Joint British Societies' guideline on management of cardiac arrest in the cardiac catheter laboratory

Version 8.0

Joel Dunning BA(Hons) BM BCh FRCS(Cth) PhD[1], R. Andrew Archbold MBBS MD FRCP[2], Joseph De Bono BM BCh MA FRCP DPhil[3], Elizabeth Butterfield[4] MSc RN PgCEHE, Nicholas Curzen BM(Hons) PhD FRCP[5], Charles D Deakin MA MD FRCA FRCP FFICM FERC[6], Ellie Gudde[7], Thomas Keeble MBBS BSc MRCP MD[7], Alan Keys[8], Mike Lewis Bsc(Hons) MB ChB FRCS(CTh) MD[9], Niall J O-Keeffe FRCA[10], Jaydeep Sarma MA PhD FRCP[11], Martin Stout PhD MSc BSc[12], Paul Swindell[8], Simon Ray BSc MD FRCP FACC FESC[11]

1. Department of Cardiothoracic Surgery, James Cook University Hospital, Middlesbrough, UK
2. Department of Cardiology, Barts Heart Centre, St. Bartholomew's Hospital, London, UK
3. Department of Cardiology, Queen Elizabeth Hospital, University of Birmingham, Birmingham, UK.
4. School of Nursing, Midwifery and Social Work, Faculty of Health and Wellbeing, Canterbury Christ Church University, Canterbury, UK.
5. Faculty of Medicine, University of Southampton & Department of Cardiology, Southampton University Hospitals NHS Trust, UK
6. Cardiac Anaesthesia and Cardiac Intensive Care, Southampton University Hospitals NHS Trust, UK
7. Essex Cardiothoracic Centre, Mid and South Essex NHS Trust, Basildon, Essex, UK, and Medical Technology Research Centre, Anglia Ruskin School of Medicine, Chelmsford, UK
8. Patient representative for the British Cardiovascular Society.
9. Department of Cardiac Surgery, Royal Sussex County Hospital, Brighton, UK.
10. Department of Cardiothoracic Anaesthesia and Intensive Care, Manchester Royal Infirmary, Manchester, UK
11. Department of Cardiology, Manchester University Hospitals, Wythenshawe, UK
12. School of Healthcare Science, Manchester Metropolitan University, Manchester, UK

Corresponding author:
Joel Dunning
Department of Cardiothoracic Surgery
James Cook University Hospital
Middlesbrough, UK
E-mail: joeldunning@doctors.org.uk
Tel: +44 7801548122
Fax: +44 1642854613

Executive Summary	442 words
Manuscript	11769 words

Keywords: Cardiac arrest, catheter laboratory, cardiac catheterisation, resuscitation, mechanical CPR, extracorporeal CPR, guideline.

Executive summary

More than 300,000 procedures are performed in cardiac catheter laboratories in the UK each year. The variety and complexity of percutaneous cardiovascular procedures have both increased substantially since the early days of invasive cardiology when it was largely focused on elective coronary angiography and single chamber (right ventricular) permanent pacemaker implantation. Modern-day invasive cardiology encompasses primary percutaneous coronary intervention (PCI), cardiac resynchronisation therapy (CRT), complex arrhythmia ablation, and structural heart interventions. These procedures all carry the risk of cardiac arrest.

We have developed evidence-based guidelines for the management of cardiac arrest of adult patients in the catheter laboratory. The guidelines include recommendations which were developed by a collaboration between nine professional and patient societies that are involved in promoting high quality care for patients with cardiovascular conditions. We present a set of protocols which use the skills of the whole catheter laboratory team and which are aimed at achieving the best possible outcomes for patients who suffer a cardiac arrest in this setting. We identified six roles and developed a treatment algorithm which should be adopted during cardiac arrest in the catheter laboratory. We recommend that all catheter laboratory staff undergo regular training for these emergency situations which they will inevitably face.

1. Introduction

More than 300,000 procedures are performed in cardiac catheter laboratories in the UK each year. The variety and complexity of procedures undertaken in the cardiac catheter laboratory have increased substantially over the last 30 years. Invasive cardiology has grown from a largely diagnostic specialty focused on elective coronary angiography to one that treats a wide spectrum of cardiovascular problems through many different types of interventional procedure, often in urgent or emergency situations. The majority of myocardial revascularisation procedures, for example, are now performed by percutaneous coronary intervention (PCI), and over a quarter of these are undertaken in the setting of acute ST elevation myocardial infarction (STEMI). Pacemaker implantations have evolved from mostly right ventricular procedures to treat bradycardias to encompass biventricular pacing to deliver cardiac resynchronisation therapy (CRT) for patients with left ventricular dysfunction and/or implantable cardioverter defibrillators (ICDs) for patients at risk of ventricular arrhythmia. Complex arrhythmia ablation procedures have become more common as their indications and success rates have increased. Recent years have seen a large increase in structural heart interventions driven by transcatheter aortic valve implantation (TAVI) to treat aortic stenosis, adding a further level of complexity to procedures undertaken in the catheter laboratory. Percutaneous interventions on the mitral valve are increasing in number while the tricuspid valve and heart failure syndromes are targets for interventional technology development. Not only have procedures become increasingly complex, they are often undertaken in patients who are elderly and comorbid, with limited cardiorespiratory reserve.

Invasive procedures undertaken in the catheter laboratory all carry the risk of complications which lead directly or indirectly to cardiac arrest. Careful assessment of the risks and benefits of the procedure is required for each patient. In many cases, the risk of cardiac arrest is low. In others, such as primary PCI, the risk is appreciable. The incidence of cardiac arrest during PCI is approximately 1.5% [3-4]. The chance of successful resuscitation is higher than in other in-hospital cardiac arrest situations [5], especially for elective procedures. The catheter laboratory benefits from the presence of an expert team which is already present at the time of cardiac arrest, the reason for the cardiac arrest may be known, and it may be reversible through an intervention in the catheter laboratory. Other specialists are usually readily available to assist, if required. Nevertheless, there is variation between catheter laboratories, for example, whether they are based in a cardiac surgical centre or in a district general hospital and in the number of practitioners and the roles which they undertake in the catheter laboratory. Furthermore, the remedial intervention to achieve restoration of spontaneous circulation (ROSC), such as PCI, may take some time to perform. Rescuers who are used to 10-15 minute cardiac arrest scenarios may need to become familiar with prolonged cardiac arrest scenarios which involve mechanical cardiopulmonary resuscitation (CPR), the administration of infusions, consideration of every aspect of the patient's physiology, and treatment more akin to that of a critically-ill patient on an intensive care unit (ICU). In undertaking invasive procedures in the catheter laboratory, our expectation should be for successful resuscitation after a cardiac arrest. In aiming to achieve the best possible outcomes, a consistent approach to the arrested patient in the catheter laboratory is needed. For this reason, we have developed evidence-based guidelines for the management of cardiac arrest in the catheter laboratory.

2. Scope and methods

The guideline covers adult patients undergoing any invasive procedure in the catheter laboratory, including coronary angiography, PCI, structural heart interventions including TAVI and mitral valve procedures, pacemaker and ICD implantation, arrhythmia ablation, atrial appendage occlusion, and pacing system extraction.
We did not consider patients who arrest and are then brought to the catheter laboratory as these patients have recently been considered in a position paper by the European Society of Cardiology published in 2020 [128].

The guideline was developed by a collaboration between nine stakeholder organisations: The British Cardiovascular Society (BCS), The British Cardiovascular Intervention Society (BCIS), The British Heart Rhythm Society (BHRS), The British Association for Nursing in Cardiovascular Care (BANCC), The British Society of Echocardiography (BSE), The Association for Cardiothoracic Anaesthesia and Critical Care (ACTACC), The Cardiovascular Care Partnership (CCPUK), The Society for Cardiothoracic Surgery in Great Britain and Ireland (SCTS), and The Resuscitation Council UK.

These guidelines were developed in accordance with The Resuscitation Council UK 2021 guidelines development process [111]. We used the European Society of Cardiology 2018 practice guidelines recommendations for grading the strength of recommendations and for assessing the levels of evidence in support of them [112]. It should be acknowledged that the literature surrounding cardiac arrest comprises mostly of papers which reported the findings of studies after either in-hospital or out-of-hospital cardiac arrest rather than of cardiac arrest in the catheter laboratory and that their findings were extrapolated into the catheter laboratory environment.

We undertook a comprehensive review of the literature and a Delphi expert consensus process in order to identify all the situations in the catheter laboratory that potentially lead to cardiac arrest and to provide team-based solutions to their management. We propose that these guidelines become the standard of care in this specialist area.

1.1 International Liaison Council on Resuscitation (ILCOR)

With regard to international guidelines, resuscitation is governed by the ILCOR which is a collaborative of 7 world resuscitation councils which was set up in 1992. The full range of all recommendations in resuscitation is reviewed and updated and a document of the 'best evidence' in resuscitation is created. The 7 resuscitation councils then take this evidence and generate guidelines adapted to the needs of their own healthcare systems.

1.2 The American Heart Association guidelines

The 2015 AHA guidelines have a two-page section entitled 'Cardiac Arrest During Percutaneous Coronary Intervention', although this was omitted from their 2020 guideline [1]. In 2015 they chose to mainly concentrate on a discussion with regard to automated CPR devices over manual compressions and also the use of extracorporeal CPR (ECPR) devices. They do not come to any firm conclusion but state that mechanical CPR devices and extracorporeal CPR devices have been used as rescue bridges to other interventions such as coronary bypass grafting, transplantation or longer term mechanical devices. In the text of the guideline it is also noted by the authors that early defibrillation within a minute is associated with excellent outcomes. No other special considerations are discussed with regard to the management of the arrest in the catheter laboratory.

1.3 The European Resuscitation Council guidelines

The ERC published more comprehensive guidance for catheter laboratory resuscitation in 2021 in the guideline entitled 'cardiac arrest in special circumstances'[2]. They have a protocol diagram, and there is a strong emphasis on ensuring that catheterisation laboratory staff are adequately trained in resuscitation technical skills including team training, and specific protocols for the initiation of mechanical CPR, temporary pacing, and pericardiocentesis, with the use of on-site emergency drills. They also recommend the availability of resuscitative equipment and the use of checklists. Mechanical CPR is recommended due to the risk to staff of manual CPR with fluoroscopy, and the requirement not to stop CPR during PCI.

1.4 The Australian and New Zealand guidelines

These guidelines discuss the use of mechanical CPR in an arrest during PCI [6]. Interestingly, they also discuss cough CPR for which they found some case reports in electrophysiology labs. They discuss drainage of pericardial tamponade in an arrest or thoracotomy and pericardiotomy with a class B recommendation if pericardiocentesis fails. In their handbook, they also state that 'The Interventionalist is heavily task burdened and as such is seldom in a good position to lead the resuscitation, and that 'CPR is likely to be required and there may be some tension between this and the ability of the interventionalist to continue with the procedure', indicating an understanding of the particular challenges faced in a catheter lab.

3. A novel protocol for the management of patients who suffer a cardiac arrest in the catheter laboratory

We have developed a modified resuscitation protocol which is specifically designed for the specialist area of the catheter laboratory. Of note this does not apply to recovery areas but does apply to hybrid laboratories where TAVI or Mitraclip procedures are being undertaken. This protocol could also be used in hybrid laboratories performing TEVAR. The full protocol is shown in figure 1 and the rationale for its development is discussed.

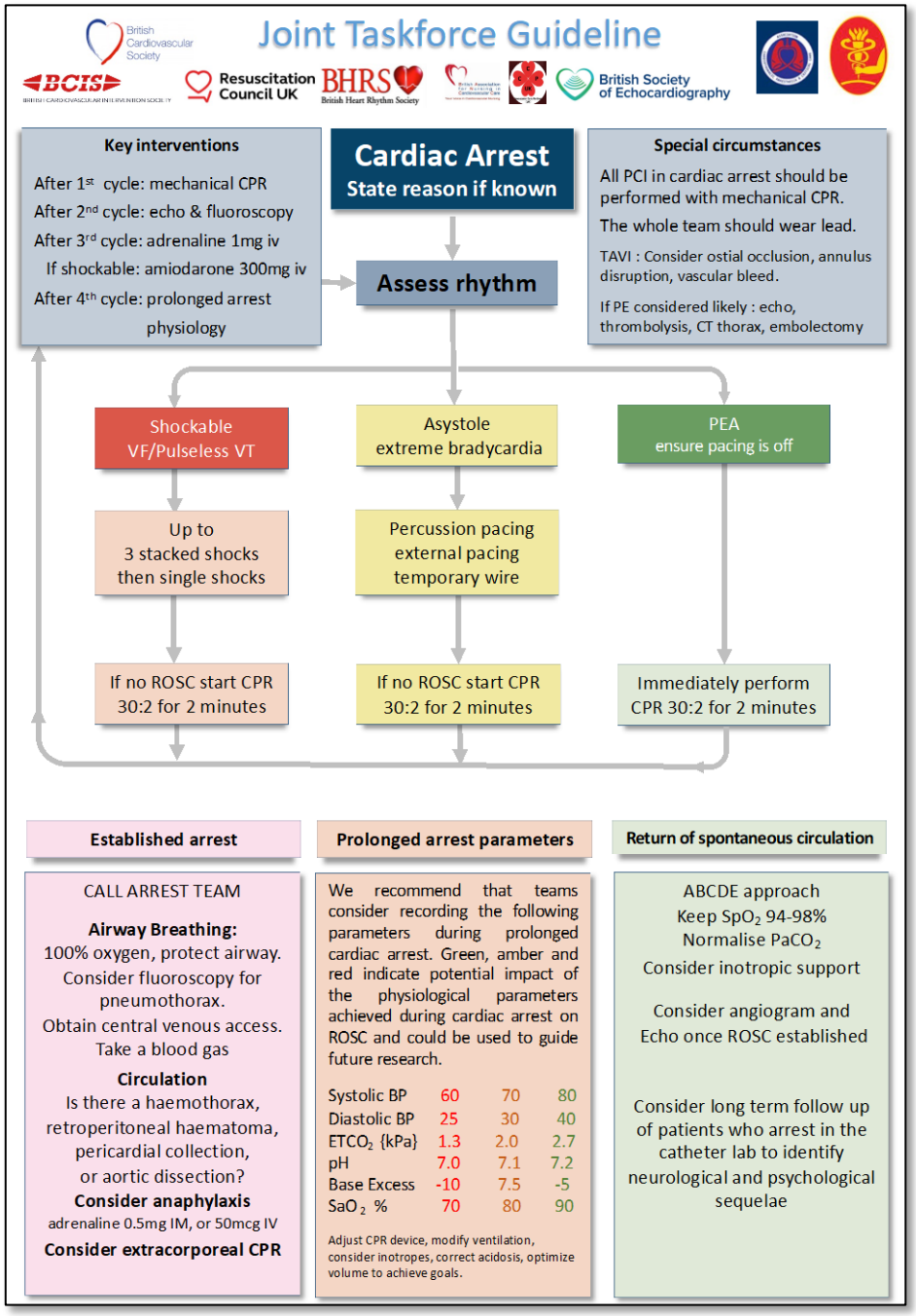


Figure 1. Protocol for resuscitation of patients who suffer a cardiac arrest in the catheter laboratory

2.1 How should cardiac arrest be identified, defined, and categorised?

In a catheter laboratory a cardiac arrest is identified much more quickly than other in-hospital arrest scenarios. VF, pulseless VT, and asystole may be diagnosed immediately when a continuous intra-arterial blood pressure is displayed, without need for an added pulse check.

It is important to define what constitutes a cardiac arrest in a catheter lab. In contrast to the two pathways in the standard arrest algorithm we have separated the protocol into three pathways: ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT), asystole or extreme bradycardia, and pulseless electrical activity (PEA).

In VF or pulseless VT, the pulse oximeter and arterial trace will confirm the absence of a cardiac output. A cardiac arrest should be called and the operator should tell the team if they know the reason for the arrest (e.g. vessel dissection or occlusion in PCI, occluded left main stem in TAVI or irritation of the ventricle in a pacing procedure for example). VF or VT is occasionally deliberately induced in electrophysiology labs and this should not trigger the arrest protocol.

Temporary asystole or extreme bradycardia (<30/min) may occur and can be anticipated with ventricular manipulation of pacing leads or EP catheters. A cardiac arrest should be called when the rhythm disturbance is unexpected and or prolonged. The pulse oximetry and any arterial transduction will show non pulsatile traces, and percussion pacing, external pacing, or temporary wire pacing may be attempted prior to chest compressions.

Many cases of PEA may be diagnosed by the absence of a pulsatile waveform on a continuous intra-arterial blood pressure display. Non or minimal pulsatility of the arterial trace and pulse oximetry in the presence of continuing electrical activity confirms the diagnosis. The operator should call a cardiac arrest and inform the laboratory team of the likely cause.

Pulseless VT can be mistaken for PEA. A regular rhythm above 140/min should be considered as pulseless VT if the arterial trace and pulse oximetry have minimal or absent pulsation and the patient has lost consciousness. Similarly extreme bradycardia may be mistaken for PEA if the arterial trace is not being transduced. It may be necessary to feel the pulse for 10 seconds or alternatively (and optimally) to perform a rapid echocardiogram to identify a cardiac output.

Occasional patients will deteriorate in the catheter laboratory with support devices in place such as LVAD, ECMO or Impella, where non pulsatility does not equate with an absent cardiac output.

Recommendations

VF, pulseless VT and asystole may all be diagnosed immediately based on the monitoring in the catheter lab (figure 1). There is no need to routinely look, listen and feel for 10 seconds.

Class I, level of Evidence C

Many cases of PEA may also be diagnosed by the absence of pulsatile traces but if in doubt then either look, listen and feel or use echocardiography to look for a cardiac output.

Class IIa, Level of Evidence C

If a patient has circulatory collapse with a rate less than 30/min then we define this as extreme bradycardia as this may respond to percussion, external or temporary wire pacing and thus we recommend following the asystole pathway.

Class I, Level of Evidence C

If a patient has an arrhythmia above a rate of 140/min without a discernible cardiac output then we recommend following the pathway for VF/pulseless VT as this may respond to defibrillation.

Class IIa, Level of Evidence C

2.2 Should all members of the resuscitation team wear lead aprons?

All clinicians coming into an arrest in the catheter laboratory should wear lead aprons. Rescuers may be tempted to come in to assist and feel that there is no time to put on lead aprons, especially anaesthetists or members of resuscitation teams. Our protocol utilises the members of the team present in the catheter laboratory in the initial stages of the arrest and, thus, it is strongly recommended that all rescuers entering the room should wear lead aprons as it is very likely that the cardiologist may need to perform fluoroscopy in many emergency situations.

We recommend that catheter laboratory team members are regularly trained in basic airway management to ensure a patent airway and good oxygenation for all patients, which should mitigate the risk of the need for immediate anaesthetic presence and allow them time to put lead on. We recommend that an individual in the catheter laboratory team is allocated to manage the personnel coming into the arrest. They will be required to assist these personnel to put on lead, and as they do this, they will be able to brief these clinicians as to the arrest situation in the catheter lab.

Catheter laboratories must also ensure that lead aprons are available for emergency team members and that a range of sizes are available for emergency use.

Recommendation

All clinicians entering a cardiac arrest situation in a catheter laboratory must first put on lead aprons prior to entry.

Advance provision should be made for enough lead aprons to be available for this situation.

Class IIa, Level of Evidence C

2.3 Should we defibrillate before external chest compressions?

In 2020 ILCOR published a literature review on this subject [91] and it was identified as a priority area for the Basic Life Support Taskforce. They found that in 5 RCTs there was no difference in outcomes with either a specified period of chest compressions (typically 1.5-3minutes) before shock delivery compared to shock delivery as soon as possible with interim brief CPR while the defibrillator was readied for use. A meta-analysis of these studies (n= 10,600 patients) also found no differences. Only when the arrest time was more than 5 minutes did any studies show an improvement with CPR before defibrillation. The ERC 2021 guidelines[2] do not recommend the routine delivery of a pre-specified period of CPR before rhythm analysis and shock delivery, and recommend shock delivery as soon as it can be applied. Deferring chest compressions until after shock delivery has been recommended in other highly monitored areas such as after cardiac surgery in the ERC 2021 guidelines[2] and they now state: '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'

Recommendation

In VF or VT without a cardiac output, external chest compressions may be deferred in order to perform up to three stacked shocks immediately.

Class IIa, Level of Evidence A

2.4 How many attempts at defibrillation should be performed prior to commencing external chest compressions?

Evidence was sought for the optimal number of attempts at external defibrillation for VF or pulseless VT prior to commencing external chest compressions. This has been subject to a literature review looking at the effectiveness of the numbers of defibrillation attempts in a range of scenarios including ICD insertions, electrophysiological studies, out-of-hospital arrests and animal studies [18]. When the data from all 15 papers are combined, the average success rate of sequential shocks declines from 78% for the first shock to 35% for the second shock and 14% for the third, and any subsequent shock will have less than a 10% chance of success. Thus, the likelihood of successful cardioversion declines dramatically from first to second shock and declines further from second to third shock.

Our guideline seeks to place a mechanical CPR device on the patient early in the pathway and it is important to consider how we modify the protocol to allow this. Firstly, it may be possible to assess the rhythm while the mechanical CPR device is ongoing. Our patients often have multi-lead ECG monitoring, and sometimes

intracardiac ECG monitoring, and thus where the team leader is satisfied that there has been no change from the shockable rhythm, there is no need to pause the CPR device every 2 minutes. If the team leader is uncertain then a pause should be performed every 2 minutes for rhythm assessment. As there is no risk to a rescuer, charging and administration of a shock may be performed while mechanical CPR is ongoing. Finally, if multiple shocks have failed to cardiovert the patient and it is clear that a coronary occlusion is the cause of the arrhythmia, then mechanical CPR should continue uninterrupted until coronary flow is restored.

Recommendations

In VF or pulseless VT, up to three stacked shocks should be given without intervening external chest compressions.

Class I, Level of Evidence B

Thereafter, a single attempt at defibrillation, if required, is performed every two minutes.

Class I, Level of Evidence C

If the arrhythmia is due to an acute coronary artery occlusion, repeat shock administration can be deferred to facilitate PCI to the occluded artery.

Class IIa, Level of Evidence C

Shocks for known VF/VT should be administered while mechanical CPR is ongoing.

Class I, Level of Evidence C

If the patient is receiving mechanical CPR and it is possible to assess the rhythm while the CPR is ongoing then it may not be necessary to pause the CPR device.

Class IIa, Level of Evidence C

2.5 Should we perform pacing in patients who undergo an asystolic arrest in the catheter laboratory prior to external chest compressions?

In an asystolic arrest in a catheter laboratory there is the potential to rapidly restore cardiac output with pacing and, as this is a witnessed arrest, if pacing is immediately performed then potentially there will be an immediate restoration of a spontaneous circulation. Furthermore, in the literature review on the effectiveness of external chest compressions in the early stages of an arrest it was found that there was little evidence to suggest harm from delaying external chest compressions for a few minutes. Periods of asystole are not uncommon in pacing and electrophysiology (EP) laboratories and most cardiologists would use external, percussion or transvenous temporary wire pacing to address this as a routine part of their practice. We recommend that pacing should be attempted prior to the initiation of external chest compressions.
Percussion pacing may initially be attempted (see the section below for further details). For external pacing the pacing pads should be applied, and the amplitude of the pacing quickly increased to regain an output. Only if capture is not obtained with maximum amplitude with the pads well applied should external chest compressions be performed. If the cardiologist suspects that the arrest is due to an extreme bradycardia due to a conduction defect transvenous pacing can be used if external pacing has been ineffective in achieving ventricular capture.

Recommendations

In a patient who arrests with asystole or extreme bradycardia with a rate of less than 30bpm, external pacing or percussion pacing should be attempted prior to chest compressions.

Class IIa, Level of Evidence C

If either external or percussion pacing is ineffective and the cardiologist feels that there is a persisting bradyarrhythmia as the cause of the arrest a temporary pacing wire should be inserted while chest compressions are performed.

Class IIa, Level of Evidence C

2.6 Interventions to address pulseless electrical activity

Our protocol using 3 categories aims to ensure that the greatest number of patients possible may benefit from either immediate defibrillation or pacing prior to the institution of external cardiac compressions. In patients presenting with PEA efforts should be directed towards identifying the underlying causes and treating them rapidly. There are a number of possibilities to consider that are relevant to the catheter laboratory:

Hypoxia – There is an airway and breathing protocol with a person allocated to address these issues in an arrest.

Hypovolaemia - bleeding. Our recommendation ensures that the four most likely areas for bleeding in the catheter laboratory (haemothorax, retroperitoneal or vascular bleed, aortic dissection and tamponade) are investigated.

Hypo/hyperkalaemia, H^+ ion imbalance and electrolyte abnormalities are addressed by a recommendation to perform an early blood gas.

Hypothermia is unusual in a catheter lab, other than following prolonged out of hospital arrest

Tension pneumothorax may arise during procedures requiring vascular access in the thorax– This is addressed in the airway and breathing protocol and by fluoroscopy.

Tamponade –where tamponade is a possibility immediate echocardiography should be performed. The clinical sign most suggestive of tamponade in a cardiac arrest is the inability to generate a systolic blood pressure of 70mmHg with external cardiac massage.

Toxins – One possible cause of a toxin-related arrest in a catheter laboratory is a drug error. We recommend that any syringe drivers or infusions should be stopped in the arrested patient to address this possibility. Careful consideration should also be given to contrast or antibiotic-induced anaphylaxis. Look for supportive signs such as rash, wheeze or facial swelling. Our protocol recommends adrenaline 0.5mg IM or otherwise 50 mcg IV.
Thrombosis- coronary or pulmonary. In the catheter laboratory this would most commonly relate to acute coronary occlusion, either due to an acute myocardial infarction or a complication of PCI which in both circumstances would be treated by re-opening of the vessels by PCI. Pulmonary embolism causing an arrest is far less common. In an arrest situation it can be very difficult to diagnose but is suggested by disproportionate right ventricular distention. If suspected, then thrombolysis or thrombectomy might be considered. This is considered in our protocol.

2.7 How deeply should we perform chest compressions?

The universal algorithm recommends compressing the chest to between 5 and 6cm over the lower half of the sternum [1-2]. For those patients with an arterial trace being transduced, we recommend 'titrating' chest compressions to achieve a systolic of 70mmHg. This allows more gentle external compressions to be performed, potentially reducing the chance of compression related injury, whilst still producing effective cerebral perfusion. Furthermore, the inability to generate an acceptable systolic pressure is suggestive of tamponade.

Recommendations

Chest compressions should be performed to a depth of 5-6cm to the lower half of the sternum.

Class IIa, Level of Evidence C

If the arterial trace is being transduced it is preferable to compress to achieve a systolic pressure of 70mmHg.

Class IIb, Level of Evidence C

2.8 Should we perform a precordial thump?

The AHA guidelines [20] state that 'The precordial thump may be considered for termination of witnessed monitored unstable ventricular tachyarrhythmias when a defibrillator is not immediately ready for use (Class IIb, LOE B), but should not delay CPR and shock delivery. ILCOR performed a worksheet on this subject in 2021 [19]. This documents that precordial thump is only effective in 2% of attempts and, in fact, rhythm deterioration is twice as common as successful cardioversion. Thus, our protocol does not recommend a precordial thump. A defibrillator should be very close to hand in every catheter laboratory, and this is much more likely to successfully cardiovert the patient.

Recommendations

A precordial thump is not recommended for patients who suffer a cardiac arrest in the catheter laboratory due to VF or pulseless VT.

Class III (no benefit), Level of Evidence C

2.9 Is cough CPR an effective alternative to external chest compressions in the catheter laboratory?

The AHA stated in 2010 that "cough" CPR may be considered in settings such as the cardiac catheterization laboratory for conscious, supine, and monitored patients if the patient can be instructed and coached to cough forcefully every 1 to 3 seconds during the initial seconds of an arrhythmic cardiac arrest. It should not delay definitive treatment (Class IIb, LOE C). The AHA made no modifications to this recommendation in 2020 [1].

The longest documented case of a patient maintaining their own spontaneous circulation is 90 seconds and most reports were around 30 seconds, in both VF as well as asystole. These patients seem able to maintain consciousness in a manner similar to the mechanism proposed for external CPR, namely a compression of the pulmonary vascular bed increasing the pressure in the left atrium then ventricle and allowing blood to flow across the aortic valve. There are case reports of its use for short periods of time in the catheter laboratory[30], including prior to defibrillation[31, 32] but the most effective use seems to be in patients with severe bradycardia who are periarrest.
ILCOR performed a systematic review in 2021 19]. Their conclusion was as follows: 'We suggest cough CPR may only be considered as a temporizing measure in an exceptional circumstance in a witnessed, monitored, in-hospital setting (such as a cardiac catheterization laboratory) if a non-perfusing rhythm is recognised promptly before loss of consciousness (weak recommendation, very-low-certainty evidence).

If a bradycardic or asystole cardiac arrest is very rapidly identified (whilst the patient is responsive), then it is reasonable to attempt to coach the patient to cough forcefully every 1 to 3 seconds if experienced clinicians choose to try this. This should not delay the commencement of the cardiac arrest protocol including the application of pads and defibrillating or pacing if necessary. Staff should be ready to perform CPR if the patient stops following the command to cough, and the arterial trace should be observed to monitor the effectiveness of cough CPR.

Recommendation

Vigorous cough CPR every 1 to 3 seconds in the catheter laboratory may only be considered as a temporising measure if a non-perfusing rhythm is recognised promptly before loss of consciousness. It is likely to be most useful in bradycardia in order to maintain consciousness until more definitive reversal measures can be instituted.

Class IIb, Level of Evidence C

2.10 Percussion (fist) pacing as an alternative to CPR in the catheter laboratory

ILCOR performed a systematic review on this subject in 2021 [19]. The total number of cases reported in the literature is around 170 patients and in the largest series of 100 patients, 69 of these maintained consciousness and 90 had percussion pacing as an alternative to CPR[89].
In a study performed in 1978 [90] 19 healthy volunteers and 31 patients with paused pacing had a right heart catheter and the authors found reliable electrical impulses could be reproduced for up to 6 minutes when the left lower sternum was struck with the clenched fist from about 20-30cm height, by causing the right ventricular pressure to rise by around 20mmHg with this action.
The ILCOR 2021 systematic review states that 'We suggest fist pacing may only be considered as a temporizing measure in an exceptional circumstance in a witnessed, monitored, in-hospital setting (such as a cardiac catheterization laboratory) if a non-perfusing rhythm is recognised promptly before loss of consciousness.
The catheterisation laboratory is a highly monitored environment where bradycardia and asystole is common. There have been no studies comparing CPR to percussion pacing directly but percussion pacing has been shown to effectively induce cardiac contraction and maintain consciousness in patients immediately identified as having an asystolic arrest and therefore, with close monitoring, we recommend that this could be a useful temporising method in the catheterisation laboratory, while preparations are made for external pacing or a temporary wire or the administration of chronotropic medications.

Recommendations

In monitored patients with onset of a non-perfusing rhythm such as asystole or extreme bradycardia (figure 1), percussion (fist) pacing may be deployed as an alternative to external pacing when successful perfusion is confirmed by a continuous arterial tracing, pulse oximetry and electrocardiography.
Class IIb, Level of Evidence C

Percussion pacing should be performed at a rate of 50-70 per minute and the ulnar side of a clenched fist should be used to strike the chest from 20-30cm above the left lower sternal edge, in order to mechanically increase the right atrial pressure by 15-20mmHg.

Class IIb, Level of Evidence C

2.11 Active pad compression for defibrillation

In atrial fibrillation there are papers including the Ottowa AF Cardioversion protocol [105] and the 2014 AHA guidelines for the management of patients with atrial fibrillation [106] that mention using paddles to provide manual compression over the defibrillator pads as a method of increasing the success of cardioversion. The original citation as evidence in favour of this intervention was by Kerber et al [107] in 1981 looking at 44 cardioversion patients, although, interestingly, the only part of this paper that actually looked at active compression was a sub-report of 4 dogs who were cardioverted with or without active compression.

Sirna et al in 1988 reported a 13% reduction in impedance with active compression when uniphasic defibrillation was being performed in 28 patients [108] and a similar result was found by Ramirez et al in 2016 with 11 participants where they concluded that 8kg of pressure could reduce the impedance by about 10% [109].

Thus, there is limited evidence from animal studies and case series, as well as a trial of cardioversion in atrial fibrillation, that active compression of the defibrillation pads using disconnected defibrillation paddles reduces intrathoracic impedance and improves shock efficacy. In the absence of any studies in ventricular arrhythmias in humans the routine use of active compression during defibrillation is not recommended. However, the use of disconnected defibrillation paddles to apply external compression to defibrillation pads may be considered in patients with arrhythmias refractory to cardioversion particularly where there is a risk of high intrathoracic impedance.

Recommendations

Active pad compression is not routinely recommended for defibrillation and the standard method of defibrillation should be via pads either in an anterior-lateral position, an anterior-posterior position or apex-posterior position.

Class III (lack of Benefit), Level of Evidence C

In situations when initial attempts at cardioversion have failed, an expert clinician who feels that increased impedance may be a factor, such as in high BMI, may elect to try active pad compression if paddles are also available to provide the compression.

Class IIb, Level of Evidence C

2.12 Does adrenaline improve outcomes in resuscitation in the catheter laboratory?

ILCOR in 2015 reviewed the literature with regard to adrenaline including a large RCT by Olasveengen et al [16] where ambulances were randomised to Group 1: CPR and defibrillation with iv cannulation and usual resuscitation medications versus Group 2: CPR and defibrillation alone. This RCT showed reduced survival to hospital discharge in group 1 and this was felt to be due to the ineffectiveness of the drugs and also the delay in CPR in order to cannulate and administer the drugs. This paper, and a more recent meta-analysis [4] (demonstrating no benefit of adrenaline in cardiac arrest) led ILCOR to write: 'despite the widespread use of adrenaline during resuscitation, and several studies involving vasopressin, there is no placebo controlled study that shows that the routine use of any vasopressor at any stage during human cardiac arrest increases survival to hospital discharge. Current evidence is insufficient to support or refute the routine use of any particular drug or sequence of drugs. Despite the lack of human data, the use of adrenaline is still recommended, based largely on animal data.'

The PARAMEDIC-2 study [23] randomised 8014 patients in an arrest situation across 5 ambulance services in the UK to receive either 1mg of adrenaline every 3-5 minutes, or identical syringes containing 0.9% saline. The average time for the ambulance to arrive was 6.6 minutes and the mean time to trial drug administration was 13 minutes after arrival. There was a large increase in the number of patients who had return of spontaneous circulation in the adrenaline arm (36% vs 11%), as well as the number who were transferred to hospital (50% vs 30%). The primary outcome measure was survival at 30 days and this was 3.2% in the adrenaline group and 2.4% in the placebo group which was significant, but the number of survivors with severe neurological impairment was 31% in the adrenaline group versus 18% in the control group, and thus the trial was negative in terms of survival with favourable neurological outcome (2.2% vs 1.9%). The triallists concluded that adrenaline significantly improved the chance of achieving return of spontaneous circulation and the patient to hospital admission but that this led only to a greater proportion surviving with severe neurological disability.

In the light of this important study, it is suggested that the current recommendations of giving adrenaline every 3-5 minutes at a dose of 1mg is supported on the basis that it is unlikely to harm the patient and may be beneficial. It is thus recommended that adrenaline (1mg) is given after the 3rd cycle. It may be acceptable to administer smaller doses of adrenaline if a senior clinician feels that there may be reactive hypertension on ROSC.

Our guideline group discussed the issue of administration of adrenaline in cases of a non-shockable rhythm. Current recommendations from the European Resuscitation Council are to give adrenaline at a dose of 1mg as soon as possible but they do caveat this by saying that 'exceptions may exist where a clear reversible cause can be rapidly addressed'. In PEA and Asystole in the catheter laboratory there are reversible causes that should be addressed, and for this reason the group concluded that we should recommend administering adrenaline at the same time as in a shockable rhythm to allow time for reversible causes to be addressed.

Recommendations

We recommend that for patients who arrest in a catheter laboratory the benefits of adrenaline which are mainly based on out-of-hospital arrest RCTs may also apply in terms of an increased return of spontaneous circulation.

Class I, Level of Evidence A

We recommend that for patients who arrest with VF or pulseless VT adrenaline 1mg iv is given after the third shock cycle.

Class I, Level of Evidence A

We recommend that for patients who arrest with a non-shockable rhythm adrenaline 1mg iv is given after the third cycle of CPR rather than immediately to allow time for reversible causes of cardiac arrest to be addressed

lkn

2.13 Waveform capnography in cardiac arrest

We recommend that waveform capnography is used for patients in established cardiac arrest. Not only does this prove that the airway is patent, and they have reasonable air entry to allow the exchange of CO_2, but more importantly the level of exhaled CO_2 correlates with the cardiac output. Furthermore, capnography can be used as a prognostic guide to the likely result of prolonged resuscitation. An end tidal CO_2 more than 20 mmHg (2.7 kPa) is a good prognostic indicator whereas an end tidal CO_2 of less than 10 mmHg (1.3 kPa) indicates a poor prognosis and may be used to indicate that further treatment is likely to be futile or that modifications are required to the CPR to improve this figure.

2.14 Goal-directed management during prolonged cardiac arrest in the catheter laboratory

A number of physiological parameters are associated with higher rates of ROSC. This has led to the hypothesis that higher rates of ROSC and better clinical outcomes might be achieved by goal-directed resuscitation techniques. This may be particularly relevant to the management of cardiac arrest in the catheter laboratory where resuscitation attempts may be prolonged and invasive monitoring is routine. Physiological parameters of interest based on our literature review on this topic are listed in Table 1. This concept was investigated in a series of 10 patients who underwent mechanical CPR and PCI to treat prolonged cardiac arrest in the catheter laboratory[82]. The average time of mechanical CPR was 43 minutes. Systolic blood pressures above 70 mmHg and diastolic blood pressures above 40 mmHg were targeted. A pigtail catheter was inserted into the right atrium via the femoral vein at the interventionists discretion to monitor CVP and to administer vasoactive drugs. The investigators aimed to keep the CVP below 25 mmHg. If this was not achieved, echocardiography was performed to exclude cardiac tamponade, the mechanical CPR device was repositioned, and inotropes or vasoconstrictors were initiated. End tidal CO_2 was measured following insertion of an endotracheal tube or a supraglottic airway with a target of >15 mmHg (>2 kPa). The SpO_2 was kept above 80%, and arterial blood gas measurement was used to guide 'normo' ventilation. Cerebral oximetry was also monitored. Vasoconstrictor infusions were used in favour of adrenaline boluses. For patients in VF, attention was directed towards opening the acutely occluded coronary artery in favour of repeated attempts at defibrillation. The protocol was simulated in training prior to its institution. Early experience identified difficulties measuring all of the parameters every 2 minutes during ongoing cardiac arrest. When the parameters were measured successfully, they regularly identified patients whose vital parameters were suboptimal.

In the AHA 'get with the guidelines registry' of 3,023 monitored cardiac arrests and 6,064 unmonitored in-hospital cardiac arrests, those who had a monitored arrest had a significantly better chance of survival based mostly on blood pressure and end tidal CO_2 monitoring [117]. The AHA recommended keeping the end tidal CO_2 above 20 mmHg and the diastolic blood pressure above 25 mmHg in their consensus statement on improving resuscitation outcomes. [118]

A group in Greece wrote a discussion document proposing the 'PERSEUS' protocol in 2019 aimed at prolonged physiological monitoring of patients in cardiac arrest [115]. They proposed mechanical CPR, and ventilating the patient with PEEP of zero, respiratory rate of 10 per min, tidal volume 6 ml//kg, 100% oxygen, I:E Ratio 1:2. In a previous observational study they had found higher airway pressure was associated with better outcomes, with a pressure of 40 – 45mmHg giving optimal outcome. They discuss the pitfalls of using end tidal CO2 to estimate cardiac output and discuss how positive pressure ventilation may be used to augment cardiac output during chest compressions. They suggest placing a CVP line with the aim being to keep the CVP below 25 mmHg. They advocate that if the CVP was low, a straight leg raise should be performed to assess volume status and then fluid be given as indicated. They suggest using optimal positioning of the mechanical CPR device and adrenaline infusions to keep the diastolic blood pressure above 40 mmHg and severe acidosis be treated immediately to prevent vasodilation and decreased central perfusion pressure (CPP).

Among over 1500 patients with out of hospital cardiac arrest in whom a venous blood gas was measured, adverse results were associated with a lower rate of survival. In

particular, patients without ROSC had a mean pH of 7.11, pCO_2 of 9.7kPa, base excess of -7 mmol/L, potassium of 4.5 mmol/L, and a lactate of 7 mmol/L. Low pH, high pCO2 and high plasma potassium concentration were predictors of poor outcome[119].

A meta-analysis of goal-directed resuscitation identified mainly animal studies but did conclude that goal- directed CPR may be superior to standard CPR, especially when end tidal CO_2 and blood pressure management were targeted [116]. It is important to emphasise that a low end-tidal CO2 may reflect inadequate ventilation rather than low cardiac, in particular when a supraglottic airway is used because of the higher airway pressures required during chest compressions and steps should be taken in these cases to place an endotracheal tube as soon as it is safe to do so.

Monitoring of the CVP allows an estimate of coronary perfusion pressure by subtracting the diastolic arterial pressure from the CVP. Ideally it should be kept above 20 mmHg.

The catheter laboratory is a unique environment in which physiological parameters can be accurately monitored during circulatory arrest. This can be used to assess the effect of interventions such as adjustment of cardiac massage technique, intravenous administration of vasoactive medications, correction of acidosis, electrolytes, and volume status, and less conventional treatments such as head-up CPR, while potentially prolonged revascularisation attempts are ongoing or preparation for extracorporeal CPR is made. Whether or not goal-directed resuscitation improves clinical outcomes, or even increases rates of ROSC, is not yet clear so firm recommendations for setting physiological parameter targets during cardiac arrest cannot be made. Nevertheless, we recommend that teams consider recording the following parameters during prolonged cardiac arrest (table 1). Green, amber and red indicate potential impact of the physiological parameters achieved during cardiac arrest on ROSC and could be used to guide future research. Clinical decisions regarding cessation of resuscitation should not be based only on these parameters.

Physiological parameters of interest

The following parameters are suggested to encourage data collection and stimulate research in a cardiac arrest management. It should be noted that they are not known markers of improved clinical outcome.

Parameter of interest	**Parameter targets**		
Systolic blood pressure (mmHg)	60	70	80
Diastolic blood pressure (mmHg)	25	30	40
Central venous pressure (mmHg)	5	10	20
Coronary perfusion pressure (mmHg)	10	15	20
End tidal CO_2 (mmHg)	10	15	20
End tidal CO_2 (kPa)	1.3	2	2.7
pH	7.0	7.1	7.2
Base excess (mmol/l)	-10	-7.5	-5
Oxygen saturation (%)	70	80	90
Cerebral oximetry (NIRS) (%)	25	30	40

Table 1. Physiological parameters of interest. Green, amber and red indicate potential impact of the physiological parameters achieved during cardiac arrest on ROSC and could be used to guide future research. Clinical decisions regarding cessation of resuscitation should not be based only on these parameters.

Recommendation

We recommend that physiological parameters are recorded at regular intervals during cardiac arrest in the catheter laboratory once mechanical CPR has been instituted.

Class IIb, level of evidence C

2.15 Is Amiodarone of use in a VF arrest in the catheter laboratory?

We sought evidence as to whether amiodarone or lidocaine may be useful for VF/pulseless VT. There is good evidence in support of Amiodarone in four large randomised trials [24-27], each demonstrating an improvement of the chance of successful cardioversion of about 10%. It must be noted that these studies are all in the out-of-hospital setting and thus there is less certainty that the results might be equivalent in the in-hospital setting or indeed in a catheter laboratory.

Amiodarone should be given as a bolus injection of 300 mg. A further dose of 150 mg may be given for recurrent or refractory VF/VT followed by an infusion of 900 mg over 24-hours. Lidocaine 1mg/kg may be used as an alternative and may have a similar efficacy [27], and there is less robust evidence regarding alternatives such as procainamide.

Recommendations

After three failed cycles of defibrillation for VF or VT without a cardiac output, a bolus of 300 mg of intravenous amiodarone should be administered.

Class I, Level of Evidence: A

2.16 The use of echocardiography during cardiac arrest

Echocardiography can help to identify the cause for the arrest and should rapidly be performed as an integral part of the resuscitation. It is important to exclude tamponade early in the resuscitative process and also potentially to repeat the echo in a prolonged arrest if the effectiveness of CPR diminishes abruptly in case of tamponade secondary to external cardiac massage or delayed onset of tamponade. Echocardiography has also been shown to reduce the time taken for pulse checks [87] by enabling visualisation of the presence or absence of organised contractions.

In patients who already have a transoesophageal echo (TOE) probe in place this has advantages compared to transthoracic echocardiography[88] in that it does not require interruptions of CPR can be performed continuously with better images, can be used to identify ROSC quickly, to look for dissection of the ascending aorta and if required can aid placement of pacing wires or the initiation of extracorporeal CPR. It is also better at monitoring the effectiveness of prolonged mechanical CPR. In addition, there may be clinicians experienced in its use available in the catheter laboratory. Thus, if it is in place already it is preferred to transthoracic echocardiography, and if it is not in place then it should be considered, especially if prolonged arrest management is being planned, allowing for the risk of damage in TOE placement of around 0.2% [88]

Recommendations

We recommend that echocardiography is performed early after cardiac arrest, particularly if immediate interventions, such as defibrillation or pacing, have failed to restore cardiac output. Transthoracic echocardiography is the most readily available modality and should generally be used first.

If a transoesophageal echo (TOE) probe is in place, this should be used in preference to TTE.

In a prolonged arrest, teams may consider the placement of a TOE probe.

Class IIa. Level of Evidence B

2.17 Fluoroscopy in order to identify a pneumothorax in an arrest in the catheter laboratory

A literature review was performed in an attempt to find cases of pneumothorax identified by fluoroscopy in a catheter laboratory and to gain an understanding of the incidence of pneumothorax causing cardiac arrest, particularly after pacing procedures, or transaxillary, transcarotid or subclavian arterial approaches.

Pneumothorax is not uncommon after attempted vessel puncture in the thorax, such as pacemaker and implantable defibrillator insertion, with an incidence of around 0.6-1,0%[32, 36].

If a pneumothorax is suspected it is straightforward to diagnose in the catheter laboratory by fluoroscopy, which has also been used to guide chest drainage in such situations [35].

In a cardiac arrest, one potential cause could be pneumothorax and, as there is immediate access to fluoroscopy, it is recommended that in a cardiac arrest with no clear cause identified, and especially if the patient is undergoing an intervention that is high risk for a pneumothorax such as pacemaker or ICD insertion, fluoroscopy is performed to exclude this as a cause.

Recommendation

If a patient arrests without a clear cause, especially in a procedure that is high risk for a pneumothorax, (pacemaker or defibrillator insertion), it is recommended that fluoroscopy be used to investigate this as a cause.

Class IIa, Level of Evidence C

2.18 How should the team balance chest compressions with attempts at percutaneous intervention in a cardiac arrest?

Interventions on the coronary arteries can is associated with occlusion, or reduced flow secondary to dissection or thrombus formation. Other complications can include no reflow and perforation. In the majority of these circumstances, a key part of the ongoing resuscitation effort will involve a further intervention to treat or reverse the underlying cause. In order to preserve cerebral perfusion until a spontaneous circulation is restored, external cardiac massage is required. Manual cardiac massage cannot be achieved at the same time as fluoroscopy due to radiation exposure for the rescuer and therefore a balance must be struck between the interventionalist and those performing external chest compressions.

The American Heart Association, the European Resuscitation Council and the Australian Guidelines all address the issue of external cardiac massage in the catheter laboratory. The AHA recommend early transfer to automated CPR devices, the ERC recommend that external cardiac massage should not be interrupted for angiography and the Australian Guidelines discuss the tension between the rescuers performing external CPR and the interventionalist wanting to continue with angiography. These statements have not translated into an agreed protocol that can be followed by the resuscitation team.

We strongly recommend using only mechanical CPR devices to administer CPR while undergoing PCI during an arrest. It is reasonable to pause manual CPR in order to perform angiography to search for a cause for the arrest, but subsequent PCI should be performed with mechanical CPR.

Recommendations

If percutaneous intervention is required during cardiac arrest, this should be performed after mechanical CPR is initiated.

Class I, Level of Evidence C

It is acceptable to pause manual CPR for less than a minute to perform diagnostic angiography to search for the cause of the arrest.

Class IIb, Level of Evidence C

2.19 Mechanical CPR devices

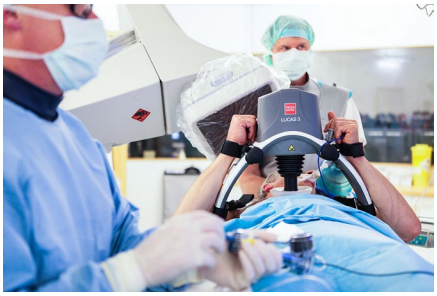
The use of mechanical CPR has been extensively investigated in at least nine randomised trials with over 12,000 patients in both out-of-hospital and in hospital arrest. Several meta-analyses exist and support the use of mechanical CPR for in hospital patients, although the evidence is less strong for use in out-of-hospital [43,46,92-94]

The AHA reviewed the feasibility of using mechanical CPR devices during PCI and identified papers where feasibility has been demonstrated in both animal [37], and human [38-41] studies. No comparative studies have examined the use of mechanical CPR devices compared with manual chest compressions during PCI procedures although, due to the inherent need to cease manual compressions during fluoroscopy, there is a clear benefit for mechanical CPR.

A number of case reports [37,38] and case series [40,41,42, 95] have reported the use of mechanical CPR devices to facilitate prolonged resuscitation in patients who have a cardiac arrest during PCI. One study demonstrated that the use of a mechanical CPR device for cardiac arrest during PCI was feasible; however, no patients survived to hospital discharge [40]. Other studies have reported good patient outcomes, including ROSC and survival to discharge with good functional outcome [37]. Of note the length of time required to perform PCI with a mechanical CPR device was around 30 minutes (ranging from 12-90 minutes), which highlights further the importance of a protocol that allows prolonged CPR while PCI is ongoing.

We are therefore strongly of the view that mechanical CPR devices are of major benefit to patients in the specialist environment of the catheter laboratory, for liberating rescuers from performing manual CPR and for the ability to perform uninterrupted CPR for at least 30 minutes while interventions are performed. In addition, we strongly advocate the immediate availability of these devices in the catheter laboratory and regular team-based training in order to be able to place these devices with a pause of less than 15 seconds [96,97].

2a Left anterior oblique caudal oblique.



2b Right anterior oblique cranial oblique


2c. Straight cranial


2d Left anterior oblique caudal oblique


2e Straight cranial

2f Straight caudal

Figure 2(a-f) demonstrate some of the Fluoroscopic projections. The Interventionist may be able to use the following projections: left anterior (LAO), cranial/caudal, right anterior (RAO), cranial/caudal, straight caudal, lateral, and straight cranial in monoplane during mechanical chest compressions (with permission from Stryker)

Recommendation

Mechanical CPR is strongly recommended if simple measures do not succeed in resuscitating the patient. We recommend that mechanical CPR is commenced after the first cycle of manual CPR.

Class I, Level of Evidence A

We recommend that all catheter laboratories have mechanical CPR immediately available in the catheter laboratory complex in case of arrest.

Class I, Level of evidence C

We recommend that teams undergo regular group training to ensure that the transfer from manual CPR to automated CPR is conducted in less than 15 seconds.

Class I, Level of evidence C

2.20 Extracorporeal CPR in the Catheter Laboratory.

The AHA and the ERC both recommend the use of ECMO to provide extracorporeal CPR (eCPR). The AHA state that 'rapid initiation of eCPR or cardiopulmonary bypass is associated with good patient outcomes in patients with hemodynamic collapse and cardiac arrest in the catheter laboratory and also the use of eCPR is feasible and associated with good outcomes when used as a bridge to coronary artery bypass grafting (AHA Class IIb, LOE C). The ERC are more equivocal, stating that very low quality evidence suggests that the use of extracorporeal life support (eCLS) can be considered as a rescue strategy if the infra-structure is available, and this should probably be preferred to the use of intra-aortic balloon pump (IABP) in such situations. The First RCT in this area called the ARREST Trial was stopped early due to the highly significant effects in favour of ECMO in OHCA. 30 patients were randomised and there were 6 survivors in the ECMO group compared to only 1 in the standard care group[114]. Furthermore, there are many case series reporting the efficacy of extracorporeal cardiopulmonary bypass [47-55] in the context of catheter laboratory based cardiac arrests. Bagai et al in 2011 reported the use of extracorporeal cardiopulmonary bypass in 39 patients in a range of situations including cardiac arrest and cardiogenic shock all in the catheter laboratory. The survival to discharge was 71% [51]. Van den Brink in 2018 [56] reported the use of extracorporeal cardiopulmonary bypass in 12 patients of whom 11 were in cardiac arrest with a survival to discharge of 67% and a 1-year survival of 42%. 9 had out of hospital arrest and a further two had in hospital arrest.

The Extracorporeal Life Support Organisation (ELSO) has published a position paper in 2018, advocating ECMO in arrests of longer than 15 minutes of duration, but centres offering ECMO are required to be looking after at least 30 patients a year and therefore will generally be located only in transplantation centres[113].

Recommendation

It is recommended that units investigate the use of ECMO as a further means of supporting patients who do not recover after cardiac arrest in the catheter laboratory and have local protocols and training in place for its effective use if it is available.

Class IIa, Level of Evidence B

2.21 Intra-Aortic Balloon Pump (IABP) insertion in the arrest situation

The evidence for the insertion of an IABP in an arrest situation was reviewed. Of note the AHA have also reviewed this evidence and concluded that whilst IABP counterpulsation increases coronary perfusion, decreases myocardial oxygen demand, and improves haemodynamics in cardiogenic shock states, it is not associated with improved patient survival. They state that the role of IABP in patients who have a cardiac arrest in the catheterisation laboratory is not known.
The IABP-SHOCK II trial which randomised nearly 600 patients who were in shock from an acute myocardial infarction did not find an improvement in the 30-day survival after the intervention [56]. This landmark study followed 13 randomised controlled trials together with meta-analyses and a Cochrane systematic review which were all unable to detect a significant improvement in 30-day survival although other small improvements were sometimes reported. [57-64] . It must be noted that although these studies were in patients with an acute myocardial infarction (rather than patients in cardiac arrest in a catheter laboratory) the IABP-SHOCK trial has led to a significant reduction in the use of IABP in cardiogenic shock in catheter laboratories.
A further small RCT looking at IABP versus control in patients who suffered a cardiac arrest with acute coronary syndrome also found no benefit [100].

There are few studies looking at the insertion of IABPs in the arrest situation, other than experimentally [65-66]. Without a spontaneous circulation to trigger the IABP, counterpulsation would be unlikely to be successful. Thus, it is concluded that there is no indication to place an IABP acutely in the cardiac arrest period in the catheter laboratory.

Recommendation

The insertion of an IABP during an arrest in the catheter laboratory or routinely in the acute post cardiac arrest period is not recommended.

Class III(Lack of benefit), Level of Evidence A

2.22 Is an Impella pump useful in an arrest?

The ERC in 2015 stated in their section on cardiac arrest in the catheter laboratory that 'There is no evidence to recommend circulatory support with the Impella pump only during cardiac arrest' and in 2021 they changed this slightly to say that they may provide circulatory support whilst performing rescue procedures but require further evaluation. They provided a single reference to support this [101] which was a case series of 8 patients who had an Impella device in an arrest, of whom 4 survived to hospital discharge. We identified a further paper documenting use in 7 patients in arrest, although only one survived, [102] and a multicentre study across 4 countries [99] of 35 patients having Impella insertion while in cardiac arrest with a 45% survival.
There have been case series and cohort studies of the use of the Impella in cardiogenic shock in adults and children [67] and in high risk PCI cases [68-70] and there is an interesting ongoing RCT currently recruiting that aims to randomise 360 patients with shock post MI to standard therapy or Impella that will report in the coming years [104].
The 2021 joint ERC and European Society of Intensive Care medicine guidelines for post resuscitation care state that 'the evidence about which type of mechanical device is superior appears inconclusive and thus their use should be decided on a case-by case basis'. [102]

Recommendation

The use of an Impella is not routinely recommended in cardiac arrest in the catheter laboratory

Class III (lack of benefit), Level of Evidence C

2.23 The identification and treatment of pericardial tamponade

Sethi et al reported the findings of the US National Inpatient Sample database from 2009 to 2013 which covers around 90% of all patients in the USA. They document 64,000 pericardiocentesis procedures and 57% of these were in unstable patients, 17% were in PCI cases, 13% in electrophysiology procedures and 14% in structural heart procedures. Thus, pericardiocentesis is performed in all types of catheter laboratory interventions [73]. As this was a database study they were unable to comment on the procedural success rate of pericardiocentesis, although the inpatient mortality in the database of these patients overall was around one in four.
Tsang et al documented a 21 year experience with a thousand pericardiocentesis procedures at the Mayo clinic, including many patients with perforation in the catheter laboratory. They report a 97% procedural success for this procedure in all settings with only a 2% major complication rate. They also reported that they saw a significant increase in the rate that clinicians left a drain during the period of the study from 25% to 75% [71].
Cho et al confirmed these findings in a report of nearly 300 echocardiographically guided pericardiocentesis procedures, with approximately 40 during PCI. They reported a 99% procedural success with a 1% complication rate. [72]

A UK observational study of 270,329 PCI procedures in the context of acute coronary syndromes describes 1013 coronary perforations (0.37%)[118]. Importantly, the adjusted odds ratios for all clinical outcomes were adversely affected by coronary perforation. The conclusion was "Coronary perforation is an infrequent event during ACS-PCI but is closely associated with adverse clinical outcomes."

The European Society of Cardiology position statement on the urgent management of cardiac tamponade [74] give a class I indication for pericardiocentesis for tamponade, preferring echocardiographic guidance where possible although fluoroscopic guidance is an acceptable alternative. If unsuccessful, surgical drainage is recommended. Of note these guidelines are mainly for non-iatrogenic causes of the tamponade

It is extremely important that all catheter laboratories have immediate access to an echo machine in order to be able to confirm or exclude tamponade in an emergency. All cardiologists who perform interventional procedures should be trained in pericardiocentesis techniques, and all catheter labs should have a dedicated and easily accessible pericardiocentesis kit, which the team are familiar with. The emergency procedures for pericardiocentesis should be familiar to all catheter laboratory staff. The pericardiocentesis/perforation kit should be stored together and include drainage equipment, coils & covered stents. There should be an agreed unit protocol as to the method of distal embolisation technique as a wide variety of options are available.

In all cases of pericardial collection, repeat TTE should be performed within 2 hours of return to the ward and often again within the following few hours. This is particularly important in the case of distal wire perforations and any case in which a perforation has apparently sealed spontaneously.

Recommendation

Pericardiocentesis should be performed for all patients with pericardial tamponade and where possible this should be with echocardiographic or fluoroscopic guidance. Surgical drainage and repair should be performed if percutaneous drainage is not successful in relieving the tamponade

Class I, Level of Evidence B

An echocardiography machine should be immediately available in all catheter laboratories in case of patient deterioration or arrest.

Class I, level of evidence C

We recommend that a repeat echocardiogram is performed to reassess the pericardial space after drain insertion to monitor for recurrence of a haemopericardium.

Class IIa, level of evidence C

2.24 Treatment of pericardial tamponade if pericardiocentesis fails

A BCIS analysis from 2006-2013 of the complete UK PCI database reported a 0.3% perforation rate with PCI [83]. This comprised of 1762 patients of whom 14% developed tamponade (246 pts) and 3% required emergency surgery (52 patients). Thus, there are roughly 250 coronary perforations per year with around 35 associated episodes of tamponade and 7 patients per year in the UK who require emergency surgery after coronary perforation.
This number is likely to have increased since 2013. Furthermore, this database does not include pacing procedures, electrophysiology or structural heart procedures. 37% of coronary perforations occurred in a unit without surgical cover. (589 coronary perforations in units without on-site surgical cover compared to 997 in units with cover.) Coronary perforations can be classified using the Ellis Classification both in the arrest and the non-arrest situation according to the significance of the defect created in the artery [85].

With regard to perforation of cardiac chambers from non PCI intervention, the National Cardiovascular Data Registry (NCDR) in the USA [84] documented 625 cardiac perforations in a 5 year period, which was one perforation for every 700 implantations of an ICD. The BHRS has provided detailed guidance in their 2016 document entitled 'Standards for Interventional Electrophysiology and catheter ablation in adults" [86].
We recommend that for coronary perforations consideration be given to heparin and antiplatelet reversal, a decision that must be balanced against the risk of producing stent thrombosis. An ACT (Activated Clotting Time) could be used to guide this decision.
We recommend there should be on site availability and experience with covered stents, embolisation coils and the ability to perform distal embolisation. There should be an agreed unit protocol as to the method of distal embolisation technique as a wide variety of options are available.
For perforation of cardiac chambers we also recommend consideration of reversal of heparin, calling for senior colleague assistance, where relevant withdrawal of the lead or wire from the perforation and echocardiographic monitoring for a tamponade.

Recommendation

For coronary perforations, consideration should be given to reversal of anticoagulants, antiplatelet medications, and glycoprotein IIb/IIIA inhibitors and an ACT should be performed.
Class IIb, Level of Evidence C

There should be on site availability and experience with covered stents, embolisation coils and the ability to perform distal embolisation. There should be an agreed unit protocol as to the method of distal embolisation technique as a wide variety of options are available.
Class IIa, Level of Evidence C

For all Cardiac Perforations, even if the patient seems stable, a decision must be taken as to whether cardiac surgical colleagues should be consulted. The threshold for surgical discussion should be low. Failure to stop the underlying cause for the tamponade should mandate emergency consultation.
Class IIa, Level of Evidence C

2.25 Surgical support

There should be access to emergency cardiothoracic surgery for all patients who have suffered a tamponade in the catheter laboratory. In units without cardiac surgical cover, an agreed written protocol must be in place in order to ensure that timely relief of a tamponade is possible. The time taken for a patient to sternotomy should be of a similar order to that possible with on-site surgical facilities where a surgical team is not on stand-by.
Options to achieve this may include rapid transfer to the cardiothoracic centre with surgeons ready to receive the patient, or utilising experienced onsite surgeons trained in emergency thoracotomy to commence relief of a tamponade while a cardiac surgeon travels to the local centre. We recommend that these protocols be documented and tested regularly to ensure equitable availability of potentially lifesaving interventions in both centres with and without on-site cardiac surgical cover.
We furthermore recommend the notification of the on call surgical team for all coronary perforations that cannot be sealed via percutaneous techniques, and all cardiac chamber perforations requiring a pericardiocentesis drain, even if they seem stable, so that the most appropriate management strategy can be agreed.

Recommendation

In units without cardiac surgical cover, an agreed written protocol must be in place in order to ensure that timely surgical relief of a tamponade is possible. The time taken for a patient to sternotomy should be of a similar order to that possible with on-site surgical facilities where a surgical team is not on stand-by.

Class IIa, Level of Evidence C

2.26 The management of pulmonary embolus

We identified papers relevant to the management of either confirmed or suspected pulmonary embolus in cardiac arrest.
In addition, the European Society of Cardiology have guidance on the treatment of Pulmonary embolus[75] and the AHA and ERC both give recommendations in this area.
It may be difficult to determine pulmonary embolus (PE) as the cause of the cardiac arrest although in-hospital arrest teams have been able to identify PE up to 85% of the time[76]. Teams may identify factors precipitating the cardiac arrest before the actual arrest which may include a high risk history such as malignancy, previous PEs or recent surgery, they may identify symptoms such as dyspnoea, tachycardia and chest pain, and there maybe signs on ECG or a distended right ventricle on echocardiography prior to the arrest.
Once the arrest has occurred, the arrest rhythm is more commonly PEA (63%) versus only 5% in VF [77]. Echocardiography during the cardiac arrest may identify a distended right ventricle with a flattened interventricular septum in cases of PE large

enough to precipitate arrest, [78] although right ventricular dilatation in arrest should be interpreted with caution. [121]

In terms of the treatment of the pulmonary embolus in the cardiac arrest Li et al published a meta-analysis in 2006 [79] of 8 papers that demonstrated that thrombolytics administered during cardiopulmonary resuscitation did improve survival, although inevitably there was also an increase in bleeding complications. In an RCT of 1000 patients with out of hospital arrests randomised to thrombolytic therapy, no improvement in survival was seen but the percentage of patients who actually had pulmonary embolus may have been low in this study.

The European Resuscitation Council recommend the use of fibrinolytics for patients suspected of arresting secondary to a massive pulmonary embolus. [2] They also recommend that cardiopulmonary resuscitation should then continue for 60-90 minutes and that a mechanical compression device may therefore be required for this. In addition, if there is return of spontaneous circulation then particular attention should be paid to identification of bleeding complications thereafter and in centres where this is available extracorporeal CPR could be considered. [122-127]

The American Heart Association gives a class IIb indication for echocardiography during cardiac arrest stating that 'if a qualified sonographer is present and use of ultrasound does not interfere with the standard cardiac arrest treatment protocol, then ultrasound may be considered as an adjunct to standard patient evaluation'.

They recommend thrombolysis with a class IIb strength of recommendation in addition to systemic anticoagulation. The AHA also mention the possibility of percutaneous mechanical thrombectomy although many units would not have access to this as it requires specialist equipment. One case series reported a successful outcome of percutaneous mechanical thrombectomy during CPR in 6 out of 7 patients.

We also discussed whether in an arrest where pulmonary embolus is suspected in the catheter laboratory pulmonary angiography should be performed, but technically this was felt to be difficult to perform. [81]

Recommendation

In confirmed or suspected acute massive pulmonary embolus in the catheter laboratory, we recommend thrombolysis and systemic anticoagulation. Cardiopulmonary resuscitation must then continue for 60 to 90 minutes.
Echocardiography may assist in making this diagnosis.

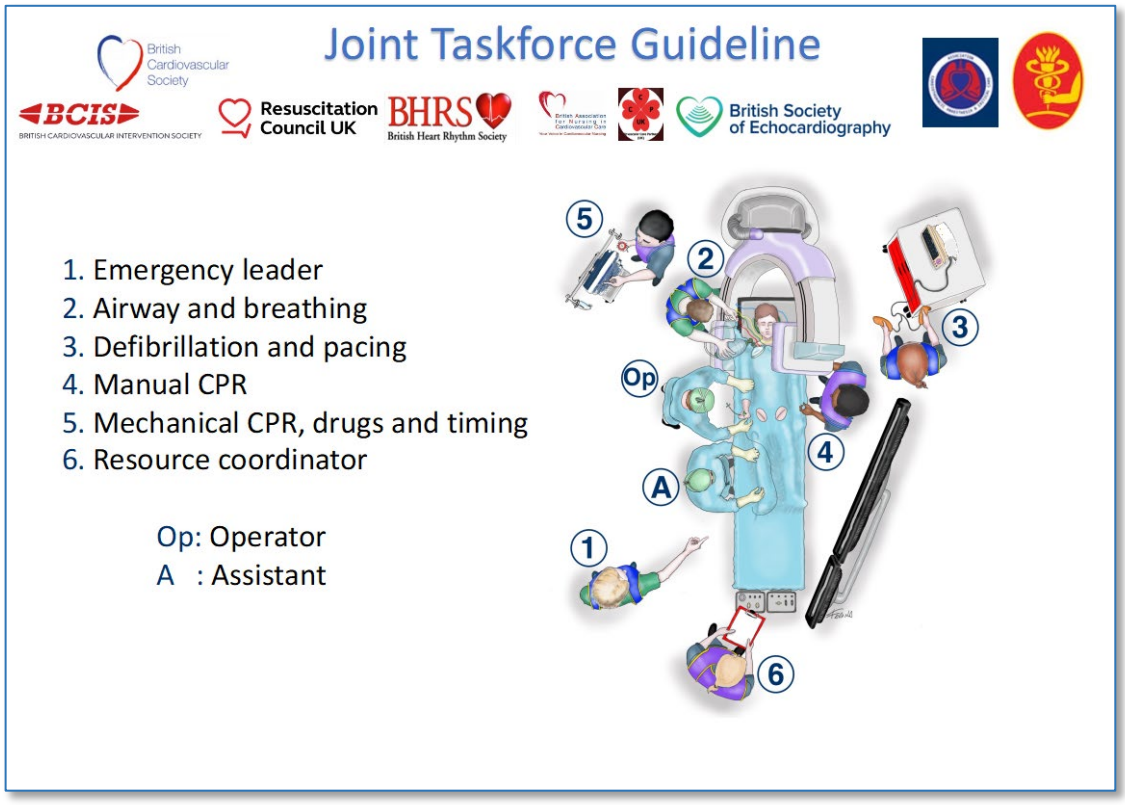
Class IIb, Level of Evidence B

2.27 Return of spontaneous circulation

Once there has been a return of spontaneous circulation a full Airway, Breathing, Circulation (ABC) examination should be performed. Angiography and echocardiography should be carried considered where appropriate. If the patient has not neurologically recovered sufficiently or their gas exchange is unfavourable it is often safer to intubate and ventilate them. Appropriate vascular access with a central line, and an arterial line, and a urinary catheter will allow cardiac monitoring and vasoactive drug use as necessary. It is important that such patients are treated in an intensive care area environment if ventilated and at least a high care area otherwise. If there has been a prolonged period of arrest then targeted temperature management has been extensively investigated especially in out of hospital arrests [120] and may help a patient who has had a prolonged arrest. However there have been no in-hospital studies to demonstrate benefit and the target temperature has not been established and therefore routine early cooling is not recommended.

Perhaps more importantly the possible longer term effects of arresting in the catheter laboratory should be considered. If the patient makes a good physical recovery, they should be fully counselled as to the events that occurred in the arrest and consideration of additional or prolonged follow up should be given to make sure that they suffer no neurological or psychological sequelae. The European Resuscitation Council and the European Society of Intensive Care Medicine have written detailed guidance in 2021 for post-resuscitation care which addresses many of these issues [103] and in addition to this there is excellent patient support at the website www.suddencardiacarrestuk.org.

4. The optimal configuration for the cardiac arrest team



In order to carry out emergency protocols efficiently, whether they be in an arrest situation or with a deteriorating patient, it is vital for all team members to know their role and responsibilities. There may be a wide variety of staff numbers and skill mixes available in the catheter laboratory area depending on the size of the institution and also the time of day or night. Therefore, there will clearly also have to be some flexibility and also additional roles that might be allocated, but we propose these 6 key roles to allow a structure for people to work towards. In addition, it is optimal that the staff members will know in advance the role that they would be expected to take in an emergency, and that this could be documented on a communication board at the start of a shift.

The operator

While the cardiologist takes the lead in the catheter lab, the main aim of our protocols is to free this person up of responsibility for resuscitation in the cardiac arrest or the emergency situation. The cardiologist should stay scrubbed at the side of the patient. They are often the person to see the emergency first, and thus must declare this early to the team but thereafter an emergency team leader should be allocated.
The cardiologist is best placed to perform the specialist interventions that may resolve the situation. They should concentrate on this aspect of the pathway and coordinate with the other staff addressing resuscitation via the team leader.

Role 1: The emergency leader

We recommend that someone other than the operating cardiologist organise the team to achieve the best outcome for the patient. We do not mandate who this person should be in terms of their discipline or qualifications, and in fact we are of the opinion that everyone who works in a catheter laboratory should be trained to be able to carry out each of the 6 key roles, although often in the day there might be another senior cardiologist who will be available to perform this role.
The role is to coordinate protocols highlighted above as the leader of the group addressing all the components of the arrest response. The leader is encouraged to have the protocol to hand on a flipchart or on a poster.
The emergency leader must make sure personnel are allocated to all required roles and will also allocate tasks to additional people , outside of the 6 key roles

Role 2: Airway and breathing

If there is any acute emergency and especially in an arrest, the scrubbed personnel will be dealing with the circulation, so another member of staff should go straight to the head of the patient to take responsibility for airway and ventilation.
For a person who is not breathing they must immediately get a bag/valve/mask at 100% oxygen and place this on the patient's face and attempt to ventilate the patient. If they are successful, then the chest will rise on both sides, and water vapour may be seen in the mask. If they are unsuccessful then an airway obstruction issue must be considered. Attempt airway manoeuvres – jaw thrust, chin lift, guedel airway and perhaps ask another person to help with squeezing the bag so you can use two hands to form a good seal around the patient's nose and mouth. We do not recommend that staff who are not fully trained in this attempt intubation. In most instances simple airway manoeuvres and airway adjuncts will suffice. A supraglottic airway is a recommended alternative to intubation. Emergency call out for anaesthetic support is mandatory in this situation.
Once air entry is established in an arrest you must coordinate 30:2 with the person performing massage or the automated CPR device. Your role also requires you to feel the trachea to see if it is central or displaced and then ask everyone to stop massage once and bag forcefully while listening bilaterally to see if you can hear a difference in breath sounds.
It is mandatory to perform these assessments in every critically ill catheter laboratory patient if you do not know the cause of their deterioration, and you must communicate that you have done this to the team leader. It is not always easy to, but if you are getting air entry from bagging but it is more difficult than you would expect, if the trachea is not central and if you bag vigorously but cannot hear breath sounds on one side then a pneumothorax or haemothorax should be suspected and this must be communicated to

the team leader.. We also recommend that fluoroscopy is performed for every arrested patient without an obvious cause for the arrest.
If a tension pneumothorax is suspected, e.g., oxygen saturations dropping and the patient complaining of being short of breath before becoming peri-arrest or arresting during a pacing procedure, then needle thoracocentesis should be performed followed by a drain or a thoracostomy.

Role 3: Defibrillation and pacing

We recommend that a single person is always allocated to this role and stays beside the defibrillator at all times, even if the rhythm is not shockable, i.e. VF or pulseless VT.
The person fulfilling role 3 should place pads on the patient wherever it is most convenient. Often they will be draped and therefore access will be limited but this will have been practiced in simulation so should not be an issue. Anterior-lateral position, an anterior-posterior position or apex-posterior positions are all acceptable.
Where the rhythm is shockable we recommend an immediate 3 stacked shocks. Once the first shock has been delivered, external cardiac massage should not be recommenced, but the rhythm assessed while the defibrillator is being charged for the next shock. If there is no ROSC and the rhythm remains shockable up to two further shocks should be delivered in rapid succession. The defibrillator operator is responsible for communicating to the team when the defibrillator is charging and before each shock.
.
If the third shock fails then further shocks may be given at 2 minute intervals as determined by the resuscitation leader and the operating cardiologist.
Most defibrillators when turned on, activate a timer, so the defibrillator operator is often the best person to time the CPR cycles.

Role 3 is also important in the two other rhythm disturbances. In asystole or extreme bradycardia without a pulse then external pacing may rapidly resolve the situation. We recommend that percussion pacing is attempted while pads are placed on the patient, and it is also important that defibrillators cannot pace and sense from the same pads and thus it is mandatory that ECG leads are placed on the patient and connected to the defibrillator prior to attempting to start pacing. We recommend that external cardiac massage is withheld until the pacing is attempted. When the pacing is activated on the defibrillator it usually defaults to the minimum amplitude, and therefore this will have to be increased to achieve capture. If capture is not achieved at maximum amplitude then it is unlikely to work unless the pads are poorly placed and the attempt can cease. If it is felt likely that the asystole or extreme bradycardia could be resolved with pacing, and both percussion and external pacing were unsuccessful then the final option would be a temporary wire to be placed in an arrest situation by the cardiologist.
Defibrillation is not required in PEA arrest but the defibrillator operator should ensure that underlying VF or asystole is not mistaken for PEA in patients with either a temporary or permanent pacemaker in place. We are aware of 3 cases when this occurred and although rare, if there is a temporary wire with pacing this can be paused to check, or if there is a permanent pacemaker then a relatively narrow QRS complex with a regular rate should raise this suspicion.

Role 4: Manual chest compressions

One person should be allocated to perform CPR. If there are very limited numbers of people in the room at night then either the cardiologist or the scrub nurse could do this but it is an important role and having an allocated person is preferable.

CPR is withheld if the arrest is VF or asystole until shocks have been administered or the external pacing has been commenced, but if this has failed then CPR must be commenced. The person performing CPR will most likely need to be on the opposite side of the table to the cardiologist, and if the table is fairly high they may need a step to stand on. Hands should be linked together and elbow straight and CPR is performed on the lower half of the sternum.
The general algorithm recommends a depth of 5-6cm and there are devices available to measure whether you are compressing adequately, but if your patient has an arterial line in place then in fact this can function as a direct measure of the quality of your CPR. If you have an arterial line then in preference to the depth recommendation you should compress the heart hard enough that you achieve a systolic pressure of 70mmHg. It is also important to note that if you have a well-functioning arterial line and you are compressing as hard as you can but you are unable to achieve a systolic pressure of 70mmHg this implies that there is a mechanical cause to the arrest such as a tamponade or a bleed, as it implies that either the heart is compressed by tamponade and cannot fill with blood to eject, or that the heart is empty of blood due to blood loss. The inability to maintain a systolic of above 70mmHg requires you to immediately notify the team leader and cardiologist.

Role 5: Mechanical CPR, drugs, timing, and vascular access

Some smaller centres or primary PCI sites in the middle of the night will not have 6 people in the catheter laboratory, but in daytime many busy catheter laboratories will have sufficient numbers of people immediately available. Therefore we considered protocols from 4 to 8 allocated members and propose 6 roles here The role of having a person in charge of mechanical CPR, drug administration, vascular access and timing we would regard as highly desirable assuming there is adequate personnel available.
This person's first role would be to immediately obtain the mechanical CPR device, turn it on and prepare it for placement after the first cycle of CPR.
Then this person can stand by the person allocated to airway and breathing and would give mediations as per protocol.
There are some key drugs that this person would need to have immediately available. Adrenaline in an arrest should be given at a dose of 1mg every 3-5 minutes. We mandate its administration after the 3rd cycle in the protocol for all arrest rhythms. It should then be given every other cycle which is again in line with the general algorithm unless we are entering a prolonged arrest situation in which case the team leader will determine whether an infusion or vasoconstrictor would be better.
If the arrest is due to a resolvable mechanical issue such as a tamponade that needs draining, it may be best to withhold the adrenaline to avoid its proarrhythmic effects and potential hypertension once the tamponade is removed which may risk further bleeding from the vessel that caused the tamponade in the first place.
The second drug in VF arrest is Amiodarone. It has been shown to have a 10% increased change of defibrillation being successful in several RCTs and is recommended in all algorithms after the 3rd cycle.
The 3rd drug to mention in cardiac arrests is atropine. It was removed from the universal algorithm in 2015 due to lack of efficacy in the arrest situation and therefore it does not appear in our arrest algorithm either. It is important to remember that it is still an important medication in bradycardia with a pulse when the patient has not arrested and it is recommended at a dose of 600mcg, repeated up to 3mg if the patient has a pulse. This issue has caused some confusion in the past.
Finally it is useful to mention that in cases of oversedation then naloxone at a dose of 400mcg repeated every 3minutes up to 10mg will immediately reverse the effects of

morphine and fentanyl, and flumazenil at 200mcg iv repeated every 30 seconds up to 3mg will equally effect a rapid reversal of midazolam and other benzodiazepines and that in a prolonged arrest infusions and bicarbonate may be required.

Role 6: Resource coordinator

There are often many members of the team available to help in an emergency situation and on simulations and observations of real world emergencies it is clear that there has to be a great deal of organisation behind the actual arrest or acute emergency. The Emergency Team leader needs to be by the patient and coordinating everything in the room but there have to be advanced lines of communication between the catheter lab, the CCU, the arrest team, the ICU, echocardiographers, and also other clinicians in the other catheter labs.

Therefore we feel this line of communication is sufficiently important to have a specific allocated role. If other personnel arrive, such as anaesthetists and surgeons then the resource coordinator can hand them lead aprons (and remind them that they must be worn) and while they are being put on then they can brief the person as to the case and what the nature of the emergency is. They may also be able to direct them to look at the communication board and to go and see the emergency leader rather than going into the room and immediately talking to the cardiologist.

It is possible that this role may fall to the radiographer who is a key member of the team and will most usually be at the foot of the table. They currently are often asked to document and provide timing in cardiac arrests and thus may be used to multitasking in this way.

5. Team training and visual aids

The ERC 2021 guidelines [2] strongly recommended that all clinicians and staff who work in the catheter laboratory be adequately trained in protocols specific to the catheter laboratory. The ERC state that ‘protocols for specific emergency procedures (initiation of mechanical CPR, emergency pacing, pericardiocentesis, ventricular assist devices) should be established. On-site emergency drills should be considered to facilitate implementation and familiarisation of the staff.’

Training in simulated catheter lab emergencies (CLEMS) is provided at the annual meeting of the British Cardiovascular Society and there is a well-established group who can assist in this training called the Cardiac Advanced Resuscitation Education group (C.A.R.E. – www.csu-als.org).

We recommend that catheter laboratory specific training be performed in every unit. ILCOR recommend the use of cognitive aids to augment the quality of the specialist resuscitation. ILCOR also recommend debriefing stating that data-driven, performance-focused debriefing of rescuers after IHCA for both adults and children is recommended[100]

Recommendation

Specific catheter laboratory focussed training should be given to all staff working in this area, including training in the protocols contained in this document, and emergency drills for mechanical CPR, emergency pacing, pericardiocentesis, and ventricular assist devices.

Class I, Level of Evidence C.

Cognitive aids may be used in the catheter laboratory to assist with the conduct of emergency protocols including the cognitive aids provided in this document.

Class IIb, Level of Evidence C.

After an unexpected cardiac arrest or a prolonged emergency situation we recommend that the senior clinician leads a debrief with his team on the same day as the emergency situation in which aspects of performance are analysed.

Class IIb, Level of Evidence C

6. Acknowledgements

We would like to thank Dr Piero Farina from the Fondazione Policlinico Universitario “Agostino Gemelli” IRCCS, in Rome who created the artwork for the Joint societies organisational poster and we would like to thank Sue Hampshire, Director of Clinical Development for the Resus Council UK who guided us through the process of creating this Resus Council approved and NICE accredited guideline.

7. Appendix 1: Competing interests statement

Name	Associations with commercial entities that provided support for the work reported in the submitted manuscript	Commercial entities that could be viewed as having an interest in the general area of the submitted manuscript	Similar financial associations involving spouse or children under 18 years of age	Non-financial associations that may be relevant to the submitted manuscript
Joseph de Bono	None	None	None	Member of council for the British Heart Rhythm Society Co-opted member of council for the Resuscitation Council UK Practising interventional cardiac electrophysiologist
Ellie Gudde	None	Abbott Vascular educational funding	None	None
Paul Swindell	None	None	None	None
Elizabeth Butterfield	None	None	None	None
Simon Ray	None	None	None	Trustee of Heart Valve Voice Immediate Past President, British Cardiovascular Society
Alan Keys	None	None	None	None
Niall O'Keeffe	None	None	None	None
Charles Deakin	None	None	None	Executive Committee, Resuscitation Council UK ALS Working Group, ILCOR
Jaydeep Sarma	None	Lead of a non-profit cardiac	None	Lead cardiology clinician, CLEMS

		catheter laboratory resuscitation educational programme based in Wythenshawe Hospital		course at Wythenshawe Hospital
Michael Lewis	None	None	None	None
R. Andrew Archbold	None	None	None	Vice President for Clinical Standards, British Cardiovascular Society
Martin Stout	None	None	None	None
Nick Curzen	None	None	None	None
Dr Thomas R Keeble	None	Advisory board member of the Zoll Medical COOL AMI EU clinical study. Received research funds to support cardiac arrest projects from Zoll. Received speaker fees from BD. (www.bd.com)	None	None
Joel Dunning	None	Co-founder of Cardiac Advanced Resuscitation Education (www.csu-als.org) which is a group that trains clinicians worldwide for emergencies in catheter laboratories, emergencies after cardiac surgery, and thoracic emergency department care.	None	Deputy Editor of www.ctsnet.org SCTS Thoracic Subcommittee ISMICS Board of Directors 2017 STS Workforce Chairman for guideline for resuscitation after cardiac surgery

8. References

[1] Merchant RM, Topjian AA, Panchal AR, Cheng A, Aziz K, Berg KM, Lavonas EJ, Magid DJ; Adult Basic and Advanced Life Support, Pediatric Basic and Advanced Life Support, Neonatal Life Support, Resuscitation Education Science, and Systems of Care Writing Groups. Part 1: Executive Summary: 2020 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2020 Oct 20;142(16_suppl_2):S337-S357.

[2] Lott C, Truhlář A, Alfonzo A, Barelli A, González-Salvado V, Hinkelbein J, Nolan JP, Paal P, Perkins GD, Thies KC, Yeung J, Zideman DA, Soar J; ERC Special Circumstances Writing Group Collaborators. European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances. Resuscitation. 2021 Apr;161:152-219.

[3] Webb JG, Solankhi NK, Chugh SK, Amin H, Buller CE, Ricci DR, Humphries K, Penn IM, Carere R. Incidence, correlates, and outcome of cardiac arrest associated with percutaneous coronary intervention. Am J Cardiol. 2002;90:1252–1254.

[4] Mehta RH, Harjai KJ, Grines L, Stone GW, Boura J, Cox D, O'Neill W, Grines CL; Primary Angioplasty in Myocardial Infarction (PAMI) Investigators. Sustained ventricular tachycardia or fibrillation in the cardiac catheterization laboratory among patients receiving primary percutaneous coronary intervention: incidence, predictors, and outcomes. J Am Coll Cardiol. 2004;43:1765–1772.

[5] Sprung J, Ritter MJ, Rihal CS, Warner ME, Wilson GA, Williams BA, Stevens SR, Schroeder DR, Bourke DL, Warner DO. Outcomes of cardiopulmonary resuscitation and predictors of survival in patients undergoing coronary angiography including percutaneous coronary interventions. Anesth Analg. 2006;102:217–224.

[6] Resuscitation : A guide for advanced rescuers. Resuscitation in Special Circumstances. The Australian and New Zealand Council on Resuscitation. Guideline 11.10 November 2011

Risks and Complicationscomplications of Coronarycoronary Angiographyangiography: Aa Comprehensivecomprehensive Reviewreview

Lockowandt U, Levine A, Strang T, Dunning J. If a patient arrests after cardiac surgery is it acceptable to delay cardiopulmonary resuscitation until you have attempted either defibrillation or pacing? Interact Cardiovasc Thorac Surg 2008;7:878-885

[8] International Liaison Committee on Resuscitation. Part 3: Defibrillation. Resuscitation 2005:67:203–211.

[9] Gazmuri RJ, Bossaert L, Mosesso V, de Paiva EF. In adult victims of ventricular fibrillation with long response times, a period of CPR before attempting defibrillation may improve ROSC and survival to hospital discharge. W68 and W177: Appendix. Circulation 2005;112:b1–b14.

[10] Wik L, Hansen TB, Fylling F, Steen T, Vaagenes P, Auestad BH, Steen PA. Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation: a randomized trial. J Am Med Assoc 2003;289:1389–1395.

[11] Jacobs IG, Finn JC, Oxer HF, Jelinek GA. CPR before defibrillation in out-of-hospital cardiac arrest: a randomized trial. Emerg Med Australas 2005;17:39–45.

[12] Cobb LA, Fahrenbruch CE, Walsh TR, Copass MK, Olsufka M, Breskin M, Hallstrom AP. Influence of cardiopulmonary resuscitation prior to defibrillation in patients with out-of-hospital ventricular fibrillation. J Am Med Assoc 1999;281:1182–1188.

[13] Stotz M, Albrecht R, Zwicker G, Drewe J, Ummenhofer W. EMS defibrillation-first policy may not improve outcome in out-of-hospital cardiac arrest. Resuscitation 2003;58:277–282.
[14] Chan PS, Krumholz HM, Nichol G, Nallamothu BK. American Heart Association National Registry of Cardiopulmonary Resuscitation Investigators. Delayed time to defibrillation after in-hospital cardiac arrest. N Engl J Med 2008:358:9–17.
[15] Spearpoint KG, McLean CP, Zideman DA. Early defibrillation and the chain of survival in 'in-hospital' adult cardiac arrest; minutes count. Resuscitation 2000:44:165–169.
[16] Addala S, Kahn JK, Moccia TF, Harjai K, Pellizon G, Ochoa A, O'Neill WW. Outcome of ventricular fibrillation developing during percutaneous coronary interventions in 19,497 patients without cardiogenic shock. Am J Cardiol. 2005;96:764–765.
[17] Miller AC, Rosati, SF, Suffredini AF, Schrump DS. A Systematic review and pooled analysis of CPR associated cardiovascular and thoracic injuries. Resuscitation 85 (2014 724-731.
[18] Richardson L, Dissanayake A, Dunning J. What cardioversion protocol for ventricular fibrillation should be followed for patients who arrest shortly post-cardiac surgery? Interact Cardiovasc Thorac Surg 2007;6:799-805.
[19] Dee R, Smith M, Rajendran K, Perkins GD, Smith CM, Vaillancourt C, Avis S, Brooks S, Castren M, Chung SP, Considine J, Escalante R, Han LS, Hatanaka T, Hazinski MF, Hung K, Kudenchuk P, Morley P, Ng KC, Nishiyama C, Semeraro F, Smyth M, Vaillancourt C; International Liaison Committee on Resuscitation Basic Life Support Task Force Collaborators. The effect of alternative methods of cardiopulmonary resuscitation - Cough CPR, percussion pacing or precordial thump - on outcomes following cardiac arrest. A systematic review. Resuscitation. 2021 Feb 11;162:73-81.
[20] Link MS, Berkow LC, Kudenchuk PJ, Halperin HR, Hess EP, Moitra VK, Neumar RW, O'Neil BJ, Paxton JH, Silvers SM, White RD, Yannopoulos D, Donnino MW. Part 7: adult advanced cardiovascular life support: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2015; 132(suppl 2):S444–S464.
[21] Lin S, Callaway CW, Shah, Prakesh S, et al. Adrenaline for out-of-hospital cardiac arrest resuscitation: A systematic review and meta-analysis of randomized controlled trials. Resuscitation 2014;85:732-40.
[22] Olasveengen TM, Wik L, Sunde K, Steen PA. Outcome when adrenaline (epinephrine) was actually given vs. not given-post hoc analysis of a randomized clinical trial. Resuscitation. 2012;83:327–332.
[23] Perkins GD, Ji C, Deakin CD, Quinn T, Nolan JP, Scomparin C, Regan S, Long J, Slowther A, Pocock H, Black JJM, Moore F, Fothergill RT, Rees N, O'Shea L, Docherty M, Gunson I, Han K, Charlton K, Finn J, Petrou S, Stallard N, Gates S, Lall R, PARAMEDIC2 Collaborators. A randomized trial of epinephrine in out-of-hospital cardiac arrest. N Engl J Med. 2018;379(8):711–21.
[24] Kudenchuk PJ, Cobb LA, Copass MK, Cummins RO, Doherty AM, Fahrenbruch CE, Hallstrom AP, Murray WA, Olsufka M, Walsh T. Amiodarone for resuscitation after out-of-hospital cardiac arrest due to ventricular fibrillation. N Engl J Med 1999;341:871-878.
[25] Dorian P, Cass D, Schwartz B, Cooper R, Gelaznikas R, Barr A. Amiodarone as compared with lidocaine for shock-resistant ventricular fibrillation. N Engl J Med 2002;346:884-890.
[26] Kudenchuk PJ, Brown SP, Daya M, Morrison LJ, Grunau BE, Rea T, Aufderheide T, Powell J, Leroux B, Vaillancourt C, Larsen J, Wittwer L, Colella MR,

Stephens SW, Gamber M, Egan D, Dorian P. Resuscitation Outcomes Consortium-Amiodarone, Lidocaine or Placebo Study (ROC-ALPS): Rationale and methodology behind an out-of-hospital cardiac arrest antiarrhythmic drug trial. Am Heart J 2014;167:653-659 e654
[27] Peter J. Kudenchuk, M.D., Siobhan P. Brown, Ph.D., Mohamud Daya, M.D., Thomas Rea, M.D., M.P.H., Graham Nichol, M.D., M.P.H., Laurie J. Morrison, M.D., Brian Leroux, Ph.D., Christian Vaillancourt, M.D., Lynn Wittwer, M.D., Clifton W. Callaway, M.D., Ph.D., James Christenson, M.D., Debra Egan, M.Sc., M.P.H., Joseph P. Ornato, M.D., Myron L. Weisfeldt, M.D., Ian G. Stiell, M.D., Ahamed H. Idris, M.D., Tom P. Aufderheide, M.D., James V. Dunford, M.D., M. Riccardo Colella, D.O., M.P.H., Gary M. Vilke, M.D., Ashley M. Brienza, B.S., Patrice Desvigne-Nickens, M.D., Pamela C. Gray, NREMT-P, Randal Gray, M.Ed., NREMT-P, Norman Seals, B.S., Ron Straight, M.Ed., and Paul Dorian, M.D., for the Resuscitation Outcomes Consortium Investigators. Amiodarone, Lidocaine, or Placebo in Out-of-Hospital Cardiac Arrest. N Engl J Med 2016; 374:1711-1722.
[28] Diana M. Cave , Raul J. Gazmuri , Charles W. Otto , Vinay M. Nadkarni , Adam Cheng , Steven C. Brooks , Mohamud Daya , Robert M. Sutton , Richard Branson , and Mary Fran Hazinski. Part 7: CPR Techniques and Devices 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2010;122:S720–S728
[29] Niemann JT, Rosborough J, Hausknecht M, Brown D, Criley JM. Cough-CPR: documentation of systemic perfusion in man and in an experimental model: a “window” to the mechanism of blood flow in external CPR. Crit Care Med. 1980;8(3):141-146.
[30] Saba SE, David SW. Sustained consciousness during ventricular fibrillation: case report of cough cardiopulmonary resuscitation. Cathet Cardiovasc Diagn. 1996;37(1):47-48.
[31] Miller B, Cohen A, Serio A, Bettock D. Hemodynamics of cough cardiopulmonary resuscitation in a patient with sustained torsades de pointes/ventricular flutter. J Emerg Med. 1994;12(5):627-632.
[32] Criley JM, Blaufuss AH, Kissel GL. Cough-induced cardiac compression. Self-administered from of cardiopulmonary resuscitation. JAMA. 1976;236(11):1246-1250.
[33] Morteza T, Ashraf S, Brener SJ. Risks and Complications of Coronary Angiography: A Comprehensive Review. Glob J Health Sci. 2012 Jan; 4(1): 65–93.
[34] Al-Hijji MA, Lennon RJ, Gulati R, El Sabbagh A, Park JY , Crusan D, Kanwar A , Behfar A, Lerman A, Holmes DR, Bell M, Singh M. Safety and Risk of Major Complications With Diagnostic Cardiac Catheterization, Cardiovascular Interventions. 2019;12 epublication https://doi.org/10.1161/CIRCINTERVENTIONS.119.007791
[35] Park GY, Oh JH, Yoon, Y, Sung, DW. J Korean Fluoroscopy Guided Percutaneous Catheter Drainage of Pneumothorax in Patients with Failed Chest Tube Drainage. Radiol Soc. 1995 Dec;33(6):889-892.
[36] Andros G, Harris RW, Dulawa LB, Oblath RW, Schneider PA, Subclavian artery catheterization: A new approach for endovascular procedures. Journal of Vascular Surgery 1994, 20, 4, 566-576,
[37] Grogaard HK, Wik L, Eriksen M, Brekke M, Sunde K. Continuous mechanical chest compressions during cardiac arrest to facilitate restoration of coronary circulation with percutaneous coronary intervention. J Am Coll Cardiol. 2007;50:1093–1094.
[38] Agostoni P, Cornelis K, Vermeersch P. Successful percutaneous treatment of an intraprocedural left main stent thrombosis with the support of an automatic mechanical chest compression device. Int J Cardiol. 2008;124:e19–e21.
[39] Steen S, Sjöberg T, Olsson P, Young M. Treatment of out-of-hospital cardiac arrest with LUCAS, a new device for automatic mechanical compression and active decompression resuscitation. Resuscitation. 2005;67:25–30.

[40] Larsen AI, Hjørnevik AS, Ellingsen CL, Nilsen DW. Cardiac arrest with continuous mechanical chest compression during percutaneous coronary intervention. A report on the use of the LUCAS device. Resuscitation. 2007;75:454–459.
[41] Wagner H, Terkelsen CJ, Friberg H, Harnek J, Kern K, Lassen JF, Olivecrona GK. Cardiac arrest in the catheterisation laboratory: a 5-year experience of using mechanical chest compressions to facilitate PCI during prolonged resuscitation efforts. Resuscitation. 2010;81:383–387.
[42] Libungan B, Dworeck C, Omerovic E. Successful percutaneous coronary intervention during cardiac arrest with use of an automated chest compression device: a case report. Ther Clin Risk Manag. 2014;10:255-257.
[43] The AutoPulse non-invasive cardiac support pump for cardiopulmonary resuscitation Medtech innovation briefing Published: 12 February 2015 by the National Institute for Health and Care Excellence. nice.org.uk/guidance/mib18
[44] Hallstrom A, Rea TD, Sayre MR et al. (2006) Manual chest compression vs use of an automated chest compression device during resuscitation following out-of-hospital cardiac arrest: a randomized trial. JAMA 295: 2620–8
[45] Wik L, Olsen JA, Persse D et al. (2014) Manual vs. integrated automatic load-distributing band CPR with equal survival after out of hospital cardiac arrest. The randomized CIRC trial. Resuscitation 85: 741–8
[46] Brooks SC, Hassan N, Bigham BL, Morrison LJ. Mechanical versus manual chest compressions for cardiac arrest. Cochrane Database Syst Rev. 2014;(2):CD007260. Published 2014 Feb 27.
[47] Ladowski JS, Dillon TA, Deschner WP, DeRiso AJ 2nd, Peterson AC, Schatzlein MH. Durability of emergency coronary artery bypass for complications of failed angioplasty. Cardiovasc Surg. 1996;4:23–27.
[48] Redle J, King B, Lemole G, Doorey AJ. Utility of rapid percutaneous cardiopulmonary bypass for refractory hemodynamic collapse in the cardiac catheterization laboratory. Am J Cardiol. 1994;73:899–900.
[49] Overlie PA. Emergency use of portable cardiopulmonary bypass. Cathet Cardiovasc Diagn. 1990;20:27–31.
[50] Shawl FA, Domanski MJ, Wish MH, Davis M, Punja S, Hernandez TJ. Emergency cardiopulmonary bypass support in patients with cardiac arrest in the catheterization laboratory. Cathet Cardiovasc Diagn. 1990;19:8–12.
[51] Bagai J, Webb D, Kasasbeh E, Crenshaw M, Salloum J, Chen J, Zhao D. Efficacy and safety of percutaneous life support during high-risk percutaneous coronary intervention, refractory cardiogenic shock and in-laboratory cardiopulmonary arrest. J Invasive Cardiol. 2011;23:141–147.
[52] Sheu JJ, Tsai TH, Lee FY, Fang HY, Sun CK, Leu S, Yang CH, Chen SM, Hang CL, Hsieh YK, Chen CJ, Wu CJ, Yip HK. Early extracorporeal membrane oxygenator-assisted primary percutaneous coronary intervention improved 30-day clinical outcomes in patients with ST-segment elevation myocardial infarction complicated with profound cardiogenic shock. Crit Care Med. 2010;38:1810–1817.
[53]Arlt M, Philipp A, Voelkel S, Schopka S, Husser O, Hengstenberg C, Schmid C, Hilker M. Early experiences with miniaturized extracorporeal life-support in the catheterization laboratory. Eur J Cardiothorac Surg. 2012;42:858–863.
[54] Huang CC, Hsu JC, Wu YW, et al. Implementation of extracorporeal membrane oxygenation before primary percutaneous coronary intervention may improve the survival of patients with ST-segment elevation myocardial infarction and refractory cardiogenic shock. Int J Cardiol. 2018;269:45-50.
[55] van den Brink, F. S., Magan, A. D., Noordzij, P. G., Zivelonghi, C., Agostoni, P., Eefting, F. D., Ten Berg, J. M., Suttorp, M. J., Rensing, B. R., van Kuijk, J. P., Klein, P., Scholten, E., & van der Heyden, J. (2018). Veno-arterial extracorporeal membrane oxygenation in addition to primary PCI in patients presenting with ST-elevation

myocardial infarction. Netherlands heart journal : monthly journal of the Netherlands Society of Cardiology and the Netherlands Heart Foundation, 26(2), 76–84.
[56] Thiele H, Zeymer U, Neumann FJ, Ferenc M, Olbrich HG, Hausleiter J, de Waha A, Richardt G, Hennersdorf M, Empen K, Fuernau G, Desch S, Eitel I, Hambrecht R, Lauer B, Böhm M, Ebelt H, Schneider S, Werdan K, Schuler G; Intraaortic Balloon Pump in cardiogenic shock II (IABPSHOCK II) trial investigators. Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock (IABPSHOCK II): final 12 month results of a randomised, open-label trial. Lancet. 2013;382:1638–1645.
[57] Ohman EM, George BS, White CJ, Kern MJ, Gurbel PA, Freedman RJ, Lundergan C, Hartmann JR, Talley JD, Frey MJ. Use of aortic counterpulsation to improve sustained coronary artery patency during acute myocardial infarction. Results of a randomized trial. The Randomized IABP Study Group. Circulation. 1994;90:792–799.
[58] Kaul U, Sahay S, Bahl VK, Sharma S, Wasir HS, Venugopal P. Coronary angioplasty in high risk patients: comparison of elective intraaortic balloon pump and percutaneous cardiopulmonary bypass support–a randomized study. J Interv Cardiol. 1995;8:199–205.
[59] Stone GW, Marsalese D, Brodie BR, Griffin JJ, Donohue B, Costantini C, Balestrini C, Wharton T, Esente P, Spain M, Moses J, Nobuyoshi M, Ayres M, Jones D, Mason D, Grines L, O'Neill WW, Grines CL. A prospective, randomized evaluation of prophylactic intraaortic balloon counterpulsation in high risk patients with acute myocardial infarction treated with primary angioplasty. Second Primary Angioplasty in Myocardial Infarction (PAMI-II) Trial Investigators. J Am Coll Cardiol. 1997;29:1459–1467.
[60] Ohman EM, Nanas J, Stomel RJ, Leesar MA, Nielsen DW, O'Dea D, Rogers FJ, Harber D, Hudson MP, Fraulo E, Shaw LK, Lee KL; TACTICS Trial. Thrombolysis and counterpulsation to improve survival in myocardial infarction complicated by hypotension and suspected cardiogenic shock or heart failure: results of the TACTICS Trial. J Thromb Thrombolysis. 2005;19:33–39.
[61] Prondzinsky R, Lemm H, Swyter M, Wegener N, Unverzagt S, Carter JM, Russ M, Schlitt A, Buerke U, Christoph A, Schmidt H, Winkler M, Thiery J, Werdan K, Buerke M. Intra-aortic balloon counterpulsation in patients with acute myocardial infarction complicated by cardiogenic shock: the prospective, randomized IABP SHOCK Trial for attenuation of multiorgan dysfunction syndrome. Crit Care Med. 2010;38:152–160.
[62] Unverzagt S, Buerke M, de Waha A, Haerting J, Pietzner D, Seyfarth M, Thiele H, Werdan K, Zeymer U, Prondzinsky R. Intra-aortic balloon pump counterpulsation (IABP) for myocardial infarction complicated by cardiogenic shock. Cochrane Database Syst Rev. 2015;3:CD007398.
[63] Sjauw KD, Engström AE, Vis MM, van der Schaaf RJ, Baan J Jr, Koch KT, de Winter RJ, Piek JJ, Tijssen JG, Henriques JP. A systematic review and meta-analysis of intra-aortic balloon pump therapy in ST-elevation myocardial infarction: should we change the guidelines? Eur Heart J. 2009;30:459–468.
[64] Lee JM, Park J, Kang J, Jeon KH, Jung JH, Lee SE, Han JK, Kim HL, Yang HM, Park KW, Kang HJ, Koo BK, Kim SH, Kim HS. The efficacy and safety of mechanical hemodynamic support in patients undergoing high risk percutaneous coronary intervention with or without cardiogenic shock: Bayesian approach network meta-analysis of 13 randomized controlled trials. Int J Cardiol. 2015;184:36–46.
[65] Iqbal MB, Al-Hussaini A, Rosser G, et al. Intra-Aortic Balloon Pump Counterpulsation in the Post-Resuscitation Period is Associated with Improved Functional Outcomes in Patients Surviving an Out-of-Hospital Cardiac Arrest: Insights from a Dedicated Heart Attack Centre. Heart Lung Circ. 2016;25(12):1210-1217.

[66] Emerman CL, Pinchak AC, Hagen JF, Hancock D. Hemodynamic effects of the intra-aortic balloon pump during experimental cardiac arrest. Am J Emerg Med. 1989;7(4):378-383.
[67] Dimas VV, Morray BH, Kim DW, et al. A multicenter study of the impella device for mechanical support of the systemic circulation in pediatric and adolescent patients. Catheter Cardiovasc Interv. 2017;90(1):124-129.
[68] Henriques JP, Remmelink M, Baan JJr, van der Schaaf RJ, Vis MM, Koch KT, Scholten EW, de Mol BA, Tijssen JG, Piek JJ, de Winter RJ. Safety and feasibility of elective high-risk percutaneous coronary intervention procedures with left ventricular support of the Impella Recover LP 2.5, Am J Cardiol, 2006, vol. 97 (pg. 990-992)
[69] Sjauw KD, Konorza T, Erbel R, et al. Supported high-risk percutaneous coronary intervention with the Impella 2.5 device the Europella registry. *J Am Coll Cardiol*. 2009;54(25):2430-2434.
[70] Vecchio S, Chechi T, Giuliani G, et al. Use of Impella Recover 2.5 left ventricular assist device in patients with cardiogenic shock or undergoing high-risk percutaneous coronary intervention procedures: experience of a high-volume center. *Minerva Cardioangiol*. 2008;56(4):391-399
[71] Tsang TS, Enriquez-Sarano M, Freeman WK, Barnes ME, Sinak LJ, Gersh BJ, Bailey KR, Seward JB. Consecutive 1127 therapeutic echocardiographically guided pericardiocenteses: clinical profile, practice patterns, and outcomes spanning 21 years. Mayo Clin Proc. 2002;77: 429–436.
[72] Cho BC, Kang SM, Kim DH, et al. Clinical and echocardiographic characteristics of pericardial effusion in patients who underwent echocardiographically guided pericardiocentesis: Yonsei Cardiovascular Center experience, 1993-2003. *Yonsei Med J*. 2004;45(3):462-468.
[73] Sethi A, Singbal Y, Kodumuri V, Prasad V. Inpatient mortality and its predictors after pericardiocentesis: An analysis from the Nationwide Inpatient Sample 2009-2013. *J Interv Cardiol*. 2018;31(6):815-825.
[74] Arsen D. Ristić, Massimo Imazio, Yehuda Adler, Aristides Anastasakis, Luigi P. Badano, Antonio Brucato, Alida L. P. Caforio, Olivier Dubourg, Perry Elliott, Juan Gimeno, Tiina Helio, Karin Klingel, Aleš Linhart, Bernhard Maisch, Bongani Mayosi, Jens Mogensen, Yigal Pinto, Hubert Seggewiss, Petar M. Seferović, Luigi Tavazzi, Witold Tomkowski, Philippe Charron, Triage strategy for urgent management of cardiac tamponade: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases, *European Heart Journal*, Volume 35, Issue 34, 7 September 2014, Pages 2279–2284
[75] Konstantinides SV, Torbicki A, Agnelli G, et al. 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J2014;35:3033–69, 69a–69k.
[76] Bergum D, Nordseth T, Mjolstad OC, Skogvoll E, Haugen BO. Causes of in-hospital cardiac arrest – incidences and rate of recognition. Resuscitation 2015;87:63–8.
[77] 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.
[78] MacCarthy P, Worrall A, McCarthy G, Davies J. The use of transthoracic echocardiography to guide thrombolytic therapy during cardiac arrest due to massive pulmonary embolism. Emerg Med J 2002;19:178–9.
[79] Li X, Fu QL, Jing XL, et al. A meta-analysis of cardiopulmonary resuscitation with and without the administration of thrombolytic agents. Resuscitation 2006;70:31–6.

[80] 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.
[81] Fava M, Loyola S, Bertoni H, Dougnac A. Massive pulmonary embolism: percutaneous mechanical thrombectomy during cardiopulmonary resuscitation. *J Vasc Interv Radiol*. 2005;16(1):119-123.
[82] Wagner H, Rundgren M, Hardig BM, Kern KB, Zughaft D, et al. A Structured Approach for Treatment of Prolonged Cardiac Arrest Cases in the Coronary Catheterization Laboratory Using Mechanical Chest Compressions. Int J Cardiovasc Res 2018 2:4.
[83] Kinnaird T, Kwok CS, Kontopantelis E, Ossei-Gerning N, Ludman P, deBelder M, Anderson R, Mamas MA; British Cardiovascular Intervention Society and the National Institute for Cardiovascular Outcomes Research. Incidence, Determinants, and Outcomes of Coronary Perforation During Percutaneous Coronary Intervention in the United Kingdom Between 2006 and 2013: An Analysis of 527 121 Cases From the British Cardiovascular Intervention Society Database. Circ Cardiovasc Interv. 2016 Aug;9(8):e003449.
[84] Hsu JC, Varosy PD, Bao H, Dewland TA, Curtis JP, Marcus GM. Cardiac perforation from implantable cardioverter-defibrillator lead placement: insights from the national cardiovascular data registry. Circ Cardiovasc Qual Outcomes. 2013 Sep 1;6(5):582-90.
[85] Ellis SG, Ajluni S, Arnold AZ, Popma JJ, Bittl JA, Eigler NL, Cowley MJ, Raymond RE, Safian RD, Whitlow PL. Increased coronary perforation in the new device era. Incidence, classification, management, and outcome. Circulation. 1994 Dec;90(6):2725-30. doi: 10.1161/01.cir.90.6.2725. PMID: 7994814.
[86] Joseph De Bono on Behlaf of the British Heart Rhythm Society Council April 2020. Standards for Interventional Electrophysiology study and catheter ablation in adults. (https://bhrs.com/wp-content/uploads/2020/04/British-Heart-Rhythm-Society-Standards-Ablation-2020-1.pdf)
[87] Clattenburg EJ, Wroe PC, Gardner K, Schultz C, Gelber J, Singh A, Nagdev A. Implementation of the Cardiac Arrest Sonographic Assessment (CASA) protocol for patients with cardiac arrest is associated with shorter CPR pulse checks. Resuscitation. 2018 Oct;131:69-73
[88] Parker BK, Salerno A, Euerle BD. The Use of Transesophageal Echocardiography During Cardiac Arrest Resuscitation: A Literature Review. J Ultrasound Med. 2019 May;38(5):1141-1151
[89] Klumbies A, Paliege R, Volkmann H. Mechanische Notfallstimulation bei Asystolie und extremer Bradykardie [Mechanical emergency stimulation in asystole and extreme bradycardia]. Z Gesamte Inn Med. 1988 Jul 1;43(13):348-52.
[90] Zeh E, Rahner E. Die manuelle extrathorakale Stimulation des Herzens. Zur Technik und Wirkung des "Präkordialschlages" [The manual extrathoracal stimulation of the heart. Technique and effect of the precordial thump (author's transl)]. Z Kardiol. 1978 Apr;67(4):299-304
[91] 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.
[92] Wang_PL, Brooks_SC. Mechanical versus manual chest compressions for cardiac arrest. Cochrane Database of Systematic Reviews 2018, Issue 8. Art. No.: CD007260. DOI: 10.1002/14651858.CD007260.pub4.
[93] 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 Jun;103:24-31. doi: 10.1016/j.resuscitation.2016.03.004. Epub 2016 Mar 11. PMID: 26976675.

[94] 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 Sep;94:91-7. doi: 10.1016/j.resuscitation.2015.07.002. Epub 2015 Jul 17. PMID: 26190673
[95] Stub D, Bernard S, Pellegrino V, Smith K, Walker T, Sheldrake J, Hockings L, Shaw J, Duffy SJ, Burrell A, Cameron P, Smit de V, Kaye DM. Refractory cardiac arrest treated with mechanical CPR, hypothermia, ECMO and early reperfusion (the CHEER trial). Resuscitation. 2015 Jan;86:88-94. doi: 10.1016/j.resuscitation.2014.09.010. Epub 2014 Oct 2. PMID: 25281189.
[96] 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.
[97] 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.
[98] Esibov A, Banville I, Chapman FW, et al. Mechanical chest compressions improved aspects of CPR in the LINC trial. Resuscitation 2015;91:116–21.
[99] Panagides V, Vase H, Shah SP, Basir MB, Mancini J, Kamran H, Batra S, Laine M, Eiskjær H, Christensen S, Karami M, Paganelli F, Henriques JPS, Bonello L. Impella CP Implantation during Cardiopulmonary Resuscitation for Cardiac Arrest: A Multicenter Experience. J Clin Med. 2021 Jan 18;10(2):339. doi: 10.3390/jcm10020339. PMID: 33477532; PMCID: PMC7831079
[100] Isman Firdaus, Yoga Yuniadi, Hananto Andriantoro, Cindy Elfira Boom, Kuntjoro Harimurti, Rochmad Romdoni, Dede Kusmana , Early Insertion of Intra-Aortic Balloon Pump after Cardiac Arrest on Acute Coronary Syndrome Patients: A Randomized Clinical Trial Cardiol Cardiovasc Med 2019; 3 (4): 193-203
[101] Vase H, Christensen S, Christiansen A, Therkelsen CJ, Christiansen EH, Eiskjær H, Poulsen SH. The Impella CP device for acute mechanical circulatory support in refractory cardiac arrest. Resuscitation. 2017 Mar;112:70-74.
[102] Kamran H, Batra S, Venesy DM, Patten RD, Waxman S, Pyne C, Shah SP. Outcomes of Impella CP insertion during cardiac arrest: A single center experience. Resuscitation. 2020 Feb 1;147:53-56.
[103] Nolan JP, Sandroni C, Böttiger BW, Cariou A, Cronberg T, Friberg H, Genbrugge C, Haywood K, Lilja G, Moulaert VRM, Nikolaou N, Olasveengen TM, Skrifvars MB, Taccone F, Soar J. European Resuscitation Council and European Society of Intensive Care Medicine guidelines 2021: post-resuscitation care. Intensive Care Med. 2021 Apr;47(4):369-421. doi: 10.1007/s00134-021-06368-4. Epub 2021 Mar 25.
[104] Udesen NJ, Møller JE, Lindholm MG, Eiskjær H, Schäfer A, Werner N, Holmvang L, Terkelsen CJ, Jensen LO, Junker A, Schmidt H, Wachtell K, Thiele H, Engstrøm T, Hassager C; DanGer Shock investigators. Rationale and design of DanGer shock: Danish-German cardiogenic shock trial. Am Heart J. 2019 Aug;214:60-68.
[105] Ramirez FD, Sadek MM, Boileau I, Cleland M, Nery PB, Nair GM, Redpath CJ, Green MS, Davis DR, Charron K, Henne J, Zakutney T, Beanlands RSB, Hibbert B, Wells GA, Birnie DH. Evaluation of a novel cardioversion intervention for atrial fibrillation: the Ottawa AF cardioversion protocol. Europace. 2019 May 1;21(5):708-715. doi: 10.1093/europace/euy285.
[106] January CT, Wann LS, Alpert JS, Calkins H, Cigarroa JE, Cleveland JC Jr, Conti JB, Ellinor PT, Ezekowitz MD, Field ME, Murray KT, Sacco RL, Stevenson WG, Tchou PJ, Tracy CM, Yancy CW; ACC/AHA Task Force Members. 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 practice guidelines and the Heart Rhythm Society. Circulation. 2014 Dec 2;130(23):e199-267.
[107] Kerber RE, Grayzel J, Hoyt R, Marcus M, Kennedy J. Transthoracic resistance in human defibrillation. Influence of body weight, chest size, serial shocks, paddle size and paddle contact pressure. Circulation. 1981 Mar;63(3):676-82.
[108] Sirna SJ, Ferguson DW, Charbonnier F, Kerber RE. Factors affecting transthoracic impedance during electrical cardioversion. Am J Cardiol. 1988 Nov 15;62(16):1048-52.
[109] Ramirez FD, Fiset SL, Cleland MJ, Zakutney TJ, Nery PB, Nair GM, Redpath CJ, Sadek MM, Birnie DH. Effect of Applying Force to Self-Adhesive Electrodes on Transthoracic Impedance: Implications for Electrical Cardioversion. Pacing Clin Electrophysiol. 2016 Oct;39(10):1141-1147.
[110] Greif R, Bhanji F, Bigham BL, Bray J, Breckwoldt J, Cheng A, Duff JP, Gilfoyle E, Hsieh MJ, Iwami T, Lauridsen KG, Lockey AS, Ma MH, Monsieurs KG, Okamoto D, Pellegrino JL, Yeung J, Finn JC, Baldi E, Beck S, Beckers SK, Blewer AL, Boulton A, Cheng-Heng L, Yang CW, Coppola A, Dainty KN, Damjanovic D, Djärv T, Donoghue A, Georgiou M, Gunson I, Krob JL, Kuzovlev A, Ko YC, Leary M, Lin Y, Mancini ME, Matsuyama T, Navarro K, Nehme Z, Orkin AM, Pellis T, Pflanzl-Knizacek L, Pisapia L, Saviani M, Sawyer T, Scapigliati A, Schnaubelt S, Scholefield B, Semeraro F, Shammet S, Smyth MA, Ward A, Zace D. Education, Implementation, and Teams: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation. 2020 Nov;156:A188-A239.
[111] Soar J, Mitchell S, Gwinnutt C, Bullock I, Cummin V. Resus Council UK. Publication: Guidelines development process manual. https://www.resus.org.uk/library/publications/publication-guidelines-development-process-manual (Accessed 22nd May 2021)
[112] 2017 European Society of Cardiology. Governing policies and procedures for the writing of ESC Clinical Practice guidelines. https://www.escardio.org/static-file/Escardio/Guidelines/About/Recommendations-Guidelines-Production.pdf (accessed 22nd of May 2021)
[113] Abrams D, Garan AR, Abdelbary A, Bacchetta M, Bartlett RH, Beck J, Belohlavek J, Chen YS, Fan E, Ferguson ND, Fowles JA, Fraser J, Gong M, Hassan IF, Hodgson C, Hou X, Hryniewicz K, Ichiba S, Jakobleff WA, Lorusso R, MacLaren G, McGuinness S, Mueller T, Park PK, Peek G, Pellegrino V, Price S, Rosenzweig EB, Sakamoto T, Salazar L, Schmidt M, Slutsky AS, Spaulding C, Takayama H, Takeda K, Vuylsteke A, Combes A, Brodie D; International ECMO Network (ECMONet) and The Extracorporeal Life Support Organization (ELSO). Position paper for the organization of ECMO programs for cardiac failure in adults. Intensive Care Med. 2018 Jun;44(6):717-729.
[114] Yannopoulos D, Bartos J, Raveendran G, Walser E, Connett J, Murray TA, Collins G, Zhang L, Kalra R, Kosmopoulos M, John R, Shaffer A, Frascone RJ, Wesley K, Conterato M, Biros M, Tolar J, Aufderheide TP. 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 Dec 5;396(10265):1807-1816.
[115] Chalkias A, Arnaoutoglou E, Xanthos T. Personalized physiology-guided resuscitation in highly monitored patients with cardiac arrest-the PERSEUS resuscitation protocol. Heart Fail Rev. 2019 Jul;24(4):473-480.
[116] Chopra AS, Wong N, Ziegler CP, Morrison LJ. Systematic review and meta-analysis of hemodynamic-directed feedback during cardiopulmonary resuscitation in cardiac arrest. Resuscitation. 2016 Apr;101:102-7.

[117] Sutton RM, French B, Meaney PA, Topjian AA, Parshuram CS, Edelson DP, Schexnayder S, Abella BS, Merchant RM, Bembea M, Berg RA, Nadkarni VM; American Heart Association's Get With The Guidelines–Resuscitation Investigators. Physiologic monitoring of CPR quality during adult cardiac arrest: A propensity-matched cohort study. Resuscitation. 2016 Sep;106:76-82.
[118] Kinnaird T, Kwok CS, Davies R, Calvert PA, Anderson R, Gallagher S, Sirker A, Ludman P, deBelder M, Stables R, Johnson TW, Kontopantelis E, Curzen N, Mamas M; British Cardiovascular Intervention Society and the National Institute for Cardiovascular Outcomes Research. Coronary perforation complicating percutaneous coronary intervention in patients presenting with an acute coronary syndrome: An analysis of 1013 perforation cases from the British Cardiovascular Intervention Society database. Int J Cardiol. 2020 Jan 15;299:37-42. doi: 10.1016/j.ijcard.2019.06.034. Epub 2019 Jun 14.
[118] Meaney PA, Bobrow BJ, Mancini ME, Christenson J, de Caen AR, Bhanji F, Abella BS, Kleinman ME, Edelson DP, Berg RA, Aufderheide TP, Menon V, Leary M; CPR Quality Summit Investigators, the American Heart Association Emergency Cardiovascular Care Committee, and the Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation. Cardiopulmonary resuscitation quality: [corrected] improving cardiac resuscitation outcomes both inside and outside the hospital: a consensus statement from the American Heart Association. Circulation. 2013 Jul 23;128(4):417-35
[119] Corral Torres E, Hernández-Tejedor A, Suárez Bustamante R, de Elías Hernández R, Casado Flórez I, San Juan Linares A. Prognostic value of venous blood analysis at the start of CPR in non-traumatic out-of-hospital cardiac arrest: association with ROSC and the neurological outcome. Crit Care. 2020 Feb 22;24(1):60
[120] Dankiewicz J, Cronberg T, Lilja G, Jakobsen JC, Levin H, Ullén S, Rylander C, Wise MP, Oddo M, Cariou A, Bělohlávek J, Hovdenes J, Saxena M, Kirkegaard H, Young PJ, Pelosi P, Storm C, Taccone FS, Joannidis M, Callaway C, Eastwood GM, Morgan MPG, Nordberg P, Erlinge D, Nichol AD, Chew MS, Hollenberg J, Thomas M, Bewley J, Sweet K, Grejs AM, Christensen S, Haenggi M, Levis A, Lundin A, Düring J, Schmidbauer S, Keeble TR, Karamasis GV, Schrag C, Faessler E, Smid O, Otáhal M, Maggiorini M, Wendel Garcia PD, Jaubert P, Cole JM, Solar M, Borgquist O, Leithner C, Abed-Maillard S, Navarra L, Annborn M, Undén J, Brunetti I, Awad A, McGuigan P, Bjørkholt Olsen R, Cassina T, Vignon P, Langeland H, Lange T, Friberg H, Nielsen N; TTM2 Trial Investigators. Hypothermia versus Normothermia after Out-of-Hospital Cardiac Arrest. N Engl J Med. 2021 Jun 17;384(24):2283-2294.
[121] Aagaard R, Caap P, Hansson NC, Botker MT, Granfeldt A, Lofgren B. Detection of Pulmonary Embolism During Cardiac Arrest-Ultrasonographic Findings Should Be Interpreted With Caution. Crit Care Med 2017; 45(7): e695-e702.
[122] 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(6): 1167-75.
[123]Javaudin F, Lascarrou JB, Esquina H, Baert V, Hubert H, Leclère B. Improving identification of pulmonary embolism-related out-of-hospital cardiac arrest to optimize thrombolytic therapy during resuscitation. Crit Care 2019; 23(1): 409.
[124]Konstantinides SV, Meyer G. The 2019 ESC Guidelines on the Diagnosis and Management of Acute Pulmonary Embolism. Eur Heart J 2019; 40(42): 3453-5.
[125]Alqahtani F, Munir MB, Aljohani S, Tarabishy A, Almustafa A, Alkhouli M. Surgical Thrombectomy for Pulmonary Embolism: Updated Performance Rates and Outcomes. Tex Heart Inst J 2019; 46(3): 172-4.
[126]Rousseau H, Del Giudice C, Sanchez O, et al. Endovascular therapies for pulmonary embolism. Heliyon 2021; 7(4): e06574.

[127]O'Malley TJ, Choi JH, Maynes EJ, et al. Outcomes of extracorporeal life support for the treatment of acute massive pulmonary embolism: A systematic review. Resuscitation 2020; 146: 132-7.
[128] Sinning C, Ahrens I, Cariou A, Beygui F, Lamhaut L, Halvorsen S, Nikolaou N, Nolan JP, Price S, Monsieurs K, Behringer W, Cecconi M, Van Belle E, Jouven X, Hassager C, Sionis A, Qvigstad E, Huber K, De Backer D, Kunadian V, Kutyifa V, Bossaert L. The cardiac arrest centre for the treatment of sudden cardiac arrest due to presumed cardiac cause: aims, function, and structure: position paper of the ACVC association of the ESC,EAPCI, EHRA, ERC, EUSEM, and ESICM. European Heart Journal: Acute Cardiovascular Care doi:10.1093/ehjacc/zuaa024