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Management of the Patient Who Has Been Resuscitated From Sudden Cardiac Death

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D is a 68-year-old man who had an anterior myocardial infarction 6 months ago. Evaluation at that time demonstrated an occluded left anterior descending artery, which was successfully stented with no other significant lesions. Left ventricular ejection fraction was 35%. He had no recurrent angina or heart failure symptoms. Early one morning he lost consciousness. Paramedics verified ventricular fibrillation (VF) and provided effective defibrillation within 5 minutes; spontaneous circulation returned before transfer to the hospital.

Sudden Cardiac Death: Scope of the Problem

Sudden cardiac death claims 300,000 to 450,000 lives a year in the United States and represents approximately 50% of all cardiac death.1–3 Despite recent declines in age-adjusted cardiac mortality and sudden death risk, the overall incidence has remained relatively stable as our population ages.3 Coronary heart disease is present in the majority (70% to 80%) of patients with sudden death, but cardiac arrest is the first manifestation of this underlying process in 50%.4 Resuscitation rates are very low, averaging from 1% to 3% in most major cities.5 There are some data to suggest that focused attempts to reduce the time to effective defibrillation, with improved training of first responders or more widespread availability of automatic external defibrillators, provide some hope for the future.

It is a widely held belief that the majority of sudden death events are due to ventricular tachycardia (VT) that degenerates into VF (Figure 1).6 This idea may well reflect our greater experience in observing patients with structural heart disease and prior myocardial infarction. Acute severe ischemia may cause primary polymorphic VT even in the absence of preexisting structural heart disease. Autopsy studies document acute changes in coronary morphology (such as plaque rupture, thrombus, etc) in >50% of sudden death events.7 Severe bradyarrhythmias or electromechanical dissociation probably represent an important cause in patients with very advanced structural heart disease; the prospects for resuscitation from these arrhythmias are even worse than for patients that present with ventricular tachyarrhythmias.

Our current understanding of the sudden death syndrome reflects a variable contribution of “pure” electrophysiological abnormalities and functional triggers (ischemia, electrolyte imbalances, autonomic nervous system input, proarrhythmic effects of drugs, emotional stress, etc).4 This complex interaction between anatomic and functional substrates obviously differs for patients with different underlying cardiac disease states. The multiplicity of influences that results in a cardiac arrest episode and our incomplete understanding of the pathophysiology of the syndrome make preventive approaches for secondary prevention far from completely successful. This realization, as well as the high risk of recurrences, forms the conceptual support for our reliance on implantable defibrillator therapy in this setting.

Evaluation of the Sudden Death Survivor

After the initial resuscitation of the patient, recurrent ventricular arrhythmias are unusual and acute antiarrhythmic therapy, though widely practiced, has little foundation. Rarely, patients will present with “arrhythmia storm,” with frequently recurring VT or VF. The prognosis in this situation is poor, and aggressive correction for acute ischemia or hemodynamic support may be helpful; intravenous amiodarone is the drug of choice in this setting.8 Because the arrest mechanism, treatment goals, and prognosis vary depending on the underlying heart disease, a complete evaluation for heart disease should be performed in all arrest survivors. At a minimum, this should consist of a 12 lead ECG (ischemia, QT interval), an evaluation of left ventricular function, and coronary angiography.
Sudden Death in Patients With Preexisting Structural Heart Disease

In patients with preexisting structural heart disease, particularly healed myocardial infarction, the preponderance of evidence supports ventricular arrhythmias as the underlying cause of sudden death (Figure 1). Although functional triggers should be evaluated and corrected, the arrest should be “blamed” on the underlying heart disease (and the anatomic arrhythmia substrate that it provides), rather than “correctable causes.” Patients with VT/VF associated with a “transient/correctable” cause included in the observational registry of the Antiarrhythmics Versus Implantable Defibrillators Trial (AVID) were shown to have a mortality rate of 17.8% in 16.9±11.5 month follow-up; this risk was similar to the general group of ventricular fibrillation arrest survivors. Several studies suggest that coronary revascularization provides important antiarrhythmic benefits. However, coronary revascularization alone provides incomplete protection from recurrent sudden death in this high risk patient group.

Three multicenter studies addressed the use of an implantable cardioverter defibrillator (ICD) versus antiarrhythmic drug therapy in secondary prevention of sudden cardiac death: the Canadian Implantable Defibrillator Study (CIDS), the Cardiac Arrest Study, Hamburg (CASH), and AVID. Although the trials had slightly different enrollment and treatment strategies, taken together they demonstrated that ICD therapy is superior to best antiarrhythmic drug therapy (predominantly amiodarone) in patients with structural heart disease who have had sustained ventricular arrhythmias. In AVID, total mortality at 2 years was 25% in the antiarrhythmic group and 18% in the ICD group (27% reduction, P=0.02). The impact of the survival benefit provided by ICD therapy, whether it applies to all patient subgroups, and whether it would stand with more complete therapy for ischemia and ventricular dysfunction, remain open questions. Nonetheless, ICDs are the therapy of choice for secondary prevention of sudden cardiac death in patients with preexisting heart disease.

Sudden Death Precipitated by Acute Coronary Ischemia/Infarction

In patients with objective evidence of acute ischemia who do not have preexisting heart disease, the presumptive cause is ischemic polymorphic VT (Figure 2). Autopsy studies imply that plaque rupture and acute occlusion are frequent causes of sudden death, but acute infarction is present in only 20% of arrest survivors. This argues either for the interaction of ischemia and electrophysiologic substrate, or for the fact that survival in the setting of severe ischemia is unlikely unless the arrest occurs in the hospital setting where resuscitation is immediate.

Coronary revascularization alone has been shown to prevent recurrent VT/VF in patients who have no previous structural heart disease and who experience cardiac arrest in the setting of acute ischemia. A negative response to programmed stimulation after surgery may be helpful in defining this group further.

Contradictory data exist regarding the management of patients who sustain ventricular arrhythmias in the peri-infarct period. In patients who received thrombolytic therapy, primary VF within the first 48 hours did not increase post-hospital risk of sudden death. The AVID registry data appear to conflict with this observation, but most of these patients had significant left ventricular dysfunction. If the index event results in Q-wave infarction, most authorities would consider the risk of recurrent VT/VF significant. The logic of this opinion centers on the “which came first” conundrum, as well as our understanding of the interaction of the now present anatomic substrate and ischemic trigger (which could redevelop despite revascularization).

Sudden Death in Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy represents a spectrum of genetic disease in which the structural abnormalities of hypertrophy and the risk of ventricular arrhythmia may be discordant. Observational studies suggest that β-blockers

Figure 1. Holter monitor tracings recorded during a sudden death episode. The initial arrhythmia is uniform ventricular tachycardia that continues for several minutes and degenerates into ventricular fibrillation and then asystole. Courtesy of Guidant Corporation educational files, with permission.

Figure 2. Surface ECG recording of ischemic polymorphic ventricular tachycardia in a patient without ventricular dysfunction. Note the anterior ST elevation in the first 3 sinus beats. The first beat of the tachycardia arises from the crest of the T wave, and the ensuing arrhythmia is disorganized from the onset.
or amiodarone provide insufficient therapy for recurrent sudden death risk, although direct comparisons with ICD therapy are lacking.22

Sudden Death in Patients Without Apparent Heart Disease
Approximately 5% of arrest victims have no demonstrable structural heart disease.23 Among many potential causes, 3 important etiologies should be considered. Congenital and acquired (by effects of drugs, often antiarrhythmic drugs) long QT syndrome can result in torsades de pointes type polymorphic VT.24 Therapeutic options include removal of all QT prolonging drugs, pacing, use of β-blockers, and ICD therapy, depending on the details of the presentation. The Brugada syndrome is recognized by ST segment elevation in the anterior precordial leads, exaggerated by type I antiarrhythmic agents, RBBB conduction pattern, and sudden cardiac death. It is a heritable disease caused by a mutation of the SCN5A cardiac sodium channel gene. ICD therapy is typically recommended for survivors of cardiac arrest because antiarrhythmic drugs have been demonstrated to be ineffective.25

Idiopathic ventricular fibrillation occurs in the absence of both structural heart disease and the electrophysiologic abnormalities discussed above. Risk of events is estimated to be 30% at 3 years, and ICD therapy is usually recommended.23

Post Hoc Application of Primary Prevention
ICD therapy provides the most appropriate treatment for recurrent ventricular tachyarrhythmias in most of the settings discussed above. This is not to imply that other forms of treatment are not useful in the overall care of the arrest survivor. Most of the interventions noted have been evaluated in trials of primary prevention of total cardiac or sudden death;26 however, extension of their findings to sudden death survivors seems reasonable. Coronary revascularization appears to reduce total and sudden death mortality and should be strongly considered when appropriate.11–14 The influence of ischemia triggers on the electrophysiologic substrate provides a strong argument for aggressive lipid management, particularly for the benefit of plaque stabilization. The beneficial effect of angiotensin-converting enzyme inhibitors in ventricular dysfunction is indisputable; studies vary as to the specific effect on sudden death. Multiple studies after myocardial infarction27 and in patients with congestive heart failure28 have demonstrated the benefit of β-blocker therapy; this therapy is also important in arrhythmia prevention in the setting of long QT syndrome and hypertrophic cardiomyopathy. Although these precautions seem obvious, a review of many secondary prevention trials demonstrates inadequate use of such important general therapies for cardiovascular disease.

Summary
Advances in contemporary therapy, particularly in ICD technology, have resulted in important reductions in subsequent mortality for patients experiencing sudden cardiac death. Nonetheless, the “holy grail” of electrophysiology, the successful primary prevention of sudden cardiac death, has proven elusive. Again, although ICD therapy has proven effective in certain well-defined high-risk subgroups,29 the majority of sudden death occurs in patients not known to be at high risk.30 Further advancements in primary prevention will depend on a more complete understanding of the pathophysiology of sudden cardiac death.

References

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