

# CHAPTER 18

## Resuscitation

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### Learning objectives

After reading this chapter, you should be able to:

- identify the clinical assessment used to identify sudden cardiac arrest (SCA)
- outline the role of the chain of survival in the management of SCA
- differentiate between monophasic and biphasic defibrillators
- outline management of common arrhythmias associated with SCA
- describe the use of advanced airway adjuncts and indications for use in SCA

- discuss indications/actions/routes of administration of medications used in advanced life support
- describe appropriate care of persons experiencing SCA including specific circumstances such as the pregnant woman, electrical injuries and drowning
- discuss current research in resuscitation.

### Key words

resuscitation  
cardiopulmonary resuscitation  
advanced life support

## INTRODUCTION

The continuum of critical illness for an individual can span the period before and beyond hospital admission. Resuscitation is often required outside the critical care environment, and the 'cardiac arrest' team has evolved to use a more proactive early intervention approach, utilising a range of systems and instruments to detect deterioration in patients' clinical status (see Chapter 5). It is well recognised that improved outcomes from cardiac arrest are dependent on early recognition and initiation of the 'chain of survival'. This chapter introduces the resuscitation systems and processes

in both the prehospital and the in-hospital settings. The chain of survival provides a framework for the management of the person experiencing cardiac arrest and resuscitation in specific circumstances. The chapter expands on the final link in the chain, advanced life support, to outline advanced airway management, rhythm recognition and administration of medications. Resuscitation involves many moral and ethical issues, such as family presence during resuscitation, deciding when to cease or initiate resuscitation, and near-death experiences.

## BACKGROUND

Coronary heart disease (CHD) is the leading cause of death in most industrialised countries, with over half of these being due to sudden cardiac arrest (SCA).<sup>1</sup> Despite advances in the management of CHD, survival outcome figures from SCA remain poor.<sup>2</sup> Survival after SCA is dependent on the presenting rhythm, early defibrillation, effective cardiopulmonary resuscitation and advanced life support.<sup>1</sup> Because the presenting rhythm with the majority of witnessed SCAs is ventricular fibrillation, bystander cardiopulmonary resuscitation and early defibrillation are the major interventions influencing outcome after SCA.<sup>1,3</sup> It is possible that the number of ventricular fibrillation/ventricular tachycardia (VF/VT) arrests is actually higher than reported, as often by the time the cardiac arrest team arrives the patient's rhythm has deteriorated to asystole.<sup>8</sup>

### Incidence/aetiology of cardiac arrests

In Australia in 2001–02, 36% of all hospitalisations for cardiovascular disease and 26,063 deaths were attributed to CHD.<sup>4</sup> As the prevalence of CHD varies worldwide, estimates of the incidence of SCA are difficult to obtain.<sup>5</sup> In adults, the commonest cause of cardiac arrest is a primary cardiac event, with coronary artery disease accounting for up to 90% of all victims.<sup>1,6</sup> Causes of cardiac arrest may be separated into two categories, primary and secondary.<sup>7</sup> These are displayed in Table 18.1.

While causes of cardiac arrest are numerous, most often it is associated with ventricular fibrillation triggered by an acutely ischaemic or infarcted myocardium or primary electrical disturbance.<sup>1,8</sup> Acute myocardial infarction (AMI) is the commonest precursor to cardiac arrest.<sup>9</sup> In victims of trauma, drug overdose and drowning, the predominant cause of cardiac arrest is asphyxia.<sup>8</sup> Similarly, cardiac arrest in children is rare and even more rarely sudden,<sup>10</sup> with the common causes hypoxia and circulatory failure. The commonest arrhythmia in infants is bradycardia, and the prognosis is especially poor if asystole is present.<sup>10</sup>

## PATHOPHYSIOLOGY

In sudden cardiac arrest of cardiac origin it is believed that myocardial ischaemia leads to ventricular irritability and the progression from ventricular tachycardia to ventricular fibrillation (VF) and ultimately asystole.<sup>11</sup> After the onset of VF (in animal studies), carotid arterial blood flow continues for approximately 4 minutes even in the absence of cardiac compressions, as coronary perfusion pressure (the pressure gradient between the aorta and the right atrium) falls over this period.<sup>11</sup> This initial phase is characterised by minimal ischaemic injury, and it is during this phase that defibrillation is most likely to result in the restoration of a perfusing rhythm, while initiation of effective cardiac compressions will increase the coronary perfusion pressure.<sup>11</sup>

TABLE 18.1 Causes of cardiac arrest

| Primary causes                                | Secondary causes       |
|---|------------------------|
| Acute myocardial infarction                   | Cessation of breathing |
| Cardiomyopathy                                | Airway obstruction     |
| Electrical shock (low- and high-voltage)      | Severe bleeding        |
| Congenital heart disease (e.g. prolonged Q-T) | Hypothermia            |
| Drugs   | Metabolic disturbance  |
|   | Electrical disturbance |
|   | Trauma                 |
|   | Neuromuscular disease  |

Progression of the cardiac arrest beyond 4 minutes results in accumulation of toxic metabolites, depletion of high-energy phosphate stores, and the initiation of ischaemic cascades.<sup>11</sup> A high probability of irreversible cellular injury exists where a cardiac arrest extends for longer than 10 minutes, and the return of a spontaneous circulation during this period may initiate a reperfusion injury<sup>11</sup> (see Chapter 10 for further discussion).

## RESUSCITATION SYSTEMS AND PROCESSES

Since the rediscovery of the effectiveness of closed-chest cardiopulmonary resuscitation (CPR) in 1960 and its subsequent widespread adoption, CPR has saved the lives of many, potentially ensuring years of productive life.<sup>12–14</sup> As CPR quickly became one of the most widely used and researched procedures, voluntary coordinating bodies developed throughout the world.<sup>13</sup> Organisations such as the European Resuscitation Council (ERC), the American Heart Association (AHA), the New Zealand Resuscitation Council (NRC), the Heart and Stroke Foundation of Canada, and the Southern African and Australian Resuscitation Councils established practice guidelines to improve standards in resuscitation, and coordinated resuscitation activities nationally.<sup>13,15</sup> However, as standardised recording of outcome data did not exist, resuscitation endeavours could not be compared meaningfully between countries.<sup>13</sup> Consequently, the International Liaison Committee on Resuscitation (ILCOR) was formed in 1992 to promote global discussion and consistency of guidelines between these international resuscitation councils.<sup>13</sup> The AHA, Australian Resuscitation Council (ARC), European Resuscitation Council and ILCOR guidelines are subject to constant review and modification based on emerging scientific data.<sup>8</sup> Guidelines and recommendations are classified according to scientific evidence. The most recent substantive guidelines from ILCOR, European Resuscitation Council, were published December 2005,<sup>8</sup> with the Australian Resuscitation Council guidelines being published in March 2006.<sup>7</sup> While it is recognised there are differences between the various councils, this chapter primarily reports on the Australian Resuscitation Council recommendations. The ACCCN

position statements on adult and paediatric resuscitation can be found in Appendix B4.

## Prehospital systems

### SURVIVAL OF OUT-OF-HOSPITAL ARRESTS

Despite recent advances in resuscitation and technology, the survival rate for out-of-hospital cardiac arrest (OHCA) remains poor.<sup>16</sup> Factors associated with 100% mortality for adults were: age over 80 years, unwitnessed arrest, defibrillation response times longer than 8 minutes, and non-ventricular tachycardia/fibrillation rhythm.<sup>17</sup> The outcome statistics for children after OHCA are similarly poor.<sup>10</sup> Marked differences in the inclusion criteria and outcome definitions may, however, also explain the wide variations in survival rates from cardiac arrests.<sup>13</sup> In recognition of these variations, the Utstein guidelines were developed and implemented to consistently document, monitor and compare out-of-hospital cardiac arrests. These guidelines:<sup>1</sup>

- establish uniform terms and definitions for out-of-hospital resuscitation;
- establish a reporting template for resuscitation studies to ensure comparability;
- define time points and time intervals relating to cardiac resuscitation;
- define clinical items and outcomes that emergency medical systems should gather; and
- develop methods for describing resuscitation systems.

## In-hospital systems

### SURVIVAL FROM IN-HOSPITAL ARRESTS

The survival rate after out-of-hospital resuscitation has improved by 30%–40% with decreases in response times, early CPR, and the skill of the advanced life support (ALS) teams.<sup>8</sup> While out-of-hospital survival lengthens, in-hospital resuscitation has had less successful outcomes, with survival rates varying around 5%–30%.<sup>18,19</sup> Many factors such as age, presence or absence of morbidity before or during the hospital admission, asystole and non-intensive care unit location contribute to the low in-hospital survival rates.<sup>19,20</sup>

## MANAGEMENT

The overall aim of management of a patient ‘in arrest’ is the prompt restoration of a spontaneous perfusing rhythm with minimal neurological dysfunction. It is well recognised that successful outcome from cardiac arrest is dependent on three key factors: (a) early initiation of CPR, (b) optimising response times, and (c) early defibrillation.<sup>19</sup> The probability of an unsuccessful outcome grows with the length of time taken to restore spontaneous circulation.

### Chain of survival

To optimise a person’s chance of survival, the AHA developed the ‘chain of survival’ strategy,<sup>21</sup> which represents the sequence of four events that must occur as quickly as possible: early

access, early CPR, early defibrillation, and early advanced life support<sup>8,21,22</sup> (see Figure 18.1). These time-sensitive, sequential actions must occur to optimise a cardiac arrest victim’s chances of survival. Communities with integrated links along this chain have demonstrated higher survival rates after OHCA than those with deficiencies in these links.<sup>1</sup>

### Early recognition

The chain of survival begins with early access, when a medical emergency is recognised and the medical system accessed.<sup>8</sup> However, the chain of survival has not always been adequate when a cardiac arrest occurs in the hospital, from the point of view of early recognition, timeliness, or availability of equipment or staff. The traditional cardiac arrest team responded to the seriously ill, but the patient was often not salvageable by the time the cardiac arrest team arrived.<sup>23</sup> Two-thirds of in-hospital cardiac arrests are potentially avoidable, with up to 84% of all in-hospital cardiac arrests demonstrating evidence of deterioration in the 8 hours preceding the arrest.<sup>24–26</sup> Consequently, in recent years there has been a move to implement programs that facilitate the early recognition and rapid management of critically ill patients, for example the medical emergency team (MET)<sup>18</sup>, the patient-at-risk team (PART) and the medical early-warning system (MEWS)<sup>18,27</sup> (see Chapter 5 for further discussion). In some hospitals these teams replace the traditional cardiac arrest team by responding to a calling criteria based primarily on abnormal vital signs (see Table 18.2).

There is a move in hospitals to replace the traditional cardiac arrest team with the MET approach.<sup>27</sup> The MET system calling criteria are widely displayed around the hospital and the MET is activated in the same manner as the cardiac arrest team, ultimately resuscitating patients earlier.<sup>28</sup> Some studies show that in clinically unstable patients, early access—including early recognition and intervention by a MET—can reduce the incidence and mortality from unexpected cardiac arrests by as much as 50%.<sup>29,30</sup> The first cluster randomised study conducted to examine the effects of the introduction of the MET system in 23 Australian hospitals found that while the MET increased the number of calls for assistance, it did not substantially affect the incidence of cardiac arrests, unplanned admissions to ICU or unexpected death.<sup>31</sup>

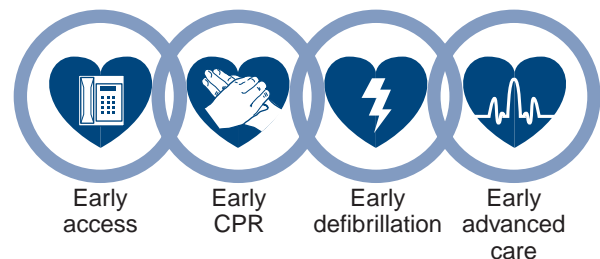


FIGURE 18.1 Chain of survival<sup>21</sup> (published with permission)

TABLE 18.2 Early calling criteria

| Area        | Adults  | Children  |   |
|-------------|---|---|---|
|             |   | 0–12 months   | 1–8 years   |
| Airway      | Threatened  | Threatened  | Threatened  |
| Breathing   | All respiratory arrests<br>Respiratory rate <5<br>Respiratory rate >36  | All respiratory arrests<br>RR <20<br>RR >50<br>Grunting respirations  | All respiratory arrests<br>RR <15<br>RR >35                                     |
| Circulation | All cardiac arrests<br>Pulse rates <40<br>Pulse rates >140<br>Systolic BP <90   | All cardiac arrests<br>Pulse rates <70<br>Pulse rates >180<br>Systolic BP <50<br>Capillary return >5 seconds<br>Marked pallor | All cardiac arrests<br>Pulse rates <50<br>Pulse rates >160<br>Systolic BP <60   |
| Neurology   | Sudden fall in the level of consciousness (fall in the Glasgow Coma Scale of >2 points)<br>Repeated or prolonged seizures | Floppy<br>Unresponsive<br>Depressed conscious level<br>Prolonged seizures   | Floppy<br>Unresponsive<br>Depressed conscious level<br>Prolonged seizures       |
| Other       | Any patient you are seriously worried about who does not fit the above criteria   | Any patient you are seriously worried about who does not fit the above criteria   | Any patient you are seriously worried about who does not fit the above criteria |

## Basic life support

When a patient is identified as in potential or actual arrest, a primary and secondary survey should be conducted in the DRABCD sequence:

- **Danger.** Check for danger.
- **Responsive.** Are they unconscious, unresponsive, not moving, not breathing normally?
- **Airway.** Airway assessment is undertaken to establish a patent airway while maintaining cervical spine support (if injury is suspected).
- **Breathing.** Breathing includes the assessment/establishment of breathing, noting rate/pattern/chest movement and tissue oxygenation. Give two initial breaths ‘rescue breaths’ if the person is not breathing normally.<sup>7</sup> (Note: European Resuscitation Council guidelines do not recommend two initial rescue breaths).
- **Compression.** Give 30 chest compressions followed by two breaths.
- **Defibrillation.** Defibrillation is initiated as soon as a defibrillator is available.

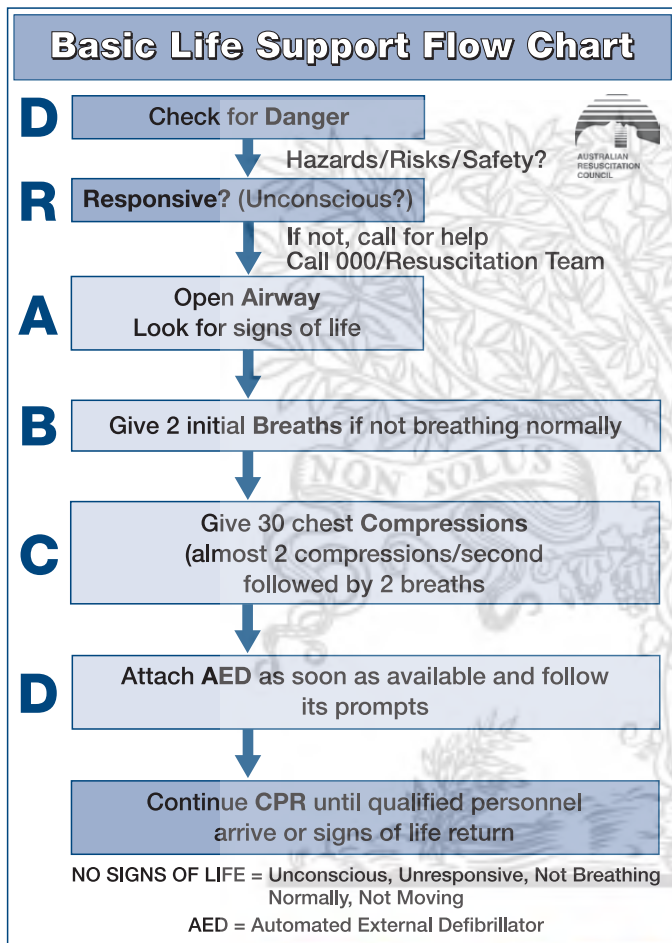
Ideally, these interventions are performed simultaneously or in rapid sequence and will take no longer than 60–90 seconds to complete. This systematic approach (see Chapter 7) correlates closely with the principles of basic life support (BLS), in that where a life-threatening abnormality is detected, immediate intervention is required before further assessment (see Figure 18.2).

### AIRWAY

Recognition of airway obstruction includes listening for

inspiratory (stridor), expiratory or grunting noises. The work of breathing can be assessed by the respiratory rate, intercostals, subcostal or sternal recession, use of accessory muscles, tracheal tug or flaring of the alae nasi. Nasal flaring is especially evident in infants with respiratory distress. Noisy breathing is obstructed breathing, but the volume of the noise is not an indicator of the severity of respiratory failure. Should obstruction to air flow be detected then the airway should be opened, using three manoeuvres: the head-tilt, chin-lift, and jaw thrust. The ARC recommends assessing a person’s airway without turning them onto the side unless the airway is obstructed with fluid (vomit or blood) or submersion injuries.<sup>7</sup>

The airway of the infant differs from that of the older child or adult in that the infant has a large head and tongue, small mouth, and the larynx is narrower, shorter, more anterior and acutely angled.<sup>10</sup> The airway of an infant is also more cartilaginous and can be easily occluded when the neck is hyperextended; in addition, the large tongue can easily fall back to obstruct the pharynx.<sup>32</sup> Hence, the head of an infant should be maintained in the neutral position, whereas the child aged 1–8 will require the ‘sniffing position’ with varying degrees according to age. The chin-lift and head-tilt manoeuvres may be used in children to obtain the appropriate amount of positioning for age. Jaw thrust may be used if head-tilt/chin-lift is contraindicated.<sup>32</sup> Do not use the finger sweep to clear the airway of an infant, as this may result in damage to the delicate palatal tissues and cause bleeding, which can worsen the situation. Use of finger sweep can force foreign bodies further down into the airway.<sup>32</sup> Suction is more useful for removing vomitus and secretions.



**FIGURE 18.2** Australian Resuscitation Council basic life support algorithm<sup>7</sup> (published with permission)

### Practice tip

Infants are obligatory nose-breathers, so it is always important to clear the nostrils.<sup>7</sup>

### BREATHING

To assess for the presence of breathing, look, listen and feel for breath sounds for 10 seconds. If no breathing is detected, rescue breathing should be commenced immediately.<sup>7</sup> Give two initial breaths at a rate of one breath per second (ERC, recommends no rescue breaths). Slow breaths will lessen the risk of inflation of the stomach.<sup>10</sup> As the airways and lungs of an infant are small, there is a subsequent rapid fall in oxygen saturation with cessation in ventilation.<sup>10</sup> Bradycardia in children may be a sign of respiratory failure, so there is particular emphasis on ventilation during paediatric resuscitation.

Studies note that the average person may not only be reluctant to initiate mouth-to-mouth resuscitation but will also take 8 seconds to deliver one breath.<sup>33</sup> When a rescuer is reluctant to perform rescue breaths, external cardiac compression (ECC) without expired air resuscitation (EAR)

should be encouraged, as the generally held belief is that ECC alone is better than no CPR at all.<sup>34</sup>

### CIRCULATION

Lay rescuers should commence cardiac compressions if the victim is unconscious, unresponsive, not moving and not breathing normally.<sup>7</sup> Pulse check by lay rescuers is not recommended.<sup>7,8</sup> Assessment of circulation by healthcare professionals may be made by palpation of the carotid artery, or femoral (in adults) and brachial artery, or femoral artery (in infants), for up to 10 seconds. CPR (the technique of lung inflation) and ECC (external cardiac compressions) in the event of a cardiac arrest, are an attempt to restore respiration and circulation.<sup>7</sup> For CPR to be effective the patient must be flat, supine and on a firm surface. The chest should be compressed in the midline over the lower half of the sternum, which equates to the ‘centre of the chest’, at a depth of up to one-third of the chest and at a rate of 100 compressions per minute for adults, infants and children, with the rate rising to 120/min for the newborn.<sup>7</sup> CPR should be initiated when the heart rate is 80 beats/min for the neonate or infant, 60 beats/min for the small child and 40 beats/min for the large child. Performed correctly, ECC can produce a systolic blood pressure peak of 60–80 mmHg (in adults) and a cardiac output of 20%–30% of normal.<sup>7</sup> With external chest compressions it takes time to reach optimal levels of coronary perfusion pressure and ultimately blood flow. Any interruption to chest compressions therefore decreases the coronary perfusion pressure and resultant blood flow, ultimately reducing survival.<sup>35,36</sup>

Survival is potentially improved when an individual receives a higher number of chest compressions during CPR, even if the person receives fewer ventilations.<sup>2</sup> Because of this, guidelines now recommend a 30:2 compression-to-ventilation ratio in adults, children and infants regardless of the number of rescuers, and 3:1 for neonates.<sup>2,7</sup> Having noted this, in the advanced life support paediatric setting the compression ratio changes to 15:2 and a ratio of 3:1 for the newborn with any number of rescuers (see Table 18.3).

**Devices to augment compression.** As ECC supplies only 25% of normal cardiac output and 15% of normal cerebral blood flow, there is a great need to find ways to improve ECC. A few of the recent devices are outlined below:

- active compression–decompression CPR;
  - minimally invasive cardiac massage device;
  - interposed abdominal compression combined with CPR; and
  - non-invasive automated chest compression device.
- Active compression–decompression (ACD-CPR) utilises a small portable device to compress and decompress the chest (‘plunger method’), and enhances ventilation and venous return by raising the negative intrathoracic pressure.<sup>9,37</sup>

### Practice tip

CPR should commence if the patient is unconscious, unresponsive, not moving and not breathing, even if the patient is taking the occasional gasp.<sup>7</sup>

**TABLE 18.3 CPR for adults, children and infants**

|                          | Airway   | Compression (CPR)  | 1 or 2 person        |
|--------------------------|--|--|----------------------|
| Infants <1 year          | Jaw support or chin-lift (no head-tilt)          | Two fingers or two overlying thumbs on the lower end of the sternum with hands encircling the chest, 100 beats/min | 30:2<br>PALS<br>15:2 |
| Younger child: 1–8 years | Head-tilt more than infants but less than adults | heel of one hand, 100 beats/min  | 30:2<br>PALS<br>15:2 |
| Older child: 9–14 years  | Head-tilt  | two hands, 100 beats/min   | 30:2<br>PALS<br>15:2 |
| Adult                    | Head-tilt  | two hands, 100 beats/min   | 30:2                 |

PALS = paediatric advanced life support

The greater negative intrathoracic pressure facilitates venous return, thus priming the heart for subsequent compressions.<sup>38</sup>

The minimally invasive direct cardiac massage device (MIDCM) requires the user to make a small incision into the anterior left fifth intercostal space of the patient's chest and insert a trocar fitted with an umbrella. Once expanded, compression of the umbrella allows direct intrathoracic compression of the heart.<sup>38,39</sup> The handle and stem of the device remain outside the thorax, which allows the operator to perform direct cardiac massage in a pumping action.<sup>39</sup> Experimental studies show that while the MIDCM can be positioned within a few seconds and provides significantly greater perfusion pressures than ECC,<sup>39</sup> the device has also caused cardiac rupture, and hence is no longer manufactured.<sup>8</sup>

The interposed abdominal compression (IAC) combined with CPR (IAC-CPR) is the least technical device, and receives the highest recommendation from the AHA.<sup>38</sup> In IAC-CPR the abdomen is compressed alternately with the rhythm of chest compression during cardiac arrest. The point of abdominal compression midway between the xiphisternum and the umbilicus results in an increase in the resistance in the descending aorta, thus raising the coronary perfusion pressure (CPP).<sup>38</sup>

The non-invasive automated chest compression device (AutoPulse) utilises a load-distributing band (LDB) to compress the anterior chest.<sup>40</sup> The device is built around a backboard that contains a motor. As the motor turns, the LDB is tightened or loosened around the patient's chest. The band distributes the compressive load over the anterior chest wall to reduce the local stress. When compared with

manual CPR, the automated chest compression device has demonstrated better coronary perfusion.<sup>40</sup>

Given the limited available evidence on the impact of any of these devices on outcome, no recommendations are available to either support or refute their use.

## DEFIBRILLATION

While CPR has been associated with improved survival to discharge from hospital, it cannot be substituted for the definitive treatment of early defibrillation. It is thought that CPR will supply sufficient oxygen to the brain and heart until defibrillation is available. Ultimately, despite the most effective CPR, the single most important cause of decreased prognosis in pulseless VT/VF cardiac arrests is a delay in electrical defibrillation.<sup>3</sup>

**Praecordial thump.** A timely delivered praecordial thump can terminate VT and VF or act as pacemaker stimulus during an episode of asystole.<sup>41</sup> A praecordial thump is a single, sharp blow delivered with a clenched fist to the midsternum of a victim's chest from a height of 25–30 cm above the sternum.<sup>7</sup> It is likely that the mechanical energy generated by the praecordial thump will generate a few joules, and therefore has the potential to result in cardioversion if applied within the first 15 seconds of a cardiac arrest.<sup>7,8,41</sup> A 'thump' is unlikely, however, to be successful after more than 30 seconds into the arrest. Currently there is no clear consensus as to whether a single or series of thumps is more effective.<sup>42,43</sup> Further, while recommended for use in children, its efficacy has not been proven.<sup>7</sup>

While generally safe, there is anecdotal evidence that a praecordial thump may convert bradycardic rhythms to VT, VF, complete heart block or asystole.<sup>7,41,43</sup> For these reasons the praecordial thump is recommended only with a witnessed or monitored VF cardiac arrest, or with electrocution-associated collapse if a defibrillator is not available.<sup>7</sup> A monitored, alert patient in pulseless VT should be encouraged to generate a strong cough as a means of returning the VT to a perfusing rhythm.

**Electrical defibrillation.** Defibrillation is the passage of a current of electricity through a fibrillating heart to simultaneously depolarise the mass of myocardial cells and allow them to repolarise uniformly to an organised electrical activity.<sup>44</sup> There are two defibrillator modes for delivery of electrical energy: monophasic and biphasic waveforms. Monophasic defibrillators operate by the current travelling in one direction from one paddle through the heart to the opposite paddle.<sup>45</sup> In comparison, the biphasic defibrillator's current travels in one direction through the heart for a predetermined time, then reverses. The nature of the biphasic waveform means that a shock delivered at 200 J is required rather than the 360 J (2 J/kg for children) required by the monophasic defibrillator protocol.<sup>7,8</sup> Biphasic defibrillators

## Practice tip

Effective BLS can slow the loss of amplitude and waveform of VF. Interruptions to effective CPR should be kept to a minimum.

may be preprogrammed to deliver a shock of anywhere between 150 and 360 J. For children, there is no difference in the joules delivered between monophasic and biphasic waveforms (i.e. 2J/kg).

As high-energy-level phosphate stores in the myocardium decrease, so does the amplitude and waveform of ventricular fibrillation.<sup>7</sup> A period of effective BLS can help to maintain myocardial and cerebral viability by slowing the loss of phosphate stores and potentially improving the likelihood of subsequent shocks.<sup>7</sup> Because of this, the single-shock strategy is used on patients in VF/VT, as opposed to the escalating sequence. However, if the arrest is a witnessed and monitored VF/VT arrest, then a salvo of three shocks should be administered in less than 30 seconds.<sup>7</sup>

Studies have demonstrated that lower-energy biphasic defibrillators are associated with greater first-shock efficacy, greater efficacy in three or fewer shocks, require lower joules, cause less myocardial dysfunction, and increase return of spontaneous circulation when compared with the monophasic defibrillator.<sup>46–48</sup> Not all the electrical energy delivered during defibrillation will traverse the myocardium. Table 18.4 outlines some of the common factors contributing to the success or failure of defibrillation.

There are two types of external defibrillators—the manual external defibrillator (MED), and the automatic external defibrillator (AED). The AED can be either fully automatic (FAED) or semiautomatic (SAED). The MED requires the user to be able to immediately and accurately recognise arrhythmias and make the decision whether to initiate defibrillation or not. In comparison, the AED automatically detects and interprets the rhythm without relying on the

user’s recognition of arrhythmias. When using an SAED, the user determines whether the person is unresponsive, not breathing and pulseless.<sup>49</sup> After checking for a pulse, the SAED requires only four steps to operate: turn power on, place self-adhesive electrodes on a victim’s chest, rhythm analysis follows, then (if advised by the machine) press the shock button.<sup>9</sup> The SAED will automatically interpret the cardiac rhythm and if VF/VT is present will advise the operator to provide a shock. FAEDs are programmed to assess the rhythm, charge the defibrillator, and deliver shocks without user intervention.

Biphasic AEDs are safe, easy to use and are effective for detecting and classifying arrhythmias (sensitivity 100%, specificity 97%).<sup>9,50</sup> Defibrillation is recommended for children with documented VF and pulseless VT cardiac arrests.<sup>51</sup> Where a manual defibrillator can be programmed, it is recommended that 2 J/kg, 2–4 J/kg or 4 J/kg be selected for the initial shock and 4 J/kg for subsequent shocks,<sup>7</sup> using a paddle size of 4.5 cm.<sup>10</sup> Generally, AEDs are not programmed for use on children under 8 years of age or below 25 kg,<sup>9,51</sup> as the SAED is preprogrammed to deliver a fixed energy, usually 200 J (which significantly exceeds the recommended 2–4 J/kg in young children and infants). Alternatively, paediatric levels on an SAED are generally preprogrammed to deliver 50 J for the 1–8-year-old child, with the adult preset level for a child over 8 years of age.<sup>7</sup>

Successful defibrillation and survival to discharge is inversely related to the time from onset of ventricular fibrillation to defibrillation.<sup>2,52</sup> For every minute that passes, the probability of survival decreases 5%–10%,<sup>2</sup> so resuscitation bodies place great emphasis on early defibrillation. To facilitate early defibrillation, ILCOR endorses the concept of non-medical individuals being authorised, educated and encouraged to use defibrillators.<sup>2,53</sup> This public access to early defibrillation has seen the placement of defibrillators on aircraft, in casinos and cricket grounds, with non-medical personnel such as police, flight attendants, security guards, family members and even children successfully initiating early defibrillation.<sup>54,55</sup> The effectiveness of training non-traditional out-of-hospital first responders to use the AED has

**Practice tip**

Remember, evaporative water loss from defibrillation pads is determined principally by the time of exposure to air and environmental conditions, rather than by the number or total energy of shocks. This means the passage of a defibrillation current across pads does not accelerate water loss, so you should change the defibrillation pads after 30 minutes.<sup>119</sup>

**TABLE 18.4 Factors contributing to the success or failure of defibrillation**

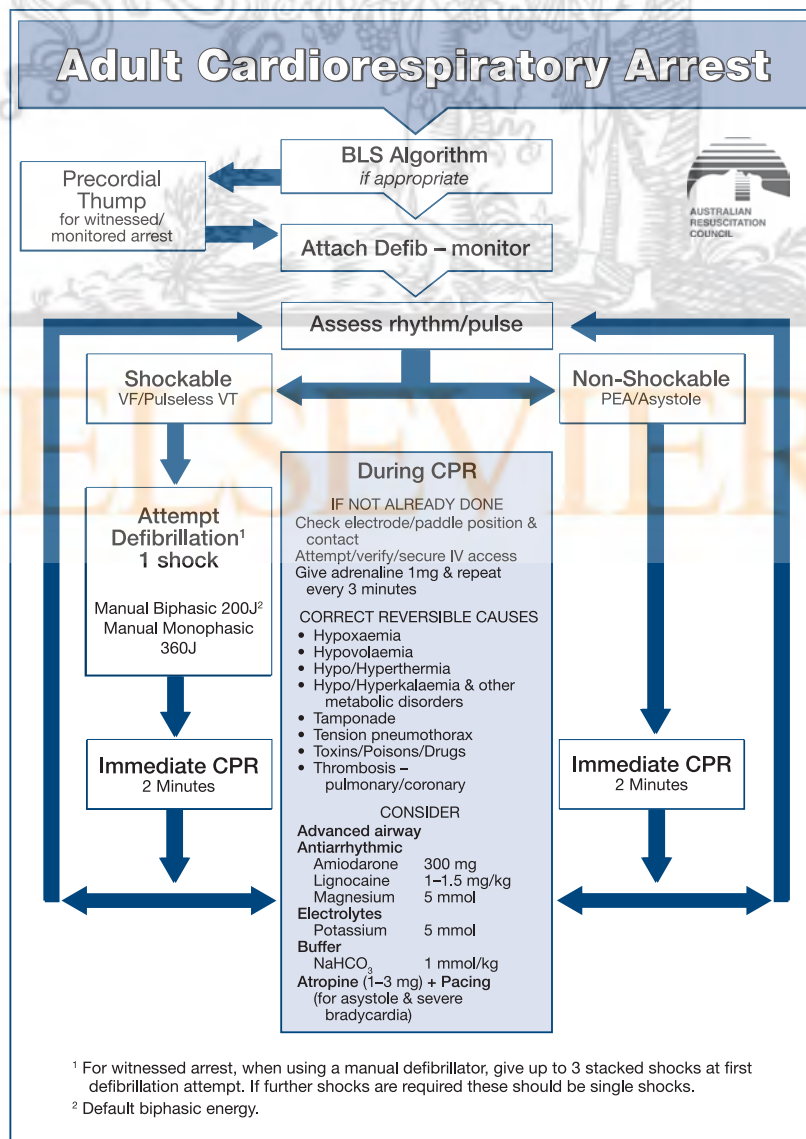
| Success   | Failure  | Precautions   |
|---|--|---|
| <ul style="list-style-type: none"> <li>• Early defibrillation (&lt;4 min)</li> <li>• Presenting rhythm (VT/VF)</li> </ul> | <ul style="list-style-type: none"> <li>• Inadequate contact with the chest</li> <li>• Inadequate pressure over paddles (8 kg required for adults)</li> <li>• Faulty positioning of the paddles</li> <li>• Synchronise button in the on position, flat battery or fractures lead</li> <li>• Positioning over bone/fat tissue</li> <li>• Drying out of gel conduction pads</li> <li>• Patient factors: acidosis, hypoxia, electrolyte imbalance, drug toxicity, hypothermia</li> <li>• Time of respiration (best delivered during expiration)</li> <li>• Paddles/electrodes too small (12 cm electrodes for adults)</li> </ul> | <ul style="list-style-type: none"> <li>• Do not defibrillate over ECG electrodes, nitrate patches, pacemakers, vascular access devices</li> <li>• Do not defibrillate unless all clear of the bed/patient</li> <li>• Do not charge/discharge paddles in the air</li> <li>• Do not have the patient in contact with metal</li> <li>• Do not allow oxygen to flow onto the patient during delivery of the shock</li> <li>• Ensure the chest is dry</li> </ul> |

improved survival to discharge rates.<sup>22,56</sup> Similarly, in-hospital cardiac arrests also occur in any area, and all healthcare workers should be capable of initiating early defibrillation.<sup>49</sup> The ARC notes that while BLS does not include the use of adjunctive equipment, the use of SAEDs by persons with education in their use is accepted.<sup>7</sup> Figure 18.3 outlines the integration of defibrillation with BLS.

For 80% of people, return of spontaneous circulation will occur after one of the first three shocks.<sup>57</sup> While early defibrillation is vital in improving outcome for pulseless VT/VF cardiac arrests, a period of CPR to maintain cardiac and cerebral perfusion is recommended.<sup>7</sup> Reducing any interruption to chest compressions during a cardiac arrest cannot be overemphasised as the passage of time reduces the chances of successful defibrillation.<sup>57</sup> For this reason resuscitation bodies now recommend single shocks, followed by 2 minutes of CPR.<sup>7,8</sup>

**The role of the chest compression prior to defibrillation.** The importance of early defibrillation is well promulgated in the ILCOR guidelines.<sup>7,8</sup> However, the guidelines do not consider the duration of the shockable rhythm. While defibrillation of prolonged VF often results in either pulseless electrical activity (PEA) or asystole, there is growing evidence to suggest that a sustained period of CPR prior to defibrillation may circumvent a poor prognosis.<sup>58</sup> Current research suggests that the practice of defibrillation of all patients regardless of the duration of shockable rhythm should be reconsidered.<sup>59,60</sup> Defibrillation of the globally ischaemic heart beyond 4 minutes may be detrimental.<sup>61</sup> In view of this, researchers propose a three-phase time-sensitive model for resuscitation (see Table 18.5).<sup>61</sup>

During the electrical phase, effectiveness of defibrillation is well established, with survival rates approaching 50%.<sup>61</sup> During the circulatory phase, return of spontaneous circulation appears to improve when defibrillation is delayed



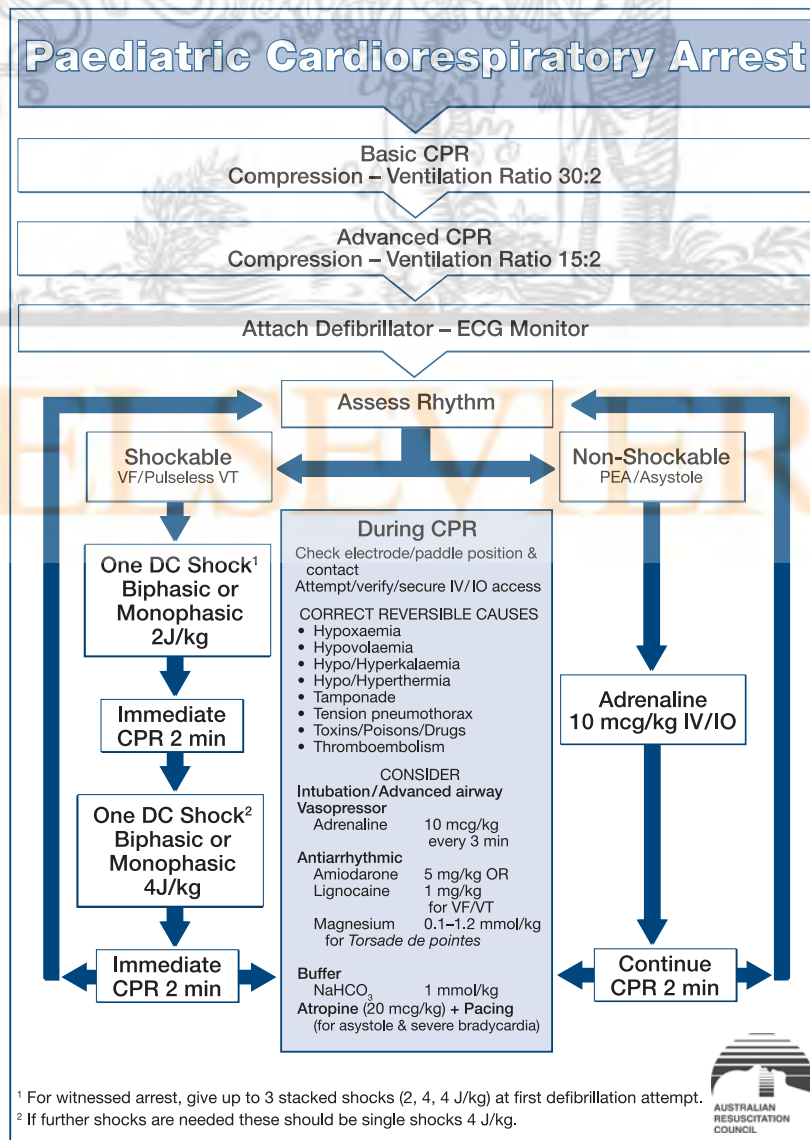
**FIGURE 18.3** Advanced life support flowchart<sup>7</sup> (published with permission)

in favour of a period of CPR and/or the administration of adrenaline.<sup>59</sup> The final or metabolic phase is reached after 10 minutes of cardiac arrest, and the effectiveness of CPR and defibrillation decreases rapidly.<sup>61</sup>

| Phase                 | Description of phase   |
|-----------------------|--|
| The electrical phase  | Time of cardiac arrest to approximately 4 minutes after the arrest |
| The circulatory phase | Approximately 4–10 minutes after the arrest                        |
| The metabolic phase   | Extending beyond approximately 10 minutes                          |

### Advanced life support

Basic life support can provide around 20%–30% of normal cardiac output and an FiO<sub>2</sub> of 0.1–0.16. Consequently, a significant number of patients rely on the provision of advanced life support (ALS) for survival.<sup>7</sup> ALS extends BLS to provide the knowledge and skills essential for the initiation of early treatment and stabilisation of people post-cardiac arrest. Advanced skills traditionally include defibrillation, advanced airway management and the administration of resuscitation drugs. While BLS generally is initiated prior to ALS, where a defibrillator and a person trained in its use are available, defibrillation takes precedence over BLS and ALS. The ARC algorithm for management of cardiopulmonary arrest (see Figures 18.3 and 18.4) outlines the two decision paths of therapy in ALS: (a) defibrillation and CPR for pulseless VT/VF; and (b) identifying and treating the underlying cause for non-VT/VF.



**FIGURE 18.4** Paediatric advanced life support flowchart<sup>7</sup> (published with permission)

## ADVANCED AIRWAY MANAGEMENT

A person with signs of acute respiratory distress should be administered oxygen at the highest possible concentration. Oxygen should never be withheld for fear of adverse effects,<sup>7</sup> as rescue breaths provide an inspired oxygen concentration of only 15%–18%. The administration of oxygen alone does not result in adequate ventilation, and as such the establishment of an effective airway is paramount. Airway management is essential in the performance of CPR, and may be administered using a variety of techniques.

The choice of advanced airway adjunct is determined by the availability of equipment and experienced personnel (see Table 18.6 and Chapter 11):

- oropharyngeal (Guedel's) airway;
- nasopharyngeal airway;
- laryngeal mask airway;
- oesophageal–tracheal Combitube;
- endotracheal intubation; and
- tracheostomy.

The endotracheal tube (ETT) is considered the 'gold standard' for airway management in a cardiac arrest,<sup>7</sup> as it protects the airway, assists effective ventilation, ensures delivery of high concentrations of oxygen and suctioning, and facilitates administration of medications.<sup>1</sup> It is vital that CPR not be interrupted for more than 20 seconds during attempts at endotracheal intubation. Given the limitations noted in Table 18.6, a variety of adjunct airway/ventilation management devices such as bag–mask–ventilation (BMV), laryngeal mask airway (LMA) and the oesophageal–tracheal Combitube (ETC) are now available. When an LMA–*Fasttrach* is in place it can be used to guide the passage of bougies, introducers, a bronchoscope or an ETT into the trachea.<sup>62</sup> Currently, there is no evidence to support the routine use of any particular advanced adjunct airway.

Once an airway has been established, continue chest compressions without interruption for ventilation. Ventilate the lungs at a rate of approximately 10 breaths a minute and an inspiratory time of 1 second and sufficient volume to produce a normal chest rise. Ventilation adjuncts may include:

- a simple face mask with filter and oxygen connector (preintubation);
- bag–valve–mask systems; and
- ventilators.

If available, automated ventilators can be used. These may be set to deliver a tidal volume of 6–7 mL/kg at a rate of 10 breaths/min.<sup>8</sup> The automated ventilator may be used with either the face mask or other adjunct airway devices such as LMA, Combitube or ETT.<sup>8</sup> Having noted this, there is currently no evidence to suggest that the use of automated ventilators during cardiac arrest are more beneficial than bag–valve–mask devices.<sup>8</sup>

## INITIAL RHYTHM

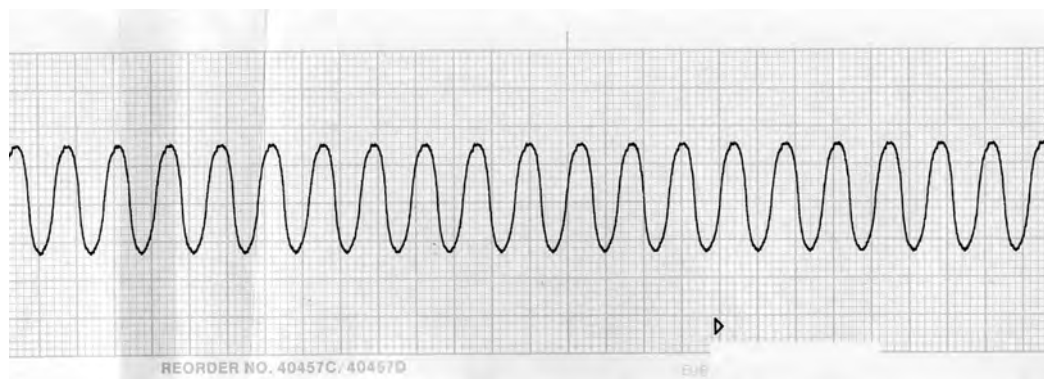
There is an association between the initial cardiac arrhythmias and survival to discharge after SCA. Cardiac arrest rhythms can be divided into two subsets:

1. ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT); and
2. non-VF/VT incorporating asystole and pulseless electrical activity (PEA).

The commonest arrhythmias observed in SCA are pulseless VT and VF, with 60%–85% of all patients initially presenting with these lethal arrhythmias.<sup>3</sup> PEA occurs as the initial rhythm in approximately 13%–22% of cases;<sup>63,64</sup> when witnessed by emergency personnel in the prehospital setting, it has been documented at as high as 50%.<sup>65</sup> Generally, patients over 70 years of age presenting in asystole after an unwitnessed cardiac arrest will not survive.<sup>9</sup> Asystole is the commonest arrest arrhythmia in children, because their hearts respond to prolonged severe hypoxia and acidosis by progressive bradycardia leading to asystole.<sup>10</sup>

**Ventricular fibrillation and pulseless ventricular tachycardia.** As previously noted, the only intervention shown to unequivocally improve long-term survival after a VF or pulseless VT arrest is BLS and early defibrillation.<sup>36</sup> VT and VF rhythms are displayed in Figures 18.5 and 18.6. Defibrillation is the definitive therapy for ventricular fibrillation and pulseless ventricular tachycardia.<sup>8,9</sup> Energy levels and subsequent shocks are equivalent for both VF and pulseless VT.

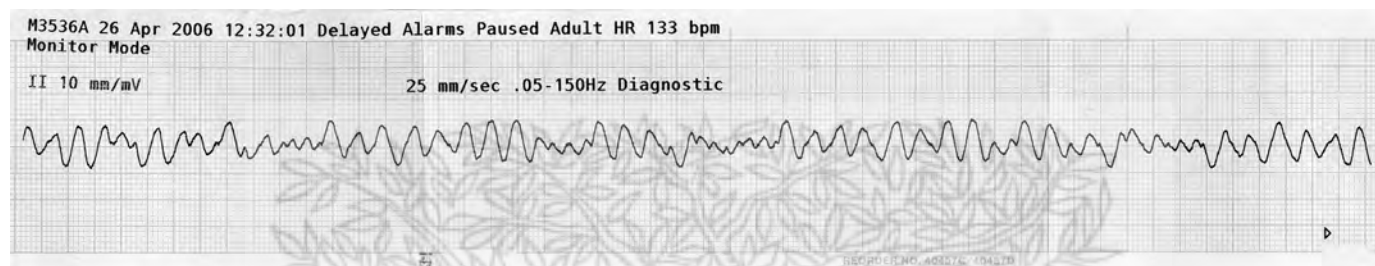
**Non-VF/VT.** This set of arrhythmias includes pulseless electrical activity and asystole. Pulseless electrical activity (PEA) or electromechanical dissociation (EMD) reflects a dissociation between the heart's electrical and mechanical activities, and the two terms are used interchangeably. It is important to note that PEA/EMD may present as any



**FIGURE 18.5** Ventricular tachycardia

TABLE 18.6 Adjuncts used during resuscitation

| Airway type                          | Description   | Practice considerations   |
|--------------------------------------|---|---|
| Oropharyngeal (Guedel's) airway      | Conforms to the curve of the palate, moving the tongue forwards away from the posterior pharyngeal wall. <sup>32</sup> Sizes from 000–5.  | Incorrect size or placement may contribute to airway obstruction by pushing the tongue back into the pharynx. Unlike adult insertion, the insertion of the oropharyngeal airway in infants and young children is inserted right-way-up; a tongue depressor or laryngoscope should be used to aid insertion. <sup>32</sup>   |
| Nasopharyngeal airway                | Soft tube inserted into the nasopharynx.  | Use with caution in patients with head injuries. <sup>7</sup> With the exception of infant's head-tilt, jaw support or jaw thrust is still necessary when using either the oropharyngeal or the nasopharyngeal. <sup>7</sup> Remember, children under 1 year of age are nose-breathers, and anything that blocks their nose is going to severely compromise their breathing. <sup>32</sup>  |
| Bag–valve–mask (BVM) systems         | A self-inflating bag that may be connected to a face mask, LMA or ETT.  | BVMs are often inappropriately used and offer no protection to the airway. <sup>120</sup> Single-person BVM ventilation may result in a poor seal around the patient's mouth and the delivery of less than optimal tidal volumes. <sup>121</sup> When using a BVM it is best performed using two rescuers, although not always possible. As the airway is not protected, smaller tidal volumes with supplementary oxygen can provide adequate oxygenation and reduce the risk of gastric inflation, regurgitation and aspiration. <sup>36</sup> The mask should be used right-way-up with children and upside-down with infants. The soft circular mask is preferred for infants, as it provides an excellent seal with low dead space. <sup>10</sup>   |
| Laryngeal mask airway (LMA)          | The LMA consists of a tube with an elliptical cuff fitted at the distal end that inflates in the hypopharynx around the posterior perimeter of the larynx. The LMA is inserted orally using a blind technique so that the distal end of the mask abuts against the base of the hypopharynx, behind the cricoid cartilage, and the cuff is inflated to form an airtight seal around the larynx. <sup>6,123</sup> | The LMA is used as a first-line adjunct when endotracheal intubation is not available (Class IIb). <sup>7,8,36</sup> The LMA is more rapidly inserted and requires less equipment than the endotracheal tube. <sup>121,122</sup> When used as a first-line airway device, the LMA provides a clear airway with a significantly lower risk of gastric overinflation and regurgitation than the BVM. <sup>122</sup> As with adults, the LMA can be used safely and effectively in infants. <sup>10</sup> Size 1 LMA is recommended for infants under 5 kg and 1.5 for heavier babies. <sup>10</sup> Complications of LMA include gastric aspiration, partial airway obstruction, coughing or gastric insufflation. <sup>62</sup> Contraindications include patients unable to open their mouths adequately; pharyngeal pathology; airway obstruction at or below level of the larynx; low pulmonary compliance or high airway resistance; or increased risk of aspiration. <sup>122</sup> |
| Oesophageal–tracheal Combitube (ETC) | The ETC is a double-lumen airway with proximal and distal cuffs that is inserted blindly.   | It is effective in maintaining an airway when performed by unskilled personnel and is a suitable alternative to tracheal intubation. <sup>124</sup> The ETC enables ventilation, whether it is positioned in the oesophagus or the trachea.   |
| Endotracheal tube (ETT)              | During intubation, direct application of firm pressure to the cricoid cartilage is required to compress the oesophagus between the trachea and vertebral column and minimise/prevent regurgitation of gastric contents.   | Endotracheal intubation is a difficult skill to acquire and maintain. In addition to routine clinical methods, ETT placement can be confirmed by either measurement of ET <sub>CO</sub> <sub>2</sub> or oesophageal detector; the latter is more reliable in a non-perfusing rhythm (Class IIb). <sup>36</sup> Immediate complications associated with intubation include oesophageal intubation; right main bronchi intubation; or ETT occlusion (kinking, sputum, cuff, blood).   |



**FIGURE 18.6** Ventricular fibrillation



**FIGURE 18.7** Asystole

rhythm normally compatible with a pulse (e.g. sinus rhythm, sinus tachycardia/bradycardia). PEA is characterised by a stroke volume insufficient to produce a palpable pulse, despite adequate electrical activity.<sup>66</sup> PEA often follows defibrillation of VF and has a survival rate of 0%–6%.<sup>8,66</sup> Management of PEA includes identifying and correcting reversible causes, summarised as the 4 Hs and 4 Ts<sup>7</sup> in Table 18.7.

Careful confirmation of asystole (see Figure 18.7) on two leads and the absence of a palpable pulse are essential when making the decision to manage asystole.<sup>36</sup> When an out-of-hospital arrest has an initial rhythm of asystole, survival to discharge is as low as 2%.<sup>8,9</sup>

### MEDICATIONS ADMINISTERED DURING CARDIAC ARREST

Resuscitation drugs can be administered during a cardiac arrest using a variety of routes including peripheral and central veins, endotracheal or intraosseous (IO). Administration by the central venous route remains the optimal method, but the decision to access peripheral versus central cannulation

will depend on the skill of the operator. Should a decision be made to insert a central line during a cardiac arrest, this must not take precedence over defibrillation attempts, CPR or airway maintenance.<sup>57</sup> Medications inserted into a peripheral line should be flushed with at least 30 mL (adults) of an isotonic solution followed by at least 1 minute of continuous external cardiac compressions.<sup>7</sup> Where there is difficulty accessing a peripheral vein, selected medications may be administered via the ETT for adults and IO in children and infants.

Intraosseous infusion involves the insertion of a metal needle with trocar into the bone marrow and provides a rapid, safe and reliable access to the circulation. The marrow sinusoids of long bones are a non-collapsible venous system in direct connection with the systemic circulation, allowing drugs to reach the central circulation as quickly as medications injected into central veins.<sup>67</sup> Intraosseal access is safe and effective for use in patients of all age groups.<sup>7</sup> General blood specimens such as biochemistry values, blood cultures, haemoglobin and cross-match studies can also be taken from the marrow at cannulation.<sup>10</sup>

Endotracheal instillation of medications is a poor substitute for medications administered by vascular access or IO.<sup>67</sup> Prior to administration of endotracheal medications, the airway should be suctioned. Medications are administered beyond the tip of the ETT and at three to 10 times the normal

**TABLE 18.7** Causes of pulseless electrical activity<sup>7</sup>

| The four Hs  | The four Ts  |
|--|--|
| <ul style="list-style-type: none"> <li>• Hypoxia</li> <li>• Hypovolaemia</li> <li>• Hypo/hyperthermia</li> <li>• Hypo/hyperkalaemia and metabolic disorders</li> </ul> | <ul style="list-style-type: none"> <li>• Tamponade</li> <li>• Tension pneumothorax</li> <li>• Toxins/poisons/drugs</li> <li>• Thrombosis—pulmonary/coronary</li> </ul> |

### Practice tip

Attempts at peripheral cannulation in children should be aborted after 2 minutes and an intraosseous needle inserted.

dose, diluted in water. A minimum of two ventilations is required to disperse the medication.<sup>7</sup> Lignocaine, atropine, naloxone, salbutamol and adrenaline may be administered by this route. Other medications must not be administered by this route, as they may cause mucosal or alveolar damage.<sup>7</sup> In children the optimal dose is unknown, and medications are diluted to 0.7–1.0 mL in the newborn, 1–2 mL for an infant, 2–5 mL for small children, 5–10 mL for larger children, and 10 mL in adults.<sup>7</sup>

**Vasopressors.** Vasopressors such as adrenaline/epinephrine and vasopressin have been used as adjuncts in cardiac arrests to improve the success of CPR. The optimal dose of adrenaline in the prehospital and in-hospital setting remains unclear (see Table 18.8). Randomised controlled trials have failed to show benefits of high-dose over low-dose adrenaline (1 mg vs 5 mg) in terms of patient survival.<sup>1</sup> Recent studies have reported that vasopressin produced no overall change in survival after cardiac arrest when compared with adrenaline.<sup>68,69</sup> Currently there is no evidence to support or refute the use of vasopressin as an alternative to or in combination with adrenaline.<sup>7</sup>

**Antiarrhythmics.** The optimal role and exact benefit of antiarrhythmic medications in cardiac resuscitation is yet to be fully elucidated, but they have very little if any role to play in the treatment of cardiac arrests.<sup>1,8</sup> The common antiarrhythmic drugs include amiodarone, lignocaine, magnesium, atropine and calcium (see Table 18.8). The efficacy of IV amiodarone in the setting of VT and VF is 51%–100%.<sup>70</sup> Lignocaine is an alternative to amiodarone, but the two should not be used together.<sup>36</sup> To date there have been no placebo-controlled studies demonstrating a survival benefit from the use of lignocaine in the management of VF.<sup>8</sup> While no antiarrhythmic has been shown to improve survival to discharge, recent trials have demonstrated that amiodarone is superior to lignocaine and placebo in improving survival to hospital admission for people with refractory VF in out-of-hospital cardiac arrests.<sup>71</sup> There is no evidence of improved survival with the use of atropine in a cardiac arrest.<sup>44</sup> Calcium chloride has little use in the management of arrhythmias unless caused by hyperkalaemia, hypocalcaemia or hypermagnesaemia, or an overdose of calcium channel-blocking drugs. Sodium bicarbonate is no longer administered routinely, as it may cause hypernatraemia, hyperosmolality and intracellular acidosis from the rapid ingress of CO<sub>2</sub> generated from its dissociation.<sup>44</sup>

There are insufficient data either for or against the routine use of magnesium in cardiac arrests.<sup>7</sup> Fibrinolysis should be considered for administration in adult patients with proven or suspected pulmonary embolism<sup>7</sup> or acute thrombotic aetiology.<sup>8</sup> Having noted this, there are insufficient data to support or refute its routine use in cardiac arrests from other causes.<sup>7</sup>

During the arrest, strategies should be initiated to prevent the development of serious periarrest arrhythmias. Whenever possible, arterial blood gases, serum electrolytes and a 12-lead ECG should be obtained to assist with determining the precise rhythm and appropriate medical interventions.<sup>8</sup> The ERC recommends the assessment and treatment of all

periarrest arrhythmias to address the stability of the patient and the nature of the arrhythmia.<sup>8</sup> The presence or absence of adverse signs and symptoms will dictate interventions. Adverse factors may include clinical evidence of:

- low cardiac output (unconscious, unresponsive, systolic BP <90 mmHg, increased sympathetic activity);
- reduced diastolic filling time (excessive tachycardia, e.g. heart rates of >150/min, wide complex tachycardia and supraventricular tachycardia);
- excessive bradycardia (heart rates of <40/min);
- raised end-diastolic filling pressure (presence of pulmonary oedema or raised jugular venous pressures); and
- reduced coronary blood flow (chest pain).

Interventions can broadly be divided into three options for intermediate treatment:

1. antiarrhythmics (refer to *periarrest* in Table 18.8);
2. electrical cardioversion; or
3. cardiac pacing.

Common periarrest arrhythmias and interventions are covered in Chapter 10. The ERC cautions that all antiarrhythmic interventions such as medications, physical manoeuvres and electrical therapies may also be proarrhythmic.<sup>8</sup>

## FLUID RESUSCITATION

Currently there are no recommendations on the routine use of fluids during a cardiac arrest. Although a 1998 systematic review concluded that the use of albumin in critically ill patients may increase mortality,<sup>72</sup> the saline versus albumin fluid evaluation (SAFE) randomised controlled study found that 28-day mortality did not differ significantly between 4% albumin and 0.9% saline<sup>73</sup> (see Chapter 15 for further discussion).

## PACING

During a cardiac arrest, temporary cardiac pacing may be required for sustained symptomatic bradycardia unresponsive to medical intervention. Two types of temporary cardiac pacing are utilised during a cardiac arrest—transvenous (invasive) and transcutaneous (external, non-invasive) pacemakers. As most current defibrillators have the capacity to pace, transcutaneous pacemakers are generally used in an arrest situation.

## SPECIAL CONSIDERATIONS

While not common, there are some clinical presentations that require special considerations in a cardiac arrest scenario: these include pregnancy, electrical injuries, and drowning. The principles of airway, breathing and circulation remain the same, although modifications must be made because of the physiological changes that occur.

**Pregnancy.** Cardiac arrest in a pregnant woman occurs only once in every 30,000 late pregnancies.<sup>74</sup> Precipitants include pulmonary embolism, trauma, peripartum haemorrhage, amniotic fluid embolism, eclamptic seizure, congenital and acquired cardiac disease, myocardial infarction, subarachnoid haemorrhage and cerebral aneurysm.<sup>74</sup> Haemorrhage, thromboembolism and hypertension are the commonest causes.<sup>74</sup> Regardless of the aetiology, resuscitation in late pregnancy is often unsuccessful.<sup>75</sup> Hence, timely delivery by

TABLE 18.8 Medications

| Action   | Dose   |  |  | Adverse events   |
|--|--|--|--|--|
|  | Indications  | Adults   | Paediatric   |  |
| <p><b>Adrenaline</b> is a catecholamine that increases aortic diastolic pressure and coronary artery perfusion by producing arteriolar vasoconstriction. Traditionally the first-line medication for the treatment of VF and refractory VT, adrenaline has not demonstrated improved outcomes after cardiac arrest and has been associated with postresuscitation myocardial dysfunction.<sup>48</sup></p> | <p>VF and pulseless VT resistant to the three initial counter shocks. PEA and asystole.</p>  | <p>Initial dose of 1 mg, repeated every 3 minutes in adults.<sup>7</sup></p>   | <p>10 µg/kg for the first dose, then 10 µg/kg for subsequent doses. 100 µg/kg via ETT.<br/><i>Periarrest:</i> Infusion 0.1–0.2 µg/kg/min.</p>  | <p>Tachyarrhythmias; hypertension; coronary vasoconstriction; increased myocardial oxygen consumption.</p>   |
| <p><b>Vasopressin</b> increases coronary perfusion pressure and end-organ perfusion more effectively than adrenaline.<sup>125</sup> It has the advantage of being more effective than other vasopressors in the acidaemic environment of a prolonged cardiac arrest.<sup>1,125</sup><br/>Note: not recommended for use in Australia.</p>   | <p>Alternative to adrenaline in refractory VF/VT.<sup>36</sup></p>   | <p>A single 40-unit dose.</p>  | <p>0.5–0.8 µg/kg</p>   | <p>Excessive vasoconstriction resulting in hypoperfusion of the gut and skin.<sup>126</sup></p>  |
| <p><b>Amiodarone</b> directly affects smooth muscle and blocks calcium channels and alpha-adrenergic receptors, resulting in coronary and peripheral arterial vasodilation and a reduction in afterload and systemic blood pressure.<sup>127</sup></p>   | <p>VT/AF refractory to three shocks.<sup>7</sup><br/>Polymorphic VT and wide complex tachycardia of uncertain origin.<br/>Control of haemodynamically stable VT when cardioversion unsuccessful (in the presence of LV dysfunction).<br/>Adjunct to electrical cardioversion of SVT.<br/>Prophylaxis of recurrent VF/VT.<sup>7</sup></p> | <p>Initial bolus dose of 300 mg in 20 mL dextrose. A further 150 mg could be considered for refractory cases.<br/><i>Periarrest:</i> An infusion of 15 mg/kg over 24 hours may be commenced.<sup>7</sup></p> | <p>Initial dose of 5 mg/kg bolus over 2 minutes, which may be repeated to a maximum of 300 mg.<sup>7</sup><br/><i>Periarrest:</i> IV infusion 5–15 µg/kg/min as continuous infusion (max of 1.2 g in 24 h).<sup>32</sup></p> | <p>Vasodilation and hypotension, bradycardia, heart block.<sup>7</sup><br/>May have negative inotropic effects.<br/>Use with caution in renal failure.<br/>Avoid use in torsades de pointes and other causes of prolonged Q-T.</p> |

TABLE 18.8 Medications—cont'd

| Action  | Dose  |   |  |
|---|---|---|--|
|   | Indications   | Adults  | Paediatric   |
| <p><b>Lignocaine</b> suppresses discharge from ectopic foci by blocking sodium channels (see Ch. 10). Impulse formation in the SA node is suppressed and conduction below the bundle of His is impeded; these actions inhibit the formation of re-entrant circuits that lead to VT or VF.</p> | <p>VF and pulseless VT refractory to defibrillation and adrenaline.<sup>7</sup></p>   | <p>Bolus of 1 mg/kg at a rate of 25–50 mg/min. <i>Periarrest</i>: May be followed by an additional bolus of 0.5 mg/kg.</p>  | <p>Initial dose of 1 mg/kg IV or IO; 2–3 mg/kg via ETT. Considered inferior to amiodarone.</p>   |
| <p><b>Magnesium</b> is a major intracellular cation resulting in smooth muscle relaxation and membrane stabilisation.</p>   | <p>Torsades de pointes with or without a pulse; cardiac arrest associated with digoxin toxicity. Failure of defibrillation and adrenaline to reverse VF and pulseless VT. Documented hypokalaemia or hypomagnesaemia.<sup>7</sup></p> | <p>Bolus of 5 mmol. <i>Periarrest</i>: May be followed by infusion of 20 mmol infused over 4 hours.<sup>7</sup> Magnesium sulphate 2.5 mL = 5 mmol. Magnesium chloride 5 mL = 5 mmol.</p> | <p>IV or IO bolus of 0.05–0.1 mmol/kg. Hypotension with rapid administration. Use with caution if renal failure present. Muscle weakness, paralysis and respiratory failure.</p>   |
| <p><b>Atropine</b> is a parasympathic blocking agent (antagonising the action of the vagus nerve) resulting in acceleration of the sinus and atrial pacemakers and AV conduction.</p>   | <p>Symptomatic bradycardia and asystole.</p>  | <p>Bolus of 1 mg, to a maximum of 3 mg.<sup>7</sup></p>   | <p>20 µg/kg via IV or IO. 30 µg/kg via ETT. Tachycardia and excitement.</p>  |
| <p><b>Calcium</b> is essential to nerve and muscle impulse formation and excitation.<sup>7</sup></p>  | <p>Hypocalcaemia, hyperkalaemia, overdose of calcium blockers.<sup>7</sup></p>  | <p>A bolus dose of 5–10 mL 10% calcium chloride (6.8 mmol).<sup>7</sup></p>   | <p>0.2 mL/kg 10% calcium chloride, or 0.7 mL/kg 10% calcium gluconate via IV or ETT (20 mg/kg).<sup>7</sup> Calcium is incompatible with a range of medications and may precipitate in IV lines. Tissue necrosis with extravasation may occur.</p> |

(Continued)

TABLE 18.8 Medications—cont'd

| Action  | Indications  | Dose  |  | Adverse events   |
|---|--|---|--|--|
|   |  | Adults  | Paediatric   |  |
| <b>Sodium bicarbonate (NaHCO<sub>3</sub>)</b> is an alkaline agent that may be used to correct an acidosis associated with SCA. | Correcting a metabolic acidosis (pH <7.1), or base deficit of ≤10 or after 15 min; pre-existing hyperkalaemia; tricyclic antidepressant overdose and urinary alkalimisation in overdose; or hypoxic lactic acidosis. | A bolus dose of 1mmol/kg administered over 2–3 min. As NaHCO <sub>3</sub> is incompatible with many medications, it should be administered by a separate line or flushed before and after administration. | 1 mmol/kg via IV or IO administered once adequate ventilation and ECC are established. <sup>7</sup>  | Should not be routinely administered. Alkalosis, hypernatraemia, hyperosmolality, paradoxical cerebral acidosis, depressed cardiac contractility and metabolic acidosis. |
| <b>Potassium</b> is an electrolyte essential for cell membrane stabilisation that is occasionally used in ALS.                  | Persistent documented VF, suspected hypokalaemia or hypomagnesaemia, and cardiac arrest associated with digoxin toxicity.  | 5 mmol via slow bolus.  | 0.05 mmol/kg via slow administration IV or IO.<br><i>Periarrest:</i> 0.2 mmol/kg/h as a continuous infusion; dilute with at least 50 times its volume and mix well, as can be fatal. <sup>32</sup><br>0.2–0.5 mmol/kg/h to a maximum of 1 mmol/kg if hypokalaemia severe but not immediately life-threatening. | Hyperkalaemia with bradycardia, hypotension with possible asystole, and extravasation may lead to tissue necrosis.   |

ARC = Australian Resuscitation Council; ECC = external cardiac compression; IO = intraosseous; PEA = pulseless electrical activity; SI = sinoatrial; SVT = supraventricular tachycardia; VF = ventricular fibrillation; VT = ventricular tachycardia.

caesarean section in the setting of maternal cardiac arrest may save both infant and mother.<sup>76</sup>

The principles of airway, breathing and circulation remain the same, but modifications must be made because of the physiological changes that occur with normal pregnancy.<sup>7,74</sup> A number of factors need to be considered when resuscitating a pregnant woman. Any situation that affects haemodynamic status will be exacerbated in a supine position,<sup>74</sup> as autocal compression may result in a fall in cardiac output of up to 25%. A patient should be placed in the left lateral tilt (15 degrees) or supine with a pillow under the right buttock, to displace the uterus from the inferior vena cava, facilitating venous return and cardiac output.<sup>7,77</sup> The uterus may also be manually and gently displaced to the left.

While ventilation:compression ratios remain the same for a pregnant woman, chest compression may be complicated by flaring of the ribs, raised diaphragm, obesity and breast hypertrophy.<sup>7,74</sup> To adjust for the shifting of pelvic and abdominal contents towards the head, chest compressions should be performed higher on the sternum. There are, however, no guidelines on how far the compression point should be shifted, so pulse checks during chest compression are used to monitor cardiac output.<sup>77</sup>

The superior displacement of stomach contents by the gravid uterus and a relaxed cardiac sphincter<sup>74</sup> contribute to an increased risk of gastric aspiration in the pregnant woman.<sup>7</sup> Because of this increased risk, cricoid pressure should be applied until after the airway is protected by a cuffed tracheal tube.<sup>77</sup> Tracheal intubation should be attended to early, utilising a short-handled laryngoscope<sup>74,77</sup> or with a blade mounted at more than 90 degrees,<sup>77</sup> as airway anatomy is altered with the larynx more anterior and superior, while pharyngeal mucosa is slightly oedematous and friable.<sup>74</sup> Defibrillation energy, drug doses and administration are in accordance with ALS guidelines.<sup>74,77</sup>

If maternal cardiac arrest occurs in the labour ward, operating room or emergency department and BLS and ALS measures are unsuccessful, the uterus should be emptied by surgical (scalpel) intervention within 4–5 minutes.<sup>77</sup> Maternal resuscitation may not be possible until the fetus is removed. Successful resuscitations have occurred after prompt surgical intervention.<sup>77</sup>

**Electrical injuries.** Electrical burn injuries (EBIs) and lightning injuries are similar in that they occur infrequently, commonly cause widespread acute and delayed tissue damage, and can arrest the heart and respiratory centre. Burn injuries are discussed in Chapter 17. This section focuses on the cardiac arrest situation. High-voltage electrocution is associated with a high incidence of cardiac abnormalities, including arrhythmias, ST and T wave changes, and myocardial infarction. Left ventricular failure can occur hours or days after the event, despite the presence of minimal initial ECG changes.<sup>78</sup> The commonest cause of death with lightning injury is cardiac arrest caused by VF or asystole.<sup>79,80</sup> Because of the potential for cardiac injuries, all patients should be admitted for cardiac monitoring.

A lightning strike may result in asystole followed by spontaneous return of circulation. If ventilation is initiated early and severe hypoxia does not ensue, a patient's chance

of recovery should be better.<sup>80</sup> Initial response of BLS should always begin with D (danger)—avoidance of injury to the rescuer. Ensure that the environment is safe for rescuers by disconnecting the electrical supply, where possible, without touching the patient. Where high-voltage lines (power lines) are in contact with the person or the vehicle, no attempt should be made to extricate the person from the vehicle until the situation is deemed safe by an authorised electricity supply person.<sup>7</sup> Once the environment is safe, commence BLS resuscitation. The neck and spine should be protected, as there may be trauma.

In lightning victims, emphasis is on the immediate resuscitation of those who appear unresponsive. Respiratory arrest may be prolonged due to paralysis of the medullary respiratory centre; if not corrected, cardiac arrest secondary to hypoxia ensues. Fixed, dilated pupils should not be used as a poor prognosis of outcome, as victims can benefit from prolonged resuscitation without major sequelae.<sup>80</sup>

**Drowning.** General issues in managing drowning presentations are discussed in Chapter 16. This section focuses on resuscitation of a cardiorespiratory arrest. Hypoxia and acute lung injury (ALI) from near-drowning results in respiratory arrest, which if not corrected may proceed to a cardiac arrest.<sup>81</sup> A patient's emotional state, associated diseases, previous hypoxia and water temperature all influence this progression.<sup>82</sup>

The primary goal of initial intervention is the relief of hypoxia, restoration of cardiovascular stability and prevention of heat loss.<sup>81</sup> Resuscitation of drowning victims follows BLS guidelines, with commencement as soon as practical. As drowning victims may have swallowed considerable amounts of water, vomiting and aspiration of gastric contents can be a major problem during resuscitation.<sup>7</sup> To minimise the risks of inhalation, abdominal compression, the Heimlich manoeuvre and attempts to drain water from the lungs are not recommended. Instead the victim should be placed on the side for the initial assessment of airway and breathing.<sup>7</sup> Attempts to rewarm the victim should be commenced immediately.<sup>81</sup> As patient outcome is improved the earlier resuscitation is initiated, in-water resuscitation is receiving considerable support.<sup>82,83</sup> Rescuers in these circumstances must consider their swimming ability and the distance to shore. Once experienced personnel arrive, ALS and administration of oxygen should be initiated. (The principles of respiratory support and ventilation are discussed in Chapter 11, and treatment of the sequelae of a drowning victim is discussed in Chapter 16.)

## EVALUATION DURING RESUSCITATION

Maintenance of an effective cardiac output during CPR is evaluated by palpating the carotid or femoral pulse in adults (brachial in children); this was once the gold standard for assessing circulation. However, neither lay persons nor professionals can rapidly (in less than 10 sec) and accurately perform this step.<sup>9</sup> Pulse checks are not recommended for evaluation after defibrillation until 2 minutes of CPR have been performed, regardless of the rhythm postdefibrillation.<sup>8</sup>

The use of capnometry as a non-invasive technique for monitoring CPR's effectiveness is recommended.<sup>7</sup> As

partial pressure of end-tidal carbon dioxide (PETCO<sub>2</sub>) concentration correlates with pulmonary blood flow during CPR, the adequacy of resuscitation efforts is evaluated by measuring this parameter. PETCO<sub>2</sub> also correlates with cardiac output, return of spontaneous circulation (ROSC) and outcomes in cardiac arrest.<sup>84</sup> A mean PETCO<sub>2</sub> of 17 mmHg or above has been associated with survival from cardiac arrest, while a mean PETCO<sub>2</sub> <10 mmHg is associated with poor outcomes.<sup>85–87</sup> A rise in PETCO<sub>2</sub> during CPR may indicate the return of spontaneous circulation.<sup>88</sup> Conversely, experimental studies have demonstrated that cardiac arrest from massive pulmonary embolism is associated with an extremely low PETCO<sub>2</sub> reading during CPR.<sup>89</sup> Having noted this, hyperventilation during CPR is not recommended and may be harmful.<sup>7,8</sup> Similarly, animal studies indicate that hyperventilation is associated with raised thoracic pressure, decreased coronary and cerebral perfusion and reduced return of spontaneous circulation.<sup>7</sup> Clinical studies show that rescuers consistently hyperventilate patients during a cardiac arrest.<sup>90</sup>

## ROLES DURING CARDIAC ARREST

Resuscitation teams should be organised to ensure that the individual skills of each member are used effectively and efficiently.<sup>79</sup> The exact composition of the resuscitation team will vary between organisations, but generally the team should possess the following skills:<sup>91</sup>

- advanced airway management and intubation skills;
- intravenous access skills including central venous access;
- defibrillation and external pacing abilities;
- medication administration skills; and
- postresuscitation skills.

As members of a resuscitation team in the hospital generally do not work together but come from all areas of the hospital, the team should have a designated leader. The team leader gives direction and guidance, assigns tasks, and makes clinical decisions without directly performing specific procedures.<sup>8,79</sup> The leader should engender the team's trust. Where leaders initiate structure within the arrest team, members not only work together better, they also perform the tasks of resuscitation quicker and more effectively.<sup>79</sup> The leader nominates the roles of arrest team members. Roles of team members include airway management, chest compression, medication administration (including IV access), documentation of events and care of family members. The team leader should be responsible for postresuscitation transfer, documentation, communicating with family members and healthcare professionals, and debriefing of the team.<sup>91</sup>

The resuscitation scenario is both complex and stressful for all participants. Often, participants express feelings that too many people are involved, with no one person in control. Unfortunately, the concept of the multidisciplinary team, where all members' contributions are equally respected, is

often not evident in the literature.<sup>92</sup> In addition, while nurses already present at a cardiac arrest in the hospital setting may be willing and competent to perform CPR, they may be prevented from doing so because of the arrival of the cardiac arrest team.<sup>93</sup>

## FAMILY PRESENCE DURING AN ARREST

The practice of family members witnessing resuscitation has over time become more evident, both in practice and in the literature. This shift in practice has been attributed to increasing patient autonomy and the presence of family at a cardiac arrest in popular television shows.<sup>100</sup> This has contributed to family members requesting and expecting to be present. However, the issue of whether the family should be present during a cardiac arrest remains controversial. Proponents argue the importance of family being with loved ones during their last moments, as this shortens the period of grieving and provides closure.<sup>100,101</sup> Indeed, paediatric advanced life support (PALS) guidelines recommend family presence in paediatric resuscitation.<sup>10</sup> However, medical personnel are not so supportive of this recommendation. Commonly cited is concern that the family may interrupt the work of the resuscitation team, the ethical and medicolegal implications, or concern about offending the family.<sup>102,103</sup>

Conflicting evidence exists as to the psychological effects of such an event on the family. Effects have been documented as ranging from no adverse effects<sup>100</sup> through to expressions of distress, haunting consequences and trauma.<sup>101</sup> Where family are provided the option of being present, a staff member should be identified to have sole responsibility of supporting the family.

## CEASING CPR

The decision to cease CPR is often difficult; continuing CPR beyond 30 minutes without return of spontaneous circulation (ROSC) is usually futile unless the arrest was compounded by hypothermia, submersion in cold water, lightning strike, drug overdose, or other identified and treatable conditions such as intermittent VF/VT.<sup>8</sup> Prolonged resuscitation of greater than 60 minutes may be made for a severely hypothermic, child victim of near-drowning. Pupillary signs should not be used as a predictor of outcome in infants and children, as 11%–33% of children with non-reactive pupils have survived long-term after CPR.<sup>10</sup> It is important to have eliminated all causes as far as possible.

Termination of resuscitation is a multifactorial process, influenced by provider comfort and experience, patient prognosis, desires previously expressed, wishes and values, the culture of the hospital, the EMS or emergency department, protocols and resource issues, and national and international guidelines that reflect changing standards of care, resource availability, global interpretations of utility and emerging

science.<sup>20</sup> With scientific advances and evidenced-based protocols becoming more widely implemented, current impressions of termination decisions will change over time.<sup>20</sup> It is appropriate to invite suggestions from team members, to ensure that all members are comfortable with a decision to stop the resuscitation attempt.<sup>8</sup> Ultimately, terminating CPR is equivalent to a determination of death, and must be made by a physician. In some out-of-hospital circumstances it may be the paramedical staff that make this decision regarding stopping CPR.

## POSTRESUSCITATION PHASE

As noted earlier, the aim of resuscitation is maintenance of cerebral and myocardial perfusion and the return of a patient to a state of best possible health.<sup>9</sup> Resuscitation does not cease with the return of spontaneous circulation. However, the ROSC after cardiac arrest does not always equate to a positive outcome for the patient. Cardiac arrest with widespread cerebral ischaemia often leads to severe neurological impairment. Only 25% of patients admitted to the ICU after a cardiac arrest will be discharged with good neurological function.<sup>104</sup>

### Role of hypothermia in adults after cardiac arrest

During cardiac arrest, prolonged global ischaemia coupled with inadequate reperfusion during the immediate postresuscitation period can lead to severe cerebral hypoxic injury.<sup>105</sup> Induced moderate hypothermia (28°–32°C) has been used in open-heart cardiac surgery since the 1950s to protect the brain against global ischaemia.<sup>106</sup> Several recent studies have shown that cooling patients post-cardiac arrest provides significant survival benefit as well as improved cardiac and neurological function.<sup>106–108</sup> Prospective randomised studies have demonstrated that mild hypothermia (32°–34°C) increases the rate of favourable neurological outcome in comatose adult patients resuscitated after out-of-hospital cardiac arrest (OHCA) due to VF.<sup>107,108</sup> A variety of cooling techniques are described in Table 18.9.

**TABLE 18.9 Cooling techniques post-cardiac arrest<sup>106–108</sup>**

|                       |   |
|-----------------------|---|
| <b>External</b>       | Cooling blankets/mattress, ice packs, wet towels, fanning and cooling helmets   |
| <b>Internal</b>       | <ul style="list-style-type: none"> <li>• IV administration of saline (30 mL/kg at 4°C to achieve a 1.5°C fall in core temperature)</li> <li>• IV heart exchange device</li> <li>• Peritoneal and pleural lavage (not generally used)</li> </ul> |
| <b>Extracorporeal</b> | Effective but very invasive   |

Based on current evidence, ILCOR recommends that unconscious adult patients with spontaneous circulation after OHCA be cooled to 32°–34°C for 12–24 hours if the initial rhythm was VF. This cooling may also be beneficial for other rhythms or in-hospital cardiac arrest.<sup>106</sup> It is important to note that shivering must be prevented during this phase. Hyperthermia occurring in the first 72 hours after a cardiac arrest should be treated with antipyretics or active cooling.<sup>7,8</sup>

## NEAR-DEATH EXPERIENCES

With the rise in survival rates after a critical illness, there are increasing numbers of documented near-death experiences (NDEs).<sup>94,95</sup> Near-death has been described as unusual experiences during a close brush with death.<sup>94</sup> Experiences have typically included memories of bright tunnels of light, deceased relatives, out-of-body sensations, feelings of presence of deity, and peace.<sup>96,97</sup> Typically, people report the experiences as pleasant, and they have resulted in positive life changes for the individual. After-effects of an NDE include absence of fear of death, more spiritual view of life, less regard for material wealth, or a heightened chemical sensitivity.<sup>95,96</sup> The incidence of NDEs after cardiac arrest is reported at 6%–18%,<sup>94,98</sup> with the frequency generally being higher in people under 60 years of age.<sup>95</sup> Hence, an awareness of the incidence of NDEs, and the sensitivity and needs of the person with an NDE are essential post-cardiac arrest.<sup>96,99</sup>

## LEGAL AND ETHICAL CONSIDERATIONS

Burgeoning technology in the 1960s enabled the support of oxygenation and circulation for people whose illnesses would have been lethal just a few years before. Enthusiasm for restoration of life led healthcare workers to routinely initiate CPR for all patients who died in hospital.<sup>110</sup> Unfortunately, this led to inappropriate resuscitation attempts and the realisation of the economic, medical and ethical burden to society when survivors had a resulting poor quality of life.<sup>111</sup> In the 1970s, growing concern about inappropriate application of CPR and patient's rights led authors to suggest means of forgoing resuscitation and involving patients in decision making.<sup>112</sup> Traditionally, the decision to initiate or withhold CPR was often made by the treating medical team in the absence of the patient or family.<sup>113</sup>

Hospitals responded by developing procedures for withholding CPR through the documentation of do not attempt to resuscitate (DNAR) orders, advance directives or living wills<sup>113</sup> (see Chapter 6). For patients or their surrogates to meaningfully participate in decision making about CPR, they must have some understanding of survival rates and adverse effects associated with CPR.<sup>114</sup> Consequently, much debate has ensued over the right of a person to forgo treatment.<sup>110</sup>

International studies show that while patients want to be involved in CPR decision making and want some form of advance directive, their knowledge is limited and often derived from television dramas.<sup>113-115</sup> Understanding of morbidity and outcomes after CPR strongly influences their preferences.<sup>114</sup> Most patients, and indeed healthcare workers, commonly hold unrealistic expectations of CPR success,<sup>116</sup> and will often reverse their preference for commencing CPR once they are informed of the true probability of survival and functional status after resuscitation.<sup>114</sup> Regardless of this, healthcare workers continue to demonstrate a reluctance to discuss CPR options with patients. Despite open discussion, poor documentation and communication can result in CPR being inappropriately commenced.<sup>117</sup> Approximately one-third of patients successfully resuscitated have subsequently stated that they did not want to be resuscitated.<sup>116</sup> Conversely, and contrary to medical and nursing opinions, some people choose CPR even when they have a terminal illness, coma or serious disability.<sup>114</sup>

With the exception of a zero survival rate there remains no formal consensus on DNAR decision-making practices

or the termination of resuscitation.<sup>118</sup> While researchers have attempted to develop prognostic indicators for cardiac arrest outcome, moralists would argue that the use of such prognostic tools alone reflect utilitarianism,<sup>116</sup> and should never be used in isolation of the input of the patient and healthcare team.<sup>118</sup>

## SUMMARY

Outcomes for patients after in-hospital sudden cardiac arrest remain poor. Successful management of a patient following SCA depends largely on the timely implementation of the chain of survival. Nurses should understand the role of the chain of survival in the resuscitation of the person following cardiac arrest. The chain emphasises the importance of early recognition and intervention, continuous uninterrupted compressions and the early use of the defibrillator as a BLS skill. Despite the plethora of research on the topic of resuscitation, there is much we still do not know.

### Clinical case study

Mr B, a previously fit and well 16-year-old, was admitted to the ICU after a cardiac arrest. Students at his school saw him collapse during a school cross-country run. Teachers and students at the scene reported that Mr B was unresponsive, not breathing and with no palpable pulse detected. BLS was immediately initiated by attending teachers. The ambulance personnel arrived on scene minutes later to find the patient unresponsive, with absent pulse and in ventricular tachycardia.

Mr B was defibrillated (monophasic) at 360 J, but failed to revert to a perfusing rhythm. Ambulance officers then intubated him with a size 7.5 ETT, inserted a cannula and administered 1 mg IV adrenaline every 3 minutes, commencing the first dose just prior to the delivery of the third shock. There was further defibrillation at 360 J and 360 J before Mr B reverted to sinus rhythm, 35 minutes after the initial collapse.

On arrival at the emergency department (ED), he displayed a flexion response to noxious stimuli, and his pupils were 2 mm and reactive. The ECG showed sinus rhythm at a rate of 60 beats/min, BP 90/50 mmHg, SpO<sub>2</sub> 89%, and temperature of 36° C. Mr B was placed on an

oxylog ventilator (FiO<sub>2</sub> 1.0, rate 12 breaths/min, EMV 5760 mL/min) and preparations were made to transfer him to the ICU.

On arrival in the ICU, Mr B experienced a sudden deterioration, with difficulty ventilating. The high inspiratory pressure alarm on the ventilator activated continually and his SpO<sub>2</sub> fell to 50%. Rapid primary assessment of ABC revealed absence of chest movement and breath sounds to the left lung. Despite the rhythm appearing as sinus at a rate of 55, the carotid and peripheral pulses were absent. On auscultation and palpation his BP was unrecordable. CPR was quickly reinitiated and adrenaline 1 mg administered IV. A tension pneumothorax was diagnosed and the left lung decompressed using a large-bore peripheral cannula, followed by the insertion of an intercostal catheter (ICC). Baseline ECG showed Q-T prolongation (500 msec). Mr B was weaned and extubated over the next 6 hours and the ICC was removed the following day. Mr B was transferred to the coronary care unit for cardiac monitoring and eventually discharged from hospital with no physical or neurological deficits. The diagnosis was long Q-T syndrome.

### Research vignette

Fulbrook P, Albarran J, Latour J. A European survey of critical care nurses' attitudes and experiences of having family members present during cardiopulmonary resuscitation. *Int J Nurs Stud* 2005; 42(5): 557-67 (published with permission).

#### ABSTRACT

This paper presents the results of a survey into the

experiences and attitudes of 124 European critical care nurses to the presence of family members during cardiopulmonary resuscitation (CPR). Nurses from mainland Europe were less experienced and less sure about the consequences of relatives witnessing resuscitation than UK nurses. Generally, nurses supported the presence of family members, although UK nurses held significantly more positive attitudes than their non-UK counterparts in the areas of decision making, processes and outcomes of

resuscitation. Differences in attitude are explored in the discussion. On the basis of results from this study, it is recommended that further policy guidance is required.

### CRITIQUE

This quantitative study used self-administered questionnaires to investigate the experiences and attitudes of European critical care nurses to the presence of family members during CPR. Justification for the study was the paucity of information on the topic from the perspective of the critical care nurse. The aim of the study and the research questions were clearly stated. A descriptive exploratory design was used to answer the research questions. The purpose-designed questionnaire was administered to a convenience sample of critical care nurses attending the 2002 European Federation of Critical Care Nursing Association's conference in Paris. All 235 delegates at the conference were invited to participate, and daily announcements were made to remind participants to return completed forms. Given the multilingual nature of the conference and to further enhance the response rate, the questionnaire was made available in four different languages. The data collection instrument asked demographic information and elicited information about participants' actual experience with family witness resuscitation (FWR). The subsequent attitudinal section utilised a five-point Likert scale to elicit participants' attitudes on decision making, and the process and outcomes of CPR.

Methodological weakness detracted from the rigour of this study. First, a pilot test of the developed questionnaire prior to implementation would have enhanced the reliability of results. Of the 235 questionnaires administered during the conference, 130 were returned, and 6 were subsequently excluded as the respondents were from outside Europe (a final response rate of 53%). Almost half the participants (n=54; 43.5%) were from the United Kingdom, 14.5% (n=18) from Denmark, 12% from Sweden (n=15), 9% from Norway (n=11), and the remaining 21% (n=26) from 11 other European countries. Random distribution of the questionnaire to critical care nurses from across Europe would have enhanced the research and allowed generalisation of the findings. The authors, however, rightly admitted the limitations of convenience sampling

and justified the method by identifying that the study purpose was not to generalise but to provide a 'snapshot' of the views of European nurses. The authors also argued that this approach was practical, inexpensive, and provided a quick means of accessing a large number of participants. A second limitation of the select population is that only those with strong opinions either for or against FWR might have completed the survey.

In general, European nurses in this study were committed to FWR, with almost half the sample reporting having experiences of FWR. Aside from the lack of generalisability of findings, the data-collection method introduced some confounding issues. Only 60% of the sample were currently engaged in direct patient care, and the majority of participants had more than 10 years' critical care experience. The findings may not reflect the views of nurses actually in a position to engage in FWR. Consistent with previous studies, participants were, however, reluctant to invite family members to be present. The authors concluded that the collaborative decision-making processes in these areas or absence of policy might have contributed to this observation. Only 38% (n=47) of participants agreed that family members should have the option of being with their family during resuscitation, and a third of nurses actually wanted the family to be present. Reasons for not wanting the family present included issues of patient confidentiality, and fear of litigation or that the family might argue with the resuscitation team.

While not the intention of the study, comparisons were made between UK and non-UK participants. UK nurses in the study held significantly more positive attitudes in the areas of decision making, processes and outcomes of FWR.

The paper was well written, clearly introduced the research questions, and extensively described the data-collection method. Concerns about the data-collection method were addressed. The authors concluded that, in view of recent practice developments and increasing consumer requests to be present during FWR and the fact that nurses remain the main target for such family requests, critical care nurses should collaborate with healthcare members to develop and implement written guidelines on FWR.

### Learning activities

*Learning activities 1–5 relate to the clinical case study.*

1. Discuss the management of this patient in relation to the ALS flowchart.
2. Discuss the tidal volume of this patient during management of the cardiac arrest.
3. Identify potential causes of PEA.
4. Discuss the pathophysiology of PEA in relation to the case study.
5. Outline the role of calcium in the management of PEA.
6. Outline the role of magnesium and potassium in the short-term management of torsades de pointes.

## Online resources

- Australian Resuscitation Council (ARC), <http://www.resus.org.au/>
  - European Resuscitation Council (ERC), <http://www.erc.edu/>
  - American Heart Association (AHA), <http://www.americanheart.org/>
  - New Zealand Resuscitation Council (NZRC), <http://www.nzrc.org.nz/>
- The sites above (ARC, AHA, ERC, NZRC) aim to foster a coordinated approach to the practice and teaching of resuscitation. These sites are useful for accessing policy, future conferences, www links and newsletters.
- The Regional Emergency Medical Services Council of New York City, <http://www.nycremsco.org/default.asp>
- This American site offers BLS and ALS protocols with a focus on the prehospital environment.
- Center for Pediatric Emergency Medicine (CPEM), <http://www.med.nyu.edu/peder/cpem>
- The CPEM aims to improve emergency medical services for children. This site offers PALS teaching resources and www links.

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