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function and HF are at substantially elevated SCD risk, only ~20% of all SCDs occur in patients with poor left ventricular function. Most SCDs occur in individuals with preserved ventricular function who would not qualify for a primary prevention ICD. Although SCD rates are elevated compared to the general population, the absolute SCD risk in patients with CHD or HF who have an LVEF >35% is not high enough to warrant consideration of ICD therapy. While the incidence of SCD is lower in patients with preserved LVEF, SCD accounts for a greater proportion of cardiac deaths, and active efforts are being made to advance SCD risk stratification in this segment of the population. However, at present, SCD prevention primarily involves cardiac risk factor modification and standard medical therapy for the underlying condition.

Preventing Sudden Death in the General Population Only about one-half of men and one-third of women who suffer SCA are recognized to have heart disease prior to the event, and only half have warning symptoms prior to the event. SCD often occurs with out warning as the first manifestation of cardiac disease. In order to prevent these SCDs, preventive interventions would need to be employed broadly to the general population. Although several risk scores have recently been developed with the intent to stratify SCD risk in low-risk populations, the clinical utility to date is limited by the low absolute incidence of SCD, which is estimated to be only 50–90 per 100,000 in the general adult population. Therefore, current efforts aimed at preventing SCD in general populations primarily focus on modification of the SCD risk factors outlined previously. Individuals who adhere to a low-risk, healthy lifestyle that includes avoidance of smoking, maintaining a healthy body weight, participating in moderate exercise, and a Mediterranean-type dietary pattern have markedly lower rates of SCD. A substantial number of SCDs are likely to be preventable through lifestyle modifications and treatment of risk factors. PART 8 Critical Care Medicine ■

■ **FURTHER READING** Al-Khatib SM et al: 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/ American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. J Am Coll Cardiol 72:e91, 2018. Callaway CW et al: Part 8: Post-cardiac arrest care: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 132:S465,

Dankiewicz J et al: Hypothermia versus normothermia after out-of-hospital cardiac arrest. *N Engl J Med* 384:2283, 2021. Deo R, Albert CM: Epidemiology and genetics of sudden cardiac death. *Circulation* 125:620, 2012. Marijon E et al: Lancet Commission to reduce the global burden of sudden cardiac death: A call for multidisciplinary action. *Lancet* 402:883, 2023. Merchant RM et al: Part 1: Executive summary: 2020 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation* 142:S337, 2020. Myerburg RJ et al: Pulseless electric activity: Definition, causes, mechanisms, management, and research priorities for the next decade: Report from a National Heart, Lung, and Blood Institute workshop. *Circulation* 128:2532, 2013. Perman SM et al: 2023 American Heart Association focused update on adult advanced cardiovascular life support: An update to the American Heart Association Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation* 149:e254, 2024. Zeppenfeld K et al: 2022 ESC guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *Eur Heart J* 43:3997, 2022.

Section 3 Neurologic Critical Care J. Claude Hemphill, III, Wade S. Smith,

S. Andrew Josephson, Daryl R. Gress

Nervous System

Disorders in Critical Care Life-threatening neurologic illness may be caused by a primary disorder affecting any region of the neuraxis or may occur as a consequence of a systemic disorder such as hepatic failure, multisystem organ failure, or cardiac arrest (Table 318-1). Neurologic critical care focuses on preservation of neurologic tissue and prevention of secondary brain injury caused by ischemia, hemorrhage, edema, herniation, and elevated intracranial pressure (ICP).

Encephalopathy is a general term describing brain dysfunction that is diffuse, global, or multifocal. Severe acute encephalopathies represent a group of various disorders due to different neurologic or systemic etiologies but that share the common themes of primary and secondary brain injury. ■

■ **PATHOPHYSIOLOGY** Brain Edema Swelling, or edema, of brain tissue occurs with many types of brain injury. The two principal types of edema are vasogenic and cytotoxic. Vasogenic edema refers to the influx of fluid and solutes into the brain through an incompetent blood-brain barrier (BBB). In the normal cerebral vasculature, endothelial tight junctions associated with astrocytes create an impermeable barrier (the BBB), through which access into the brain interstitium is dependent upon specific transport mechanisms. The BBB may be compromised in ischemia, trauma, infection, and metabolic derangements, and typically develops rapidly following injury. Cytotoxic edema results from cellular swelling, membrane breakdown, and ultimately cell death. Clinically significant brain edema usually represents a combination of vasogenic and cytotoxic components. Edema can lead to increased ICP as well as tissue shifts and brain displacement or herniation from focal processes (Chap. 30). These tissue shifts can cause injury by mechanical distention and compression in addition to the ischemia of impaired perfusion consequent to the elevated ICP. Ischemic Cascade and Cellular Injury When delivery of substrates, principally oxygen and glucose, is inadequate to sustain cellular function, a series of interrelated biochemical reactions known as the ischemic cascade is initiated (see Fig. 437-2). The release of excitatory amino acids, especially glutamate, leads to influx of calcium and sodium ions, which disrupt cellular homeostasis. An increased intracellular calcium concentration may activate proteases and lipases, which then lead to lipid peroxidation and free radical-mediated cell membrane injury. Cytotoxic edema ensues, and ultimately necrotic cell death and tissue infarction occur. This pathway to

irreversible cell death is common to ischemic stroke, global cerebral ischemia, and traumatic brain injury. Penumbra refers to areas of ischemic brain tissue that have not yet undergone irreversible infarction, implying that these regions are potentially salvageable if ischemia can be reversed. Factors that may exacerbate ischemic brain injury include systemic hypotension and hypoxia, which further reduce substrate delivery to vulnerable brain tissue, and fever, seizures, and hyperglycemia, which can increase cellular metabolism, outstripping compensatory processes. Clinically, these events are known as secondary brain insults because they lead to exacerbation of the primary brain injury. Prevention, identification, and treatment of secondary brain insults are fundamental goals of management. An alternative pathway of cellular injury is apoptosis. This process implies programmed cell death, which may occur in the setting of ischemic stroke, global cerebral ischemia, traumatic brain injury, and

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