Ventilation Perfusion Relationships

Lecturer: Sally Osborne, Ph.D. Department of Cellular & Physiological Sciences

email: sosborne@interchange.ubc.ca **Useful links**: www.sallyosborne.com

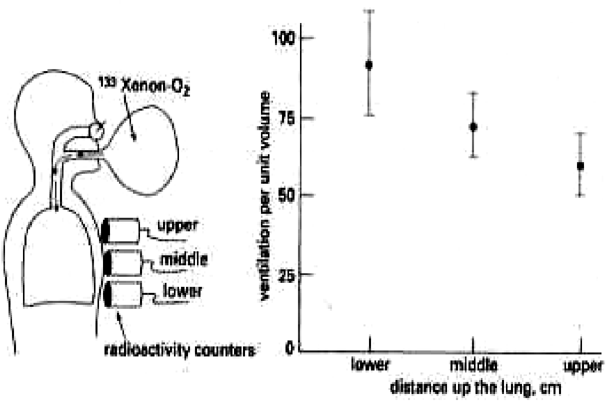
Required Reading: Respiratory Physiology, A Clinical Approach, Schwartzstein & Parker pp.23-28 & 95-105.

Objectives

1. What is the pattern of distribution of ventilation (V) and perfusion (Q), and the relationship between ventilation and perfusion (V/Q ratio) in normal lungs
2. What factors cause the distribution of ventilation in the lungs to be uneven?
3. What factors cause the distribution of perfusion in the lung to be uneven?
4. What is the impact of uneven matching of ventilation and perfusion (V/Q mismatch) on regional and global exchange in the lungs?
5. How does the respiratory system compensate for the gas exchange abnormalities caused by V/Q mismatch?

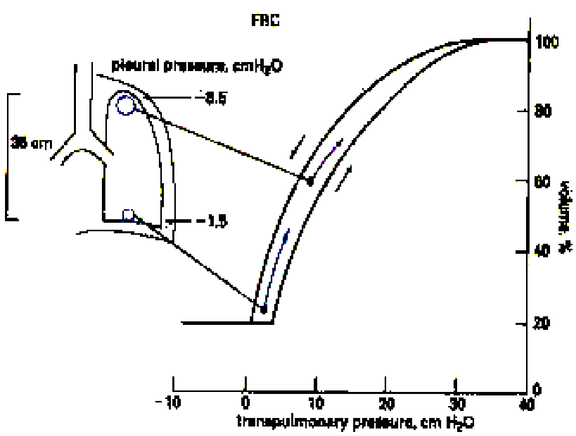
1. Distribution of Ventilation in the Normal Upright Lung

Healthy subjects seated in the upright posture breathing quietly from FRC show differences in regional ventilation. Ventilation is directed preferentially to the bases of the lungs. Similar studies on healthy subjects in lying on their left side show less difference in regional ventilation. However, ventilation is directed preferentially to the left lung compared to the right. The regional differences are therefore considered to be influenced by gravity. The term "gravity dependent region" denotes the regions of the lung lower with respect to gravity and better ventilated than those above them ("gravity independent").



Regional distribution of ventilation determined by a single breath of oxygen and radioactive xenon 133 gas mixture.

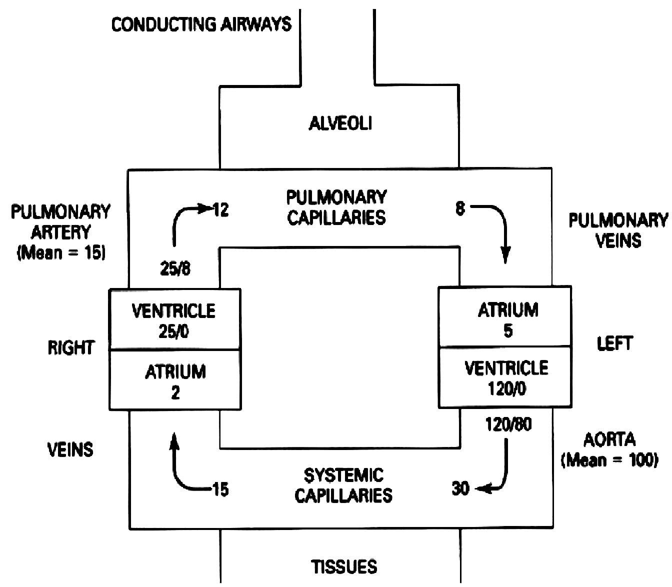
2. Explanation for Differences in Regional Ventilation



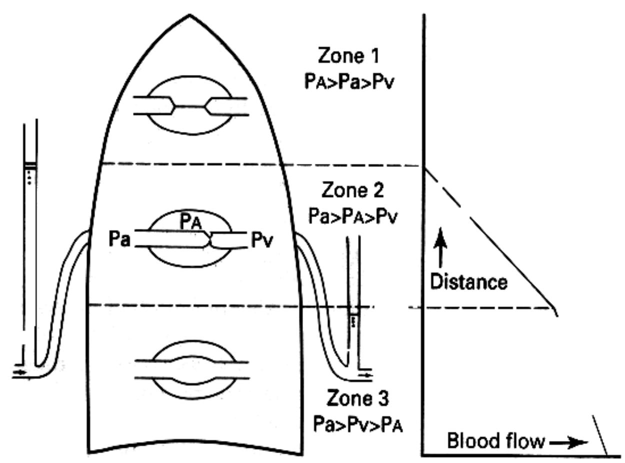
In the upright lung intrapleural pressure varies from the top to the base of the lungs. For every centimeter of vertical displacement from the tip of the lung to the base, intrapleural pressure increases by about 0.2 cm H_2O. For an average healthy male, the lung is about 35 cm long and at FRC the intrapleural pressure at the apex of the lung is about – 8 cm H_2O and at the base about – 1.5 cm H_2O. This means that the alveoli at the apex are exposed to a greater distending pressure (P_A-P_{pl}= 0 - -8= 8 cm H_2O) compared to those at the base (P_A-P_{pl}= 0 - -1.5= 1.5 cm H_2O). It is this difference in initial volume that results in the preferential distribution of ventilation to the alveoli at the base of the lungs. To understand this you need to recall the relationship between pressure and volume in the lungs as described by the static pressure volume curve of the lungs. The alveoli at the base of the lung have a smaller initial volume and are operating at the steep portion of the pressure volume curve. Therefore, being relatively more compliant, the alveoli at the base fill to a greater extend for a given change in intrapleural pressure during inspiration compared to the alveoli at the apex. Conversely, the alveoli at the apex are exposed to a larger distending pressure, have a greater initial volume, operate at a less compliant segment of the pressure volume curve and fill less during inspiration.

3. Distribution of Perfusion in the Normal Upright Lung

The walls of the vasculature in the pulmonary circulation are much thinner than the corresponding parts of the systemic circulation. In addition, there is less smooth muscle in the walls of the pulmonary arterial tree and there are no highly muscular vessels that correspond to the systemic arterioles. The physiologic consequence of thin walls and small amount of smooth muscle in pulmonary vasculature is that pulmonary vessels offer relatively low resistance to blood flow and are much more distensible and compressible than their systemic counterpart. These factors result in much lower intravascular pressures in the pulmonary circulation compared to the systemic circulation. Since pulmonary vessels are located in the thorax, they are subject to alveolar pressures



In healthy individuals at rest with normal cardiac output, pulmonary blood flow is distributed unevenly in the upright lung. Similar to the distribution of ventilation, pulmonary blood flow is preferentially directed to the base of the lungs. This distribution is dependent on three relative pressures: alveolar pressure, pulmonary arterial pressure and pulmonary venous pressure. Three functional zones based on these pressure relationships where first suggested by John West and characterized in experiments on excised, perfused upright animal lungs under low arterial perfusion pressures. The most apical region, **zone I**, where **PA>Pa>Pv**, alveolar pressure exceeds vascular pressures resulting in capillary collapse and no blood flow. The alveoli in this zone do not participate in gas exchange and are part of the lung's **alveolar dead space**. In healthy subjects under normal perfusion pressures, zone I is not present because arterial pressures is just sufficient to raise blood to the top of the lung and exceed alveolar pressure. Zone I may be present if pulmonary arterial pressure is reduced (following severe hemorrhage) or if alveolar pressure is raised (during positive pressure ventilation). In **zone II** where **Pa>PA>Pv**, the driving pressure for blood flow is determined by the difference between arterial and alveolar pressure. Alveolar pressure is constant throughout the lung. In contrast, arterial pressure increases from the apex to the base of the lungs due to an increase in blood hydrostatic pressure. Therefore, there is a gradual increase in blood flow down zone II as a result in an increase in the driving pressure (Pa-PA). In **zone III**, both vascular pressures are greater than alveolar pressure, **Pa>Pv>PA** and the driving pressure for blood flow is simply pulmonary

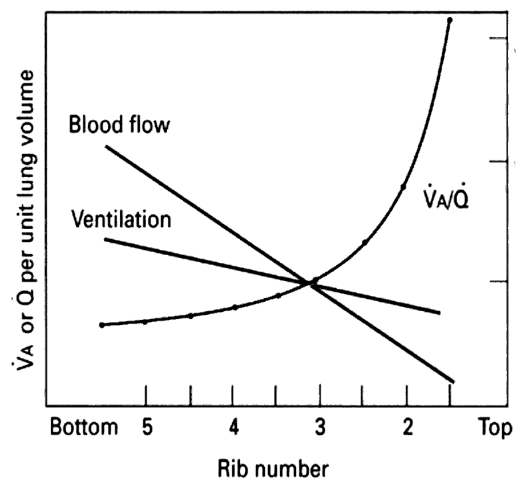


arterial pressure minus pulmonary venous pressure. The increase in blood flow in zones II and III reflects also the recruitment and distention of pulmonary vessels with increasing intravascular pressures down the lung.

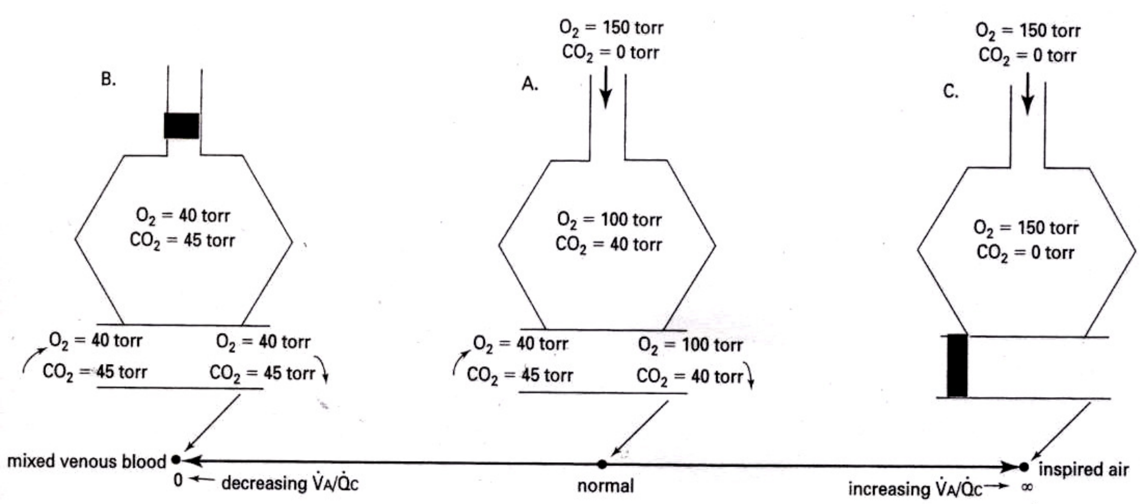
A key point to appreciate is that the zones characterized above and their boundaries are physiological, not anatomical landmarks. The borders between zones can be moved by many physiological and pathophysiological alterations or conditions including the following list of examples. **Alveolar pressure** changes are only a few centimeters of water during the course of a quiet breath but they are much greater during speech, exercise and other conditions; patients on positive pressure ventilation with positive end expiratory pressure (PEEP) may have substantial zone I due to high alveolar pressures. **Pulmonary arterial pressure** can be after a severe hemorrhage or during general anesthesia resulting in zone I conditions. Pulmonary artery pressure is high during exercise eliminating any existing zone I into zone II and moving the boundary between zone III and II upward. **Changes in body position** alter the orientation of the zones with respect to the anatomic locations in the lung but the same relationship with respect to gravity and vascular pressure remains.

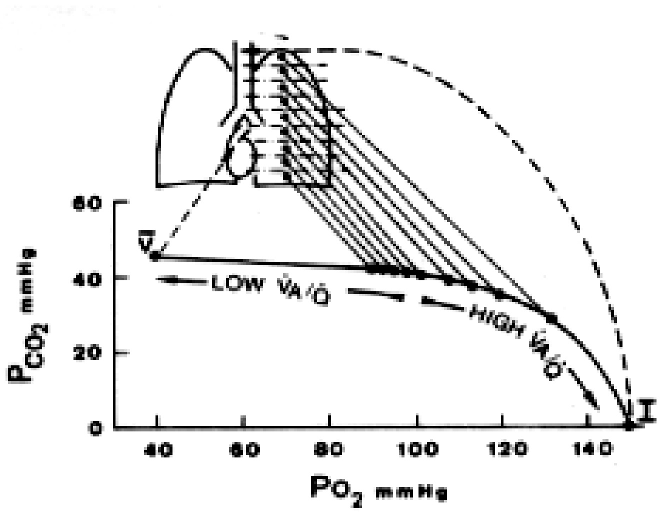
4. Ventilation Perfusion Ratio

In the upright lung the gradient for perfusion is greater than the gradient for ventilation (see figure on the right). Hence the greatest ventilation perfusion ratio is at the top of the lungs and the lowest ratio at the base of the lungs.



The alveolar partial pressure of oxygen and carbon dioxide are determined by the ratio of ventilation to perfusion. The compartments below describe the consequence of extremely high and low V/Q ratios on the composition of alveolar gases.





The O2-CO2 diagram depicts the regional differences in alveolar partial pressures of oxygen and carbon dioxide in the upright healthy lung within the context of possible extreme V/Q ratios. Normally, the blood in the pulmonary capillary equilibrates with the alveolar PO2 and PCO2 so the effects of regional differences in V/Q on regional end capillary blood values can be predicted. The O2-CO2 diagram shows that the upper regions of the lungs with higher V/Q ratio have relatively higher PO2s and lower PCO2s compared to the lower regions. At first glance this may seem to indicate that there is greater gas exchange at the upper regions of the lungs but recall that there is greater ventilation and perfusion to the bases of the lungs and therefore greater gas exchange at the base of the lungs. Overall this arrangement results in adequate gas exchange but if disease states affect either the distribution of ventilation or perfusion, there will be an increase in V/Q mismatching and gas exchange worsens (more on this in the lecture on causes of hypoxemia in week 3).

5. Physiologic responses that minimize V/Q mismatching

Unlike the systemic circulation, the resistance of the pulmonary vessels is not affected predominately by the balance between sympathetic and parasympathetic inputs. The key determinant of pulmonary vessel size is alveolar hypoxia. Alveolar hypoxia has direct effect on pulmonary vessel smooth muscle resulting in vasoconstriction. The hypoxic pulmonary vasoconstriction response is graded and occurs at alveolar PO_2 levels ranging from 100-20 mmHg. With regional hypoxia, where local airway obstruction has reduced oxygenation of an alveolus, local pulmonary vasoconstriction is a useful physiologic measure. Blood is diverted from the poorly ventilated alveolus to areas with adequate degree of oxygenation. With global hypoxia where the whole lung is exposed low PO_2 in inspired air such as encountered in high altitude or hypoventilation, the vasoconstriction occurs throughout the lung. There is potential that the global vasoconstriction can lead to pulmonary hypertension and ultimately with chronic exposure to pulmonary edema.