AIRWAY MANAGEMENT

Describe the anatomy of the upper airway, larynx and trachea, including its innervation and endoscopic appearance

Upper airway:

- airway from nares + lips to larynx includes: nose, oral cavity, pharynx, larynx
- Pharynx divided into: nasopharynx, oropharynx, laryngopharynx

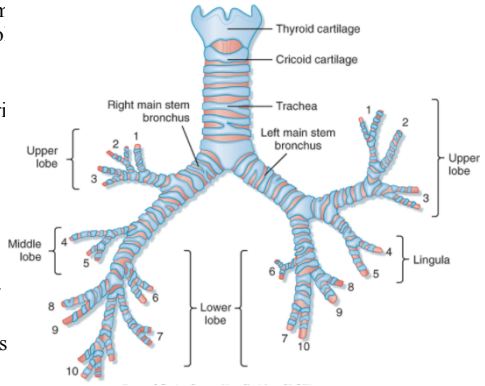
	Structure	**Function**
Nose	R + L cavities due to nasal septum Lined with mucosa	1. **Olfaction** 2. **respiration** 3. **filtration** – vibrissae hairs 4. **humidification** of inspired air – nasal septum + turbinates ↑ SA of mucosa available for evaporation + turbulent flow ↑contact 5. reception of **secretions** 6. Resistance to flow → flow >35L/min → oral breathing
Oral cavity	Oral vestibule + oral cavity proper Roof = palate (hard + soft) Communicates posteriorly with oropharynx	- Breathing at rest (nasal) - Oral breathing (or swallowing): soft palate rigid + arches up and back under control of tensor and levator palate to lie against superior constrictor
Pharynx	from skull base to oesophagus at C6 3 constrictors (sup, mid, inf) Widest = hyoid 5cm Narrowest = oesophageal opening 1.5cm *Nasopharynx:* post. to nasal cavity *Oropharynx:* soft palate to tip of epiglottis. Sensory nerve supply = glossopharyngeal, maxillary, mandibular nerves *Laryngopharynx:* tip of epiglottis to cricoid C6; communicates with larynx via laryngeal inlet	1. **Conducts** air to larynx, trachea, lungs 2. **Directs food** to oesophagus – pharyngeal muscles constrict during swallowing *Nasopharynx:* resp function *Oropharynx:* digestive + conductive function *Laryngopharynx:* phonation + protect lower airway from contents of GIT
Larynx	Level of C3-C6 Cartilages linked together by ligaments Lies opposite to C4-C6 *Laryngeal skeleton:* 9 cartilages - 3 single: thyroid, cricoid, epiglottic - 3 double: arytenoid, corniculate, cuneiform *Interior larynx:* - laryngeal inlet to inf border of cricoid - *epiglottis*: fibrous cartilage + valleculae - *glottis* = vocal apparatus of larynx; made up of vocal folds, vocal processes, rima glottidis Nerve supply: VA + superior and recurrent laryngeal branches - ext + int branches of sup laryngeal nerve → sensation above vocal cords - recurrent laryngeal nerves supply sensation below the cords + motor supply to intrinsic laryngeal muscles	1. **airway protection**: prevents aspiration during swallowing by elevating + occluding aryepiglottic folds 2. **Phonation / speech:** adjusting tension of vocal cords by cricothyroid 3. **Inspiration**: cricoarytenoid muscles rotate arytenoid cartilage + abduct vocal cords → ↓resistance to airflow 4. **Expiration**: thyroarytenoid muscles adduct cords + ↑resistance → intrinsic PEEP (3-4cmH2O PEEP a. maintains patency of small airways + prevents alveolar collapse + maintains FRC 5. **Effort closure:** tighter occlusion of laryngeal inlet → aryepiglottic muscles contract + act as sphincter → airway withstands up to 120cm H2O e.g. cough

Summary of upper airway function

- Nasal breathing: filtration + humidification
- Oral breathing: ↑flow
- Deglutition: nasopharynx occluded; laryngx elevated; aryepiglottic folds approximated
- Airway protection:
  - trisphincteric mechanism (aryepiglottic folds, false vocal cords, true vocal cords)
  - Glottis closure reflex: aryepiglottic muscles
  - Laryngospasm
- Phonation/ speech (upper + lower airways)
  - Tone: larynx
  - Pitch: cricothyroid + thyroarytenoid muscles
- Effort closure: cough/ sneeze/ vomit

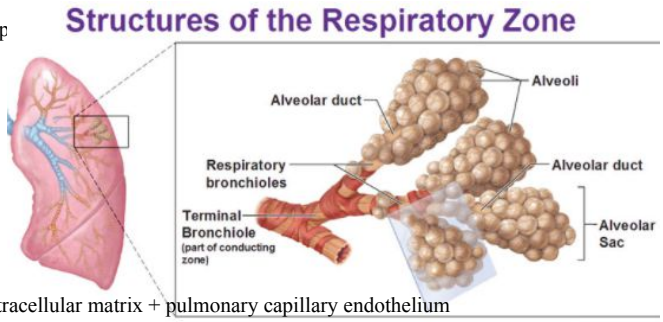
Lower airways

- tracheobronchial tree
- Trachea to alveolus → airways divide 23 times
  - conduction zone: 1st 16 divisions: trachea → main bronchi → lobar bronchi → segm
  - respiratory zone: last 7 divisions: respiratory bronchioles → alveolar ducts → alveo
- **Conducting zone:**
  - *Trachea*
    - fibrocartilaginous tube supported by incomplete cartilaginous rings anteri
    - extends from inf end of larynx into thorax
    - bifurcates at level of transverse thoracic plane
    - mean diameter 2cm; length 10cm
    - external pressure 40cmH2O → occlusion of extrathoracic trachea
  - *Bronchi*
    - 1st 4 divisions of trachea
    - R main bronchus: wider; deviates less from axis of trachea (L has tighter
    - 2 main bronchi divide into 5 lobar bronchi → 18 segmental bronchi
    - segmental bronchi travel with branches of pulmonary artery + lymphatics
  - *Bronchioles*
    - embedded in lung parenchyma
    - do not have cartilage – held open by lung volume
    - resistance to flow is negligible due to large cross sectional area
  - *Terminal bronchioles*



- Flow in conducting zone during inspiration = fast + turbulent
- No gas exchange in conducting zone = anatomical dead space ~150ml in adults
- Blood supply = via **bronchial circulation**
- Mucus secreted by goblet cells in bronchial wall = trap inhaled p
- Cilia move staircase of mucus to epiglottis

- **Respiratory zone**
  - Majority of lung volume
  - Blood supply via **pulmonary circulation**
  - *Respiratory bronchioles*
  - *Alveolar ducts*
  - *Alveolar sacs*
    - Total surface area of lung alveoli: 50-100m2
    - Thin walls: 0.2-0.3um
    - Dense mesh of capillaries 7-10um thick
    - Alveolar-capillary barrier: type 1 pneumocytes + extracellular matrix + pulmonary capillary endothelium
    - Alveoli = composed of 3 types of cells:
      - Type 1 pneumocytes: thin walled optimised for gas exchange; 90% alveolar surface area
      - Type 2 pneumocytes: secrete surfactant → ↓surface tension
      - Alveolar macrophages

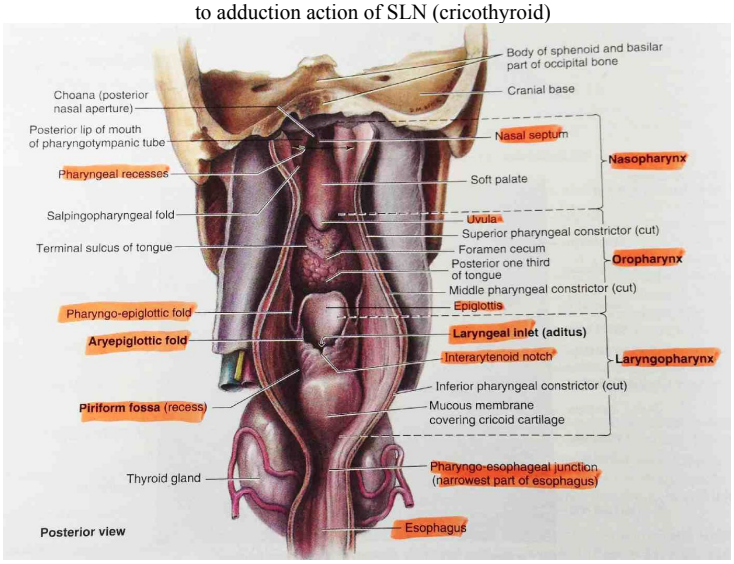


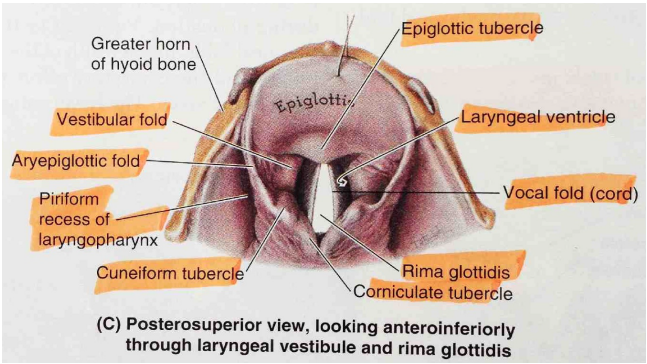
Function of lower airways

1. Prime function = exchange of O2 and CO2
2. Blood/ gas barrier to diffusion: 50-80m2
3. Other:
   a. Blood reservoir
   b. Clotting mechanism: mast cells containing heparin in interstitium
   c. Defence: mechanical; IgA in bronchial mucus; pulmonary macrophages
   d. Synthetic function: surfactant; protein synthesis (collagen and elastin)
   e. Heat regulation + heat exchange
   f. Pharmacokinetics: route of administration e.g. volatiles; effect site e.g. bronchodilators; route of elimination e.g. volatiles

Note: nerves of the larynx

- superior laryngeal nerve
  - Divides into:
    - Internal branch: sensory to: ipsilateral larynx from sup boundary to true cords; pyriform sinus; epiglottis
    - External branch: motor to cricothyroid muscle; sensory to anterior infraglottic larynx cricothyroid membrane
    - Unilateral paralysis → failure of ipsilateral cord closure event with intact RLNs
- Recurrent laryngeal nerve
  - Motor: all ipsilateral intrinsic muscles of larynx except cricothyroid (external laryngeal from vagus)
  - Sensory: ipsilateral mucosa below true cords
  - R RLN longer course, turns aortic arch; R RLN turns around subclavian artery → paralysis of RLN → paramedian vocal cord position due to adduction action of SLN (cricothyroid)





Describe the physiology of the airway including airway reflexes

NB density of sensory innervation greatest at laryngeal inlet

Stretch reflexes

- Inflation
  - Inhibito-inspiratory reflex (**Hering-Breuer**)
    - Pulmonary stretch R in smooth muscle of bronchi + bronchioles → sustained discharge on lung inflation (no adaptation)
    - Stimulated by rate + extent of inflation
    - Effect → ↓ or cessation of insp muscle activity
    - Largely inactive at Vt <1
- Deflation
  - Opposite of Hering-Breuer reflex
  - Lung deflation → ↑frequency + force of resp effort
- Juxtacapillary Rs: non-myelinated C fibres in alveolar walls; activated by tissue damage, oedema, emboli → VA → brady, apnoea, rapid shallow breathing, bronchoconstriction
- Irritant Rs: nociceptive, chemoreceptive, noxious gases → stimulates respiration + bronchoconstriction

Glottic closure reflex

- reflex laryngeal closure produced by rapid contraction of tyroarytenoid muscle in response to SLN stimulation

Laryngospasm

- exaggeration of glottis closure reflex
- maintained well beyond end of stimuli
- most likely when: intubation, light anaesthesia, manipulation of upper aerodigestive tract, FB, blood or mucus
- causes of inhibition of reflex: ↑PaCO2, ↓PaO2, +ve intrathoracic pressure, insp phase of resp

Cough reflex:

- voluntary or involuntary
- 3 phases
  - inspiratory: larynx opens wide → rapid deep insp
  - compressive: tight closure of glottis + strong activation of exp muscles
  - expulsive: larynx opens widely → sudden outflow of air 6-10L/sec

Apnoea reflex

- supraglottic larynx has chemical and thermal sensors → apnoea
- prevents aspiration
- initiation: water

Circulatory reflexes

- stimulation of larynx → ↑BP, ↑VA stimulation → bradycardia → ↓BP
- afferent limb: SLN

Describe the effect of anaesthetic agents and other drugs on airway reflexes

- loss of airway patency due to relaxation of pharyngeal muscles + post displacement of tongue
- ↓ability to manage secretions: build up of saliva + mucus → obstruct oropharyngx
- Loss of cough reflex → allows secretions or gastric contents onto vocal cords → laryngospasm/ bronchospasm/ aspiration

Describe the physiological consequences of anaesthesia and patient positioning on the respiratory system and their management

For patient positioning see General Anaesthesia section

Consequences of anaesthesia

GA effects: ventilation + gas exchange

1. **Ventilation**
   - i. Airway obstruction:
     - loss of airway patency 2° relaxation of pharyngeal muscles + post displacement of tongue
     - ↓ability to manage secretions: ↑saliva + mucus → obstruct oropharyngx
     - Loss of cough reflex → allows secretions or gastric contents onto vocal cords → laryngospasm/ bronchospasm/ aspiration
   - ii. ↓ventilation
     - dose dependent ↓MV: ↓RR (opioids), ↓TV (volatiles), or both (Propofol) → ↓alveolar ventilation → ↑PaCO2 → vasodilation, tachycardia, arrhythmias, HTN, CO2 narcosis, displacement of O2 from alveloli
     - ↓ventilatory response to CO2 → hypercapnia
     - ↓ventilatory response to acidaeamia
     - ↓response to hypoxia
2. **Gas exchange**
   - i. Changes in FRC
     - O2 content of FRC: preoxygenation
     - Vol of FRC: GA relaxes diaphragm + intercostals → ↓FRC 20%
     - Relationship to closing capacity
       - CC = lung vol at which small airways collapse → impeding flow of gas into alveoli
       - During GA the CC approaches the FRC → small airway collapse in normal expiration → atelectasis (effect compounded in neonates, elderly, smokers, resp disease)
   - ii. Changes in ventilation + perfusion
     - Alters distribution of gas + blood within lungs → ↑V/Q mismatch, ↑shunt (atelectasis) → ↓oxygenation of blood
   - iii. ↓HPV: volatiles
   - iv. other:
     - ↓ciliary activity
     - dry gas → mucous plugging
     - volatiles can be directly irritatnt to airways → cough
     - ketamine + neostigmine → ↑saliva + mucous production

Oxygenation dependent on:

- FiO2
- Patent airway
- Adequate alveolar ventilation
- V/Q matching
- Transfer of O2 across alveolar + endothelial membranes

Effects of mechanical ventilation on lung tissue

- ARDS
  - Direct damage to lung parenchyma
  - TV >12ml/kg → alveoli shear stress → release of inflammatory mediators IL6, IL8 → interstitial alveolar oedema → ↓lung compliance +

↓gas transfer → hypoxia
    - Protective lung strategies: high PEEP to maintain alveolar patency + low TV 6ml/kg to ↓shearing stress
- Barotrauma (pneumothorax)
    - High insp pressures or large TV
    - More likely in stiff, non compliant lungs (ARDS) or non elastic lungs (COPD)
    - Effect exacerbated if uneven distribution of disease (and therefore compliance) in lung

Managing effects of anaesthesia on resp system
- Preparation
    - Positioning patients at 45o prior to induction to ↓fall in FRC
    - Preoxygenation to maximise O2 content of FRC → ↑ time from apnoea to hypoxia
    - Antimuscurinic drugs: ↓saliva in airway
- Intraoperatively
    - Mechanical ventilation:
        - ↓airway collapse + atelectasis
        - PEEP maintains alveolar patency + prevents hypoxia
    - PEEP + recruitment manoeuvres:
        - Open collapsed portions of the lung
        - Recruitment: high PEEP e.g. 5 breaths at 30cm H2O and then 10 breaths at 20cmH2O
        - PEEP 5-10cmH2O throughout anaesthesia can maintain lung expansion → ↑oxygenation + compliance
    - Lung protective ventilation
    - Avoid long periods of 100% O2 → absorption atelectasis
    - Maintain adequate perfusion pressures with IV fluids + vasopressors to ↑perfusion of non-dependent lung areas + ↓V/Q mismatch
    - HME: maintains mucous clearance + prevents tracheal dyskariosis + mucous plugging
- Post-operatively
    - O2 post anaesthetic: maintains adequate alveolar concentration to correct effects of hypoventilation, V/Q mismatch, diffusion of anaesthetic gases into alveoli
    - Obese patients: CPAP/ bilevel
    - Postop analgesia: deep breaths + cough

Table 1. Summary of the effects of the main anaesthetic drugs on the respiratory system.

Inhalational agents	*Positive effects*	*Negative effects*
Nitrous oxide	No significant change in MV – CO_2 normal range Non-irritant	
Isoflurane	Bronchodilation	Reduced MV due to reduced TV Reduced response to hypoxia & hypercarbia Pungent – causes coughing Increased secretions
Sevoflurane	MV stable due to increased RR at lower doses. Bronchodilation Non-irritant	Hypercarbia Depressed response to CO2 Inhibition HPV
Halothane	Bronchodilation Non- irritant Reduced bronchial secretions	Reduced MV due to reduced TV Blunted response to hypoxia and hypercarbia Inhibits HPV
Enflurane	Non-irritant Bronchodilation No increase in secretions	Reduced MV (more than other volatiles) Hypercarbia Blunted response to hypoxia & hypercarbia Inhibits pulmonary macrophages and mucociliary transport

Iv Induction agents	*Positive effects*	*Negative effects*
Thiopentone		Dose dependent respiratory depression Increased bronchial smooth muscle tone with increased bronchospasm & laryngospasm
Propofol	Laryngeal relaxation – ease of LMA insertion Bronchodilation May preserve HPV	Respiratory depression Reduced response to hypoxia & hypercarbia
Etomidate		Dose dependent reduction in MV Apnoea Coughing
Ketamine	Preserved laryngeal reflexes Maintain patent airway Less respiratory depression Reduction in bronchial smooth muscle tone	Increased saliva and mucous production
Opiates	Anti-tussive	Respiratory depression Chest wall rigidity Bronchospasm

NB: Hypoxaemia due to small shunts can be corrected by increasing the inspired O2 concentration, but once the amount of blood being shunted exceeds 30%, the hypoxia cannot be corrected even by breathing 100% oxygen.

Describe different modes of ventilation available on modern ventilators and their physiological consequences

IPPV
- ↑pleural pressure
- ↑intraabdo pressure
- ↑lung volumes
- Effects:
    - ↓preload: ↑intrathoracic pressure → ↑RAP → ↓VR
    - ↑PVR 2o compression + stretching pulmonary vessels
    - ↓LV afterload
    - ↓myocardial O2 demand
    - ΔCO: ↓CO in preload dependent; ↑CO in afterload dependent

Modes of venstilation
- **Assist control modes**
    - Breaths can be initiated by patient (assisted breaths) or ventilator (control breaths)
    - Characteristics of the breath are the same regardless of whether tha patient or ventilator initiates the breath
    - If pt does not initiate breaths to ensure min vent rate reached → vent will initiate enough breathes to make up the difference
    - If pts spont breathing rate is > minimum set rate → vent will not initiate any breaths (cf SIMV)
    - Can be: volume control, pressure control, PRVC
        - **Volume control:**
            - VT, duration of inspiration, insp pause time, insp flow = determined by vent settings
            - Flow rises rapidly then remains constant → constant ↑vol and ↑airway pressure
            - Flow pattern = square wave
        - **Pressure control**

- Insp pressure set instead of tidal vol
- Application of constant pressure during inspiration → high initial flow → ↓exponentially to 0 by end of inspiration
- Oxygenation may be better because of the flow pattern
- Volume rises less exponentially
- Insp pause is often built into the breath as there is minimal flow in later part of insp phase

- **PRVC**
  - Constant pressure applied throughout inspiration (like pressure control), however operator sets desired VT
  - Ventilator adjustsinsp pressure from breath to breath as pts airway resistance and resp system compliance changes, in order to deliver that tidal vol

- **SIMV**
  - Usually combined with pressure support
  - Patient receives **set number of mandatory breaths**, which are synchronised with any attempts by patient to breathe
  - Pt can take additional breathes between mandatory breaths - usually pressure supported (designed to ↑synchrony)
  - When pt attempts to take a breath + triggers vent, whether a synchronised mandatory breath or PSV breath depends on whether triggering occurs during SIMV period or during spont period
    - If triggered during SIMV period → delivers synchronised mandatory breath
    - If triggered during spont period → delivers PSV
    - If not triggered during SIMV period → delivers mandatory breath towards the end of SIMV period

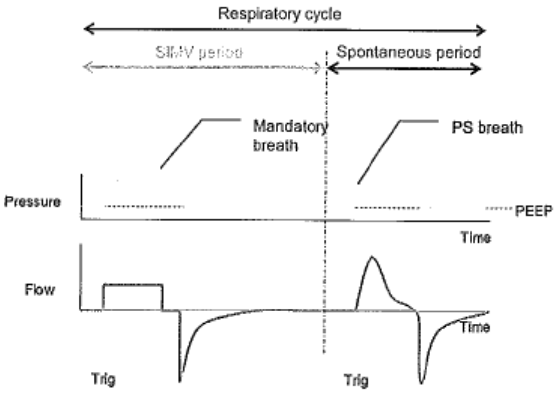
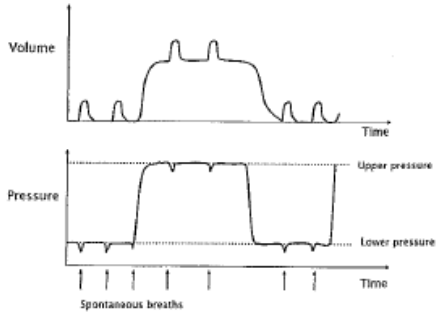


Figure 1. Pressure and flow waveforms in SIMV mode.

- **Assist modes**
  - **PSV**
    - Operator sets inspiratory pressure → delivered every time the patient initiates a breath
    - If pt doesn't initiate breath → no breaths are given (newer vents switch to backup mandatory)
    - Cycles from insp to exp when insp flow ↓s to preset proportion of peak insp flow
    - NB PS of 4-15 is required to overcome the additional WOB due to breathing through ETT and demand valve of ventilator
  - Volume support
    - Spont breathing equivalent of PRVC
    - Form of PSV in which insp pressure is adjusted on breath by breath basis to achieve pre-set target tidal vol
    - Set VT delivered with differing amount of pressure support depending on patient effort, resistance, compliance
  - Neurally adjusted ventilatory assist
    - Form of PSV in which breath is initially triggered by the diaphragmatic EMG and then the pressure delivered ∝ amplitude of EMG
    - EMG continuously sensed by electrodes on modified NGT → allows pressure to be adjusted with each breath
    - Insp to exp cycling occurs when diaphragmatic electrical activity ↓to 70% of the peak activity
- Other modes
  - Bilevel airway pressure
    - 2 levels of positive airway pressure for preset periods of time → pt able to take spont breaths at both high and low pressure levels
    - Oxygenation can be ↑ by ↑both high + low pressures or by ↑time at high pressure
    - CO2 elimination is dependent on difference between high + low pressure, frequency of cycling between high + low pressures, and spont breaths



    - *Figure 1. Bilevel airway pressure ventilation.*
  - Airway pressure release ventilation (APRV)
    - Inverse ratio ventilation
    - Pt breaths spontaneously at optimal FRC (maintained by high pressure CPAP) that supports oxygenation; and release periods to low pressure CPAP augment ventilation and CO2 removal
    - Time at low pressure is very short; almost all spont breaths occur at high pressure
    - Allows spont brathing, ↑alveolar recruitment, ↓sedation + paralysis

Adjusting mechanical ventilation

Action	Adverse effects	Range of values associated with low risk of adverse effects

↑FiO2	O2 toxicity	0.21-0.5
↑PEEP	CVS effects due to ↑mean intrathoracic pressure ↑airway and alveolar pressures with risk of barotrauma	0-10
↑insp time	Shorter expiratory time with risk of gas trapping CVS effects due to ↑mean intrathoracic pressure	<50% resp cycle
↑tidal vol or insp P	↑airway and aveolar pressures with risk of barotrauma only minor effect on oxygenation and may ↑dead space	Tidal vol <8ml/kg Insp pressure (including PEEP) <30cmH2O
↑insp ppause	↓insp flow time	5-10% resp cycle time
Position patient	Potential haemodynamic disturbances	
↑RR	Shorter expiratory time → gas trapping → barotrauma, haemodynamic compromise ↑dead space ventilation	

Trouble shooting mechanical ventilation

- airway pressure
  - airway pressure = flow x resistance + alveolar pressure
    - measured in the ventilator, not the patients airway
    - under normal circumstances airway pressure ~ = alveolar pressure because resistance is low
    - ↑↑ airway pressure → ALI, ARDS; pressure transmitted from alveoli to pleural space will have haemodynamic effects
  - ↑ airway pressure may be due to:
    - problem in ventilator: inappropriate setting; vent malfunction
    - problem in circuit: kinking; pooling condensed water vapour; wet filters → ↑resistance
    - problem with ETT: kinked; obstructed; endobronch intubation
    - problem in patient : bronchospasm; ↓lung compliance (pulmonary oedema, consolidation, collapse); ↓pleural compliance (pneumothorax); ↓chest wall compliance (abdo distension); pt/vent dysynchrony, coughing
  - Distinguish problem with vent/ circuit from patient by disconnecting pt from vent and BVM
- Alveolar pressure
  - Alveolar pressure = (volume/ compliance) + PEEP
  - Insp pause pressures used to obtain estimate of alveolar pressure
  - Pressure measured at end inspiration when there is no flow
    - Airway pressure = flow x resistance + alveolar pressure. If there is no flow then…
    - Airway pressure = 0 x resistance + alveolar pressure and therefore …
    - Airway pressure = alveolar pressure
  - ↑alveolar pressure due to excessive tidal vol, gas trapping, PEEP, or low compliance

Basics

- Pressure = flow x resistance
- Flow = volume / time
- Alveolar pressure = (volume / compliance) + PEEP
- Airway pressure (total pressure) during inspiration: airway pressure = flow x resistance + alveolar pressure
  - $\text{Airway pressure} = \text{Flow} \times \text{Resistance} + \frac{\text{Volume}}{\text{Compliance}} + \text{PEEP}$
- alveolar ventilation = RR x (tidal vol – dead space)
- transmural pressure = alveolar pressure – pleural pressure
- time constant = lung compliance x airway resistance
  - ime constant = the time required for inflation to 63% of the final vol that is attained if inflation is prolonged indefinitely
  - directly related to lung compliance and airway resistance
  - fast alveoli = short time constants → filled faster

Ways to ↑oxygenation and ventilation

- **↑oxygenation by:**
  - **↑FiO2**
  - **↑mean alveolar pressure** → ↓shunting by reopening alveoli + keeping them open with PEEP. Ways to ↑mean alveolar pressure:
    - **prolong inspiration**: allows for more even distribution of ventilation + ↑ventilation of less compliant alveoli
    - **↑tidal vol or insp pressure** → recruitment (re-expansion of collapsed alveoli) → ↓shunt
    - **PEEP**
  - ↑alveolar ventilation (to a lesser extent)
- **↑ventilation**
  - **↑minute ventilation**: ↑RR or ↑tidal vol
  - ↑RR is less efficient method of ↑alveolar vent as dead space vent is ↑. NB that excessive ↑tidal vol → over distension of alveoli + compression and stretching of alveolar capillaries → perfusion to these alveoli and ↑dead space ventilation
  - **↓dead space**

Minimising lung injury

- minimise FiO2
- tidal vol <8ml/kg predicted lean body weight (calculated from height and gender)
- maintain alveolar pressure <30cmH2O
- PEEP 5-10cmH2O

FRC

- Lung volume at the end fo a normal expiration
- Results from balance between inward elastic recoil of lung and outward movement of chest wall due to elastic recoil and residual end-expiratory muscle tone
- Provides O2 reserve and buffer to maintain steady arterial O2 concentration
- Maintenance of FRC:
  - Prevents atelectasis
  - Keeps airway resistance low
  - ↓V/Q mismatch

- ↓WOB
- GA → ↓FRV 15-20%
  - Supine position → compression of lung
  - Loss of N2 splinting if using ↑FiO2
  - Change in chest wall shape by ↓ cross sectional area of ribcage
  - Loss of muscle tone
  - Result: FRC < CC → atelectasis

Regional ventilation

- distending pressure of alveolus = transmural pressure
- transmural pressure = alveolar pressure – pleural pressure
- ventilation not homogenous 2o differential effects of gravity on alveolar and pleural pressure
  - alveolar pressure does not vary with vertical height of lung
  - intrapleural pressure becomes ↑-ve (or less +ve in the case of ventilated pts) in uppermost part of the lung due to effect of gravity → larger transmural pressure in uppermost part → larger alveoli

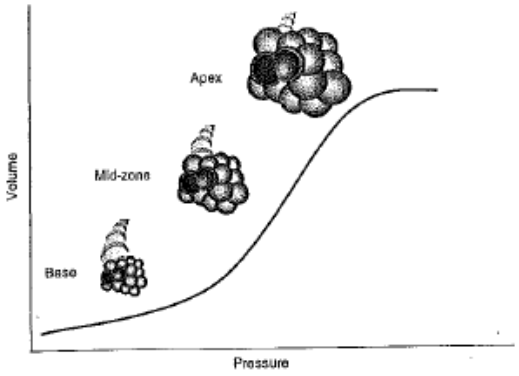


Figure 5a. Healthy adult in upright position at FRC. Lung units at apex are already well inflated while basal units are deflated but not collapsed. Units at the apex are on flat part of compliance curve so that an increase in transmural pressure produces little change in volume (figure 5b), in contrast to units in mid and basal zones.

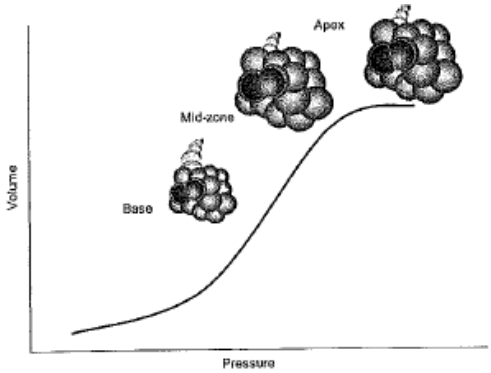


Figure 5b. After lung inflation.

  -
  -

Perfusion

- pulmonary circulation = ↓resistance, ↓pressure system
- regional differences in perfusion
- driving pressure is affected by gravity: driving pressure is lowest in uppermost portion of lung and highest in dependent portions
- ↑PVR → ↓blood flow
- Pulmoanry vascular resistance is affected by:
  - Pulmonary arterial pressure: ↑PAP → ↓resistance due to recruitment + distension of blood vessels
  - Lung vol: over distension of alveoli stretches and compresses pulmonary capillaries
  - Alveolar partial pressure O2:
    - Alveolar collapse → local HPV → ↓perfusion to collapsed areas → ↓shunt
    - Conversely, reexpansion → ↑perfusion
  - PaCO2 and pH: ↑PaCO2 + acidosis → ↑PVR
  - Neural / hormonal control
  - Blood viscocity and Hct

PPV results in:

- ↑pleural pressure (more closely related to alveolar pressure and lung vol than aurway pressure)
- ↑intraabdo pressure
- ↑lung volumes
- above changes affect preload, afterload, PVR, myocardial O2 consumption, and total body O2 consumption
  - ↑inrathoracic pressure → ↑RAP → ↓VR → ↓preload. Effect minimised by:
    - compensatory ↑SY tone
    - ↑intra-abdo pressure → ↑mean systemic venous pressure
  - Overinflation of lungs
    - ↑PVR 2o compression and stretching of pulmonary vessels
    - may cause or exacerbate RHF → RV distension
  - LV afterload
    - LV wall tension (afterload) = transmural pressure x radius / 2x wall thickness
    - Transmural pressure = intraventricular pressure – pleural pressure
    - PPV → ↑pleural pressure
    - Therefore transmural pressure and afterload are ↓by PPV
  - Myocardial O2 consumption
    - Determined by sum fo stroke work and elastance defined potential wprk (potential energy in the ventricle at end systole)
    - Mechanical ventilation generally ↓ preload and afterload → shifts the pressure-vol loop to the left and down → ↓elastance defined potential work and thus myocardial O2 consumption
    - In pts with myocardial ischaemia → ↓myocardial O2 consumption may improve balance between O2 demand and supply → improvement in LV function. In these patients mechanical ventilation may ↑LV contractility
  - Cardiac output
    - Overall effect depends on whether CVS is more preload dependent or afterload dependent
    - Pts who have normal CVS are usually more preload dependent. In normal pts, or hypovolaemic: PPV → ↓CO
    - Pts with RV failure + hyperinflation may → ↓CO
    - Pts with congestive heart failure are usually more afterload dependent: PPV → ↑CVS function

Outline the equipment required to be immediately available for basic airway management and the 'can't intubate, can't oxygenate' situation

- Basic airway management
  - BVM
  - Oropharyngeal + nasopharyngeal airway
  - LMA
  - Laryngoscopy
  - ETT 2 sizes
  - Bougie
- Surgical airway kit

Note: cricothyroidotomy

- Boundaries of the cricothyroid membrane:
  - Superiorly: thyroid cartilage
  - Inferiorly: cricoid cartilage
  - Laterally: cricothyroideus muscles on both sides
- cricothyroid membrane is ~2cm caudal to laryngeal prominence
- cricothyroid arteries = branches of superior thyroid arteries; course along both sides of cricothyroid membrane + anastomose in midline, closer to the superior border of membrane. → avoid by incising membrane in its lower 1/3.
- Equipment + technique

	Equipment	Technique
Dilational cricothyroidotomy	- kink resistant cannula - 10ml syringe - NS - high pressure ventilation system e.g. manujet	- ID cricothyroid membrane → stabilise cartilage → midline vertical incision - Attach needle to syringe filled with NS → advance needle through cricothyroid membrane → confirm posit by asp of air - Seldinger technique needle/ wire/ dilator - Leave airway caheter in place → attach to vent system - if ventilation fails or surgical emphysema or complication develops → convert to surgical cricothyroidotomy
Surgical cricothyroidotomy	- scalpel - bougie - cuffed tracheal or tracheostomy tube	- identify cricothyroid membrane → horizontal incision → rotate 90o to make triangular hole - hold scalel → insert bougie using blade of scalpel as guide - insert 6mm cuffed ETT over bougie - ventilate with low pressure source - verify tube position + pulmonary ventilation

Jet ventilation

- Manujet + ventrain
- Deliver O2 at high pressures (1000-4000cmH2O; 1-4 ATM)
- Deliver O2 at small bursts at pressures of 1-4ATM
  - Ventrain: flow rate 15L/min
  - Manujet: gradually ↑ inflation pressure; minimum 2-2.5ATM
- Complications
  - Barotrauma: pneumothorax; pneumomediastinum; pneumopericardium; pneumoperitoneum
  - Subcut emphysema
  - Malposition of catheters
  - Gastric distension
  - Dysrhythmias
- Contraindications
  - Do not have facility to assist expiration – don't use in pts with complete upper airway obstruction

Note: tracheostomy

- Trachea extends from cricoid cartilate (C6) to carina (T4), manubiosternal junction
- Anterior structure superiorly, but descends posteriorly becoming deep mediastinal structure inferiorly
- 15cm long; 1/3 in neck
- supported by C shaped cartilages anteriorly and laterally; joined posteriorly by membranous wall

Anatomy

- Anterior relations
  - Skin → superficial fascia → sternohyoid + sternothyroid muscles → deep (pretracheal) fascia
  - Thyroid isthmus at level of 2-4th rings; branches of sup thyroid artery run along sup aspect of isthmus
  - Inf thyroid veins lie ant to lower part of cervical trachea; post to strap muscles
  - Ant jugular veins often connected by vein that runs superficially across lower neck, ant to sternohyoid and sternothyroid muscles
  - Brachiocephalic artery crosses mid trachea (in young pts can run close to sternal notch)
- Posterior: oesophagus
- Laterally: thyroid lobes
- Posteriolaterally:
  - Carotid sheaths + VA nerves
  - RLN in groove between trachea + oesophagus

Indications

- Upper airway obstruction
- ↓duration of orotracheal intubation: ↑comfort; facilitate nursing care; ↑communication; facilitate PO intake of food; psychological benefit; facilitate tracheal suction

Contraindications

- Soft tissue infection of neck
- Grossly abnormal anatomy of neck

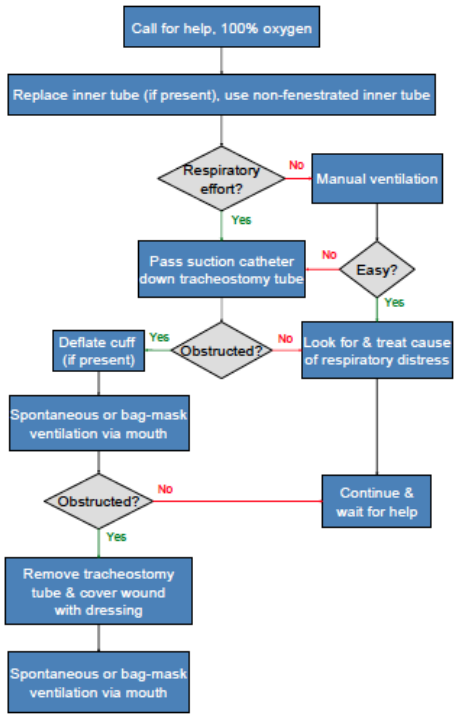


Figure 24 Management of possible blocked tracheostomy

Complications

- Blockage
- Bleeding
    - Early or late: early usually not significant; late rare but ↑mortality (97-100%)
    - Most common cause: peristomal infection or erosion of trache tube through innominate artery (usually heralded by sentinel bleed)
    - Resus + haemorrhage control:
        - Ensure patent airway – suction
        - Hyperinflate trache cuff using 50ml → withdraw trache tube slightly to apply pressure to anterior tracheal wall by hyperinflated balloon
        - If fails; consider replacing trache tube with entotracheal tube via PO route and hyperinflating cuff
        - Apply digital pressure in sternal notch to compress innominate artery
        - Wide bore IV access; massive blood transfusion; emergency transfer to theatre exploration
        - NB profuse tracheal bleeding can rapidly lead to obstruction of trachea by clot; if doesn not respond to tracheal suction → use bougie to push the clot down main bronchi to allow vent of at least one lun

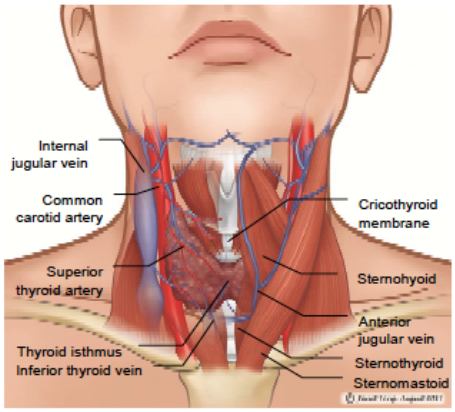
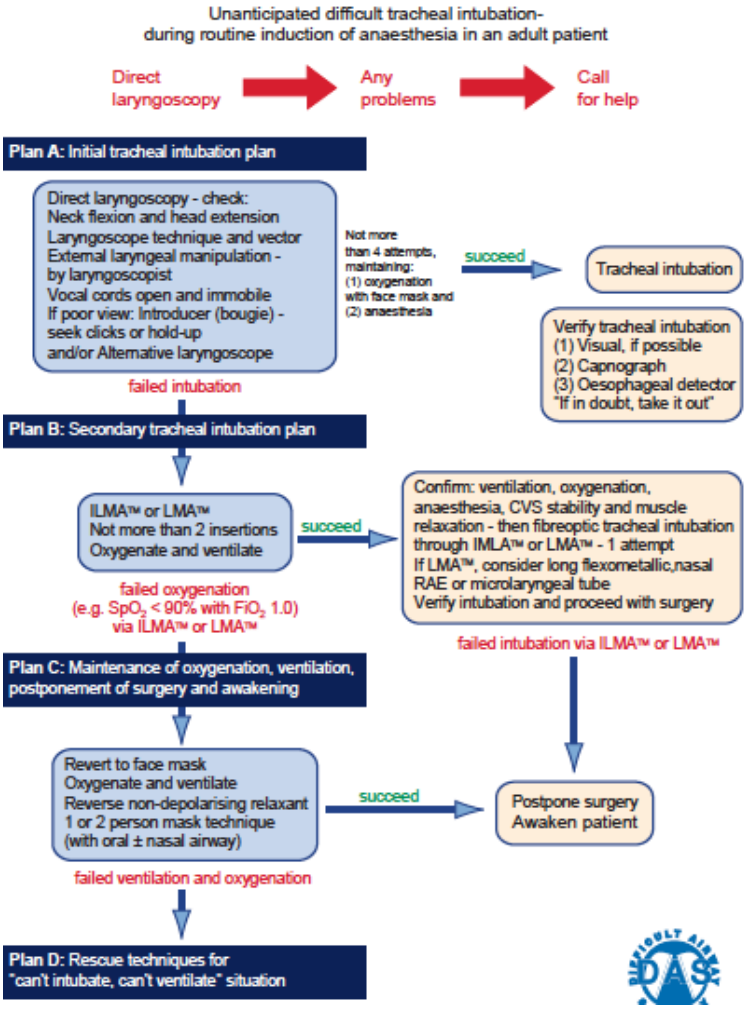
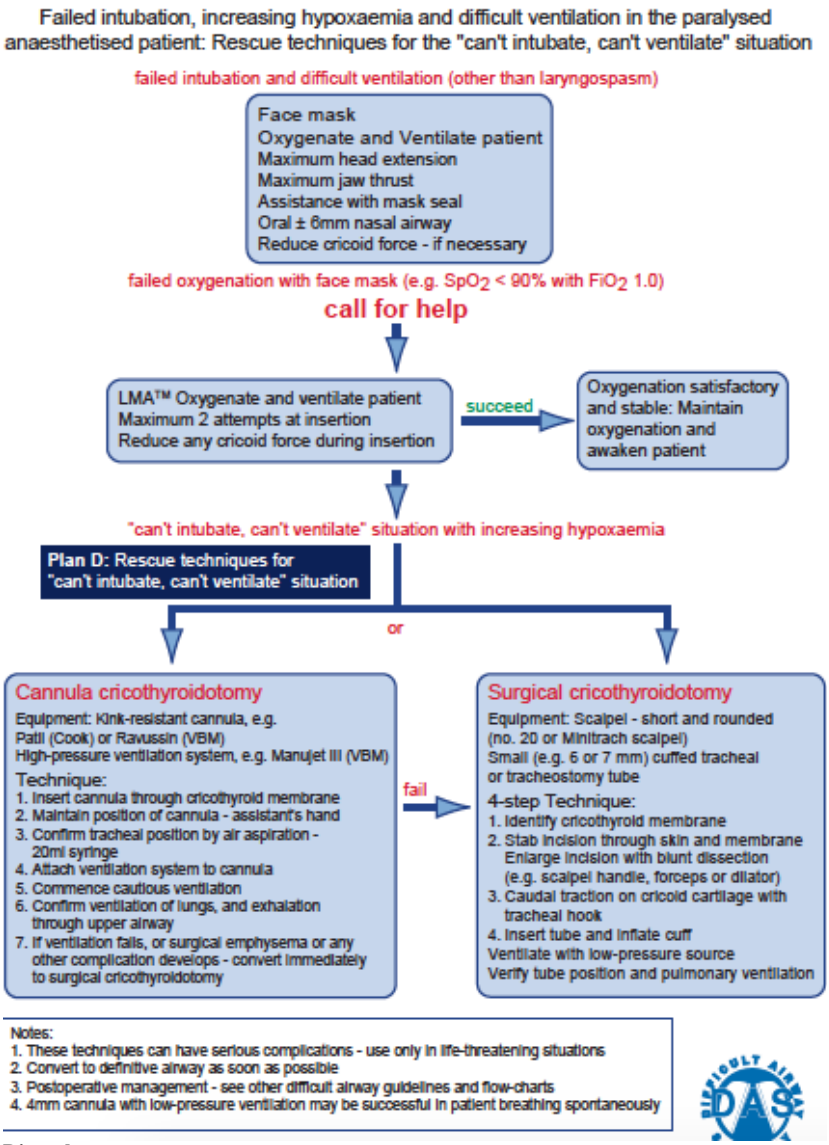


Figure 1. Anatomy of the neck relevant to tracheostomy. On the right the more superficial structures (eg sternal head of sternomastoid, sternohyoid and omohyoid muscles, anterior jugular vein) have been removed to allow deeper structures to be shown.





Direct laryngoscopy

- align oral, pharyngeal, laryngeal axis + manage tongue and epiglottis
- laryngoscope blades
  - macintosh
    - curved blade; sweeps away tongue; elevates epiglottis
    - benefits: non-traumatic tip, shielded light bulb, indirect elevation of epiglottis (due to hypoepiglottic ligament tensioning when blade correctly placed in vallecula)
  - Straight (miller) blade
    - Positioned posterior to epiglottic, trapping it while exposing glottis + vocal folds
    - Benefits: better tongue control; improved epiglottic elevation; ↓force; ↓head extension
    - Indivations: laryngeal/ lingular lesions; hypoplastic mandible; awkward teeth
  - Angled head; angled blades (Kessel), short handles
    - Good for obese patients/ large breasts
  - McCoy blade
    - Macintosh-shaped with hinged tip to flex when lever pressed
    - Indications: floppy epiglottis

LMA

- silicone tube connected to distal elliptical spoon shaped mask with inflatable rim
- positioned blindly into pharynx
- forms low-pressure seal against laryngeal inlet
- uses:
  - Rescue device in failed intubation
  - Quick surgical procedures not involving paralysis; pneumoperitoneum, asp risk, obese etc
- Contraindications
  - Inability to open mouth
  - Pharyngeal pathology
  - Airway obstruction at or below larynx
  - Low pulmonary complianceor high airway resistance
- Types of LMA
  - Classic
  - Intubating LMA: conduit for blind or fibreoptic intubation; specially designed to allow passage of bougies, bronch, ETT
- Insertion:

- Tip of cuff continuously applied to hard palate → R index finger placed at junction of mask and tube and guides tube along back of tongue until firm resistance encountered
- Cuff inflated to 20-40ml air
- Lifting jaw elevates tongue + epiglottic rom post pharyngeal wall and may aid positioning

- Complications
  - Aspiration
  - Gastric insufflation
  - Partial airway obstruction
  - Coughing
  - Laryngospasm
  - Post-extubation stridor

Fibreoptic intubation

- Bronchoscopic anatomy
  - Ant trachea: semi-circular rings; post wall has vertical lines running towards carina
  - Carina: demarcation between L and R main bronchi = sharp; runs directly ant-post
  - L main bronchus runs 5cm before giving off LUL bronchus
    - LUL bronchus runs 1cm → divides into sup and inf (ilngular) divisions
    - Sup division → divides into apical, ant, and post segments of upper lobe
    - Inf division of LUL bronchus deflets down to lingular portion of LUL → divides into sup branch + 3 basal branches
  - R main bronchus: short; gives off RUL bronchus → trifurcates → continues as bronchus intermedius → gives off smaller RML and larger RLL bronchi
    - RML branches into 2; RLL into 5 segments (1 apical; 4 basal)
- Use of fibreoptic scopes
  - Airway assessment
  - ETT insertion
  - Confirmation of ETT position
  - Endobronchial suction
  - Insertion of airway catheter e.g. Aintree

Describe preoxygenation, including its physiological basis

Preoxygenation = breathing 100% O2 for 3-5 mins (or 4 VC breaths)

- aim: denitrogenate lungs → oxygenation of FRC >1800mls O2 → ↑time to desaturation of 7-8 mins
- best way to measure effectiveness of preoxygenation is measure ET O2 fraction (FEO2)
  - FEO2 ~ = FAO2
  - Use alveolar gas equation to understand % of O2 in lung
    - 149-40/0.8 = 100mmHg
    - 100mmHg as % of 1 atmosphere (760mmHg) = 100/760 x 100 = 13%
  - therefore typical FRC vol = 2.2 L which in RA contains 13% O2 = 270mls O2
  - in normal adult with complete preoxygenation (FAO2 >0.9) → lungs should contain around **2000ml O2**
  - Total body O2 consumption = ~250ml/min
    - Therefore apnoea with normal stores takes ~1min (270/250)
  - If FRC preoxygenated with FiO2 1
    - 760 – 47 (40/0.8) = 663
    - 663/760 x 100 = 0.87
    - 2200 x 0.87 = 1914mls
    - 1914 / 250 = 7.65mins

Total ventilation

- Vt = 500ml X RR
- 500 x 15 = 7500ml/min → vol of air entering is slightly greater as >O2 is taken in than CO2 is given out

Alveolar ventilation

- VT – dead space x RR
- Amount getting to respiratory zone
- Anatomic dead space = 150mls → therefore alveolar vent = 500 – 150 x 15 = 5250ml/min

Partial pressure of gas

- partial pressure of gas = concentration x total pressure
- when air inhaled it is warmed + moistened
  - water vapour pressure = 47mmHg → total dry gas pressure = 760 – 47 = 713
  - therefore PiO2 inspired air = 21/100 x 714 = 149mmHg

Alveolar gas equation

- allows relationship between fall in PO2 and rise in PCO2