Chapter 15

Ventilation and oxygenation management

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Learning objectives

After reading this chapter, you should be able to:

• describe complications associated with oxygen therapy and management priorities
• state nursing priorities for airway management strategies including laryngeal masks, endotracheal tubes and tracheostomy tubes
• summarise current knowledge on the physiological benefits, indications for use, associated monitoring priorities, complications, modes, settings and interfaces for non-invasive ventilation
• state the indications for use, associated monitoring priorities, complications, classification framework, modes and settings for invasive mechanical ventilation
• outline the weaning continuum and current evidence for optimising safe and efficient weaning from mechanical ventilation
• discuss ventilation management strategies for refractory hypoxaemia
• discuss ventilation management strategies for severe airflow limitation.

Introduction

Support of oxygenation and ventilation are two of the most common interventions in intensive care; in 2012–13, approximately 41% of patients in Australian and New Zealand intensive care units (ICUs) received invasive mechanical ventilation and 8% received non-invasive ventilation (NIV). Similar numbers of critically ill patients receive ventilation in the UK, whereas in the USA reported numbers range from 21% to 39%. The technology available for supporting oxygenation and ventilation is complex, ranging from simple interventions such as nasal cannulae through to invasive mechanical ventilation and extracorporeal support. Additionally, the meaning of ventilator terminology is often unclear and terms may be used interchangeably. Critical care nurses must have a strong knowledge of the underlying principles of oxygenation and ventilation that will facilitate an understanding of respiratory support devices, associated monitoring priorities and risks.
Oxygen therapy

Oxygen is required for aerobic cellular metabolism and ultimately for human survival, with some cells, such as those in the brain, being more sensitive to hypoxia than others. Refer to Chapter 13 for a discussion of oxygen delivery and consumption, the oxygen–haemoglobin dissociation curve, hypoxaemia and tissue hypoxia; this material provides rationales for clinical decisions regarding the administration of oxygen therapy or ventilation strategies. Oxygen therapy should be considered for patients with a significant reduction in arterial oxygen levels, irrespective of diagnosis and especially if the patient is drowsy or unconscious.

Indications

Indications for oxygen therapy include:

- cardiac and respiratory arrest
- type I respiratory failure
- type II respiratory failure
- chest pain or acute coronary syndrome with hypoxia (i.e. SpO₂ <93%) or evidence of shock
- low blood pressure, cardiac output
- increased metabolic demands
- carbon monoxide poisoning.

Complications

Administration of oxygen, regardless of the delivery device, has potential adverse effects. High concentrations of oxygen cause nitrogen washout, resulting in absorption atelectasis.

Hypoventilation and CO₂ narcosis

High-dose oxygen therapy may lead to hypoventilation, hypercapnia and CO₂ narcosis in a small proportion of patients with chronic obstructive pulmonary disease (COPD). The processes underpinning these physiological changes are described in Chapter 13. These patients require close monitoring of PaCO₂ levels when oxygen therapy is instituted or increased. Although COPD patients frequently may have a lower baseline SpO₂ (88–94% compared to 96–100% in patients with no lung pathology), treatment of hypoxia is still essential, and oxygen should not be withheld or withdrawn while hypoxia remains, even if hypercapnia worsens.5,6

Practice tip

Oxygen should not be withheld or withdrawn while hypoxia remains, even if hypercapnia worsens.

Oxygen toxicity

Administration of high oxygen concentrations may lead to oxygen toxicity; symptoms include non-productive cough, substernal pain, reduced lung compliance, interstitial oedema, and pulmonary capillary haemorrhage. These symptoms may be mistakenly attributed to the underlying illness, especially in a sedated and ventilated patient. Many of the symptoms abate once the fraction of inspired oxygen (FiO₂) is reduced, although irreversible pulmonary fibrosis may occur (see Box 15.1). The concentration and duration of oxygen exposure that induces oxygen toxicity varies between patients;7 the lowest possible FiO₂ should therefore be used to achieve the target partial pressure of oxygen in arterial blood (PaO₂) or peripheral oxygen saturation (SpO₂).

BOX 15.1

Signs and symptoms of oxygen toxicity

Central nervous system:
- Nausea and vomiting
- Anxiety
- Visual changes
- Hallucinations
- Tinnitus
- Vertigo
- Hiccups
- Seizures
Pulmonary:
- Dry cough
- Substernal chest pain
- Shortness of breath
- Pulmonary oedema
- Pulmonary fibrosis

Oxygen administration devices

Initial management of hypoxia in a spontaneously-breathing patient with an intact airway is low-flow oxygen via nasal cannulae (up to 6 L/min) or face mask (up to 15 L/min). Although oxygen devices have traditionally had FiO₂ ascribed to specific flow rates, the FiO₂ delivered to the alveoli is influenced by:

- patient factors – inspiratory flow rate, respiratory rate, tidal volume (VT), respiratory pause
- oxygen device factors – oxygen flow rate, volume of mask/reservoir, air vent size, tightness of fit.

Normal inspiratory flow in a healthy adult ranges between 25 and 35 L/min. Patients with respiratory failure tend to increase their flow demand from 50 up to 300 L/min. Patients in respiratory distress are characterised by high respiratory rates and low VT that can significantly decrease the FiO₂ available via an oxygen delivery device, depending on the type in use.

All oxygen delivery devices use some type of reservoir to support oxygen delivery and prevent CO₂ rebreathing. For face masks, the reservoir is the mask; for nasal cannulae, it is the patient’s pharynx. Patients with high
inspiratory flow demand will deplete the reservoir faster than it can be replenished, resulting in air entrainment and dilution of the oxygen concentration.

**Variable flow devices**

Various low- or variable-flow oxygen delivery devices are available. These devices range from nasal cannulae and oxygen masks with different features, through to bag-mask ventilation.

**Low-flow nasal cannulae**

Traditional low-flow nasal cannulae sit at the external nares and deliver 3–4 L/min of oxygen. Higher flows may cause discomfort and damage from the drying effect on respiratory mucosa. Increased flow demand with respiratory distress dilutes the oxygen, reducing the FiO\(_2\) to the alveoli.

**High-flow nasal cannulae**

High-flow nasal cannulae have slightly larger prongs that facilitate oxygen flow of up to 60 L/min, leading to less air entrainment than with other oxygen delivery systems.

High-flow nasal cannulae generate low levels of end-expiratory pressure, though this is dependent on the flow rate, trachea size and mouth closing, and can therefore reduce tachypnoea and work of breathing. The high gas flow may flush CO\(_2\) from the anatomical dead space preventing CO\(_2\) rebreathing and thereby decreasing PaCO\(_2\), although this is not well supported by the literature. These systems are generally well-tolerated, but must be used with heated humidification to avoid drying the respiratory mucosa. High-flow nasal cannulae are now used frequently in clinical practice to avoid, or as an alternative to, more invasive therapies but there is limited high-quality evidence on their use in adults and children other than neonates.

**Oxygen masks**

Loose-fitting oxygen masks include simple (Hudson) face masks, aerosol masks used in combination with heated humidification and nebuliser treatments, tracheostomy masks and face tents. All are considered low-flow or variable-flow devices, with the delivered FiO\(_2\) varying with patient demand. Flow rates ≥5 L/min minimise CO\(_2\) rebreathing. The addition of ‘tusks’ to a Hudson mask may increase the oxygen reservoir but does not guarantee a consistent FiO\(_2\) and has probably been superseded by high-flow systems.

Partial rebreather and non-rebreather masks have an attached reservoir bag that enables delivery of higher levels of FiO\(_2\). Both mask types have a one-way valve precluding expired gas entering the reservoir bag. A non-rebreather mask has two one-way valves preventing air entrainment. The maximum FiO\(_2\) delivery with non-rebreather masks is 0.85 with low flow demand, with a steep decline in alveolar oxygen concentration as minute volume increases. Non-rebreather masks may perform worse than a Hudson mask without a reservoir bag.

**Venturi systems**

Venturi systems use the Venturi effect to entrain gas via a narrow aperture via a side port increasing gas speed and augmenting kinetic energy. FiO\(_2\) concentration can be altered by widening or narrowing the Venturi device aperture to a maximum FiO\(_2\) of 0.6. The FiO\(_2\) concentration using a Venturi system is less affected by changes in respiratory pattern and demand compared to other low-flow oxygen devices.

**Bag–mask ventilation**

Bag-mask ventilation with a self-inflating bag (and reservoir), non-return valve and masks delivers assisted ventilation at an FiO\(_2\) of 1.0. Addition of a positive expiratory pressure (PEEP) valve will improve oxygenation. Manual ventilation requires a good seal between the patient’s face and the mask; this may be difficult to achieve as a single operator. One person should hold the mask and lift the patient’s chin, while another squeezes the bag. Effective bag-mask ventilation is confirmed when the chest visibly rises as the bag is squeezed and oxygen saturations improve. Bag-mask ventilation may cause gastric insufflation, increasing the risk of vomiting and subsequent aspiration.

**Practice tip**

Transparent face masks are recommended for bag-mask ventilation as they allow immediate recognition if a patient vomits.

**Airway support**

The most common cause of partial airway obstruction in an unconscious patient is loss of oropharyngeal muscle tone, particularly of the tongue. This may be alleviated by tilting the head slightly back and lifting the chin, or thrusting the jaw forward. The head-tilt/chin-lift manoeuvre is not used if cervical spine injury is suspected. The jaw-thrust manoeuvre may require two hands to maintain. If more prolonged support is required, an oro- or nasopharyngeal airway can be used, which may also facilitate bag-mask ventilation.

**Oro- and nasopharyngeal airways**

The Guedel oropharyngeal airway is available in various sizes (a medium-sized adult requires a size 4). The airway is inserted into the patient’s mouth past the teeth, with the end facing up into the hard palate, then rotated 180°, taking care to bring the tongue forward and not push it back. Oropharyngeal airways are poorly tolerated in conscious patients and may cause gagging and vomiting. A nasopharyngeal airway (see Figure 15.1) is inserted through the nares into the oropharynx; it can be difficult to insert and requires generous lubrication to minimise trauma. This type of airway should not be used for patients with a suspected head injury. As well as opening the airway,
suction catheters can be passed to facilitate secretion clearance. Once inserted, these airways are better tolerated than an oropharyngeal airway.

Laryngeal mask airway and insertion

The classic laryngeal mask airway (cLMA) (see Figure 15.2) is positioned blindly into the pharynx to form a low-pressure seal against the laryngeal inlet. It is easier and quicker to insert than an endotracheal tube, and is particularly useful for operators with limited airway skills; the cLMA does not carry the same potentially fatal complications such as oesophageal intubation although the risk of aspiration remains.

Mechanical ventilation can be delivered with low-airway pressures (<20 cmH₂O) via a cLMA. This device is widely used in elective general anaesthesia,21 and can be used in critical care as an alternative to bag–mask ventilation22 or endotracheal intubation when initial attempts at intubation have failed.23 The ‘intubating’ LMA

is most commonly used when a difficult intubation is anticipated or encountered. This device has a handle and is more rigid, wider and curved than the cLMA, enabling passage of a purpose-made endotracheal tube.23

Combitube

The combitube is more widely used in North America for emergency situations than in Australia and the UK.21 It is a dual-lumen, dual-cuff oesophageal–tracheal airway that enables ventilation if inserted into either the oesophagus or trachea. Inexperienced operators may find a combitube more difficult to insert correctly than a cLMA.23 Complications may occur in up to 40% of patients and include aspiration pneumonitis, pneumothorax, airway injuries and bleeding, oesophageal laceration and perforation and mediastinitis.26

Endotracheal tubes

Endotracheal intubation is the ‘gold standard’ for airway support, providing airway protection in the presence of airway oedema, absent gag, cough or swallow reflex. Intubation facilitates mechanical ventilation and pulmonary secretion clearance.22

Endotracheal tubes (ETT) have common design characteristics, are generally made from polyvinyl chloride, are available with internal diameters ranging from 2–10 mm (common adult sizes are 7–9 mm) and are up to 30 cm long. A longitudinal radio-opaque line allows visualisation of tube placement on a chest X-ray. Markings at 1-cm intervals indicate the length from the distal end, a design feature that facilitates the ability to gauge insertion depth and monitor tube movement.27 Tubes are available with and without a distal cuff, an inflatable balloon that seals the trachea, facilitates positive pressure ventilation and prevents aspiration of oropharyngeal contents. Cuffs come in a range of profiles and volumes, but are commonly high-volume, low-pressure enabling application of a safe pressure over a larger surface area (see Figure 15.3). A smaller inflatable balloon, attached to the cuff via a pilot line, provides a tactile gauge of cuff pressure and a small air reservoir to prevent minor changes in cuff pressure.28
Endotracheal tubes reinforced with a wire coil embedded within the plastic along the entire tube length prevent kinking and occlusion. These tubes are more commonly used in the operating room.29 The wire coils can be irreversibly compressed by a strong bite occluding the airway. Reinforced tubes also increase the risk of tracheal damage and should be replaced with a standard ETT on ICU arrival. Most ETTs have a ‘Murphy eye’, an oval-shaped hole in the side of the tube between the cuff and the tube end that provides a patent aperture if the distal opening is occluded.30

### Practice tip
During intubation, know who to call for help, and do not hesitate to do so.

#### Procedure
The patient is preoxygenated to minimise desaturation during apnoea and laryngoscopy, commonly via bag and mask, although other methods such as non-invasive ventilation have been suggested.32 The practice of apnoeic oxygenation during endotracheal intubation through the administration of 15 litres per minute via nasal cannula has become very popular in emergency departments. To date, there is insufficient evidence to recommend its routine use during endotracheal intubation in the critically ill. Intubation in ICU is usually performed via laryngoscopy with insertion of an oral ETT. Intubation may be performed using a fibre optic bronchoscope when difficulty is encountered, or for nasal intubation.

#### Oral vs nasal intubation
Oral intubation is preferred unless there are specific indications for nasal intubation. Oral intubation is easier to perform and allows use of a larger diameter ETT. While nasal intubation provides better splinting for the ETT and facilitates oral hygiene, it can damage nasal structures, is contraindicated in skull fractures and increases the risk of maxillary sinusitis and ventilator-associated pneumonia (VAP).33

#### Cricoid pressure
The cricoid cartilage, situated below the thyroid prominence, is a closed tracheal ring which, when compressed, closes the oesophagus while the trachea remains open. Cricoid pressure is performed by placing the thumb on one side of the patient's trachea, middle finger on the other side and index finger directly on the cricoid.34 Although widely used, its efficacy is questionable as technique is frequently poor,35 and there is wide anatomical variation in the exact orientation of the oesophagus in relation to the trachea.36

#### Backwards, upwards, rightward pressure manoeuvre
The backwards, upwards, rightward pressure (BURP) manoeuvre on the thyroid cartilage was introduced in the mid-1990s to improve visualisation during difficult laryngoscopy. The patient's jaw is thrust forward, so the head is in the 'sniffing' position. The thumb and third finger are placed on either side of the thyroid cartilage and the index finger on top. Pressure is applied in the sequence backwards (towards the spine), upwards (towards the head), rightward (towards the patient's right side). This is easier to perform following administration of muscle relaxants.

#### Cuff management
Endotracheal and tracheostomy tube cuffs prevent airway contamination by pharyngeal secretions and gastric
waveforms reflect airway pressure (Paw) during inspiration and expiration and can be used to evaluate peak and plateau inspiratory pressures and end-expiratory pressures, inspiratory and expiratory times and appropriateness of flow (see Figure 15.4). The peak pressure represents the maximum pressure achieved during inspiration. The plateau pressure is measured during an inspiratory hold and represents the pressure applied to the small airways and alveoli. The expiratory pressure is the pressure measured once the patient has expired. Pressure–time scalars vary in appearance depending on the control variable (volume vs pressure). In volume-control breaths (see Figure 15.4) the inspiratory waveform continues to rise until peak inspiratory pressure is achieved according to VT set. If an inspiratory hold is applied a plateau pressure will be generated. As the patient expires airway pressure will drop back to the set PEEP level. In pressure control breaths, the inspiratory waveform reaches its peak at the beginning of inspiration and remains at this elevation until cycling to expiration.

**FIGURE 15.4** Pressure, flow, volume and time waveforms.
When interpreting the pressure waveform it is important to recognise that the graphic waveforms display circuit pressure, which does not always represent alveolar pressure. Periods of no flow (inspiratory and expiratory holds/pauses) are required to estimate alveolar pressure. The plateau pressure is a more reliable estimate of inspiratory alveolar pressure than the peak inspiratory pressure. An expiratory hold is required to determine end-expiratory alveolar pressure. Estimating alveolar pressure may be useful to assess the patient’s respiratory resistance and compliance. Comparing the difference between the peak inspiratory pressure and plateau pressure can give an indication of the patient’s inspiratory resistance. Comparing the difference between the plateau pressure and the end-expiratory pressure can provide information about the patient’s compliance. A large difference between peak and plateau pressures indicates high airway resistance. An elevated plateau pressure indicates reduced compliance.

Spontaneous triggering of ventilation can be identified by examination of the pressure–time scalar at the beginning of inspiration. A small negative deflection indicates patient effort. When pressure-triggering is used, a breath is triggered when the pressure drops below baseline. The depth of the deflection is proportional to patient effort required to trigger inspiration (see Figure 15.5). A flow-triggered breath occurs when the flow rises above baseline, although this is frequently accompanied by a small negative deflection in the pressure–time scalar. Patient inspiratory attempts that fail to trigger the ventilator can also be identified as negative deflections in the pressure waveform without corresponding responses from the ventilator. Appropriateness of flow can be detected from the pressure–time scalar. If the flow is set too high or the rise time is too short this can be seen as a sharp peak in the waveform (see Figure 15.5). Conversely, if flow is inadequate or the rise time is too long, the incline of the inspiratory portion of the pressure waveform may be dampened or even negative.

**Flow vs time scalar**

The flow–time scalar presents the inspiratory phase above the horizontal axis and the expiratory phase below (see Figure 15.6). The shape of the inspiratory flow waveform is influenced by the selection of flow pattern (constant, decelerating, sinusoidal) in volume-control breaths or the variable and decelerating flow waveform associated with pressure-control breaths. The inspiratory flow waveform of spontaneous breaths, those triggered and cycled by the patient, is influenced by the presence or absence of pressure support and the expiratory sensitivity.

Evaluation of the expiratory limb of the flow–time scalar assists with detection of gas trapping and the patient’s response to bronchodilators. In the absence of gas trapping, the expiratory limb drops sharply below baseline then gradually returns to zero before the next breath. Failure to return to baseline indicates gas trapping whereby the inspired gas is not totally expired. Gas trapping results in development of intrinsic or ‘auto-PEEP’. This can adversely affect a patient’s haemodynamic

**FIGURE 15.5** Triggering and rise time.

<table>
<thead>
<tr>
<th>Breath 'A'</th>
<th>Breath 'B'</th>
<th>Breath 'C'</th>
</tr>
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<tbody>
<tr>
<td><strong>Pressure</strong></td>
<td><strong>Time</strong></td>
<td>0 cmH₂O</td>
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**Pressure time scalar demonstrating:**

1) how negative dips in pressure prior to the breath are indicative of patient effort required to trigger inspiration. Compare the drop in pressure in breath ‘A’ to breath ‘B’. The greater drop in pressure in breath ‘B’ indicates a greater patient effort to trigger gas flow.

2) the effect on the pressure time scalar when the rise time is set too short. Note the ‘overshoot’ in the pressure waveform on breath ‘C’. This sometimes occurs in patients with a high airway resistance (e.g. acute severe asthma) and a short rise time. It is questionable whether this pressure overshoot has any effect on the patient but it can trigger the high-pressure alarm and compromise ventilation.
status and cause patient–ventilator asynchrony.\textsuperscript{86} Gas trapping may occur in patients with airflow limitation such as those with COPD and asthma. Consequences of gas trapping include dynamic hyperinflation, reduced respiratory compliance and respiratory muscle fatigue.\textsuperscript{87} Evaluation of the expiratory flow waveform also enables evaluation of the effects of bronchodilator therapy as, if efficacious, the expiratory flow waveform will return to baseline (see Figure 15.7).\textsuperscript{86} Patient–ventilator asynchrony can be detected in the flow waveform as abrupt decreases in expiratory flow in the expiratory limb and abrupt increases in flow in the inspiratory limb.\textsuperscript{85}

**Volume vs time scalar**

The volume–time waveform originates from the functional residual capacity (baseline), rises as inspiratory flow is delivered to reach the maximum inspiratory VT, then returns to baseline during expiration. The volume waveform is useful in troubleshooting circuit leaks (see Figure 15.8) as it will fail to return to baseline if a leak in the circuit–patient interface is present.

**Loops: Pressure/volume, flow/volume**

Most contemporary critical care ventilators allow for monitoring of pressure, flow and volume parameters integrated into graphic loops enabling measurement of airway resistance, chest wall and lung compliance.

**Pressure–volume loops**

The two parameters, $P_{aw}$ and $V_T$, are plotted against each other, with $P_{aw}$ on the x axis. For mandatory breaths, the loop is drawn counterclockwise (see Figure 15.9). Spontaneous (triggered and cycled) breaths are drawn in a clockwise fashion. When low gas flow is delivered and the patient is unable to initiate ventilation, pressure–volume loops may be used to identify the lower and upper inflection points. The lower inflection point begins near the beginning of inspiration as the $P_{aw}$ starts to rise with little change in $V_T$. As $P_{aw}$ continues to rise, the $V_T$ increases exponentially as alveoli are recruited, resulting in a marked increase in the inspiratory limb slope. This point represents alveolar recruitment and is referred to
as the lower inflection point, and may be used to guide PEEP selection.\textsuperscript{88,89} The inspiratory limb continues until peak inspiratory pressure and maximal $V_T$ are achieved. The bend in the inspiratory limb towards the end of inspiration is referred to as the upper inflection point, and denotes the point at which small volume increases produce large pressure increases indicating lung overdistension.\textsuperscript{88} The expiratory limb represents lung derecruitment and is also useful in guiding PEEP selection.\textsuperscript{90,91}

For patient-triggered mandatory breaths, the initial part of the loop occurs to the left of the $y$ axis and flows...
in a clockwise fashion, reflecting patient effort. The loop then shifts to the right of the y axis and moves in a counterclockwise fashion as the ventilator assumes the work of breathing. Pressure–volume loops reflect dynamic compliance between the lungs and the ventilator circuit. Decreased compliance requires greater pressure to achieve VT and is reflected in a flattened pressure–volume loop. The area between the loops represents the resistance to inspiration and expiration, known as hysteresis. As resistance increases, less VT is delivered resulting in a shorter and wider loop; conversely, as resistance decreases, a longer, wider loop is generated (see Figure 15.10).

Flow–volume loops
Flow–volume loops recorded during positive pressure ventilation depict inspiration above the baseline and expiration below it. These loops are useful in determining response to bronchodilators and examining changes in airway resistance.

**Ventilator circuits**
Delivery of mechanical ventilation requires a ventilator circuit to transport gas flow to the patient. To prevent condensation from cooling of warm humidified gas, inspired gas is heated via a wire inside the circuit wall in either the inspiratory limb alone or both the inspiratory and expiratory limbs. Historically, ventilator circuits were changed frequently (48–72 hours) to decrease the risk of VAP. Current guidelines for VAP prevention found evidence that the frequency of ventilator circuit changes had no relationship to the VAP incidence and, therefore, recommended routine circuit changes were not necessary and circuits should only be changed when soiled or damaged.

**Humidification**
Humidification warms and moistens gas to facilitate cilia action and mucus removal as well as to prevent drying and irritation of respiratory mucosa and solidification of secretions. During endotracheal intubation and mechanical ventilation, the normal humidification processes of the nasopharynx are bypassed. This, in combination with the use of dry medical gas at high flow rates, means alternative methods of humidification are required. The best conditions for mucosal health and function over prolonged periods are when inspired gas is warmed to core body temperature and is fully saturated with water.

**Absolute and relative humidity**
Absolute humidity refers to the amount of water vapour in a given volume of gas at a given temperature. Absolute humidity rises with increasing temperature; during mechanical ventilation gas is heated to increase the amount of water vapour it will hold. Relative humidity is expressed as a percentage, and is the actual amount of water vapour in a gas compared to the maximum amount this gas can hold (ratio of absolute to maximal humidity). Ideal humidification is achieved when the:

1. inspired gas delivered into the trachea is at 37°C with a water content of 30–43 g/m³ (relative humidity is 100% at 37°C in the bronchi)
2. set temperature remains constant without fluctuation
3. humidification and temperature are unaffected by large or differing types of gas flow
4. device is simple to use
5. humidifier can be used with spontaneously breathing and ventilated patients
6. safety alarms prevent overheating, overhydration and electrocution
7. resistance, compliance and dead space characteristics do not adversely affect spontaneous breathing modes
8. sterility of the inspired gas is not compromised.

Humidification is applied using either a heat–moisture exchanger (HME) or a heated water bath reservoir device in combination with a heated ventilator circuit.
Heat–moisture exchanger

HMEs conserve heat and moisture during expiration enabling inspired gas to be heated and humidified. Two types of HMEs exist: hygroscopic and hydrophobic. Hygroscopic HMEs absorb moisture onto a chemically impregnated foam or paper material and have been shown to be more effective than hydrophobic HMEs. HMEs are placed distally to the circuit Y-piece in line with the ETT and increase dead space by an amount equal to their internal volume. HMEs should be changed every 24 hours or when soiled with secretions and are usually reserved for short-term humidification.

Heated humidification

Generally, heated humidification is used for patients requiring more than 24 hours of mechanical ventilation. Various models of heater bases and circuits are on the market and we recommend their use in accordance with manufacturer instructions. A recent systematic review and meta-analysis reported no overall effect on artificial airway occlusion, mortality, pneumonia or respiratory complications when HMEs were compared to heated humidification, although it noted that the PaCO₂ and minute ventilation were increased and body temperature was lower with the use of HMEs.

Non-invasive ventilation

NIV is an umbrella term describing the delivery of mechanical ventilation without the use of an invasive airway, via an interface such as an oronasal, nasal or full-face mask or helmet. NIV techniques include both negative and positive pressure ventilation, although in critical care positive pressure ventilation is primarily used.

Terminology

Positive pressure NIV can be further categorised as non-invasive positive pressure ventilation (NIPPV) or continuous positive airway pressure (CPAP). NIPPV is the provision of inspiratory pressure support, also referred to as inspiratory positive airway pressure (IPAP), usually in combination with positive end-expiratory pressure (PEEP). PEEP is also referred to as expiratory positive airway pressure (EPAP). CPAP does not actively assist inspiration but provides a constant positive airway pressure throughout inspiration and expiration. The terms biphasic (or bilevel) positive airway pressure (BiPAP) and non-invasive pressure support ventilation (NIPSV) are also used to refer to NIPPV. The acronym BiPAP is registered to Respironics (Murrayville, PA), a company that produces non-invasive ventilators including the BiPAP Vision, which is commonly used in the ICU. The acronym NIPSV is primarily used in European descriptions of NIPPV.

Physiological benefits

The efficacy of NIV in patients with acute respiratory failure is, at least in part, related to avoidance of inspiratory muscle fatigue through the addition of inspiratory positive pressure, thus reducing inspiratory muscle work. Application of positive pressure during inspiration increases transpulmonary pressure, inflates the lungs, augments alveolar ventilation and unloads the inspiratory muscles. Augmentation of alveolar ventilation, demonstrated by an increase in V̇, increases CO₂ elimination and reverses acidaemia. High levels of inspiratory pressure may also relieve dyspnoea.

The main physiological benefit in patients with congestive heart failure (CHF) is attributed to the increase in functional residual capacity associated with the use of PEEP that reopens collapsed alveoli and improves oxygenation. Increased intrathoracic pressure associated with the application of positive pressure also may improve cardiac performance by reducing myocardial work and oxygen consumption through reductions to ventricular preload and left ventricular afterload. NIV also preserves the ability to speak, swallow, cough and clear secretions, and decreases risks associated with endotracheal intubation.

Indications for NIV

The success of NIV treatment is dependent on appropriate patient selection. Table 15.3 outlines indications and contraindications to NIV.

Acute respiratory failure

Evidence supporting the role of NIV in patients with hypoxaemic respiratory failure is limited and conflicting. For patients with community-acquired pneumonia, NIV has been shown to reduce intubation rates, ICU length of stay and 2-month mortality but only in the subgroup of patients with COPD. Pneumonia also has been identified as a risk factor for NIV failure.

Acute exacerbation of COPD and CHF

Strong evidence exists to support the use of NIV for patients with acute exacerbation of COPD and CHF. Three meta-analyses have shown a reduction in intubation rates, hospital length of stay and mortality for COPD patients managed with NIPPV compared to standard medical treatment. COPD patients most likely to respond favourably to NIPPV include those with an unimpaired level of consciousness, moderate acidaemia, a respiratory rate of <30 breaths/minute and who demonstrate an improvement in respiratory parameters within 2 hours of commencing NIPPV.

Early use of NIV in combination with standard therapy for patients with CHF has also been shown to reduce intubation rates and mortality when compared
to standard therapy alone. The most recent meta-analysis including 32 trials found NIV reduced hospital mortality and reintubation rates by nearly 50% compared to standard medical care and no increased risk of acute myocardial infarction. The authors recommended CPAP may be considered as the first option for NIV due to more robust evidence for its effectiveness, safety and lower cost compared with NIPPV. Practice surveys indicate CPAP may be the preferred method of NIV for patients with CHF in Australia and internationally.

**NIV in weaning**

NIV may be used as an adjunct to weaning to reduce the duration of invasive ventilation and associated complications. Patients are extubated directly to NIV and then weaned to standard oxygen therapy. This use of NIV differs from its role in preventing reintubation in patients that develop, or who are at high risk of, postextubation respiratory failure. A recent systematic review and meta-analysis of 16 trials with 994 participants (mostly COPD) using NIV immediately after extubation reported reductions in mortality, weaning failure, VAP, tracheostomy, reintubation, ICU and hospital lengths of stay and total duration of ventilation. Conversely, the effectiveness of NIV for postextubation respiratory failure is not so clear. The largest study of NIV use in postextubation respiratory failure reported worsened survival rates hypothesised to be as a result of delayed reintubation. A subsequent meta-analysis suggested NIV may have a role in preventing the development of respiratory failure postextubation for those at risk, but should be used with caution once respiratory failure has developed and should not delay the decision to reintubate.

**Other indications**

Other indications for NIV include:

- asthma
- pulmonary infiltrates in immunocompromised patients
- neuromuscular disorders (e.g. muscular dystrophy, amyotrophic lateral sclerosis)
- fractured ribs
- obesity and central hypoventilation syndromes
- palliation.

**Patient selection**

Selection of patients to receive NIV depends on the presence of an indication listed above as well as bedside observations and gas exchange parameters found in Table 15.3.

**Interfaces and settings**

NIV requires an interface that connects the patient to a ventilator, portable compressor or flow generator with a CPAP valve. The selection of an appropriate interface can influence NIV success or failure. Oronasal masks cover both the mouth and nose and are the preferred mask type for the management of acute respiratory failure. Nasal masks enable speech, eating and drinking, and therefore are employed more frequently for long-term NIV use. An oronasal mask enables delivery of higher ventilation

<table>
<thead>
<tr>
<th>TABLE 15.3</th>
<th>Indications and contraindications for non-invasive ventilation</th>
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<tbody>
<tr>
<td><strong>INDICATIONS</strong></td>
<td><strong>CONTRAINDICATIONS</strong></td>
</tr>
<tr>
<td>Bedside observations</td>
<td>Increased dyspnoea; moderate-to-severe tachypnoea:</td>
</tr>
<tr>
<td></td>
<td>( &gt;24 \text{ breaths per min {obstructive} } )</td>
</tr>
<tr>
<td></td>
<td>( &gt;30 \text{ breaths per min {restrictive} } )</td>
</tr>
<tr>
<td></td>
<td>Signs of increased work of breathing, accessory muscle use and abdominal paradox</td>
</tr>
<tr>
<td>Gas exchange</td>
<td>Acute or acute-on-chronic ventilatory failure (best indication), ( \text{PaCO}_2 &gt;45 \text{ mmHg, pH &lt;7.35} )</td>
</tr>
<tr>
<td></td>
<td>Hypoxaemia (use with caution), ( \text{PaO}_2/\text{FiO}_2 \text{ ratio &lt;200} )</td>
</tr>
<tr>
<td>Absolute</td>
<td>Respiratory arrest</td>
</tr>
<tr>
<td></td>
<td>Unable to fit mask</td>
</tr>
<tr>
<td>Relative</td>
<td>Medically unstable: hypotensive shock, uncontrolled cardiac ischaemia or arrhythmia, uncontrolled upper gastrointestinal bleeding</td>
</tr>
<tr>
<td></td>
<td>Agitated, uncooperative</td>
</tr>
<tr>
<td></td>
<td>Unable to protect airway</td>
</tr>
<tr>
<td></td>
<td>Swallowing impairment</td>
</tr>
<tr>
<td></td>
<td>Excessive secretions not managed by secretion clearance techniques</td>
</tr>
<tr>
<td></td>
<td>Multiple (i.e. two or more) organ failure</td>
</tr>
<tr>
<td></td>
<td>Recent upper airway or upper gastrointestinal surgery</td>
</tr>
</tbody>
</table>

\( \text{PaCO}_2 = \text{partial pressure of carbon dioxide in arterial blood; PaO}_2 = \text{partial pressure of oxygen in arterial blood; PaO}_2/\text{FiO}_2 = \text{ratio of partial pressure of oxygen in arterial blood to fraction of inspired oxygen.} \)
pressures with less leak and greater comfort for the patient. Other interfaces include full-face masks that seal around the perimeter of the face and cover the eyes as well as the nose and mouth, nasal pillows, mouthpieces that are placed between the patient’s lips and helmets that cover the whole head and consist of a transparent plastic hood attached to a soft neck collar. These alternative interfaces may increase patient tolerance by reducing pressure ulceration, air leaks and patient discomfort.

Initiation and monitoring priorities

Successful initiation of NIV is dependent on patient acceptance and tolerance. Patient acceptance may be aided by a brief explanation of the procedure and its benefits. Strategies to enhance patient tolerance include: use of an interface that fits the patient’s facial features, commencing with low pressure levels, holding the mask gently in position prior to securing with the straps/headgear and ensuring straps prevent major leaks but are not so tight they increase discomfort. Once NIV is commenced, the patient should be monitored for respiratory and haemodynamic stability, response to NIV treatment, ongoing tolerance and presence of air leaks (Table 15.4). Arterial blood gas analysis should be performed at baseline and within the first 1 to 2 hours of commencement. During the initiation and stabilisation period, patients should be monitored using a nurse-to-patient ratio of 1:1 with ongoing coaching to promote NIV tolerance throughout the early stabilisation period.

### Practice tip

NIV tolerance may be promoted with a simple explanation of the therapy, reassurance and constant monitoring for your patient. During initiation, allow them to take short breaks from the mask if they are in discomfort or experiencing claustrophobia.

### Potential complications

Masks need to be tight-fitting to reduce air leaks; however, this contributes to pressure ulceration on the bridge of the nose or above the ears (due to mask straps/headgear). Air leaks may cause conjunctival irritation and the high flow of dry medical gas results in nasal congestion, oral or nasal dryness and insufflation of air into the stomach. Claustrophobia associated with the NIV interface may also lead to agitation, reducing the efficacy of NIV treatment due to poor coordination of respiratory cycling between the patient and NIV unit.

More serious, yet infrequent, complications include aspiration pneumonia, haemodynamic compromise associated with increased intrathoracic pressures and pneumothorax.

### Detecting NIV failure

Failure to respond to NIV within 1–2 hours of commencement is demonstrated by unchanged or worsening gas exchange, as well as ongoing or new onset of rapid shallow breathing and increased haemodynamic instability. Decreased level of consciousness may be indicative of imminent respiratory arrest.

### Weaning from NIV

Existing guidelines provide little guidance on weaning of NIV. In many cases, NIV may be simply withdrawn as opposed to weaned. Those commencing on high levels of IPAP and/or EPAP may need weaning based on ongoing assessment of dyspnoea and chest wall movement, as well as ventilation and oxygenation parameters. Another weaning method may be progressive extension of time off NIV, while monitoring tolerance.

### Invasive mechanical ventilation

Critically ill patients with persistent respiratory insufficiency (hypoxaemia and/or hypercapnia), due to drugs, disease or other conditions, may require intubation and mechanical ventilation to support oxygenation and ventilatory demands. Clinical criteria for intubation and ventilation should be based on individual patient assessment and patient response to measures aimed at reversing hypoxaemia.

---

**TABLE 15.4**

<table>
<thead>
<tr>
<th>PRIORITY</th>
<th>ASSESSMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient comfort</td>
<td>Restlessness</td>
</tr>
<tr>
<td>Anxiety level</td>
<td>Dyspnoea score</td>
</tr>
<tr>
<td>Conscious level</td>
<td>Glasgow Coma Score</td>
</tr>
<tr>
<td>Work of breathing</td>
<td>Chest wall motion</td>
</tr>
<tr>
<td>Gas exchange parameters</td>
<td>Continuous SpO2</td>
</tr>
<tr>
<td>Haemodynamic status</td>
<td>Continuous heart rate</td>
</tr>
<tr>
<td>Ventilator parameters</td>
<td>Air leak around mask</td>
</tr>
<tr>
<td>PaCO2 = partial pressure of carbon dioxide in arterial blood; PaO2 = partial pressure of oxygen in arterial blood; SpO2 = saturation of peripheral oxygen; V̇t = tidal volume.</td>
<td></td>
</tr>
</tbody>
</table>

Indications
Indications for intubation and mechanical ventilation include:
- apnoea
- inability to protect airway; e.g. loss of gag/cough reflex; decreased Glasgow Coma Scale score
- clinical signs indicating respiratory distress; e.g. tachypnoea, activation of accessory and expiratory muscles, abnormal chest wall movements, tachycardia and hypertension
- inability to sustain adequate oxygenation for metabolic demands; e.g. cyanosis, $\text{SpO}_2 < 88\%$, with supplemental $\text{FiO}_2 \geq 0.5$
- respiratory acidosis (e.g. acute decrease in $\text{pH} < 7.25$)
- postoperative respiratory failure
- shock.

The goals of mechanical ventilation are to achieve and maintain adequate pulmonary gas exchange, minimise the risk of lung injury, reduce patient work of breathing and optimise comfort.

Mechanical ventilators
Contemporary ventilators use sophisticated microprocessor controls with sensitive detection, response and control of pressure and gas flow characteristics. These ventilators are more sensitive to patient ventilatory demands, enabling improved patient–ventilator synchrony during both inspiratory and expiratory breath phases. Parameters commonly manipulated during mechanical ventilation are detailed in Table 15.5. Parameters often observed and documented are discussed below.

### Phases of breath delivery
The respiratory cycle comprises both inspiratory and expiratory phases (see Figure 15.11). Pressure, flow, volume and time are parameters used to describe or classify mechanical ventilator breaths during the phases of inspiration. Ventilator breaths are classified by: 1) the mechanism (ventilator or patient) that ‘triggers’ the start of inspiration; 2) the parameter that is ‘targeted’ (also referred to as ‘controlled’ or ‘limited’) during inspiration; and 3) the parameter that ‘cycles’ the breath from inspiration to expiration.138

### BOX 15.2
**Mechanical ventilation of the elderly patient**
- Elderly survivors of mechanical ventilation may have a greater increase in disability than those hospitalised and not requiring ventilation;138 this is information that should be shared with patients and family members when considering treatment options.
- Frail elderly are at increased risk of delirium resulting in prolonged mechanical ventilation.137

### Table 15.5
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\text{Fraction of inspired oxygen (FiO}_2$</td>
<td>The fraction of inspired oxygen delivered on inspiration to the patient</td>
</tr>
<tr>
<td>Tidal volume ($V_t$)</td>
<td>Volume (mL) of each breath</td>
</tr>
<tr>
<td>Set breath rate (f)</td>
<td>The clinician-determined set rate of breaths delivered by the ventilator (bpm)</td>
</tr>
<tr>
<td>Inspiratory trigger or sensitivity</td>
<td>Mechanism by which the ventilator senses the patient’s inspiratory effort. May be measured in terms of a change in pressure or flow</td>
</tr>
<tr>
<td>Inspiratory pressure ($P_{\text{insp}}$, $P_{\text{high}}$)</td>
<td>Clinician-determined pressure that is targeted during inspiration</td>
</tr>
<tr>
<td>Inspiratory time ($T_{\text{insp}}$)</td>
<td>The duration of inspiration (s)</td>
</tr>
<tr>
<td>Inspiratory: expiratory ratio (I:E)</td>
<td>The ratio of the inspiratory time to expiratory time</td>
</tr>
<tr>
<td>Flow (V)</td>
<td>The speed gas travels during inspiration (L/min)</td>
</tr>
<tr>
<td>Pressure support (PS)</td>
<td>The flow of gas that augments a patient’s spontaneously initiated breath to a clinician-determined pressure (cmH$_2$O)</td>
</tr>
<tr>
<td>Positive end-expiratory pressure (PEEP)</td>
<td>Application of airway pressure above atmospheric pressure at the end of expiration (cmH$_2$O)</td>
</tr>
<tr>
<td>Rise time</td>
<td>Time to achieve maximal flow at the onset of inspiration for pressure-targeted breaths</td>
</tr>
<tr>
<td>Expiratory sensitivity</td>
<td>During a spontaneous breath, the ventilator cycles from inspiration to expiration once flow has decelerated to a percentage of initial peak flow</td>
</tr>
<tr>
<td>Minute volume ($V_{\text{E}}$)</td>
<td>Generally not set directly but is determined by $V_t$ and f settings. Tidal volume multiplied by the respiratory rate over one minute (L/min)</td>
</tr>
<tr>
<td>Airway pressure ($P_{\text{aw}}$)</td>
<td>The pressure measured in cmH$_2$O by the ventilator in the proximal airway</td>
</tr>
<tr>
<td>Plateau pressure ($P_{\text{plat}}$)</td>
<td>The pressure, measured in cmH$<em>2$O, applied to the small airways and alveoli. $P</em>{\text{plat}}$ is not set but can be measured by performing an inspiratory hold manoeuvre</td>
</tr>
</tbody>
</table>
Pressure vs volume delivery

Traditionally, clinicians have favoured volume control ventilation (VCV) due to the ability to regulate minute ventilation ($V_E$) and CO$_2$ elimination with straightforward manipulation of respiratory rate and $V_T$. VCV provides consistent $V_T$ delivery, independent of lung mechanics. The set $V_T$ and flow rates used in VCV mean that the ventilator is unable to increase volume or flow rates in response to the patient’s inspiratory demands during mandatory breaths. This inability to respond to the patient’s inspiratory flow requirements can lead to dyssynchrony. Another disadvantage of VCV is the lack of control over peak airway pressure that changes in response to altered compliance and resistance. Elevated plateau pressure may cause alveolar overdistension, barotrauma and haemodynamic effects such as reduced venous return and cardiac output resulting in hypotension and thus decreased organ perfusion. Clinicians need to carefully monitor ventilation to avoid injurious pressures. In VCV the peak airway pressure is achieved towards the end of inspiration, and only for a short duration; therefore, distribution of gas may not
be optimised and shearing stress can occur. This can be overcome with the use of a decelerating waveform and an inspiratory time that produces an inspiratory hold.

Pressure controlled ventilation (PCV) allows control over the peak inspiratory pressure and inspiratory time. Clinicians must monitor minute ventilation and gas exchange due to the lack of a guaranteed VT and possible changes in respiratory compliance and resistance. The variable flow and VT mean that there is the potential for greater interaction between patient efforts and ventilator breaths than is present in volume controlled ventilation. The variable and decelerating inspiratory gas flow pattern of PCV enables rapid alveolar filling and more even gas distribution compared to the constant flow pattern that may be used with volume control. This decelerating flow pattern also results in improved gas exchange, decreased work of breathing and prevention of overdistension in healthy alveoli.

During PCV, the set inspiratory pressure is achieved at the beginning of the inspiratory cycle and maintained for the set inspiratory time. This promotes recruitment of alveoli with high opening pressures and long time-constants.

**Ventilator parameters**

**Fraction of inspired oxygen**

The FiO₂ is expressed as a decimal, between 0.21 and 1.0, when supplemental oxygen is applied. Room air has an oxygen content of 0.21 (21%). Ventilation is commonly commenced on a high FiO₂ setting but, as noted earlier, clinicians should consider the risks of oxygen toxicity, which include disruption to the alveolar-capillary membrane and alveolar wall fibrosis.

**Tidal volume**

Vₜ is the volume, measured in mL, of each breath. Set or targeted Vₜ is calculated using the patient’s ideal body weight using height and gender-specific tables to achieve 6–8 mL/kg (see Table 15.6). Strong evidence indicates

<table>
<thead>
<tr>
<th>TABLE 15.6</th>
<th>ARDSnet tables for predicted body weight for females and males</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PBW</strong></td>
<td><strong>4 ML</strong></td>
</tr>
<tr>
<td><strong>PBW</strong></td>
<td><strong>4 ML</strong></td>
</tr>
<tr>
<td>31.7</td>
<td>127</td>
</tr>
<tr>
<td>34</td>
<td>136</td>
</tr>
<tr>
<td>36.3</td>
<td>145</td>
</tr>
<tr>
<td>38.6</td>
<td>154</td>
</tr>
<tr>
<td>40.9</td>
<td>164</td>
</tr>
<tr>
<td>43.2</td>
<td>173</td>
</tr>
<tr>
<td>45.5</td>
<td>182</td>
</tr>
<tr>
<td>47.8</td>
<td>191</td>
</tr>
<tr>
<td>50.1</td>
<td>200</td>
</tr>
<tr>
<td>52.4</td>
<td>210</td>
</tr>
<tr>
<td>54.7</td>
<td>219</td>
</tr>
<tr>
<td>57</td>
<td>228</td>
</tr>
<tr>
<td>59.3</td>
<td>237</td>
</tr>
<tr>
<td>61.6</td>
<td>246</td>
</tr>
<tr>
<td>63.9</td>
<td>256</td>
</tr>
<tr>
<td>66.2</td>
<td>265</td>
</tr>
<tr>
<td>68.5</td>
<td>274</td>
</tr>
<tr>
<td>70.8</td>
<td>283</td>
</tr>
<tr>
<td>73.1</td>
<td>292</td>
</tr>
<tr>
<td>75.4</td>
<td>302</td>
</tr>
<tr>
<td>77.7</td>
<td>311</td>
</tr>
<tr>
<td>80</td>
<td>320</td>
</tr>
<tr>
<td>82.3</td>
<td>329</td>
</tr>
<tr>
<td>84.6</td>
<td>338</td>
</tr>
<tr>
<td>86.9</td>
<td>348</td>
</tr>
</tbody>
</table>

PBW = predicted body weight.

The formulae, when using height in centimetres, are:

- **Females** = 45.5 + 0.91 × (height in cm − 152.3)
- **Males** = 50 + 0.91 × (height in cm − 152.3).

Adapted from information courtesy of ARDSnet. Further information available at http://www.ardsnet.org/node/77460.
a mortality benefit for using 6 mL/kg in patients with acute respiratory distress syndrome (ARDS).\(^{148,121}\) Increasingly, evidence indicates protective lung ventilation using 6 mL/kg as a target for patients without ARDS is associated with improved clinical outcomes.\(^{149-151}\) While further studies are required, clinicians should consider aiming for 6–8 mL/kg in all ventilated patients.

**Respiratory rate**

Mandatory frequency (\(f\)) or respiratory rate (RR) is set with consideration of the patient’s own respiratory effort, anticipated ventilatory requirements and the effect on the I:E ratio. Use of high doses of sedation with or without neuromuscular blockade requires setting a mandatory rate that facilitates adequate gas exchange and meets oxygenation requirements. A lower frequency can be set for a patient able to breathe spontaneously in modes such as synchronised intermittent mandatory ventilation (SIMV) and assist control (A/C) (see below) to enable spontaneous triggering. Physiologically normal respiratory rates are 12–20 breaths per minute. Patients with hyperoxic respiratory failure generally breathe 20–30 breaths per minute.\(^{152}\)

**Triggering of inspiration**

Depending on the ventilation mode, breaths are triggered by the ventilator or patient in various sequences. A breath may be triggered by the ventilator in response to time elapsed in modes with clinician-determined set frequency, such as controlled mandatory ventilation (CMV), and in A/C and SIMV in the absence of spontaneous effort. Patient triggering requires the ventilator to sense the patient’s inspiratory effort. Most modern ventilators now use flow triggering, as evidence indicates that flow triggering may be more responsive to patient effort than pressure triggering.\(^{153}\) Pressure triggering requires the patient to create a negative pressure within the ventilator circuit of sufficient size to enable the ventilator to sense the effort and commence flow of gas. Flow triggering is sometimes used in conjunction with a predetermined flow of gas, usually 5–10 L/min, referred to as the bias (or base) flow, that travels continuously through the ventilator circuit. When the patient makes an inspiratory effort, they divert flow, which is sensed by the ventilator. If the flow diversion reaches a clinician-determined set value, a breath is initiated.\(^{154}\) The flow trigger is usually set at 1–3 L/min (1 L/min represents less patient effort and 3 L/min represents greater patient effort). Despite advances in ventilator technology, various studies continue to identify missed patient triggers that contribute to patient–ventilator asynchrony.\(^{155}\) Conversely, ‘auto-triggering’ is ventilator triggering in the absence of spontaneous inspiratory effort. Auto triggering is sometimes observed in patients with an increased cardiac output, such as those fulfilling brain death criteria.

**Rise time**

The rise time controls how quickly the ventilator reaches the clinician-determined inspiratory pressure (\(P_{\text{insp}}\)) for mandatory breaths and pressure support for spontaneous breaths. Reducing the rise time to its lowest value will enable the ventilator to reach target pressure in the shortest time frame resulting in a more rapid delivery of flow in the early phase of inspiration. This reduces work of breathing and improves synchrony. In patients with a high airway resistance (e.g., severe asthma), a short inspiratory time may cause oscillation and overshoot of the pressure waveform. The clinical relevance of this is questionable but it produces an abnormal pressure waveform and results in alarm violations. Increasing the rise time may alleviate this problem. In other patients, an increased rise time may unnecessarily increase their work of breathing.\(^{156}\)

**Inspiratory time and inspiratory-to-expiratory ratio**

The total time available for each mandatory breath is determined by the set inspiratory time and breath frequency. Normal inspiratory time is 0.8 to 1.2 seconds. Total breath time comprises the inspiratory (I) and expiratory (E) time, which can be expressed as a ratio (I:E). In normal spontaneous breathing, expiratory time is approximately twice the inspiratory time (1:2 ratio). Gas flow also influences inspiratory time, with higher gas flows resulting in decreased time to achieve the target \(V_T\). The I:E ratio can be manipulated to create an inverse relationship (1:1, 2:1, 4:1) with the goal of increased mean airway pressure resulting in alveolar recruitment and improved oxygenation. Prolonging the inspiratory time beyond normal or using inverse ratio ventilation in any mode can result in patient ventilator dyssynchrony and increased risk of barotrauma.\(^{157}\)

**Inspiratory flow and flow pattern**

The flow rate refers to the speed of gas, is measured in litres per minute (L/min) and generally delivered at speeds of 30–60 L/min. Higher flow rates cause turbulent gas flow, resulting in increased peak airway pressures. Lower flow rates result in laminar flow, increased inspiratory time, improved gas distribution and lower peak airway pressures.\(^{158}\) The flow of inspiratory gas can be delivered in three styles: constant or square wave, decelerating ramp and sinusoidal pattern (see Figure 15.6). In a constant flow pattern, the peak flow is achieved at the beginning of inspiration and is held constant throughout the inspiratory phase. This may result in higher peak airway pressures. Using a decelerating ramp, the gas flow is highest at the beginning of inspiration and tapers throughout the inspiratory phase. Sinusoidal gas flow resembles spontaneous ventilation.

**Peak airway pressure**

Airway pressures vary across the respiratory cycle with a number of pressures identifiable (e.g. peak inspiratory, end-expiratory). The airway pressure (\(P_{\text{aw}}\)) is an important parameter in assessing respiratory compliance and patient–ventilator synchrony, and will vary depending on \(V_T\), RR, ventilator flow pattern, dynamic compliance and airway resistance.
resistance. In pressure-targeted modes the peak inspiratory pressure is equivalent to the $P_{\text{insp}}$. In volume-targeted modes the peak inspiratory pressure is determined by the set $V_T$ and patient compliance and resistance.

**Positive end-expiratory pressure**

PEEP is the pressure applied at the end of the expiratory cycle to prevent alveolar collapse. PEEP increases residual lung volume thereby recruiting collapsed alveoli, improving ventilation/perfusion match and enhancing movement of fluid out of the alveoli.\(^{159,160}\) PEEP was originally introduced by Ashbaugh and colleagues\(^{161}\) in the 1960s as a technique for treating refractory hypoxaemia in patients with ARDS. Animal studies suggest ventilator-associated lung injury may be prevented using PEEP by recruiting atelectic alveoli and bronchioles and preventing cyclic opening and closing of alveoli.\(^{162-165}\) PEEP may be beneficial, however, only if the lung is recruitable such as in collapsed, as opposed to consolidated, lung.\(^{159}\) Selection of optimal PEEP remains controversial. Low PEEP levels have been shown to be associated with higher mortality for ARDS patients in several studies.\(^{166-169}\) Two randomised, controlled trials comparing low tidal volume ventilation and conventional PEEP to low tidal volume ventilation and high PEEP, with and without additional recruitment manoeuvres (40 cmH\(_2\)O applied for 40 s),\(^{170,171}\) did not demonstrate a reduction in hospital\(^{170}\) or 28-day\(^{171}\) mortality.

**Pressure support**

When triggered by the patient, the ventilator delivers flow to achieve the clinician-determined set pressure support. The flow is variable, depending on the patient demand. The $V_T$ achieved with pressure support is dependent on chest and lung compliance as well as airflow and ventilator resistance. Pressure support is generally set at 5–20 cmH\(_2\)O. Increasing the level of pressure support will result in increased $V_T$ and improvements in gas exchange if compliance and resistance remain constant.

**Expiratory sensitivity**

Expiratory sensitivity describes the percentage of decay in peak flow reached during the inspiratory phase that signals the ventilator to cycle to expiration for spontaneous breaths. In some ventilator models this is predetermined at 25%, while others allow clinician selection. Premature termination of a breath will increase inspiratory muscle workload whereas delayed breath termination increases expiratory muscle load.\(^{172}\) Reducing the expiratory sensitivity in patients with COPD may prolong the inspiratory time, thereby increasing the $V_T$ and reducing the respiratory rate and gas trapping.\(^{173}\)

**Practice tip**

The $P_{\text{insp}}$ setting reflects a different value on different ventilators. $P_{\text{insp}}$ equals total pressure including PEEP on some ventilators and $P_{\text{insp}}$ above PEEP on others. Use the pressure–time scalar to confirm.

**Ventilator modes**

The mode of ventilation describes inspiratory phase variables; how the ventilator controls pressure, volume and flow during a breath; as well as describing how breaths are sequenced. All breaths have trigger, limit and cycle inspiratory phase variables.\(^{174}\) Each breath is triggered (started) either by the patient or by the ventilator. During inspiration, the breath is limited to a set target of pressure, volume or flow. This target cannot be exceeded during each breath. The cycling variable determines the end of the inspiratory phase. Again this variable may be pressure, flow, volume or time. Gas delivery during each breath is described by the control variable. There are five control variables: pressure, volume, flow, time and dual control (such as used in the mode pressure-regulated volume control). Breathing sequencing refers to the sequence of mandatory and spontaneous breath. A spontaneous breath is one during which inspiration is both started (triggered) and stopped (cycled) by the patient. Spontaneous breaths may be assisted, as with pressure support, or unassisted. Mandatory breaths are either triggered or cycled by the ventilator.\(^{175}\) A complete mode description should include: 1) the control variable; 2) the breath sequence; and 3) the targeting scheme (limit variable).

**Commonly employed ventilation modes**

Contemporary ventilators now provide a range of modes to facilitate mechanical ventilation. See Table 15.7.

**Controlled mandatory ventilation**

CMV is a mandatory mode, and is the original and most basic mode of ventilation.\(^{176}\) CMV delivers all breaths at a clinician-determined set frequency (rate); the patient’s spontaneous effort is not acknowledged by the ventilator.\(^{81}\) VCV requires clinician selection of the frequency, PEEP, FiO\(_2\), tidal volume, flow waveform, peak inspiratory flow and either the inspiratory time or I:E ratio. PCV requires clinician selection of rate, PEEP, FiO\(_2\), inspiratory pressure, as opposed to tidal volume, and inspiratory time or I:E ratio depending on the ventilator type. Peak inspiratory flow and the flow waveform are manipulated by the ventilator, to achieve the clinician-selected inspiratory pressure within the set inspiratory time. The inability to breathe spontaneously during CMV contributes to diaphragm muscle dysfunction and atrophy, which may result in difficulty weaning from the ventilator.\(^{177}\)

**Assist control**

In A/C the patient can trigger the ventilator; however, unlike SIMV, every patient-initiated breath is assisted to the same clinician-determined $V_T$ (volume targeted) or inspiratory pressure (pressure targeted). All breaths are cycled by the ventilator irrespective of being patient- or ventilator-triggered. In the absence of spontaneous breathing, A/C resembles CMV.
TABLE 15.7

<table>
<thead>
<tr>
<th>MODE</th>
<th>DESCRIPTION</th>
<th>CLINICAL IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controlled mandatory ventilation (CMV)</td>
<td>All breaths are mandatory, no patient triggering is enabled. Also called volume controlled ventilation (volume targeted) (VCV) and pressure controlled ventilation (pressure targeted) (PCV)</td>
<td>Patients with respiratory effort require sedation and neuromuscular blockade. Potential for respiratory muscle atrophy due to disuse.</td>
</tr>
<tr>
<td>Assist-control (A/C)</td>
<td>Breaths may be either machine or patient triggered but all are cycled by the ventilator. Assist control may be delivered as volume (AC-VC) or pressure (AC-PC) targeted</td>
<td>Activation of the diaphragm with patient triggering. Potential for respiratory alkalosis if tachypnoea develops.</td>
</tr>
<tr>
<td>Synchronised intermittent mandatory ventilation (SIMV)</td>
<td>Mandatory breaths are delivered using a set rate and volume (SIMV-VC) or pressure (SIMV-PC). Mandatory breaths are synchronised with patient triggers within a timing window. Between mandatory breaths the patient can breathe spontaneously.</td>
<td>Reduced need for sedation. Activation of the diaphragm with patient triggering.</td>
</tr>
<tr>
<td>Pressure support ventilation (PSV)</td>
<td>All breaths are patient triggered and cycled. Pressure applied by the ventilator during inspiration (pressure support) augments patient effort</td>
<td>Reduced need for sedation. Facilitates ventilator weaning. Level of PS can be adjusted to achieve desired VT. Sustains respiratory muscle tone and decreases work of breathing.</td>
</tr>
<tr>
<td>Continuous positive airway pressure (CPAP)</td>
<td>All breaths are patient triggered and cycled. Positive pressure is applied throughout inspiratory and expiratory phases of the respiratory cycle.</td>
<td>Requires intact respiratory drive and patient ability to maintain adequate tidal volumes.</td>
</tr>
<tr>
<td>Volume support (VS)</td>
<td>Spontaneous mode with clinician preset target tidal volume delivery achieved with the lowest inspiratory pressure.</td>
<td>Requires intact respiratory drive.</td>
</tr>
<tr>
<td>Pressure-regulated volume control (PRVC)</td>
<td>Mandatory rate and target tidal volume are set, and the ventilator then delivers the breaths using the lowest achievable pressure.</td>
<td>Dual control of volume and pressure enables guarantee of volume and pressure.</td>
</tr>
<tr>
<td>Airway pressure release ventilation (APRV)</td>
<td>Ventilator cycles between 2 preset pressure levels for defined time periods. I:E ratio is inverse, often with a prolonged inspiratory time (4 s) and shortened expiratory time (0.8 s). Patient can breathe spontaneously at both pressure levels.</td>
<td>Reduced need for sedation. Activation of the diaphragm with patient triggering. Promotes alveolar recruitment. Considered a rescue mode in ARDS when used with extreme inverse ratio.</td>
</tr>
<tr>
<td>Biphasic positive airway pressure (BiPAP/ BILEVEL/ Bivent)</td>
<td>As with APRV, the ventilator cycles between 2 preset pressure levels for defined time periods and the patient can breathe spontaneously at both pressure levels. The inspiratory time is generally shorter than, or the same length, as the expiratory time.</td>
<td>Reduced need for sedation. Activation of the diaphragm with patient triggering. Promotes alveolar recruitment.</td>
</tr>
<tr>
<td>Mandatory minute ventilation (MMV)</td>
<td>The patient's spontaneous minute ventilation is monitored by the ventilator. When the minute ventilation falls below the clinician-determined target, the ventilator increases the mandatory rate or size of tidal volumes to regain the desired minute ventilation.</td>
<td>Guarantees minute ventilation for patients with fluctuating respiratory drive and muscle innervation such as patients awakening from anaesthesia and those with Guillain–Barré.</td>
</tr>
<tr>
<td>Proportional assist ventilation (PAV)²⁶⁸</td>
<td>Delivers positive pressure throughout inspiration in proportion to patient generated effort, and dependent on the set levels of flow assist (offsets resistance) and volume assist (offsets elastance).²⁶⁹</td>
<td>Requires intact respiratory drive. Patients with high respiratory drive as the ventilator may over-assist and continue to apply support when the patient has stopped inspiration.²⁷⁰</td>
</tr>
<tr>
<td>Proportional assist ventilation (PAV⁺)²⁷⁰</td>
<td>Clinician only sets a percentage of work for the ventilator. The ventilator assesses total work of breathing by randomly measuring compliance and resistance every 4–10 breaths.</td>
<td>Requires intact respiratory drive. Decreases work of breathing and improves patient ventilator synchrony. Potential for use as a weaning mode.</td>
</tr>
<tr>
<td>Adaptive support ventilation (ASV)</td>
<td>Automatic adaptation of respiratory rate and pressure levels based on a clinician-set desired percentage of minute ventilation.²⁷⁰</td>
<td>Automatically sets all ventilator settings except PEEP and FiO₂. Potential for use as a weaning mode.</td>
</tr>
<tr>
<td>Volume assured pressure support (VAPS)</td>
<td>The ventilator switches from pressure control to volume control, or pressure support to volume control during inspiration.</td>
<td>Enables maintenance of a preset minimum VT and reduces work of breathing.</td>
</tr>
</tbody>
</table>
Synchronised intermittent mandatory ventilation

SIMV delivers breaths at a set frequency (rate), and can be either pressure- or volume-targeted. Setting of the ventilator is similar to setting VCV or PCV. The availability of patient triggering with SIMV facilitates provision of gas flow in recognition of a patient’s spontaneous effort. SIMV uses a timing window to deliver mandatory breaths in synchrony with patient inspiratory effort. Additional spontaneous breaths occurring outside of the timing window may be assisted with pressure support to augment the patient’s spontaneous effort to a pre-set pressure level.

Pressure support ventilation

Pressure support ventilation (PSV) is a spontaneous mode in which the patient initiates and cycles all breaths, with support of the patient’s inspiratory effort by the ventilator using rapid acceleration of flow to achieve a preset level of inspiratory pressure. Unlike CMV, SIMV or A/C, PSV does not require setting of ventilator (mandatory) breaths. PSV is usually employed with PEEP, which maintains partial inflation of alveoli during the expiratory phase to promote alveolar recruitment and oxygenation.

Continuous positive airway pressure

CPAP applies a set baseline positive pressure throughout the inspiratory and expiratory phases. In this spontaneous breathing mode, unlike PSV, no additional positive pressure is provided to the patient during inspiration. Due to nomenclature used on some ventilator models, PSV is frequently misrepresented as CPAP.

Volume-targeted pressure control breaths

A number of hybrid ventilator modes are commercially available that use an algorithm to target a set VT by regulating the inspiratory pressure during pressure-controlled breaths based on the patient’s resistance, compliance and inspiratory effort. Examples include pressure-regulated volume control, available on the Servo 300 and Servo 1 (Maquet, Solna, Sweden) and SIMV with autoflow (Dräger, Lübeck, Germany). On initiation of these modes, the ventilator delivers a number of breaths during a ‘learning period’ to establish an estimate of the pressure required to achieve the targeted VT. The patient’s resistance, compliance and inspiratory effort continue to influence the pressure and flow delivered to attain the targeted VT. The ventilator constantly regulates inspiratory pressure based on the pressure/volume calculation of the previous breaths and the clinician-determined target tidal volume.

Airway pressure release ventilation and biphasic positive airway pressure

Airway pressure release ventilation (APRV) and BIPAP are ventilator modes that allow unrestricted spontaneous breathing independent of ventilator cycling, using an active expiratory valve that allows patients to exhale even in the inspiratory phase. Both modes are pressure-limited and time-cycled. In the absence of spontaneous breathing, these modes resemble conventional pressure-limited, time-cycled ventilation. In North America the acronym BiPAP® is registered to Respironics non-invasive ventilators (Murrayville, PA). Therefore ventilator companies have developed brand names such as BiLevel (Puritan Bennett, Pleasanton, CA, GE Healthcare, Madison, WI) Bivent (Maquet, Solna, Sweden), DuoPAP (Hamilton Medical, Rhazins, Switzerland), PCV+ (Dräger Medical, Lübeck, Germany) or BiPhasic (Viasys, Conshohnack, PA) to describe essentially equivalent modes. Ambiguity exists in the criteria that distinguish APRV and BIPAP. When applied with the same I:E ratio, no difference exists between the two modes. APRV, as opposed to BIPAP, however, is more frequently described with an extreme inverse ratio and advocated as a method to improve oxygenation in refractory hypoxemia.

Automatic tube compensation

Automatic tube compensation is active during spontaneous breathing and compensates for the work of breathing associated with ETT resistance via closed-loop control of continuously calculated tracheal pressure. During spontaneous inspiration, a pressure gradient exists between the proximal and distal ends of the ETT due to resistance created by the tube. A reduced pressure at the proximal end of the tube means a patient needs to produce a greater inspiratory force (greater negative pressure) to generate an adequate VT. Higher flow rates generate larger pressure gradients and greater resistance. Automatic tube compensation requires the airway type and size to be selected as well as a percentage of automatic tube compensation to be applied. It appears to have most use in reducing the work of breathing for patients with high respiratory drive who require high inspiratory flow.

Neurally adjusted ventilatory assist

Neurally adjusted ventilatory assist (NAVA®) is available on the Servo-I ventilator (Maquet, Solna, Sweden) and uses the electrical activity of the diaphragm to control patient-ventilator interaction. Electrical activity of the diaphragm, measured using an oesophageal catheter, should result in optimal patient-ventilator synchrony as it represents the end point of neural output from the respiratory centres, and thus is the earliest signal of patient inspiratory trigger and expiratory cycling. Pressure delivered to the airways is proportional to inspiratory diaphragmatic electrical activity using a clinician-determined proportionality factor set on the ventilator. NAVA® provides breath-by-breath assist in synchrony with, and in proportion to, respiratory demand. Although clinical data on NAVA® are currently limited, this mode shows promise for improving patient-ventilator synchrony.
Managing the mechanically ventilated patient

Management of refractory hypoxaemia

Refractory hypoxaemia may require strategies in addition to conventional lung-protective mechanical ventilation. These include recruitment manoeuvres (RMs), high frequency oscillatory ventilation (HFOV), extracorporeal membrane oxygenation (ECMO) and nitric oxide (NO).

Recruitment manoeuvres

RMs refer to brief application of high levels of PEEP to raise the transpulmonary pressure to levels higher than achieved during tidal ventilation with the goals of opening collapsed alveoli, recruiting slow opening alveoli, preventing alveolar derecruitment and reducing shearing stress. The most common RM is elevation of PEEP to achieve a peak pressure of 40 cmH\textsubscript{2}O for a sustained period of 40 s, although studies report peak pressure elevations ranging from 25–50 cmH\textsubscript{2}O for durations ranging from 20–40 s. The best method in terms of pressure, duration and frequency has yet to be determined. RMs in humans have not produced consistent results in clinical studies, with a recent systematic review demonstrating no mortality benefit despite transient increases in oxygenation. Effective recruitment may be difficult to assess with the potential for either alveolar overdistension or failure to recruit. Once the RM is terminated, derecruitment may occur rapidly. Serious adverse effects have been noted during RMs due to increased intrathoracic and intrapulmonary pressures resulting in reductions in venous return and cardiac output, cardiac arrest and increased risk of barotrauma.

High frequency oscillatory ventilation

HFOV requires a specialised ventilator and manipulation of four variables: mean airway pressure (cmH\textsubscript{2}O), frequency (Hz), inspiratory time and amplitude (or power [AP]). Alveolar overdistension is limited through the use of sub-dead space tidal volumes whereas alveolar cyclic collapse is prevented by maintenance of high end-expiratory lung pressures. High frequency (between 3 and 15 Hz) oscillations at extremely fast rates (300–420 breaths/min) create pressure waves enabling CO\textsubscript{2} elimination. Oxygenation is facilitated through application of a constant mean airway pressure via the bias flow (rate of fresh gas). In adults, recommendations for the initiation of HFOV state mean airway pressure should be set 5 cmH\textsubscript{2}O above the peak airway pressure achieved with conventional ventilation. The recommended frequency range is 3–10 Hz with 5 Hz conventionally used to initiate HFOV. Inspiratory time is set at 33% and the amplitude setting is determined by adequate CO\textsubscript{2} elimination. Increased CO\textsubscript{2} elimination is achieved by lowering the frequency and increasing the amplitude.

HFOV is generally considered a rescue mode for adult patients with ARDS experiencing refractory hypoxaemia and failing conventional ventilation. A recent large, multicentre, randomised controlled trial of HFOV was stopped early for harm with increased mortality in the HFOV arm. A contemporaneous trial conducted in the UK found no effect for HFOV compared to usual ventilatory care on 30-day mortality in patients undergoing mechanical ventilation for ARDS.

Extracorporeal membrane oxygenation

ECMO improves total body oxygenation using an external (extracorporeal) oxygenator, while allowing intrinsic recovery of lung pathophysiology by resting the lung. Indications for ECMO include acute severe cardiac or respiratory failure such as severe ARDS and refractory shock or as a bridge to transplantation. Due to the need for rescue treatment with ECMO during the 2009 H1N1 outbreak and a randomised clinical trial indicating a survival benefit with venous–venous ECMO compared to conventional ventilation, ECMO is being used more frequently for refractory respiratory failure. Bleeding as a complication of anticoagulation is a major risk of ECMO, with cerebral bleeds being the most catastrophic. Another serious complication is limb ischaemia when the femoral artery is used.

ECMO consists of three key components:

1. a blood pump (either a simple roller or centrifugal force pump)
2. a membrane oxygenator (bubble, membrane or hollow fibre)
3. a countercurrent heat exchanger, where the blood is exposed to warmed water circulating within metal tubes.

In addition, essential safety features include: bubble detectors that detect gas in the arterial line and shut the pump off; arterial line filters between the heat exchanger and arterial cannula, to trap air thrombi and emboli; pressure monitors placed before and after the oxygenator that measure the pressure within the circuit and detect rising circuit pressures commonly caused by thrombus or circuit or cannulae occlusion; and continuous venous oxygen saturation and temperature monitoring. On commencement of ECMO the circuit is primed with fresh blood. The acid–base balance and blood gas of the primer is adjusted to ensure that the pH is within the normal range (7.35–7.45) and PaO\textsubscript{2} is adequate. ECMO can be delivered via veno-arterial access, which requires cannulation of an artery. This method bypasses the pulmonary circulation while providing cardiac support to the systemic circulation and achieves a higher PaO\textsubscript{2} with lower perfusion rates. The alternative is veno-venous access, used for patients in respiratory failure with adequate cardiac function as there is no support of systemic circulation. Perfusion rates are higher, the mixed venous PO\textsubscript{2} is elevated and the PaO\textsubscript{2} is lower.

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**Nitric oxide**

NO is an endothelial smooth muscle relaxant. Inhaled NO is effective for dilation of pulmonary arteries resulting in reduced pulmonary shunting and reduced right ventricular afterload due to reduced pulmonary artery tone. Pulmonary shunting refers to failure of uptake of alveolar gas by the pulmonary vascular bed due to vascular constriction or interstitial oedema. Inhaled NO has a role in the management of pulmonary hypertension and was previously thought to have a role in management of refractory hypoxaemia for patients with ARDS. However, the most recent systematic review and meta-analysis of NO in ARDS comprising 14 RCTs and 1303 participants reported no effect on overall mortality despite a statistically significant improvement in oxygenation in the first 24 hours.215

**Positioning**

Regular repositioning of critically ill patients is essential for lung recruitment, prevention of atelectasis and maintenance of skin integrity (see Chapter 6).

**Head-of-bed elevation**

Supine positioning has been associated with aspiration of abnormally colonised oropharyngeal and gastric contents216–218 and increased incidence of VAP compared to a semirecumbent position, defined as backrest elevation at 45°.219 Guidelines and care bundles for VAP prevention recommend semirecumbent positioning for all mechanically ventilated patients.96,220,221 A more recent trial has, however, questioned the feasibility of 45° semirecumbent positioning as this backrest elevation was only achieved for 15% of study observations.222 There was also no difference in VAP incidence between the supine and semirecumbent group. Contraindications to backrest elevation include:

- suspected or existing spinal injury
- intracranial hypertension (for 45° elevation)
- unstable pelvic fractures
- prone positioning
- haemodynamic support devices (intra-aortic balloon pumps, left ventricular assist devices and ECMO)
- femoral catheterisation for continuous renal replacement therapy
- large abdominal wounds
- following femoral sheath removal.

As some degree of semirecumbent positioning is preferable to supine positioning, patients with suspected or existing spinal injury, pelvic fractures or being managed with prone positioning can have the head elevated by tilting the whole bed. Patients with femoral cannulation and large abdominal wounds can usually achieve 25–30° positioning.

Clinical practice audits conducted internationally and in Australia and New Zealand indicate that compliance with a 45° semirecumbent position rarely occurs, even when taking into consideration contraindications.223–227 Similarly, interventions to improve compliance failed to demonstrate adherence to the 45° semirecumbent position that can be sustained by the patient over time.228,229 Due to uncertainty over compliance with 45° semirecumbent positioning in the original trial conducted by Drakulovic,219 and the lack of difference in VAP rates despite difficulty achieving compliance with semirecumbency in the van Niewenhoven study,222 new studies are required to confirm the equivalence or lack of inferiority of lower degrees of backrest elevation to the strict 45° semirecumbent position.

**Practice tip**

Backrest elevation is difficult to estimate accurately. Use an objective measurement device such as an inclinometer or protractor.

**BOX 15.3**

**Mechanical ventilation and the bariatric patient**

- Bariatric patients are at increased risk of atelectasis and have decreased chest wall compliance due to the weight of the abdomen.
- Avoid the supine position as this will further decrease lung volumes.
- Bariatric patients may require higher airway pressures to generate adequate tidal volumes.
- Recruitment manoeuvres may improve oxygenation.
- Frail elderly may experience difficult weaning due to the presence of comorbidities such as CHF, ischaemic heart disease and COPD.

**Lateral positioning**

Patients with unilateral lung disease experience a mismatch of ventilation to perfusion if the consolidated or atelectic lung is placed in the dependent position.231 Temporary and early positioning of the affected lung in the dependent position, amongst other strategies such as avoiding manual hyperinflation, for patients with unilateral pneumonia or following aspiration may be beneficial in preventing the movement of bacteria or acidic gastric contents into the non-affected lung.232 This theory has been coined ‘propagation prevention’. While appealing, as yet there have been no adequately powered randomised controlled trials to support its use. Continuous lateral rotational therapy is a positioning therapy advocated for the prevention and management of respiratory complications associated with immobility.233 The most recently reported multi-centre randomised controlled trial found a significant reduction in VAP and shorter durations of ventilation and ICU stay.234 Continuous lateral rotation therapy requires a special bed system enabling rotation of the upper part of the body to a maximum angle of 90°.
Prone positioning

Prone positioning has been shown to improve oxygenation and intrapulmonary shunt fraction when compared with rotational turning during the first 72 hours of ARDS and in patients with multiorgan failure. Prone positioning may also decrease the risk of VAP due to improved bronchial secretions drainage, limitation of colonisation of distal lung, decreased atelectasis, and increased alveolar recruitment but may increase the spread of pathogens in the lung and may increase the risk of aspiration.

Prone positioning results in changes to the distribution of ventilation and pulmonary blood flow. Pleural pressures are lower in non-dependent regions and higher in dependent regions due to gravitational forces, the weight of the overlying lung and mismatch between the local physical structures of the lung and chest wall. The weight of the overlying lung increases in ARDS due to parenchymal oedema and fluid within the alveoli. This gradient in pleural pressures means transpulmonary pressure is higher in non-dependent lung regions, compared to dependent regions. Perfusion also increases from previously non-dependent to dependent lung regions resulting in optimal matching of ventilation and perfusion to promote gas exchange.

Increased pleural pressure in the dependent dorsal regions in the supine position can result in airway closure, atelectasis and hypoxaemia. The difference in pleural pressures from non-dependent and dependent lung regions is greater in the supine compared to the prone position. In the supine position, the heart and abdominal contents also compress lung bases and decrease functional residual capacity, whereas in prone positioning, the weights of these structures are lifted from the lung.

The benefits of prone positioning continue to be debated. Although oxygenation improves in 70–80% of patients turned from supine to prone, a mortality benefit has not been shown in all trials. The most recent systematic review and meta-analysis that included 11 randomised controlled trials found reduced mortality and improved oxygenation for patients with ARDS managed with protective lung ventilation. Adverse events related to prone positioning were increased risk of decubitus ulcer formation, endotracheal obstruction and thoracotomy tube dislodgement.

Implementing prone positioning requires forward planning to ensure eye care and protection, mouth care, wound dressings and tracheal suction are attended to before positioning the patient prone. Intravenous lines, electrocardiogram leads, urinary catheter drainage, chest drains and ostomy bags need to be secured and repositioned appropriately once the patient is positioned. Prone positioning can be achieved by manual handling of the patient, requiring up to five staff, although commercial devices are available that facilitate the turning and positioning.

Complications of mechanical ventilation

Physiological complications associated with mechanical ventilation include ventilator-associated lung injury and nosocomial infection, including VAP. Ventilator-associated lung injury occurs through alveolar overdistension and cyclic opening and closing of alveoli resulting in diffuse alveolar damage, increased permeability, pulmonary oedema, cell contraction and cytokine production. VAP substantially increases the duration of ICU stay and is associated with attributable mortality of 5.8–8.5%. Additional complications associated with mechanical ventilation are listed in Table 15.8. Complications can occur due to inappropriate application of mechanical ventilation. This may result in extra-alveolar gas causing pneumothoraces or subcutaneous emphysema due to high peak Ppeak and alveolar stretch and oedema formation as a result of large f/VT.

Weaning from mechanical ventilation

Weaning traditionally occurs via clinician-directed adjustments to the level of support provided by the ventilator, culminating in a spontaneous breathing trial (SBT) comprising either low level pressure support or a T piece trial.

Current recommendations

No ventilation strategy is more lung-protective than the timely and appropriate discontinuation of mechanical ventilation. Weaning refers to the transition from ventilatory support to spontaneous breathing. Evidence-based consensus guidelines published for weaning in 2001 and 2007 emphasise the importance of preventing unnecessary delays in the weaning process, early recognition of a patient’s ability to breathe spontaneously and the use of a systematic method to identify the potential for extubation.

Weaning predictors

Clinician judgement regarding prediction of weaning readiness is known to be imperfect, with unnecessary prolongation of ventilation or high rates of reintubation as resultant consequences, both of which are associated with adverse outcomes. An evidence-based review evaluating over 50 objective physiological measurements for determining readiness for weaning and extubation found most had only a modest relationship with weaning outcome; no single factor or combination of factors demonstrated superior accuracy. Of all predictors studied, the respiratory frequency to tidal volume ratio (f/VT) appears to be most accurate. However, inclusion of the f/VT as part of a weaning protocol was found in one randomised study to increase, as opposed to decrease, the duration of weaning. At present, consensus guidelines do not recommend routine inclusion of weaning predictors.

Weaning methods

Various studies have attempted to identify the best weaning method. Two of the most frequently-cited
studies have produced conflicting results. Brochard and colleagues261 compared PSV, T piece trials and SIMV, and concluded that PSV reduced the duration of mechanical ventilation compared with the other methods. Esteban and colleagues262 compared PSV, T piece trials, CPAP and progressive reduction of SIMV support, and found a once-daily T piece trial led to extubation three times more quickly than SIMV and nearly twice as quickly as PSV. A recent systematic review comparing trials of PSV and T piece found no clear evidence of a difference between these weaning strategies for weaning success.263 Failure to produce consistent results favouring a single weaning style suggests it is not the mode that is important but rather the application of a systematic process.264

### Spontaneous breathing trials

SBTs incorporate a focused assessment of a patient’s capacity to breathe prior to extubation265 and are recommended as the major diagnostic test to determine extubation readiness.264 SBTs can be conducted using either a T piece or low levels of pressure support266 and should need to last only 30 minutes.267 This method of weaning is less common in Australia and New Zealand, in contrast to international findings.78,79

### Weaning protocols

Implementation of various organisational strategies such as weaning teams and non-physician-led weaning protocols may assist in the timely recognition of weaning and extubation readiness.268–272 Coupling of a sedation and weaning protocol was found to result in a three-day reduction in the duration of ventilation compared to standard care in four North American hospitals.273 A systematic review and meta-analysis of 11 weaning protocol trials including 1971 patients demonstrated a reduction in the duration of mechanical ventilation.274 However, the authors cautioned that the effect of weaning protocols may vary according to ICU organisational characteristics such as an intensivist-led ICU model, high levels of physician staffing, structured ward rounds, collaborative discussion and more frequent medical review; all are characteristics reported for ICUs in Australia and New Zealand.275,276

### Automated weaning

Automated computerised systems potentially enable more efficient weaning by providing improved adaptation of ventilatory support through continuous monitoring and real-time intervention.277 One such

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### TABLE 15.8 Complications of mechanical ventilation

<table>
<thead>
<tr>
<th>ITEM</th>
<th>COMPLICATION(S)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barotrauma</td>
<td>Pneumothorax, Pneumomediastinum, Pulmonary interstitial emphysema, Subcutaneous emphysema</td>
</tr>
<tr>
<td>Volutrauma</td>
<td>Shearing stress, endothelial and epithelial cell injury, fluid retention and pulmonary oedema, perivasculary and alveolar hemorrhage, alveolar rupture</td>
</tr>
<tr>
<td>Volutrauma</td>
<td>Shearing stress, endothelial and epithelial cell injury, fluid retention and pulmonary oedema, perivasculary and alveolar hemorrhage, alveolar rupture</td>
</tr>
<tr>
<td>Biotrauma</td>
<td>Activation of systemic and local inflammatory mechanisms</td>
</tr>
<tr>
<td>Ventilation/perfusion</td>
<td>Alveolar distension causes compression of the adjacent pulmonary capillaries resulting in dead space ventilation</td>
</tr>
<tr>
<td>mismatch</td>
<td></td>
</tr>
<tr>
<td>↓ Cardiac output</td>
<td>Resulting in hypotension, ↓ cerebral perfusion pressure (CPP), ↓ renal and hepatic blood flow</td>
</tr>
<tr>
<td>↑ Right ventricular</td>
<td>Due to ↑ intrathoracic pressure may result in ↓ left ventricular compliance and preload</td>
</tr>
<tr>
<td>afterload</td>
<td></td>
</tr>
<tr>
<td>↓ Urine output</td>
<td>Due to ↓ glomerular filtration rate, ↑ sodium reabsorption and activation of the renin-angiotensin-aldosterone system</td>
</tr>
<tr>
<td>Fluid retention</td>
<td>Due to above renal factors as well as ↑ antidiuretic hormone and ↓ atrial natriuretic peptide</td>
</tr>
<tr>
<td>Impaired hepatic function</td>
<td>Due to ↑ pressure in the portal vein, ↓ portal venous blood flow, ↓ hepatic vein blood flow</td>
</tr>
<tr>
<td>↑ Intracranial pressure</td>
<td>Due to ↓ cerebral venous outflow</td>
</tr>
<tr>
<td>Oxygen toxicity</td>
<td>Alterations to lung parenchyma similar to those found in ARDS</td>
</tr>
<tr>
<td>Pulmonary emboli and deep</td>
<td>Due to immobility</td>
</tr>
<tr>
<td>vein thrombosis</td>
<td></td>
</tr>
<tr>
<td>Ileus, diarrhoea</td>
<td>Due to alterations in gastric motility</td>
</tr>
<tr>
<td>Gastrointestinal haemorrhage</td>
<td>Gastritis and ulceration may occur due to stress, anxiety and critical illness</td>
</tr>
<tr>
<td>ICU-acquired weakness</td>
<td>Neuropathies and myopathies develop in association with critical illness, corticosteroids and neuromuscular blockade</td>
</tr>
<tr>
<td>Psychological issues</td>
<td>Delirium, anxiety, depression, agitation and post-traumatic stress disorder may be experienced by critically ill ventilated patients in the acute and recovery phases</td>
</tr>
</tbody>
</table>
system, SmartCare\textsuperscript{TM}/PS, monitors three respiratory parameters, \( f, V_T \) and end-tidal carbon dioxide concentration, every 2 or 5 minutes and periodically adapts PS.\textsuperscript{277,278} SmartCare\textsuperscript{TM}/PS establishes a respiratory status diagnosis, based on evaluation of the three parameters, and may either decrease or increase PS, or leave it unchanged to maintain the patient in a defined ‘respiratory zone of comfort’.\textsuperscript{279,280} Once SmartCare\textsuperscript{TM}/PS has successfully minimised the level of PS, a 1-hour observation period occurs. For patients who remain within the respiratory zone of comfort throughout the observation period, SmartCare\textsuperscript{TM}/PS recommends to ‘consider separation’, indicating the patient’s respiratory status now suggests the patient will tolerate extubation.

A recent meta-analysis of automated weaning systems has shown that SmartCare\textsuperscript{TM}/PS may reduce the duration of weaning, total ventilation and ICU length of stay when compared to protocolised or usual care weaning.\textsuperscript{281}

The difficult-to-wean patient

International reports indicate patients that require mechanical ventilation for \( \geq 21 \) days account for less than 10\% of all mechanically ventilated patients, but occupy 40\% of ICU bed days and accrue 50\% of ICU costs.\textsuperscript{282,283} A recommendation from the National Association for Medical Direction of Respiratory Care states that prolonged mechanical ventilation should be defined as ‘\( \geq 21 \) consecutive days of ventilation required for \( \geq 6 \) hours per day’.\textsuperscript{254} Prolonged weaning has been defined as \( >7 \) days of weaning after the first SBT or more than three SBTs.\textsuperscript{254} Little evidence defines the optimal method for managing the difficult-to-wean patient. One trial found no difference in weaning duration or success when comparing tracheostomy trials to low-level pressure support in patients with COPD experiencing weaning difficulty.\textsuperscript{284} A recent randomised controlled trial demonstrated increased weaning success with use of a once daily progressive tracheostomy mask trial compared to pressure support weaning.\textsuperscript{285} These patients are most likely to benefit from an individualised and structured approach to weaning using progressive lengthening of tracheostomy trials with supportive ventilation in between in combination with early physical therapy.

Practice tip

Tachypnoea and decreased \( V_T \) during weaning are indicators that a patient is not ready for extubation.

Summary

Support of oxygenation and ventilation during critical illness are key activities for nurses in ICU. Oxygen therapy promotes aerobic metabolism but has adverse effects that need to be considered. Various oxygen delivery devices provide low or variable flows of oxygen.

Strong evidence supports the use of NIV for COPD and CHF, but caution is required when it is used for other diagnoses such as pneumonia. NIV success is dependent on patient tolerance, with common complications including pressure ulcers, conjunctival irritations, nasal congestion, insufflation of air into the stomach and claustrophobia.

Airway support can be provided with oro- or nasopharyngeal airways, LMAs and endotracheal intubation; oral intubation is the preferred method. For a patient with an ETT, the key points for practice are:

- ETT placement should be confirmed with end-tidal CO\textsubscript{2} monitoring
- the aim of endotracheal cuff management is to prevent airway contamination and enable positive pressure ventilation
- closed suctioning reduces alveolar derecruitment compared to open suctioning
- instillation of normal saline is not recommended during routine tracheal suctioning.

The optimal timing of tracheostomy remains uncertain; however, tracheostomy should be considered for patients experiencing weaning difficulty.

The goals of mechanical ventilation are to promote gas exchange, minimise lung injury, reduce work of breathing and promote patient comfort:

- Despite its life-saving potential, mechanical ventilation carries the risk of serious physical and psychological complications.
- Humidification of dry medical gas is required during mechanical ventilation to prevent drying of secretions, mucus plugging and airway occlusion.
- The pressure required to deliver a volume of gas into the lungs is determined by elastic and resistive forces.
- Contemporary ventilators now provide a range of modes to facilitate mechanical ventilation.
- Analysis of ventilator graphics provides clinicians with the ability to assess patient–ventilator interaction, appropriateness of ventilator settings and lung function.
- Semirecumbent positioning at 45\° elevation has been shown to reduce VAP but compliance is poor.
- RMs, HFOV, ECMO and prone positioning are strategies that may facilitate management of refractory hypoxaemia.
Case study

A 60-year-old female, Martha, was admitted to the ICU with acute respiratory failure due to community-acquired pneumonia. Martha was morbidly obese (190 kg) with extensive central obesity and a body mass index of 74.2. She had a history of COPD but was not prescribed steroids and had not been investigated for sleep apnoea.

Martha was commenced on broad spectrum antibiotic cover in the emergency department. On arrival to the ICU, Martha was placed in an isolation room, and respiratory isolation using droplet precautions for possible H1N1 were initiated. The patient was commenced on oral oseltamivir at 150 mg twice a day. Martha had a trial of NIV with FiO₂ 0.7, PEEP 7 cmH₂O (EPAP 7 cmH₂O) and pressure support 5 cmH₂O (IPAP 12 cmH₂O) to reduce her work of breathing and improve gas exchange. The NIV trial was discontinued as Martha’s dyspnoea was unrelieved, and hypoxia and hypercapnia persisted. She was intubated with a size 7 oral ETT and a bronchial alveolar lavage was performed to obtain samples for bacterial and viral screening. Nasopharyngeal swabs were also obtained. Ventilator settings following intubation were A/C, FiO₂ 1.0, respiratory rate 16, P_{insp} 30 cmH₂O, PEEP 15 cmH₂O and inspiratory time of 1.1 seconds. Initial blood gases were as follows: pH 7.07, PaO₂ 71 mmHg, PaCO₂ 71 mmHg, HCO₃⁻ 16.4 mmol, base excess -9.5, sodium 123 mmol, chloride 94 mmol, lactate 0.7, SpO₂ 94% and PaO₂/FiO₂ (PF) ratio 71. Dynamic compliance was 25.6 mL/cmH₂O, resistance was 8.6 cmH₂O/(L/s). A chest X-ray showed bilateral pulmonary infiltrates and a lobular pneumonia. Chest auscultation revealed bilateral crackles, late in the inspiratory phase.

Nursing assessment indicated the following issues:

1. Notable audible cuff leak on inspiration despite a cuff pressure of 30 cmH₂O
2. Atelectasis as evidenced by decreased air entry in lung bases, reduced compliance, diminished gas exchange and obliteration of costophrenic angles on the chest X-ray.

To address the cuff leak, nursing staff connected rigid manometer tubing between the cuff pressure gauge and the ETT pilot tube to enable continuous cuff pressure measurement. Cuff pressure did not decrease over time indicating that the ETT cuff was intact. Therefore, the audible air leak was not caused by a leaking ETT cuff but was due to an air leak around the cuff. On careful examination of the chest X-ray the ETT cuff was found to be above the level of the vocal cords and therefore needed repositioning.

To address the atelectasis, Martha was repositioned in a high semi-Fowler position (≥45° HOB elevation). This change in positioning resulted in an immediate improvement in compliance from a baseline of 25.6 to 38.4 mL/cmH₂O. V₆ also increased from 300 mL to 400 mL. These improvements enabled rapid downward titration of FiO₂ to 0.7 while maintaining SpO₂ >90%. An RM using 40 cmH₂O for 40 seconds was performed with further improvement of Martha’s oxygenation indicating her lungs were responsive to this strategy. The ventilator mode was changed to APRV with a P_{insp} of 27 cmH₂O for 6 seconds and an expiratory pressure of 5 cmH₂O for 0.4 seconds. Further improvements in oxygenation were noted (PaO₂ 180 mmHg and PF ratio 225).

**DISCUSSION**

A cuff leak may be assumed to be secondary to a hole in either the cuff or pilot tube; however, this is relatively rare. Audible cuff leaks are more frequently due to a malpositioned ETT. It is important to note that each time cuff pressure is measured, a small volume of gas leaves the cuff to pressurise the pressure gauge. Repeated cuff pressure measurement may cause reduced cuff pressure over time, which may be falsely assumed to indicate the cuff is losing volume due to other causes. Attaching rigid tubing between the cuff and pressure gauge eliminates this problem and facilitates continuous cuff pressure measurement. This is a useful strategy for assessing cuff leak problems. In this case scenario, careful troubleshooting averted the need for ETT replacement and avoided unnecessary risk to the patient.

Patient positioning is extremely important in managing the bariatric patient. Central obesity causes cephalic displacement of the diaphragm resulting in a positive pleural pressure and subsequent alveolar collapse. Inspiratory crackles late in the inspiratory phase indicate late alveolar opening and an increased potential for lung injury due to cyclic alveolar inflation and deflation. Positive pleural pressure decreases transpulmonary pressure and often necessitates the use of higher levels of PEEP to prevent collapse. Positioning in the high semi-Fowler position can have a dramatic and positive effect on lung mechanics for these patients evidenced by the increase in compliance in this case study. RMs typically have a limited
period of effectiveness, i.e. derecruitment generally occurs following the manoeuvre. APRV maintains the higher level of pressure for a prolonged time thus sustaining alveolar recruitment. In this case study APRV and position changes appeared to promote recruitment and improved oxygenation enabling downwards titration of the FiO₂. When considering extubation for the bariatric patient, maintaining PEEP at a high level prior to extubation and using CPAP following extubation may prevent alveolar derecruitment.

**CASE STUDY QUESTIONS**

1. In this case study Martha received relatively low levels of PEEP and pressure support prior to intubation. What are the potential advantages and disadvantages of increasing these parameters for this patient?

2. What is the rationale for using oxygen therapy in patients with COPD and low SpO₂?

3. Explain why Martha’s lung compliance increased when positioning was changed from the supine to high semi-Fowler position.

**RESEARCH VIGNETTE**


**Abstract**

**Background:** Previous trials involving patients with the ARDS have failed to show a beneficial effect of prone positioning during mechanical ventilatory support on outcomes. We evaluated the effect of early application of prone positioning on outcomes in patients with severe ARDS.

**Methods:** In this multicenter, prospective, randomized, controlled trial, we randomly assigned 466 patients with severe ARDS to undergo prone-positioning sessions of at least 16 hours or to be left in the supine position. Severe ARDS was defined as a ratio of the partial pressure of arterial oxygen to the FiO₂ of less than 150 mmHg, with a FiO₂ of at least 0.6, a positive end-expiratory pressure of at least 5 cmH₂O, and a tidal volume close to 6 mL per kilogram of predicted body weight. The primary outcome was the proportion of patients who died from any cause within 28 days after inclusion.

**Results:** A total of 237 patients were assigned to the prone group, and 229 patients were assigned to the supine group. The 28-day mortality was 16.0% in the prone group and 32.8% in the supine group (P<0.001). The hazard ratio for death with prone positioning was 0.39 (95% confidence interval [CI], 0.25 to 0.63). Unadjusted 90-day mortality was 23.6% in the prone group versus 41.0% in the supine group (P<0.001), with a hazard ratio of 0.44 (95% CI, 0.29 to 0.67). The incidence of complications did not differ significantly between the groups, except for the incidence of cardiac arrests, which was higher in the supine group.

**Conclusion:** In patients with severe ARDS, early application of prolonged prone-positioning sessions significantly decreased 28-day and 90-day mortality.

**Critique**

This well conducted randomised controlled trial has a number of strengths. Patients with severe ARDS (defined as a PF ratio of <150 mmHg, with an FiO₂ of ≥0.6, PEEP of ≥5 cmH₂O and a Vₜ of approximately 6 mL per kilogram of predicted body weight) were recruited early (within 36 hours of ventilation) ensuring the intervention was applied in the early phase of ARDS and not as a rescue measure. Patients were assessed for 12 to 24 hours prior to randomization, thus confirming the presence of severe ARDS. All patients received standardised protective lung ventilation thereby removing the style of ventilation as a potential confounder. In the intervention arm, all patients were proned within 1 hour of randomisation and proning sessions extended for at least 16 consecutive hours, thereby producing maximal effects of proning. Considerations for translation of this research into practice are as follows. All participating centres had extensive (greater than 5 years) experience with proning. The study found no difference in adverse event rates between the prone and supine groups. This finding may not be generalisable to centres without this level of experience. Additionally, the notable reduction in 28-day mortality found in this study with proning applies to patients with severe ARDS. Again, the findings are not generalisable to patients with mild-to-moderate ARDS.
Learning activities

1. Describe how the terms IPAP and EPAP used on some NIV ventilators correlate with the more generic terms of PEEP and pressure support.
2. Why is it important to consider the patient’s respiratory rate and tidal volume when using a low flow (variable flow) oxygen delivery device?
3. How do increasing PEEP and recruitment manoeuvres increase oxygenation?
4. Identify some of the potential risks of recruitment manoeuvres and the nursing observations to detect signs of deterioration.
5. Explain how a reduction in the FiO₂ from 1.0 to 0.8 can increase the SpO₂.

Online resources

American Association for Respiratory Care, www.aarc.org/resources
Anaesthesia UK, www.frca.co.uk/default.aspx
ARDS network, www.ardsnet.org/
College of Intensive Care Medicine of Australia and New Zealand, www.cicm.org.au
Covidien education resources, www.nellcor.com/educ/OnlineEd.aspx
Critical Care Medicine Tutorials, www.ccmtutorials.com
Department of Anaesthesia and Intensive Care, Chinese University of Hong Kong, http://aic-server4.aic.cuhk.edu.hk/web8
Intensive Care Coordination and Monitoring Unit, http://intensivecare.hsnet.nsw.gov.au
Vent World, www.ventworld.com

Further reading


References


