

Recognition of the Deteriorating Patient and Prevention of Cardiorespiratory Arrest

Learning outcomes

To understand:

- ▶ **The importance of early recognition of the deteriorating patient**
- ▶ **The causes of cardiorespiratory arrest in adults**
- ▶ **How to identify and treat patients at risk of cardiorespiratory arrest using the Airway, Breathing, Circulation, Disability, Exposure (ABCDE) approach**

Introduction

Early recognition of the deteriorating patient and prevention of cardiac arrest is the first link in the chain of survival. Once cardiac arrest occurs, fewer than 20% of patients having an in-hospital cardiac arrest will survive to go home. Prevention of in-hospital cardiac arrest requires staff education, monitoring of patients, recognition of patient deterioration, a system to call for help, and an effective response.

Survivors from in-hospital cardiac arrest usually have a witnessed and monitored ventricular fibrillation (VF) arrest, primary myocardial ischaemia as the cause, and receive immediate and successful defibrillation.

Most cardiorespiratory arrests in hospital are not sudden or unpredictable events: in approximately 80% of cases there is deterioration in clinical signs during the few hours before cardiac arrest. These patients often have slow and progressive physiological deterioration, particularly hypoxia and hypotension (i.e. Airway, Breathing, Circulation

problems) that is unnoticed by staff, or is recognised but treated poorly. The cardiac arrest rhythm in this group is usually non-shockable (PEA or asystole) and the survival rate to hospital discharge is very low.

Early recognition and effective treatment of the deteriorating patient might prevent cardiac arrest, death or an unanticipated intensive care unit (ICU) admission. Closer attention to patients who have a 'false' cardiac arrest (i.e. a 'cardiac arrest team' call when the patient has not had a cardiac arrest) may also improve outcome, because up to one third of these patients die during their in-hospital stay. Early recognition will also help to identify individuals for whom cardiorespiratory resuscitation is not appropriate or who do not wish to be resuscitated.

Prevention of in-hospital cardiac arrest: the Chain of Prevention

The Chain of Prevention can assist hospitals in structuring care processes to prevent and detect patient deterioration and cardiac arrest. The five rings of the chain represent: staff education; the monitoring of patients; the recognition of patient deterioration; a system to call for help; and an effective response (Figure 3.1):

- **Education:** how to observe patients; interpretation of observed signs; the recognition of signs of deterioration; and the use of the ABCDE approach and simple skills to stabilise the patient pending the arrival of more experienced help.
- **Monitoring:** patient assessment and the measurement and recording of vital signs, which may include the use of electronic monitoring devices.

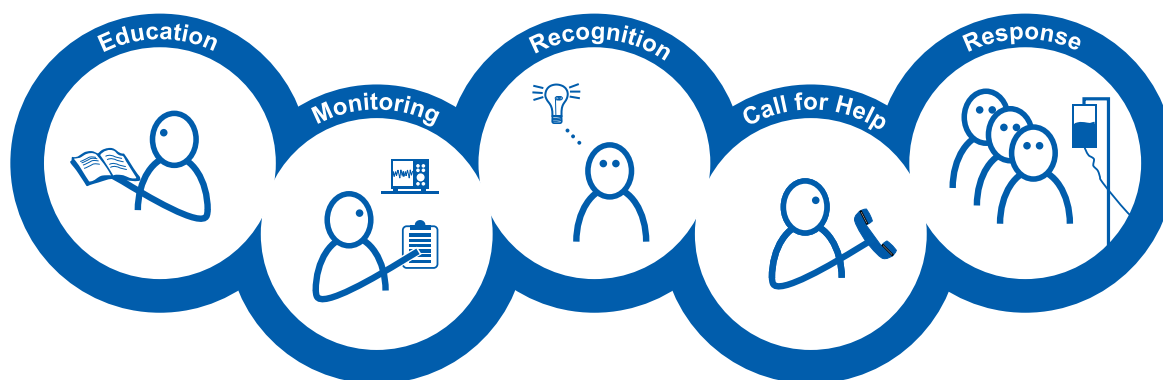


Figure 3.1 Chain of Prevention

Score	3	2	1	0	1	2	3
Pulse (min ⁻¹)		≤ 40	41 - 50	51 - 90	91 - 110	111 - 130	≥ 131
Respiratory rate (min ⁻¹)	≤ 8		9 - 11	12 - 20		21 - 24	≥ 25
Temperature (°C)	≤ 35.0		35.1 - 36.0	36.1 - 38.0	38.1 - 39.0	≥ 39.1	
Systolic BP (mmHg)	≤ 90	91 - 100	101 - 110	111 - 249	≥ 250		
Oxygen saturation (%)	≤ 91	92 - 93	94 - 95	≥ 96			
Inspired oxygen				Air			Any oxygen therapy
AVPU				Alert (A)			Voice (V) Pain (P) Unresponsive (U)

TABLE 3.1 Example of early warning scoring (EWS) system*

* From Prytherch et al. ViEWS - Towards a national early warning score for detecting adult in-patient deterioration. Resuscitation. 2010;81(8):932-7

- **Recognition** encompasses the tools available to identify patients in need of additional monitoring or intervention, including suitably designed vital signs charts and sets of predetermined 'calling criteria' to 'flag' the need to escalate monitoring or to call for more expert help.
- **Call for help** protocols for summoning a response to a deteriorating patient should be universally known and understood, unambiguous and mandated. Doctors and nurses often find it difficult to ask for help or escalate treatment as they feel their clinical judgement may be criticised. Hospitals should ensure all staff are empowered to call for help. A structured communication tool such as SBAR (Situation, Background, Assessment, Recommendation) or RSVP (Reason, Story, Vital signs, Plan) should be used to call for help.
- **Response** to a deteriorating patient must be assured, of specified speed and by staff with appropriate acute or critical care skills, and experience.

observations, or the total EWS, indicates the level of intervention required, e.g. increased frequency of vital signs monitoring, or calling ward doctors or resuscitation teams to the patient. An example of an EWS system is shown in Table 3.1.

Early warning scores are dynamic and change over time and the frequency of observations should be increased to track improvement or deterioration in a patient's condition. If it is clear a patient is deteriorating help should be called for early rather than waiting for the patient to reach a specific score.

The patient's EWS is calculated based on Table 3.1. An increased score indicates an increased risk of deterioration and death. There should be a graded response to scores according to local hospital protocols (Table 3.2).

Alternatively, systems incorporating calling criteria are based on routine observations, which activate a response when one or more variables reach an extremely abnormal value. It is not clear which of these two systems is better. Some hospitals combine elements of both systems.

Even when doctors are alerted to a patient's abnormal physiology, there is often delay in attending to the patient or referring to higher levels of care.

Response to critical illness

The traditional response to cardiac arrest is reactive: the name 'cardiac arrest team' implies that it will be called only after cardiac arrest has occurred. In some hospitals the cardiac arrest team has been replaced by other resuscitation teams (e.g. rapid response team, critical care outreach team, medical emergency team). These teams can be activated according to the patient's EWS (see above) or according to specific calling criteria. For example, the medical emergency team (MET) responds

Recognising the deteriorating patient

In general, the clinical signs of critical illness are similar whatever the underlying process because they reflect failing respiratory, cardiovascular, and neurological systems i.e. ABCDE problems (see below). Abnormal physiology is common on general wards, yet the measurement and recording of important physiological observations of acutely ill patients occurs less frequently than is desirable. The assessment of very simple vital signs, such as respiratory rate, may help to predict cardiorespiratory arrest. To help early detection of critical illness, many hospitals use early warning scores (EWS) or calling criteria. Early warning scoring systems allocate points to measurements of routine vital signs on the basis of their derangement from an arbitrarily agreed 'normal' range. The weighted score of one or more vital sign

EWS	Minimal observation frequency	Escalation	
		Recorder's action	Doctor's action
3 - 5	4 hourly	Inform nurse in charge	
6	4 hourly	Inform doctor	Doctor to see within 1 h
7 - 8	1 hourly	Inform doctor Consider continuous monitoring	Doctor to see within 30 min and discuss with senior doctor and/or outreach team
≥9	30 min	Inform doctor Start continuous monitoring	Doctor to see within 15 min and discuss with senior doctor and ICU team

TABLE 3.2 Example escalation protocol based on early warning score (EWS)

not only to patients in cardiac arrest, but also to those with acute physiological deterioration. The MET usually comprises medical and nursing staff from intensive care and general medicine and responds to specific calling criteria (Table 3.3). Any member of the healthcare team can initiate a MET call. Early involvement of the MET may reduce cardiac arrests, deaths and unanticipated ICU admissions, and may facilitate decisions about limitation of treatment (e.g. do-not-attempt-resuscitation [DNAR] decisions). Medical emergency team interventions often involve simple tasks such as starting oxygen therapy and intravenous fluids. The benefits of the MET system remain to be proved.

MET calling criteria	
Airway	Threatened
Breathing	All respiratory arrests Respiratory rate < 5 min ⁻¹ Respiratory rate > 36 min ⁻¹
Circulation	All cardiac arrests Pulse rate < 40 min ⁻¹ Pulse rate > 140 min ⁻¹ Systolic BP < 90 mmHg
Neurology	Sudden decrease in level of consciousness Decrease in GCS of > 2 points Repeated or prolonged seizures
Other	Any patient causing concern who does not fit the above criteria

TABLE 3.3 Medical emergency team (MET) calling criteria

In the UK, a system of pre-emptive ward care known as critical care outreach, has developed. Outreach services

exist in many forms ranging from a single nurse to a 24-hour, seven days per week multiprofessional team. An outreach team or system may reduce ward deaths, postoperative adverse events, ICU admissions and readmissions, and increase survival.

All critically ill patients should be admitted to an area that can provide the greatest supervision and the highest level of organ support and nursing care. This is usually in a critical care area, e.g. ICU, high dependency unit (HDU), or resuscitation room. These areas should be staffed by doctors and nurses experienced in advanced resuscitation and critical care skills.

Hospital staffing tends to be at its lowest during the night and at weekends. This influences patient monitoring, treatment and outcomes. Admission to general wards in the evening, or to hospital at weekends, is associated with increased mortality. Studies have shown that in-hospital cardiac arrests occurring in the late afternoon, at night or at weekends are more often non-witnessed and have a lower survival rate. Patients discharged at night from ICUs to general wards have an increased risk of ICU readmission and in-hospital death compared with those discharged during the day and those discharged to HDUs.

Causes of deterioration and cardiorespiratory arrest

Deterioration and cardiorespiratory arrest can be caused by primary airway and/or breathing and/or cardiovascular problems.

Airway obstruction

For a detailed review of airway management see Chapter 7.

Causes

Airway obstruction can be complete or partial. Complete airway obstruction rapidly causes cardiac arrest. Partial

obstruction often precedes complete obstruction. Partial airway obstruction can cause cerebral or pulmonary oedema, exhaustion, secondary apnoea, and hypoxic brain injury, and eventually cardiac arrest.

Causes of airway obstruction

- Central nervous system depression
- Blood
- Vomitus
- Foreign body (e.g. tooth, food)
- Direct trauma to face or throat
- Epiglottitis
- Pharyngeal swelling (e.g. infection, oedema)
- Laryngospasm
- Bronchospasm – causes narrowing of the small airways in the lung
- Bronchial secretions
- Blocked tracheostomy

Central nervous system depression may cause loss of airway patency and protective reflexes. Causes include head injury and intracerebral disease, hypercarbia, the depressant effect of metabolic disorders (e.g. diabetes mellitus), and drugs, including alcohol, opioids and general anaesthetic agents. Laryngospasm can occur with upper airway stimulation in a semi-conscious patient whose airway reflexes remain intact.

In some people, the upper airway can become obstructed when they sleep (obstructive sleep apnoea). This is more common in obese patients and obstruction can be worsened in the presence of other factors (e.g. sedative drugs).

Recognition

Assess the patency of the airway in anyone at risk of obstruction. A conscious patient will complain of difficulty in breathing, may be choking, and will be distressed. With partial airway obstruction, efforts at breathing will be noisy. Complete airway obstruction is silent and there is no air movement at the patient's mouth. Any respiratory movements are usually strenuous. The accessory muscles of respiration will be involved, causing a 'see-saw' or 'rocking-horse' pattern of chest and abdominal movement: the chest is drawn in and the abdomen expands on inspiration, and the opposite occurs on expiration.

Treatment

The priority is to ensure that the airway remains patent. Treat any problem that places the airway at risk; for example, suck blood and gastric contents from the airway and, unless contraindicated, turn the patient on their side. Give oxygen as soon as possible to achieve an arterial blood oxygen saturation by pulse oximetry (SpO₂) in the range of 94 - 98%. Assume actual or impending airway obstruction in anyone with a depressed level of

consciousness, regardless of cause. Take steps to safeguard the airway and prevent further complications such as aspiration of gastric contents. This may involve nursing the patient on their side or with a head-up tilt. Simple airway opening manoeuvres (head tilt/chin lift or jaw thrust), insertion of an oropharyngeal or nasal airway, elective tracheal intubation or tracheostomy may be required. Consider insertion of a nasogastric tube to empty the stomach.

Breathing problems

Causes

Breathing inadequacy may be acute or chronic. It may be continuous or intermittent, and severe enough to cause apnoea (respiratory arrest), which will rapidly cause cardiac arrest. Respiratory arrest often occurs because of a combination of factors; for example, in a patient with chronic respiratory inadequacy, a chest infection, muscle weakness, or fractured ribs can lead to exhaustion, further depressing respiratory function. If breathing is insufficient to oxygenate the blood adequately (hypoxaemia), a cardiac arrest will occur eventually.

Respiratory drive

Central nervous system depression may decrease or abolish respiratory drive. The causes are the same as those for airway obstruction from central nervous system depression.

Respiratory effort

The main respiratory muscles are the diaphragm and intercostal muscles. The latter are innervated at the level of their respective ribs and may be paralysed by a spinal cord lesion above this level. The innervation of the diaphragm is at the level of the third, fourth and fifth segment of the spinal cord. Spontaneous breathing cannot occur with severe cervical cord damage above this level.

Inadequate respiratory effort, caused by muscle weakness or nerve damage, occurs with many diseases (e.g. myasthenia gravis, Guillain-Barré syndrome, and multiple sclerosis). Chronic malnourishment and severe long-term illness may also contribute to generalised weakness.

Breathing can also be impaired with restrictive chest wall abnormalities such as kyphoscoliosis. Pain from fractured ribs or sternum will prevent deep breaths and coughing.

Lung disorders

Lung function is impaired by a pneumothorax or haemothorax. A tension pneumothorax causes a rapid failure of gas exchange, a reduction of venous return to the heart, and a fall in cardiac output. Severe lung disease will impair gas exchange. Causes include infection, aspiration, exacerbation of chronic obstructive pulmonary disease (COPD), asthma, pulmonary embolus, lung contusion, acute respiratory distress syndrome (ARDS) and pulmonary oedema.

Recognition

A conscious patient will complain of shortness of breath and be distressed. The history and examination will usually indicate the underlying cause. Hypoxaemia and hypercarbia can cause irritability, confusion, lethargy and a decrease in the level of consciousness. Cyanosis may be visible but is a late sign. A fast respiratory rate ($>25 \text{ min}^{-1}$) is a useful, simple indicator of breathing problems. Pulse oximetry is an easy, non-invasive measure of the adequacy of oxygenation (see Chapter 15). However, it is not a reliable indicator of ventilation and an arterial blood gas sample is necessary to obtain values for arterial carbon dioxide tension (PaCO_2) and pH. A rising PaCO_2 and a decrease in pH are often late signs in a patient with severe respiratory problems.

Treatment

Give oxygen to all acutely ill hypoxaemic patients and treat the underlying cause. Give oxygen at 15 l min^{-1} using a high-concentration reservoir mask. Once the patient is stable, change the oxygen mask and aim for a SpO_2 in the range of 94 - 98%. For example, suspect a tension pneumothorax from a history of chest trauma and confirm by clinical signs and symptoms. If diagnosed, decompress it immediately by inserting a large-bore (14 G) cannula into the second intercostal space, in the mid-clavicular line (needle thoracocentesis).

Patients who are having difficulty breathing or are becoming tired will need respiratory support. Non-invasive ventilation using a face mask or a helmet can be useful and prevent the need for tracheal intubation and ventilation. For patients who cannot breathe adequately, sedation, tracheal intubation and controlled ventilation is needed.

Circulation problems

Causes

Circulation problems may be caused by primary heart disease or by heart abnormalities secondary to other problems. Most often, circulation problems in acutely ill patients are due to hypovolaemia. The heart may stop suddenly or may produce an inadequate cardiac output for a period of time before stopping.

Primary heart problems

The commonest cause of sudden cardiac arrest is an arrhythmia caused by either ischaemia or myocardial infarction. Cardiac arrest can also be caused by an arrhythmia due to other forms of heart disease, by heart block, electrocution and some drugs.

Sudden cardiac arrest may also occur with cardiac failure, cardiac tamponade, cardiac rupture, myocarditis and hypertrophic cardiomyopathy.

Causes of ventricular fibrillation

- Acute coronary syndromes (Chapter 4)
- Hypertensive heart disease
- Valve disease
- Drugs (e.g. antiarrhythmic drugs, tricyclic antidepressants, digoxin)
- Inherited cardiac diseases (e.g. long QT syndromes)
- Acidosis
- Abnormal electrolyte concentration (e.g. potassium, magnesium, calcium)
- Hypothermia
- Electrocution

Secondary heart problems

The heart is affected by changes elsewhere in the body. For example, cardiac arrest will occur rapidly following asphyxia from airway obstruction or apnoea, tension pneumothorax, or acute severe blood loss. Severe hypoxia and anaemia, hypothermia, oligoemia and severe septic shock will also impair cardiac function and this may lead to cardiac arrest.

Recognition

The signs and symptoms of cardiac disease include chest pain, shortness of breath, syncope, tachycardia, bradycardia, tachypnoea, hypotension, poor peripheral perfusion (prolonged capillary refill time), altered mental state, and oliguria.

Most sudden cardiac deaths (SCDs) occur in people with pre-existing cardiac disease, which may have been unrecognised previously. Although the risk is greater for patients with known severe cardiac disease, most SCDs occur in people with unrecognised disease. Asymptomatic or silent cardiac disease may include hypertensive heart disease, aortic valve disease, cardiomyopathy, myocarditis, and coronary disease.

Recognition of risk of sudden cardiac death out of hospital

Coronary artery disease is the commonest cause of SCD. Non-ischaemic cardiomyopathy and valvular disease account for some other SCD events. A small percentage of SCDs are caused by inherited abnormalities (e.g. long and short QT syndromes, Brugada syndrome, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy), and by congenital heart disease.

In patients with a known diagnosis of cardiac disease, syncope (with or without prodrome - particularly recent or recurrent) is as an independent risk factor for increased risk of death. Apparently healthy children and young adults who have SCD may also have symptoms and signs (e.g. syncope/pre-syncope, chest pain, palpitation, heart murmur)

that should alert healthcare professionals to seek expert help to prevent cardiac arrest. Features that indicate a high probability of arrhythmic syncope include:

- syncope in the supine position;
- syncope occurring during or after exercise (although syncope after exercise is often vasovagal);
- syncope with no or only brief prodromal symptoms;
- repeated episodes of unexplained syncope;
- syncope in individuals with a family history of sudden death or inherited cardiac condition.

Assessment in a clinic specialising in the care of those at risk for SCD is recommended in family members of young victims of SCD or those with a known cardiac disorder resulting in an increased risk of SCD. Specific and detailed guidance for the care of individuals with transient loss of consciousness is available (<http://guidance.nice.org.uk/CG109>).

Treatment

Treat the underlying cause of circulatory failure. In many sick patients, this means giving intravenous fluids to treat hypovolaemia. Assess patients with chest pain for an acute coronary syndrome (ACS). A comprehensive description of the management of ACS is given in Chapter 4.

Most patients with cardiac ischaemic pain will be more comfortable sitting up. In some instances lying flat may provoke or worsen the pain. Consider using an anti-emetic, especially if nausea is present.

Survivors of an episode of VF are likely to have a further episode unless preventative treatment is given. These patients may need percutaneous coronary intervention, coronary artery bypass grafting, or an implantable defibrillator.

Treating the underlying cause should prevent many secondary cardiac arrests; for example, early goal-directed therapy to optimise vital organ perfusion decreases the risk of death in severe sepsis. Cardiovascular support includes correction of underlying electrolyte or acid-base disturbances, and treatment to achieve a desirable cardiac rate, rhythm and output. Advanced cardiovascular monitoring and echocardiography may be indicated. Appropriate manipulation of cardiac filling may require fluid therapy and vasoactive drugs. Inotropic drugs and vasoconstrictors may be indicated to support cardiac output and blood pressure. In some situations, mechanical circulatory support (e.g. intra-aortic balloon pump) or consideration of heart transplantation will be necessary.

The ABCDE approach

Underlying principles

The approach to all deteriorating or critically ill patients is the same. The underlying principles are:

1. Use the **A**irway, **B**reathing, **C**irculation, **D**isability, **E**xposure approach to assess and treat the patient.
2. Do a complete initial assessment and re-assess regularly.
3. Treat life-threatening problems before moving to the next part of assessment.
4. Assess the effects of treatment.
5. Recognise when you will need extra help. Call for appropriate help early.
6. Use all members of the team. This enables interventions, e.g. assessment, attaching monitors, intravenous access, to be undertaken simultaneously.
7. Communicate effectively - use the SBAR or RSVP approach (see Chapter 2).
8. The aim of the initial treatment is to keep the patient alive, and achieve some clinical improvement. This will buy time for further treatment and making a diagnosis.
9. Remember - it can take a few minutes for treatments to work.

First steps

1. Ensure personal safety. Wear apron and gloves as appropriate.
2. First look at the patient in general to see if the patient appears unwell.
3. If the patient is awake, ask "How are you?". If the patient appears unconscious or has collapsed, shake him and ask "Are you alright?" If he responds normally he has a patent airway, is breathing and has brain perfusion. If he speaks only in short sentences, he may have breathing problems. Failure of the patient to respond is a clear marker of critical illness.
4. This first rapid 'Look, Listen and Feel' of the patient should take about 30 s and will often indicate a patient is critically ill and there is a need for urgent help. Ask a colleague to ensure appropriate help is coming.

5. If the patient is unconscious, unresponsive, and is not breathing normally (occasional gasps are not normal) start CPR according to the guidance in Chapter 5. If you are confident and trained to do so, feel for a pulse to determine if the patient has a respiratory arrest. If there are any doubts about the presence of a pulse start CPR.
6. Monitor the vital signs early. Attach a pulse oximeter, ECG monitor and a non-invasive blood pressure monitor to **all** critically ill patients, as soon as possible.
7. Insert an intravenous cannula as soon as possible. Take bloods for investigation when inserting the intravenous cannula.

Airway (A)

Airway obstruction is an emergency. Get expert help immediately. Untreated, airway obstruction causes hypoxia and risks damage to the brain, kidneys and heart, cardiac arrest, and death.

1. Look for the signs of airway obstruction:
 - Airway obstruction causes paradoxical chest and abdominal movements ('see-saw' respirations) and the use of the accessory muscles of respiration. Central cyanosis is a late sign of airway obstruction. In complete airway obstruction, there are no breath sounds at the mouth or nose. In partial obstruction, air entry is diminished and often noisy.
 - In the critically ill patient, depressed consciousness often leads to airway obstruction.
2. Treat airway obstruction as a medical emergency:
 - Obtain expert help immediately. Untreated, airway obstruction causes hypoxaemia (low PaO₂) with the risk of hypoxic injury to the brain, kidneys and heart, cardiac arrest, and even death.
 - In most cases, only simple methods of airway clearance are required (e.g. airway opening manoeuvres, airways suction, insertion of an oropharyngeal or nasopharyngeal airway). Tracheal intubation may be required when these fail.
3. Give oxygen at high concentration:
 - Provide high-concentration oxygen using a mask with an oxygen reservoir. Ensure that the oxygen flow is sufficient (usually 15 l min⁻¹) to prevent collapse of the reservoir during inspiration. If the patient's trachea is intubated, give high concentration oxygen with a self-inflating bag.
 - In acute respiratory failure, aim to maintain an oxygen saturation of 94 - 98%. In patients at risk of hypercapnic respiratory failure (see below) aim for an oxygen saturation of 88 - 92%.

Breathing (B)

During the immediate assessment of breathing, it is vital to diagnose and treat immediately life-threatening conditions, e.g. acute severe asthma, pulmonary oedema, tension pneumothorax, and massive haemothorax.

1. Look, listen and feel for the general signs of respiratory distress: sweating, central cyanosis, use of the accessory muscles of respiration, and abdominal breathing.
2. Count the respiratory rate. The normal rate is 12 - 20 breaths min⁻¹. A high (≥ 25 min⁻¹), or increasing, respiratory rate is a marker of illness and a warning that the patient may deteriorate suddenly.
3. Assess the depth of each breath, the pattern (rhythm) of respiration and whether chest expansion is equal on both sides.
4. Note any chest deformity (this may increase the risk of deterioration in the ability to breathe normally); look for a raised jugular venous pulse (JVP) (e.g. in acute severe asthma or a tension pneumothorax); note the presence and patency of any chest drains; remember that abdominal distension may limit diaphragmatic movement, thereby worsening respiratory distress.
5. Record the inspired oxygen concentration (%) and the SpO₂ reading of the pulse oximeter. The pulse oximeter does not detect hypercapnia. If the patient is receiving supplemental oxygen, the SpO₂ may be normal in the presence of a very high PaCO₂.
6. Listen to the patient's breath sounds a short distance from his face: rattling airway noises indicate the presence of airway secretions, usually caused by the inability of the patient to cough sufficiently or to take a deep breath. Stridor or wheeze suggests partial, but significant, airway obstruction.
7. Percuss the chest: hyper-resonance may suggest a pneumothorax; dullness usually indicates consolidation or pleural fluid.
8. Auscultate the chest: bronchial breathing indicates lung consolidation with patent airways; absent or reduced sounds suggest a pneumothorax or pleural fluid or lung consolidation caused by complete bronchial obstruction.
9. Check the position of the trachea in the suprasternal notch: deviation to one side indicates mediastinal shift (e.g. pneumothorax, lung fibrosis or pleural fluid).
10. Feel the chest wall to detect surgical emphysema or crepitus (suggesting a pneumothorax until proven otherwise).
11. The specific treatment of respiratory disorders depends upon the cause. Nevertheless, all critically

ill patients should be given oxygen. In a subgroup of patients with chronic obstructive pulmonary disease (COPD), high concentrations of oxygen may depress breathing (i.e. they are at risk of hypercapnic respiratory failure - often referred to as type 2 respiratory failure). Nevertheless, these patients will also sustain end-organ damage or cardiac arrest if their blood oxygen tensions are allowed to decrease. In this group, aim for a lower than normal PaO₂ and oxygen saturation. Give oxygen via a Venturi 28% mask (4 l min⁻¹) or a 24% Venturi mask (4 l min⁻¹) initially and reassess. Aim for target SpO₂ range of 88 - 92% in most COPD patients, but evaluate the target for each patient based on the patient's arterial blood gas measurements during previous exacerbations (if available). Some patients with chronic lung disease carry an oxygen alert card (that documents their target saturation) and their own appropriate Venturi mask.

12. If the patient's depth or rate of breathing is judged to be inadequate, or absent, use bag-mask or pocket mask ventilation to improve oxygenation and ventilation, whilst calling immediately for expert help. In cooperative patients who do not have airway obstruction consider the use of non-invasive ventilation (NIV). In patients with an acute exacerbation of COPD, the use of NIV is often helpful and prevents the need for tracheal intubation and invasive ventilation.

Circulation (C)

In almost all medical and surgical emergencies, consider hypovolaemia to be the primary cause of shock, until proven otherwise. Unless there are obvious signs of a cardiac cause, give intravenous fluid to any patient with cool peripheries and a fast heart rate. In surgical patients, rapidly exclude haemorrhage (overt or hidden). Remember that breathing problems, such as a tension pneumothorax, can also compromise a patient's circulatory state. This should have been treated earlier on in the assessment.

1. Look at the colour of the hands and digits: are they blue, pink, pale or mottled?
2. Assess the limb temperature by feeling the patient's hands: are they cool or warm?
3. Measure the capillary refill time (CRT). Apply cutaneous pressure for 5 s on a fingertip held at heart level (or just above) with enough pressure to cause blanching. Time how long it takes for the skin to return to the colour of the surrounding skin after releasing the pressure. The normal value for CRT is usually < 2 s. A prolonged CRT suggests poor peripheral perfusion. Other factors (e.g. cold surroundings, poor lighting, old age) can prolong CRT.
4. Assess the state of the veins: they may be under-filled or collapsed when hypovolaemia is present.

5. Count the patient's pulse rate (or preferably heart rate by listening to the heart with a stethoscope).
6. Palpate peripheral and central pulses, assessing for presence, rate, quality, regularity and equality. Barely palpable central pulses suggest a poor cardiac output, whilst a bounding pulse may indicate sepsis.
7. Measure the patient's blood pressure. Even in shock, the blood pressure may be normal, because compensatory mechanisms increase peripheral resistance in response to reduced cardiac output. A low diastolic blood pressure suggests arterial vasodilation (as in anaphylaxis or sepsis). A narrowed pulse pressure (difference between systolic and diastolic pressures; normally 35 - 45 mmHg) suggests arterial vasoconstriction (cardiogenic shock or hypovolaemia) and may occur with rapid tachyarrhythmia.
8. Auscultate the heart. Is there a murmur or pericardial rub? Are the heart sounds difficult to hear? Does the audible heart rate correspond to the pulse rate?
9. Look for other signs of a poor cardiac output, such as reduced conscious level and, if the patient has a urinary catheter, oliguria (urine volume < 0.5 ml kg⁻¹ h⁻¹).
10. Look thoroughly for external haemorrhage from wounds or drains or evidence of concealed haemorrhage (e.g. thoracic, intra-peritoneal, retroperitoneal or into gut). Intra-thoracic, intra-abdominal or pelvic blood loss may be significant, even if drains are empty.
11. The specific treatment of cardiovascular collapse depends on the cause, but should be directed at fluid replacement, haemorrhage control and restoration of tissue perfusion. Seek the signs of conditions that are immediately life threatening, e.g. cardiac tamponade, massive or continuing haemorrhage, septicaemic shock, and treat them urgently.
12. Insert one or more large (14 or 16 G) intravenous cannulae. Use short, wide-bore cannulae, because they enable the highest flow.
13. Take blood from the cannula for routine haematological, biochemical, coagulation and microbiological investigations, and cross-matching, before infusing intravenous fluid.
14. Give a rapid fluid challenge (over 5 - 10 min) of 500 ml of warmed crystalloid solution (e.g. Hartmann's solution or 0.9% sodium chloride) if the patient is normotensive. Give one litre, if the patient is hypotensive. Use smaller volumes (e.g. 250 ml) for patients with known cardiac failure or trauma and use closer monitoring (listen to the chest for crackles after each bolus, consider a CVP line).

15. Reassess the heart rate and BP regularly (every 5 min), aiming for the patient's normal BP or, if this is unknown, a target > 100 mmHg systolic.
16. If the patient does not improve, repeat the fluid challenge.
17. If symptoms and signs of cardiac failure (dyspnoea, increased heart rate, raised JVP, a third heart sound and pulmonary crackles on auscultation) occur, decrease the fluid infusion rate or stop the fluids altogether. Seek alternative means of improving tissue perfusion (e.g. inotropes or vasopressors).
18. If the patient has primary chest pain and a suspected ACS, record a 12-lead ECG early, and treat initially with aspirin, nitroglycerine, oxygen, and morphine.
19. Immediate general treatment for ACS includes:
 - Aspirin 300 mg, orally, crushed or chewed, as soon as possible.
 - Nitroglycerine, as sublingual glyceryl trinitrate (tablet or spray).
 - Oxygen, aiming at a SpO₂ of 94 - 98%; do not give supplementary oxygen if the patient's SpO₂ is within this range when breathing air alone.
 - Morphine (or diamorphine) titrated intravenously to avoid sedation and respiratory depression.

Disability (D)

Common causes of unconsciousness include profound hypoxia, hypercapnia, cerebral hypoperfusion, or the recent administration of sedatives or analgesic drugs.

1. Review and treat the ABCs: exclude or treat hypoxia and hypotension.
2. Check the patient's drug chart for reversible drug-induced causes of depressed consciousness. Give an antagonist where appropriate (e.g. naloxone for opioid toxicity).
3. Examine the pupils (size, equality and reaction to light).
4. Make a rapid initial assessment of the patient's conscious level using the AVPU method: **A**lert, responds to **V**ocal stimuli, responds to **P**ainful stimuli or **U**nresponsive to all stimuli. Alternatively, use the Glasgow Coma Scale score.
5. Measure the blood glucose to exclude hypoglycaemia using a rapid finger-prick bedside testing method. If the blood sugar is below 4.0 mmol l⁻¹, give an initial dose of 50 ml of 10% glucose solution intravenously. If necessary, give further doses of intravenous 10% glucose every minute until the patient has fully regained consciousness, or a total of 250 ml of 10%

glucose has been given. Repeat blood glucose measurements to monitor the effects of treatment. If there is no improvement consider further doses of 10% glucose.

6. Nurse unconscious patients in the lateral position if their airway is not protected.

Exposure (E)

To examine the patient properly full exposure of the body may be necessary. Respect the patient's dignity and minimise heat loss.

Additional information

1. Take a full clinical history from the patient, any relatives or friends, and other staff.
2. Review the patient's notes and charts:
 - Study both absolute and trended values of vital signs.
 - Check that important routine medications are prescribed and being given.
3. Review the results of laboratory or radiological investigations.
4. Consider which level of care is required by the patient (e.g. ward, HDU, ICU).
5. Make complete entries in the patient's notes of your findings, assessment and treatment. Where necessary, hand over the patient to your colleagues.
6. Record the patient's response to therapy.
7. Consider definitive treatment of the patient's underlying condition.

Key learning points

- Most patients who have an in-hospital cardiac arrest have warning signs and symptoms before the arrest.
- Early recognition and treatment of the deteriorating patient will prevent some cardiorespiratory arrests.
- Use strategies such as early warning scoring (EWS) systems to identify patients at risk of cardiorespiratory arrest.
- Airway, breathing and circulation problems can cause cardiorespiratory arrest.
- Use the ABCDE approach to assess and treat critically ill patients.

Further reading

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