## **VENTILATION PERFUSION RATIO**

# @ CBU SCHOOL OF MEDICINE

☐This is the ratio between the volume of alveolar ventilation <i>I</i> minute and the pulmonary blood flow <i>I</i> minute.
□Ventilation/perfusion (V/Q) matching is essential for normal gas exchange in the lungs. For normal gas exchange, alveoli must be in close proximity to pulmonary capillaries i.e. ventilation must be close to blood flow.
☐The V/Q ratio expresses the matching of ventilation (V in L/min)

to perfusion or blood flow (Q in L/min). It is useless if ventilated

are not near ventilated alveoli.

alveoli are not near perfused capillaries, or if perfused capillaries

□Normally, the former is about 4.2 litrers while the latter is about 5.5 litres (= the cardiac output of the right ventricle). Therefore, the average normal V/P is 4.2 I 5.5 = 0.8. ☐ This means for the whole lung, ventilation (L/min) is 80% of perfusion (L/min). ☐ However, V/Q is not uniformly 0.8 throughout the entire normal lung; some regions have higher V/Q and some regions have lower V/Q. □An average V/Q of 0.8 results in an arterial PO2 of 100 mm Hg and arterial PCO2 of 40 mm Hg, the normal values.

□When V (alveolar ventilation) is normal for a given alveolus and Q (blood flow) is also normal for the same alveolus, the ventilation-perfusion ratio (V/Q) is also said to be normal.
□When the ventilation (V) is zero, yet there is still perfusion (Q) of the alveolus, the V/Q is zero Or, at the other extreme, when there is adequate ventilation (V a) but zero perfusion (Q), the ratio V/Q is infinity

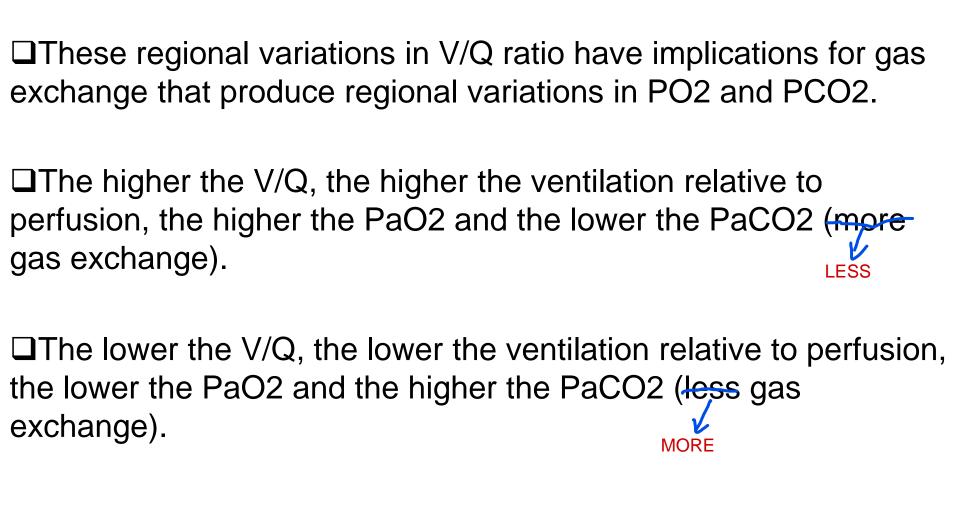
☐At a ratio of either zero or infinity, there is no exchange of gases through the respiratory membrane of the affected alveoli, which explains the importance of this concept

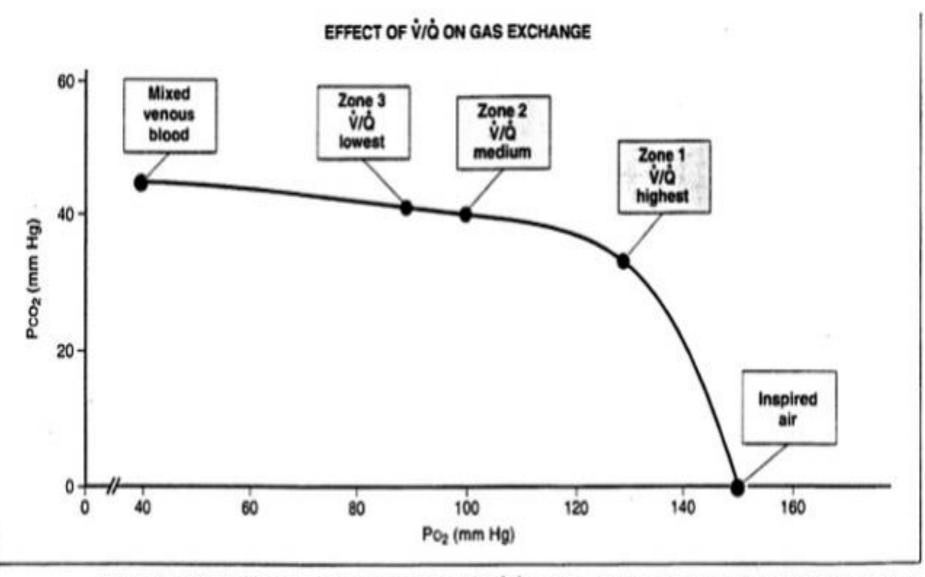
### REGIONAL VARIATIONS IN V/Q

☐ In the upright lung, there are regional variations in both ventilation and blood flow. ☐ Gravitational effects that cause blood flow to be highest at the base and lowest at the apex ☐ There are also regional variations in ventilation that occur in the same direction as those for blood flow; thus, ventilation is highest at the base and lowest at the apex. ☐ However, and importantly, the variations in blood flow are greater than the variations for ventilation, such that the apex has a higher V/Q and base has a lower V/Q.

Apex	Blood Flow (Q)	Alveolar Ventilation (V)	å	Pa <sub>02</sub>	Pa <sub>CO2</sub>
Zone 1	Lowest	Lower	Highest (3.0)	Highest (130 mm Hg)	Lower (28 mm Hg)
Zone 2			-	-	-
Zone 3	Highest	Higher	Lowest (0.6)	Lowest (89 mm Hg)	Higher (42 mm Hg)

Variation in ventilation/perfusion ( $\hat{V}/\hat{Q}$ ) in the three zones of the lung. The effects of regional differences in  $\hat{V}/\hat{Q}$  on Pa<sub>co2</sub> and Pa<sub>co2</sub> also are shown.



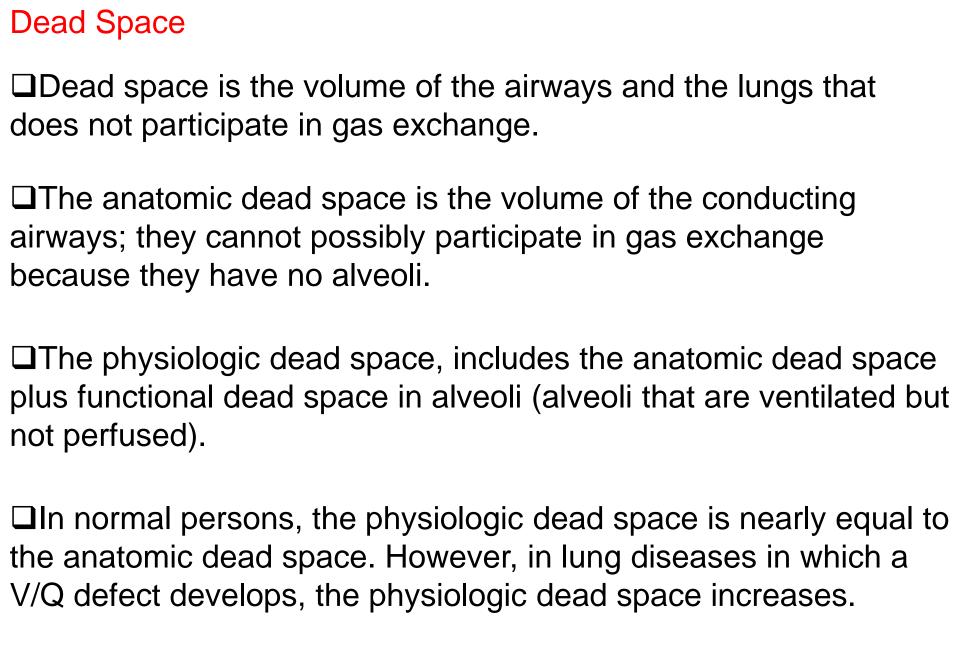


Effect of regional differences in ventilation/perfusion ( $\hat{V}/\hat{Q}$ ) on Pco<sub>2</sub> and Po<sub>2</sub>. Regional differences in Po<sub>2</sub> are much greater than the regional differences in Pco<sub>2</sub>.

#### V/Q DEFECTS

- □V/Q matching means that ventilation and perfusion are "matched up", that ventilated alveoli are close to perfused capillaries, which provides for ideal gas exchange.
- □A mismatch of ventilation and perfusion (called V/Q mismatch or V/Q defect) causes a defect in gas exchange.
- The defect can range from ventilated alveoli that are not perfused (called "dead space") to perfused capillaries that are not ventilated (called "shunt"), and every possibility in between (high V/low Q = high V/Q; low V/high Q = low V/Q). Any V/Q mismatch implies that inadequate gas exchange will occur.

V/a matching Capillary "Dead s pace "Shunt" V/Q=0 1/a=∞ airway ofstruction Pulmonary embolism R->L cardiac V/Q Defects



- ☐So one extreme of V/Q mismatch is called dead space. It refers to alveoli that are ventilated, but not perfused. No O2 or CO2 can be exchanged with air entering these alveoli because there is no blood flow to pick up O2 or to release CO2
- □In regions of the lung where there is dead space, alveolar PO2 and PCO2 approach their values in inspired air.

- □Physiologic dead space is calculated by Bohr's equation, which assumes that
- all of the CO2 in expired air comes from functional alveoli (alveoli that are perfused);
- (2) that inspired air has no CO2, and
- (3) that alveolar and arterial PCO2 are equal.

$$VD = VT x Pa_{CO2} PE_{CO2}$$

$$Pa_{CO2}$$

VD is physiologic dead space (ml), VT is tidal volume, Pa<sub>CO2</sub> is the P<sub>CO2</sub> of arterial blood, and PE<sub>CO2</sub> is the P<sub>CO2</sub> of expired air.

 $\square$ If there is no dead space, then  $PE_{CO2}$  equals  $PA_{CO2}$  (same as PaCO2), and VD comes out to be zero in the calculation

□If dead space is the whole tidal volume then PE<sub>CO2</sub> is zero and VD equals VT in the calculation. (That would be really bad, the person would be dead.)

#### **Shunts**

□Shunts occur when a portion of the pulmonary blood flow bypasses the alveoli; gas exchange cannot occur in shunted blood, i.e., the PO2 and PCO2 of shunted blood equals their values in mixed venous blood.

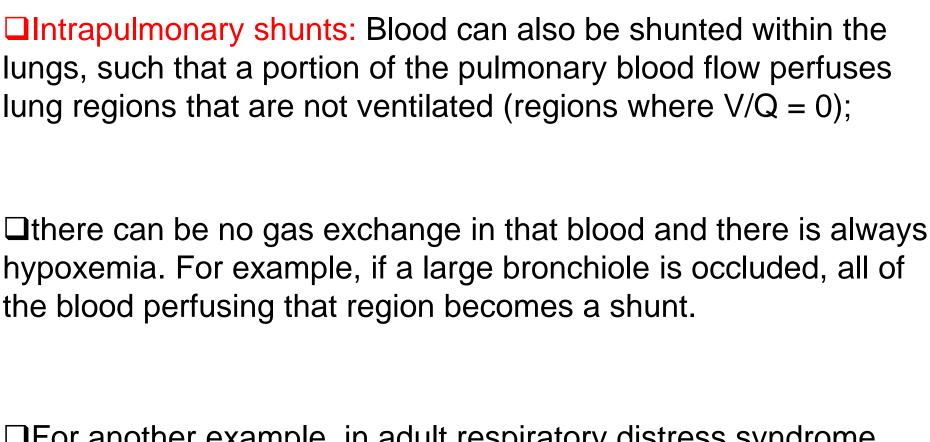
Physiologic shunt: Normally, a small portion (2%) of the pulmonary blood flow bypasses the alveoli (a portion of bronchial blood flow drains into the pulmonary veins and a portion of coronary blood flow drains directly into the left ventricle via the Thebesian veins).

☐ Thus, a small physiologic shunt is always present and causes PaO2 to be slightly less than PAO2, a difference we usually ignore.

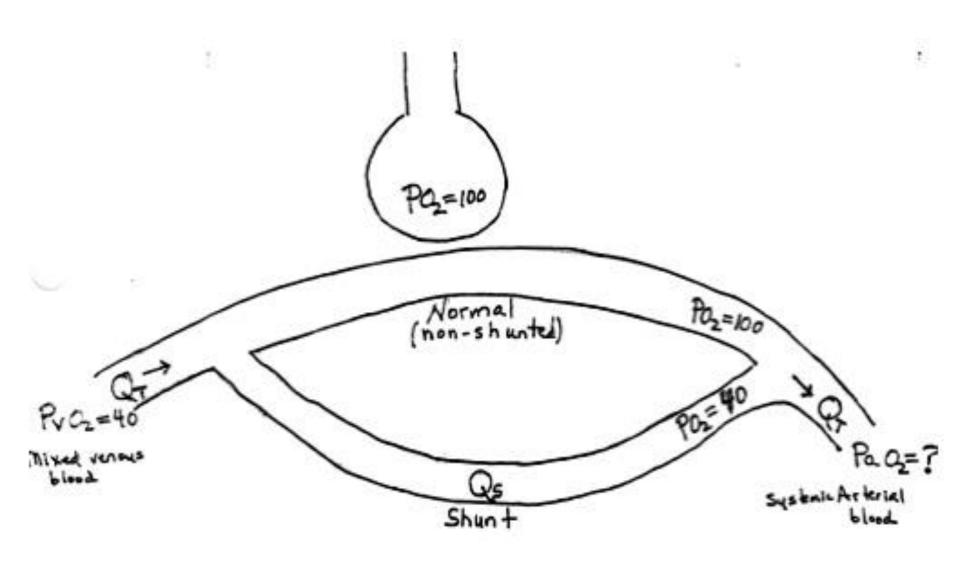
- □Right-to-left cardiac shunts: Defects in the intraventricular septum can result in as much as 50% of the cardiac output being routed from the right ventricle to the left ventricle without going to the lungs for gas exchange.
- □In cardiac right-to-left shunts, there is always hypoxemia (decreased arterial PO2) -- shunted blood is not oxygenated in the lungs and dilutes the non-shunted (normal) blood that is oxygenated.

☐ More common are left-to-right cardiac shunts, which do not cause hypoxemia.

□When blood is shunted from the left heart to the right heart, there is a decrease in cardiac output of the left heart and an increase in cardiac output of the right heart, but no "problem" with oxygenation.	h
☐A portion of the oxygenated blood from the left heart is recycled to the lungs, raising PO2 on the right side of the heart.	b



□ For another example, in adult respiratory distress syndrome (ARDS), certain cytokines released by the lung cause local vasoconstriction and re-route blood to regions that are not ventilated.



## Alveolar-arterial gradient

- □ The Alveolar–arterial gradient (A-aO<sub>2</sub>, or A–a gradient), is a measure of the difference between the alveolar concentration ( $\mathbf{A}$ ) of oxygen and the arterial ( $\mathbf{a}$ ) concentration of oxygen. It is used in diagnosing the source of hypoxemia
- □The A–a gradient helps to assess the integrity of alveolar capillary unit. For example, in high altitude, the arterial oxygen PaO2 is low but only because the alveolar oxygen (PAO2) is also low.
- ☐ However, in states of ventilation perfusion mismatch, such as pulmonary embolism or right-to-left shunt, oxygen is not effectively transferred from the alveoli to the blood which results in elevated A-a gradient

□Even though partial pressure of oxygen is about equilibrated between the pulmonary capillaries and the alveolar gas, this equilibrium is not maintained as blood travels further through pulmonary circulation.

 $\square$ As a rule,  $P_AO_2$  is always higher than  $P_aO_2$  by at least 5–10 mmHg, even in a healthy person with normal ventilation and perfusion.

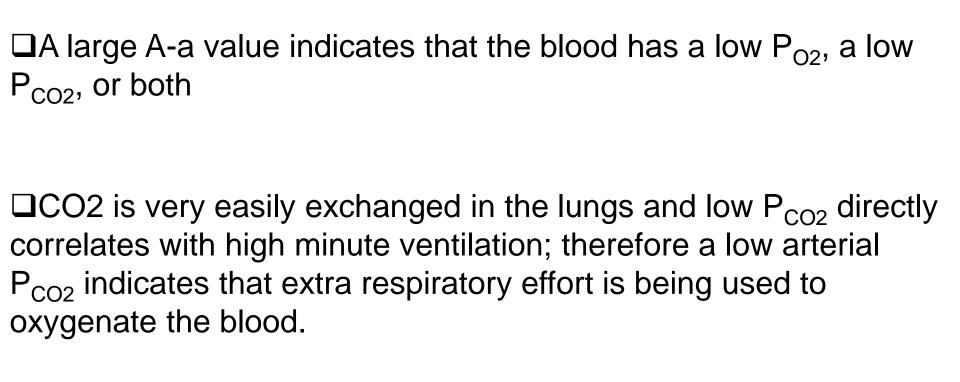
☐ This gradient exists due to both physiological right-to-left shunting and a physiological V/Q mismatch caused by gravity-dependent differences in perfusion to various zones of the lungs.

$$\text{A-a Gradient} = \begin{cases} \left(150 \text{ mmHg} - \frac{5}{4}(P_a \text{CO}_2)\right) - P_a \text{O}_2 & \text{or} \\ \left(20 \text{ kPa} - \frac{5}{4}(P_a \text{CO}_2)\right) - P_a \text{O}_2 \end{cases}$$

The A-a gradient is useful in determining the source of hypoxemia. The measurement helps isolate the location of the problem as either intrapulmonary (within the lungs) or extrapulmonary (elsewhere in the body).

□A normal A—a gradient for a young adult non-smoker breathing air, is between 5–10 mmHg. Normally, the A—a gradient increases with age. For every decade a person has lived, their A—a gradient is expected to increase by 1 mmHg.

- □ A conservative estimate of normal A—a gradient is less than [age in years/4] + 4. Thus, a 40-year-old should have an A-a gradient less than 14. □An abnormally increased A–a gradient suggests a defect in diffusion, V/Q (ventilation/perfusion ratio) mismatch, or right-toleft shunt.
- □Because A–a gradient is approximated as: (150 5/4(pCO2)) PaO2 at sea level and on room air (0.21x(760-47) = 149.7 mmHg for the alveolar oxygen partial pressure, after accounting for the water vapor)



□A low Pa<sub>O2</sub> indicates that the patient's current minute ventilation (whether high or normal) is not enough to allow adequate oxygen diffusion into the blood.

☐the A—a gradient essentially demonstrates a high respiratory
effort (low arterial P <sub>CO2</sub> ) relative to the achieved level of
oxygenation (arterial P <sub>O2</sub> ).

□A high A—a gradient could indicate a patient breathing hard to achieve normal oxygenation, a patient breathing normally and attaining low oxygenation, or a patient breathing hard and still failing to achieve normal oxygenation.

☐ Treatment with 100% O2 tests for a shunt. When a person	with
a shunt (and therefore an increased A - a gradient) breathes	100%
O2, their A - a gradient will remain increased.	

□Although the high O2 treatment will raise the PO2 of the nonshunted blood, the PO2 of the shunted blood remains at the value for mixed venous blood; thus, overall PaO2 remains lower than PAO2, i.e., increased A-a gradient.

□The "quick and dirty" wisdom you will hear is that a shunt is not "treatable" with 100% O2. This wisdom is superficial.
□Correctly speaking, the A - a gradient and O2 delivery are not correctable. The overall PO2 of arterial blood will be somewhat increased by giving 100% O2, but the extent of increase arterial PO2 depends on the size of the shunt.

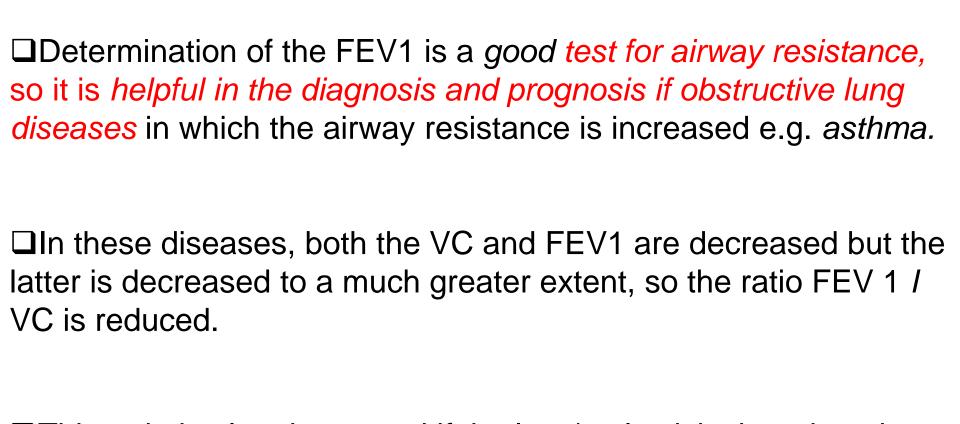
☐ The shunt equation calculates the fraction of total pulmonary blood flow (QT) that is shunted (QS).

 $Q_S = O_2$  content of non-shunted blood - arterial  $O_2$  content  $O_2$  content  $O_2$  content of non-shunted blood - venous  $O_2$  content

- a. Qs is blood flow through the shunt
- Q<sub>T</sub> is total pulmonary blood flow, or cardiac output
- c.  $O_2$  content of non-shunted blood is calculated based on equilibration of that blood with alveolar gas, i.e.,  $P_{O2} = 100 \text{ mm Hg}$
- d. Arterial O<sub>2</sub> content is calculated based on the measured arterial P<sub>O2</sub>
- Venous O<sub>2</sub> content is calculated based on the measured venous P<sub>O2</sub>

## THE TIMED VITAL CAPACITY (TIMED VC OR FEV,)

- ☐A test of vital capacity of the lungs expressed with respect to the volume of air that can be quickly and forcibly breathed out in a certain amount of time.
- ☐ The vital capacity (VC) is normally expelled in about 4 seconds, and the timed VC is the fraction that is expelled during the first second only (so it is also called the forced expiratory volume in one second or FEV1).
- □It is expressed as a % of the VC (i.e. FEV<sub>1</sub>/VC %). Normally, the FEV<sub>1</sub> is at least 80 % of the VC [the normal value after 2 seconds(FEV2) is about 94% while that after 3 seconds (FEV1) is 97 %]



☐ This ratio is also decreased if the lung's elasticity is reduced (e.g. in *emphysema*), but it is *usually normal in restrictive lung diseases* because both the VC and the FEV1 are *frequently decreased equally in these diseases* 

## THE MAXIMAL BREATHING CAPACITY (MBC)

- ☐ The MBC is the maximal volume of air that can be inhaled per minute by the greatest voluntary respiratory effort possible, so it is also called the *maximal voluntary ventilation (MVV)*
- □ It is estimated by measuring the volume of expired air given out by the subject while breathing as deep and as fast as possible in a spirometer.

□Such hyperpnea is performed for only I0-15 seconds then the MBC is calculated per minute. This is because prolonged maximal breathing leads to excessive elimination of C02 which causes alkalosis and may be tetany.

☐ The determination of the MBC is the best test to assess the strength of the respiratory muscles, and its average normal value is 120 (up to 170) litres per minute in young adult males, and is less in females and in old persons.

□ It is affected by the same factors that affect the vital capacity but it is a *better pulmonary function test because in the early stages of certain lung diseases, the VC may be normal while the MBC is decreased.* 

## THE BREATHING RESERVE (BR)

☐ The BR is the *difference between the MBC and the resting*MRV, thus if the MBC is 125 litres and the MRV is 6 litres, then the BR will be 119 litres per minute.

□ Its measurement is a good test for the functional reserve of the respiratory system as well as the state of physical fitness.

#### THE DYSPNEIC INDEX

☐ The ratio: BR I MBC % is normally higher than 90 %, and dyspnea occurs if it drops below about 70 %, so it is also called the dyspneic index.

#### **DYHPNEA**

- □ Dyspnea means difficulty or shortness of breathing of which the person is aware.
- □It develops when the *dyspneic index decreases below about 70* %
- ☐ This occurs due to either a decrease in the MBC or an increase in the MRV (e.g in cases of acidosis and hyperthyroidism). Dyspnea also occurs in the following conditions:
- 1. If the work of breathing increases due to any cause.
- 2. If the mechanical efficiency decreases (e.g. in obese persons).
- 3. In certain psychological conditions.