



## RESPIRATORY FAILURE

by Bill Wojciechowski, MS, RRT

The function of the respiratory system, of course, is gas exchange. Oxygen is constantly being removed from the lungs by the flowing pulmonary capillary blood, and carbon dioxide is continuously being eliminated from the lungs during expiration. Assuming a normal alveolar-capillary membrane, as long as four liters per minute of alveolar ventilation match five liters per minute of pulmonary capillary perfusion, gas exchange will be normal. Normal gas exchange produces a PaO<sub>2</sub> of 100 torr and a PaCO<sub>2</sub> of 40 torr. Even during exercise when a greater ventilatory demand and cardiac output are needed, alveolar ventilation and pulmonary capillary perfusion match to meet the body's increased metabolic requirements.

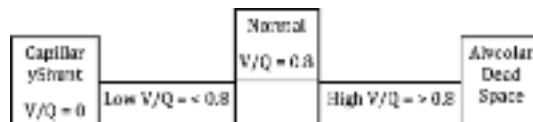
Periodically, the gas exchange mechanism of the lungs goes awry. The flow of air into the alveoli fails to match pulmonary capillary perfusion and vice versa. Consequently, the PaO<sub>2</sub> falls below normal and the PaCO<sub>2</sub> rises beyond normal.

This situation constitutes respiratory failure. When this situation develops suddenly, the condition that develops is acute respiratory failure. When it progresses more slowly it is called chronic respiratory failure. Acute respiratory failure is life-threatening, while chronic respiratory failure is less spectacular. Aside from being either acute or chronic, respiratory failure has two pathophysiologic forms, i.e., hypoxemic (type I) respiratory failure or hypercapnic (type II) respiratory failure.

**All respiratory failure has two pathophysiologic forms, hypoxemic (type I) or hypercapnic (type II)**

As a cause of hypoxemic respiratory failure, hypoventilation produces both hypoxemia and hypercapnia. As alveolar ventilation diminishes, hypercapnia develops. Interestingly, hypoventilation can be distinguished from shunting and V<sub>A</sub>Q<sub>C</sub> mismatching as a cause of hypoxemic respiratory failure by observing an alveolar-arterial oxygen tension gradient [P(A-a)O<sub>2</sub>] that decreases compared to its value when an FIO<sub>2</sub> of 0.21 is breathed. The normal P(A-a)O<sub>2</sub> gradient, which is age-specific, is about 10 to 15 torr when a person breathes room air. Hypoxemia causes the P(A-a)O<sub>2</sub> gradient to increase beyond 15 torr. If shunting is the cause of the hypoxemia, breathing an FIO<sub>2</sub> of 1.0 causes the P(A-a)O<sub>2</sub> gradient to become even greater than the room air P(A-a)O<sub>2</sub> gradient. On the other hand, if the cause of the hypoxemia is either hypoventilation or V<sub>A</sub>Q<sub>C</sub> mismatching, breathing an FIO<sub>2</sub> of 1.0 will cause the P(A-a)O<sub>2</sub> gradient to increase less in comparison to the P(A-a)O<sub>2</sub> gradient at an FIO<sub>2</sub> of 0.21.

The most common cause of hypoxemic respiratory failure is V<sub>A</sub>Q<sub>C</sub> disturbances. In the presence of pulmonary disease, V<sub>A</sub>Q<sub>C</sub> ratios extend across the entire ventilation-perfusion ratio spectrum. They vary from extremely low (bordering shunting) to exceptionally high (approaching alveolar dead space). The schematic below illustrates the V<sub>A</sub>Q<sub>C</sub> spectrum.



Low V<sub>A</sub>Q<sub>C</sub> ratios are described as perfusion in excess of ventilation and contribute to hypoxemia and hypercapnia. Generally, low V<sub>A</sub>Q<sub>C</sub> ratios are amenable to oxygen therapy, for example, COPD or an early asthma attack. These diseases are not characterized by significant amounts of severely low V<sub>A</sub>Q<sub>C</sub> ratios and shunting. However, high V<sub>A</sub>Q<sub>C</sub> ratios, which produce a dead-space-like effect because they feature ventilation in excess of perfusion, tend to have less influence on gas exchange. Pulmonary embolism is a pulmonary vascular disease that is characterized by high V<sub>A</sub>Q<sub>C</sub> ratios and alveolar dead space units. However, some lungs units may develop low V<sub>A</sub>Q<sub>C</sub> ratio because a portion of the pulmonary perfusion may be diverted to alveoli receiving normal ventilation.

Capillary shunt was defined previously as alveoli receiving pulmonary capillary blood flow, but no ventilation. Therefore, the mixed venous blood flowing to these gas exchange units does not become arterialized because no ventilation is present to provide for oxygen uptake and carbon dioxide excretion. The ultimate fate of blood flowing from these units is to continue to the left atrium where it will mix with arterialized blood and lower the PaO<sub>2</sub> and elevate the PaCO<sub>2</sub>. Keep in mind that hypoxemia caused by capillary shunt-

### Hypoxemic Respiratory Failure

The primary problem associated with hypoxemic respiratory failure is the inability of the patient to maintain oxygenation. As a clinical guide, failure to achieve a PaO<sub>2</sub> of 60 torr or less while breathing room air constitutes hypoxemic respiratory failure. At the same time, the PaCO<sub>2</sub> may be high, low, or normal. The essential problem with type I respiratory failure is impeded diffusion of oxygen across the alveolar-capillary membrane. The most common causes of hypoxemic respiratory failure are (1) hypoventilation, (2) ventilation-perfusion (V<sub>A</sub>Q<sub>C</sub>) abnormalities, and (3) shunting.

As a cause of hypoxemic respiratory failure, hypoventilation can develop from central nervous system depression caused by drugs (opiates) or obesity-hypoventilation syndrome, and from neuromuscular diseases such as myasthenia gravis or Guillain-Barré syndrome. Hypoventilation produces both hypoxemia and hyper-

ing is not amenable to oxygen therapy. The amount of shunt can be quantified using the following equation:

$$\frac{Q_s}{Q_T} = \frac{C_{cO_2} - C_{aO_2}}{C_{aO_2} - C_{vO_2}}$$

The  $Q_s/Q_T$  ratio represents the shunt fraction. The  $C_{cO_2}$  signifies the end-pulmonary capillary  $O_2$  content. The  $C_{aO_2}$  and the  $C_{vO_2}$  refer to the total arterial  $O_2$  content and the total mixed-venous  $O_2$  content, respectively. The alveolar air equation is needed to calculate the  $C_{cO_2}$  and a pulmonary artery catheter is required to determine the  $C_{vO_2}$ . As a cause of type I respiratory failure, shunting is associated with pneumonia and atelectasis. An elevated  $PaCO_2$  generally occurs when the shunt fraction ( $Q_s/Q_T$ ) exceeds 0.60. The normal shunt fraction is between 0.025 and 0.050 as a result of the normal anatomic shunt that occurs because of the mixed-venous blood contributed to arterial blood by the bronchial, pleural, and Thebesian veins. The anatomic shunt accounts for the fact that the normal  $SaO_2$  on room air ranges from 95% to 97.5%, as opposed to being 100% if the anatomic shunt were non-existent.

In addition to treating the underlying disease, the treatment of hypoxemic respiratory failure involves airway management, and supplemental oxygen. Continuous positive airway pressure, noninvasive positive pressure ventilation, or invasive positive pressure mechanical ventilation may be indicated.

### **Hypercapnic Respiratory Failure**

When the respiratory system functions normally and when alveolar ventilation and  $CO_2$  production are in phase with each other, the  $PaCO_2$  ranges between 35 and 45 torr. On the other hand, when alveolar ventilation outpaces  $CO_2$  production, the  $PaCO_2$  will fall below 35 torr. This relationship is known as hyperventilation. Conversely, hypoventilation, which is characterized by a  $PaCO_2$  greater than 45 torr, develops when alveolar ventilation lags behind  $CO_2$  production. The relationship among these three factors, that is, the  $PaCO_2$ , carbon dioxide production ( $VCO_2$ ), and alveolar ventilation ( $V_A$ ) is illustrated in the following formula:

$$PaCO_2 = \frac{K \times VCO_2}{V_A}$$

The value of K equals 0.0863 and is a constant. Alveolar ventilation diminishes when minute ventilation ( $V_E$ ) decreases or



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when dead space ventilation ( $V_D$ ) increases. Common causes of decreased  $V_E$  are neuromuscular diseases such as myasthenia gravis and Guillain-Barré syndrome. Rapid shallow breathing or ventilating nonperfused alveoli will increase  $V_D$ . Hypercapnic respiratory failure involves severely compromised alveolar ventilation, and is often defined as a  $PaO_2$  less than 60 torr and a  $PaCO_2$  of 50 torr or greater while a patient breathes room air. Oxygen therapy will typically correct the hypoxemia caused by hypercapnic respiratory failure.

The pH associated with hypercapnic respiratory failure depends on whether the condition is acute or chronic. In the acute form, which develops suddenly within minutes, the pH falls commensurate with the rise in  $PaCO_2$  because the bicarbonate ion concentration will still be essentially normal. With chronic type II respiratory failure, which evolves over days or even years, renal compensation will have become established preventing the pH from falling significantly.

Some causes of hypercapnic respiratory failure include COPD exacerbation, status asthmaticus, drug overdose, neuromuscular diseases, and head and spinal cord injuries. Aside from attending to the underlying pathophysiology, treatment of hypercapnic respiratory failure entails airway management, oxygen administration, and mechanical ventilation. Not only must the respiratory therapist apply assessment skills and diagnostic techniques to differentiate between the presence of hypoxemic and hypercapnic respiratory failure, but also they must distinguish the underlying cause of each form. Ascertaining whether the respiratory failure is acute or chronic is likewise critical to appropriate treatment. The underlying disease responsible for the respiratory failure often determines the prognosis. For example, acute respiratory distress syndrome has a mortality rate that varies between 40% and 50%. For COPD patients with exacerbations, mortality may be as low as 10% or as high as 30%.

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