Mechanical ventilation for the non-anaesthetist 2: practical tips

In the second of this pair of articles on mechanical ventilation, five cases are used to explain the initiation, benefits and potential complications of mechanical ventilation. The first article (https://doi.org/10.12968/hmed.2018.79.12.C188) explained the physiology and mechanics of mechanical ventilation. Positive pressure mechanical ventilation uses positive pressure to deliver a set pressure or a set tidal volume to the patient. Airway resistance and lung compliance influence the volumes and pressures achieved. This article explains the importance of maintaining pressures and tidal volumes within appropriate limits in order to reduce the occurrence of ventilator-induced lung injury.

Case 1: initial ventilator settings

Mr Brown is a previously fit and well 67-year-old man who was intubated in the emergency department for severe hypoxia secondary to pneumonia. His ideal bodyweight is 70 kg. He has been transferred to intensive care for mechanical ventilation.

What is the aim of mechanical ventilation?

The aim of mechanical ventilation is to keep the patient alive while doing the least harm possible. In pneumonia, inflammatory exudate in the alveoli of the affected lung reduces compliance, increasing the work of breathing. Blood flowing to poorly ventilated areas of lung creates a physiological shunt leading to hypoxaemia (Light, 1999). Mechanical ventilation reduces the work of breathing, reducing oxygen consumption and can improve oxygenation by re-opening alveoli that have closed, thereby increasing the surface area available for gas exchange. Collapsed alveoli are recruited by increasing mean alveolar pressure via:

1. Increasing the inspiratory positive airway pressure
2. Increasing the time spent in inspiration
3. Increasing the positive end-expiratory pressure.

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Which initial mode of mechanical ventilation should be selected?

Intubation requires the use of a paralysing agent to allow the endotracheal tube to be passed between the vocal cords. The paralysing agent also paralyses the muscles of respiration, leaving the patient unable to breathe independently, and thus a mandatory mode of ventilation must be used in which the ventilator delivers a set number of breaths per minute. In pressure control ventilation, the inspiratory positive airway pressure is set. In volume control ventilation the tidal volume is set. The decision to use volume or pressure control ventilation is often unit and clinician specific (Esteban et al, 2000).

What initial settings should be used?

Mechanical ventilation can itself induce or worsen lung injury, known as ventilator-induced lung injury. High pressure in the alveoli can cause barotrauma, lung overdistension volutrauma and the shear stress of repetitive opening and closing of alveoli, atelectotrauma (Tremblay and Slutsky, 1998). All of these can lead to biotrauma, in which inflammatory mediators spread from the damaged lungs to distant organs, causing multi-organ dysfunction (Slutsky and Ranieri, 2013). Selecting appropriate ventilator settings, known as lung protective ventilation, can reduce the occurrence of ventilator-induced lung injury.

In pressure control ventilation, the pressure delivered during inspiration (inspiratory positive airway pressure) is set. In this case, a starting inspiratory positive airway pressure of 14 cmH_2O case could be trialled. The tidal volume achieved at this pressure depends on airway resistance and lung or chest wall compliance. Too low a tidal volume will lead to inadequate gas exchange, too high a tidal volume will damage delicate alveoli. Studies have shown a target tidal volume of ≤6 ml/kg of ideal bodyweight reduces mortality in mechanically ventilated patients (Brower et al, 2000; Serpa Neto et al, 2012, 2015). Therefore an appropriate target tidal volume in Mr Brown's case would be ≤420 ml (70 kg x 6 ml/kg).

In volume controlled ventilation, the tidal volume is set (420 ml). The pressures required to achieve such tidal volumes must be closely monitored, as high pressures (particularly >30 cmH_2O) can lead to barotrauma (Howell and Davis, 2018).

Positive end-expiratory pressure helps prevent airway collapse during expiration and increases mean alveolar pressure, thereby increasing oxygenation. A minimum positive end-expiratory pressure of 5 cmH_2O is usually set.

The inspired fraction of oxygen (FiO_2) should initially be set to 1 and then reduced once the patient's condition has stabilized. Both hypoxia and hyperoxia (oxygen saturations above 98%) should be avoided. An arterial blood gas should be obtained to guide necessary changes in ventilator settings.

Carbon dioxide elimination is directly proportional to alveolar minute volume, which is respiratory rate multiplied by alveolar tidal volume. The appropriate minute volume is one which maintains adequate acid–base status. If the respiratory rate is set too low, then minute volume and thus carbon dioxide elimination are inadequate, carbon dioxide levels subsequently rise, reducing pH. In mandatory ventilation, the appropriate set respiratory rate is one that maintains adequate acid–base status and is usually 12–25 breaths per minute.

The following initial settings are selected for Mr Brown:

Pressure control ventilation with an inspiratory positive airway pressure of

14 cmH_2O and a positive end-expiratory pressure of 5 cmH_2O. These pressures achieve tidal volumes of 380–400 ml. Respiratory rate is set at 20 breaths per minute and inspiratory:expiratory ratio (I:E) is 1:2. The FiO_2 is reduced from 1.0 to 0.5 and oxygen saturations of 93% are achieved. An arterial blood gas shows pH 7.37, partial pressure of oxygen (PaO_2) 9.1 kPa, partial pressure of carbon dioxide ($PaCO_2$) 4.7 kPa, bicarbonate (HCO_3-) 24 mEq/litre.

Case 2: acute respiratory distress syndrome

As the paralysing agent wears off, Mr Brown begins to trigger his own breaths and he is switched to a synchronised intermittent mandatory ventilation mode with the following settings: inspiratory positive airway pressure 14 cmH_2O, positive end-expiratory pressure 5 cmH_2O, respiratory rate 16 breaths per minute, I:E 1:2, FiO_2 0.5. Four hours later, Mr Brown's nurse calls to say he is desaturating and that she has had to increase FiO_2 to 0.7 to maintain oxygen saturations of >90%. An arterial blood gas shows pH 7.35, PaO_2 7.1 kPa, $PaCO_2$ 5 kPa, HCO_3- 24 mEq/litre. Bilateral crepitations can be heard on auscultation and a chest X-ray shows new diffuse bilateral opacities. An echocardiogram obtained a few hours before was normal, with a left ventricular ejection fraction of 60%.

The intensive care consultant diagnoses acute respiratory distress syndrome – an acute and diffuse inflammatory lung injury which affects approximately 25% of mechanically ventilated patients and has a mortality of 30–40% (Bellani et al, 2016). Acute respiratory distress syndrome is defined as acute hypoxaemia (defined as PaO_2:FiO_2 ratio of ≤300 mmHg or ≤40 kPa) with a minimum positive end-expiratory pressure of 5 cmH_2O, plus the presence of bilateral lung opacities on radiographic imaging not entirely explained by heart failure or fluid overload (Ranieri et al, 2012).

In acute respiratory distress syndrome, the permeability of the pulmonary vasculature increases, with fluid and plasma proteins leaking into the alveoli and interstitial tissues ('non-cardiogenic pulmonary oedema') creating a diffusion barrier. The increase in total lung water reduces compliance. Plasma proteins denature surfactant leading to alveolar collapse and shunting (Ranieri et al, 2012). The result is acute hypoxaemia.

How can oxygenation be increased now?

The aim is to improve oxygenation without causing further damage to the lungs. The difficulty in acute respiratory distress syndrome is that some alveoli are normal while others are stiff and collapsed. The aim is to try and open collapsed alveoli without damaging the normal areas. To recruit alveoli the mean alveolar pressure can be increased through increasing inspiratory positive airway pressure, positive end-expiratory pressure or the time spent in inspiration.

The inspiratory positive airway pressure is currently set at 14 cmH_2O, achieving tidal volumes of approximately 400 ml. Mr Brown's target tidal volume is ≤420 ml (≤6 ml/kg ideal bodyweight). A landmark study showed a 22% relative reduction in mortality in patients with acute respiratory distress syndrome when low tidal volumes of 6 ml/kg were used compared to 12 ml/kg (Brower et al, 2000). Increasing inspiratory positive airway pressure here may result in tidal volumes over the target 6 ml/kg.

The current positive end-expiratory pressure of 5 cmH_2O is low and could be increased. Increasing positive end-expiratory pressure can improve oxygenation by recruiting alveoli and putting the lung on the steeper part of the compliance curve. In patients undergoing low tidal volume of acute respiratory distress syndrome, positive end-expiratory pressure can be increased to 20 cmH_2O (Brower et al, 2000). It is important to ensure patients are adequately fluid resuscitated before increasing positive end-expiratory pressure, as high positive pressures in the thorax can reduce venous return to the right side of the heart, reducing pre-load and thus cardiac output (Schmitt et al, 2001). High positive end-expiratory pressure can also lead to barotrauma.

Another option to improve oxygenation is to increase the time spent in inspiration. Normally, twice as long is spent in expiration than inspiration giving an I:E ratio of 1:2. Increasing the I:E ratio to 1:1 increases the proportion of time spent in inspiration, allowing more time for poorly compliant lung units to be ventilated.

If achieving sufficient oxygenation continues to be difficult, paralysing agents can be used to paralyse the muscles of respiration thereby reducing oxygen consumption and any patient–ventilator asynchrony (Papazian et al, 2010). Turning the patient over to the prone position can help improve oxygenation by decreasing ventilation–perfusion mismatch, but is not without risks (Guérin et al, 2013).

Case 3: high peak pressures

Over the next 3 days, Mr Brown progresses well although the nurse reports thick secretions on endotracheal suctioning. The patient is sedated for endotracheal tube tolerance, but is breathing spontaneously with a respiratory rate of 18 breaths per minute. A pressure support mode of ventilation is being used, with an inspiratory positive airway pressure of 10 cmH_2O and a positive end-expiratory pressure of 5 cmH_2O, FiO_2 0.4. Suddenly the peak inspiratory pressure alarms showing pressures of 35 cmH_2O.

How should this patient be assessed to determine the cause?

The highest level of pressure measured by the ventilator during inspiration is the peak inspiratory pressure. A high inspiratory pressure can be caused by a problem with the ventilator, the patient or the tubing connecting the two. A systematic approach is important to determine the cause.

Tubing and ventilator problems

The tubing connecting the patient and the ventilator should be checked for kinks and fluid causing increased resistance. The endotracheal or tracheostomy tube should be reviewed for blockages or displacement. Disconnecting the patient from the ventilator and manually ventilating the patient eliminates any ventilator issues and provides tactile information as to how easy or difficult the patient is to ventilate.

Patient problems

Patient causes of high inspiratory pressures can be divided into increased airway resistance, or reduced lung or chest wall compliance. The main causes are shown in *Table 1*. Clinical examination is key, with a portable chest X-ray for confirmation if time allows. Looking at the expiratory flow and performing an inspiratory hold can also provide useful information.

Plateau pressure

High pressures in the alveoli causes barotrauma. The peak inspiratory pressure is the pressure in the airways and alveolar pressure combined, as shown in *Figure 1*. To obtain an estimate of the alveolar pressure, known as the plateau pressure, a manoeuvre

Table 1. Causes of high inspiratory pressures with suggested examination findings and possible treatments

Increased airway resistance	Bronchospasm – listen for wheeze, treat with bronchodilators
	Thick airway secretions – reduced air entry, treat with suctioning, saline nebulisers, chest physiotherapy
Decreased compliance	Pulmonary oedema – listen for crepitations, assess fluid balance, treat with diuretics
	Consolidation – crackles, lobar shadowing on chest X-ray, treat with antibiotics
	Lobar collapse – reduced air entry, treat with antibiotics, chest physiotherapy, bronchoscopy
	Pneumothorax – reduced air entry, treat with chest drain
	Pleural effusion – dull lung base, treat with diuretics and/or chest drain
	Abdominal distension splinting diaphragm – abdominal examination, nasogastric tube insertion, consider draining ascites if present

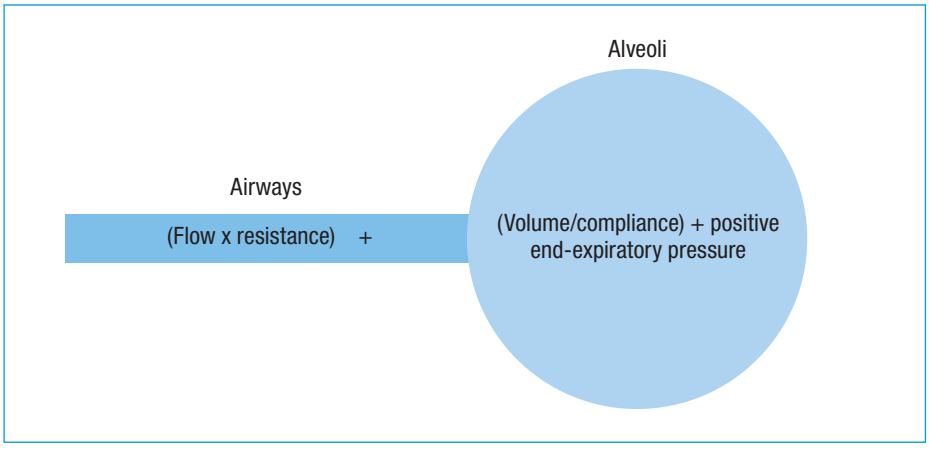


Figure 1. Pictorial representation of the inspiratory pressure generated in the airways and the alveoli during positive pressure ventilation.

called an inspiratory hold is performed. In volume control ventilation, flow is paused for 0.5–1 second (*Figure 2*). As airway pressure equals flow multiplied by resistance, if flow is paused and therefore zero, the airway pressure will be zero. The remaining plateau pressure is an estimate of alveolar pressure at end-inspiration.

High alveolar pressures can lead to overdistension and rupture of alveoli, known as barotrauma (Ioannidis et al, 2015). To reduce the risk of barotrauma, plateau pressures should not exceed 30 cmH_2O (Howell and Davis, 2018). Plateau pressure measurements can also help differentiate between resistance and lung or chest wall compliance issues. Increased airway resistance (e.g. from kinking of the endotracheal tube) will increase peak inspiratory pressure but not plateau pressure. Decreased lung or chest wall compliance will increase both peak inspiratory pressure and plateau pressure.

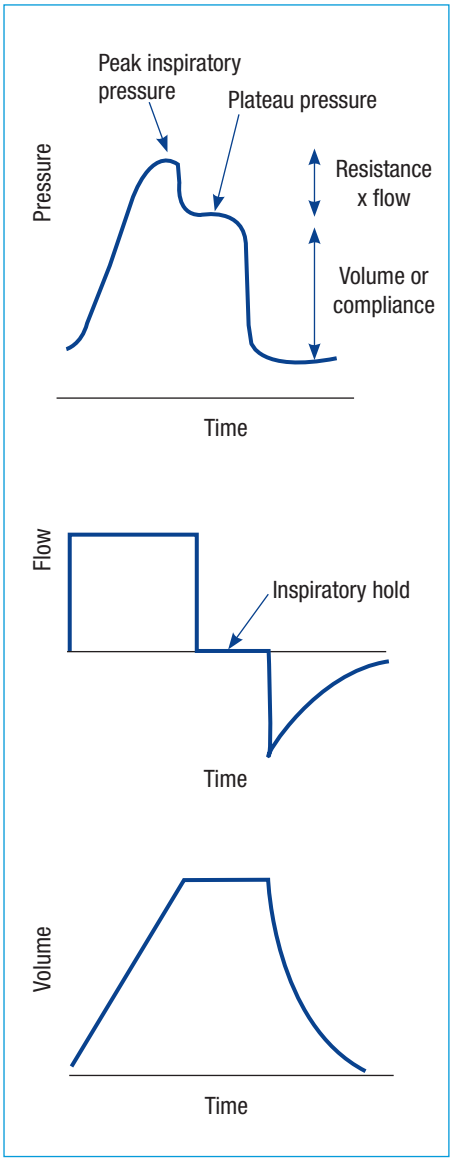
Driving pressure

Driving pressure is the difference between plateau pressure and positive end-expiratory pressure, e.g. a plateau pressure of 22 cmH_2O and a positive end-expiratory pressure of 10 cmH_2O gives a driving pressure of 12 cmH_2O. Driving pressure describes the pressure gradient which generates air flow into the lungs. High driving pressures (above 15 cmH_2O) increase ventilator-associated lung injury (Bugedo et al, 2017). Reduced driving pressures have been associated with improved survival (Amato et al, 2015).

Expiratory flow

Looking at expiratory flow during positive pressure ventilation can help differentiate between airway resistance and compliance issues. High airway resistance impedes both inspiration and expiration so flow out is very slow. Low compliance impedes inspiration but does not impede expiration so flow out is quick and normal.

Figure 2. Pressure, flow and volume graphs during an inspiratory hold to obtain a plateau pressure measurement.



Mr Brown

The doctor attending Mr Brown notes reduced air entry bilaterally on auscultation. While the peak inspiratory pressure is 35 cmH_2O, the plateau pressure is only 8 cmH_2O, indicating a resistance issue. On suctioning, thick secretions are removed from the endotracheal tube and the peak inspiratory pressure returns to normal. The impression is of a partially blocked endotracheal tube, leading to increased airway resistance with high peak inspiratory pressures and normal plateau pressures.

Case 4: intrinsic positive end-expiratory pressure

Mrs Smith is a 63-year-old woman who was intubated and ventilated for a severe exacerbation of chronic obstructive pulmonary disease. The intensive care consultant has set the respiratory rate to 30 breaths per minute in an attempt to increase carbon dioxide elimination. An hour later, her blood pressure drops from 140/80 mmHg to 90/50 mmHg. On examination she has a prolonged expiratory wheeze. A chest X-ray shows hyperinflated lung fields.

What has caused the drop in blood pressure?

The differential diagnosis for Mrs Smith's hypotension is broad, including tension pneumothorax, hypoxia from a displaced endotracheal tube, hypovolaemia and high positive end-expiratory pressure. A prompt examination is required to determine the underlying cause.

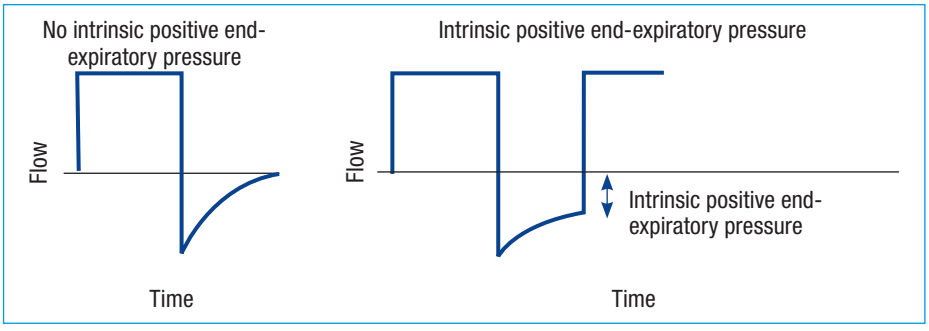
The intensive care doctor notices that the flow graph is not returning to zero, indicating intrinsic positive end-expiratory pressure.

The high respiratory rate has left insufficient time for expiration. If the patient is not given enough time to exhale all the tidal volume, flow does not return to baseline before the next breath starts and air becomes trapped, known as intrinsic positive end-expiratory pressure or auto positive end-expiratory pressure (Blanch et al, 2005). High positive pressure in the thorax reduces venous return to the right side of the heart (Magder, 2011). This decreases pre-load which reduces cardiac output and thus blood pressure.

The flow and volume graphs displayed on the ventilator should always return to baseline by the end of expiration, as shown in *Figure 3*. Intrinsic positive end-expiratory pressure can be measured using a static measurement known as an expiratory hold, in which flow is paused at the end of expiration.

Patients with bronchoconstriction in particular need sufficient time to exhale. In mechanical ventilation this can be achieved by decreasing the I:E ratio (e.g. from 1:2 to 1:3), so proportionally longer is spent in expiration. Reducing the respiratory rate can also allow more time for expiration. Reducing the tidal volume reduces the volume that needs to be exhaled. Bronchodilators and steroids should also be used to treat the underlying bronchoconstriction and inflammation.

Figure 3. Flow over time graph showing intrinsic positive end-expiratory pressure.



Case 5: ventilator-associated pneumonia

Two days later, Mrs Smith's C-reactive protein level jumps from 36 to 131 mg/litre and she spikes two fevers of 38°C. A chest X-ray shows new right lower lobe shadowing and ventilator-associated pneumonia is diagnosed. Sputum samples are sent and antibiotics started.

Endotracheal and tracheostomy tubes allow microorganisms to bypass the normal nasopharyngeal and oropharyngeal defence mechanisms, greatly increasing the risk of ventilator-associated pneumonia. Around 10–20% of patients receiving over 48 hours of mechanical ventilation will develop ventilator-associated pneumonia and these patients are twice as likely to die than patients without ventilator-associated pneumonia (Safdar et al, 2005). The incidence of ventilator-associated pneumonia can be reduced using bundles of care, in which evidence-based practices are implemented together to improve outcomes (Hellyer et al, 2016). These include elevating the head of the bed to 30° and daily sedation interruption and assessment of readiness to extubate (Hellyer et al, 2016).

Assessing a mechanically ventilated patient on the ward round

Table 2 provides a systematic approach to assessing breathing in a mechanically ventilated patient. Assessment of breathing should be followed by a full examination, review of drug chart and relevant imaging, with thorough documentation of findings and management plan.

Conclusions

Mechanical ventilation is a life-saving treatment, with the potential to cause great harm. Ventilator-induced lung injury can be reduced by using target tidal volumes ≤6 ml/kg of ideal body weight, driving pressures ≤15 cmH_2O and plateau pressures ≤30 cmH_2O. Other complications of positive pressure ventilation include reduced cardiac output and ventilator-associated pneumonia. It is important for doctors working in intensive care to understand the basics of mechanical ventilation in order to adequately assess patients and to troubleshoot should problems arise. **BJHM**

Conflict of interest: none.

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KEY POINTS

- Peak inspiratory pressure is the highest pressure measured during inspiration and increases with high airway resistance and/or low lung compliance.
- Plateau pressure is an estimate of the pressure in the alveoli and should not exceed 30 cmH_2O.
- Driving pressure is the difference between plateau pressure and positive end-expiratory pressure and should be kept below 15 cmH_2O.
- High positive end-expiratory pressure can reduce cardiac output.
- The incidence of ventilator-associated pneumonia can be reduced using care bundles.

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Table 2. A systematic approach to assessing breathing in a mechanically ventilated patient

Airway	Assess the patency of the airway (endotracheal tube, tracheostomy)	
Ventilator mode	Which mode of ventilation is being used? Mandatory or spontaneous? Set volume or set pressure?	
	If set pressure mode of ventilation	Note the set inspiratory positive airway pressure and the positive end-expiratory pressure
		What tidal volumes are being achieved with these pressures?
		The target tidal volume is normally ≤6 ml/kg (to avoid volutrauma)
	If set volume mode of ventilation	What inspiratory pressures are required to achieve the set volume?
		Avoid plateau pressures >30 cmH_2O (to avoid barotrauma)
Oxygenation	What is the fraction of inspired oxygen? What is the oxygenation saturation?	
	Review the latest arterial blood gases, note the trend	
	Aim for an arterial partial pressure of oxygen ≥8.0 kPa	
	If arterial partial pressure of oxygen is <8 kPa, investigate and treat the underlying cause. Increase the fraction of inspired oxygen or the mean alveolar pressure by increasing inspiratory positive airway pressure, tidal volume, inspiratory time or positive end-expiratory pressure	
Carbon dioxide	What is the arterial partial pressure of carbon dioxide?	
	An arterial partial pressure of carbon dioxide of over 8 kPa is permitted in some patients if pH is >7.1 (known as permissive hypercapnia)	
	If arterial partial pressure of carbon dioxide is rising, increase tidal volume or time spent in expiration	
Clinical examination of the respiratory system	Does the patient look comfortable on the ventilator? Ventilator–patient asynchrony can be caused by pain, anxiety or inappropriate ventilator settings	
	Note the volume, colour and consistency of respiratory secretions	
	Auscultate and percuss the chest	
Cardiovascular system	High intra-thoracic pressures generated during mechanical ventilation can reduce pre-load and thus cardiac output	
	Oxygen delivery is dependent on cardiac output, haemoglobin concentration and arterial oxygen saturation. These must be optimized to improve oxygen delivery	