SPECIAL CONSIDERATIONS

Risk of Iatrogenic Cardiogenic Shock In the treatment of pulmonary edema, vasodilators lower BP, and their use, particularly in combination, may lead to hypotension, coronary artery hypoperfusion, and shock (Fig. 326-1). In general, patients with a *hypertensive* response to pulmonary edema tolerate and benefit from these medications. In normotensive patients, low doses of single agents should be instituted sequentially, as needed.

Acute Coronary Syndromes (See also Chap. 295) Acute STEMI complicated by pulmonary edema is associated with in-hospital mortality rates of 20–40%. After immediate stabilization, coronary artery blood flow must be reestablished rapidly. When available, primary PCI is preferable; alternatively, a fibrinolytic agent should be administered. Early coronary angiography and revascularization by PCI or CABG also are indicated for patients with non-ST elevation acute coronary syndrome. Assist devices may be used selectively as noted for refractory pulmonary edema.

Extracorporeal Membrane Oxygenation For patients with acute, severe noncardiogenic edema with a potential rapidly reversible cause, ECMO may be considered as a temporizing supportive measure to achieve adequate gas exchange. Usually venovenous ECMO is used in this setting.

Unusual Types of Edema Specific etiologies of pulmonary edema may require particular therapy. Reexpansion pulmonary edema can develop after removal of longstanding pleural space air or fluid. These patients may develop hypotension or oliguria resulting from rapid fluid shifts into the lung. Diuretics and preload reduction are contraindicated, and intravascular volume repletion often is needed while supporting oxygenation and gas exchange.

High-altitude pulmonary edema often can be prevented by use of dexamethasone, calcium channel–blocking drugs, or long-acting inhaled β_2-adrenergic agonists. Treatment includes descent from altitude, bed rest, oxygen, and, if feasible, inhaled nitric oxide; nifedipine may also be effective.

For pulmonary edema resulting from upper airway obstruction, recognition of the obstructing cause is key, because treatment then is to relieve or bypass the obstruction.

327 Cardiovascular Collapse, Cardiac Arrest, and Sudden Cardiac Death

Robert J. Myerburg, Agustin Castellanos

OVERVIEW AND DEFINITIONS

Sudden cardiac death (SCD) is defined *as natural death due to cardiac causes* in a person who may or may not have previously recognized heart disease but in whom the time and mode of death are *unexpected*. The term "sudden," in the context of SCD, is defined for most clinical and epidemiologic purposes as *1 h or less* between a change in clinical status heralding the onset of the terminal clinical event and the cardiac arrest itself. One exception is unwitnessed deaths, in which pathologists may expand the temporal definition to 24 h after the victim was last seen to be alive and stable.

Another exception is the variable interval between cardiac arrest and biological death that results from community-based interventions, following which victims may remain biologically alive for days or even weeks after a cardiac arrest that has resulted in irreversible central nervous system damage. Confusion in terms can be avoided by adhering strictly to definitions of cardiovascular collapse, cardiac arrest, and death (Table 327-1). Although cardiac arrest is often potentially reversible by appropriate and timely interventions, death is biologically, legally, and literally an absolute and irreversible event. Biological death may be delayed by interventions, but the relevant pathophysiologic event remains the sudden and unexpected cardiac arrest. Accordingly, for statistical purposes, deaths that occur during hospitalization or within 30 days after resuscitated cardiac arrest are counted as sudden deaths.

The majority of natural deaths are caused by cardiac disorders. However, it is common for underlying heart diseases—often far advanced—to go unrecognized before the fatal event. As a result, up to two-thirds of all SCDs occur as the first clinical expression of previously undiagnosed disease or in patients with known heart disease, the extent of which suggests low individual risk. The magnitude of sudden *cardiac* death as a public health problem is highlighted by the estimate that ~50% of all cardiac deaths are sudden and unexpected, accounting for a total SCD burden estimated to range from <200,000 to >450,000 deaths each year in the United States. SCD is a direct consequence of cardiac arrest, which may be reversible if addressed promptly. Because resuscitation techniques and emergency rescue systems are available to respond to victims of out-of-hospital cardiac arrest, which was uniformly fatal in the past, understanding the SCD problem has practical clinical importance.

CLINICAL DEFINITION OF FORMS OF CARDIOVASCULAR COLLAPSE

Cardiovascular collapse is a general term connoting loss of sufficient cerebral blood flow to maintain consciousness due to acute dysfunction of the heart and/or peripheral vasculature. It may be caused by vasodepressor syncope (vasovagal syncope, postural hypotension with syncope, neurocardiogenic syncope; Chap. 27), a transient severe bradycardia, or cardiac arrest. The latter is distinguished from the transient forms of cardiovascular collapse in that it usually requires an active intervention to restore spontaneous blood flow. In contrast, vasodepressor syncope and other primary bradyarrhythmic syncopal events are transient and non-life-threatening, with spontaneous return of consciousness.

In the past, the most common electrical mechanism for cardiac arrest was ventricular fibrillation (VF) or pulseless sustained ventricular tachycardia (PVT). These were the initial rhythms recorded in 60–80% of cardiac arrests, with VF being the far more common of the two. Severe persistent bradyarrhythmias, asystole, and pulseless electrical activity (PEA; organized electrical activity, unusually slow, without mechanical response, formerly called electromechanical dissociation [EMD]) caused another 20–30%. Currently, asystole has emerged as the most common mechanism recorded at initial contact (45–50% of cases). PEA accounts for 20–25%, and VF is now present on initial contact in 25–35%. Undoubtedly, a significant proportion of the asystole cases began as VF and deteriorated to asystole because of long response times, but there are data suggesting an absolute reduction in VF as well. Acute low cardiac output states, having a precipitous onset, also may present clinically as a cardiac arrest. These hemodynamic causes include massive acute pulmonary emboli, internal blood loss from a ruptured aortic aneurysm, intense anaphylaxis, and cardiac rupture with tamponade after myocardial infarction (MI).

ETIOLOGY, INITIATING EVENTS, AND CLINICAL EPIDEMIOLOGY

Clinical, epidemiologic, and pathologic studies have provided information on the underlying *structural substrates* in victims of SCD and identified subgroups at high risk for SCD. In addition, studies of clinical physiology have begun to identify *transient functional factors* that may convert a long-standing underlying structural abnormality from a stable to an unstable state, leading to the onset of cardiac arrest (Table 327-2).

Cardiac disorders constitute the most common causes of sudden *natural* death. After an initial peak incidence of sudden death between birth and 6 months of age (sudden infant death syndrome [SIDS]), the incidence of sudden death declines sharply and remains low through childhood and adolescence. Among adolescents and young adults, the incidence of SCD is approximately 1 per 100,000 population per year. The incidence begins to increase in adults over age 30 years, reaching a second peak in the age range of 45–75 years, when it approximates 1–2 per 1000 per year among the unselected adult population. Increasing