Chapter 129
Cardiac Arrest and Resuscitation

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The modern era of cardiopulmonary resuscitation (CPR) has been marked by the development of closed-chest cardiac massage, artificial respiration, and electrical defibrillation into a practical set of techniques for the treatment of patients in cardiac arrest. After its introduction in 1960, closed-chest CPR was shown to be successful in resuscitating approximately 25% of patients from in-hospital cardiac arrests. However, it was soon realized that the vast majority of patients suffered cardiac arrest outside a hospital and that most patients who die of myocardial infarction succumb to arrhythmias within the first hour after the infarction. The concept of extending treatment into the community was developed in the 1970s. Pioneering studies in Belfast, New York, Seattle, and other cities demonstrated that emergency medical services (EMS) providing advanced cardiac life support (ACLS) by trained paramedics could significantly improve resuscitation and survival in patients suffering cardiac arrest. Thus an effective approach to cardiac arrest required the development of new concepts for bringing emergency care into the community.6,15

Improvements in the treatment of patients in cardiac arrest have generally come by evaluating cardiac arrest as a systems problem that requires new solutions beyond the traditional medical model. Advances have come with community solutions such as the development of an out-of-hospital community emergency medical care system and the training of laypersons to deliver CPR.16 More recently, the development of the automatic external defibrillator further extends the possibilities for community treatment. Public-access defibrillation holds the promise of providing early defibrillation in the community by trained persons, such as security guards and flight attendants, prior to the arrival of the EMS medics.6,15,16

BASIC CARDIOPULMONARY RESUSCITATION

The function of basic CPR is to provide some blood flow to vital organs until more definitive treatment, such as defibrillation, can be initiated. There has been some debate about the efficacy of CPR in the resuscitation of patients in cardiac arrest. Although studies from one city with a rapid-response EMS system have failed to show the benefit of layperson CPR in the resuscitation of victims of cardiac arrest, other studies from several cities have demonstrated that the earlier CPR is applied, the better is the outcome.6,15 It is important to remember, however, that CPR itself usually does not resuscitate patients from cardiac arrest. CPR must be accompanied by the early institution of advanced life support.

The importance of early defibrillation is emphasized by the concept of “phone first,” in which laypersons are taught to activate the EMS system before CPR is started in adults in cardiac arrest.15 This concept recognizes the importance of getting the defibrillator to the scene as soon as possible. The standard technique of CPR includes chest compression and ventilation. It is designed to provide some blood flow to vital organs until more definitive

therapy, such as defibrillation, is available.

There has also been much debate about the mechanism of blood flow during closed-chest compression. Some authorities believe that the thorax acts as a pump when the chest is compressed and that pressure gradients are generated between the intrathoracic and extrathoracic structures. Other authorities believe that the heart itself is compressed during chest massage and that pressure gradients are developed between intracardiac and extracardiac structures. If the mechanism of blood flow during CPR were known, techniques could be developed to increase blood flow to vital organs. For example, if the thorax-pump mechanism of blood flow predominates, techniques that maximize thoracic pressure differences, such as simultaneous ventilation and compression, or abdominal binding, might improve cardiac output during CPR. On the other hand, if the heart acts as the pump during chest compression, one could improve cardiac output by increasing the number of chest compressions per minute. Although studies investigating these alternative forms of CPR have yielded encouraging results in specific laboratories or animal models, there are not yet enough data in humans to determine which mechanism of blood flow predominates. There may even be a dimorphic population, meaning that thorax compression may predominate in some persons and cardiac compression in others.

Thus far, no alternative CPR techniques have yielded improved resuscitation or survival rates in clinical studies. Therefore, the standard technique of CPR has changed little over the past decade.

Airway

In approaching the victim of cardiac arrest, one must assess unresponsiveness, activate the system for initiating advanced life support, and position the patient on the floor or a backboard to assess the airway. The tongue may fall back and obstruct the airway. Rescuers may open the airway by using the head tilt-chin lift maneuver (Fig. 129.1). The rescuer places backward pressure

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on the patient's forehead while lifting the chin anteriorly. This maneuver pushes the tongue and epiglottis forward, removing the obstruction.

Figure 129.1. Opening the airway. **(Top)** Airway obstruction produced by tongue and epiglottis. **(Bottom)** Obstruction is relieved by head tilt-chin lift. (Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care, I: introduction. *JAMA* 1992;268:2172.)

The head tilt-chin lift maneuver should not be used if neck injury is suspected for any reason. In patients with possible neck injury, the jaw thrust maneuver is used. Pressure applied to the angles of the mandible moves the jaw anteriorly, opening the airway.

Breathing

Once the airway is open, the rescuer looks, listens, and feels for an exchange of air. If the

patient is not breathing, mouth-to-mouth respirations are administered by pinching the victim's nostrils and blowing slowly into the mouth for two breaths. The rescuer watches for the rise and fall of the chest during ventilation. The patient receives ten to 12 breaths per minute, with each ventilation lasting 1.5 to 2.0 seconds. Slow ventilations, with time for complete exhalations, may result in less gastric distention and less potential for regurgitation and aspiration. The patient should be intubated and ventilated with oxygen as soon as it is practical in the ACLS sequence. After endotracheal intubation, the rescuers do not need to pause between compressions for ventilation to be given.

Obstructed Airway

If the rescuer has optimally positioned the jaw, chin, and tongue and still cannot detect adequate air exchange, one must be concerned about an obstructed airway. The Heimlich maneuver, or subdiaphragmatic abdominal thrust, is the recommended procedure for clearing the obstructed airway of a foreign body (Fig. 129.2). The rescuer's hand is positioned between the patient's xiphoid and navel, and several quick thrusts are administered in an attempt to relieve the foreign-body obstruction. In the unconscious victim, the Heimlich maneuver may be accompanied by attempts to visualize the foreign body in the pharynx and by finger sweeps to remove the foreign body. Complications of the Heimlich maneuver include regurgitation, aspiration, and traumatic injury to the abdomen.15

Figure 129.2. (Top) Administration of Heimlich maneuver to *conscious* victim of foreign-body airway obstruction. **(Bottom)** Administration of Heimlich maneuver to *unconscious* victim of foreign-body airway obstruction. (Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care, I: introduction. *JAMA* 1992;268:2172.)

Circulation

The rescuer determines pulselessness through palpation of the carotid. If no pulse is detected, chest compressions should be instituted.

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After placing the victim on a backboard or firm surface, the heel of the hand is placed on the lower half of the sternum and depressed 1.5 to 2.0 in. The recommended compression rate is 80 to 100 times per minute.15 The duration of compression should be 50% of the entire compression-release cycle. A 5:1 ratio of compression to ventilation is used when there are two or more rescuers. When there is only one rescuer, the ratio should be 15 compressions to two ventilations.15

Closed-chest compression can provide 10% to 30% of normal cardiac output. The key hemodynamic parameter that predicts whether resuscitation will be successful is the aortic diastolic pressure. Animal studies have shown that if an aortic diastolic pressure of 40 mm Hg can be generated during CPR, there is an excellent chance of successful resuscitation, presumably because it is during diastole that the coronary arteries are filled.

Mechanical resuscitators are commercially available that deliver standard American Heart Association chest compression and ventilation. These resuscitators have been shown to produce pressures comparable to manual CPR. Once applied, the resuscitators deliver standard CPR throughout the resuscitation effort, freeing personnel to perform ACLS.

Alternative Cardiopulmonary Resuscitation Techniques

A number of alternatives to the standard CPR technique have been proposed, based on theoretical advantages in achieving higher cardiac output during CPR. Although many of the techniques have shown promise in laboratory investigations and limited clinical studies, none has yielded improved survival from cardiac arrest when compared with standard CPR. The most promising techniques include interposed abdominal compression CPR (IAC-CPR), active compression-decompression CPR (ACD-CPR), simultaneous ventilation compression CPR (SVC-CPR), and high-frequency CPR.

IAC-CPR involves compression of the abdomen by a second rescuer during the relaxation phase of chest compression. IAC-CPR has been shown to improve the aortic systolic and diastolic pressure, as well as cardiac output and myocardial profusion pressures, when compared with standard CPR in experimental models. Clinical studies comparing IAC-CPR with standard CPR have shown some promise.13 The technique requires an additional rescuer, however, and compression of the abdomen may predispose to complications such as abdominal injury and aspiration if used in patients with unprotected airways. The role of IAC-CPR needs to be defined by future clinical studies.

ACD-CPR makes use of a suction device that is applied to the anterior thorax and creates negative intrathoracic pressure during the relaxation phase of CPR. Studies in experimental models and humans in cardiac arrest have demonstrated improvement in some hemodynamic parameters but not in others. Clinical trials of the device have demonstrated mixed results.4

SVC-CPR attempts to use the entire thorax as the pump to produce forward blood flow. Chest compression is applied at full inspiration to maximize the gradient between intrathoracic and extrathoracic structures. A newer circumferential vest CPR has shown improved hemodynamics in limited clinical trials.8 High-frequency or rapid-compression-rate CPR has been shown in experimental models to improve cardiac output, myocardial perfusion pressures, and 24-hour survival compared with standard CPR. Clinical studies are limited, but there is some evidence for improved hemodynamics using rapid manual compression rates.9

Other studies have questioned the role of ventilation as a standard part of layperson CPR. In experimental models of cardiac arrest, chest-compression-only CPR provides outcomes comparable to conventional CPR. While professional health-care providers will continue to provide ventilation with basic life support, further clinical trials are needed before any recommendations can be made regarding the option of chest-compression-only CPR for lay rescuers.2

Studies evaluating these alternative techniques of CPR appear to show benefits in some groups of patients but not in others. One explanation may be that certain physiologic characteristics may be more important in some subsets of patients than in others. For

example, patients who are obese or have large anterior-posterior chest diameters probably receive no direct cardiac compression during standard CPR but might benefit from techniques designed to take advantage of the thorax mechanism of forward blood flow, such as SVC-CPR. Patients with a thin body habitus, for whom cardiac compression may be the primary mechanism of forward blood flow, might benefit from techniques such as rapid-compression-rate CPR. It may be that the use of one standard method of CPR for all patients in cardiac arrest is inappropriate. At the present time, however, these alternative techniques of CPR should be considered experimental.

ADVANCED CARDIAC LIFE SUPPORT

Because the recommended treatment protocols are based on the patient's underlying rhythm, it is important that electrocardiographic (ECG) monitor leads be placed early and followed throughout the resuscitation attempt. If the patient is in ventricular fibrillation, immediate defibrillation is recommended even before CPR, intubation, and other interventions. As soon as possible, the patient is endotracheally intubated and an intravenous line is placed. Peripheral lines may be placed in the antecubital area, or central lines through the internal or external jugular veins. Peripheral lines should not be placed in the lower half of the body, because external chest compression results in retrograde blood flow through the vena cava (because there are no valves in the inferior vena cava).15

Defibrillation

Early defibrillation is the one intervention that has been repeatedly shown to be efficacious for patients in pulseless ventricular tachycardia or ventricular fibrillation. The longer the patient remains in fibrillation, the more likely it is that defibrillation and resuscitation will be unsuccessful.6,15 It is the current passing directly through the heart that allows defibrillation. Current is directly related to the energy set on the defibrillator and inversely related to the transthoracic impedance. The transthoracic impedance can be minimized by attention to several factors, including position of the defibrillation paddles, firm pressure on the paddles, the use of conductive material between the skin and paddles, employing sets of shocks (pairs or triplets), and coordinating the shock with the end-expiratory phase of ventilation.15

A precordial thump generates 0.5 to 1.0 joule of energy. Rarely, when applied early to a patient in cardiac arrest, a precordial thump can convert ventricular fibrillation to a perfusing rhythm; pulseless ventricular tachycardia may be converted to a perfusing rhythm in 11% to 25% of cases. However, a thump can also convert ventricular tachycardia to a more malignant dysrhythmia, such as ventricular fibrillation, asystole, or pulseless electrical activity (PEA). The precordial thump may be used for patients with witnessed cardiac arrest while another rescuer is preparing the defibrillator. It should, however, never delay the definitive treatment—electrical defibrillation.

The automatic external defibrillator (AED) is capable of analyzing the rhythm of a patient in cardiac arrest and shocking the patient per ACLS protocols. Semiautomatic external defibrillators

analyze the rhythm and advise the rescuer to attempt defibrillation through the sensing electrodes.19 These devices extend the capability of EMS systems to reach victims of cardiac arrest earlier. Rescue workers and even spouses of high-risk patients can be trained to operate the AED. Some communities have trained basic emergency medical technicians (EMTs) in the recognition of ventricular fibrillation and the use of defibrillators, providing for early defibrillation in communities in which paramedics cannot respond to cardiac arrests within a few minutes.15 AEDs have also been advocated for use by other professionals who may come in contact with patients in cardiac arrest. These include security guards, airline flight attendants, and police officers in situations in which EMS responders might not be immediately available. The use of AEDs might help some communities solve the difficult issue of prompt defibrillation. The place of public access defibrillation in community EMS systems remains to be determined by future clinical studies and individual community needs and resources.

Pressor Agents

Epinephrine, a mixed α- and β-adrenergic agent, is the pressor drug of choice for patients in cardiac arrest.15 Epinephrine increases peripheral vascular resistance and raises aortic diastolic pressure, thus augmenting coronary blood flow. It also increases cerebral blood flow during cardiac arrest. The recommended dose is 1 mg initially, with repeated doses every 3 to 5 minutes,15 but the optimal dose has not been established. Although there is some evidence that higher doses of epinephrine may increase myocardial and central nervous system blood flow, several outcome studies have failed to demonstrate any difference in survival to hospital discharge with standard-dose compared with high-dose epinephrine.3 In fact, there is some evidence that high doses of epinephrine may result in worse neurologic outcome.1,7 Thus, after the initial 1-mg dose, subsequent doses may be higher based on the treating physician's discretion. Epinephrine can also be given through the endotracheal tube; the dose is two to two and one-half times the intravenous dose. Vasopressin is a potent vasopressor that has been shown to improve cardiac as well as cerebral blood flows in cardiac arrest. The role of vasopressin as an alternative to epinephrine is being investigated in larger clinical trials.12

Acid-Base Abnormalities

The optimal treatment of acidosis during cardiac arrest is hyperventilation. The role of bicarbonate administration is controversial. The patient in cardiac arrest develops lactic acidemia from poor tissue perfusion, but lactic acid does not accumulate significantly until relatively late in the course (20 or more minutes of total arrest time). Many patients in cardiac arrest also develop a venous respiratory acidosis because they are unable to transport CO_2 to the lungs where it may be excreted.20 This CO_2 readily crosses membranes and may worsen intracellular and central nervous system acidosis. If bicarbonate is given to a patient in cardiac arrest, CO_2 is generated by the buffering of metabolic acids and PCO_2 increases locally. Thus, bicarbonate may actually worsen the acid-base status. In addition, overzealous administration of bicarbonate can produce alkalosis and hyperosmolarity, both of which are associated with a poor prognosis. Clearly, however, there are patients in cardiac arrest in whom aggressive treatment with bicarbonate can be life-saving. These include patients with hyperkalemia and those with

tricyclic antidepressant overdosage. In addition, patients with other drug overdosages and those with preexisting metabolic acidosis may benefit from early treatment with bicarbonate. Arterial and central venous blood gases may be useful in assessing acid-base status and the advisability of administering bicarbonate for patients in cardiac arrest.

Antiarrhythmic Agents

No studies have shown that antiarrhythmics improve survival from cardiac arrest. Many of the antiarrhythmic agents raise the fibrillation threshold, however, and are thus useful in preventing recurrent ventricular fibrillation in patients who have been successfully defibrillated. Antiarrhythmic agents that may be beneficial in the treatment of patients in ventricular fibrillation include amiodarone, lidocaine, bretylium, magnesium sulfate, and procainamide. Amiodarone may be an important drug for the treatment of patients in persistent ventricular fibrillation or pulseless ventricular tachycardia resistant to electrical defibrillation. In one study,11 it improved the rate of return of spontaneous circulation and admission to the hospital for patients with out-of-hospital ventricular fibrillation unresponsive to electrical defibrillation. Studies assessing the effects of amiodarone on the rate of discharge from the hospital and on long-term survival will determine the place of amiodarone in ACLS algorithms. Lidocaine has been used in the treatment of patients in cardiac arrest for many years. In clinical studies, no difference in overall resuscitation or survival has been seen when lidocaine and bretylium are compared in patients with ventricular fibrillation. In general, when antiarrhythmic agents are administered, they should be followed by immediate attempts at electrical defibrillation so that the sequence of drug-shock-drug-shock is maintained.

Atropine

Atropine may be useful in some patients with asystolic cardiac arrest who have an excess of parasympathetic stimulation. Studies in the prehospital use of atropine have shown improved resuscitation rates but not improved overall survival in patients in asystolic arrest.17 Atropine is also useful in patients with symptomatic bradycardia.

Pacemakers in Cardiac Arrest

The wide adoption of transcutaneous cardiac pacing has extended the availability of pacemakers to many patients. Transcutaneous pacing capability is often a feature of newer defibrillators. Pacing is indicated in patients with hemodynamically unstable bradycardia and may also be useful for overdrive pacing patients with tachycardia unresponsive to pharmacologic therapy. Although the successful use of pacing for patients in asystolic cardiac arrest has been reported anecdotally, most studies have failed to show significant improvements in resuscitation rates. Thus, pacemakers cannot be recommended for routine use in asystolic cardiac arrest. If pacing is used in the management of patients in cardiac arrest, however, it should be applied early.

GUIDELINES FOR CARDIAC ARREST

Guidelines for the treatment of patients in cardiac arrest have been developed by the American Heart Association.15 They are updated every few years; clinicians should be

aware of the updated versions. Although the ACLS treatment for patients in cardiac arrest is based on the ECG monitor rhythm, the clinician must always consider the etiology of the arrest in developing a treatment plan. Cardiac arrest is the end point of a physiologic process that results in the loss of circulation or ventilation. Etiologies such as respiratory failure, asthma, medication toxicity, hypovolemia, pulmonary embolism, sepsis, trauma, acute myocardial infarction, anaphylaxis, metabolic abnormalities, and others may be treated specifically while going through the ACLS algorithm.10

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Patients in cardiac arrest present with either ventricular fibrillation-pulseless ventricular tachycardia (Fig. 129.3), asystole (Fig. 129.4), or PEA (Fig. 129.5). Treatment modalities have been classified by a consensus of experts based on the scientific evidence of their efficacy and safety, as follows:

Figure 129.3. Ventricular fibrillation-pulseless ventricular tachycardia (VF/VT) algorithm. (*1997–99 Handbook of emergency cardiovascular care for healthcare providers.* American Heart Association, Dallas, 1997.)

Figure 129.4. Asystole algorithm. (*1997–99 Handbook of emergency cardiovascular care for healthcare providers*. American Heart Association, Dallas, 1997.)

Figure 129.5. Pulseless electrical activity (PEA) algorithm. (*1997–99 Handbook of emergency cardiovascular care for healthcare providers*. American Heart Association, Dallas, 1997.)

Class I: Definitely helpful

Class IIA: Acceptable, probably helpful

Class IIB: Acceptable, possibly helpful

Class III: Not indicated, may be harmful

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Ventricular Fibrillation and Pulseless Ventricular Tachycardia

The key to the treatment of patients with ventricular fibrillation or pulseless ventricular tachycardia is to remember that successful resuscitation depends on electrical defibrillation. All other treatments, including CPR, intubation, and drugs, are merely attempts to prepare the patient for successful defibrillation. Defibrillation should be attempted with up to three shocks as soon as the diagnosis is made.15 Shocking the patient several times in a row decreases the transthoracic impedance and increases the

chances for successful defibrillation. If defibrillation attempts are unsuccessful, CPR is initiated, the trachea is intubated, and an intravenous line is placed as soon as possible. Epinephrine is the first recommended pharmacologic agent; repeat doses should be given every 3 to 5 minutes until defibrillation is successful. Each administration of drug should be followed by attempts at electrical defibrillation. Antiarrhythmic drugs may be useful in preventing refibrillation once the patient has been electrically defibrillated. Lidocaine is frequently given as the first antiarrhythmic agent. In the future, amiodarone may be the first antiarrhythmic drug recommended. If this is ineffective, treatment with bretylium, magnesium sulfate, and procainamide may be considered. Treatment with bicarbonate should be initiated if there is any clinical suspicion of hyperkalemia, tricyclic antidepressant, or preexisting acidosis.15

Asystole

The asystolic patient has a poor prognosis for resuscitation. However, because some patients who appear to be asystolic are actually in fine ventricular fibrillation, the diagnosis of asystole must be confirmed by checking for a straight-line rhythm on three leads. If there is any suspicion that ventricular fibrillation is present, one should proceed with defibrillation as in the ventricular fibrillation protocol. For asystole, one must consider possible causes such as hypoxia, drug overdose, hypothermia, and electrolyte imbalance. As noted previously, transcutaneous pacing has not generally been efficacious for patients in asystole. If it is used in selected patients, however, it should be applied

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early in the treatment protocol. Pharmacologic treatment of asystole involves the use of epinephrine to improve myocardial and cerebral blood flow during CPR, and atropine to antagonize parasympathetic stimulation.15

Pulseless Electrical Activity

PEA describes a situation in which there is an electrical rhythm other than ventricular tachycardia or fibrillation, but no palpable pulses. This condition may be due to true electromechanical dissociation (EMD), in which the heart muscle itself is not contracting despite the presence of electrical activity. True EMD has a very poor prognosis. However, PEA may also be due to states of severely decreased flow, so patients must be evaluated for cardiac and extracardiac causes of severe shock. Bedside ultrasonography may allow the treating physician to determine whether the heart is beating and whether pericardial fluid is present. Alternatively, the placement of an arterial line can be used to differentiate severe shock states from true EMD. In cases in which these modalities are not available, the clinician must

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consider causes such as hypovolemia, hypoxia, cardiac tamponade, tension pneumothorax, hypothermia, pulmonary embolism, drug overdose, acidosis, and electrolyte imbalance. A thorough reevaluation of the respiratory system is also critical. This may involve reintubation of the patient or confirmation of endotracheal tube placement both visually and with capnometry. The possibility of pneumothorax should be evaluated by listening to breath sounds bilaterally; if necessary, needle decompression can be performed to rule out a tension pneumothorax. Pericardiocentesis may help to identify cardiac tamponade if

bedside ultrasonography is not available. Arterial blood gases may be particularly useful in patients with PEA to determine oxygen and acid-base status. Empiric treatment with a fluid bolus while evaluating for acute blood loss from gastrointestinal hemorrhage, ruptured aortic aneurysm, or other causes for hypovolemia may also be useful. Once extracardiac causes of EMD are ruled out, treatment recommendations include epinephrine to improve myocardial and cerebral blood flow during cardiac arrest, and atropine if the ECG shows bradycardia.

Initiating and Discontinuing Resuscitation Efforts

CPR should be instituted for all patients in cardiac arrest, except in two circumstances:

1. The patient made it clear, while competent and informed, that he or she did not want resuscitation efforts to be instituted.
2. Successful resuscitation would be futile because the patient has clear signs of irreversible death, such as rigor mortis, dependent lividity, or decapitation.

Emergency physicians and paramedics are obligated to respect patients' wishes regarding resuscitation. In most cases, these wishes may be known through documentation on advance directives. Many EMS communities throughout the United States have developed a standardized “do not attempt resuscitation”' (DNAR) form that patients and their physicians can fill out, documenting their wishes, and present to paramedics. Portable or prehospital DNAR orders are likely to become more commonplace over the next decade and should be respected by emergency health-care professionals.

Once undertaken, resuscitation efforts are generally continued until it is clear that the patient is not responding to them. The longer the patient remains in cardiac arrest without response, the worse the prognosis for survival. There is, however, no definitive time interval that can predict the impossibility of survival after cardiac arrest. Indeed, special circumstances, such as hypothermic arrest in a child, may dictate prolonged resuscitation efforts. Thus, clinicians must use their judgment in going through the ACLS protocols and determining unresponsiveness to resuscitation.

INVASIVE CARDIOPULMONARY RESUSCITATION

Studies in animal models have shown that techniques of invasive CPR, such as open chest CPR and cardiopulmonary bypass, and the use of direct mechanical assist devices improve hemodynamics, resuscitation, and the chances of surviving cardiac arrest. It has also been demonstrated that when invasive CPR is applied late in the treatment protocol (after more than 15 to 20 minutes of total arrest time), there is no improvement in resuscitation. At the present time, however, these techniques should be used only in the context of well-defined experimental protocols and should not be used as last-ditch efforts for patients who do not respond to standard ACLS protocols.

QUALITY OF RESUSCITATION EFFORTS

How well CPR is performed and ACLS delivered does make a difference in the outcome for patients in cardiac arrest. Unfortunately, the performance of CPR is frequently not optimal.18 Ideally, the quality of resuscitation efforts might be best assessed by monitoring pressures during CPR so that the diastolic pressure gradient could be maximized, but this is clearly impractical except under special circumstances. The use of capnometry to monitor end-tidal CO_2 levels during CPR gives clinicians a noninvasive assessment of the cardiac output generated by CPR.14 The quality of resuscitation efforts should also be assessed retrospectively by systematically tracking key variables and the outcome of resuscitation efforts in both the in-hospital and out-of-hospital setting.5

PROGNOSIS

The prognosis for survival of cardiac arrest depends on many factors. These include patient variables such as the etiology of the arrest and comorbid diseases, arrest variables such as initial rhythm, and system variables such as time to CPR and defibrillation.5 None of these variables, by themselves or in combination, can accurately predict survival or neurologic outcome. However, it has been demonstrated in several studies that the longer the patient remains in cardiac arrest, the poorer the chances for successful resuscitation.6

The presenting rhythm is the most important factor in overall prognosis. Rhythms that are amenable to treatment with electrical defibrillation (ventricular tachycardia and ventricular fibrillation) have a relatively good prognosis, whereas asystole and EMD usually have poor outcomes. Resuscitation success for patients in ventricular fibrillation depends on the time elapsed before the patient is defibrillated; the longer the patient remains in ventricular fibrillation, the less likely is successful defibrillation.6,15 In units where staff are prepared to treat cardiac patients for ventricular arrhythmias promptly, successful defibrillation is nearly universal.

Another important factor in predicting resuscitation is the time that elapses before CPR is initiated. By providing some blood flow to the heart and other vital organs, one can extend the time in which defibrillation is effective. For example, data from Seattle show that if ACLS is started within 8 minutes of cardiac arrest, there is a successful outcome in 27% of patients. For those patients who have CPR started within 4 minutes in addition to having ACLS within 8 minutes, successful resuscitation is increased to 43%.6

Finally, the cause of the cardiac arrest is another factor important for prognosis.5 In cardiac arrest due to extracardiac causes such as pulmonary embolism and hypovolemia, the prognosis is very poor. In addition, specific pathophysiologic entities, such as complete thrombosis of the left main coronary artery, have poor prognoses, because providing adequate coronary blood flow is impossible no matter how quickly CPR and ACLS are provided.

COMMON PITFALLS

- It is important to remember that resuscitation efforts require a well-disciplined team in which each member plays a role. Often, cardiac arrests are managed in a chaotic, undisciplined environment that is not conducive to optimal patient care. Well-

disciplined teams develop through frequent drills and critique. Each resuscitation should be carefully reviewed. Hospitals and prehospital care systems should constantly

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monitor the success of their resuscitation efforts and compare them with studies in the literature.

- After the placement of an endotracheal tube, bilateral equal breath sounds should be heard in the axilla. Correct placement of the tube can be confirmed by quantitative capnometry. Breath sounds heard anteriorly may be transmitted from the stomach. Breath sounds and capnometry readings should be checked repeatedly during the resuscitation. Asymmetry of breath sounds or a sudden absence of CO_2 excretion mandates prompt evaluation—the endotracheal tube may have slipped out or down into the right bronchus or the patient may have developed a pneumothorax secondary to the trauma of chest compressions. Either of these serious conditions can be easily treated if recognized early.
- Intracardiac medications should be avoided. If an intravenous line cannot be started, many standard advanced life-support medications, including epinephrine, atropine, and lidocaine (*but not bicarbonate*), can be administered through the endotracheal tube.
- Hyperkalemia should be suspected as the cause of cardiac arrest in any patient with renal failure. The ECG may show a characteristic sine wave pattern, which should not be mistaken for ventricular fibrillation. Patients with hyperkalemia will not respond until the potassium level is lowered. Vigorous treatment with bicarbonate and calcium is indicated when hyperkalemia is suspected.
- Most patients in ventricular fibrillation can be defibrillated. Repeated unsuccessful defibrillation attempts may be due to poor technique. Successful defibrillation involves the depolarization of a critical mass of left ventricular myocardium. One paddle should be placed to the right of the sternum below the right clavicle, and the other in the midaxillary line at the level of the nipple. If the paddles are too close together, the skin, rather than the heart, may be getting the current. Alternatively, the anteroposterior position may be used over the left precordium. Firm pressure of approximately 25 lb should be applied to each paddle, and the appropriate contact gel must be used. The chest should be in full expiration when shocks are administered, because air in the lungs can significantly add to transthoracic impedance. Repeated shocks decrease the transthoracic impedance and improve the chances of successful defibrillation; therefore, the shocks may be delivered in pairs or triplets, with only a short pause between shocks to check for a pulse and the rhythm on the monitor.
- Torsades de pointes can be mistaken for ventricular tachycardia or fibrillation. The ECG reveals cycles of QRS complexes “twisting around” the isoelectric point of the ECG. It is associated with a congenital or drug-induced prolonged QT interval, most frequently due to quinidine. Although antiarrhythmic drugs are commonly ineffective in terminating torsades, electrical cardioversion is usually successful. Further

treatment with magnesium sulfate, isoproterenol, or overdrive pacing decreases the QT interval and prevents recurrence.15

- There are no good parameters that can be used to assess the effectiveness of ongoing CPR or to guide treatment during the resuscitation effort. Femoral pulsations may reflect venous rather than arterial blood flow. Carotid pulsations may reflect some blood flow but cannot be used as a guide to decision making during resuscitation. The longer the patient remains in cardiac arrest, the worse is the prognosis for resuscitation. After 30 minutes of resuscitation attempts, the prognosis is very poor, although there are case reports of successful resuscitation after hours of CPR. Therefore, no clear guidelines can be given as to when resuscitation efforts should be stopped.
- In patients who are hypothermic, as in near-drowning victims, resuscitation efforts should continue until the patient has been adequately warmed. Strong consideration should be given to cardiopulmonary bypass for these patients. In other patients, resuscitation efforts should be continued until the patient is unresponsive to ACLS protocols as outlined.

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