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1. **INTRODUCTION TO MECHANICAL VENTILATION:**

In the past few years there has been an increase in the number of methods by which positive pressure ventilation can be delivered. The increasing number of methods available to deliver mechanical ventilation has made it difficult for clinicians to learn all that is necessary in order to provide a safe and effective level of care for patients receiving mechanical ventilation. Despite the method by which mechanical ventilation is applied the primary factors to consider when applying mechanical ventilation are:

- the components of each individual breath, specifically whether pressure, flow, volume and time are set by the operator, variable or dependent on other parameters
- the method of triggering the mechanical ventilator breath/gas flow,
- how the ventilator breath is terminated:
- potential complications of mechanical ventilation and methods to reduce ventilator induced lung injury
- methods to improve patient ventilator synchrony; and
- the nursing observations required to provide a safe and effective level of care for the patient receiving mechanical ventilation

The following sections will provide an overview of each of the above considerations. This section - an introduction to mechanical ventilation will provide a rather detailed overview of four key parameters that are necessary to consider when evaluating and classifying ventilator delivered breaths. These parameters are

- pressure,
- volume,
- flow and
- time.

If you are relatively inexperienced in the application of mechanical ventilators you may find this and later sections challenging. Keep in mind as you work through this package that that the intended aims of this package are to provide you with resource material and introduce you to topic areas that will form the basis for your future professional development. Questions throughout the package, and summary pages at the end of relevant sections, will direct you to the key concepts that are necessary to provide a safe and effective level of care, for the patient receiving mechanical ventilatory support. If you are interested in a specific topic area readings are highlighted in the package and a full reference list is provided. There is purposely no space provided within the package to answer questions. This, it is hoped, will enable you to develop notes and explore areas of interest. While attempts have been made to provide you with several answers to the questions within the text of this document, you may find it useful to discuss answers / queries with your colleagues. Answers relating to causes of alarm violations, for example, may be difficult to find in journals or text books. Many of the answers are developed through experience and it would be useful to utilise some of your colleagues knowledge in these areas.
1.1 Airway Pressures (Paw)

For gas to flow to occur there must be a positive pressure gradient. In spontaneous respiration gas flow occurs due to the generation of a negative pressure in the alveoli relative to atmospheric or circuit pressure (as in CPAP) (refer to following diagram).

Mechanical ventilation delivers flow and volume to the patient’s as a result of the development of a positive pressure gradient between the ventilator circuit and the patient’s gas exchange units as illustrated in the diagram above. There are four pressures to be aware of in regards to mechanical ventilation. These are the:

1. Peak
2. Plateau
3. Mean; and
4. End expiratory pressures.
1.1.1 Definitions

- **Peak Inspiratory Pressure (PIP).** The peak pressure is the maximum pressure obtainable during active gas delivery.

- **Plateau Pressure.** The plateau pressure is defined as the end inspiratory pressure during a period of no gas flow.

- **Mean Airway Pressure.** The mean airway pressure is an average of the system pressure over the entire ventilatory period.

- **End Expiratory Pressure.** End expiratory pressure is the airway pressure at the termination of the expiratory phase and is normally equal to atmospheric or the applied PEEP level. 1,2,3

1.1.2 Pressure Measurement

During the delivery of a positive pressure breath, system pressure can be measured in a variety of locations, these include:

- internal to the ventilator - inspiratory / expiratory;
- at the Y piece of the ventilator circuit;
- at the airway opening; and
- at the carina - by applying the pressure monitoring line to a tracheal tube with an extra lumen. 2

The farther away the site of measurement is from the alveoli the greater the potential for difference between the pressure reading on the ventilator and the pressure in the alveoli 2. Increased resistance to airflow in the ventilator circuit, the endotracheal tube, or the patients conducting airway will be reflected in an increased difference between peak inspiratory and alveolar pressure. 4
This means that the pressures measured by the mechanical ventilator will not always be indicative of alveolar pressure. During inspiration, for example, gas is moving from the circuit into the alveoli and the pressure in the circuit will be greater than alveolar pressure. Conversely during expiration, gas is moving from the alveoli into the circuit and the pressure in the alveoli will be greater than the pressure in the circuit. The only time in which alveolar pressure equals circuit pressure is during a period of no flow. Periods of no gas flow occur during an inspiratory hold (pause) or at the end of exhalation after which time expiratory gas flow has ceased. (Refer to the following diagram).

Because of these considerations the observation of airways pressure during periods of no flow and when there is no flow can provide useful information.
Alveolar vs Circuit Pressure

The following diagram depicts how circuit pressure and alveolar pressure differ during mechanical ventilation. You will note in the pressure trace there are two pressure recordings. The darkened line ( ) represents circuit pressure whereas the broken line ( ) represents alveolar pressure. During inspiration circuit pressure is greater than alveolar pressure. Conversely during expiration alveolar pressure exceeds circuit pressure. You will note that the only time when these pressures are equal when there is a period of no flow i.e., during an inspiratory pause or after expiration has ceased.
1.1.3 Peak Inspiratory and Plateau Pressures

When pressure is plotted against time for a ventilator machine breath a waveform is results that is illustrated in the figure on page 5. Two points of significance have been identified on this graph;

1. **Peak Inspiratory Pressure (PIP)**
   This pressure a function of the compliance of the lung and thorax and the airway resistance including the contribution made by the tracheal tube and the ventilator circuit (if the pressure is measured from a site in the circuit that is close to the ventilator).

2. **Plateau Pressure**
   If volume is kept constant at the end of inspiratory flow the peak inspiratory pressure will fall to a pressure level called the plateau pressure. The plateau pressure reflects lung and chest wall compliance.

The drop in pressure from PIP to Plateau results from the fact that inspiratory flow has ceased therefore pressure is not required to overcome resistance to flow. The pressure also falls as a result of redistribution of gas within the lungs, “elastic give” (this is a property of elastic materials which results in a drop in pressure after a period of time at the same volume) recruitment of alveoli and the effect of surfactant.

As the plateau pressure is the pressure when there is no flow within the circuit and patient airways it most closely represents the alveolar pressure and thus is of considerable significance as it desirable to limit the pressure that the alveoli are subjected to. Excessive pressure may result in extrapulmonary air (eg pneumothorax) and acute lung injury.

An increase in airways resistance (including ETT resistance) will result in an increase in PIP. An increase in resistance will result in a widening of the difference between PIP and plateau pressure. A fall in compliance will elevate both PIP and plateau pressure.

While recognising that the causes of ventilator-induced lung injury are multi-factorial, it is generally believed that end inspiratory occlusion pressure (ie plateau pressure) is the best clinically applicable estimate of average peak alveolar pressure. Although controversial it has been generally recommended that the plateau pressure should be limited to 35 cms H\textsubscript{2}O.
Question 1) Which of the following waveforms indicate
1. an increased resistance
2. a decreased compliance
3. and increased resistance and a decreased compliance

Which type of patient might experience both a decreased compliance and an increased resistance?
1.1.4 PEEP and CPAP

Positive end expiratory pressure (PEEP) refers to the application of a fixed amount of positive pressure applied during mechanical ventilation cycle. Continuous positive airway pressure (CPAP) refers to the addition of a fixed amount of positive airway pressure to spontaneous respirations, in the presence or absence of an endotracheal tube. PEEP and CPAP are not separate modes of ventilation as they do not provide ventilation. Rather they are used together with other modes of ventilation or during spontaneous breathing to improve oxygenation, recruit alveoli, and/or decrease the work of breathing.6, 7, 8

The major benefit of PEEP and CPAP is achieved through their ability to increase functional residual capacity (FRC) and keep FRC above Closing Capacity. The increase in FRC is accomplished by increasing alveolar volume and through the recruitment of alveoli that would not otherwise contribute to gas exchange. Thus increasing oxygenation and lung compliance. 6,7,8.

The potential ability of PEEP and CPAP to open closed lung units increases lung compliance and tends to make regional impedances to ventilation more homogenous.

Physiological Responses to CPAP / PEEP

PEEP and CPAP may decrease cardiac output and mean arterial blood pressure through a decrease in venous return and hence ventricular filling, as illustrated in the following diagram. In patients with poor left ventricular function and pulmonary oedema the addition of CPAP or PEEP may improve cardiac output through an improvement of stroke volume. 6,7,8.

Fluid retention and diminished urinary output are commonly observed in patients receiving PEEP, particularly in conjunction with mechanical ventilation. Mechanical ventilation and PEEP increase the production of antidiuretic hormone, decrease mean renal artery perfusion pressure, redistribute perfusion from the cortex, reduce urine flow, reduce creatinine clearance and diminish fractional excretion of sodium.
Effects of CPAP

CPAP results in
- increased CVP
- decreased RVEDV (preload)
- increased PVR (RV afterload)
- increased PAWP (wedge pressure)
- decreased LVEDV (preload)
- decreased LV afterload

Schematic Representation Of The Multiple Effects Of Positive Pressure Ventilation On Renal Function (adapted from Perel & Stock 1992 P 69)

Positive Pressure Ventilation

Increased Intrathoracic Pressure

Decreased Cardiac Filling

Pressure, Inferior Vena Cava

Decreased Atrial Natriuretic Factor

Decreased Left Ventricular Size

Decreased Cardiac Output

Decreased Mean Arterial Pressure

Increased Baroreceptors

Increased Antidiuretic Hormone

Increased Renal Nerve Stimulation

Increased Renin - angiotensin - aldosterone.

Decreased Urine Volume

Decreased Urine Sodium Excretion
1.1.5 AutoPEEP

You will recall from the previous section on pressure measurement that circuit pressure is not always indicative of alveolar pressure. During expiration alveolar pressure is greater than circuit pressure until expiratory flow ceases. If expiratory flow does not cease prior to the initiation of the next breath gas trapping may occur. Gas trapping increases the pressure in the alveoli at the end of expiration and has been termed:

- dynamic hyperinflation;
- autoPEEP;
- inadvertent PEEP;
- intrinsic PEEP; and
- occult PEEP

Although we quite commonly aim for patients to have increased pressure in their alveoli at the end of expiration (PEEP), autoPEEP is potentially harmful as we may not be aware of its presence. The effects of autoPEEP are the same as PEEP/CPAP and can predispose the patient to:

- an increased risk of barotrauma;
- fall in cardiac output;
- hypotension;
- fluid retention; and
- an increased work of breathing.

---

**Question 2) Your patient is on the following ventilator settings**

- Tidal Volume: 700 mls
- Plateau pressure: 45
- PEEP 5 cmsH2O

*The PEEP is increased to 15 cmH2O and the plateau pressure decreases to 35 cmsH2O. Provide a rationale for the change in inspiratory pressure.*
1.1.6 How does the presence of AutoPEEP increase work of breathing?

In health the pressure in the alveoli at the end of expiration is the equivalent to atmospheric pressure. The pressure between the parietal and visceral pleura at this time is negative. To achieve gas flow into the alveoli the diaphragm and external intercostal muscles contract creating a more negative intrapleural pressure. This causes the alveoli to expand and results in a sub atmospheric alveolar pressure that produces gas flow. When autoPEEP is present the pressure in the alveoli at end expiration is greater than atmospheric, the size of the thorax is expanded and the respiratory muscles have returned to their lengthened resting state. To generate gas flow the respiratory muscles must shorten enough to expand the thorax beyond its increased dimensions and create a sub atmospheric alveolar pressure. If this pressure is not generated no gas flow will occur. When a patient is intubated and on a ventilator the demand response time of the ventilator may further exacerbate this problem.

Through the addition of CPAP / PEEP the pressure gradient between the alveoli and the circuit is reduced, thereby decreasing the inspiratory work of breathing.
Alveolar Pressure during Inspiration with 20 cms AutoPEEP & 15 cms CPAP
1.1.7 The Measurement of AutoPEEP

AutoPEEP, unlike externally applied PEEP, is not registered on the ventilator’s pressure monometer. This is because the ventilator registers circuit pressure and not alveolar pressure. If however the exhalation valve on the ventilator is occluded immediately before the onset of the next breath, the pressure in the alveoli and the ventilator circuit will equilibrate. By performing this manoeuvre the level of auto-PEEP will be displayed on the ventilator (see following diagram).

It is important to note that this method of measuring autoPEEP can only be used when the patient is receiving controlled breaths. When the patient is taking spontaneous or assisted breaths the pressure in the circuit will obviously drop, to initiate gas flow, and a measurement of autoPEEP will be unattainable. To ascertain if a patient has autoPEEP during spontaneous or assisted breaths it is necessary to view the flow waveforms on the ventilator or insert an oesophageal balloon. An analysis of flow waveforms on the graphics menu will allow you to detect if autoPEEP is present. If the expiratory flow does not return to baseline before the next breath autoPEEP is present (see following diagram).
By inserting an oesophageal balloon in a patient the presence of autoPEEP can be ascertained and measured. The basis for using an oesophageal balloon is that oesophageal pressure has been shown to closely reflect intrapleural pressure. Thus the amount of oesophageal pressure required to initiate gas flow is reflective of the level of autoPEEP. Monitors, such as the Bicore Pulmonary Monitor, utilise an oesophageal balloon with a flow transducer and pressure sensor that can be added to the Y-piece of the ventilator circuit or connected to a T-piece on a CPAP circuit. The Bicore Pulmonary Monitor defines auto-PEEP as the difference in end expiratory oesophageal pressure and oesophageal pressure at the start of inspiratory flow minus sensitivity (see following diagram). The Bicore defines sensitivity as the measurement of ventilator demand valve sensitivity. It is calculated as the airway pressure before the onset of inspiratory flow.

**Question 3) Complete the following summary of AutoPEEP:**

- AutoPEEP is:__________________________
- AutoPEEP can occur in patients who aren’t ventilated True / False
- AutoPEEP can be measured in spontaneously breathing patients by occluding the exhalation valve True / False
- Which of the following diagrams demonstrates autoPEEP 1, 2, or 3

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Further Reading - AutoPEEP

1.2 Volume (VT)

Tidal volume refers to the size of the breath that is delivered to the patient. Normal physiologic tidal volumes are approximately 5-7 mls / kg whereas the traditional aim for tidal volumes has been approximately 10 - 15 mls / kg. The rationale for increasing the size of the tidal volume in ventilated patients has been to prevent atelectasis and overcome the deadspace of the ventilator circuitry and endotracheal tube. Inspired and expired tidal volumes are plotted on the y axis against time as depicted in the following diagram.

The inspired and expired tidal volumes should generally correlate although certain circumstances may cause a difference between inspired and expired tidal volumes. Expired tidal volumes may be less than inspired tidal volumes if:
- there is a leak in the ventilator circuit - causing some of the gas delivered to the patient to leak into the atmosphere
- there is a leak around the endotracheal / tracheostomy tube - due to tube position, inadequate seal or cuff leak - causing some of the gas delivered to the patient to leak into the atmosphere
- there is a leak from the patient, such as a bronchopleural fistula - causing some of the gas delivered to the patient to leak into the atmosphere

Expired tidal volumes may be larger than inspired tidal volumes due to:
- the addition of water vapour in the ventilator circuitry from a hot water bath humidifier.

The following diagrams depict examples where inspired and expired tidal volumes do not correlate.
1.3 Flow (V)

Flow rate refers to the speed at which a volume of gas is delivered, or exhaled, per unit of time. Flow is described in litres per minute (lpm). (Banner and Lampotang) The peak (inspiratory) flow rate is therefore the maximum flow delivered to a patient per ventilator breath. Flow is plotted on the y axis of the ventilator graphics against time on the x axis (refer to following diagram). You will note on the following diagram that inspiratory flow is plotted above the zero flow line, whereas expiratory flow is plotted as a negative deflection. When the graph depicting flow is at zero there is no gas flow going into or out of the patient.

Question 4): What factors might contribute to the development of atelectasis in the intubated and ventilated patient?
1.4 Time (Ti)

Time in mechanical ventilation is divided between inspiratory and expiratory time. Inspiratory time is a combination of the inspiratory flow period and time taken for inspiratory pause. The following diagram depicts how the addition of an inspiratory pause extends total inspiratory time.

![Diagram showing inspiratory time (Ti) with and without inspiratory pause]

Normal inspiratory time on the spontaneously breathing healthy adult is approximately 0.8 - 1.2 seconds, with an inspiratory expiratory (I: E) ratio of 1:1.5 to 1:2. At times it may be advantageous to extend the inspiratory time in order to:

- improve oxygenation - through the addition of an inspiratory pause; or to
- increase tidal volume - in pressure controlled ventilation

Adverse effects of excessively long inspiratory times are haemodynamic compromise, patient ventilator dysynchrony, and the development of autoPEEP.

**Question 5). What are the factors to consider when prolonging inspiratory time beyond normal parameters?**

The previous sections have provided an overview of pressure, flow, volume and time. While there are many methods by which mechanical ventilation could be applied the following guidelines should assist you in providing a safe and effective level of care for your assigned patients, regardless of what type of ventilation is implemented.

Pressure:

**Peak and Plateau Pressure.** While recognising that the causes of ventilator induced lung injury are multifactorial increased intrathoracic pressures have been identified as a potential mechanism of inducing lung injury. It is generally accepted that the plateau pressure should not exceed 35 cm H$_2$O. An elevated peak pressure above this level may still be a cause for concern as some alveoli may be receiving this pressure.

**End Expiratory Pressure.** PEEP and CPAP improve oxygenation through their ability to increase functional residual capacity. PEEP and CPAP may not only be of benefit in increasing the level of oxygenation but may also be useful in the recruitment of alveoli, reduction of work of breathing and the prevention of acute lung injury. Both PEEP and CPAP however may cause a decrease in cardiac output, fluid retention, and increase the risk of the development of extra pulmonary air (eg pneumothorax).

Where there is inadequate time for expiration gas trapping may occur and autoPEEP may be present. AutoPEEP is not registered on the ventilator’s pressure monitor but may be identified by:
- observing the flow - time waveform on the graphic waveforms;
- performing and expiratory hold manoeuvre - machine initiated breaths only; or
- by inserting an oesophageal balloon and observing the appropriate pressures.

AutoPEEP is potentially harmful as it represents an additional amount of PEEP that has not been set by the operator.

**Volume.** The size of the tidal volume to be delivered is generally dictated by unit practice (eg 10 mls / kg) but is usually set to ensure adequate elimination of carbon dioxide without producing excessive inspiratory pressure.

**Flow.** The peak inspiratory flow rate should be set to match the patient’s inspiratory flow requirements. Where flow does not meet this requirement the patient’s work of breathing may be unnecessarily increased. A type of ventilation with a variable flow delivery system eg pressure support and pressure controlled ventilation may be more effective in matching the patient’s inspiratory flow requirements than a ventilation mode with a set flow, eg volume cycled ventilation.

Where available, a decelerating flow waveform may be preferable as it will result in less high peak inspiratory pressure than the other flow waveforms.
**Time.** Normal inspiratory time is 0.8 - 1.2 seconds with an inspiratory: expiratory of 1:2 or 1:1.5. Prolonging inspiratory time through the addition of an inspiratory pause may be beneficial in improving oxygenation and recruiting alveoli. In pressure controlled ventilation increased inspiratory time may also be beneficial in increasing tidal volume (see section on Pressure Controlled Ventilation). Extending inspiratory time however, may increase the potential for the development of autoPEEP, cause haemodynamic compromise and be a source of discomfort to the patient - causing them to “fight” the ventilation (refer to section on patient ventilator synchrony).
2. TRIGGERING

Triggering refers to the mechanism through which the ventilator senses inspiratory effort and delivers gas flow or a machine breath in concert with the patient’s inspiratory effort. In modern ventilators the demand valve is triggered by either a fall in pressure (pressure triggered) or a change in flow (flow triggered). With pressure triggered a preset pressure sensitivity has to be achieved before the ventilator delivers fresh gas into the inspiratory circuit. With flow triggered a preset flow sensitivity is employed as the trigger mechanism.

2.1 Pressure Triggering

In pressure triggering the sensitivity refers to the amount of negative pressure the patient must generate to receive a breath/gas flow. If the sensitivity is set at 1 cm then the patient must generate 1 cm H\textsubscript{2}O of negative pressure, at the site of pressure measurement, for the machine to sense the patient's effort and deliver a breath / gas flow. The sensitivity should be set as close to zero as possible, without allowing the machine to cycle spontaneously. If the sensitivity is too high the patient's work of breathing will be unnecessarily increased. **It is not a reasonable course of action to increase the sensitivity to reduce the patient's respiratory rate as you will only increase their work of breathing.**

Through observation of the pressure-time trace on the graphics or the ventilators pressure manometer you will note that quite frequently the pressure drops well below the set sensitivity. The reason for this drop in pressure is due to the time lag between when the patient drops the pressure in the circuit and when the ventilator actually delivers flow. **This is known as the demand responsiveness of the ventilator.** In some ventilators the airway pressure drop from end expiratory pressure level is as large as 6-8 cmH\textsubscript{2}O with a 0.3-0.7 second time delay. The above mentioned factors are partly determined by the characteristics of the demand valve and the added resistance of the inspiratory and expiratory circuits. This leads to an increased inspiratory muscle work and oxygen consumption.

Note the greater negative inflection in the second pressure time trace - indicative of a poorer demand response time than the first example.
2.2 Flow Triggering

The flow triggered system has two preset variables for triggering, the base flow and flow sensitivity. The base flow consists of fresh gas that flows continuously through the circuit and out the exhalation port, where flow is measured. The patient’s earliest demand for flow is satisfied by the base flow. The flow sensitivity is computed as the difference between the base flow and the exhaled flow. Hence the flow sensitivity is the magnitude of the flow diverted from the exhalation circuit into the patient’s lungs. As the subject inhales and the set flow sensitivity is reached the flow pressure control algorithm is activated, the proportional valve opens, and fresh gas is delivered.\textsuperscript{5}

The time taken for the onset of inspiratory effort to the onset of inspiratory flow is considerably less with flow triggering when compared to pressure triggering. At a flow triggering sensitivity of 2 litres per minute, for example, the time delay is 75 milliseconds, whereas the time delay for a pressure sensitivity of 1 cm H\textsubscript{2}O is 115 milliseconds - depending on the type of ventilator used. The use of flow triggering decreases the work involved in initiating a breath.\textsuperscript{15}
3. **VOLUME CYCLED VENTILATION**

Volume cycled ventilation delivers a:
- set volume;
- with a variable **Pressure** - determined by resistance, compliance, inspiratory effort;
- set flow; and an
- **inspiratory time** that is determined by the inspiratory pause (if activated), flow rate, and tidal volume.

### 3.1 Inspiratory Pressures

Because pressure is the variable parameter in volume cycled ventilation it is critical to observe the patient's inspiratory pressures and act appropriately in response to increased inspiratory pressures.

In volume cycled ventilation the inspiratory pressures vary in response
- to the size of the breath delivered to the patient;
- the resistance of the endotracheal / tracheostomy tube;
- the resistance of the upper airways;
- the patients compliance; and
- inspiratory effort.

By monitoring the peak and plateau pressures in volume cycled ventilation it is possible to get an estimate of the patient's resistance and compliance.

A large difference between the peak and plateau pressures indicates an increased resistance. An elevated plateau pressure indicates a decreased compliance. Note it is possible to have both an increased resistance and decreased compliance, in which case there may be a large difference between the peak and the plateau pressures as well as elevated plateau pressures.
3.2 Flow Waveforms

In volume cycled ventilation inspiratory flow is controlled by setting the peak flow and flow waveform. The peak flow rate is the maximum amount of flow delivered to the patient during inspiration, whereas the flow waveform determines the how quickly gas will be delivered to the patient throughout various stages of the inspiratory cycle. There are four different types of flow waveforms available. These include the square, decelerating (ramp), accelerating and sine/sinusoidal waveform, as illustrated below.

![Flow Waveform Diagram]

It is important to note that these flow waveforms only effect inspiration. Expiratory flow is determined by the patient.

**Square waveform.** The square flow waveform delivers a set flow rate throughout ventilator inspiration. If for example the peak flow rate is set at 60 lpm then the patient will receive 60 lpm throughout ventilator inspiration.

**Decelerating waveform.** The decelerating flow waveform delivers the peak flow at the start of ventilator inspiration and slowly decreases until a percentage of the peak inspiratory flow rate is attained.

**Accelerating waveform.** The accelerating flow waveform initially delivers a fraction of the peak inspiratory flow and steadily increasing the rate of flow until the peak flow has been reached.

**Sine / sinusoidal waveform.** The sine waveform was designed to match the normal flow waveform of a spontaneously breathing patient.

**Setting the Peak Flow and Flow Waveform**

The flow rate should be set to match the patient’s inspiratory demand. Where the patient’s inspiratory flow requirements exceed the preset flow rate there will be an imposed work of breathing which may cause the patient to fight the ventilator and become fatigued. Where flow rate is unable to match the patient’s inspiratory flow requirements the pressure waveform on the ventilator graphics screen may show a depressed or “scooped out” pressure waveform, refer to the following diagram. This is often referred to as flow starvation. 25
The decelerating flow waveform is the most frequently selected flow waveform as it produces the lowest peak inspiratory pressures of all the flow waveforms. This is because of the characteristics of alveolar expansion. Initially a high flow rate is required to open the alveoli. Once alveolar opening has occurred a lower flow rate is sufficient to procure alveolar expansion. Flow waveforms which produce a high flow rate at the end of inspiration (ie. square and accelerating flow waveforms) exceed the flow requirements for alveolar expansion, resulting in elevated peak inspiratory pressures.\textsuperscript{2}
### 3.3 Inspiratory Time

In most volume cycled ventilators used in the intensive care environment it is not possible to set the inspiratory time. The inspiratory time is determined by the peak inspiratory flow rate, flow waveform and inspiratory pause. Where inspiratory time is able to be set, flow becomes dependent on inspiratory time and tidal volume. The following examples illustrate how these parameters effect inspiratory time.

**Ventilator settings**

- Tidal volume: 1000mls
- Peak Flow: 60 lpm
- Flow Waveform: square / constant
- Insp. Pause: 0 secs

The inspiratory time for this patient would be 1 second because gas is constantly being delivered at a flow rate of 60 lpm, which equals 1 litre per second. If an inspiratory pause of 0.5 seconds were applied then the inspiratory time would be increased to 1.5 seconds.

Changing the patients flow waveform from a square to a decelerating flow waveform, without changing the flow rate, will result in an increase in inspiratory time, because the flow of gas is only initially set at 60 lpm and decreases throughout inspiration (refer to following diagrams).
3.4 Summary Page - Advantages and Disadvantages of Volume Cycled Ventilation

Advantages

Ease of Use: Due to the widespread implementation of volume cycled ventilation it is a type of ventilation that is familiar to many clinicians.

Set Volumes: One of the major advantages of volume cycled ventilation is the ability to set a tidal volume. This is of critical importance to patient's requiring tight regulation of carbon dioxide elimination. Neurosurgical patients - post surgery / head injury and patients suffering a neurological insult (eg post cardiac arrest) often require CO\textsubscript{2} regulation. This is because carbon dioxide is a potent vasodilator. Increased levels of carbon dioxide, in these groups of patients, may therefore increase cerebral blood volume with a concomitant elevation of intracranial pressure. A raised intracranial pressure may decrease the delivery of oxygenated blood to the brain - resulting in cerebral ischaemia. Conversely a low CO\textsubscript{2} may cause constriction of the cerebral vasculature also resulting in decreased oxygen delivery and cerebral ischaemia. For these reasons volume cycled ventilation is often the mode of choice for patients requiring CO\textsubscript{2} regulation.

Disadvantages

The major disadvantages of volume cycled ventilation are the variable pressure and set flow rate. It is therefore a necessary part of nursing practice to closely monitor the patient's inspiratory pressure and observe the patient for signs of “flow starvation”.

Due to the limitations of volume cycled ventilation methods of ventilating patients with a set pressure and variable flow rate (eg pressure support and pressure controlled ventilation) are now widely available. Newer types of ventilation are now available which combine the ability to set a target tidal volume, maximum pressure and variable flow rate.

Question6). What are the factors could cause the:

- high inspiratory pressure alarm;
- low inspiratory pressure alarm;
- low tidal volume alarm; and
- low minute volume alarm

to be activated in volume cycled ventilation?

Describe appropriate action to be taken in order to rectify the problem.
4. **Pressure Support Ventilation**

The main goal of pressure support ventilation is to adequately assist respiratory muscle activity in a way that will improve the efficacy of a patient’s effort and allow a reduction in workload.

Pressure support only applies to spontaneous breaths. Pressure support has a:
- set **pressure** (pressure support added to the CPAP/PEEP);
- variable **volume** - determined by the resistance, compliance, inspiratory effort and level of pressure support;
- variable **flow** rate determined by the resistance, compliance, inspiratory effort and level of pressure support;
- variable inspiratory **time**; and
- is **cycled** off when the patient's inspiratory flow declines to a value determined by the manufacturer of the ventilator.
Pressure support ventilation is a pressure preset mode in which each breath is patient triggered and supported. It provides a means of a positive pressure that is synchronised with the inspiratory effort of the patient. Pressure support breaths are both patient initiated and patient terminated. During inspiration the airway pressure is raised to the preset level of pressure support level. The speed of pressurisation may be fixed by the ventilator or adjustable by setting the rise time (refer to the section on rise time / pressurisation)-5,16,17.

The **inspiratory pressures** in pressure supported breath are set by the operator. The peak pressure is determined by the addition of the level of pressure support to the level of CPAP/PEEP, ie peak pressure = pressure support + CPAP/PEEP. There is no plateau pressures in pressure supported breaths as it is impossible to achieve an inspiratory pause.

**Question7): Why is it impossible to have an inspiratory pause in pressure support ventilation?**

Because the ventilator’s algorithm is set to attain a preset pressure, the flow rate on the ventilator must respond to the:
- resistance of the endotracheal / tracheostomy tube
- resistance of the patient’s airway
- patient’s compliance; and
- inspiratory effort
The flow in pressure support must vary so that the preset level of pressure support is achieved and maintained throughout the breath. Flow cannot, therefore, be set by the operator. Likewise the flow waveform cannot be set but tends to be decelerating in nature. Initially a high flow rate is delivered to the patient in order distend the alveoli and overcome the resistance of the endotracheal tube. Once the alveoli opening occurs and the preset pressure has been obtained the rate of flow decreases - producing a decelerating flow waveform.

The termination of the pressure support breath is based on the decline of inspiratory flow. Inspiration cycles off when inspiratory flow falls to a preset value. This value may be a percentage of peak inspiratory flow (eg 25%) or a fixed amount of flow (eg 4 litres / min). The decline of inspiratory flow suggests that the patient’s inspiratory muscles are relaxing and that the patient is approaching the end of inspiration. At this point the inspiratory phase is cycled off. The ventilator terminates the pressure support and opens its exhalation valve. The expiratory phase is free of assistance, and returns to baseline pressure which may be level of CPAP/PEEP that is applied. 5,16,17

Pressure support ventilation is thus defined as a mode of ventilation:

- that is patient initiated,
- with a preset pressure,
- variable flow, volume and inspiratory time
- and is flow cycled

4.1 Application of Pressure Support

Pressure support ventilation may help to compensate for the increased respiratory muscle work required for breathing through an endotracheal tube and a demand valve. The level of pressure support ventilation required to compensate for the added inspiratory work caused by endotracheal tube resistance and a ventilator demand system is dependent on the resistance of the endotracheal tube and the underlying lung disease. 5,16,17

At high levels of Pressure Support Ventilation may used as a ventilation mode in it’s own right. 5,16,17
Question 8) You are caring for a 24 year old, 70 kg male post head injury with a size 8 tracheostomy tube. He is being ventilated with 6 cms of pressure support and 10 cms H$_2$O of CPAP. You are trying to wean the ventilation - intracranial pressure / cerebral perfusion are no longer a problem. The ventilator is continually activating its apnoea mode (set at 20 seconds) because the patient periodically stops breathing. The tidal volumes are approximately 1,100 mls and the respiratory rate is 6. What would be an appropriate action at this point in time? The graphic waveforms, for a 30 second time period, are supplied below.
Further Reading - Pressure Support


4.2 Pressurisation - Rise Time

Once inspiration has been initiated the ventilator delivers a high inspiratory flow that decreases, in response to the patient’s efforts, throughout the cycle of inspiration. The servo regulatory mechanism of the ventilator adjusts the required flow necessary to reach and maintain the appropriate pressure until expiration occurs. The flow regulation varies amongst ventilators. Pressure increases according to a time interval that is system specific or adjustable by the operator. A high speed of pressurisation results in a quicker achievement of the preset pressure support level.

- A low speed of pressurisation can cause the patient to breathe with excessive effort, especially when respiratory drive is high and respiratory mechanics are poor.

- A high speed of pressurisation may make it difficult for the ventilator to properly maintain the pressure throughout inspiration according to the servo control mechanism, especially in patients with low compliance or high resistance. For instance a very sudden rise in pressure under the action of a high flow rate and a high resistance may interfere with the pressure mechanism that cycles from inspiration to expiration. 6,17 The following diagram illustrates the how too quick a rise time may result in premature breath termination and a resulting ineffectual tidal volume. The first breath represents a normal pressure support breath. The second breath illustrates an initial rapid flow (due to a short rise time) that has caused the inspiratory pressure to rise higher than the set level of pressure support. The ventilator has compensated by rapidly decreasing the flow - which in turn has caused the ventilator to cycle the pressure support breath off. A prolonged rise time may be beneficial in this instance.

![Diagram showing pressurisation - Rise Time](image-url)
Question 9. What are the factors could cause the:
- high inspiratory pressure alarm;
- low inspiratory pressure alarm;
- low tidal volume alarm; and
- low minute volume alarm

to be activated in pressure support ventilation?

Describe appropriate action to be taken in order to rectify the problem.
5. **Pressure Controlled Ventilation**

Pressure controlled ventilation has a:
- set **pressure** (pressure controlled ventilation pressure added to the CPAP/PEEP);
- variable **volume** - determined by the resistance, compliance, inspiratory effort and level of pressure control ventilation pressure;
- variable **flow** rate determined by the resistance, compliance, inspiratory effort and level of pressure controlled ventilation pressure;
- set inspiratory **time**; and
- is **cycled** off by the inspiratory time (pressure controlled time cycled ventilation) or I:E ratio (pressure controlled ventilation I:E ratio cycled)

Pressure controlled: time cycled ventilation differs from pressure cycled ventilation as the rate of **flow is variable**, while the **inspiratory time is set**. The inspiratory time governs how long the pressure limit, known as pressure controlled ventilation pressure (PCVP), will be maintained. After the inspiratory time has been reached inspiration phase stops and the expiration phase begins. Hence it is pressure controlled as the pressure is "controlled" at a level for a set time and it is time cycled because "time" is what determines the duration of inspiration.

The time taken for the PCVP to be reached is able to be adjusted on some machines by manipulating the initial flow rate or rise time. On other ventilators it is possible to manipulate the maximum flow rate. This will allow the patient to generate greater flow rates, if required, during periods of potential flow starvation eg, during suctioning.

**Tidal volume is not set in pressure controlled: time cycled ventilation and will be influenced by inspiratory effort, inspiratory time, resistance to flow, and lung/thorax compliance.** By having a set inspiratory time the tidal volume is going to be less variable...
than that delivered by a simple pressure cycled ventilator. The advantage of PCV over volume cycled ventilation is that similar tidal volumes may be delivered with limited airway pressures. Furthermore there is probably less "flow starvation" experienced by the patient on assisted breaths. 18,19

It is sometimes possible to increase a patient's tidal volume by increasing the inspiratory time. Diagram 1 shows a representation of how extending the patient's inspiratory time may increase their tidal volume. Note that as a result of increasing the inspiratory time (as indicated by the dotted line) flow continues to be delivered to the patient and the tidal volume is increased. In diagram 2 however, the flow has already returned to zero before the end of the inspiratory time. By increasing the inspiratory time in this patient (as indicated by the dotted line) there is no improvement in the patient's tidal volume. All that is achieved by increasing the inspiratory time in the second patient is an inspiratory pause/hold. This may be useful for improving distribution of gases and perhaps open more alveoli but there is no increase in tidal volume.

The last diagram shows one of the benefits of pressure controlled ventilation over that of volume cycled ventilation. Notice that on the pressure tracing there is a negative pressure that indicates that this is an assisted breath, i.e., patient initiated machine breath. In this breath the patient is making a large inspiratory effort and because the flow rate in pressure controlled ventilation is variable the ventilator is able to give the patient the flow they demand. In this breath the tidal volume is greater than the previous breath due to the patient's inspiratory effort.
Question 10). What are the factors could cause the:

- high inspiratory pressure alarm
- low inspiratory pressure alarm
- low tidal volume alarm
- low minute volume alarm

to be activated in pressure controlled ventilation?

Describe appropriate action to be taken in order to rectify the problem.

Critical Care Medicine, vol. 22, no. 1, pp. 22-32.


Place a tick (✓) next to the set parameters and a cross (x) next to the variable parameters for each of the following modes of ventilation:

<table>
<thead>
<tr>
<th>Ventilator Setting</th>
<th>Pressure Controlled: Time Cycled Ventilation</th>
<th>Volume Cycled Ventilation</th>
<th>Pressure Support / CPAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>FiO₂</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PEEP/CPAP</td>
<td></td>
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</tr>
<tr>
<td>IMV Rate</td>
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<tr>
<td>Tidal Volume</td>
<td></td>
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<td></td>
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<tr>
<td>Pressure Support</td>
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<td></td>
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<tr>
<td>Peak</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flow Waveform</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sensitivity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inspiratory Hold</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inspiratory Time</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCV Pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Place a tick (✓) next to the observations that are required for each of the following modes of ventilation:

<table>
<thead>
<tr>
<th>Ventilator Observation</th>
<th>Pressure Controlled: Time Cycled Ventilation</th>
<th>Volume Cycled Ventilation</th>
<th>Pressure Support / CPAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous Exhaled Tidal Volume</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Machine Exhaled Tidal Volume</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expired Minute Volume</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rate</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>I:E ratio</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak / Plateau Pressures</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
6. **MODES OF VENTILATION**

In both volume cycled and pressure controlled: time cycled ventilation the following modes of ventilation may be available:

- **Controlled Mandatory Ventilation**
- **Synchronised Intermittent Mandatory Ventilation**
- **Assist / Control Ventilation**

### 6.1 Controlled Mandatory Ventilation (CMV)

In this mode of ventilation the operator sets a rate to a predetermined pressure, volume or time limit and the patient receives this breath in a set time interval. For example if the patient is on a rate of 10, then they will receive a breath every 6 seconds, regardless of their inspiratory effort (see following diagram). In this mode there are no spontaneous or assisted breaths.

![Diagram of CMV](image)

### 6.2 Intermittent Mandatory Ventilation (IMV)/ Synchronised Intermittent Mandatory Ventilation (SIMV).

Intermittent mandatory ventilation (IMV) was an earlier version of the more advanced SIMV. In this mode of ventilation a preset respiratory rate is delivered at a specified time interval. For a patient receiving 10 breaths per minute, a breath is delivered every six seconds regardless of the patient's efforts. The theoretical disadvantage of this form of ventilation is that the patient may take a spontaneous breath and could receive a machine delivered breath at the same time or during expiration, causing hyperinflation and high peak airway pressures. SIMV is said to avoid this problem by monitoring the patient's respiratory efforts and delivering breaths in response to the patient's inspiratory efforts.
SIMV is similar to IMV and CMV in that it will still deliver a minimum number of breaths, despite the potential lack of inspiratory effort from the patient. If the ventilator is set to deliver 10 bpm the patient will receive these breaths if they are breathing or not. SIMV utilises a window of time in which a breath is due and will look to deliver this breath within a specified time frame. If the patient makes a sufficient inspiratory effort (governed by sensitivity) the machine will sense this effort and give the patient the breath during this time, synchronised to their own effort.

NB SIMV is available in both volume cycled and pressure controlled time cycled ventilation. In the above example the theoretical decrease in peak inspiratory pressures that may occur during assisted breaths does not apply to pressure controlled time cycled ventilation, as the pressure is constant in this type of ventilation. In pressure controlled: time cycled ventilation the tidal volume may be increased during assisted breaths.
It is also important to note that during assisted breaths the patient continues to inspire even after the machine detects the patient's effort. Thus the work of breathing during assisted breaths is comparable to that of spontaneous breaths. For this reason pressure control time cycled ventilation may have greater synchrony with patients demands during assisted breaths as the patient can have as big a tidal volume or flow rate as they require.

6.3 Assist / Control Ventilation

Assist Control ventilation is available in both pressure control and volume cycled ventilation. In this form of ventilation a fixed number of breaths, with a set tidal volume or time limit, will be delivered to the patient if they are breathing spontaneously or not. If the patient makes any inspiratory effort above this number of breaths, they will receive extra breaths with that same fixed pressure or volume, ie all breaths will either be controlled (ventilator initiated) or assisted (patient initiated) with the same tidal volume (eg 500mls in volume cycled) or pressure limit and inspiratory time (eg. 30 cmsH₂O, 1.2 seconds in pressure controlled ventilation).

For Further Reading on Mechanical Ventilation refer to the following texts and articles:
- "A Clinical Guide to Cardiopulmonary Therapies" and "Pressure Controlled Ventilation", produced by Puritan Bennett
Question 10. Which of the following methods of ventilating patients would be suitable for a) a spontaneously breathing patient and b) a patient who is receiving muscle relaxants?

* SIMV
* Assist control

* CMV
* Pressure Support

Provide a rationale for your answer.
7. **IMPOSED WORK OF BREATHING.**

**Work of Breathing**

This is the work done to overcome the resistance to gas flow and to overcome the elastic work properties of the lungs and chest wall. It is a function of:

- airflow resistance
- pulmonary and chest wall compliance
- minute ventilation

The work of breathing is reflected by the oxygen cost of breathing. This is usually approximately 3% of total oxygen delivery under normal conditions but is very much greater in extreme conditions.

**Factors affecting work of breathing for intubated ventilated patients**

A) **Imposed work of breathing**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initiation of gas flow</td>
<td>Trigger sensitivity and responsiveness</td>
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<tr>
<td></td>
<td>Continuous flow circuit</td>
</tr>
<tr>
<td></td>
<td>flow triggering</td>
</tr>
<tr>
<td></td>
<td>proximal pressure measurement</td>
</tr>
<tr>
<td>Circuit resistance (including ETT)</td>
<td>titration of pressure support</td>
</tr>
<tr>
<td></td>
<td>use of appropriate size of ETT</td>
</tr>
<tr>
<td>Poor patient - ventilator synchrony</td>
<td>Titration of flow</td>
</tr>
<tr>
<td></td>
<td>Pressure support ventilation</td>
</tr>
<tr>
<td></td>
<td>pressure controlled ventilation</td>
</tr>
</tbody>
</table>

B) **Physiological work imposed by disease increases physiological work of breathing**

This component of work is reduced by the appropriate application of PEEP or CPAP

C) **Normal physiological workload.**
Attaching a patient to a breathing circuit (Ventilator, CPAP circuit, ETT, T-piece) imposes a workload on the muscles of ventilation over and above the workload required to breathe normally.

Sources Of Work Load:

1. **Ventilator with demand valve spontaneous breathing**
   - **Demand valve** - an initial pressure or flow must be generated, as a result of the work of the respiratory muscles, to trigger the machine. Triggering the machine results in gas flow.
   - **Time delay** - a time delay between the opening of the demand valve and fresh gas flow will result in a period of respiratory muscle tension without flow - therefore increased work.

2. **Ventilator and Continuous Flow CPAP Circuit.**
   - **Fresh gas flow rate** - once flow is triggered if it does not meet patient demand this will be the same as the patient's sucking on the circuit ie doing work on the circuit to obtain gas to meet demand. Patient discomfort (fighting the ventilator) results from the degree to which these problems exist in a ventilator setup
   - **Resistance and Inertia of Circuit Components**
     - narrow tubing ETT/TT
     - humidifier
     - one way valves
     - filters (especially if wet)
     - exhale valves

Further reading:

1. Ten Eyck LG, & MacIntyre NR. "Imposed work of breathing during mechanical
8. WEANING FROM MECHANICAL VENTILATORY SUPPORT

Weaning involves the transfer of the work of breathing from the ventilator to the patient. Weaning can be seen as a continuation of the process of minimising the work imposed by the breathing circuit and the work imposed by the lung pathology.

Weaning considerations

- Limiting the imposed work of breathing
- Assessing the function of respiratory centre - respiratory drive
- Assessing function of the nerves and the neuromuscular junction
- Assessing loads imposed by the chest wall and abdomen (e.g., fail segment, sternotomy, abdominal distension)
- Assessing respiratory muscle strength and stamina

<table>
<thead>
<tr>
<th>Weaning Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consideration</td>
</tr>
<tr>
<td>---------------------</td>
</tr>
<tr>
<td>Ventilatory Drive</td>
</tr>
<tr>
<td>Muscle Strength</td>
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<tr>
<td></td>
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<tr>
<td>Endurance</td>
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<td>Fatigue</td>
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</table>

8.1 Respiratory Drive (P100 or P0.1)

The P 0.1 is the most negative pressure that a patient can generate against a closed system during the first one hundred milliseconds of a spontaneous effort. The P 100 can be measured via the ventilator circuit or by an oesophageal balloon. Airway occlusion pressure has been shown to correlate with phrenic nerve activity and is considered to be a measure of central respiratory drive. The P 100 value is thus indicative of the amount of neural activity that is driving diaphragmatic movement (respiratory center drive). The normal range is 2-4 cm H₂O and a P 100 < 6 cm H₂O is an indicator of readiness for weaning. It is important to realise that the P 100 will be affected by the level of ventilatory assistance that the patient is receiving. A patient may have an adequate respiratory drive but a low P 100 due to high levels of assistance from the ventilator e.g., too much pressure support. Conversely, the P 100 could also be high due to an inadequate amount of support from the ventilator or a high resistive component, such as tube occlusion.
8.2 Maximum Inspiratory Pressure (MIP) & Negative Inspiratory Pressure (NIP)

MIP is the pressure change measured by an oesophageal balloon, that the patient can when the airway is occluded for several breaths. Cooperative patients can perform this manoeuvre in a short period of time; however many patients require longer occlusion periods in order for the neural drive to increase and produce a maximum effort. MIP is a reflection of diaphragmatic strength and may also be used to monitor respiratory muscle endurance when serial measurements are made. MIP differs slightly from negative inspiratory force, which is measured at the mouth but also reflects or measures diaphragmatic strength. The normal range for MIP is from -30 cm H₂O (low effort) to -140 cm H₂O (high effort).  

8.3 Delta Oesophageal Pressure.

This value is measured as the total downward or negative deflection in the oesophageal pressure trace during a patient generated breath beginning from the end resting oesophageal pressure. It is an indication of the pressure generated by the patient in stretching the lung and drawing gas into the lung. Although it has been described in the literature as a strength parameter, it also has endurance implications because of the work that must be performed with each patient breath. Normal range is 5-10 cm H₂O and delta pressure <15 cm H₂O is an indication of readiness for weaning. This measurement, like the P 100 should also be assessed in conjunction with the level of assistance that the patient is receiving from the ventilator.

---

Question 11). You are trying to wean your patient from the ventilator, on the following settings, when the patient becomes distressed and desatrates to 75%. The ventilator is alarming high inspiratory pressure and low tidal volume on the following settings. Describe the immediate action you would take and provide a rationale for the patient's condition.

- Volume Cycled SIMV
- Rate 8
- TV 700mls
- Pressure support 20 cms H₂O
- PEEP 10 cms H₂O
- PIP 35
- Plateau pressure 30
- High inspiratory pressure alarm set at 20 cms H₂O


9. **Patient Ventilator Synchrony**

Dysynchrony between vigorous spontaneous efforts and machine delivered breaths is often referred to as fighting the ventilator. Because increased inspiratory efforts are often followed by active expiration, discrepancies between machine and patient inspiratory time can cause peak airway pressure to increase and violate the high pressure alarm, which in turn terminates the breath. Inappropriate ventilator mode or improper selection of ventilator settings can contribute to the development of dysynchrony between the patient and mechanical ventilator. Dysynchrony may result in:

- respiratory and metabolic acidosis - due to unnecessary motor activity;
- a deterioration in gas exchange
- a compromised cardiac output - due to excessive intrathoracic pressures; and
- a prolonged weaning phase - due to the need for sedation.

As mentioned previously (see section 2 - Triggering) excessive time delays between the patient’s initial efforts to initiate a breath and the delivery of gas flow can impose a significant work of breathing. While this may be decreased with the use of flow triggering, the potential for patient ventilator dysynchrony can still occur due to improper selection of flow, volume and timing settings on the mechanical ventilator.

**Question12** Describe how the improper selection of flow, volume and timing settings might contribute to patient ventilator dysynchrony? Discuss the strategies or settings that may be implemented to decrease the risk of patient ventilator dysynchrony.
10. **Pathophysiology and Management of Acute Lung Injury**

In recent years there has been increasing interest in the methods through which positive pressure ventilation is applied. While there have been several reports within the literature pertaining to the adverse effects of high alveolar volumes and pressures, it was not until the introduction of “volutrauma” and “baby” lung concepts that the need to reduce alveolar distension became an established part of intensive care practice. The widespread acceptance of the adverse complications of high airway pressures has led to a concomitant rise in the application of pressure limited modes of ventilation and the increasing use of permissive hypercapnia. Of perhaps equal importance in the prevention of acute lung injury has been the need to utilise enough PEEP to exceed alveolar opening pressure and prevent “inflation deflation” lung injury. Most of the research regarding the application of mechanical ventilation has been based on ARDS. Following is a brief review of ARDS.

During the early phase of acute respiratory distress syndrome the lung is homogeneously (uniformly) affected by disease processes. This leads to an homogenous alteration of the vascular permeability. Consequently the oedema, present in ARDS, accumulates evenly in all lung regions and is not distributed by gravity. The increased oedema however leads to an increase in lung weight through the transmission of hydrostatic pressure, which causes collapse of the lung regions along the vertical axes. In the supine patient the collapse of lung regions occurs in the dorsal aspect of the lung. The collapse in lung regions has been confirmed by CT scans and is termed “compression atelectasis”. Due to the decreased number of alveoli participating in gas exchange there is a risk that we are over ventilating a small portion of the lung in the ARDS patient. To prevent injury to the alveoli located in the ventral lung regions (supine position) plateau pressures should be kept below 35 cms H2O. In patients with non compliant lungs or an increased resistance it may not be possible to eliminate enough CO2 and achieve normocarbia. In the absence of a severe head injury or cerebral insult it may be desirable to allow the patient’s CO2 to rise (permissive hypercapnia).

The acknowledgment of compression atelectasis has lead to the introduction of the ‘baby’ lung concept. The current hypothesis for the need to reduce the amount of ventilation that is delivered to the patient is based on the risk of over ventilating the alveoli in the ventral...
lung regions (supine patient). CT scans of patient’s with prolonged ARDS however reveals the development of cysts or bullae in the dependant lung regions. The proposed mechanisms for the development of this lung injury is thought to be the shear forces which are applied to the alveoli during repeated airway opening and collapse. One mechanism through which this form of lung injury may be prevented is through the application of a level of PEEP which exceeds airway opening pressure.

The initial pressure required to expand the alveoli is large. You will note through observation of the following diagram that on the lower part of the curve there is a large rise in pressure with little increase in volume. This rise in pressure represents airway opening. Along the vertical section of the curve, enclosed by the dotted lines, there is a large rise in volume for a small increment in pressure. This part of the curve represents the optimal area for ventilation to occur. At the top of the curve there is a large rise in pressure for a small increase in volume. This horizontal part of the curve is indicating that the lung is overdistended and has approached its total lung capacity.

The goal when adding PEEP is to detect the pressure at which alveolar opening occurs and apply sufficient PEEP to prevent airway closure. Airway opening may be assessed through the analysis of volume pressure curve on the ventilator, as indicated in the following diagram.
The titration of PEEP through observation of the inflection point is not ideally assessed from the mechanical ventilator. Ideally this inflection point should be determined through the use of a super syringe.

The Importance and Under Utilisation of Positioning as a Mechanism to Reduce Lung Injury

While there has been widespread acceptance of the need for low airway pressures, the use of permissive hypercapnia and the optimal use of PEEP there are some investigators who suggest that these therapies do not have as much supportive evidence as patient positioning in the management and prevention of acute lung injury. There are several studies and editorials in the literature that discuss the use of prone positioning in the management of acute bilateral lung injury.

Lying a patient prone will cause a more negative intrapleural pressure in the non dependant lung region which may facilitate the recruitment of alveoli, drainage of secretions, and improved gas exchange. The improvement in gas exchange between patients is variable. Where there is improvement the improvement may be short lived or continue for an extended period.

The risks to airway security must be carefully considered if a decision has been made for a trial of prone ventilation. Additionally the frequent turning of patients may prevent the consolidation of secretions and compression atelectasis associated with acute lung injury. Given the relative ease of frequent patient repositioning there is little reason why it is not part of our routine practice.

References and Further Reading - Acute Lung Injury and Prone Ventilation

34. Albert, J. 1997, “For every thing (turn…turn…turn) editorial, American Respiratory Critical Care Medicine, vol. 155, pp 393-394
Question 13): Observe the following waveforms and comment where indicated.

Mode

Problem

Solution
Mode

Problem

Solution
11. REFERENCES AND RESOURCES - MECHANICAL VENTILATION


8. Smith, R. 1992, “Positive end-expiratory pressure (PEEP) and continuous positive


25. Nilsestuen, J & Hargett, K. 1996, “Managing the patient-ventilator system using graphic analysis; an overview and introduction to graphics corner” Respiratory Care, vol. 41, no. 12, pp 1105-


34. Albert, J. 1997, “For every thing (turn...turn...turn) editorial, American Respiratory Critical Care Medicine, vol. 155, pp 393-394


12. Humidification

12.1 Introduction - Principles of Humidity and Humidification

12.1.1 What Is Humidity?

Humidity is water as vapour in a gas mixture. The amount of water present as vapour is dependent upon the gas temperature. Increasing gas temperature increases the ability of the gas to hold water. A gas has a certain capacity for holding water at a given temperature. When a gas mixture is unable to hold any more water vapour it is said to be saturated. The temperature of the gas at this state is called the dew point.

Humidity is described in terms of absolute humidity and relative humidity.

- Absolute Humidity (AH) is the actual mass of water in a volume of gas. AH is expressed as grams/cubic meter (g/m$^3$) or milligrams/Litre (mg/L).

- Relative Humidity (RH) is the actual amount of water present in a gas divided by the capacity of the gas at a given temperature ie. $\% \text{ RH} = (\text{content/capacity}) \times 100$.

Water Vapour Content Of Saturated Air (ie 100% RH)

<table>
<thead>
<tr>
<th>Temp $^\circ$C</th>
<th>AH at Saturation (mg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>12.1</td>
</tr>
<tr>
<td>20</td>
<td>17.3</td>
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<td>25</td>
<td>23.1</td>
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<td>35</td>
<td>39.6</td>
</tr>
<tr>
<td>37</td>
<td>44.0</td>
</tr>
<tr>
<td>40</td>
<td>51.2</td>
</tr>
</tbody>
</table>

Another important concept is that of the energy content of a gas mixture. The total energy content of air is made up of sensible heat and latent heat. Sensible heat is reflected by the gas temperature whilst latent heat is reflected by the water mass. Latent heat refers to the heat associated with a change of state ie. from liquid to vapour and from vapour to liquid. The change of state involves taking up of energy in the case of vaporisation or a release of energy in the case of condensation. It follows from this that humid air has more energy to give up than dry air.
12.1.2 Heat and Moisture Pathways

Normally the nose and upper respiratory tract are responsible for heating, humidifying, and filtering inspired air. This is done so effectively that by and large the heat and humidity of the gas within the lungs is in equilibrium. This occurs when the gas is at body temperature and fully saturated with water vapour at this temperature. There is thus no nett movement of heat or water between the gas and tissues. The conditions in the lung remain constant despite changes in the temperature and humidity of inspired gas as well as changes in minute ventilation and inspiratory flow rate.

Water and heat are transferred to the inspired gas as the gas passes through the upper airway. Heating and humidification of gas is complete at a point just below the carina. This point is called the \textit{isothermic saturation boundary (ISB)}. The exact point where equilibrium occurs varies under normal conditions. For instance the boundary would move more distal if large tidal volumes of dry cold air were inhaled.

Nose breathing is more effective in warming and humidifying the inspired gas than mouth breathing. In either case equilibrium will still be reached at the ISB.

\textbf{On exhalation} heat and moisture is returned progressively to the mucosa. This process results in some heat and water loss from the mucosa as the exhaled gas is generally warmer and has a greater water content than the inspired gas. The heat and moisture that is lost is replenished from systemic reserves to enable conditioning of the next breath. Refer to Figure 1.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{humidification_diagram.png}
\caption{Adapted from Jackson C. Humidification in the upper respiratory tract: a physiological overview. Intensive and Critical Care Nursing, 1996;12:27032}
\end{figure}
12.1.3 Airway Clearance and Inspired Gas Conditioning.

The airway mucosa consists of cellular, aqueous, and viscoelastic gel layers. Together these layers make up the mucociliary transport system. This system functions to remove surface liquids and particles from the lung. The cellular layer consists of cells that secrete mucous gel, secrete aqueous fluid, absorb aqueous fluid, and cells that are ciliated. The ciliated cells have cilia on their surface that beat in synchrony with the cilia of neighbouring cells. The cilia propel the gel layer forward. The gel layer floats on an aqueous layer.

The effectiveness of the mucociliary transport system depends upon maintaining an effective mucociliary transport velocity. The velocity of transport is effected by changes to the aqueous and gel layers as well as the beat frequency of the cilia. If the gel layer “dries out” then transport deteriorates. Also extremes of inspired gas temperature can reduce cilia beat frequency and thus mucociliary clearance.

“Changes in the properties of any layer have a direct effect on mucociliary function. Consequently, mucociliary transport dysfunction reflects one of the earliest changes that would occur if there were inadequate heat or moisture”(1)

Lung Mechanics and Inspired Gas Conditioning

“Inspired humidity can alter lung mechanics by directly affecting airway patency and lung compliance. Extremes of humidity can compromise airway patency by either altering the viscosity of tracheobronchial secretions, delivering excess water, slowing mucociliary clearance, or causing oedema or bronchoconstriction in asthmatic patients. Lung compliance is compromised by decreased patency, dilution of surfactant by excess water, high/low humidity causing airway oedema or bronchoconstriction, or altered airway tissue characteristics through thermal injury”(1)”

Pulmonary Lesions Associated With Inadequate Humidification (6)

- Loss of ciliary action
- Damage to mucous glands
- Disorganisation of airway epithelium
- Disorganisation of basement membranes
- Cytoplasmic and nuclear degeneration
- Cellular desquamation
- Ulceration of mucosa
- Reactive hyperaemia
12.2.2 Basic Requirements Of A Humidifier (11)

- The inspired gas is delivered into the trachea at 32-36°C with a water content of 30-43g/m³.
- The set temperature remains constant and does not fluctuate.
- Humidification and temperature remain unaffected by a large range of fresh gas flows, especially high flows.
- The device is simple to use and to service.
- Humidification can be provided for air, oxygen, or any mixture of inspired gas, including anaesthetic agents.
- The humidifier can be used with spontaneous or controlled ventilation.
- There are safety mechanisms, with alarms, against overheating, overhydration and electrocution.
- The resistance, compliance and dead-space characteristics do not adversely affect spontaneous breathing modes.
- The sterility of the inspired gas is not compromised.
12.3 Methods Of Humidification

Two methods of humidification are used in conjunction with ventilation / artificial airway management in the intensive care unit;

- Hot water humidifiers
- Heat and moisture exchangers

12.3.1 Hot Water Humidifiers

Hot water bath humidifiers such as the Fisher & Paykel humidification systems utilise a heated passover system.

“The humidifier consists of a microprocessor control unit, a water-filled humidification chamber and a delivery circuit that may be heated. The water in the humidification chamber is heated to produce a molecular vapour. Thus as the gas is passed through the chamber it is heated to the required temperature and at the same time collects the water vapour.......... The warmed, humidified gas passes to the patient along a tube which can be heated. This has the advantage of maintaining the correct gas temperature along the length of the breathing circuit, minimising condensation or “rainout”, in which bacteria can survive .......... Temperature sensors located at both the humidification chamber outlet and the patient end of the delivery circuit accurately monitor the temperature of the gas. This information is fed back to the microprocessor which automatically regulates both the heater plate and the heater wire to ensure the temperature of the gas remains at the set level” (14)

Clinical Practice Considerations

- Maintain chamber temperature at “body temperature” ie. 37°C (Ideally should be set at “core temperature)

- Set “delivered temperature” at 39 - 40°C

The aim of these settings is to provide gas that is saturated at core temperature. As the tubing is maintained at a temperature greater than chamber temperature the RH of the inspired gas will be reduced thus avoiding “rainout” and a “wet circuit”. “Wet circuits” are associated with a higher incidence of bacterial contamination. Although the inspired gas is heated to 40°C heating ceases prior to the patient Y and the gas cools down between the patient Y and the endotracheal tube. The result is that the isothermic saturation boundary is maintained at the level of the endotracheal tube ensuring that the whole of the airway distal to the ET tube is an environment in which there is no net flux of water or heat. The mucociliary transport system is maintained at an optimal level and the risk of tube occlusion due to “dry” secretions is minimised.

Movement of fluid retrogradely in the circuit should be avoided – although
• Humidifier chambers have been shown not to support the growth of bacteria as they are maintained at a high temperature and retrograde contamination has been shown to be unlikely.

• The greatest concentration of bacteria occurs closer to the ET tube and “flex” connector. Bacterial contamination of the environment (and attendant carers) can occur as a result of the “spray” that is produced when ventilator tubing is disconnected.

• The use of closed suction systems and closed and/or continuous humidifier chamber water feed sets may be helpful infection control measures.

• Continuous feed humidifier chamber water feed sets reduce nursing time required for the care of the system.

• If there is excessive rainout check temperature settings has the tube heater wire been switched off?

| Relative humidity is an important consideration - for instance if the delivered temperature is set at 37°C and the chamber temperature set well below this (say -5°C) to prevent rainout the delivered gas will have a RH of 75%. The gas will take water from the trachea and more proximal sections of the bronchi to saturate the inspired gas at body temperature. This results in greater likelihood of thick secretions and tube occlusion. |
12.3.2 Heat and Moisture Exchange Devices

These types of humidifiers contain a material from which heat and water is exchanged with the inspired and expired gas.

They are of three basic types;

- Heat and Moisture Exchanger (HME)
- Hygroscopic HME
- Hydrophobic HME

**Heat Moisture Exchangers**

These devices utilise an element that has high thermal conductivity. The element is colder than exhaled gas thus water condenses on the element and the element is warmed as a result of heat transfer from the exhaled gas to the element. The element is now warmer than the inspired gas. The gas on inspiration is warmed by heat transfer from the element and as the gas is warmed it takes up water.

These devices are generally inefficient and result in poor humidification, water and heat loss.

Hygroscopic and hydrophobic devices have been developed in order to increase the humidifying and warming capacity
Hygroscopic HME

These devices utilise an element of low thermal conductivity that has been impregnated with a water retaining (hygroscopic) compound (usually calcium chloride or lithium chloride). The element acts as a condenser humidifier but the efficiency is increased by the hygroscopic material that is able to absorb water which is then taken up by the inspired gas. (see Figure 2)

Devices of this sort are the most effective of the HMEs

Hydrophobic HME

These devices contain a material that is impervious to water. Water is retained on the element and a thermal gradient established that results in water and heat exchange. Hydrophobic humidifiers are also effective microbiologic filters.

Hybrid devices are available that combine a hygroscopic membrane and a hydrophobic membrane. These are called Heat Moisture Exchange Filters. (see Figure 2)

Overall the moisture output of these devices is variable. The composite devices (HMEF) are able to produce a water output in the range recommended for effective humidification. However the output of these devices declines with increasing tidal and minute ventilation. (see Table 1 for comparisons)

The use of Hydrophobic HME has been associated with increased incidence of tube occlusion.

Figure 2. adapted from Shelly MP. Inspired gas conditioning. Respiratory Care, 1992;37(9):1070-1080
Clinical Considerations When Using HMEs

There is an increased risk of tube occlusion when using HMEs in the setting of high tidal volumes and minute ventilation (> 10L/min)

The safety and effectiveness of HMEs has not been established for long term ventilation in ICUs

HMEs should not be used as the sole source of humidification when secretions are copious, thick, and or bloody

Where secretions are bloody there is an increased risk of tube occlusion regardless of the method of humidification

HMEs can increase the flow resistance and thus increase work of breathing

HMEs are cheaper and less labour intensive than hot water humidifiers

HMEs can be used as microbiological filters

HMEs increase mechanical dead space

Use of Nebulisers and Saline Instillation as Humidification Adjuncts

Nebulisers increase the risk of bacterial contamination and have the potential to “overhumidify”

Normal saline instillation has no place in the management of intubated patients. It does not loosen or moisten secretions nor increase sputum removal
## 12.4 Studies Evaluating Performance of Humidifiers

<table>
<thead>
<tr>
<th>Study</th>
<th>Description</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Martin (1)</td>
<td>Clinical study, prospective randomised, controlled. Compared Pall Ultipor HME with hot water humidifier. Long term ventilation.</td>
<td>6 tube occlusions in HME group (31 patients in group) with one death as a result of tube occlusion. No tube occlusions in Hot water humidifier group. Greater incidence of hypothermia on HME group. Increased number of days with thick secretions in HME group. NB. Hot water humidifier set to deliver at only 31°C</td>
</tr>
<tr>
<td>Branson (2)</td>
<td>Lung model - test bench evaluation. Tested 7 devices; Airlife Humid air (hygro) Engstrom Edith (hygro-hydro) Mallinckrodt Inline Foam Nose (hygro) Pall conserve (hydro) Portex Humid-Vent 1 (HME) Siemens Servo Humid 150 (hygro) Terumo Breathaid (hygro)</td>
<td>All humidifiers delivered greater than 21mgH₂O/L. Moisture output fell as TV increased. Hydrophobic unit (Pall) did not meet minimum standard. Recommended that use of HMEs limited to only short term duration in well hydrated, normothermic patients.</td>
</tr>
<tr>
<td>Eckerbom (3)</td>
<td>Test bench according to Draft International Standard. Tested 6 devices; Pall Ultipor Filter Mallinckrodt Inline Siemens Servo 152 Engstrom Edith Triplus Icor Portex Humid Vent 1</td>
<td>Icor, Servo, Inline, Edith - very good performance. Pall - good performance for TV up to approx. 700mL Humid Vent 1 - acceptable performance for TV up to 500mL</td>
</tr>
<tr>
<td>Stoutenbeck (4)</td>
<td>Test bench - lung model plus clinical test Compared Siemens Servo 150 (Hygro) with Portex Humid Vent (HME)</td>
<td>NB. Clinical trial only short term ventilation Hygro unit superior to non hygro unit</td>
</tr>
</tbody>
</table>

Hygro = hygroscopic
Hydro = hydrophobic
Hygro - hydro = hygroscopic + hydrophobic
HME = standard filter


Question 14) Why might heat and moisture exchangers be an acceptable means of humidification for a patient post cardiac surgery?

Question 15) Why is it generally unnecessary to humidify gas for receiving 50% oxygen via a venturi mask?

Question 16) When is it necessary to provide artificial humidification?


