

Available online at www.sciencedirect.com

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

European Resuscitation Council Guidelines 2021: Adult advanced life support

Jasmeet Soar^{a,*}, Bernd W. Böttiger^b, Pierre Carli^c, Keith Couper^d, Charles D. Deakin^e, Therese Djärv^f, Carsten Lott^g, Theresa Olasveengen^h, Peter Paalⁱ, Tommaso Pellis^j, Gavin D. Perkins^k, Claudio Sandroni^{l,m}, Jerry P. Nolanⁿ

^a Southmead Hospital, North Bristol NHS Trust, Bristol, UK

^b Department of Anaesthesiology and Intensive Care Medicine, University Hospital of Cologne, Cologne, Germany

^c SAMU de Paris, Centre Hospitalier Universitaire Necker Enfants Malades, Assistance Publique Hôpitaux de Paris, and Université Paris Descartes, Paris, France

^d Critical Care Unit, University Hospitals Birmingham NHS Foundation Trust, Birmingham, UK; Warwick Medical School, University of Warwick, Coventry, UK

^e University Hospital Southampton NHS Foundation Trust, Southampton, UK; South Central Ambulance Service NHS Foundation Trust, Otterbourne, UK

^f Dept of Acute and Reparative Medicine, Karolinska University Hospital, Stockholm, Sweden, Department of Medicine Solna, Karolinska Institutet, Stockholm, Sweden

^g Department of Anesthesiology, University Medical Center, Johannes Gutenberg-Universität Mainz, Germany

^h Department of Anesthesiology, Oslo University Hospital and Institute of Clinical Medicine, University of Oslo, Norway

ⁱ Department of Anaesthesiology and Intensive Care Medicine, Hospitallers Brothers Hospital, Paracelsus Medical University, Salzburg, Austria

^j Department of Anaesthesia and Intensive Care, Azienda Sanitaria Friuli Occidentale, Italy

^k University of Warwick, Warwick Medical School and University Hospitals Birmingham NHS Foundation Trust, Coventry, UK

^l Department of Intensive Care, Emergency Medicine and Anaesthesiology, Fondazione Policlinico Universitario A. Gemelli-IRCCS, Rome, Italy

^m Institute of Anaesthesiology and Intensive Care Medicine, Università Cattolica del Sacro Cuore, Rome, Italy

ⁿ University of Warwick, Warwick Medical School, Coventry, CV4 7AL; Royal United Hospital, Bath, UK

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 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

* Corresponding author.

E-mail address: jasmeet.soar@nbt.nhs.uk (J. Soar).

<https://doi.org/10.1016/j.resuscitation.2021.02.010>

Available online xxx

0300-9572/© 2021 European Resuscitation Council. Published by Elsevier B.V. All rights reserved

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.

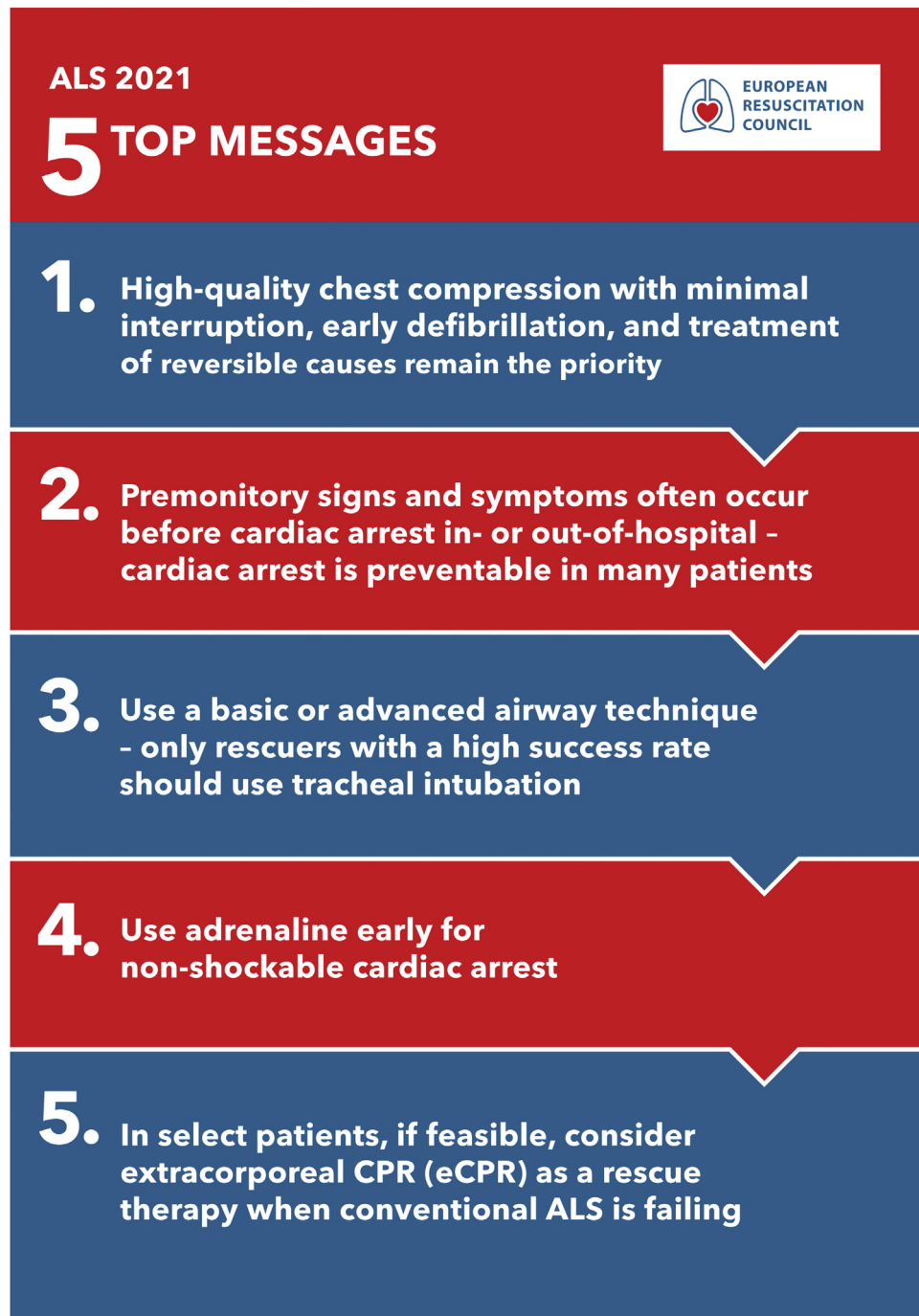


Fig. 1 – ALS summary

- 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 (ETCO₂), 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 refrillation 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 ETCO₂ during CPR may indicate that ROSC has occurred. However, chest compression should not be interrupted based on this sign alone.
- Although high and increasing ETCO₂ values are associated with increased rates of ROSC and survival after CPR, do not use a low ETCO₂ 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).

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

Table 1 – Causes of sudden cardiac arrest (SCD).

Adapted from Kandala⁴⁶ 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

adolescent SCD, 50% of patients had misinterpreted symptoms before death.⁴⁴ CHD is the most frequent cause of explained SC DY; 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 SC DY 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 SC DY 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 SC DY.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 supine⁸⁰

Sudden onset palpitation immediately followed by syncope⁸⁰

Minor

No warning symptoms or short (<10 s) prodrome^{80–83}

Family history of SCD at young age⁸⁴

Syncope in the sitting position⁸⁵

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 fibrillation⁸⁷

Pre-excited QRS complex

Short QTc interval (<= 340 ms)⁸⁶

Atypical Brugada patterns⁸⁶

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 Guidelines²¹ 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/RRRT (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

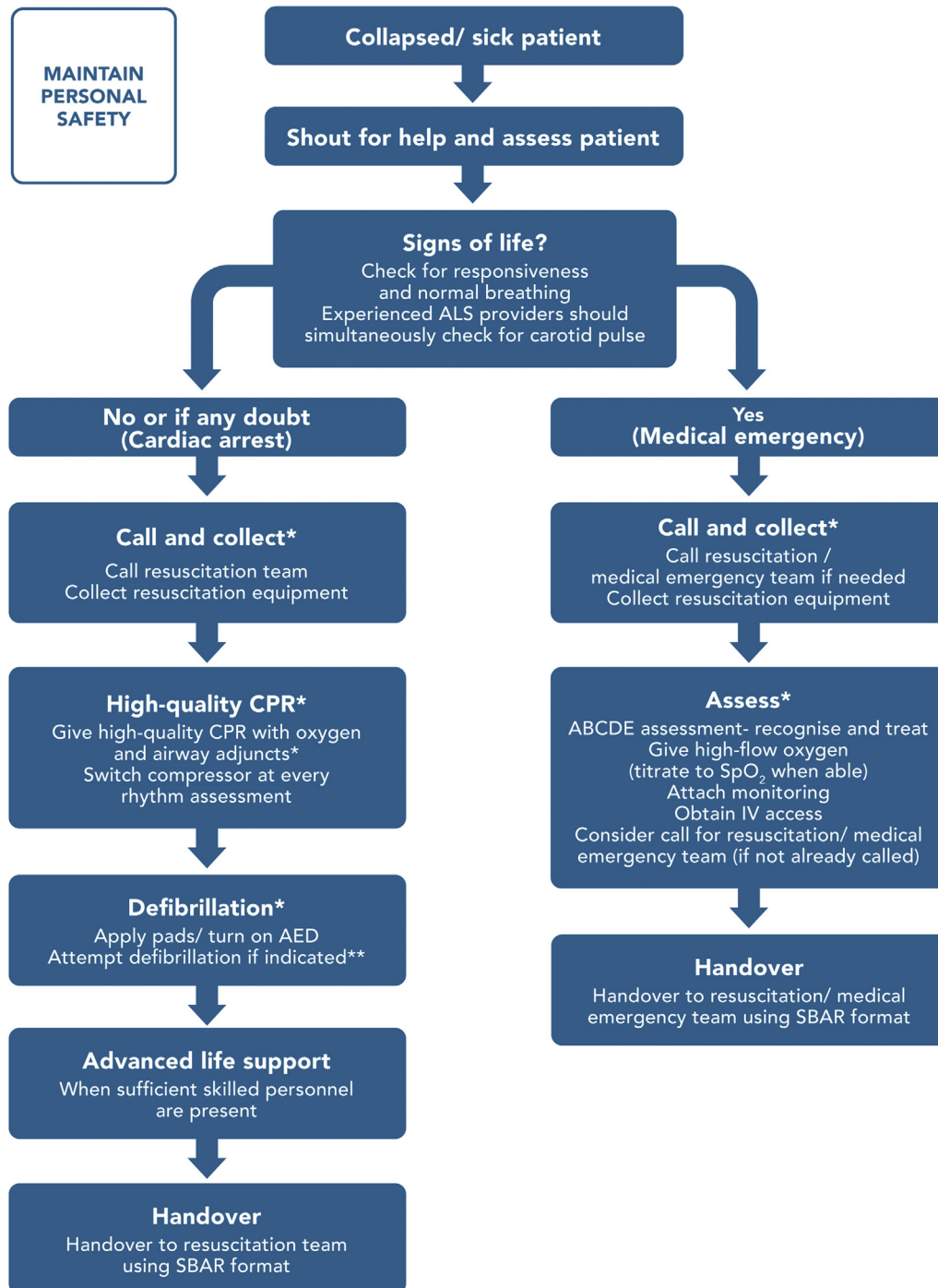
EMS personnel and interventions

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: ≤ 6 exposures (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. ≥ 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

IN-HOSPITAL RESUSCITATION



* Undertake actions concurrently if sufficient staff available

**Use a manual defibrillator if trained and device available

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 ET_{CO₂}), 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

ADVANCED LIFE SUPPORT

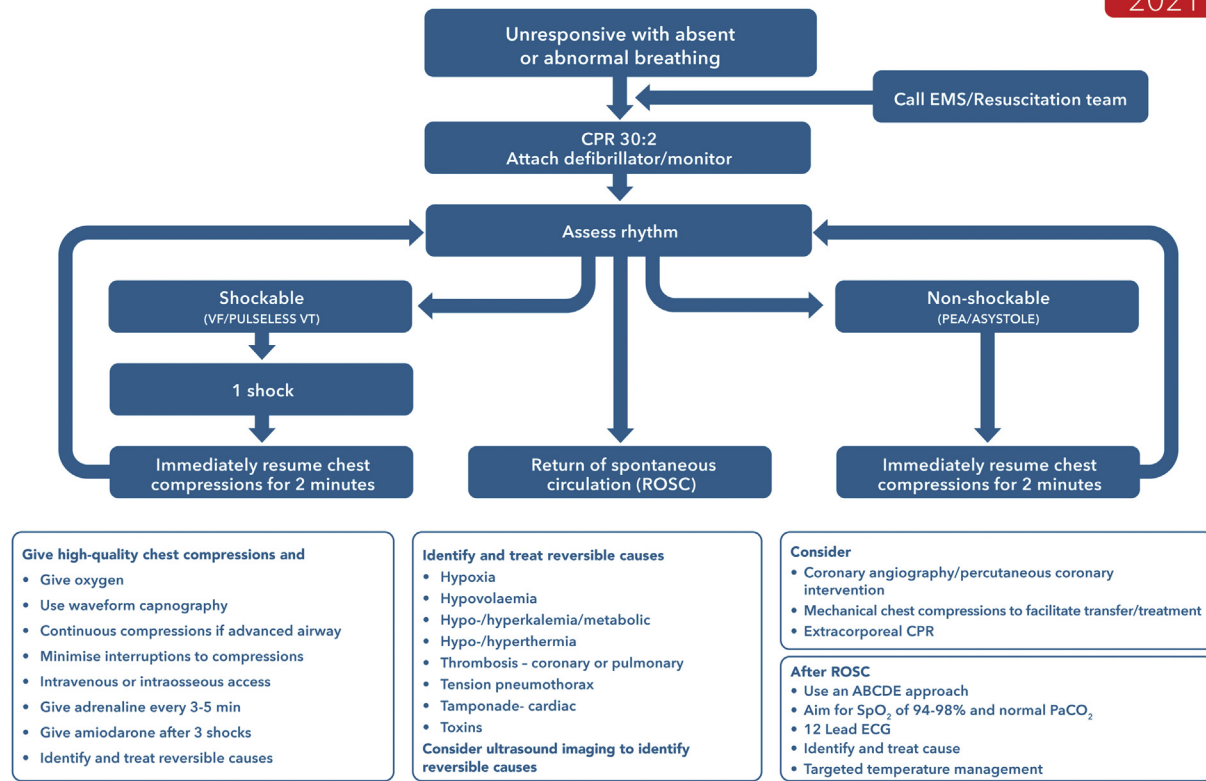


Fig. 3 – Advanced Life Support algorithm. ABCDE airway, breathing, circulation, disability, exposure CPR cardiopulmonary resuscitation; ECG electrocardiogram; EMS emergency medical system; PEA pulseless electrical activity; PaCO₂ arterial partial pressure of carbon dioxide; ROSC return of spontaneous circulation; SpO₂ 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

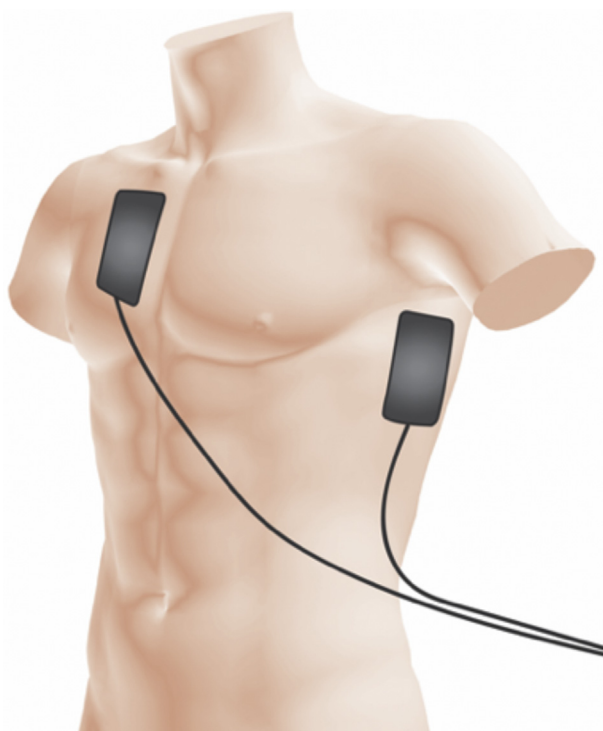


Fig. 4 – Correct pad placement for defibrillation (© Charles Deakin).

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 transmural 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, extracardiac 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 OHCA 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 trial²⁴⁰ 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 intubation²⁷¹ 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 CO₂ 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 CO₂ 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 CO₂ 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 trials²⁸⁸ 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 trial²⁹⁸ 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 study³⁰⁰ 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 OHCA³⁰⁶ 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 review³¹⁰ and the previous 2015 ERC ALS Guidelines.²¹ End-tidal carbon dioxide is the partial pressure of carbon dioxide (PCO₂) 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 PCO₂ in the exhaled air during CPR. In the typical capnogram, the ETCO₂ recorded at the end of the plateau phase best reflects the alveolar PCO₂. End-tidal CO₂ 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 hyper-ventilation during CPR. In a paediatric resuscitation model a greater depth of chest compression was associated with higher end-tidal CO₂ 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 CO₂ 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 CO₂ has been identified for reliable diagnosis of ROSC. The increase in ETCO₂ can start several minutes before a palpable pulse is detected.^{315–317}

- **Prognostication during CPR.** Failure to achieve an ETCO₂ 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 ETCO₂ 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 CPR³²⁶ and the use of adrenaline.^{327,328} In general, ETCO₂ 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, ETCO₂ 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 ETCO₂ have not been blinded, which may have caused a self-fulfilling prophecy. For this reason, although an ETCO₂ > 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 ETCO₂ 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 transoesophageal 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 (≤ 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 μ g min starting dose) and adrenaline (2–10 μ g 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.

TACHYCARDIA

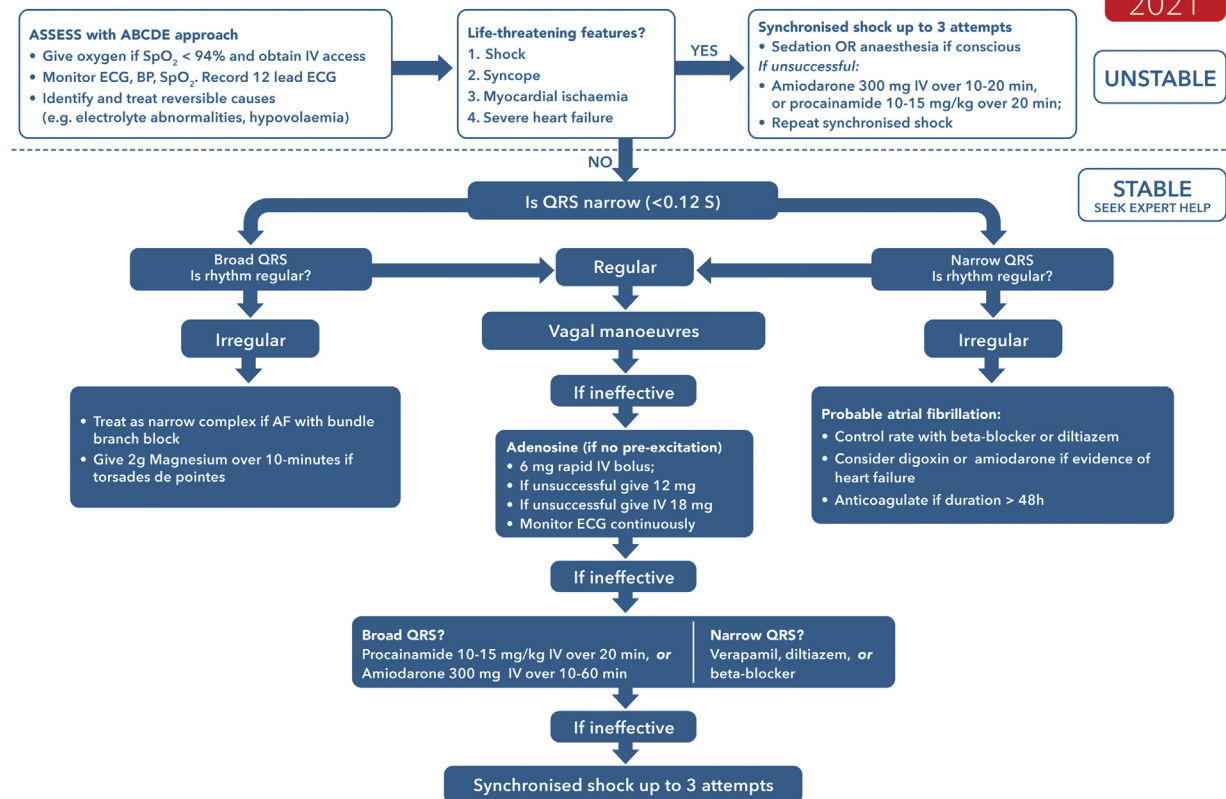


Fig. 5 – Tachycardia algorithm. ABCDE airway, breathing, circulation, disability, exposure BP blood pressure; DC direct current; ECG electrocardiogram; IV intravenous; SpO₂ arterial oxygen saturation; VT ventricular tachycardia.

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

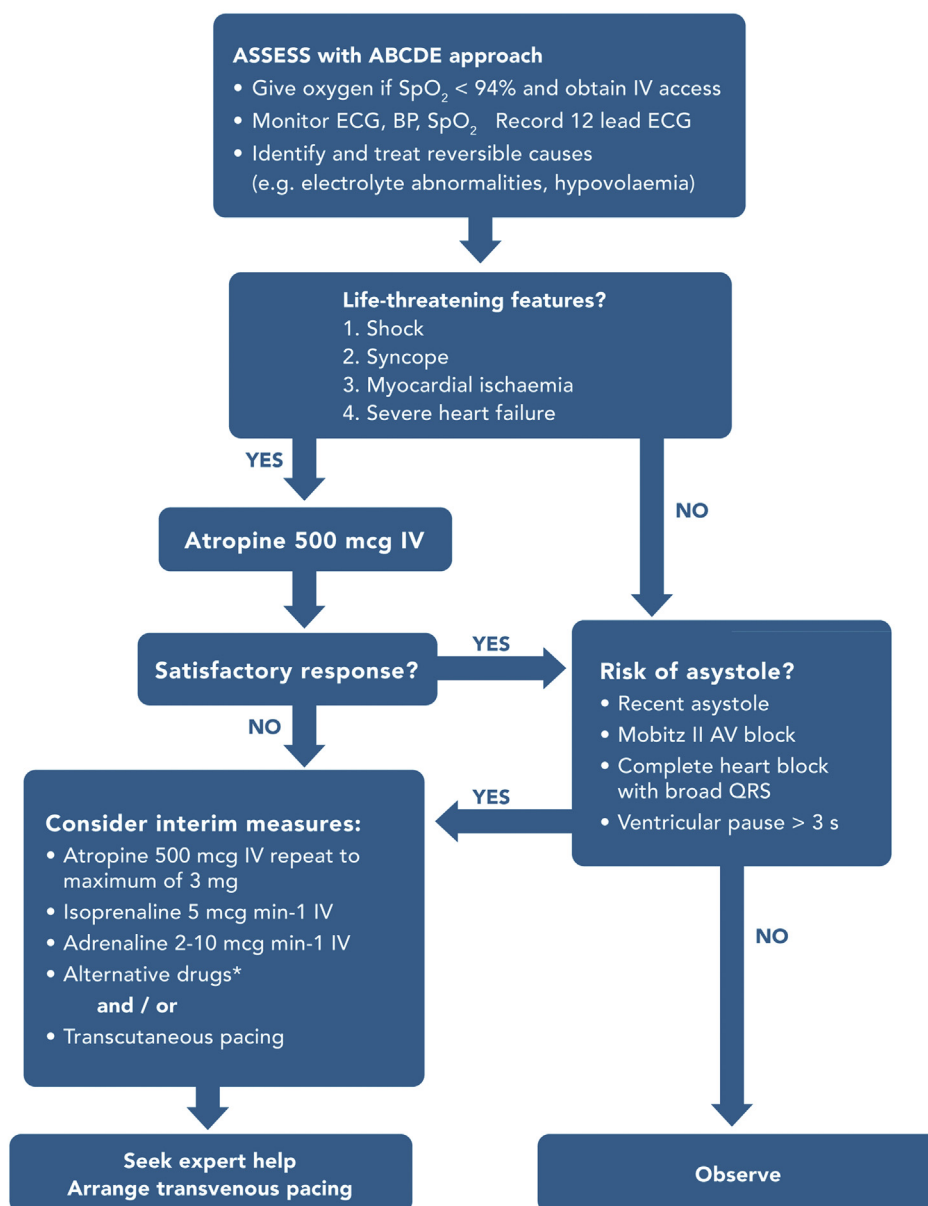
Cardioversion for pulsatile ventricular tachycardia

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

BRADYCARDIA



* Alternatives include:

- Aminophylline
- Dopamine
- Glucagon (if bradycardia is caused by beta-blocker or calcium channel blocker)
- Glycopyrrolate (may be used instead of atropine)

Fig. 6 – Bradycardia algorithm. ABCDE airway, breathing, circulation, disability, exposure BP blood pressure; ECG electrocardiogram; IV intravenous; SpO₂ 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.

BB declared speakers honorarium from Baxalta, ZOLL, FomF, Bard, Stemple, Novartis Pharma, Philips Market DACH, Bioscience Valuation BSV.

TP declares Speakers honorarium from BARD.

Acknowledgements

The authors acknowledge the following individuals who contributed to the 2015 version of this guideline: Markus B. Skrifvars, Gary B. Smith, Kjetil Sunde, Rudolph W. Koster, Koenraad G. Monsieurs, Nikolaos I. Nikolaou. GDP is supported by the National Institute for Health Research (NIHR) Applied Research Collaboration (ARC) West Midlands. The views expressed are those of the author(s) and not necessarily those of the NIHR or the Department of Health and Social Care.

REFERENCES

1. Soar J, Berg KM, Andersen LW, et al. Adult Advanced Life Support: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Resuscitation* 2020;156: A80–A119.
2. Nolan JP, Monsieurs KG, Bossaert L, et al. European Resuscitation Council COVID-19 guidelines executive summary. *Resuscitation* 2020;153:45–55.
3. Perkins GD, Morley PT, Nolan JP, et al. International Liaison Committee on Resuscitation: COVID-19 consensus on science, treatment recommendations and task force insights. *Resuscitation* 2020;151:145–7.
4. Couper K, Taylor-Phillips S, Grove A, et al. COVID-19 in cardiac arrest and infection risk to rescuers: A systematic review. *Resuscitation* 2020;151:59–66.
- 4a. Perkins GD, Graesner JT, Semeraro F, et al. European resuscitation council guidelines 2021 – executive summary. *Resuscitation* 2021;161.
5. Andersen LW, Holmberg MJ, Berg KM, Donnino MW, Granfeldt A. In-Hospital Cardiac Arrest: A Review. *JAMA* 2019;321: 1200–10.
6. Hessulf F, Karlsson T, Lundgren P, et al. Factors of importance to 30-day survival after in-hospital cardiac arrest in Sweden - A population-based register study of more than 18,000 cases. *International Journal of Cardiology* 2018;255:237–42.
7. Aune S, Herlitz J, Bang A. Characteristics of patients who die in hospital with no attempt at resuscitation. *Resuscitation* 2005;65: 291–9.
8. Skrifvars MB, Hilden HM, Finne P, Rosenberg PH, Castren M. Prevalence of ‘do not attempt resuscitation’ orders and living wills among patients suffering cardiac arrest in four secondary hospitals. *Resuscitation* 2003;58:65–71.
9. Fritz ZB, Heywood RM, Moffat SC, Bradshaw LE, Fuld JP. Characteristics and outcome of patients with DNACPR orders in an acute hospital; an observational study. *Resuscitation* 2014;85: 104–8.
10. Perkins GD, Griffiths F, Slowther AM, et al. Do-not-attempt-cardiopulmonary-resuscitation decisions: an evidence synthesis. Southampton (UK). 2016.
11. Moskowitz A, Berg KM, Cocchi MN, et al. Cardiac arrest in the intensive care unit: An assessment of preventability. *Resuscitation* 2019;145:15–20.
12. Roberts D, Djarv T. Preceding national early warnings scores among in-hospital cardiac arrests and their impact on survival. *Am J Emerg Med* 2017;35:1601–6.

13. Smith GB. In-hospital cardiac arrest: is it time for an in-hospital' chain of prevention'? *Resuscitation* 2010;81:1209–11.
14. Physicians RCo. National Early Warning Score (NEWS) 2: Standardising the assessment of acute-illness severity in the NHS. Updated report of a working party. London: RCP; 2017.
15. National Institute for Health and Clinical Excellence. NICE clinical guideline 50 Acutely ill patients in hospital: recognition of and response to acute illness in adults in hospital. London: National Institute for Health and Clinical Excellence. 2007.
16. Greif R. Education, Implementation, and Teams 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2020.
17. Nolan JP, Soar J, Smith GB, et al. Incidence and outcome of in-hospital cardiac arrest in the United Kingdom National Cardiac Arrest Audit. *Resuscitation* 2014;85:987–92.
18. Smith GB, Redfern O, Maruotti A, Recio-Saucedo A, Griffiths P. The Missed Care Study G. The association between nurse staffing levels and a failure to respond to patients with deranged physiology: A retrospective observational study in the UK. *Resuscitation* 2020;149:202–8.
19. Griffiths P, Recio-Saucedo A, Dall'Orta C, et al. The association between nurse staffing and omissions in nursing care: A systematic review. *J Adv Nurs* 2018;74:1474–87.
20. Thoren A, Rawshani A, Herlitz J, et al. ECG-monitoring of in-hospital cardiac arrest and factors associated with survival. *Resuscitation* 2020;150:130–8.
21. Soar J, Nolan JP, Bottiger BW, et al. European Resuscitation Council Guidelines for Resuscitation 2015: Section 3. Adult advanced life support. *Resuscitation* 2015;95:100–47.
22. Douw G, Schoonhoven L, Holwerda T, et al. Nurses' worry or concern and early recognition of deteriorating patients on general wards in acute care hospitals: a systematic review. *Crit Care* 2015;19:230.
23. Douw G, Huisman-de Waal G, van Zanten ARH, van der Hoeven JG, Schoonhoven L. Capturing early signs of deterioration: the dutch-early-nurse-worry-indicator-score and its value in the Rapid Response System. *Journal of clinical nursing* 2017;26:2605–13.
24. Marshall S, Harrison J, Flanagan B. The teaching of a structured tool improves the clarity and content of interprofessional clinical communication. *Qual Saf Health Care* 2009;18:137–40.
25. Buljac-Samardzic M, Doekhie KD, van Wijngaarden JDH. Interventions to improve team effectiveness within health care: a systematic review of the past decade. *Hum Resour Health* 2020;18:2.
26. Muller M, Jurgens J, Redaelli M, Klingberg K, Hautz We, Stock S. Impact of the communication and patient hand-off tool SBAR on patient safety: a systematic review. *BMJ Open* 2018;8:e022202.
27. Brady PW, Zix J, Brilli R, et al. Developing and evaluating the success of a family activated medical emergency team: a quality improvement report. *BMJ Qual Saf* 2015;24:203–11.
28. Albutt AK, O'Hara JK, Conner MT, Fletcher SJ, Lawton RJ. Is there a role for patients and their relatives in escalating clinical deterioration in hospital? A systematic review. *Health Expect* 2017;20:818–25.
29. McKinney A, Fitzsimons D, Blackwood B, McGaughey J. Patient and family-initiated escalation of care: a qualitative systematic review protocol. *Syst Rev* 2019;8:91.
30. Subbe CP, Bannard-Smith J, Bunch J, et al. Quality metrics for the evaluation of Rapid Response Systems: Proceedings from the third international consensus conference on Rapid Response Systems. *Resuscitation* 2019;141:1–12.
31. Winters Bd, Weaver Sj, Pfoh Er, Yang T, Pham Jc, Dy Sm. Rapid-response systems as a patient safety strategy: a systematic review. *Ann Intern Med* 2013;158:417–25.
32. Virani SS, Alonso A, Benjamin EJ, et al. Heart Disease and Stroke Statistics-2020 Update: A Report From the American Heart Association. *Circulation* 2020;141:e139–596.
33. Kiguchi T, Okubo M, Nishiyama C, et al. Out-of-hospital cardiac arrest across the World: First report from the International Liaison Committee on Resuscitation (ILCOR). *Resuscitation* 2020;152:39–49.
34. Grasner JT, Wnent J, Herlitz J, et al. Survival after out-of-hospital cardiac arrest in Europe - Results of the EuReCa TWO study. *Resuscitation* 2020;148:218–26.
35. Grasner JT. Epidemiology of cardiac arrest in Europe. *Resuscitation* 2021.
36. Basso C, Carturan E, Pilichou K, Rizzo S, Corrado D, Thiene G. Sudden cardiac death with normal heart: molecular autopsy. *Cardiovasc Pathol* 2010;19:321–5.
37. Goldberger JJ, Basu A, Boineau R, et al. Risk stratification for sudden cardiac death: a plan for the future. *Circulation* 2014;129:516–26.
38. Harmon KG, Drezner JA, Wilson MG, Sharma S. Incidence of sudden cardiac death in athletes: a state-of-the-art review. *Heart* 2014;100:1227–34.
39. Kramer MR, Drori Y, Lev B. Sudden death in young soldiers. High incidence of syncope prior to death. *Chest* 1988;93:345–7.
40. Mazzanti A, O'Rourke S, Ng K, et al. The usual suspects in sudden cardiac death of the young: a focus on inherited arrhythmogenic diseases. *Expert Rev Cardiovasc Ther* 2014;12:499–519.
41. Quigley F, Greene M, O'Connor D, Kelly F. A survey of the causes of sudden cardiac death in the under 35-year-age group. *Ir Med J* 2005;98:232–5.
42. Winkel BG, Risgaard B, Sadjadieh G, Bundgaard H, Haunso S, Tfelt-Hansen J. Sudden cardiac death in children (1-18 years): symptoms and causes of death in a nationwide setting. *Eur Heart J* 2014;35:868–75.
43. Wisten A, Forsberg H, Krantz P, Messner T. Sudden cardiac death in 15-35-year olds in Sweden during 1992-99. *J Intern Med* 2002;252:529–36.
44. Wisten A, Messner T. Symptoms preceding sudden cardiac death in the young are common but often misinterpreted. *Scandinavian Cardiovascular Journal* 2005;39:143–9.
45. Wisten A, Messner T. Young Swedish patients with sudden cardiac death have a lifestyle very similar to a control population. *Scandinavian Cardiovascular Journal* 2005;39:137–42.
46. Kandala J, Oommen C, Kern KB. Sudden cardiac death. *Br Med Bull* 2017;122:5–15.
47. Winkel BG, Jabbari R, Tfelt-Hansen J. How to prevent SCD in the young? *International Journal of Cardiology* 2017;237:6–9.
48. Wellens HJ, Schwartz PJ, Lindemans FW, et al. Risk stratification for sudden cardiac death: current status and challenges for the future. *Eur Heart J* 2014;35:1642–51.
49. Buxton AE, Lee KL, DiCarlo L, et al. Electrophysiologic testing to identify patients with coronary artery disease who are at risk for sudden death. Multicenter Unsustained Tachycardia Trial Investigators. *N Engl J Med* 2000;342:1937–45.
50. Buxton AE. Sudden death in ischemic heart disease - 2017. *International Journal of Cardiology* 2017;237:64–6.
51. Levine YC, Rosenberg MA, Mittleman M, et al. B-type natriuretic peptide is a major predictor of ventricular tachyarrhythmias. *Heart Rhythm* 2014;11:1109–16.
52. Levine YC, Matos J, Rosenberg MA, Manning WJ, Josephson ME, Buxton AE. Left ventricular sphericity independently predicts appropriate implantable cardioverter-defibrillator therapy. *Heart Rhythm* 2016;13:490–7.
53. Brignole M, Moya A, de Lange FJ, et al. 2018 ESC Guidelines for the diagnosis and management of syncope. *Eur Heart J* 2018;39:1883–948.
54. Stecker EC, Reinier K, Marijon E, et al. Public health burden of sudden cardiac death in the United States. *Circ Arrhythm Electrophysiol* 2014;7:212–7.
55. Jayaraman R, Reinier K, Nair S, et al. Risk Factors of Sudden Cardiac Death in the Young: Multiple-Year Community-Wide Assessment. *Circulation* 2018;137:1561–70.
56. Bjune T, Risgaard B, Kruckow L, et al. Post-mortem toxicology in young sudden cardiac death victims: a nationwide cohort study. *Europace* 2018;20:614–21.

57. Behr ER, Dalageorgou C, Christiansen M, et al. Sudden arrhythmic death syndrome: familial evaluation identifies inheritable heart disease in the majority of families. *Eur Heart J* 2008;29:1670–80.
58. Giudicessi JR, Ackerman MJ. Role of genetic heart disease in sentinel sudden cardiac arrest survivors across the age spectrum. *International Journal of Cardiology* 2018;270:214–20.
59. Maron BJ. Clinical Course and Management of Hypertrophic Cardiomyopathy. *N Engl J Med* 2018;379:655–68.
60. Stecker EC, Vickers C, Waltz J, et al. Population-based analysis of sudden cardiac death with and without left ventricular systolic dysfunction: two-year findings from the Oregon Sudden Unexpected Death Study. *J Am Coll Cardiol* 2006;47:1161–6.
61. Marijon E, Uy-Evanado A, Dumas F, et al. Warning Symptoms Are Associated With Survival From Sudden Cardiac Arrest. *Ann Intern Med* 2016;164:23–9.
62. Muller D, Agrawal R, Arntz HR. How sudden is sudden cardiac death? *Circulation* 2006;114:1146–50.
63. Nishiyama C, Iwami T, Kawamura T, et al. Prodromal symptoms of out-of-hospital cardiac arrests: a report from a large-scale population-based cohort study. *Resuscitation* 2013;84:558–63.
64. Deakin CD, Quartermain A, Ellery J. Do patients suffering an out-of-hospital cardiac arrest present to the ambulance service with symptoms in the preceding 48 h? *Eur Heart J Qual Care Clin Outcomes* 2020;6:308–14.
65. Priori SG, Blomstrom-Lundqvist C. 2015 European Society of Cardiology Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death summarized by co-chairs. *Eur Heart J* 2015;36:2757–9.
66. Authors/Task Force m, Elliott PM, Anastasakis A, et al. 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy: the Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). *Eur Heart J* 2014;35:2733–79.
67. Brugada J, Brugada R, Brugada P. Determinants of sudden cardiac death in individuals with the electrocardiographic pattern of Brugada syndrome and no previous cardiac arrest. *Circulation* 2003;108:3092–6.
68. Jons C, Moss AJ, Goldenberg I, et al. Risk of fatal arrhythmic events in long QT syndrome patients after syncope. *J Am Coll Cardiol* 2010;55:783–8.
69. Krahn AD, Healey JS, Simpson CS, et al. Sentinel symptoms in patients with unexplained cardiac arrest: from the cardiac arrest survivors with preserved ejection fraction registry (CASPER). *J Cardiovasc Electrophysiol* 2012;23:60–6.
70. Marcus FI, McKenna WJ, Sherrill D, et al. Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia: proposed modification of the Task Force Criteria. *Eur Heart J* 2010;31:806–14.
71. Marjamaa A, Hiippala A, Arrhenius B, et al. Intravenous epinephrine infusion test in diagnosis of catecholaminergic polymorphic ventricular tachycardia. *J Cardiovasc Electrophysiol* 2012;23:194–9.
72. Nava A, Bauce B, Basso C, et al. Clinical profile and long-term follow-up of 37 families with arrhythmogenic right ventricular cardiomyopathy. *J Am Coll Cardiol* 2000;36:2226–33.
73. Priori SG, Napolitano C, Gasparini M, et al. Natural history of Brugada syndrome: insights for risk stratification and management. *Circulation* 2002;105:1342–7.
74. Schinkel Af. Implantable cardioverter defibrillators in arrhythmogenic right ventricular dysplasia/cardiomyopathy: patient outcomes, incidence of appropriate and inappropriate interventions, and complications. *Circ Arrhythm Electrophysiol* 2013;6:562–8.
75. Schwartz PJ, Spazzolini C, Priori SG, et al. Who are the long-QT syndrome patients who receive an implantable cardioverter-defibrillator and what happens to them?: data from the European Long-QT Syndrome Implantable Cardioverter-Defibrillator (LQTS ICD) Registry. *Circulation* 2010;122:1272–82.
76. Spirito P, Autore C, Rapezzi C, et al. Syncope and risk of sudden death in hypertrophic cardiomyopathy. *Circulation* 2009;119:1703–10.
77. Quinn J, McDermott D, Stiell I, Kohn M, Wells G. Prospective validation of the San Francisco Syncope Rule to predict patients with serious outcomes. *Ann Emerg Med* 2006;47:448–54.
78. Reed MJ, Newby DE, Coull AJ, Prescott RJ, Jacques KG, Gray AJ. The ROSE (risk stratification of syncope in the emergency department) study. *J Am Coll Cardiol* 2010;55:713–21.
79. Quinn JV, Stiell IG, McDermott DA, Sellers KL, Kohn MA, Wells GA. Derivation of the San Francisco Syncope Rule to predict patients with short-term serious outcomes. *Ann Emerg Med* 2004;43:224–32.
80. Del Rosso A, Ungar A, Maggi R, et al. Clinical predictors of cardiac syncope at initial evaluation in patients referred urgently to a general hospital: the EGSYS score. *Heart* 2008;94:1620–6.
81. Alboni P, Brignole M, Menozzi C, et al. Diagnostic value of history in patients with syncope with or without heart disease. *J Am Coll Cardiol* 2001;37:1921–8.
82. Calkins H, Shyr Y, Frumin H, Schork A, Morady F. The value of the clinical history in the differentiation of syncope due to ventricular tachycardia, atrioventricular block, and neurocardiogenic syncope. *Am J Med* 1995;98:365–73.
83. Costantino G, Perego F, Dipaola F, et al. Short- and long-term prognosis of syncope, risk factors, and role of hospital admission: results from the STePS (Short-Term Prognosis of Syncope) study. *J Am Coll Cardiol* 2008;51:276–83.
84. Colman N, Bakker A, Linzer M, Reitsma JB, Wieling W, Wilde AA. Value of history-taking in syncope patients: in whom to suspect long QT syndrome? *Europace* 2009;11:937–43.
85. Jamjoom AA, Nikkar-Esfahani A, Fitzgerald JE. Operating theatre related syncope in medical students: a cross sectional study. *BMC Med Educ* 2009;9:14.
86. Priori SG, Blomstrom-Lundqvist C, Mazzanti A, et al. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: The Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). *Eur Heart J* 2015;36:2793–867.
87. Sheldon R, Rose S, Connolly S, Ritchie D, Koshman ML, Frenneaux M. Diagnostic criteria for vasovagal syncope based on a quantitative history. *Eur Heart J* 2006;27:344–50.
88. Mahmood S, Lim L, Akram Y, Alford-Morales S, Sherin K, Committee APP. Screening for sudden cardiac death before participation in high school and collegiate sports: American College of Preventive Medicine position statement on preventive practice. *Am J Prev Med* 2013;45:130–3.
89. Hainline B, Drezner J, Baggish A, et al. Interassociation consensus statement on cardiovascular care of college student-athletes. *Br J Sports Med* 2017;51:74–85.
90. Malhotra A, Dhutia H, Finocchiaro G, et al. Outcomes of Cardiac Screening in Adolescent Soccer Players. *N Engl J Med* 2018;379:524–34.
91. Brugada J, Katritsis DG, Arbelo E, et al. 2019 ESC Guidelines for the management of patients with supraventricular tachycardia The Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC). *Eur Heart J* 2020;41:655–720.
92. Perez MV, Mahaffey KW, Hedlin H, et al. Large-Scale Assessment of a Smartwatch to Identify Atrial Fibrillation. *N Engl J Med* 2019;381:1909–17.
93. Guo Y, Wang H, Zhang H, et al. Mobile Photoplethysmographic Technology to Detect Atrial Fibrillation. *J Am Coll Cardiol* 2019;74:2365–75.
94. ERC Quality standards for cardiopulmonary resuscitation practice and training. 2019. (Accessed 05 October 2020, 2020, at https://www.erc.edu/sites/5714e77d5e615861f00f7d18/assets/5dedf6664c84860818e4d3c0/CPR_quality_standards_In_hosp_accute_ERC_V3_Final_1_.pdf).
95. Chan PS, Krumholz HM, Nichol G, Nallamothu BK. Delayed time to defibrillation after in-hospital cardiac arrest. *N Engl J Med* 2008;358:9–17.

96. Larsen MP, Eisenberg MS, Cummins RO, Hallstrom AP. Predicting survival from out-of-hospital cardiac arrest: a graphic model. *Ann Emerg Med* 1993;22:1652–8.
97. Whitaker DK, Nolan JP, Castren M, Abela C, Goldik Z. Implementing a standard internal telephone number 2222 for cardiac arrest calls in all hospitals in Europe. *Resuscitation* 2017;115:A14–A5.
98. Featherstone P, Chalmers T, Smith GB. RSVP: a system for communication of deterioration in hospital patients. *Br J Nurs* 2008;17:860–4.
99. De Meester K, Verspuy M, Monsieurs KG, Van Bogaert P. SBAR improves nurse-physician communication and reduces unexpected death: a pre and post intervention study. *Resuscitation* 2013;84:1192–6.
100. Ornato JP, Peberdy MA, Reid RD, Feeser VR, Dhindsa HS. Impact of resuscitation system errors on survival from in-hospital cardiac arrest. *Resuscitation* 2012;83:63–9.
101. Weng TI, Huang CH, Ma MH, et al. Improving the rate of return of spontaneous circulation for out-of-hospital cardiac arrests with a formal, structured emergency resuscitation team. *Resuscitation* 2004;60:137–42.
102. Panesar Ss, Ignatowicz Am, Donaldson Lj. Errors in the management of cardiac arrests: an observational study of patient safety incidents in England. *Resuscitation* 2014;85:1759–63.
103. Bray J, Nehme Z, Nguyen A, Lockey A, Finn J. Education Implementation Teams Task Force of the International Liaison Committee on R. A systematic review of the impact of emergency medical service practitioner experience and exposure to out of hospital cardiac arrest on patient outcomes. *Resuscitation* 2020;155:134–42.
- 103a. Smyth M, Perkins G, Coppola A, et al. - on behalf of the International Liaison Committee on Resuscitation Education, Implementation and Teams Task Force. Prehospital termination of resuscitation (TOR) rules Draft Consensus on Science with Treatment Recommendations. International Liaison Committee on Resuscitation (ILCOR) Education, Implementation and Teams Task Force, 2020, January 6. Available from: <http://ilcor.org>.
104. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation* 2020;142:S41–91.
105. Nichol G, Sayre MR, Guerra F, Poole J. Defibrillation for Ventricular Fibrillation: A Shocking Update. *J Am Coll Cardiol* 2017;70:1496–509.
106. Dyson K, Bray Je, Smith K, Bernard S, Straney L, Finn J. Paramedic Exposure to Out-of-Hospital Cardiac Arrest Resuscitation Is Associated With Patient Survival. *Circ Cardiovasc Qual Outcomes* 2016;9:154–60.
107. Tuttle JE, Hubble MW. Paramedic Out-of-hospital Cardiac Arrest Case Volume Is a Predictor of Return of Spontaneous Circulation. *West J Emerg Med* 2018;19:654–9.
108. Yeung J, Matsuyama T, Bray J, Reynolds J, Skrifvars MB. Does care at a cardiac arrest centre improve outcome after out-of-hospital cardiac arrest? - A systematic review. *Resuscitation* 2019;137:102–15.
109. Edelson DP, Abella BS, Kramer-Johansen J, et al. Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. *Resuscitation* 2006;71:137–45.
110. Eftestol T, Sunde K, Steen PA. Effects of interrupting precordial compressions on the calculated probability of defibrillation success during out-of-hospital cardiac arrest. *Circulation* 2002;105:2270–3.
111. Cheskes S, Schmicker RH, Christenson J, et al. Perishock pause: an independent predictor of survival from out-of-hospital shockable cardiac arrest. *Circulation* 2011;124:58–66.
112. Cheskes S, Schmicker RH, Verbeek PR, et al. The impact of peri-shock pause on survival from out-of-hospital shockable cardiac arrest during the Resuscitation Outcomes Consortium PRIMED trial. *Resuscitation* 2014;85:336–42.
113. Gundersen K, Kvaloy JT, Kramer-Johansen J, Steen PA, Eftestol T. Development of the probability of return of spontaneous circulation in intervals without chest compressions during out-of-hospital cardiac arrest: an observational study. *BMC Med* 2009;7:6.
114. Sell RE, Sarno R, Lawrence B, et al. Minimizing pre- and post-defibrillation pauses increases the likelihood of return of spontaneous circulation (ROSC). *Resuscitation* 2010;81:822–5.
115. Edelson DP, Robertson-Dick BJ, Yuen TC, et al. Safety and efficacy of defibrillator charging during ongoing chest compressions: a multi-center study. *Resuscitation* 2010;81:1521–6.
116. Perkins GD, Davies RP, Soar J, Thickett DR. The impact of manual defibrillation technique on no-flow time during simulated cardiopulmonary resuscitation. *Resuscitation* 2007;73:109–14.
117. Otto Q, Musiol S, Deakin CD, Morley P, Soar J. Anticipatory manual defibrillator charging during advanced life support: A scoping review. *Resuscitation Plus* 2020 1–2:100004.
118. Miller PH. Potential fire hazard in defibrillation. *JAMA* 1972;221:192.
119. Hummel 3rd RS, Ornato JP, Weinberg SM, Clarke AM. Spark-generating properties of electrode gels used during defibrillation. A potential fire hazard. *JAMA* 1988;260:3021–4.
120. ECRI. Defibrillation in oxygen-enriched environments [hazard]. *Health Devices* 1987;16:113–4.
121. Lefever J, Smith A. Risk of fire when using defibrillation in an oxygen enriched atmosphere. *Medical Devices Agency Safety Notices* 1995;3:1–3.
122. Ward ME. Risk of fires when using defibrillators in an oxygen enriched atmosphere. *Resuscitation* 1996;31:173.
123. Theodorou AA, Gutierrez JA, Berg RA. Fire attributable to a defibrillation attempt in a neonate. *Pediatrics* 2003;112:677–9.
124. Pagan-Carlo LA, Spencer KT, Robertson CE, Dengler A, Birkett C, Kerber RE. Transthoracic defibrillation: importance of avoiding electrode placement directly on the female breast. *J Am Coll Cardiol* 1996;27:449–52.
125. Foster AG, Deakin CD. Accuracy of instructional diagrams for automated external defibrillator pad positioning. *Resuscitation* 2019;139:282–8.
126. Deakin CD, Sado DM, Petley GW, Clewlow F. Is the orientation of the apical defibrillation paddle of importance during manual external defibrillation? *Resuscitation* 2003;56:15–8.
127. Kirchhof P, Eckardt L, Loh P, et al. Anterior-posterior versus anterior-lateral electrode positions for external cardioversion of atrial fibrillation: a randomised trial. *Lancet* 2002;360:1275–9.
128. Botto GL, Politi A, Bonini W, Broffoni T, Bonatti R. External cardioversion of atrial fibrillation: role of paddle position on technical efficacy and energy requirements. *Heart* 1999;82:726–30.
129. Alp NJ, Rahman S, Bell JA, Shahi M. Randomised comparison of antero-lateral versus antero-posterior paddle positions for DC cardioversion of persistent atrial fibrillation. *International Journal of Cardiology* 2000;75:211–6.
130. Mathew TP, Moore A, McIntyre M, et al. Randomised comparison of electrode positions for cardioversion of atrial fibrillation. *Heart* 1999;81:576–9.
131. Kirkland S, Stiell I, AlShawabkeh T, Campbell S, Dickinson G, Rowe BH. The efficacy of pad placement for electrical cardioversion of atrial fibrillation/flutter: a systematic review. *Acad Emerg Med* 2014;21:717–26.
132. Zhang B, Li X, Shen D, Zhen Y, Tao A, Zhang G. Anterior-posterior versus anterior-lateral electrode position for external electrical cardioversion of atrial fibrillation: a meta-analysis of randomized controlled trials. *Arch Cardiovasc Dis* 2014;107:280–90.
133. Walsh SJ, McCarty D, McClelland AJ, et al. Impedance compensated biphasic waveforms for transthoracic cardioversion of atrial fibrillation: a multi-centre comparison of antero-apical and antero-posterior pad positions. *Eur Heart J* 2005;26:1298–302.
134. Manegold JC, Israel CW, Ehrlich JR, et al. External cardioversion of atrial fibrillation in patients with implanted pacemaker or cardioverter-defibrillator systems: a randomized comparison of monophasic and biphasic shock energy application. *Eur Heart J* 2007;28:1731–8.

135. Alferness CA. Pacemaker damage due to external countershock in patients with implanted cardiac pacemakers. *Pacing Clin Electrophysiol* 1982;5:457–8.
136. Pitcher D, Soar J, Hogg K, et al. Cardiovascular implanted electronic devices in people towards the end of life, during cardiopulmonary resuscitation and after death: guidance from the Resuscitation Council (UK), British Cardiovascular Society and National Council for Palliative Care. *Heart* 2016;102(Suppl 7):A1–A17.
137. Olsen JA, Brunborg C, Steinberg M, et al. Pre-shock chest compression pause effects on termination of ventricular fibrillation/tachycardia and return of organized rhythm within mechanical and manual cardiopulmonary resuscitation. *Resuscitation* 2015;93:158–63.
138. Deakin CD, Lee-Shrewsbury V, Hogg K, Petley GW. Do clinical examination gloves provide adequate electrical insulation for safe hands-on defibrillation? I: Resistive properties of nitrile gloves. *Resuscitation* 2013;84:895–9.
139. Deakin CD, McLaren RM, Petley GW, Clewlow F, Dalrymple-Hay MJ. Effects of positive end-expiratory pressure on transthoracic impedance—implications for defibrillation. *Resuscitation* 1998;37:9–12.
140. Jacobs I, Sunde K, Deakin CD, et al. Part 6: Defibrillation: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation* 2010;122:S325–37.
141. Sunde K, Jacobs I, Deakin CD, et al. Part 6: Defibrillation: 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Resuscitation* 2010;81(Suppl 1):e71–85.
142. Jost D, Degrange H, Verret C, et al. DEFI 2005: a randomized controlled trial of the effect of automated external defibrillator cardiopulmonary resuscitation protocol on outcome from out-of-hospital cardiac arrest. *Circulation* 2010;121:1614–22.
143. Berdowski J, Schulten RJ, Tijssen JG, van Alem AP, Koster RW. Delaying a shock after takeover from the automated external defibrillator by paramedics is associated with decreased survival. *Resuscitation* 2010;81:287–92.
144. Sunde K, Eftestol T, Askenberg C, Steen PA. Quality assessment of defibrillation and advanced life support using data from the medical control module of the defibrillator. *Resuscitation* 1999;41:237–47.
145. Rea TD, Shah S, Kudenchuk PJ, Copass MK, Cobb LA. Automated external defibrillators: to what extent does the algorithm delay CPR? *Ann Emerg Med* 2005;46:132–41.
146. Pierce AE, Roppolo LP, Owens PC, Pepe PE, Idris AH. The need to resume chest compressions immediately after defibrillation attempts: an analysis of post-shock rhythms and duration of pulselessness following out-of-hospital cardiac arrest. *Resuscitation* 2015;89:162–8.
147. Link MS, Berkow LC, Kudenchuk PJ, et al. Part 7: Adult Advanced Cardiovascular Life Support: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation* 2015;(132):S444–64.
148. Eftestol T, Wik L, Sunde K, Steen PA. Effects of cardiopulmonary resuscitation on predictors of ventricular fibrillation defibrillation success during out-of-hospital cardiac arrest. *Circulation* 2004;110:10–5.
149. Eftestol T, Sunde K, Aase SO, Husoy JH, Steen PA. Predicting outcome of defibrillation by spectral characterization and nonparametric classification of ventricular fibrillation in patients with out-of-hospital cardiac arrest. *Circulation* 2000;102:1523–9.
150. Callaway CW, Sherman LD, Mosesso Jr. VN, Dietrich TJ, Holt E, Clarkson MC. Scaling exponent predicts defibrillation success for out-of-hospital ventricular fibrillation cardiac arrest. *Circulation* 2001;103:1656–61.
151. Weaver WD, Cobb LA, Dennis D, Ray R, Hallstrom AP, Copass MK. Amplitude of ventricular fibrillation waveform and outcome after cardiac arrest. *Ann Intern Med* 1985;102:53–5.
152. Brown CG, Dzwonczyk R. Signal analysis of the human electrocardiogram during ventricular fibrillation: frequency and amplitude parameters as predictors of successful countershock. *Ann Emerg Med* 1996;27:184–8.
153. Callahan M, Braun O, Valentine W, Clark Dm, Zegans C. Prehospital cardiac arrest treated by urban first-responders: profile of patient response and prediction of outcome by ventricular fibrillation waveform. *Ann Emerg Med* 1993;22:1664–77.
154. Strohmenger HU, Lindner KH, Brown CG. Analysis of the ventricular fibrillation ECG signal amplitude and frequency parameters as predictors of countershock success in humans. *Chest* 1997;111:584–9.
155. Strohmenger HU, Eftestol T, Sunde K, et al. The predictive value of ventricular fibrillation electrocardiogram signal frequency and amplitude variables in patients with out-of-hospital cardiac arrest. *Anesth Analg* 2001;93:1428–33.
156. Podbregar M, Kovacic M, Podbregar-Mars A, Brezocnik M. Predicting defibrillation success by 'genetic' programming in patients with out-of-hospital cardiac arrest. *Resuscitation* 2003;57:153–9.
157. Menegazzi JJ, Callaway CW, Sherman LD, et al. Ventricular fibrillation scaling exponent can guide timing of defibrillation and other therapies. *Circulation* 2004;109:926–31.
158. Povoas HP, Weil MH, Tang W, Bisera J, Klouche K, Barbatsis A. Predicting the success of defibrillation by electrocardiographic analysis. *Resuscitation* 2002;53:77–82.
159. Noc M, Weil MH, Tang W, Sun S, Pernat A, Bisera J. Electrocardiographic prediction of the success of cardiac resuscitation. *Crit Care Med* 1999;27:708–14.
160. Strohmenger HU, Lindner KH, Keller A, Lindner IM, Pfenninger EG. Spectral analysis of ventricular fibrillation and closed-chest cardiopulmonary resuscitation. *Resuscitation* 1996;33:155–61.
161. Noc M, Weil MH, Gazmuri RJ, Sun S, Biscera J, Tang W. Ventricular fibrillation voltage as a monitor of the effectiveness of cardiopulmonary resuscitation. *Journal of Laboratory and Clinical Medicine* 1994;124:421–6.
162. Lightfoot CB, Nremt P, Callaway CW, et al. Dynamic nature of electrocardiographic waveform predicts rescue shock outcome in porcine ventricular fibrillation. *Ann Emerg Med* 2003;42:230–41.
163. Marn-Pernat A, Weil MH, Tang W, Pernat A, Bisera J. Optimizing timing of ventricular defibrillation. *Crit Care Med* 2001;29:2360–5.
164. Hamprecht FA, Achleitner U, Krismer AC, et al. Fibrillation power, an alternative method of ECG spectral analysis for prediction of countershock success in a porcine model of ventricular fibrillation. *Resuscitation* 2001;50:287–96.
165. Amann A, Achleitner U, Antretter H, et al. Analysing ventricular fibrillation ECG-signals and predicting defibrillation success during cardiopulmonary resuscitation employing N(alpha)-histograms. *Resuscitation* 2001;50:77–85.
166. Brown CG, Griffith RF, Van Ligten P, et al. Median frequency—a new parameter for predicting defibrillation success rate. *Ann Emerg Med* 1991;20:787–9.
167. Amann A, Rheinberger K, Achleitner U, et al. The prediction of defibrillation outcome using a new combination of mean frequency and amplitude in porcine models of cardiac arrest. *Anesth Analg* 2002;95:716–22.
168. Firoozabadi R, Nakagawa M, Helfenbein ED, Babaeizadeh S. Predicting defibrillation success in sudden cardiac arrest patients. *J Electrocardiol* 2013;46:473–9.
169. Ristagno G, Li Y, Fumagalli F, Finzi A, Quan W. Amplitude spectrum area to guide resuscitation—a retrospective analysis during out-of-hospital cardiopulmonary resuscitation in 609 patients with ventricular fibrillation cardiac arrest. *Resuscitation* 2013;84:1697–703.
170. Ristagno G, Mauri T, Cesana G, et al. Amplitude spectrum area to guide defibrillation: a validation on 1617 patients with ventricular fibrillation. *Circulation* 2015;131:478–87.

171. Freese JP, Jorgenson DB, Liu PY, et al. Waveform analysis-guided treatment versus a standard shock-first protocol for the treatment of out-of-hospital cardiac arrest presenting in ventricular fibrillation: results of an international randomized, controlled trial. *Circulation* 2013;128:995–1002.
172. Coult J, Blackwood J, Sherman L, Rea TD, Kudenchuk PJ, Kwok H. Ventricular Fibrillation Waveform Analysis During Chest Compressions to Predict Survival From Cardiac Arrest. *Circ Arrhythm Electrophysiol* 2019;12:e006924.
173. Aiello S, Perez M, Cogan C, et al. Real-Time Ventricular Fibrillation Amplitude-Spectral Area Analysis to Guide Timing of Shock Delivery Improves Defibrillation Efficacy During Cardiopulmonary Resuscitation in Swine. *Journal of the American Heart Association* 2017;6.
174. Nakagawa Y, Amino M, Inokuchi S, Hayashi S, Wakabayashi T, Noda T. Novel CPR system that predicts return of spontaneous circulation from amplitude spectral area before electric shock in ventricular fibrillation. *Resuscitation* 2017;113:8–12.
175. He M, Lu Y, Zhang L, Zhang H, Gong Y, Li Y. Combining Amplitude Spectrum Area with Previous Shock Information Using Neural Networks Improves Prediction Performance of Defibrillation Outcome for Subsequent Shocks in Out-Of-Hospital Cardiac Arrest Patients. *PLoS One* 2016;11:e0149115.
176. Shandilya S, Ward K, Kurz M, Najarian K. Non-linear dynamical signal characterization for prediction of defibrillation success through machine learning. *BMC Med Inform Decis Mak* 2012;12:116.
177. Nakagawa Y, Sato Y, Kojima T, et al. Electrical defibrillation outcome prediction by waveform analysis of ventricular fibrillation in cardiac arrest out of hospital patients. *Tokai J Exp Clin Med* 2012;37:1–5.
178. Lin LY, Lo MT, Ko PC, et al. Detrended fluctuation analysis predicts successful defibrillation for out-of-hospital ventricular fibrillation cardiac arrest. *Resuscitation* 2010;81:297–301.
179. Balderston JR, Gertz ZM, Ellenbogen KA, Schaaf KP, Ornato JP. Association between ventricular fibrillation amplitude immediately prior to defibrillation and defibrillation success in out-of-hospital cardiac arrest. *Am Heart J* 2018;201:72–6.
180. Agerskov M, Hansen MB, Nielsen AM, Moller TP, Wissenberg M, Rasmussen LS. Return of spontaneous circulation and long-term survival according to feedback provided by automated external defibrillators. *Acta Anaesthesiol Scand* 2017;61:1345–53.
181. Coult J, Kwok H, Sherman L, Blackwood J, Kudenchuk PJ, Rea TD. Ventricular fibrillation waveform measures combined with prior shock outcome predict defibrillation success during cardiopulmonary resuscitation. *J Electrocardiol* 2018;51:99–106.
182. Hulleman M, Salcido DD, Menegazzi JJ, et al. Predictive value of amplitude spectrum area of ventricular fibrillation waveform in patients with acute or previous myocardial infarction in out-of-hospital cardiac arrest. *Resuscitation* 2017;120:125–31.
183. Jin D, Dai C, Gong Y, et al. Does the choice of definition for defibrillation and CPR waveform impact the predictability of ventricular fibrillation waveform analysis? *Resuscitation* 2017;111:48–54.
184. Hidano D, Coult J, Blackwood J, et al. Ventricular fibrillation waveform measures and the etiology of cardiac arrest. *Resuscitation* 2016;109:71–5.
185. Coult J, Sherman L, Kwok H, Blackwood J, Kudenchuk PJ, Rea TD. Short ECG segments predict defibrillation outcome using quantitative waveform measures. *Resuscitation* 2016;109:16–20.
186. Indik JH, Conover Z, McGovern M, et al. Association of amplitude spectral area of the ventricular fibrillation waveform with survival of out-of-hospital ventricular fibrillation cardiac arrest. *J Am Coll Cardiol* 2014;64:1362–9.
187. Howe A, Escalona OJ, Di Maio R, et al. A support vector machine for predicting defibrillation outcomes from waveform metrics. *Resuscitation* 2014;85:343–9.
188. Wu X, Bisera J, Tang W. Signal integral for optimizing the timing of defibrillation. *Resuscitation* 2013;84:1704–7.
189. Hall M, Phelps R, Fahrenbruch C, Sherman L, Blackwood J, Rea TD. Myocardial substrate in secondary ventricular fibrillation: insights from quantitative waveform measures. *Prehosp Emerg Care* 2011;15:388–92.
190. Foomany FH, Umapathy K, Sugavaneswaran L, et al. Wavelet-based markers of ventricular fibrillation in optimizing human cardiac resuscitation. *Annu Int Conf IEEE Eng Med Biol Soc* 2010;2010:2001–4.
191. Endoh H, Hida S, Oohashi S, Hayashi Y, Kinoshita H, Honda T. Prompt prediction of successful defibrillation from 1-s ventricular fibrillation waveform in patients with out-of-hospital sudden cardiac arrest. *Journal of Anesthesia* 2011;25:34–41.
192. Kerber RE. External defibrillation: new technologies. *Ann Emerg Med* 1984;13:794–7.
193. Joglar JA, Kessler DJ, Welch PJ, et al. Effects of repeated electrical defibrillations on cardiac troponin I levels. *Am J Cardiol* 1999;83:270–2, A6.
194. Kerber RE, Martins JB, Kienzle MG, et al. Energy, current, and success in defibrillation and cardioversion: clinical studies using an automated impedance-based method of energy adjustment. *Circulation* 1988;77:1038–46.
195. Deakin CD, Nolan JP, Soar J, et al. European Resuscitation Council Guidelines for Resuscitation 2010 Section 4. Adult advanced life support. *Resuscitation* 2010;(81):1305–52.
196. van Alem AP, Chapman FW, Lank P, Hart AA, Koster RW. A prospective, randomised and blinded comparison of first shock success of monophasic and biphasic waveforms in out-of-hospital cardiac arrest. *Resuscitation* 2003;58:17–24.
197. Martens PR, Russell JK, Wolcke B, et al. Optimal Response to Cardiac Arrest study: defibrillation waveform effects. *Resuscitation* 2001;49:233–43.
198. Carpenter J, Rea TD, Murray JA, Kudenchuk PJ, Eisenberg MS. Defibrillation waveform and post-shock rhythm in out-of-hospital ventricular fibrillation cardiac arrest. *Resuscitation* 2003;59:189–96.
199. Gliner BE, Jorgenson DB, Poole JE, et al. Treatment of out-of-hospital cardiac arrest with a low-energy impedance-compensating biphasic waveform automatic external defibrillator. *The LIFE Investigators. Biomed Instrum Technol* 1998;32:631–44.
200. White RD, Blackwell TH, Russell JK, Snyder DE, Jorgenson DB. Transthoracic impedance does not affect defibrillation, resuscitation or survival in patients with out-of-hospital cardiac arrest treated with a non-escalating biphasic waveform defibrillator. *Resuscitation* 2005;64:63–9.
201. Morrison LJ, Henry RM, Ku V, Nolan JP, Morley P, Deakin CD. Single-shock defibrillation success in adult cardiac arrest: a systematic review. *Resuscitation* 2013;84:1480–6.
202. Stiell IG, Walker RG, Nesbitt LP, et al. BIPHASIC Trial: a randomized comparison of fixed lower versus escalating higher energy levels for defibrillation in out-of-hospital cardiac arrest. *Circulation* 2007;115:1511–7.
203. Walsh SJ, McClelland AJ, Owens CG, et al. Efficacy of distinct energy delivery protocols comparing two biphasic defibrillators for cardiac arrest. *Am J Cardiol* 2004;94:378–80.
204. Olsen JA, Brunborg C, Steinberg M, et al. Survival to hospital discharge with biphasic fixed 360 joules versus 200 escalating to 360 joules defibrillation strategies in out-of-hospital cardiac arrest of presumed cardiac etiology. *Resuscitation* 2019;136:112–8.
205. Anantharaman V, Tay SY, Manning PG, et al. A multicenter prospective randomized study comparing the efficacy of escalating higher biphasic versus low biphasic energy defibrillations in patients presenting with cardiac arrest in the in-hospital environment. *Open Access Emerg Med* 2017;9:9–17.
206. Schneider T, Martens PR, Paschen H, et al. Multicenter, randomized, controlled trial of 150-J biphasic shocks compared with 200- to 360-J monophasic shocks in the resuscitation of out-of-hospital cardiac arrest victims. Optimized Response to Cardiac Arrest (ORCA) Investigators. *Circulation* 2000;102:1780–7.

207. Higgins SL, Herre JM, Epstein AE, et al. A comparison of biphasic and monophasic shocks for external defibrillation. *Physio-Control Biphasic Investigators. Prehosp Emerg Care* 2000;4:305–13.
208. Berg RA, Samson RA, Berg MD, et al. Better outcome after pediatric defibrillation dosage than adult dosage in a swine model of pediatric ventricular fibrillation. *J Am Coll Cardiol* 2005;45:786–9.
209. Killingsworth CR, Melnick SB, Chapman FW, et al. Defibrillation threshold and cardiac responses using an external biphasic defibrillator with pediatric and adult adhesive patches in pediatric-sized piglets. *Resuscitation* 2002;55:177–85.
210. Tang W, Weil MH, Sun S, et al. The effects of biphasic waveform design on post-resuscitation myocardial function. *J Am Coll Cardiol* 2004;43:1228–35.
211. Xie J, Weil MH, Sun S, et al. High-energy defibrillation increases the severity of postresuscitation myocardial dysfunction. *Circulation* 1997;96:683–8.
212. Koster RW, Walker RG, Chapman FW. Recurrent ventricular fibrillation during advanced life support care of patients with prehospital cardiac arrest. *Resuscitation* 2008;78:252–7.
213. Walker RG, Koster RW, Sun C, et al. Defibrillation probability and impedance change between shocks during resuscitation from out-of-hospital cardiac arrest. *Resuscitation* 2009;80:773–7.
214. Hess EP, Russell JK, Liu PY, White RD. A high peak current 150-J fixed-energy defibrillation protocol treats recurrent ventricular fibrillation (VF) as effectively as initial VF. *Resuscitation* 2008;79:28–33.
215. Deakin CD, Ambler JJ. Post-shock myocardial stunning: a prospective randomised double-blind comparison of monophasic and biphasic waveforms. *Resuscitation* 2006;68:329–33.
216. Khaykin Y, Newman D, Kowalewski M, Korley V, Dorian P. Biphasic versus monophasic cardioversion in shock-resistant atrial fibrillation. *J Cardiovasc Electrophysiol* 2003;14:868–72.
217. Koster RW, Dorian P, Chapman FW, Schmitt PW, O'Grady SG, Walker RG. A randomized trial comparing monophasic and biphasic waveform shocks for external cardioversion of atrial fibrillation. *Am Heart J* 2004;147:e20.
218. Mittal S, Ayati S, Stein KM, et al. Transthoracic cardioversion of atrial fibrillation: comparison of rectilinear biphasic versus damped sine wave monophasic shocks. *Circulation* 2000;101:1282–7.
219. Kmec J. Comparison the effectiveness of damped sine wave monophasic and rectilinear biphasic shocks in patients with persistent atrial fibrillation. *Kardiologia* 2006;15:265–78.
220. Kosior DA, Szulec M, Torbicki A, Opolski G, Rabaczynski D. A decrease of enlarged left atrium following cardioversion of atrial fibrillation predicts the long-term maintenance of sinus rhythm. *Kardiologia Polska* 2005;62:428–37.
221. Hess EP, Agarwal D, Myers LA, Atkinson EJ, White RD. Performance of a rectilinear biphasic waveform in defibrillation of presenting and recurrent ventricular fibrillation: a prospective multicenter study. *Resuscitation* 2011;82:685–9.
222. Eilevstjonn J, Kramer-Johansen J, Sunde K. Shock outcome is related to prior rhythm and duration of ventricular fibrillation. *Resuscitation* 2007;75:60–7.
223. Mapp JG, Hans AJ, Darrington AM, et al. Prehospital Double Sequential Defibrillation: A Matched Case-Control Study. *Acad Emerg Med* 2019;26:994–1001.
224. Ross EM, Redman TT, Harper SA, Mapp JG, Wampler DA, Miramontes DA. Dual defibrillation in out-of-hospital cardiac arrest: A retrospective cohort analysis. *Resuscitation* 2016;106:14–7.
225. Cortez E, Krebs W, Davis J, Keseg DP, Panchal AR. Use of double sequential external defibrillation for refractory ventricular fibrillation during out-of-hospital cardiac arrest. *Resuscitation* 2016;108:82–6.
226. Beck LR, Ostermayer DG, Ponce JN, Srinivasan S, Wang HE. Effectiveness of Prehospital Dual Sequential Defibrillation for Refractory Ventricular Fibrillation and Ventricular Tachycardia Cardiac Arrest. *Prehosp Emerg Care* 2019;23:597–602.
227. Emmerson AC, Whitbread M, Fothergill RT. Double sequential defibrillation therapy for out-of-hospital cardiac arrests: The London experience. *Resuscitation* 2017;117:97–101.
228. Cabanas JG, Myers JB, Williams JG, De Maio VJ, Bachman MW. Double Sequential External Defibrillation in Out-of-Hospital Refractory Ventricular Fibrillation: A Report of Ten Cases. *Prehosp Emerg Care* 2015;19:126–30.
229. Cheskes S, Wudwud A, Turner L, et al. The impact of double sequential external defibrillation on termination of refractory ventricular fibrillation during out-of-hospital cardiac arrest. *Resuscitation* 2019;139:275–81.
230. Cheskes S, Dorian P, Feldman M, et al. Double sequential external defibrillation for refractory ventricular fibrillation: The DOSE VF pilot randomized controlled trial. *Resuscitation* 2020;150:178–84.
231. Deakin CD, Morley P, Soar J, Drennan IR. Double (dual) sequential defibrillation for refractory ventricular fibrillation cardiac arrest: A systematic review. *Resuscitation* 2020;155:24–31.
232. Friedman DJ, Parzynski CS, Varosy PD, et al. Trends and In-Hospital Outcomes Associated With Adoption of the Subcutaneous Implantable Cardioverter Defibrillator in the United States. *JAMA Cardiol* 2016;1:900–11.
233. Knops RE, Olde Nordkamp LRA, Delnoy PHM, et al. Subcutaneous or Transvenous Defibrillator Therapy. *N Engl J Med* 2020;383:526–36.
234. Stockwell B, Bellis G, Morton G, et al. Electrical injury during "hands on" defibrillation-A potential risk of internal cardioverter defibrillators? *Resuscitation* 2009;80:832–4.
235. Peran D, Cmurek PC, Pekara J. Bystander hit by leakage current from S-ICD. *Resuscitation* 2019;138:297–8.
236. Petley GW, Albon B, Banks P, Roberts PR, Deakin CD. Leakage current from transvenous and subcutaneous implantable cardioverter defibrillators (ICDs): A risk to the rescuer? *Resuscitation* 2019;137:148–53.
237. Monsieurs KG, Conraads VM, Goethals MP, Snoeck JP, Bossaert LL. Semi-automatic external defibrillation and implanted cardiac pacemakers: understanding the interactions during resuscitation. *Resuscitation* 1995;30:127–31.
238. Bengier JR, Kirby K, Black S, et al. Effect of a Strategy of a Supraglottic Airway Device vs Tracheal Intubation During Out-of-Hospital Cardiac Arrest on Functional Outcome: The AIRWAYS-2 Randomized Clinical Trial. *JAMA* 2018;320:779–91.
239. Jabre P, Penaloza A, Pinero D, et al. Effect of Bag-Mask Ventilation vs Endotracheal Intubation During Cardiopulmonary Resuscitation on Neurological Outcome After Out-of-Hospital Cardiorespiratory Arrest: A Randomized Clinical Trial. *JAMA* 2018;319:779–87.
240. Wang HE, Schmicker RH, Daya MR, et al. Effect of a Strategy of Initial Laryngeal Tube Insertion vs Endotracheal Intubation on 72-Hour Survival in Adults With Out-of-Hospital Cardiac Arrest: A Randomized Clinical Trial. *JAMA* 2018;320:769–78.
241. Granfeldt A, Avis SR, Nicholson TC, et al. Advanced airway management during adult cardiac arrest: A systematic review. *Resuscitation* 2019;139:133–43.
242. Soar J, Maconochie I, Wyckoff MH, et al. 2019 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2019;145:95–150.
243. Andersen LW, Granfeldt A, Callaway CW, et al. Association Between Tracheal Intubation During Adult In-Hospital Cardiac Arrest and Survival. *JAMA* 2017;317:494–506.
244. Soar J, Maconochie I, Wyckoff MH, et al. 2019 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations: Summary From the Basic Life Support; Advanced Life Support; Pediatric Life Support; Neonatal Life Support; Education, Implementation, and Teams; and First Aid Task Forces. *Circulation* 2019;140:e826–e80.
245. Voss S, Rhys M, Coates D, et al. How do paramedics manage the airway during out of hospital cardiac arrest? *Resuscitation* 2014;85:1662–6.
246. Nolan JP. European Resuscitation Council and European Society of Intensive Care Medicine Guidelines for Post-resuscitation Care 2020. *Resuscitation* 2021.

247. Olasveengen TM, Semeraro F, Ristagno G, et al. European Resuscitation Council Guidelines for Basic Life Support. *Resuscitation* 2021.
248. Couper K, Abu Hassan A, Ohri V, et al. Removal of foreign body airway obstruction: A systematic review of interventions. *Resuscitation* 2020;156:174–81.
249. Nichol G, Leroux B, Wang H, et al. Trial of Continuous or Interrupted Chest Compressions during CPR. *N Engl J Med* 2015;373:2203–14.
250. Olasveengen TM, de Caen AR, Mancini ME, et al. 2017 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations Summary. *Resuscitation* 2017;121:201–14.
251. Vissers G, Soar J, Monsieurs KG. Ventilation rate in adults with a tracheal tube during cardiopulmonary resuscitation: A systematic review. *Resuscitation* 2017;119:5–12.
252. Deakin CD, O'Neill JF, Tabor T. Does compression-only cardiopulmonary resuscitation generate adequate passive ventilation during cardiac arrest? *Resuscitation* 2007;75:53–9.
253. Saissy JM, Boussignac G, Cheptel E, et al. Efficacy of continuous insufflation of oxygen combined with active cardiac compression-decompression during out-of-hospital cardiorespiratory arrest. *Anesthesiology* 2000;92:1523–30.
254. Bertrand C, Hemery F, Carli P, et al. Constant flow insufflation of oxygen as the sole mode of ventilation during out-of-hospital cardiac arrest. *Intensive Care Med* 2006;32:843–51.
255. Bobrow BJ, Ewy GA, Clark L, et al. Passive oxygen insufflation is superior to bag-valve-mask ventilation for witnessed ventricular fibrillation out-of-hospital cardiac arrest. *Ann Emerg Med* 2009;54:656–62 e1.
256. Grmec S. Comparison of three different methods to confirm tracheal tube placement in emergency intubation. *Intensive Care Med* 2002;28:701–4.
257. Lyon RM, Ferris JD, Young DM, McKeown DW, Oglesby AJ, Robertson C. Field intubation of cardiac arrest patients: a dying art? *Emergency Medicine* 2010;27:321–3.
258. Jones JH, Murphy MP, Dickson RL, Somerville GG, Brizendine EJ. Emergency physician-verified out-of-hospital intubation: miss rates by paramedics. *Acad Emerg Med* 2004;11:707–9.
259. Pelucio M, Halligan L, Dhindsa H. Out-of-hospital experience with the syringe esophageal detector device. *Acad Emerg Med* 1997;4:563–8.
260. Jemmett ME, Kendal KM, Foure MW, Burton JH. Unrecognized misplacement of endotracheal tubes in a mixed urban to rural emergency medical services setting. *Acad Emerg Med* 2003;10:961–5.
261. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. *Ann Emerg Med* 2001;37:32–7.
262. Wang HE, Simeone SJ, Weaver MD, Callaway CW. Interruptions in cardiopulmonary resuscitation from paramedic endotracheal intubation. *Ann Emerg Med* 2009;54: 645–52 e1.
263. Garza AG, Gratton MC, Coontz D, Noble E, Ma OJ. Effect of paramedic experience on orotracheal intubation success rates. *J Emerg Med* 2003;25:251–6.
264. Bradley JS, Billows GL, Olinger ML, Boha SP, Cordell WH, Nelson DR. Prehospital oral endotracheal intubation by rural basic emergency medical technicians. *Ann Emerg Med* 1998;32:26–32.
265. Sayre MR, Sakles JC, Mistler AF, Evans JL, Kramer AT, Pancioli AM. Field trial of endotracheal intubation by basic EMTs. *Ann Emerg Med* 1998;31:228–33.
266. Bernhard M, Mohr S, Weigand MA, Martin E, Walther A. Developing the skill of endotracheal intubation: implication for emergency medicine. *Acta Anaesthesiol Scand* 2012;56:164–71.
267. Cook TM, Boniface NJ, Sellar C, et al. Universal videolaryngoscopy: a structured approach to conversion to videolaryngoscopy for all intubations in an anaesthetic and intensive care department. *Br J Anaesth* 2018;120:173–80.
268. Goto Y, Goto T, Hagiwara Y, et al. Techniques and outcomes of emergency airway management in Japan: An analysis of two multicentre prospective observational studies, 2010–2016. *Resuscitation* 2017;114:14–20.
269. Lee DH, Han M, An JY, et al. Video laryngoscopy versus direct laryngoscopy for tracheal intubation during in-hospital cardiopulmonary resuscitation. *Resuscitation* 2015;89:195–9.
270. Park SO, Kim JW, Na JH, et al. Video laryngoscopy improves the first-attempt success in endotracheal intubation during cardiopulmonary resuscitation among novice physicians. *Resuscitation* 2015;89:188–94.
271. Jiang J, Kang N, Li B, Wu AS, Xue FS. Comparison of adverse events between video and direct laryngoscopes for tracheal intubations in emergency department and ICU patients—a systematic review and meta-analysis. *Scand J Trauma Resusc Emerg Med* 2020;28:10.
272. Kim JW, Park SO, Lee KR, et al. Video laryngoscopy vs. direct laryngoscopy: Which should be chosen for endotracheal intubation during cardiopulmonary resuscitation? A prospective randomized controlled study of experienced intubators. *Resuscitation* 2016;105:196–202.
273. Jiang J, Ma D, Li B, Yue Y, Xue F. Video laryngoscopy does not improve the intubation outcomes in emergency and critical patients—a systematic review and meta-analysis of randomized controlled trials. *Crit Care* 2017;21:288.
274. Deakin CD, Morrison LJ, Morley PT, et al. Part 8: Advanced life support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Resuscitation* 2010;81(Suppl 1):e93–e174.
275. Soar J, Callaway CW, Aibiki M, et al. Part 4: Advanced life support: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Resuscitation* 2015;95:e71–120.
276. Paiva EF, Paxton JH, O'Neil BJ. The use of end-tidal carbon dioxide (ETCO₂) measurement to guide management of cardiac arrest: A systematic review. *Resuscitation* 2018;123:1–7.
277. Grmec S, Mally S. Prehospital determination of tracheal tube placement in severe head injury. *Emergency Medicine* 2004;21:518–20.
278. Knapp S, Kofler J, Stoiser B, et al. The assessment of four different methods to verify tracheal tube placement in the critical care setting. *Anesth Analg* 1999;88:766–70.
279. Takeda T, Tanigawa K, Tanaka H, Hayashi Y, Goto E, Tanaka K. The assessment of three methods to verify tracheal tube placement in the emergency setting. *Resuscitation* 2003;56:153–7.
280. Cook TM, Harrop-Griffiths AW, Whitaker DK, McNarry AF, Patel A, McGuire B. The 'No Trace=Wrong Place' campaign. *Br J Anaesth* 2019;122:e68–e9.
281. Salem MR, Khorasani A, Zeidan A, Crystal GJ. Cricoid Pressure Controversies: Narrative Review. *Anesthesiology* 2017;126:738–52.
282. Higgs A, McGrath BA, Goddard C, et al. Guidelines for the management of tracheal intubation in critically ill adults. *Br J Anaesth* 2018;120:323–52.
283. Granfeldt A, Avis SR, Lind PC, et al. Intravenous vs. intraosseous administration of drugs during cardiac arrest: A systematic review. *Resuscitation* 2020;149:150–7.
284. Feinstein BA, Stubbs BA, Rea T, Kudenchuk PJ. Intraosseous compared to intravenous drug resuscitation in out-of-hospital cardiac arrest. *Resuscitation* 2017;117:91–6.
285. Kawano T, Grunau B, Scheuermeyer FX, et al. Intraosseous Vascular Access Is Associated With Lower Survival and Neurologic Recovery Among Patients With Out-of-Hospital Cardiac Arrest. *Ann Emerg Med* 2018;71:588–96.
286. Mody P, Brown SP, Kudenchuk PJ, et al. Intraosseous versus intravenous access in patients with out-of-hospital cardiac arrest: Insights from the resuscitation outcomes consortium continuous chest compression trial. *Resuscitation* 2019;134:69–75.

287. Nolan JP, Deakin CD, Ji C, et al. Intraosseous versus intravenous administration of adrenaline in patients with out-of-hospital cardiac arrest: a secondary analysis of the PARAMEDIC2 placebo-controlled trial. *Intensive Care Med* 2020;46:954–62.
288. Daya MR, Leroux BG, Dorian P, et al. Survival After Intravenous Versus Intraosseous Amiodarone, Lidocaine, or Placebo in Out-of-Hospital Shock-Refractory Cardiac Arrest. *Circulation* 2020;141:188–98.
289. Perkins GD, Ji C, Deakin CD, et al. A Randomized Trial of Epinephrine in Out-of-Hospital Cardiac Arrest. *N Engl J Med* 2018;379:711–21.
290. Finn J, Jacobs I, Williams TA, Gates S, Perkins GD. Adrenaline and vasopressin for cardiac arrest. *Cochrane Database Syst Rev* 2019;1:CD003179.
291. Holmberg MJ, Issa MS, Moskowitz A, et al. Vasopressors during adult cardiac arrest: A systematic review and meta-analysis. *Resuscitation* 2019;139:106–21.
292. Perkins GD, Kenna C, Ji C, et al. The effects of adrenaline in out of hospital cardiac arrest with shockable and non-shockable rhythms: Findings from the PACA and PARAMEDIC-2 randomised controlled trials. *Resuscitation* 2019;140:55–63.
293. Perkins GD, Kenna C, Ji C, et al. The influence of time to adrenaline administration in the Paramedic 2 randomised controlled trial. *Intensive Care Med* 2020;46:426–36.
294. Soar J, Donnino MW, Maconochie I, et al. 2018 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations Summary. *Resuscitation* 2018;133:194–206.
295. Ali MU, Fitzpatrick-Lewis D, Kenny M, et al. Effectiveness of antiarrhythmic drugs for shockable cardiac arrest: A systematic review. *Resuscitation* 2018;132:63–72.
296. Kudenchuk PJ, Brown SP, Daya M, et al. Amiodarone, Lidocaine, or Placebo in Out-of-Hospital Cardiac Arrest. *N Engl J Med* 2016;374:1711–22.
297. Soar J, Perkins GD, Maconochie I, et al. European Resuscitation Council Guidelines for Resuscitation: 2018 Update - Antiarrhythmic drugs for cardiac arrest. *Resuscitation* 2019;134:99–103.
298. Bottiger BW, Arntz HR, Chamberlain DA, et al. Thrombolysis during resuscitation for out-of-hospital cardiac arrest. *N Engl J Med* 2008;359:2651–62.
299. Yousuf T, Brinton T, Ahmed K, et al. Tissue Plasminogen Activator Use in Cardiac Arrest Secondary to Fulminant Pulmonary Embolism. *J Clin Med Res* 2016;8:190–5.
300. Kurkciyan I, Meron G, Sterz F, et al. Pulmonary embolism as a cause of cardiac arrest: presentation and outcome. *Arch Intern Med* 2000;160:1529–35.
301. Janata K, Holzer M, Kurkciyan I, et al. Major bleeding complications in cardiopulmonary resuscitation: the place of thrombolytic therapy in cardiac arrest due to massive pulmonary embolism. *Resuscitation* 2003;57:49–55.
302. Javaudin F, Lascarrou JB, Le Bastard Q, et al. Thrombolysis During Resuscitation for Out-of-Hospital Cardiac Arrest Caused by Pulmonary Embolism Increases 30-Day Survival: Findings From the French National Cardiac Arrest Registry. *Chest* 2019;156:1167–75.
303. Böttiger BW, Böhler H, Bach A, Motsch J, Martin E. Bolus injection of thrombolytic agents during cardiopulmonary resuscitation for massive pulmonary embolism. *Resuscitation* 1994;28:45–54.
304. Wu JP, Gu DY, Wang S, Zhang ZJ, Zhou JC, Zhang RF. Good neurological recovery after rescue thrombolysis of presumed pulmonary embolism despite prior 100 minutes CPR. *J Thorac Dis* 2014;6:E289–93.
305. Summers K, Schultheis J, Raiff D, Dahhan T. Evaluation of Rescue Thrombolysis in Cardiac Arrest Secondary to Suspected or Confirmed Pulmonary Embolism. *Ann Pharmacother* 2019;53:711–5.
306. Bernard SA, Smith K, Finn J, et al. Induction of Therapeutic Hypothermia During Out-of-Hospital Cardiac Arrest Using a Rapid Infusion of Cold Saline: The RINSE Trial (Rapid Infusion of Cold Normal Saline). *Circulation* 2016;134:797–805.
307. Kim F, Nichol G, Maynard C, et al. Effect of prehospital induction of mild hypothermia on survival and neurological status among adults with cardiac arrest: a randomized clinical trial. *JAMA* 2014;311:45–52.
308. Maynard C, Longstreth Jr. WT, Nichol G, et al. Effect of prehospital induction of mild hypothermia on 3-month neurological status and 1-year survival among adults with cardiac arrest: long-term follow-up of a randomized, clinical trial. *Journal of the American Heart Association* 2015;4:e001693.
309. Soar J, Foster J, Breitkreutz R. Fluid infusion during CPR and after ROSC—is it safe? *Resuscitation* 2009;80:1221–2.
310. Sandroni C, De Santis P, D'Arrigo S. Capnography during cardiac arrest. *Resuscitation* 2018;132:73–7.
311. Gutierrez JJ, Ruiz JM, Ruiz de Gauna S, et al. Modeling the impact of ventilations on the capnogram in out-of-hospital cardiac arrest. *PLoS One* 2020;15:e0228395.
312. Hamrick JL, Hamrick JLT, Lee JK, Lee BH, Koehler RC, Shaffner DH. Efficacy of chest compressions directed by end-tidal CO₂ feedback in a pediatric resuscitation model of basic life support. *Journal of the American Heart Association* 2014;3:e000450.
313. Sheak KR, Wiebe DJ, Leary M, et al. Quantitative relationship between end-tidal carbon dioxide and CPR quality during both in-hospital and out-of-hospital cardiac arrest. *Resuscitation* 2015;89:149–54.
314. Garnett AR, Ornato JP, Gonzalez ER, Johnson EB. End-tidal carbon dioxide monitoring during cardiopulmonary resuscitation. *JAMA* 1987;257:512–5.
315. Pokorna M, Necas E, Kratochvil J, Skripsky R, Andriks M, Franek O. A sudden increase in partial pressure end-tidal carbon dioxide (P(ET)CO₂) at the moment of return of spontaneous circulation. *J Emerg Med* 2010;38:614–21.
316. Lui Ct, Poon Km, Tsui KI. Abrupt rise of end tidal carbon dioxide level was a specific but non-sensitive marker of return of spontaneous circulation in patient with out-of-hospital cardiac arrest. *Resuscitation* 2016;104:53–8.
317. Sandroni C, Ristagno G. End-tidal CO₂ to detect recovery of spontaneous circulation during cardiopulmonary resuscitation: We are not ready yet. *Resuscitation* 2016;104:A5–6.
318. Levine RL, Wayne MA, Miller CC. End-tidal carbon dioxide and outcome of out-of-hospital cardiac arrest. *N Engl J Med* 1997;337:301–6.
319. Sutton RM, French B, Meaney PA, et al. Physiologic monitoring of CPR quality during adult cardiac arrest: A propensity-matched cohort study. *Resuscitation* 2016;106:76–82.
320. Conseil francais de reanimation c, Societe francaise d'anesthesie et de r, Societe francaise de c, et al. Guidelines for indications for the use of extracorporeal life support in refractory cardiac arrest. French Ministry of Health. *Ann Fr Anesth Reanim* 2009;28:182–90.
321. Kolar M, Krizmaric M, Klemen P, Grmec S. Partial pressure of end-tidal carbon dioxide successfully predicts cardiopulmonary resuscitation in the field: a prospective observational study. *Crit Care* 2008;12:R115.
322. Poppe M, Stratil P, Clodi C, et al. Initial end-tidal carbon dioxide as a predictive factor for return of spontaneous circulation in nonshockable out-of-hospital cardiac arrest patients: A retrospective observational study. *European Journal of Anaesthesiology* 2019;36:524–30.
323. Grmec S, Lah K, Tusek-Bunc K. Difference in end-tidal CO₂ between asphyxia cardiac arrest and ventricular fibrillation/pulseless ventricular tachycardia cardiac arrest in the prehospital setting. *Crit Care* 2003;7:R139-R44.
324. Heradstveit BE, Sunde K, Sunde GA, Wentzel-Larsen T, Heltne JK. Factors complicating interpretation of capnography during advanced life support in cardiac arrest—A clinical retrospective study in 575 patients. *Resuscitation* 2012;83:813–8.

325. Gazmuri RJ, Ayoub IM, Radhakrishnan J, Motl J, Upadhyaya MP. Clinically plausible hyperventilation does not exert adverse hemodynamic effects during CPR but markedly reduces end-tidal PCO₂. *Resuscitation* 2012;83:259–64.
326. Grieco DL, L JB, Drouet A, et al. Intrathoracic Airway Closure Impacts CO₂ Signal and Delivered Ventilation during Cardiopulmonary Resuscitation. *Am J Respir Crit Care Med* 2019;199:728–37.
327. Callaham M, Barton C, Matthay M. Effect of epinephrine on the ability of end-tidal carbon dioxide readings to predict initial resuscitation from cardiac arrest. *Crit Care Med* 1992;20:337–43.
328. Hardig BM, Gotberg M, Rundgren M, et al. Physiologic effect of repeated adrenaline (epinephrine) doses during cardiopulmonary resuscitation in the cath lab setting: A randomised porcine study. *Resuscitation* 2016;101:77–83.
329. Brinkrolf P, Borowski M, Metelmann C, Lukas RP, Pidde-Kullenberg L, Bohn A. Predicting ROSC in out-of-hospital cardiac arrest using expiratory carbon dioxide concentration: Is trend-detection instead of absolute threshold values the key? *Resuscitation* 2018;122:19–24.
330. Reynolds JC, Issa MS, T CN, et al. Prognostication with point-of-care echocardiography during cardiac arrest: A systematic review. *Resuscitation* 2020;152:56–68.
331. Huis In't Veld MA, Allison MG, Bostick DS, et al. Ultrasound use during cardiopulmonary resuscitation is associated with delays in chest compressions. *Resuscitation* 2017;119:95–98.
332. Clattenburg EJ, Wroe P, Brown S, et al. Point-of-care ultrasound use in patients with cardiac arrest is associated prolonged cardiopulmonary resuscitation pauses: A prospective cohort study. *Resuscitation* 2018;122:65–8.
333. Berg RA, Sorrell VL, Kern KB, et al. Magnetic resonance imaging during untreated ventricular fibrillation reveals prompt right ventricular overdistention without left ventricular volume loss. *Circulation* 2005;111:1136–40.
334. Querrelou E, Leyral J, Brun C, et al. [In and out-of-hospital cardiac arrest and echography: a review]. *Ann Fr Anesth Reanim* 2009;28:769–78.
335. Blanco P, Volpicelli G. Common pitfalls in point-of-care ultrasound: a practical guide for emergency and critical care physicians. *Crit Ultrasound J* 2016;8:15.
336. Aagaard R, Granfeldt A, Botker MT, Mygind-Klausen T, Kirkegaard H, Lofgren B. The Right Ventricle Is Dilated During Resuscitation From Cardiac Arrest Caused by Hypovolemia: A Porcine Ultrasound Study. *Crit Care Med* 2017;45:e963–e70.
337. Teran F. Resuscitative Cardiopulmonary Ultrasound and Transesophageal Echocardiography in the Emergency Department. *Emerg Med Clin North Am* 2019;37:409–30.
338. Perkins GD, Lall R, Quinn T, et al. Mechanical versus manual chest compression for out-of-hospital cardiac arrest (PARAMEDIC): a pragmatic, cluster randomised controlled trial. *Lancet* 2015;385:947–55.
339. Rubertsson S, Lindgren E, Smekal D, et al. Mechanical chest compressions and simultaneous defibrillation vs conventional cardiopulmonary resuscitation in out-of-hospital cardiac arrest: the LINC randomized trial. *JAMA* 2014;311:53–61.
340. Hallstrom A, Rea TD, Sayre MR, et al. Manual chest compression vs use of an automated chest compression device during resuscitation following out-of-hospital cardiac arrest: a randomized trial. *JAMA* 2006;295:2620–8.
341. Wik L, Olsen JA, Persse D, et al. Manual vs. integrated automatic load-distributing band CPR with equal survival after out of hospital cardiac arrest. The randomized CIRC trial. *Resuscitation* 2014;85:741–8.
342. Lu XG, Kang X, Gong DB. [The clinical efficacy of Thumper modal 1007 cardiopulmonary resuscitation: a prospective randomized control trial]. *Zhongguo Wei Zhong Bing Ji Jiu Yi Xue* 2010;22:496–7.
343. Smekal D, Johansson J, Huzevka T, Rubertsson S. A pilot study of mechanical chest compressions with the LUCAS device in cardiopulmonary resuscitation. *Resuscitation* 2011;82:702–6.
344. Dickinson ET, Verdile VP, Schneider RM, Salluzzo RF. Effectiveness of mechanical versus manual chest compressions in out-of-hospital cardiac arrest resuscitation: a pilot study. *Am J Emerg Med* 1998;16:289–92.
345. Halperin HR, Tsitlik JE, Gelfand M, et al. A preliminary study of cardiopulmonary resuscitation by circumferential compression of the chest with use of a pneumatic vest. *N Engl J Med* 1993;329:762–8.
346. Koster RW, Beenen LF, van der Boom EB, et al. Safety of mechanical chest compression devices AutoPulse and LUCAS in cardiac arrest: a randomized clinical trial for non-inferiority. *Eur Heart J* 2017;38:3006–13.
347. Gao C, Chen Y, Peng H, Chen Y, Zhuang Y, Zhou S. Clinical evaluation of the AutoPulse automated chest compression device for out-of-hospital cardiac arrest in the northern district of Shanghai. China. *Arch Med Sci* 2016;12:563–70.
348. Liu M, Shuai Z, Ai J, et al. Mechanical chest compression with LUCAS device does not improve clinical outcome in out-of-hospital cardiac arrest patients: A systematic review and meta-analysis. *Medicine (Baltimore)* 2019;98:e17550.
349. Zhu N, Chen Q, Jiang Z, et al. A meta-analysis of the resuscitative effects of mechanical and manual chest compression in out-of-hospital cardiac arrest patients. *Crit Care* 2019;23:100.
350. Wang PL, Brooks SC. Mechanical versus manual chest compressions for cardiac arrest. *Cochrane Database Syst Rev* 2018;8:CD007260.
351. Gates S, Quinn T, Deakin CD, Blair L, Couper K, Perkins GD. Mechanical chest compression for out of hospital cardiac arrest: Systematic review and meta-analysis. *Resuscitation* 2015;94:91–7.
352. Couper K, Yeung J, Nicholson T, Quinn T, Lall R, Perkins GD. Mechanical chest compression devices at in-hospital cardiac arrest: A systematic review and meta-analysis. *Resuscitation* 2016;103:24–31.
353. Khan SU, Lone AN, Talluri S, Khan MZ, Khan MZU, Kaluski E. Efficacy and safety of mechanical versus manual compression in cardiac arrest - A Bayesian network meta-analysis. *Resuscitation* 2018;130:182–8.
354. Li H, Wang D, Yu Y, Zhao X, Jing X. Mechanical versus manual chest compressions for cardiac arrest: a systematic review and meta-analysis. *Scand J Trauma Resusc Emerg Med* 2016;24:10.
355. Poole K, Couper K, Smyth MA, Yeung J, Perkins GD. Mechanical CPR: Who? When? How?. *Crit Care* 2018;22:140.
356. Brouwer TF, Walker RG, Chapman FW, Koster RW. Association Between Chest Compression Interruptions and Clinical Outcomes of Ventricular Fibrillation Out-of-Hospital Cardiac Arrest. *Circulation* 2015;132:1030–7.
357. Yost D, Phillips RH, Gonzales L, et al. Assessment of CPR interruptions from transthoracic impedance during use of the LUCAS mechanical chest compression system. *Resuscitation* 2012;83:961–5.
358. Levy M, Yost D, Walker RG, Scheunemann E, Mendive SR. A quality improvement initiative to optimize use of a mechanical chest compression device within a high-performance CPR approach to out-of-hospital cardiac arrest resuscitation. *Resuscitation* 2015;92:32–7.
359. Esibov A, Banville I, Chapman FW, Boomars R, Box M, Rubertsson S. Mechanical chest compressions improved aspects of CPR in the LINC trial. *Resuscitation* 2015;91:116–21.
360. Couper K, Velho RM, Quinn T, et al. Training approaches for the deployment of a mechanical chest compression device: a randomised controlled manikin study. *BMJ Open* 2018;8:e019009.
361. Richardson ASC, Tonna JE, Nanjaya V, et al. Extracorporeal cardiopulmonary resuscitation in adults. Interim guideline consensus statement from the extracorporeal life support organization. *ASAIO J* 2021;67(3):221–8, doi:http://dx.doi.org/10.1097/MAT.0000000000001344.
362. Richardson AS, Schmidt M, Bailey M, Pellegrino VA, Rycus PT, Pilcher DV. ECMO Cardio-Pulmonary Resuscitation (ECPR),

- trends in survival from an international multicentre cohort study over 12-years. *Resuscitation* 2017;112:34–40.
363. Hutin A, Abu-Habsa M, Burns B, et al. Early ECPR for out-of-hospital cardiac arrest: Best practice in 2018. *Resuscitation* 2018;130:44–8.
 364. Swol J, Belohlavek J, Brodie D, et al. Extracorporeal life support in the emergency department: A narrative review for the emergency physician. *Resuscitation* 2018;133:108–17.
 365. Dennis M, Lal S, Forrest P, et al. In-Depth Extracorporeal Cardiopulmonary Resuscitation in Adult Out-of-Hospital Cardiac Arrest. *Journal of the American Heart Association* 2020;9:e016521.
 366. Holmberg MJ, Geri G, Wiberg S, et al. Extracorporeal cardiopulmonary resuscitation for cardiac arrest: A systematic review. *Resuscitation* 2018;131:91–100.
 367. Yannopoulos D, Bartos J, Raveendran G, et al. Advanced reperfusion strategies for patients with out-of-hospital cardiac arrest and refractory ventricular fibrillation (ARREST): a phase 2, single centre, open-label, randomised controlled trial. *Lancet* 2020;396:1807–16.
 368. Guglin M, Zucker MJ, Bazan VM, et al. Venoarterial ECMO for Adults: JACC Scientific Expert Panel. *J Am Coll Cardiol* 2019;73:698–716.
 369. Debaty G, Babaz V, Durand M, et al. Prognostic factors for extracorporeal cardiopulmonary resuscitation recipients following out-of-hospital refractory cardiac arrest. A systematic review and meta-analysis. *Resuscitation* 2017;112:1–10.
 370. Yu HY, Wang CH, Chi NH, et al. Effect of interplay between age and low-flow duration on neurologic outcomes of extracorporeal cardiopulmonary resuscitation. *Intensive Care Med* 2019;45:44–54.
 371. Dennis M, Zmudzki F, Burns B, et al. Cost effectiveness and quality of life analysis of extracorporeal cardiopulmonary resuscitation (ECPR) for refractory cardiac arrest. *Resuscitation* 2019;139:49–56.
 372. Kawashima T, Uehara H, Miyagi N, et al. Impact of first documented rhythm on cost-effectiveness of extracorporeal cardiopulmonary resuscitation. *Resuscitation* 2019;140:74–80.
 373. Bharmal MI, Venturini JM, Chua RFM, et al. Cost-utility of extracorporeal cardiopulmonary resuscitation in patients with cardiac arrest. *Resuscitation* 2019;136:126–30.
 374. Page RL, Joglar JA, Caldwell MA, et al. 2015 ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol* 2016;67:e27–e115.
 375. Kirchhof P, Benussi S, Kotecha D, et al. 2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. *Eur Heart J* 2016;37:2893–962.
 376. January CT, Wann LS, Calkins H, et al. 2019 AHA/ACC/HRS Focused Update of the 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol* 2019;74:104–32.
 377. Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol* 2019;74:e51–e156.
 378. Hindricks G, Potpara T, Dagres N, et al. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association of Cardio-Thoracic Surgery (EACTS). *Eur Heart J* 2020.
 379. Ortiz M, Martin A, Arribas F, et al. Randomized comparison of intravenous procainamide vs. intravenous amiodarone for the acute treatment of tolerated wide QRS tachycardia: the PROCAMIO study. *Eur Heart J* 2017;38:1329–35.
 380. Bernheim A, Fatio R, Kiowski W, Weilenmann D, Rickli H, Rocca HP. Atropine often results in complete atrioventricular block or sinus arrest after cardiac transplantation: an unpredictable and dose-independent phenomenon. *Transplantation* 2004;77:1181–5.
 381. Roth A, Elkayam I, Shapira I, et al. Effectiveness of prehospital synchronous direct-current cardioversion for supraventricular tachyarrhythmias causing unstable hemodynamic states. *Am J Cardiol* 2003;91:489–91.
 382. Wittwer MR, Rajendran S, Kealley J, Arstall MA. A South Australian registry of biphasic cardioversions of atrial arrhythmias: efficacy and predictors of success. *Heart Lung Circ* 2015;24:342–7.
 383. Reisinger J, Gstrein C, Winter T, et al. Optimization of initial energy for cardioversion of atrial tachyarrhythmias with biphasic shocks. *Am J Emerg Med* 2010;28:159–65.
 384. Lown B. Electrical reversion of cardiac arrhythmias. *Br Heart J* 1967;29:469–89.
 385. Deakin CD, Connelly S, Wharton R, Yuen HM. A comparison of rectilinear and truncated exponential biphasic waveforms in elective cardioversion of atrial fibrillation: a prospective randomized controlled trial. *Resuscitation* 2013;84:286–91.
 386. Schmidt AS, Lauridsen KG, Torp P, Bach LF, Rickers H, Lofgren B. Maximum-fixed energy shocks for cardioverting atrial fibrillation. *Eur Heart J* 2020;41:626–31.
 387. Pinski SL, Sgarbossa EB, Ching E, Trohman RG. A comparison of 50-J versus 100-J shocks for direct-current cardioversion of atrial flutter. *Am Heart J* 1999;137:439–42.
 388. Kerber RE, Kienle MG, Olshansky B, et al. Ventricular tachycardia rate and morphology determine energy and current requirements for transthoracic cardioversion. *Circulation* 1992;85:158–63.
 389. Hedges JR, Syverud SA, Dalsey WC, Feero S, Easter R, Shultz B. Prehospital trial of emergency transcutaneous cardiac pacing. *Circulation* 1987;76:1337–43.
 390. Barthell E, Troiano P, Olson D, Stueven HA, Hendley G. Prehospital external cardiac pacing: a prospective, controlled clinical trial. *Ann Emerg Med* 1988;17:1221–6.
 391. Cummins RO, Graves JR, Larsen MP, et al. Out-of-hospital transcutaneous pacing by emergency medical technicians in patients with asystolic cardiac arrest. *N Engl J Med* 1993;328:1377–82.
 392. Ornato JP, Peberdy MA. The mystery of bradyasystole during cardiac arrest. *Ann Emerg Med* 1996;27:576–87.
 393. Niemann JT, Adomian GE, Garner D, Rosborough JP. Endocardial and transcutaneous cardiac pacing, calcium chloride, and epinephrine in postcountershock asystole and bradycardias. *Crit Care Med* 1985;13:699–704.
 394. Quan L, Graves JR, Kinder DR, Horan S, Cummins RO. Transcutaneous cardiac pacing in the treatment of out-of-hospital pediatric cardiac arrests. *Ann Emerg Med* 1992;21:905–9.
 395. Dalsey WC, Syverud SA, Hedges JR. Emergency department use of transcutaneous pacing for cardiac arrests. *Crit Care Med* 1985;13:399–401.
 396. Knowlton AA, Falk RH. External cardiac pacing during in-hospital cardiac arrest. *Am J Cardiol* 1986;57:1295–8.
 397. Ornato JP, Carveth WL, Windle JR. Pacemaker insertion for prehospital bradyasystolic cardiac arrest. *Ann Emerg Med* 1984;13:101–3.
 398. Chan L, Reid C, Taylor B. Effect of three emergency pacing modalities on cardiac output in cardiac arrest due to ventricular asystole. *Resuscitation* 2002;52:117–9.
 399. Eich C, Bleckmann A, Schwarz SK. Percussion pacing—an almost forgotten procedure for haemodynamically unstable bradycardias? A report of three case studies and review of the literature. *Br J Anaesth* 2007;98:429–33.
 400. Appelboam A, Reuben A, Mann C, et al. Postural modification to the standard Valsalva manoeuvre for emergency treatment of supraventricular tachycardias (REVERT): a randomised controlled trial. *Lancet* 2015;386:1747–53.

401. Smith GD, Fry MM, Taylor D, Morgans A, Cantwell K. Effectiveness of the Valsalva Manoeuvre for reversion of supraventricular tachycardia. *Cochrane Database Syst Rev* 2015;CD009502.
402. Smith G, Morgans A, Boyle M. Use of the Valsalva manoeuvre in the prehospital setting: a review of the literature. *Emergency Medicine* 2009;26:8–10.
403. Lim SH, Anantharaman V, Teo WS, Goh PP, Tan AT. Comparison of treatment of supraventricular tachycardia by Valsalva maneuver and carotid sinus massage. *Ann Emerg Med* 1998;31:30–5.
404. Brady Jr. WJ, DeBehnke DJ, Wickman LL, Lindbeck G. Treatment of out-of-hospital supraventricular tachycardia: adenosine vs verapamil. *Acad Emerg Med* 1996;3:574–85.
405. Glatzer K, Cheng JPD, et al. Electrophysiologic effects of adenosine in patients with supraventricular tachycardia. *Circulation* 1999;99:1034–40.
406. Delaney B, Loy J, Kelly AM. The relative efficacy of adenosine versus verapamil for the treatment of stable paroxysmal supraventricular tachycardia in adults: a meta-analysis. *Eur J Emerg Med* 2011;18:148–52.
407. Van Gelder IC, Rienstra M, Crijns HJ, Olshansky B. Rate control in atrial fibrillation. *Lancet* 2016;388:818–28.
408. Dougherty AH, Jackman WM, Naccarelli GV, Friday KJ, Dias VC. Acute conversion of paroxysmal supraventricular tachycardia with intravenous diltiazem and esmolol in terminating supraventricular tachycardia. *J Assoc Physicians India* 1999;47:969–72.
409. Hood MA, Smith WM. Adenosine versus verapamil in the treatment of supraventricular tachycardia: a randomized double-cross-over trial. *Am Heart J* 1992;123:1543–9.
410. Gupta A, Naik A, Vora A, Lokhandwala Y. Comparison of efficacy of intravenous diltiazem and esmolol in terminating supraventricular tachycardia. *J Assoc Physicians India* 1999;47:969–72.
411. Lim SH, Anantharaman V, Teo WS, Chan YH. Slow infusion of calcium channel blockers compared with intravenous adenosine in the emergency treatment of supraventricular tachycardia. *Resuscitation* 2009;80:523–8.
412. Das G, Tschida V, Gray R, et al. Efficacy of esmolol in the treatment and transfer of patients with supraventricular tachyarrhythmias to alternate oral antiarrhythmic agents. *J Clin Pharmacol* 1988;28:746–50.
413. Amsterdam EA, Kulcyski J, Ridgeway MG. Efficacy of cardioselective beta-adrenergic blockade with intravenously administered metoprolol in the treatment of supraventricular tachyarrhythmias. *J Clin Pharmacol* 1991;31:714–8.
414. Brubaker S, Long B, Koyfman A. Alternative Treatment Options for Atrioventricular-Nodal-Reentry Tachycardia: An Emergency Medicine Review. *J Emerg Med* 2018;54:198–206.
415. Gorgels AP, van den Dool A, Hofs A, et al. Comparison of procainamide and lidocaine in terminating sustained monomorphic ventricular tachycardia. *Am J Cardiol* 1996;78:43–6.
416. Scheinman MM, Levine JH, Cannom DS, et al. Dose-ranging study of intravenous amiodarone in patients with life-threatening ventricular tachyarrhythmias. The Intravenous Amiodarone Multicenter Investigators Group. *Circulation* 1995;92:3264–72.
417. Levine JH, Massumi A, Scheinman MM, et al. Intravenous amiodarone for recurrent sustained hypotensive ventricular tachyarrhythmias. Intravenous Amiodarone Multicenter Trial Group. *J Am Coll Cardiol* 1996;27:67–75.
418. Tzivoni D, Banai S, Schuger C, et al. Treatment of torsade de pointes with magnesium sulfate. *Circulation* 1988;77:392–7.
419. Manara AR, Dominguez-Gil B, Perez-Villares JM, Soar J. What follows refractory cardiac arrest: Death, extra-corporeal cardiopulmonary resuscitation (E-CPR), or uncontrolled donation after circulatory death? *Resuscitation* 2016;108:A3–5.
420. Thuong M, Ruiz A, Evrard P, et al. New classification of donation after circulatory death donors definitions and terminology. *Transpl Int* 2016;29:749–59.
421. Champigneulle B, Fieux F, Cheisson G, et al. French survey of the first three-years of liver transplantation activity from uncontrolled donors deceased after cardiac death. *Anaesth Crit Care Pain Med* 2015;34:35–9.
422. Dupriez F, De Pauw L, Darius T, et al. Fourteen years of experience in uncontrolled organ donation after cardio-circulatory death. *Transplantation Proceedings* 2014;46:3134–7.
423. Fieux F, Losser MR, Bourgeois E, et al. Kidney retrieval after sudden out of hospital refractory cardiac arrest: a cohort of uncontrolled non heart beating donors. *Crit Care* 2009;13:R141.
424. Fondevila C, Hessheimer AJ, Flores E, et al. Applicability and results of Maastricht type 2 donation after cardiac death liver transplantation. *Am J Transplant* 2012;12:162–70.
425. Gamez P, Cordoba M, Ussetti P, et al. Lung transplantation from out-of-hospital non-heart-beating lung donors. one-year experience and results. *J Heart Lung Transplant* 2005;24:1098–102.
426. Mateos-Rodriguez AA, Navalpotro-Pascual JM, Del Rio Gallegos F, Andres-Belmonte A. Out-hospital donors after cardiac death in Madrid, Spain: a 5-year review. *Australasian emergency nursing journal : AENJ* 2012;15:164–9.
427. Ortega-Deballon I, Hornby L, Shemie SD. Protocols for uncontrolled donation after circulatory death: a systematic review of international guidelines, practices and transplant outcomes. *Crit Care* 2015;19:268.
428. Peters-Sengers H, Homan van der Heide JJ, Heemskerk MBA, et al. Similar 5-Year Estimated Glomerular Filtration Rate Between Kidney Transplants From Uncontrolled and Controlled Donors After Circulatory Death-A Dutch Cohort Study. *Transplantation* 2017;101:1144–51.
429. Mateos-Rodríguez A, Pardillos-Ferrer L, Navalpotro-Pascual JM, Barba-Alonso C, Martín-Maldonado ME, Andrés-Belmonte A. Kidney transplant function using organs from non-heart-beating donors maintained by mechanical chest compressions. *Resuscitation* 2010;81:904–7.
430. Sanchez-Fructuoso AI, Marques M, Prats D, et al. Victims of cardiac arrest occurring outside the hospital: a source of transplantable kidneys. *Ann Intern Med* 2006;145:157–64.
431. Minambres E, Rubio JJ, Coll E, Dominguez-Gil B. Donation after circulatory death and its expansion in Spain. *Curr Opin Organ Transplant* 2018;23:120–9.
432. West S, Soar J, Callaway CW. The viability of transplanting organs from donors who underwent cardiopulmonary resuscitation: A systematic review. *Resuscitation* 2016;108:27–33.
433. Dominguez-Gil B, Duranteau J, Mateos A, et al. Uncontrolled donation after circulatory death: European practices and recommendations for the development and optimization of an effective programme. *Transpl Int* 2016;29:842–59.
434. Dalle Ave AL, Bernat JL. Uncontrolled Donation After Circulatory Determination of Death: A Systematic Ethical Analysis. *J Intensive Care Med* 2018;33:624–34.
435. Molina M, Dominguez-Gil B, Perez-Villares JM, Andres A. Uncontrolled donation after circulatory death: ethics of implementation. *Curr Opin Organ Transplant* 2019;24:358–63.
436. Gordon L, Pasquier M, Brugger H, Paal P. Autoresuscitation (Lazarus phenomenon) after termination of cardiopulmonary resuscitation - a scoping review. *Scand J Trauma Resusc Emerg Med* 2020;28:14.
437. Bruce CM, Reed MJ, MacDougall M. Are the public ready for organ donation after out of hospital cardiac arrest? *Emergency Medicine* 2013;30:226–31.
438. Joffe AR, Carcillo J, Anton N, et al. Donation after cardiocirculatory death: a call for a moratorium pending full public disclosure and fully informed consent. *Philos Ethics Humanit Med* 2011;6:17.
439. Rodriguez-Arias D, Tortosa JC, Burant CJ, Aubert P, Aulisio MP, Youngner SJ. One or two types of death? Attitudes of health professionals towards brain death and donation after circulatory death in three countries. *Med Health Care Philos* 2013;16:457–67.

440. Manara A, Shemie SD, Large S, et al. Maintaining the permanence principle for death during in situ normothermic regional perfusion for donation after circulatory death organ recovery: A United Kingdom and Canadian proposal. *Am J Transplant* 2020;20:2017–25.
441. Mentzelopoulos SD, Couper K, Van de Voorde P, et al. Ethics of resuscitation and end of life decisions. *Resuscitation* 2021.
442. Bleijenberg E, Koster RW, de Vries H, Beesems SG. The impact of post-resuscitation feedback for paramedics on the quality of cardiopulmonary resuscitation. *Resuscitation* 2017;110:1–5.
443. Couper K, Kimani PK, Davies RP, et al. An evaluation of three methods of in-hospital cardiac arrest educational debriefing: The cardiopulmonary resuscitation debriefing study. *Resuscitation* 2016;105:130–7.
444. Edelson DP, Litzinger B, Arora V, et al. Improving in-hospital cardiac arrest process and outcomes with performance debriefing. *Arch Intern Med* 2008;168:1063–9.
445. Wolfe H, Zebuhr C, Topjian AA, et al. Interdisciplinary ICU cardiac arrest debriefing improves survival outcomes*. *Crit Care Med* 2014;42:1688–95.
446. Couper K, Perkins GD. Debriefing after resuscitation. *Curr Opin Crit Care* 2013;19:188–94.