ANZCOR Guideline 11.1 – Introduction to Advanced Life Support

Summary

Who does this guideline apply to?

This guideline applies to adults who require advanced life support.

Who is the audience for this guideline?

This guideline is for health professionals and those who provide healthcare in environments where equipment and drugs are available.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) make the following recommendations:

1. As soon as possible, Advanced Life Support treatments are used to supplement any adult receiving Basic Life Support.
2. Hospitals consider using early warning, rapid response team, or medical emergency team systems to reduce the incidence of in-hospital cardiac arrests and in-hospital mortality.
3. Hospitals use a system validated for their specific patient population to identify individuals at increased risk of serious clinical deterioration, cardiac arrest, or death, both on admission to hospital and during their stay.

Guideline

1 Definitions

Cardiopulmonary resuscitation (CPR) is the technique of chest compressions combined with rescue breathing. The purpose of cardiopulmonary resuscitation is to temporarily maintain a circulation sufficient to preserve brain function until specialised treatment is available.

CPR has 3 fundamental components:

A Airway assessment and management.
B Breathing assessment and management.
C Circulation assessment and management.

Basic Life Support (BLS) is the preservation or restoration of life by the establishment of and/or the maintenance of airway, breathing and circulation, and related emergency care. Adjunctive equipment is **NOT** essential for basic life support, however the use of Automated External Defibrillators (AEDs) by persons trained in their use but not trained in ALS techniques is encouraged by ANZCOR.

Advanced Life Support (ALS) is basic life support with the addition of invasive techniques e.g. manual defibrillation, advanced airway management, intravenous access and drug therapy.

Patients requiring BLS and ALS commonly have underlying problems including:

- ischaemic heart disease
- chronic respiratory disease
- drug overdose / toxicity
- drowning
- trauma
- electrolyte abnormalities
- peri-arrest arrhythmias.

2 Background

BLS is only a temporary measure to maintain ventilation and circulation. Effective external cardiac compression provides a cardiac output of only 20-30% of the pre-arrest value[1], and expired air resuscitation provides ventilation with an inspired oxygen concentration of only 15-18%[2]. Electrical defibrillation is the mainstay of treatment for ventricular fibrillation and pulseless VT. The chance of successful defibrillation decreases with time. Therefore performance of good CPR and decreasing the time to defibrillation are the first priorities in resuscitation from sudden cardiac arrest. The purpose of BLS is to help maintain myocardial and cerebral oxygenation until ALS personnel and equipment are available.

- Effective BLS may increase the likelihood of successful defibrillation[3].
- Effective BLS buys time until reversible causes can be diagnosed and/or treated.

Monitoring what we do is becoming even more important, including:

- the effectiveness of compressions (depth, rate and hands off periods);
- the adequacy of ventilation (avoiding over-ventilation and consequent deleterious effects);
- the timing of defibrillation with regard to likelihood of success (eg compressions before and after).

Emphasis is now also being focused on the pre-arrest period (early detection and prevention of cardiac arrest) and the post-resuscitation management.

An extensive review of many aspects of advanced life support was performed as part of the 2010 and 2015 Consensus on Science process [5-12]. The information from this process has been incorporated into the following guidelines wherever appropriate.

3 Prevention of Cardiac Arrest

Children and young adults presenting with characteristic symptoms of arrhythmic syncope should have a specialist cardiology assessment, which should include an ECG and in most cases an echocardiogram and exercise test [11] [Class A, Expert consensus opinion].

Characteristics of arrhythmic syncope include: syncope in the supine position, occurring during or after exercise, with no or only brief prodromal symptoms, repetitive episodes, or in individuals with a family history of sudden cardiac death (SCD). In addition, non-pleuritic chest pain, palpitations associated with syncope, seizures (when resistant to treatment, or occurring at night) should raise suspicion of increased risk of arrhythmic syncope. Systematic evaluation in a clinic specializing in the care of those at risk for SCD is recommended in family members of young victims of SCD or those with a known cardiac disorder resulting in an increased risk of SCD [11] [Class B; Expert consensus opinion].

4 In-Hospital Pre-Arrest Detection and Management

In adult patients admitted to hospital, there is variable evidence regarding the use of early warning systems/rapid response team (RRT) systems or medical emergency team (MET) systems (compared with no such systems) to reduce cardiac and respiratory arrests and hospital mortality [11].

ANZCOR suggests that hospitals consider the introduction of an EWS/response team/MET system to reduce the incidence of IHCA and in-hospital mortality (CoSTR 2015 weak recommendation, low-quality evidence) [12]. It is reasonable, and increasingly made mandatory by health authorities, that hospitals provide a system of care that includes [11,13]:

- staff education about the signs of patient deterioration,;
- appropriate and regular vital signs monitoring of patients;
- clear guidance (e.g. via calling criteria or early warning scores) to assist staff in the early detection of patient deterioration;
- a clear, uniform system of calling for assistance, and;
- a clinical response to calls for assistance.

[Class A; Expert consensus opinion]

There is insufficient evidence to identify the best methods for the delivery of these components and, based on current evidence, this should be based on local circumstances.[11, 13] [Class A; Expert consensus opinion]

Hospitals should use a system validated for their specific patient population to identify individuals at increased risk of serious clinical deterioration, cardiac arrest, or death, both on admission and during hospital stay [11] [Class A; Expert consensus opinion].

There is insufficient evidence to identify specific educational strategies that improve outcomes (e.g. early recognition and rescue of deteriorating patient at risk of cardiac/respiratory arrest). Educational efforts have a positive impact on knowledge, skills, attitudes/confidence, and increase the frequency of activation of a response and should therefore be considered [11] [Class A; Expert consensus opinion].

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ANZCOR Guideline 11.2 – Protocols for Adult Advanced Life Support

Summary

Who does this guideline apply to?

This guideline applies to adults who require advanced life support (ALS).

Who is the audience for this guideline?

This guideline is for health professionals and those who provide healthcare in environments where equipment and drugs are available.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) make the following recommendations:

1. That the Adult ALS algorithm be used as a tool to manage all adults who require advanced life support.
2. Good quality CPR and reducing time to defibrillation are the highest priorities in resuscitation from sudden cardiac arrest.
3. Rescuers should aim to minimise interruptions to CPR during any ALS intervention.

Guideline

1 Advanced Life Support Algorithm

The flow diagram illustrates the sequence of actions to be undertaken once equipment and drugs are available. Several tasks in the diagram may be undertaken at the same time.

The algorithm is based on the following considerations:

1. The importance of good CPR and early defibrillation in achieving successful outcomes. Ventricular Fibrillation (VF) is in many situations the primary rhythm in sudden cardiac arrest. The vast majority of survivors come from this group.

   The chance of successful defibrillation decreases with time. Therefore the performance of good CPR and decreasing the time to defibrillation are the highest priorities in resuscitation from sudden cardiac arrest.

   The amplitude and waveform of VF deteriorate as high energy phosphate stores in the myocardium decrease. This rate of decrease can be slowed, or even reversed by effective BLS.[1]

2. Automated External Defibrillators (AEDs) can accurately diagnose cardiac rhythms and separate them into two groups:

   a. "Shockable" = those responsive to defibrillation
   b. "Non-shockable" = those unresponsive to defibrillation

3. There are interventions that are indicated in all causes of cardiac arrest.

4. There is a group of potentially reversible conditions that, if unrecognised or left untreated during cardiac arrest, may prevent successful resuscitation.

2 Notes on the Algorithm

2.1 Good quality CPR

The provision of good quality CPR is the cornerstone of advanced life support. As outlined in Guideline 11.1.1 this includes delivery of chest compressions over the lower half of the sternum at a depth of at least 5 cm, and at a rate of approximately 100-120 per minute, while minimising interruptions to compressions at all times.

2.2 Assess rhythm

As soon as the defibrillator is available, the pads should be placed on the patient's chest, it should be charged and, the rhythm analyzed. If a rhythm compatible with spontaneous circulation is observed, the defibrillator should be disarmed and the pulse checked [Class A; Expert Consensus Opinion].

2.3 Shockable Rhythm

- Ventricular fibrillation is asynchronous chaotic ventricular activity that produces no cardiac output.
- Pulseless ventricular tachycardia is a wide complex regular tachycardia associated with no clinically detectable cardiac output.
- A defibrillator shock should be administered according to the algorithm.
- Administer a single shock and immediately resume CPR for 2 minutes after delivery of shock. Do not delay recommencing CPR to assess the rhythm.
  [Class A; LOE II to IV][2]

2.4 Energy levels

- **Monophasic:** the energy level for adults should be set at maximum (usually 360 Joules) for all shocks. [Class A; LOE II][2]

- **Biphasic waveforms:** the default initial energy level for adults should be set at 200J. Other energy levels may be used providing there is relevant clinical data for a specific defibrillator that suggests that an alternative energy level provides adequate shock success (eg. usually greater than 90%) [Class A; LOE II][2] .

ANZCOR suggests that if the first shock is not successful and the defibrillator is capable of delivering shocks of higher energy, it is reasonable to increase the energy to the maximum available for subsequent shocks (CoSTR 2015 weak recommendation, very low quality evidence).[3]

2.5 Immediate CPR

Interruptions to CPR decrease the chance of survival from cardiac arrest. While defibrillation is of paramount importance for VF/VT, a period of well performed CPR immediately after each shock can help maintain myocardial and cerebral viability, and improves the likelihood of subsequent shock success.[1]

- During CPR advanced life support interventions are applied and potential causes of arrest sought.
- After each defibrillation continue a further 2 minutes of CPR, unless responsiveness or normal breathing become apparent.
- If using a defibrillator in manual mode, the defibrillator should be charged during CPR as the end of the 2 minute loop of CPR approaches, to minimise interruptions to CPR and increase the likelihood of shock success.[4]
- Rhythm is then reassessed and treatment is directed as necessary. If rhythm assessment results in a significant interruption to CPR then a further 2-minute period of CPR is recommended before further shocks are delivered. This is done to obtain the benefits of CPR on VF waveform and increase the likelihood of shock success.
- Consideration should be given to administration of a vasopressor in the period of CPR after the second failed defibrillation attempt. Consideration should be given to administration of an antiarrhythmic after the third failed defibrillation attempt. The sequence of escalating advanced life support would then be:
  1. attempt defibrillation ensure good CPR
  2. attempt defibrillation add vasopressor (adrenaline 1 mg)
  3. attempt defibrillation, add anti-arrhythmic (amiodarone 300 mg).

  [Class A; LOE II to IV][2]

2.6 Non-shockable rhythm (Non VF/VT)

- Asystole is characterised by the absence of any cardiac electrical activity.
- Pulseless Electrical Activity (PEA) (sometimes referred to Electromechanical Dissociation [EMD]) is the presence of a coordinated electrical rhythm without a detectable cardiac output.
- The prognosis in this group of cardiac rhythms or asystole is much less favourable than with VF/VT.
- During CPR advanced life support interventions are applied and potential causes of arrest sought.
- Defibrillation is not indicated and the emphasis is on CPR and other ALS interventions (e.g.. intravenous access, consideration of advanced airway, drugs and pacing).

[Class A; Expert consensus opinion].

2.7 During CPR

The following interventions apply to all rhythms and are carried out continuously or during each loop of the algorithm. Each loop comprises 5 sets of 30 compressions (at approximately 100-120 per minute) : 2 breaths, which equates to approximately 2 minutes.

Other management priorities during CPR:

- Minimise interruptions to CPR during ALS interventions [Class A; LOE III-2].
- Administer 100% oxygen when available (CoSTR 2015 weak recommendation, very low quality evidence).[3]
- Obtain intravenous or intra-osseous access [Class A; LOE II].
- Consider airway adjuncts, but attempts to secure the airway should not interrupt CPR for more than 5 seconds [Class A; Expert consensus opinion].
- Waveform capnography should be used to confirm airway placement and monitor the adequacy of CPR (CoSTR 2015 strong recommendation, low quality evidence).[3]
- Adrenaline should be administered every second loop (approximately every 4 minutes) [Class A; Expert consensus opinion].
- Other drugs/electrolytes should be considered depending on the individual circumstances [Class A; Expert consensus opinion].

2.8 Medications during CPR

Vasopressors

There are no placebo-controlled studies that show that the routine use of any vasopressor at any stage during human cardiac arrest increases survival to hospital discharge, though they have been demonstrated to increase Return of Spontaneous Circulation. Current evidence is insufficient to support or refute the routine use of any particular drug or sequence of drugs. Despite the lack of human data it is reasonable to continue to use vasopressors on a routine basis.[5]

Adrenaline (1 mg), when indicated, should be administered after rhythm analysis (± shock), at the time of recommencement of CPR [Class A; Expert consensus opinion].

Antiarrhythmics

There is no evidence that giving any antiarrhythmic drug routinely during human cardiac arrest increases rate of survival to hospital discharge. In comparison with placebo and lignocaine the use of amiodarone in shock-refractory VF improves the short-term outcome of survival to hospital admission. Despite the lack of human long-term outcome data it is reasonable to continue to use antiarrhythmic drugs on a routine basis.[5]

Amiodarone (300 mg) should be administered after the third failed attempt at defibrillation, at the time of recommencement of CPR [Class A; LOE II].

Other drugs

There is no evidence that routinely giving other drugs (e.g. buffers, aminophylline, atropine, calcium, magnesium) during human cardiac arrest increases survival to hospital discharge.[5]

2.9 Correct Reversible Causes

Very few data address the aetiology of cardiac arrest directly. One prospective study and one retrospective study suggested that rescuers can identify some non-cardiac causes of some arrests.[6,7] The physical circumstances, history, precipitating events, clinical examination, or the use of adjunct techniques (such as ultrasound) may enable the rescuer to determine a cardiac or non-cardiac cause of the cardiorespiratory arrest. The rescuer should undertake interventions based on the presumed aetiology (cardiac or non-cardiac).

4 Hs and 4 Ts are a simple reminder of conditions that may precipitate cardiac arrest or decrease the chances of successful resuscitation. These conditions should be sought and, if present, corrected in every case [Class A; Expert consensus opinion].

- **H**ypoxaemia
- **H**ypovolaemia
- **H**yper/hypokalaemia & metabolic disorders
- **H**ypo/hyperthermia
- **T**ension pneumothorax
- **T**amponade
- **T**oxins / poisons / drugs
- **T**hrombosis-pulmonary / coronary

Fluid administration

There is insufficient evidence to recommend for or against the routine infusion of intravenous fluids during cardiac arrest resuscitation.[5]

Fluids should be infused if hypovolemia is suspected (hypovolaemic shock would normally require the administration of at least 20 mL/kg) [Class A; Expert consensus opinion].

Thrombolytics

Routine administration of fibrinolytics for the treatment of in-hospital and out-of hospital cardiac arrest is not recommended.[5]

Fibrinolysis should be considered in adult patients with cardiac arrest with proven or suspected pulmonary embolism [Class A; Expert consensus opinion].

Checking Resuscitation Equipment

ANZCOR is aware of cases where equipment failure (e.g. oxygen pipes being incorrectly connected resulting in hypoxic gases being administered, and resuscitation bag valve devices incorrectly assembled) that have led to adverse outcomes. The checking and maintenance of hospital and resuscitation equipment is covered by National Standards and local policies.

Practitioners involved in resuscitation should always be alert to errors of equipment installation, assembly or use and have checking processes to minimise these risks.

2.10 Post Resuscitation Care

After the return of spontaneous circulation (ROSC), post-resuscitation care commences (see Guideline 11.7 and 11.8).

Re-evaluate the patient using the standard ABCDE approach: Airway Breathing Circulation Disability and Exposure.

Other considerations include obtaining a 12 lead ECG and a chest radiograph. The adequacy of perfusion should be assessed, and the need for reperfusion therapy should be considered (eg. thrombolytics or percutaneous coronary intervention). The adequacy of oxygenation and ventilation should be confirmed and maintained (and advanced airway may be required).

Targeted Temperature Management may be instituted if indicated, and further investigation for reversible causes should be continued, and treatment instituted where necessary. See also guideline 11.7 and 11.8 [Class A; Expert consensus opinion].

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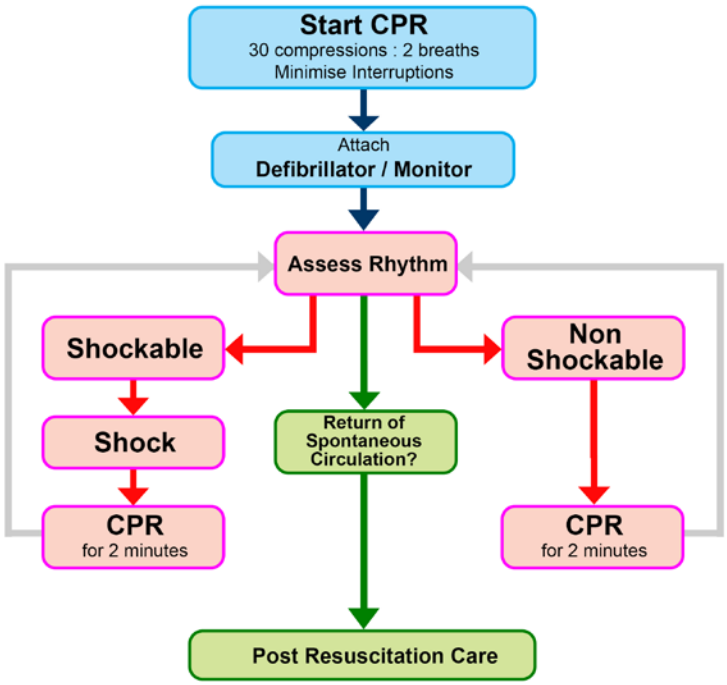
Advanced Life Support for Adults



During CPR
Airway adjuncts (LMA / ETT)
Oxygen
Waveform capnography
IV / IO access
Plan actions before interrupting compressions
(e.g. charge manual defibrillator)

Drugs

Shockable
* Adrenaline 1 mg after 2nd shock
(then every 2nd loop)
* Amiodarone 300mg after 3 shocks

Non Shockable
* Adrenaline 1 mg immediately
(then every 2nd loop)

Consider and Correct
Hypoxia
Hypovolaemia
Hyper / hypokalaemia / metabolic disorders
Hypothermia / hyperthermia
Tension pneumothorax
Tamponade
Toxins
Thrombosis (pulmonary / coronary)

Post Resuscitation Care
Re-evaluate ABCDE
12 lead ECG
Treat precipitating causes
Aim for: SpO2 94-98%, normocapnia and normoglycaemia
Targeted temperature management

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GUIDELINE 11.3

PRECORDIAL THUMP & FIST PACING

INTRODUCTION

A precordial thump is a single sharp blow delivered by the rescuer's fist to the mid sternum of the victim's chest.

RECOMMENDATIONS

The precordial thump may be considered for patients with monitored, pulseless ventricular tachycardia if a defibrillator is not immediately available. [Class B; LOE IV]

The precordial thump is relatively ineffective for ventricular fibrillation, and it is no longer recommended for this rhythm.[1]

There is insufficient evidence to recommend for or against the use of the precordial thump for witnessed onset of asystole caused by AV-conduction disturbance.[1]

The precordial thump should not be used for unwitnessed cardiac arrest.[1]

A precordial thump should not be used in patients with a recent sternotomy (eg. for coronary artery grafts or valve replacement), or recent chest trauma.

TECHNIQUE

The clenched fist of the rescuer is held approximately 25-30cm (10-12 inches) above the sternum of the victim. The fist is then brought down sharply so the inside (medial, ulna) side of the fist makes contact with the mid-sternum of the victim's chest.

The precordial thump should not be taught as an isolated technique. It should be taught as part of an ALS course in which the student learns to identify life threatening arrhythmias and the appropriate steps to undertake if the chest thump fails. It is best taught with the skill of defibrillation.

DISCUSSION

In five prospective case series of out-of-hospital and two series of in-hospital VF cardiac arrest, healthcare provider administration of the precordial thump did not result in ROSC.

In three prospective case series of ventricular tachycardia in the electrophysiology lab administration of the precordial thump by experienced cardiologists was of limited use (1.3% ROSC). When events occurred outside of the electrophysiology lab, in 6 case series of in and out of the hospital VT the precordial thump was followed by ROSC in 19% of patients. Rhythm deterioration following precordial thump occurred in 3% of patients and was observed predominantly in patients with prolonged ischemia or digitalis-induced toxicity.

In three case series of asystolic arrest the precordial thump, but not fist-pacing, was sometimes successful in promoting ROSC when administered by health care providers to patients with witnessed asystole (some clearly p-wave asystolic arrest) for out-of-hospital cardiac arrest (OHCA) and in-hospital cardiac arrest (IHCA).[1]

Two case series and a case report documented the potential for complications from use of the precordial thump including sternal fracture, osteomyelitis, stroke, and rhythm deterioration in adults and children.[1]

PERCUSSION (FIST) PACING

The administration of serial rhythmic blows to the chest has been proposed as a technique to provide mechanical pacing until an electrical pacemaker is available.

There is little evidence supporting fist or percussion pacing in cardiac arrest, particularly when the effect of the maneuver cannot be confirmed by continuous electrocardiographic monitoring and assessment of a pulse. Evidence consists of six single-patient case reports and a moderate sized case series with mixed asystole and bradycardia.[1]

Recommendation

For patients in cardiac arrest, percussion (fist) pacing is not recommended.[1] However, percussion (fist) pacing may be considered in haemodynamically unstable bradyarrhythmias until an electrical pacemaker (transcutaneous or transvenous) is available.[2]

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ANZCOR Guideline 11.4 – Electrical Therapy for Adult Advanced Life Support

Summary

Defibrillation as soon as possible provides the best chance of survival in victims with VF or pulseless VT.

Who does this guideline apply to?

This guideline applies to adults who require advanced life support.

Who is the audience for this guideline?

This guideline is for health professionals and those who provide healthcare in environments where equipment and drugs are available.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) make the following recommendations:

1. A defibrillation shock is delivered as soon as a defibrillator is available.
2. Paddles or pads are placed on the exposed chest in an anterior-lateral position or an anterior-posterior position.
3. In patients with an ICD or a permanent pacemaker the defibrillator pad/paddle is placed on the chest wall ideally at least 8 cm from the generator position.
4. Self-adhesive defibrillation pads are used for defibrillation.
5. Biphasic waveforms are used for defibrillation.
6. For **Monophasic waveforms:** the initial energy level for adults is set at maximum (usually 360 Joules) for all shocks.
7. For **Biphasic waveforms:** the default energy level for adults is set at 200J for all shocks. Other energy levels may be used providing there is relevant clinical data for a specific defibrillator that suggests that an alternative energy level provides adequate shock success (e.g. Usually greater than 90%).
8. If the first shock is not successful and the defibrillator is capable of delivering shocks of higher energy, it is reasonable to increase the energy to the maximum available for subsequent shocks.
9. A single shock strategy is used in patients in cardiac arrest requiring defibrillation for VF or pulseless VT.

10. The use of AEDs to facilitate early defibrillation in hospitals is reasonable, but services that introduce AEDs must be aware of the possible adverse impact of interruptions to CPR, especially in non-shockable rhythms.

Guideline

A defibrillation shock when applied through the chest produces simultaneous depolarization of a mass of myocardial cells and may enable resumption of organised electrical activity.

1 Indications

A defibrillation shock is indicated for treating Ventricular Fibrillation (VF) and pulseless Ventricular Tachycardia (VT).

2 Timing of Defibrillation

The likelihood of defibrillation success decreases with time until definitive treatment (i.e. defibrillation) is initiated.[5] Interruptions to external cardiac compression (e.g. for rhythm assessment or pulse checks) should be minimised. However, good CPR may even increase the likelihood of defibrillation success.[1] The results of clinical studies assessing the usefulness of a strategy providing a period of CPR before defibrillation rather than a strategy providing immediate defibrillation are not consistent.

In two randomized controlled trials, a period of 1.5 to 3 minutes of CPR by EMS personnel before defibrillation did not improve return of spontaneous circulation (ROSC) or survival to hospital discharge in patients with out-of-hospital VF or pulseless VT, regardless of EMS response interval. One before and after study and another case series failed to demonstrate significant improvements in ROSC or survival to hospital discharge when a strategy of CPR before defibrillation (CPR first) was compared to a shock first strategy. In the Hayakawa study, the CPR first group showed a higher rate of favourable neurologic outcome 30 days and one year after cardiac arrest.

One randomized controlled trial and one clinical trial with historic controls comparing CPR first versus shock first also found no overall difference in outcomes.

However, in both studies, improvements in ROSC, survival to hospital discharge, neurologic outcome and one-year survival were observed in a subgroup of patients who received CPR first where the EMS response interval was greater than 4 to 5 minutes.[2]

Recommendation

ANZCOR suggest delivering a defibrillation shock as soon as a defibrillator is available [Class A; Consensus expert opinion].

3 Positioning of Electrodes

There are no studies in patients with VF/pulseless VT comparing directly the effects of various positions of pad/paddle placement on defibrillation success and ROSC. Most studies evaluate cardioversion (e.g. AF) or secondary endpoints (e.g. transthoracic impedance). Eleven studies found all four positions (anterior-apex, anterior-posterior, anterior-left infrascapular, anterior right-infrascapular) to be equally effective in defibrillation (for VF/pulseless VT) or elective AF cardioversion success.

Four studies support the anterior-posterior position, one study supports the anterior-lateral position and one study supports the anterior-apex position.

Five studies found no effect of electrode position on transthoracic impedance. One study showed that pads/paddles should be placed under the breast tissue and two studies showed that hirsute males should be shaved before to application of pads. Of the 36 studies reviewed, only four examined biphasic waveforms that have gained widespread use.[2]

Recommendation

It is reasonable to place paddles or pads on the exposed chest in an anterior-lateral position. One paddle or pad is placed on the midaxilliary line over the 6th left intercostal space and the other on the right parasternal area over the 2nd intercostal space [Class A; LOE III-2]. Acceptable alternative positions are the anterior-posterior (for paddles and pads) and apex-posterior (for pads). In large-breasted individuals it is reasonable to place the left electrode pad (or paddle) lateral to or underneath the left breast, avoiding breast tissue. Consideration should be given to the rapid removal of excessive chest hair prior to the application of pads/paddles but emphasis must be on minimizing delays in shock delivery [2] [Class B; LOE IV].

3.1 Positioning of electrodes in the presence of a pacemaker/internal defibrillator

Two case series reported pacemaker or implantable cardioverter defibrillator (ICD) malfunction after external defibrillation when the pads were placed in close proximity to the device generator. One small study on atrial cardioversion demonstrated that positioning the pads on the chest at least 8 cm from the device generator did not produce significant damage to pacing sensing and capturing. [2]

Recommendation

In patients with an ICD or a permanent pacemaker, the placement of pad/paddles should not delay defibrillation. When treating an adult with a permanent pacemaker or an implantable cardioverter defibrillator, the defibrillator pad/paddle should be placed on the chest wall ideally at least 8 cm from the generator position [Class A; LOE IV] .The anterior-posterior and anterior-lateral pad/paddle placements on the chest are acceptable in patients with a permanent pacemaker or ICD [2] [Class B; Extrapolated evidence].

One case report suggested that pacemaker spikes generated by devices programmed to unipolar pacing may confuse AED software and emergency personnel and may prevent the detection of VF.[2]

4 Size of Electrodes

One study has demonstrated that transthoracic impedance decreased and shock success increased with increasing pad size (from 8 to 12 cm). Ten other studies showed that larger pad/paddle sizes (8 to 12 cm diameter) lowered transthoracic impedance and that maximum pad/paddle size was limited by the chest wall size and anatomy. No data related to survival outcome were included in these studies.[2]

There is insufficient evidence to recommend a specific electrode size for optimal external defibrillation in adults. However, it is reasonable to use a pad size >8 cm^2 [Class B; Extrapolated evidence].

5 Paddles / Self Adhesive Pads

Evidence from one small, good quality retrospective control study in 1987 showed that self-adhesive pads were associated with a significantly improved rate of ROSC and hospital admission compared with hand-held paddles. Several studies have shown the practical benefits of pads over paddles for routine monitoring and defibrillation.[2]

One prospective study comparing pads and paddles found lower transthoracic impedance when paddles applied at an optimal force of 8 kg were compared with pads. In a cohort study in patients with atrial fibrillation (AF) the use of hand-held paddles placed in the anterior-posterior position increased the success rate of monophasic cardioversion compared with similarly placed self-adhesive electrodes for monophasic defibrillation. The overall cardioversion success rate for biphasic defibrillators was high (>95%) in all groups. In the majority of other studies, self-adhesive electrodes are associated with similarly high cardioversion success rates.[2]

Recommendation

ANZCOR recommend using self-adhesive defibrillation pads in preference to paddles for defibrillation [Class A; Expert consensus opinion]. They are safe and effective and offer advantages (e.g. facilitating pacing, charging during compressions, safety [including removing risk of fires]) over defibrillation paddles [Class A;LOE III-3, Extrapolated evidence]. If paddles are used, the application of firm pressure and conductive gel pads are recommended for maximum electrical contact. Care should be taken to ensure that pads or electrodes are applied in accordance with manufacturer's instructions and are not in electrical contact with each other [Class A; Expert consensus opinion].

The composition of the conductive material of defibrillation electrodes influences transthoracic impedance. In terms of cardiac arrest outcomes, there is insufficient evidence to recommend a specific composition of the defibrillation electrode conductive material.[6]

6 Defibrillation Waveform

In three randomized trials and four other human studies biphasic waveforms had higher shock success rates compared with monophasic defibrillation. Shock success is usually defined as termination of ventricular fibrillation (VF) 5 seconds after the shock.[6] Another randomized study comparing transthoracic incremental monophasic with biphasic defibrillation for out-of-hospital pulseless VT/VF cardiac arrest found no differences in any outcome. A single cohort study using the 2000 International Guidelines demonstrated better hospital discharge and neurologic survival with biphasic than with monophasic waveforms. However, there are confounding factors in that the intervals between the first and second shocks (of three stacked shocks) were shorter with the biphasic defibrillators. There is no clinical evidence for superiority for any specific biphasic waveform over another.[2]

Recommendation

Biphasic waveforms are recommended to be used for defibrillation [Class A; Expert consensus opinion]. There is insufficient evidence to recommend any specific biphasic waveform. In the absence of biphasic defibrillators, monophasic defibrillators are acceptable [Class B; Expert consensus opinion].

7 Energy Levels

7.1 Biphasic truncated exponential waveform

Evidence from one well-conducted randomized trial and one other human study employing biphasic truncated exponential (BTE) waveforms suggest that higher energy levels are associated with higher shock success rates. In the randomized trial, the first shock success rate was similar with 150 J and 200 J.[2]

7.2 Biphasic pulsed waveform

In one study using pulsed biphasic waveforms at 130J the first shock success rate was 90%. [2]

7.3 Rectilinear biphasic waveform

When defibrillation success was defined as ROSC (this differs from the definition used in other studies), one study using a rectilinear biphasic waveform showed that an organized rhythm was restored by the first shock (120 J) in 23% of cases. Success rate for the termination of VF at 5 seconds was not published for this waveform.[2]

7.4 Monophasic waveform (damped sinusoid or truncated exponential)

Evidence from three studies of monophasic defibrillation suggest equivalent outcomes with lower and higher starting energies.[2]

7.5 Myocardial damage associated with higher energy level shocks

Several animal studies have suggested the potential for myocardial damage with higher energy shocks using BTE or monophasic waveforms. Human studies involving BTE waveforms have not shown harm as indicated by biomarker levels, ECG findings, and ejection fractions with energy levels up to 360J.[2]

7.6 Fixed versus escalating energy levels

One randomized trial of 150 J fixed versus 200 J-300 J-360 J shocks and one study (with concurrent controls) of 150 J fixed versus 100 J-150 J-200 J shocks supported the use of an escalating energy biphasic defibrillation protocol compared with a fixed dose defibrillation protocol.

In one study (escalating 200J-200J-360J shocks) the success rate of defibrillation for recurrent VF declined with the number of recurrences. However, these studies were not designed to demonstrate an improvement in the rate of ROSC or survival to hospital discharge.

One study of fixed-dose biphasic defibrillation suggested that defibrillation success improved with three shocks. All of these studies were done with the three shock protocol (before the change in Guidelines 2005).[2]

Recommended Energy Levels

- **Monophasic:** the energy level for adults should be set at maximum (usually 360 Joules) for all shocks. [Class A; LOE III-2][1]
- **Biphasic waveforms:** the default energy level for adults should be set at 200J for all shocks. Other energy levels may be used providing there is relevant clinical data for a specific defibrillator that suggests that an alternative energy level provides adequate shock success (e.g. Usually greater than 90%) [Class A; LOE II]. [2]

ANZCOR suggest if the first shock is not successful and the defibrillator is capable of delivering shocks of higher energy, it is reasonable to increase the energy to the maximum available for subsequent shocks [CoSTR 2015, weak recommendation, very low quality evidence].[3] Escalating shock energy may prevent the risk of refibrillation and is in line with current practices [CoSTR 2015, values and preferences statement].

8 Single Shock Protocol

One study showed no survival benefit from a protocol that included a single shock protocol compared to a three-shock protocol. Evidence from three pre-post design studies suggested significant survival benefit with a single shock defibrillation protocol compared with three stacked shock protocols. However, these studies included confounders related to pre-post design and the multiple interventions that were included as part of the defibrillation protocol. Another pre-post study, with fewer confounding factors, showed a significantly lower hands-off-ratio (ie, percentage of total CPR time when no compressions were provided) with the one-shock protocol but no statistical difference in survival.[2]

One observational study of fixed-dose biphasic defibrillation suggested higher defibrillation success with three shocks. The same paper also suggested that chest compressions immediately following a shock did not result in recurrence of VF. In contrast, another study showed earlier recurrence of VF when chest compressions were resumed immediately after the shock compared with delayed resumption of compressions. There was no difference in total incidence of recurrent VF or outcome. A single study demonstrated early termination of recurrent VF was associated with increased ROSC, but quality of CPR was poor and few patients achieved ROSC. Another study showed decreased survival when defibrillation for recurrent VF was, for a variety of reasons, delayed.[2]

One randomised controlled clinical trial has been published since 2010 comparing single versus stacked shocks and showed no difference in outcome.[7]

Priorities in resuscitation should include early assessment of the need for defibrillation, provision of CPR until a defibrillator is available, and minimization of interruptions in chest compressions. Rescuers can optimize the likelihood of defibrillation success by optimizing the performance of CPR, timing of shock delivery with respect to CPR, and the combination of waveform and energy levels. Rescuers can safely continue CPR while charging a manual defibrillator.[8]

Recommended shock protocol

It is recommended that a single shock strategy be used in patients in cardiac arrest requiring defibrillation for VF or pulseless VT [Class A; Expert consensus opinion]. When using this strategy, CPR should be resumed immediately following shock delivery and interruptions minimised [Class A; LOE IV].

CPR should be continued during charging of the defibrillator, and CPR should not be interrupted until rhythm reanalysis is undertaken [Class A; Expert consensus opinion].

9 Precautions

Be aware of electrical hazards in the presence of water, metal fixtures, oxygen and flammable substances. Warn of impending discharge by a "stand clear" command.

9.1 Oxygen and fire risk

Four case reports involving adults and one case report involving a neonate described fires caused by sparks generated during defibrillation attempts when paddles were used in the vicinity of high flow (>10 L/min) oxygen.[2]

In two manikin studies the oxygen concentration in the zone of defibrillation was not increased when ventilation devices (bag-valve device, self-inflating bag, Hamilton Viola ventilator) were left attached to a tracheal tube or when the oxygen source was vented at least 1 meter behind the patient's mouth. One study described higher oxygen concentrations and longer washout periods when oxygen is administered in confined spaces without adequate ventilation. There are no case reports of fires caused by sparking when shocks were delivered using adhesive pads.[6]

9.2 Recommended technique

Rescuers should take precautions to minimize sparking (by paying attention to pad/paddle placement, contact, etc) during attempted defibrillation. Rescuers should try to ensure that defibrillation is not attempted in an oxygen-enriched atmosphere (e.g. when high-flow oxygen is directed across the chest) [Class A; Expert consensus opinion].

Rescuers should minimise interruptions to CPR while defibrillating the patient. Rescuers should be able to safely charge a manual defibrillator during CPR when using pads. The defibrillator should be disarmed if a shock is not required [Class B; Expert consensus opinion]. Manual chest compressions should not continue during the delivery of a shock because safety has not been established.

Specifically, rescuers should:

- AVOID charging the paddles unless they are placed on the victim's chest
- AVOID placing the defibrillator paddles/pads over ECG electrodes (risk of burns or sparks), ECG leads (may melt), medication patches, an implanted device (e.g. a pacemaker), or a central line insertion site
- AVOID having, or allowing any person to have, any direct or indirect contact with the victim during defibrillation (a shock may be received)

- AVOID having the victim in contact with metal fixtures e.g. bed rails (risk of burn)
- AVOID delivering the shock with a gap between the paddle/pad and chest wall (spark hazard)
- AVOID defibrillating if victim, operator and/or close bystander are situated in an explosive/flammable (e.g. petrol) environment
- AVOID allowing oxygen from a resuscitator to flow onto the victim's chest during delivery of the shock when using paddles (risk of fire) [Class A; LOE IV].

10 Confirmation of Shock Delivery

Check that the victim has a muscle response to the shock and there is ECG (electrocardiogram) evidence of shock delivery. If it does not appear that the shock has been delivered, consider that the "synchronize" mode of the defibrillator may be on or there may be a flat battery, lead fracture, charge dump etc.

11 Failure of Defibrillation

If the attempt at defibrillation is unsuccessful:

- Recommence CPR with oxygen (follow algorithm in Guideline 11.2).
- Check paddle or electrode position.
- Check that there is adequate skin contact (clipping or shaving of body hair under the defibrillator paddle/pad may be required).
- Consider changing the defibrillator pads.

12 Use of Automated External Defibrillators (AEDs)

AED use should not be restricted to trained personnel. Allowing use of AEDs by individuals without prior formal training can be beneficial and may be life saving. Since even brief training improves performance (e.g. speed of use, correct pad placement), it is recommended that training in the use of AEDs be provided.

Implementation of AED programs in public settings should be based on the characteristics of published reports of successful programs in similar settings.[9] Services that implement the use of AEDs must be aware of the possible adverse impact of interruptions to CPR, especially in non-shockable rhythms.[4]

Home AED use, for high-risk individuals who do not have an ICD, is safe and feasible and may be considered on an individual basis, but has not been shown to change overall survival rates.[9]

Because population (e.g. rates of witnessed arrest) and program (e.g. response time) characteristics affect survival, when implementing an AED program, community and program leaders should consider factors such as location, development of a team with a responsibility for monitoring and maintaining the devices, training and retraining programs for those who are likely to use the AED, coordination with the local EMS, and identification of a group of paid or volunteer individuals who are committed to using the AED for victims of arrest.[9]

12.1 AEDs in manual mode

Modern defibrillators can be operated in both manual and semi-automatic modes. However, few studies compare these two options. One randomized controlled study showed no difference in survival to hospital discharge rate but significant reduction in time to first shock in the AED group versus the manual group (1.1 vs 2.0 minutes). One good concurrent controlled out-of-hospital cardiac arrest study in 36 rural communities showed no improvements in ROSC, survival and neurological outcome but significantly shorter times to first shock and higher VF conversion rates when paramedics used AEDs in semi-automatic mode compared with manual mode. One retrospective study demonstrated no improvement in survival to hospital discharge for in-hospital adult cardiac arrest when comparing AED with manual defibrillators.

In patients with initial asystole or pulseless electrical activity (PEA), AEDs were associated with a significantly lower survival (15%) compared with manual defibrillators (23%, $p = 0.04$).

In a study of three different EMS systems and one in-hospital center, the manual mode of defibrillation was associated with a lower total hands-off ratio (ie, percentage of total CPR time when no compressions were provided) than AED mode. However, more shocks were delivered inappropriately by rescuers using manual defibrillators (26% manual vs. 6% AEDs). A randomized manikin study simulating cardiac arrest showed a lower hands-off ratio, mainly due to a shorter pre-shock pause, when trained paramedics used the defibrillator in manual mode compared with semi-automatic mode. More inappropriate shocks (12% vs 0), were delivered in manual mode. All episodes of VF were detected and shocked appropriately. A shorter pre-shock pause and lower total hands-off-ratio increase vital organ perfusion and the probability of ROSC.[2]

There are no survival differences between defibrillation in semiautomatic and manual modes during in- and out-of-hospital resuscitation; however, the semi-automatic mode is preferred because it is easier to use and may deliver fewer inappropriate shocks. Trained personnel may deliver defibrillation in manual mode. Use of the manual mode enables chest compressions to be continued during charging, thereby minimizing the pre-shock pause. When using the defibrillator in manual mode, frequent team training and ECG recognition skills are essential.

The defibrillation mode that results in the best outcome will be influenced by the system, and provider skills, training and ECG recognition.[6]

In one in-hospital study, the use of AEDs was not associated with improved survival in those patients with shockable rhythms, but was associated with lower survival in those with non-shockable rhythms.[4]

Recommendation

The use of AEDs is reasonable to facilitate early defibrillation in hospitals [2], but services that introduce AEDs must be aware of the possible adverse impact of interruptions to CPR, especially in non-shockable rhythms [4] [Class B; LOE IV].

13 Use of the Defibrillator for Quality Assurance

13.1 Data collection

Collection of data from defibrillators enables a comparison of actual performance during cardiac arrests and training events. The results of many observational studies suggest that the rate and depth of external cardiac compressions and ventilation rate were at variance with current guidelines. Monitor/defibrillators modified to enable collection of data on compression rate and depth and ventilation rate may be useful for monitoring and improving process and outcomes after cardiac arrest.[2] However, rescuers should be aware of the potential overestimation of compression depth when the victim is on a soft surface.[10]

13.2 Waveform analysis

Retrospective analysis of the VF waveform analysis in multiple clinical and animal studies and theoretical models suggest that it is possible to predict the success of defibrillation from the fibrillation waveform with varying reliability. One animal study was neutral.

No human studies have specifically evaluated whether treatment altered by predicting success of defibrillation can improve successful defibrillation, ROSC or survival from cardiac arrest. Multiple waveform parameters have been examined without consensus on the most important parameters to predict outcome.[2]

There is insufficient evidence to support routine use of VF waveform analysis to guide defibrillation management in adult in hospital and out of hospital cardiac arrest.[2] There is insufficient evidence to support or refute the use of artefact filtering algorithms for analysis of ECG rhythm during CPR.[10]

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AUSTRALIAN RESUSCITATION COUNCIL

NEW ZEALAND Resuscitation Council WHAKAHAUORA AOTEAROA

ANZCOR Guideline 11.5 – Medications in Adult Cardiac Arrest

Summary

Who does this guideline apply to?

This guideline applies to adults who require advanced life support (ALS).

Who is the audience for this guideline?

This guideline is for health professionals and those who provide healthcare in environments where equipment and drugs are available.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) make the following recommendations:

1. Intravenous (IV) administration is the preferred means of administering medications to patients during or after cardiac arrest, followed by intraosseous (IO) access.
2. Given the observed benefit on short-term outcomes, standard does adrenaline (epinephrine) is administered to adult patients in cardiac arrest.
3. Vasopressin is not be added to standard dose adrenaline (epinephrine) during cardiac arrest.
4. Given the observed benefit on short-term outcomes, amiodarone is used in adult patients with refractory VF/pVT.
5. Other drugs, including calcium, lidocaine (lignocaine), magnesium (magnesium sulfate heptahydrate), potassium, sodium bicarbonate (and other buffers) may be considered to help manage particular conditions that are associated with patients who have arrested.
6. Fibrinolytics should not be used routinely in cardiac arrest, but may be used when pulmonary embolus is the suspected cause of cardiac arrest.

While the listed drugs have theoretical benefits in selected situations, no medication has been shown to improve long-term survival in humans after cardiac arrest. Priorities are defibrillation, oxygenation and ventilation together with external cardiac compression.

1 Administration

1.1 Intravenous (IV) route

Intravenous (IV) drug administration is preferable and IV access is quickly and most easily achieved via a peripheral cannula inserted into a large peripheral vein. If there are no visible peripheral veins, the external jugular vein should be considered. Lower limb veins should be avoided due to impairment of venous return below the diaphragm during cardiac arrest. Intravenous drug administration must be followed by a fluid flush of at least 20-30 mL and external cardiac compression. If a central line is present it should be used. Central access provides more rapid drug delivery but insertion of a new line may be difficult, takes time to establish and has major risks [Class A; Expert consensus opinion].

1.2 Intraosseous (IO) route

Intraosseous is the preferred route if intravenous access is not available. Two prospective trials in adults and children and 6 other studies documented that IO access is safe and effective for fluid resuscitation, drug delivery, and laboratory evaluation, and is attainable in all age groups. If IV access cannot be established, intraosseous (IO) delivery of resuscitation drugs will achieve adequate plasma concentrations.[1] A number of devices are now available for use in adults [2] [Class A; Expert consensus opinion].

1.3 Endotracheal route

If IV/IO access cannot be attained and an endotracheal tube is present, endotracheal administration of some medications is possible, although the absorption is variable and plasma concentrations are substantially lower than those achieved when the same drug is given by the intravenous route (increase in dose 3-10 times may be required). There are no benefits from endobronchial injection compared with injection of the drug directly into the tracheal tube. Dilution with water instead of 0.9% saline may achieve better drug absorption. Adrenaline (epinephrine), lidocaine (lignocaine) and atropine (atropine sulfate monohydrate) may be given via endotracheal tube, but other cardiac arrest drugs should NOT be given endotracheally as they may cause mucosal and alveolar damage.[1] This route cannot be used if a laryngeal mask airway is present [Class A; Expert consensus opinion].

1.4 Intracardiac injection

Intracardiac injection is **not** recommended because of the limited benefit and the high risk of complications.

2 Classes of Drugs and Order of Drug Administration

It is recognised that the vast majority of studies assessing the effects of drugs on survival have not been able to control for the quality of cardiopulmonary resuscitation. Furthermore, most drug evaluations to date have been conducted before recent advances in post-cardiac arrest care including Targeted Temperature Management. Since most drug trials have, at most, demonstrated only short-term outcome advantage it may be important to evaluate long-term outcome when these drugs are combined with optimized post-cardiac arrest care. One study compared the use of all drugs (adrenaline (epinephrine), amiodarone, atropine (atropine sulfate monohydrate), vasopressin), without isolating the effect of each individual drug alone, with placebo in adult out-of-hospital cardiopulmonary resuscitation and demonstrated improvement in return of spontaneous circulation and survival to hospital and intensive care unit admission, but no difference in survival to discharge or neurologic outcomes at discharge and at 1-year follow-up; however, this study was not powered to detect clinically meaningful differences in long-term outcome.[3]

There are no studies that addressed the order of drug administration.[4] There is inadequate evidence to define the optimal timing or order for drug administration. An incomplete review of animal studies suggests that timing of vasopressor administration may affect circulation and further investigations are important to help guide the timing of drug administration.[4]

There is some evidence from 8 observational studies (of cardiac arrest both in and out-of-hospital), to suggest that for cardiac arrest with an initial non-shockable rhythm, if adrenaline (epinephrine) is to be administered, it is given as soon as feasible after the onset of the arrest (CoSTR 2015 weak recommendation, low quality evidence).[5]

For cardiac arrest with an initial shockable rhythm, there is insufficient evidence to make a treatment suggestion regarding the timing of administration of adrenaline (epinephrine), particularly in relation to defibrillation, and the optimal timing may vary for different groups of patients and different circumstances.

2.1 Vasopressors

Despite the continued widespread use of adrenaline (epinephrine) and increased use of vasopressin during resuscitation in some countries, there is no placebo-controlled study that shows that the routine use of any vasopressor during human cardiac arrest increases survival to hospital discharge.[4] Although there is evidence that vasopressors (adrenaline (epinephrine) or vasopressin) may improve return of spontaneous circulation and short-term survival, there is insufficient evidence to suggest that vasopressors improve survival to discharge and neurologic outcome. There is insufficient evidence to suggest the optimal dosage of any vasopressor in the treatment of adult cardiac arrest. Given the observed benefit in short-term outcomes, ANZCOR suggest that standard dose adrenaline (epinephrine) is administered to adult patients in cardiac arrest (CoSTR 2015, weak recommendation, very low quality evidence). [5]

2.2 Other drugs

There is no convincing evidence that the routine use of other drugs (atropine (atropine sulfate monohydrate), amiodarone, lidocaine (lignocaine), procainamide, bretylium, magnesium (magnesium sulfate heptahydrate), buffers, calcium, hormones or fibrinolytics) during human CPR increases survival to hospital discharge.[4]

3 Specific Resuscitation Drugs

3.1 Adrenaline (Epinephrine)

This is a naturally occurring catecholamine with alpha and beta effects. It is administered in cardiac arrest to cause peripheral vasoconstriction via its alpha-adrenergic action (directing available cardiac output to myocardium and brain). It may facilitate defibrillation by improving myocardial blood flow during CPR.

One study retrospectively compared adrenaline (epinephrine) with no adrenaline (epinephrine) for sustained VF and PEA/asystole and found improved ROSC with adrenaline (epinephrine) for both rhythms but no difference in survival. In a large retrospective registry-based study from Sweden adrenaline (epinephrine) was an independent predictor of poor outcome.

Three randomised studies and a meta-analysis demonstrated no difference in outcomes (ROSC, survival to discharge, or neurologic outcome) with vasopressin when compared with adrenaline (epinephrine) as a first line vasopressor in cardiac arrest.

Two randomised studies demonstrated no difference in outcomes (ROSC, survival to discharge, or neurologic) comparing adrenaline (epinephrine) in combination with vasopressin with adrenaline (epinephrine) alone in cardiac arrest.

No study has demonstrated a long term survival benefit with high-dose versus standard-dose adrenaline (epinephrine) in cardiac arrest. Pooled data from 4 RCTs comparing standard dose adrenaline (epinephrine) (SD) with High Dose adrenaline (epinephrine) (HD) shows a survival to hospital admission advantage with HD (CoSTR 2015, weak recommendation, low quality evidence). [5]

Six randomised studies reported improvement in ROSC using high-dose adrenaline (epinephrine). One meta-analysis of pooled data from 5 studies supported improvement in ROSC with high-dose adrenaline (epinephrine) but no change in survival outcomes.[4]

Indications

There is insufficient evidence to suggest the optimal dosage of any vasopressor in the treatment of adult cardiac arrest. ANZCOR suggest that, given the observed benefit in short-term outcomes, standard does adrenaline (epinephrine) is administered to adult patients in cardiac arrest (CoSTR 2015 weak recommendation, very low quality evidence). [5]

Give for:

- Ventricular Fibrillation/pulseless Ventricular Tachycardia after initial counter shocks have failed (after 2nd shock then after every second loop)

- Asystole and electromechanical dissociation (pulseless electrical activity) in initial loop (then every second loop).

[Class A; Expert consensus opinion]

ANZCOR suggests that vasopressin should not be used instead of adrenaline (epinephrine) in cardiac arrest (CoSTR 2015 weak recommendation, low quality evidence). [5] However, if there are settings where vasopressin is already being used instead of adrenaline (epinephrine), this use may be continued (CoSTR 2015 weak recommendation, low quality evidence). [5]

ANZCOR suggest against adding vasopressin to standard dose adrenaline (epinephrine) during cardiac arrest (CoSTR 2015 weak recommendation, moderate quality evidence).[5]

Values and Preferences

There is no evidence to indicate that settings that use adrenaline (epinephrine) should switch to using vasopressin, but it is acknowledged that vasopressin is already used in some settings, and the available data do not indicate any reason to change this. There is no evidence to suggest that adding vasopressin to the use of adrenaline (epinephrine) results in patient benefit.

Adverse effects:

- Tachyarrhythmias
- Severe hypertension after resuscitation
- Tissue necrosis if extravasation occurs.

Dosage:

The initial adult dose is 1mg (1 mL of 1:1,000 or 10 mL of 1:10,000) and this should be repeated at regular intervals (every 2nd loop) during CPR. Higher doses of adrenaline (epinephrine) have not been shown to improve long-term outcome. Adrenaline (epinephrine) may be required in repeated small doses or by infusion to produce an adequate blood pressure after return of a patient generated pulse. In this situation adrenaline (epinephrine) by infusion (1-20 mcg/min) should be delivered by a dedicated central line as soon as possible.

3.2 Amiodarone

Amiodarone is an antiarrhythmic drug with complex pharmacokinetics and pharmacodynamics. It has effects on sodium, potassium and calcium channels as well as alpha and beta-adrenergic blocking properties.

One RCT has shown a higher rate of ROSC for amiodarone (after adrenaline (epinephrine)) compared with no drug.

Two randomized trials demonstrated the benefit of amiodarone over standard of care, which included lidocaine (lignocaine) in 80% of cases, or routine use of lidocaine (lignocaine) for

shock refractory or recurrent VT/VF for the endpoint of survival to hospital admission, but not to survival to hospital discharge.[4]

An additional nine studies document consistent improvement in defibrillation response when amiodarone is given to humans or animals with VF or hemodynamically unstable VT. There was little evidence to suggest a survival-to-discharge advantage with any antiarrhythmic drug used during resuscitation from out-of-hospital or in-hospital cardiac arrest.[4]

ANZCOR suggest the use of amiodarone in adult patients with refractory VF/pVT to improve rates of ROSC (CoSTR 2015, weak recommendation, moderate quality evidence). [5]

Give for: VF/pulseless VT (between the third and fourth shock, when refractory to defibrillator shocks and a vasopressor) [Class A; Expert consensus opinion].

Consider administration for:

- Prophylaxis of recurrent VF/VT.

Adverse effects:

- Hypotension
- Bradycardia
- Heart block.

Dosage:

Initial bolus dose is 300 mg. An additional dose of 150 mg could be considered. This may be followed by an infusion (i.e. 15 mg/kg over 24 hours).

3.3 Calcium

Calcium is essential for normal muscle and nerve activity. It transiently increases myocardial excitability and contractility and peripheral resistance.

Three randomized control trials and three cohort studies and one case series demonstrated no effect on survival when calcium was given to in-hospital or out-of-hospital cardiac arrest patients. Two adult studies suggest that calcium administration during cardiac arrest was associated with decreased survival to hospital discharge.[4] In VF, calcium did not restore a spontaneous circulation.

In one study of PEA arrests, calcium demonstrated improved ROSC, without reporting long-term survival, but only in a subgroup of patients with wide QRS. Another study showed improved ROSC and survival to hospital arrival; however, there was no significant effect on survival. Another study showed decreased rate of ROSC in the calcium group. In two studies of asystole calcium administration failed to show any improvement in ROSC or survival to hospital discharge. One study showed reduced ROSC in the calcium group.[4]

Routine administration of calcium for treatment of in-hospital and out of hospital cardiac arrest is not recommended [Class A; Expert consensus opinion].

Consider administration for:

- Hyperkalaemia
- Hypocalcaemia
- Overdose of calcium-channel blocking drugs.

Adverse effects:

- Possible increase in myocardial and cerebral injury by mediating cell death
- Tissue necrosis with extravasation.

Dosage:

The usual adult bolus dose in these settings is 5-10 mL of 10% calcium chloride (calcium chloride dihydrate) (10 mL 10% calcium chloride dihydrate = 6.8 mmol Ca ions = 360 mg elemental calcium). An alternative formulation is calcium gluconate (calcium gluconate monohydrate) (10 mL of 10% calcium gluconate (calcium gluconate monohydrate = 2.2 mmol Ca ions).

3.4 Lidocaine (lignocaine)

Lidocaine (lignocaine) acts as a sodium channel blocker.

Two randomized trials demonstrated the benefit of amiodarone over standard of care, which included lidocaine (lignocaine) in 80% of cases, or routine use of lidocaine (lignocaine) for shock refractory or recurrent VT/VF for the endpoint of survival to hospital admission, but not to survival to hospital discharge. A retrospective review demonstrated improved survival to admission with lidocaine (lignocaine) (compared with standard treatment) for patients in VF out of hospital.[6] There is inadequate evidence to support or refute the use of lidocaine (lignocaine) in VT/VF not terminated by defibrillation, or VT/VF recurrence in out-of-hospital cardiac arrest or in-hospital cardiac arrest.[4]

ANZCOR suggest that lidocaine (lignocaine) may be used as an alternative to amiodarone in patients with refractory VF/pVT (CoSTR 2015, weak recommendation, very low quality evidence).[5]

Consider administration for:

- VF/pulseless VT as an alternative to amiodarone
- Prophylaxis in the setting of recurrent VF or VT.

Adverse effects:

- Slurred speech, altered consciousness, muscle twitching, and seizures
- Hypotension, bradycardia, heart block and asystole.

Dosage:

Lidocaine (lignocaine) is given initially as a 1mg/kg bolus. During resuscitation an additional bolus of 0.5 mg/kg may be considered. It is not recommended to commence a lidocaine (lignocaine) infusion until return of spontaneous circulation.

3.5 Magnesium (magnesium sulfate heptahydrate)

Magnesium is an electrolyte essential for membrane stability. Hypomagnesaemia causes myocardial hyperexcitability particularly in the presence of hypokalaemia and digoxin. Four randomized controlled trials did not show any increase in ROSC or survival when magnesium was compared with placebo for patients in VF in the prehospital, intensive care unit and emergency department settings.[4]

ANZCOR suggest that magnesium (magnesium sulfate heptahydrate) should not be routinely used in adult cardiac arrest (CoSTR 2015 strong recommendation, low quality evidence).[5] Magnesium (magnesium sulfate heptahydrate) should be given for hypomagnesemia and torsades de pointes.

Consider administration for:

- Torsade de pointes
- Cardiac arrest associated with digoxin toxicity
- VF/pulseless VT (usually administered when refractory to defibrillator shocks and a vasopressor)
- Documented hypokalaemia
- Documented hypomagnesium.

[Class A; Expert consensus opinion]

Adverse effects:

- Excessive use may lead to muscle weakness and respiratory failure.

Dosage:

A bolus of 5 mmol of magnesium (magnesium sulfate heptahydrate), which may be repeated once and followed by an infusion of 20 mmol over four hours.

3.6 Potassium

Potassium is an electrolyte essential for membrane stability. Low serum potassium, especially in conjunction with digoxin therapy and hypomagnesaemia, may lead to life threatening ventricular arrhythmias.

Consider administration for:

- Persistent VF due to documented or suspected hypokalaemia.

[Class A; Expert consensus opinion]

Adverse effects:

- Inappropriate or excessive use will produce hyperkalaemia with bradycardia, hypotension and possible asystole
- Extravasation may lead to tissue necrosis.

Dosage:

A bolus of 5 mmol of potassium chloride is given intravenously.

3.7 Sodium Bicarbonate (and other buffers)

Sodium bicarbonate is an alkalising solution, which combines with hydrogen ions to form a weak carbonic acid. This breaks down to produce CO_2 and H_2O. In most cardiac arrests early efficient CPR and adequate ventilation negate the need for any $NaHCO_3$.

Two studies evaluated buffering agents during cardiopulmonary resuscitation. Both had limitations but showed no improvement in outcome. Two retrospective cohort studies also showed no benefit in the use of buffering agents during cardiopulmonary resuscitation. Two studies demonstrated increased return of spontaneous circulation, hospital admission and survival at hospital discharge with bicarbonate use. Four cohort studies reported that bicarbonate use was associated with poor short- and long-term outcome.[4]

Routine administration of sodium bicarbonate for treatment of in-hospital and out-of hospital cardiac arrest is not recommended. [Class A; Expert consensus opinion]

Consider administration for:

- Hyperkalaemia
- Treatment of documented metabolic acidosis
- Overdose with tricyclic antidepressants
- Protracted arrest (greater than 15 mins).

[Class A; Expert consensus opinion]

Adverse effects:

- Metabolic alkalosis, hypokalaemia, hypernatraemia and hyperosmolality
- Intra cellular acidosis may develop or worsen when the CO_2 liberated from $NaHCO_3$ freely enters the cells
- Sodium bicarbonate and adrenaline (epinephrine) or calcium when mixed together may inactivate each other, precipitate and block the IV line

Dosage:

1mmol/kg, is initially given over 2-3 minutes, then as guided by arterial blood gases.

3.8 Vasopressin

Vasopressin is commonly referred to as antidiuretic hormone. In high doses vasopressin acts as a nonadrenergic peripheral vasoconstrictor and therefore is an effective vasopressor.

Three randomized studies and a meta-analysis demonstrated no difference in outcomes (ROSC, survival to discharge, or neurologic outcome) with vasopressin when compared with adrenaline (epinephrine) as a first line vasopressor in cardiac arrest.[4]

Six RCTs have shown no improvement in outcomes (ROSC, survival to discharge, or neurologic) with the addition of vasopressin to adrenaline (epinephrine).[5]

There is insufficient evidence to suggest the optimal dosage of any vasopressor in the treatment of adult cardiac arrest.

ANZCOR suggest against using vasopressin instead of adrenaline (epinephrine) for cardiac arrest (CoSTR 2015, weak recommendation, moderate quality evidence). [5]

ANZCOR suggest against adding vasopressin to standard dose adrenaline (epinephrine) during cardiac arrest (CoSTR 2015 weak recommendation, low quality evidence). [5]

4 Other Drugs and Fluids

4.1 Aminophylline

One case series and 3 small randomized trials indicate that aminophylline does not increase ROSC when given for brady-asystolic cardiac arrest. No studies have shown an effect of aminophylline on rates of survival to hospital discharge. There is no evidence of harm from giving aminophylline in brady-asystolic cardiac arrest.[1]

4.2 Fluids

No published human study directly compared outcome of routine intravenous fluid administration with no fluid administration during CPR. Two animal studies report that normothermic fluid infusion during CPR cause a decrease in coronary perfusion pressure and another animal study shows that the coronary perfusion pressure rise with adrenaline (epinephrine) during CPR is not improved with the addition of a fluid infusion. Most animal studies of fluid infusion during CPR do not have a control group that receives no fluids to enable an assessment of benefit or harm from fluid therapy.[4] Hypertonic fluid: One small RCT in adults found no significant return of spontaneous circulation or survival benefit with hypertonic intravenous fluid infusion when compared to isotonic intravenous fluid infusion during CPR. One animal study shows that hypertonic saline improves cerebral blood flow during CPR. Two animal studies found neither benefit nor harm with infusion of hypertonic saline.[4]

Chilled Fluid vs. Room Temperature fluid: Two adult studies and two animal studies showed no improvement in return of spontaneous circulation when cold intravenous fluids (compared with room temperature intravenous fluids) are infused during CPR. One of the reported animal studies showed that the infusion of cold fluids during CPR caused a decrease in coronary perfusion pressure when compared to no fluids.[4]

There is insufficient evidence to recommend for or against the routine infusion of intravenous fluids during cardiac arrest resuscitation.[4]

Fluids should be infused if hypovolemia is suspected (hypovolemic shock would normally require the administration of at least 20 mL/kg) [Class A; Expert consensus opinion].

4.3 Steroids

For OHCA, one RCT and one non-RCT did not show benefit in survival with the addition of steroids during cardiac arrest. Additionally, the RCT did not show improvement in ROSC, but the non-RCT did.

For IHCA, two RCTs (from the same investigators) showed improved outcome (ROSC) with methylprednisolone, vasopressin, and adrenaline (epinephrine) during cardiac arrest, and improved outcomes (survival and neurology) with the addition of hydrocortisone to those with post-ROSC shock compared with only adrenaline (epinephrine) and placebo.

ANZCOR suggests against the routine use of steroids during CPR for OHCA (CoSTR 2015 weak recommendation, very low quality evidence).[5]

ANZCOR makes no recommendation either for or against the use of steroids for in-hospital cardiac arrest.

Values and Preferences

For IHCA, it is acknowledged that there are no studies assessing the effect of the addition of steroids alone to standard treatment for IHCA. Also, although the triple-agent drug regimen used (methylprednisolone, vasopressin and adrenaline (epinephrine)) appears to suggest an association with improved outcome, the population studied had very rapid advanced life support, a high incidence of asystolic cardiac arrest, and low baseline survival compared to other IHCA studies, so some of the observed effects might be peculiar to the population studied.

4.4 Thrombolytics

Two randomised studies failed to show any improvement in short or long term outcomes with the use of fibrinolytics. One study showed an increased risk of intracranial bleeding associated with the routine use of fibrinolytics during cardiac arrest. Seven studies showed benefit from fibrinolytic therapy in the treatment of victims of cardiopulmonary arrest unresponsive to standard therapy; however, these studies had significant limitations.[4] Routine administration of fibrinolytics for the treatment of in-hospital and out-of hospital cardiac arrest is not recommended [Class A; Expert consensus opinion].

Fibrinolysis should be considered in adult patients with cardiac arrest with proven or suspected pulmonary embolism (CoSTR 2015, weak recommendation, very low quality evidence).[5] If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60–90 min before termination of resuscitation attempts [Class A; Expert consensus opinion].

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ANZCOR Guideline 11.6 – Equipment and Techniques in Adult Advanced Life Support

Summary

Who does this guideline apply to?

This guideline applies to adults who require advanced life support (ALS).

Who is the audience for this guideline?

This guideline is for health professionals and those who provide healthcare in environments where equipment and drugs are available.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) make the following recommendations:

1. The highest possible inspired oxygen concentration is used on all patients during cardiopulmonary resuscitation (CPR). Oxygen should never be withheld because of the fear of adverse effects.
2. Either an advanced airway or a bag-mask device may be used for airway management during CPR for cardiac arrest in any setting.
3. Waveform capnography should be used to confirm and continuously monitor the position of a tracheal tube during CPR in addition to clinical assessment.
4. Either a supraglottic airway or tracheal tube may be used as the initial advanced airway during CPR for cardiac arrest in any setting.
5. When ventilating a victim without an advanced airway, ventilation should be continued at a ratio of 30 compressions to 2 ventilations.
6. CPR prompt / feedback devices may be considered for clinical use to provide data as part of an overall strategy to improve quality of CPR at a systems level.
7. $ETCO_2$ cut-off values alone should not be used as a mortality predictor or for the decision to stop a resuscitation attempt.
8. If cardiac ultrasound is available and can be performed without interfering with standard ACLS, it may be considered to try and identify potentially reversible causes of cardiac arrest.
9. An ITD should not be routinely used in addition to standard CPR.

10. Automated mechanical chest compression devices should not be routinely used to replace manual chest compressions.
    However, they may be a reasonable alternative to high-quality manual chest compressions in situations where sustained high-quality manual chest compressions are impractical or compromise provider safety.
11. eCPR is a reasonable rescue therapy for selected patients with cardiac arrest when initial standard CPR is failing in settings where this can be implemented.

A wide range of equipment is available for use in ALS. The role of such equipment should be subject to constant evaluation. The use of any item of equipment requires that the operator is appropriately trained and maintains competency in its use. Frequent retraining (theory and practice) is required to maintain both Basic Life Support (BLS) and ALS skills. The optimal interval for retraining has not been established.

Airway adjuncts can be used to facilitate ventilation, to better maintain the airway, or to provide access to the airway (e.g. for suctioning) [Class B; Expert consensus opinion].

1 Oxygen during CPR

There are no adult human studies that directly compare maximal inspired oxygen with any other inspired oxygen concentration. In one observational study of patients receiving 100% oxygen and tracheal intubation during CPR, a higher measured PaO_2 during CPR was associated with improved return of spontaneous circulation (ROSC) and hospital admission.[1]

ANZCOR suggests that the highest possible inspired oxygen concentration is used on all patients during CPR (CoSTR 2015 weak recommendation, very low quality evidence)[2]. Oxygen should never be withheld because of the fear of adverse effects.

There is insufficient evidence to support or refute the use of passive oxygen delivery during compression only CPR to improve outcomes (ROSC, hospital discharge rate and improve neurological survival) when compared with oxygen delivery by positive pressure ventilation.

2 Airway

2.1 Airway manoeuvres

The BLS techniques of chin lift and head tilt are covered in Guideline 4.

Jaw thrust

In this technique, the rescuer is commonly positioned at the top of the victim's head, although a jaw thrust may be applied from the side or in front. The jaw is clasped with both hands and the mouth is held open by the thumbs.

Pressure is applied with the index (or middle) fingers behind the angles of the jaw. The jaw is gently thrust upwards and away from the chest, moving the tongue away from the back of the throat. Gentle head tilt may also be necessary to maintain airway patency with this technique.

A jaw thrust may be required in the recovery position if the victim's airway is not patent [Class A; Expert consensus opinion].

2.2 Basic airway adjuncts

Oro- and nasopharyngeal airways have long been used in cardiac arrest, despite never being studied in this clinical context. It is reasonable to continue to use oral and naso-pharyngeal airways when performing bag-mask ventilation in cardiac arrest, but in the presence of a known or suspected basal skull fracture an oral airway is preferred. It is still necessary to use head tilt and jaw support, or jaw thrust [Class B; Expert consensus opinion].

Oropharyngeal airway

Oral airways should be appropriately sized and not be forcibly inserted. They should be reserved for unconscious, obtunded victims. Laryngospasm or vomiting with aspiration may result in those patients who still have a gag reflex [Class B; Expert consensus opinion].

Nasopharyngeal airway

Despite frequent successful use of nasopharyngeal airways by anaesthetists, there are no published data on the use of these airway adjuncts during CPR. One study in anesthetised patients showed that nurses inserting nasopharyngeal airways were no more likely than anaesthetists to cause nasopharyngeal trauma. One study showed that the traditional methods of sizing a nasopharyngeal airway (measurement against the patient's little finger or anterior nares) do not correlate with the airway anatomy and are unreliable. In one report insertion of a nasopharyngeal airway caused some airway bleeding in 30% of cases. Two case reports involve inadvertent intracranial placement of a nasopharyngeal airway in patients with basal skull fractures. In the presence of a known or suspected basal skull fracture, an oral airway is preferred, but if this is not possible and the airway is obstructed, gentle insertion of a nasopharyngeal airway may be lifesaving (ie. the benefits may far outweigh the risks)[3] [Class B; Expert consensus opinion].

2.3 Advanced airway devices

The endotracheal tube has generally been considered the optimal method of managing the airway during cardiac arrest. There is evidence that without adequate training and experience, the incidence of complications, such as unrecognized oesophageal intubation, is unacceptably high. Alternatives to the tracheal tube that have been studied during CPR include the bag-valve mask device and advanced airway devices such as the laryngeal mask airway (LMA), i-gel, laryngeal tube, and oesophageal-tracheal combitube (Combitube).

There is insufficient data to support the routine use of any specific approach to airway management during cardiac arrest.

ANZCOR suggests using either an advanced airway or a bag-mask device for airway management during CPR for cardiac arrest in any setting (CoSTR 2015, weak recommendation, very-low-quality evidence).[2]

The choice of airway used should depend on the skills and training of the healthcare provider. Tracheal intubation may result in increased hands-off time in comparison with insertion of a supraglottic airway (e.g. LMA, laryngeal tube) or a bag-mask device. Both a bag-mask device and an advanced airway are frequently used in the same patient as part of a stepwise approach to airway management, but this has not been formally assessed.[2]

There is inadequate evidence to define the optimal timing of advanced airway placement during cardiac arrest.

The airway devices/adjuncts used during a cardiac arrest must be chosen according to local training and availability [Class A; Expert consensus opinion]. To avoid substantial interruptions in chest compressions providers may defer attempts to insert devices/adjuncts until return of spontaneous circulation (ROSC) [Class B; Expert consensus opinion].

2.4 Endotracheal intubation

The only published randomised controlled trial that compared tracheal intubation with BVM ventilation was performed in children who required airway management out-of-hospital. In this study there was no difference in survival-to-discharge rates but it is unclear how applicable this paediatric study is to adult resuscitation.[4] The study had some important limitations, including the provision of only 6 hours of additional training for intubation, limited opportunity to perform intubations, and short transport times. Two studies compared outcomes from out-of-hospital cardiac arrest in adults treated by either emergency medical technicians or paramedics. The skills provided by the paramedics, including intubation and intravenous (IV) cannulation and drug administration, made no difference in survival to hospital discharge.

The reported incidence of unrecognised oesophageal intubation in cardiac arrest ranges from 0-14% with a mean of 4.3%.[2] An additional problem common to any advanced airway is that intubation attempts generally require interruptions in chest compressions. Rescuers must weigh the risks and benefits of intubation versus the need to provide effective chest compressions. The intubation attempt will require interruption of chest compressions, but once an advanced airway is in place ventilation will not require interruption or even pausing of chest compressions.

To avoid substantial interruptions in chest compressions providers may defer an intubation attempt until return of spontaneous circulation (ROSC) [Class B; Expert consensus opinion]. To ensure competence, healthcare systems that utilise advanced airways should address factors such as adequacy of training and experience, and quality assurance. Providers must confirm tube placement and ensure that the tube is adequately secured [Class A; Expert consensus opinion].

In addition to providing optimal isolation and patency of the airway, intubation allows ventilation with 100% oxygen and suctioning of the airway and also provides possible access for the delivery of some drugs. However, if endotracheal intubation is attempted, ongoing CPR must be maintained, laryngoscopy should be performed during chest compressions and attempts at intubation should not interrupt cardiac compressions for more than 5 seconds [Class A; Expert consensus opinion].

Once an endotracheal tube has been passed:

- Inflate cuff with enough air to prevent a leak
- Confirm placement by assessing chest inflation, auscultation, by direct observation, and waveform capnography. Then, firmly secure the tube.

Confirmation of placement of endotracheal tube

Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation. Routine confirmation of correct placement of the tracheal tube should reduce this risk.

Two studies of waveform capnography to verify tracheal tube position in victims of cardiac arrest after intubation demonstrated 100% sensitivity and 100% specificity in identifying correct tracheal tube placement. One of these studies included 246 intubations in cardiac arrest with 9 oesophageal intubations and the other included 51 cardiac arrests with an overall oesophageal intubation rate of 23% but it is not specified how many of these occurred in the cardiac arrest group.

Three studies with a cumulative total of 194 tracheal and 22 oesophageal tube placements demonstrated an overall 64% sensitivity and 100% specificity in identifying correct tracheal tube placement when using the same model capnometer (no waveform capnography) on prehospital cardiac arrest victims. The sensitivity may have been adversely affected by the prolonged resuscitation times and very prolonged transport times of many of the cardiac arrest victims studied. Intubation was performed after arrival at hospital and time to intubation averaged more than 30 minutes.

Studies of colorimetric $ETCO_2$ detectors, the syringe aspiration oesophageal detector device the self-inflating bulb oesophageal detector device and non-waveform End Tidal CO_2 capnometers show that the accuracy of these devices is similar to the accuracy of clinical assessment for confirming the tracheal position of a tracheal tube in victims of cardiac arrest.

ANZCOR recommends using waveform capnography to confirm and continuously monitor the position of a tracheal tube during CPR in addition to clinical assessment (CoSTR 2015, strong recommendation, low-quality evidence).[2] See also Guideline 11.1.1

It is also recommended that if waveform capnography is not available, a non-waveform carbon dioxide detector, esophageal detector device or ultrasound, in addition to clinical assessment, are alternatives (CoSTR 2015, strong recommendation, low quality evidence). [2]

Values and Preferences

These are strong recommendations despite the low quality evidence, as a high value is placed on avoiding unrecognised oesophageal intubation. In 11 studies assessed, the mean incidence of unrecognised oesophageal intubation in cardiac arrest was 4.3% (range 0–14%).[2]

Additionally, waveform capnography is recommended as it may have other potential uses during CPR (e.g. monitoring ventilation rate, assessing quality of CPR, and alerting the presence of ROSC).

Alternatives to endotracheal intubation

Supraglottic airway (SGA) devices (e.g. LMA, Laryngeal tube, i-gel, Combitube) are generally considered easier to insert than tracheal tubes. They can be inserted without interrupting chest compressions, and their use in cardiac arrest has been increasing. Ten studies have compared a variety of SGA devices with the tracheal tube during out of hospital cardiac arrest.[2]

No studies comparing alternative advanced airway devices and tracheal intubation have been of a high quality and adequately powered to study long term survival. Studies comparing supraglottic airway to tracheal intubation have generally compared insertion time and ventilation success rates. There is insufficient data to support the routine use of any specific approach to airway management during cardiac arrest.[2]

ANZCOR suggests using either a supraglottic airway or tracheal tube as the initial advanced airway during CPR for cardiac arrest in any setting. Supraglottic airways are also a backup or rescue airway in a difficult or failed tracheal intubation (CoSTR 2015, weak recommendation, very-low-quality evidence).[2]

Values and Preferences

In the absence of sufficient data obtained from studies of IHCA, it is necessary to extrapolate from data derived from OHCA. The type of airway used should depend on the skills and training of the healthcare provider. Tracheal intubation requires considerably more training and practice. Attempted tracheal intubation may result in unrecognised oesophageal intubation and increased hands-off time in comparison with insertion of an SGA. Both an SGA and tracheal tube are frequently used in the same patients as part of a stepwise approach to airway management.[2]

3 Ventilation

3.1 Bag-Valve-Mask Device

Where difficulty with bag-mask-valve resuscitation is experienced, two trained operators may be required i.e. the first to manage the airway and the second to operate the bag [Class B; Expert consensus opinion].

3.2 Oxygen-Powered Resuscitators

These devices have a limited place but can provide high oxygen concentrations in experienced hands [Class B; Expert consensus opinion]. Devices that do not comply with current Australasian Standards should not be used.

3.3 Mechanical Ventilators

One pseudo-randomised study suggests that use of an automatic transport ventilator with intubated patients may enable the EMS team to perform more tasks while subjectively providing similar ventilation to that of a bag-valve device.[5]

One study suggests that use of an automatic transport ventilator with intubated patients provides similar oxygenation and ventilation as use of a bag-valve device with no difference in survival.[6]

ANZCOR considers that there is insufficient evidence to support or refute the use of an automatic transport ventilator over manual ventilation during resuscitation of the cardiac arrest victim with an advanced airway.

Both manual ventilation and mechanical ventilation have advantages and disadvantages in the initial management of cardiac arrests. These relate largely to the risks of hyperventilation (with manual ventilation), and hypoventilation (with mechanical breaths not being delivered). Irrespective of the mode of delivery of breaths, the adequacy of delivery of those delivered breaths should be regularly assessed [Class B; Expert consensus opinion].

3.4 Hyperventilation may be harmful

Reports containing both a small case series and an animal study showed that hyperventilation is associated with increased intrathoracic pressure, decreased coronary and cerebral perfusion, and, in animals, decreased return of spontaneous circulation (ROSC). In a secondary analysis of the case series that included patients with advanced airways in place after out-of-hospital cardiac arrest, ventilation rates of >10 per minute and inspiration times >1 second were associated with no survival. Extrapolation from an animal model of severe shock suggests that a ventilation rate of 6 ventilations per minute is associated with adequate oxygenation and better haemodynamics than ≥12 ventilations per minute [8] [Class B; LOE IV].

3.5 Inadvertent gas trapping

Eighteen articles involving 31 cases reported unexpected return of circulation (and in some cases prolonged neurologically intact survival) after cessation of resuscitation attempts. One case series suggested that this occurred in patients with obstructive airway disease. Four studies reported unexpected return of circulation in 6 cases in which resuscitation had ceased and ventilation was shown on repeated occasions (or was highly likely) to result in gas trapping and consequent hemodynamic compromise. The authors of all these studies suggested that a period of disconnection from ventilation during resuscitation from PEA may be useful to exclude gas trapping [7] [Class B; LOE IV].

Recommendation for frequency of ventilation

When ventilating a victim without an advanced airway, ventilation should be continued at a ratio of 30 compressions to 2 ventilations, irrespective of the number of rescuers, until an advanced airway is in place.

After an advanced airway (e.g. tracheal tube, LMA, Combitube,) is placed, ventilate the patient's lungs with supplementary oxygen to make the chest rise. During CPR for a patient with an advanced airway in place it is reasonable to ventilate the lungs at a rate of 6 to 10 ventilations per minute without pausing during chest compressions to deliver ventilations. (CoSTR 2015, weak recommendation , very low quality evidence). [2]

Simultaneous ventilation and compression may adversely effect coronary perfusion[9] and has been associated with decreased survival.[10] One starting point to provide consistent ventilation and an adequate minute volume while minimising interruptions to CPR, and minimising the likelihood of excessive ventilation, is to provide one breath after each 15 compressions (delivering the breath during the relaxation phase of compression, without a significant pause)[11] [Class B; Expert Consensus Opinion]. See also Guideline 11.1.1

The adequacy of ventilation with supraglottic airway devices during uninterrupted chest compressions is however unknown. Theoretically, a compression to ventilation ratio of 30:2 may be continued in patients with an advanced airway (ETT, LMA and other supraglottic airways).

This has advantages for simplicity of teaching, allows intermittent assessment of adequacy of ventilation, and also overcomes the problems associated with inefficient ventilation if breaths are delivered at the same time as the peak of the compressions [Class B; Expert consensus opinion].

Use the same initial tidal volume and rate in patients regardless of the cause of the cardiac arrest. Carbon dioxide estimation via arterial blood gas analysis and capnography may assist with monitoring ventilation and assessing quality of CPR, though these are more reliable once ROSC has been achieved [Class B; Expert consensus opinion].

3.6 Monitoring of ventilation

There is insufficient evidence to support or refute the use of peak pressure and minute ventilation monitoring to improve outcome from cardiac arrest. There is indirect evidence that monitoring the respiratory rate with real time feedback is effective in avoiding hyperventilation and achieving ventilation rates closer to recommended values, but there is no evidence that ROSC or survival is improved.[3]

4 Circulation

Healthcare providers should perform chest compressions for adults at a rate of approximately 100-120 compressions per minute **(**CoSTR 2015, strong recommendation, very low-quality evidence)[2] and to compress the lower half of the sternum by approximately 5 cm (approximately 1/3 of the antero-posterior diameter of the chest) (CoSTR 2015, strong recommendation, low-quality evidence).[2] Rescuers should allow complete recoil of the chest after each compression.

When feasible, rescuers should frequently alternate "compressor" duties (i.e. every 2 minutes), regardless of whether they feel fatigued, to ensure that fatigue does not interfere with delivery of adequate chest compressions. It is reasonable to use a duty cycle (i.e. ratio between compression and release) of 50% [Class A; Expert consensus opinion]. CPR with the patient in a prone position is a reasonable alternative for intubated hospitalised patients who cannot be placed in the supine position [Class B; LOE Expert consensus opinion].

Rescuers should minimise interruptions of chest compressions. It is reasonable for instructors, trainees and providers to monitor and improve the process of CPR to ensure adherence to recommended compression and ventilation rates and depths [Class B; LOE III-2]. See also Guideline 11.1.1.

4.1 CPR prompt or feedback devices [2,12,13]

Evidence from 22 manikin studies consistently demonstrated that CPR prompt/feedback devices used during CPR improved the quality of CPR performance on manikins. Three additional manikin studies examined the utility of video/animations on mobile phone devices: two studies showed improved checklist scores and quality of CPR and faster initiation of CPR while the third study showed that participants using multi-media phone CPR instruction took longer to complete tasks than dispatcher-assisted CPR. Two manikin studies that used two-way video communication to enable the dispatcher to review and comment on CPR in real time produced equivocal findings.

There is no high level evidence that the use of CPR feedback devices during real time CPR improves survival or return of spontaneous circulation (2015 CoSTR, weak recommendation, very low quality evidence).[2] One study each in children and adults showed that metronomes improved chest compression rate and increased end-tidal carbon dioxide. Five studies evaluating the introduction of CPR prompt/feedback devices in clinical practice (pre/post comparisons) found improved CPR performance.

There may be some limitations to the use of CPR prompt/feedback devices. Two manikin studies report that chest compression devices may overestimate compression depth if CPR is being performed on a compressible surface such as a mattress on a bed.[14] One study reported harm to a single participant when a hand got stuck in moving parts of the CPR feedback device. A further manikin study demonstrated that additional mechanical work is required from the CPR provider to compress the spring in one of the pressure sensing feedback devices. One case report documented soft tissue injury to a patient's chest when an accelerometer device was used for prolonged CPR. Instructors and rescuers should be made aware that a compressible support surface (e.g. mattress) may cause a feedback device to overestimate depth of compression.[14]

Recommendations

CPR prompt / feedback devices may be considered for clinical use to provide data as part of an overall strategy to improve quality of CPR at a systems level (CoSTR 2015, weak recommendation, very low quality evidence).[2]

ANZCOR places a higher value on resource allocation and cost effectiveness than widespread implementation of a technology with uncertain effectiveness during real time CPR. We acknowledge that data provided by CPR feedback devices may benefit other victims as part of a broader quality improvement system (2015 CoSTR, Values and Preferences Statement). [2]

4.2 Pacing

Four studies addressed the efficacy of pacing in cardiac arrest. These studies found no benefit from routine pacing in cardiac arrest patients. Use of pacing (transcutaneous, transvenous, needle) in cardiac arrest (in-hospital or out-of-hospital) did not improve ROSC or survival. There was no apparent benefit related to the time at which pacing was initiated (early or delayed in established asystole), location of arrest (in-hospital or out-of-hospital), or primary cardiac rhythm (asystole, PEA). Five case series, a review with two additional case reports, and a moderate sized case series, support percussion pacing in p-wave asystolic cardiac arrest/complete heart block or hemodynamically unstable patients with bradycardia. In these reports, sinus rhythm with a pulse was restored using different pacing techniques.

Electrical pacing is not effective as routine treatment in patients with asystolic cardiac arrest.[3]

The routine use of pacing (electrical or fist) is not recommended.

The use of pacing after cardiac surgery is considered in Guideline 11.10, 'Resuscitation in Special Circumstances'.

5 Monitoring during CPR

5.1 Waveform capnography (End-tidal carbon dioxide [$ETCO_2$])

Waveform capnography during CPR has potential roles in:

- Confirming tracheal tube placement
- Monitoring the ventilation rate to assist in avoiding hyperventilation
- Assessing the quality of chest compressions during CPR (CO_2 values are associated with compression depth and ventilation rate)
- Identifying ROSC during CPR (by an increased CO_2 value)
- Assessing prognosis during CPR (low CO_2 values may indicate a poor prognosis and less chance of ROSC). Failure to achieve a CO_2 value >10 mmHg after 20 min of CPR is associated with a poor outcome in observational studies.

Recommendations

ANZCOR recommends against using $ETCO_2$ cut-off values *alone* as a mortality predictor, or for the decision to stop a resuscitation attempt (CoSTR 2015, strong recommendation, low-quality evidence).[2]

ANZCOR suggests that an $ETCO_2$ 10 mm Hg or greater measured after tracheal intubation or after 20 min of resuscitation, may be a predictor of ROSC (CoSTR 2015, weak recommendation, low-quality evidence).[2]

ANZCOR suggests that an $ETCO_2$ of 10 mm Hg or greater measured after tracheal intubation, or an $ETCO_2$ 20 mm Hg or greater measured after 20 min of resuscitation may be a predictor of survival to discharge (CoSTR 2015, weak recommendation, moderate-quality evidence).[2]

Values and Preferences

ANZCOR has put a higher value on not relying on a single variable ($ETCO_2$) and cut-off value when their usefulness in actual clinical practice, and variability according to the underlying cause of cardiac arrest, has not been established. The aetiology (e.g. asphyxia, PE) of cardiac arrest could affect $ETCO_2$ values, and there is concern about the accuracy of $ETCO_2$ values during CPR.[2]

5.2 Arterial Blood Gas

There is evidence from 11 studies that arterial blood gas values are an inaccurate indicator of the magnitude of tissue acidosis during cardiac arrest and CPR in both the in-hospital and out-of-hospital settings. The same studies indicate that both arterial and mixed venous blood gases are required to establish the degree of acidosis.[7]

Arterial blood gas analysis alone can disclose the degree of hypoxemia and highlight the extent of metabolic acidosis. Arterial CO_2 is an indicator of adequacy of ventilation during CPR. If ventilation is constant an increase in $PaCO_2$ is a potential marker of improved perfusion during CPR.

Arterial blood gas monitoring during cardiac arrest enables estimation of the degree of hypoxemia and the adequacy of ventilation during CPR, but should not interfere with overall performance of good CPR [7] [Class B; LOE II and IV].

5.3 Ultrasound during cardiac arrest

The use of cardiac ultrasound during cardiac arrest may allow identification of many cardiac and non-cardiac causes of cardiac arrest, and three studies have examined the prognostic value of the presence or absence of sonographic cardiac motion in cardiac arrest.

Absence of cardiac motion on sonography during resuscitation of patients in cardiac arrest was highly predictive of death. [3]

One RCT compared the use of cardiac ultrasound during ALS to no use of cardiac ultrasound in adult patients with PEA arrest. This study enrolled 100 patients in a convenience sample and reported return of spontaneous circulation (ROSC) for at least 10 seconds in 34% of patients in the ultrasound group versus 28% in the group with no ultrasound (p=0.52).

Recommendation

If cardiac ultrasound is available and can be performed without interfering with standard ALS, it may be considered to try and identify potentially reversible causes of cardiac arrest (CoSTR 2015, weak recommendation, very low quality evidence).[2]

5.4 Other techniques and devices for circulatory support during CPR

Several techniques or adjuncts to standard CPR have been investigated and the relevant data was reviewed extensively as part of the 2010 ILCOR Consensus on Science process.[15] The success of any technique depends on the education and training of the rescuers and/or the resources available (including personnel). Techniques reviewed include: Open-chest CPR, Interposed Abdominal Compression CPR, Active Compression-Decompression CPR, Open Chest CPR, Load Distributing Band CPR, Mechanical (Piston) CPR, Lund University Cardiac Arrest System CPR, Impedance Threshold Device, and Extracorporeal Techniques.[15]

Because information about these techniques and devices is often limited, conflicting, or supportive only for short-term outcomes, no recommendations can be made to support or refute their routine use.

While no circulatory adjunct is currently recommended instead of manual CPR for routine use, some circulatory adjuncts are being routinely used in both out-of-hospital and in-hospital resuscitation. If a circulatory adjunct is used, rescuers should be well-trained and a program of continuous surveillance should be in place to ensure that use of the adjunct does not adversely affect survival [Class B; LOE IV].

New evidence for specific techniques to assist circulation during CPR was reviewed in the 2015 ILCOR Consensus on Science process.[2]

Three technologies for which there have been significant developments since 2010 have been considered:

(1) The impedance threshold device (ITD)

(2) Automated mechanical chest compression devices

(3) Extracorporeal CPR (eCPR).

(1) The impedance threshold device (ITD)

For standard CPR, 1 RCT showed no clinically significant benefit in survival from the addition of the ITD.

ANZCOR recommends against the routine use of the ITD in addition to standard CPR (CoSTR 2015, strong recommendation, high quality of evidence). [2]

For Active Compression CPR, 2 RCTs showed no clinically significant benefit in survival from the addition of the ITD to ACD CPR in a total of 421 out-of-hospital cardiac arrests.

Additionally, 2 RCTs did not demonstrate a clinically significant benefit in survival or neurological status from the addition of the ITD to ACD CPR compared with standard CPR.

(2) Automated mechanical chest compression devices (ACTs)

Two RCTs demonstrated no improvement in survival or neurological outcome at 30, 180 days or 1 yr compared with manual CPR. Three RCTs showed variable survival with good neurology at hospital discharge. Of two studies using the load-distributing band one study, showed harm, while the other showed no effect, and one study using the Lund University Cardiac Arrest System (LUCAS) device showed no effect. Five RCTs showed variable results for survival to hospital discharge. One RCT of IHCA showed benefit with use of a piston device compared with manual chest compressions. Two other RCTs of the LUCAS and 1 using a load-distributing band device showed neither benefit nor harm. Seven RCTs looked at the effect of ACDs on establishing ROSC: 2 showed a benefit, 1 showed harm and four showed no effect.

ANZCOR suggests against the routine use of automated mechanical chest compression devices to replace manual chest compressions (CoSTR 2015 weak recommendation, moderate quality of evidence).[2]

ANZCOR suggests that automated mechanical chest compression devices are a reasonable alternative to high-quality manual chest compressions in situations where sustained high-quality manual chest compressions are impractical or compromise provider safety (CoSTR 2015, weak recommendation, low quality evidence).[2]

Values and Preferences

ANZCOR believes the emphasis in resuscitation should be on providing high-quality chest compressions with adequate depth, rate and minimal interruptions, regardless of whether they are delivered by machine or human. We acknowledge that application of a mechanical chest compression device without a focus on minimising interruptions in compressions and delay to defibrillation could cause harm.

However, we also acknowledge that 1 large RCT showed equivalence between very high-quality manual chest compressions and mechanical chest compressions delivered with a load-distributing band in a setting with rigorous training and CPR quality monitoring, and we recognise that there are situations where sustained high-quality manual chest compressions may not be practical. Examples include CPR in a moving ambulance, the need for prolonged CPR (eg, hypothermic arrest), and CPR during certain procedures (eg. coronary angiography or preparation for extracorporeal CPR).

(3) Extracorporeal CPR (eCPR)

For IHCA, two observational studies demonstrated improved neurological survival at 180 days, but no difference at 1 year. These studies also showed improved survival at 30 and 180 days, but not at 1 year, and improved outcome (in both survival and neurology) at hospital discharge.

For OHCA, 1 observational study showed improved functional survival with eCPR at 30 and 180 days, and another at 90 days. One of these studies also showed improved survival to hospital discharge, though not in propensity matched samples.

ANZCOR suggests eCPR is a reasonable rescue therapy for selected patients with cardiac arrest when initial standard CPR is failing in settings where this can be implemented (CoSTR 2015 weak recommendation, very low quality of evidence).[2]

Values and Preferences

ANZCOR acknowledges that the published series used selected patients for eCPR and that guidelines for clinical practice should apply to similar populations. We recognise that eCPR is a complex intervention that is not universally available, but we consider that it may be successful in individuals where usual CPR techniques have failed and may also buy time for another treatment such as coronary angiography or percutaneous coronary intervention (PCI).

5.5 Open Chest CPR

There are no published randomised controlled trials and very limited data in humans comparing open-chest CPR to standard CPR in cardiac arrest. Four relevant human studies, 2 after cardiac surgery and 2 after out-of-hospital cardiac arrest, showed that open-chest cardiac massage improved coronary perfusion pressure and increased ROSC. Evidence from animal studies indicates that open-chest CPR produces greater survival rates, perfusion pressures, and organ blood flow than closed-chest CPR. Open-chest CPR should be considered for patients with cardiac arrest in the early postoperative phase after cardiothoracic surgery or when the chest or abdomen is already open. Open chest CPR should also be considered after penetrating chest injures [15] [Class B; LOE III-2].

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AUSTRALIAN RESUSCITATION COUNCIL

NEW ZEALAND Resuscitation Council WHAKAHAUORA AOTEAROA

ANZCOR Guideline 11.6.1 – Targeted Oxygen Therapy in Adult Advanced Life Support

Summary

This guideline provides advice on the administration of oxygen in the peri-arrest period.

Who does this guideline apply to?

This guideline applies to adults who require advanced life support.

Who is the audience for this guideline?

This guideline is for health professionals and those who provide healthcare in environments where equipment and drugs are available.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) make the following recommendations:

1. Oxygen therapy should only be used by personnel trained in its use, and its effects should be monitored whenever possible, usually by the use of pulse oximetry.
2. In patients requiring advanced life support, oxygen should be administered if the oxygen saturation (SpO_2) falls below 94% unless contraindications exist.
3. ANZCOR recommends the use of 100% oxygen during adult cardiac arrest.
4. ANZCOR recommends avoiding hypoxia in adults with ROSC after cardiac arrest in any setting.
5. ANZCOR suggests avoiding hyperoxia in adults with ROSC after cardiac arrest in any setting.
6. Once ROSC has been established and the oxygen saturation of arterial blood (SaO2) can be monitored reliably (by pulse oximetry [SpO2] and/or arterial blood gas analysis [SaO2]), it is reasonable to titrate the inspired oxygen to achieve a target saturation between 94 – 98%.
7. In patients with suspected or proven acute coronary syndromes, the routine use of supplemental oxygen is not recommended.
8. Oxygen therapy is indicated for patients with suspected or proven acute coronary syndromes, with hypoxia and those with evidence of shock, to correct tissue hypoxia.

   It is recommended that oxygen administration be targeted to achieve oxygen saturation (SpO_2) of 94-98%.

9. In patients with suspected or proven acute coronary syndromes and the absence of hypoxia, the benefit of oxygen therapy is uncertain, and in some cases oxygen therapy may be harmful.
10. Patients who have experienced an acute stroke and are hypoxic should be given supplemental oxygen. It is recommended that oxygen administration be targeted to achieve oxygen saturation (SpO_2) of 94-98%.
11. The routine use of supplemental oxygen is not recommended in acute stroke patients who are not hypoxic.
12. All patients with shock, major trauma, sepsis or other critical illness should be managed initially with high concentration oxygen therapy from a reservoir mask. It is recommended that oxygen administration be targeted to achieve an oxygen saturation (SpO_2) of 94-98%.
13. The routine use of supplemental oxygen high-dose oxygen via a reservoir mask is recommended for a patient with carbon monoxide poisoning.
14. Patients developing symptoms of decompression sickness after diving should be treated with high flow oxygen as soon as possible.
15. In patients with Paraquat poisoning or bleomycin lung injury the routine use of supplemental oxygen is not recommended.
16. In patients with Paraquat poisoning or bleomycin lung injury it is recommended that oxygen administration be targeted to achieve oxygen saturation (SpO_2) of 88-92%.
17. In patients who are at risk of hypercapnic respiratory failure, the routine use of supplemental oxygen is not recommended.
18. In patients who are at risk of hypercapnic respiratory failure it is recommended that oxygen administration be targeted to achieve oxygen saturation (SpO_2) of 88-92%.

The use of high concentrations of inspired oxygen has been routine during Advanced Life Support. The use of supplemental oxygen is not without risk, and its routine use has been questioned. Oxygen therapy should only be used by personnel trained in its use, and its effects should be monitored whenever possible, usually by the use of pulse oximetry. The pulse oximeter usually reads within ± 2%, but is less accurate in some specific situations (including poor perfusion, carbon monoxide toxicity etc).[1,2]

Adverse effects of the administration of oxygen include: worsened ventilation/perfusion matching; absorption atelectasis; myocardial ischaemia; reduced cardiac output; reduced coronary, cerebral and renal blood flow; increased peripheral resistance and blood pressure; and increased reactive oxygen species.[2]

Of particular concern in the peri-arrest period is the concern about increased oxidative damage, increased neuronal death, and worse neurologic function.[3,4]

Recommendations

Oxygen therapy should only be used by personnel trained in its use, and its effects should be monitored whenever possible, usually by the use of pulse oximetry. [Class A; Expert consensus opinion]

In patients requiring advanced life support, oxygen should be administered if the oxygen saturation (SpO_2) falls below 94% unless contraindications exist. [Class A; Expert consensus opinion]

1 Oxygen Use During Cardiac Arrest

There were no adult (>8 years of age) human studies that addressed directly whether titrated oxygen compared with 100% oxygen during CPR affects outcome. Two animal studies that used a fibrillatory model of cardiac arrest suggested that use of 100% oxygen during CPR and for 15–60 min after ROSC results in worse neurological outcomes compared with normoxic (21% oxygen, room air) resuscitation, whereas one animal study using an asphyxial model documented that ventilation with either 100% oxygen or 21% oxygen during resuscitation did not affect outcome.[5]

A recent publication observed an association between improved oxygenation during cardiac arrest and improved rates of hospital admission.[6]

ANZCOR recommends the use of 100% oxygen during adult cardiac arrest (CoSTR 2015, weak recommendation, very low quality evidence). [7]

2 Oxygen Use after Return of Spontaneous Circulation (ROSC)

After ROSC, toxic oxygen byproducts (reactive oxygen species, free radicals) are produced that may damage cell membranes, proteins, and DNA (reperfusion injury).

A number of animals studies have suggested that significant harm may result from the use of high concentrations of oxygen in the early resuscitation period.[3,5]

One randomised prospective clinical trial in patients who had been resuscitated from a cardiac arrest compared ventilation with 30% oxygen or 100% oxygen for the first 60 min after ROSC. Mean partial pressure of oxygen in arterial blood (PaO2) at 60min after ROSC was 110 ± 25 mmHg in the 30% oxygen group and 343 ± 174mmHg in the 100% oxygen group. No statistical difference was detected in serum biomarkers of acute brain injury, survival to hospital discharge, or the percent of patients with good neurological outcome (cerebral performance category of 1 or 2) at hospital discharge. However, this study was not adequately powered to detect important differences in survival and cerebral performance category at hospital discharge (n = 14 per group). A significant subset of patients in this study (30%) who were ventilated with 30% oxygen after ROSC required increased FiO2 to maintain a pulse oximetry reading of >95%. The study was underpowered to determine efficacy or harm. [8]

The results of recently published large observational studies which assessed the association between hyperoxia (utilising a number of ways of defining hyperoxia) after ROSC and in-hospital mortality in humans have been inconsistent and conflicting.[9-15]

Recommendations

ANZCOR recommends avoiding hypoxia in adults with ROSC after cardiac arrest in any setting (CoSTR 2015, strong recommendation, very low quality evidence).[7]

ANZCOR suggests avoiding hyperoxia in adults with ROSC after cardiac arrest in any setting (CoSTR 2015, weak recommendation, very low quality evidence).[7]

Once ROSC has been established and the oxygen saturation of arterial blood (SaO2) can be monitored reliably (by pulse oximetry [SpO2] and/or arterial blood gas analysis [SaO2]), it is reasonable to titrate the inspired oxygen to achieve a target saturation between 94 – 98%. [Class A; LOE III-2, Expert consensus opinion]

3 Oxygen use in other specific scenarios

3.1 Acute Coronary Syndromes (also see Guideline 14.2)

The routine use of supplemental oxygen is not recommended. Supplemental oxygen should be initiated if the patient has breathlessness, hypoxaemia and signs of heart failure or shock.[16] There is relatively limited evidence from clinical studies to support the routine use of oxygen therapy in ACS.[17] The use of oxygen saturation monitoring by non-invasive techniques such as pulse oximetry, may be very useful in guiding oxygen therapy.[18] However it is important to understand that hyperoxaemia may be potentially harmful in uncomplicated myocardial infarction.[16,19]

Recommendations

In patients with suspected or proven acute coronary syndromes, the routine use of supplemental oxygen is not recommended. [Class A; Expert consensus opinion]

Oxygen therapy is indicated for patients with hypoxia and those with evidence of shock, to correct tissue hypoxia. It is recommended that oxygen administration be targeted to achieve oxygen saturation (SpO_2) of 94-98%. [Class A; Expert consensus opinion]

In the absence of hypoxia, the benefit of oxygen therapy is uncertain, and in some cases oxygen therapy may be harmful. [Class A; Expert consensus opinion]

3.2 Stroke

The use of oxygen in acute stroke is still controversial, and evidence is still being collected.[20] The routine use of supplemental oxygen is not recommended in acute stroke patients who are not hypoxic. Patients with oxygen saturation <95% should be given supplemental oxygen.[21]

Recommendations

Patients who have experienced an acute stroke and are hypoxic should be given supplemental oxygen. It is recommended that oxygen administration be targeted to achieve oxygen saturation (SpO_2) of 94-98%. [Class A; Expert consensus opinion]

The routine use of supplemental oxygen is not recommended in acute stroke patients who are not hypoxic. [Class A; Expert consensus opinion]

3.3 Other critical illnesses

Treatment recommendation:

All patients with shock, major trauma, sepsis or other critical illness should be managed initially with high concentration oxygen therapy from a reservoir mask. It is recommended that oxygen administration be targeted to achieve an oxygen saturation (SpO_2) of 94-98%. [2] [Class A; Expert consensus opinion]

4 Oxygen Use in Other Specific Scenarios: High Oxygen Goals

4.1 Carbon monoxide poisoning

The most important treatment for a patient with carbon monoxide poisoning is to give high-dose oxygen via a reservoir mask. Pulse oximetry cannot screen for carbon monoxide exposure as it does not differentiate carboxyhaemoglobin from oxyhaemoglobin. The blood carboxyhaemoglobin level must be measured to assess the degree of carbon monoxide poisoning.[2]

Recommendations

The routine use of supplemental oxygen high-dose oxygen via a reservoir mask is recommended for a patient with carbon monoxide poisoning. [Class A; Expert consensus opinion]

4.2 Diving emergencies

Musculoskeletal or neurologic symptoms occuring soon after diving may be signs of decompression sickness and should be treated with high flow oxygen as soon as possible.[22,23]

Recommendations

Patients developing symptoms of decompression sickness after diving should be treated with high flow oxygen as soon as possible. [Class A; Expert consensus opinion]

5 Oxygen Use in Specific Scenarios: Lower Oxygen Goals

5.1 Paraquat poisoning and bleomycin lung injury

Oxygen is known to be hazardous to patients with paraquat poisoning. Oxygen worsens bleomycin lung injury. Because of these risks, supplemental oxygen should be given to patients with these conditions only if needed, aiming for a target range of saturation (SpO_2) of 88–92%.[2]

Recommendations

In patients with Paraquat poisoning or bleomycin lung injury the routine use of supplemental oxygen is not recommended. [Class A; Expert consensus opinion]

It is recommended that oxygen administration be targeted to achieve oxygen saturation (SpO_2) of 88-92%. [Class A; Expert consensus opinion]

5.2 Patients at risk of hypercapnic respiratory failure

Uncontrolled supplemental oxygen therapy can be harmful to patients who are at risk of hypercapnic respiratory failure. If high concentrations of oxygen are given to these patients, the oxygen level in the blood will rise but the level of carbon dioxide will also rise and this can cause acidosis with subsequent organ dysfunction and, when severe, coma. The target SpO2 is 88–92% if the patient is at risk of hypercapnic respiratory failure.

A small reduction in ventilation may be a contributing factor to the rise in carbon dioxide levels during oxygen therapy in COPD. Much of the rise in carbon dioxide which occurs is due to deterioration in the matching of blood flow and gas flow in the lungs.[2] This can be avoided by giving controlled lower concentration oxygen therapy to vulnerable patients.

It is not possible to predict if individual patients with COPD will develop hypercapnia during an acute exacerbation, so all patients with moderate or severe COPD should be considered to be at risk of this complication until the results of blood gas measurements are available.

If the diagnosis is unknown, patients aged >50 years who are long-term smokers with a history of chronic breathlessness on minor exertion such as walking on level ground and no other known cause of breathlessness should be treated as if having COPD. Patients without diagnosed COPD, but at risk of hypercapnic respiratory failure include patients with:

- cystic fibrosis
- bronchiectasis
- severe kyphoscoliosis or severe ankylosing spondylitis
- severe lung scarring from old tuberculosis (especially with thoracoplasty)
- morbid obesity (body mass index >40 kg/m2)
- musculoskeletal disorders with respiratory muscle weakness (especially if on home ventilation)
- overdose of opioids, benzodiazepines or other respiratory depressant drugs.

Recommendations

In patients who are at risk of hypercapnic respiratory failure, the routine use of supplemental oxygen is not recommended. [Class A; Expert consensus opinion]

In these patients, it is recommended that oxygen administration be targeted to achieve oxygen saturation (SpO_2) of 88-92%. [Class A; Expert consensus opinion]

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AUSTRALIAN RESUSCITATION COUNCIL

NEW ZEALAND Resuscitation Council WHAKAHAUORA AOTEAROA

ANZCOR Guideline 11.7 – Post-resuscitation Therapy in Adult Advanced Life Support

Summary

This guideline provides advice on post-resuscitation care because a comprehensive treatment protocol including multiple interventions provided in a structured way may improve survival after cardiac arrest

Who does this guideline apply to?

This guideline applies to adults who require advanced life support

Who is the audience for this guideline?

This guideline is for health professionals and those who provide healthcare in environments where equipment and drugs are available.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) make the following recommendations:

1. ANZCOR suggests hemodynamic goals (e.g., mean arterial pressure [MAP], systolic blood pressure [SBP]) be considered during postresuscitation care and as part of any bundle of post-resuscitation interventions.
2. ANZCOR recommends avoiding hypoxia in adults with ROSC after cardiac arrest in any setting.
3. ANZCOR suggests avoiding hyperoxia in adults with ROSC after cardiac arrest in any setting.
4. Once ROSC has been established and the oxygen saturation of arterial blood (SaO2) can be monitored reliably (by pulse oximetry [SpO2] and/or arterial blood gas analysis [SaO2]), it is reasonable to titrate the inspired oxygen to achieve a target saturation between 94 – 98%.
5. ANZCOR suggests maintaining $PaCO_2$ within a normal physiological range as part of a post-ROSC bundle of care.
6. ANZCOR suggests no modification of standard glucose management protocols for adults with ROSC after cardiac arrest.
7. Providers should monitor blood glucose frequently after cardiac arrest and should treat hyperglycemia (>10 mmol/l) with insulin but avoid hypoglycemia.
8. It may be reasonable to continue an infusion of an antiarrhythmic drug that successfully restored a stable rhythm during resuscitation (e.g. lignocaine 2-4 mg/min or amiodarone 0.6 mg/kg/hr for 12-24 hours).

9. If no antiarrhythmic drug was used during resuscitation from a shockable rhythm, an antiarrhythmic drug may be considered to prevent recurrent VF.
10. ANZCOR recommends targeted temperature management (TTM) for adults victims of cardiac arrest who remain unresponsive after ROSC (see Guideline 11.9 for details).
11. ANZCOR suggests against routine seizure prophylaxis in post–cardiac arrest patients.
12. ANZCOR recommends the treatment of seizures in post–cardiac arrest patients.
13. Maintenance therapy for seizures should be started after the first event and potential precipitating causes (e.g. intracranial haemorrhage, electrolyte imbalance, etc) should be excluded.
14. In patients with STEMI or new LBBB on ECG following ROSC after OHCA, immediate angiography and percutaneous coronary intervention (PCI) should be considered.
15. ANZCOR recommends emergency cardiac catheterization laboratory evaluation in comparison with cardiac catheterization later in the hospital stay or no catheterization in select adult patients with ROSC after OHCA of suspected cardiac origin with ST elevation on ECG.
16. It is reasonable to perform immediate angiography and PCI in selected patients, despite the absence of ST segment elevation on the ECG or prior clinical findings, such as chest pain.
17. ANZCOR suggests emergency cardiac catheterization laboratory evaluation in comparison with cardiac catheterization later in the hospital stay or no catheterization in select adult patients who are comatose with ROSC after OHCA of suspected cardiac origin with-out ST elevation on ECG.
18. It is reasonable to include cardiac catheterization in standardized post-cardiac arrest protocols as part of an overall strategy to improve neurologically intact survival in this patient group.
19. Targeted temperature management is recommended in combination with primary PCI, and should be started as early as possible, preferably prior to initiation of PCI.
20. After resuscitation all patients should be reassessed and re-evaluated for resuscitation-related injuries. The extent of injuries is often underestimated by standard investigations (e.g. chest radiograph). Other complications of resuscitation (e.g. incorrect placement of tubes) should be identified and treated. Intravascular lines inserted under emergency conditions may need to be replaced.
21. ANZCOR suggests that OHCA patients should be considered for transport to a specialist cardiac arrest center as part of wider regional system of care for management of patients with OHCA.

Prognostication and cardiac arrest

22. Relying on the neurologic exam during or immediately after cardiac arrest to predict outcome is not recommended and should not be used.

Prognostication with TTM

23. ANZCOR suggest against the use of clinical criteria alone before 72 hours after ROSC to estimate prognosis.
24. ANZCOR suggests that multiple modalities of testing (clinical exam, neurophysiological

measures, imaging, or blood markers) be used to estimate prognosis instead of relying on single tests or findings.

25. ANZCOR recommends using bilaterally absent pupillary light reflexes (PLRs) or the combined absence of both pupillary and corneal reflexes at least 72 hours after ROSC to predict poor outcome in patients who are comatose after resuscitation from cardiac arrest and who are treated with TTM.

26. ANZCOR suggests against using an absent (M1) or extensor motor response to pain (M2) alone to predict poor outcome, given its high FPR. However, due to its high sensitivity, this sign may be used to identify the population with poor neurologic status needing prognostication or to predict poor outcome in combination with other more robust predictors.

27. ANZCOR suggests against the use of myoclonus during the first 72 hours from ROSC as a predictor for prognosticating a poor neurologic outcome.

28. ANZCOR suggests that the presence of status myoclonus during the first 72 hours from ROSC be considered at 72 hours after ROSC (in combination with other factors) as a predictor for prognosticating a poor neurologic outcome.

29. ANZCOR suggests prolonging the observation of clinical signs when interference from residual sedation or paralysis is suspected, so that the possibility of obtaining false-positive results is minimized. We recommend that the earliest time to prognosticate a poor neurologic outcome is 72 hours after ROSC, and should be extended longer if the residual effect of sedation and/or paralysis confounds the clinical examination.

30. ANZCOR recommends using bilateral absence of N20 somatosensory evoked potentials (SSEP) wave measured at least 72 hours after ROSC to predict poor outcome in patients who are comatose after resuscitation from cardiac arrest and who are treated with TTM.

31. AZCOR suggests using persistent absence of EEG reactivity to external stimuli at 72 hours or longer after ROSC, presence of persistent burst suppression after rewarming, or intractable and persistent status epilepticus (SE) to predict poor outcome in patients who are comatose after resuscitation from cardiac arrest and who are treated with TTM.

32. ANZCOR recommends against using Bispectral Index (BIS) to predict poor outcome during TTM in patients who are comatose after resuscitation from cardiac arrest and are treated with TTM.

33. ANZCOR suggests using utmost care and preferably sampling at multiple serial time points (24–72 hours) when assessing neuron-specific enolase (NSE), to avoid false-positive results due to hemolysis.

34. ANZCOR suggests using serial high-serum values of NSE at 48 to 72 hours from ROSC in combination with other predictors for predicting poor neurologic outcome in patients who are comatose after cardiac arrest and who are treated with TTM.

35. ANZCOR suggests using brain imaging studies for prognostication only in centers where specific experience is available.

36. ANZCOR suggests using the presence of a marked reduction of the gray matter/white matter (GM/WM) ratio on brain CT within 2 hours after ROSC or the presence of extensive diffusion restriction on brain MRI at 2 to 6 days after ROSC in combination with other predictors for prognosticating a poor neurologic outcome in patients who are comatose after cardiac arrest and who are treated with TTM.

Prognostication without TTM

37. ANZCOR suggest against the use of clinical criteria alone before 72 hours after ROSC to estimate prognosis.
38. ANZCOR suggests that multiple modalities of testing (clinical exam, neurophysiological measures, imaging, or blood markers) be used to estimate prognosis instead of relying on single tests or findings.
39. ANZCOR recommends using the absence of PLR (or the combined absence of both pupillary and corneal reflexes) at 72 hours or greater from ROSC to predict poor outcome in patients who are comatose after resuscitation from cardiac arrest and who are not treated with TTM.
40. ANZCOR suggests against using an absent or extensor motor response to pain (M≤2) alone to predict poor outcome, given its high FPR.
41. ANZCOR suggests using the presence of myoclonus or status myoclonus within 72 hours from ROSC in combination with other predictors to predict poor outcome in comatose survivors of cardiac arrest.
42. ANZCOR suggests prolonging the observation of clinical signs when interference from residual sedation or paralysis is suspected, so that the possibility of obtaining false-positive results is minimized.
43. ANZCOR recommends using bilateral absence of the N20 SSEP wave within 72 hours from ROSC to predict poor outcome in patients who are comatose after cardiac arrest and who are not treated with TTM.
44. ANZCOR suggests using the presence of burst suppression on EEG at 72 hours from ROSC in combination with other predictors for prognosticating a poor neurologic outcome in patients who are comatose after cardiac arrest and who are not treated with TTM.
45. ANZCOR suggests against using EEG grades for prognostication due to the inconsistencies in their definitions.
46. ANZCOR suggests against using low-voltage EEG for prognostication, given the potential interferences of technical factors on EEG amplitude.
47. ANZCOR suggests using high serum values of NSE at 24 to 72 hours from ROSC in combination with other predictors for prognosticating a poor neurologic outcome in patients who are comatose after cardiac arrest and who are not treated with TTM.
48. ANZCOR suggests using the presence of a marked reduction of the GM/WM ratio on brain CT within 48 hours after ROSC or the presence of extensive reduction in diffusion on brain MRI at 2 to 6 days after ROSC only in combination with other more-established predictors for prognosticating a poor neurologic outcome in patients who are comatose after resuscitation from cardiac arrest and who are not treated with TTM).
49. ANZCOR suggests using brain-imaging studies for prognostication only in centers where specific experience is available.

Outcome for Survivors

50. Cardiac arrest survivors may experience post-arrest problems including anxiety, depression, post-traumatic stress, and difficulties with cognitive function. Clinicians should be aware of these potential problems, screen for them and, if found, treat them.

Organ Donation

51. ANZCOR recommends that all patients who have restoration of circulation after CPR and who subsequently progress to death be evaluated for organ donation.

After the return of a spontaneous circulation (ROSC), resuscitation DOES NOT STOP. It is essential to continue maintenance of airway, breathing and circulation. ROSC is just the first step toward the goal of complete recovery from cardiac arrest. Interventions in the post-resuscitation period are likely to significantly influence the final outcome. A comprehensive treatment protocol including multiple interventions provided in a structured way may improve survival after cardiac arrest.[1]
Hypoxic brain injury, myocardial injury or subsequent organ failure are the predominant causes of morbidity and mortality after cardiac arrests.[2]

The aims of therapy after initial resuscitation are to:

- Continue respiratory support.
- Maintain cerebral perfusion.
- Treat and prevent cardiac arrhythmias.
- Determine and treat the cause of the arrest.

In addition treatable causes of cardiac arrest need to be addressed. These include:

- Hypoxaemia
- Hypovolaemia
- Hypo/Hyperkalaemia and other metabolic disorders including acidosis and disturbances of magnesium and calcium
- Hypo/Hyperthermia
- Tension pneumothorax
- Tamponade: pericardial
- Toxins/poisons/drugs including carbon monoxide, and cyclic antidepressants
- Thrombosis: pulmonary embolus /acute myocardial infarction.

A full history and examination will guide the possible investigations. Electrolyte disorders such as hypo- and hyper-natraemia may cause continuing cerebral damage. Serum electrolytes, arterial blood gases and ECG should be performed to guide further treatment.[1]

1.1 Blood Pressure

It is imperative to ensure an adequate systemic arterial blood pressure as soon as practicable after return of spontaneous circulation. Despite limited clinical data, the known pathophysiology of post-cardiac arrest syndrome provides a rationale for titrating hemodynamics to optimize organ perfusion.[1]

Recommendations

ANZCOR suggests hemodynamic goals (e.g., mean arterial pressure [MAP], systolic blood pressure [SBP]) be considered during postresuscitation care and as part of any bundle of postresuscitation interventions (CoSTR 2015, weak recommendation, low-quality evidence).[3]

There is insufficient evidence to recommend specific hemodynamic goals; such goals should be considered on an individual patient basis and are likely to be influenced by post-cardiac arrest status and pre-existing comorbidities (CoSTR 2015, weak recommendation, low-quality evidence).[3]

Values and preferences

In making these recommendations, we place a higher value on the recognition that while hemodynamic goals are likely important to optimize outcome, specific targets remain unknown and likely vary depending on individual physiology and comorbid status.[3]

Aim for a blood pressure equal to the patient's usual blood pressure or at least a systolic pressure greater than 100mg Hg. If the blood pressure falls, a vasopressor may be given by small intravenous increments (e.g. adrenaline 50 to 100 mcg) or infusion until fluid status and the need for intravascular volume expansion can be assessed. [Class A; Expert consensus opinion]

There is insufficient evidence to support or refute the routine use of intravenous fluids following sustained return on spontaneous circulation after cardiac arrest. Rapid infusion of cold 0.9% saline or lactacted Ringers appears to be well tolerated when used to induce therapeutic hypothermia. Based on the pathophysiology of postcardiac arrest syndrome,[2] it is reasonable to use intravenous fluids as part of a package of post-cardiac arrest care.[1]

There is insufficient evidence to support or refute the routine use of vasopressors and/ or inotropes for improving survival in adult patients with cardiovascular dysfunction after resuscitation from cardiac arrest.[1] If vasoactive drugs are used, then as soon as possible any vasoconstricting drugs should be given by a dedicated central venous line. [Class A; Expert consensus opinion]

There is insufficient evidence to support or refute the use of mechanical circulatory support (e.g. an intra-aortic balloon pump) in post-cardiac arrest patients who have cardiovascular dysfunction.[1]

Intubation and ventilation are continued in the immediate post arrest period guided by appropriate monitoring.

1.2 Oxygenation

Recommendations

ANZCOR recommends avoiding hypoxia in adults with ROSC after cardiac arrest in any setting (CoSTR 2015, strong recommendation, very low quality evidence).[3]

ANZCOR suggests avoiding hyperoxia in adults with ROSC after cardiac arrest in any setting (CoSTR 2015, weak recommendation, very low quality evidence).[3]

Once ROSC has been established and the oxygen saturation of arterial blood (SaO2) can be monitored reliably (by pulse oximetry [SpO2] and/or arterial blood gas analysis [SaO2]), it is reasonable to titrate the inspired oxygen to achieve a target saturation between 94 – 98%. [Class A; LOE III-2, Expert consensus opinion]. See also Guideline 11.6.1.

1.3 Control of arterial carbon dioxide

Five studies in adults and numerous animal studies documented harmful effects of hypocapnia (cerebral ischemia) after cardiac arrest. Two studies provide neutral evidence.

There are no data to support the targeting of a specific $PaCO_2$ after resuscitation from cardiac arrest. Data extrapolated from patients with brain injury however, imply that ventilation to normocarbia (e.g. $PaCO_2$ 35 to 40 mmHg) is appropriate.[4]

Routine hyperventilation may be detrimental (e.g. result in cerebral vasoconstriction) and should be avoided. [Class A; Extrapolated evidence] Arterial blood gas measurements should be used to titrate ventilation in the immediate post-resuscitation period, rather than End Tidal CO_2 levels.[1]

Recommendations

ANZCOR suggests maintaining $PaCO_2$ within a normal physiological range as part of a post-ROSC bundle of care (CoSTR 2015, weak recommendation, very-low-quality evidence).[3]

Values and preferences

There is no good evidence to suggest or recommend either hypercarbia or hypocarbia. In the absence of evidence to that end, combined with a potential suggestion of harm, we suggest maintaining normocarbia. Many physiological considerations may influence selection of $PaCO_2$ goals for individual patients.

1.4 Blood glucose control

Several human studies have documented a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurologic outcome. There is good evidence that persistent hyperglycemia after stroke is associated with a worse neurologic outcome.

One human randomized interventional study that prospectively evaluated strict glucose control (72-108 mg/dl, 4-6 mmol/l]) compared to moderate glucose control (108-144 mg/dl, 6-8 mmol/l) in patients resuscitated from prehospital cardiac arrest with ventricular fibrillation found no survival benefit with strict glucose control. Five retrospective studies in post-cardiac arrest patients suggested an association of higher glucose levels with increased mortality and worse neurological outcomes, but these findings may be related to other factors.

Based on these studies, the suggested target ranges for glucose values have been variable. A good randomized trial of intensive glucose control versus conventional glucose control in the largest number of ICU patients to date reported increased mortality in patients treated with intensive glucose control. Two meta-analyses of studies of tight glucose control versus conventional glucose control in critically ill patients showed no significant difference in mortality but found tight glucose control was associated with a significantly increased risk of hypoglycemia.[1]

The optimal blood glucose target in critically ill patients has not been determined. Comatose patients were at particular risk from unrecognized hypoglycemia, and the risk of this complication occurring increases as the target blood glucose concentration is lowered.[1]

Recommendation

ANZCOR suggests no modification of standard glucose management protocols for adults with ROSC after cardiac arrest (CoSTR 2015, weak recommendation, moderate-quality evidence).[3]

Providers should monitor blood glucose frequently after cardiac arrest and should treat hyperglycemia (>10 mmol/l) with insulin but avoid hypoglycemia.[1] [Class B; LOE II]

1.5 Prophylactic anti-arrhythmic agents

No studies specifically and directly addressed the prophylactic use of antiarrhythmic therapy started immediately after resuscitation from cardiac arrest. Observational studies document inconsistent improvement in long-term survival when prophylactic antiarrhythmics were given to survivors of cardiac arrest from all causes.[3]

Recommendation

It may be reasonable to continue an infusion of an antiarrhythmic drug that successfully restored a stable rhythm during resuscitation (e.g. lignocaine 2-4 mg/min or amiodarone 0.6 mg/kg/hr for 12-24 hours). [Class B; Expert consensus opinion]

If no antiarrhythmic drug was used during resuscitation from a shockable rhythm, an antiarrhythmic drug may be considered to prevent recurrent VF. [Class B; Expert consensus opinion]

1.6 Temperature control

Targeted temperature management has been shown to be beneficial in some patients still comatose after return of spontaneous circulation. When actively rewarming a severely hypothermic patient, practitioners should take this information into account. Hyperthermia should be avoided.[3]

Recommendation

ANZCOR recommends targeted temperature management for adults victims of cardiac arrest who remain unresponsive after ROSC (CoSTR 2015, strong recommendation, low-quality evidence) (see Guideline 11.9 for details).[3]

1.7 Sedation and paralysis

Apart from the data related to induced hypothermia, there were no data to support or refute the use of a defined period of ventilation, sedation, and neuromuscular blockade after cardiac arrest. One observational study in adults documents increased incidence of pneumonia when sedation is prolonged beyond 48 hours after prehospital or in-hospital cardiac arrest.[4]

There is insufficient data to recommend for or against the use of neuroprotective drugs (such as thiopental, glucocorticoids, nimodipine, lidoflazine, or diazepam) in comatose cardiac arrest post return of spontaneous circulation not treated with hypothermia or as an adjunct to targeted temperature management in the post arrest treatment of adult cardiac arrest.[1]

1.8 Seizure control

Studies document a 3-44% incidence of seizures after sustained return of spontaneous circulation.[1] Seizures increase the oxygen requirements of the brain and can cause life-threatening arrhythmias and respiratory arrest.

Studies report no difference in neurologic outcome after use of single dose diazepam or magnesium or both; or thiopental given after sustained return of spontaneous circulation.[5-7]

There are no studies addressing prompt and aggressive treatment after the first seizure occurring after circulation was restored. Seizures in the post arrest period may be refractory to multiple medications. There are insufficient data to support or refute the use of specific anti seizure medication in the prevention or treatment of seizures in after return of spontaneous circulation.[1]

Recommendation

ANZCOR suggests against routine seizure prophylaxis in post–cardiac arrest patients (CoSTR 2015, weak recommendation, very-low-quality evidence).[3]

ANZCOR recommends the treatment of seizures in post–cardiac arrest patients (CoSTR 2015, strong recommendation, very-low-quality evidence).[3].

Maintenance therapy for seizures should be started after the first event and potential precipitating causes (e.g. intracranial haemorrhage, electrolyte imbalance, etc) should be excluded. [Class A; Expert consensus opinion]

1.9 Treatment of underlying cause of the cardiac arrest

If not already undertaken, management should be directed toward the treatment of underlying causes that have been identified (e.g. management of myocardial infarction, correction of electrolyte abnormalities, treatment of tension pneumothorax etc.).

Myocardial infarction

There is evidence of underlying ischemic heart disease in the majority of patients who have an out-of-hospital cardiac arrest.

Acute coronary artery occlusion is known to be the precipitating factor in many of these patients. While coronary artery occlusion after cardiac arrest is associated with ECG ST elevation or LBBB, it can also occur in the absence of these findings.[8]

Clinical findings of coma in patients prior to PCI are commonly present in OHCA patients, and should not be a contraindication to consider immediate angiography and PCI.

Recommendations

In patients with STEMI or new LBBB on ECG following ROSC after OHCA, immediate angiography and percutaneous coronary intervention (PCI) should be considered. [Class A; LOE III-3]

ANZCOR recommends emergency cardiac catheterization laboratory evaluation in comparison with cardiac catheterization later in the hospital stay or no catheterization in select adult patients with ROSC after OHCA of suspected cardiac origin with ST elevation on ECG (CoSTR 2015, strong recommendation, low-quality evidence).[3] Also see Guideline 11.3

It is reasonable to perform immediate angiography and PCI in selected patients, despite the absence of ST segment elevation on the ECG or prior clinical findings, such as chest pain. [Class A; LOE III-3]

ANZCOR suggests emergency cardiac catheterization laboratory evaluation in comparison with cardiac catheterization later in the hospital stay or no catheterization in select adult patients who are comatose with ROSC after OHCA of suspected cardiac origin with-out ST elevation on ECG (CoSTR 2015, weak recommendation, very-low-quality evidence).

It is reasonable to include cardiac catheterization in standardized post-cardiac arrest protocols as part of an overall strategy to improve neurologically intact survival in this patient group. [Class A; LOE III-3]

Targeted temperature management is recommended in combination with primary PCI, and should be started as early as possible, preferably prior to initiation of PCI. [Class A; LOE III-3]

1.10 Pulmonary embolus

Despite good theoretical reasons why fibrinolysis following cardiac arrest in patients with suspected pulmonary embolism might be beneficial, there is no direct evidence to that effect. Several studies showed no significant increase in survival to hospital discharge. There was an increase in bleeding complications following fibrinolysis in most of those studies. One study suggested that the risk of major haemorrhage was further increased in patients who have undergone CPR.[1]

In patients with diagnosed or suspected pulmonary embolism after return of spontaneous circulation following cardiac arrest, there is inadequate evidence to recommend for or against the use of fibrinolytic therapy in addition to heparin. The mortality with surgical embolectomy for suspected or diagnosed pulmonary embolism is high if it follows cardiac arrest and it should be avoided in patients who have received CPR. There are few data on percutaneous mechanical thromboembolectomy, but it may be beneficial and may be considered in patients sustaining cardiac arrest from a pulmonary embolism who are not candidates for fibrinolytic therapy.[1]

1.11 Resuscitation related injuries

Rib fractures and other injuries are common[9] but acceptable consequences of CPR given the alternative of death from cardiac arrest.

Recommendation

After resuscitation all patients should be reassessed and re-evaluated for resuscitation-related injuries. The extent of injuries is often underestimated by standard investigations (e.g. chest radiograph).[10] Other complications of resuscitation (e.g. incorrect placement of tubes) should be identified and treated. Intravascular lines inserted under emergency conditions may need to be replaced. [Class B; Expert consensus opinion]

1.12 Resuscitation centres

While extrapolation from randomized and observational studies of systems of care for other acute time-sensitive conditions (trauma, ST elevation MI, stroke) suggests that specialist cardiac arrest centres and systems of care may be effective, there is insufficient direct evidence to recommend for or against their use.[11]

Recommendation

ANZCOR suggests that OHCA patients should be considered for transport to a specialist cardiac arrest center as part of wider regional system of care for management of patients with OHCA (CoSTR 2015, weak recommendation, low-quality evidence).[11]

Values and preferences

In making this recommendation, we recognize the development of cardiac arrest centers may be considered as a health improvement initiative, as has been performed for other critical conditions, including myocardial infarction, stroke, and major trauma, without the evidence of randomized trials.[3]

2 Prognosis

Many comatose post-cardiac arrest patients die or will survive with a poor neurologic outcome. Therefore, reliable methods to assess prognosis are needed to prevent continued unnecessary treatment and accurately inform families. Post-cardiac arrest prognostication was extensively reviewed in the 2010 and 2015 CoSTR processes.[1,3] The consensus of the task forces was that multimodal assessments should be used, should never rely on a single prognostication element, and supplementary tests should be considered in the context of the clinical examination. The most reliable combination and timing for assessments have not been fully determined.[3]

Recommendations address prognostication of comatose post-cardiac arrest patients treated with hypothermic TTM and patients not treated with hypothermic TTM. This approach was chosen because hypothermic TTM may alter the natural history of coma and may delay recovery of CNS function. Patients may be exposed to large doses of sedative and neuromuscular blockade during TTM. The metabolism of these agents maybe delayed during hypothermic TTM. Prognostic elements that are reliable in comatose post-cardiac arrest patients not treated with hypothermic TTM may be less reliable at the same time point inpatients treated with TTM.

Clinical signs, neurophysiological measurements, blood or cerebrospinal fluid markers, and imaging studies with high specificity for poor neurologic outcome, defined as death, vegetative state, or severe cerebral disability (CPC 3–5) have been assessed. These assessments are justified because they are likely to be used to justify limiting life-sustaining treatments. To quantify the specificity of the findings, CoSTR 2015 examined the false positive rate (FPR) of each sign for predicting unfavorable neurologic outcome, with a goal of 0% FPR. The 95% CI of the FPR was calculated, and the tendancy was to recommend a finding as useful if the FPR was less than 5%, and suggest that a finding might be useful if the FPR was less than 10%. In most cases, clinical signs and findings were considered individually, because few studies considered combinations of clinical findings.[3]

2.1 Prognostication during a cardiac arrest

Studies document some ability to predict outcome in adults when neurologic examination is undertaken during cardiac arrest, but there is insufficient negative predictive value for this assessment to be used clinically. It is impossible to predict accurately the degree of neurological recovery during or immediately after a cardiac arrest. After cessation of sedation (and/or induced hypothermia) the probability of awakening decreases with each day of coma.

Recommendation

Relying on the neurologic exam during cardiac arrest to predict outcome is not recommended and should not be used.[4] [Class A, Expert consensus opinion]

3 Prognostication for comatose cardiac arrest victims treated with TTM

Recommendations

ANZCOR suggest against the use of clinical criteria alone before 72 hours after ROSC to estimate prognosis (CoSTR 2015, weak recommendation, low-quality evidence).[3]

ANZCOR suggests that multiple modalities of testing (clinical exam, neurophysiological measures, imaging, or blood markers) be used to estimate prognosis instead of relying on single tests or findings (CoSTR 2015, weak recommendation, low-quality evidence). [3]

3.1 Clinical Examination

ANZCOR recommends using bilaterally absent pupillary light reflexes (PLRs) or the combined absence of both pupillary and corneal reflexes at least 72 hours after ROSC to predict poor outcome in patients who are comatose after resuscitation from cardiac arrest and who are treated with TTM (CoSTR 2015, strong recommendation, low-quality evidence). [3]

ANZCOR suggests against using an absent (M1) or extensor motor response to pain (M2) alone to predict poor outcome, given its high FPR. However, due to its high sensitivity, this sign may be used to identify the population with poor neurologic status needing prognostication or to predict poor outcome in combination with other more robust predictors (CoSTR 2015, weak recommendation, very low-quality evidence). [3]

ANZCOR suggests against the use of myoclonus during the first 72 hours from ROSC as a predictor for prognosticating a poor neurologic outcome (CoSTR 2015, weak recommendation, low-quality evidence). [3]

ANZCOR suggests that the presence of status myoclonus during the first 72 hours from ROSC be considered at 72 hours after ROSC (in combination with other factors) as a predictor for prognosticating a poor neurologic outcome (CoSTR 2015, weak recommendation, low-quality evidence). [3]

ANZCOR suggests prolonging the observation of clinical signs when interference from residual sedation or paralysis is suspected, so that the possibility of obtaining false-positive results is minimized.

ANZCOR recommends that the earliest time to prognosticate a poor neurologic outcome is 72 hours after ROSC, and should be extended longer if the residual effect of sedation and/or paralysis confounds the clinical examination (CoSTR 2015, weak recommendation, low-quality evidence). [3]

3.2 Electrophysiology

ANZCOR recommends using bilateral absence of N20 smatosensory evoked potentials (SSEP) wave measured at least 72 hours after ROSC to predict poor outcome in patients who

are comatose after resuscitation from cardiac arrest and who are treated with TTM (CoSTR 2015, strong recommendation, low-quality evidence).[3]

SSEP recording requires appropriate skills and experience, and utmost care should be taken to avoid electrical interference from muscle artifacts or from the ICU environment, as well as confounding drugs. This test is only ordered in the appropriate clinical context.

AZCOR suggests using persistent absence of EEG reactivity to external stimuli at 72 hours or longer after ROSC (weak recommendation, low-quality evidence), presence of persistent burst suppression after rewarming, or intractable and persistent status epilepticus (SE) (CoSTR 2015, weak recommendation, very-low-quality evidence) to predict poor outcome in patients who are comatose after resuscitation from cardiac arrest and who are treated with TTM.[3]

ANZCOR recommends against using Bispectral Index (BIS) to predict poor outcome during TTM in patients who are comatose after resuscitation from cardiac arrest and are treated with TTM (CoSTR 2015, strong recommendation, very-low-quality evidence).[3]

3.3 Blood Markers

ANZCOR suggests using utmost care and preferably sampling at multiple serial time points (24–72 hours) when assessing neuron-specific enolase (NSE), to avoid false-positive results due to hemolysis (CoSTR 2015, weak recommendation, very-low-quality evidence).[3]

ANZCOR suggests using serial high-serum values of NSE at 48 to 72 hours from ROSC in combination with other predictors for predicting poor neurologic outcome in patients who are comatose after cardiac arrest and who are treated with TTM (CoSTR 2015, weak recommendation, verylow-quality evidence). However, no threshold-enabling prediction with 0 FPR can be recommended, and NSE levels are insufficiently specific to be used alone for estimating prognosis.[3]

3.4 Imaging

ANZCOR suggests using brain imaging studies for prognostication only in centers where specific experience is available (CoSTR 2015, weak recommendation, very-low-quality evidence).[3]

ANZCOR suggests using the presence of a marked reduction of the gray matter/white matter (GM/WM) ratio on brain CT within 2 hours after ROSC or the presence of extensive diffusion restriction on brain MRI at 2 to 6 days after ROSC in combination with other predictors for prognosticating a poor neurologic outcome in patients who are comatose after cardiac arrest and who are treated with TTM (CoSTR 2015, weak recommendation, very-low-quality evidence).

Early imaging markers of poor prognosis should not prevent support for a sufficient period of time to observe other clinical features, although some extreme CT scan findings are consistent with herniation and brain death.[3]

4 Prognostication for comatose cardiac arrest victims not treated with TTM

ANZCOR suggest against the use of clinical criteria alone before 72 hours after ROSC to estimate prognosis (CoSTR 2015, weak recommendation, low-quality evidence).

ANZCOR suggests that multiple modalities of testing (clinical exam, neurophysiological measures, imaging, or blood markers) be used to estimate prognosis instead of relying on single tests or findings (CoSTR 2015, weak recommendation, low-quality evidence).

4.1 Clinical Examination

ANZCOR recommends using the absence of PLR (or the combined absence of both pupillary and corneal reflexes) at 72 hours or greater from ROSC to predict poor outcome in patients who are comatose after resuscitation from cardiac arrest and who are not treated with TTM (CoSTR 2015, strong recommendation, very-low-quality evidence).[3]

ANZCOR suggests against using an absent or extensor motor response to pain (M≤2) alone to predict poor outcome, given its high FPR (CoSTR 2015, weak recommendation, very-low-quality evidence). However, due to its high sensitivity, this sign may be used to identify the population with poor neurologic status needing prognostication or to predict poor outcome in combination with other more-robust predictors.[3]

ANZCOR suggests using the presence of myoclonus or status myoclonus within 72 hours from ROSC in combination with other predictors to predict poor outcome in comatose survivors of cardiac arrest (CoSTR 2015, weak recommendation, very-low-quality evidence).[3]

ANZCOR suggests prolonging the observation of clinical signs when interference from residual sedation or paralysis is suspected, so that the possibility of obtaining false-positive results is minimized (CoSTR 2015, weak recommendation, very-low-quality evidence).[3]

4.2 Electrophysiology

ANZCOR recommends using bilateral absence of the N20 SSEP wave within 72 hours from ROSC to predict poor outcome in patients who are comatose after cardiac arrest and who are not treated with TTM (CoSTR 2015, strong recommendation, very-low-quality evidence). SSEP recording requires appropriate skills and experience, and utmost care should be taken to avoid electrical interference from muscle artifacts or from the ICU environment.[3]

ANZCOR suggests using the presence of burst suppression on EEG at 72 hours from ROSC in combination with other predictors for prognosticating a poor neurologic outcome in patients who are comatose after cardiac arrest and who are not treated with TTM (CoSTR 2015, strong recommendation, very-low-quality evidence).[3]

ANZCOR suggests against using EEG grades for prognostication due to the inconsistencies in their definitions (CoSTR 2015, weak recommendation, very-low-quality evidence).[3]

ANZCOR suggests against using low-voltage EEG for prognostication, given the potential interferences of technical factors on EEG amplitude (CoSTR 2015, weak recommendation, very-low quality evidence).[3]

4.3 Blood Markers

ANZCOR suggests using high serum values of NSE at 24 to 72 hours from ROSC in combination with other predictors for prognosticating a poor neurologic outcome in patients who are comatose after cardiac arrest and who are not treated with TTM (CoSTR 2015, weak recommendation, very-low-quality evidence). However, no threshold-enabling predictionwith 0 FPR can be recommended. We suggest using utmost care and preferably sampling at multiple time points when assessing NSE, to avoid false-positive results due to hemolysis. [3]

4.4 Imaging

ANZCOR suggests using the presence of a marked reduction of the GM/WM ratio on brain CT within 48 hours after ROSC or the presence of extensive reduction in diffusion on brain MRI at 2 to 6 days after ROSC only in combination with other more-established predictors for prognosticating a poor neurologic outcome in patients who are comatose after resuscitation from cardiac arrest and who are not treated with TTM (CoSTR 2015, weak recommendation, very-low quality evidence). [3]

ANZCOR suggests using brain-imaging studies for prognostication only in centers where specific experience is available (CoSTR 2015, weak recommendation, very-low-quality evidence). [3]

5 Outcome of Resuscitation

Resuscitation after cardiac arrest produces a good quality of life in most long-term survivors. There is little evidence to suggest that resuscitation leads to a large pool of survivors with an unacceptable quality of life. Survivors may however suffer a variety of post-arrest problems that affect quality of life.

Recommendation

Cardiac arrest survivors may experience post-arrest problems including anxiety, depression, post-traumatic stress, and difficulties with cognitive function. Clinicians should be aware of these potential problems, screen for them and, if found, treat them.[6] [Class A; Expert consensus opinion]

5.1 Organ donation

ANZCOR recommends that all patients who have restoration of circulation after CPR and who subsequently progress to death be evaluated for organ donation (CoSTR 2015, strong recommendation, low-quality evidence).[3]

The Australian and New Zealand Intensive Care Society Death and Organ Donation Committee provides relevant advisory statements at:
http://www.anzics.com.au/Pages/DaOD.aspx

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ANZCOR Guideline 11.8 – Targeted Temperature Management (TTM) after Cardiac Arrest

Summary

This guideline provides advice on targeted temperature management (TTM) during the post-arrest period which is a therapy associated with improved outcomes.

Who does this guideline apply to?

This guideline applies to adults who require advanced life support after cardiac arrest

Who is the audience for this guideline?

This guideline is for health professionals and those who provide healthcare in environments where equipment and drugs are available.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) make the following recommendations:

1. ANZCOR recommends TTM as opposed to no TTM for adults with out-of-hospital cardiac arrest (OHCA) with an initial shockable rhythm who remain unresponsive after ROSC.
2. ANZCOR suggests TTM as opposed to no TTM for adults with OHCA with an initial non-shockable rhythm who remain unresponsive after ROSC.
3. ANZCOR suggests TTM as opposed to no TTM for adults with in-hospital cardiac arrest (IHCA) with any initial rhythm who remain unresponsive after ROSC.
4. ANZCOR recommends selecting and maintaining a constant target temperature between 32°C and 36°C for those patients in whom TTM is used.
5. No studies specifically address cardiac arrests due to non-cardiac causes, but it is reasonable to assume that these patients might also benefit from targeted temperature management.
6. Rapid infusion of ice-cold intravenous fluid, up to 30 ml kg-1 or ice packs are feasible, safe and simple methods for initially lowering core temperature up to 1.5 degrees. When intravenous fluids are used to induce hypothermia additional cooling strategies will be required to maintain hypothermia.

7. ANZCOR recommends against routine use of pre-hospital cooling with rapid infusion of large volumes of cold intravenous fluid immediately after ROSC.

8. ANZCOR suggests that if TTM is used, duration should be at least 24 hours.

9. ANZCOR suggests that percutaneous coronary intervention during TTM is feasible and safe and may be associated with improved outcome.

10. ANZCOR suggests institutions or communities planning to implement complex guidelines, such as targeted temperature management should consider using a comprehensive, multifaceted approach, including: clinical champions; a consensus-building process; multidisciplinary involvement; written protocols; detailed process description; practical logistic support; multi-modality, multi-level education; and rapid cycle improvement methods.

11. ANZCOR suggests prevention and treatment of fever in persistently comatose adults after completion of TTM between 32°C and 36°C.

Guideline

Induced hypothermia has been successfully used during cardiac surgery to protect against global cerebral ischaemia. Its use has been described in other clinical settings since the 1950s, particularly following cardiac arrest. Several animal and human studies have demonstrated the potential for therapeutic hypothermia to improve survival and neurological outcome in victims of cardiac arrest. The term targeted temperature management (TTM) is preferred to therapeutic hypothermia.

1 Who to cool and what temperature to cool to?

All studies of post-cardiac arrest therapeutic hypothermia have included only patients in coma. One trial defined coma as "not responding to verbal commands". The other trials defined coma similarly, used GCS ≤8, or did not provide a clear definition.[1]

One randomized controlled trial[2] (RCT) and a quasi-randomised trial[3] demonstrated improved neurological outcome at hospital discharge or at 6 months after hospital discharge in comatose patients after out-of-hospital ventricular fibrillation cardiac arrest. Cooling was initiated within minutes to hours after return of spontaneous circulation and a temperature range of 32-34ºC was maintained for 12- 24 hours. Studies with historical control groups have shown improvement in neurological outcome after therapeutic hypothermia for comatose survivors of ventricular fibrillation cardiac arrest and a systematic review demonstrated that conventional cooling methods were more likely to reach a best cerebral performance category score of 1 or 2 (five point scale where one is good and five is brain death) with a relative risk of 1.55 995% CI 1.22-1.96) and more likely to survive to hospital discharge (relative risk of 1.35 95% CI 1.1 to 1.65) compared with standard post resuscitation care.[1, 4]

Cohort studies comparing mild induced hypothermia (32–34◦C) to no temperature management in OHCA found no difference in neurological outcome.[5-7] While a retrospective registry study of 1830 patients documented an increase in poor neurological out-come among those with non shockable OHCA and treated with mild hypothermia.[8] One retrospective cohort study of 8316 in-hospital cardiac arrest (IHCA) patients of any initial rhythm showed no difference in survival to hospital discharge among those who were treated with mild induced hypothermia compared with no active temperature management.[9]

One large RCT (the TTM trial, 939 patients) compared cooling to 33°C compared with tight temperature control at 36°C in adult patients with OHCA of any initial rhythm except unwitnessed asystole, and found no benefit in outcome (survival or neurological).[10]

The vast majority of the patients studied with induced hypothermia were from cardiac arrests due to presumed cardiac causes.

Recommendations

ANZCOR recommends TTM as opposed to no TTM for adults with out-of-hospital cardiac arrest (OHCA) with an initial shockable rhythm who remain unresponsive after ROSC (CoSTR 2015, strong recommendation, low-quality evidence).[11]

ANZCOR suggests TTM as opposed to no TTM for adults with OHCA with an initial non-shockable rhythm who remain unresponsive after ROSC (CoSTR 2015, weak recommendation, very low-quality evidence).[11]

ANZCOR suggests TTM as opposed to no TTM for adults with in-hospital cardiac arrest (IHCA) with any initial rhythm who remain unresponsive after ROSC (CoSTR 2015, weak recommendation, very low-quality evidence).[11]

ANZCOR recommends selecting and maintaining a constant target temperature between 32°C and 36°C for those patients in whom TTM is used. (CoSTR 2015, strong recommendation, moderate-quality evidence). Whether certain subpopulations of cardiac arrest patients may benefit from lower (32-34°C) or higher (36°C) temperatures remains unknown.[11]

No studies specifically addressed cardiac arrests due to non-cardiac causes, but it is reasonable to assume that these patients might also benefit from targeted temperature management. [Class B; Expert consensus opinion]

Values and preferences

We place a higher value on the potential for increased survival with good neurologic outcome as compared with the possible risks (which appear to be minimal) and the cost of TTM. We emphasize that the mortality after cardiac arrest is high and the treatment options are limited. Although the evidence for TTM compared with no temperature management is of low quality, it is the only post-ROSC intervention that has been found to improve survival with good neurologic outcome.[11]

2 How to cool?

Nineteen studies indicated that cooling could be initiated safely with intravenous ice-cold fluids (30 ml/kg of saline 0.9% or Ringer's lactate).[1] Six studies indicated that cooling with IV cold saline could be initiated in the prehospital phase.[1] Thirteen studies documented the use of an intravascular heat exchanger to induce and maintain hypothermia.[1] Twelve studies documented the use of ice packs and either water or air circulating blankets to induce and maintain hypothermia.[1]

Seven studies documented the use of ice packs (sometimes combined with wet towels) alone to induce and maintain hypothermia. Four studies documented the use of ice packs alone to maintain hypothermia. Seven studies documented the use of cooling blankets or pads alone to induce and maintain hypothermia.[1] Eight studies documented the use of water circulating gel-coated pads to induce and maintain, or just maintain, hypothermia.[1]

One randomized controlled trial used a cold air tent and another used a cooling helmet to induce and maintain hypothermia. In one registry study, cooling was maintained with ice-packs (17%), air cooling (8%), circulating water blankets (63%), an intravascular cooling device (16%) and other methods (8%).[1] More recently transnasal evaporative cooling and cooling by extracorporeal circulation have also been studied.[12-14]

There are currently no data indicating that any specific cooling technique increases survival when compared with any other cooling technique.

Studies that documented improved outcome with therapeutic hypothermia after cardiac arrest used continuous temperature monitoring.[1] Shivering may necessitate sedation and intermittent or continuous neuromuscular blockade. Use of continuous neuromuscular blockade could mask seizure activity.[1]

Recommendation

Rapid infusion of ice-cold intravenous fluid, up to 30 ml kg-1 or ice packs are feasible, safe and simple methods for initially lowering core temperature up to 1.5 degrees. When intravenous fluids are used to induce hypothermia additional cooling strategies will be required to maintain hypothermia.[1] [Class B; LOE III-3, IV]

3 When to cool?

One registry-based case series of 986 comatose post-cardiac arrest patients suggested that time to initiation cooling (median 90 min; interquartile range 60 -165 min) was not associated with improved neurological outcome post discharge.[15]

A case series of 49 consecutive comatose post-cardiac arrest patients who were intravascularly cooled after out-of-hospital cardiac arrest also documented that time to target temperature (median 6.8 hours [IQR 4.5 to 9.2 hours]) was not an independent predictor of neurologic outcome.[16]

5 trials (1,867 subjects) of OHCA showed no difference in neurologic outcomes after initiation of induced hypothermia in the pre-hospital environment compared with later initiation. Seven trials found no improvement in mortality for patients treated with pre-hospital cooling compared to those who did not receive pre-hospital cooling.[11]

Four RCTs showed an increased risk of re-arrest for those who received pre-hospital induced hypothermia. This result was driven by data from the largest trial (the TTM trial).[10]

Three trials reported no pulmonary oedema in any group. Two small pilot trials found no difference between groups, and one trial showed an increase in pulmonary oedema in patients who received pre-hospital cooling.[11]

Recommendation

ANZCOR recommends against routine use of pre-hospital cooling with rapid infusion of large volumes of cold intravenous fluid immediately after ROSC (CoSTR 2015, strong recommendation, moderate-quality evidence).[11]

Values and Preferences

Pre-hospital cooling has not demonstrated benefit despite a large number of patients studied, and there has been the suggestion of increased risk of re-arrest with pre-hospital induction of mild hypothermia using rapid infusion of cold intravenous fluids.

However, it is acknowledged that cold intravenous fluid might still be used in patients who have been further evaluated or in other settings.[11]

4 Duration of Cooling

No human trials have compared different durations of targeted temperature management after cardiac arrest. Published trials have treated patients with cooling for 12-28 hours and the TTM trial maintained strict normothermia (<37.5◦C) after hypothermia until 72 h after ROSC.[10] Two observational studies found no difference in outcome with 24 h compared with 72 h of hypothermia[17,18]

Recommendation

ANZCOR suggests that if TTM is used, duration should be at least 24 hours (CoSTR 2015, weak recommendation, very low-quality evidence).[11]

5 Safety with Percutaneous Coronary Intervention?

Five studies indicate that the combination of therapeutic hypothermia and primary percutaneous intervention was feasible and safe after cardiac arrest caused by acute myocardial infarction.[1]

Recommendation

ANZCOR suggests that percutaneous coronary intervention during TTM is feasible and safe and may be associated with improved outcome.[1] [Class B; LOE III-3, IV]

ANZCOR suggests institutions or communities planning to implement complex guidelines, such as targeted temperature management should consider using a comprehensive, multifaceted approach, including: clinical champions; a consensus-building process; multidisciplinary involvement; written protocols; detailed process description; practical logistic support; multi-modality, multi-level education; and rapid cycle improvement methods.[19] [Class B; Expert consensus opinion]

6 Avoidance of Fever after ROSC

In the absence of the use of targeted temperature management, five observational studies have demonstrated poor outcome with fever after ROSC.

When targeted temperature management has been used, six observational studies have not shown an association between fever after TTM and outcome (survival or neurological). However, two observational studies have demonstrated poorer outcome (survival or neurological) with fever after TTM.[11]

Recommendation

ANZCOR suggests prevention and treatment of fever in persistently comatose adults after completion of TTM between 32°C and 36°C (CoSTR 2015, weak recommendation, very low quality evidence).[11]

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AUSTRALIAN RESUSCITATION COUNCIL

NEW ZEALAND Resuscitation Council
Whakahauora, Aotearoa

GUIDELINE 11.10

RESUSCITATION IN SPECIAL CIRCUMSTANCES

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

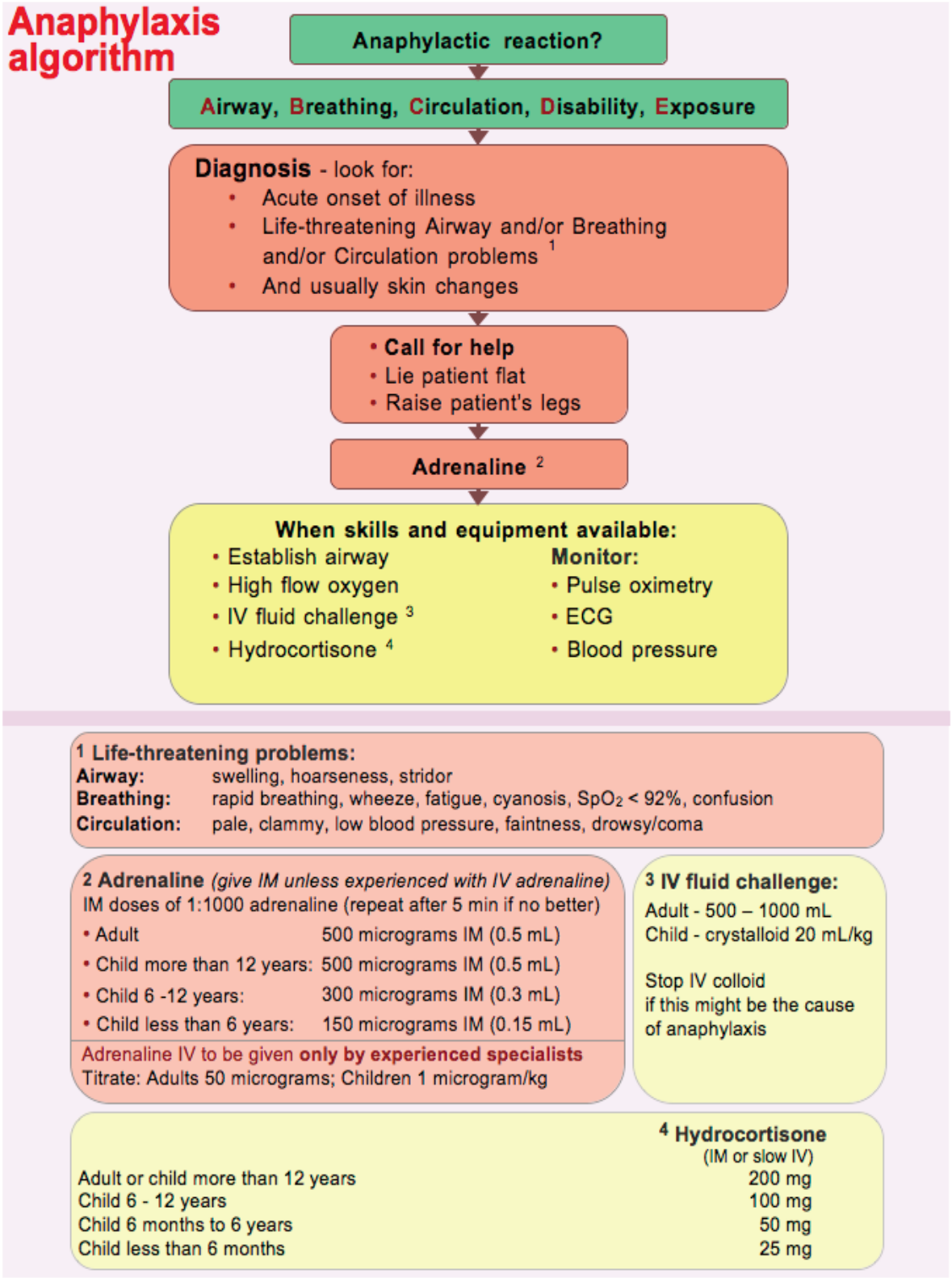
Anaphylaxis is a severe, life-threatening, generalised or systemic hypersensitivity reaction. This is characterised by rapidly developing life-threatening airway, and/or breathing and/or circulation problems usually associated with skin and mucosal changes. Airway obstruction may occur rapidly in severe 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. ß-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 randomized 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 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. [Class A, Expert Consensus Opinion]

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 optimized 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 [ie:. <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, 5mg with repeat dosing up to 15mg. [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 intra-osseous 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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AUSTRALIAN RESUSCITATION COUNCIL

NEW ZEALAND Resuscitation Council WHAKAHAUORA AOTEAROA

ANZCOR Guideline 11.10.1
Management of Cardiac Arrest due to Trauma

Summary

To whom does this guideline apply?
This guideline applies to adult and paediatric patients in cardiac arrest, or peri-arrest, due to physical trauma. Specific isolated traumatic mechanisms such as near-hanging and burns are not addressed.

Who is the audience for this guideline?
This guideline applies to first-aiders, prehospital clinicians and hospital teams.

Recommendations
The Australian and New Zealand Committee on Resuscitation (ANZCOR) recommends:

- Unless there are injuries obviously incompatible with life, attempted resuscitation of patients with cardiac arrest due to trauma is not futile and should be attempted.
- The first priority in peri-arrest trauma patients is to stop any obvious bleeding.
- Depending on the likely aetiology of the cardiac arrest, restoration of the circulating blood volume may have a higher priority than airway and breathing. If hypovolaemia is likely, an initial fluid bolus of 20mL/kg should be given as rapidly as possible.
- All patients in cardiac arrest with suspected chest trauma who are not responding to airway opening and restoration of circulating blood volume should have their chest decompressed.
- Surgical drainage of traumatic cardiac tamponade (with repair of cardiac laceration if required) is preferable to needle pericardiocentesis.
- Cardiac arrest due to penetrating trauma is more likely to respond to emergency thoracotomy than is true in blunt trauma. A favourable outcome is rarely possible (even in penetrating trauma) if thoracotomy is initiated more than 10 minutes after the onset of cardiac arrest.
- Cardiac arrest due to isolated head injury, crush syndrome and commotio cordis are special circumstances necessitating specific therapies.
- In cardiac arrest due to trauma, haemorrhage control, restoration of circulating blood volume, opening the airway and relieving tension pneumothorax should have priority over conventional cardiopulmonary resuscitation (CPR) (i.e. external chest compressions, defibrillation and adrenaline) unless a medical cause for cardiac arrest is reasonably suspected to have preceded the traumatic event. However, if there are sufficient resources available and this does not interfere with necessary procedures, conventional CPR should occur simultaneously.
- Prolonged (>10 minutes) CPR in traumatic cardiac arrest after reversible causes have been addressed is almost never associated with a good outcome.

Most deaths due to trauma occur in the first five minutes following the traumatic event, and most of these deaths cannot be prevented, even with skilled and timely treatment.

There are, however, three common causes of *preventable* early death in trauma:

- Airway obstruction
- Tension pneumothorax
- Haemorrhage

Post-mortem studies of military trauma suggest haemorrhage is responsible for around 60% of preventable deaths amongst patients who *actually died* following trauma, with 33% due to tension pneumothorax and 7% due to airway obstruction.[1] Civilian data is similar, but also identifies cardiac tamponade as the cause of traumatic cardiac arrest in 10% of patients.[2] These figures are often quoted with the suggestion that they should determine priorities for intervention in trauma, but this is misleading. Based on post-mortem studies, these figures do not account for patients who *would have died* were it not for *successful interventions* such as airway control. Opening the airway (in particular) is likely to be proportionately more important than these figures suggest.

While patients in cardiac arrest due to trauma have a worse prognosis than other cardiac arrest patients, several large series show survival after appropriate treatment is possible, with recent studies showing 5.1%[3], 6.6%[4], 7%[5] and 7.5%[6] survival to discharge, albeit with varying rates of neurological recovery (2%[5] to 6.6%[4]). Therefore, **unless there are injuries or wounds that are obviously incompatible with life (such as substantial body disruption), attempted resuscitation of patients with cardiac arrest due to trauma is not futile and should be attempted (Class A, LOE III). Conversely, prolonged (>10 minutes) cardiopulmonary resuscitation after reversible causes have been addressed is almost never associated with a good outcome.**

This guideline applies to first-aiders, prehospital clinicians and hospital teams managing trauma patients who are peri-arrest or in cardiac arrest. However, the equipment, skills and experience required to provide many of the interventions described will not exist in all circumstances. **Nothing in this guideline suggests that first-aiders or clinicians should work outside their scope of practice, or perform procedures for which they have insufficient training.** Particularly with respect to highly invasive procedures such as resuscitative thoracotomy, the risk to a patient from a procedure performed incorrectly or for a presumed indication that is, in reality, not present may be greater than not attempting such a procedure. **No fault should be attributed to clinicians who, by remaining within their scope of practice, do not perform one of the procedures this guideline recommends or lists for consideration.**

Guideline

1 Pre-Arrest: Stop the Bleeding

Trauma patients who are able to maintain their circulation may have injuries or wounds that will lead to cardiac arrest if not adequately treated. The first priority in such patients is to **stop the bleeding** using one of the techniques in ANZCOR Guideline 9.1.1 or (in hospital) more advanced interventions such as surgical exploration or interventional radiology. **Only after such interventions are commenced should attention be directed to the airway or breathing (Class A, LOE IV),** unless there are sufficient people to perform interventions simultaneously.

2 Airway

ANZCOR Guideline 11.6 describes basic and advanced techniques to open the airway. A further consideration in trauma is the possibility of cervical spine injury, the assessment and management of which is described in ANZCOR guideline 9.1.6.

A patient in cardiac arrest due to trauma should have the airway opened as quickly as possible while not delaying blood volume expansion and relief of possible tension pneumothorax. Chin lift / jaw thrust are preferred techniques but head tilt and / or positioning in the recovery position (likely to be associated with more cervical spine movement) may be required. **(Class B, LOE IV).**

Direct trauma to the airway may contraindicate usual supraglottic approaches. Therefore, insertion of an endotracheal, tracheostomy or purpose-designed cricothyroidotomy tube through the cricothyroid membrane or trachea may be required. Whether these procedures should take priority over restoration of the circulating blood volume will depend on the likely principal cause of the cardiac arrest or impending arrest.

3 Restoration of Circulating Blood Volume

Depending on the condition of the patient and the likely aetiology of the cardiac arrest, restoration of the circulating blood volume may be the highest priority for patients in cardiac arrest due to trauma.

3.1 Intravenous or intraosseous access

Intravenous or intraosseous access should be established as rapidly as possible. If peripheral intravenous cannulation can be accomplished prior to cardiac arrest, ideally two cannulae of at least 18G should be inserted **(Class A, LOE IV).**

Peripheral venous cannulation is likely to be impossible in cardiac arrest due to exsanguination, regardless of whether or not cardiac compressions are being performed. In adults, central venous cannulation may be possible and should be attempted using a Seldinger technique and a short, large-bore catheter (such as a catheter-introducer for a pulmonary artery catheter or a dialysis catheter) **(Class B, LOE IV).** Small-bore central venous catheters are not recommended due to their low maximal flow rates. One study found subclavian central venous cannulation more likely to be successful (success in 94% of attempts) than femoral venous cannulation (success in only 77%)[7]. In young children, intraosseous (IO) access is preferred to central venous access.

IO access may be more rapidly and reliably achieved than venous access, especially by clinicians inexperienced with central venous catheter insertion, and especially in children. In adults, IO cannulae in the sternum or humeral head achieve more rapid fluid administration than those in the medial proximal tibia.[8] In children, IO cannulae should be placed in either the humeral head or medial proximal tibia. Two IO access devices may be warranted to increase the maximum possible rate of fluid infusion. The choice between central IV, peripheral IV and IO access will depend on the condition of the patient and the equipment and personnel available; there is no evidence-based threshold time at which attempted IV access should be abandoned in favour if IO access. When sufficient clinicians are available, simultaneous approaches are suggested.

3.2 Fluid therapy

If hypovolaemia is a possible cause or contributor to traumatic cardiac arrest, an initial (ideally warmed) fluid bolus of 20mL/kg (approximately 1.4L in a 70kg patient) should be given as rapidly as possible **(Class B; LOE IV).** In exsanguinating haemorrhage, this should be a 1:1 or 1:2 mixture of thawed (**Class B; LOE III**) thawed fresh-frozen plasma : packed red blood cells.[9,10] Use of other blood products (such as cryoprecipitate and platelets) should be guided by an institutional Massive Transfusion Protocol informed by consensus guidelines,[9] and in particular should avoid dilution of platelets and fibrinogen. Several ambulance services have shown that prehospital administration of packed red blood cells and plasma is feasible. Administration of large volumes of packed red blood cells anticoagulated with citrate will reduce plasma ionised calcium, necessitating replacement with calcium gluconate or calcium chloride, ideally titrated to measured levels.

If blood products are not available, a crystalloid solution should be used. Blood products should be substituted for crystalloid as soon as possible.

Further fluid boluses of 5-10mL/kg should be given if hypovolaemia is suspected as the ongoing cause of persistent cardiac arrest. **(Class B; LOE IV).**

In adults, once spontaneous cardiac output is restored, prior to surgical haemorrhage control and at least for the first hour, further fluid should be titrated either to a systolic blood pressure of 90mmHg (permissive hypotension) or to consciousness. **(Class B; LOE IV).** Due to the risk of raised intracranial pressure reducing cerebral perfusion pressure, a patient with substantial trauma to the head may benefit from a target systolic blood pressure of 110mmHg. **(Class B; LOE IV).** The optimal endpoint of fluid resuscitation after the first hour or after surgical haemorrhage control is unclear; however, progressive improvements in plasma lactate and base deficit are reasonable targets in the absence of advanced haemodynamic monitoring. Achieving such targets may require a higher target systolic blood pressure than during the first hour hypotensive resuscitation phase. Acidosis should not be corrected with IV sodium bicarbonate (**Class B; LOE IV**) unless it is used as a specific treatment for hyperkalaemia, as (for example) in crush syndrome. In children, there is insufficient evidence for or against a strategy of permissive hypotension during the first hour of resuscitation. Resuscitation targeting a low to normal (for age) systolic pressure for age is recommended in the absence of such evidence.

3.3 Haemorrhage control

A patient in traumatic cardiac arrest will have little active bleeding. However, bleeding may resume upon restoration of circulation. Therefore, during the resuscitation phase but without delaying the other effective interventions listed here, the haemorrhage control techniques listed in ANZCOR Guideline 9.1.1, such as direct wound pressure, proximal arterial tourniquets, pelvic binders, junctional haemorrhage devices and haemostatic dressings, should be applied.

4 Chest Decompression

All patients in cardiac arrest with suspected chest trauma who are not responding to airway opening and restoration of circulating blood volume should have their chest decompressed as described below. **(Class B; LOE IV).** Resuscitative thoracotomy may be indicated in limited situations.

Finger thoracostomy (initially on the most affected side of the chest) is the preferred method of chest decompression. Finger thoracostomy involves incising 3-4cm of skin over the 4th intercostal space just anterior to the mid-axillary line followed by blunt dissection to the pleura to allow introduction of a finger into the pleural space. This should be followed by insertion of an intercostal catheter if available. Intercostal catheters should be connected to underwater seal drains or one-way valves. If an intercostal catheter is not available, an adhesive one-way chest seal is acceptable. The same procedure should be performed on the contralateral side if there is suspicion of bilateral tension pneumothoraces. If more than 1000mL blood drains immediately, or there is ongoing bleeding >200mL/hr for the subsequent 2-4 hours, surgical exploration is warranted. **(Class B; LOE IV).** Finger thoracostomies should be accompanied by intubation and mechanical ventilation because of the risk of inadequate ventilation due to air entering the pleural space. **(Class B; LOE IV).**

An alternative to finger thoracostomy that may allow more rapid chest decompression in some circumstances is insertion of a long, large bore (ideally 8cm, 12- or 14-gauge) cannula into the pleural cavity. The optimal location for cannula insertion is unknown, and probably depends on individual body habitus.

Options include the second intercostal space at or just lateral to the midclavicular line, with the needle directed away from the heart, or alternatively the 4th or 5th intercostal space just anterior to the mid-axillary line. **(Class B; LOE IV).** If resuscitation continues, needle chest decompression must always be followed by insertion of an intercostal catheter.

5 Pericardiocentesis

The commonest cause of pericardial tamponade due to trauma is a penetrating injury or wound to the myocardium, which will require surgical intervention via thoracotomy. Urgent bedside echocardiography should be used to identify or exclude pericardial tamponade due to trauma **(Class B; LOE IV)**.

Needle pericardiocentesis is almost never the optimal means of decompressing the pericardium in trauma, as it does not address the commonest cause (myocardial laceration) and because pericardial blood is often clotted, preventing aspiration. However, when no surgeon or other clinician with the required skills and experience is present to surgically manage the patient, needle pericardiocentesis (ideally under ultrasound guidance) can be attempted in a patient who is peri-arrest or in cardiac arrest with a high suspicion of cardiac tamponande **(Class B; LOE IV).**

6 Resuscitative Thoracotomy

A resuscitative thoracotomy can:

- release tension pneumothorax or cardiac tamponade;
- allow direct control of intrathoracic haemorrhage;
- allow cross-clamping the descending aorta (in so doing stopping blood loss below the diaphragm and improving brain and cardiac perfusion); and
- permit open cardiac compression and defibrillation.

In one case series, resuscitative thoracotomy was successfully performed by adequately trained and experienced prehospital clinicians.[6] However, in general in the Australian and New Zealand civilian context this will remain a hospital intervention performed by a surgeon or a specifically trained and experienced emergency or critical care physician. Whether a clam-shell or anterolateral approach is best will be determined by the pattern of injury or wounding and the available surgical expertise. The decision on proceeding with resuscitative thoracotomy will rest on the mechanism of injury or wounding, whether there is likely to be a surgically-correctable problem given the expertise and resources available, and the duration since the traumatic event and the onset of cardiac arrest. Ideally, hospitals should develop local guidelines relevant to their institution.

As a general guide, cardiac arrest due to penetrating trauma is more likely to respond to emergency thoracotomy than cardiac arrest due to blunt trauma. A favourable outcome is rarely possible (even in penetrating trauma) if resuscitative thoracotomy is initiated more than 10 minutes after the onset of cardiac arrest. **(Class A; LOE IV).**

A resuscitative thoracotomy may be indicated prior to cardiac arrest – if time allows, ideally by an appropriately trained surgical team in an adequately-equipped operating theatre.

7 Special Circumstances

7.1 Crush syndrome

ANZCOR Guideline 9.1.7 outlines the recommended early management of a patient with crush injury. In addition to the Basic Life Support (BLS) measures outlined in that guideline, Advanced Life Support (ALS) for a crushed patient in cardiac arrest or peri-arrest should consider the possibility of hyperkalaemia as a contributing aetiology. ANZCOR recommends urgent check of the serum potassium level if possible, with urgent treatment indicated for potassium levels >6.5 mmol/L. ECG changes (peaked T waves, loss of P waves, prolonged QRS interval, or a "sine wave" appearance) prior to cardiac arrest also suggest hyperkalaemia. In the absence of diagnostic tests, empiric treatment on the basis of trauma mechanism alone may be required. Treat a crushed patient with cardiac arrest due to hyperkalaemia with:

- Calcium chloride 10% 5-10mL or calcium gluconate 10% 10-20mL IV. (weight adjust for children);
- Glucose 50% 50mL and (simultaneously) insulin 10 units IV (weight adjust for children); and/or
- Sodium bicarbonate 1mmol/kg IV.

(Class A; LOE IV).

7.2 Direct cardiac trauma resulting in commotio cordis

Commotio cordis occurs when a force applied to the anterior chest precipitates cardiac arrhythmia, most commonly ventricular fibrillation. The most common precipitant is being struck by a hard ball during sport. Cardiac arrest due to commotio cordis is greatest when a force is applied at 65 kilometres per hour,[11] with the chance of structural heart damage increasing at greater velocities. Suspected traumatic cardiac arrest due to commotio cordis should be managed according to the general principles for CPR outlined in ANZCOR Guideline 11.2, with early defibrillation of shockable rhythms accorded the same high priority **(Class A; LOE IV).**

7.2 Isolated major head injury

Isolated traumatic brain injury (TBI) without substantial structural brain pathology has been noted in case series and animal models occasionally to cause apnoea associated with a transient catecholamine surge followed by cardiovascular collapse.[12] Several unconscious apnoeic TBI patients with preserved spontaneous circulation have reportedly been resuscitated, with complete neurological recovery, by means of artificial respiration alone. Whether spontaneous respiration might have resumed prior to hypoxic cardiac arrest in these patients remains speculative. However, artificial respiration of an apnoeic patient requires little justification. In the absence of other injuries, such patients should be managed according to the general principles for CPR outlined in ANZCOR Guideline 11.2.

8 Conventional Basic and Advanced Life Support

A small minority of patients will have a medical cause that precipitates cardiac arrest that is followed by trauma: for example, in drivers involved in road traffic accidents. If the severity of the trauma mechanism or injuries observed appear insufficient to have caused cardiac arrest, a medical cause (such as myocardial infarction) should be suspected and managed according to the general principles for CPR outlined in ANZCOR Guidelines 6 and 11.2.

In cardiac arrest due to trauma, all of the interventions aimed at addressing underlying causes take priority over chest compressions, defibrillation and adrenaline. However, if there are sufficient resources available and there is no interference with essential procedures, conventional CPR can occur simultaneously. The effectiveness of conventional CPR will depend on correcting the causes of the cardiac arrest.

8.1 External Chest Compressions

An exsanguinated patient theoretically derives little benefit from external cardiac compressions until blood volume is restored to a minimally sufficient quantity. External chest compressions may exacerbate haemorrhage and cardiac tamponade, and positive pressure ventilation may further reduce critically low venous return or cause air embolism.[13] Several case series demonstrate that external chest compressions are virtually never effective for patients in traumatic cardiac arrest unless the underlying cause of the arrest is simultaneously and rapidly addressed.[13-16] Conversely, there is no clinical evidence that chest compressions worsen outcome in trauma. Therefore, external chest compressions (as recommended in ANZCOR Guideline 6) should be commenced as a secondary priority, after airway opening, commencement of restoration of circulating blood volume (if the required equipment is available), and (if appropriate) decompression of the chest **(Class B; LOE IV)**. If there is any other indication for resuscitative thoracotomy and this is possible in the circumstances, internal cardiac compression is preferable to external chest compressions.[17] **(Class A; LOE IV).**

In the absence of the requisite equipment or expertise to address the underlying aetiology of cardiac arrest in trauma, first aiders should summon skilled assistance then proceed directly to BLS.

8.2 Adrenaline

There is little evidence for or against the use of adrenaline in cardiac arrest due to trauma. Retrospective observational studies have found vasopressor use associated with worse outcome in haemorrhagic shock,[18,19] but conversely, a large case series found patients in established traumatic cardiac arrest had a higher chance of survival to hospital discharge if they were treated with adrenaline.[20] Such studies are almost certainly confounded by residual indication bias despite attempts at adjustment for severity of injury, and there have been no prospective clinical trials. Guidance is therefore based on extrapolation from physiological principles.

ANZCOR does not recommend adrenaline for patients in traumatic cardiac arrest until haemorrhage control, opening the airway, commencement of restoration of circulating blood volume and (if appropriate) decompression of tension pneumothorax have been addressed. **(Class A; LOE IV).**

Once spontaneous cardiac output is restored, hypotension is usually the result of hypovolaemia and should be treated initially with ongoing volume replacement. **(Class B; LOE IV).** In the later phases of post-arrest care, vasodilation or myocardial depression may require adrenaline or other vasoactive infusions.

8.3 Defibrillation

Only 7.5% of patients in traumatic cardiac arrest are initially found in VF or VT.[21] Therefore defibrillation is not the priority for the majority of trauma patients in cardiac arrest. ANZCOR suggests not using defibrillation prior to opening the airway, commencement of restoration of circulating blood volume, and (if appropriate) decompression of the chest **(Class B; LOE IV).** In the small minority of patients who are in traumatic cardiac arrest with VF or VT, reversible causes must be addressed, and defibrillation should be performed promptly, especially in those suspected of having a cardiac co-morbidity. **(Class A; LOE IV).**

8.4 Four Hs and Four Ts

ANZCOR Guideline 11.2 recommends consideration of "Four Hs and four Ts" (hypoxaemia, hypovolaemia, hyper/hypokalaemia and other metabolic disorders, hypo/hyperthermia, tension pneumothorax, tamponade, toxins, and thrombosis – pulmonary / coronary) in any patient in cardiac arrest, particularly those in asystole. Following this cardiac arrest due to trauma guideline will identify and treat all these conditions, with the exceptions of hypo/hyperthermia, toxins and thrombosis. These relatively infrequent causes should be considered in a patient who has not responded to other interventions. **(Class A, LOE IV).**

9 Transport to Hospital

Several potentially effective interventions (such as resuscitative thoracotomy) may only be available in hospital, suggesting speed of transport should be prioritised over time taken performing prehospital interventions. Conversely, the case series demonstrating the highest survival rates after traumatic cardiac arrest reported on protocols that mandated not leaving the incident scene until spontaneous circulation was restored.[4] On the basis of the best available evidence, ANZCOR suggests that patients in cardiac arrest due to trauma should only be transported to hospital after return of spontaneous circulation, unless the hospital is in such close proximity that a patient thought to require an emergency room thoracotomy would have a realistic chance of this occurring within 10 minutes.

Patients who have a cardiac arrest due to trauma during transportation should have the interventions described in this guideline, noting that those who have no spontaneous circulation for >10 minutes are unlikely to survive.

In the current Australian and New Zealand civilian context, a prehospital resuscitative thoracotomy will rarely, if ever, be appropriate. **(Class B; LOE IV).**

10 Terminating Attempted Resuscitation

There is no consensus on how long resuscitation attempts should continue after cardiac arrest due to trauma. Most case series of traumatic cardiac arrests do not report informative time data. Restoration of a circulating blood volume sufficient to sustain spontaneous circulation may take several minutes, depending on the equipment and venous access available. In the absence of data, ANZCOR recommends continuation of BLS or ALS (including external cardiac compressions) for up to 10 minutes after these potentially reversible causes have been addressed, following which resuscitation attempts should be stopped if there is no ROSC. **(Class A; LOE IV).**

11 Debriefing

The circumstances of the traumatic event and the physical appearance of the patient can be more distressing to first aiders and clinicians than might be the case for victims of cardiac arrest due to non-traumatic causes. Sensitive, professional debriefing of people involved in resuscitation efforts is likely to have value.

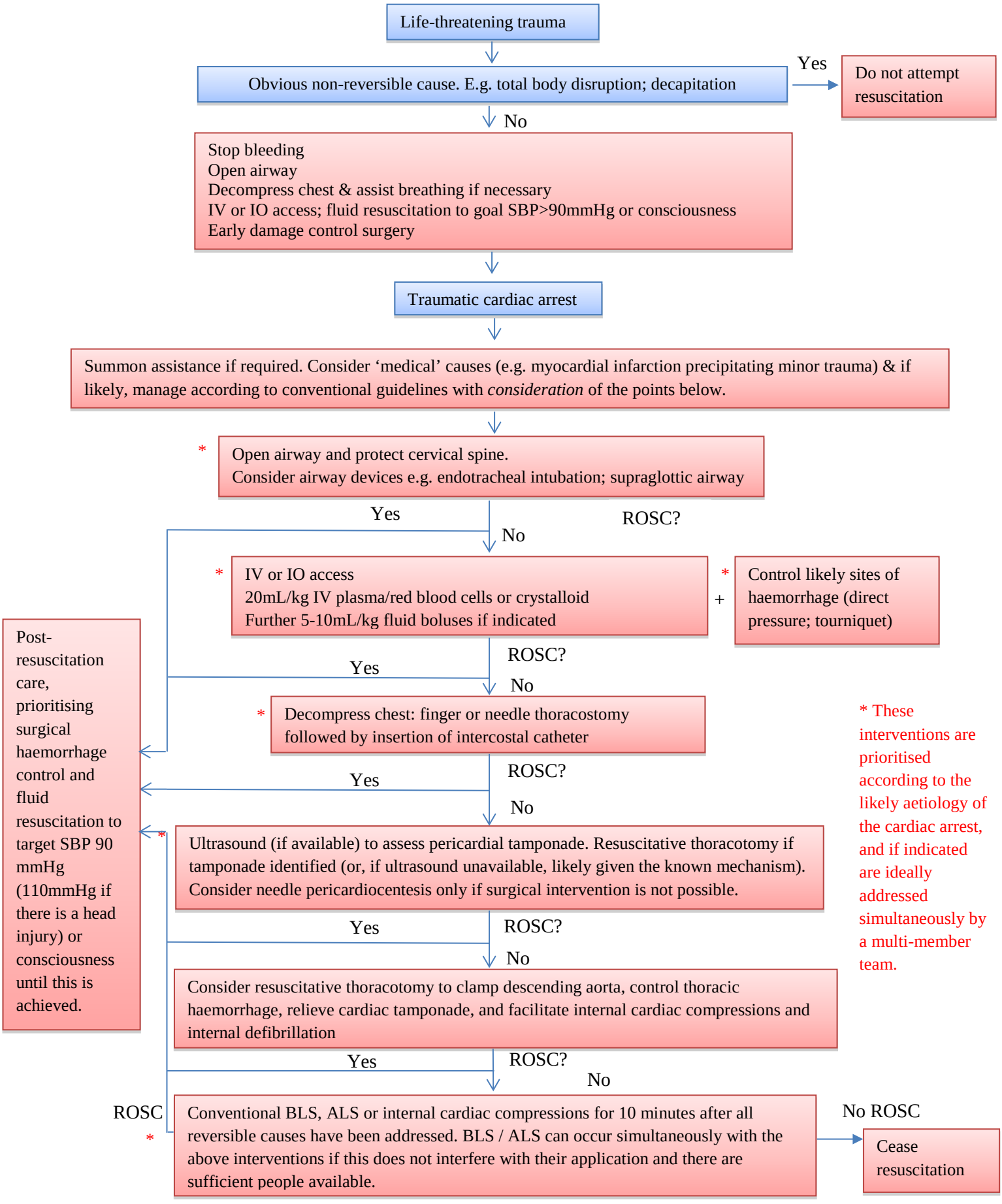
12 Advice to First-Aiders

Many of the potentially beneficial interventions for patients in cardiac arrest due to trauma will only be possible for trained prehospital clinicians or hospital personnel with the required equipment. Speed of ambulance arrival to a patient in traumatic cardiac arrest is a strong predictor of survival. Therefore, **first-aiders and clinicians lacking necessary equipment should prioritise calling for skilled help over attempting BLS (Class A; LOE IV).** Conversely, first-aiders can apply simple interventions that might effectively prevent the progression to cardiac arrest after trauma – in particular, opening the airway (ANZCOR Guideline 4) and stopping the bleeding (ANZCOR Guideline 9.1.1) **(Class A; LOE IV).**

Further reading

ANZCOR Guideline 4 Airway
ANZCOR Guideline 9.1.1 Principles for the Control of Bleeding for First Aiders
ANZCOR guideline 9.1.6 Management of Suspected Spinal Injury
ANZCOR Guideline 9.1.7 Emergency Management of a Crushed Victim
ANZCOR Guideline 11.2 Protocols for Adult Advanced Life Support
ANZCOR Guideline 11.6 Equipment and Techniques in Adult Advanced Life Support

Summary


Life-threatening trauma
Obvious non-reversible cause. E.g. total body disruption; decapitation
Yes
Do not attempt resuscitation
No
Stop bleeding
Open airway
Decompress chest & assist breathing if necessary
IV or IO access; fluid resuscitation to goal SBP>90mmHg or consciousness
Early damage control surgery
Traumatic cardiac arrest
Summon assistance if required. Consider 'medical' causes (e.g. myocardial infarction precipitating minor trauma) & if likely, manage according to conventional guidelines with consideration of the points below.
* Open airway and protect cervical spine.
Consider airway devices e.g. endotracheal intubation; supraglottic airway
Yes
ROSC?
No
* IV or IO access
20mL/kg IV plasma/red blood cells or crystalloid
Further 5-10mL/kg fluid boluses if indicated
+
* Control likely sites of haemorrhage (direct pressure; tourniquet)
ROSC?
Yes
No
* Decompress chest: finger or needle thoracostomy followed by insertion of intercostal catheter
ROSC?
Yes
No
* Ultrasound (if available) to assess pericardial tamponade. Resuscitative thoracotomy if tamponade identified (or, if ultrasound unavailable, likely given the known mechanism). Consider needle pericardiocentesis only if surgical intervention is not possible.
ROSC?
Yes
No
Consider resuscitative thoracotomy to clamp descending aorta, control thoracic haemorrhage, relieve cardiac tamponade, and facilitate internal cardiac compressions and internal defibrillation
ROSC?
Yes
No
ROSC
*
Conventional BLS, ALS or internal cardiac compressions for 10 minutes after all reversible causes have been addressed. BLS / ALS can occur simultaneously with the above interventions if this does not interfere with their application and there are sufficient people available.
No ROSC
Cease resuscitation
Post-resuscitation care, prioritising surgical haemorrhage control and fluid resuscitation to target SBP 90 mmHg (110mmHg if there is a head injury) or consciousness until this is achieved.
* These interventions are prioritised according to the likely aetiology of the cardiac arrest, and if indicated are ideally addressed simultaneously by a multi-member team.

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