Vice Presidential Address

The Psychophysiology of cardiovascular reactivity

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Abstract

In this presentation cardiovascular reactivity (CVR) was discussed in relation to several prominent hypotheses regarding its stability and personality and social factors that appear as moderators of this reactivity. A call for additional research on cardiovascular reactivity in women was made in view of the relatively scarce amount of work on CVR in women. © 1997 Elsevier Science B.V. All rights reserved

Keywords: Cardiovascular reactivity, Coronary heart disease, Personality, Type A, Type B, Hostility, Anger, Gender

The American writer Washington Irving alluded to the effects of personality on cardiovascular reactivity in his story about Rip Van Winkle written over 175 years ago (Irving, 1820). In his story Irving described Rip Van Winkle's spouse as a "termagant wife" which, in my dictionary, is defined as "a violent, quarreling, scolding woman." On the other hand, Mr. Van Winkle was an easy-going person who had "an unsuperable aversion to all kinds of profitable labor" (Irving, 1820, p. 36). Rip's wife nagged him persistently for his ineptitude, carelessness, and the ruin he was causing his family. His wife's sharp tongue often drove Rip into the woods with his rifle and hunting dog. In Irving's story Rip went into the woods one day where he met a band of strange people. He drank a great deal of their potent liquor and fell into a deep sleep from which he did not waken until 20 years had passed!

When Rip returned home he found that his very angry, hostile wife had died some time before. It appears that she broke a blood vessel while yelling at a peddler in a fit of rage! Irving was writing about "folk wisdom" of the time that if you were an ill-tempered person you might die of a stroke. This common sense belief of over 175 years ago has been transformed into contemporary research on personality factors that might make an individual more prone to develop cardiovascular disease and therefore die from "cardiovascular accidents."

Cardiovascular reactivity as a psychophysiological trait

Modern researchers have been attempting to link cardiovascular reactivity to personality and
Cardiovascular reactivity refers to the magnitude and patterns of cardiovascular responses to challenge. Various researchers have provided evidence that reactivity is influenced by a number of factors. Among these factors are personality, the type of task, social influences, and gender. Researchers are interested in the hypothesis that exaggerated reactivity to psychological stressors may play a role in the development of cardiovascular disease, including coronary heart disease and high blood pressure.

Sherwood and Turner (1992) presented an overview of cardiovascular reactivity research. They pointed out that heart rate (HR) and blood pressure (BP) have been the main measures of reactivity, but that others are now being used. For example, cardiac output (amount of blood pumped by the heart per minute), stroke volume (amount of blood pumped per beat), and total peripheral resistance (resistance to blood flow in the body) are commonly used. These measures provide convenient indices of cardiovascular activity compared to some baseline—usually a state of quiet or rest. An individual who shows an increase in HR of 30 beats per minute (bpm) from baseline, when asked to perform some task, is more reactive than one who increases by 10 bpm for the same task. These individual differences are of interest to psychophysicists because they have been found to have predictive significance for health outcomes—for example, the development of high blood pressure. A prominent hypothesis in this area is that degree of reactivity may be predictive of later development of cardiovascular disease (Light, Dolan, Davis and Sherwood, 1992).

Sherwood and Turner (1992) concluded that some individuals appear to be more reactive than others in showing typically large cardiovascular responses to a variety of stressful stimuli. In fact, research has shown the very large magnitude of individual differences in reactivity to a variety of laboratory stressors including mental arithmetic, reaction time, speech presentation, cold pressor, and mirror tracing (Sherwood, Davis, Dolan and Light, 1992). These researchers have also provided evidence for the stability of the hemodynamics of cardiovascular reactivity over time (Sherwood, Turner, Light and Blumenthal, 1990).

The mechanisms responsible for these responses are relatively stable, suggesting that cardiovascular reactivity has attributes of a psychophysiological trait (Sherwood and Turner, 1992).

Possible link between reactivity and CHD

The link between cardiovascular reactivity and CHD has not been definitely established, but it has been hypothesized that stress reactions cause the release of epinephrine and norepinephrine which may increase blood pressure to levels high enough to injure the inner arterial walls if reactivity is chronic (Barnett, Biener and Baruch, 1987; Saab, Matthews, Stoney and McDonald, 1989; van Doornan and van Blokland, 1992). Additionally, elevated blood pressure may become chronic as a result of repeated reactivity involving the desensitization of beta-adrenergic receptors (Bertel, Buhler, Kiowski and Lutold, 1980). This could compromise the ability of these receptors to homeostatically regulate blood pressure. Increased SNS activity may also increase the level of blood lipids. Stress increases the quantity of lipids and may act to increase production or decrease removal of lipoproteins (McCann, 1992). These two factors, injuries to blood vessels, and increased lipids with stress, may worsen the athero-sclerotic process and contribute to vascular disease.

Personality

Research on personality factors in cardiovascular reactivity has been dominated by comparisons of Type A and Type B individuals (Andreassi, 1995). The Type A person is impatient, hard driving, and competitive. The Type B lacks these behaviors. The concept of Type A was advanced by two cardiologists who noticed personality differences in their patients (Friedman and Rosenman, 1974). Coronary heart disease (CHD) is a major cause of death and one longitudinal study indicated that Type A persons have three times the death rate from CHD than Type Bs (Jenkins, 1976). One view is that these disorders may be due in part to greater cardiovascular and neuroendocrine activity of Type As compared to Type
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