CHEAT SHEETS FOR VENTILATION

- Ventilators create the drive for inspiration
-Expiration is passive
-When picturing ventilation think of trying to inflate a balloon with a long straw
  - To get gas through the straw (The ETT and the conducting airways) to the balloon (alveolus) needs a Pressure difference from the atmosphere to the alveolus
  - This pressure difference in spont breathing patients (you and me) is a negative force (i.e the alveolus has a relative negative pressure to the alveolus created by chest wall expansion)
  - In ventilation we need positive pressure (hence the term positive pressure ventilation) to inflate the alveolus

Modes
- There are lots of variables we need to consider when setting up a ventilator
  - Resp rate
  - Pressure
  - Volume
  - PEEP
  - Flow
  - Inspiratory time/ inspiratory: Expiratory time
- The main choices or variables when we set the ventilator is whethe we want to control the volume delivered to the lungs (volume control) or the pressure delivered to the lungs
  - If we prescribe a volume mode the ventilator will adjust the pressure required to deliver that volume as the patients resistance and compliance changes
If we prescribe a pressure mode, the pressure remains constant and the resulting volume delivered will change with the patient's compliance and resistance.

This relationship between pressure/volume is described in a formula later.

- The trigger for breathing can either be mandatory (control) or patient triggered
  - Volume modes
    - Control
      - ACV/CMV
        - Assist control or control mandatory ventilation
          - ACV will still allow the patient to breath but the way the breath is delivered is the same whether the machine or patient triggers the breath
      - SIMV
        - Where the patients desire to breath is more synchronized with the ventilator
        - Will still give mandatory breaths if needed
        - IF the patient wants to breath it will generally act as pressure support, unless it is nearly time for a mandatory breath in which case it will appear the same as a machine initiated breath
    - Spontaneous
      - PSV
  - Pressure modes
    - Control
      - PCV (pressure control ventilation)
      - PC-SIMV (same as SIMV but pressure the controlled variable)
    - Spontaneous
      - PSV
o Pressure support ventilation

The waveforms that appear on the ventilator interface differ with volume vs pressure modes

- The main waveforms we use are
  - Pressure vs time
  - Flow vs time
  - Volume vs time
- Time is on the x axis

For example in **Pressure control modes**

![Pressure control modes diagram](image)
- Tying it together
  - Pressure vs time
    - Note the waveform has a square waveform
      - The pressure has been set, and the ventilator aims to keep this constant during the patient's inspiration
  - Flow vs time
    - This is called a decelerating flow pattern
      - In early inspiration there is a rapid increase in flow to reach the preset pressure
      - Flow then peters off to keep that pressure constant
  - Volume vs time
    - The resulting volume varies as the patient’s compliance and resistance changes
    - Again this is explained below

- The purported advantage of this mode is that by keeping the pressure constant, you allow a better equilibration of oxygen throughout all the alveoli hence improving oxygenation
  - However in multiple studies in hypoxia it does not matter whether you set pressure or volume as the controlled variable

- It also arguably lowers the peak pressure delivered (but has a higher mean airway pressure (which improves oxygenation)
  - Will be explained in a different handout on PEEP and oxygenation
In volume control modes

- Pressure vs time
  - Note the pressure gradually climbs as the volume is delivered
  - The pressure required to increase the volume changes with the patient’s characteristics
- Flow vs time
  - This is a square waveform
In other words the flow of gas is kept constant until the volume is delivered.
  - Remember flow x time will equal the volume delivered

Often in volume control we can set an inspiratory pause. This is where flow stops for a brief period before exhalation. The idea is to allow better equilibration of gas through the alveoli. The pressure vs time waveforms will subsequently look like this

- Note the will also be a pause in the flow vs time waveform
- Our default mode at Nepean ICU is SIMV (Volume)
  - However if you look at our waveforms they appear more like a pressure control mode
  - That is because we actually set our ventilators to have SIMV with autoflow
    - Basically makes this mode a volume guaranteed pressure control SIMV
  - Gives us the advantage of
    - Lower peak pressures but higher mean pressures
    - Better oxygenation/ distribution of gas
    - But guaranteeing a set volume!
THE DREADED PRESSURE ALARM

- An important alarm to keep an eye on in volume control modes is the airway pressure. Higher pressures are a significant risk to the patient. In some ventilators if the pressure is deemed unacceptable the breath may be held or dumped (and not reach the desired volume)

- The amount of pressure needed to shift gas to the alveolus can be summed up as

Pairway (Peak Pressure) = Flow x Resistance + Volume/ Compliance + PEEP

- We can use this formula to actually figure out what the hell is causing the high airway pressures
- Flow x Resistance is the Pressure component needed to get gas past the airways
  - If the airways are bronchospastic (narrowed) or the ETT is obstructed or kinked this pressure will increase
- Because the resistance will increase to the power of 4 (remember Poiseuille's law)

- The pressure that is needed to blow up the balloon (alveolus) is Volume/Compliance
  - Compliance is the change in volume for a change in pressure

- IF the alveolus becomes stiffer (APO, pneumonia, ARDS) then clearly it is harder to inflate (less compliant) and will require a higher pressure to achieve a similar volume
- The compliance of the alveolus also varies with how open the alveolus is to begin with
  - Remember that the hardest part of blowing up a balloon is the beginning (trying to open it up) and the last bit (when it is over distended and the elastic is stretched)
• Hence Compliance varies with the volume of the alveoli
• This is one of the advantages of using PEEP – improving compliance and ultimate work of breathing (of the patient or the ventilator)

- We need to closely monitor ventilator pressure
  o Risk of barotrauma
  o Sign of deterioration
  o Hence watch the pressure vs time curve

- If the pressure increases it can be due to patient factors
  o Increased resistance
    ▪ Kinked tube
    ▪ Bronchospasm
    ▪ Obstructed tube
  o Decreased compliance
    ▪ Pulmonary
      • Endobronchial migration of ETT
      • APO
      • Pneumonia
      • Atelectasis
    ▪ Pleural
      • Effusion
      • Empyema
      • Pneumothorax
    ▪ Abdominal
      • Ascites etc
- Chest wall
  - Kyphoscoliosis
  - SC emphysema
- Can also be due to
  - Ventilator problems
    - Condensation in tubing, kinked ventilator tubing
    - Inappropriate ventilator settings (excessive PEEP, volume or inappropriate flow)

- How do you tell what has caused the rise in Peak pressure?
  - Look at the God Damn Patient
    - Disconnect from the ventilator and place on 100% O2 via Bag
      - If easy to bag then was probably ventilator
      - If no improvement then move on
    - Check the Tube for kinks
    - Suction the tube
    - Ensure tube has not migrated
    - Palpate trachea
    - Auscultate chest
      - Bronchospasm → COPD/Asthma or cardiogenic wheeze (APO)
      - Creps
      - Absence of Air entry
        - Endobronchial tube
        - Pneumothorax
    - Examine abdomen if needed

- Look at the ventilator waveforms- takes practice and knowing what is normal.
  - Increased resistance
Pressure vs time curve

- The Plateau pressure is low relative to the Peak pressure.
- The plateau pressure is considered to be the alveolar pressure, and can be measured when there is a pause in inspiration (i.e., there is no flow $\rightarrow 0 \times$ resistance $= 0$!!)

ETCO2 trace - if resistance increases, it is more difficult to exhale, hence it takes longer for the ETCO2 to reach a plateau.

Flow vs time curve

- You may see evidence that expiration does not completely finish before the next breath happens.
  - This is evidence of gas trapping.

Example of increased airway resistance.
• Decreased compliance
  o Plateau pressure is increased
  o The flow vs time curve looks sharper
    (steeper incline and a rapid decline) when using PCV
      ▪ This is because the flow required to reach the set pressure is less.

  o Note if the question was hypoxia, you can do a similar approach.
  However if pressure hasn’t changed (i.e. compliance or resistance is not a problem, then you need to consider causes of increased dead space.... See below

  ▪ Get some history/ context
• What was the time course of the rise (sudden vs gradual)
• Any recent changes to ventilator settings
• Any recent attempts at CVC etc for pneumothorax
HYPOXIA

- There are different ways of classifying causes of hypoxia
  o By system
  o Time course
  o Physiology

Time course is the best for helping at MET calls for SOB and hypoxia. It automatically narrows the field for you, and you can start treating and planning disposition.

- Sudden onset
  o APO (Particularly what is referred to as SCAPE – sympathetic crashing acute pulmonary edema- bloody Americans.)
  o PE
  o Pneumothorax
  o Aspiration
  o +/- Anaphylaxis

- Over Hours
  o Asthma
  o Pneumonia
  o Pulmonary oedema- non cardiogenic and cardiogenic
  o Metabolic acidosis- something that is commonly missed (shouldn’t cause hypoxia but does cause tachypnea)

- Days to weeks
  o Pleural effusion
  o Exacerbation of COPD
  o Pneumonia
- Anaemia - again will not cause low saturations (because we are measuring the % of Hb that is saturated with O2!) but will cause SOB

Other things to consider
- Don't forget that low cardiac output state (including due to hypovolaemia) will give a low sats trace.
- SOB can be caused by poor abdominal compliance – ascites, haemoperitoneum, pancreatitis, SBO etc.

Physiological classification of Hypoxia –

- It is sometimes useful to use this model when considering the hypoxic patient on a ventilator.
  - Low inspired concentration of oxygen (altitude- not in Nepean!)
  - Hypoventilation
  - VQ mismatch
    - Shunt
    - Dead space
  - Diffusion abnormality

Hypoventilation
- Hypercapnoea and hypoxia
- Do not assume that just that there is hypercapnoea present that there is not a second process. This is where we need our alveolar gas equation to make sure we are not missing a shunt, dead space or diffusion abnormality

Alveolar gas equation

\[ PAO2 (\text{Note capital A - Alveolar, not arterial}) = FiO2 \times (\text{Barometric pressure – Saturated water vapour pressure}) - PaCO2/0.8 \]

- 0.8 refers to the average respiratory quotient for the body
- Barometric pressure 760mmHg at sea level
  o We are converting the inspired fraction of oxygen in air to its relative partial pressure
  o Need to consider that part of the vapour we breath is saturated with water
- If there is a large difference between the Alveolar partial Pressure of oxygen and the arterial partial pressure, then there must be something stopping oxygen crossing the alveolar-capillary membrane.
  o i.e. shunt, dead space or diffusion
  o There is always a normal difference between PAO2 and PaO2 but the normal range varies with age.

**VQ Mismatch**
- Error with either ventilation or blood flow.
- There is a normal VQ mismatch
  o When standing
    ▪ Ventilation increases as you go from the apex to the base
    ▪ However blood flow increases more from the apex to the base
    ▪ Hence V/Q falls from the apex to the base

1) Shunt
  a. Commonest cause of hypoxia
  b. Where there is blood flow past the alveolus, but the alveolus itself is not ventilated as it is clogged up with fluid or pus etc
  c. Examples
    i. APO
    ii. Pneumonia
    iii. Atelectasis
    iv. Sputum plugging
  d. The blood perfusing these alveoli will not be oxygenated
  e. CO2 will not climb that much due to compensatory tachypnea.
f. Oxygen does not relieve the hypoxia completely – as the alveoli that are ventilating normally cannot saturate the blood any further

g. However in time hypoxic pulmonary vasoconstriction occurs, and more blood is preferentially diverted to the normal alveoli

h. These are the guys that need PEEP to re open the alveoli!

i. May exhibit worsening compliance on the ventilator with resulting higher Peak and Plateau Pressures as above

2) Dead Space

a. Dead space refers to alveoli that are ventilated but not perfused.

b. Examples

i. Massive PE

ii. Hypovolaemia

iii. Cardiogenic shock

c. Excessive PEEP can be detrimental here

i. If you over expand the alveolus, it will impede the flow of blood in the capillaries, in effect worsening the shunt

d. These patients have a large difference between their Alveolar Expired CO2 (ETCO2) and the arterial CO2

i. If the alveolus does not have blood flow, then the PCO2 in that alveolus will be 0

ii. When exhaled, and mixed with other alveolar air, the total ETCO2 will be reduced

e. Note the compliance does not change with these patients, so the peak and plateau pressure will not have increased
Diffusion Abnormality

- Thickening of the alvelo-capillary membrane reduces the ease with which gas diffuses
  - Fick’s law of diffusion states in part that diffusion is inverse to the thickness of the membrane
- These include conditions like pulmonary fibrosis
- Relatively rare as cause of hypoxia acutely or in ICU