RESUSCITATION FROM CARDIAC ARREST

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In recent years, organisations such as the European Resuscitation Council, the American Heart Association and the International Liaison Committee on Resuscitation have produced guidelines in an attempt to improve the quality of cardiopulmonary resuscitation (CPR). They are based on international consensus views and the most recent of them, relating to Advanced Life Support, were published in 1998. The techniques of CPR, based on such guidelines, have now become a standard part of health professional training in many parts of the world.

The aim of this article is to provide an overview of resuscitation based on these guidelines and will be confined to the management of cardiac arrest including some comment on the more specialised areas of electrocution, drowning and arrests related to anaesthesia.

BACKGROUND PHYSIOLOGY AND PATHOPHYSIOLOGY

The maintenance of normal tissue metabolism relies principally on an adequate delivery of oxygen, in a functioning circulation. Failure of delivery rapidly results in the following changes:

Hypoxia After a brief period of cardiac arrest, P_aO_2 falls dramatically as oxygen continues to be consumed. In addition, progressive accumulation of carbon dioxide shifts the oxygen-haemoglobin dissociation curve to the right. This initially improves oxygen transfer to the tissues but without further delivery tissue hypoxia ensues. In the brain, the P_aO_2 falls from 13kPa to 2.5 kPa within 15 seconds and consciousness is lost. After a minute, the P_aO_2 will have fallen to zero.

Acidosis The brain and heart have a relatively high rate of oxygen consumption (4mls/min and 23mls/min respectively) and O_2 delivery to them will fall below critical levels during cardiac arrest. In the case of ventricular fibrillation, myocardial metabolism continues at an approximately normal rate, exhausting oxygen and high energy phosphate supplies. Acidosis then arises as the result of increased anaerobic metabolism and the accumulation of carbon dioxide in the tissues.

The degree of acidosis developing in the brain, even with basic life support, will threaten tissue survival within 5 - 6 minutes. Also, in the heart, even with the restoration of a perfusing rhythm, acidosis depresses contractility and there is a higher risk of further arrhythmias.

Cardiovascular collapse prompts a massive stress response. Catecholamines are released in large amounts, together with adrenal corticosteroids, anti-diuretic hormone and other hormonal responses. The possible detrimental effects of these changes include hyperglycaemia, hypokalaemia, increased lactate levels and a tendency towards further arrhythmias.

CAUSES OF CARDIAC ARREST

There are many causes of cardiac arrest. In the developed world most are related to ischaemic heart disease. Table 1 lists other common causes.

Table 1. Causes of Cardiac Arrest

Cardiac disease
- Ischaemic heart disease
- Acute circulatory obstruction
- Fixed output states
- Cardiomyopathies
- Myocarditis
- Trauma and tamponade
- Direct myocardial stimulation

Circulatory causes
- Hypovolaemia
- Tension pneumothorax
- Air or pulmonary embolism
- Vagal reflex mechanisms

Respiratory causes
- Hypoxia (usually causes asystole)
- Hypercapnia

Metabolic changes
- Potassium disturbances
- Acute hypercalcaemia
- Circulating catecholamines
- Hypothermia

Drug effects
- Direct pharmacological actions
- Secondary effects

Miscellaneous causes
- Electrocution
- Drowning

PREVENTION OF CARDIAC ARREST

Patients who develop a cardiac arrest may have been severely ill for some hours prior to the event. Warning signs such as: *hypotension, tachycardia, chest pain, dyspnoea, fever, restlessness or confusion* indicate that a patient is seriously ill. *Hypoxaemia, hypovolaemia and sepsis* may progress to cardiac arrest unless rapidly diagnosed and corrected. CPR for patients who are septic or hypovolaemic usually fails.

RESUSCITATION

The primary aim of resuscitation is to restore a beating heart and a functioning circulation. This article considers basic and advanced life support.

BASIC LIFE SUPPORT

Basic Life Support (BLS) establishes a clear airway followed by assisted ventilation and support of the circulation, all without the aid of specialised equipment. The recommended sequence for BLS is shown in figure1.

When approaching a patient who appears to have suffered a cardiac arrest the rescuer should check that there are no hazards to himself before proceeding to treat the patient. Although this rarely arises in hospital, patients may suffer a cardiac arrest due to electric shocks or toxic substances. In these situations the rescuer may be in considerable danger, and must ensure that any hazard is taken account of and eliminated as a risk.

Checking responsiveness is best done by speaking loudly to the casualty, and trying to rouse them by shaking a shoulder. If there is no response send for help as the patient is being treated.

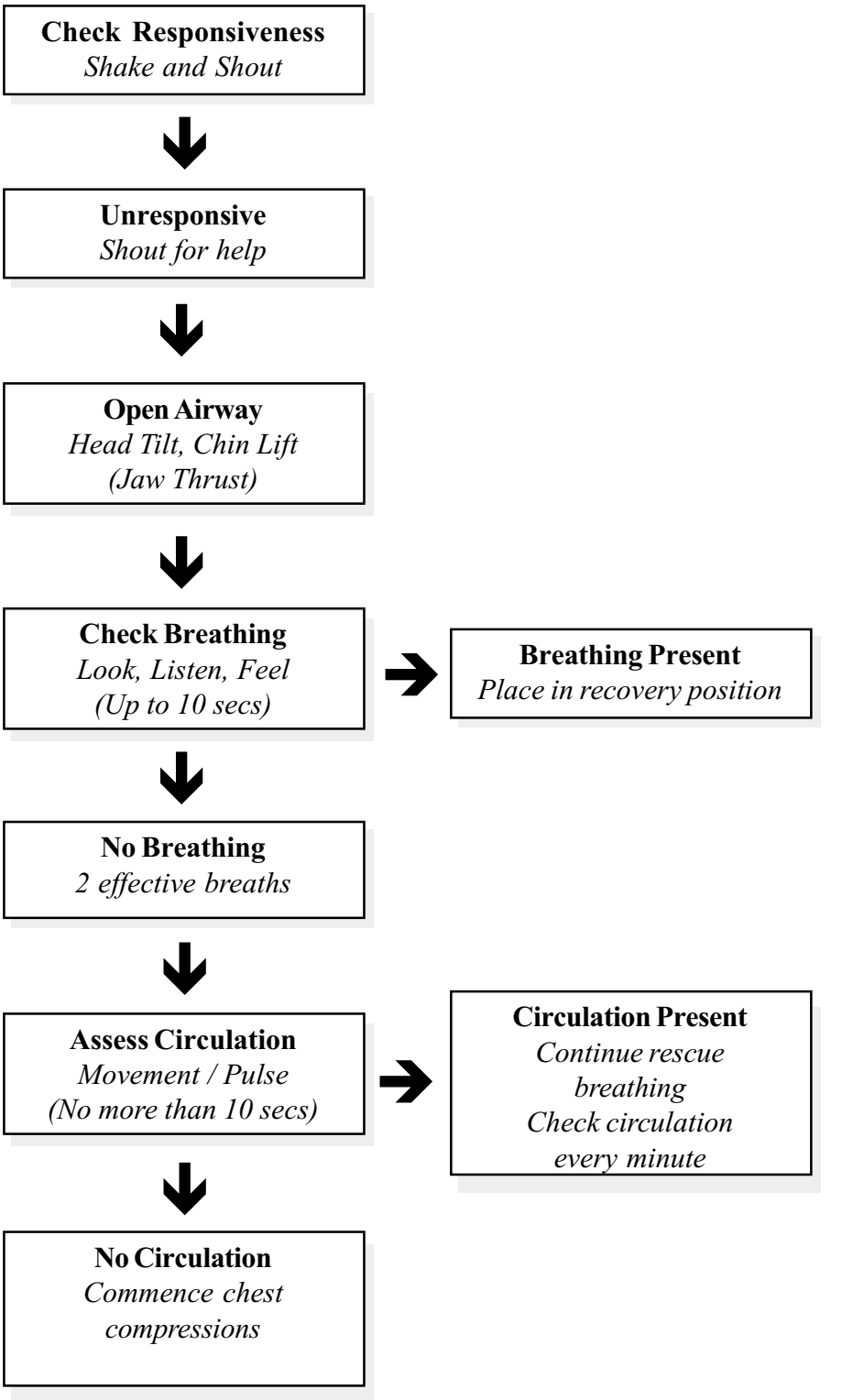
Opening the airway This can normally be done by simply extending the head and performing a chin lift. In some patients a jaw thrust will be required along with the insertion of an oropharyngeal airway. False teeth that are loose or other debris within the airway should be removed.

Assisted ventilation should be provided if the patient is not breathing. It may be provided using expired air ventilation (mouth to mouth, mouth to nose, using a Laerdel pocket mask) or by using a self inflating bags, usually with supplemental oxygen. Oxygen should be added to self inflating bag, using a reservoir on the inlet side of the bag. Adequacy of ventilation is judged by each breath producing adequate movement of the chest on inspiration. In general tidal volumes of 400 – 500mls are optimal.

Chest compressions (previously known as **cardiac massage**) are used whenever a central pulse (carotid) is absent. The technique creates positive pressure within the chest and forces blood out of the chest during the compression phase. Due to the valves within the venous system and the heart, most of the blood flows forward through the arteries. When the chest recoils to its normal position blood returns to the chest from the venous side of the circulation. A small amount of flow is produced by direct compression of the heart between the sternum and the spine. During chest compressions approximately 25% of the normal cardiac output is produced.

Current guidelines advise that 5 chest compressions are carried out for each ventilation when two rescuers are available. In the event of only one rescuer, 15 compressions

ADULT BASIC LIFE SUPPORT



Send or go for assistance as soon as possible according to guidelines

Figure 1: Recommended sequence for BLS

should be followed by 2 ventilations. The overall rate of chest compressions should be 100/minute.

When starting chest compressions:

- Get the patient on a firm surface
- Feel the xiphisternum, and measure 2 finger breadths up on the sternum (figure 2). Without moving your fingers, place the heel of the second hand on the sternum. Put both hands together as shown in figure 3 and depress the sternum 4-5cm in an adult.
- Keep your elbows (figure 4) straight, and ensure that all the pressure is directed through the sternum and not through the ribs. To perform chest compressions adequately, it is necessary to be above the patient. Stand on a platform if necessary.
- During a cardiac arrest change the person performing chest compressions regularly, as it is tiring when performed properly.
- The rescuer performing chest compressions should count out loud "1,2,3,4,5", and the rescuer ventilating the patient should count the number of cycles completed.

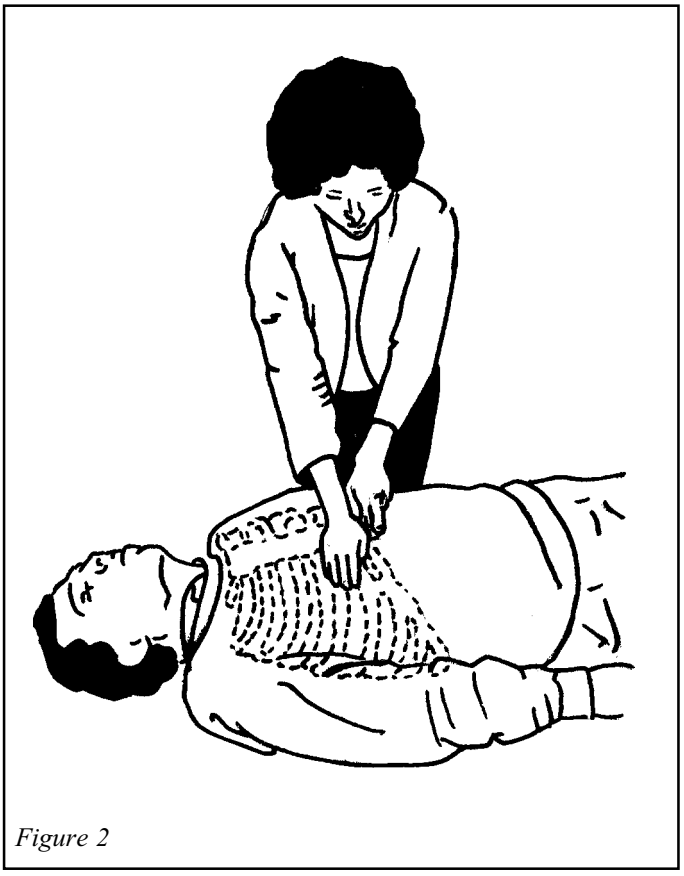


Figure 2

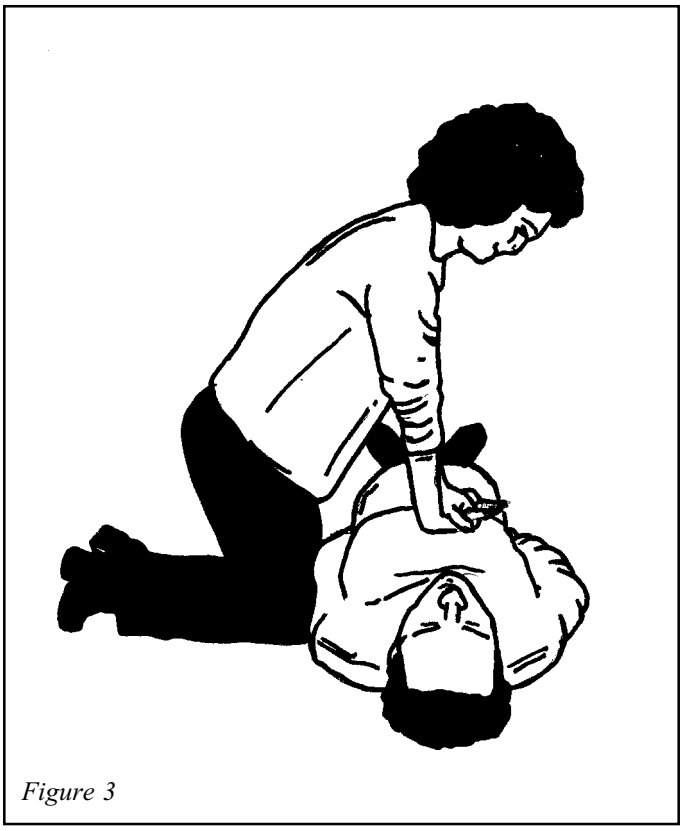


Figure 3

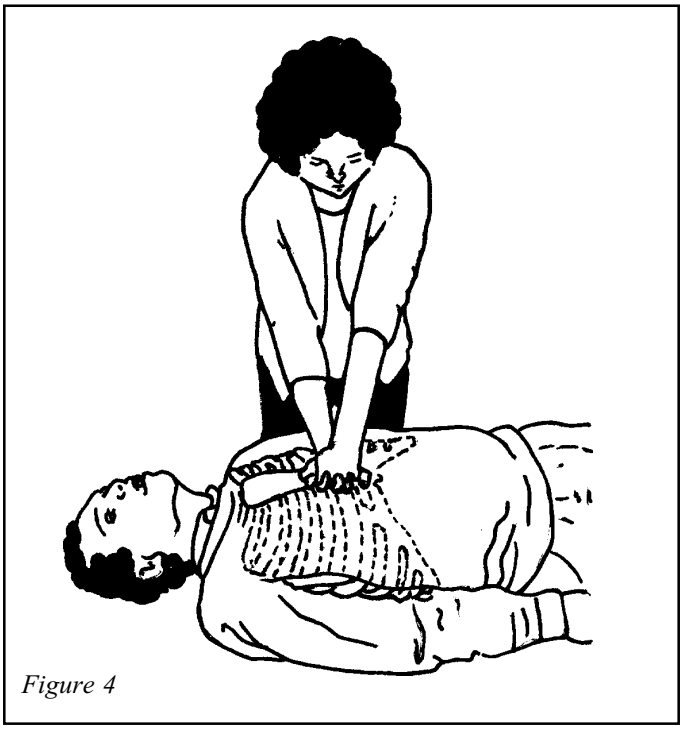


Figure 4

Early BLS has been shown to improve outcome, particularly when access to advanced airway management and defibrillation is likely to be delayed. Although the barely adequate level of oxygen delivery achieved during BLS may be regarded as a holding measure, it is of great importance and will occasionally reverse the primary cause of the cardiac arrest and restore some circulation preventing the rhythm degenerating into asystole.

Table. 2. General Management Principles for Cardiac Arrest

1. Establish the safety of the victim and potential rescuer.
2. Confirm the diagnosis of an arrest
3. Send for help
4. Establish Basic Life Support
5. Aim for early and frequent defibrillation if indicated, with regular doses of adrenaline and CPR.
6. If there is doubt about the rhythm, or no ECG monitor is available, treat adults as being in VF.
7. Except for defibrillation, chest compressions should not be interrupted for more than 10 seconds to allow invasive procedures or advanced airway management.
8. Administer drugs intravenously whenever possible. Use a 20-50ml 0.9% saline flush with the peripheral route.
9. Consider and treat any underlying causes
10. Consider antiarrhythmic drugs and sodium bicarbonate as described below.

ADVANCED LIFE SUPPORT

Advanced Life Support refers to the use of specialised techniques, in an attempt to rapidly restore an effective rhythm to the heart. The most important components of the advanced life support techniques are direct current defibrillation and efficient BLS. The general principles involved with resuscitation from a cardiac arrest are shown in Table 2, and each technique involved with ALS is described below.

SPECIALISED TECHNIQUES IN ADVANCED LIFE SUPPORT

Advanced Airway Management

Advanced airway management requires specialised equipment and skills and should be used in an apnoeic patient receiving basic life support.

Oral and nasopharyngeal airways are easy to insert with minimal experience. The commonest forms are the Guedel oropharyngeal airway and the more easily tolerated nasopharyngeal airway.

An oropharyngeal airway is sized by matching the distance between the corner of the mouth and the angle of the jaw. The nasopharyngeal airway is matched approximately to the diameter of the patients little finger and should be well lubricated before insertion. Do not use a nasopharyngeal airway if there is any suspicion of a basal skull fracture.

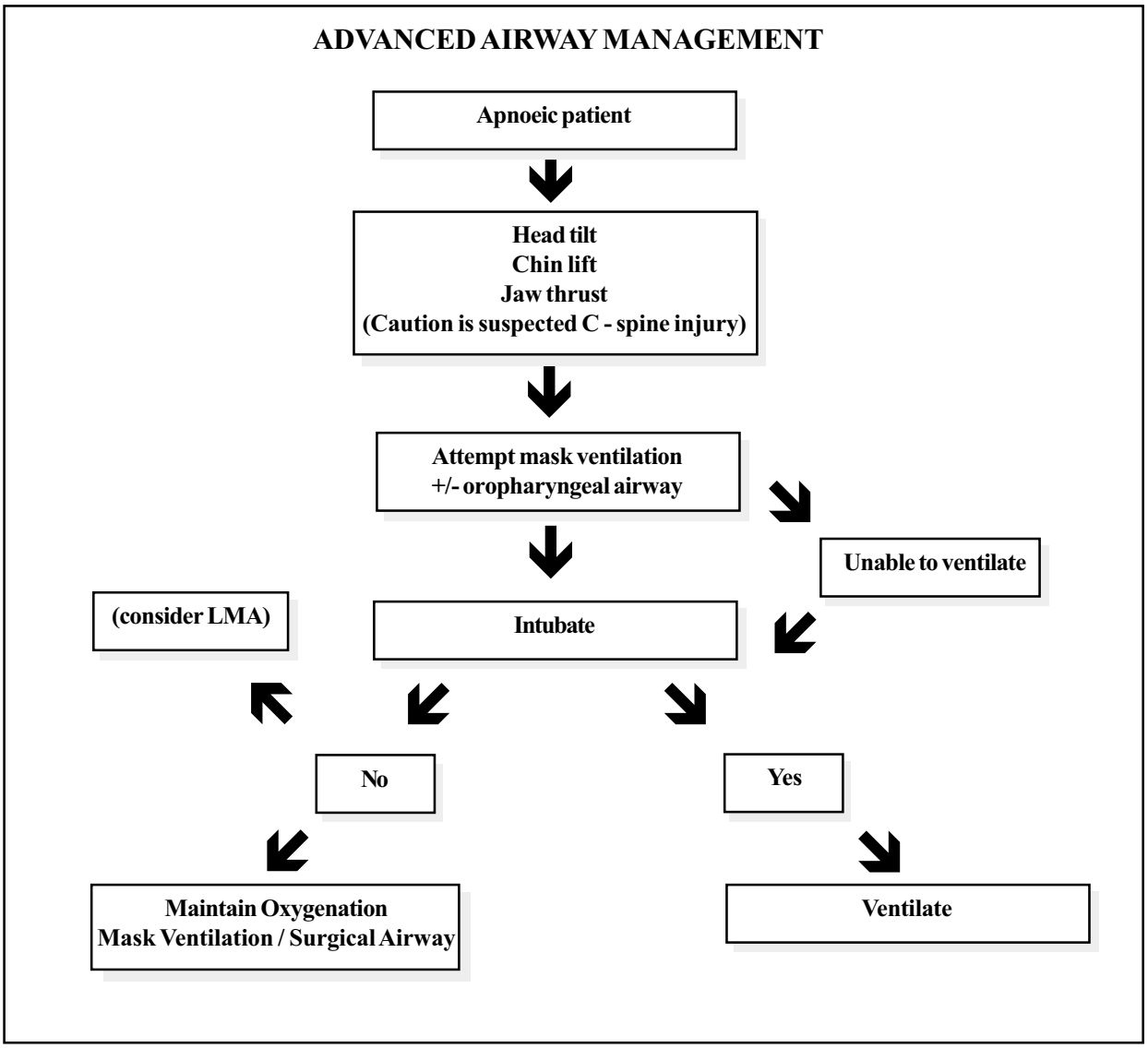
Tracheal intubation is the best way of providing a secure and reliable airway. However, the technique requires special skills and equipment and attempts at intubation may cause further complications and delay if performed incorrectly. Confirmation of the tube's position is most reliably achieved by seeing it pass between the cords during intubation, auscultation of the chest and, if available, end-tidal carbon dioxide measurement. Various simple oesophageal detector devices are also available (see Update 1997;7:30).

Application of cricoid pressure should be considered if there is a major risk of gastric contents contaminating the airway. It should be applied until the airway is secured with a cuffed endotracheal tube. However it may make intubation more difficult for the inexperienced operator, particularly if it is not done completely correctly (see Update 1994;4:1-5).

Other oropharyngeal airway devices

Although it has been part of routine anaesthetic practice for around ten years in the UK, the laryngeal mask airway (LMA) has also been used for failed intubation and, more recently in resuscitation.

The insertion technique is easily taught and it provides a similarly efficiency of ventilation as a bag and mask technique. However in a few cases LMA's are difficult to position correctly, ventilation of poorly compliant lungs is uncertain, and they do not reliably protect the airway from gastric contents. The double lumen Combitube® has also been used during resuscitation. It is inserted blindly into the oesophagus and then used to inflate the lungs via the second lumen. See Update 1998;9:37-45 for more details of the LMA and the Combitube.



Flow chart for the advanced management of the airway and ventilation

Surgical airway techniques are required when life-threatening airway obstruction is present and other means of establishing an airway have failed. Emergency access to the airway is via the relatively avascular cricothyroid membrane. This membrane is identified by locating the midline depression between the easily identifiable cricoid cartilage and the lower edge of the thyroid cartilage.

Cricothyroid emergency airway. A 12 or 14 gauge cannula with a syringe attached is introduced through the cricothyroid membrane until air can be aspirated. The cannula is then advanced off the needle down the trachea. The hub of the cannula is connected to an oxygen source at 15 litres/minute and the patient ventilated for one second and then allowed to exhale for 4 seconds. In the absence of an oxygen supply, short-term improvised connections can be made by:

- The cricothyroid cannula is connected to a 10ml syringe with the plunger removed. An 8.0mm endotracheal tube is inserted into the barrel, the cuff inflated and a self-inflating bag connected and ventilation attempted.
- A 3.5mm endotracheal tube connector will usually fit directly into the cricothyroid cannula and allow connection to a self inflating bag.

Although the patient may be oxygenated in this way, ventilation to remove CO_2 cannot be achieved and respiratory acidosis will ensue. Spontaneous respiration is impossible through a needle cricothyrotomy and careful observation is required to prevent barotrauma. A clear expiratory pathway is required to allow the oxygen to escape as an intravenous cannula is not adequate by itself.

A needle cricothyrotomy will ensure a supply of oxygen for a maximum of 10-20 minutes and it should be converted to a surgical cricothyrotomy to allow adequate ventilation. A horizontal incision is made through the membrane and a small (size 5.0-6.5) endotracheal or tracheostomy tube is inserted and connected to a self inflating bag, providing highly efficient ventilation and airway security. Although a simple concept, the equipment may take time to assemble and there is a significant complication and failure rate. Therefore in theatre, or accident/ emergency, the equipment should be prepared and ready for use.

Blind, single-stage cricothyrotomy techniques
Several kits are commercially available (Portex, Cook Critical Care, Rusch) which are designed to pass a tube through the cricothyroid membrane in a single manoeuvre. They use either a guidewire, introducer or dilational technique and all provide a 22mm connection to standard ventilation equipment.

Defibrillation
The majority of adult cardiac arrests involve ventricular fibrillation that may be reversed by electrical defibrillation. The likelihood of successful defibrillation decreases with the duration of cardiac arrest (an estimated 2 - 7% for every minute of the arrest) and, although BLS measures will slow the deterioration, asystole will inevitably ensue.

Defibrillation delivers an electrical current through the heart simultaneously depolarising a critical mass of the myocardium and introducing a co-ordinated absolute refractory period. This results in a period during which another action potential cannot be triggered by a stimulus of any magnitude and, if successful will stop the chaotic electrical activity of ventricular fibrillation momentarily. The pacemaker cells of the sino-atrial (SA) node have the opportunity to re-establish sinus rhythm as they are the earliest myocardial cells to depolarise spontaneously.

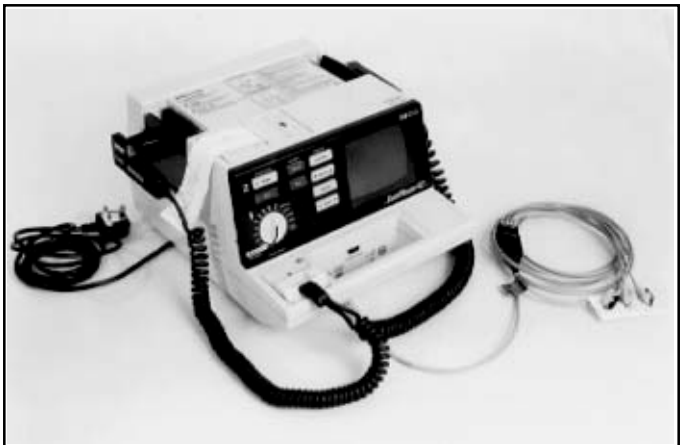


Figure 5: A defibrillator

All defibrillators consist of a power source, an energy selector, an AC/DC converter, a capacitor and a set of electrode paddles (Figure 5). Modern machines allow ECG monitoring via the paddles or via leads attached to the machine. The power output is expressed in terms of delivered energy (in Joules), which is the energy delivered through the paddles to the chest wall.

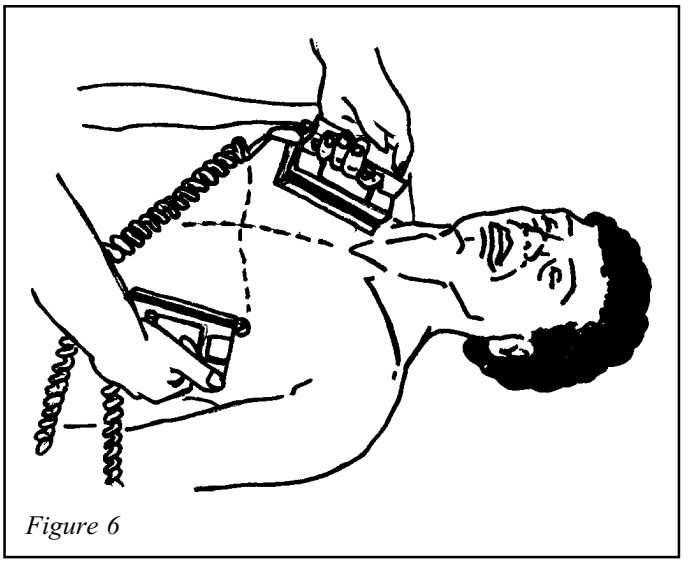


Figure 6

Only a relatively small proportion of the energy is delivered to the heart and variations in transthoracic impedance (resistance to current flow caused by chest tissues) will occur. The energy requirement for defibrillation (defibrillation threshold) will tend to increase with the duration of the arrest. Empirical energy levels of 200 Joules (J) for the first two shocks and 360J subsequently have been decided upon for adult resuscitation. DC shocks should be delivered with the correct paddle position and good contact using conductive pads or a coupling medium. Although the polarity of the paddles is not crucial, the cardiac complexes are upright on the screen if the paddles labelled "sternum" and "apex" are placed correctly. The sternal paddle is placed high on the right side of the anterior chest wall, lateral to the upper part of the sternum and below the clavicle; the apex paddle is placed just lateral to the position of the normal apex beat (figure 6), avoiding breast tissue in females. Other positions, such as apex-posterior may be tried if the conventional paddle position is not successful.

In recent years, semi and fully automatic defibrillators have been developed. When connected to the patient these are able to interpret cardiac rhythms and deliver shocks when

DEFIBRILLATION SEQUENCE

Action	Announcements
1. Switch on.	
2. Place coupling pads/gel in correct position	
3. Apply paddles	
4. Check ECG rhythm and confirm no pulse	
5. Select non-synchronised (VF) setting	
6. Charge to required energy level	*"Charging"*
7. Ensure no-one is in contact with anything touching the patient	*"Stand clear"*
8. Press paddle buttons simultaneously	*"Shocking now"*
9. Check ECG rhythm	
10. Check for output if rhythm change	*"Check pulse"*
11. Return to ALS algorithm for further steps	

appropriate. Some are also able to measure the transthoracic impedance of the patient and attempt to match the energy delivery to the required current flow. The very latest generation of machines use bi- and tri-phasic energy wave forms to achieve successful defibrillation at lower energy levels.

Regardless of the type of defibrillator available, it is essential that the staff using it are familiar with its operation, and are trained regularly in its use.

Cardiac Arrest: Defibrillation Technique

It is assumed that the rhythm has been confirmed as suitable for defibrillation. The first three shocks of the ALS algorithm should be completed within 90 seconds. Unless the rhythm changes on the ECG trace, there is no need to check the pulse between cycles of defibrillation.

Drug Therapy

Adrenaline (epinephrine) is the main drug used during resuscitation from cardiac arrest. A 1mg dose should be given at least every three minutes during the arrest. Intravenous adrenaline enhances cerebral and myocardial blood flow by increasing peripheral vascular resistance and raising aortic diastolic pressure. These peripheral vascular actions are primarily alpha1 ($\alpha 1$), and alpha2 ($\alpha 2$), receptor-mediated. Beta1($\beta 1$) and Beta2 ($\beta 2$) receptor actions also occur though a beta effect has not been shown to be beneficial in restoring spontaneous circulation in VF, asystole or EMD. Indeed, $\beta 1$ effects may increase myocardial oxygen demand and increase the risk of arrhythmias in a beating heart. Recently, high dose adrenaline (5mg) has been tried during resuscitation in an attempt to improve the survival of cardiac arrest but there was no improvement in outcome.

The ALS algorithm suggests the use of antiarrhythmics, buffers, atropine and pacing. Antiarrhythmic drugs are considered on page 30.

Atropine as a single dose of 3mg is sufficient to block vagal tone completely and should be used once in cases of asystole. It is also indicated for symptomatic bradycardia in a dose of 0.5mg – 1mg.

Sodium bicarbonate In prolonged arrests, the effects of acidosis become significant. The use of sodium bicarbonate as a buffer has been controversial; it is associated with hyperosmolarity and carbon dioxide production, and may worsen intra-cellular acidosis. Carbon dioxide-consuming buffers, such as Carbicarb and THAM have been developed, but no buffer has been shown to improve outcome. Nevertheless, sodium bicarbonate continues to be recommended (50mls of 8.4% solution) after 15 minutes of cardiac arrest or when the arterial pH is less than 7.1, or the base deficit is more negative than –10. It should be used early in arrests caused by acidosis, hyperkalaemia or tricyclic overdosage, but must not be given by the tracheal route or mixed with calcium or adrenaline solutions.

Drug Delivery

The optimal route of administration for these drugs is via a central venous cannula. However, they are usually given through a peripheral cannula and in this situation, drug

administration should be followed by a 20-50ml 0.9% saline flush and elevation of the limb to assist entry to the central circulation.

CPR should not be interrupted for more than 10 seconds to permit intravenous cannulation and consideration should be given to the tracheal route if no intravenous access exists. Although a second-line choice, endotracheal tube placement will often precede intravenous access and adrenaline, atropine and lignocaine can all be given intra-tracheal in doses 2 times the normal intravenous dose, diluted up to 10mls in 0.9% saline. When gaining iv access during a cardiac arrest, choose the most proximal large vein that can be easily cannulated: the external jugular vein is often suitable. ***Central venous cannulation should only be attempted by those experienced in the technique.***

The unpredictable drug delivery and risk of damage to the left anterior descending coronary artery make direct intra-cardiac injection impractical and unsafe.

Advanced Life Support Algorithm

The algorithm (figure 7) guides the response to cardiac arrest. If the arrest is witnessed, a precordial thump should be considered. This is delivered with a heel of a clenched fist from a height of around 8 inches from the chest. This generates a few joules of electrical current within the heart which, in the early phase of a cardiac arrest, may be enough to return sinus rhythm. A precordial thump should not be administered by people who have not been trained in the technique, or if the arrest has not been witnessed. Perform a pulse check after delivering a precordial thump.

The priority in advanced life support is to determine the underlying rhythm causing the cardiac arrest and whether any underlying treatable cause can be found. The algorithm details the management according to whether the underlying rhythm falls into the category of Ventricular Fibrillation (VF) / Pulseless Ventricular Tachycardia (VT) or, Asystole / Pulseless Electrical Activity (in the figure = Non VF /VT).

Ventricular Fibrillation or Pulseless Ventricular Tachycardia

When VF or VT are diagnosed the patient should be defibrillated as quickly as possible using three shocks of 200J, 200J then 360J. Unless the rhythm changes on the ECG trace, there is no point in checking the pulse between shocks as this will delay the next defibrillation attempt. Palpation of a major artery is carried out if the ECG appearances are compatible with an output, or if purposeful movements are made. If these shocks are not successful, CPR should be resumed for one minute while the airway is secured and iv access is achieved. A dose of iv adrenaline (1mg) is injected and consideration is given to any specifically treatable causes of VF such as hypothermia and toxins. After another 10 cycles of CPR, the ECG trace is re-examined. Persistent VF is treated with a further three shocks of 360J as required. These take priority over any continuing attempts at securing the airway or establishing iv access. It is recommended that this sequence is followed for at least 9-12 shocks before consideration is given to the use of antiarrythmic drugs. Adrenaline should be administered every 2 –3 minutes during resuscitation.

If there is no cardiac monitor but a defibrillator is available, it is better to treat the rhythm as VF, as this rhythm has the best prognosis.

Asystole or Pulseless Electrical Activity (PEA)

Asystole occurs when there is no detectable electrical activity in the heart and is associated with a very poor prognosis. Pulseless Electrical Activity (or Electro-Mechanical Dissociation - EMD) is present when the ECG shows a rhythm normally associated with an output but with no detectable central pulse. In either case, the defibrillation-based treatment loop is not appropriate.

In asystole or PEA treatment options are more limited. The right-hand loop of the algorithm is followed. The airway is secured and iv access obtained as soon as possible and CPR is continued with doses of adrenaline administered every three minutes. Atropine (3mg) is given once in asystole. The chance of surviving asystole or EMD is improved if a reversible cause can be identified which can be treated. The most likely ones are listed in the algorithm. Acute hypovolaemia is the most commonly treatable cause, and always results from extremely severe haemorrhage (>50% blood volume). These patients usually need immediate surgery to control haemorrhage and rapid fluid replacement. Any change of the ECG consistent with VF should prompt an immediate transfer to the other treatment loop.

Stopping Resuscitation

The decision to stop resuscitation attempts is usually made by the team treating the arrest. It is usually the responsibility of the most experienced doctor present and should involve the whole team. Patients in asystole or PEA, who have no underlying cause diagnosed, and who do not respond to BLS and adrenaline, have a very poor prognosis and in our experience resuscitation attempts are normally stopped after 10 – 15 minutes.

The ALS Algorithm for the management of Cardiac Arrest in Adults

Note that each successive step is based on the assumption that the one before has been successful.

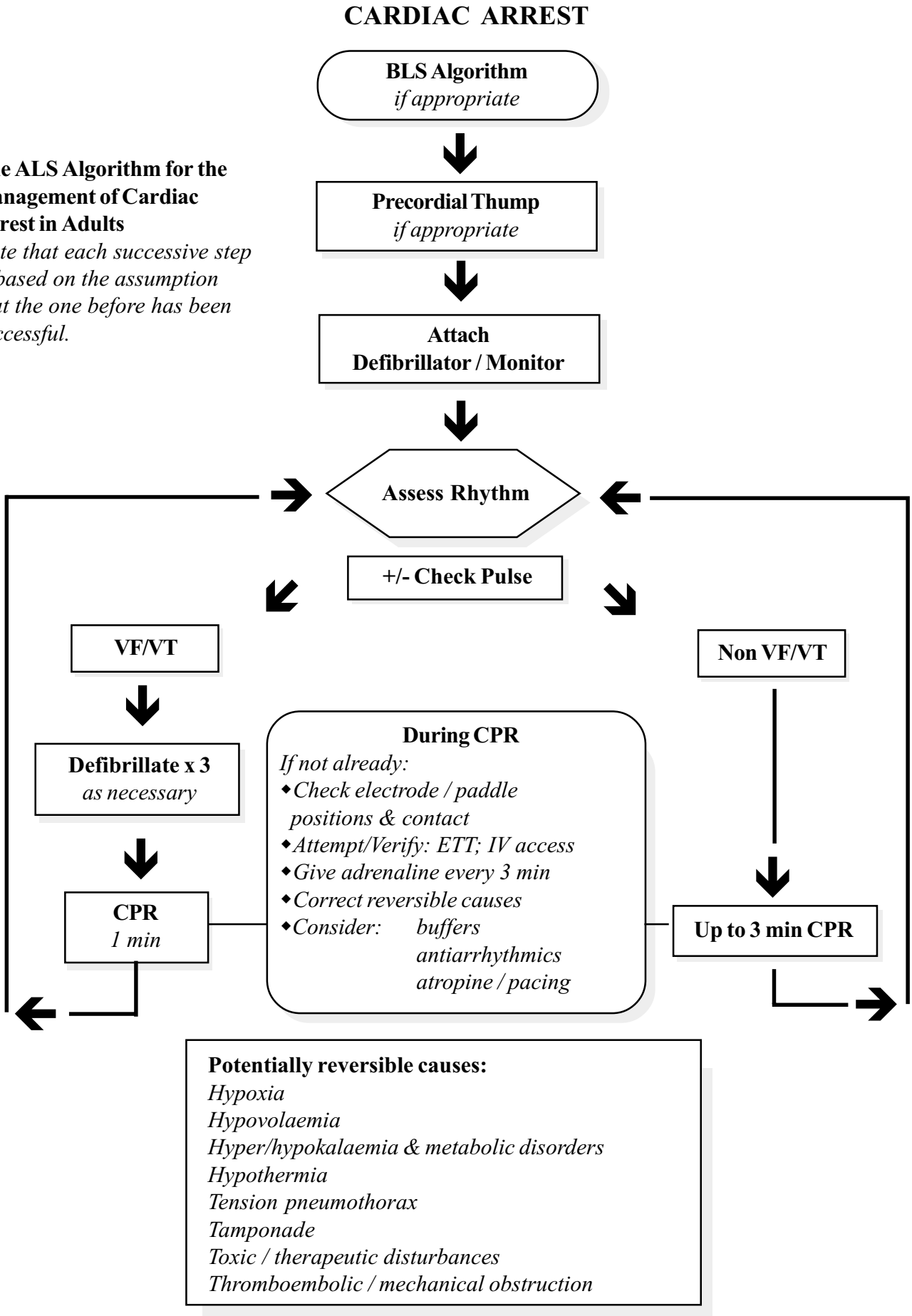


Figure 7: ALS flowchart

Treatment would normally be continued while the ECG trace indicates the presence of VF. However successful resuscitation becomes unlikely after 12 shocks, and rare after 15 – 20 minutes of attempted resuscitation. The highest survival rates occur in witnessed VF arrests, when BLS has been started immediately and defibrillation is very rapid. Outcome studies from VF carried out in hospitals in the developed world in the 1990's indicated an initial resuscitation success rate of up to 50% but a survival to discharge rate of up to only 20% in this population.

Patients who have severe underlying illnesses or terminal conditions usually have a cardiac arrest as a terminal event and resuscitation in these patients is usually unsuccessful, and often inappropriate. In many hospitals patients in this category may be designated "Not for Resuscitation" after discussion with the relatives and or the patient, and the medical and nursing teams caring for them. The legal position of such decisions, and the methods for making them varies from country to country.

Patients who suffer unwitnessed cardiac arrrests and have delayed BLS / defibrillation as a result have a dismal outlook and resuscitation attempts will be unsuccessful in most cases.

Managing Cardiac Arrests without a Defibrillator.
Clearly without the aid of a defibrillator, cardiac arrest management is more limited and the diagnosis and treatment of the likely underlying problem provides the best chance of survival. Basic life support should be initiated, adrenaline given and resuscitation attempted while any reversible factors (such as hypovolaemia) are diagnosed and treated.

Other Antiarrhythmic Therapy
Although the defibrillator remains the main technique, a number of antiarrhythmic drugs may prove useful. They may be used to treat a persistent, life-threatening arrhythmia, to lower the threshold for successful defibrillation or as prophylaxis against further rhythm disturbances.

Each agent has specific indications but most are negatively inotropic - clearly undesirable in resuscitation. Lignocaine, bretylium, amiodarone and magnesium are the most commonly used agents. There is a lack of human-based evidence for their effectiveness, reflecting the difficulty in performing meaningful clinical studies in resuscitation.

Lignocaine (lidocaine) has antiarrhythmic properties derived from sodium channel blockade, resulting in membrane stabilisation. The pacemaker action of the SA node is suppressed and conduction within the ventricular muscle is inhibited. There is little effect on the atrio-ventricular (AV) node and its myocardial depressant and pro-arrhythmic effects are minimal.

Lignocaine is established for the treatment of ventricular tachycardia. The ability of lignocaine to improve the chances of successful defibrillation of persistent VF is less certain, but it is often tried when repeated unsuccessful attempts at defibrillation have been made. Lignocaine is also used to treat haemodynamically stable VT.

The dose of lignocaine for refractory ventricular fibrillation is 100mg iv and for haemodynamically stable ventricular tachycardia is 1mg/kg iv – repeated once if necessary - and followed by an intravenous infusion of 4mg/min for 30 minutes, 2 mg/min for 2 hours and then 1mg/minute.

Amiodarone produces potassium channel blockade with some inhibition of sodium channel mediated depolarisation, a lengthening of the myocardial action potential and a degree of ß-blockade. This gives it gives it an antifibrillatory action and lowers the defibrillation threshold with a minimal effect on myocardial contractility.

Its routine use during cardiac arrest is yet to be proven and it is generally reserved for the second-line treatment of peri-arrest tachyarrhythmias. Amiodarone is preferably administered centrally and slowly. Usually a 300mg loading dose is given over one hour followed by an infusion of 900mg in 1000ml of 5% glucose over the following 24hrs. In more urgent situations, the first 300mg dose can be given peripherally over 5-15 minutes and followed by a further 300mg over one hour.

Bretylium tosylate stabilises the action potential duration throughout the myocardium. This increases the resistance to VF and lowers the defibrillation threshold. However it is slow to act (15-20minutes) and there is a tendency for it to produce pulseless electrical activity and greater post-arrest hypotension that expected.

Magnesium is a critical factor in myocardial cell stability. A decreased intracellular level promotes myocardial excitability but, even in the absence of a low magnesium level, a bolus of iv magnesium will suppress ventricular ectopic beats. The use of magnesium in cardiac arrest is unproven, but it may be useful when hypokalaemia may have contributed to the arrest and a dose of 10mls of 50% magnesium sulphate may be given if this is the case.

Calcium has a specific indication as emergency protection against the effects of hyperkalaemia or the unusual condition of calcium channel blocker (eg verapamil) overdose. Despite its crucial role in the myocardial action

potential and contraction, its administration for any other reason appears to be ineffective, or even detrimental, as high intracellular calcium concentration are damaging to injured myocardial and neuronal cells. However, if the serum potassium level is above 6mmol/L, 10ml of 10% calcium chloride should be given.

Although the individual drugs are chosen largely for their lack of effect on myocardial contractility, administration of several antiarrhythmic agents will result in a cumulative, deleterious effect even if a perfusing rhythm is restored.

If resuscitation is successful, arrythmias remain a likely sequel. The management of subsequent brady-arrythmias and narrow or broad complex tachycardias are beyond the scope of this article.

Cardiac Arrests in Special Circumstances

There are a few circumstances in which the ALS principles need adapted.

Drowning and near-drowning Victims of immersion who are in cardiac arrest on arrival in hospital are a difficult and controversial group to treat. Occasional reports of apparently miraculous recovery after prolonged immersion and resuscitation have been made, particularly in children. These cases have involved rapid, profound cooling in **very cold water**. Children transferred rapidly to hospital who have suffered a short duration of immersion in cold water should have rectal core temperature measurement and ECG monitoring established immediately. Often asystole is present but occasionally the ECG will show slow sinus rhythm when the patient appears to be dead. If it is thought worth attempting resuscitation:

- Resuscitation should follow standard principles with BLS.
- Early intubation and ventilation with 100% oxygen should be a priority and prolonged BLS may be needed while attempts are made to rewarm the victim to 31^0C as attempts to defibrillate the hypothermic heart below this temperature are unlikely to be successful
- Although rewarming the patient can be extremely difficult and sometimes impossible without facilities for cardio-pulmonary bypass, resuscitation should not normally cease until the core temperature has reached 31^0C or attempts to achieve this have failed.
- Surface warming, heated inspired gases, warm iv fluids and intra-gastric balloons are of limited value, but must be tried. Warmed peritoneal dialysis has been recommended.

The prognosis for victims of drowning discovered in cardiac arrest is very poor. Most will die or be significantly brain damaged. With few signs to indicate the speed of cooling, triage can be difficult. Hyperkalaemia is caused by the pre-hypothermic phase of the cardiac arrest and serum potassium of >10mmol/L measured during resuscitation is incompatible with survival.

Electrocution

The effects of electrocution depend on the conversion of electrical energy into heat energy. The degree of damage depends on:

- Energy delivered
- Tissue resistance to current flow
- Type of current. Alternating current (AC) is more dangerous. It is more likely to reach central tissues and the resulting tetanic muscle contractions prevent the victim from releasing the electrical source.
- Current pathway through the body.

Asystolic arrests are more likely with currents greater than 10 Amps but VT and VF are also common. ***Rescuers must take great care to avoid receiving an electric shock.***

Drug overdose

Deliberate overdose or poisoning should always be considered in an unconscious patient. Cardiac arrhythmias or haemodynamic effects are particularly associated with certain drugs and may require specific treatment or prolonged resuscitation. Cardiac arrhythmias following a tricyclic overdose may respond to an infusion of sodium bicarbonate to maintain the pH within the high normal range and also potassium to keep the serum potassium >4.0mmol. Bupivacaine is able to bind to the myocardium and following a cardiac arrest due to toxicity from this drug, resuscitation should be prolonged (1 hour).

Cardiovascular Features of Common Drug Poisoning

Tachyarrhythmias
- anticholinergics
- tricyclic antidepressants
- cardiac glycosides
- chloral hydrate
- local anaesthetics especially bupivacaine

Bradyarrhythmias
- cardiac glycosides
- organophosphates
- calcium channel blockers
- chloroquine

Asystole
- cyanide

Hypotension
- barbiturates
- chloroquine
- theophylline

Cardiac Arrests during Anaesthesia

The management of intra-operative cardiac arrests differs from the standard guidelines in that the event is normally witnessed and some form of airway maintenance and intravenous access has already been established. A primary cardiac event may be the cause, but treatment is often needed for an underlying problem such as vagal stimulation, blood loss, hypoxia, bronchospasm, myocardial depression, hypokalaemia, hyperkalaemia etc. The most common cause of cardiac arrests during anaesthesia are hypoxia, vagal stimulation or hypovolaemia. Most can be prevented by careful anaesthesia and close clinical monitoring. Specific treatment should be towards the underlying cause as well as initiating resuscitation procedures.

POST-ARREST MANAGEMENT

Following the restoration of a spontaneous cardiac output, the metabolic changes and the likelihood of the injured heart developing arrhythmias make further monitoring and intensive care essential. There may also be a need to provide a period of brain protection to maximise chances

Special Points for Intra-Operative Arrests (to be used in conjunction with normal guidelines and treatment of the specific problem if known)

1. Stop all anaesthetic agents, administer 100% oxygen and ventilate the lungs.
2. Ask the surgical team to begin chest compressions at 5 chest compressions to 1 ventilation.
3. If the patient is pregnant, create at least $10\text{-}15^0$ of left lateral tilt to allow CPR to be effective. Deliver fetus.
4. Commence ECG and end-tidal CO_2 monitoring if not already in place.
5. Convert the airway to a tracheal tube and check its position and patency:
   - Observe chest movement.
   - Auscultate the chest and clinically exclude a pneumothorax.
   - Observe the end-tidal CO_2 output if available.
   - If in any doubt, change the tube.
6. Check the oxygen supply. If in doubt, change to a cylinder or air.
7. Check the fresh gas delivery from the ventilator to the patient. If in doubt, change to a self-inflating bag with an oxygen reservoir
8. If possible, send blood for arterial blood gases (ABGs), electrolytes and calcium. Aim to repeat ABGs, acid base values and potassium every 10-15minutes.
9. If the arrest is accompanied by significant hypothermia or is due to local anaesthetic toxicity, resuscitation is likely to be prolonged.

of recovery. To allow this, the optimum place for a recovering arrest victim is in a high dependency, coronary care or intensive care area. Even if successful resuscitation is achieved very rapidly, the heart may still be significantly damaged and is at risk of further arrhythmias.

If the patient is alert, maintaining an airway and breathing adequately, he may be extubated and admitted to a coronary care unit for monitoring and observation. Support of the cardiac output and circulation may be required with the guidance of invasive monitoring, including central venous cannulation, if available. Monitoring of end organ function, such as urine output is also required. Post cardiac arrest investigations should include serial 12-lead ECGs, CXR and basic blood tests including electrolytes, full blood count, magnesium and cardiac enzyme measurement if available. In the case of a proven myocardial infarction (ECG, enzymes) streptokinase may be considered.

If the resuscitation was prolonged, there is a significant metabolic disturbance, if the patient is cerebrally obtunded or requires a high level of inotropic support, admission to an intensive therapy unit for mechanical ventilation may be indicated, depending on the patient's prognosis and available facilities. The detailed management of post resuscitation care is beyond the scope of this article. Failure to recover consciousness 24hours after resuscitation indicates a poor prognosis.

Further Reading

ABC of Resuscitation. British Medical Association UK

Advanced Life Support Course Provider Manual (3rd edition) Resuscitation Council UK1998.

European Resuscitation Council Guidelines for Resuscitation. European Resuscitation Council (1998). Ed. L. Bossaert. Elseviar.