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.

Summary of Recommendations

This Australian and New Zealand Committee on Resuscitation (ANZCOR) guideline has been reviewed and updated based on evidence reviews including the 2018, 2019, 2020, 2022, 2023, 2024 and 2025 International Liaison Committee on Resuscitation (ILCOR) Advanced Life Support (ALS) Taskforce evidence reviews.[1-7] ANZCOR makes the following recommendations:

1. Intravenous (IV) access is the preferred means of administering medications to patients during or after cardiac arrest, when compared to intraosseous (IO) access.
2. If IV access cannot be rapidly achieved within two attempts, it is reasonable to consider IO access as an alternative during adult cardiac arrest.
3. For non-shockable rhythms (pulseless electrical activity/asystole), ANZCOR recommends administration of 1mg adrenaline (epinephrine) as soon as feasible during cardiopulmonary resuscitation (CPR) (and then every second loop of the ALS algorithm).
4. For shockable rhythms (ventricular fibrillation (VF)/pulseless ventricular tachycardia (pVT)), ANZCOR suggests administration of 1mg adrenaline (epinephrine) after initial defibrillation attempts are unsuccessful during CPR (after 2nd shock and after every second loop of the ALS algorithm).
5. ANZCOR suggests against the administration of vasopressin in place of adrenaline (epinephrine) during CPR.
6. ANZCOR suggests against the addition of vasopressin to adrenaline (epinephrine) during CPR.

7. ANZCOR suggests against the use of the combination of vasopressin and corticosteroids in addition to usual care for adults in cardiac arrest.
8. ANZCOR suggests administration of 300mg amiodarone for shock-refractory VF/pVT during CPR (after 3rd shock, if still refractory an additional dose of 150mg may be considered after the 5th shock).
9. ANZCOR suggests 1mg/kg lidocaine (lignocaine) may be used as an alternative to amiodarone in shock-refractory VF/pVT (if still refractory an additional dose of 0.5mg/kg may be considered).
10. ANZCOR recommends against routine administration of calcium for the treatment of out-of-hospital cardiac arrest (OHCA) and suggests against routine administration of calcium for treatment of in-hospital cardiac arrest (IHCA).
11. Calcium, magnesium, potassium, sodium bicarbonate (and other buffers) may be considered to help manage particular conditions that are associated with patients who have arrested.
12. Fibrinolytics should not be used routinely in cardiac arrest but may be used when pulmonary embolus is the suspected cause of cardiac arrest.

Guideline

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.

1.0 | 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 may 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 to 30mL 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 [Good Practice Statement].

1.2 | Intraosseous (IO) route

Intraosseous (IO) is the preferred route if IV access is not available. Doses and delivery are the same as the IV route. IO access is effective for fluid resuscitation, drug delivery and laboratory evaluation, and is attainable in all age groups. If IV access cannot be established, IO delivery of resuscitation drugs will achieve adequate plasma concentrations.[8] A number of devices are now available for use in adults[9] [Good Practice Statement].

1.3 | Intravenous (IV) route versus Intraosseous (IO) route

ILCOR identified new evidence from 3 randomised controlled clinical trials published in 2024 that were included in a systematic review during the 2025 CoSTR review process.[10]

Recommendations

ANZCOR suggests IV access is the preferred means of administering medications to patients during or after cardiac arrest, when compared to IO access [CoSTR 2025, weak recommendation, low certainty evidence].

If IV access cannot be rapidly achieved within two attempts, it is reasonable to consider IO access as an alternative during adult cardiac arrest [Good Practice Statement].

1.4 | 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 IV dose at least 3 times is 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% sodium chloride solution 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 by the endotracheal route as they may cause mucosal and alveolar damage. This route cannot be used if a supraglottic airway is present [Good Practice Statement].

1.5 | Intracardiac injection

Intracardiac injection is **not** recommended because of the limited benefit and the high risk of complications [Good Practice Statement].

2.0 | Drugs and Order of Drug Administration

It is recognised that most studies assessing the effects of drugs on survival have not been able to control for the quality of CPR. Furthermore, many drug evaluations were conducted before recent advances in post-cardiac arrest care, including temperature control. Since most drug trials have, at most, demonstrated only short-term outcome advantage, it is important to evaluate long-term outcome when these drugs are combined with optimized post-cardiac arrest care.

2.1 | Vasopressors

ILCOR and ANZCOR have previously reviewed the use of vasopressors[8,11,12] during cardiac arrest and the ILCOR ALS Task Force targeted another update after the 2018 publication of a large RCT on the use of epinephrine in OHCA.[13]

ILCOR commissioned a systematic review and meta-analysis[14] which the ALS Task Force analysed for the 2019 CoSTR.[2] An update of this review was included in the 2025 CoSTR.

2.1.1 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.

Adrenaline (epinephrine) compared with placebo

For the comparison of adrenaline (epinephrine) with placebo, there was evidence from 2 randomised control trials (RCTs) with a total of 8500 OHCA patients; there were no RCTs for IHCA. The UK PARAMEDIC2 trial[13] of 8000 patients with OHCA, and the Australian PACA trial (Placebo-Controlled Trial of Adrenaline in Cardiac Arrest)[15] of 500 patients were combined in a meta-analysis.[14] The meta-analysis found no benefit in favourable neurological outcome at discharge but showed higher rates of survival to discharge, survival to admission, and return of spontaneous circulation (ROSC) in the adrenaline (epinephrine) group. In the subgroup of patients with non-shockable rhythms, combined evidence from the two RCTs showed benefit of adrenaline (epinephrine) for survival to discharge. There was no difference in survival to discharge with favourable neurological outcome (low certainty evidence). In the subgroup of patients with shockable rhythms, combined evidence from the two RCTs showed benefit of adrenaline (epinephrine) for ROSC but no difference for survival to discharge (moderate certainty evidence). The 2025 updated review was consistent with previous findings.[7]

2.1.2 Vasopressin

This is a hormone released from the posterior pituitary and has two primary functions. Vasopressin increases the amount of water reabsorbed from the kidney tubules and also constricts arterioles, which increases peripheral vascular resistance and blood pressure.

Vasopressin compared with adrenaline (epinephrine)

Three RCTs (all published >10 years ago) with >1500 patients with OHCA compared vasopressin with adrenaline (epinephrine).[16-18]

The combined results showed no benefit of vasopressin compared with adrenaline (epinephrine) across all outcomes and initial rhythms.

Only one study has examined the use of vasopressin compared with adrenaline (epinephrine) for IHCA and there was no statistically significant benefit from the administration of vasopressin compared with adrenaline (epinephrine) for IHCA.[19]

Initial adrenaline (epinephrine) plus vasopressin compared with (epinephrine) only

Four RCTs have compared adrenaline (epinephrine) plus vasopressin with adrenaline (epinephrine) alone.[20-23] The combined results of these studies showed no benefit across all outcomes and initial rhythms.

2.1.3 Vasopressin and Steroids

The use of vasopressin with steroids during cardiac arrest was prioritised for review by the ALS Task Force after the publication of an RCT.[24] A subsequent systematic review and meta-analysis,[25] was identified as suitable for adolopment for the 2022 CoSTR.[4]

Three RCTs were identified, all of which included patients with IHCA only, and compared the effect of the addition of vasopressin and steroids to standard cardiac arrest care.[24,26,27]

In-Hospital Cardiac Arrest. One of the included trials,[24] which enrolled 501 patients, assessed health-related quality of life at 90 days measured by the EuroQol 5 Dimension 5 Level tool. Data were available from all 44 patients who survived to 90 days, and there was no difference in the EuroQol 5 Dimension 5 Level score.

For the critical outcomes of favourable functional outcome at hospital discharge, and survival to discharge, there was low certainty evidence from all 3 RCTs including 869 patients, suggesting a possible small beneficial effect – OR 1.64 (95% CI 0.99-2.72) and 1.39 (95% CI 0.90-2.14) respectively.

For the important outcome of ROSC, there was moderate certainty of evidence from all 3 RCTs of a positive effect – OR 2.09 (95% CI 1.54-2.84).

Out-of-Hospital Cardiac Arrest. No evidence specific to OHCA was identified. Therefore, all the results for this population were the same as those for IHCA, with the evidence downgraded for

indirectness for the OHCA population.

Recommendations

ANZCOR recommends administration of adrenaline (epinephrine) during CPR [CoSTR 2025, strong recommendation, low certainty of evidence].

For non-shockable rhythms (pulseless electrical activity (PEA)/asystole), ANZCOR recommends administration of adrenaline (epinephrine) as soon as feasible during CPR (and then every second loop) [CoSTR 2025, strong recommendation, very low certainty of evidence].

For shockable rhythms (VF/pVT), ANZCOR suggests administration of adrenaline (epinephrine) after initial defibrillation attempts are unsuccessful during CPR (after 2nd shock and subsequently after every second loop) [CoSTR 2025, weak recommendation, very low certainty of evidence].

ANZCOR suggests against the administration of vasopressin in place of adrenaline (epinephrine) during CPR [CoSTR 2025, weak recommendation, very low certainty of evidence].

ANZCOR suggests against the addition of vasopressin to adrenaline (epinephrine) during CPR [CoSTR 2025, weak recommendation, low certainty of evidence].

ANZCOR suggests against the use of the combination of vasopressin and corticosteroids in addition to usual care for adults with IHCA because of low confidence in effect estimates for critical outcomes [CoSTR 2022, weak recommendation, low to moderate certainty evidence].

ANZCOR suggests against the use of the combination of vasopressin and corticosteroids in addition to usual care for adults with OHCA [CoSTR 2022, weak recommendation, very low to low certainty evidence].

Adrenaline (epinephrine) dose

There is limited evidence to suggest an optimal dose of any vasopressor in the treatment of adult cardiac arrest. The 2 OHCA RCTs comparing epinephrine with placebo that were evaluated for the 2019 CoSTR used standard-dose epinephrine (1mg IV or IO every 3 to 5 minutes). Although this CoSTR did not separately evaluate high-dose epinephrine because no new evidence was found, a previous ILCOR review did not find evidence of a survival benefit for high-dose epinephrine. Thus, the evidence to date supports the “standard” dosing used in the 2 RCTs included in the 2019 meta-analysis.

The initial adult dose is 1mg (1mL of 1:1,000 or 10mL of 1:10,000) and should be repeated at regular intervals (every 2nd loop of the ALS algorithm, which equates to approximately 4 minutes) during CPR. Adrenaline (epinephrine) may be required in repeated small doses or by infusion to produce an adequate blood pressure after ROSC. In this situation adrenaline (epinephrine) by infusion (1 to 20microgram/min*)* may be administered via a peripheral line, but delivery should be changed to a dedicated central line as soon as possible. There are special circumstances (e.g. post-operative cardiac surgery) where standard dosing may not be appropriate and may be modified.

Adverse effects

- Tachyarrhythmia
- Severe hypertension after resuscitation
- Tissue necrosis if extravasation occurs

ANZCOR acknowledges the identified knowledge gaps related to the following:

- The long-term neurological benefit of adrenaline (epinephrine) in cardiac arrest.
- The optimal dose of adrenaline (epinephrine) and dosing interval.
- The use and optimal timing of adrenaline (epinephrine) administration in patients with shockable rhythms.
- The use of adrenaline (epinephrine) for IHCA.
- The cost-effectiveness of adrenaline (epinephrine).
- The effect of different routes of administration (IV versus IO).
- The effect of increased ROSC on organ donation.
- Effective therapies to prevent or mitigate against neurological injury associated with cardiac arrest.

2.2 | Antiarrhythmic drugs

Antiarrhythmic drugs have a potential role in the treatment of VF or pVT that is refractory to defibrillation. The reported incidence of adult VF/pVT cardiac arrest varies, across Australia and New Zealand it has been reported in around 30% of cardiac arrest patients.[28]

ILCOR and ANZCOR have reviewed the use of antiarrhythmic drugs during cardiac arrest many times, and the ILCOR ALS Task Force prioritized an evidence update on the role of antiarrhythmic drugs in 2018 after publication of a RCT comparing amiodarone, lidocaine (lignocaine), and placebo.[29]

ILCOR commissioned a systematic review[30] which the ALS Task Force analysed for the 2019 CoSTR.[2] This review was updated for the for the 2023, 2024 and 2025 CoSTRs.

Use of antiarrhythmic drugs during resuscitation of adults with VF/pVT cardiac arrest or immediately after ROSC

Consensus on Science

The systematic review identified comparative data on the use of amiodarone versus placebo, lidocaine (lignocaine) versus placebo, amiodarone versus lidocaine (lignocaine), magnesium versus placebo, bretylium versus placebo, lidocaine (lignocaine) versus bretylium, amiodarone versus nifekalant, lidocaine (lignocaine) versus nifekalant, and lidocaine (lignocaine) versus sotalol.

Amiodarone versus placebo

Combined evidence from two RCTs -ARREST[31] and ROC-ALPS[29] trials comparing amiodarone with placebo for OHCA showed, with very low certainty of evidence, no statistically significant difference in survival to hospital discharge with good neurological outcome, survival to hospital discharge, or ROSC. The ROC-ALPS trial enrolled patients from 2013 to 2015, while the ARREST trial enrolled from 1994 to 1997, and the contemporary resuscitation practices changes over time was a significant confounder. An additional confounder resulted from the fact that the placebo groups in both trials received polysorbate 80, which is not inactive. The ARREST trial compared Cordarone (amiodarone in polysorbate 80) with a polysorbate 80 placebo. This study showed no statistically significant difference in survival to hospital discharge with good neurological outcome, or survival to hospital discharge, but it did show a statistically significant increase in ROSC. The ROC-ALPS trial, compared the Nexterone preparation of amiodarone with a saline placebo, and showed no statistically significant difference in survival to hospital discharge with good neurological outcome, survival to hospital discharge or ROSC.

Lidocaine (lignocaine) versus placebo

The ROC-ALPS trial, compared lidocaine (lignocaine) with placebo, and showed, with moderate certainty of evidence, no statistically significant difference in survival to hospital discharge with good neurological outcome, or survival to hospital discharge. However, the trial did show a statistically significant increase in ROSC favouring lidocaine (lignocaine).

Amiodarone versus lidocaine (lignocaine)

Two combined RCTs, the ALIVE trial[32] and the ROC-ALPS trial[29], compared amiodarone with lidocaine (lignocaine). These trials showed, with very low certainty of evidence, no statistically significant difference in survival to hospital discharge. The certainty of this combined evidence is confounded because the ALIVE trial enrolled from 1995 to 2001 when resuscitation differed to current practice, and because lidocaine (lignocaine) was mixed with polysorbate 80, the effects of which are uncertain.

The ROC-ALPS trial showed, with moderate certainty of evidence, no statistically significant difference in survival to hospital discharge with good neurological outcome, survival to hospital discharge, or ROSC.

The ALIVE trial showed, with very low certainty of evidence, no statistically significant difference in survival to hospital discharge.

Magnesium versus placebo

Three RCTs comparing magnesium with placebo showed, with very low certainty of evidence, no statistically significant difference in survival to hospital discharge with good neurological outcome.[5] The 3 trials and an additional RCT compared magnesium with placebo and showed, with very low certainty of evidence, no statistically significant difference in survival to hospital discharge or ROSC.[33-36]

Bretylium versus placebo

One RCT comparing bretylium with placebo showed, with very low certainty, no statistically significant difference in survival to hospital discharge.[37] Resuscitation practice at the time of patient enrolment differed substantially from current practice and bretylium is no longer available.

Lidocaine (lignocaine) versus bretylium

Two RCTs comparing lidocaine (lignocaine) with bretylium in 237 patients showed, with very low certainty, no statistically significant difference in survival to hospital discharge or ROSC.[38,39] Resuscitation practice at the time of patient enrolment differed substantially from current practice and bretylium is no longer available.

Amiodarone versus nifekalant

One small (30 patient) controlled trial comparing amiodarone with nifekalant showed, with very low certainty of evidence, no statistically significant difference in survival to hospital discharge with good neurological outcome, survival to hospital discharge or ROSC.[40]

Nifekalant has been used in Japan but is not available in Australia and New Zealand.

Lidocaine (lignocaine) versus nifekalant

One controlled trial comparing lidocaine (lignocaine) with nifekalant showed, with very low certainty, no statistically significant difference in survival to hospital discharge or ROSC.[41] Resuscitation practice at the time of patient enrolment differed substantially from current practice and nifekalant is not available in Australia and New Zealand.

Lidocaine (lignocaine) versus sotalol

One randomised controlled trial comparing lidocaine (lignocaine) with sotalol showed, with low certainty, no statistically significant difference in survival to hospital discharge with good neurological outcome, survival to hospital discharge, or ROSC.[42] The quality of this evidence was downgraded as a result of concerns about imprecision because the Confidence Intervals (CIs) around the point estimates were wide, because of the number of events, and because the sample size did not meet the optimal information size criteria. Also, resuscitation practice at the time of patient enrolment differed substantially from current practice.

Prophylactic use of antiarrhythmic drugs immediately after ROSC

There were no RCTs identified for the prophylactic use of antiarrhythmic drugs in patients during the first hour after ROSC following a VF/pVT cardiac arrest.

Recommendations

ANZCOR suggests the use of amiodarone in adults with shock-refractory VF/pVT [CoSTR 2018, weak recommendation, low-certainty evidence].

ANZCOR suggests lidocaine (lignocaine) may be used as an alternative to amiodarone in adults with shock-refractory VF/pVT [CoSTR 2018, weak recommendation, low certainty evidence].

ANZCOR suggests against the routine use of magnesium in adults with shock-refractory VF/pVT [CoSTR 2018, weak recommendation, very low-certainty evidence].

ANZCOR suggests against the routine use prophylactic antiarrhythmic drugs immediately after ROSC in adults with VF/pVT cardiac arrest [Good Practice Statement].

ANZCOR acknowledges identified knowledge gaps related to the following:

- Do antiarrhythmic drugs improve patient-centred outcomes (survival with good neurological outcome, health-related quality of life), and do the outcomes differ within or across specific populations (OHCA or IHCA) or conditions (e.g. witnessed arrest, monitored arrest, bystander CPR, number of shocks?).
- Does the use of adrenaline (epinephrine) alter the effectiveness of antiarrhythmic drugs during CPR for VF/pVT cardiac arrest and, if so, how?
- Is the use of multiple antiarrhythmic drugs (e.g. amiodarone followed by lidocaine (lignocaine) more effective than the use of a single drug during CPR for VF/pVT cardiac arrest?
- Does the route of administration (IV vs IO) of antiarrhythmic drugs given during CPR for VF/pVT alter their effectiveness?
- Does treatment with prophylactic antiarrhythmic drugs (including β-blockers) given immediately after ROSC, improve outcome following VF/pVT cardiac arrest?

Timing and Dosage of Antiarrhythmic

2.2.1 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.

Recommendations

ANZCOR suggests the use of amiodarone in adults with shock-refractory VF/pVT after the 3rd shock [CoSTR 2018, weak recommendation, low-certainty evidence]. Amiodarone prolongs the QT interval so should be avoided in Torsades de Pointes as it can make the situation worse [Good Practice Statement].

Consider amiodarone for prophylaxis after recurrent VF/pVT [Good Practice Statement].

Adverse Effects

- Hypotension
- Bradycardia
- Heart block

Amiodarone dosage

Initial bolus dose of amiodarone is 300mg. An additional dose of 150mg can be considered during refractory VF/pVT after the 5th defibrillation attempt. An infusion may be considered after recurrent VF/pVT (15 mg/kg over 24 hours).

Amiodarone should be avoided in *Torsades de pointes* as it can make the situation worse.

2.2.2 Lidocaine (lignocaine)

Lidocaine (lignocaine) acts as a sodium channel blocker.

Recommendations

ANZCOR suggests that lidocaine (lignocaine) may be used as an alternative to amiodarone in adults with shock-refractory VF/pVT [CoSTR 2018, weak recommendation, low certainty evidence].

Consider administration for prophylaxis after recurrent VF/pVT [Good Practice Statement].

Adverse effects

- Slurred speech
- Altered consciousness
- Muscle twitching, and seizures
- Hypotension
- Bradycardia
- Heart block

Lidocaine (lignocaine) Dosage

Initial bolus dose of lidocaine (lignocaine) is 1mg/kg.

An additional bolus of 0.5mg/kg may be considered during refractory VF/pVT after the 5th defibrillation attempt.

A lidocaine (lignocaine) infusion may be considered after recurrent VF/pVT (dose 1 to 4mg/min).

2.2.3 Magnesium (magnesium sulfate heptahydrate)

Magnesium is an electrolyte essential for membrane stability. Hypomagnesaemia causes myocardial hyper excitability particularly in the presence of hypokalaemia and digoxin.

Recommendations

ANZCOR suggests against the routine use of magnesium in adults with shock-refractory VF/pVT [CoSTR 2018, weak recommendation, very low-quality evidence].

Consider administration for:

- Torsade de pointes

- Cardiac arrest associated with digoxin toxicity
- Documented hypokalaemia
- Documented hypomagnesium.

[Good Practice Statement]

Adverse effects

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

Magnesium Dosage

Initial bolus dose of magnesium is 5mmol.

This may be repeated once during on-going resuscitation and followed by an infusion of 20mmol over 2 to 4 hours.

2.3 | Calcium

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

The ALS taskforce of ILCOR carried out a systematic review of the effects of calcium on outcomes from cardiac arrest in preparation for the 2023 CoSTR (https://costr.ilcor.org/document/calcium-during-cardiac-arrest-als-tfsr). This identified 12 studies of adult patients, including 4 studies from 3 randomised trials for adult OHCA patients and 8 observational studies for adult OHCA and/or IHCA patients.

For the important outcome of ROSC, 3 RCTs were identified. There was very low certainty evidence from 1 RCT of 90 people with OHCA[43], showing no significant difference in outcome with calcium chloride compared to placebo. There was very low certainty evidence from one RCT of 73 patients in refractory asystole[44], of no significant difference in outcome with calcium chloride. There was moderate certainty evidence from one RCT of 397 patients with OHCA[45] that found no significant difference in ROSC with calcium chloride compared to placebo. This trial was stopped early due to concerns for possible harm.

For the critical outcome of mid-term survival, there was moderate certainty evidence from one RCT enrolling 391 patients[45] showing no difference in 30-day, 90-day, or 6-month survival with calcium chloride compared to placebo.

For the critical outcome of survival with favourable neurologic outcome at 30 days or 6 months, there was moderate certainty evidence from one RCT[45] showing no significant difference with calcium chloride compared to placebo. However, for the critical outcome of survival with favourable neurological outcome at 90 days, there was moderate certainty evidence from the same RCT, showing harm from calcium chloride compared to placebo.

Additionally, for the critical outcomes of survival and survival with favourable neurological outcome at one year, there was moderate certainty evidence from the same RCT, suggesting a possible decrease in survival in the calcium chloride group compared to placebo.

Finally, for the critical outcome of quality of life, there was moderate certainty evidence from the one RCT[45], suggesting lower quality-of-life scores in survivors (5-dimensional, 5-level EuroQol score) in the calcium group at 30 days, 90 days, and 6 months compared with the placebo group at 6 months, though the difference was not statistically significant.

Recommendations

ANZCOR recommends against routine administration of calcium for the treatment of OHCA in adults [CoSTR 2023, strong recommendation, moderate certainty of evidence].

ANZCOR suggests against routine administration of calcium for the treatment of IHCA in adults [CoSTR 2023, weak recommendation, low certainty of evidence].

Consider administration for:

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

[Good Practice Statement]

Adverse effects

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

Calcium Dosage

The usual adult bolus dose in these settings is 10mL of 10% calcium chloride (10mL 10% calcium chloride = 6.8 mmol Ca ions). An alternative is 30mL calcium gluconate (30mL of 10% calcium gluconate = 6.6 mmol Ca ions).

Calcium dosage in cardiac arrest associated with hyperkalaemia:

Give 10mL calcium chloride 10% IV by rapid bolus injection. Consider repeating dose if cardiac arrest is prolonged or refractory.

2.4 | 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. Hypokalaemia is defined as a serum potassium level < 3.5mmol/L. Severe hypokalaemia is defined as a serum potassium level < 2.5mmol/L and may be associated with symptoms.

Recommendations

Consider IV administration for:

Persistent VF/pVT due to documented or suspected hypokalaemia [Good Practice Statement].

Adverse effects

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

Potassium Dosage

Initial bolus dose of potassium chloride of 5mmol intravenously.

2.5 | Sodium Bicarbonate (and other buffers)

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

Sodium bicarbonate use during cardiac arrest was previously reviewed by ILCOR in 2010 and updated in 2020 and 2025[11,3,7].

Recommendations

ANZCOR suggests against the routine use of buffering agents such as sodium bicarbonate for treatment of IHCA and OHCA unless a special circumstance is present [CoSTR 2025, weak recommendation, low-very low certainty evidence].

Consider administration for:

- Hyperkalaemia
- Treatment of documented metabolic acidosis
- Overdose with tricyclic antidepressants

- Protracted arrest (greater than 15 minutes).

[Good Practice Statement]

Adverse effects

- Metabolic alkalosis, hypokalaemia, hypernatraemia and hyperosmolality.
- Intracellular acidosis may develop or worsen when the CO_2liberated 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.

Sodium Bicarbonate Dosage

Initial bolus dose of Sodium Bicarbonate is 1mmol/kg, given over 2 to 3 minutes, then as guided by arterial blood gases.

2.6 | 2.6 Drugs for Torsades de Pointes

This subject was last reviewed in 2010.[11] An Evidence Update was included in the ILCOR 2020 review process. No new studies meeting inclusion criteria were identified. The treatment recommendation is therefore unchanged from 2010.

Recommendations

ANZCOR suggests polymorphic wide-complex tachycardia associated with familial long QT may be treated with IV magnesium to achieve supranormal levels levels (>2 mmol/L while avoiding toxicity) and correcting hypokalaemia and hypocalcaemia, pacing, and/or beta-blockers; however, isoprenaline should be avoided [Good Practice Statement].

ANZCOR suggests polymorphic wide-complex tachycardia associated with acquired long QT may be treated with IV magnesium to achieve supranormal levels (>2 mmol/L while avoiding toxicity) and correcting hypokalaemia and hypocalcaemia. The addition of pacing or IV isoprenaline may be considered when polymorphic wide-complex tachycardia is accompanied by bradycardia or appears to be precipitated by pauses in rhythm [Good Practice Statement].

3.0 | Other Drugs and Fluids

3.1 | Fluids

No published human study has directly compared outcome of routine intravenous fluid administration with no fluid administration during CPR. Two animal studies report that normothermic fluid infusion during CPR causes a decrease in coronary perfusion pressure, and another animal study showed that the coronary perfusion pressure associated with administration of 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.[11]

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 showed that hypertonic saline improves cerebral blood flow during CPR. Two animal studies found neither benefit nor harm with infusion of hypertonic saline.[11]

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.[11]

Recommendations

ANZCOR suggests that there is insufficient evidence to recommend for or against the routine infusion of intravenous fluids during cardiac arrest resuscitation.

ANZCOR recommends that fluids should be infused if hypovolemia is suspected (hypovolemic shock would normally require the administration of at least 20 mL/kg crystalloid solution e.g. 0.9% sodium chloride solution) [Good Practice Statement].

3.2 | Steroids

Steroids during CPR

Steroid use during CPR was previously reviewed by ILCOR in 2015. An evidence update in 2020 identified 2 large, population-based observational studies published since 2015.[46,47] Two additional

studies[24,48] and a systematic review and meta-analysis[25] looking at steroid in combination with vasopressin (see also section 2.2.3 above) were considered by ILCOR to be suitable for adolopment, and included in the 2022 CoSTR.[4] An evidence update was also included in CoSTR 2025.

Recommendations

ANZCOR suggests against the routine use of steroids during CPR for OHCA [CoSTR 2020 weak recommendation, very-low-certainty evidence].

ANZCOR suggests against the routine use of steroids during CPR for IHCA [Good Practice Statement].

ANZCOR suggests against the use of the combination of vasopressin and corticosteroids in addition to usual care for adult IHCA because of low confidence in effect estimates for critical outcomes [CoSTR 2022, weak recommendation, low- to moderate-certainty evidence].

ANZCOR suggest against the use of the combination of vasopressin and corticosteroids in addition to usual care for adult OHCA [CoSTR 2022, weak recommendation, very low to low-certainty evidence].

Post-ROSC Steroids

The use of steroids *after* the return of ROSC following cardiac arrest, for IHCA and OHCA, was addressed in the 2010 CoSTR and an Evidence Update was included in the 2020 CoSTR. ILCOR found insufficient evidence to make a recommendation.

Recommendations

ANZCOR suggests against the routine the use of corticosteroids for patients with ROSC after cardiac arrest [Good Practice Statement].

3.3 | 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.[11]

Recommendations

ANZCOR recommends against routine administration of fibrinolytics for the treatment of IHCA and OHCA [Good Practice Statement].

ANZCOR suggests administering fibrinolytic drugs (e.g. alteplase 100mg) for cardiac arrest when pulmonary embolism (PE) is the **suspected** cause of cardiac arrest [CoSTR 2020, weak recommendation, very low certainty evidence].[3]

ANZCOR suggests the use of fibrinolytic drugs or surgical embolectomy or percutaneous mechanical thrombectomy for cardiac arrest when PE is the **known** cause of cardiac arrest [CoSTR 2020, weak recommendation, very low certainty evidence].[3]

If a fibrinolytic drug is given in these circumstances, consider performing CPR for 60 to 90 minutes before termination of resuscitation attempts [Good Practice Statement].

Abbreviations

Abbreviations

Abbreviation	**Meaning/Phrase**
ALS	advanced life support
ANZCOR	Australian and New Zealand Committee on Resuscitation
CoSTR	Consensus on Science with Treatment Recommendations
CO_2	carbondioxide
CPR	cardiopulmonary resuscitation
IHCA	in-hospital cardiac arrest
ILCOR	International Liaison Committee on Resuscitation
IO	intraosseous
IV	intravenous
OHCA	out of hospital cardiac arrest
$NaHCO_3$	sodium bicarbonate
PEA	pulseless electrical activity
PE	pulmonary embolus
pVT	pulseless ventricular tachycardia
RCT	randomised control trial

ROSC	return of spontaneous circulation
VF	ventricular fibrillation

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About this Guideline

Search date/s	ILCOR literature search details and dates are available on the CoSTR page of the ILCOR website (https://costr.ilcor.org) and the relevant CoSTR documents: https://costr.ilcor.org/document/antiarrhythmic-drugs-for-cardiac-arrest-adults https://costr.ilcor.org/document/vasopressors-in-adult-cardiac-arrest ILCOR evidence review worksheets available at: https://costr.ilcor.org/document/antiarrhythmic-drugs-for-cardiac-arrest-adults
Questions/PICOs:	Are described in the CoSTR documents (https://costr.ilcor.org)
Method:	Mixed methods including ARC NHMRC methodology before 2017 and ILCOR GRADE methodology described in ILCOR publications since 2017.

Main changes:	No major changes to the clinical aspects of the guideline. Updating of review evidence, references, and terminology to increase consistency with GRADE terminology.
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