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3.Ventilation

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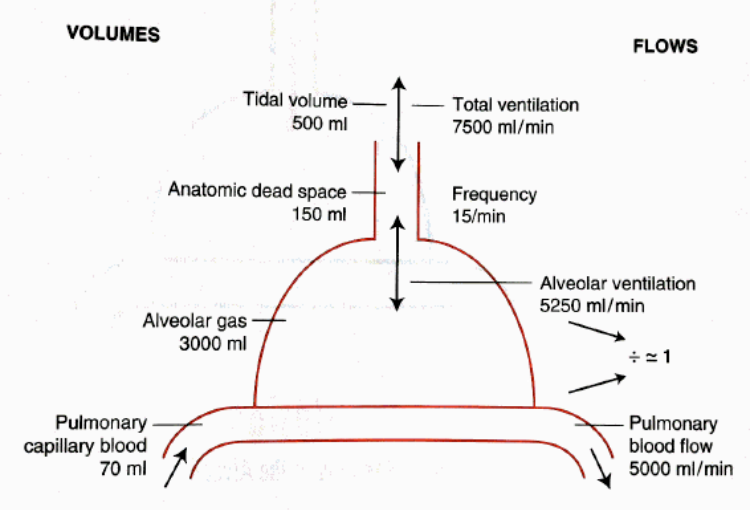
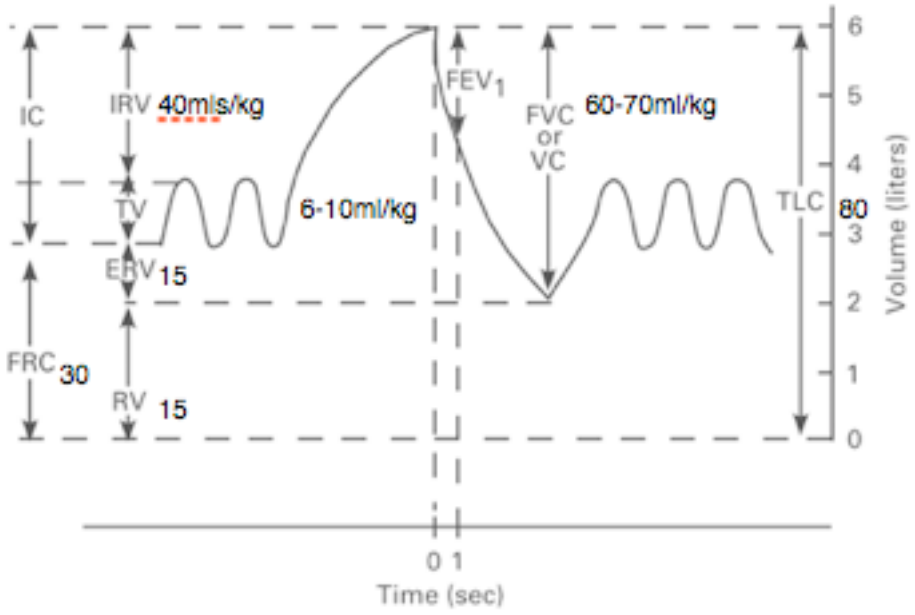
Volumes

- Prime role of lung = gas exchange



Static Volumes

- This = spirometry trace which includes a max insp & expiratory effort:



- Volumes:
  - RV = residual volume (**15**-20ml/kg)
    - Volume left after forced expiration
  - ERV (10-**15**ml/kg)
    - Volume forcefully expired after normal tidal exp
  - IRV = (**45**ml/kg) volume inspired over norm tidal insp

↳ All above values can be measured by spirometry.
↳ spirometry cannot measure RV

- Volumes added together = capacity
  - Total lung capacity (**75**-80ml/kg) (RV+ERV+TV+IRV)
  - Vital capacity (**60**-70ml/kg) (TV+IRV+ERV)
  - Functional residual capacity (**30**ml/kg) (ERV+RV)

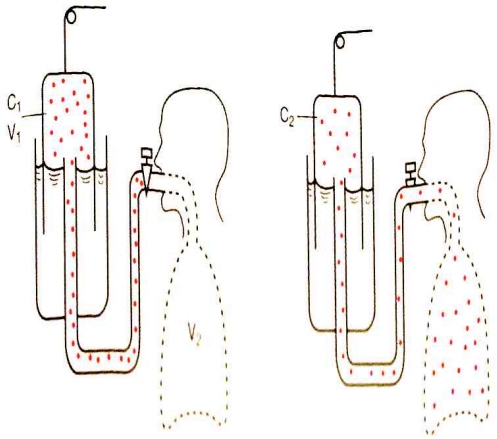
7,5 ml/kg	**- TV**
15	**-RV**
15	**-ERV**
30	**-FRC**
45	**- IRV**
60	**-VC**
75	**-TLC**

Techniques to Measure RV/FRC/TLC

Helium Dilution Technique

- Helium dilution technique & spirometer:
  - Virtually insoluble in blood
  - Method:
    - Closed circuit system
    - Amount of helium in in spirometer is known at beginning of test
      ↳ concentration (C1) x volume (V1) = amount
    - Pt then breathe number of tidal volume starting at FRC ⇒ helium spreads & equilibrates into lungs from circuit
    - Spirometer measures change in concentration of helium in whole circuit (now including lungs)
    - ∴ can now derive volume of lungs :

$$V2 = \frac{V1 \times (C1 - C2)}{C2} \qquad V2 = FRC$$



Before equilibration After equilibration

- Nitrogen washout (dilutional method) – see later

Body Plethysmograph

- Body plethysmograph (BPG):
  - Whole person in airtight box, breathes normally
  - At some stage shutter closes off airway ie pt making resp effort against closed airway:
    - End normal expiration = FRV
    - End full expiration = RV
  - 2 manometers in place:
    - Box pressure (P1 & P2)
    - Mouth pressure = airway pressure (P3 & P4)
  - Boyles Law (P x V = K) = pressure x volume = constant (at a constant temp)
  - Calculation of values in the box:
    - Before resp effort is made certain volumes are known:
      - Pressure in box = P1
      - Volume in box = V1
    - Pt now makes resp effort against closed airway and chest expands by unknown (ΔV)
    - Volume of box must be decreased by ?amount as chest expands
    - Boyles Law ∴ says pressure must go up = P2 which measured in box
    - Leaves calculation:

$$P_1 \times V_1 = P_2 \times (V_1 - \Delta V)$$

Want to know ΔV so rearrange equation:

- Calculation of values in the patient:
  - Before effort against closed airway the mouth pressure (P3) and lung volume = FRC
    ↳ or RV (depending on when closed)
    - After effort pts lung volume ↑s by ΔV (which we know from box pressures)
    - ∴(boyles) mouth pressure must decrease (P4)
    - so for the patient:

      $P_3 \times FRC = P_4 \times (FRC + \Delta V)$

    - ∴ FRC is only unknown variable which can be calculated by rearranging equation

- which to chose:
  - ∴ in diseased lung should use BPG
    ↳ other techniques wont measure volume behind blocked airways

Ventilation

Total Ventilation

- Vt = 500ml & RR 15/min:
  - Total ventilation: = Vt x RR
    500 x 15 = 7500ml/min
    ↳volume of air entering is slightly greater as more 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 ∴ alveolar vent = 500 – 150 x 15 = 5250ml/min
- represents amount fresh air available for gas exchange
- can ↑alveolar vent by ↑ing Vt or ↑ing RR
  ↳but ↑ing Vt is more efficient as less wasted to dead space
- can be measured by
  - calculating dead space ventilation :
    - (volume x resp frequency) and subtracting from total ventilation
    - but measuring dead space can be difficult
  - concentration of CO2 in expired gas
    - no gas exchange in anatomic dead space ∴ no CO2 at end inspiration
    - PCO2 of alveolar gas & arterial blood are virtually identical
      ↳allows arterial PCO2 to determine alveolar ventilation
    - If alveolar vent is halved ⇒ x2↑ alveolar & arterial PCO2 (if Co2 production constant)

Dead Space

Definitions

- Anatomical DS = volume of conducting airways (ie volume of gas in airways NOT alveoli)
- Alveolar DS = volume of air beyond conducting airways which does not participate in gas exchange (ie V/Q infinity)
- Physiological DS = that part of tidal volume which does not participate in gas exchange
  ↳ ie sum of anatomical + alveolar DS

= a functional assessment

- Alveolar gas = gas from alveoli that are both ventilated and perfused ie alveoli taking part in gas exchange & ∴ contain CO2
- Ideal alveolar gas = theoretical gas from alveoli with V/Q 1
- Mixed expired gas:
  - = one or more complete breaths of expired gas coming thoroughly mixed from physiological dead space & alveoli
  - ∴ contains content from:
    - ideal alveolar gas
    - alveolar DS
    - anatomical DS
  - usual indicated by $\bar{E}$. (eg $P_{\bar{E}}CO_2 = pCO_2$ in mixed exp gas)
- End expired gas (aka end tidal gas):
  - Contains:
    - Ideal alveolar gas
    - Alveolar DS
  - Inidicated by E'

Effects on Dead Space

Anatomical dead space is ***increased*** in:
i. Old age
ii. Neck extension
iii. Jaw protrusion
iv. Bronchodilators
v. Increasing lung volume
vi. Atropine (causes bronchodilation)
vii. Anesthesia mask, circuits
viii. Intermittent positive pressure ventilation (IPPV) and positive end expiratory pressure (PEEP).

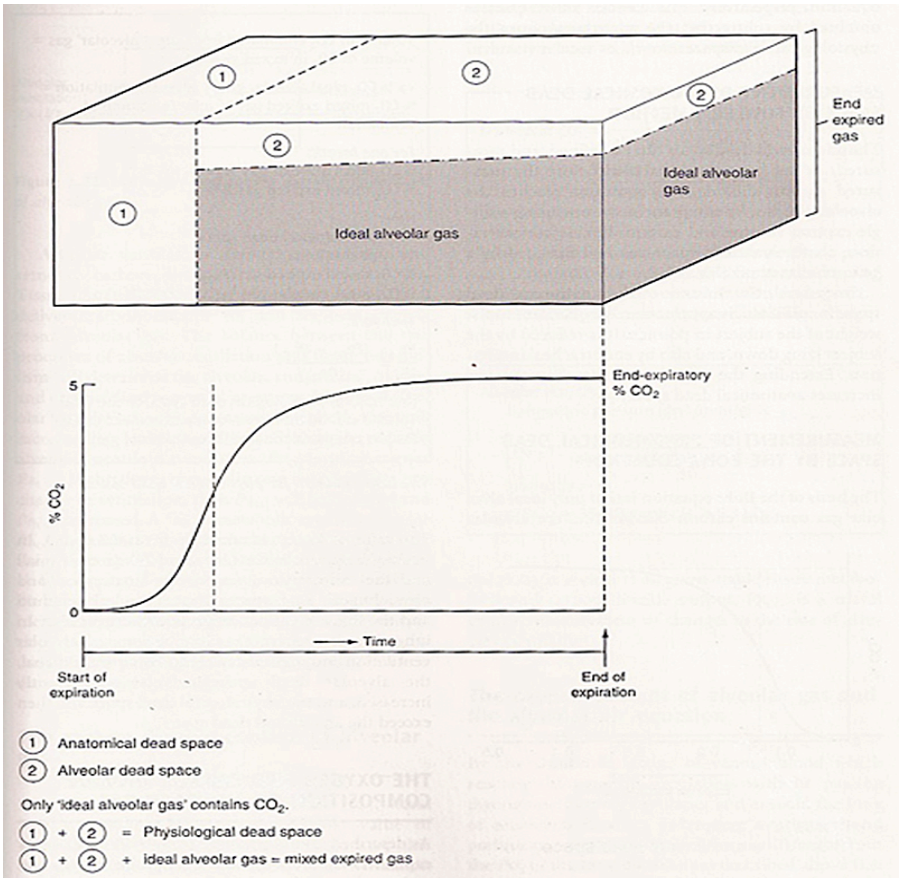
Anatomical dead space is ***decreased*** by:
i. Intubation (nasal cavity is bypassed and diameter of tube is less than airway diameter)
ii. Tracheostomy (upper airways and nasal cavity bypassed)
iii. Hyperventilation (decreasing lung volume)
iv. Neck flexion
v. Bronchoconstrictors

Alveolar Dead Space
It is ***increased*** by:
1. Lung pathologies affecting diffusion at alveolar capillary membrane like interstitial lung disease, pulmonary embolism, pulmonary edema and ARDS.
ii: General anesthesia.
iii. IPPV (Intermittent positive pressure ventilation).
iv. PEEP (Positive end expiratory pressure).
v. Hypotension.

Alveolar DS Calculations

- Index for amount of alveolar DS = D(a-E')CO2 (arterial - end expiratory PCO2 difference)
  - ↳ is possible as
    - $PaCO2 \sim P_ACO2$ in ideal alveolus – as CO2 highly diffusible
  - Normal value = ~3-5mmHg
  - ↑ed in PE
  - ↓ed in CO
- NB:
  - P_ACO2 roughly = to pulmonary end capillary blood
  - BUT see a slight ↑ PaCO2: PACO2 because of V/Q mismatch (or shunt):
    - 10% shunt ⇒ 0.7mmHg diff
    - 30% shunt ⇒ ~2mmHg

    ↳this would obviously confound the alveolar DS calculation as above



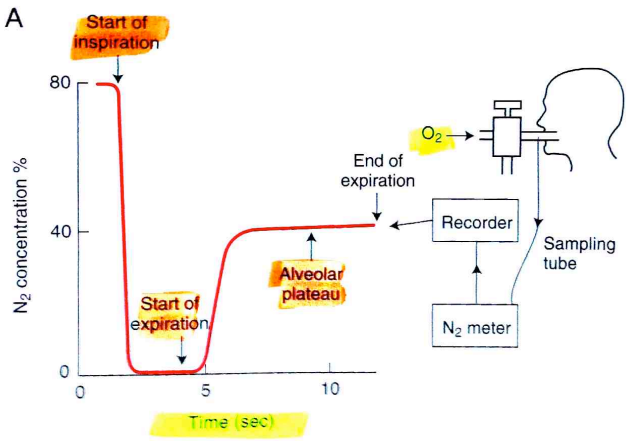
(2) + ideal alveolar gas = end expired gas

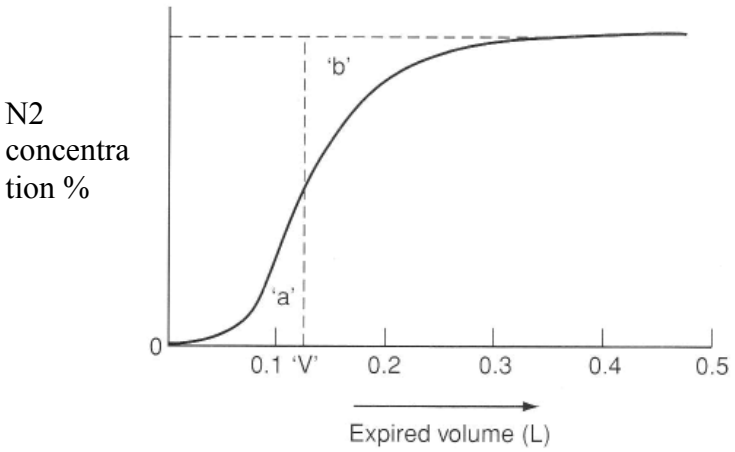
Physiological Importance of Dead Space

- essentially it causes alveolar hypoventilation
- this can lead to:
  - hypoxaemia – can usually be overcome by ↑ing Fio2 (see alveolar gas equation)
  - hypercapnia – impt as ↑PaCO2 can cause:
    - hypoxaemia – esp on RA (alveolar gas equation)
    - resp acidosis
    - ↑symp n.s. stim
    - arrhythmias – combo of ↑SNS & hypoxaemia
    - variable effects on SVR:
      - [initially] VC due to symp n.s.
      - [later] VD due to direct effect CO2
    - CNS depression – PaCo2 >100mmHg = direct anaesthetic effect
    - ↑Cerebral blood flow ⇒ ↑ICP
    - ↑RR (if spont vent) ⇒ ↑WOB

Anatomic Dead Space

- Norm ~150ml or (2.2ml/kg)
- Factors influence size:
  - large inspirations: ↑size due to pull on bronchi by surrounding lung parenchyma
  - size
  - posture
- Use **Fowlers method** to measure:
  - Take tidal breath (Vt) of 100% O2
  - A rapid nitrogen analyser is placed in circuit (can also be CO2)
  - Expired N2 is then plotted against volume exhaled (**not time**)





If area 'a' = area 'b'
Then 'V' = anatomical dead space

- Initially expire pure O2 (ie no N2) as last in, first out
- Then the N2 concentration will slowly rise as alveolar gas washes out the dead space gas
  ↳ ie 100% O2
- This continues until uniform gas concentration seen
  ↳ = alveolar plateau which represents make up of pure alveolar gas
- Alveolar plateau – close to flat plateau in norm in healthy subjects
  ↳in lung diseases may rise steeply
- The vertical line V on the graph is drawn where area a & area b are equal
  - V = volume of conducting airway up to where rapid dilution of inspired gas with alveolar gas occurs ie anatomical dead space

- Normal value of dead space ~ 2.2ml/kg (~150ml in adult)
- Factors which increase:
  - Neck extension
  - Jaw protrusion
- Factors decrease:
  - ETT
  - Erect ⇒ supine

Physiologic Dead Space

- Bohr's Method – measures volume of lung which does not eliminate CO2
- ∴ physiologic dead space

$$\frac{\text{Volume of Dead Space } (V_D)}{\text{Volume of Tidal Volume } (V_T)} = \frac{\text{Alveolar Expired CO2 } (P_ACO2) - \text{Mixed Expired PCO2 } (P_ECO_2)}{P_ACO2 \text{ (ideal alveolar PCO2)}}$$

- can be re-arranged:

$$V_D = \frac{P_ACO_2 - P_ECO_2}{P_ACO_2} \times V_T \text{ - (minus) apparatus DS}$$

- Principle:
  - All expired CO2 comes from (ideal) alveolar gas & none from dead space
  - Physiologic DS thus =
    - part of Vt that does not eliminate CO2
    - includes anatomical & alveolar DS
    - functional measurement
- An average true representation of alveolar gas sample cannot be measured DUE to regional variations in V/Q ratio's in normal lung
  - ↳∴ term 'ideal alveolar gas' is used which would exist if all of lung had same V/Q ratio (=1)
- In practise PaCo2 is used as an estimate of ideal PACO2 and substituted into equation:
  - ↳ = Enghoff modification:

$$V_D / V_T = \frac{P_aCO_2 - P_ECO_2}{P_aCO_2} \quad \text{or} \quad V_D = \frac{P_aCO_2 - P_ECO_2}{P_aCO_2} \times V_T \text{ - apparatus DS}$$

- Can create an estimate of alveolar DS with substitution into equation:

$$V_{D\text{alveolar}}/V_T = \frac{PaCO2 - P_ECO2}{PaCO2}$$

- In ICU/theatre practise we use PaCO2 - P_ECO2 difference as an index of alveolar DS

- Physiological & anatomical dead space in healthy very similar
  - ↳in lung disease: unequal blood flow & ventilation ⇒ ↑↑physiological ds

Functional Residual Capacity

Definition

- Different definitions – simpler the better:
  - = volume of gas which remains in the lungs at end of normal expiration
  - = equilibrium point between tendancy of chest wall (& diaphragm) to move out vs tendency of lungs to collapse
    - ↳ these opposing forces create –ve intrapleural pressure
- normal value = ~30ml/kg
- volume established withing 30-60mins after birth and remains same for whole life
  - ↳ what does change (esp neonates & elderly) is closing volume
    - ↳ ↑closing capacity ⇒ ↑closing volume ⇒ ↑shunting
      - ↳ no change in FRC
- capacity ∴ = RV + ERV

Effects on FRC

- Major factors :
  - **Height**
  - **Weight**
  - **Position**
  - **Disease**
  - **Muscle relaxation** (ie anaesthesia +/- muscle relaxants)

- Increase in FRC:
  - ↑ Height
  - Supine → erect (largest change is from 0 → 60 degrees)
  - ↓ in lung elastic recoil (emphysema) ie ↑ compliance
  - PEEP
  - Males (10% ↑)

- Decrease in FRC:
  - Obesity
  - Muscle paralysis (loss of diaphragmatic end expir tone)
  - Erect → supine
  - Disease causing ↑ elastic recoil of lungs
  - Pregnancy
  - Anaesthesia (supine , ↓ muscle tone)

Functions of FRC:

- Oxygen store
- Buffer to maintain steady PaO2 (esp during expiration)
- Prevention of atelectasis
- ↓ Work of breathing (keep lungs on steep part of compliance curve)
- Keeps Pulmonary vascular resistance (PVR) at a *minimum*.
- ↓ V/Q mismatch
- Keeps airways resistance low (not at minimum though)
  - ↳ Below FRC, airways resistance increases dramatically

Oxygen Store

- Norm lungs contain ~290ml O2 in adult
- With denitrogenation of lungs (ie preoxygenation ~3min of 3-4VC breaths) ⇒ O2 store ↑1800ml
- ↑time to desaturation of 7-8mins
- Best way to measure effectiveness of preoxygenation is measure ET O2 fraction (FEO2)

- FEO2 ≈ FAO2 (alveolar o2 fraction)
- Typical FRC volume = 2.2 litres which normally contains 21% O2 = 462mls O2
- In norm adult with complete preoxygenation (FAO2 >0.9) lungs should contain around 2000ml O2
- Total body oxygen consumption ≈ 250mls/min
- ∴ apnoea with norm store takes around 1-2min (462/250)
- If FRC preoxygenated to FAO2 0.9:
  - 2200*0.9 = 1980mls
  - 1980/250 = 7.92mins

NB the importance of 100% O2 pre-ox is ↑O2 store (only slight ↑CaO2 & Spo2 seen)

Minimizing PVR

- PVR varies with lung volume – it is high at large and small lung volumes
- Minimum value for PVR = at FRC:
  - Above FRC: ↑PVR due to stretching of pulmon capillaries (alveolar vessels)
  - Below FRC: ↑PVR due to ↓caliber of extra-alveolar vessels
    ↳ at larger lung volumes these vessels stretched opne by elastic fibres in lung parenchyma
- Alveolar & extra-alveolar vessels are in series
- PVR = Pulmon capillaries contribute 50-60% of total PVR
- (SVR = arterioles contribute ~80%)

Closing Volume/Capacity

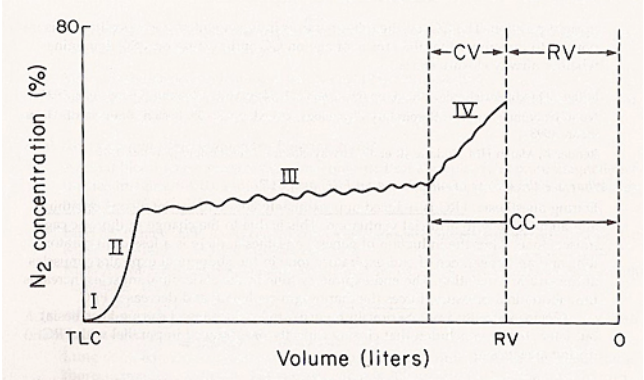
- closing capacity (CC) = the residual lung volume at which small airways in the dependant parts of the lungs **start** closing during expiration
- closing volume (CV) = lung volume from beginning of airway closure to the end of max expiration
  ↳ ie the portion of VC which can (with max effort) be exhaled after the **onset** of airways closure

  ↳ ∴ CV = CC – RV

  normal = ~7ml/kg or 10% of vital capacity

Measurement

- single N2 breath test
- most common = N2 breath test:
  (↳ other techniques eg bolus technique with inert tracer gas like Helium/Xenon/Argon)
  - comparable to Fowlers' method for measurement of anatomical DS:
    - full expiration (from RV)
    - vital capacity breath of 100% O2
    - expired N2 measured & plotted against volume



- III⇒IV interface: sudden ↑N2 = early airway closure
- Phase IV ends at max expiration ie RV

  ↳ ∴ phase IV = CV

- To find CC then need to measure RV: see on next pages

Reason for ↑N2 at Start of Small airway closure

- Starting at RV just before single breath of 100% (prior to beginning of test):
  - N2 conc is nearly uniform through lung – (slight incr in N2 at base)
    - Basal alveoli start out much smaller than apical alveoli
    - @ end of VC inspiration all alveoli = same size
      ↳ basal alveoli have ↑ed size > apical ones ∴ basal N2 must be more diluted
    - subsequent expiration: upper & lower zones initially empty together ⇒ expired N2 constant
    - dependant airways begin to close
    - higher conc N2 in upper zones contribute more to expired gas ⇒ sudden rise (phase 4)
- this test only good if have small amount of small airways disease
  ↳too much disease and trace distorts so much cannot identify CV

Measurement of FRC or RV

- Can use nitrogen washout method:
  - FRC – start breathing 100% O2 at end of normal end expiration
  - RV – start breathing 100% O2 at end full expiration
- Total volume of expired gas over several minutes is then analysed to determine N2 content as it is washed out
  ↳content = concentration x volume
- This N2 could only have come from FRC or RV (depending on which phase of expiration started at)
- Once content known can then calculate volume **because** we know original conc of N2 in the FRC (RV) in room air (ie 79%)

Ie concentration = amount/volume

Or volume (FRC) = amount/concentration

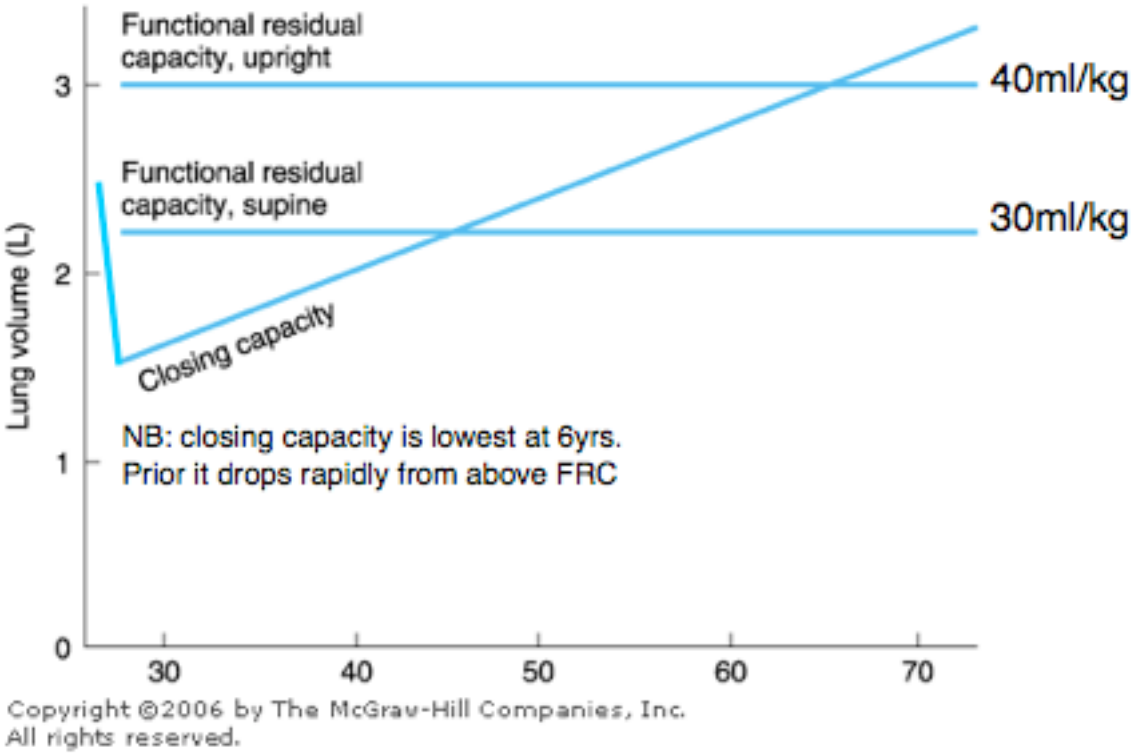
- As with helium dilution method – doe not measure gas trapped behind closed airways

Relationship b/w FRC, CC & V/Q Matching

- If CC > FRC ≈ dependant airways will start to close during norm tidal breathing
  ↳ ∴ ↑ing V/Q mismatching (ratio <1) will occur ⇒ ↓PaO2
- Balance between sizes of CC & FRC:
  - ↑CC
  - ↓FRC
  - or both together

  ↳ will lead to CC >FRC
- factors effecting FRC prev discussed
- factors ↑ing CC:
  - extremes of age ie neonates and elderly
    - via changes in intrapleural pressure ie less –ve (↑ing positive):
      - neonates – floppy chest which collapses in
      - elderly - ↓ed elasticity of lung parenchyma
  - disease: emphysema/asthma – air trapping
  - smoking
- CC = FRC in varying positions ie both ↑with age:

- 44yr in supine
- 66yr in erect



Age…..