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New Developments in Cardiac Arrest Management

Matthias L. Riess, MD, PhD^{a,b,c,*}

^aDepartment of Anesthesiology, Vanderbilt University, 1161 21st Avenue South, T4202 MCN, Nashville, TN 37232-2520, USA

^bDepartment of Pharmacology, Vanderbilt University, 2220 Pierce Avenue, Nashville, TN 37232, USA

^cDepartment of Anesthesiology, TVHS VA Medical Center, 1310 24th Avenue South, Nashville, TN 37212, USA

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INTRODUCTION

Cardiac arrest continues to be a substantial health care problem worldwide because of its combination of high frequency and low survival. In the United States alone, an estimated 350,000 out-of-hospital cardiac arrests (OHCA) occur every year [1], with similarly high numbers in Europe [2]. Despite significant regional variations, overall chances for functionally favorable survival remain low, between 5% and 10% [3,4]. According to the Cardiac Arrest Registry to Enhance Survival, patients with OHCA are on average 64 years old, 61% are male, and 22% are pronounced dead before arrival at the hospital [5]. Only about one-third receive cardiopulmonary resuscitation (CPR) by by-standers, and less than 4% are treated with an automated external defibrillator before the arrival of emergency medical service personnel. A cardiac cause has been described in 70% to 85% of OHCA; the remainder is a consequence of noncardiac causes, such as trauma, drowning, overdose, asphyxia, electrocution, primary respiratory arrests, or other etiologies. The highest survival rates are achieved when the arrest is witnessed by a bystander and the patient has a shockable rhythm, such as pulseless ventricular tachycardia or ventricular fibrillation (VF). Thus, predictors of OHCA survival (Table 1) are (1) witnessed by a bystander, (2) witnessed by emergency medical service, (3) bystander CPR, (4) shockable cardiac rhythm (only 23% of all OHCA), and (5) return of spontaneous circulation (ROSC) in the field [6].

The situation for in-hospital cardiac arrest (IHCA) differs only marginally. According to the British registry [7], the incidence of IHCA is estimated to be 1.6 per 1000 hospital

*Department of Anesthesiology, 1161 21st Avenue South, T4202 MCN, Nashville, TN 37232-2520. matthias.riess@vanderbilt.edu.

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admissions, with 17% being in a shockable rhythm versus 72% in asystole or pulseless electrical activity. Overall survival rates have improved from 10% in the 1950s to 17% in the 1990s. A large-scale study, however, has reported a plateau of the overall average survival-to-discharge rate at 18% [8], comprising 49% for shockable rhythms, but only 11% for non-shockable rhythms. About half of the IHCA occur on regular wards, between 5% and 10% in intensive care and coronary care units. Predictors for survival of IHCA (Table 2) are (1) witnessed arrest, (2) initial rhythm shockable, (3) epinephrine dose administered within 2 minutes of arrest, and (4) arrest in the intensive care unit rather than on the ward [9].

Perioperative cardiac arrests are rare. Their incidence is estimated to be about 7.4 per 10,000 anesthetics with the risk of death from cardiac arrest attributable to anesthesia being 0.6 per 10,000 anesthetics [10]. Risk factors for perioperative cardiac arrest include older age; male gender; American Society of Anesthesiologists physical status of IV or higher; upper abdominal, thoracic, spine, and/or emergency surgery; longer and after hours procedures; and general versus nongeneral anesthesia [10]. Although the cause of OHCA and even IHCA is often unknown the list of probable causes for cardiac arrest in the perioperative period is more succinct (Box 1) [11].

Overall, the public health burden of cardiac arrest remains immense. A reasonable analogy of the impact of cardiac arrest on public health are two full Boeing 747 aircraft crashing with total loss of life per day on each continent [2]. With recent discoveries and insights into improved resuscitation technique, overall functionally favorable survival rate could be improved and a proportion of this premature death prevented. This article highlights some important recent developments in the area of cardiocerebral resuscitation after cardiac arrest.

HISTORY OF CARDIOPULMONARY RESUSCITATION

In 1958, Safar [12] described effective pulmonary ventilation by mouth-to-mouth breathing without an artificial airway. In 1960, Kouwenhoven and co-workers [13] for the first time documented 14 cardiac arrest victims who were successfully treated with closed chest cardiac massage and survived. In the same year, the Maryland Medical Society introduced the combination of chest compressions and rescue breathing, the foundation of what is recognized today as CPR [14]. In 1962, Lown and colleagues [15] described for the first time direct-current, monophasic waveform defibrillation. Four years later, the American Heart Association (AHA) developed the first CPR guidelines, which since then have been updated regularly. The process of generating CPR guide-lines has evolved over several decades. The AHA, the European Resuscitation Council, the Heart and Stroke Foundation of Canada, the Australian Resuscitation Council, and others have been comprehensively evaluating the available resuscitation science and collaborating extensively for decades, which has led to similar CPR guidelines published in different regions of the world.

The most current (2015) AHA Advanced Cardiac Life Support (ACLS) guidelines (www.heart.org/eccguidelines) stress several key points (Box 2, Fig. 1) [16]: (1) early defibrillation and early chest compressions; (2) high-quality compressions, that is, a rate of 100–120 compressions per minute, a compression depth of at least 2 inches, and complete chest recoil; (3) no unnecessary interruptions of chest compressions; and (4) a ventilation

rate no greater than 8 to 10 breaths per minute. The scientific basis for these recommendations is outlined next.

EARLY CHEST COMPRESSIONS AND EARLY DEFIBRILLATION

To achieve ROSC, early CPR and defibrillation are critical for survival when a shockable rhythm is encountered. Defibrillation is the best treatment of VF cardiac arrest because with immediate CPR survival rates decrease by only 3% to 4% per minute, whereas without immediate CPR this rate is 7% to 10% per minute. Therefore, CPR can double to triple survival, not only by providing blood flow to vital organs, but also by prolonging VF and delaying the onset of asystole, thus extending the window for successful defibrillation [17].

HIGH-QUALITY CHEST COMPRESSIONS

The highest rates of ROSC and survival are achieved at chest compression rates between 100 and 120 per minute [18]. Compression depth is linearly related to survival, with chest compressions of only 1 inch depth instead of 2 inches resulting in 50% lower survival rates as reported by Edelson and colleagues [19]. Compression depths of more than 2.4 inches, however, should be avoided because of increased risk for injury [20]. Equally important as sufficient compression depth is complete recoil to a neutral sternum position on the upstroke of CPR. Incomplete chest recoil leads to increased intrathoracic pressure, which in turn increases intracerebral pressure and decreases filling of the right heart with subsequently lower cardiac output, both of which synergistically reduce cerebral blood flow [21,22]. Incomplete recoil or “leaning” is prevented with appropriate training [23] and an optimal hand technique [22].

PRIORITIZING CHEST COMPRESSIONS AND MINIMIZING PAUSES

Blood flow is now prioritized over ventilation. The study by Edelson and colleagues [19] suggests that chest compressions should not be interrupted for more than 10 seconds; preshock pauses of 30 seconds, for example, led to a 60% lower success of a subsequent shock compared with pauses less than 10 seconds. This resulted in a fundamental guideline change from A-B-C to C-A-B: instead of starting with airway assessment (A) and breathing (B), the provider should initiate chest compressions (C) immediately. Biphasic shocks should be delivered only once at a time instead of as previously recommended as a stack of three [16]. When shocks were limited to one time, allowing for immediate continuation of chest compressions instead of prolonged pauses, Rea and colleagues [24] reported a 24% increase in ROSC at hospital arrival and a 40% increase in hospital discharge and 1-year survival. The rationale is that the diminishing probability of a second or third shock to be successful, after the first shock has failed, no longer justifies prolonged pauses.

AVOID HYPERVENTILATION

Just as incomplete recoil should be avoided to not lead to continuously increased intrathoracic pressure, excessive positive pressure ventilation can contribute significantly to a decrease in return of blood to the right heart and to increased intracerebral pressure. Aufderheide and colleagues [25] could show in their landmark porcine study increased

intrathoracic pressure over time, rather than the hypocapnia associated with hyperventilation, is responsible for a dramatically reduced survival after cardiac arrest and CPR with hyperventilation. Thus, hyperventilation should be avoided by limiting ventilations to 8 to 10 breaths per minute; metronomes may be considered to guide normoventilation.

ROLE OF ENDTIDAL CARBON DIOXIDE PARTIAL PRESSURE IN RESUSCITATION

Endtidal (ET)CO₂ is the partial pressure of exhaled CO₂ at the end of expiration and determined by CO₂ production, pulmonary blood flow, and alveolar ventilation [16]. Waveform capnography visualizes the actual CO₂ waveform during inspiration and expiration. In contrast to stable cardiovascular conditions where ETCO₂ partial pressure serves to differentiate between hypoventilation, normoventilation, and hyperventilation, the primary determinant of ETCO₂ during and after CPR is pulmonary blood flow, and its value becomes more an indicator of passive cardiac output during chest compressions or active cardiac output after ROSC. Consequently, the ACLS guidelines emphasize the need to improve the quality of chest compressions for ETCO₂ values less than 10 mm Hg (see Fig. 1) [16]; conversely, a sustained increase to greater than 40 mm Hg, especially when abrupt, may be an indicator of ROSC. In intubated and normoventilated patients, failure to achieve more than 10 mm Hg ETCO₂ after 20 minutes CPR might be taken into consideration as one element of a multimodal approach in the decision process when to stop resuscitative efforts; ETCO₂, however, should never be used alone or in nonintubated patients [16].

ADJUNCT DEVICES

Even with the best technique, external chest compressions can only produce about 20% to 30% of the normal cardiac output [26,27]. Limited endurance of the rescuer [28] and the difficulty of providing high-performance CPR during patient transport [29] contributes to decreasing quality of manual chest compressions over time. Two newer adjunct devices work synergistically by more efficiently using the heart and chest to pump blood during CPR through lowering intrathoracic and intracranial pressure to improve cardiac and cerebral blood flow during chest compressions.

ACTIVE COMPRESSION-DECOMPRESSION

A case report of a son rescuing his father after cardiac arrest by using a toilet plunger [30] inspired Lurie [31] to the development of active compression-decompression (ACD), where a suction cup is used to actively decompress the chest after a preceding compression, thus lowering intrathoracic pressure faster, and improving cardiac output and generated systolic pressure when used alone [32]. It is marketed as ResQPUMP (ZOLL Medical Corporation, Chelmsford, MA) in different countries, and recently received Food and Drug Administration approval in the United States.

AUTOMATED CHEST COMPRESSION

Automated chest compressors (Fig. 2) are programmed to not exceed normal sternum levels during decompression. The Lund University Cardiopulmonary Assist System (LUCAS-2; PhysioControl, Redmond, WA) is a newer version of the original LUCAS, is battery-powered, and runs with a constant compression rate of 100 min^{-1} , a force of 600 N, a compression depth of 2 inches, and 1:1 duty cycle [33]. In comparison, AutoPulse (ZOLL Medical Corporation) is a battery-powered compression device that uses a circumferential band to squeeze the entire chest as developed by Halperin and colleagues [34,35]. AutoPulse delivers 80 compressions per minute with an even distribution of the compression across the entire chest, while the compression depth can be adjusted to the individual patient's chest diameter. For an overview of more mechanical devices from different manufacturers, please refer to recent review articles in this area [33,36]. Earlier studies have reported a higher incidence of rib and sternal fractures after manual ACD versus conventional manual CPR [37]; other case reports have described rare complications, such as tension pneumothorax [38] and liver [39] or splenic lacerations [40]. Nevertheless, neither of these are thought unique to automated versus conventional manual chest compressions [41]. There seem to be, however, subtle differences among devices; Koster and colleagues [42] reported that circumferential compression devices had a higher incidence of visceral damage than LUCAS when compared with manual CPR controls. Clear advantages of mechanical CPR devices are reliable compressions of constant rate, depth, and location, thus avoiding inconsistencies of manual compressions, provider fatigue [43], and injury [44]; at the same time, personnel is freed up for other important tasks (Box 3) [45].

IMPEDANCE THRESHOLD DEVICE

Most importantly, however, the concept of ACD works best in tandem with an impedance threshold device (ITD; Fig. 3; commercially available as ResQPOD, ZOLL Medical Corporation) [46] and vice versa. The ITD creates a small negative intrathoracic pressure during passive chest recoil or active decompression to increase venous return to the heart. The ITD is placed between a tight-sitting face mask or the endotracheal tube on one end, and the ventilation bag or ventilator on the other end as early as possible during CPR. The ITD opens for spontaneous or for positive pressure ventilation and for negative intrathoracic pressures of minus 10 mm Hg or lower. Enhancing venous return to the right heart, ACD and ITD synergistically result in largely improved systemic blood pressures, decreased intracranial pressure, and improved cerebral perfusion during CPR in animal models [46] and in patients [47]. Their combination has been shown to improve neurologically favorable survival in patients with cardiac arrest by 50% on hospital discharge with modified Rankin Scale scores comparable with the control group 1 year later [48].

When used in conjunction with conventional manual CPR alone [49–51], the generation of negative intrathoracic pressure in the presence of an ITD largely depends on the intrinsic elastic recoil of the chest and the quality of CPR. Fractured ribs, for example, or a rigid, noncompliant chest reduce elastic recoil. Moreover, limited recoil through leaning has detrimental physiologic effects on venous return and intracranial pressure as described previously [21,22]. Although the Resuscitation Outcomes Consortium trial with 8718

patients randomly assigned to conventional manual CPR with a sham or an active ITD could not show a benefit of the ITD alone across all-comers [51], a recent post hoc analysis of the data showed a clear benefit in those patients treated with high-quality CPR with regards to a compression rate and depth recommended by the guidelines as opposed to those who did receive CPR outside of the current recommendations [52].

Whether a continuous negative intrathoracic pressure through an intrathoracic pressure regulator by combining an ITD with a vacuum suction to maintain a constant intrathoracic vacuum between minus 5 and 10 mm Hg except during intermittent positive pressure ventilations is an improvement over the ITD during CPR, remains the subject of ongoing animal studies [53–55]. First promising results in humans, however, have been reported during coronary bypass graft surgery [56] and hemorrhagic shock [57].

IMPORTANT CHANGES FROM 2010 AMERICAN HEART ASSOCIATION GUIDELINES

The 2015 AHA guidelines serve less as a comprehensive revision than an update of the 2010 AHA guidelines for CPR and have to be interpreted in this context [58]. Moreover, as of 2015, the guidelines have now moved from periodic revisions and updates every 5 years to a continuously updated World Wide Web–based format (<https://eccguidelines.heart.org/>) enabling a more rapid translation of new scientific discoveries into daily patient care. Systems of care and continuous quality improvement are important new components that emphasize integrated structures and processes essential for OHCA and IHCA resuscitation capable of measuring quality and improving patient outcomes [59]. Another new feature is education of lay rescuers and health care providers to better facilitate implementation of the most current guidelines into clinical practice [60].

Pertinent changes in the 2015 ACLS guidelines include the addition of an upper limit of 120 compressions per minute [16] resulting from one large registry study that found an association between fast compression rates and inadequate compression depth [61]. Recognizing potential differences in pathophysiologic diseases between shockable and nonshockable initial presenting rhythms leads to differential recommendations with regard to timing of epinephrine administration [58]. The standard dose of 1 mg epinephrine every 3 to 5 minutes remained unchanged, even after treatment of local anesthetic toxicity with Intralipid [62], because no human data to date support a modification in the AHA ACLS recommendations; the latter contrasts with recommendations by other organizations, such as the American Society of Regional Anesthesia and Pain Medicine, which recommend a reduction of epinephrine boluses to less than 1 µg/kg [63]. Epinephrine should be administered as soon as feasible in case of nonshockable compared with shockable rhythms where timely defibrillation remains the treatment of choice. Furthermore, vasopressin has been removed from the algorithm as a vasopressor therapy (see Fig. 1) [16] because of its equivalent effect when compared with epinephrine and the desire to simplify the algorithm. Finally, data indicating that low ETCO_2 in intubated patients after 20 minutes of CPR is strongly associated with failure of resuscitation, led to the inclusion of this important

parameter as a prognosticator; it should, however, not be used in isolation or in nonintubated patients [16].

New guidelines also exist for the prevention and management of resuscitative emergencies related to opioid and other drug overdoses including local anesthetic systemic toxicity, during the second half of pregnancy, cardiac arrests caused by pulmonary embolism, and those during percutaneous coronary interventions [62].

With regards to mechanical devices, the guidelines have undergone small, but important revisions [64]. Although a clear benefit with the use of automated piston devices versus manual chest compressions has not been found, they provide a reasonable alternative when the delivery of high-quality manual compressions may be challenging or dangerous for the provider, such as with limited personnel, prolonged CPR, in moving ambulances, or during ongoing CPR under fluoroscopy during coronary interventions. In the latter case, the radiolucent backboard of LUCAS seems superior to the one in the AutoPulse device. Furthermore, the combination of an ITD with ACD-CPR was upgraded to a reasonable alternative to conventional CPR when available and used by properly trained personnel [64].

PERIOPERATIVE CONSIDERATIONS

Cardiac arrest during surgery is unique because it is not only witnessed and can be immediately treated, but also because the patient's history is usually well known and vital signs are continuously assessed. Depending on the circumstances, there is a short list of possible causes (see Table 2) that may have led to the perioperative circulatory collapse and that can be treated specifically [11]. Plethysmography or, when available, arterial line traces and $ETCO_2$ can help assess pulse pressure and rate and cardiac output, respectively; intravenous access and most resuscitative drugs are immediately available in the operating room. All of these allow for a more etiology-specific and immediate management, which largely contributes to an overall higher survival rate [10] than for OHCA and IHCA.

EXPERIMENTAL POSTCONDITIONING TO AMELIORATE GLOBAL ISCHEMIA-REPERFUSION INJURY

Ideally, most patients would receive high-quality CPR by a bystander immediately after cardiac arrest. Unfortunately, in most communities only a minority of patients receives CPR before the arrival of first responders. This translates into an absent circulation with whole-body ischemia for an average of 8 to 10 minutes. In a recent multicenter randomized CPR trial, no patient was discharged from the hospital with favorable neurologic outcome when the time from the emergency call to CPR initiation exceeded 10 minutes [48]. Paradoxically, reperfusion, that is, the reintroduction of blood flow to vital organs after prolonged untreated ischemia, is thought to add significantly to overall ischemia-reperfusion (IR) injury, with mitochondrial dysfunction and the production of reactive oxygen species contributing to cell death [65]. Faced with this challenge, recent research has focused on ways to improve outcomes by mitigating IR injury after cardiac arrest beyond what was previously thought possible. Although preconditioning requires prior knowledge of an ischemic event, postconditioning strategies have the distinct advantage that they can be used on reperfusion

[66]. In the context of cardiac arrest, it is important to recognize that the actual reperfusion starts with the onset of CPR, not later with ROSC. Postconditioning is achieved by targeting known intracellular signaling pathways early that lead to cellular dysfunction and eventually cell death. Although its exact signaling mechanisms are still unclear, postconditioning likely includes activation of the reperfusion injury salvage kinase and survival activating factor enhancement pathways, and result in reduced apoptosis and improved cardiac and neurologic function and eventually increased survival in different animal models of cardiac arrest [67]. For example, the introduction of several short well-defined pauses limited to only the very beginning of CPR [68], or the very early administration of pharmacologic agents [69–71] to trigger postconditioning pathways show highly promising results in the experimental setting. Before their translation into clinical practice, however, conclusive clinical trials are needed to understand and validate their benefit in humans.

TARGETED TEMPERATURE MANAGEMENT

The benefits of therapeutic hypothermia to decrease metabolism and thus organ injury following cardiac arrest have been described for several decades [72,73] before the first human trial by Bernard and colleagues [74] in 1997. In 2002, two landmark publications by Bernard and colleagues [75] and the Hypothermia after Cardiac Arrest Study Group [76] built the basis for the current standard of care [77]; therapeutic hypothermia (32°C–34°C) for 18 [75] to 24 hours [76] increased the rate of a favorable neurologic outcome and reduced mortality significantly after cardiac arrest compared with no hypothermia. In 2013, however, Nielsen and colleagues [78] reported in a large multicenter trial that controlled hypothermia did not have any benefit compared with controlled normothermia with regards to mortality or neurologic outcome in unconscious survivors of OHCA. Despite considerable attention and criticism of this study, a possible consensus could be that it may not be the mild hypothermia, but rather the active avoidance of hyperthermia that is critically important for a more favorable outcome post cardiac arrest [78,79]. This notion is now reflected in the 2015 post cardiac arrest care guidelines that now use the new term “targeted temperature management” to refer to induced hypothermia and to the active control of temperature at any target, and recommend that comatose adult patients with ROSC after cardiac arrest are kept at a constant temperature between 32°C and 36°C for at least 24 hours after achieving target temperature [80]. More well-planned studies are necessary to shed more light on this complex topic; underpowered studies [81] with an only presumably negative outcome despite clear evidence to the contrary are of limited usefulness in this context.

SUMMARY

Delivering immediate and high-quality CPR (chest compressions at 100–120 per minute and at least 2 inch deep, full chest recoil, no interruptions, early defibrillation if in a shockable rhythm, no hyperventilation) after cardiac arrest remains the focus of substantial system-based, educational, clinical, and translational research efforts. The high frequency of OHCA and IHCA combined with currently still dismal survival rates lend great significance to any improvement in early restoration of cerebral blood flow and decreasing cerebral IR injury postarrest. Adjunct mechanical devices, such as ACD or automated chest compression devices in combination with an ITD, aim to decrease intrathoracic and intracerebral

pressure, thus improving cerebral blood flow and survival; when using an ITD, high-quality CPR is absolutely necessary to benefit the patient. Perioperative cardiac arrest is rare and a special situation because it is typically witnessed and can be treated immediately and specifically according to a short list of likely differential diagnoses. Measuring ETCO_2 can add to improve resuscitative efforts, identify ROSC, and contribute to prognostication after prolonged CPR. Postarrest targeted temperature management and, at least in the experimental setting, postconditioning strategies aim to ameliorate cerebral IR injury. Any progress in strengthening one or several links in the chain of survival has the potential to save the lives and to improve the neurologic function of tens of thousands of cardiac arrest patients worldwide each year.

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Key points

- Cardiac arrest continues to be a substantial health care problem worldwide because of its combination of high frequency and low rate of neurologically favorable survival.
- Immediate and high-quality cardiopulmonary resuscitation remains the focus of the most current 2015 ACLS guidelines and of substantial system-based, educational, clinical, and translational research efforts.
- Mechanical adjunct devices, such as active compression/decompression or automated chest compression devices, in combination with an impedance threshold device aim to improve cerebral blood flow and survival.
- High-quality CPR is absolutely necessary to benefit the patient when using an impedance threshold device.
- Perioperative cardiac arrest is rare and its management is largely etiology-driven.
- Endtidal carbon dioxide measurement helps improve resuscitative efforts, recognize return of spontaneous circulation, and can add to prognostication.
- Postarrest targeted temperature management and postconditioning strategies aim to ameliorate cerebral ischemia-reperfusion injury.

Box 1: Common causes of perioperative cardiac arrest**Cardiovascular**

High parasympathetic tone: vagal reflex, electroconvulsive therapy

Hypovolemia/hemorrhage

Embolism: thrombi, gas, cement

Increased intra-abdominal pressure

Anaphylaxis/transfusion reaction

Pulmonary hypertension

Acute coronary syndrome

Pacemaker failure

Electrolyte imbalance: hyperkalemia, hypocalcemia

Respiratory

Hypoxemia/hypercarbia

Increased intrathoracic pressure: bronchospasm, auto positive end-expiratory pressure, tension pneumothorax

Anesthesia

Anesthetic overdose

High neuroaxial block with sympathectomy

Local anesthetic toxicity

Malignant hyperthermia

Drug errors

Adapted from Moitra VK, Gabrielli A, Maccioli GA, et al. Anesthesia advanced circulatory life support. Can J Anaesth 2012;59(6):586–603.

Box 2: Important features of current (2015) CPR guidelines

High-quality chest compressions

Compression rate at 100 to 120^a per minute

Compression depth at least 2 inches

Complete recoil

Uninterrupted chest compressions

Start with compressions

Only one shock at a time followed by immediate CPR

Avoid excessive ventilation

30:2 compression/ventilation ratio for single rescuers of adults, children, and infants

Advanced airway: continuous chest compressions, ventilation every 6 to 8 seconds

Removal of vasopressin from algorithm^a

^aDifferent from 2010 guidelines.

Adapted from Link MS, Berkow LC, Kudenchuk PJ, et al. Part 7: adult advanced cardiovascular life support: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation* 2015;132(18 Suppl 2):S444–64

Box 3: Advantages of mechanical CPR devices

Mechanical CPR devices enable reliable compressions with

- Constant rate

- Constant depth

- Constant location

Mechanical CPR devices avoid

- Inconsistencies of manual compressions

- Provider fatigue

- Injury

Mechanical CPR devices free up personnel for other important tasks

Fig. 1. 2015 CPR guidelines according to the American Heart Association. ET, endotracheal tube; IO, intraosseous; IV, intravenous; PEA, pulseless electrical activity; pVT, pulseless ventricular tachycardia. (From Link MS, Berkow LC, Kudenchuk PJ, et al. Part 7: adult advanced cardio-vascular life support: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation* 2015;132(18 Suppl 2):S452; with permission.)

Fig. 2. Examples of automated chest compression devices. LUCAS-2 (*left*); AutoPulse (*right*). (*Courtesy of* [*Left*] Lund University Cardiopulmonary Assist System; PhysioControl, Redmond, WA, with permission; and [*Right*] ZOLL Medical Corporation, Chelmsford, MA; with permission.)

Fig. 3.
The impedance threshold device is placed between a tight sitting face mask (*left*) or endotracheal tube (*right*) and the ventilation bag. (*Courtesy of ZOLL Medical Corporation, Chelmsford, MA; with permission.*)

Table 1

Predictors of survival after out-of-hospital cardiac arrest

Predictors of survival	Survival probability, %
Witnessed by a bystander	6.4–13.5
Bystander CPR	3.9–16.1
Witnessed by EMS personnel	4.9–18.2
Initial rhythm shockable	14.8–23.0
Return of spontaneous circulation in the field	15.5–33.6

Abbreviation: EMS, emergency medical system.

Adapted from Sasson C, Rogers MA, Dahl J, et al. Predictors of survival from out-of-hospital cardiac arrest: a systematic review and meta-analysis. *Circ Cardiovasc Qual Outcomes* 2010;3(1):63–81.

Table 2

Predictors of survival after in-hospital cardiac arrest

Predictors of survival	Survival probability, %
Initial rhythm shockable	85
Arrest in the intensive care unit	56
Epinephrine administered within 2 min of arrest	54
Witnessed arrest	52

Adapted from Fennelly NK, McPhillips C, Gilligan P. Arrest in hospital: a study of in hospital cardiac arrest outcomes. *Ir Med J* 2014;107(4):105–7.