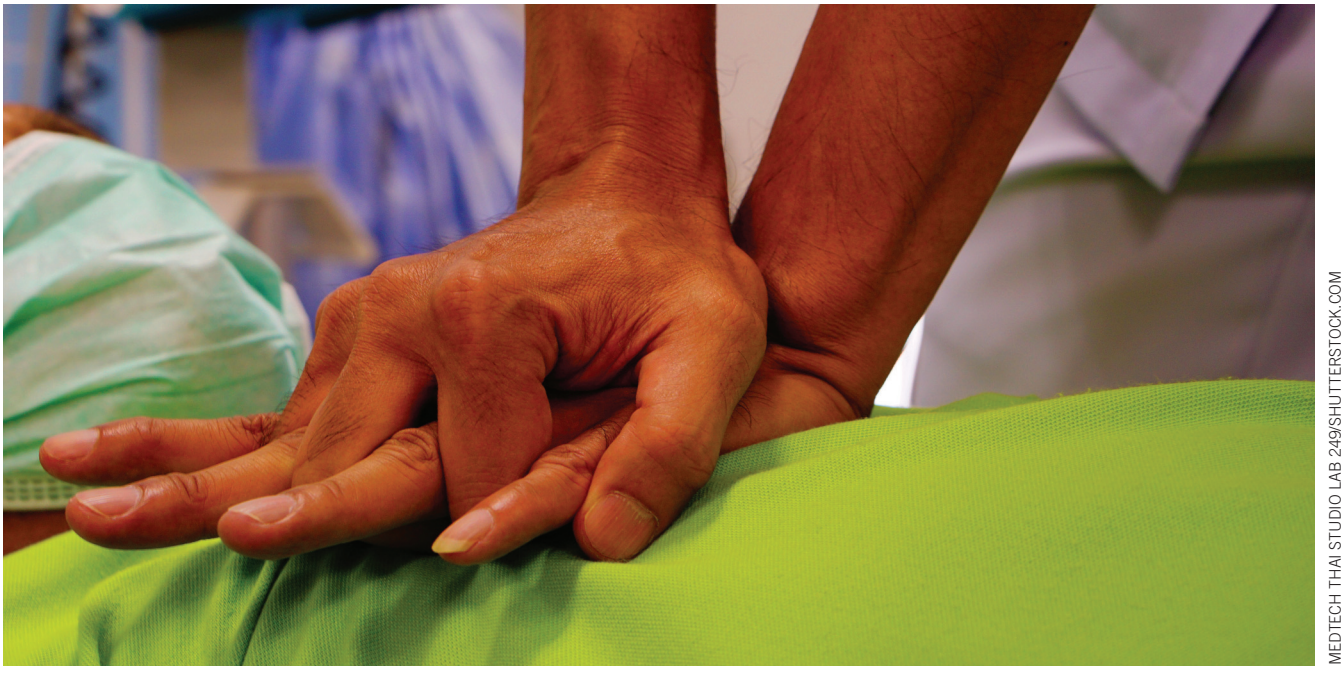

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Optimizing the management of inpatient cardiac arrest

Michael Carter, MPAS, PA-C

ABSTRACT

Cardiac arrest is a common event in the inpatient setting, and although attempts have been made to simplify the approach to its management via systemwide training in CPR and Advanced Cardiac Life Support, significant challenges remain in implementing these guidelines in the management of cardiac arrest.

Keywords: cardiac arrest, inpatient, Advanced Cardiac Life Support, management, resuscitation, CPR

Learning objectives

- Understand the importance of early recognition and early intervention in cardiac arrest.
- Recognize the underlying physiology behind high quality CPR and how best to implement it.
- Explain the importance of early defibrillation.
- Demonstrate an understanding of the use of pharmacotherapies in resuscitation.
- Be able to recognize deficiencies in team performance and offer suggestions for improvement.

About 650,000 cardiac arrests occur every year in the United States, with 290,000 occurring in the hospital setting.[1] Most (77%) patients in cardiac arrest present with a nonshockable rhythm such as asystole or pulseless electrical activity (PEA).[1] This distribution is similar to arrests occurring in the outpatient setting, with the largest difference being outcomes; about 26% of patients with inhospital cardiac arrest survive to hospital discharge, compared with 10% of patients suffering out-of-hospital arrests.[1,2] Among these survivors, 21% of those with inhospital arrests and 9% of those with out-of-hospital arrests have a good functional outcome (**Figure 1**).[1] The American Heart Association has published data demonstrating opportunities for quality

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Key points

- The outcomes for patients with inhospital cardiac arrest are better than for those with out-of-hospital arrests; however, room for improvement remains.
- Early recognition, early, high-quality CPR, and rapid defibrillation are crucial.
- Evidence is mixed about the use of pharmacologic agents in patients with cardiac arrest.
- Targeted temperature management in the postarrest phase can help improve patient neurologic outcomes.

improvement in a variety of categories, specifically early recognition and intervention of cardiac arrest, including delivery of high-quality CPR and advanced support such as defibrillation and medication administration.[2-5]

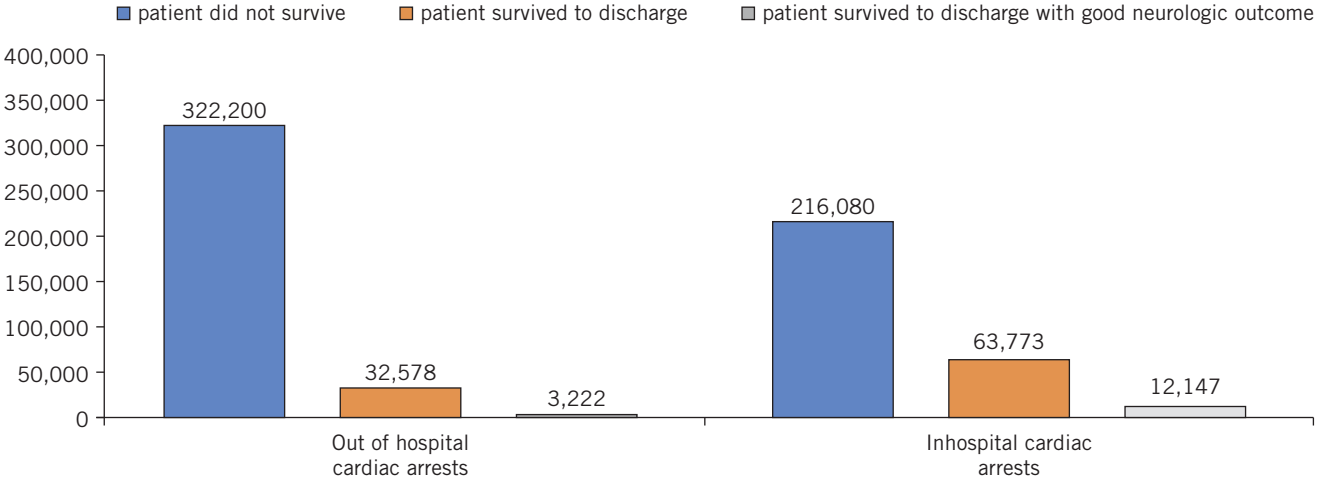
PREARREST

Early recognition, prevention and response Delayed recognition of cardiac arrest, or failure to recognize and intervene early in the management of a patient who is deteriorating, is a leading cause of morbidity and mortality because each minute of delay in receiving treatment reduces patient survival by 10%.[6] This was a driving factor in the Institute of Medicine's push for the development of a rapid response team model with the goal of identifying these patients early in their course.[3-5,7] Rapid response teams commonly are used on general medical units and are an interdisciplinary group activated by the bedside nurse or other caregiver when certain criteria are met; the goal is to prevent patient decompensation.[7] Activation criteria often differ from facility to facility, with some using standardized systems such as the Early Warning Score, and others using hemodynamic parameters, dysrhythmias, or caregiver gestalt (**Table 1**).[1,7,8] Systematic reviews have demonstrated a reduction in non-ICU cardiac arrests without a change in overall mortality.[7,8] Previous studies have also demonstrated a consistent time-related trend in mortality, with significantly worsened outcomes occurring in the "off-hours" or overnight, likely due to reduced staffing, lack of familiarity with patients, and proven decreases in psychomotor function resulting in worsened performance of CPR.[2,3] One way to help increase patient survivability is through Advanced Cardiac Life Support (ACLS) training. The rate of survival from cardiac arrest was shown to increase by almost four times when the nurse who initially found the patient down was ACLS-certified.[9]

Code Blue teams or medical emergency response teams (MERT) often are composed of specially trained members of the hospital staff who respond to cardiac arrests and other medical emergencies. The composition often varies between members of the ICU, ED, and/or anesthesia and may include nurses, physicians, PAs and NPs, pharmacists, and respiratory care practitioners. The American Heart Association strongly suggests formal education for the code team, training of the leadership, and simulation with mock codes.[10] As resuscitations are high-risk, low-frequency events, clinicians often experience a high level of stress during the event, which can have a detrimental effect on team performance. Items that are commonly identified as areas for improvement include number of people in the room, noise level, identification of roles, and leadership. This stress can be mitigated through simulation and mock code training, as well as with the development of an organized code team structure with assigned roles, responsibilities, and locations in the room during an arrest.[11] Literature has demonstrated that adopting a high-fidelity simulation training program can improve the quality of CPR, code team confidence, and rhythm identification.[12] Participation in mock codes has resulted in increased rates of return of spontaneous circulation (ROSC) as well as patient survival to discharge.[13] Structured debriefings

FIGURE 1. Cardiac arrest outcomes in the United States

Based on AHA heart disease and stroke statistics



postevent have been shown to drastically improve ROSC in some studies.[14] These debriefings often include audiovisual tools to demonstrate chest compression fraction, time to defibrillation, and other areas for improvement.

INTRA-ARREST

Compressions Rapid delivery of chest compressions upon recognition of pulselessness is known to improve patient outcomes.[15] In theory, compression of the sternum results in positive intrathoracic pressure, displacing blood from the ventricles and the great vessels into their respective circulation, while the passive decompression phase allows for drainage of the cerebral circulation and venous return to the right ventricle.[16] During the decompression phase, the heart itself is perfused via the coronary arteries, much as in the standard diastolic portion of the cardiac cycle. If compressions are performed well, at the appropriate rate and depth, they assist in perfusion of the cerebral and coronary circulation. The goal of this perfusion is twofold: delivery of oxygen to the systemic tissues mitigating hypoxic-ischemic injury, and providing substrate for the formation of adenosine triphosphate (ATP), all while buying the patient physiologic time as one attempts to find and reverse the cause of the arrest. Any delay in administering compressions can further worsen ischemic time. One study demonstrated that compressions were not being immediately performed in up to 6% of patients with recognized arrests, reducing their odds of survival, and that compressions were too slow in 23% of patients and/or too shallow in 36% of patients.[12,15]

The major components of high-quality chest compressions are ensuring an appropriate rate, depth, and release. Current evidence suggests that a rate of between 110 and 120 compressions per minute is ideal, with a significant dropoff in patient survival on either side of this range.[17] A rate lower than this does not provide the needed bloodflow for adequate perfusion; a more accelerated rate does not allow the necessary amount of ventricular filling time to ensure adequate stroke volume.[15,17] A variety of smartphone applications can provide a metronome to help clinicians keep tempo during compressions; some monitor-defibrillators provide real-time feedback on compression rate.

Reaching an adequate compression depth can be challenging in the hospital setting because patients are most commonly lying on a mattress when pulselessness is recognized. Delivering compressions without a solid support structure under the patient results in inadequate posterior displacement of the sternum, preventing the generation of intrathoracic pressure. Recent data suggest that a compression depth of 40 mm to 50 mm is needed to ensure the highest rate of patient survivability.[18] This can be achieved by either placing a hard board between the patient and mattress, or moving the patient from the bed to the floor.

An often-overlooked component of chest compressions is the release phase; as noted above it is during this time frame that blood is drained from the cerebral venous circulation as well as the remainder of the body and returned to the heart. Coronary perfusion also occurs during this period. If full release does not occur, or if the clinician remains leaning on the patient's chest during this phase between compressions, intrathoracic pressure remains elevated, which increases the gradient for venous return.[19] This commonly occurs due to muscle fatigue and is best prevented by ensuring that clinicians performing compressions are switched out at each rhythm check or earlier if compression quality is poor.[19]

Maximizing the amount of time spent performing compressions also is paramount to providing the best possible patient outcomes. A chest compression fraction (the total amount of time spent providing continuous compressions throughout the arrest) of more than 80% has been shown to greatly increase the possibility of ROSC.[20] This is best achieved by improving logistics, such as the switching of clinicians providing compressions, and charging the defibrillator before the rhythm check so that patients with shockable rhythms can be rapidly defibrillated before a return to compressions. Avoiding prolonged pulse checks also is important; instead, rely on objective data such as capnography or evaluation of an arterial line tracing.

Automated CPR devices are being used by some code teams because they provide continuous, uninterrupted compressions. These machines consist of either a load-distributing band or a piston-like device that is attached to the patient with a short backboard device posteriorly. The benefits of these machines include reducing the cognitive load of the team leader, as well as reducing the number of staff required to respond to an arrest. The literature is unfortunately lacking for inhospital use, with several small

TABLE 1. Common rapid response team activation criteria

Central nervous system
- Seizures
- Obtundation
- Severe agitation

Cardiovascular
- Sustained heart rate greater than 125 or lower than 45 beats/minute
- Ventricular dysrhythmia
- Systolic BP less than 90 mm Hg or greater than 180 mm Hg
- Chest pain

Pulmonary
- Respiratory rate greater than 30 or less than 10
- Spo_2 less than 90%

Hematologic
- Exsanguination

Renal
- Urine output of less than 50 mL in 4 hours

Other
- Staff or family concern

trials having been performed with a low level of recommendation from major societies. A Cochrane Review of 11 trials including patients who experienced either inhospital or out-of-hospital cardiac arrest found that establishing protocols to include mechanical CPR devices has limited utility other than in situations where providing compressions would be dangerous to rescuers or impossible.[21] The heterogeneity of these trials likely contributes to the results, as one could suspect providing resolution of the underlying cause of the dysrhythmia (for example, coronary intervention) with ongoing resuscitation may prove beneficial in that specific subset.

DEFIBRILLATION

In patients with shockable rhythms such as ventricular tachycardia or ventricular fibrillation, early defibrillation is the key to a successful resuscitation. The goal of early defibrillation is to interrupt the macro or micro-reentry rhythms and let the physiologic pacemaker resume control of the patient's cardiac rhythm. A potential benefit to earlier intervention is the higher levels of intra-myocyte ATP, levels which have been correlated with higher success in the restoration of a perfusing rhythm.[22] Multiple studies have shown that delays in the delivery of electrical therapy result in decreased ROSC and worsened outcomes.[15,16,23] Earlier interventions can be achieved by improving access to monitor-defibrillators as well as the implementation of well-trained cardiac arrest response teams. Improving the time to defibrillation to less than 2 minutes from onset of cardiac arrest may help up to 2,000 additional patients survive to discharge.[23]

AIRWAY MANAGEMENT

Basic airway management during cardiac arrest consists of asynchronous ventilation in a 30:2 ratio of compressions to breaths, most commonly delivered by a bag-valve mask device. Once an advanced airway is in place, be it a supraglottic airway or an endotracheal (ET) tube, deliver breaths at a rate of 10 per minute in a synchronous fashion without pauses.[24] The volume delivered should be the minimal amount to induce chest rise, with a strict adherence to the suggested rate, as overzealous ventilation results in decreased venous return and potentially harmful cerebral vasoconstriction.[25] All efforts should be made to avoid interrupting compressions for insertion of an advanced airway; a large randomized controlled trial demonstrated no difference in outcomes with placement of a supraglottic airway compared with an ET tube, and time on the chest is most important.[26] If the decision is made to pursue advanced airway management, the most experienced clinician (anesthesia, ED, or intensivist) should be the one attempting the intervention to ensure first-pass success and to prevent interruptions in compressions.

VASOPRESSORS

Epinephrine, an endogenous catecholamine with alpha and beta agonistic properties, is the primary vasopressor used for patients in cardiac arrest. This drug initially showed promise in resuscitation of canine models from trials in the mid-20th century, and has remained in the ACLS algorithm in some shape ever since.[21] Recent studies have questioned its role and current dosing strategies, with large prehospital studies demonstrating improved patient survival to hospital discharge compared with placebo, although without an improvement in neurologic outcomes.[27] Although epinephrine remains in the 2019 ACLS update without change, controversy remains about its utility in the field of resuscitation science in its current form.[28]

Two trials evaluated data from the Get With the Guidelines-Resuscitation registry about the timing of epinephrine administration. The first study suggested early administration, defined as within 2 minutes of the initial defibrillation, during shockable rhythms was associated with significantly worse odds of achieving ROSC as well as a decrease in patient functional outcomes.[29] In contrast, the second study demonstrated an improvement in both outcomes with earlier administration in patients with nonshockable rhythms.[30] When the differing causes of these types of arrest are considered, the results make sense from a physiologic standpoint. Classically the H's and T's (**Table 2**) are considered when evaluating the possible causes of nonshockable rhythms; these underlying derangements commonly respond to vasopressors by increasing cardiac automaticity and vascular tone. Also consider profound vasoplegia from metabolic derangements and sepsis—these will present in a similar fashion to the conditions above. Shockable rhythms are more likely to be due to a re-entry mechanism often brought on via ischemia with subsequent early after depolarizations (more commonly known as premature ventricular contractions or PVCs) and heterogeneity of the myocardial electrical background. As these are more likely to be responsive to rapid defibrillation, the potential negative effects of epinephrine, such as the increase in myocardial oxygen demand and increase in automaticity, may outweigh the benefits.

TABLE 2. Common reversible causes of cardiac arrest

Based on the AHA cardiac arrest algorithm

- Hypovolemia
- Hypoxia
- Hypo- and hyperkalemia
- Hydrogen ions (metabolic acidosis)
- Tamponade
- Toxins
- Tension pneumothorax
- Thrombosis (coronary or pulmonary)

ANTIARRHYTHMICS

Two core medications are included in the cardiac arrest algorithms for shockable rhythms: amiodarone and lidocaine.[31] Although amiodarone and lidocaine are quite

similar in use, they achieve their antiarrhythmic properties in markedly different ways.

Amiodarone is a Vaughn-Williams class III antiarrhythmic with a complex pharmacodynamic profile. The drug exhibits components of beta antagonism and calcium channel blockade and inhibits the potassium rectifier current in phase 3 of the depolarization cycle. These combined mechanisms result in a multifactorial solution to the problem of dysrhythmias, enabling amiodarone to be used in atrial and ventricular disorders. By prolonging the repolarization phase of the ventricular myocytes, the drug lengthens the absolute refractory period of the cells, inhibiting re-entry circuits. These same mechanisms, although helpful in treating dysrhythmias, also can cause adverse reactions such as significant hypotension and refractory bradycardia.

Lidocaine, a Vaughn-Williams class Ib sodium channel blocking agent, helps prolong the initial depolarization phase by reducing the slope of phase 0, resulting in a reduction in the propagation of an action potential. Although lidocaine has been one of the oldest antiarrhythmics in the ACLS algorithm, it was removed from the 2010 algorithm due a lack of evidence compared with amiodarone.[32] Lidocaine was returned to the algorithm after a 2016 study demonstrated no statistically significant difference in survival to hospital discharge between amiodarone, lidocaine, and placebo with the exception of witnessed arrest in which amiodarone and lidocaine both showed benefit.[33]

> Significant debate remains about the most appropriate temperature goals.

POSTARREST CARE

Targeted temperature management (TTM) and therapeutic hypothermia A key intervention possible in the inhospital setting is the initiation of TTM or therapeutic hypothermia after cardiac arrest. Studies have demonstrated that reducing body temperature provides a neurologic benefit and reduces cerebral metabolic demand, reperfusion injury, and post-ischemic brain injury.[34] This approach is used in patients who demonstrate no purposeful activity or movement after obtaining ROSC.[34] Significant debate remains about the most appropriate temperature goals, which range from 32° C to 36° C (89.6° F to 96.8° F), although a review of available data shows that avoidance of pyrexia for a minimum of 48 hours is key to improved patient outcomes.[35] A thorough description of the different devices used in TTM is beyond the scope of this article, although it is commonly accomplished via insertion of a cooling catheter or cooling blankets/pads applied to the body.

Hemodynamic stability Multiple observational studies have addressed the relationship between postarrest BP and outcomes, but none with a specific intervention or approach.[36,37] These studies have demonstrated worsened outcomes with systolic BPs of less than both 90 and 100 mm Hg.[36,37] The optimum goal would be to meet the patient's current perfusion needs by evaluating their current volume status, myocardial contractility, and systemic vascular resistance. Because this will vary between patients, as will the ability to assess each of these factors, the AHA recommends maintaining a systolic BP greater than 90 mm Hg and a mean arterial pressure greater than 65 mm Hg.[38] Ideally, these goals will provide the appropriate degree of systemic blood flow to the most vulnerable of organs.

CONCLUSION

The evidence surrounding advanced interventions such as ET intubation, antiarrhythmics, and vasopressors for patients with inhospital cardiac arrest remains elusive. What has been demonstrated time and again is the positive effect high-quality CPR has on patient outcomes. This is something that comes with time spent in practice, simulation, and education in concert with a well-developed quality improvement program. To ensure the best outcomes for patients, hospitals must emphasize the basics and:

- Recommend ACLS education for staff
- Frequently train rapid response and code teams, ideally with regular simulation exercises including mock codes
- Reduce time to starting compressions while ensuring minimal time off of the chest
- Provide rapid defibrillation
- Choose the airway management approach that would most benefit the patient
- Identify the underlying cause of the arrest to enable rapid reversal
- Assess neurologic status immediately postarrest to determine need for TTM
- Work toward resolution of unstable patient hemodynamics to ensure adequate cardiac and cerebral perfusion. **JAAPA**

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REFERENCES

1. Virani SS, Alonso A, Benjamin EJ, et al. Heart disease and stroke statistics—2020 update: a report from the American Heart Association. *Circulation*. 2020;141(9):e139–e596.
2. Ofoma UR, Basnet S, Berger A, et al. Trends in survival after in-hospital cardiac arrest during nights and weekends. *J Am Coll Cardiol*. 2018;71(4):402-411.

3. Abella BS, Alvarado JP, Myklebust H, et al. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. *JAMA*. 2005;293(3):305-310.
4. Meaney PA, Bobrow BJ, Mancini ME, et al. Cardiopulmonary resuscitation quality: improving cardiac resuscitation outcomes both inside and outside the hospital. *Circulation*. 2013;128(4): 417-435.
5. Wallace SK, Abella BS, Becker LB. Quantifying the effect of cardiopulmonary resuscitation quality on cardiac arrest outcome: a systematic review and meta-analysis. *Circ Cardiovasc Qual Outcomes*. 2013;6(2):148-156.
6. Larsen MP, Eisenberg MS, Cummins RO, Hallstrom AP. Predicting survival from out-of-hospital cardiac arrest: a graphic model. *Ann Emerg Med*. 1993;22(11):1652-1658.
7. Winters BD, Weaver SJ, Pfoh ER, et al. Rapid-response systems as a patient safety strategy. *Ann Intern Med*. 2013;158(5 Part 2):417-425.
8. Beckett DJ, Inglis M, Oswald S, et al. Reducing cardiac arrests in the acute admissions unit: a quality improvement journey. *BMJ Qual Saf*. 2013;22(12):1025-1031.
9. Dane FC, Russell-Lindgren KS, Parish DC, et al. In-hospital resuscitation: association between ACLS training and survival to discharge. *Resuscitation*. 2000;47(1):83-87.
10. Morrison LJ, Neumar RW, Zimmerman JL, et al. Strategies for improving survival after in-hospital cardiac arrest in the United States: 2013 consensus recommendations. *Circulation*. 2013; 127(14):1538-1563.
11. Prince CR, Hines EJ, Chyou P-H, Heegeman DJ. Finding the key to a better code: code team restructure to improve performance and outcomes. *Clin Med Res*. 2014;12(1-2):47-57.
12. Seethala RR, Esposito EC, Abella BS. Approaches to improving cardiac arrest resuscitation performance. *Curr Opin Crit Care*. 2010;16(3):196-202.
13. Josey K, Smith ML, Kayani AS, et al. Hospitals with more-active participation in conducting standardized in-situ mock codes have improved survival after in-hospital cardiopulmonary arrest. *Resuscitation*. 2018;133:47-52.
14. Edelson DP, Litzinger B, Arora V, et al. Improving in-hospital cardiac arrest process and outcomes with performance debriefing. *Arch Intern Med*. 2008;168(10):1063-1069.
15. Bircher NG, Chan PS, Xu Y, et al. Delays in cardiopulmonary resuscitation, defibrillation, and epinephrine administration all decrease survival in in-hospital cardiac arrest. *Anesthesiology*. 2019;130(3):414-422.
16. Harris AW, Kudenchuk PJ. Cardiopulmonary resuscitation: the science behind the hands. *Heart*. 2018;104(13):1056-1061.
17. Idris AH, Guffey D, Aufderheide TP, et al. Relationship between chest compression rates and outcomes from cardiac arrest. *Circulation*. 2012;125(24):3004-3012.
18. Stiell IG, Brown SP, Nichol G, et al. What is the optimal chest compression depth during out-of-hospital cardiac arrest resuscitation of adult patients? *Circulation*. 2014;130(22):1962-1970.
19. Yannopoulos D, McKnite S, Aufderheide TP, et al. Effects of incomplete chest wall decompression during cardiopulmonary resuscitation on coronary and cerebral perfusion pressures in a porcine model of cardiac arrest. *Resuscitation*. 2005;64(3): 363-372.
20. Christenson J, Andrusiek D, Everson-Stewart S, et al. Chest compression fraction determines survival in patients with out-of-hospital ventricular fibrillation. *Circulation*. 2009;120(13):1241-1247.
21. Wang PL, Brooks SC. Mechanical versus manual chest compressions for cardiac arrest. *Cochrane Database Syst Rev*. 2018;8(8): CD007260.
22. Neumar RW, Brown CG, Ligten PV, et al. Estimation of myocardial ischemic injury during ventricular fibrillation with total circulatory arrest using high-energy phosphates and lactate as metabolic markers. *Ann Emerg Med*. 1991;20(3):222-229.
23. Chan PS, Nichol G, Krumholz HM, et al. Hospital variation in time to defibrillation after in-hospital cardiac arrest. *Arch Intern Med*. 2009;169(14):1265-1273.
24. Panchal AR, Berg KM, Hirsch KG, et al. 2019 American Heart association focused update on advanced cardiovascular life support: use of advanced airways, vasopressors, and extracorporeal cardiopulmonary resuscitation during cardiac arrest: an update to the American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2019;140(24):e881-e894.
25. Aufderheide TP, Sigurdsson G, Pirrallo RG, et al. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. *Circulation*. 2004;109(16):1960-1965.
26. Benger JR, Kirby K, Black S, et al. Effect of a strategy of a supraglottic airway device vs tracheal intubation during out-of-hospital cardiac arrest on functional outcome: the AIRWAYS-2 randomized clinical trial. *JAMA*. 2018;320(8): 779-791.
27. Perkins GD, Ji C, Deakin CD, et al. A randomized trial of epinephrine in out-of-hospital cardiac arrest. *N Engl J Med*. 2018;379(8):711-721.
28. Soar J. Epinephrine for cardiac arrest: knowns, unknowns and controversies. *Curr Opin Crit Care*. 2020;26(6):590-595.
29. Andersen LW, Kurth T, Chase M, et al. Early administration of epinephrine (adrenaline) in patients with cardiac arrest with initial shockable rhythm in hospital: propensity score matched analysis. *BMJ*. 2016;353:i1577.
30. Donnino MW, Salciccioli JD, Howell MD, et al. Time to administration of epinephrine and outcome after in-hospital cardiac arrest with non-shockable rhythms: retrospective analysis of large in-hospital data registry. *BMJ*. 2014;348:g3028.
31. Panchal AR, Berg KM, Kudenchuk PJ, et al. 2018 American Heart Association focused update on advanced cardiovascular life support use of antiarrhythmic drugs during and immediately after cardiac arrest: an update to the American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2018;138(23):e740-e749.
32. Neumar RW, Otto CW, Link MS, et al. Part 8: Adult advanced cardiovascular life support: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2010;122(18 suppl 3):S729-S767.
33. Kudenchuk PJ, Daya M, Dorian P, et al. Amiodarone, lidocaine, or placebo in out-of-hospital cardiac arrest. *N Engl J Med*. 2016;375(8):801-803.
34. Nielsen N, Wetterslev J, Cronberg T, et al. Targeted temperature management at 33°C versus 36°C after cardiac arrest. *N Engl J Med*. 2013;369(23):2197-2206.
35. Lascarrou J-B, Merdji H, Le Gouge A, et al. Targeted temperature management for cardiac arrest with nonshockable rhythm. *N Engl J Med*. 2019;381(24):2327-2337.
36. Bray JE, Bernard S, Cantwell K, et al. The association between systolic blood pressure on arrival at hospital and outcome in adults surviving from out-of-hospital cardiac arrests of presumed cardiac aetiology. *Resuscitation*. 2014;85(4):509-515.
37. Kilgannon JH, Roberts BW, Reihl LR, et al. Early arterial hypotension is common in the post-cardiac arrest syndrome and associated with increased in-hospital mortality. *Resuscitation*. 2008;79(3):410-416.
38. Callaway CW, Donnino MW, Fink EL, et al. Part 8: Post-cardiac arrest care: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2015;132(18 suppl 2):S465-S482.