Chapter 3: Invasive mechanical ventilation

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Introduction

Conventional mechanical ventilation is the delivery of positive pressure to the airway to allow removal of CO2 and delivery of O2. In invasive mechanical ventilation, this is delivered through an endotracheal tube, be it oral, nasal or a tracheostomy.

Indications of mechanical ventilation

1. Respiratory failure, hypoxic, hypercarbic or mixed
2. Control of PaCO2, e.g. for cerebral protection
3. Facilitate anaesthesia

Physiology of ventilation

In order for normal spontaneous ventilation to occur, the respiratory muscles must overcome two forces in order to allow movement of air into lung. These are:

1. The elastic recoil of the lung and chest wall
2. The resistance of the lung and chest wall

In mechanical ventilation/positive pressure ventilation, the pressure delivered also need to overcome similar issues:

1. The elastic recoil of the lung in which the pressure required is dependent on how much the lung is inflated (i.e. the volume of the lung). As an analogy, the bigger you blow up a balloon, the greater the pressure that is required.
2. The resistance of the respiratory system which is a function of the air flow i.e. the pressure required to simply move air through the bronchi, trachea etc. As an analogy, this is the pressure required to blow through a straw. This work is increased if the tube has a smaller radius and how fast the air flow is (the faster, the harder due to increase in turbulent flow).
3. The pressure to maintain a residual lung volume at the end of expiration, also known as positive end expiratory pressure (PEEP).

Modes of ventilation

How patients are ventilated depends on the mode of the ventilation and the type of ventilator. In this chapter we will discuss the most common modes of positive pressure ventilation.

Controlled mechanical ventilation

This is the most basic form of ventilation, while it is uncommon to see the pure form of this in the ICU, it does explain the basics behind positive pressure ventilation. We will discuss the two main modes: Volume control ventilation (VCV) and pressure control ventilation (PCV).

Volume control ventilation

There two key settings in order to deliver ventilation; the tidal volume (Vt) and the respiratory rate (f). The Vt is the volume that is delivered to the lungs at each breath and the frequency is the number of breaths that will delivered within each minute. Minute volume (MV), which is a standard of quantifying ventilation, measures the total volume delivered to the lungs in a minute and is the product of Vt and f (i.e. Minute volume= Vt x f).

Minute volume

The typical minute volume is around 8-10ml/kg. However it is important to know that in ICU patients the actual minute volume required is very variable depending on their illness and what drugs they are on. Therefore, the best marker for adequate ventilation is the arterial partial pressures of CO2 (PaCO2). Increasing ventilation will blow out CO2 and will therefore PaCO2 will drop and decreasing ventilation will mean that the body will retain CO2 and therefore the CO2 will rise. The end tidal CO2 (ETCO2) monitors the CO2 at the end expiration of a breath is a surrogate marker of PaCO2. It is not an accurate marker, and will underestimate PaCO2, but it is monitored on all ventilated patients and therefore is easily accessible compared to the PaCO2, which needs a blood gas to be sampled and analysed.

Tidal volume

In the traditional VCV, the flow is a set rate and that the tidal volume is delivered by adjusting the inspiration time. For example, if a ventilator delivers a flow rate of 500ml/s, if you adjust the tidal volume to 250ml, then the inspiratory time would be adjusted to 0.5s. If 500ml is required, then the ventilator would deliver the volume by adjusting the inspiratory time to 1sec.

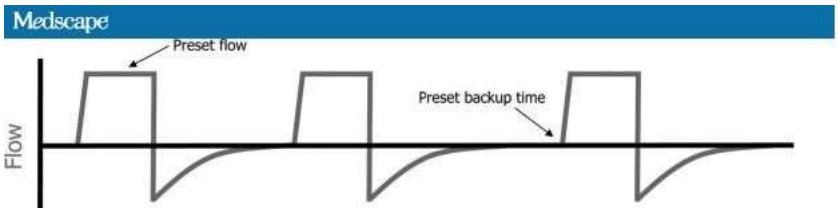

Figure 1: volume control ventilator waveforms. This is the flow vs time waveform. The flow accelerates to a set rate and inspiration will occur at that fixed flow rate at an inspiratory time that is adjusted to achieve the given tidal volume.

In a typical patient, this is normally in the range of 8-10ml/kg. In recent years, there is more evidence that higher tidal volumes can cause damage to the lung, specifically where the lung is injured. In patients who are at risk of or have acute lung injury (ALI) or acute respiratory

distress syndrome (ARDS), where the lung is essentially inflamed, we aim for lower tidal volumes of 6ml/kg.

Frequency aka respiratory rate

Typically 10-16/min. It should be adjusted to maintain adequate minute ventilation which can be monitored by using ETCO2 or PaCO2. Increasing the frequency or the respiratory rate (RR) can increase CO2 clearance, but this will increase the chance of gas trapping, particular in patients with airway narrowing e.g. asthmatics. Gas trapping is where the air that enters the lung does not have time to completely exit the lung during expiration before the next inspiration begins. The result is that the lung becomes more inflated with each breath (called dynamic hyperinflation) and if left unchecked, this can cause the lung to be so inflated that the blood from the right ventricle cannot cross the lung into the left heart causing cardiac arrest. If you notice haemodynamic instability, the solution is to disconnect the patient from the ventilator to allow for expiration. If there is no haemodynamic instability then you should reduce the respiratory rate until gas trapping no longer occurs. This can be diagnosed by looking at the ventilator waveforms (1).

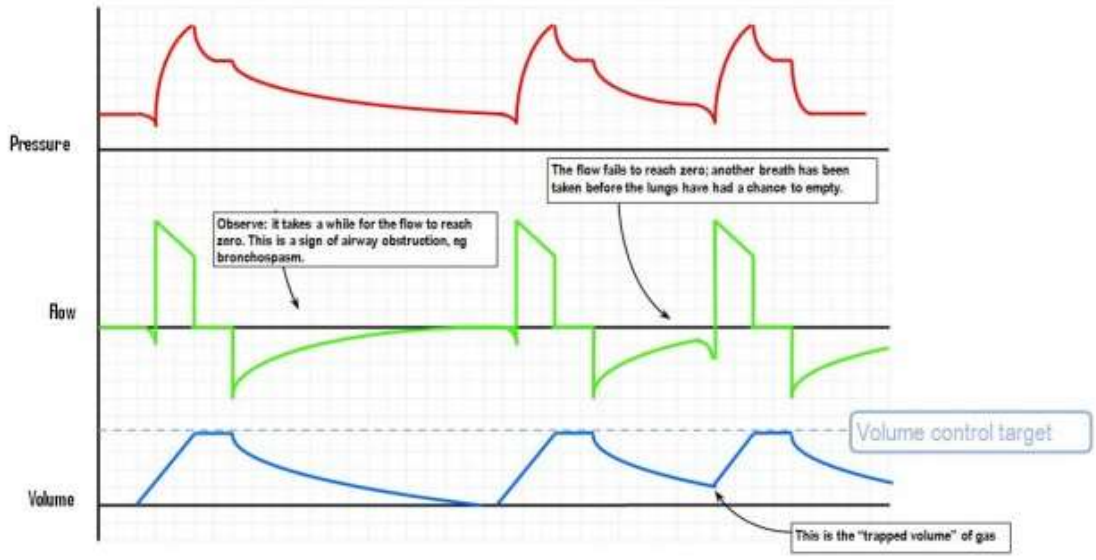

Fig. 2: Gas trapping and ventilator waveforms: There are 3 waveforms in the diagram. The top wave is a pressure time waveform, the middle is flow time waveform and the bottom is the volume time waveform. Note that in gas trapping, the flow does not reach 0 (i.e. the baseline) before the next inspiration and in the volume waveform also does not reach baseline before inspiration.

Pressure control ventilation

This is similar to volume control ventilation in all respects except that instead of setting a tidal volume, you set a pressure that the ventilator will deliver. The ventilator will deliver the set pressure at inspiration and the flow of the breath will change during the inspiratory cycle depending on the pressure difference between the alveolus and the set pressure. Now,

we have another thing to consider; when will inspiration stop? In volume control mode, inspiration stops when the target tidal volume is achieved. In pressure control, you will need to set the amount of time you want the ventilator in inspiration as there is no volume target. In most ventilators you can set an ***inspiration time (Ti),*** the duration you want for the patient to inspire at the set pressure. In pressure control, setting times are important and that inspiratory time interacts with respiratory rate and expiratory times. For instance if you have an inspiratory time of 2secs and set a respiratory rate of 30/min, then the amount of time spent in inspiration in that minute:

Total inspiratory time in a minute= Inspiratory time x frequency

=2sec x 30/min

=60sec

In other words, the patient will inspire the whole time, which would be unsurvivable. Expiration time needs to be considered, which brings in another concept, which is the ***inspiratory/expiration ratio (I:E ratio)***.

Inspiratory/expiratory ration (I:E ratio)

This describes the relationship of inspiration time to expiration time. A normal I:E ratio is 1:2 i.e. for each second the patient breaths in, 2 seconds will be given for expiration. Remember that inspiration is an active process whereas expiration is a passive process, and therefore will need to be **generally** longer than inspiration in most, but not all circumstances.

High I:E ratio

Situations where I:E ratio needs to be high i.e. longer inspiratory time relative to expiratory time where is ratio is as high as 1:1 (2), are where the lungs are stiff and non-compliant. Examples include:

- Acute lung injury or acute respiratory distress syndrome (ARDS)
- Infants
- Obese patients

Low I:E ratio

Situations where low ratios are required are where there is increase airway resistance. Remember that inspiration is an active process where increased pressure can drive air flow into the lungs, but that expiration is a passive process where the lung recoils. The air cannot be “forced” out and therefore for the air to be expired, you have to wait for the air to slowly expire. In this instance, you would decrease the I:E ratio to 1:3 or 1:4. Examples of this include:

- Bronchospasm is asthmatics or COPD
- Airway obstruction
- Relatively small endotracheal tubes (e.g. Size 5 ETT in an adult male)

Synchronised intermittent ventilation SIMV

This is the most common mode that you will see used in the ICU. In the section above, we have talked about controlled mechanical ventilation, where ventilator does all the breathing. This generally require a patient that is either deeply anaesthetised and/or paralysed with muscle relaxants. In reality, most patients in the modern ICU are only moderately to mildly sedated with no paralysis. This means the patients will try to breath on their own which means they fight with ventilator if it was in a fixed controlled ventilation mode e.g. if the patient breaths out while the ventilator tries to make them inspire. This is called patient-ventilator asynchrony (3). In SIMV, the ventilator will allow the patients to breath by their own effort. Most ventilators will allow a pressure support breath to be delivered when the patients breath on their own. The patient does this by "triggering" a breath, which could be detected by air flow or a drop in pressure within the ventilator circuit. The ventilator will also deliver the mandatory breath set on the ventilator, if the patients do not achieve this by themselves.

Pressure support ventilation

This is used commonly used when we wean a patient off the ventilator (4). Patients are awake enough that they can trigger their own breaths and have the ability to do a reasonable amount of respiratory work themselves. Like SIMV, in pressure support the ventilator detects when the patient breathes either by detecting the flow or the negative pressure within the circuit (settings which can be adjusted) and delivers flow at a fixed pressure while the patient inspires. As the flow of the inspiration reduces as patient transitions to expiration, the pressure delivered by the ventilator stops so that the patient isn't trying to expire while the ventilator is delivering pressure. The set pressure that the ventilator delivers is called ***pressure support (PS)***.

Oxygenation

We have talked about the mechanics of ventilation in the early parts of this chapter. Ventilation is about getting air in and out of the lungs. Oxygenation talks about how we maintain oxygen supply to the tissues, commonly using partial pressure of oxygen in arterial blood (PaO2) as the marker for this. In the previous chapter, we have talked about the causes of hypoxia. In this section, we will be focussing on ventilation strategies which can increase PaO2.

1. Increase FIO2. By increasing the oxygen concentration you deliver to the alveolus, you can potentially increase the PaO2 in the blood. However for this to occur, the oxygen must be delivered to the alveoli (i.e. adequate alveoli ventilation) and that there is no shunt.
2. Ensure adequate ventilation. There is a relationship between alveoli ventilation and PaO2. This makes sense, because if you don't breathe, fresh oxygen can't get to your lungs to replace the oxygen that your body uses. Below is the alveolar gas equation (5) which shows the mathematical relationship

$PaO_2 = PAO_2 + PaCO_2/R$

PaO_2=Partial pressure of oxygen in the arterial blood

PAO_2=partial pressure of oxygen in the Alveolus

$PaCO_2$=Partial pressure of CO_2 in the arterial blood

R=respiratory ratio, and is the relationship between O_2 consumption and $PaCO_2$. $PaCO_2/R$ reflects the predicted oxygen consumption of the body

3. Increase Positive end expiratory pressure (PEEP). PEEP functions by preventing the alveolus collapsing that can happen during end expiration by maintaining positive pressure (6). The collapse of alveolus has two effects: The first is that of hypoxia as the alveolus will not be oxygenated, and blood that passes through that alveolus will not be oxygenated and therefore eventually will reduce the total amount of oxygen that is carried back into the left atrium. This is in effect a shunt i.e. blood that passes from the right side to the left side of the heart without being oxygenated. The second is that of decrease in the compliance of the lung i.e. how easily the lung can be expanded, because you now have to blow into the lungs harder to expand the alveolus that is already collapsed.
4. Increase the mean airway pressure. This can be done by increasing PEEP, as well as increase the inspiratory time or the I:E ratio. The mechanism of this is similar to the reasons of increasing PEEP. Note that the downsides of increasing inspiratory time is that you may not give the lungs enough time to fully expire and therefore causing breath stacking i.e. that lungs expands at each breath because it can't fully expire during each breath cycle. The other downside to increasing the mean airway pressure is that although the short term physiological changes will improve oxygenation, excessive pressure (>30-35mmH_2O) can cause barotrauma and actually injure the lung and cause an increase in patient mortality.
5. Recruitment strategies. The goal of this is to inflate the lungs and reduce the amount of lungs that are collapsed and therefore improved oxygenation by reducing shunt. There are variety of ways to do this (7), but the most simple way is to do an inspiratory hold at 30-40mmH_2O using the pressure control mode. Other ways include the use of Bi-level ventilation and step wise increase in PEEP (8).

Other non-mechanical ventilation strategies:

1. Changes in posture. Gravity alters how the lung ventilates and alters the blood flow through the lung. Blood flow will generally increase at dependent regions. Below describes changes at different posture in general (not necessarily on a specific patients)
    a. Sitting up position: Increased blood flow to the base of the lungs, but most importantly reduces the compression of the lungs by the abdominal contents that happens when the patient is in a supine position. This reduces the collapse of the alveolus and coupled with increased blood flow at the base of the lungs, oxygenation normally improves.
    b. Lateral position: In diseased lungs, generally ventilation does not alter significantly, but blood flow does. In general, when the patient is turned on

the pathology side down, blood flow increases in the disease lung where ventilation is poor, causing hypoxia.

c. Prone positioning: If you think a patient requires prone positioning, the consultant should be consulted. Prone positioning is used in patients with severe ARDS (9) where inflammation is more severe in the dependent areas i.e. dorsally, while there is overdistension in the ventral regions of the lung. Proning reverse these effects by opening up the alveolus of the dorsal part of the lung, while preventing overdistension on the ventral part of the lung. This allows for better V/Q matching as the "good lungs" ventrally will be perfused with more blood at the same time recruiting the dorsal lung.

2. Reducing mucus plugging. Patients who are ventilated have poor clearance of their secretion in addition to any sputum/blood which they may produce. These can obstruct branches of the bronchial tree which can cause no ventilation in large parts of lungs. This creates a shunt and can cause significant hypoxia. Suctioning of mucus or secretions in such instance can relieve the obstruction and therefore improve oxygenation.
3. The use of inhaled Nitric oxide (10): This is a vasodilator. When inhaled, it only goes to only the ventilated part of the lung. The better the ventilation and higher the concentration and hence the better the alveolus is ventilated, the greater the vasodilation of its blood supply. This improves the V/Q mismatch and thereby increases oxygenation.
4. ECMO. New Zealand has a national centre in ECMO at CVICU, Auckland Hospital. Referral is via specialist and is used where conventional ventilation has failed.

Lung protection strategies

The above strategies will increase oxygenation, but it is important to note, in patients with ARDS, you need to take a lung protection approach. This mean the use of lower tidal volumes of 6ml/kg (11) of lean body weight, peak airway pressure <30cmH2O, and use of permissive hypercapnia i.e. disregarding of a high CO2 and accepting under ventilation. Some centres also accept a low SpO2 target (90-92%). This is to prevent applying too much pressure and distension of the lung, avoiding prolonged high oxygen concentrations which in themselves can cause lung damage and increase mortality.

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