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Manual of Emergency Medicine

Third Edition

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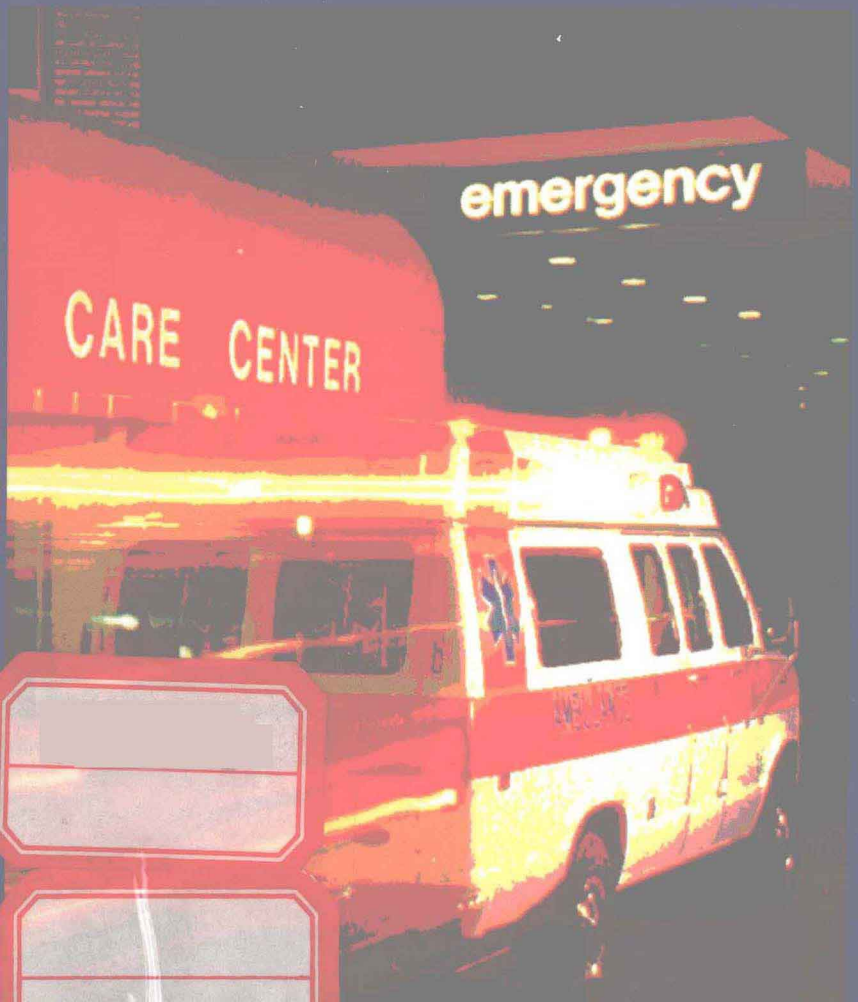

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Manual of Emergency Medicine

To Cathy, Anita, and Kate
for their patience and
understanding and to Anna Maye

Preface

The practice of emergency medicine represents a unique combination of the essential aspects of acute care from many of the various subspecialties. The extensive body of knowledge and technical expertise that the emergency physician must master to maintain adequate skills have expanded over the years to such a degree that continuous study is essential, both at the training level and thereafter.

This text has been written as a guide in the practice of this increasingly complex specialty. Many chapters begin with a differential diagnosis of the complaint or symptom in question; this is followed by a discussion of the various historical, physical, and laboratory features that may allow the physician to arrive at an appropriate diagnosis. This general section, which will be of most use when a specific diagnosis is not yet clear, is followed by a detailed discussion of the individual disorders included in the differential diagnosis, the focus of which is toward rapid confirmation of the diagnosis and the institution of specific treatment. The very nature of a text written in this format dictates the need for extensive cross-referencing. Consequently, we have reproduced in several chapters the descriptions of common disorders that may be associated with more than a single presenting symptom. Frequently, less common disorders with more than one possible presenting symptom are simply cross-referenced by page number.

The intent of the authors is that this type of format will make the text practical, rapidly referable, and sufficiently informative for physicians involved in the practice of emergency medicine at any level.

J. L. J.
J. L.
G. R. B.

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Contents

Cardiopulmonary Arrest

Notice. The indications and dosages of all drugs in this book have been recommended in the medical literature and conform to the practices of the general medical community. The medications described do not necessarily have specific approval by the Food and Drug Administration for use in the diseases and dosages for which they are recommended. The package insert for each drug should be consulted for use and dosage as approved by the FDA. Because standards for usage change, it is advisable to keep abreast of revised recommendations, particularly those concerning new drugs.

1 Cardiopulmonary Resuscitation

Techniques, strategies, and guidelines for CPR have developed over the years into a rational system of care for any patient who presents pulseless or apneic. In the emergency department, basic CPR must proceed simultaneously with advanced resuscitation using both medications and electrotherapy.

I. **Basic CPR** focuses on the "ABCs," ensuring first that the airway is patent and adequate; second, that breathing is effective and results in appropriate air exchange; and third, that the circulation is restored. These three aspects of basic CPR are prioritized in this manner because the establishment of a functional airway must be the initial and primary concern.

A. **Airway maintenance.** In the obtunded or unconscious patient, the upper airway may become obstructed owing to relaxation of muscle groups in the upper respiratory tract. Should upper airway obstruction by a foreign body be suspected, the airway should be cleared either manually or, if unsuccessful, by the Heimlich maneuver (performed by applying anterior chest thrusts to the lower abdomen with the patient in a supine position).

1. **When respiratory effort exists,** airway patency can often be obtained by a variety of simple mechanical maneuvers that involve the mouth, chin, and mandible. When injury to the cervical spine is not present, simply tilting the head backward may be dramatically effective in opening the airway, and if so, signs of respiratory obstruction, such as stridor, may disappear. In some patients, the insertion of an oral or nasal airway, provided that the former does not result in gagging or vomiting, followed by bag-mask–assisted ventilation as required, may provide adequate oxygenation while the physician attends to other aspects of CPR. In other patients with respiratory effort, the jaw thrust (which involves placing the fingers bilaterally behind the mandibular angles and displacing the mandible forward or anteriorly) or the chin lift may provide complete control of the upper airway. The jaw thrust, which results in little or no movement of the neck, is the preferred initial maneuver in patients with possible injury to the cervical spine. In all patients, supplemental oxygen should be administered.

   Despite respiratory effort by the patient, the use of supplemental oxygen, and the application of techniques to open the airway, patients with inadequate oxygenation urgently require establishment of a patent airway. Endotracheal intubation is the preferred maneuver unless the following relative contraindications exist: potential injury to the cervical spine, mechanical upper airway obstruction, severe restriction of cervical spine mobility, severe perioral trauma, or an inability to open the patient's mouth, e.g., during seizure or associated with teeth clenching. In many such cases, nasotracheal intubation remains a valuable technique that may safely be used in the presence of contraindications to endotracheal intubation. In the patient with potential cervical spine injury, insertion of a nasotracheal tube while the cervical spine remains immobilized is both safe and effective. Nasotracheal intubation should be avoided in patients with significant maxillofacial trauma, since intracranial penetration along fracture lines may occur. Because of a variety of

factors, in some patients it may not be possible to obtain an airway by endotracheal or nasotracheal intubation. In these patients, bag-mask–assisted ventilation using an oral or nasal airway (during which time the adequacy of oxygenation should be ensured by serial arterial blood gas analysis), cricothyrotomy, and tracheostomy remain additional options.

2. **In patients without respiratory effort,** immediate intervention is required to establish an airway and provide oxygenation. Begin with bag-mask–assisted ventilation and 100% supplemental oxygen with the assistance of an oral or nasal airway. When possible, evaluate oxygenation with pulse oximetry. Endotracheal intubation is then indicated, with cricothyrotomy or tracheostomy considered an alternative for the patient who can be neither oxygenated nor endotracheally intubated.

B. Breathing. Once airway patency is established, patients without adequate spontaneous respiratory effort require artificial ventilation; mouth-to-mouth, mouth-to-nose, mouth-to-stoma, and mouth-to-airway techniques have all been used with variable success. In one-rescuer CPR, two initial breaths of 1–1½ seconds each should be performed, after which two breaths should be administered after every 15 compressions (at a rate of 80–100 compressions/min). In two-rescuer CPR, one ventilation should be provided for every five chest compressions; compressions should occur at a rate of approximately 80–100/min. When available, an Ambu-bag used with an oral or nasopharyngeal airway and supplemental oxygen (100% FIO_2) are preferred to mouth-to-mouth ventilation and are more effective. Effective, sustained bag-mask–assisted ventilation is also preferable to the interrupted ventilation that can occur during multiple failed attempts at endotracheal intubation. The adequacy of ventilation is assessed by determining that breath sounds are present bilaterally, that an inspiratory increase in chest volume occurs with each inspiration, that skin color improves, and that arterial blood gases or pulse oximetry reflect appropriate oxygenation.

C. Circulation. Chest compressions should begin simultaneously with the establishment of an airway and the initiation of ventilation. A precordial thump is occasionally effective in initiating heart action, particularly for a witnessed cardiac arrest where the patient is pulseless and a defibrillator is not immediately available. A precordial thump may convert 11–25% of cases of ventricular tachycardia (VT), but it may also cause VT to deteriorate to ventricular fibrillation or asystole. (The American Heart Association feels that the precordial thump is an advanced life support technique that should not be used by the lay public.) With the patient placed in a supine position on a hard surface, external cardiac compressions are initiated by placing the heel of one hand over the lower half of the sternum and the heel of the second hand on top of the first. Pressure over the xiphoid process should be avoided. With the elbows extended, rhythmic compressions should be provided by depressing the sternum 1.5–2.0 inches posteriorly in adults. Current evidence suggests that changes in intrathoracic pressure related to compression result in or enhance cardiac output; actual mechanical ventricular compression is no longer felt to be the primary mechanism of effective chest compressions. New techniques based on these principles that are currently under study to enhance blood flow during CPR include simultaneous chest compression and ventilation, abdominal compression with synchronized ventilation, CPR augmented with military antishock trousers (MAST), interposed abdominal compressions, and continuous abdominal binding. Compressions should be smooth and should be performed at the rate of approximately 80–100/min. The efficacy of external compressions can be checked by palpating the carotid or femoral pulse after four cycles of compressions and ventilations (15 : 2 ratio); an impulse should be noted at these sites with each compression to demonstrate the hemodynamic adequacy of resuscitation. As suggested, ventilations should be timed to occur between compressions at a rate of one ventilation for every five compressions in two-rescuer CPR.

II. **Advanced cardiopulmonary resuscitation.** Immediately after arrival in the emergency department, or as soon as possible if in the field, the patient's underlying rhythm should be determined. This is most easily and rapidly accomplished via defibrillator paddles that sense underlying cardiac activity when applied to the anterior chest; external electrodes are also useful but require somewhat more time to apply. When ventricular fibrillation is identified, immediate **defibrillation** using 200 joules should be employed, which if unsuccessful should immediately be followed by defibrillations using 300 and then 360 joules. If an effective rhythm is not restored, or if asystole is the initial or subsequent rhythm, basic CPR should proceed, intravenous access should be obtained, and **intubation** undertaken (if the patient has not been intubated) using endotracheal or nasotracheal techniques. In contrast to the "blind" nature of nasotracheal intubation, safe endotracheal intubation requires visualization of the vocal cords and arytenoids before actual intubation. Pressure applied by an assistant to the cricoid will help to visualize these structures as well as protect against regurgitation of gastric contents. If possible, particularly in profoundly hypotensive patients, Doppler ultrasound should be used to assess pulses and blood flow.

A. **Intravenous access.** Initial venous access should be established in a peripheral vein (e.g., using veins in the antecubital fossa, generally the most accessible peripheral veins). Central venous sites are initially best avoided because of the increased time associated with their placement and the unavoidable interruption of CPR; hand and wrist sites are also less useful, as is femoral venous catheterization (unless a long catheter can be passed above the diaphragm). One must remember that 1–2 minutes is required for medications administered at a peripheral site to reach the heart; this is true even when CPR is adequate. Most authorities therefore recommend that a central line be started if, after the initial administration of pharmacologic agents, the circulation remains unrestored. In the intubated patient, the internal jugular approach results in less interference with chest compressions. When venous access is unobtainable, several medications can be given by endotracheal tube: epinephrine, atropine, and lidocaine, which are administered in the usual doses. Bicarbonate cannot be given via an endotracheal tube because of potential adverse effects on the lung.

B. **Additional recommendations.** In the past, the use of **sodium bicarbonate** was encouraged to treat acidosis associated with cardiac arrest. Recently, the use of sodium bicarbonate has been discouraged in routine CPR. The rationale for this change involves the lack of evidence supporting the use of this alkali in changing the outcome of routine CPR as well as a number of factors suggesting a negative effect. For example, bicarbonate (1) does not facilitate defibrillation or improve survival in experimental animals in ventricular fibrillation; (2) shifts the oxyhemoglobin saturation curve to the left, inhibiting the release of oxygen to the tissues; (3) produces a paradoxical acidosis in cells, which results from the ability of carbon dioxide, released from sodium bicarbonate, to diffuse freely into cells, depressing cellular function; (4) may inactivate administered catecholamines; and (5) induces a number of other adverse effects owing to systemic alkalosis produced from overvigorous administration. Bicarbonate is therefore not recommended in routine CPR. In certain specific circumstances, bicarbonate may be of use, but only when the diagnosis on which such therapy is based has been clearly defined. For example, patients with pronounced systemic acidosis associated with renal failure or patients with hyperkalemia, both documented immediately prior to arrest, may benefit from the prompt administration of bicarbonate. The blind, routine administration of bicarbonate should, however, be avoided. Prior protocols also recommended the use of **calcium** in asystolic arrests and those associated with electromechanical dissociation; new protocols stress the very limited role of calcium in routine CPR. Specifically, calcium should only be used in arrests associated with hyperkalemia, hypocalcemia, or calcium channel blocker toxicity.

C. Treatment of rhythm disturbances

1. **Ventricular fibrillation**, once diagnosed, should be treated with immediate defibrillation using 200 joules, which, if unsuccessful, should be followed by defibrillations using 200–300 and then 360 joules. Defibrillations should be repeated immediately if unsuccessful (so-called back-to-back discharges), thereby taking advantage of an increasing decrement in chest wall impedance afforded by three rapid, repeated discharges. If ventricular fibrillation persists, epinephrine (10 ml of a 1 : 10,000 solution or 1 mg) should be administered, either intravenously or, if venous access has not been obtained, by endotracheal tube. Defibrillation should then be repeated using maximal energies (360 joules) within 30–60 seconds.

   If these maneuvers fail, lidocaine (1 mg/kg) should be administered intravenously over 1–2 minutes, after which defibrillation may be repeated. If unsuccessful, bretylium (5 mg/kg), given intravenously over 1–2 minutes, should be administered, followed by electrical defibrillation. The dose may be doubled and repeated in 5 minutes and electrical defibrillation repeated as well if necessary. Magnesium sulfate (1–2 g IV) may be useful in torsade de pointes or in suspected hypomagnesemia. Procainamide (30 mg/min IV to maximum total of 17 mg/min) may also be considered in refractory cases. If these maneuvers fail to restore a favorable rhythm, epinephrine should be repeated at 5-minute intervals.
2. **Ventricular tachycardia** generally requires the least energy of all arrhythmias for cardioversion. In the **conscious patient** with minimal hemodynamic compromise, the treatment of choice is lidocaine, which should be administered intravenously as 1.0 mg/kg over 1–2 minutes, following which a continuous infusion at 1–4 mg/min may be initiated if chemical cardioversion has occurred. In the **unconscious patient** or the patient with impaired hemodynamics, a precordial thump may be used initially if a defibrillator is not available (providing 1–2 joules), although its success is limited (as discussed previously) and if unsuccessful should be followed by electrical cardioversion using 10–100 joules. If successful, lidocaine should then be administered (1.0 mg/kg loading dose followed by a 1–4 mg/min continuous infusion) to maintain normal sinus rhythm (NSR). If unsuccessful, procainamide may be administered and electrical cardioversion repeated as necessary. Procainamide is a second-line ventricular antiarrhythmic that may be given intravenously in a dosage of 20 mg/min or 100 mg at 5-minute intervals until the dysrhythmia is suppressed, the QRS complex widens by more than 50%, or a total of 1 g has been administered. Loading doses should be followed by continuous infusions of 1–4 mg/min once cardioversion has restored NSR. Bretylium may also be considered in patients with ventricular tachycardia refractory to lidocaine or procainamide.
3. **Asystole** may represent any one of three possible electrophysiologic events: extremely fine ventricular fibrillation, pronounced bradycardia (supraventricular, junctional, or idioventricular), or true asystole. Therapy is predicated on one's inability to distinguish among these three etiologies using the scalar ECG. At least two leads should be briefly analyzed before the diagnosis of asystole is made. Hence, epinephrine (1 mg), followed by atropine (1 mg), should be employed; if unsuccessful, external or transvenous pacing may be useful, and the use of bicarbonate should be considered. Atropine should be repeated once, at 5 minutes, and epinephrine every 5 minutes. When fine ventricular fibrillation is a definite possibility (given antecedent rhythms or transient responses to epinephrine), electrical defibrillation may be attempted.

   Major causes for this dysrhythmia should be considered, including hypoxia, hyperkalemia, hypokalemia, preexisting acidosis, drug overdose, and hypothermia. After treatment of the asystole and correctable causes has failed, terminate the resuscitation. As quoted from the ACLS guide-

lines of the American Heart Association, "asystole most often represents a confirmation of death rather than a rhythm to be treated."

4. **Bradycardia.** Bradycardias may be sinus, ectopic atrial, junctional, or idioventricular in origin. When associated with hypotension, clinical or ECG evidence of ischemia, or congestive heart failure, treatment should be initiated with atropine (0.5–1.0 mg IV, repeated as needed up to 2.0 mg), followed by external or transvenous pacing, then dopamine, 5–20 μg/kg/min, or epinephrine, 2–10 μg/min.

   Once a more rapid sinus or junctional rhythm is obtained (i.e., > 60 beats/min), the presence of a pulse should be sought. If present, blood pressure should be determined; if low, volume repletion and pressors (dopamine, 2–50 μg/kg/min, or norepinephrine, 16–24 μg/min) may be instituted.

5. **Electromechanical dissociation.** In this disorder, there is ECG evidence of organized electrical activity but failure of effective myocardial contraction (absent pulses and heart sounds). Causes include hypoxemia, tension pneumothorax, massive pulmonary embolus, pericardial tamponade, hypovolemia, hypothermia, drug overdoses (including beta-blockers, calcium channel blockers, digitalis and tricyclics, antidepressants), hyperkalemia, acidosis, ventricular rupture, or massive myocardial infarction. Treatment includes oxygenation, volume repletion, epinephrine (1 mg IV push q3–5 min), atropine (1 mg IV, q3–5 min up to a total of 0.04 mg/kg), and consideration of bicarbonate therapy, pericardiocentesis, and needle aspiration of the chest.

6. **Supraventricular tachycardias.** Although supraventricular tachycardias may produce cardiopulmonary arrest, they are rarely sustained; ventricular tachycardia and fibrillation usually evolve with continued hypotension. Thus, these dysrhythmias are noted most commonly **after** successful cardioversion or defibrillation. Occasionally, patients may be seen immediately before an arrest precipitated by a hemodynamically intolerable supraventricular tachycardia; they must be immediately electrically cardioverted.

   a. **Carotid sinus pressure** (CSP) is a useful diagnostic (and occasionally therapeutic) maneuver for distinguishing among supraventricular mechanisms. In response to CSP, sinus tachycardia (rates of 100–180 beats/min) may gradually slow or not change, whereas paroxysmal supraventricular tachycardia (PSVT), or paroxysmal atrial tachycardia (PAT), or reentrant nodal tachycardia (rates of 140–220 beats/min) will abruptly terminate or fail to change. Atrial flutter (atrial rates of 260–340 beats/min usually with 2 : 1 atrioventricular [AV] block) will also either not change or will block down in a stepwise fashion, returning to the original rate once the effect of CSP wanes. The response of tachyarrhythmias involving accessory bypass tracts is variable. Atrial fibrillation with a rapid ventricular response and multifocal atrial tachycardia (MAT) also respond inconsistently to CSP.

   b. **Adenosine** (6 mg rapid IV push over 1–3 seconds, followed in 1–2 minutes by 12 mg rapid IV push over 1–3 seconds, repeated in 1–2 minutes) is the drug of choice for a hemodynamically stable PSVT or PAT. It is safer than verapamil because it produces less hypotension and has a very short half-life.

   c. **Verapamil** (5 mg IV, repeated twice if necessary) after adenosine has been tried is a useful adjunct in the management of some supraventricular tachycardias: PSVT or PAT will typically terminate abruptly; 5–10% of patients in atrial flutter will revert to sinus rhythm; MAT may also slow significantly. Verapamil is, however, contraindicated in patients with accessory bypass tracts, given the unpredictable response of these patients to this agent. If the patient becomes symp-