Lung function

Always consider all the parts separately and together

Control of breathing

Mechanics of breathing

Airways both large and small

Gas transfer

Transfer of oxygen to tissues (consider iron deficiency in LVF?COPD)

The end organs that we use to move

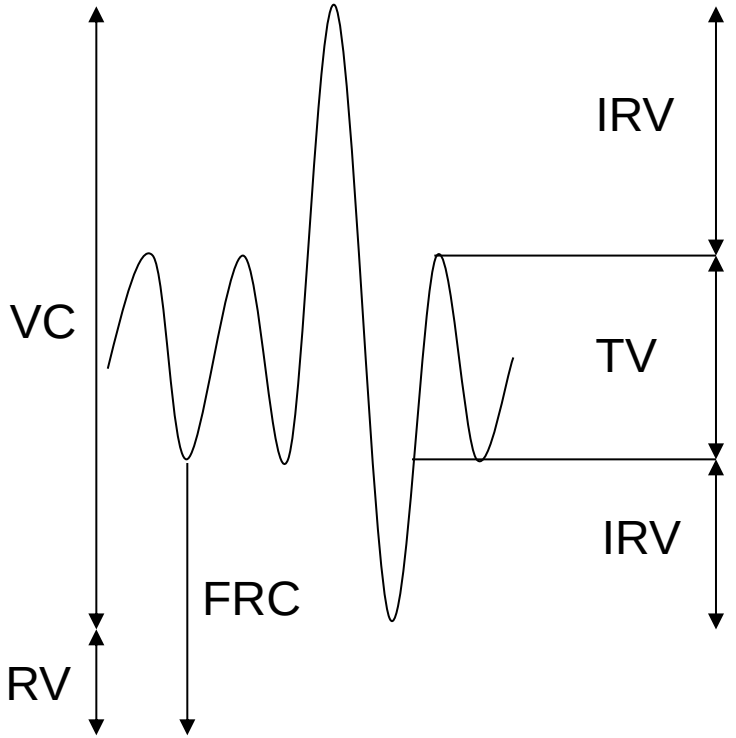
How we perceive the load on our breathing

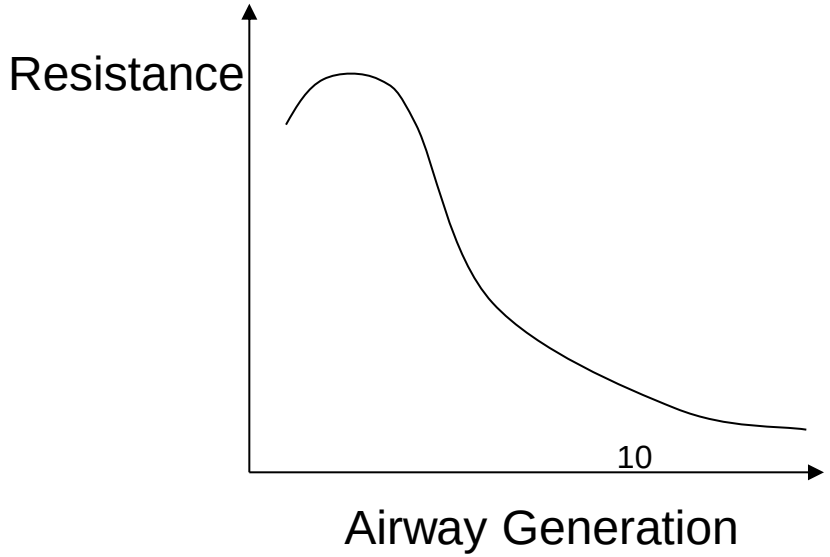
LIMITATIONS OF TESTING

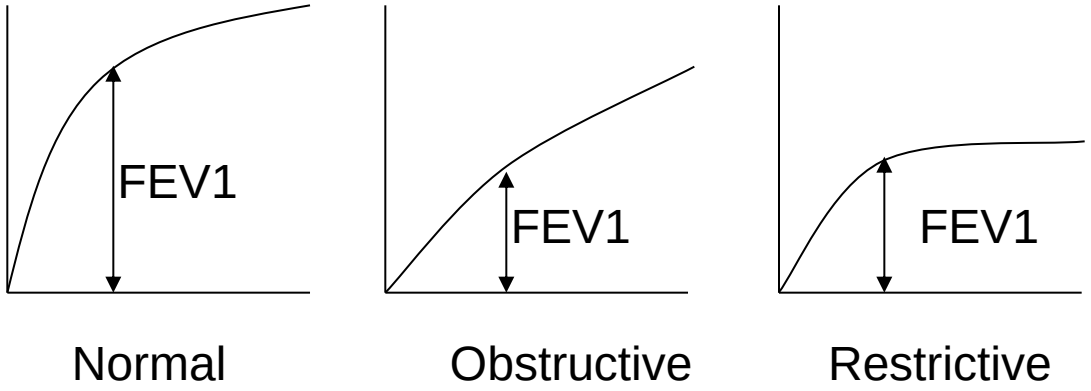
- Need to understand the orders
- Need to breathe out from TLC
- Maximum effort
- Mouthpiece leak
- Ability to carry out orders (IPD/CVA)

Airways

1. Laryngeal dysfunction presents as “asthma” with difficulty breathing in and normal lung function, it may only appear on exertion so may need laryngoscopy and exercise to prove it.
2. Some patients have upper airway obstruction due to fixed compression and it shows on flow volume loop, but a few have tracheal collapse so flows are reduced after FRC on breathing out
3. Current tests require comprehension and ability to breathe to order, so if you cannot do so then forced oscillation gives useful data


IRV
VC
TV
IRV
FRC
RV


Resistance
10
Airway Generation


FEV1
FEV1
FEV1
Normal
Obstructive
Restrictive

Obstructive defect

- The volume in 1 second is reduced more than the vital capacity
- FEV1/VC is <75%
- Occurs with obstruction at any level
- Typical in asthma, COPD and upper Airway obstruction

Restrictive defect

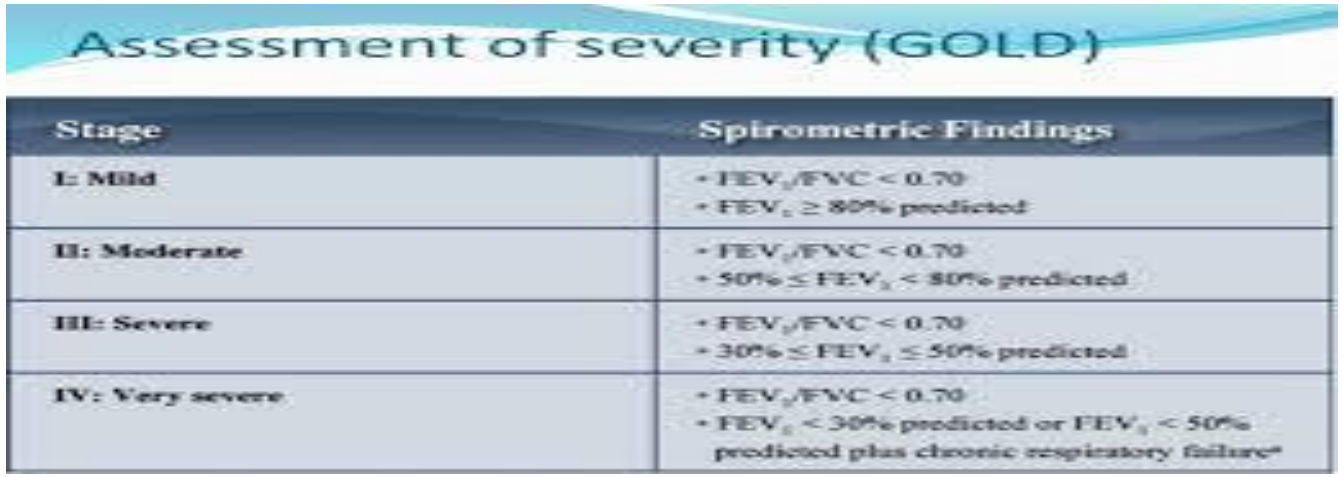
- The FEV 1 is reduced no more than the VC
- FEV1/VC ratio is >75% and may be 100%
- Vital capacity is reduced
- Typical with restrictive lung disease, lung fibrosis, reduced chest wall movement and muscle disease

Definition of COPD

Chronic obstructive pulmonary disease shows an obstructive defect which does not improve with bronchodilators

FEV1 <80%

FV1/VC <70%



Peak Flow Measurement

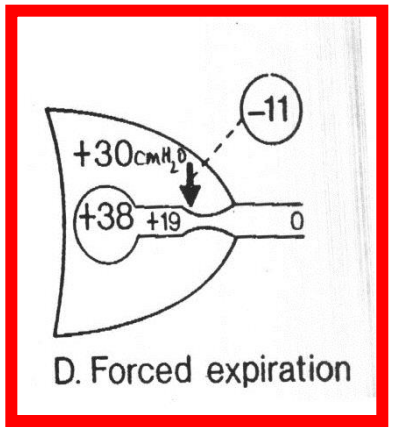
- Only valid if done at TLC and with maximum effort
- Easy to use repeatedly and follow change
- Cheap machines for patient use
- Typically reduced in large airway obstruction , upper airway obstruction or asthma
- Less affected in COPD and small airways disease

Peak Flow over Time

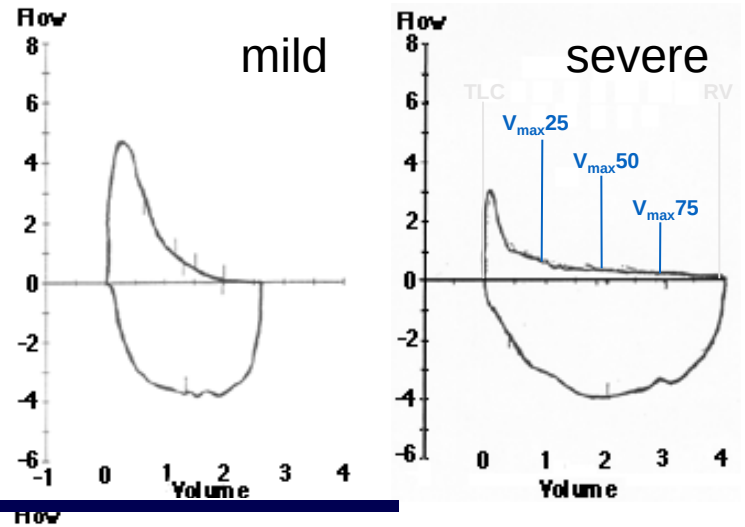
- Peak flow is lowest in the morning
- Saw tooth pattern in asthma
- Pattern of peak flow after challenge or contact with irritant or allergen may indicate cause of change
- >20% diurnal variation for .3days /week for 2 weeks on PEFR diary = asthma
- Peak Flow may increase with inspiratory muscle training

Response to BronchoDilators

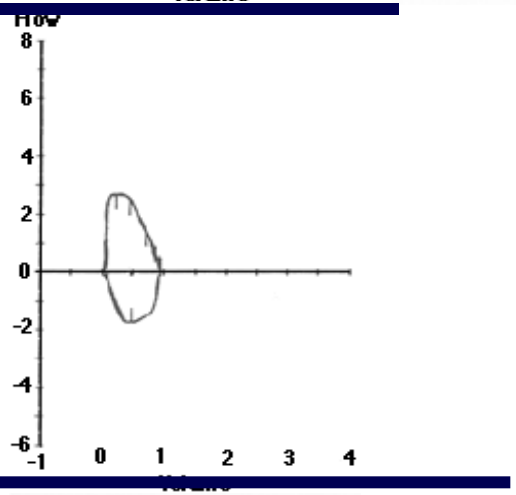
- Increase in FEV1 or PEFR in response to bronchodilators suggests reversibility of obstruction
- FEV1 >15% and 200ml after 400microgram salbutamol by spacer
- or 2.5mg via nebuliser,
- or 30mg/day of steroids for 14 days



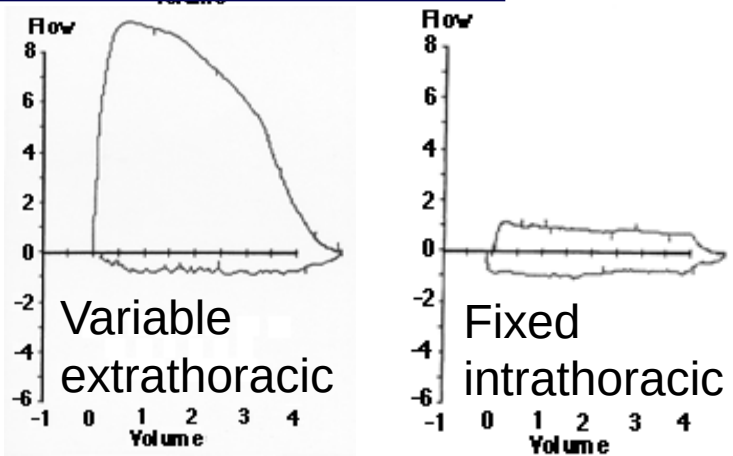
Small airways disease
e.g. COPD



Restrictive
Lung disease



Airways
Obstruction



Large airways or small airways

- Spirometry and peak flow and flow volume loops are useful for large and moderate sized airways
- Beyond the 11the generation of airway there is only diffusion and no bulk flow
- In between you need to look with nitrogen washout or radioactive markers such as SF6

Dynamic Hyperinflation

- There is always static hyperinflation in patients with COPD but it gets worse on breathing more
- On exertion there is air trapping as airways collapse and the pressure of air in the emphysaematous areas increases
- Breathing pattern changes and there is gradually less and less inspiratory reserve
- Once inspiratory reserve = <500ml there is increased breathlessness

What happens with CPAP and NIV

- In babies the main problem is airways which are collapsible, so applying CPAP reduces collapse, and atelectasis and does not need intubation which is technically difficult with a 3-4mm trachea
- In adults with COPD and airways which close early CPAP might increase the time of opening of the airways including the larynx
- But the more CPAP you deliver then the more difficult to exhale and an increase in transthoracic pressure and air trapping
- Increasing CPAP to 15 cmH2O makes inspiration passive and expiration and active process of the abdominal muscles. NIV reduces work of breathing

Heliox

- Gas has density and viscosity
- Helium is very light but viscous so flows smoothly more so than nitrogen
- Heliox flows through upper airway obstruction and lower airway collapse better than Nitrogen and oxygen
- But your voice sounds odd

Anatomical Dead space

Ventilation includes the airways where there is no gas transfer and the alveolae where gas transfer occurs

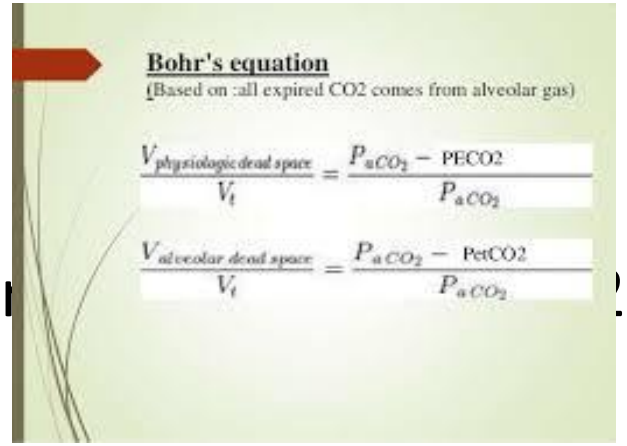
Alveolar ventilation = tidal volume – anatomical dead space

This is usually 1ml/lb of body weight

Alveolar ventilation (VA)=VT (tidal volume – dead space) x frequency

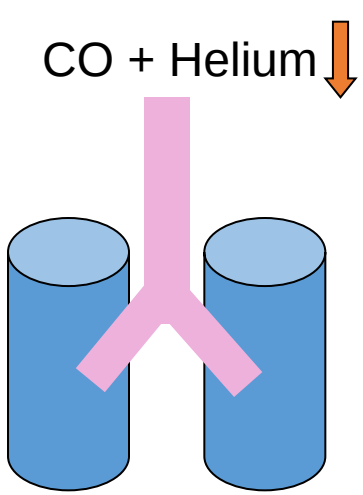
Physiological Dead space

- Physiological dead space describes the area of the lungs which is not involved in gas transfer, both the anatomical dead space and those areas of lung which are ventilated and not perfused.
- It can be calculated using the Bohr equation

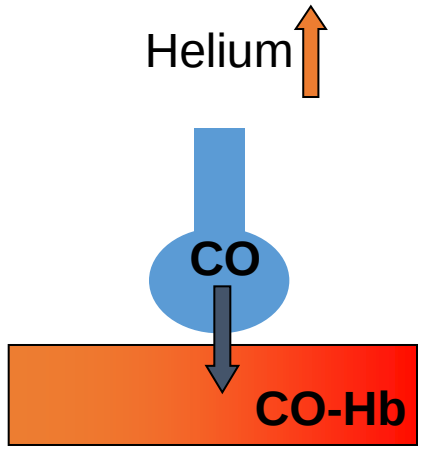


- Where PaCO2 = arter = expired CO2 pressure

Gas Transfer



TLCO = KCO x Va



Decreased TLCO:
Decreased Perfusion
Decreased Ventilation
V/Q mismatch
Anaemia

Increased TLCO:
Increased Cardiac Output
Polycythaemia
Alveolar Haemorrhage

Fick Principle formula

$$V_{gas} = \frac{A}{T} \times D \times (P_1 - P_2)$$

Where V = volume of gas that diffuses/sec

A = area of the sheet

T = thickness of the sheet

D = coefficient of diffusion

P2 and P1 are the pressure on each side of the sheet

carbon monoxide is used as the gas for diffusion

Ficks's Principle and Disease

Gas transfer is reduced with

reduced surface area, in pneumonectomy, lobectomy or reduced ventilation from airway obstruction or reduced effective area with emphysaema or increased dead space

increased thickness of membrane with pulmonary fibrosis, alveolar proteinosis, and acute lung injury

reduced oxygen concentration as in high altitude

inadequate time for gas transfer on exercise if

there is lung disease

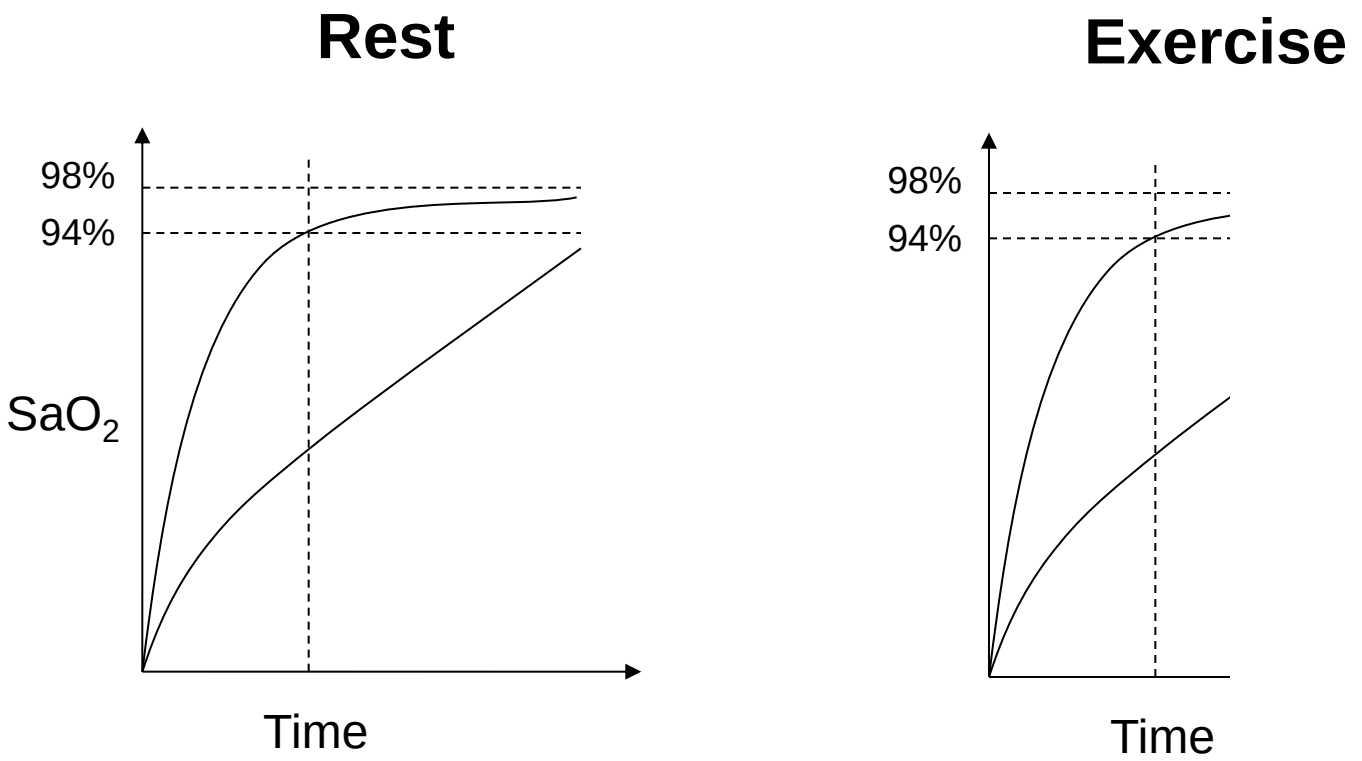
Gas transfer

- Gas transfer depends on the area of alveolar membrane, the thickness of the membrane, and the partial pressure of a gas on each side.
- Measured with a single 10 second breath hold of a mixture of oxygen, helium and carbon monoxide,
- BUT, although transfer is important generally it is the locality in the lung which determines its importance
- Lower zone emphysaema or fibrosis or thromboembolism can leave very low gas transfer
- If you cannot d the standard test you can do a rebreathing method to measure DLCO and KCO

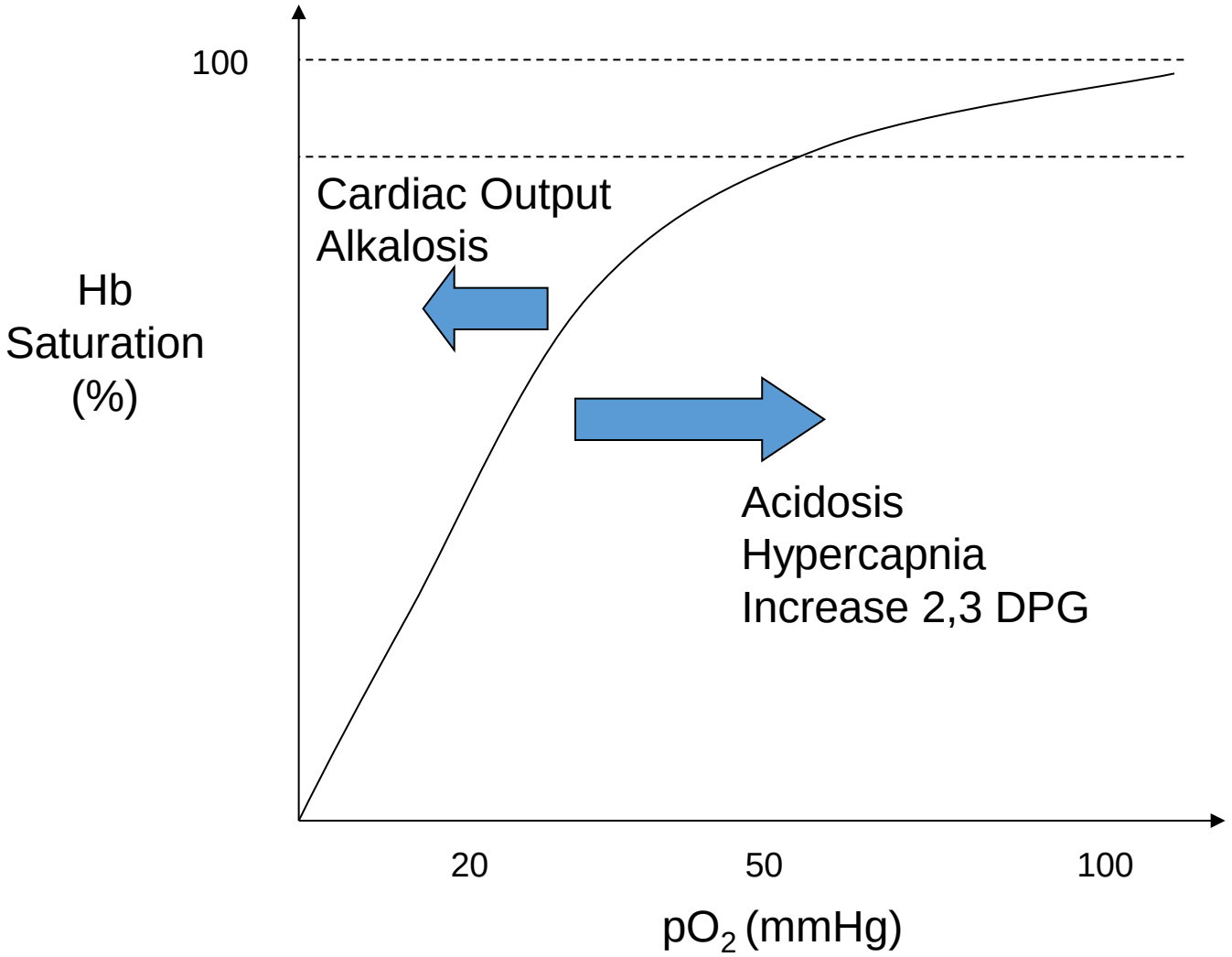
Gas transfer for oxygen

- You can measure gas transfer by measuring A-a gradient, knowing how much goes in and how much gets in.
- As CO2 transfer is easier than for oxygen, PO2 tends to fall but CO2 is cleared
- At rest 250ml oxygen required per minute, so oxygen saturation m ay be Ok even with poor gas transfer, and there is enough time for the oxygen to transfer as well
- But with exercise the amount of oxygen required by the body increases, and there may not be adequate time for gas transfer with a low SaO2.

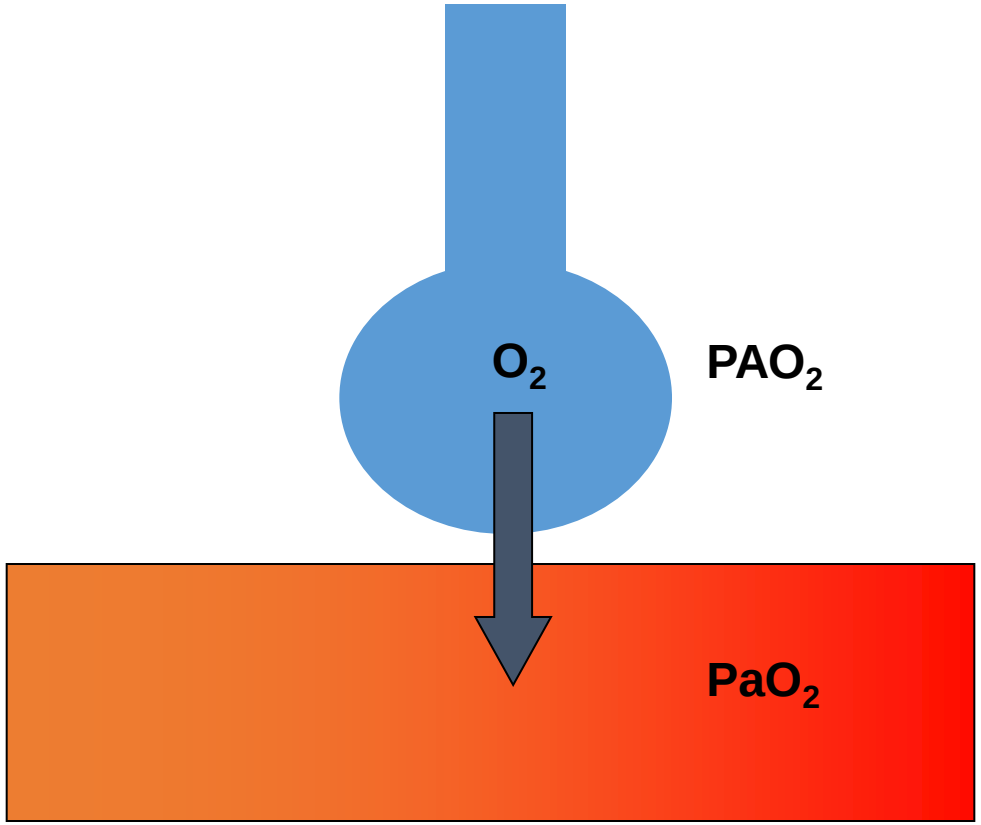
Exercise De-saturation



Oxygen Dissociation Curve



Alveolar-arterial (A-a) Gradient



$$PAO_2 = FiO_2 (P_{atm} - P_{H20}) - PaCO_2/R$$

Measurement of lung volumes

- The circuit allows for addition of oxygen at 0.2 litres/min and absorption of CO2.
- The volume for functional residual capacity is calculated by comparing the concentration of helium before rebreathing with that after stabilisation.
- **FRC = V (He1-He2/He2) where V is the volume of added Helium, He1 and He2 are concentrations of helium at beginning and end**
- This can be corrected for temperature and pressure.
- From the spirometric trace TLC, IRV, ERV, and RV can be calculated.

Lung volumes

- Standard measure is by helium dilution , but this under measures if there is air trapped with poor ventilation
- Helium dilution is not that useful for measuring residual volume when lung reduction surgery is being considered
- RV may be under measured by up to 25-50%
- Use the body box instead

AIRWAY RESISTANCE AND CONDUCTANCE

- Airway resistance is defined as the pressure difference between the alveolae and the mouth.
- This can be measured in a body plethysmograph or by direct measurement of intrathoracic oesophageal pressures
- Conductance is the reciprocal of resistance

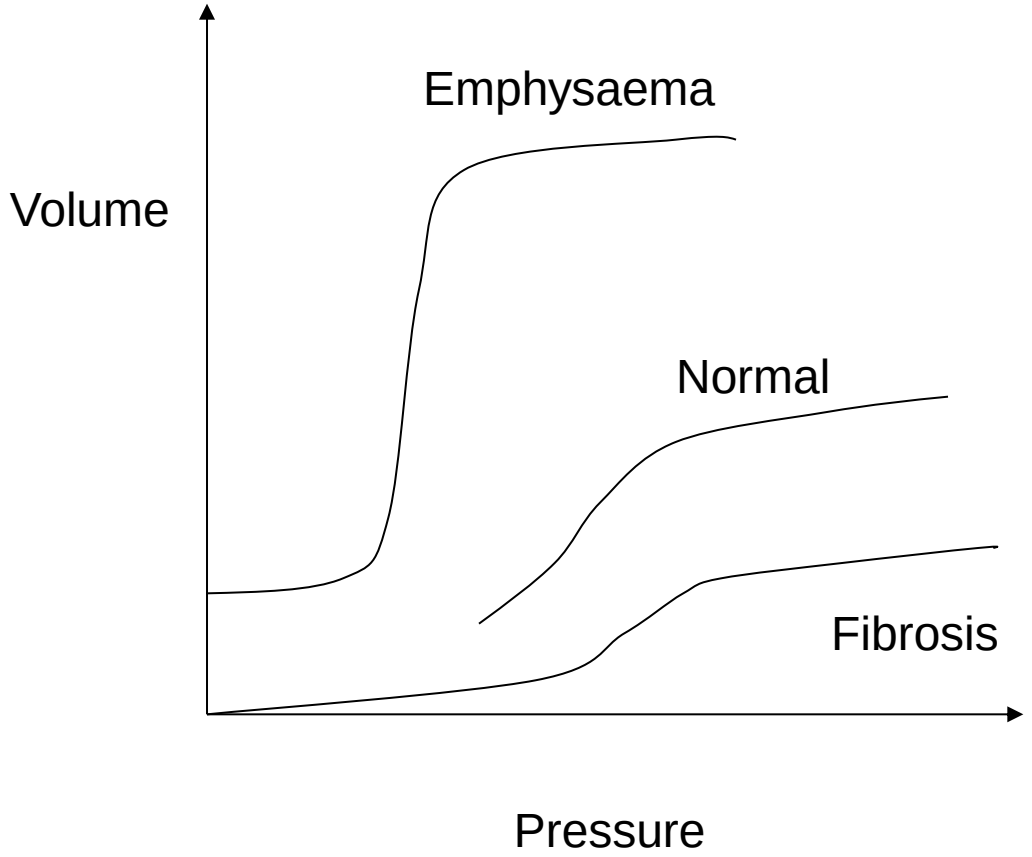
Airway resistance 2

- Most resistance to air flow is in the first 5 generations of airways
- Beyond the 11^{th} generation there is diffusion not bulk flow
- Increasing gas viscosity improves laminar flow and allows better flow (Heliox)

ELASTIC RECOIL AND COMPLIANCE

- Lung compliance is defined as the volume change per unit of pressure
- Depends on alveolar surface tension and distensibility of lung tissue
- Is increased in emphysaema with decreased elastic recoil
- Is decreased in lung fibrosis with increased elastic recoil
- Chest wall compliance is reduced by deformities of the thorax

Lung Compliance



$$\text{Compliance} = \Delta V/\Delta P$$

Control of breathing

- Airways need to be kept open and collapse of upper airways with sedation leads to sleep apnoea
- Poor cerebral function or perfusion leads to Cheyne Stokes respiration and central apnoea
- The time constant for the different parts of breathing control are all different
- PO2 is fastest , via carotid body reset sensitivity no hypoxia
- PCO2 is next via central PCO2 levelsand a bit via carotid body
- pH is slowest via central H+ receptors, and take a long time to wear off
- BUT you increase ventilation even before you start walking

Control of breathing: hierarchy of control

- Information from muscles, tendons, joints
- Lung receptors, chest wall,
- Information of intent to exercise
- Automatic breathing control in the brainstem
- Voluntary control
- All fit together to give the final effect
- There is a wide range of CO2 drive levels, asthmatics who develop type 2 respiratory failure have low CO2 response even when well

(Read 1971)

Breathlessness and control of breathing

- Breathlessness comes from lung receptors and chest wall muscle spindles, and information reaches subcortical areas of the brain shared with pain perception and fear
- We can improve breathlessness by teaching breathing control, slow breathing, not panting, pacing of exertion, and removal of the fear which comes with breathlessness
- This is an intrinsic part of pulmonary rehabilitation
- Confidence comes from knowing you can cope with breathlessness
- Panting cools airways and leads to bronchospasm , reversed by anti-muscarinic agents as in asthma
- Pursed lip breathing gives 2-5cm of PEEP

Muscles

- Muscles are abnormal in patients with COPD, with abnormal mitochondrial function and build up of products of oxidation, and with fewer less efficient mitochondria
- A week of ventilation means a loss of up to 1/3 of diaphragm function
- Many patients are overloaded with weight (30% BMI >30 in Basildon area) or have no muscles (up to 10% BMI <18)
- If you do not exert you lose muscle and get deconditioned
- Inspiratory muscle training probably does not work
- Flat diaphragms are less efficient than curved ones so severe air trapping gives reduced inspiratory pressures

US exams

- Anatomical and physiological dead space caclulations,
- Shunt
- Alveolar ventilation
- Calculation of alveolar pressure PAO2
- Airway resistance
- Bohr equation
- PVR
- A-a gradient
- BUT these are not required for the UK
- **John West: Pulmonary Physiology and Pathophysiology ----has the answers you would need otherwise use**
- **Peter Gibson Clinical Tests of Respiratory Function or for Anaesthetics Nunn Applied Respiratory Physiology**