CARDIAC ARREST AND CPR

Assessment, Planning, and Intervention
Second Edition

Paul S. Auerbach, MD
Susan A. Budassi, RN, MSN, CEN
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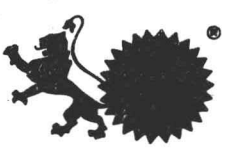

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Preface

Cardiopulmonary resuscitation is a charged medical-political issue that evokes strong biases, highly publicized and grandiose research, and community scrutiny that is unique to the practice of medicine. In another view, it is the challenge to the intervening rescuer, which must evoke automatic and accurate responses.

Properly regarded, the practice of medicine is a team effort in which the team members share the goals of health and preservation of the patient. In no area is teamwork more important than in cardiopulmonary resuscitation. At all skill levels, cooperation is the keynote of successful resuscitation. As with any other disaster, cardiopulmonary arrest must be approached enthusiastically and in an organized fashion.

In this second edition of *Cardiac Arrest and CPR*, we have attempted to address the major aspects of resuscitation in a way that will be of clinical use to the reader. The evolution of prehospital care and layperson awareness has focused public attention on the management of cardiac arrest and trauma. It is crucial therefore that medical professionals and paraprofessionals be able to perform crisis intervention and to justify their actions.

In this book on CPR, the approaches are open minded. Because of the nature of the topic, there is some overlap between chapters; however, each chapter can stand alone. Our intent is to provide the reader with both a comprehensive review and in-depth discussions of specific subjects. As it was with the first edition, it is our hope that reader response will help us to improve this text and to anticipate new trends in cardiopulmonary resuscitation.

Forthcoming developments are difficult to predict, as the cycles of simplicity and complexity in medicine are related to rhetoric, cost consciousness, and scientific research. We do anticipate, however, a more straightforward and expeditious approach to prehospital care, maximizing the successes that have followed crisp resuscitations and transport times. In addition, we can predict a global effort at the hospital level of care to validate the efficacy of cerebral resuscitation and great ethical discussion about the intensive care unit responsibilities it will incur. Some of the old standards, such as the use of calcium chloride, megawatt countershock, and precordial thump, will most likely disappear; the use of calcium channel antagonists, increased intrathoracic pressure, barbiturate coma, and endotracheal administration of medications will probably gain new support. It is imperative that the new data be clean and that we examine the data honestly. Only time will tell if any of these measures will improve survival statistics.

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Editors
November 1982

Acknowledgments

We would like to acknowledge the monumental accomplishments of the American Heart Association and the American Red Cross in training the lay public, as well as paraprofessional and professional personnel, in both basic and advanced cardiac life support techniques. We would also like to acknowledge the lay people who have had the courage to "get involved" and have unselfishly performed "bystander CPR." A very special mention of the remarkable prehospital care personnel of the United States, many of whom are not paid for their services; their dedicated efforts on behalf of the people of their communities have made survival following cardiopulmonary arrest more of a reality.

We praise our colleagues, who have provided straightforward and current recommendations for assessment, planning, and intervention in the setting of cardiopulmonary arrest.

We wish our readers success with their rescues and hope that this textbook has made the effort somewhat easier.

Contents

1. Pathophysiology of Cardiac Arrest

HERBERT J. ROGOVE, DO

Equating cardiac arrest solely with ischemic heart disease invariably leads to oversight and failure in cardiopulmonary resuscitation (CPR). Although it is true that most cardiac arrests are secondary to ischemic heart disease, the experienced resuscitation leader should be able to draw upon and apply a basic knowledge of the pathophysiology of cardiac arrest in treating patients in cardiac arrest of any etiology. Three basic events are predominant in the evolution of cardiac arrest: (1) hypoxia and anoxia, (2) neurologic dysfunction, and (3) metabolic and chemical abnormalities. Two or more of these mechanisms often work in concert to produce ventricular fibrillation or asystole.

HYPOXIA AND ANOXIA

Whether the pathogenesis of cardiac arrest is asphyxia, pulmonary embolism, or carbon monoxide poisoning, it is hypoxia that ultimately leads to cardiopulmonary arrest. The term *hypoxia* indicates that the oxygen content or oxygen tension of arterial blood is diminished, while the term *anoxia* usually connotes total depletion of oxygen throughout the body. At this time, the body's highly efficient cellular metabolism begins to fail. Mitochondrial damage ensues, and the driving force of the cell becomes inoperative. Failure to correct this problem invariably leads to death.

Classification

Approximately 60 years ago, anoxia was classified into four major divisions: (1) anoxic, (2) anemic, (3) stagnant, and (4) histotoxic.[1] When anoxia occurs, as in asphyxia or drowning, oxygen is denied access to the intravascular spaces. Anemic anoxia results when the amount of blood available to transport oxygen to tissues is insufficient. In stagnant anoxia, typified by the shock state, circulation of the blood to tissues is reduced. Histotoxic anoxia is synonymous with cell poisoning; although oxygen is available, it is not used by the tissues. The most common examples of histotoxic anoxia are cyanide and carbon monoxide poisoning, hypoglycemia, and uremia.

Response to Hypoxia and Anoxia

The heart responds to hypoxia by increasing its output. Attempts are made to increase peripheral vascular resistance even though the circulatory system may already be maximally vasodilated. If anoxia is not corrected immediately, ectopic cardiac rhythms may result. In addition, coronary blood flow may decrease, creating a potential for acute myocardial infarction, arrhythmias, or sudden death. In the presence of anoxia, stimulation of the vagus nerve may be a major factor in the etiology of bradycardia, leading to asystole.[1] Anoxia alone induces the release of catecholamines, and excessive amounts

of catecholamines lower the threshold for ventricular fibrillation.

Simultaneously with the cardiac response to hypoxia, the pulmonary blood flow, pulmonary vascular resistance, and pulmonary artery pressures increase. Other organs and tissues fail to function when oxygen saturation falls below 50%. In this setting, although the cerebral blood vessels dilate as much as possible, the brain cells are unable to extract oxygen from blood, especially when capillary oxygen pressure levels are below 15 to 20 mm Hg. Also, hepatic cells cannot survive when arterial oxygen content is less than 9 vol%. These are but some of the many physiologic responses to hypoxemia.[2]

Structural Damage

In addition to the physiologic reactions to oxygen deficiency, subendocardial hemorrhagic necrosis may occur, often resulting in low cardiac output with conduction abnormalities. Representative anoxic damage involves three major areas: the myocardial cell, the coronary microvasculature, and the cardiac conduction system.[3]

The Myocardial Cell

Myocardial cells require oxygen for energy production and survival. The energy elicited from the cardiac mitochondria supports the sodium-potassium "pump" of the sarcoplasmic reticulum, both of which help to maintain the heart's intrinsic rhythm and contractility. When the oxygen supply is diminished, energy requirements of the heart may not be fulfilled. Following the onset of severe hypoxia, for example, the amount of oxygen in reserve is adequate for less than five minutes. There is a threefold increase in oxygen consumption during fibrillation as compared with asystole. Oxygen consumption in the arrested fibrillating heart may approach 30% of that of the normal working heart.

During anaerobic metabolism, glycogen becomes one of the primary substrates used. Unfortunately, however, myocardial anaerobic glycolysis produces only enough glycogen to last for four minutes.[3] One of the more interesting of the therapeutic interventions that have been proposed for anoxic cardiac arrest involves the use of glycogen as an energy substrate during anoxia.[4] In one animal study, glycogen was administered to several dogs during induced anoxic arrest. The results indicated significant protection and preservation of cardiac function during the arrest. Further studies are required before this can be considered for human clinical application, however.

Hypoxia, hypercapnia, and acidemia stimulate the release of catecholamines prior to cardiac arrest. With increased levels of circulating catecholamines, myocardial oxygen consumption increases, the rate of glycolysis increases, and the automaticity of the His-Purkinje fibers is enhanced. Combined, these factors create an extremely precarious situation for the heart that needs to conserve rather than consume energy.

Ischemia may cause myocardial cell damage and electrolyte shifts, which in turn may precipitate arrhythmias and alter cellular metabolism and mitochondrial function. Hypercapnia and elevated lactic acid levels can alter the myocardial cellular pH, diminishing cardiac contractility.

The Coronary Microvasculature

Microscopically, an early myocardial lesion of anoxia is seen as an alteration of the capillary membrane.[5] In the human adult, there is close to a one-to-one ratio of capillaries to myocardial fibers; therefore any damage to a capillary affects an entire contractile unit. Fragmentation and swelling of myocardial fibers distort the myocardial ultrastructure; consequently, there is a decrease in the work capacity of the heart, which corresponds with a decrease in the adenosine triphosphate (ATP) concentration.[6] When anoxia becomes irreversible,

mitochondria may disappear. The loss of lysosomal membrane integrity causes a release of hydrolytic enzymes, which further damages the myocardial ultrastructure.[3]

A decrease in systemic blood flow and an increase in capillary permeability accompany anoxia. Plasma protein is lost from the intravascular compartment, decreasing volume and thus contributing to red cell sludging. This initiates a cascade of events, eg, red cell agglutination, platelet adhesion to endothelial walls, and release of adenosine diphosphate (ADP). The result is vascular endothelial damage.

When the vascular cell membrane becomes permeable, uncontained vasoactive substances and acid metabolites further damage the capillary endothelium. The anoxic heart muscle releases adenosine, potassium, phosphate, and lactate, which contribute to vasodilation and increase vascular permeability.[5]

The Cardiac Conduction System

Anoxia may alter the normal sequence of depolarization and encourage irregular ventricular contractions and arrhythmias. A large number of Purkinje's fibers are located in the left ventricular subendocardium. If this vulnerable area suffers hemorrhagic necrosis, cardiac output may be low.

Metabolic Influence of Anoxia

A comprehensive study of the metabolic influences of asphyxia and anoxia demonstrated the remarkable similarity of the cardiac and respiratory responses.[7] In asphyxia, hypercapnia progressively paralyzed the catecholamine-induced myocardial contraction; however, death was attributed more directly to hypoxia and acidosis, which synergistically produced hypotension. Despite this particular synergism, it was demonstrated that each acted discretely in the pathophysiology of cardiac failure.[7]

In anoxia, the progression of the arres was rapid, there was a high incidence ot myocardial irritability (ventricular fibrillation), and autopsy showed the heart to be firmly contracted. Arrests associated with respiratory acidemia progressed less rapidly and had a lower associated incidence of ventricular irritability; the heart appeared dilated and relaxed at autopsy. The therapeutic implications of such data are unclear.

Depletion of oxygen inhibits the respiratory drive prior to total cardiac decompensation. Therefore respiratory arrest (present within two to six minutes) precedes circulatory collapse (present within five to ten minutes). Although experimental animal hearts undergo significant structural damage during arrest, the lungs of these same animals appear grossly normal at autopsy. The only evidence of pulmonary injury is mild dependent congestion. This supports the notion that respiratory arrest is centrally mediated.

An appreciation of the pathophysiology of anoxia makes it easier to understand how certain conditions may ultimately result in cardiac arrest and death. At all times, the clinician must consider anaphylaxis, exsanguination, asphyxia, electrocution, lightning injuries, tension pneumothorax, pulmonary embolism, carbon monoxide poisoning, near-drowning, and seizures.[8-10]

NEUROLOGIC MECHANISMS

The Vagus Nerve

Increased vagal tone not only may propagate a sinus bradycardia, but also may depress atrioventricular conduction, causing atrial and ventricular slowing or cardiac standstill. Because visceral structures, particularly gastrointestinal and respiratory organs, contain vagal nerve fibers, procedures such as endotracheal intubation, bronchoscopy, colonoscopy, and esophagogastroscopy may precipitate arrhythmias or even a full arrest.[11] Anoxia may further

contribute to the lethality of vagal stimulation, because it may be highly vagomimetic. Hypoxia and hypercapnia can enhance vagal stimulation. Estimates in one series of 1,200 cardiac arrests suggested that 25% were mediated in some form by the vagus nerve.[12]

Certain common clinical procedures are associated with vagal stimulation and the risk of a cardiopulmonary arrest, eg, abdominal surgery involving traction on the gallbladder, displacement of the stomach, mobilization of the omentum, and distention of the common bile duct.[11] Diagnostic procedures such as gastroscopy, the administration of a barium enema, or even a routine ventipuncture also carry these risks.[11,13-15] Patients who undergo eye surgery may experience vagus-related cardiac arrest.[12] With increased external ocular pressure, an afferent oculocardiac reflex that involves both the vagus and trigeminal nerve may cause bradycardia, premature ventricular contractions, nodal escape beats, or ventricular tachycardia. The premedication of patients to relieve anxiety has probably diminished the risk of these procedures, particularly in patients with underlying cardiovascular disease.

Jaundiced patients may experience episodes of bradycardia, probably because of increased vagal tone secondary to the deposition of bile salts in body tissue. Elderly patients who have a sensitive carotid sinus may experience episodes of ventricular fibrillation or asystole, apparently because of an indirect link between the carotid sinus reflex and the vagus nerve. A similar mechanism has been postulated in patients with glossopharyngeal neuralgia who experience cardiac syncope. In these situations, the ninth cranial nerve has an indirect connection to the vagus.[16] Subarachnoid hemorrhage may cause cardiac arrest through several mechanisms, one of which is stimulation of area 13 of the brain, which has the major cortical influence upon the vagus nerve.[12]

Cases of cardiac arrest related to excessive vagal discharge have been reported in patients with quadriplegia and in patients treated with electroconvulsive shock therapy; for the most part, the mechanisms are quite similar to those involved in the other vagus-induced arrests.[11,17] Clearly, it is of crucial therapeutic significance to anticipate overstimulation of the vagus nerve, particularly in patients who are unable to compensate with the appropriate cardiovascular response.

Autonomic Abnormalities

A neurologic basis for cardiac arrest is seen in patients whose autonomic nervous system cannot accommodate the stress of illness, such as a diabetic with an associated autonomic neuropathy.[18] These patients have inadequate respiratory responses to afferent impulses from the carotid body and carotid arch. Diabetics are often further jeopardized by concurrent cardiovascular disease.

In contrast to those with autonomic hypofunction, some patients have an excessive amount of autonomic activity. In tetanus infections, for example, cardiac standstill may occur, partly because of toxin-induced sympathetic overactivity.[19]

Sudden Death

Psychologic as well as physiologic imbalance may cause sudden death.[20] There appears to be a close correlation between the mechanisms of vasovagal syncope and sudden death. In the face of emotional despair, a patient may experience vasodepressor syncope. The hemodynamic response to vasodepression involves two phases. Initially, the heart rate, blood pressure, peripheral vascular resistance, and cardiac output increase. In the second phase, all of these parameters reverse, resulting in bradycardia with frequent sinus arrest and escape rhythms. In addition to the vagal influence, a corticospinal response is reputed to be intimately involved.

There have been numerous examples of patients under extreme stress who have

experienced syncope or sudden death. Simply arriving at an emergency department has been related to dramatic, life-threatening arrhythmias.[20] A decreased arrhythmia threshold is associated with stress. For example, when they are helpless, animals exposed to noxious stimuli developed lethal arrhythmias.[20] The animal response varied from a sympathetic system-induced tachycardia to a parasympathetic system-mediated bradycardia. This rapid fluctuation of sympathetic and parasympathetic discharge may contribute to electrical instability that provides the milieu for ominous arrhythmias.

Although the psychophysiologic input into sudden death is significant, organic heart disease remains the predominant factor in sudden cardiac arrest. Most of the 400,000 to 600,000 annual sudden deaths have been attributed to ventricular arrhythmias.[21,22] In a group of patients resuscitated from sudden death, angiographic studies revealed that 94% had significant (greater than 70%) occlusion of one or more coronary vessels. Measured ventricular contraction abnormalities suggested that extensive scarring contributed to ventricular fibrillation.[23]

Recently, attention has been focused on the long QT interval syndrome. In the idiopathic type, the patient is usually asymptomatic until a syncopal episode reveals a prolonged QT interval.[19] The acquired form is usually secondary to drugs, electrolyte imbalance, hypothermia, cerebrovascular disease, or neck surgery. The suspected mechanism is an imbalance of cardiac sympathetic innervation, a suspicion supported by the fact that a prolonged QT interval is produced in animals when the right stellate ganglion is blocked. Sudden death associated with a prolonged QT interval has also been attributed to liquid protein diets.[24–27]

METABOLIC AND CHEMICAL ABNORMALITIES

Alterations of the overall metabolic state may have direct cardiac effects that predispose to cardiopulmonary arrest. In general, metabolic acidemia decreases the threshold for ventricular fibrillation.[28] In dogs, the ventricular fibrillation threshold was not influenced by pH changes of respiratory origin[28]; thus hyperventilation of an acidemic dog did not diminish the risk of ventricular fibrillation. Changes of P_{CO_2} are indeed detrimental, however. Hypercapnia associated with both a lower pH and an elevated potassium level may greatly sensitize the myocardium to the effects of catecholamines.[29]

Fluxes or absolute deficiencies or excesses of electrolytes, such as potassium, calcium, and magnesium, may alter myocardial excitability and precipitate arrhythmias. Patients with progressive hyperkalemia may develop delayed ventricular conduction, arrhythmias, and cardiac standstill.[29] Excessive calcium, particularly in concert with digitalis, may stimulate arrhythmias and induce sudden death. Excessive magnesium, a major intracellular ion, may cause intraventricular conduction delays and arrest the heart in diastole.

Hypothermia may induce cardiac arrest. In fact, much of the understanding of anoxic and hypothermic arrest has been derived from observing patients whose hearts were intentionally arrested during cardiac surgery. Hypothermia may directly affect the myocardium, causing ventricular fibrillation and asystole.[30,31]

Chemical mechanisms of cardiac arrest are numerous and involve primarily anesthesia and medications. In one study of unexpected cardiac arrests in healthy patients undergoing elective surgery (1/3,400 operations), the causes included anesthetic mismanagement, hypoxia secondary to hypoventilation, malignant hyperpyrexia, severe bronchospasm, and air emboli.[32] The more common medications associated with cardiac arrests are aminophylline, aminoglycosides, penicillin, chlorpromazine, propranolol, potassium supplements, diazepam, and lidocaine.[33,34] An increased vigilance for arrhythmias is necessary when these agents are used.

EFFECTS OF PROLONGED ARREST

The longer it takes to restore normal perfusion after cardiac arrest, the more likely that the patient will suffer such complications as acute tubular necrosis of the kidney, bowel infarction, or hypoxic encephalopathy. While hemodialysis and timely surgical removal of devitalized bowel are treatments for the first two complications, it is only recently that clinically applicable treatments have been developed for cerebral resuscitation.

The changes in cerebral perfusion and histologic damage to the brain during and following cardiac arrest have been carefully documented in many animal studies. The most commonly suggested mechanisms of the hypoxic encephalopathy that accompanies cardiac arrest are neuronal vulnerability to oxygen deprivation and elevation of lactic acid levels as a consequence of ischemia.[35] When the patient's body temperature is normal, irreversible ischemia usually occurs eight to ten minutes after the onset of cardiac arrest.[35] Within two to three minutes, red blood cells begin to sludge; within five to ten minutes, microemboli form and obstruct the cerebral vasculature in a scattered distribution. In animals, the affected areas include, in order, the brain stem, basal ganglia and thalamus, and the cortex.[36] Interestingly, fibrin clots or platelet thrombi are not preeminent, making the administration of heparin as yet an inappropriate therapy.

Following cerebral ischemia, cerebral perfusion is diminished as a result of mechanical obstruction of the edematous endothelial cells, potassium-induced elevation of cerebrovascular resistance, and slightly increased intracranial pressures.[37] These are the most likely explanations for what has been called the "no-reflow phenomenon." The basis for post–cardiac arrest cerebral insufficiency appears to be the brain's low tolerance for anoxia. Further cerebral insult apparently results from a combination of depleted glucose reserves and extreme loss of cerebral energy metabolism.[38] World-wide studies are needed to determine whether treatment, such as with barbiturates or calcium channel antagonists, will protect and promote recovery of the ischemic brain.

THE FINAL MECHANISM

Whatever the cause, the finale in the pathophysiology of cardiac arrest is the cessation of cardiac muscle contraction. Usually, it requires 20 to 30 minutes for electrical activity to cease completely, although it requires only 10 minutes for brain cell death.[8] The two final arrhythmias in the termination of cardiovascular activity are ventricular fibrillation and asystole.

Ventricular fibrillation has been described as an uncoordinated, disorderly and extremely bizarre contractile process.[39,40] When it occurs, the intraventricular pressure is not sufficient to eject blood. Without adequate myocardial contraction, perfusion pressures cannot satisfy the needed blood flow to the brain and other vital organs.

The vast majority of deaths related to ventricular fibrillation are complications of a myocardial infarction. One widely accepted theory of fibrillation, the reentry theory, is that a single impulse is propagated in a circus movement. A second explanation is that repetitive discharges originate from a single ectopic focus.[39,40] Because of the increasing awareness of the importance of metabolic and biochemical factors that increase cardiac automaticity and thus the potential for fibrillation,[41] drugs that alter the fibrillation threshold have been developed.

Asystole may be classified as either primary or secondary.[8] Primary asystole is a sudden and overwhelming event associated with myocardial depressant drugs, sustained high-energy shock, and advanced atrioventricular block. Secondary asystole may be the consequence of vagal stimulation, exsanguination, or asphyxia. Within

ten seconds of asystole, either primary or secondary, the patient is unconscious. This is followed immediately by apnea and, in 60 seconds, by pupillary dilatation.

CONCLUSION

The medical literature and anecdotal physician experiences provide an enormous data base on cardiopulmonary arrest. There are protean studies and case reports on the pathogenesis of cardiac arrest; what is necessary now is a search for common ground based on the pathophysiology of cardiac arrest so that a reasonable approach to resuscitation can be taken. Whether the patient is in full arrest from near-drowning, electrocution, or ischemic heart disease, all physiologic parameters must be rapidly corrected. If both brain and heart are to be saved, resuscitators must advance beyond arrhythmia management and employ maneuvers that demonstrate an understanding of the pathophysiology of cardiopulmonary and cerebral arrest.

REFERENCES

1. Lucas BGB: Role of anoxia and hypoxia in etiology of cardiac arrest, in Stephenson HE (ed): *Cardiac Arrest & Resuscitation,* ed 4. St. Louis, CV Mosby Co, 1974, pp 166–174.

2. Sykes MK, McNicol MW, Campbell LJM: Causes, time course and effects of respiratory failure, *Respiratory Failure,* ed 2. London, Blackwell Scientific Publications, 1976, pp 95–111.

3. Iyengar SRK, et al: Myocardial protection during anoxic cardiac arrest in aortocoronary bypass surgery, in Norman JC (ed): *Coronary Artery Medicine and Surgery: Concepts & Controversies.* New York, Appleton-Century-Crofts, 1975, pp 495–512.

4. Hewitt RL, et al: Protective effect of glycogen and glucose on the anoxic arrested heart. *Surgery* 1974; 75:1–10.

5. Iyengar SRK, et al: An experimental study of subendocardial hemorrhagic necrosis after anoxic cardiac arrest. *Ann Thorac Surg* 1972;13:214–224.

6. Stephenson HE: Effect of anoxia on myocardial contractility, in Stephenson HE (ed): *Cardiac Arrest & Resuscitation,* ed 4. St Louis, CV Mosby Co, 1974, pp 192–193.

7. Kristoffersen MB. Rattenborg CC, Holaday DA: Asphyxial death: The roles of acute anoxia, hypercarbia and acidosis. *Anesthesiology* 1967;28:488–497.

8. Safar P: The mechanisms of dying and their reversal, in Schwartz GR (ed): *Principles and Practice of Emergency Medicine.* Philadelphia, WB Saunders Co, 1978, pp 17–50.

9. Stephenson HE: Toxic response and cardiac arrest, in Stephenson HE (ed): *Cardiac Arrest & Resuscitation,* ed 4. St Louis, CV Mosby Co, 1974, pp 207–214.

10. Kirimli B, Kampschulte S, Safar P: Pattern of dying from exsanguinating hemorrhage in dogs. *J Trauma* 1970;10:393–404.

11. Stephenson HE: Pathophysiology of cardiac arrest, in Stephenson HE (ed): *Cardiac Arrest & Resuscitation,* ed 4. St Louis, CV Mosby Co, 1974, pp 141–165.

12. Stephenson HE: Yes, Virginia there is a vasovagal reflex. *Chest* 1973;64:3–5.

13. Eastwood GL: ECG abnormalities associated with the barium enema. *JAMA* 1972;219:719–721.

14. Levy N, Abinader E: Continuous electrocardiographic monitoring with Holter electrocardiocorder through all stages of gastroscopy. *Am J Dig Dis* 1977;22:1091–1096.

15. Tizes R: Cardiac arrest following routine venipuncture. *JAMA* 1976;236:1846–1847.

16. Jamshidi A, Masroor MA: Glossopharyngeal neuralgia with cardiac syncope. *Arch Intern Med* 1976;137:843–845.

17. Frankel HL, Mathias CJ, Spalding JMK: Mechanism of reflex cardiac arrest in tetraplegic patients. *Lancet* 1975;2:1183–1185.

18. Page MMcB, Watkins PJ: Cardiorespiratory arrest and diabetic autonomic neuropathy. *Lancet* 1978;1:14–16.

19. Moss AJ, Schwartz PJ: Sudden death and the idiopathic long QT syndrome. *Am J Med* 1979;66:6–7.

20. Engle GL: Psychologic stress, vasodepressor (vasovagal) syncope, and sudden death. *Ann Intern Med* 1978;89:403–412.

21. Levitt B, et al: Ventricular rhythm disorders and sudden cardiac death. *Am J Med* 1976;61:449–450.

22. Greene HL, Redi PR, Schaeffer AH: The repetitive ventricular response in man—A predictor of sudden death. *N Engl J Med* 1978;299:729–734.

23. Lawry GV: Ventricular premature beats and sudden death. *N Engl J Med* 1978;298:512.

24. Brown JM, et al: Cardiac complications of protein-sparing modified fasting. *JAMA* 1978;240:120–122.

25. Michiel RR, et al: Sudden death in a patient on a liquid protein diet. *N Engl J Med* 1978;298:1005–1007.

26. Felig P: Four questions about protein diets. *N Engl J Med* 1978;298:1025–1026.

27. Hastings DH: Letters to the editor. *N Engl J Med* 1978;299:420.

28. Gerst PH, Fleming WH, Malm JR: Increased susceptibility of the heart to ventricular fibrillation during metabolic acidosis. *Circ Res* 1966;19:63–70.

29. McDonald HL: Acid-base and electrolyte imbalance, in Stephenson HE (ed): *Cardiac Arrest & Resuscitation*, ed 4. St Louis, CV Mosby Co, 1974, pp 194–206.

30. Bristow G, et al: Resuscitation from cardiopulmonary arrest during accidental hypothermia due to exhaustion and exposure. *Can Med Assoc J* 1977; 117:247–249.

31. Reuler JB: Hypothermia: Pathophysiology, clinical settings and management. *Ann Intern Med* 1978; 89:519–527.

32. Taylor G, Larson CP, Prestwich R: Unexpected cardiac arrest during anesthesia and surgery. *JAMA* 1976;236:2758–2760.

33. Cheng TO, Wadhwa K: Sinus standstill following intravenous lidocaine administration. *JAMA* 1973;223:790–792.

34. Camarata SJ, et al: Cardiac arrest in the critically ill. *Circulation* 1971;44:688–695.

35. Lin SR, et al: Effect of cardiac arrest on cerebral circulation. *Acta Radiol Suppl* 1976;347:149–165.

36. Fisher EG: Impaired perfusion following cerebrovascular stasis. *Arch Neurol* 1973;29:361–366.

37. Tweed WA, Wade JG, Pandson WJ: Mechanisms of the "low flow" state during resuscitation of the totally ischemic brain. *Can J Neurol Sci* 1977;4:19–23.

38. Hossman V, Hossman KA: Return of neuronal function after prolonged cardiac arrest. *Brain Res* 1973;60:423–438.

39. Granefield PF: Ventricular fibrillation. *N Engl J Med* 1973;289:732–736.

40. Stephenson HE: Ventricular fibrillation, in Stephenson HE (ed): *Cardiac Arrest & Resuscitation*, ed 4. St Louis, CV Mosby Co, 1974; pp 240–252.

41. Opie LH, Nathan D, Lubbe WF: Biochemical aspects of arrhythmogenesis and ventricular fibrillation. *Am J Cardiol* 1979;43:131–148.