

# How can we reduce sudden death in the community?

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*Cardiac arrest outcomes have not improved for 30 years. Survival from defibrillation within 3 minutes of ventricular tachycardia/fibrillation (VT/VF) onset is 70% to 80%. However, VT/VF accounts for only 30% to 40% of arrests. In non-VT/VF arrest and VT/VF arrest without an automated external defibrillator (AED) on site, survival from manual cardiopulmonary resuscitation (CPR) is 2% to 8%, vs 20% to 30% from witnessed arrests in communities offering bystander CPR and rapid arrival of trained personnel with an AED. Hence the drive to simplify CPR instruction, emphasizing proper chest compression, full release between compressions, and avoidance of overventilation. Drugs and devices other than defibrillators have demonstrated no long-term survival benefit. Technological advances and raised community awareness offer the best hopes for improving quality survival.*

**Keywords:** CPR (cardiopulmonary resuscitation) mechanics; CPR devices; AED (automated external defibrillator); sudden death; cardiac arrest

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Improving survival from cardiac arrest is an illusive goal of modern medicine. Defibrillation within 3 minutes of onset of ventricular tachycardia or ventricular fibrillation (VT/VF) results in survival of 70% to 80%. But only 30% to 40% of arrests have an initial recorded rhythm of VT/VF. In non-VT/VF arrests and in VT/VF arrests without an automated external defibrillator (AED) on site, the best hope is to apply optimal manual cardiopulmonary resuscitation (CPR) as soon as possible. In communities where bystander CPR is common, and trained personnel with an AED arrives promptly, 20% to 30% of witnessed arrests survive. In most com-

munities, 4% to 8% survive. Drugs and devices other than defibrillators have no demonstrated long-term survival benefit. Currently, concentration of effort is on simplifying CPR instruction with emphasis on proper chest compression. Overventilation and lack of full release between chest compressions are of importance in performance of manual CPR. Hypothermia may benefit those who survive to reach the hospital, but remain unconscious. Community efforts and technology advances do hold considerable hope for improving quality survival from cardiac arrest.

## DEFINITION

Cardiac arrest is the "cessation of cardiac mechanical activity, as confirmed by the absence of signs of circulation."<sup>1</sup> This syndrome leads to death within 15 to 30 minutes from onset of first symptoms. First symptoms are most commonly loss of consciousness or cessation of spontaneous breathing. No pulse is detectable. If an emergency medical service (EMS) provider or physician did not witness the event, then it may be difficult to ascertain whether a cardiac arrest has occurred. "Presumed cardiac etiology" is frequently used to subcategorize cardiac arrest, but can only accurately be determined by conducting a postmortem examination. Since it is impractical to conduct an autopsy on every out-

### SELECTED ABBREVIATIONS AND ACRONYMS

<b>ACS</b>	acute coronary syndrome
<b>AED</b>	automated external defibrillator
<b>CPR</b>	cardiopulmonary resuscitation
<b>EMS</b>	emergency medical service
<b>ICD</b>	implantable cardioverter-defibrillator
<b>MI</b>	myocardial infarction
<b>PAD</b>	public-access defibrillation
<b>VF</b>	ventricular fibrillation
<b>VT</b>	ventricular tachycardia

of-hospital death, the likelihood of underlying disease remains uncertain, and many studies presume that an arrest is of cardiac origin unless there is another obvious cause.<sup>1</sup> Traumatic sudden death is usually considered separately from unexpected cardiac death because the treatment and prognosis of traumatic and nontraumatic arrest differ from each other. For the purpose of this article, we will use unexpected cardiac death, EMS-treated cardiac arrest, or nontraumatic out-of-hospital cardiac arrest as synonyms for cardiac arrest of presumed cardiac etiology.

## INCIDENCE

The incidence of cardiac arrest is the attack rate of cardiac arrest in the community. Risk factors for increased incidence of cardiac arrest include cellular (eg, gene mutations that predispose to arrhythmias), environmental, social, educational, behavioral (eg, activity level, smoking), clinical (eg, atherosclerosis, reduced ventricular function, diabetes), or health system risk factors.<sup>2</sup> Unexpected cardiac death generally occurs in persons with known or previously unrecognized ischemic heart disease. Early identification of modifiable risk factors and use of certain pharmaceutical agents (aspirin and  $\beta$ -blockers) can reduce an individual's risk of cardiac arrest.

The true incidence of out-of-hospital cardiac arrest is unknown. Overall, cardiovascular disease contributes 30.9% of global mortality. About half of coronary heart deaths are sudden.<sup>3</sup> Since there were 7.2 million coronary heart deaths worldwide in 2002 (<http://www3.who.int/whosis/>), this implies there were 3.6 million unexpected cardiac deaths. About two thirds of these occur without prior recognition of cardiac disease. About 60% are treated by EMS.<sup>4</sup>

Although the proportion of those treated by EMS is likely less in those countries that have limited access to emergency services, this implies that as many as 2.2 million cardiac arrests are treated by EMS worldwide annually.

The reported incidence of unexpected cardiac death in the US is 1.9/1000 person years among those aged 50 to 79 years. Since the US population aged 50 to 79 is 68 million (<http://www.census.gov/>), this implies 130 000 out-of-hospital cardiac arrests. The reported incidence of EMS-treated cardiac arrest is 36/100 000 to 81/100 000 total population.<sup>4,5</sup> Since the total US population is 300 million (<http://www.census.gov/>), this implies 107 000 to 240 000 treated arrests occur in the United States annually. Of these, 20% to 38% have VF/VT as the first recorded rhythm.<sup>5,6</sup> This implies 21 000 to 91 000 treated ventricular fibrillation arrests annually.

## Children

Unexpected cardiac death in children merits special consideration because its incidence, etiology, prognosis, and treatment differ from that of out-of-hospital adult episodes. The reported incidence of out-of-hospital pediatric unexpected cardiac death is 2.6 to 19.7 annual cases per 100 000.<sup>7</sup> It is usually due to trauma, sudden infant death syndrome, respiratory causes, or submersion, but ventricular fibrillation is still commonly observed.

## High-risk subgroups

Patients with acute coronary syndromes (ACS) or myocardial infarction (MI) are at high risk of mortality (ie, >5%) within 30 days of presentation, regardless of whether ST-segment elevation is present or absent. The risk of death remains elevated

for at least 6 months in patients with ACS, and for at least 1 year in patients with MI. Patients with ventricular dysfunction are at higher risk than those without. At least one half of these deaths are suspected to have an arrhythmic mechanism.

## Secular changes in cardiac arrest epidemiology

There has been a steady decline in morbidity and mortality from most cardiovascular diseases in high-income countries over the last 30 years. Much of this reduction has been attributed to risk factor modification. Although the incidence of ventricular fibrillation and cardiac arrest with any first initial rhythm is decreasing over time,<sup>5</sup> there has been little improvement in survival from cardiac arrest.<sup>8,9</sup>

## SURVIVAL

There is a wide geographic variation in outcomes after the onset of cardiac arrest.<sup>10,11</sup> This is attributable in part to regional differences in the availability of emergency cardiac care. Also, there is regional variation in EMS processes such as EMS service level provided, number of EMS providers responding, use of procedures or drugs in field, training, quality assurance/feedback, and response time intervals.<sup>11-15</sup> No published analysis has had adequate power to detect the independent effects of all of these factors.

The median reported rate of survival to discharge after out-of-hospital unexpected cardiac death with any first recorded rhythm is only 6.4%.<sup>11</sup> It is likely that this overestimates the actual rate of survival in many communities because of publication bias. In many large urban areas the rate is less than 2%,<sup>16-18</sup> and it is even lower in some rural areas. The reported average survival to dis-



charge after out-of-hospital pediatric cardiac arrest is 6.7%.<sup>7</sup> Most communities are not aware of their own survival rates, as cardiac arrest data are not routinely tracked. Yet, a city with an organized EMS system and dedicated quality assurance can achieve survival of 15% to 20% (see reference 5, and unpublished data, L. Cobb). If the average survival could be improved from 5% to 20% by optimizing the chain of survival, the premature deaths of 16 000 to 36 000 subjects in the United States could be prevented each year.

### EVOLUTION OF PRESENTATION OF SUDDEN DEATH

From the dawn of defibrillation and modern CPR, the ECG of the vast majority of adult patients recorded in the first 10 to 15 min after cardiac arrest showed VT/VF. Beginning 10 years ago, an increasing incidence of asystole, or an organized electrical rhythm without perfusion, has been recognized. The evolution of presentation has been rapid and profound numerically: from 80% to 90% VT/VF to as low as 20% to 30% VT/VF today. At the same time, there has been some evolution of demographics, which may in part be causal. The average age of the victims of cardiac arrest is strikingly older with the overall incidence in the US not much changed.

Another likely possibility is that primary prevention of acute myocardial infarction, revascularization of patients with severe coronary stenosis, and effective medical (drug) management of patients with coronary disease and particularly chronic coronary disease with congestive heart failure have reduced VT/VF as the cause of cardiac arrest. A final and rapidly growing participant in this prevention strategy is the implantation of defibrillators in large

groups of patients with a history of nonfatal arrhythmic events or a left ventricular ejection fraction of less than 35%. In such populations, randomized clinical trials suggest that about 5% of these patients with an implantable cardioverter-defibrillator (ICD) implant will be saved from VT/VF cardiac arrest per year. If the number of appropriate implants performed in the US is 100 000 per year in the US, 75 000 VT/VF arrests will be prevented over 5 years.

The etiology of VT/VF arrest is acute coronary occlusion or reentry around scar in chronic coronary disease with heart failure, but the underlying cause and the proximate cause for non-VT/VF arrests are unclear. Also, effective therapeutic strategies for such arrests are poorly studied and remain elusive. Rapid electrical intervention such as defibrillation and pacing have little if any role in survival of this growing and important group of out-of-hospital cardiac arrest victims.

### CARDIOPULMONARY RESUSCITATION

#### Bystander CPR

CPR is important both before and after shock delivery in patients with pulseless ventricular tachycardia or ventricular fibrillation, as well as throughout resuscitative efforts in those with asystole or pulseless electrical activity. When performed immediately after collapse from VT/VF, CPR can double or quadruple the victim's chance of survival.<sup>19-22</sup> After 5 minutes of untreated VF, outcome may or may not be better if attempted defibrillation is preceded by a period of CPR.<sup>23-25</sup> Therefore, it remains unclear whether rhythm analysis to confirm the presence or absence of VF should precede CPR for undifferentiated cardiac arrest.

#### Compressions-only CPR

Multiple animal studies of arrhythmic arrest suggest that compressions-only CPR may be as effective as standard CPR.<sup>26-32</sup> However, there are no directly relevant prospective human studies. Observational or secondary analyses of a trial dataset provide limited insight into the effectiveness of compressions-only CPR. In a Belgian cohort, 14-day survival after treatment by bystanders was 5% with ventilation alone (n=97 eligible), 9% with compression alone (n=258) and 12% with ventilation and compressions (n=561,  $P=0.09$  calculated from data).<sup>33,34</sup> In a Swedish cohort, survival to discharge was 4.3% with ventilation alone (n=620 eligible), 6.8% with compression alone (n=278), and 9.7% with ventilation and compressions (n=8979,  $P<0.01$  calculated from data).<sup>21</sup> In a secondary analysis of a Dutch randomized trial, survival to discharge was 7% with ventilation alone (n=15 eligible), 15% with compression alone (n=41) and 14% with ventilation and compressions (n=437,  $P=0.7$ ).<sup>35</sup> In all of these studies, lay responders who had been trained to perform compression and ventilations chose at the time of an emergency response to provide either or both. Therefore, these studies were subject to selection bias because the decision to provide some, but not all, components of CPR likely reflected the presence of responder discomfort with CPR skills or adverse patient characteristics. It is difficult to distinguish asphyxial versus arrhythmic arrest at the time of resuscitative efforts. Compressions-only CPR may be detrimental to patients in asphyxial arrests.

Thus, in adults with cardiac arrest treated by lay responders trained in standard CPR, survival with compressions-only CPR is almost certainly better than with no CPR, but

may not as good as with both compressions and ventilation. Until there is evidence to the contrary, compressions and ventilations should be given during CPR unless a responder is unwilling to provide mouth-to-mouth ventilation.

### Dispatcher instruction in CPR

A randomized trial in Seattle, Washington, evaluated the effectiveness of CPR telephone instructions provided when no one on the scene was performing CPR and the caller was willing to be instructed.<sup>36</sup> Survival was better among patients assigned to lay responder instruction in chest compressions alone than among those assigned to instruction in compression and ventilations (14.6% vs 10.4%,  $P=0.18$ ). However, instructions were delivered completely in only 62% of compressions and ventilation episodes versus 81% of episodes assigned to compressions alone ( $P=0.005$ ). It is unclear whether the differential survival reflects the benefit of compressions-alone, or the harm of prolonged dispatcher instructions for compressions and ventilations.

### NEED FOR IMPROVED CPR PERFORMANCE

Recent studies of cardiac arrest in the out-of-hospital setting in Milwaukee, Wisconsin,<sup>37</sup> Tucson, Arizona,<sup>38</sup> and Europe<sup>39</sup> have suggested

that CPR is not performed as recommended. These results were confirmed by a recent study in the hospital setting in Chicago, Ill.<sup>40</sup> Relevant factors include interruption of CPR, and adequate provision of key components of CPR and ventilation.

### Interruption

Several observational studies show that chest compressions are interrupted for 24% to 49% of the duration of resuscitation efforts in patients with cardiac arrest.<sup>38-40</sup> The small size and lack of controls in these studies make it difficult to correlate interruption of CPR with clinical outcomes. However, a surrogate measure of the effectiveness of CPR is coronary perfusion pressure. This is the pressure gradient generated between aorta and right atrium during the decompression phase of CPR.<sup>41</sup> Blood flows through coronary arteries predominantly during this chest decompression phase. Coronary perfusion pressures increase with ongoing chest compressions, but rapidly diminish with cessation of compressions to administer ventilation or other maneuvers.<sup>42</sup> Greater survival in animals (Figure 1)<sup>43</sup> and greater restoration of circulation in humans<sup>41</sup> are associated with greater coronary perfusion pressure. Therefore, interruptions of compressions should be minimized.

### Components of CPR

There are several key components of CPR. At least 80 chest compressions should be given per minute (ie, rate of 100 per minute to allow for no compressions during pauses) with sternal compression of 1.5 to 2 inches in adults to achieve optimal forward flow during CPR.<sup>44</sup> Complete chest wall recoil improves hemodynamics during CPR by generating a relatively negative intrathoracic pressure during decompression, which increases venous return and hence cardiac preload for the next compression.<sup>45</sup> In a single EMS system (n=13 arrests), persistently positive airway pressures were recorded in 46% of episodes (Figure 2).<sup>46</sup>

Such pressures are likely due to incomplete chest wall recoil, and are potentially deleterious. Therefore, rescuers should allow complete chest recoil after each compression, while maintaining hand position in the lower half of the sternum between the nipples.

### Components of ventilation

Hyperventilation during resuscitation of patients in cardiac arrest has been observed in some, but not all, settings. Excess ventilation increases intrathoracic pressure during the diastolic phase of chest compression, decreases venous return, and may decrease survival.<sup>37</sup>

The rate of ventilation should not be reduced by prolonged inspiration, which is associated with persistent positive intrathoracic pressure and decreased venous return. Retraining, quality assurance, and use of devices such as metronomes or timing lights may reduce hyperventilation and integrate ventilation with performance of chest compressions.

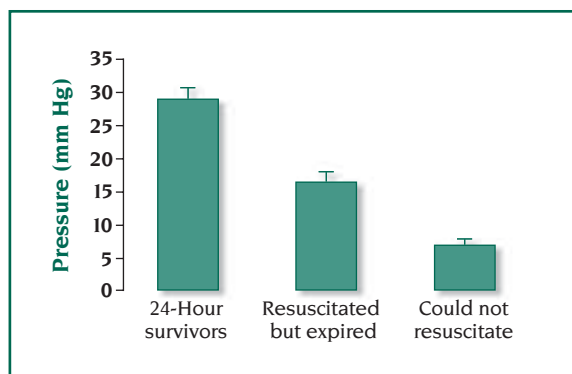
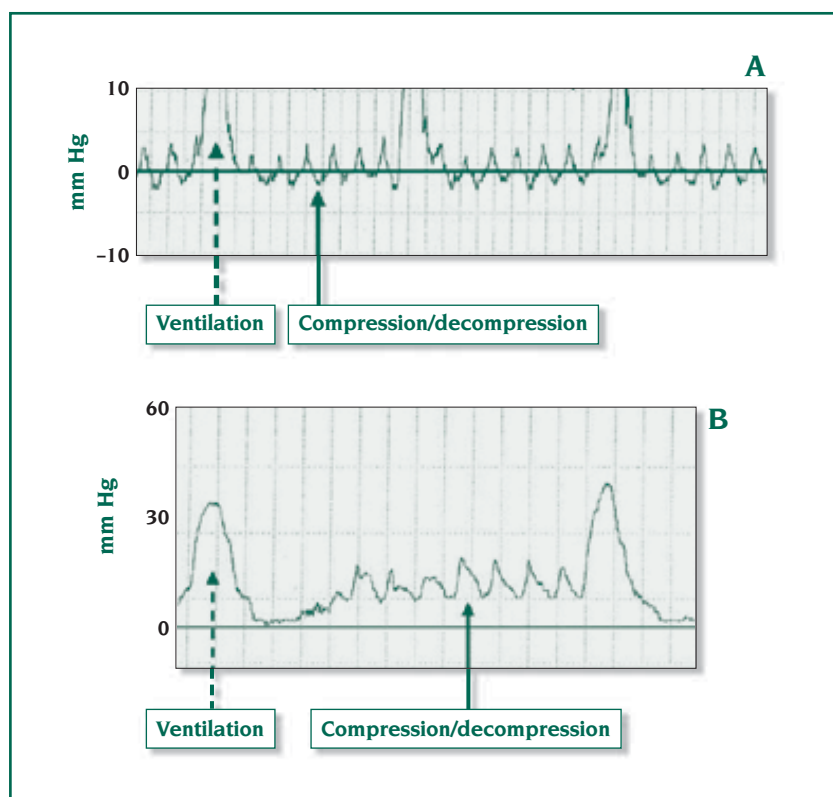


Figure 1. Chance of survival after prolonged cardiac arrest is positively correlated with coronary perfusion pressure generated during chest compression in dog models.

Modified from reference 43: Ewy GA. Cardiocerebral resuscitation: the new cardiopulmonary resuscitation. *Circulation*. 2005;111:2134-2142. Copyright © 2005, Lippincott Williams & Wilkins.



**Figure 2.** A: This 16-s airway pressure recording is representative of cardiopulmonary resuscitation performed by ambulance personnel at the scene of cardiac arrest allowing complete chest wall recoil, confirmed by an independent observer. Note the generation of a small, negative intrathoracic pressure following each compression. B: A representative example of an airway pressure recording taken during visually observed incomplete chest wall recoil. Note the continuously positive airway pressure following each compression. The potential cause of the consistently positive airway pressures recorded during observations includes incomplete chest wall recoil, prolonged positive ventilations, intrinsic positive end expiratory pressure, increased airway resistance, or some combination of these etiologies, and cannot be solely ascribed to incomplete chest wall recoil.

Modified from reference 46: Aufderheide TP, Pirralo RG, Yannopoulos D, et al. Incomplete chest wall decompression: a clinical evaluation of CPR performance by EMS personnel and assessment of alternative manual chest compression-decompression techniques. *Resuscitation*. 2005;4:353-362. Copyright © 2005, Elsevier Inc.

### Importance of feedback to improve cardiopulmonary resuscitation

Real-time monitoring and feedback of the components of the CPR process is associated with significant improvement in compliance with resuscitation guidelines and may be associated with improved short-term outcomes (see references 32 to 47 and personal communication, P. Steen, January 25, 2006). Ongoing study is needed to determine whether feedback improves survival to discharge.

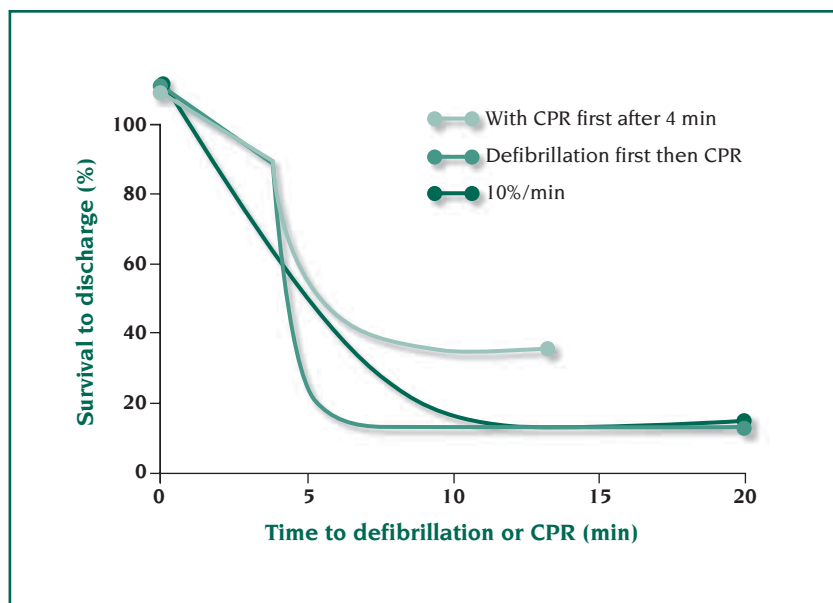
### TIMING OF DEFIBRILLATION

Based on reasonably solid experimental animal and correlative human data, Weisfeldt and Becker proposed that effective therapy of VT/VF arrest could be conceptualized in three sequential phases.<sup>30</sup> The most effect therapy for VT/VF is defibrillation during the first 4 to 5 minutes of cardiac arrest. This was called the “electrical phase of resuscitation.” After 4 to 5 minutes, and for at least 10 minutes after cardiac arrest, is the second or circulatory

phase. During this phase, immediate defibrillation provides worse outcomes in pigs and dogs than delaying defibrillation for 90 seconds to 3 minutes of effective CPR.<sup>17</sup> Finally, there is a somewhat ill-defined metabolic phase where hypothermia and/or yet to be discovered metabolic interventions may aid circulation and defibrillation in leading to survival with reasonably good neurologic status.

Studies of rapid defibrillation for VT/VF arrest in the first 3 minutes of out of hospital cardiac arrest suggest that 70% to 80% long-term survival can be expected.<sup>31</sup> Findings from ICD studies show that defibrillations within 20 seconds result in almost uniform survival. These studies suggest that epidemiologic studies<sup>15,16</sup> understate the survival results of early defibrillation likely due to imprecise timing of the onset of cardiac arrest without bystander CPR has taken place. Clinical studies<sup>17,18</sup> suggest that after 5 minutes of cardiac arrest, prompt defibrillation is associated with a less than 5% survival, but that a period of CPR followed by defibrillation could result in 20% to 30% survival (*Figure 3, next page*).

The evidence of the high survival from defibrillation during the electrical phase led to the concept of public-access defibrillation (PAD): simple self-instructional AEDs are made available to any willing and trained provider for use. A randomized trial of community implementation of AEDs showed that survival is doubled in community sites with this approach.<sup>32</sup> Today, this approach is of unproven value in the home, where the majority of arrests occur. Also, AEDs are not cost-effective if less than one cardiac arrest



**Figure 3.** Theoretical relationship of survival to timing and sequence of cardiopulmonary resuscitation (CPR) and defibrillation.

The dark green line is based on a large number of epidemiological studies<sup>15,16</sup> that suggest that there is a 10% decrease in survival with every minute before defibrillation is provided in patients with ventricular tachycardia/fibrillation (VT/VF) arrest. The other lines between 0 and 4 minutes are based on the Casino data,<sup>31</sup> which use exact time from onset of arrest. Similar data are available from other studies with very accurate time estimates. The 0 time point is from data from implanted defibrillators. The decline in survival after 5 min is based on the study of Wik and associates<sup>17</sup> where survival with CPR first is about 20% after 5 min of down time and much lower if defibrillation is provided before a period of CPR.

is likely to occur in 5 years. The higher the frequency of cardiac arrest in a given site, the greater value of a PAD-AED program. Increasingly, transportation facilities, airplanes, gaming and high stress and exercise environments, and large public buildings are being equipped with AEDs in clear public view. Technologic advances may bring lay or partner/neighbor defibrillation into the home. Soon a small implantable ECG monitor may, through an electronic page system, signal a partner or neighbor that VT/VF is present. Even more remarkable, a global position sensor may tell the partner and/or the EMS system the victim's location. Such an approach in high- or moderate-risk patients may be more acceptable and have greater cost effectiveness than defibrillator implantation.

### CPR ASSIST DEVICES

Since conventional CPR, even when performed optimally, leads to brain blood flow of 30% to 50% of normal and myocardial blood flow of 10% to 20% of normal, there is considerable interest in devices that may enhance survival by improving blood

flow. One of the closest correlates of survival in man is the coronary perfusion pressure gradient.<sup>26</sup> Studies primarily in animal models with correlates in man establish that in general blood moves forward during CPR as a result of the increase in intrathoracic pressure accompanying chest compression. In some patients, direct cardiac compression between bony structures may also occur during chest compression and is additive to the thoracic pressure rise.

Mechanical sternal compression devices have long been employed in some EMS systems and hospitals and provide a solution to rescuer fatigue and CPR during transportation. Of greater interest are devices that increase the rise in intrathoracic pressure during the compression phase such as inflatable vests or mechanical chest squeezing devices. Also, devices have been developed to obstruct airflow back into the chest between compressions. The latter type of device increases blood flow during manual CPR by providing increased venous return between compressions as a result of the induced negative intrathoracic pressure.

To date, none of these devices has been proven in randomized trials to improve long-term survival over conventional manual CPR. There is some evidence that the more complicated vest and chest-squeezing devices are inferior in terms of survival compared with best manual CPR, perhaps because these devices are bulky and burdensome compared with rapid performance of best manual CPR. The negative airway devices are small and simple and have resulted in improved short-term survival (ie, admission to the hospital). They require an occlusive airway device (intubation) or tight sealing mask and require optimal chest compression technique with full "hands or pressure off" the chest between chest compression. Such proper chest compress requires careful instruction and likely leads to earlier fatigue of the resuscitator. The AHA and the International Liaison Committee have given this airway device a favorable IIa rating in the most recent guidelines document.

Other mechanical or invasive approaches to CPR are performed from time to time in settings with spe-



cialized interests or skills. These approaches include internal cardiac massage and use of various degrees of cardiopulmonary bypass.<sup>36</sup> One group reports remarkably favorable results of full cardiopulmonary bypass with cardiac decompression and correction of reversible cardiac problems in patients who have failed "in-hospital" prolonged periods of CPR.<sup>37</sup>

### DRUGS DURING CPR

No drug has been shown to improve long-term survival from cardiac arrest in clinical trials in man. Short-term survival was shown in two studies to be associated with administration of amiodarone intravenously in refractory VT/VF: either persistent or recurrent VT/VF despite CPR and defibrillation.<sup>38,39</sup> In neither of the two reported studies was there even a trend toward improved survival to hospital discharge. Over the years, opinions, some based on animal data, have dominated the guidelines with regard to advice regarding the use of drugs. For example, repeat administration of sodium bicarbonate has come and gone from the guidelines, as has high-dose epinephrine. Epinephrine continues to be advised on the basis of animal studies. The 1-mg dose IV continues to be a mainstay of pharmacological management of cardiac arrest once initial CPR and defibrillation fail or with non-VT/VF arrest. Magnesium and calcium are used in special cases: the former when torsades de pointes is present and the latter with hyperkalemia. Other antiarrhythmics are now rarely employed. Vasopressin showed impressive results in animal models, but unimpressive results in man, and is considered in general an alternative to epinephrine. Vasopressin may have added value to epinephrine in the very challenging non-VT/VF arrest patient.<sup>37</sup> Studies of fibrinolytic

agents are ongoing in special subsets of cardiac arrest patients and there is interest in alternative vasoconstrictors, inotropic agents,  $\beta$ -blockers, and agents to reduce ischemic damage such as erythropoietin, antioxidants, and neuroprotective or antiapoptotic or anti-inflammatory agents. A safe aqueous preparation of amiodarone would also warrant testing in man during CPR since there is suspicion that the hypotensive effects of the diluent of currently available amiodarone accounts for its lack of long-term survival benefit.

### TAILORED APPROACH TO RESUSCITATION

A tailored approach to prevention and treatment of unexpected cardiac death is desirable since there is wide geographic variation in the incidence of cardiac arrest, resources available to treat it, existing rate of survival, and how much society is willing to pay to avert an arrhythmic death. Although some segments of the population are at higher risk of cardiac arrest than others, more episodes occur in those at lower risk

because there are many more low-risk individuals. Consequently, primary prevention of cardiac arrest by reducing modifiable risk factors in individuals without apparent cardiac arrest could have a large public health impact.

The risk of cardiac arrest varies by location as well as by individual (*Table I*). Each site participating in the PAD trial<sup>32</sup> identified distinct units within their service area (eg, office buildings, public areas). Due to the large variety of settings studied in this trial, there was limited precision in the estimate of the incidence of cardiac arrest by location. However, a cohort study of out-of-hospital cardiac arrest in Seattle and King County, Washington, described the incidence of cardiac arrest by public location.<sup>48</sup>

### HYPOTHERMIA

Therapeutic hypothermia consists of initiation and maintenance of reduced whole body temperature. Hypothermia reduces intracranial pressure as well as production of glutamate and oxygen-free radicals

Unit at risk	Incidence	EMS response	Intervention
Rural	+	+++	Bystander CPR, AED programs
Committed neighborhood	+	++	Primary prevention, bystander CPR, AED programs
Large city	+	+ / ++	Optimize dispatch Dispatcher instruction in CPR
Selected high-risk public locations	++	+ / ++ / +++	Bystander CPR, AED programs
Selected high-risk individuals (eg, acute coronary syndrome in last month)	+++	+ / ++ / +++	Secondary prevention, Family CPR

**Table I.** Tailored resuscitation.

**Abbreviations:** AED, automated external defibrillation; CPR, cardiorespiratory resuscitation; EMS, emergency medical service.

that are associated with reperfusion injury after restoration of spontaneous circulation.<sup>49</sup> Two moderately sized randomized trials demonstrated that initiating and maintaining mild hypothermia (32°C to 34°C for at least 12 hours) via external cooling methods is safe and improves neurologic outcomes in comatose survivors of out-of-hospital VT/VF arrest.<sup>50,51</sup> Endovascular methods of therapeutic hypothermia may achieve more rapid, controlled, or sustained cooling that would be associated with additional benefits, but have not been evaluated in adequately powered randomized trials in patients with cardiac arrest. Concurrent medication including buspirone and meperidine may be necessary to facilitate cooling and patient comfort. The duration of re-warming may be as important as the duration or depth of cooling, since rapid re-warming of patients who underwent therapeutic hypothermia has been associated with adverse effects.

### FUTILITY

In recent years, the assessment and determination of futility with regard to meaningful patient survival after cardiac arrest has progressively become a mandate. In the presence of futility, CPR is avoided or stopped. In some states in the US, with no "do not resuscitate" (DNR) order, there is no legal basis for such withholding or stopping of CPR efforts when a judgment of futility is made. Such legal statutes are, in the judgment of these authors, unfortunate. By oath, physicians are committed

to not provide care that has no possible benefit to the patient. The unfortunate conflicts between legal statutes and futility has resulted in the ethically corrupt performance of slow or improper CPR. Instead, CPR should be liberally and fully provided if meaningful survival is possible and withheld by patient preference (DNR documentation) or futility.

### HORIZONS

The current state of resuscitation resembles a Dickensian novel. It is the best of times; it is the worst of times: there are multiple promising interventions, but outcomes have not improved for 30 years. Future interventions may yet improve outcomes associated with cardiac arrest in the future. These include effective devices and/or drugs (as discussed), methods of early identification (eg, a wearable or implantable ECG alarm); defibrillation by family members of those at moderate or high-risk of cardiac arrest (<http://www.clinicaltrials.gov/show/NCT00047411>); tailored therapy (eg, defibrillation guided by waveform analysis)<sup>52,53</sup>; and faster or simpler methods of implementing and maintaining therapeutic hypothermia. Broad training of the lay public in simpler methods of CPR is particularly promising because it requires less time and is at least as efficacious as traditional methods of training.<sup>54</sup> Training of advanced EMS or hospital providers in advanced cardiac life support may be facilitated by adoption of simulation methods.

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