





A (3) موجود في كل الرنة في عاله الإضلقاء على الطهر

8 - Ventilation / perfusion ratio -II

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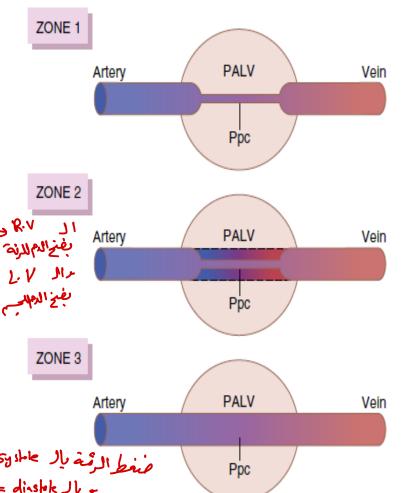
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Zones 1, 2, and 3 of Pulmonary Blood Flow

- Zone 1: No blood flow during all portions of the cardiac cycle because the local alveolar capillary pressure in that area of the lung never rises higher than the alveolar air pressure during any part of the cardiac cycle
 Zone 2: Intermittent blood flow only during the appropriate
- Peaks of pulmonary arterial pressure because the عبير المرادة peaks of pulmonary arterial pressure because the systolic pressure is then greater than the alveolar air pressure, but the diastolic pressure is less than the alveolar air pressure So if Cant over one if

Zone 3: Continuous blood flow because the alveolar capillary pressure remains greater than alveolar air pressure during the entire cardiac cycle

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- -In normal healthy lung during **standing**, there are zone II (Apex) and zone III (at base) and during recumbent position all lung are of zone III.
- **Zone I** presents abnormally if the person breaths air under positive pressure in which intra-alveolar pressure reaches 10 mmHg **also** occur in hypovolemic shock.
- -During muscular exercise, the pulmonary blood flow increases in all parts of the lung via opening of new capillaries especially the apex which has already closed capillaries during rest.

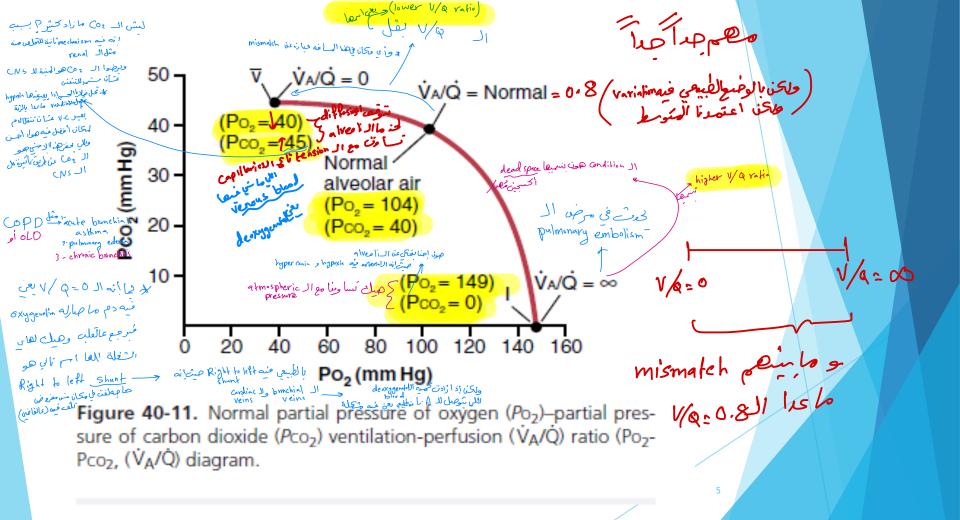
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الفازات pressur exerted الفازمين معن الفازات portial الفازات by this gas Just to Remember

The partial pressures of gases PARTIAL PRESSURES in the alveoli differ from those in the atmosphere. This difference is caused by a combination of several factors: Humidification of inhaled air Gas exchange between alveoli PO2 = 104 mm Hg _ and pulmonary capillaries nthe alverti PCO2 = 40 mm Hg Mixing of new and old air PH2O = 47 mm Hg Atmosphere lo 4 mm Hg (mmHg) 159 PO2 0.3 PO2 = atmispheric X fraction by percentage pressure X ef 02 atmospheric pressure = 760 mmllg



Pathophysiology

Extreme alterations of V/Q

-An area with perfusion but no ventilation (and thus a V/Q of zero) is termed (shunt)

With airway obstruction, the composition of alveolar air approaches that of mixed venous blood

i.e. PO2 = 40 mmHg PCO2 = 45 mmHg

-An area with ventilation but no perfusion (and thus a V/Q undefined though approaching infinity) is termed "dead space".

With pulmonary embolus, the composition of alveolar air approaches that of inspired air

i.e. PO2 = 149 mmHg PCO2 = 0 mmHg

-The term V/Q mismatch is more appropriate for conditions in between these two extremes.

A lower V/Q ratio (with respect to the expected value for a particular lung area in a defined position) impairs pulmonary gas exchange and is a cause of low arterial partial pressure of oxygen (pO2).

Excretion of carbon dioxide is also impaired, but a rise in the arterial partial pressure of carbon dioxide (pCO2) is very uncommon because this leads to respiratory stimulation and the resultant increase in alveolar ventilation returns arterial partial pressure CO2 to within the normal range.

These abnormal phenomena are usually seen in chronic bronchitis, asthma, and acute pulmonary edema.

A high V/Q ratio decreases pCO2 and increases pO2 in alveoli.

- -Because of the increased dead space ventilation, the pO2 is **reduced (in blood vessels)** and thus also the peripheral oxygen saturation is **lower** than normal, leading to tachypnea.
- This finding is typically associated with **pulmonary embolism** (where blood circulation is impaired by an embolus).
- Ventilation is wasted, as it fails to oxygenate any blood.
- -A high V/Q can also be observed in emphysema as a mal-adaptive ventilatory overwork of the damaged lung parenchyma. Because of the loss of alveolar surface area, there is proportionally more ventilation per available perfusion area. As a contrast, this loss of surface area leads to decreased arterial pO2 due to impaired gas exchange.

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1. V/Q ratio in airway obstruction

If the airways are completely blocked (e.g., by a piece of steak caught in the trachea), then ventilation is zero. If blood flow is normal, then V/Q is zero, which is called a right-to-left shunt.

There is no gas exchange in a lung that is perfused but not ventilated.

The PO2 and PCO2 of alveolar air will approach their values in mixed venous blood.

2. V/Q ratio in pulmonary embolism

If blood flow to a lung is completely blocked (e.g., by an embolism occluding a pulmonary artery), then blood flow to that lung is zero. If ventilation is normal, then V/Q is infinite, which is called dead space.

There is no gas exchange in a lung that is ventilated but not perfused.

The PO2 and PCO2 of alveolar gas will approach their values in inspired air.

V/Q DEFECTS Normal Airway obstruction Pulmonary embolus (dead space) (shunt) V/Q 8.0 0 ∞ PA_{O2} 100 mm Hg 150 mm Hg PACO₂ 40 mm Hg 0 mm Hg Pa_{O2} 100 mm Hg 40 mm Hg Pa_{CO2} 40 mm Hg 46 mm Hg

FIGURE 4.13 Effect of ventilation/perfusion (V/Q) defects on gas exchange. With airway obstruction, the composition of systemic arterial blood approaches that of mixed venous blood. With pulmonary embolus, the composition of alveolar gas approaches that of inspired air. $PA_{02} = alveolar Po_2$; $PA_{CO_2} = alveolar Pco_2$; $Pa_{02} = arterial Pco_2$.

Additional notes:

Dead Space

Dead space is the volume of the airways and lungs that **does not participate in gas exchange.** Dead space is a general term that refers to both the **anatomic dead space of the conducting airways and a functional, or physiologic, dead space.**

Physiologic Dead Space

The physiologic dead space is the total volume of the lungs that does not participate in gas exchange. Physiologic dead space includes the anatomic dead space of the conducting airways plus a functional dead space in the alveoli. The functional dead space can be thought of as ventilated alveoli that do not participate in gas exchange. The most important reason that alveoli do not participate in gas exchange is a mismatch of ventilation and perfusion, or so-called ventilation/perfusion defect, in which ventilated alveoli are not perfused by pulmonary capillary blood. In normal persons, the physiologic dead space is nearly equal to the anatomic dead space. In other words, alveolar ventilation and perfusion (blood flow) are normally well matched and functional dead space is small.

In certain pathologic situations, however, the physiologic dead space can become larger than the anatomic dead space, suggesting a ventilation/perfusion defect,