RESUSCITATION

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INTRODUCTION

The patient with sudden cardiac arrest requires a bystander to initiate a number of actions in rapid sequence for there to be any hope of successful resuscitation. These steps are known as the ‘chain of survival’. After the first step, a call to ambulance, the bystander needs to institute basic life support (BLS) whilst awaiting the arrival of the emergency medical services (EMS). The BLS procedures may be undertaken by personnel with little or no medical training and are applicable in the patient who has become unconscious as a result of airway obstruction, respiratory arrest or cardiac arrest. In general, BLS includes interventions that involve minimal training in the use of equipment, but also now include the application of a semi-automatic external defibrillator (SAED), if one is available close to the site of the cardiac arrest.

DEVELOPMENT OF PROTOCOLS

It is important that the guidelines for BLS be nationally consistent. To achieve this, many countries have established national expert committees to advise the community, ambulance services and medical profession on BLS guidelines. Table 1.1.1 shows the national associations that make up the International Liaison Committee on Resuscitation (ILCOR). This group meets every 6 years to review the BLS guidelines and to consider changes to these guidelines. The most recent revision of BLS guidelines occurred in 2000. Subsequently, each national committee may determine regional variations to these guidelines to take into account local practices.

One of the major considerations of changes to protocols for these committees is the feasibility of these protocols to be implemented by personnel with minimal training. The major handicap to consideration of the protocols is the relative paucity of good scientific evidence for the strategies that have been taught over many years.

| Table 1.1 |
| American Heart Association |
| Australian Resuscitation Council |
| European Resuscitation Council |
| Heart and Stroke Foundation of Canada |
| Inter-American Heart Foundation |
| New Zealand Resuscitation Council |
| Resuscitation Council of Southern Africa |

INITIAL EVALUATION

A flow chart for the initial evaluation of the collapsed patient is shown in Figure 1.1.1. This flow chart commences with the recognition that a patient has collapsed. The initial steps are as follows:

Check for dangers

As the patient is approached, the bystander should immediately consider any dangers that may be associated with...
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the collapse of the patient. For example, the patient may have been electrocuted and there could be further casualties if the power source is not switched off prior to patient contact.

In the case of a motor-vehicle accident where a patient is unconscious, there is the risk of additional injury from further collisions involving passing vehicles and another bystander should be tasked to direct traffic at the scene. There may also be a risk of fire if fuel has leaked onto hot engine parts or there is an electrical fault. Therefore, the ignition should be switched off, and it is advised that unconscious patients should be immediately removed from vehicles prior to the arrival of emergency medical services, whilst taking care to minimize movement of the neck. It is considered that the risk of injury from a sudden fire or explosion exceeds that of moving an unconscious patient prior to immobilization of the cervical spine with a hard collar.

In the case of a patient who has collapsed in a confined space, the possibility of poisoning with carbon monoxide or a similar toxic gas should be considered, and the scene not entered until it can be made safe by emergency services.

Check for response
The patient who has collapsed must be quickly assessed to determine whether there is coma, indicating possible cardiac arrest or just a simple fall. This is assessed by a gentle ‘shake and shout’, followed by an examination of the motor and verbal response of the patient.

If the patient is unresponsive, cardiac arrest due to ventricular fibrillation should be suspected and emergency medical services telephoned immediately (‘call first’). Alternatively, if the collapse is due to suspected airway obstruction (choking) or inadequate ventilations (drowning, hanging, etc.), then resuscitation should be commenced for approximately 1 minute prior to the calling of emergency medical services (‘call fast’).

Airway
If the unconscious patient has collapsed in a prone position, then he/she should be placed on his/her side in the coma position (see Fig. 1.1.2) and an assessment of the airway should be made. Alternatively, if the patient has collapsed and is supine, the airway may be checked in that position.

One exception to this initial step is if the unconscious patient has been retrieved from near drowning. In this setting, the initial step of rolling onto the side to allow assessment of airway and facilitate airway clearance has been practised by surf life-savers for many years and is still recommended by some authorities as the initial position for patient assessment.

The airway is checked with visual inspection, careful forward movement of the jaw (‘jaw thrust’) and sweeping out any foreign material or vomitus with a finger. In general, ‘noisy breathing is obstructed breathing’, although an exception to this rule might include the patient with severe asthma who has such a low tidal volume that there are no sounds from the upper airway.

If the airway is obstructed with a foreign body, there are a number of manoeuvres that have been described as helpful in this setting. In North America, the Heimlich manoeuvre is endorsed as the technique of choice. However, this technique is associated with some significant complications, including intra-abdominal injuries. In Australia, the recommended technique for clearing an airway that is completely obstructed by a foreign body includes back blows and lateral chest thrusts.

In a study comparing standard chest compressions and Heimlich manoeuvre on cadavers with a simulated complete airway obstruction, the mean peak airway pressure was significantly lower with abdominal thrusts compared to chest compressions. This study concluded that standard chest compressions have the potential of being more effective than the Heimlich manoeuvre for the management of complete airway obstruction by a foreign body in an unconscious patient.

Breathing
Adequate respirations are assessed by visually inspecting movement of the chest wall. In cases of cardiac arrest, infrequent, deep (agonal) respirations may continue for some minutes.

If the patient is found to have inadequate or absent breathing on initial assessment, then expired air resuscitation, mouth-to-mask or assisted ventilation with a bag/valve/mask will be required. On the other hand, if the initial assessment of the unconscious patient reveals
adequate respirations, the victim should be turned and maintained in a recovery position.

Circulation

Previously, it was recommended that a bystander attempt to palpate a pulse in order to diagnose cardiac arrest and, if absent, commence external cardiac massage (ECM). However, it is now recognized that the pulse check is quite inaccurate in this setting. Since cardiac arrest may be presumed if breathing is absent and is very unlikely if breathing is adequate, this step has now been deleted from BLS assessment.

Cardiopulmonary Resuscitation

If cardiac arrest is diagnosed, and the EMS has been summoned, the bystander should now commence CPR, using both expired air resuscitation (EAR) and ECM until a defibrillator arrives.

Expired air resuscitation

Since first described in 1958, EAR has become the standard in BLS for patients who have absent or inadequate respirations. However, there is often considerable reluctance for bystanders to perform EAR due to the perceived difficulty and contact with saliva resulting in the possibility of cross-infection.

It has been demonstrated in animal models that some ventilation occurs during chest compressions and it has been proposed that EAR may be withheld in patients who have cardiac arrest. In a study of 520 patients with pre-hospital cardiac arrest, bystanders were given instructions by ambulance dispatchers to perform EAR plus ECM or ECM alone. There was a trend towards better survival to hospital discharge in the ECM only group compared with the EAR plus ECM (14.6% vs. 10.4%), however, this difference was not statistically significant (P=0.18). In this study, response times for EMS were very short (mean 5 minutes), consequently, the role of EAR in EMS areas where response times are longer remains uncertain.

There are a number of simple pieces of equipment that may be used as an alternative to EAR. These include mouth-to-mask and bag-valve/mask, with or without an oral-pharyngeal airway. This equipment has the advantage that there is no possible cross-infection risk, however, some training in the use of these devices is required. Within BLS, Guidelines 2000 recommends specific tidal volumes in bag-valve mask ventilation. Whichever technique of assisted ventilation is used, adequate tidal volume is assessed by the rise of victim’s chest, whether there is any distension of the stomach, and by listening and feeling for air being exhaled from the patient’s mouth.

The use of supplemental oxygen has previously not been considered as part of BLS, however, some advisory committees within ILCOR are now incorporating oxygen use within BLS training. Although there are little data on the effect on outcome, it is intuitive that supplemental oxygen during CPR would increase the oxygen content of the blood and oxygen delivery to vital organs.

External cardiac compressions

External cardiac massage was first described in 1960. Subsequently, ECM has been adopted as the standard of care for patients with cardiac arrest. However, there is debate as to whether ECM generated blood flow via a ‘cardiac pump’ mechanism or a ‘thoracic pump’. The thoracic pump theory had been supported by early transthoracic echocardiographic studies during CPR showing that the cardiac valves remained open during the relaxation phase of ECM. There had also been observations that ‘cough CPR’ resulted in some blood flow. It was presumed that the changes in intra-thoracic pressure led to forward blood flow and that valves in the venous system prevented back flow.

However, more recent studies of trans-osophageal echocardiography during CPR in humans has found that during the compression phase, the left ventricle is compressed; the mitral valve remains closed and the aortic valve opens only at the end of compression. During the relaxation phase, the mitral valve opens and the left ventricle is filled. These findings suggest that blood flow during ECM is a result of cardiac compression, at least early in the course of ECM.

Whatever the predominant mechanism of blood flow, ECM results in only 15%-20% of cardiac output in the adult, mainly owing to the relative rigidity of the chest wall. Consequently, there is progressive metabolic acidosis during CPR and very few adults survive when ECM has been given for more than 30 minutes.

The current recommended rate for ECM is 100 per minute, to ensure the delivery of a minimum of 60 compressions per minute. The sternum should be depressed at least 4 cm in the adult with compression being approximately 50% of the cycle. Pauses in chest compressions result in a prolonged decrease in mean arterial blood pressure, therefore, it is recommended that two breaths for every 15 compressions be delivered without a pause in chest compressions if there are two rescuers.

Defibrillation

Semi-automatic external defibrillation (SAED) is now considered part of BLS. The SAED devices are extremely sensitive and specific for the correct diagnosis of ventricular fibrillation or ventricular tachycardia and are simple for bystanders to use with minimal training. Following the switching on of the device and the application of the pads, the SAED will request confirmation of coma and absent respirations, and advise the bystander to ‘stand clear’. The bystander is then advised to press a button to deliver a shock.

There are four situations that have been proposed where non-medical personnel could use a SAED. First, the SAED may be used by first responders such as fire services who co-respond with ambulance services. For example, Ontario, Canada, has implemented an extensive programme to introduce rapid defibrillation across that state. The use of fire department first responders resulted in 92.5% of cardiac arrest patients being de-
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fibrillated in under 8 minutes compared with 76.7% under the previous system (P<0.001). Survival to hospital discharge improved from 3.9% (183/4690 patients) to 5.2% (85/1614 patients) (P=0.03). This study demonstrated that an inexpensive, multifaceted system optimization approach to rapid defibrillation can lead to significant improvements in survival after cardiac arrest in a large system. In Melbourne, Australia, a study of a fire-service first responder programme found that the mean time to defibrillation was reduced from a mean of 7.1 minutes for ambulance services to 6.0 minutes for the combined approach.9 However, this study did not have the power to assess the impact on patient outcomes.

Second, the SAED may be placed in a public area for use by designated personnel such as security staff who undergo a short training programme. In places such as casinos10 and a large football stadium,11 this approach has been shown to be effective.

Third, the SAED may be placed in a public area for use by personnel with no previous training at all in the use of an SAED. For example, at Chicago airport, there were defibrillators placed in strategic locations with signs advising of the correct use of the SAED.12 Over a 2-year period, there were 21 patients with cardiac arrests, of whom 18 had an initial rhythm of ventricular fibrillation. A defibrillator was applied by a ‘good Samaritan’ bystander in 14/18 patients with ventricular fibrillation and 11 were successfully resuscitated, with 10 patients alive and well at 1 year.

However, most cardiac arrests occur in the home and it is estimated that the widespread implementation of this approach to all public areas would be very costly and result in relatively few lives saved.13

An SAED may be placed in the home of a patient who is at increased risk of sudden cardiac death for use by a partner or relative who (hopefully) would witness the cardiac arrest. Clinical trials are required to assess the cost-effectiveness of this approach prior to widespread implementation.

SUMMARY

Basic life support for the patient with sudden cardiac arrest has been described as a ‘chain of survival’ and includes recognition of cardiac arrest, a call to emergency medical services, the performance of EAR and ECM, and early defibrillation using a semi-automatic external defibrillator.

Current research explores the cost-effective means of delivering early defibrillation using public access defibrillation by personnel with no previous training, first responders with minimal training such as security personnel, co-responders with ambulance services (such as fire fighters) or even defibrillators in the homes of high-risk patients.

REFERENCES


rate was the sum of the three coefficients, or 5.5% per minute.

The importance of rapid treatment of cardiac arrest has led clinicians to develop a systems management approach, represented by the concept of the ‘chain of survival’, which has become a widely accepted model for the emergency medical services (EMS) systems. The chain of survival idea maintains that more people survive sudden cardiac arrest when a sequence of events occurs as rapidly as possible. This sequence is:

1. Early access to the EMS system
2. Early BLS
3. Early defibrillation
4. Early advanced care

A weakness in any link of the chain reduces the probability of patient survival, and all of the links must be connected. By convention, ALS involves continuation of BLS as appropriate, with the addition of defibrillation, invasive airway and vascular access techniques, and the administration of pharmacologic agents.

AETIOLOGY AND INCIDENCE OF CARDIAC ARREST

The commonest cause of adult sudden cardiac arrest is ischaemic heart disease. Other causes of cardiac arrest include respiratory failure, drug overdose, metabolic derangements, trauma, hypothermia, immersion and hypovolaemia. While ALS guidelines are universally applicable, in these situations specific modifications may be appropriate.

The incidence of sudden cardiac death (within 24 hours of the onset of symptoms) in the USA has been estimated as 1.24/1000/year. In western metropolitan Melbourne, Australia, in 1995 the incidence of cardiac arrest notified to ambulance was approximately 0.72/1000/year. In 20 communities from developed nations worldwide an average of 0.62/1000/year received attempted resuscitation after out-of-hospital cardiac arrest.

ADVANCED LIFE SUPPORT GUIDELINES AND ALGORITHMS

In 1997 the International Liaison Committee on Resuscitation (ILCOR), with delegates from Australia, Canada, Europe, South Africa and the USA published an advisory statement on ALS, The Universal ALS Algorithm, based largely on the belief that valid scientific evidence supports only three interventions as unequivocally effective in adult cardiac resuscitation:

1. Basic CPR
2. Defibrillation if the dysrhythmia is VF or pulseless VT
3. Tracheal intubation

The algorithm (Fig. 1.2.1) recommends a specific sequence in which the above interventions should be performed, and prompts consideration of other therapies and potentially reversible causes of cardiac arrest. It is uncomplicated, concise, easy to memorize and adapt into poster format and is readily applied to the clinical situation. The guidelines and algorithm were ratified by the European Resuscitation Council (ERC) in Copenhagen, in June 1998 and published as The 1998 European Resuscitation Council guidelines for adult advanced life support.

In 2000, the American Heart Association (AHA) convened the International Guidelines 2000 Conference on CPR and Emergency Cardiac Care (ECC). Following this conference, the International Guidelines 2000 for CPR and ECC were developed and published. These guidelines represent a consensus of expert individuals and resuscitation councils and similar organizations from many countries.
cultures and disciplines. The underlying principles that assisted decision-making when developing the guidelines were that additions to existing guidelines had to pass a rigorous evidence-based review, and revisions or deletions occurred because of:

- lack of evidence to confirm effectiveness and/or
- additional evidence to suggest harm or ineffectiveness and/or
- evidence that superior therapies had become available.

The guidelines include a version of the Universal ALS Algorithm and several other algorithms that expand on more specific areas of ALS assessment and management, and are a very valuable and clinically useful adjunct to the Universal ALS Algorithm.5

In 2002 the Australian Resuscitation Council published Protocols for Adult Advanced Life Support, which are succinct and include a slightly modified version of the Universal ALS Algorithm.9

The most exciting and clinically relevant advances in ALS over the last decade have been the development of guidelines and algorithms that are becoming universal in both inclusion of scientifically proven therapies and widespread acceptance, and have substantially simplified the management of cardiac arrest. Nevertheless, our resuscitation knowledge is still incomplete and many of the ALS techniques that we currently use are not supported by scientifically rigorous evidence.10 Rigid adherence to guidelines is neither practical nor advisable and they should be interpreted with common sense. Individuals with specialist knowledge should take the opportunity to modify them according to the level of their expertise and the specific clinical situation or environment in which they practice.

CONFIRMATION OF CARDIAC ARREST RHYTHM AND INITIATION OF ALS

BLS is only a temporary and inefficient substitute for normal cardiorespiratory function. ALS is almost always necessary to produce return of spontaneous circulation (ROSC) after circulatory arrest. The purpose of BLS is to maintain the patient as effectively as possible until drugs and equipment, particularly a defibrillator, are available.4, 5, 8

The point of entry to the ALS algorithm is dependent upon the circumstances of the cardiac arrest. In many situations, such as out-of-hospital cardiac arrest, BLS will already have been initiated and should continue while the monitor/defibrillator is being prepared. When the patient is being monitored at the time of cardiac arrest, diagnosis should be swift and the defibrillator attached without delay.

For the rescuer with a manual defibrillator the critical decision is whether or not the rhythm present is VF/VT.4 Nearly 70% of patients with an out-of-hospital cardiac arrest are in VF at the time of arrival of EMS personnel with a monitor/defibrillator.11 Most eventual survivors come from this group.5, 4

VF is a pulseless, chaotic, disorganized dysrhythmia characterized by an undulating, irregular pattern that varies in
amplitude and morphology with a ventricular waveform of more than 150/minute. \( ^1 \) Pulseless VT is characterized by broad, bizarrely shaped ventricular complexes associated with no detectable cardiac output. By definition the rate is greater than 100/minute and is usually well in excess of 150.

The absence of a detectable cardiac output in the presence of a coordinated electrical rhythm is called electromechanical dissociation (EMD) or pulseless electrical activity (PEA). \( ^9 \)

Asystole is identified by the absence of any cardiac electrical activity. Occasionally asystole is incorrectly diagnosed because:

1. ECG leads may be broken or disconnected. The presence of electrical artifact during external chest compression indicates that the leads are connected and probably intact.

2. Lead sensitivity may be inappropriate. The sensitivity should be increased to maximum: an accompanying increase in the size of electrical artifact will confirm that the sensitivity selection is functioning.

3. On occasions VF has a predominant axis. If this is at right angles to the selected monitor lead even coarse VF may cause minimal undulation in the baseline and resemble asystole. At least two leads should be selected in succession before asystole is diagnosed, preferably leads at right angles, such as II and aVL.

If there is any difficulty in diagnosing the rhythm in a patient with cardiac arrest the VF/VT protocol should be followed. \( ^{5,8} \)

**DEFIBRILLATION**

The only proven effective treatment for VF is electrical defibrillation. \( ^1 \), \( ^{10,12} \) When a defibrillator is available, it should be brought immediately to the side of the person in cardiac arrest and, if the rhythm is VF/VT, defibrillation should be attempted without delay.

The chances of defibrillation restoring a sustained, perfusing rhythm, and also of a long-term favourable outcome are optimal for as little as 90 seconds after cardiac arrest and decline rapidly thereafter as myocardial high-energy phosphate stores are consumed. \( ^5 \), \( ^8 \), \( ^9 \) BLS may be expected only to slow further myocardial deterioration, but is critical to the maintenance of cerebral circulation. Effective BLS will sustain the cerebral circulation at viable levels for 5–10 minutes or more. However, restoration of an effective spontaneous circulation provides the only means of completely reversing the effects of ischaemia and should be achieved as rapidly as possible. \( ^8 \) Defibrillation should only ever be delayed by the commencement or continuation of BLS if this can be expected to improve the cellular biochemistry of the myocardium or if restoration of some cerebral circulation is essential. \( ^{1,4,8} \)

All of the resuscitation guidelines referred to previously stress the importance of minimum delay in the administration of defibrillating shocks. \( ^{1,5,8,9} \) Furthermore, the ILCOR, ERC and ARC guidelines and algorithms qualify the commencement of BLS with the statement – ‘if appropriate’. \( ^{1,8,9} \) This approach is justified because:

- the prospects of successful defibrillation decrease relatively rapidly over a few minutes after cardiac arrest
- BLS is unlikely to improve the odds of successful defibrillation
- modern defibrillators have very rapid charge times: three shocks of appropriate energy levels can be given within 30 seconds by a trained and well-equipped team. \( ^{1,8} \)

**Technique**

For defibrillation to be successful, a critical myocardial mass must be depolarized synchronously to interrupt the fibrillation and allow recapture by a single pacemaker. The technique used must minimize transthoracic impedance in order to maximize the probability of success. \( ^{4,11,12,13} \)

- paddles of 10–13 cm in diameter for adults. Smaller paddles allow too concentrated a discharge of energy and may cause focal myocardial damage. \( ^{11,12} \)
- conductive electrode paste or pads reduce impedance by 30%. \( ^{11} \)
- pressure of about 5 kg on each paddle. \( ^{11} \)
- defibrillation in expiration. \( ^{13} \)
- repeated countershocks with a short interval between. \( ^{11,13} \)

**Paddle placement**

There are two widely accepted positions for the defibrillation paddles that optimize current delivery to the heart. The most convenient is the antero-apical position, where one paddle is placed to the right of the sternum just below the clavicle, and the other is centred lateral to the normal cardiac apex in the anterior or midaxillary line (V5-6 position). An alternative is the anteroposterior position with the anterior paddle placed over the precordium or apex and the posterior paddle on the back in the left or right infrascapular region. Paddles are often labelled sternum and apex, which is irrelevant for transthoracic defibrillation, but allows correct orientation of rhythms detected by the paddles for synchronized cardioversion. \( ^{4,11,12,13} \)

Defibrillation should not be attempted over ECG electrodes or medicated patches, and placement of paddles over the breast tissue in female patients should be avoided. \( ^{4,12} \) If the patient has an implanted pacemaker module or cardioverter defibrillator, the paddles should be placed at least 12–15 cm away from the module and pulse generator respectively. Pacemaker function should be checked as soon as practicable following successful defibrillation. \( ^{11,12} \)
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Technical problems
If attempted defibrillation is not accompanied by skeletal muscle contraction, care should be taken to ensure good contact and that the defibrillator is turned on, charged, develops sufficient power and is in asynchronous mode. The majority of defibrillator problems are due to operator errors or faulty care and maintenance. The operational status of defibrillators should be checked regularly and a stand-by machine should be available when possible.

Timing and energy of shocks
When using a conventional defibrillator with a damped monophasic sinusoidal waveform (see below) the first shock is given with an energy level of 200 joules (J), which represents the best compromise between the probability of success and the risk of myocardial damage. If the first attempt at defibrillation is unsuccessful, a shock of the same energy is repeated. If still unsuccessful a third shock is given, this time at 360 J. The paddles need not be removed from the chest while being recharged and CPR need not be recommenced between these initial shocks, unless there is a delay exceeding 20 seconds in recharging the defibrillator. With modern defibrillators it is possible to administer three shocks within 60 seconds.

The carotid pulse should be palpated if, after a defibrillating shock, an ECG rhythm compatible with cardiac output is obtained. However, if the monitor indicates persistent VF then additional shocks in the sequence of three can be administered without a further pulse check. After a defibrillating shock there is typically a delay of several seconds before an ECG trace of diagnostic quality is obtained. Additionally, even when defibrillation is successful, there is often a temporary impairment of cardiac function associated with a weak, or difficult to palpate, central pulse for seconds to minutes. It is important to recognize these phenomena and allow for them rather than hastily conclude that defibrillation has been unsuccessful or that EMD has developed.

COMPLICATIONS OF DEFIBRILLATION
- Skin burns can occur; these are usually superficial.
- Skeletal muscle injury or thoracic vertebral fractures are possible, though are uncommon.
- Myocardial injury and post-defibrillation dysrhythmias can happen with cumulative high-energy shocks.
- Health-care providers can receive electrical injuries due to electrical contact with the patient during defibrillation. These range from paraesthesiae to deep partial thickness burns. Cardiac arrest is a theoretical possibility. The operator should ensure that all rescue personnel are clear of the patient before delivering a defibrillation shock. A particular concern should be to ensure that the patient, rescuers and equipment are dry before defibrillation is attempted, especially outdoors or around a swimming pool area.

DEVELOPMENTS IN DEFIBRILLATION

Automated external defibrillators
Automated external defibrillators (AEDs) were first introduced in 1979 and have become standard equipment in many EMS systems primarily for use outside of the hospital. The AED is attached by two connecting cables to adhesive pads that are placed on the patient’s chest in the standard antero-apical positions for defibrillation. An internal microprocessor analyses the ECG signal and, if VF/VT are detected, causes the AED to display an alarm and either delivers a shock (automatic) or advises the operator to do so (semi-automatic).

AEDs are highly accurate with some models demonstrating 100% specificity and 90–92% sensitivity for coarse VF. Although their precision is less for fine VF and least for VT, their accuracy overall is comparable to that of experienced cardiologists. Several EMS systems equipped with AEDs have shown that they can deliver the first shock up to 1 minute faster than when using conventional defibrillators and rates of survival to hospital discharge are equivalent to those achieved when first responders used manual defibrillators.

The major advantage of AEDs over conventional defibrillators is their simplicity, which has markedly reduced the skill required to defibrillate a patient in cardiac arrest. This decreases the time and expense of initial training and continuing education, and increases the number of persons who can operate the device. Members of the public have been trained to use AEDs in a variety of community settings and have demonstrated that they can retain skills for up to 1 year. Encouraging results have been produced when AEDs have been placed with community responders, such as fire fighters, police officers, security guards at large public assemblies and public transportation vehicle crews.

Current-based defibrillation
Conventional defibrillators are designed to deliver a specified number of joules (J). However, depolarization of myocardial tissue is accomplished by the passage of electrical current through the heart, and clinical studies have determined that the optimal current is 30–40 amperes (A). At a fixed energy, the current delivered is inversely related to the transthoracic impedance and a standard energy dose of 200 J delivers about 30 A to a patient with average transthoracic impedance. In patients with greater than average impedance, the current generated may be inadequate, whereas a patient with smaller than average impedance may sustain myocardial damage from excessive current flow.

Some newer defibrillators automatically measure transthoracic impedance and then predict and adjust the energy delivered to avoid inappropriately high or low transmyocardial currents. These devices have defibrillation success rates comparable to conventional defibrillators while cumulatively delivering less energy. The decreased energy should result in less myo-
Cardiac damage and may reduce post-
defibrillation complications.\textsuperscript{5, 11, 12, 13}

**New defibrillator waveforms**

Most conventional defibrillators use a 
damped monophasic sinusoidal waveform, 
which is a single pulse lasting for 
3–4 ms. Many studies over the last decade 
have shown that biphasic (bidirectional) 
truncated transthoracic shocks are as 
effective at lower energies as standard 
damped sine wave shocks and result in 
fewer post-defibrillation ECG abnormalities.\textsuperscript{4, 11, 12, 13} In a recent review, 
the reviewers concluded that lower-energy 
biphasic shocks, delivered without an 
increase in energy, achieved clinical 
outcomes equivalent to those of mono-
phasic shocks with increasing energy 
levels, in out-of-hospital cardiac arrest.\textsuperscript{14}

Defibrillators using biphasic waveforms 
with impedance compensation are now 
available and will likely become the 
standard in the near future. Research is 
still needed to determine the optimal 
biphasic waveform and energy levels 
for first and subsequent shocks, but the 
potential advantages are equivalent or 
more effective external defibrillation 
with a reduction in myocardial injury 
produced by the defibrillatory shocks.\textsuperscript{5}

**FAILURE OF DEFIBRILLATION, EMD 
AND ASYSTOLE**

Most patients who will survive cardiac 
arrest are successfully defibrillated by one 
of the first three shocks and even if this 
first sequence is unsuccessful, the best 
chance for restoring a perfusing rhythm 
is still defibrillation.\textsuperscript{4} However, at this 
stage it is necessary to recommence BLS 
in an attempt to restore some myocardial 
and cerebral perfusion and maintain 
cellular viability. Additionally, efforts 
should be made to secure advanced air-
way management and ventilation, and to 
institute vascular access for administration 
of drugs.

Potentially reversible causes or 
aggravating factors of cardiac arrest 
(Fig. 1.2.1) should be considered and 
specific therapy commenced as indicated.

These interventions should occur during 
the 1 minute period of CPR, although it 
is unlikely that even a highly trained 
team will be able to complete all of these 
aspects of management within this 
interval. Further opportunities will occur 
with subsequent cycles.\textsuperscript{4}

The ECG rhythm is then reassessed 
and if VF persists the next sequence of 
three defibrillating shocks is started 
without delay. These shocks are all at 
360 J, or its equivalent if the defibrillator 
is current-based or uses a biphasic 
waveform.\textsuperscript{4, 11, 12, 13}

If VF/VT is definitely excluded at the 
time of initial or later rhythm analysis, 
defibrillation is not appropriate and may 
be deleterious.\textsuperscript{4, 11, 12, 13} These patients will 
have EMD or asystole and the prognosis 
for these rhythms is much less favourable 
than for VF/VT. There are some situa-
tions where EMD or asystole may have 
been provoked by conditions that are 
treatable. The common causes are listed 
in Figure 1.2.1 and may be recalled under 
the headings of the four Hs and four Ts. 
Apart from treating potentially remedi-
ble conditions the management of EMD 
and asystole is largely the application 
of other ALS therapies and the 
continuation of BLS.\textsuperscript{1, 4, 8, 9}

**OTHER ALS INTERVENTIONS**

Except for defibrillation, no single 
ALS intervention has been scientifically 
proven to enhance patient outcome,\textsuperscript{10, 15} 
although the ILCOR considers that there 
is valid scientific evidence to support 
tracheal intubation as unequivocally 
effective.\textsuperscript{11} Many clinicians maintain that 
ALS has an incremental benefit compared 
with defibrillation alone.\textsuperscript{3, 15, 16} While 
there are some data to support this 
contention, it remains impossible to prove.\textsuperscript{10}

**Advanced airway management**

No randomized controlled studies exist 
that demonstrate the life-saving effect 
of endotracheal intubation compared to 
Basic airway management.\textsuperscript{15, 16} However, 
intuitively some benefit would be expected.

Direct expired air resuscitation and bag/
valve-mask ventilation are less effective 
than ventilation via an endotracheal tube 
and provide no protection against aspira-
tion, which is found in 28% of patients 
examined by the coroner after failed 
resuscitation from cardiac arrest.\textsuperscript{16, 17, 18} 
Also, EMS systems that use endotracheal 
intubation report higher survival rates 
than systems that do not.\textsuperscript{15, 17}

Endotracheal intubation is the gold 
standard for advanced management of 
the airway during cardiac arrest. It 
provides a clear and secure airway, allowing 
ventilation, oxygenation, suction and 
administration of medications if 
indicated.\textsuperscript{1, 6, 17, 18} Attempts at intubation 
should not interrupt BLS for longer than 
15–20 seconds. If intubation is not 
accomplished within that time, addi-
tional attempts should be delayed until 
the cycle of CPR following the next 
sequence of three defibrillation shocks, 
or with EMD and asystole until after a 
further 3 minutes of CPR.\textsuperscript{4, 8, 9}

**Ventilation and oxygenation**

During cardiac arrest, carbon dioxide 
(CO\textsubscript{2}) production and delivery to the 
pulmonary circulation is limited by the 
relatively low cardiac output achieved 
during CPR. As a consequence, relatively 
low minute volumes are sufficient to 
achieve adequate CO\textsubscript{2} excretion and 
prevent hypercapnia. This situation may 
be altered if CO\textsubscript{2}-producing buffers such 
as sodium bicarbonate are administered, 
and relative increases in minute ventila-
tion are required to prevent the develop-
ment of respiratory acidosis.\textsuperscript{4}

Several animal studies and evidence 
from humans in cardiac arrest indicate 
that, when the airway is patent, spontan-
eous gasping can provide sufficient 
ventilation during CPR to maintain 
normal arterial CO\textsubscript{2} levels. Similarly, 
chest compression alone provides some 
pulmonary ventilation and gas exchange, 
which can approach normal values with 
active compression–decompression CPR. 
Ventilation may actually be unnecessary 
during the first few minutes of CPR, 
although under conditions of prolonged 
cardiac arrest, it is essential for survi-
val.\textsuperscript{10, 18} In most cardiac arrest situations,
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a tidal volume of 400–500 mL (5–6 mL/kg) is sufficient to clear CO₂ during CPR and will cause a visible rise and fall of the patient’s chest.\(^1\)

Cardiac arrest and CPR cause an increase in dead space and a reduction in lung compliance that may compromise gas exchange. As adequate oxygenation is of paramount importance in cardiac arrest, the aim should be to provide a fractional inspired oxygen concentration (FIO₂) of 1.0.\(^4,\)\(^8\)

Tidal volumes of 400–500 mL and FIO₂ approaching 1.0 are attainable using self-inflating bag/valve-mask and intubation devices, which remain the mainstay of ventilation in ALS.\(^4,\)\(^8,\)\(^18\)

Drug therapy

No drug used in resuscitation has been shown to improve long-term survival in humans after cardiac arrest.\(^10,\)\(^15,\)\(^20,\)\(^21\)

Despite this knowledge, a number of pharmacotherapeutic agents continue to be employed, largely for historical reasons based on theoretical, retrospective or anecdotal evidence of efficacy.\(^10,\)\(^20\)

Adrenaline (epinephrine)

Actions The beneficial actions of adrenaline (epinephrine) in cardiac arrest are due to its α-adrenergic effects, whereas the β-adrenergic activity appears, at best, to be unimportant and may be detrimental. A series of experiments have demonstrated that adrenaline (epinephrine) maintains tone in intrathoracic arteries, preventing their collapse during external chest compression, and also increases resistance in non-cerebral and non-coronary arteries. These actions result in decreased blood flow to non-cerebral and non-coronary vessels, increased aortic blood pressure and increased perfusion of the cerebral and coronary vascular beds.\(^22\)

Indications and dose Adrenaline (epinephrine) is recommended in VF/VT cardiac arrest if there is no ROSC after the first three attempts at defibrillation. It is recommended in EMD and asystole after commencement of CPR. The standard adult dose is 1 mg intravenously (IV) every 3 minutes.\(^1,\)\(^4,\)\(^5,\)\(^8,\)\(^20,\)\(^21\)

For a number of years there has been considerable interest in high-dose adrenaline, usually defined as amounts in excess of 45 μg per kg every 5 minutes. However, no prospective randomized clinical trials in humans have demonstrated a significant improvement in survival to hospital discharge for adult patients treated with either standard-dose or high-dose adrenaline.\(^5,\)\(^10,\)\(^15,\)\(^16,\)\(^20\)

Potential complications Particularly in higher doses adrenaline (epinephrine) may increase myocardial oxygen requirements, induce myocardial contraction band necrosis and predispose to tachydysrhythmias. After ROSC it can produce severe hypertension. Tissue necrosis occurs commonly after extravasation.\(^5,\)\(^18,\)\(^21,\)\(^22\)

Vasopressin

Actions Vasopressin is an endogenous peptide hormone synthesized in the hypothalamus and secreted from the posterior pituitary in response to a variety of osmotic and non-osmotic stimuli. The principal physiologic effects of vasopressin are direct vasoconstriction of the systemic circulation, mediated by V₁ receptors on vascular smooth muscle and renal water retention, mediated by renal V₂ receptors. During the last decade research has indicated that vasopressin may be an important hormone during cardiac arrest and levels of vasopressin during CPR are significantly higher in eventual survivors than in those who do not survive. A possible advantage of vasopressin during cardiac arrest and CPR is that it produces vasoconstriction in non-vital tissues while preserving blood flow to the coronary and cerebral circulations.\(^23\)

Indications and dose The AHA in the International Guidelines 2000 recommend that vasopressin is an effective vasopressor and can be used as an alternative to epinephrine for the treatment of adult shock-refractory VF.\(^5\) The ERC and ARC do not include vasopressin in their recently published algorithms.\(^8,\)\(^21\) Although there are still insufficient clinical data to support the use of vasopressin as a first-line drug in the management of cardiac arrest, reports to date are promising and further research may well provide definitive information in the near future.\(^23\) The dose currently recommended by the AHA is an i.v. bolus of 40 U administered once during an episode of cardiac arrest.\(^5\)

Potential complications The beneficial effect of vasopressin on the cerebral circulation during CPR may increase the risk of cerebral oedema or haemorrhage after ROSC. Vasopressin has a relatively long half-life (10–20 minutes) and persistent vasoconstriction following ROSC may exacerbate myocardial ischaemia and interfere with left ventricular function. Vasopressin also exerts a procoagulant effect on platelets.\(^23\)

Lidocaine

Actions Lidocaine is a Vaughan-Williams class IB agent that depresses myocardial excitability by blocking sodium channels without extending action potential duration. In animal models it also has an antifibrillatory action.

The role of lidocaine in assisting resuscitation from refractory VF is contentious. Several experimental studies in animals have shown that it increases the defibrillation threshold (the energy required for defibrillation).\(^12,\)\(^18\) Other studies have shown no effect, or a decrease in the defibrillation threshold.\(^4,\)\(^24\) A recently reported retrospective observational study of outcome from cardiac arrest with sustained VF, compared the survival of patients who received lidocaine with those who received no lidocaine. This study showed a significant increase in ROSC in the lidocaine group but there was no difference in the rate of hospital discharge between the two groups.\(^23\) Controlled prospective trials of lidocaine and alternative antifibrillatory drugs are still needed.\(^4,\)\(^20\)

Indications and dose Lidocaine cannot be recommended as first-line therapy in cardiac arrest, but may be considered if multiple DC shocks and adrenaline have failed to revert VF/VT.
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It can also be used to help prevent reversion to VF/VT after successful defibrillation.5, 12, 20, 21 The initial bolus dose is 1–1.5 mg/kg with an additional bolus of 0.5 mg/kg after 5–10 minutes if indicated.4, 5, 21

Potential complications Cardiovascular effects include hypotension, bradycardia and asystole. Neurological toxicity causes central nervous system (CNS) excitation with anxiety, tremor and convulsions followed by CNS depression.4, 20, 21

**Amiodarone**

**Actions** Amiodarone is a class III agent that has some class I activity and weak non-competitive β-blocking effects. Amiodarone lowers the defibrillation threshold and has a potent antiarrhythmic effect. Its broad spectrum of antiarrhythmic activity and minimal adverse haemodynamic effects make it a potentially useful agent, but its value during cardiac arrest has not been extensively explored.20, 24

**Indications and dose** Amiodarone cannot be recommended as first-line therapy in cardiac arrest, but may be considered if multiple DC shocks and adrenaline have failed to revert VF/VT. The initial dose is 5 mg/kg given as a slow intravenous infusion over 5–15 minutes. This may be repeated if indicated.

**Potential complications** Cardiovascular effects include hypotension and bradycardia.24

**Atropine**

**Actions** Atropine antagonizes parasympathetic nervous effects on the heart by blocking cholinergic muscarinic receptors, leading to increased sinoatrial and atrioventricular automaticity and rate of conduction.5, 20, 21 Atropine may be effective when increased vagal tone results in a haemodynamically significant bradyasystole but its effect on EMD or asystole caused by prolonged, widespread myocardial ischaemia is negligible.24

**Indications and dose** Atropine may be considered in bradyasystolic cardiac arrest that does not respond to initial CPR and adrenaline (epinephrine). The dose is 3 mg, which is considered to be the vagolytic dose.5, 8, 20

**Potential complications** Adverse effects include tachycardia, CNS excitement and delirium, which are usually regarded as benign.21, 24

**Magnesium**

**Actions** Magnesium is an essential electrolyte that may be depleted by diuretics, severe diarrhoea and alcohol abuse. Hypomagnesaemia may cause cardiac dysrhythmias.4, 21 Several case reports and trials have yielded contradictory results concerning the effect of magnesium in cardiac arrest, and there is little to support its routine use as present.10, 20, 24

**Indications and dose** Magnesium may be considered in refractory VF/VT, particularly if hypokalaemia is present, and as an agent of choice in torsades de pointes. The initial dose is 5 mmol (2.5 mL of 49.3% solution) given over 1 minute, which may be repeated if indicated and followed by an infusion of 20 mmol over 4 hours.5, 20, 21

**Potential complications** Adverse effects include hypotension and heart block. Muscle weakness and paralysis may occur if excessive quantities are administered.5, 21

**Calcium**

**Actions** Calcium is a divalent cation essential to neuromuscular function. Human studies have shown that its pharmacotherapeutic effects in cardiac arrest are negligible and may be adverse.5, 20

**Indications and dose** Calcium should not be administered unless there is evidence that cardiac arrest is caused or exacerbated by hyperkalaemia, hypocalcaemia or overdose of calcium-channel-blocking drugs.4, 5, 21 The dose is 5–10 mL of 10% calcium chloride or three times that dose of 10% calcium gluconate.5, 21

**Potential complications** Calcium may increase the damage caused by profound ischaemia of myocardial and cerebral cells.4, 20, 21 Extravasation causes tissue necrosis.21

**Sodium bicarbonate**

**Actions** Sodium bicarbonate (NaHCO₃) is an alkalinizing agent that, theoretically, reverses the metabolic acidosis associated with profound ischaemia. However, provided CPR is effective, acidosis does not develop rapidly or severely in otherwise healthy individuals during cardiac arrest.4, 20, 21 There is no strong clinical evidence supporting the administration of alkalinizing agents in cardiac arrest.5, 15, 20 Some benefits have been reported, particularly when the dose can be titrated to avoid alkalosis and with concurrent use of adrenaline (epinephrine).16, 20 It is probably unwise to completely abandon NaHCO₃ therapy for all patients with cardiac arrest, and an objective reappraisal is needed to define its role.10, 20

**Indications and dose** Sodium bicarbonate is unnecessary in brief resuscitations when the patient has been previously well but can be considered if cardiac arrest exceeds 10–15 minutes duration.10, 20 It should also be considered when cardiac arrest occurs in a patient with a pre-existing profound acidosis or in special circumstances, such as tricyclic antidepressant overdose and hyperkalaemia.5, 21

**Potential complications** Adverse effects of NaHCO₃ include alkalosis, hyperosmolality and CO₂ production, causing paradoxical intracellular acidosis.4, 5, 21

**VASCULAR ACCESS AND DRUG DELIVERY DURING CARDIAC ARREST**

The ideal route of drug delivery combines rapid and easy vascular access with quick delivery to the central circulation. Central venous cannulae deliver drugs
1.2 ADVANCED LIFE SUPPORT

rapidly to the central circulation but re-quire considerable technical proficiency to insert during CPR and some methods of insertion interfere with defibrillation and CPR, which is unacceptable.5,20 The most appropriate method of vascular access will usually be via a peripheral venous cannula. When drugs are admin-istered by this route the extremity should be elevated and a 20 mL bolus of IV fluid should follow the agent to facilitate delivery to the central circulation.4,20

Intratracheal deposition is an alter-na-tive route, and during cardiac arrest tracheal intubation often precedes venous access.4,20 If there is a delay in achieving vascular access most ALS drugs, includ-ing adrenaline (epinephrine), lidocaine and atropine, may be safely administered through the endotracheal tube.5,20 The ideal dose and dilution of drugs given by this route is uncertain but current recommendations are to use two to three times the standard IV dose diluted in 10 mL of normal saline. This solution should be delivered via a catheter placed beyond the tip of the endotracheal tube and followed by five ventilations to aid dispersion.4,20,26

Crystalloid solutions are preferred as the standard vehicle of drug delivery as administration of glucose-containing solutions during CPR may contribute to post-arrest hyperglycaemia, which is detrimental to cerebral recovery.26

HAEMODYNAMIC MONITORING DURING CPR

Researchers and clinicians have proposed and measured numerous physiological parameters as a means of monitoring the effectiveness of resuscitation during cardiac arrest. The techniques for measuring these parameters are usually invasive, technically difficult and time consuming to establish and maintain, thereby limiting their utility in sudden and unexpected cardiac arrest.27

Numerous animal and clinical experi-ments indicate that measurement of end-tidal CO2 (ETCO2) may be an effective and informative method of determining the progress of CPR.10,27 The normal ETCO2 is 4–5% and typically falls to less than 1% at the onset of cardiac arrest. With effective CPR, the ETCO2 rises to between one-quarter and one-third of normal and ROSC is associated with a rise to normal or supranormal levels over the next minute. These changes parallel proportionally similar alterations in cardiac output.27

An ETCO2 of less than 1% during attempted resuscitation from cardiac arrest is an indication of ineffective CPR. This may be due to inadequate ventilation due to airway obstruction or oesophageal intubation, or due to the cardiac output being less than expected because of poor technique or causes such as hypo-volaemia, pulmonary embolism or peri-cardial tamponade. A sharp rise in ETCO2 may be the first indication of ROSC.27

End-tidal CO2 measured immediately after commencement of CPR may also have a prognostic value in out-of-hospital cardiac arrests as it is higher in patients who have had a short interval of cardiac arrest, as compared with those who have had a longer period prior to the initiation of resuscitation. There is also evidence that patients who are eventu-ally resuscitated have a higher ETCO2 during CPR than those who will never have ROSC. Caution should be exercised in interpreting ETCO2 following adrena-line (epinephrine), as this agent causes a decrease in ETCO2, which is not necessarily a poor prognostic indicator.27

DISCONTINUING ALS

With the introduction of effective EMS systems, initiation of ALS for patients with out-of-hospital cardiac arrest moved from the institution into the community. Research indicates that the vast majority of patients who survive out-of-hospital cardiac arrest have ROSC before arrival at the emergency department (ED). In 18 papers published between 1981 and 1995, only 33 (0.6%) of 5444 patients who were transported to an ED still in cardiac arrest after unsuccessful prehospital resuscitation, survived to hospital discharge.28 Twenty-four of the surviving patients arrived in the ED in VF and 11 of these patients had their initial arrest in the ambulance en route to the hospital or had temporary ROSC before arrival at the ED.

In 1993 a recommendation was made that after out-of-hospital cardiac arrest in the normothermic patient, resuscitation should cease if there is no ROSC after 25 minutes of ALS.29 The two exceptions to this practice are if the cardiac arrest occurs in the presence of ambulance personnel, or the patient demonstrates persistent VF. These recommendations were applied and considered to be valid in a prospective study in the same year.30

Early in the resuscitation of patients with in-hospital cardiac arrest there are no absolute predictors of futility but some variables are associated with a greater or lesser chance of survival to discharge. Ventricular tachydysrhythmias, commence-ment of resuscitation within 5 minutes and ROSC within 15 minutes are all linked to better outcomes. Pre-existing conditions such as metastatic cancer, renal failure, sepsis, acute cerebrovas-cular accident and cardiogenic shock are all linked to poor outcome. Age is not an independent predictor of outcome and this has also been confirmed in out-of-hospital cardiac arrest.31,32 Resuscitation efforts lasting more than 30 minutes without ROSC appear to be so uniformly unsuccessful that they should be aban-doned except in unusual circumstances.10,32

PROGNOSIS AFTER CARDIAC ARREST

The prognosis for survival after an out-of-hospital cardiac arrest fluctuates from community to community. Some of the variation is due to differences in EMS systems but much is also due to diverse research methodology and data report-ing. In King County, Washington, where a sophisticated EMS system has been in place for over two decades, survival to hospital discharge was between 15% and 20% from 1976 to 1987. These figures are in contrast to US national rates of survival, which were estimated to be from 1% to 8% in a 1991 report from the
CONTROVERSIES

1. Acceptance of universal guidelines and algorithms.
2. The role of new technologies in defibrillation.
3. The need for ventilation as initial therapy in cardiac arrest.
4. The role of vasopressors in ALS.
5. When is CPR futile?

REFERENCES


AHA. In 1987–88 in Perth, Australia survival was 22.7% of 231 cases of out-of-hospital cardiac arrest due to VF and in western metropolitan Melbourne in 1995 the survival rate for all arrest dysrhythmias was 3%. In a 1996 meta-analysis of 36 articles published between 1973 and 1992 describing 41 EMS systems in six countries, survival varied from 0% to 21% with an overall mean survival of 8%. Prognosis for survival from in-hospital cardiac arrest is only marginally better with survival to hospital discharge averaging 13.8% of 12961 patients described in reports published between 1961 and 1984. In a further seven reports published between 1978 and 1989, 11% of 1804 patients survived to hospital discharge.

UNIFORM REPORTING IN CARDIAC ARREST RESEARCH

Cardiac arrest research and the interpretation of available data has often been hampered by inconsistent methodology and reporting. An important initiative has been the recognition of the need for uniform, internationally recognized definitions and guidelines for reporting of cardiac arrest data. During the last few years a number of templates have been developed to include the most relevant variables for describing and comparing cardiac arrest research results. These are referred to as Utstein style guidelines or templates after Utstein Abbey, near Stavanger, Norway, where expert researchers and clinicians gathered in 1990.

REFERENCES

INTRODUCTION

Appropriate airway management is the initial step in the resuscitation of the patient with critical illness. Basic airway manoeuvres include jaw thrust, chin lift and finger sweeps to clear the airway, together with expired air or bag/valve/mask breathing for ventilation. Advanced airway management includes endotracheal intubation (ETI) to provide a secure airway and allow assisted ventilation. In many emergency departments, advanced airway management is undertaken by an appropriately trained emergency physician rather than an anaesthetist. This chapter details the equipment, drugs and techniques that may be used in advanced airway management in the emergency department (ED).

Patients with respiratory arrest require immediate ETI and ventilation with oxygen. Although a trial of non-invasive ventilation may be used initially in conscious patients with severe hypo-oxaemia or hypercapnoea, this may be unsuccessful and ETI will be required. Patients with a decreased conscious state and/or depression of the cough reflex may require ETI for airway protection to minimize the risk of aspiration pneumonitis. In patients with severe head injury, ETI and controlled hyperventilation may be required for the short-term treatment of intracranial hypertension. Finally, ETI may be indicated as part of general anaesthesia in combative patients who require investigations and/or procedures.

There are additional challenges to urgent intubation of the critically ill or injured patient in the ED compared with elective intubation in the operating room. Details of current medications, previous anaesthetics and allergies may not be available. There may be inadequate time for a complete clinical assessment of the upper airway or thorough consultation with the patient and/or family. In patients with coma following severe head injury, the status of the cervical spine will be uncertain even if initial imaging is normal.

RAPID SEQUENCE INTUBATION

Unless the patient is deeply comatose or in cardiac arrest, ETI will require the use of sedative and neuromuscular blocking drugs to facilitate laryngoscopy and placement of the endotracheal tube. Rapid sequence intubation (RSI) is the technique of choice when definitive airway management is required in the ED, to minimize hypoxaemia or the risk of aspiration of vomitus. Possible exceptions include patients with upper airway obstruction or severe facial trauma, where alternate initial techniques, such as awake intubation or awake tracheostomy, may be preferred (see later).

Careful preparation for RSI is required. If time and patient condition allow, a history should be sought of current medication, allergies and time of last meal. A careful examination of the upper airway is required, looking for anatomical features that may predict difficult intubation.

The conscious, cooperative patient should receive explanation and reassurance. Preoxygenation with 100% oxygen by mask is commenced using a non-rebreathing circuit. Optimal pre-oxygenation requires tidal volume breathing for 3–5 minutes using 10 litres/minute oxygen flow. A pillow under the head is essential, unless the patient has suspected spinal column injury, in which case the neck must be immobilized in an anatomically neutral position. Reliable intravenous access is required, as well as equipment for suctioning the airway.

Appropriate monitoring includes continuous ECG and pulse oximetry. The blood pressure should be measured, either invasively using an intra-arterial cannula or non-invasively using an automated blood-pressure monitoring device. Capnography must be prepared for end-tidal CO₂ (ETCO₂) measurement following intubation.

The required drugs should be chosen and will depend on operator preference and the clinical situation. In general, a narcotic and benzodiazepine are used in combination with a rapid-onset neuromuscular blocking drug. Details of the indications, dosages and side effects of the commonly used drugs for rapid sequence intubation are shown in Table 1.3.1. These must be drawn up, checked and the syringes clearly labelled. A spare laryngoscope must be available, in case of failure of the first and the appropriate size ETT opened, lubricated and the cuff checked. Another ETT (one size smaller) should be immediately available. At least two assistants will be required, one to assist the operator with the drugs and equipment, and another to provide cricoid pressure following the induction of sedation and muscle relaxation. Further equipment in case of difficult intubation should be immediately available (see later).

When all preparations are complete,
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### Table 1.3.1 Drugs commonly used in RSI

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Action</th>
<th>Onset (min)</th>
<th>Duration (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Premedication agents</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atropine</td>
<td>0.02 mg/kg</td>
<td>Vagal blockade</td>
<td>1</td>
<td>30</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1.5 mg/kg</td>
<td>Decreases ICP</td>
<td>0.5</td>
<td>10</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>1.5 µg/kg</td>
<td>Analgesic</td>
<td>1</td>
<td>30</td>
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<tr>
<td>Morphine</td>
<td>0.15 mg/kg</td>
<td>Analgesic</td>
<td>2</td>
<td>30</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.05 mg/kg</td>
<td>Anxiolytic</td>
<td>2</td>
<td>30</td>
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<tr>
<td>Vecuronium</td>
<td>0.01 mg/kg</td>
<td>Defasciculation</td>
<td>2</td>
<td>10</td>
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<tr>
<td><strong>Induction agents</strong></td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>Thiopentol</td>
<td>1–5 mg/kg</td>
<td>Rapid-onset sedation</td>
<td>0.5</td>
<td>10</td>
</tr>
<tr>
<td>Methohexital</td>
<td>1.0 mg/kg</td>
<td>Rapid-onset sedation</td>
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<td>5</td>
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<tr>
<td>Midazolam</td>
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<td>10</td>
</tr>
<tr>
<td>Diazepam</td>
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<td>Rapid-onset sedation</td>
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<td>20</td>
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<tr>
<td>Ketamine</td>
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<td>Dissociative state</td>
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<td>20</td>
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<td>Propofol</td>
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<td>Sedation</td>
<td>1</td>
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<td>Sedation, analgesic</td>
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<td>20</td>
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<td><strong>Muscle relaxants</strong></td>
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<tr>
<td>Suxamethonium</td>
<td>1.5 mg/kg</td>
<td>Depolarizing MR</td>
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<td>5</td>
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<tr>
<td>Vecuronium</td>
<td>0.2 mg/kg</td>
<td>Non-depolarizing MR</td>
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<td>40</td>
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<tr>
<td>Rocuronium</td>
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<td>Roccuronium</td>
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<td>Pancuronium</td>
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<td>Non-depolarizing MR</td>
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ICP: intracranial pressure
MR: muscle relaxant
RSI: rapid sequence intubation

Premedication with adjunctive agents such as atropine, a benzodiazepine and/or a narcotic are administered as clinically indicated. The sedative drug is then given and, as consciousness is lost, the muscle relaxant (usually suxamethonium) is given with cricoid pressure applied. Following fasciculations and the loss of muscle tone, laryngoscopy is performed and the larynx sighted. The endotracheal tube is then placed through the vocal cords into the trachea, the cuff inflated and the ETT is secured with tapes. Cricoid pressure must be maintained until the ETT position is checked and secured.

Clinical methods of ensuring tracheal position include sighting the passage of the ETT through the vocal cords, misting of the ETT during exhalation, auscultation of breath sounds in the lung fields and palpation of the ETT cuff in the suprasternal notch by the squeeze test. However, when visualization of the vocal cords has been difficult, these clinical tests may be misleading and confirmatory tests will be required. Although capnography is regarded as the gold standard for confirmation of tracheal placement, during cardiac arrest there may be inadequate delivery of carbon dioxide to the lungs and hence a false-negative reading. In this setting, the use of an oesophageal detector device (ODD) has been shown to be more accurate.

After intubation, an orogastric or nasogastric tube should be inserted and chest X-ray taken to exclude right main bronchus intubation and confirm placement of the orogastric or nasogastric tube in the stomach. As the drugs used for sedation and muscle relaxation wear off, further drugs for the maintenance of sedation and paralysis will be required. Appropriate monitoring of vital signs, pulse oximetry and capnography with visual and audible alarms must be maintained at all times. Humidification of the inspired oxygen is desirable using a disposable filter. If the patient is placed on mechanical ventilation, the PaCO₂ should be checked to ensure adequate ventilation and to confirm correlation with ETCO₂. The unconscious patient requires eye care, pressure area care, temperature control and catheterization of the urinary bladder.

Hypotension following intubation must be treated promptly, especially in patients with neurological injury. The causes include vasodilator and/or negative inotropic effects of the sedative drug(s) and/or positive pressure ventilation decreasing venous return and cardiac output. Treatment consists of a fluid challenge and/or inotrope administration, depending on the clinical setting. Rarely, hypotension may be due to tension pneumothorax occurring after the commencement of positive-pressure ventilation. Hypertension usually indicates inadequate sedation and should be treated with supplemental sedation.

In patients with severe head injury, the following additional measures need to be considered. As there is the possibility of cervical spine instability, an assistant must hold the head in the neutral position, which increases the difficulty in visualizing the larynx. Also, laryngoscopy may raise intracranial pressure, however, the benefit of lidocaine at 1.5 mg/kg as premedication is uncertain.

The technique of RSI is not advised for patients with upper airway pathology and impending upper airway obstruction. Following the administration of a muscle relaxant, the larynx may not be visualized and ventilation of the apnoeic patient may be impossible, the ‘can’t intubate, can’t ventilate’ situation. In these patients, an initial awake technique may be performed instead. Alternatively, an inhalational anaesthetic agent or intravenous propofol is utilized, as their effects will rapidly be reversed and spontaneous respirations resume if intubation is impossible.

**DIFFICULT INTUBATION**

The intubation of the trachea under direct vision may be easy or difficult, depending on the view of the larynx during laryngoscopy. This has been
1.3 ADVANCED AIRWAY MANAGEMENT

classified into Grades 1-4 by Cormack and Lehane. In Grade 1 laryngoscopy, there is a clear view of the entire laryngeal aperture. In Grade 2, only the posterior part of the larynx is visible. In Grade 3, only the epiglottis is able to be visualized and in Grade 4 only the soft palate is seen. A difficult intubation is defined as a Grade 3 or 4 view at laryngoscopy.

Difficult intubation may be anticipated in the presence of pathological disorders such as congenital facial and upper airway disorders, maxillofacial and airway trauma, airway tumours and abscesses, or cervical spine immobility. There may also be anatomical reasons for Grade 3-4 laryngoscopy and a range of clinical tests have been proposed that may predict difficulty in visualization of the larynx, including relative tongue/pharyngeal size, atlanto-occipital joint mobility and a thyromental distance < 6 cm. However, these are unlikely to be clinically useful in the emergency setting.

When the larynx is not visualized, attempts at blind placement of the ETT into the trachea are unlikely to be successful and repeated attempts at intubation result in patient hypoxaemia. Failed intubation drills have been described for use in the operating theatre and a failed intubation drill more suitable for use in the ED is shown in Figure 1.3.1.

Initial simple manoeuvres to visualize the larynx include the addition of pillows to further flex the neck (unless cervical spine injury is suspected), the use of a straight laryngoscope blade and backward/upward/rightward external pressure (BURP) on the thyroid cartilage. If the larynx is still unable to be visualized, blind placement of a gum elastic bougie and subsequent placement of the ETT by rail-roading the ETT over the bougie should be attempted as the initial manoeuvre. If resistance to ETT passage at the larynx occurs, rotation of the ETT through 90° in an anti-clockwise direction may be helpful.

If these initial steps are unsuccessful, oxygenation must be maintained using a bag/valve/mask with a Guedel’s airway and alternative equipment suitable for use in the ED should be prepared for

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**Fig. 1.3.1**

- **Failed Intubation Drill:**
  - Unable to see vocal cords during initial laryngoscopy?
    - Bag/mask/Oropharyngeal airway
  - Call for difficult intubation tray
    - Add introducer and additional pillow (unless contraindicated)
  - Trial of “blind” placement of a gum-elastic bougie.
    - Railroad lubricated ETT (One size smaller) over bougie
  - Immediate definitive check of position (ETCO₂/Air aspiration test)
  - Retry using additional pillow/introducer/different laryngoscope blade
  - Immediate definitive check of position (ETCO₂/Air aspiration test)

- **ETT in trachea**
  - Able to oxygenate/ventilate, allow patient to awaken and/or try alternative technique
  - Yes, now able to oxygenate and ventilate with LMA, allow patient to awaken and/or try alternative technique

- **ETT in esophagus**
  - Not able to oxygenate/ventilate
  - Insert laryngeal mask airway
  - Still not able to oxygenate/ventilate
    - Cricothyroidotomy

**ETT** = endotracheal tube
**ETCO₂** = end-tidal CO₂
**LMA** = laryngeal mask airway
1.3 ADVANCED AIRWAY MANAGEMENT

use. A summary of these devices is given below. However, if oxygenation and ventilation is considered unsatisfactory during the failed intubation drill, immediate cricothyroidotomy is indicated.

THE LARYNGEAL MASK AIRWAY

The laryngeal mask airway (LMA) is now used routinely for airway management during elective general anaesthesia. During a failed intubation drill, the LMA may be superior to bag/mask and oral airway for oxygenation and ventilation of the patient. However, there has been a limited role for the LMA in the ED for two reasons. First, if pulmonary compliance is low or airway resistance is high, there will be a leak around the cuff of the LMA when peak inspiratory pressures exceed 20–30 mmHg. Second, there is a potential risk of aspiration pneumonitis, since the airway remains unprotected. To minimize the risk of aspiration pneumonitis, further developments of the LMA now include a modified cuff to improve the seal and a drainage tube to provide access to the gastrointestinal tract (the ProSeal). The technique of retrograde intubation may be useful in the ED when other techniques fail and time allows. The cricothyroid membrane is punctured by a needle/cannula and a guide wire is passed through the cannula cephalad. This is brought out through the mouth using Magill’s forceps. An ETT is placed over a special bougie that is passed over the guide wire through the larynx. Resistance will be felt when the ETT reaches the larynx. When the level of the cricothyroid is reached, the guide-wire is removed and the ETT passed further into the trachea.

THE INTUBATING LARYNGEAL MASK AIRWAY

The LMA may be also used to assist in blind orotracheal intubation, either using a 6 mm ETT passed through the LMA, or an intubating LMA that has been developed for this purpose. Preliminary reports of the intubating LMA indicate a high success rate in the prehospital setting, ED and operating theatre.

THE OESOPHAGEAL TRACHEAL COMBITUBE (COMBITUBE™)

The oesophageal tracheal combitube combines the functions of the oesophageal obturator airway and a conventional ETT and may be useful in the failed intubation algorithm. A 97% success rate for oxygenation and ventilation of patients undergoing elective anaesthesia and a 91% success rate for successful insertion and ventilation by paramedics in cardiac arrest patients has been reported. However there are little data on emergency department use of this device.

THE LARYNGEAL TUBE AIRWAY

The laryngeal tube airway (Airway Management Device ™) combines the functions of the LMA and an oesophageal obturator airway and consists of a tube placed in the oesophagus with a proximal cuff that inflates in the oropharynx to form a seal for ventilation and a distal cuff that inflates to seal the oesophagus and prevent aspiration of vomitus and/or insufflation of the stomach. Evaluation of the laryngeal tube airway in emergency medicine is limited to a manikin study, which found that adequate ventilation at the first insertion attempt was possible in 96%.

BLIND NASOTRACHEAL INTUBATION

Blind nasotracheal intubation (BNTI) is a technique that is now rarely used in the operating theatre, but may occasionally be useful in the ED, either as the initial technique of choice, or as part of a failed intubation drill. Requirements for successful BNTI include spontaneous respirations and depressed gag/cough reflexes. Contraindications include coagulopathy, fractured base of skull, maxillary fractures, upper airway obstruction or suspected laryngeal injury.

To perform BNTI, high-flow oxygen is administered by mask and the nare checked to assess size and patency. The nare is prepared with a pledget soaked in local anaesthetic and vasoconstrictor such as 5 ml of lidocaine 2% with adrenaline (epinephrine) 1:100 000. After several minutes, the pledget is removed and sterile lubricant applied. Local anaesthetic may also be applied by spray to the pharynx and larynx. If required and clinically appropriate, sedation using midazolam 1–2 mg may be administered. An ETT of one size less than the predicted oral size is passed via the nose to the pharynx and advanced slowly to the larynx, with the operator listening for breath sounds. To facilitate entry into the larynx, the head may need to be flexed, extended or rotated, the ETT rotated clockwise through 90°, and/or a suction catheter used to guide the ETT. When the ETT passes into the trachea, spontaneous respirations through the ETT confirm placement. However, complications of BNTI including epistaxis, injuries to the turbinates, perforation of the posterior pharynx, laryngospasm and injuries to the larynx currently limit enthusiasm for this technique.

RETROGRADE INTUBATION

The technique of retrograde intubation may be useful in the ED when other techniques fail and time allows. The cricothyroid membrane is punctured by a needle/cannula and a guide wire is passed through the cannula cephalad. This is brought out through the mouth using Magill’s forceps. An ETT is placed over a special bougie that is passed over the guide wire through the larynx. Resistance will be felt when the ETT reaches the larynx. When the level of the cricothyroid is reached, the guide-wire is removed and the ETT passed further into the trachea.

FIBRE-OPTIC BRONCHOSCOPE ASSISTED INTUBATION

The fibre-optic bronchoscope may assist in the intubation of the patient where RSI fails or is contraindicated. If the patient is awake, the nasal passage and upper airway should have topical anaesthetic applied as for BNTI. Initially, a well lubricated ETT is introduced nasally and passed to the posterior pharynx. The bronchoscope is then passed through the
1.3 ADVANCED AIRWAY MANAGEMENT

ETT and the vocal cords visualized. The suction port may be used to clear any secretions. The bronchoscope is then advanced into the trachea and the ETT is railroaded over the bronchoscope. Following removal of the bronchoscope, the patient is ventilated with oxygen. If a LMA has been used during a failed intubation drill, this may be used to guide the bronchoscope with the ETT already placed over it.

The use of the fibre-optic bronchoscope in the ED is limited by several factors. The bronchoscope and light source must be immediately available for use during a failed intubation drill. The larynx may be difficult to visualize in the presence of blood, vomitus or secretions. Finally, the equipment is expensive to purchase and maintain.

CRICOThYROIDOTOMY

Cricothyroidotomy is an essential skill for the emergency physician and must be considered immediately in cases of ‘can’t intubate-can’t ventilate’.

To perform cricothyroidotomy, a small vertical incision is made over the cricothyroid membrane and artery forceps are used for blunt dissection to the cricothyroid membrane. The artery forceps are then passed into the trachea and the cricothyroid membrane opened horizontally. A size 6 mm ETT is passed through the opening into the trachea, the cuff is inflated and bag/valve ventilation commenced.

Alternatively, there are proprietary kits that allow a cricothyroidotomy tube to be passed over a guide-wire using the Seldinger technique. In this approach, the cricothyroid membrane is punctured with a needle/cannula mounted on a syringe and free aspiration of air confirms placement in the airway. The cannula is advanced as the needle is withdrawn and a guidewire is passed through the cannula down the trachea. The cannula is removed and a dilator passed along the guide-wire. Finally, a 4.5–6 mm cricothyroidotomy tube mounted on a dilator is passed along the guide-wire and placed in the trachea. The position of the cricothyroidotomy tube must be carefully checked, since misplacement in the mediastinum, anterior to the trachea is possible.

In children, puncture of the cricothyroid membrane with a 14 gauge needle/cannula and insufflation with oxygen is preferred, as injury to the tracheal mucosa at the level of the cricothyroid may lead to tracheal stenosis. In adults, placement of a too large tube (>6 mm) through the cricothyroid membrane is also considered unsatisfactory in the longer term because of possible stenosis occurring at the level of the cricoid. Therefore, conversion of the cricothyroidotomy to a sub-cricoid tracheostomy is generally undertaken at the earliest time it is safe and convenient.

TRACHEOSTOMY

Compared with cricothyroidotomy, a surgical tracheostomy is more time-consuming and difficult to perform in the ED, although it is recommended in suspected direct laryngeal trauma when an emergency airway is needed. Pre-tracheal dissection requires adequate lighting, instruments and diathermy. Percutaneous dilatational tracheostomy may be performed without these requirements, however, there is little published experience with this technique in the ED.

REFERENCES


CONTROVERSIES/PITFALLS

1. A number of clinical signs have been described that may predict difficult intubation, but none are completely accurate.
2. When the larynx is unable to be visualized, a failed intubation drill must be followed to prevent patient hypoxaemia.
3. The laryngeal mask airway, intubating laryngeal mask airway, Combitube™, retrograde intubation and fibre-optic-assisted intubation may have a role when orotracheal intubation is difficult or contraindicated.
4. Capnography is recommended for confirmation of tracheal placement of the endotracheal tube.
5. The air aspiration test is more accurate for patients in cardiac arrest.
6. Cricothyroidotomy is an essential skill for the emergency physician and must be considered immediately in cases of ‘can’t intubate-can’t ventilate’.
1.4 CEREBRAL RESUSCITATION AFTER CARDIAC ARREST

STEPHEN BERNARD

ESSENTIALS

1. Brain injury following global cerebral ischaemia is common following out-of-hospital cardiac arrest and is associated with high morbidity and mortality rates.

2. Reperfusion of the ischaemic brain results in glutamate-mediated calcium influx into cells, with biochemical cascades to cell death.

3. A number of pharmacological interventions have failed to improve neurological outcome in human randomized controlled trials.

4. Prospective, controlled trials indicate that induced hypothermia (33°C) for 12–24 hours after resuscitation from cardiac arrest is an effective treatment for anoxic neurological injury.

5. There is some evidence that hypotension and/or hyperglycaemia are deleterious to the injured brain and should be promptly treated.

INTRODUCTION

Prolonged cardiac arrest causing global cerebral ischaemia may lead to permanent neurological injury, despite effective cardiopulmonary resuscitation. Many patients who suffer out-of-hospital cardiac arrest remain comatose in the emergency department (ED) and the neurological injury accounts for much of the disability and death following hospital admission.

This chapter details the pathophysiology of anoxic neurological injury and current cerebral resuscitation treatment strategies.

DEFINITION

Cerebral resuscitation involves the use of pharmacological or other strategies to minimize injury to the brain following a prolonged ischaemic insult.

PATHOPHYSIOLOGY

The brain is highly dependent on an adequate supply of oxygen and glucose for aerobic metabolism. When cerebral oxygen delivery falls below 20 mL/100 g brain/minute, anaerobic glycolysis predominates with a marked decrease in the generation of adenosine triphosphate. After 3–6 minutes of complete cerebral ischaemia, the supply of adenosine triphosphate is exhausted and cellular metabolism ceases. The failure of the sodium/potassium transmembrane pump leads to persistent depolarization of the cell membrane and allows the equilibration of intracellular and extracellular ions, with the shift of sodium and water leading to cell swelling.

In addition, hydrogen ions with lactate ions are generated and the resulting intracellular metabolic acidosis is toxic to intracellular enzyme systems. The pH shift is partly dependent on the concentration of glucose, with hyperglycaemia leading to an intracellular acidosis.

THE REPERFUSION INJURY

Following reperfusion, additional injury occurs. The intracellular levels of glu-
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1.4 CEREBRAL RESUSCITATION AFTER CARDIAC ARREST

tamate, an excitatory neurotransmitter released from presynaptic terminals, increase dramatically during reperfusion. The glutamate activates ion channel complexes including N-methyl-D-aspartate receptors and α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid receptors. When activated, these ion channels increase calcium conductance from the extracellular to intracellular fluid. In addition, glutamate activates G-protein associated metabotropic receptors, inducing changes in phospho-inositol metabolism resulting in the production of inositol triphosphate. This acts as a second messenger, which releases further calcium from stores within mitochondria and endoplasmic reticulum.

Multiple biochemical cascades are initiated by calcium influx into cells, leading to the production of oxygen free-radicals and the activation of degradative enzymes, including proteases, endonucleases, phospholipases and xanthine oxidase. Phospholipase activation results in lipid peroxidation, which causes cell membrane destruction and neuronal death. Activated phospholipase A2 generates arachidonic acid, which mediates injury by several mechanisms, including the uncoupling of oxidative phosphorylation, inactivation of membrane Na+/K-ATPase and increased release of glutamate. Activated proteases (calpains) degrade cytoskeletal and regulatory proteins.

Intracellular iron also plays an important role in free-radical production. Iron is usually maintained in a ferric state and is sequestered to intracellular proteins. During ischaemia, iron is reduced to the soluble ferrous form and reacts with peroxide, generating damaging hydroxyl free-radicals.

The generation of free-radicals activates an upregulation of adhesion molecules in the leucocytes, endothelium and platelets. These adhesion molecules mediate leucocyte adhesion and extravasation into brain parenchyma, with increased cerebral ischaemia by causing microvessel occlusion with leucocyte-platelet complexes. There is also experimental evidence of marked activation of blood coagulation without endogenous fibrinolysis and platelet activation during reperfusion after cardiac arrest.5 In addition, vasoconstriction may occur secondary to the production of thromboxane A2 and prostaglandin F2α from arachidonic acid.

Ischaemia is also a stimulus for nitric oxide synthase activation. Nitric oxide synthase converts L-arginine to nitric oxide, a potent mediator of excitotoxic injury. The nitric oxide may combine with superoxide to form peroxynitrite radicals, which are potent activators of lipid peroxidation. Other proposed actions of nitric oxide include DNA damage, increased glutamate release and microvascular vasoconstriction.

Finally, some neurones which survive the initial anoxic insult proceed to programmed cell death (apoptosis). After reperfusion, this delayed neuronal death may occur at different rates, varying from 6 hours for neurones in the striatum to 7 days for hippocampal CA1 neurones. Apoptosis is characterized by cellular and nuclear shrinkage, chromatin condensation and DNA fragmentation. The complex biochemical pathways for this phenomenon are currently under investigation.7

CEREBRAL HAEMODYNAMICS AFTER REPERFUSION

After restoration of a spontaneous circulation, cerebral haemodynamics may remain abnormal.8 Following an initial hyperaemia, cerebral blood flow decreases, despite normal mean arterial blood pressure, whilst cerebral metabolic rate for oxygen increases. Thus, there may be continuing cerebral ischaemia for 12–24 hours following resuscitation from prolonged cardiac arrest. Cerebral oxygen supply/demand is also adversely affected by systemic hypoxaemia, raised intracranial pressure and/or seizure activity.

PHARMACOLOGICAL INTERVENTIONS

As much of the neurological injury seen following ischaemic injury occurs after reperfusion, there has been considerable interest and research into pharmacological interventions that alter the above metabolic pathways and ameliorate the reperfusion injury.5,9

A number of drugs that have shown improved neurological outcome in animal models of global cerebral ischaemia have undergone large randomized controlled human trials, including thiopentone,10 a corticosteroid11 and the calcium antagonists, lidoflazine12 and nimodipine.13 However, none of these showed improved neurological or overall outcome. Magnesium has been shown to improve neurological outcome in patients resuscitated from in-hospital cardiac arrest;14 however a study of patients with anoxic neurological injury after resuscitation from out-of-hospital cardiac arrest showed no benefit.15

Currently, there is some interest in the possible role of thrombolytic drugs during and after cardiac arrest with the goal of decreasing cerebral microcirculatory fibrin formation and improving cerebral blood flow.16

INDUCED HYPOTHERMIA

Induced hypothermia is theoretically beneficial after cardiac arrest and resuscitation. Hypothermia decreases cerebral oxygen demand without decreasing cerebral oxygen supply and potentially decreases the reperfusion injury by reducing the production of oxygen free-radicals after reperfusion.

Two prospective, controlled human studies have suggested improved outcome using moderate hypothermia in comatose survivors of prehospital cardiac arrest.16,17 In one study, 43 patients were randomized to induced hypothermia (IH) at 33°C for 12 hours and 34 patients were maintained at normothermia.16 Hypothermia was induced in the ED using ice-packs and neuromuscular blockade. At hospital discharge, 21/43 (49%) in the IH group had a good outcome compared with 9/34 (26%) in the control group (P=0.046). Following multivariate analysis for differences at baseline, the odds ratio for good out-
1.4 CEREBRAL RESUSCITATION AFTER CARDIAC ARREST

come in the hypothermic group was 5.25 (95% confidence intervals 1.47 to 18.76: P=0.011). There were no apparent adverse effects of IH such as sepsis, lactic acidosis or coagulopathy.

A second clinical trial of induced hypothermia was conducted in Europe.17 This study enrolled 273 comatose survivors of pre-hospital cardiac arrest, with 136 patients undergoing IH (33°C for 24 hours), and 137 patients maintained at normothermia. Hypothermia was induced in the ED using a refrigerated air mattress. At 6 months, 55% of the IH patients had good outcome, compared with 39% of normothermic controls (odds ratio 1.4, 95% confidence interval 1.08 to 1.81). The complication rate did not differ between the two groups.

However, these two studies also demonstrated that surface cooling had significant limitations. First, surface cooling is a slow method of decreasing core temperature, with 0.9°C/hour using ice packs16 and 0.5°C/hour using forced cold air cooling.17 Second, covering the patient with ice packs or cooling blankets during resuscitation is inconvenient and impractical for medical and nursing staff. Finally, the use of ice packs or refrigerated units (for forced air cooling) limit the use of these techniques to the hospital environment.

Since there is evidence from animal studies that outcome may be improved if cooling is initiated during or immediately after return of spontaneous circulation,4 current research focuses on the development of techniques for the induction of hypothermia that may be feasible in the out-of-hospital setting.

One approach, which has been recently studied in 16 patients who were resuscitated from out-of-hospital cardiac arrest, is the use of a cooling helmet.18 However, this technique is also a relatively slow, with the target core temperature of 34°C only reached after 180 minutes.

An alternative approach is the use of large volume, ice-cold (4°C) intravenous fluid.19 In a recent study of 22 patients who had been resuscitated from out-of-hospital cardiac arrest, a rapid intravenous infusion of large-volume (30 mL/kg) lactated Ringers solution at 4°C was shown to be an effective and safe technique for the induction of mild hypothermia. This therapy decreased core temperature by 1.7°C over 25 minutes and there were improvements in mean arterial blood pressure, as well as acid-base and renal function. There were no apparent complications of this therapy such as pulmonary oedema. If subsequent studies confirm this finding, this approach may be applicable to the out-of-hospital environment.

OTHER INTERVENTIONS

Animal studies of anoxic brain injury have suggested that outcome may be improved if elevated blood pressure is maintained in the post-resuscitation period and it seems reasonable to provide elevated mean arterial blood pressure after cardiac arrest.20 Hyperglycaemia is also associated with worse outcome following cerebral ischaemia and should also be corrected.21

CEREBRAL MONITORING

Recent developments in cerebral resuscitation include the use of invasive and non-invasive neurological monitoring to detect persistent cerebral ischaemia. Currently, monitoring for neurological deterioration after global or focal ischaemic injury is largely clinical, with observation of pupil reactivity, level of conscious state and the development or progression of focal neurologic signs. However, any change in clinical signs due to an increase in cerebral ischaemia may be delayed, with irreversible damage occurring before appropriate intervention. Thus, there is considerable interest in newer methods of non-clinical monitoring of the central nervous system.

The monitoring of cerebral perfusion pressure equating to the mean arterial blood pressure (MAP) minus the intracranial pressure (ICP) may give an estimation of cerebral oxygen delivery. Although increased ICP may occur after global ischaemia,22 there are little data on ICP monitoring in adults following prolonged cardiac arrest.

Cerebral oxygen delivery may also be estimated by the continuous or intermittent measurement of jugular venous oxygen saturation. However, this technique does not appear to provide useful information that alters management.23

Other neurological monitoring techniques that are currently being evaluated include near-infra-red spectroscopy, cerebral microdialysis and the direct continuous measurement of cerebral blood flow using jugular venous thermodilution.

SUMMARY

In patients with prolonged cardiac arrest and global cerebral ischaemia, neurological injury is mainly related to the time interval between cardiac arrest and the return of spontaneous circulation. Comatose patients should be intubated and have assisted ventilation with supplemental oxygen to protect the airway and assure adequate oxygenation and ventilation. Following the return of spontaneous circulation, a normal or elevated mean arterial blood pressure should be maintained using fluid and/or vasoactive drug therapy. In addition, hyperglycaemia should be promptly corrected with insulin therapy. Since adult out-of-hospital cardiac arrest often occurs in the setting of an ischaemic coronary syndrome, the usual cardiac care for this, such as the use of aspirin, heparin and thrombolysis, may be required.

There is now evidence that mild hypothermia for 12–24 hours following resuscitation is beneficial. The optimal technique for the induction of hypothermia is yet to be determined, however, preliminary evidence suggests that the rapid infusion of large-volume (30 mL/kg), ice-cold (4°C) intravenous fluid has advantages compared with surface cooling. Other pharmacologic and non-pharmacologic interventions are yet to be proven.

Admission to an intensive care unit will be required for most patients with anoxic brain injury following resuscitation for out-of-hospital cardiac arrest for further supportive care.
CONTROVERSIES
1. The biochemical pathways of neuronal death following ischaemia and reperfusion are complex and require further study.
2. Induced hypothermia is the only neuroprotective therapy that has shown benefit in clinical studies of global anoxic neurological injury.
3. Future studies will focus on techniques for the rapid induction of hypothermia either in the ambulance or the emergency department.

REFERENCES

ESSENTIALS
1. The three broad categories of shock include disorders of cardiac rate or rhythm; volume or vascular resistance problems; and myocardial pump dysfunction.
2. Hypotension, although characteristic of shock, should be considered a late finding.
3. Hypovolaemia, and hence volume resuscitation, should be carefully considered and excluded in every patient with undiagnosed shock.
4. The mortality following cardiogenic shock is improved by revascularization strategies, including angioplasty and coronary artery bypass grafting. Thrombolysis has no proven benefit but lysis may be supplemented with intra-aortic balloon counterpulsation where available.
5. Cyclo-oxygenase inhibitors, opioid antagonists and cytokine inhibitors confer little additional benefit in septic shock over fluid resuscitation, the use of inotropes and appropriate antibiotics or surgery. Activated protein C has been shown to improve mortality in severe sepsis and severe organ dysfunction.

DEFINITION
Shock is a clinical syndrome where tissue perfusion, and hence oxygenation, is inadequate to maintain normal metabolic function. Insufficient ATP (adenosine triphosphate) is generated intracellularly to maintain the function and structural integrity of tissues. This causes a switch to anaerobic metabolism, resulting in an oxygen debt and tissue acidosis.

The clinical recognition of shock may be difficult, particularly at the extremes of age. Pre-existing disease and the use of medications modify the compensatory mechanisms that protect vital organ perfusion. The emergency management of shock requires a high clinical suspicion and early, aggressive resuscitation.
SOURCES OF SHOCK

Shock is due to malfunction of the cardiovascular system for which there may be more than one contributing mechanism. A simple classification recognizes three broad categories:

❶ Disorders of cardiac rate or rhythm
 pudo
Volume or vascular resistance problems
a. volume loss – ‘empty tank’ (Table 1.5.1)
b. altered vascular resistance – ‘inappropriately sized tank’ (Table 1.5.2)

❷ Myocardial pump dysfunction (Table 1.5.3).

This classification is not exhaustive and contributing causes may feature in more than one category.

PATHOPHYSIOLOGY

The consequences of shock are cellular injury and death occurring by common mechanisms. Shock and reperfusion cause intracellular calcium overload, producing ATP reduction, diastolic dysfunction and decreased contractile forces in excitable tissues. Calcium overload is also related to free-radical oxidative damage, degradation of cell ionic pumps, and the destruction of cytosol, nuclear and mitochondrial macromolecules.

The accumulation of hydrogen ions downregulates catecholamine receptors, resulting in a reduction in catecholamine effectiveness and a decrease in intracellular energy production. Shock also causes a catabolic state, with increased circulating catecholamines, angiotensins, glucagon and corticosteroids. Metabolism becomes glycolysis dependent, and circulating glucose, lactic acid, free fatty acids and triglycerides increase.

Oxygen consumption is defective in some tissues in shock. Many causes are responsible, including physical barriers to diffusion such as dysfunctional endothelium, and interstitial and intracellular oedema, as well as metabolic dysfunction. In humans, most shock states result in flow-dependent oxygen uptake. However, the role of supranormal oxygen delivery in therapy has yet to be resolved.

Prolonged shock results in myocardial dysfunction. An early compensatory increase in heart rate is common. Diastolic dysfunction has also been described where active ventricular relaxation is impaired through the disruption of ATP-dependent sarcoplasmic reticular calcium ion uptake. Circulating catecholamines are initially increased, but ultimately in decompensated shock the heart fails, owing partly to circulating myocardial depressant factors described in haemorrhagic and septic shock.

Organ blood flow changes are important in shock, as shock raises the threshold at which vital organ blood flow decreases. These and other specific pathophysiologic changes are discussed later.

DIAGNOSIS OF SHOCK

- Hypotension is a sentinel clinical sign, defined as a systolic blood pressure <90 mmHg or a reduction of >30 mmHg in a previously hypertensive patient. However, a low blood pressure should be considered a late finding. Aggressive management aims to prevent the development of hypotension.

| Table 1.5.1. Examples of volume loss contributing to shock: |
| Blood loss (haemorrhagic shock) |
| External |
| − trauma |
| − gastrointestinal tract bleeding |
| Concealed |
| − haemothorax |
| − haemoperitoneum |
| − ruptured abdominal aortic aneurysm |
| − ruptured ectopic pregnancy |
| Loss of plasma |
| − Burns |
| − Exfoliative dermatitis |
| Loss of fluid and electrolytes |
| External |
| − vomiting |
| − diarrhoea |
| − excessive sweating |
| − urinary losses |
| − adrenal insufficiency (aldosterone deficiency) |
| − diabetes mellitus |
| − diabetes insipidus |
| − diuretics |
| − renal disease |
| Concealed |
| − pancreatitis |
| − ascites |
| − bowel obstruction |
| − peritonitis |
| − splanchic ischaemia |

| Table 1.5.2. Examples of shock resulting from altered vascular resistance: |
| Septic shock |
| Anaphylactic shock |
| Spinal neurogenic shock |
| Vasodilator drugs or toxins |
| Adrenal insufficiency (cortisol deficiency) |
| Central nervous system injury |
| Prolonged shock from any cause |

| Table 1.5.3. Examples of myocardial dysfunction resulting in shock: |
| Primary cause of myocardial dysfunction (cardiogenic shock) |
| Acute myocardial infarction |
| Cardiac contusion |
| Cardiomyopathy |
| Congestive heart failure |
| Myocarditis |
| Ruptured ventricular septum or free wall |
| Acute valvular dysfunction |
| − aortic insufficiency |
| − chordae tendineae rupture |
| − papillary muscle dysfunction |
| − prosthetic valve thrombus/dysfunction |
| − severe aortic stenosis |
| Secondary causes of myocardial dysfunction |
| Obstructive causes |
| − tension pneumothorax |
| − pericardial disease (tamponade, constriction) |
| − pulmonary vascular disease (thromboembolism, pulmonary hypertension) |
| − atrial myxoma |
| − left atrial mural thrombus |
| − obstructive valvular disease (aortic, mitral) |
| Drugs |
| Systemic toxins or myocardial depressant factors |
1.5 SHOCK

- Tachycardia is usually present, but may be masked by drugs or advanced age. The trend with serial observation is more significant than absolute values. Bradycardia may occur, for instance, in catastrophic haemorrhage from a ruptured ectopic pregnancy, or following an inferior myocardial infarction (MI).
- An abnormal respiratory rate of <10 or >29 per minute depends on the cause, such as narcotic overdose or early sepsisemia, but may also be part of the shock syndrome.
- Core temperature may be low, normal or elevated, and will be affected by age, environment, volume status, coexisting disease and drug therapy.
- SaO2 should be measured to detect early hypoxaemia.
- The mental state reflects cerebral perfusion and may range from normal to confused or coma.
- The peripheral circulation usually reveals vasoconstriction, decreased peripheral temperature, diaphoresis and pallor. Capillary return may be prolonged beyond 4 seconds. Peripheral or central cyanosis is a late sign. However, spinal neurogenic shock and sepsis may lead to warm, dry skin as a consequence of vasodilatation.
- Urine output is the most useful bedside monitor of the adequacy of end-organ perfusion. Levels below 0.5 mL/kg/h indicate underperfusion.

Invasive monitoring in the ED may also include:
- Intra-arterial blood pressure monitoring via the non-dominant radial artery
- Central venous pressure (CVP) monitoring via the subclavian or internal jugular veins. Trends in CVP may be followed in response to volume loading, but should be interpreted in relation to the other observed parameters
- End-tidal CO2 in ventilated patients

Pulmonary artery catheterization, gastric tonometry, Doppler cardiac output studies and other more sophisticated investigations are best performed in an integrated intensive care environment.

Lactate measurements are an objective marker of the presence and severity of shock. Bedside lactate analysis is now available. Normal levels are <2 mmol/L, and levels of >4 mmol/L are associated with increased mortality. Similarly, base deficit (BD) is a useful indicator of hypoperfusion and may be used to assess the adequacy of resuscitation. BD levels reflect the volume of fluid required, the presence and severity of intra-abdominal haemorrhage and mortality. It may also be used to identify compensated shock and to predict transfusion requirements, the need for ICU and the length of stay.

The incidence of adult respiratory distress syndrome (ARDS), multiple organ failure, renal failure and coagulopathy all increase with rising levels of BD.

The following shock syndromes are discussed in detail.

- Hypovolaemic shock
- Cardiogenic shock
- Septic shock
- Neurogenic shock

Hypovolaemic shock

Table 1.5.4 provides a guide to the pathophysiologic responses to acute haemorrhage. They relate to sympathetically mediated vasoconstriction and the release of endogenous catabolic hormones. Total peripheral resistance increases at the arteriolar level, venous capacitance increases, and blood flow to the brain and heart is maintained at the expense of renal, splanchnic, skin and muscle blood flow. Cerebral vascular autoregulation maintains cerebral blood flow and oxygen transport down to a mean blood pressure of 50–60 mmHg, below which acidosis and brain ischaemia develop, followed by a progressive fall in cerebral perfusion pressure and coma. Lung parenchymal water increases with decreasing surfactant and alveolar collapse. Pulmonary dysfunction develops through intravenous access is obtained with large-bore peripheral cannulae. Cardiac rhythm and pulse oximetry (SaO2) should be monitored. A fluid challenge may be given after drawing blood for investigations, including a bedside glucose level, and arterial blood gases are measured.

Vital signs are recorded and any available history obtained, followed by a directed physical examination. Observations should be continued regularly.

A chest X-ray, ECG and other emergency investigations are performed, e.g. bedside ultrasound to exclude pericardial tamponade, assess cardiac filling and ventricular function, or to seek intra-abdominal free fluid.
multiple insults at the microvascular and cellular level. Renal perfusion is decreased and glomerular filtration rate falls with intra-renal shunting of blood flow. Pancreatic blood flow may decrease to as little as 15% of normal, which may persist post resuscitation. A myocardial depressant factor has been isolated from the pancreas. Splanchnic blood flow is significantly reduced, with some preservation of mucosal circulation. Hypoperfusion may persist after the hypotension is corrected, which contributes to a failure of gut barrier function, leading to bacterial translocation and contributing to the development of multiple organ failure.

Clinical presentation

Signs and symptoms: The pulse rate, blood pressure, pulse pressure, respiratory rate, urine output and mental status change are detailed in Table 1.5.4. Neck veins will be flat as a consequence of low central venous pressure, where cardiac function is normal. A specific cause for blood volume loss must be sought (see Table 1.5.1).

Investigations:
- Bedside glucose estimation, arterial blood gases and serum lactate
- Full blood examination, haematocrit, coagulation profile, blood group and cross-match
- Urea, creatinine, electrolytes and liver function tests
- β-HCG in females of childbearing age
- Chest X-ray and 12-lead electrocardiogram.

Additional studies will be indicated in specific situations, and all tests are repeated serially according to the clinical picture.

Diagnosis Hypovolaemia, and hence volume resuscitation, should be carefully considered in every patient presenting with undiagnosed shock.

Therapy
- General supportive care is provided as described previously, with supplemental oxygen, cervical spine immobilization in trauma, and early endotracheal intubation and mechanical ventilation for airway or respiratory failure or progressive shock.
- The Trendelenburg position provides no consistent effect on systemic vascular resistance or venous return. Passive leg raising is more effective in increasing left ventricular end-diastolic volume, stroke volume and cardiac output, but these effects are transient.
- Application of the pneumatic antishock garment (PASG or MAST suit) results in a minimal autotransfusion effect. There is no evidence that it improves recovery in bleeding trauma patients and it has no place in the management of hypovolaemic shock.
- External haemorrhage is controlled with firm direct pressure.
- Intravenous access is gained with two 14 g cannulae, and fluid warmers and infusers capable of rapid delivery are employed.
- O-negative or type-specific blood must be transfused as soon as possible in patients presenting with class III or IV haemorrhage. Surgical consultation is also urgently required.
- Efforts to return blood pressure to normal in bleeding trauma patients may be counter-productive and occasionally harmful. Contemporary resuscitation practice, in the absence of evidence for the effectiveness of currently recommended resuscitation protocols, might best be regarded as experimental. Patients with class I or II haemorrhagic shock or non-haemorrhagic hypovolaemic shock should continue to receive warmed crystalloid. Timely restoration of perfusion and oxygen delivery should be the primary objective in bleeding patients presenting in rural and remote communities, in the elderly and in those with controlled haemorrhage. In those patients with uncontrolled haemorrhage following penetrating truncal trauma, in close proximity to facilities capable of definitive care, less aggressive fluid resuscitation, pending prompt surgical intervention, may be used.
- There is little evidence supporting the continued use of colloids in the resuscitation of critically ill or injured patients.
- A clinical role for hypertonic saline (HS) has yet to be defined as significant advantages of HS over standardized crystalloid solutions are unproven. HS may improve

<table>
<thead>
<tr>
<th>Table 1.5.4 Estimated volume losses in a 70 kg man at initial presentation</th>
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<tbody>
<tr>
<td><strong>Class</strong></td>
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<tr>
<td>Blood loss (mL)</td>
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<tr>
<td>Blood loss as a % of blood vol.</td>
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<tr>
<td>Pulse rate per min</td>
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<tr>
<td>Blood pressure (mmHg)</td>
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<tr>
<td>Pulse pressure</td>
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<td>Respiratory rate per min</td>
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<td>Urine output (mL/h)</td>
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<td>Mental status</td>
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<td>Fluid replacement</td>
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outcomes in a subgroup of patients with shock and traumatic brain injury and has been recommended as the initial fluid of choice in haemorrhaging battlefield casualties.9 There are no current definitive recommendations concerning the use of modified haemoglobin or perfluorocarbon blood substitutes.8

- Tension pneumothorax, cardiac tamponade and myocardial contusion should always be considered in the hypotensive trauma patient, although such patients should be assumed to be hypovolaemic until proven otherwise.
- Continuous monitoring and reassessment should take place in the ED pending definitive care and admission. Early ICU involvement should be considered.

**Cardiogenic shock**

**Definition**

Cardiogenic shock is the inability of the heart to deliver sufficient blood to the tissues to meet resting metabolic demands,9 i.e. a systolic blood pressure of <90 mmHg or ≥30 mmHg below basal levels for at least 30 minutes; alternatively, a significant arteriovenous oxygen difference and a cardiac index of <2.2 L/min/m² where pulmonary capillary wedge pressure is >15 mmHg are seen. There is clinical evidence of poor tissue perfusion in the form of oliguria, cyanosis and altered mentation. Failure to respond to correction of hypoxaemia, hypovolaemia, arrhythmias and acidosis is a requirement for the diagnosis.9

**Aetiology**

The commonest cause of cardiogenic shock is MI or ischaemia, resulting in at least 40% dysfunctional myocardium (see Table 1.5.3).

**Epidemiology**

Cardiogenic shock complicates 6–20% of patients with acute myocardial infarction (AMI). The mortality rate exceeds 80%. It is the commonest cause of in-hospital post-infarct mortality, and there has been no change in its incidence or prognosis since the early 1970s.9,10 Ten per cent of patients present already in established cardiogenic shock, while 90% develop it during admission.11

Older patients with anterior AMI, previous AMI, angina or congestive heart failure are at greater risk. One-quarter of patients will have reinfarcted. There is also a significant incidence of diabetes mellitus. There is a higher prevalence of patients with multivessel disease and persistent occlusion of the infarct-related artery. Patients with an occluded left anterior descending artery also have an increased incidence, termed the ‘left main shock syndrome’, with a mortality approaching 100%.12 Only aggressive revascularization within 12 hours of symptom onset makes any difference to these patients.

In a series of 231 patients with cardiogenic shock, 214 presented with symptoms and/or signs of left ventricular failure: 42% received thrombolysis, 26% had percutaneous transluminal coronary angioplasty (PTCA), and 8% had emergency coronary artery by-pass grafting (CABG). The overall mortality rate was 66%. Mortality in patients given intravenous thrombolitics was 61%, compared to 71% in those not receiving lysis. This was not statistically significant. Over 50% of patients with inferior AMI died, compared to 67% of those with anterior infarctions.13

**Pathophysiology**

The clinical consequences of pump dysfunction have been well described.9,14 The activation of the sympathetic nervous and renin-angiotensin systems contributes to the failure to increase or a fall in myocardial oxygen demand, which contributes to infarct expansion, and further decreases in contractility, coronary perfusion pressure and oxygen extraction. Systolic dysfunction results in an increase in end-systolic volumes and falls in ejection fraction, stroke volume and cardiac output. Diastolic dysfunction is also well described. Systemic hypoperfusion and selective vascular redistribution lead to organ failure and metabolic acidosis.

**Clinical presentation**

**Signs and symptoms** These vary with the cause. Non-specific findings are similar to those described under hypovolaemic shock.

- Jugular venous pressure is frequently elevated, but is a non-specific finding as it is also seen in the following conditions:
  - pericardial tamponade
  - constrictive pericarditis
  - pulmonary hypertension
  - right ventricular infarction
  - superior vena caval obstruction
  - tension pneumothorax
  - tricuspid valve insufficiency or stenosis.
- Blood pressure may be within normal limits as a result of compensatory mechanisms, which also produce tachycardia and a narrowed pulse pressure.
- Signs of pulmonary venous congestion are common, but may be absent in pure right ventricular infarction.
- Precordial examination may demonstrate a dyskinetic apex beat or thrill. A fourth heart sound suggests decreased ventricular compliance, and third sound increased ventricular diastolic pressure. Murmurs common in systole may be due to mitral regurgitation or, rarely, rupture of the ventricular septum.

**Investigations**

- 12-lead ECG. Leads V4R and V7-9 are indicated in suspected right ventricular and posterior infarction, respectively
- Chest X-ray
- Full blood examination and film
- Bedside blood glucose, urea and creatinine, electrolytes, liver-function tests, calcium, magnesium and phosphorus levels
- Serial CK (creatinine kinase), CKMB (creatinine kinase, muscle-brain), myoglobin and troponin I or T levels, depending on local availability
- Blood-gas estimation, except in candidates for lysis or revascularization.
Bedside echocardiography should be available to all patients who present with undiagnosed shock, as an extension of the physical examination. Pericardial effusion or tamponade may be excluded and global systolic function or wall motion abnormalities confirmed. The presence of hypovolaemia with a hyperkinetic heart and small right-side chambers, or right ventricular dysfunction may also be ascertained.15

**Diagnosis** Cardiogenic shock must be considered in any patient presenting with a primary or secondary cause for cardiac dysfunction (see Table 1.5.3) in the presence of symptoms and signs of hypoperfusion despite optimizing circulating volume following a fluid challenge.

**Therapy**
- Initial care and monitoring should be provided, as described earlier.
- Tracheal intubation should be considered for airway stabilization and ventilatory support in the presence of worsening hypoxia.
- Tension pneumothorax should be excluded.
- Arrhythmias considered as contributory to the presence of cardiogenic shock should be treated according to ACLS principles.
- Hypovolaemia must be sought and corrected in all patients. A 250 mL aliquot of 0.9% saline should be given cautiously as a bolus and the response assessed. Further boluses may be indicated. Volume loading to maintain right atrial filling pressures is essential in inferior MI with right ventricular (RV) involvement. CVP monitoring may be indicated, although it is of limited value in the presence of pulmonary oedema.
- Persistence of the shock state following adequate fluid challenge in the presence of end-organ dysfunction is an indication for inotropic support.
- Dobutamine is a β1-adrenergic agonist with some β2 effects that, although weak, lead to increased contractility, cardiac output, stroke volume and heart rate (at the higher end of the dose range). Peripheral vasodilatation is also produced and coronary and collateral blood flow augmented.9,10 Dobutamine is indicated at 2–20 μg/kg/min in patients with an SBP of 90–100 mmHg. It is the preferred inotrope in RV infarction and may be commenced by the peripheral route in the absence of central venous access. Tachycardia must be avoided, and obstructive cardiac lesions contraindicate its use.
- Dopamine is the endogenous precursor of noradrenaline, with dose-dependent effects16 as listed below:
  - 1–2 μg/kg/min: Increases renal plasma flow, glomerular filtration rate (GFR) and sodium excretion via dopamine-1 and dopamine-2 receptors. This effect is no longer valid.
  - 2–10 μg/kg/min: Significant inotropic effects at the β-adrenoreceptor increase cardiac output.
  - >10 μg/kg/min: Increasing peripheral vasoconstriction through α-adrenergic stimulation.
- Dopamine is useful as an inotropic agent when SBP is less than 90 mmHg in order to restore perfusion pressure to vital organs. It may increase the risk of arrhythmias and cause tissue necrosis following local extravasation. Its beneficial effects on renal blood flow in low dosage are debatable.
  - Adrenaline (epinephrine) is a potent α and β1 agonist and a moderate β2 agonist: 0.04–0.1 μg/kg/min produces increased heart rate and contractility with unchanged or lowered peripheral vascular resistance. Higher doses produce α-receptor mediated vasoconstriction. Adrenaline (epinephrine) is indicated in profound hypotension accompanying cardiogenic shock, and in those unresponsive to dobutamine and dopamine.
  - Noradrenaline (norepinephrine) is the main neurotransmitter at sympathetic postganglionic fibres, producing β1- and potent α1- and α2-agonist effects. It is indicated in severe cardiogenic shock to increase perfusion pressure. Its peripheral vasoconstricting effects may be counterbalanced by a vasodilator. The early use of noradrenaline (norepinephrine) in profound undifferentiated shock helps restore vital organ perfusion while awaiting the effects of fluid loading, and allows the introduction of other inotropes or vasodilators. The starting dose is 0.5–10 μg/min.
- Systolic blood pressure should remain at least 100 mmHg. A minimum value of 60 mmHg is suggested for mean arterial pressure (MAP). The use of pressors in cardiogenic shock has not been shown to improve survival.11
- Vasodilators are indicated when peripheral and organ perfusion fails to respond to restoration of blood pressure alone. Glycerol trinitrate is the vasodilator of choice in myocardial ischaemia, in a dose range of 10–200 μg/min. Sodium nitroprusside may be used when pulmonary oedema occurs in the absence of ischaemia. The dose range is 0.5–2.0 μg/kg/min, to a maximum of 10 μg/kg/min.
- MI must be sought and active management pursued dependent primarily on local facilities and expertise. Thrombolytic therapy in cardiogenic shock following AMI results in inadequate exposure of occlusive thrombus to the thrombolytic17. Intra-aortic balloon counterpulsation increases aortic diastolic pressure and cardiac output with no increase in oxygen demand and may be combined with thrombolitics, but when used alone confers no survival advantage unless revascularization is contemplated. Complications include leg ischaemia, dissection, thromboembolism and thrombocytopenia.11

Early transfer and revascularization confers a survival advantage in patients with MI and cardiogenic shock.9,10,13,18
Early revascularisation of the infarct-related artery by percutaneous transcoronary angioplasty (PTCA) or coronary artery bypass grafting (CABG) is the only intervention that decreases mortality. The greatest benefit is in those less than 75 years, with the optimal management of those over this age remaining unclear. If catheterization facilities are unavailable, thrombolytics should be given to eligible patients while emergent transfer to an interventional facility is arranged.

Cardiac tamponade should be excluded by transthoracic echocardiography in the following patient groups:

- Blunt or penetrating cardiac trauma
- Pericarditis due to infection such as TB or viral, radiation, connective tissue disorders
- Ureemia
- Anticoagulant use
- Aortic dissection
- Iatrogenic, e.g. CVP insertion
- Pneumopericardium from barotrauma, gas-forming infection, Valsalva, PEEP, cocaine.

Coexistent hypovolaemia may mask the clinical signs of tamponade, such as a full JVP rising on inspiration (Kussmaul’s sign). Volume loading is indicated with inotropic support until pericardiocentesis under echo-control or surgical pericardiectomy can be performed;

Aortic dissection should be considered in patients with risk factors, e.g. Marfan’s syndrome, Ehlers-Danlos syndrome, bicuspid aortic valve, aortic coarctation, Ebstein’s anomaly, hypertension in pregnancy and cocaine use.

Other causes of cardiogenic shock presenting to the emergency physician have been reviewed extensively.

In summary, those patients with large infarctions and a resting tachycardia should be identified early and stabilized with inotropic and/or vasodilator agents when indicated. Intra-aortic balloon counterpulsation should be instituted urgently while emergent revascularization is contemplated. Patients managed outside centres with interventional capabilities should be considered for thrombolytics where eligible.

Disposition
Patients presenting with cardiogenic shock require admission to CCU or ICU, depending on the requirement for ventilation, invasive monitoring or active management of myocardial ischaemia. Direct transfer from the ED to the catheter laboratory or operating theatre, or transfer to a tertiary centre, may be indicated.

Septic shock
Definitions
The following definitions were formulated at the consensus conference of the American College of Chest Physicians and Society of Critical Care Medicine (ACCP-SCCM) in 1992:

- Bacteraemia: the presence of viable bacteria in the blood, usually confirmed by positive blood culture.
- Sepsis: clinical evidence of infection, accompanied by a systemic response, including two or more features from the following:
  - tachypnoea, RR >20/min or PaCO₂ <32 mmHg (4.3 kPa)
  - where the patient is mechanically ventilated, minute ventilation >10 L/min
  - tachycardia >90 beats/min
  - hyper- or hypothermia, core or rectal temperature >38°C or <36°C and/or elevation or reduction in the leucocyte count >12000 cells/µL or <4000 cells/µL, or 10% or more bands.
- Systemic inflammatory response syndrome (SIRS): the presence of a severe clinical insult accompanied by two or more of the systemic responses outlined above.
- Severe sepsis: hypoperfusion (altered mentation, lactic acidosis and/or oliguria), hypotension or organ dysfunction associated with sepsis.
- Septic shock: sepsis accompanied by hypotension, systolic BP <90 mmHg or 40 mmHg or more below normal baseline and perfusion abnormalities despite adequate fluid resuscitation. Refractory septic shock is present when hypotension lasts >1 hour, despite adequate volume replacement and high-dose vasopressor use.
- Multiple organ dysfunction syndrome (MODS): a syndrome of altered organ function in an acutely ill patient requiring intervention to maintain homeostasis.

Debate surrounds these criteria, which, although still employed, should not form the sole basis for the clinical diagnosis of sepsis. Symptoms and signs that lead the clinician to suspect sepsis are as follows:

Clinical signs:
- Fever/hypothermia
- Unexplained tachycardia
- Unexplained tachypnoea
- Signs of peripheral vasodilatation
- Unexplained shock
- Changes in mental state.

Laboratory parameters:
- Leucocytosis/neutropenia
- Unexplained lactic acidosis
- Unexplained alteration in renal or liver function tests
- Thrombocytopenia/disseminated intravascular coagulation
- Increased procalcitonin levels
- Increased cytokines, c-reactive protein levels.

Aetiology
Infection of bacterial, viral or fungal origin is the precursor of septic shock, which may complicate up to 50% of bacteraemic episodes. Typically in Gram-negative sepsis, Escherichia coli, Klebsiella, Pseudomonas aeruginosa, Enterobacter, Acinetobacter, Proteus, Serratia, Aeromonas, Xanthomonas, Citrobacter, Achronobacter, Salmonella or Shigella species are responsible.

The pathogenic mechanisms in septic shock have been well reviewed. A nidus of infection is formed through multiplication of micro-organisms, which may invade the bloodstream or proliferate locally, releasing various mediators consisting of structural components and exotoxins into the circulation. Such mediators in turn stimulate plasma precursors or cells to release further endogenous mediators of sepsis. More than 100 me-
1.5 SHOCK

Mediators have been identified, including tumour necrosis factor-α (TNFα), interleukin-1 and interleukin-6, which are the most extensively studied. The effects of mediator release include direct organ injury, hepatic failure, disseminated intravascular coagulation (DIC), vasodilation, altered capillary permeability, myocardial depression, endothelial cell dysfunction and leucocyte aggregation.

Circulatory changes
Nitric oxide overproduction in the peripheral vasculature causes the loss of vascular control seen in septic shock.25 The vasodilatation and decreased systemic peripheral vascular resistance may be masked by hypovolaemia. Capillary blood flow is reduced, and decreased deformability of red and white blood cells underlies microvessel plugging, resulting in a potential trap for bacterial overgrowth in the microcirculation.26

Cardiac dysfunction
Ventricular dilatation with decreased ejection fraction is the commonest finding in septic shock, associated with a reduced stroke volume that is compensated for by an increase in heart rate to maintain or increase cardiac index.27 This ventricular dilatation is necessary, and usually reverses in 7–10 days in patients who survive. Decreased right ventricular (RV) function and increased pulmonary artery pressure are associated with a poor outcome. As in decreased left ventricular function, circulating mediators have been implicated, as poor RV performance is not entirely explained by raised pulmonary vascular resistance.

Mortality
Reported mortality ranges from 20% to 80%. Patients admitted to the ICU with hypotension associated with sepsis have a mortality rate of over 50%.

High-risk groups
These include the young and the old, those with burns, alcohol dependence, chronic renal failure, diabetes mellitus, immunosuppression, chronic cardiopulmonary disease, infection, malnutrition and the multiply injured.

Clinical presentation
Signs and symptoms
- Early: tachypnoea, tachycardia, temperature instability, oliguria, altered mental state, peripheral vasodilatation;
- Late: reduced capillary refill, hypotension, further altered mental status and reduction in urine output, evidence of myocardial dysfunction, metabolic acidosis;
- Evidence of genitourinary, respiratory or gastrointestinal infection. Many other sites are possible, including iatrogenic sources such as CVP lines and indwelling catheters. A careful secondary survey following resuscitation is essential.

Investigations
- Full blood examination, urea, creatinine, electrolytes, bedside glucose, coagulation profile, and β-HCG in females of reproductive age.
- Arterial blood gases, arterial or venous lactate.
- Two sets of blood cultures, including a set through any indwelling cannulae or central venous lines. Consider arterial blood cultures in the immunosuppressed, e.g. intravenous drug users, and cultures through a pre-existing arterial line.
- Urinalysis, microscopy, Gram stain and culture.
- Chest X-ray and 12-lead electrocardiogram.
- Additional studies as indicated by the clinical situation, likely source of sepsis and search for possible foci of infection.

Diagnosis
This is made utilizing ACCP-SCCM definitions. More than one cause for shock may exist in the same patient. Volume depletion must be treated with an appropriate fluid challenge.

Therapy
- Initial care is provided as outlined previously during the primary survey.

Early airway control with mechanical ventilation is necessary to optimize oxygenation and ventilation.
- Volume replacement should commence with 250 mL boluses of 0.9% saline titrated against observed parameters, and frequent clinical reassessment. CVP line insertion should be performed rapidly in the ED under strict asepsis if expertise is immediately available. Trends in CVP response to fluid infusion should be followed, rather than absolute values, in conjunction with other monitored parameters. CVP is of least value in the presence of myocardial dysfunction or elevated pulmonary artery pressures. An intra-arterial blood pressure monitoring line should be inserted in addition.
- Persistent hypotension and/or signs of organ hypoperfusion are indications for inotropic support. Dopamine is used commonly in higher doses, but is a weak vasoconstrictor in septic shock.28 There is no role for low-dose dopamine in improving renal function. The early use of noradrenaline is recommended where hypotension is severe, SBP 70 mmHg or less, and the response to dopamine is suboptimal. Noradrenaline may preserve vital organ perfusion while volume is replaced and hypoxaemia corrected. It may effectively optimize renal blood flow and renal vascular resistance.29
- Oxygen consumption is maintained in haemorrhagic and cardiogenic shock by an ability to increase oxygen extraction where oxygen delivery is extremely low.30 This ability is impaired in septic shock. Thus, oxygen delivery aims to reverse hyperlacticacidaemia to increase survival rates. The use of dobutamine is only advocated in patients with adequate central pressures, in order to increase cardiac index and hence oxygen delivery and consumption. However, routinely maintaining supranormal oxygen
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Vancomycin should not be used in Cefpirome, ceftepime or piperacillin + Gentamicin 4–6 mg/kg daily PLUS <0.5×10^9/L + predicted decline to <0.5×10^9/L + fever > 38°C. Empirical regimes should cover *Pseudomonas aeruginosa*.

**Neutropenic patients:**

<0.5×10^9/L or <1×10^9/L + predicted decline to <0.5×10^9/L + fever > 38°C. Empirical regimes should cover *Pseudomonas aeruginosa*.

Gentamicin 4–6 mg/kg daily PLUS either ceftazidime 1 g IV 8 hourly OR ticarcillin+clavulanate 3.1 g IV 6 hourly.

Cefpirome, cefepime or piperacillin + tazobactam may be substituted for ceftazidime or ticarcillin+clavulanate. Alternately monotherapy with ceftazidime, cefpirome, cefepime, imipenem or meropenem in maximal dosage is equally effective.

Vancomycin should not be used in febrile neutropenics unless the patient is in shock, is known to be colonized with methicillin-resistant *Staphylococcus aureus* (MRSA) or has clinical evidence of a catheter infection from a unit with a high incidence of MRSA infection: vancomycin 1 g 12 hourly IV.

Appropriate antibiotic therapy confirmed by in-vitro testing reduces mortality by 50%, and should always be discussed with infectious disease specialists and microbiologists. Further advice on condition-specific antibiotic regimes is available elsewhere.

• Sodium bicarbonate is not recommended for the treatment of lactic acidosis.

• Corticosteroids should only be given for adrenal insufficiency, or if the patient is already receiving corticosteroids. Corticosteroids in low dosage have also been found to be beneficial in a sub-set of patients with refractory septic shock.

• Cyclo-oxygenase inhibitors such as ibuprofen have no significant effect on outcome. Opioid antagonists in high dosage reverse low SBP and mean arterial pressure, but have no effect on survival. The use of prostaglandins, pentoxifylline, N-acetylcysteine, selenium, antithrombin III, immunoglobulins, growth hormone and granulocyte colony stimulating factor in non-neutropenics is currently unsupported and is not recommended.

• The use of cytokine inhibitors has not been associated with improvement in mortality rates in severe sepsis or septic shock.

Pathophysiology

Low thoracic lesions result in loss of lower extremity sympathetic tone and subsequent venous pooling. Upper thoracic lesions result in venous pooling in the lower extremities and the abdominal viscera. Cervical lesions result in the absence of intrinsic cardiovascular sympathetic tone, with loss of thoraco-lumbar vascular tone.

One in three patients with a complete cervical-cord injury requires support for their hypotension. The presence of hypotension has no implications regarding the degree of completeness of cord injury or prognosis.

Clinical features

• This is a diagnosis of exclusion in the injured patient. Hypotension should be accompanied by flaccidity and areflexia distal to the suspected level of the lesion. There should be no compensatory tachycardia or peripheral pallor, sweating or vasoconstriction.

• Tension pneumothorax and cardiac tamponade must always be considered, and hypovolaemic shock, which may be masked and should be excluded by appropriate fluid challenge and investigations, such as abdominal ultrasound or CT.
scanning for possible intra-abdominal or retroperitoneal blood loss.

**Therapy**
- Supportive therapy should be instituted to airway, ventilation and circulation, as indicated.
- Atropine 0.5–1 mg is given to counter unopposed parasympathetic vagal tone to a maximum dose of 3 mg, if bradycardia and symptomatic hypotension are present.
- Euvolaemia should be assessed with a fluid challenge: 250–1000 mL aliquots of 0.9% saline are given and the clinical response followed.
- If the above measures fail to return blood pressure and measurable signs of perfusion to normal, consider pharmacologic vasoconstriction. Ephedrine 5–10 mg IV, or phentylephrine 0.2–1 mg IV may be used. Noradrenaline (norepinephrine) at 2–5 mcg/kg/min titrated to response is used in refractory cases providing other causes of hypotension such as haemorrhage, tamponade, pneumothorax, etc., have been excluded.

**Disposition**
Patients should be cared for in an ICU or a dedicated spinal injury unit, depending on other injuries and local facilities.

**CONCLUSION**
The aetiology of shock in patients presenting to the emergency department is varied. Hypovolaemia should be sought in all cases, although specific management will depend on the underlying cause.

**REFERENCES**

### CONTROVERSIES
1. There is currently no evidence to support the use of colloids over crystalloids in shock management.
2. The overall benefits of hypertonic fluids in shock are yet to be confirmed by prospective clinical studies.
3. The use of fluid restriction until early surgery, although accepted in the management of penetrating truncal trauma, requires further evaluation in the blunt trauma patient.
4. Corticosteroids have shown some benefit in certain subgroups of patients with severe sepsis and septic shock
5. Only activated protein C can be currently recommended as adjunctive therapy in severe sepsis associated with organ failure.

1.6 ETHICS OF RESUSCITATION

INTRODUCTION

A working definition of ethics is the study of morality. It is reasonable to describe moral behaviour as that which is ‘the right thing to do’. Thus, medical ethical deliberation is the process of determining what is the right thing to do when considering any of the dilemmas that arise in medical practice.

The approach to these dilemmas may vary according to the philosophical perspective adopted. Although there are a variety of models describing moral decision making, only a pragmatic overview will be given here. In general terms one may adopt a utilitarian approach, which values the positive balance of good over bad brought about by any action, or a deontological approach, which values actions that adhere to overriding moral principles. However, moral philosophers have recognized that moral principles may compete against each other when specific actions are considered. Moral principles should be honoured, but when they are competing, in a given circumstance, we should then consider the relative balances of good and bad that ensue from the application of each principle. In other words, we have a composite philosophy wherein the principles and the consequences of their application could both be considered.

Beauchamp and Childress have recently developed this further into a practical framework for medical ethical deliberation. They describe four principles that should be honoured in medical decision making. When these principles compete, the relative balance of good and bad should be considered. These principles are: respect for patient autonomy, beneficence, non-maleficence and justice.

RESPECT FOR PATIENT AUTONOMY

Autonomy is the patient’s moral right to determine his or her own destiny. It is a principle that has grown in stature in recent years. The realization of the importance of informed consent is a consequence of this.

Although the principle of respect for patient autonomy remains sound, there are many occasions in resuscitation medicine where the patient’s competence is impaired, as he or she is unable to receive information, undertake rational deliberation or express a decision free from coercion. Although we still endeavour to respect the patient’s autonomy, we will struggle to define what the patient’s autonomous wishes would be, if he or she was not impaired. This will be discussed further later in the chapter.

BENEFICENCE

Beneficence is the principle of acting in a way that benefits the patient. Historically, this and non-maleficence have been the overriding governing principles in medical practice. When these principles are enforced without due consideration of, or in contradiction to, the patient’s perceived or expressed wishes, the action is termed paternalistic. When the principle of respect for patient autonomy is not honoured, the patient is deprived of a fundamental right and is treated as less worthy by being reduced to a position where he or she is considered incapable of self-governance. This harm to the patient needs to be considered when the relative benefits and harms of any action are considered.

ESSENTIALS

1. Ethical deliberation may be aided by considering the four principles of: respect for patient autonomy; beneficence; non-maleficence and justice.

2. During deliberation, if the ethical principles seem to be competing the relative benefits and harms of the application of each should be considered.

3. During resuscitation, urgency and impaired patient competence conspire against adequate consideration of these principles, especially non-maleficence and respect for patient autonomy.

4. Resuscitation can be harmful in a number of ways. These should be considered when assessing the balance of benefit and harm of any resuscitation endeavour.

5. All medical interventions need some form of consent, including resuscitation, despite the urgency and the impaired patient competence. Of the consent options available, presumed consent is the one most commonly employed. Presumed consent using professional substituted judgement is a model that best respects patient autonomy.

6. The practice of resuscitation procedures, particularly endotracheal intubation, on the newly dead is common and valuable. Some form of consent is required for this to occur, but currently there is no suitable model. Presumed consent would be appropriate if the practice was explicit and if the public was well informed. In so doing those who would not consent are protected by the opportunity to decline. Until then, practising on the newly dead is ethically wrong.
NON-MALEFICENCE

Non-maleficence, or the principle of avoiding harm in therapeutic endeavours, is an established maxim attributed to Hippocrates. Although this is an obvious and commonsense principle, we will commonly tolerate some harm, for example when delivering chemotherapy for cancer, or undertaking surgery that is known to have certain complications, because consideration of the other principles tells us our actions are right.

The principles of beneficence and non-maleficence risk being poorly considered because they ‘go without saying’. However, we should be reasonably certain of the benefits and harms of our interventions before they may be considered right. For example, the performance of gastric lavage on a non-consenting patient after a trivial overdose several hours earlier is ethically unjustifiable as there is insufficient benefit to override our principles of respect for autonomy and non-maleficence. In order to consider the benefits and harms, information is required regarding the outcomes of our interventions, and to this end research becomes an ethical necessity to provide the evidence upon which to judge competing principles.

JUSTICE

The principle of justice is an essential balance to the first three principles, which apply primarily to the individual. Justice, or the concept of fairness, is best addressed by questioning whether there are others who might be adversely affected by a particular action. For example, in a mass casualty incident the performance of a hopeless resuscitation may be unjust (in addition to harming the patient) as it deprives another of resuscitation facilities.

THE HARMS OF RESUSCITATION

The benefits of resuscitation include the avoidance of death and the restoration of good health. The harms of resuscitation, may be of the following five types:

1. The first is if resuscitation is unnecessary because the patient’s condition is insufficiently serious to justify it. As a consequence, the harm includes pain and other discomfort to the patient, iatrogenic illness and unnecessary use of limited resources, thereby depriving others in need of these resources. In resuscitation medicine the extent of overtreatment may be difficult to predict, as it is hard to know whether the patient would survive intact without the treatment. The most promising way of minimizing this harm is to have senior staff present during a resuscitation to draw upon their experience.

2. The second harm of resuscitation is if it is unsuccessful because the patient’s condition is too far advanced. When resuscitation will not produce the desired effects because the patient is too sick, there is the potential for a great number of harms. Harms to the patient include physical discomfort, loss of dignity, a prolonged death, and survival with an unacceptable quality of life. Harms to the family include the psychological discomfort of surrogate pain and loss of dignity, unfulfilled hope, loss of control of a loved one’s destiny, the cost of lost earnings while at the bedside, and the cost of supporting a disabled survivor. The harms to the health workers include frustration and sadness at lack of success, guilt at inflicting harm, and the cost of being unable to treat others waiting for resources. The harms to the community include the loss of resources to treat others, the deception that resuscitation offered hope, and the worry that death must be preceded by a loss of dignity.

3. The third harm of resuscitation is if it is unkind because it brings about an outcome with which the patient or their family is unhappy. Resuscitation may condemn the patient to a quality of life below that considered acceptable. This is potentially a tragic harm, with an ongoing burden from which the patient and their carers may have no means of escape.

4. The fourth harm of resuscitation is if it is unwise because it diverts resources from alternative healthcare activities that would bring more
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Resuscitation is a significant user of resources: if it is futile and beneficial healthcare activities cannot proceed for lack of resources, then resuscitation is causing significant harm.

Finally, resuscitation is harmful if it is against the patient’s wishes. A preconceived ‘do not resuscitate’ order written by or negotiated with the patient, or consent declined by a competent patient, must be honoured, in keeping with the ethical principle of respect for autonomy. However, preconceived orders must be carefully considered as they relate to the situation in which the patient finds him or herself. For example, a written signed and witnessed statement (often called an advance directive, or living will) declining resuscitation from cardiac arrest means that the patient should not be resuscitated from cardiac arrest: it does not mean that the patient has declined resuscitation from haemorrhagic shock. Similarly, a ‘no intensive care’ directive does not mean the patient has declined aggressive treatment for pulmonary oedema with a nitrate infusion and continuous positive airway pressure ventilation. Although such directives may occasionally apply specifically to the patient’s illness, on other occasions they may not. In the setting of some form of an advance directive, three questions should be considered: 1) Did the patient make this decision based on well informed deliberation? 2) Is the context they find themselves in now what they had in mind when they made the decision? If not, how closely does it relate to what they had in mind? 3) Since they made this decision, is there any indication they might have changed their mind? If the answers to these questions suggest some doubt as to how applicable the advance directive is to this resuscitation, at this time, then it should be considered an indication of the patient’s wishes, rather than morally binding.

These harms of resuscitation must be considered when weighing the relative merits of the principles of respect for patient autonomy, beneficence, non-maleficence and justice.

An ill-considered approach will lead to underrepresentation of patient autonomy in these deliberations, and an inadequate appreciation of the extent of the harm that may ensue from resuscitation efforts.

FUTILITY

The concept of futility has been widely discussed in the medical literature, with particular emphasis on resuscitation medicine. Regrettably, discussions of the harms of resuscitation have become stalled by attempts to define futility. The word is derived from the Latin *futilis*, meaning ‘that which easily pours or melts’. The current usage stems from the story in which the daughters of the King of Argos murdered their husbands and were then condemned to collect water for eternity in leaking buckets. To arrive at your destination with an empty bucket when the intention of the journey was to bring water is undoubtedly a futile endeavour. However, futility in medicine is much more difficult to define. Some emphasize physiological futility, meaning the inability to produce a physiological objective, for example if CPR produces no pulse, or transfusion produces no blood pressure. The proponents of this definition suggest that it has the least risk of unilaterally imposed physician value judgements. Others consider futility in terms of quantitative or qualitative measures. A quantitative estimate of futility is one in which an intervention is considered futile if it has failed in, for example the last 100 times attempted. The qualitative component describes futility if the patient’s resultant quality of life falls below a threshold considered minimal by general professional judgement. It is unlikely that there will ever be agreement as to what physiological measure or quality of outcome measures are most appropriate, and what threshold measure separates futility from benefit. Although these arguments are interesting, it is unfortunate that they have taken on more importance than they merit. Futility defines the absence of acceptable benefit for any given intervention, whereas reasonable ethical deliberation demands we consider the ratio of benefit and harm. If there is no benefit, any harm at all would make the benefit-harm ratio unfavourable. However, even if the endeavour is not futile and brings about measurable benefit, this does not necessarily mean that the endeavour is the right thing to do, as the amount of harm that ensues, as defined above, may outweigh any benefit. It is the benefit-harm balance, as assessed by considering the four ethical principles and the five types of harm described above, that has the most relevance in determining whether to start or stop resuscitation.

CONSENT, WITHHOLDING AND WITHDRAWING RESUSCITATION

Consent must be obtained for any medical intervention, including resuscitation. Informed consent, as is appropriate for elective surgery, may be inappropriate during resuscitation owing to the urgency of the treatment and impaired patient competence. However, if informed consent is not relevant, other forms of consent still are. The two most common forms of consent employed in resuscitation scenarios, where there is both urgency and impaired patient competence, are presumed consent and proxy consent. Presumed consent uses the concept that a reasonable patient under similar circumstances – or this patient if he or she were able to – would consent to the resuscitation endeavours proposed. This form of consent has merit and is commonly employed, but occasionally attracts criticism as being a form of medical paternalism, in that it may be perceived to be respecting the principle of beneficence, as the resucitators perceive it, while ignoring respect for patient autonomy.
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Proxy consent involves obtaining consent for resuscitation from a family member or other person who is perceived to be able to speak on behalf of the patient. Proxy consent avoids the criticism of medical paternalism as the decision is taken out of the physician’s hands, but it suffers as a model as the decision maker may be unable to adequately receive information, understand it, and deliberate over it during a hurried and rapidly evolving resuscitation. In addition, the proxy may not reflect the views of the patient. There may be occasional circumstances where the proxy declines resuscitation because of some financial or other benefit that would accrue from the patient’s death. More commonly, proxies have a tendency to demand more resuscitation than the patient would have wanted, for fear of becoming responsible for their death. When this form of consent is used there is greater scope for the harms of resuscitation. A modification of proxy consent that better addresses the issue of respect for patient autonomy is proxy consent with substituted judgement. This involves not asking what the proxy would want done for the patient, but instead what the proxy thinks the patient would want done. In other words, it attempts to see the resuscitation from the patient’s perspective as viewed by the proxy.

A modification of presumed consent is presumed consent using professional substituted judgement. This means the resuscitators gathering as much information about the patient as they possibly can to attempt to understand how the patient would view this decision. This usually involves speaking with the patient’s loved ones. Then, with some knowledge of the likely outcome of the resuscitation proposed, based on previous experience and a knowledge of the medical literature, they can exercise their moral imagination by asking ‘Would I want this treatment if I was this patient?’ In this way the patient’s autonomy is as best respected as it can be under difficult circumstances, by combining a knowledge of the harms and benefits of the resuscitation with an appreciation of this balance from the patient’s perspective. If presumed consent using professional substituted judgement is employed and the answer to the question is ‘No’, then the resuscitation cannot proceed. To resuscitate without regard for the patient’s explicit or perceived wishes is a harmful disrespect for their autonomy. Often, and appropriately, a decision to proceed will be made on the basis of a perceived marginal benefit over harm. This balance is made more appealing by the alternative of certain death if resuscitation is not undertaken. However, the balance is dynamic, with a clearer view of the likely benefits and harms emerging as the patient responds to the resuscitation. If the treatment does not procure the hoped-for benefits, all concerned should be willing to minimize the ongoing harms of resuscitation by withdrawing treatment as the balance becomes more unfavourable.

The concept of withholding and withdrawing treatment is somewhat misdirected in that it implies a need for permission to stop the intervention, whereas the precedent in medicine is to obtain permission to proceed. It is wrong to withhold a resuscitation endeavour because of the concern that the life-saving treatment cannot be withdrawn at a later date if things are not going well. When resuscitation is withheld a small but significant number of patients may miss out on an opportunity for a good outcome had the resuscitation been offered to them. Similarly, it is wrong to be unable to withdraw treatment because of the ill-conceived concept that once resuscitation has begun it must continue.

By employing professional substituted judgement, the resuscitators should recognize when the balance of benefit and harm becomes unfavourable from the patient’s perspective. At this point they have a moral obligation to withdraw resuscitation as they can no longer presume the patient’s consent. By appreciating the benefits and harms of resuscitation, the use of professional substituted judgement to view these from the patient’s perspective, and by a commitment to stop resuscitation when we cannot presume the patient’s consent, we will minimize the harms of resuscitation medicine.

PRACTISING RESUSCITATION PROCEDURES ON THE NEWLY DEAD

Practising resuscitation procedures – most commonly endotracheal intubation – on patients who have died after an unsuccessful resuscitation is a common practice in many parts of the world. However, some view this with a repugnance that can be rationally argued. Others would propose that the benefit of this practice to subsequent patients outweighs any repugnance felt by others who witness it, or any harm done to the recently deceased. However, like all other interventions in medicine, this procedure requires permission before it may proceed. Informed consent may be obtained from the terminally ill for permission to perform procedures after they die, but this has limited relevance to the practice as it occurs in many emergency departments. Implied consent argues that consent is implicit in the fact that the patient used the emergency services and, therefore, is agreeable to all that this entails, including being used for teaching. Implied consent criteria are commonly used for those who present of their own volition for non-invasive medical care. However, patients who die in the emergency department most often do not present of their own volition, but instead are brought in by others, usually ambulance staff, in a state of impaired competence. Furthermore, implied consent confers the right to administer treatment that the patient would reasonably expect at the time of presentation. Therefore, if a patient’s attendance is non-voluntary with impaired competence or with ignorance of the procedure, he or she cannot imply consent and we cannot infer it.

Construed consent is a modification of implied consent, suggesting that if consent was obtained for a procedure it can be construed for a related procedure. If we concede that a form of consent (presumed consent, as suggested above) is obtained to intubate a patient during resuscitation, may we construe that
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Consent also applies to intubation after death? There is a superficial logic to this, as to perform the same procedure on the same patient with the same equipment one minute before and one minute after death seems a continuum of the same therapeutic relationship. However, on close analysis there is a difference sufficiently significant to render previous consent null and void. The consent to resuscitate is based on a contract between medical staff and patient dedicated to helping the patient. When the objective is no longer to help the patient the previous contract is a violation of the trust inherent in the previously formed therapeutic relationship. An appreciation of this violation contributes to the repugnance of the procedure.

Presumed consent is appropriate when impaired competence renders the patient unable to give informed consent. Although it is likely that most would consent to postmortem procedures for the benefit of medical staff and subsequent patients, presumed consent does disadvantage the minority who would not. Formal application of a presumed consent rule for performing procedures on the recently dead mandates that the community should be well informed, so that individuals have the opportunity to explicitly decline consent if they so desire.

Proxy consent has also been argued in relation to this procedure. However, when proxy consent rules have been enforced the procedure tends not to occur, because staff are uncomfortable about obtaining consent in this way, or because relatives decline consent in an effort to protect their loved one from further harm.

The value of practising endotracheal intubation and other procedures on the newly dead is well argued and, therefore, there is a cost if it is disallowed. However, the current prevailing policy of ‘don’t ask, don’t tell’ is ethically unjustifiable. If we presume a patient’s consent and do not ask, we are obliged to tell. The significant minority that would not consent are thereby protected by an opportunity to decline. Therefore, to proceed with presumed consent we must have a well informed public, and preferably a statute to formalize consent. An extrapolation of this, which is the most convincing solution, is called ‘mandated choice’, which proposes a process whereby, as a matter of public policy, individuals must choose on a variety of issues and these choices are recorded on, for example, their driving licence. This process informs and honours individual choice, gives the significant minority the opportunity to decline, and avoids deception. However, in the absence of a suitably informed public from whom we can presume consent, or a mandated choice, we do not have permission to proceed with postmortem resuscitation practice. Therefore, to do so is ethically wrong.

CONCLUSION

Emergency medicine abounds with clinical dilemmas requiring ethical deliberation. Such deliberation may be influenced by theories regarding the consequences of action, theories based on moral principles, or some combination of these two. Beauchamp and Childress present a model for deliberation based on the principles of respect for autonomy, non-maleficence, beneficence and justice. Although this model frequently will not provide an answer beyond dispute, it does allow a rational examination of the important issues so that our consequential actions will at least be better directed than they might otherwise have been.

Resuscitation medicine demands such deliberation despite the pressure of urgency and the common impairment of patient competence. Patient autonomy must be respected by employing a suitable consent process, such as the use of presumed consent using professional substituted judgement. In this way we can attempt to honour the patient’s autonomy by viewing the benefits and harms of resuscitation from their perspective. Often, particularly in the early stages of resuscitation, the relative benefits and harms may be difficult to establish and the patient’s perspective may be difficult to formalize. It is appropriate to continue with resuscitation until these variables become more clear. However, as soon as there is a negative answer to the question ‘Would I want this done if I was this patient, knowing what I know about the patient and knowing what I know about the likely outcome?’, the resuscitators have a moral obligation to stop resuscitation.

CONTROVERSIES

1. Performing resuscitation procedures where a poor outcome is expected or where there may be reasons to suspect that the patient may not wish to be resuscitated.

2. Withholding resuscitation procedures on the basis of an argument of futility.

3. Practising procedures on the newly dead.

REFERENCES


