Editorial Comment

Impact of Stress Monitoring and Lifestyle in the Development of Cardiovascular Disease

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Several lines of evidence suggest that psychosocial factors contribute to the pathogenesis and expression of cardiovascular disease (*1*). Largely on the basis of studies in middle-aged men, this evidence is composed of data relating coronary artery disease (CAD) risk to three specific psychosocial domains: 1) depression and anxiety, 2) personality factors and character traits and 3) psychological interaction with the organization of work (job strain). Depression is a risk factor for cardiac morbidity and mortality in patients with CAD, especially after an acute myocardial infarction (MI) (*2–4*). Major depression is associated with a 4-fold increase in the risk of mortality during the first 6 months after an acute MI, and its prognostic significance is comparable to that of left ventricular dysfunction and a history of MI (*3*). Depression is not unusual among individuals with CAD, with studies indicating that about 20–25% of patients suffer from depression after a cardiac event (*5*).

Barefoot and Schroll (*6*) reported that high scores on a measure of symptoms of depressed mood were associated with an increased risk for acute MI and early mortality during a 27-year follow-up, and that the impact of a depressed affect on health did not differ between men and women. We also observed in middle-aged community-dwelling people that a depressive mood predicted the occurrence of vascular diseases beyond the prediction provided by age, gender, ambulatory blood pressure (BP), lifestyle and environmental conditions (*7*).

Most studies have shown an increased incidence of cardiovascular events mainly related to episodic BP elevation in the morning, peaking between 6:00 AM and 12:00 PM. Ambulatory BP monitoring has become an important tool in the diagnosis and management of hypertension. We reported that circadian and other patterns are synchronized by socio-ecologic factors, such as human lifestyles, as well as economic and environmental conditions (*8*). We demonstrated the presence of a weekly BP variation in community-dwelling subjects, and it was most prominent in depressed people (*9*). A Monday peak has been reported for the incidence of acute myocardial infarction, sudden cardiac death, and stroke. Our previous studies showed that the morning BP surge on Mondays was significantly higher compared with other weekdays (*10*). Although a direct association between the Monday surge in BP and cardiovascular events could not be demonstrated directly, it is possible that a morning surge of BP on Mondays triggers cardiovascular events.

A positive association between job strain and ambulatory BP levels has been found, but the relationship between occupational stress and higher BP in women is rather ambiguous compared with that in men (*11*). Clays *et al*. (*12*) have detected strong and consistent associations between high job strain and ambulatory BP. The impact of job strain on ambulatory BP was not limited to an increased BP at work, but also extended to an increased BP at home and during sleep. It is important to note that the associations in this study were independent of other risk factors of high BP (*i.e.*, gender, age, body mass index, smoking, physical demands of the job, stress outside work, and physical activity prior to BP measurements). However, a single assessment based on perceived job strain increases the risk of misclassification due to inaccuracy in measurement. The associations are likely underestimations, or at least conservative estimates of the actual relationships between job stress and BP. Further research

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with more appropriate study designs is needed in order to clarify the exact nature of the relationship between job strain and BP.

In view of the strong relation between mental stress and adverse cardiac events, studies should be done to ascertain that stress is properly diagnosed and treated. Sawai *et al*. (*13*) developed a new device to estimate the influence of mental stress on cardiovascular parameters, including BP, heart rate, and sympathetic activity, with the use of a quantitative indicator of energy expenditure. We suggest that future models of this kind of device be used to assess all dimensions of the cardiovascular response to stress, focusing on the cumulative duration of response.

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