



Paediatric Advanced Life Support

Introduction

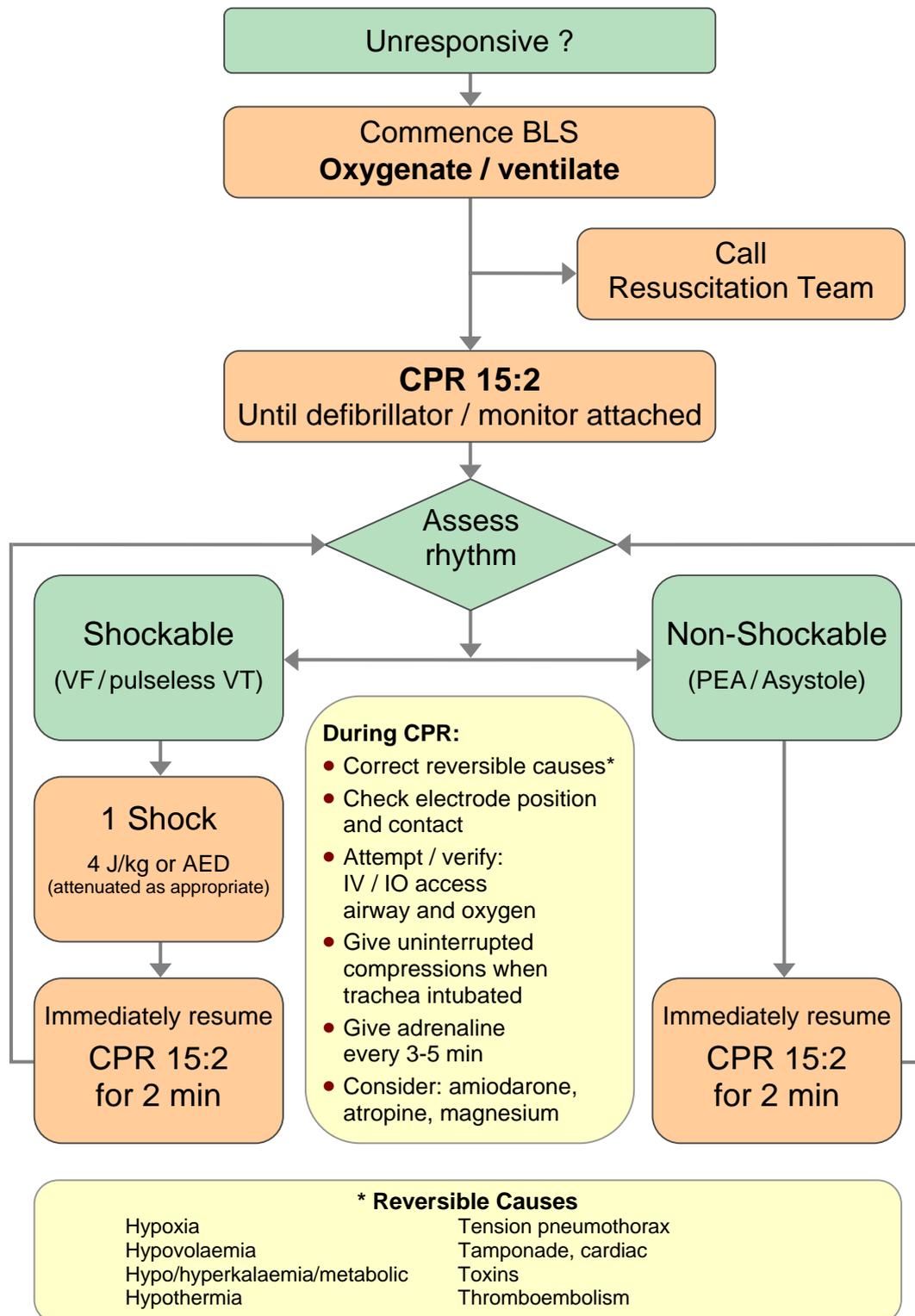
There is concern that resuscitation from cardiac arrest is not performed as well as it might because the variations in guidelines for different age groups cause confusion to providers, and therefore poor performance. Most of the changes in paediatric guidelines for 2005 have been made for simplification and to minimise differences between adult and paediatric protocols. It is hoped that this will assist teaching and retention. The guidelines have not, however, been simplified in the face of contradictory evidence or against an understanding of pathophysiology.

There remains a paucity of good quality evidence on which to base the resuscitation of infants and children. Most conclusions have had to be drawn from extrapolated adult studies and from experimental work.

Guideline Changes

- Where possible, give drugs intravascularly (intravenous or intraosseous), rather than by the tracheal route.
- Either uncuffed or cuffed tracheal tubes may be used in infants and children in the hospital setting.
- One defibrillating shock, rather than three 'stacked' shocks, is recommended for ventricular fibrillation/pulseless ventricular tachycardia (VF/VT).
- When using a manual defibrillator, the shock energy for children is 4 J kg^{-1} for all shocks.
- A standard AED can be used in children over 8 years.
- Purpose-made paediatric pads, or programs which attenuate the energy output of an AED, are recommended for children between 1 and 8 years.
- If no such system or manually adjustable machine is available, an unmodified adult AED may be used for children older than 1 year.
- There is insufficient evidence to support a recommendation for or against the use of AEDs in children less than 1 year.
- The dose of adrenaline (epinephrine) during cardiac arrest is $10 \text{ microgram kg}^{-1}$ on each occasion.

Paediatric Advanced Life Support



Sequence of Actions

1 Establish basic life support.

2 Oxygenate, ventilate, and start chest compression:

- Provide positive-pressure ventilation with high-concentration inspired oxygen.
- Provide ventilation initially by bag and mask. Ensure a patent airway by using an airway manoeuvre as described in the paediatric basic life support section.
- As soon as is feasible, an experienced operator should intubate the child. This will both control the airway and enable chest compression to be given continuously, thus improving coronary perfusion pressure.
- Take care to ensure that ventilation remains effective when continuous chest compressions are started.
- Use a compression rate of 100 min⁻¹.
- Once the child has been intubated and compressions are uninterrupted, use a ventilation rate of approximately 10 min⁻¹.

3 Attach a defibrillator or monitor:

- Assess and monitor the cardiac rhythm.
- If using a defibrillator, place one defibrillator pad or paddle on the chest wall just below the right clavicle, and one in the left anterior axillary line.
- Pads or paddles for children should be 8 - 12 cm in size, and 4.5 cm for infants. In infants and small children it may be best to apply the pads or paddles to the front and back of the chest.
- Place monitoring electrodes in the conventional chest positions.

4 Assess rhythm and check for signs of a circulation (signs of life):

- Look for signs of a circulation, which include responsiveness, coughing, and normal breathing.
- Check the pulse if trained to do so:
 - **Child** – feel for the carotid pulse in the neck.
 - **Infant** – feel for the brachial pulse on the inner aspect of the upper arm.
- Take no more than 10 sec for the pulse check.
- Assess the rhythm on the monitor:
 - Non-shockable (asystole or pulseless electrical activity) **OR**
 - Shockable (VF/VT).

5 A Non-shockable (asystole or pulseless electrical activity):

This is the more common finding in children.

- **Perform continuous CPR:**
 - Continue to ventilate with high-concentration oxygen.
 - If ventilating with bag-mask give 15 chest compressions to 2 ventilations for all ages.
 - If the patient is intubated, chest compressions can be continuous as long as this does not interfere with satisfactory ventilation.
 - Use a compression rate of 100 min^{-1} .
 - Once the child has been intubated and compressions are uninterrupted use a ventilation rate of approximately 10 min^{-1} .

Note: Once there is return of spontaneous circulation (ROSC) the ventilation rate should be $12 - 20 \text{ min}^{-1}$. Measure exhaled CO_2 to ensure correct tracheal tube placement.

- **Give adrenaline:**
 - If venous or intraosseous (IO) access has been established, give adrenaline $10 \text{ microgram kg}^{-1}$ (0.1 ml kg^{-1} of 1 in 10,000 solution).
 - If there is no circulatory access, attempt to obtain IO access.
 - If circulatory access is not present, and cannot be quickly obtained, but the patient has a tracheal tube in place, consider giving adrenaline $100 \text{ microgram kg}^{-1}$ via the tracheal tube (1 ml kg^{-1} of 1 in 10,000 or 0.1 ml kg^{-1} of 1 in 1,000 solution). This is the least satisfactory route (see routes of drug administration).
- **Continue CPR.**
- **Repeat the cycle:**
 - Give adrenaline $10 \text{ microgram kg}^{-1}$ every 3 to 5 min, (i.e. every other loop), while continuing to maintain effective chest compression and ventilation without interruption. Unless there are exceptional circumstances, the dose should be $10 \text{ microgram kg}^{-1}$ again for this and subsequent doses.
 - Once the airway is protected by tracheal intubation, continue chest compression without pausing for ventilation. Provide ventilation at a rate of 10 min^{-1} and compression at 100 min^{-1} .
 - When circulation is restored, ventilate the child at a rate of $12 - 20 \text{ breaths min}^{-1}$ to achieve a normal pCO_2 , and monitor exhaled CO_2 .

- **Consider and correct reversible causes:**
 - Hypoxia
 - Hypovolaemia
 - Hyper/hypokalaemia (electrolyte disturbances)
 - Hypothermia
 - Tension pneumothorax
 - Tamponade
 - Toxic/therapeutic disturbance
 - Thromboembolism
- **Consider the use of other medications such as alkalisating agents.**

5 B Shockable (VF/VT)

This is less common in paediatric practice but likely when there has been a witnessed and sudden collapse. It is commoner in the intensive care unit and cardiac ward.

- **Defibrillate the heart:**
 - Give 1 shock of 4 J kg^{-1} if using a manual defibrillator.
 - If using an AED for a child of 1-8 years, deliver a paediatric-attenuated adult shock energy.
 - If using an AED for a child over 8 years, use the adult shock energy.
- **Resume CPR:**
 - Without reassessing the rhythm or feeling for a pulse, resume CPR **immediately**, starting with chest compression.
- **Continue CPR for 2 min.**
- **Pause briefly to check the monitor:**
 - If still VF/VT, give a second shock at 4 J kg^{-1} if using a manual defibrillator, **OR** the adult shock energy for a child over 8 years using an AED, **OR** a paediatric-attenuated adult shock energy for a child between 1 year and 8 years.
- **Resume CPR immediately after the second shock.**
- **Consider and correct reversible causes (see above: 4Hs and 4Ts).**
- **Continue CPR for 2 min.**

- **Pause briefly to check the monitor:**
 - **If still VF/VT:**
 - Give adrenaline 10 microgram kg^{-1} followed immediately by a (3rd) shock.
 - Resume CPR immediately and continue for 2 min.

- **Pause briefly to check the monitor.**
 - **If still VF/VT:**
 - Give an intravenous bolus of amiodarone 5 mg kg^{-1} and an immediate further (4th) shock.
 - Continue giving shocks every 2 min, minimising the breaks in chest compression as much as possible.
 - Give adrenaline immediately before every other shock (i.e. every 3-5 min) until return of spontaneous circulation (ROSC).

Note: After each 2 min of uninterrupted CPR, pause briefly to assess the rhythm.

- **If still VF/VT:**
 - Continue CPR with the shockable (VF/VT) sequence.

- **If asystole:**
 - Continue CPR and switch to the non-shockable (asystole or pulseless electrical activity) sequence as above.

- **If organised electrical activity is seen,** check for a pulse:
 - If there is ROSC, continue post-resuscitation care.
 - If there is **no** pulse, and there are no other signs of a circulation, give adrenaline 10 microgram kg^{-1} and continue CPR as for the non-shockable sequence as above.

Important note

Uninterrupted, good-quality CPR is vital. Chest compression and ventilation should be interrupted only for defibrillation. Chest compression is tiring for providers. The team leader should continuously assess and feed back on the quality of the compressions, and change the providers every 2 min.

Explanatory notes

Routes of drug administration

Studies in children and adults have shown that atropine, adrenaline, naloxone, lidocaine, and vasopressin are absorbed via the trachea, albeit resulting in lower blood concentrations than the same dose given intravascularly. However,

experimental studies suggest that the lower adrenaline concentrations achieved in this way may produce transient beta-adrenergic effects. These effects can be detrimental, causing hypotension and lower coronary artery perfusion pressure, thereby reducing the likelihood of return of spontaneous circulation.¹ On the other hand, prospective, randomised trials in adults and children show that intraosseous access is safe and effective; practice indicates that this route is increasingly being used successfully.

Tracheal tubes

Several studies^{2, 3} have shown no greater risk of complications for children less than 8 years when cuffed tracheal tubes rather than uncuffed tubes are used in the operating room and intensive care unit. Cuffed tracheal tubes are as safe as uncuffed tubes for infants (except newborns) and children if rescuers use the correct tube size and cuff inflation pressure, and verify tube position. Under certain circumstances (e.g. poor lung compliance, high airway resistance, and large glottic air leak) cuffed tracheal tubes may be preferable. Therefore, either uncuffed or cuffed tracheal tubes may be used in infants and children, but only in the hospital setting.

Shock sequence

For VF/VT, one defibrillating shock rather than three 'stacked' shocks is now recommended. This new recommendation for the sequence of defibrillation in children is based on extrapolated data from adult and experimental studies with biphasic devices. Evidence shows a high rate of success for first-shock conversion of ventricular fibrillation (VF).⁴ Furthermore, interruption of chest compression reduces coronary perfusion pressure, myocardial viability, and the chance of successful defibrillation.

Shock energy level

The ideal energy level for safe and effective defibrillation in children is unknown. The recommendation of 2 - 4 J kg⁻¹ in Guidelines 2000 was based on a single historical study of effective outcomes.

Extrapolation from adult data and experimental studies shows that biphasic shocks are at least as effective as monophasic shocks and produce less post-shock myocardial dysfunction. A few studies have shown that an initial monophasic or biphasic shock level of 2 J kg⁻¹ generally terminates paediatric VF. Paediatric case series have reported that shock levels of more than 4 J kg⁻¹ (up to 9 J kg⁻¹) have effectively defibrillated children less than 12 years with negligible adverse effects. In experimental studies, high energy levels cause less myocardial damage in young hearts than in adult hearts.⁵⁻⁸

A variable-dose manual defibrillator, or an AED able to recognise paediatric shockable rhythms and equipped with dose attenuation, is preferred in paediatric practice.

- Standard AEDs may be used in children over 8 years.

- Purpose-made paediatric pads, or devices/programs which attenuate the energy output of an AED, are recommended for children between 1 and 8 years.
- If no such system or manually adjustable machine is available, however, an unmodified adult AED may be used for children older than 1 year.

There is insufficient information to recommend for or against the use of an AED in infants less than one year. Manual defibrillation must be available for defibrillating infants.

Dose of adrenaline

In paediatric studies^{9,10}, no improvement in survival rates, and a trend towards worse neurological outcomes, have been shown after the administration of high-dose adrenaline during cardiac arrest. Children in cardiac arrest should, therefore, be given adrenaline 10 microgram kg⁻¹ for the first and subsequent IV doses. Routine use of high-dose IV adrenaline (100 microgram kg⁻¹) is not recommended and may be harmful, particularly in asphyxial arrests. High-dose adrenaline should be considered only in exceptional circumstances, for example after beta-blocker overdose.

Drugs used in CPR

Adrenaline

This is an endogenous catecholamine with potent alpha, beta 1, and beta 2 adrenergic actions. Its use has never been subjected to trial in humans, but the drug still plays a major role in the treatment algorithms both for non-shockable and shockable cardiac arrest rhythms. This is supported by experimental studies and its known effect of improving relative coronary and cerebral perfusion.

Adrenaline induces vasoconstriction, increases coronary perfusion pressure, enhances the contractile state of the heart, stimulates spontaneous contractions, and increases the intensity of VF so increasing the likelihood of successful defibrillation.

The recommended IV/IO dose of adrenaline in children is 10 microgram kg⁻¹. The dose of adrenaline via the tracheal tube route is ten times the IV dose (100 microgram kg⁻¹). This route should be avoided if at all possible as evidence shows that there may be a paradoxical effect. Subsequent doses of adrenaline, if needed, should be given every 3-5 min. The use of a higher dose of adrenaline via the IV or IO route is not recommended routinely in children. High-dose adrenaline has not been shown to improve survival or neurological outcome after cardiopulmonary arrest.

Amiodarone

Amiodarone is a membrane-stabilising anti-arrhythmic drug that increases the duration of the action potential and refractory period in atrial and ventricular myocardium. Atrioventricular conduction is slowed, and a similar effect is seen with accessory pathways. Amiodarone has a mild negative inotropic action and causes peripheral vasodilation through non-competitive alpha-blocking effects. The hypotension that occurs with IV amiodarone is related to the rate of delivery and is due more to the solvent (Polysorbate 80), which causes histamine release, than the drug itself.

An initial IV dose of amiodarone 5 mg kg^{-1} , diluted in 5% dextrose, should be considered if VF or pulseless VT persists after a third shock. Amiodarone can cause thrombophlebitis when injected into a peripheral vein: use a central vein if a central venous catheter is in situ; if not, use a large peripheral vein and a generous flush of dextrose or saline.

Lidocaine

Until the publication of Guidelines 2000, lidocaine was the anti-arrhythmic drug of choice. Comparative studies with amiodarone have displaced it from this position and lidocaine is now recommended only for use when amiodarone is unavailable.

Atropine

When bradycardia is unresponsive to improved ventilation and circulatory support, atropine may be used. The dose of atropine is $20 \text{ microgram kg}^{-1}$, with a maximum dose of 600 microgram, and a minimum dose of 100 microgram to avoid a paradoxical effect at low doses.

Magnesium

This is a major intracellular cation and serves as a cofactor in a number of enzymatic reactions. Magnesium treatment is indicated in children with documented hypomagnesemia or with polymorphic VT ('torsade de pointes'), regardless of cause.

Give magnesium sulphate by intravascular infusion over several minutes, at a dose of $25 - 50 \text{ mg kg}^{-1}$ (to a maximum of 2 g).

Calcium

Calcium plays a vital role in the cellular mechanisms underlying myocardial contraction, but there are very few data supporting any beneficial action of therapeutic calcium following most cases of cardiac arrest. High plasma concentrations achieved after injection may have detrimental effects on the ischaemic myocardium and may impair cerebral recovery. Thus, calcium is given during resuscitation only when specifically indicated, for example in

hyperkalaemia, hypocalcaemia, and clinically severe overdose of calcium-channel-blocking drugs.

The dose of calcium chloride is 0.2 ml kg^{-1} of the 10% solution. Calcium can slow the heart rate and precipitate arrhythmias. In cardiac arrest, calcium may be given by rapid intravenous injection. In the presence of a spontaneous circulation it should be given slowly. Calcium solutions and sodium bicarbonate should not be administered simultaneously by the same route.

Sodium bicarbonate

Cardiac arrest results in combined respiratory and metabolic acidosis, caused by cessation of pulmonary gas exchange, and the development of anaerobic cellular metabolism respectively. The best treatment for acidaemia in cardiac arrest is a combination of chest compression and ventilation. Furthermore, giving bicarbonate causes generation of carbon dioxide which diffuses rapidly into the cells. This has the following effects:

- It exacerbates intracellular acidosis.
- It produces a negative inotropic effect on ischaemic myocardium.
- It presents a large, osmotically active, sodium load to an already compromised circulation and brain.
- It produces a shift to the left in the oxygen dissociation curve further inhibiting release of oxygen to the tissues.

The routine use of sodium bicarbonate in cardiac arrest is not recommended. It may be considered in prolonged arrest, and it has a specific role in hyperkalaemia and the arrhythmias associated with tricyclic antidepressant overdose. The dose is $1\text{-}2 \text{ ml kg}^{-1}$ of the 8.4% solution given by the IV or IO routes.

Hypovolaemia

Hypovolaemia is a potentially reversible cause of cardiac arrest. If hypovolaemia is suspected, infuse intravenous or intraosseous fluids rapidly. In the initial stages of resuscitation there are no clear advantages in using colloid. Use isotonic saline solutions. Avoid dextrose-based solutions – these will be redistributed rapidly away from the intravascular space and will cause hyponatraemia and hyperglycaemia, which may worsen neurological outcome after cardiac arrest.

Therapeutic hypothermia

Mild hypothermia is thought to suppress many of the chemical reactions associated with reperfusion injury. Two randomised clinical trials showed improved outcome in adults remaining comatose after initial resuscitation from

out-of-hospital VF cardiac arrest. There are insufficient data for a firm recommendation for children.

Current guidance is that post-arrest infants and children with core temperatures less than 37.5°C should not be actively rewarmed, unless the core temperature is less than 33°C when they should be rewarmed to 34°C.

Hyperthermia has been shown to be correlated with a poorer outcome, so infants and children with core temperatures over 37.5°C should be actively cooled to a normal level. Shivering should be prevented by ensuring adequate sedation and giving neuromuscular blocking drugs.

Complications of mild therapeutic hypothermia include increased risk of infection, cardiovascular instability, coagulopathy, hyperglycaemia, and electrolyte abnormalities such as hypophosphataemia and hypomagnesaemia.

Parental Presence

Many parents would like to be present during a resuscitation attempt; they can see that everything possible is being done for their child. Reports show that being at the side of the child is comforting to the parents or carers, and helps them to gain a realistic view of attempted resuscitation and death. Families who have been present in the resuscitation room show less anxiety and depression several months after the death.

A dedicated staff member should be present with the parents at all times to explain the process in an empathetic and sympathetic manner. He or she can also ensure that the parents do not interfere with the resuscitation process or distract the medical and nursing staff. If the presence of the parents is impeding the progress of the resuscitation, they should be gently asked to leave. When appropriate, physical contact with the child should be allowed.

The team leader of the resuscitation, not the parents, will decide when to stop the resuscitation effort; this should be expressed with sensitivity and understanding. After the event, debriefing of the team should be conducted, to express any concerns and to allow the team to reflect on their clinical practice in a supportive environment.

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