Cardiac Arrest Management

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Introduction

It is currently estimated that over 300,000 out-of-hospital cardiac (OHCA) arrests occur in the United States. Over half of OHCA cases are managed by EMS systems [1]. The national average for survival from an OHCA is approximately 12 % however; there is considerable variation by region and EMS system [2, 3]. Factors associated with an improved survival from OHCA include crew witnessed arrest and bystander CPR [4]. Survival from witnessed VF arrest decreases by 8 % for every minute delay in CPR and defibrillation [5]. Overall outcomes correlate with early implementation of chest compression. There is a strong suggestion that bystander CPR whether with "chest compression only" or standard CPR is associated with better mortality and neurologic outcomes [6, 7].

Early effective chest compressions and attention to basic life support components are part of high quality CPR. Various organizations have revisited each of the components of cardiac arrest resuscitation over the last couple of years and thus the elements of high-quality CPR are "ever-evolving" [8]. Rapid activation of the "Chain of Survival" and meticulous attention to early defibrillation and chest compressions may lead to greater overall trends in survival.

Case Presentation

A 68 year-old male was playing cards at a casino when suddenly he clutched his chest and became unresponsive. Casino security arrived within less than a minute and applied an automated external defibrillator (AED). AED displayed an audio prompt that "no shock was advised". Bystander cardiopulmonary resuscitation (CPR) was begun within another 10 s. Paramedics arrived and provided two-rescuer CPR with a compression rate of 110 compressions/minute. In the ambulance rescuers used a mechanical compression device to administer continuous chest compressions at a rate of 110 and depth of 2.5 inches with manual ventilation using a bag mask valve. This support was continued until their arrival at the hospital in approximately 9 min. Patient was not intubated in the field. During CPR there was no evidence of an organized cardiac rhythm. Patient had received a total of 3 doses of epinephrine totaling 3 mg via humeral intra-osseous line. Upon arrival in the emergency department endotracheal intubation was performed without incident and capnography displayed a good waveform with an $ETCO_2$ of 15 mm Hg. On arrival emergency physicians administered another dose of epinephrine while continuing CPR. At this point the team leader

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paused and solicited ideas from the team about possible etiologies for persistent pulseless electrical activity (PEA).

Question What are methods to assess the quality of chest compressions during CPR?

Answer Capnography, arterial blood pressure and coronary perfusion pressure

Components of high quality CPR include minimizing interruptions of chest compressions with a chest compression fraction of >60 %, correct chest compression rate and depth. Recommended chest compression rates are greater than 100 and a depth of 50 mm with allowance for chest recoil between compression and minimizing ventilations to no more than 10–12 breaths/minute [9–12]. The emphasis of chest compressions over positive pressure ventilation has been supported by studies, which have shown a mortality benefit of compression only CPR in witnessed arrest compared with traditional CPR with compressions and ventilation [13]. How well chest compressions are meeting the goal of providing circulatory flow to the brain and vital systems is often difficult to ascertain.

Of the readily available parameters capnography and arterial blood pressure monitoring are the most easily applied measurements to provide feedback of the quality of chest compressions. Close attention to the physiologic response to chest compressions is desirable, as studies indicate that even healthcare professionals have poor recall and variable quality when performing chest compressions [14].

Capnography has long-been seen as a potential surrogate for blood flow through heart and the pulmonary circulation [15]. As the technology has improved so has the portability. Capnography can now be measured by sidestream technology in non-intubated patients and or mainstream capnography in intubated patients. During cardiopulmonary resuscitation the goal of high quality chest compressions is to achieve an end-tidal CO_2 of 20 mm Hg or higher and to maintain an end-tidal CO_2 greater than 10 mm Hg at all times with compressions. A rapid rise in end-tidal CO_2 with chest compression to close to 35 mm Hg may signal return of spontaneous circulation (ROSC) [15–17].

Achieving a higher blood pressure during CPR makes intuitive sense, as thoracic compression and thus, cardiac output will be the driving force behind improving cerebral and systemic perfusion. However, the hemodynamics of cardiac arrest is complex and patient-specific factors may be responsible for the variable responses to chest compressions, vasopressors and ventilation. One of the main determinants of successful resuscitation is the coronary perfusion pressure (CPP), which is the difference between the right atrial pressure (or CVP) and aortic pressure during diastole (relaxation phase of chest compression). In the arrested patient there is a delay until there is a complete cessation of flow through the cardiac chambers and by 1 min there is no flow to the coronary arteries. In a human study a CPP < 15 mm Hg was associated with not achieving ROSC [18]. In animal studies it has been shown that higher levels of CPP are required to provide cerebral blood flow when CPR is delayed [19]. Moreover, in studies where ROSC was achieved in humans, it was closely tied to CPP and aortic diastolic pressure [20]. CPR guided by blood pressure has also shown improved outcomes [21]. In the observational human study evaluating coronary perfusion pressure as the correlate to ROSC a mean maximal aortic relaxation pressure (aka diastole) was 35.2 ± 11.5, thus the diastolic pressure target should be approximately 40 mm Hg [18]. In settings where a patient is instrumented with an arterial and a central venous line, aortic diastolic pressure and right atrial pressure can be substituted by arterial diastolic pressure and central venous pressure. The difference between arterial diastolic pressure and central venous pressure may provide a rough estimate of CPP [22]. When the ability to monitor CPP is unavailable a strategy to assess the efficacy of chest compressions may depend upon capnography and diastolic pressure.

Lastly, emerging technology, which provides instantaneous feedback about the quality of chest compressions is now available. This CPR-sensing feedback (FB) system often utilizes accelerome-

ters to detect rate and depth of compressions while delivering audio cues to the rescuer. Currently available CPR-FB systems include the Phillips Q-CPR ®, Zoll Real CPR Help ® and Physio-Control compression metronome and Code Stat ® [23]. It is not known at this time whether utilizing these CPR-FB systems improves outcomes.

Principles of Management

Standard Approach to Resuscitation

Introduction

Achieving optimal outcomes from cardiac arrest requires collaboration between several disciplines including pre-hospital providers, emergency physicians, cardiologists, cardiac interventionalist as well as several other medical professionals and specialists. All providers in this paradigm should understand each other's role as well as what measures can be expected to be offered to a victim of sudden cardiac arrest.

Recognition of Sudden Cardiac Arrest

The ability of laypersons as well as health professionals to detect a pulse has been reported to be extremely poor [14, 24]. Additionally, agonal breaths may be seen for several minutes after cardiac arrest confounding the confirmation that a patient has arrested. Despite these limitations it is best to activate emergency response system as soon as a patient is unresponsive with a faint or absent pulse.

Chest Compressions

After a pulse check of no more than 10 s chest compressions should be initiated at once. Health care providers should re-double their efforts to improve their knowledge and maintain technical skills relevant to chest compressions. Evidence for maintaining these skills may be gleaned from a multi-center study whereby healthcare providers often performed suboptimal chest compression rates. Specifically, the mean chest compression rate was below the recommended rate and lowest for patients without ROSC (79 ± 18) compared to patients with ROSC (90 ± 17) [3].

Specific goals of high quality CPR include achieving a compression rate of at least 100–120 compression/minute and a compression depth of at least 50 mm (2 inches) with an upper limit of 60 mm (2.4 inches) [9]. Additionally, high quality chest compressions should include a chest compression fraction >60 %, meaning when CPR is performed chest compressions should occupy at least 60 % of the resuscitation [9]. Patient positioning, vascular or intraosseous access, medication administration, airway establishment, rhythm analysis and defibrillation should occupy the remaining fraction of time. Maintaining a chest compression rate of 100–120 compressions/minute can lead to rescuer fatigue. Switching compressors every 2 min may minimize rescuer fatigue but lead to frequent interruptions and may negatively impact chest compression fraction. One suggestion to decrease this "hands-off" time is to have rescuers switch from opposite sides of the victim [25]. Between compressions there should be time allowed for full chest recoil in order for heart to refill with blood and maximizing CPP.

Adjuncts to CPR: Oxygen and Ventilation

During the initial rounds of chest compressions rescuers should focus on the quality of the compressions and use passive oxygenation with the highest concentration of oxygen available at the time. The delivery of this oxygen is dependent upon the systemic perfusion that may be established by chest compressions.

Attempts to establish a definitive airway should be postponed unless there is difficulty ventilating a patient with a bag valve mask. Furthermore, hyperventilation should be avoided as this has been tied to reducing cardiac output [26, 27]. When two providers are resuscitating a patient ventilations are delivered in a 30:2 compression-to-ventilation ratio until a definitive airway has been established [28]. Using a 1 L bag mask device a second provider should provide approximately 600 cc of tidal volume over 1 s. This should be performed a total of two times after every 30 compressions. With an advanced

airway in place rescuers may provide a breath every 6 s while chest compressions are performed continuously.

Defibrillation

Defibrillation is indicated for ventricular fibrillation or pulseless ventricular tachycardia. Although traditionally monophasic defibrillators have been used to administer a counter shock, biphasic defibrillators are preferred due to the greater first shock success. Newer waveforms have been studied which provide patient-specific impedance current delivery using biphasic truncated exponential, rectilinear biphasic or pulsed biphasic wave. At this time there is no specific recommendation regarding which waveform is superior. Current recommendations are to administer a single counter shock at an optimal energy level (between 120 and 360 J for biphasic defibrillators) with minimal interruptions in CPR before and after the shock [28]. In situations requiring repeated defibrillations use manufacturers' guidelines or consider escalating energy. For refractory VF and pulseless VT, administration of epinephrine and an anti-dysrhythmic agent should be instituted.

Search for Precipitating Cause of Cardiac Arrest

Ventricular Dysrhythmias

Survival to discharge for patients with an initial rhythm of VT or VF is between 15 and 23 % for out-of-hospital cardiac arrest and up to 37 % for patients with an in-hospital cardiac arrest [29, 30]. Resuscitation team leaders must simultaneously look for reversible etiologies while administering time-sensitive interventions. Ventricular fibrillation is usually found in patients with abnormal myocardial perfusion from a prior infarct or ongoing ischemia. Similarly ventricular tachycardia usually results from foci below the AV node which progresses into a wide and regular tachycardia. When confronted with these malignant ventricular dysrhythmias diagnostic considerations include medication toxicity, pre-existing channelopathy (Brugada syndrome) or an electrolyte abnormality. If medication toxicity and electrolyte abnormalities are ruled out persistence of these dysrhythmias should prompt search for myocardial ischemia.

Different forms of ventricular tachycardia exist including: monomorphic VT, polymorphic VT, torsade de pointes, right ventricular outflow tachycardia (idiopathic and arrhythmogenic right ventricular dysplasia), fascicular tachycardia, bidirectional VT and ventricular flutter. Monomorphic VT accounts for the majority of VT encountered. Most cases of monomorphic VT are associated with myocardial ischemia. Torsade de pointe is a specific form of polymorphic VT where there is progressive widening of the QT interval. Although most forms of VT are associated with myocardial ischemia there are forms of idiopathic VT. Of the idiopathic forms of VT, most cases are due to abnormalities in the outflow tract of the right ventricle. A small number of these have an anatomically identified focus termed "arrhythmogenic right ventricular dysplasia". Ventricular flutter is an extreme form of VT that has a sinusoidal appearance and may degrade into ventricular fibrillation. Ventricular flutter usually has a rate >200 beats/min. Thus when confronted with a patient with refractory ventricular dysrhythmias providers should strongly consider consulting a cardiology specialist to evaluate patient for the possibility of a diagnostic and percutaneous intervention.

Pulseless Electrical Activity (PEA)

Patients with PEA have a survival to discharge of 2.77 % for patients with out-of-hospital cardiac arrest as compared to patient in an in-hospital cardiac arrest of only 12 % [30, 31]. PEA is defined as the absence of a pulse when electrical cardiac activity is present. This is further classified as "true" PEA, which is when there is no pulse, the presence of an electrical signal but no evidence of cardiac activity usually detected by echocardiography. "Pseudo-PEA" is defined as the absence of a pulse, presence of an electrical signal and cardiac activity observed by echocardiography.

It is important to make these distinctions, as there is pathophysiologic and prognostic significance. True PEA is when electromechanical uncou-

pling of cardiac cells which propagate an electrical signal but the myocytes are unable to coordinate ventricular contraction. This situation is usually seen in severe hypoxia, acidosis or necrosis.

In pseudo-PEA, there is an electrical signal and weak cardiac contractions due to conditions such as hypovolemia, massive pulmonary embolism or other mechanical impediments to flow. In these situations the predominant rhythm is a tachydysrhythmia.

A mnemonic, which has been modified over the years to remind providers of the common precipitants of PEA, is "4Hs-4Ts". This mnemonic represents – hypoxia, hypovolemia, hypo/hyperkalemia and hypothermia as well as thrombosis (pulmonary emboli), tamponade (cardiac), toxins and tension pneumothorax [32].

Point of care ultrasound whenever possible should be used to assist clinicians to investigate many of the above-mentioned etiologies. For instance, a subcostal view on ultrasound may reveal a large pericardial effusion with diastolic collapse of right ventricle representing cardiac tamponade (Video 1.1). A parasternal short axis view may demonstrate bowing of the intra-ventricular septum, the so called "D-sign" appearance of the left ventricle being compressed by a volume-overloaded right ventricle contracting against a massive pulmonary embolism (Video 1.2).

Littman et al. proposes a diagnostic guide to evaluate causes of PEA which includes evaluating the width of the QRS complexes on EKG as well as combining sonographic findings to suggest whether a mechanical, ischemic or metabolic cause are to blame [33]. There is no randomized study to support ultrasound-guided resuscitations over resuscitations without ultrasound but a recent study has suggested a trend that when modifications in traditional approaches employ ultrasound there may be a higher rate of ROSC to hospital admission [34].

Quality Assurance

Every cardiac arrest should have some method to monitor the quality of the resuscitation. The ability of rescuers to retain critical resuscitation skills wanes after 6 months thus implementing simulated resuscitations may be useful in retaining skills [35]. Short debriefing sessions after performing a cardiac resuscitation have been shown to improve team performance and outcomes [36].

Evidence Contour

Are Outcomes with Mechanical Compressions Superior to Manual Compressions During Active CPR?

Based upon currently available data mechanical compressions do not appear to be superior to manual compressions in terms of outcomes. Manual compressions are the most readily applicable and commonly taught method of providing chest compressions. Despite this many prehospital and hospital systems have chosen to use mechanical compression devices to administer chest compression for various logistical reasons.

Over the last 40 years various technologies have emerged to provide high quality and consistency of chest compressions. These technologies are based upon one of two predominant theories of how chest compressions promote forward flow of blood into the thoracic aorta and systemic circulation. The so-called "cardiac pump" theory expounds that external chest compressions places pressure simultaneously on the right and left ventricle. During the active compression phase a pressure gradient pushes blood out of ventricles, while closing the atrio-ventricular valves and then to the pulmonary artery and the aorta. During the decompression phase blood re-enters the right and left atrium and feeds coronary arteries [37].

The "thoracic pump" theory relies upon the compliance of the whole thorax as the main pressure determinant of flow and not on compression of the heart. During compressions in the "thoracic pump" theory intra-thoracic pressure is increased driving blood into the thoracic, extra-thoracic aorta and other large arteries preferentially. This compression however does not seem to affect the venous system as much due to valves and the vast network of venous plexuses. During

Fig. 1.1 Thumper ™ piston driven chest compression device (Courtesy of Michigan Instruments, Inc.)

Fig. 1.2 LUCAS™ Chest compression device (Used with permission of Physio-Control, Inc.)

the decompression phase intra-thoracic pressure falls below the extra-thoracic pressure and blood flows to the lungs.

Mechanical compression devices became available for research and clinical applications during the 1970s with the Thumper ® created by Michigan Instrument which utilized a hydraulically powered piston to provide chest compressions similar to the "cardiac pump theory" (Fig. 1.1). Newer devices emerged including the Lund University Cardiac Arrest System (aka. LUCAS 1 and 2 ®) by Physio Control and the Auto Pulse ® by Zoll employ different mechanisms to enhance chest compressions namely through an active compression and decompression mode. The LUCAS ® device is predominantly a piston-driven device with a suction area which makes contact with the chest (Fig. 1.2). The Auto Pulse ® utilizing a load-distributing band which wraps around the torso and squeezes to increase intra-thoracic pressure (Fig. 1.3).

Fig. 1.3 Auto Pulse ™ Load distributing chest compression device (Courtesy of ZOLL Medical Corporation)

Despite various studies utilizing transthoracic and trans esophageal dopplers to investigate the hemodynamics during chest compressions there is no consistent data to support either the "cardiac pump or "thoracic pump" as the main mechanism of flow during CPR [19, 38]. It is possible that there are elements of both pump models active during CPR.

Advocates of mechanical compressions support its consistency of depth and compression rate. Mechanical compressions can maximize "hands on" time compared to manual compressions due to the fact that there is no need to switch

rescuers or halt compressions when performing defibrillation. Despite these theoretical advantages a study completed in 2014 did not show a mortality benefit of mechanical CPR over manual compressions [39]. This study, which used the LUCAS device showed no difference in survival or neurologic outcome. The added cost of these devices and their upkeep may prohibit widespread use however providers point to the advantage of reducing rescuer fatigue and diminishing risk to rescuers during transport while CPR is in progress [40, 41].

What Physiologic Parameters Can Guide the Administration of Vasoactive Medications and Provide Feedback Regarding Quality of CPR?

Hemodynamic Directed Resuscitation

Hemodynamic directed resuscitation (or patient-centric cardiopulmonary resuscitation) is a concept whereby the decision to administer a pharmacologic agents such as epinephrine is guided by hemodynamic variables. This concept may have developed due to various studies indicating that epinephrine may have several deleterious effects on the heart [42]. A strategy whereby resuscitation is guided by hemodynamic parameters rather than protocolized and repetitive administration of epinephrine may potentially minimize inadvertent epinephrine toxicity [42].

Ever since the seminal studies by Ewy and Paradis which tied successful resuscitation to coronary perfusion pressure (CPP) there has been a resurgence of interest in evaluating CPP or a "surrogate of CPP" during CPR [18, 43]. In an animal study which compared CPR guided by CPP vs. chest compressions at 33 mm depth plus standard dose epinephrine vs. chest compressions at 51 mm depth plus standard dose epinephrine the largest improvement in cerebral perfusion pressure was found in the group that was guided by CPP [44]. In another similar study, CPR guided by systolic blood pressure was associated with the highest 24-h survival compared to conventional guideline care [21]. Despite controversy surrounding the use of central venous oxygen saturation ($ScvO_2$) in the management of sepsis its application may be used as an estimate of tissue perfusion. $ScvO_2$ represents the residual oxygen content returning to the right side of the heart after systemic perfusion. Studies have shown that persistently low $ScvO_2$ correlates with decrease cardiac output. If available, chest compressions may be titrated to a $ScvO_2$ concentration greater than 30 % [45, 46].

Whether hemodynamic directed resuscitation leads to better outcomes in humans remains unknown but if efforts to improve the rate of bystander CPR and high quality chest compressions are successful there may be less dependence upon the pharmacologic treatment to achieve ROSC.

References

1. Daya MR, et al. Out of Hospital Cardiac Arrest survival improving over time: results from the Resuscitation Outcomes Consortium (ROC). Resuscitation. 2015;91:108–15.
2. Prevention, C.f.D.C.a. Cardiac Arrest Registry to Enhance Survival (CARES) National Summary Report: Non-Traumatic National Survival Report. 2014. https://mycares.net/sitepages/reports2014.jsp. Accessed 4 Jan 2016.
3. Abella BS, Sandbo N, Vassilatos P. Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest. Circulation. 2005;111:428–34.
4. Hollenberg J, Herlitz J, Lindqvist J. Improved survival after out of hospital cardiac arrest is associated with an increase in proportion of emergency crew-witnessed cases and bystander cardiopulmonary resuscitation. Circulation. 2008;118:389–96.
5. Sutton R, Nadkarni V, Abella BS. "Putting it all together" to improve resuscitation quality. Emerg Med Clin N Am. 2012;30(1):105–22.
6. Bernard SA, Buist M. Induced hypothermia in critical care medicine: a review. Crit Care Med. 2003;31(7):2041–51.
7. Bohm K, et al. In patients with out-of-hospital cardiac arrest, does the provision of dispatch cardiopulmonary resuscitation instructions as opposed to no instructions improve outcomes: a systematic review of the literature. Resuscitation. 2011;82:1490–5.
8. Hazinski M, et al. Part 1: Executive Summary: 2015: International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Circulation. 2015;132:S2–39.

9. Neumar N, et al. Executive Summary Part 1: 2015 American Heart Association Guidelines Update for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation. 2015;132 Suppl 2:S315–67.
10. Wik L, Kramer-Johansen J, Myklebust H. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. JAMA. 2005;293(3):299–304.
11. Idris A, Guffey D, Pepe P. Chest compression rates and survival following out-of-hospital cardiac arrest. Crit Care Med. 2015;43(4):840–8.
12. Meaney P, Bobrow B, Mancini M. CPR quality: improving cardiac resuscitation outcomes both inside and outside the hospital. Circulation. 2013;128(4):417–35.
13. International Liaison Committee on Resuscitation. 2005 International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Resuscitation. 2005;67:181–341.
14. Ochoa F, Ramalle-Gomara E, Carpintero J. Competence of health professionals to check carotid pulse. Resuscitation. 1998;37:173–5.
15. Sanders A, Atlas M, Ewy G. Expired PCO2 as an index of coronary perfusion pressure. Am J Emerg Med. 1985;1985(3):147–9.
16. Rieke H, et al. Virtual arterial blood pressure feedback improves chest compression quality during simulated resuscitation. Resuscitation. 2013;84:1585–90.
17. Vaillancourt C, et al. In out-of-hospital cardiac arrest patients, does the description of any specific symptoms to the emergency medical dispatcher improve the accuracy of the diagnosis of cardiac arrest: a systematic review of the literature. Resuscitation. 2011;82:1483–9.
18. Paradis N, et al. Coronary perfusion pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. JAMA. 1990;263:1106–13.
19. Andreka P, Frenneaux MP. Haemodynamics of cardiac arrest and resuscitation. Curr Opin Crit Care. 2006;12(3):198–203.
20. Sanders A, Ewy G. Prognostic and therapeutic importance of the aortic diastolic pressure in resuscitation from cardiac arrest. Crit Care Med. 1984;12(10): 871–3.
21. Sutton R, Friess S, Naim M. Patient-centric blood pressure-targeted cardiopulmonary resuscitation improves survival from cardiac arrest. Am J Resp Crit Care Med. 2014;190(11):1255–62.
22. Sutton R, Friess S, Maltese M. Hemodynamic-directed cardiopulmonary resuscitation during in-hospital cardiac arrest. Resuscitation. 2014;85: 983–6.
23. Vadeboncoeur T, Bobrow BJ, Cone DC, Chikani V, Abella BS. Instant replay: perform CPR with immediate feedback. JEMS. 2011;36(3):50–63.
24. Eberle B, Dick W, Schneider T. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. Resuscitation. 1996;33:107–16.
25. Kim Y, Lee J, Lee D. Differences in hands-off time according to the position of a second rescuer when switching compression in pre-hospital cardiopulmonary resuscitation provided by two bystanders: a randomized, controlled, parallel study. J Korean Med Sci. 2015;30:1347–53.
26. Aufderheide TP. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. Circulation. 2004;109:1960.
27. Aufderheide TP, Lurie K. Death by hyperventilation: a common and life-threatening problem during cardiopulmonary resuscitation. Crit Care Med. 2004;32:S345.
28. Neumar RW, et al. 2010 "American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science". Circulation. 2010;122:S729–67.
29. Sasson C, et al. Predictors of survival from out of hospital cardiac arrest: a systematic review and meta-analysis. Circulation. Cardiovasc Quality Outcomes. 2010;3(1):63–81.
30. Meaney P, Nadkarni V, Kern K. Rhythms and outcomes of adult in-hospital cardiac arrest. Crit Care Med. 2010;38(1):101–8.
31. Thomas A, Newgard C, Fu R. Survival in out-of-hospital cardiac arrests with initial asystole or pulseless electrical activity and subsequent shockable rhythms. Resuscitation. 2013;84(9):1261–6.
32. Kloeck W. A practical approach to the aetiology of pulseless electrical activity. A simple 10-step training mnemonic. Resuscitation. 1995;30:157–9.
33. Littman L, Bustin D, Haley M. A simplified and structured teaching tool for the evaluation and management of pulseless electrical activity. Med Princ Pract. 2014;23:1–6.
34. Prosen G, Krizmaric M, Zavrsnik J. Impact of modified treatment in echocardiographically confirmed pseudo-pulseless electrical activity in out-of-hospital cardiac arrest patients with constant end-tidal carbon dioxide pressure during compression pauses. J Int Med Res. 2010;38:1458–67.
35. Niles D, Sutton R, Donoghue A. "Rolling Refreshers": a novel approach to maintain CPR psychomotor skill competence. Resuscitation. 2009;2009(80):909–12.
36. Edelson D, Litzinger B, Arora V. Improving in-hospital cardiac arrest process and outcomes with performance debriefing. Arch Intern Med. 2008; 168(10):1063–9.
37. Rudikoff M, Maughan W, Effron M. Mechanisms of blood flow during cardiopulmonary resucitation. Circulation. 1980;61(2):345–52.
38. Georgiou M, Papathanassoglou E, Xanthos T. Systematic review of the mechanisms driving effective blood flow during adult CPR. Resuscitation. 2014;85:1586–93.
39. Rubertsson S, et al. Mechanical chest compressions and simultaneous defibrillation vs conventional cardiopulmonary resuscitation in out-of-hospital cardiac arrest: the LINC randomized trial. JAMA. 2014;311(1):53–61.
40. Chang WT, Ma MH, Chien KL. Post resuscitation myocardial dysfunction: correlated factors and prognostic implications following prolonged ventricular

fibrillation and cardiopulmonary resuscitation. Intensive Care Med. 2007;33:88–95.
41. Brooks S, et al. Mechanical versus manual chest compressions for cardiac arrest. Cochrane Database Syst Rev. 2014;2:1–46.
42. Dumas F, Bougouin W, Geri G. Is epinephrine during cardiac arrest associated with worse outcomes in resuscitated patients. J Am Coll Cardiol. 2014;64(22):2360–7.
43. Go AS, et al. Heart disease and stroke statistics – 2013 update: a report from the American Heart Association. Circulation. 2013;127:e6–245.
44. Friess S, Sutton R, French B. Hemodynamic directed CPR improves cerebral perfusion pressure and brain tissue oxygenation. Resuscitation. 2014;85(9): 1298–303.
45. Rivers E, et al. The clinical implications of continuous central venous oxygen saturation during human CPR. Ann Emerg Med. 1992;21:1094–101.
46. Snyder A, Salloum L, Barone J. Predicting short-term outcome of cardiopulmonary resuscitation using central venous oxygen tension measurements. Crit Care Med. 1991;19:111–3.