Cardiopulmonary Resuscitation: Current Status

Joseph Varon, MD, FACP, FCCP, FCCM
George L. Sternbach, MD, FACEP
Paul E. Marik, MD, FCCP, FCCM

In its earliest forms, cardiopulmonary resuscitation (CPR) is most likely as old as human society itself.[1] Depictions of mouth-to-mouth ventilation appear in ancient Egyptian hieroglyphics, and descriptions appear in the Bible. Modern CPR techniques emerged in the late 1950s and early 1960s, particularly through the refinements of Kouwenhoven, Jude, and Knickerbocker, who demonstrated the efficacy of closed-chest compression in cases of cardiac arrest.[2] Since that time, there has been widespread acceptance of CPR as the cornerstone of basic cardiac life support, and external chest compression has become part of the standard method of managing cardiac arrest. Besides providing information on the etiology and epidemiology of cardiac arrest, this article will discuss in detail the steps involved in CPR, focusing on standard techniques but also addressing some variations in resuscitation methods. In addition, possible innovations in drug therapy and in prediction of outcome after cardiac arrest will be examined.

ETIOLOGY AND EPIDEMIOLOGY OF CARDIAC ARREST

Cardiac arrest is usually the result of a cardiac dysrhythmia. Approximately 80% to 90% of adults with sudden nontraumatic cardiac arrest are found to be in ventricular fibrillation (VF) when an initial electrocardiogram is obtained.[1–4] When VF-associated cardiac arrest occurs outside a hospital setting, it presumably reflects chronic myocardial ischemia with electrical instability, rather than acute myocardial infarction.[4] In contrast, cardiac arrest occurring in a hospital most often follows acute myocardial infarction or results from severe multisystem disease. Asystole, bradydysrhythmias, and pulseless electrical activity are the usual electrical rhythms encountered in these patients.[5]

The success rate following in-hospital cardiac arrest has remained unchanged over the past 3 decades; return of spontaneous circulation occurs in approximately 30% of patients, and approximately 15% are discharged neurologically intact.[6–8] CPR performed in a hospital is most likely to be successful in patients with primary VF and ischemic heart disease, patients with cardiac arrest in the absence of other life-threatening conditions, and patients with hypothermia, drug overdose, airway obstruction, or primary respiratory arrest.[8]

Patients who have an out-of-hospital cardiac arrest generally have a worse outcome. Initial survival of these patients, as determined by hospital admission rates, ranges between 8% and 22%, with only 1% to 8% of patients with out-of-hospital cardiac arrests being discharged neurologically intact.[9–11] The higher discharge percentage generally involves patients in whom CPR is initiated within 4 minutes of the arrest.[12]

INITIAL MANAGEMENT OF CARDIAC ARREST

Assessment

The first and most crucial step in the management of a patient with cardiac arrest is assessment. No one should undergo the intrusive and potentially injurious procedures of CPR unless the need for resuscitation has been established by an appropriate assessment. Consequently, determining whether the patient is unconscious, whether the patient is breathing, and whether the patient has a carotid pulse are all essential steps.

Defibrillation

Once a thorough assessment has been performed, the initial approach may vary, depending on the availability of equipment to monitor cardiac rhythm and apply electrical countershock. In a hospital, such

Dr. Varon is an Associate Professor of Medicine, Pulmonary & Critical Care Section, Baylor College of Medicine, and the former Research Director, Department of Emergency Services, The Methodist Hospital, Houston, TX; he is also a member of the Hospital Physician *Editorial Board. Dr. Sternbach is a Clinical Professor of Surgery, Department of Emergency Medicine, Stanford University Medical Center, Stanford, CA. Dr. Marik is the Director, Medical Intensive Care Unit, Mercy Hospital, Pittsburgh, PA.*

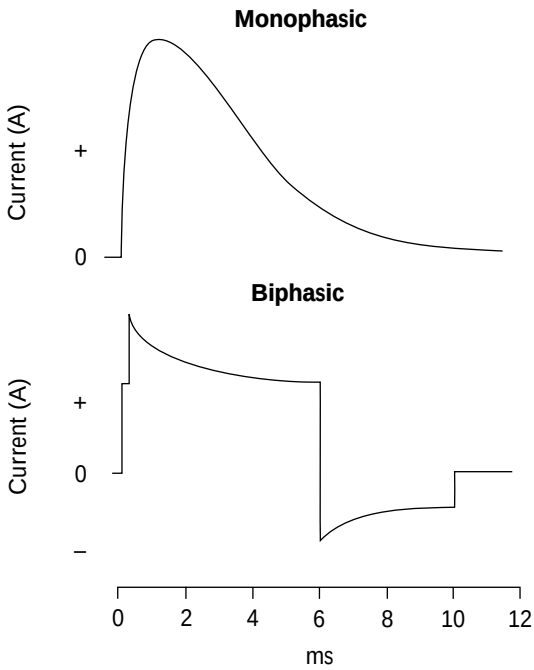

Figure 1. Waveforms generated by defibrillators. ms = milliseconds.

equipment should be available immediately. Should VF or ventricular tachycardia (VT) be documented in a hospital setting by conventional monitoring or with "quick look" defibrillation paddles, 3 sequential countershocks should be delivered as soon as possible at energy settings of 200, 300, and 360 J, respectively.[3]

Electrical defibrillation is the only effective method of terminating VF.[4] The survival following VF drops precipitously with time, with a survival rate of more than 70% when defibrillation is applied within 1 minute of cardiac arrest but dropping by 10% for each minute that defibrillation is delayed. Precordial chest compressions should initially be regarded as a secondary priority. If VF or VT is not the initial dysrhythmia or if defibrillation is not successful, chest compressions and artificial ventilation should be immediately initiated. A recent study suggests that in out-of-hospital cases in which there is a long delay before the arrival of medical personnel, chest compressions and ventilation can be administered before defibrillation is attempted.[13]

The aim of electrical therapy is to provide a current through the myocardium that will change enough myocardial cells to the same electrical state for a sufficient time to break the re-entrant circuits or to produce electrical homogeneity, thus allowing a stable rhythm to be re-established from the sinus node. The current delivered to the heart is determined by both the energy level chosen (J) and the transthoracic impedance present. For example, the use of bare paddles without a couplant (eg, gel, cream) results in very high transthoracic impedance; impedance can be reduced by applying strong pressure on the paddles, by anteroposterior positioning of the paddles, by delivery of repetitive shocks, and by delivery of the shock during expiration.

The energy level chosen for the initial shock is a compromise between risking myocardial damage from excessive current and risking unsuccessful defibrillation from too low a current. The American Heart Association and the European Resuscitation Council recommend an energy level of 200 J for initial attempts at defibrillation in adults.[3] Because thoracic impedance declines with successive shocks, the second attempt can be from 200 to 300 J. If a second attempt fails, then another shock at the maximum amount (360 J) should be administered. The defibrillator paddles should be placed in a position that will maximize current flow through the myocardium. The most commonly employed position is apex-anterior: the anterior electrode is placed on the right of the upper sternum below the clavicle, while the apex electrode is placed to the left of the nipple with the center of the electrode in the midaxillary line.

Most currently available defibrillators use a damped sine-wave monophasic waveform, which is not impedance compensated. At a given energy setting, the current delivered is dependent on transthoracic impedance; consequently, excessive current may be delivered to patients with low transthoracic impedance (causing increased myocardial damage), and insufficient current may be delivered to patients with high impedance (resulting in decreased efficacy).

Recently, biphasic waveform defibrillators have been developed (**Figure 1**). The rectilinear biphasic waveform is more advantageous in patients with high impedance because it is relatively insensitive to changes in transthoracic impedance. Multiple studies have shown that biphasic waveforms have greater efficacy than do monophasic waveforms for endocardial defibrillation, and more recent studies of transthoracic ventricular defibrillation have shown that biphasic shocks (of 150 J) are more effective and result in less post-shock myocardial dysfunction compared to monophasic shocks.[14–16]

A promising alternative to traditional energy-based defibrillation is current-based defibrillation. With this modality, the operator selects the electrical dose (in amperes) rather than the energy (in joules). The

defibrillator instantaneously measures the transthoracic impedance; consequently, this approach avoids delivery of low energy in cases of high transthoracic impedance or of an excessive current in the face of low impedance. A microprocessor in the defibrillator then determines and delivers the exact current that the operator has specified. It is likely that current-based defibrillation and low-energy biphasic waveform defibrillation will replace standard monophasic waveform defibrillation in the near future.

If cardiac arrest occurs outside a hospital setting, chest compressions and artificial ventilation should be initiated and continued until defibrillation or cardiac monitoring equipment becomes available. A precordial thump can sometimes terminate VT and, more rarely, VF and should be considered in cases of witnessed cardiac arrest when a defibrillator is not immediately available.[17,18]

The advent of paramedical services in the 1960s has led to increased survival from cardiac arrest, primarily from successful resuscitation from VF.[19] Emergency medical technicians have subsequently been trained to recognize VF and to operate manual defibrillators; automated external defibrillators (AEDs) were first introduced in 1979.[20] Developed to allow personnel with minimal training to provide early defibrillation, AEDs are able to analyze the electrical activity of the heart and to detect VT or VF, based on the rate, frequency, amplitude, slope, and isoelectric characteristics of the QRS complex.[21] Fine or low-voltage VF is the most difficult dysrhythmia to recognize on an AED.[22] In clinical practice, however, this distinction may not be significant, because low-voltage VF is most frequently seen after a long delay has occurred between arrest and initiation of treatment, and such cases have a low rate of successful resuscitation.[23]

Widespread availability of AEDs allows for early defibrillation by both ambulance crews and lay responders. Lay rescuers with minimal training have demonstrated an impressive ability to use AEDs during simulated cardiac arrest.[24] The impact of these devices on survival following actual out-of-hospital cardiac arrest, however, remains unclear.

Chest Compressions and Ventilation

The overall objective of closed-chest cardiac massage is to generate adequate flow of oxygenated blood to the heart and brain until more definitive therapy can be applied and effective spontaneous circulation restored. The basic life-support guidelines established by the American Heart Association include a combination of external chest compressions and ventilation.[3] Compressions are performed over the lower part of the sternum, at a depth of 1.5 in and at a rate of 80 to 100 compressions per minute (in adults). Arterial pressure is maximal when the duration of each compression is 50% of the compression-release cycle.

Because of the frequent absence of sufficient muscle tone during cardiac arrest, the tongue and epiglottis can obstruct the pharynx. If there is no evidence of head or neck trauma, the rescuer should use the head-tilt, chin-lift, or jaw-thrust maneuver to open the airway. Any foreign material or vomitus in the mouth should be removed. Rescue breathing can be provided by the mouth-to-mouth or mouth-to-nose technique. Barrier devices are available for mouth-to-mouth ventilation to prevent possible spread of pathogens to the rescuer. In one-rescuer CPR, ventilations are performed in a cycle of 15 compressions and two breaths, with 1.5 to 2 seconds devoted to the two rescue breaths. In two-rescuer CPR, ventilations are provided at the end of each fifth compression.[3]

During cardiac arrest, properly performed chest compressions can produce systolic arterial blood pressure peaks of 60 to 80 mm Hg, but diastolic pressure remains low.[25] This fact is significant because coronary perfusion occurs during diastole. Cardiac output resulting from chest compressions is only one fourth to one third of normal, and mean blood pressure in the carotid artery seldom exceeds 40 mm Hg.

The mechanism that produces forward blood flow during chest compressions has been widely debated, and 2 distinct theories have been proposed.[26] Initial studies suggested that forward flow results from compression of the heart between the sternum and paraspinal structures, with ejection of blood from the ventricles into the systemic and pulmonary circulation[2]; this "cardiac pump" theory held that when compression is released, intrathoracic and intracardiac pressures fall, promoting venous return and ventricular filling. This theory assumes that cardiac valve function is preserved during cardiac arrest, with mitral valve closure preventing backflow into the atria. Later studies have suggested, however, that forward flow during CPR results from an increase in intrathoracic pressure, producing an arteriovenous pressure gradient in which the left ventricle acts as a passive conduit and not as a pump (the "thoracic pump" theory). It is likely that both mechanisms result in forward blood flow.

Supplemental oxygen should be administered as soon as possible to patients with cardiac arrest. Rescue breathing will deliver only approximately 16% to 17% oxygen to the patient, ideally producing an alveolar oxygen tension of 80 mm Hg.[3] Bag-valve devices,

consisting of a self-inflating bag and nonrebreathing valve, can be used with a mask or endotracheal tube.

In patients who have had a cardiac arrest, low minute ventilation is required to maintain an optimal ventilation-perfusion relationship. Arterial carbon dioxide levels are often reduced to levels less than 20 mm Hg during CPR, because ventilation exceeds the minute volume required for normal gas exchange in the setting of low pulmonary flow.[27] Ventilation is performed with a recommended tidal volume of approximately 1000 mL, delivered slowly.

As soon as is practical during the resuscitative effort, the trachea should be intubated by trained personnel. Endotracheal intubation isolates the airway, keeps it patent, reduces the risk of aspiration, permits suctioning of the trachea, ensures delivery of a high concentration of oxygen, and provides a route for the administration of certain drugs.[28] In patients with suspected or confirmed cervical spine injuries, extreme caution should be used when intubating, with every effort taken to avoid hyperextension of the neck.

VARIATIONS IN RESUSCITATION METHODS

Ventilation

Recent evidence suggests that ventilation may not be necessary in the very initial stage of cardiac arrest. In canine studies, intubated dogs maintained an arterial oxygen saturation greater than 90% for the first 5 minutes of VF when chest compression were performed without ventilation.[29] Similarly, the Belgian Cerebral Resuscitation Study Group found no statistically significant difference in outcome in patients revived by bystanders, regardless of whether mouth-to-mouth ventilation was performed in addition to chest compressions.[30,31] This observation has been confirmed in a randomized controlled trial in which the outcome after CPR with chest compression alone was similar to that after chest compression with mouth-to-mouth ventilation.[32] These data suggest that chest compressions alone may suffice until qualified professionals and appropriate equipment become available.

Abdominal Compressions

Initially described in 1906, the concept of abdominal compressions during CPR was not discussed in the literature again until 1972; their potential usefulness constitutes a promising line of research.[33] Abdominal compressions are performed by a second or third rescuer. Two different techniques have been studied: synchronized (ie, simultaneous) chest and abdominal compressions and interposed (ie, alternating) abdominal compressions. Barranco and colleagues reported that pressure applied simultaneously to the chest and abdomen generated significantly higher thoracic aortic pressures than did interposed abdominal compressions.[34] In another study, Sack and colleagues[35] randomized patients to receive CPR with either interposed abdominal compressions or standard methods. Return of spontaneous circulation and subsequent hospital discharge were significantly greater for the group receiving interposed abdominal compressions than for those receiving standard CPR.[35] In contrast, another study found that interposed abdominal compressions failed to improve survival in patients with out-of-hospital cardiac arrest.[36]

Mechanical Aids

A number of mechanical devices have been developed to aid in resuscitation efforts, including the cardiac press, pneumatically powered mechanical resuscitators, and pneumatic compression vests. Interpretation of studies evaluating these devices is difficult, because of the small number of patients included in these trials. The pneumatic compression vest, analogous to a large blood-pressure cuff, is placed circumferentially around the thorax and programmed to inflate and deflate sequentially. Although the vest device may hold promise,[37] its effect on survival in patients receiving CPR is currently unknown.

Finally, active compression-decompression CPR is performed with a hand-held suction device applied to the chest wall. Suction applied by withdrawal of the device during the diastolic phase of CPR creates a negative pressure that, presumably, promotes forward blood flow. Investigation has, however, failed to demonstrate an improvement in outcome with this technique when compared to standard CPR.[38]

End-Tidal Carbon Dioxide Measurement

Attention has recently turned to the measurement of end-tidal carbon dioxide ($ETCO_2$) as a predictor of outcome after cardiac arrest. The introduction of battery-powered mainstream capnographs with infrared analysis of $ETCO_2$ has made possible quantitative capnography in patients following a cardiac arrest. $ETCO_2$ has been shown to be of prognostic value in cases of in-hospital and out-of-hospital cardiac arrest.[39] However, definite recommendations regarding use of $ETCO_2$ measurement still await larger clinical studies.

DRUG THERAPY FOR PATIENTS WITH CARDIAC ARREST

Venous Access

Once closed-chest compressions, defibrillation (when indicated), and airway management have been

performed, resuscitation personnel can begin intravenous administration of medications. The antecubital vein is the first choice for venous cannulation, because CPR does not have to be interrupted. However, there are also advantages to having central venous access, including more rapid delivery of drugs to their site of action, the ability to give multiple medications and fluids simultaneously, rapid volume resuscitation, and decreased likelihood of extravasation. A study reported that prompt drug delivery was enhanced with subclavian and internal jugular injection of medication during CPR in a canine arrest model.[40] A short femoral venous line should not be used for drug administration during CPR, because limited cephalad flow occurs below the diaphragm.[41]

If an endotracheal tube has been placed but venous access is delayed, medication (specifically, epinephrine, lidocaine, atropine) can be administered by instillation into the endotracheal tube. These medications should be administered intravenously at 2 to 2.5 times the recommended dose after first being diluted in 10 mL of normal saline.[42]

Epinephrine

Adrenergic drugs that are effective in improving rates of resuscitation share the common property of α-agonist activity. Myocardial blood flow is determined by the pressure gradient created between the aorta and the right atrium at the onset of diastole (ie, coronary perfusion pressure). The addition of any α-agonist agent to basic CPR has been shown to increase this pressure. The mechanism by which these agents increase coronary perfusion pressure is via systemic arteriolar vasoconstriction, which maintains peripheral vascular tone and prevents arteriolar collapse—both of which are necessary to sustain blood flow through the cerebral and coronary circulations during the relaxation phase of chest compressions.

The α-agonist agent currently recommended by the American Heart Association is epinephrine, which has been in clinical use since 1906. Studies of epinephrine in 1906 showed that a dose of 0.1 mg/kg body weight clearly improved resuscitation rates and survival in 10-kg dogs.[43] This dose, when applied in humans, showed no evidence of comparable clinical efficacy; subsequent studies suggested the inadequacy of the standard dose of epinephrine.[44,45] A dose-dependent increase in arterial pressures and myocardial and cerebral blood flow was demonstrated when epinephrine was used in a dose range of 0.045 mg/kg to 0.2 mg/kg.[44–46] On a mg/kg basis, the equivalent dose for a 70-kg human would be 3 to 14 mg. Following these investigations, a number of studies have evaluated the use of high-dose epinephrine in terms of survival and neurologic outcome, but the results of these studies have been mixed.[9,46]

Vasopressin

Vasopressin is a potent vasoconstrictor commonly used to control bleeding in patients with esophageal varices. Recently, vasopressin has also been used in patients with cardiac arrest with encouraging results.[47] In fact, the new guidelines of the American Heart Association recommend the use of a single dose of vasopressin (40 U) in lieu of epinephrine in cases of VF.

Bretylium and Lidocaine

Bretylium and lidocaine have been the 2 major antiarrhythmic agents used during CPR for the past 2 decades. Although these drugs have been shown to be effective in suppressing ventricular dysrhythmias following myocardial infarction, their actual benefit is cases of cardiac arrest is less clear. Studies of patients with out-of-hospital VF have shown that bretylium and lidocaine are equally effective in their ability to restore an organized rhythm in patients with VF who are not responsive to defibrillation and epinephrine.[48]

Amiodarone

Amiodarone is an antidysrhythmic agent shown to be particularly effective in the treatment of refractory ventricular dysrhythmias; reports have described its ability to rapidly suppress malignant ventricular dysrhythmias when administered intravenously.[49, 50] Animal studies have shown a decrease in the defibrillation threshold when amiodarone is administered intravenously and an improved response to defibrillation in resistant VF. A clinical trial randomly assigned 504 patients with nontraumatic out-of-hospital cardiac arrest who had not responded to three initial electrical shocks to receive a bolus of either amiodarone or placebo. In this study, survival to a hospital was significantly higher in the amiodarone group (44% versus 35%).[51] These studies suggest that amiodarone may become the antidysrhythmic agent of choice in patients who fail initial attempts at defibrillation. Although there is still some controversy surrounding the use of this agent, the authors of this review favor its use in cases of cardiac arrest.

Magnesium

There is considerable evidence showing that magnesium can be helpful in the suppression of ventricular dysrhythmias. The benefits of magnesium are not limited to

patients with hypomagnesemic states but apply as well to cases of torsades de pointes and monomorphic VT.[52,53] However, empiric magnesium supplementation does not improve the rate of successful resuscitation or the survival of patients with in-hospital cardiac arrest.[54]

Sodium Bicarbonate

The use of buffer therapy during CPR has been a contentious issue. In the absence of pre-existing acidosis, arterial pH usually stays within normal limits during the first 5 to 10 minutes of CPR. The bulk of current evidence suggests that patients receiving optimal CPR derive no clinical benefit from early administration of buffers.[55] To date, evidence has failed to show a survival benefit caused by administration of sodium bicarbonate during CPR.[56] Although buffer agents do not improve the success of resuscitation, however, they may ameliorate postresuscitation myocardial dysfunction and thereby improve postresuscitation survival.[57]

At present, sodium bicarbonate is the recommended agent for patients with cardiac arrest in whom there is a known pre-existing metabolic acidosis, hyperkalemia, or specific intoxication (eg, cyclic antidepressant overdose). There may also be some benefit in the administration of sodium bicarbonate in cases of prolonged resuscitation times (ie, more than 10 minutes).

The lack of demonstrable efficacy of sodium bicarbonate in mitigating the acidosis of cardiac arrest has produced interest in carbon dioxide–consuming or noncarbon dioxide–producing buffer agents (eg, tromethamine, sodium carbonate). Unfortunately, despite improvements in arterial and venous pH, none of these agents buffers the profound intramyocardial acidosis that develops during cardiac arrest or improves the hemodynamics of CPR or resuscitation outcomes.[55,58]

Calcium

Because of the potential role of intracellular calcium accumulation in causing irreversible ischemic cell injury, the use of calcium with CPR is now recommended only in patients with hyperkalemia, calcium-channel blocker toxicity, or hypocalcemia.

CONCLUSIONS

Early defibrillation has the greatest influence on survival in patients with cardiac arrest. Consequently, all responders—including nonmedical personnel—should be instructed and encouraged to use defibrillators. In cases of unwitnessed cardiac arrest in which there is a delay before defibrillation, use of chest compressions prior to the first shock may improve the survival rate. Although defibrillation and chest compressions are essential for successful resuscitation, recent evidence suggests that ventilation may not be required in the early stages of CPR. Although administration of epinephrine is recommended in current advance cardiac life support guidelines, evidence of the drug's efficacy is lacking. Early experimental and clinical data suggest that vasopressin may be the pressor of choice in the management of patients with cardiac arrest. Similarly, amiodarone may prove to be a more efficacious antidysrhythmic agent than lidocaine in patients who have had unsuccessful defibrillation. **HP**

REFERENCES

1. Varon J, Sternbach GL. Cardiopulmonary resuscitation: lessons from the past. J Emerg Med 1991;9:503–7.
2. Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed chest cardiac massage. JAMA 1960;173:1064–7.
3. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of communitywide emergency cardiac care. JAMA 1992;268:2289–95.
4. Kremers MS, Black WH, Wells PJ. Sudden cardiac death: etiologies, pathogenesis, and management. Dis Mon 1989;35:381–445.
5. Varon J, Fromm RE Jr. In-hospital resuscitation among the elderly: substantial survival to hospital discharge. Am J Emerg Med 1996;14:130–2.
6. Schneider AP 2nd, Nelson DJ, Brown DD. In-hospital cardiopulmonary resuscitation: a 30-year review. J Am Board Fam Pract 1993;6:91–101.
7. McGrath RB. In-house cardiopulmonary resuscitation—after a quarter of a century. Ann Emerg Med 1987; 16:1365–8.
8. Marik PE, Craft M. An outcomes analysis of in-hospital cardiopulmonary resuscitation: the futility rationale for do not resuscitate orders. J Crit Care 1997;12:142–6.
9. Stiell IG, Hebert PC, Weitman BN, et al. High-dose epinephrine in adult cardiac arrest. N Engl J Med 1992; 327:1045–50.
10. Becker LB, Ostrander MP, Barrett J, Kondos GT. Outcome of CPR in a large metropolitan area—where are the survivors? Ann Emerg Med 1991;20:355–61.
11. Lombardi G, Gallagher J, Gennis P. Outcome of out-of-hospital cardiac arrest New York City: The Pre-Hospital Arrest Survival Evaluation (PHASE) study. JAMA 1994; 271:678–83.
12. Eisenberg MS, Bergner L, Hallstrom A. Cardiac resuscitation in the community. Importance of rapid provision and implications for program planning. JAMA 1979; 241:1905–7.
13. Cobb LA, Fahrenbruch CE, Walsh TR, et al. Influence of cardiopulmonary resuscitation prior to defibrillation in patients with out-of-hospital ventricular fibrillation. JAMA 1999;281:1182–8.

14. Tang W, Weil MH, Sun S. Low-energy biphasic waveform defibrillation reduces the severity of postresuscitation myocardial dysfunction. Crit Care Med 2000;28: N222–4.
15. Leng CT, Paradis NA, Calkins H, et al. Resuscitation after prolonged ventricular fibrillation with use of monophasic and biphasic waveform pulses for external defibrillation. Circulation 2000;101:2968–74.
16. Mittal S, Ayati S, Stein KM, et al. 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;34:1595–601.
17. Auble TE, Menegazzi JJ, Paris PM. Effect of out-of-hospital defibrillation by basic life support providers on cardiac arrest mortality: a metaanalysis. Ann Emerg Med 1995; 25:642–8.
18. Caldwell G, Millar G, Quinn E, et al. Simple mechanical methods for cardioversion: defence of the precordial thump and cough version. Br Med J (Clin Res Ed) 1985; 291:627–30.
19. Cummins RO. Options to provide earlier definitive care. In: Eisenberg MS, Bergner L, Hallstrom AP, editors. Sudden cardiac death in the community. New York: Praeger Scientific; 1984:87–100.
20. Diack AW, Wellborn WS, Rullman RG, et al. An automatic cardiac resuscitator for emergency treatment of cardiac arrest. Med Instrum 1979;13:78–83.
21. Cobbe SM, Redmond MJ, Watson JM, et al. "Heartstart Scotland"—initial experience of a national scheme for out of hospital defibrillation. BMJ 1991;302:1517–20.
22. Weaver WD, Cobb LA, Dennis D, et al. Amplitude of ventricular fibrillation waveform and outcome after cardiac arrest. Ann Intern Med 1985;102:53–5.
23. Lerman BB, DiMarco JP, Haines DE. Current-based versus energy-based ventricular defibrillation: a prospective study. J Am Coll Cardiol 1988;12:1259–64.
24. Fromm RE, Varon J. Automated external versus blind manual defibrillation by untrained lay rescuers. Resuscitation 1997;33:219–21.
25. Paradis NA, Martin GB, Goetting MG, et al. Simultaneous aortic, jugular bulb, and right atrial pressures during cardiopulmonary resuscitation in humans. Insights into mechanisms. Circulation 1989;80:361–8.
26. Varon J, Fromm RE Jr. Cardiopulmonary resuscitation. New and controversial techniques. Postgrad Med 1993; 93:235–9.
27. Weil MH, Rackow EC, Trevino R, et al. Difference in acid-base state between venous and arterial blood during cardiopulmonary resuscitation. N Engl J Med 1986; 315:153–6.
28. Varon J, Fromm RE. Special techniques. In: Varon J, editor. Practical guide to the care of the critically ill patient. St. Louis: Mosby; 1994:321–39.
29. Chandra BN, Gruben KG, Tsitlik JE, et al. Ventilation during CPR [abstract]. Circulation 1991;84(suppl 2): II–9.
30. Van Hoeyweghen RJ, Bossaert LL, Mullie A, et al. Quality and efficiency of bystander CPR. Belgian Cerebral Resuscitation Study Group. Resuscitation 1993;26: 47–52.
31. Ornato JP. Should bystanders perform mouth-to-mouth ventilation during resuscitation? Chest 1994;106:1641–2.
32. Hallstrom A, Cobb L, Johnson E, Copass M. Cardiopulmonary resuscitation by chest compression alone or with mouth-to-mouth ventilation. N Engl J Med 2000; 342:1546–53.
33. Molokhia FA, Ponn RB, Robinson WJ, et al. A method of augmenting coronary perfusion during internal cardiac massage. Chest 1972;62:610–3.
34. Barranco F, Lesmes A, Irles JA, et al. Cardiopulmonary resuscitation with simultaneous chest and abdominal compression: comparative study in humans. Resuscitation 1990;20:67–77.
35. Sack JB, Kesselbrenner MB, Bregman D. Survival from in-hospital cardiac arrest with interposed abdominal counterpulsation during cardiopulmonary resuscitation. JAMA 1992;267:379–85.
36. Mateer JR, Stueven HA, Thompson BM, et al. Interposed abdominal compression CPR versus standard CPR in prehospital cardiopulmonary arrest: preliminary results. Ann Emerg Med 1984;13:764–6.
37. Halperin HR, Tsitlik JE, Gelfand M, et al. A preliminary study of cardiopulmonary resuscitation by circumferential compression of the chest with use of a pneumatic vest. N Engl J Med 1993;329:762–8.
38. Stiell IG, Hebert PC, Wells GA, et al. The Ontario trial of active compression-decompression cardiopulmonary resuscitation for in-hospital and prehospital cardiac arrest. JAMA 1996;275:1417–23.
39. Asplin BR, White RD. Prognostic value of end-tidal carbon dioxide pressures during out-of-hospital cardiac arrest. Ann Emerg Med 1995;25:756–61.
40. Emerman CL, Pinchak AC, Hancock D, Hagen JF. Effect of injection site on circulation times during cardiac arrest. Crit Care Med 1988;16:1138–41.
41. Emerman CL, Bellon EM, Lukens TW, et al. A prospective study of femoral versus subclavian vein catheterization during cardiac arrest. Ann Emerg Med 1990;19:26–30.
42. Aitkenhead AR. Drug administration during CPR: what route? Resuscitation 1991;22:191–5.
43. Crile G, Dolley DN. An experimental research into the resuscitation of dogs killed by anesthetics and asphyxia. J Exp Med 1906;8:713–25.
44. Brown CG, Taylor RB, Werman HA, et al. Effect of standard doses of epinephrine on myocardial oxygen delivery and utilization during cardiopulmonary resuscitation. Crit Care Med 1988;16:536–9.
45. Gonzalez ER, Orneto JP, Garnett AR, et al. Dose-dependent vasopressor response to epinephrine during CPR in human beings. Ann Emerg Med 1989;18: 920–6.
46. Barton C, Callaham M. High-dose epinephrine improves the return of spontaneous circulation rates in

human victims of cardiac arrest. Ann Emerg Med 1991; 20:722–5.
47. Lindner KH, Dirks B, Strohmenger HU, et al. Randomised comparison of epinephrine and vasopressin in patients with out-of-hospital ventricular fibrillation. Lancet 1997;349:535–7.
48. Olson DW, Thompson BM, Darin JC, Milbrath MH. A randomized comparison study of bretylium tosylate and lidocaine in resuscitation of patients from out-of-hospital ventricular fibrillation in a paramedic system. Ann Emerg Med 1984;13:807–10.
49. Ochi RP, Goldenberg IF, Almquist, A, et al. Intravenous amiodarone for the rapid treatment of life-threatening ventricular arrhythmias in critically ill patients with coronary artery disease. Am J Cardiol 1989;64:599–603.
50. Williams ML, Woelfel A, Cascio WE, et al. Intravenous amiodarone during prolonged resuscitation from cardiac arrest. Ann Intern Med 1989;110:839–42.
51. Kudenchuk PJ, Cobb LA, Copass MK, et al. Amiodarone for resuscitation after out-of- hospital cardiac arrest due to ventricular fibrillation. N Engl J Med 1999;341:871–8.
52. Tzivoni D, Banai S, Schuger C, et al. Treatment of torsade de pointes with magnesium sulfate. Circulation 1988;77:392–7.
53. Allen BJ, Brodsky MA, Capperelli EV, et al. Magnesium sulfate therapy for sustained monomorphic ventricular tachycardia. Am J Cardiol 1989;64:1202–4.
54. Thel MC, Armstrong AL, McNulty SE, et al. Randomised trial of magnesium in in-hospital cardiac arrest. Duke Internal Medicine Housestaff. Lancet 1997;350:1272–6.
55. Stahmer SA, Varon J, Fromm RE Jr. Controversies in cardiopulmonary resuscitation pharmacotherapy. Hosp Physician 1994;30(7):23–30.
56. Federiuk CS, Sanders AB, Kern KB, et al. The effect of bicarbonate on resuscitation from cardiac arrest. Ann Emerg Med 1991;20:1173–7.
57. Heimbach DM, Waeckerle JF. Inhalation injuries. Ann Emerg Med 1988;17:1316–20.
58. Rattenborg CC. Effect of bicarbonate and THAM on apnea-induced hypercarbia. In: Safar P, Elam J, editors. Advances in cardiopulmonary resuscitation. New York: Springer-Verlag; 1977:128–38.

Copyright 2001 by Turner White Communications Inc., Wayne, PA. All rights reserved.