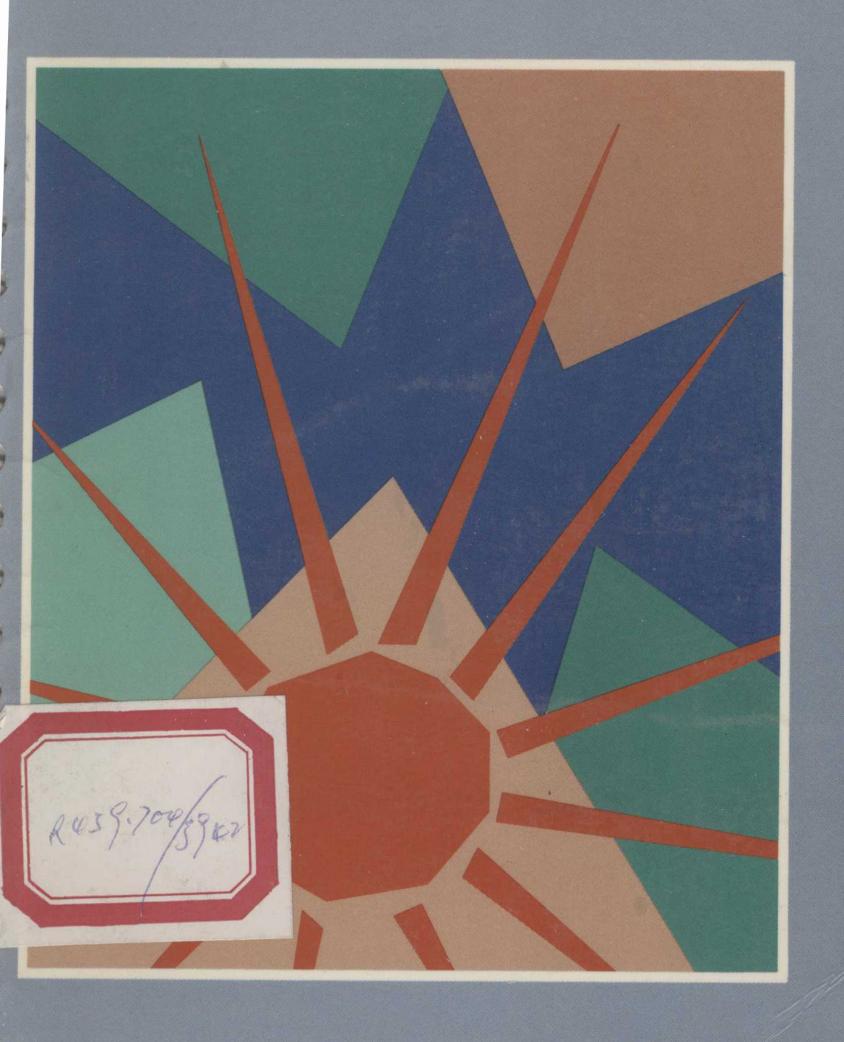


Manual of Emergency Medicine

Diagnosis and Treatment

Jon L. Jenkins, M.D. Joseph Loscalzo, M.D.



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First Edition Second Printing July 1987 Third Printing December 1987

Fourth Printing April 1988 Fifth Printing July 1989

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Library of Congress Catalog Card No. 85-81924

ISBN 0-316-46052-4

Printed in the United States of America

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146-4



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

Acknowledgments

We would like to acknowledge the contribution of Dr. Bruce E. Shannon to the chapters involving Radiation Exposure, Poisoning, High Altitude Illness, Sexually Transmitted Diseases, Thermoregulation, and Electrical Injuries. We would also like to thank Dr. Thomas Mulvaney for his review of selected otolaryngologic material.

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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 that the emergency physician must master to maintain adequate skills has 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; it is divided into three sections-medical disorders, trauma, and special problems in emergency medicine. The medical section is presented in a chief complaint format and begins with a differential diagnosis of the 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 crossreferencing. 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.

Contents

Assessment of the			
TOP IS A RESIDENCE OF THE	Preface	ix	
	Acknowledgments	xi	
the anthropy tend later	Medical Disorders		
1	Cardiopulmonary Resuscitation	on 3	
2 amubida ata pi an	Abdominal Pain	9	
3	Back Pain	29	
4	Change in Bowel Habits	37	
5	Chest Pain	47	
6	Coma and Other Disorders of Consciousness	55	
7	Cough	69	
8	Dizziness	77	
9	Ear Pain	81	
10	Extremity Pain—Atraumatic	Extremity Pain—Atraumatic 89	
11	Eye—The Acutely Red Eye	107	
12	Facial Pain—Atraumatic	115	
13 mg same in a market in the	Fever, Heat Illness, and Cold Exposure	그리는 그 그는 그 그리는 그리는 그는	
14	Gastrointestinal Bleeding	127	
15	Headache	135	
16	Hematuria 80	143	
17	Hemoptysis	145	
18	Hiccups (Singultus)	149	
19	Hoarseness	151	
20	Hypertension	153	
21	Jaundice	157	
22	Joint Pain—Atraumatic	163	
23	Nausea and Vomiting	171	
24	Palpitations	175	
25	Rash	185	
26	Sexually Acquired Disorders	199	
27	Shortness of Breath	205	

28	Sore Throat	213
29	Testicular, Scrotal, and Inguinal Pain or Swelling	219
30	Vaginal Bleeding	223
31	Visual Disturbances	227
32	Weakness	233
	II Trauma	
33	Initial Assessment of the Multiply Traumatized Patient	245
34	Trauma to the Head, Neck, and Face	253
35	Trauma to the Eye and Periorbital Area	265
36	Trauma to the Chest	271
37	Trauma to the Abdomen	285
38	Trauma to the Genitourinary Tract	289
39	Trauma to the Pelvis	291
40	General Principles of Extremity Trauma	293
41	Abscesses	321
42	Abrasions, Avulsions, Lacerations, and Puncture Wounds	323
43	Burns	337
44	Epistaxis	341
45	Foreign Bodies	343
	III Special Problems in Emergency Medicine and Trauma	
46	Bites and Stings	353
47	Child Abuse	363
48	Childbirth and Emergency Delivery	367
49	Diving Accidents	371
50	Electrical Injuries	373
51	High Altitude Illness	377
52	Near Drowning	381
53	Poisoning and Ingestions	385
54	Radiation Illness and Exposure	437
55	Sexual Assault	447
56	Smoke Inhalation	453
57	Suicide	455
	Index	457



Medical Disorders

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.



Cardiopulmonary Resuscitation

The techniques and strategies of cardiopulmonary resuscitation (CPR) have developed over the years into a rational system of approach to any patient who presents pulseless or apneic. Resuscitation must proceed simultaneously at two levels, basic and advanced, with the latter using both pharmacologic and electrotherapies. The experienced emergency physician must be skilled in the techniques and strategies of both basic and advanced cardiac life support.

I. Basic cardiopulmonary resuscitation. Basic cardiopulmonary resuscitation focuses on the "ABCs," ensuring first that the airway is patent and adequate, second, that breathing is effective and results in appropriate air exchange within the chest, 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 for the physician.

A. Airway maintenance. In the obtunded or unconscious patient, the upper airway may readily become obstructed due to relaxation of muscle groups in the upper respiratory tract. 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 backwards 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 simply 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. When these simple maneuvers do not provide adequate oxygenation, endotracheal or nasotracheal intubation (while the neck remains immobilized if necessary) or cricothyrotomy are immediately indicated. 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 patients without respiratory effort, immediate intervention is clearly required to obtain an airway and provide oxygenation. When contraindications to endotracheal intubation do not exist, this maneuver is preferred. A number of relative contraindications to endotracheal intubation exist; these include 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. In many such cases, nasotracheal intubation remains a valuable technique. Although this maneuver shares many of the complications of endotracheal intubation, it nonetheless avoids some of the contraindications previously mentioned. In the patient with potential cervical spine injury for example, insertion of a nasotracheal tube while the cervical spine remains immobilized is both safe and effective. Nasotracheal intubation should not be attempted in patients with significant maxillofacial trauma, since intracranial penetration along fracture lines has

been clearly documented. As in the case of patients with respiratory effort, bagmask-assisted ventilation using an oral or nasal airway, cricothyrotomy, or

tracheostomy remain additional options.

B. Breathing. Once airway patency is established, patients without adequate spontaneous respiratory effort require artificial ventilation; mouth-to-mouth, mouth-to-nose, and mouth-to-airway techniques have all been used with variable success. In general, ventilations should be provided for every five chest compressions; compressions should occur at a rate of approximately 70 per minute. When available, an Ambu-bag used with an oral or nasopharyngeal airway and supplemental oxygen are preferred to mouth-to-mouth ventilation and are more effective. Effective, sustained bag-mask—assisted ventilation is also clearly 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 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 and restoring normal blood pressure and may thereby obviate the need for other interventions. 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 approximately 2 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. Compressions should be smooth and should be performed at the rate of approximately 70 per minute. The efficacy of external compressions can be checked by palpating carotid or femoral pulse; an impulse should be noted at these sites with each compression to show the hemodynamic adequacy of resuscitation. As suggested above, ventilations should be timed to occur between compressions at a rate of 1/5 compressions.
- 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 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, intubation should be undertaken (if the patient has not been intubated), and intravenous access should be obtained.
 - A. Intravenous access. It is currently recommended that initial venous access be established in the antecubital fossa, and we agree with this recommendation. 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 appropriate, 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.

- B. New 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 buffers 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 freely diffuse into cells, depressing cellular function (such as myocardial and cerebral); (4) may inactivate administered catecholamines; and (5) induces a number of other adverse effects owing to systemic alkalosis. Bicarbonate is therefore not recommended in routine CPR. 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. In specific, calcium should only be used in arrests associated with hyperkalemia, hypocalcemia, or calcium channel blocker toxicity.
- C. Treatment of rhythm disturbances
- 1. Ventricular fibrillation and tachycardia. Ventricular fibrillation may be either coarse or fine. Once diagnosed, however, regardless of type, defibrillation should be immediately attempted using 200 joules, which, if unsuccessful, should be followed by defibrillations using 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 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 (360–400 joules).

If these maneuvers fail, lidocaine (1 mg/kg) should be administered intravenously over 1 to 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. Bretylium has been said to be the only drug that may spontaneously defibrillate an individual without the use of electricity. The dose may be doubled and repeated and electrical defibrillation repeated as well if necessary. If these maneuvers fail to restore a favorable rhythm, epinephrine should be repeated at 5-minute intervals and use of bicarbonate should be considered. Bretylium may be

repeated every 15 minutes as needed.

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 to 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 (providing 1-2 joules), and if unsuccessful should be followed by electrical cardioversion using 10 to 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, bretylium may be administered as for ventricular fibrillation and electrical cardioversion repeated as necessary. Procainamide is a second-line ventricular antiarrhythmic that may be given in a dosage of 20 mg/min until the dysrhythmia is suppressed, the QRS complex widens by more than 50 percent, or a total of 1 gm has been administered. Boluses should be followed by continuous infusions of bretylium, 1 to 2 mg/min, or procainamide, 1 to 4 mg/min, once cardioversion has restored 2. Asystole. 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. 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.

3. **Bradycardia.** Bradycardias may be sinus, ectopic atrial, junctional, or idioventricular in origin; these may all be treated initially with atropine (0.5–1.0 mg IV, repeated as needed up to 2.0 mg), followed by isoproterenol (2–10

μg/min), and/or external or transvenous pacing.

Once a more rapid sinus or junctional rhythm is obtained (i.e., greater than 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.

4. 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, ventricular rupture, or massive myocardial infarction. Treatment includes epinephrine, oxygenation, volume repletion, and consideration of bicarbonate

therapy, pericardiocentesis, and needle aspiration of the chest.

5. 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. 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 PAT) or reentrant nodal tachycardia (rates of 140-220 beats/min) will abruptly terminate or not change. Atrial flutter (atrial rates of 260–340 beats/min usually with 2: 1 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.

Verapamil (5 mg, IV, repeated twice if necessary) is a useful adjunct in the management of some supraventricular tachycardias: PSVT or PAT will typically terminate abruptly; 5 to 10 percent 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 un-

predictable response of these patients to this agent.

Broad-complex tachycardias present a special problem in diagnosis and management. These arrhythmias may represent either ventricular tachycardia or a supraventricular tachycardia conducted aberrantly; distinguishing between these two mechanisms is essential for appropriate termination of the arrhythmia. When hemodynamic stability is present, a full 12-lead ECG is most useful in differentiating these two rhythms. Table 1-1 lists the electrocardiographic features that may differentiate ventricular from su-

Table 1-1. Electrocardiographic features differentiating ventricular tachycardia from supraventricular tachycardia with aberrancy

Si Minda sur ni encare de	VT	SVT with aberrancy
QRS width	Often > 0.14 sec	Usually < 0.14 sec
Axis	Bizarre	Normal
V_1	Rs, RsR', Rsr'	rsR'
V_6	S wave present	S wave absent
Fusion beats	Present	Absent
AV dissociation	Present	Absent

VT = ventricular tachycardia; SVT = supraventricular tachycardia.

praventricular tachycardia with aberrancy. It is important to note that the presence of a consistent relationship between P waves and QRS complexes does *not* exclude ventricular tachycardia. In some patients with ventricular tachycardia, the atria may be activated in a retrograde fashion producing a 1:1 relationship; in other patients, isorhythmic but independent depolarizations of the atria and ventricles occur and may artifactually appear as if the former were conducted.

Treatment depends on the appropriate diagnosis and in all cases, if hemodynamic instability or myocardial ischemia exists, electrical cardioversion must be used promptly regardless of the specific etiology. For all supraventrical tachyarrythmias (except atrial flutter, which may be cardioverted with as little as 20 joules) relatively high energies are required (100–200 joules). Overdrive pacing (with rates up to 800 per sec) may be required in extreme cases of PSVT or refractory atrial flutter.

III. Pediatric cardiopulmonary resuscitation. Resuscitation of the infant or child involves the same basic principles of airway management, circulatory assistance, and pharmacologic interventions discussed above with respect to the adult, with a

few specific differences.

A. Airway and breathing. Since children less than 6 months of age are obligate nose breathers, obstruction of the nose, from whatever cause, can rapidly result in respiratory failure; because of this, nasogastric tube placement is generally deferred in the acute setting. In addition, the anatomy of the upper airway differs in the small child; the oral cavity is small, the tongue is relatively large, the larynx is anterior, and the trachea is short. These anatomic differences must be appreciated if the delivery of oxygen and intubation are to be successful and entry into the bronchi prevented. In addition, during the administration of oxygen and in preparation for intubation, hyperextension of the neck, particularly in the infant, is to be avoided because the anatomic topography of the oropharynx is such that optimal airway patency is achieved with the neck held in the neutral position. With respect to intubation, for neonates and children less than 2 years of age the position of sniffing is most appropriate while for older children extension of the head will be helpful. Obstruction of the airway (e.g., secondary to foreign body, epiglottitis) remains an important cause of respiratory arrest in children and must be considered in all patients. Mouth-to-mouth, mouth-to-nose, and bag-mask ventilation of the infant and child, when successful, are associated with appropriate thoracic motion and auscultatory evidence of air exchange; these techniques may provide adequate oxygenation for prolonged periods of time and are preferable to multiple failed attempts at intubation during which time oxygenation is interrupted. Ventilations should never provide excessive tidal volumes; "cheek" breathing may be sufficient for the resuscitation of children less than 2 years of age.

In children 5 to 7 years old, teeth may easily be avulsed and enter the trachea; insertion of an oral airway, if necessary to improve oxygenation, should not be