Prediction and Prevention of Sudden Cardiac Death

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KEYWORDS

• Sudden cardiac death • Sudden cardiac arrest • Ischemic heart disease
• Nonischemic cardiomyopathy • Left ventricular ejection fraction

KEY POINTS

- As the most common cause of death worldwide, sudden cardiac death (SCD) has important implications for not only individuals, but for entire populations.
- The most common underlying abnormality associated with SCD is ischemic heart disease, but several other pathophysiological processes (both inherited and acquired) can also predispose to SCD.
- Methods of risk stratification for SCD, and treatments aimed at reducing that risk, have been developed.
- The definitive therapy for most patients at high risk for SCD is the implanted cardioverter-defibrillator (ICD).
- Following the recently published DANISH study, the utility of ICDs in nonischemic cardiomyopathy (NICM) has had renewed interest, and risk stratification in NICM is an area of active investigation.

INTRODUCTION

Sudden cardiac death (SCD), defined as death due to cardiac causes heralded by abrupt loss of consciousness, is the most common cause of death worldwide, accounting for 50% of deaths from cardiovascular disease and approximately 350,000 annual deaths in the United States.[1–6] Although the terms sudden cardiac arrest (SCA) and SCD are commonly used interchangeably, by definition SCA is the sudden cessation of cardiac activity so that the victim becomes unresponsive, with no normal breathing and no signs of circulation.[1] If definitive measures are not taken rapidly, SCA progresses to SCD.[1] Many of these events can be predicted and prevented by implementing evidence-based, guideline-endorsed recommendations for primary or secondary prevention of SCD.[1–6] Risk stratification techniques now allow identification of individuals at risk for SCD because of structural heart disease or inherited channelopathies (**Box 1**).[1–6] Several cardiovascular conditions predisposing to athletic sudden death have been identified.[7–9] Multiple interventions have been identified that can reduce the risk of SCD in these patient populations.[1–9]

Despite contemporary risk stratification techniques, prediction and prevention of SCD represent major challenges, because most SCD events occur in patients who were not previously identified as being at risk for SCD.[1–6] Because most individuals experiencing SCD currently are not identifiable as being at high risk, community-based public access to defibrillation programs is

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Box 1
Conditions associated with sudden cardiac death

- Structural heart disease
  - Ischemic heart disease
  - Nonischemic cardiomyopathy
  - Valvular heart disease
  - Congenital heart disease
  - Hypertrophic cardiomyopathy
  - Arrhythmogenic right ventricular dysplasia
  - Anomalous coronary artery origin
- Primary electrophysiological conditions
  - Congenital long QT syndromes
  - Short QT syndrome
  - Ventricular pre-excitation (Wolff-Parkinson-White syndrome)
  - Idiopathic ventricular fibrillation
  - Brugada syndrome
  - Catecholaminergic polymorphic ventricular tachycardia

essential to save lives and improve outcomes for cardiac arrest victims.

ISCHEMIC HEART DISEASE

Several strategies have evolved to predict and prevent SCD in patients with ischemic heart disease.[1–6] Primary prevention of coronary artery disease (CAD), the most common condition predisposing to SCD, is 1 approach.[1–6] SCD is the first manifestation of CAD in approximately 40% of patients. Risk factors for both CAD and SCD include advanced age, male sex, cigarette smoking, hypertension, diabetes mellitus, hypercholesterolemia, obesity, and a family history of premature CAD.[1,6] These risk factors for SCD are also predictors of myocardial infarction, CAD-related death, and all-cause mortality.[1,6] Lifestyle-based optimization of blood pressure, weight, glucose, cholesterol, smoking, diet, and physical activity is included in this strategy.[1,6] Although this approach is intuitively appealing, evidence showing that it directly prevents SCD does not currently exist.[1,6]

Primary prevention of SCD in patients with CAD, via risk stratification and pharmacologic intervention, revascularization, and/or the implantable cardioverter defibrillator (ICD), represents a strategy supported by multiple clinical trials.[1–6] Impaired left ventricular ejection fraction (LVEF) and other genetic, anatomic, and electrophysiological risk factors for SCD have been identified.[1–6] Pharmacologic interventions that reduce the risk of SCD in patients with impaired LVEF and CAD include beta-blockers, angiotensin-converting enzyme inhibitors, and statins.[6] Antiarrhythmic agents for suppression of ventricular arrhythmias have a neutral or negative effect on mortality based on prospective, randomized trials.[6] Multiple clinical trials randomizing several thousand patients have demonstrated that the ICD reduces SCD and improves overall mortality in selected patient populations, including those with ischemic heart disease and left ventricular dysfunction.[1,6]

Secondary prevention of SCD refers to interventions in patients who have survived a prior cardiac arrest or sustained ventricular tachyarrhythmia.[1–6] Multiple prospective randomized trials have shown the ICD to be superior to antiarrhythmic drug therapy for prolonging survival in such patients.[1–6] All recommendations for ICD therapy apply only to patients who are receiving optimal medical therapy (OMT) and have a reasonable expectation of survival with good functional status for 1 year.[1–6] When indicated for primary or secondary prevention, ICDs have been demonstrated to reduce SCD and improve total mortality, with favorable cost-effectiveness.[1–6] Despite this evidence, many patients who have indications for this therapy are not receiving ICDs.[1–6]

Prevention of SCD immediately after a myocardial infarction (MI) represents a vexing clinical challenge.[6] With advances in the treatment of MI with primary percutaneous coronary intervention and pharmacologic therapy, SCD and total mortality after MI have decreased.[6] Despite optimal therapy with revascularization and drugs, the risk of SCD is highest in the first 30 days.[6] However, clinical trials have demonstrated no mortality improvement from early ICD placement in patients at even extremely high risk for SCD after MI.[6] Autopsy evaluation of patients experiencing SCD in the immediate post-MI period has demonstrated that there is a high frequency of cardiac rupture or recurrent MI in the first month after the index MI, whereas arrhythmic death becomes more likely subsequently.[6] These observations may help to explain the lack of benefit of early ICD therapy after MI.[6]

Contemporary clinical guidelines restrict ICD implants to patients at least 40 days after MI with continued impairment of LVEF despite OMT.[1–6] The home automated external defibrillator in high-risk post-MI patients does not improve survival compared with conventional resuscitation methods.[1–6] Based on these considerations, clinicians commonly employ the wearable cardioverter defibrillator (WCD).[5] Although the short-term use of the WCD is a reasonable approach for high-risk