TABLE 327-1 DISTINCTION BETWEEN CARDIOVASCULAR COLLAPSE, CARDIAC ARREST, AND DEATH

Term	Definition	Qualifiers	Mechanisms
Cardiovascular collapse	Sudden loss of effective blood flow due to cardiac and/or peripheral vascular factors that may reverse spontaneously (e.g., neurocardiogenic syncope, vasovagal syncope) or require interventions (e.g., cardiac arrest)	Nonspecific term: includes cardiac arrest and its consequences and transient events that characteristically revert spontaneously	Same as "Cardiac Arrest," plus vasodepressor syncope or other causes of transient loss of blood flow
Cardiac arrest	Abrupt cessation of cardiac mechanical function, which may be reversible by a prompt intervention but will lead to death in its absence	Rare spontaneous reversions; likelihood of successful intervention relates to mechanism of arrest, clinical setting, and prompt return of circulation	Ventricular fibrillation, ventricular tachycardia, asystole, bradycardia, pulseless electrical activity, noncardiac mechanical factors (e.g., pulmonary embolism)
Sudden cardiac death	Sudden, irreversible cessation of all biological functions	None	

Source: Modified from RJ Myerburg, A Castellanos: Cardiac arrest and sudden cardiac death, in P Libby et al (eds): *Braunwald's Heart Disease*, 8th ed. Philadelphia, Saunders, 2008.

TABLE 327-2 CARDIAC ARREST AND SUDDEN CARDIAC DEATH

Structural Substrates and Causes

- I. Coronary heart disease
  - A. Coronary artery abnormalities
    - 1. Chronic atherosclerotic lesions
    - 2. Active lesions (plaque fissuring, platelet aggregation, acute thrombosis)
    - 3. Anomalous coronary artery anatomy
  - B. Myocardial infarction
    - 1. Healed
    - 2. Acute
- II. Myocardial hypertrophy
  - A. Secondary
  - B. Hypertrophic cardiomyopathy
    - 1. Obstructive
    - 2. Nonobstructive
- III. Dilated cardiomyopathy—primary muscle disease
- IV. Inflammatory and infiltrative disorders
  - A. Myocarditis
  - B. Noninfectious inflammatory diseases
  - C. Infiltrative diseases
- V. Valvular heart disease
- VI. Electrophysiologic abnormalities, structural
  - A. Anomalous pathways in Wolff-Parkinson-White syndrome
  - B. Conducting system disease
- VII. Inherited disorders associated with electrophysiologic abnormalities (congenital long QT syndromes, right ventricular dysplasia, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia, etc.)

Triggers for Expression of Cardiac Arrest

- I. Alterations of coronary blood flow
  - A. Transient ischemia
  - B. Reperfusion after ischemia
- II. Low cardiac output states
  - A. Heart failure
    - 1. Chronic
    - 2. Acute decompensation
  - B. Shock
- III. Systemic metabolic abnormalities
  - A. Electrolyte imbalance (e.g., hypokalemia)
  - B. Hypoxemia, acidosis
- IV. Neurologic disturbances
  - A. Autonomic fluctuations: central, neural, humoral
  - B. Receptor function
- V. Toxic responses
  - A. Proarrhythmic drug effects
  - B. Cardiac toxins (e.g., cocaine, digitalis intoxication)
  - C. Drug interactions

age within this range is associated with increasing risk for sudden *cardiac* death (Fig. 327-1*A*). From 1 to 13 years of age, only one of five sudden *natural* deaths is due to cardiac causes. Between 14 and 21 years of age, the proportion increases to 30%, and it rises to 88% in the middle-aged and elderly.

Young and middle-aged men and women have different susceptibilities to SCD, but the sex differences decrease and ultimately disappear with advancing age. The difference in risk for SCD parallels the differences in age-related risks for other manifestations of coronary heart disease (CHD) between men and women. As the gender gap for manifestations of CHD closes in the sixth to eighth decades of life, the excess risk of SCD in males progressively narrows. Despite the lower incidence among younger women, coronary risk factors such as cigarette smoking, diabetes, hyperlipidemia, and hypertension are highly influential, and SCD remains an important clinical and epidemiologic problem. The incidence of SCD among the African-American population appears to be higher than it is among the white population; the reasons remain uncertain.

Genetic factors contribute to the risk of acquiring CHD, and a genetic basis for its expression as SCD is being explored. A genetic hypothesis for at least part of the SCD risk is supported by data suggesting a familial predisposition to SCD as a specific form of expression of CHD. A parental history of SCD as a first cardiac event increases the probability that an acute coronary event in the offspring will express similarly. In a number of less common syndromes, such as hypertrophic cardiomyopathy, congenital long QT interval syndromes, right ventricular dysplasia, and the syndrome of right bundle branch block and nonischemic ST-segment elevations (Brugada syndrome), and other more rare syndromes, there is a specific inherited risk of ventricular arrhythmias and SCD (Chap. 277).

The etiologic structural substrates and functional factors contributing to expression of the SCD syndrome are listed in Table 327-2. Worldwide, and especially in Western cultures, coronary atherosclerotic heart disease is the most common structural abnormality associated with SCD in middle-aged and older adults. Up to 80% of all SCDs in the United States are due to the consequences of coronary atherosclerosis. The nonischemic cardiomyopathies (dilated and hypertrophic, collectively; Chap. 273e) account for another 10–15% of SCDs, and all the remaining diverse etiologies cause only 5–10% of all SCDs. The inherited arrhythmia syndromes (see above and Table 327-2) are proportionally more common causes in adolescents and young adults. For some of these syndromes, such as hypertrophic cardiomyopathy (Chap. 287), the risk of SCD increases significantly after the onset of puberty.

Transient ischemia in a previously scarred or hypertrophied heart, hemodynamic and fluid and electrolyte disturbances, fluctuations in autonomic nervous system activity, and transient electrophysiologic changes caused by drugs or other chemicals (e.g., proarrhythmia) have all been implicated as mechanisms responsible for the transition from electrophysiologic stability to instability. In addition, reperfusion of ischemic myocardium may cause transient electrophysiologic instability and arrhythmias.