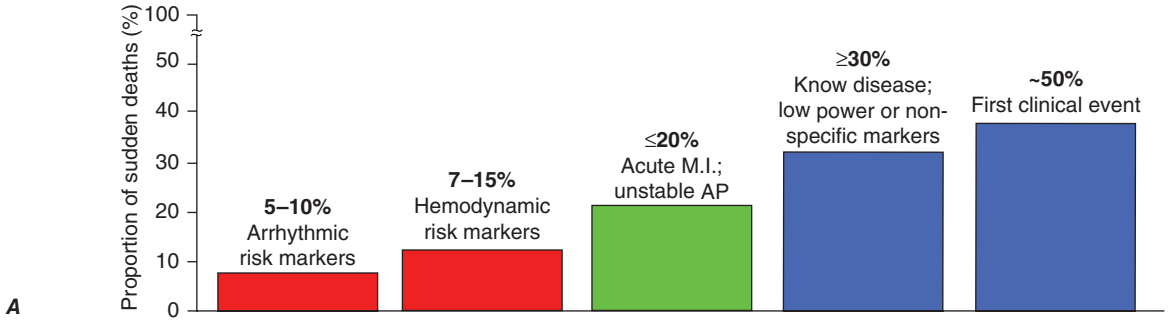

B

Target	*Examples*	*Goal*	*Sensitivity*
• ASHD risk factors	• Framingham risk index	• Predict evolution of disease	• Very low
• Anatomic screening	• CT imaging	• Identify CAD	• Moderate for anatomy
• Clinical markers	• EF; angiography	• Define extent of disease	• High for extent of disease; variable for specificity of risk
	• AM; EPS		
	• History of heart failure	• Identify arrhythmia markers	• Low-to-intermediate for screening
	• EP and hemodynamic variations	• Define high-risk subgroups	• High for specific groups
• Transient risk predictors	• Autonomic fluctuations	• Clinical markers of instability	• Primary predictive value unknown
	• Predictors of ischemia	• Quantify autonomic triggers	• Uncertain; some measures useful (?)
	• Familial/genetic profiles	• Predict unstable plaques	• Unknown; potentially high
• Individual risk predictors		• Predict specific SCD risk before disease expression	• High potential for future profiling

FIGURE 327-2 Population subsets, risk predictors, and distribution of sudden cardiac deaths (SCDs) according to clinical circumstances. *A.* The population subset with high-risk arrhythmia markers in conjunction with low ejection fraction is a group at high risk of SCD but accounts for <10% of the total SCD burden attributable to coronary artery disease. In contrast, 50% of all SCD victims present with SCD as the first and only manifestation of underlying disease, and up to 30% have known disease but are considered relatively low risk because of the absence of high-risk markers. ***B.*** Profiling for individual prediction and prevention of SCD is difficult. The highest absolute numbers of events occur among the general population who may have risk factors for coronary heart disease or expressions of disease that do not predict high risk. This results in a low sensitivity for predicting and preventing SCD. New approaches that include epidemiologic modeling of transient risk factors and genetic predictors of individual patient risk offer hope for greater sensitivity in the future. AM, ambulatory monitoring; AP, angina pectoris; ASHD, arteriosclerotic heart disease; CAD, coronary artery disease; CT, computed tomography; EF, ejection fraction; EP, electrophysiologic; EPS, electrophysiologic study; MI, myocardial infarction. *(Modified from RJ Myerburg: J Cardiovasc Electrophysiol 12:369–381, 2001.)*

Among patients in the acute, convalescent, and chronic phases of MI (Chap. 295), subgroups at high absolute risk of SCD can be identified. During the acute phase, the potential risk of cardiac arrest from onset through the first 48 h used to be as high as 15%, but is now reported in the range of 2.3–4.4% because of early patient awareness of the significance of symptoms and the availability of emergency revascularization strategies. Those who survive acute-phase VF are not at continuing risk for recurrent cardiac arrest indexed to that event. During the convalescent phase after MI (3 days to ~6 weeks), an episode of sustained ventricular tachycardia (VT) or VF, which is usually associated with a large infarct, predicts a natural history mortality risk of >25% at 12 months. At least one-half of the deaths are sudden. Aggressive intervention techniques may reduce this incidence.

During the chronic phase after MI, the longer-term risk for total mortality and SCD mortality is predicted by a number of factors (Fig. 327-2*B*). The most important for both SCD and nonsudden death is the extent of myocardial damage sustained as a result of the acute MI. This is measured by the magnitude of reduction of the ejection fraction (EF) and/or the occurrence of heart failure. Various studies have demonstrated that ventricular arrhythmias identified by ambulatory monitoring contribute significantly to this risk, especially in patients with an EF <40%. In addition, inducibility of VT or VF during electrophysiologic testing of patients who have ambient ventricular arrhythmias (premature ventricular contractions [PVCs] and nonsustained VT) and an EF <35% is a strong predictor of SCD risk. Patients in this subgroup are now considered candidates for ICDs (see below). Risk falls off sharply with EFs >35% and the absence of ambient arrhythmias after MI, and conversely is high with EFs <30% even without the ambient arrhythmia markers.

The cardiomyopathies (dilated and hypertrophic, Chap. 287) are the second most common category of diseases associated with risk of SCD (Table 327-2). Some risk factors have been identified, largely related to extent of disease, presence of heart failure, documented ventricular arrhythmias, and syncope thought to be due to arrhythmias. The less common causes of SCD include valvular heart disease (primarily aortic) and inflammatory and infiltrative disorders of the myocardium. The latter include viral myocarditis, sarcoidosis, and amyloidosis.

Among adolescents and young adults, rare inherited disorders such as hypertrophic cardiomyopathy, the long QT interval syndromes, right ventricular dysplasia, and the Brugada syndrome have received attention as important causes of SCD, as has acute myocarditis and other less common acquired diseases. Among the subgroup of young competitive athletes, the incidence of SCD may be higher than it is for the general adolescent and young adult population, perhaps up to 1 in 75,000–100,000. Hypertrophic cardiomyopathy (Chap. 287) is the most common cause in the United States.

Secondary prevention strategies should be applied to survivors of cardiac arrest that was not associated with an acute MI or other controllable transient risk factors, such as certain drug exposures and correctable electrolyte imbalances. Multivessel coronary artery disease and dilated cardiomyopathy, especially with markedly reduced left ventricular EF, predict a high risk of recurrence of cardiac arrest or SCD and are indications for specific interventions, such as ICDs (see