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CPR UPDATE

In-hospital Cardiac Arrest: an Update

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ABSTRACT

In-hospital cardiac arrest has not been the focus of intensive research over the recent years. Since the last American Heart Association (AHA) and European Resuscitation Council (ERC) Guidelines in 2010, a number of studies and a consensus statement by AHA have been published concerning possible improvements in the management of in-hospital cardiac arrest, which are herein reviewed.

KEY WORDS: *cardiac arrest; in-hospital cardiac arrest; cardiopulmonary resuscitation; sudden death; basic life support; advanced life support; defibrillation; automatic external defibrillator*

Abbreviations
ACLS = Advanced Cardiac Life Support
AED = automated external defibrillator
AHA = American Heart Association
BLS = Basic Life Support
CPR = cardiopulmonary resuscitation
DNAR = Do not Attempt Resuscitate
ERC = European Resuscitation Council
ICU = intensive care unit
IHCA = in-hospital cardiac arrest
IO = intraosseous
IV = intravenous
OHCA = out-of-hospital cardiac arrest
PCI = percutaneous coronary intervention
PEA = pulseless electric activity
ROSC = return of spontaneous circulation
VF = ventricular fibrillation
VT = ventricular tachycardia

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IN-HOSPITAL CARDIAC ARREST

Data from a number of emergency medical services (EMS) systems suggest that advances in resuscitation care during the past decade have led to higher rates of survival among patients with out-of-hospital cardiac arrest (OHCA).[1-4] In-hospital cardiac arrest (IHCA) has not received the same level of focused research as OHCA. Thus, despite advances in resuscitation care in recent years, it is not clear whether survival and neurologic function after in-hospital cardiac arrest have improved over time. The latest guidelines about the management of cardiac arrest have been published in 2010, both from the American Heart Association (AHA) and the European Resuscitation Council (ERC). Since then, a consensus statement by AHA concerning possible improvements in the management of in-hospital cardiac arrest has been published. In this article, after a quick overview of the Basic Life Support (BLS) and Advanced Cardiac Life Support (ACLS) algorithm, there will be a focus on the key messages of the 2013 AHA Consensus Statement and a brief review of recent studies concerning IHCA.

EPIDEMIOLOGY

Published Estimates of Incidence

Single-institution studies have reported large variations in hospital-wide incidence rates of adult IHCA, ranging from 3.8 to 13.1 per 1000 admissions.[5,6] A systematic review involving >1 million admissions described an incidence of IHCA occurring outside of intensive care units (ICUs) of 3.66 per 1000 adult admissions and 1.14 per 1000 pediatric admissions.[7] Taking into account the fact that 45% of adult arrests and 65% of pediatric arrests occur in ICUs,[8] it is estimated that the total rate of cardiac arrests is approximately 6.65 and 3.26 per 1000 admissions for adults and children, respectively.

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 ICU and

critical care units, or operating room should be counted in, while patients with a Do not Attempt Resuscitate (DNAR) order should be excluded.

Published Estimates of Outcome

Overall survival to hospital discharge has remained essentially unchanged for decades.[9] In a retrospective analysis in the US, it was suggested that there was a 3% increase in in-hospital survival rates among IHCA patients between 2000 and 2004.[10] 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%).[8]

Recommended Definition of Outcome

In order to define the outcome of IHCA, calculating survival to hospital discharge is the minimum standard, while survival to 30 days is preferred. Outcomes should be reported for all admitted patients who are treated with either chest compressions or defibrillation, except those who have a DNAR order before the arrest.

CPR OVERVIEW

The universal Adult Basic Life Support (BLS) Algorithm is presented in Figure 1.[11] When encountering an adult victim of sudden cardiac arrest, the lone rescuer must first recognize that the victim has experienced a cardiac arrest, based on unresponsiveness and lack of normal breathing.[12,13] Agonal gasps are common early after cardiac arrest and should not be confused with normal breathing.[14-17] Pulse detection alone, even when performed by trained rescuers, is often unreliable and time consuming .[18-21] Thereby, rescuers should start cardiopulmonary resuscitation (CPR) immediately if the adult victim is unresponsive and not breathing normally (i.e., only gasping). The directive to "look, listen, and feel for breathing" to aid recognition is no longer recommended. After recognition, the rescuer should immediately activate the emergency response system, get an automated external defibrillator (AED)/ manual defibrillator, if available, and start CPR with chest compressions. If an AED is not close by, the rescuer should proceed directly to CPR. If other rescuers are present, the first rescuer should direct them to activate the emergency response system and get the AED/defibrillator; the first rescuer should start CPR immediately.[11]

Chest Compressions

The prompt initiation of effective chest compressions is imperative and improves the victim's chance of survival. In order to deliver high-quality CPR, rescuers should focus on providing chest compressions of adequate rate (at least 100/

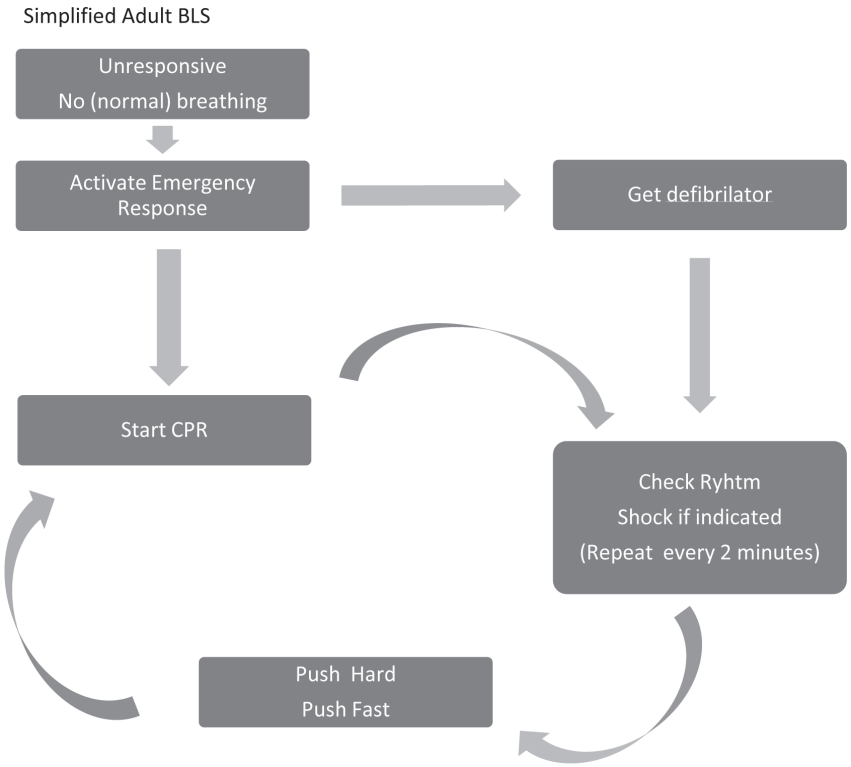

FIGURE 1. Adult BLS Algorithm. Modified from Travers et al.[11]

minute) and depth (at least 2 inches/5 cm for adults or at least one third the anterior-posterior diameter of the chest for infants and children), while allowing complete chest recoil after each compression. Interruptions in compressions should be minimized and excessive ventilation should be avoided.[11]

Airway and Ventilations

Opening the airway with maneuvers such as head tilt– chin lift or jaw thrust, followed by rescue breaths can improve oxygenation and ventilation, but can be technically challenging and require interruptions of chest compressions, particularly for a lone rescuer who has not been trained. Ventilations should be provided if the victim has a high likelihood of an asphyxial cause of the arrest (e.g., infant, child, or drowning victim).

Optimal ventilation is provided via an endotracheal tube; alternatives include the bag-mask or supraglottic airway devices, such as the laryngeal mask airway, esophageal-tracheal combitube and laryngeal tube. Once an advanced airway is in place, providers should deliver ventilations at a regular rate 1 breath every 6 to 8 seconds (8 to 10 breaths/minute) and chest compressions can be delivered without interruption. Multiple rescuers, when available, should rotate the task of compressions every 2 minutes.[11]

Defibrillation

When the AED/defibrillator arrives, the pads should be applied, preferably, without interrupting chest compressions and the AED should be turned "on." The AED will analyze the rhythm and direct the rescuer either to provide a shock (i.e., attempt defibrillation) or to continue CPR. If an AED/defibrillator is not available, continue CPR without interruptions until more experienced rescuers assume care.[11] The victim's chance of survival decreases with an increasing interval between the arrest and defibrillation.[22-23] Thus, early defibrillation remains the cornerstone therapy for ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT). Defibrillation outcome is improved if interruptions (for rhythm assessment, defibrillation, or advanced care) in chest compressions are minimized.[24-32]

OVERVIEW OF THE MANAGEMENT OF CARDIAC ARREST

This section provides an overview of the 2010 ACLS Adult Cardiac Arrest Algorithms and details about the general care of a patient in cardiac arrest (Figure 2). Cardiac arrest can be caused by 4 rhythms: VF, pulseless VT, pulseless electric activity (PEA), and asystole.[33]

Basic Life support

CPR technique, with respect to rate and depth of chest compressions and rate of ventilation with synchronized compression ventilation ratio of 30:2, in the absence of an advanced airway and asynchronous delivery of 1 breath every 6 to 8 seconds –8 to 10 breaths per minute–, after the placement of a supraglottic airway or an endotracheal tube, is the same as in the BLS algorithm. Monitoring and optimizing quality of CPR on the basis of either mechanical parameters, such as chest compression rate and depth, adequacy of relaxation, and minimization of pauses or, when feasible, physiologic parameters, such as partial pressure of end-tidal CO2, arterial pressure during the relaxation phase of chest compressions, or central venous oxygen saturation, are encouraged. Periodic pauses in CPR should be as brief as possible and only as necessary to assess rhythm, shock VF/VT, and perform a pulse check when an organized rhythm is detected, or place an advanced airway.[33]

Rhythm-Based Management of Cardiac Arrest

In addition to high-quality CPR, the only therapy proven to increase survival to hospital discharge is defibrillation of VF/pulseless VT and, therefore considered part of the CPR cycle when the rhythm check reveals VF/pulseless VT. Paddles and electrode pads should be placed on the exposed chest in an anterior-lateral position. Acceptable alternative positions are anterior-posterior, anterior-left infrascapular, and anterior-right infrascapular. Rhythm checks should be brief, and in case an organized rhythm is observed, a pulse check should be performed. If a cardiac monitor is attached to the patient at the time of arrest, the rhythm can be diagnosed before CPR is initiated.

When a rhythm check by an AED reveals VF/VT, the AED will typically prompt to charge and "clear" the victim for shock delivery, which should be performed as quickly as possible. CPR should be resumed immediately after shock delivery (without a rhythm or pulse check and beginning with chest compressions) and continue for 2 minutes before the next rhythm check.[33]

When a rhythm check by a manual defibrillator reveals VF/VT, the first provider should resume CPR while the second provider charges the defibrillator. Once the defibrillator is charged, CPR is paused to "clear" the patient for shock delivery. After the patient is "clear," the second provider gives a single shock as quickly as possible to minimize the interruption in chest compressions ("hands-off interval"). The first provider resumes CPR immediately after shock delivery (again without a rhythm or pulse check and beginning with chest compressions) and continues for 2 minutes. After 2 minutes of CPR the sequence is repeated, beginning with a new rhythm check.[33]

When a rhythm check by an AED reveals a nonshockable rhythm, CPR should be resumed immediately, beginning with chest compressions, and should continue for 2 minutes before the next rhythm check. When a rhythm check using a manual defibrillator or cardiac monitor reveals an organized rhythm, a pulse check is performed. If a pulse is detected, post– cardiac arrest care should be initiated immediately. If the rhythm is asystole or the pulse is absent (e.g., PEA), CPR should be resumed immediately, beginning with chest compressions,

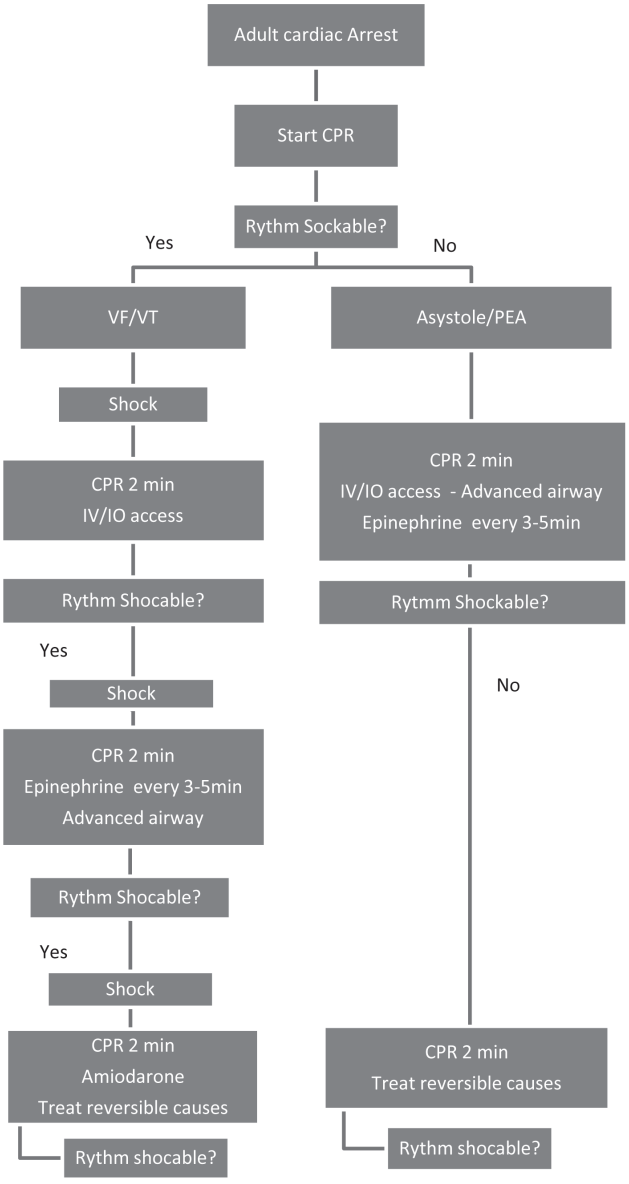

FIGURE 2. ACLS Algorithm. Modified From Neumaret al.[33]

and should continue for 2 minutes before the rhythm check is repeated.[33]

Defibrillation Strategies

If a biphasic defibrillator is available, providers should use the manufacturer's recommended energy dose (e.g., initial dose of 120 to 200 J) for terminating VF (Class I, level of evidence-LOE B). If the provider is unaware of the effective dose range, the provider may use the maximal dose (Class IIb, LOE C). Second and subsequent energy levels should be at least equivalent, and higher energy levels may be considered if available (Class IIb, LOE B). If a monophasic defibrillator is used, providers should deliver an initial shock of 360 J and use that dose for all subsequent shocks. If VF is terminated by a shock but then recurs later in the arrest, deliver subsequent shocks at the previously successful energy level. The benefit of using a multimodal defibrillator in manual instead of automatic mode during cardiac arrest is uncertain (Class IIb, LOE C). Performing CPR while a defibrillator is readied for use is strongly recommended for all patients in cardiac arrest (Class I, LOE B). However, the benefit of delaying defibrillation to perform CPR before defibrillation is unclear (Class IIb, LOE B). Also, the value of VF waveform analysis to guide management of defibrillation in adults with in-hospital and out-of-hospital cardiac arrest is also uncertain (Class IIb, LOE C).[33]

Drug Therapy in Cardiac Arrest

Other ACLS interventions during cardiac arrest may be associated with an increased rate of return of spontaneous circulation (ROSC), but have not yet been proven to increase survival to hospital discharge. Therefore, they are recommended as long as they do not cause significant interruptions in chest compression or delay defibrillation. In order to administer drug therapy during CPR, vascular (intravenous-IV) access should be established, preferably by placing a central line via the subclavian, jugular or femoral vein route; alternative access routes include via the endotracheal tube or via intraosseous injection.

When VF/pulseless VT persists after at least 1 shock and a 2-minute CPR period, a vasopressor can be given with the primary goal of increasing myocardial blood flow during CPR and achieving ROSC (Class IIb, LOE A). The peak effect of an intravenous (IV)/ intraosseous (IO) vasopressor, when given as a bolus dose during CPR, is delayed for at least 1 to 2 minutes. The optimal timing has not been defined. Prompt administration after a shock, could be beneficial if the shock does not generate a rhythm with a palpable pulse, but could have a detrimental effect in the opposite scenario. In the case of PEA/asystole, a vasopressor can be given as soon as possible with the goal of increasing blood flow in the myocardium and the brain, during CPR and achieving ROSC (Class IIb, LOE A). However, routine use of atropine during PEA or asystole is unlikely to have a therapeutic benefit (Class IIb, LOE B) and thus, atropine has been removed from the cardiac arrest algorithm.[33]

Amiodarone is the first-line antiarrhythmic agent given during cardiac arrest in adults with refractory VF/pulseless VT, as it has demonstrated improved rate of ROSC and hospital admission, in this group (Class IIb, LOE A). If amiodarone is unavailable, lidocaine may be considered (Class IIb, LOE B). Magnesium sulfate should be considered only for torsades de pointes associated with a long QT interval (Class IIb, LOE B).[33]

The proposed sequence (order) of drug administration and advanced airway placement during cardiac arrest is not supported by sufficient evidence, and in most cases the timing of

these interventions will depend on factors such as the number of providers participating in the resuscitation, their skill levels and whether vascular access has already been established or an advanced airway has been placed before cardiac arrest.[33]

Treating Potentially Reversible Causes of Cardiac Arrest

Diagnosing and treating the underlying cause is fundamental to management of all cardiac arrest rhythms. During management of cardiac arrest the provider should consider reversible causes of the arrest or factors that may be complicating the resuscitative effort (grouped as the 6 H's and 6 T's) (Table 1). In the case of refractory VF/pulseless VT, cardiac ischemia or acute coronary syndrome should be suspected as a potential etiology. Reperfusion strategies such as coronary angiography and percutaneous coronary intervention (PCI) during CPR or emergency cardiopulmonary bypass have been demonstrated to be feasible in a number of selected cases, but have not been tested in large randomized clinical trials.[34-44] On the other hand, fibrinolytic therapy administered during CPR for acute coronary occlusion does not improve outcome.[45]

Pulseless electrical activity (PEA) is often caused by reversible conditions and during each 2-minute period of CPR the provider should recall the H's and T's to identify factors that may have caused the arrest or may be complicating the resuscitative effort. Due to the association of PEA with hypoxemia, placement of an advanced airway is theoretically more important than during VF/pulseless VT and might be necessary to achieve adequate oxygenation or ventilation. PEA caused by severe volume loss or sepsis will potentially benefit from administration of empirical IV/IO crystalloid, while blood transfusion could be of critical importance for a patient with PEA caused by severe blood loss. When pulmonary embolism is presumed to be the cause of cardiac arrest, empirical fibrinolytic therapy can be considered (Class IIa, LOE B). Finally, clinical suspicion of tension pneumothorax requires needle decompression as initial management. If available, echocardiography can be used to guide management of PEA. Asystole is commonly the end-stage rhythm that follows prolonged VF or PEA, and for this reason the prognosis is generally much worse.[33]

TABLE 1. Reversible Causes[33]

Hypovolemia	Tension Pneumothorax
Hypoxia	Tamponade, cardiac
Hydrogen ion (acidosis)	Toxins
Hypo-/hyperkalemia	Thrombosis, pulmonary
Hypothermia	Thrombosis, coronary
Hypoglycemia	Trauma

It is common for the arrest rhythm to evolve during the course of resuscitation. In such cases treatment algorithm should shift according to the current rhythm, and particular care should be taken to deliver a timely shock when a patient with an initially non-shockable rhythm is found to be in VF/pulseless VT during a repeat rhythm check. There is no evidence that the resuscitation strategy for a new cardiac arrest rhythm should necessarily be altered based on the characteristics of the previous rhythm, unless maximum doses of specific medications have already been administered during earlier resuscitation. Thus, medication doses should be monitored in order to avoid potential toxicity.[33]

Return of Spontaneous Circulation (ROSC) after Cardiac Arrest

If the patient achieves ROSC, post– cardiac arrest care should be immediately initiated to avoid rearrest and optimize the patient's outcome. In case of ROSC after VF/Pulseless VT, treatment of hypoxemia and hypotension, early diagnosis and treatment of ST-elevation myocardial infarction (STEMI) (Class I, LOE B) and therapeutic hypothermia in comatose patients are critical (Class I, LOE B). In the case of ROSC after PEA/asystole, the treatment of hypoxemia and hypotension and early diagnosis and treatment of the underlying cause of cardiac arrest is of particular importance. Therapeutic hypothermia may be considered in this case, when the patient is comatose but its value is less established (Class IIb, LOE C).[33]

MAIN DIFFERENCES BETWEEN AHA AND ERC GUIDELINES

1. Healthcare providers should, "look, listen and feel" for breathing keeping the airway open, in an unresponsive victim of cardiac arrest.
2. When treating VF/VT cardiac arrest, adrenaline 1 mg is given after the third shock once chest compressions have restarted and then every 3–5 min (during alternate cycles of CPR). Amiodarone 300 mg is also given after the third shock.
3. The use of up to 3 sequential shocks may be considered if VF/VT occurs during cardiac catheterization or in the early postoperative period following cardiac surgery. This three-shock strategy may also be considered for an initial, witnessed VF/VT cardiac arrest when the patient is already connected to a manual defibrillator.[45]

STRATEGIES FOR IMPROVING SURVIVAL AFTER IN-HOSPITAL CARDIAC ARREST: 2013 AHA CONSENSUS RECOMMENDATIONS

The goal of this statement was to develop consensus recommendations aimed at measuring and optimizing outcomes after in-hospital cardiac arrest (IHCA).[46]

PREARREST PERIOD

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 (e.g., DNAR status).[46]

Defibrillators and Code Carts: In general, a defibrillator and code cart should be in close proximity to enable defibrillation of any patient in cardiac arrest within 2 minutes. To minimize delays and confusion, it may be advisable to standardize defibrillator equipment across the institution. Code carts should be stocked with the necessary ACLS medications and intubation and respiratory supplies.[46]

Rapid Response Teams: These teams, composed of varying combinations of physicians, nurses, respiratory therapists, and pharmacists, were established to prevent IHCA in patients whose condition is deteriorating.[47,48] These teams 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. 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.

Code Teams: A designated emergency response team (e.g., **code blue** in some hospitals) must be available at all times. Code team composition is defined locally and may consist of nurses, respiratory therapists, pharmacists, physicians, and clergy, as well as security personnel. A code team leader is responsible for guiding the resuscitative efforts. Code team members must be available to respond to codes at all times. 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.[46]

Education and Training: All hospital staff responsible for the care of patients should be trained in basic life support, including 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. One of the more promising training strategies may involve the use of simulation-based mock codes.[49]

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 hospital staff.[50] 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.[51,52]

ECG and Physiological Monitoring: Improved survival to discharge and favorable neurological outcome has been reported with either monitoring or direct observation compared with unmonitored or unwitnessed IHCA,[53] and selected patients should have continuous ECG monitoring.[54]

Plan for Routine Debriefing: It is important to put in place a process for post-event debriefing that best fits the culture of the institution, the resources, and the timing of data capture and analysis.[46]

DNAR Orders: Advance planning by the patient or proxy decision maker is ultimately in the best interest of the patient as these decisions are associated with better care, quality of life, and bereavement adjustment by caregivers.[55] Advance directives should be discussed with and documented for all patients admitted to the hospital.

INTRA-ARREST PERIOD

High-quality CPR, with optimal chest compressions and ventilations, and early defibrillation are cornerstones of intra-arrest treatment that have improved survival from OHCA.[56]

Mechanical Chest Compressions: The use of mechanical chest compression devices (that automatically compress the chest) 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 and PCI.[57] Improved coronary perfusion pressures during IHCA compared with manual chest compressions have been reported with these devices.[58]

Automated External Defibrillators: AEDs may play a role in improving early defibrillation times, particularly in less intensively monitored areas of the hospital, where half of all IHCAs occur.[9] Automated external cardioverter-defibrillators may play a role in more intensively monitored areas of the hospital. These devices provide continuous cardiac monitoring with 2 pads placed on the patient's chest and can automatically defibrillate shockable rhythms, granting a short interval between onset of arrhythmia and first defibrillation as short as 33 seconds in a prospective study.[59]

Decrease Interruptions in Chest Compressions: Interruptions in chest compressions may decrease the compression fraction, which has been associated with decreased survival rates in OHCA.[56] Some out-of-hospital strategies that include continuous compressions without pauses for ventilations have been associated with improved outcomes.[6,60] Newer versions of defibrillator software enable interpretation of the ECG and continuous charging of the capacitor during chest compressions, which minimizes pauses. [61]

Avoid Hyperventilation: Excessive ventilation rates are often observed during CPR for OHCA[62] and IHCA.[63] Fast ventilation rates in the laboratory are associated with increased intrathoracic pressures, lower coronary perfusion pressures, and decreased survival rates.[62,64]

Performance of CPR: A major opportunity for hospitals to improve patient care involves monitoring and improving CPR performance.[65-67] 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.[63] Despite the importance of chest compressions in cardiac arrest outcome, they are rarely performed according to guideline recommendations.[63] In studies of IHCA, chest compression rates were too slow >30% of the time.[63,68] 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.[69]

Optimize Chest Compression Depth and Rate: Greater chest compression depth and a decreased pre-shock pause interval before defibrillation have been associated with increased defibrillation success and ROSC after IHCA.[70] 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.[71,72]

Use Real-Time Feedback: Devices that prompt or time ventilation (e.g., timing lights) and guide rhythm of chest compressions (e.g., metronome) and quality of compressions (e.g., quantitative end-tidal CO_2 waveform capnography) during IHCA may be helpful, but no difference was found in either ROSC or survival to hospital discharge.[17] 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.[71,72]

Provide Early Defibrillation: Approximately 25% of patients with IHCA have a shockable rhythm of pulseless VT/VF.[9] Despite proximity to advanced health care, >30% of patients with IHCA have defibrillation times of >2 minutes after arrest.[73] Defibrillation times longer than 2 minutes after IHCA have been associated with decreased rates of survival to hospital discharge.

Identify and Treat Underlying Causes: The most common causes of IHCA include cardiac arrhythmia, acute respiratory insufficiency, and hypotension.[9] 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,[9] whereas children were more likely to present with asystole (40% versus 35% in adults).[8] Only 10% of patients with IHCA who present with an initial rhythm of PEA or asystole have neurologically intact survival.[9] Thus, identification and treatment of the reversible causes that may present with PEA/asystole are important during IHCA.

POST-ARREST PERIOD

For patients who achieve ROSC, variability in survival rates between hospitals exists and can range from 54% to 32%.[74] Key components include (1) post-arrest brain injury, (2) post-arrest myocardial dysfunction, (3) systemic ischemia/reperfusion response, and (4) persistent acute and chronic pathology that precipitated cardiac arrest.[75] Comprehensive post-arrest care requires access to and collaboration between a multidisciplinary team of providers, including emergency medicine (if the arrest occurs in the emergency department), cardiology, interventional cardiology, cardiac electrophysiology, intensive care, and neurology.[46]

Induction of Goal-Directed Mild Therapeutic Hypothermia: Mild therapeutic hypothermia (32°C to 34°C) improves outcome of comatose survivors of witnessed out-of-hospital cardiac arrest when the initial rhythm is VF.[76,77] Similar studies have not been performed in patients who achieve ROSC after IHCA, or with initial rhythms other than VF.

Coronary Reperfusion for ST-Segment Elevation Myocardial Infarction (STEMI): Percutaneous Coronary Intervention (PCI) for patients resuscitated from IHCA is an important therapeutic consideration. While only 11% of treated IHCAs are caused by acute myocardial infarction,[8] patients who undergo cardiac catheterization are more likely to survive,[78] and 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.

Early Hemodynamic Optimization: Data extrapolated from randomized trials on postoperative patients and those presenting with sepsis,[79-82] emphasize the importance of goal-directed therapy based on hemodynamic monitoring, and it is important to consider the volume status of the patient before IHCA.

Seizure Prophylaxis and Treatment: There are no firm data regarding the issue of seizure prophylaxis in post-IHCA patients, and the incidence of seizures in post-IHCA patients is 5% to 21% with or without therapeutic hypothermia. The 2010 AHA Guidelines for CPR recommend frequent or continuous electroencephalographic monitoring of all comatose survivors of cardiac arrest and advocate prompt and aggressive treatment after the first seizure in post-IHCA patients,[83-84] although post-IHCA seizures may be challenging to treat and respond poorly to anticonvulsant therapies.[85-87]

Glucose Control: Hyperglycemia is likely to develop in the post-IHCA patient, but the optimum blood glucose concentration or intervention is unknown. There is an association of higher glucose concentrations with increased mortality or worse neurological outcomes,[88-92] but strict glucose control (72 to 108 mg/dL) compared with moderate glucose control (108 to 144 mg/dL) in survivors of OHCA presenting with VF and treated with induced hypothermia did not demonstrate a survival benefit at 30 days. Thus, glucose concentrations of 144 to 180 mg/dL are reasonable in adult patients after cardiac arrest, and control of glucose concentration within a lower range (<110 mg/dL) should not be implemented after arrest because of the increased risk of hypoglycemia.[46]

PROCESS ISSUES

Use of a Comprehensive Protocol: the successful implementation of a standardized and comprehensive protocol, addressing the management post-cardiac arrest patients, results in a decrease in in-hospital mortality.[93-94]

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.[9] Guidelines state that there is not a single prognostic test that can safely and adequately predict outcome.[95-96] Thus, 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%.[9,97] These patients can and should be considered for organ donation.

RECENTLY PUBLISHED STUDIES CONCERNING IHCA

EPIDEMIOLOGY

In a recent report by Girotra et al, up to 79.3% of hospitalized patients with cardiac arrest had an initial rhythm of asystole or PEA, and 20.7% had VF or pulseless VT and that proportion increased over time. Risk-adjusted rates of survival to discharge increased from 13.7% in 2000 to 22.3% in 2009 and survival improvement was similar in the two rhythm groups and was due to improvement in both acute resuscitation survival and post-resuscitation survival, while rates of clinically significant neurologic disability among survivors seem to decrease over time.[98]

PREVENTION AND TRAINING

Regardless of whether cardiac arrest was witnessed or unwitnessed, telemetry use was an independent and strong predictor of survival to hospital discharge.[99] Also, the implementation of rapid response team in a single center trial was effective in reducing cardiopulmonary arrests and total hospital mortality for medical ward patients, improving the outcomes of patients who needed ICU admission and reduced readmissions and mortality of patients who were discharged from the ICU.[100] McEvoy et al have shown that adherence to ACLS protocols throughout an event is correlated with increased ROSC in the setting of cardiac arrest and that both wrong actions and omissions of indicated actions lead to decreased ROSC after IHCA.[101] In a Greek study, certified providers had remarkably higher successful resuscitation rates for IHCA than non-certified providers.[102] In addition, Avisar et al pointed out that confidence and CPR skills of preclinical medical students deteriorate significantly within 1 year post-training, reaching an unacceptable level 2 years post-training, thus requiring refresher training at least every year.[103] Moreover, the use of an electronic decision support tool provided a significant improvement in the management of simulated IHCA by senior medical students as measured by adherence to published guidelines.

CPR TECHNIQUE

In a study, mechanical chest compression with the miniaturized chest compressor (MCC) significantly improved calculated Cerebral Perfusion Pressure P but did not compromise intracranial pressure during CPR.[104] On the contrary, there is no clear benefit from active compression-decompression cardiopulmonary resuscitation (CPR) in OHCA or IHCA.[105] Nordsetha et al suggested that the "optimal" first loop duration may be 4 min in initial PEA and 6–8 min in initial asystole, and If secondary PEA/asystole is encountered, 2-min loop duration seems appropriate.[106] Hellevuoa et al reported that the number of iatrogenic injuries in male patients was associated with chest compressions during CPR increased as the measured compression depth exceeded 6 cm. While there is an increased risk of complications with deeper compressions, it is important to realize that the injuries were by and large not fatal.[107]

DEFIBRILLATION

It has been estimated that the correct diagnosis of shockable rythms seems to be greater than the one of nonshockable rhythms (95.2 % vs 88.6 %) and can usually be performed within 4 seconds.[108] In a recent review biphasic waveforms did not seem superior to monophasic ones with respect to VF termination, ROSC, or survival to hospital discharge in OHCA patients with initial VF rhythm under the context of current guidelines. However, most trials were conducted in accordance with previous guidelines for CPR.[109] In a case report of intractable VF that did not respond to typical antidysrhythmics and defibrillation, the simultaneous use of two biphasic defibrillators, at a total of 400 Joules terminated VF and restored a perfusing rhythm.[110]

DRUGS DURING CPR

The latest guidelines state that delivery of drugs via a tracheal tube is no longer recommended and that if intravenous access cannot be achieved, drugs should be given by the intraosseous (IO) route. Newer insertion devices are easy to use, making the intraosseous route an attractive alternative for venous access during a resuscitation event.[111]

In a recent study, a less frequent average epinephrine dosing than recommended by consensus guidelines was associated with improved survival of in-hospital cardiac arrest,[112] while in another, conducted among patients with cardiac arrest requiring vasopressors, combined vasopressin-epinephrine and methylprednisolone during CPR and stress-dose hydrocortisone in postresuscitation shock, compared with epinephrine/saline

placebo, resulted in improved survival to hospital discharge with favorable neurological status.[113]

In a swine model of cardiac arrest, early administration of amiodarone did not improve ROSC or 48-hour survival rates, and was associated with worse haemodynamics.[114] In a study involving children with in-hospital pulseless VT/VF, lidocaine use was independently associated with improved ROSC and 24-hour survival. Amiodarone use was not associated with superior rates of ROSC, survival at 24 hours. Neither drug was associated with survival to hospital discharge.[115]

On the other hand, nitric oxide breathing exerts protective effects after cardiac arrest/CPR even when therapeutic hypothermia is ineffective due to impaired endogenous nitric oxide production,[116] and early neuromuscular blockade for a 24-hour period is associated with an increased probability of survival and an improved lactate clearance.[117]

MONITORING DURING CPR

The integration of cerebral oximetry (regional cerebral oxygen saturation-rSO2) into cardiac arrest resuscitation provides a novel noninvasive method to determine the quality of cerebral perfusion and oxygen delivery to the brain.[118] Cerebral oximetry may have a role as a real-time, non-invasive predictor of ROSC during cardiac arrest. The main utility of rSO2 in determining ROSC appears to apply to asystole and PEA subgroups of cardiac arrest, rather than VF/VT. Furthermore, a persistently low rSO2 <30% in spite of optimal resuscitation methods may indicate futility of resuscitation efforts.[119]

PROGNOSIS

Girotra et al recently described the Cardiac Arrest Survival Post-resuscitation In-hospital (CASPRI) score which includes 11 predictor variables: age, initial cardiac arrest rhythm, defibrillation time, baseline neurological status, duration of resuscitation, mechanical ventilation, renal insufficiency, hepatic insufficiency, sepsis, malignancy, and hypotension. The score is simple to use at the bedside, has excellent discrimination and calibration, and provides robust estimates of the probability of favorable neurological survival after an in-hospital cardiac arrest.[120]

Hypo- and hypercapnia are common after ICU admission post-cardiac arrest and compared with normocapnia, hypocapnia was independently associated with worse clinical outcomes and hypercapnia a greater likelihood of discharge home among survivors. In a study by Lee et al, mean PaO2 had no independent association with in-hospital mortality whereas hypocarbia was independently associated with in-hospital mortality.[121] A V-shaped independent association between the mean PaO2 and poor neurologic outcome at hospital discharge was also noted.[122]

In a study by Chan et al, it was found that 59% of elderly survivors of an in-hospital cardiac arrest were alive at 1 year, and one third were not readmitted to the hospital during that time. Survival and readmission rates differed according to the patients' age, sex, race, and neurologic status at discharge.[123] It is important to note that most elderly patients in Greece, despite their poor knowledge regarding CPR, would like to be resuscitated in case they suffered an in-hospital cardiac arrest, and their attitudes regarding resuscitation are not different from others.[124]

CONCLUSION

While awaiting the updated AHA and ERC guidelines on 2015, it is important to stress the need to implement actions for the prevention and proper management of IHCA. Staff training, appropriate site organization and close monitoring of patients at risk for IHCA remain paramount, along with the need for adherence to the current Guidelines, with respect to high quality uninterrupted CPR and early defibrillation when applicable.

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