## **Instructor's Answer Key**

## **Chapter 16: Respiratory Physiology**

## **Answers to Test Your Understanding of Concepts and Principles**

- Contraction of the diaphragm → lowering of diaphragm → increase in volume of lungs → decrease in intrapulmonary pressure (Boyle's Law) → air enters lungs. [Note: This question is also answered in the Student Study Guide.]
- 2. The lungs are normally stuck to the wall of the thorax (the visceral and parietal pleural membranes are against each other). In this state, the lungs are stretched and exert an inward elastic tension against the rib cage. When a pneumothorax occurs air enters the intrapleural space, the lungs pull away from the thoracic wall and collapse. This relieves the elastic tension that was exerted by the lungs on the rib cage. The ribs, as a result of their own elastic recoil, thus become expanded and spaced farther apart.
- 3. A rise in the  $P_{CO2} \rightarrow$  fall in blood pH  $\rightarrow$  stimulation of peripheral chemoreceptors. Also, the rise in arterial  $P_{CO2} \rightarrow$  fall in pH of cerebrospinal fluid  $\rightarrow$  stimulation of central chemoreceptors. Both peripheral and central chemoreceptors  $\rightarrow$  stimulation of respiratory control center in the medulla  $\rightarrow$  increased ventilation.
- 4. Ketoacidosis is a type of metabolic acidosis; and the fall in arterial pH thus produced generates action potentials from the aortic and carotid bodies that stimulate the medullary respiratory center neurons to increase total minute volume. Since the increased ventilation was not caused initially by a rise in blood P<sub>CO2</sub>, this increased ventilation is excessive (hyperventilation) and causes an abnormally low arterial P<sub>CO2</sub> to be produced. Administration of bicarbonate can buffer the excessive H<sup>+</sup> released from the ketoacids and thus help to return the blood pH to normal. As this occurs, the hyperventilation will cease.
- (a) Red blood cell counts, hematocrit, and hemoglobin concentration measurements.
  (b) Percent carboxyhemoglobin saturation.
  (c) Arterial P<sub>CO2</sub> and arterial percent oxyhemoglobin saturation.
- 6. In *hyperventilation* the arterial P<sub>CO2</sub> is decreased. Therefore, a lower-than-normal carbonic acid concentration will cause the blood pH to rise. With less carbonic acid, there will be less free bicarbonate in the blood, since bicarbonate is derived from carbonic acid. In *hypoventilation*, conversely, the arterial P<sub>CO2</sub> is increased, carbonic acid concentration is increased, blood pH falls, and free bicarbonate concentration is increased.
- 7. Upon exercise, the rate and depth of breathing increase to produce a total minute volume that is many times the resting value. This increase is matched by an increase in oxygen consumption and carbon dioxide production by the exercising muscles. Because of the match between demand and supply, the arterial blood P<sub>O2</sub>, pH, and P<sub>CO2</sub> remain constant during exercise.

- 8. An increase in the red blood cell content of 2,3-DPG lowers the affinity of hemoglobin for oxygen, and thus produces increased oxygen unloading. At a given P<sub>O2</sub> in the tissue capillaries, more oxygen carried to the tissues in the arteries will be unloaded, so that less oxygen remains in the venous blood draining the tissues. The P<sub>O2</sub> of venous blood will thus be decreased.
- 9. When a person goes from sea level to a high altitude the P<sub>O2</sub> of arterial blood falls. The decreased P<sub>O2</sub> stimulates hyperventilation (hypoxic ventilatory response). Hyperventilation increases tidal volume, thus reducing the proportionate contribution of air from the anatomical dead space and increasing the proportion of fresh air brought to the alveoli. As part of the compensatory response, the levels of hemoglobin concentration also increase at high elevation, as does the number of red blood cells (rise in hematocrit). However on the negative side, polycythemia leads to increases the viscosity of blood, which can lead to pulmonary hypertension, ventricular hypertrophy, and heart failure.
- 10. When a person goes from sea level to a high altitude the P<sub>O2</sub> of arterial blood falls. Also, the affinity of hemoglobin for oxygen is reduced, so that a higher proportion of oxygen is unloaded. The percent oxyhemoglobin and thus the oxygen content of the blood falls. Furthermore, the rate at which oxygen can be delivered to the cells after release from hemoglobin is reduced at high altitude. This is because the maximum concentration of oxygen that can be dissolved in the plasma decreases in a linear fashion with the fall in P<sub>O2</sub> of arterial blood. The hypoxic ventilatory response produces hyperventilation that lowers the arterial P<sub>CO2</sub> and produces a respiratory alkalosis that helps to blunt the hyperventilation. Furthermore, there is evidence that NO produced in the lungs may be carried by the amino acid cysteine in hemoglobin to distant vessels. There, the lower oxygen concentration favors the unloading of NO, which may then produce vasodilation and increased blood flow. NO may be transferred from the blood to the respiratory control center, where it may stimulate breathing and contribute to the hypoxic drive at high altitudes.
- 11. Asthma is characterized as an obstructive disorder in which inflammation, mucous secretion, and bronchoconstriction cause obstruction of airflow through the bronchioles. Vital capacity is normal, but FEV is decreased because of the difficulty in moving air through the air passages. Emphysema is a chronic, progressive restrictive disorder in which alveolar tissue is destroyed, resulting in fewer but larger alveoli. This reduces the surface area for gas exchange. The loss of alveoli reduces the lateral tension exerted on the bronchiolar walls and thereby reduces the ability of bronchioles to remain open during expiration. Vital capacity decreases as air trapping occurs, but FEV tests are normal.
- 12. A quiet inspiration results from contraction of the diaphragm, the parasternal intercostals, and probably also by the external intercostal. Other muscles become involved in a forced inspiration including the scalenus, pectoralis minor, and the sternocleidomastoid. Quiet expiration is a passive process in which muscles relax and the lungs and thorax recoil. During a forced expiration, the internal intercostals contract and depress the rib cage, and the abdominal muscles decrease the volume of the thorax.

13. In late fetal life surfactant molecules are secreted into the alveoli by type II alveolar cells. Surfactant consists of phospholipids – primarily phosphatidylcholine and phosphatidylglycerol – together with hydrophobic surfactant proteins. Surfactant functions to lower surface tension of fluids lining the alveoli, thus helping to prevent the alveoli from collapsing during expiration. In the absence of surfactant, alveoli will collapse under surface tension during expiration. This condition is called *respiratory distress syndrome* (RDS). Babies with this condition can be saved by mechanical ventilators and by exogenous surfactant delivered to the baby's lungs by means of an endotracheal tube until the baby's lungs mature and can manufacture sufficient surfactant.

## Answers to Test Your Ability to Analyze and Apply Your Knowledge

- 1. Emphysema is a chronic, progressive destruction of alveolar tissue, resulting in fewer but larger alveoli. The weakened bronchioles have difficulty remaining open during exhalation and often collapse, trapping extra air in the alveoli. The percussion sounds on the chest therefore would result in a hyperresonance, or booming as trapped air in the destroyed alveolar tissue and collapsed bronchioles amplify the tapping. The presence of air surrounding the collapsed lung would also cause a hyperresonance percussion sound when compared to the more solid sound of the healthy side. The percussion sounds in a lung partially filled with fluid would sound much more muffled, or solid sounding over the area of fluid but would change to a more hollow sound over the area above the collapsed lung.
- 2. The neonate's first breath is the most difficult because the newborn must overcome great alveolar surface tension forces in order to inflate partially collapsed alveoli for the first time. The transpulmonary pressure required for the first breath is fifteen to twenty times that required for subsequent breaths. Premature infants often require a mechanical ventilator to keep their lungs inflated because they may lack adequate amounts of surfactant in the lining of the alveoli, and as a result their alveoli are collapsed (law of Laplace). This condition is called respiratory distress syndrome (RDS). RDS babies can also be treated with exogenous surfactant delivered to the baby's alveoli by means of an endotracheal tube or by surfactant-laced mist in the ventilator.
- 3. The buildup of mucus and particulate matter from inadequate cleansing by the cilia lining the respiratory tract would qualify as an obstructive disorder. In this case of nicotine poisoning the vital capacity is normal because lung tissue is not damaged, only obstructed. However, the rate of expiration as measured by the forced expiratory volume (FEV) test would be severely reduced due to the obstructions. If smoking progresses to emphysema, the FEV test results would continue to decline while alveolar tissue destruction, bronchiolar collapse, and air trapping would reveal a reduction in the vital capacity (restrictive disorder).

- 4. Carbon monoxide (CO) poisoning results in the formation of carboxyhemoglobin in the red blood cells instead of oxyhemoglobin. Since CO-hemoglobin bond is 210 times stronger than the bond with oxygen, oxygen is displaced and its delivery of is reduced. CO-hemoglobin is also a bright cherry red in color, especially in the mucous membranes. The hemoglobin concentration and hematocrit should remain the same, while the percent oxyhemoglobin saturation would be abnormally low due to the displaced oxygen. If CO poisoning and the oxyhemoglobin saturation decline is chronic, the production of 2,3-DPG in the red blood cells is increased. The bonding of 2,3-DPG with deoxyhemoglobin makes the latter more stable, encouraging the unloading of oxygen from the remaining hemoglobin molecules. The increased concentration of 2,3-DPG in red blood cells thus increases oxygen unloading at the tissues and shifts the oxyhemoglobin dissociation curve to the right.
- 5. High altitude dizziness is most likely due to the decreased P<sub>O2</sub> in the inspired air resulting in hyperventilation and a drop in P<sub>CO2</sub> accompanied by a similar decrease in arterial P<sub>O2</sub> to the brain (hypoxia). The benefit is you immediately know you are no longer at sea level and your brain is not getting adequate oxygen. The detriment is that you have begun a series of compensatory mechanisms in response to the hypoxia and until compensation has been achieved you should not exert yourself or increase your utilization of the little oxygen you are inhaling. Eventually, your kidney production of erythropoietin will stimulate an increase in hemoglobin and in RBC production (polycythemia), thereby raising your hematocrit and oxygen carrying capacity of the blood. Finally, the increased production of 2,3-DPG within the RBCs will result in a higher percent unloading of oxygen from hemoglobin in the tissues.