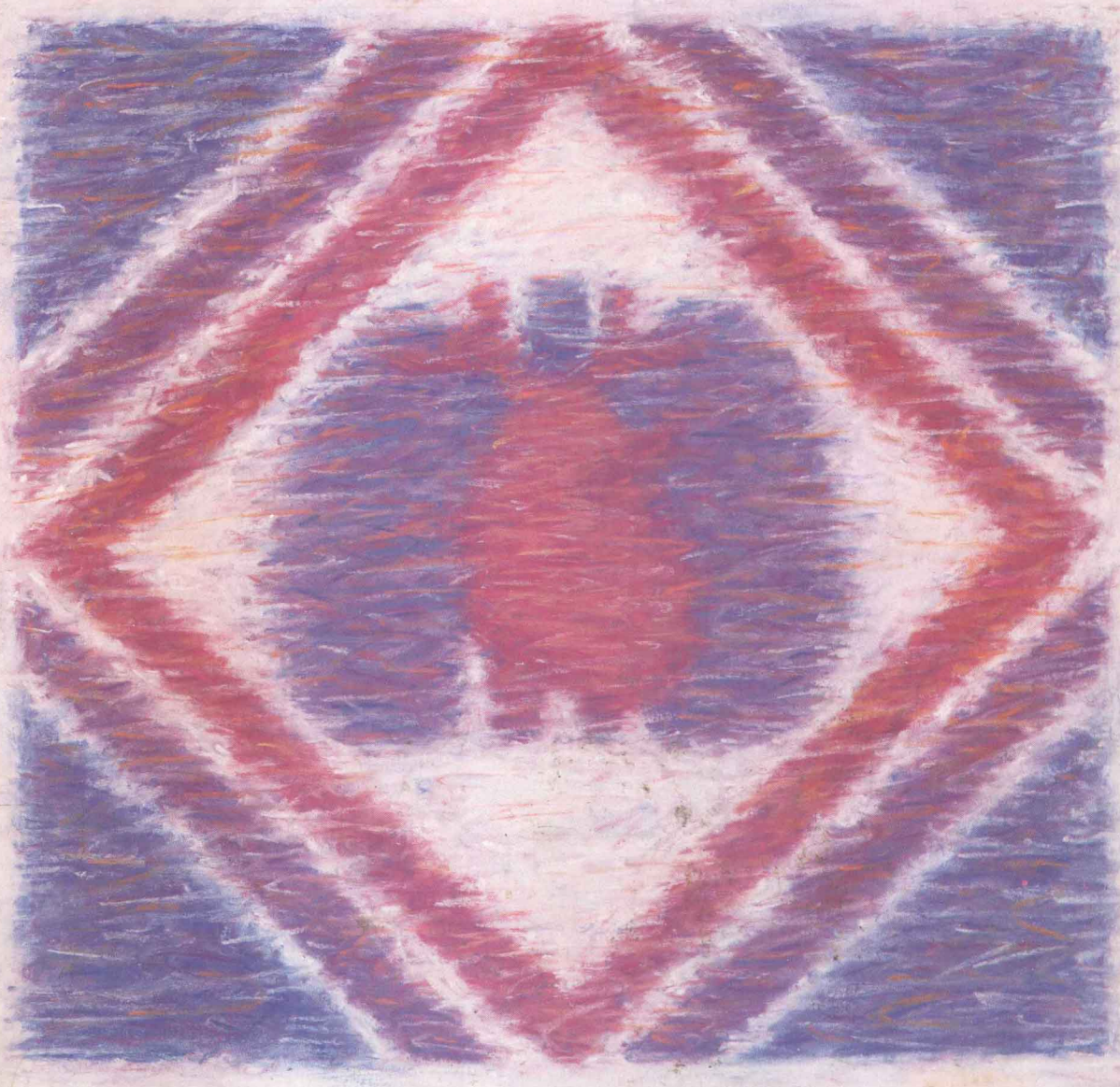
CARDIOPULMONARY EMERGENCIES

- Causes and characteristics
- Assessment and diagnosis
- Treatment options
- Nursing management



CARDIOPULMONARY EMERGENCIES

Springhouse Corporation
Springhouse, Pennsylvania

Staff

Executive Director, Editorial
Stanley Loeb

Editorial Director
Helen Klusek Hamilton

Clinical Director
Barbara F. McVan, RN

Art Director
John Hubbard

Clinical Editors
Joanne Patzek DaCunha, RN, BS,
Diane Schweisguth, RN, BSN, CCRN, CEN

Drug Information Editor
George J. Blake, RPh, MS

Editors
Stephanie Forbes, Carole Gan, Kathy Goldberg, Peter Johnson, Brigid Wallace, Mary Lou Webster

Copy Editor
Doris Weinstock

Editorial Assistants
Maree DeRosa, Beverly Lane

Designers
Stephanie Peters (associate art director), Mary Stangl, Lesley Weissman-Cook

Art Production
Robert Perry (manager), Anna Brindisi, Donald Knauss, Catherine Mace, Robert Wieder

Typography
David Kosten (director), Diane Paluba (manager), Elizabeth Bergman, Joyce Rossi Biletz, Phyllis Marron, Robin Rantz, Valerie L. Rosenberger

Manufacturing
Deborah Meiris (manager), T.A. Landis, Jennifer Suter

Production Coordination
Aline S. Miller (manager), Laurie J. Sander

Library of Congress Cataloging-in-Publication Data
Cardiopulmonary emergencies.
p cm.
Includes bibliographical references.
Includes index.
1. Cardiopulmonary system—Diseases—Nursing. 2. Intensive care nursing. I. Springhouse Corporation.
[DNLM: 1. Angina Pectoris—nursing. 2. Asthma—nursing. 3. Emergencies—nursing. 4. Heart Arrest—nursing. 5. Heart Failure, Congestive—nursing. 6. Myocardial Infarction—nursing. 7. Respiration, Artificial—nursing. 8. Respiratory Distress Syndrome, Adult—nursing. 9. Respiratory Insufficiency—nursing. WY 154 C267]
RC702.C36 1991
610.73'691—dc20
DNLM/DLC 90-9760
ISBN 0-87434-269-4 CIP

CPE-021190

Contributors and consultants

Contributors

CHAPTER 1: Cardiac arrest

Lynne Atkinson Rosenberg, RN, BSN, CEN
MSN Candidate, Gwynedd-Mercy College
Gwynedd Valley, Pa.
Consultant in Emergency Nursing
Past President, Philadelphia Chapter
Emergency Nurses Association

Mark Rosenberg, DO
Director, Emergency Services
Roxborough Memorial Hospital
Philadelphia

CHAPTER 2: Angina

Dawn M. Angst, RN, BSN
Staff Nurse, Critical Care
Lehigh Valley Hospital Center
Allentown, Pa.

Dana A. Bensinger, RN, CCRN
Staff Nurse, Emergency Medicine
St. Luke's Hospital
Bethlehem, Pa.

CHAPTER 3: Myocardial infarction

Kathryn Dolter, RN, BSN, MA, CCRN, CPT/AN
Head Nurse, Neurosurgical Intensive Care
Brooke Army Medical Center
Fort Sam Houston, Tex.

CHAPTER 4: Congestive heart failure

Deborah Panozzo Nelson, RN, MS, CCRN
Cardiovascular Clinical Specialist
Consultant, Visiting Assistant Professor, Staff Nurse
EMS Nursing Education
Purdue University–Calumet
LaGrange, Ill.

CHAPTER 5: Acute respiratory failure

Donald St. Onge, RN, MSN
Pulmonary Clinical Specialist
Allegheny General Hospital
Pittsburgh

CHAPTER 6: Adult respiratory distress syndrome

Phyllis Roach Sutton, RN, MA, CCRN
Clinical Assistant Director of Nursing
New York University Medical Center

CHAPTER 7: Acute asthma

Janet D'Agostino Taylor, RN, MSN
Pulmonary Clinical Specialist
St. Elizabeth's Hospital
Brighton, Mass.

CHAPTER 8: Mechanical ventilation

Karen Sudhoff Allard, RN, MSN, CCRN
Staff Development Educator, Critical Care
University of Cincinnati Hospitals

Consultants

Linda S. Baas, RN, MSN, CCRN
Cardiac Clinical Nurse Specialist
Nursing Consultation Department
University of Cincinnati Hospitals

Sheila Glennon, RN, MA, CCRN
Critical Care Consultant
New York

Foreword

As changing systems of reimbursement have led to rising acuity levels among hospitalized patients, many patients once cared for in the intensive care unit are now in med/surg units, in outpatient rehabilitation centers, or in home care. As a result, nurses in all clinical settings must keep pace with accelerating technology and be prepared to provide patient care that's consistent with the latest approaches to treatment of major cardiac and respiratory disorders. For example, they must be ready to maintain adequate respiration in critically ill patients because respiratory failure can strike suddenly as a complication of many disease states. They must provide quick remedial interventions based on a thorough knowledge of cardiovascular and respiratory mechanisms and the effects of their dysfunction on interacting vital systems. They must know how to help patients recover by correctly using drug therapy, ventilation support, nursing diagnoses, care plans, and patient-teaching plans that enlist the patient's family in helping to manage acute cardiac and respiratory illness.

Cardiopulmonary Emergencies provides an updated and comprehensive guide to nursing care of acute cardiac and respiratory illness that can be applied to a wide range of practice settings—from critical care units to outpatient care. It provides this information in a unique format that encompasses the latest technical information within a holistic nursing approach.

Each of the first seven chapters in this volume focuses on a major cardiac or respiratory disorder: cardiac arrest, angina, myocardial infarction, congestive heart failure, acute respiratory failure, adult respiratory distress syndrome, and acute asthma. The final chapter, on mechanical ventilation, explains the mechanisms and uses of mechanical ventilation and offers specific recommendations for successfully managing nursing care of patients who require it.

Each chapter begins with an introduction that summarizes its importance to practicing nurses and offers a review of fundamentals related to pathophysiology, assessment, and diagnostic tests. Interwoven among major sections of the text, a complete case study illustrates the practical applications of the information contained in the text. By highlighting real nursing transactions during the treatment of a single patient, the case study personalizes the educational content and encourages the reader to relate specific content of this volume to her own patients.

Each chapter also provides current treatment options with emphasis on nursing management. For example, the chapter on cardiac arrest incorporates American Heart Association guidelines for Advanced Cardiac Life Support, including the use of external pacing and defibrillation; nursing measures to prevent or recognize clinical situations that may lead to an arrest; ethical issues related to resuscitation and organ donation; and the special emotional needs of the survivors of a cardiac arrest and their families.

At the end of each chapter, a section on nursing management emphasizes nursing diagnosis as the foundation for nursing care decisions and priorities. This section offers assessment guidelines and interventions for potential and actual complications, with recommendations for patient and family teaching and discharge planning. To provide continuity, each chapter ends with a summary of the case study and briefly discusses the patient outcome.

The final chapter covers care of patients who require mechanical ventilation. It discusses types of ventilatory assistance, therapeutic adjuncts to mechanical ventilation, and monitoring of potential complications. It concludes with guidelines for weaning the patient from the ventilator and thoroughly explores the relevant psychological issues and techniques for successful weaning.

Cardiopulmonary Emergencies offers a wealth of practical information that can help every nurse—from novice to expert—to provide superior care for patients with major cardiac or respiratory illness.

Sandra K. Goodnough, RN, PhD
Critical Care Consultant
Texas Woman's University
Houston

Linda S. Baas, RN, MSN, CCRN
Cardiac Clinical Nurse Specialist
Nursing Consultation Department
University of Cincinnati Hospitals

Contents

CHAPTER 1
Cardiac arrest

Cardiac arrest leads to biological death if resuscitation does not begin within minutes of shutdown of the body's breathing and circulation. Every year, sudden cardiac death takes an estimated toll of 300,000 lives in the United States.

Lifesaving response to cardiac arrest, which includes both basic life support and advanced cardiac life support, is typically handled by an efficiently organized resuscitation team. Successful team intervention can restore the patient's spontaneous respiration and circulation while preserving the function of vital organs.

During resuscitation, nursing concerns include continual assessment and monitoring, as well as participation in cardiopulmonary resuscitation (CPR). After successful resuscitation, nursing concerns include major legal, ethical, and psychosocial issues—for example, questions regarding life-support systems and subsequent quality of life for the patient. After resuscitation and throughout the patient's hospitalization, nursing management should include reassurance and emotional support for both the patient and family.

Case study: Part 1

Collapsed, unconscious, and with no signs of breathing—that's how the security officer described the man he discovered in the restroom of a downtown office building. The security officer notified the operator of the building's switchboard and immediately started CPR. The operator who took the call dispatched an emergency medical services (EMS) mobile intensive care unit (MICU) to the building. When the team arrived, they found the patient—a white man who looked about 35 years old—still unresponsive, with no spontaneous respirations or pulse. Traces of a white powder resembling cocaine were visible in his left nostril. The paramedics took over the CPR at once, established radio contact with the EMS base at the local hospital, and applied a cardiac monitor.

When they saw the chaotic rhythm of ventricular fibrillation on the ECG, they immediately defibrillated the patient at 200 joules; he didn't respond. After a second defibrillation, however, the monitor

Case study: Part 1 *(continued)*

showed a supraventricular tachycardia (SVT) rhythm with occasional premature ventricular contractions (PVCs).

Although the patient's pupils were equal and reactive, he remained unresponsive. Still unable to detect respirations, the paramedics inserted an esophageal obturator airway attached to a bag-mask device. Then they performed manual ventilation at a rate of 12 ventilations/minute and administered a 100-mg bolus of lidocaine by a peripheral vein. As the van approached the emergency department (ED), the paramedics measured a pulse rate of 150 beats/minute and a mean blood pressure of 88 mm Hg. They also noted that the patient's skin was warm, dry, and pale.

Causes and characteristics

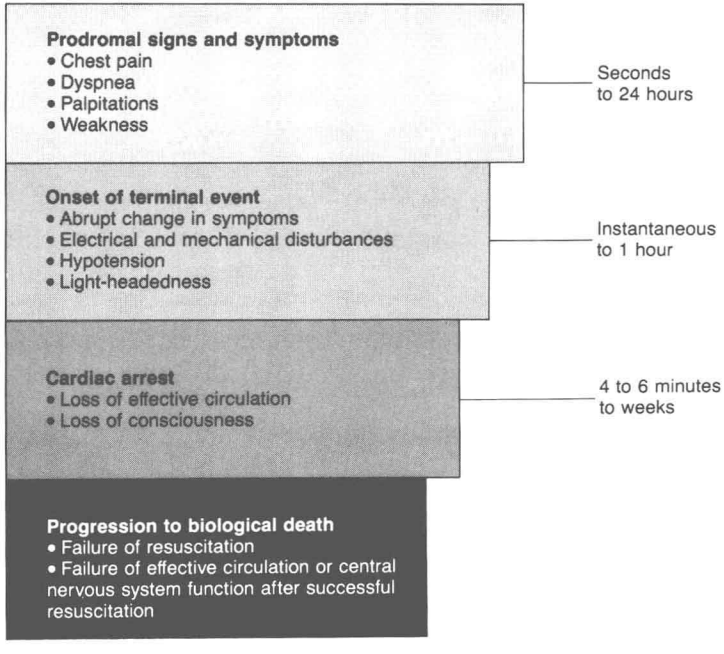
Cardiac arrest is the abrupt cessation of effective heart action resulting in failure to maintain adequate oxygenation of the brain and other vital organs. Sudden cardiac death (SCD) is defined as an abrupt loss of consciousness within 1 hour of the onset of acute cardiac symptoms in an individual with or without preexisting heart disease, in whom the time and mode of death are unexpected. In adults, SCD includes cardiac arrest that is unexpected and independent of a preexisting heart condition. In infants and children, cardiopulmonary arrest is rarely a sudden, primary cardiac event; it is usually the end result of progressive deterioration of both respiration and circulation.

Four factors must be present to satisfy the medical and legal requirements for SCD: prodromal signs and symptoms, onset of the terminal event, cardiac arrest, and biological death. However, progression through all four stages may not be identifiable, depending on the underlying cause. For instance, if the cause is a dysrhythmia, prodrome may not occur and onset of cardiac arrest may be instantaneous; if the cause is cardiac failure, all four stages may be identifiable. (See *Stages of progression: Sudden cardiac death.*)

The underlying cause of most SCDs is coronary artery disease (CAD) and pathologic changes related to it. For example, intraventricular conduction disturbances are known to influence the incidence of SCD in patients with CAD. Anterior myocardial infarctions (MIs) and bundle branch block during the first 30 days after infarction are associated with a risk of ventricular

Stages of progression: Sudden cardiac death

Sudden cardiac death develops in the following sequential stages: prodromal signs and symptoms, onset of the terminal event, the cardiac arrest itself, and progression to biological death. These stages vary greatly. Some individuals, usually when the cause is dysrhythmia, have no prodromal signs and symptoms, with onset leading almost instantaneously to cardiac arrest; some, usually when the cause is cardiac failure, have an onset lasting up to 1 hour before cardiac arrest; and some survive for weeks after the cardiac arrest before progression to biological death if life-support systems are used. These modifying factors influence application of the 1-hour definition of the terminal event. The onset of the terminal event and the cardiac arrest itself are the major clinical factors; the time of biological death has major legal and social importance.



fibrillation. Hypertrophic cardiomyopathy is the most common cause of SCD in athletes under age 35; ischemic heart disease is the most common cause in athletes over age 35.

Other factors known to lead to cardiac arrest include drugs; surgery and invasive procedures; and physiologic factors, such

The deadly hours

Harvard Medical School researchers showed that of 2,203 persons who died within 1 hour of the onset of cardiac symptoms, 31% died in the morning, between 6 a.m. and noon. The peak hour, in which 6% of those struck died, was between 10 a.m. and 11 a.m. The "quiet" hour, in which only 3% died, was between 4 a.m. and 5 a.m.

These statistics match those reported for victims of nonfatal myocardial infarction and strokes. Coincidental? Probably not, say several researchers. Their explanations for the similarity in timing include increased blood pressure peaks and blood platelet adhesion (both of which are common in the morning), leading to loosening of fatty acid deposits inside blood vessels and subsequent thrombosis.

as metabolic abnormalities and reflex stimulation. (See *The deadly hours.*)

Whatever the cause, sudden cardiac arrest produces failure of cardiac muscle contraction. Consequently, electrical activity usually shuts down completely in about 20 to 30 minutes; brain cell death usually follows cardiac arrest in about 10 minutes.

Drugs

Toxic doses of sympathomimetic drugs may indirectly cause SCD by inducing ventricular fibrillation. Toxic levels of acetylcholine or other parasympathomimetic drugs are known to induce asystole, which can lead to SCD. Recent therapy with antiarrhythmic drugs (quinidine, disopyramide phosphate)—particularly those that prolong the QT interval (quinidine), decrease the fibrillation threshold, or increase myocardial irritability—has also been linked to SCD.

Abuse of cocaine is now known to sometimes cause SCD even in persons without a history of heart disease. Cocaine raises heart rate and blood pressure and increases the myocardial oxygen demand. Researchers believe cocaine could create a sudden increase in myocardial oxygen demand while simultaneously interfering with blood flow; this would explain, at least partially, the myocardial ischemia observed in cocaine users. (Cocaine has pronounced vasoconstrictor effects.) Further research may identify the mechanism by which cocaine induces cardiac arrest.

Surgery and invasive procedures

Cardiac arrest during surgery can result directly from the effects of general anesthetics and from hypovolemia, hypoxemia, hypercapnia, or respiratory acidosis, which may occur during surgery. It can also follow certain common surgical procedures that are associated with vagal stimulation, such as abdominal surgery involving traction on the gallbladder and eye surgery. With increased external ocular pressure, an afferent oculocardiac reflex that involves both the vagus and trigeminal nerves may cause dysrhythmias.

Certain invasive procedures or complications of diagnostic procedures, such as cardiac catheterization, can result in SCD. For example, insertion of a pacemaker or pulmonary artery catheter may irritate the ventricular myocardium, thereby causing

ventricular fibrillation and triggering cardiac arrest. Endotracheal intubation, bronchoscopy, and colonoscopy also tend to precipitate lethal dysrhythmias through stimulation of vagal nerve fibers in the gastrointestinal (GI) and respiratory organs.

Physiologic factors

Central nervous system–related effects, notably those associated with the heart's electrical stability, have also been cited as contributory factors in SCD. Sympathetic nervous system imbalance is identified in some hereditary forms of prolonged QT-interval syndrome. Psychological stress, behavioral abnormalities, and emotional extremes have also been identified as risk factors in SCD. Curiously, auditory stimulation and auditory auras have also been linked to SCD.

Sudden death can also follow metabolic derangements, such as hypothermia and acid-base disorders, and fluid and electrolyte disturbances, such as hypovolemia, hypokalemia, hyperkalemia, hypocalcemia, and hypomagnesemia. In patients with diabetes, autonomic denervation increases the risk of "silent MI" and of SCD.

Previous CAD or certain other cardiac abnormalities may place a person at risk for SCD. For example, 75% of all patients who have died suddenly have a history of earlier MI. Other predisposing cardiac conditions include the following.

Ventricular ectopy or PVC. In persons over age 30, ventricular ectopy greater than 6/minute is a predisposing factor for CAD and for SCD. Survivors of MI who experience frequent or complex forms of PVCs are at an even greater risk. Complex forms of PVCs that carry an increased risk of SCD include multifocal PVCs, bigeminy, short coupling intervals (R-on-T phenomenon), and, especially, salvos of three or more ectopic beats.

Left ventricular dysfunction. This condition can be considered both a co-factor and an independent predictor for SCD. Left ventricular dysfunction with postmyocardial infarction PVCs greatly increases the risk of death, especially in the first 6 months after infarction. Complex postinfarction PVCs seem to carry a greater risk in patients with non-Q-wave infarctions than in those with transmural infarctions.

Reflex stimulation. Stimulation of vagal reflexes—for example, by straining (Valsalva's maneuver) — can precipitate car-

diac dysrhythmia. Remember that increased vagal tone may not only propagate sinus bradycardia by slowing the heart rate, but may also depress atrioventricular (AV) conduction, causing atrial and ventricular slowing or asystole.

Individual characteristics

Certain individual characteristics may influence or increase the patient's risk of SCD. These include age, heredity, gender, psychosocial factors, or a history of cardiac disease (especially ventricular ectopy or left ventricular dysfunction).

Age. Two major high-risk age-groups are children between birth and age 6 months and adults between ages 45 and 75. In adults, the incidence of SCD related to CAD rises with advancing age; however, the incidence related to all other causes decreases with advancing age. In children older than those at risk for sudden infant death syndrome, SCD is associated with identifiable heart disease. SCD usually occurs in children who have undergone surgery for congenital cardiac disease (about 25%) or who have cardiac lesions, typically one of the following: congenital aortic stenosis, Eisenmenger's syndrome, tetralogy of Fallot, pulmonary stenosis or atresia, or hypertrophic obstructive cardiomyopathy.

Heredity. Although not a common cause of SCD, genetic predisposition is linked to such syndromes as hereditary and congenital QT-interval prolongation, hypertrophic obstructive cardiomyopathy, and familial SCD in children and adolescents. Progressive familial conduction system disease also carries a risk of SCD.

Gender. SCD is far more common in men than in women. Apparently, premenopausal women benefit from greater protection from coronary atherosclerosis.

Psychosocial factors. Certain life-styles place individuals at greater risk for SCD. Cigarette smokers between ages 30 and 59, for example, have a twofold to threefold increased risk of SCD. Because smoking lowers the threshold for ventricular fibrillation, it is the only CAD risk factor that is also an independent risk factor for ventricular fibrillation.

Obesity— often associated with high cholesterol levels, hypertension, physical inactivity, and heavy consumption of alcohol—is also thought to increase the risk of SCD, although

controversy surrounds some of the supporting evidence for this view. Recent changes in health, work, home and family, and personal and social factors cause high levels of stress and increase the risk for SCD. Other significant psychosocial factors include social isolation, a history of psychiatric treatment, and a low educational level.

Cardiac history. A history of CAD, ventricular ectopy, or left ventricular dysfunction is associated with higher risk of SCD.

Pathophysiology

The underlying pathophysiologic cause of deadly tachydysrhythmia, severe bradydysrhythmia, or asystole typically includes complex interactions among coronary vascular events, myocardial injury, and variations in the autonomic, metabolic, and conductive state of the myocardium. Probably multiple pathophysiologic abnormalities combine to induce SCD. However, no single hypothesis explains the mechanisms of these interactions.

Lethal dysrhythmias usually arise as a consequence of acute myocardial ischemia—a condition marked by dramatic electrophysiologic changes, including those associated with transmembrane action potentials and refractory periods. Furthermore, reperfusion after transient ischemia may cause lethal dysrhythmias. Consequently, onset of acute ischemia is now known to cause immediate electrical, mechanical, and biochemical dysfunction of myocardial cells.

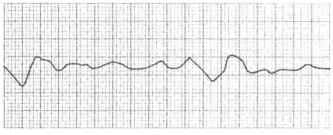
Several changes immediately follow ischemia at the cellular level: Myocardial cell membranes lose their selectivity, so potassium ions now flow out of the cell and calcium ions enter the cell, transmembrane resting potentials are reduced, acidosis results, and automaticity is enhanced in some tissues.

During reperfusion, a different series of changes occur: the continued influx of calcium ions, responses to alpha- or beta-adrenoceptor stimulation, and neurophysiologically induced afterdepolarization.

Myocardial tissue status at the time of ischemic injury is another important factor. Abnormal and chronically hypertrophied myocardial tissue, for example, seems more susceptible to the destabilizing electrical effects of acute ischemia. Hypokalemia is known to render the ventricular myocardium more susceptible to potentially lethal dysrhythmias. Such dysrhythmias

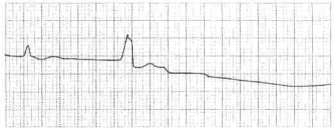
Common ECG rhythms in cardiac arrest

VENTRICULAR FIBRILLATION



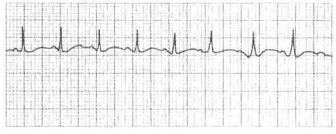
Characteristics
- Ventricular rhythm rapid and chaotic, indicating varying degrees of depolarization and repolarization; QRS complexes not identifiable
- Patient unconscious at onset
- Absent pulses, heart sounds, and blood pressure
- Dilated pupils; rapid development of cyanosis

VENTRICULAR ASYSTOLE



Characteristics
- Totally absent ventricular electrical activity
- Possible P waves
- Possible severe metabolic deficit or extensive myocardial damage

ELECTROMECHANICAL DISSOCIATION



Characteristics
- Electrical activity but no pulse
- Organized electrical activity without any evidence of effective myocardial contraction
- Possible failure in the calcium transport system (can cause electromechanical dissociation)
- Possible association with profound hypovolemia, cardiac tamponade, myocardial rupture, massive myocardial infarction, or tension pneumothorax

then result from a triggering event imposed on a susceptible myocardium. For example, premature impulses in a chronically abnormal myocardium may trigger multiple uncoordinated reentrant pathways—that is, ventricular fibrillation. (See *Common ECG rhythms in cardiac arrest.*)

Ventricular fibrillation. This condition is often a direct result of ischemia. (Physiologically, ischemia is characterized by a sudden drop in the transmembrane resting potential, amplitude, and duration of the action potential in the affected area.) Before ischemic cells become completely unable to transmit electrical signals to neighboring cells, they show reduced excitability with slow conduction and electrophysiologically unstable membranes. In this situation, premature impulses generated between adjacent pockets of ischemic and normal heart tissue create a setting for reentry—slow conduction in a unidirectional block—which makes the myocardium vulnerable to reentrant dysrhythmias. Such a condition almost invariably leads to ventricular fibrillation.

Ventricular asystole. When the heart fails to maintain automatic pacing because of electrophysiologic disruptions at the sinus node or the AV junction, bradycardia and asystole may result. Bradydysrhythmias and asystolic arrests are more common in severely diseased hearts.

Electromechanical dissociation. With electromechanical dissociation (EMD), electrical rhythmicity continues in the absence of effective mechanical function. In the primary form, the muscular walls of the ventricles fail to sustain contractions although electrical activity continues. The secondary form results from sudden cessation of cardiac venous return, as in massive pulmonary embolism, acute malfunction of prosthetic valves, severe blood loss, or cardiac tamponade. EMD is the least common mechanism of SCD.

Case study: Part 2

As soon as the MICU arrived at the ED, the medical team connected the patient to another cardiac monitor. Again, the ECG showed SVT with occasional PVCs. They continued the lidocaine drip at an infusion rate of 2 mg/minute.

The patient's pulse rate and mean arterial blood pressure remained unchanged: 150 beats/minute and 88 mm Hg, respectively. His skin was still warm, dry, and pale; his pupils were equal and reactive; and he remained unresponsive without spontaneous respirations. The ED team intubated him and placed him on an MA2 mechanical ventilator. A nurse drew blood samples for analysis of serum electrolytes, cardiac enzymes, and arterial blood gases (ABGs). A chest X-ray and 12-lead ECG showed no abnormalities.

As the patient was being prepared for transfer to the intensive care unit (ICU), an ED nurse searched his belongings for identification and information concerning his medical history. Reaching into the pocket of his suit jacket, she felt a powdery substance, then discovered a small sterling silver pillbox with its top unhinged. Inside the box, she saw a small mound of white powder.

She also found a wallet and began looking for a Medic Alert card or other medical history information. She found none—but she did find a driver's license and business card. The patient, she learned, was Garon Gardner II, a 33-year-old corporate attorney for a downtown law firm. After finding Mr. Gardner's telephone number in the directory, she called his home. His wife Marilyn answered and said she'd be at the hospital as soon as possible.

In the meantime, the laboratory report arrived. ABG values were as follows: pH, 7.20; $Paco_2$, 50 mm Hg; Pao_2, 60 mm Hg; HCO_3^-, 15 mEq/liter; and Sao_2, 0.90 (90%).

Assessment and diagnosis

In the patient with SCD, emergency assessment and intervention must be performed simultaneously. This section will review confirmation of the four progressive stages of SCD: prodromal signs and symptoms, onset of the terminal event, cardiac arrest, and biological death.

Prodromal signs and symptoms

Common signs and symptoms of impending cardiac arrest or SCD include onset of chest pain, chest pain unrelieved by nitroglycerin, chest pain at rest, palpitations and dysrhythmias, shortness of breath at rest, dyspnea, increased orthopnea, diaphoresis, and sudden changes in blood pressure and level of consciousness. Other common but nonspecific symptoms are weakness or fatigue and restlessness. (See *Diagnostic profile: Laboratory tests.*)

Assessment of patients with prodromal signs and symptoms should be performed quickly and accurately. In emergency situations, assessment usually may have to proceed simultaneously with treatments, such as insertion of an intravenous (I.V.) line and administration of supplemental oxygen.

The patient's chief complaint will often guide you in obtaining a personal history. Phrase your questions so the patient can provide "yes," "no," or short responses—for example, "Do you have chest pain now?" or "Are you having difficulty breathing?" Take a few moments to look at your patient. Observe skin color. Is it cyanotic? Ruddy? Is his skin cool to the touch or clammy? Watch your patient's body language. Is he grimacing? Are his fists clenched? During such assessment, careful listening is vital. Commonly, the patient in prodromal stages subtly expresses a feeling of "impending doom." More often than not, this feeling reliably signals an impending emergency. Learn to listen for such clues and develop your own intuitive "sixth sense" that so often helps expert nurses give excellent care.

While obtaining the patient's history, remember that cardiac arrest or SCD may occur from noncardiac-related complaints. For this reason, check vital signs immediately and constantly monitor:

- cardiovascular system (heart rate, rhythm, and sounds; rate, rhythm, equality and presence of arterial pulses; neck veins for distention and pulsation; point of maximum impulse for thrills)

DIAGNOSTIC PROFILE

Laboratory tests

Tests that measure serum electrolytes and cardiac enzymes are commonly performed to help determine the cause of cardiac arrest or evaluate treatment. *Note:* Laboratory values may vary.

Serum electrolytes

Electrolyte profiles commonly measure potassium, sodium, chloride, calcium, phosphate, magnesium, blood urea nitrogen (BUN), carbon dioxide (CO_2) content, and glucose.

- Serum potassium

Physiologic values: 3.5 to 5 mEq/liter
Possible abnormalities: hyperkalemia, hypokalemia

- Serum sodium

Physiologic values: 135 to 145 mEq/liter
Possible abnormalities: hypernatremia, hyponatremia

- Serum chloride

Physiologic values: 95 to 105 mEq/liter
Possible abnormalities: hyperchloremia, hypochloremia (commonly associated with hypokalemia and metabolic alkalosis)

- Serum calcium

Total serum calcium values: 8.5 to 10.5 mg/dl or 4.0 to 5.5 mEq/liter
Serum ionized calcium values: usually 50% of total serum calcium
Possible abnormalities: acidosis, alkalosis

- Serum phosphate

Physiologic values: 2.5 to 4.8 mg/dl or 1.8 to 2.6 mEq/liter
Possible abnormalities: hyperphosphatemia, hypophosphatemia

- Serum magnesium

Physiologic values: 1.8 to 3.0 mg/dl or 1.5 to 2.5 mEq/liter
Possible abnormalities: hypermagnesemia, hypomagnesemia

- BUN

Physiologic values: 10 to 20 mg/dl
Possible abnormalities: hypovolemia, excessive protein intake, increased catabolism, overhydration, reduced protein intake

- CO_2 content

Physiologic values: 24 to 30 mEq/liter *Note:* CO_2 content reflects total serum bicarbonate and carbonic acid levels.
Possible abnormalities: metabolic alkalosis, metabolic acidosis

- Serum glucose

Physiologic values: 70 to 100 mg/dl
Possible abnormalities: osmotic diuresis, hypovolemia.

Cardiac enzymes

Enzymes and subtypes of enzymes (isoenzymes) can be detected in the circulation roughly 30 to 60 minutes after irreversible myocardial tissue injury. Clinicians typically examine the pattern of enzyme levels when they suspect myocardial infarction (MI). Elevated levels of creatine phosphokinase (CPK), lactic dehydrogenase (LDH), and serum aspartate aminotransferase (AST), formerly SGOT, indicate MI.

- CPK is considered the most accurate indicator of MI. The isoenzyme CPK-MB concentrates mainly in the heart; it rises markedly 4 to 8 hours after MI onset and may remain elevated for 72 hours.

Physiologic values: undetectable to 7 IU/liter
Possible abnormalities: myocarditis, cardiac trauma (possibly resulting from cardiopulmonary resuscitation)

- LDH levels peak 3 to 6 days following onset of chest pain.

Physiologic values: 48 to 115 IU/liter. The isoenzymes LDH_1 and LDH_2 concentrate primarily in the heart.
Possible abnormalities: anemia, leukemia, myocarditis

- AST concentrates in the heart, but it is not a reliable marker of cardiac dysfunction. Elevated AST readings can also result from liver disease, shock, and pulmonary embolism. Many hospitals no longer use AST levels as part of their cardiac enzyme analysis.

- respiratory system (breath sounds, respiratory rate and quality, and chest symmetry)
- renal system (urine output for amount, color, and odor)
- skin (color, temperature, turgor, nail bed capillary refill in the extremities)