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European Resuscitation Council Guidelines 2021: Adult advanced life support

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Abstract

These European Resuscitation Council Advanced Life Support guidelines, are based on the 2020 International Consensus on Cardiopulmonary Resuscitation Science with Treatment Recommendations. This section provides guidelines on the prevention of and ALS treatments for both in-hospital cardiac arrest and out-of-hospital cardiac arrest.

Introduction

Adult advanced life support (ALS) includes the advanced interventions that follow basic life support (BLS) and use of an automated external defibrillator (AED). Basic life support continues during and overlaps with ALS interventions.

This ALS section includes the prevention and treatment of both in-hospital cardiac arrest (IHCA) and out-of-hospital cardiac arrest cardiac arrest (OHCA), the ALS algorithm, manual defibrillation, airway management during cardiopulmonary resuscitation (CPR), drugs and their delivery during CPR, and the treatment of peri-arrest arrhythmias.

These Guidelines are based on the International Liaison Committee on Resuscitation (ILCOR) 2020 Consensus on Science and Treatment Recommendations (CoSTR) for ALS.[1] For these ERC

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Guidelines the ILCOR recommendations were supplemented by focused literature reviews undertaken by the ERC ALS Writing Group for those topics not reviewed in the 2020 ILCOR CoSTR. When required, the guidelines were informed by the expert consensus of the writing group membership.

The ERC has also produced guidance on cardiac arrest for patients with coronavirus disease 2019 (COVID-19),[2] which is based on an ILCOR CoSTR and systematic review.[3,4] Our understanding of the optimal treatment of patients with COVID-19 and the risk of virus transmission and infection of rescuers is poorly understood and evolving. Please check ERC and national guidelines for the latest guidance and local policies for both treatment and rescuer precautions.

Guidelines were drafted and agreed by the ALS Writing Group members before posting for public comment between 21 October and 5 November 2020. Twenty-five individuals from 11 countries made 109 comments. Review of these comments led to 46 changes. The Guideline was presented to and approved by the ERC General Assembly on 10th December 2020. The methodology used for guideline development is presented in the Executive summary.[4a]

Summary of key changes

- There are no major changes in the 2020 Adult ALS Guidelines.
- There is a greater recognition that patients with both in- and out-of-hospital cardiac arrest have premonitory signs, and that many of these arrests may be preventable.
- High quality chest compressions with minimal interruption and early defibrillation remain priorities.
- During CPR, start with basic airway techniques and progress stepwise according to the skills of the rescuer until effective ventilation is achieved. If an advanced airway is required, rescuers with a high tracheal intubation success rate should use tracheal intubation. The expert consensus is that a high success rate is over 95% within two attempts at intubation.
- When adrenaline is used it should be used as soon as possible when the cardiac arrest rhythm is non-shockable cardiac arrest, and after 3 defibrillation attempts for a shockable cardiac arrest rhythm.
- The guideline recognises the increasing role of point-of-care ultrasound (POCUS) in peri-arrest care for diagnosis, but emphasise that it requires a skilled operator, and the need to minimise interruptions during chest compression.
- The guideline reflects the increasing evidence for extracorporeal CPR (eCPR) as a rescue therapy for selected patients with cardiac arrest when conventional ALS measures are failing or to facilitate specific interventions (e.g. coronary angiography and percutaneous coronary intervention (PCI), pulmonary thrombectomy for massive pulmonary embolism, rewarming after hypothermic cardiac arrest) in settings in which it can be implemented.
- These ERC guidelines have followed European and international guidelines for the treatment of peri-arrest arrhythmias.

Key messages from this section are presented in Fig. 1.

Concise guidelines for clinical practice

Prevention of in-hospital cardiac arrest

- The ERC supports 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. These plans should be recorded in a consistent manner (See Ethics section).
- Hospitals should 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 should train staff in the recognition, monitoring and immediate care of the acutely-ill patient.
- Hospitals should 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 should 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 should use structured communication tools to ensure effective handover of information.
- Patients should 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.

Prevention of out-of-hospital cardiac arrest

- Symptoms such as syncope (especially during exercise, while sitting or supine), palpitations, dizziness and sudden shortness of breath that are consistent with an arrhythmia should be investigated.
- Apparently healthy young adults who suffer sudden cardiac death (SCD) can also have 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 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.

Treatment of in-hospital cardiac arrest

- Hospital systems should aim to recognise cardiac arrest, start CPR immediately, and defibrillate rapidly (<3 min) when appropriate.
- All hospital staff should be able to rapidly recognise cardiac arrest, call for help, start CPR and defibrillate (attach an AED and follow the AED prompts, or use a manual defibrillator).
- European hospitals should adopt a standard “Cardiac Arrest Call” telephone number (2222).
- Hospitals should have a resuscitation team that immediately responds to IHCAs.

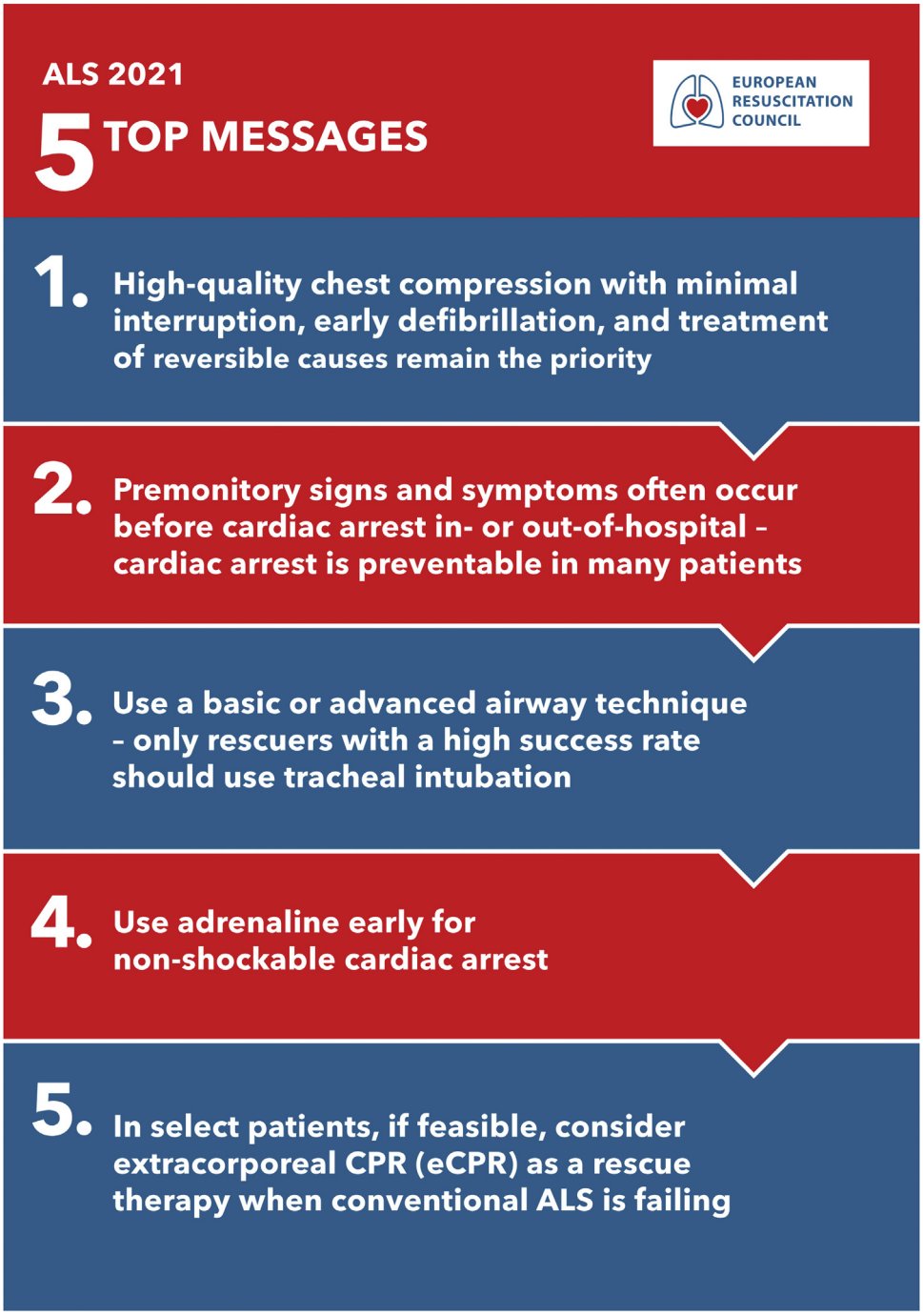

Fig. 1 – ALS summary

- The hospital resuscitation team should include team members who have completed an accredited adult ALS course.
- 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.
- Start ALS as early as possible.
- Emergency medical systems (EMS) should consider implementing criteria for the withholding and termination of resuscitation (TOR) taking in to consideration specific local legal, organizational and cultural context (see Ethics section)
- Systems should define criteria for the withholding and termination of CPR, and ensure criteria are validated locally (see the Ethics section).
- Emergency medical systems (EMS) should monitor staff exposure to resuscitation and low exposure should be addressed to increase EMS team experience in resuscitation.

- Adult patients with non-traumatic OHCA should be considered for transport to a cardiac arrest centre according to local protocols (see Systems saving lives)

Manual defibrillation

Defibrillation strategy

- Continue CPR while a defibrillator is retrieved and pads applied.
- Give a shock as early as possible when appropriate.
- Deliver shocks with minimal interruption to chest compression, and minimise the pre-shock and post-shock pause. This is achieved by continuing chest compressions during defibrillator charging, delivering defibrillation with an interruption in chest compressions of less than 5 s and then immediately resuming chest compressions.
- 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 waking, purposeful movement, arterial waveform or a sharp rise in end-tidal carbon dioxide ($ETCO_2$), consider stopping chest compressions for rhythm analysis, and if appropriate a pulse check.

Safe and effective defibrillation

- Minimise the risk of fire by taking off any oxygen mask or nasal cannulae and place them at least 1 m away from the patient's chest. Ventilator circuits should remain attached.
- Antero-lateral pad position is the position of choice for initial pad placement. Ensure that the apical (lateral) pad is positioned correctly (mid-axillary line, level with the V6 pad position) i.e. below the armpit.
- In patients with an implantable device, place the pad $>$ 8 cm away from the device, or use an alternative pad position. Also consider an alternate pad position when the patient is in the prone position (bi-axillary), or in a refractory shockable rhythm (see below).
- A shock can be safely delivered without interrupting mechanical chest compression.
- During manual chest compressions, 'hands-on' defibrillation, even when wearing clinical gloves, is a risk to the rescuer.

Energy levels and number of shocks

- Use single shocks where indicated, followed by a 2 min 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.
- Defibrillation shock energy levels are unchanged from the 2015 guidelines:
  - For biphasic waveforms (rectilinear biphasic or biphasic truncated exponential), deliver the first shock with an energy of at least 150 J.
  - For pulsed biphasic waveforms, deliver the first shock at 120–150 J.
- If the rescuer is unaware of the recommended energy settings of the defibrillator, for an adult use the highest energy setting for all shocks.

Recurrent or refractory VF

- Consider escalating the shock energy, after a failed shock and for patients where refibrillation occurs.
- For refractory VF, consider using an alternative defibrillation pad position (e.g. anterior- posterior)
- Do not use dual (double) sequential defibrillation for refractory VF outside of a research setting.

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.
- If an advanced airway is required, rescuers with a high tracheal intubation success rate should use tracheal intubation. The expert consensus is that a high success rate is over 95% within two attempts at intubation.
- Aim for less than a 5 s interruption in chest compression for tracheal intubation.
- Use direct or video laryngoscopy for tracheal intubation according to local protocols and rescuer experience
- Use waveform capnography to confirm tracheal tube position.
- Give the highest feasible inspired oxygen during CPR.
- Give each breath over 1 s to achieve a visible chest rise.
- Once a tracheal tube or a supraglottic airway (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.

Drugs and fluids

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

Vasopressor drugs

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

Antiarrhythmic drugs

- Give amiodarone 300 mg IV(IO) 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 (IO) for adult patients in cardiac arrest who are in VF/pVT after five shocks have been administered.
- Lidocaine 100 mg IV (IO) 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 thrombolytic drug therapy when pulmonary embolus is the suspected or confirmed cause of cardiac arrest.
- Consider CPR for 60–90 min after administration of thrombolytic drugs.

Fluids

- Give IV (IO) fluids only where the cardiac arrest is caused by or possibly caused by hypovolaemia.

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.
- Although high and increasing $ETCO_2$ values are associated with increased rates of ROSC and survival after CPR, 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 be useful to diagnose treatable causes of cardiac arrest such as cardiac tamponade and pneumothorax.
- Right ventricular dilation in isolation during cardiac arrest should not be used to diagnose massive pulmonary embolism.
- Do not use POCUS for assessing contractility of the myocardium as a sole indicator for terminating CPR.

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 use by using only trained teams familiar with the device.

Extracorporeal CPR

- Consider extracorporeal CPR (eCPR) as a rescue therapy for selected patients with cardiac arrest when conventional ALS measures are failing or to facilitate specific interventions (e.g. coronary angiography and percutaneous coronary intervention (PCI), pulmonary thrombectomy for massive pulmonary embolism, rewarming after hypothermic cardiac arrest) in settings in which it can be implemented.

Peri-arrest arrhythmias

- The assessment and treatment of all arrhythmias addresses the condition of the patient (stable versus unstable) and the nature of the arrhythmia. Life-threatening features in an unstable patient include:
  - Shock – appreciated as hypotension (e.g. systolic blood pressure < 90 mmHg) and symptoms of increased sympathetic activity and reduced cerebral blood flow.
  - Syncope – as a consequence of reduced cerebral blood flow.
  - Severe 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).

Tachycardias

- Electrical cardioversion is the preferred treatment for tachyarrhythmia in the unstable patient displaying potentially life-threatening adverse signs.
- Conscious patients require anaesthesia or sedation, before attempting synchronised cardioversion.
- To convert atrial or ventricular tachyarrhythmias, the shock must be synchronised to occur with the R wave of the electrocardiogram (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 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.
- If the patient with tachycardia is stable (no adverse signs or symptoms) and is not deteriorating, pharmacological treatment may be possible.
- Consider amiodarone for acute heart rate control in AF patients with haemodynamic instability and severely reduced left ventricular ejection fraction (LVEF). For 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 caused by inferior myocardial infarction, cardiac transplant or spinal cord injury, consider giving aminophylline (100–200 mg slow intravenous injection).
- Consider giving glucagon if beta-blockers or calcium channel blockers are a potential cause of the bradycardia.
- Do not give atropine to patients with cardiac transplants – it can cause a high-degree AV block or even sinus arrest – use aminophylline.
- Consider pacing in patients who are unstable, with symptomatic bradycardia refractory to drug therapies.
- If transthoracic pacing is ineffective, consider transvenous pacing.

- 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 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.

Debriefing

- Use data-driven, performance-focused debriefing of rescuers to improve CPR quality and patient outcomes.

Evidence informing the guidelines

Prevention of in-hospital cardiac arrest (IHCA)

In-hospital cardiac arrest (IHCA) occurs in about 1.5 patients per 1000 admitted to hospital.[5,6] There are two main strategies to prevent cardiac arrest and the need for attempted CPR:

- Patient-focussed 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.[7–10] The ERC Ethics guidelines 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 Ethics section.

Physiological deterioration

In-hospital cardiac arrest is often preceded by physiological deterioration.[11,12] This provides an opportunity to recognise deterioration and prevent the 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'.[13] 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.[14–16]

Staff education

Education should include measurement of vital signs, 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.[15] Staff should also know how to implement local policies about do-not-attempt CPR (DNACPR) decisions, treatment escalation plans, and starting end-of-life care.

Monitoring

Most cases of IHCA have an initial non-shockable rhythm and preceding signs of respiratory depression or shock are common.[5,6,17] 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. This can be addressed by using a standardised early warning score (EWS) system for all patients. The choice of system depends on local circumstances and should align with national guidelines. For example in the UK the National Early Warning Score 2 (NEWS2) is endorsed by the National Institute for Health and Care Excellence (NICE) guidelines.[14,15] Higher trained nurse staffing levels are associated with lower rates of failure-to-respond to abnormal vital signs, and the quality of patient care.[18,19] There is a lack of randomised controlled trials (RCTs) or consensus on which patients should undergo continuous ECG monitoring. In a registry-based study, settings where patients are closely monitored are associated with improved survival irrespective of initial rhythm.[20]

Recognition

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 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.[21] Clinical concern from nurses and other members of the multidisciplinary team can also indicate patient deterioration.[22,23]

The call for help

All staff should be empowered to call for help and also trained to use structured communication tools such as SBAR (situation-background-assessment-recommendation) to ensure effective communication.[24–26] The response to patients who are critically ill or who are at risk of becoming critically ill is often provided by a medical emergency team (MET), rapid response team (RRT), or critical care outreach team (CCOT). Any member of the health-care team can initiate a MET/RRT/CCOT call. In some hospitals, the patient, and their family and friends, are also encouraged to activate the team.[27–29]

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. Systematic reviews, meta-analyses and multicentre studies suggest that RRT/MET/CCOT systems reduce the rate of IHCA and hospital mortality.[30,31] These data led ILCOR to suggest that hospitals consider the introduction of rapid response systems (rapid response team/medical emergency team) to reduce the incidence of IHCA and in-hospital mortality (weak recommendation, low-quality evidence).[16] Team interventions often involve simple tasks such as starting oxygen therapy and intravenous 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 Ethics section). 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.

Prevention of out-of-hospital cardiac arrest (OHCA)

In industrialised countries, sudden cardiac death (SCD) is the third leading cause of death. Survival following out-of-hospital cardiac arrest (OHCA) is only 10% or less,[32–34] which makes prevention of OHCA important.[35] Apparently healthy young adults who sustain SCD can also have 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.[36–45]

There is no systematic review on this topic. A search on 26 February 2020 using the terms "out-of-hospital cardiac arrest" AND "prevention" limited to clinical trials and reviews since 1 January 2015 identified 65 articles. The references of these articles were also reviewed. Existing guidelines of the European Society of Cardiology (ESC), the American Heart Association (AHA) and European Resuscitation Council (ERC) were considered.

Epidemiology and pathophysiology of sudden cardiac death

Coronary heart disease (CHD) accounts for 80% of SCD, especially in older patients, and non-ischaemic cardiomyopathies account for another 10–15%.[46] In the young, inherited diseases, congenital heart disease, myocarditis and substance abuse are predominant causes. Knowledge of the causes of SCD will assist in early treatment and the prevention of OHCA (Table 1).

Table 1 – Causes of sudden cardiac arrest (SCD).
Adapted from Kandala[46] and Winkel.[47].

Coronary heart disease
ST-segment elevation
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 (ABiMVPS))
Drug or medication induced
Non-atherosclerotic coronary artery anomalies
Congenital heart disease
Hypertrophic cardiomyopathy (HCM)
Dilated cardiomyopathy (DCM)
Valvular heart disease

Coronary heart disease (CHD)

Arrhythmias triggered by acute myocardial infarction (AMI) or subsequent myocardial scarring can result in SCD.[48] About two-thirds of SCDs occur as the first CHD event or in individuals considered to be at low risk.[46] During the last 50 years primary prevention and secondary revascularisation have reduced CHD age-adjusted mortality.[46] 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.[46] Cardiac electrophysiology studies can identify patients with CHD at high versus low risk of SCD.[49] Additional factors such as heart failure (HF) and left ventricular hypertrophy (LVH) predispose to ventricular arrhythmias (polymorphic ventricular tachycardia [VT] and VF]). How to identify patients at high risk of SCD with HF and LVH is uncertain.[50] Changes in left ventricular geometry affect the likelihood of developing VT and VF. High blood levels of B-type natriuretic peptide (BNP) and its N-terminal fragment (NT-proBNP) are associated with higher rates of appropriate implantable cardioverter defibrillator (ICD) placement and mortality.[51,52] 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).[48] LVEF is used to indicate the need for an implantable cardioverter defibrillator (ICD) for the primary and secondary prevention of SCD.[53] Despite considerable progress, the ability to recognise the risk of SCD before the event remains very limited.[48]

SCD in the young

SCD in the young (SCDY, 5–35 years of age) accounts for 7% of all SCDs;[47] the incidence is 1–8/100 000 fatalities per year.[54] In adolescent SCD, 50% of patients had misinterpreted symptoms before death.[44] CHD is the most frequent cause of explained SCDY; 25–31% of the cases remain unexplained after post mortem examination (Sudden Arrhythmic Death Syndrome- SADS).[47] The majority of inherited cardiac diseases can be treated if diagnosed, yet most young SCD victims are not diagnosed.[42] Premonitory signs of SCDY were present in only 29% in one study, and thus lower than in older patients.[55] QT-prolonging and psychotropic drugs, alone or in combination, increase the risk of SCD.[56] 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. This screening resulted in a diagnosis of an inherited cardiac disease in over half of the families.[57] In a large retrospective SCDY study, a cause was identified in 113/180 patients (62.8%), the rest were classified as idiopathic VF.[58] With improvements in diagnosis (e.g. provocation drug testing for cardiac channelopathies and coronary vasospasm, genetical testing), the number of unexplained SCDs should decrease.[58] (See the Epidemiology section).[35]

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.[46,55]

Hypertrophic cardiomyopathy (HCM)

Hypertrophic cardiomyopathy is the most common genetic disorder of the heart, with 1 in 200–500 cases, and it is the most frequent cause of SCDY.[59] It often remains clinically silent until SCD presents as the first cardiac event. The incidence of SCD in families with HCM may be 2-4% a year and 4-6% in children and adolescents.[46]

Premonitory signs

Approximately 50% of cardiac arrests occur in individuals with undiagnosed CHD. [48,60] 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.[61,62] 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.[62,63] In 1960 OHCA patients, 9.4% had been assessed by an ambulance crew within the preceding 48 h.[64] Emergency care in patients with symptoms is associated with improved survival.[61] Early recognition of acute coronary syndrome (ACS) by emergency medical system (EMS) teams with 12-lead ECG capabilities and reduction of time to reperfusion may prevent SCD.[65] 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.[65] 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.[65] 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.[53,59,66–76] 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 2).[53] Early EMS acquisition of a 12 lead-ECG may be helpful.

Screening programs for athletes may be helpful but vary between countries.[88,89] 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).[90]

Preventive measures against SCD

Prevention of SCD is focused on the associated medical conditions that may contribute to or exacerbate arrhythmia, the risk posed by arrhythmia and the risk-benefit of a given therapy. Interventions include anti-arrhythmic drugs, implantable cardioverter defibrillators (ICD), and ablation or surgery.[53,91] Noninvasive telemetry or implantable devices transmitting the ECG are currently used in selected group of patients to detect high risk arrhythmias and prevent SCD. More recently, connected devices with arrhythmia detection capabilities (smartwatch, smartphone applications) have been

Table 2 – High risk features suggesting a serious condition in patients with syncope at initial evaluation in the emergency department. Adapted from Brignole 2018.[53] ECG electrocardiogram; ICD implantable cardioverter defibrillator; LVEF left ventricular ejection fraction; SCD sudden cardiac death; VT ventricular tachycardia.

Syncopal event features
Major
New onset of chest discomfort, breathlessness, abdominal pain or headache[77–79]
Syncope during exertion or when supine[80]
Sudden onset palpitation immediately followed by syncope[80]
Minor
No warning symptoms or short (<10 s) prodrome [80–83]
Family history of SCD at young age[84]
Syncope in the sitting position[85]
Past medical history
Major
Severe structural or coronary artery disease (heart failure, low LVEF or previous myocardial infarction)[77,79]
Physical examination
Major
Unexplained systolic blood pressure <90 mmHg[77,79]
Persistent bradycardia (<40 min^{-1}) in 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 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[78,83]
Sustained and non-sustained VT
Dysfunction of an implantable cardiac device (pacemaker or ICD)
Type 1 Brugada pattern
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)[86]
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.)[83]
Paroxysmal supraventricular (SVT) or atrial fibrillation[87]
Pre-excited QRS complex
Short QTc interval (< = 340 ms)[86]
Atypical Brugada patterns[86]
Negative T waves in right precordial leads, epsilon waves suggestive of arrhythmogenic right ventricular cardiomyopathy (ARVC)[86]

introduced and may be helpful in detecting asymptomatic AF, however their potential role in the general population to detect SCD arrhythmias is unknown.[92,93] Public education to report on symptoms before SCD and to help a persons in cardiac arrest are important.[61]

Treatment of in-hospital cardiac arrest (IHCA)

Cardiac arrest treatment principles, such as rapid defibrillation 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 can 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 (see Fig. 2). These guidelines are based on the ILCOR CoSTR,[1] the 2015 ERC ALS Guidelines[21] and ERC Quality Standards for CPR Practice and Training.[94]

ILCOR undertook a systematic review of accredited training in adult ALS. The review included eight observational studies and identified benefits of ALS for ROSC and survival to hospital discharge or 30-days.[16] ILCOR also undertook a systematic review on team and leadership training including sixteen RCTs and three observational studies identifying a benefit for patient survival as well as skill performance.[16]

First responders

The clinical skill of a first 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 first 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.[95,96]

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.[97]

Following the completion of initial actions and provided sufficient staff are available, staff should collect ALS equipment and prepare to handover to the resuscitation team using either the SBAR (Situation, Background, Assessment, Recommendation) or RSVP (Reason, Story, Vital Signs, Plan) systems.[24,98,99] Each clinical area in a hospital should consider patient acuity, risk of cardiac arrest, and geographical location (i.e. 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 MET/RRT (medical emergency team/ rapid response team) that responds to both cardiac arrests and critically unwell patients. The ILCOR recommends accredited ALS level training for healthcare staff (weak recommendation based on very low certainty evidence) as ALS training is associated with increased ROSC and patient survival.[16] ILCOR also recommends team and leadership training (weak recommendation based on very low certainty evidence) because it is associated with improved patient and process-outcomes.[16] Resuscitation teams often form on an ad hoc basis depending on hospital work rosters and include individuals from a range of specialities (e.g. acute medicine, cardiology, critical care). Lack of knowledge of team member roles, including who is acting as team leader can lead to errors during CPR for IHCA.[100,101] A team meeting at the beginning of each shift for introductions and allocation of roles may support effective team-working during resuscitation.

Equipment

Hospitals should ensure that clinical areas should have immediate access to resuscitation equipment and drugs to facilitate rapid resuscitation of the patient in cardiac arrest. Missing or malfunctioning equipment contributes to treatment delays.[100,102] Equipment should be standardised throughout the hospital and equipment checked regularly.

Treatment of out-of-hospital cardiac arrest

This section provides an overview of specific ALS issues related to CPR for OHCA. Further information is available in the sections Basic life support (BLS), Cardiac Arrest in Special Circumstances, Systems of Care, Epidemiology, Post-resuscitation care, and Ethics. 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.

Three ILCOR systematic reviews were identified.[103,103a108] A focused search on 13 March limited to clinical trials and reviews since 1 January 2015 identified 612 articles. The titles and abstracts were screened and pertaining articles included.

Initial ALS treatment of OHCA

Several patient and CPR factors affect outcome from OHCA (Table 3). Community programs of lay bystander CPR and AED use improve outcome from OHCA.[104] Chest compressions and early defibrillation are the cornerstones of CPR in OHCA. The only definitive treatment for VF remains prompt defibrillation.[105]

EMS personnel and interventions

ILCOR performed a systematic review of EMS exposure to and experience of OHCA on outcome.[103] The largest study in this review linked exposure of paramedics to OHCA, defined as the number of times a paramedic had attended an OHCA, to patient survival to hospital discharge.[106] Increasing exposure in the preceding three years was associated with increased survival to discharge: $\leq$ 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).[106] Another large observational study reported that increased exposure of the treating paramedic was associated with increased ROSC (<15 exposures, control group vs. $\geq$ 15 exposures (aOR 1.22, 95%CI 1.11−1.36).[107] 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).

Termination of CPR rules

Termination of resuscitation (TOR) rules are used by many EMS. An ILCOR systematic review on the use of TOR rules found that implementing the currently studied TOR rules would result in some missed survivors.[103a] ILCOR recommended the use of TOR rules to

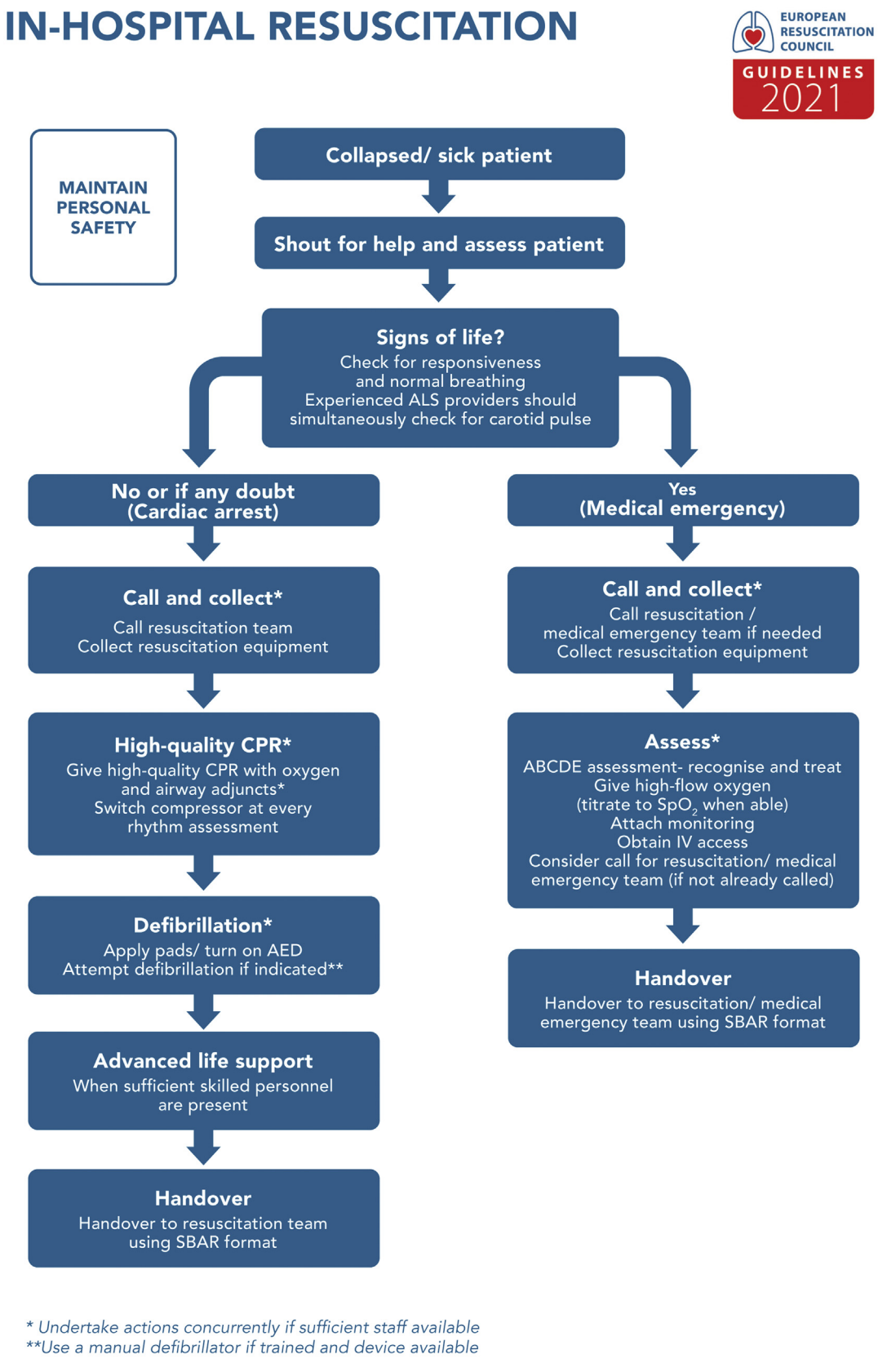

Fig. 2 – In-hospital resuscitation algorithm. AED automated external defibrillator; ALS advanced life support; CPR cardiopulmonary resuscitation; SBAR situation, background, assessment, recommendation.

Table 3 – Patient and CPR factors affecting outcome from OHCA. Adapted from Kandala 2017.[46] AED automated external defibrillation; CPR cardiopulmonary resuscitation.

Patient
Age
Sex
Comorbidities
Cardiac function
Pulmonary function
Renal function
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

assist clinicians in deciding whether to discontinue resuscitation efforts at the scene or to transport the patient to the hospital with ongoing CPR (weak recommendation, very-low certainty evidence). Decisions to terminate resuscitation should also take into account the local legal, organizational, and cultural context. EMS personnel working in systems where TOR by non-physicians is not legal or culturally acceptable should transport patients with ongoing CPR to hospital. The Ethics section provides more specific guidance on the use of termination of resuscitation rules.

Care at cardiac arrest centres

An ILCOR systematic review assessed the benefits of care at a dedicated cardiac arrest centre (CAC).[16,108] The resulting ILCOR treatment recommendations included:

- We suggest adult non-traumatic OHCA cardiac arrest patients be cared for in cardiac arrest centres rather than in non-cardiac arrest centres (weak recommendation, very low certainty of evidence).
- We cannot make a recommendation for or against regional triage of OHCA patients to a cardiac arrest centre by primary EMS transport (bypass protocols) or secondary interfacility transfer.

For further information about cardiac arrest centres see sections on Systems Saving Lives and Post Resuscitation Care.

ALS treatment algorithm

Cardiac arrest is associated with either shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT)) or non-shockable rhythms (asystole and pulseless electrical activity (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 ventilation, venous access, administration of adrenaline and the identification and treatment of reversible causes, are common for all arrests. The ALS algorithm (Fig. 3) provides an overview of these key interventions. These are based on the expert consensus of the writing group. The ALS cardiac arrest algorithm is applicable to all cardiac arrests. Additional interventions may be indicated for cardiac arrest caused by special circumstances.

Manual defibrillation

Defibrillation is a vital component of CPR as it has the potential to terminate VF/pVT and achieve ROSC. Defibrillation is indicated in approximately 20% of cardiac arrests. As its effectiveness decreases with time and VF duration, defibrillation attempts must be timely, whilst remaining efficient and safe. Knowledge of how to use a defibrillator (manual or AED) is key for rescuers performing advanced life support. 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 in order to minimise interruption to chest compressions.

Since 2015, ERC defibrillation guidelines have referred solely to biphasic energy waveforms and in these 2020 guidelines we refer only to the use of defibrillation pads (rather than paddles).[21]

The evidence for this section is based on ILCOR 2020 CoSTRs, the ERC 2015 ALS Guidelines, and expert consensus.[1,21,104]

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 successful.[109–114] 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.[115,116] 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.[1] If there are clinical and physiological signs of ROSC (e.g. 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 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.[104] Therefore, the routine delivery of a pre-specified period of CPR (e.g. 2–3 min) before rhythm analysis and a shock is delivered is not recommended. Rescuers should provide a short period of CPR until the defibrillator is ready for rhythm analysis in unmonitored cardiac arrest (weak recommendation, low-certainty evidence). 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 min cycle.

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.[21] We wish to clarify that when the rhythm is clearly judged to be VF a shock should be given.

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. When compressions are paused briefly to check rhythm, a shock can be delivered immediately (if indicated) from a defibrillator that is already charged, avoiding a period of further chest compressions

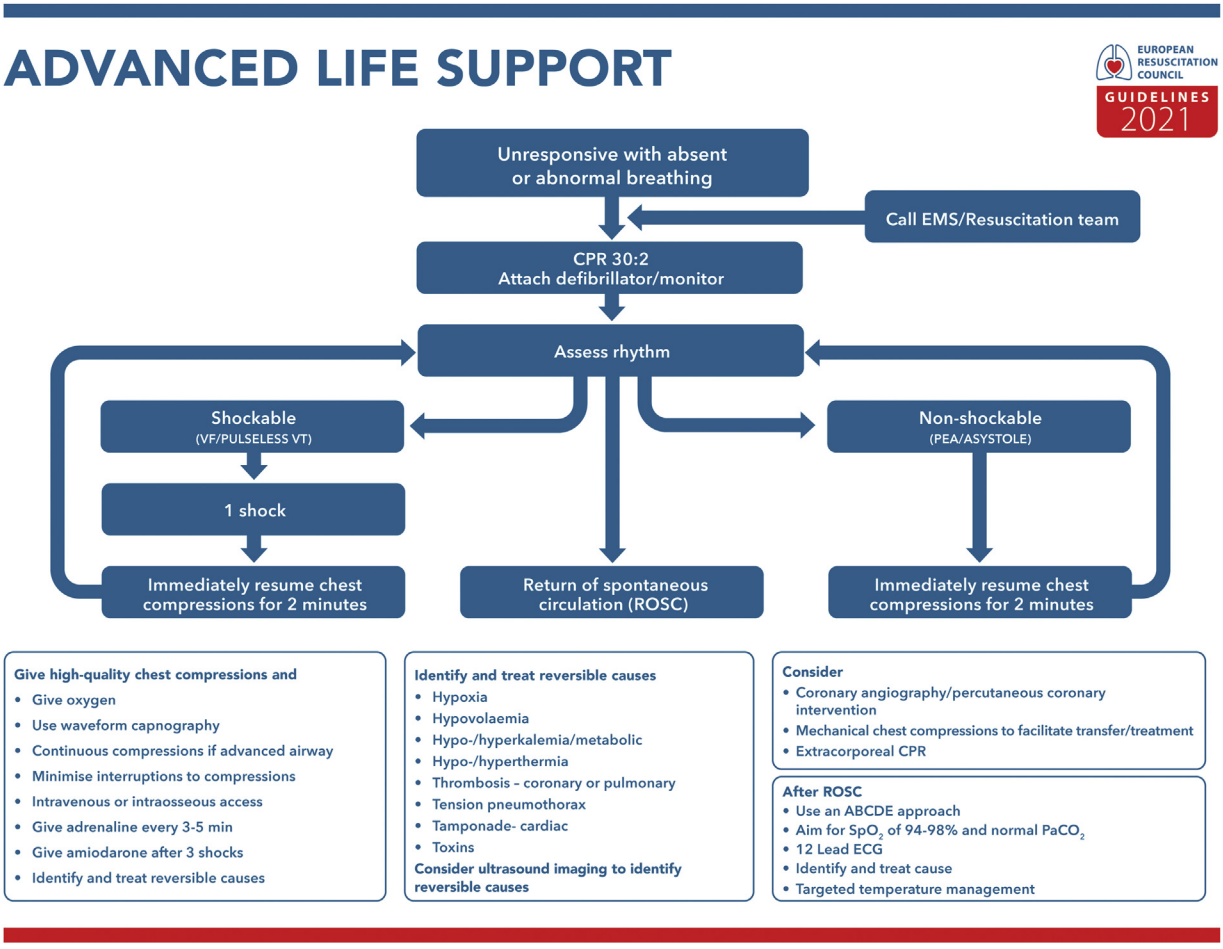

Fig. 3 – Advanced Life Support algorithm. ABCDE airway, breathing, circulation, disability, exposure CPR cardiopulmonary resuscitation; ECG electrocardiogram; EMS emergency medical system; PEA pulseless electrical activity; $PaCO_2$ arterial partial pressure of carbon dioxide; ROSC return of spontaneous circulation; SpO_2 arterial oxygen saturation; VF ventricular fibrillation; VT ventricular tachycardia.

while the defibrillator is charged. This method was reviewed by ILCOR in 2020 as the technique is already in use as an alternative to the conventional sequence.[117] Manikin studies show anticipatory charging is feasible, can reduce the overall interruption to chest compression, but increases pre- post, and peri-shock pause duration. This technique may be a reasonable alternative for use by well-drilled teams that can minimise pre- post, and peri-shock pause duration. 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.[118–123] 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 these guidelines. The risk of fire during attempted defibrillation can be minimised by taking the following precautions:

- Take off any oxygen 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, 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.

Pad contact with the chest and anatomical position

There is no new evidence since the 2015 guidelines regarding optimal defibrillation pad position.[21] 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.[104] Transmyocardial current during defibrillation is likely to be maximal when 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.

Pad placement for ventricular arrhythmias and cardiac arrest

Place pads in the conventional antero-lateral (sternal-apical) position. The right (sternal) pad is placed to the right of the sternum, below the clavicle. The apical pad is placed in the left mid-axillary line, approximately level with the V6 ECG electrode. This position should be clear of any breast tissue.[124] It is important that this pad is placed

sufficiently laterally (Fig. 4) and in practical terms, the pad should be placed just below the armpit.[125] 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 apical position and the other on the right upper back.
- One pad anteriorly, over the left precordium, and the other pad posteriorly to the heart just inferior to the left scapula.

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 impedance was lower when the paddle was orientated in a cranio-caudal direction.[126] 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,[127,128] the majority have failed to show any clear advantage of any specific pad position.[129–132] Efficacy of cardioversion may be less dependent on pad position when using biphasic impedance-compensated waveforms.[131–133] 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 inferior to 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. These devices may be damaged during defibrillation if current is discharged through pads placed directly over the device.[134,135] Place the pad away from the device (at least 8 cm) or use an alternative pad position (anterior-lateral, anterior-posterior).[134,136]

Hands-on defibrillation

By allowing continuous chest compressions during the delivery of the defibrillation shock, hands-on defibrillation can minimise peri-shock pause and allow continuation of chest compressions during defibrillation. 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.[137] Only Class 1 electrical safety gloves, but not standard clinical examination gloves (or bare hands) provide a safe level of electrical insulation for hands-on defibrillation.[138] There have been no new studies since the 2015 guidelines and the recommendation therefore remain unchanged.[21]

Respiratory phase

Positive end expiratory pressure (PEEP) increases transthoracic impedance and should be minimised where possible during

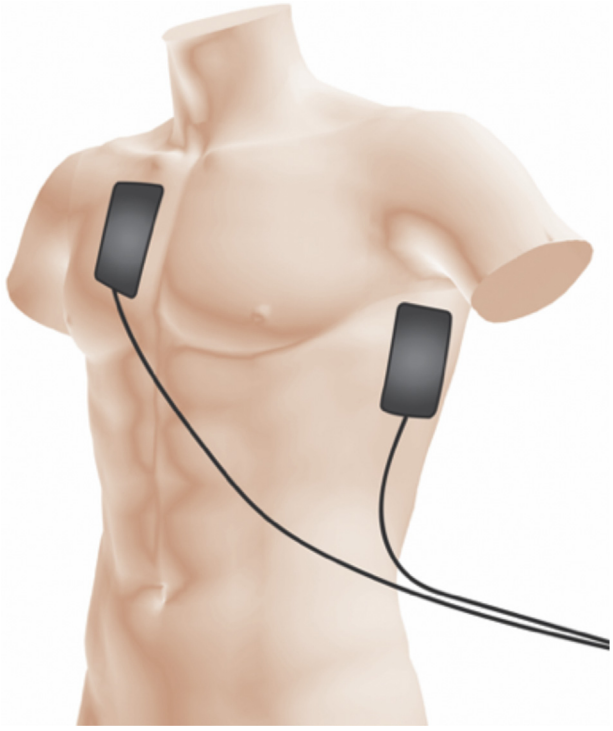

Fig. 4 – Correct pad placement for defibrillation (© Charles Deakin).

defibrillation. Auto-PEEP (gas trapping) may be particularly high in patients with asthma and may necessitate higher than usual energy values for defibrillation.[139]

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.[140,141] 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 end-point.[142,143] 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 in view of the evidence suggesting that outcome is improved by minimising interruptions to chest compressions, we continue in 2020 to recommend single shocks for most situations (see below).

When defibrillation is warranted, give a single shock and resume chest compressions immediately following the shock.[104] 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.[144,145] 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.[146] In patients where defibrillation achieves a perfusing rhythm, the effect of chest compressions on re-inducing VF is not clear.[147]

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, immediately after onset of VF/pVT (expert opinion).

Fibrillation waveform analysis

It is possible to predict, with varying reliability, the success of defibrillation from the fibrillation waveform.[148–170] 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. This technology is under active development and investigation, but current sensitivity and specificity are insufficient to enable introduction of VF waveform analysis into clinical practice. Although one large RCT,[171] and 20 observational studies[172–191] published since the 2010 guidelines review [140,141] have shown promise and some improvements in this technology, there remains insufficient evidence to support routine use of VF waveform analysis to guide the optimal timing for a shock attempt.[1,104]

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.[21]

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.[192] Selection of an appropriate energy level also reduces the number of repetitive shocks, which in turn limits myocardial damage.[193]

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 flow that achieves defibrillation; the electrical current correlates well with successful defibrillation and cardioversion.[194] Defibrillation shock energy levels are unchanged from the 2015 guidelines.[21]

First shock

Relatively few studies have been published with which to refine the current defibrillation energy levels set in the 2010 guidelines.[195] 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 86-98%.[196–200] First shock efficacy of the RLB waveform using 120 J has been reported as 85%.[201] Four studies have suggested equivalence with lower and higher starting energy biphasic defibrillation.[202–205] although one has suggested that initial low energy (150 J) defibrillation is associated with better survival.[206] Although human studies have not shown harm (raised biomarkers, ECG changes, ejection fraction) from any biphasic waveform up to 360 J,[202,207] several animal studies have suggested the potential for harm with higher energy levels.[208–211]

The initial biphasic shock should be no lower than 120 J for RLB waveforms and at least 150 J for BTE waveforms. For pulsed biphasic waveforms, begin at 120–150 J. Ideally, the initial biphasic shock energy should be at least 150 J for all biphasic waveforms in order 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 (expert opinion).

Second and subsequent shocks

The 2010 guidelines recommended either a fixed or escalating energy strategy for defibrillation. Several studies show that although an escalating strategy reduces the number of shocks required to restore an organised rhythm compared with fixed-dose biphasic defibrillation, and may be needed for successful defibrillation,[212,213] rates of ROSC or survival to hospital discharge are not significantly different between strategies.[202–204] Conversely, a biphasic protocol using a fixed energy level showed high cardioversion rates (>90%) but significantly lower ROSC rate for recurrent VF could not be excluded.[214] 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.[215–220]

In 2020, 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.[212] Two studies showed termination rates of subsequent refibrillation were unchanged when using fixed 120 J or 150 J shock protocols respectively,[214,221] 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.[212] 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.[222]

In view of the larger study suggesting benefit from higher subsequent energy levels for refibrillation,[212] we recommend that if a shockable rhythm recurs after successful defibrillation with ROSC, and the defibrillator is capable of delivering shocks of 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 and occurs in approximately 20% of patients who present in VF.[212] Duration of VF correlates negatively with good outcome. Actively search for and correct any reversible causes (Fig. 3 ALS algorithm). Ensure that the defibrillation energy output is on the maximum setting – an escalating protocol may be more effective in treating refractory VF. Check that the defibrillation pads are placed correctly (particularly the apical pad, when using the antero-lateral pad position). Consider using an alternative defibrillation pad orientation (e.g. antero-posterior).

Dual/double sequential defibrillation

Patients in refractory VF have significantly lower rates of survival than patients who respond to standard resuscitation treatments. Double sequential defibrillation 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 anterior-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,[223–230] ILCOR reviewed the efficacy of this technique and based on very low certainty evidence made a weak recommendation against the routine use of a double sequential defibrillation strategy in comparison with standard defibrillation strategy for cardiac arrest with a refractory shockable rhythm.[1,231]

Analysis of rhythm during chest compression

New software technology in some defibrillators enables removal of ECG motion artefact generated during chest compressions in order 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 artifact-filtering algorithms for analysis of electrocardiographic rhythm during CPR.[104] 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 artifact-filtering algorithms for rhythm analysis during CPR, 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.[232] In a recent randomised controlled trial the subcutaneous ICD was non-inferior to the transvenous ICD with respect to device-related complications and inappropriate shocks.[233]

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 disable the defibrillation function in these circumstances.[136]

Discharge of an ICD may cause pectoral muscle contraction in the patient, and shocks to the rescuer have been documented.[234] In view of 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.[235,236] 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.[237] The diagnostic algorithms of modern AEDs can be insensitive to such spikes.

Airway and ventilation

In 2015 the ERC recommended a stepwise approach to airway management during CPR.[21] Three large RCTs of airway management for OHCA have been published since 2015.[238–240] Check the latest ERC guidelines for COVID-19 precautions required during airway management.

An ILCOR systematic review addressed whether a specific advanced airway management strategy improved outcome from cardiac arrest (CA) in comparison with an alternative airway management strategy.[241,242] Seventy-eight observational studies were included; nine of these addressed the question of timing of advanced airway management. Eleven controlled trials were included but only three of these were RCTs.[238–240] The first of these RCTs compared early tracheal intubation (TI) with bag-mask ventilation (TI delayed until after ROSC) in a physician-staffed EMS system.[239] The result of this non-inferiority trial that recruited over 2000 patients was inconclusive (4.3% versus 4.2% for 28-day survival with favourable functional outcome (CPC 1–2), no significant difference). Notably, the TI 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 (LT) insertion with TI in 3000 OHCAs by paramedics in the United States, 72-h survival (primary outcome) was higher in the LT group (18.2% versus 15.3%; $p = 0.04$).[240] However, the overall TI success rate was just 51% making it possible that the lower survival rate in the TI group was a reflection of the poor TI success rate. The third of these RCTs was a comparison of the initial insertion of an i-gel supraglottic airway (SGA) with TI in OHCA treated by paramedics in the United Kingdom (UK).[238] Among the more than 9000 patients enrolled, there was no difference in the primary outcome of favourable functional survival (mRS $\leq$ 3; 6.4% versus 6.8%; $P = 0.33$).

A large observational cohort study of IHCA from the American Heart Association (AHA) Get with the Guidelines-Resuscitation (GWTG-R) registry matched patients intubated at any given minute within the first 15 min after cardiac arrest onset, with patients still receiving CPR at risk of being intubated within the same minute.[243] The matching was based on a time-dependent propensity score and matched 43,314 intubated patients with patients with same propensity for intubation but who were not intubated in the same minute. Compared with not intubating, TI 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; $p < 0.001$).

After reviewing the evidence for airway management during cardiac arrest, the ILCOR ALS Task Force made the following treatment recommendations:[244]

- 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 TI success rate (weak recommendation, low certainty of evidence).
- If an advanced airway is used, we suggest an SGA or TI for adults with OHCA in settings with a high TI success rate (weak recommendation, very low certainty of evidence).
- If an advanced airway is used, we suggest an SGA or TI 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.[238,245] 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. Most patients with ROSC remain comatose and will need tracheal intubation (TI) and mechanical ventilation (See Post-resuscitation Care).[246]

Airway obstruction

Patients requiring resuscitation often have an obstructed airway, usually secondary to loss of consciousness, but occasionally it may be the primary cause of cardiorespiratory arrest. Prompt assessment, with control of the airway and ventilation of the lungs, is essential. This will help to prevent secondary hypoxic damage to the brain and other vital organs. Without adequate oxygenation it may be impossible to achieve ROSC. These principles may not apply to the witnessed primary cardiac arrest in the vicinity of a defibrillator; in this case, the priority is immediate defibrillation.

Basic airway management and adjuncts

There are three manoeuvres that 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 is low even with effective CPR. Based on the physiological rationale and expert opinion, ILCOR recommends giving the highest feasible inspired oxygen concentration during cardiac arrest to maximise oxygen delivery to the brain thereby minimising hypoxic-ischaemic injury.[1] Immediately after ROSC, as soon as arterial blood oxygen saturation can be monitored reliably (by pulse oximetry or arterial blood gas analysis), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation between 94–98% or arterial partial pressure of oxygen (PaO_2) of 10 –13 kPa or 75–100 mmHg. (See Post Resuscitation Care).[246]

Choking

The initial management of foreign body airway obstruction (choking) is addressed in the BLS section.[247,248] 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.[104]

Ventilation

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 (expert opinion). 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. 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.[249] 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).[250] In Europe, the most common approach during CPR with an unprotected airway is to give two ventilations after each sequence of 30 chest compressions.

Once a tracheal tube (TT) or an SGA has been inserted, ventilate the lungs at a rate of 10 min^{-1} and continue chest compressions without pausing during ventilations (expert opinion).[251] 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, e.g. see ERC COVID-19 Guidelines), 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.

Passive oxygen delivery

In the presence of a patent airway, chest compressions alone may result in some ventilation of the lungs.[252] Oxygen can be delivered passively, either via an adapted TT (Boussignac tube),[253,254] or with the combination of an oropharyngeal airway and standard oxygen mask with non-rebreather reservoir.[255] In theory, an 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 VF OHCA, but this was a retrospective analysis and is subject to numerous confounders.[255] The 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.[249]

Choice of airway devices

Disadvantages of TI over bag-mask ventilation include:

- The risk of an unrecognised misplaced TT; in patients with OHCA the reliably documented incidence ranges from 0.5% to 17%: emergency physicians – 0.5%;[256] paramedics - 2.4%,[257] 6%,[258,259] 9%,[260] 17%.[261]
- A prolonged period without chest compressions while TI is attempted. In a study of prehospital TI by paramedics during 100 CA the total duration of the interruptions in CPR associated with TI attempts was 110 s (IQR 54 - 198 s; range 13–446 s) and in 25% the interruptions were more than 3 min.[262] Tracheal intubation attempts accounted for almost 25% of all CPR interruptions.
- A comparatively high failure rate. Intubation success rates correlate with the TI experience attained by individual paramedics.[263] The high failure rate of 51% documented in the PART trial[240] is similar to those documented in some prehospital systems more than 20 years ago.[264,265]
- Tracheal intubation is a difficult skill to acquire and maintain. In one study, anaesthesia residents required about 125 intubations in the operating room setting before they were able to achieve a TI success rate of 95% under such optimal conditions.[266]

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

The ILCOR recommendation is that only systems that achieve high tracheal intubation success rates should use this technique.[242] 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 (TI delayed until after ROSC) in a physician-staffed EMS system.[239] The TI success rate in this study was 98%. The expert consensus of this writing group is that a high success rate is greater than 95% with up to 2 intubation attempts.

Rescuers must weigh the risks and benefits of 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 TI 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 TI attempt should interrupt chest compressions for less than 5 s (expert opinion); if intubation is not achievable within these constraints, recommence bag-mask ventilation. After TI, tube placement must be confirmed immediately (see below) and the tube must be secured adequately.

Videolaryngoscopy

Videolaryngoscopy is being used increasingly in anaesthetic and critical care practice.[267,268] Preliminary studies indicate that compared with direct laryngoscopy, videolaryngoscopy during CPR improves laryngeal view and TI success rates,[269,270] reduces the risk of oesophageal intubation[271] and reduces interruptions to chest compressions.[272] One systematic review concluded that in the prehospital setting, videolaryngoscopy decreased the first-attempt TI 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.[273] Several different videolaryngoscopy systems are available and they do not all perform in the same way. The expert consensus of the writing group is that the rescuer's choice of direct laryngoscopy or videolaryngoscopy should be guided by local protocols and rescuer experience.

Confirmation of correct placement of the tracheal tube

Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation. The evidence supporting the guideline is summarised in longstanding ILCOR recommendations.[1,274,275] Routine use of clinical assessment and immediate capnography reduces this risk significantly.[275,276] Initial assessment includes observation of bilateral chest expansion, bilateral lung auscultation in the axillae (breath sounds should be equal and adequate) and over the epigastrium (breath sounds should be absent). Clinical signs of correct TT placement (condensation in the tube, chest rise, breath sounds on auscultation of lungs, and inability to hear gas entering the stomach) are not reliable. The reported sensitivity (proportion of TI correctly identified) and specificity (proportion of oesophageal intubations correctly identified) of

clinical assessment varies: sensitivity 74 – 100%; specificity 66 – 100%.[256,277–279]

The ILCOR ALS Task Force recommends using waveform capnography to immediately confirm and continuously monitor the position of a TT during CPR in addition to clinical assessment (strong recommendation, low quality evidence).[275] Waveform capnography is given a strong recommendation because it has other potential uses during CPR (see below). The persistence of exhaled CO_2 after six ventilations indicates placement of the TT in the trachea or a main bronchus.[256] The 'No Trace = Wrong Place' campaign by the UK Royal College of Anaesthetists emphasises that immediately after TI (even during CA) the absence of exhaled CO_2 strongly suggests oesophageal intubation.[280]

Waveform capnography is the most sensitive and specific way to confirm and continuously monitor the position of a TT in victims of cardiac arrest and must supplement clinical assessment (visualization of TT through cords and auscultation). Existing portable monitors make capnographic initial confirmation and continuous monitoring of TT position feasible in all out- and in-of-hospital settings where TI is performed.

Ultrasonography of the neck or visualisation with a fibreoptic scope by skilled operators can also be used to identify the presence of a tracheal tube in the trachea. This requires additional equipment and skills. These techniques were not formally reviewed for this guideline.

Cricoid pressure

The use of cricoid pressure in CA is not recommended (expert consensus). Cricoid pressure can impair ventilation, laryngoscopy, TT and SGA insertion, and may even cause complete airway obstruction.[281]

Securing the tracheal tube and supraglottic device

Accidental dislodgement of a TT 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 TT.[238] The most effective method for securing the TT or a SGA has yet to be determined. Use either conventional tapes or ties, or purpose-made holders (Expert opinion).

Cricothyroidotomy

Occasionally it will be impossible to ventilate an apnoeic patient with a bag-mask, or to pass a TT or SGA. This may occur in patients with extensive facial trauma or laryngeal obstruction caused by oedema, tumour or foreign material. In these circumstances, delivery of oxygen through a surgical cricothyroidotomy may be lifesaving.[282] A tracheostomy is contraindicated in an emergency because it is time consuming, hazardous and requires considerable surgical skill and equipment.

Surgical cricothyroidotomy provides a definitive airway that can be used to ventilate the patient's lungs until semi-elective intubation or tracheostomy is performed. Needle cricothyroidotomy is a much more temporary procedure providing only short-term oxygenation and minimal if any pulmonary CO_2 removal.

Drugs and fluids

Vascular access

ILCOR suggests the intravenous route as opposed to the intraosseous route is used as the first attempt for drug administration during adult cardiac arrest.[1,283] This weak recommendation is based on very low-certainty evidence drawn from three retrospective observational studies which included 34,686 adult out-of-hospital cardiac arrests which suggests worse outcomes when the IO route was used.[284–286] Since the ILCOR review, secondary analyses of the PARAMEDIC2.[287] and ALPS randomised trials[288] suggested no significant effect modification by drug administration route although the studies were underpowered to assess for differences between the IV and IO routes.

Consistent with ILCOR, the ERC suggests attempting IV access first to enable drug delivery in adults in cardiac arrest. IO access may be considered if unable to obtain IV access in adults in cardiac arrest.

Vasopressors

ILCOR reviewed the use of vasopressors in cardiac arrest following the publication of the PARAMEDIC2 trial.[242,289] Systematic reviews and meta-analyses examined 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.[290,291] The reviews reported evidence that adrenaline (1 mg) improved the rate of survival to hospital admission and long-term survival (to 3 months) but did not improve favourable neurological outcome. By contrast, the use of high-dose adrenaline or vasopressin (with or without adrenaline) did not improve long term survival or favourable neurological outcome.

These data led to ILCOR upgrading the strength of recommendation to strong recommendation in favour of the use of adrenaline during CPR (strong recommendation, low to moderate certainty of evidence).[242] 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 PARAMEDIC2 trial followed the ERC ALS 2015 Guidelines, which recommended that adrenaline was given as soon as vascular access is obtained for non-shockable rhythms and for shockable rhythms, refractory to 3 attempts at defibrillation.[21] Meta-analysis of the two placebo-controlled trials (PACA and PARAMEDIC2) found that the effects of adrenaline on ROSC relative to placebo were greater for patients with an initially non-shockable rhythm than those with a shockable rhythms.[292] Similar patterns were observed for longer term survival and favourable neurological outcomes, although the differences in effects were less pronounced.[292] A secondary analysis which examined the time to drug administration in the PARAMEDIC2 trial found that whilst the relative treatment effects of adrenaline did not change over time, survival rates and favourable neurological outcomes decreased over time, suggesting early intervention would lead to the best outcomes.[293]

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

Consistent with the ILCOR Treatment Recommendations, the ERC recommends adrenaline 1 mg IV (IO) 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 3 initial shocks, give adrenaline 1 mg IV (IO). Repeat adrenaline 1 mg IV (IO) every 3–5 min whilst ALS continues.

If 3 stacked shocks have been given for a witnessed and monitored shockable cardiac arrest, these initial 3 stacked shocks should be

considered as the first shock with regards to timing of the first dose of adrenaline.

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

Antiarrhythmic drugs

ILCOR updated the Consensus on Science and Treatment Recommendation for antiarrhythmic drugs in 2018.[294] No further relevant studies were identified upon searching the literature to 10 February 2020.

The ILCOR systematic review identified evidence from 14 randomised controlled trials and 17 observational studies which evaluated lidocaine, amiodarone, magnesium, bretylium, nifekalant and procainamide.[295] Meta-analysis of randomised trials in adults, found that none of the anti-arrhythmic drugs improved survival or favourable neurological outcome compared to placebo. Meta-analysis showed that lidocaine compared to placebo improved ROSC (RR = 1.16; 95% CI, 1.03−1.29, p = 0.01).

The largest and most recent 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.[296] 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).[294] 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 defibrillation attempts, irrespective of whether they are consecutive shocks, or interrupted by CPR, or for recurrent VF/pVT during cardiac arrest.[297] 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.[297] 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.[297]

Thrombolytic therapy

The 2020 ILCOR Consensus on Science with Treatment Recommendations pooled evidence from a sub-group analysis of the TROICA trial[298] and 4 observational studies[299–302] which examined the use of thrombolytic drugs in cardiac arrest caused by suspected or confirmed pulmonary embolus (PE). The studies did not find evidence that thrombolytic drugs improved neurological outcome.[298,301] By contrast, in one study, 30-day survival was higher in the intervention group (16% vs 6%; P = 0.005)[302] but not in 3 other studies which examined survival to discharge.[299–301] ROSC also improved in one study[300] but not two others.[299,301] 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 the potential benefits outweighed the potential harm from bleeding.[1]

The ERC endorses the recommendation from ILCOR, which aligns with the ERC guidelines in 2015.[21] 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, consider continuing CPR attempts for at least 60 −90 min before termination of resuscitation attempts.[303–305]

Fluid therapy

No randomised controlled trials have evaluated the routine administration of fluids versus no fluids as a treatment strategy for cardiac arrest. Two large randomised trials provide indirect evidence from treatment strategies designed to induce hypothermia which included administration of up to 2 L ice cold intravenous fluids during OHCA[306] or immediately after ROSC.[307] The studies found no improvement in short[306,307] or long-term outcomes.[308] The studies reported evidence of reduced ROSC in patients with VF,[306] increased rate of re-arrest,[307] and higher rates of pulmonary oedema.[306,307] It is not possible to determine from these studies whether the harmful effects were related to fluid volume per se or the temperature of the infused fluids.[309] Nevertheless, based on expert consensus, the ERC maintains its recommendation to avoid the routine infusion of large volume fluids in the absence of evidence of suspicion of a hypovolaemic cause of the cardiac arrest.

Waveform capnography during advanced life support

This guideline is based on an ILCOR evidence update and scoping review,[1] a recent systematic review,[276] a narrative review[310] and the previous 2015 ERC ALS Guidelines.[21] End-tidal carbon dioxide is the partial pressure of carbon dioxide (PCO_2) measured at the end of expiration. It reflects cardiac output, tissue perfusion and pulmonary blood flow, as well as the ventilation minute volume. 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 plateau phase best reflects the alveolar PCO_2. End-tidal CO_2 is most reliable when the patient's trachea is intubated, but it can also be used with a SGA or bag mask.[311]

The aims of monitoring waveform capnography during CPR include:[21,310]

- **Confirming correct tracheal tube** placement (see airway section).
- **Monitoring the quality of CPR** (ventilation rate and chest compressions). Monitoring ventilation rate helps avoiding hyperventilation during CPR. In a paediatric resuscitation model a greater depth of chest compression was associated with higher end-tidal CO_2 values.[312] Whether this can be used to guide care and improve outcome requires further study.[313]
- **Detecting ROSC during CPR**. When ROSC occurs, end-tidal CO_2 may increase up to three times above the values during CPR.[314] Capnography may therefore help detect ROSC during resuscitation and avoid unnecessary chest compression or adrenaline in a patient with ROSC. However, no specific threshold

for the increase in end-tidal CO_2 has been identified for reliable diagnosis of ROSC. The increase in $ETCO_2$ can start several minutes before a palpable pulse is detected.[315–317]

- **Prognostication during CPR**. Failure to achieve an $ETCO_2$ value >1.33 kPa (10 mmHg) during CPR is associated with a poor outcome in observational studies[276,318,319] This threshold has also been suggested as a criterion to withhold e-CPR in refractory cardiac arrest.[320] However values of $ETCO_2$ during CPR depend on several factors including the timing of measurement (initial vs. final,[321,322] cause of cardiac arrest,[323,324] chest compression quality,[312] ventilation rate and volume,[325] presence of airway closure during CPR[326] and the use of adrenaline.[327,328] In general, $ETCO_2$ tends to decrease during CPR in patients in whom resuscitation is unsuccessful and tends to increase in those who go on to achieve ROSC.[318,329] For this reason, $ETCO_2$ trends might be more appropriate than point values for predicting ROSC during CPR.[276] However, evidence on this is still limited.[329] Studies assessing the prognostic value of $ETCO_2$ have not been blinded, which may have caused a self-fulfilling prophecy. For this reason, although an $ETCO_2$ > 1.33 kPa (10 mmHg) measured after tracheal intubation or after 20 min of CPR may be a predictor of ROSC or survival to discharge, using $ETCO_2$ threshold values alone as a mortality predictor or for the decision to stop a resuscitation attempt is not recommended.[1] In selected patients, continue CPR to facilitate the implementation of other technologies such as E-CPR, that buy time for treatments that address a reversible cause of the cardiac arrest (e.g. re-warming following accidental hypothermia, intra-arrest primary percutaneous coronary intervention for acute myocardial ischaemia).

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.[21]

An ILCOR systematic review assessed the role of POCUS during cardiac arrest as a prognostic tool.[330] 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.[330] The review concluded that no sonographic finding had sufficiently or consistently high sensitivity to support its use as a sole criterion to terminate CPR. The authors of the ILCOR systematic review advised that clinicians should be cautious about introducing additional interruptions in chest compressions with a transthoracic approach to POCUS during cardiac arrest.[1,331,332]

POCUS can be used to diagnose treatable causes of cardiac arrest such as cardiac tamponade or pneumothorax. The ERC ALS 2015 guidelines recommended a sub-xiphoid probe position placed just before chest compressions are paused for a planned rhythm assessment.[21] [Soar 2015 100] These applications were not covered in the ILCOR systematic review; however, the review stressed the issue of over-interpreting the finding of right ventricular dilation in isolation as a diagnostic indicator of massive pulmonary embolism. Right ventricular dilation begins a few minutes after onset of cardiac arrest as blood shifts from the systemic circulation to the right heart along its pressure gradient.[333–335] Right ventricular dilation was consistently observed in a porcine model of cardiac arrest caused by hypovolaemia, hyperkalaemia, and primary arrhythmia,[336] and is a common finding regardless of the cause of OHCA during trans-oesophageal echocardiography performed in the emergency department.[337] At present, there is limited knowledge about the use POCUS during CPR to assess deep vein thrombosis to help diagnose pulmonary embolism, to assess for pleural effusion and FAST (Focussed Assessment with Sonography for Trauma) assessment of the abdomen and aorta.

Mechanical chest compression devices

Informed by evidence from 8 RCTs[338–345] the ILCOR 2015 CoSTR and ERC Guidelines did not recommend the routine use of automated mechanical chest compression devices but did suggest that they are a reasonable alternative when sustained high-quality manual chest compressions are impractical or compromise provider safety.[21,275]

This evidence update focused on randomised controlled trials and systematic reviews.

Two new randomised trials were identified.[346,347] One study examined the use of the Autopulse applied in the emergency department following OHCA (n = 133). The trial found the rate of survival to hospital discharge was higher in the Autopulse group (18.8% versus 6.3%, p = 0.03) but no difference in favourable neurological outcome (16.2% versus 13.4%). A randomized non-inferiority safety study, involving 374 patients, reported that LUCAS device did not cause significantly more serious or life-threatening visceral damage than manual chest compressions. For the Autopulse device, significantly more serious or life-threatening visceral damage than manual compressions cannot be excluded.[346]

Six systematic reviews and meta-analyses were published since the ILCOR review, including a Cochrane review.[348–353] Significant methodological errors in one systematic review and meta-analysis led to its exclusion.[354] Four reviews drew conclusions similar to the ILCOR 2015 review, that mechanical CPR did not improve critical or important outcomes.[348–351] A review focusing solely on mechanical CPR in the in-hospital setting, reported very low-certainty evidence that mechanical chest compressions improved patient outcomes in that setting.[352] A Bayesian network meta-analysis reported that manual CPR was more effective than Autopulse mechanical chest compression device and comparable to LUCAS mechanical chest compression device.[353]

The writing group considered that the new data did not materially alter the previous ERC guidelines on the use of mechanical chest compression devices in cardiac arrest.[21]

Circumstances to consider mechanical chest compression devices

A review identified several specific circumstances where it is difficult to deliver high-quality manual CPR where mechanical CPR can be considered as an alternative.[355] Examples include transporting to hospital in an ambulance or helicopter, during percutaneous coronary intervention, diagnostic imaging such as a CT scan, as a bridge to establishing extra-corporeal CPR or maintaining circulation prior to organ retrieval when resuscitation is unsuccessful. The expert consensus is that mechanical devices should be considered when high-quality manual compressions are not practical or pose a risk to rescuer safety.

Device deployment

Observational studies show that interruptions in chest compressions, particularly immediately before or around the time of attempted defibrillation are harmful.[111,356] Some studies report long pauses in chest compressions associated with mechanical chest compression device deployment.[357–359] Training those responsible for mechanical device deployment can reduce interruptions to less than 15 s.[358,360] The expert consensus is that mechanical devices should be used only in settings where teams are trained in their deployment.

Extracorporeal CPR

Extracorporeal CPR (eCPR) is defined by the ELSO (Extracorporeal Life Support Organization) as the application of rapid-deployment veno-arterial extracorporeal membrane oxygenation (VA-ECMO) to provide circulatory support in patients in whom conventional CPR is unsuccessful in achieving sustained ROSC.[361] The use of eCPR has increased for both IHCA and OHCA in recent years.[362–365]

The 2019 ILCOR CoSTR informed by a systematic review made the following recommendation:[242,244,366]

- We suggest that eCPR may be considered as a rescue therapy for selected patients with cardiac arrest when conventional CPR is failing in settings in which it can be implemented (weak recommendation, very low certainty of evidence).

There is one recent small randomised controlled trial of eCPR for OHCA refractory VF cardiac arrest,[367] and several others in progress. There are no universally agreed indications for eCPR regarding which patients and the optimum time-point during conventional ALS. There are guidelines on when to start eCPR.[320,363,368–370] Inclusion criteria have not been used consistently or prospectively tested in trials.[365] Commonly used criteria include:

- Witnessed cardiac arrest with bystander CPR.
- Time to establishing eCPR is less than 60 min from starting CPR.
- Younger patients (e.g. less than 65–70 years) and no major comorbidities precluding a return to independent life.
- Known or suspected treatable underlying cause of cardiac arrest.

The role of eCPR for specific causes of cardiac arrest is addressed. Cardiac Arrest in Special Circumstances. Establishing an eCPR programme requires a whole system approach (in- and out-of hospital) and considerable resources to implement effectively, and not all healthcare systems will have sufficient resources.[371–373]

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 peri-arrest arrhythmias that cause life-threatening instability. If patients are stable there is time to seek advice from a specialist or more experienced physician. Other international organisations have produced comprehensive evidence-based arrhythmia guidelines.[86,91,374–377] Electrical cardioversion is required in the peri-arrest patient with a clinical unstable arrhythmia while pacing is used in refractory bradycardia. The key interventions are summarised in Fig. 5 and 6.

These guidelines follow recommendations published by international cardiology societies including the European Society of Cardiology (ESC), the American Heart Association (AHA), the American College of Cardiology (ACC) and the Heart Rhythm Society (HRS).[86,91,374–377] Table 4 summarises the supporting evidence for vagal manoeuvres and some of the more commonly used drugs for the treatment of arrhythmias.

Pharmacological cardioversion restores sinus rhythm in approximately 50% of patients with recent-onset AF. Among the several drugs for pharmacological conversion suggested by the ESC,[378] beta-blockers and diltiazem/verapamil are preferred over digoxin because of their rapid onset of action and effectiveness at high sympathetic tone. For patients with LVEF $<$ 40%, consider the smallest dose of beta-blocker to achieve a heart rate less than 110 min^{-1} and add digoxin if necessary. Amiodarone is the drug most likely to be familiar to non-specialists and can be considered for acute heart rate control in atrial fibrillation (AF) patients with haemodynamic instability and severely reduced left ventricular ejection fraction (LVEF).

The ESC has published recent guidelines for the acute management of regular tachycardias in the absence of an established diagnosis.[91] The guidelines for treating regular narrow QRS ($\leq$ 120 ms) and 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.

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.[379] However, in many countries procainamide is either unavailable and/or unlicensed.

Evidence for the treatment of patients with bradycardia was included in ACC/AHA/HRS guidelines published in 2019 (Fig. 6 Bradycardia algorithm).[377] If bradycardia is accompanied by adverse signs, atropine remains the first choice drug.[21] When atropine is ineffective, second line drugs include isoprenaline (5 μg min starting dose) and adrenaline (2–10 μg min). For bradycardia caused by inferior myocardial infarction, heart transplant or spinal cord injury, consider giving aminophylline (100–200 mg slow intravenous injection). Atropine can cause a high-degree atrioventricular (AV) block or even sinus arrest in heart transplant patients.[380] Consider giving intravenous glucagon if beta-blockers or calcium channel blockers are a potential cause of the bradycardia. Consider pacing in patients who are unstable, 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 signs (Fig. 5. Tachycardia algorithm).[381–383] The shock must be synchronised to occur with the R wave of the electrocardiogram rather than with the T wave: VF can be induced if a shock is delivered during the relative refractory portion of the cardiac cycle.[384] 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 or pulseless VT require unsynchronised shocks. Conscious patients require anaesthesia or sedation, before attempting synchronised cardioversion.

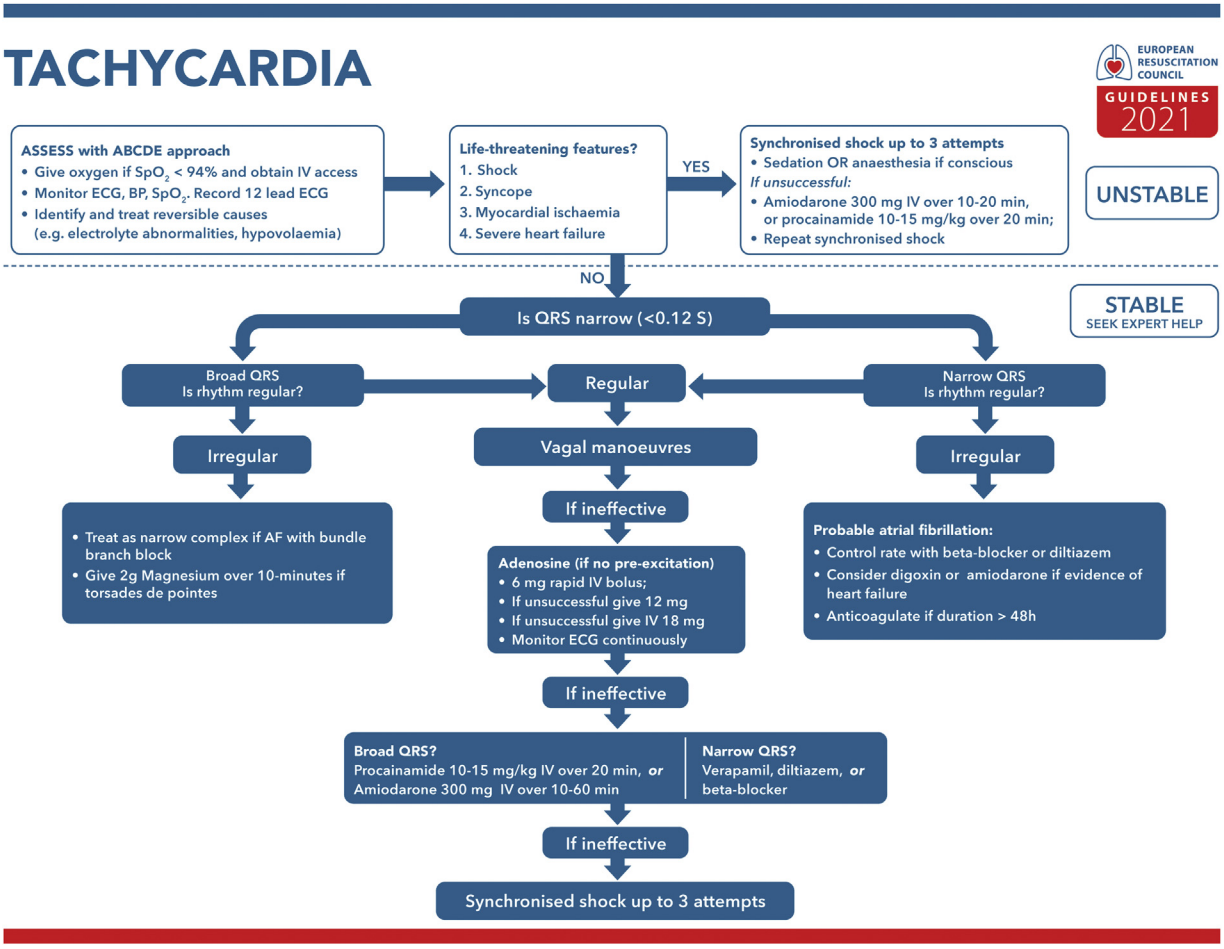

Fig. 5 – Tachycardia algorithm. ABCDE airway, breathing, circulation, disability, exposure BP blood pressure; DC direct current; ECG electrocardiogram; IV intravenous; SpO_2 arterial oxygen saturation; VT ventricular tachycardia.

Cardioversion for atrial fibrillation

Some studies,[127,128] but not all,[130,133] have suggested that antero-posterior pad position is more effective than antero-lateral pad position, but both are acceptable positions.[131] 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) waveform show similar high efficacy in the elective cardioversion of atrial fibrillation.[385] A recent RCT showed that maximum fixed energy electrical cardioversion (360 J BTE in this study) was more effective in achieving sinus rhythm one minute after cardioversion than an energy-escalating strategy.[386] There was no increase in adverse events. An initial synchronised shock at maximum defibrillator output 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.[378]

Cardioversion for atrial flutter and paroxysmal supraventricular tachycardia

Atrial flutter and paroxysmal supraventricular tachycardia (SVT) generally require less energy than atrial fibrillation for cardioversion.[387] Give an initial shock of 70–120 J. Give subsequent shocks using stepwise increases in energy.[194]

Cardioversion for pulsatile ventricular tachycardia

The energy required for cardioversion of VT depends on the morphological characteristics and rate of the arrhythmia.[388] Ventricular tachycardia with a pulse responds well using energy levels of 120–150 J for the initial shock. Consider stepwise increases if the first shock fails to achieve sinus rhythm.[388]

Pacing

Consider pacing in patients who are unstable, with symptomatic bradycardia refractory to drug therapy. Immediate pacing is indicated especially when the block is at or below the His-Purkinje level. If transthoracic (transcutaneous) pacing is ineffective, consider transvenous pacing. Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because this will likely respond to cardiac pacing. The use of epicardial wires to pace the myocardium following cardiac surgery is effective and discussed elsewhere. Do not attempt pacing for asystole unless P waves are present; it does not increase short or long-term survival in- or out-of-hospital.[389–397] For haemodynamically unstable, conscious patients with bradyarrhythmia, percussion pacing as a bridge to electrical pacing may be attempted, although its effectiveness has not been established.[104,398,399] Give serial rhythmic blows with the closed fist over the left lower edge of the sternum to pace the heart at a

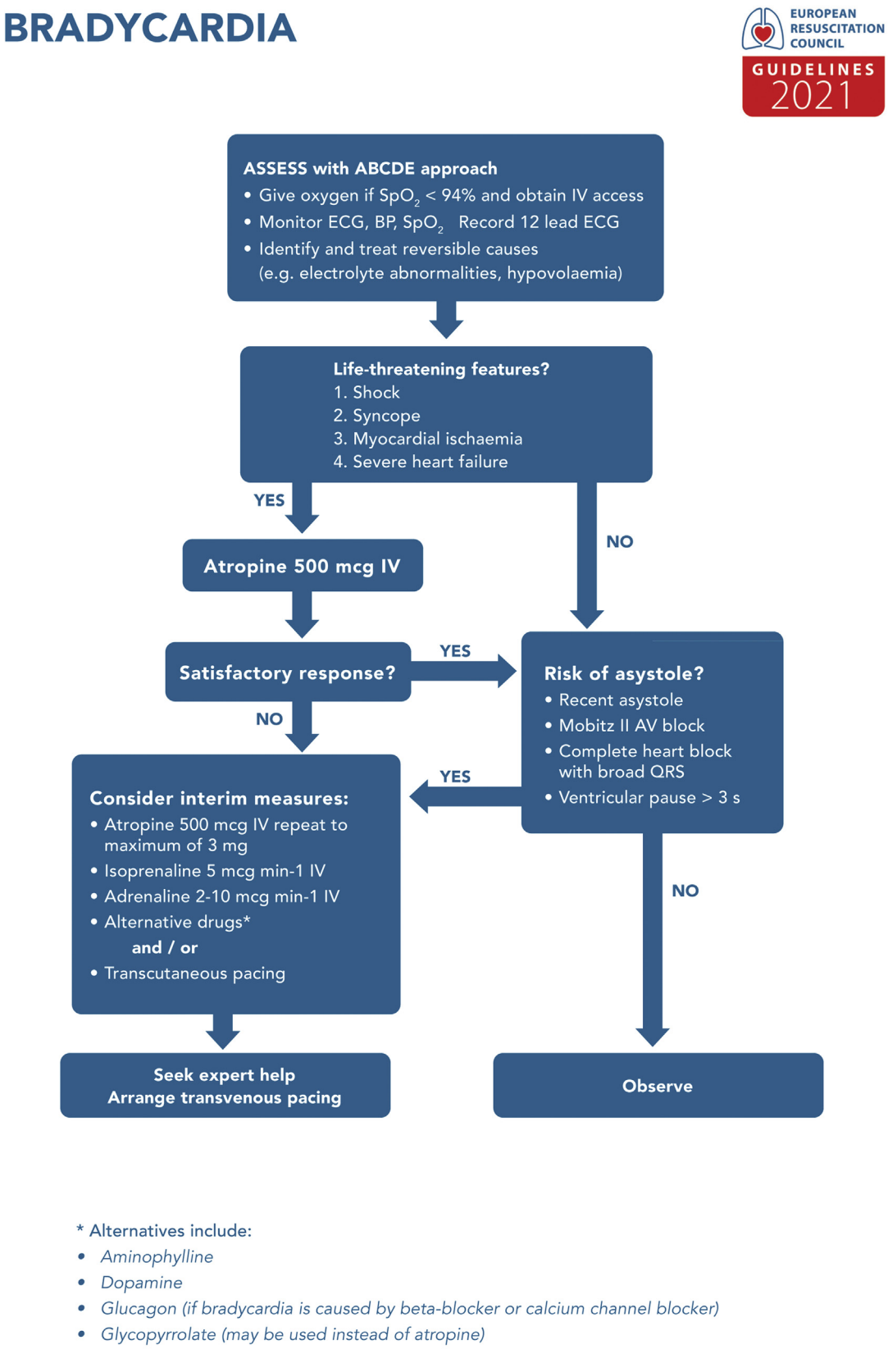

Fig. 6 – Bradycardia algorithm. ABCDE airway, breathing, circulation, disability, exposure BP blood pressure; ECG electrocardiogram; IV intravenous; SpO_2 arterial oxygen saturation.

Table 4 – Recommendations for the acute management of narrow and wide QRS tachycardia (Drugs may be administered via peripheral IV in an emergency. HF heart failure; LV left ventricular).

Drug /procedure	Indication	Timing	Dose/delivery	Notes
Vagal Manoeuvre	Narrow QRS tachycardia Wide QRS tachycardia		Blow into a 10 mL syringe with sufficient force to move the plunger	Preferably in the supine position with leg elevation[400–403]
Adenosine	Narrow QRS tachycardia Wide QRS tachycardia	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[404–406] When using an 18 mg dose, take into account the tolerability/side effects in the individual patient.
Verapamil or diltiazem	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%).[404,406–411]
Beta-blockers (IV esmolol or metoprolol)	Narrow QRS tachycardia	Consider if vagal manoeuvres and adenosine fail	Esmolol (0.5 mg/kg IV bolus or 0.05–0.3 mg/kg/min infusion) Metoprolol (2.5–15 mg given IV in 2.5 mg boluses),	More effective in reducing the heart rate than in terminating tachycardia.[410,412–414]
Procainamide	Wide QRS tachycardia	Consider if vagal manoeuvres and adenosine fail	10–15 mg/kg IV over 20 min	[379,415]
Amiodarone	Narrow and wide QRS tachycardia	Consider if vagal manoeuvres and adenosine fail	300 mg IV over 10–60 min according to circumstances – followed by infusion of 900 mg in 24h	[416,417]
Magnesium	Polymorphic wide QRS tachycardia (torsades de pointes -TdP)		2 g 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[361,418]

physiological rate of 50–70 min^{-1}. Transthoracic and percussion pacing can cause discomfort Consider giving analgesic or sedative drugs in conscious patients

Uncontrolled organ donation after circulatory death

Following cardiac arrest, less than a half of patients achieve ROSC.[17,34] When standard ALS fails to achieve ROSC, there are three broad treatment strategies:[419]

- Stop resuscitation and declare death.
- In selected patients, continue CPR to facilitate the implementation of other technologies such as E-CPR, that buy time for treatments that address a reversible cause of the cardiac arrest (e.g. re-warming following accidental hypothermia, intra-arrest primary percutaneous coronary intervention for acute myocardial ischaemia).
- Continue CPR to maintain organ perfusion and transfer to a hospital with an uncontrolled donation after circulatory death (uDCD) pathway.

This guideline focuses on uDCD (Maastricht category I/II donors).[420] The post-resuscitation care guidelines includes guidance for organ donation pathways following brain death or controlled donation after circulatory death (Maastricht category III donors) in patients who achieve ROSC or are treated with eCPR.[246,420] We acknowledge the ethical, cultural and legislative issues that lead to variation in the use of uDCD.

Across Europe, demand for transplanted organs continues to outstrip supply. Uncontrolled donation after circulatory death (uDCD) provides an opportunity for cardiac arrest victims in whom ROSC cannot be achieved, to donate their organs. In Europe, uDCD is currently undertaken in regions of Spain, France, The Netherlands, Belgium, and Italy.[421–430] Organs that can be recovered include kidneys, liver, pancreas and lungs. Observational data show that long-term uDCD graft success is comparable to other organ recovery approaches.[428,430–432]

There is no universal consensus on selection criteria for uDCD, and the identification of a potential donor currently follows regional/national protocols. These generally include: age above 18 year (for adults) and not over 55 or 65 years, a no-flow time (the interval between cardiac arrest and CPR start) within 15–30 min, and a total warm ischaemia time (the interval between cardiac arrest and the start of organ preservation) not longer than 150 min.[433] Exclusion criteria generally include trauma, homicide, or suicide as a cause of arrest, and comorbidities such as cancer, sepsis, and, according to local programme and the targeted organ to transplant, kidney and liver disease.[433]

Uncontrolled donation after circulatory death is a time-critical, resource-intensive, complex and ethically challenging process.[434,435] Following completion of aggressive resuscitation efforts and

confirmation of death, a 'no-touch' period is observed to rule-out the possibility of auto-resuscitation.[436] Organ preservation procedures are then immediately started and continued whilst family consent for organ recovery is sought, and organs are assessed for suitability for donation. [437–439] For abdominal organs, organ preservation typically uses an extracorporeal circulation with membrane oxygenation via a femoro-femoral bypass.[434] Catheters with balloons are used to limit circulation to the abdominal cavity.[440] Following consent and completion of practical arrangements, the patient is transferred to the operating theatre for organ recovery.

Consent to organ donation is obtained as soon as possible during the process from a surrogate decision maker (e.g., a family member) or by retrieving previous consent registered on a donor card or in a public registry, if available. The urgency and nature of the process creates several ethical challenges that are unique to uDCD, highlighting the importance of clear local protocols, and legislative and societal acceptance of the process.[434] These issues are discussed in the ethics section of the guidelines.[441]

Debriefing

ILCOR undertook a systematic review of debriefing following cardiac arrest in 2020.[16] The review included four observational studies and identified that debriefing was associated with improvements in hospital survival, ROSC and CPR quality.[442–445] These studies all described use of a cold debrief that incorporated data on CPR quality downloaded from defibrillators.[446] Based on these data, ILCOR continues to make a weak recommendation based on very low certainty evidence supporting the use of data-driven performance-focused debriefing. The justification and evidence to decision framework noted the substantial heterogeneity in debriefing intervention between studies. ILCOR also noted that the intervention is highly likely to be acceptable to stakeholders and cost of implementation may be modest. A potential harm of debriefing is the psychological effect on rescuers of discussing challenging clinical events. The ILCOR summary recorded no evidence of harm from included studies but highlighted the need to consider this effect when implementing debriefing interventions.

Conflict of interest

JS declares his role of Co-editor Resuscitation; he declares institutional research funding for the NAP7 project from the Royal College of Anaesthetists.

GDP reports funding from Elsevier for his role as an editor of the journal Resuscitation. He reports research funding from the National Institute for Health Research in relation to the PARAMEDIC2 trial and the RESPECT project.

JN reports funding from Elsevier for his role as Editor in Chief of the journals Resuscitation and Resuscitation Plus. He reports research funding from the National Institute for Health Research in relation to the PARAMEDIC2 trial and the AIRWAYS2 trial.

CD declares research grants from RCUK and funding from the National Institute for Health Research in relation to the PARAMEDIC2 trial and the AIRWAYS2 trial.

TO declares research grants from Swedish Heart and Lung Foundation, Laerdal Foundation and Lof -Swedish patient insurance and Swedish state under the agreement between the Swedish government and the county councils, the ALF-agreement.

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