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In-Hospital Cardiac Arrest:

A Review

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Abstract

IMPORTANCE—In-hospital cardiac arrest is common and associated with a high mortality rate. Despite this, in-hospital cardiac arrest has received little attention compared with other high-risk cardiovascular conditions, such as stroke, myocardial infarction, and out-of-hospital cardiac arrest.

OBSERVATIONS—In-hospital cardiac arrest occurs in over 290 000 adults each year in the United States. Cohort data from the United States indicate that the mean age of patients with in-

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hospital cardiac arrest is 66 years, 58% are men, and the presenting rhythm is most often (81%) nonshockable (ie, asystole or pulseless electrical activity). The cause of the cardiac arrest is most often cardiac (50%-60%), followed by respiratory insufficiency (15%-40%). Efforts to prevent in-hospital cardiac arrest require both a system for identifying deteriorating patients and an appropriate interventional response (eg, rapid response teams). The key elements of treatment during cardiac arrest include chest compressions, ventilation, early defibrillation, when applicable, and immediate attention to potentially reversible causes, such as hyperkalemia or hypoxia. There is limited evidence to support more advanced treatments. Post–cardiac arrest care is focused on identification and treatment of the underlying cause, hemodynamic and respiratory support, and potentially employing neuroprotective strategies (eg, targeted temperature management). Although multiple individual factors are associated with outcomes (eg, age, initial rhythm, duration of the cardiac arrest), a multifaceted approach considering both potential for neurological recovery and ongoing multiorgan failure is warranted for prognostication and clinical decision-making in the Post–cardiac arrest period. Withdrawal of care in the absence of definite prognostic signs both during and after cardiac arrest should be avoided. Hospitals are encouraged to participate in national quality-improvement initiatives.

CONCLUSIONS AND RELEVANCE—An estimated 290 000 in-hospital cardiac arrests occur each year in the United States. However, there is limited evidence to support clinical decision making. An increased awareness with regard to optimizing clinical care and new research might improve outcomes.

In-hospital cardiac arrest is an acute event that can potentially affect any hospitalized patient. For the purposes of clinical care, research, and guideline development, in-hospital cardiac arrest (as opposed to death without resuscitation) is most commonly defined as the loss of circulation prompting resuscitation with chest compressions, defibrillation, or both.

Traditionally, in-hospital cardiac arrest has been viewed as a condition with such poor outcomes that resuscitation may not even be warranted. Although outcomes remain poor, recent data suggest improvement over the past 2 decades.[1,2] One reason for this improvement might be an increased awareness of the influence that clinical management can have on outcomes in patients with in-hospital cardiac arrest and cardiac arrest in general. Despite this increased interest, in-hospital cardiac arrest remains a somewhat neglected condition compared with out-of-hospital cardiac arrest and other cardiovascular conditions, such as stroke and myocardial infarction. For example, in a systematic review of all randomized clinical cardiac arrest trials (n = 92) involving at least 50 patients from 1995 to 2014, only 4 (4%) exclusively involved patients with in-hospital cardiac arrest.[3] Although guidelines for in- and out-of-hospital cardiac arrest are almost identical,[4,5] there are important differences between the conditions that warrant consideration (Table 1).

In this review, we discuss adult in-hospital cardiac arrest, including epidemiology, causes, management during and after cardiac arrest, characteristics related to outcomes, prognostication, and quality improvement. There are relatively few randomized clinical trials of patients with in-hospital cardiac arrest (Table 2). Therefore, much of the current knowledge is based on observational studies primarily from large registries, extrapolation of results from trials of out-of-hospital cardiac arrest, and expert opinion.

Methods

This review was based on a series of informal searches of PubMed addressing each relevant topic, including incidence and outcomes, causes, prevention, treatment, and prognostication of in-hospital cardiac arrest. In addition, we performed a systematic search to identify all randomized trials including patients with in-hospital cardiac arrest published within the past 30 years. Additional details regarding the systematic search are provided in Supplement 1.

Results

Incidence and Outcomes

The global incidence of in-hospital cardiac arrest in adults has not been well described,[29,30] and the majority of data are derived from the American Heart Association's Get With The Guidelines-Resuscitation (GWTG-R) registry[31] and the National Cardiac Arrest Audit from the Resuscitation Council (UK) and the Intensive Care National Audit and Research Centre.[32] Based on GWTG-R data from 2003 to 2007, the estimated incidence of in-hospital cardiac arrests in the United States was 211 000 annually, or roughly 6 to 7 cardiac arrests per 1000 admissions.[30,31] Data from 2008 to 2017 showed the incidence increased to 292 000 annually, or 9 to 10 in-hospital cardiac arrests per 1000 admissions.[33] In contrast, an incidence of 1.6 in-hospital cardiac arrests per 1000 admissions in the United Kingdom from 2011-2013 was estimated using data from the UK National Cardiac Arrest Audit.[32]

Based on data from the GWTG-R registry, the mean age of patients with in-hospital cardiac arrest in the United States is 66 years, 58% are men, and the presenting rhythm is most often (81%) nonshockable (ie, asystole or pulseless electrical activity). Approximately half of in-hospital cardiac arrests occur in wards, with the remaining half occurring in other locations, such as intensive care units and operating rooms.[2,32,34]

In a review from 2007, survival (most commonly to hospital discharge) varied from 0% to 42% between studies, although most larger studies reported survival around 20%.[29] Survival has been increasing over the last 2 decades (Figure 1)[1,2] and, in 2017, survival to hospital discharge was 25% in the GWTG-R registry. Among patients alive at hospital discharge, 85% were discharged with a favorable neurological outcome (cerebral performance category 1 or 2).[2] Data from 2011 to 2013 indicate 18% survival to hospital discharge in the United Kingdom[32] while, in Denmark and Sweden, 30-day survival is approximately 30% in contemporary national registries.[35,36] A 2018 systematic review that included more than 1 million in-hospital cardiac arrests and 39 studies from 1992 to 2016 found an overall 1-year survival rate of 13%, with large between-study variability and an increase in 1-year survival over time.[37] In elderly (aged ≥65 years) patients with in-hospital cardiac arrest in the United States who survived to hospital discharge, 59% were alive after 1 year and 34% had not been readmitted to a hospital.[38]

The variability between countries in both incidence of and survival after in-hospital cardiac arrest likely reflects differences in (1) the definitions used to identify in-hospital cardiac arrest, (2) the proportion of cardiac arrests captured by various registries, (3) the patient populations, (4) country-specific culture surrounding cardiopulmonary resuscitation (CPR),

do-not-resuscitate orders, and withdrawal of care, and (5) treatment during and after cardiac arrest. Comparisons between countries or registries should therefore be performed carefully.

Causes of Cardiac Arrest

Historically, the etiologies of cardiac arrest have been dichotomized as cardiac or noncardiac. Because patients with no obvious cause are generally classified as cardiac, and because discrepancies often exist between clinical and postmortem diagnoses, the causes of cardiac arrest are often uncertain. In general, cardiac causes of cardiac arrest, such as myocardial infarction, arrhythmia, or heart failure, are most frequent, with a prevalence of approximately 50% to 60%. Respiratory insufficiency is the second most common cause (15%-40%).[34,39,40] The median admission duration prior to cardiac arrest is 1 to 2 days, with a higher prevalence of respiratory insufficiency as the cause of cardiac arrest with longer duration of preceding hospitalization.[32,36,40] Neurological causes of cardiac arrest are rare in the in-hospital setting.[36,41]

Identifying the cause of cardiac arrest serves several purposes. During cardiac arrest, resuscitation guidelines emphasize that potential reversible causes should be identified, which are categorized into 4 *h*'s and *t*'s (Box).[42] Although not all of these categories (eg, hypothermia) are applicable in the in-hospital setting, the majority of in-hospital cardiac arrests can be categorized using this approach.[43] Identifying the cause of cardiac arrest could improve outcomes.[44] Identification of the cause of cardiac arrest also has implications if return of spontaneous circulation is achieved, because Post–cardiac arrest organ dysfunction is partly dependent on the underlying cause, and post–cardiac arrest treatment should be tailored accordingly (see "Treatment After Cardiac Arrest"). Additional research is needed to create a useful and more precise framework for the classification of causes of cardiac arrest. Such a framework might not only improve research efforts but may eventually improve care provided during and after cardiac arrest.

Prevention of cardiac arrest will be best achieved by addressing the mechanisms underlying the cause of the cardiac arrest. For example, the prescription of QT interval–prolonging drugs during hospital admission can lead to arrhythmias, while the prescription of opioids or sedatives may lead to respiratory insufficiency [45,46] Another potentially preventable cause of cardiac arrest is sepsis. The prevalence of preexisting sepsis in patients with in-hospital cardiac arrest varies across studies, ranging from 13% to 27%.[34,47] Organ failure from sepsis contributes to multiple potential causes of arrest, including circulatory failure, respiratory insufficiency, and metabolic derangements.

Prevention of Cardiac Arrest

Contrary to the out-of-hospital setting, in-hospital cardiac arrest facilitates observation of a patient's clinical condition prior to cardiac arrest. Clinical deterioration is common prior to in-hospital cardiac arrest,[48] and many in-hospital cardiac arrests are considered preventable or avoidable on retrospective review.[49] Prevention has therefore been added as the first link in the Chain of Survival for in-hospital cardiac arrest in the 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care.[50] Key elements of success include identification of at-risk patients

combined with early interventions to prevent deterioration to cardiac arrest. Such identification might occur through the use of early warning systems triggered by specific vital sign abnormalities, a scoring system based on multiple criteria, or by staff concern. However, current prediction models often lack optimal sensitivity and/or specificity to identify at-risk patients and may be limited by differences in the logistical structure of individual hospital systems.[51] Further, although interventions in terms of rapid response teams are generally supported by the literature, the evidence is weak because there are few rigorous randomized clinical trials and heterogeneity between hospital systems.[52] Despite these limitations, individual hospitals should create a 2-part system for preventing cardiac arrest that includes (1) identifying at-risk or deteriorating patients (requiring relevant education, monitoring, and recognition) and (2) creating appropriate interventional responses (eg, rapid response teams).

Treatment During Cardiac Arrest

Chest compressions, ventilation, and early defibrillation, when applicable, are the cornerstones of cardiac arrest treatment.[4,5] Early initiation of CPR is associated with improved outcomes for both out-of-hospital[53] and in-hospital[54] cardiac arrest. CPR training for all hospital personnel has, therefore, been mandatory in many hospital systems for decades, facilitating the rapid identification and management of cardiac arrest prior to the arrival of the cardiac arrest team. Quality of chest compressions and of CPR in general have been associated with better outcomes in patients with cardiac arrest.[55] Optimization of CPR quality is therefore a priority.

Although only approximately 20% of patients with in-hospital cardiac arrest have an initial shockable rhythm, rapid defibrillation is associated with improved outcomes for these patients.[56] It is not clear whether automated (compared with manual) external defibrillators, which have been associated with markedly improved outcomes in the out-of-hospital setting,[57] are of any benefit in the in-hospital setting.[58]

Data supporting the efficacy of medications during in-hospital cardiac arrest are sparse. Current guidelines recommend the use of epinephrine and amiodarone, both of which improve short-term outcomes in out-of-hospital cardiac arrest, but there is limited evidence supporting substantial neurological improvement when these medications are used.[59,60] Given the differences between in-hospital and out-of-hospital cardiac arrest (Table 1), especially the much earlier administration of drugs in the in-hospital setting, it is unclear whether findings from studies of out-of-hospital cardiac arrest apply to in-hospital cardiac arrest. For in-hospital events, early administration of epinephrine in patients with a nonshockable rhythm is associated with better outcomes.[61] In contrast, early epinephrine for patients with shockable rhythms is associated with worse outcomes.[62] The combination of vasopressin and methylprednisolone during in-hospital cardiac arrest has been tested in 2 small randomized clinical trials, with promising results.[18,21] This combination of drugs is not recommended in the US or European guidelines because of insufficient evidence to support their use (see eTable 1 in Supplement 1 for an overview of ongoing randomized clinical trials).[42,63]

Airway management is a key component of advanced life support during cardiac arrest. Endotracheal intubation has traditionally been considered the preferred approach to ensure adequate ventilation and oxygenation, but emerging evidence in both out-of-hospital[64-66] and in-hospital[67] cardiac arrest suggests that alternative approaches, such as bag-valve-mask ventilation or supraglottic airways, may be equally or even more effective. How to optimize ventilation and oxygenation likely depends on the specific clinical conditions of the patient with cardiac arrest.

Extracorporeal circulation during CPR is being assessed for use in cardiac arrest and it may be useful for in-hospital events and in certain patient populations, such as in patients who have recently undergone cardiac surgery. Some reports have shown good outcomes from extracorporeal circulation during CPR but, overall, the evidence of benefit is scarce and limited by the observational nature of the studies.[68] Extracorporeal circulation during CPR may help carefully selected patients, but balance must be achieved between efficient resource use and clinical benefits.

Treatment After Cardiac Arrest

Management in the Post–cardiac arrest period generally focuses on the precipitating cause, hemodynamic and respiratory support, and neuroprotective care. Conditions before and during cardiac arrest determine the severity of the Post–cardiac arrest syndrome (see “Characteristics Related to Outcomes”) and the need for various interventions. For example, a patient admitted with acute coronary syndrome who develops ventricular fibrillation that is rapidly treated with defibrillation may be cognitively intact and require treatment specific for the cardiac condition without need of neuroprotection. Conversely, a patient who has a prolonged cardiac arrest and significant ischemia-reperfusion injury may have multiorgan injury and require numerous treatments. One of the more distinguishing components of in-hospital cardiac arrest is that the event may result from progressively worsening underlying disease, whereas out-of-hospital arrest is often sudden and unpredictable.

Despite some controversy, targeted temperature management at 32°C to 36°C for at least 24 hours after cardiac arrest remains the primary neuroprotective approach following out-of-hospital cardiac arrest.[69,70] To our knowledge, no randomized trials have evaluated targeted temperature management following in-hospital cardiac arrest. Outcomes data are limited to observational studies and extrapolation from the out-of-hospital investigations. The largest observational study of targeted temperature management in patients with in-hospital cardiac arrest (N = 26 183) found that it was associated with worse overall outcomes.[71] However, this study did not consider coma status, which could have biased the results against the intervention because targeted temperature management is only used in comatose patients.[72] While future studies are needed to better evaluate targeted temperature management for patients with in-hospital cardiac arrest (eTable 1 in Supplement 1), the current American Heart Association recommendation is to provide targeted temperature management for at least 24 hours.[70]

Other proposed neuroprotective strategies for in-hospital cardiac arrest supported by indirect observational data include minimizing supplemental oxygen therapy when oxygen levels are adequate and maintaining normal carbon dioxide levels.[73,74] Observational studies that

examined the association between the maintenance of low tidal-volume ventilation and outcomes in patients with out-of-hospital cardiac arrest[75] and in-hospital cardiac arrest[76] have shown conflicting results. While further studies are necessary, one important element to note is that patients who experienced cardiac arrest rarely die from refractory respiratory failure,[77,78] potentially limiting the effectiveness of ventilator-based interventions.

Hemodynamic management is essential, but no specific differences have been defined in the management of patients after cardiac arrest compared with other critically ill patients. Preexisting disease, underlying diagnoses, and myocardial stunning from ischemia reperfusion all contribute to the hemodynamic profile of patients after cardiac arrest. Targeted temperature management appears to have some effects on hemodynamics and data from a small 2002 trial suggested that patients receiving targeted temperature management had lower heart rates, increased systematic vascular resistance, and slightly decreased cardiac outputs.[79] A 2015 analysis of patients cooled to 33°C vs 36°C reported higher vasopressor dosages and lactate levels for patients maintained at 33°C.[80] Consequently, patients can be maintained with higher temperatures (ie, 36°C) if there is a concern that targeted temperature management negatively influences hemodynamics.[81] Targeted temperature management at 33°C should probably be avoided in patients with sepsis and septic shock[82] or bacterial meningitis,[83] because recent trials suggest worse outcomes in these patient groups with targeted temperature management.

Characteristics Related to Outcomes

Many patient and event characteristics are associated with the clinical outcomes of in-hospital cardiac arrest. Some of these characteristics cannot be modified, such as age, gender, and preexisting conditions, while other characteristics, such as time to drug administration and monitoring, are modifiable and the subject of quality-improvement efforts. While many risk factors, such as advanced age, are predictive of clinical outcomes, none can individually be used to estimate prognosis following cardiac arrest (see “Prognostication”).

Increased age is associated with decreased survival following cardiac arrest in most studies, especially for patients older than 70 years.[84] The association with outcomes of other demographic features, such as sex and race, is less clear. Although the incidence of in-hospital cardiac arrest is higher among men, men and women have similar clinical outcomes, [85] although women of child-bearing age (15 to 44 years) may have better outcomes compared with men of the same age.[86] Studies that investigated the relationship between race and outcomes have found black and Hispanic patients to have lower rates of neurological recovery and survival following in-hospital cardiac arrest compared with white patients.[47,87] Data from the GWTG-R registry have shown that racial disparities in outcomes have narrowed over time, with a reported absolute survival difference between black and white patients of 4.5% in 2000 and 1.8% in 2014.[87] Differing distribution of risk factors[88] or variability in patient- and hospital-level care during and after cardiac arrest may explain why these racial differences exist.

The presence of preexisting medical and surgical conditions is strongly associated with outcomes following in-hospital cardiac arrest. For example, malignancy, sepsis, poor

functional status prior to the cardiac arrest, pneumonia, hypotension, renal dysfunction, and hepatic dysfunction have been identified as significant predictors of poor survival.[84,89] Conversely, acute myocardial infarction causing an in-hospital cardiac arrest is associated with increased survival compared with cardiac arrest not caused by myocardial infarction.[90]

Factors related to early detection of cardiac arrest, such as the event being witnessed[47,91] or occurring in a monitored location,[34,47,84] are associated with improved outcomes. However, the association between monitoring/location and outcomes[34,47,84,91] is complex given the different case-mix of patients in various locations.

Two of the factors most strongly associated with outcomes are the presenting rhythm and the duration of the cardiac arrest.[84,92,93] Patients with a shockable rhythm have 2 to 3 times higher survival to hospital discharge compared with patients with a nonshockable rhythm (Figure 1).[2,92] While this difference might reflect the potential for more effective treatment in the shockable group (ie, defibrillation), part of the difference is likely also explained by differences in patient characteristics and preexisting conditions, which may influence the presenting rhythm. The chance of 30-day survival markedly decreases with increasing duration of CPR.[93]

Prognostication

During Cardiac Arrest—Deciding when to stop CPR during cardiac arrest remains challenging, with limited guidance in contemporary guidelines.[94,95] Although longer duration of resuscitation is associated with worse outcomes, survival with good neurological outcome is possible with prolonged CPR.[93] Additionally, Goldberger et al[96] found that hospitals where CPR is performed for longer durations have better outcomes, suggesting that average CPR duration may be too short at some hospitals.

Several observational studies have reported that cardiac standstill on point-of-care cardiac ultrasonography is associated with very low likelihood of survival.[97] However, there are significant concerns with interrater variability in image interpretation and potential interference of ultrasonography with CPR.[98] End-tidal carbon dioxide values less than 10 mm Hg after 20 minutes of CPR are also strongly associated with poor outcomes.[99] However, studies regarding point-of-care cardiac ultrasonography and end-tidal carbon dioxide should be interpreted carefully because blinding was rarely performed and the findings described might reflect a self-fulfilling prophecy where resuscitation is terminated based on these specific findings. Neither cardiac ultrasonography nor end-tidal carbon dioxide should be used in isolation, but might be considered together with other factors, such as the initial rhythm and the duration of the cardiac arrest (see “Characteristics Related to Outcomes”).

After Cardiac Arrest—Current guidelines on prognostication are based on literature describing out-of-hospital cardiac arrest and focus exclusively on neurological status.[70] While two-thirds of patients with out-of-hospital cardiac arrest who survive to intensive care unit admission die of neurological causes, neurologic death only occurs in one-fourth of patients with in-hospital cardiac arrest, for whom multiorgan dysfunction drives mortality.[78] Because in-hospital cardiac arrest is most often witnessed, times from cardiac arrest to

initiation of CPR and to return of spontaneous circulation are shorter than in out-of-hospital cardiac arrest, which may contribute to the lower rate of neurological injury. These differences leave clinicians without clear guidance regarding how to best prognosticate outcomes (ie, survival and survival with good neurological/functional recovery) following in-hospital cardiac arrest.

Chan et al attempted to address this issue by developing a scoring system for estimation of survival to hospital discharge with a favorable neurological outcome in patients with return of spontaneous circulation after in-hospital cardiac arrest (Figure 2; Supplement 2).[84] The final score, which has been externally validated,[100] is based on 11 parameters available immediately after the cardiac arrest. The score successfully categorized patients with varying chances of hospital survival.[84] Such a scoring system can inform conversations about goals of care when patients survive the initial cardiac arrest but remain seriously ill. However, the score is not able to accurately identify many patients with a very low/no chance of survival[84] and should therefore not be used in isolation for decisions regarding withdrawal of care.

Treatment recommendations for patients with in-hospital cardiac arrest with presumed severe neurological impairment following the arrest are based on studies of patients with out-of-hospital cardiac arrest.[70] A key recommendation is that neuroprognostication should not be performed too early, especially when targeted temperature management is used, because slower metabolism of sedatives and neuromuscular blockade may occur. Several studies have found that delayed awakening (>48 hours after cessation of sedation) is common in patients after cardiac arrest.[101] In a study of patients still in a coma 7 days after cardiac arrest, 22% obtained a favorable neurological outcome at 6 months.[102] The American Heart Association recommends that neuroprognostication based on physical examination findings should be deferred until at least 72 hours after return of spontaneous circulation or rewarming (if targeted temperature management is used), and often longer if effects of sedation or neuromuscular blockade may still be present.[70]

The clinical examination findings most predictive of poor outcomes are absent pupillary light reflexes and absent corneal reflexes after 72 hours, as well as status myoclonus (continuous, prolonged, and generalized myoclonus) within 72 to 120 hours of the cardiac arrest. Extensor motor responses and intermittent myoclonus are less predictive of outcomes and should not be relied upon. On electroencephalography, persistent burst suppression after rewarming and nonreactivity predict poor outcomes, as does the absence of somatosensory evoked potentials at 72 hours. Cerebral edema on early head computed tomographic imaging and restricted diffusion on magnetic resonance imaging at 2 to 6 days after return of spontaneous circulation are potentially helpful in predicting outcomes, but cannot preclude a favorable neurological outcome. Biomarkers for the degree of neurological injury (eg, neuron-specific enolase and S100 calcium-binding protein B) do not adequately predict outcomes by themselves, but may be helpful when considered with other clinical features that predict outcomes.[70,103]

Little evidence exists for any one tool for prognostication after in-hospital cardiac arrest. A multifaceted approach that assesses the neurological prognosis combined with ongoing

organ failure is prefered.[102,104-106] Information regarding neurological prognosis and organ failure can be considered along with patient and cardiac arrest characteristics to support clinical decision making.

Quality Improvement

Organizations have proposed strategies and quality-improvement initiatives to improve outcomes after in-hospital cardiac arrest. For example, the GWTG-R registry currently tracks a number of quality-improvement measures, including the proportion of cardiac arrests that are monitored or witnessed, the time to relevant intervention (defibrillation for shockable rhythms and epinephrine for nonshockable rhythms), and confirmation of correct airway placement.[107] Observational studies have shown that longer duration of participation in the GWTG-R registry is associated with both improved quality of care[108] and increased return of spontaneous circulation.[109] Specific adherence to performance measures is also associated with improvement in outcomes.[110] Other aspects of cardiac arrest care, such as training, monitoring, and cardiac arrest team composition, are also potential targets for quality improvement. To monitor and improve quality of care for patients with in-hospital cardiac arrest, hospitals are encouraged to participate in national registries (eTable 2 in Supplement 1).

Limitations

The aim of this review was to provide an overview of in-hospital cardiac arrest. As such, many topics are only briefly discussed and others have been omitted. We did not perform comprehensive systematic reviews to address all topics and, therefore, relevant studies might have inadvertently been omitted. The data and recommendations provided herein are limited by the available data and, in some cases, reflect expert opinion.

Conclusions

An estimated 290 000 in-hospital cardiac arrests occur each year in the United States. However, there is limited evidence to support clinical decision making. An increased awareness with regard to optimizing clinical care and new research might improve outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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REFERENCES

1. Girotra S, Nallamothu BK, Spertus JA, Li Y, Krumholz HM, Chan PS; American Heart Association Get with the Guidelines–Resuscitation Investigators. Trends in survival after in-hospital cardiac arrest. N Engl J Med. 2012;367(20):1912–1920. doi:10.1056/NEJMoa1109148 [PubMed: 23150959]

2. Benjamin EJ, Virani SS, Callaway CW, et al.; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2018 Update: a report from the American Heart Association. Circulation. 2018;137(12):e67–e492. doi:10.1161/CIR.0000000000000558 [PubMed: 29386200]
3. Sinha SS, Sukul D, Lazarus JJ, et al. Identifying important gaps in randomized controlled trials of adult cardiac arrest treatments: a systematic review of the published literature. Circ Cardiovasc Qual Outcomes. 2016;9(6):749–756. doi:10.1161/CIRCOUTCOMES.116.002916 [PubMed: 27756794]
4. Neumar RW, Shuster M, Callaway CW, et al. Part 1: Executive Summary: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2015;132(18)(suppl 2):S315–S367. doi:10.1161/CIR.0000000000000252 [PubMed: 26472989]
5. Monsieurs KG, Nolan JP, Bossaert LL, et al.; ERC Guidelines 2015 Writing Group. European Resuscitation Council Guidelines for Resuscitation 2015: Section 1. Executive summary. Resuscitation. 2015;95:1–80. doi:10.1016/j.resuscitation.2015.07.038 [PubMed: 26477410]
6. Brain Resuscitation Clinical Trial II Study Group. A randomized clinical study of a calcium-entry blocker (lidoflazine) in the treatment of comatose survivors of cardiac arrest. N Engl J Med. 1991;324 (18):1225–1231. doi:10.1056/NEJM199105023241801 [PubMed: 2014035]
7. Stiell IG, Hebert PC, Weitzman BN, et al. High-dose epinephrine in adult cardiac arrest. N Engl J Med 1992;327(15):1045–1050. doi:10.1056/NEJM199210083271502 [PubMed: 1522840]
8. Sack JB, Kesselbrenner MB, Bregman D. Survival from in-hospital cardiac arrest with interposed abdominal counterpulsation during cardiopulmonary resuscitation. JAMA. 1992;267(3): 379–385. doi:10.1001/jama.1992.03480030057037 [PubMed: 1727961]
9. Sack JB, Kesselbrenner MB, Jarrad A. Interposed abdominal compression-cardiopulmonary resuscitation and resuscitation outcome during asystole and electromechanical dissociation. Circulation. 1992;86(6):1692–1700. doi:10.1161/01.CIR.86.6.1692 [PubMed: 1451240]
10. Cohen TJ, Goldner BG, Maccaro PC, et al. A comparison of active compression-decompression cardiopulmonary resuscitation with standard cardiopulmonary resuscitation for cardiac arrests occurring in the hospital. N Engl J Med. 1993;329(26):1918–1921. doi:10.1056/NEJM199312233292603 [PubMed: 8018138]
11. Lipman J, Wilson W, Kobilski S, et al. High-dose adrenaline in adult in-hospital asystolic cardiopulmonary resuscitation: a double-blind randomised trial. Anaesth Intensive Care. 1993;21(2):192–196. [PubMed: 8517510]
12. Tucker KJ, Galli F, Savitt MA, Kahsai D, Bresnahan L, Redberg RF. Active compression-decompression resuscitation: effect on resuscitation success after in-hospital cardiac arrest. J Am Coll Cardiol. 1994;24(1):201–209. doi:10.1016/0735-1097(94)90564-9 [PubMed: 8006266]
13. Woodhouse SP, Cox S, Boyd P, Case C, Weber M. High dose and standard dose adrenaline do not alter survival, compared with placebo, in cardiac arrest. Resuscitation. 1995;30(3):243–249. doi:10.1016/0300-9572(95)00890-X [PubMed: 8867714]
14. Patrick WD, Freedman J, McEwen T, Light RB, Ludwig L, Roberts D. A randomized, double-blind comparison of methoxamine and epinephrine in human cardiopulmonary arrest. Am J Respir Crit Care Med. 1995;152(2):519–523. doi:10.1164/ajrccm.152.2.7633701 [PubMed: 7633701]
15. Stiell IG, Hébert PC, Wells GA, et al. The Ontario trial of active compression-decompression cardiopulmonary resuscitation for in-hospital and prehospital cardiac arrest. JAMA. 1996;275(18):1417–1423. doi:10.1001/jama.1996.03530420045034 [PubMed: 8618367]
16. Thel MC, Armstrong AL, McNulty SE, Califf RM, O'Connor CM; Duke Internal Medicine Housestaff. Randomised trial of magnesium in in-hospital cardiac arrest. Lancet. 1997;350(9087):1272–1276. doi:10.1016/S0140-6736(97)05048-4 [PubMed: 9357406]
17. Stiell IG, Hébert PC, Wells GA, et al. Vasopressin versus epinephrine for inhospital cardiac arrest: a randomised controlled trial. Lancet. 2001;358(9276):105–109. doi:10.1016/S0140-6736(01)05328-4 [PubMed: 11463411]
18. Mentzelopoulos SD, Zakynthinos SG, Tzoufi M, et al. Vasopressin, epinephrine, and corticosteroids for in-hospital cardiac arrest. Arch Intern Med. 2009;169(1):15–24. doi:10.1001/archinternmed.2008.509 [PubMed: 19139319]

19. Weidman EK, Bell G, Walsh D, Small S, Edelson DP. Assessing the impact of immersive simulation on clinical performance during actual in-hospital cardiac arrest with CPR-sensing technology: a randomized feasibility study. Resuscitation. 2010;81(11):1556–1561. doi:10.1016/j.resuscitation.2010.05.021 [PubMed: 20724057]
20. Pittl U, Schratter A, Desch S, et al. Invasive versus non-invasive cooling after in- and out-of-hospital cardiac arrest: a randomized trial. Clin Res Cardiol. 2013;102(8):607–614. doi:10.1007/s00392-013-0572-3 [PubMed: 23644718]
21. Mentzelopoulos SD, Malachias S, Chamos C, et al. Vasopressin, steroids, and epinephrine and neurologically favorable survival after in-hospital cardiac arrest: a randomized clinical trial. JAMA. 2013;310(3):270–279. doi:10.1001/jama.2013.7832 [PubMed: 23860985]
22. Vahedian-Azimi A, Hajiesmaeili M, Amirsavadkouhi A, et al. Effect of the Cardio First Angel device on CPR indices: a randomized controlled clinical trial. Crit Care. 2016;20(1):147. doi:10.1186/s13054-016-1296-3 [PubMed: 27184664]
23. Eastwood GM, Schneider AG, Suzuki S, et al. Targeted therapeutic mild hypercapnia after cardiac arrest: A phase II multi-centre randomised controlled trial (the CCC trial). Resuscitation. 2016;104:83–90. doi:10.1016/j.resuscitation.2016.03.023 [PubMed: 27060535]
24. Movahedi A, Mirhafez SR, Behnam-Voshani H, et al. A comparison of the effect of interposed abdominal compression cardiopulmonary resuscitation and standard cardiopulmonary resuscitation methods on end-tidal CO2 and the return of spontaneous circulation following cardiac arrest: a clinical trial. Acad Emerg Med. 2016;23(4): 448–454. doi:10.1111/acem.12903 [PubMed: 26801358]
25. Anantharaman V, Tay SY, Manning PG, et al. A multicenter prospective randomized study comparing the efficacy of escalating higher biphasic versus low biphasic energy defibrillations in patients presenting with cardiac arrest in the in-hospital environment. Open Access Emerg Med. 2017;9:9–17. doi:10.2147/OAEM.S109339 [PubMed: 28144168]
26. Koster RW, Beenen LF, van der Boom EB, et al. Safety of mechanical chest compression devices AutoPulse and LUCAS in cardiac arrest: a randomized clinical trial for non-inferiority. Eur Heart J. 2017;38(40):3006–3013. doi:10.1093/eurheartj/ehx318 [PubMed: 29088439]
27. Zhang Q, Li C, Shao F, Zhao L, Wang M, Fang Y. Efficacy and safety of combination therapy of shenfu injection and postresuscitation bundle in patients with return of spontaneous circulation after in-hospital cardiac arrest: a randomized, assessor-blinded, controlled trial. Crit Care Med. 2017;45(10):1587–1595. doi:10.1097/CCM.0000000000002570 [PubMed: 28661970]
28. Look X, Li H, Ng M, et al. Randomized controlled trial of internal and external targeted temperature management methods in post- cardiac arrest patients. Am J Emerg Med. 2018;36(1):66–72. doi:10.1016/j.ajem.2017.07.017 [PubMed: 28698133]
29. Sandroni C, Nolan J, Cavallaro F, Antonelli M. In-hospital cardiac arrest: incidence, prognosis and possible measures to improve survival. Intensive Care Med. 2007;33(2):237–245. doi:10.1007/s00134-006-0326-z [PubMed: 17019558]
30. Morrison LJ, Neumar RW, Zimmerman JL, et al.; American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular and Stroke Nursing, Council on Clinical Cardiology, and Council on P. Strategies for improving survival after in-hospital cardiac arrest in the United States: 2013 consensus recommendations: a consensus statement from the American Heart Association. Circulation. 2013;127(14):1538–1563. doi:10.1161/CIR.0b013e31828b2770 [PubMed: 23479672]
31. Merchant RM, Yang L, Becker LB, et al.; American Heart Association Get With The Guidelines-Resuscitation Investigators. Incidence of treated cardiac arrest in hospitalized patients in the United States. Crit Care Med. 2011;39(11):2401–2406. doi:10.1097/CCM.0b013e3182257459 [PubMed: 21705896]
32. Nolan JP, Soar J, Smith GB, et al.; National Cardiac Arrest Audit. Incidence and outcome of in-hospital cardiac arrest in the United Kingdom National Cardiac Arrest Audit. Resuscitation. 2014;85(8):987–992. doi:10.1016/j.resuscitation.2014.04.002 [PubMed: 24746785]
33. Holmberg M, Ross C, Chan P, et al. Incidence of adult in-hospital cardiac arrest in the United States. Abstract presented at: American Heart Association Resuscitation Science Symposium; November 10-11, 2018; Chicago, IL.

34. Perman SM, Stanton E, Soar J, et al.; American Heart Association's Get With the Guidelines®—Resuscitation Investigators. Location of in-hospital cardiac arrest in the United States: variability in event rate and outcomes. J Am Heart Assoc. 2016;5(10):e003638. doi:10.1161/JAHA.116.003638 [PubMed: 27688235]
35. DANARREST Steering Commitee. DANARREST: Registrering af hjertestop på hospital. Årsrapport 2015 [Danish]. https://www.sundhed.dk/content/cms/83/70283_danarrest-%C3%A5rsrapport-2015.pdf. Published 6 30, 2016 Accessed June 5, 2017.
36. Tran S, Deacon N, Minokadeh A, et al. Frequency and survival pattern of in-hospital cardiac arrests: the impacts of etiology and timing. Resuscitation. 2016;107:13–18. doi:10.1016/j.resuscitation.2016.07.006 [PubMed: 27456394]
37. Schluep M, Gravesteijn BY, Stolker RJ, Endeman H, Hoeks SE. One-year survival after in-hospital cardiac arrest: A systematic review and meta-analysis. Resuscitation. 2018;132:90–100. doi:10.1016/j.resuscitation.2018.09.001 [PubMed: 30213495]
38. Chan PS, Nallamothu BK, Krumholz HM, et al.; American Heart Association Get with the Guidelines–Resuscitation Investigators. Long-term outcomes in elderly survivors of in-hospital cardiac arrest. N Engl J Med. 2013;368(11):1019–1026. doi:10.1056/NEJMoa1200657 [PubMed: 23484828]
39. Wallmuller C, Meron G, Kurkciyan I, Schober A, Stratil P, Sterz F. Causes of in-hospital cardiac arrest and influence on outcome. Resuscitation. 2012;83(10):1206–1211. doi:10.1016/j.resuscitation.2012.05.001 [PubMed: 22595441]
40. Radeschi G, Mina A, Berta G, et al.; Piedmont IHCA Registry Initiative. Incidence and outcome of in-hospital cardiac arrest in Italy: a multicentre observational study in the Piedmont Region. Resuscitation. 2017;119:48–55. doi:10.1016/j.resuscitation.2017.06.020 [PubMed: 28655621]
41. Legriel S, Bougouin W, Chocron R, et al.; for Paris-SDEC investigators. Early in-hospital management of cardiac arrest from neurological cause: diagnostic pitfalls and treatment issues. Resuscitation. 2018;132:147–155. doi:10.1016/j.resuscitation.2018.08.004 [PubMed: 30086373]
42. Soar J, Nolan JP, Böttiger BW, et al.; Adult advanced life support section Collaborators. European Resuscitation Council Guidelines for Resuscitation 2015: Section 3. Adult advanced life support. Resuscitation. 2015;95:100–147. doi:10.1016/j.resuscitation.2015.07.016 [PubMed: 26477701]
43. Tirkkonen J, Hellevuo H, Olkkola KT, Hoppu S. Aetiology of in-hospital cardiac arrest on general wards. Resuscitation. 2016;107:19–24. doi:10.1016/j.resuscitation.2016.07.007 [PubMed: 27492850]
44. Bergum D, Haugen BO, Nordseth T, Mjølstad OC, Skogvoll E. Recognizing the causes of in-hospital cardiac arrest—a survival benefit. Resuscitation. 2015;97:91–96. doi:10.1016/j.resuscitation.2015.09.395 [PubMed: 26449872]
45. De Bruin ML, Langendijk PN, Koopmans RP, Wilde AA, Leufkens HG, Hoes AW. In-hospital cardiac arrest is associated with use of non-antiarrhythmic QTc-prolonging drugs. Br J Clin Pharmacol. 2007;63(2):216–223. doi:10.1111/j.1365-2125.2006.02722.x [PubMed: 16869820]
46. Overdyk FJ, Dowling O, Marino J, et al. Association of opioids and sedatives with increased risk of in-hospital cardiopulmonary arrest from an administrative database. PLoS One. 2016;11(2):e0150214. doi:10.1371/journal.pone.0150214 [PubMed: 26913753]
47. Larkin GL, Copes WS, Nathanson BH, Kaye W. Pre-resuscitation factors associated with mortality in 49,130 cases of in-hospital cardiac arrest: a report from the National Registry for Cardiopulmonary Resuscitation. Resuscitation. 2010;81(3):302–311. doi:10.1016/j.resuscitation.2009.11.021 [PubMed: 20047786]
48. Andersen LW, Kim WY, Chase M, et al.; American Heart Association's Get With the Guidelines(®) – Resuscitation Investigators. The prevalence and significance of abnormal vital signs prior to in-hospital cardiac arrest. Resuscitation. 2016;98:112–117. doi:10.1016/j.resuscitation.2015.08.016 [PubMed: 26362486]
49. Galhotra S, DeVita MA, Simmons RL, Dew MA; Members of the Medical Emergency Response Improvement Team (MERIT) Committee. Mature rapid response system and potentially avoidable cardiopulmonary arrests in hospital. Qual Saf Health Care. 2007;16(4):260–265. doi:10.1136/qshc.2007.022210 [PubMed: 17693672]

50. Kronick SL, Kurz MC, Lin S, et al. Part 4: Systems of Care and Continuous Quality Improvement: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2015;132(18)(suppl 2):S397–S413. doi:10.1161/CIR.0000000000000258 [PubMed: 26472992]
51. Smith ME, Chiovaro JC, O'Neil M, et al. Early warning system scores for clinical deterioration in hospitalized patients: a systematic review. Ann Am Thorac Soc. 2014;11(9):1454–1465. doi:10.1513/AnnalsATS.201403-102OC [PubMed: 25296111]
52. Maharaj R, Raffaele I, Wendon J. Rapid response systems: a systematic review and meta-analysis. Crit Care. 2015;19:254. doi:10.1186/s13054-015-0973-y [PubMed: 26070457]
53. Hasselqvist-Ax I, Riva G, Herlitz J, et al. Early cardiopulmonary resuscitation in out-of-hospital cardiac arrest. N Engl J Med. 2015;372(24):2307–2315. doi:10.1056/NEJMoa1405796 [PubMed: 26061835]
54. Bircher NG, Chan PS, Xu Y; American Heart Association's Get With The Guidelines-Resuscitation Investigators. Delays in cardiopulmonary resuscitation, defibrillation, and epinephrine administration all decrease survival in in-hospital cardiac arrest. Anesthesiology. 2019;130:414–422. doi:10.1097/ALN.0000000000002563 [PubMed: 30707123]
55. 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. doi:10.1161/CIRCOUTCOMES.111.000041 [PubMed: 23481533]
56. Chan PS, Krumholz HM, Nichol G, Nallamothu BK; American Heart Association National Registry of Cardiopulmonary Resuscitation Investigators. Delayed time to defibrillation after in-hospital cardiac arrest. N Engl J Med. 2008;358(1):9–17. doi:10.1056/NEJMoa0706467 [PubMed: 18172170]
57. Andersen LW, Holmberg MJ, Granfeldt A, et al.; CARES Surveillance Group. Neighborhood characteristics, bystander automated external defibrillator use, and patient outcomes in public out-of-hospital cardiac arrest. Resuscitation. 2018;126:72–79. doi:10.1016/j.resuscitation.2018.02.021 [PubMed: 29477731]
58. Chan PS, Krumholz HM, Spertus JA, et al.; American Heart Association National Registry of Cardiopulmonary Resuscitation (NRCPR) Investigators. Automated external defibrillators and survival after in-hospital cardiac arrest. JAMA. 2010;304(19):2129–2136. doi:10.1001/jama.2010.1576 [PubMed: 21078809]
59. Kudenchuk PJ, Brown SP, Daya M, et al.; Resuscitation Outcomes Consortium Investigators. Amiodarone, lidocaine, or placebo in out-of-hospital cardiac arrest. N Engl J Med. 2016;374(18):1711–1722. doi:10.1056/NEJMoa1514204 [PubMed: 27043165]
60. Perkins GD, Ji C, Deakin CD, et al.; PARAMEDIC2 Collaborators. A randomized trial of epinephrine in out-of-hospital cardiac arrest. N Engl J Med. 2018;379(8):711–721. doi:10.1056/NEJMoa1806842 [PubMed: 30021076]
61. Donnino MW, Salciccioli JD, Howell MD, et al.; American Heart Association's Get With The Guidelines-Resuscitation Investigators. 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. doi:10.1136/bmj.g3028 [PubMed: 24846323]
62. Andersen LW, Kurth T, Chase M, et al.; American Heart Association's Get With The Guidelines-Resuscitation Investigators. Early administration of epinephrine (adrenaline) in patients with cardiac arrest with initial shockable rhythm in hospital: propensity score matched analysis. BMJ. 2016;353:i1577. doi:10.1136/bmj.i1577 [PubMed: 27053638]
63. 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–S464. doi:10.1161/CIR.0000000000000261 [PubMed: 26472995]
64. Jabre P, Penaloza A, Pinero D, et al. Effect of bag-mask ventilation vs endotracheal intubation during cardiopulmonary resuscitation on neurological outcome after out-of-hospital cardiorespiratory arrest: a randomized clinical trial. JAMA. 2018;319(8):779–787. doi:10.1001/jama.2018.0156 [PubMed: 29486039]
65. Wang HE, Schmicker RH, Daya MR, et al. Effect of a strategy of initial laryngeal tube insertion vs endotracheal intubation on 72-hour survival in adults with out-of-hospital cardiac arrest: a

randomized clinical trial. JAMA. 2018;320(8):769–778. doi:10.1001/jama.2018.7044 [PubMed: 30167699]

66. 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. doi:10.1001/jama.2018.11597 [PubMed: 30167701]
67. Andersen LW, Granfeldt A, Callaway CW, et al.; American Heart Association's Get With The Guidelines–Resuscitation Investigators. Association between tracheal intubation during adult in-hospital cardiac arrest and survival. JAMA. 2017;317(5):494–506. doi:10.1001/jama.2016.20165 [PubMed: 28118660]
68. Holmberg MJ, Geri G, Wiberg S, et al.; International Liaison Committee on Resuscitation's (ILCOR) Advanced Life Support and Pediatric Task Forces. Extracorporeal cardiopulmonary resuscitation for cardiac arrest: A systematic review. Resuscitation. 2018;131:91–100. doi: 10.1016/j.resuscitation.2018.07.029 [PubMed: 30063963]
69. Soar J, Callaway CW, Aibiki M, et al.; Advanced Life Support Chapter Collaborators. Part 4: Advanced life support: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation. 2015;95:e71–e120. doi:10.1016/j.resuscitation.2015.07.042 [PubMed: 26477429]
70. 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. doi:10.1161/CIR.0000000000000262 [PubMed: 26472996]
71. Chan PS, Berg RA, Tang Y, Curtis LH, Spertus JA; American Heart Association's Get With the Guidelines–Resuscitation Investigators. Association between therapeutic hypothermia and survival after in-hospital cardiac arrest. JAMA. 2016;316(13):1375–1382. doi:10.1001/jama.2016.14380 [PubMed: 27701659]
72. Berg KM, Grossestreuer AV, Uber A, Patel PV, Donnino MW. Intubation is not a marker for coma after in-hospital cardiac arrest: a retrospective study. Resuscitation. 2017;119:18–20. doi:10.1016/j.resuscitation.2017.07.024 [PubMed: 28750886]
73. Roberts BW, Kilgannon JH, Hunter BR, et al. Association between early hyperoxia exposure after resuscitation from cardiac arrest and neurological disability: prospective multicenter protocol-directed cohort study. Circulation. 2018;137(20):2114–2124. doi:10.1161/CIRCULATIONAHA.117.032054 [PubMed: 29437118]
74. Roberts BW, Kilgannon JH, Chansky ME, Mittal N, Wooden J, Trzeciak S. Association between postresuscitation partial pressure of arterial carbon dioxide and neurological outcome in patients with Post-cardiac arrest syndrome. Circulation. 2013;127(21):2107–2113. doi:10.1161/CIRCULATIONAHA.112.000168 [PubMed: 23613256]
75. Beitler JR, Ghafouri TB, Jinadasa SP, et al. Favorable neurocognitive outcome with low tidal volume ventilation after cardiac arrest. Am J Respir Crit Care Med. 2017;195(9):1198–1206. doi: 10.1164/rccm.201609-1771OC [PubMed: 28267376]
76. Moskowitz A, Grossestreuer AV, Berg KM, et al.; Center for Resuscitation Science. The association between tidal volume and neurological outcome following in-hospital cardiac arrest. Resuscitation. 2018;124:106–111. doi:10.1016/j.resuscitation.2017.12.031 [PubMed: 29292026]
77. Laver S, Farrow C, Turner D, Nolan J. Mode of death after admission to an intensive care unit following cardiac arrest. Intensive Care Med. 2004;30(11):2126–2128. doi:10.1007/s00134-004-2425-z [PubMed: 15365608]
78. Witten L, Gardner R, Holmberg MJ, et al. Reasons for death in patients successfully resuscitated from out-of-hospital and in-hospital cardiac arrest. Resuscitation. 2019;136:93–99. doi:10.1016/j.resuscitation.2019.01.031 [PubMed: 30710595]
79. Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. N Engl J Med. 2002;346(8):557–563. doi:10.1056/NEJMoa003289 [PubMed: 11856794]
80. Bro-Jeppesen J, Annborn M, Hassager C, et al.; TTM Investigators. Hemodynamics and vasopressor support during targeted temperature management at 33°C versus 36°C after out-of-

hospital cardiac arrest: a post hoc study of the target temperature management trial. Crit Care Med. 2015;43(2):318–327. doi:10.1097/CCM.0000000000000691 [PubMed: 25365723]

81. Annborn M, Bro-Jeppesen J, Nielsen N, et al.; TTM-trial investigators. The association of targeted temperature management at 33 and 36 °C with outcome in patients with moderate shock on admission after out-of-hospital cardiac arrest: a post hoc analysis of the Target Temperature Management trial. Intensive Care Med. 2014;40(9):1210–1219. doi:10.1007/s00134-014-3375-8 [PubMed: 25001475]
82. Itenov TS, Johansen ME, Bestle M, et al.; Cooling and Surviving Septic Shock (CASS) Trial Collaboration. Induced hypothermia in patients with septic shock and respiratory failure (CASS): a randomised, controlled, open-label trial. Lancet Respir Med. 2018;6(3):183–192. doi:10.1016/S2213-2600(18)30004-3 [PubMed: 29325753]
83. Mourvillier B, Tubach F, van de Beek D, et al. Induced hypothermia in severe bacterial meningitis: a randomized clinical trial. JAMA. 2013;310(20):2174–2183. doi:10.1001/jama.2013.280506 [PubMed: 24105303]
84. Chan PS, Spertus JA, Krumholz HM, et al.; Get With the Guidelines-Resuscitation Registry Investigators. A validated prediction tool for initial survivors of in-hospital cardiac arrest. Arch Intern Med. 2012;172(12):947–953. doi:10.1001/archinternmed.2012.2050 [PubMed: 22641228]
85. Harrison DA, Patel K, Nixon E, et al.; National Cardiac Arrest Audit. Development and validation of risk models to predict outcomes following in-hospital cardiac arrest attended by a hospital-based resuscitation team. Resuscitation. 2014;85(8):993–1000. doi:10.1016/j.resuscitation.2014.05.004 [PubMed: 24830872]
86. Topjian AA, Localio AR, Berg RA, et al.; American Heart Association National Registry of Cardiopulmonary Resuscitation Investigators. Women of child-bearing age have better inhospital cardiac arrest survival outcomes than do equal-aged men. Crit Care Med. 2010;38(5):1254–1260. doi:10.1097/CCM.0b013e3181d8ca43 [PubMed: 20228684]
87. Joseph L, Chan PS, Bradley SM, et al.; American Heart Association Get With the Guidelines–Resuscitation Investigators. Temporal changes in the racial gap in survival after in-hospital cardiac arrest. JAMA Cardiol. 2017;2(9):976–984. doi:10.1001/jamacardio.2017.2403 [PubMed: 28793138]
88. Galea S, Blaney S, Nandi A, et al. Explaining racial disparities in incidence of and survival from out-of-hospital cardiac arrest. Am J Epidemiol. 2007;166(5):534–543. doi:10.1093/aje/kwm102 [PubMed: 17584756]
89. Ebell MH, Afonso AM. Pre-arrest predictors of failure to survive after in-hospital cardiopulmonary resuscitation: a meta-analysis. Fam Pract. 2011;28(5):505–515. doi:10.1093/fampra/cmr023 [PubMed: 21596693]
90. Ebell MH. Prearrest predictors of survival following in-hospital cardiopulmonary resuscitation: a meta-analysis. J Fam Pract. 1992;34(5):551–558. [PubMed: 1533659]
91. Brady WJ, Gurka KK, Mehring B, Peberdy MA, O'Connor RE; American Heart Association's Get with the Guidelines (formerly, NRCPR) Investigators. In-hospital cardiac arrest: impact of monitoring and witnessed event on patient survival and neurologic status at hospital discharge. Resuscitation. 2011;82(7):845–852. doi:10.1016/j.resuscitation.2011.02.028 [PubMed: 21454008]
92. Nadkarni VM, Larkin GL, Peberdy MA, et al.; National Registry of Cardiopulmonary Resuscitation Investigators. First documented rhythm and clinical outcome from in-hospital cardiac arrest among children and adults. JAMA. 2006;295(1):50–57. doi:10.1001/jama.295.1.50 [PubMed: 16391216]
93. Rohlin O, Taeri T, Netzereab S, Ullemark E, Djärv T. Duration of CPR and impact on 30-day survival after ROSC for in-hospital cardiac arrest: a Swedish cohort study. Resuscitation. 2018;132:1–5. doi:10.1016/j.resuscitation.2018.08.017 [PubMed: 30138650]
94. Mancini ME, Diekema DS, Hoadley TA, et al. Part 3: Ethical Issues: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2015;132(18)(suppl 2):S383–S396. doi:10.1161/CIR.0000000000000254 [PubMed: 26472991]
95. Bossaert LL, Perkins GD, Askitopoulou H, et al.; ethics of resuscitation and end-of-life decisions section Collaborators. European Resuscitation Council Guidelines for Resuscitation 2015: Section

11. The ethics of resuscitation and end-of-life decisions. Resuscitation. 2015;95:302–311. doi: 10.1016/j.resuscitation.2015.07.033 [PubMed: 26477419]

96. Goldberger ZD, Chan PS, Berg RA, et al.; American Heart Association Get With The Guidelines—Resuscitation (formerly National Registry of Cardiopulmonary Resuscitation) Investigators. Duration of resuscitation efforts and survival after in-hospital cardiac arrest: an observational study. Lancet. 2012;380(9852):1473–1481. doi:10.1016/S0140-6736(12)60862-9 [PubMed: 22958912]

97. Wu C, Zheng Z, Jiang L, et al. The predictive value of bedside ultrasound to restore spontaneous circulation in patients with pulseless electrical activity: a systematic review and meta-analysis. PLoS One. 2018;13(1):e0191636. doi:10.1371/journal.pone.0191636 [PubMed: 29364925]

98. Hu K, Gupta N, Teran F, Saul T, Nelson BP, Andrus P. Variability in interpretation of cardiac standstill among physician sonographers. Ann Emerg Med. 2018;71(2):193–198. doi:10.1016/j.annemergmed.2017.07.476 [PubMed: 28870394]

99. Paiva EF, Paxton JH, O'Neil BJ. The use of end-tidal carbon dioxide ($ETCO_2$) measurement to guide management of cardiac arrest: a systematic review. Resuscitation. 2018;123:1–7. doi: 10.1016/j.resuscitation.2017.12.003 [PubMed: 29217394]

100. Wang CH, Chang WT, Huang CH, et al. Validation of the Cardiac Arrest Survival Postresuscitation In-hospital (CASPRI) score in an East Asian population. PLoS One. 2018;13(8):e0202938. doi:10.1371/journal.pone.0202938 [PubMed: 30138383]

101. Paul M, Bougouin W, Geri G, et al. Delayed awakening after cardiac arrest: prevalence and risk factors in the Parisian registry. Intensive Care Med. 2016;42(7):1128–1136. doi:10.1007/s00134-016-4349-9 [PubMed: 27098348]

102. Velly L, Perlbarg V, Boulier T, et al.; MRI-COMA Investigators. Use of brain diffusion tensor imaging for the prediction of long-term neurological outcomes in patients after cardiac arrest: a multicentre, international, prospective, observational, cohort study. Lancet Neurol 2018;17(4):317–326. doi:10.1016/S1474-4422(18)30027-9 [PubMed: 29500154]

103. Sandroni C, D'Arrigo S, Nolan JP. Prognostication after cardiac arrest. Crit Care. 2018;22(1):150. doi:10.1186/s13054-018-2060-7 [PubMed: 29871657]

104. Youn CS, Callaway CW, Rittenberger JC; Post Cardiac Arrest Service. Combination of initial neurologic examination and continuous EEG to predict survival after cardiac arrest. Resuscitation. 2015;94:73–79. doi:10.1016/j.resuscitation.2015.06.016 [PubMed: 26164682]

105. Youn CS, Callaway CW, Rittenberger JC; Post Cardiac Arrest Service. Combination of initial neurologic examination, quantitative brain imaging and electroencephalography to predict outcome after cardiac arrest. Resuscitation. 2017;110:120–125. doi:10.1016/j.resuscitation.2016.10.024 [PubMed: 27840004]

106. Coppler PJ, Elmer J, Calderon L, et al.; Post cardiac arrest Service. Validation of the Pittsburgh Cardiac Arrest Category illness severity score. Resuscitation. 2015;89:86–92. doi:10.1016/j.resuscitation.2015.01.020 [PubMed: 25636896]

107. Get With the Guidelines-Resuscitation Registry. Resuscitation Fact Sheet. American Heart Association website 2014 http://www.heart.org/idc/groups/heart-public/@private/@wcm/@hcm/@gwtg/documents/downloadable/ucm_434082.pdf. Accessed February 25, 2019.

108. Starks MA, Dai D, Nichol G, et al.; American Heart Association's Get With the Guidelines-Resuscitation Investigators. The association of duration of participation in get with the guidelines-resuscitation with quality of care for in-hospital cardiac arrest. Am Heart J. 2018;204:156–162. doi:10.1016/j.ahj.2018.04.018 [PubMed: 30121017]

109. Bradley SM, Huszti E, Warren SA, Merchant RM, Sayre MR, Nichol G. Duration of hospital participation in Get With the Guidelines-Resuscitation and survival of in-hospital cardiac arrest. Resuscitation. 2012;83(11):1349–1357. doi:10.1016/j.resuscitation.2012.03.014 [PubMed: 22429975]

110. Anderson ML, Nichol G, Dai D, et al.; American Heart Association's Get With the Guidelines–Resuscitation Investigators. Association between hospital process composite performance and patient outcomes after in-hospital cardiac arrest care. JAMA Cardiol. 2016;1(1):37–45. doi: 10.1001/jamacardio.2015.0275 [PubMed: 27437652]

Box. Potential Reversible Causes of Cardiac Arrest

H's
Hypokalemia/hyperkalemia[a]
Hypothermia
Hypovolemia
Hypoxia
t's
Tamponade
Tension pneumothorax
Thrombosis (coronary or pulmonary)
Toxins

[a]Can include other metabolic alterations such as severe acidosis.

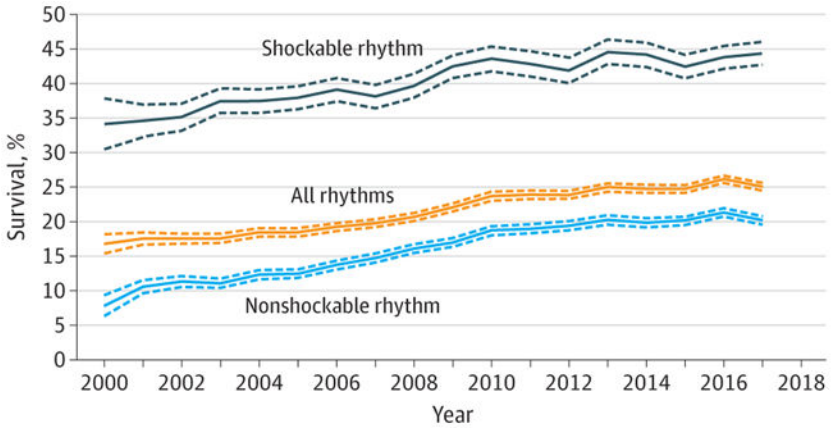

Figure 1. Survival After In-Hospital Cardiac Arrest, 2000 to 2017

Based on data from the Get With The Guidelines-Resuscitation registry on all adult in-hospital cardiac arrests from 2000 to 2017 The dotted lines represent 95% CIs. Shockable rhythms include ventricular fibrillation and pulseless ventricular tachycardia. Nonshockable rhythms include asystole and pulseless electrical activity. Adapted from Benjamin et al.[2]

Determination of the CASPRI Score

For this cardiac arrest risk score, points for each variable are determined, and a summary score is obtained.

1. Age group, y	Points
<50	0
50-59	0
60-69	1
70-79	2
≥80	4

2. Initial arrest rhythm; VF/VT time to defibrillation	Points
≤2 minutes	0
3 minutes	0
4-5 minutes	2
>5 minutes	3
Pulseless electrical activity	6
Asystole	7

3. Prearrest CPC score	Points
1	0
2	2
3	9
≥4	9

4. Hospital location	Points
Telemetry unit	0
Intensive care	1
Nonmonitored unit	3

5. Duration of resuscitation, min	Points
<2	0
2-4	0
5-9	3
10-14	5
15-19	6
20-24	6
25-29	6
≥30	8

Factors present prior to arrest	Points
6. Mechanical ventilation	3
7. Renal insufficiency	2
8. Hepatic insufficiency	4
9. Sepsis	3
10. Malignant disease	4
11. Hypotension	3

Figure 2. The Cardiac Arrest Survival Postresuscitation In-hospital (CASPRI) Score

Reprinted from Chan et al,[84] where a detailed description of the score's interpretation is presented. Scores of 0-4 are associated with 83% survival, 15-19 are associated with 23% survival, and 30-34 are associated with 2% survival. CPC indicates cerebral performance score; VF/VT, ventricular fibrillation or ventricular tachycardia.

Table 1.

Comparison of Out-of-Hospital and In-Hospital Cardiac Arrest

	In-Hospital Cardiac arrest	**Out-of-Hospital Cardiac Arrest**
Incidence	290 000 per year in the United States	350 000 per year in the United States[a]
Patient characteristics	Mean age: 66 y Approximately 60% men	Median age: 65 y Approximately 60% men
Presenting rhythm	Often nonshockable (approximately 80%)	Often nonshockable (approximately 80%)
Cause	Primarily cardiac and respiratory	Primarily cardiac
Prevention	Potentially possible with recognition of deterioration and early intervention	Often impossible given the lack of pre-cardiac arrest monitoring
Timing of basic life support	Often instantaneously	Variable depending on bystander involvement
Timing of advanced life support drugs	Within 5 to 10 min	On average, approximately 20 min after the onset of cardiac arrest
Airway management	Approximately one-third of patients already intubated (eg, intensive care unit patients); often performed by physicians	Often performed by clinicians (eg, paramedics) with variable experience in advanced airway management
Drugs	Limited evidence; epinephrine and amiodarone recommended	Some evidence; epinephrine and amiodarone recommended
Post-cardiac arrest treatment	Limited evidence; supportive care and targeted temperature management recommended	Some evidence; supportive care and targeted temperature management recommended
Prognostication	Limited evidence; focuses on both neurological status and organ failure	Some evidence; focuses on neurological status
Survival to discharge	Approximately 25%	10% to 12%

[a]Assessed by emergency medical services but not necessarily treated.[2]

Table 2.

Randomized Trials Including In-Hospital Cardiac Arrest Patients Between 1988 and 2018

Study	Country	Sites	Years of Inclusion	Main Inclusion Criteria	Main Exclusion Criteria	Patients	Intervention	Control	Main Findings
Brain Resuscitation Clinical Trial II Study Group,[6] 1991[a]	Multiple	24	1984-1989	IHCA, age ≥12 y, ROSC, comatose	Terminal illness, central nervous system disease, atrial fibrillation, women of childbearing age	184	Lidoflazine (calcium channel blocker)	Placebo	No difference in survival at 6 mo (12% vs 15%)
Stiell et al,[7] 1992[a]	Canada	2	1989-1992	IHCA, age ≥16 y	Terminal illness, acute trauma, in the operating or recovery rooms of hospitals	315	High-dose epinephrine (7 mg; max 5 doses)	Standard-dose epinephrine (1 mg)	No difference in ROSC (25% vs 31%) or survival to hospital discharge (5% vs 7%)
Sack et al,[8] 1992	United States	1	1989-1890	IHCA, age ≥18 y	Traumatic or respiratory cause, pregnancy, abdominal aortic aneurysm	103	Interposed abdominal counterpulsation CPR	Standard CPR	Higher ROSC (60% vs 25%), survival to hospital discharge (25% vs 7%), and survival to hospital discharge neurologically intact (17% vs 6%) in the intervention group
Sack et al,[9] 1992	United States	1	1990-1991	IHCA, age ≥18 y, initial nonshockable rhythm	Traumatic or respiratory cause, pregnancy, abdominal aortic aneurysm, recent abdominal surgery, prolonged intubation	143	Interposed abdominal counterpulsation CPR	Standard CPR	Higher ROSC (49% vs 28%) and survival at 24 h (33% vs 13%) in the intervention group. Few survived to hospital discharge
Cohen et al,[10] 1993	United States	1	1992-1993	IHCA, age ≥18 y, witnessed cardiac arrest	Traumatic cause, inability to achieve endotracheal intubation within 15 min	62	Active compression-decompression CPR	Standard CPR	Higher ROSC (62% vs 30%) and survival at 24 h (45% vs 9%) in the intervention group. Few survived to hospital discharge
Lipman et al,[11] 1993	South Africa	1	1990-1991	IHCA in the ICU, age ≥18 y, witnessed cardiac arrest, asystole	NA	37	High-dose epinephrine (10 mg)	Standard-dose epinephrine (1 mg)	No difference in ROSC (68% vs 66%) or survival at 24 h (21% vs 31%). Few

Study	Country	Sites	Years of Inclusion	Main Inclusion Criteria	Main Exclusion Criteria	Patients	Intervention	Control	Main Findings
									patients survived to hospital discharge
Tucker et al,[12] 1994	United States	1	1992-1993	IHCA, age ≥18 y	Traumatic or respiratory cause, pregnancy	53	Active compression-decompression CPR	Standard CPR	Higher ROSC (60% vs 32%) and survival at 24 h (48% vs 21%) in the intervention group. No difference in survival to hospital discharge (24% vs 11%) or neurologically intact at hospital discharge (20% vs 11%)
Woodhouse et al,[13] 1995	Australia	1	1989-1992	IHCA or OHCA with ongoing CPR in the ED	Noncardiac cause	194 (109 with IHCA)	Epinephrine (10 mg; max 2 doses)	Placebo (max 2 doses)	No difference in ROSC (10% vs 7%). No patients survived to hospital discharge
Patrick et al,[14] 1995	Canada	1	1990-1992	Witnessed IHCA or OHCA with ongoing CPR in the ED	Traumatic cause, severe hypothermia/hemorrhage	145 (91 with IHCA)	Methoxamine (40 mg per dose)	Epinephrine (2 mg per dose)	No difference in ROSC (42% vs 53%), 24-h survival (30% vs 37%), or survival to hospital discharge (12% vs 15%)
Stiell et al,[15] 1996[a]	Canada	5	1993-1995	IHCA, age ≥16 y	Terminal illness; acute trauma or exsanguination; recent sternotomy; arrest in operating, recovery, or delivery room	773	Active compression-decompression CPR	Standard CPR	No difference in survival at 1 h (35% vs 35%) or at hospital discharge (10% vs 11%)
Thel et al,[16] 1997	United States	1	1993-1996	IHCA, age ≥18 y	Arrest in emergency, operating, or recovery room; clinical indication for magnesium; signs of irreversible death	156	Magnesium (2 g bolus + 8 g infusion over 24 h)	Placebo	No difference in ROSC (54% vs 60%), survival at 24 h (43% vs 50%), or at hospital discharge (21% vs 21%)
Stiell et al,[17] 2001	Canada	3	1997-1998	IHCA, age ≥16 y	Terminal illness; traumatic injury prior to admission; arrest due to exsanguination; arrest in operating, recovery, or delivery room	200	Vasopressin (40 IU; max 1 dose)	Epinephrine (1 mg)	No difference in survival at 1 h (39% vs 35%), 24 h (26% vs 24%), hospital discharge (12%

Study	Country	Sites	Years of Inclusion	Main Inclusion Criteria	Main Exclusion Criteria	Patients	Intervention	Control	Main Findings
									vs 14%), or 30 d (13% vs 14%)
Mentzelopoulos et al,[18] 2009	Greece	1	2006-2007	IHCA, age ≥18 y	Terminal illness, exsanguination, treatment with IV corticosteroids prior to the cardiac arrest	100	Vasopressin (20 IU; max 5 doses) + methylprednisolone (40 mg) + hydrocortisone (300 mg/ 24 h) if in shock after cardiac arrest	Placebo	Higher ROSC (81% vs 52%) and survival to hospital discharge (19% vs 4%) in the intervention group
Weidman et al, [19] 2010	United States	1	2007-2008	IHCA, age ≥18 y	Arrest in emergency or operating room	98	Immersive simulation training of residents	Standard training	No difference in CPR quality between groups
Pittl et al,[20] 2013	Germany	1	2008-2009	IHCA or OHCA, age ≥18 y, ROSC, cardiac origin, comatose	Pregnancy, coagulation disorder, cardiogenic shock, terminal illness	80[b]	Invasive cooling	Surface cooling	No difference in survival to hospital discharge (62% vs 54%) or with a good neurological outcome (36% vs 36%). More bleeding complications with invasive cooling
Mentzelopoulos et al,[21] 2013	Greece	3	2008-2010	IHCA, age ≥18 y	Terminal illness, exsanguination, treatment with intravenous corticosteroids prior to the cardiac arrest	268	Vasopressin (20 IU; max 5 doses) + methylprednisolone (40 mg) + hydrocortisone (300 mg/ 24 h) if in shock after cardiac arrest	Placebo	Higher ROSC (84% vs 66%) and survival to hospital discharge with a good neurological outcome (14% vs 5%) in the intervention group
Vahedian-Azimi et al,[22] 2016	Iran	4	2014	IHCA in the ICU, age ≥18 y	NA	83	CPR with feedback devise	Standard CPR	Higher CPR quality and ROSC (72% vs 35%) in the intervention group
Eastwood et al, [23] 2016	Australia and New Zealand	4	2012-2014	IHCA or OHCA, age ≥18 y, mechanically ventilated	Traumatic cause, pregnancy, imminent death, raised intracranial pressure/ bleeding, severe chronic airflow limitation, severe metabolic acidosis	86 (16 with IHCA	Mild hypercapnia after cardiac arrest ($PaCO_2$ 50-55 mm Hg) for 24 h	Normocapnia after cardiac arrest ($PaCO_2$ 35-45 mm Hg) for 24 h	Lower NSE values in the intervention group. No difference in survival to hospital discharge (74% vs 63%) and good neurological

Study	Country	Sites	Years of Inclusion	Main Inclusion Criteria	Main Exclusion Criteria	Patients	Intervention	Control	Main Findings
									outcome at 6 mo (59% vs 46%)
Movahedi et al,[24] 2016	Iran	1	2014	IHCA, endotracheal intubation, age 18-85 y, arrest in hospital ward or ED	Traumatic cause, pregnancy, abdominal surgery in the past 2 weeks, history of abdominal aortic aneurysm, coagulopathy, ascites, active gastrointestinal bleeding, pulmonary embolism	83	Interposed abdominal counterpulsation CPR	Standard CPR	Higher end-tidal carbon dioxide in the intervention group. No difference in ROSC (60% vs 53%) or survival at 24 h (38% vs 38%)
Anantharaman et al,[25] 2017	Singapore	4	2005-2008	IHCA or OHCA with ongoing CPR in the ED, age ≥21 y, initial shockable rhythm	Traumatic cause, pregnancy	235 (89 with IHCA)	Escalating high-energy shocks (200-300-360 J)	Low-energy shocks (150-150 - 150 J)	No difference in first shock success (67% vs 64%), ROSC (55% vs 55%), or 30-d survival (21% vs 28%)
Koster et al,[26] 2017[a]	The Netherlands	1	2006-2014	IHCA, age ≥18 y	Traumatic cause	199	Mechanical chest compressions (AutoPulse or LUCAS)	Manuel chest compressions	No difference in serious or life-threatening visceral damage (9% vs 8%)
Zhang et al,[27] 2017	China	50	2012-2015	IHCA, age ≥18 y	Pregnancy, malignancy, HIV, arrest caused by brain/liver/lung disease, end-stage heart disease, Shenfu (a traditional Chinese medication including ginseng and aconite) allergy	978	Shenfu (200 mL/d for 14 d)	Standard care	Higher 28-d (43% vs 30%) and 90-d survival (40% vs 26%) in the intervention group
Look et al[28] 2018	Singapore	1	2006-2014	IHCA or OHCA, age 18-80 y	Traumatic cause, hemodynamically unstable, pregnancy, poor premorbid status	45 (7 with IHCA)	Invasive cooling	Surface cooling	No difference in 30-d survival (48% vs 32%) or good neurological outcome (30% vs 23%)

Abbreviations: CPR, cardiopulmonary resuscitation; ED, emergency department; IHCA, in-hospital cardiac arrest; ICU, intensive care unit; NA, not applicable; NSE: neuron-specific enolase; OHCA, out-of-hospital cardiac arrest; $PaCO_2$, partial pressure of carbon dioxide; ROSC, return of spontaneous circulation.

[a] The trial also included patients with OHCA, but those patients are not included in this table.

[b] Number of IHCAs not reported.