

RESPIRATORY PHYSIOLOGY ANAESTHESIA TUTORIAL OF THE WEEK 147

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QUESTIONS

Before continuing, try to answer the following questions. The answers can be found at the end of the article, together with an explanation.

1. Lung measurements:

- a) Fowlers method measures physiological dead space
- b) TLC can be measured using a spirometer
- c) the FRC in an average adult is 2.2litre
- d) vital capacity is the volume of air expired from full inspiration to full expiration
- e) helium dilution over estimates FRC in patients' with bullous lung disease

2. The functional residual capacity:

- a) is increased in the obese
- b) is the residual volume plus the inspiratory reserve volume
- c) falls with general anaesthesia
- d) is not affected by posture
- e) falls with increasing age

3. Surfactant:

- a) is a mucopolypeptide
- b) causes a decrease in surface tension
- c) keeps alveoli dry
- d) causes an increase in compliance
- e) production is reduced after a prolonged reduction in pulmonary blood flow

4. A pressure-volume curve can be used for measuring:

- a) the work of breathing
- b) functional residual capacity
- c) anatomical dead space
- d) compliance
- e) respiratory quotient

5. The oxyhaemoglobin dissociation curve is shifted to the left by:

- a) an increase in arterial PCO₂
- b) acidosis
- c) a decrease in 2,3 DPG
- d) carbon monoxide
- e) a fall in temperature

FUNCTIONAL ANATOMY

To maintain gas exchange function, the anatomy of the respiratory system is designed in such a way to make the process as efficient as possible.

The nose, mouth and pharynx conduct air to the larynx, humidify and filter the air gases. The larynx aids phonation and conducts the gas into the trachea which in an adult is about 18mm in diameter and 11 cm in length. It is lined with columnar ciliated epithelium and divides into the left and right major bronchi at the carina (T4). The bronchi divide 23 times in total (23 generations) in order to increase the surface area available for gas exchange.

The first 16 generations are termed the conducting zone, no bronchi in this region take part in gas exchange and this forms the anatomical dead space. In an average adult the volume of this space is about 150ml.

From generation 17 onwards, small alveoli bud off the bronchi, these are where gas exchange takes place. Generation 17 -23 is the respiratory zone where gas exchange occurs. The volume of this zone is about 2-3 litres and there are about 300 million alveoli present within an average lung.

Cells types and functions in the lung

Type I alveolar cells

These cells are derived from type II alveolar cells and provide a thin layer of cytoplasm which covers about 80% of the gas exchange zone

Type II alveolar cells

These cells allow the formation of surfactant and other enzymes

Type III alveolar cells

These cells are the main lung defence system – they are the alveolar macrophages

Surfactant

Surfactant is stored in the lamellar bodies of type II alveolar cells and made up of a mixture of phospholipids, plasma proteins and carbohydrate. It is an amphipathic molecule – with a charged hydrophilic head and hydrophobic tail. Functions of surfactant are:

- Surfactant reduces surface tension within the alveoli which helps to increase the compliance of the lung
- It improves alveolar stability
- It keeps alveoli dry by opposing water movement from the pulmonary interstitium

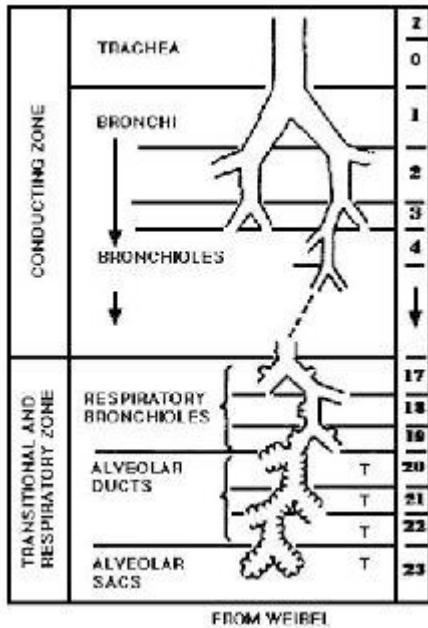


Figure 1: Weibel's classification of the airways - Generations 1 – 23

LUNG VOLUMES

There are many lung volumes which can be measured and used to give information about diagnosis and progression of disease processes as well as a guide to patients' respiratory reserve.

Most volumes are measured using basic spirometry at B,T,P,S (Body Temperature and Standard Pressure) using a Wright's Respirometer.

Key points

- The conducting zone begins at the mouth and continues until the 16th generation. This zone transports gas but plays no part in gas exchange.
- The respiratory zone continues from the 17th until the 23rd generation and is the region where gas exchange occurs.
-

Clinical Points

- The ETT in an adult should lie 1-2cm superior to the carina
- On an X-ray the carina is the point at which the trachea can be seen dividing into the right and left bronchi – around T4
- The right major bronchus divides from the trachea at a much less acute angle than the left making it more prone to endobronchial intubation
- The right upper lobe bronchus arises only a few centimeters from the carina therefore for one lung ventilation a left sided double lumen tube is favoured to avoid the risks of right upper lobe collapse with a right sided double lumen tube

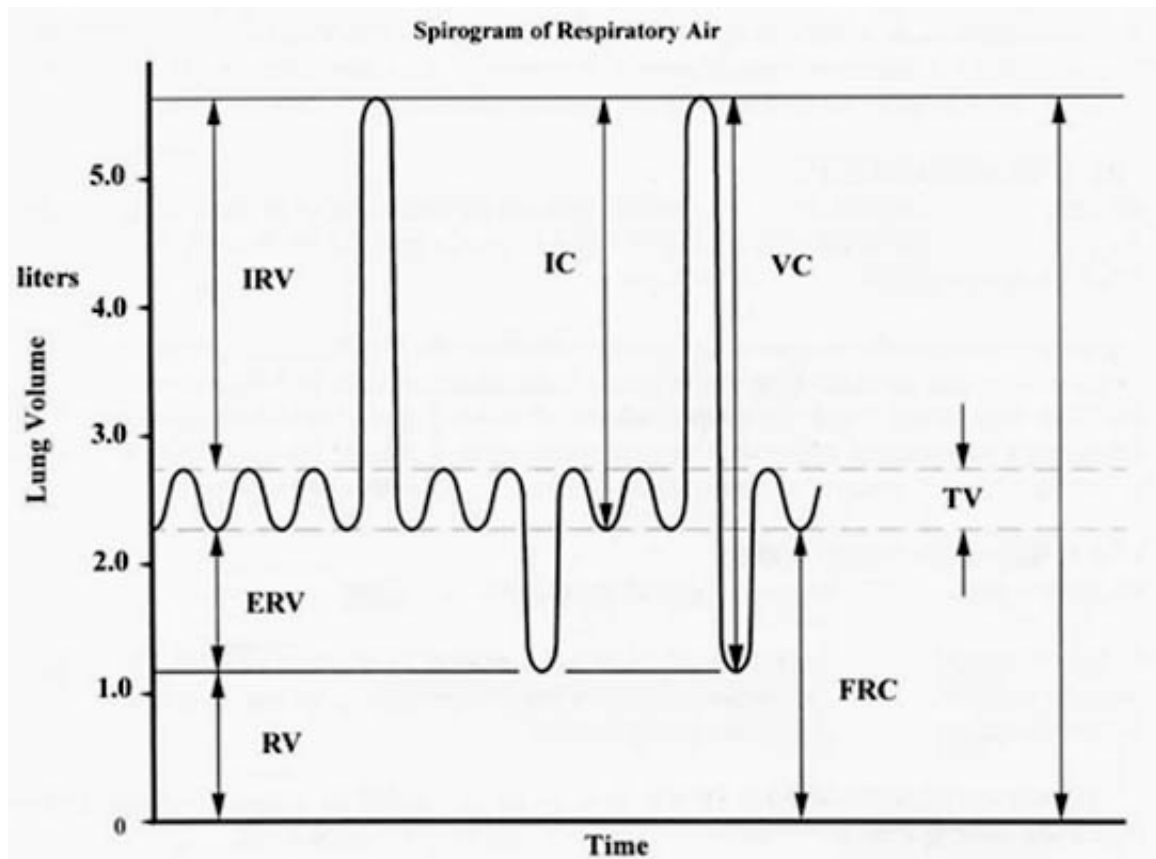


Figure 2: Basic spirometry trace of lung volumes and capacities

Lung volumes vary with age, sex, height and weight, and are formulated into nomograms.

- Residual volume RV - Volume of gas remaining in lungs after a forced expiration (15-20ml/kg)
- Expiratory reserve volume ERV – Volume of gas forcefully expired after normal tidal expiration (15ml/kg)
- Tidal volume TV – Volume of gas inspired and expired during normal breathing (6ml/kg)
- Inspiratory reserve volume IRV – Volume of gas inspired over normal tidal inspiration (45ml/kg)

Any 2 or more volumes added together = a capacity

- Total lung capacity – volume of gas in lungs at the end of maximal inspiration (80ml/kg)
- Vital Capacity – sum of IRV, TV and ERV (60-70ml/kg)
- Functional residual capacity – Sum of ERV and RV (30ml/kg)

Functional Residual capacity

$$FRC = ERV + RV$$

The FRC is the balance between the tendency of the chest wall to spring outwards and the tendency of the lung to collapse. FRC is not the same volume all the time; it can be disrupted by many factors

Factors decreasing FRC

- Age
- Posture – supine position
- Anaesthesia – muscle relaxants
- Surgery - Laparoscopic
- Pulmonary fibrosis

- Pulmonary oedema
- Obesity
- Abdominal Swelling
- Reduced muscle tone – Reduced diaphragm tone will reduce pull away from the lungs
- Pregnancy – Increased abdominal pressure

Factors increasing FRC

- Increasing height of patient
- Erect position – diaphragm and abdominal organs less able to encroach upon bases of the lungs
- Emphysema – decreased elastic recoil of lung therefore less tendency of lung to collapse
- Asthma – air trapping

Measurement of FRC

A spirometer is unable to measure TLC, FRC and RV.
There are 2 methods used to measure FRC:

1. Helium dilution

The patient is connected to a spirometer containing a known volume of gas (V1) with a known concentration of helium (C1)

Helium is not metabolised by the body.

The patient begins to breathe normally. The helium concentration will change as the helium gets diluted in a larger volume of gas due to the patient's lung volume. (V1 + FRC)

The final helium concentration is measured.

The value for FRC can then be derived since 3 of the 4 values are known.

$$C1 \times V1 = C2 \times (V1 + V2)$$

$$V2 = V1 (C1 - C2) / C2$$

This method measures the volume of gas in the lungs which is participating in gas exchange. It will underestimate the FRC if there are significant areas of gas trapping.

2. Body Plethysmography

FRC is measured by placing the patient in a closed chamber and measuring the pressure and volume changes occurring when the subject makes an inspiratory effort against a closed airway. Boyle's gas law ($P1V1 = P2V2$) is applied as ventilation takes place to derive the FRC.

This technique also takes into account any gas trapped behind closed airways e.g. in the case of patients with emphysematous bullae. Helium dilution techniques do not calculate this.

Closing capacity (CC)

This is the volume at which the small airways close during expiration. Under normal circumstances the FRC is always greater than the CC however if the FRC was to decrease then this would no longer be the case and the small airways may close at the end of normal tidal expiration. This leads to hypoxaemia, atelectasis and worsening gas exchange due to increasing V/Q mismatch.

Closing capacity increases with age. Typically closing capacity is equal to FRC at the age of 66 in the erect position or 44 in the supine position.

Preoxygenation

The major oxygen store within the body is the Functional Residual Capacity. A typical volume for FRC is about 2.2 litres in an average adult and normally contains 21% oxygen. Since total body oxygen consumption is about 250mls per minute this normal store of oxygen will only last just over 1 minute with apnoea. Preoxygenation is defined as breathing 100% oxygen from a close fitting mask for 3-5 minutes. Breathing 100% oxygen for this time will denitrogenate the lungs and increases the oxygen store to in excess of 1800mls thus increasing the time to desaturation to about 7-8 minutes assuming an oxygen consumption of 250mls/min.

Key points

- Spirometry can be used to provide values for all basic lung volumes except TLC, RV and FRC
- FRC is determined by the balance between lung collapse and chest wall springing outward
- FRC affects oxygenation
- FRC is the main oxygen store in the body and can be easily and quickly enriched with oxygen
- FRC will change due to a large number of factors

Clinical points

- The FRC is reduced by anaesthesia and therefore hypoxia is common in a patient with a decrease in FRC. The application of PEEP enables the FRC to remain greater than CC and improves oxygenation
- PEEP maintains the lungs on the steep part of the compliance curve which lessens collapse at the bases of the lungs.
- Preoxygenation should take place for 3-5 minutes or 4 vital capacity breaths
- Preoxygenation is essential in patients likely to have a decreased FRC e.g. pregnant, obese
- Adequacy of preoxygenation can be assessed by monitoring end tidal oxygen – aim for $ETO_2 > 90\%$

VENTILATION

Total ventilation (MV) = $V_T \times RR$

With each tidal volume about a third the total amount of gas flowing into the airway and lung does not participate in gas exchange. This is the physiological dead space.

What is dead space?

Dead space can be defined as a volume of gas which does not take part in gas exchange.

Dead space can be classified into 3 types

1. Anatomical dead space

This includes any breathing system or airway plus mouth, trachea and the airways up until the start of the respiratory zone. The typical volume in an adult is about 150mls (See section 1)

2. Alveolar dead space

This occurs when areas of the lung are being ventilated but not being perfused and this leads to what is known as V/Q mismatch.

Large increases in alveolar dead space commonly occur in the following conditions: pneumonia, pulmonary oedema, pulmonary embolism

3. Physiological dead space

This is a combination of alveolar and anatomical dead space added together.

Dead space is usually 30% of V_T

Measurement of dead space

Fowlers method

This is used to measure anatomical dead space.

A patient takes a breath of 100% oxygen to rid the conducting zone gases of nitrogen and then exhales through a mouthpiece capable of analysing nitrogen concentration at the lips.

Initially the exhaled gases contain no nitrogen as this is dead space gas; the nitrogen concentration will increase as the alveolar gases are exhaled. Nitrogen which is measured following the breath of 100% oxygen must then have come only from gas exchanging areas of the lung and not dead space.

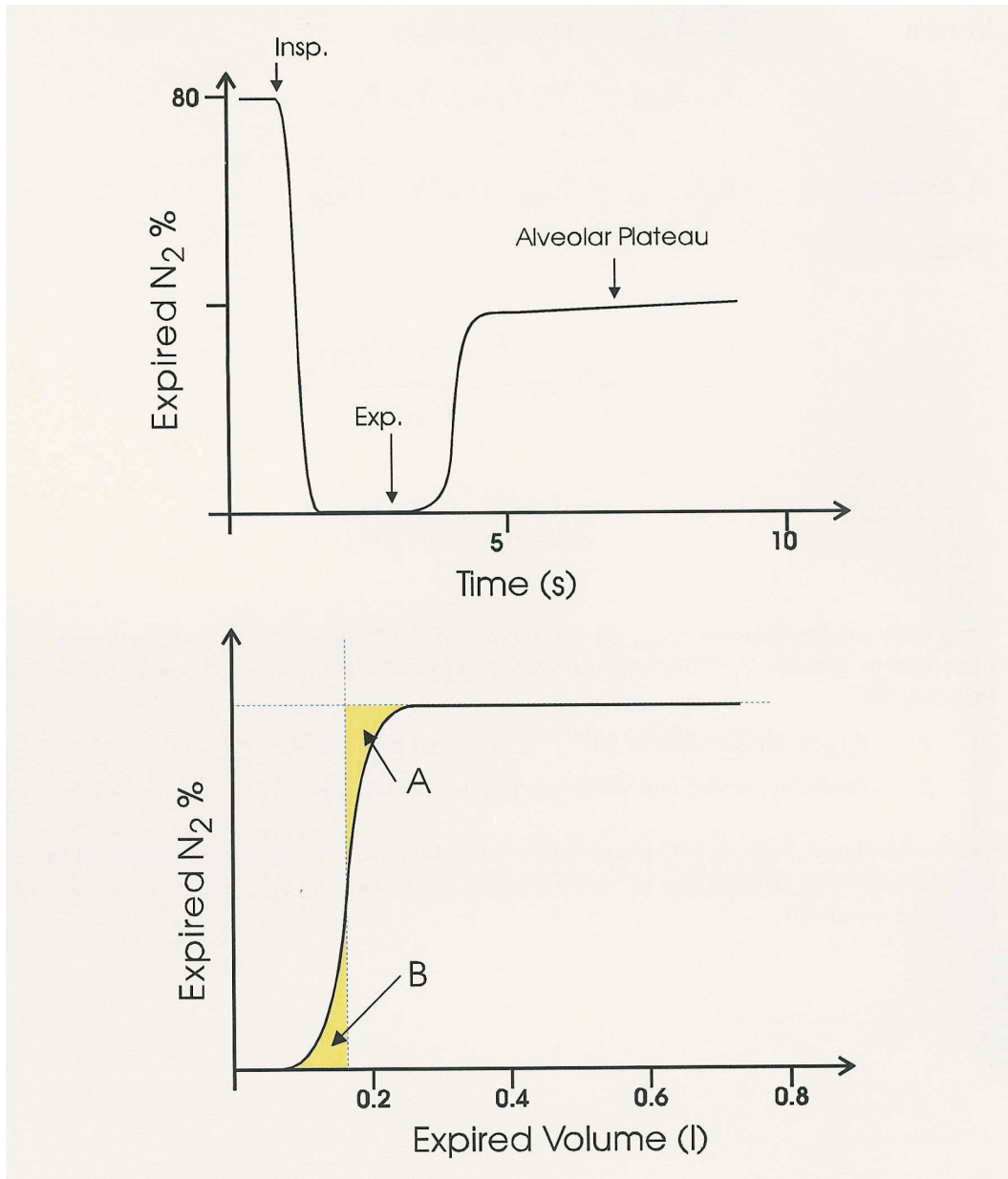


Figure 3: Fowlers method. Initially expired nitrogen concentration is plotted against time as seen in top graph. To calculate the anatomical dead space expired nitrogen is plotted against volume. The upstroke of the curve is equally divided in half to give areas A and B. The anatomical dead space equals volume 0 up to and including area B.

The Bohr Equation

This measures physiological dead space. It is a complicated equation based upon the fact that all CO₂ comes from alveolar gas and the exhalation of CO₂ can therefore be used to measure gas exchange or lack of gas exchange if there is alveolar dead space (no perfusion of these alveoli).

For each tidal volume there will be a proportion of dead space (anatomical) but the amount of gas that is left over should take part in gas exchange. In order to derive the equation:

FACO₂- Alveolar CO₂
FeCO₂-CO₂ from mixed expired gases
VT- Tidal volume
VD- Dead space volume (Physiological)

In an adult with normal lungs the value for VD/VT is between 0.20 and 0.35

The expiration of CO₂ is calculated by either

$$VTCO_2 = V_A \times FACO_2$$

or

$$VTCO_2 = V_T \times FeCO_2$$

As we said above though with each breath there will be a component of dead space, therefore:

$$V_A = V_T - V_D$$

Therefore:

$$(V_T - V_D) \times FACO_2 = V_T \times FeCO_2$$

This can be expressed as:

$$V_D/V_T = (FACO_2 - FeCO_2)/FACO_2$$

There is not really a truly ideal value for FACO₂ that can be used in the equation and therefore arterial pCO₂ is used. This is substituted into the equation giving:

$$V_D/V_T = (PaCO_2 - PeCO_2)/PaCO_2$$

Key points

- Approximately 1/3 of VT is dead space
- There are 3 types of dead space: Anatomical, Physiological and Alveolar
- Physiological dead space is a combination of alveolar and anatomical dead space
- Anatomical dead space is about 150mls in an average adult and is measured by fowlers method
- Physiological dead space is measured using the Bohr equation

Clinical points

- Minimise dead space by using the correct sized equipment e.g HME, breathing circuits
Consider the application of PEEP to prevent atelectasis and V/Q mismatch

RESPIRATORY MECHANICS

Respiratory muscles

Inspiratory muscles

- Diaphragm – very powerful, has the ability to contract 10cm in forced inspiration
- External intercostals – pull the ribs up and forwards
- Accessory inspiratory muscles – scalene muscles (elevate first 2 ribs) and sternomastoids (raise the sternum)
- Muscles of neck and head (seen in small babies in respiratory distress)

Expiratory muscles

Expiration is usually passive and relies on the elastic recoil of the lungs and the chest wall. Under anaesthesia or extreme exercise expiration may become active due to the activation of abdominal muscles. Muscles have their use in forced expiration.

- Abdominal wall muscles – rectus abdominus, internal and external oblique
- Internal intercostal muscles – pull ribs down and inwards

Compliance

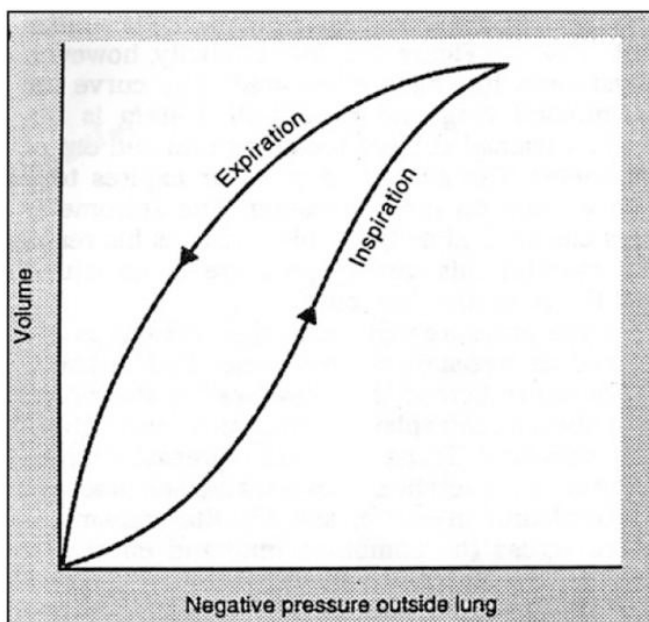
Compliance is defined as the volume change per unit pressure change and is usually expressed in ml/cmH₂O

$$\text{Compliance} = \Delta V / \Delta P$$

It is classified into chest wall, lung or total lung compliance. (Distensibility)

Compliance can be measured by inserting an oesophageal probe into a co-operative patient, the patient inhales and exhales to a set volume. At each volume the intrapleural pressure is estimated using the oesophageal probe. A pressure volume curve can then be plotted.

If during the measurement process no gas flow occurs at each set volume then this is static compliance. (Gas flow ceases and equilibration occurs) If gas flow continues throughout measurement then this is dynamic compliance.



Critical Care

Figure 4: This graph illustrates lung compliance.

Using a spirometer certain fixed volumes can easily be measured e.g. TLC, RV and FRC.

The pressure along the x axis is often plotted as the transpulmonary pressure (Alveolar pressure – Intrapleural pressure)

Initially as can be seen from the above curve at lower lung volumes the compliance of the lung is poor and greater pressure change is required to cause a change in volume. This occurs if the lungs become collapsed for a period of time.

At FRC compliance is optimal since the elastic recoil of the lung towards collapse is balanced by the tendency of the chest wall to spring outwards.

At higher lung volumes the compliance of the lung again becomes less as the lung becomes stiffer.

Expiration is deemed a passive process due to the elastic recoil of the lung; because of this the inspiratory curve is not identical to the expiratory curve on a correctly drawn compliance curve. This is known as hysteresis.

Compliance increases in old age and emphysema as elastic lung tissue is destroyed. It is decreased in pulmonary fibrosis, pulmonary oedema, atelectasis and in the extremes of lung volume.

Work of breathing

The energy required for the work of breathing is mainly used in the process of inspiration as energy is required to overcome airway resistance, the elastic recoil of the tissues and the chest wall and tissue resistance.

The energy stored within the elastic tissues is used to provide for expiration.

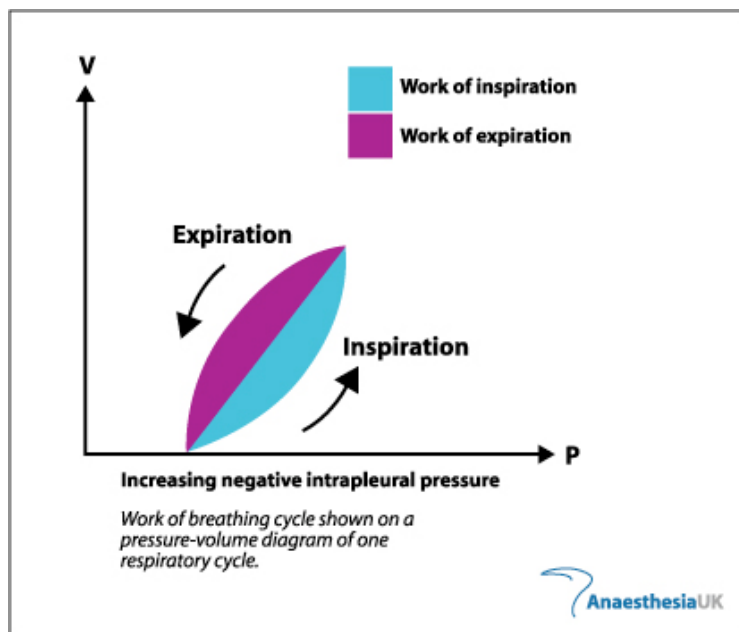


Figure 5: The work of breathing

Key Points

- Expiration is normally a passive process
- Compliance is the change in volume per unit change in pressure
- Compliance can be dynamic or static depending on whether the gas flow is continuing or allowed to equilibrate during pressure measurements
- A spirometer cannot measure TLC, FRC or RV
- Compliance of the lungs is poor at very low or very high lung volumes
- Compliance is optimal at or just above FRC

Clinical points

- Patients using their accessory muscles may indicate increased work of breathing
- PEEP can help to maintain the lungs at FRC
- If used correctly CPAP can reduce the work of breathing by increasing FRC

PERFUSION OF THE LUNG

Blood flow to and around the lung is similar to any other organ but at much lower pressures than the systemic system (see diagram). The blood vessels in the lungs continually branch and get consistently smaller very like the branching of the airways. The pulmonary arteries whose walls are very thin in comparison to that of the arteries in the main circulation feed the lung up to the level of the terminal bronchioles and then split into the capillary bed. The capillaries have great capability to distend thus enhancing gas exchange and reservoir action. Once the red blood cells have become oxygenated the capillary bed is drained into venules which then join to form the pulmonary veins. It is the ability of the blood vessels to distend and be recruited which allows the pressures in the pulmonary system to stay low despite very high blood flow.

The pulmonary arteries only supply blood flow and oxygen to the lungs and must have the ability to accept huge blood volumes at times. The low pulmonary pressures are important to minimise the work of the right heart.

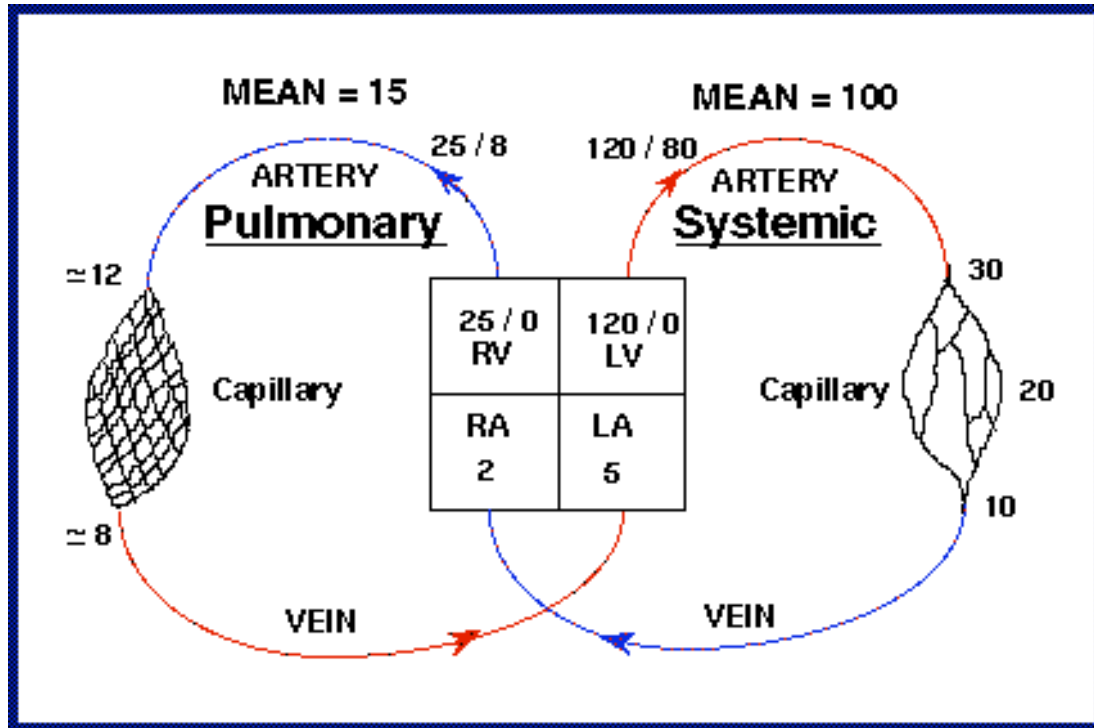


Figure 6: The pressures within the main and pulmonary circulatory systems

PULMONARY VASCULAR RESISTANCE

$$PVR = 80 \times (MPAP - PCWP) / CO$$

Acute and chronic lung disease

Both can cause an increase in PVR. Chronic PVR can lead to right sided heart failure

Lung volumes

PVR depend on the alveolar and extra-alveolar resistances. Both low and high lung volumes will increase PVR, it is at its lowest at FRC.

Alveolar vessels or pulmonary capillaries will become overly stretched longitudinally at high lung volumes and therefore their resistance will increase.

With extra – alveolar vessels resistance will increase at low lung volumes since they are not encouraged to expand by lung parenchyma.

PVR can also be altered through the distension and recruitment of pulmonary vessels which have a large ability to increase their capacitance.

Hypoxic pulmonary vasoconstriction

In response to episodes of low partial pressure of alveolar oxygen the lung will divert its own blood supply to areas of properly ventilated lung tissue. This is important in matching and maintaining ventilation to perfusion in the lung. HPV is also obtunded by the general anaesthetic inhalational agents, it is relatively preserved by TIVA.

Metabolic substances

Endothelin – released from the lung endothelial cells in response to tissue damage – is a very potent vasoconstrictor

Prostacyclin – a naturally occurring vasodilator

Nitric oxide – Also known as endothelin derived relaxing factor – a potent vasodilator

Perfusion throughout the lungs

Taking all of the above into account it is easy to understand then why blood flow is not always even throughout the lung. Gravity plays a large part in directing blood flow by setting up a hydrostatic pressure gradient which is higher at the base of the lung than at the top. Blood is preferentially directed to the lung bases.

As well as the affect of gravity on perfusion and ventilation, the differing pressures within the alveoli, arteries and venous systems heavily influences the outcome. The relationship between these factors describes West zones.

West zones within the lung are 3 vertically split zones (in the upright subject) which explain how alveoli, arterial and venous pressures differ in each zone and thus affect perfusion and ventilation throughout the lung.

In zone 1 the alveolar pressure may exceed that of the arterial and venous pressure and thus little perfusion will occur as the vessels collapse, this then leads to dead space.

In zone 2 the arterial pressure will exceed that of the alveoli pressure but not the venous pressure.

In zone 3 both the arterial and venous pressures exceed the alveoli pressure.

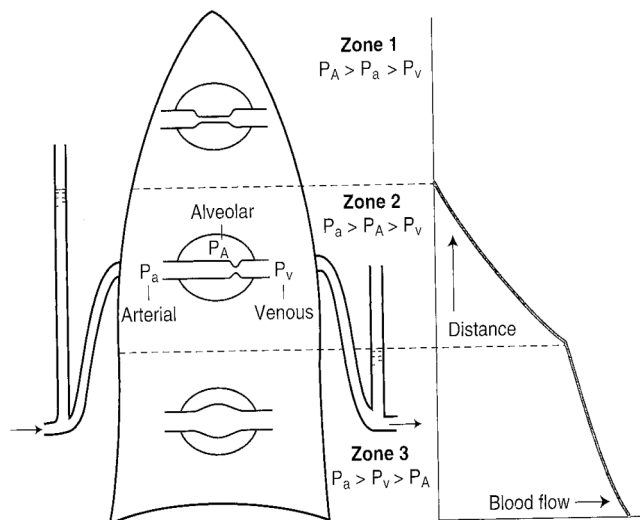


Figure 7: West Zones

Key points

- The lung is a low pressure system
- Low pulmonary pressure minimises the work of the right heart
- The pulmonary vessels distend and recruit to maintain low pressures
- Very high and very low lung volumes increase pulmonary vascular resistance
- Hypoxic pulmonary vasoconstriction diverts blood flow away from poorly ventilated regions of lung
- HPV is obtunded by anaesthesia

Clinical points

- Ventilatory strategies can prevent increases in pulmonary pressures e.g low tidal volumes, permissive hypercapnia
- TIVA does not affect HPV

GAS EXCHANGE

The partial pressure of oxygen that is inhaled from our natural environment through normal inhalation is not maintained at the same partial pressure by the time it reaches the alveoli and indeed the mitochondria. The process by which this decrease in partial pressure occurs is called the oxygen cascade.

The air surrounding us – how much oxygen does it contain?

Dry atmospheric air gas – 21% of 100KPa

Therefore 21 KPa or 160mmHg

However as gas is inspired it is diluted by water vapour which reduces the partial pressure of oxygen

Water vapour – 6.3 KPa/ 47mmHg

$$PO_2 = 0.21 \times (760-47) = 149\text{mmHg}$$

$$PO_2 = 0.21 \times (100-6.3) = 19.8\text{KPa}$$

When the gas reaches the alveoli the partial pressure of oxygen will again decrease as some oxygen is absorbed and CO₂ is excreted. The partial pressure at this point in the oxygen cascade can be determined by using the alveolar gas equation.

$$PAO_2 = PIO_2 - PACO_2/RQ$$

The RQ stands for respiratory quotient and is normally 0.8. It is determined by the amount of CO₂ produced/ oxygen consumed.

$$PAO_2 = 0.21 - 5/0.8 = 14\text{KPa (106mmHg)}$$

Again when the gas reaches the arterial blood a further small drop in partial pressure will have occurred as blood known as venous admixture with a lower oxygen content mixes with the oxygenated alveolar blood.

Venous admixture is made up of blood that has passed through poorly ventilated regions of lung and thus has a lower O₂ partial pressure. Venous admixture is also composed of venous blood which has drained the lungs and left side of the heart. This blood is known as true shunt and drains directly into the left side of the heart.

Extraction of oxygen from this blood then causes the end capillary oxygen partial pressure to be 6-7KPa (40 - 50mmHg)

In the mitochondria the PO₂ varies hugely from 1-5KPa (7.5-40mmHg)

This provides us with an explanation for the following graph, the oxygen cascade.

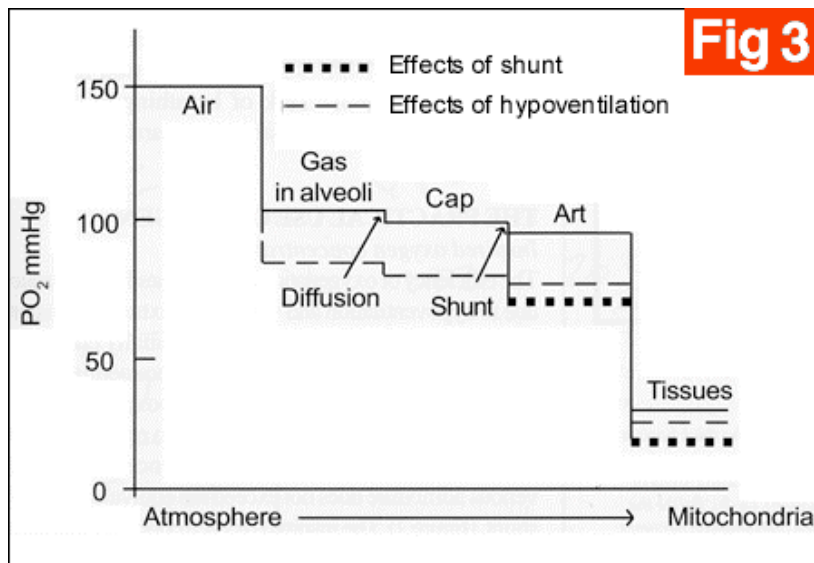


Figure 8: Diffusion of gases from alveoli to blood – the oxygen cascade

Diffusion of Gases Across the Alveolar Membrane

The speed and ease of diffusion are controlled by the laws of diffusion.

Fick's law of diffusion states that gas transfer across a membrane is directly proportional to the concentration gradient.

Graham's law states that diffusion of a gas is inversely proportional to the square root of the molecular weight of the molecule.

Other factors which increase diffusion:

- Large surface area
- Thin membrane
- High solubility

The following equation incorporates the important factors

Diffusion is proportional to $A/T \cdot D \cdot (P_1 - P_2)$

A = Area

T = Thickness

D = Diffusion constant

$P_1 - P_2$ = Concentration gradient

Diffusion in the lungs can be limited in the presence of disease states e.g pulmonary oedema and thickening of the alveolar membrane in pulmonary fibrosis.

Oxygen transport

Oxygen is carried in 2 forms in the blood:

- Oxygen combined to haemoglobin (97%)

Haemoglobin molecule consists of 2 alpha and 2 beta chains; each chain is formed from an iron-porphyrin molecule - haem. Each haemoglobin molecule can bind 4 oxygen molecules (20ml oxygen per 100ml blood) or 15ml oxygen per 100ml in venous blood

- Oxygen dissolved in the blood – this accounts for a minimal amount (0.3ml per dl)

The amount dissolved obeys Henry's law – amount is proportional to the partial pressure
0.023ml per kPa per 100ml blood

Oxygen content in the blood

Total content of oxygen in the blood can be calculated from the Oxygen flux equation:

Flux = $(CO \times Hb \times \text{Saturation} \times \text{Huffners constant}(1.39)) + (0.023 \times PO_2)$

Oxygen dissociation curve

- Sigmoid shaped curve relating the fact that binding of oxygen to the haemoglobin molecule is a co-operative process
- Describes the relationship of saturation of haemoglobin with oxygen at varying partial pressures
- Be aware of the P_{50} – (point at which Hb is 50% saturated)
- Decreasing pH, increasing temperature, 2,3-DPG and CO_2 tension will cause a right shift of the curve
- Increased pH, and reduction in CO_2 tension, temperature and 2,3-DPG produce a left shift of the curve
- If a right shift occurs the Hb molecule is more likely to offload oxygen to the tissues
- In a left shifted situation the Hb is less likely to release oxygen to the tissues

Haemoglobin Oxygen dissociation curve

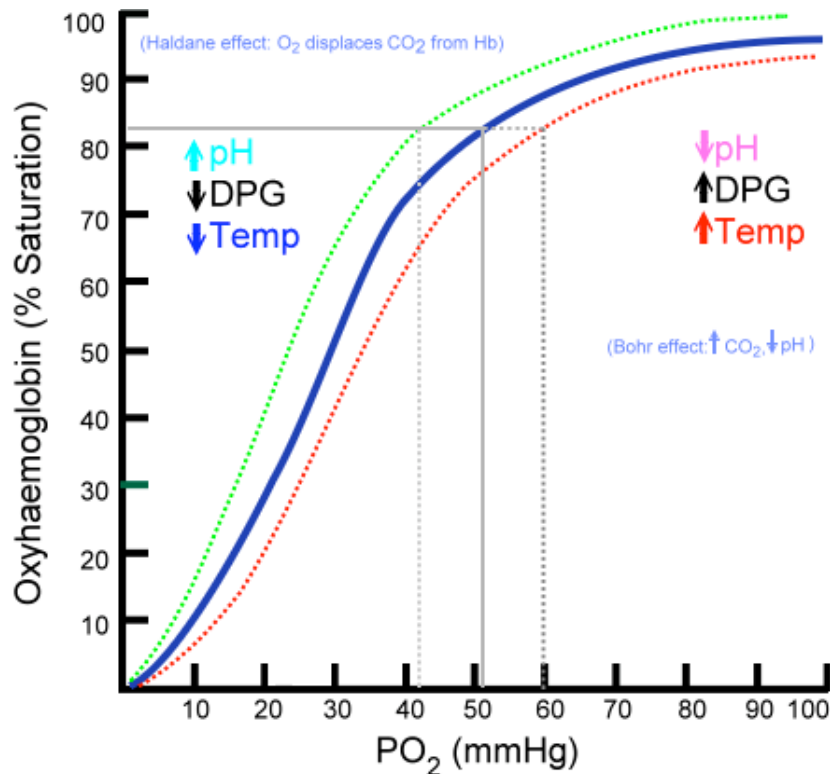


FIGURE 9: The Oxyhaemoglobin dissociation curve

2,3-DPG

This molecule binds to deoxygenated Hb – it reduces the affinity of haemoglobin for oxygen and therefore ensures offloading of oxygen to the tissues.

The Bohr Effect

This describes the affect that CO₂ has on influencing the release of oxygen to the tissues. On entering red blood cells the following reaction occurs:



An increase in H⁺ will cause an acidosis and therefore encourage the release of oxygen from Hb. In the lungs where the CO₂ is being removed, the alkalosis will encourage the uptake of oxygen.

Oxygen Delivery (DO₂)

This can be calculated as follows:

Cardiac output X arterial O₂ content

Carbon dioxide transport

Carbon dioxide is carried in the blood in 3 ways:

- As bicarbonate – 90%
- As dissolved CO₂ – 5%
- As carbamino compounds – 5%

Carbamino compounds are formed by the reaction of the CO_2 with terminal amino groups of proteins and side chains of arginine and lysine. Haemoglobin is essential for this process to occur since it has 4 amino groups per molecule. Albumin also provides amino groups but only 1 per molecule.

The Haldane effect

This phenomenon refers to the increased ability of blood to carry CO_2 when haemoglobin is deoxygenated. Deoxyhaemoglobin is 3.5 times more effective than oxyhaemoglobin in forming carbamino compounds.

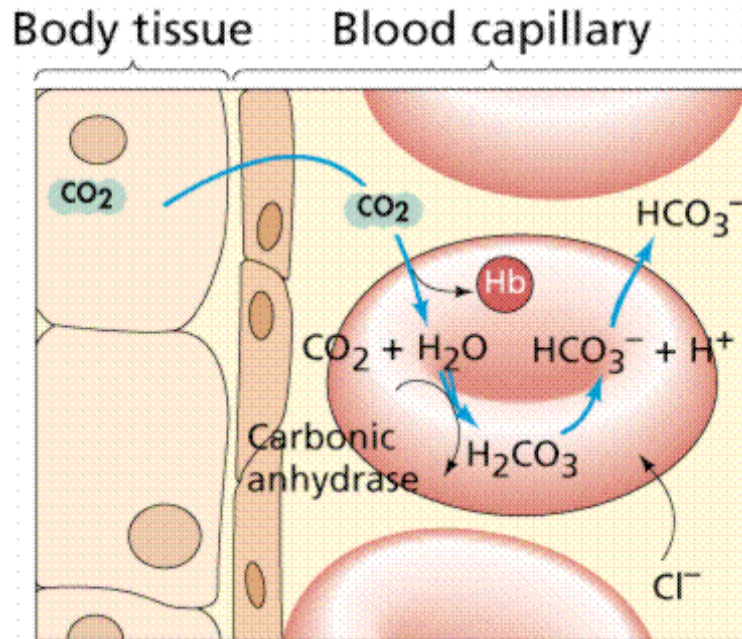


Figure 10: Carbon dioxide transport in blood

Ventilation perfusion Relationships

As mentioned earlier any discrepancy between ventilation and blood flow in the lung will result in V/Q mismatch and potentially dangerous irregularities in gas exchange. If flow of blood to the lung units is to match that of ventilation to the same unit then the ratio of ventilation to perfusion should be in a ratio of 1:1

If the lung is being underventilated but perfused as normal then we say that the V/Q ratio is <1

If the lung is under perfused then the V/Q is >1

Even in a normal lung the V/Q ratio is not uniformly 1 throughout the lung as perfusion and ventilation both have favoured parts of the lung.

Differences between the apices and bases of the lungs

At the apices there is less ventilation than the bases as alveoli are already very stretched however there is proportionally less perfusion therefore the overall V/Q ratio is higher compared to the base of the lung.

Blood flow is directly affected by gravity and naturally has a tendency to flow to the bases of the lungs thus V/Q ratios towards the lower segments of the lung are usually greater than 1.

The vertical change in V/Q ratios in the lung is because although both ventilation and perfusion increase from top to bottom of the lung, perfusion increases much quicker than ventilation. Thus the V/Q ratio at the top of the lung is 3.3 whereas at the bases it is around 0.6. See the following illustration (figure 11).

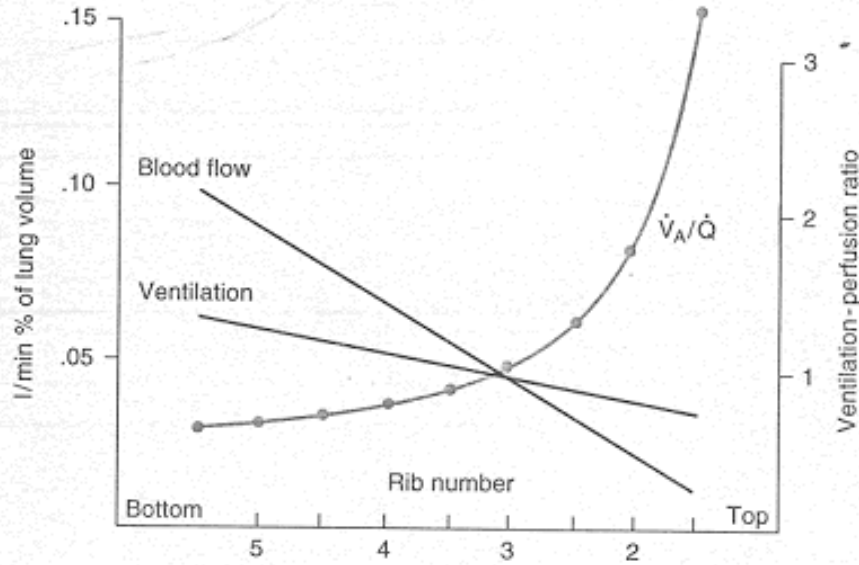


Figure 5-8. Distribution of ventilation and blood flow down the upright lung (compare Figures 2-7 and 4-7). Note that the ventilation-perfusion ratio decreases down the lung.

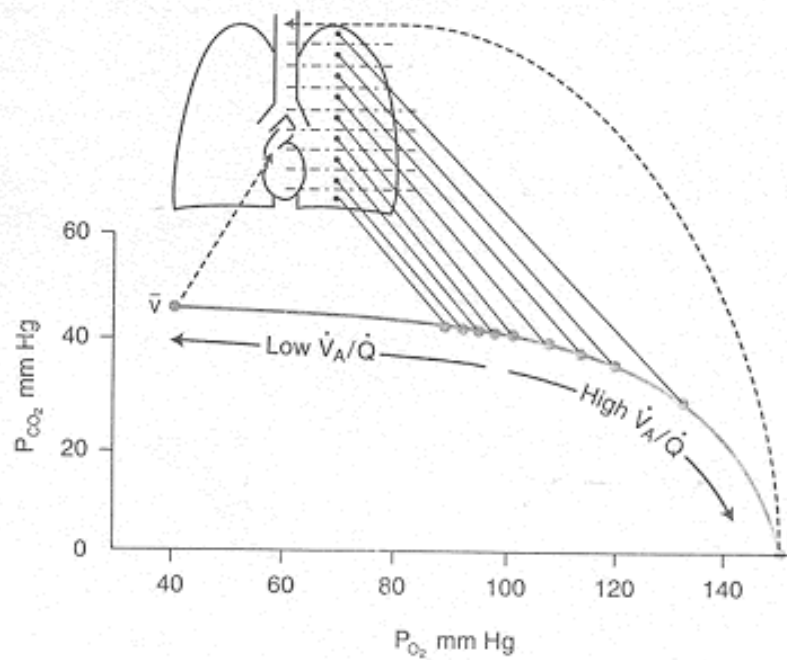


Figure 5-9. Result of combining the pattern of ventilation-perfusion ratio inequality shown in Figure 5-8 with the effects of this on gas exchange as shown in Figure 5-7. Note that the high ventilation-perfusion ratio at the apex results in a high P_{O_2} and low P_{CO_2} there. The opposite is seen at the base.

Figure 11: Ventilation perfusion relationships in the lungs

Alveolar –Arterial P_{O₂} gradient

The value for the A-a gradient gives the clinician some idea about the amount of VQ mismatch and shunt that is present in the lungs. A typical normal value would be around 0.5-1 KPa (5mmHg) though values up to around 15mmHg may be accepted.

It is calculated as PAO₂ – PaO₂. The PAO₂ is calculated using the alveolar gas equation.

Shunt

True shunt refers to a VQ = 0. That is to say that blood has passed through areas of the lung where no ventilation is occurring. As discussed earlier VQ mismatch is also referred to as shunt. Blood passes through areas of the lung which are poorly ventilated ie VQ<1.

Physiological shunt refers to the amount of venous admixture which is directly added to main circulatory blood without having passed through the oxygenating mechanism of the lung. Blood from the bronchial veins draining the lung parenchyma and the thebesian veins draining the cardiac muscle represent the physiological shunt (around 5% of cardiac output.)

The shunt equation allows calculation of the amount of shunt present in an individual subject.

The Shunt Equation:

Q_s = Shunted blood flow

Q_t = Cardiac output

Q_t-Q_s = Blood flow through the lungs minus the shunted blood

Cc_{O₂} = Oxygen content of end pulmonary capillary blood

Ca_{O₂} = Oxygen content of arterial blood

Cv_{O₂} = Oxygen content of mixed venous blood

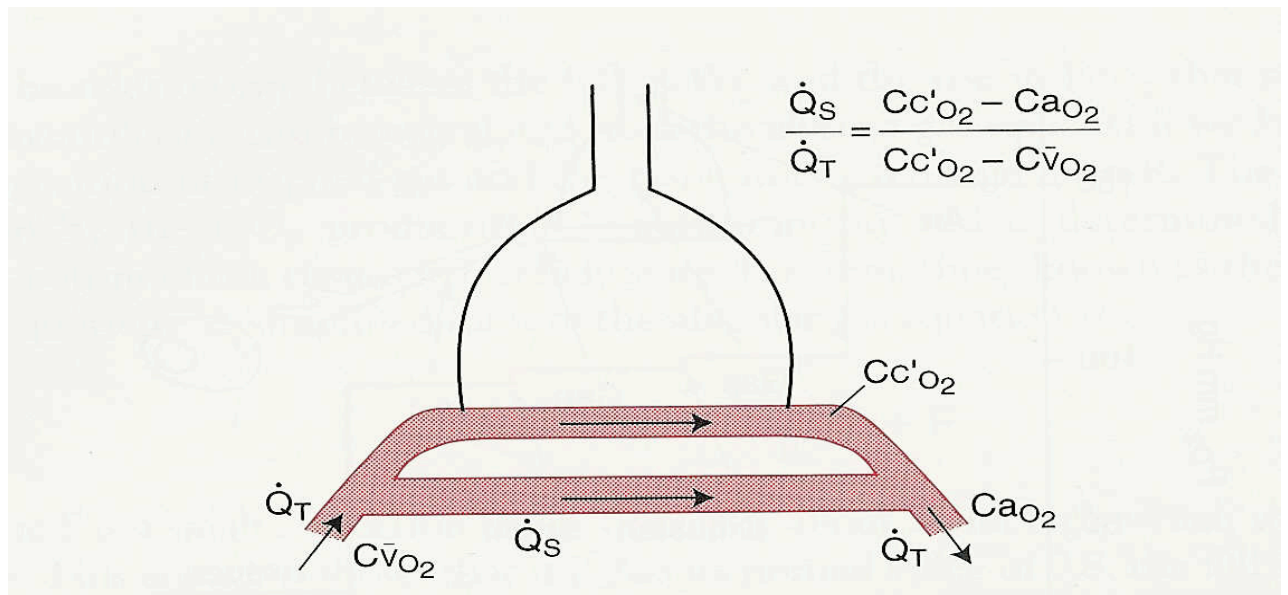


Figure 12: A visualization of the shunt equation

The amount of oxygen leaving the lungs is Q_t x CaO₂. This is equal to the shunted blood flow plus the oxygen content from the lung which would be (Q_s x CvO₂) + (Q_t-Q_s) x CcO₂. (shunt flow x mixed venous O₂ content + pulmonary capillary flow x pulmonary capillary O₂ content).

When these equations are rearranged it provides the classic shunt equation:

$$Q_s/Q_t = CcO_2 - CaO_2 / CcO_2 - CvO_2$$

Key points

- The amount of oxygen inhaled is greatly different from the amount reaching the mitochondria
- The oxygen cascade describes the fall in oxygen as it passes from the air to the mitochondria
- Diffusion of gas is affected by molecular size, surface area and thickness of diffusion barrier and concentration gradient of the gas
- Oxygen content in the blood can be calculated by the flux equation
- CO₂ is mainly carried as bicarbonate in the blood
- Deoxygenated haemoglobin is more effective at carrying CO₂ than oxygenated haemoglobin

Clinical points

- CO, Hb saturation and haemoglobin concentration are the prime factors affecting oxygen delivery
- These factors can be manipulated by the anaesthetist to optimise a patient pre-operatively
- Insertion of haemodynamic monitoring, fluid therapy, transfusion, supplemental oxygen therapy and the correct use of inotropes are the key components in optimisation strategies to improve oxygen delivery

ANSWERS TO MCQS

1. FFTTF

Fowlers method uses expired nitrogen concentration to measure anatomical dead space. TLC, RV and FRC are measured using either helium dilution or body plethysmography techniques. FRC is about 2.2 litres in an average adult. Vital capacity is the combination of inspiratory reserve volume, tidal volume and expiratory volume. Helium dilution will underestimate the FRC in patients with bullous lung disease

2. FFTFT

The FRC decreases in the obese population and in the supine position. It is also affected by age and sex. It is the combination of the residual volume plus the expiratory reserve Volume.

3. FTTTT

Surfactant is an amphipathic molecule, with a charged hydrophilic head and hydrophobic tail. It is a mucopolysaccharide.

4. TFFTF

See explanation in answer 1 for stems a and b. The respiratory quotient is the amount of CO₂ produced/O₂ consumed and is measured by indirect calorimetry using a respirometer.

5. FTTTT

The oxyhaemoglobin dissociation curve is shifted to the left with a decrease in CO₂, 2,3 DPG, temperature and an alkalosis. Carbon monoxide prevents haemoglobin from releasing oxygen to the tissues therefore it causes a shift in the curve to the left.

WEB LINKS

www.frca.co.uk

REFERENCES

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