Acute respiratort failure:

Definitions and basic physiology

- Respiratory system is no longer able to meet the metabolic demands of the body

- 2 major functions of the lungs:
  1. oxygenation of blood
  2. elimination of CO_2

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Acute respiratory failure

Definitions and basic physiology

Oxygenation + CO_2 elimination

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Derived from this there are 2 types of respiratory failure

1. ***hypoxemic*** respiratory failure (***type 1*** resp failure)

   PaO2 < kPa = 60 mmHg on room air (21% O2)

2. ***hypercapnic*** respiratory failure (***type 2*** resp failure)

   PaCO2 > 6,7 kPa = 50 mmHg

Acute respiratory failure

Definitions and basic physiology

- may be *acute* or *chronic*, depending on duration
- May be *acute on chronic* (superimposition)
- May be *combined* and may change during the clinical course

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Physiology

Helps to understand and categorize the causes and diseases in a logical way, including identification of the right management

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1. O2 from atmosphere > lung > blood

   ventilation *diffusion*

2. CO2 from blood > lung > atmosphere

   diffusion ventilation

Physiology

Gas exchange requires:

- a pressure gradient between alveolar air and blood
- a short distance for diffusion of gases and intervening tissues which are permeable to oxygen and CO2.

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Physiology – Getting oxygen in

The alveolar partial pressure of oxygen (P_AO_2) is dependent on the total alveolar pressure and the partial pressures of the other gases in the alveolus

the sum of the partial pressures of all gases is equal to the total alveolar pressure

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Physiology – Getting oxygen in

Alv.Pressure = PaO_2 + $PaCO_2$ + PaH_2O + PaN_2

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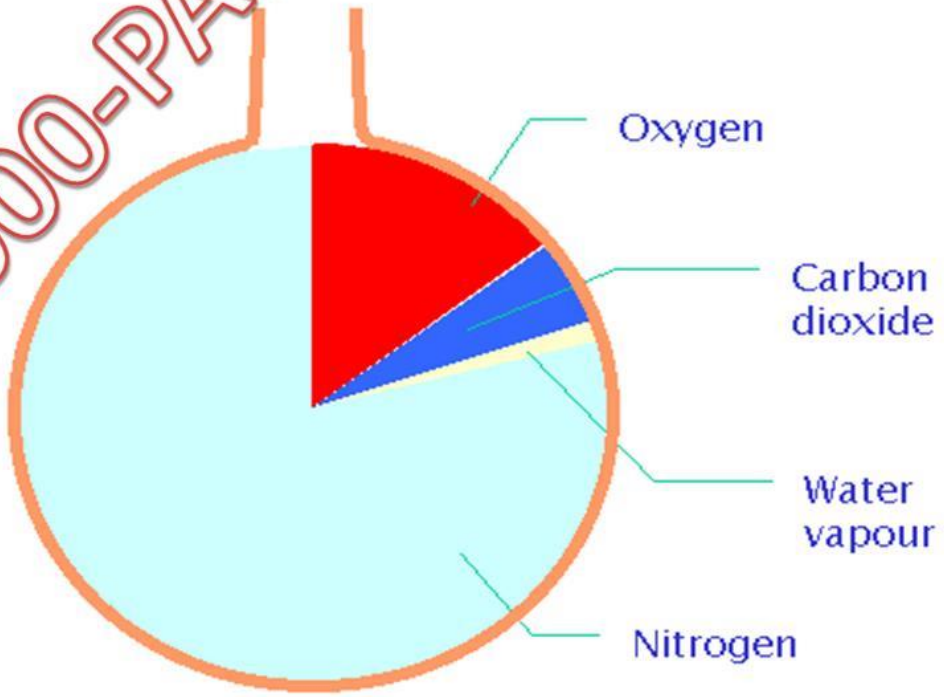

Alveolar pressure = $P_AO_2 + P_ACO_2 + P_AH_2O + P_AN_2$

Physiology – Getting oxygen in

- Partial pressure of each gas in a mixture of gases is directly related to the proportions in which they are present
- Therfore partial pressure of oxygen can be increased by:
  1. Increasing the proportion of oxygen in the mixture
  2. Increasing alveolar pressure
- Increasing FiO_2 increases the proportion of oxygen in alveolar gas while reducing the proportion of nitrogen
- Alveolar partial pressure of water vapour remains largely constant and therefore does not contribute to changes in PAO_2. The proportion of carbon dioxide in alveolar gas does, however, change and therefore factors which affect $PACO_2$ also affect PAO_2

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Physiology – getting oxygen in

- as CO_2 passes into the alveolus and O_2 passes into the blood the P_ACO_2 rises and the P_AO_2 falls. Ventilation is required to resplenish the alveolar gas with fresh gas
- Thus the factors that result in changes in P_AO_2 are:
- > P_ACO_2
- > alveolar pressure
- > inspired oxygen concentration
- > ventilation

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Physiology - Getting CO_2 out

- CO_2 elimination is largely dependent on alveolar ventilation (CO_2 crosses the alveolar membrane very readily and so diffusion abnormalities and shunting have little effect on CO_2 elimination)
- Alveolar Ventilation = RRate x (TV – Dead Space)
- RR = respiratory rate; TV = tidal volume
- Anatomical deadspace is constant, but physiological deadspace depends on the relationship between ventilation and perfusion
-

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Physiology – getting CO_2 out

- Therefore changes in P_ACO_2 are dependent on:
- > respiratory rate
- > tidal volume
- > ventilation - perfusion matching

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Pathophysiological mechanisms

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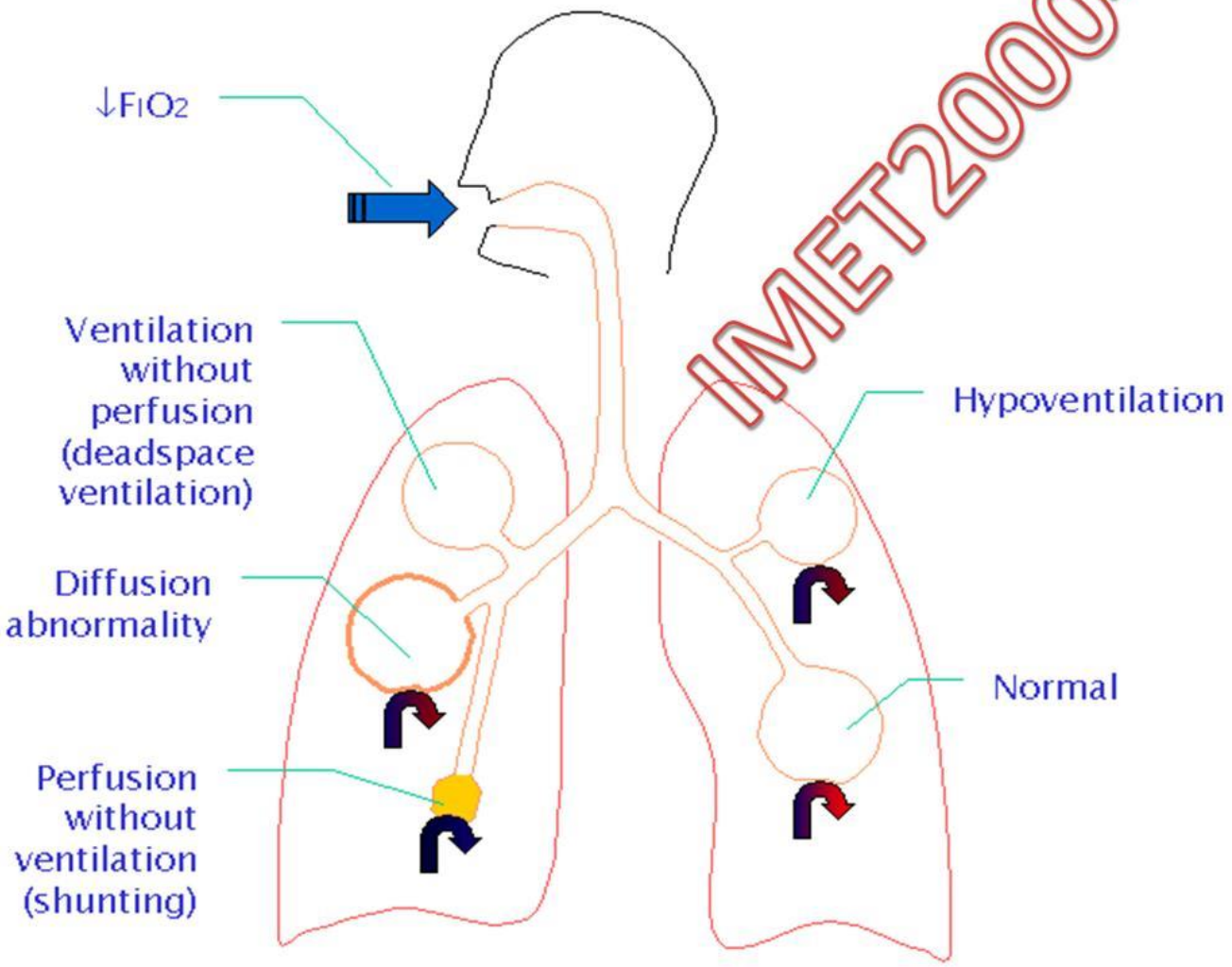

Pathophysiological mechanisms

- ***Shunting:***
- The most common cause for hypoxaemic respiratory failure in ICU patients is perfusion of non-ventilated alveoli
- It is a form of ventilation - perfusion mismatch in which alveoli which are not ventilated (due to collapse or pus or oedema fluid) but are still perfused. As a result blood traversing these alveoli is not oxygenated

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Pathophysiology and mechanisms

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Figure 2. Schematic representation showing shunting results in a fall in oxygen saturation. The alveolus on the right is filled with pus and is not ventilated. As a result the capillary blood is not oxygenated. This deoxygenated blood mixes with blood which has perfused normally ventilated alveoli and is 100% saturated, reducing the saturation in the pulmonary vein. With time hypoxic pulmonary vasoconstriction reduces the proportion of blood perfusing non-ventilated lung units thus minimizing shunting. Increasing FiO_2 has relatively little effect because blood leaving well ventilated units is already 100% saturated and the oxygen does not reach the blood perfusing unventilated lung units

Pathophysiology and mechanisms

- Shunting is relatively resistant to oxygen therapy. Increasing FiO_2 has little effect because it can not reach alveoli where shunting is occuring and blood leaving normal alveoli is already 100% saturated
- Shunting is the commenest cause of hypoxaemic respiratory failure in critically ill patients
- Hypoxic pulmonary vasoconstriction reduces the blood flow to non-ventilated alveoli and reduces the severety of hypoxaemia

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Pathophysiology and mechanisms

- Causes of shunting:
- > intracardiac
- any cause of right to left shunt eg. Fallot`s tetralogy, Eisenmenger`s syndrome
- > pulmonary
- pneumonia

pulmonary oedema
atelectasis
collapse
pulmonary haemorrhage
pulmonary contusion

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Pathophysiology and mechanisms

- ***Ventilation without perfusion***
- this is the opposite extreme of ventilation – perfusion mismatch
- gas passes in and out the alveoli but no gas exchange occurs because the alveoli are not perfused. The alveoli become a part of `***dead space***` (physiological dead space)
- unless the patient is able to compensate for it the reduction in effective ventilation results in an increase in $PaCO_2$

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Pathophysiology and mechanisms

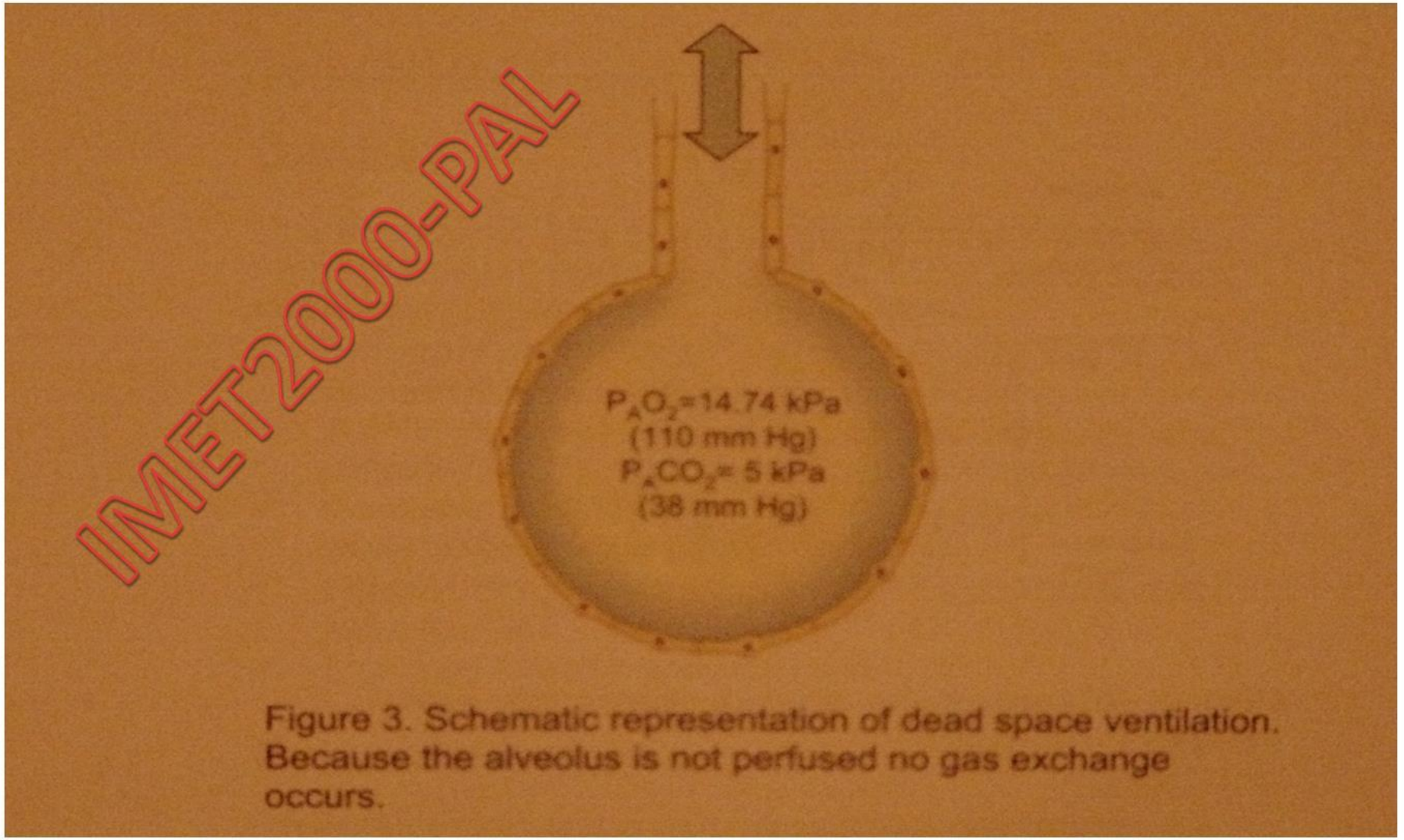

Figure 3. Schematic representation of dead space ventilation. Because the alveolus is not perfused no gas exchange occurs.

Pathophysiology and mechanisms

Causes include:

> low cardiac output

> high intra-alveolar pressure leading to compression or stretching of alveolar capillary (mechanically ventilated patients

> pulmonary embolism

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Pathophysiology and mechanisms

- ***Diffusion abnormality***
- less common
- may be due to an abnormality of the alveolar membrane or a reduction in the number of alveoli resulting in a reduction in alveolar surface area
- Causes include:
- Acute Respiratory Distress Syndrome (ARDS)
- Fibrotic lung disease

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Pathophysiology and mechanisms

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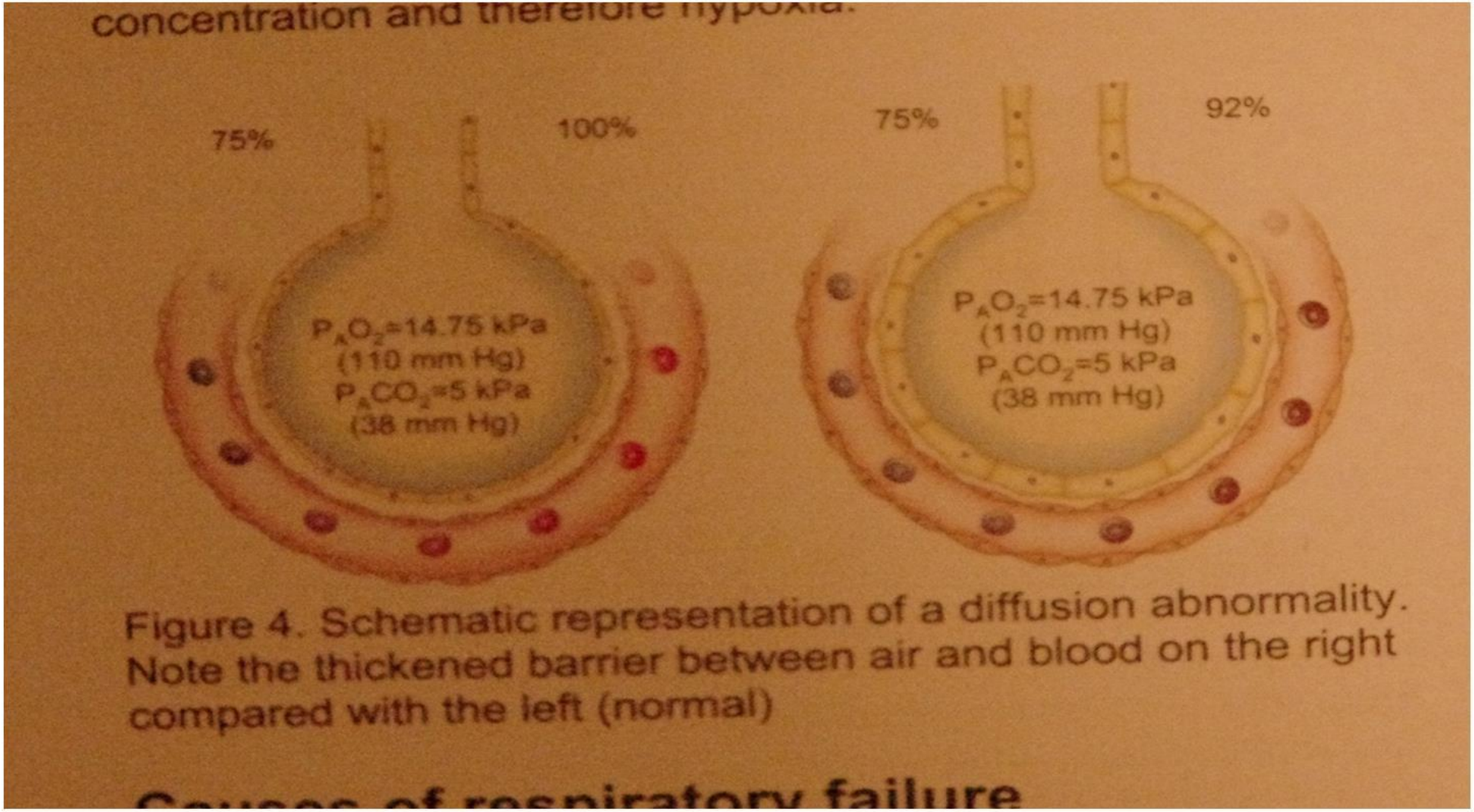

Figure 4. Schematic representation of a diffusion abnormality. Note the thickened barrier between air and blood on the right compared with the left (normal)

Causes of respiratory failure

Pathophysiology and mechanisms

- ***Alveolar hypoventilation***
- as CO_2 passes into the alveolus and O_2 passes into the blood the pressure gradients between alveolar gas and blood are gradually reduced. Ventilation is required to restore the pressure gradients
- hypoventilation is marked by a rise in PaO_2 and a fåll in PaO_2

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Pathophysiology and mechanisms

- ***Causes of hypoventilation:***
- *Brainstem*:
  injury due to trauma, haemorrhage, infarction, hypoxia
  infection, etc
- *Spinal cord:*
  trauma, tumor, transverse myelitis
- *Nerve root injury*
- *Nerve:*
  trauma
  neuropathy eg Guillain Barre
  motor neuron disease

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Pathophysiology and mechanisms

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Pathophysiology and mechanisms

Causes of hypoventilation (cont.)

Respiratory muscles:

- fatigue
- disuse atrophy
- myopathy
- malnutrition

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Respiratoty system:

- airway obstruction (upper or lower)
- decreased lung, pleural or chest wall compliance

Pathophysiology and mechanisms

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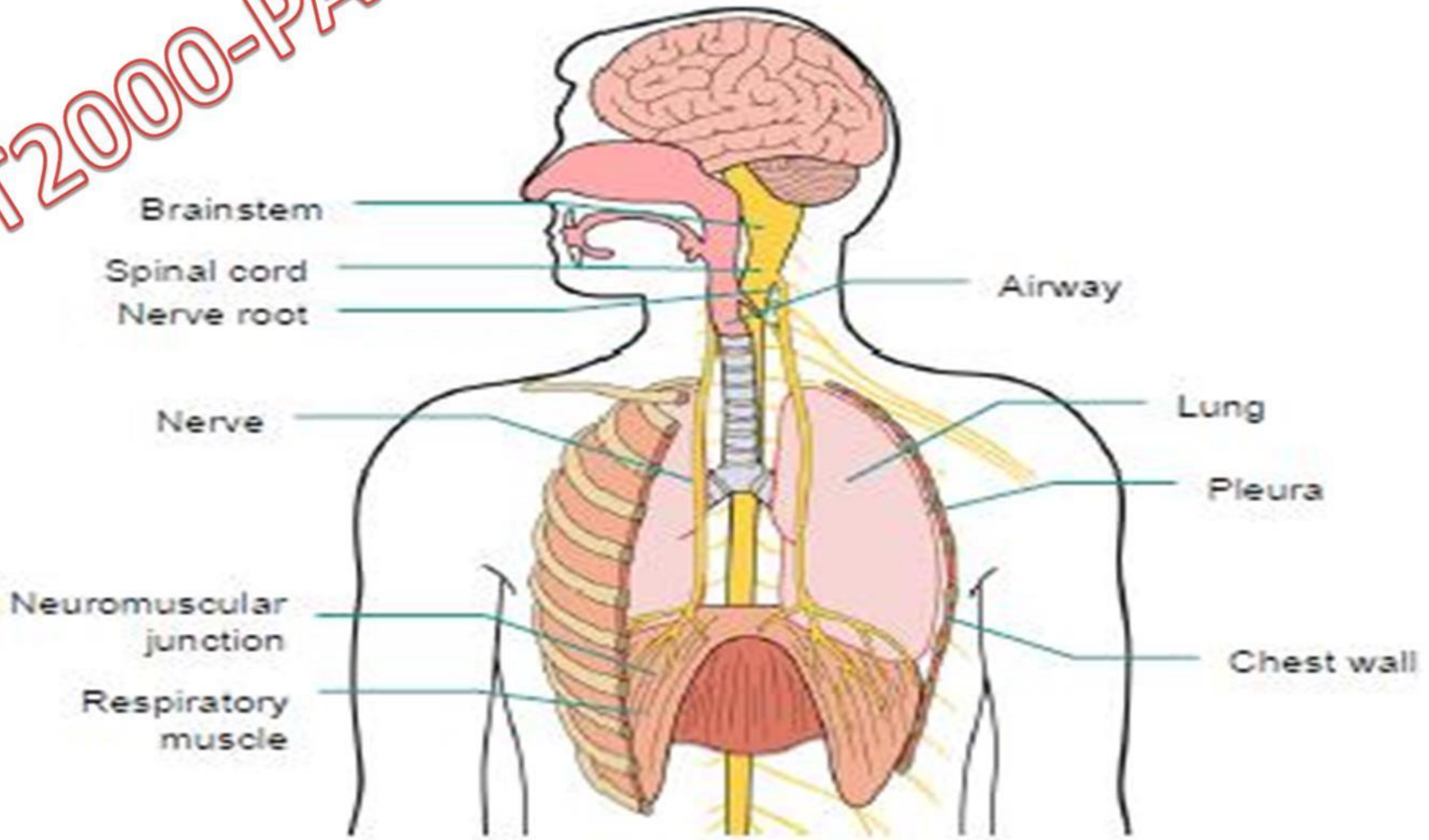

Respiratory monitoring - basic

Clinical: The signs of respiratory failure are signs of respiratory compensation, increased sympathetic tone, end-organ hypoxia, haemoglobin desaturation

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- Signs of ***respiratory compensation***:
  >tachypnoea > is a very good indicator of severe illness always monitor respiratory rate >use of accessory muscles >nasal flaring
  >interxcostal, suprasternal, supraclavicular recessions

Respiratory monitoring - basic

- Increased ***sympathetic tone***
  >tachycardia
  >hypertension
  >sweating
- End-organ ***hypoxia***
  >altered mental status
  >bradycardia and hypotension (late signs)
- Haemoglobin ***desaturation***
  >cyanosis

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Respiratory monitoring - basic

- ***Pulse oxymetry***
- estimates arterial haemoglobin saturation, not PaO_2, using absorption of 2 different wavelengths of infraread light
- the oxyhaemoglobin dissociation curve describes the relation between saturation and PaO_2
- *sources of error:*
  poor peripheral perfusion
  dark skin (pulsoximeter overreads slightly)
  fals nails or nail varnish
  excessive motion
  carboxyhaemoglobin ($SpO_2 > SaO_2$)
  poorly adherant probe

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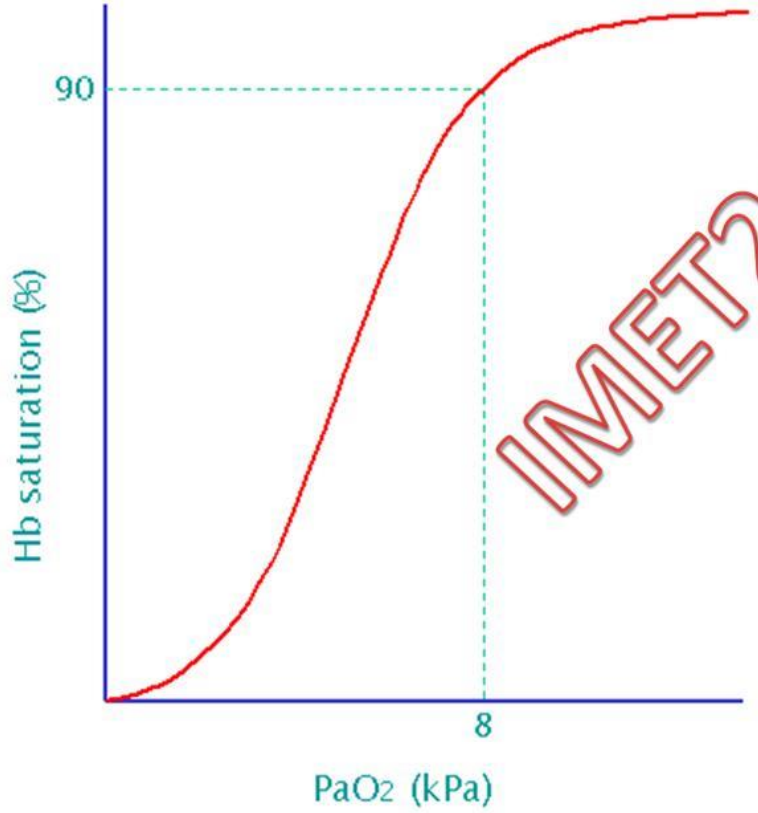
Respiratory monitoring - basic

- A pulse oxymetry saturation (SpO2) – 90% is a critical threashhold. Below this a small fall in PaO2 produces a sharp fall in SpO2



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Respiratory monitoring - basic

- Capnography
- Arterial blood gases

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