

Ventilation-Perfusion Ratio; Consequences on Arteriole pO₂

Definitions

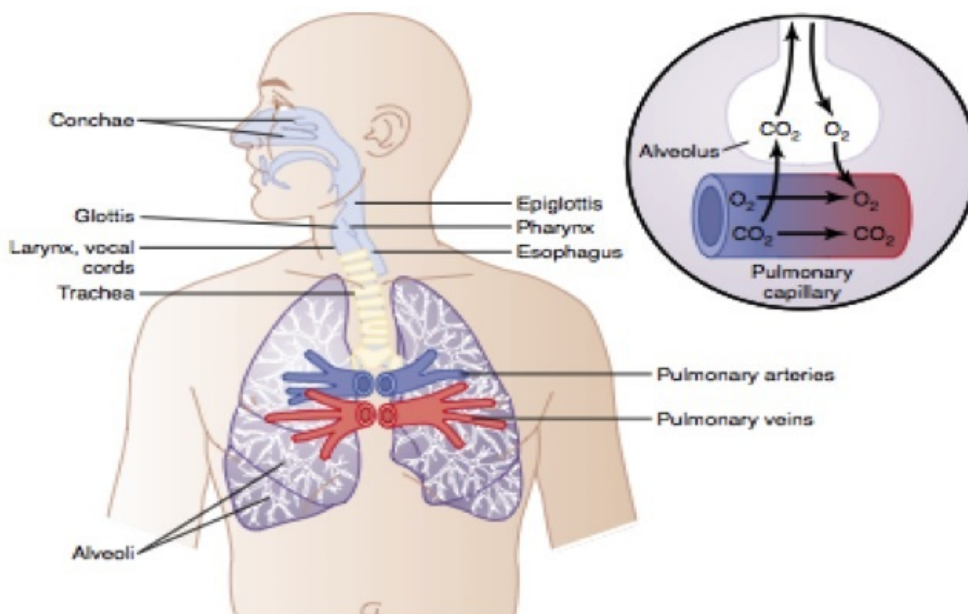
Ventilation (Breathing): the movement of air (& thus gases) between the environment and alveoli. 12-20 breaths/min @ Rest

Perfusion* (Blood Flow): the process of nutritive delivery of arterial blood to a capillary bed. Pressure ~25/8 mm Hg, therefore Low Resistance; High Flow System

Ventilation-Perfusion Ratio: a measurement used to assess the efficiency and adequacy of the matching of two variables: ventilation & perfusion.

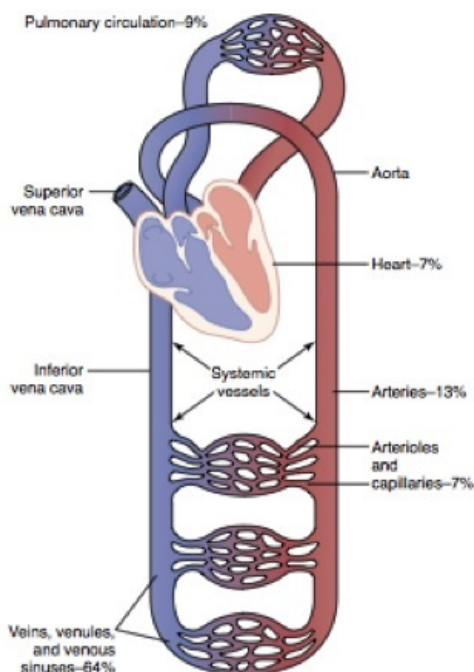
- Opposite in pulmonary circulation.

Ventilation



Gas Laws: Dalton's Law $P_{Total} =$

PA + PB + PC...



Gases (singly/mixture) move from areas of HIGH to LOW Pressure (i.e. P)

Boyle's Law $P_1V_1 = P_2V_2$

Perfusion

10% of Total Blood Volume in Pulmonary Circulation (~0.5L @ CO ≈ 5L/min) HIGH Flow; LOW Resistance System F is (Proportional) to the (Change) P/R [= (Change)Pr⁴/Ln * (pie/8)] Receives the ENTIRE CO per minute R (Proportional) Ln/r⁴ LOW because of SMALL L(ength) & LARGE cross-sectional Area 90% of R due to Trachea & Bronchi (rigid structures with small cross-sxn area)

Ventilation-Perfusion Ratio

Main determinant of blood concentration* of O₂ Variables V = Ventilation (expressed as O₂ mass reaching alveoli per minute) V = g / min Q = Perfusion (flow of blood in the lungs per minute) Q = L / min Therefore: (V/Q) = [(g/min)/(L/min)] => g / L*

- Usually, however, the density of the gas (in g/l) can be used to convert the V/Q into a dimensionless ratio, and this is how it is most often expressed.

Physiologic Shunt (V/Q below norm): measure of the amount of (shunted) blood that is not oxygenated passing through lungs. (PICS 3 & 4; ppg 8)

QPS=physio. shunt blood flow per min QT=CO per min C_iO₂= arterial PO₂ w/ ideal V/Q ratio. CaO₂ = measured PO₂

in arterial blood. C_vO₂ =arterial PO₂ in mixed venous blood. $\frac{\dot{Q}_{PS}}{\dot{Q}_T} = \frac{C_{iO_2} - C_{aO_2}}{C_{iO_2} - C_{\bar{v}O_2}}$

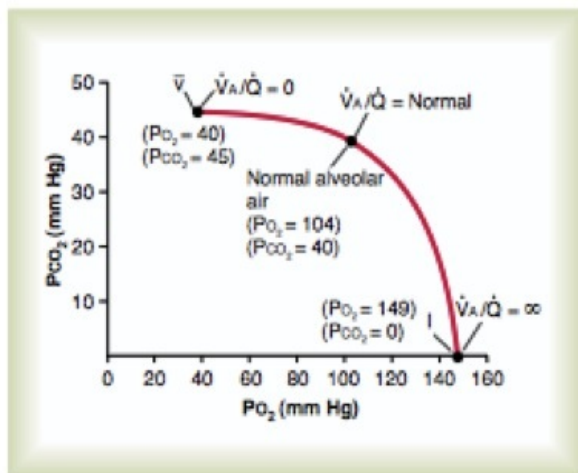


Figure 39-11

Normal PO₂-PCO₂, V_A/Q diagram.

Physiologic Dead Space (V/Q above norm): measure of the amount of (wasted) ventilation. (PICS 5 & 6; ppg 9)

VDphys=physiologic dead space VT=tidal volume PaCO₂=arterial PCO₂ w/ ideal V/Q ratio. PECO₂=average PCO₂ in

expired air. $\frac{V_{D_{phys}}}{V_T} = \frac{P_{aCO_2} - \bar{P}_{E_{CO_2}}}{P_{aCO_2}}$

1° - By Regulating the diameters of Arterioles & Bronchioles Arterioles Pulmonary Capillary Properties (*Collapsible) @ Rest: Apical Cap. Beds = Collapsed (V/Q ≈ 2.5x Ideal) Base Cap. Beds = Perfused (gravity-induced increase in hydrostatic pressure; V/Q ≈ 0.6x Ideal) During Exercise: Apical Cap Beds = Perfused (CO-induced increase in B.P.) Local Factors (Resistance is 1° regulated by [PO₂] in surrounding interstitial fluid.) PO₂ PCO₂ Bronchioles (Diameter is 1° regulated by [PCO₂] in exhaled air)

Arterioles

(Decrease) Pulmonary Arteriole PO₂ (surrounding tissue)→ * Pulmonary Arteriole Vasoconstriction → (Decrease) Pulmonary Venous PO₂ → (Decrease) Systemic Arteriole PO₂

- Note: vasoconstriction in Pulmonary arterioles is in response to low PO₂; opposite to Systemic arterioles.

Bronchioles

(Increase) Alveolar PCO_2 (exhaled air) → (Increase) Bronchodilation → (Increase) Alveolar PO_2 (inspired air) → (Increase) Systemic Arteriole PO_2

- PSNS = bronchoconstriction; SNS ≈ NOT significant, BUT respond to Epinephrine (Adrenal Medulla; β_2 Recepto

Gas Composition	Bronchioles	Pulmonary Arterioles	Systemic Arterioles
P_{CO_2} Increases	Dilate	(Constrict)	Dilate
P_{CO_2} Decreases	Constrict	(Dilate)	Constrict
P_{O_2} Increases	(Constrict)	Dilate	Constrict
P_{O_2} Decreases	(Dilate)	Constrict	Dilate

- Note: Responses in parentheses indicate weak responses.

Summary

1. Matching ventilation to alveolar sacs with blood flow past those alveoli, is a two-part process involving both air- & blood-flow. 2. V/Q is a measure of efficiency and adequacy of, ultimately, gas exchange/delivery. 3. Bronchioles & Arterioles are subject to reflex control via NS & hormones. **HOWEVER:** 4. Bronchioles & Arterioles are controlled on a minute-to-minute basis via paracrines.

References

Dee U. Silverthorn (2004). Texas: Human Physiology, (3rd Ed.) Daryl Fox Arthur C. Guyton & John E. Hall (2006). Mississippi: Textbook of Medical Physiology, (11th Ed.) Elseiver Inc. <http://www.wikipedia.org/>