Acute management of sudden cardiac death in adults based upon the new CPR guidelines

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Received 3 February 2006; accepted after revision 20 August 2006

Purpose of the review

The aim of this article is to provide a comprehensive description of interventions that can improve outcomes in adults with sudden cardiac death. The new American Heart Association 2005 Guidelines introduced a number of changes for the initial management of cardiopulmonary arrest based on new data that accumulated over the last 5 years.

Acute management of sudden cardiac death

Appropriate interventions targeting the three phases of cardiopulmonary resuscitation (CPR) (electrical, circulatory, and metabolic) should be implemented. Early defibrillation in early witnessed arrest with one shock is very effective and can improve survival outcomes. When resuscitation efforts are delayed and CPR is performed by paramedics, 2 min of CPR before shock is recommended. Emphasis has been placed on fast and forceful continuous compressions with minimal interruptions, adequate decompression, and decrease in the rate of ventilations to 8–10/min for intubated patients with two rescuers and a universal increase in compression to ventilation ratio to 30:2 for lone rescuers. Mechanical adjuncts to improve circulation have been adapted in the recommendations. The inspiratory impedance threshold device that enhances negative intrathoracic pressure and improves venous preload has been recommended for application in intubated and bag-mask ventilated patients. Owing to the difficulty of endotracheal intubation, airway management devices (Combitube and Laryngeal Mask Airway) can be used as alternatives with minimal extra training.

Conclusion

The new guidelines for CPR have focused on early defibrillation, uninterrupted compressions, complete decompression, fewer ventilations, and simplification and uniformity of the process.

Keywords

Sudden cardiac death; Cardiopulmonary resuscitation; Circulation; Ventilation

Introduction

Approximately 1,000,000 sudden cardiac deaths occur yearly in the USA and Europe.¹,² Despite a dramatic improvement in many aspects of treatment strategies for emergency clinical situations, the survival from both out-of-hospital and in-hospital cardiac arrest remains poor. Even in the most advanced emergency medical systems in Western societies,¹,³ neurologically intact survival rates remain <20%. On the basis of the large body of scientific advances in the arena of cardiopulmonary resuscitation (CPR), the American Heart Association and European Resuscitation Council have developed new guidelines for CPR and emergency management of sudden cardiac death.⁴

This article is focused on the major and critical interventions that have been proposed for the initial management of cardiac arrest. Special emphasis will be given to the basic simple ways to improve circulation and vital organ perfusion pressures in an effort to improve the grave prognosis of sudden cardiac death. Most of this article is focused on relatively simple ways to increase the blood flow to the heart and brain during CPR to improve the overall chances of meaningful survival.

The complexity of cardiac arrest has led to the generation of a theory for its study, which divides cardiac arrest into three phases. Although the existence of those three phases is not scientifically proved, they are widely used as a basis for research. Specific treatments targeting the pathophysiology of each phase increase the chances of a meaningful recovery.⁵ The first phase, the electrical, where most patients have ventricular fibrillation, lasts ~4 min after the initial collapse and is characterized by a high degree of responsiveness to early defibrillation. The second phase, the circulatory, lasts from min 4 to 10, depending on the surrounding temperature and conditions. Good quality CPR, with emphasis given to improve delivery of oxygenated blood to brain and the heart before

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defibrillation, is of paramount importance and techniques that increase circulation have been shown to improve outcomes. The third phase, the metabolic, usually starts after 10 min. Current treatments are poor. These late efforts generally target the metabolic effects of prolonged ischaemia. Survival is inversely related to the time of untreated cardiac arrest. Most of the victims of cardiac arrest get professional assistance during the second phase of cardiac arrest, namely, the circulatory phase. This is one of the reasons that the new guidelines have placed extra emphasis on ways to improve circulation and to simplify the delivery of compressions and ventilations.

Immediate resuscitation
The earlier the intervention after the cardiac collapse, the higher the chances of survival. On the basis of the idea that most of the victims of cardiac arrest are initially approached by laypersons, a specific emphasis has been placed, increasing the rate of bystander CPR. Specifically, the new guidelines recommend that radio dispatchers should encourage chest-compressions-only CPR when there is unwillingness to provide rescue breathing in order to facilitate rapid initiation of chest compression. Fear of transmission from mouth-to-mouth ventilation and the challenges of trying to teach mouth-to-mouth by telephone dispatch have shifted the emphasis of pre-emergency medical service (EMS) arrival phone instructions to focus rescuers on chest compressions only. For lay rescuers interested in learning traditional CPR, a new and effective home-instruction self-learning programme was developed to teach basic life support (BLS) and automatic external defibrillator (AED) techniques in 30 min rather than the traditional 4 h course.

In the new guidelines, early defibrillation is re-emphasized as an essential therapy for the electrical phase of cardiac arrest. Direct current defibrillation can restore a perfusing rhythm in 80% of patients within 1–2 min. However, after 10 min, the success rate falls to <5% without CPR. That is the reason that broad deployment of public access defibrillators for layperson use has been encouraged. In a study that tested early defibrillation in US casinos, the survival of patients suffering from ventricular fibrillation was 50% overall. Patients who received defibrillation within 3 min of collapse had a 75% hospital discharge rate. The new 2005 guidelines give a Class I recommendation for early defibrillation in a witnessed arrest, reinforcing the previous 2000 guidelines. A Class I or IIa recommendation is given to techniques and devices that have been shown in clinical trials to be effective and considered acceptable and useful.

The type of waveform recommended for defibrillation has also been changed. Biphasic electrical counter-shocks have been shown to decrease defibrillation thresholds when compared with monophasic shock. Although there are no clinical data demonstrating improved short- (1 h) or long-term survival rates between the monophasic and biphasic waveforms for the treatment of ventricular fibrillation during the electrical phase of cardiac arrest, a single high energy (150–200 J) biphasic defibrillation shock was recommended, Class IIa, for the treatment of both in-hospital and out-of-hospital ventricular fibrillation. Class IIa recommendations are given in the guidelines when the level of evidence or the consensus of expert opinion (when evidence is absent) suggests a benefit without harm from any particular recommendation. Nearly, all new external defibrillators are currently manufactured to deliver biphasic shocks, but the AEDs need to be reprogrammed to minimize the time when there is no CPR or circulation to the heart and brain.

Improving circulation
Most of the pre-hospital cardiac arrests cannot be treated within 4 min of the electrical phase. During this phase, there is a need for immediate compressions to generate blood flow and partially replete the membrane’s energy required for generation of an organized rhythm. It has been recently shown that when the time between the emergency call and the paramedic arrival is longer than 4–5 min, CPR first, before shock, improves survival rates. If the time to defibrillation was <5 min, there were no differences in survival. When the time from the emergency call to the arrival of the ambulance was >5 min, CPR first improved survival and hospital discharge rates five-fold (4–22%).

The new guidelines focus on more compressions and fewer ventilations. They call for immediate chest compressions and, once an advanced airway has been established, continuous chest compressions without interruption for ventilations. For BLS, the compression to ventilation ratio was increased from 15:2 to 30:2 in order to provide fewer interruptions of compressions for ventilation. During CPR, even in the best of circumstances, the generated cardiac output is <20% of normal. Each positive pressure ventilation immediately decreases the blood flow to the heart and brain. Moreover, respiratory exchange is adequate with less than normal minute ventilation, in part, because gas exchange is limited by the severely reduced pulmonary flow.

This change in the guidelines was based upon a consensus of experts in CPR. However, it has been recently shown in pigs that with the shift from a 15:2 to 30:2 compression:ventilation ratio, there is doubling of the common carotid blood flow and a 25% increase in cardiac output without any compromise in oxygenation and acid–base balance (Figure 1).

During advanced life support (ALS), uninterrupted compressions with a rate of 100/min are recommended. The rescuers who are responsible for the ventilation should deliver 8–10 breaths/min with special care not to hyperventilate. Rescuers should rotate frequently (every 2–3 min) to avoid excessive fatigue, which is known to diminish the quality of CPR (discussed subsequently).

Ventilations
Periodic ventilation during CPR is important to provide oxygen to the blood and tissues. However, a fundamental shift in the new guidelines is to prevent excessive ventilation rates, which have been shown to be life-threatening, if not deadly. As in the prior recommendation, obtaining an open airway is of paramount importance, which for endotracheal intubation is recommended as Class I intervention. For the same reason, the use of a Combitube, that is placed in the oropharyngeal cavity and allows for non-selective airway isolation for the purpose of ventilation, and the Laryngeal Mask Airway (LMA), are recommended as a Class IIa intervention in the new guidelines. Neither has been shown to alter outcomes after cardiac arrest, but both provide a means to maintain airway patency. More
Importantly, however, the guidelines stress the importance of reducing the frequency of ventilation during CPR, along with the importance of delivering a breath rapidly to minimize the duration of positive airway pressures. Each time intrathoracic pressure is increased with a positive pressure ventilation, venous return to the heart is inhibited and intracranial pressure is increased. As such, the benefits of positive pressure ventilation must be weighed against the harm associated with too much ventilation (Figure 2).

Hyperventilation increases intrathoracic and intracranial pressures and concomitantly decreases coronary perfusion and mean arterial pressures and survival rates in animals. Intracranial pressures are regulated, in part, by intrathoracic pressures: each time ventilation is delivered, there is a rise in the pressure inside the thorax and the brain, which reduces cardiac and cerebral perfusion pressures and survival rates (Figure 3). A simple change in the compression to ventilation ratio from 15:2 to 15:1 resulted in an increase in diastolic aortic pressure and higher cerebral perfusion pressure in pigs.

Based in part upon this rediscovered physiology, the new guidelines recommend a reduced ventilation rate during BLS of two breaths after 30 continuous compressions and of only 8–10 breaths/min for ALS. Moreover, each BLS and ALS breath should be delivered with a tidal volume of only ~500 cc and over a period of only 1 s. These subtle, but fundamental, changes in ventilation technique assure optimal circulation during conventional manual closed chest CPR.

Compressions

Generation of blood flow during compression results from an increase in intrathoracic pressure (thoracic pump theory), the mechanical effect of compressing the heart between the sternum and the spine (cardiac pump theory), and the cardiac valvular system that allows mainly unidirectional flow. The new guidelines, recognizing the importance of compressions during CPR, recommend pushing "hard and fast". A depth of 1.5–2 in. (5 cm) is considered adequate compression depth.

The rate should be 100 compressions/min as lower rates decrease forward blood flow. Interruptions should be minimized, because every time compressions are stopped, it takes a significant amount of time to re-establish adequate aortic and coronary perfusion pressures. For example, pulse checks should not last more than 10 s. In observational studies, the average time without

![Figure 1](image1.png) After 6 min of untreated ventricular fibrillation, 6 min of either 15:2 or 30:2 compression to ventilation ratio CPR was performed. At the end, the inspiratory ITD was added for another 4 min. There was a significant increase in cardiac output, common carotid artery flow, end tidal CO₂ in the animals that received 30:2 C/V ratio. There was a further increase with the addition of an ITD, although it was applied late. PaCO₂-EtCO₂ is a marker of pulmonary ventilation/perfusion matching. Asterisk means statistical significant difference compared with the ratio of 15:2 (P < 0.05).

![Figure 2](image2.png) When after 6 min of untreated ventricular fibrillation pigs received CPR with either 12 or 30 breaths/min (as observed frequently in a clinical trial), the mean intrathoracic pressure was inversely related to coronary perfusion pressure and 1 h survival rates (P < 0.05).

![Figure 3](image3.png) Representative real-time tracings from a single animal during 15:1 and 15:2 compression to ventilation ratios. Aortic pressure and intracranial pressure are shown as well as intrathoracic pressure at the level of the carina. Filled with black is the cerebral perfusion pressure area. Notice the significant decrease in the area when two breaths instead of one are delivered. There is an increase in intracranial pressure with positive pressure ventilation and it more pronounced with the 15:2 ratio. ICP, intracranial pressure; AoP, aortic pressure; CerPP, cerebral perfusion pressure; ITP, intrathoracic pressure. Arrow heads show when the positive pressure ventilation is delivered.
compressions during resuscitation varies from 25 to 50%. This can be extremely detrimental as no compressions means no perfusion. Studies, in animals and humans, have shown that for the initial few minutes of CPR, uninterrupted chest compressions only are an alternative to standard CPR that has the advantage to be performed by any layperson who is not willing to perform mouth to mouth ventilation.

An important change in the new guidelines is that uninterrupted chest compressions should be delivered both before and immediately after the delivery of a shock. Chest compressions for 90 s to 3 min before defibrillation help to prime the pump, making successful return of spontaneous circulation most likely after defibrillation. Chest compressions for 60 s to 2 min immediately after defibrillation are thought to help prevent the hypotension and asystole often observed when a defibrillation shock is delivered. As a result, rather than check for a pulse after a defibrillatory shock in a patient who has been in ventricular fibrillation for >4 min, the guidelines recommend the rescuers immediately resume CPR to maintain circulation, even if the heart is spontaneously beating. It is important to note that this recommendation was based upon a consensus of experts rather than a clinical trial demonstrating increased short- or long-term survival rates with this new approach. Despite the theoretical risk of re-inducing fibrillation with chest compression, there are no human data to support a significant risk in performing a 2–3 min of CPR before checking for rhythm and pulses after defibrillation.

Decompressions

Newly emphasized in the guidelines is the importance of the decompression phase. With each chest wall recoil, the negative intrathoracic pressure naturally generated by the elastic recoil properties of the chest wall acts to promote venous return to the heart, thereby increasing preload for the next compression cycle. Incomplete decompression, such as hyperventilation, is a common mistake that also decreases blood flow to the heart and brain during CPR. Fatigue and ineffective technique, as well as inappropriate hand positioning, can result in incomplete chest wall recoil. From a recent randomized trial, it was shown that many rescuers fail to decompress completely. This results in a sustained end-diastolic increase in intrathoracic pressure. This phenomenon, when examined in a porcine model of cardiac arrest, revealed two fundamental effects. First, incomplete chest wall recoil caused a significant decrease in mean arterial pressure, an increase in right atrial pressure, and thus decreased coronary perfusion pressures. Second, incomplete chest wall recoil caused an increase in intracranial pressure leading to a significant decrease in cerebral and systemic perfusion pressures. Strikingly, when incomplete decompression and positive pressure ventilation occurred simultaneously, cerebral perfusion ceased: the cerebral perfusion gradient was essentially zero for at least three to four compression-decompression cycles (Figure 4). Thus, the guidelines re-emphasize the importance of full chest wall recoil after each compression to avoid the deleterious effects of incomplete chest wall recoil and the combined and dangerous effects of hyperventilation and incomplete decompression.

Alternative hand positioning has been described in order to eliminate incomplete decompression.

Devices

Inspiratory impedance threshold device

The dynamic energy of the expanding chest wall during the decompression phase can be harnessed in order to increase venous return, lower intracranial pressure, and increase circulation to the heart and brain. The inspiratory impedance threshold device (ITD) regulates the entry of air through the Airways into the chest during the decompression phase of CPR. It causes a decrease in intrathoracic pressure to −5 to −10 mmHg and thereby helps to generate a greater intrathoracic vacuum to draw blood back to the heart during the recoil phase of CPR. Although this device is attached to an airway, it is used during CPR to enhance circulation. The ITD has been shown repeatedly to improve blood flow to vital organs and survival in animal and human studies. In clinical studies, the ITD doubles the systolic blood pressure during CPR and increases the chances of short-term survival. This is the only CPR device with a Class Ila level of recommendation in the new guidelines, and it is recommended as a way to enhance circulation during CPR and to increase the chances of successful resuscitation and a return of spontaneous circulation. The ITD needs to be applied early and it can be used both with intubated patients connected to the endotracheal tube and with a facemask and a good seal (Figure 5).

Oesophageal-tracheal Combitube, laryngeal mask airway, and facemask ventilation

Owing to the practical issues of adequate training for endotracheal intubation, the Combitube has been recognized as a reasonable alternative when endotracheal intubation is not possible or feasible. This device isolates the oesophagus and allows for airway isolation for ventilation and avoidance of gastric inflation. One of the most important issues for prevention of potentially fatal complications is the correct identification of the distal port. Laryngeal mask airway has been shown in many clinical non-cardiac arrest situations to be simple to use and to provide a safe alternative to intubation. The new guidelines recommend its use in a manner similar to the Combitube, when endotracheal intubation is not possible or feasible. The only disadvantage is that a small percentage of patients cannot be adequately ventilated and an alternative airway management technique should be available. Both these airway adjuncts have been recommended as a Class Ila interventions by the new AHA 2005 guidelines.

As in earlier guidelines, two-person technique is recommended when using a facemask for ventilation. One person should maintain the correct head position, the complete seal, and a jaw thrust to maintain airway patency and the second person should squeeze the resuscitation bag. This approach can be used for more prolonged period of time when sufficient personnel is available as it enables rescuers to perform high quality CPR without stopping compressions and interrupting circulation to place an advanced airway device. One of the hallmarks of the new guidelines is to maintain continuous chest compressions without interruptions. Delaying intubation by providing a
A good facemask ventilation technique is an important way to maximize chest compression time.

Compression devices

Several chest compression devices were again evaluated in the new guidelines. The Autopulse, an automated band-compression device, has shown significant improvement in vital organ perfusion and systemic pressures in animals and humans.\textsuperscript{45-47}

This device was given a Class IIb level of recommendation, suggesting that it is probably of benefit. However, a recent randomized trial (ASPIRE) showed that the use of the Autopulse increased mortality rates.\textsuperscript{48} As such, this trial was prematurely stopped. There are concerns about the device, including weight and delays associated with placement. At the time of writing, it is not clear whether the increased death rate associated with the use of the Autopulse is caused by the way the device performs CPR or whether it is due to a device implementation issue, or both.

Active compression–decompression (ACD) CPR devices were recommended with a Class IIb for in-hospital use and

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**Figure 4** The effect of positive pressure ventilation and incomplete decompression on CerPP is shown. The first tracing shows real-time aortic and ICP waveforms from a pig with full chest wall recoil after a ventilation cycle, whereas the second tracing shows the aortic and ICP waveforms with incomplete chest wall recoil after a ventilation cycle. Positive pressure gradient (Ao–ICP) is shown in black. Note the marked difference in total area during each compression–decompression cycle with and without a positive pressure breath. The bar graph shows the mean four beat area of all animals during and after a ventilation cycle. The mean ± SEM values during 100 and 75\% decompression are illustrated. Arrows show when the positive pressure ventilations are delivered. ITP, intrathoracic pressure.

**Figure 5** Concept and design of the inspiratory ITD. ITD can be placed on an endotracheal tube or on a specially designed facemask for bag-mask ventilation provided that a good air seal is applied. Timing lights flash at a rate of 10/min to guide ventilations and compressions (10 compressions between 2 flashes).
Class indeterminate (more data needed) for pre-hospital use. Two in-hospital studies with this device have shown an increase in short-term survival rates.\textsuperscript{49,50} Multiple out-of-hospital survival studies have been performed with ACD CPR.\textsuperscript{51–56} Some have shown significant improvements in up to 1-year survival, whereas others have shown no significant benefit with the device.

Another ACD device is the Lund University Compression Assist Device or LUCAS device. There are no randomized controlled trials with this device. Thus, it too was given a Class indeterminate level of recommendation.

**Therapeutic hypothermia**

During the metabolic phase of cardiac arrest, decreasing core body temperature has been shown to have protective effect on the myocardium from reperfusion injury (deep hypothermia 25°C).\textsuperscript{57,58} Hypothermia also protects the brain, possibly by lowering intracranial pressures\textsuperscript{59,60} and by protecting the brain from ischaemic injury.\textsuperscript{61–64} In two large randomized human trials, mild-to-moderate hypothermia (32–34°C), post-resuscitation, resulted in an improvement (16–23% absolute risk reduction) for poor neurological outcomes in patients who had a witnessed ventricular fibrillation arrest. There was a significant improvement in 6-month survival rates in the hypothermic groups.\textsuperscript{65,66} In resuscitated victims of cardiac arrest, especially after prolonged resuscitation efforts, hypothermia should be considered and implemented when possible. The new guidelines have given hypothermia a Class IIa level of recommendation for comatose patients with ventricular fibrillation arrest.

For all other presenting rhythms of cardiac arrest, hypothermia was given a IIb recommendation. Recent studies have shown that it is possible to cool during CPR before reperfusion is achieved to minimize tissue damage before it occurs. Cooling during CPR in animals with venovenous access systems or with cold IV saline and use of ACD CPR plus the ITD may eventually offer a means of rapidly decreasing cerebral temperatures during CPR and improving neurological outcomes.\textsuperscript{67,68} On the basis of the data in support of therapeutic hypothermia, the guidelines recommend cooling of comatose patients after successful resuscitation when possible, as long as there is a protocol in place to assure careful monitoring of core temperatures and haemodynamics, prevention of shivering, and maintenance of adequate perfusion pressures during the recommended 24 h period of cooling. Further study is needed to evaluate the therapeutic potential of very early cooling and to investigate the best way of achieving cerebral hypothermia in a timely and practical manner.

**Pharmacological management**

There were very few major changes in the pharmacological management of patients in cardiac arrest.

**Vasoactive medications**

Evidence for the broad use of vasoactive medication during CPR comes primarily from animal studies. There are no placebo-controlled trials that have demonstrated long-term benefit of either adrenaline or vasopressin. As such, the new guidelines recommend the use of either of these agents as Class IIb.

Adrenaline is the most commonly used vasopressor during CPR. The beneficial haemodynamic effects of adrenaline during CPR are due to its potent alpha-adrenergic effects. The significant increase in central aortic pressures results in significant increase in coronary and cerebral perfusion pressures and possibly rates of successful resuscitation.\textsuperscript{69,70}

However, on the basis of the multiple clinical trials, the use of high dose adrenaline is contraindicated and harmful in patients in cardiac arrest (Class III). The guidelines continue to recommend 1 mg of adrenaline every 3–5 min (Class IIb) for adults in cardiac arrest. If no venous access has been obtained, endotracheal or intra-osseous administration can also be effective.

Vasopressin is recommended as an alternative vasopressor during CPR. It too has potent vasoconstricting properties. No study has shown that vasopressin use will result in an increase in hospital discharge rates when used in patients in cardiac arrest. A recent study showed that the combination of adrenaline plus vasopressin resulted in higher rates of resuscitation, no increase in long-term survival rates, but a strong trend towards worsening of neurological outcomes, except in those with an initial rhythm of asystole arrest.\textsuperscript{71,72} On the basis of these findings, 40 U of vasopressin can be used instead of the first or second dose of adrenaline during CPR (guidelines recommendation: Class indeterminate). The authors of this article recommend that one to two doses of adrenaline are used prior to vasopressin given the lack of definitive data and levels of recommendation in the new guidelines.

There is no good treatment for asystole. Atropine, a vagolytic medication, has no known untoward effects in patients with asystole and can be given for severe bradycardia and asystole with doses of 1 mg IV every minute to a total dose of 3 mg. (Class indeterminate). There is no randomized animal or human study to support the administration of atropine for the improvement of outcomes.

**Anti-arrhythmic agents**

As with the other intravenous medications, there were insufficient data or consensus among the experts regarding the use of anti-arrhythmic agents during CPR. Amiodarone is now considered the drug of choice and as an intravenous bolus of 150–300 mg for ventricular fibrillation or pulseless ventricular tachycardias that are unresponsive to the initial sequences of CPR–shock–CPR–vasoconstrictors. The recommendation is based on limited clinical trials showing improvement in hospital admission, but no definitive increase in hospital discharge rates when compared with placebo or lignocaine. Given the lack of definitive data, lignocaine (initial dose of 1–1.5 mg/kg IV) can also be used in patients in cardiac arrest (Class indeterminate).

**Summary**

In the new 2005 AHA CPR guidelines, significant emphasis has been placed on early defibrillation, quality and continuity of compressions, less frequent ventilations, and adequate decompression. In order to simplify CPR...
performance, a unifying 30:2 compression to ventilation ratio has been introduced for all single rescuers. Adjuncts to improve circulation during CPR, such as the inspiratory ITD, have also been recommended, although the evidence for their effectiveness has still to be determined. Comatose patients with ventricular fibrillation arrest could be treated with mild therapeutic hypothermia as long as the infrastructure needed to support the patients is promptly available.

Acknowledgement

This manuscript was partially supported by an American Heart Association Postdoctoral Fellowship Grant for DY: 0425714Z.

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