Perfusion

The pulmonary vascular bed differs from the systemic circulation in several respects. The pulmonary vascular bed receives the entire cardiac output of the right ventricle, whereas the cardiac output from the left ventricle is dispersed among several organ systems. Despite receiving the entire cardiac output, the pulmonary system is a low-resistance, low-pressure circuit. The normal mean systemic arterial pressure is about 100 mm Hg, whereas the normal mean pulmonary artery pressure is in the range of 15 mm Hg. The vascular bed can passively accommodate an increase in blood flow without raising arterial pressure by recruiting more vessels in the lung. During exercise, for example, there is little increase in pulmonary artery resistance despite a large increase in pulmonary blood flow. Hypoxic vasoconstriction, another feature unique to the pulmonary vascular system, regulates regional blood flow. This regulation aids in matching blood flow to ventilation by reducing flow to poorly ventilated regions of the lung.

Perfusion ($\dot{Q}$) refers to the blood flow through an organ (i.e., the lung). In the upright individual, there is greater perfusion of the lung bases than of the apices (Fig. 15-12). In a low-pressure system such as the pulmonary circulation, the effects of gravity on blood flow need to betaken into account. The arterial-venous pressure difference usually provides the "driving" pressure for blood flow in the systemic circulation, but this is true only for certain regions of the lung. Pulmonary blood flow also needs to be considered in the context of alveolar pressure. Venous and arterial pressures are importantly affected by gravity, whereas alveolar pressure remains constant throughout the lung, assuming the airways are open. Therefore, as one descends from the apex to the base of the lung, arterial and venous pressures increase because of gravity but alveolar pressure remains constant.

At the apex, alveolar pressure may be greater than arterial pressure. This region of the lung is referred to as *zone 1,* and, in theory, it receives no blood flow. The alveolar pressure may be greater than arterial pressure, for example, in special circumstances such as hypovolemic shock, which lowers the arterial pressure, or with very high levels of positive end-expiratory pressure (PEEP), which increases alveolar pressure.

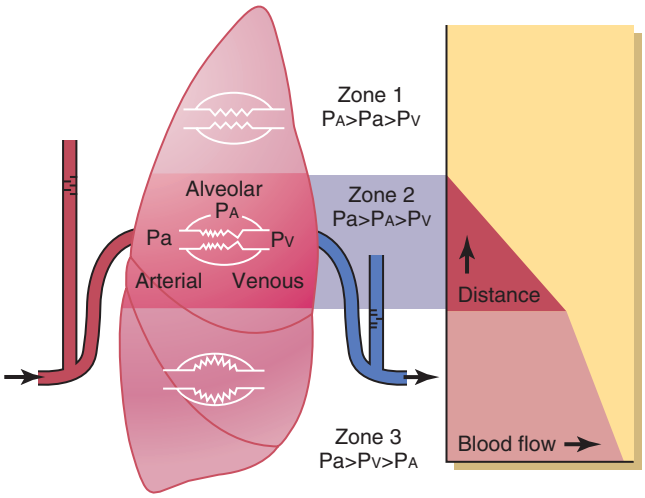

FIGURE 15-12 Zonal model of blood flow in the lung. Because of the inter-relationship of arterial (Pa) and venous (Pv) vascular pressures and alveolar (PA) pressures, the lung base receives the most flow (see text for explanation). (From West JB, Dollery CT, Naimark A: Distribution of blood flow in isolated lung: relation to vascular and alveolar pressures, J Appl Physiol 19:713–724, 1964.)

As one descends from the apex toward the midzone of the lung, arterial and venous pressures increase, whereas alveolar pressure remains constant. At some point, arterial pressure becomes greater than alveolar pressure. In this region, the driving pressure for blood flow is the arterial-alveolar pressure difference. This region is referred to as *zone 2* of the lung. Normally, zone 2 is very small because alveolar pressure is less than venous pressure in most of the lung. However, with high levels of PEEP, alveolar pressure becomes greater than venous pressure in more lung regions.

Further toward the base of the lung, the effects of gravity on arterial and venous pressures are more pronounced, venous pressure becomes greater than alveolar pressure, and the arterial-venous pressure difference provides the driving pressure for blood flow, as in the systemic circulation. This region is referred to as *zone 3* of the lung.

Normally, most of the lung is in zone 3, and most of the perfusion is to the lung base. This inequality in perfusion from apex to base is *qualitatively* similar to the inequality of ventilation from apex to base. However, blood flow increases from apex to base *more* than ventilation does, and this accounts for the small amount of ventilation-perfusion inequality that exists in the normal lung.

Gas Transfer

Oxygen and carbon dioxide are easily dissolved in plasma. Nitrogen is much less soluble and is not significantly exchanged across the alveolar-capillary interface. The driving force for the diffusion of a gas across a tissue barrier is the difference in partial pressure of the gas across the barrier. The partial pressure of oxygen in inspired room air entering the trachea is 150 mm Hg; this is derived from the equation, $P_{O_2} = (P_{atm} - P_{H_2O}) \times F_{IO_2}$, assuming that P_{atm} (atmospheric pressure) is 760 mm Hg, P_{H_2O} (the partial pressure of water vapor) is 47 mm Hg, and F_{IO_2} (the fraction of oxygen in inspired air) is 20.9%. In the alveolus, however, the partial pressure of oxygen is reduced to 100 mm Hg because the inspired V_T mixes with about 3 L of "oxygen-poor" air already in the lungs and is diluted by carbon dioxide moving into the alveolus from the pulmonary capillaries. The partial pressure of oxygen in the alveolus (P_{AO_2}) is set by the balance of these processes. Increasing minute ventilation increases the amount of oxygen added to the alveolus while lowering the P_{ACO_2}—the opposite result from hypoventilation. This reciprocal relationship between alveolar carbon dioxide and alveolar oxygen is described by the *alveolar gas equation*:

$$P_{AO_2} = [(P_{atm} - P_{H_2O}) \times F_{IO_2}] - (P_{ACO_2}/RER)$$

where RER is the respiratory exchange ratio, usually about 0.8.

The pressure gradient that drives diffusion of oxygen from the alveolus to the capillary is the difference between the alveolar P_{O_2} (100 mm Hg) and the arterial P_{O_2} (40 mm Hg) in the capillary blood entering the alveolus. By the time the blood leaves the alveolus, the P_{O_2} in the capillary blood has risen to 100 mm Hg.