given 1 meq/kg $NaHCO_3$ initially and an additional 50% of the dose repeated every 10–15 min. However, it should not be used routinely.

After initial unsuccessful defibrillation attempts or with persistent/recurrent electrical instability, antiarrhythmic therapy should be instituted. Intravenous amiodarone has emerged as the initial treatment of choice (150 mg over 10 min, followed by 1 mg/min for up to 6 h and 0.5 mg/min thereafter) (Fig. 327-3*A*). For cardiac arrest due to VF in the early phase of an acute coronary syndrome, a bolus of 1 mg/kg of lidocaine may be given intravenously as an alternative, and the dose may be repeated in 2 min. It also may be tried in patients in whom amiodarone is unsuccessful. Intravenous procainamide (loading infusion of 100 mg/5 min to a total dose of 500–800 mg, followed by continuous infusion at 2–5 mg/min) is now rarely used in this setting but may be tried for persisting, hemodynamically stable arrhythmias. Intravenous calcium gluconate is no longer considered safe or necessary for routine administration. It is used only in patients in whom acute hyperkalemia is known to be the triggering event for resistant VF, in the presence of known hypocalcemia, or in patients who have received toxic doses of calcium channel antagonists.

Cardiac arrest due to bradyarrhythmias or asystole (B/A cardiac arrest) is managed differently **(Fig. 327-3*B*)**. The patient is promptly intubated, CPR is continued, and an attempt is made to control hypoxemia and acidosis and identify other reversible causes. Epinephrine may be given intravenously or by an intraosseous route. Atropine is no longer considered effective for asystole or PEA, but can be used for bradyarrhythmias. External pacing devices are used to attempt to establish a regular rhythm when atropine fails for a bradyarrhythmia, but chronotropic agents given intravenously are now recognized as an equally effective alternative.

The success rate may be good when B/A arrest is due to acute inferior wall MI or to correctable airway obstruction or drug-induced respiratory depression or with prompt resuscitation efforts. For acute airway obstruction, prompt removal of foreign bodies by the Heimlich maneuver or, in hospitalized patients, by intubation and suctioning of obstructing secretions in the airway is often successful. The prognosis is generally very poor in other causes of this form of cardiac arrest, such as end-stage cardiac or noncardiac diseases. Treatment of PEA is similar to that for bradyarrhythmias, but its outcome is also dismal.

POST-CARDIAC ARREST SYNDROME AND POSTRESUSCITATION CARE

After return of spontaneous or stable assisted circulation, attention shifts to the diagnostic and therapeutic elements of the post-cardiac arrest syndrome. This recently developed clinical classification emerged from the organization of the elements of injury following cardiac arrest into a multidisciplinary continuum. The four components of post-cardiac arrest syndrome include brain injury, myocardial dysfunction, systemic ischemia/reperfusion responses, and control of persistent precipitating factors. The therapeutic goal is to maintain a stable electrical, hemodynamic, and central nervous system status.

Postresuscitation care is determined by the specific clinical circumstances. The most pressing is the presence of anoxic encephalopathy, which is a strong predictor of in-hospital death and postarrest disability. Mild therapeutic hypothermia is indicated for resuscitated cardiac arrest victims who are hemodynamically stable, but remain comatose. Core body temperature is decreased to 32–34°C, by several available techniques (external and/or internal [core]), as soon as practical after resuscitation and maintained for a minimum of 12–24 h. By reducing metabolic demands and cerebral edema, this intervention improves probability of survival with better neurologic outcome.

Primary VF in acute MI (not accompanied by low-output states) **(Chap. 295)** is generally very responsive to life support techniques and easily controlled after the initial event. In the in-hospital setting, respirator support is usually not necessary or is needed for only a short time, and hemodynamics stabilize promptly after defibrillation or cardioversion. In *secondary VF* in acute MI (those events in which hemodynamic abnormalities predispose to the potentially fatal arrhythmia), resuscitative efforts are less often successful, and in patients who are successfully resuscitated, the recurrence rate is high. The clinical picture and outcome are dominated by hemodynamic instability and the ability to control hemodynamic dysfunction. Bradyarrhythmias, asystole, and PEA are commonly secondary events in hemodynamically unstable patients.

The outcome after in-hospital cardiac arrest associated with noncardiac diseases is poor, and in the few successfully resuscitated patients, the postresuscitation course is dominated by the nature of the underlying disease. Patients with end-stage cancer, renal failure, acute central nervous system disease, and uncontrolled infections, as a group, have a survival rate of <10% after in-hospital cardiac arrest. Some major exceptions are patients with transient airway obstruction, electrolyte disturbances, proarrhythmic effects of drugs, and severe metabolic abnormalities, most of whom may have a good chance of survival if they can be resuscitated promptly and stabilized while the transient abnormalities are being corrected.

LONG-TERM MANAGEMENT AFTER SURVIVAL OF OUT-OF-HOSPITAL CARDIAC ARREST

Patients who survive cardiac arrest without irreversible damage to the central nervous system and who achieve hemodynamic stability should have diagnostic testing to define appropriate therapeutic interventions for their long-term management. This approach is driven by the fact that survival after out-of-hospital cardiac arrest is followed by a 10–25% mortality rate during the first 2 years after the event, and there are data suggesting that significant survival benefits can be achieved by prescription of an ICD.

Among patients in whom an acute ST elevation MI or transient and reversible myocardial ischemia is identified as the specific mechanism triggering an out-of-hospital cardiac arrest, the management is dictated in part by the transient nature of life-threatening arrhythmia risk during the acute coronary syndrome (ACS) and in part by the extent of permanent myocardial damage that results. Cardiac arrest during the acute ischemic phase is not an ICD indication, but survivors of cardiac arrest not associated with an ACS do benefit. In addition, patients who survive MI with an EF less than 30–35% appear to benefit from ICDs.

For patients with cardiac arrest determined to be due to a treatable transient ischemic mechanism, particularly with higher EFs, catheter interventional, surgical, and/or pharmacologic antiischemic therapy is generally accepted for long-term management.

Survivors of cardiac arrest due to other categories of disease, such as the hypertrophic or dilated cardiomyopathies and the various rare inherited disorders (e.g., right ventricular dysplasia, long QT syndrome, Brugada syndrome, catecholaminergic polymorphic VT, and so-called idiopathic VF), are all considered ICD candidates.

PREVENTION OF SCD IN HIGH-RISK INDIVIDUALS WITHOUT PRIOR CARDIAC ARREST

Post-MI patients with EFs <35% and other markers of risk such as ambient ventricular arrhythmias, inducible ventricular tachyarrhythmias in the electrophysiology laboratory, and a history of heart failure are considered candidates for ICDs 40 days or more after the MI. Total mortality benefits in the range of a 20–35% reduction over 2–5 years have been observed in a series of clinical trials. One study suggested that an EF <30% was a sufficient marker of risk to indicate ICD benefit, and another demonstrated benefit for patients with Functional Class 2 or 3 heart failure and EFs ≤35%, regardless of etiology (ischemic or nonischemic) or the presence of ambient or induced arrhythmias **(Chaps. 277 and 279)**. For patients with newly diagnosed heart failure and an EF <35%, the required delay between diagnosis and institution of medical therapy, and subsequent implantation of an ICD, is 90 days. In general, there appears to be a gradient of increasing ICD benefit with EFs ranging lower than the threshold indications. However, patients with very low EFs (e.g., <20%) may receive less benefit.