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Practice Guideline

European Resuscitation Council Guidelines 2025 Adult Advanced Life Support

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Abstract

These European Resuscitation Council (ERC) Guidelines 2025 Adult Advanced Life Support (ALS) are based on the International Liaison Committee on Resuscitation (ILCOR) Consensus on Cardiopulmonary Resuscitation Science with Treatment Recommendations (CoSTR). The evidence informing the ALS Guidelines is also included. When ILCOR has not addressed a specific topic, the ERC ALS Writing Group has provided its own guidance and the evidence supporting it. This section provides recommendations for ALS for adults with in- or out-of-hospital cardiac arrest. The ERC Guidelines 2025 ALS emphasise providing *early* and *effective* ALS interventions to improve survival from cardiac arrest in adults.

Keywords: Cardiac arrest, cardiopulmonary resuscitation, advanced life support

Introduction

These European Resuscitation Council (ERC) Guidelines 2025 Adult Advanced Life Support (ALS) include the advanced interventions that can be used in addition to basic life support (BLS) and automated external defibrillator (AED) by health care professionals. These ALS treatments are helpful when started quickly and early during cardiac arrest. ALS includes the prevention and treatment of both in-hospital cardiac arrest (IHCA) and out-of-hospital cardiac arrest (OHCA), the ALS algorithm, manual defibrillation, airway management during cardiopulmonary resuscitation (CPR), medication and their delivery, and the treatment of peri-arrest arrhythmias. These ERC Guidelines 2025 are based on the International Liaison Committee on Resuscitation (ILCOR) Consensus on Science and Treatment Recommendations (CoSTR) for ALS.[1] For these ERC Guidelines, the ILCOR recommendations were supplemented by focused literature reviews undertaken by the ERC ALS Writing Group for those topics not reviewed by ILCOR. When required, the guidelines were informed by the expert consensus of the writing group membership. For the first time we have had a patient public representative (GC) on the ALS Writing Group.

Abbreviations: AED, automated external defibrillator, AF, atrial fibrillation, ALS, advanced life support, BLS, basic life support, BP, blood pressure, CHD, coronary heart disease, CoSTR, Consensus on Science and Treatment Recommendation, CPR, cardiopulmonary resuscitation, DC, direct current, DNACPR, do not attempt cardiopulmonary resuscitation, DSD, dual (double) sequential defibrillation, ECG, electrocardiogram, ECPR, extra-corporeal cardiopulmonary resuscitation, EMS, emergency medical service, ERC, European Resuscitation Council, $ETCO_2$, End-tidal carbon dioxide, EWS, early warning score, ICD, implantable cardioverter defibrillator, IHCA, in-hospital cardiac arrest, ILCOR, International Liaison Committee on Resuscitation, IO, intraosseous, IV, intravenous, OHCA, out-of-hospital cardiac arrest, PEA, pulseless electrical activity, POCUS, point of care ultrasound, ROSC, return of spontaneous circulation, SBAR, situation, background, assessment, recommendation., SCD, sudden cardiac death, SGA, supraglottic airway, STEMI, ST-elevation myocardial Infarction, TOR, termination of resuscitation, VT, ventricular tachycardia, VF, ventricular fibrillation, VF/pVT, ventricular fibrillation/pulseless VT

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The scope of the 2025 ALS Guidelines was posted for feedback from National Resuscitation Councils (NRCs) and public comment and several new topics were added based on the scoping process that included a survey of NRCs.[2] The ALS Guidelines were drafted and agreed by the ALS Writing Group members and the ERC Guidelines 2025 Steering Committee. These Guidelines were posted for public comment between 5 and 30 May 2025. A total of 203 individuals or organisations submitted 325 comments, which resulted in 66 significant changes in the final version. The Guidelines were presented to and approved by the ERC Board and the General Assembly in June 2025. The methodology used for guideline development is presented in the Executive summary.[3]

The key messages are presented in Fig. 1. Table 1 summarises the major changes that have been made. Fig. 2 shows the 2025 ALS Algorithm.

Concise guidelines for clinical practice

Prevention of in-hospital cardiac arrest

The ERC recommends that:

- Shared decision making and advance care planning which integrates resuscitation decisions with emergency care treatment plans is used to increase clarity of treatment goals and also prevent inadvertent deprivation of other indicated treatments, besides CPR. These plans should be recorded in a consistent manner (See ERC Guidelines 2025 Ethics in Resuscitation).[4]
- Hospitals use a track and trigger early warning score system for the early identification of patients who are critically ill or at risk of clinical deterioration.
- Hospitals train staff in the recognition, monitoring and immediate care of the acutely ill patient.
- Hospitals empower all staff to call for help when they identify a patient at risk of physiological deterioration. This includes calls based on clinical concern, rather than solely on vital signs.
- Hospitals have a clear policy for the clinical response to abnormal vital signs and critical illness. This may include a critical care outreach service and, or emergency team (e.g. medical emergency team, rapid response team).
- Hospital staff use structured communication tools to ensure effective handover of information.
- Patients receive care in a clinical area that has the appropriate staffing, skills, and facilities for their severity of illness.
- Hospitals should review cardiac arrest events to identify opportunities for system improvement and share key learning points with hospital staff.
- Participation in national cardiac arrest audit as a benchmark for local performance.

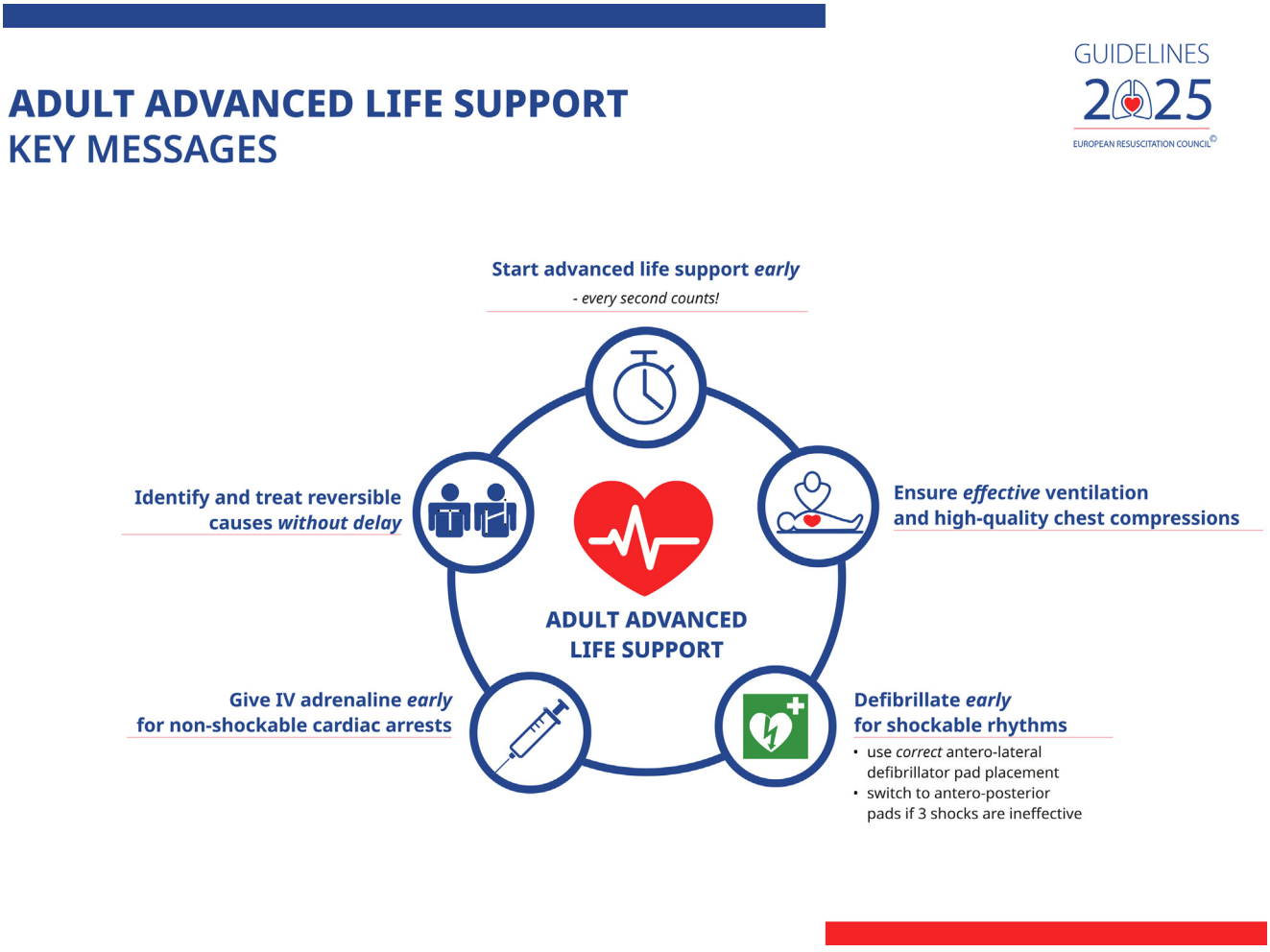

Fig. 1 – Advanced life support – Key messages.

Table 1 – What's new in the ERC Guidelines 2025 adult Advanced Life Support?

	Guidelines 2021	Guidelines 2025
Headlines	• Emphasis on: ○ High-quality chest compression ○ Premonitory signs to prevent cardiac arrest ○ Using basic and advanced airway techniques in a step wise fashion – only rescuers with a high success rate should use tracheal intubation ○ Early adrenaline for non-shockable rhythms	• Greater emphasis on: ○ Starting ALS as early as possible to help save more lives. ○ Effective oxygenation and ventilation breaths with high-quality chest compressions. ○ Correct apical (lateral) pad position for defibrillation. ○ Use of waveform capnography to confirm correct tracheal tube placement • What's out? o Calcium and sodium bicarbonate have no role during CPR except for very specific indications. o Precordial thump is no longer included in these Guidelines.
ALS in low resource settings	• Not mentioned in guidelines 2021	• ALS guidelines may have to be adapted according to resources and there may need to be a greater focus on prevention, early first aid, and basic life support measures. • Rescuers should be aware that even in high-income settings, ALS may be constrained by limited resources. • A two-tiered approach incorporating basic and advanced interventions may be the safest and most effective.
CPR-induced consciousness	• Not mentioned in guidelines 2021	• Rescuers may consider using sedative or analgesic drugs (or both) in small doses to prevent pain and distress to patients who are conscious during CPR (without ROSC). • Neuromuscular blocking drugs alone should not be given to conscious patients. • Drug regimens may be based on those used in critically ill patients and according to local protocols such as small doses of opioids, ketamine and/or midazolam.
Defibrillation: AED versus manual defibrillation during ALS	• Not clarified in 2021	• Manual defibrillators should only be used by rescuers who can quickly and accurately identify a cardiac arrest rhythm (within 5 s) and, if needed, deliver a safe shock with minimal interruption (aim for less than 5 s) to chest compressions. • ALS providers must be proficient in using both an AED and a manual defibrillator. • If an AED is already in use when ALS providers arrive, they should follow its shock prompts. When possible, they should transition to a manual defibrillator during a 2-minute CPR cycle.
Manual defibrillation strategy	• Mentioned in the supporting text: The 2015 ERC ALS Guideline stated that if there is doubt about whether the rhythm is asystole or extremely fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation. We wish to clarify that when the rhythm is clearly judged to be VF a shock should be given.	• Immediate defibrillation of (ventricular fibrillation) VF of any amplitude (even fine VF) should be attempted.
Refractory ventricular fibrillation	• For refractory VF, consider using an alternative defibrillation pad position (e.g. antero-posterior) • Do not use dual (double) sequential defibrillation for refractory VF outside of a research setting.	• For refractory VF, defined as continuous VF after three consecutive shocks, and having ensured correct antero-lateral pad positioning, consider using a defibrillation vector change by using an alternative defibrillation pad position (e.g. antero-posterior). • Dual (double) sequential defibrillation (DSD), involves using a combination of antero-lateral and antero-posterior pad positioning, discharged in close succession.

(*continued on next page*)

Table 1 (continued)

	Guidelines 2021	Guidelines 2025
		Given the practical challenges of using two defibrillators to deliver DSD and the limited evidence for its efficacy the ERC does not recommend its routine use.
Bag-mask ventilation	• Not emphasised in 2021	• Deliver effective bag-mask ventilation breaths by optimising mask seal and airway patency and if necessary use a two-person technique for bag-mask ventilation.
Choice of supraglottic airway	• This was not specified in 2021	• When using a supraglottic airway (SGA), an i-gel is preferred to a laryngeal tube.
Confirmation of correct tracheal tube placement	• Use waveform capnography to confirm tracheal tube position.	• A sustained $ETCO_2$ trace on waveform capnography must be used to exclude oesophageal placement of the tracheal tube.
Mechanical ventilator settings during chest compressions	• This was not specified in 2021	• If a mechanical ventilator is used during CPR, use a volume-controlled or pressure regulated mode during chest compressions set the ventilator to a tidal volume of 6–8 mL kg^{-1} (predicted body weight), or to achieve a visible chest movements, the maximum fraction of inspired oxygen, a respiratory rate of 10 min^{-1}, an inspiratory time of 1–1.5 s, a positive end expiratory pressure (PEEP) 0–5 cm H_2O, the peak pressure alarm at 60–70 cm H_2O, and turn off the inspiratory trigger. Ensure mechanical ventilation is effective and if not, use manual ventilation.
Vascular access	• Attempt intravenous (IV) access first to enable drug delivery in adults in cardiac arrest. • Consider intraosseous (IO) access if attempts at IV access are unsuccessful or IV access is not feasible	• Attempt intravenous (IV) rather than intraosseous (IO) access first, to enable drug delivery in adults in cardiac arrest. • If IV access cannot be rapidly achieved within two attempts, it is reasonable to consider IO access as an alternative route for vascular access during adult cardiac arrest.
Use of calcium, sodium bicarbonate and corticosteroids	• This was not made explicit in 2021	• Do not routinely give calcium, sodium bicarbonate or corticosteroids during cardiac arrest.
ALS in highly-monitored cardiac arrest, and physiology-guided CPR	• Not mentioned in Guidelines 2021	• A sudden decrease in $ETCO_2$ may indicate a cardiac arrest or very low cardiac output state. • Consider starting chest compressions if the systolic blood pressure decreases and remains < 50 mmHg despite interventions. • In adults with continuous intra-arterial blood pressure monitoring, we suggest that adrenaline is initially given in small increments (e.g., 50–100 μg IV) rather than a 1 mg bolus. • A pragmatic approach during physiology-guided CPR is to aim for a diastolic blood pressure of ≥30 mmHg (when using intra-arterial blood pressure monitoring) and an $ETCO_2 \geq 25$ mmHg (3.3 kPa).
Peri-arrest arrhythmias		• The 2025 has a greater emphasis on those arrhythmias that require immediate treatment before or after cardiac arrest. • The tachycardias section has been renamed tachyarrhythmias. • There is greater emphasis on use of electrical cardioversion with a synchronised shock for patients immediately after ROSC, or who are unstable.

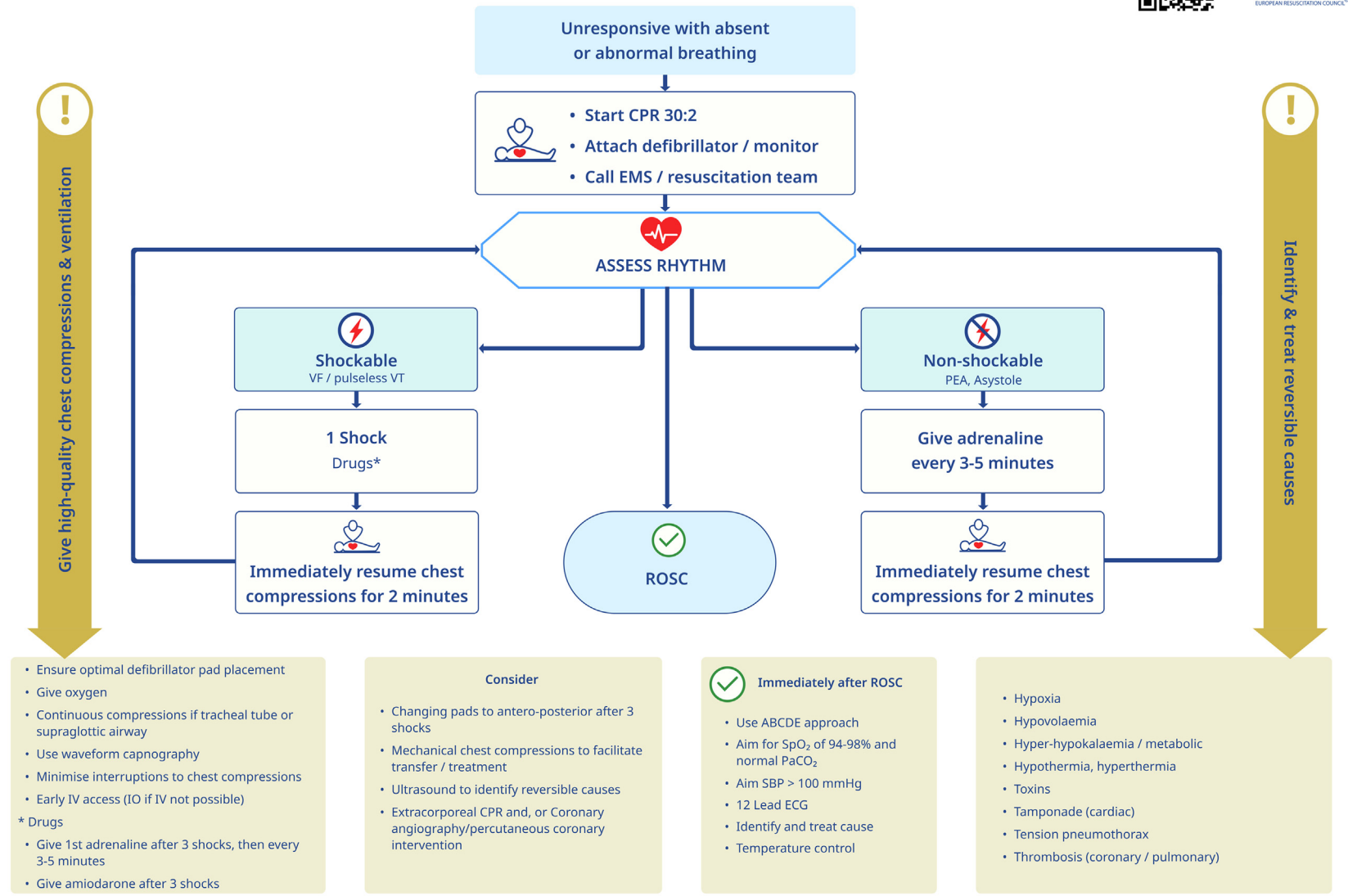

Fig. 2 – Advanced Life Support algorithm

ABCDE airway, breathing, circulation, disability, exposure; CPR cardiopulmonary resuscitation; ECG electrocardiogram; EMS emergency medical system; IO intraosseous; IV intravenous; PEA pulseless electrical activity; $PaCO_2$ arterial partial pressure of carbon dioxide; ROSC return of spontaneous circulation; SpO_2 oxygen saturation measured with pulse oximetry; VF ventricular fibrillation; VT ventricular tachycardia.

Prevention of out-of-hospital cardiac arrest

- Coronary heart disease (CHD) is the leading cause of sudden cardiac death (SCD), responsible for 80 % of cases, particularly in older patients. Non-ischaemic cardiomyopathies contribute to 10–15 % of SCD cases. In younger individuals, the main causes of SCD include inherited heart diseases, congenital heart defects, myocarditis, and substance misuse. In these patient groups, risk stratification is possible, and preventive treatments may be effective.
- Predicting SCD is challenging because most cases happen in individuals with undiagnosed heart disease. As a result, detecting early warning signs, implementing an efficient emergency medical services (EMS) system, and focusing on the prevention of cardiovascular disease (CVD) risk factors are crucial in the general population.
- Symptoms such as chest pain, syncope (especially during exercise, while sitting or supine), palpitations, dizziness or sudden shortness of breath that are consistent with cardiac ischaemia or an arrhythmia should be investigated.
- Overtly healthy young adults who have SCD can also have preceding signs and symptoms (e.g. syncope/pre-syncope, chest pain and palpitations) that should alert healthcare professionals to seek expert help to prevent cardiac arrest.
- Young adults presenting with characteristic symptoms of arrhythmic syncope should have a specialist cardiology assessment, which should include an electrocardiogram (ECG) and in most cases echocardiography, 24-hour ECG monitoring and an exercise test.
- Systematic evaluation in a clinic specialising 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.
- Identification of individuals with inherited conditions and screening of family members can help prevent deaths in young people with inherited heart disorders.
- Follow current European Society of Cardiology (ESC) guidelines for the diagnosis and management of syncope and arrhythmias.

Treatment of in-hospital cardiac arrest

- Start ALS as early as possible.
- Hospital systems should aim to recognise cardiac arrest, start CPR immediately, defibrillate rapidly (<3 min) for shockable rhythms, give adrenaline rapidly for non-shockable rhythms, and identify and treat reversible causes.
- All hospital staff should be able to recognise cardiac arrest rapidly, call for help, start CPR and defibrillate (attach an AED and follow the AED prompts, or use a manual defibrillator).
- Hospitals should adopt a standard 'Cardiac Arrest Call' telephone number (2222).
- Hospitals should have a resuscitation team that immediately responds to IHCAs.
- The hospital resuscitation team should include team members who have completed an accredited adult ALS course that incorporates teamwork and leadership training.
- Resuscitation team members should have the key skills and knowledge to manage a cardiac arrest including manual defibrillation, advanced airway management, intravenous access, intra-osseous access, and identification and treatment of reversible causes.
- The resuscitation team should meet at the beginning of each shift for introductions and allocation of team roles.
- Hospitals should standardise resuscitation equipment.
- Termination of resuscitation rules (TOR) should not be used as a sole strategy for terminating an in-hospital resuscitation attempt (See Ethics in Resuscitation[4]).

Treatment of out-of-hospital cardiac arrest

- Start ALS as early as possible – EMS systems should be organised to provide a rapid ALS response with sufficient qualified personnel. This may include a prehospital critical care team.
- Adults with non-traumatic OHCA should be considered for transport to a cardiac arrest centre according to local protocols and take into account which interventions can be provided on scene.
- Emergency medical systems should consider implementing validated criteria for the withholding and termination of resuscitation (TOR) taking into consideration specific local legal, organisational and cultural context (See Ethics in Resuscitation[4]).
- Emergency medical systems should monitor staff exposure to resuscitation and low exposure should be addressed to increase EMS team experience in resuscitation.

Debriefing

Use data-driven, performance-focused debriefing of rescuers to improve CPR quality and patient outcomes (See Education ERC Guidelines 2025 Education for Resuscitation[5]).

ALS in low-resource settings

- Advanced Life Support guidelines may have to be adapted according to resources and there may need to be a greater focus on prevention, early first aid, and basic life support measures (See the ERC Guidelines 2025 System Saving Lives[6] and First Aid[7]).
- Rescuers should be aware that even in high-income settings, ALS may be constrained by limited resources.
- A two-tiered approach incorporating basic and advanced interventions may be the safest and most effective.

CPR-induced consciousness

- Cardiopulmonary resuscitation induced consciousness (without ROSC) is uncommon but increasingly reported. Rescuers may consider using sedative or analgesic drugs (or both) in small doses to prevent pain and distress to patients who are conscious during CPR.
- Neuromuscular blocking drugs alone should not be given to conscious patients.
- The optimal drug regimen for sedation and analgesia during CPR is uncertain. Regimens may be based on those used in critically ill patients and according to local protocols such as small doses of fentanyl, ketamine and/or midazolam.

Defibrillation

Automated external defibrillation (AED) versus manual defibrillation during ALS

- Manual defibrillators should only be used by rescuers who can quickly and accurately identify a cardiac arrest rhythm (within 5 s) and, if needed, deliver a safe shock with minimal interruption (aim for less than 5 s) to chest compressions.
- Advanced Life Support providers must be proficient in using both an AED and a manual defibrillator.

- If an AED is already in use when ALS providers arrive, they should follow its shock prompts. When possible, they should transition to a manual defibrillator during a 2-minute CPR cycle.

Defibrillation strategy

- Continue CPR while a defibrillator is retrieved and defibrillation pads applied. High-quality CPR improves the chances of successful defibrillation.
- Give a shock as early as possible when appropriate.
- Deliver shocks with minimal interruption to chest compressions and minimise the pre-shock and post-shock pause. This is achieved by continuing chest compressions during defibrillator charging, delivering defibrillation aiming for an interruption in chest compressions of less than 5 s and then immediately resuming chest compressions.
- Immediate defibrillation of (ventricular fibrillation) VF of any amplitude (even fine VF) should be attempted.
- Immediately resume chest compressions after shock delivery. If there is a combination of clinical and physiological signs of return of spontaneous circulation (ROSC) such as return of consciousness, purposeful movement, arterial waveform or a sharp rise in $ETCO_2$, consider stopping chest compressions for rhythm analysis, and if appropriate, a pulse check.
- When using a defibrillator that displays the ECG with the motion artefact caused by chest compressions removed, the underlying cardiac arrest rhythm may guide the decision to perform a rhythm and pulse check every two minutes. If asystole is displayed there would be no need to pause chest compressions for a rhythm check.

Safe and effective defibrillation

- Minimise the risk of fire by taking off any oxygen mask, nasal cannulae or bag mask and by placing them at least 1 m away from the patient's chest. When using a mechanical ventilator, oxygen exhaust from ventilation circuits should be directed away from the chest. A self-inflating bag or the ventilator circuit should remain attached to a supraglottic airway or tracheal tube.
- Charging the defibrillator in anticipation of each rhythm check may minimise hands-off time prior to shock delivery and is an acceptable alternative strategy if delivered without prolonging the peri-shock pause.
- A shock with a manual defibrillator can be safely delivered without interrupting mechanical chest compression.
- Do not defibrillate during manual chest compressions (even when wearing clinical gloves), as that practice is not safe to the rescuer.

Defibrillation pads and paddles

- There is insufficient evidence to recommend a specific pad or paddle size for optimal external defibrillation in adults.
- When available, defibrillation pads are preferable to paddles as they offer practical benefits for routine monitoring and defibrillation. Pads enable the operator to stand clear during defibrillation and minimise pre- and post-shock pauses to chest compressions by enabling hands-free operation. Better contact with the chest wall may also reduce the risk of arcing and subsequent fires.
- When using defibrillation paddles, apply firm force to both defibrillation paddles to optimise skin contact, minimise transthoracic impedance and reduce the risk of electrical arcing.
- Antero-lateral pad position is the position of choice for initial pad/-paddle placement. In particular, ensure that the apical (lateral) pad is positioned correctly (i.e. below the armpit in the mid-axillary line).
- Consider and antero-posterior pad position for vector change defibrillation following three failed shocks in cases of refractory shockable rhythms. The anterior pad is placed to the left of the sternum, avoiding as much breast tissue as possible. The posterior pad is placed at the same height, centred just medial to the left scapula.
- In patients with an implantable pacemaker/defibrillator (ICD), place the pad more than 8 cm away from the device, or use an alternative pad position.
- Consider an alternative pad position when the patient is in the prone position (bi-axillary), or in a refractory shockable rhythm.

Energy levels and number of shocks

- Use single shocks followed by a 2-minute cycle of chest compressions.
- The use of up to three stacked shocks may be considered only if initial ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT) occurs during a witnessed, monitored cardiac arrest with a defibrillator immediately available, e.g. during cardiac catheterisation or in a high dependency area. (For the purposes of adrenaline and amiodarone administration after three failed shocks, the initial three stacked shocks should be counted as the initial shock).
- Energy levels:
  - For biphasic waveforms (rectilinear biphasic or truncated exponential biphasic, but not pulsed biphasic), defibrillation shock energy levels for the first shock is at least 150 J.
  - For pulsed biphasic waveforms, deliver the first shock at 130–150 J.
- 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 for subsequent shocks.
- If the rescuer is unaware of the recommended energy settings of the defibrillator, for an adult use the highest energy setting for all shocks.
- Use standard energy levels in obese patients.

Refractory ventricular fibrillation

- Consider escalating the shock energy, after a failed shock.
- For refractory VF, defined as continuous VF after three consecutive shocks, and having ensured correct antero-lateral pad positioning, consider using a defibrillation vector change by using an alternative defibrillation pad position (e.g. antero-posterior). After a failed third shock, prepare to place a fresh set of defibrillation pads, at the time of the following rhythm check. Optimise transthoracic impedance by shaving the anticipated areas of pad placement (if necessary).

Dual (double) sequential defibrillation (DSD), involves using a combination of antero-lateral and antero-posterior pad positioning, discharged in close succession and has been advocated for use in refractory shockable rhythms. Given the practical challenges of using two defibrillators to deliver DSD and the limited evidence for its efficacy the ERC does not recommend its routine use.

Ventricular fibrillation waveform analysis for optimising shock success

- Rescuers should give defibrillation shocks according to AED prompts or use a manual defibrillator for ventricular fibrillation/ pulseless ventricular tachycardia (VF/pVT) according to the ALS algorithm – there is currently no role for VF waveform analysis (e.g., based on amplitude) for identifying the optimal time for defibrillation.

Patients with actively discharging implantable cardioverter defibrillators

- Rescuers may sense a significant shock across their arms if a shock is delivered by an ICD while they are performing external chest compressions, even when wearing clinical gloves.
- If an ICD fails to terminate a shockable rhythm, conventional external shocks should be delivered, placing any defibrillation pad/paddle more than 8 cm from the defibrillator box (as above).
- If the ICD is incorrectly detecting arrhythmias and shocking inappropriately, a magnet placed over the ICD can temporarily stop shocks but will not disable pacing (if programmed).

Airway and ventilation

- During CPR, start with basic airway techniques and progress stepwise according to the skills of the rescuer until effective ventilation is achieved.
- Give the highest feasible inspired oxygen during CPR.
- Start effective ventilation breaths as soon as possible ensuring the rate and tidal volume are appropriate to prevent both inadequate ventilation (hypoventilation) and excessive ventilation (hyperventilation).
- Deliver effective bag-mask ventilation breaths by optimising mask seal and airway patency and if necessary, use a two-person technique for bag-mask ventilation.
- Give each inspiratory breath over 1 s to achieve a visible chest movement.
- When using a supraglottic airway (SGA), an i-gel is preferred to a laryngeal tube.
- Tracheal intubation should only be attempted by rescuers with a high success rate and with the use of continuous waveform capnography. The expert consensus is that a high tracheal intubation success rate is over 95 % within two attempts at intubation.
- Aim for less than a 5-second interruption in chest compression for tracheal intubation.
- Use direct or video laryngoscopy for tracheal intubation according to local protocols and rescuer experience. In settings where video laryngoscopy is immediately available, it is preferable to use video laryngoscopy instead of direct laryngoscopy.
- A sustained $ETCO_2$ trace on waveform capnography must be used to exclude oesophageal placement of the tracheal tube.
- Once a tracheal tube or a SGA has been inserted, ventilate the lungs at a rate of 10 min^{-1} and continue chest compressions without pausing during ventilations. With a SGA, if gas leakage results in inadequate ventilation, pause compressions for ventilation using a compression-ventilation ratio of 30:2.
- If using mechanical ventilation, use a volume-controlled mode during chest compressions set the ventilator to a tidal volume of 6–8 mL kg^{-1} (predicted body weight), or to achieve a visible chest movement, the maximum inspired oxygen, a respiratory rate of 10 min^{-1}, an inspiratory time of 1–2 s, a positive end expiratory pressure (PEEP) 0–5 cm H_2O, the peak pressure alarm at 60–70 cm H_2O, and the flow trigger off. Ensure mechanical ventilation is effective and if not, use manual ventilation.
- If standard airway management strategies (oropharyngeal airway and bag-mask/supraglottic airway/ tracheal tube) fail during cardiac arrest, appropriately trained rescuers should attempt surgical cricothyroidotomy to enable oxygenation and ventilation.

Medication and fluids

Vascular access

- Attempt intravenous (IV) rather than intraosseous (IO) access first, to enable drug delivery in adults in cardiac arrest.
- If IV access cannot be rapidly achieved within two attempts, it is reasonable to consider IO access as an alternative route for vascular access during adult cardiac arrest.

Vasopressor drugs

- Give adrenaline 1 mg as soon as possible for adult patients in cardiac arrest with a non-shockable rhythm.
- Give adrenaline 1 mg after the third shock for adult patients in cardiac arrest with a shockable rhythm.
- Repeat adrenaline 1 mg every 3–5 min whilst ALS continues.

Antiarrhythmic drugs

- Give amiodarone 300 mg IV for adult patients in cardiac arrest who are in VF/pVT after three shocks have been administered.
- Give a further dose of amiodarone 150 mg IV for adult patients in cardiac arrest who are in VF/pVT after five shocks have been administered.
- Give the first dose of amiodarone after three shocks, and the second dose after five shocks, irrespective of whether the shockable rhythms are sequential (refractory) or intermittent (recurrent).
- Lidocaine 100 mg IV may be used as an alternative if amiodarone is not available or a local decision has been made to use lidocaine instead of amiodarone. An additional bolus of lidocaine 50 mg can also be given after five defibrillation attempts.

Thrombolytic drugs

- Consider immediate thrombolytic drug therapy when pulmonary embolism is the suspected or confirmed cause of cardiac arrest.
- In select patients with suspected pulmonary embolism, consider CPR for 60–90 min after administration of thrombolytic drugs.

Fluids

- Give fluids during CPR only if cardiac arrest is caused by hypovolaemia.
- Use either isotonic saline or balanced crystalloids for fluid infusion during CPR.

Other medication

- Do not routinely give calcium, sodium bicarbonate or corticosteroids during cardiac arrest.

ALS in highly-monitored cardiac arrest, and physiology-guided CPR

- A sudden decrease in $ETCO_2$ may indicate a cardiac arrest or very low cardiac output state.
- Consider starting chest compressions if the systolic blood pressure decreases and remains <50 mmHg despite interventions.

- In adults undergoing continuous intra-arterial blood pressure monitoring, we suggest that adrenaline is initially given in small increments (e.g., 50–100 μg IV) rather than a 1 mg bolus. If a total of 1 mg has been given with no response, ensure that there is no extravasation and consider giving further IV adrenaline doses of 1 mg every 3–5 min.
- A pragmatic approach during physiology-guided CPR is to aim for a diastolic blood pressure of ≥30 mmHg (when using intra-arterial blood pressure monitoring) and an $ETCO_2$ - ≥ 25 mmHg (3.3 kPa).

Waveform capnography during advanced life support

- Use waveform capnography to confirm correct tracheal tube placement during CPR.
- Use waveform capnography to monitor the quality of CPR.
- An increase in $ETCO_2$ during CPR may indicate that ROSC has occurred. However, chest compression should not be interrupted based on this sign alone. Use a combination of clinical and physiological signs of ROSC (e.g., consciousness, purposeful movement, arterial waveform, rise in $ETCO_2$) before stopping chest compressions for rhythm analysis, and if appropriate, a pulse check.
- Do not use a low $ETCO_2$ value alone to decide if a resuscitation attempt should be stopped.

Use of ultrasound imaging during advanced life support

- Only skilled operators should use intra-arrest point-of-care ultrasound (POCUS).
- POCUS must not cause additional or prolonged interruptions in chest compressions.
- POCUS may help identify treatable causes of cardiac arrest such as cardiac tamponade and tension pneumothorax.
- Right ventricular dilation in isolation during cardiac arrest should not be used to diagnose pulmonary embolism.
- Do not use POCUS for assessing contractility of the myocardium as a sole indicator for terminating CPR.

Devices

Mechanical chest compression devices

- Consider mechanical chest compressions only if high-quality manual chest compression is not practical or compromises provider safety.
- When a mechanical chest compression device is used, minimise interruptions to chest compression during device application by using only trained teams familiar with the device.

Resuscitative endovascular balloon occlusion of the aorta (REBOA)

- The ERC does not recommend the routine use of REBOA for cardiac arrest unless being evaluated in a clinical trial.

Intra-arrest cooling

- We do not recommend intra-arrest cooling during advanced life support (unless there is severe hyperthermia).

Extracorporeal CPR

- ECPR may be considered as a rescue therapy for selected adults with IHCA and OHCA when conventional CPR is failing to restore spontaneous circulation, in settings in which this can be implemented.

Peri-arrest arrhythmias

- The ERC Guidelines 2025 ALS and the algorithms focus on those arrhythmias that require immediate treatment before or after cardiac arrest.
- Rescuers should seek expert advice if the arrhythmia and/or life-threatening features persist.
- The assessment and treatment of all arrhythmias address the condition of the patient (stable versus unstable) and the nature of the arrhythmia. Persistent arrhythmias require careful evaluation, as they are often linked to underlying structural heart disease and may indicate unresolved issues such as myocardial ischaemia. In addition to an arrhythmia occurring immediately after ROSC, life-threatening features in an unstable patient include:
  - Shock – recognised by hypotension (e.g., systolic blood pressure <90 mmHg) along with signs of compensatory mechanisms, such as increased sympathetic activity, and evidence of inadequate organ perfusion
  - Syncope – as a consequence of reduced cerebral blood flow.
  - Heart failure – manifested by pulmonary oedema (failure of the left ventricle) and/or raised jugular venous pressure (failure of the right ventricle).
  - Myocardial ischaemia – may present with chest pain (angina) or may occur without pain as an isolated finding on the 12-lead ECG (silent ischaemia).

Tachyarrhythmias

- Electrical cardioversion is the preferred treatment for tachyarrhythmia in the unstable patient displaying potentially life-threatening adverse signs or immediately after ROSC.
- Electrical cardioversion is recommended for stable patients with monomorphic VT who have structural heart disease or when it is unclear whether there is underlying heart muscle damage.
- Conscious patients require careful anaesthesia or sedation before attempting synchronised cardioversion – be aware of the risk of haemodynamic deterioration with anaesthesia/sedation.
- When cardioverting atrial or ventricular tachyarrhythmias, the shock must be synchronised to occur with the R wave of the ECG.
- For atrial fibrillation:
  - An initial synchronised shock at maximum defibrillator output, rather than an escalating approach, is a reasonable strategy based on current data.
- For atrial flutter and paroxysmal supraventricular tachycardia:
  - Give an initial shock of 70–120 J.
  - Give subsequent shocks using stepwise increases in energy.
- For ventricular tachycardia with a pulse:
  - Use energy levels of 120–150 J for the initial shock.
  - Consider stepwise increases in energy if the first shock fails to achieve sinus rhythm.
- If cardioversion fails to restore sinus rhythm and the patient remains unstable, give amiodarone 300 mg intravenously over 10–20 min (or procainamide 10–15 mg/kg over 20 min) and re-attempt electrical cardioversion. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 h.
- Pharmacological treatment may be considered in haemodynamically stable patients with monomorphic ventricular tachycardia if there is an increased risk with sedation or anaesthesia.

- Consider amiodarone for acute heart rate control in patients with AF and haemodynamic instability and severely reduced left ventricular ejection fraction (LVEF). For stable patients with LVEF < 40 % consider the smallest dose of beta-blocker to achieve a heart rate less than 110 min^{-1}. Add digoxin if necessary.

Bradycardia

- If bradycardia is accompanied by adverse signs, give atropine 500 μg IV (IO) and, if necessary, repeat every 3–5 min to a total of 3 mg.
- If treatment with atropine is ineffective, consider second-line drugs. These include isoprenaline (5 μg min^{-1} starting dose), and adrenaline (2–10 μg min^{-1}).
- For bradycardia in patients with cardiac transplant or spinal cord injury, consider giving aminophylline (100–200 mg slow intravenous injection). Do not give atropine to patients with cardiac transplants – it can cause a high-degree atrioventricular block or even sinus arrest – use aminophylline.
- Consider giving glucagon if beta-blockers or calcium channel blockers are a potential cause of the bradycardia.
- Do not give atropine to patients with high-degree atrioventricular block and wide QRS. It is ineffective and may worsen the block.
- Consider pacing in patients who are unstable, with symptomatic bradycardia refractory to drug therapies.
  - Establish early transvenous pacing in unstable patients with symptomatic bradycardia.
  - Consider transthoracic (transcutaneous) pacing as a bridge to transvenous pacing or when transvenous pacing is not readily available.
- Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because, unlike true asystole, this is more likely to respond to cardiac pacing.
- If atropine is ineffective and transvenous/transcutaneous pacing is not immediately available, fist pacing can be attempted while waiting for pacing equipment.

Uncontrolled organ donation after circulatory death

- When there is no ROSC, consider uncontrolled organ donation after circulatory death in settings where there is an established programme, and in accordance with local protocols and legislation.

Evidence informing the guidelines

Prevention of in-hospital cardiac arrest

IHCA occurs in about 1.5 patients per 1000 admitted to hospital.[8–12] There are two main strategies to prevent cardiac arrest and the need for attempted CPR:

- Patient-focused decision-making to determine if CPR is appropriate.
- Identifying and treating physiological deterioration early to prevent cardiac arrest.

Emergency care treatment and CPR decisions

Most patients who die in hospital do not have a resuscitation attempt.[13–16] The ERC Guidelines 2025 on Ethics in Resuscitation promote shared decision-making and advanced care planning which integrates resuscitation decisions with emergency care treatment plans to increase clarity of treatment goals and also prevent inadvertent deprivation of other indicated treatments, besides CPR. Further information is provided in the ERC Guidelines 2025 Ethics in Resuscitation.[4]

Physiological deterioration

In-hospital cardiac arrest is often preceded by physiological deterioration.[17,18] This provides an opportunity to recognise deterioration and prevent cardiac arrest. The 5 key steps have been conceptualised as the in-hospital chain of survival: 'staff education', 'monitoring', 'recognition', the 'call for help' and the 'response'.[19] This ERC guidance is based on an ILCOR COSTR and systematic review of adult rapid response systems, and UK guidance for early warning scores and recognising and responding to deterioration of acutely ill adults in hospital, the ILCOR´s 'Ten Steps Toward Improving In-Hospital Cardiac Arrest Quality of Care and Outcomes' and the Society of Critical Care Medicine´s guidelines on "Recognizing and Responding to Clinical Deterioration Outside the ICU".[10,20–23]

Staff education. Education should include knowing the importance of timely and serial measurement of vital signs for the early prediction of patient deterioration, a structured ABCDE-type approach that includes assessment and initial treatment interventions, use of structured communication tools such as Situation-Background-Assessment-Recommendation (SBAR), and how to call for help and escalate care.[21] Staff should also be aware of treatment escalation plans, protocols for critical care admission, implementation of local policies regarding do-not-attempt CPR (DNACPR) decisions and the management of end-of-life care. Timely treatment escalation and DNACPR decisions will avoid ineffective treatments or treatments patients may not wish to have(see Ethics in Resuscitation).[4]

Monitoring. Most patients with IHCA have an initial non-shockable rhythm, and preceding signs of respiratory depression or shock are common.[8,9,24] Although the supporting evidence is low to very low certainty, there is consensus that to help detect deterioration and critical illness early, all patients should have a documented plan for vital sign monitoring that includes which physiological measurements should be recorded and how frequently.[25,26] This can be addressed by using a standardised early warning score (EWS) system for all patients.[27] The choice of system depends on local circumstances and should align with national guidelines. For example in the UK, the National Early Warning Score (NEWS) is endorsed by the National Institute for Health and Care Excellence (NICE) guidelines.[20,21] Higher trained and nurse staffing levels are associated with lower rates of failure to respond to abnormal vital signs, and improved patient outcomes.[28,29] There is a lack of randomised controlled trials (RCTs) or consensus on which patients should undergo continuous ECG or other continuous vital sign monitoring.[12] In a registry-based study, settings where patients are closely monitored were associated with improved survival irrespective of initial rhythm.[30] The use of artificial intelligence to predict patient deterioration has gained interest during recent years but currently the evidence does not support its wider adoption without further studies on effectiveness and impact on clinical management.[31] Implementation of an automated predictive model to identify high-risk patients in 19 United States hospitals was associated with decreased mortality.[32]

Recognition. In non-ICU patients, strategies to simplify and standardise tracking of a patient's condition, and recognising acute illness or deterioration, and triggering a response include early warning score (EWS) systems. These scoring systems have a predefined graded and escalating response according to the patient's EWS. The EWS is used to identify ward patients needing escalation of care, increasing vital sign monitoring, and may improve identification of deterioration, and reduce time to emergency team activation.[33] Clinical concern from nurses and other members of the multidisciplinary team can also indicate patient deterioration.[34,35]

The call for help. All staff should be empowered to call for help and also trained to use structured communication tools such as SBAR to ensure effective communication.[36–38] The response to patients who are critically ill or who are at risk of becoming critically ill is often provided by a rapid response system (which includes medical emergency team (MET), rapid response team (RRT), or critical care outreach team (CCOT)). Any member of the healthcare team can initiate a call to such a team according to explicit activation criteria. In some hospitals, the patient, and their family and friends, are also encouraged to activate the team.[39–41] Such a patient safety initiative is being implemented throughout English hospitals.[42]

Response. The response to patients who are or at risk of being critically ill is often provided by a MET/RRT/CCOT. These teams usually comprise critical care medical and nursing staff who respond to specific calling criteria. They replace or coexist with traditional cardiac arrest teams, which typically only respond to patients already in cardiac arrest. These teams regardless of composition should function 24/7. Systematic reviews, meta-analyses and multicentre studies suggest that RRT/MET/CCOT systems reduce the rate of IHCA and hospital mortality.[43,44] These data led ILCOR to suggest that hospitals consider the introduction of rapid response systems to reduce the incidence of IHCA and in-hospital mortality (weak recommendation, low-certainty evidence).[22] Team interventions often involve simple tasks such as starting oxygen therapy and IV fluids, as well as more complex decision-making such as transferring the patient to the intensive care unit (ICU) or initiating discussions regarding DNACPR, treatment escalation or end-of-life care plans (See ERC Guidelines 2025 Ethics in Resuscitation[4]). An important part of the response is to place a patient at risk of deterioration, or an already deteriorating patient, in an appropriate setting. Patients should be treated in a clinical area that is equipped and staffed to meet the patient's needs. A quality improvement process should be implemented for RRT/MET/CCOT systems.[10]

Prevention of out-of-hospital cardiac arrest

In high-income settings, sudden cardiac death (SCD) is the third leading cause of death. Survival following OHCA is generally no more than 10 % or less,[45–47] and a positive survival trend has been observed in only half of the countries studied recently which underscores the importance of OHCA prevention.[48,49]

Even seemingly healthy young adults who experience SCD may present with prodromal signs and symptoms—such as syncope, presyncope, chest pain, or palpitations—that should prompt healthcare professionals to seek expert evaluation to prevent cardiac arrest.[50–59]

There is no ILCOR systematic review on this topic and existing guidelines of the European Society of Cardiology (ESC), the American Heart Association (AHA) and ERC[60] were also considered.

Epidemiology and pathophysiology of sudden cardiac death

Coronary heart disease (CHD) is the underlying cause of SCD in 80 % of cases, especially in older patients, and non-ischaemic cardiomyopathies account for another 10–15 %.[61] In the young, inherited diseases, congenital heart disease, myocarditis and substance misuse are predominant causes. Knowledge of the causes of SCD assists in early treatment and the prevention of OHCA (Table 2).

Coronary heart disease (CHD)

Arrhythmias triggered by acute myocardial infarction (AMI) or subsequent myocardial scarring can result in SCD.[63] About two-thirds of SCDs occur as the first CHD event or in individuals considered to be at low risk.[61] During the last 50 years primary prevention and secondary revascularisation have reduced CHD age-adjusted mortality.[61] The percentage of SCDs associated with CHD remains unchanged, suggesting that there are interactions between CHD and triggering events such as autonomic nervous system dysfunction, electrolyte disturbances, drug toxicity and individual genetic profiles.[61] Cardiac electrophysiology studies can identify patients with CHD at high versus low risk of SCD.[64] Additional factors such as heart failure (HF) and left ventricular hypertrophy (LVH) predispose to ventricular arrhythmias (polymorphic VT and VF). How to effec-

Table 2 – Causes of sudden cardiac arrest (SCD).

Coronary heart disease
ST-segment elevation myocardial infarction
Other myocardial infarction
Unstable angina
Silent ischaemia
Electrical heart disease, often associated with SCD in the young
Long QT-syndrome (LQTS)
Short QT syndrome
Brugada syndrome
Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)
Triadin knock-out syndrome (TKOS)
Arrhythmogenic bi-leaflet mitral valve prolapse
Drug or medication induced
Non-atherosclerotic coronary artery anomalies
Congenital heart disease
Hypertrophic cardiomyopathy (HCM)
Dilated cardiomyopathy (DCM)
Valvular heart disease

Adapted from Kandala[61] and Winkel.[69]

tively identify patients at high risk of SCD with HF and LVH is uncertain.[65] Changes in left ventricular geometry affect the likelihood of developing VT and VF. The only indicator that has been identified to be consistently associated with an increased risk of SCD in the setting of CHD and left ventricular (LV) dysfunction is LV ejection fraction (LVEF).[63] LVEF is used to indicate the need for an ICD for the primary and secondary prevention of SCD.[66]

Cardiac MRI has been proposed as a tool for detecting scar burden and assessing risk of SCD. Recently, artificial intelligence (AI) and deep learning analysis of the scars have been used to assess patient-specific prognosis.[67] Despite considerable progress, the ability to recognise the risk of SCD before the event remains very limited.[63]

SCD in the young

SCD in the young (SCDY, 5–35 years of age) accounts for 7 % of all SCDs[62]; with an incidence of 1–8/100 000 fatalities per year.[68] In adolescent SCD, 50 % of patients had misinterpreted symptoms before death.[58] CHD is the most frequent cause of explained SCDY, but 25–31 % of the cases remain unexplained after post mortem examination (Sudden Arrhythmic Death Syndrome- SADS).[69] The majority of inherited cardiac diseases can be treated if diagnosed, yet most young SCD victims are not diagnosed.[56] Premonitory signs of SCDY were present in only 29 % in one study, and thus less common than in older patients.[70] QT-prolonging and psychotropic drugs, alone or in combination, increase the risk of SCD.[62] Investigation of cardiac arrest survivors or post mortem examination is crucial to identify inherited cardiac disease in unexplained cases of SCD; this should result in a cardiac investigation of first-degree relatives. In one study, this screening resulted in a diagnosis of an inherited cardiac disease in over half of the families.[71] In a large retrospective SCDY study, a cause was identified in 113/180 patients (62.8 %), the rest were classified as idiopathic VF.[72] With improvements in diagnosis (e.g. provocation drug testing for cardiac channelopathies and coronary vasospasm, genetic testing), the number of unexplained SCDs should decrease.[72] (See ERC Guidelines 2025 Epidemiology in Resuscitation).[48]

Non-atherosclerotic coronary artery anomalies

Coronary artery embolism, coronary arteritis (e.g. Kawasaki disease, polyarteritis nodosa), spasm and myocardial bridging have all been described with SCD.

Congenital heart disease

Congenital coronary anomalies are present in 1 % of all patients. SCD because of congenital coronary anomalies is exercise-related and accounts for 17 % of SCD in young athletes.[61,70]

Hypertrophic cardiomyopathy (HCM)

Hypertrophic cardiomyopathy is the most common genetic disorder of the heart, and the most common cause of SCDY.[73] It often remains clinically silent until SCD presents as the first cardiac event. The incidence of SCD in families with HCM is 2–4 % a year and 4–6 % in children and adolescents.[61]

The 2022 ESC guideline on the management of ventricular arrhythmias and prevention of SCD death proposed 10 new key aspects that may improve the management of SCD.[74] (Table 3).

The prediction of SCD presents an epidemiological paradox: although high-risk patients have a greater individual risk, the absolute number of OHCA is higher in the much larger low-risk general population. Preventing and predicting SCD is challenging, as most events occur in individuals from the general population without known heart disease. In response, *The Lancet Commission on SCD* recently issued a call for multidisciplinary action to reduce the burden of SCD, addressing all aspects of prevention and treatment.[75] The development of high-quality, population-based registries of OHCA is important for improving our understanding and prediction of SCD.

Table 3 – Key points from the European Society of Cardiology guidelines for the treatment of ventricular arrhythmias and sudden cardiac death.

1 Development of public BLS and access to automatic defibrillators
2 Focus on the management of electrical storm
3 Increased relevance of cardiac MRI
4 Increased relevance of catheter ablation
5 Implementation of SCD risk scores and calculators
6 New algorithms for diagnostic evaluation
7 Upgrade of genetic counselling and testing
8 Algorithm for antiarrhythmic drug therapy
9 Individualized risk stratification
10 Change regarding primary electrical diseases

(Adapted from Könemann, 2023[74]).

However, there are currently no established strategies or guidelines for preventing OHCA in the general population.

SCD may be associated with a range of factors—some related to cardiovascular disease, others linked to the broader socioeconomic environment (e.g., obesity, climate, pollution, lifestyle).

Many drugs prescribed commonly (antibiotics, antidepressants) impacting cardiac electrophysiology mainly by prolonging the QT interval, may also increase the risk of OHCA.[76] Recently, proton pump inhibitors, were also identified to be associated with this risk even in patients without cardiovascular disease.[77]

AI and machine learning models offer new possibilities by linking cardiac arrest patient's files and the health records of the general population. It may identify new factors driving risk of SCD and lead to improved targeted screening at the patient level.[78] AI may also help to predict SCD with pulseless electrical activity (PEA) and the understanding of mechanisms and warning symptoms, in this population with a poorer prognosis of survival.[79]

Premonitory signs

Approximately 50 % of cardiac arrests occur in individuals with undiagnosed CHD.[63,80] Many SCD victims have a history of cardiac disease and warning signs before cardiac arrest, most commonly chest or upper abdominal pain or dyspnoea that has not been acted on by the patient or health care professionals.[81,82] Approximately one third of elderly patients will have symptoms in the days or hours before cardiac arrest; primarily chest pain, dyspnoea, syncope, and/or cold sweats.[82,83] In 1960 OHCA patients, 9.4 % had been assessed by an ambulance crew within the preceding 48 h.[84] Emergency care in patients with symptoms is associated with improved survival.[81] Early recognition of acute coronary syndrome (ACS) by EMS teams with 12-lead ECG capabilities and reduction of time to reperfusion may prevent SCD.[85]

Table 4 – High risk features suggesting a serious condition in patients with syncope at initial evaluation in the emergency department.

Syncopal event features
Major
New onset of chest discomfort, breathlessness, abdominal pain or headache[98–100]
Syncope during exertion or when supine[101]
Sudden onset palpitation immediately followed by syncope[101]
Minor
No warning symptoms or short (<10 s) prodrome [101–104]
Family history of SCD at young age[105]
Syncope in the sitting position[106]
Past medical history
Major
Severe structural or coronary artery disease (heart failure, low LVEF or previous myocardial infarction)[98,100]
Physical examination
Major
Unexplained systolic blood pressure <90 mmHg[98,100]
Persistent bradycardia (<40 min^{-1}) in an awake state, in absence of physical training
Undiagnosed systolic murmur
ECG
Major
ECG changes consistent with acute ischaemia
Mobitz II second- and third-degree atrioventricular (AV) block
Slow atrial fibrillation (AF) (<40 min^{-1})
Persistent sinus bradycardia (<40 min^{-1}) or repetitive sinoatrial block or sinus pauses >3 s in an awake state, in absence of physical training
Bundle branch block, intraventricular conduction disturbance, ventricular hypertrophy or Q waves consistent with ischaemic heart disease or cardiomyopathy[99,104]
Sustained and non-sustained VT
Dysfunction of an implantable cardiac device (pacemaker or ICD)
ST-segment elevation with type 1 morphology in leads V1-V3 (Brugada pattern)
QTc >460 ms in repeated 12-lead ECGs indicating long QT syndrome (LQTS)[85]
Minor (high-risk only if history consistent with arrhythmic syncope)
Mobitz I second-degree AV block and 1st degree AV block with markedly prolonged PR interval
Asymptomatic inappropriate mild sinus bradycardia (40–50 bpm.)[104]
Paroxysmal supraventricular (SVT) or atrial fibrillation[107]
Pre-excited QRS complex
Short QTc interval ($\leq$340 ms)[85]
Atypical Brugada patterns[86]
Negative T waves in right precordial leads, epsilon waves suggestive of arrhythmogenic right ventricular cardiomyopathy (ARVC)[86]

Adapted from Brignole 2018.[66] ECG electrocardiogram, ICD implantable cardioverter defibrillator, LVEF left ventricular ejection fraction, SCD sudden cardiac death, VT ventricular tachycardia.

The most effective approach to prevent SCD in the general population remains the quantification of the individual risk of developing CHD, followed by control of risk factors.[86] Syncope can be an important premonitory sign of SCD.

Syncope

Syncope occurring during strenuous exercise, while sitting or in the supine position should always raise the suspicion of a cardiac cause; in other situations it is more likely to be vasovagal syncope or postural hypotension.[85] In patients with known cardiac disease, syncope (with or without prodrome, particularly recent or recurrent) is an independent risk factor for increased risk of death.[66,73,87–97] High-risk (suggesting a serious condition) and low-risk features (suggesting a benign condition) of patients with syncope at initial evaluation in the emergency department have been published by the ESC (Table 4).[66] Early EMS acquisition of a 12 lead-ECG may be helpful.

Screening programs for athletes may be helpful but vary between countries.[108–110] In one study from the United Kingdom between 1996 and 2016, 11,168 athletes received cardiovascular screening and diseases associated with SCD were identified in 0.38 % (n = 42).[111] The incidence of SCD in competitive athletes is higher than in non-athletes.[111] Subpopulations that have been identified as at increased risk include male, black ethnicity, basketball or football players.[112] Screening commonly includes physical examination and ECG. The ECG has a false positive risk because of some ECG features that are particular to athletes. Despite more specialised screening, the risk decreases globally but SCD may still occur. Consequently awareness, CPR training, and availability of AEDs during sport remain important for the protection of athletes.[113,114]

Preventive measures against SCD

Prevention of SCD focuses on identifying and managing medical conditions that may contribute to or exacerbate arrhythmias, assess-

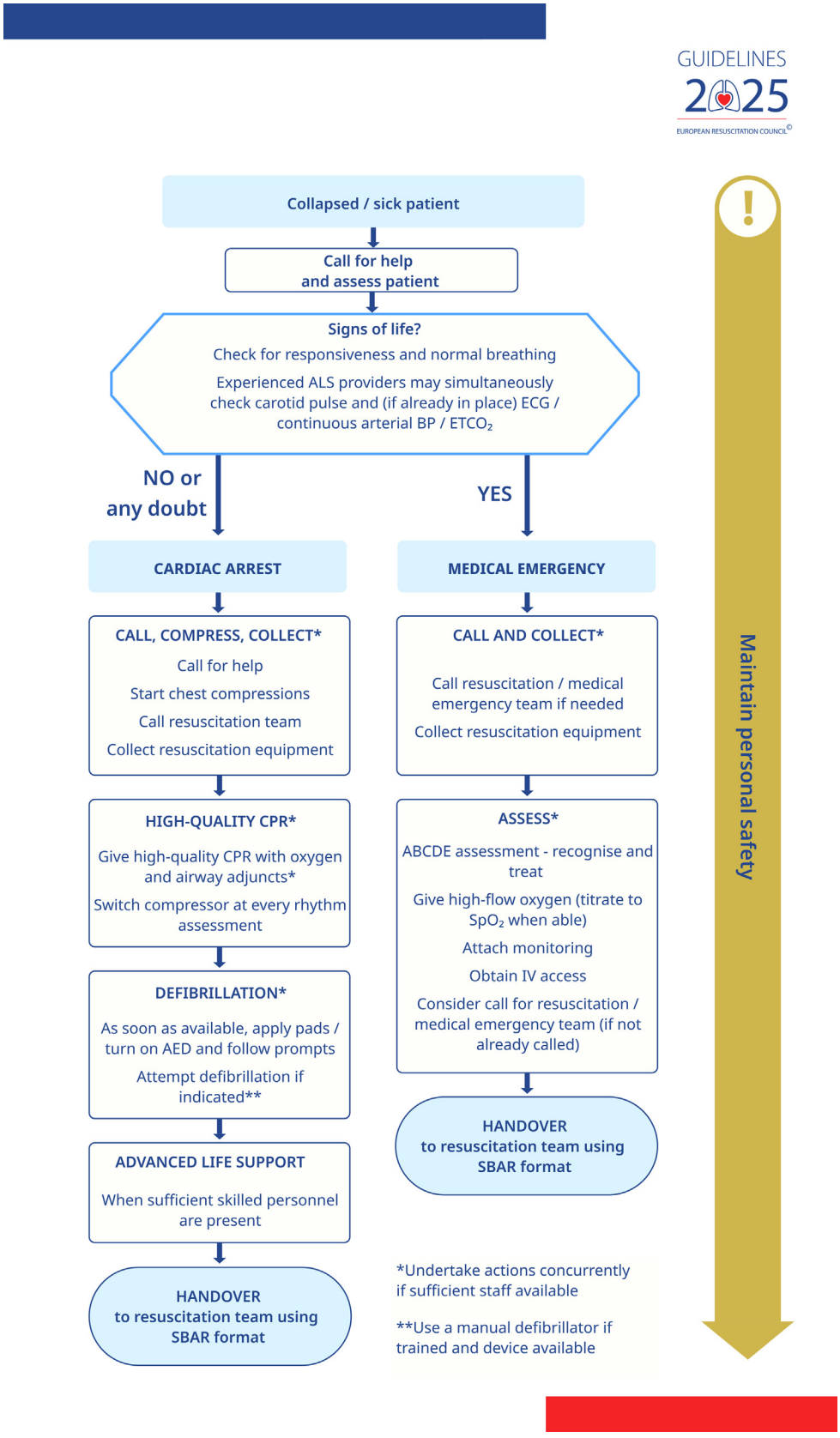

Fig. 3 – In-hospital resuscitation algorithm

ABCDE airway breathing circulation disability exposure; AED automated external defibrillator; ALS advanced life support; BP blood pressure; CPR cardiopulmonary resuscitation; $ETCO_2$ end-tidal carbon dioxide; IV intravenous; SBAR situation, background, assessment, recommendation; SpO_2 oxygen saturation measured with pulse oximetry.

ing the risk posed by the arrhythmia itself, and evaluating the risk–benefit ratio of potential therapies. Interventions may include anti-arrhythmic medications, ICDs, and catheter ablation or surgery.[66,115] The effective management of non-cardiovascular diseases associated with an increased risk of cardiac arrest has also been proposed as a strategy for preventing SCD.[116] For instance, a large registry study found that treatment of sleep apnoea with continuous positive airway pressure (CPAP) was associated with a lower risk of OHCA compared with patients who did not receive treatment.[117]

Non-invasive telemetry or implantable devices transmitting the ECG are currently used in selected groups of patients to detect high risk arrhythmias and prevent SCD. More recently, connected devices with arrhythmia detection capabilities (smartwatch, smartphone applications) have been introduced and may be helpful in detecting asymptomatic AF, however their potential role in the general population to detect SCD arrhythmias is unknown.[118,119] A recent review on smartwatches identified 57 publications including 24 cohort studies mostly focused on AF, and often detected by Apple Watch™.[120] Automated cardiac arrest diagnosis, using smart devices such as wearables and phones, remains an innovative field of research. It promotes the possibility of transforming unwitnessed SCD to witnessed events. However, most of the studies published have been aimed at feasibility and concern a small population.[121] They need to be validated in diverse populations and then integrated into EMS protocols. They carry the risk of false positive alarms which may be responsible for patient anxiety and stress and inappropriate activation of the EMS response, overwhelming the available resources.[122]

Educating the public to report on symptoms before SCD and to help a person in cardiac arrest is important.[81] An awareness campaign on chest pain was associated with an increase in EMS calls and a reduction in OHCA incidence and may serve in part, as an effective primary prevention strategy for OHCA. The campaign period was associated with an 8.8 % (Incident rate ratio [IRR] 1.09, 95 % CI: 1.07, 1.11) increase in the incidence of EMS attendances for chest pain and a 5.6 % (IRR 0.94, 95 % CI: 0.92, 0.97) reduction in OHCA attendances.[123]

Treatment of in-hospital cardiac arrest

Cardiac arrest treatment principles, such as early defibrillation, early delivery of adrenaline and delivery of high-quality CPR, are consistent across both the IHCA and OHCA settings. In the hospital setting, the immediate availability of trained clinical staff and equipment provides an opportunity for the rapid identification of cardiac arrest and initiation of treatment. An IHCA can be defined as any cardiac arrest that occurs on the hospital premises. This may include a cardiac arrest in patients, hospital visitors or staff, in a variety of hospital settings. For IHCA, BLS and ALS interventions can often start and take place at the same time (Fig. 3).

Initial responders

The clinical skill of the initial responder may range from a non-clinical member of staff trained in BLS to an ALS provider. Irrespective of skill level, the initial action of the initial responder is to recognise cardiac arrest, immediately start CPR, call for help and facilitate rapid defibrillation. Delays in starting treatment reduce the likelihood of a successful outcome.[124,125]

The process for calling for help may differ between hospitals or locations within a hospital. If the responder is alone, they may need to leave the patient to call for help. Where a telephone system is used to activate the emergency team, the standard European number (2222) should be used.[126] European Board of Anaesthesiology has also included the 'Cardiac Arrest Call 'telephone number 2222 as a Principal Requirement in the 2025 update of the Helsinki Declaration on Patient Safety in Anaesthesiology 2.0 launched in May 2025. To date, hospital uptake of the standard number across European countries has been variable.[127–129] This is despite changing the number being relatively easy and completely safe if 2222 and the old number are both operated in parallel for a transition period.

Following the completion of initial actions and provided sufficient staff are available, staff should collect ALS equipment and prepare to hand over to the resuscitation team using a standardised communication system, such as SBAR (Situation, Background, Assessment, Recommendation) or RSVP (Reason, Story, Vital Signs, Plan).[36,130,131] Each clinical area in a hospital should consider patient acuity, risk of cardiac arrest, and geographical location (e.g. distance for the resuscitation team to travel) in determining the specific training needs of staff.

Resuscitation team

The resuscitation team may take the form of a traditional cardiac arrest team that responds only to cardiac arrest events or a medical emergency team/ rapid response team (MET/RRT) that responds to both cardiac arrests and critically unwell patients. ILCOR recommends accredited ALS-level training for healthcare staff (strong recommendation based on very low certainty evidence) as this type of training is associated with improved patient outcomes.[22,132] ILCOR also recommends that ALS training incorporates team and leadership training (weak recommendation based on very low certainty evidence) because it is associated with improved patient and process outcomes.[22]

Resuscitation teams often form on an ad hoc basis depending on hospital work rosters and may include individuals from a range of specialities (e.g. emergency medicine, acute medicine, cardiology, critical care, anaesthesia). Lack of knowledge of team member roles, including who is acting as team leader can lead to errors during ALS for IHCA.[133,134] A team meeting at the beginning of each shift for introductions and allocation of roles is straightforward to implement and may support effective team-working during resuscitation, although its effect on patient outcomes is uncertain.[135]

Equipment

Hospitals should ensure that clinical areas have immediate access to resuscitation equipment and medication to facilitate rapid resuscitation of the patient in cardiac arrest. Missing or malfunctioning equipment contributes to treatment delays.[133,136] Equipment should be standardised throughout the hospital and regularly checked to ensure proper functioning. In contrast to OHCA, most patients who have an IHCA will have vascular access at the time of cardiac arrest, facilitating rapid administration of time-critical drugs, such as adrenaline.[137–139]

Termination of CPR rules for in-hospital cardiac arrest

Research on termination of resuscitation (TOR) rules for IHCA remains limited. An ILCOR systematic review published in 2021 identified only very low-certainty evidence for a single clinical decision rule, the UN10 rule, which included three variables: unwitnessed, initial non-shockable rhythm, and resuscitation duration of more than 10 min.[140–142] While the rule had a false-positive rate of 0 % in the derivation cohort, later validation revealed a false-positive rate above the 1 % threshold deemed acceptable for clinical use.[4] Consequently, ILCOR concluded that no identified tool was reliable in predicting death after IHCA and specifically recommended against using the UN10 as a sole strategy for termination of resuscitation in IHCA (strong recommendation, very low–certainty evidence).[143]

Following the 2021 ILCOR systematic review, a Scandinavian study enrolling IHCA patients from Denmark, Sweden, and Norway developed and validated five TOR rules for IHCA.[144] The best-performing rule included four variables (unwitnessed, unmonitored, initial rhythm of asystole, and resuscitation duration of 10 min or more). This rule incorrectly predicted 30-day mortality in 6 per 1000 cases, and proposed termination in 110 per 1000 cardiac arrests, potentially reducing futile resuscitation attempts. Notably, a large observational study demonstrated that survival to hospital discharge is possible even after prolonged IHCA (>1 h), although survival rates were less than 1 % after 40 min of resuscitation.[145] An Austrian study found that machine learning models effectively predicted failure to achieve ROSC and poor functional outcomes while CPR was ongoing; however, the positive predictive value was insufficient to justify early termination of resuscitation efforts.[146]

In alignment with ILCOR, the ERC does not recommend using TOR rules as the sole basis for TOR in IHCA. While the results of the Scandinavian study are promising, further external validation is needed before the rule can be considered for use in clinical practice.[144] TOR rules should be validated in local, regional, or national cohorts before implementation and revisited as survival rates change. Decisions to terminate resuscitation should also consider the local legal, organisational, and cultural context. The ERC Guidelines 2025 on Ethics in Resuscitation provide additional guidance on the use of termination of resuscitation rules.[4]

Treatment of out-of-hospital cardiac arrest

This section provides an overview of specific ALS issues related to resuscitation for OHCA. Further information is available in the ERC Guidelines 2025 Basic Life Support, Cardiac Arrest in Special Circumstances, Systems Saving Lives, Epidemiology, Post Resuscitation Care, and Ethics in Resuscitation.[4,6,114,129,147,148]

Transfer of patients with OHCA

The aim of ALS for OHCA is to provide the same interventions as available in hospital as early as possible, and to rapidly transfer the patient to hospital for those interventions that are not feasible out-of-hospital. A recent systematic review addressed the benefit of rapid transport from the scene to definitive in-hospital care versus extended on-scene resuscitation in OHCA.[149] Nine studies (8 cohort studies, one RCT) were included. In pooled analysis, expedited (or earlier) transfer was not predictive of survival to discharge (odds ratio [OR] 1.16, 95 % confidence interval [CI] 0.53 to 2.53, $I^2 = 99$ %, $p = 0.65$) or favourable neurological outcome (OR 1.06, 95 % CI 0.48 to 2.37, $I^2 = 99$ %, $p = 0.85$). The certainty of evidence was assessed as very low with a moderate risk of bias. Significant heterogeneity was observed mostly related to the region of the EMS studied. In a large North American registry a propensity-matched cohort, which included 27,705 patients, survival to hospital discharge occurred in 4.0 % of patients who underwent intra-arrest transport compared with 8.5 % who received on-scene resuscitation (risk difference, 4.6 % [95 % CI, 4.0–5.1 %]).[150] The decision regarding at which stage conveyance to hospital should be undertaken will depend on EMS system factors that include the clinical skills of rescuers, the ALS interventions available on scene, and the use of termination of resuscitation protocols.[149]

Care at cardiac arrest centres

An ILCOR systematic review assessed the benefits of care at a dedicated cardiac arrest centre. The resulting ILCOR treatment recommendations include[151]:

- Adult patients with non-traumatic OHCA should be considered for transport to a cardiac arrest centre, according to local protocols.
- Adult patients with non-traumatic OHCA should be cared for at a cardiac arrest centre whenever possible.
- Health care networks should establish local protocols to develop and maintain a cardiac arrest network.

The ERC has adopted these recommendations on cardiac arrest centres and further details can be found in ERC Guidelines 2025 Systems Saving Lives and Post Resuscitation Care.

Initial treatment of OHCA

Several patient and CPR factors affect outcome from OHCA (Table 5). Community programmes of lay bystander CPR and AED use improve outcome from OHCA.[152] Chest compressions and early defibrillation are the cornerstones of CPR in OHCA. The only definitive treatment for VF remains prompt defibrillation.[153]

EMS personnel and interventions

ILCOR conducted a systematic review examining how EMS exposure to and experience with OHCA impacts outcomes.[154] The largest study in this review studied the association between the number of times a paramedic had attended an OHCA with patient survival to hospital discharge.[155] Increasing paramedic exposure to OHCA in the preceding three years was associated with increased survival to discharge: ≤6 exposure (control group), >6–11 exposures (adjusted odds ratio [aOR] 1.26, 95 % CI 1.04–1.54), 11–17 exposures (aOR 1.29, 95 %CI 1.04–1.59), >17 exposures (aOR 1.50, 95 %CI 1.22–1.86).[155] Another large observational study reported that increased exposure of the treating paramedic was associated with increased ROSC (<15 exposures [control group] vs. ≥ 15 exposures, aOR 1.22, 95 %CI 1.11–1.36).[156] The ILCOR CoSTR concluded that EMS should monitor exposure of their clinical personnel to resuscitation and implement strategies to address low exposure or ensure that treating teams have members with recent exposure (weak recommendation, very-low certainty of evidence).[23]

There is no recommendation on the optimal number of members in prehospital ALS teams. A recent review of 22 articles published between 2005 and 2023 assessed the effectiveness of ALS CPR performed by two-member teams and found insufficient evidence to support adapting ALS protocols to such settings. [157] A recent review in 2023 identified four non-RCT studies and concluded that

Table 5 – Patient and resuscitation factors affecting outcome from OHCA.

Patient
Age
Sex
Comorbidities (cardiac, pulmonary, renal, trauma)
Special circumstances
Cardiopulmonary resuscitation
Location (private vs. public)
Witnessed vs. unwitnessed cardiac arrest
Bystander CPR
Type of bystander CPR (compression only vs. standard)
First cardiac arrest rhythm
Use of AED by bystander
Time to return of spontaneous circulation

Adapted from Kandala 2017.[61] AED denotes automated external defibrillator, CPR cardiopulmonary resuscitation.

prehospital ALS care with a ratio of on-scene ALS-trained personnel >50 % could improve survival-to-discharge with a certainty of evidence rated as very low.[158] A large national cohort analysis conducted in a system with prehospital ALS and the initial dispatch of more than one EMS crew found both a higher number of EMS crew members (three or more) and a higher proportion of ALS providers in the first-contact EMS crew. This was associated with improved neurological recovery in adults with non-traumatic OHCA. Specifically, good neurological recovery was associated with an adjusted odds ratio (95 % confidence interval) of 1.23 (1.06–1.43) for three-member crews, and 1.28 (1.17–1.40) for crews with a higher proportion of ALS-trained providers.[159]

A 2024 ILCOR review compared prehospital critical care for OHCA with standard prehospital ALS.[23,160] Prehospital critical care was defined as care involving enhanced clinical competencies beyond standard ALS, delivered by dedicated EMS teams dispatched to critically ill patients. These teams were staffed by physicians (specialised in emergency medicine, anaesthesia, critical care, or intensive care) or specially trained critical care paramedics. This was compared with standard prehospital ALS. The review included 15 articles. Prehospital critical care was associated with improved outcomes for several measures: survival to hospital admission, ROSC (OR 1.95, 95 % CI 1.35–2.82), survival to hospital discharge (OR 1.34, 95 % CI 1.10–1.63), 30-day survival (OR 1.56, 95 % CI 1.38–1.75), and favourable neurological outcome at 30 days (OR 1.56, 95 % CI 1.38–1.75). ILCOR recommends that adults with non-traumatic OHCA receive care from prehospital critical care teams in EMS systems with sufficient resource infrastructure (weak recommendation, low certainty of evidence). However, the included studies did not report on resource costs, cost-effectiveness, impact on health equity, or implementation feasibility; therefore, these aspects were not analysed.

Prehospital critical care teams also provide the opportunity to complement ALS with more advanced and invasive resuscitation techniques, such as ECPR, balloon occlusion, and emergency thoracotomy.[161] These techniques, their targets populations, and their potential impacts on outcome are discussed in other parts of the guidelines.

Termination of resuscitation rules for out-of-hospital cardiac arrest

Termination of resuscitation (TOR) rules guide EMS in deciding whether to continue resuscitation or transport a patient with ongoing CPR. An ILCOR review found that current TOR rules may result in some missed survivors but do also prevent premature termination of resuscitation.[1,23,162] ILCOR makes a conditional recommendation based on very low certainty evidence that EMS systems may use TOR rules to guide decisions on stopping resuscitation or transporting with ongoing CPR – the rules should only be implemented after local validation, ensuring acceptable specificity and alignment with local culture, values, and context. Further ethical guidance is available in the ERC Guidelines 2025 Ethics in Resuscitation.[4]

Debriefing

In 2020, ILCOR conducted a systematic review of debriefing following cardiac arrest[22], including four observational studies.[163–166] At that time, debriefing was associated with improved hospital survival, ROSC, and CPR quality. In 2024, ILCOR conducted a new systematic review, incorporating ten non-randomised studies—six involving adult patients[135,163,165–168] one paediatric,[164] and two neonatal cardiac arrest.[169,170] This updated review revealed that, despite the very low certainty of evidence because of significant risks of bias and inconsistency, post-event debriefing was either associated with no effect or with improvements in ROSC, survival to hospital discharge, favourable neurological outcomes, and enhanced CPR quality. The ILCOR review did not identify any negative consequences, such as emotional trauma to the debriefed team or significant resource demands (including costs), associated with debriefing after cardiac arrest in the studies examined. Based on these findings, ILCOR suggests, and the ERC recommends, implementing post-event debriefing following adult cardiac arrest (weak recommendation, very low certainty of evidence). This recommendation stems from the review's conclusion that debriefing has a neutral to positive impact on critical and important outcomes, which likely outweighs any potential undesirable effects. The ERC Guidelines 2025 Education for Resuscitation include further details on these issues.[5]

ALS in low-resource settings

This review was informed by a narrative ILCOR review.[171] Low-resource settings in CPR are most often associated with low-income countries. High-income countries may also have scenarios where the resources are low (e.g., mass-casualty incidents, at night, or during bad weather, natural disasters, pandemics or war) or due to location (e.g., difficult access – mountains and sea, remote locations – aircraft, oil platforms, ships). Implementation of resuscitation guidelines from well-resourced settings may not be applicable in low-resource settings because of lack of logistics, personnel and infrastructure. Several organisations from the global south and the World Health Organization (WHO) have developed and are further developing resuscitation guidelines tailored to low-resource settings.[171–191] These guidelines often focus on emergency prevention, first aid and basic life support. The ERC Guidelines 2025 Special Circumstances have addressed some settings which can also have low resources, such as cardiac arrest inflight, on cruise ships and mass casualty incidents.[114,186] An adapted approach towards the chain of survival has been proposed to address the different needs and opportunities in low-resource settings[171] and is described in more detail in the ERC Guidelines for Systems Saving Lives.[6]

CPR-induced recovery of consciousness

CPR induced consciousness (CPRIC) occurs when the patient is conscious during CPR but has not achieved ROSC. This guidance is based on an ILCOR summary statement 2024,[192] which is an update of the previous 2021 summary statement[193] and a scoping review from 2022, which included eight observational studies, 26 case studies, and three reviews.[194] A 2025 scoping review identified two additional observational studies,[195,196] a case series,[197] and one review on prehospital guidelines to treat CPR-induced consciousness.[198] ILCOR has made the following good practice statements:

- In settings in which it is feasible, rescuers may consider using sedative or analgesic drugs (or both) in very small doses to prevent pain and distress to patients who are conscious during CPR.
- Neuromuscular-blocking drugs alone should not be given to conscious patients.
- The optimal drug regimen for sedation and analgesia during CPR is uncertain. Regimens can be based on those used in critically ill patients and according to local protocols.

CPR-induced consciousness can be defined as 'a demonstration of consciousness whilst undergoing CPR with no measurable spontaneous cardiac output displayed'.[194] The incidence of CPR-induced conscious-

ness is 0.23–0.90 % in observational studies of patients[199] and 48–57 % of experienced healthcare professionals have reported observing patients with CPR-induced consciousness.[200] CPR-induced consciousness is associated with witnessed and shockable cardiac arrests, younger age, and better outcomes.[197] Longer CPR attempts in patients with CPR-induced consciousness may be reasonable.[197]

Patients with CPR-induced consciousness may interfere with and prevent effective CPR[194,197] and sedation may be required. Consider using sedatives or analgesics (or both) in very small doses to prevent pain and distress in patients who are conscious or where consciousness cannot be ruled out during CPR. The optimal drug regimen for sedation and analgesia during CPR is uncertain, and local and national protocols exist.[198] Use drug regimens based on those used in critically ill patients such as small doses of fentanyl, ketamine and/or midazolam. Do not give neuromuscular blocking drugs alone to conscious patients. Consciousness during CPR or awareness without visible consciousness may lead to post-traumatic stress disorder for clinicians, bystanders, and cardiac arrest survivors.[196,201]

ALS treatment algorithm

Cardiac arrest is associated with either shockable rhythms (VF/pVT) or non-shockable rhythms (asystole and PEA). The main difference in the treatment of shockable rhythms is the need for attempted defibrillation. Other interventions, including high-quality chest compressions with minimal interruption, airway management and effective ventilation, venous access, administration of adrenaline and the identification and treatment of reversible causes, are common for all arrests. The ALS algorithm (Fig. 2) provides an overview of these key interventions. These are based on the expert consensus of the ERC ALS Writing Group. The ALS cardiac arrest algorithm is applicable to all adult cardiac arrests. Additional interventions may be indicated for cardiac arrest caused by special circumstances.[114,202] To improve understanding of timing of shocks and drugs we have provided some example flow charts – these examples do not cover every possible scenario or transition between cardiac arrest rhythms or ROSC (QR code 1). Although asystole and PEA are both treated as non-shockable rhythms, the treatment of PEA can be more nuanced in highly monitored settings (e.g., with continuous invasive blood pressure monitoring) and this is addressed in the sections below on ALS in highly monitored settings, and physiologically guided CPR.

QR code 1 – Four examples on advanced life support for timing of shocks and drugs (not every possible scenario is included).

Defibrillation

Automated external defibrillation versus manual defibrillation during ALS

This guidance is based on an ILCOR Evidence update from 2020[203] and a scoping review from January 2020 to January 2025 which identified three systematic reviews,[204–206] and two observational studies.[207,208]

ALS providers require frequent training and advanced ECG recognition skills for manual defibrillation.[203] They should preferentially use manual defibrillation but also be skilled in AED use. Despite the faster time to first shock with AEDs, studies suggest that this does not translate into improved survival when used instead of manual defibrillators. For instance, a study in paramedics reported that while AEDs improved the time to first shock within 2 min (aOR 1.72; 95 % CI 1.32–2.26; $P < 0.001$), they were associated with a reduction in survival to hospital discharge (aOR 0.71; 95 % CI 0.55–0.92; $P = 0.009$), event survival (aOR 0.74; 95 % CI 0.62–0.88; $P = 0.001$), and prehospital ROSC (aOR 0.81; 95 % CI, 0.68–0.96; $P = 0.01$) compared with manual defibrillation.[209] Manual defibrillation has been associated with shorter pauses in chest compressions, which is critical for maintaining coronary and cerebral perfusion during resuscitation.[210] Software that filters chest compression artefacts and enables rhythm analysis during CPR and performing chest compressions while charging for defibrillation also provides an advantage for manual defibrillation over use of AEDs.

In two-tier systems, ALS providers may arrive after an AED has already been attached to the patient and is in use. If an AED is already in use when ALS providers arrive, they should follow its shock prompts. When possible, they should transition to a manual defibrillator during a 2-minute CPR cycle. There is insufficient evidence to recommend the placement of an additional manual defibrillator in a vector-changing position when an AED is already in place.[211]

Compared with following AED prompts, manual defibrillation may result in more inappropriate defibrillation shocks (because a shock would not have been advised by an AED), and more missed shocks (because a shock would have been advised by an AED).[212] A shock should be given if the ALS provider is in doubt whether fine VF or asystole are displayed on the monitor. In recommending this the ERC balanced the risks of not shocking VF versus shocking a patient in asystole, and variability between rescuers in deciding if a rhythm is VF or fine VF. If rescuers are not confident in making shockable versus non-shockable rhythm decisions rapidly (within 5 s) during a resuscitation attempt they should use the defibrillator in an AED mode.

Manual defibrillation

Attempted defibrillation is a vital component of CPR as it has the potential to terminate VF/pVT and achieve ROSC. Although defibrillation is indicated in approximately 20 % of cardiac arrests, more than 80 % of those who survive present in a shockable rhythm.[213] As defibrillation effectiveness decreases with both time and VF duration, defibrillation attempts must be timely, whilst remaining efficient and safe.[214] Knowledge of how to use a defibrillator (manual or AED) is key for rescuers performing ALS. Rescuers who use a manual defibrillator should aim to take less than 5 s to recognise a shockable cardiac arrest rhythm and make the decision to give a shock to minimise interruption to chest compressions. If the delay exceeds 5 s, consider resuming chest compressions, switch the device to AED mode and then allow the device to analyse immediately.

Since 2015, ERC defibrillation guidelines have referred solely to biphasic energy waveforms and defibrillation pads. In this ERC Guidelines 2025 ALS, we reintroduce recommendations on the use of defibrillation paddles which in some countries, remain in clinical use.[215] The evidence for this section is based on ILCOR 2020 CoSTRs, the ERC 2015 ALS Guidelines, and expert consensus.[152,203,215]

Strategies for minimising the peri-shock pause. The delay between stopping chest compressions and shock delivery (the pre-shock pause) must be kept to an absolute minimum; even a 5–10 s delay will reduce the chances of the shock being success-

ful.[216–221] The pre-shock pause can be reduced to less than 5 s by continuing compressions during charging of the defibrillator and by having an efficient team coordinated by a leader who communicates effectively.[222,223] The safety check to avoid rescuer contact with the patient at the moment of defibrillation should be undertaken rapidly but efficiently. The delay between shock delivery and recommencing chest compressions (the post-shock pause) is minimised by immediately resuming chest compressions after shock delivery.[224] If there are both clinical and physiological signs of ROSC (e.g., return of consciousness, movement, arterial waveform, increase in $ETCO_2$), chest compressions can be paused briefly for rhythm analysis. The entire process of manual defibrillation should be achievable with less than a 5 s interruption to chest compressions.

CPR versus defibrillation as the initial treatment. A 2020 ILCOR systematic review addressed whether a specified period (typically 1.5–3 min) of chest compressions before shock delivery compared with a short period of chest compressions (to enable defibrillator start-up) before shock delivery affected resuscitation outcomes. Outcomes were no different when CPR was provided for up to 180 s before attempted defibrillation, compared with rhythm analysis and attempted defibrillation first.[152] Therefore, the routine delivery of a pre-specified period of CPR (e.g., 2–3 min) before rhythm analysis and shock delivery is not recommended. ILCOR made a weak recommend based on low certainty evidence for rescuers providing a short period of CPR until the defibrillator is ready for rhythm analysis in unmonitored cardiac arrest. Defibrillation should then be delivered as indicated, without delay. Immediate defibrillation of VF of any amplitude should be attempted at the end of each 2-minute cycle.

Anticipatory defibrillator charging. Using this method, the defibrillator is charged as the end of a compression cycle is approached, but before the rhythm is checked – this method is also called 'pre-charging'. When compressions are paused briefly to check the rhythm, a shock can be delivered immediately (if indicated) from a defibrillator that is already charged, thereby avoiding a period of further chest compressions while the defibrillator is being charged. This method was reviewed by ILCOR in 2020 as the technique is already in use in some countries as an alternative to the conventional sequence.[225] Manikin studies show anticipatory charging is feasible, and can reduce the overall number of interruptions to chest compression, but can increase pre-, post, and peri-shock pause duration. A Danish study showed an association between pre-charging and the total hands-off fraction, a longer pre-shock pause, and increased ROSC.[226] This technique may be a reasonable alternative for use by well-drilled teams that can minimise pre- post, and peri-shock pause duration. Further clinical studies are required to determine the best technique for manual defibrillation.

Safe use of oxygen during defibrillation

In an oxygen-enriched atmosphere, sparking from poorly applied defibrillator paddles can cause a fire and significant burns to the patient.[227–232] Although defibrillation pads may be safer than paddles with regards to arcing and spark generation, recommendations for the safe use of oxygen during defibrillation remain unchanged in this ERC Guidelines 2025 ALS. The use of non-invasive ventilation and high flow nasal oxygen increases the risk of an oxygen enriched environment. The risk of fire during attempted defibrillation can be minimised by taking the following precautions:

- Take off any oxygen mask (including bag mask) or nasal cannulae and place them at least 1 m away from the patient's chest.
- Leave the ventilation bag or ventilation circuit connected to the tracheal tube or supraglottic airway, and ensure any oxygen exhaust is directed away from the chest.
- If the patient is connected to a ventilator, for example in the operating room or critical care unit, leave the ventilator tubing (breathing circuit) connected to the tracheal tube. Oxygen exhaust from ventilators should be directed away from the chest.

Pad contact with the chest and anatomical position

A 2024 ILCOR systematic review found no RCTs since the 2021 guidelines regarding optimal defibrillation pad position and evidence from two observational studies in OHCA patients was rated as very low certainty.[192]

The techniques described below aim to place external defibrillation pads (self-adhesive pads) in an optimal position to maximise transmyocardial current density and minimise transthoracic impedance. No human studies have evaluated the pad position as a determinant of ROSC or survival from VF/pVT.[152] Transmyocardial current during defibrillation is likely to be maximal when defibrillation pads are placed so that the area of the heart that is fibrillating lies directly between them (i.e. ventricles in VF/pVT, atria in AF). Therefore, the optimal pad position may not be the same for ventricular and atrial arrhythmias. The antero-lateral pad position is preferred as the initial pad position for VF/VT because it is easier to place and avoids interruptions to CPR while the posterior pad is positioned.

Pad placement for ventricular arrhythmias and cardiac arrest. In adults, place defibrillator pads or paddles in the antero-lateral position to optimise placement speed and minimise interruptions to chest compressions. One pad/paddle should be positioned below the patient's right clavicle, just to the right of the upper sternal border. The other pad/paddle should be placed on the patient's left mid-axillary line, below the armpit (Fig. 4).

In adults, if the initial antero-lateral position is not feasible, consider using the antero-posterior pad position if trained (Fig. 5). Place the anterior pad on the left side of the chest, between the midline and the nipple. For female patients, place the anterior pad to the left of the lower sternum, ensuring it avoids breast tissue as much as possible.[233] The posterior pad should be placed on the left side of the patient's spine, just below the scapula.

A study of initial antero-posterior (AP) versus antero-lateral (AL) pad positioning on outcomes in patients with shockable OHCA due to VF or pVT found that initial AP pad placement was significantly associated with higher odds of achieving ROSC at any time compared to AL placement (aOR 2.64; 95 % CI, 1.50–4.65). Approximately 74.1 % of patients with AP placement achieved ROSC versus 50.5 % with AL placement. Given the limitations of a non-randomised study and the potential delays in applying AP rather than AL pads, we continue to recommend AL pads as the preferred initial pad position.[234]

Other acceptable pad positions include:

- Placement of each pad on the lateral chest walls, one on the right and the other on the left side (bi-axillary).
- One pad in the standard axillary position and the other on the right upper back.

Either pad can be placed in either position (apex or sternal). An observational study in patients undergoing elective cardioversion with external defibrillator paddles showed that transthoracic impe-

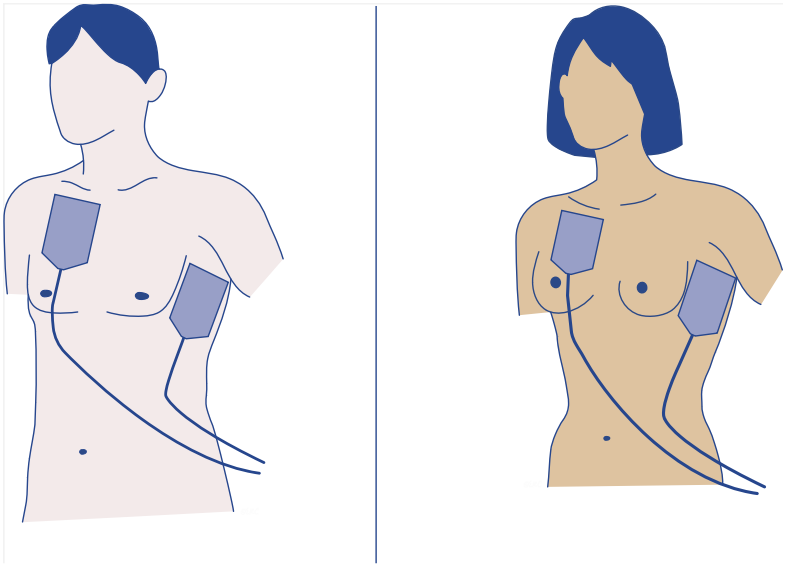

Fig. 4 – Correct antero-lateral pad placement for defibrillation.

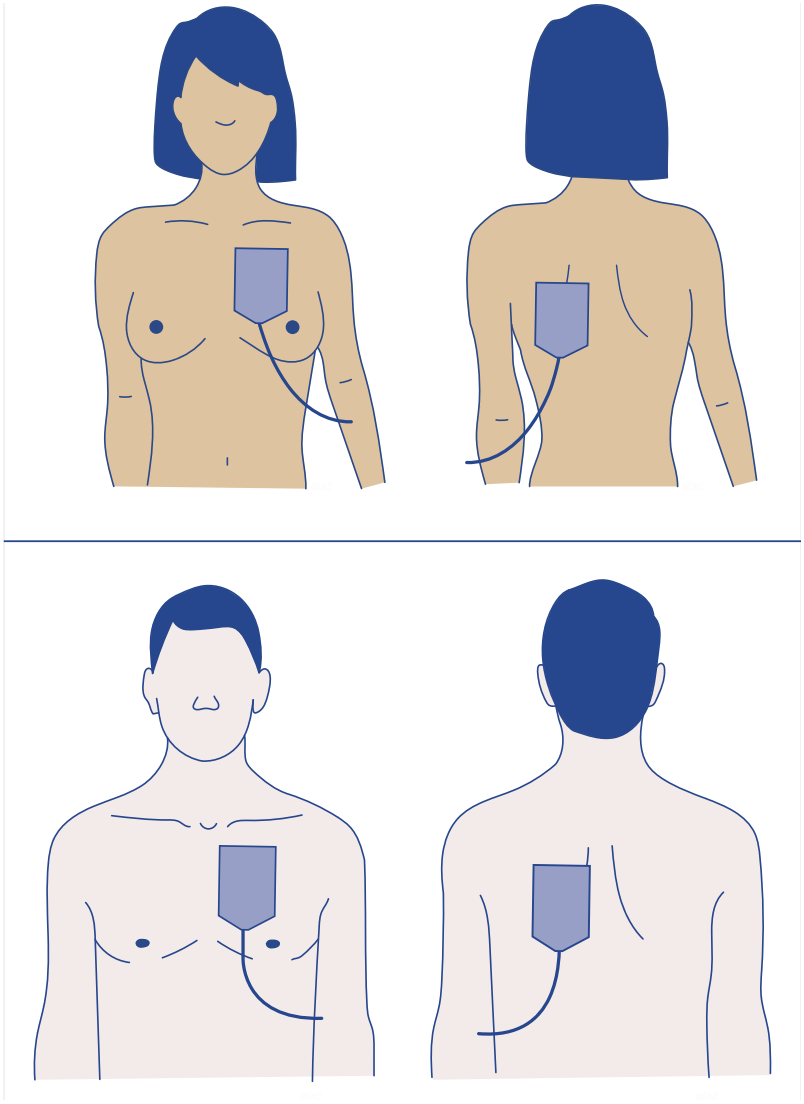

Fig. 5 – Antero-posterior pad placement.

dance was lower when the paddle was oriented in a cranio-caudal direction.[235] Consider shaving the chest if it is very hairy and the electrodes will not stick firmly. Do not delay shock delivery, and consider alternative pad positions if necessary.

Pad placement for atrial arrhythmias. Atrial fibrillation is usually maintained by functional re-entry circuits in the left atrium. As the left atrium is located posteriorly in the thorax, pad positions that result in a more posterior current pathway may theoretically be more effective for atrial arrhythmias. Although some studies have shown that antero-posterior pad placement is more effective than the traditional antero-apical position in elective cardioversion of atrial fibrillation,[236,237] the majority have failed to show any clear advantage of any specific pad position.[238–241] Efficacy of cardioversion may be less dependent on pad position when using biphasic impedance-compensated waveforms.[240–242] The following pad positions are safe and effective for cardioversion of atrial arrhythmias:

- Traditional sternal-apical position.
- Antero-posterior position (one pad anteriorly, over the left precordium, and the other pad posteriorly to the heart, just below the left scapula).

Pad placement to avoid implantable medical devices. More patients are presenting with implantable medical devices (e.g. permanent pacemaker, implantable cardioverter defibrillator (ICD)). Medic Alert bracelets are recommended for these patients. Implantable medical devices may be damaged during defibrillation if current is discharged through defibrillation pads placed directly over the device.[243,244] Place the pad away from the device (at least 8 cm) or use an alternative pad position (antero-lateral, antero-posterior).[243,245,246]

Hands-on defibrillation

By allowing continuous chest compressions during the delivery of the defibrillation shock, hands-on defibrillation would minimise peri-shock pause and enable continuation of chest compressions during defibrillation. However, the benefits of this approach are unproven, and further studies are required to assess the safety and efficacy of this technique. A post-hoc analysis of a multi-centre trial did not observe any benefit when shocks were delivered without pausing manual or mechanical chest compressions.[246] Only Class 1 electrical safety gloves, but not standard clinical gloves (or bare hands), provide a safe level of electrical insulation for hands-on defibrillation.[247] There have been no new studies since the 2021 guidelines and the ERC recommendation 2025 therefore remains unchanged.[60,215]

Respiratory phase

High levels of positive end expiratory pressure (PEEP) increase transthoracic impedance and should be minimised where possible during defibrillation. Auto-PEEP (gas trapping) may be particularly high in patients with asthma and may necessitate higher than usual energy values for defibrillation.[248]

One shock versus three stacked shock sequence

In 2010, it was recommended that when defibrillation was required, a single shock should be provided with immediate resumption of chest compressions after the shock.[249] This recommendation was made for two reasons. Firstly, to minimise peri-shock interruptions to chest compressions and secondly, given the greater efficacy of biphasic shocks, if a biphasic shock failed to defibrillate, a further period of chest compressions could be beneficial. Studies have not shown that any specific shock strategy is of benefit for any survival endpoint.[250,251] There is no conclusive evidence that a single-shock strategy is of benefit for ROSC or recurrence of VF compared with three stacked shocks, but given the evidence suggesting that outcome is improved by minimising interruptions to chest compressions, the ERC continued in 2020 to recommend single shocks for most situations.

When defibrillation is warranted, give a single shock and resume chest compressions immediately following the shock.[152] Do not delay CPR for rhythm reanalysis or a pulse check immediately after a shock. Continue CPR for 2 min until rhythm reanalysis is undertaken and another shock given (if indicated). Even if the defibrillation attempt is successful, it takes time until the post shock circulation is established and it is very rare for a pulse to be palpable immediately after defibrillation.[252,253] Patients can remain pulseless for over 2 min and the duration of asystole before ROSC can be longer than 2 min in as many as 25 % of successful shocks.[254] In patients where defibrillation achieves a perfusing rhythm, the effect of chest compressions on re-inducing VF is not clear.[255]

Monitored and witnessed shockable cardiac arrest. If a patient has a monitored and witnessed cardiac arrest (e.g. in the catheter laboratory, coronary care unit, or other monitored critical care setting in or out-of-hospital) and a manual defibrillator is rapidly available:

- Confirm cardiac arrest and shout for help.
- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks.
- Rapidly check for a rhythm change and, if appropriate, ROSC after each defibrillation attempt.
- Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful.

This three-shock strategy may also be considered for an initial, witnessed VF/pVT cardiac arrest if the patient is already connected to a manual defibrillator. Although there are no data supporting a three-shock strategy in any of these circumstances, it is unlikely that chest compressions will improve the already very high chance of ROSC when defibrillation occurs early in the electrical phase,[256] immediately after onset of VF/pVT. For giving drugs after three stacked shocks:

- Give the first dose of amiodarone (or lidocaine) after the third shock if VF/pVT persists.
- Consider the stacked shocks as the first shock in the ALS algorithm for the purposes of adrenaline and amiodarone dosing.

Waveforms

Biphasic waveforms are now well established as a safe and effective waveform for defibrillation. Biphasic defibrillators compensate for the wide variations in transthoracic impedance by electronically adjusting the waveform magnitude and duration to ensure optimal current delivery to the myocardium, irrespective of the patient's size (impedance compensation). There are two main types of biphasic waveform: the biphasic truncated exponential (BTE) and rectilinear biphasic (RLB). A pulsed biphasic waveform is also in clinical use, in which the current rapidly oscillates between baseline and a positive value before inverting in a negative pattern.[33]

Energy levels

Defibrillation requires the delivery of sufficient electrical energy to defibrillate a critical mass of myocardium, abolish the wavefronts of VF and enable restoration of spontaneous synchronised electrical activity in the form of an organised rhythm. The optimal energy for defibrillation is that which achieves defibrillation whilst causing the minimum of myocardial damage.[257] Selection of an appropriate energy level also reduces the number of repetitive shocks, which in turn limits myocardial damage.[258]

Optimal energy levels for defibrillation are unknown. The recommendations for energy levels are based on a consensus following careful review of the current literature. Although delivered energy levels are selected for defibrillation, it is the transmyocardial current that achieves defibrillation; the electrical current correlates well with successful defibrillation and cardioversion.[259] A recent large database study revealed no significant association between initial energy level and outcomes, such as ROSC or survival to hospital discharge.[260] Defibrillation shock energy levels for the common BTE and RLB waveforms are unchanged from the 2021 guidelines, with pulsed biphasic energy levels increased to a minimum to 130 J to reflect the clinical evidence.[60,261] Studies of defibrillation energy levels in obese patients have generally found that defibrillation efficacy for any given energy level is unchanged.[262]

First shock. Few studies have been published with which to refine the current defibrillation energy levels set in the 2010 guidelines.[263] There is no evidence that one biphasic waveform or device is more effective than another. First shock efficacy of the BTE waveform using 150–200 J has been reported as 86–100 %.[264–269] First shock efficacy of the RLB waveform using 120 J has been reported as 85 %.[270] Four studies have suggested equivalence with lower and higher starting energy biphasic defibrillation (BTE waveform)[271–274] although one has suggested that initial low energy (150 J) defibrillation is associated with better survival.[275] Although human studies have not shown harm (raised biomarkers, ECG changes, ejection fraction) from any biphasic waveform up to 360 J[271,276], several animal studies have suggested the potential for harm with higher energy levels.[277–280]

The initial biphasic shock should be no lower than 150 J for RLB and BTE waveforms. For pulsed biphasic waveforms, begin at 130–150 J. Ideally, the initial biphasic shock energy should be at least 150 J for all biphasic waveforms to simplify energy levels across all defibrillators, particularly because the type of waveform delivered by a defibrillator is not marked. Manufacturers should display the effective waveform dose range on the face of the biphasic defibrillator. If the rescuer is unaware of the recommended energy settings of the defibrillator, for an adult, use the highest energy setting for all shocks.

Second and subsequent shocks. The 2010 guidelines recommended either a fixed or escalating energy strategy for defibrillation. Several studies of BTE waveform show that although an escalating strategy reduces the number of shocks required to restore an organised rhythm compared with low fixed-dose biphasic defibrillation, and may be needed for successful defibrillation,[281,282] rates of ROSC or survival to hospital discharge are not significantly different between strategies.[271–273] Additionally, a rectilinear biphasic protocol using a fixed low energy level showed high cardioversion rates (>90 %) but a significantly lower ROSC rate for recurrent VF could not be excluded.[283] Several in-hospital studies using an escalating shock energy strategy have shown improvement in cardioversion rates (compared with fixed dose protocols) in non-arrest rhythms.[284–289]

In 2025, there remains no evidence to support either a fixed or escalating energy protocol. Both strategies are acceptable; however, 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 for subsequent shocks.

Recurrent ventricular fibrillation (refibrillation)

Recurrence of fibrillation is usually defined as 'recurrence of VF during a documented cardiac arrest episode, occurring after initial termination of VF while the patient remains under the care of the same providers (usually out-of-hospital).' Refibrillation is common and occurs in >50 % of patients following initial first-shock termination of VF.[281] Two studies showed termination rates of subsequent refibrillation were unchanged when using fixed 120 J or 150 J shock protocols respectively,[283,290] but a larger study showed termination rates of refibrillation declined when using repeated 200 J shocks, unless an increased energy level (360 J) was selected.[281] In a retrospective analysis, conversion of VF to an organised rhythm was higher if the VF had first appeared after a perfusing rhythm, than after PEA or asystole.[223]

In view of the larger study suggesting benefit from higher subsequent energy levels for refibrillation,[281] the ERC recommends that if a shockable rhythm recurs after successful defibrillation, and the defibrillator can deliver shocks of a higher energy, it is reasonable to increase the energy for subsequent shocks.

Refractory ventricular fibrillation

Refractory VF is defined as fibrillation that persists after three or more shocks. It was initially reported as occurring in approximately 20 % of patients who present in VF,[281] but more recent studies have suggested that the true incidence may be a low as 5 %. The majority of patients cardiovert successfully, but refibrillate during the two-minute period of chest compression following the shock.[291] These cases of refibrillation may therefore make up a large proportion of cases of refractory VF. Duration of VF correlates negatively with good outcome. Actively search for and correct any reversible causes (Fig. 2. ALS algorithm). Ensure that the defibrillation energy output is on the maximum setting. Check that the defibrillation pads are placed correctly (particularly the apical [lateral] pad, when using the antero-lateral pad position). Consider using an alternative defibrillation pad position (e.g., antero-posterior) – this is unchanged from previous guidance but has been given increased prominence.

Dual/double sequential defibrillation (DSD). Patients in refractory VF have significantly lower rates of survival than patients who respond to standard resuscitation treatments. Double sequential defibrillation (DSD) is the use of two defibrillators to deliver two overlapping shocks or two rapid sequential shocks, one with standard pad placement and the other with either antero-posterior or additional antero-lateral pad placement. The technique has been suggested as a possible means of increasing VF termination rates. With numerous case reports and some observational studies,[292–299] in 2020 ILCOR reviewed the efficacy of this technique

and based on very low certainty evidence made a weak recommendation against the routine use of a DSD strategy in comparison with standard defibrillation strategy for cardiac arrest with a refractory shockable rhythm.[84,224] More recently, ILCOR again reviewed the evidence[1] following the publication of a RCT (DOSE VF) of OHCA incorporating DSD.[211] The updated ILCOR recommendation suggested that a DSD (weak recommendation, low certainty of evidence) or a vector change defibrillation strategy (weak recommendation, very low certainty of evidence) may be considered for adults with cardiac arrest who remain in VF/pVT after three or more consecutive shocks. However, in view of the practical challenges of delivering DSD using two defibrillators and the limited evidence for its efficacy, the ERC guidelines do not recommend its introduction into routine practice. Following the public comment period in May 2025, the writing group further considered DSD. The group had already considered the evidence for DSD and in particular the DOSE VF RCT in detail. Following the public feedback, we did not identify any new evidence to change the recommendations. Our considerations included that the DOSE VF study was stopped early due to the COVID pandemic, recruited fewer than half the planned number of patients, and the final results included data from the initial feasibility study. We noted the lack of support from manufacturers and the potential for defibrillator damage.[300,301] We also considered the need for additional resources (equipment and training) required to implement DSD for routine use. Equipoise still exists regarding DSD and there are two European trials (STRAT-DEFI [NCT06781892], The Dual Defib Trial [NCT06672159]) of DSD that will help inform guideline updates. These guidelines are reviewed annually, and if necessary the guidance on DSD will be updated. Finally, considering vector change for refractory VF cardiac arrest has been part of previous ERC ALS guidelines and is unchanged.

Manual pressure augmentation. The application of manual pressure on defibrillation pads to reduce impedance and thereby increase defibrillation shock success has been studied. There is evidence that this technique may be helpful for treating resistant cardioversion of atrial fibrillation.[302] However, the safety and efficacy of manual pressure augmentation has not been studied in the context of shockable OHCA.

Percutaneous stellate ganglion block in refractory electrical storm. Percutaneous stellate ganglion block (PSGB) is an emerging, minimally invasive strategy for electrical storm treatment.[303] A large, prospective, multicentre study provides some evidence in favour of the effectiveness and safety of PSGB for treating refractory electrical storm.[304] A secondary analysis of this study, including 14 patients who were treated with PSGB during IHCA due to refractory or recurrent VF, found no major complications; 11 (78 %) patients survived for at least 24 h, and 7 (50 %) were discharged with favourable neurological outcome.[305] However, data on the use of PSGB in patients experiencing refractory cardiac arrest is very limited, and larger studies are needed before its introduction into routine practice.

Analysis of rhythm during chest compression

New software technology in some defibrillators enables the removal of ECG motion artefact generated during chest compressions to show the real-time underlying waveform during CPR. An ILCOR systematic review found no studies in humans evaluating this technology, leading to a weak recommendation based on very low certainty evidence to suggest against the routine use of artefact-filtering algorithms for analysis of electrocardiographic rhythm during CPR.[152] In making its recommendation ILCOR placed a priority on avoiding the costs of a new technology where effectiveness remains to be determined. The ILCOR task force acknowledged that some EMS already use artefact-filtering algorithms for rhythm analysis during chest compressions, and strongly encouraged EMS to report their experience to build the evidence base regarding these technologies in clinical practice.

Implantable cardioverter defibrillators. Implantable cardioverter defibrillators (ICDs) are becoming increasingly common as they are implanted more frequently in an aging population. They are implanted because a patient is at risk from, or has had, a life-threatening shockable arrhythmia. They are usually embedded under the pectoral muscle below the left clavicle (in a similar position to pacemakers, from which they cannot be immediately distinguished). More recently, extravascular devices can be implanted subcutaneously in the left chest wall, with a lead running parallel to the left of the sternum.[306] In a recent RCT, the subcutaneous ICD (S-ICD) was non-inferior to the transvenous ICD with respect to device-related complications and inappropriate shocks.[307]

On sensing a shockable rhythm, an ICD will discharge approximately 40 J (approximately 80 J for subcutaneous devices) through an internal pacing wire embedded in the right ventricle. On detecting VF/pVT, ICD devices will discharge no more than eight times but may reset if they detect a new period of VF/pVT. Patients with fractured ICD leads may suffer repeated internal defibrillation as the electrical noise is mistaken for a shockable rhythm. In these circumstances, the patient is likely to be conscious, with the ECG showing a relatively normal rate. A magnet placed over the ICD will turn off the defibrillation function.[245]

Discharge of an ICD may cause pectoral muscle contraction in the patient, and shocks to the rescuer have been documented.[308] Given the low energy values discharged by conventional ICDs, it is unlikely that any harm will come to the rescuer, but minimising contact with the patient whilst the device is discharging is prudent. Surface current from subcutaneous ICDs is significant and may cause a perceptible shock to the rescuer.[309,310] Cardioverter and pacing function should always be re-evaluated following external defibrillation, both to check the device itself and to check pacing/defibrillation thresholds of the device leads.

Pacemaker spikes generated by devices programmed to unipolar pacing may confuse AED software and emergency personnel and may prevent the detection of VF.[311] The diagnostic algorithms of modern AEDs can be insensitive to such spikes.

Ultraportable automated external defibrillators. A recent ILCOR scoping review found no evidence of ultraportable AED device performance, clinical or safety outcomes, particularly in relation to their novel low-energy waveforms.[312] ILCOR recommends that the safety and efficacy of these devices are established prior to clinical introduction and this recommendation is supported by the ERC.

Patients with actively discharging implantable cardioverter defibrillators (ICD)

Patients considered at risk of cardiovascular collapse from malignant arrhythmias may have a pre-emptive ICD placed. There are two main types:

a) Transvenous (standard) ICD: Conventional placement beneath the left pectoral muscle, with a lead inserted into the right ventricle (Fig. 6). Typically delivers 40 J on discharge.

b) Subcutaneous ICD: Placed beneath the skin in the left lateral chest wall with a lead placed subcutaneously along the left sternal border (Fig. 7). Typically delivers 80 J on discharge.

If either device senses a shockable rhythm, the ICD will deliver a defibrillation shock between the ICD and the distal section of the wire. Externally, this is often not apparent with a standard ICD, but a S-ICD depolarises more chest muscle to make the shock visible externally.

Most ICDs deliver up to 6–8 shocks per episode before stopping. During this time, the shock intervals are approximately 5–20 s (depending on the internal programming). A lead fracture may cause intermittent erroneous sensing, resulting in resetting of the discharge programme and near-continuous internal defibrillation. Because the S-ICD does not have intracardiac leads, it requires a higher energy output to ensure effective defibrillation. Rescuers may sense a significant shock across their arms while performing external chest compressions, even when wearing clinical gloves. If an ICD/S-ICD fails to terminate a shockable rhythm, conventional external shocks should be delivered, placing any defibrillation pad/paddle >8 cm from the defibrillator box (as above).

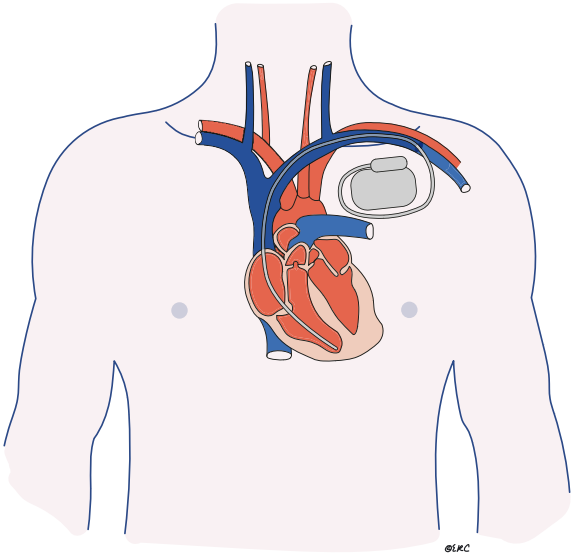

Fig. 6 – Position of a transvenous (standard) Implantable Cardioverter Defibrillator.

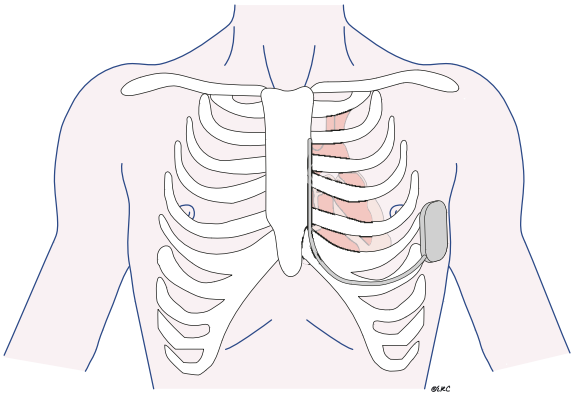

Fig. 7 – Position of a subcutaneous Implantable Cardioverter Defibrillator.

Waveform analysis during CPR

It is possible to predict, with varying reliability, the success of defibrillation from the fibrillation waveform.[220,313–334] If optimal defibrillation waveforms and the optimal timing of shock delivery can be determined in prospective studies, it should be possible to prevent the delivery of unsuccessful high energy shocks and minimise myocardial injury. Since the 2021 guidelines,[60] one small RCT, 10 observational studies, one systematic review and one narrative review were identified.[205,335–346] Most of the studies identified were retrospective and observational, assessing the ability of VF waveform analysis to predict defibrillation success or ROSC. The only RCT showed the prospective real-time use of VF waveform analysis using Amplitude Spectrum Area (AMSA) analysis during CPR.[335] Amplitude Spectrum Area is the most studied parameter (9/10 studies) and showed the highest accuracy to predict defibrillation success[304,336–338,341,342], although its predictivity was inferior to a machine learning algorithm during chest compression.[342] The small RCT that compared AMSA-guided CPR with standard CPR showed no evidence of improvement in termination of VF (primary outcome), ROSC or in long-term survival.[335] Recent developments include the use of convolutional neural networks to calculate AMSA during continuous CPR.[344] Additionally, information other than the outcome of an immediate defibrillation can be obtained from the waveform analysis. For instance, two retrospective observational studies assessed the ability of AMSA to detect coronary occlusion, showing lower AMSA values in case of acute myocardial infarction.[345] A recent ILCOR systematic review update[347] found two observational studies focusing on software-based cardiac rhythm analysis during CPR in OHCA patients.[348,349] Both studies were observational and used historical controls, showing improved CPR quality metrics with rhythm analysis during compressions. None of the studies evaluated survival rates or neurological outcomes, leaving it uncertain whether these technologies enhance patient outcomes. ILCOR found insufficient evidence to make a treatment recommendation, given the absence of RCTs or well-controlled observational studies. Consistent with previous recommendations, there remains insufficient evidence to support routine use of VF waveform analysis to guide the optimal timing for a shock attempt.

Airway and ventilation

Since 2015 the ERC has recommended a stepwise approach to airway management during CPR.[215] Three large RCTs of airway management for OHCA have been published since 2015 that have supported this guidance.[350–352] An ILCOR systematic review addressed whether a specific advanced airway management strategy (use of supraglottic airway devices (SGA) or tracheal intubation) improved outcome from cardiac arrest in comparison with an alternative airway management strategy.[353,354] Seventy-eight observational studies were included; nine of these addressed the timing of advanced airway management. Eleven controlled trials were included but only three of these were RCTs.[354] The first of these RCTs compared early tracheal intubation with bag-mask ventilation (tracheal intubation delayed until after ROSC) in a physician-

staffed EMS system.[351] The result of this non-inferiority trial that recruited over 2,000 patients was inconclusive –4.3 % versus 4.2 % for 28-day survival with favourable functional outcome (Cerebral Performance Category [CPC] 1–2), no significant difference. Notably, the tracheal intubation success rate was 98 % and 146 patients in the bag-mask ventilation group underwent 'rescue intubation' (i.e., crossed over); 100 of these were because of regurgitation. In a comparison of initial laryngeal tube (although technically an infraglottic airway, it is usually included within the SGA group of devices) insertion with tracheal intubation in 3,000 OHCAs by paramedics in the United States, 72-h survival (primary outcome) was higher in the laryngeal tube group (18.2 % versus 15.3 %; $p = 0.04$).[352] However, the overall tracheal intubation success rate was just 51 % making it possible that the lower survival rate in the tracheal intubation group was a reflection of the poor tracheal intubation success rate. The third of these RCTs was a comparison of the initial insertion of an i-gel supraglottic airway (SGA) with tracheal intubation in OHCA treated by paramedics in the United Kingdom (UK).[350] Among the more than 9,000 patients enrolled, there was no difference in the primary outcome of favourable functional survival (modified Rankin Scale [mRS] ≤ 3; 6.4 % versus 6.8 %; $P = 0.33$).

A cluster RCT in Taiwan compared insertion of i-gel with tracheal intubation in 936 OHCA patients and found no difference in the rate of sustained ROSC, the primary outcome.[355] Three observational studies have compared the use of the laryngeal tube with i-gel in OHCA. Rates of successful airway placement and survival to hospital discharge were higher with the i-gel than the laryngeal tube.[356–358]

In a cluster-randomised trial in patients with OHCA in Singapore, use of the laryngeal tube by paramedics was associated with more complications and fewer successful placements compared with a laryngeal mask airway (Ambu Aura-i LMA).[359] There are many different SGAs currently available, but only a few have been studied in recent RCTs. Ultimately it will be a local system decision on which SGA to use.

A large observational cohort study of IHCA from the American Heart Association (AHA) Get with the Guidelines-Resuscitation (GWTG-R) registry, compared outcomes in patients intubated at any given minute within the first 15 min after cardiac arrest onset against matched patients still receiving CPR at risk of being intubated within the same minute.[360] The matching was based on time-dependent propensity scores, pairing 43,314 intubated patients and non-intubated patients with similar propensity for intubation. Compared with not intubating, tracheal intubation was associated with a lower rate of ROSC (risk ratio [RR] 0.97; 95 % CI 0.96–0.99; $p < 0.001$), lower survival to hospital discharge (RR 0.84; 95 % CI 0.81–0.87; $p < 0.001$), and worse neurological outcome (RR 0.78; 95 % CI 0.75–0.81; $p < 0.001$). Two ongoing RCTs are comparing insertion of an i-gel with tracheal intubation in IHCA.[361,362]

After reviewing the evidence for airway management during cardiac arrest, the ILCOR ALS Task Force made the following treatment recommendations,[354] which the ERC has adopted, and these remain unchanged following an evidence update in 2025[1]:

- We suggest using bag-mask ventilation or an advanced airway strategy during CPR for adult cardiac arrest in any setting (weak recommendation, low to moderate certainty of evidence).
- If an advanced airway is used, we suggest a SGA for adults with OHCA in settings with a low tracheal intubation success rate (weak recommendation, low certainty of evidence).
- If an advanced airway is used, we suggest an SGA or tracheal intubation for adults with OHCA in settings with a high tracheal intubation success rate (weak recommendation, very low certainty of evidence).
- If an advanced airway is used, we suggest an SGA or tracheal intubation for adults with IHCA (weak recommendation, very low certainty of evidence).

Patients often have more than one type of airway intervention, typically starting with basic and advancing to more complex techniques that are inevitably applied later during cardiac arrest – the stepwise approach.[350,363] The best airway, or combination of airway techniques will vary according to patient factors, the phase of the resuscitation attempt (during CPR, after ROSC), and the skills of rescuers. If basic airway techniques enable effective ventilation, there may be no need to progress to advanced techniques until after ROSC. One potential advantage of inserting an advanced airway is that it enables chest compressions to be delivered continuously without pausing during ventilation. The quality of the seal between the glottis and an SGA is variable – in some cases the oropharyngeal leak pressure is very low and excessive gas leakage will occur, rendering effective ventilation impossible and resulting in imperceptible chest movement.[364] If this occurs, revert to bag-mask ventilation or, if the appropriately skilled personnel are available, intubate the trachea. Most patients with ROSC remain comatose and will need tracheal intubation and mechanical ventilation (See Post-resuscitation Care).[147]

Airway obstruction

Patients requiring CPR often have an obstructed airway, usually secondary to loss of consciousness, but occasionally it may be the primary cause of cardiorespiratory arrest.

Basic airway management and adjuncts. Three manoeuvres may improve the patency of an airway obstructed by the tongue or other upper airway structures: head tilt, chin lift, and jaw thrust. Despite a total lack of published data on the use of nasopharyngeal and oropharyngeal airways during CPR, they are often helpful, and sometimes essential, to maintain an open airway, particularly when CPR is prolonged.

Oxygen during CPR

During cardiac arrest, the blood flow and oxygen reaching the brain are low, even with effective CPR. Based on the physiological rationale and expert opinion, ILCOR and ERC recommends giving the highest feasible inspired oxygen concentration during cardiac arrest to maximise oxygen delivery to the brain thereby minimising hypoxic-ischaemic injury.[203] Observational studies have shown that a higher PaO_2 during CPR is associated with a higher likelihood of ROSC and patient survival.[365,366] After ROSC, as soon as SpO_2 can be measured reliably or arterial blood gas values are obtained, titrate the inspired oxygen to achieve an arterial oxygen saturation of 94–98 % or arterial partial pressure of oxygen (PaO_2) of 10–13 kPa (75–100 mmHg). Rescuers should be aware that pulse oximetry

can overestimate the true oxygen saturation in people with darker skin tones.[367,368]

Choking

The initial management of foreign body airway obstruction (choking) is addressed in the ERC Guidelines 2025 First Aid.[7] This topic has been recently assessed by an ILCOR evidence update[369] and a previous systematic review.[370] In an unconscious patient with suspected foreign body airway obstruction if initial basic measures are unsuccessful use laryngoscopy and forceps to remove the foreign body under direct vision. To do this effectively requires training. Some patients with foreign body airway obstruction may require emergency front of neck access and this is addressed further below.

Ventilation

Ventilation breath volume. Recent studies suggest that effective ventilation and oxygenation is often done poorly during ALS and this is associated with poorer outcomes.[371] This is increasingly important given that patients have often received a period of chest compression only CPR before ALS trained providers arrive. Advanced life support providers should give artificial ventilation as soon as possible for any patient in whom spontaneous ventilation is inadequate or absent. This is usually achieved with a self-inflating bag attached to a facemask or an advanced airway. Deliver each breath over approximately 1 s, giving a volume that corresponds to normal chest movement. The chest should visibly rise; this represents a compromise between giving an adequate volume, minimizing the risk of gastric inflation, and allowing adequate time for chest compressions. The choice of self-inflating bag may also effect outcome – in a recent observational study of adult OHCA patients the use of a smaller self-inflating bag was associated with poorer survival.[372]

30:2 versus asynchronous ventilation. Although the delivery of continuous chest compressions during face-mask ventilation was previously thought to increase the risk of regurgitation, a trial of continuous versus interrupted chest compressions during CPR (CCC Trial) that enrolled more than 23,000 patients showed no statistically significant difference in survival to discharge.[373] ILCOR has subsequently recommended that when using bag mask, EMS providers perform CPR either using a 30:2 compression-ventilation ratio (pausing chest compressions for ventilation) or continuous chest compressions without pausing while delivering positive pressure ventilation (strong recommendation, high-quality evidence).[374] In Europe, the most common approach during CPR with an unprotected airway is to give two ventilations after each sequence of 30 chest compressions. A secondary analysis of the CCC trial examined the frequency of effective ventilations delivered with bag-mask and measured via thoracic bioimpedance during pauses in chest compressions among 1976 OHCA patients of the 30:2 arm of the trial.[371] The study revealed that the quality of ventilation was notably poor: in 60 % of the patients, lung inflation was observed in less than half of the chest compression pauses, with a median time to the first ventilation greater than 4 min. Patients who experienced lung inflations in at least 50 % of pauses showed higher rates of ROSC (RR 1.3 [95 % CIs 1.2–1.5]; $p < 0.0001$), survival at hospital discharge (RR 2.2 [95 % CIs 1.6–3.0]; $p < 0.0001$) and survival with favourable neurological outcomes (RR 2.8 [95 % CIs 1.8–4.3]; $p < 0.0001$) (1). The ERC recommends, that advanced life support providers should ensure effective ventilation when using a bag-mask. If ventilation is inadequate, efforts should be made to optimise bag-mask ventilation by improving the mask seal, maintaining airway patency, and, using a two-person technique – rescuers who do not regularly and frequently use a bag-mask should use a two-person technique to ensure effective ventilation.[375,376]

Once a tracheal tube or an SGA has been inserted, ventilate the lungs at a rate of 10 min^{-1} and continue chest compressions without pausing during ventilations.[377] The laryngeal seal achieved with an SGA may not be good enough to prevent at least some gas leaking when inspiration coincides with chest compressions. Moderate gas leakage is acceptable (unless there is a significant risk of infection),[378,379] particularly as most of this gas will pass up through the patient's mouth. If excessive gas leakage results in inadequate ventilation of the patient's lungs, chest compressions will have to be interrupted to enable ventilation, using a compression – ventilation ratio of 30:2. One observational study of OHCA patients who had received prolonged CPR with a mechanical device has documented worse blood gases among those whose airway was managed with an SGA compared with tracheal intubation.[380]

Ventilation rate. Since the 2021 guidelines review, one small RCT, a secondary analysis of an RCT, and three observational studies assessing ventilation rate during CPR have been identified through a systematic search strategy.[372,381–384] A small RCT randomised 46 patients after tracheal intubation to receive either 10 or 20 breaths/min delivered with mechanical ventilation in volume-controlled mode with a tidal volume of 6 mL kg^{-1}.[381] The study was terminated early, failing to achieve the planned sample size, and showed a higher minute ventilation in the group randomised to 20 breaths min^{-1} (primary outcome) with no evidence of improvement in hypercapnia, hypoxia, and ROSC rates. A secondary analysis of the Pragmatic Airway Resuscitation Trial (PART) compared laryngeal tubes with tracheal intubation evaluated the association between ventilation rates and outcomes.[382] A median ventilation rate of 8 min^{-1} was observed in both groups, and the duration of hypoventilation (defined as <6 min^{-1}) was associated with decreased rates of ROSC and hospital survival. The duration of mild hyperventilation (>12 to 16 min^{-1}) was associated with improved ROSC, survival to hospital discharge and survival with favourable neurological outcome. Two retrospective observational studies failed to demonstrate an association between ventilation rates and patient outcomes.[372,384] Although there is increasing evidence in ALS settings showing that respiratory rate is frequently below 10 min^{-1} during CPR and that the duration of hypoventilation is associated with worse outcomes, there remains insufficient evidence to establish an optimal ventilation rate during CPR.

Mechanical ventilation during CPR. Two RCTs and two observational studies comparing mechanical ventilation and bag ventilation during CPR were identified.[385–388] The RCTs were feasibility studies comparing mechanical ventilation delivered in volume-controlled mode (tidal volume 6–7 mL kg^{-1}, RR 10 min^{-1}, FiO_2 1.0) with bag ventilation after advanced airway placement. Each study included 60 patients and no sample size calculations were performed. They showed that the use of mechanical ventilation during both mechanical[385] and manual[386] chest compressions was feasible. Neither of these trials found differences in oxygenation, ROSC, or survival. A prospective observational study indicated that mechanical ventilation is associated with reduced $PaCO_2$ values; however, it did not affect the rates of ROSC, survival, or survival with favourable neurological outcomes.[387] The retrospective study compared

mechanical ventilation delivered in cardiopulmonary ventilation mode (298 patients) with bag ventilation (2268 patients), showing that mechanical ventilation may increase the rate of ROSC with no effect on survival with favourable neurological outcomes.[388]

Although studies show that the use of mechanical ventilation during chest compressions is feasible, there is insufficient evidence to support the use of mechanical ventilation over manual bag ventilation or to recommend a specific ventilation mode. We suggest that if using mechanical ventilation (volume controlled or pressure regulated modes) during CPR, the following initial settings are used: tidal volume of 6–8 mL kg^{-1} (predicted body weight) or sufficient to cause a visible chest movements, the maximum fraction inspired oxygen, a respiratory rate of 10 min^{-1}, an inspiratory time of 1–1.5 s, PEEP 0–5 cmH_2O, alarm for peak pressure set at 60–70 cm H_2O, and the inspiratory trigger turned off. If mechanical ventilation is not effective, switch to manual ventilation.

Ventilation during mechanical chest compressions. Ventilation during mechanical chest compressions may be particularly challenging because of the mechanical forces applied to the chest, causing lung volume to decrease below functional residual capacity (FRC).[389,390] One RCT and one retrospective observational study evaluated ventilation during mechanical chest compressions.[391,392] The pilot RCT randomised 30 OHCA patients undergoing mechanical chest compressions after tracheal intubation into one of the following three ventilation strategies: 1) biphasic positive airway pressure (BIPAP) with assisted spontaneous breathing; 2) continuous positive airway pressure (CPAP) and 3) volume-controlled ventilation. This study showed a higher tidal volume delivered in BIPAP mode than in CPAP mode, while no differences in the rate of ROSC were detected. The retrospective observational study evaluated the frequency of bag-ventilation during pauses in mechanical chest compressions delivered at a 30:2 ratio and showed inadequate ventilation during 3-second pauses in mechanical chest compressions – two inflations were successfully provided in 45 % of compression pauses, and no ventilation was delivered in 19 % of compression pauses.[392] A particularly low likelihood of delivering two successful insufflations was detected in the first four minutes after mechanical chest compression device placement. The best method for ventilation during mechanical chest compression is uncertain.

Passive oxygen delivery. In the presence of a patent airway, chest compressions alone may result in some ventilation of the lungs.[393] Oxygen can be delivered passively, either via an adapted tracheal tube (Boussignac tube),[394,395] or with the combination of an oropharyngeal airway and standard oxygen mask with a non-rebreather reservoir.[396] In theory, a SGA can also be used to deliver oxygen passively but this has yet to be studied. One study has shown higher neurologically favourable survival with passive oxygen delivery (oral airway and oxygen mask) compared with bag-mask ventilation after shockable OHCA, but this was a retrospective analysis and is subject to numerous confounders.[396] A trial of continuous or interrupted chest compressions during CPR (CCC Trial) included a subgroup of patients who were treated with passive oxygenation but until further data are available, passive oxygen delivery without ventilation is not recommended for routine use during CPR.[373]

Choice of airway devices

Disadvantages of tracheal intubation over bag-mask ventilation include:

- The risk of an unrecognised misplaced tracheal tube – in patients with OHCA the reliably documented incidence ranges from 0.5 % to 17 %: emergency physicians – 0.5 %[397]; paramedics – 2.4 %[398], 6 %[399,400], 9 %,[401] and 17 %[402]
- A prolonged period without chest compressions while tracheal intubation is attempted. In a study of prehospital tracheal intubation by paramedics during 100 cardiac arrests the median duration of the interruptions in CPR associated with tracheal intubation attempts was 110 s (interquartile range 54–198 s; range 13–446 s).[403] Tracheal intubation attempts accounted for almost 25 % of all CPR interruptions.
- A comparatively high failure rate. Intubation success rates correlate with the tracheal intubation experience attained by individual paramedics.[404] The high failure rate of 51 % documented in the PART trial[352] is similar to those documented in some prehospital systems more than 20 years ago.[405,406]
- Tracheal intubation is a difficult skill to acquire and maintain. In one study, anaesthesia residents required about 125 intubations with direct laryngoscopy in the operating room setting before they were able to achieve a tracheal intubation success rate of 95 % under such optimal conditions.[407]

Healthcare personnel who undertake prehospital tracheal intubation should do so only within a structured, monitored program, which should include comprehensive competency-based training and regular opportunities to refresh skills.

The ILCOR recommendation is that only systems that achieve high tracheal intubation success rates should use this technique.[354] ILCOR did not recommend a particular success rate but suggested it should be similar to that achieved in the RCT comparing early tracheal intubation with bag-mask ventilation (tracheal intubation delayed until after ROSC) in a physician-staffed EMS system.[351] The tracheal intubation success rate in this study was 98 %. The expert consensus of the ERC 2021 ALS Guidelines writing group was that a high success rate is greater than 95 % with up to two intubation attempts.[60]

Rescuers must weigh the risks and benefits of tracheal intubation against the need to provide effective chest compressions. To avoid any interruptions in chest compressions, unless alternative airway management techniques are ineffective, it is reasonable to defer tracheal intubation until after ROSC. In settings with personnel skilled in advanced airway management, laryngoscopy should be undertaken without stopping chest compressions; a brief pause in chest compressions will be required only as the tube is passed through the vocal cords. The tracheal intubation attempt should interrupt chest compressions for less than 5 s; if intubation is not achievable within these constraints, recommence bag-mask ventilation. After tracheal intubation, tube placement must be confirmed immediately (see below) and the tube must be secured adequately.

Video laryngoscopy. Video laryngoscopy (VL) is being used increasingly in anaesthetic and critical care practice.[408,409] Preliminary studies indicate that compared with direct laryngoscopy (DL), VL during CPR improves laryngeal view and tracheal intubation success rates,[410,411] reduces the risk of oesophageal intubation[412] and reduces interruptions to chest compressions.[413] One systematic review concluded that in the prehospital setting, VL decreased the first-attempt tracheal intubation success rate (RR, 0.57; $P < 0.01$; high-quality evidence) and overall success rate (RR, 0.58; 95 % CI, 0.48–0.69; moderate-quality evidence) by experienced operators.[414] A later meta-analysis of OHCAs and IHCAs

that included six observational studies and one RCT documented higher first pass success rate and better grade of view with VL compared with DL.[415] A secondary analysis of an RCT also documented a higher rate of successful tracheal intubation on the first attempt with VL compared with DL.[416] Several different VL systems are available and they do not all perform in the same way. The expert consensus of the writing group and recommendation of the ERC is that the rescuer's choice of direct laryngoscopy or video laryngoscopy should be guided by local protocols and rescuer experience. However, in settings where VL is immediately available, it is preferable to use VL instead of DL.

Confirmation of correct placement of the tracheal tube

Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation in patients with and without cardiac arrest. One large RCT comparing bag-mask ventilation and tracheal intubation during CPR by skilled prehospital physicians reported accidental but recognised oesophageal intubation in 10 % of attempts.[351] Another study found the rate of unrecognised oesophageal intubation in OHCA by paramedics was 5 %.[400] Therefore, accurate means to verify correct tracheal tube placement in CPR are of greatest importance. The previous evidence supporting these guidelines is summarised in longstanding ILCOR recommendations.[33,224,417] The 2022 PUMA Guidelines for the prevention of unrecognised oesophageal intubation state clearly that the only accurate method to confirm tracheal placement of the tracheal tube is the presence of a sustained $ETCO_2$ trace for at least seven breaths on waveform capnography in patients with and without cardiac arrest.[418] Clinical assessment such as chest and abdominal auscultation, observation of chest expansion, and fogging of the tube cannot be used to confirm tracheal placement if waveform capnography suggests otherwise. The 'No Trace = Wrong Place' campaign by the UK Royal College of Anaesthetists emphasised that immediately after tracheal intubation (even during cardiac arrest) the absence of exhaled CO_2 strongly suggested oesophageal intubation.[419] It is accepted that this is not sufficient and that in some cases of unrecognised oesophageal intubation there has been some CO_2 detected – this has led to the need for sustained exhaled CO_2 trace to exclude oesophageal intubation.[420] If waveform capnography does not confirm correct placement of the tracheal tube in patients without cardiac arrest the PUMA Guidelines suggest using repeat laryngoscopy to view the passage of the tube, use of a flexible bronchoscope or ultrasound of the neck. Portable monitors make capnographic initial confirmation and continuous monitoring of tracheal tube position feasible in both in- and out-of-hospital settings where tracheal intubation is performed. Although a sustained $ETCO_2$ trace confirms that the tube is not in the oesophagus, it could still be misplaced in a bronchus which can cause severe hypoxaemia. Check the tube length at the teeth to ensure that it is not inserted too far. Auscultation of the lungs may also help to exclude endobronchial intubation, but this is challenging during cardiac arrest and must not delay chest compressions.

Ultrasonography of the neck or visualisation with a flexible fibreoptic scope by skilled operators can also be used to confirm the presence of a tracheal tube in the trachea.[421]

Cricoid pressure

The use of cricoid pressure in cardiac arrest is not recommended (expert consensus). Cricoid pressure can impair ventilation, laryngoscopy, tracheal tube and SGA insertion, and may even cause complete airway obstruction.[422]

Securing the tracheal tube and supraglottic airway

Accidental dislodgement of a tracheal tube can occur at any time but may be more likely during CPR and during transport. An SGA is more prone to being dislodged than a tracheal tube.[350] The most effective method for securing the tracheal tube or SGA has yet to be determined. Use either conventional tapes or ties, or purpose-made holders.

Emergency front of neck access (eFONA)

In rare cases, it may be impossible to oxygenate a patient in cardiac arrest using bag-mask ventilation, or to insert a tracheal tube or supraglottic airway. This may occur in patients with extensive facial trauma or laryngeal obstruction caused by oedema, tumour or a foreign body. In 2023, ILCOR undertook a scoping review which summarised evidence from 69 studies across four domains: incidence of eFONA; success rates of eFONA attempts; clinical outcomes in patients with an eFONA attempt; and complications associated with eFONA attempts.[423] The review found that none of the identified studies focused specifically on cardiac arrest, and noted that the available evidence was highly heterogeneous. Consistent with a systematic review on pre-hospital eFONA success rates, the scoping review observed that, when attempted, eFONA success rates were typically high.[424] Based on the available evidence, ILCOR identified that it was not possible to undertake a systematic review, but made a good practice statement supporting the use of cricothyroidotomy in patients where standard airway management and ventilation strategies have been unsuccessful.[1] An Australian observational study, published in 2024, reported on 80 cricothyroidotomy attempts of which 56 occurred in OHCA patients.[425] The reported incidence was 1.1 cricothyroidotomies per 1000 attempted resuscitations with a success rate in cardiac arrest of 58.9 %. The use of a surgical approach was associated with a higher success rate than a needle approach (88.2 % v. 54.6 %, $p = 0.003$). Consistent with Difficult Airway Society recommendations, the ERC recommends, where feasible, the use of a scalpel-bougie cricothyroidotomy.[426]

Medication and fluids

Vascular access

The effectiveness of medication in cardiac arrest is time-dependent.[427–429] The IO route has been proposed as an alternative strategy for initial vascular access in adult cardiac arrest, based on the perceived ease of insertion and an RCT showing that the time to drug administration was fastest with a tibial IO strategy, compared to both a peripheral IV or humeral IO strategy.[430,431] Observational studies show that the use of the IO route in clinical practice has increased over recent years.[432,433]

In 2024, three large RCTs compared an IO-first with an IV-first strategy in adult cardiac arrest.[434–436] In contrast to earlier research, none of these trials found that the IO route facilitated more rapid drug administration. These three trials, which comprised 9,332 patients, formed the basis of a systematic review and meta-analysis led by the ILCOR ALS Task Force.[437] The meta-analysis showed that an IO-first strategy, compared with an IV-first strategy, did not improve the rate of survival to 30-days or discharge with favourable neurological outcome (OR 1.07; 95 % CI 0.88–1.30; low-certainty evidence) and 30-day survival (OR 0.99: 95 % CI 0.84–1.17; moderate-certainty evidence), but may reduce the rate of sustained ROSC

(OR 0.89, 95 % CI 0.80–0.99; moderate-certainty evidence). There was no evidence that the treatment effect was influenced by baseline patient characteristics or the anatomical site of the IO cannula. In formulating its treatment recommendation, ILCOR noted the routine availability of the peripheral IV route across international systems, the high cost of IO cannulae, the additional training requirement associated with intraosseous use, and the evidence from the meta-analysis suggesting that an IO-first strategy reduces the odds of achieving ROSC.[1] ILCOR noted that no RCTs had been undertaken in an IHCA but observed that the IO route is infrequently required in such cases.

Consistent with ILCOR, the ERC recommends that the IV route should be used as the primary route for vascular access in adult cardiac arrest. We recognise that the IO route may be a reasonable vascular access strategy where IV access cannot be rapidly achieved. Consistent with the two European RCTs, we recommend that two IV vascular access attempts are made before considering attempting IO access.[435,436]

There has been recent interest in the use of intramuscular (IM) adrenaline for cardiac arrest based on animal studies and observational data.[438–441] In a single-centre before-and-after observational study, a single dose of 5 mg IM adrenaline in 420 patients with OHCA was associated with improved survival to hospital admission, survival to hospital discharge, and functional survival compared with standard adrenaline dosing.[441] The role of the IM route for adrenaline in cardiac arrest needs to be studied with RCTs before considering its inclusion in guidelines.

Vasopressors

ILCOR recently updated their review of vasopressors in cardiac arrest.[1] The systematic reviews and *meta*-analyses evaluated standard dose adrenaline (1 mg) versus placebo, high dose (5–10 mg) versus standard dose (1 mg) adrenaline, adrenaline versus vasopressin, and adrenaline and vasopressin versus adrenaline alone.[192,442–444] The evidence showed that adrenaline (1 mg) improved ROSC, survival to hospital admission, survival to hospital discharge, and long-term survival (up to 12 months), although the effect on favourable neurological outcome remains uncertain. By contrast, the use of high-dose adrenaline or vasopressin (with or without adrenaline) did not improve long-term survival or favourable neurological outcome.

Based on this evidence, ILCOR makes a strong recommendation in favour of adrenaline during CPR (strong recommendation, low certainty of evidence).[192] The justification and evidence to decision framework highlights that the Task Force placed a very high value on the apparent life-preserving benefit of adrenaline, even if the absolute effect size is likely to be small and the effect on survival with favourable neurological outcome is uncertain.

The evidence supporting adrenaline use comes primarily from two placebo-controlled trials.[445,446] The PARAMEDIC2 trial followed the ERC ALS 2015 Guidelines, which recommended adrenaline administration as soon as vascular access was obtained for non-shockable rhythms, and after three unsuccessful defibrillation attempts for shockable rhythms.[215,446] Analysis of this trial continues to provide new evidence, with long-term follow-up data showing sustained survival benefits at 12 months.[447] Meta-analyses with the earlier PACA trial found greater effects of adrenaline on ROSC and survival to hospital discharge in initial non-shockable rhythms compared to shockable rhythms, although this difference was less pronounced for longer-term survival and favourable neurological outcomes.[213] A secondary analysis of time to drug administration found that while relative treatment effects of adrenaline remained constant, survival and favourable neurological outcome rates decreased over time, suggesting potential benefit from early intervention.[427]

Consequently, ILCOR recommends that adrenaline is administered as soon as feasible for non-shockable rhythms (PEA/asystole) (strong recommendation, very low certainty of evidence). For shockable rhythms (VF/pVT), ILCOR suggests administration of adrenaline after initial defibrillation attempts are unsuccessful during CPR (weak recommendation, very low certainty of evidence).

Consistent with the ILCOR Treatment Recommendations, the ERC recommends that adrenaline 1 mg is administered as soon as possible for adult patients in cardiac arrest with a non-shockable rhythm. For patients with a shockable rhythm persisting after three initial shocks, administer adrenaline 1 mg. Repeat adrenaline 1 mg every 3–5 min whilst ALS continues. While practical challenges may affect the timing of adrenaline in the prehospital setting, early administration remains a priority, particularly for non-shockable rhythms where alternative interventions are limited.

The optimal dose of adrenaline remains unclear. Pharmacokinetic and observational data suggest further investigation is needed regarding dosing strategies.[448,449] This includes the standard 1 mg dose, the cumulative effects of repeated doses, and potential alternative approaches such as titrated dosing in closely monitored settings. Alternative routes such as intracoronary administration, have been studied in cardiac catheterisation laboratories, but evidence is insufficient to support these approaches.[450,451]

If three stacked shocks have been given for a witnessed and monitored shockable cardiac arrest where immediate defibrillation is possible, these initial three stacked shocks should be considered as the first shock with regards to timing of the first dose of adrenaline and amiodarone. After these stacked shocks, providers should continue resuscitation attempts and adrenaline and amiodarone dosing according to the standard ALS algorithm.

A recent cost-effectiveness analysis of PARAMEDIC2 incorporating both direct survival benefits and increased organ donation rates supports the use of adrenaline during cardiac arrest, although costs may vary between healthcare systems.[452]

Consistent with the ILCOR treatment recommendation, the ERC does not support the use of vasopressin during cardiac arrest.

Antiarrhythmic drugs

ILCOR updated the CoSTR for antiarrhythmic drugs in 2018.[453] This was followed by ILCOR evidence updates in July 2023 and October 2024.[1,192] The 2018 CoSTR made the following recommendations:

- We suggest the use of amiodarone or lidocaine in adults with shock refractory VF/pVT (weak recommendation, low-quality evidence).
- We suggest against the routine use of magnesium in adults with shock-refractory VF/pVT (weak recommendation, very low-quality evidence).
- The confidence in effect estimates is currently too low to support an ALS Task Force recommendation about the use of bretylium, nifekalant, or sotalol in the treatment of adults in cardiac arrest with shock-refractory VF/pVT.
- The confidence in effect estimates is currently too low to support an ALS Task Force recommendation about the use of prophylactic antiarrhythmic drugs immediately after ROSC in adults with VF/pVT cardiac arrest.

Despite many studies, the ILCOR evidence updates in 2023 did not identify any compelling new data that would justify a further systematic review or changes to the treatment recommendations.[192] The 2024 evidence update [1] identified one additional small pilot RCT of the beta-blocker landiolol including 36 patients.[454] Given the interest in beta-blockers for cardiac arrest and in particular esmolol and landiolol, and the fact that sotalol is not considered a primary beta-blocker, the ILCOR ALS Task Force clarified the third bullet point of its 2018 treatment recommendation to state:

- The confidence in effect estimates is currently too low to support an ALS Task Force recommendation about the use of beta-blocker drugs, bretylium, or nifekalant, or sotalol in the treatment of adults in cardiac arrest with shock-refractory VF/pVT.

This 2025 guidance is therefore primarily based on the 2018 ILCOR systematic review that identified evidence from 14 RCTs and 17 observational studies that included lidocaine, amiodarone, magnesium, bretylium, nifekalant, procainamide, sotalol and beta-blockers.[455] Meta-analysis of randomised trials in adults, found that none of the antiarrhythmic drugs improved survival or favourable neurological outcome compared to placebo.

The largest randomised trial compared amiodarone, lidocaine or placebo in patients with VF/pVT refractory after at least one defibrillation attempt. Compared with placebo, amiodarone and lidocaine increased survival to hospital admission. However, there was no difference in survival to discharge or favourable neurological survival at discharge between groups.[456] In the pre-defined sub-group of bystander-witnessed cardiac arrests, amiodarone and lidocaine increased survival to hospital discharge compared with placebo. Survival was also higher with amiodarone than with placebo after EMS-witnessed arrest.

These data led ILCOR to suggest that amiodarone or lidocaine could be used in adults with shock refractory VF/pVT (weak recommendation, low quality evidence).[453] The values and preferences analysis indicates that the Task Force prioritised the pre-defined and reported sub-group analysis from the ALPS study, which showed greater survival with amiodarone and lidocaine in patients with a witnessed cardiac arrest. ILCOR did not support the use of magnesium, bretylium, nifekalant or procainamide.

The ERC updated its guidelines in 2018 to recommend that amiodarone should be given after three unsuccessful defibrillation attempts, irrespective of whether they are consecutive shocks, or interrupted by CPR, or for recurrent VF/pVT during cardiac arrest.[457] The initial recommended dose is amiodarone 300 mg; a further dose of 150 mg may be given after five defibrillation attempts. The recommendation in favour of amiodarone was based on 21 of 24 National Resuscitation Councils of Europe reporting that amiodarone was the main drug used during CPR.[457] Lidocaine 100 mg may be used as an alternative if amiodarone is not available, or a local decision has been made to use lidocaine instead of amiodarone. An additional bolus of lidocaine 50 mg can also be given after five defibrillation attempts.[457]

Thrombolytic therapy

ILCOR evidence updates in 2022 and 2024 for the treatment of pulmonary embolism did not identify sufficient new evidence to justify an update of the previous 2020 ILCOR systematic review.[192,193] We also considered a recent systematic review that identified 13 studies with 804 patients.[458] The 2020 ILCOR CoSTR pooled evidence from a sub-group analysis of the TROICA trial[459] and four observational studies which examined the use of thrombolytic drugs in cardiac arrest caused by suspected or confirmed pulmonary embolism (PE).[460–463] The studies did not find evidence that thrombolytic drugs improved neurological outcome.[459,462] By contrast, in one study, 30-day survival was higher in the intervention group (16 % vs 6 %; $P = 0.005$)[463] but not in three other studies which examined survival to discharge.[460–462] ROSC also improved in one study [461]but not in two others. [460,462] In making a weak recommendation for the use of thrombolytic drugs for suspected or confirmed PE and cardiac arrest based on very low certainty evidence, the ILCOR Task Force considered that the potential benefits outweighed the potential harm from bleeding.[224]

The ERC endorses the recommendation from ILCOR, which aligns with the ERC guidelines in 2015 and 2021.[60,215] The ERC does not support the routine use of thrombolytic drugs in cardiac arrest, unless the cause is suspected or confirmed PE. When thrombolytic drugs have been administered, based on evidence from case series consider continuing CPR attempts for at least 60–90 min before termination of resuscitation attempts.[464–466] As for all cardiac arrests the duration of the CPR attempt should take into account the possibility of reversing the underlying cause, and other factors such as patient comorbidity and frailty.

Fluids and blood components

ILCOR has not recently addressed the use of fluids during cardiac arrest. The ERC ALS Writing Group performed its own search up to February 2025. No RCTs have evaluated the routine administration of fluids versus no fluids as a treatment strategy for cardiac arrest. Two large RCTs provide indirect evidence from treatment strategies designed to induce hypothermia, which included the administration of up to 2 L of ice-cold IV fluids during OHCA[467] or immediately after ROSC.[468] The studies found no improvement in short[467,468] or long-term outcomes.[469] The studies reported evidence of reduced ROSC in patients with VF,[467] increased rate of re-arrest,[468] and higher rates of pulmonary oedema.[467,468] It is not possible to determine from these studies whether the harmful effects were related to fluid volume, the rate of infusion, or the temperature of the infused fluids.[470] The routine rapid infusion of large-volume fluids should be avoided unless there is evidence or suspicion of a hypovolaemic cause of the cardiac arrest. A clinical assessment, the history of events before cardiac arrest, and when the skills are available, POCUS can help identify hypovolaemia during resuscitation.

A recent systematic review on fluid therapy during and after CPR for non-traumatic CA confirmed the limited evidence for the use of either isotonic saline or balanced crystalloids, and the potential benefit of hypertonic fluids, and mentioned the potential role of optimised fluid resuscitation with accurate clinical assessment of volumes status during CPR, including considering medical history, exam findings and POCUS.[471]

Either isotonic saline or balanced crystalloids can be considered during CPR. A multicentre RCT ($n = 432$) compared the infusion of two units each of packed red blood cells (PRBC) and fresh frozen plasma versus 1 L of isotonic saline in patients with traumatic haem-

orrhagic shock.[472] The study was stopped earlier than the planned sample size of 490 patients due to the COVID-19 pandemic. No difference was observed in the primary outcome parameter (composite of mortality and lactate clearance failure) and secondary outcomes: i) mortality and ii) lactate clearance failure); serious adverse events were comparable. The role of intra- and peri-arrest administration of hypertonic fluids,[473] blood, and blood products for non-traumatic cardiac arrest remains uncertain,[472] and these products should only be used in clinical trials. Small bolus doses (e.g., 20 mL) of fluid, or a slow fluid infusion should be used to flush drugs given during CPR.

Other drugs

Calcium. ILCOR updated the CoSTR for calcium administration during cardiac arrest based on a systematic review that included three randomized trials and eight observational studies in adult cardiac arrest.[474] The largest and most recent trial of 391 patients comparing calcium chloride with placebo during OHCA was terminated early because an interim analysis suggested potential harm.[475,476] While no statistically significant differences were observed in ROSC, neurological outcomes at 30 days, or survival up to 1 year, patients receiving calcium had lower rates of favourable neurological outcome at 90 days and 1 year, with a signal of harm across multiple outcomes. Two older, smaller trials of cardiac arrest patients with refractory asystole or pulseless electrical activity, along with most observational studies, also failed to show any survival benefit.[474,477,478] These data led ILCOR to recommend against routine calcium administration in both OHCA (strong recommendation, moderate certainty evidence) and IHCA (weak recommendation, low certainty evidence).[1] Consistent with ILCOR, the ERC recommends not routinely giving calcium during cardiac arrest. As noted in the ERC Guidelines 2025 Special Circumstances, for cardiac arrest caused by suspected hyperkalaemia, ILCOR found insufficient evidence to recommend for or against calcium administration.[114]

Sodium bicarbonate. ILCOR updated the CoSTR for sodium bicarbonate administration during cardiac arrest based on a systematic review including three RCTs and three observational studies in OHCA.[1] Meta-analyses found no benefit of sodium bicarbonate administration compared to standard care for short-term survival, survival to hospital discharge, and survival with favourable neurological outcome at one month.[479–481] Supporting observational data showed similar results.[482–487] Although the evidence is inconclusive, sodium bicarbonate is commonly given during cardiac arrest.[487–489] ILCOR suggests against routine sodium bicarbonate administration in both OHCA (weak recommendation, low certainty evidence) and IHCA (weak recommendation, very low certainty evidence).[1] The ERC does not support the routine administration of sodium bicarbonate during cardiac arrest, unless there is a specific indication.

Corticosteroids. The guidance for administering corticosteroids during cardiac arrest, whether given alone or in combination with vasopressin, is based on an ILCOR evidence update and systematic review of individual participants data.[1,490,491] In adult IHCA, three RCTs comparing vasopressin and methylprednisolone to placebo found higher rates of ROSC and favourable neurological outcome at hospital discharge, but no significant improvement in survival, longer-term neurological outcome, or health-related quality of life up to 90 days.[137,492,493] Secondary analyses of the largest trial also found no difference in haemodynamic or long-term outcomes for vasopressin and methylprednisolone.[494,495] The two earlier smaller trials that administered corticosteroids both during and after ROSC reported improved outcomes overall.[492,493] Given that no consistent survival benefit was identified in meta-analyses, ILCOR suggests against using vasopressin and corticosteroids in addition to usual care for adult cardiac arrest.[1] For corticosteroids as a standalone intervention during cardiac arrest, early small trials have found no meaningful benefit, including three trials in OHCA and one in IHCA.[496,497] Based on these data, and considering the practical challenges of incorporating additional medication into resuscitation protocols, the ERC recommends against the routine use of corticosteroids either alone or in combination with vasopressin during cardiac arrest except when this is done as part of a clinical trial.

ALS for cardiac arrest in highly-monitored settings

Patients in highly monitored settings such as critical care areas, operating or recovery rooms, and cardiac catheterisation laboratories are closely and continuously monitored, and causes of unexpected cardiac arrest may be promptly reversible, particularly when detected immediately (e.g., a sudden arrhythmia in the ICU or a relative overdose of induction drug in the operating room). If blood pressure is monitored continuously using an indwelling arterial cannula, sudden changes in blood pressure can be detected almost immediately and a pulsatile arterial waveform may be seen even if peripheral and central pulses are no longer palpable. A sudden decrease in cardiac output, including cardiac arrest, may be detected very quickly by a sudden decrease in $ETCO_2$ if this is being monitored continuously. These are just some of the features of cardiac arrest in a highly monitored setting that may warrant adjustment of the doses of resuscitation drugs, changes in defibrillation strategy, and different indications for starting chest compressions.

A recent Royal College of Anaesthetists National Audit in the United Kingdom (National Audit Project 7) estimated the incidence of intra-operative cardiac arrest to be 3 in 10,000.[498] Of the 548 patients with intraoperative cardiac arrest during the one-year period of study, half had invasive blood pressure monitoring. Sustained ROSC was achieved in 78 % of patients and 62 % were alive at the time of reporting. Only 70 patients (13 %) had a shockable rhythm. Among the patients with an initial rhythm of PEA or bradycardia, the three most reported triggers for starting CPR were an impalpable pulse (57 %), severe hypotension (47 %), and a reduction in $ETCO_2$ (25 %).

In adults during general anaesthesia, it has been suggested that chest compressions should be started if the systolic blood pressure decreases and remains <50 mmHg despite interventions.[499,500] This approach is likely also applicable to any patient undergoing invasive arterial monitoring (e.g., in an critical care setting). A systolic blood pressure <50 mmHg is likely to be associated with impalpable pulses[501] and the benefit from the additional cerebral and coronary blood flow produced by chest compressions will likely outweigh the risk of harm from chest compressions.[499]

Although the ERC Guidelines 2025 ALS recommend a dose of 1 mg adrenaline, the optimal dose of adrenaline to treat cardiac arrest is unknown.[502] Smaller doses are likely appropriate when adrenaline is first given IV for profound hypotension, when there is a high probability of a low-flow state during PEA or severe bradycardia, or when there is a very short time between the onset of cardiac arrest and injection of adrenaline. With continuous monitoring, car-

diac arrest can be diagnosed immediately and treated rapidly with the likelihood of restoring spontaneous circulation very quickly. Under these circumstances, giving a 1 mg bolus of adrenaline may lead to severe hypertension and tachyarrhythmias. Thus, we suggest that adrenaline is given initially in increments (e.g., 50–100 μg IV) rather than a 1 mg bolus, but if 1 mg in total has been given with no response, further IV adrenaline doses of 1 mg are given at the usual 3–5 min intervals. Australasian guidelines for resuscitation after cardiac surgery include similar recommendations.[503] When continuously monitoring arterial blood pressure it is reasonable to aim to achieve a diastolic blood pressure higher than 30 mmHg by combining high-quality chest compressions and titrated adrenaline (by giving 50–100 μg boluses or an infusion) (see physiology-guided CPR).[504,505]

The International Liaison Committee on Resuscitation does not recommend the precordial thump for established cardiac arrest, citing its low success rate documented in a systematic review – although already de-emphasised in previous ERC guidelines, we have removed it completely from these Guidelines.[506] In two studies of patients undergoing electrophysiological testing, it terminated malignant ventricular arrhythmias (VT or VF) in only 1 of 80 and 2 of 155 cases, respectively—all cases were VT, and none of the 49 VF cases responded.[507,508] In an OHCA study, a precordial thump restored circulation in 5 of 103 patients (3 VF, 2 pVT) but worsened rhythms in 10 cases.[509] Another Italian OHCA study found it effective in only 3 of 144 patients, all initially in asystole.[510]

Physiology-guided CPR

The quality of CPR is associated with survival and the ability to monitor the quality of CPR is fundamental to its improvement.[511] The quality of CPR can be measured by monitoring the performance of the rescuer to ensure adherence to guidelines; this might include compression rate, depth and recoil, chest compression fraction and ventilation rate and volume. Many of the current defibrillator-monitors are capable of monitoring and providing these metrics in real time. Another way of monitoring CPR performance is to assess its effect on the patient's physiology using intra-arterial blood pressure, capnography or cerebral oximetry.

During low flow conditions, $ETCO_2$ values are more strongly associated with cardiac output and pulmonary blood flow than with ventilation.[512] The use of $ETCO_2$ to guide the quality of chest compressions was first described almost 40 years ago.[513] Two observational studies have subsequently shown that chest compression depth but not rate is associated with $ETCO_2$ values,[514,515] although a third observational study did show a small association between compression rate and $ETCO_2$.[516] In a pig model of neonatal asphyxial cardiac arrest, chest compressions aimed at maximising $ETCO_2$ increased the rate of ROSC compared with a control group of standard CPR.[517]

A systematic review of haemodynamic-directed feedback during CPR identified six animal studies.[518] Four of these studies examined the effect of haemodynamic-directed CPR on survival in swine. The hemodynamic-directed CPR groups had chest compression depth titrated to a systolic blood pressure (SBP) of 100 mmHg and vasopressors titrated to maintain a coronary perfusion pressure (CPP) >20 mmHg. Pooled results showed that 35/37 (94.6 %) of the animals survived in the haemodynamic-directed CPR groups and 12/35 (34.3 %) survived in the control groups ($p < 0.001$).

Most of the clinical studies of physiology-directed CPR have involved children. In a stepped-wedge cluster randomised trial in 18 paediatric intensive care units, a bundled intervention comprising physiologically focused CPR training at the point of care and structured clinical event debriefings did not improve survival to hospital discharge with favourable functional outcome.[519] In this study, when an arterial line or $ETCO_2$ was in place, clinicians were educated to aim for a systolic blood pressure above 100 mmHg in older children (i.e. not neonates or infants) and an $ETCO_2$ above 25 mmHg. Observational data from this study demonstrate that achieving a mean diastolic BP (DBP) during CPR greater than 30 mmHg for older children is associated with higher rates of survival to hospital discharge.[504]

In one of the first clinical studies in adults on this topic, invasive blood pressure was measured during CPR in 104 patients.[520] A chest compression rate of 100–120 min^{-1} and compression depth $\geq$6 cm was associated with a DBP $\geq$ 30 mmHg in both femoral and radial recordings. However, although there was a weak upward trend in blood pressure as compression depth increased in individual subjects, deeper compression depth did not result in a higher blood pressure in all patients. In a more recent study, 80 OHCA adults had invasive blood pressure monitoring during CPR delivered by helicopter emergency medicine service (HEMS) clinicians.[505] The maximum, average and delta-DBP (difference between the initial and maximum values); and maximum and average mean arterial pressure (MAP) were positively associated with ROSC. Maximum DBP had an optimal threshold value of 35 mmHg (sensitivity 94.1 %; specificity 58.7 %) for predicting ROSC. The odds ratio for ROSC was 1.05 (95 % CI 1.03–1.08) for every 1 mmHg increase in maximum DBP.

A consensus statement from the American Heart Association suggested several physiological targets during CPR, all of which were based on expert consensus:[511]

- If both an arterial line and a central venous catheter are in situ during cardiac arrest, aim to achieve a coronary perfusion pressure (CPP) >20 mmHg.
- If only an arterial line is in situ during cardiac arrest, aim to maintain a DBP > 25 mmHg.
- If only capnography is available during cardiac arrest, aim to achieve an $ETCO_2$ > 20 mmHg (2.67 KPa).

Given the recent observational data,[504,505,520] the ERC suggests aiming for a diastolic blood pressure of $\geq$30 mmHg when using intra-arterial blood pressure monitoring for physiology-guided CPR. This is only feasible in very specialist settings with expert resuscitators – assessing diastolic BP in real time may be challenging in some patients. Further studies are still needed to increase our understanding of the optimum blood pressure target during CPR and how to measure it in real time.

Other potential physiological targets during CPR include cerebral oximetry and EEG. Higher cerebral oxygenation saturation during CPR is associated with ROSC,[521] but although theoretically cerebral oxygenation could be used as a physiological target during CPR, this has yet to be investigated (see below).

Waveform capnography during advanced life support

End-tidal CO_2 ($ETCO_2$) is the partial pressure of carbon dioxide (PCO_2) measured at the end of expiration. It reflects cardiac output, tissue perfusion, pulmonary blood flow, and minute ventilation. Carbon dioxide is produced in perfused tissues by aerobic metabolism, transported by the venous system to the right side of the heart and

pumped to the lungs by the right ventricle, where it is removed by alveolar ventilation.

Waveform capnography enables a continuous, non-invasive measurement of PCO_2 in the exhaled air during CPR. In the typical capnogram, the $ETCO_2$ recorded at the end of the expiratory plateau best reflects the alveolar PCO2.[522] $ETCO_2$ is most reliable when the patient's trachea is intubated, but it can also be detected with a supraglottic airway device or bag mask.[523]

The aims of monitoring waveform capnography during CPR include:

- **Confirming tracheal tube placement** (this has been addressed above).
- **Monitoring the quality of CPR**. $ETCO_2$ monitoring during CPR enables the measurement of the ventilation rate helping rescuers avoid hypo- or hyperventilation.[384,523] Observational studies on adults with in-hospital or OHCA have shown that $ETCO_2$ values are proportional to the chest compression depth measured using transthoracic impedance. In contrast, variations in the chest compression rate do not affect $ETCO_2$.[514,515]
- **Detecting ROSC during CPR**. When ROSC occurs, $ETCO_2$ may rise three times above the values during CPR. Capnography during CPR might, therefore, help avoid unnecessary chest compression or potentially harmful administration of adrenaline in a patient with ROSC. However, no consistent amount of $ETCO_2$ rise has been identified as a criterion for diagnosing that ROSC has occurred. In addition, the rise in $ETCO_2$ associated with ROSC is not immediate and can start 10 min or more before a palpable pulse is detected.[524–526]
- **Prognostication during CPR**. Lower $ETCO_2$ values during CPR indicate a lower likelihood of ROSC. Previous evidence suggested that failure to achieve an $ETCO_2$ value >10 mmHg (1.33 kPa) was associated with a poor outcome.[527,528] This threshold has also been suggested as a criterion for withholding e-CPR in refractory cardiac arrest.[529] However, recent evidence has shown that patients may survive with ETCO2 values below 10 mmHg during CPR. In a study on 617 adults with refractory OHCA from non-shockable rhythm, of whom 615 (99.3 %) died before hospital discharge, $ETCO_2$ at 30 min was still above 10 mmHg in 88 % of non-survivors. In one of the two survivors, 30-min $ETCO_2$ was below 10 mmHg.[530] In a study on 14,122 adult non-traumatic OHCA from all rhythms, 4.2 % of the 9,226 patients who subsequently achieved ROSC and 3.3 % of those who survived to hospital discharge had $ETCO_2 < 10$ mmHg at 20 min.[531] In that study, the adjusted odds of mortality in patients with maximum prehospital $ETCO_2$ values above 50 mmHg (6.67 Kpa) were 50 % higher than in patients with $ETCO_2$ values of 30–40 mmHg (4.0–5.33 KPa), probably reflecting hypoventilation. Other factors affecting $ETCO_2$ during cardiac arrest include the presence of airway closure,[532] and the cause of arrest.[533–535]

Besides single values of $ETCO_2$, the changes in $ETCO_2$ during CPR have also been investigated to predict ROSC.[536,537] In a recent secondary analysis of 1113 patients enrolled in the multicentre cluster randomised PART trial, in which patients were ventilated with supraglottic airways or tracheal tube, the median $ETCO_2$ increased from 30.5 at 10 min to 43.0 mmHg (p for trend <0.001) five minutes before the end of resuscitation in ROSC patients, while it declined from 30.8 to 22.5 mmHg (p for trend <0.001) in non-ROSC patients, respectively.[538] After adjusting for major confounders, the slope of the $ETCO_2$ during CPR remained associated with both ROSC and 72-h survival (OR 1.45 [1.31–1.61] and 1.33 [1.20–1.45] respectively). $ETCO_2$ trends are probably more appropriate than point values for predicting ROSC during CPR.[528] In the study on refractory non-shockable OHCA mentioned above, while only one of the two survivors to discharge had an $ETCO_2$ above 10 mmHg at 30 min, both had an $ETCO_2$ increase from the initial value to 30 min (specificity 13 % vs 33 %).[530] In a multicentre study on 668 OHCA patients of any aetiology and rhythm, both an $ETCO_2$ value at intubation greater than 20 mmHg (2.67 KPa) and its increase 10 min later were independent predictors of survival to hospital admission and survival at hospital discharge. However, after adjustment for bystander and CPR status, presenting rhythm and EMS arrival time, only the $ETCO_2$ change at 10 min remained an independent predictor of outcome.[539]

Evidence regarding the prognostic value of $ETCO_2$ is based on observational, unblinded studies, which may have caused a self-fulfilling prophecy. Although $ETCO_2$ values below 10 mmHg after prolonged CPR are strongly associated with unfavourable outcome (no ROSC or death before hospital discharge), the ERC recommends that they should not be used alone as a mortality predictor or for deciding to stop a resuscitation attempt.

Near infra-red spectroscopy (NIRS), and EEG monitoring during CPR

Although NIRS and EEG monitoring during CPR were within the scope of the ERC Guidelines 2025 ALS update, the ERC has not made any recommendations for their use and the rea sons are set out below. Near infrared spectroscopy is a non-invasive method to monitor the oxygenation of the combination of arterial and venous blood in the brain. NIRS monitoring can be conducted with external electrodes placed on the forehead of the patient. With the emission of infrared light, the monitor can provide the regional oxygen saturation proportion in tissue and blood. When applied to the forehead of a patient this will provide an estimation of the amount of oxygen saturation in brain tissue. Given the risk of hypoxic-ischaemic brain injury in patients undergoing CPR this has raised much interest. According to a systematic review conducted in 2021, NIRS monitoring may provide information on the likelihood of ROSC.[540] The systematic review including 17 studies showed that higher initial cerebral regional oxygen saturation (rSO_2) values were associated with the likelihood of ROSC. Thus far only one study has compared $ETCO_2$ and NIRS and this study suggested superiority for NIRS compared with $ETCO_2$.[541] This finding should however be confirmed in future studies. On the other hand, studies have failed to show any association of NIRS values with the quality of chest compressions, the amount of oxygen in the blood or the administration of adrenaline.[366,542] Thus, it is unclear if NIRS can be used to modify the delivery of resuscitation measures in any way. No study to date has assessed the cost effectiveness of NIRS monitoring. Therefore, there is not enough evidence to recommend NIRS as a monitoring tool for patients undergoing CPR.

EEG monitors the electric activity of the brain noninvasively. Changes in EEG correlate with changes in cerebral blood flow. Some studies have assessed the feasibility of using EEG during resuscitation to monitor the efficacy and outcome of CPR.[543] The EEG methods used include traditional multichannel EEG as well as the raw EEG signal from monitors of the depth of anaesthesia, including the bispectral index (BIS®). The existing evidence is inconclusive and comes mainly from smaller observational studies and case-reports. At present, the use of EEG is not recommended by the

ERC either for monitoring the performance of CPR or estimating the outcome.

Use of ultrasound imaging during advanced life support

Point-of-care ultrasound (POCUS) imaging is already commonly used in emergency care settings. Its use during CPR is also increasing. Previous and current guidance emphasises the need for skilled POCUS operators and minimising interruption in chest compression to acquire images.[60,215]

An ILCOR systematic review assessed the role of POCUS during cardiac arrest as a prognostic tool in which assessment of cardiac motion informs the likelihood of achieving ROSC and clinical decisions to terminate resuscitation.[544] The review identified several limitations such as inconsistent definitions and terminology around sonographic evidence of cardiac motion, low inter-rater reliability of findings, low sensitivity and specificity for outcomes, confounding from self-fulfilling prophecy when terminating resuscitation in unblinded settings as well as unspecified timing of POCUS. The review concluded that no sonographic finding had sufficiently diagnostic accuracy to support its use as a sole criterion to terminate resuscitation. A 2025 ILCOR Evidence Update[1] identified additional studies with small sample sizes, and heterogeneous POCUS findings and clinical outcomes.[545–548] All studies were potentially biased by lack of blinding of the resuscitating team to POCUS findings. There was a lack of agreement in the interpretation of acquired views in most of studies, which highlighted the inherent real-time clinical challenge of time-limited image acquisition and the need for skilled POCUS operators.

A further ILCOR systematic review assessed the role of POCUS to diagnose treatable causes of cardiac arrest such as cardiac tamponade, pneumothorax, pulmonary embolism, myocardial infarction, aortic dissection, and hypovolaemia.[549] There was a high degree of clinical heterogeneity and a critical risk of bias, which precluded a meta-analysis. The certainty of evidence was very low, and individual studies were difficult to interpret. However, the review stressed the issue of misinterpreting POCUS findings as the cause of cardiac arrest as opposed to an incidental finding. For example, unilaterally absent lung sliding could indicate a small pneumothorax or mainstem bronchial intubation, rather than a tension pneumothorax. Likewise, visualised peritoneal fluid could be ascites, rather than an acute haemorrhage; a pericardial effusion could be present without cardiac tamponade; and right heart dilation can occur during CPR without a massive pulmonary embolism. Right ventricular dilation a few minutes after the onset of cardiac arrest as blood moves from the systemic circulation to the right heart along its pressure gradient [550] was consistently observed in a porcine model of cardiac arrest caused by hypovolaemia, hyperkalaemia, and primary arrhythmia.[551] This is a common trans-oesophageal echocardiography (TOE) observation in patients with OHCA regardless of the cause.[552]

Since 2015, the ERC ALS guidelines recommend a transthoracic sub-xiphoid view with the probe placed just before chest compressions are paused for a planned rhythm assessment,[215] minimising additional interruptions in chest compressions, which may delay or impede other therapies.[553,554] A strategy to deal with this is to record brief sonographic video clips during pulse/rhythm checks (less than 10 s) and then view/interpret them after the resumption of chest compressions. In addition, sonographers may pre-localise the approximate acoustic window during chest compressions with subsequent fine-tuning during pauses in CPR.[555] Finally, to avoid prolonged pauses in chest compressions, when feasible the sonographer should not be simultaneously leading the team and, or doing rhythm checks.[554]

Another possibility for reducing hands-off time during cardiac arrest is the use of TOE. In 2021, a systematic review of TOE in cardiac arrest concluded that because of the heterogeneity of studies, small sample size and inconsistent reference standard, the evidence for TOE in cardiac arrest resuscitation is of low certainty and is affected by a high risk of bias.[556] Further studies are needed to better understand the true diagnostic accuracy of TOE in identifying reversible causes of arrest and cardiac contractility. Since then, additional case series and observational studies have been published, which showed that in experienced hands, TOE provides useful diagnostic and therapeutic information and that the rate of adequate cardiac visualisation can be excellent with a low complication rate.[557] During CPR, TOE may help to improve hand positioning for chest compression and improve left ventricular compression.[558,559] In an observational study, improving left ventricular compression was associated with increased ROSC.[548] The use of TOE during cardiac arrest requires additional equipment and expertise.

Devices

Mechanical chest compression devices

Since the 2021 guidelines,[60] ILCOR has published updated recommendations for the use of mechanical chest compression. The ILCOR 2024 systematic review identified 14 reports of 11 RCTs conducted post-2000.[1] Trials before this date were not included because of the significant changes to CPR and cardiac arrest treatment that have occurred since 2000. Three new RCTs were identified since the 2021 ERC guidelines, each providing very low certainty evidence.[560–562] One study, enrolling 1191 patients, assessed the use of the LUCAS device following OHCA. No difference in ROSC (RR 0.90 [95 % CI 0.62 to 1.32]) or survival to 30 days (RR 0.89 [95 % CI 0.41 to 1.92]) was found when comparing LUCAS with manual CPR.[560] Two studies compared LUCAS with manual CPR following IHCA.[561,562] One study enrolled patients with IHCA (but not in the emergency department) with non-shockable rhythms (n = 127).[561] No benefit in survival with favourable neurological outcome was found with LUCAS compared with manual CPR (RR 1.13 [95 % CI 0.13 to 9.72]). A trial enrolling patients sustaining witnessed cardiac arrest in the emergency department found no difference in ROSC when using a LUCAS device compared with manual CPR (RR 0.80 [95 % CI 0.55 to 1.173]).[562] No new RCTs reporting safety outcomes were identified in the ILCOR systematic review. Consistent with the ILCOR treatment recommendations, the ERC recommends considering mechanical chest compression only if high-quality manual chest compressions are not practical (e.g., during percutaneous coronary intervention or ECMO cannulation) or compromise provider safety (e.g. during transport). When a mechanical device is used, delays in the initial defibrillation attempt should be avoided and pauses to chest compression during device deployment minimised. Mechanical chest compression should only be used by trained teams familiar with the device.

Resuscitative endovascular balloon occlusion of the aorta (REBOA)

Resuscitative endovascular balloon occlusion of the aorta (REBOA) is a technique in which blood flow through the aorta is

occluded by inflating an intra-aortic balloon. The use of REBOA has not yet been addressed by ILCOR but was included in the scope of the ERC guidelines. REBOA has been used in the management of haemorrhagic shock and traumatic cardiac arrest.[563,564] However, in a recent RCT the in-hospital use of REBOA in patients with exsanguinating traumatic haemorrhage did not improve 90-day survival and may even have increased mortality compared with standard care alone.[565] It has recently been proposed as an adjunctive treatment for patients with non-traumatic cardiac arrest because of its potential to redistribute blood flow to organs proximal to the occlusion, thereby increasing cerebral and coronary perfusion.[566]

In animal models of non-traumatic cardiac arrest, inflation of REBOA increases central diastolic arterial pressure, which is a surrogate marker of coronary artery pressure and cerebral perfusion.[567,568] However, human evidence is currently limited to case reports[569–573] and small case series with a total of 78 patients.[574–582] These studies aimed to assess the feasibility of positioning REBOA during resuscitation. REBOA positioning was successful in 72 cases (92 %) and ROSC was achieved in 35 cases (45 %) but only 16 (20.5 %) had sustained ROSC. Approximately 80 % of patients with sustained ROSC died (13/16); of these, nearly half died from brain injury[570,571,577,582] and less frequently from withdrawal of life-sustaining treatment or re-arrest after balloon deflation.[576]

Currently, there are no data to support the use of REBOA in non-traumatic OHCA. There are two (one pre-hospital; one emergency department) ongoing efficacy RCTs which aim to evaluate the ability of REBOA to increase ROSC in adult, witnessed, non-traumatic cardiac arrest and its potential effects on survival and neurological outcome.[583,584]

Intra-arrest cooling

Cooling of patients during CPR may potentially alleviate reperfusion injury and prevent or reduce hypoxic-ischaemic brain injury. The most recent ILCOR systematic review on temperature control in cardiac arrest patients found no evidence of improved outcome in patients treated with pre-hospital cooling in patients with OHCA.[585–587] These reviews did not separate cooling conducted during or after ROSC. This may be important with respect of both the effects and side effects of the intervention. A systematic review focusing on cooling during CPR was published in 2021.[588] This review identified four RCTs (two of high certainty and two of moderate certainty of evidence) including 2305 patients that compared intra-arrest cooling with cooling started in the hospital.[467,589–591] Two studies used evaporative intranasal cooling: one used cooling with cold IV fluids (up to 2000 mL) and one used a combination of cold fluids (up 2000 mL) and external cooling with gel pads. In comparison with the control patients, the use of cooling during CPR was not associated with improved favourable neurological outcome (OR 0.96 [95 % CIs 0.68–1.37]; $p = 0.84$), any change in ROSC (OR 1.11 [95 % CIs 0.83–1.49]; $p = 0.46$) or survival to hospital discharge (OR 0.91 [95 % CIs 0.73–1.14]; $p = 0.43$). A further analysis of the method of intra-arrest cooling did not appear to influence the results. Therefore, the use of intra arrest cooling is not currently recommended by the ERC (unless there is severe hyperthermia). The ongoing PRINCESS 2 trial will assess the effect of intranasal evaporative cooling in patients resuscitated from a shockable initial rhythm.[592]

Extracorporeal CPR

Extracorporeal CPR (ECPR) is the rapid deployment of veno-arterial extracorporeal membrane oxygenation (VA-ECMO) during ongoing CPR to restore and maintain organ perfusion in patients in whom conventional CPR is unsuccessful in achieving a sustained ROSC.[593] The use of ECPR has continued to increase for both IHCA and OHCA in recent years[594] despite uncertainty in evidence.[595,596]

Since 2020, three RCTs of ECPR have been published. The Advanced Reperfusion Strategies for Patients with OHCA and Refractory Ventricular Fibrillation Trial (ARREST) demonstrated higher survival to hospital discharge with ECPR compared to conventional CPR in OHCA patients with an initial shockable rhythm.[597] The Prague OHCA study found improved survival with a favourable neurological outcome at 30 days but not at six months (primary outcome) among 264 patients with all-rhythms refractory OHCA.[598] Of note, these were both single-centre trials performed in well-established ECPR systems. A third, multi-centre trial conducted in 10 cardiosurgical centres, Early Initiation of Extracorporeal Life Support in Refractory OHCA Trial (INCEPTION) showed no difference between ECPR and conventional CPR in OHCA patients with an initial shockable rhythm.[599] Additionally, a small pilot RCT, the Extracorporeal Cardiopulmonary Resuscitation for Refractory Out-of-Hospital Cardiac Arrest (EROCA) trial, failed to meet the feasibility goal of transporting patients to an ECPR-capable emergency department within 30 min from OHCA.[600] A meta-analysis of these four trials demonstrated higher survival with favourable neurological outcome with ECPR compared to conventional CPR.[601] A Bayesian meta-analysis found a posterior probability of a clinically relevant ECPR-based treatment effect on 6-month neurologically favourable survival of 71 % in patients with all rhythms and 76 % in shockable rhythms.[602] For IHCA, RCTs have not been conducted, and a meta-analysis of observational data found that ECPR was effective in improving survival and favourable neurological outcomes.[603]

The use of ECPR during cardiac arrest was addressed by an ILCOR systematic review in 2022, which was updated in 2024.[192,596] The ILCOR ALS Task suggested, and the ERC recommends that ECPR may be considered as a rescue therapy for selected adults with IHCA and OHCA when conventional CPR is failing to restore spontaneous circulation in settings where this can be implemented (weak recommendation). The overall certainty of evidence was rated as low for OHCA and as very low for IHCA (downgraded further because all evidence was in OHCA).[1]

ECPR is not intended for the entire population of cardiac arrest patients, with only approximately 10 % of OHCA cases being eligible candidates.[598] However, there are no universally agreed selection criteria for ECPR, and practices vary widely among centres, particularly regarding the inclusion of older patients, those with initial non-shockable rhythms (pulseless electrical activity or asystole), and those with longer low-flow times. A recent systematic review and meta-analysis found that younger age, initial shockable rhythm, witnessed arrest, immediate CPR, pre-cannulation ROSC at any time, and shorter low-flow times are associated with increased likelihood of survival with favourable neurological outcome.[604] Adopting more liberal selection criteria impacts survival and survival with favourable neurological outcome[605–607] but also influences organ donation rates.[608] Conversely, more restrictive criteria may exclude potential survivors and organ donors.

The ERC, European Extracorporeal Life Support Organization (EuroELSO), European Society of Intensive Care Medicine (ESICM),

European Society of Emergency Medicine (EuSEM), European Society of Anaesthesia and Intensive Care (ESAIC), and the European Board of Cardiovascular Perfusion (EBCP) have produced consensus guidelines for ECPR in adults and the brief guidance below is based on these guidelines. Given the time-sensitive nature of ECPR, in systems where ECPR is available, it is crucial for clinicians to promptly recognise when a patient with ongoing ALS is in refractory cardiac arrest (i.e., three consecutive unsuccessful defibrillation attempts or 10 min of resuscitation in case of non-shockable rhythms) and might be a suitable candidate, enabling rapid activation of the ECPR team.

Currently used selection criteria for initiating ECPR include:

- Younger patients (upper age limit of 50–75 years), no perceived frailty, and absence of major comorbidities.
- Witnessed cardiac arrest with immediate CPR (i.e., a no-flow duration of $\leq$ 5 min)

In addition to the previous criteria, the following criteria are commonly applied:

- Initial shockable or PEA rhythms.
- Estimated time to establish ECPR from starting CPR (i.e., low-flow time) is ideally within 45 to 60 min.
- Known or suspected treatable underlying cause of cardiac arrest.
- ROSC at any time prior to cannulation.
- Presence of signs of life during CPR.
- $ETCO_2$ > 10 mmHg (1.3 kPa).
- Mechanical CPR during transport.

Whether initiating ECPR for OHCA in the pre-hospital setting is superior to in-hospital setting remains uncertain,[609–611] and ongoing clinical trials aim to address this question (ON-SCENE [ClinicalTrials.gov NCT04620070], RACE [NCT06789978], PACER [NCT06177730]).

Peri-arrest arrhythmias

Prompt identification and treatment of life-threatening arrhythmias may prevent cardiac arrest or its recurrence. This section offers guidance and treatment algorithms for the non-specialist ALS provider. The scope is to focus on arrhythmias occurring pre-arrest or immediately post-ROSC and causing life-threatening instability. In case of persistent arrhythmia, the first goal should be to ascertain the patient's stability and seek advice from a specialist or more experienced physician. Electrical cardioversion is required in the peri-arrest patient with a clinically unstable tachyarrhythmia, while pacing is used in refractory, clinically important bradycardia. The key interventions are summarised in Fig. 8. and Fig. 9.

These ERC Guidelines 2025 ALS follow recommendations published by international cardiology societies, including the European Society of Cardiology (ESC), the European Heart Rhythm Association (EHRA), the European Society of Cardiothoracic Surgeons, the American Heart Association (AHA), the American College of Cardiology (ACC) and the Heart Rhythm Society (HRS).[86,115,612–615] Table 6 summarises the supporting evidence for vagal manoeuvres and some of the more commonly used drugs for the treatment of arrhythmias.

Ventricular arrhythmias occurring peri-arrest usually originate from a complex interplay between underlying structural or electrical heart disease, external triggering factors and dominance of the sympathetic limb of the autonomic nervous system.[616] Reversible triggering factors include acute myocardial ischaemia, electrolyte imbalance, fever or hypothermia, hormonal factors, sepsis, starvation and decompensated heart failure.[617,618] For patients with wide QRS arrhythmias peri-arrest, expert help should be pursued whenever possible to either help with the termination of the arrhythmia or to prevent recurrences.

Treatment of ventricular arrhythmias depends on the haemodynamic consequences of the arrhythmia, its morphology on ECG and the underlying myocardial substrate.[617] For patients with monomorphic VT and structural heart disease or uncertain myocardial substrate, current European Society of Cardiology (ESC) guidelines (which are supported by the ERC) recommend synchronised external cardioversion, even when the patient is haemodynamically stable, as haemodynamic deterioration may occur if monomorphic VT is not treated. Pharmacological treatment may be an alternative if the risk of sedation/anaesthesia is high.[612]

For patients with polymorphic VT, precipitating factors should be sought and removed.[619] In case there is a prolongation of the QTc interval at baseline, IV Mg^{2+} and K^+ infusion might help. Also increasing heart rate with isoproterenol or temporary pacing should be considered.[620]

In the case of recurrent ventricular arrhythmias, uncontrolled with non-invasive measures, consider reprogramming the ICD (if one is already in place), suppressing sympathetic activity (beta-blockers, sedation, autonomic modulation), starting mechanical circulatory support and referring the patient for catheter ablation.[616,620]

The ESC has published recent guidelines for the acute management of regular tachycardias without an established diagnosis. The guidelines for treating regular narrow QRS ($\leq$ 120 ms) and life-threatening wide QRS (> 120 ms) tachycardias have been incorporated into the tachycardia algorithm. The ESC Guidelines provide more detailed recommendations and evidence for treating rhythms once a specific diagnosis of the rhythm has been made.[115]

In a randomised trial involving haemodynamically stable patients with wide QRS-complex tachycardia of unknown aetiology, procainamide was associated with fewer major adverse cardiac events and a higher proportion of tachycardia termination within 40 min, compared with amiodarone.[621] However, in many countries, procainamide is either unavailable or unlicensed.

In critical low-perfusion conditions like hypovolaemic, cardiogenic, or distributive shock, sinus tachycardia is a compensatory response to enhance blood flow to ischaemic tissues. In patients with sinus tachycardia, the primary focus of treatment should be to address the underlying cause of sinus tachycardia, such as through fluid administration. Rescuers should avoid trying to normalise the heart rate through rate-control strategies, such as beta-blocker administration, as this may result in patient deterioration or even haemodynamic collapse.

In acute settings, rescuers should evaluate and manage underlying causes or triggering factors that initiate AF such as sepsis, fluid overload or cardiogenic shock.[622] The selection of rate versus rhythm control strategy and the appropriate drug will depend on the patient's characteristics, the presence of heart failure and the haemodynamic profile.[623] In patients with AF and acute or worsening haemodynamic instability, electrical cardioversion is recommended.[624,625]

The rate of pharmacological cardioversion to sinus rhythm in patients with recent onset AF may be high, ranging from 50 to 95 % in 10 min to 6 h with the use of fast-acting drugs such as flecainide (200–300 mg orally once), propafenone (450–600 mg orally once), ibutilide (if patient <60 kg, 0.6 mg kg^{-1}; if patient >60 Kg, 1 mg IV. The dose may be repeated after 10 min if the initial dose is ineffective) and vernakalant (3 mg kg^{-1} IV over a 10-min period.

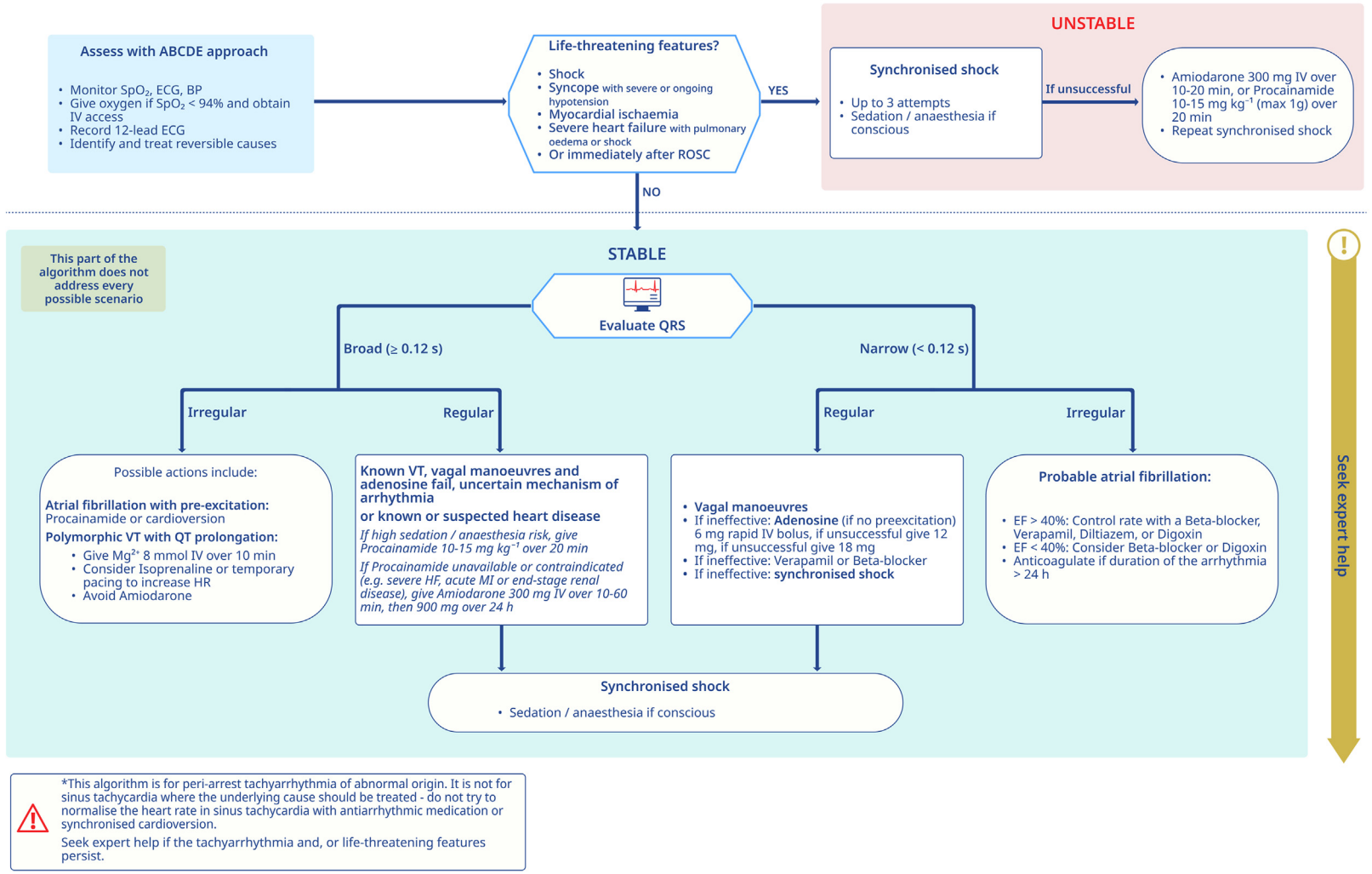

Fig. 8 – Peri-arrest Tachyarrhythmia Algorithm

ABCDE airway, breathing, circulation, disability, exposure; BP blood pressure; DC direct current; ECG electrocardiogram; EF ejection fraction; IV intravenous; ROSC return of spontaneous circulation; SpO_2 oxygen saturation measured with pulse oximetry; VT ventricular tachycardia.

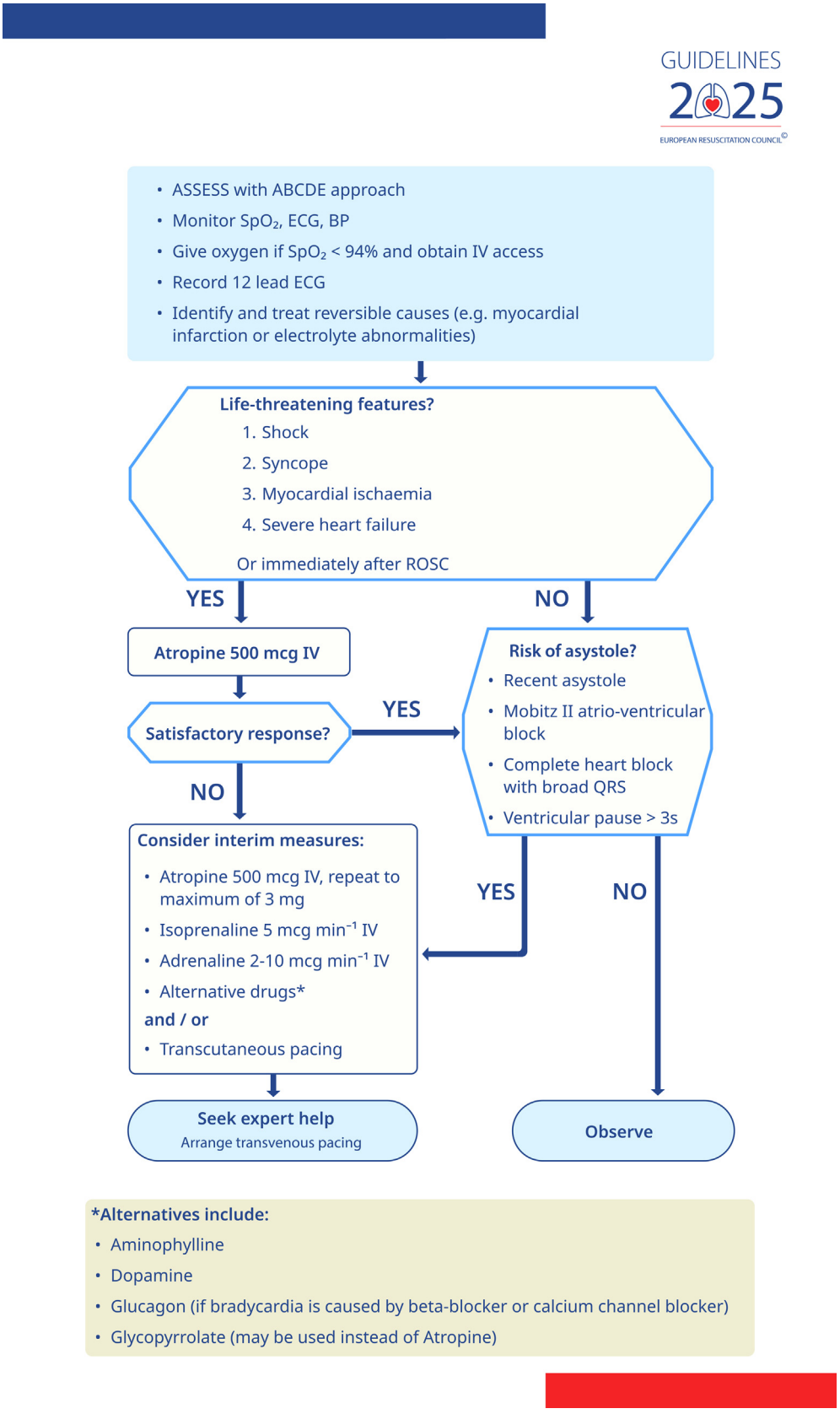

Fig. 9 – Bradycardia Algorithm

ABCDE airway, breathing, circulation, disability, exposure; BP blood pressure; ECG electrocardiogram; IV intravenous; SpO_2 oxygen saturation measured with pulse oximetry.

Table 6 – Recommendations for the acute management of narrow and wide QRS tachycardia. Drugs may be administered via peripheral IV in an emergency.

Drug/ procedure	Indication	Timing	Dose/delivery	Notes
Vagal Manoeuvres	Regular narrow QRS tachycardia Wide QRS tachycardia[612]		Blow into a 10 mL syringe with sufficient force to move the plunger	Preferably in the supine position with leg elevation[661,662]
Adenosine	Regular narrow QRS tachycardia Wide QRS tachycardia[612]	Recommended if vagal manoeuvres fail	Incremental, starting at 6 mg, followed by 12 mg IV. An 18 mg dose should then be considered	If no evidence of pre-excitation on resting ECG[115,663] When using an 18 mg dose, consider the tolerability/side effects in the individual patient.
Verapamil or diltiazem	Regular narrow QRS tachycardia	Consider if vagal manoeuvres and adenosine fail	Verapamil (0.075 – 0.15 mg/kg IV [average 5–10 mg] over 2 min) Diltiazem [0.25 mg/kg IV (average 20 mg) over 2 min].	Should be avoided in patients with haemodynamic instability, HF with reduced LV ejection fraction (<40)%[663]
Esmolol	Narrow QRS tachycardia Wide QRS	Heart rate control in SVT including AF and Atrial Flutter, and perioperative tachycardias Electrical storm	500 μg/kg IV optional bolus over 1 min followed by 50–300 μg/kg/min infusion	Short acting (elimination half-life 9 min), beta-1 selective, may be considered in patients with haemodynamic instability[625 616]
Landiolol	Narrow QRS tachycardia Wide QRS	Heart rate control in SVT including AF and Atrial Flutter, and perioperative tachycardias Electrical storm	Optional loading dose of 100 μg/kg IV over 1 min followed by 10–40 μg/kg/min infusion, or 1–10 μg/kg/min if impaired LV function	Ultra short half-life (about 4 min), beta-1 selective, may be considered in patients with haemodynamic instability[625 616]
Metoprolol	Narrow QRS tachycardia	Consider if vagal manoeuvres andadenosine fail	Metoprolol (2.5–15 mg given IV in 2.5 mg boluses),	For settings where Esmolol and Landiolol are not available
Procainamide	Wide QRS tachycardia	Consider if vagal manoeuvres and adenosine fail[612]	10–15 mg/kg IV over 20 min	[115,621]
Amiodarone	Narrow and wide QRS tachycardia	For acute management of VT or wide QRS tachycardia in the absence of an established diagnosis if vagal manoeuvres and adenosine fail[612]	300 mg IV over 10–60 min according to circumstances – followed by infusion of 900 mg in 24 h	Contraindicated in pre-excited AF-can – recognised by the fast, broad, irregular 'FBI' ECG pattern[115,612,664]
Lidocaine	Wide QRS tachycardia		50–200 mg bolus, then 2–4 mg/min	Reduce dose in shock states.
Magnesium	Polymorphic wide QRS tachycardia (torsades de pointes –TdP)		8 mmol[665] IV over 10 min. Can be repeated once if necessary.	Magnesium can suppress episodes of TdP without necessarily shortening QT, even when serum magnesium concentration is normal[612,616,666]

HF heart failure; LV left ventricular.

If AF persists 15 min after the completion of the initial infusion, a second dose of 2 mg kg^{-1} may be administered over 10 min).[626] However, only amiodarone is indicated in patients with severe left ventricular hypertrophy, heart failure with reduced ejection fraction and coronary artery disease, which are common in patients presenting with cardiac arrest.[627,628] The rate of successful restoration of sinus rhythm with amiodarone is 44 % at 8–12 h to several days after IV administration.[627] In selected patients, rate control may be preferred. For this purpose, beta-blockers and diltiazem/verapamil are preferred over digoxin because of their rapid onset of action and effectiveness in patients with high sympathetic tone. For patients with left ventricular ejection fraction less than 40 %, consider the smallest dose of beta-blocker to achieve a heart rate of less than 110 min^{-1} and add digoxin if necessary.[625]

Evidence for the treatment of patients with bradycardia was included in the American College of Cardiology (ACC)/American Heart Association (AHA)/Heart Rhythm Society (HRS) guidelines published in 2019, and these form the basis of the ERC bradycardia algorithm (Fig. 9. Bradycardia algorithm).[613] If bradycardia is accompanied by adverse haemodynamic signs, atropine remains the first-choice drug.[215] Conversely, atropine is ineffective and may worsen the block in patients with high-degree atrioventricular block and wide QRS complexes.[613] When atropine is ineffective, second-line drugs include isoprenaline (5 mcg.min^{-1} starting dose) and adrenaline (2 to 10 μg.min^{-1}). For bradycardia in cases of sympathetic denervation, such as heart transplant or spinal cord injury, consider giving aminophylline (100–200 mg, slowly IV).[613] Atropine can cause a high-degree AV block or even sinus arrest in heart transplant patients.[629]

Consider giving IV glucagon (one or more initial IV 3 to 10 mg push doses over 1–2 min. If effective, start a continuous infusion at 2–5 mg.h^{-1}) if beta-blockers or calcium channel blockers are themselves a potential cause of the bradycardia.[630] Consider pacing in unstable patients, with symptomatic bradycardia refractory to drug therapy (see below).

Cardioversion

Electrical cardioversion is the preferred treatment for tachycardia in the unstable patient displaying potentially life-threatening adverse haemodynamic signs (Fig. 8. ERC Peri-arrest Tachyarrhythmia algorithm).[612,614,616] The shock must be synchronised to occur with the R wave of the ECG: VF can be induced if a shock is delivered during the T wave which is a relative refractory portion of the cardiac cycle.[631] Synchronisation can be difficult in VT because of the wide-complex and variable forms of ventricular arrhythmia. Inspect the synchronisation marker carefully for consistent recognition of the R wave. If needed, choose another lead and/or adjust the amplitude. If synchronisation fails, give unsynchronised shocks to the unstable patient in VT to avoid prolonged delay in restoring sinus rhythm. Ventricular fibrillation (VF) or pulseless VT (pVT) require unsynchronised shocks. Conscious patients require anaesthesia or sedation before attempting synchronised cardioversion.

Cardioversion for atrial fibrillation. There is no single optimal position for external defibrillation electrodes. A meta-analysis of 10 RCTs comparing antero-posterior with antero-lateral electrode positioning in AF showed no difference in sinus rhythm restoration.[632] Applying active compression to the anterior defibrillation pad with antero-posterior pads is associated with lower defibrillation thresholds, lower total energy delivery, fewer shocks for successful electric cardioversion, and higher success rates.[302] More data are needed before specific recommendations can be made for optimal biphasic energy levels and different biphasic waveforms. Biphasic rectilinear and biphasic truncated exponential (BTE) waveforms show similar high efficacy in the elective cardioversion of atrial fibrillation.[633] An RCT showed that maximum fixed energy electrical cardioversion (360 J BTE) was more effective in achieving sinus rhythm one minute after cardioversion than an energy-escalating strategy.[634] There was no increase in adverse events. Starting synchronised shocks using the maximum defibrillator energy rather than an escalating approach is a reasonable strategy based on current data. In stable patients, follow appropriate guidelines on the need for anticoagulation before cardioversion to minimise stroke risk.[625]

Cardioversion for atrial flutter and paroxysmal supraventricular tachycardia. Atrial flutter and paroxysmal supraventricular tachycardia (SVT) generally require less energy than atrial fibrillation for cardioversion.[635] The ERC recommends giving an initial shock of 70–120 J. Give subsequent shocks using stepwise increases in energy, as required.

Cardioversion for ventricular tachycardia with a pulse. The energy required for cardioversion of VT with pulse depends on the morphological characteristics and rate of the arrhythmia. Ventricular tachycardia with a pulse responds well to initial shocks with energy levels of 120–150 J. Consider stepwise increases if the first shock fails to achieve sinus rhythm.[257]

Bradycardias and pacing. The treatment of bradycardia is summarised in Fig. 9. Consider pacing in unstable patients with symptomatic bradycardia refractory to drug therapy. Immediate pacing is indicated especially when the block is at or below the His-Purkinje level. Transvenous pacing should be established as soon as possible.[613,615] Consider transthoracic (transcutaneous) pacing as a bridge to transvenous pacing or when transvenous pacing is not readily available. Whenever a diagnosis of asystole is made start CPR, and when chest compressions are paused for a rhythm check look at the ECG carefully for the presence of P waves because this may respond to cardiac pacing. If there is no immediate electrical and mechanical capture with pacing restart CPR. The use of epicardial wires to pace the myocardium following cardiac surgery is effective. Do not attempt pacing for asystole unless P waves are present; it does not increase short or long-term survival in-hospital or out-of-hospital.[636,637] Transcutaneous pacing is a short term bridging measure and may not always be successful.[638] Monitor patients closely, and seek expert help early and arrange transvenous pacing for those patients who do not respond to medication, require transcutaneous pacing, or have an ongoing risk of asystole (Fig. 9).

For haemodynamically unstable, conscious patients with bradyarrhythmia, for whom transthoracic pacing is not readily available, percussion pacing may be attempted as a bridge to electrical pacing, although its effectiveness has not been established.[94,152,639] Give serial rhythmic blows with the closed fist over the left lower edge of the sternum to pace the heart at a physiological rate of 50–70 min^{-1}. Transthoracic and percussion pacing can cause discomfort, so consider giving analgesic or sedative drugs to conscious patients.

Uncontrolled organ donation after circulatory death

Less than half of patients achieve ROSC after resuscitation from cardiac arrest.[47,640] When standard ALS fails to achieve ROSC, there are three broad treatment strategies:[641]

- Stop resuscitation and declare death.
- In selected patients, consider ECPR.
- In settings with an uncontrolled organ donation after circulatory death (uDCD) programme, continue CPR to preserve organ perfusion and transport the patient to a hospital with a designated uDCD pathway, following local protocols and legal requirements.

This ERC Guidelines 2025 ALS focuses on uDCDs. These are defined as donors after unsuccessful resuscitation from unwitnessed (Maastricht category I) or witnessed (Maastricht category II) cardiac arrest, either in-hospital or out-of-hospital.[642] The ERC Guidelines 2025 on Post Resuscitation Care include guidance for organ donation pathways following brain death (donation after brain death: DBD) or controlled donation after circulatory death (cDCD, Maastricht category III donors) in patients who achieve ROSC or are treated with ECPR.[147,643] We acknowledge the ethical, cultural, and legislative issues that lead to variations in the use of uDCD.

There is a mismatch between the availability of organs and demand across the world. Uncontrolled donation after circulatory death (uDCD) provides an opportunity for cardiac arrest victims in whom ROSC cannot be achieved to become organ donors. In Europe, uDCD is currently undertaken in Austria, Belgium, Israel, Italy, Lithuania, Portugal, Russia, and Spain.[642,644] Organs that can be recovered include kidneys, liver, pancreas and lungs. In 2025, an ILCOR systematic review showed that the rates of early dysfunction (primary non-function or delayed graft function) of kidneys and long-term function of livers recovered from uDCD were higher than in DBD or cDCD.[1,645] However, in most studies, long-term graft function was comparable.[644,646–649] This difference may be partly due to the longer warm ischaemia time of uDCD compared with other organ recovery approaches.

There is no universal consensus on selection criteria for uDCD, and identifying a potential donor currently follows regional/national protocols. Criteria generally include age above those of consent (variable by nation, but usually above18 years) and not over 55–65 years, a no-flow time (the interval from emergency call or witnessed arrest to CPR start) within 15 min, and a total warm ischaemia time (the interval between cardiac arrest and the start of organ preservation) not longer than 150 min. Exclusion criteria generally include trauma, homicide, or suicide as a cause of arrest and comorbidities such as cancer, sepsis, and, according to local programmes and the targeted organ to transplant, kidney and liver disease.[650] In a 2024 study conducted on the Parisian registry, of 19,976 adult patients who were resuscitated from 2011 to 2020, 12,890 (65 %) had no ROSC, 9461 (47 %) met termination of resuscitation (TOR) criteria, and 6720 (52 %) could be considered for uDCD kidney donors.[640]

Uncontrolled donation after circulatory death is a time-critical, resource-intensive, complex and ethically challenging process.[651] Following the termination of resuscitation efforts, a 'no-touch' period is observed to rule out the possibility of autoresuscitation, i.e., ROSC after CPR has been stopped or life-sustaining measures have been withdrawn in the intensive care unit. In most countries where uDCD is practised, the prescribed duration of the no-touch period is five minutes,[652] but some require 20 min.[653] An updated systematic review on autoresuscitation identified seven observational studies, of which one investigated OHCA.[654] Among 840 patients whose resuscitation measures were terminated on site, the study reported five cases of ROSC occurring 3 to 8 min after cessation of CPR. Three of these five patients died on scene, while two died in hospital, one within 1.5 h and the other within 26 h.

After the no-touch period, organ preservation procedures are started and continued until consent for organ recovery is ascertained.[655–657] Obtaining consent from a surrogate decision maker (e.g., a family member) is particularly challenging for uDCD, given the unexpected nature of the arrest, the considerable time pressure, and the difficult environment of an emergency department. Establishing clear local protocols and legislative and societal acceptance is crucial for this process.[658] Previous consent registered on a donor card or a public registry is invaluable and must be rapidly retrieved. Adopting an opt-out system with implied consent to donation is an effective strategy to improve the rates of organ donation if the legal and cultural context allows.[659] The ERC Guidelines 2025 Ethics in Resuscitation includes further details on these issues.[4]

For abdominal organs, organ preservation typically uses extracorporeal circulation with membrane oxygenation via a femoro-femoral bypass.[658] Catheters with balloons are used to limit circulation to the abdominal cavity.[660]

Disclosures

Declarations of competing interests for all ERC Guidelines authors are displayed in a COI table which can be found online at https://doi.org/10.1016/j.resuscitation.2025.110769.

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