

CHAPTER 20

Respiratory emergencies

SARAH LOUISE POWER

LEARNING OBJECTIVES

- Describe the anatomy and physiology of the lungs and discuss complications due to related diseases.
- Demonstrate an accurate and detailed assessment of a patient presenting to the emergency department (ED) with a respiratory emergency.
- Recognise and effectively manage the complications surrounding respiratory emergencies and potential hypoxaemic and hypercapnic compromise.
- Demonstrate and discuss troubleshooting complications associated with an underwater sealed drain.
- Describe the principles of non-invasive ventilation.

INTRODUCTION

Australian Lung Foundation research shows that chronic obstructive pulmonary disease (COPD) is the fourth biggest killer in Australia and approximately one million Australians have COPD. It estimates the cost to taxpayers is about \$800 to \$900 million each year.¹

This chapter discusses the essential components of assessing respiratory emergencies and managing the respiratory patient in the ED.

ANATOMY AND PHYSIOLOGY

Structures which constitute the upper respiratory system include the nose and nasal cavity, epiglottis, pharynx and associated structures (Fig 20.1). The lower respiratory system comprises the larynx, trachea, bronchi and the lungs. Within the thorax are the major organs of respiration and these include the thoracic cavity, lungs, pleura and muscles of ventilation.²

The epiglottis is situated in the upper airways and protects the airways by closing the opening of the trachea during swallowing.

The pharynx is a funnel shaped tube which functions as a passage for food and air. Its space acts as a resonating chamber for sound. The purpose of the upper respiratory system is to heat, filter and moisten air as it passes through its structures before entering the lungs.^{2,3}

The larynx or voice box is composed of nine pieces of cartilage. The thyroid cartilage or 'Adams apple' and the cricoid cartilage are the most commonly known. The cricoid cartilage is a ring of cartilage which forms the inferior wall of the larynx and is attached to the first ring of cartilage of the trachea. The cricoid cartilage is the clinical landmark for applying cricoid pressure during intubation and creating an emergency tracheostomy.

The trachea or windpipe is a passageway for air and is about 12 cm in length. It is situated anterior to the oesophagus

and stretches from the trachea to the 5th thoracic vertebrae; at this point it divides in the right and left main bronchi. The trachea is lined with ciliated epithelium which traps particles of dust and other debris, preventing contamination of the lower airways (Fig 20.2).^{2,3}

The right and left bronchi are anatomically different. The left bronchi are narrower and angled at 45° to 55° towards the left lung because of the position above the heart. The right bronchi are wider and angled at 20° to 30° degrees. Because of the angle of the right bronchi and the force of gravity they are the most susceptible sites for aspiration of foreign objects into the lower lobe of the right lung. Each branching from the main bronchus produces a new generation of tubes. With each branch the diameter decreases but the number of bronchi increases.²⁻⁴

The final division results in the bronchioles which are 1 to 2 mm in diameter. Bronchioles consist of smooth muscle and contain no cartilage. The last branch is formed by the terminal bronchioles, of which there are more than 32,000; this is where the gas exchange of the lungs takes place.

Terminal bronchioles further divide into branches called respiratory bronchioles, which in turn subdivide into several alveolar ducts. The primary site of gas exchange is the alveolus, which is the endpoint of the respiratory tract. Within both lungs there are approximately 300 million alveoli.²⁻⁴

The lungs are a pair of cone shaped organs, which lie within the thoracic cavity. They are separated by the heart

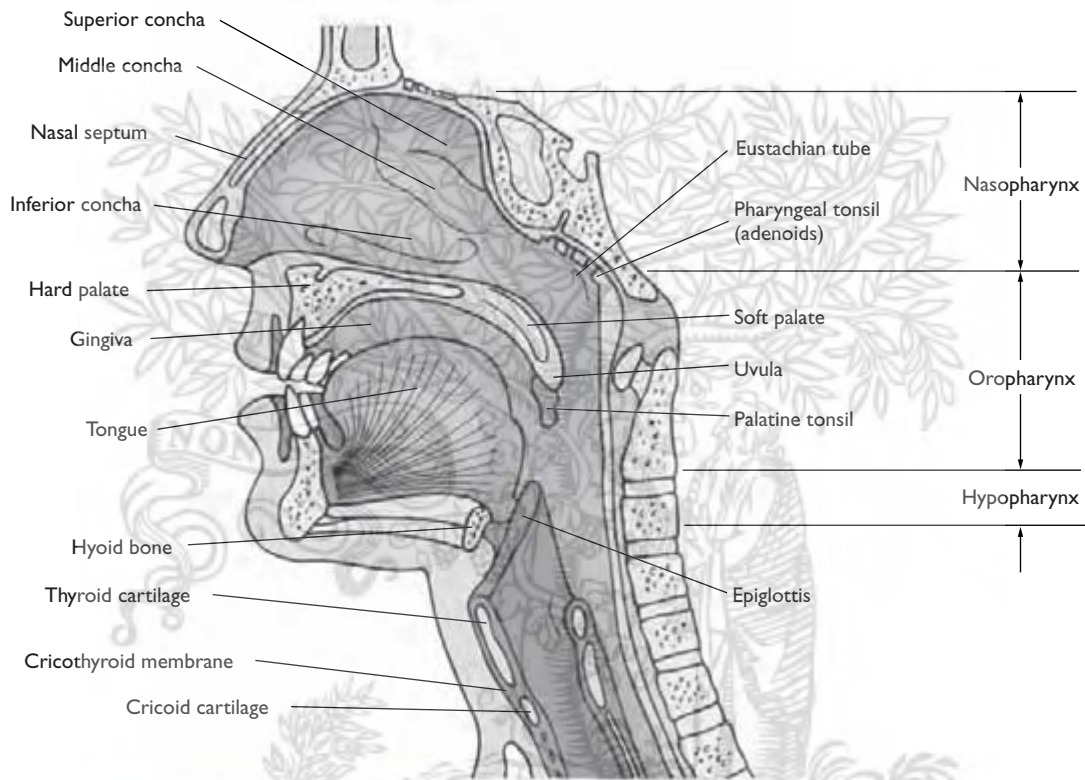


FIGURE 20.1 Structures of the upper airways. Note the placement of the epiglottis. From Ellis PD, Billings DM. *Cardiopulmonary resuscitation: procedures for basic and advanced life support*. St Louis: Mosby; 1980.

and other structures in the mediastinum, which also acts to separate the thoracic cavity into two distinct anatomical chambers. The lungs are enclosed and protected by two layers of serous membrane called the pleural membrane.^{2,4} The parietal pleura are the superficial layer which lines the inside of the thoracic cavity. The visceral pleura are the deeper layer and cover each lung. There is a small space between each of the pleural layers which is called the pleural cavity. The pleural cavity contains a lubricating fluid that is secreted by the membranes; this fluid reduces friction between the membranes during inspiration and expiration and allows the membranes to glide easily over one another.²⁻⁴

The lungs are divided into lobes and segments. The right lung consists of three lobes, the upper, middle and lower lobe. The left lung is divided into only two lobes, the upper and lower lobe. The lobes of the lungs are separated by pleural membrane covered segments or fissures.²⁻⁴

The main muscle required for inhalation is the diaphragm which contracts and flattens, pushes down and causes the abdomen to displace outwards. To some degree the contraction of the diaphragm lifts and expands the rib cage. The other muscles which govern inhalation include, most importantly, the external intercostal muscles, which lift the ribs and expand the chest outwards (Fig 20.3).^{2,4}

Exhalation is passive and occurs when the diaphragm relaxes; therefore there are no true muscles of exhalation. The elastic recoil of the lungs is intrinsic and also assists in

exhalation. Accessory muscles include the sternocleidomastoid and other chest and back muscles; these are not active during normal breathing (Fig 20.4).²⁻⁵

The exchange of respiratory gases—oxygen (O_2) and carbon dioxide (CO_2)—between the circulatory blood and the lungs takes place by diffusion across the alveolar–capillary membrane. At this site a network of vessels completely surrounds the alveoli which are just large enough for red blood cells to pass through in single file, enabling the membrane of the alveolar cell to touch the capillary walls and making it highly efficient for gas exchange (Fig 20.5).^{2,4}

Oxygen is transported to the tissues via the blood. Oxygenation of the cells is dependant on the transport of adequate oxygen to the cells, the affinity of haemoglobin for oxygen and how easily haemoglobin will release oxygen to the cells.

The oxygen–haemoglobin dissociation curve describes haemoglobin's affinity for oxygen (Fig 20.6).^{4,5} When the curve shifts towards the left, then haemoglobin can pick up oxygen in the lungs more readily but does not release it to the tissues easily. When the curve shifts to the right haemoglobin picks up oxygen less rapidly but delivers it to the cells more easily. Factors which can affect the oxygen dissociation curve are temperature, acid–base balance and CO_2 pressure levels.⁴

The basic rhythm of respiration is controlled by the medulla oblongata and the pons. Impulses reach the diaphragm

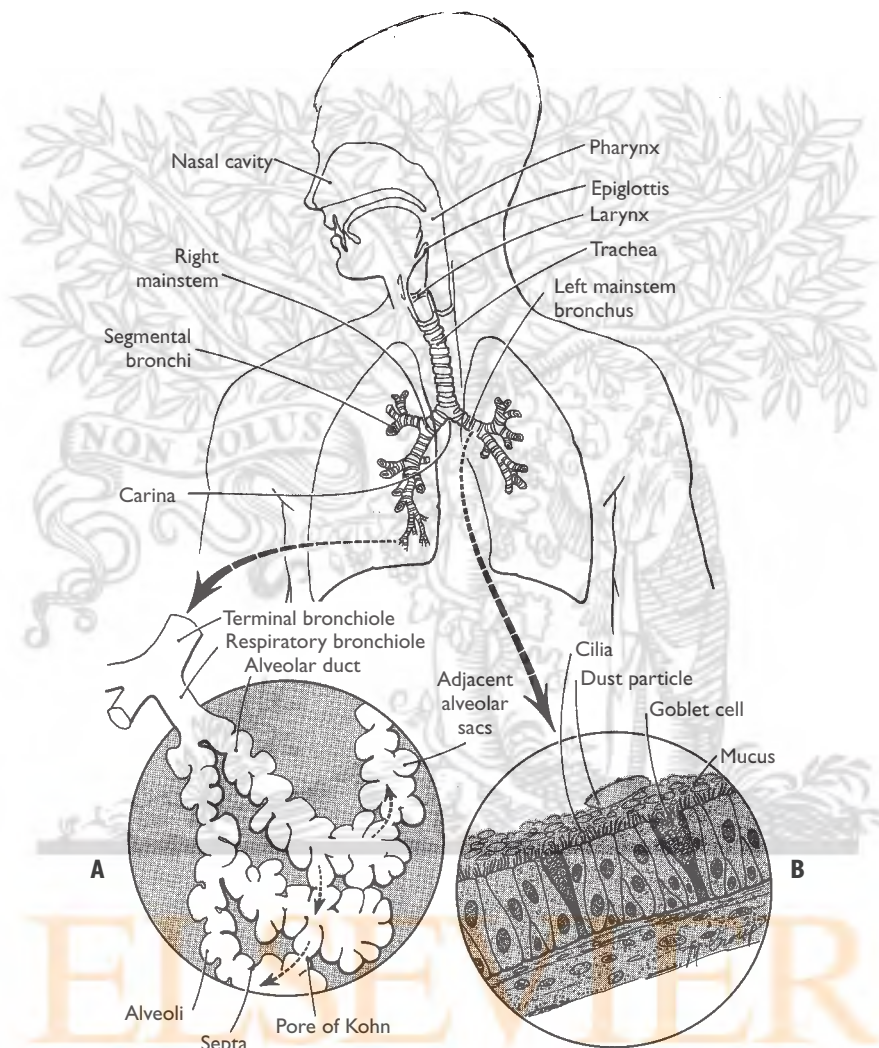


FIGURE 20.2 Structures of the respiratory tract. **A**, Pulmonary functional unit. **B**, Ciliated mucous membrane. From Price SA, Wilson LM. Pathophysiology: clinical concepts of disease processes. 5th edn. St Louis: Mosby; 1997.

via the phrenic nerve and when these impulses reach the intercostal muscles, the muscles contract and inspiration occurs. During the resting state inspiration occurs over 2 seconds and expiration occurs over 3 seconds as a result of the passive elastic recoil of the muscles.^{2,4}

Chemical regulation of respiration serves to maintain the correct levels of O₂ and CO₂. Chemoreceptors are highly sensitive to subtle changes in hydrogen ions (H⁺), CO₂ concentrations or both.^{3,5}

Ventilation is the movement of air in and out of the lungs by inspiration and expiration. Respiration is the process of gas exchange within the lungs at a cellular level. Ventilation and perfusion of the lungs is represented by a ratio. During normal respiration the lungs are in balance and every litre of alveolar ventilation is approximately matched by one litre of pulmonary capillary blood flow. Changes in this balance are expressed as a ratio called the ventilation/perfusion

ratio (\dot{V}/\dot{Q}). A high \dot{V}/\dot{Q} is indicative of greater than normal ventilation and lower than normal perfusion or both. A low \dot{V}/\dot{Q} is indicative of lower than normal ventilation and greater than normal perfusion or both (Fig 20.7).²⁻⁶

PATIENT ASSESSMENT

Assessment of the patient with a respiratory emergency should begin with evaluation of airway, breathing and circulation status (ABC).^{5,6} After this is done, the four techniques for physical assessment should be used. *Inspection* is the process of observation that requires an intensive look at the patient. *Palpation* requires touching the patient to judge the shape, size, temperature and texture of the skin, the body surface and underlying anatomical structures. *Percussion* is the creation of sound waves on the surface of the body to assess and determine the normal from abnormal sounds, resonance and

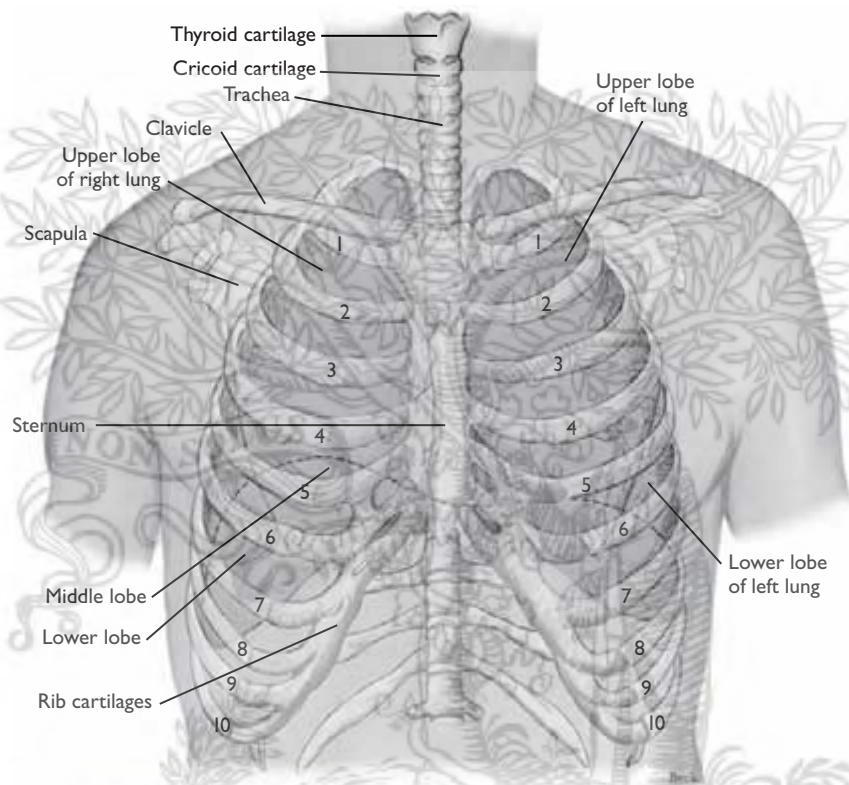


FIGURE 20.3 Ventilatory structures of the chest and lungs, showing ribs (numbered) and lobes of the lungs. Each intercostal space takes the number of the rib above it. The dotted line indicates the location of the diaphragm at inhalation and exhalation. Note the apex of each lung rising above the clavicle. From Thibodeau GA. *Anthony's textbook of anatomy and physiology*. 13th edn. St Louis: Mosby; 1990.

density of the underlying areas. *Auscultation* is the process of listening with a stethoscope to determine body function characteristic sounds.^{3,5,6}

Inspection should focus on the mucous membranes of the tongue and sublingual area for signs of central cyanosis. Discolouration of the fingers and toes can indicate peripheral cyanosis. Assessment of the chest wall should identify the shape and size of the patient's chest and assessment of bilateral chest wall movement. Evaluation of respiratory effort should include assessment of rate, rhythm, symmetry, patient position and the ability to lay supine, effort to breathe and use of accessory muscles, the ability to speak in sentences and the level of consciousness.^{3,5,6}

Palpation evaluates the position of the trachea and assesses the degree and symmetry of respiratory movement. Percussion evaluates and assesses the underlying lung structure. Auscultation assesses and identifies normal and abnormal breath sounds.^{3,5,6}

Other important observations include vital signs, oxygen saturation level (SpO_2) peak flow and spirometry. Monitoring of cardiac rhythm can alert staff to arrhythmias related to myocardial ischaemia precipitated by the respiratory emergency (see Ch 10).^{3,4} Obtaining a thorough history should include the establishment and onset of the symptoms, a medical history including any recent hospital admissions,

use of tobacco, occupation and any exposure to an infectious disease (Table 20.1).³⁻⁶

Peak flow

Measurement of peak flow in the emergency department is done using a simple assessment device that measures airflow, or peak expiratory flow (PEF). Patients blow into the device quickly and forcefully, and the resulting peak flow reading indicates how open the airways are, or how difficult it is for the patient to breathe.^{6,7} Peak flow meters have limited accuracy and provide only a single-effort-dependent assessment of ventilatory function. They are also dependent on patient technique. PEF is reduced in diseases causing airway obstruction and for asthmatics. The meter is a very useful tool in the home for patients to assess changes in condition and prevent unnecessary attendance at the ED.^{3,4,6,7}

Spirometry

Spirometry is a physiological test to measure lung function. It is the broadest non-invasive test of ventilatory function. Spirometry is used to detect and assess diseases which limit ventilatory capacity and affect the mechanics of the chest wall and lungs and it assesses the function of the airways.

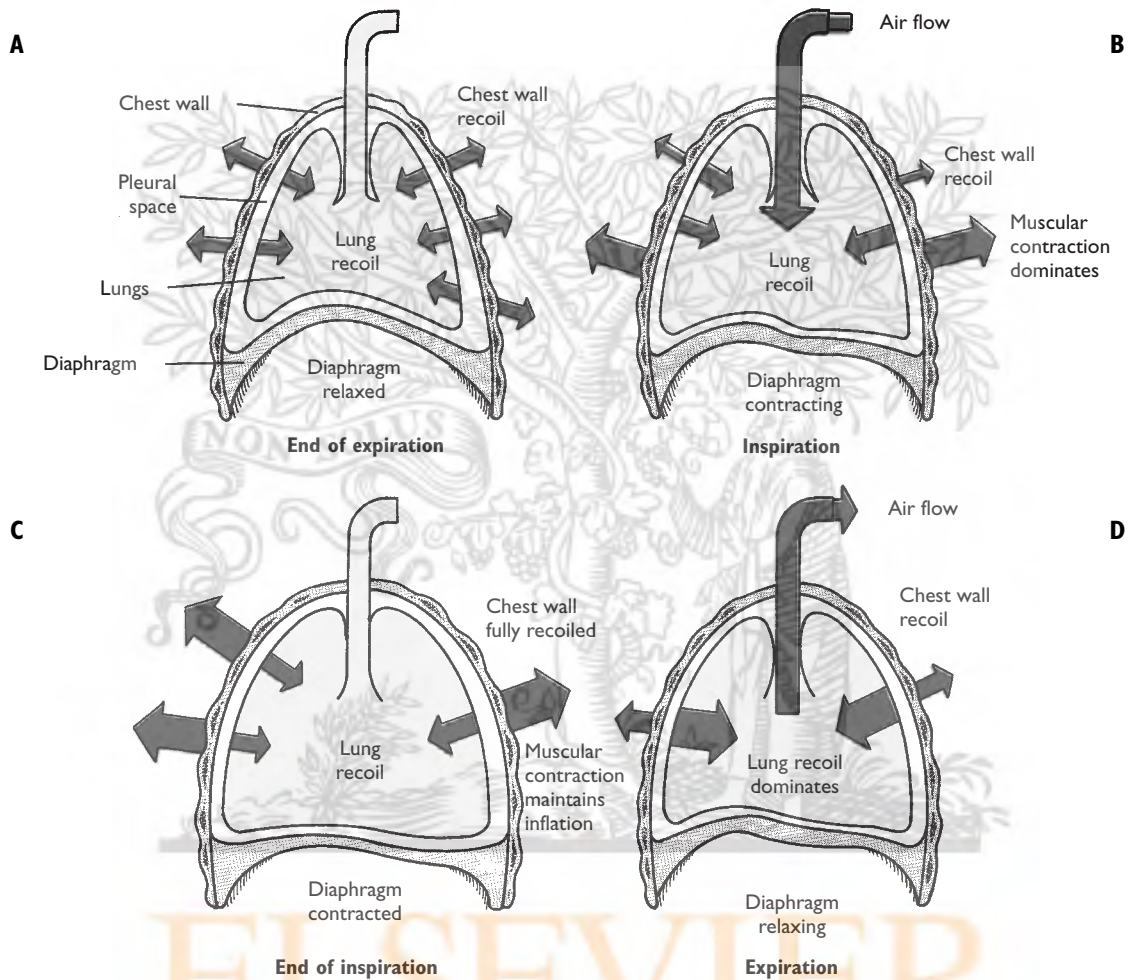


FIGURE 20.4 Interactions of forces during inspiration and expiration. **A**, Outward recoil of the chest wall equals inward recoil of the lungs at the end of expiration. **B**, During inspiration, contraction of respiratory muscles, assisted by chest wall recoil, overcomes tendency of lungs to recoil. **C**, At the end of inspiration, respiratory muscle contraction maintains lung expansion. **D**, During expiration, respiratory muscles relax, allowing elastic recoil of the lungs to deflate the lungs. From Huether SE, McCance KL. *Understanding pathophysiology*. 2nd edn. St Louis: Mosby; 2000.

Spirometry is used to measure timed expired and inspired volumes and, from this, calculation of how readily the lungs can be emptied and filled is possible.^{6,7} This assessment provides information on whether the lung disease is of an obstructive or restrictive nature. Acceptable spirometry measurement requires cooperation by the patient and knowledge of the technique required by the operator. Constant verbal reinforcement of technique throughout the spirometry test will help to produce favourable results (Fig 20.8).³⁻⁷

Overview of arterial blood gas (ABG) analysis

ABG sampling and analysis is the gold standard assessment of the patient's oxygenation and acid-base balance. It is commonly used in emergency departments and is vital to the management of the patient's condition. Knowledge of normal

values for each patient is paramount. Interpretation of these results should follow 5 simple steps (Box 20.1).^{3-6,8}

Step 1. PaO_2 refers to the measurement of the partial pressure of oxygen dissolved in arterial blood. The normal range should be 80 to 100 mmHg for a person breathing room air at sea level. Normal levels vary in infants and in people over 60 years of age. For older persons normal levels decrease as a result of a ventilation/perfusion (V/Q) mismatch and the normal ageing of the lung.^{2,3} A PaO_2 of less than the normal value is indicative of hypoxaemia which means the amounts of oxygen dissolved in the plasma is lower than normal. A PaO_2 level of 40 mmHg or below, at any age, indicates a life-threatening situation. The patient needs immediate administration of oxygen and/or mechanical ventilation as oxygenation of the tissues is severely compromised.^{3-6,8}

Step 2. pH refers to the acidity or alkalinity of the blood and the hydrogen ion (H^+) concentration of the plasma. The normal values for arterial blood pH are 7.35 to 7.45. The

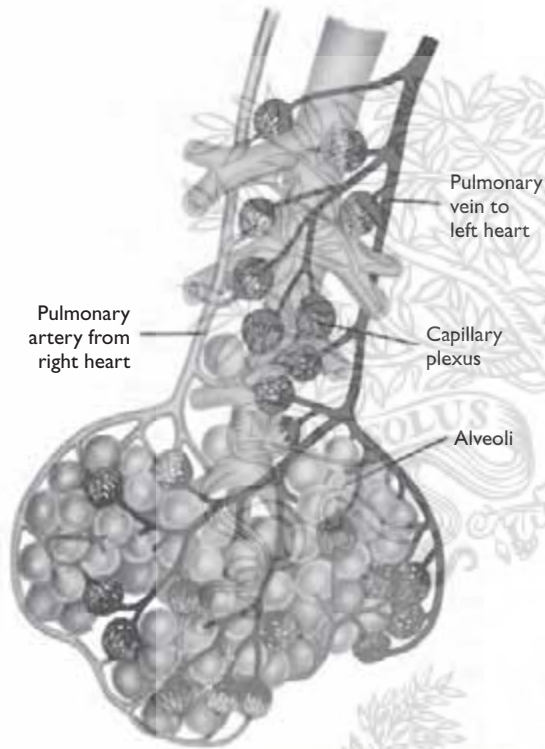


FIGURE 20.5 Terminal ventilation and perfusion units of the lung. Pulmonary arterial blood is venous (light grey), and pulmonary venous blood is oxygenated (dark grey). From Thompson JM, et al: Mosby's clinical nursing, 5th edn. St Louis: Mosby; 2002.

mean value for arterial blood pH is 7.40. Any result below this value is on the acidic side of average and any result above this value is on the alkaline side of average. An arterial blood pH of less than 7.35 is referred to as acidosis and a result of greater than 7.45 is referred to as alkalosis.^{3-6,8}

Step 3. $PaCO_2$ refers to the measurement of the partial pressure of carbon dioxide dissolved in arterial blood. The normal range should be 35 to 45 mmHg and does not change as a person ages. The body produces carbon dioxide during normal metabolism. The PaO_2 values indicate the ability of the patient to effectively ventilate to rid the body of carbon dioxide. The definition of respiratory acidosis is a $PaCO_2$ value above 45 mmHg. This is due to hypoventilation and can be the result of a number of conditions including COPD, over-sedation, head trauma or drug overdose. When the levels of $PaCO_2$ are greater than 50 mmHg ventilatory failure occurs.^{3-6,8} Acute ventilatory failure will have abnormal pH values of 7.30; this is because the body has not had enough time to compensate by attempting to bring the pH value back towards the normal value. Chronic ventilatory failure has near normal pH values of greater than 7.30. The definition of respiratory alkalosis is a $PaCO_2$ value of less than 35 mmHg. Respiratory alkalosis is due to hyperventilation and can be the result of a number of conditions including hypoxia, pulmonary embolus, hyperventilation and pregnancy. It can also be the result of a compensatory mechanism to a metabolic acidosis.^{3-6,8}

Step 4. The bicarbonate (HCO_3^-) result is a reflection of the renal function. The normal range is 22 to 26 mEq/L. The definition of a metabolic acidosis is a level below 22 mEq/L. This can be a result of renal failure, lactic acidosis, ketoacidosis or diarrhoea. The definition of a metabolic alkalosis is a level

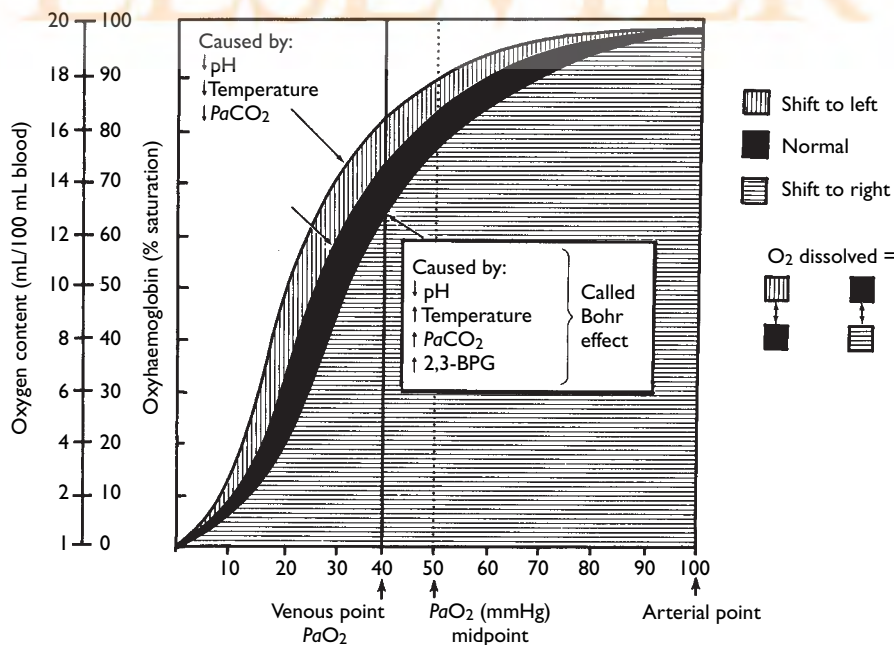


FIGURE 20.6 The oxygen-haemoglobin dissociation curve. Effects of acidity and temperature changes are shown. BPG, 2,3-biphosphoglycerate. Adapted from Thompson JM, et al. Mosby's clinical nursing, 5th edn. St Louis: Mosby; 2001.

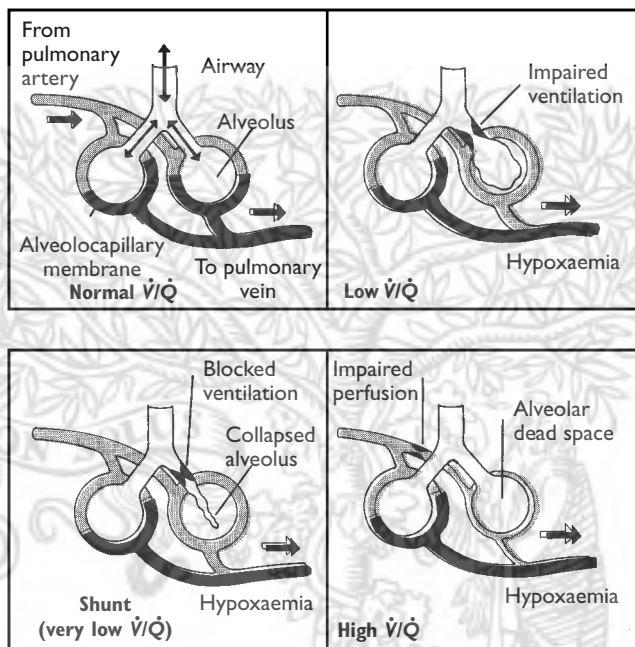


FIGURE 20.7 Ventilation–perfusion abnormalities. From Huether SE, McCance KL. Understanding pathophysiology. 2nd edn. St Louis: Mosby; 2000.

TABLE 20.1 Chest examination findings in common pulmonary problems				
Problem	Inspection	Palpation	Percussion	Auscultation
Chronic bronchitis	Barrel chest; cyanosis	↓Movement ↑Fremitus	Hyperresonant or dull if consolidation	Crackles; rhonchi; wheezes
Emphysema	Barrel chest; tripod position; use of accessory muscles	↓Movement	Hyperresonant or dull if consolidation	Crackles; rhonchi; diminished if no exacerbation
Asthma: In exacerbation	Prolonged expiration; tripod position; pursed lips	↓Movement	Hyperresonance sounds	Wheezes; ↓breath sounds ominous sign if no improvement (severely diminished air movement)
Not in exacerbation	Normal	↓Fremitus if hyperinflation Normal	Normal	Normal
Pneumonia	Tachypnoea; use of accessory muscles; dusky skin or cyanosis	Unequal movement if lobar involvement; ↑fremitus over affected area	Dull over affected areas	Early: bronchial sounds lower in chest Later: crackles; rhonchi
Atelectasis	No change unless involves entire segment, lobe	If small, no change; if large, ↑movement; ↓fremitus	Dull over affected areas	Crackles (may disappear with deep breaths); absent sounds if large
Pulmonary oedema	Tachypnoea; laboured respirations; cyanosis	↓Movement or normal movement	Dull or normal depending on amount of fluid	Fine or coarse crackles
Pleural effusion	Tachypnoea; use of accessory muscles	↓Movement ↑Fremitus above effusion; absent fremitus over effusion	Dull	Diminished or absent over effusion; egophony over effusion
Pulmonary fibrosis	Tachypnoea	↓Movement	Normal	Crackles

From Lewis SM, Heitkemper MM, Dirksen SR. Medical–surgical nursing: assessment and management of clinical problems. 5th edn. St. Louis: Mosby; 2000.

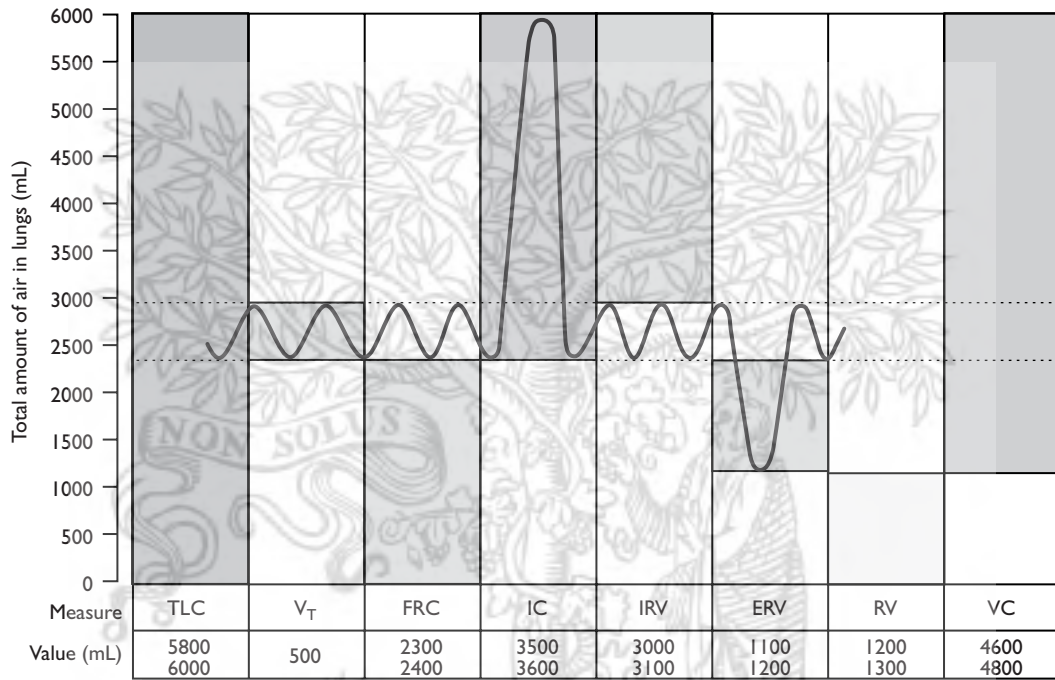


FIGURE 20.8 Lung volume measurements. All values are approximately 25% less in women. *TLC*, Total lung capacity; *V_T*, tidal volume; *FRC*, functional residual capacity; *IC*, inspiratory capacity; *IRV*, inspiratory reserve volume; *ERV*, expiratory reserve volume; *RV*, residual volume; *VC*, vital capacity. From Urden LD, Stacy KM, Lough ME. *Thelan's critical care nursing: diagnosis and management*. 5th edn. St Louis: Mosby; 2006.

BOX 20.1 Steps for interpretation of blood gas levels

- Step 1**
Look at the *PaO₂* level and answer the question, 'Does the *PaO₂* level show hypoxaemia?'
- Step 2**
Look at the pH level and answer the question, 'Is the pH level on the acid or alkaline side of 7.40?'
- Step 3**
Look at the *PaCO₂* level and answer the question, 'Does the *PaCO₂* level show respiratory acidosis, alkalosis, or normalcy?'
- Step 4**
Look at the *HCO₃⁻* level and answer the question, 'Does the *HCO₃⁻* level show metabolic acidosis, alkalosis, or normalcy?'
- Step 5**
Okay back at the pH level, and answer the question, 'Does the pH show a compensated or an uncompensated condition?'

From Urden LD, Stacy KM, Lough ME. *Thelan's critical care nursing: diagnosis and management*. 5th edn. St Louis: Mosby; 2006.

above 26 mEq/L and can be the result of loss of fluid and diuretic therapy.^{3-6,8}

Step 5. In an uncompensated condition (respiratory or metabolic): if the pH is abnormal, then the *PaCO₂*, *HCO₃⁻*, or both, will also be abnormal, because the body has not had enough time to return the pH value back to the normal range.

In a compensated condition (respiratory or metabolic): if the pH is normal and both the *PaCO₂* and *HCO₃⁻* values are abnormal then the body has had time to return the pH values back to the normal range (Table 20.2).^{3-6,8}

ASTHMA

Australasia has one of the highest rates of asthma in the world.⁹ There are about 600,000 people in New Zealand and 2.2 million Australians with asthma.^{9,10}

Asthma is a chronic inflammatory disease affecting the airways. It is characterised by narrowing of the airways and attacks are triggered by spasms of the smooth muscle in the walls of the smaller bronchioles.^{9,10} This spasm causes the airways to close, either partially or completely, and this process is known as bronchoconstriction. In the acute phase excessive mucus is also produced, which can further clog the bronchioles and worsen the attack. Asthmatics have hyperreactive airways and are susceptible to certain triggers. Common triggers include:

- allergies (foods, pollen, animals, household dust and mould)
- tobacco smoke and air pollutants
- exercise and cold weather
- chest infections, rhinitis and sinusitis
- medications (aspirin and NSAIDs).

Signs and symptoms of asthma include wheezing and cough, dyspnoea and orthopnoea, tachycardia and tachypnoea,

TABLE 20.2 Arterial blood gases with various stages of compensation

Cause		$PaCO_2$	Uncompensated		Partially compensated		Fully compensated	
Values			pH	HCO_3^-	pH	HCO_3^-	pH	HCO_3^-
Respiratory alkalosis	Hyperventilation	↓	↑	Normal	↑	↓	Normal	↓
Respiratory acidosis	Drug ingestion (hypoventilation)	↑	↓	Normal	↓	↑	Normal	↑

Cause		HCO_3^-	Uncompensated		Partially compensated		Fully compensated	
Values			pH	$PaCO_2$	pH	$PaCO_2$	pH	$PaCO_2$
Metabolic alkalosis	Severe vomiting	↑	↑	Normal	↑	↑	*	↑
Metabolic acidosis	Diabetic ketoacidosis	↓	↓	Normal	↓	↓	*	↓

*Metabolic cannot be fully compensated to a normal pH by the respiratory system. HCO_3^- , bicarbonate; $PaCO_2$, carbon dioxide pressure.

low oxygen saturation, chest tightness and hyperresonance as a result of lung hyperinflation, use of accessory muscles and low peak flow.^{3,4}

Oxygen and corticosteroids should be given as a priority to reduce airway inflammation in asthma; steroids usually take about 6 hours to work. Nebulised or inhaled medication should include a beta₂-agonist to produce bronchodilation and an anticholinergic medication to inhibit contraction of bronchial smooth muscle.^{3-6,9,10}

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

COPD is the single leading cause of death and it continues to increase; it is now the third largest contributor to the burden of disease in Australia.^{11,12} COPD involves a combination of asthma, chronic bronchitis and emphysema which all produce a degree of airway obstruction.³⁻⁶ Smoking remains the leading cause of COPD. Less common factors include genetic disorders such as α_1 -antitrypsin deficiency, environmental and occupational exposure to pollutants. COPD patients are classically described as 'pink puffers' (predominantly emphysema) or 'blue bloaters' (predominantly chronic bronchitis).^{3-6,11,12}

In chronic bronchitis hypertrophy and hyperplasia of the goblet cells lining the bronchial airways give rise to a productive cough. This secretion signifies underlying inflammation and the productive cough is present for at least 3 months per year for at least 2 consecutive years. 'Blue bloaters' are usually obese, have a reduced cardiac output resulting in hypoxaemia, right ventricular failure and retain CO_2 .^{3-6,11,12} In emphysema the alveolar walls diminish, resulting in permanently large air spaces that remain filled with air during expiration. This loss of surface area for gas exchange results in a reduction of oxygen levels and to compensate, hyperventilation occurs. 'Pink puffers' are usually thin or even cachectic with a barrel chest (Table 20.3).^{4-6,11,12}

Signs and symptoms of COPD include dyspnoea and cyanosis, a productive cough and orthopnoea, wheeze, crackles and hyperresonance. Treatment should include oxygen therapy, focusing on the potential for CO_2 retention, so an oxygen saturation level of 90% via pulse oximetry is usually acceptable. Medication should include nebulised or inhaled beta₂-agonist and anticholinergic drugs to produce bronchodilation. Corticosteroids are useful if there is an asthmatic component and antibiotic therapy may be necessary as bacteria are a common cause of exacerbations. Non-invasive ventilation should also be considered and is discussed later in this chapter.^{3-6,11,12}

TABLE 20.3 Chronic Obstructive Pulmonary Disease

Pink puffer	Blue bloater
<i>Prominent disease</i>	
Emphysema	Chronic bronchitis
<i>History</i>	
Dyspnoea for a long period Cough at late onset Minimal to no sputum production	Productive cough Eventual dyspnoea
<i>Physical assessment</i>	
Pursed lip breathing; distant heart sounds; barrel chest; sitting in tripod position; accessory muscle use; wheezing	Cyanotic; symptoms of right heart failure; rhonchi; obese; use of accessory muscles
<i>Course of disease</i>	
Progressive dyspnoea Eventual cachexia Respiratory failure	Frequent, recurrent, pulmonary infections Worsening cough Cardiac or respiratory failure

PNEUMONIA

Pneumonia is an acute inflammation or infection of the alveoli. The alveolar sacs fill with fluid and dead white blood cells thus diminishing the capacity for air within the lungs. The blood PaO_2 will be drastically reduced because oxygen is unable to diffuse as readily through the inflamed alveoli. CO_2 levels are usually normal as it diffuses more easily than O_2 .^{3,4} Bacterial invasion can occur as a result of inhalation, aspiration, migration and colonisation. Most cases presenting to the emergency department are community acquired and are usually less virulent than hospital acquired pneumonia.^{5,6}

Signs and symptoms include cough and fever, hypoxaemia and dyspnoea, tachycardia and tachypnoea, chest pain, crackles or wheezing (Table 20.4). Treatment with oxygenation and monitoring by pulse oximetry may be required if oxygen levels are low. Antibiotics are routinely given empirically and the use of nebulised or inhaled bronchodilators as needed may relieve symptoms. Blood and sputum cultures may be obtained.³⁻⁶

ACUTE PULMONARY OEDEMA (APO)

Causes of APO are either cardiac or non-cardiac in origin. Non-cardiogenic pulmonary oedema can be caused by a variety of conditions such as high altitude, blood transfusion and acute lung injury.

Cardiogenic pulmonary oedema occurs when there is damage to the left ventricle from acute myocardial infarction (AMI), left ventricular dysfunction, valvular heart disease or fluid overload.¹³⁻¹⁵

The increased blood or pressure in the pulmonary circulation increases capillary pressure forcing intravascular fluid to seep out into the lung tissue and alveoli. This increase of fluid diminishes the degree of oxygen in the blood and impairs gas

exchange, which leads to respiratory distress. APO interferes with the diffusion of oxygen, depressing the arterial partial pressure of oxygen and leading to tissue hypoxia.²⁻⁴

Signs and symptoms of APO include breathlessness and anxiety as the patient may have a feeling of suffocation. Respiratory rate increases and the patient may be gasping for breath and using accessory muscles to breathe. Patients typically cannot lie flat and have to sit bolt upright. On chest auscultation loud crackles can be heard and are occasionally audible without the use of a stethoscope. Pink frothy sputum caused by red blood cells and fluid from the alveoli is characteristic of APO as is diaphoresis and cold, ashen skin which is indicative of low cardiac output.⁴⁻⁶

Therapy for APO should focus on patient comfort and reducing anxiety. Close observation of vital signs and oxygen saturation, cardiac rhythm and a 12-lead ECG should be used to assess the underlying cause. IV morphine will help reduce anxiety and produce peripheral vasodilation which reduces venous return and therefore reduces preload and subsequent afterload. IV diuretics such as frusemide are given to decrease preload and eliminate fluid from the circulatory system. IV nitrates are given also to decrease preload and can serve to dilate the coronary arteries if ischaemic heart disease is thought to be the precipitating factor. Administration of oxygen to maintain SpO_2 levels above 90% will help resolve the respiratory distress. The use of non-invasive ventilation by continuous positive airway pressure (CPAP) may be considered.^{3-6,13,16}

ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

ARDS refers to a form of non-cardiogenic pulmonary oedema which results in hypoxaemic respiratory failure. ARDS is an inflammatory syndrome in which the alveolar capillary membrane is damaged; as a result a protein rich oedema fills the air spaces.^{4-6,17,18} Furthermore, surfactant production

TABLE 20.4 Precipitating conditions of pneumonia

Condition	Aetiologies
Depressed epiglottal and cough reflexes	Unconsciousness, neurologic disease, endotracheal or tracheal tubes, anaesthesia, ageing
Decreased cilia activity	Smoke inhalation, smoking history, oxygen toxicity, hypoventilation, intubation, viral infections, ageing, COPD
Increased secretion	COPD, viral infections, bronchiectasis, general anaesthesia, endotracheal intubation, smoking
Atelectasis	Trauma, foreign body obstruction, tumour; splinting, shallow ventilations, general anaesthesia
Decreased lymphatic flow	Heart failure, tumour
Fluid in alveoli	Heart failure, aspiration, trauma
Abnormal phagocytosis and humoral activity	Neutropenia, immunocompetent disorders, patients receiving chemotherapy
Impaired alveolar macrophages	Hypoxaemia, metabolic acidosis, cigarette smoking history, hypoxia, alcohol use, viral infections, ageing

Urden LD, Stacy KM, Lough ME. Thelan's critical care nursing: diagnosis and management. 5th edn. St Louis: Mosby; 2006.
COPD, chronic obstructive pulmonary disease

decreases which all contributes to the end result of smaller airway diameters, injury to the pulmonary vasculature and compromised oxygenation.

A number of conditions can predispose a patient to ARDS and these include pneumonia and sepsis, aspiration and chemical inhalation of toxic fumes, trauma, chronic alcohol abuse and massive blood transfusion (Box 20.2).^{4-6,17,18}

Signs and symptoms are similar to cardiogenic APO and therapeutic interventions include the maintenance of ABC. Patients require endotracheal intubation and admission to ICU.^{3-6,13,17,18}

INHALATION INJURY

Three factors to consider when evaluating a patient with an inhalation injury are exposure to asphyxiants (or asphyxiation), thermal or heat injury, and smoke poisoning (or pulmonary irritation). Exposure to asphyxiants is the most frequent cause of early mortality, with carbon monoxide (CO) the most frequent asphyxiant from a fire.⁴⁻⁶

Carbon monoxide poisoning

The affinity of CO for haemoglobin is 240 times greater than that of oxygen, resulting in oxygen being displaced from haemoglobin. Without a mechanism for oxygen transport, tissue becomes hypoxic. Carboxyhaemoglobin (COHb) levels greater than 10% indicate smoke inhalation; however, smokers or individuals exposed to automobile exhaust fumes can have baseline COHb levels of 10% to 15%. Fetal haemoglobin binds even more quickly with CO, so the fetus is at greater risk for injury from smoke inhalation.

The signs and symptoms of acute CO poisoning can be related to the concentration at the time of exposure. Acute exposure to CO usually causes central nervous system and cardiovascular toxicity. These tissues have high blood flow and oxygen demands. Table 20.5 summarises the signs and symptoms and Table 20.6 summarises the clinical features of CO poisoning.¹⁹

DIAGNOSIS

The differential diagnosis includes cerebrovascular disease, psychiatric illness, migraine and cardiovascular disease. These conditions may co-exist with acute CO poisoning. The diagnosis of CO poisoning should be considered whenever multiple members of the same family or from the same workplace present with non-specific symptoms, especially headache, within 24 hours of each other.

Measurement of an elevated COHb concentration confirms the diagnosis; arterial blood gas analysis may demonstrate an acidosis, although alkalosis has been reported. Blood gas machines calculate the oxygen saturation from measured partial pressure and give a false elevated result in CO poisoning. Similarly, pulse oximeters do not distinguish between oxy-Hb and COHb and give false high readings.

MANAGEMENT

Initial management is directed towards securing the airway and stabilising respiration and circulation. The patient should immediately be given 100% oxygen therapy, ideally through a tight fitting nonrebreathing facemask. ET tube intubation is advised for agitated or comatose patients. Hyperbaric oxygen (HBO) has been purported to offer additional benefits, in particular in reducing the risk of neuropsychological sequelae.

BOX 20.2 Conditions predisposing to acute respiratory distress syndrome

<i>Infectious causes</i>	<i>Metabolic disorders</i>	<i>Drug-related</i>
Gram-negative sepsis	Pancreatitis	Dextran 40
Bacterial pneumonia	Uraemia	Heroin
Viral pneumonia	Diabetic ketoacidosis	Methadone
<i>Pneumocystis carinii</i>	<i>Inhaled toxic agents</i>	Salicylates
Tuberculosis	Oxygen	Thiazides
<i>Aspiration</i>	Smoke	Propoxyphene
Gastric	Toxic gases	Colchicine
Freshwater and salt water (drowning)	<i>Haematologic disorders</i>	<i>Other</i>
Ethylene glycol	Massive blood transfusion	Radiation pneumonitis
Hydrocarbon fluids	Disseminated intravascular coagulation	Amniotic fluid emboli
<i>Shock</i>	Transfusion reaction	Increased intracranial pressure
Septic	Postcardiopulmonary bypass or	High altitude
Traumatic	resuscitation	Fluid overload
Haemorrhagic	<i>Immunologic reactions</i>	Eclampsia
<i>Trauma</i>	Drug allergy	Goodpasture's syndrome
Generalised	Anaphylaxis	Drug overdose
Fat embolism		Bowel infarction
Lung contusion		Dead fetus
Multiple major fractures		
Head injury		
Burns		

From Newberry L, ed. Sheehy's emergency nursing: principles and practice. 5 edn. St Louis: Mosby; 2003.

HBO acts by increasing dissolved O₂ content in the blood, accelerating elimination of CO, and possible prevention of lipid peroxidation in the brain.¹⁹

Cardiac monitoring for arrhythmias and 12-lead ECG should be performed as should intravenous cannulation and blood testing for full blood count, electrolyte imbalance, urea and creatinine, COHb, blood sugar level and cardiac enzymes. Serum lactate is a better guide to tissue acidosis than arterial blood gases.¹⁹

An additional consideration with CO poisoning is the potential for exposure to cyanide which can be produced from the combustion of wool, silk, plastics, paper products, rubber and polyurethane (see Ch 27).¹⁹

TABLE 20.5 Signs and symptoms related to COHb level at time of exposure

COHb level (%)	Signs and symptoms
0	None
10	Frontal headache
20	Throbbing headache, shortness of breath on exertion
30	Impaired judgement, nausea, fatigue, visual disturbances, dizziness
40	Confusion, loss of consciousness
50	Seizures, coma
60	Hypotension, respiratory failure
70	Death

From Cruise DC. Carbon monoxide. In: Cameron P, Jelinek G, Kelly A, et al, eds. Textbook of adult emergency medicine. 2nd edn. Edinburgh: Churchill Livingstone; 2004:834.

THERMAL OR HEAT INJURY

Unless the cause of heat injury is from steam, explosive or volatile gas, or hot liquid aspiration, it is rare to have heat injury below the oropharyngeal airway. The respiratory tract's ability to efficiently exchange heat, combined with closure of the glottis, protects the airway from extreme heat. Extreme heat in the upper airway initially causes erythema, oedema and blisters of the mucosa. Within the first 24 to 48 hours after the injury, the airway can become obstructed from the increasing mucosal oedema.¹⁹

Smoke poisoning

Smoke poisoning refers to inhalation of toxic gases, such as hydrogen chloride, phosgene, ammonia and sulfur dioxide. These toxins damage pulmonary epithelial cells and destroy epithelial cilia, leading to mucosal oedema. Surfactant production decreases, followed by atelectasis. Clinically the patient develops pulmonary oedema, usually within 24 to 48 hours of the initial injury.

Initial nursing care is directed at maintaining a patent airway and supporting the patient's haemodynamic status. A secure airway and oxygen administration are important in all patients with an inhalation injury or the possibility of inhalation injury.⁵

NEAR DROWNING

The rate of near drowning is not accurately known because of nomenclature, definitions, and the difficulty in collecting the numbers of attendances related to near drowning, but it is estimated at between 2 and 20:1.¹⁹ Submersion related injuries occur when a victim is submerged unexpectedly, air

TABLE 20.6 Clinical features of carbon monoxide poisoning

System	Symptoms	Pathology	Diagnostic
CNS	Early: confusion, coma, seizures Late: psychoses, dementia, parkinsonism, ataxia, peripheral neuropathy, gait disturbance	Brain oedema, encephalopathy Cerebral atrophy, basal ganglia lesions	EEG CT scan
Cardiac	Arrhythmias, hypotension angina, tachycardia	Myocardial ischaemia	ECG, CK, CK-MB, Troponin
Pulmonary	Shortness of breath	Pulmonary oedema	CXR
Ophthalmological	Visual disturbances	Flame-shaped retinal haemorrhages, cerebral lesions, retrobulbar neuritis, papilloedema	Fundoscopy
Renal	Acute failure	Myoglobinuria	Renal function tests, serum myoglobin, urine myoglobin
Muscular	Ischaemia	Compartment syndrome, rhabdomyolysis	
Auditory and vestibular	Hearing loss, nystagmus, tinnitus		

From Cruise DC. Carbon monoxide. In: Cameron P, Jelinek G, Kelly A, et al, eds. Textbook of adult emergency medicine. 2nd edn. Edinburgh: Churchill Livingstone; 2004:834.

hunger develops and hypoxia ensues. The hypoxaemia occurs as a result of laryngospasm, an involuntary gasp occurs and water is aspirated into the lungs: this is referred to as 'wet drowning'. 'Dry drowning' occurs in approximately 10% to 20% of drownings and is a result of severe laryngospasm, which actually prevents water from entering the lungs. Freshwater and seawater submersion leads to profound hypoxia, pulmonary damage and death from the aspiration of water. Water floods the alveoli and thus impairs gas exchange and loss of surfactant. Contaminants such as algae, chlorine sand and mud can worsen pulmonary injury and increase the risk of infection.^{19,20} There is a risk of submersion hypothermia if the water temperature is less than 25°C. If submersion occurs in water temperatures of less than 5°C hypothermia rapidly develops and provides some protection against hypoxia but this protection is usually only seen in small children.

Injuries to the spinal cord are frequently seen in adolescents and young adults from diving or falling into the water head first and spinal immobilisation is required. Causes of submersion injury vary: they can be the result of a lack of child supervision, the inability to swim, alcohol use around water and risk taking behaviour. All these can result in submersion injuries as can an underlying medical condition.^{19,20}

Signs and symptoms vary and are directly related to the extent of hypoxia and the length of time submerged. The clinical signs include respiratory distress, bronchospasm and loss of consciousness, hypotension and arrhythmias. Outcomes are determined by age, length of submersion, temperature and type of fluid and associated injuries. Treatment should begin with ABC and advanced resuscitation if necessary. Supplemental oxygen and respiratory status should be assessed and intubation and mechanical ventilation are indicated when there is inadequate oxygen saturation. Submersion produces an insult to many of the body's systems and ideally needs to be managed in a critical care setting.

Victims are at risk from acute lung injury that can progress to ARDS.^{3,19,20} Assisted ventilation may be required with the use of continuous positive airway pressure. Intravenous fluids should be administered with care and there should be close observation of haemodynamic status to prevent the precipitation of pulmonary oedema. Hypothermic patients should be re-warmed at a rate that corresponds to the rate of onset of hypothermia, but this is often difficult to gauge. A nasogastric tube can aid decompression and emptying of the stomach and investigations should include chest radiography, 12-lead ECG and arterial blood gas analysis.^{3,6,20}

PULMONARY EMBOLUS (PE)

In the general population PE affects 0.5 to 1 per 1000 people in Australia and it is the third largest cause of death in the USA. PE remains one of the commonest causes of preventable death among hospital inpatients in Australia.²¹ Clinical diagnosis of PE is unreliable and diagnostic imaging procedures such as VQ scan must be used. The most common cause of PE is the embolisation of thrombi from the deep leg veins. This is due to stasis of the blood, damage to the epithelium of the blood

vessel wall or changes in coagulation. The clot dislodges and travels within the venous system through the right side of the heart and into the pulmonary circulation until it becomes lodged in a pulmonary vessel and obstructs blood flow to the lung, thus reducing perfusion to the affected area. Other sources of PE include the right ventricle and the pelvic veins. Nonthrombotic emboli can include fats, amniotic fluid and tumours.^{3,4} Risk factors are listed in Box 20.3.

Alveolar dead space occurs when a portion of the lung is being adequately ventilated but receiving no perfusion due to the clot blocking the circulating blood. This ventilation is wasted, as it is not associated with gas exchange. Bronchoconstriction occurs locally to limit alveolar dead space and as a result of low levels of O₂ and CO₂. Shunting of blood occurs as circulation is redistributed to the unaffected areas of the lungs; this results in the blood returning to the left side of the heart without being oxygenated which further precipitates hypoxaemia. Pulmonary vascular resistance increases during PE and impacts on the workload of the right ventricle as the result of pulmonary hypertension, and failure of the right ventricle occurs.³⁻⁶

Signs and symptoms of PE are varied and the condition is usually under-diagnosed. Nonspecific findings can lead the emergency nurse to pursue other diagnostic avenues as symptoms are related to other complaints.^{3,4} Common symptoms include chest pain and dyspnoea, tachycardia and tachypnoea, diaphoresis, fever and cough, haemoptysis, anxiety, apprehension and restlessness, pleural friction rub, crackles and wheeze on auscultation, pallor or cyanosis and hypotension and syncope. Therapy should include the administration of supplemental oxygen and monitoring of oxygen saturation along with observation of vital signs. Analgesia and anticoagulation can be administered in the emergency department. Oral anticoagulation is not usually administered in the emergency department. Intravenous heparinisation or the use of a low-molecular weight-based heparin can be commenced immediately. Fibrinolytic therapy can be administered and will depend on the clinical status of the patient.^{3-6,21}

Computed tomography pulmonary angiography (CTPA)

CTPA is considered the gold standard for diagnosing a PE. There are other diagnostic tests that cannot reliably be used to diagnose PE. These include D-Dimer and the \dot{V}/\dot{Q} scan. The CT scanner can differentiate between structures that are different in density, such as tissue and air, it can visualise the lung parenchyma and pulmonary vasculature. It is this high sensitivity which makes CTPA convenient, cost effective, accurate and rapid in the diagnosis of PE.^{21,22}

UNDERWATER SEALED DRAINS (UWSD)

UWSD are used for the treatment of a variety of complications. They are inserted for the reduction of a pneumothorax, haemothorax, and large pleural effusion and to remove empyema from the pleural space. A pneumothorax can

BOX 20.3 Risk factors for pulmonary thromboembolism*Predisposing factors*

Venous stasis
 Atrial fibrillation
 Decreased cardiac output (CO)
 Immobility
 Injury to vascular endothelium
 Local vessel injury
 Infection
 Incision
 Atherosclerosis
 Hypercoagulability
 Polycythaemia

Precipitating conditions

Previous pulmonary embolus
 Cardiovascular disease
 Heart failure
 Right ventricular infarction
 Cardiomyopathy
 Cor pulmonale
 Surgery
 Orthopaedic
 Vascular
 Abdominal
 Cancer
 Ovarian
 Pancreatic
 Stomach
 Extrahepatic bile duct system
 Trauma (injury or burns)
 Lower extremities
 Pelvis
 Hips
 Gynaecologic status
 Pregnancy
 Postpartum
 Birth control pills
 Oestrogen replacement therapy

From Urden LD, Stacy KM, Lough ME: *Thelan's critical care nursing: diagnosis and management*. 3rd edn. St Louis: Mosby; 1998:669.

develop as the result of traumatic injury to the chest, as an iatrogenic cause from the insertion of a central line or surgery or spontaneously.^{3,4,23} Spontaneous primary pneumothoraces occur in individuals without any underlying lung disease. They are usually tall thin men under the age of 40 years and are as a result of a ruptured subpleural bleb into the pleural space. Secondary pneumothoraces occur in individuals with underlying COPD or pulmonary fibrosis. A haemothorax usually develops as a result of chest injury. A pleural effusion is the collection of fluid within the pleural space. Pleural effusions are usually transudative (watery) and are associated with a problem from outside the lungs and are usually bilateral (Box 20.4). Exudative pleural effusions consist of a protein rich substance that is usually associated with inflammation or infection. Empyema is a collection of pus in the pleural space.^{3,4,23}

BOX 20.4 Causes of pleural effusion*Transudative*

After abdominal surgery
 Cirrhosis
 Congestive heart failure
 Pericardial disease
 Peritoneal dialysis
 Pulmonary embolus

Exudative

Collagen vascular diseases (systemic lupus erythematosus, rheumatoid pleuritis)
 Infectious diseases
 Neoplastic diseases
 Trauma

From Lewis SM, et al. *Medical–surgical nursing: assessment and management*. 5th edn. St Louis: Mosby; 2000.

Insertion of an UWSD is aimed at evacuating the substance (air, blood, fluid or pus) from the pleural cavity. The volume of the lung is reduced first by elastic recoil and then compression of the lung. With each breath the lung is seriously compromised and the mechanics of breathing are drastically impaired and a ventilation/perfusion imbalance occurs. Chest tubes or smaller pigtail catheters are inserted into the pleural space and the distal end is connected to the drainage system. During exhalation there is positive pressure in the chest. This pressure pushes the air or liquid from the pleural space into the chest tube or pigtail catheter and out into the drainage system. This reinflates the lung and the water seal chamber of the drainage system acts as a one-way valve allowing the air or liquid to evacuate from the chest but not enter it.^{3,4,23,24}

If the patient has a small, uncomplicated pneumothorax with little or no drainage that doesn't require suction, the chest tube may be connected to a Heimlich valve. The Heimlich valve was first produced in 1962 and introduced into more widespread use in the mid-1960s during the conflict in Southeast Asia. It was originally designed to allow emergency treatment of pneumothorax on the frontlines so that servicemen would survive to receive definitive care at field hospitals, or to facilitate patient transport by helicopter between hospital stations.²⁵

The device is constructed from a piece of latex rubber tubing, like a Penrose drain, encased in a plastic tube. The tubing collapses on itself at rest, but when positive pressure is applied to the device from the chest—air exiting the chest through a chest tube from a pleural air leak during exhalation—the tubing opens and air escapes. During inspiration, the tubing closes again, creating a one-way valve through which air can leave the chest but not re-enter.²⁶

Complications associated with UWSD are perforation of the heart, lung, liver, stomach and aorta, infection and subcutaneous emphysema. Common problems with an UWSD include decreased drainage volume; this can be due to a reduction of the liquid within the pleural space and the improvement of the condition or an obstruction in the tubing. Tubing should be checked for kinks and tubing should be changed if it is occluded or blocked. Bubbling can be indicative

of air being removed from the pleural space when the patient coughs or exhales.²³ Continuous bubbling may signify a leak in the system, either from loose connections or around the insertion site. If the collection bottle falls over it should be quickly repositioned upright to re-establish the underwater seal. The patient's vital signs and UWSD observations should be performed as per hospital policy.^{3,4,23} If there is accidental disconnection of the chest tube from the drainage bottle the tube can be immediately clamped until a clean system can replace the underwater seal or the end of the chest tube can be immersed into a bottle of sterile water to temporarily seal the system until a clean system can be reconnected. It is also important never to lift the UWSD bottle above the insertion site or level of the chest.^{23,24} On insertion it is common practice to insert a purse string suture around the chest tube. If the chest tube were accidentally removed the purse string suture should be immediately tied. Vital signs and close observation of the patient should follow in conjunction with notification of the medical team.^{3–6,23,24}

Rapid sequence intubation (RSI)

Rapid sequence intubation (i.e. rapid sequence induction) is a technique used for the emergency management of the airway. The basic theory behind RSI is that all patients presenting to the emergency department have a full stomach and therefore pose a significant aspiration risk during intubation.⁴ To minimise the risks of aspiration intubation must be performed in a rapid, safe and controlled manner to stabilise and maintain the airway. RSI is the almost simultaneous administration of an anaesthetic induction agent with a paralysing dose of a neuromuscular blocking agent followed by the insertion of an endotracheal (ET) tube.^{4,27} Preoxygenation is maintained for a few minutes prior to the administration of the induction agents and paralysing agents. Cricoid pressure is applied during intubation and is maintained until the patient is successfully intubated and the emergency physician is happy with the ET tube placement.^{4,27}

PRINCIPLES OF VENTILATION

Mechanical ventilation is used for a variety of clinical conditions. The principle aim of mechanical ventilation is to support gas exchange by increasing alveolar ventilation and subsequent arterial oxygenation (Table 20.7). Mechanical ventilation increases lung volumes by increasing tidal volumes and minute ventilation, increasing functional residual capacity and reducing the work of breathing with positive airway pressures.

Mechanical ventilation is used for the management and intervention of respiratory failure. There are two types of respiratory failure, Type I respiratory failure (hypoxaemic) and Type II respiratory failure (hypercapnic).^{2–4}

Primary causes of hypoxaemia include:

- \dot{V}/\dot{Q} mismatch
- hypoventilation of the alveoli
- diffusion impairment (refers to the movement of gas

across the alveolar–capillary membrane and is common in interstitial lung disease such as pulmonary fibrosis)

- decreased inspired oxygen (when inspired oxygen is less than the atmospheric 21%, such as at high altitudes). Primary causes of hypercapnic respiratory failure include:
- a decreased ventilatory drive (due to CNS depression by drugs, obesity, sleep apnoea, trauma and brainstem lesions)
- respiratory muscle fatigue or failure (neuromuscular dysfunction, Guillain-Barré syndrome, muscular dystrophy and polymyositis)
- increased work of breathing (from COPD, asthma, pneumothorax, pleural effusion, rib fractures) (see Ch 46).^{2–4}

Non-invasive ventilation

Non-invasive positive pressure ventilation (NIPPV) techniques do not require endotracheal intubation and are increasingly used for Types I and II respiratory failure. There are two types of non-invasive mechanical ventilation used widely in the ED.^{2–6}

Continuous positive airway pressure (CPAP) exerts a positive pressure throughout the respiratory cycle. The positive end expiratory pressure (PEEP) improves the functional residual capacity and recruits alveoli by keeping the collapsed alveoli opened and partly inflated, thus preventing atelectasis. CPAP/PEEP improves oxygenation because the residual air in the recruited alveoli continues to exchange gases. CPAP/PEEP also reduces pulmonary oedema (cardiogenic) by forcing the interstitial fluid out of the alveoli, back into the pulmonary circulation.^{3,4,27–29}

Bi-level positive airway pressure (BiPAP) administers an inspiratory positive airway pressure (IPAP) and an expiratory positive airway pressure (EPAP). When the patient takes a breath in the bi-level ventilator delivers a higher level of positive airway pressure (IPAP) which increases tidal volume and minute volume by increasing the size of the breath. In the fatigued patient this reduces the work of breathing and improves the ability to effectively clear carbon dioxide.^{3,4,28} The lower level of positive airway pressure occurs during expiration (EPAP) and is the same as CPAP and PEEP. During BiPAP therapy two levels are prescribed: IPAP and EPAP. The difference between the two numbers creates an amount of pressure support which splints open the airways and allows more volume and therefore carbon dioxide to be exhaled with each breath.^{3,4,27–29}

Common complications associated with NIPPV are:

- patient discomfort (resistance to expiration from the PEEP/EPAP makes breathing out uncomfortable. Also the tight fitting mask can cause discomfort)
- pressure ulcers (ulcers occur around the bridge of the nose and the ears from masks and certain head straps)
- hypercapnia (CPAP can also trap gases within the circuit resulting in high CO₂ levels)
- cardiovascular instability (CPAP/BiPAP increases intrathoracic pressure impeding venous return)

TABLE 20.7 Modes of mechanical ventilation

Mode of ventilation	Clinical application	Nursing implications
Continuous mandatory (volume or pressure) ventilation (CMV) also known as assist-control (A/C) ventilation: delivers gas at preset tidal volume or pressure (depending on selected cycling variable) in response to patient's inspiratory efforts and will initiate breath if patient fails to do so within preset time	Volume controlled– (VC-) CMV is used as the primary mode of ventilation in spontaneously breathing patients with weak respiratory muscles Pressure controlled– (PC-) CMV is used in patients with decreased lung compliance or increased airway resistance particularly when the patient is at risk for volutrauma	Hyperventilation can occur in patients with increased respiratory rates Sedation may be necessary to limit the number of spontaneous breaths Patient on VC-CMV should be monitored for volutrauma Patient on PC-CMV should be monitored for hypercapnia
Pressure-regulated volume control ventilation (PRVCV): a variation of CMV that combines both volume and pressure features; delivers a preset tidal volume using the lowest possible airway pressure; airway pressure will not exceed preset maximum pressure limit	PRVCV is used in patients with rapidly changing pulmonary mechanics (airway resistance and lung compliance), thus limiting potential complications	
Pressure-controlled inverse ratio ventilation (PC-IRV): PC-CMV mode in which the inspiratory-to-expiratory (I:E) time ratio is greater than 1:1	PC-IRV is used in patients with hypoxaemia refractory to PEEP; the longer inspiratory time increases functional residual capacity and improves oxygenation by opening collapsed alveoli, and the shorter expiratory time induces auto-PEEP that prevents alveoli from recollapsing	Requires sedation and/or pharmacologic paralysis because of discomfort Increased intrathoracic pressure can result in excessive air trapping and decreased cardiac output
Intermittent mandatory (volume or pressure) ventilation (IMV) also known as synchronous intermittent mandatory ventilation (SIMV): delivers gas at preset tidal volume or pressure (depending on selected cycling variable) and rate while allowing patient to breathe spontaneously; ventilator breaths are synchronised to patient's respiratory effort	Volume controlled– (VC-) IMV is used both as a primary mode of ventilation in a wide variety of clinical situations and as a weaning mode Pressure controlled– (PC-) IMV is used in patients with decreased lung compliance or increased airway resistance when the need to preserve the patient's spontaneous effects is important	May increase the work of breathing and promote respiratory muscle fatigue Patient should be monitored for hypercapnia, particularly with PC-IMV
Adaptive support ventilation (ASV): ventilator automatically adjusts settings to maintain 100 mL/min/kg of minute ventilation; pressure support	ASV is a computerised mode of ventilation that increases or decreases ventilatory support based on patient needs; can be used with any patient requiring volume controlled ventilation	Not intended as a weaning mode. Adapts to changes in patient position
Constant positive airway pressure (CPAP): positive pressure applied during spontaneous breaths; patient controls rate, inspiratory flow, and tidal volume	CPAP is a spontaneous breathing mode used in patients to increase functional residual capacity and improve oxygenation by opening collapsed alveoli at end expiration; it is also used for weaning	Side effects include decreased cardiac output, volutrauma, and increased intracranial pressure No ventilator breaths are delivered in PEEP and CPAP mode unless used with CMV or IMV
Airway pressure release ventilation (APRV): two different levels of CPAP (inspiratory and expiratory) are applied for set periods of time, allowing spontaneous breathing to occur at both levels	APRV is a spontaneous breathing mode used in patients to maintain alveolar recruitment without imposing additional peak inspiratory pressures that could lead to barotrauma	Patient needs to be monitored for hypercapnia
Pressure support ventilation (PSV): preset positive pressure used to augment patient's inspiratory efforts; patient controls rate, inspiratory flow, and tidal volume	PSV is a spontaneous breathing mode used as the primary mode of ventilation in patients with stable respiratory drive to overcome any imposed mechanical resistance (e.g. artificial airway) PSV can also be used with IMV to support spontaneous breaths	Patient should be monitored for hypercapnia Advantages include reduced patient work of breathing and improved patient-ventilator synchrony

Continued

TABLE 20.7 Modes of mechanical ventilation (continued)

Mode of ventilation	Clinical application	Nursing implications
Volume-assured pressure support ventilation (VAPSV) also known as pressure augmentation (PA): a variation of PSV with a set tidal volume to ensure that patient receives minimum tidal volume with each pressure support breath	VAPSV is a spontaneous breathing mode used to treat acute respiratory illness and to facilitate weaning	Advantages include increased patient comfort, decreased work of breathing and decreased respiratory muscle fatigue, and promotion of respiratory muscle conditioning
Independent lung ventilation (ILV): each lung is ventilated separately	ILV is used in patients with unilateral lung disease, bronchopleural fistulas, and bilateral asymmetric lung disease	Requires a double-lumen endotracheal tube, two ventilators, sedation, and/or pharmacologic paralysis
High-frequency ventilation (HFV): delivers a small volume of gas at a rapid rate High-frequency positive-pressure ventilation (HFPPV): delivers 60–100 breaths/min High-frequency jet ventilation (HFJV): delivers 100–600 cycles/min High-frequency oscillation (HFO): delivers 900–3000 cycles/min	HFV is used in situations in which conventional mechanical ventilation compromises haemodynamic stability, with bronchopleural fistulas, during short-term procedures, and with diseases that create a risk of volutrauma	Patients require sedation and/or pharmacologic paralysis Inadequate humidification can compromise airway patency Assessment of breath sounds is difficult
Adapted from Urden LD, Stacy KM, Lough ME. <i>Thelan's critical care nursing: diagnosis and management</i> . 5th edn. St Louis: Mosby; 2006. PEEP, positive end-expiratory pressure		

BOX 20.5 Essential nursing care for mechanical ventilation

Definition: Use of an artificial device to assist a patient to breathe

Activities

- Monitor for respiratory muscle fatigue
- Monitor for impending respiratory failure
- Consult with other healthcare personnel in selection of a ventilator mode
- Initiate setup and application of the ventilator
- Instruct the patient and family about the rationale and expected sensations associated with use of mechanical ventilators
- Routinely monitor ventilator settings
- Monitor for decrease in exhale volume and increase in inspiratory pressure
- Ensure that ventilator alarms are on
- Administer muscle-paralysing agents, sedatives, and narcotic analgesics, as appropriate
- Monitor the effectiveness of mechanical ventilation on patient's physiologic and psychologic status
- Initiate calming techniques, as appropriate
- Provide patient with a means for communication (e.g. paper and pencil or alphabet board)
- Check all ventilator connections regularly
- Empty condensed water from traps, as appropriate
- Use aseptic technique, as appropriate
- Monitor ventilator pressure reading and breath sounds

- Stop nasogastric feedings during suctioning and 30 to 60 min before chest physiotherapy
- Silence ventilator alarms during suctioning to decrease frequency of false alarms
- Monitor patient's progress on current ventilator settings and make appropriate changes as ordered
- Monitor for adverse effects of mechanical ventilation: infection, barotraumas and reduced cardiac output
- Position to facilitate ventilation/perfusion matching ('good lung down'), as appropriate
- Collaborate with physician to use CPAP or PEEP to minimise alveolar hypoventilation, as appropriate
- Perform chest physical therapy, as appropriate
- Perform suctioning, based on presence of adventitious sounds and/or increased ventilatory pressures
- Promote adequate fluid and nutritional intake
- Provide routine oral care
- Monitor effects of ventilator changes on oxygenation: ABG, SaO_2 , SvO_2 , end-tidal CO_2 , \dot{Q}_s/\dot{Q}_T , and $A-aO_2$ levels and patient's subjective response
- Monitor degree of shunt, vital capacity, \dot{V}_I/\dot{V}_E , mandatory minute ventilation (MMV) inspiratory force, and FEV₁ for readiness to wean from mechanical ventilation based on agency protocol

From Dochterman JM, Bulechek GM. *Nursing interventions classification (NIC)*. 4th edn. St Louis: Mosby; 2004.

- gastric distension (CPAP/BiPAP can force air into the stomach).^{3,4,27–30}

Invasive ventilation

The principle aim of ventilation was discussed earlier and is the same for both invasive and non-invasive ventilation.

Invasive ventilation is associated with the need for patient sedation and the use of neuromuscular blocking agents as an endotracheal tube is inserted into the airway. Invasive ventilation is associated with more complications than non-invasive ventilation and is usually required as a life-saving measure. Complications are similar for both forms of ventilatory support and include barotrauma from over

distension of the alveoli, intestinal distension from inflation pressures and nosocomial pneumonia from the ET tube bypassing many of the lungs' normal defence mechanisms. A number of different ventilatory modes are used for a variety of patient conditions; invasive ventilation ensures that the patient receives the minute volume and appropriate gases to meet respiratory needs without damaging the lungs.^{3,4} As there are a number of ventilators available on the market today, it is important that the emergency nurse is familiar with the technology and modes of ventilation prior to caring for a ventilated patient.

CARE OF THE VENTILATED PATIENT IN THE EMERGENCY DEPARTMENT

Emergency nursing management of the patient on a ventilator is outlined in Box 20.5. Routine assessment of a patient on a ventilator includes monitoring the patient for both patient-related and ventilator-related complications. Particular care is required for the pulmonary system, placement of endotracheal tube and when observing for subcutaneous emphysema and synchrony with the ventilator. Assessment of the ventilator includes a review of all the ventilator settings and alarms.^{3,4,31}

The bedside evaluation of the patients' vital capacity, minute ventilation, ABG values, end-tidal CO₂, invasive arterial pressure monitoring and pulse oximetry should be undertaken as per hospital policy. The continuous monitoring, assessment of oxygenation is required and documented contemporaneously. It is imperative that the emergency nurse maintains the patency and placement of the ET tube by regular closed system suctioning and ventilator tubing to avoid kinking, and checking that it is connected to oxygen.^{3,4,7,31}

SUMMARY

Respiratory emergencies are commonplace in any emergency department. With the ageing population and chronic diseases such as COPD, the emergency nurse must maintain sound knowledge and skill and promote a calming environment to reduce patient anxiety when dealing with such an emergency.

Sound knowledge of anatomy and physiology, and the pathophysiology of respiratory disease will enable the emergency nurse to critically assess patient symptoms and identify potential complications associated with a respiratory emergency. The emergency nurse must be able to evaluate treatments and therapeutic interventions and be ever prepared in the event of an emergency if the patient's respiratory condition deteriorates.

REVIEW QUESTIONS

- 1 Describe how we breathe. Include the muscles involved and how long a normal respiratory cycle takes.

- 2 Discuss the difference between ventilation and respiration.
- 3 What are the four ways to assess a patient's respiratory status?
- 4 What are the differences between peak flow and spirometry measurements?
- 5 Discuss what is happening to a patient with a respiratory acidosis.
- 6 What are the underlying diseases described by the terms 'pink puffers' and 'blue bloaters'?
- 7 Discuss the indications for the insertion of an underwater sealed drain.
- 8 Discuss the difference between Type I and Type II respiratory failure.
- 9 What is the difference between CPAP and BiPAP? Discuss the clinical implications of each.
- 10 What does the term 'cricoid pressure' mean?

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