Adult Advanced Cardiovascular Life Support

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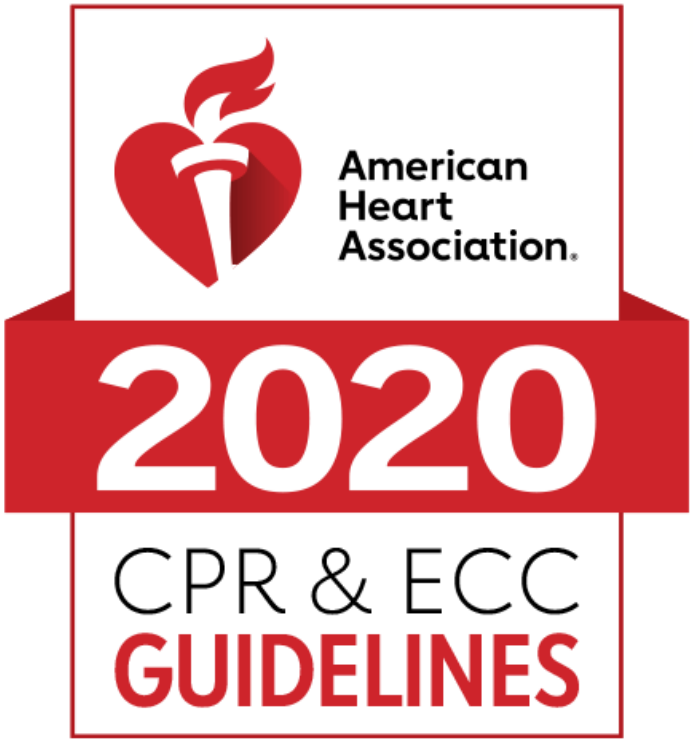
American
Heart
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2020
CPR & ECC
GUIDELINES


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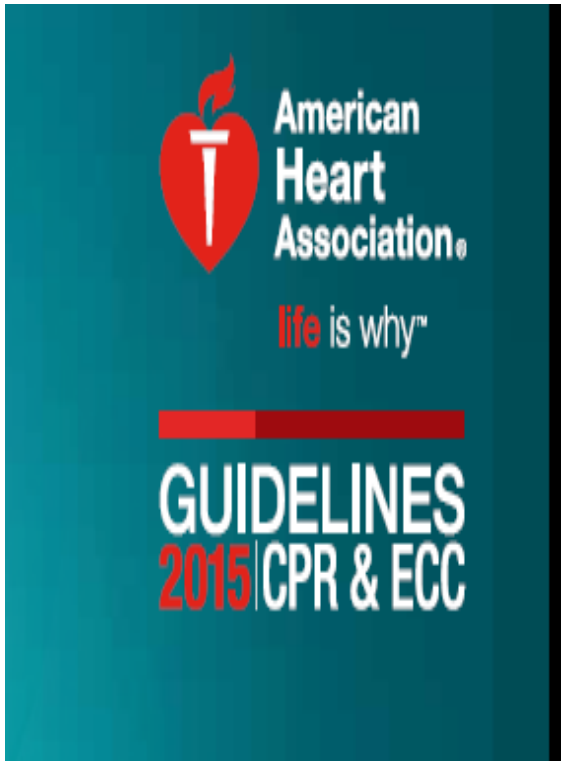
American
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life is why
GUIDELINES
2015 CPR & ECC

refrence

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.

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.

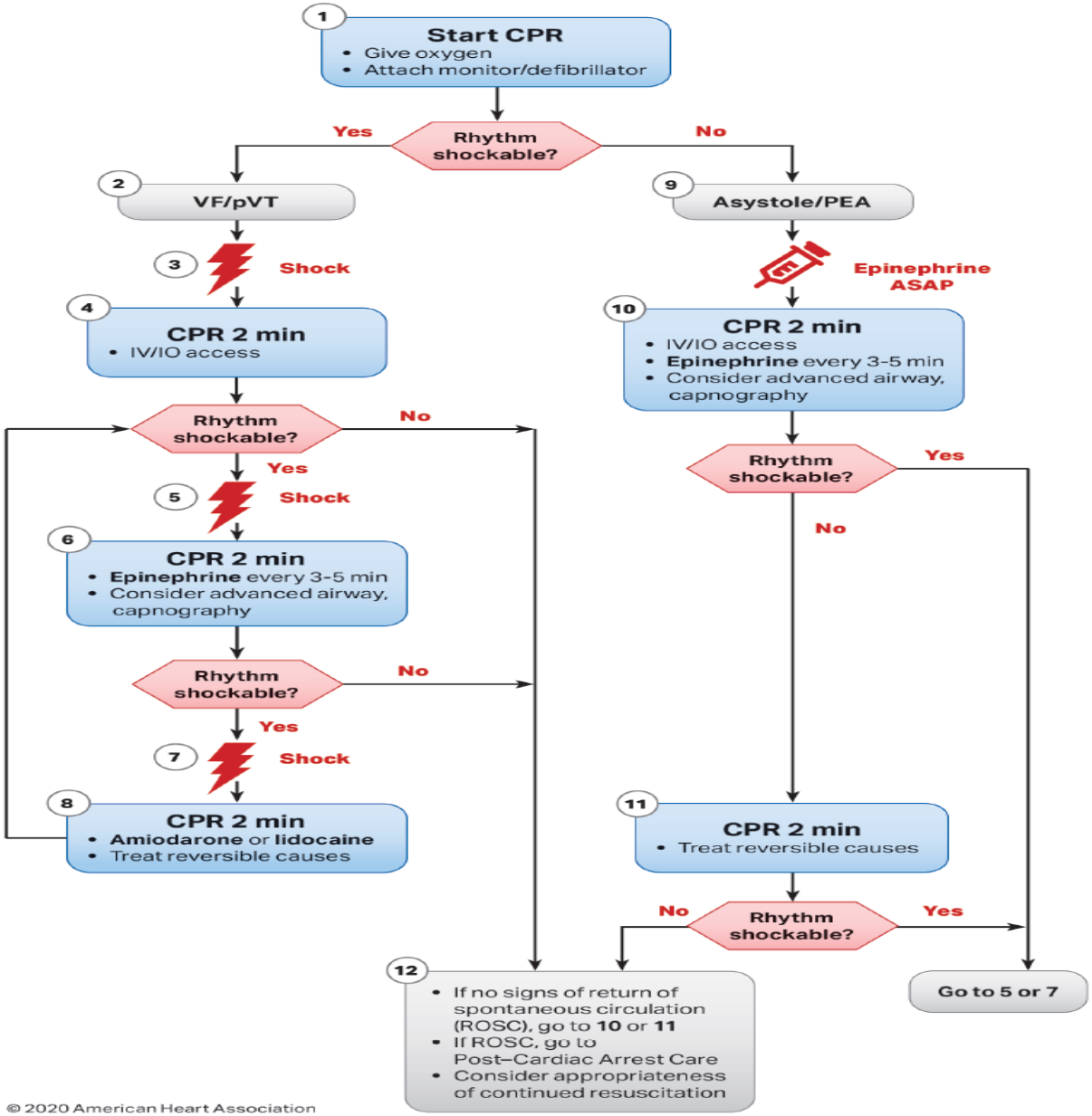
Adjunctive equipment is NOT essential for basic life support, however the use of Automated External Defibrillators (AEDs) by persons trained in their use but not trained in ALS

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

Figure 4. Adult Cardiac Arrest Algorithm.



CPR Quality

- Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
  - If $PETCO_2$ is low or decreasing, reassess CPR quality.

Shock Energy for Defibrillation

- **Biphasic**: Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic**: 360 J

Drug Therapy

- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus. Second dose: 150 mg.
  or
  Lidocaine IV/IO dose: First dose: 1-1.5 mg/kg. Second dose: 0.5-0.75 mg/kg.

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in $PETCO_2$ (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

defibrillation shock

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

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.
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.

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.

Indications

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

Timing of Defibrillation

The likelihood of defibrillation success decreases with time until definitive treatment (i.e. defibrillation) is initiated. 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. 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.

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.

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.

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 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

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

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.The anterior-posterior and anterior-lateral pad/paddle placements on the chest are acceptable in patients with a permanent pacemaker or ICD. 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

Medications in Adult Cardiac Arrest

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.

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 .

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. A number of devices are now available for use in adults

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.

This route cannot be used if a laryngeal mask airway is present.

Intracardiac injection

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

Specific Resuscitation Drugs

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.

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).

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.

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

Consider administration for:

- Prophylaxis of recurrent VF/VT.

A d v e r s e e f f e c t s :

- Hypotension
- Bradycardia
- Heart block.

D o s a g e :

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).

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 .

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.

Routine administration of calcium for treatment of in-hospital and out of hospital cardiac arrest is **not recommended**

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).

Lidocaine

Lidocaine acts as a sodium channel blocker.

Two randomized trials demonstrated the benefit of amiodarone over standard of care, which included lidocaine in 80% of cases, or routine use of lidocaine for shock refractory or recurrent VT/VF for the endpoint of survival to hospital admission, but not to survival to hospital discharge. There is **inadequate** evidence to support or refute the use of lidocaine in VT/VF not terminated by defibrillation, or VT/VF recurrence in out-of-hospital cardiac arrest or in-hospital cardiac arrest.

suggest that lidocaine may be used as an alternative to amiodarone in patients with **refractory** VF/pVT

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.

D o s a g e :

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.

Magnesium

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. suggest that magnesium should not be routinely used in adult cardiac arrest .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.

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.

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.

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.

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.

✓ Routine administration of sodium bicarbonate for treatment of in-hospital and out-of hospital cardiac arrest **is not** recommended.

Consider administration for:

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

Adverse effects:

- Metabolic alkalosis, hypokalaemia, hypernatraemia and hyperosmolality
- Intra cellular acidosis may develop or worsen when the CO2 liberated from NaHCO3 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.

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

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

- suggest against using vasopressin instead of adrenaline (epinephrine) for cardiac arrest
- suggest against adding vasopressin to standard dose adrenaline (epinephrine) during cardiac arrest

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.

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.

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 ported animal studies showed that the infusion of cold fluids during CPR caused a decrease in coronary perfusion pressure when compared to no fluids.

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

Fluids should be infused if hypovolemia is suspected (hypovolemic shock would normally require the administration of at least **20** mL/kg)

Steroids

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

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.

suggests against the routine use of steroids during CPR for OHCA

T h r o m b o l y t i c s

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.

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

Fibrinolysis should be considered in adult patients with cardiac arrest with proven or suspected pulmonary embolism If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60–90 min before termination of resuscitation attempts.

Post–Cardiac Arrest Care

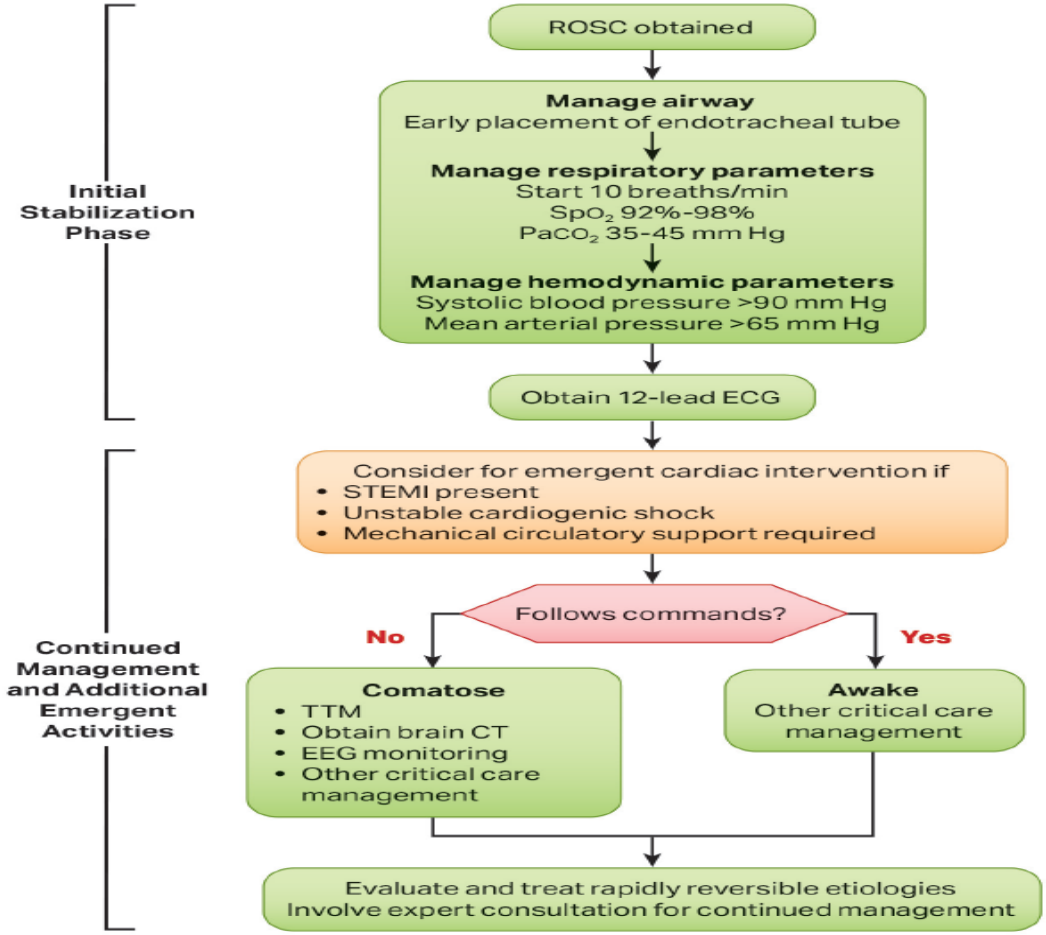
Summary of Key Issues and Major Changes

Key issues and major changes in the 2015 Guidelines Update recommendations for post–cardiac arrest care include the following:

- Emergency coronary angiography is recommended for all patients with ST elevation and for hemodynamically or electrically unstable patients without ST elevation for whom a cardiovascular lesion is suspected.
- TTM recommendations have been updated with new evidence suggesting that a range of temperatures may be acceptable to target in the post–cardiac arrest period.
- After TTM is complete, fever may develop. While there are conflicting observational data about the harm of fever after TTM, the prevention of fever is considered benign and therefore is reasonable to pursue.

- Identification and correction of hypotension is recommended in the immediate post–cardiac arrest period.
- Prognostication is now recommended no sooner than 72 hours after the completion of TTM; for those who do not have TTM, prognostication is not recommended any sooner than 72 hours after ROSC.
- All patients who progress to brain death or circulatory death after initial cardiac arrest should be considered potential organ donors.

Figure 7. Adult Post–Cardiac Arrest Care Algorithm.



Initial Stabilization Phase

Resuscitation is ongoing during the post-ROSC phase, and many of these activities can occur concurrently. However, if prioritization is necessary, follow these steps:

- Airway management: Waveform capnography or capnometry to confirm and monitor endotracheal tube placement
- Manage respiratory parameters: Titrate FIO_2 for SpO_2 92%–98%; start at 10 breaths/min; titrate to $PaCO_2$ of 35–45 mm Hg
- Manage hemodynamic parameters: Administer crystalloid and/or vasopressor or inotrope for goal systolic blood pressure >90 mm Hg or mean arterial pressure >65 mm Hg

Continued Management and Additional Emergent Activities

These evaluations should be done concurrently so that decisions on targeted temperature management (TTM) receive high priority as cardiac interventions.

- Emergent cardiac intervention: Early evaluation of 12-lead electrocardiogram (ECG); consider hemodynamics for decision on cardiac intervention
- TTM: If patient is not following commands, start TTM as soon as possible; begin at 32-36°C for 24 hours by using a cooling device with feedback loop
- Other critical care management
  - Continuously monitor core temperature (esophageal, rectal, bladder)
  - Maintain normoxia, normocapnia, euglycemia
  - Provide continuous or intermittent electroencephalogram (EEG) monitoring
  - Provide lung-protective ventilation

H's and T's

Hypovolemia
Hypoxia
Hydrogen ion (acidosis)
Hypokalemia/**h**yperkalemia
Hypothermia
Tension pneumothorax
Tamponade, cardiac
Toxins
Thrombosis, pulmonary
Thrombosis, coronary

Coronary Angiography

2015 (Updated): Coronary angiography should be performed emergently (rather than later in the hospital stay or not at all) for OHCA patients with suspected cardiac etiology of arrest and ST elevation on ECG. Emergency coronary angiography is reasonable for select (eg, electrically or hemodynamically unstable) adult patients who are comatose after OHCA of suspected cardiac origin but without ST elevation on ECG. Coronary angiography is reasonable in post–cardiac arrest patients for whom coronary angiography is indicated, regardless of whether the patient is **comatose or awake**.

Why: Multiple observational studies found positive associations between emergency coronary revascularization and both survival and favorable functional outcome. In the absence of cardiac arrest, guidelines already recommend emergency treatment of **STEMI** and emergency treatment **of non–ST-segment elevation ACS with electrical or hemodynamic instability**. Because the outcome of coma may be improved by correction of cardiac instability, and the prognosis of coma cannot be reliably determined in the first few hours after cardiac arrest, emergency treatment of post– cardiac arrest patients should follow identical guidelines.

Targeted Temperature Management

2015 (Updated): All comatose (ie, lacking meaningful response to verbal commands) adult patients with ROSC after cardiac arrest should have TTM, with a target temperature between 32°C and 36°C selected and achieved, then maintained constantly for at least 24 hours.

Why: Initial studies of TTM examined cooling to temperatures between 32°C and 34°C compared with no well-defined TTM and found improvement in neurologic outcome for those in whom hypothermia was induced. Taken together, the initial studies suggest that TTM is beneficial, so the recommendation remains to select a single target temperature and perform TTM.

Continuing Temperature Management Beyond 24 Hours

2015 (New): Actively preventing fever in comatose patients after TTM is reasonable.

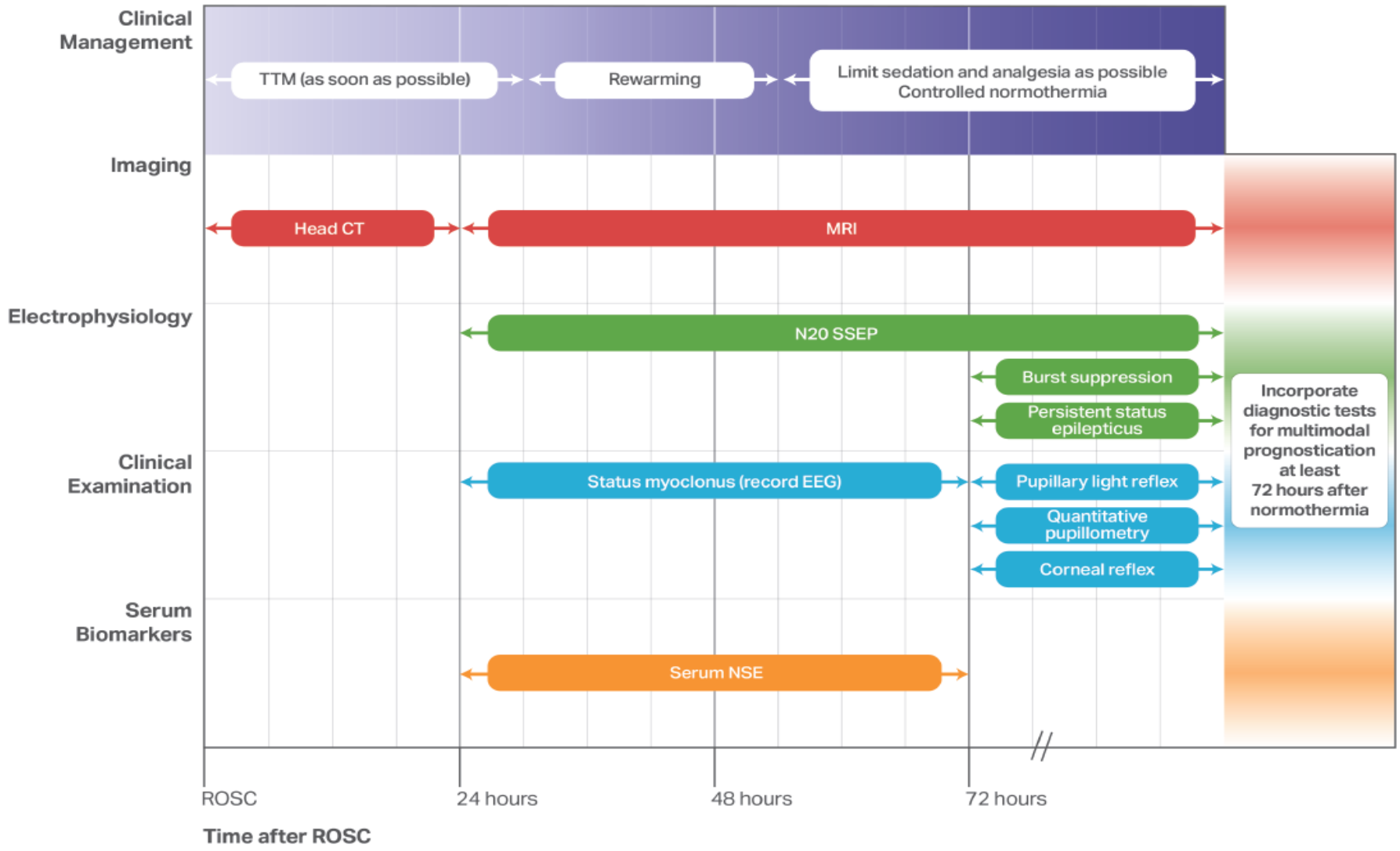
Why: In some observational studies, fever after rewarming from TTM is associated with worsened neurologic injury, although studies are conflicting. Because preventing fever after TTM is relatively benign and fever may be associated with harm, preventing fever is suggested.

Out-of-Hospital Cooling

2015 (New): The routine prehospital cooling of patients with rapid infusion of cold IV fluids after ROSC is not recommended.

Why: Before 2010, cooling patients in the prehospital setting had not been extensively evaluated. It had been assumed that earlier initiation of cooling might provide added benefits and also that prehospital initiation might facilitate and encourage continued in-hospital cooling. Recently published high-quality studies demonstrated no benefit to prehospital cooling and also identified potential complications when using cold IV fluids for prehospital cooling.

Figure 8. Recommended approach to multimodal neuroprognostication in adult patients after cardiac arrest.



Hemodynamic Goals After Resuscitation

2015 (New): It may be reasonable to avoid and immediately correct hypotension (systolic blood pressure less than 90 mm Hg, mean arterial pressure less than 65 mm Hg) during post–cardiac arrest care.

Why: Studies of patients after cardiac arrest have found that a systolic blood pressure less than 90 mm Hg or a mean arterial pressure of less than 65 mm Hg is associated with higher mortality and diminished functional recovery, while systolic arterial pressures of greater than **100** mm Hg are associated with better recovery. Also, because baseline blood pressure varies from patient to patient, different patients may have different requirements to maintain optimal organ perfusion.

Over the years, there have been many proposals about how long EMS should continue CPR and when to decide that further resuscitation is futile.

A National Association of EMS Physicians (NAEMSP) position statement in 2000 proposed that “an adequate effort” of CPR was 20 minutes, based on limited data, and this number has persisted in many protocols throughout the country

The American Heart Association (AHA) 2015 Guidelines discuss the limited data on how long to continue resuscitation, and ultimately choose not to make any recommendation because of such insufficient data.

This leaves open the question, “How long should we continue CPR when pulses don’t return

- For BLS-only resuscitation, termination of resuscitation is recommended for patients who:

Aren’t witnessed to arrest by EMS;

Never received a rescue shock; and

Never have return of pulses prior to commencing transport

- ✓ *For ALS resuscitations, termination of resuscitation is recommended for patients who:*

Aren’t witnessed to arrest by EMS;

Aren’t witnessed to collapse by bystanders;

Never receive a rescue shock; and

Never have return of pulses prior to commencing transport

WAVEFORM CAPNOGRAPHY

Waveform capnography is one tool to recognize the patient with preserved circulation during CPR. Excretion of CO_2 requires good blood flow to the lungs and continued metabolism by the patient.

High end-tidal carbon dioxide ($EtCO_2$) (> 20 mmHg) during CPR is a sign of life just like continued electrical activity in the ECG. It's reasonable to continue resuscitative efforts longer in a patient who's excreting CO_2 during CPR.

Conversely, the 2015 AHA Guidelines suggest that failure to achieve $EtCO_2$ > 10 mmHg after 20 minutes of CPR may be used to support termination of CPR.

Because measurement of CO_2 may have technical glitches, the Guidelines caution that this number should be used in conjunction with other clinical considerations and ideally measured via endotracheal tube.

Taken together with the termination of resuscitation algorithms, these data suggest a clinical approach for deciding how long to continue CPR that's based on clinical features and clinical response specific to the patient, not a set time interval.

CONCLUSION

There are some patients for whom prolonged CPR is futile. The termination of resuscitation algorithms usually can identify these patients with less than 20 minutes of CPR. When these rules aren’t met, 90% of patients who will respond to conventional CPR do so within 16–24 minutes.

When preserved excretion of CO_2 on waveform capnography, persistent cardiac electrical activity, or other clinical signs suggest that CPR is generating good blood flow in an individual patient, prolonged resuscitation still may result in good outcomes.