CASE 15

A 34-year-old woman with diabetes presents to the emergency department with complaints of fever, chills, back pain, dizziness, and shortness of breath. She reports a new-onset nonproductive cough and denies having chest pain. She reports no sick contacts. On examination, she is ill-appearing, febrile, hypotensive, and tachycardic. She has marked right costovertebral (flank) tenderness. Her lung examination demonstrates course rales and rhonchi throughout both lung fields. Her heart rate is tachycardic, but with a regular rhythm. Her oxygen saturation on room air is very low at 80% (normal > 94%). Urinalysis reveals numerous bacteria and leukocytes, consistent with a urinary tract infection. She is diagnosed with pyelonephritis and septic shock and has evidence of adult respiratory distress syndrome (ARDS) with bilateral pulmonary infiltrates on chest x-ray. The emergency room physician explains to the patient that pulmonary injury has led to leaky pulmonary capillaries.

◆ How does pulmonary capillary leakage cause hypoxia?

◆ After a patient takes a normal breath and exhales, what lung volume remains?

◆ How do obstructive lung diseases such as asthma affect forced expiratory volume?
ANSWERS TO CASE 15: PULMONARY STRUCTURE AND LUNG CAPACITIES

Summary: A 34-year-old diabetic woman has pyelonephritis, septic shock, and ARDS.

◆ Pulmonary capillary leakage and hypoxia: Accumulation of excess fluid outside the capillaries leads to altered local ventilation and perfusion and makes gas exchange inefficient.

◆ Lung volume remaining after normal breath: Functional residual capacity (FRC; cannot be measured with spirometry alone).

◆ Forced expiratory volume with obstructive airway disease: Decreased.

CLINICAL CORRELATION

Knowledge of respiratory physiology is essential for understanding, diagnosing, and treating many medical conditions. When a patient presents with hypoxia, there may be many different etiologies, but there are two general categories: diseases that affect perfusion of the lungs (pulmonary embolus) and diseases of the bronchial tree (eg, pneumonia, pneumothorax, ARDS, pulmonary edema, asthma, bronchitis). A patient’s history and physical examination will help determine the cause of most disorders. A chest x-ray and other radiologic studies may help as well. A patient with pulmonary difficulties may undergo pulmonary function tests to measure and test many of the lung volumes, capacities, and flows. For example, patients with a decrease in forced expiratory volume could have an obstructive lung disorder such as asthma. When the normal balance of reabsorption and lymphatic drainage is overwhelmed by the filtered fluid, fluid will accumulate in the interstitium. The edema can be hydrostatic, which may occur with fluid overload or congestive heart failure, or may be protein-rich, as with ARDS. ARDS is characterized by increased permeability of the capillary endothelium, resulting in a protein-rich fluid collection.

APPROACH TO PULMONARY PHYSIOLOGY

Objectives

1. Diagram lung volumes and capacities.
2. Describe the anatomy of the pulmonary tree.
3. Discuss pulmonary blood flow and regulation.
Definitions

**Dead space:** The volume of airways and of alveoli in which gas exchange does not take place.

**FRC:** The volume of air in the lungs when all the muscles of respiration are relaxed.

**Vital capacity:** The volume of air that can be expelled from the lungs during a forced expiration that starts at total lung capacity (TLC).

**FEV₁:** The volume of air expelled from the lungs during the first second of a forced expiration that starts at TLC.

**DISCUSSION**

The **exchange of oxygen and carbon dioxide** necessary for metabolism requires that **air and blood be brought into close contact** over a large surface area. This is accomplished by **matching ventilation** of the approximately **300 million alveoli** that constitute the lungs with blood flow to those alveoli. Neither the distribution of alveolar ventilation nor the distribution of blood flow is uniform within the lung; certain regions receive more of both, whereas other regions receive less of both. Normally, however, ventilation and flow are matched to provide optimal gas exchange.

Ventilation of alveoli is accomplished by the regulated movement of air into and out of the lung. During normal breathing while a person is at rest, a fairly constant volume of air moves in and out of the alveoli and airways with each breath. This is called the **tidal volume** and can be measured with a device called a **spirometer**. There are several kinds of spirometers, but all are able to monitor airflow without adding extra resistance or causing significant changes in air composition. In addition to tidal volume measurements, several other lung volumes and lung capacities can be measured with a spirometer (Figure 15-1).

If one makes a normal inspiration and then continues to inhale as much air as possible, the additional air inspired after the tidal volume is the **inspiratory reserve volume**. In a similar manner, the **expiratory reserve volume** is the volume of air exhaled if as much air as possible is exhaled after the exhalation of a normal tidal volume. The **vital capacity (VC)**, which can be determined by inhaling as much as possible and then exhaling as much as possible, is composed of the **tidal volume** and the **inspiratory and expiratory reserve volumes**.

Although spirometry can provide much useful information, **residual volume (RV)**, **functional residual capacity (FRC)**, and **total lung capacity (TLC)** cannot be measured using spirometry alone. However, FRC can be determined by rebreathing air containing a known volume and concentration of an inert gas that is not readily absorbed and then determining its concentration in the expired gas at the end of the exhalation of a normal tidal volume. If one knows the initial volume and concentration of inert gas inhaled and the concentration of the inert gas at equilibrium, FRC can be calculated. With knowledge of this capacity, both RV and TLC also can be calculated.
Measuring lung volumes can provide much information. For example, FRC, which is the **volume of air in the lungs when no air is moving and the muscles of respiration are relaxed**, is determined by the balance of lung elastic recoil and chest wall elastic recoil. Thus, changes in tissue composition of the lung (eg, fibrosis) or chest wall will affect FRC.

Combining lung volume measurements with airflow measurements can provide information about resistance to airflow. If a person inhales to TLC and then exhales as forcefully and completely as possible, the **forced expired volume during the first second of expiration (FEV₁)** and the **forced vital capacity (FVC)** can be determined. The ratio FEV₁/FVC provides information about resistance to airflow. Normally, about 80% of the FVC will be exhaled during the first second. If resistance is increased, the percentage exhaled during the first second will be decreased. Resistance to airflow lies almost completely in the **airways** and is a function mainly of airway radius. A small decrease in radius will lead to a large increase in resistance because resistance to flow through a tube is inversely proportional to the fourth power of its radius. Airway radius can be affected in a number of ways. Many airways are enveloped in smooth muscle whose contraction can decrease radius. Many airways have **mucus**-secreting cells and glands; increased or abnormally thick mucus secretion can occlude airways partially. Inflammation and edema also can reduce airway radius.

**Figure 15-1.** Pulmonary spirometry. Lung volumes in milliliters are depicted.
As a result of the structure of the lung, not all the inspired air takes part in gas exchange even in a normal individual. Starting with the trachea, which branches into the right and left bronchi, each lung is composed of a set of branching airways that increase in number and decrease in radius with each branching. The airways that constitute the final seven or so branches have outpouches or alveoli, and those forming the final branching end blindly, feeding millions of alveoli. There is just one set of airways leading to and from the alveoli; thus, not all the air inhaled reaches the alveoli and not all the gas leaving the alveoli during expiration is expelled into the air. Because exchange of gas between the air and the blood occurs only within alveoli, the volume of air remaining in the airways at the end of inspiration is called dead space volume. In a normal individual with a tidal volume of 500 mL, the dead space volume is around 150 mL.

The pulmonary circulation delivers blood to and from the lungs. The main pulmonary artery divides to the right and left to supply each lung. Each of these arteries branches to form smaller, more numerous arteries, which themselves branch. Successive branching continues and provides capillaries to each alveolus and to many of the airways. The capillaries then coalesce into venules, which coalesce into larger veins and finally into the pulmonary vein, which returns oxygenated blood back to the heart to be pumped through the systemic circulation. Although blood flows through the pulmonary and systemic circulations are equal, pressures and resistances in the pulmonary circulation are about one-tenth of those in the systemic circulation. In fact, in a person who is standing or sitting upright, pulmonary arterial pressure is not sufficient to perfuse the tops of the lungs as well as other regions. Also, resistance to flow in the pulmonary circulation, in contrast to resistance in the systemic circulation, depends as much if not more on the diameter and number of capillaries that are open as on the arteriolar radius.

Optimal gas exchange requires a thin alveolar-capillary interface. As in the systemic circulation, there is a net filtration of fluid out of the capillaries into the interstitial space. This fluid normally flows along the surface and is carried away in the lymphatic vessels in the walls of the small airways. However, during inflammatory conditions such as the one encountered in this case and in conditions in which pulmonary capillary hydrostatic pressure is increased, such as left-sided heart failure, capillary filtration exceeds lymphatic flow and pulmonary edema occurs. The edema fluid fills alveoli, disrupting ventilation and gas exchange.
COMPREHENSION QUESTIONS

[15.1] In a 58-year-old woman with difficulty breathing, the TLC and FRC are lower than normal and FEV₁/FVC is slightly higher than normal. These findings are most consistent with which of the following?

A. Decreased pulmonary blood flow
B. Decreased strength of the chest wall muscles
C. Increased airway resistance
D. Increased chest wall elastic recoil
E. Increased lung elastic recoil

[15.2] A patient has reduced TLC and increased RV. FRC is normal. These findings are most consistent with which of the following?

A. Decreased pulmonary blood flow
B. Decreased strength of the muscles of respiration
C. Increased airway resistance
D. Increased chest wall elastic recoil
E. Increased lung elastic recoil

[15.3] A chest x-ray of a patient with left-sided heart failure indicates pulmonary edema. Additional examination probably would reveal which of the following?

A. Decreased pulmonary artery pressure
B. Decreased pulmonary lymph flow
C. Increased pulmonary venous pressure
D. Normal arterial oxygen partial pressure
E. Normal vital capacity

Answers

[15.1] E. A lung with increased elastic recoil (decreased compliance) will be harder to fill on inspiration and will tend to pull the chest wall inward on relaxation of the muscles of breathing. Thus, both TLC and FRC will be decreased. Because airway radius is normal or even increased, FEV₁ normalized to FVC will be normal or increased even though FVC will be reduced. Decreased muscle strength could cause a decrease in TLC, but it would not alter FRC.

[15.2] B. If the muscles of inspiration are weak, lungs cannot be inflated as well, thus reducing the inspiratory reserve volume and TLC. If the muscles of expiration are weak, not as much air can be forced from the lungs and expiratory reserve volume will be decreased, thus increasing RV. Increases in elastic recoil of either the chest wall or the lungs and increases in airway resistance will alter TLC and/or FRC.
As a result of the decrease in myocardial contractility, end diastolic pressure in the left ventricle increases, leading to an increase in the pulmonary venous and pulmonary capillary pressures. The increased pulmonary capillary hydrostatic pressure leads to increased pulmonary capillary filtration, and when filtration exceeds lymph flow, pulmonary edema develops. Pulmonary artery pressure is likely to be increased in this condition. The edema interferes with gas exchange and with lung inflation; thus, arterial oxygen partial pressure and vital capacity will be decreased.

**PHYSIOLOGY PEARLS**

- Changes in FRC point toward changes in lung and chest wall structure such as those caused by pulmonary fibrosis.
- Decreases in forced airflow, especially normalized to FVC, point toward increased airway resistance such as that seen in asthma, bronchitis, and emphysema.
- Pulmonary edema results in a ventilation-perfusion inequality and a decrease in diffusion of oxygen, leading to a decrease in oxygen transfer and arterial oxygen partial pressure.
- In a normal person who is standing or sitting upright, both blood flow and alveolar ventilation are lowest at the top of the lung and greatest near the bottom of the lung.

**REFERENCES**


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