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Impact of Exercise Training on Psychological Risk Factors

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Abstract Although the role of psychological risk factors has been underemphasized, considerable evidence indicates the adverse effects of various psychosocial stressors in the pathogenesis and recovery from cardiovascular diseases. Substantial data, especially from cardiac rehabilitation and exercise training programs, have demonstrated the role of physical activity, exercise training, and cardiorespiratory fitness, to improve psychological risk factors, including depression, anxiety, hostility, and total psychological stress, as well as stress-related mortality. (Prog Cardiovasc Dis 2011;53:464-470)

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> "It is exercise alone that supports the spirit, and keeps the mind in vigor." – Cicero[1]

There is considerable evidence that various psychosocial stressors play a role in cardiovascular (CV) health.[2,3] Despite widespread belief by the general public that psychological stress (PS) leads to various medical ailments, including CV diseases (CVDs), the medical community has not fully embraced this conclusion. Clearly, throughout medicine, "stress" is one of the most common complaints from patients,[4] and PS has negative impact on many chronic diseases, including diabetes, cancer, rheumatoid arthritis, chronic obstructive pulmonary disease, human immunodeficiency virus, and CVD.[5]

The purposes of this article are to briefly review the epidemiologic and clinical data on the relationship between PS and CVD and to review the potential impact of physical activity, exercise training (ET), and increased cardiorespiratory fitness (CRF) to reduce PS and to reduce PS-related CVD events and mortality.

Does PS increase CVD?

Although the lay public has generally been extremely concerned about the impact of PS in general and CV health, this topic has been underemphasized by clinicians, especially in the field of CVD prevention and treatment. In a recent major review, PS was defined to exist when an individual perceives that environmental demands tax or exceed his or her adaptive capacity.[6] The link between PS and CVD has clearly been identified as an important public health concern.

The body of data exploring the complex relationship between PS and CVD comes primarily from epidemiologic and prospective studies that have explored laboratory responses to acute stressors with long-term follow-up of subjects for major CVD events. The presence of high levels of PS, assessed by validated screening instruments, has been associated with incident CVD in many cohort studies, with clinical impact that seems to be comparable with many standard CVD risk factors, including smoking, hypertension, obesity, and physical inactivity.[7]

Statement of Conflict of Interest: see page 469.

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Abbreviations and Acronyms

CHD = coronary heart disease

CRET = cardiac rehabilitation and exercise training

CRF = cardiorespiratory fitness

CV = cardiovascular

CVD = cardiovascular diseases

ET = exercise training

HF = heart failure

MI = myocardial infarction

PS = psychological stress

VO_2 = oxygen consumption

Although most of the literature in PS has focused on depression as a CVD risk factor, there is substantial evidence that other psychosocial risk factors, including anxiety, hostility, isolation, time urgency, strong adverse emotions, and total PS, have significant and important acute and chronic effects on CVD prognosis.[2-4,7] Although precisely how PS effects CV prognosis is an area of intense debate and research, considerable existing evidence indicates that psychosocial factors are strong risk factors for CVD and may adversely effect recovery after major CVD events, especially from coronary heart disease (CHD; Fig 1).[2] Practical issues certainly may be involved, such as engaging in self-destructive behaviors like substance abuse, noncompliance with prescribed medical regimens, or failure to follow prudent nonpharmacologic recommendations (eg, smoking cessation, diet, and exercise and other behaviors like social isolation and failure to get adequate sleep). However, PS also directly disturbs hormonal homeostasis, which may result in insulin resistance, inflammation, hypertension, hyperglycemia, and other metabolic disorders and lead to endothelial dysfunction, a common denominator in CVD.[2,7]

Probably the most impressive data to date on the role of PS and CHD come from the INTERHEART study,[8] which evaluated CHD risk factors and psychosocial stress among a diverse group of 11,119 myocardial infarction (MI) patients and 13,648 age- and sex-matched controls from 262 centers in 52 countries. In this landmark study, among the 9 major modifiable CVD risk factors, PS was behind only lipids and smoking in importance, and PS accounted for fully one-third of the total attributable risk.[8]

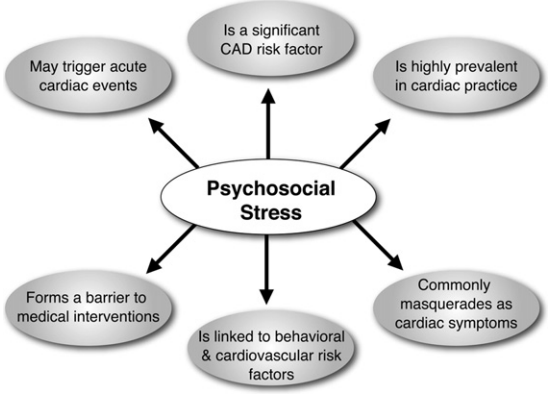

Fig 1. Several reasons for interest by medical practices in the evaluation and treatment of psychosocial stress. Reprinted with permission from Elsevier from Rozanski et al.[2]

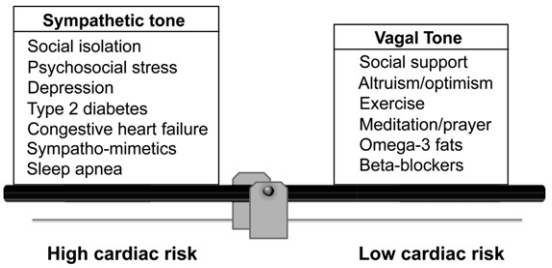

Fig 2. Important mediators of autonomic tone and CV risk. Reprinted with permission from Elsevier from Das and O'Keefe.[7]

Role of the autonomic nervous system

Chronic imbalance in the autonomic nervous system (ANS) with a shift toward higher sympathetic predominance can be considered a CV risk factor[7,9,10,11] and, when present, worsens CV prognosis (Fig 2).[7] Sympathetic over activity has been shown to produce adverse effects on endothelial function and coronary artery tone and to predispose to dysrhythmias, left ventricular hypertrophy, platelet activation, and thrombosis. Many forms of PS will adversely impact the ANS by increasing levels of norepinephrine and suppressing vagal tone, which often manifests clinically as reduced heart rate variability, increased resting heart rate, blunted peak exercise heart rate, and delayed postexercise heart rate recovery. Notwithstanding, there are many mechanisms other than ANS-related disturbances by which PS may adversely impact CV health, including elevations in cortisol, renin, and insulin.[7,12]

Although there are many aspects of PS and CVD, our research has specifically focused on anxiety, hostility, and, especially, depression in CVD.[3]

Anxiety and CVD

A causal relationship has been suggested between anxiety and CHD, although the evidence for this association may be less than with depression. Also, as with depression, there are similar concerns about causality, including the concern about reverse causation.[3,13-19] However, several recent studies have suggested that anxiety is an independent predictor of major CHD morbidity and mortality. Clearly, both acute and chronic anxiety seem to adversely affect CV risk. Although anxiety may increase the risk of CHD events including acute MI, the link between anxiety and CV death, especially sudden cardiac death, where a dose-dependent relationship exists, seems to be stronger.[2,20,21] Anxiety