PHTY300:CARDIORESPIRATORY SCIENCE and PRACTICE OVERVIEW

Important Terms and Concepts		
Term	**Abb**	**Formula/Comments**
Minute Ventilation	VE	- Total volume of air moved in/out of the lungs in a minute - VE = Vt x RR (L/min)
Dead Space	Vd	- Non gas exchange areas i.e. anatomic dead space (conducting airways – trachea, bronchi) and physiologic dead space (alveoli which are ventilated but not perfused)
Dead Space Ventilation	VD	- The Dead Space volume over a minute - VD = Vd x RR
Alveolar Ventilation	VA	- The amount of gas which reaches the alveoli (for gas exchange) per minute - VA = (vt – Vd) x RR OR VE-VD
Peak Inspiratory Pressure	PIP	- Maximum pressure reached with a set Vt - Varies with airway resistance and respiratory compliance
Plateau Pressure		- End pressure after a period of no gas flow (inspiratory pause) - Plateau pressure is a function of lung and chest wall compliance
End Expiratory Pressure		- Airway pressure at the termination of the expiratory phase - Equal to atmospheric pressure or the applied PEEP level
Continuous positive Airway Pressure	CPAP	- Positive pressure during inspiration AND expiration. - Used for spontaneous breaths in non-intubated patient
Positive End Expiratory Pressure	PEEP	- Positive pressure that remains in the lungs at the end of the respiratory cycle (end of expiration) - Mechanically ventilated patients
Ventilator Induced Injury	VILI	- Further lung injury caused by use of 'conventional settings' during ventilation (barotrauma, volutrauma, biotrauma, atelectrauma etc.). Can also result in pneumothorax, pneumomediastinum and subcutaneous emphysema +/- high FiO2 causing more damage (inflammation etc.)
Barotrauma		- Pressure induced lung damage (in stiff areas)
Volutrauma		- Alveolar overdistension (in high compliane regions)
Biotrauma		- Ventilator induced inflammation
Atelectrauma		- Repeated alveolar recruitment and collapse

Normal ABG ranges

pH	7.35-7.42	(7.38-7.42)
PaCO2	34-45 mmHg	(38 – 42)
PaO2	80-100 mmHg	(85-100)
HCO3-	22-26 mmol/L	
BE	-2 to 2	
SaO2	95-100%	(96-98%)

PaO2 adequacy = O2 % x 5 = FiO2 % x 5
Acidaemia: Low pH (<7.35) **(**Conditioned marked by high concentration of hydrogen ions in the blood)
Alkalaemia: High pH (>7.45)

	ABG	**Causes**	**Signs and symptoms**
Respiratory Acidosis	Low pH (<7.35) High PaCO2 (>45)	- Decreased gas movement overall - Low Vt without increased RR - Low RR without increased Vt	- Drowsiness, confusion - Headache - Unsteady/falls - Increased ICP
Respiratory Alkalosis	High pH (<7.45) Low PaCO2 (<35 mmHg)	- Hyperventilation, blowing off too much CO2 - Anxiety, pain, acute (severe) hypoxaemia - Fever, sepsis	- Dizziness/fainting (from cerebral vasoconstriction) - Tingling lips and fingers, cramps - Confusion - Increased RR (tachypnoea) and/or volume (hyperpnoea)
Metabolic Acidosis	Low pH (<7.35) Low HCO3- (<22 mmHg) Low BE (<-2)	- From acid gain, failure to remove acids or bicarbonate loss - Diabetic ketoacidosis - Methanol (alcohol) poisoning - Lactic acidosis (decreased O2 to tissues) - Severe hydration, starvation or diarrhoea - Renal failure	- Confusion, drowsiness - Headache - Hyperventilation/hyperpnoea (long, deep breaths, normal rate) - If severe → cardiac arrhythmia, tachycardia, hypotension

Metabolic Alkalosis	High pH (>7.45) High HCO3- (>26 mmHg) High BE (>+2)	- From acid loss, alkali gain or renal bicarbonate retention - Acid: gastro (prolonged vomiting or nasogastric suction), urinary (excess diuretics – increased urinary acid secretion) - Alkali gain (ingestion of excess antacids in the presence of kidney failure) - Bicarbonate retention (reduced blood volume, metabolic syndromes)	- Headaches, lethargy - Muscle cramps, weakness - Decreased minute ventilation (decreased RR +/- Vt) - Arrhythmias

Factors that Affect PaO2 in a person

- O2 concentration: increased FiO2 = increased PaO2
- Barometric pressure: decreased PB (increased altitude) = decreased PiO2, decreased PaO2
- Age: increased age = decreased PaO2
- PaCO2: increased PaCO2 = decreased PaO2
- Lung pathologies: decreased SA for gas exchange = decreased PaO2

Oxygen Therapy is used for: hypoxaemia and some cardiac conditions

SpO2 targets

- 94-98% for acutely ill
- 88-92% (or patient specific) for those at risk of hypercapnic respiratory failure

Oxygen Transport System

- Oxygen perfusion follows a pressure gradient defined by the decreasing partial pressure of oxygen.
  - Air > upper airway > alveoli > interstitium > blood > tissues/cells
- In the air PO2 is affected by FiO2, barometric pressure and amount of fresh gas inspired. Generally PO2 = 159 mmHg
- In the upper airway air is humidified and warmed, decreasing PO2 to 149 mmHg.
- In the Alveolar dead space, gas mixing occurs, decreasing PaO2 to 100 mmHg.
  - In the alveoli PaO2 is affected by surface area for gas exchange, in the interstitium by pulmonary oedema.
- The PaO2 of oxygen is the driving force for perfusion into the blood, where PaO2 is 80-100 mmHg. The 5-25 mmHg difference between alveoli and blood is the A-A gradient. PaO2 in the blood is also affected by decreased lung perfusion, Hb and cardiac output.
- Perfusion into body tissues can also be affected by decreased O2 extraction/utilisation i.e. sepsis.

This system is used in oxygen therapy: increasing FiO2 by using oxygen therapy, increases the PO2 in air being delivered. By doing so, increasing the downstream oxygen perfusion.

Low Flow	Deliver 1-8 L/min, less than patients own inspiratory rate (normal adult PIFR = 15 L/min) O2 is highly diluted in room air, leading to low concentrations delivered to lungs
High Flow	Deliver high flows of >60 L/min, higher than patients own inspiratory rate Control of RA entrained during inspiration leads to more accurate FiO2 and more O2 delivered to lungs *When SOB, can match their high PIFR to deliver accurate O2*

Variable Performance O2				
	Description	**Advantages**	**Disadvantages**	**Delivery**
Nasal Prongs	- Low flow rate (1-4L/min) - Open system, O2 is diluted by RA through normal inhalation	- Comfortable, non-invasive - Can eat, drink, communication - Suitable for long term - Natural humidification can occur via nose	- Only for low flows (<5L/min) - Dry nasal mucosa and lead to nose bleeds - Inaccurate FiO2 (estimated)	1L/min → 0.24 FiO2 2L/min → 0.28 3L/min → 0.32 4L/min → 0.36
Simple Face Mask	- Short term use, mask acts as a reservoir.	- Suited for mouth breathers - Mask acts as a reservoir	- Inaccurate FiO2 (estimated) - Must maintain minimal 5L/min flow rate - Can't eat, drink - No humidifcation - Pressure areas	6L/min → 0.4 FiO2 8L/min → 0.5 10L/min → 0.60
Reservoir mask	-	- Delivers high concentrations O2, entrains small RA amounts - Reservoir stores O2, allowing consistent high O2 concentrations, even with high RR - Exact FiO2 amount known	- Very drying to mucosa - Can't eat or drink	10-15L/min → FiO2 0.6-0.9
Non-Rebreather mask	-	- Higher O2 concentration with reservoir bag - Minimises rebreathing	- Non humidified - Cant eat, drink - Pressure areas	10-15L/min → 0.6-0.9 FiO2

Fixed Performance				
Venturi Mask	- Use Bernouli principle to entrain room air when 100% O2 delivered	- FiO2 can be fixed by either altering size of gas orifice where O2 enters or size of entrainment port where RA enters - Devices allows controllable O2	- Can't eat or drink - Not humidified - Pressure areas	Up to 15L/min → 0.24-0.5 FiO2

Dangers and complications of O2 therapy	
Hypoxaemic Drive to Breathe (e.g. COPD)	- Normal drive to breathe is related to PaCO2 levels driven by central chemoreceptors (c PaCO2 ⇒ ↑ ventilation) - ↓ sensitivity with COPD, relying on low O2 levels (driven by peripheral receptors) instead - So, with high FiO2 oxygen therapy, drive to breathe can be 'turned off'
Oxygen Toxicity	- Occurs with high concentration O2 over prolonged periods - Adults result in: acute tracheobronchitis, diffuse alveolar damage, reduced cilial activity - Neonates results in: bronchopulmonary dysplasia, retrolental dysplasia (blindness)
Absorption Atelectasis	- N2 normally helps splint open alveoli, during 100% O2 therapy N2 is washed out of alveoli and replaced by O2 - O2 diffuses into pulmonary vasculature, causing alveolar collapse
Fire	- O2 is highly combustible

Keep FiO2 <0.6, and use length of exposure decreased to decrease complications

Humidification Importance			
General	**Indications**	**Benefits**	**Disadvantages**
- Cilia heat and humidify air being breathed in. Humidification counteracts cold, dry O2 therapy air or when URT is artificially bypassed - Heating a gas ↑ capacity to hold water	- O2 >4L/min - Use artificial airway/bypass URT for ventilation - ↓ airway resistance in asthma/croup - Presence of thick, tenacious sputum	- ↓ irritation nasal/oropharyngeal surfaces - Maintain airway hydration - Prevent crusting around & blockage artificial airways - Facilitate removal of secretions	- Over humdification/ saturation and overheating of airways - Colonisation of bacteria - Some patients find uncomfortable - cost
Heated Water Bath Humidifier	- active humidifier, 'wet' circuit - electrically powered heated water bath - capable of fully saturation and heating inspired gas to 37 at high flow rates (up to 60L/min)		
Heat and Moisture Exchanger (HME)	- passive humidifier, 'dry' circuit - contains layer of foam/paper with salt (e.g. CaCl2), traps heat and moisture from inspired breath which is then inspired - used in short term mechanical ventilation (<24hr)		
Combined O2 Therapy and Humidifcation AIRVO	- High flow oxygen therapy 15-60L/min (FiO2 0.21-0.6) with built in humidification - Delivers high O2 concentrations - Gives 3Cm H2O PEEP - Improves airway clearance (humidification), while well tolerated and comfortable		

Nebulisation

- Used to deliver saline (normal or hypertonic) and medication (e.g. salbutamol/Ventolin) by converting liquid to a fine mist for inhalation
- Facilitates airway clearance (saline)
    - Normal saline (0.9%) → adjunct to other therapies
    - Hypertonic saline (3-7%) → patients with viscous secretions (CF). Osmotic agent, drawing water into mucus, decreasing its viscosity making it easier to clear. Must be medically prescribed.

Respiratory Failure: a syndrome in which the respiratory system fails in one or both of its exchange functions (oxygenation and carbon dioxide elimination). i.e. inability to ventilate adequately or provide sufficient O2 to the blood and system organs
Acute Respiratory Failure: rapid onset (minutes to hours), short cause
Chronic Respiratory Failure: long term duration of poor ABG values, can be life threatening. Will be (metabolic) compensation, therefore pH can be normal.
Acute on Chronic Respiratory Failure: e.g. acute exacerbation of COPD

	Hypoxaemic Respiratory Failure	Hypercapnic Respiratory Failure
Description	- *Lung failure* - O2 gas movement issue - reduced regional ventilation - lung disease severe enough to interfere O2 exchange	- *Pump failure* - primarily CO2 movement issue - ↓ alveolar ventilation - inadequate respiratory pump (cannot maintain ventilation to eliminate CO2 produced by metabolism
Signs and Symptoms	- Dyspnoea - Changes in POB (e.g. ↑ RR)	- Depends on rate of CO2 rise and extent of metabolic compensation

	- Agitation followed by somnolence (drowsiness) - ↓ mental acuity (PaO2 <40-50mmHg) - Organ failure (e.g. renal injury, brain injury)	- Dyspnoea - ↑ respiratory rate, change in POB (COPD: accessory muscle use, paradoxical breathing, intercostal space or rib indrawing, PLB) - Agitation, tremor - Confusion → coma - ↑ ICP, headache
	PaO2 <60mmHg PaCO2 normal or low Type I Respiratory Failure	PaO2 Lower than normal PaCO2 high; <45; Type II Respiratory Failure if PaCO2 >50mmHg
Mechanisms and Causes	Reduced surface area (SA) for gas exchange - Consolidation/pneumonia, collapse, emphysema Inadequate fresh air reaching gas exchange area (airway dysfunction) - Acute asthma, airway blockage, hypoventilation Diffusion Problems (Interstitial or vascular dysfunction) - Lung fibrosis, pulmonary oedema Perfusion problems Interstitial or vascular dysfunction) - Pulmonary embolism	- Depressed drive to breathe - Impaired neuromuscular function (e.g. Cervical Spinal cord injury, Guillain Barre syndrome, Respiratory muscle function) - Increased respiratory load (issue with compliance or resistance, increased resistance e.g. asthma/COPD, decreased lung compliance e.g. lung collapse/consolidation
Physiotherapy Implications	- Watch patient signs and symptoms of respiratory failure - Determine type of respiratory failure → Hypoxaemic vs. Hypercapnic - Determine cause of respiratory failure → impairment problem list (↓ O2 +/- CO2 gas movement) - Review medical Ax and management - Choose appropriate interventions - Medical Management: ventilatory support +/- intubation	
Acute RF Treatment	Hypoxaemia Type I RF - Oxygen therapy and CPAP (first call) - High flow Nasal Prongs - Bilevel Ventilation - Intubation and MV (*last call*)	Respiratory Acidosis and Hypercapnia (Type II RF) - Ventilatory support - Invasive ventilation - Non-invasive ventilation → bilevel ventilation
Severe Hypoxaemia Treatment		
Oxygen Therapy	- Low or high flow devices - ↑ FiO2 → ↑ PO2 lower respiratory tract → ↑ PaO2 → ↑ driving pressure for O2 to diffuse to capillaries → ↑ PaO2 **↑ FiO2 → ↑ diffusion of O2**	
CPAP and PEEP	- CPAP: intubated or non-intubated patient, positive pressure during inspiration and expiration in an non intubated patient (some intubated patients) - PEEP: only used on a mechanical ventilator. Positive pressure remains in lungs at end of respiratory cycle. Physiologically effective by ↑ FRC: - ↑ size/volume of already open alveoli: +ve pressure at end expiration → ↓ alveolar emptying → ↑ alveolar size → ↑ FRC → ↑ gas exchange surface area → ↑ PaO2 - Alveoli recruitment via collateral channel ventilation (open previously closed alveoli): +ve pressure at end expiration → Re-expands collapsed alveoli → ↑ number of alveoli participating in gas exchange → ↑ FRC → ↑ gas exchange surface area → ↑ PaO2 **↑ surface rea for gas exchange**	
Hyperbaric Oxygen Therapy Chamber (Alt)	- Administration 100% O2 in pressurised environment (up to 3xx atmospheric pressure) Used for - Decompression illness ('the bends' - Non healing wounds, ulcers and serous soft-tissue infections	
Nitric Oxide (NO) (Alt)	- Inhaled NO is a selective pulmonary vasodilator Positives - NO goes to areas of lung being ventilated, resulting in: improved blood flow to open alveoli (↑ PaO2), ↓ pulmonary artery pressure, ↓ pulmonary vascular resistance **↑ perfusion (blood supply) to those alveoli that are ventilated (with O2**)	
Extracorporeal Membrane Oxygenation (ECMO) (Alt)	- Cardiopulmonary support provided outside the body using artificial heart and/or lung support - The extracorporeal circuit allows for the oxygenation and removal of CO2 from blood - Used as supportive strategy in patients who have a high risk of death despite conventional therapy (e.g. awaiting heart transplant) Challenging for Physio to mobilise out of bed to prevent ICU complications **Adds O2 to blood through a machine outside the lungs**	
Physiotherapy Implications	Physio limited in severe respiratory failure and medically unstable patients, especially in requiring high levels of PEEP/CPAP	