

Part 6: Electrical Therapies

Automated External Defibrillators, Defibrillation, Cardioversion, and Pacing

2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Mark S. Link, Chair; Dianne L. Atkins; Rod S. Passman; Henry R. Halperin; Ricardo A. Samson; Roger D. White; Michael T. Cudnik; Marc D. Berg; Peter J. Kudenchuk; Richard E. Kerber

Overview

This chapter presents guidelines for defibrillation with manual defibrillators and automated external defibrillators (AEDs), synchronized cardioversion, and pacing. AEDs may be used by lay rescuers and healthcare providers as part of basic life support. Manual defibrillation, cardioversion, and pacing are advanced life support therapies.

Defibrillation Plus CPR: A Critical Combination

Early defibrillation is critical to survival from sudden cardiac arrest (SCA) for several reasons¹: the most frequent initial rhythm in out-of-hospital witnessed SCA is ventricular fibrillation (VF),² the treatment for ventricular fibrillation is defibrillation,³ the probability of successful defibrillation diminishes rapidly over time,⁴ and VF tends to deteriorate to asystole over time.^{1,5,6}

Several studies have documented the effects of time to defibrillation and the effects of bystander CPR on survival from SCA. For every minute that passes between collapse and defibrillation, survival rates from witnessed VF SCA decrease 7% to 10% if no CPR is provided.¹ When bystander CPR is provided, the decrease in survival rates is more gradual and averages 3% to 4% per minute from collapse to defibrillation.^{1,2,5,7} CPR can double^{1,3} or triple⁴ survival from witnessed SCA at most intervals to defibrillation.

If bystanders provide immediate CPR, many adults in VF can survive with intact neurologic function, especially if defibrillation is performed within 5 to 10 minutes after SCA.^{8,9} CPR prolongs VF, delays the onset of asystole,^{10–12} and extends the window of time during which defibrillation can occur. Basic CPR alone, however, is unlikely to terminate VF and restore a perfusing rhythm.

New Recommendations to Integrate CPR and AED Use

To treat VF SCA, rescuers must be able to rapidly integrate CPR with use of the AED. To give the victim the best chance of survival, 3 actions must occur within the first moments of a

cardiac arrest¹: activation of the emergency medical services (EMS) system,² provision of CPR, and operation of an AED.³ When 2 or more rescuers are present, activation of EMS and initiation of CPR can occur simultaneously.

Delays to either the start of CPR or the start of defibrillation reduce survival from SCA. In the 1990s, some predicted that CPR could be rendered obsolete by the widespread development of community AED programs. However, Cobb⁹ noted that as more of Seattle's first responders were equipped with AEDs, survival rates from SCA unexpectedly fell. This decline was attributed to reduced emphasis on CPR, and there is growing evidence to support this view. Part 5: "Adult Basic Life Support" summarizes the evidence on the importance of provision of high-quality CPR (including chest compressions of adequate rate and depth, allowing full chest recoil after each compression and minimizing interruptions in compressions).

Two critical questions about integration of CPR with defibrillation were evaluated during the 2010 International Consensus Conference on CPR and Emergency Cardiovascular Care.¹³ The first question concerned whether CPR should be provided before defibrillation is attempted. The second question concerned the number of shocks to be delivered in a sequence before the rescuer resumes CPR.

Shock First Versus CPR First

When any rescuer witnesses an out-of-hospital arrest and an AED is immediately available on-site, the rescuer should start CPR and use the AED as soon as possible. Healthcare providers who treat cardiac arrest in hospitals and other facilities with AEDs on-site should provide immediate CPR and should use the AED/defibrillator as soon as it is available. These recommendations are designed to support early CPR and early defibrillation, particularly when an AED is available within moments of the onset of SCA.

In studies in which EMS call-to-arrival intervals were 4⁹ to 5⁸ minutes or longer, 1 ½ to 3 minutes of CPR before defibrillation increased the rate of initial resuscitation (return of spontaneous circulation or ROSC), survival to hospital discharge,^{8,9} and 1-year survival⁸ when compared with immediate defibrillation

The American Heart Association requests that this document be cited as follows: Link MS, Atkins DL, Passman RS, Halperin HR, Samson RA, White RD, Cudnik MT, Berg MD, Kudenchuk PJ, Kerber RE. Part 6: electrical therapies: automated external defibrillators, defibrillation, cardioversion, and pacing: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122(suppl 3):S706–S719.

(*Circulation*. 2010;122[suppl 3]:S706–S719.)

© 2010 American Heart Association, Inc.

Circulation is available at <http://circ.ahajournals.org>

DOI: 10.1161/CIRCULATIONAHA.110.970954

for VF SCA. However, in 2 randomized controlled trials,^{14,15} a period of 1 ½ to 3 minutes of CPR by EMS personnel before defibrillation did not improve ROSC or survival to hospital discharge in patients with out-of-hospital VF or pulseless ventricular tachycardia (VT) compared with immediate defibrillation, regardless of EMS response interval, in systems with low overall survival. In 1 retrospective before/after study,¹⁶ immediate CPR by EMS personnel was associated with no significant difference in survival to discharge but significantly improved neurological status at 30 days or 1 year compared with immediate defibrillation in patients with out-of-hospital VF. In a retrospective observational study,¹⁷ probability of survival was increased if chest compressions were performed during a higher proportion of the initial CPR period as compared to a lower proportion.

When VF is present for more than a few minutes, the myocardium is depleted of oxygen and metabolic substrates. A brief period of chest compressions can deliver oxygen and energy substrates, increasing the likelihood that a shock may terminate VF (defibrillation) and a perfusing rhythm will return (ie, ROSC).¹⁸

When an out-of-hospital cardiac arrest is not witnessed by EMS personnel, EMS may initiate CPR while checking the ECG rhythm and preparing for defibrillation. There is insufficient evidence to determine if 1 ½ to 3 minutes of CPR should be provided prior to defibrillation. CPR should be performed while a defibrillator is being readied (Class I, LOE B). One cycle of CPR consists of 30 compressions and 2 breaths. When compressions are delivered at a rate of about 100 per minute, 5 cycles of CPR should take roughly 2 minutes (range: about 1 ½ to 3 minutes).

EMS system medical directors may consider implementing a protocol that allows EMS responders to provide CPR while preparing for defibrillation of patients found by EMS personnel to be in VF. In practice, however, CPR can be initiated while the AED is being readied.

With in-hospital SCA, there is insufficient evidence to support or refute CPR before defibrillation. However, in monitored patients, the time from VF to defibrillation should be under 3 minutes. When 2 or more rescuers are present, one rescuer should begin CPR while the other activates the emergency response system and prepares the defibrillator.

1-Shock Protocol Versus 3-Shock Sequence

At the time of the 2010 Consensus Conference, there were 2 new published human studies that compared a 1-shock protocol versus a 3-stacked-shock protocol for treatment of VF cardiac arrest. Evidence from these 2 well-conducted pre/post design^{19,20} studies suggested significant survival benefit with the single-shock defibrillation protocol compared with 3-stacked-shock protocols. If 1 shock fails to eliminate VF, the incremental benefit of another shock is low, and resumption of CPR is likely to confer a greater value than another shock. This fact, combined with the data from animal studies documenting harmful effects from interruptions to chest compressions and human studies suggesting a survival benefit with a 1-shock protocol, indicate that it is reasonable to use 1-shock for VF, then immediate CPR (Class IIa, LOE B).

First-shock efficacy for biphasic shocks is comparable or better than 3 monophasic shocks.^{21–25} Although the optimal

energy level for defibrillation using any of the monophasic or biphasic waveforms has not been determined, a recommendation for higher initial energy when using a monophasic waveform was weighed by expert consensus with consideration of the potential negative effects of a high first-shock energy versus the negative effects of prolonged VF. The consensus was that rescuers using monophasic defibrillators should give an initial shock of 360 J; if VF persists after the first shock, second and subsequent shocks of 360 J should be given. This single dose for monophasic shocks is designed to simplify instructions to rescuers but is not a mandate to recall monophasic AEDs for reprogramming. If the monophasic AED being used is programmed to deliver a different first or subsequent dose, that dose is acceptable.

After shock delivery, the rescuer should not delay resumption of chest compressions to recheck the rhythm or pulse. After about 5 cycles of CPR (about 2 minutes, although this time is not firm), ideally ending with compressions, the AED should then analyze the cardiac rhythm and deliver another shock if indicated (Class I, LOE B). If a nonshockable rhythm is detected, the AED should instruct the rescuer to resume CPR immediately, beginning with chest compressions (Class I, LOE B).

Concern that chest compressions in the presence of a postshock organized rhythm might provoke recurrent VF has been expressed by 1 animal and 2 human studies,^{26–28} but this has not been shown to adversely affect survival if the current algorithms are followed.^{19,20}

Furthermore, in animal studies, frequent or long interruptions in precordial chest compressions for rhythm analysis²⁹ or rescue breathing^{30,31} were associated with postresuscitation myocardial dysfunction and reduced survival rates. Data from a prospective observational study showed that interruption in chest compressions is associated with a decreased probability of successful conversion of VF to a perfusing rhythm after shock.³² In a recent clinical observational study of out-of-hospital CPR³³ and an in-hospital study of CPR³⁴ by healthcare providers, chest compressions were performed only for 51%³³ to 76%³⁴ of total CPR time.

The rhythm analysis for a 3-shock sequence performed by commercially available AEDs can result in delays of up to 37 seconds between delivery of the first shock and delivery of the first postshock compression.²⁹ This delay is difficult to justify in light of the first-shock efficacy of >90% reported by current biphasic defibrillators.^{28,35–39}

AED manufacturers should seek innovative methods to decrease the amount of time chest compressions are interrupted for AED operation. Training materials for lay rescuers should emphasize the importance of continued CPR until basic or advanced life support personnel take over CPR or the victim begins to move.

Shortening the interval between the last compression and the shock by even a few seconds can improve shock success (defibrillation and ROSC).^{18,32,40} Thus, it is reasonable for healthcare providers to practice efficient coordination between CPR and defibrillation to minimize the hands-off interval between stopping compression and administering shock (Class IIa, LOE C). For example, when 2 rescuers are present, the rescuer operating the AED should be prepared to deliver a shock as soon as the compressor removes his or her hands from the victim's chest and all rescuers are "clear" of contact with the victim. Rescue

breathing prior to the shock will increase the time from compression to shock, and thus it is reasonable to proceed immediately to shock without rescue breathing (Class IIa, LOE B).

Defibrillation Waveforms and Energy Levels

The term *defibrillation* (shock success) is typically defined as termination of VF for at least 5 seconds following the shock.^{41,42} VF frequently recurs after successful shocks, but this recurrence should not be equated with shock failure.^{21,28}

Shock success using the typical definition of *defibrillation* should not be confused with resuscitation outcomes such as restoration of a perfusing rhythm (ROSC), survival to hospital admission, or survival to hospital discharge.^{41,43} Since resuscitation outcomes, including survival, depend on many variables in addition to shock delivery, defibrillation programs must strive to improve patient survival, not just shock success.

Modern defibrillators are classified according to 2 types of waveforms: monophasic and biphasic. Monophasic waveform defibrillators were introduced first, but biphasic waveforms are used in almost all AEDs and manual defibrillators sold today. Energy levels vary by type of device and manufacturer.

Monophasic Waveform Defibrillators

Monophasic waveforms deliver current of one polarity (ie, direction of current flow). Monophasic waveforms can be further categorized by the rate at which the current pulse decreases to zero. The monophasic damped sinusoidal waveform (MDS) returns to zero gradually, whereas the monophasic truncated exponential waveform (MTE) current returns abruptly (is truncated) to zero current flow.

Few monophasic waveform defibrillators are being manufactured, but many are still in use, and most use MDS waveforms. As noted above, no specific waveform characteristic (either monophasic or biphasic) is consistently associated with a greater incidence of ROSC or higher survival to hospital discharge rates after cardiac arrest.

Biphasic Waveform Defibrillators

Data from both out-of-hospital and in-hospital studies indicate that lower-energy biphasic waveform shocks have equivalent or higher success for termination of VF than either MDS or MTE monophasic waveform shocks.^{21,23,39,44–46} However, the optimal energy for first-shock biphasic waveform defibrillation has not been determined. One study⁴⁷ in which a pulsed biphasic waveform was used showed a first-shock success rate of 90%. There is no new evidence regarding the first-shock success rate with the rectilinear biphasic waveform since publication of the 2005 Guidelines. Several randomized^{21,23,39} and observational studies^{22,48} have shown that defibrillation with biphasic waveforms of relatively low energy (≤ 200 J) is safe and has equivalent or higher efficacy for termination of VF than monophasic waveform shocks of equivalent or higher energy.^{42,49–53}

Evidence from 3 randomized trials^{21,23,39} and 3 other human studies^{22,42,54} suggests that defibrillation with biphasic waveforms improves the short-term outcome of termination of VF, but no individual study has demonstrated improved survival to discharge using biphasic waveforms when compared with studies using monophasic waveforms. There is no human study to support defibrillation with a multiphasic waveform when com-

pared with any biphasic waveform. Data from animal studies suggest that multiphasic waveforms (triphase, quadriphase, or higher) may defibrillate at lower energies and induce less postshock myocardial dysfunction. These results are limited by studies of only short-duration VF (approximately 30 seconds) and lack of human studies for validation of these experimental observations.

Biphasic waveforms are safe and have equivalent or higher efficacy for termination of VF when compared with monophasic waveforms. In the absence of biphasic defibrillators, monophasic defibrillators are acceptable (Class IIb, LOE B). Different biphasic waveforms have not been compared in humans with regard to efficacy. Therefore, for biphasic defibrillators, providers should use the manufacturer's recommended energy dose (eg, initial dose of 120 to 200 J) (Class I, LOE B). If the manufacturer's recommended dose is not known, defibrillation at the maximal dose may be considered (Class IIb, LOE C).

In pediatric defibrillation, there are limited data regarding the lowest effective dose or the upper limit for safe defibrillation. Initial monophasic doses of 2 J/kg are effective in terminating 18% to 50% of VF^{55–57} and 48% of VF using similar doses of biphasic energy.⁵⁷ However, even with higher energies (up to 9 J/kg), defibrillation has been successful with no clear adverse effects.^{58–61} Thus, for pediatric patients, it is acceptable to use an initial dose of 2 to 4 J/kg (Class IIa, LOE C), but for ease of teaching an initial dose of 2 J/kg may be considered. For refractory VF, it is reasonable to increase the dose to 4 J/kg. Subsequent energy levels should be at least 4 J/kg, and higher energy levels may be considered, not to exceed 10 J/kg or the adult maximum dose (Class IIb, LOE C).

Fixed and Escalating Energy

Commercially available biphasic AEDs provide either fixed or escalating energy levels. Multiple prospective human clinical studies^{23,52,53} and retrospective studies^{21,22,39,48,62,63} have failed to identify an optimal biphasic energy level for first or subsequent shocks. Human studies^{50,52} have not demonstrated evidence of harm from any biphasic waveform defibrillation energy up to 360 J, with harm defined as elevated biomarker levels, ECG findings, and reduced ejection fraction. Conversely, several animal studies have shown the potential for myocardial damage with much higher energy shocks.^{64–66} Therefore, it is not possible to make a definitive recommendation for the selected energy for subsequent biphasic defibrillation attempts. However, based on available evidence, we recommend that second and subsequent energy levels should be at least equivalent and higher energy levels may be considered, if available (Class IIb, LOE B).

Current-Based Defibrillation

Modern defibrillators deliver current based on stored energy. Because it is accepted that defibrillation is accomplished by the passage of sufficient current through the heart, the concept of current-based defibrillation is appealing. Energy is a nonphysiologic descriptor of defibrillation despite its entrenchment in traditional jargon. Current-based defibrillation has been assessed^{67,68} and in 1 study was superior to energy-based defibrillation with monophasic waveforms.⁶⁹ This concept merits exploration in light of the variety of biphasic waveforms available that

deliver current in different ways. Peak current amplitude, average current, phasic duration, and phasic current flow need to be examined as determinants of shock efficacy. Transition to current-based defibrillation is timely and should be encouraged.

Clinical studies using MDS waveform shocks have tried to identify the range of current necessary to achieve defibrillation and cardioversion. The optimal current for ventricular defibrillation appears to be 30 to 40 A MDS.⁶⁷ Comparable information on current dose for biphasic waveform shocks is under investigation.

Electrodes

Electrode Placement

Data demonstrate that 4 pad positions (anterolateral, anteroposterior, anterior-left infrascapular, and anterior-right-infrascapular)⁷⁰ are equally effective to treat atrial or ventricular arrhythmias.^{71–75} There are no studies directly pertaining to placement of pads/paddles for defibrillation success with the end point of ROSC. All 4 positions are equally effective in shock success.^{71–74,76–82} Any of the 4 pad positions is reasonable for defibrillation (Class IIa, LOE B). For ease of placement and education, anterolateral is a reasonable default electrode placement (Class IIa, LOE C). Alternative pad positions may be considered based on individual patient characteristics.

Lateral pads/paddles should be placed under breast tissue,⁸³ and hirsute males should be shaved prior to application of pads.^{84,85} Ten studies^{65,81,86–93} indicated that larger pad/paddle size (8 to 12 cm diameter) lowers transthoracic impedance.

Defibrillation With Implanted Cardioverter Defibrillator

If the patient has an implantable cardioverter defibrillator (ICD) that is delivering shocks (ie, the patient's muscles contract in a manner similar to that observed during external defibrillation), allow 30 to 60 seconds for the ICD to complete the treatment cycle before attaching an AED. Occasionally, the analysis and shock cycles of automatic ICDs and AEDs will conflict.⁹⁴ There is the potential for pacemaker or ICD malfunction after defibrillation when the pads are in close proximity to the device.^{95,96} One study with cardioversion⁹⁵ demonstrated that positioning the pads at least 8 cm away did not produce changes in pacing thresholds or sensing measurements. Pacemaker spikes with unipolar pacing may confuse AED software and may prevent VF detection.⁹⁴ The anteroposterior and anterolateral locations are acceptable in patients with these devices. In patients with ICDs or pacemakers, pad/paddle placement should not delay defibrillation. It might be reasonable to avoid placing the pads or paddles over the device (Class IIb, LOE C).

Do not place AED electrode pads directly on top of a transdermal medication patch, (eg, patch containing nitroglycerin, nicotine, analgesics, hormone replacements, antihypertensives) because the patch may block delivery of energy from the electrode pad to the heart and may cause small burns to the skin.⁹⁷ If shock delivery will not be delayed, remove medication patches and wipe the area before attaching the electrode pad (Class IIb, LOE C).

If an unresponsive victim is lying in water or if the victim's chest is covered with water or the victim is extremely diaphoretic, it may be reasonable to remove the victim from water and briskly wipe the chest before attaching electrode

pads and attempting defibrillation (Class IIb, LOE C). AEDs can be used when the victim is lying on snow or ice (Class IIb, LOE C). Attempt to remove excess chest hair by briskly removing an electrode pad (which will remove some hair) or rapidly shaving the chest in that area provided chest compressions are not interrupted and defibrillation is not delayed.

Electrode Size

In 1993 the Association for the Advancement of Medical Instrumentation recommended a minimum electrode size of 50 cm² for individual electrodes.⁹⁸ However, advances in electrode design and chemical composition may soon require modification of this recommendation. For adult defibrillation, both handheld paddle electrodes and self-adhesive pad electrodes 8 to 12 cm in diameter perform well, although defibrillation success may be higher with electrodes 12 cm in diameter rather than with those 8 cm in diameter.^{86,99} Small electrodes (4.3 cm) may be harmful and may cause myocardial necrosis.⁸⁸ When using handheld paddles and gel or pads, rescuers must ensure that the paddle is in full contact with the skin. Even smaller pads have been found to be effective¹⁰⁰ in VF of brief duration. Use of the smallest (pediatric) pads, however, can result in unacceptably high transthoracic impedance in larger children.⁹³ For adults, an electrode size of 8 to 12 cm is reasonable (Class IIa, LOE B).

Transthoracic Impedance

The average adult human impedance is ≈ 70 to 80Ω .^{67,86,101} When transthoracic impedance is too high, a low-energy shock will not generate sufficient current to achieve defibrillation.^{101–103} To reduce transthoracic impedance, the defibrillator operator should use conductive materials. This is accomplished with the use of gel pads or electrode paste with paddles or through the use of self-adhesive pads. No existing data suggest that one of these modalities is better than the others in decreasing impedance.

Automated External Defibrillators

AEDs are sophisticated, reliable computerized devices that use voice and visual prompts to guide lay rescuers and healthcare providers to safely defibrillate VF and (pulseless) rapid ventricular tachycardia (VT) SCA.^{44,46,104,105} In recent clinical trials,^{33,34} modified prototype AEDs recorded information about frequency and depth of chest compressions during CPR. These devices are now commercially available and can prompt rescuers to improve CPR performance.

Lay Rescuer AED Programs

Since 1995 the American Heart Association (AHA) has recommended the development of lay rescuer AED programs to improve survival rates from out-of-hospital SCA.^{106–108} These programs are also known as public access defibrillation or PAD programs. The goal of these programs is to shorten the time from onset of SCA VF/pulseless VT until CPR and shock delivery by ensuring that AEDs and trained lay rescuers are available in public areas where SCA is likely to occur. To maximize the effectiveness of these programs, the AHA has emphasized the importance of organizing, planning, training, linking with the EMS system, and establishing a process of continuous quality improvement.^{109,110}

Studies of lay rescuer AED programs in airports¹¹¹ and casinos^{112,113} and of first-responder programs with police officers^{22,44,46,63,114–116} have shown survival rates of 41% to 74% from out-of-hospital witnessed VF SCA when immediate bystander CPR is provided and defibrillation occurs within about 3 to 5 minutes of collapse.^{70,117a} Other studies^{117b,118} have demonstrated decreased time intervals from collapse to delivery of the first shock when AEDs were used during adult out-of-hospital cardiac arrest. However, if no decrease in time to defibrillation is achieved, then high survival rates are not observed.^{119–121}

In the large prospective randomized trial Public Access Defibrillation Trial (PAD),¹²² lay rescuer CPR + AED programs in targeted public settings doubled the number of survivors from out-of-hospital VF SCA when compared with programs that provided early EMS call and early CPR. The programs included a planned response, lay rescuer training, and frequent retraining/practice. In another large population-based study, AED use prior to EMS arrival resulted in a doubling of survival.¹²³ In a prospective population-based study of >300 000 patients, increased penetration of AEDs resulted in increased defibrillation by bystanders and increased survival compared to historical control.¹²⁴

Lay rescuer AED programs will have the greatest potential impact on survival from SCA if the programs are created in locations where SCA is likely to occur. In the PAD trial, programs were established at sites with a history of at least 1 out-of-hospital cardiac arrest every 2 years or where at least 1 out-of-hospital SCA was predicted during the study period (ie, sites having >250 adults over 50 years of age present for >16 hours/d).¹²² Other data suggest that there is benefit when 1 out-of-hospital arrest is likely every 5 years.^{125,126}

CPR and AED use by public safety first responders (traditional and nontraditional) is recommended to increase survival rates for SCA (Class I, LOE B). Establishment of AED programs in public locations where there is a reasonable likelihood of witnessed cardiac arrest (eg, airports, casinos, and sports facilities) is recommended (Class I, LOE B).

Because the improvement in survival rates in AED programs is affected by the time to CPR and to defibrillation, it is reasonable for sites that deploy AEDs to establish a response plan, train likely responders in CPR and AED use, maintain equipment, and coordinate with local EMS systems (Class IIa, LOE B).^{109,110} Sites without these components are unlikely to demonstrate any improvement in survival rates.¹²⁶

Approximately 80% of out-of-hospital cardiac arrests occur in private or residential settings.¹²⁷ One study¹²⁸ demonstrated that survival was not improved in homes of high-risk individuals equipped with AEDs compared with homes where only CPR training had been provided.

AEDs are of no value for arrest not caused by VF/pulseless VT, and they are not effective for treatment of nonshockable rhythms that may develop after termination of VF. Nonperfusing rhythms are present in most patients after shock delivery,^{22,28,63,129} and in general, CPR is required until a perfusing rhythm returns. Therefore, the AED rescuer should be trained not only to recognize emergencies and use the AED, but also to provide CPR until the AED is retrieved and ready for shock delivery and immediately after shock delivery.

The mere presence of an AED does not ensure that it will be used when SCA occurs. Even in the PAD trial, in which almost 20 000 rescuers were trained to respond to SCA, lay rescuers attempted resuscitation before EMS arrival for only half of the victims of witnessed SCA, and the on-site AED was used for only 34% of the victims who experienced an arrest at locations with AED programs.¹²² These findings suggest that lay rescuers need frequent practice to optimize response to emergencies.

It is reasonable for lay rescuer AED programs to implement processes of continuous quality improvement (Class IIa, LOE C). These quality improvement efforts should use both routine inspections and postevent data (from AED recordings and responder reports) to evaluate the following^{110,130}:

- Performance of the emergency response plan, including accurate time intervals for key interventions (such as collapse to shock or no shock advisory to initiation of CPR), and patient outcome
- Responder performance
- AED function, including accuracy of the ECG rhythm analysis
- Battery status and function
- Electrode pad function and readiness, including expiration date

Automated Rhythm Analysis

AEDs analyze multiple features of the surface ECG signal, including frequency, amplitude, and some integration of frequency and amplitude, such as slope or wave morphology. Filters check for QRS-like signals, radio transmission, or 50- or 60-cycle interference, as well as loose electrodes and poor electrode contact. The AHA has recommended performance goals for AED arrhythmia analysis algorithms, specifying sensitivity and specificity for various arrhythmias.¹³¹

AEDs have been tested extensively both in vitro against libraries of recorded cardiac rhythms and clinically in many field trials in adults^{131,132} and children.^{133–135} They are extremely accurate in rhythm analysis. Although AEDs are not designed to deliver synchronized shocks (ie, cardioversion for VT with pulses), AEDs will recommend a (nonsynchronized) shock for monomorphic and polymorphic VT if the rate and R-wave morphology exceed preset values.

Some devices are programmed to detect spontaneous movement by the patient or others. Prototype defibrillators were used in 2 recent clinical trials evaluating quality of CPR in the out-of-hospital and in-hospital settings, which led to the development of AEDs that prompt rescuers to improve the quality of CPR provided.^{33,34}

AED Use in Children

Cardiac arrest is less common in children than adults, and its causes are more diverse.^{136–139} Although VF is not a common arrhythmia in children, it is observed in 5% to 15% of pediatric and adolescent arrests.^{138,140–143} In these patients rapid defibrillation may improve outcomes.^{143,144} The lowest-energy dose for effective defibrillation in infants and children is not known. The upper limit for safe defibrillation is also not known, but doses >4 J/kg (as high as 9 J/kg) have effectively defibrillated children^{60,61} and pediatric animal models¹⁴⁵ with no significant

adverse effects. Based on adult clinical data^{21,39} and pediatric animal models,^{145–147} biphasic shocks appear to be at least as effective as monophasic shocks and are less harmful than monophasic shocks. As noted above, it is acceptable to use an initial dose of 2 to 4 J/kg (Class IIa, LOE C), but for ease of teaching an initial dose of 2 J/kg may be considered. For refractory VF, it is reasonable to increase the dose to 4 J/kg. Subsequent energy levels should be at least 4 J/kg, and higher energy levels may be considered, not to exceed 10 J/kg or the adult maximum dose (Class IIb, LOE C).

Many AEDs can accurately detect VF in children of all ages^{133–135} and differentiate shockable from nonshockable rhythms with a high degree of sensitivity and specificity.^{133–135} Some AEDs are equipped with pediatric attenuator systems (eg, pad-cable systems or a key) to reduce the delivered energy to a dose suitable for children.

For children 1 to 8 years of age, it is reasonable to use a pediatric dose-attenuator system if one is available (Class IIa, LOE C).^{61,148,149} If the rescuer provides CPR to a child in cardiac arrest and does not have an AED with a pediatric attenuator system, the rescuer should use a standard AED.

For infants (<1 year of age), a manual defibrillator is preferred. If a manual defibrillator is not available, an AED with pediatric attenuation is desirable. If neither is available, an AED without a dose attenuator may be used. AEDs with relatively high-energy doses have been successfully used in infants with minimal myocardial damage and good neurological outcomes (Class IIb, LOE C).^{150,151}

If an AED program is established in systems or institutions that routinely provide care to children, the program should be equipped with AEDs with a pediatric attenuator system. This statement, however, should not be interpreted as a recommendation for or against AED placement in specific locations where children are present. Ideally, healthcare systems that routinely provide care to children at risk for cardiac arrest should have available manual defibrillators capable of dose adjustment.¹⁴⁸

In-Hospital Use of AEDs

At the time of the 2010 Consensus Conference, there were no published in-hospital randomized trials of AEDs versus manual defibrillators. Evidence from 1 study with historic controls,¹⁵² 1 case series,¹⁵³ and 2 retrospective studies^{117,118} indicated higher rates of survival to hospital discharge when AEDs were used to treat adult VF or pulseless VT in the hospital. However, 1 before/after study did not show an improvement in survival to discharge or ROSC when in-hospital AEDs were implemented in noncritical areas of a hospital,¹⁵⁴ and 1 observational study with historical controls observed no improvement in survival to discharge when comparing biphasic AEDs to standard monophasic defibrillators.¹⁵⁵ The Gombotz and Hanefeld studies observed a decrease in the time interval from collapse to first shock delivery as well as increased ROSC and survival.

Defibrillation may be delayed when patients develop SCA in unmonitored hospital beds and in outpatient and diagnostic facilities. In such areas, several minutes may elapse before centralized response teams arrive with the defibrillator, attach it, and deliver shocks.¹⁵⁶ Despite limited evidence, AEDs may be considered for the hospital setting as a way to facilitate early defibrillation (a goal of ≤ 3 minutes from collapse), especially in

areas where staff have no rhythm recognition skills or defibrillators are used infrequently (Class IIb, LOE C).

When hospitals deploy AEDs, first-responding personnel should also receive authorization and training to use an AED, with the goal of providing the first shock for any SCA within 3 minutes of collapse. The objective is to make goals for in-hospital use of AEDs consistent with goals established in the out-of-hospital setting.¹⁵⁷ Early defibrillation capability should be available in ambulatory care facilities, as well as throughout hospital inpatient areas. Hospitals should monitor collapse-to-first shock intervals and resuscitation outcomes.

Fibrillation Waveform Analysis to Predict Outcome

There is evidence that VF waveforms change over time.^{158,159} Several retrospective case series, animal studies, and theoretical models suggest that it is possible to predict, with varying reliability, the success of attempted defibrillation by analyzing the VF waveform.^{18,40,160–177} However, there are currently no prospective studies that have identified optimal waveforms and/or timing. The value of VF waveform analysis to guide defibrillation management is uncertain (Class IIb, LOE C).

“Occult” Versus “False” Asystole

In certain cases of cardiac arrest, it is difficult to be certain whether the rhythm is fine VF or asystole. In 1989, Losek¹⁷⁸ published a retrospective review of initial shock delivery for 49 children (infants through 19 years of age) in asystole compared with no shock delivery for 41 children in asystole and found no improvement in rhythm change, ROSC, or survival in the group that received the shocks. In 1993, the Nine City High-Dose Epinephrine Study Group published an analysis of 77 asystolic patients who received initial shock compared with 117 who received standard therapy.¹⁷⁹ There was a worse outcome of ROSC and survival for those who received shocks. Thus, it is not useful to shock asystole (Class III, LOE B).

Fire Hazard

Several case reports have described fires ignited by sparks from poorly applied defibrillator paddles in the presence of an oxygen-enriched atmosphere.^{180–185} Fires have been reported when ventilator tubing is disconnected from the endotracheal tube and then left adjacent to the patient’s head, blowing oxygen across the chest during attempted defibrillation.^{181,183,185} It may be reasonable for rescuers to take precautions to minimize sparking during attempted defibrillation; try to avoid defibrillation in an oxygen-enriched atmosphere (Class IIb, LOE C).

The use of self-adhesive defibrillation pads and ensuring good pad–chest-wall contact will likely minimize the risk of sparks igniting during defibrillation. If manual paddles are used, gel pads are preferable to electrode pastes and gels, because the pastes and gels can spread between the 2 paddles, creating the potential for a spark (Class IIb, LOE C).

Synchronized Cardioversion

Synchronized cardioversion is shock delivery that is timed (synchronized) with the QRS complex. This synchronization avoids shock delivery during the relative refractory portion of the cardiac cycle, when a shock could produce VF.¹⁸⁶ For

additional information, see Part 8.3: “Management of Symptomatic Bradycardia and Tachycardia.”

Synchronized cardioversion is recommended to treat supraventricular tachycardia due to reentry, atrial fibrillation, atrial flutter, and atrial tachycardia. Synchronized cardioversion is also recommended to treat monomorphic VT with pulses. Cardioversion is not effective for treatment of junctional tachycardia or multifocal atrial tachycardia.

Synchronized cardioversion must not be used for treatment of VF as the device may not sense a QRS wave and thus a shock may not be delivered. Synchronized cardioversion should also not be used for pulseless VT or polymorphic (irregular VT). These rhythms require delivery of high-energy *unsynchronized* shocks (ie, defibrillation doses). Electric therapy for VT is discussed further below. For additional information see Part 8.2: “Management of Cardiac Arrest.”

Supraventricular Tachycardias (Reentry Rhythms)

The recommended initial biphasic energy dose for cardioversion of adult atrial fibrillation is 120 to 200 J (Class IIa, LOE A).^{187–191} If the initial shock fails, providers should increase the dose in a stepwise fashion. Cardioversion of adult atrial flutter and other supraventricular tachycardias generally requires less energy; an initial energy of 50 J to 100 J is often sufficient.¹⁹¹ If the initial shock fails, providers should increase the dose in a stepwise fashion.¹⁰² Adult cardioversion of atrial fibrillation with monophasic waveforms should begin at 200 J and increase in a stepwise fashion if not successful (Class IIa, LOE B).^{187–189} For cardioversion of SVT in children, use an initial dose of 0.5 to 1 J/kg. If unsuccessful, increase the dose up to 2 J/kg (Class IIb, LOE C). For further information, see Part 14: “Pediatric Advanced Life Support.”

Ventricular Tachycardia

The energy dose and timing of shocks for treatment of VT with pulses are determined by the patient’s condition and the morphological characteristics of the VT.¹⁹² Pulseless VT is treated as VF (see Part 8.2: “Management of Cardiac Arrest”). Management of stable VT is summarized in Part 8.3: “Management of Symptomatic Bradycardia and Tachycardia.” Unstable monomorphic (regular) VT with pulses is treated with synchronized cardioversion. Unstable polymorphic (irregular) VT with or without pulses is treated as VF using *unsynchronized* high-energy shocks (ie, defibrillation doses).

Adult monomorphic VT (regular form and rate) with a pulse responds well to monophasic or biphasic waveform cardioversion (synchronized) shocks at initial energies of 100 J. If there is no response to the first shock, it may be reasonable to increase the dose in a stepwise fashion. No studies were identified that addressed this issue. Thus, this recommendation represents expert opinion (Class IIb, LOE C).

For electric cardioversion in children the recommended starting energy dose is 0.5 to 1 J/kg. If that fails, increase the dose up to 2 J/kg (Class I, LOE C). For further information, see Part 14: “Pediatric Advanced Life Support.”

Although synchronized cardioversion is preferred for treatment of an organized ventricular rhythm, for some arrhythmias synchronization is not possible. The many QRS configurations and irregular rates that comprise polymorphic ventricular

tachycardia make it difficult or impossible to reliably synchronize to a QRS complex. If there is any doubt whether monomorphic or polymorphic VT is present in the *unstable* patient, do not delay shock delivery to perform detailed rhythm analysis—provide high-energy unsynchronized shocks (ie, defibrillation doses).

The recommended shock doses for high-energy, *unsynchronized* shocks (defibrillation) with a biphasic or monophasic device are those presented earlier in this section (Defibrillation Waveforms and Energy Levels). After shock delivery, the healthcare provider should be prepared to provide immediate CPR (beginning with chest compressions) and follow the ACLS Cardiac Arrest Algorithm if pulseless arrest develops (for further information see Part 8.2: “Management of Cardiac Arrest”).

Pacing

Pacing is not recommended for patients in asystolic cardiac arrest. Randomized controlled trials^{193–195} and additional studies^{196–202} indicate no improvement in the rate of admission to hospital or survival to hospital discharge when paramedics or physicians attempted to provide pacing in asystolic patients in the prehospital or hospital (emergency department) setting. Pacing is not effective for asystolic cardiac arrest and may delay or interrupt the delivery of chest compressions. Pacing for patients in asystole is not recommended (Class III, LOE B).

In symptomatic bradycardia with a pulse, 2 randomized adult trials comparing transcutaneous pacing to drug therapy showed no difference in survival.^{203,204} It is reasonable for healthcare providers to be prepared to initiate pacing in patients who do not respond to atropine (or second-line drugs if these do not delay definitive management) (Class IIa, LOE B). Immediate pacing might be considered if the patient is severely symptomatic (Class IIb, LOE C). If the patient does not respond to drugs or transcutaneous pacing, transvenous pacing is probably indicated (Class IIa, LOE C). For further information see Part 8.3: “Management of Symptomatic Bradycardia and Tachycardia.”

Maintaining Devices in a State of Readiness

User checklists have been developed to reduce equipment malfunction and operator errors. Failure to properly maintain the defibrillator or power supply is responsible for the majority of reported malfunctions. Many currently available defibrillators do an automated check and display readiness. Checklists are useful when designed to identify and prevent such deficiencies. It is recommended to maintain devices in a state of readiness (Class I, LOE C).

Summary

The recommendations for electrical therapies described in this section are designed to improve survival from SCA and life-threatening arrhythmias. Whenever defibrillation is attempted, rescuers must coordinate high-quality CPR with defibrillation to minimize interruptions in chest compressions and to ensure immediate resumption of chest compressions after shock delivery. The high first-shock efficacy of newer biphasic defibrillators led to the recommendation of single shocks plus immediate CPR instead of 3-shock sequences that were recommended prior to 2005 to treat VF. Further data are needed to refine recommendations for energy levels for defibrillation and cardioversion using biphasic waveforms.

Disclosures

Guidelines Part 6: Electrical Therapies: Writing Group Disclosures

| Writing Group Member | Employment | Research Grant | Other | Speakers' Bureau/Honoraria | Ownership Interest | Consultant/Advisory | |
|----------------------|---|---|------------------|---|---------------------------|-------------------------------------|---|
| | | | Research Support | | | Board | Other |
| Mark S. Link | Tufts Medical Center—MD | None | None | None | None | None | None |
| Dianne L. Atkins | University of Iowa: University and Medical School—Professor *Compensated works sheet editor for the Guidelines 2010 Process. Money is paid approximately 2/3 to my institution and 1/3 to directly me. My salary from my institution is not changed by this reimbursement | None | None | None | None | None | *Serving as defense expert witness |
| Rod S. Passman | Northwestern University—Associate Professor | None | None | None | None | None | None |
| Henry R. Halperin | Johns Hopkins University—Professor | †Zoll Circulation | None | None | *Surgivision Lexmed | †Zoll Circulation *Cardiac Concepts | †State of Hawaii *US Department of Justice |
| Ricardo A. Samson | University of Arizona: clinical care, teaching and research with pediatric cardiology in an academic setting—Professor | None | None | None | None | None | None |
| Roger D. White | Mayo Clinic—staff physician | None | None | None | None | None | None |
| Michael T. Cudnik | The Ohio State University Medical Center—Assistant Professor, Dept of Emergency Medicine | †Current Funding AHA Scientist Development Grant. I am the PI on this 4 year project (July 2008-June 2012) that is evaluating the impact of transport distance, transport time, and hospital level factors on survival from CA. There is no perceived conflict with this project. The money from the AHA goes to the Ohio State Research Foundation. Pending Funding R03 Small Research Grant Program, Funding Agency AHRQ. This grant is pending. It is a 1 year project designed to look at the location of current AEDs in the city of Columbus relative to the location of the out of hospital CA in order to determine the optimal location of AEDs in a community. If funded, the money will go to the Ohio State Research Foundation | None | None | None | None | None |
| Marc D. Berg | University of Arizona/University Physician's Healthcare (UPH): Attending pediatric intensivist and Board Member of UPH. UPH is a physician group of the faculty of the College of Medicine. The Board oversees three distinct entities: the physician group, the UPH managed care plan, and the operations of UPH Hospital.- Associate Prof. of Clinical Pediatrics and Member, BOD | None | None | None | None | None | None |
| Peter J. Kudenchuk | University of Washington - Medical Professor of Medicine; Contracted Associate Medical Director, King County Emergency Medical Services - Associate Medical Director | Resuscitation Outcomes Consortium (NIH) —multicenter study of resuscitation. Funds come to the University of Washington | None | *Network for Continuing Medical Education, Academy for Healthcare Education, Sanofi-Aventis, Pri-Med, Horizon CME, with honoraria | *Sanofi-Aventis, Novartis | None | *Occasional expert witness in medical malpractice cases |

(Continued)

Guidelines Part 6: Electrical Therapies: Writing Group Disclosures, *Continued*

| Writing Group Member | Employment | Research Grant | Other Research Support | Speakers' Bureau/Honoraria | Ownership Interest | Consultant/Advisory Board | Other |
|----------------------|---|----------------|------------------------|--|--------------------|--|---|
| Richard E. Kerber | University of Iowa Hospitals and Clinics: Staff Cardiologist-Professor of Medicine | None | None | *Occasional speaker at Cardiology Grand Rounds at other hospitals. Usual honorarium is \$1000 for such talks, about 3/year. The money is paid by the institution that invites me to speak, and is paid to me personally. I gave a talk several months ago to Philips Medical Co. on my hypothermia research, and provided advice on aspects of defibrillator design \$1000 honorarium; one-time event I am a member of a DSMB of a clinical trial of a new Resuscitation product of Zoll. There have been 2 meetings of this DSMB in the past 2 years, & expect subsequent meetings to review/discuss the trial as data are acquired. Compensation so far about \$2000 | None | *See previous comments about relationships with Philips (one-time) and Zoll (DSMB) | †I have served as an expert witness in lawsuits in the past. Occasionally such suits have involved cardiac resuscitation, although not for several years. |

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

*Modest.

†Significant.

References

- Larsen MP, Eisenberg MS, Cummins RO, Hallstrom AP. Predicting survival from out-of-hospital cardiac arrest: a graphic model. *Ann Emerg Med.* 1993;22:1652-1658.
- Valenzuela TD, Roe DJ, Cretin S, Spaite DW, Larsen MP. Estimating effectiveness of cardiac arrest interventions: a logistic regression survival model. *Circulation.* 1997;96:3308-3313.
- Swor RA, Jackson RE, Cynar M, Sadler E, Basse E, Boji B, Rivera-Rivera EJ, Maher A, Grubb W, Jacobson R, Dalbec DL. Bystander CPR, ventricular fibrillation, and survival in witnessed, unmonitored out-of-hospital cardiac arrest. *Ann Emerg Med.* 1995;25:780-784.
- Holmberg M, Holmberg S, Herlitz J. Incidence, duration and survival of ventricular fibrillation in out-of-hospital cardiac arrest patients in Sweden. *Resuscitation.* 2000;44:7-17.
- Chan PS, Krumholz HM, Nichol G, Nallamothu BK. Delayed time to defibrillation after in-hospital cardiac arrest. *N Engl J Med.* 2008;358:9-17.
- Kudenchuk PJ. Electrical therapies. In: Field JM, Kudenchuk JP, O'Conner RE, Vanden Hoek TL, Bresler MJ, Mattu A, Silvers SM, eds. *The Textbook of Emergency Cardiovascular Care and CPR.* Philadelphia, PA: Lippincott Williams & Wilkins; 2008:362-378.
- Stiell IG, Wells GA, Field B, Spaite DW, Nesbitt LP, De Maio VJ, Nichol G, Cousineau D, Blackburn J, Munkley D, Luinstra-Toohey L, Campeau T, Dagnone E, Lyver M. Advanced cardiac life support in out-of-hospital cardiac arrest. *N Engl J Med.* 2004;351:647-656.
- Wik L, Hansen TB, Fylling F, Steen T, Vaagenes P, Auestad BH, Steen PA. Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation: a randomized trial. *JAMA.* 2003;289:1389-1395.
- Cobb LA, Fahrenbruch CE, Walsh TR, Copass MK, Olsufka M, Breskin M, Hallstrom AP. Influence of cardiopulmonary resuscitation prior to defibrillation in patients with out-of-hospital ventricular fibrillation. *JAMA.* 1999;281:1182-1188.
- Cummins RO, Eisenberg MS, Hallstrom AP, Litwin PE. Survival of out-of-hospital cardiac arrest with early initiation of cardiopulmonary resuscitation. *Am J Emerg Med.* 1985;3:114-119.
- Holmberg M, Holmberg S, Herlitz J. Effect of bystander cardiopulmonary resuscitation in out-of-hospital cardiac arrest patients in Sweden. *Resuscitation.* 2000;47:59-70.
- Waalwijk RA, Tijssen JG, Koster RW. Bystander initiated actions in out-of-hospital cardiopulmonary resuscitation: results from the Amsterdam Resuscitation Study (ARRESUST). *Resuscitation.* 2001;50:273-279.
- Jacobs I, Sunde K, Deakin CD, Hazinski MF, Kerber RE, Koster RW, Morrison LJ, Nolan JP, Sayre MR, on behalf of Defibrillation Chapter Collaborators. Part 6: defibrillation: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Circulation.* 2010;122(suppl 2):S325-S337.
- Baker PW, Conway J, Cotton C, Ashby DT, Smyth J, Woodman RJ, Grantham H. Defibrillation or cardiopulmonary resuscitation first for patients with out-of-hospital cardiac arrests found by paramedics to be in ventricular fibrillation? A randomised control trial. *Resuscitation.* 2008;79:424-431.
- Jacobs IG, Finn JC, Oxer HF, Jelinek GA. CPR before defibrillation in out-of-hospital cardiac arrest: a randomized trial. *Emerg Med Australas.* 2005;17:39-45.
- Hayakawa M, Gando S, Okamoto H, Asai Y, Uegaki S, Makise H. Shortening of cardiopulmonary resuscitation time before the defibrillation worsens the outcome in out-of-hospital VF patients. *Am J Emerg Med.* 2009;27:470-474.
- Christenson J, Andrusiek D, Everson-Stewart S, Kudenchuk P, Hostler D, Powell J, Callaway CW, Bishop D, Vaillancourt C, Davis D, Aufderheide TP, Idris A, Stouffer JA, Stiell I, Berg R. Chest compression fraction determines survival in patients with out-of-hospital ventricular fibrillation. *Circulation.* 2009;120:1241-1247.
- Eftestol T, Wik L, Sunde K, Steen PA. Effects of cardiopulmonary resuscitation on predictors of ventricular fibrillation defibrillation success during out-of-hospital cardiac arrest. *Circulation.* 2004;110:10-15.
- Bobrow BJ, Clark LL, Ewy GA, Chikani V, Sanders AB, Berg RA, Richman PB, Kern KB. Minimally interrupted cardiac resuscitation by emergency medical services for out-of-hospital cardiac arrest. *JAMA.* 2008;299:1158-1165.
- Rea TD, Helbock M, Perry S, Garcia M, Cloyd D, Becker L, Eisenberg M. Increasing use of cardiopulmonary resuscitation during out-of-hospital ventricular fibrillation arrest: survival implications of guideline changes. *Circulation.* 2006;114:2760-2765.
- van Alem AP, Chapman FW, Lank P, Hart AA, Koster RW. A prospective, randomised and blinded comparison of first shock success of monophasic and biphasic waveforms in out-of-hospital cardiac arrest. *Resuscitation.* 2003;58:17-24.

22. Carpenter J, Rea TD, Murray JA, Kudenchuk PJ, Eisenberg MS. Defibrillation waveform and post-shock rhythm in out-of-hospital ventricular fibrillation cardiac arrest. *Resuscitation*. 2003;59:189–196.
23. Morrison LJ, Dorian P, Long J, Vermeulen M, Schwartz B, Sawadsky B, Frank J, Cameron B, Burgess R, Shield J, Bagley P, Mausz V, Brewer JE, Lerman BB. Out-of-hospital cardiac arrest rectilinear biphasic to monophasic damped sine defibrillation waveforms with advanced life support intervention trial (ORBIT). *Resuscitation*. 2005;66:149–157.
24. Kudenchuk PJ, Cobb LA, Copass MK, Olsufka M, Maynard C, Nichol G. Transthoracic incremental monophasic versus biphasic defibrillation by emergency responders (TIMBER): a randomized comparison of monophasic with biphasic waveform ascending energy defibrillation for the resuscitation of out-of-hospital cardiac arrest due to ventricular fibrillation. *Circulation*. 2006;114:2010–2018.
25. Leng CT, Paradis NA, Calkins H, Berger RD, Lardo AC, Rent KC, Halperin HR. Resuscitation after prolonged ventricular fibrillation with use of monophasic and biphasic waveform pulses for external defibrillation. *Circulation*. 2000;101:2968–2974.
26. Osorio J, Dossdall DJ, Robichaux RP Jr, Tabereaux PB, Ideker RE. In a swine model, chest compressions cause ventricular capture and, by means of a long-short sequence, ventricular fibrillation. *Circ Arrhythm Electrophysiol*. 2008;1:282–289.
27. Berdowski J, Tijssen JG, Koster RW. Chest compressions cause recurrence of ventricular fibrillation after the first successful conversion by defibrillation in out-of-hospital cardiac arrest. *Circ Arrhythm Electrophysiol*. 3:72–78.
28. Hess EP, White RD. Ventricular fibrillation is not provoked by chest compression during post-shock organized rhythms in out-of-hospital cardiac arrest. *Resuscitation*. 2005;66:7–11.
29. Yu T, Weil MH, Tang W, Sun S, Klouche K, Povoas H, Bisera J. Adverse outcomes of interrupted precordial compression during automated defibrillation. *Circulation*. 2002;106:368–372.
30. Berg RA, Sanders AB, Kern KB, Hilwig RW, Heidenreich JW, Porter ME, Ewy GA. Adverse hemodynamic effects of interrupting chest compressions for rescue breathing during cardiopulmonary resuscitation for ventricular fibrillation cardiac arrest. *Circulation*. 2001;104:2465–2470.
31. Kern KB, Hilwig RW, Berg RA, Sanders AB, Ewy GA. Importance of continuous chest compressions during cardiopulmonary resuscitation: improved outcome during a simulated single lay-rescuer scenario. *Circulation*. 2002;105:645–649.
32. Eftestol T, Sunde K, Steen PA. Effects of interrupting precordial compressions on the calculated probability of defibrillation success during out-of-hospital cardiac arrest. *Circulation*. 2002;105:2270–2273.
33. Wik L, Kramer-Johansen J, Myklebust H, Sorebo H, Svensson L, Fellows B, Steen PA. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. *JAMA*. 2005;293:299–304.
34. Abella BS, Alvarado JP, Myklebust H, Edelson DP, Barry A, O'Hearn N, Vanden Hoek TL, Becker LB. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. *JAMA*. 2005;293:305–310.
35. Bain AC, Swerdlow CD, Love CJ, Ellenbogen KA, Deering TF, Brewer JE, Augostini RS, Tchou PJ. Multicenter study of principles-based waveforms for external defibrillation. *Ann Emerg Med*. 2001;37:5–12.
36. Poole JE, White RD, Kanz KG, Hengstenberg F, Jarrard GT, Robinson JC, Santana V, McKenas DK, Rich N, Rosas S, Merritt S, Magnotto L, Gallagher JV III, Gliner BE, Jorgenson DB, Morgan CB, Dillon SM, Kronmal RA, Bardy GH. Low-energy impedance-compensating biphasic waveforms terminate ventricular fibrillation at high rates in victims of out-of-hospital cardiac arrest. LIFE Investigators. *J Cardiovasc Electrophysiol*. 1997;8:1373–1385.
37. White RD, Blackwell TH, Russell JK, Snyder DE, Jorgenson DB. Transthoracic impedance does not affect defibrillation, resuscitation or survival in patients with out-of-hospital cardiac arrest treated with a non-escalating biphasic waveform defibrillator. *Resuscitation*. 2005;64:63–69.
38. Mittal S, Ayati S, Stein KM, Knight BP, Morady F, Schwartzman D, Cavlovich D, Platia EV, Calkins H, Tchou PJ, Miller JM, Wharton JM, Sung RJ, Slotwiner DJ, Markowitz SM, Lerman BB. Comparison of a novel rectilinear biphasic waveform with a damped sine wave monophasic waveform for transthoracic ventricular defibrillation. ZOLL Investigators. *J Am Coll Cardiol*. 1999;34:1595–1601.
39. Schneider T, Martens PR, Paschen H, Kuisma M, Wolcke B, Gliner BE, Russell JK, Weaver WD, Bossaert L, Chamberlain D. Multicenter, randomized, controlled trial of 150-J biphasic shocks compared with 200- to 360-J monophasic shocks in the resuscitation of out-of-hospital cardiac arrest victims. Optimized Response to Cardiac Arrest (ORCA) Investigators. *Circulation*. 2000;102:1780–1787.
40. Eftestol T, Sunde K, Aase SO, Husoy JH, Steen PA. Predicting outcome of defibrillation by spectral characterization and nonparametric classification of ventricular fibrillation in patients with out-of-hospital cardiac arrest. *Circulation*. 2000;102:1523–1529.
41. White RD. External defibrillation: the need for uniformity in analyzing and reporting results [editorial]. *Ann Emerg Med*. 1998;32:234–236.
42. Gliner BE, White RD. Electrocardiographic evaluation of defibrillation shocks delivered to out-of-hospital sudden cardiac arrest patients. *Resuscitation*. 1999;41:133–144.
43. Cummins RO, Chamberlain DA, Abramson NS, Allen M, Baskett PJ, Becker L, Bossaert L, Deloos HH, Dick WF, Eisenberg MS, Evans TR, Holmberg S, Kerber R, Mullie A, Ornato JP, Sandoe E, Skulberg A, Tunstall-Pedoe H, Swanson R, Thies WH. Recommended guidelines for uniform reporting of data from out-of-hospital cardiac arrest: the Utstein Style. A statement for health professionals from a task force of the American Heart Association, the European Resuscitation Council, the Heart and Stroke Foundation of Canada, and the Australian Resuscitation Council. *Circulation*. 1991;84:960–975.
44. White RD, Hankins DG, Bugliosi TF. Seven years' experience with early defibrillation by police and paramedics in an emergency medical services system. *Resuscitation*. 1998;39:145–151.
45. Cummins RO, Eisenberg MS, Bergner L, Hallstrom A, Hearne T, Murray JA. Automatic external defibrillation: evaluations of its role in the home and in emergency medical services. *Ann Emerg Med*. 1984;13:798–801.
46. White RD, Vukov LF, Bugliosi TF. Early defibrillation by police: initial experience with measurement of critical time intervals and patient outcome. *Ann Emerg Med*. 1994;23:1009–1013.
47. Didon JP, Fontaine G, White RD, Jekova I, Schmid JJ, Cansell A. Clinical experience with a low-energy pulsed biphasic waveform in out-of-hospital cardiac arrest. *Resuscitation*. 2008;76:350–353.
48. Stothert JC, Hatcher TS, Gupton CL, Love JE, Brewer JE. Rectilinear biphasic waveform defibrillation of out-of-hospital cardiac arrest. *Prehosp Emerg Care*. 2004;8:388–392.
49. Schwarz B, Bowdle TA, Jett GK, Mair P, Lindner KH, Aldea GS, Lazzara RG, O'Grady SG, Schmitt PW, Walker RG, Chapman FW, Tacker WA. Biphasic shocks compared with monophasic damped sine wave shocks for direct ventricular defibrillation during open heart surgery. *Anesthesiology*. 2003;98:1063–1069.
50. Higgins SL, Herre JM, Epstein AE, Greer GS, Friedman PL, Gleva ML, Porterfield JG, Chapman FW, Finkel ES, Schmitt PW, Nova RC, Greene HL. A comparison of biphasic and monophasic shocks for external defibrillation. Physio-Control Biphasic Investigators. *Prehosp Emerg Care*. 2000;4:305–313.
51. Martens PR, Russell JK, Wolcke B, Paschen H, Kuisma M, Gliner BE, Weaver WD, Bossaert L, Chamberlain D, Schneider T. Optimal Response to Cardiac Arrest study: defibrillation waveform effects. *Resuscitation*. 2001;49:233–243.
52. Stiell IG, Walker RG, Nesbitt LP, Chapman FW, Cousineau D, Christenson J, Bradford P, Sookram S, Berringer R, Lank P, Wells GA. BIPHASIC Trial: a randomized comparison of fixed lower versus escalating higher energy levels for defibrillation in out-of-hospital cardiac arrest. *Circulation*. 2007;115:1511–1517.
53. Walsh SJ, McClelland AJ, Owens CG, Allen J, Anderson JM, Turner C, Adgey AA. Efficacy of distinct energy delivery protocols comparing two biphasic defibrillators for cardiac arrest. *Am J Cardiol*. 2004;94:378–380.
54. Freeman K, Hendej GW, Shalit M, Stroh G. Biphasic defibrillation does not improve outcomes compared to monophasic defibrillation in out-of-hospital cardiac arrest. *Prehosp Emerg Care*. 2008;12:152–156.
55. Berg MD, Samson RA, Meyer RJ, Clark LL, Valenzuela TD, Berg RA. Pediatric defibrillation doses often fail to terminate prolonged out-of-hospital ventricular fibrillation in children. *Resuscitation*. 2005;67:63–67.
56. Rodriguez-Nunez A, Lopez-Herce J, Garcia C, Dominguez P, Carrillo A, Bellon JM. Pediatric defibrillation after cardiac arrest: initial response and outcome. *Crit Care*. 2006;10:R113.
57. Tibballs J, Carter B, Kiraly NJ, Ragg P, Clifford M. External and internal biphasic direct current shock doses for pediatric ventricular fibrillation and pulseless ventricular tachycardia. *Pediatr Crit Care Med*. 2010. [epub ahead of print].

58. Atkins DL, Hartley LL, York DK. Accurate recognition and effective treatment of ventricular fibrillation by automated external defibrillators in adolescents. *Pediatrics*. 1998;101:393–397.
59. Rossano JW, Quan L, Kenney MA, Rea TD, Atkins DL. Energy doses for treatment of out-of-hospital pediatric ventricular fibrillation. *Resuscitation*. 2006;70:80–89.
60. Gurnett CA, Atkins DL. Successful use of a biphasic waveform automated external defibrillator in a high-risk child. *Am J Cardiol*. 2000;86:1051–1053.
61. Atkins DL, Jorgenson DB. Attenuated pediatric electrode pads for automated external defibrillator use in children. *Resuscitation*. 2005;66:31–37.
62. Gliner BE, Jorgenson DB, Poole JE, White RD, Kanz KG, Lyster TD, Leyde KW, Powers DJ, Morgan CB, Kronmal RA, Bardy GH. Treatment of out-of-hospital cardiac arrest with a low-energy impedance-compensating biphasic waveform automatic external defibrillator. The LIFE Investigators. *Biomed Instrum Technol*. 1998;32:631–644.
63. White RD, Russell JK. Refibrillation, resuscitation and survival in out-of-hospital sudden cardiac arrest victims treated with biphasic automated external defibrillators. *Resuscitation*. 2002;55:17–23.
64. Berg RA, Samson RA, Berg MD, Chapman FW, Hilwig RW, Banville I, Walker RG, Nova RC, Anavy N, Kern KB. Better outcome after pediatric defibrillation dosage than adult dosage in a swine model of pediatric ventricular fibrillation. *J Am Coll Cardiol*. 2005;45:786–789.
65. Killingsworth CR, Melnick SB, Chapman FW, Walker RG, Smith WM, Ideker RE, Walcott GP. Defibrillation threshold and cardiac responses using an external biphasic defibrillator with pediatric and adult adhesive patches in pediatric-sized piglets. *Resuscitation*. 2002;55:177–185.
66. Tang W, Weil MH, Sun S, Jorgenson D, Morgan C, Klouche K, Snyder D. The effects of biphasic waveform design on post-resuscitation myocardial function. *J Am Coll Cardiol*. 2004;43:1228–1235.
67. Lerman BB, DiMarco JP, Haines DE. Current-based versus energy-based ventricular defibrillation: a prospective study. *J Am Coll Cardiol*. 1988;12:1259–1264.
68. Kerber RE, McPherson D, Charbonnier F, Kieso R, Hite P. Automated impedance-based energy adjustment for defibrillation: experimental studies. *Circulation*. 1985;71:136–140.
69. Kerber RE, Kieso RA, Kienzle MG, Olshansky B, Waldo AL, Carlson MD, Wilber DJ, Aschoff AM, Birger S, Charbonnier F. Current-based transthoracic defibrillation. *Am J Cardiol*. 1996;78:1113–1118.
70. England H, Hoffman C, Hodgman T, Singh S, Homoud M, Weinstock J, Link M, Estes NA III. Effectiveness of automated external defibrillators in high schools in greater Boston. *Am J Cardiol*. 2005;95:1484–1486.
71. Boodhoo L, Mitchell AR, Bordoli G, Lloyd G, Patel N, Sulke N. DC cardioversion of persistent atrial fibrillation: a comparison of two protocols. *Int J Cardiol*. 2007;114:16–21.
72. Brazdionyte J, Babarskiene RM, Stanaitiene G. Anterior-posterior versus anterior-lateral electrode position for biphasic cardioversion of atrial fibrillation. *Medicina (Kaunas)*. 2006;42:994–998.
73. Chen CJ, Guo GB. External cardioversion in patients with persistent atrial fibrillation: a reappraisal of the effects of electrode pad position and transthoracic impedance on cardioversion success. *Jpn Heart J*. 2003;44:921–932.
74. Stanaitiene G, Babarskiene RM. [Impact of electrical shock waveform and paddle positions on efficacy of direct current cardioversion for atrial fibrillation]. *Medicina (Kaunas)*. 2008;44:665–672.
75. Krasteva V, Matveev M, Mudrov N, Prokopova R. Transthoracic impedance study with large self-adhesive electrodes in two conventional positions for defibrillation. *Physiol Meas*. 2006;27:1009–1022.
76. Kerber RE, Martins JB, Ferguson DW, Jensen SR, Parke JD, Kieso R, Melton J. Experimental evaluation and initial clinical application of new self-adhesive defibrillation electrodes. *Int J Cardiol*. 1985;8:57–66.
77. Garcia LA, Kerber RE. Transthoracic defibrillation: does electrode adhesive pad position alter transthoracic impedance? *Resuscitation*. 1998;37:139–143.
78. Dodd TE, Deakin CD, Petley GW, Clewlow F. External defibrillation in the left lateral position—a comparison of manual paddles with self-adhesive pads. *Resuscitation*. 2004;63:283–286.
79. Kerber RE, Jensen SR, Grayzel J, Kennedy J, Hoyt R. Elective cardioversion: influence of paddle-electrode location and size on success rates and energy requirements. *N Engl J Med*. 1981;305:658–662.
80. Mathew TP, Moore A, McIntyre M, Harbinson MT, Campbell NP, Adgey AA, Dalzell GW. Randomised comparison of electrode positions for cardioversion of atrial fibrillation. *Heart*. 1999;81:576–579.
81. Camacho MA, Lehr JL, Eisenberg SR. A three-dimensional finite element model of human transthoracic defibrillation: paddle placement and size. *IEEE Trans Biomed Eng*. 1995;42:572–578.
82. Lateef F, Lim SH, Anantharaman V, Lim CS. Changes in chest electrode impedance. *Am J Emerg Med*. 2000;18:381–384.
83. Pagan-Carlo LA, Spencer KT, Robertson CE, Dengler A, Birkett C, Kerber RE. Transthoracic defibrillation: importance of avoiding electrode placement directly on the female breast. *J Am Coll Cardiol*. 1996;27:449–452.
84. Bissing JW, Kerber RE. Effect of shaving the chest of hirsute subjects on transthoracic impedance to self-adhesive defibrillation electrode pads. *Am J Cardiol*. 2000;86:587–589.
85. Sado DM, Deakin CD, Petley GW, Clewlow F. Comparison of the effects of removal of chest hair with not doing so before external defibrillation on transthoracic impedance. *Am J Cardiol*. 2004;93:98–100.
86. Kerber RE, Grayzel J, Hoyt R, Marcus M, Kennedy J. Transthoracic resistance in human defibrillation. Influence of body weight, chest size, serial shocks, paddle size and paddle contact pressure. *Circulation*. 1981;63:676–682.
87. Connell PN, Ewy GA, Dahl CF, Ewy MD. Transthoracic impedance to defibrillator discharge. Effect of electrode size and electrode-chest wall interface. *J Electrocardiol*. 1973;6:313–M.
88. Dahl CF, Ewy GA, Warner ED, Thomas ED. Myocardial necrosis from direct current countershock: effect of paddle electrode size and time interval between discharges. *Circulation*. 1974;50:956–961.
89. Hoyt R, Grayzel J, Kerber RE. Determinants of intracardiac current in defibrillation. Experimental studies in dogs. *Circulation*. 1981;64:818–823.
90. Thomas ED, Ewy GA, Dahl CF, Ewy MD. Effectiveness of direct current defibrillation: role of paddle electrode size. *Am Heart J*. 1977;93:463–467.
91. Atkins DL, Kerber RE. Pediatric defibrillation: current flow is improved by using “adult” electrode paddles. *Pediatrics*. 1994;94:90–93.
92. Atkins DL, Sirna S, Kieso R, Charbonnier F, Kerber RE. Pediatric defibrillation: importance of paddle size in determining transthoracic impedance. *Pediatrics*. 1988;82:914–918.
93. Samson RA, Atkins DL, Kerber RE. Optimal size of self-adhesive preapplied electrode pads in pediatric defibrillation. *Am J Cardiol*. 1995;75:544–545.
94. Monsieurs KG, Conraads VM, Goethals MP, Snoeck JP, Bossaert LL. Semi-automatic external defibrillation and implanted cardiac pacemakers: understanding the interactions during resuscitation. *Resuscitation*. 1995;30:127–131.
95. Manegold JC, Israel CW, Ehrlich JR, Duray G, Pajitnev D, Wegener FT, Hohnloser SH. External cardioversion of atrial fibrillation in patients with implanted pacemaker or cardioverter-defibrillator systems: a randomized comparison of monophasic and biphasic shock energy application. *Eur Heart J*. 2007;28:1731–1738.
96. Alferness CA. Pacemaker damage due to external countershock in patients with implanted cardiac pacemakers. *Pacing Clin Electrophysiol*. 1982;5:457–458.
97. Panacek EA, Munger MA, Rutherford WF, Gardner SF. Report of nitropatch explosions complicating defibrillation. *Am J Emerg Med*. 1992;10:128–129.
98. *American National Standard: Automatic External Defibrillators and Remote Controlled Defibrillators (DF39)*. Arlington, VA: Association for the Advancement of Medical Instrumentation; 1993.
99. Stults KR, Brown DD, Cooley F, Kerber RE. Self-adhesive monitor/defibrillation pads improve prehospital defibrillation success. *Ann Emerg Med*. 1987;16:872–877.
100. Wilson RF, Sirna S, White CW, Kerber RE. Defibrillation of high-risk patients during coronary angiography using self-adhesive, preapplied electrode pads. *Am J Cardiol*. 1987;60:380–382.
101. Kerber RE, Kouba C, Martins J, Kelly K, Low R, Hoyt R, Ferguson D, Bailey L, Bennett P, Charbonnier F. Advance prediction of transthoracic impedance in human defibrillation and cardioversion: importance of impedance in determining the success of low-energy shocks. *Circulation*. 1984;70:303–308.
102. Kerber RE, Martins JB, Kienzle MG, Constantin L, Olshansky B, Hopson R, Charbonnier F. Energy, current, and success in defibrillation

- and cardioversion: clinical studies using an automated impedance-based method of energy adjustment. *Circulation*. 1988;77:1038–1046.
103. Dalzell GW, Cunningham SR, Anderson J, Adgey AA. Electrode pad size, transthoracic impedance and success of external ventricular defibrillation. *Am J Cardiol*. 1989;64:741–744.
 104. Cummins RO, Eisenberg M, Bergner L, Murray JA. Sensitivity, accuracy, and safety of an automatic external defibrillator. *Lancet*. 1984;2:318–320.
 105. Davis EA, Moseoso VN Jr. Performance of police first responders in utilizing automated external defibrillation on victims of sudden cardiac arrest. *Prehosp Emerg Care*. 1998;2:101–107.
 106. Weisfeldt ML, Kerber RE, McGoldrick RP, Moss AJ, Nichol G, Ornato JP, Palmer DG, Riegel B, Smith SCJ. American Heart Association Report on the Public Access Defibrillation Conference December 8–10, 1994. Automatic External Defibrillation Task Force. *Circulation*. 1995;92:2740–2747.
 107. Weisfeldt ML, Kerber RE, McGoldrick RP, Moss AJ, Nichol G, Ornato JP, Palmer DG, Riegel B, Smith SC Jr. Public access defibrillation. A statement for healthcare professionals from the American Heart Association Task Force on Automatic External Defibrillation. *Circulation*. 1995;92:2763.
 108. Nichol G, Hallstrom AP, Ornato JP, Riegel B, Stiell IG, Valenzuela T, Wells GA, White RD, Weisfeldt ML. Potential cost-effectiveness of public access defibrillation in the United States. *Circulation*. 1998;97:1315–1320.
 109. American Heart Association in collaboration with International Liaison Committee on Resuscitation. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2000;102(suppl):I1–I384.
 110. Hazinski MF, Idris AH, Kerber RE, Epstein A, Atkins D, Tang W, Lurie K. Lay rescuer automated external defibrillator (“public access defibrillation”) programs: lessons learned from an international multicenter trial: advisory statement from the American Heart Association Emergency Cardiovascular Committee; the Council on Cardiopulmonary, Perioperative, and Critical Care; and the Council on Clinical Cardiology. *Circulation*. 2005;111:3336–3340.
 111. Caffrey SL, Willoughby PJ, Pepe PE, Becker LB. Public use of automated external defibrillators. *N Engl J Med*. 2002;347:1242–1247.
 112. Valenzuela TD, Bjerke HS, Clark LL, Hardman R, Spaite DW, Nichol G. Rapid defibrillation by nontraditional responders: the Casino Project. *Acad Emerg Med*. 1998;5:414–415.
 113. Valenzuela TD, Roe DJ, Nichol G, Clark LL, Spaite DW, Hardman RG. Outcomes of rapid defibrillation by security officers after cardiac arrest in casinos. *N Engl J Med*. 2000;343:1206–1209.
 114. White RD, Asplin BR, Bugliosi TF, Hankins DG. High discharge survival rate after out-of-hospital ventricular fibrillation with rapid defibrillation by police and paramedics. *Ann Emerg Med*. 1996;28:480–485.
 115. White RD. Early out-of-hospital experience with an impedance-compensating low-energy biphasic waveform automatic external defibrillator. *J Interv Card Electrophysiol*. 1997;1:203–208.
 116. White RD, Bunch TJ, Hankins DG. Evolution of a community-wide early defibrillation programme experience over 13 years using police/fire personnel and paramedics as responders. *Resuscitation*. 2005;65:279–283.
 - 117a. Rea TD, Olsufka M, Bemis B, White L, Yin L, Becker L, Copass M, Eisenberg M, Cobb L. A population based investigation of public access defibrillation: Rose of emergency medical services care. *Resuscitation*. 2010;81:163–167.
 - 117b. Gombotz H, Weh B, Mitterdorfer W, Rehak P. In-hospital cardiac resuscitation outside the ICU by nursing staff equipped with automated external defibrillators—the first 500 cases. *Resuscitation*. 2006;70:416–422.
 118. Hanefeld C, Lichte C, Mentges-Schroter I, Sirtl C, Mugege A. Hospital-wide first-responder automated external defibrillator programme: 1 year experience. *Resuscitation*. 2005;66:167–170.
 119. Groh WJ, Newman MM, Beal PE, Fineberg NS, Zipes DP. Limited response to cardiac arrest by police equipped with automated external defibrillators: lack of survival benefit in suburban and rural Indiana—the police as responder automated defibrillation evaluation (PARADE). *Acad Emerg Med*. 2001;8:324–330.
 120. de Vries W, van Alem AP, de Vos R, van Oostrom J, Koster RW. Trained first-responders with an automated external defibrillator: how do they perform in real resuscitation attempts? *Resuscitation*. 2005;64:157–161.
 121. Sayre MR, Evans J, White LJ, Brennan TD. Providing automated external defibrillators to urban police officers in addition to a fire department rapid defibrillation program is not effective. *Resuscitation*. 2005;66:189–196.
 122. The Public Access Defibrillation Trial Investigators. Public-access defibrillation and survival after out-of-hospital cardiac arrest. *N Engl J Med*. 2004;351:637–646.
 123. Weisfeldt ML, Sitlani CM, Ornato JP, Rea T, Aufderheide TP, Davis D, Dreyer J, Hess EP, Jui J, Maloney J, Sopko G, Powell J, Nichol G, Morrison LJ. Survival after application of automatic external defibrillators before arrival of the emergency medical system: evaluation in the resuscitation outcomes consortium population of 21 million. *J Am Coll Cardiol*. 2010;55:1713–1720.
 124. Kitamura T, Iwami T, Kawamura T, Nagao K, Tanaka H, Hiraide A. Nationwide public-access defibrillation in Japan. *N Engl J Med*. 2010;362:994–1004.
 125. Cram P, Vijan S, Fendrick AM. Cost-effectiveness of automated external defibrillator deployment in selected public locations. *J Gen Intern Med*. 2003;18:745–754.
 126. Folke F, Lippert FK, Nielsen SL, Gislason GH, Hansen ML, Schramm TK, Sorensen R, Fosbol EL, Andersen SS, Rasmussen S, Kober L, Torp-Pedersen C. Location of cardiac arrest in a city center: strategic placement of automated external defibrillators in public locations. *Circulation*. 2009;120:510–517.
 127. Becker L, Eisenberg M, Fahrenbruch C, Cobb L. Public locations of cardiac arrest: implications for public access defibrillation. *Circulation*. 1998;97:2106–2109.
 128. Bardy GH, Lee KL, Mark DB, Poole JE, Toff WD, Tonkin AM, Smith W, Dorian P, Packer DL, White RD, Longstreth WT Jr, Anderson J, Johnson G, Bischoff E, Yallop JJ, McNulty S, Ray LD, Clapp-Channing NE, Rosenberg Y, Schron EB. Home use of automated external defibrillators for sudden cardiac arrest. *N Engl J Med*. 2008;358:1793–1804.
 129. Weaver WD, Cobb LA, Copass MK, Hallstrom AP. Ventricular defibrillation: a comparative trial using 175-J and 320-J shocks. *N Engl J Med*. 1982;307:1101–1106.
 130. 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2005;112(suppl):IV1–203.
 131. Kerber RE, Becker LB, Bourland JD, Cummins RO, Hallstrom AP, Michos MB, Nichol G, Ornato JP, Thies WH, White RD, Zuckerman BD. Automatic external defibrillators for public access defibrillation: recommendations for specifying and reporting arrhythmia analysis algorithm performance, incorporating new waveforms, and enhancing safety. A statement for health professionals from the American Heart Association Task Force on Automatic External Defibrillation, Subcommittee on AED Safety and Efficacy. *Circulation*. 1997;95:1677–1682.
 132. Dickey W, Dalzell GW, Anderson JM, Adgey AA. The accuracy of decision-making of a semi-automatic defibrillator during cardiac arrest. *Eur Heart J*. 1992;13:608–615.
 133. Atkinson E, Mikysa B, Conway JA, Parker M, Christian K, Deshpande J, Knilans TK, Smith J, Walker C, Stickney RE, Hampton DR, Hazinski MF. Specificity and sensitivity of automated external defibrillator rhythm analysis in infants and children. *Ann Emerg Med*. 2003;42:185–196.
 134. Cecchin F, Jorgenson DB, Berul CI, Perry JC, Zimmerman AA, Duncan BW, Lupinetti FM, Snyder D, Lyster TD, Rosenthal GL, Cross B, Atkins DL. Is arrhythmia detection by automatic external defibrillator accurate for children? Sensitivity and specificity of an automatic external defibrillator algorithm in 696 pediatric arrhythmias. *Circulation*. 2001;103:2483–2488.
 135. Atkins DL, Scott WA, Blaufox AD, Law IH, Dick M II, Geheb F, Sobh J, Brewer JE. Sensitivity and specificity of an automated external defibrillator algorithm designed for pediatric patients. *Resuscitation*. 2008;76:168–174.
 136. Kuisma M, Suominen P, Korpela R. Paediatric out-of-hospital cardiac arrests: epidemiology and outcome. *Resuscitation*. 1995;30:141–150.
 137. Sirbaugh PE, Pepe PE, Shook JE, Kimball KT, Goldman MJ, Ward MA, Mann DM. A prospective, population-based study of the demographics, epidemiology, management, and outcome of out-of-hospital pediatric cardiopulmonary arrest. *Ann Emerg Med*. 1999;33:174–184.
 138. Hickey RW, Cohen DM, Strausbaugh S, Dietrich AM. Pediatric patients requiring CPR in the prehospital setting. *Ann Emerg Med*. 1995;25:495–501.
 139. Atkins DL, Everson-Stewart S, Sears GK, Daya M, Osmond MH, Warden CR, Berg RA. Epidemiology and outcomes from out-of-hospital

- cardiac arrest in children: the Resuscitation Outcomes Consortium Epistudy—Cardiac Arrest. *Circulation*. 2009;119:1484–1491.
140. Appleton GO, Cummins RO, Larson MP, Graves JR. CPR and the single rescuer: at what age should you “call first” rather than “call fast”? *Ann Emerg Med*. 1995;25:492–494.
 141. Ronco R, King W, Donley DK, Tilden SJ. Outcome and cost at a children’s hospital following resuscitation for out-of-hospital cardiopulmonary arrest. *Arch Pediatr Adolesc Med*. 1995;149:210–214.
 142. Losek JD, Hennes H, Glaeser P, Hendley G, Nelson DB. Prehospital care of the pulseless, nonbreathing pediatric patient. *Am J Emerg Med*. 1987;5:370–374.
 143. Mogayzel C, Quan L, Graves JR, Tiedeman D, Fahrenbruch C, Herndon P. Out-of-hospital ventricular fibrillation in children and adolescents: causes and outcomes. *Ann Emerg Med*. 1995;25:484–491.
 144. Safranek DJ, Eisenberg MS, Larsen MP. The epidemiology of cardiac arrest in young adults. *Ann Emerg Med*. 1992;21:1102–1106.
 145. Berg RA, Chapman FW, Berg MD, Hilwig RW, Banville I, Walker RG, Nova RC, Sherrill D, Kern KB. Attenuated adult biphasic shocks compared with weight-based monophasic shocks in a swine model of prolonged pediatric ventricular fibrillation. *Resuscitation*. 2004;61:189–197.
 146. Tang W, Weil MH, Jorgenson D, Klouche K, Morgan C, Yu T, Sun S, Snyder D. Fixed-energy biphasic waveform defibrillation in a pediatric model of cardiac arrest and resuscitation. *Crit Care Med*. 2002;30:2736–2741.
 147. Clark CB, Zhang Y, Davies LR, Karlsson G, Kerber RE. Pediatric transthoracic defibrillation: biphasic versus monophasic waveforms in an experimental model. *Resuscitation*. 2001;51:159–163.
 148. Samson RA, Berg RA, Bingham R, Biarent D, Coovadia A, Hazinski MF, Hickey RW, Nadkarni V, Nichol G, Tibballs J, Reis AG, Tse S, Zideman D, Potts J, Uzark K, Atkins D. Use of automated external defibrillators for children: an update: an advisory statement from the pediatric advanced life support task force, International Liaison Committee on Resuscitation. *Circulation*. 2003;107:3250–3255.
 149. Jorgenson D, Morgan C, Snyder D, Griesser H, Solosko T, Chan K, Skarr T. Energy attenuator for pediatric application of an automated external defibrillator. *Crit Care Med*. 2002;30(suppl):S145–S147.
 150. Bar-Cohen Y, Walsh EP, Love BA, Cecchin F. First appropriate use of automated external defibrillator in an infant. *Resuscitation*. 2005;67:135–137.
 151. Konig B, Bengler J, Goldsworthy L. Automatic external defibrillation in a 6 year old. *Arch Dis Child*. 2005;90:310–311.
 152. Zafari AM, Zarter SK, Heggen V, Wilson P, Taylor RA, Reddy K, Backscheider AG, Dudley SC Jr. A program encouraging early defibrillation results in improved in-hospital resuscitation efficacy. *J Am Coll Cardiol*. 2004;44:846–852.
 153. Destro A, Marzaloni M, Sermasi S, Rossi F. Automatic external defibrillators in the hospital as well? *Resuscitation*. 1996;31:39–43.
 154. Smith M. Service is improving everywhere . . . but what about EMS? *EMS Mag*. 2009;38:26.
 155. Forcina MS, Farhat AY, O’Neil WW, Haines DE. Cardiac arrest survival after implementation of automated external defibrillator technology in the in-hospital setting. *Crit Care Med*. 2009;37:1229–1236.
 156. Kaye W, Mancini ME, Richards N. Organizing and implementing a hospital-wide first-responder automated external defibrillation program: strengthening the in-hospital chain of survival. *Resuscitation*. 1995;30:151–156.
 157. Peberdy MA, Kaye W, Ornato JP, Larkin GL, Nadkarni V, Mancini ME, Berg RA, Nichol G, Lane-Trullitt T. Cardiopulmonary resuscitation of adults in the hospital: a report of 14720 cardiac arrests from the National Registry of Cardiopulmonary Resuscitation. *Resuscitation*. 2003;58:297–308.
 158. Asano Y, Davidenko JM, Baxter WT, Gray RA, Jalife J. Optical mapping of drug-induced polymorphic arrhythmias and torsade de pointes in the isolated rabbit heart. *J Am Coll Cardiol*. 1997;29:831–842.
 159. Gray RA, Jalife J, Panfilov A, Baxter WT, Cabo C, Davidenko JM, Pertsov AM. Nonstationary vortexlike reentrant activity as a mechanism of polymorphic ventricular tachycardia in the isolated rabbit heart. *Circulation*. 1995;91:2454–2469.
 160. Callaway CW, Sherman LD, Mosesso VN Jr, Dietrich TJ, Holt E, Clarkson MC. Scaling exponent predicts defibrillation success for out-of-hospital ventricular fibrillation cardiac arrest. *Circulation*. 2001;103:1656–1661.
 161. Weaver WD, Cobb LA, Dennis D, Ray R, Hallstrom AP, Copass MK. Amplitude of ventricular fibrillation waveform and outcome after cardiac arrest. *Ann Intern Med*. 1985;102:53–55.
 162. Brown CG, Dzwonczyk R. Signal analysis of the human electrocardiogram during ventricular fibrillation: frequency and amplitude parameters as predictors of successful countershock. *Ann Emerg Med*. 1996;27:184–188.
 163. Callahan M, Braun O, Valentine W, Clark DM, Zegans C. Prehospital cardiac arrest treated by urban first-responders: profile of patient response and prediction of outcome by ventricular fibrillation waveform. *Ann Emerg Med*. 1993;22:1664–1677.
 164. Strohmenger HU, Lindner KH, Brown CG. Analysis of the ventricular fibrillation ECG signal amplitude and frequency parameters as predictors of countershock success in humans. *Chest*. 1997;111:584–589.
 165. Strohmenger HU, Eftestol T, Sunde K, Wenzel V, Mair M, Ulmer H, Lindner KH, Steen PA. The predictive value of ventricular fibrillation electrocardiogram signal frequency and amplitude variables in patients with out-of-hospital cardiac arrest. *Anesth Analg*. 2001;93:1428–1433.
 166. Podbregar M, Kovacic M, Podbregar-Mars A, Brezocnik M. Predicting defibrillation success by ‘genetic’ programming in patients with out-of-hospital cardiac arrest. *Resuscitation*. 2003;57:153–159.
 167. Menegazzi JJ, Callaway CW, Sherman LD, Hostler DP, Wang HE, Fertig KC, Logue ES. Ventricular fibrillation scaling exponent can guide timing of defibrillation and other therapies. *Circulation*. 2004;109:926–931.
 168. Povoas HP, Weil MH, Tang W, Bisera J, Klouche K, Barbatsis A. Predicting the success of defibrillation by electrocardiographic analysis. *Resuscitation*. 2002;53:77–82.
 169. Noc M, Weil MH, Tang W, Sun S, Pernat A, Bisera J. Electrocardiographic prediction of the success of cardiac resuscitation. *Crit Care Med*. 1999;27:708–714.
 170. Strohmenger HU, Lindner KH, Keller A, Lindner IM, Pfenninger EG. Spectral analysis of ventricular fibrillation and closed-chest cardiopulmonary resuscitation. *Resuscitation*. 1996;33:155–161.
 171. Noc M, Weil MH, Gazmuri RJ, Sun S, Biscera J, Tang W. Ventricular fibrillation voltage as a monitor of the effectiveness of cardiopulmonary resuscitation. *J Lab Clin Med*. 1994;124:421–426.
 172. Lightfoot CB, Nremt P, Callaway CW, Hsieh M, Fertig KC, Sherman LD, Menegazzi JJ. Dynamic nature of electrocardiographic waveform predicts rescue shock outcome in porcine ventricular fibrillation. *Ann Emerg Med*. 2003;42:230–241.
 173. Marn-Pernat A, Weil MH, Tang W, Pernat A, Bisera J. Optimizing timing of ventricular defibrillation. *Crit Care Med*. 2001;29:2360–2365.
 174. Hamprecht FA, Achleitner U, Krismer AC, Lindner KH, Wenzel V, Strohmenger HU, Thiel W, van Gunsteren WF, Amann A. Fibrillation power, an alternative method of ECG spectral analysis for prediction of countershock success in a porcine model of ventricular fibrillation. *Resuscitation*. 2001;50:287–296.
 175. Amann A, Achleitner U, Antretter H, Bonatti JO, Krismer AC, Lindner KH, Rieder J, Wenzel V, Voelckel WG, Strohmenger HU. Analysing ventricular fibrillation ECG-signals and predicting defibrillation success during cardiopulmonary resuscitation employing N(alpha)-histograms. *Resuscitation*. 2001;50:77–85.
 176. Brown CG, Griffith RF, Van Ligten P, Hoekstra J, Nejman G, Mitchell L, Dzwonczyk R. Median frequency—a new parameter for predicting defibrillation success rate. *Ann Emerg Med*. 1991;20:787–789.
 177. Amann A, Rheinberger K, Achleitner U, Krismer AC, Lingnau W, Lindner KH, Wenzel V. The prediction of defibrillation outcome using a new combination of mean frequency and amplitude in porcine models of cardiac arrest. *Anesth Analg*. 2002;95:716–722.
 178. Losek JD, Hennes H, Glaeser PW, Smith DS, Hendley G. Prehospital countershock treatment of pediatric asystole. *Am J Emerg Med*. 1989;7:571–575.
 179. Martin DR, Gavin T, Bianco J, Brown CG, Stueven H, Pepe PE, Cummins RO, Gonzalez E, Jastremski M. Initial countershock in the treatment of asystole. *Resuscitation*. 1993;26:63–68.
 180. Miller PH. Potential fire hazard in defibrillation. *JAMA*. 1972;221:192.
 181. Hummel RS III, Ornato JP, Weinberg SM, Clarke AM. Spark-generating properties of electrode gels used during defibrillation. A potential fire hazard. *JAMA*. 1988;260:3021–3024.
 182. Fires from defibrillation during oxygen administration. *Health Devices*. 1994;23:307–309.
 183. Lefever J, Smith A. Risk of fire when using defibrillation in an oxygen enriched atmosphere. *Medical Devices Agency Safety Notices*. 1995;3:1–3.

184. Ward ME. Risk of fires when using defibrillators in an oxygen enriched atmosphere. *Resuscitation*. 1996;31:173.
185. Theodorou AA, Gutierrez JA, Berg RA. Fire attributable to a defibrillation attempt in a neonate. *Pediatrics*. 2003;112:677–679.
186. Lown B. Electrical reversion of cardiac arrhythmias. *Br Heart J*. 1967;29:469–489.
187. Mittal S, Ayati S, Stein KM, Schwartzman D, Cavlovich D, Tchou PJ, Markowitz SM, Slotwiner DJ, Scheiner MA, Lerman BB. Transthoracic cardioversion of atrial fibrillation: comparison of rectilinear biphasic versus damped sine wave monophasic shocks. *Circulation*. 2000;101:1282–1287.
188. Page RL, Kerber RE, Russell JK, Trouton T, Waktare J, Gallik D, Olgin JE, Ricard P, Dalzell GW, Reddy R, Lazzara R, Lee K, Carlson M, Halperin B, Bardy GH. Biphasic versus monophasic shock waveform for conversion of atrial fibrillation: the results of an international randomized, double-blind multicenter trial. *J Am Coll Cardiol*. 2002;39:1956–1963.
189. Scholten M, Szili-Torok T, Klootwijk P, Jordaens L. Comparison of monophasic and biphasic shocks for transthoracic cardioversion of atrial fibrillation. *Heart*. 2003;89:1032–1034.
190. Glover BM, Walsh SJ, McCann CJ, Moore MJ, Manoharan G, Dalzell GW, McAllister A, McClements B, McEneaney DJ, Trouton TG, Mathew TP, Adgey AA. Biphasic energy selection for transthoracic cardioversion of atrial fibrillation. The BEST AF Trial. *Heart*. 2008;94:884–887.
191. Reisinger J, Gstrein C, Winter T, Zeindlhofer E, Hollinger K, Mori M, Schiller A, Winter A, Geiger H, Siostrzonek P. Optimization of initial energy for cardioversion of atrial tachyarrhythmias with biphasic shocks. *Am J Emerg Med*. 2010;28:159–165.
192. Kerber RE, Kienle MG, Olshansky B, Waldo AL, Wilber D, Carlson MD, Aschoff AM, Birger S, Fugatt L, Walsh S. Ventricular tachycardia rate and morphology determine energy and current requirements for transthoracic cardioversion. *Circulation*. 1992;85:158–163.
193. Hedges JR, Syverud SA, Dalsey WC, Feero S, Easter R, Shultz B. Pre-hospital trial of emergency transcutaneous cardiac pacing. *Circulation*. 1987;76:1337–1343.
194. Barthell E, Troiano P, Olson D, Stueven HA, Hendley G. Prehospital external cardiac pacing: a prospective, controlled clinical trial. *Ann Emerg Med*. 1988;17:1221–1226.
195. Cummins RO, Graves JR, Larsen MP, Hallstrom AP, Hearne TR, Ciliberti J, Nicola RM, Horan S. Out-of-hospital transcutaneous pacing by emergency medical technicians in patients with asystolic cardiac arrest. *N Engl J Med*. 1993;328:1377–1382.
196. Ornato JP, Peberdy MA. The mystery of bradyasystole during cardiac arrest. *Ann Emerg Med*. 1996;27:576–587.
197. Niemann JT, Adomian GE, Garner D, Rosborough JP. Endocardial and transcutaneous cardiac pacing, calcium chloride, and epinephrine in postcountershock asystole and bradycardias. *Crit Care Med*. 1985;13:699–704.
198. Quan L, Graves JR, Kinder DR, Horan S, Cummins RO. Transcutaneous cardiac pacing in the treatment of out-of-hospital pediatric cardiac arrests. *Ann Emerg Med*. 1992;21:905–909.
199. Dalsey WC, Syverud SA, Hedges JR. Emergency department use of transcutaneous pacing for cardiac arrests. *Crit Care Med*. 1985;13:399–401.
200. Knowlton AA, Falk RH. External cardiac pacing during in-hospital cardiac arrest. *Am J Cardiol*. 1986;57:1295–1298.
201. Ornato JP, Carveth WL, Windle JR. Pacemaker insertion for prehospital bradyasystolic cardiac arrest. *Ann Emerg Med*. 1984;13:101–103.
202. White JD. Transthoracic pacing in cardiac asystole. *Am J Emerg Med*. 1983;1:264–266.
203. Smith I, Monk TG, White PF. Comparison of transesophageal atrial pacing with anticholinergic drugs for the treatment of intraoperative bradycardia. *Anesth Analg*. 1994;78:245–252.
204. Morrison LJ, Long J, Vermeulen M, Schwartz B, Sawadsky B, Frank J, Cameron B, Burgess R, Shield J, Bagley P, Mausz V, Brewer JE, Dorian P. A randomized controlled feasibility trial comparing safety and effectiveness of prehospital pacing versus conventional treatment: “PrePACE.” *Resuscitation*. 2008;76:341–349.

KEY WORDS: arrhythmia ■ automatic external defibrillator ■ cardioversion ■ ventricular fibrillation

**Part 6: Electrical Therapies: Automated External Defibrillators, Defibrillation,
Cardioversion, and Pacing** 2010 American Heart Association Guidelines for
Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Mark S. Link, Dianne L. Atkins, Rod S. Passman, Henry R. Halperin, Ricardo A. Samson,
Roger D. White, Michael T. Cudnik, Marc D. Berg, Peter J. Kudenchuk and Richard E. Kerber

Circulation. 2010;122:S706-S719

doi: 10.1161/CIRCULATIONAHA.110.970954

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 2010 American Heart Association, Inc. All rights reserved.

Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the
World Wide Web at:

http://circ.ahajournals.org/content/122/18_suppl_3/S706

An erratum has been published regarding this article. Please see the attached page for:

</content/123/6/e235.full.pdf>

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the [Permissions and Rights Question and Answer](#) document.

Reprints: Information about reprints can be found online at:
<http://www.lww.com/reprints>

Subscriptions: Information about subscribing to *Circulation* is online at:
<http://circ.ahajournals.org/subscriptions/>

Correction

In the article by Link et al, “Part 6: Electrical Therapies: Automated External Defibrillators, Defibrillation, Cardioversion, and Pacing: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care,” which published ahead of print on October 18, 2010, and appeared with the November 2, 2010, issue of the journal (*Circulation*. 2010;122[suppl 3]:S706–S719), a change was needed.

On page S708, in the right column, the first complete paragraph, the third sentence read, “Therefore, for biphasic defibrillators, providers should use the manufacturer’s recommended energy dose (120 to 200 J) (Class I, LOE B).” It has been updated to read, “Therefore, for biphasic defibrillators, providers should use the manufacturer’s recommended energy dose (eg, initial dose of 120 to 200 J) (Class I, LOE B).”

This correction has been made to the current online version of the article, which is available at http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S706.

DOI: 10.1161/CIR.0b013e31820ff4b0