

Advances In The Acute Management Of Cardiac Arrest

A 47-year-old man presents with nonspecific chest discomfort intermittently over the past 3 days. Episodes are not related to exertion and last 10 to 30 minutes. He has a history of hypertension and smokes 1 pack per day. In the ED, he is pain free and has an ECG with evidence of left ventricular hypertrophy and j-point elevation. You doubt that he has an acute cardiac syndrome but decide to err on the conservative side and admit him to your observation unit. The patient looks well, his first troponin is negative, and the monitor continues to show a normal sinus rhythm. Two hours later you go to check on the patient and find him disconnected from his monitor, unresponsive, and with no pulse (no wonder there was so much beeping coming from the obs unit). The nurse has been on break for the past 30 minutes, and due to "sick calls" there was no cross coverage. You call for help which doesn't immediately come, and you must decide what is more important — beginning chest compressions, securing the airway, getting intravenous access, or getting the defibrillator. You decide on chest compressions but are not inclined to begin mouth to mouth — you wonder if that is negligence. When the crash cart finally arrives, you note the new biphasic defibrillator and wonder what voltage to start at and if you should "stack" shocks the way you used to. The nurse asks if you want to stop CPR to establish intravenous access and what drugs you want. You begin to realize there is more that you're unsure of than you would like to admit.

Cardiac arrest is the cessation of effective cardiac output as a result of either ventricular asystole, ventricular tachycardia, or ventricular fibrillation (VT/VF): the end result is sudden cardiac death (SCD).¹ Sudden cardiac death describes the unexpected natural death from cardiac cause within 1 hour of onset of symptoms in a person without

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CME Objectives

Upon completion of this article, you should be able to:

1. Identify the significant changes in the 2005 American Heart Association guidelines.
2. Examine the evidence which prompted changes to the American Heart Association guidelines.
3. Indicate future therapies that may impact outcomes from sudden cardiac death.

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any prior condition that appears fatal.²

In 2005, the American Heart Association (AHA) released updated guidelines based on the International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment recommendations.³ These recommendations are based on both experimental data and expert consensus. The new guidelines incorporated significant changes in the algorithms in the treatment of cardiac arrest (**Table 1**). The AHA also identified future areas of research that may impact outcomes in cases of cardiac arrest. These changes include the manner in which CPR is to be carried out with increased emphasis on the continuity of chest compressions with minimal interruptions. This issue of *Emergency Medicine Practice* highlights significant changes in the 2005 AHA guidelines, examines the evidence that prompted the changes, and explores future therapies that may impact outcomes from SCD.

Critical Appraisal Of The Literature

A literature search for articles between 1966 and 2008 was performed using PubMed. Search terms included sudden cardiac death, cardiac arrest, and VT/VF. Both animal and human studies were included. The broad search yielded approximately 4000 articles in addition to the 2005 AHA guidelines for CPR and emergency cardiovascular care. Abstracts were reviewed, and 120 articles were identified, 89 of which are cited.

The closed chest method of CPR was first described by Kouwenhoven et al in a landmark article in 1960.⁵ Due to the nature of the problem of SCD, prospective randomized trials are difficult to conduct.

Even after 48 years, a significant portion of management of SCD is based on animal experiments and expert consensus. However, over the past 15 years an increasing number of evidence-based management strategies were put into practice, as reflected by the most updated AHA guidelines. The classification of AHA recommendations is presented in **Table 2**. In this review, we use the classification system consistent with the AHA and the American College of Cardiology collaboration on evidence-based guidelines.⁶ **Class I** recommendations were based on high-level prospective studies where the benefit substantially outweighs the potential of harm. **Class IIa** recommendations were based on cumulative weight of evidence, and the therapy is considered acceptable and useful.⁶ When a therapy demonstrates only short-term benefit or when a positive result was based on lower level of evidence, a **Class IIb** recommendation was used. For **Class III** therapies, there is evidence and/or general agreement that the procedure/treatment is not useful/effective and in some cases may be harmful. **Class Indeterminate** are therapies for which further research is required.⁶ Generally, **Class I** and **Class IIa** recommendations support standard of care. Deviation from the recommendation should be addressed in a clinical decision making note on the chart.

Epidemiology, Etiology, Pathophysiology

Sudden cardiac death accounts for 300,000 to 400,000 deaths every year in the United States.² The incidence of SCD is 54 to 55 per 100,000 persons.⁷ Rea et al calculated that SCD accounts for 5.6% of the annual mortality in the United States.⁸ Zheng and colleagues reported 63% of all cardiac deaths as

Table 1. Important Changes In The 2005 AHA Guidelines For CPR And Emergency Cardiovascular Care

Measure	2000 Recommendation	2005 Recommendation
Immediate defibrillation for unwitnessed cardiac arrest	Recommended	5 cycles of CPR prior to shock is recommended
Compression: ventilation ratio	15:2	30:2
Sequence of defibrillation	3 stacked shocks	1 shock only followed by immediate CPR
Rhythm/pulse check	After each shock	After 5 cycles of CPR following each shock

Adapted from Ali et al. *Ann Intern Med* 2007;147:171-179.

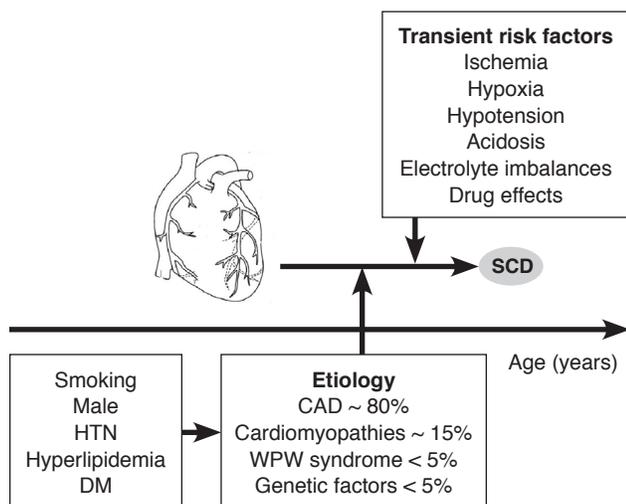
Table 2. The AHA Classification Of Recommendations And Level Of Evidence

Class I	Conditions for which there is evidence and/or general agreement that a given procedure or treatment is useful and effective.
Class II	Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment. IIa. Weight of evidence/opinion is in favor of usefulness/efficacy IIb. Usefulness/efficacy is less well established by evidence/opinion
Class III	Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful/effective and in some cases may be harmful.
Class Indeterminate	Conditions for which there is Insufficient research, continuing area of research, or no recommendation until further research.

SCD.⁹ The proportion of cardiovascular death from SCD has remained constant over the past several years despite the fact that mortality from cardiovascular cause has decreased.¹⁰ This may be due to the infrequency of bystander CPR and the fact that approximately 80% of SCDs occur at home.

The most common etiology for SCD is CAD followed by cardiomyopathies (Figure 1). Together, these cardiovascular diseases account for 95% of SCDs (Table 3). It is important to account for uncommon causes of SCD as they may have treatment implications. These diseases include aortic stenosis, congenital heart disease, Wolff-Parkinson-White (WPW) syndrome, prolonged QT, and Brugada syndrome, a common etiology of SCD in Asian men less than 50 years of age. Acute insults including hypoxia, ischemia, acidosis, electrolyte imbalances, and toxic effects of certain drugs may act on the structural substrate and produce arrhythmias leading to SCD and cardiac arrest.^{11,12} The presenting rhythm in cardiac arrest is variable, with new studies suggesting a decreasing incidence of VT/VF (21%-32%) for cardiac arrest and a higher incidence of asystole and pulseless electrical activity (PEA).¹³⁻¹⁵ In a multicenter, randomized trial (N= 757) studying out-of-hospital cardiac arrest, 31% of subjects presented with an initial rhythm of VT/VF. In another study with a cohort of 783 out-of-hospital cardiac arrest subjects, 22% presented with an initial rhythm of VT/VF.^{13,14} The National Registry of Cardiopulmonary Resuscitation (NRCPR) reported 25% of initial rhythm in 14,720 victims of in-hospital cardiac arrest as VT/VF.¹⁶ A heart in VT/VF is thought to deteriorate

Figure 1. A Confluence Of Risk Factors Act Together To Produce Sudden Cardiac Death



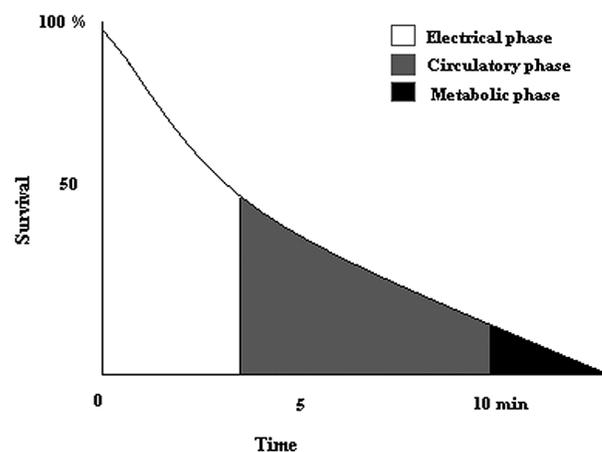
Long-term medical problems (coronary artery disease and cardiomyopathies) produce structural pathology in the myocardium on which transient factors act and trigger ventricular tachycardia and ventricular fibrillation. People with risk factors for coronary artery disease are at high risk for sudden cardiac death.

rate to PEA and asystole with time, conditions which are less responsive to treatment.

The temporal sequence of cardiac arrest can be understood by a 3-phased time sensitive model as proposed by Weisfeldt and Becker (Figure 2).¹⁷ These phases include electrical (lasting 0 to 4 minutes from time of cardiac arrest), circulatory (lasting approximately 4 to 10 minutes from time of cardiac arrest), and metabolic (lasting > 10 minutes from time of cardiac arrest), and they require specific treatments. During the electrical phase, defibrillation is the most effective treatment for cardiac arrest. In the circulatory phase, good quality CPR gains increasing importance along with defibrillation. In the third and final metabolic phase, there is global ischemic injury, where therapeutic strategies that focus on metabolic derangements are critical.¹⁷ Therapeutic hypothermia for comatose survivors of SCD may assist in neurologic recovery at this stage.

Patients with cardiac arrest present both in-hospital and out-of-hospital. The majority of SCDs occur at home and are witnessed by relatives of cardiac arrest victims.¹⁸ In a prospective study of out-of-hospital SCDs conducted in Europe, bystander interviews were conducted by emergency physicians on site after return of spontaneous circulation (ROSC) or death. The study identified 406 cardiac arrest patients out of 5831 rescue missions. In 72% of the cardiac arrest patients, events occurred at home. Of the witnessed cardiac arrest victims, only 14% received bystander resuscitation even though 66% of witnesses were relatives of the victim.¹⁸ Most notably, 55% of SCD victims reported cardiac symptoms 1 hour prior to collapse.¹⁸ These symptoms included chest pain, syncope, and dyspnea. The

Figure 2. Graphic Representation Of The 3-Phase Time Sensitive Model Of Cardiac Arrest



This model predicts 50% survival rate for defibrillation provided in the electrical phase where electrical phase = 0 to 4 minutes, circulatory phase = 4 to 10 minutes, and metabolic phase > 10 minutes (based on the model described by Weisfeldt and Becker. JAMA. 2002).

majority of SCD victims have a known history of either cardiovascular disease (CVD) or cardiac symptoms.¹⁸ However, almost half of the patients will present without any symptoms and will present as unresponsive with no spontaneous respirations or pulse.¹⁸

Differential Diagnosis

Sudden cardiac death occurs in the setting of an acute insult acting most commonly on a pathological structural substrate (Table 4). They include acidosis, acute myocardial infarction, cardiac tamponade, hypoxia, hypovolemia, hyperkalemia, hypokalemia, hypoglycemia, hypothermia, pulmonary embolism, effect of certain toxins or drugs, and tension pneumothorax.^{11,12} During CPR, it is critical for the clinician to seek clues from the medical history and family and to treat for the contributing factors, some of which may be rapidly reversible. Point of care testing can guide the need for treatment of hyperglycemia, hypoglycemia, acidosis, hyperkalemia, or hypokalemia. Bedside sonography when immediately available is increasingly used by trained emergency physicians to check for cardiac activity in PEA/asystole, pericardial effusion, or suspected aortic catastrophe.

Hypoxia, hypovolemia, and hypoglycemia can be rapidly assessed and treated through adequate ventilation, fluid resuscitation, and a finger stick test and dextrose water. If acidosis is suspected, it can be reversed by infusion of sodium bicarbonate solution. Hyperkalemia can cause bradycardic arrest. It may or may not produce the typical ECG features of prolonged PR intervals and peaked T waves (Figure 3). It should be treated with 10 units of regular insulin with glucose in normoglycemic patients. If hyperkalemia is detected prior to cardiac arrest, calcium gluconate, 10 mL in 10% solution over 10 to 20 minutes, should be given to stabilize electrical effects on cardiac myocytes.¹⁹ If hyperkalemia is suspected during cardiac arrest, a much faster rate should be used.

Digitalis toxicity may lead to sustained VT which is characterized by right bundle branch block

Table 3. Etiologies of Sudden Cardiac Death

Etiology	Frequency
Coronary Artery Disease Acute Coronary Syndrome Chronic Myocardial Scar	Approximately 80%
Cardiomyopathies Dilated Cardiomyopathies Hypertrophic Cardiomyopathies	Approximately 10% to 15%
Uncommon Causes Valvular/Congenital Heart Disease Myocarditis, Genetic Ion-Channel Abnormalities, etc.	< 5%

Adapted and modified from Myerberg et al. *Am J Cardiol.* 1997;80:10F-19F and Huikuri HV et al. *N Engl J Med.* 2001;345:1473-1482

configuration and alternating left and right axis deviation (Figure 4). It can be treated with infusion of digoxin Fab fragments.¹⁹ Certain drugs can prolong the QT interval in genetically predisposed individuals. These medications include:¹⁹

- tricyclic antidepressants
- neuroleptics
- macrolide and quinolone antibiotics
- antifungal agents
- procainamide, quinidine, disopyramide (class IA antiarrhythmics)
- sotalol, dofetilide, and ibutilide (class III antiarrhythmics)

In cardiac tamponade, the patient may have symptoms and signs prior to cardiac arrest (e.g. pulses paradoxus, elevated jugular venous pulsation, distant heart sounds, and electrical alternans on ECG). Chest x-ray may show an enlarged heart. If cardiac tamponade is suspected, emergent pericardiocentesis should be performed.¹⁹

Tension pneumothorax may occur in a patient with a history of emphysema and chest wall trauma. Decreased breath sounds on one of side of the chest wall suggests pneumothorax, and in the event of cardiac arrest, it requires immediate decompression.¹⁹

Symptoms consistent with acute myocardial infarction (e.g. angina, dyspnea, diaphoresis) may precede prior to collapse. If acute coronary syndromes and pulmonary embolism are suspected, they should be ruled out after resuscitation.¹⁸ Following ROSC in cardiac arrest, a 12-lead ECG may show ST-segment elevation myocardial infarction (STEMI). It is recommended that survivors of cardiac arrest be considered for emergent percutaneous coronary intervention if another etiology is not obvious.²⁰

Initial Management

Cardiac arrest is an emergency situation in which death can occur within minutes. Factors associated with improved outcomes in cardiac arrest are listed in

Table 4. Contributing Causes Of Cardiac Arrest

The 6 Hs	The 5 Ts
Hypovolemia	Toxins
Hypoxia	Tamponade, cardiac
Hydrogen ion (acidosis)	Tension, pneumothorax
Hypokalemia/Hyperkalemia	Thrombosis (coronary or pulmonary)
Hypothermia	Trauma
Hypoglycemia	

Adapted from 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care, part 7.2: management of cardiac arrest. *Circulation.* 2005;112 (suppl):IV-58-66.

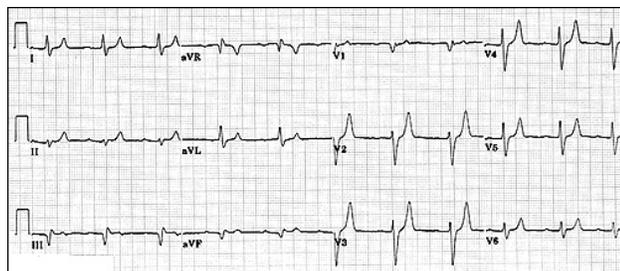
Table 5. The first priority is to check the ABCs following basic cardiovascular life support (BLS) and advanced cardiovascular life support (ACLS) protocols (see *Clinical Pathway on page 10*). During the resuscitative effort and after the patient is stabilized, the underlying etiologies should be continuously explored.

Basic Cardiovascular Life Support

The first step when encountering a victim of cardiac arrest is activation of the emergency response system and immediate initiation of CPR. If the airway is clear, 2 rescue breaths are delivered, and the carotid pulse is checked for no more than 10 seconds (*Class IIa*). If the patient does not have a pulse, cycles of compressions and ventilations should be started at the ratio of 30:2 (*Class IIa*). Chest compressions should allow for complete recoil of the chest and should be at the rate of 100 per minute (*Class IIa*). Each breath should be given for 1 second and should produce visible chest rise (*Class IIa*). The chest is compressed at the center of the nipple line at the approximate depth of 1.5 to 2 inches.²¹ Effective chest compressions are necessary to maintain adequate coronary perfusion (*Class I*). Team leader monitoring of ventilation rate in resuscitation is essential, as it is invariably too fast — even among trained providers. The team leader must also monitor adequacy of compressions. The most effective method to coordinate chest compressions and ventilations and the best compression and ventilation ratio is yet to be determined.

The compression ventilation ratio has been changed from 15:2 to 30:2 to minimize interruptions to chest compressions and to prevent hyperventilation. Animal models have demonstrated that interruptions to chest compressions lead to decreased myocardial blood flow and 24-hour survival.^{22, 23} In a clinical observational study, Aufderheide et al demonstrated an average ventilation rate of 30 ± 3.2 per minute by professional rescuers during CPR in 13 consecutive adults.²⁴ In the second part of the study, ventilation rates of 30 per minute led to high intrathoracic pressures and low coronary perfusion pressures in animal models.²⁴ Similarly,

Figure 3. Hyperkalemia



Example of a patient with hyperkalemia. Note the peaked T waves with a narrow base and the slightly widened QRS complexes. (Reproduced with permission.)

animal models have demonstrated that PaO₂ levels are maintained in the first 14 minutes of cardiac arrest when proper CPR is provided.²³ In contrast, an experimental animal model and a prospective observational study provided support that interruptions to chest compressions decrease the probability of return to spontaneous circulation and low coronary perfusion pressures.^{23,25} When trained health care professionals and BLS-trained subjects were studied, it was shown that rescue breaths interrupted chest compressions for 14 to 16 seconds.^{26, 27}

The efficacy of ventilation in CPR for cardiac arrest victims is not well established. Recently there has been increasing interest in “cardiocerebral resuscitation” which is defined as “chest compression only resuscitation.” In a retrospective study of 135 patients, Kellum et al showed improved survival (20% vs. 57%) and neurological outcomes (15% vs. 48%; P = 0.001) with application of a protocol of cardiocerebral resuscitation in victims of out-of-hospital cardiac arrest with initially shockable rhythms.²⁸

At the very least, a body of evidence supports the critical importance of minimal interruptions during CPR chest compressions. One of the most important factors impacting survival in cardiac arrest is early provision of good quality CPR and early defibrillation when indicated. Stiell et al reported the threefold higher survival rate of 2.98 (95% CI, 2.07-4.29) when CPR was provided by a bystander in an out-of-hospital cardiac arrest.²⁹ In a study based on cardiac arrest victims in Las Vegas casinos, 74% of victims survived to discharge when defibrillation was delivered within 3 minutes as opposed to 49% survival rate when defibrillation was delivered after 3 minutes of downtime (P = 0.02).³⁰ The results from the Swedish cardiac arrest registry demonstrated a 17.4% survival rate at 1 month for patients if CPR was provided within 2 minutes of cardiac arrest vs.

Figure 4. Bidirectional Ventricular Tachycardia Caused By Digitalis Toxicity



Note the right bundle branch block pattern and alternating QRS axis. (Adapted from Kummer JL, Nair R, Krishnan SC. *Circulation*. 2006;113:e156-157)

6.9% if provided after 2 minutes ($P = 0.001$). The authors also reported an odds ratio of 3.5 (95% CI, 2.9-4.3) for survival with bystander CPR.³¹ In the setting of in-hospital cardiac arrest, 38% of patients survived to discharge when defibrillation was provided within 3 minutes vs. 21% when shock was provided after 3 minutes ($P = 0.001$).¹⁶

In a 2008 study, Chan et al studied in-hospital cardiac arrest and the impact of delay in defibrillation on outcome.³² Delay was defined as more than 2 minutes from loss of pulse to defibrillation. The study identified 6789 patients with VT/VF cardiac arrest from 369 hospitals. In 2045 patients (30.1%), defibrillation occurred after 2 minutes. The authors reported that delay resulted in a lower probability of survival to hospital discharge when compared to defibrillation without delay (22% vs. 39.3%, $P = 0.001$).³² In a prospective study involving 193 patients, White et al reported a mean time to shock interval of 5.6 ± 1.5 minutes for survival to discharge for out-of-hospital cardiac arrest and 6.7 ± 1.8 minutes for non-survivors ($P < 0.001$).³³

The second important change in the BLS guidelines distinguishes between witnessed and unwitnessed cardiac arrest. If the cardiac arrest is witnessed and of short duration with an initial rhythm of VT/VF, the patient should be immediately defibrillated (*Class I*). However, if the downtime is unknown, 2 minutes of CPR is recommended prior to defibrillation for a shockable rhythm (*Class IIb*). These changes were prompted by results of 2 important studies.^{34,35} The first study was a prospective observational study in the out-of-hospital VT/VF cardiac arrest setting; when the response time was greater than 4 minutes, the victims benefited from 90 seconds of CPR prior to defibrillation.³⁴ The “CPR first” group had a 27% survival rate vs. 17% when shock was delivered prior to CPR ($P = 0.01$). The “CPR first” group also showed improved neurological outcomes regardless of response time.³⁴ These findings were confirmed by Wik and colleagues in a randomized trial.³⁵ They studied out-of-hospital VT/VF cardiac arrest victims, with the first group receiving 3 minutes of CPR prior to defibrillation and the second group receiving immediate defibrillation. In patients with a response time of ≥ 5 minutes, the “CPR first” group had a 22% survival to discharge

Table 5. Factors Associated With Improved Outcomes in Cardiac Arrest

- Presenting rhythm of VT/VF
- Early/bystander CPR
- Early defibrillation
- CPR prior to defibrillation in the circulatory phase of cardiac arrest
- Minimal interruptions to chest compressions
- In-hospital and out-of-hospital use of AEDs
- Amiodarone use in shock-resistant VT/VF
- Therapeutic hypothermia in comatose cardiac arrest victims

vs. a 4% survival to discharge for the “defibrillation first” group ($P = 0.006$). This survival difference was also confirmed at 1 year (20% vs. 4%, $P = 0.01$).³⁵ Conversely, no difference in survival was observed in a prospective randomized trial involving 256 patients of out-of-hospital VT/VF cardiac arrest in which one group received 90 seconds of CPR prior to shock and the other group received immediate defibrillation.³⁶ Therefore, the current guidelines use permissive language while recommending CPR first in an unobserved cardiac arrest.

The most recent guidelines recommend a single shock protocol in BLS instead of the previously recommended protocol using 3 stacked shocks. There is no evidence that the protocol utilizing 1 shock is better than 3 stacked shocks in the management of VT/VF cardiac arrest. However, there is evidence that the 1-shock protocol may lead to better quality of CPR. Studies have demonstrated that the protocol using 3 stacked shocks results in unacceptably prolonged interruptions in the delivery of CPR.³⁷⁻³⁹ Probability of ROSC decreases if there is an interruption of CPR for > 20 seconds.²⁵ In a study by Van Alem et al, the mean delay for the resumption of CPR was 40 seconds after the first shock, and in none of the 123 patients was CPR resumed within 20 seconds.³⁷ Another study described a “hands off” interval of 19 to 25 seconds between shocks with no CPR.³⁸ Berg and colleagues described a mean delay of 38 seconds for resumption of post-shock CPR.³⁹ The interruption of chest compressions during CPR leads to poor outcomes. In addition, interrupted chest compressions during CPR for rhythm analysis correlated with low arterial pressures, low coronary perfusion, and decreased ejection fraction.^{22,40} Analysis of heart rhythms have shown that the initial rhythm after defibrillation is either asystole or some other non-perfusing rhythm approximately 60% of the time.³⁶⁻⁴⁰ Therefore, immediate CPR after delivery of the first shock is advocated in the current recommendations (*Class IIa*). The first shock should be followed by 2 minutes of CPR which comprises of 5 cycles of CPR at a ratio of 30:2 chest compressions to ventilations. Only then should the rhythm be analyzed. If an organized rhythm is seen, then the pulse is checked; otherwise, CPR is continued.

Monophasic And Biphasic Waveforms

Currently, defibrillation devices use 2 kinds of waveforms: monophasic and biphasic (**Table 6**). A successful defibrillation is defined as absence of VT/VF at 5 seconds after shock delivery.⁴¹ Even though there is a lack of clear-cut evidence for the superiority of biphasic devices in terms of survival, use of biphasic devices is increasing in prevalence. Direction of current is unidirectional in monophasic waveforms and bidirectional in biphasic waveforms, with typical energy levels of 200 to 360 J for monophasic devices and 120 to 200 J for biphasic devices. Weaver et al demonstrated a higher incidence of atrioventricular block when

repeated shocks of high energy monophasic waveforms were delivered.⁴² In a prospective randomized trial comparing efficacy of monophasic and biphasic waveforms, first shock success rates were 96% for biphasic devices compared to 54% to 77% for monophasic devices.⁴³ Similarly, another study demonstrated a higher first shock success rate with biphasic devices compared to monophasic devices (98% vs. 69%; $P = 0.0001$), and the authors described better neurological status for patients defibrillated with biphasic devices (87% vs. 53%).⁴⁴ The authors failed to demonstrate the mechanism of improved cerebral outcomes, and a recent study comparing monophasic and biphasic devices demonstrated neither higher first shock success rates nor improved cerebral outcomes for biphasic devices.⁴⁵ None of the studies comparing the 2 waveforms demonstrated any advantage in terms of ROSC or survival rates for any one waveform. There is no strong evidence backing the increased use of biphasic devices. Replacement of obsolete defibrillators is favoring the biphasic defibrillators, the theory being that lower joules with equivalent efficacy save more myocardium.

In the interest of applicability, the AHA guidelines recommend a dose of 360 J in a non-escalating manner when a monophasic device is used.⁴⁶ The appropriate energy level for biphasic waveforms is device-specific, typically 120 J with a rectilinear waveform and 150 to 200 J for a truncated exponential waveform. Subsequent shocks may be given at the same or higher energy level (**Class IIa**).⁴⁶ CPR should be continued until the defibrillator is charged and the patient is cleared for shock delivery.

Automated External Defibrillators

AEDs are simple, safe, and effective devices designed to be used by both medical professionals and lay people during CPR. There is evidence that use of AEDs leads to early defibrillation and better survival in both in-hospital and out-of-hospital cardiac arrest. When the use of "CPR+AED" in public areas by the general public was compared to CPR by general public and AED use by emergency medical services only,

the "CPR+AED" group not only had shorter time for initial rhythm assessment (6.0 ± 4.7 minutes vs. 8.7 ± 5.5 minutes; $P = 0.001$) but also doubled the number of patients (30 vs. 15; $P = 0.03$) surviving to hospital discharge.⁴⁷ In the case of in-hospital cardiac arrest, a single site study encouraging early defibrillation with use of AEDs demonstrated a 2.6-fold increase in survival to discharge from 4.9% to 12.8%; $P = 0.001$.⁴⁸

Advanced Cardiovascular Life Support

BLS measures have more impact on survival in cardiac arrest when compared to measures of advanced cardiovascular support. The Ontario Prehospital Advanced Life Support (OPALS) study was designed as a multicenter, prospective, observational study and compared outcomes for 4247 patients of out-of-hospital cardiac arrest who received prehospital ACLS vs. a historical cohort of 1391 patients who received only prehospital BLS and defibrillation. The study showed that even though institution of ACLS increased the number of people admitted alive to the hospital (10.9% vs. 14.6%; $P < 0.001$), no advantage was present if survival to hospital discharge was taken into account (5.0% vs. 5.1%; $P = 0.83$).⁴⁹ High quality CPR and rapid defibrillation are the most important measures in the management of cardiac arrest.

Administration Of Medications During CPR

Traditionally, medications have been administered via intravenous or endotracheal route during CPR. It is a common practice in hospitals to attempt placing a central venous access line emergently. However, there is limited evidence of superiority of central venous access over peripheral venous access during CPR. In addition, there is potential of interruption of CPR during attempted placement of a central access line which is clearly detrimental for the patient.⁵⁰ Use of central venous access leads to higher peak drug levels and earlier attainment of effective drug levels compared to peripheral access, but this advantage may not be present for femoral access lines.⁵¹⁻⁵³

If intravenous access cannot be established, drugs may be administered through an intraosseous route or through the endotracheal tube. New devices for rapid intraosseous access in adults using a drill can be placed promptly.⁵⁰ This has often been an access route in pediatric CPR but appears to be gaining favor in adults as well when traditional access cannot be achieved.⁵⁰ Epinephrine and atropine are among the drugs that can be given via the endotracheal route.⁵⁰ The endotracheal route requires higher concentrations of epinephrine than the intravenous route.⁵⁴ In a retrospective study comparing the outcomes of endotracheal vs. intravenous drug administration in out-of-hospital cardiac arrest patients, Niemann et al found that none of the patients who received drugs via the endotracheal tube survived to discharge.⁵⁵ Endotracheal drugs should only be administered if no intravenous access is available.

Table 6. Comparison Of Monophasic And Biphasic Waveforms

	Monophasic	Biphasic
Current direction	Unidirectional	Bidirectional
Typical energy level	200 J to 300 J	120 J to 200 J
First shock success rates*	90% to 95%	60% to 90%
Higher rates of ROSC	Not demonstrated	Not demonstrated
Survival benefit	Not demonstrated	Not demonstrated
AV nodal block	Demonstrated with repeated high energy shocks	Not demonstrated

Rates obtained from Martens PR et al Resuscitation 2001 and Carpenter J et al Resuscitation 2003

Peripheral access is preferable over central, and intravenous access is preferable over endotracheal for drug administration during CPR.

Role Of Medications During CPR

Epinephrine: Use of epinephrine in cardiopulmonary resuscitation dates back to more than a century.⁵⁶ Epinephrine is an α - and β -receptor agonist. The main benefit during CPR is derived from increased peripheral vascular resistance via the stimulation of α -receptors of the blood vessels. This results in the effective redistribution of blood flow from visceral organs to the heart and brain.^{57, 58} There is a possible detrimental effect of β -receptor stimulation by causing increased metabolic demand in the heart.

The appropriate dosing regimen of epinephrine has been subject to some controversy, with experimental data in animal models showing benefit for higher doses. In a double blind, prospective study in France involving 536 patients with out-of-hospital cardiac arrest, with one group receiving standard 1 mg of epinephrine and the second group receiving 5 mg of epinephrine, no statistically significant differences were observed in the ROSC as well as in short-term and long-term survival rates.⁶⁰ This study was followed by a larger, multi-center, prospective, and double-blind study involving 3327 patients suffering out-of-hospital cardiac arrest.⁶¹ Patients were randomized to a standard group receiving 1 mg of epinephrine and a high-dose group receiving 5 mg of epinephrine for up to a total of 15 doses given at 3-minute intervals along with standard CPR. Even though the high-dose group achieved a higher rate of ROSC and higher hospital admission rate, only a small number of patients survived to hospital discharge. In fact, the high-dose group had a higher in-hospital mortality rate during the first 24 hours. There was no statistically significant difference in the rates of discharge from hospital or cerebral performance at discharge in survivors between the groups.⁶¹

Currently, 1 mg of epinephrine intravenously is recommended in a concentration of 1:10,000 every 3 to 5 minutes in order to resuscitate victims of all forms of cardiac arrest (*Class IIb*).⁵⁰

Vasopressin: Another area of controversy is the role of vasopressin in CPR. Vasopressin causes peripheral vasoconstriction by acting on vasopressin receptors and bypassing the adrenergic system. It has a longer half-life than epinephrine, approximately 20 minutes, and has the ability to act in an acidic environment in contrast to epinephrine.^{62, 63}

Use of vasopressin was initially established by a small study involving 40 patients in which 40 IU of vasopressin showed better ROSC and better survival in the first 24 hours in out-of-hospital cardiac arrest caused by VT/VF; thus, vasopressin 40 IU was recommended in the management of refractory VT/VF in the 2000 AHA guidelines.^{64,65} This small study was

followed by 2 larger prospective studies.^{66,67} The first study (N = 200) compared 40 IU of vasopressin with 1 mg of epinephrine in cardiac arrest victims regardless of presenting rhythm.⁶⁶ This study did not find any difference in ROSC, survival, and neurological outcomes between the 2 groups. The second study involved 1219 patients and compared 1 mg of epinephrine to 40 IU of vasopressin in out-of-hospital cardiac arrest patients.⁶⁷ This study did not show significant differences in hospital admission and survival rates between the groups. Nonetheless, if presenting rhythms were considered, asystolic patients in the vasopressin group showed a higher rate of hospital admission (29% vs. 20.3%; P = 0.02) and survival to discharge (4.7% vs. 1.5%; P = 0.04) compared to the epinephrine group. Patients in asystole who received additional epinephrine along with vasopressin showed a higher rate of hospital admission (22.5% vs. 13.3%; P = 0.02) and survival to discharge (3.8% vs. 0; P = 0.008) when compared to patients who received repeated doses of epinephrine alone.⁶⁶ However, there was a disturbingly high incidence of cerebral dysfunction in patients who were discharged after asystole. A retrospective analysis of 298 out-of-hospital cardiac arrests patients showed better pulse return with the combination of epinephrine and vasopressin (40% vs. 13%; P = 0.008) and better ROSC (33% vs. 8.7%; P = 0.004) when the presenting rhythm was asystole.⁶³ However, the authors did not describe any improved outcomes in terms of survival.⁶³ Therefore, even though use of vasopressin is allowed by the current AHA guidelines, there is limited evidence of superiority of vasopressin over epinephrine in any form of cardiac arrest. There is no compelling reason to prefer vasopressin over epinephrine, and further trials need to be conducted to further define the role of vasopressin, particularly in asystolic cardiac arrest.

Currently, vasopressin 40 IU intravenously is recommended as an alternative to epinephrine for refractory VT/VF as well as PEA/asystole when intravenous or intraosseous access is established (*Class Indeterminate*).⁵⁰

Atropine: Experimental evidence for the efficacy of atropine in cardiac arrest is limited. One mg of atropine intravenously, every 3 to 5 minutes (maximum dose 3 mg), is recommended for use in asystole and slow PEA along with epinephrine and vasopressin (*Class Indeterminate*). Atropine is an acetylcholine receptor antagonist of the muscarinic type. Parasympathetic stimulation of the heart results in negative inotropic and chronotropic effects, and atropine is used to block the parasympathetic effect on the heart.⁶⁸

A small prospective study involving 21 patients did not show any advantage of atropine in patients with out-of-hospital cardiac arrest.⁶⁹ In contrast, a retrospective study of refractory asystole showed an advantage of atropine in out-of-hospital cardiac arrest.⁷⁰

In the latter study, the atropine group had a higher number of patients admitted alive to the emergency department but none survived to hospital discharge.⁷⁰

Amiodarone: Amiodarone is the first-line antiarrhythmic for shock refractory VT. In contrast to lidocaine, efficacy of amiodarone to convert VT/VF rhythms to perfusing rhythms in cardiac arrest has been established by 2 prospective randomized trials. The first trial was a randomized, double blind, placebo-controlled study involving 504 eligible patients. This study compared 300 mg of amiodarone to placebo in patients with out-of-hospital cardiac arrest due to ventricular fibrillation. Amiodarone was administered when CPR, 3 shocks, and epinephrine failed to convert the rhythm. Amiodarone showed a higher rate of successful resuscitation and admission to hospital than placebo, odds ratio 1.6 (95% CI, 1.1-2.4; P = 0.02).⁷¹ The study was underpowered to detect differences in survival to hospital discharge. The Amiodarone vs. Lidocaine in Prehospital Ventricular Fibrillation Evaluation (ALIVE) trial compared amiodarone 5 mg/kg to lidocaine 1.5 mg/kg for shock-resistant ventricular fibrillation.⁷² This study enrolled 347 patients and demonstrated superiority of amiodarone over lidocaine in terms of survival to hospital admission (22.8 % vs. 12 %; P = 0.009); this superiority was present regardless of presenting rhythm and was demonstrated over an extended period of time. The lidocaine group had a higher incidence of asystole after defibrillation, following the study-drug delivery.⁷² Neither of the 2 trials showed long-term survival benefit for amiodarone.

Polysorbate 80 and benzoyl alcohol are used as diluents for amiodarone, which may cause hypotension during the resuscitative efforts.⁷³ However, the incidence of hypotension was not statistically significant when compared to lidocaine in the ALIVE trial. In addition, studies by the amino-aqueous investigators on the aqueous formulation of amiodarone did not show any increase in incidence of hypotension. The added advantage of aqueous formulation of amiodarone is that it can be infused rapidly compared to the standard formulation.^{73,74}

Amiodarone 300 mg is used intravenously when CPR, 3 shocks, and vasopressors have failed to convert the rhythm in cardiac arrest (**Class IIIb**). The **Class IIIb** recommendation is indicative of the short-term benefit of amiodarone.⁶ The initial dose can be followed by a second dose of 150 mg.

Lidocaine: Use of lidocaine in shock refractory VF/VT is not established by any clinical evidence. It should not be used as a first-line antiarrhythmic agent during the management of cardiac arrest (**Class Indeterminate**). Lidocaine may increase the incidence of asystole in cardiac arrest due to ventricular arrhythmias.^{72,73}

Magnesium Sulfate: One to 2 g of magnesium sulfate diluted in 10 mL of dextrose water is used to treat VT/VF presenting as *torsades de pointes* during CPR (**Class IIa**). *Torsades de pointes* is a polymorphic VT associated with a prolonged QT interval. The evidence to support this indication is very limited, and there are no randomized trials to support it. Despite the paucity of evidence, the AHA has chosen to make it a **Class IIa** recommendation.⁵⁰ In a small study, Tzivoni et al terminated VT in 11 out of 12 patients by using boluses of 2 g magnesium sulfate followed by continuous infusion of 3 to 20 g/minute.⁷⁶

Post-Resuscitative Care

In patients with return of spontaneous circulation (ROSC) with CPR, the objectives of post-resuscitative care include optimization of hemodynamic, respiratory, and neurologic support as well as identification and treatment of reversible causes of cardiac arrest, temperature regulation, and control of metabolic abnormalities.⁷⁷

Metabolic causes of cardiac arrest like hypovolemia, hypoxia, acidosis, hypokalemia, hyperkalemia, hypoglycemia, and hypothermia must be treated as soon as possible as they are reversible.⁷⁷ Other treatable causes include tension pneumothorax, cardiac tamponade, pulmonary embolism, myocardial infarction, and toxins.⁷⁷

Hypothermia

Moderate hypothermia after cardiac arrest due to ventricular fibrillation is a promising therapy that can be instituted in the intensive care unit. Two randomized, prospective clinical trials have shown improved survival and cerebral performance when therapeutic hypothermia was initiated in comatose out-of-hospital cardiac arrest patients after admission to the hospital.^{78,79} The hypothermia after cardiac arrest study showed 41% mortality after the application of mild hypothermia (target temperature 32°C to 34°C) vs. 55% for standard of care, odds ratio for mortality: 0.74 (95% CI, 0.58-0.95; P = 0.02). The study also demonstrated a statistically significant favorable neurological outcome for the hypothermia group.⁷⁸ Bernard et al also showed a 49% survival to discharge with good neurological outcomes vs. 26% for standard of care in out-of-hospital VT/VF cardiac arrest when cardiac arrest patients were cooled to a target temperature of 33°C for 12 hours (P = 0.046). The odds ratio for a good outcome in the hypothermia group compared to the normothermia group was 5.25 (95% CI, 1.47-18.76; P = 0.011).⁷⁹ A protocol of therapeutic hypothermia with a target temperature of 33°C can successfully be implemented in intensive care units for comatose cardiac arrest patients with major benefit in patient outcome (**Class IIa**).^{77,80}

Core temperature should be monitored continuously and the patient can be externally cooled for 12 to 24 hours. Rewarming should be passive.⁸⁰ Hypo-

thermic intervention may impact long-term survival as patients with severe neurological disability have poor long-term outcomes. Hypothermia may prevent neurological damage by decreasing the metabolic rate of neurons and by preventing reperfusion injury.^{78, 79}

Avoidance Of Hyperthermia

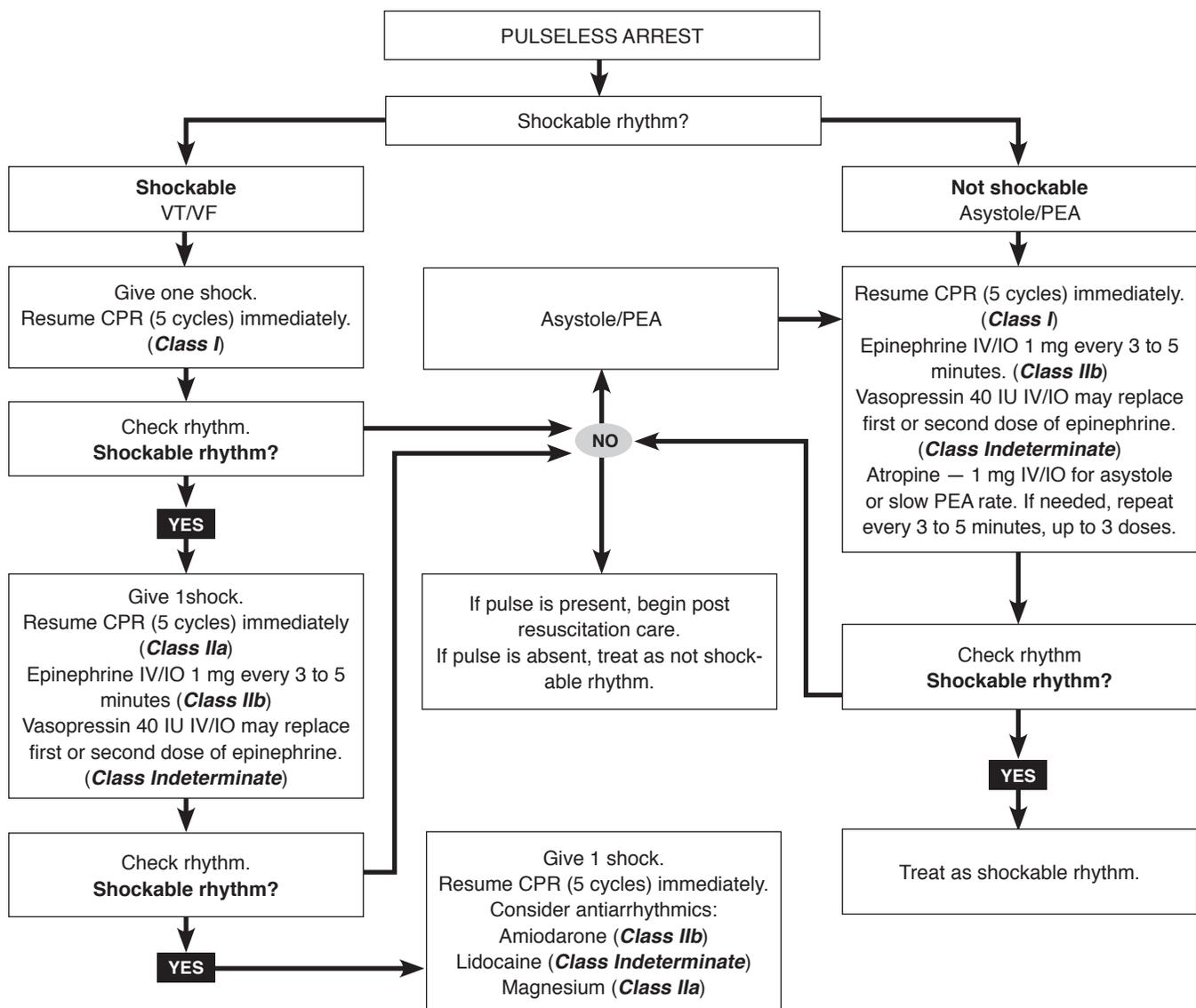
Hyperthermia after successful resuscitation in cardiac arrest is associated with poor outcomes. In cardiac

arrest patients in intensive care units, Zeiner et al demonstrated that the risk of poor neurological outcome increases 2.26 times (95% CI, 1.24-4.12) for each degree above 37°C. Thus, hyperthermia should be treated promptly during post-resuscitative care.⁸¹

Control of Blood Glucose Levels

There is no specific evidence suggesting that glycemic control is as beneficial in cardiac arrest

Clinical Pathway for Advanced Cardiovascular Life Support Pulseless Arrest



See Table 2 on page 2 for class of evidence definitions.

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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Adapted and modified from 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care, part 7.2: management of cardiac arrest. *Circulation*. 2005;112(suppl):IV-58-66

victims as it is in critically ill patients in intensive care units.⁸² Maintaining strict glycemic control by keeping the blood glucose level between 80 and 110 mg per deciliter reduces mortality and morbidity in critically ill patients. Blood glucose levels of cardiac arrest patients in intensive care units should be monitored every 1 to 4 hours, and elevated blood glucose levels may be treated with an insulin infusion (*Class Indeterminate*).^{77,82}

Future Therapies

Therapy With Epinephrine And Vasopressin

Combined use of epinephrine and vasopressin in cardiac arrest patients has shown improved outcomes, especially when the presenting rhythm is asystole.

Further clinical trials evaluating this measure are necessary to confirm potential benefits before this approach can become a recommended step.⁸³

Cardiocerebral Resuscitation

In the past few years, there has been increasing evidence for a paradigm shift in resuscitation science, which is the implementation of cardiocerebral resuscitation in the management of cardiac arrest. Cardiocerebral resuscitation is the protocol of continuous chest compressions without any interruptions for ventilation.^{84,85} It is an attractive idea because the majority of cases of cardiac arrest in the out-of-hospital setting are caused by a cardiovascular event and less likely by acute respiratory compromise. Early bystander-initiated CPR is an independent

Risk Management Pitfalls For Cardiac Arrest Cases

Survival of the majority of cardiac arrest victims depends on the basic interventions of CPR and early delivery of electrical therapy rather than any of the currently applied or applicable advanced measures. Hypothermia is the only advanced intervention that has shown any survival benefit.

- **Don't forget to activate the emergency response system.** Activation of the emergency response system both in-hospital and out-of-hospital will ensure timely arrival of trained personnel and a defibrillator. CPR should begin immediately after the activation of the emergency response system. Bystander CPR is an independent predictor of survival.
- **Make sure to check the carotid pulse for at least but no longer than 10 seconds.** This recommendation is not for lay people and is only for health care providers. Be careful not to take too long to check for carotid pulse. When pulse is not present, initiate chest compressions.
- **Make sure chest compressions are hard and fast.** Chest compression rates should be 100 per minute with avoidance of interruptions, which are common mistakes of health care providers.
- **Avoid rescuer fatigue.** Rescuers performing chest compressions should be frequently rotated. This will help to maintain continuous and fast chest compressions, which will ensure adequate cerebral and coronary perfusion pressures.
- **Allow chest wall to recoil completely.** Complete recoil increases negative intrathoracic pressure and facilitates venous return to the heart during CPR.
- **Attempt peripheral venous access prior to central access.** There is no evidence that central venous access is superior to peripheral venous access in terms of outcomes. Peripheral venous access should be attempted first as it does not interrupt CPR. If central access is attempted, make sure there is minimal interruption to CPR.
- **Do not hyperventilate the patient.** Even trained health care professionals can hyperventilate the patient during CPR. Hyperventilation may actually be harmful to the patient by impeding venous return to the heart because of high intrathoracic pressure.
- **Give 2 minutes of CPR prior to defibrillation if you suspect that the duration of cardiac arrest is longer than 4 to 5 minutes.** If the duration of cardiac arrest is greater than 4 minutes, the patient most likely is in the circulatory phase of cardiac arrest. At this time, circulatory support in the form of good quality CPR becomes as important as defibrillation. Hearts that are well perfused are more likely to respond to a defibrillatory shock.
- **Begin CPR immediately after each shock.** Cardiopulmonary resuscitation must resume immediately after each shock, as the heart is most likely in a non-perfusing rhythm in the first few seconds after defibrillation. Check the rhythm after 2 minutes of CPR; with identification of an organized rhythm, check the pulse. There is no evidence that chest compressions induce arrhythmias.
- **Use amiodarone as your first line antiarrhythmic drug.** Amiodarone is the only antiarrhythmic drug to show potential benefit in randomized clinical trials in the setting of shock refractory cardiac arrest. Lidocaine should not be used as a first-line agent.

Table 7. Patients Eligible For Therapeutic Hypothermia After Cardiac Arrest

- Presents with a VT/VF rhythm
- Is 18 years of age or older
- Has restoration of spontaneous circulation after CPR
- Is comatose and/or non-responsive to verbal commands after ROSC
- Has presumed cardiac origin of arrest
- Has no evidence of causes of coma other than cardiac arrest (e.g. drug overdose, CVA, head trauma)

predictor of survival in cardiac arrest, and a protocol of cardiocerebral resuscitation has shown an increased likelihood of bystander-initiated CPR.^{86, 87} In addition, the new protocol would lead to decreased interruptions for ventilation and could result in better quality of chest compressions, leading to more continuous perfusion of the heart and the brain.

A protocol utilizing 200 uninterrupted chest compressions prior to defibrillation lead to increased survival from 20% to 57% and increased neurologically intact survivor rate from 15% to 48% when compared to a historical cohort.³¹ However, no randomized clinical trial data supporting cardiocerebral resuscitation are currently available.

At this time, cardiocerebral resuscitation or compression-only CPR has been approved by AHA for out-of-hospital cardiac arrest when the bystander is not confident in providing ventilation; this approach is not recommended for medical teams.⁸⁸

Therapeutic Hypothermia

Even though there is strong evidence for induced hypothermia, it is not currently being widely implemented. Induced hypothermia has the potential to play a critical role in the post-resuscitative care of a small subset of cardiac arrest victims. Increased awareness about and induction of therapeutic hypothermia in select patients could translate into better outcomes with ACLS. Benefit of induced hypothermia was observed in a select subset of patients

who were initially comatose but hemodynamically stable after a witnessed cardiac arrest with VF (Table 7).^{78,79} In the Hypothermia After Cardiac Arrest (HACA) study, only 8% of cardiac arrest victims were eligible to receive induced hypothermia.⁷⁸ Similar therapy may be beneficial for patients with non-VF arrest in the out-of-hospital or in-hospital settings.⁷⁷ Further experimental studies and clinical trials are required to determine optimal methods of cooling and optimal timing, duration, and intensity of cooling in order to achieve a measurable impact on CPR outcomes.^{87, 89}

Education Of Cardiovascular Patients And Their Families

The majority of SCDs occur at home and are witnessed by relatives of cardiac arrest victims. In such cases, cardiac arrest victims do not receive adequate CPR from their relatives. See Table 8 for some of the most common CPR errors. Notably, 55% of SCD victims report cardiac symptoms 1 hour prior to collapse.¹⁸ The majority of SCD victims have a known history of either cardiovascular disease or cardiac symptoms. Educating relatives of patients with CVD in basic CPR may be an effective strategy to improve outcomes in out-of-hospital cardiac arrest.¹⁸

Summary

Early initiation of CPR and defibrillation are the most effective measures with the highest impact on survival in patients suffering cardiac arrest. Increased public awareness is required, as witnessed arrests and bystander CPR are positive predictors of outcomes in out-of-hospital cardiac arrest. Cardiopulmonary resuscitation must be performed with a compression to ventilation ratio of 30:2, with minimal interruptions, and delivery of rescue breaths taking no more than 1 second. Basic BLS interventions take precedence over ACLS, as the latter is of limited efficacy. CPR must be resumed immediately after each shock for 5 cycles. Amiodarone is the only

Table 8. Common Errors in CPR Performance

Error	Remedy	Pathophysiology
Inadequate chest compression rates during CPR	Ensure chest compressions at a rate of 100/minute	Higher rates maintains Cerebral Perfusion Pressure (CPP) and coronary perfusion
Incomplete recoil of the chest wall during chest compressions	Allow for complete recoil	Negative intrathoracic pressures allows for better venous return to the heart
Prolonged interruptions to chest compressions for ventilation and central access placement	Minimize interruptions to chest compressions; obtain peripheral access	Interruptions to chest compressions results in low Cerebral Perfusion Pressure (CPP) and coronary perfusion
Hyperventilation of the cardiac arrest victims	30:2 compression:ventilation ratio; give rescue breath for 1 second	Higher intrathoracic pressures impede venous return to the heart
Failure to resume CPR after each shock	Immediately resume CPR after each shock for 5 cycles only, then check the rhythm and pulse	Heart may be in a non-perfusing rhythm immediately after defibrillation

antiarrhythmic with proven efficacy for the restoration of an organized rhythm in cardiac arrest. Benefit of lidocaine in restoring an organized rhythm is not yet established. Automated external defibrillators are simple, safe, and effective devices designed to be used by the general public, first responders, and hospital staff to convert VT/VF to perfusing rhythms in cardiac arrest patients. Educating cardiovascular patients and their families to recognize symptoms preceding SCD, in order to call for help when symptoms are present and to provide CPR when collapse occurs, are important steps to improve outcomes. High quality post-resuscitative care is an important component of management of cardiac arrest with emphasis on treatment of reversible causes and metabolic conditions. Therapeutic hypothermia is effective in a select subset of cardiac arrest patients.

Case Conclusion

Cardiopulmonary resuscitation was immediately initiated. As the downtime was not known, the patient received 2 minutes of CPR, with a chest compression and ventilation ratio of 30:2. While CPR was ongoing, a biphasic AED was attached, which showed coarse VF. Peripheral venous access was established without interruption of CPR. After 2 minutes of CPR (approximately 5 cycles of compression and ventilation), the patient received his first shock. A pulse was detected after 2 minutes of CPR after the first shock, and the AED showed sinus rhythm. The patient regained consciousness with establishment of spontaneous circulation. The patient's ECG immediately after resuscitation showed ST-segment elevation in V1 to V3 with reciprocal changes in inferior leads, consistent with anterior myocardial injury. He was immediately taken to the cardiac catheterization laboratory and received percutaneous coronary intervention to the left anterior descending epicardial coronary artery. Subsequent blood chemistry revealed elevated cardiac enzymes. Since he was not comatose, he was not deemed to be a candidate for induced hypothermia. His blood glucose was closely monitored and kept between 80 and 110 mg/dL by an insulin infusion. He had 1 episode of elevated temperature of 38°C after the procedure, which was promptly treated. The patient's electrolytes were closely monitored and any deficiency was corrected. He was discharged home neurologically intact.

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Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

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CME Questions

1. What is the correct chest compression to ventilation ration in adult CPR?
 - a. 15:2
 - b. 10:2
 - c. 30:2
 - d. 30:4
2. What is the incidence of VT/VF cardiac arrest in adults?
 - a. 50% to 60%
 - b. 21% to 32%
 - c. 10% to 16%
 - d. 72% to 82%
3. What are the 2 most common underlying etiologies of SCD?
 - a. CAD and cardiomyopathies
 - b. CAD and WPW syndrome
 - c. Cardiomyopathies and WPW syndrome
 - d. Drug effects and WPW syndrome
4. Which of the following statements is false?
 - a. Biphasic devices have higher first shock success rates than monophasic devices.
 - b. Repeated shocks with monophasic devices may produce atrioventricular block.
 - c. Biphasic devices use lower energy levels than monophasic devices.
 - d. Biphasic devices have shown a survival benefit when compared to monophasic devices.

5. Which CPR intervention has the largest impact on survival in cardiac arrest?
 - a. Good quality CPR and early defibrillation
 - b. Frequent ventilation of the patient
 - c. Intubation and placement of a central venous access
 - d. Use of antiarrhythmics
 - e. Controlled hypothermia
6. Immediately after the delivery of a first shock you should:
 - a. Check the rhythm on the monitor.
 - b. Palpate the carotid pulse.
 - c. Begin CPR for 2 minutes.
 - d. Check the patient's blood pressure.
7. Regarding the use of vasopressors in cardiac arrest, which of the following is true?
 - a. Use of higher doses of epinephrine results in a higher rate of survival to discharge.
 - b. Vasopressin is superior to epinephrine in all forms of cardiac arrest.
 - c. Epinephrine is effective in an acidic environment where vasopressin is not.
 - d. Vasopressin 40 IU can replace the first or second dose of epinephrine during ACLS.
8. The use of which antiarrhythmic in cardiac arrest has been shown in clinical trials to improve survival to hospital admission?
 - a. Amiodarone
 - b. Lidocaine
 - c. Procainamide
 - d. Ibutilide
9. Which of the post-resuscitative measures have shown to improve outcomes in cardiac arrest patients?
 - a. Hyperthermia
 - b. Hypothermia
 - c. Hyperglycemia
 - d. Hypoglycemia
10. Use of amiodarone in cardiac arrest results in higher incidence of hypotension when compared to lidocaine
 - a. True
 - b. False

Physician CME Information

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