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Risk Stratification and Treatment for Sudden Cardiac Death

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Table of Contents

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next page

Risk Stratification and Treatment for Sudden Cardiac Death

George M. Soliman, MD, and Samuel C. Dudley, Jr., MD, PhD

I. INTRODUCTION

Cardiovascular disease remains among the most common causes of death in developed nations despite significant advances during the past 30 to 40 years. Sudden death from cardiac problems accounts for 50% of all deaths from cardiovascular causes and for 300,000 to 400,000 deaths in the United States annually.[1,2] The most common initiating event for sudden cardiac death (SCD) is a ventricular tachyarrhythmia that may occur with or without previously known structural heart disease.[2,3] The typical sequence of arrhythmias leading to SCD is ventricular tachycardia (VT) that degenerates to ventricular fibrillation followed by asystole. Acute myocardial ischemia is considered to be the most common trigger of fatal arrhythmias.[4] The next most frequent setting for SCD is the presence of myocardial scarring, usually resulting from previous infarction. Nonischemic cardiomyopathy also is associated with SCD. In the absence of underlying heart disease, SCD commonly results from polymorphic VT and torsades de pointes caused by congenital or acquired long-QT syndrome or by hypertrophic cardiomyopathy. Despite an emphasis on tachycardia, bradyarrhythmias and electromechanical dissociation are recognized as the initial event in SCD, especially in patients with advanced heart disease.[5,6] The most common causes of SCD are listed by age group in **Table 1**.

MECHANISMS OF TACHYARRHYTHMIAS

Three basic mechanisms seem to be responsible for tachyarrhythmias. Reentry, which is thought to be the most common mechanism, is one or more "short circuits" that produce continuous electrical activity and disorganized contraction without adequate ventricular filling or ejection. Reentry requires at least a transient unidirectional conduction block, and it is favored by slow and heterogeneous conduction that occurs transiently after a premature ventricular contraction or permanently after a myocardial infarction (MI). Other causes of tachyarrhythmia include enhanced normal or abnormal automaticity and triggered activity. Ischemia promotes all 3 mechanisms. Recent evidence suggests that gene regulation events in cardiomyopathy may contribute to risk by prolonging the action potential or by reducing the abnormal currents needed to initiate arrhythmias.[7] In order to prevent sudden death, it will be important to refine tests that will predict those at risk so primary prevention can be instituted. Some commonly recognized predictors of SCD are listed in **Table 2**.

CLINICAL TRIALS

Recent randomized clinical trials have improved our understanding of the likelihood and predictability of SCD. Although great advances have been made in the treatment of high-risk patients, the effect on the overall incidence of SCD has been relatively small because most SCD occurs among persons who would not fit in the high-risk populations that have been studied. **In other words, the current tests still lack positive predictive value for moderate-risk to low-risk patients.** In this article, we will use 2 clinical scenarios to review the state of the art in risk stratification and treatment for SCD.

II. CASE PATIENT 1

PRESENTATION

Patient 1 is a 64-year-old man with a history of diabetes, hypertension, and tobacco abuse who presents with complaints of chest pain consistent with angina. The symptoms began with exertion 3 days before presentation. On the day of presentation, he experienced severe chest discomfort at rest, and he was brought by ambulance to the emergency department. Physical examination reveals a blood pressure (BP) of 145/92 mm Hg, a heart rate of 98 bpm, and an audible S_4 gallop; jugular venous distention or rales are not noted. He is in moderate distress and is diaphoretic. The electrocardiogram (ECG) during this episode shows normal sinus rhythm with left-axis deviation, a large R-wave in precordial lead V_1, Q-waves in the inferior leads consistent with an age-indeterminate inferior MI, 1-mm ST depression, and lateral T-wave inversion.

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