# Strategies for Improving Survival After In-Hospital Cardiac Arrest in the United States: 2013 Consensus Recommendations : A Consensus Statement From the American Heart Association

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on behalf of the 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 Peripheral Vascular Disease

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# Strategies for Improving Survival After In-Hospital Cardiac Arrest in the United States: 2013 Consensus Recommendations

## A Consensus Statement From the American Heart Association

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The goal of this statement is to develop consensus recommendations aimed at measuring and optimizing outcomes after in-hospital cardiac arrest (IHCA). For the purposes of this statement, IHCA is defined as a cardiac arrest that occurs in a hospital (whether the patient is admitted or not) and for which resuscitation is attempted with chest compressions, defibrillation, or both.

### IHCA Consensus Process

Members of the writing group were selected for their expertise in cardiac resuscitation and post–cardiac arrest care. Monthly telephone conferences and "webinars" over a 10-month period were used to define the scope of the statement and to assign writing teams for each section. The first draft of each section was discussed and sent to the chair to be compiled into a single document. Revised versions were then sent to all writing group members until consensus was achieved. The final draft underwent 3 sets of independent peer review before publication.

### IHCA Conflict of Interest or Relationships With Industry

The American Heart Association (AHA) is committed to the highest ethical standards. The AHA believes that having experts who have a relationship with industry or other relevant relationships on writing groups can strengthen the writing group effort when these relationships are transparent and managed.

The AHA conflict of interest policy requires each member to declare relevant and current conflicts of interest. The chair may not have any relationship with industry relevant to the topic. The majority of writing group members (defined as >50% +1) must be free of relevant relationships with industry. Every writing group member agrees to maintain his or her current status with respect to relationships with industry throughout the development of the manuscript to publication. In addition, each member formally declares his or her conflict of interest or relationship with industry at the time of publication. All members of this writing group were compliant with this policy ("Writing Group Disclosures").

### Brief Overview

IHCA has not received the same level of focused research as out-of-hospital cardiac arrest (OHCA). There are many gaps in science, policy, and institutional application and accountability for the care of these patients. There is variation across hospitals, regions, and nations in how IHCAs are defined and

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This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on January 3, 2013. A copy of the document is available at http://my.americanheart.org/statements by selecting either the "By Topic" link or the "By Publication Date" link. To purchase additional reprints, call 843-216-2533 or e-mail kelle.ramsay@wolterskluwer.com.

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counted and whether they are reported annually as an accreditation requirement or a metric of hospital performance. This scientific statement is organized into the following 4 sections to provide consensus recommendations based on scientific evidence from IHCA studies or reasonable extrapolation from the OHCA literature:

1. Epidemiology (incidence and outcome)
2. Best practices (institutional infrastructure, care pathways, and process of care for each time interval [prearrest, intra-arrest, and postarrest])
3. Appeal for a culture change and standardized reporting and benchmarking
4. Conclusions and recommendations

This consensus statement on IHCA provides healthcare providers, clinical leaders, administrators, regulators, and policy makers with an overview of the various issues related to reporting, planning, and performing best practices as they relate to IHCA.

## Epidemiology

Without a comparable data set composed of uniform definitions and reliable data abstraction across hospitals, it is challenging to identify interventions that are effective and safe. It is also difficult to count and report the incidence and outcomes of IHCA without a standardized method of defining the denominator, which has led to confusion in the literature and affects the generalizability of study results. More important, there is a common assumption that scientific advances in OHCA are directly applicable to the epidemiology and treatment of IHCA, with no consideration given to the different causes and burden of comorbidities that contribute to IHCA epidemiology.[1] This assumption may be flawed, but current guidelines lump the literature together to guide resuscitation.[2]

In most institutions, counting IHCAs is challenging. One method is to count the number of times the hospital's emergency response team is activated. This may be a flawed measure, because it can overcount (by including nonarrests) or undercount (by missing arrests in which victims were resuscitated by local staff without activation of the emergency response team, or missing arrests that occur in the emergency department [ED], operating rooms, cardiac procedure suites, and sometimes intensive care units [ICUs]).

The incidence of IHCA is not just a measure of the burden of illness; it is also a measure of the institutional response and system of care in the prevention of IHCA. Whereas IHCA outcomes may be a more refined measure of institutional readiness and effectiveness in the treatment of IHCA. The Joint Commission[3] requires a common standard of care across the inpatient and contiguous outpatient areas of the hospital, yet in practice, variability may exist in the institutional response based on the geography of the event (Table 1). Because all arrests that occur within the confines of a hospital test that hospital's response and system of care, a strategy should be in place to ensure comprehensive monitoring and institutional reporting of outcomes for arrests in patients, employees, and visitors in all areas, including the ED, diagnostic services, surgical suites, long-term care, and employee areas.

Hospitals that provide care for both acute and long-term patients may not consistently include or separate these patients when reporting incidence. Long-term care facilities and specialized facilities (eg, psychiatric care) may be physically located within a hospital but operate under a separate license. Another important issue that must be addressed to ensure consistent reporting of institutional IHCA is how to count multiple arrests in the same patient during the same admission; each arrest in the same patient may be counted differently across institutions.

Finally, institutional variation in implementation of do-not-attempt-resuscitation (DNAR) orders for patients before or after IHCA and how DNAR patients are counted may skew reported incidence and survival rates.[4] Hospitals that frequently implement DNAR orders before IHCA may report lower incidences and higher survival rates than hospitals that infrequently implement DNAR orders. The institutional rate of survival will be dramatically affected if the institutional practice is to declare most patients DNAR after IHCA or to withdraw life-sustaining therapy. By one estimate from a registry of 207 hospitals, as many as 63% of patients with IHCA who achieve return of spontaneous circulation (ROSC) may be declared DNAR, and 44% may have life support withdrawn.[5] In 1 study, there was a significant increase (15%) in the calculated survival-to-discharge rate when patients who were declared DNAR after an initial arrest were excluded.[6] This suggests that DNAR rates can have a significant effect on reported outcome measures, and standard methods that account for the use of DNAR orders before or after IHCA must be implemented.

### Published Estimates of Incidence

Given the lack of consistency in reporting, estimates of incidence and outcome should be reviewed and compared with caution. Single-institution studies using Utstein criteria have reported large variations in hospital-wide incidence rates of adult IHCA, ranging from 3.8 to 13.1 per 1000 admissions.[7,8]

A systematic review and meta-analysis of rapid response systems within 41 hospitals (academic and community) involving >1 million admissions described an incidence of IHCA occurring outside of ICUs of 3.66 per 1000 adult admissions and 1.14 per 1000 pediatric admissions.[9] Because 45% of adult arrests and 65% of pediatric arrests occur in ICUs,[10] by extrapolation, the hospital-wide rate of cardiac arrests is likely to be closer to 6.65 and 3.26 per 1000 admissions for adults and children, respectively. Given the estimated 32.2 million adult admissions and 1.8 million pediatric admissions (Healthcare Cost and Utilization Project data),[11] extrapolation of the rapid response team data[9] yields ≈200 000 adult cardiac arrests and ≈6000 pediatric cardiac arrests in the United States each year (Table 2). The adult estimate was confirmed by a recently published extrapolation that used data from 150 hospitals participating in the Get With The Guidelines–Resuscitation registry.[12] This volunteer registry, funded by the AHA, was formerly known as the National Registry for Cardiopulmonary Resuscitation,

**Table 1. Variability of Institutional Response to In-Hospital Cardiac Arrest**

| Type of Patient/Arrest | Potential Responders | | | |
|---|---|---|---|---|
| OHCA | | | | |
| Arrives alive at ED with pulse | ED staff | | | |
| Arrives with ongoing active resuscitation attempt | ED staff | | | |
| OHCA rearrest in ED | ED staff | | | |
| Outpatient cardiac arrest | | | | |
| ED patient | ED staff | | | |
| ED patient admitted to hospital, waiting for inpatient bed | ED staff | | | |
| Same-day surgery | ED staff | Operating staff | | Cardiac arrest team |
| Diagnostic tests and therapy | ED staff | | | Cardiac arrest team |
| IHCA | | | | |
| Inpatient | ED staff* | | | Cardiac arrest team |
| Operating room | ED staff* | Operating staff | | |
| Critical care unit | ED staff* | | Critical care staff | Cardiac arrest team |
| Recovery room | ED staff* | | Critical care staff | Cardiac arrest team |
| Nonpatient cardiac arrest | | | | |
| Staff with arrest anywhere | ED staff* | | | Cardiac arrest team |
| Visitors with arrest anywhere | ED staff* | | | Cardiac arrest team |

Institutional response may differ across various locations where cardiac arrest occurs. This variability stresses the importance of good tracking of incidence and outcome to know how well the institution is performing in terms of prevention, response, and outcome.

ED indicates emergency department; IHCA, in-hospital cardiac arrest; and OHCA, out-of-hospital cardiac arrest.

*ED staff refers to institutions where the ED provides 24/7 (24 hours a day, 7 days a week) coverage for the hospital. This may occur in hospitals without 24/7 in-house support for critical care units.

or the NRCPR.[13] Remarkably, these estimates are similar to those for emergency medical services–assessed (treated and untreated) OHCAs. On the basis of US census data and available incidence data, it is estimated that each year ≈300 000 adult[14] and 7000 pediatric[15] OHCAs occur.[16]

Published estimates can be affected by rearrest rates as well. Ninety-two percent of admitted patients who have a cardiac arrest have only 1 arrest during the index hospitalization; however, 7% have 2 arrests during the same admission, and, surprisingly, 1% have ≥3 arrests.[5]

## Recommended Definition of Incidence

The incidence of IHCA in admitted patients should be calculated by dividing the total number of patients who receive chest compressions, defibrillation, or both by the number of patients admitted to the hospital. Admitted patients in the

**Table 2. Published Incidence and Outcome Estimates of ICHA (Adult and Pediatric)**

| | Estimate by Admissions | Estimate by Population/Year | Reference |
|---|---|---|---|
| Incidence | | | |
| Adult | 6.65/1000 | 200 000 | Chan et al[9]<br>Nadkarni et al[10] |
| Pediatric | 3.26/1000 | 6000 | Chan et al[9]<br>Nadkarni et al[10] |
| Outcomes | | | |
| Adult | | | |
| Survival to discharge | … | 18% | Nadkarni et al[10] |
| CPC good | … | 73% | Nadkarni et al[10] and Fiser[19]<br>Booth et al[20] |
| Survival at 1 year | … | 6.60% | Booth et al[20] |
| Survival at 3 years | … | 5.20% | Booth et al[20] |
| Pediatric | | | |
| Survival to discharge | … | 27% | Nadkarni et al[10] |
| CPC good | … | 65% | Nadkarni et al[10] and Fiser[19]<br>Booth et al[20] |

CPC indicates cerebral performance categories; and IHCA, in-hospital cardiac arrest.

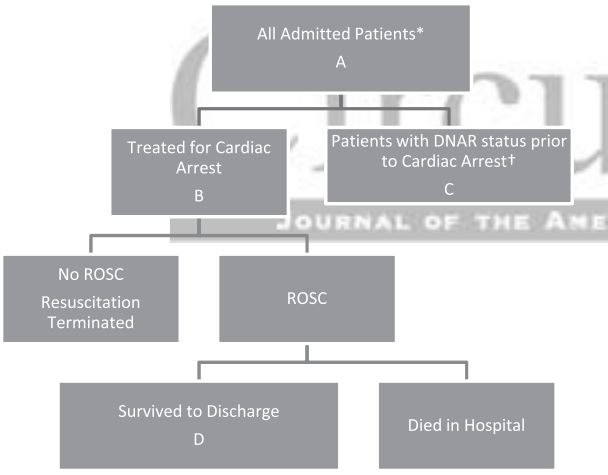
ICU and critical care units, recovery room, and operating room should be counted in the denominator, and the number of patients who experience an arrest in these areas should be included in the numerator. All patients with a DNAR order before the index cardiac event should be excluded. This seems intuitive, because the index event will be the patient's last event; however, the presence of a DNAR order is often missed, which leads to activation of the emergency response team and initiation of resuscitative efforts only for hospital staff to find out about the DNAR order midarrest and then withdraw care. These patients should not be counted in IHCA incidence or outcome measures (Figure 1).

The incidence of IHCA in ED patients should include all patients who were registered in the ED and patients admitted to the hospital but who remained in the ED awaiting a bed before their index IHCA. This group of patients would exclude those with OHCA that occurred before their arrival in the ED and patients with OHCA who experienced another arrest on arrival in the ED. The incidence calculation should exclude all nonadmitted patients with OHCA or cardiac arrest that occurred outside the ED (outpatient settings) who were transferred to the ED after resuscitation while awaiting an in-hospital bed or were admitted directly to a hospital bed, because the true denominator of this type of patient is unknown (Figure 2). The incidence of IHCA among long-term care patients should be reported separately, using their respective denominator in a fashion similar to that shown in Figure 1.

For patients with >1 IHCA during a single hospitalization, only the first IHCA is counted as the index cardiac arrest regardless of how many times the patient rearrests. If a patient has an arrest during >1 admission, then the first cardiac arrest that occurs in each separate admission is counted.

## Published Estimates of Outcome

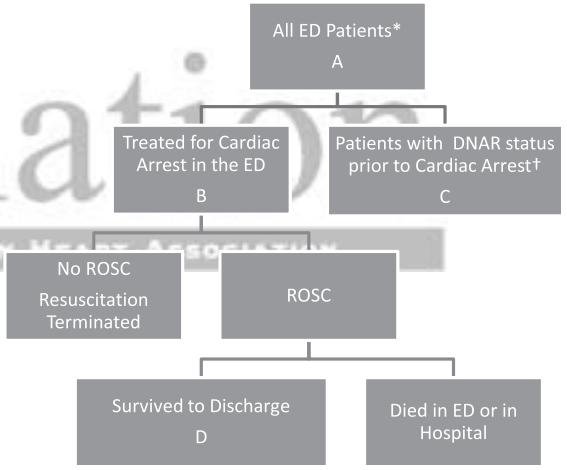
Although survival-to-discharge rates vary between studies, overall survival to hospital discharge has remained essentially unchanged for decades[5] (Figure 3). In a retrospective analysis of the data from ≈1000 US hospitals in the Nationwide Inpatient Sample, survival of post-IHCA patients was determined by use of the *International Statistical Classification of Diseases and Related Health Problems (ICD)-9* code 427.5, "cardiac arrest," to identify patients with IHCA and patients who presented to the ED in cardiac arrest who were eventually admitted to the hospital.[17] The study suggested that there was a 3% increase in in-hospital survival rates among IHCA patients between 2000 and 2004 (Figure 4). In a registry of 36 902 adults (≥18 years of age) and 880 children (<18 years old), survival to discharge after IHCA was higher in children than in adults for all rhythms (27% versus 18%); however, arrests that occurred in the delivery room and the ICU were




Rate Calculations

| | | |
|---|---|---|
| Incidence | Treated for Cardiac Arrest / All admitted Patients | B / A |
| Survival | Survived to Discharge / Treated for Cardiac Arrest | D / B |
| DNAR | Patients with DNAR status prior to Cardiac Arrest / All Admitted Patients | C / A |

**Figure 1.** Reporting of incidence, survival, and do-not-attempt-resuscitation (DNAR) rate for patients admitted to an in-hospital bed with in-hospital cardiac arrest (IHCA). ROSC indicates return of spontaneous circulation. *All admitted patients includes all patients admitted in any in-hospital bed in any location of the hospital, including operating rooms, recovery, critical care units, procedural and diagnostic laboratories, and public areas. †Excludes admitted patients in the emergency department and patients designated DNAR after treatment for IHCA.




Rate Calculations

| | | |
|---|---|---|
| Incidence | Treated for Cardiac Arrest / All admitted Patients | B / A |
| Survival | Survived to Discharge / Treated for Cardiac Arrest | D / B |
| DNAR | Patients with DNAR prior to Cardiac Arrest / All Admitted Patients | C / A |

**Figure 2.** Reporting of incidence, survival, and do-not-attempt-resuscitation (DNAR) rate for patients who have a cardiac arrest in the emergency department (ED). ROSC indicates return of spontaneous circulation. *All ED patients refers to all patients registered in the ED who have an arrest at any time in the ED before moving from the ED to an in-hospital ward. Patients who have had an out-of-hospital cardiac arrest, staff, and visitors are excluded, as are outpatients attending clinics who have an arrest and are transported to the ED for postresuscitation care until they are admitted to the hospital. †Patients with DNAR status before cardiac arrest and who were not treated or treated initially until the DNAR status was verified and resuscitation terminated.

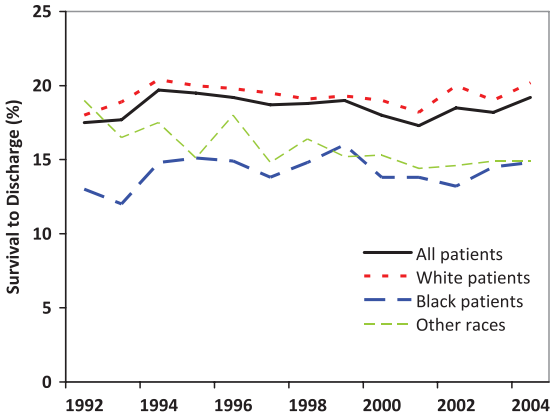



**Figure 3.** Survival to hospital discharge after in-hospital cardiopulmonary resuscitation, according to year and race. Survival rates are poorer for black and other nonwhite patients ($P$<0.001). There is no significant change in overall survival rate from 1992 to 2005 ($P$=0.57 with use of the likelihood-ratio test). From Ehlenbach et al.[23] Copyright © 2009, Massachusetts Medical Society. Reprinted with permission from Massachusetts Medical Society.

excluded in this comparison[10] (Table 3). One study compared short- and long-term survival after IHCA and found 6.6% alive at discharge, 5.2% alive at 1 year, and 3% alive at 3 years.[18] Good short-term neurological outcomes after IHCA, as measured by cerebral performance category, were reported in 64% of children and 75% of adults who survived to discharge[10,19,20] (Table 4).

Survival outcomes across different types of institutions, at different times, and of various subgroups have also been reported. A higher survival rate has been correlated with larger, teaching, and urban hospitals in some studies,[5,17] but others report lower survival rates in metropolitan or teaching hospitals, perhaps related to the severity of underlying illness[18] (Table 5; Figure 5). In addition, survival rates after IHCA have been reported to be lower at night and during weekends[21] (Figure 6). In one report, survival-to-discharge rates after IHCA among critical care patients were 15.9% overall but only 3.9% in patients who received vasopressors before the arrest.[22] In a study of 433 985 elderly patients (≥65 years of age) who underwent in-hospital cardiopulmonary resuscitation (CPR), 18.3% survived to discharge (95% confidence interval, 18.2–18.5). There was no significant change in survival rates over time. Survival rates were lower in this subgroup of elderly patients when they were admitted to a skilled nursing facility and in patients who received care in a metropolitan or teaching hospital, and these findings were attributed to an overrepresentation of patients with more severe illness in these treatment facilities.[23]

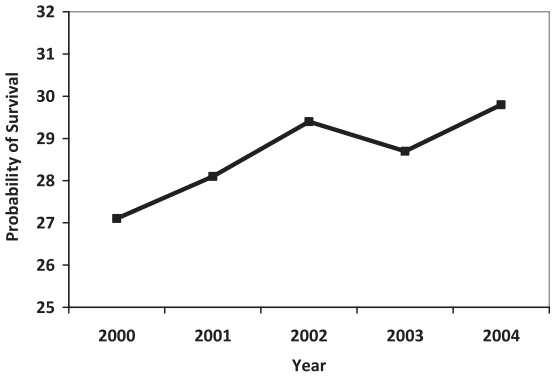



**Figure 4.** US national data and adjusted probability of survival by year. Adjusted for age, sex, hospital location (urban/rural), hospital teaching status, hospital bed size (large, medium, small), median income for patient's zip code, and region of the country in which the hospital is located. Modified from Carr et al[17] and used with kind permission from Springer Science+Business Media. Copyright © 2008, Springer-Verlag.

**Table 3. First Documented Rhythm in Pediatric and Adult Cardiac Arrests**

| First Documented Pulseless Rhythm | Pediatric Cardiac Arrest (n=880) | Adult Cardiac Arrest (n=36 902) | *P* |
|---|---|---|---|
| Asystole | 350 (40) | 13 024 (35) | 0.006 |
| VF or pulseless VT | 120 (14) | 8361 (23) | <0.001 |
| VF | 71 (8) | 5170 (14) | <0.001 |
| Pulseless VT | 49 (6) | 3191 (9) | 0.001 |
| PEA | 213 (24) | 11 963 (32) | <0.001 |
| Unknown by documentation | 197 (22) | 3554 (10) | <0.001 |

PEA indicates pulseless electrical activity; VF, ventricular fibrillation; and VT, ventricular tachycardia.

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## Recommended Definition of Outcome

Survival to hospital discharge is the minimum standard, and survival to 30 days is preferred. In addition, a measure of functional survival (eg, cerebral performance category or Modified Rankin Scale at discharge or at 30 days). Outcomes should be reported for all patients who are admitted to the hospital who do not have a DNAR order before arrest who are treated with either chest compressions or defibrillation (Figure 1). Arrests that occur in the ED should be reported separately and should not include patients whose initial arrest occurred out of hospital or people who were visitors, staff, or outpatients (Figure 2). DNAR rates should be defined by the number of patients with a DNAR status (before an index cardiac arrest) per 1000 admitted patients. The DNAR status of the patient after arrest is not included in the DNAR rate. The DNAR rate of patients before arrest should be reported separately for acute and long-term care inpatients. The rates of DNAR status assignment after arrest should be reported and compared with similar institutions to ensure that performance is in line with the standard of care.

## Best Practices

The best practices are divided into 3 temporal sections: Prearrest, intra-arrest, and postarrest. The discussion for each period includes (1) a brief introduction, (2) the structural aspects of the institutional response (personnel, training, equipment), (3) care pathways followed during the time interval (early identification, focus on CPR and early defibrillation, comprehensive postarrest care), and (4) process issues related to how care is provided and quality improvement measures (real-time feedback, automated equipment that can replace staff and deliver similar care, withdrawal of life-sustaining therapy).

**Table 4. Outcomes of In-Hospital Pulseless Cardiac Arrest by First Documented Pulseless Arrest Rhythm***

| | VF or Pulseless VT | | Asystole | | PEA | | Unknown Rhythm | |
|---|---|---|---|---|---|---|---|---|
| | Pediatric (n=120) | Adult (n=8361) | Pediatric (n=350) | Adult (n=13 024) | Pediatric (n=213) | Adult (n=11 963) | Pediatric (n=197) | Adult (n=3554) |
| Any ROSC | 80 (66.7) | 5629 (67.3) | 184 (52.6) | 5858 (45.0) | 123 (57.7) | 6270 (52.4) | 137 (69.5) | 2062 (58.0) |
| ROSC >20 min | 74 (61.7) | 5185 (62.0) | 157 (44.9) | 4997 (38.4) | 108 (50.7) | 5135 (42.9) | 120 (60.9) | 1866 (52.5) |
| Survival to discharge | 35 (29.2) | 3013 (36.0) | 78 (22.3) | 1379 (10.6) | 57 (26.8) | 1340 (11.2) | 66 (33.5) | 753 (21.2) |
| Neurological outcome | | | | | | | | |
| Good | 22 (62.9) | 2268 (75.3) | 43 (55.1) | 841 (61.0) | 36 (63.2) | 834 (62.2) | 35 (53.0) | 447 (59.4) |
| Poor | 1 (2.9) | 264 (8.8) | 16 (20.5) | 243 (17.6) | 13 (22.8) | 222 (16.6) | 11 (16.7) | 111 (14.7) |
| Unknown | 12 (34.3) | 481 (16.0) | 19 (24.4) | 295 (21.4) | 8 (14.0) | 284 (21.2) | 20 (30.3) | 195 (25.9) |

Values are number (%) of patients.

PEA indicates pulseless electrical activity; ROSC, return of spontaneous circulation; VF, ventricular fibrillation; and VT, ventricular tachycardia.

*First documented pulseless rhythm was defined as the first electrocardiographic rhythm documented at the time the patient became pulseless. Good neurological outcome was prospectively defined as cerebral performance category (CPC) 1 or 2 for adults; the comparable pediatric cerebral performance category (PCPC) of 1, 2, or 3 for children on hospital discharge; or no change from baseline CPC or PCPC.



## Best Practices: Prearrest

### *Brief Introduction to Prearrest*

In the pre-IHCA period, several aspects of preparation are important. These include placement of defibrillators and code carts (or crash carts); establishment of emergency response teams; training of IHCA code team personnel in clinical resuscitation care, as well as team leadership and resource management; and development of a comprehensive performance review process, cardiac monitoring, and documentation in the medical record about the level of resuscitation appropriate for the patient (eg, DNAR status).

### *Structural Aspects of the Institutional Response*

*Defibrillators and Code Carts*

Manual defibrillators or automated external defibrillators (AEDs) and code carts should be readily accessible to any patient area, and all staff should know the location of this equipment and how to use it. In general, a defibrillator and code cart should be in close proximity to enable defibrillation of any patient in cardiac arrest within 2 minutes.[24] This may be achieved by staff carrying resources to the scene of the arrest rather than moving a cart; regardless of how staff access the defibrillator and code cart, it is most important that access be rapid. To minimize delays and confusion, it may be advisable to standardize defibrillator equipment across the institution.[25] Ideally, staff should have the capacity to receive feedback on the quality of CPR at the point of care. This may include voice or visual cues on the quality of CPR (depth, interruptions or hands-off time, compression rate) that are measured and reported by the defibrillator, a handheld device, or alternative technology.[26] Additionally, staff should have access to physiological feedback about the quality of CPR at the point of care (eg, quantitative end-tidal $CO_2$ or waveform capnography defined as continuous noninvasive measurement and graphic display of end-tidal $CO_2$), at a minimum and intra-arterial pressure monitoring as outlined in the *2010 AHA guidelines for CPR and ECC*.[27] There should be a process in place to

**Table 5. Severity of Disease Predictors for Nonsurvivors and Survivors of Cardiac Arrest**

| Variable | Nonsurvivors (n=683) | Survivors (n=49) | *P** |
|---|---|---|---|
| Age at arrest, y | 66±12 | 59±12 | <0.01 |
| Comorbidity score on discharge | 3.0±1.5 | 2.6±1.6 | 0.03 |
| Duration of resuscitation attempt, min | 22.6±13 | 19.9±18 | 0.58 |
| Ejection fraction, % | 42±18 | 42±18 | 0.67 |
| VT/VF | 122 (18) | 25 (52) | <0.01 |
| Medication use | | | |
| ACE-I/ARB | 270 (40) | 30 (67) | 0.01 |
| β-Blocker | 217 (31) | 27 (56) | <0.01 |
| Antiarrhythmic | 50 (8) | 10 (20) | 0.05 |
| Calcium channel blocker | 216 (32) | 19 (39) | 0.62 |

Data are presented as mean±SD or n (%).

ACE-I indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; VF, ventricular fibrillation; and VT, ventricular tachycardia.

**P* value contrasts the 2 categories.



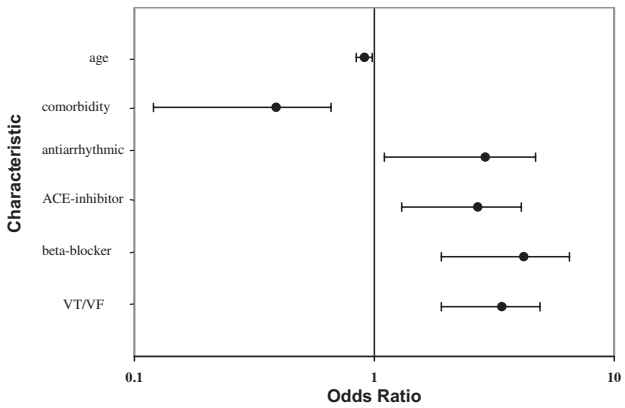
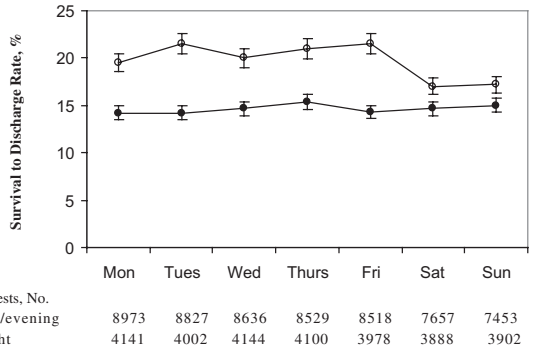



**Figure 5.** Odds ratios for survival by significant clinical characteristics between survivors to discharge and nonsurvivors of in-hospital cardiac arrest. ACE indicates angiotensin-converting enzyme; and VT/VF, ventricular tachycardia/ventricular fibrillation. Reprinted from Bloom et al[18] with permission from Elsevier. Copyright © 2007, Elsevier Inc.




| Total arrests, No. | | | | | | | |
|---|---|---|---|---|---|---|---|
| Day/evening | 8973 | 8827 | 8636 | 8529 | 8518 | 7657 | 7453 |
| Night | 4141 | 4002 | 4144 | 4100 | 3978 | 3888 | 3902 |

**Figure 6.** Survival to discharge rate and total arrests by time category and day of week. Error bars represent 95% confidence intervals. Reprinted with permission from Peberdy et al.[21] Copyright © 2008, American Medical Association. All rights reserved.

collect and review the resuscitation data from the defibrillator and any other device or source documentation that captures data at the scene in a timely manner as a source of postevent feedback to the team.[28] Code carts should be stocked with the necessary ACLS medications and intubation and respiratory supplies, and, where applicable, specialty-specific supplies (eg, pediatric supplies, cesarean section tray).

*Rapid Response Teams*

Rapid response teams were established to prevent IHCA in patients whose condition is deteriorating.[29,30] These teams are usually composed of varying combinations of physicians, nurses, respiratory therapists, and pharmacists and can be summoned to the bedside of a patient who is noted to have an acute clinical decompensation or is thought to be at immediate risk of IHCA and other immediate life-threatening events. Although the theory is compelling, data on the effectiveness of these teams have actually been mixed.[31,32] A recent meta-analysis suggests that although rapid response teams may decrease the incidence of IHCA outside the ICU, they have not convincingly demonstrated significant improvements in survival rates.[9] Possibilities for these counterintuitive results are (1) early identification and transfer of the patient to the ICU, where the patient subsequently experiences an IHCA, and (2) increased use of DNAR orders.[33] Other possibilities include failure to trigger the team when signs of deterioration are noted and poor surveillance methods for identifying clinical deterioration.[34]

*Code Teams*

The Joint Commission[3] requires that resuscitation services and equipment be provided to patients according to the hospital's protocol and that resuscitation outcomes be collected and reviewed. In addition, The Joint Commission requires that evidence-based programs be used to train staff in the need for resuscitation and the use of resuscitation equipment and techniques. However, The Joint Commission does not mandate the composition of code teams. The American Board of Internal Medicine and the Accreditation Council for Graduate Medical Education likewise do not mandate composition or even training of code teams, although the new Accreditation Council for Graduate Medical Education common program requirements clearly emphasize the need for adequate supervision and graded progressive responsibility as core tenets within graduate training in medicine.[35] To fulfill the requirements of The Joint Commission, all hospital staff responsible for the care of patients should be trained in basic life support. This training should include how to recognize a patient whose condition is deteriorating, call for help, start CPR, direct others to get the nearest AED, and use the AED. A designated emergency response team (eg, *code blue* in some hospitals) must be available at all times. Code team composition is mandated by individual hospitals and may consist of nurses, respiratory therapists, pharmacists, physicians, and clergy, as well as security personnel. Mechanisms for triggering a specialty-specific emergency response team for unique situations such as pediatric and maternal-fetal arrests should be available if such clinical situations are a possibility at a specific hospital. A code team leader is responsible for guiding the resuscitative efforts. Code team members must have ACLS Provider cards and be on duty in the hospital and available to respond to codes at all times.[27]

*Education and Training*

All hospital staff should know how to recognize cardiac arrest, call for help, perform chest compressions, and use an AED at the level of a bystander until staff with training in the care of patients with cardiac arrest respond to the event. Some hospitals have made this a minimum requirement for hiring, and others have mandated it as a minimum requirement for continued employment, with annual retraining of all staff.

Education and training of IHCA code team staff are critical to improved performance and better outcomes.[36] IHCA is a relatively low-frequency event, and IHCA code team members have reported feeling ill prepared to lead and participate as members of the team.[37] IHCA treatment generally relies on code teams whose personnel composition changes frequently, and members may not be focused solely on providing emergency resuscitation care. Therefore, there may be aspects of training and skills retention related to providing intra-arrest care that are unique to the hospital setting and require frequent retraining of the team to maintain skills, minimize errors, and optimize outcome.[38,39] Simulation training in addition to ACLS training of house staff at an academic hospital ICU was associated with greater adherence to the *AHA Guidelines for CPR and ECC*.[40]

Very few studies have reported the effect of training on survival from IHCA. In 1 study performed at a 550-bed tertiary care center, the survival rate of patients initially resuscitated by a nurse trained in ACLS was almost 4 times higher (37.5% versus 10.3%) than when resuscitation was initiated by a nurse without ACLS training.[41] One study with some methodological concerns reported an increase in short- and long-term survival rates with ACLS-trained personnel.[42]

One of the more promising training strategies may involve the use of simulation-based mock codes. A recent study

conducted over the course of 48 months suggested that monthly random mock codes that used a simulator and occurred in various patient locations within the hospital were correlated with improved survival rates for pediatric arrest of >50%. These rates were higher than the 2008 national average.[43] In a simulation model evaluation of ventricular fibrillation (VF) IHCA resuscitation, patients were more likely to receive more defibrillations when the physician team arrived early (median arrival 50 seconds after onset). In all cases, there was a median delay of 85 seconds until CPR was started, and 100 seconds elapsed before the first defibrillation. These data suggest that gaps in knowledge, reluctance to act, and team work all need to be addressed through improved training.[44]

### *Care Pathways*

*Prevention Through Early Identification*

IHCA is frequently preceded by clinical deterioration that is evident in symptoms and changes in vital signs that could be identified and treated by trained in-hospital staff.[45] As a result, greater emphasis has been placed on prevention of these events, based on the assumption that earlier identification and intervention to stabilize these patients can prevent IHCA.[46,47] In 2008, The Joint Commission[47] introduced patient safety goals, in which goal 16 specifically targets improved recognition of and response to changes in a patient's condition.[48]

An observational study of surgical and medical wards reported that 1 of 5 patients developed abnormal vital signs, and >50% of these events went unnoticed by nursing staff. The patients with abnormal vital signs had a 3-fold higher 30-day mortality rate.[49] A nested, controlled, in-hospital trial comparing prearrest patients with control subjects at 48 hours before the event suggested that the Modified Early Warning Scores were different, but the authors noted that this scoring system does not include significant predictors such as diastolic and pulse pressures.[50] A study that examined circadian variability and a large registry study of >58 000 IHCAs both demonstrated lower survival rates during nights and weekends.[8,21] Interventions to address consistent and comprehensive staff training in monitoring vital signs, including quantitative end-tidal $CO_2$ waveform capnography and electrocardiographic (ECG) tracings, as well as anticipation of bad outcomes, initial response, and ACLS skills may enable earlier detection, better treatment, and better survival rates regardless of time of day or day of the week.

Very few high-quality evaluations of training interventions to improve the early identification of prearrest patients exist.[51] The more promising educational interventions are the Immediate Life Support[52] and the Acute Life-Threatening Events Recognition and Treatment[53] courses; however, high-quality evaluations of their efficacy are still pending. A longitudinal multicenter study[54] of the Acute Life-Threatening Events Recognition and Treatment course suggested an increase in prearrest calls, a reduction in the number of IHCAs, and improved survival-to-discharge rate after IHCA. A before-and-after comparison of a 1-day course, based on a needs assessment, failed to show any difference; however, only 67% of the nursing staff were trained.[55]

*ECG and Physiological Monitoring*

It is important to keep in mind that many arrests are unmonitored and unwitnessed. Brady et al[56] reported improved survival to discharge and favorable neurological outcome with either monitoring or direct observation compared with unmonitored or unwitnessed IHCA. This retrospective look at registry data also suggested that there was no additional advantage of cardiac monitoring compared with staff observation of the event, which reinforces that early identification and trained response are key.[56] That said, it is very difficult to predict who will experience a cardiac arrest. A tiered approach to the use of ECG monitoring may alert hospital personnel to a life-threatening arrhythmia before the clinical discovery of an unconscious patient; thus, it can save critical minutes from the onset of IHCA to the start of resuscitative efforts.

A 2004 AHA scientific statement provided some guidance on who should be monitored with electrocardiography.[57] Class I indications for monitoring include the following:

- Patients resuscitated from sudden cardiac death
- Patients in the early phase of acute coronary syndromes
- Patients with unstable coronary syndromes and newly diagnosed high-risk coronary lesions
- Adults and children who have undergone cardiac surgery
- Patients who have undergone nonurgent percutaneous coronary intervention (PCI) with complications
- Patients who have undergone implantation of an automated defibrillator lead or a pacemaker lead and who are considered pacemaker dependent
- Patients with a temporary pacemaker or transcutaneous pacing pads
- Patients with atrioventricular block
- Patients with arrhythmias and Wolff-Parkinson-White syndrome
- Patients with long-QT syndrome and arrhythmias
- Patients with intra-aortic balloon pumps
- Patients with acute heart failure
- Patients with indications for intensive care
- Patients undergoing conscious sedation
- Patients with unstable arrhythmias
- Pediatric patients with symptoms of arrhythmia

Class II indications (may be beneficial in some patients) include the following:

- Patients with post–acute myocardial infarction (AMI)
- Patients with chest pain syndromes not thought to be acute coronary syndromes
- Patients who have undergone uncomplicated nonurgent coronary intervention
- Patients who have been administered antiarrhythmic drugs that are not potentially proarrhythmic
- Patients with implanted pacemakers who are not pacemaker dependent
- Patients with uncomplicated ablation of arrhythmia
- Patients with diagnostic coronary angiography
- Patients evaluated for syncope thought to be noncardiac
- Patients with DNAR orders who may have arrhythmias that cause discomfort

Selected low-risk patients admitted with chest pain may not need ECG monitoring.[58,59] ECG monitoring must be of high quality in capturing the trigger of true arrests (sensitivity) and

for avoidance of false alarms (specificity). Sufficient staffing is critical to allow a prompt and appropriate response to the alarms by a nurse or technician.

Other physiological monitoring is also necessary in disease-specific subgroups of patients. Arterial blood pressure monitoring may be performed noninvasively or invasively for patients at risk for hemodynamic instability.[60] Respiratory monitoring is of particular use in patients with sleep apnea. Pulse oximetry for monitoring of patients with pulmonary disease is quite valuable. Quantitative end-tidal $CO_2$ waveform capnography is recommended for ventilation with a bag-mask, for intubated patients,[27,61] and for those undergoing conscious sedation.

### *Process Issues*

*Plan for Routine Debriefing*

It is important to put in place a process for postevent debriefing that best fits the culture of the institution, the resources, and the timing of data capture and analysis. It is important to define a priori who will lead the debriefing (preferably people trained as facilitators) and when it will occur. Debriefing is used to identify best practices unique to the institution, to optimize performance, and to address emotional responses related to the specific event. The impact of debriefing to date has been measured against performance and short- and long-term survival; however, other outcomes, such as factors related to team building, psychological responses, and retention, have not been studied. Debriefing sessions that review clinical and defibrillator-recorded information from a code may improve some but not all aspects of code team performance.[28] In 1 study, debriefing with audiovisual feedback was associated with significantly improved rates of ROSC (59.6% versus 44.6%, $P$=0.03) but did not change survival-to–hospital discharge rates (7.4% versus 8.9%, $P$=0.69). Further study is needed to evaluate routine debriefing with respect to the capacity to build and retain teams, who should conduct the debriefing, when the debriefing should occur, and to define the cost-effectiveness of this intervention.

*DNAR Orders*

Resuscitation is not always desired by the individual, and in many cases it is medically futile. Advance directives, living wills, and durable power of attorney for health care and patient self-determination ensure that patient preferences will guide care even when the patient is unable to make decisions on his or her own. Advance planning by the patient or proxy decision maker is ultimately in the best interest of the patient, because studies have shown that these decisions are associated with better care, quality of life, and bereavement adjustment by caregivers.[62] Advance directives should be discussed with and documented for all patients admitted to the hospital. DNAR orders should be completed, signed, and dated by the physician after a documented discussion with the patient and/or family or legal representative. This will avoid unwanted or futile resuscitation and the subsequent need for early withdrawal of life-sustaining therapy. It is important to be frank with the patient and explain the probability of surviving IHCA, because most older patients readily understand prognostic information and can make decisions on whether they would like to receive CPR.[63–65] The DNAR order should preferably state either full resuscitation or no attempt at resuscitation; however, certain situations or patient or family preferences may warrant explicit instructions about which interventions to withhold or provide (eg, CPR without intubation, medications without CPR). This may include but is not limited to vasopressor agents, blood products, advanced airway interventions, nutrition, fluids, analgesia, sedation, antiarrhythmic drugs, and defibrillation.[66]

## Best Practices: Intra-arrest

### *Brief Introduction to Intra-arrest Care*

High-quality CPR, with optimal chest compressions and ventilations, and early defibrillation are cornerstones of intra-arrest treatment that have improved survival from OHCA.[67] There is growing evidence that optimizing these treatment cornerstones for IHCA could also improve outcomes in this setting.[68,69] Periodic evaluation of residents trained in pediatric advanced life support revealed that they did not meet performance standards specified by the *2010 AHA Guidelines for CPR and ECC,* which suggests that training is not enough to ensure performance.[70] Implementation strategies to ensure timely access to equipment, visual reminders, regular testing, and point-of-care feedback may be required to optimize the translation of guidelines into practice during cardiac arrest.

### *Structural Aspects of the Institutional Response*

*Mechanical Chest Compressions*

The use of mechanical chest compression devices in the in-hospital setting has been reported, particularly in settings where the performance of manual CPR is difficult, such as during in-hospital transport[71] and PCI.[72] Mechanical devices include active compression-decompression and load-distributing band devices that automatically compress the chest. There are reports that mechanical chest compression devices improve coronary perfusion pressures during IHCA compared with manual chest compressions.[73] IHCA studies of mechanical compression devices have been limited to small case series involving a handful of patients. As an example of the current literature on this subject, a recent case series of 28 patients with IHCA who presented in pulseless electrical activity (PEA) and were treated with mechanical chest compressions demonstrated a 50% rate of survival to discharge and a 46% rate of good neurological outcome.[74] If these devices are used, it is important to provide training that minimizes interruptions in chest compressions during use of the device; however, there are limited data to support the routine use of these devices for IHCA.

*Automated External Defibrillators*

AEDs may play a role in improving early defibrillation times, particularly in less intensively monitored areas of the hospital. Approximately half of all IHCAs occur outside the ICU.[5] Implementation of a public access defibrillation program at a tertiary care hospital included targeted placement of AEDs in areas where time from arrest to arrival of a defibrillator would be >3 minutes, including time spent in parking garages and on walkways between buildings.[75] In a study of 439 patients with IHCA, a program to equip and train nurses outside of the ICU setting to use AEDs resulted in an 86% rate of ROSC for patients with pulseless ventricular tachycardia (VT)/VF and a 47% rate of survival to hospital discharge.[76] In another study, placement of AEDs in 14 locations that could be easily reached from all wards and diagnostic rooms within 30 seconds was

combined with a 2-hour AED training program for medical officers, nurses, and administrative and technical staff. In the 18 recorded cases of pulseless VT/VF, rates of ROSC and survival to hospital discharge were 88.9% and 55.6%, respectively.[77] Although these studies did not compare AED resuscitation rates with prior non-AED rates, 1 study did show an improvement in outcomes of patients with pulseless VT/VF. After implementation of a program that included education and encouraged use of manual biphasic defibrillators in AED mode, as well as placement of AEDs in all outpatient clinics and chronic care units, IHCA survival to discharge improved by 2.6 times from 4.9% to 12.8%.[78] AEDs performed similarly to biphasic manual defibrillators in AED mode. A recent large registry study of IHCAs suggested that there was no association with increased survival and use of an AED with VF and pulseless VT and decreased survival with the nonshockable initial rhythms.[79] The decrease in survival from nonshockable rhythms could be attributed to the mandatory time off the chest to allow for analysis and shock delivery with an AED. Time to first contact by the cardiac arrest team was not compared in this study, and it is likely that AEDs were placed in areas less well served by the cardiac arrest team, representing a potential selection bias. In addition, AEDs were grouped with manual defibrillators that could be used in AED mode, but it was unknown whether the latter were used in manual or automatic mode, which makes it harder for the AED group to demonstrate superiority over the manual mode.[79] Additional randomized clinical trials are required to evaluate and optimize use of AEDs in the hospital.

*Automated External Cardioverter-Defibrillators*

Automated external cardioverter-defibrillators (AECDs) may play a role in more intensively monitored areas of the hospital. These devices differ from AEDs in that they provide continuous cardiac monitoring with 2 pads placed on the patient's chest and can automatically defibrillate shockable rhythms. In 1 prospective study of AECD monitoring of ED patients considered to be at risk for pulseless VT/VF (n=55), the average interval between onset of arrhythmia and first defibrillation was 33 seconds and resulted in a 94.4% rate of ROSC.[80] A prospective trial (Automatic External Defibrillation Monitoring in Cardiac Arrest; ClinicalTrials.gov, unique identifier NCT00382928) has completed enrollment of telemetry patients with IHCA randomly assigned to a cardiac arrest team or standard CPR plus AECD monitoring.[81] The AECD in the study was programmed to deliver one 150-J biphasic shock to patients in sustained pulseless VT/VF. The primary end point was time to defibrillation, with secondary outcomes including neurological status and survival to discharge and 3-year follow-up. Preliminary data demonstrated that 1 of 192 enrolled patients experienced sustained pulseless VT during AECD monitoring, and this patient was successfully defibrillated within 17 seconds. There were no events in the control group; however, during the same period, mean time to shock for pulseless VT/VF IHCA that occurred outside the telemetry ward was 230±50 seconds.

### Care Pathways

*Performance of CPR*

A major opportunity for hospitals to improve patient care involves monitoring and improving CPR performance.[26,28,82] Optimizing ventilations (a ratio of 30:2) and providing chest compressions at a rate of 100/min and a depth of at least 5 cm while minimizing pauses (hands-off time) will improve outcomes from IHCA.[69] Despite the importance of chest compressions in cardiac arrest outcome, they are rarely performed according to guideline recommendations.[69] In studies of IHCA, chest compression rates were too slow >30% of the time.[68,69] In addition, 33% of compressions were too shallow, and ≈20% of resuscitation time consisted of interruptions and no-flow time. Rescuer fatigue contributes to poor-quality CPR, and rescuers who provide ventilations and compressions should be replaced or should switch places after each 2-minute cycle.[83] Strategies for improving the quality of each component of CPR are reviewed below.

*Decrease Interruptions in Chest Compressions*

Bystander CPR and CPR provided by healthcare professionals improve outcomes in OHCA and IHCA, respectively.[69,84] Interruptions in chest compressions may decrease the compression fraction, which has been associated with decreased survival rates[67] in OHCA. Some out-of-hospital strategies that include continuous compressions without pauses for ventilations have been associated with improved outcomes.[85,86] Interruptions for even a few seconds can decrease coronary blood flow,[87] and are associated with worsened neurological outcome in animal models,[88] and may decrease survival to discharge in OHCA.[89] Pauses in chest compressions of ≥10 seconds' duration have been associated with decreased success of defibrillation.[90] A correctly performed compression-to-ventilation ratio of 30:2 should be consistent, with 2 ventilations delivered within 2 seconds off the chest for each set of 30 compressions or 4 to 6 seconds off the chest per minute during the 2 minutes between rhythm analyses. To reduce hands-off time during analysis and charging, newer versions of defibrillator software enable interpretation of the ECG and continuous charging of the capacitor during chest compressions,[91] which minimizes the pause to a few seconds before the shock is delivered.

*Avoid Hyperventilation*

Excessive ventilation rates are often observed during CPR for OHCA[92] and IHCA.[69] Fast ventilation rates in the laboratory are associated with increased intrathoracic pressures, lower coronary perfusion pressures, and decreased survival rates.[92,93] Devices that prompt or time ventilation through timing lights or audio cue during CPR may be useful to prevent excessive ventilation. In addition to improving quality of chest compressions, code team debriefing with audiovisual feedback has been associated with a decrease in mean ventilation rates from 18/min to 13/min.[28]

*Optimize Chest Compression Depth*

Greater chest compression depth and a decreased preshock pause interval before defibrillation have been associated with increased defibrillation success and ROSC after IHCA.[90] Adequate chest compression depth in OHCA has been associated with survival to hospital admission,[94] but improved survival to discharge as a function of adequate chest compression depth has not yet been demonstrated in either OHCA or IHCA. The *2010 AHA Guidelines for CPR and ECC* have changed the emphasis to a depth of at least 5 cm with each compression.[95]

The depth of compression in the IHCA setting has likely been overestimated because of movement of the patient's mattress with manual chest compressions.[96,97] Recent simulation studies demonstrated that even with the use of a backboard, mattress compression can account for as much as 40% of measured compression depth in patients with IHCA.[96,97] When a single accelerometer is applied to the sternum to measure chest compressions, as used in the majority of clinical studies, the actual compression depth is overestimated by as much as 4 to 13 mm, depending on mattress type.[96,97] Thus, deeper chest compressions in the IHCA setting may be needed to compensate for mattress movement if it cannot be neutralized by the use of a backboard.

*Provide Early Defibrillation*

Approximately 25% of patients with IHCA have a shockable rhythm of pulseless VT/VF.[5,98] Despite proximity to advanced health care, >30% of patients with IHCA have defibrillation times of >2 minutes after arrest.[99] Defibrillation times longer than 2 minutes after IHCA have been associated with decreased rates of survival to hospital discharge. Delays were also associated with black race, noncardiac admitting diagnosis, time of arrest during evenings and weekends, and hospitals with <250 beds. When nonphysicians are allowed to perform defibrillation, it can save critical seconds to minutes until the emergency response team arrives.[76] Strategies used to increase early defibrillation after IHCA include use of hands-free pads (which decrease preshock pause),[100] use of AEDs in non-ICU settings, and use of AECDs.

*Identify and Treat Underlying Causes*

The most common causes of IHCA include cardiac arrhythmia, acute respiratory insufficiency, and hypotension.[5] Studies show that asystole and PEA are more common than VF in adult IHCA, with only 25% of patients having VF or pulseless VT as the initial rhythm,[5,79] whereas children were more likely to present with asystole (40% versus 35% in adults).[10] The frequency of PEA as the first presenting rhythm in adult IHCA is 30% and has remained unchanged over many years.[5,10] Only 10% of patients with IHCA who present with an initial rhythm of PEA or asystole have neurologically intact survival.[5] Thus, identification and treatment of the reversible causes that may present with PEA/asystole are important during IHCA. Very little has been published on the specific causes of PEA in this setting; however, expert opinion suggests that a substantial number of cases may be secondary to respiratory insufficiency and shock and may respond to targeted therapy. A number of special situations may cause IHCA and require unique interventions that are disease specific. A detailed overview of these situations is provided in the sections on special considerations and pediatrics of the *2010 AHA Guidelines for CPR and ECC*.[101,102]

### *Process Issues*

*Use Real-Time Feedback*

Devices that prompt or time ventilation (eg, timing lights) and guide rhythm of chest compressions (eg, metronome) and quality of compressions (eg, quantitative end-tidal $CO_2$ waveform capnography) during IHCA may be helpful. A cohort study with historical controls demonstrated improvements in chest compressions and ventilations with point-of-care feedback, but no difference was found in either ROSC or survival to hospital discharge.[26] Audio prompting of chest compressions through use of technology as simple as a metronome has been found to improve blood flow during CPR both in animal models and during resuscitation attempts in humans.[103,104] A recent randomized trial on OHCA demonstrated that point-of-care feedback did not improve patient outcomes in well-trained services participating in randomized controlled trials,[105] whereas Edelson et al demonstrated the usefulness of employing quality of CPR measures during real IHCA to evaluate the efficacy of training,[38] and this finding was subsequently verified for both OHCA and IHCA in a systematic review[39] in 2009. The latter suggested that there was good evidence to support the use of point-of-care feedback in training and that it may be beneficial in clinical application.[39] Point-of-care feedback on CPR quality is generally thought to be helpful, and it makes sense in IHCA, because staff are accustomed to using technology to guide care.

## Best Practices: Postarrest

### *Brief Introduction to Postarrest*

For patients who achieve ROSC, variability in survival rates between hospitals exists and can range from 54% to 32%.[106] Higher-volume hospitals and teaching hospitals have the highest survival rate, which averages 38% for patients who have an arrest outside the ICU and 32% for patients who have an arrest in the ICU.[10] Clinical investigation focused on improving outcomes of patients who achieve ROSC after IHCA has been limited and has made it necessary for practitioners to extrapolate from OHCA studies when developing diagnostic and treatment strategies. Patients with ROSC after cardiac arrest in any setting will suffer from a complex combination of pathophysiological processes previously described as the post–cardiac arrest syndrome.[107] Key components include (1) postarrest brain injury, (2) postarrest myocardial dysfunction, (3) systemic ischemia/reperfusion response, and (4) persistent acute and chronic pathology that precipitated cardiac arrest.[107] Persistence of preexisting conditions and precipitating pathologies after ROSC provide significant challenges in management of patients resuscitated from IHCA (Table 6). Multisystem organ failure is a more common cause of death in the ICU after initial resuscitation from IHCA than after OHCA.[108] Patient management is also affected by the location of the arrest within the hospital (Table 1), the intensity of support (eg, mechanical ventilation and vasopressor therapy), and invasive monitoring in place at the time of arrest (Table 7). In all cases, optimal post-IHCA care requires a well-coordinated multidisciplinary team. Clinical trials evaluating treatment strategies in postarrest patients are lacking for both OHCA and IHCA. Much of the evidence is based on animal studies, cohort comparisons, and extrapolations from diseases that share similar characteristics, such as sepsis.

### *Structural Aspects of the Institutional Response*

Comprehensive postarrest care requires access to and collaboration between a multidisciplinary team of providers, including emergency medicine (if the arrest occurs in the ED), cardiology, interventional cardiology, cardiac electrophysiology, intensive care, and neurology. If these services are not available, the institution needs to have in place an interhospital transfer agreement

**Table 6. Post–Cardiac Arrest Syndrome: Pathophysiology, Clinical Manifestations, and Potential Treatments**

| Syndrome | Pathophysiology | Clinical Manifestation | Potential Treatments |
|---|---|---|---|
| Post–cardiac arrest brain injury | • Impaired cerebrovascular autoregulation<br>• Cerebral edema (limited)<br>• Postischemic neurodegeneration | • Coma<br>• Seizures<br>• Myoclonus<br>• Cognitive dysfunction<br>• Persistent vegetative state<br>• Secondary parkinsonism<br>• Cortical stroke<br>• Brain death | • Therapeutic hypothermia<br>• Early hemodynamic optimization<br>• Airway protection and mechanical ventilation<br>• Seizure control<br>• Controlled reoxygenation ($Sa_{O_2}$ 94%–96%)<br>• Supportive care |
| Post–cardiac arrest myocardial dysfunction | • Global hypokinesis (myocardial stunning)<br>• ACS | • Reduced cardiac output<br>• Hypotension<br>• Dysrhythmias<br>• Cardiovascular collapse | • Early revascularization of AMI<br>• Early hemodynamic optimization<br>• Intravenous fluid<br>• Inotropes<br>• IABP<br>• LVAD<br>• ECMO |
| Systemic ischemia/reperfusion response | • Systemic inflammatory response syndrome<br>• Impaired vasoregulation<br>• Increased coagulation<br>• Adrenal suppression<br>• Impaired tissue oxygen delivery and utilization<br>• Impaired resistance to infection | • Ongoing tissue hypoxia/ischemia<br>• Hypotension<br>• Cardiovascular collapse<br>• Pyrexia (fever)<br>• Hyperglycemia<br>• Multiorgan failure<br>• Infection | • Early hemodynamic optimization<br>• IV fluid<br>• Vasopressors<br>• High-volume hemofiltration<br>• Temperature control<br>• Glucose control<br>• Antibiotics for documented infection |
| Persistent precipitating pathology | • Cardiovascular disease (AMI/ACS, cardiomyopathy)<br>• Pulmonary disease (COPD, asthma)<br>• CNS disease (CVA)<br>• Thromboembolic disease (PE)<br>• Toxicological (overdose, poisoning)<br>• Infection (sepsis, pneumonia)<br>• Hypovolemia (hemorrhage, dehydration) | • Specific to cause but complicated by concomitant PCAS | • Disease-specific interventions guided by patient condition and concomitant PCAS |

ACS indicates acute coronary syndrome; AMI, acute myocardial infarction; CNS, central nervous system; COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; IV, intravenous; LVAD, left ventricular assist device; PCAS, post–cardiac arrest syndrome; and PE, pulmonary embolism.





and a process to ensure access to these resources within the first 6 hours after arrest if therapeutic hypothermia cannot be initiated in the sending facility. The timing of transfers should take into consideration the time-sensitive nature of potential interventions such as PCI and therapeutic hypothermia.

### *Care Pathways*

*Induction of Goal-Directed Mild Therapeutic Hypothermia*

Mild therapeutic hypothermia (32°C to 34°C) improves outcome of comatose survivors of witnessed OHCA when the initial rhythm is VF.[109,110] Similar studies have not been performed in patients who achieve ROSC after IHCA. The potential detrimental or beneficial effect of mild therapeutic hypothermia on active pathologies, comorbidities, and ongoing therapies must be considered. The role of therapeutic hypothermia in the management of IHCA and with initial rhythms other than VF in either the out-of-hospital or in-hospital setting is an important knowledge gap that needs to be addressed by future research. Despite this gap in research, the *2010 AHA Guidelines for CPR and ECC* recommend that induced hypothermia may be considered for comatose adult patients with ROSC after IHCA of any initial rhythm.[111]

*Coronary Reperfusion for ST-Segment Elevation Myocardial Infarction*

PCI for patients resuscitated from IHCA is an important therapeutic consideration. According to the Get With The Guidelines–Resuscitation data, only 11% of treated IHCAs are caused by AMI.[10] In a retrospective review of 110 survivors of IHCA caused by VF, only 30 patients (27%) underwent cardiac catheterization on the day of the arrest, and of these, only 13 patients had an ECG with results consistent with ST-segment–elevation myocardial infarction or new left bundle-branch block. Patients who underwent cardiac catheterization were more likely to survive than those who did not receive cardiac catheterization. Of those who underwent catheterization, 17 patients had a successful PCI.[112] In post-IHCA patients, management of suspected AMI should be similar to management of AMI in the nonarrest population; however, the extension of indications for immediate PCI beyond ST-segment–elevation myocardial infarction or new left bundle-branch block remains controversial. A recent observational study of survivors of OHCA who were treated with therapeutic hypothermia and selected for cardiac catheterization demonstrated that at least 1 significant coronary lesion existed in 58% of patients without any ST-segment elevation.[113]

**Table 7. Post–Cardiac Arrest Syndrome: Monitoring Options**

| General Intensive Care Monitoring | More Advanced Hemodynamic Monitoring | Cerebral Monitoring |
|---|---|---|
| • Arterial catheter<br>• Oxygen saturation by pulse oximetry<br>• Continuous ECG<br>• CVP<br>• $Scvo_2$<br>• Temperature (bladder, esophagus)<br>• Urine output<br>• Arterial blood gases<br>• Serum lactate<br>• Blood glucose, electrolytes, CBC, and general blood sampling<br>• Chest radiograph | • Echocardiography<br>• Cardiac output monitoring (either noninvasive or PA catheter) | • EEG (on indication/continuously): early seizure detection and treatment<br>• CT/MRI |

CBC indicates complete blood count; CT/MRI, computed tomography/magnetic resonance imaging; CVP, central venous pressure; ECG, electrocardiogram; EEG, electroencephalogram; PA, pulmonary artery; and $Scvo_2$, central venous oxygen saturation.

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This association suggests that a more liberal practice of offering emergent cardiac catheterization with PCI may be helpful in all arrests of presumed cardiac origin independent of ECG findings after IHCA; however, clinical trials are required before practice should change to manage this controversial subgroup of patients with angiography and PCI. It is recommended that all IHCA patients with ROSC after arrest with ST-segment–elevation myocardial infarction or new left bundle-branch block undergo emergent angiography and, when indicated, PCI.[27]

*Early Hemodynamic Optimization*

For patients with ROSC after IHCA, the high prearrest prevalences of respiratory insufficiency, hypotension/hypoperfusion, congestive heart failure, and infection are likely to exacerbate the cardiovascular dysfunction observed in the post-IHCA period.[114] These complicating factors emphasize the importance of goal-directed therapy based on hemodynamic monitoring. The therapeutic approach has been extrapolated from randomized trials on postoperative patients and those presenting with sepsis,[115–117] which seems appropriate given the similarities in myocardial dysfunction and ischemic reperfusion response with both sepsis and postarrest patients.[118] Perhaps most important to consider is the volume status of the patient before IHCA. For example, IHCA caused by unrecognized severe sepsis or septic shock could require volume resuscitation that exceeds what is typically needed for the treatment of postarrest syndrome alone. Conversely, patients who have an arrest in the ICU despite optimized preload and infusion of vasopressors and inotropes are likely to require different interventions to optimize oxygen delivery.

*Seizure Prophylaxis*

There are no controlled clinical trials that address the issue of seizure prophylaxis in post-IHCA patients.[119,120] Randomized trials comparing single-dose diazepam, magnesium, or thiopental with placebo suggest that there is no difference in neurological outcome.[121,122] The *2010 AHA Guidelines for CPR and ECC* do not recommend seizure prophylaxis in post-IHCA patients.[111]

*Seizure Treatment*

Two studies, 1 cohort[123] and 1 randomized trial,[122] suggest that the incidence of seizures in post-IHCA patients is 5% to 21% with or without therapeutic hypothermia. It is anticipated that the actual rates may be even higher than reported because the occurrence of seizures in the comatose patient may not be clinically apparent without continuous electroencephalography. The *2010 AHA Guidelines for CPR and ECC* recommend frequent or continuous electroencephalographic monitoring of all comatose survivors of cardiac arrest.[111] Some studies suggest that post-IHCA seizures may be challenging to treat and respond poorly to anticonvulsant therapies[124–126]; however, the guidelines advocate prompt and aggressive treatment after the first seizure in post-IHCA patients and status epilepticus when it occurs.[119,120]

*Glucose Control*

Hyperglycemia is likely to develop in the post-IHCA patient, but the optimum blood glucose concentration or interventional strategy to manage blood glucose in the post-IHCA period for in-hospital or out-of-hospital arrest is unknown. Evidence from several retrospective studies[127–131] suggests an association of higher glucose concentrations with increased mortality or worse neurological outcomes. Only 1 study examined the association of glucose concentration with outcomes in survivors of IHCA.[127] These studies do not provide evidence that an interventional strategy to manage glucose concentrations will alter outcomes in post-IHCA patients.

Only 1 randomized trial[132] evaluated strict glucose control (72 to 108 mg/dL [4 to 6 mmol/L]) compared with moderate glucose control (108 to 144 mg/dL [6 to 8 mmol/L]) in survivors of OHCA presenting with VF and treated with induced hypothermia. The use of strict glucose control did not demonstrate a survival benefit at 30 days, and the study was stopped for futility after only 25% of the planned sample size was enrolled. Randomized interventional trials of glucose control in survivors of IHCA have not been performed. Studies performed in critically ill patients also do not support efforts to control glucose in a low range.[133,134] The largest randomized trial of intensive glucose control (81 to 108 mg/dL [4.5 to 6 mmol/L]) versus conventional glucose control (144 to 180 mg/dL [8 to 10 mmol/L]) in ICU patients[135] reported increased mortality in patients treated with intensive glucose control (odds ratio for intensive control, 1.14; 95% confidence interval, 1.02–1.28; $P$=0.02). Intensive therapy to control glucose concentration in critically ill patients consistently results in more

frequent episodes of severe hypoglycemia (usually defined as blood glucose concentration ≤40 mg/dL [2.2 mmol/L]), and hypoglycemia may be associated with worse outcomes in critically ill patients.[136,137] On the basis of current evidence and experience, glucose concentrations of 144 to 180 mg/dL (10 mmol/L) are reasonable in adult patients after cardiac arrest (both IHCA and OHCA), and control of glucose concentration within a lower range (<110 mg/dL [<6.1 mmol/L]) should not be implemented after arrest because of the increased risk of hypoglycemia.

### *Process Issues*

*Use of a Comprehensive Protocol*

There are no randomized controlled trials evaluating the use of a comprehensive protocol of care for post-IHCA patients. Before-and-after studies suggest that the successful implementation of a standardized protocol addressing the complexity and comprehensiveness of care for post-OHCA patients results in a decrease in in-hospital mortality.[123,138]

*Withdrawal of Life-Sustaining Therapy and Prognostication After IHCA*

Registry data suggest that prognostication of futility in care may be premature, especially given improvement in outcomes emerging with the use of therapeutic hypothermia.[5] The average length of stay for survivors of IHCA was 2 weeks; it was <2 days for those who died in the hospital despite aggressive treatment or for those whose care was withdrawn.[5] Two days is too short, and futility cannot be accurately prognosticated in most cases at this interval after arrest.[5] Among 24 132 comatose survivors of IHCA admitted to critical care units in the United Kingdom, treatment was withdrawn in 28.2% at a median of 2.4 days (interquartile range, 1.5 to 4.1 days).[129] The Get With The Guidelines–Resuscitation data suggest that 63% of patients with ROSC after an IHCA are declared DNAR, and 44% of these have life-sustaining care withdrawn, yet only 8% are declared clinically brain dead.[2] The average length of stay was 1.5 days for those who died in the hospital, which may suggest that many patients have withdrawal of life support before accurate neurological prognostication. The current guidelines suggest that prognostication should wait until after 24 hours in a patient who was not treated with therapeutic hypothermia and after 72 hours in a patient who was treated with therapeutic hypothermia (Table 8). Beyond these intervals, the accuracy of prognostication will depend on both the modalities used and the time after ROSC. Most importantly, current guidelines state that there is not a single prognostic test that can safely and adequately predict outcome[66,111] (Table 8). This means that a composite of prognostic modalities combined with clinical judgment continues to guide decisions about when to withdraw life-sustaining therapy after aggressive treatment.

*Organ Donation After IHCA*

The reported incidence of clinical brain death in patients with sustained ROSC after IHCA ranges from 8% to 16%.[5,169] These patients can and should be considered for organ donation. In the 2003 Get With The Guidelines–Resuscitation study, only 1.3% of patients had organ recovery, which suggests much more aggressive procurement strategies are required.[5]

## Changing Culture and Standardizing Reporting and Benchmarking

IHCA resuscitative efforts are perceived by healthcare providers to be futile, and post-IHCA patients are often assigned a hopeless prognosis.[195,196] Improving outcomes for IHCA requires a change in the culture through standardized reporting, knowledge, training, and better systems of care. IHCA outcomes will respond to aggressive management, attention to detail, implementation of guideline-based management, and standardized performance measurement, as well as improvement initiatives.[107,115,138]

### Changing the Cultural Aspects of IHCA Care

The current culture of hopelessness in outcomes from IHCA may stem from the lack of knowledge and uniform reporting standards and may breed impatience and early withdrawal of life-sustaining therapy, which no doubt contributes to the consistently low survival rates that have remained unchanged for decades. Survival is a product of science, education, and implementation[123,197,198]:

Survival = Science × Education × Implementation

Of the 3 factors, there is the least evidence to guide implementation in IHCA, but there is substantial literature on knowledge translation in other disease entities from which one can extrapolate to ICHA care. Perception drives institutional culture and individual behavior. To change perception requires information from a credible source; institutional acceptance by a majority, including the leadership; and translation into point-of-care tools that assist with adherence with each and every patient. Information must be useful, desirable, accessible, credible, findable, and usable.[199] Pathman et al[200] suggested that there are 4 stages to change: Awareness, agreement, adoption, and adherence. Publication of guidelines alone does not incite the rapid dissemination of information and eager adoption. Prehospital services and providers took an average of 1.5 years to implement the *2005 AHA Guidelines for CPR and ECC*[201]and even longer to adopt the process of uploading the CPR process data and using the data to guide care through feedback and universal reporting of outcomes.[202] Social networks suggest that stories trump data and relationships trump stories. Perhaps institutions working to transform practice should celebrate their stories and build relationships across departments and programs internally and partners externally (community survivor groups). Inherently, every healthcare provider wants to give the best possible care. Drawing on what we know about knowledge translation to guide effective implementation and the power of the social network to engage and effect change, it is possible for institutions to transform the culture of hopelessness into one of hopefulness and pride.

### Capturing and Reporting the Data: IHCA Benchmarking

The current approach to surveillance of IHCA incidence, process, and outcomes relies heavily on billing information, which is insufficient because of limitations of coding as it relates to IHCA in all 3 commonly used coding systems: Diagnosis-Related Group (DRG), the ICD, and Current Procedural

Terminology (CPT). This approach needs to change to conform to universal reporting standards of incidence and outcome.

There are a number of DRG and *ICD-10* codes for cardiac arrest and postarrest care, and this permits discretionary recording based on the preference or training of the coder (Table 9). For example, DRG 129 is "cardiac arrest, unexplained," and *ICD-9* 427.5 is "cardiac arrest"; however, there are probably a dozen other DRGs that could be used for "explained" that are not specific to cardiac arrest; for example, DRG 121 is "circulatory disorder with AMI and major complications discharged alive." The *ICD-9* code 427.5 is labeled "cardiac arrest"; however, this coding produces substantial noise when used to report IHCA. For example, this code can be used as a primary diagnosis for (1) patients who have only transient ROSC and die in the ED or (2) patients who have ROSC and survive to be admitted to the hospital but die before the cause of the arrest is determined, and as a secondary diagnosis for (1) patients who have an IHCA during the course of hospitalization and are resuscitated or (2) patients who present to the ED in cardiac arrest with a discernible cause and survive to be admitted to the hospital. Furthermore, health data coders may not include code 427.5, preferring to rank other codes higher (eg, comorbidities), which will skew the coded incidence of IHCA. The *ICD-9* code 427.5 appears to be preferred but not exclusively used by coders for patients who achieve ROSC.

A study of all non-DNAR hospitalized patients reported that *ICD-9* code 427.5 had 43% sensitivity for identifying patients with treated IHCA. Similarly, *ICD-9* code 427.4, "ventricular fibrillation and flutter," could be used both for patients undergoing in-patient electrophysiological procedures and those with IHCA.

A recent study attempted to use billing data to determine the incidence of IHCA in Medicare patients >65 years of age.[23] Investigators used 2 procedural codes derived from *ICD-9*,[203] 99.60 for "cardiopulmonary resuscitation, not otherwise specified," and 99.63 for "closed chest cardiac massage," to calculate an incidence of 2.73 IHCAs per 1000 admissions. This estimate included resuscitations irrespective of location within the hospital, and patients who did not get billed for either procedure, which is not uncommon in the in-hospital setting, would have been excluded. Furthermore, this study may not be representative of the hospitalized patient population as a whole, because it was limited to patients >65 years of age.

To add to these coding challenges, a current CPT code for therapeutic hypothermia does not exist. Specific ICD codes for "inpatient cardiac arrest" and "postarrest syndrome" and a CPT code for "postarrest therapeutic hypothermia" would greatly enhance the utility of billing data based on IHCA surveillance. CPT codes for defibrillation and CPR exist and are tied to remuneration, but they are infrequently and inconsistently used because the incremental benefit of billing is modest and may contribute to undercounting IHCA. To bill these codes requires the presence of the attending physician at the resuscitation, which does not always occur in teaching hospitals.

As a result, tracking of incidence and mortality rates for IHCA as a measured outcome for performance at a local, regional, or national level has not been applied with the same scrutiny as that directed at other cardiac processes such as AMI and congestive heart failure.[204,205] As health care evolves and performance measurement plays an increasing role in reimbursement and performance improvement, it is critical that IHCA incidence, outcome, and treatment variables become measurable entities and that they be reported as mandatory components for regulators and reimbursement bodies.

The Get With The Guidelines–Resuscitation registry[13] provides a universal platform to report IHCA and outcomes in a comparable way across institutions that choose to and can afford to capture the data. Participating hospitals require that some staffing be directed to support data capture; however, in return for this investment, the hospital receives consistent comparable reports on institutional performance relevant to IHCA, including outcomes.

## Need for Public Reporting of IHCA

Currently, IHCA statistics are internally collected but not publicly reported. Public reporting of comparative information on patient outcomes among physicians and hospitals is intended to facilitate informed decision making by patients and referring physicians. Such quality information also has the potential benefit of providing feedback on patient care, thereby identifying areas for quality improvement and motivation to improve adherence to guidelines and overall performance.[206,207] Among the potential drawbacks of reported outcome measures is the pressure placed on physicians and hospitals to avoid performing measurable and reported interventions in the sickest, highest risk patients.[208,209] Despite careful efforts to develop risk adjustment methods that account for severity of illness in mortality reporting, there is concern that current models do not accurately reflect mortality risk in the sickest patients undergoing PCI[210–212] and that the consequent effect is physician mistrust of outcomes measures and avoidance of PCI in the highest-risk patients, who may nevertheless benefit from the procedure.[209,213]

Suggested strategies to address this dilemma include the following:

1. Improve risk adjustment methods for the highest-risk patients to include a "compassionate use" category and bring attention to hospitals and physicians who appropriately treat this population.
2. Provide adequately resourced data collection methods as part of mandated outcomes reporting.
3. Develop risk-adjusted reporting standards that can be applied nationally as opposed to state-specific standards.
4. Develop and report measures that address appropriateness of care given to the sickest patients.[213]

Thus, any call for public reporting of outcomes and interventions in IHCA first requires the development of measurement and adjustment tools to adequately account for severity of illness and other comorbid factors and incorporate these strategies.

## The Impact on IHCA of Establishment of Cardiac Arrest Centers Is Unknown

IHCA may occur in any institution with or without the capacity to provide comprehensive timely post-IHCA care. Optimal treatment of patients with postarrest syndrome requires

**Table 8. Neuroprognostication When Treated or Not Treated With Therapeutic Hypothermia**

| | No Treatment With Therapeutic Hypothermia* | | | | | | Treatment With Therapeutic Hypothermia* | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | ≥24 h | | At 48 h | | ≥72 h | | ≥24 h | | ≥72 h | |
| Diagnostic Tests | Poor Outcome (CPC 3 or 4 or Death) | Reliability | Poor Outcome (CPC 3 or 4 or Death) | Reliability | Poor Outcome (CPC 3 or 4 or Death) | Reliability | Poor Outcome (CPC 3 or 4 or Death) | Reliability | Poor Outcome (CPC 3 or 4 or Death) | Reliability |
| GCS total score | … | … | <5 at 48 h | FPR 0%, 95% CI 0%–13%[139,140] | <5 at 72 h | FPR 0%, 95% CI 0%–6%[139,141] | … | … | … | … |
| GCS motor score | … | … | … | … | 1 at 72 h | FPR 5%, 95% CI 2%–9%[139,142] | … | … | ≤2 at 72 h | FPR 14%, 95% CI 3%–4%[139,143] |
| Pupillary light and corneal reflexes | … | … | … | … | Absence of both at 72 h | FPR 0%, 95% CI 0%–9%[139,142] | … | … | Absence of both at 72 h | FPR 0%, 95% CI 0%–48%[139,143,144] |
| Vestibulo-ocular reflex | Absence at ≥24 h | FPR 0%, 95% CI 0%–14%[139,145,146] | … | … | … | … | … | … | … | … |
| Median nerve somatosensory-evoked potentials measured | >24–72 h Bilateral absence of N20 component of evoked potentials in comatose patients secondary to hypoxic-anoxic origin is fatal | FPR 0.7%, 95% CI 0.1%–3.7%[139,147] | … | … | Bilateral absence of N20 component of evoked potentials at 72 h in comatose patients secondary to hypoxic-anoxic origin is fatal | FPR 0.7%, 95% CI 0.1%–3.7%[139,147] | >24–72 h Bilateral absence of N20 component of evoked potentials in comatose patients secondary to hypoxic-anoxic origin is fatal | FPR 0%, 95% CI 0%–69%; FPR 3%, 95% CI 0%–14%; and FPR 0% 95% CI 0%–13%[143,144,148] 95% CI 0%–13% | … | … |
| Brainstem auditory evoked potentials | Abnormal recorded 1–56 d after | FPR 0%, 95% CI 0%–14%[139,144,149] | … | … | … | … | … | … | … | … |
| Myoclonus | Observed | Not recommended: accurate diagnosis is problematic; some have had complete recovery[139,150–154] | … | … | … | … | Observed | Not recommended: accurate diagnosis is problematic; some have had complete recovery and CI wide; FPR 0%, 95% CI 0%–40%[139,143,144] | … | … |
| EEG | Generalized suppression to <20 μV | FPR 3%, 95% CI 0.9%–11%[139,142,146,147,155–158] | … | … | Generalized suppression to <20 μV | FPR 3%, 95% CI 0.9%–11%[139,142,146,147,155–158] | Status epilepticus | FPR 7%, 95% CI 1%–25% and FPR 11.5%, 95% CI 3%–31%[139,159,160] | … | … |
| | Burst-suppression pattern | … | … | … | Burst-suppression pattern | … | … | … | … | … |
| | Diffuse periodic complexes on a flat background | … | … | … | Diffuse periodic complexes on a flat background | … | Unreactive EEG background | FPR 0%, 95% CI 0%–13%[139,147] | … | … |
| Blood and CSF biochemical markers | BNP, procalcitonin, interleukins, TNF, sTREM-1, CSF-CKBB, S100, NSE, LDH, GOT, neurofilament, acid phosphatase, lactate, pyruvate, T- lymphocyte cell markers | Not recommended: Clinical application limited because cutoff values for PPV vary across studies and because of lack of adjustment for confounders that affect marker performance[139,147,161–174] | … | … | … | … | Glial fibrillary acidic protein, NSE, S100b | Not recommended: Clinical application limited because cutoff values for PPV vary across studies and because of lack of adjustment for confounders that affect marker performance[139,175–178] | … | … |

(Continued)

**Table 8. (Continued)**

| | No Treatment With Therapeutic Hypothermia* | | | | | | Treatment With Therapeutic Hypothermia* | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | ≥24 h | | At 48 h | | ≥72 h | | ≥24 h | | ≥72 h | |
| Diagnostic Tests | Poor Outcome (CPC 3 or 4 or Death) | Reliability | Poor Outcome (CPC 3 or 4 or Death) | Reliability | Poor Outcome (CPC 3 or 4 or Death) | Reliability | Poor Outcome (CPC 3 or 4 or Death) | Reliability | Poor Outcome (CPC 3 or 4 or Death) | Reliability |
| Brain MRI | … | … | … | … | … | … | … | … | … | … |
| Brain CT | … | … | … | … | … | … | … | … | … | … |
| Brain SPECT | Decreased cerebral blood flow, particularly in frontal and temporal lobes, which persists on repeated imaging | … | … | … | … | … | … | … | … | … |
| Cerebral angiography | Delayed cerebral circulation time | … | … | … | … | … | … | … | … | … |
| Transcranial Doppler | Delayed hyperemia | … | … | … | … | … | … | … | … | … |
| Nuclear medicine | Abnormal uptake in cerebral cortices | … | … | … | … | … | … | … | … | … |
| Near-infrared spectroscopy | … | Limitations include lack of comparative standard, nonmodern technique, sample size too small, and lack of uniform interval after arrest and withdrawal of care[139,179–192] | … | … | … | … | … | … | … | … |
| Bispectral index monitoring | … | … | … | … | … | … | Initial score ≥22 | Not recommended: FPR 7%, 95% CI 1%–26%[139,193] | … | … |
| Bispectral index monitoring | … | … | … | … | … | … | Suppression ratio of ≥48 h or level 0 ≤72 h | Not recommended: FPR 0%, 95% CI 0%–27%[139,158] | … | … |
| Clinical decision rule | … | … | … | … | … | … | Between 36 and 72 h, presence of 2: ≥1 absent brainstem reflexes, early myoclonus, unreactive EEG background, and bilaterally absent cortical somatosensory evoked potentials | Not recommended: FPR 0%, 95% CI 0%–14%[139,194] | … | … |

BNP indicates brain natriuretic peptide; CI, confidence interval; CKBB, creatine kinase BB; CPC, cerebral performance categories; CSF, cerebrospinal fluid; CT, computed tomography; EEG, electroencephalographic; ellipses (…), unknown; FPR, false-positive rate; GCS, Glasgow Coma Scale; GOT, glutamate oxaloacetate transaminase; LDH, lactate dehydrogenase; MRI, magnetic resonance imaging; NSE, neuron-specific enolase; PPV, positive predictive value; SPECT, single-photon emission computed tomography; sTREM-1, soluble triggered receptor expressed on myeloid cells-1; and TNF, tumor necrosis factor.

*In the absence of confounders, including hypotension, use of sedatives, or neuromuscular blockers.

**Table 9. Coding Variability for Cardiac Arrest**

| Source | Code | Name |
|---|---|---|
| *ICD-10* | 427.5 | Cardiac arrest |
| *ICD-9* | 99.6 | Cardiopulmonary resuscitation not otherwise specified (CPR) |
| *ICD-9* | 99.63 | Closed chest cardiac massage |
| DRG | 129 | Cardiac arrest, unexplained |
| DRG | 121 | Circulatory disorder with acute myocardial infarction and major complication discharged alive |

CPR indicates cardiopulmonary resuscitation; ICD, *International Statistical Classification of Diseases and Related Health Problems*; and DRG, Diagnosis-Related Group.

a multidisciplinary approach that is resource intensive and depends on a high level of cooperation among physician specialists, nurses, coordinators, and allied healthcare professionals.[107,138] The concept of level 1 cardiac arrest centers has been proposed for postarrest care of OHCA patients,[214] based on the comprehensive systems concept applied successfully to level 1 trauma centers,[215] management of ST-segment–elevation myocardial infarction,[216] and a regional "cardiac center" model with a well-developed integrated transfer system.[217] Improved health outcomes with the use of cardiac arrest centers have not been studied with a randomized design. Large registry and population-based studies have not shown that transport to critical care centers or high-volume sites with PCI capacity was associated with increased survival to discharge in OHCA when adjusted for all other predictors of a favorable outcome.[218,219] When resources are focused on cardiac arrest centers without addressing a system of care for those who have an arrest in a nondesignated center, outcomes from IHCA are unlikely to improve and may actually worsen. It is important for all hospitals to recognize that improved survival of postarrest patients may be associated with patient volume (>50 cases per year).[106] However, if a region redirects all OHCAs to a cardiac arrest center, it means that all nondesignated center hospitals are at risk for skills depreciation and a decline in health outcomes (survival to discharge) for IHCA occurring in these institutions, and the overall survival rate from IHCA in the community may suffer.

Hence, all hospitals should be prepared to initiate optimized post–cardiac arrest care and, where appropriate, to transfer the patient to a higher level of care in a timely and safe manner. This means that where appropriate, a mutual aid system of interhospital transfer must be established in advance to ensure optimized, timely post-IHCA care to all patients, regardless of where patients are when they have an IHCA, and to enable the treating clinician to access this system of care easily and rapidly. In turn, it should be acknowledged that regional cardiac arrest centers provide postarrest care to both OHCA and IHCA patients, who still have very high rates of morbidity and mortality. Caring for a high volume of postarrest patients can negatively influence a performance ranking that is based on rates for PCI, cardiac catheterization laboratory, and ICU mortality. Adjustments should be made when these performance rankings are compared across institutions with or without regional designation as a postarrest center.

## Conclusions

This consensus statement on IHCA provides healthcare providers, clinical leaders, administrators, regulators, and policy makers with an overview of the various issues related to reporting, planning, and performing best practices as related to IHCA. This statement also documents what is unknown about IHCA and what aspects of IHCA need to be changed to advance the care of IHCA. Much of the science behind the current guidelines for IHCA has been extrapolated from OHCA, which may not be appropriate, given the differences in causes and outcomes, team configuration, and access to resources. IHCA lacks uniformity in documentation and reporting, lacks the science to guide some of the therapeutic interventions in this patient population, and is perceived by many to be hopeless, which suggests a cultural impediment to change. Current regulatory and accreditation standards do not include the required incentives nor the mandate for universal reporting, and as a result, the institutional response is inconsistent. Very few registry or population-based options exist to capture the data, and some interventions have good levels of evidence but suffer from lack of adherence to practice guidelines. Much more could be done to improve IHCA care at the level of the provider, the institution, and the healthcare system. This consensus document will guide implementation strategies to assist clinicians in adhering to current practice guidelines when providing care and achieving benefit for patients and the system of healthcare delivery that pertains to IHCA. It identifies the gaps in science and provides justification for future studies. To enable this needed transformation, a list of recommendations is provided to guide institutional leaders, regulatory bodies, and research funding agencies.

## Recommendations

### *Institutional Leaders and Healthcare Providers*

1. Establish and report patient self-determination of care documentation, including explicit DNAR status, as a routine practice in all admissions.
2. Establish competency of all hospital staff in recognizing cardiac arrest, performing chest compressions, and using an AED.
3. Implement best practices across all phases of IHCA care with a continuous quality improvement program.
4. Track and report complete IHCA incidence and survival to hospital discharge, as well as functional outcome at discharge, using universal definitions to ensure that the numerator and denominator are standardized for IHCA across all hospitals.
5. Implement a standardized, evidence-based prognostication approach to prevent premature withdrawal of life-sustaining therapy.
6. Optimize the process for successful organ and tissue recovery after death.

### *Regulatory*

7. Mandate reporting of IHCA incidence and survival as an accreditation benchmark using universal definitions to

ensure a standardized numerator and denominator across all hospitals.
8. Mandate reporting of rates of DNAR status (before index cardiac arrest) per 1000 admissions.
9. Modify ICD coding to collect reliable administrative data on IHCA.

### *Research Funding Agencies*

10. Make all aspects of IHCA care, including but not limited to epidemiology, therapy, education, and implementation issues such as patient safety, team composition, and debriefing, priorities for research funding to address the significant gaps in knowledge.

### *National and International Bodies Setting Resuscitation Guidelines*

11. Consider developing guidelines for OHCA that are separate from those for IHCA so that gaps in knowledge are more readily documented and levels of evidence can be adjusted accordingly.

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*Modest.

†Significant.

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This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (1) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (2) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

## References


1. 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;81:302–311.
2. Field JM, Hazinski MF, Sayre MR, Chameides L, Schexnayder SM, Hemphill R, Samson RA, Kattwinkel J, Berg RA, Bhanji F, Cave DM, Jauch EC, Kudenchuk PJ, Neumar RW, Peberdy MA, Perlman JM, Sinz E, Travers AH, Berg MD, Billi JE, Eigel B, Hickey RW, Kleinman ME, Link MS, Morrison LJ, O'Connor RE, Shuster M, Callaway CW, Cucchiara B, Ferguson JD, Rea TD, Vanden Hoek TL. Part 1: executive summary: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122(suppl 3):S640–S656.
3. The Joint Commission Web site. http://www.jcrinc.com/Joint-Commission-Requirements/Hospitals/. Accessed February 24, 2013.
4. Shepardson LB, Youngner SJ, Speroff T, O'Brien RG, Smyth KA, Rosenthal GE. Variation in the use of do-not-resuscitate orders in patients with stroke. *Arch Intern Med*. 1997;157:1841–1847.
5. Peberdy MA, Kaye W, Ornato JP, Larkin GL, Nadkarni V, Mancini ME, Berg RA, Nichol G, Lane-Trultt T. Cardiopulmonary resuscitation of adults in the hospital: a report of 14720 cardiac arrests from the National Registry of Cardiopulmonary Resuscitation. *Resuscitation*. 2003;58:297–308.
6. Niemann JT, Stratton SJ. The Utstein template and the effect of in-hospital decisions: the impact of do-not-attempt resuscitation status on survival to discharge statistics. *Resuscitation*. 2001;51:233–237.
7. Hodgetts TJ, Kenward G, Vlackonikolis I, Payne S, Castle N, Crouch R, Ineson N, Shaikh L. Incidence, location and reasons for avoidable in-hospital cardiac arrest in a district general hospital. *Resuscitation*. 2002;54:115–123.
8. Jones-Crawford JL, Parish DC, Smith BE, Dane FC. Resuscitation in the hospital: circadian variation of cardiopulmonary arrest. *Am J Med*. 2007;120:158–164.
9. Chan PS, Jain R, Nallmothu BK, Berg RA, Sasson C. Rapid response teams: a systematic review and meta-analysis. *Arch Intern Med*. 2010;170:18–26.
10. Nadkarni VM, Larkin GL, Peberdy MA, Carey SM, Kaye W, Mancini ME, Nichol G, Lane-Truitt T, Potts J, Ornato JP, Berg RA; National Registry of Cardiopulmonary Resuscitation Investigators. First documented rhythm and clinical outcome from in-hospital cardiac arrest among children and adults. *JAMA*. 2006;295:50–57.
11. Elixhauser A. Hospital Stays for Children, 2006. HCUP Statistical Brief #56. Rockville, MD: Agency for Healthcare Research and Quality; July 2008.
12. Merchant RM, Yang L, Becker LB, Berg RA, Nadkarni V, Nichol G, Carr BG, Mitra N, Bradley SM, Abella BS, Groeneveld PW; 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;39:2401–2406.
13. American Heart Association. Get With The Guidelines–Resuscitation Web site. 2011. http://www.heart.org/HEARTORG/HealthcareResearch/GetWithTheGuidelines-Resuscitation/Get-With-The-Guidelines-Resuscitation_UCM_314496_SubHomePage.jsp. Accessed March 16, 2012.
14. Nichol G, Thomas E, Callaway CW, Hedges J, Powell JL, Aufderheide TP, Rea T, Lowe R, Brown T, Dreyer J, Davis D, Idris A, Stiell I; Resuscitation Outcomes Consortium Investigators. Regional variation in out-of-hospital cardiac arrest incidence and outcome [published correction appears in *JAMA*. 2008;300:1763]. *JAMA*. 2008;300:1423–1431.
15. Atkins DL, Everson-Stewart S, Sears GK, Daya M, Osmond MH, Warden CR, Berg RA; Resuscitation Outcomes Consortium Investigators. Epidemiology and outcomes from out-of-hospital cardiac arrest in children: the Resuscitation Outcomes Consortium Epistry-Cardiac Arrest. *Circulation*. 2009;119:1484–1491.
16. Day JC. *Population Projections of the United States, by Age, Sex, Race, and Hispanic Origin: 1992 to 2050*. US Bureau of the Census, Current Population Reports. Washington, DC: US Government Printing Office; 1992:P25–1092.
17. Carr BG, Goyal M, Band RA, Gaieski DF, Abella BS, Merchant RM, Branas CC, Becker LB, Neumar RW. A national analysis of the relationship between hospital factors and post-cardiac arrest mortality. *Intensive Care Med*. 2009;35:505–511.
18. Bloom HL, Shukrullah I, Cuellar JR, Lloyd MS, Dudley SC Jr, Zafari AM. Long-term survival after successful inhospital cardiac arrest resuscitation. *Am Heart J*. 2007;153:831–836.
19. Fiser DH. Assessing the outcome of pediatric intensive care. *J Pediatr*. 1992;121:68–74.
20. Booth CM, Boone RH, Tomlinson G, Detsky AS. Is this patient dead, vegetative, or severely neurologically impaired? Assessing outcome for comatose survivors of cardiac arrest. *JAMA*. 2004;291:870–879.
21. Peberdy MA, Ornato JP, Larkin GL, Braithwaite RS, Kashner TM, Carey SM, Meaney PA, Cen L, Nadkarni VM, Praestgaard AH, Berg RA; National Registry of Cardiopulmonary Resuscitation Investigators. Survival from in-hospital cardiac arrest during nights and weekends. *JAMA*. 2008;299:785–792.
22. Tian J, Kaufman DA, Zarich S, Chan PS, Ong P, Amoateng-Adjepong Y, Manthous CA; American Heart Association National Registry for Cardiopulmonary Resuscitation Investigators. Outcomes of critically ill patients who received cardiopulmonary resuscitation. *Am J Respir Crit Care Med*. 2010;182:501–506.
23. Ehlenbach WJ, Barnato AE, Curtis JR, Kreuter W, Koepsell TD, Deyo RA, Stapleton RD. Epidemiologic study of in-hospital cardiopulmonary resuscitation in the elderly. *N Engl J Med*. 2009;361:22–31.
24. Aufderheide T, Hazinski MF, Nichol G, Steffens SS, Buroker A, McCune R, Stapleton E, Nadkarni V, Potts J, Ramirez RR, Eigel B, Epstein A, Sayre M, Halperin H, Cummins RO. Community lay rescuer automated external defibrillation programs: key state legislative components and implementation strategies: a summary of a decade of experience for healthcare providers, policymakers, legislators, employers, and community leaders from the American Heart Association Emergency Cardiovascular Care Committee, Council on Clinical Cardiology, and Office of State Advocacy. *Circulation*. 2006;113:1260–1270.
25. Abella BS, Edelson DP. Resuscitation errors: a shocking problem. In: WebM&M: Morbidity & Mortality Rounds on the Web. Rockville, MD: Agency for Healthcare Research and Quality; July/August 2007. http://www.webmm.ahrq.gov/case.aspx?caseID=155. Accessed March 16, 2012.
26. Abella BS, Edelson DP, Kim S, Retzer E, Myklebust H, Barry AM, O'Hearn N, Hoek TL, Becker LB. CPR quality improvement during in-hospital cardiac arrest using a real-time audiovisual feedback system. *Resuscitation*. 2007;73:54–61.
27. Neumar RW, Otto CW, Link MS, Kronick SL, Shuster M, Callaway CW, Kudenchuk PJ, Ornato JP, McNally B, Silvers SM, Passman RS, White RD, Hess EP, Tang W, Davis D, Sinz E, Morrison LJ. Part 8: adult advanced cardiovascular life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care [published correction appears in *Circulation*. 2011;123:e236]. *Circulation*. 2010;122(suppl 3):S729–S767.
28. Edelson DP, Litzinger B, Arora V, Walsh D, Kim S, Lauderdale DS, Vanden Hoek TL, Becker LB, Abella BS. Improving in-hospital cardiac arrest process and outcomes with performance debriefing. *Arch Intern Med*. 2008;168:1063–1069.
29. Peberdy MA, Cretikos M, Abella BS, DeVita M, Goldhill D, Kloeck W, Kronick SL, Morrison LJ, Nadkarni VM, Nichol G, Nolan JP, Parr M, Tibballs J, van der Jagt EW, Young L. Recommended guidelines for monitoring, reporting, and conducting research on medical emergency team, outreach, and rapid response systems: an Utstein-style scientific statement: a scientific statement from the International Liaison Committee on Resuscitation (American Heart Association, Australian Resuscitation Council, European Resuscitation Council, Heart and Stroke Foundation of Canada, InterAmerican Heart Foundation, Resuscitation Council of Southern Africa, and the New Zealand Resuscitation Council); the American Heart Association Emergency Cardiovascular Care Committee; the Council on Cardiopulmonary, Perioperative, and Critical Care; and the Interdisciplinary Working Group on Quality of Care and Outcomes Research. *Circulation*. 2007;116:2481–2500.
30. Devita MA, Bellomo R, Hillman K, Kellum J, Rotondi A, Teres D, Auerbach A, Chen WJ, Duncan K, Kenward G, Bell M, Buist M, Chen J, Bion J, Kirby A, Lighthall G, Ovreveit J, Braithwaite RS, Gosbee J, Milbrandt E, Peberdy M, Savitz L, Young L, Harvey M, Galhotra S. Findings of the first consensus conference on medical emergency teams [published correction appears in *Crit Care Med*. 2006;34:3070]. *Crit Care Med*. 2006;34:2463–2478.
31. Bellomo R, Goldsmith D, Uchino S, Buckmaster J, Hart GK, Opdam H, Silvester W, Doolan L, Gutteridge G. A prospective before-and-after trial of a medical emergency team. *Med J Aust*. 2003;179:283–287.
32. Hillman K, Chen J, Cretikos M, Bellomo R, Brown D, Doig G, Finfer S, Flabouris A; MERIT Study Investigators. Introduction of the medical emergency team (MET) system: a cluster-randomised controlled trial [published correction appears in *Lancet*. 2005;366:1164]. *Lancet*. 2005;365:2091–2097.

33. Chan PS, Khalid A, Longmore LS, Berg RA, Kosiborod M, Spertus JA. Hospital-wide code rates and mortality before and after implementation of a rapid response team. *JAMA*. 2008;300:2506–2513.
34. Edelson DP. A weak link in the rapid response system. *Arch Intern Med*. 2010;170:12–13.
35. Accreditation Council for Graduate Medical Education Web site. Common program requirements. http://www.acgme.org/acgmeweb/Portals/0/dh_dutyhoursCommonPR07012007.pdf. Accessed February 24, 2013.
36. Moretti MA, Cesar LA, Nusbacher A, Kern KB, Timerman S, Ramires JA. Advanced cardiac life support training improves long-term survival from in-hospital cardiac arrest. *Resuscitation*. 2007;72:458–465.
37. Hayes CW, Rhee A, Detsky ME, Leblanc VR, Wax RS. Residents feel unprepared and unsupervised as leaders of cardiac arrest teams in teaching hospitals: a survey of internal medicine residents. *Crit Care Med*. 2007;35:1668–1672.
38. 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;81:1556–1561.
39. Yeung J, Meeks R, Edelson D, Gao F, Soar J, Perkins GD. The use of CPR feedback/prompt devices during training and CPR performance: a systematic review. *Resuscitation*. 2009;80:743–751.
40. Wayne DB, Didwania A, Feinglass J, Fudala MJ, Barsuk JH, McGaghie WC. Simulation-based education improves quality of care during cardiac arrest team responses at an academic teaching hospital: a case-control study. *Chest*. 2008;133:56–61.
41. Dane FC, Russell-Lindgren KS, Parish DC, Durham MD, Brown TD. In-hospital resuscitation: association between ACLS training and survival to discharge. *Resuscitation*. 2000;47:83–87.
42. Sanders AB, Berg RA, Burress M, Genova RT, Kern KB, Ewy GA. The efficacy of an ACLS training program for resuscitation from cardiac arrest in a rural community. *Ann Emerg Med*. 1994;23:56–59.
43. Andreatta P, Saxton E, Thompson M, Annich G. Simulation-based mock codes significantly correlate with improved pediatric patient cardiopulmonary arrest survival rates. *Pediatr Crit Care Med*. 2011;12:33–38.
44. Marsch SC, Tschan F, Semmer N, Spychiger M, Breuer M, Hunziker PR. Performance of first responders in simulated cardiac arrests. *Crit Care Med*. 2005;33:963–967.
45. Smith AF, Wood J. Can some in-hospital cardio-respiratory arrests be prevented? A prospective survey. *Resuscitation*. 1998;37:133–137.
46. Winters BD, Pham J, Pronovost PJ. Rapid response teams: walk, don't run. *JAMA*. 2006;296:1645–1647.
47. The Joint Commission announces the 2009 National Patient Safety Goals and requirements. *Jt Comm Perspect*. 2008;28:1, 11–15.
48. Revere A, Eldridge N. National patient safety goals for 2008. *Topics in Patient Safety.* 2008;12:1–4. http://www.patientsafety.gov/TIPS/Docs/TIPS_JanFeb08.pdf. Accessed July 22, 2012.
49. Fuhrmann L, Lippert A, Perner A, Østergaard D. Incidence, staff awareness and mortality of patients at risk on general wards. *Resuscitation*. 2008;77:325–330.
50. Churpek MM, Yuen TC, Huber MT, Park SY, Hall JB, Edelson DP. Predicting cardiac arrest on the wards: a nested case-control study. *Chest*. 2012;141:1170–1176.
51. Lighthall GK, Lippert A. In hospital staff (P), does the use of any specific educational strategies (I) compared with no such strategies (C) improve outcomes (eg, early recognition and rescue of the deteriorating patient at risk of cardiac/respiratory arrest (O)? Worksheet EIT-026A. In: Appendix: evidence-based worksheets. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations and 2010 American Heart Association and American Red Cross International Consensus on First Aid Science With Treatment Recommendations. *Circulation*. 2010;122(suppl 2):S606–S638.
52. Soar J, Perkins GD, Harris S, Nolan J. The immediate life support course. *Resuscitation*. 2003;57:21–26.
53. Smith GB, Poplett N. Impact of attending a 1-day multi-professional course (ALERT) on the knowledge of acute care in trainee doctors. *Resuscitation*. 2004;61:117–122.
54. Spearpoint KG, Gruber PC, Brett SJ. Impact of the Immediate Life Support course on the incidence and outcome of in-hospital cardiac arrest calls: an observational study over 6 years. *Resuscitation*. 2009;80:638–643.
55. Fuhrmann L, Perner A, Klausen TW, Østergaard D, Lippert A. The effect of multi-professional education on the recognition and outcome of patients at risk on general wards. *Resuscitation*. 2009;80:1357–1360.
56. 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;82:845–852.
57. Drew BJ, Califf RM, Funk M, Kaufman ES, Krucoff MW, Laks MM, Macfarlane PW, Sommargren C, Swiryn S, Van Hare GF. Practice standards for electrocardiographic monitoring in hospital settings: an American Heart Association scientific statement from the Councils on Cardiovascular Nursing, Clinical Cardiology, and Cardiovascular Disease in the Young [published correction appears in *Circulation*. 2005;111:378]. *Circulation*. 2004;110:2721–2746.
58. Saleem MA, McClung JA, Aronow WS, Kannam H. Inpatient telemetry does not need to be used in the management of older patients hospitalized with chest pain at low risk for in-hospital coronary events and mortality. *J Gerontol A Biol Sci Med Sci*. 2005;60:605–606.
59. Hollander JE, Sites FD, Pollack CV Jr, Shofer FS. Lack of utility of telemetry monitoring for identification of cardiac death and life-threatening ventricular dysrhythmias in low-risk patients with chest pain. *Ann Emerg Med*. 2004;43:71–76.
60. Troy P, Smyrnios NA, Howell MD. Routine monitoring of critically ill patients. In: Rippe JM, Irwin RS, eds. *Irwin and Rippe's Intensive Care Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2011:258–276.
61. Galvagno SM, Kodali BS. Critical monitoring issues outside the operating room. *Anesthesiol Clin*. 2009;27:141–156.
62. Wright AA, Zhang B, Ray A, Mack JW, Trice E, Balboni T, Mitchell SL, Jackson VA, Block SD, Maciejewski PK, Prigerson HG. Associations between end-of-life discussions, patient mental health, medical care near death, and caregiver bereavement adjustment. *JAMA*. 2008;300:1665–1673.
63. Murphy DJ, Burrows D, Santilli S, Kemp AW, Tenner S, Kreling B, Teno J. The influence of the probability of survival on patients' preferences regarding cardiopulmonary resuscitation. *N Engl J Med*. 1994;330:545–549.
64. Kaldjian LC, Erekson ZD, Haberle TH, Curtis AE, Shinkunas LA, Cannon KT, Forman-Hoffman VL. Code status discussions and goals of care among hospitalised adults. *J Med Ethics*. 2009;35:338–342.
65. Cotter PE, Simon M, Quinn C, O'Keeffe ST. Changing attitudes to cardiopulmonary resuscitation in older people: a 15-year follow-up study. *Age Ageing*. 2009;38:200–205.
66. Morrison LJ, Kierzek G, Diekema DS, Sayre MR, Silvers SM, Idris AH, Mancini ME. Part 3: ethics: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122(suppl 3):S665–S675.
67. Christenson J, Andrusiek D, Everson-Stewart S, Kudenchuk P, Hostler D, Powell J, Callaway CW, Bishop D, Vaillancourt C, Davis D, Aufderheide TP, Idris A, Stouffer JA, Stiell I, Berg R; Resuscitation Outcomes Consortium Investigators. Chest compression fraction determines survival in patients with out-of-hospital ventricular fibrillation. *Circulation*. 2009;120:1241–1247.
68. Abella BS, Sandbo N, Vassilatos P, Alvarado JP, O'Hearn N, Wigder HN, Hoffman P, Tynus K, Vanden Hoek TL, Becker LB. Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest. *Circulation*. 2005;111:428–434.
69. Abella BS, Alvarado JP, Myklebust H, Edelson DP, Barry A, O'Hearn N, Vanden Hoek TL, Becker LB. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. *JAMA*. 2005;293:305–310.
70. Hunt EA, Vera K, Diener-West M, Haggerty JA, Nelson KL, Shaffner DH, Pronovost PJ. Delays and errors in cardiopulmonary resuscitation and defibrillation by pediatric residents during simulated cardiopulmonary arrests. *Resuscitation*. 2009;80:819–825.
71. Bonnemeier H, Olivecrona G, Simonis G, Götberg M, Weitz G, Iblher P, Gerling I, Schunkert H. Automated continuous chest compression for in-hospital cardiopulmonary resuscitation of patients with pulseless electrical activity: a report of five cases. *Int J Cardiol*. 2009;136:e39–e50.
72. Larsen AI, Hjørnevik AS, Ellingsen CL, Nilsen DW. Cardiac arrest with continuous mechanical chest compression during percutaneous coronary intervention: a report on the use of the LUCAS device. *Resuscitation*. 2007;75:454–459.
73. Timerman S, Cardoso LF, Ramires JA, Halperin H. Improved hemodynamic performance with a novel chest compression device during treatment of in-hospital cardiac arrest. *Resuscitation*. 2004;61:273–280.
74. Bonnemeier H, Simonis G, Olivecrona G, Weidtmann B, Götberg M, Weitz G, Gerling I, Strasser R, Frey N. Continuous mechanical chest compression during in-hospital cardiopulmonary resuscitation of patients with pulseless electrical activity. *Resuscitation*. 2011;82:155–159.

75. Friedman FD, Dowler K, Link MS. A public access defibrillation programme in non-inpatient hospital areas. *Resuscitation*. 2006;69:407–411.
76. Gombotz H, Weh B, Mitterndorfer W, Rehak P. In-hospital cardiac resuscitation outside the ICU by nursing staff equipped with automated external defibrillators: the first 500 cases. *Resuscitation*. 2006;70:416–422.
77. Hanefeld C, Lichte C, Mentges-Schröter I, Sirtl C, Mügge A. Hospital-wide first-responder automated external defibrillator programme: 1 year experience. *Resuscitation*. 2005;66:167–170.
78. Zafari AM, Zarter SK, Heggen V, Wilson P, Taylor RA, Reddy K, Backscheider AG, Dudley SC Jr. A program encouraging early defibrillation results in improved in-hospital resuscitation efficacy. *J Am Coll Cardiol*. 2004;44:846–852.
79. Chan PS, Krumholz HM, Spertus JA, Jones PG, Cram P, Berg RA, Peberdy MA, Nadkarni V, Mancini ME, Nallamothu BK; American Heart Association National Registry of Cardiopulmonary Resuscitation (NRCPR) Investigators. Automated external defibrillators and survival after in-hospital cardiac arrest. *JAMA*. 2010;304:2129–2136.
80. Bento AM, Cardoso LF, Timerman S, Moretti MA, Peres ED, de Paiva EF, Ramires JA, Kern KB. Preliminary in-hospital experience with a fully automatic external cardioverter-defibrillator. *Resuscitation*. 2004;63:11–16.
81. Ali B, Bloom H, Veledar E, House D, Norvel R, Dudley SC, Zafari AM. Automated external cardioversion defibrillation monitoring in cardiac arrest: a randomized trial. *Trials*. 2008;9:36.
82. Kramer-Johansen J, Edelson DP, Abella BS, Becker LB, Wik L, Steen PA. Pauses in chest compression and inappropriate shocks: a comparison of manual and semi-automatic defibrillation attempts. *Resuscitation*. 2007;73:212–220.
83. Sugerman NT, Edelson DP, Leary M, Weidman EK, Herzberg DL, Vanden Hoek TL, Becker LB, Abella BS. Rescuer fatigue during actual in-hospital cardiopulmonary resuscitation with audiovisual feedback: a prospective multicenter study. *Resuscitation*. 2009;80:981–984.
84. Wik L, Kramer-Johansen J, Myklebust H, Sørebø H, Svensson L, Fellows B, Steen PA. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. *JAMA*. 2005;293:299–304.
85. Bobrow BJ, Clark LL, Ewy GA, Chikani V, Sanders AB, Berg RA, Richman PB, Kern KB. Minimally interrupted cardiac resuscitation by emergency medical services for out-of-hospital cardiac arrest. *JAMA*. 2008;299:1158–1165.
86. Rea TD, Helbock M, Perry S, Garcia M, Cloyd D, Becker L, Eisenberg M. Increasing use of cardiopulmonary resuscitation during out-of-hospital ventricular fibrillation arrest: survival implications of guideline changes. *Circulation*. 2006;114:2760–2765.
87. Berg RA, Sanders AB, Kern KB, Hilwig RW, Heidenreich JW, Porter ME, Ewy GA. Adverse hemodynamic effects of interrupting chest compressions for rescue breathing during cardiopulmonary resuscitation for ventricular fibrillation cardiac arrest. *Circulation*. 2001;104:2465–2470.
88. Ewy GA, Zuercher M, Hilwig RW, Sanders AB, Berg RA, Otto CW, Hayes MM, Kern KB. Improved neurological outcome with continuous chest compressions compared with 30:2 compressions-to-ventilations cardiopulmonary resuscitation in a realistic swine model of out-of-hospital cardiac arrest. *Circulation*. 2007;116:2525–2530.
89. SOS-KANTO Study Group. Cardiopulmonary resuscitation by bystanders with chest compression only (SOS-KANTO): an observational study. *Lancet*. 2007;369:920–926.
90. Edelson DP, Abella BS, Kramer-Johansen J, Wik L, Myklebust H, Barry AM, Merchant RM, Hoek TL, Steen PA, Becker LB. Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. *Resuscitation*. 2006;71:137–145.
91. Edelson DP, Robertson-Dick BJ, Yuen TC, Eilevstjønn J, Walsh D, Bareis CJ, Vanden Hoek TL, Abella BS. Safety and efficacy of defibrillator charging during ongoing chest compressions: a multi-center study. *Resuscitation*. 2010;81:1521–1526.
92. Aufderheide TP, Sigurdsson G, Pirrallo RG, Yannopoulos D, McKnite S, von Briesen C, Sparks CW, Conrad CJ, Provo TA, Lurie KG. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. *Circulation*. 2004;109:1960–1965.
93. Yannopoulos D, Tang W, Roussos C, Aufderheide TP, Idris AH, Lurie KG. Reducing ventilation frequency during cardiopulmonary resuscitation in a porcine model of cardiac arrest. *Respir Care*. 2005;50:628–635.
94. Kramer-Johansen J, Myklebust H, Wik L, Fellows B, Svensson L, Sørebø H, Steen PA. Quality of out-of-hospital cardiopulmonary resuscitation with real time automated feedback: a prospective interventional study. *Resuscitation*. 2006;71:283–292.
95. Berg RA, Hemphill R, Abella BS, Aufderheide TP, Cave DM, Hazinski MF, Lerner EB, Rea TD, Sayre MR, Swor RA. Part 5: adult basic life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care [published correction appears in *Circulation*. 2011;124:e402]. *Circulation*. 2010;122(18 Suppl 3):S685–S705.
96. Nishisaki A, Nysaether J, Sutton R, Maltese M, Niles D, Donoghue A, Bishnoi R, Helfaer M, Perkins GD, Berg R, Arbogast K, Nadkarni V. Effect of mattress deflection on CPR quality assessment for older children and adolescents. *Resuscitation*. 2009;80:540–545.
97. Perkins GD, Kocierz L, Smith SC, McCulloch RA, Davies RP. Compression feedback devices over estimate chest compression depth when performed on a bed. *Resuscitation*. 2009;80:79–82.
98. Chan PS, Nichol G, Krumholz HM, Spertus JA, Nallamothu BK; American Heart Association National Registry of Cardiopulmonary Resuscitation (NRCPR) Investigators. Hospital variation in time to defibrillation after in-hospital cardiac arrest. *Arch Intern Med*. 2009;169:1265–1273.
99. Chan PS, Krumholz HM, Nichol G, Nallamothu BK; American Heart Association National Registry of Cardiopulmonary Resuscitation Investigators. Delayed time to defibrillation after in-hospital cardiac arrest. *N Engl J Med*. 2008;358:9–17.
100. Perkins GD, Davies RP, Soar J, Thickett DR. The impact of manual defibrillation technique on no-flow time during simulated cardiopulmonary resuscitation. *Resuscitation*. 2007;73:109–114.
101. Vanden Hoek TL, Morrison LJ, Shuster M, Donnino M, Sinz E, Lavonas EJ, Jeejeebhoy FM, Gabrielli A. Part 12: cardiac arrest in special situations: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care [published corrections appear in *Circulation*. 2011;124:e405 and *Circulation*. 2011;123:e239]. *Circulation*. 2010;122(suppl 3):S829–S861.
102. Berg MD, Schexnayder SM, Chameides L, Terry M, Donoghue A, Hickey RW, Berg RA, Sutton RM, Hazinski MF. Part 13: pediatric basic life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122(suppl 3):S862–S875.
103. Kern KB, Sanders AB, Raife J, Milander MM, Otto CW, Ewy GA. A study of chest compression rates during cardiopulmonary resuscitation in humans: the importance of rate-directed chest compressions. *Arch Intern Med*. 1992;152:145–149.
104. Milander MM, Hiscok PS, Sanders AB, Kern KB, Berg RA, Ewy GA. Chest compression and ventilation rates during cardiopulmonary resuscitation: the effects of audible tone guidance. *Acad Emerg Med*. 1995;2:708–713.
105. Hostler D, Everson-Stewart S, Rea TD, Stiell IG, Callaway CW, Kudenchuk PJ, Sears GK, Emerson SS, Nichol G; Resuscitation Outcomes Consortium Investigators. Effect of real-time feedback during cardiopulmonary resuscitation outside hospital: prospective, cluster-randomised trial. *BMJ*. 2011;342:d512.
106. Carr BG, Kahn JM, Merchant RM, Kramer AA, Neumar RW. Inter-hospital variability in post-cardiac arrest mortality. *Resuscitation*. 2009;80:30–34.
107. Neumar RW, Nolan JP, Adrie C, Aibiki M, Berg RA, Böttiger BW, Callaway C, Clark RS, Geocadin RG, Jauch EC, Kern KB, Laurent I, Longstreth WT Jr, Merchant RM, Morley P, Morrison LJ, Nadkarni V, Peberdy MA, Rivers EP, Rodriguez-Nunez A, Sellke FW, Spaulding C, Sunde K, Vanden Hoek T. Post-cardiac arrest syndrome: epidemiology, pathophysiology, treatment, and prognostication: a consensus statement from the International Liaison Committee on Resuscitation (American Heart Association, Australian and New Zealand Council on Resuscitation, European Resuscitation Council, Heart and Stroke Foundation of Canada, InterAmerican Heart Foundation, Resuscitation Council of Asia, and the Resuscitation Council of Southern Africa); the American Heart Association Emergency Cardiovascular Care Committee; the Council on Cardiovascular Surgery and Anesthesia; the Council on Cardiopulmonary, Perioperative, and Critical Care; the Council on Clinical Cardiology; and the Stroke Council. *Circulation*. 2008;118:2452–2483.
108. 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;30:2126–2128.
109. Bernard SA, Gray TW, Buist MD, Jones BM, Silvester W, Gutteridge G, Smith K. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med*. 2002;346:557–563.
110. Hypothermia after Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest [published correction appears in *N Engl J Med*. 2002;346:1756]. *N Engl J Med*. 2002;346:549–556.
111. Peberdy MA, Callaway CW, Neumar RW, Geocadin RG, Zimmerman JL, Donnino M, Gabrielli A, Silvers SM, Zaritsky AL, Merchant

R, Vanden Hoek TL, Kronick SL. Part 9: post-cardiac arrest care: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care [published corrections appear in *Circulation*. 2011;123:e237 and *Circulation*. 2011;124:e403]. *Circulation*. 2010;122(suppl 3):S768–S786.
112. Merchant RM, Abella BS, Khan M, Huang KN, Beiser DG, Neumar RW, Carr BG, Becker LB, Vanden Hoek TL. Cardiac catheterization is underutilized after in-hospital cardiac arrest. *Resuscitation*. 2008;79:398–403.
113. Dumas F, Cariou A, Manzo-Silberman S, Grimaldi D, Vivien B, Rosencher J, Empana JP, Carli P, Mira JP, Jouven X, Spaulding C. Immediate percutaneous coronary intervention is associated with better survival after out-of-hospital cardiac arrest: insights from the PROCAT (Parisian Region Out of hospital Cardiac ArresT) registry. *Circ Cardiovasc Interv*. 2010;3:200–207.
114. Laurent I, Monchi M, Chiche JD, Joly LM, Spaulding C, Bourgeois B, Cariou A, Rozenberg A, Carli P, Weber S, Dhainaut JF. Reversible myocardial dysfunction in survivors of out-of-hospital cardiac arrest. *J Am Coll Cardiol*. 2002;40:2110–2116.
115. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Peterson E, Tomlanovich M; Early Goal-Directed Therapy Collaborative Group. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001;345:1368–1377.
116. Pölönen P, Ruokonen E, Hippeläinen M, Pöyhönen M, Takala J. A prospective, randomized study of goal-oriented hemodynamic therapy in cardiac surgical patients. *Anesth Analg*. 2000;90:1052–1059.
117. Pearse R, Dawson D, Fawcett J, Rhodes A, Grounds RM, Bennett ED. Early goal-directed therapy after major surgery reduces complications and duration of hospital stay: a randomised, controlled trial [ISRCTN38797445]. *Crit Care*. 2005;9:R687–R693.
118. Adrie C, Adib-Conquy M, Laurent I, Monchi M, Vinsonneau C, Fitting C, Fraisse F, Dinh-Xuan AT, Carli P, Spaulding C, Dhainaut JF, Cavaillon JM. Successful cardiopulmonary resuscitation after cardiac arrest as a "sepsis-like" syndrome. *Circulation*. 2002;106:562–568.
119. El Sanadi N. In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of seizure prophylaxis or effective seizure control (I) as opposed to standard care (no prophylaxis or ineffective seizure control)(C), improve outcome (O) (eg. survival)? Worksheet ALS-PA-050A. In: Appendix: evidence-based worksheets. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations and 2010 American Heart Association and American Red Cross International Consensus on First Aid Science With Treatment Recommendations. *Circulation*. 2010;122(suppl 2):S606–S638.
120. Castrén M. In adult and pediatric patients with ROSC after cardiac arrest (P), does the use of seizure prophylaxis or effective seizure control (I) as opposed to standard care (no prophylaxis) (C) improve outcome (O)? Worksheet ALS-PA-050B. In: Appendix: evidence-based worksheets. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations and 2010 American Heart Association and American Red Cross International Consensus on First Aid Science With Treatment Recommendations. *Circulation*. 2010;122(suppl 2):S606–S638.
121. Longstreth WT Jr, Fahrenbruch CE, Olsufka M, Walsh TR, Copass MK, Cobb LA. Randomized clinical trial of magnesium, diazepam, or both after out-of-hospital cardiac arrest. *Neurology*. 2002;59:506–514.
122. Brain Resuscitation Clinical Trial I Study Group. Randomized clinical study of thiopental loading in comatose survivors of cardiac arrest. *N Engl J Med*. 1986;314:397–403.
123. Sunde K, Pytte M, Jacobsen D, Mangschau A, Jensen LP, Smedsrud C, Draegni T, Steen PA. Implementation of a standardised treatment protocol for post resuscitation care after out-of-hospital cardiac arrest. *Resuscitation*. 2007;73:29–39.
124. Krumholz A, Stern BJ, Weiss HD. Outcome from coma after cardiopulmonary resuscitation: relation to seizures and myoclonus. *Neurology*. 1988;38:401–405.
125. Wijdicks EF, Parisi JE, Sharbrough FW. Prognostic value of myoclonus status in comatose survivors of cardiac arrest. *Ann Neurol*. 1994;35:239–243.
126. Hui AC, Cheng C, Lam A, Mok V, Joynt GM. Prognosis following postanoxic myoclonus status epilepticus. *Eur Neurol*. 2005;54:10–13.
127. Beiser DG, Carr GE, Edelson DP, Peberdy MA, Hoek TL. Derangements in blood glucose following initial resuscitation from in-hospital cardiac arrest: a report from the national registry of cardiopulmonary resuscitation. *Resuscitation*. 2009;80:624–630.
128. Losert H, Sterz F, Roine RO, Holzer M, Martens P, Cerchiari E, Tiainen M, Müllner M, Laggner AN, Herkner H, Bischof MG. Strict normoglycaemic blood glucose levels in the therapeutic management of patients within 12h after cardiac arrest might not be necessary. *Resuscitation*. 2008;76:214–220.
129. Nolan JP, Laver SR, Welch CA, Harrison DA, Gupta V, Rowan K. Outcome following admission to UK intensive care units after cardiac arrest: a secondary analysis of the ICNARC Case Mix Programme Database. *Anaesthesia*. 2007;62:1207–1216.
130. Langhelle A, Tyvold SS, Lexow K, Hapnes SA, Sunde K, Steen PA. In-hospital factors associated with improved outcome after out-of-hospital cardiac arrest: a comparison between four regions in Norway. *Resuscitation*. 2003;56:247–263.
131. Müllner M, Sterz F, Binder M, Schreiber W, Deimel A, Laggner AN. Blood glucose concentration after cardiopulmonary resuscitation influences functional neurological recovery in human cardiac arrest survivors. *J Cereb Blood Flow Metab*. 1997;17:430–436.
132. Oksanen T, Skrifvars MB, Varpula T, Kuitunen A, Pettilä V, Nurmi J, Castrén M. Strict versus moderate glucose control after resuscitation from ventricular fibrillation. *Intensive Care Med*. 2007;33:2093–2100.
133. Griesdale DE, de Souza RJ, van Dam RM, Heyland DK, Cook DJ, Malhotra A, Dhaliwal R, Henderson WR, Chittock DR, Finfer S, Talmor D. Intensive insulin therapy and mortality among critically ill patients: a meta-analysis including NICE-SUGAR study data. *CMAJ*. 2009;180:821–827.
134. Wiener RS, Wiener DC, Larson RJ. Benefits and risks of tight glucose control in critically ill adults: a meta-analysis [published correction appears in *JAMA*. 2009;301:936]. *JAMA*. 2008;300:933–944.
135. Finfer S, Chittock DR, Su SY, Blair D, Foster D, Dhingra V, Bellomo R, Cook D, Dodek P, Henderson WR, Hebert PC, Heritier S, Heyland DK, McArthur C, McDonald E, Mitchell I, Myburgh JA, Norton R, Potter J, Robinson BG, Ronco JJ. Intensive versus conventional glucose control in critically ill patients. *N Engl J Med*. 2009;360:1283–1297.
136. Krinsley JS, Grover A. Severe hypoglycemia in critically ill patients: risk factors and outcomes. *Crit Care Med*. 2007;35:2262–2267.
137. Arabi YM, Tamim HM, Rishu AH. Hypoglycemia with intensive insulin therapy in critically ill patients: predisposing factors and association with mortality. *Crit Care Med*. 2009;37:2536–2544.
138. Gaieski DF, Band RA, Abella BS, Neumar RW, Fuchs BD, Kolansky DM, Merchant RM, Carr BG, Becker LB, Maguire C, Klair A, Hylton J, Goyal M. Early goal-directed hemodynamic optimization combined with therapeutic hypothermia in comatose survivors of out-of-hospital cardiac arrest. *Resuscitation*. 2009;80:418–424.
139. Morrison LJ, Deakin CD, Morley PT, Callaway CW, Kerber RE, Kronick SL, Lavonas EJ, Link MS, Neumar RW, Otto CW, Parr M, Shuster M, Sunde K, Peberdy MA, Tang W, Hoek TL, Böttiger BW, Drajer S, Lim SH, Nolan JP; on behalf of the Advanced Life Support Chapter Collaborators. Part 8: advanced life support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation*. 2010;122(suppl 2):S345–S421.
140. Bassetti C, Bomio F, Mathis J, Hess CW. Early prognosis in coma after cardiac arrest: a prospective clinical, electrophysiological, and biochemical study of 60 patients. *J Neurol Neurosurg Psychiatr*. 1996;61:610–615.
141. Edgren E, Hedstrand U, Kelsey S, Sutton-Tyrrell K, Safar P. Assessment of neurological prognosis in comatose survivors of cardiac arrest. BRCT I Study Group. *Lancet*. 1994;343:1055–1059.
142. Zandbergen EG, Hijdra A, Koelman JH, Hart AA, Vos PE, Verbeek MM, de Haan RJ; PROPAC Study Group. Prediction of poor outcome within the first 3 days of postanoxic coma. *Neurology*. 2006;66:62–68.
143. Al Thenayan E, Savard M, Sharpe M, Norton L, Young B. Predictors of poor neurologic outcome after induced mild hypothermia following cardiac arrest. *Neurology*. 2008;71:1535–1537.
144. Tiainen M, Kovala TT, Takkunen OS, Roine RO. Somatosensory and brainstem auditory evoked potentials in cardiac arrest patients treated with hypothermia. *Crit Care Med*. 2005;33:1736–1740.
145. Edgren E, Hedstrand U, Nordin M, Rydin E, Ronquist G. Prediction of outcome after cardiac arrest. *Crit Care Med*. 1987;15:820–825.
146. Young GB, Doig G, Ragazzoni A. Anoxic-ischemic encephalopathy: clinical and electrophysiological associations with outcome. *Neurocrit Care*. 2005;2:159–164.
147. Wijdicks EF, Hijdra A, Young GB, Bassetti CL, Wiebe S; Quality Standards Subcommittee of the American Academy of Neurology. Practice parameter: prediction of outcome in comatose survivors after cardiopulmonary resuscitation (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology*. 2006;67:203–210.

148. Leithner C, Ploner CJ, Hasper D, Storm C. Does hypothermia influence the predictive value of bilateral absent N20 after cardiac arrest? *Neurology*. 2010;74:965–969.
149. Fischer C, Luauté J, Némoz C, Morlet D, Kirkorian G, Mauguière F. Improved prediction of awakening or nonawakening from severe anoxic coma using tree-based classification analysis. *Crit Care Med*. 2006;34:1520–1524.
150. Arnoldus EP, Lammers GJ. Postanoxic coma: good recovery despite myoclonus status. *Ann Neurol*. 1995;38:697–698.
151. Celesia GG, Grigg MM, Ross E. Generalized status myoclonicus in acute anoxic and toxic-metabolic encephalopathies. *Arch Neurol*. 1988;45:781–784.
152. Datta S, Hart GK, Opdam H, Gutteridge G, Archer J. Post-hypoxic myoclonic status: the prognosis is not always hopeless. *Crit Care Resusc*. 2009;11:39–41.
153. English WA, Giffin NJ, Nolan JP. Myoclonus after cardiac arrest: pitfalls in diagnosis and prognosis. *Anaesthesia*. 2009;64:908–911.
154. Morris HR, Howard RS, Brown P. Early myoclonic status and outcome after cardiorespiratory arrest. *J Neurol Neurosurg Psychiatr*. 1998;64:267–268.
155. Zandbergen EG, de Haan RJ, Stoutenbeek CP, Koelman JH, Hijdra A. Systematic review of early prediction of poor outcome in anoxic-ischaemic coma. *Lancet*. 1998;352:1808–1812.
156. Sakurai A, Kinoshita K, Moriya T, Utagawa A, Ebihara T, Furukawa M, Tanjoh K. Reduced effectiveness of hypothermia in patients lacking the wave V in auditory brainstem responses immediately following resuscitation from cardiac arrest. *Resuscitation*. 2006;70:52–58.
157. Shibata S, Imota T, Shigeomi S, Sato W, Enzan K. Use of the bispectral index during the early postresuscitative phase after out-of-hospital cardiac arrest. *J Anesth*. 2005;19:243–246.
158. Stammet P, Werer C, Mertens L, Lorang C, Hemmer M. Bispectral index (BIS) helps predicting bad neurological outcome in comatose survivors after cardiac arrest and induced therapeutic hypothermia. *Resuscitation*. 2009;80:437–442.
159. Rossetti AO, Oddo M, Liaudet L, Kaplan PW. Predictors of awakening from postanoxic status epilepticus after therapeutic hypothermia. *Neurology*. 2009;72:744–749.
160. Rossetti AO, Logroscino G, Liaudet L, Ruffieux C, Ribordy V, Schaller MD, Despland PA, Oddo M. Status epilepticus: an independent outcome predictor after cerebral anoxia. *Neurology*. 2007;69:255–260.
161. Zandbergen EG, de Haan RJ, Hijdra A. Systematic review of prediction of poor outcome in anoxic-ischaemic coma with biochemical markers of brain damage. *Intensive Care Med*. 2001;27:1661–1667.
162. Martens P. Serum neuron-specific enolase as a prognostic marker for irreversible brain damage in comatose cardiac arrest survivors. *Acad Emerg Med*. 1996;3:126–131.
163. Martens P, Raabe A, Johnsson P. Serum S-100 and neuron-specific enolase for prediction of regaining consciousness after global cerebral ischemia. *Stroke*. 1998;29:2363–2366.
164. Roine RO, Somer H, Kaste M, Viinikka L, Karonen SL. Neurological outcome after out-of-hospital cardiac arrest: prediction by cerebrospinal fluid enzyme analysis. *Arch Neurol*. 1989;46:753–756.
165. Mussack T, Biberthaler P, Kanz KG, Wiedemann E, Gippner-Steppert C, Mutschler W, Jochum M. Serum S-100B and interleukin-8 as predictive markers for comparative neurologic outcome analysis of patients after cardiac arrest and severe traumatic brain injury. *Crit Care Med*. 2002;30:2669–2674.
166. Fries M, Kunz D, Gressner AM, Rossaint R, Kuhlen R. Procalcitonin serum levels after out-of-hospital cardiac arrest. *Resuscitation*. 2003;59:105–109.
167. Sodeck GH, Domanovits H, Sterz F, Schillinger M, Losert H, Havel C, Kliegel A, Vlcek M, Frossard M, Laggner AN. Can brain natriuretic peptide predict outcome after cardiac arrest? An observational study. *Resuscitation*. 2007;74:439–445.
168. Geppert A, Zorn G, Delle-Karth G, Koreny M, Siostrzonek P, Heinz G, Huber K. Plasma concentrations of von Willebrand factor and intracellular adhesion molecule-1 for prediction of outcome after successful cardiopulmonary resuscitation. *Crit Care Med*. 2003;31:805–811.
169. Adrie C, Haouache H, Saleh M, Memain N, Laurent I, Thuong M, Darques L, Guerrini P, Monchi M. An underrecognized source of organ donors: patients with brain death after successfully resuscitated cardiac arrest. *Intensive Care Med*. 2008;34:132–137.
170. Adib-Conquy M, Monchi M, Goulenok C, Laurent I, Thuong M, Cavaillon JM, Adrie C. Increased plasma levels of soluble triggering receptor expressed on myeloid cells 1 and procalcitonin after cardiac surgery and cardiac arrest without infection. *Shock*. 2007;28:406–410.
171. Longstreth WT Jr, Clayson KJ, Chandler WL, Sumi SM. Cerebrospinal fluid creatine kinase activity and neurologic recovery after cardiac arrest. *Neurology*. 1984;34:834–837.
172. Kärkelä J, Pasanen M, Kaukinen S, Mörsky P, Harmoinen A. Evaluation of hypoxic brain injury with spinal fluid enzymes, lactate, and pyruvate. *Crit Care Med*. 1992;20:378–386.
173. Clemmensen P, Strandgaard S, Rasmussen S, Grande P. Cerebrospinal fluid creatine kinase isoenzyme BB levels do not predict the clinical outcome in patients unconscious following cardiac resuscitation. *Clin Cardiol*. 1987;10:235–236.
174. Rosén H, Karlsson JE, Rosengren L. CSF levels of neurofilament is a valuable predictor of long-term outcome after cardiac arrest. *J Neurol Sci*. 2004;221:19–24.
175. Kaneko T, Kasaoka S, Miyauchi T, Fujita M, Oda Y, Tsuruta R, Maekawa T. Serum glial fibrillary acidic protein as a predictive biomarker of neurological outcome after cardiac arrest. *Resuscitation*. 2009;80:790–794.
176. Tiainen M, Roine RO, Pettilä V, Takkunen O. Serum neuron-specific enolase and S-100B protein in cardiac arrest patients treated with hypothermia. *Stroke*. 2003;34:2881–2886.
177. Oksanen T, Tiainen M, Skrifvars MB, Varpula T, Kuitunen A, Castrén M, Pettilä V. Predictive power of serum NSE and OHCA score regarding 6-month neurologic outcome after out-of-hospital ventricular fibrillation and therapeutic hypothermia. *Resuscitation*. 2009;80:165–170.
178. Rundgren M, Karlsson T, Nielsen N, Cronberg T, Johnsson P, Friberg H. Neuron specific enolase and S-100B as predictors of outcome after cardiac arrest and induced hypothermia. *Resuscitation*. 2009;80:784–789.
179. Allen JS, Tranel D, Bruss J, Damasio H. Correlations between regional brain volumes and memory performance in anoxia. *J Clin Exp Neuropsychol*. 2006;28:457–476.
180. De Volder AG, Michel C, Guérit JM, Bol A, Georges B, de Barsy T, Laterre C. Brain glucose metabolism in postanoxic syndrome due to cardiac arrest. *Acta Neurol Belg*. 1994;94:183–189.
181. Fujioka M, Nishio K, Miyamoto S, Hiramatsu KI, Sakaki T, Okuchi K, Taoka T, Fujioka S. Hippocampal damage in the human brain after cardiac arrest. *Cerebrovasc Dis*. 2000;10:2–7.
182. Tommasino C, Grana C, Lucignani G, Torri G, Fazio F. Regional cerebral metabolism of glucose in comatose and vegetative state patients. *J Neurosurg Anesthesiol*. 1995;7:109–116.
183. Lovblad K, Senn P, Walpoth B, et al. Increased brain tolerance for ischemia in accidental deep hypothermia and circulatory arrest. *Riv Neuroradiol*. 1998;11(suppl 2):224–226.
184. Zhang YX, Liu JR, Jiang B, Liu HQ, Ding MP, Song SJ, Zhang BR, Zhang H, Xu B, Chen HH, Wang ZJ, Huang JZ. Lance-Adams syndrome: a report of two cases. *J Zhejiang Univ Sci B*. 2007;8:715–720.
185. Choi SP, Park HK, Park KN, Kim YM, Ahn KJ, Choi KH, Lee WJ, Jeong SK. The density ratio of grey to white matter on computed tomography as an early predictor of vegetative state or death after cardiac arrest. *Emerg Med J*. 2008;25:666–669.
186. Tanaka H, Masugata H, Fukunaga R, Mandai K, Sueyoshi K, Abe H. Sequential change of heterogeneous cerebral blood blow patterns after diffuse brain ischemia. *Resuscitation*. 1992;24:273–281.
187. Heckmann JG, Lang CJ, Pfau M, Neundörfer B. Electrocerebral silence with preserved but reduced cortical brain perfusion. *Eur J Emerg Med*. 2003;10:241–243.
188. Roine RO, Launes J, Nikkinen P, Lindroth L, Kaste M. Regional cerebral blood flow after human cardiac arrest: a hexamethylpropyleneamine oxime single photon emission computed tomographic study. *Arch Neurol*. 1991;48:625–629.
189. Arishima H, Ishii H, Kubota T, Maeda H, Shigemori K. Angiographic features of anoxic encephalopathy in the acute phase: a case report [in Japanese]. *No To Shinkei*. 2003;55:977–982.
190. Iida K, Satoh H, Arita K, Nakahara T, Kurisu K, Ohtani M. Delayed hyperemia causing intracranial hypertension after cardiopulmonary resuscitation. *Crit Care Med*. 1997;25:971–976.
191. Hung GU, Lee JD, Lee JK. Bilateral cranial Tc-99m MDP uptake due to hypoxic-ischemic encephalopathy. *Clin Nucl Med*. 2007;32:328–329.
192. Buunk G, van der Hoeven JG, Meinders AE. A comparison of near-infrared spectroscopy and jugular bulb oximetry in comatose patients resuscitated from a cardiac arrest. *Anaesthesia*. 1998;53:13–19.
193. Seder DB, Fraser GL, Robbins T, Libby L, Riker RR. The bispectral index and suppression ratio are very early predictors of neurological outcome during therapeutic hypothermia after cardiac arrest. *Intensive Care Med*. 2010;36:281–288.

194. Rossetti AO, Oddo M, Logroscino G, Kaplan PW. Prognostication after cardiac arrest and hypothermia: a prospective study. *Ann Neurol*. 2010;67:301–307.
195. Geocadin RG, Buitrago MM, Torbey MT, Chandra-Strobos N, Williams MA, Kaplan PW. Neurologic prognosis and withdrawal of life support after resuscitation from cardiac arrest. *Neurology*. 2006;67:105–108.
196. Hemphill JC 3rd, White DB. Clinical nihilism in neuroemergencies. *Emerg Med Clin North Am*. 2009;27:27–37, vii.
197. Chamberlain DA, Hazinski MF; European Resuscitation Council; American Heart Association; Heart and Stroke Foundation of Canada; Australia and New Zealand Resuscitation Council; Resuscitation Council of Southern Africa; Consejo Latino-Americano de Resuscitación. Education in resuscitation. *Resuscitation*. 2003;59:11–43.
198. Chamberlain DA, Hazinski MF; European Resuscitation Council; American Heart Association; Heart and Stroke Foundation of Canada; Resuscitation Council of Southern Africa; Australia and New Zealand Resuscitation Council; Consejo Latino-Americano de Resusucitación. Education in resuscitation: an ILCOR symposium: Utstein Abbey: Stavanger, Norway: June 22-24, 2001. *Circulation*. 2003;108:2575–2594.
199. Evans M. Navigating health change, knowledge, media, and life: can we make it easier to do the right thing? http://www.theopma.com/pastmeetings/publish/meetings-08/08_04_06_M-Evans.shtml. Accessed March 16, 2012.
200. Pathman DE, Konrad TR, Freed GL, Freeman VA, Koch GG. The awareness-to-adherence model of the steps to clinical guideline compliance: the case of pediatric vaccine recommendations. *Med Care*. 1996;34:873–889.
201. Bigham BL, Koprowicz K, Aufderheide TP, Davis DP, Donn S, Powell J, Suffoletto B, Nafziger S, Stouffer J, Idris A, Morrison LJ; ROC Investigators. Delayed prehospital implementation of the 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiac care. *Prehosp Emerg Care*. 2010;14:355–360.
202. Stiell IG, Nichol G, Leroux BG, Rea TD, Ornato JP, Powell J, Christenson J, Callaway CW, Kudenchuk PJ, Aufderheide TP, Idris AH, Daya MR, Wang HE, Morrison LJ, Davis D, Andrusiek D, Stephens S, Cheskes S, Schmicker RH, Fowler R, Vaillancourt C, Hostler D, Zive D, Pirrallo RG, Vilke GM, Sopko G, Weisfeldt M; ROC Investigators. Early versus later rhythm analysis in patients with out-of-hospital cardiac arrest. *N Engl J Med*. 2011;365:787–797.
203. *The International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM)*. http://www.cdc.gov/nchs/icd/icd9cm.htm. Accessed February 24, 2013.
204. Werner RM, Bradlow ET. Relationship between Medicare's hospital compare performance measures and mortality rates [published correction appears in *JAMA*. 2007;297:700]. *JAMA*. 2006;296:2694–2702.
205. Peterson ED, Roe MT, Mulgund J, DeLong ER, Lytle BL, Brindis RG, Smith SC Jr, Pollack CV Jr, Newby LK, Harrington RA, Gibler WB, Ohman EM. Association between hospital process performance and outcomes among patients with acute coronary syndromes. *JAMA*. 2006;295:1912–1920.
206. Lindenauer PK, Remus D, Roman S, Rothberg MB, Benjamin EM, Ma A, Bratzler DW. Public reporting and pay for performance in hospital quality improvement. *N Engl J Med*. 2007;356:486–496.
207. Tu JV, Donovan LR, Lee DS, Wang JT, Austin PC, Alter DA, Ko DT. Effectiveness of public report cards for improving the quality of cardiac care: the EFFECT study: a randomized trial. *JAMA*. 2009;302:2330–2337.
208. Werner RM, Asch DA. The unintended consequences of publicly reporting quality information. *JAMA*. 2005;293:1239–1244.
209. Narins CR, Dozier AM, Ling FS, Zareba W. The influence of public reporting of outcome data on medical decision making by physicians. *Arch Intern Med*. 2005;165:83–87.
210. Cutlip DE, Ho KK, Kuntz RE, Baim DS. Risk assessment for percutaneous coronary intervention: our version of the weather report? *J Am Coll Cardiol*. 2003;42:1896–1899.
211. Holmes DR, Selzer F, Johnston JM, Kelsey SF, Holubkov R, Cohen HA, Williams DO, Detre KM; National Heart, Lung, and Blood Institute Dynamic Registry. Modeling and risk prediction in the current era of interventional cardiology: a report from the National Heart, Lung, and Blood Institute Dynamic Registry. *Circulation*. 2003;107:1871–1876.
212. Matheny ME, Ohno-Machado L, Resnic FS. Discrimination and calibration of mortality risk prediction models in interventional cardiology. *J Biomed Inform*. 2005;38:367–375.
213. Resnic FS, Welt FG. The public health hazards of risk avoidance associated with public reporting of risk-adjusted outcomes in coronary intervention. *J Am Coll Cardiol*. 2009;53:825–830.
214. Nichol G, Aufderheide TP, Eigel B, Neumar RW, Lurie KG, Bufalino VJ, Callaway CW, Menon V, Bass RR, Abella BS, Sayre M, Dougherty CM, Racht EM, Kleinman ME, O'Connor RE, Reilly JP, Ossmann EW, Peterson E; on behalf of the American Heart Association Emergency Cardiovascular Care Committee; Council on Arteriosclerosis, Thrombosis, and Vascular Biology; Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation; Council on Cardiovascular Nursing; Council on Clinical Cardiology; Advocacy Committee; Council on Quality of Care and Outcomes Research. Regional systems of care for out-of-hospital cardiac arrest: a policy statement from the American Heart Association [published correction appears in *Circulation*. 2010;122:e439]. *Circulation*. 2010;121:709–729.
215. MacKenzie EJ, Rivara FP, Jurkovich GJ, Nathens AB, Frey KP, Egleston BL, Salkever DS, Scharfstein DO. A national evaluation of the effect of trauma-center care on mortality. *N Engl J Med*. 2006;354:366–378.
216. Henry TD, Atkins JM, Cunningham MS, Francis GS, Groh WJ, Hong RA, Kern KB, Larson DM, Ohman EM, Ornato JP, Peberdy MA, Rosenberg MJ, Weaver WD. ST-segment elevation myocardial infarction: recommendations on triage of patients to heart attack centers: is it time for a national policy for the treatment of ST-segment elevation myocardial infarction? *J Am Coll Cardiol*. 2006;47:1339–1345.
217. Henry TD, Sharkey SW, Burke MN, Chavez IJ, Graham KJ, Henry CR, Lips DL, Madison JD, Menssen KM, Mooney MR, Newell MC, Pedersen WR, Poulose AK, Traverse JH, Unger BT, Wang YL, Larson DM. A regional system to provide timely access to percutaneous coronary intervention for ST-elevation myocardial infarction. *Circulation*. 2007;116:721–728.
218. Kajino K, Iwami T, Daya M, Nishiuchi T, Hayashi Y, Kitamura T, Irisawa T, Sakai T, Kuwagata Y, Hiraide A, Kishi M, Yamayoshi S. Impact of transport to critical care medical centers on outcomes after out-of-hospital cardiac arrest. *Resuscitation*. 2010;81:549–554.
219. Callaway CW, Schmicker R, Kampmeyer M, Powell J, Rea TD, Daya MR, Aufderheide TP, Davis DP, Rittenberger JC, Idris AH, Nichol G; Resuscitation Outcomes Consortium (ROC) Investigators. Receiving hospital characteristics associated with survival after out-of-hospital cardiac arrest. *Resuscitation*. 2010;81:524–529.

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