

Principle of Mechanical ventilation for non-critical care nurses

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This article aims to introduce non-critical care nurses to the principles of mechanical ventilation.

Learning Outcomes

1. To understand the different types of respiratory failure and the differences between them
2. To appreciate respiratory pathophysiology
3. To be introduced to mechanical ventilation principles, ventilator modes, ventilator settings, and risks of artificial ventilation, and weaning
4. To explore and develop knowledge for underpinning evidence base principles of mechanical ventilation using national guidelines and care bundles.

Ventilation is the process by which gases move in and out of the lungs (Woodrow, 2019). In critically unwell patients, self-ventilation may become inadequate. Artificial ventilation may be required when self-ventilation is, or is likely to be, inadequate due to respiratory or neurological distress causing loss of consciousness; the use of recreational drugs, sedatives; opioid medications; head injury and neurological disorders.

Respiratory failure

There are two types of respiratory failure (O'Driscoll et al, 2017, for the British Thoracic Society):

- Type 1: oxygenation failure—hypoxia ($\text{PaO}_2 < 8 \text{ kPa}$) with normocapnia (normal CO_2) or hypocapnia (low CO_2 as a result of increased respiratory rate)
- Type 2: ventilatory failure—hypoxia ($\text{PaO}_2 < 8 \text{ kPa}$) with hypercapnia ($\text{PaCO}_2 > 6 \text{ kPa}$).

Gas exchange in the lungs is determined by three factors (Woodrow, 2019):

- Ventilation (V): breath size
- Perfusion (Q): pulmonary blood flow
- Diffusion: movement of gases across the tissue between pulmonary blood and alveolar air.

Pathophysiology

The main pathophysiological mechanisms of respiratory failure are hypoventilation and ventilation/perfusion (V/Q) mismatch (Shebl and Burns, 2019):

Hypoventilation

Hypoventilation is a condition that arises when air entering the alveoli is reduced. This causes levels of O₂ to decrease and the levels of CO₂ to increase. Hypoventilation may occur when breathing is too slow or shallow and is usually a consequence of other medical conditions, such as neuromuscular disorders, head injury, chest wall abnormalities, obesity hypoventilation, and chronic obstructive pulmonary disease (Fayyaz and Lessnau, 2018). It can also be caused by medications, such as sedatives, opioid-based analgesia, and substances that depress brain function, eg alcohol and recreational drugs.

Hypoventilation leads to an abnormal retention of CO₂ in the blood due to poor gas exchange within the lungs. There can be a variety of underlying reasons for the poor exchange of CO₂, but the result is higher volumes of CO₂ (hypercapnia), which displaces and lowers the volume of O₂ carried in the blood (hypoxaemia) (Woodrow, 2019). The abnormal retention of CO₂ in the bloodstream is significant because it can lead to respiratory acidosis, whereby the pH level of the blood is raised making it too acidic. Consequently, cellular respiration is disrupted and, in extreme cases, this will lead to type 2 respiratory failure (O'Driscoll et al, 2017).

Ventilation/perfusion mismatch

V/Q mismatch is the most common cause of hypoxaemia.

Shunt

Shunt or pulmonary shunt is one of the two contributors to V/Q mismatch (Woodrow, 2019). Generally, pulmonary shunt can occur in two ways: anatomical shunt and capillary shunt. Anatomical shunt happens when arterial blood returns to the pulmonary veins without passing through the pulmonary capillaries. A capillary shunt occurs when the blood passes through the capillaries of alveoli without ventilation. Therefore, it decreases the oxygenation of the deoxygenated blood (Whitten, 2017). This poorly oxygenated blood subsequently returns to the heart, where it mixes with blood that has been oxygenated in well-ventilated areas of the lungs.

Dead space

Dead space, total dead space or physiologic dead space is the second contributor to V/Q mismatch (Woodrow, 2019). Generally, it has two components: anatomical dead space and alveolar dead space. Typically, anatomical dead space is the portion of the air that remains in the conducting airways where no gas exchange is possible. In comparison, alveolar dead space is the condition that results when the alveoli of the lungs have adequate ventilation and inadequate perfusion (Whitten, 2017).

Mechanical ventilation

A mechanical ventilator is a machine that assists a patient to breathe (ventilate) when they are unable to breathe on their own (*Figure 1*). The ventilator blows gas (air plus oxygen, as needed) into a patient's lungs. It can help by doing all the patient's breathing for them or by assisting their breathing.

Figure 1. Patient on ventilator

The patient is connected to a ventilator via a endotracheal tube (ETT), which is a flexible plastic tube inserted through the mouth or nose into the trachea, enabling

oxygen to be delivered to the lungs. An ETT—or artificial airway—is necessary when a patient is unable to breathe independently; when it is necessary to sedate and ‘rest’ someone who is critically unwell; or to protect the airway due to reduced consciousness (Eldridge and Paul, 2020). It maintains the airway so that O₂ can be inhaled into lungs, and CO₂ can be expelled from the lungs. It also offers access to the lower airways for suctioning and removal of secretions.

The ventilator can deliver higher concentrations of O₂ than that delivered by a mask or other devices. The machine can also provide what is called positive end-expiratory pressure (PEEP). This helps to hold the alveolar open preventing them from collapse at the end of expiration.

Mechanical ventilation is one of the most common interventions in intensive care (Scottish Intensive Care Society (SICS), 2020)—it is often lifesaving, but can have life-threatening physiological and psychological side-effects (see Risks of mechanical ventilation section, p473). The main purpose of ventilation is to provide life support without causing harm to the patient and, for this, a sound knowledge of the underlying principles of mechanical ventilation is essential for its effective and safe implementation. The information required can be obscured by the confusing terminology attached to the various modes of ventilation.

Anaesthetists, intensivists and advanced critical care practitioners (ACCPs) are expert airway clinicians. The procedure of having the tube inserted is commonly referred to as ‘intubation’. Some people undergo surgery to have an incision in their neck through which a tracheostomy tube is inserted into the trachea—this is called a stoma. ETTs and tracheotomy tubes can remain in place for as long as needed but should be changed if they become blocked or if they leak, as per local trust policy and in accordance with the manufacturer's guidelines. Sometimes, a patient can talk with a tracheostomy tube in place by using a special adapter called a speaking valve.

Patients remain on mechanical ventilation until their condition improves enough for them to breathe on their own or end-of-life care decisions are made.

Artificial ventilation

Oxygenation relies on functional alveolar surface area and therefore is determined by:

- Mean airway pressure
- Inspiration time
- Positive end-expiratory pressure (PEEP)
- Fraction of inspired oxygen (FiO₂) and pulmonary blood flow.

CO₂ removal requires active tidal ventilation and therefore is affected by:

- Tidal volume
- Expiratory time
- Frequency and flow of breath
- Resistance to expiration (gas trapping).

Modes of ventilation

Historically, positive pressure ventilators were classified by their cycles as follows (Woodrow, 2019):

- Time (controlled by rate or inspiration: expiration (I:E) ratio)
- Volume (delivers gas until preset tidal volume is reached)
- Pressure (delivers gas until preset airway pressure is reached)
- Flow (speed in litres per minute at which the ventilator delivers breaths).

Patients at greatest risk from alveolar trauma usually have poor lung compliance, low functional lung volumes and hypoxia (Woodrow, 2019). The Acute Respiratory Distress Syndrome Network (ARDSNet) (2008) recommends an initial tidal volume of 8 ml/kg of ideal body weight, reducing quickly to 6 ml/kg, which may necessitate accepting permissive hypercapnia. Permissive hypercapnia is a ventilation strategy to allow for an unphysiologically high partial pressure of carbon dioxide (PCO₂) to permit lung protective ventilation with low tidal volumes (Fuchs et al, 2017).

Ventilator settings

Minute ventilation (Vm)

Most modern ventilators will use respiratory rate (RR) and tidal volume (Vt) to work out a set minute volume. Many basic mechanical ventilators and transport ventilators may only use minute volume as a key setting and not have a Vt or RR setting. This means that the clinician responsible for the mechanical ventilator would need to calculate an acceptable minute volume number. Remember that it is recommended to initiate ventilation at between 6–8 ml/kg of ideal body weight, and that a normal respiratory rate in adults is 12–20 breaths per minute (bpm) (Royal College of Physicians, 2017).

Minute ventilation = tidal volume × respiratory rate (ie 500 ml × 12 bpm = a minute volume setting of 6).

Fraction of inspired oxygen

Fraction of inspired oxygen (FiO₂) is the percentage of O₂ delivered to the patient. The FiO₂ ranges from 0.21 (room air) to 1 (100% O₂). FiO₂ will be increased and decreased depending on the patient's arterial blood gas (ABG), aiming for a normal reference range ie pH: 7.35–7.45, PCO₂: 4.6–6.4 kPa, PO₂: 11.0–14.4 kPa.

Positive end-expiratory pressure

PEEP is the amount of pressure in the breathing circuit at the end of exhalation. The initial PEEP for patients admitted to ICU is usually between 5 cm and 10 cm H₂O (SICS, 2020). According to Elliott and Elliott (2018), normal physiological breathing prevents the alveoli from completely collapsing at the end of expiration because the epiglottis closes the airway, leaving a residual volume of air in the lungs. This is not possible if the larynx is permanently open because of the presence of an endotracheal or tracheostomy tube.

The term 'open lung' is used to refer to the lungs being directly open to the atmosphere by a tube. PEEP ensures that a certain amount of pressure remains in the lungs throughout the entire ventilatory cycle. Usually, PEEP will be at least 5 cm H₂O,

increasing depending on the patient's physiology, for example stiffer lungs may require an increase in PEEP. However, in prolonged ventilation, alveoli may continue to collapse as a result of several physiological and mechanical processes, such as lack of surfactant, sputum retention and endotracheal suctioning. In this case, PEEP should be intermittently increased to higher levels to enable the alveoli to reopen. This is known as a lung recruitment manoeuvre.

The non-invasive equivalent of PEEP is continuous positive airway pressure (CPAP). Delivering CPAP requires an inward flow of air against exhalation, which can be uncomfortable for patients. Patients who are critically unwell with deteriorating respiratory failure will require either non-invasive ventilation (NIV), such as facial CPAP or facial BiLevel, or invasive ventilation via an ETT tube attached to mechanical ventilation. [Table 1](#) outlines the positive and negative issues associated with PEEP.

Table 1. Positive end-expiratory pressure (PEEP)

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Positives	Negatives
Prevents atelectasis	Can cause barotrauma
Recruits collapsed alveoli	Gas trapping and hypercapnia
Facilitates oxygen exchange during expiratory pause, so improving oxygenation	Reduces venous return (increasing cardiac workload)
	Increased work of breathing on self-ventilating modes by increasing resistance to expiration

High PEEP (>10 cm H₂O) may increase extravascular lung water and increase pulmonary oedema (Woodrow, 2019). Therefore, in conditions such as acute respiratory distress syndrome (ARDS), PEEP should remain at a minimum 5 cm H₂O if possible (ARDSNet, 2008); however, with increased oxygen demand and increased FiO₂, it is likely that PEEP will be increased.

Volume control

This refers to modes of ventilation where the volume of the tidal breath is set; for example, setting the ventilator to deliver a breath of 500 ml. Setting the rate is normally mandatory for this method of controlling ventilation, eg setting the V_t at 500 ml to be delivered at a rate of 14 bpm. The ventilator in this example is set to deliver a minute

volume of $500 \times 14 = 7$ L/minute. The airway pressure in this mode of ventilation will depend on lung and chest wall compliance. Indeed, the airway pressure might change from breath to breath and will depend on many factors.

Pressure control

Refers to modes of ventilation where the pressure of the tidal breath is set, eg the ventilator is set to deliver a breath pressure of 30 cm H₂O. Setting the rate is normally mandatory for this method of controlling ventilation. For example, the set breath pressure may be at 30 cm H₂O to be delivered at a rate of 14 bpm. The tidal volume in this mode of ventilation will depend on lung and chest wall compliance. Indeed, tidal volume might change from breath to breath, and will depend on many factors.

Inspiratory:expiratory ratio

The inspiratory:expiratory (I:E) ratio refers to the ratio of inspiratory time:expiratory time. In normal spontaneous breathing the expiratory time is about twice the duration of the inspiratory time. This gives an I:E ratio of 1:2 and is read 'one to two'. This ratio is typically changed in patients with asthma due to the prolonged time of expiration. Patients might have an I:E ratio of 1:3 or 1:4. Even longer expiratory times are required sometimes for patients who are in type 2 respiratory failure to expel excessive build-up of PCO₂, but this must be set by an experienced clinician.

Inverse ratio

An inverse ratio refers to when the I:E ratio is 2:1 or higher and is typically used to ventilate non-compliant lungs. This requires expertise. Nurses must consult a senior clinician if they are unsure of what to do, and only work within their scope of clinical practice (Nursing and Midwifery Council, 2018). Pressure control modes of ventilation should be used when employing inverse ratios as the use of volume control modes might lead to 'breath dyssynchrony stacking' (BDS) and an increase in airway pressure.

BDS refers to the unintended high tidal volumes that occur due to incomplete exhalation between consecutive inspiratory cycles delivered by the ventilator (European Society of Intensive Care Medicine, 2020). This can commonly occur

during volume-preset assist control modes during lung protective ventilation for ARDS. BDS typically occurs during either double or reverse triggering in assist control modes. Double-triggering refers to a sustained inspiratory effort that lasts longer than the patient-triggered respiratory cycle. This results in triggering the subsequent inspiratory cycle with incomplete interval exhalation. Reverse-triggering occurs when rhythmic passive ventilator-induced insufflations result in diaphragm entrainment out of phase with the machine. The patient's inspiratory muscle effort determines the number and timing of ventilator inspiratory cycles, allowing the patient to determine the true tidal volume delivered, irrespective of the assist mode used.

Work of breathing

The Medical Dictionary for the Health Professions and Nursing (2012) defines the work of breathing as the total expenditure of energy necessary to accomplish the act of breathing. It may be calculated in terms of the pulmonary pressure multiplied by the change in pulmonary volume, or in terms of the O₂ cost of breathing (ie O₂ consumption above basal metabolic O₂ use attributable to breathing). It is determined by lung and thoracic compliance, airway resistance, and the use of accessory muscles for inspiration or forced expiration. It is measured in joules/L and joules/min. The measurement of the work of breathing is analogous to the typical description of work in physics (work = force x distance). In respiratory physiology, work = pressure x volume.

Synchronisation

Synchronisation is a feature of some modes of ventilation that allow the tidal volume to be delivered to be synchronised with the patient's breathing. Different ventilators do this differently; consult the specific ventilator manual for a detailed description of the synchronisation method used.

Humidification

Restrepo and Walsh (2012) identified that, when the upper airway is bypassed during invasive mechanical ventilation, humidification is necessary to prevent hypothermia, disruption of the airway epithelium, bronchospasm, atelectasis, and airway

obstruction. In severe cases, inspissation of airway secretions may cause occlusion of the endotracheal tube. While there is no clear consensus on whether or not additional heat and humidity are always necessary when the upper airway is not bypassed, such as in NIV, humidification is highly suggested to improve comfort (Woodrow, 2019).

The upper airway provides 75% of the heat and moisture supplied to the alveoli. When bypassed, a humidifier is required to supply this missing heat and moisture. There are two main systems for warming and humidifying gases delivered to mechanically ventilated patients: active humidification through a heated humidifier (HH), and passive humidification through a heat and moisture exchanger (HME).

There are various types of HME filter (or artificial nose): hydrophobic, hygroscopic and filtered. Heated humidifiers operate actively to increase the heat and water vapour content of inspired gas. HMEs operate passively by storing heat and moisture from the patient's exhaled gas and releasing it to the inhaled gas.

Prone positioning

The Faculty of Intensive Care Medicine (FICM) (2019) notes that there is currently a lack of evidence for an optimal method of proning a patient (nursing them face down). The following recommendations are therefore based on common themes that appear in the literature and intend to provide an example of safe and effective practice.

Indications (FICM, 2019)

- Moderate-to-severe ARDS with a PaO₂:FiO₂ ratio <150mmHg and FiO₂ ≥0.6
- Implement proning early on the course of the disease (ideally <48 hours) following 12–24 hours of mechanical ventilation, allowing for treatment optimisation.
- Best outcomes achieved using tidal volumes of 6 ml/kg predicted body weight and consider the use of neuromuscular blocking drugs if there is evidence of ventilator dyssynchrony.

Positioning

- Ensure the patient is in the centre of the bed and remove the slide sheet, ensuring counter traction on the patient to prevent them slipping off the bed
- Absorbent pad placed under patient's head to catch secretions
- Carefully position the patient's arms in the 'swimmer's position'. This involves raising one arm on the same side to which the head is facing, while placing the other arm by the patient's side. The shoulder should be abducted to 80° and the elbow flexed 90° on the raised arm. The position of both the head and arms should be alternated every 2 to 4 hours. The patient should be nursed at 30° in the reverse Trendelenburg prone position.

Benefits of mechanical ventilation

The main benefits of mechanical ventilation are (American Thoracic Society (ATS), 2017):

- The patient does not have to work as hard to breathe—their respiratory muscles rest
- The patient is allowed time to recover in the hope that breathing becomes normal again
- It helps the patient receive adequate oxygen and clears carbon dioxide
- It preserves a stable airway and prevents injury from aspiration.

It is important to note that mechanical ventilation does not heal the patient. Rather, it allows the patient a chance to be stable while the medications and treatments help them recover.

Risks of mechanical ventilation

The main risks include (ATS, 2017):

- Haemodynamic instability due to positive pressure ventilation (PPV), eg diminishing venous blood return, increasing right ventricular (RV) afterload, decreasing left ventricular (LV) filling and depressing cardiac output (CO), and overall organ perfusion (Wiesen et al, 2013)

- Infections: the ET or tracheostomy tube allows bacteria to enter the patient's lungs. Wu et al (2019) state that ventilator-associated pneumonia (VAP) is a hospital-acquired infection that occurs >48 hours after initiation of mechanical ventilation. It is a common complication of mechanical ventilation and has a high mortality rate. VAP can cause patients to have difficulty weaning off the ventilator and to stay in hospital longer, resulting in a huge financial impact for hospitals and an increased demand for medical resources.
- Collapsed lung (pneumothorax): pneumothorax can be difficult to recognise in a critically ill patient. Physical examination and clinical signs and symptoms are unreliable and non-specific but may raise clinical suspicion for pneumothorax—decreased breath sounds on one side, pulsus paradoxus, haemodynamic instability with tachycardia, hypotension, contralateral tracheal deviation, and sudden increase in airway pressures (peak and plateau)—in a mechanically ventilated patient. Because clinical signs are unreliable, radiography is required to diagnose pneumothorax. A chest drain can be placed between the ribs to draw out the extra air. The chest drain allows the lung to re-expand and seal the leak. Chest drains usually must stay in place until it has been established that the leak has stopped, which can take some time. Rarely, a collapse of the lung can cause death (ATS, 2017)
- Lung damage: the pressure of putting air into the lungs via a ventilator can damage the lungs. Clinical staff will try to keep this risk at a minimum by using the lowest amount of pressure needed. Very high levels of oxygen may be harmful to the lungs. A major form of harm is ventilator-induced lung injury (Woodrow, 2019). Jackson et al (2019) identified that oxygen toxicity is due to the production of oxygen free radicals, such as superoxide anion, hydroxyl radical, and hydrogen peroxide. Oxygen toxicity can cause a variety of complications, ranging from mild tracheobronchitis and absorptive atelectasis to diffuse alveolar damage that is indistinguishable from ARDS. No consensus has been established for the level of FiO₂ required to cause oxygen toxicity, but this complication has been reported in patients given a maintenance FiO₂ of 0.5 or greater. The clinician is encouraged to use the lowest FiO₂ that accomplishes satisfactory oxygenation (Jackson et al, 2019)
- Side-effects of medications: sedatives and pain medications can cause a patient to seem confused or delirious, and these side-effects may continue to

affect the patient even after the medications have been discontinued. The healthcare team will use sedation scores and neurological screening tools to assess the patient's neurological deficit. If a neuromuscular blockade has been used to prevent muscle movement, the muscles may be weak for a period of time after the medication is stopped. This will improve over time. Unfortunately, in some cases, depending on the severity of critical illness, this weakness will remain for weeks to months.

- Inability to discontinue ventilator support: sometimes the condition that led a patient to require mechanical ventilation does not improve, despite active treatment. When this happens, clinicians will discuss alternative treatments regarding continued ventilator support. Often clinicians will have these discussions with the patient's family because the patient may not be able to participate due to the severe nature of their illness and/or their lack of mental capacity. In situations where a patient is not recovering or is getting worse, a decision may be made to discontinue ventilator support and allow death to occur.

Care ventilator bundle

VAP is an important healthcare-associated infection (HCAI) (Hellyer et al, 2016). Interventions for the prevention of VAP are often used within bundles of care. VAP is a common HCAI that occurs in 10–20% of patients who are mechanically ventilated in the ICU (Hellyer et al, 2016). Although the exact attributable mortality has proved difficult to define, it has significant consequences, with increased mortality, length of ICU stay and hospital stay, and an increase in healthcare costs (Hellyer et al, 2016). Furthermore, within a global setting of worsening antimicrobial resistance, the treatment of respiratory tract infections represents a significant burden on antimicrobials in the ICU.

Recommended bundle of interventions to prevent VAP (Hellyer et al, 2016)

- Elevation of head of bed (30°–45°) to prevent aspiration
- Daily sedation holds (daily sedation interruption and assessment of readiness to extubate)
- Use of subglottic secretion drainage

- Avoidance of scheduled ventilator circuit changes
- Use of chlorhexidine mouthwash (in cardiac patients) because it has been shown to help decrease the incidence of VAP in combination with mechanical cleansing of the oral cavity (Lorente et al, 2007).

Weaning from mechanical ventilation

In patients with severe respiratory failure artificial ventilation may be life saving. Prolonging ventilation unnecessarily is costly, both in terms of patient morbidity but also in terms of workload and finances. Debate persists over optimal weaning criteria, but the European federation of Critical Care Nursing associations (2012) suggests that readiness to wean should be identified by the following:

- Improvement in the patient's underlying medical condition
- Adequate respiratory rate and gas exchange
- Stable cardiovascular function
- An acceptable state of consciousness.

Specific respiratory parameters may include (Woodrow, 2019):

- PEEP 5 cm H₂O, FiO₂<0.5; pressure support 8–10 cm H₂O
- Arterial blood pH: 7.35–7.45, PO₂: 11.0-14.4 kPa, PCO₂: 4.6–6.4 kPa.

According to Mora Carpio and Mora (2019), weaning patients from mechanical ventilation is not without complications. Shortening ventilator time has been shown to reduce ventilation-related complications such as pneumonia, so actively pursuing liberation from mechanical ventilation (so-called 'ventilation weaning') is imperative in every ventilated patient.

Simple criteria that should be satisfied before a patient is deemed ready for extubation include:

- The indication for intubation and mechanical ventilation must be resolved
- The patient has to be able to maintain adequate gas exchange on their own, without the help of positive pressure ventilation

- There must be no auto-PEEP. Intrinsic PEEP or auto-PEEP is a complication of mechanical ventilation that most frequently occurs in patients with chronic obstructive pulmonary disease or asthma who require a prolonged expiratory phase of respiration. These patients may have difficulty in totally exhaling the ventilator-delivered tidal volume before the next machine breath is delivered. When this problem occurs, a portion of each subsequent tidal volume may be retained in the patient's lungs, a phenomenon sometimes referred to as 'breath stacking'. If this goes unrecognised, the patient's peak airway pressure may increase to a level that results in barotrauma, volutrauma, hypotension, patient-ventilator dyssynchrony, or death
- The patient must have adequate cardiovascular reserve
- There should not be copious amounts of secretions in the ET tube that could generate high airway resistance and obstruction after extubation
- The patient must have a level of consciousness to be able to protect their airway, a Glasgow Coma Scale score above 8 (Woodrow, 2019).

After these criteria have been satisfied it is time to perform a spontaneous breathing trial (SBT). To perform this, two processes must be completed:

1. A sedation-hold trial should be done daily to assess readiness for extubation. This should be performed in every patient who is stable and in whom the indication for mechanical ventilation has resolved. During these daily trials, sedation is reduced to a minimum or eliminated until the patient is awake and co-operative but comfortable
2. The second parameter is the SBT itself. To perform this, ventilator support should be reduced to a minimum. This can be done either via T-piece or pressure support ventilation (PSV). CPAP has been used in the past, although it has been suggested to be inferior to the other two methods. PSV has been identified as a superior weaning mode for performing SBTs among patients with simple weaning, when compared with T-piece and CPAP (Mora Carpio and Mora, 2019).

SBT should be performed for 30–120 minutes at a time, and the patient should be monitored closely for any signs of respiratory distress. This may include an elevated RR (>20 bpm), nasal flaring, obvious changes in body position, use of accessory

muscles, shallow breaths, peripheral and central cyanosis, and compensated tachycardia (Bickley, 2016).

If any of these signs are observed, the patient should be placed back on their prior ventilator settings. If the patient is deemed ready, the ETT should be removed and the patient should be monitored closely. In patients with high risk for re-intubation, the use of non-invasive positive pressure ventilation should be considered, via CPAP or high-flow nasal cannula. Both methods have also shown reduced re-intubation rates, although no effect on mortality has been noted.

Nurses caring for patients on mechanical ventilation require specialist knowledge and skills to monitor, identify and prevent the potential deleterious effects associated with it, so it is important to ensure that nurses caring for such patients have gained appropriate qualifications and experience.

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