



Guideline 11.10 Resuscitation in special circumstances

Summary

Most cardiac arrests are associated with coronary heart disease, but about 30% of out-of-hospital events are believed to be of a non-cardiac cause. These conditions account for a large proportion of cardiac arrests in young patients with no co-existing disease. Early recognition and effective treatment of these conditions may prevent cardiac arrest or increase the chance of a successful outcome. Survival in all these conditions still relies on using the ABCDE (Airway, Breathing, Circulation, Disability, Exposure) approach.

Resuscitation needs to be modified in specific circumstances. Early recognition of signs and symptoms and effective treatment will often prevent cardiac arrest. These conditions account for a large proportion of cardiac arrests in younger patients with no co-existing disease. It is essential to seek appropriate expert help early for most of these conditions, as they will require specialist interventions.

Survival in all these conditions still relies on using the ABCDE (Airway Breathing Circulation Disability Exposure) approach to help prevent cardiac arrest. If cardiac arrest does occur, high quality CPR with minimal interruption and treatment of reversible causes are still the most important interventions.

[These topics are covered in more detail in the Advanced Life Support Course manual.](#)

Anaphylaxis

Anaphylaxis is a severe, life-threatening, generalised or systemic hypersensitivity reaction. This is characterised by rapidly developing life-threatening airway, and/or breathing and/or circulation problems usually associated with skin and mucosal changes. Airway obstruction may occur rapidly in severe anaphylaxis, particularly in patients with angioedema.

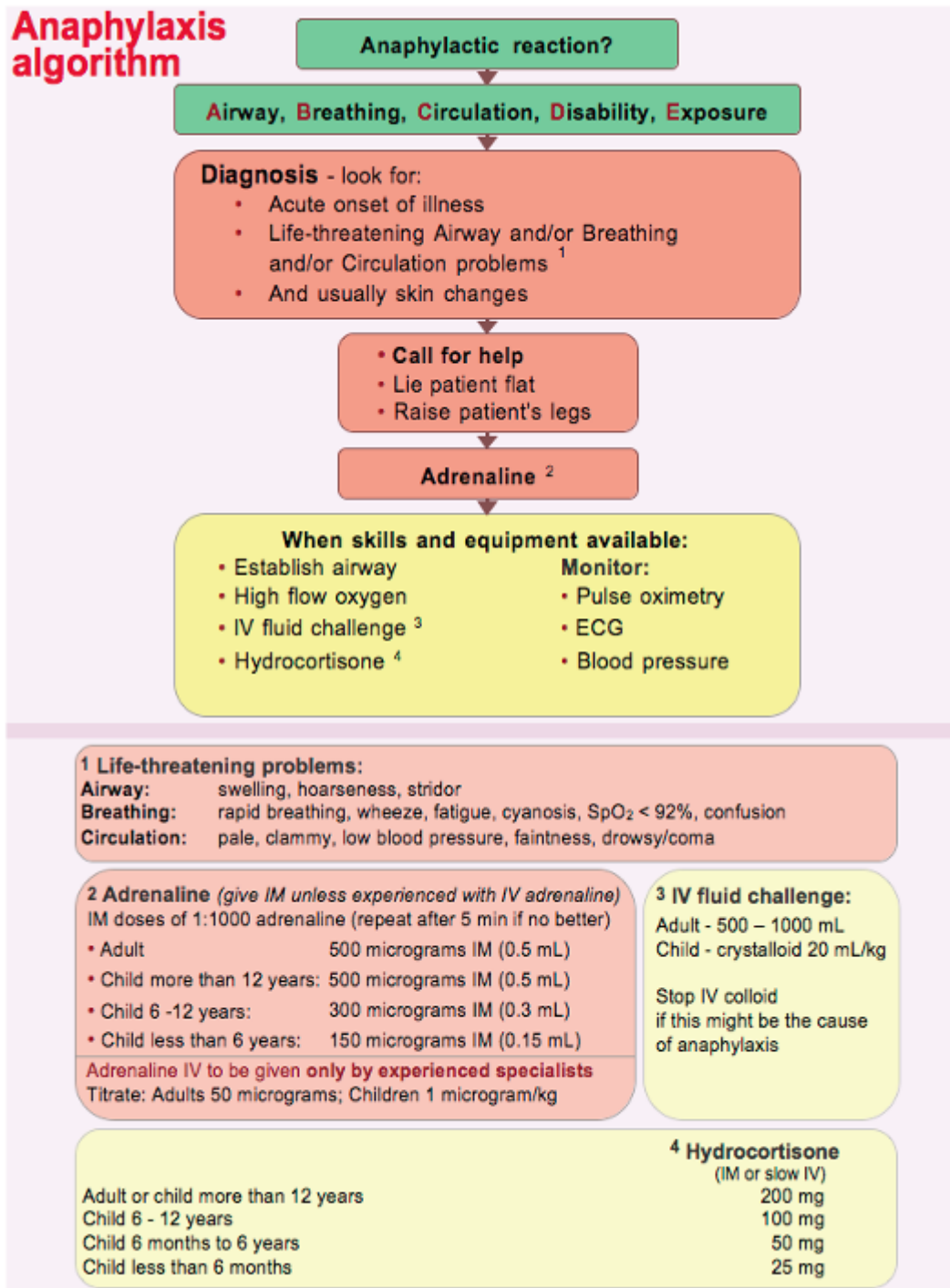
Recommendations

If cardiorespiratory arrest occurs follow the standard ALS protocol as well as large volumes of intravenous fluids. Prolonged resuscitation may be necessary. [Class A, Expert Consensus

Opinion]

See Figure 11.10.1: Peri-arrest management of Anaphylaxis algorithm. For more details see Cardiac Arrest in Special Circumstances chapter in ARC ALS text.

Figure 11.10.1: Peri-arrest management of Anaphylaxis algorithm



Asthma

Cardiac arrest in the asthmatic is often a terminal event after a period of hypoxaemia; occasionally, it may be sudden.

Cardiac arrest in asthmatics has been linked to:

- severe bronchospasm and mucous plugging leading to asphyxia;
- cardiac arrhythmias due to hypoxia, stimulant drugs (e.g. β -adrenergic agonists, aminophylline) or electrolyte abnormalities;
- dynamic hyperinflation, i.e. auto-positive end-expiratory pressure (auto-PEEP), can occur in mechanically ventilated asthmatics. Auto-PEEP is caused by air trapping and 'breath stacking' (air entering the lungs and being unable to escape). Gradual build-up of pressure occurs and reduces venous return and blood pressure;
- tension pneumothorax (often bilateral).

There are no randomised controlled trials that specifically evaluate or compare adjuvant treatment with standard treatment for cardiac arrest in asthmatics. Most of the literature comprises case reports and case series.

Evidence from three non-cardiac arrest case series involving 35 patients suggests that asthmatic patients are at risk for gas trapping during cardiac arrest, especially if their lungs are ventilated with high tidal volumes and/or rapid rates.

One volunteer adult study demonstrated that increasing PEEP caused increased transthoracic impedance.

Seven case series involving 37 patients suggested increased ease of ventilation and return of spontaneous circulation with lateral chest compressions at the base of the ribs. In a single case report, lateral chest compressions were associated with cardiac arrest and poor cardiac output. Three single case reports (two intra-operative and one emergency department) involving cardiac arrest caused by asthma, suggested improvement in ease of ventilation and return of spontaneous circulation with thoracotomy and manual lung compression. [Deakin 2010]

Recommendations

If cardiac arrest occurs in the setting of asthma, follow standard resuscitation guidelines [Deakin 2010]

Ventilation may be difficult, so early endotracheal intubation should be considered. If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest wall and/or a period of apnoea (eg. disconnection of tracheal tube after 2 minutes of CPR) may relieve gas-trapping. Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation. [Class A, LOE IV/Expert Consensus Opinion]

Consideration should also be given to the possible coexistence of anaphylaxis. [Class A, Expert Consensus Opinion]

Avalanches

Avalanches occur in areas that are difficult to access by rescuers in a timely manner, and burials frequently involve multiple victims. The decision to initiate full resuscitative measures should be determined by the number of victims and the resources available, and should be informed by the likelihood of survival.

Avalanche victims are not likely to survive when they are:

- buried > 35 minutes and in cardiac arrest with an obstructed airway on extrication
- buried initially and in cardiac arrest with an obstructed airway on extrication, and an initial core temperature of <32 degrees
- buried initially and in cardiac arrest on extrication with an initial serum potassium of >12 mmol [Deakin 2010][Boyd 2010]

Recommendations

Full resuscitative measures, including extracorporeal rewarming, when available, are indicated for all avalanche victims who do not show evidence of an unsurvivable injury. [Class A, LOE IV/Expert Consensus Opinion]

Cardiac Surgery

After major cardiac surgery, cardiac arrest is relatively common in the immediate post-operative phase, with a reported incidence of 0.7% - 2.9%. Cardiac arrest is usually preceded by physiological deterioration, although it may occur suddenly in stable patients. Continuous monitoring on the intensive care unit (ICU) enables immediate intervention at the time of arrest. Survival to hospital discharge of patients having a cardiac arrest during the first 24 h after cardiac surgery is reported as 54 - 79% in adults and 41% in children.

There are usually specific causes of cardiac arrest that are all potentially reversible. The main causes of cardiac arrest in the initial post-operative period include:

- cardiac tamponade;
- myocardial ischaemia;
- haemorrhage causing hypovolaemic shock;
- disconnection of the pacing system in a pacing dependent patient;
- tension pneumothorax;
- electrolyte disturbances (particularly hypo/hyperkalaemia). [Deakin 2010]

The adverse effects of external cardiac compressions may be more significant after cardiac surgery (eg. disruption of grafts, valves etc). In this setting, early defibrillation of a shockable rhythm is even more important. [Dunning 2009]

Recommendations

The use of 3 stacked shocks may be considered at any time in the immediate post-operative period for cardiac arrests due to shockable rhythms after cardiac surgery to minimise the potential harm of chest compressions, but only if the defibrillator is immediately available (eg. first shock able to be delivered within 20 seconds) (see “Immediate defibrillation” below). [Class A, Expert Consensus Opinion]

In the event of a brady-asystolic cardiac arrest after cardiac surgery where epicardial wires have previously been attached, these wires should be used to attempt to pace the heart. Emergency asynchronous pacing (such as VOO or DOO) should be used. Atrial pacing modes are unlikely to be successful. [Class A, Expert Consensus Opinion]

Opening the chest after recent cardiac surgery (re-sternotomy) allows visualisation of, or treatment of cardiac tamponade, visualisation of problems, direct cardiac compressions, and facilitates institution of cardiopulmonary bypass if required. Re-sternotomy for patients with cardiac arrest following cardiac surgery should be considered in an appropriately staffed and equipped intensive care unit.

Re-sternotomy performed outside these specialised environments has poor results. [Deakin 2010] [Class A, LOE III-2/Expert Consensus Opinion]

Chest compressions should not be withheld while preparing for emergency re-sternotomy. Transthoracic or trans-oesophageal echocardiography are very useful when they are readily available to help elucidate the cause of the cardiac arrest (including haemoperitoneum, haemothorax, tension pneumothorax and cardiac tamponade). [Class A, LOE IV/Expert Consensus Opinion]

Mechanical circulatory support may be considered in the setting of cardiac arrest following cardiac surgery. [Class B, LOE III-3] There is insufficient evidence to make any recommendations about epinephrine dose, anti-arrhythmic use, or any other intervention separate from those recommended in standard protocols. [Deakin 2010]

Electrolyte disorders

[See ANZCOR Guideline 11.5 Medications in Adult Cardiac Arrest.](#)

Percutaneous Coronary Interventions

There is evidence of underlying ischemic heart disease in the majority of patients who have cardiac arrests. Recommendations for the use of angiography and percutaneous coronary intervention (PCI) in the setting of cardiac arrest are included in [Guideline 11.7](#) Post-Resuscitation Therapy in Adult Advanced Life Support. See also [Guideline 14.3](#) Acute Coronary Syndromes: Reperfusion Strategy.

Mechanical CPR during PCI

Three adult human case reports, three adult human case series, and one animal study reported that the use of a mechanical chest-compression device in cardiac arrest during percutaneous coronary intervention (PCI) maintained circulation and enabled the procedure to be completed. A small number of patients in the case series survived. [Wagner 2010] [Deakin 2010]

Cough CPR

A few case reports documented limited benefit of cough CPR during the initial seconds to minutes of cardiac arrest in patients who remained conscious in a controlled, monitored setting of electrophysiology testing with patient instruction prior to the onset of anticipated cardiac arrest. [Deakin 2010]

Recommendations

There are insufficient data to support or refute the use of mechanical chest compression, cough CPR or emergency cardiopulmonary bypass to improve outcome of cardiac arrest during percutaneous coronary intervention.

Use of cough CPR may be considered only for patients maintaining consciousness during the initial seconds to minutes of VF or pulseless VT cardiac arrest in a witnessed, monitored, hospital setting (such as a cardiac catheterization laboratory). [Class B, Expert Consensus Opinion]

Pericardial tamponade

Five case series indicate that echocardiographically guided pericardiocentesis is a safe and effective method of relieving tamponade, especially when used in conjunction with a pericardial drain, and it may obviate the need for subsequent treatment in the operating room. [Deakin 2010]

Recommendations

Pericardial tamponade in nontraumatic arrests

Pericardiocentesis guided by ultrasound/echocardiography should be considered for treatment of cardiac arrest associated with suspected cardiac tamponade while non-image guided pericardiocentesis is an acceptable alternative only if echocardiography is not available. [Class A, Expert Consensus Opinion]

Placement of a pericardial drain may be beneficial and may obviate the need for subsequent operating room treatment. [Class B, Expert Consensus Opinion]

Emergency room thoracotomy and pericardiotomy can be considered for use in the treatment of nontraumatic cardiac arrest when pericardiocentesis is unsuccessful in relieving cardiac tamponade. [Class B, Expert Consensus Opinion]

Fluid resuscitation should be continued as indicated while awaiting definitive management. [Class A, Expert Consensus Opinion]

Pericardial tamponade in traumatic cardiac arrests

The optimal management of pericardial tamponade in traumatic cardiac arrest is definitive surgical drainage and treatment of the underlying cause. [Class A, Expert Consensus Opinion]

Emergency room thoracotomy and pericardiotomy should be considered as an acceptable alternative to operating room thoracotomy and pericardiotomy for treatment of traumatic cardiac arrest associated with cardiac tamponade. [Class A, LOE IV/Expert Consensus Opinion]

If neither of the above are possible, pericardiocentesis (ideally with ultrasound guidance) should be attempted. [Class A, Expert Consensus Opinion]

Fluid resuscitation should be continued as indicated while awaiting definitive management.

Pregnancy

Cardiac arrest in pregnancy is most commonly caused by:

- Cardiac disease
- Pulmonary thrombo-embolism
- Haemorrhage
- Sepsis
- Hypertensive disorders of pregnancy
- Poisoning and self-harm
- Amniotic fluid embolism
- Pregnant women can also have the same causes of cardiac arrest as females of the same age group (e.g. anaphylaxis, drug overdose, trauma).

The physiological changes associated with during pregnancy (increased cardiac output, blood volume, minute ventilation, oxygen consumption and reduced lung volumes) complicate basic management of the ABCs. Hypoxia occurs quickly, intubation is more difficult and gastric reflux is more likely.; for example, cardiac output, circulatory volume, minute ventilation, and oxygen consumption all increase. The gravid uterus causes compression of the abdominal organs and iliac and abdominal vessels when the mother is in the supine position. This results in reduced cardiac output and hypotension. There are no randomised controlled trials evaluating the effect of specialised obstetric resuscitation versus standard care in post-arrest pregnant women.

Although there is concern for the viability of the unborn child, effective resuscitation of the mother is the best way to optimise fetal outcome.

Resuscitation guidelines for pregnancy are based largely on case series, extrapolation from nonpregnant arrests, manikin studies and expert opinion based on the physiology of pregnancy and changes that occur in normal labour.

There is insufficient evidence to support or refute the use of specialised obstetric resuscitation techniques in maternal cardiac arrest and the use of therapeutic hypothermia in the post arrest period. Treatment may be guided by understanding the physiology of pregnancy, the importance of releasing aortocaval compression, the increased risk for hypovolemia, the compression advantage through positioning and the value of perimortem caesarean section early in maternal cardiac arrest. [Deakin 2010][Jeejeebhoy 2011]

Recommendations

In cardiac arrest, all the principles of basic and advanced life support apply. Specific additional factors include:

- Summon help immediately. For effective resuscitation of mother and fetus, expert help must be obtained; this should include an obstetrician and neonatologist.
- Manually displace the uterus to the left to remove caval compression. Add left lateral tilt if this is feasible (the optimal angle of tilt is unknown). Aim for between 15 and 30 degrees. The angle of tilt used needs to allow high quality chest compressions and if needed permit

Caesarean delivery of the fetus.

- Consider preparation for emergency Caesarean section, as the fetus will need to be delivered if initial resuscitation efforts fail. [Class A, LOE IV/Expert Consensus Opinion]

Pulmonary embolus

One double-blind RCT showed no improvement in survival to discharge with the use of tissue plasminogen activator following cardiac arrest with pulseless electrical activity. One RCT of fibrinolytics showed no difference in short- or long-term (30 days) survival or bleeding in patients randomised to receive tenecteplase or placebo during CPR. Patients with suspected pulmonary embolism were excluded from the study if open thrombolysis was possible in the prehospital setting. Thirty-seven cases with suspected pulmonary embolism were randomised in the trial. Of those, 2 of 15 patients survived when treated with tenecteplase compared with no survivors in the 22 patients of the placebo-treated group.

One meta-analysis of eight retrospective cohort studies with a variety of causes of cardiac arrest (pulmonary embolism, two studies; myocardial infarctions, four studies; cardiology diseases, one study; and nontraumatic etiologies, one study) demonstrated an increased rate of ROSC, survival to discharge, and long-term neurological function with fibrinolytic, but it also showed an increased risk of severe bleeding.

Nine studies of patients with presumed pulmonary embolism or all patients with cardiopulmonary arrests showed improvement with fibrinolysis in ROSC and admission to the hospital or ICU, but no improvement in survival to discharge. Three studies showed good neurological function in those who survived after successful fibrinolysis during CPR).

There is insufficient data to recommend any specific time period to continue external cardiac compressions after administration of fibrinolytic therapy, but 30 minutes was used in the largest controlled trial. [Deakin 2010]

Recommendations

Fibrinolytic therapy may be considered when pulmonary thrombo-embolism is suspected as the cause of the cardiac arrest. [Class B; Expert consensus opinion].

If a fibrinolytic drug is given in these circumstances, to allow time for optimal effect, CPR should be performed for at least an additional 30 min before termination of resuscitation attempts. [Class A; Expert consensus opinion] Consideration should be given to performing CPR for at least 60–90 min. [Class B; Expert consensus opinion].

See also [Guideline 11.5](#) Medications in Adult Cardiac Arrest.

Three stacked shocks

There are some situations where the patient with a perfusing rhythm, develops a shockable rhythm in a witnessed and monitored setting and the defibrillator is immediately available (eg.

first shock able to be delivered within 20 seconds). This may occur in the pre-hospital setting, emergency department, critical care and coronary care unit, cardiac catheter laboratory or the operating room. In these settings it may be appropriate to use a 3 stacked-shock technique (ie. without commencing chest compressions between shocks), especially where there may be a relative contraindication to external cardiac compressions (eg. after cardiac surgery).

The “3 stacked-shock sequence” (delivery of up to three shocks if needed) can be optimised by immediate rhythm analysis and charging of the defibrillator. This protocol may be of benefit in scenarios where the first shock is able to be delivered within 20 seconds, the time required for rhythm recognition and for recharging the defibrillator is short (i.e.: <10 seconds). The trade off here is interruptions in external cardiac compressions, so the patients need to be in a well-oxygenated, well-perfused state immediately before the arrest.

Recommendations

A sequence of up to 3 stacked shocks can be considered in patients with a perfusing rhythm who develop a shockable rhythm where the setting is:

- a witnessed and monitored setting and
- the defibrillator is immediately available (eg. first shock able to be delivered within 20 seconds and
- the time required for rhythm recognition and for recharging the defibrillator is short [i.e.: <10 seconds]).

If the patient does not have return of spontaneous circulation within 10 seconds of the delivery of the third shock compressions should be started immediately. If after any shock the patient develops a non-shockable rhythm, but does not have ROSC within 10 seconds, compressions should be started immediately. [Class B, Expert Consensus Opinion]

Toxicology

There is very little data regarding the success or otherwise of specific therapies during the management of cardiac arrest due to toxic substances. However there is a larger amount of lower level data, including case reports that have reported the use of a number of additional therapeutic interventions especially in situations of severe cardiac toxicity. [Smith 2010] [Deakin 2010]

Benzodiazepines

Overdose of benzodiazepines can cause loss of consciousness, respiratory depression and hypotension. No human studies or reports of any patients who had cardiac arrest solely resulting from benzodiazepine toxicity alone were identified.

Five reports of cardiac arrests resulting from exposure to combinations of medication that included one of the benzodiazepines were identified. One case report indicated that standard care alone was sufficient to reverse the severe cardiovascular toxicity attributed to an anaphylactic reaction to a benzodiazepine.

One case report described improved outcome when minor cardiovascular toxicity caused by

benzodiazepines was treated with flumazenil. Four studies indicated that flumazenil is unlikely to improve haemodynamic function in the setting of benzodiazepine overdose and may complicate other therapy

Flumazenil may improve ventilation in the deteriorating patient but it should be used with caution as two studies described serious adverse effects such as seizure, arrhythmia, hypotension, and withdrawal syndrome after flumazenil was given to patients presenting with decreased level of consciousness attributed to either benzodiazepine toxicity or an unknown cause. These side effects were more common with co-ingestants (such as tricyclic antidepressant and opioids), chronic benzodiazepine use or abuse, and known seizure disorder. [Deakin 2010]

Recommendation

There are no specific modifications required for cardiac arrest caused by benzodiazepines. Administration of flumazenil during cardiac arrest is not recommended [Class A, Expert Consensus Opinion].

Beta-blockers

There are no RCTs evaluating conventional versus alternative treatment of cardiac arrest caused by beta-blockers. Evidence is limited to case reports, extrapolations from nonfatal cases, severe cardiovascular toxicity cases, and animal studies. The wide variety of beta-blockers with differing pharmacological and physiochemical profiles makes it difficult to generalise from the limited data available.

In 13 case studies (n = 16) of human patients with severe cardiovascular toxicity caused by beta-blockers refractory to standard treatment, including vasopressors, the administration of glucagon (50–150mcg/kg) was followed by haemodynamic improvement and survival.

In two animal studies, high-dose insulin infusions (1U/kg/hr) given with glucose supplementation and electrolyte monitoring appeared effective (as measured by rates of improved haemodynamic stability and survival) in the setting of cardiovascular toxicity associated with beta-blockers. A single human case report documented that high-dose insulin (10U/kg/h IV), given with glucose supplementation and electrolyte monitoring, was followed by improved haemodynamic stability and survival to hospital discharge in the setting of severe cardiovascular toxicity associated with beta-blocker toxicity.

Case reports described the use of phosphodiesterase inhibitors calcium salts, extracorporeal support, intraaortic balloon pumps, and ECMO. Animal studies supported the use of calcium salts and the phosphodiesterase inhibitor amrinone. Animal studies suggested that dopamine, a combination of dopamine and isoprenaline, and milrinone may decrease the effectiveness of glucagon as an antidote for beta-blocker toxicity. [Deakin 2010]

Recommendations

If cardiac arrest occurs in the setting of toxicity due to beta-blockers, follow standard resuscitation guidelines [Class A, Expert Consensus Opinion]

Calcium Channel Blockers

There are no RCTs evaluating conventional versus alternative therapies for the treatment of cardiac arrest caused by calcium channel blockers. Evidence is limited to extrapolations from

nonfatal case reports of severe cardiovascular toxicity.

In 16 human case series (n = 28) high-dose insulin (bolus 0.5–2U/kg followed by 0.5U/kg/h infusion) given with glucose supplementation and electrolyte monitoring appeared effective (as measured by improved haemodynamic stability [25/28] and survival [26/28]) in the setting of severe cardiovascular toxicity associated with calcium channel blockers. [Deakin 2010]

Recommendations

If cardiac arrest occurs in the setting of toxicity due to calcium channel blockers, follow standard resuscitation guidelines [Class A, LOE IV/Expert Consensus Opinion].

Carbon monoxide

Three studies suggested that most patients who develop cardiac arrest from carbon monoxide poisoning will not survive to hospital discharge, regardless of whether hyperbaric oxygen therapy is administered following ROSC.

Two studies suggested that neurological outcomes were improved in patients (all severity excluding cardiac arrest; and mild-to-moderate, excluding loss of consciousness and cardiac instability who received hyperbaric oxygen therapy for carbon monoxide poisoning. However, two studies found no difference in neurologically intact survival. Two systematic reviews concluded that improvement in neurologically intact survival following the administration of hyperbaric oxygen to carbon monoxide poisoning patients was possible but unproven.

Two studies demonstrated that patients with carbon monoxide toxicity treated with hyperbaric oxygen who developed myocardial infarction have an increased risk of cardiovascular and all-cause mortality lasting at least seven years after the event. [Deakin 2010]

Recommendations

Patients who develop cardiac arrest caused by carbon monoxide rarely survive to hospital discharge, even if return of spontaneous circulation is achieved; however, hyperbaric oxygen therapy may be considered in these patients as it may reduce the risk of developing persistent or delayed neurological injury. The risks inherent in transporting critically ill post arrest patients to a hyperbaric facility may be significant, and must be weighed against the possibility of benefit on a case-by-case basis. [Class B, Expert Consensus Opinion].

Patients who develop myocardial injury caused by carbon monoxide have an increased risk of cardiac and all-cause mortality lasting at least seven years after the event; it is reasonable to recommend cardiology follow-up for these patients. [Class B, Expert Consensus Opinion]

Cocaine/Amphetamines

Sympathetic overstimulation associated with amphetamine/cocaine toxicity may cause agitation, symptomatic tachycardia, hypertensive crisis, hyperthermia and myocardial ischaemia with angina. Small doses of intravenous benzodiazepines (midazolam, diazepam, lorazepam) are effective first-line drugs. Glyceryl trinitrate and phentolamine can reverse amphetamine/cocaine induced coronary vasoconstriction. Possible myocardial necrosis should be assessed using the ECG and cardiac markers (e.g. troponin) in patients with amphetamine/cocaine-related chest pain. [Deakin 2010]

Recommendations

If cardiac arrest occurs in the setting of toxicity due to cocaine/amphetamines, follow standard resuscitation guidelines. [Class A, Expert Consensus Opinion]

Cyanide

There have been no relevant comparative or placebo-controlled human trials. Nine case series were identified. Treatment with hydroxocobalamin was reported in a total of 361 cases. No serious adverse effects of hydroxocobalamin were reported, and many patients with otherwise presumably fatal poisoning survived. Sodium thiosulfate use was reported in two case series, similarly with no adverse effects. Treatment with sodium nitrite, amyl nitrite and sodium thiosulfate was reported in 74 patients, with results indistinguishable from those of hydroxocobalamin and sodium thiosulfate. No case series using dicobalt edetate or 4-dimethylaminophenol (4-DMAP) were identified, but successful use in single cases has been reported. Hydroxocobalamin and sodium thiosulfate differ from alternatives in having negligible adverse effects, and on the basis of current evidence are the antidotes of choice. [Deakin 2010][Reade 2011]

Recommendations

Patients with severe cardiotoxicity (cardiac arrest, cardiovascular instability, metabolic acidosis, or altered mental status) caused by known or suspected cyanide poisoning should receive cyanide antidote therapy in addition to standard resuscitation guidelines. [Class A, Expert Consensus Opinion] The ARC recommends that adult patients with suspected severe cyanide poisoning (including those in cardiac arrest) should receive immediate parenteral hydroxocobalamin, 5g with repeat dosing up to 15g. [Class A, Expert Consensus Opinion]

Digoxin

There are no RCTs evaluating conventional versus alternative treatments for cardiac arrest caused by digoxin. Evidence is limited to 14 studies demonstrating the usefulness of antidigoxin Fab fragments for severe cardiac glycoside toxicity. [Deakin 2010]

Recommendations

If cardiac arrest occurs in the setting of toxicity due to digoxin, follow standard resuscitation guidelines.[Class A, Expert Consensus Opinion]

In adults and children with severe cardiovascular toxicity caused by digoxin and related cardiac glycosides, anti-digoxin Fab fragment therapy should be administered. [Class A, Expert Consensus Opinion]

Local anaesthetic agents

Local anaesthetic toxicity typically occurs in the setting of regional anaesthesia, when a bolus of local anaesthetic inadvertently enters the arterial or venous system, leading to refractory seizures, dysrhythmias or rapid cardiovascular collapse. There are no RCTs evaluating conventional versus alternative therapies for the treatment of cardiac arrest caused by local anaesthetics. Evidence is limited to case reports involving cardiac arrest and severe cardiovascular toxicity and animal studies.

Five single-case reports describe patients in cardiac arrest attributed to local anaesthetic intoxication, who were refractory to advanced life support conventional treatment, but who obtained ROSC soon after treatment with IV lipid emulsion. Five single-case reports describe

patients with acute, life-threatening cardiovascular toxicity from local anaesthetic intoxication, but who were not pulseless at the time of lipid administration. In three cases severe cardiovascular toxicity resolved rapidly following IV lipid, but in two other cases the patient's condition deteriorated to cardiac arrest after IV lipid, although the patients were resuscitated and survived to hospital discharge. [Deakin 2010][Morley 2011]

Recommendations

There is insufficient clinical evidence to suggest any change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest caused by local anaesthetics. Animal studies and case reports suggest severe cardiovascular toxicity or cardiac arrest attributable to local anaesthetic intoxication may respond to treatment with intravenous lipid emulsion. [Class B, LOE IV/Expert Consensus Opinion]

Opioids

There are no RCTs evaluating conventional versus alternative treatments for cardiac arrest caused by opioids. Evidence is limited to studies of mild, moderate, and severe cardiovascular toxicity. Evidence from studies assessing other endpoints (efficacy of naloxone), as well as animal studies, support the use of assisted ventilation before giving naloxone in opioid-poisoned patients with severe cardiopulmonary toxicity.

The use and safety of naloxone is supported by human case studies as well as those assessing other endpoints (alternate routes of administration). Naloxone can be given intravenously, intramuscularly, intranasally, and into the trachea. [Deakin 2010]

Recommendations

In cardiac arrest:

If cardiac arrest occurs in the setting of toxicity due to opioids, follow standard resuscitation guidelines. Administration of naloxone offers no survival advantage in this setting. [Class A, Expert Consensus Opinion]

In patients with cardio-respiratory instability (pre-arrest setting):

In adults with severe cardio-respiratory toxicity caused by opioids, ventilation should be assisted using a bag-mask before administration of naloxone, and tracheal intubation if there is no response to naloxone. Naloxone can be given via intravenous, intramuscular intranasal, or intraosseous routes. [Class A, Expert Consensus Opinion]

Tracheal route may be used if conditions preclude other routes of administration. [Class A, Expert Consensus Opinion]

In opiate induced respiratory arrest the dose of naloxone varies between small aliquots of 100mcg (a more conservative approach to achieve return of spontaneous respiration and airway control) and a large 2mg dose aimed at achieving immediate and complete reversal. The conservative approach has the advantage of maintaining a safe airway without precipitating an acute withdrawal response with associated risk of harm to both patient and clinician. [Class A, Expert Consensus Opinion]

Rescuers need to be aware that the effects of naloxone may wear off and signs of opioid toxicity may reappear.

Tricyclic anti-depressants

There are no RCTs evaluating conventional versus alternative treatments for cardiac arrest caused by tricyclic antidepressant toxicity. Evidence from COSTR 2010 was limited to one small case series of cardiac arrest patients which demonstrated improvement with the use of sodium bicarbonate and adrenaline. The evidence for the management of cardiotoxicity caused by tricyclic antidepressant was limited to case reports, case series, and animal studies. The use of sodium bicarbonate has been described in two case series and six animal studies. The use of hyperventilation was described in one small case series and one animal study. For a more detailed summary of the relevant studies, see Deakin 2010.

Recommendations

There is insufficient clinical evidence to suggest any change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest or cardiotoxicity caused by tricyclic antidepressants. Since sodium bicarbonate bolus is the mainstay of therapy in the setting of tricyclic-induced cardiac conduction abnormalities. This treatment strategy should be considered during the arrest and in the post arrest period of care for patients surviving cardiac arrest caused by tricyclic antidepressant toxicity associated with wide QRS complexes. [Class A, Expert Consensus Opinion] When mechanical ventilation is required respiratory acidosis should be avoided. [Class A, Expert Consensus Opinion]

Trauma

Common causes of cardiac arrest associated with trauma include hypovolaemia, tension pneumothorax and pericardiac tamponade.

Recommendations

If available, ultrasound will help rapidly diagnose potentially reversible causes such as haemoperitoneum, haemothorax, tension pneumothorax and cardiac tamponade. This requires a trained operator and should not delay treatment. [Class A, Expert Consensus Opinion] Treatment should be based on the identified underlying cause. [Class A, Expert Consensus Opinion]

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