea. Other signs and symptoms include periorbital or circumoral cyanosis,

diaphoresis, accessory muscle use, diminished lung sounds, inability to speak in full sentences, an impending sense of doom, and an altered mental status. The patient may assume the tripod position in an attempt to further expand the chest during the inspiratory phase of respiration. In chronic respiratory failure, the only consistent clinical indicator is protracted shortness of breath.

Be aware that pulse oximetry measures the percentage of hemoglobin saturated with oxygen, but it doesn't give information about

## Ventilation and perfusion: A critical relationship

For adequate organ and tissue oxygenation, ventilation ( $V$ , the amount of air reaching the alveoli) should match perfusion ( $Q$ , the amount of blood reaching the alveoli). Although ventilation and perfusion to the lungs aren't distributed uniformly, they tend to match: Where ventilation is maximal, blood flow is maximal, and vice versa. The  $V/Q$  ratio reflects the matching of these two components.

Because air rises, alveoli in the apex of each lung are always partially inflated and thus don't accommodate much further ventilation. The lung bases, on the other hand, have a much lower alveolar pressure and ventilate more readily than the apices. The low pulmonary arterial pressure is just enough to pump blood to the top of the lung. Although all parts of the lung receive some perfusion, hydrostatic pressure in the vasculature directs most of the blood flow through the lower (dependent) portion of the lung. So when a person sits or stands, blood flow to the lung bases exceeds blood flow to the apices.

### Balancing ventilation and perfusion

The lung uses hypoxic vasoconstriction to balance ventilation and perfusion. When a lung region becomes hypoxic, small arteries feeding that region sense hypoxia in alveolar gas and constrict in response. Constriction redirects blood away from the hypoxic region to better-oxygenated lung areas. When a  $V/Q$  mismatch or imbalance occurs, as from lung disease, hypoxemia

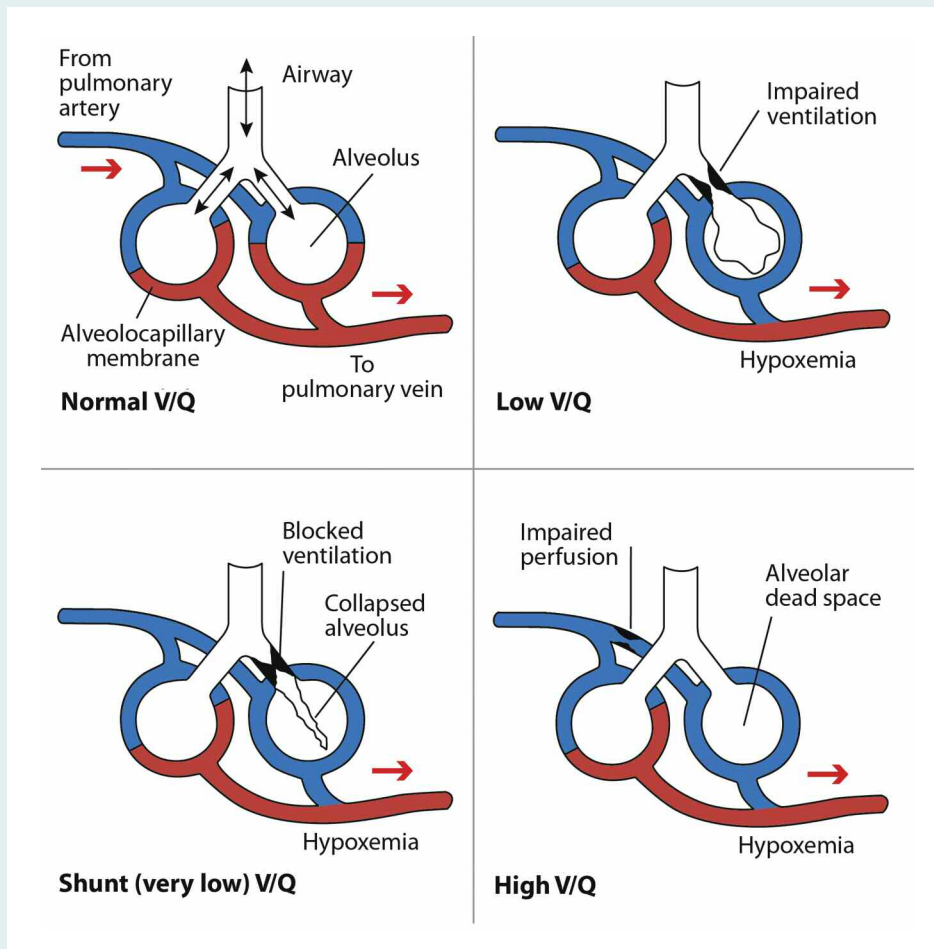
may result. Causes of extreme  $V/Q$  mismatching include:

- shunting, in which alveolar perfusion is normal but ventilation is absent
- dead space—portions of the respiratory tract where air doesn't participate in gas exchange
- nonperfused lung areas.

A low  $V/Q$  ratio occurs when pulmonary gas exchange is impaired—a situation that typically causes hypoxemia. Carbon dioxide ( $\text{CO}_2$ ) excretion also is impaired, which can lead to increased partial pressure of  $\text{CO}_2$  in arterial blood ( $\text{PaCO}_2$ ). However, this is rare because increased  $\text{PaCO}_2$  typically triggers respiratory stimulation and increased alveolar ventilation, which returns  $\text{PaCO}_2$  to normal. This scenario is most common in patients with chronic bronchitis, asthma, and acute pulmonary edema.

On the other hand, a high  $V/Q$  ratio occurs when a well-ventilated lung area is poorly perfused; ventilation is wasted as it fails to oxygenate the blood. The most common cause is pulmonary embolism, which impairs blood flow. A high  $V/Q$  ratio also may occur in emphysema due to air trapping.

These illustrations show what happens with a normal  $V/Q$  ratio, a low  $V/Q$  ratio, a very low  $V/Q$  ratio (shunt), and a high  $V/Q$  ratio.



## To detect changes in respiratory status early, assess the patient's tissue oxygenation status regularly.

oxygen delivery to the tissues or the patient's ventilatory function. So be sure to consider the patient's entire clinical presentation. Compared to SpO<sub>2</sub>, an ABG study provides more accurate information on acid-base balance and blood oxygen saturation. Capnography is another tool used for monitoring patients receiving anesthesia and in critical care units to assess a patient's respiratory status. It directly monitors inhaled and exhaled concentration of CO<sub>2</sub> and indirectly monitors PaCO<sub>2</sub>.

### Treatment and management

In acute respiratory failure, the healthcare team treats the underlying cause while supporting the patient's respiratory status with supplemental oxygen, mechanical ventilation, and oxygen saturation monitoring. Treatment of the underlying cause, such as pneumonia, COPD, or heart failure, may require diligent administration of antibiotics, diuretics, steroids, nebulizer treatments, and supplemental O<sub>2</sub> as appropriate.

For chronic respiratory failure, despite the wide range of chronic or end-stage pathology present (such as COPD, heart failure, or systemic lupus erythematosus with lung involvement), the mainstay of treatment is continuous supplemental O<sub>2</sub>, along with treatment of the underlying cause.

### Nursing care

Nursing care can have a tremendous impact in improving efficiency of the patient's respiration and ventilation and increasing the chance for recovery. To detect changes in respiratory status early, assess the patient's tissue oxygenation status regularly. Evaluate ABG results and indices of end-organ perfusion. Keep in mind that the brain is extremely sensitive to O<sub>2</sub> supply; decreased O<sub>2</sub> can lead to an altered mental status. Also, know that angina signals inadequate

coronary artery perfusion. In addition, stay alert for conditions that can impair O<sub>2</sub> delivery, such as elevated temperature, anemia, impaired cardiac output, acidosis, and sepsis.

As indicated, take steps to improve V/Q matching, which is crucial for improving respiratory efficiency. To enhance V/Q matching, turn the patient on a regular and timely basis to rotate and maximize lung zones. Because blood flow and ventilation are distributed preferentially to dependent lung zones, V/Q is maximized on the side on which the patient is lying.

Regular, effective use of incentive spirometry helps maximize diffusion and alveolar surface area and can help prevent atelectasis. Regular rotation of V/Q lung zones by patient turning and repositioning enhances diffusion by promoting a healthy, well-perfused alveolar surface. These actions, as well as suctioning, help mobilize sputum or secretions.

### Nutritional support

Patients in respiratory failure have unique nutritional needs and considerations. Those with acute respiratory failure from primary lung disease may be malnourished initially or may become malnourished from increased metabolic demands or inadequate nutritional intake. Malnutrition can impair the function of respiratory muscles, reduce ventilatory drive, and decrease lung

defense mechanisms. Clinicians should consider nutritional support and individualize such support to ensure adequate caloric and protein intake to meet the patient's respiratory needs.

### Patient and family education

Provide appropriate education to the patient and family to promote adherence with treatment and help prevent the need for readmission. Explain the purpose of nursing measures, such as turning and incentive spirometry, as well as medications. At discharge, teach patients about pertinent risk factors for their specific respiratory condition, when to return to the healthcare provider for follow-up care, and home measures they can take to promote and maximize respiratory function. ★

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**PURPOSE/GOAL**

To provide nurses with information on how to better care for patients in respiratory failure.

**LEARNING OBJECTIVES**

1. Discuss the physiology of ventilation and respiration.
2. Differentiate the types of respiratory failure.
3. Describe the treatment of respiratory failure.

**Please mark the correct answer online.**

**1. Which statement about the respiratory center's response to changes in carbon dioxide (CO<sub>2</sub>) and hydrogen-ion concentration is correct?**

- a. The respiratory center can compensate for changes in hours or days.
- b. The respiratory center can compensate for changes in seconds or minutes.
- c. Alkalemia stimulates the respiratory center.
- d. Acidemia inhibits the respiratory center.

**2. The most important variable in gas exchange is:**

- a. amount of wasted air.
- b. amount of dead space.
- c. bronchial ventilation.
- d. alveolar ventilation.

**3. Which statement about lung compliance is correct?**

- a. Compliance is not related to the work it takes to breathe.
- b. High compliance is associated with less work required to breathe.
- c. Elastic recoil relates inversely to compliance.
- d. Elastic recoil relates directly to compliance.

**4. Which of the following clinical findings indicate acute respiratory failure?**

- a. Partial pressure of CO<sub>2</sub> in arterial blood (PaCO<sub>2</sub>) above 50 mm Hg and pH below 7.35
- b. PaCO<sub>2</sub> below 50 mm Hg and pH above 7.35
- c. Partial pressure of arterial oxygen (PaO<sub>2</sub>) increase of 10 mm Hg from baseline in patients with chronic lung disease
- d. PaCO<sub>2</sub> decrease of 10 mm Hg from baseline in patients with chronic lung disease

**5. Which statement about ventilation and perfusion is correct?**

- a. When a lung region becomes hypoxic, small arteries constrict in response.
- b. When a lung region becomes hypoxic, small arteries dilate in response.
- c. A high ventilation/perfusion (V/Q) ratio occurs when pulmonary gas exchange is impaired.
- d. A low V/Q ratio occurs when a well-ventilated lung area is poorly perfused.

**6. In which type of respiratory failure do patients who are breathing room air commonly have hypoxemia?**

- a. Type 1
- b. Type 2
- c. Type 3
- d. Type 4

**7. Shunting and V/Q mismatching are common causes of which type of respiratory failure?**

- a. Postoperative
- b. Perioperative
- c. Hypercapnic
- d. Hypoxemic

**8. Which type of respiratory failure results from lung or alveolar atelectasis?**

- a. Postoperative
- b. Perioperative
- c. Hypercapnic
- d. Hypoxemic

**9. Signs and symptoms of impending respiratory failure include:**

- a. bradypnea.
- b. tachypnea.
- c. bradycardia.
- d. increased SpO<sub>2</sub>.

**10. Which statement about pulmonary care for patients with respiratory failure is accurate?**

- a. V/Q matching is minimized on the side on which the patient is lying.
- b. Incentive spirometry should be used sparingly.
- c. Regular turning helps improve V/Q matching.
- d. Slight decreases in temperature can impair O<sub>2</sub> delivery.

**11. Which statement about the nutritional needs of patients with respiratory failure is correct?**

- a. Malnutrition can increase ventilatory drive.
- b. Malnourishment typically is not a problem.
- c. Nutritional support requirements are universal.
- d. Increased metabolic demands can cause malnourishment.