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Changes in heart beat timing: reactivity, resetting, or perturbation?

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Abstract

A widely held hypothesis within behavioral medicine is that cardiovascular reactivity is a risk factor for cardiovascular disease. The measurement model for this cardiovascular reactivity is rather simple. A basal level of function is seen to increase while the organism is stressed and then return to basal function. We argue that this model is incomplete and that other forms of 'reactivity' may be relevant to pathophysiology. A pathophysiological hypothesis is discussed which assumes a cyclic heart beat generation mechanism that is sensitive to stimulation only at certain phases of its cycle. Implications of this hypothesis for measurement are developed to illustrate the point that models of normal function can determine the measures most relevant to pathophysiology.

Keywords: Behavioral medicine; Cardiovascular reactivity; Heart rate; Phase sensitivity; Cardiac cycle time effect; Arrhythmia; Cardiac sudden death

Psychophysiology's traditional concern with concepts and mechanisms should be central to the field of 'cardiovascular reactivity'. This collection of papers expresses the vitality and variety of alternative psychophysiological concepts. The under-

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standing of the pathological should be guided by a concept of the usual. The last 15 years have witnessed the development of a major field, cardiovascular behavioral medicine. Spurred largely by the apparently successful prediction of heart disease incidence by the Type A coronary-prone behavior pattern (Rosenman et al., 1975, 1976), many psychologists became interested in how psychological processes contributed to cardiovascular disease. What made the competitive, hurried, job-involved, somewhat hostile Type A behavior pattern coronary prone? One answer was that Type A behavior would increase exposure to stress. Stress in turn should induce cardiovascular responses. Investigators in the fields of psychophysiology and psychosomatic medicine had already established that the heart and blood vessels would respond to stress (e.g. Sternbach, 1966; Lipowski et al., 1977). These cardiovascular reactions might disrupt normal cardiovascular regulation and thus induce a heightened risk for cardiovascular disease. The reasons for this were reviewed by a number of authors (Matthews, 1982; Jennings, 1983; Manuck and Krantz, 1986). An important paper by Manuck and Krantz (1986) developed a conceptual and measurement model of cardiovascular reactivity as a risk factor. For example, a model in which blood pressure reacted to work stress with an elevation lasting throughout the day was contrasted with a 'recurrent stress' model in which blood pressure transiently increased many times throughout the day in response to stressors. Both were seen as possible pathophysiological patterns, although laboratory investigations typically induce changes suggestive of 'recurrent stress'.

Investigators in cardiovascular reactivity were not initially overly concerned with the physiological mechanisms of reactivity. When discussed, the prevalent view was that the mechanism was a sympathetic nervous system and adrenal medullary response to stress (Dembroski et al., 1979). As might be expected, however, both the Type A concept and that of cardiovascular reactivity required further elaboration to fit emerging empirical findings (Pfiffner and Bättig, 1989; Manuck et al., 1990; Pickering and Gerin, 1990). The promise of cardiovascular reactivity remains, but significant research must be done. Many different neural and neuroendocrine responses play upon the cardiac and vascular system and these responses vary significantly over time and between individuals—particularly those of different ages and with different disease states. The field can no longer sustain the hope of identifying 'hot reactors' to 'stress' using a single videogame challenge and an automated blood pressure recorder. In this article, we suggest that an understanding of cardiac mechanisms may reveal possibilities of dysregulation quite different from the initial concept of how cardiovascular reactivity may lead to cardiovascular disease.

The implicit measurement model frequently used to study reactivity views the heart as producing a fixed level of activity. During a baseline, extraneous factors are controlled so a simple clocklike pumping is revealed. A manipulation, i.e. stress, then momentarily speeds the pump and heightens the pressure. Physiology tells us this model is too simple, (e.g. Mountcastle, 1968; Larsen et al., 1986); and our data (Jennings et al., 1992a) on baselines confirmed the difficulty of isolating a fixed resting level of heart rate or blood pressure. It is quite difficult to establish a stable,