The Changing Epidemiology of Sudden Cardiac Death

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KEYWORDS

• Sudden cardiac death • Cardiac arrest
• Cardiovascular disease • Global health • SCD risk

Sudden cardiac death (SCD) is a devastating complication of myocardial infarction (MI). Long-term population studies outlining the incidence of SCD after MI in the community are decades old.[1–3] The in-hospital mortality after MI has decreased substantially, and the long-term prognosis after MI has improved greatly, yielding a growing number of MI survivors at risk for sudden cardiac death.[4,5] Patterns of disease progression predict a globally increased incidence of heart disease by 2020.[6–8] Already in the first decade of the new millennium, these predictions are becoming a reality. In the developing world, the greatest increases in prevalence of diabetes, coronary artery disease and heart failure are being seen.[9] As a consequence of the increased prevalence of coronary artery disease and heart failure will be an increasing incidence of SCD. As a result, SCD will manifest as a shared worldwide public health problem. The purpose of this article is to summarize SCD epidemiology, with a focus on the anticipated global rise in incidence.

WHAT IS SCD?

The most widely accepted definition of SCD is the sudden and unexpected death within an hour of symptom onset.[10] Despite a substantial overall decline in morbidity and mortality caused by cardiovascular diseases in the second half of the last century, the survival after an episode of SCD remains in the range of 5%.[11] The sudden nature of this condition, and its occurrence in many instances without warning, are major impediments to improving outcome. Awareness of this important deficiency has led to considerable interest on mechanisms of SCD. Despite a renewed focus, the significant delay in development of effective measures for risk stratification and prevention of SCD can be attributed directly to a poor understanding of mechanisms involved in fatal arrhythmogenesis.[12]

There are several important factors contributing to the challenges faced when assessing true rates of SCD. SCD occurs in the general population in an unpredictable manner, and whenever possible, it is crucial to exclude subjects who are likely to have had a noncardiac cause of sudden death. Additionally, an accurate estimate of SCD incidence requires prospective ascertainment of cases. Studies that have used retrospective analysis of the death certificate to identify cases of SCD likely overestimate SCD incidence by as much as 200% to 300%.[13] Therefore, the US estimates published by the US Centers for Disease Control and Prevention (CDC) (400 to 450 000 per year)[3] are likely significant overestimates.[14]

ARRHYTHMIC MECHANISMS OF SCD

In the last 30 years, there have been important alterations in the prevalence trends of arrhythmias causing SCD (**Fig. 1**). Early studies reported that ventricular fibrillation (VF) was the initial rhythm in

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most SCD cases—75% in a longitudinal study from Seattle[15] and 84% in a series of patients wearing Holter monitors.[16] Despite the spectrum of etiologic conditions, in most cases, SCD results from either ventricular tachycardia (VT)/VF or severe bradycardia/pulseless electrical activity (PEA).[17,18] Of the lethal arrhythmias, VF consistently has been shown to have the highest likelihood of survival. As a result, there has been great emphasis on VF detection and treatment in resuscitation protocols. Since the first descriptions of external defibrillation in the 1960s and the first human implantable cardioverter defibrillator (ICD) in 1980 by Mirowski,[19] the paradigm for the prevention of SCD shifted away from antiarrhythmic drug therapy. Since 1990, however, a declining rate of VF and a rise in prevalence of PEA have been reported (**Fig. 2**). This was reported initially in in-hospital studies and later confirmed in prehospital studies. Data from Seattle from 1990 to 1996 indicated that 41% of SCDs were attributable to VF and 24% to PEA.[20] Another study from Seattle that looked at prevalence trends between 1980 and 2000 reported a 56% decrease in VF as the first identified rhythm from 1980 to 2000 (from 0.85 to 0.38 per 1000 population; relative risk [RR], 0.44; 95% confidence interval [CI], 0.37 to 0.53).[21] Similar reductions were seen in blacks and whites, and the phenomenon was most evident in men (57%; RR, 0.43; 95% CI, 0.35 to 0.53), in whom the baseline incidence was relatively high. An amiodarone versus lidocaine trial reported VF prevalence among prehospital cardiac arrests of only 26%.[22] Similar reports were published from Sweden, documenting a decrease in prevalence of VF by 39% over 17 years despite significant improvements in response time and rates of bystander cardiopulmonary resuscitation.[23] Prevalence of PEA among sudden cardiac arrest cases rose from 6% to 26% from 1980 to 1996.[23] From the mechanistic standpoint, as VF more commonly is related to coronary disease and PEA to noncardiac factors,[24] the significant decrease in age-adjusted mortality from coronary artery disease over the last 50 years[25] may be responsible in part for the decline seen in VF prevalence. As the survival rates from these arrhythmias is markedly different, ongoing investigation focused on improving the understanding of PEA mechanisms and treatment is underway, with the aim of improving current rates of survival from sudden cardiac arrest in the community.

WHO IS AT RISK FOR SCD?

SCD is the consequence of complex interaction between genetic elements, various cardiac conditions, comorbidities, and epidemiologic and environmental factors (**Fig. 3**). Approximately 80% of SCDs are attributed to coronary artery disease.[10,14,26] The two major mechanisms of fatal ventricular arrhythmias include acute coronary ischemia, usually as a result of plaque rupture, and occlusion of an epicardial coronary artery and re-entry associated with areas of slow conduction and previous myocardial scarring. The former is more likely to result in polymorphic VT or VF and may occur in individuals with preserved left ventricular (LV) function. The latter occurs in the setting of an ischemic

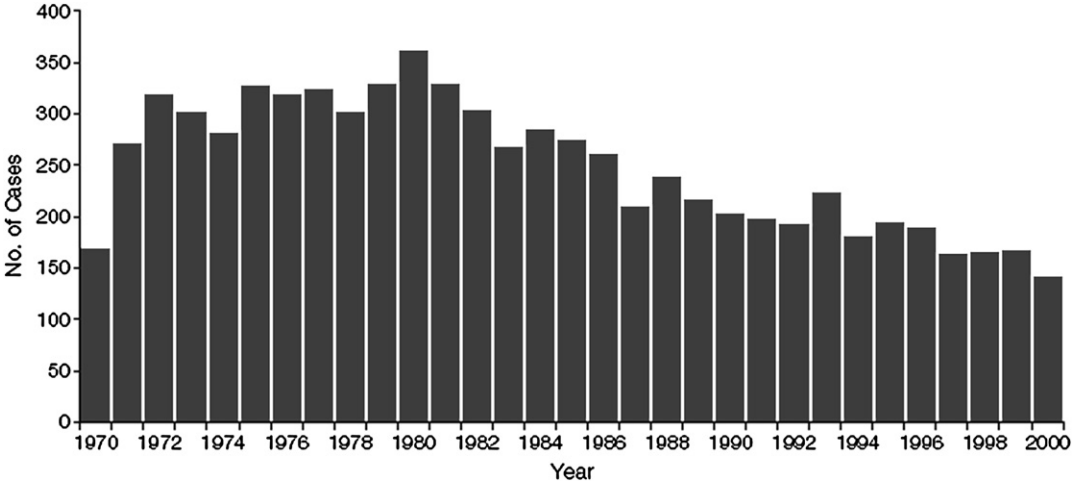

Fig. 1. Annual numbers of all patients treated for out-of-hospital ventricular fibrillation from 1970 to 2000 in Seattle, Washington. (*Modified from* Cobb LA, Fahrenbruch CE, Olsufka M, et al. Changing incidence of out-of-hospital ventricular fibrillation, 1980–2000. JAMA 2002;288:3008; with permission.)