The activity of the autonomic nervous system exerts a pervasive influence on cardiovascular health. For many major forms of cardiovascular disease, evidence has been accrued to implicate neurogenic factors either as acute triggers or as long-term catalysts or both. Among the most prominent conditions with an underlying neural component exacerbating cardiovascular dysfunction are hypertension, heart failure, coronary artery disease, and arrhythmias (1). The underlying mechanisms are multifactorial and complex. There are both direct and intermediary factors responsible for the adverse influence of disturbed autonomic activity on cardiovascular health. These range from perturbations in hemodynamic function, particularly elevated arterial blood pressure, and direct effects of neurotransmitters on the myocardium and vascular endothelium.

The goals of this brief review are to summarize the current knowledge regarding sympathetic and parasympathetic neural influences on cardiac electrical stability and to discuss the evidence which implicates behavioral stress as a bona fide risk factor in cardiac events. We will focus primarily on propensity to arrhythmias, because of the relevance of ambulatory ECG monitoring to the audience and the fact that the basic principles have been elucidated by research on this topic.

SYMPATHETIC NERVE INFLUENCES

Sympathetic nerve activation has been extensively implicated in provoking life-threatening arrhythmias both in animals and humans (2,3). Stimulation of
TABLE 1

DIRECT CARDIAC ELECTROPHYSIOLOGIC EFFECTS OF SYMPATHETIC NERVOUS SYSTEM STIMULATION

- Shifts pacemaker from sinus node to junctional region
- Alters P-wave morphology
- Abbreviates P-R interval
- Increases Purkinje fiber automaticity
- Increases early afterdepolarizations
- Prolongs QT-interval on body surface
- Increases TQ-depression and enhances reentry during acute myocardial ischemia
- Decreases ventricular fibrillation threshold
- Induces T-wave alternans in the long QT-syndrome and during acute myocardial ischemia


The mechanisms whereby enhanced sympathetic nerve activity increases cardiac vulnerability in the normal and ischemic heart are complex (Table 1).