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Part 5: Electrical Therapies

Automated External Defibrillators, Defibrillation, Cardioversion, and Pacing

This chapter presents guidelines for defibrillation with automated external defibrillators (AEDs) and manual defibrillators, 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 reasons: (1) the most frequent initial rhythm in witnessed SCA is ventricular fibrillation (VF), (2) the treatment for VF is electrical defibrillation, (3) the probability of successful defibrillation diminishes rapidly over time, and (4) VF tends to deteriorate to asystole within a few minutes.¹

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} CPR can double¹⁻³ 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 about 5 minutes after SCA.^{5,6} CPR prolongs VF⁷⁻⁹ (ie, the window of time during which defibrillation can occur) and provides a small amount of blood flow that may maintain some oxygen and substrate delivery to the heart and brain.¹⁰ Basic CPR alone, however, is unlikely to eliminate 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 arrest: (1) activation of the emergency medical services (EMS) system or emergency medical response system, (2) provision of CPR, and (3) operation of an AED. When 2 or more rescuers are present, activation of EMS and initiation of CPR can occur simultaneously.

Delays to either start of CPR or defibrillation can reduce survival from SCA. In the 1990s some predicted that CPR could be rendered obsolete by the widespread development of community AED programs. Cobb⁶ noted, however, that as more Seattle first responders were equipped with AEDs, survival rates from SCA unexpectedly fell. He attributed this decline to reduced emphasis on CPR, and there is growing evidence to support this view. Part 4: “Adult Basic Life Support” summarizes the evidence on the importance of effective chest compressions and minimizing interruptions in providing compressions.

Two critical questions about integration of CPR with defibrillation were evaluated during the 2005 Consensus Conference.¹¹ The first question concerns whether CPR should be provided before defibrillation is attempted. The second question concerns 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 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.

When an out-of-hospital cardiac arrest is not witnessed by EMS personnel, they may give about 5 cycles of CPR before checking the ECG rhythm and attempting defibrillation (Class IIb). 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 1½ to 3 minutes). This recommendation regarding CPR prior to attempted defibrillation is supported by 2 clinical studies (LOE 2⁵; LOE 3⁶) of adult out-of-hospital VF SCA. In those studies when EMS call-to-arrival intervals were 4⁶ to 5⁵ minutes or longer, victims who received 1½ to 3 minutes of CPR before defibrillation showed an increased rate of initial resuscitation, survival to hospital discharge,^{5,6} and 1-year survival⁵ when compared with those who received immediate defibrillation for VF SCA. One randomized study,¹² however, found no benefit to CPR before defibrillation for non-paramedic-witnessed SCA.

EMS system medical directors may consider implementing a protocol that would allow EMS responders to provide about 5 cycles (about 2 minutes) of CPR before defibrillation of patients found by EMS personnel to be in VF, particularly when the EMS system call-to-response interval is >4 to 5

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minutes. There is insufficient evidence to support or refute CPR before defibrillation for in-hospital cardiac arrest.

1-Shock Protocol Versus 3-Shock Sequence

At the time of the 2005 Consensus Conference, no published human or animal studies were found that compared a 1-shock protocol with a 3-stacked shock protocol for treatment of VF cardiac arrest. In animal studies, however, frequent or long interruptions in precordial chest compressions for rhythm analysis¹³ or rescue breathing^{14,15} were associated with post-resuscitation myocardial dysfunction and reduced survival rates. Secondary analyses of 2 randomized trials^{16,17} showed that interruption in chest compressions is associated with a decreased probability of conversion of VF to another rhythm. In 2 recent clinical observational studies (LOE 4) of out-of-hospital¹⁸ and in-hospital¹⁹ CPR by healthcare providers, chest compressions were performed only 51%¹⁸ to 76%¹⁹ of total CPR time.

In 2005 the rhythm analysis for a 3-shock sequence performed by commercially available AEDs resulted in delays of up to 37 seconds between delivery of the first shock and delivery of the first post-shock compression.¹³ This delay is difficult to justify in light of the first-shock efficacy of >90% reported by current biphasic defibrillators.^{20–25} 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, suggests that a 1-shock scenario plus immediate CPR is reasonable.

When VF/pulseless ventricular tachycardia (VT) is present, the rescuer should deliver 1 shock and should then immediately resume CPR, beginning with chest compressions (Class IIa). The rescuer should not delay resumption of chest compressions to recheck the rhythm or pulse. After 5 cycles (about 2 minutes) of CPR, the AED should then analyze the cardiac rhythm and deliver another shock if indicated (Class IIb). If a nonshockable rhythm is detected, the AED should instruct the rescuer to resume CPR immediately, beginning with chest compressions (Class IIb). Concern that chest compressions might provoke recurrent VF in the presence of a post-shock organized rhythm does not appear to be warranted.²⁵

AED voice prompts should not instruct the lay user to reassess the patient at any time. AED manufacturers should seek innovative methods to decrease the amount of time chest compressions are withheld 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.

First-shock efficacy for monophasic shocks is lower than first-shock efficacy for biphasic shocks.^{17,26,27} 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

AEDs 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.

One study compared the effectiveness of 175 J versus 320 J monophasic waveform shocks for out-of-hospital VF cardiac arrest.²⁸ Approximately 61% of patients who received shocks with either 175 J or 320 J monophasic damped sine waveform were defibrillated with the first shock, which was delivered an average of 10.6 minutes after the call to EMS. There was no significant difference in the percentage of patients who developed advanced atrioventricular (AV) block after 1 shock. AV block was more likely to develop after 2 or 3 shocks of 320 J than after 2 or 3 shocks of 175 J, but the block was transient and did not affect survival to hospital discharge.²⁸

Healthcare providers must practice efficient coordination between CPR and defibrillation. 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 perfusing rhythm will return after defibrillation (elimination of VF).²⁹ Analyses of VF waveform characteristics predictive of shock success have documented that the shorter the time between a chest compression and delivery of a shock, the more likely the shock will be successful.^{29,30} Reduction in the interval from compression to shock delivery by even a few seconds can increase the probability of shock success.¹⁶

The rescuer providing chest compressions should minimize interruptions in chest compressions for rhythm analysis and shock delivery and should be prepared to resume CPR, beginning with chest compressions, as soon as a shock is delivered. 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. The lone rescuer should practice coordination of CPR with efficient AED operation.

Defibrillation Waveforms and Energy Levels

Defibrillation involves delivery of current through the chest and to the heart to depolarize myocardial cells and eliminate VF. The energy settings for defibrillators are designed to provide the lowest effective energy needed to terminate VF. Because defibrillation is an electrophysiologic event that occurs in 300 to 500 milliseconds after shock delivery, the term *defibrillation* (shock success) is typically defined as termination of VF for at least 5 seconds following the shock.^{31,32} VF frequently recurs after successful shocks, but this recurrence should not be equated with shock failure.^{17,25}

Shock success using the typical definition of *defibrillation* should not be confused with resuscitation outcomes such as restoration of a perfusing rhythm, survival to hospital admission, or survival to hospital discharge.^{31,33} Although resuscitation outcomes including survival may be affected by many variables in addition to shock delivery, defibrillation pro-

grams 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. No specific waveform (either monophasic or biphasic) is consistently associated with a higher rate of return of spontaneous circulation (ROSC) or rates of survival to hospital discharge after cardiac arrest.

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 is abruptly returned to baseline (truncated) to zero current flow.

Few monophasic waveform defibrillators are being manufactured but many are still in use. Most of these use MDS waveforms. As noted above, no specific waveform (either monophasic or biphasic) is consistently associated with a greater incidence of ROSC or survival to hospital discharge rates after cardiac arrest than any other specific waveform. Research indicates, however, that when doses equivalent to or lower than monophasic doses are used, biphasic waveform shocks are safe and effective for termination of VF.

Biphasic Waveform Defibrillators

Researchers have collected data from both out-of-hospital^{34–36} and in-hospital studies (electrophysiologic studies and implantable cardioverter-defibrillator [ICD] testing and evaluation).³⁷ Overall this research indicates that lower-energy biphasic waveform shocks have equivalent or higher success for termination of VF than either damped sinusoidal or truncated exponential monophasic waveform shocks delivering escalating energy (200 J, 300 J, 360 J) with successive shocks. No direct comparison of the different biphasic waveforms has been made.

The optimal energy for first-shock biphasic waveform defibrillation yielding the highest termination rate for VF has not been determined. Several randomized (LOE 2)^{17,24,27} and observational studies (LOE 5)^{26,38} 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 (Class IIa).^{32,39–41}

Compensation for patient-to-patient differences in impedance may be achieved by changes in duration and voltage of shocks or by releasing the residual membrane charge (called *burping*). Whether there is an optimal ratio of first-phase to second-phase duration and leading-edge amplitude is unclear. It is unknown whether a waveform more effective for *immediate outcomes* (defibrillation) and *short-term outcomes* (ROSC, survival to hospital admission) results in better *long-term outcomes* (survival to hospital discharge, survival for 1 year). Given the high efficacy of all biphasic wave-

forms, other determinants of survival (eg, interval from collapse to CPR or defibrillation) are likely to supersede the impact of specific biphasic waveforms or energies.

Fixed and Escalating Energy

Commercially available biphasic AEDs provide either fixed or escalating energy levels.

Multiple prospective human clinical studies (LOE 2)^{27,42} and retrospective^{17,24,26,38,43,44} studies have failed to identify an optimal biphasic energy level for first or subsequent shocks. Therefore, it is not possible to make a definitive recommendation for the selected energy for the first or subsequent biphasic defibrillation attempts.

Biphasic defibrillators use one of two waveforms, and each waveform has been shown to be effective in terminating VF over a specific dose range. The ideal shock dose for a biphasic device is one that falls within the range that has been documented to be effective using that specific device. Current research confirms that it is reasonable to use selected energies of 150 J to 200 J with a biphasic truncated exponential waveform or 120 J with a rectilinear biphasic waveform for the initial shock. For second and subsequent biphasic shocks, use the same or higher energy (Class IIa). In this context “selected” refers to the energy dose selected by the operator (or programmed by the AED manufacturer). With the rectilinear biphasic waveform device, selected and delivered energies usually differ; delivered energy is typically higher in the usual range of impedance. For example, in a patient with 80 Ω impedance, a selected energy of 120 J will deliver 150 J.

None of the available evidence has shown superiority of either nonescalating or escalating energy biphasic waveform defibrillation for termination of VF. Nonescalating and escalating energy biphasic waveform shocks can be used safely and effectively to terminate short-duration and long-duration VF (Class IIa). The safety and efficacy data related to specific biphasic waveforms, the most effective initial shock, and whether to use escalating sequences require additional studies in both the in-hospital and out-of-hospital settings.

Automated External Defibrillators

AEDs are sophisticated, reliable computerized devices that use voice and visual prompts to guide lay rescuers and health-care providers to safely defibrillate VF SCA.^{34,36,45,46} In recent clinical trials,^{18,19} modified prototype AEDs recorded information about frequency and depth of chest compressions during CPR. If such devices become commercially available, AEDs may one day 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.^{47–49} 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 VF 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 organization, planning, training, linking with the EMS system, and establishing a process of continuous quality improvement.^{50,51}

Studies of lay rescuer AED programs in airports⁵² and casinos^{53,54} and first-responder programs with police officers^{26,34,36,44,55–57} have shown a survival rate 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. These high survival rates, however, are not attained in programs that fail to reduce time to defibrillation.^{58–60}

In a large prospective randomized trial (LOE 1)⁶¹ funded by the AHA, the National Heart, Lung, and Blood Institute (NHLBI), and several AED manufacturers, 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. The following elements are recommended for community lay rescuer AED programs^{50,51}:

- A planned and practiced response; typically this requires oversight by a healthcare provider
- Training of anticipated rescuers in CPR and use of the AED
- Link with the local EMS system
- Process of ongoing quality improvement

More information is available on the AHA website: www.americanheart.org/cpr. Under the topic “Links on this site,” select “Have a question?” and then select “AED.”

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 NHLBI 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 h/d).⁶¹

To be effective, AED programs should be integrated into an overall EMS strategy for treating patients in cardiac arrest. CPR and AED use by public safety first responders (traditional and nontraditional) are recommended to increase survival rates for SCA (Class I). AED programs in public locations where there is a relatively high likelihood of witnessed cardiac arrest (eg, airports, casinos, sports facilities) are recommended (Class I). Because the improvement in survival rates in AED programs is affected by the time to CPR and to defibrillation, sites that deploy AEDs should establish a response plan, train likely responders in CPR and AED use, maintain equipment, and coordinate with local EMS systems.^{50,51}

Approximately 80% of out-of-hospital cardiac arrests occur in private or residential settings (LOE 4).⁶² Reviewers found no studies that documented the effectiveness of home AED deployment, so there is no recommendation for or against personal or home deployment of AEDs (Class Indeterminate).

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,^{25,26,28,44} and 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 support ventilation and circulation with CPR as needed.

The mere presence of an AED does not ensure that it will be used when SCA occurs. Even in the NHLBI 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.⁶¹ 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). These quality improvement efforts should use both routine inspections and postevent data (from AED recordings and responder reports) to evaluate the following^{50,51}:

- 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 have microprocessors that 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. 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 hospital settings, and they hold promise for future AEDs that may prompt rescuers to improve the quality of CPR provided.^{18,19}

AEDs have been tested extensively, both in vitro against libraries of recorded cardiac rhythms and clinically in many field trials in adults^{63,64} and children.^{65,66} 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.

Electrode Placement

Rescuers should place AED electrode pads on the victim's bare chest in the conventional sternal-apical (anterolateral) position (Class IIa). The right (sternal) chest pad is placed on

the victim's right superior-anterior (infraclavicular) chest and the apical (left) pad is placed on the victim's inferior-lateral left chest, lateral to the left breast (Class IIa). Other acceptable pad positions are placement on the lateral chest wall on the right and left sides (biaxillary) or the left pad in the standard apical position and the other pad on the right or left upper back (Class IIa).

When an implantable medical device is located in an area where a pad would normally be placed, position the pad at least 1 inch (2.5 cm) away from the device (Class Indeterminate). If the victim has an 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.⁶⁷

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.⁶⁸ Remove medication patches and wipe the area before attaching the electrode pad.

If an unresponsive victim is lying in water or if the victim's chest is covered with water or the victim is extremely diaphoretic, remove the victim from water and briskly wipe the chest before attaching electrode pads and attempting defibrillation. AEDs can be used when the victim is lying on snow or ice. Most victims do not need any special preparation of the chest other than removal of the clothes from the chest. If the victim has a very hairy chest, it may be necessary to remove some hair so that the electrode pads will adhere to the chest. This may be accomplished by briskly removing an electrode pad (which will remove some hair), or it may be necessary to shave the chest in that area.

AED Use in Children

Cardiac arrest is less common in children than adults, and its causes are more diverse.^{69–71} Although VF is not a common arrhythmia in children, it is observed in 5% to 15% of pediatric and adolescent arrests.^{71–75} In these patients rapid defibrillation may improve outcomes.^{75,76}

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^{77,78} and pediatric animal models⁷⁹ with no significant adverse effects. Based on adult clinical data^{17,24} and pediatric animal models,^{79–81} biphasic shocks appear to be at least as effective as monophasic shocks and less harmful. Recommended manual defibrillation (monophasic or biphasic) doses are 2 J/kg for the first attempt (Class IIa; LOE 5⁸² and 6⁷⁹) and 4 J/kg for subsequent attempts (Class Indeterminate).

Many AEDs can accurately detect VF in children of all ages^{65,66} and differentiate shockable from nonshockable rhythms with a high degree of sensitivity and specificity.^{65,66} Some 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 the rescuer should use a pediatric dose-attenuator system if one is available.^{78,83,84} 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.

There is insufficient data to make a recommendation for or against the use of AEDs for infants <1 year of age (Class Indeterminate). During infancy the risk of VF SCA is unknown, and most cardiac arrest is thought to be related to progression of respiratory failure or shock. As a result there is concern that repeated interruption of CPR to try to detect and treat a rhythm uncommon in that age group may introduce more risk than benefit.⁸³

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 high specificity for pediatric shockable rhythms and with a pediatric attenuator system (eg, pediatric pad-cable system or other method of attenuating the shock dose). This statement, however, should not be interpreted as a recommendation for or against AED placement in specific locations where children are present. Ideally health-care systems that routinely provide care to children at risk for cardiac arrest should have available manual defibrillators capable of dose adjustment.⁸³

In-Hospital Use of AEDs

At the time of the 2005 Consensus Conference, there were no published in-hospital randomized trials of AEDs versus manual defibrillators. Evidence from 1 study of fair quality (LOE 4)⁸⁵ and a case series (LOE 5)⁸⁶ indicated higher rates of survival to hospital discharge when AEDs were used to treat adult VF or pulseless VT in the hospital.

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.⁸⁷ Despite limited evidence, AEDs should 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. An effective system for training and retraining should be in place.

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.⁸⁸ 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 (see Part 3: "Overview of CPR").

Manual Defibrillation

Shock Energies

At present it is clear that both low-energy and high-energy biphasic waveform shocks are effective, but definitive recommendations for the first and subsequent energy levels for all devices cannot be made because devices vary in waveform

and reported shock success. Although both escalating-energy and nonescalating-energy defibrillators are available, there is insufficient data to recommend one approach over another. Any claim of superiority at this time is unsupported.

As noted, biphasic defibrillators use one of two waveforms, and each waveform has been shown to be effective in terminating VF over a specific dose range. The ideal shock dose with a biphasic device is one that falls within the range that has been documented to be effective using that specific device. Manufacturers should display the device-specific effective waveform dose range on the face of the device, and providers should use that dose range when attempting defibrillation with that device. Providers should be aware of the range of energy levels at which the specific waveform they use has been shown to be effective for terminating VF, and they should use that device-specific dose for attempted defibrillation. At this time there is no evidence that one biphasic waveform is more effective than another.

With a biphasic defibrillator it is reasonable to use selected energies of 150 J to 200 J with a biphasic truncated exponential waveform or 120 J with a rectilinear biphasic waveform for the initial shock. For second and subsequent shocks, use the same or higher energy (Class IIa). In this context “selected” refers to the energy dose selected by the operator (or programmed by the AED manufacturer). With the rectilinear biphasic waveform device, selected and delivered energies usually differ; delivered energy is typically higher in the usual range of impedance. For example, in a patient with 80 Ω impedance, a selected energy of 120 J will deliver 150 J.

If a provider is operating a manual biphasic defibrillator and is unaware of the effective dose range for that device to terminate VF, the rescuer may use a selected dose of 200 J for the first shock and an equal or higher dose for the second and subsequent shocks. The 200-J “default” energy level is not necessarily an optimal dose, but it was selected because it falls within the reported range of doses effective for first and subsequent biphasic shocks. In addition, this dose can be provided by every biphasic manual defibrillator available in 2005. Thus, it is a consensus default dose and not a recommended ideal dose. If devices are clearly labeled and providers are familiar with the devices they will use for clinical care, the device-specific dose will be used and there will be no need for the “default” 200-J dose.

If a monophasic defibrillator is used, select a dose of 360 J for all shocks. If VF is initially terminated by a shock but then recurs later in the arrest, deliver subsequent shocks at the previously successful energy level.

Defibrillation is achieved by generating amplitude of current flow and sustaining that flow for a time interval. Although the defibrillator operator selects the shock energy (in joules), it is the current flow (in amperes) that actually depolarizes the myocardium. Current depends in part on the selected shock dose and is affected by the thoracic pathway between the 2 defibrillator electrodes and the position of the heart in that pathway and impedance to current flow between the electrodes. The complexity of thoracic current flow has been observed experimentally.⁸⁹

The most important determinant of survival in adult VF SCA is rapid defibrillation by either a monophasic or biphasic

device. Thus, in the hospital it is acceptable to deliver 1 shock with a monophasic or biphasic defibrillator followed by immediate initiation of CPR, beginning with compressions. The goal is to minimize the time between chest compressions and shock delivery and between shock delivery and resumption of chest compressions. In specific settings (eg, critical care units with hemodynamic monitoring in place), this sequence may be modified at the physician’s discretion (see Part 7.2: “Management of Cardiac Arrest” and Part 12: “Pediatric Advanced Life Support”).

Transthoracic Impedance

The average adult human impedance is ≈ 70 to 80Ω .^{90–92} When transthoracic impedance is too high, a low-energy shock will not generate sufficient current to achieve defibrillation.^{91,93,94} 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 suggests that one of these modalities is better than the others in decreasing impedance (Class Indeterminate).

In a male patient with a hairy chest, electrode-to-chest contact may be poor, and the hair may cause air trapping between the electrode and skin. This, as well as improper use of paddles, may result in high impedance, with occasional current arcing. Although extremely rare, in oxygen-rich environments such as critical care units, this arcing has been known to cause fires if an accelerant is present (see below). When using paddles, rescuers should apply them firmly to gel pads on the chest wall, avoiding contact with ECG leads. Use of self-adhesive pads will reduce the risk of arcing. It may be necessary to shave the area of intended pad placement.

Electrode Position

An overview of adhesive pad placement was provided in the AED section above. If electrode paddles are used instead of pads, the paddles should be well separated, and the paste or gel used to create the interface between the paddles and the skin should not be smeared on the chest between the paddles. Smearing of the paste or gel may allow current to follow a superficial pathway (arc) along the chest wall, “missing” the heart. Self-adhesive monitor/defibrillator electrode pads are as effective as gel pads or paste (LOE 3^{95–97}), and they can be placed before cardiac arrest to allow for monitoring and then rapid administration of a shock when necessary.⁹⁸ Consequently, self-adhesive pads should be used routinely instead of standard paddles (Class IIa; LOE 2, 4).

When providing cardioversion or defibrillation for patients with permanent pacemakers or ICDs, do not place the electrodes over or close to the device generator, because defibrillation can cause the pacemaker to malfunction. A pacemaker or ICD also may block some current to the myocardium during defibrillation attempts, resulting in suboptimal energy delivery to the heart. Because some of the defibrillation current flows down the pacemaker leads, permanent pacemakers and ICDs should be reevaluated after the patient receives a shock.⁹⁹

Electrode Size

In 1993 the Association for the Advancement of Medical Instrumentation recommended a minimum electrode size of 50 cm² for individual electrodes.¹⁰⁰ 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.^{90,95} Small electrodes (4.3 cm) may be harmful and may cause myocardial necrosis.¹⁰¹ 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 effective¹⁰² in VF of brief duration. Use of the smallest (pediatric) pads, however, can result in unacceptably high transthoracic impedance in larger children.¹⁰³ It is best to use the largest pads that can fit on the chest without overlap.

Fibrillation Waveform Analysis

Several retrospective case series, animal studies, and theoretical models (LOE 4^{29,30,104–110} and LOE 6^{111–121}) suggest that it is possible to predict, with varying reliability, the success of attempted defibrillation by analyzing the VF waveform. If prospective studies can select optimal defibrillation waveforms and optimal timing of shock delivery (eg, before or after a period of CPR), shock delivery may be more likely to result in return of spontaneous perfusion, and the delivery of unsuccessful high-energy shocks may be prevented. At present there is insufficient evidence to recommend for or against analysis of VF ECG characteristics (Class Indeterminate).

At issue is whether analysis of the VF waveform is useful in predicting therapeutic outcome and modifying therapy prospectively. Potential applications include prediction of success of cardioversion, selection of appropriate waveform type, and optimization of timing of defibrillation relative to CPR and medication delivery.

Current-Based Defibrillation

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^{92,122} but has not yet been used clinically as a better physiologic descriptor of defibrillation dose. 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. Another difficulty with using energy as a descriptor was described earlier with regard to differences between operator-selected energy and that delivered with the rectilinear biphasic waveform. Transition to current-based description 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.⁹² Comparable

information on current dosage for biphasic waveform shocks is under investigation.

“Occult” Versus “False” Asystole

There is no evidence that attempting to “defibrillate” asystole is beneficial. In 1989 Losek¹²³ 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.¹²⁴ There was no benefit from shock delivery for asystole. In fact, in all outcomes studied, including ROSC and survival, the group that received shocks showed a trend toward a *worse* outcome than the group that did not receive shocks. With recent recognition of the importance of minimizing interruptions in chest compressions, it is difficult to justify any interruption in chest compressions to attempt shock delivery for asystole.

Fire Hazard

Several case reports have described fires ignited by sparks from poorly applied defibrillator paddles in the presence of an oxygen-enriched atmosphere (LOE 5).^{125–130} Severe fires have been reported when ventilator tubing is disconnected from the tracheal tube and then left adjacent to the patient’s head, blowing oxygen across the chest during attempted defibrillation (LOE 5).^{126,128,130}

The use of self-adhesive defibrillation pads is probably the best way to 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). Do not use medical gels or pastes with poor electrical conductivity, such as ultrasound gel.

Rescuers should take precautions to minimize sparking during attempted defibrillation; try to ensure that defibrillation is not attempted in an oxygen-enriched atmosphere (Class IIa). When ventilation is interrupted for shock delivery, rescuers should try to ensure that oxygen does not flow across the patient’s chest during defibrillation attempts.

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.¹³¹ The energy (shock dose) used for a synchronized shock is lower than that used for unsynchronized shocks (defibrillation). These low-energy shocks should always be delivered as synchronized shocks because if they are delivered as *unsynchronized* shocks they are likely to induce VF. If cardioversion is needed and it is impossible to synchronize a shock (eg, the patient’s rhythm is irregular), use high-energy unsynchronized shocks.

Delivery of synchronized shocks (cardioversion) is indicated to treat unstable tachyarrhythmias associated with an

organized QRS complex and a perfusing rhythm (pulses). The unstable patient demonstrates signs of poor perfusion, including altered mental status, ongoing chest pain, hypotension, or other signs of shock (eg, pulmonary edema).

Synchronized cardioversion is recommended to treat unstable supraventricular tachycardia due to reentry, atrial fibrillation, and atrial flutter. These arrhythmias are all caused by reentry, an abnormal rhythm circuit that allows a wave of depolarization to travel in a circle. The delivery of a shock can stop these rhythms because it interrupts the circulating (reentry) pattern. Synchronized cardioversion is also recommended to treat unstable monomorphic VT. For additional information see Part 7.3: “Management of Symptomatic Bradycardia and Tachycardia.”

Cardioversion will not be effective for treatment of junctional tachycardia or ectopic or multifocal atrial tachycardia because these rhythms have an automatic focus. Automatic rhythms are created when local cells are stimulated to spontaneously depolarize at a rapid rate. Sinus tachycardia is a good example of an automatic rhythm. It results when the cells in the sinus node are stimulated (eg, by catecholamines) to depolarize at a rapid rate. Junctional tachycardia and ectopic or multifocal atrial tachycardia also result when cells are stimulated to depolarize at a rapid rate. Delivery of a shock cannot stop these rhythms. In fact, shock delivery to a heart with a rapid automatic focus may increase the rate of the tachyarrhythmia.

Synchronized cardioversion is not used for treatment of VF, pulseless VT, or unstable polymorphic (irregular) VT. These rhythms require delivery of high-energy *unsynchronized* shocks (ie, defibrillation doses). Electrical therapy for VT is discussed further below. For additional information see Part 7.2: “Management of Cardiac Arrest.”

Supraventricular Tachycardias (Reentry SVT)

The recommended initial monophasic energy dose for cardioversion of atrial fibrillation is 100 J to 200 J. Cardioversion of atrial flutter and other supraventricular tachycardias generally requires less energy; an initial energy of 50 J to 100 J MDS waveform is often sufficient. If the initial 50-J shock fails, providers should increase the dose in a stepwise fashion.⁹³ These recommendations are consistent with those contained in the *ECC Guidelines 2000*.⁵⁰ Cardioversion with biphasic waveforms is now available,¹³² but the optimal doses for cardioversion with biphasic waveforms have not been established with certainty. Extrapolation from published experience with elective cardioversion of atrial fibrillation using rectilinear and truncated exponential waveforms supports an initial dose of 100 J to 120 J with escalation as needed.^{133,134} This initial dose has been shown to be 80% to 85% effective in terminating atrial fibrillation. Until further evidence becomes available, this information can be used to extrapolate biphasic cardioversion doses to other tachyarrhythmias.^{135–138}

A recent prospective randomized study that compared the rectilinear biphasic waveform (200 J maximum selected energy) with a biphasic truncated exponential waveform (360 J maximum energy) for elective cardioversion found no significant differences in efficacy between the 2 waveforms.¹³⁴

Ventricular Tachycardia

The amount of energy and timing of shocks for treatment of VT with pulses are determined by the patient’s condition and the morphologic characteristics of the VT.¹³⁹ Pulseless VT is treated as VF (see Part 7.2: “Management of Cardiac Arrest”). Management of stable VT is summarized in Part 7.3: “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).

Monomorphic VT (regular form and rate) with a pulse responds well to monophasic waveform cardioversion (synchronized) shocks at initial energies of 100 J. If there is no response to the first shock, increase the dose in a stepwise fashion (eg, 100 J, 200 J, 300 J, 360 J). These recommendations are consistent with the recommendations in the *ECC Guidelines 2000*.⁵⁰

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. In addition, the patient with persistent polymorphic VT will probably not maintain perfusion/pulses for very long, so any attempt to distinguish between polymorphic VT with or without pulses quickly becomes moot. A good rule of thumb is that if your eye cannot synchronize to each QRS complex, neither can the defibrillator/cardioverter. 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 (see “Manual Defibrillation, Shock Energies”). After shock delivery the healthcare provider should be prepared to provide immediate CPR (beginning with chest compressions) and follow the ACLS Pulseless Arrest Algorithm if pulseless arrest develops (for further information see Part 7.2: “Management of Cardiac Arrest”).

There is limited data about the treatment of polymorphic (irregular) VT. Providers should consider consultation with an expert in arrhythmia management. Treatment of the patient with polymorphic VT is presented in section 7.3: “Management of Symptomatic Bradycardia and Tachycardia.”

Pacing

Pacing is not recommended for patients in asystolic cardiac arrest. Pacing can be considered in patients with symptomatic bradycardia.

Three randomized controlled trials (LOE 2)^{140–142} of fair quality and additional studies (LOE 3 to 7)^{143–149} 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 prehos-

pital or hospital (emergency department) setting. Given the recent recognition of the importance of maximizing chest compressions as well as the lack of demonstrated benefit of pacing for asystole, withholding chest compressions to attempt pacing for patients with asystole is not recommended (Class III).

Transcutaneous pacing is recommended for treatment of symptomatic bradycardia when a pulse is present. Healthcare providers should be prepared to initiate pacing in patients who do not respond to atropine (or second-line drugs if these do not delay definitive management). Immediate pacing is indicated if the patient is severely symptomatic, especially when the block is at or below the His Purkinje level. If the patient does not respond to transcutaneous pacing, transvenous pacing is needed. For further information see Part 7.3: "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. Checklists are useful when designed to identify and prevent such deficiencies.

Summary

The new recommendations for electrical therapies described in this section are designed to improve survival from SCA and life-threatening arrhythmias. For any victim of cardiac arrest, good CPR—push hard, push fast, allow complete chest recoil, and minimize interruptions in chest compressions—is essential. Some victims of VF SCA may benefit from a short period of CPR before attempted defibrillation. Whenever defibrillation is attempted, rescuers must coordinate good 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 formerly recommended to treat VF. Further data is needed to refine recommendations for use of electrical therapies, particularly for the use of biphasic waveforms.

References

1. 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.
2. 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.
3. Swor RA, Jackson RE, Cynar M, Sadler E, Basse E, Boji B, Rivera-Rivera EJ, Maher A, Grubb W, Jacobson R, et al. Bystander CPR, ventricular fibrillation, and survival in witnessed, unmonitored out-of-hospital cardiac arrest. *Ann Emerg Med.* 1995;25:780–784.
4. 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.
5. 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.
6. 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.
7. 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.
8. Holmberg S, Holmberg M, Herlitz J. Effect of bystander cardiopulmonary resuscitation in out-of-hospital cardiac arrest patients in Sweden. *Resuscitation.* 2000; 47:59–70.
9. Waalewijn 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.
10. Weaver WD, Copass MK, Bui D, Ray R, Hallstrom AP, Cobb LA. Improved neurologic recovery and survival after early defibrillation. *Circulation.* 1984;69:943–948.
11. International Liaison Committee on Resuscitation. 2005 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care With Treatment Recommendations. *Circulation.* 2005; 112:III-1–III-136.
12. 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.
13. 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.
14. 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.
15. Kern K, Hilwig R, Berb R, Sanders A, Ewy G. Importance of continuous chest compressions during CPR. *Circulation.* 2002;105:645–649.
16. 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.
17. 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.
18. 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.
19. 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.
20. 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.
21. 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.
22. 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.
23. 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.
24. Schneider T, Martens PR, Paschen H, Kuism 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. *Circulation.* 2000;102:1780–1787.
25. 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.

26. 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.
27. Morrison LJ, Dorian P, Long J, Vermeulen M, Schwartz B, Sawadsky B, et al. 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.
28. 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.
29. 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.
30. 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.
31. White RD. External defibrillation: the need for uniformity in analyzing and reporting results [editorial]. *Ann Emerg Med*. 1998;32:234–236.
32. Gliner BE, White RD. Electrocardiographic evaluation of defibrillation shocks delivered to out-of-hospital sudden cardiac arrest patients. *Resuscitation*. 1999;41:133–144.
33. Cummins RO, Chamberlain DA, Abramson NS, Allen M, Baskett P, Becker L, Bossaert L, Delooy L, Dick W, Eisenberg M, et al. 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.
34. 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.
35. 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.
36. 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.
37. Faddy SC, Powell J, Craig J. Biphasic and monophasic shocks for transthoracic defibrillation: a metaanalysis of randomized controlled trials. *Resuscitation*. 2000;58:9–16.
38. 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.
39. 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.
40. 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.
41. 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.
42. 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.
43. 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.
44. 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.
45. Cummins RO, Eisenberg M, Bergner L, Murray JA. Sensitivity, accuracy, and safety of an automatic external defibrillator. *Lancet*. 1984;2:318–320.
46. Davis EA, Mosesso 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.
47. 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.
48. 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.
49. 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.
50. American Heart Association in collaboration with International Liaison Committee on Resuscitation. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: International Consensus on Science. *Circulation*. 2000;102(suppl):I1–I384.
51. 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 Care Committee; the Council on Cardiopulmonary, Perioperative and Critical Care; and the Council on Clinical Cardiology. *Circulation*. 2005;111:3336–3340.
52. Caffrey SL, Willoughby PJ, Pepe PE, Becker LB. Public use of automated external defibrillators. *N Engl J Med*. 2002;347:1242–1247.
53. Valenzuela TD, Bjerke HS, Clark LL, et al. Rapid defibrillation by nontraditional responders: the Casino Project. *Acad Emerg Med*. 1998;5:414–415.
54. 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.
55. 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.
56. 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.
57. 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;279–283.
58. 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.
59. 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.
60. Sayre M, Evans J, White L, Brennan T. Providing automated external defibrillators to urban police officers in addition to fire department rapid defibrillation program is not effective. *Resuscitation*. 2005;66:189–196.
61. 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.
62. Becker L, Eisenberg M, Fahrenbruch C, Cobb L. Public locations of cardiac arrest: implications for public access defibrillation. *Circulation*. 1998;97:2106–2109.
63. 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.
64. 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.
 65. Atkinson E, Mikysa B, Conway JA, Parker M, Christian K, Deshpande J, Knilians 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.
 66. 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.
 67. 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.
 68. Panacek EA, Munger MA, Rutherford WF, Gardner SF. Report of nitro patch explosions complicating defibrillation. *Am J Emerg Med*. 1992;10:128-129.
 69. Kuisma M, Suominen P, Korpela R. Paediatric out-of-hospital cardiac arrests: epidemiology and outcome. *Resuscitation*. 1995;30:141-150.
 70. 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 [published correction appears in *Ann Emerg Med*. 1999;33:358]. *Ann Emerg Med*. 1999;33:174-184.
 71. Hickey RW, Cohen DM, Strausbaugh S, Dietrich AM. Pediatric patients requiring CPR in the prehospital setting. *Ann Emerg Med*. 1995;25:495-501.
 72. 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.
 73. 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.
 74. 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.
 75. 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.
 76. Safranek DJ, Eisenberg MS, Larsen MP. The epidemiology of cardiac arrest in young adults. *Ann Emerg Med*. 1992;21:1102-1106.
 77. 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.
 78. Atkins D, Jorgenson D. Attenuated pediatric electrode pads for automated external defibrillator use in children. *Resuscitation*. 2005;66:31-37.
 79. 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.
 80. 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.
 81. 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.
 82. Gutgesell HP, Tacker WA, Geddes LA, Davis S, Lie JT, McNamara DG. Energy dose for ventricular defibrillation of children. *Pediatrics*. 1976;58:898-901.
 83. 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.
 84. 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:S145-S147.
 85. 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.
 86. Destro A, Marzalani M, Sermasi S, Rossi F. Automatic external defibrillators in the hospital as well? *Resuscitation*. 1996;31:39-43.
 87. Kaye W, Mancini M, 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.
 88. Peberdy MA, Kaye W, Ornato JP, Larkin GL, Nadkarni V, Mancini ME, Berg RA, Nichol G, Lane-Trullt 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.
 89. Yoon RS, DeMonte TP, Hasanov KF, Jorgenson DB, Joy ML. Measurement of thoracic current flow in pigs for the study of defibrillation and cardioversion. *IEEE Trans Biomed Eng*. 2003;50:1167-1173.
 90. 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.
 91. 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.
 92. Lerman BB, DiMarco JP, Haines DE. Current-based versus energy-based ventricular defibrillation: a prospective study. *J Am Coll Cardiol*. 1988;12:1259-1264.
 93. 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.
 94. 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.
 95. Stults KR, Brown DD, Cooley F, Kerber RE. Self-adhesive monitor/defibrillation pads improve prehospital defibrillation success. *Ann Emerg Med*. 1987;16:872-877.
 96. Kerber RE, Martins JB, Kelly KJ, Ferguson DW, Kouba C, Jensen SR, Newman B, Parke JD, Kieso R, Melton J. Self-adhesive preapplied electrode pads for defibrillation and cardioversion. *J Am Coll Cardiol*. 1984;3:815-820.
 97. 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.
 98. Perkins GD, Roberts C, Gao F. Delays in defibrillation: influence of different monitoring techniques. *Br J Anaesth*. 2002;89:405-408.
 99. Levine PA, Barold SS, Fletcher RD, Talbot P. Adverse acute and chronic effects of electrical defibrillation and cardioversion on implanted unipolar cardiac pacing systems. *J Am Coll Cardiol*. 1983;1:1413-1422.
 100. *American National Standard: Automatic External Defibrillators and Remote Controlled Defibrillators (DF39)*. Arlington, Va: Association for the Advancement of Medical Instrumentation; 1993.
 101. 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.
 102. 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.
 103. Samson RA, Atkins DL, Kerber RE. Optimal size of self-adhesive preapplied electrode pads in pediatric defibrillation. *Am J Cardiol*. 1995;75:544-545.
 104. 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.
 105. 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.

106. 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.
107. 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.
108. Strohmer 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.
109. Strohmer 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.
110. 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.
111. 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.
112. 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.
113. 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.
114. Strohmer HU, Lindner KH, Keller A, Lindner IM, Pfenninger EG. Spectral analysis of ventricular fibrillation and closed-chest cardiopulmonary resuscitation. *Resuscitation.* 1996;33:155–161.
115. 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.
116. 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.
117. Marn-Pernat A, Weil MH, Tang W, Pernat A, Bisera J. Optimizing timing of ventricular defibrillation. *Crit Care Med.* 2001;29:2360–2365.
118. Hamprecht FA, Achleitner U, Krismer AC, Lindner KH, Wenzel V, Strohmer 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.
119. Amann A, Achleitner U, Antretter H, Bonatti JO, Krismer AC, Lindner KH, Rieder J, Wenzel V, Voelckel WG, Strohmer HU. Analysing ventricular fibrillation ECG-signals and predicting defibrillation success during cardiopulmonary resuscitation employing N(alpha)-histograms. *Resuscitation.* 2001;50:77–85.
120. Brown CG, Griffith RF, Van Ligtan 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.
121. 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.
122. Kerber RE, McPherson D, Charbonnier F, Kieso R, Hite P. Automated impedance-based energy adjustment for defibrillation: experimental studies. *Circulation.* 1985;71:136–140.
123. Losek JD, Hennes H, Glaeser PW, Smith DS, Hendley G. Prehospital countershock treatment of pediatric asystole. *Am J Emerg Med.* 1989;7:571–575.
124. 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.
125. Miller PH. Potential fire hazard in defibrillation. *JAMA.* 1972;221:192.
126. 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.
127. Fires from defibrillation during oxygen administration. *Health Devices.* 1994;23:307–309.
128. Lefever J, Smith A. Risk of fire when using defibrillation in an oxygen enriched atmosphere. *Med Devices Agency Safety Notices.* 1995;3:1–3.
129. Ward ME. Risk of fires when using defibrillators in an oxygen enriched atmosphere. *Resuscitation.* 1996;31:173.
130. Theodorou AA, Gutierrez JA, Berg RA. Fire attributable to a defibrillation attempt in a neonate. *Pediatrics.* 2003;112:677–679.
131. Lown B. Electrical reversion of cardiac arrhythmias. *Br Heart J.* 1967;29:469–489.
132. Page RL, Kerber R, Russell JK, et al. Biphasic vs. monophasic shock waveform for conversion of atrial fibrillation: the results of an international randomized, double-blind multicenter trial. *Circulation.* 2000;102:II-574.
133. 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.
134. Alatawi F, Gurevitz O, White R. Prospective, randomized comparison of two biphasic waveforms for the efficacy and safety of transthoracic biphasic cardioversion of atrial fibrillation. *Heart Rhythm.* 2005;2:382–387.
135. Adegay AA, Walsh SJ. Theory and practice of defibrillation: (1) atrial fibrillation and DC conversion. *Heart.* 2004;90:1493–1498.
136. Koster RW, Dorian P, Chapman FW, Schmitt PW, O'Grady SG, Walker RG. A randomized trial comparing monophasic and biphasic waveform shocks for external cardioversion of atrial fibrillation. *Am Heart J.* 2004;147:e20.
137. Neal S, Ngarmukos T, Lessard D, Rosenthal L. Comparison of the efficacy and safety of two biphasic defibrillator waveforms for the conversion of atrial fibrillation to sinus rhythm. *Am J Cardiol.* 2003;92:810–814.
138. Kim ML, Kim SG, Park DS, Gross JN, Ferrick KJ, Palma EC, Fisher JD. Comparison of rectilinear biphasic waveform energy versus truncated exponential biphasic waveform energy for transthoracic cardioversion of atrial fibrillation. *Am J Cardiol.* 2004;94:1438–1440.
139. Kerber RE, Kienzle MG, Olshansky B, Waldo AL, Wilber D, Carlson MD, Aschoff AM, Birger S, Fugatt L, Walsh S, et al. Ventricular tachycardia rate and morphology determine energy and current requirements for transthoracic cardioversion. *Circulation.* 1992;85:158–163.
140. Hedges JR, Syverud SA, Dalsey WC, Feero S, Easter R, Shultz B. Prehospital trial of emergency transcutaneous cardiac pacing. *Circulation.* 1987;76:1337–1343.
141. 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.
142. 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.
143. Ornato JP, Peberdy MA. The mystery of bradyasystole during cardiac arrest. *Ann Emerg Med.* 1996;27:576–587.
144. 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.
145. 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.
146. Dalsey WC, Syverud SA, Hedges JR. Emergency department use of transcutaneous pacing for cardiac arrests. *Crit Care Med.* 1985;13:399–401.
147. Knowlton AA, Falk RH. External cardiac pacing during in-hospital cardiac arrest. *Am J Cardiol.* 1986;57:1295–1298.
148. Ornato JP, Carveth WL, Windle JR. Pacemaker insertion for prehospital bradyasystolic cardiac arrest. *Ann Emerg Med.* 1984;13:101–103.
149. White JD. Transthoracic pacing in cardiac asystole. *Am J Emerg Med.* 1983;1:264–266.