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Relative effects of harassment, frustration, and task characteristics on cardiovascular reactivity

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Abstract

Effects of anger induction procedures such as frustration and harassment on cardiovascular reactivity have been demonstrated in a wide range of experimental situations. Similarly, heightened cardiovascular reactivity has been associated with a diverse range of tasks involving active coping, competition and interpersonal interaction. The present study sought to directly compare the relative effects of these two important ways of inducing cardiovascular changes. One hundred and five university students performed two tasks that differed in the degree of active coping and interpersonal competition: a competitive psychomotor task and a problem-solving task. States of anger were induced during both tasks by means of harassment, frustration or frustration + harassment. Task-related changes in heart rate, systolic blood pressure, diastolic blood pressure, pulse volume amplitude and respiratory sinus arrhythmia amplitude were monitored. The competitive psychomotor task produced greater cardiovascular reactivity than did the problem-solving task. Harassment and frustration + harassment provoked more cardiovascular reactivity than did frustration alone. However, harassment and frustration + harassment had the greatest cardiovascular effects in the competitive task, whereas frustration had the greatest cardiovascular effects in the problem-solving task. In this sense, the increases on cardiovascular reactivity seem to depend on the interaction between anger induction procedures and the context in which anger is provoked.

Keywords: Active coping; Anger; Cardiovascular reactivity; Competition; Frustration; Harassment; Task characteristics

1. Introduction

Research on cardiovascular reactivity to stress as a risk factor for disease has focussed on two major questions (Hodapp et al., 1990, 1992): the identification of stressors that are specifically linked to a harmful pattern of cardiovascular responses, and the identification of specific emo-

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tions or personality traits associated with heightened cardiovascular activity. Regarding the former, most research on specific cardiovascular stressors during the last few decades has addressed the distinction between active and passive coping. With regard to the latter issue, researchers have paid most attention to the emotion of anger and its related trait of hostility, because of the long history of their association with cardiovascular disease. In both areas, differential cardiovascular reactivity has been demonstrated.

Active coping refers to the mental or physical effort exerted to achieve actual or perceived control over the outcome of a situation (Light and Obrist, 1983). It is also related to perceptions of task difficulty, task predictability and self-efficacy (Gerin et al., 1996). The cardiovascular response pattern produced during active coping is predominantly characterized by β-adrenergic stimulation (β-1), which induces increases in skeletal muscle vasodilatation, heart rate (HR), systolic blood pressure (SBP) and cardiac output, with smaller effects on diastolic blood pressure (DBP) and vascular resistance (Obrist, 1981; Lovallo et al., 1985; Smith and Frohm, 1985; Johnston et al., 1994; Bongard et al., 1997; Waldstein et al., 1997; Hartley et al., 1999; Brownley et al., 2000). *Passive coping* tasks, on the other hand, are those in which subjects are not allowed to exert control over the outcome of a situation. Such tasks have been reported to induce a vascular response pattern, fundamentally reflecting α-adrenergic influences, that produces increased skeletal muscle vasoconstriction and vascular resistance, with stronger effects on DBP and weaker effects on HR, SBP and cardiac output (Obrist, 1981; Lovallo et al., 1985; Hodapp et al., 1990; Waldstein et al., 1997; Hartley et al., 1999; Brownley et al., 2000). A *mixed pattern of active and passive coping* during psychological stressors has also been described (Schneiderman and McCabe, 1989). This pattern may reflect characteristics of the behavioural task itself and its context, as well as differences in individual history and in perceptual and response styles, among other variables.

The above approaches to active–passive coping have emphasized the role of β- and α-adrenergic influences in cardiovascular reactivity. Lately, the importance of parasympathetic influences and sympathetic–parasympathetic interactions during active and passive tasks has also been suggested (Reyes del Paso, 1992; Reyes del Paso et al., 1993, 1996; Waldstein et al., 1997). Using measures of respiratory sinus arrhythmia (RSA) and baroreceptor cardiac reflex sensitivity, it has been shown that stressful situations are generally also characterized by a withdrawal of vagal cardiac control.

It should be noted, however, that the differentiation between active and passive coping is always relative, because, by definition, any laboratory task always requires some kind of active effort, regardless of the experimenter's manipulation of the outcome. Moreover, as pointed out by Waldstein et al. (1997), there is substantial variability among active coping tasks in terms of psychological responses and concomitant haemodynamic response patterns. Indeed, there is evidence that some properties of active coping tasks differentially induce cardiovascular reactivity. This is the case, for example, with active coping stressors that involve competition and social interaction (Ewart and Kolodnar, 1991; Winzer et al., 1999; Harrison et al., 2001). Ewart and Kolodnar (1991) reported larger blood pressure responses to a social competence interview than to non-social laboratory stressors. Similarly, Harrison et al. (2001) have shown that social competition produces blood pressure and HR increases and a significant shortening of the pre-ejection period, an index of enhanced β-adrenergic influences on the heart.

In relation to the identification of specific emotions, several investigations have confirmed the role of anger to evoke an increase in cardiovascular reactivity (Bongard et al., 1997; Waldstein et al., 1997). The emotion of anger refers to the temporal experience of subjective feelings of annoyance, irritation or rage when we are injured, deceived, frustrated, controlled, or betrayed. Anger has usually been associated with higher HR, cardiac output, and SBP responses, but the greatest responses have been obtained in vascular tonus and DBP, specially when anger cannot be expressed by direct action (Sinha et al., 1992; Bongard et al., 1997).

Anger is produced by situations in which we are injured, deceived or betrayed, by situations in