Chapter 3
The patient who needs respiratory support

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NMC Standards for Pre-registration Nursing Education

This chapter will address the following competencies:

Domain 3: Nursing practice and decision-making

Generic competencies:

3. All nurses must carry out comprehensive, systematic nursing assessments that take account of relevant physical, social, cultural, psychological, spiritual, genetic and environmental factors, in partnership with service users and others through interaction, observation and measurement.
4. All nurses must ascertain and respond to the physical, social and psychological needs of people, groups and communities. They must then plan, deliver and evaluate safe, competent, person-centred care in partnership with them, paying special attention to changing health needs during different life stages, including progressive illness and death, loss and bereavement.

Field-specific competencies:

3.1. Adult nurses must safely use a range of diagnostic skills, employing appropriate technology, to assess the needs of service users.

4.1. Adult nurses must safely use invasive and non-invasive procedures, medical devices, and current technological and pharmacological interventions, where relevant, in medical and surgical nursing practice, providing information and taking account of individual needs and preferences.

NMC Essential Skills Clusters

This chapter will address the following ESCs:

Cluster: Care, compassion and communication

3. People can trust the newly registered graduate nurse to respect them as individuals and strive to help them preserve their dignity at all times.

By entry to the register:

v. Is proactive in promoting and maintaining dignity.

Chapter aims

By the end of this chapter, you should be able to:

- identify why patients may need advanced respiratory (ventilatory) support;
- demonstrate an awareness of the importance of arterial blood gas analysis in the management of patients with respiratory failure;
- describe non-invasive ventilation (NIV) and mechanical invasive ventilation (MIV);
- demonstrate an awareness of the factors influencing the choice of appropriate respiratory support;
- describe the fundamentals of providing a safe holistic approach to caring for patients receiving NIV and MIV.

Introduction

In this chapter you are introduced to Mrs Jenny Matthews. She is 43 years old and for 20 years has had a history of acute exacerbation of asthma. She had eczema as a child and hay fever but wasn't diagnosed with asthma until she was in her twenties. We will follow her on her journey through healthcare as she experiences an acute exacerbation of asthma. The scenario box below provides a summary of her admission to the emergency department.

Scenario: Jenny Matthews

Situation

Mrs Jenny Matthews, age 43 years.

Admitted to the emergency department with a seven-day history of shortness of breath and productive cough. She had previously been seen by her GP and treated with a combination of broad spectrum antibiotics and an increased dose of salbutamol. She was progressing well at home but a sudden onset of increased shortness of breath at 2 a.m. on the morning of the eighth day prompted a 999 call for help from her husband.

Background

History of acute asthma for 20 years. Jenny has been admitted to hospital on five occasions during the last seven years. On the last occasion she required emergency intubation and ventilation, staying in intensive care for 48 hours.

She did smoke 30 cigarettes a day for 22 years but has reduced this to ten a day. She has a strong family history of reactive airways disease.

Assessment in the emergency room

Airway (A): *Patient is agitated and struggling to breathe.*

Breathing (B): *Dyspnoea, with use of her accessory muscles.*
Unable to complete a full sentence when responding to questions.

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Chest auscultation indicates an expiratory wheeze and bilateral crackles.
Respiration (R): 36 bpm.
SpO_2 91%.
Blood gases showed:
PH: 7.42
PaO_2: 8.7 kPa
$PaCo_2$: 3.6 kPa
HCO_3: 24 mmol/L.
Peak expiratory flow rate (PEF): 120 ml/min (Jenny's normal PEF: 300 ml/min).

Circulation (C): HR: 125/min.
BP: 125/72 mmHg.
Disability (D): Blood glucose: 6.8 mmol/L.
Alert and agitated.
Exposure (E): Temp: 37.4°C.

Recommendation

Humidified high-flow oxygen 60%.
Salbutamol 5 mg nebulisers continuously until improvement in PEF.
Hydrocortisone 100 mg IV.

Aminophylline infusion.

IV antibiotics.
Plan to transfer to high dependency for monitoring and evaluation of her treatment.

Jenny's husband, Brendan, has arrived and when he is informed that she needs to be admitted to the high dependency unit he becomes very angry and starts shouting at his wife 'I told you this would happen, I've had enough of this! Why didn't you just give up smoking! I'm going home, you're on your own now!' Jenny seems not to be listening and Brendan Matthews is asked to leave. The staff invite him to stay in a quiet room and approach him for more information. At this point he refuses to stay and instead gives the GP's address and Jenny's parents' phone number saying that he has had enough and he's leaving her.

Why did Jenny's situation deteriorate at home and lead to an emergency admission to HDU?

Less than 24 hours ago Jenny was seemingly making a good recovery from a chest infection when she experienced a sudden deterioration in her condition. Asthma is a chronic inflammatory disorder of the mucosal lining of the bronchi which is associated with bronchial hyper-responsiveness, reversible airway constriction and variable airflow obstruction (McCance and Huether, 2014). Factors involved in triggering an acute asthma attack can include:

- evidence of a family history of asthma;
- exposure to an allergen;
- urban residence;
- air pollution;
- tobacco smoke;
- recurrent respiratory tract infections;
- psychological factors and anxiety.

In Jenny's case, after seven days of antibiotic treatment, she decided it was time to resume smoking, as this was her main way of coping with the stresses of life. This triggered a long and aggressive argument with her husband that continued over the course of the evening. It was later that night that Jenny developed the acute exacerbation of asthma reported in her story. According to Polosa and Thompson (2013) cigarette smoking in asthma is associated with a higher frequency and severity of exacerbations and a higher risk of mortality than non-smokers. There is also evidence to suggest that anxiety and depression is often associated with smoking and an increased frequency of exacerbations for patients with asthma (Leader et al., 2014). For Jenny, the combination of tobacco smoke, stress, a recent respiratory tract infection and her family history combined to trigger the asthma attack.

When Jenny was admitted to the emergency room she was presenting signs and symptoms of the 'early response' phase. According to McCance and Huether (2014) this phase is initiated by exposure to the inhaled irritant and triggers a cascade of inflammatory events that lead to acute and chronic airway dysfunction. The combined impact of mast cell activation releasing vasoactive mediators with degranulation of their inflammatory mediators and an immune activation, leads to:

- vasodilation and increased capillary permeability;
- vascular congestion;
- bronchospasm;
- increased contractile response of the bronchial smooth muscle;
- mucus secretion;
- thickening of airway walls.

This cascade of events leads to bronchial hyper-responsiveness and airway obstruction.

For Jenny the presence of a PEF of less than 50% of her predicted normal range, a respiratory rate of 36 bpm, a pulse of 125 bpm and an inability to complete a sentence in one breath indicated the presence of acute severe asthma (BTS and SIGN, 2014). With oxygen saturations of 91% and signs of type 1 respiratory failure indicated by her arterial blood gas result of PaO_2 8.7 kPa (see Chapter 2), Jenny's condition was becoming life threatening and she required high dependency (level 2) care (BTS and SIGN, 2014).

Why are the arterial blood gas results significant?

In the body, acids (substances that release hydrogen ions (H^+) in solution) are constantly being produced as by-products of normal cell metabolism. For example, the metabolism of proteins produces acids such as sulphuric acid and hydrochloric acid. During the metabolism of carbohydrates about 15,000 mmol of carbon dioxide (CO_2) is produced each day, and although it is not an acid, it is influential in maintaining pH balance.

Carbon dioxide is transported in the circulation in the following ways.

- 20% of CO_2 is attached to haemoglobin and carried as carbaminohaemoglobin ($HHbCO_2$).
- 10% is dissolved in the plasma as carbonic acid (H_2CO_3).
- 70% of CO_2 is carried as a bicarbonate base (a substance that uses up hydrogen ions). Carbonic acid in the presence of an enzyme called carbonic anhydrase is converted to bicarbonate ions (HCO_3^-) and hydrogen ions (H^+).

The relationship between carbonic acid and bicarbonate is a very important factor in how the body regulates the pH (the calculated acidity of the blood) in the circulation, as well as other buffer systems such as the kidney and renal excretion of hydrogen ions. In order to maintain a pH of 7.4 (normal blood pH) the ratio between carbonic acid and bicarbonate should stay at one part carbonic acid to 20 parts bicarbonate (1 H_2CO_3: 20 $HCO3^- + H^+$). This means that if the amount of $HCO3^-$ in the blood falls so must the amount of H_2CO_3 in order to maintain a ratio of 1:20. The body achieves this by increasing the rate and depth of respiration so that more CO_2 is eliminated through respiration and the ratio is maintained. This is called respiratory compensation and can be seen in patients who are producing an excess of metabolic acids such as lactic acid in shock (Chapters 6 and 7) and ketone acid in diabetic ketoacidosis (Chapters 8 and 12).

Left in the circulation, an imbalance in acids or bases would destroy cells and organs, so it is imperative the body has ways to maintain a pH balance at a pH value of between 7.35 and 7.45 in order to maintain normal cell function (Hall, 2011). Should the pH value fall above or below this range, the impact on the body can be critical and in extreme cases lead to death. The body also needs to maintain acid-base balance inside cells so that cells continue to function effectively and intracellular proteins, such as haemoglobin, help to buffer acids inside cells.

What should we be monitoring?

The results obtained from analysis of arterial blood provides information about a number of factors involved in the process of acid-base balance as well as information about the amount of oxygen available to the cells. These include:

- pH value of arterial blood;
- the amount of O_2 in arterial blood (expressed as the partial pressure of oxygen or PaO_2);

- the amount of CO_2 in arterial blood (expressed as the partial pressure of carbon dioxide or $PaCO_2$);
- the amount of bicarbonate and bases available to buffer acids in arterial blood (expressed as mmol/l);
- arterial blood potassium levels;
- arterial haemoglobin;
- blood urea nitrogen, creatinine and glomerular filtration rate to monitor kidney function.

Why are these values important?

The body has a number of ways of maintaining the acid-base balance in health and we will look at five now.

Buffer systems are control mechanisms that can either increase or decrease the number of hydrogen ions in a solution, thus making the solution more acid if the hydrogen ions increase in number or more alkaline if the hydrogen ions are reduced in number (Mattson Porth and Matfin, 2009). These include:

- inside cells proteins act as buffer systems such as the plasma proteins;
- in the circulation it is the bicarbonate buffer system that converts a strong acid that releases large numbers of H^+ to a weak acid that releases much fewer H^+. For example, hydrochloric acid (HCL: strong acid) can be substituted by carbonic acid (H_2CO_3: weak acid), thus reducing the overall H^+:

$$\underset{\text{(sodium bicarbonate)}}{HCl + NaHCO_3} \rightleftharpoons \underset{\text{(sodium chloride)}}{H_2CO_3 + NaCl}$$

  This equation is reversible and is accelerated by the presence of the enzyme carbonic anhydrase. The carbonic acid produced dissociates into H^+ and HCO_3^- (bicarbonate ions). The H^+ combines with haemoglobin and the bicarbonate diffuses into plasma where it continues to participate in buffering acids.

Respiratory control mechanisms act as another line of defence against alterations in acid-base balance. An increase in ventilation decreases levels of CO_2 in the circulation and a decrease in ventilation increases CO_2 in the blood. Chemoreceptors in the brain stem, carotid and aortic bodies (see Figure 6.1, page 134) sense changes in CO_2, hydrogen ions and O_2 and alter the respiratory rate accordingly. The respiratory control of pH is rapid and occurs within minutes of a change in pH balance but is only approximately 50–70% effective as a buffer system. It is the second line of defence against large changes in pH.

Renal control mechanisms are slower to react but can continue to function for days until the pH value has returned to the normal range. The mechanisms are:

- reabsorption of bicarbonate ions into the circulation;
- excretion of hydrogen ions from acids produced as a result of protein and fat metabolism.

Buffers are the body's first line of defence	The lungs are the body's second line of defence	The kidneys are the body's third line of defence
They act within seconds	They act within seconds to minutes	They act more slowly, measured in hours and days
They remove or release H^+ to correct acid-base balance	They eliminate or retain CO_2 to maintain the ratio of carbonic acid to bicarbonate at 1:20	The have a number of functions: – retention of carbonate ions – elimination of H^+

Table 3.1: How the body defends against abnormal alterations in acid-base balance

Hydrogen-potassium exchange: when there is excess H^+ in the blood, some is able to move into cells in exchange for potassium ions (K^+), and when there is excess K^+ in the blood, it moves into cells and exchanges with H^+. Thus potassium levels and hydrogen levels can change dramatically in some clinical situations such as a patient with diabetic ketoacidosis (see Chapter 8).

Blood urea nitrogen (BUN), creatinine and glomerular filtration rate through creatinine clearance: the measurement of creatinine and glomerular filtration rate are very important measures of renal function and will give an indication of how efficient the patient's kidney function is. If the patient has impaired kidney function the ability for the kidneys to act as the third line of defence in maintaining acid-base balance is impaired. A metabolic acidosis in the context of other indicators can suggest acute kidney injury or chronic renal disease (see Chapter 9).

What do these values tell us?

The pH value determines the presence of acidaemia and alkalaemia.

- Acidaemia: pH <7.35 (a value below 7.35).
- Alkalaemia: pH >7.45 (a value above 7.45).

The partial pressures of oxygen and carbon dioxide give a measure of respiratory function and the presence of respiratory acidosis/alkalosis.

- **Respiratory acidosis:**
  - pH <7.35 and $PaCO_2$ >6.0 kPa;
  - dyspnoea/increased or decreased respiratory function;
  - headache;
  - restlessness, confusion;
  - drowsiness/unconsciousness;
  - tachycardia and arrhythmias.
- **Respiratory alkalosis:**
  - pH >7.45 and $PaCO_2$ <4.9 kPa;
  - feeling light-headed;

- numbness and tingling in the mouth and peripheries;
- inability to concentrate, confusion;
- palpitations.

The levels of bicarbonate and base excess give a measure of metabolic function and represent either a failure to buffer hydrogen ion concentrations with bases leading to acidosis or a failure to buffer bicarbonate concentrations with acids leading to an alkalosis.

- **Metabolic acidosis:**
  - pH <7.35 and HCO_3 <22 mmol/L;
  - headache;
  - restlessness, confusion;
  - coma;
  - cardiac arrhythmias;
  - Kussmaul respirations (rapid shallow)/**respiratory depression**;
  - skin warm and flushed.
- **Metabolic alkalosis:**
  - pH >7.45 and HCO_3 >27 mmol/L;
  - muscle twitching and cramps;
  - feeling dizzy;
  - confusion;
  - lethargy;
  - seizures/coma;
  - nausea and vomiting.

In Table 3.2 you will find clinical examples of patients who have experienced an acid-base imbalance.

What does Jenny's arterial blood gas result tell us about her condition?

By using the step-by-step guide in Table 3.3, an analysis of Jenny's arterial blood gas results and general condition indicate the following.

- **Step 1: Assess oxygenation**

  PaO2 – 8.7 kPa: there is evidence of hypoxaemia with SpO_2 91% with supplemental oxygen of 60%.
- **Step 2: Assess pH level**

  pH – 7.42: there is no evidence of respiratory acidosis or alkalosis.
- **Step 3: Assess respiratory component**

  $PaCo_2$ – 3.6 kPa: this indicates that Jenny has been hyperventilating and expiring CO_2 in an attempt to cope with reduced volumes of air movement in her lungs due to bronchospasm. This is supported by her reduced peak expiratory flow rate (PEF) of 120 ml/min (her normal PEF: 300 ml/min).

Arterial blood gas analysis	Patient examples
Respiratory acidosis: pH <7.35 $PaCO_2$ >6.0 kPa	Gladys Cabrera (62 years) suffers from COPD and she is admitted to hospital with an acute exacerbation of her condition. She is unable to talk due to her breathlessness, rate of 40 bpm, SpO_2 is 72%, she is centrally cyanosed and she is unable to respond to commands. The results of an arterial blood sample are: pH 7.29, PaO_2 4.8 kPa, $PaCo_2$ 8.4 kPa, HCO_3 28.5 mmol/L. Following a rapid assessment of Gladys's condition she was admitted to intensive care for respiratory support and intensive treatment for type II respiratory failure (see Chapter 2).
Respiratory alkalosis: pH >7.45 $PaCO_2$ <4.9 kPa	Joan Butcher (50 years) suffers from anxiety attacks, and these have become worse since progressing to the menopause. On this occasion she has been involved in a minor road traffic collision and she has no obvious injuries. However, when the paramedics arrived at the scene they found her to be breathless and disorientated. She was complaining of pins and needles in her hands and arms, and she felt she couldn't get her breath. Joan was taken to accident and emergency where her arterial blood gas result following admission was: pH: 7.49; $PaCO_2$: 3.2 kPa; HCO_3: 24.2 mmol/L; BE (base excess): –1.0. Joan was hyperventilating and needed to be encouraged to reduce her respiratory rate and allow her carbon dioxide levels to rise back to normal levels.
Metabolic acidosis: pH <7.3 HCO_3 <22 mmol/L	Mary Bevan (58 years) was found by her neighbour lying at the front door in a drowsy and confused state. Mary has type 2 diabetes and has recently developed a severe infection on her leg. Mary's neighbour called the emergency services and Mary was admitted to accident and emergency. Her arterial blood gas following admission was: pH: 7.24; $PaCO_2$: 3.8 kPa; HCO_3: 15.1 mmol/L; BE: –13.7. Mary had Kussmaul respirations at a rate of 35 bpm and a blood glucose of 22 mmol/L. Mary had developed a metabolic acidosis secondary to infection that triggered an increase in blood glucose that necessitated management with insulin.

Metabolic alkalosis: pH >7.45 HCO_3 >26 mmol/L	Gary Smith (54 years) has been suffering from indigestion-type pain for several days. Rather than go to the GP he has been treating himself with large doses of antacids such as bicarbonate of soda. That afternoon he felt nauseated, weak and tired and still had the persistent indigestion. He visited the GP who decided to admit him to hospital for an assessment of his chest pain. His arterial blood gas following admission was: pH: 7.49; $PaCO_2$: 5.6 kPa; HCO_3: 29.7 mmol/L; BE: +9.0.
Respiratory and metabolic acidosis: pH <7.35 $PaCO_2$ >6.0 kPa HCO_3 <22 mmol/L	Peter Baker (41 years) was admitted to an acute ward with a history of abdominal pain, nausea and vomiting. Peter's condition deteriorated during the first 24 hours, and that evening he had a cardiac arrest. He was resuscitated and transferred to ICU for respiratory support and management of acute pancreatitis. His arterial blood gas following admission was: pH: 7.15; $PaCO_2$: 7.6 kPa; HCO_3: 16.7 mmol/L; BE: –9.8. Peter has developed a combined acidosis as a result of his cardiac arrest (failed respiration) and severe sepsis associated with pancreatitis and lactic acidosis (see Chapter 7).

Table 3.2: Clinical examples of patients with changes in acid-base balance

- **Step 4: Assess the metabolic component**

  HCO_3- 24 mmol/L: this indicates that Jenny has no evidence of metabolic acidosis or alkalosis.

- **Step 5: Combine your findings**

  Jenny is not experiencing any form of acidosis or alkalosis based on these blood gas results, however, the presence of a $PaCo_2$ of 3.6 kPa indicates that Jenny's hyperventilation and lower than normal $PaCo_2$ is correcting any potential for acidosis.

- **Step 6: Clinical interpretation and recommendation**

  Clinically Jenny is showing signs of type I respiratory failure (see Chapter 2), she is experiencing increased work of breathing and a reducing peak expiratory flow. The combination of salbutamol (bronchodilation) nebulisers and hydrocortisone will have a direct anti-inflammatory effect on her hypersensitive bronchi and should relieve her symptoms. Jenny, however, continues to be at risk of an escalation of her condition due to a secondary or late response to the initial trigger and requires close monitoring and support during this critical stage (BTS and SIGN, 2014; McCance and Huether, 2014).

Always risk assess	**Look: Listen: Feel: Measure**
ABG: Step 1 Assess oxygenation. • Normal: PaO_2 11.5–13.5 kPa	• Is there evidence of hypoxaemia? • Is there evidence of high levels of oxygenation? • Is the patient receiving supplemental oxygen?
ABG: Step 2 Assess pH level. • Normal: 7.35–7.45	• Is there evidence of acidosis? pH <7.35 • Is there evidence of alkalosis? pH >7.45
ABG: Step 3 Assess the respiratory component. • $PaCO_2$: 4.5–6.0 kPa	• Is the $PaCO_2$ <4.5 kPa? • Is the $PaCO_2$ >6.0 kPa?
ABG: Step 4 Assess the metabolic component. • HCO_3 : 22–27 mmol/L	• Is the HCO_3 <22 mmol/L? • Is the HCO_3 >27 mmol/L? • The base excess level (BE) is the quantity of acid or base required to restore the pH to 7.4. Base excess will mirror the bicarbonate level and simply reinforces evidence of a metabolic component (Jevon and Ewens, 2007).
ABG: Step 5 Combine your findings	• Combine your findings from steps 2/3/4 and identify if there is evidence of: o respiratory acidosis; o respiratory alkalosis; o metabolic acidosis; o metabolic alkalosis;

	• signs that the respiratory system has compensated for a metabolic acidosis by increasing the respiratory rate and reducing the CO_2 level; • signs that the renal system has compensated for chronic respiratory acidosis by increasing the level of HCO_3.
ABG: Step 6 Clinical interpretation and recommendation	• Interpret the ABGs in the context of all available patient data.

Table 3.3: A step-by-step approach to assessing arterial blood gas results (ABG)

Activity 3.1 *Decision making*

Read the scenario below and think about the significance of the arterial blood gas results.

Joseph Baglio (age 68 years) has smoked 40 cigarettes a day since his twenties and has experienced angina on exertion for the last five years although this has been managed by the use of beta blockers and GTN. He was admitted to the medical ward six hours ago following a diagnosis of pneumonia. Following his admission he seemed to be responding well to the oxygen therapy and IV antibiotics when he pressed the buzzer and fell forward clutching his chest. When the nurse arrived she found that Joseph was unresponsive with absent respirations and pulse. A cardiac arrest call was placed and he was resuscitated successfully. His arterial blood gas results 30 minutes after his resuscitation were:

pH: 7.05
PaO_2: 8.5 kPa on 60% high-flow oxygen (SpO_2 82%)
$PaCO_2$: 14.1 kPa
HCO_3: 20.5 mmol/L
BE: –3.0
Joseph was conscious, flushed and anxious. His vital signs were T: 38.0°C, R: 32/min, P: 95, BP: 110/70 mmHg.

- Using the step-by-step guide in Table 3.3, what can you interpret from the arterial blood gas result?

One hour later Joseph was conscious but confused. His skin was cold and clammy to touch and his vital signs were T: 38.0°C, R: 32/min, P: 94, BP: 110/70 mmHg. He was receiving 60% humidified high-flow oxygen and diagnosed with acute coronary

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syndrome with evidence of ST elevation myocardial infarction (STEMI). As well as his beta blockers, he has been prescribed statins for reducing cholesterol (he had previously refused to commence statins when prescribed before), clopidogrel to reduce the risk of another thrombotic event and morphine and nitrates for chest pain. Joseph was not considered a suitable candidate for percutaneous coronary angiography because of his pneumonia and was assessed as a candidate for thrombolysis instead but this was also decided against in light of his traumatic resuscitation. He now had two acute morbidities affecting his respiratory and cardiac system. A second arterial blood gas result was:

pH: 7.20
PaO_2: 9.4k Pa (SpO_2 89%)
$PaCO_2$: 6.70 kPa
HCO_3: 21.4 mmol/L

- Using the step-by-step guide in Table 3.3, what can you interpret from the arterial blood gas result?
- What are your priorities of care for this patient?

Answers are given at the end of the chapter.

With reference to Jenny's story we can see the importance of using a holistic approach to rapid assessment. There are a number of factors that now become significant when monitoring Jenny's condition. For example we know that:

- Jenny has now been awake and fighting for breath since 2 a.m. and it is now 4 a.m.;
- she is emotionally distressed after her husband appears to have left her;
- she is still recovering from an acute respiratory infection and is presenting with type I respiratory failure and about to be transferred to high dependency care.

Case study: Jenny's transfer to HDU

Following her assessment in the emergency room, Jenny was considered to be a level 2 patient requiring high dependency care for assessment and monitoring of her respiratory system. Following admission to HDU the results of her assessment were as follows.

A: Responding to commands and maintaining her airway.
B: SpO_2: 91%
60% high-flow humidified O_2
R: 36/min
ABGs:
pH 7.36

PaO_2 8.8 kPa
$PaCo_2$ 4.2 kPa

C: HR: 128/min
BP: 130/72 mmHg
D: Blood glucose 6.7 mmol/L
Agitated but disorientated
E: Temp: 37.5°C

According to BTS and SIGN (2014), the evidence of a severe hypoxia in the presence of a normalizing $PaCO_2$, and persistent disorientation following intensive treatment, indicates Jenny is having a life-threatening attack. She has been prescribed a once only dose of intravenous magnesium sulphate in an attempt to produce further bronchodilation (Blitz et al., 2005). When her current situation is assessed in the context of her previous admission to ICU, Jenny is referred to the ICU specialist who suggests that she meets the criteria for non-invasive ventilation (NIV) and that commencement of NIV could prevent her from needing intubation and invasive ventilation (Lim et al., 2012). The plan for Jenny is to commence her on NIV and monitor her ABCDE continuously for signs of improvement or deterioration.

What is NIV and why is it appropriate to use this respiratory support for Jenny?

Pulmonary ventilation, or breathing, is essential for life, and the purpose of NIV is to provide varying levels of positive pressure air flow through a tight-fitting mask in order to improve the patient's levels of PaO_2 and $PaCO_2$. Breathing involves the inhalation of gases in air into the lungs and exhalation of gases from the lungs into the atmosphere. All gases in air collectively exert a pressure known as atmospheric pressure. The gases in the lungs also exert a pressure known as alveolar pressure. In air, gases always flow from an area of high pressure to an area of low pressure. During inspiration the thoracic space expands as a result of contraction of the intercostal muscles and diaphragm. This increase in space reduces the overall alveolar pressure in the lungs and air flows into the airways in order to equalise the pressure. Expiration involves relaxation of the respiratory muscles and natural elastic recoil of the lung tissue so that air flows back into the atmosphere. Normal breathing therefore relies on negative pressure ventilation.

For 120 years the principal method of supporting ventilation for patients with respiratory failure was based on the principle of negative pressure ventilation. For example, the **iron lung** was used successfully for patients with respiratory failure caused by neuromuscular diseases such as polio. In the 1950s, during the polio epidemic in Europe, the demand for iron lungs outstripped supply and alternative methods for providing respiratory support were attempted (Lassen et al., 1954). This led to the development of mechanical **invasive ventilation** (MIV), which involved air being forced under pressure into patients' lungs via a tracheostomy tube or endotracheal tube at a rate

of between 10 and 20 per minute in order to mimic normal respiration. This dramatically reduced the mortality rate of patients suffering from respiratory failure and became the mainstay treatment (Borthwick et al., 2003).

In the last 25 years the use of non-invasive positive pressure ventilation (NIV) techniques that supply air through a tight-fitting face mask rather than a tube have escalated, and this method has now become the first-line therapy for adult patients with:

- sleep apnoea;
- acute exacerbations of COPD;
- pulmonary oedema;
- neuromuscular disease;
- pneumonia;
- weaning from MIV (BTS, 2000; BTS, 2008; NICE, 2010c).

In Jenny's case the use of NIV to manage an acute severe asthma attack does not have such a strong evidence base (Medoff, 2008). However, BTS and SIGN (2014) recommend that it should be considered as an option to prevent the risk of intubation in patients with acute severe asthma but should be based on skilled clinical assessment and knowledge of the patient's condition. Jenny's respiratory function is compromised but not so impaired that she is in imminent danger of complete respiratory collapse. She is able to protect her own airway, has only mild disorientation and there is no evidence of a pneumothorax on chest X-ray (Medoff, 2008). The types of NIV and their use are explained in Table 3.4.

Activity 3.2 *Reflection*

Reflect back on patients you have nursed and ask yourself the following questions.

- Have I looked after patients with acute respiratory failure either in hospital or the community?
- If so, how did I assess and document the patient care?
- Did the patient need support with oxygen therapy or NIV?
- Did the patient have support from the physiotherapist, dietitian and respiratory nurse?

Hint: This reflection is meant to encourage you to think critically about assessing and managing care and should help you to identify good practice and areas for improvement.

As this answer is based on your own reflection, there is no outline answer at the end of the chapter.

Contraindications for using NIV

The success of NIV techniques in the support of respiratory function relies on effective patient selection. For Jenny, CPAP (can also be referred to as pressure support when given through some ventilators) was chosen as the optimum treatment regime, but this does not mean that the use of NIV will always lead to a successful outcome for every patient. Patients need to be risk assessed

Type of NIV	Benefits	Risks	Patient examples
Continuous positive airways pressure: CPAP. This method provides a continuous flow of positive pressure even at the end of expiration so that some air always remains trapped in the alveoli. This enables oxygen exchange to continue during the whole respiratory cycle and prevents alveolar collapse (atelactasis).	• Improves oxygenation in patients with type I respiratory failure. • Reduces the risk of atelactasis.	• There is reduced clearance of CO_2 due to air being trapped in the alveoli. Not suitable for patients with type II respiratory failure where there are increased levels of CO_2. • The airway is not protected so patients must be able to maintain their own airway.	• Mrs Smith is admitted with severe breathlessness and is producing excessive amounts of pink frothy secretions from her airways. She is diagnosed with acute pulmonary oedema and is commenced on CPAP starting at 5 cm H_2O as part of her ongoing treatment to reduce pulmonary secretions by increasing alveolar pressure to above capillary hydrostatic pressure. • Chao Chan is diagnosed with pneumonia and type I respiratory failure. His PaO_2 is 6.2 kPa and his $PaCO_2$ is 3.6 kPa. He is commenced on CPAP at 5 and then 10 cm H_2O. • Bryn Jones has been diagnosed with **obstructive sleep apnoea**. He suffers from morbid obesity, snoring and daytime fatigue. He has now been fitted with a face mask and CPAP machine for home use. The equipment delivers CPAP at 10 cm H_2O, to be used at night while sleeping.

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Table 3.4 (Continued)

Type of NIV	Benefits	Risks	Patient examples
Bilevel NIV or bilevel positive airways pressure ventilation: BiPAP. This method provides two alternating levels of positive pressure during respiration. During inspiration, there is an inspired pressure level (IPAP) and during expiration, an expired pressure level (EPAP).	• Improves oxygenation and CO_2 clearance in patients with type II respiratory failure. • IPAP reduces the work of breathing and conserves the use of oxygen by the body. • A lower EPAP pressure reduces air trapping but still allows continuous gas exchange during respiration while preventing atelactasis.	• The airway is not protected so patients must be able to maintain their own airway.	• Henry Jones has pneumonia. His PaO_2 is 6.8 kPa and his $PaCO_2$ is 6.5 kPa. He is breathless and agitated. He is commenced on BiPAP with an inspiratory pressure of 10 cm H_2O and an expiratory pressure of 4 cm H_2O. • Gladys Cabrera (62 years) suffers from COPD and she is admitted to hospital with an acute exacerbation of her condition. She was commenced on BiPAP at an inspiration pressure (IPAP) of 12 cm H_2O and an expired pressure (EPAP) of 5 cm H_2O with 40% oxygen. She didn't like the face mask but was prepared to give it a try as long as the nurse reminded her.

Table 3.4: Types of non-invasive ventilation and their use

for any contraindications before commencing the therapy and then risk assessed for evidence of any change or deterioration in their condition. This is illustrated in Table 3.5. The contraindications of NIV rarely exist in isolation: often patients will present with one or more of these factors. Knowing the patient and their medical history is an essential part of the rapid decision-making process required when determining a patient's suitability for NIV and relies on good communication between all the carers involved (RCP et al., 2008). Contraindications include:

- life-threatening hypoxaemia;
- severe confusion/agitation/cognitive impairment;
- unconscious patient;
- airway obstruction due to vomiting or a foreign object;
- facial trauma/burns/surgery;
- **pneumothorax**;
- patient unable to protect their own airway;
- copious amounts of respiratory secretions/sputum;
- recent surgery in the upper gastro-intestinal tract;
- severe co-morbidity;
- haemodynamic instability;
- presence of bowel obstruction.

Table 3.5 offers a summary of the risk assessment and nursing interventions required to care for patients receiving NIV.

Risk assessment	Nursing interventions
Contraindications for use of NIV	• Rapid assessment of ABCDE using 'Look: Listen: Feel: Measure' is important to measure the risk of contraindications to treatment with NIV. In particular, the risk of pneumothorax should be ruled out by reviewing the patient's chest X-ray following their admission. • A patient may decide to refuse treatment.
Preparation of the patient and technology	• If the patient has consented and is able to proceed, ensure the equipment has been prepared and checked to ensure it is in working order. • Sit the patient upright and, with their cooperation, attach the face mask. The patient will need a few minutes to get used to the mask. Often NIV is commenced at a low level and increased according to the clinical state of the patient (RCP et al., 2008). • Document baseline clinical data. • Agree and document a treatment plan for escalating and identifying a ceiling of treatment.

(Continued)

Table 3.5 (Continued)

Risk assessment	Nursing interventions
Airway and respirations	• Monitor the patient's airway and respiratory rate, look for signs of respiratory distress and air entry as illustrated in Figure 2.1, page 36. • Monitor SpO_2 for evidence of improvement or deterioration. • Monitor the patient's arterial blood gas results after: ○ one hour: if there is no change in the patient's condition or a slight improvement, then monitor again in four hours; ○ one hour: if there is a deterioration in the patient's condition: ❍ assess patient and check the equipment; ❍ consider either increasing the oxygen or pressures; ❍ consider a change to mechanical ventilation.
Haemodynamic state	• The increase in pulmonary airway pressure from NIV can cause a rebound reduction in the patient's blood pressure, particularly with CPAP pressures above 10 cm H_2O. • Monitor the patient's blood pressure every five minutes during the first 30 minutes and then at 30 minutes to hourly as the patient's blood pressure stabilises. Continuous arterial monitoring of blood pressure provides an effective way to monitor BP as well as obtaining arterial samples for blood gas analysis.
Mental state and level of consciousness	• Monitor for signs of increased confusion or agitation. Any deterioration in level of consciousness is an indication that the NIV should be discontinued and the treatment plan utilised. • Patients on NIV should not normally be sedated as this can compromise their airway and compliance with treatment.
Fluid balance and gastro-intestinal function	• There is a risk of fluid retention triggered by the stress response (Chapter 6). Look for evidence of reduced urine output and interstitial oedema. • There is a risk of increased air swallowing and gastric distension associated with the air flow. This may be reduced by inserting a nasogastric tube.
Psychological distress	• Patients receiving NIV experience discomfort and distress due to the tight-fitting mask and side effects of the treatment. Communication is difficult with the face mask in place, although this may be resolved for some patients by using a nasal mask. Alternative techniques for delivering the air under pressure include a mouth piece and a helmet. • The role of the nurse in providing support and reassurance is essential. Frequent removal of the mask is counterproductive, and it is important to encourage the patient to keep the mask in situ for at least 30 minutes if any benefit is to be achieved.

	• If a patient is becoming very distressed, this will impact on their physiological state and is often an indication to discontinue the NIV and refer to the treatment plan (Jarvis, 2006). • Optimum management of patients with acute respiratory failure and NIV is achieved in ICU. However, patients can be nursed in acute wards and accident and emergency provided there is an appropriate skill mix and staff ratios of 1 or 2 patients to 1 nurse.

Table 3.5: Risk assessment and management of patients receiving NIV

Case study: Jenny's condition changes

Jenny consented to the use of NIV and commenced the support at a low level of positive pressure (5 cm) and this was gradually increased to 10 cm with support and encouragement from the nursing staff. There seemed to be an initial improvement with an increase in SpO_2 (94%). However, two hours after commencing the NIV Jenny's condition rapidly deteriorated as illustrated in the following assessment.

A: Difficult to rouse but still able to maintain her airway.

B: SpO_2 91% on O_2 60% high-flow humidified O_2
R 39/min, with shallow respirations
Blood gases showed:
pH 7.20
PaO_2 8.2 kPa
$PaCo_2$ 9.8 kPa
HCO_3 24 mmol/L

C: HR: 128/min
BP: 110/72 mmHg

D: Blood glucose 6.7 mmol/L
GCS had dropped to 9 (eyes opening to pain 2, inappropriate words 3 and flexion to pain 4).

E: Temp: 37.5°C

ABG analysis

- *Jenny has persistent hypoxaemia.*
- *She is acidotic.*
- *The high CO_2 indicates a respiratory acidosis.*
- *No sign of a metabolic acidosis.*

(Continued)

continued . . .

- *Jenny has hypoxia and a respiratory acidosis.*
- *Her worsening clinical condition of a high respiratory rate, reduction in her level of consciousness and increasing heart rate, combined with hypoxia and respiratory acidosis, indicates severe type II respiratory failure. Immediate intervention with intubation and mechanical ventilation is now required.*

What happens when NIV is not suitable: the case for mechanical invasive ventilation (MIV)

The benefits of supporting patients with respiratory failure with NIV include the following (RCP et al., 2008).

- There is reduced risk of ventilator-acquired pneumonia.
- The patient is fully awake and an active partner in their care.
- The patient may be nursed in an acute care setting.
- The use of NIV may prevent the requirement for invasive respiratory support.

There are, however, a number of reasons why patients may require an escalation of treatment to MIV or direct intervention with MIV without NIV (Brainard and Deutschman, 2010). These include:

- life-threatening hypoxic (PaO_2 below 8.0 kPa) respiratory failure accompanied by patient confusion and/or exhaustion;
- life-threatening hypercarbic ($PaCO_2$ above 6.0 kPa) respiratory failure accompanied by patient confusion and/or exhaustion;
- impaired consciousness and/or the patient's inability to protect their airway.

For patients in these situations, clinical assessment, combined with medical and nursing experience, is the most important tool for judging when invasive support with intubation and mechanical ventilation is required. Based on Jenny's assessment following her deterioration, she meets all of the three criteria above for intubation and mechanical ventilation. Jenny's sudden deterioration may have been related to the combined effects of physical exhaustion and the latent release of inflammatory mediators triggered by the initial inflammatory response several hours before. This can lead to further bronchospasm, oedema, mucus secretion and obstruction of air flow, an increase in variable and uneven airway obstruction and air trapping in the alveoli and hyperventilation (McCance and Huether, 2014). According to Medoff (2008) and Brenner et al. (2009) it is the combination of progressive airways obstruction and physical exhaustion caused by the increased work of breathing that leads to a reduction in the patient's respiratory tidal volume, retention of carbon dioxide, respiratory acidosis and deteriorating cardiovascular

Reason for MIV	Look: Listen: Feel: Measure	Patient examples
Hypoxaemic respiratory failure. • Pneumonia. • Lung consolidation. • Atelectasis. • Pulmonary oedema. • Acute respiratory distress syndrome (ARDS). • **Pulmonary embolism**. • **Carbon monoxide poisoning**.	Central cyanosis. Altered respiratory pattern. Agitation/irritability. Confusion. Exhaustion. Seizures. SpO_2 <85%. PaO_2 <8.0 kPa.	Chao Chan (Table 3.4) is diagnosed with pneumonia and type I respiratory failure. His PaO_2 is 6.2 kPa and his $PaCO_2$ is 3.6 kPa. He was commenced on CPAP at 5 cm H_2O then 10 cm H_2O. However, after the first hour he was confused and agitated, pulling off his mask and refusing to put it back on. His ABGs were PaO_2 5.7 kPa and $PaCO_2$ 5.0 kPa. It was agreed that treatment should be escalated to MIV.
Hypercarbic respiratory failure. • COPD. • Asthma. • Airway obstruction/anatomical. • Deformity. • Cervical injury above level C4 and/or damage to the brain stem. • Excessive sedation. • Guillain-Barré syndrome. • Cardiac arrest. • Heart failure. • Pulmonary embolism.	Increased work of breathing. Use of accessory muscles. Shallow breathing. Dyspnoea. Agitation/irritability. Confusion. Exhaustion. Seizures. Cardiovascular collapse and cardiac arrest. $PaCO_2$ >6.0 kPa.	Mariana Banica (27 years) has a severe scoliosis of her spine (the spine is curved from side to side in an S shape). Since childhood she has been prone to respiratory infections due to reduced and uneven lung capacity. Mariana was admitted to ICU after having collapsed at home following a flu-like illness for three days. On admission she was very confused, cyanosed and her breathing was shallow. Her ABGs were pH: 7.19; PaO_2: 12.7 kPa; $PaCO_2$: 10.7 kPa; HCO_3: 24.0 mmol/L; BE: 0.1. Mariana was intubated and commenced on BiPAP at a rate of 15/min, with an IPAP of 20 cm H_2O and EPAP of 5 cm H_2O.

(Continued)

Table 3.6 (Continued)

Reason for MIV	Look: Listen: Feel: Measure	Patient examples
Impaired consciousness and/or the patient's inability to protect his/her airway. • Glasgow Coma Scale (GCS) score of <8 indicates the potential for further deterioration in consciousness, reduced ventilation and poor airway protection, for example: ○ severe brain injury; ○ prolonged effects of general anaesthetic; ○ traumatic injury of the face and neck.	Inability to maintain airway. Unconscious. GCS <8.	Pete Williams (19 years) was assaulted on his way home from the pub. A witness said that Pete had been kicked repeatedly on the head while he lay on the floor. In ICU he was agitated and unable to communicate except with grunts. He was opening his eyes and flexing his arms to pain, GCS 7. The computerised tomography scan showed evidence of progressive brain swelling. The management plan for Pete in the first 24 hours was to intubate him with an oral endotracheal tube and provide continuous pressure ventilation (IPAP 30 cm H_2O) with a rate of 15/min in order to protect his airway and maintain PaO_2 >8.0 kPa and $PaCO_2$ 4.5–6.0 kPa. Pete developed ventilator-acquired pneumonia on day four and stayed on MIV for seven days.

Table 3.6: Indications for mechanical invasive ventilation in the critically ill patient

and neurological state, as illustrated in Jenny's case study. Other clinical examples of situations when MIV is required are included in Table 3.6.

Mechanical invasive ventilation in adults can only take place when a patient is intubated with a cuffed endotracheal or tracheostomy tube. The cuff provides a seal around the tube and prevents leaks. The purpose of MIV is to push air under pressure into the patient's lungs to ensure there is effective movement of oxygen and carbon dioxide in and out of the lungs (pulmonary ventilation). There are increasing numbers of types and modes of MIV, but for the purposes of this chapter we will limit discussion to two core modes: pressure-controlled ventilation and volume-controlled ventilation (Carbery, 2008; Grossbach et al., 2011). In Table 3.7 you will find an explanation of these modes together with the advantages and disadvantages of both.

In both pressure-controlled and volume-controlled ventilation the patient's respiratory rate can be managed in one of three ways.

- The patient breathes spontaneously and controls their own rate.
- The patient's respiratory rate is set and controlled by the machine.
- The patient's respiratory rate is supported by a minimum respiratory rate set by the machine and supplemented by the patient's own respiratory rate.

The option of as much or as little respiratory support through MIV allows the patients to be involved in the process of respiratory support and aids their readiness to wean from MIV as they improve.

Case study: Jenny's intubation and ventilation with MIV

Jenny now required immediate intubation and mechanical ventilation and she was induced into anaesthesia with ketamine and alfentanil (short-acting anaesthetic agents) and paralysed with suxamethonium (a fast-acting muscle relaxant) to facilitate safe tracheal intubation with an oral endotracheal tube. Because of the combination of risks related to hyperinflation of the lungs, air trapping and increased airways resistance caused by bronchospasm, inflammation and mucus production, it was decided that the best clinical intervention for Jenny was controlled ventilation with SIMV (Table 3.7), with an inspired tidal volume set at 400 ml and a rate of 16 breaths per minute and a plateau airways pressure of 30 cm. The respiratory rate was set to give Jenny a short inspiration time and a prolonged expiratory time to reduce the risks of further air trapping and barotrauma (Brenner et al., 2009). In order to achieve this type of controlled ventilation it was necessary to fully sedate and paralyse Jenny with neuromuscular blockade and this was achieved by the use of propofol (a short-acting anaesthetic) and cisatracrium (a short-acting neuromuscular blocking agent). Jenny's airway resistance was reduced by suctioning of the airways to remove secretions. A chest X-ray was performed to check the position of the endotracheal tube.

MIV mode	Risks	Benefits
Pressure-controlled/pressure-support ventilation: air is pushed into the lungs until a preset alveolar pressure is reached. For example: • bilevel positive airways pressure (BiPAP) (see NIV). • continuous positive airways pressure (CPAP) (see NIV). • pressure-support ventilation (PS). • positive end expiratory pressure (PEEP).	Ineffective ventilation. Hypo ventilation and variable tidal volumes triggered by reduced lung compliance in the presence of acute lung injury, sputum and/or bronchospasm. Compliance measures the 'ease of stretch' ability in the lungs. The more compliant the lungs are, the less pressure is required to open the airways during MIV.	Reduces the risk of ventilator-associated lung injury.
Volume-controlled ventilation: a preset volume of air is delivered to the lungs with each breath. For example: • synchronised intermittent mandatory ventilation (SIMV).	Ventilator-associated lung injury: • barotrauma: over-distension of some alveoli; • volutrauma: over-distension of the alveoli caused by large tidal volumes; • biotrauma: the release of inflammatory mediators that may increase patient mortality.	The machine delivers a set tidal volume with each breath, thus improving overall ventilation.
Modes that deliver a combination of both. For example: • pressure-regulated volume-controlled ventilation.		Reduces the risk of ventilator-associated lung injury. Ensures effective tidal volumes and pulmonary ventilation.

Table 3.7: A comparison of pressure-controlled and volume-controlled ventilation modes

Research summary: Sedation

The aim of using drugs to sedate patients during MIV is to promote comfort, relieve distress and anxiety, and facilitate effective respiratory function. The majority of drugs used for this purpose, however, can cause side effects, including: depression of the cardiovascular system leading to reduced BP; respiratory depression and delayed weaning from respiratory support; reduced motility of the gastro-intestinal tract with delayed absorption of nutrients and poor quality sleep (Whitehouse et al., 2014). The use of sedation assessment scales and sedation protocols have been recommended as a method for getting the balance right between the advantages and disadvantages of using sedation. The Ramsay scale, Riker Sedation-Agitation scale and Richmond Agitation and Sedation scale are examples of tools adapted for patients on MIV (Ramsay et al., 1974; Riker et al., 2001; Ely et al., 2003). There is limited evidence, however, that such scales and protocols can improve patient outcomes (O'Connor et al., 2010; Williams et al., 2008; Whitehouse et al., 2014). There is evidence, however, that daily sedation interruption combined with patient assessment can improve patient outcome (Chen et al., 2014). There is also evidence that healthcare staff do not always follow sedation recommendations due to lack of awareness, lack of conceptual agreement with the guidance, poor strength of evidence in their use and lack of clarity over who is responsible for prescribing the guidance (Sneyers et al., 2014; Miller et al., 2012). In summary, the use of sedation protocols and daily sedation interruption while seen to be clinically effective continue to be areas that require further research and should always be used in the context of the patient's clinical condition.

Why are tidal volume, respiratory rate and airway pressure important in promoting optimum ventilation?

The tidal volume (TV) is the volume of air in each breath and can be measured as inspired (ITV) and expired (ETV) tidal volume. The respiratory rate (R) describes the total number of respirations in a minute. If a patient is on MIV this may include set ventilator breaths and the patient's own breaths. Minute volume is the total volume of air either inspired (IMV) or expired (EMV) in one minute and is equal to tidal volume times respiratory rate (Hall, 2011). Airway pressure is the same as alveolar pressure and is the pressure required or allowed to push air into the patient's lungs.

When assessing and monitoring a patient receiving MIV, tidal volume, rate, minute volume and airway pressure are some of the important indicators for measuring effective ventilation. For example, increasing ITV, R or IMV can improve the elimination of CO_2. If, however, by doing this the inspired airway pressure goes above 30–35 cm H_2O, then the patient becomes at risk of acute lung injury. Patients such as Jenny often have high airway resistance and it becomes harder to push air into the lungs. In this situation it is important to reduce the risks of barotrauma and pneumothorax caused by high inflation pressures by balancing the controlled respiratory rate

and ITV to ensure inspired airway pressure does not exceed 30–35 cm H_2O. Promoting effective patient ventilation therefore requires assessment, monitoring, communication and collaboration with the patient, nurse, intensivist (anaesthetist) and physiotherapist to promote optimum lung function, and with the dietitian to promote optimum nutrition to support the patient's metabolic requirements and promote recovery (Woodrow, 2012). A summary of the risk assessment and management of patients such as Jenny is illustrated in Table 3.8.

Risk assessment	Nursing interventions
Airway	
• Risk of the endotracheal tube/tracheostomy (tube) occluding due to poor humidification, the patient biting down on the tube and/or secretions. • Risk of airway irritation. • Risk of the tube becoming dislodged. • Risk of unplanned extubation.	• Look for evidence of distress and agitation such as coughing and biting on the tube, assess the patient's sedation score and reassure. If the patient continues to be distressed, there is a higher risk of unplanned extubation and/or trauma to the patient's airways. If necessary, increase the sedation according to the prescribed guideline until the patient is comfortable. • Humidification of the airways can be achieved by: o heat/moisture exchange (HME) filters that are attached to the ventilator circuit close to the endotracheal tube; o hot water humidifiers (37°C); o cold water humidifiers.
Breathing	
• Risk of airways becoming partially occluded leading to a rise in airway pressure and ineffective ventilation. • Risk of air leak due to poor connections. • Risk of inappropriately set alarm parameters. • Risk of ventilator-associated lung injury and ventilator-associated pneumonia (VAP).	• Narrowing or occlusion of the patient's airway can be identified by an increase in the inspired airway pressure and evidence of patient agitation, rattling/bubbling on chest auscultation. • Endotracheal suction is used to remove secretions in the trachea but should only be performed when there is evidence of the above. Suction can be painful, distressing and increase the risk of infection and trauma to the airways. • A loose connection can be identified by a reduction in inspired airway pressure, tidal volume and reduction in SpO_2. • Assess respirations, inspired and expired tidal volumes and airway pressure, SpO_2 and ABG analysis if the patient's condition changes. • Set alarm limits to between 5 and 10 marks above and below the prescribed range and assess the patient hourly. • Adhere to the ventilator bundle (see Concept summary: Care bundles).

Circulation	
• Risk of impaired circulation and cardiac function: MIV increases venous return pressure because the right side of the heart has to pump against a higher alveolar pressure, thus raising the patient's CVP. Left ventricular cardiac output is reduced due to more blood staying in the venous circulation. Thus the patient is at risk of hypotension and oedema. • Risk of liver dysfunction leading to clotting disorders, immunosuppression and reduced albumin production.	• Assess the patient's vital signs for evidence of impaired circulation using continuous monitoring: heart rate and rhythm; BP; CVP; chest X-ray; signs of venous thrombosis; urine output, which should be ≥0.5 ml/kg/hr (> about 30 ml/hr). • Adhere to the ventilator bundle to reduce the risk of VAP. • Assess the patient for signs of peripheral oedema, bruising. • Assess blood results including: serum electrolytes; urea and creatinine; liver function tests; clotting. • Assess and screen for sepsis daily (Chapter 7).
Disability	
• Inability to communicate verbally due to the endotracheal tube and sedation. • Risk of pain. • Risk of poor skin integrity, dry eyes and mouth. • Risk of anxiety, delirium and/or boredom.	• When appropriate, encourage the patient to use non-verbal means of communication, picture cards and alphabet cards. Use eye contact and explain all procedures before they are attempted. • Assess the patient's pain using non-verbal cues and pain scores and manage appropriately. • Assess the integrity of the patient's eyes and mouth hourly and manage appropriately according to each patient's needs. • Adopt the Institute for Healthcare Improvement (IHI, 2009) care bundle for pressure ulcer prevention: risk assess on admission; reassess daily: inspect skin, manage moisture on the skin, optimise nutrition and hydration, minimise pressure through positioning. • Help the patient to be orientated to night and day, and assess for signs of delirium (Chapter 8). • Encourage family-centred care and patient-focused care. • Encourage the patient to be involved in decisions and, where possible, life outside the unit.

(Continued)

Table 3.8 (Continued)

Risk assessment	Nursing interventions
Exposure and safe environment • Risk of infection associated with the use of invasive procedures. • Risk of noise and the environment disturbing sleep and rest.	• Risk assess and manage the patient with due regard to the ventilator bundle and risk assessment for sepsis. • Assess noise levels and reduce noise pollution where possible. Reorientate the patient to their environment and offer reassurance when appropriate.

Table 3.8: Risk assessment and plan of care for a ventilated patient

Concept summary: Care bundles

Evidence-based practice is concerned with ensuring that the best available evidence is applied to practice. One method for achieving this is through the use of care bundles. Care bundles are a group of evidence-based interventions that, when combined, provide the most clinically effective method for reducing risk and improving patient outcome (Fulbrook and Mooney, 2003). The ventilator care bundle is an example of how combining selective interventions appears to have reduced the incidence of ventilator-acquired pneumonia (Lawrence and Fullbrook, 2011; Eom et al., 2014). The bundle recommended by the IHI (2014) combines the following five elements.

- Elevation of the head of the bed to 30–45%.
- Periodic interruption of the patient's sedation and daily assessment of the patient's readiness for extubation.
- Peptic ulcer disease prophylaxis.
- Venous thromboembolism prophylaxis.
- Daily oral care with chlorhexidine.

Case study: Jenny's ventilation with MIV

Jenny continued to be ventilated, sedated and paralysed for a further 12 hours until her clinical condition improved and her neuromuscular blocking agent was discontinued. Jenny's sedation was ceased and she was assessed:

A: Airway maintained through an oral endotracheal tube.
Responding to commands.

B: *SpO_2: 95%*
O_2 60%
Ventilation mode: SIMV
ITV: 400 ml
Controlled R: 16/min
Blood gases showed:
pH 7.32
PaO_2 11 kPa
$PaCO_2$ 6.0 kPa

C: *HR: 120/min*
BP: 110/70 mmHg

D: *Blood glucose 6.7 mmol/L*
Sedated with reducing levels of propofol according to protocol

E: *Temp: 37.5°C*

ABG analysis

- *Jenny's oxygen levels have improved and are now within the accepted safe range.*
- *She is slightly acidotic but considerably improved from her results prior to MIV.*
- *The high CO_2 indicates the upper limit of normal.*
- *No sign of a metabolic acidosis.*
- *Jenny no longer has hypoxia or hypercapnia.*
- *Her clinical condition has improved and she no longer has evidence of respiratory failure. The recommendation is to continue to reduce her sedation and encourage her to trigger her own breaths while reducing the preset ventilator rate. Once she is breathing without the help of the MIV the plan is to extubate her and monitor her condition.*

Chapter summary

In this chapter you have been introduced to patients who need advanced respiratory support. The technology and assessment strategies for patients in these situations are often complex and the patient's condition can change suddenly. We have seen how Jenny's condition initially deteriorated but continuous assessment of her condition alerted staff to changes in her condition and she received intensive care. To complete her story, Jenny's condition improved sufficiently for her to be transferred to a general ward and she was discharged from hospital five days later. She decided to separate from her husband and has managed to give up smoking and make a new life for herself. She can still remember her time in the intensive care unit and is determined to improve how she manages her asthma in order to reduce the risk of readmission.

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The important messages to gain from this chapter are as follows.

- Always begin by assessing the patient's airway, breathing and circulation, disability and environment, and you will always be able to prioritise care and communicate your concerns.
- Interpretation of the patient's condition through blood gas analysis means much more if the results are assessed in the context of the patient's story.

Activities: brief outline answers

Activity 3.1: Decision making (pages 63–4)

Using the step-by-step guide in Table 3.3, what can you interpret from the arterial blood gas result?

These are the first set of ABG results.

- Joseph was showing signs of hypoxaemia on 60% oxygen.
- pH 7.07: shows evidence of acidosis.
- $PaCO_2$: 14.1 kPa shows evidence of respiratory acidosis.
- HCO_3: 20.5 mmol/L shows evidence of metabolic acidosis also.
- Joseph shows signs of both a respiratory and metabolic acidosis.
- This result in the context of his clinical situation are consistent with a period of inadequate oxygenation and tissue perfusion related to his cardiac arrest. Re-establishment and maintenance of Joseph's respiration and circulation will provide an opportunity for acid-base balance to be restored.

Using the step-by-step guide in Table 3.3, what can you interpret from Joseph's second arterial blood gas result?

- Joseph's oxygen levels had improved however his PaO_2 and SpO_2 are still below the accepted level.
- His pH of 7.20 is still showing signs of acidaemia.
- $PaCO_2$: 6.70 kPa still shows evidence of respiratory acidosis but has improved from his previous results.
- HCO_3: 22.5 mmol/L shows evidence of a resolving metabolic acidosis compared to the previous result.
- Joseph still shows signs of both a respiratory and metabolic acidosis, however, this is not as severe as his post cardiac arrest results.
- Joseph's ABGs still indicate evidence of type II respiratory failure which has now been complicated by a diagnosis of a STEMI.

What are your priorities of care for this patient?

- Joseph needs to receive support for his respiratory failure and should be assessed to determine the most suitable treatment plan. This may be a combination of nebulised short-acting **beta agonist**, short-acting **muscarinic antagonist** and intravenous antibiotics. He should be encouraged to sit up in the most comfortable breathing position and be assessed for NIV. Following an assessment from the critical care outreach team, Joseph was transferred to ICU for NIV and was commenced on bilevel positive airways pressure.
- Your role is to risk assess the patient and reassure him, and if he is commenced on NIV, to support him to promote his comfort.
- Joseph will require continuous assessment of his respiratory and cardiac function with a view to reducing the NIV support over the next few hours if his condition continues to improve.
- He will need continued support and reassurance to maximise the effect of the respiratory support and his cardiovascular status should be monitored to assess for signs of deterioration following his cardiac event.

Further reading

Moore, T and Woodrow, P (2009) *High Dependency Nursing Care: Observation, Intervention and Support for Level 2 Patients.* Second edition. London: Routledge.

This book offers practical help with learning how to use the technology when involved in the care of level 2 patients.

San Diego Patient Safety Council (2009) *Tool Kit: ICU Sedation Guidelines of Care.* San Diego: San Diego Patient Safety Council. http://www.carefusion.com/pdf/The_Center/2008-PCA-toolkit-disclaimer-updated-may-30-2014.pdf

This document gives you a helpful introduction to some of the drugs used to promote safety and pain relief for patients with mechanical ventilation. It also gives you examples of some of the assessment tools available for monitoring pain and sedation.

Useful websites

http://www.ics.ac.uk/ics-homepage/guidelines-and-standards

The Intensive Care Society site provides access to relevant innovations and standards that relate to the care of patients who are critically ill. The website is multidisciplinary and offers information to patients and relatives in user-friendly guides. A revised edition of sedation guidance is now available on this website.

www.ihi.org

The Institute for Healthcare Improvement website offers evidence-based and practical ways in which to provide safe and effective care for patients with acute and critical care needs.