Additional information for trainees/trainers to underpin power point presentations

PACU Breathing Learning and Teaching Resource

Presentation : Basic sciences

Introduction : Breathing

The respiratory system consists of the airway, lungs and pulmonary circulation. Breathing enables gases to travel to and from the atmosphere via the airways. In the alveolar network, gases exchange into the capillary circulation for transport to the body tissue cells, and waste products back from the tissues for expiration by the lungs. Cells need oxygen and glucose to enable oxidative metabolism and create energy to fire all biochemical reactions essential for life. A waste product of metabolism, carbon dioxide, must be exhaled constantly to maintain the acid base balance in the body. Carbon dioxide retention causes blood pH to become more acid which damages the cells.

External and internal respiration

External respiration occurs in the lung, between the alveoli and pulmonary capillary circulation where gases exchange either way across the alveolar-capillary membrane. Internal respiration occurs in the body tissues where oxygen diffuses from blood stream to cell, and carbon dioxide from cell to the venous circulation.

External Respiration

We will examine the structure and function of the lungs to understand how breathing enables external respiration.

a. Overview of lungs anatomy and physiology
b. Respiratory Centre
c. Mechanics of breathing
d. Lung volumes
e. Transfer of gases at the alveolar-capillary membrane

a. Overview of the lungs - anatomy and physiology

The respiratory system is composed of the airways and lungs. We have examined the airway [upper and lower] as far as the alveolar network in BARNA Airway presentation. Please revise these structures. The airway must be patent to transport air from the atmosphere via the nose : mouth : pharynx : larynx : trachea : left and right main bronchus and smaller airways until the terminal airways, the alveoli where gas exchange occurs.

- The lungs are two cone shaped organs lying on either side of the heart in the mediastinum
- The proximity of lungs to the heart allows both organs to work simultaneously to deliver oxygen to the tissues and eliminate carbon dioxide : if the one is failing, the other compensates
- The left lung has three lobes, the right lung has two lobes. Fissures divide the lung into lobes. Each lobes receives it's won secondary [lobar] bronchus.
- The trachea feeds bifurcates at the carina from where the left and right main bronchus emerge. The left main bronchus is angled more sharply than the right main bronchus. In endotracheal intubation this can lead to the endotracheal tube slipping down the right main bronchus, resulting in oxygen delivery to one lung only. Post intubation auscultation of the lungs is essential to verify placement.
- The trachea and main bronchi contain cartilage which gives these airways a semi rigid structure. In the smaller bronchi and bronchioles this cartilage gives way to smooth muscle with no cartilage enervated by the autonomic nervous system. This allows the airways to dilate with sympathetic input and constrict with parasympathetic input. Bronchospasm occurs when the airways constrict – a post anaesthetic respiratory complication.
- The medium size bronchi offer resistance to air flow on expiration. This explains why asthmatics have more difficulty exhaling than inhaling. Resistance plays an important role in ventilation : if there is airway constriction it is more difficult to ventilate patients.

- Finally the smallest airways end at the terminal alveolar network which makes up the vast expanse of lung tissue [and sponge like quality]. Approximately 300 million alveoli – like clusters of grapes, provide surface area for gas exchange matching the area of half a tennis court.

- Each bunch of alveoli are supplied with blood from the pulmonary artery which transports deoxygenated blood from the right heart to the venous end of the pulmonary capillary. Here carbon dioxide diffuses from blood into the alveoli to be exhaled. At the arterial end of the pulmonary capillary oxygen diffuses across into the blood stream for mass transport to the body tissues.

- VQ ratio. This refers to the ventilation perfusion match in the lung. The close relationship between alveoli and the pulmonary artery capillary network throughout the lung should ensure that good ventilation matches excellent blood supply. When people move around normally there is constant distribution of blood flow and opening up of the air sacs. In perioperative practice where the patient lies static and supine on the table for longs lengths of time the air sacs at the lung apices of the lung are open while those of the bases often shut down. By contrast the blood flow distribution is healthiest at the bases [not the apices]. Thus there is an immediate mismatch between gas and blood supply. The factors that disrupt VQ ratio are numerous including patient positioning, immobility, damage to alveoli, impaired blood supply. This may produce over perfused, under ventilated alveoli at the base, and over ventilation and under perfusion in the lung apex which can contribute to hypoxia.

- Diffusion : this is a passive process and occurs because gas always travels across a membrane from an area of high to low pressure until both sides are equal. Carbon dioxide is at a higher pressure in the pulmonary capillary than the alveoli – $C0_2$ diffuses across. Oxygen is higher in the alveoli than the capillary – oxygen diffuses across – in both cases diffusion happens until the pressure gradients are equal.

- The alveolar-capillary membrane consists of two membranes only one cell thick lying adjacent to each other. This allows for RAPID diffusion of gases across. $C0_2$ travels much faster than oxygen.

- The alveoli are kept open partly by surfactant a polyprotein secreted by the walls of the sac – this reduces surface tension and allows the alveoli to keep the bubble shape. Prolonged exposure to positive pressure ventilation intraoperatively can break down surfactant leading to alveoli collapse and less surface area for gas transfer. This can be a problem in the PACU – where stir up regime is necessary to recruit the alveoli.

- The lungs are framed by the rib cage and are connected to them by the outer protective visceral layer of tissue [pleura] surrounding them. This visceral pleura is fixed to the undersurface of the ribs. When the ribs move upwards and outwards the lung expands with this action. The inner pleura which actually covers the surface of lung tissue is the parietal pleura. Between the two is the pleural cavity which secretes a film of fluid allowing surfaces to glide over each other. If the parietal pleura is punctured the lung collapses [pneumothorax]. This may happen in practice with central line placement, or a spontaneous ruptured bullae.

- Between the ribs lie swathes of intercostal muscles which shorten and tighten to allow rib expansion upwards and outwards.

- At the base of the lungs sits the diaphragm a large muscle shaped like a dome. When the rib cage expands upwards and out the diaphragm descends on inspiration leading to an increase in lung volume. The diaphragm action is responsible for 75% of air entering lungs.

- The property of elasticity is very important in allowing the lung to expand and contract. Elastic fibres are embedded in lung tissue. Lung compliance refers to the effort required to stretch the lungs and the chest wall. Low compliance means that the lungs are stiff and unable to expand easily making assisted ventilation difficult.

- Surface tension lowers the potential of alveolus to stretch and expand with fresh gas infusion and causing alveoli to collapse. Surfactant is a protein excreted by the alveoli wall and is a formidable force in maintaining open alveoli since it breaks down

surface tension. Surfactant is lost during artificial ventilation, and patients undergoing prolonged surgery may have significant alveolar collapse post operatively.

b. The Respiratory Centre and the control of breathing:

- The respiratory centre in the medulla oblongata is a specialized group of cells lying near to other vital centres in the brain stem [such as the cardiovascular centre]. It controls breathing; instigating breath and modifying the size of each breath.

- The respiratory centre receives messages from chemoreceptors found in the aortic arch and the carotid bodies. These special 'detector' cells sense the levels of oxygen and $C0_2$ in the blood stream and alert the respiratory centre that a breath is needed to eliminate $C0_2$ – and to inspire oxygen. $C0_2$ excess is the primary stimulus. Oxygen deficit is the secondary stimulus. Similarly acid-base balance is detected near to the RC – if blood is acid – this denotes there is an excess of $C0_2$ – this is another central respiratory stimulus.

- Clinical application : in peri-anaesthesia practice many drugs/agents depress the respiratory centre. This means that they render the centre less sensitive to high $C0_2$ levels. When this happens, rate and depth of respiration falls which can lead to hypoventilation with resultant hypoxia and a rise in $C0_2$. The residual effect of these drugs makes central respiratory depression a relatively common problem in the PACU.

c. The mechanics of breathing

- The respiratory centre alerts the intercostal muscles to expand the rib cage upwards and out – and the diaphragm downwards [by action of the phrenic nerve]. The lung volume enlarges and the pressure drops. At this point the pressure in the lungs is lower than that in the atmosphere / alveoli. Air containing oxygen rushes into the lungs. On expiration, the rib cage falls back to its original size passively, and the diaphragm ascends, thus contracting the lung volume. As the volume

contracts, the pressure goes up until the lung pressure is higher than atmospheric pressure. Air is exhaled from lungs to atmosphere

- **Remember this important fact : Boyles Law states that gases always travel from an area of high to lower pressure until equilibrium is reached**

- Clinical application : the strength and tone of the intercostal respiratory muscles is very important in maintaining strong respirations and efficient ventilation. In clinical practice the use of long lasting muscle relaxants can weaken muscle strength resulting in hypoventilation. Residual paralysis is a serious complication when the muscle relaxants have not been reversed and muscular activity is deranged leading to disco-ordinated breathing and hypoxia.

d. Lung volumes

- Adults at rest exchange around 500 mls of air on one breath known as the tidal volume. Around 12-15 bpm are taken. Multiplying 500mls X 12 bpm = around 6 litres of air exchanged over one minute = minute volume. We can measure breath volumes with incentive spirometry but in practice usually observe the rate and depth of breaths when doing respiratory observations.

- Not all the fresh air inspired is used in respiration. Around 150mls is stale air that is trapped in the airways and never gets shunted to the alveolar surface. This is known as **dead space.** The volume of air containing oxygen which reaches the alveoli for gas transfer is known as the **Alveolar Ventilation Rate.** This is an important measurement : effectively **500 – 150mls = 350mls per breath.** Multiply this by 12 bpm and result is 4,200mls of gas per minute used for alveolar ventilation. In order to maintain this volume for gas exchange the respiratory apparatus must be working well : respiratory central trigger to breathe, muscle power for lung excursion, alveoli free from fluid. Post operatively residual loss of muscle power and respiratory central depression all combine to cause hypoventilation. If alveolar ventilation falls below dead space volume – carbon dioxide retention occurs.

- The **Functional Residual Capacity** is another important lung volume. It is the pool of gas left in the lungs at the end of quiet expiration. This is in effect the reserve supply of oxygen in emergency situations. Prolonged assisted ventilation also reduces this reserve – so patients have less to fall back on in a respiratory emergency.

e. Transfer of gases at the alveolar-capillary membrane

- Gases diffuse across the alveolar-capillary membrane due to a pressure gradient between the gas in the alveoli and the pulmonary capillary. Gases always flow across a membrane from an area of high to low pressure until equilibrium is reached.

- We need to examine how gases behave in order to understand this transfer.

- Gas consists of millions of molecules [of whatever gas] jostling around for position, colluding, exerting pressure in a fixed space. The smaller the space, the higher the pressure.

- Atmospheric air consists of mixed gases in fixed percentages. Nitrogen [80%] Oxygen [21%] C02 [0.03%]. Dalton's Law states that each gas in a mixture of gases exerts its won pressure as if no other gas were present. That is why partial pressure refers to each individual gas.

- The total atmospheric pressure is 760mmHg at sea level. Oxygen at 21% therefore exerts a partial pressure of 21% of 760mmHg = 160mmHg. This is known as PO_2 [partial pressure of oxygen in the atmosphere]. Carbon dioxide at 0.03% of 760mmHg = This is known as $PC0_2$.

- By the time oxygen reaches the alveoli – it has been mixed with water vapour and has lost some of this pressure: it now stands at 100-105mmHg or 13.3kPa.

- Oxygen diffuses across the alveolar-capillary membrane across a pressure gradient from the alveolus 13.3kPa to the capillary [40mmHg or 5.3kPa. In other words from higher to lower pressure until equilibrium has been reached.

- Carbon dioxide has a partial pressure in the capillary of 45mmHg or 5.8kPa. It diffuses across the membrane into the alveoli where its partial pressure is 40mmHg or 5.3kPa.

- Any obstacle to the diffusion of gases can lead to hypoxia/hypercarbia. Loss of alveolar ventilation caused by respiratory depression, weak muscles, airway obstruction can be serious. Loss of surface area for gas exchange though the collapse of alveoli, or lung disorders such as emphysema decrease gas exchange rate. Build up of interstitial fluid between alveoli [pulmonary oedema] slows gas exchange.

Carriage of gases in the blood : oxygen

- The main purpose of circulation is to carry oxygen and nutrients to tissue cells by mass transport. If this fails tissues become hypoxic –anaerobic metabolism takes place and lactic acidosis develops. This disturbs the acid base balance.

- Oxygen is carried in the blood in simple solution [2% dissolved in plasma]. The remaining 98% of oxygen is carried in combination with haemoglobin [oxyhaemaglobin] in the red blood cell. This combination is a reversible chemical combination and describes oxygen content. Oxygen content is different from partial pressure, oxygen content only refers to the amount of oxygen carried, not its partial pressure.

- When all the molecules of oxygen are attached to the haemaglobin the blood is said to be fully saturated at 100%. Pulse oximetry measure this saturation level. When only half of oxygen molecules are attached, patient is only 50% saturated.

- The higher the partial pressure of oxygen $Pa0_2$ [measured in ABG's] the stronger will be the loading of the oxygen molecule onto the haemaglobin atom. Partial pressure of 0_2 is reliant on **2% of oxygen** which diffuses into the blood. However, the 2% is vital. If the patient become short of oxygen – and partial pressure falls, the oxygen molecules quickly dissociate from haemaglobin and oxygen saturation [$Sa0_2$] falls.

- The RBC is concave shaped 'S' shape. This is to ensure that oxygen adheres to Hb up to a point. The dissociation curve [see diagram on presentation] is S shaped. At the higher end $Sa0_2$ of 90% of oxygen adheres to Hb even at a $Pa0_2$ of 8.3kPa. Below this pressure oxygen rapidly dissociates from Hb. In clinical practice this means that below saturation of 90% fast deterioration occurs as the $Pa0_2$ falls below 8.3kP – unless fresh oxygen reaches the blood stream via unobstructed lungs, and efficient breathing the patient is heading for DANGEROUS HYPOXIA. Hypoventilation cause by central respiratory depression, residual muscle weakness is dangerous.

- While pulse oximetry allows a real time reading of $Sa0_2$, the partial pressure of oxygen [$Pa0_2$] must be measured with arterial blood gas analysis.

- 4 factors are involved in the supply of oxygen to the tissues.
  a. healthy lungs [consider how lungs are affected by general anaesthesia with potential for hypoventilation]
  b. cardiac output : determines speed at which oxygen reaches the tissues carried in the blood
  c. haemaglobin : lack of Hb in anaemic patients mean that there are limited Hb molecules for the oxygen to attach to : while $Sa0_2$ may read 100% oxygen content is less than nonanaemic patients
  d. metabolic factors : hypothermia and alkalaemia decrease amount of oxygen carried by Hb

Carriage of gases in the blood : Carbon Dioxide

- Approximately 200ml of $C0_2$ is produced by the tissues per minute as a by product of cellular metabolism. Carbon dioxide readily dissolves in the blood and combines with water to form carbonic acid [H_2C0_3].

Carbonic acid is weak, and quickly dissociates to form H+ ions and sodium bicarbonate [$HC0_3$]

$C0_2 + H_20 = H_2C0_3 \geq H+ + HC0_3$

Carbonic Acid — Hydrogen + Bicarbonate

- This equation can fall either way. It maintains an acid-base equilibrium in the blood of between 7.35 and 7.42. The hydrogen is the acid part which must be neutralized by bicarbonate [a buffer]. In the lungs the equation shifts to the left, carbon dioxide is breathed out as a gas.

- In clinical practice carbon dioxide levels in the blood are measured by capnography which gives the real time end tidal $C0_2$ [approximates to the $PaC0_2$ in the blood as gas equilibrates.]

- Carbon dioxide is 20 times more soluble than oxygen and diffuses very quickly from the tissue cells into the blood. Again diffusion of $C0_2$ in the lung is so efficient that diffusion defects that cause hypoxaemia do not cause hypercapnia. This is due to the vital importance of constant elimination of $C0_2$.

- NORMAL $PaC0_2$ = 4.5-6.0kPa – at end of normal expiration $C0_2$ in expired air is the same as $PaC0_2$

- Low $ETC0_2$ = low $PaC0_2$ = lower than 4.5kPa - indicates hyperventilation

- High $ETC0_2$ = high $PaC0_2$ – higher than 6.0kPa indicates patient retains $C0_2$ - hypoventilation

Hypoventilation while causing hypoxia – can lead to hypercapnia as $C0_2$ fails to be expired from the lungs, H+ ions accumulate and the body cells become acid and start to malfunction.

Internal Respiration

- At cellular level gases again diffuse across the capillary membrane [one cell thick] to the cell membrane [also one cell thick] from high to low pressure until equilibrium is reached. Oxygen in the arterial capillary end is 13.3kPa and in the tissues 5.3kPa. Molecules diffuse across until equilibrium is reached. $C0_2$ in tissue cells is 5.8kPa and 5.3kPa at the venous end of the capillary. Molecules diffuse from high to lower pressure from cells to circulation – and $C0_2$ is transported back to lungs for expiration.

barna

Trainer's notes on power point presentations

Breathing : Basic Sciences

TRAINER NOTES

BREATHING : Basic Sciences Presentation

Slides 3-5: Re-establish basic facts on metabolism [already discussed in airway PPP]. Understanding how and why oxygen + carbon dioxide are involved in metabolism is basic to rationalizing the need to prevent hypoxia.

6-8 : Important to explain why lack of oxygen causes eventual tissue cell death. The terms hypoxia and hypoxaemia are often misused – need to clarify from the start. A clinical focus – how we monitor oxygenation in practice [with oximetry and ABG's] – makes this science relevant.

9-12 : Establishing ventilation as indicating effective elimination of carbon dioxide is vital in peri-anaesthesia. Measurement of carbon dioxide with $ETC0_2$ again provides clinical focus. Good lead up to introduction of acid base.

13: Introducing acid base early here immediately explains why $C0_2$ elimination is vital for cell function and why hypoventilation in clinical practice can lead to build up of this gas and acid base inbalance. From now on we can talk about acidity, alkalinity with less confusion.

14: Breathing is second priority : keep establishing this prioritization with rationale.

15-18 : External/internal respiration – not difficult concepts / help divide up the long journey of oxygen to tissues!

19: Next section deals with anatomy and physiology of lungs in sections.

21-22: Close proximity of heart and lungs/make point as explains how they work in collaboration and if one fails the other compensates.

23-26 : Focus on rib cage, intercostal muscles, pleurae, diaphragm / how they are connected to enable inspiration. In particular emphasise the important role of the diaphragm in breathing.

27-29: Get students to examine which of airways have cartilage – which have smooth muscle and why. Refer to bronchodilaton and constriction and action of sympathetic nervous system.

30-31: Two slides alveolar network / gross facts about surface area for gas exchange / and pulmonary blood supply – both key factors in exchange of gases. Refer to fact that in prolonged procedures the match of blood to fresh gas is disturbed [VQ ratio] which may lead to hypoxia.

33-35: Role of respiratory centre : key topic to understanding rate/depth of respirations and how this can be affected by GA mix. Students should understand how the respiratory centre is stimulated **primarily by $C0_2$ excess**. Opioids render the Respiratory Centre less sensitive to $C0_2$ levels – hence breathing becomes slow and shallow accumulation of hydrogen ions and acidity. Carbon Dioxide must be constantly eliminated.

36-37 : Diagrams demonstrate normal respiratory stimulus and effect of anaesthetic mix on breathing. Emphasise the common causes of hypoventilation in the clinical reference.

41: Stress again that CO_2 excess is primary respiratory stimulant – body needs to breathe out carbon dioxide all the time to keep pH in balance.

42-43: Very important that students understand how the visceral pleura attaches to the ribs and the action of intercostal muscles in elevating rib cage. This is key to understanding muscle weakness caused by muscle relaxants in practice.

44-45: Combined action of intercostal muscles and diaphragm needed to expand the lungs. Diaphragmatic action vital in this process.

46-50 : Boyles Law is not difficult, take time to ensure students understand this. This is KEY to understanding the mechanics of breathing. No need to cite pressure differences in detail here : the essence is to understand that as lung volume increases, pressure falls lower than atmospheric pressure – air rushes in. On expiration the converse takes place. **GAS TRAVELS FROM AREA OF HIGHER TO AREA OF LOWER PRESSURE** – this is fundamental to respiratory physiology. We will keep coming back to this fact as oxygen and carbon dioxide diffuse across pressure gradients.

52-53: Lung volumes. Basic equation of RR X TV = MV is essential knowledge to understanding breathing. This equation should be used at all times so student know it by heart. Diagram of lung volumes can appear complicated until you work it out! Essentialy we are only interested in TV, and Expiratory Reserve Volume or FRC [functional residual capacity]. FRC is the amount of oxygen left in the lungs after normal expiration – and is the reserve that can be used in emergency. IPPV [Intermittent Positive Pressure Ventilation] in clinical practice lowers FRC.

54-56 : The concept of 'dead space' is key to understanding 'alveolar ventilation rate'. The fact that not all air is used in gas exchange is critical. This means that the effective, working volume of air used in gas exchange [alveolar ventilation rate] must be greater than 'dead space' or carbon dioxide will be retained. Alveolar ventilation is a KEY CONCEPT – anaesthetic agents can cause hypoventilation meaning that alveolar ventilation is impaired. Hypoxia and hypercarbia will follow.

58 -62: Exchange of gases : again the concepts are not complex [gases diffuse across the membrane from high to low pressure until equilibrium established]. More than one slide explains this.

63: Units of measurement : quick reminder of 2 systems of measurement : kPa and mmHg [refer to ABG results in both].

64-67: Slides that demonstrate via diagrams how important it is to understand the close proximity of capillary and alveoli basement membranes. Also need to understand how many alveoli there are and how much surface area for gas exchange they provide. Role of surfactant in maintaining alveoli open an important concept for peri-anaesthesia nursing as this is destroyed by ventilation.

68: Reminder of how surgery and anaeasthesia can disrupt normal exchange of gases, collapse of alveoli.

70 – 74: How gases behave / partial pressures. It is important to understand Daltons Law and how oxygen and $C0_2$ percentage of atmospheric pressure results in 100mmHg and 40mmHg. Again not a difficult concept but needs time. Students should learn a little about how gases behave – since this makes the concept of 'pressure' much easier and later on $Pa0_2$ and $PaC0_2$ will be understandable.

76: The importance of circulation in transporting gases must be remembered. Explanation of lactic acidosis useful here, students often don't know about this.

77 – 81 : Carriage of oxygen in the blood. While this takes some understanding – it can be made simple. 2% of oxygen dissolves and creates $Pa0_2$ [partial pressure in the plasma]. 98% of oxygen travels on the back of Hb molecule. It is the partial pressure [2%] that **pushes oxygen onto the Hb.** If $Pa0_2$ falls, oxygen dissociates from Hb with a resultant fall in $Sa0_2$. Hypoxaemia means the $Pa0_2$ has fallen [arterial blood gas analysis would reveal this]. If fresh oxygen is not immediately taken in by the lungs, then dissociation will happen and $Sa0_2$ will fall. Hypoxia will result fast. The oxyhaemaglobin dissociation curve means that above 8.9 kPa – oxygen sticks fast to Hb. Below 8.9kPa – oxygen dissociates fast [i.e. below 90%]. Take time taken to explain the relationship between AGB analysis and Sa02 – and why in clinical practice Sa02 **FALLS RAPIDLY WHEN IT HITS 90** is important.

84-85: Recap of how oxygen saturation is measured by pulse oximetry and oxygen pressure by arterial blood gases in practice.

86: Carriage of $C0_2$ in the blood. Students should understand the basic equation given here – that $C0_2$ + water [end products of metabolism] create carbonic acid – a weak acid which then dissociates to bicarbonate [alkaline] and Hydrogen atoms. The equation can work either way – acid base balance is maintained [at respiratory level] via this mechanism.

87: High $ETC0_2$ is equal to the partial pressure of carbon dioxide in the blood. High $ETC0_2$ = hypoventilation as patient retains $C0_2$. Low $ETC0_2$ = hyperventilation as patient breaths off $C0_2$. The respiratory component of pH maintenance is important. Student should know that $C0_2$ easily dissolves – and easily transfers [much more than oxygen] – because pH MUST BE MAINTAINED WITHIN SAFE LIMIT.

88-92 : Concepts of gas diffusion now very familiar. Internal Respiration should be straightforward.

93: It is useful to summarise all the factors needed in transporting oxygen to the cells. It will help students consider whether hypoxia is due to airway, breathing or circulatory problem in practice.

94: A diagram to pull together all the possible respiratory complications in PACU – to prepare students for next presentation [all about practice]