Review Article

Primary Care

CARDIAC RESUSCITATION

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At least 225,000 people in the United States will die suddenly this year from coronary heart disease before they reach a hospital.[1,2] In addition, an estimated 370,000 to 750,000 patients will have a cardiac arrest and undergo attempted resuscitation during hospitalization.[3] The causes of cardiac arrest are numerous; by far the most common in adults is ischemic cardiovascular disease.[4-6] The arrest is usually associated with the lethal arrhythmia of ventricular fibrillation triggered by an acutely ischemic or infarcted myocardium or by a primary electrical disturbance. The precipitants of a life-threatening arrhythmia such as ventricular fibrillation are poorly understood.[6,7] The demographic profile of persons with out-of-hospital cardiac arrest due to underlying cardiovascular disease is shown in Table 1.

TABLE 1. DEMOGRAPHIC CHARACTERISTICS OF 5213 PERSONS WITH OUT-OF-HOSPITAL CARDIAC ARREST DUE TO PRESUMED CARDIOVASCULAR DISEASE IN KING COUNTY, WASHINGTON, 1990–1999.*

VARIABLE	VALUE
Annual incidence	0.5/1000
Male sex — no. (%)	3573 (69)
Female sex — no. (%)	1635 (31)
Average age — yr	69
Average age of males — yr	67
Average age of females — yr	72
Arrest before arrival of emergency medical services — no./total no. (%)	4457/4914 (90)
Arrest after arrival of emergency medical services — no./total no. (%)	484/4914 (10)
Witnessed collapse — no./total no. (%)	2895/4914 (59)
Cardiopulmonary resuscitation by bystander — no./total no. (%)	2416/4339 (56)
Rhythm — no./total no. (%)	
Asystole	1606/5213 (31)
Pulseless electrical activity	496/5213 (10)
Ventricular fibrillation	2334/5213 (45)
Ventricular tachycardia	57/5213 (1)
Other	720/5213 (14)
Location of arrest — no./total no. (%)	
Home or other residence	3725/5213 (71)
Public place	1077/5213 (21)
Nursing home	411/5213 (8)

*The table is based on unpublished data from King County Emergency Medical Services Division, King County, Washington. Deaths due to presumed cardiovascular disease represent 71 percent of all nontraumatic sudden deaths treated by emergency medical services. Presumptive causes are based on death certificates and paramedics' reports. Other causes of cardiac arrest include respiratory causes, 8 percent; cancer, 4 percent; neurologic causes, 3 percent; suicide, 3 percent; drug overdose, 3 percent; sudden infant death syndrome, 2 percent; and miscellaneous causes, 6 percent. Not all information was available for all cases. Percentages may not add to 100 due to rounding. Only cases in which cardiopulmonary resuscitation was initiated or continued by emergency-medical-services personnel are included. Cases in which the person was dead on arrival at the hospital are not included; such cases account for approximately 25 percent of calls for nontraumatic cardiac arrest.

THE CHAIN OF SURVIVAL

Cardiac resuscitation in adults follows the same sequence whether it occurs in the community, the clinic, or an unmonitored hospital setting. First, one should call for help by activating the emergency-medical-services system in the community (by telephoning 911 in most U.S. locales) or the code team in the hospital. Second, one should begin cardiopulmonary resuscitation until advanced help arrives. Third, one should assess the heart rhythm and defibrillate the heart if indicated. Fourth, one should administer medications and protect the airway. This sequence (rapid access, rapid cardiopulmonary resuscitation, rapid defibrillation, and rapid advanced care) is termed the chain of survival.[8] If all four links in the chain come together quickly, there is a good chance of a successful resuscitation.[9-12] There are no national statistics on survival of out-of-hospital ventricular fibrillation in the United States; communities report rates ranging from 4 to 33 percent.[13-15] The higher rates are in communities able to provide the links rapidly. With inpatient cardiac arrest, the survival rates are variously reported as 0 to 29 percent.[16]

Other factors influencing survival include the rhythm associated with the arrest, whether the collapse was witnessed, and the underlying health of the patient.[17,18] The outcomes for out-of-hospital cardiac arrest in King County, Washington, are shown in Figure 1.[19] The rate of survival to hospital discharge for patients with a witnessed collapse who are found to be in ventricular fibrillation is 34 percent. The rates for patients with an unwitnessed collapse or with rhythms other than ventricular fibrillation are considerably lower.

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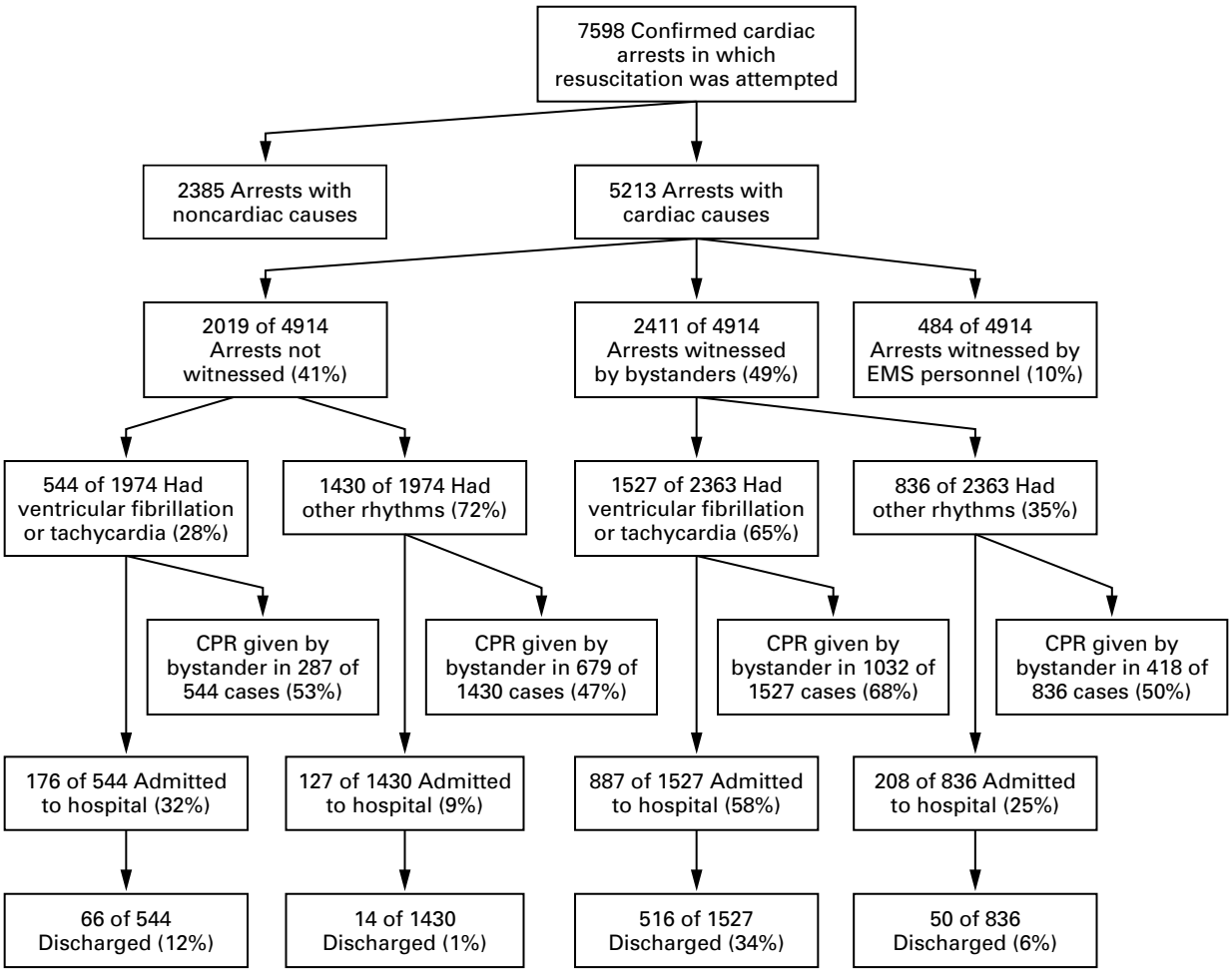

Figure 1. Survival after Nontraumatic Cardiac Arrest Due to Presumed Cardiovascular Disease, King County, Washington, 1990–1999. The figure is based on unpublished data from King County Emergency Medical Services Division. Deaths due to presumed cardiovascular disease represent 71 percent of all nontraumatic sudden deaths treated by emergency medical services. Presumptive causes are based on death certificates and paramedics' reports. Other causes of cardiac arrest include respiratory causes, 8 percent; cancer, 4 percent; neurologic causes, 3 percent; suicide, 3 percent; drug overdose, 3 percent; sudden infant death syndrome, 2 percent; and miscellaneous causes, 6 percent. Not all information was available for all cases. Percentages are calculated from the known data only. EMS denotes emergency medical services, and CPR cardiopulmonary resuscitation. The number of confirmed cardiac arrests includes only cases in which cardiopulmonary resuscitation was initiated or continued by emergency-medical-services personnel. Cases in which the person was dead on arrival at the hospital are not included; such cases account for approximately 25 percent of calls for nontraumatic cardiac arrest.

Rapid Access

Someone who encounters a collapsed person should immediately determine whether the person is unresponsive by shaking or shouting at him or her. If there is no response, help should be summoned. A person in cardiac arrest will be unresponsive and will have no pulse, but agonal respirations may be present for several minutes.

Rapid Cardiopulmonary Resuscitation

Since its description more than 40 years ago,[20,21] the fundamentals of cardiopulmonary resuscitation have remained unchanged. Mouth-to-mouth ventilation oxygenates the blood, and chest compressions cause forward blood flow. The latter is apparently caused by a combination of direct compression of the heart and changes in intrathoracic pressure.[22,23] Cardiopulmonary resuscitation by itself cannot defibrillate a heart. Its main benefit is to extend the patient's viability, thus allowing a defibrillator to reach the patient in time to be effective. Cardiopulmonary resuscitation, though hardly as efficient as a contracting heart, can result in some cardiac output. When cardiopulmonary resuscitation is started within four minutes after collapse, the likelihood of survival to hospital discharge doubles.[24] Millions of people have learned the funda-

mentals of cardiopulmonary resuscitation, and instructions for cardiopulmonary resuscitation are available on the Internet at http://www.learncpr.org.

The sequence for cardiopulmonary resuscitation performed by one person is as follows: The airway is opened by tilting the patient's head backward, lifting the chin, or both. (In the case of suspected trauma to the neck, the airway is opened by thrusting the jaw forward.) The rescuer looks, listens, and feels for respirations. If the respirations are agonal or absent, two mouth-to-mouth ventilations are performed (with each breath delivered slowly over a period of two seconds). Adequacy of ventilation is determined by observing the chest rise and fall. Health care professionals should then feel for a carotid pulse, taking 5 to 10 seconds to do so. If there is no pulse, 15 chest compressions are administered. The American Heart Association no longer recommends that lay rescuers perform pulse checks.[25] (Lay rescuers should initiate chest compressions if the patient is not breathing, coughing, or moving after the initial two breaths.) Chest compression should be administered in the center of the chest on the lower half of the sternum. The depth of compression should be 4 to 5 cm (1½ to 2 in.) in adults, and the rate of compression should be approximately 100 per minute. Resuscitation is continued, with 2 breaths alternating with 15 compressions, until a defibrillator arrives. When cardiopulmonary resuscitation is performed by two persons, the sequence is similar, and the ratio of compressions to breaths remains 15:2. The person performing chest compression should pause during the ventilations, and each ventilation should take two seconds. After endotracheal intubation, the ratio should be five compressions to one ventilation, and there should be no pause in chest compressions for the ventilatory breath.

Rapid Defibrillation

Defibrillation is definitive therapy for ventricular fibrillation and pulseless ventricular tachycardia. The sooner defibrillation occurs, the higher the likelihood of resuscitation. When it is provided immediately after the onset of ventricular fibrillation, the success rate of defibrillation is extremely high.[26] In a recent study of patients with sudden cardiac arrest in gambling casinos, the rate of survival to hospital discharge was 74 percent for patients who received their first defibrillation within three minutes after collapse.[27] If a defibrillator is immediately at hand, its use takes precedence over cardiopulmonary resuscitation for patients with ventricular fibrillation or pulseless ventricular tachycardia. Cardiopulmonary resuscitation should be performed until the defibrillator arrives. When a monophasic wave-form defibrillator is used, the initial shock should be 200 J. If the arrhythmia persists, a second shock of 200 to 300 J should be given, followed by a third shock of 360 J if still necessary. All three shocks should be given in quick succession, with a pause only long enough to assess the rhythm on the monitor between shocks. After the third shock, cardiopulmonary resuscitation should be started or resumed. New biphasic, impedance-compensating defibrillation wave forms appear to work at least as effectively as standard monophasic damped sinusoidal wave forms, and they allow effective defibrillation at energy levels of approximately 150 J without escalation of the energy levels for subsequent shocks.[28-31]

Rapid Advanced Care

Definitive airway control (usually with endotracheal intubation), intravenous access, and pharmacologic therapy constitute advanced care. Frequently, cardiopulmonary resuscitation and defibrillation alone are not enough to sustain a perfusing rhythm, making advanced procedures necessary.

MANAGEMENT OF CARDIAC ARREST

The management of specific cardiac arrhythmias is based on recommendations of the American Heart Association[25] and the International Liaison Committee on Resuscitation.[32] These guidelines are subject to constant review and modification based on emerging scientific data. Like all guidelines, they must be modified according to individual circumstances. Readers interested in learning the skills of advanced cardiovascular life support should contact the American Heart Association at 1-800-242-8721 or at http://www.cpr-ecc.org.

Ventricular Fibrillation and Pulseless Ventricular Tachycardia

The treatment of ventricular fibrillation and pulseless ventricular tachycardia is outlined in Table 2. Medications that are useful in cardiac resuscitation are listed in Table 3.

Pulseless Electrical Activity

The prognosis for patients with pulseless electrical activity is grim, with generally reported rates of survival to hospital discharge of 1 to 4 percent. The best hope for a successful resuscitation involves finding and treating the cause of the pulseless electrical activity (Table 4). It is prudent to check for a pulse in more than one location, since a blocked carotid artery may lead to a false positive diagnosis of pulseless electrical activity. The management of pulseless electrical activity (Table 5), in addition to attempts to identify and treat its cause, includes cardiopulmonary resuscitation, endotracheal intubation, intravenous epinephrine every three to five minutes for as long as the patient is pulseless, and atropine if the rate of the rhythm on the cardiac monitor is too slow.

Asystole

The rate of survival for patients with asystole is near zero. Management of asystole (Table 5), in addi-

TABLE 2. MANAGEMENT OF VENTRICULAR FIBRILLATION AND PULSELESS VENTRICULAR TACHYCARDIA.*

1. Perform cardiopulmonary resuscitation until defibrillator arrives.
2. Shock using 200 J, 200–300 J, and then 360 J.†
3. Intubate the trachea and confirm that tube placement is correct; establish intravenous catheter access.
4. Administer epinephrine (1 mg by intravenous push, repeated every 3–5 min) or vasopressin (a single dose of 40 U intravenously).
5. Shock using 360 J (up to three times).
6. Administer antiarrhythmic drug or drugs‡
   - Lidocaine or amiodarone
   - Magnesium sulfate (if hypomagnesemia is suspected)
   - Procainamide (for recurrent or intermittent ventricular fibrillation or ventricular tachycardia).
7. Shock using 360 J (up to three times).
8. Simultaneously with the above, identify and treat potential causes.

*Management is based on American Heart Association guidelines.[25] The sequence of steps assumes continued ventricular fibrillation or pulseless ventricular tachycardia and ongoing cardiopulmonary resuscitation. Cardiopulmonary resuscitation should be interrupted only briefly as required for defibrillation or quickly performed procedures or interventions. Administration of medications should be followed by 30 to 60 seconds of cardiopulmonary resuscitation before giving shocks.

†Biphasic nonescalating shocks at lower energy (150–200 J) are equivalent.

‡Dosages are listed in Table 3.

tion to the identification and treatment of reversible causes (Table 4), should include confirmation of asystole through a check of the rhythm in more than one lead and with the gain maximized on the cardiac monitor. Immediate transcutaneous pacing may be tried, but its likelihood of success is extremely low.

Immediate Postresuscitation Care

Hypotension should be treated by the administration of fluids unless the patient has pulmonary edema, in which case dopamine should be started (with a target systolic blood pressure of 90 to 100 mm Hg). Appropriate analgesia and sedation should be considered for patients who are intubated. If the arrest rhythm was either ventricular fibrillation or ventricular tachycardia, parenteral administration of an antiarrhythmic medication (e.g., lidocaine or amiodarone) is usually started (or continued) in the immediate postresuscitation period to prevent a recurrence of the arrhythmia. If, during perfusion, the postarrest rhythm is idioventricular or third-degree heart block with an idioventricular escape rhythm, antiarrhythmic-drug treatment should not be started at this time, because it may eliminate the ventricular perfusing focus — with return of cardiac arrest. Finally, a meticulous search for the cause of the cardiac arrest, starting with the clinically relevant conditions listed in Table 4, should be performed.

COMMON ERRORS IN CARDIAC RESUSCITATION

Given the complexity and stress of a resuscitation, many errors are possible. These commonly relate to leadership and procedural skills. In hospital settings, especially teaching hospitals, there are frequently too many health professionals at the resuscitation scene, resulting in disorder and diffusion of responsibility. One person, generally a senior physician knowledgeable about cardiac resuscitation, should assume leadership and immediately assign appropriate resuscitation tasks to team members. The team leader should direct the resuscitation and make clinical decisions without directly performing specific procedures. It is appropriate to invite suggestions from team members, to ensure that all members are comfortable with a decision to stop the resuscitation attempt, and to debrief the team so that all may learn from the experience. Empathy and skill are also needed in compassionately informing the patient's family about the outcome.[33,34]

Procedural errors commonly involve airway management and use of defibrillators. Endotracheal intubation must be immediately confirmed and regularly reconfirmed during and after the resuscitation. Although endotracheal intubation is preferred for airway control, sometimes it cannot be easily accomplished. In such situations, adequate airway management can often be obtained with manual manipulation of the patient's jaw and use of a properly fitting face mask attached to a bag-valve device. Placement of a laryngeal mask airway, an easily learned skill, is an acceptable alternative when endotracheal intubation cannot be readily accomplished.[35]

Defibrillation will not succeed if electricity is not appropriately delivered to the arrested heart. Conducting gel or conducting pads should be used. If the patient has considerable chest hair, it should be shaved off where the pad or paddles are to be placed. If perspiration or conducting gel has been smeared across the chest during cardiopulmonary resuscitation, it should be removed with a towel before defibrillation.

Some defibrillation errors are a result of unfamiliarity with the machine. A common problem occurs when the synchronization mode has been accidentally selected before defibrillation is attempted. In synchronization mode, the machine will not deliver a shock if it does not detect a clear QRS signal, and this signal is not present in patients with ventricular fibrillation. Yet another error is inattention to the selection of leads on the defibrillator. For example, the operator may think that lead I, II, or III is being displayed, when in fact the selection is set to paddles, so that asystole is falsely shown.

CONTROVERSIES AND NEW DEVELOPMENTS

The current treatment for ventricular fibrillation or pulseless ventricular tachycardia is defibrillation administered as quickly as possible. A recent observational study questioned whether defibrillation should be provided as soon as possible for all cases of ventricular fibrillation. It is possible that in some cases (such as

TABLE 3. DRUGS USEFUL IN CARDIAC RESUSCITATION.*

DRUG	INDICATIONS	ADULT DOSAGE
Amiodarone	Ventricular fibrillation or pulseless ventricular tachycardia unresponsive to initial defibrillatory shocks and epinephrine Stable ventricular tachycardia Supraventricular tachyarrhythmias: rate control or conversion of atrial fibrillation or flutter (especially in patients with ejection fraction <40%, congestive heart failure, or preexcitation syndrome) and rate control of ectopic atrial tachycardia or multifocal atrial tachycardia (especially in patients with ejection fraction <40% or congestive heart failure)	Ventricular fibrillation or pulseless ventricular tachycardia: 300 mg diluted in 20–30 ml normal saline or 5% dextrose in water by rapid intravenous push; repeated doses of 150 mg may be given if required Stable ventricular tachycardia or supraventricular tachyarrhythmias: 150 mg intravenously over 10 min, followed by 1 mg/min infusion for 6 hr, then 0.5 mg/min maintenance. Administer another bolus of 150 mg intravenously over 10 min for breaththrough arrhythmia, if necessary Maximal dose should not exceed 2200 mg in 24 hr Acute side effects may include hypotension and bradycardia, which may necessitate changing infusion rate or additional corrective therapy
Atropine†	Symptomatic bradycardia Symptomatic heart block at nodal level or above Asystole Pulseless electrical activity (if rate of rhythm is slow)	For bradycardia or for supranodal or nodal heart block: 0.5–1.0 mg intravenously every 3–5 min, up to a total of 3 mg For asystole or pulseless electrical activity: 1 mg intravenously every 3–5 min, up to a total of 3 mg
Bicarbonate	Clinically significant metabolic acidosis unresponsive to optimal cardiopulmonary resuscitation, oxygenation, and ventilation Clinically significant hyperkalemia Overdoses of certain drugs, including tricyclic antidepressants and aspirin	Metabolic acidosis: 1 mmol/kg of body weight by slow intravenous push; may repeat half initial dose after 10 min Hyperkalemia therapy: 50 mmol intravenously over 5 min Use in overdose: discuss with toxicologist Acute side effects may include sodium overload, hypokalemia, and metabolic alkalosis
Dopamine	Shock secondary to a cardiac or vascular-resistance problem unresponsive to volume infusion or when volume infusion is contraindicated Postresuscitation hypotension unresponsive to volume infusion or when volume infusion is contraindicated Symptomatic bradycardia or heart block unresponsive to atropine or pacing	Start intravenous infusion at 2–5 μg/kg/min and titrate until desired effect is achieved (e.g., systolic blood pressure 95–100 mm Hg); dose range: 2–20 μg/kg/min
Epinephrine‡	Ventricular fibrillation or pulseless ventricular tachycardia unresponsive to initial defibrillatory shocks Pulseless electrical activity Asystole Anaphylaxis Hypotension unresponsive to volume infusion when vasopressor is needed Symptomatic bradycardia or heart block unresponsive to atropine or pacing	Cardiac arrest: 1 mg by intravenous push, may be repeated every 3–5 min Anaphylaxis: 0.3–0.5 mg intramuscularly or subcutaneously; may be repeated every 15–20 min if condition requires. If hypotension is present, 0.1 mg intravenously slowly over 5–10 min, followed by continuous infusion of 1–10 μg/min Vasopressor infusion: 1–10 μg/min intravenously, titrated until desired effect is achieved Symptomatic bradycardia or heart block not responsive to atropine or pacing: 1–10 μg/min intravenously, titrated until desired effect is achieved
Lidocaine§	Ventricular fibrillation or pulseless ventricular tachycardia unresponsive to initial defibrillatory shocks and epinephrine shock or shocks Stable ventricular tachycardia Frequent premature ventricular contractions compromising hemodynamic status	Initial dose: 1–1.5 mg/kg intravenously; for refractory ventricular fibrillation or unstable ventricular tachycardia, may repeat 1–1.5 mg/kg intravenously in 3–5 min; maximal dose is 3 mg/kg If lidocaine is effective, initiate continuous intravenous infusion at 2–4 mg/min when patient has return of a perfusing rhythm; for patients in stable condition with ventricular tachycardia or hemodynamically significant premature ventricular contractions: 0.5–0.75 mg/kg intravenously every 5–10 min, up to maximal dose of 3 mg/kg
Magnesium sulfate	Ventricular fibrillation or ventricular tachycardia if hypomagnesemic state is suspected Torsade de pointes	For life-threatening arrhythmia: administer 1–2 g diluted in 100 ml of 5% dextrose in water intravenously over 1–2 min
Norepinephrine	Severe hypotension (systolic blood pressure <70 mm Hg) accompanied by signs and symptoms of shock due to a vascular-resistance or cardiac problem when volume infusion is contraindicated or ineffectual	Initial dose: 0.5 to 1.0 μg/min intravenously, titrated until desired clinical effect is achieved; refractory shock may require doses of 8–30 μg/min

*Indications and dosages are based on American Heart Association guidelines.[25] All medications used during cardiac arrest, when given through a peripheral venous site in an arm or leg, should be followed by a 20-ml intravenous bolus of saline and elevation of the arm or leg for 10–20 seconds.

†Atropine can be given by the endotracheal route (2–3 mg diluted with normal saline to a total volume of 10 ml).

‡Epinephrine can be given by the endotracheal route (2–2.5 mg diluted with normal saline to a total volume of 10 ml).

§Lidocaine can be given by the endotracheal route (2–4 mg/kg diluted with normal saline to a total volume of 10 ml).

¶Vasopressin can be given by the endotracheal route (40 IU diluted with normal saline to a total volume of 10 ml).

TABLE 3. CONTINUED.

DRUG	INDICATIONS	ADULT DOSAGE
Procainamide	Intermittent or recurrent ventricular fibrillation or pulseless ventricular tachycardia not responsive to earlier interventions Monomorphic ventricular tachycardia (ejection fraction >40%, no congestive heart failure) Polymorphic ventricular tachycardia with normal base-line QT interval Wide complex tachycardia of unknown type (ejection fraction >40%, no congestive heart failure), patient in stable condition Refractory paroxysmal supraventricular tachycardia Atrial fibrillation or flutter (ejection fraction >40%, no congestive heart failure), including preexcitation syndrome	Intermittent or recurrent ventricular fibrillation or pulseless ventricular tachycardia: 20–30 mg/min intravenously (up to 50 mg/min if necessary) Other indications: 20 mg/min intravenously Maximal dose is 17 mg/kg (reduced to 12 mg/kg in setting of cardiac or renal dysfunction) Procainamide should be stopped when arrhythmia is suppressed, hypotension occurs, QRS interval widens to >50% of original duration, or maximal dose of 17 mg/kg is administered
Vasopressin¶	Pulseless ventricular tachycardia or ventricular fibrillation not responsive to initial defibrillatory shocks Possibly effective in pulseless electrical activity (insufficient data) Possibly effective in asystole (insufficient data)	A single dose of 40 IU by intravenous push If no response after 10 min of continued resuscitation, administer epinephrine

when the collapse is unwitnessed or there is a long interval before the defibrillator arrives at the scene), cardiopulmonary resuscitation should be briefly administered before defibrillation is attempted.[36] Future defibrillators may be able to interpret the form and amplitude of the electrocardiographic signal and recommend either immediate defibrillation or a period of cardiopulmonary resuscitation first. It is not difficult to envision biosensors that will guide therapy during resuscitation in the future. End-tidal carbon dioxide monitors are already being used and are recommended to indicate the adequacy of cardiopulmonary resuscitation and the likelihood of a successful resuscitation.[37] Low carbon dioxide values are due to inadequate or absent circulation.

There have been no placebo-controlled studies demonstrating a survival benefit from the use of lidocaine in the management of ventricular fibrillation. Nevertheless, lidocaine has been used for decades in this setting. In a recent study comparing amiodarone with placebo for patients with shock-refractory ventricular fibrillation before they reached the hospital, intravenous amiodarone improved the rate of survival to arrival at the hospital, but not survival to discharge from the hospital.[38] The optimal role and exact benefit of antiarrhythmic medications in cardiac resuscitation have yet to be fully elucidated. For now, lidocaine and amiodarone can be considered acceptable treatments for the management of ventricular fibrillation or pulseless ventricular tachycardia that is refractory to repeated shocks.

Hypoxic brain damage is a regrettable outcome in some resuscitations. Decades of research have failed to find a means to improve brain resuscitation significantly. Current research is exploring induced hypothermia[39,40] as well as pharmacologic intervention to reduce brain damage.

The precordial thump may generate a few joules, and therefore it has the potential to cause cardioversion if it is applied immediately in patients with pulseless ventricular tachycardia. For a monitored arrest, when the patient is pulseless and no defibrillator is immediately available, a precordial thump is a reasonable intervention. In practice, it seldom is of benefit, especially outside the hospital.

Recommendations to improve cardiopulmonary resuscitation range from the use of mechanical vests to devices that actively compress and decompress the chest (active compression–decompression resuscitation).[41,42] Randomized studies have shown equivocal benefit, and these new techniques have yet to receive widespread acceptance. Another technique, known as interposed abdominal compression cardiopulmonary resuscitation, which requires three rescuers, alternates chest compression with abdominal compression.[43,44] This technique appears to be equivalent or superior to standard cardiopulmonary resuscitation and is recommended as an alternative for professional rescuers.

Some investigators have questioned the value of mouth-to-mouth ventilation, postulating that chest compression alone may be sufficient to oxygenate the blood in the early phases of a cardiac arrest.[45] There are no data in humans to support such a recommendation. A recent observational study of patients with out-of-hospital cardiac arrest, however, showed similar rates of survival to hospital discharge between dispatcher-delivered instructions to lay rescuers with mouth-to-mouth ventilation and those without.[46]

Automated external defibrillators have been available for over a decade. They are being increasingly placed in airplanes,[47] airports, shopping malls, stadiums, casinos,[27] exercise facilities, office buildings, and other public locations. For example, Chicago's O'Hare airport has 33 automated external defibrilla-

TABLE 4. POTENTIALLY TREATABLE CONDITIONS ASSOCIATED WITH CARDIAC ARREST.

CONDITION	COMMON CLINICAL SETTINGS	CORRECTIVE ACTIONS
Acidosis	Preexisting acidosis, diabetes, diarrhea, drugs and toxins, prolonged resuscitation, renal disease, and shock	Reassess adequacy of cardiopulmonary resuscitation, oxygenation, and ventilation; reconfirm endotracheal-tube placement Hyperventilate Consider intravenous bicarbonate if pH <7.20 after above actions have been taken
Cardiac tamponade	Hemorrhagic diathesis, cancer, pericarditis, trauma, after cardiac surgery, and after myocardial infarction	Administer fluids; obtain bedside echocardiogram, if available Perform pericardiocentesis. Immediate surgical intervention is appropriate if pericardiocentesis is unhelpful but cardiac tamponade is known or highly suspected
Hypothermia	Alcohol abuse, burns, central nervous system disease, debilitated or elderly patient, drowning, drugs and toxins, endocrine disease, history of exposure, homelessness, extensive skin disease, spinal cord disease, and trauma	If hypothermia is severe (temperature <30°C), limit initial shocks for ventricular fibrillation or pulseless ventricular tachycardia to three; initiate active internal rewarming and cardiopulmonary support. Hold further resuscitation medications or shocks until core temperature is >30°C If hypothermia is moderate (temperature 30–34°C), proceed with resuscitation (space medications at intervals greater than usual), passively rewarm, and actively rewarm truncal body areas
Hypovolemia, hemorrhage, anemia	Major burns, diabetes, gastrointestinal losses, hemorrhage, hemorrhagic diathesis, cancer, pregnancy, shock, and trauma	Administer fluids Transfuse packed red cells if hemorrhage or profound anemia is present Thoracotomy is appropriate when a patient has cardiac arrest from penetrating trauma and a cardiac rhythm and the duration of cardiopulmonary resuscitation before thoracotomy is <10 min
Hypoxia	Consider in all patients with cardiac arrest	Reassess technical quality of cardiopulmonary resuscitation, oxygenation, and ventilation; reconfirm endotracheal-tube placement
Hypomagnesemia	Alcohol abuse, burns, diabetic ketoacidosis, severe diarrhea, diuretics, and drugs (e.g., cisplatin, cyclosporine, pentamidine)	Administer 1–2 g magnesium sulfate intravenously over 2 min (see Table 3)
Myocardial infarction	Consider in all patients with cardiac arrest, especially those with a history of coronary artery disease or prearrest acute coronary syndrome	Consider definitive care (e.g., thrombolytic therapy, cardiac catheterization or coronary-artery reperfusion, circulatory-assist device, emergency cardiopulmonary bypass)
Poisoning	Alcohol abuse, bizarre or puzzling behavioral or metabolic presentation, classic toxicologic syndrome, occupational or industrial exposure, and psychiatric disease	Consult toxicologist for emergency advice on resuscitation and definitive care, including appropriate antidote Prolonged resuscitation efforts may be appropriate; immediate cardiopulmonary bypass should be considered, if available
Hyperkalemia	Metabolic acidosis, excessive administration of potassium, drugs and toxins, vigorous exercise, hemolysis, renal disease, rhabdomyolysis, tumor lysis syndrome, and clinically significant tissue injury	If hyperkalemia is identified or strongly suspected, treat with all of the following: 10% calcium chloride (5–10 ml by slow intravenous push; do not use if hyperkalemia is secondary to digitalis poisoning), glucose and insulin (50 ml of 50% dextrose in water and 10 units of regular insulin intravenously), sodium bicarbonate (50 mmol intravenously; most effective if concomitant metabolic acidosis is present), and albuterol (15–20 mg nebulized or 0.5 mg by intravenous infusion)
Hypokalemia	Alcohol abuse, diabetes, use of diuretics, drugs and toxins, profound gastrointestinal losses, hypomagnesemia	If profound hypokalemia (<2–2.5 mmol of potassium per liter) is accompanied by cardiac arrest, initiate urgent intravenous replacement (2 mmol/min intravenously for 10–15 mmol), then reassess
Pulmonary embolism	Hospitalized patient, recent surgical procedure, peripartum, known risk factors for venous thromboembolism, history of venous thromboembolism, or prearrest presentation consistent with diagnosis of acute pulmonary embolism	Administer fluids; augment with vasopressors as necessary Confirm diagnosis, if possible; consider immediate cardiopulmonary bypass to maintain patient's viability Consider definitive care (e.g., thrombolytic therapy, embolectomy by interventional radiology or surgery)
Tension pneumothorax	Placement of central catheter, mechanical ventilation, pulmonary disease (including asthma, chronic obstructive pulmonary disease, and necrotizing pneumonia), thoracentesis, and trauma	Needle decompression, followed by chest-tube insertion

tors mounted on walls throughout the facility, in a manner not unlike that for fire extinguishers. An automated external defibrillator automatically interprets the cardiac rhythm and, if ventricular fibrillation is present, advises the operator to provide a shock. The devices are highly sensitive and specific.[48-50] The increase in the number of automated external defibrillators parallels efforts by the American Heart Association and other national organizations to encourage public-access defibrillation.[51]

Automated external defibrillators are small (the size of a notebook laptop computer) and easy to use (instruction takes minutes) and have batteries that last five years. The cost of an automated external defibrillator is $2,500 to $3,000, and future devices are likely to be less expensive. Most states have passed legislation limiting liability for those prescribing or using automated external defibrillators.[52]

Because most cardiac arrests occur in the home, an argument can be made for home defibrillators for patients at high risk. Automated external defibrillators currently must be prescribed by a physician. It is conceivable that in the next decade they will be sold over the counter as consumer devices. Growing aware-

TABLE 5. MANAGEMENT OF PULSELESS ELECTRICAL ACTIVITY AND ASYSTOLE.*

PULSELESS ELECTRICAL ACTIVITY	ASYSTOLE
1. Perform cardiopulmonary resuscitation.	1. Perform cardiopulmonary resuscitation.
2. Intubate the trachea and confirm placement; establish intravenous catheter access.	2. Intubate the trachea and confirm placement; establish intravenous catheter access.
3. Administer epinephrine (1 mg by intravenous push every 3–5 min).	3. Institute immediate transcutaneous pacing, if available.
4. Administer atropine (1 mg by intravenous push if heart rate is too slow, repeated every 3–5 min if necessary, up to a total of 3 mg).	4. Administer epinephrine (1 mg by intravenous push every 3–5 min).
5. Simultaneously with the above, identify and treat potential causes.	5. Administer atropine (1 mg by intravenous push every 3–5 min, up to a total of 3 mg).
	6. Simultaneously with the above, identify and treat potential causes.
	7. Consider termination of resuscitation if confirmed asystole lasts more than 10 min and no treatable condition exists.

*Management is based on American Heart Association guidelines.[25]

ness will probably prompt patients to ask their doctors about the potential benefit of automated external defibrillators. They are not currently paid for by medical insurance. Patients at the highest risk for ventricular fibrillation should have implanted cardioverter–defibrillators.[53] The role of home automated external defibrillators is not yet known.[54,55]

WHEN TO STOP

The decision to stop cardiac resuscitation is difficult. Prolonging resuscitation efforts beyond 30 minutes without a return of spontaneous circulation is usually futile, unless the cardiac arrest is compounded by hypothermia, submersion in cold water, drug overdose, other identified and treatable conditions, or intermittent ventricular fibrillation or ventricular tachycardia.[56,57] It is reasonable to stop resuscitation after a patient has been in asystole for more than 10 minutes if there is no readily identified and reversible cause. In the hospital, patients with unwitnessed arrest, rhythms other than ventricular fibrillation or ventricular tachycardia, and no pulse after 10 minutes of resuscitation do not survive.[58] In the community setting (given that proper equipment and medications are available), full resuscitation should be attempted at the scene of a nontraumatic cardiac arrest in preference to rapid transportation to an emergency department.

WHEN NOT TO START

The phrase "hearts too good to die" was coined by Claude Beck, one of the inventors of defibrillation. It best describes persons with structurally good hearts who are struck down in the prime of their lives by fatal arrhythmias.[59] If cardiac resuscitation is successful, patients can continue to lead productive lives. Most survivors of sudden cardiac arrest have good functional outcomes.[60] Today, we expect a resuscitation effort to avert sudden and unexpected death. But what about expected death? The sudden death of a person who is not hospitalized precludes knowing that person's wishes regarding resuscitation, unless they were previously stated. The emergency-medical-services system has only one response when called to the scene of a cardiac arrest — namely, resuscitate, regardless of the medical history or the wishes of relatives (unless there are obvious signs of death).

The power of modern technology and pharmacologic therapy can sometimes bring persons back to life with "hearts too bad to live,"[61] leading to continued suffering in the hospital and prolongation of the process of dying. The best way to prevent such a tragedy is to address the issue before it occurs. Many patients, particularly those with terminal diseases, sincerely do not wish to be resuscitated, and options in the event of a sudden collapse should be discussed. These patients should have a signed do-not-attempt-resuscitation document on the premises. Relatives and friends also need to know that they should not call 911 under these circumstances. In many states, emergency-medical-services crews can honor these orders when proper documentation (for example, a bracelet indicating such an order) is present.

The powerful tools and techniques of cardiac resuscitation can benefit thousands of people in the community and hospital setting. Conversely, when inappropriately applied, cardiac resuscitation may prolong human suffering. Ideally, the patient's wishes about resuscitation should be made known beforehand. What is needed, of course, at all times are judgment and balance. Death is, after all, inevitable. Only unexpected or sudden death is the enemy.

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