CHAPTER

6

The management of cardiac arrest

LEARNING OBJECTIVES

In this chapter you will learn:

- How to assess the cardiac arrest rhythm and perform advanced life support

6.1. INTRODUCTION

Cardiac arrest has occurred when there is no effective cardiac output. Before any specific therapy is started effective basic life support must be established as described in chapter 4. Four cardiac arrest rhythms will be discussed in this chapter:

1. Asystole
2. Pulseless electrical activity (including electro mechanical dissociation)
3. Ventricular fibrillation
4 Pulseless ventricular tachycardia

The four are divided into two groups: two that do not require defibrillation (called "non-shockable") and two that do require defibrillation ("shockable"). The cardiac arrest algorithm is shown in Figure 6.1

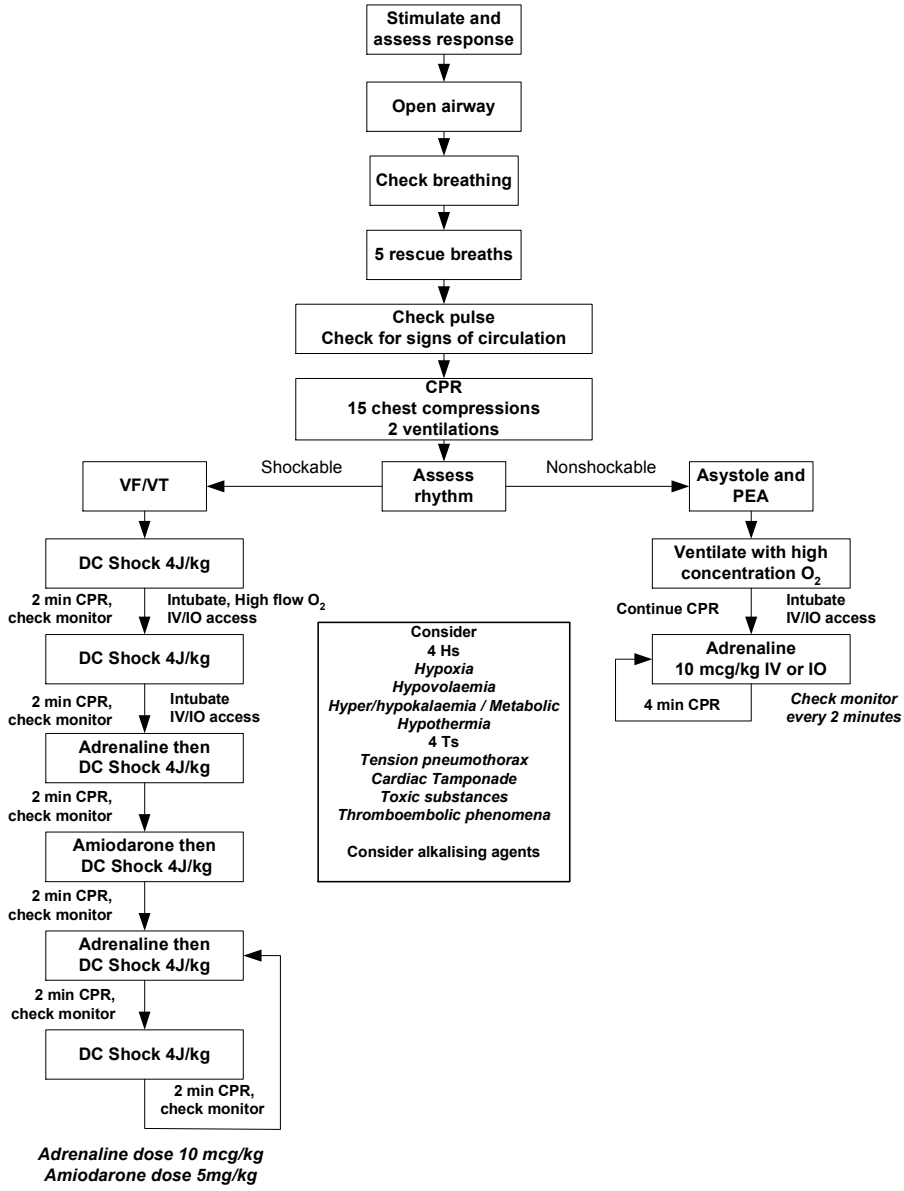

Figure 6.1. Cardiac arrest algorithm

6.2 NON – SHOCKABLE RHYTHMS

This includes asystole and pulseless electrical activity.

Asystole

This is the most common arrest rhythm in children, because the response of the young heart to prolonged severe hypoxia and acidosis is progressive bradycardia leading to asystole.

The ECG will distinguish asystole from ventricular fibrillation, ventricular tachycardia and pulseless electrical activity. The ECG appearance of ventricular asystole is an almost straight line; occasionally P-waves are seen. Check that the appearance is not caused by an artifact, e.g. a loose wire or disconnected electrode. Turn up the gain on the ECG monitor.

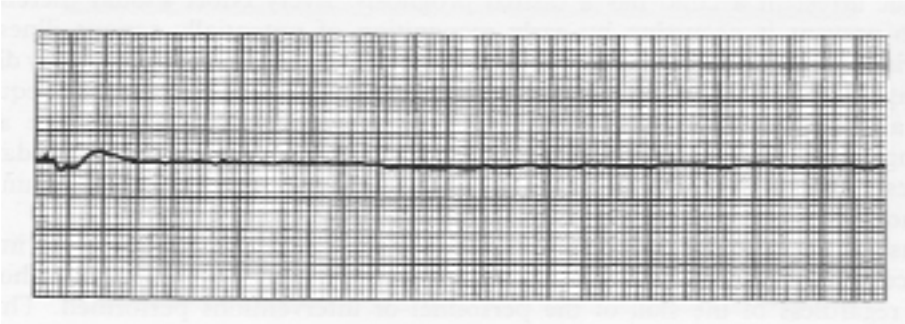

Figure 6.2. Asystole

Pulseless Electrical Activity (PEA)

This is the absence of a palpable pulse or other signs of circulation despite the presence on the ECG monitor of recognisable complexes which normally produce a pulse. PEA is treated in the same way as asystole and is often a pre-asystolic state.

Pulseless electrical activity may be due to an identifiable and reversible cause. In children, trauma is most often associated with a reversible cause of PEA. This may be severe hypovolaemia, tension pneumothorax or pericardial tamponade. PEA is also seen in hypothermic patients and in patients with electrolyte abnormalities, including hypocalcaemia from calcium channel blocker overdose. Rarely in children it may be seen after massive pulmonary thromboembolus.

Figure 6.3. Pulseless Electrical Activity (PEA)

Management of Asystole/PEA

The first essential is to establish ventilations and chest compressions effectively. Ventilations are provided initially by bag and mask with high concentration oxygen. Ensure a patent airway, initially using an airway manoeuvre to open the airway and stabilising it with an airway adjunct.

Provide effective chest compressions at a rate of 100 per minute with a compression/ ventilation ratio of 15: 2. The child should have a cardiac monitor attached and the **heart's rhythm assessed.**

Although the procedures to stabilise the airway and gain circulatory access are now described sequentially, they should be undertaken simultaneously under the direction of a resuscitation team leader.

If asystole or PEA is identified give **adrenaline (epinephrine) 10 micrograms per kilogram intravenously or intraosseously.** Adrenaline (epinephrine) is the first line drug for asystole. Through α-adrenergic–mediated vasoconstriction, its action is to increase aortic diastolic pressure during chest compressions and thus coronary perfusion pressure and the delivery of oxygenated blood to the heart. It also enhances the contractile state of the heart and stimulates spontaneous contractions, The intravenous or interosseous dose is 10 micrograms/kg (0.1 ml of 1:10,000 solution). This is best given through a central line but if one is not in place it may be given through a peripheral line. In the child with no existing intravenous access the intraosseous route is recommended as the route of choice as it is rapid and effective. In each case the adrenaline (epinephrine) is followed by a normal saline flush (2-5 mls).

If circulatory access cannot be gained, the tracheal tube can be used but this is the least satisfactory route and should be avoided if possible as it may cause transient β adrenergic effects, decreasing coronary perfusion. If the route is used, ten times the intravenous dose (100 micrograms/kg) should be given. The drug should be injected quickly down a narrow bore suction catheter beyond the tracheal end of the tube and then flushed in with 1 or 2 mls of normal saline.

As soon as is feasible a skilled and experienced operator should **intubate the child's airway.** This will both control and protect the airway and enable chest compressions to be given continuously, thus improving coronary perfusion. Once the child has been intubated and compressions are uninterrupted, the ventilation rate should be 10 per minute. It is important for the team leader to assess that the ventilations remain adequate when chest compressions are continuous.

The protocol for asystole and PEA is shown in Figure 6.4.

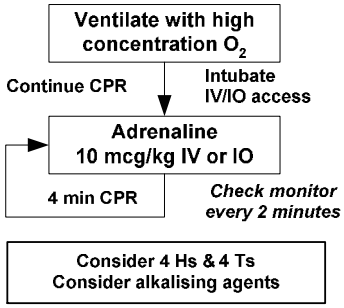

Figure 6.4. Protocol for asystole and PEA

During and following the administration of adrenaline (epinephrine), chest compressions and ventilations should continue. Giving chest compressions is tiring for the operator so the team leader should continuously ensure that the compressions are adequate and change the operator every few minutes.

At intervals of about 2 minutes briefly pause in the delivery of chest compressions to assess the rhythm on the monitor. If asystole remains, continue CPR while again checking the electrode position and contact. If there is an organised rhythm check for a pulse or signs of a circulation. If there is a return of spontaneous circulation (ROSC), continue post-resuscitation care. If there is no pulse and no signs of a circulation continue the protocol. Give adrenaline (epinephrine) about every 4 minutes at a dose of 10 micrograms per kilogram.

Reversible causes

Continually, during CPR consider and correct reversible causes of the cardiac arrest based on the history of the event and any clues that are found during resuscitation.

These factors are remembered as the **4Hs and 4Ts:**

Hypoxia is a prime cause of cardiac arrest in childhood and is key to successful resuscitation
Hypovolaemia may be significant in arrests associated with trauma, anaphylaxis and sepsis and requires infusion of crystalloid (see Chapter 13)
Hyperkalaemia, hypokalaemia, hypocalcaemia and other metabolic abnormalities may be suggested by the patient's underlying condition (e.g. renal failure), tests taken during the resuscitation or clues given in the ECG (see Appendices A and B). Intravenous calcium (0.2 mls/kg of 10% calcium gluconate) is indicated in hyperkalaemia, hypocalcaemia and calcium channel blocker overdose
Hypothermia is associated with drowning incidents and requires particular care: a low reading thermometer must be used to detect it (see Chapter 19)
Tension pneumothorax and cardiac **tamponade** are especially associated with PEA and found in trauma cases (see Chapter 14).
Toxic substances either as a result of accidental or deliberate overdose or from a iatrogenic mistake may require specific antidotes (see Appendix H)
Thromboembolic phenomena are much less common in children than in adults.

Adrenaline (epinephrine) dosage

Adrenaline (epinephrine) has been used for many years although its place has never been subjected to trial against placebo in humans. Its use is supported by animal studies and its known effects in improving relative coronary and cerebral perfusion. There has been a trend to the use of higher doses of adrenaline (epinephrine) in past years but evidence now links high dosage to poorer outcome, especially in asphyxial arrests. High dose (100microgm/kg) adrenaline (epinephrine) should be used therefore only in very specific circumstances e.g. if necessary after cardiac arrest associated with β blocker overdose.

Alkalizing Agents

Children with asystole will be acidotic as cardiac arrest has usually been preceded by respiratory arrest or shock. However, the routine use of alkalizing agents has not been shown to be of benefit. Sodium bicarbonate therapy increases intra-cellular carbon dioxide levels so administration, if used at all should follow assisted ventilation with oxygen, and effective BLS. Once ventilation is ensured and adrenaline (epinephrine) plus chest compressions are provided to maximize circulation, use of sodium bicarbonate may be considered for the patient with prolonged cardiac arrest or cardiac arrest associated with documented severe metabolic acidosis These agents should be administered only in cases where profound acidosis is likely to adversely

affect the action of adrenaline (epinephrine). An alkalizing agent may be considered if spontaneous circulation has not returned after the first or second dose of adrenaline (epinephrine). In addition sodium bicarbonate is recommended in the treatment of patients with hyperkalaemia (Appendix B) and tricyclic antidepressant overdose. (Appendix H).

In the arrested patient arterial pH does not correlate well with tissue pH. Mixed venous or central venous pH should be used to guide any further alkalizing therapy and it should always be remembered that good basic life support is more effective than alkalizing agents at raising myocardial pH.

Bicarbonate is the most common alkalizing agent currently available, the dose being 1 mmol/kg (1 ml/kg of an 8.4% solution). Certain caveats must be kept in mind.

- Bicarbonate must not be given in the same intravenous line as calcium because precipitation will occur.
- Sodium bicarbonate inactivates adrenaline (epinephrine) and dopamine and therefore the line must be flushed with saline if these drugs are subsequently given.
- Bicarbonate may not be given by the intra-tracheal route.

Calcium

In the past, calcium was recommended in the treatment of PEA and asystole, but there is no evidence for its efficacy and there is evidence for harmful effects as calcium is implicated in cytoplasmic calcium accumulation in the final common pathway of cell death. This results from calcium entering cells following ischaemia and during reperfusion of ischaemic organs. Administration of calcium in resuscitation of asystolic patients is not recommended. Calcium is indicated only for treatment of documented hypocalcemia and hyperkalemia, and for the treatment of hypermagnesemia and of calcium channel blocker overdose.

6.3 SHOCKABLE RHYTHMS

Ventricular Fibrillation and Pulseless Ventricular Tachycardia

ECGs showing ventricular fibrillation and ventricular tachycardia are shown in Figures 6.5 and 6.6 respectively.

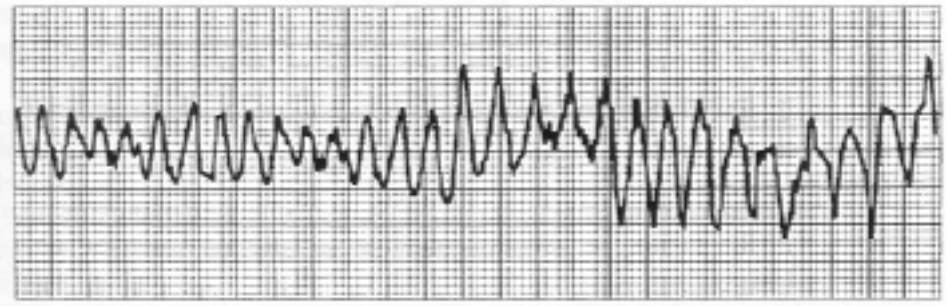

Figure 6.5. Ventricular fibrillation

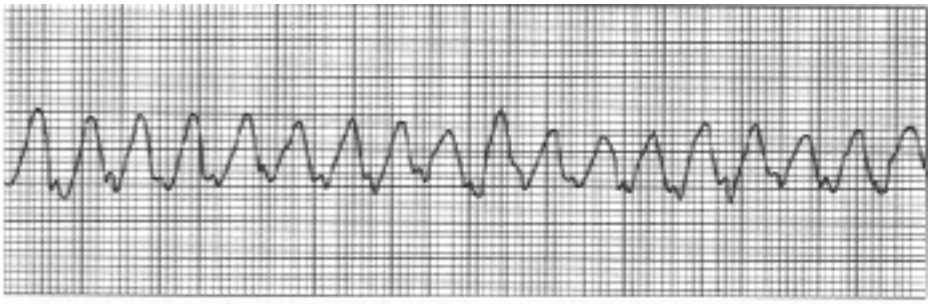

Figure 6.6. Ventricular tachycardia

These arrhythmias are less common in children but either may be expected in sudden collapse, those suffering from hypothermia, poison by tricyclic antidepressants and with cardiac disease. The protocol for ventricular fibrillation and pulseless ventricular tachycardia is the same and is shown in Figure 6.7.

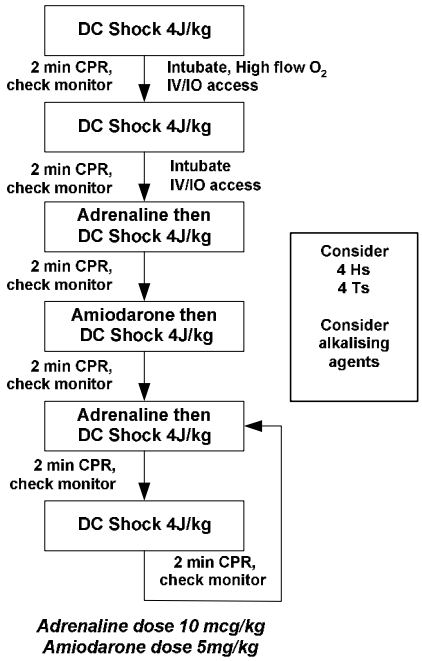

Figure 6.7. Protocol for ventricular fibrillation and ventricular tachycardia

If the patient is being monitored, the rhythm can be identified before significant deterioration. With immediate identification of VF/pulseless VT asynchronous electrical defibrillation of 4 joules per kilogram should be carried out immediately and the protocol continued as below.

In un-monitored children BLS will have been started in response to the collapse and the identification of VF/pulseless VT will occur when the cardiac monitor is put in place.

An **asynchronous shock of 4 joules per kilogram** should be given immediately and **CPR immediately resumed** without reassessing the rhythm or feeling for a pulse. Immediate resumption of CPR is necessary because there is sometimes a pause between successful defibrillation and the appearance of a perfusing rhythm on the monitor. Cessation of chest compressions will reduce the chance of a successful outcome if a further shock is needed. No harm accrues from “unnecessary” compressions.

Paediatric paddles (4.5 cm) should be used for children under 10 kg. One electrode is placed over the apex in the mid axillary line, whilst the other is put immediately below the clavicle just to the right of the sternum. If only adult paddles are available for an infant under 10 kg. one may be placed on the infant's back and one over the left lower part of the chest at the front.

If the shock fails to defibrillate, attention must revert to supporting coronary and cerebral perfusion as in asystole. Although the procedures to stabilise the airway and gain circulatory access are now described sequentially, they should be undertaken simultaneously under the direction of a resuscitation team leader.

The airway should be secured, the patient **ventilated** with high flow oxygen and **effective chest compressions** continued at a rate of 100 per minute and a ratio of 15 compressions to 2 ventilations. As soon as is feasible, a skilled and experienced operator should **intubate the child's airway.** This will both control and protect the airway and enable chest compressions to be given continuously, thus improving coronary perfusion. Once the child has been intubated and compressions are uninterrupted, the ventilation rate should be 10 per minute. It is important for the team leader to assess that the ventilations remain adequate when chest compressions are continuous. **Gain circulatory access.** If a central line is in place this is the most effective route but if one is not in place the drug may be given through a peripheral line. In the child with no existing intravenous access the intraosseous route is recommended as the route of choice as it is rapid and effective.

After 2 minutes, pause chest compressions briefly to check the monitor. If VF/VT is still present, give a **second shock of 4 joules per kilogram** and **immediately resume CPR** without checking the monitor or feeling for a pulse.

Consider and correct reversible causes (4Hs and 4Ts) while continuing CPR for a further 2 minutes. Pause briefly to check the monitor.

If the rhythm is still VF/VT give **adrenaline (epinephrine) 10 micrograms/kg** intravenously or intraosseously followed immediately by a **third shock of 4 joules per kilogram** and **resume CPR immediately**.

After a further 2 minutes of CPR, pause briefly to check the monitor and if the rhythm is still VF/VT give an **intravenous bolus of amiodarone** 5 mg per kilogram and an immediate **fourth shock of 4 joules per kilogram.**

Continue giving shocks every 2 minutes, minimising the pauses in CPR as much as possible. Give adrenaline (epinephrine) before every other shock and continue to seek and treat reversible causes

Note: After each 2 minutes of uninterrupted CPR, pause briefly to assess the rhythm on the monitor

- If still VF/VT, continue with the sequence as above
- If asystole, change to the asystole/PEA sequence
- If organised electrical activity is seen, check for a pulse, if there is ROSC, continue post resuscitation care. If there is no pulse and no signs of a circulation continue the asystole/PEA sequence

A precordial thump may be given in monitored children in whom the onset of the arrhythmia is witnessed and if the defibrillator is not immediately to hand. This is performed by giving a sharp blow to the lower part of the child's sternum from the ulnar side of the clenched fist

Anti-arrhythmic drugs

Amiodarone is the treatment of choice in shock resistant ventricular fibrillation and pulseless ventricular tachycardia. This is based on evidence from adult cardiac arrest and experience with the use of amiodarone in children in the catheterisation laboratory setting. The dose of amiodarone for VF/pulseless VT is 5 mg/kg via rapid i.v. bolus.

There may be circumstances where the routine use of amiodarone should be omitted. This includes VF/pulseless VT caused by an overdose of an arrhythmogenic drug. Expert advice should be obtained from a Poisons Centre. Amiodarone is likely to be unhelpful in the setting of VF caused by hypothermia but may be used, nevertheless.

Lidocaine (lignocaine) is an alternative to amiodarone of the latter is unavailable. The dose is 1 mg/kg IV or IO. It is DC shock that converts the heart back to a perfusing rhythm not the drug. The purpose of the anti-arrhythmic drug is to stabilise the converted rhythm and the purpose of adrenaline (epinephrine) is to improve myocardial oxygenation by increasing coronary perfusion pressure adrenaline (epinephrine) also increases the vigour and intensity of ventricular fibrillation which increases the success of defibrillation.

Magnesium is indicated in children with hypomagnesemia or with polymorphic VT (torsades de pointes) regardless of cause.

Reversible causes

During CPR consider and correct reversible causes of the cardiac arrest based on the history of the event and any clues that are found during resuscitation.

These factors are remembered as the **4Hs and 4Ts**

Hypoxia is a prime cause of cardiac arrest in childhood and is key to successful resuscitation

Hypovolaemia may be significant in arrests associated with trauma, anaphylaxis and sepsis and requires infusion of crystalloid (see Chapter 13)

Hyperkalaemia, hypokalaemia, hypocalcaemia and other metabolic abnormalities may be suggested by the patient's underlying condition (e.g. renal failure), tests taken during the resuscitation or clues given in the ECG (see Appendices A and B). Intravenous calcium chloride is indicated in hyperkalaemia, hypocalcaemia and calcium channel blocker overdose. Hyperkalaemia is treated then with bicarbonate, insulin and glucose (see Appendix A and B).

Hypothermia is associated with drowning incidents and requires particular care: a low reading thermometer must be used to detect it and defibrillation may be resistant until core temperature is increased. Active rewarming should be commenced (see Chapter 19)

Tension pneumothorax and cardiac **tamponade** are especially associated with PEA and found in trauma cases (see Chapter 14).

Toxic substances either as a result of accidental or deliberate overdose or from a iatrogenic mistake may require specific antidotes. If the VF/VT has been caused by an overdose of tricyclic antidepressants then the patient should be alkalised and anti-arrhythmic drugs avoided except under expert guidance (see Appendix H).

Thromboembolic phenomena are much less common in children than in adults.

If there is still resistance to defibrillation, different paddle positions or another defibrillator may be tried. In the infant in whom paediatric paddles have been used, larger paddles applied to the front and back of the chest may be an alternative.

If the rhythm initially converts and then deteriorates back to ventricular fibrillation or pulseless ventricular tachycardia then the sequence should continue to cycle with the exception of a further dose of amiodarone. If further amiodarone is thought necessary an infusion should be given of 600 microgms/kg/hr to a maximum of 1.2 G in 24 hours.

Automatic External Defibrillators (AEDs)

The introduction of automatic external defibrillators in the pre-hospital setting and especially for public access is likely to significantly improve the outcome for VF/VT cardiac arrest.

In the prehospital setting, automatic external defibrillators (AEDs) are commonly used in adults to assess cardiac rhythm and to deliver defibrillation. In children AEDs can accurately detect ventricular fibrillation at all ages, but there is concern over their ability to identify correctly tachycardic rhythms in infants. At present, therefore, AEDs can be used to identify rhythms in children but not in infants.

Many devices now have paediatric attenuation pads which decrease the energy to a level more appropriate for the child (1-8 years) or leads reducing the total energy to 50-80 joules. This means that AEDs can be used for all ages of children except the under ones. Institutions treating infants who might need defibrillation must provide manual defibrillators.

- with a manual defibrillator use 4 joules/kg to defibrillate at all ages
- with an unattenuated AED (see above) the child of over 8 years can be defibrillated
- with an AED with paediatric pads or paddles, the child of 1-8 years can be defibrillated

Modern defibrillators now use biphasic wave forms. Defibrillation appears to be as effective at lower energy doses than conventional wave forms in adults and the energy appears to cause less myocardial damage than monophasic shocks Both monophasic and biphasic wave form defibrillators are acceptable for use in childhood.

Therapeutic hypothermia

Recent data suggest that there is some evidence that post-arrest hypothermia (core temperatures of 33 to 36°C) may have beneficial effects on neurological recovery but there is insufficient evidence or experience to recommend the routine use of hypothermia. Current recommendations are that post arrest patients with core temperatures less than 37.5^{o}C should not be actively rewarmed, unless the core temperature is < 33°C when they should be rewarmed to 34°C. Conversely, increased core temperature increases metabolic demand by 10-13% for each degree Centigrade increase in temperature above normal. Therefore in the post arrest patient hyperthermia should be treated with active cooling to achieve a normal core temperature. Shivering should be prevented, since it will increase metabolic demand. Sedation may be adequate to control shivering, but neuromuscular blockade may be needed.

Hypoglycaemia

All children, especially infants can become hypoglycaemic when seriously ill. Blood glucose should be checked frequently and hypoglycaemia corrected carefully. It is important not to cause

hyperglycaemia as this will promote an osmotic diuresis and also hyperglycaemia is associated with worse neurological outcome in animal models of cardiac arrest.

6.4 WHEN TO STOP RESUSCITATION

Resuscitation efforts are unlikely to be successful and can be discontinued if there is no return of spontaneous circulation at any time with up to 30 mins of cumulative life support and in the absence of recurring or refractory VF/VT. Exceptions are patients with a history of poisoning or a primary hypothermic insult in whom prolonged attempts may occasionally be successful. Seek expert help from a toxicologist or paediatric intensivist.

6.5 PARENTAL PRESENCE

Evidence suggests that presence at the child's side during resuscitation enables parents gain a realistic understanding of the efforts made to save their child and they subsequently may show less anxiety and depression.

Important points:

- A staff member must be designated to be the parents support and interpreter of events at all times
- The team leader, not the parents, decides when it is appropriate to stop the resuscitation
- The team needs a debriefing session to support staff and reflect on practice

6.6 SUMMARY

The teaching in this chapter is consistent with the ILCOR guidelines, Resuscitation 2005, and there are an enormous number of references which have informed this process. These are available on the ALSG Web site. See details on the "Contact Details and Further Information" page.