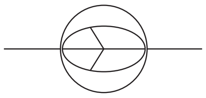
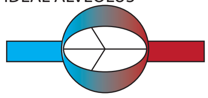
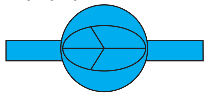
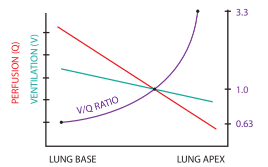
VENTILATION - PERFUSION RELATIONSHIPS

RILEYS THREE COMPARTMENT MODEL	V/Q RATIOS	MEASUREMENT	ALVEOLAR AIR	CONSEQUENCE	TREATMENT
DEAD SPACE 	∞ 3.3	Dead Space → Bohr equation $P_{ECO_2}(TV) = P_{aCO_2}(TV - \text{Dead Space})$	Alveolar air approaches inspired air concentrations (no exchange wasted air) $PO_2 = 149$ $CO_2 = 0$	Decreased perfusion leads wasted ventilation therefore decreased minute alveolar ventilation & primarily to increased blood CO2	Increased tidal volumes will reduce the effect of dead space (note that Alveolar Vent = TV - PDS)
IDEAL ALVEOLUS 	1 0.63	V/Q Mismatch → Using the multiple inert gas elimination technique (MIDGET) or nuclear med studies Alveolar air equation $P_{AO_2} = FiO_2(P_{atm} - P_{H_2O}) - P_{aCO_2}/RQ$	Alveolar air equals concentration of postcap blood due to ideal exchange $PO_2 = 104$ $CO_2 = 40$	V/Q scatter leads to decreased PaO2 because a majority of mismatch flow is at ratios < 1 and a small drop is acentuated by the point on the Hb dissociation curve	Increased FiO2 will improve oxygenation unless the V/Q ratio is 0 (true shunt). High FiO2 will remove the V/Q scatter effect.
TRUE SHUNT 	0	Shunt equation (Venous admixture) $QT(\text{Art O2 cont}) = QS(\text{precap O2 cont}) + (\text{postcap 02 cont})(QT-QS)$	Alveolar air approaches mixed venous concentrations (no exchange wasted blood) $PO_2 = 40$ $CO_2 = 46$	Shunt leads to both ↑CO2 and ↓O2 but the decrease in PO2 is more pronounced because it is on the flat of the dissociation curve and the CO2 dissociation is near linear	Improved recruitment may work unless the shunt is extra-pulmonary. ↑FiO2 is decreasingly effective in true shunts > 30%

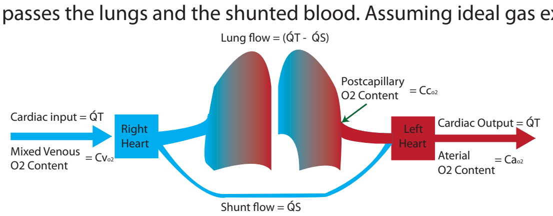
Ventilation refresher - the regional distribution of ventilation is dependent on two main processes and one minor. The first is the most important and is the effect of gravity. As the lung has weight, the lower parts of the lung are generally more compressed and less inflated than the upper part of the lungs which leads to the more dependent parts of the lung having greater compliance (compliance = △volume/ △pressure) thus they generally receive greater ventilation. In the upright position, with slow VC inspirations the uppermost slices of the lung have ventilation at around one third of the slices at the bases. When there is slow inspiration from FRC there is a smaller but still significant gradient of 1.5 :1. In horizontal positions this gradient is significantly reduced. The second regional variation is due to the different time constants of alveoli. The time constant is dependent on the compliance of the alveoli unit and the resistance of the tube supplying it. If the time constants for two units is equal then they will fill and empty at the same rate. The ultimate volume will be dependent only on regional compliances. If however the constants are not equal some units will fill and empty quicker, if inspiration is long enough then all units will eventually fill and again final volume will be dependent on compliance only. Therefore the inspiratory duration (and thus RR) will cause regional variation of ventilation if it is not prolonged. In patients with the commoner forms of lung disease there is usually different time constants and sequential filling and emptying of lung units leading to ventialtion variation. The last cause of regional variation in ventilation is the anatomy of the conducting airways which have been observed to increase ventilation to central lung units.

Perfusion refresher - because pulmonary circulation operates at low pressures, it is partcularly influenced by the force of gravity. This has previously covered in reference to West's zones of the lung and the waterfall analogy. A fourth zone is sometimes described where the weight of the lung reduces flow due to interstital forces at the very based of the lung. The other factor which causes variation is due the the vascualar anatomy and the pattern of branching which mathematical models show contribute significantly to the heterogeneity of flow.

Ventilation/Perfusion Ratio (V/Q) - Whilst the blood flow and ventilation to the lungs is roughly equal each usually between 4-5 litres, in different parts of the lung they are not necessairly equally matched. The effect of gravity which is the most important factor for both V and Q is more significant in terms of perfusion. This leads to a scatter of V/Q ratios which is exacerbated in older subjects even in the absence of lung disease. V/Q ratios at the apex of the lungs (where ventilation is relatively greater than perfusion) has values of 3.3, at the base (where perfusion dominates) is 0.6 in normal subjects. In pathologial states alveolus may recieve no perfusion and thus forms part of the physiological dead space. Examples of this include a pulmonary embolism, or a sudden decrease in cardiac output resulting in decreased perfusion to the apicies. The V/Q ratio will approach infinity. If the alveolus receives no ventilation it represents a form of true shunt and the V/Q ratio will be 0 . Compensatory mechanisms for V/Q scatter include hypoxic pulmonary vasoconstriction discussed previously.



The shunt equation is based on the assumption that the total oxygen carried by the aterial blood may be calculated by adding the oxygen contents of the blood that passes the lungs and the shunted blood. Assuming ideal gas exchange (to calculate Postcap O2 content) it is possible to create this in an equation as follows:



Cardiac Output X Aterial O2 Content = Lung Flow X Postcap O2 Content + Shunt Flow X Mixed Venous O2 Content

Using the abbreviations shown in the diagram this becomes;

$$\dot{Q}T \times Ca_{o_2} = (\dot{Q}S \times Cv_{o_2}) + (\dot{Q}T - \dot{Q}S)Cc_{o_2}$$

rearranged gives the shunt equation;

$$\frac{\dot{Q}S}{\dot{Q}T} = \frac{Cc_{o_2} - Ca_{o_2}}{Cc_{o_2} - Cv_{o_2}}$$

The Bohr equation is based on the assumption that CO2 exchange is ideal (alveolar and post capillary values are equal) therefore any difference in the expired and arterial CO2 is due to dead space.

$$P_{ECO_2}(VT) = P_{ACO_2}(VT - VD)$$

it is commonly rearranged to

$$\frac{VD}{VT} = \frac{P_{ECO_2} - P_{ACO_2}}{P_{ACO_2}}$$

$P_{\text{EXPIRED } CO_2}$(Tidal Vol) = $P_{\text{ARTERIAL } CO_2}$(Tidal Vol - Dead Space)

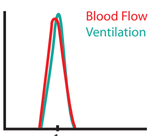
The alveolar equation calculates the ideal alveolar P_{AO_2}. It can then be used to assess the Alveolar-arterial gradient of O2. It is never equal due to V/Q mismatch-ing. The normal value in a 20 year old patient is around 7, but this increases gradually with age, an 70 year old patient having a normal A-a gradient of 17 (the formula is 2.5 + age x 0.21). The alveolar equation needs to account for water vapour and air pressure and is therefore represented by the following equation:

$$PAO_2 = (FiO_2 \times [Patm - PH_2O]) - (PaCO_2 \div R)$$

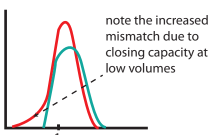
R is usually 0.8
Patm is 760 at sea level
PH2O is 47 at body temp

$$\text{the A-a gradient} = P_{AO_2} - P_{aO_2}$$

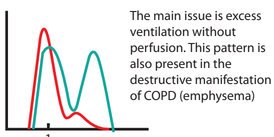
Measurement of V/Q mismatch is problematic. The shunt equation measures **venous admixture** which is the amount of venous blood that is needed to add to the arterial blood to compensate for the difference between ideal and actual O2 content of the post capillary and arterial blood. In reality it consists of both V/Q mismatching and true shunting. A three compartment model aggregates the mismatch compenent with the true shunt component. Response to increasing FiO2 can tease out this difference. VQ Mismatch with correct however true shunt will not, especially if the shunt is >30% Accurate measurement of V/Q relationships is possible using **Multiple Inert Gas Elimination Techniques (MIGET)** which uses six different gases with varialble solubilty to measure the V/Q ratios. This method is very complicated which reduces its utility but it gives a much more accurate picture than the three compartment model.



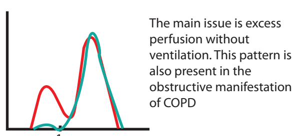
Normal 20 year old Patient



Normal 50 year old patient



Patient with Pulmonary Emboli



Patient with Asthma