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Hypertension and pain sensitivity: effects of gender and cardiovascular reactivity

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Abstract

Repeatedly, hypertensives have been found to appraise physical stressors as less aversive than normotensives. The main aim of the present study was to examine the effects of gender and cardiovascular reactivity in the relationship between hypertension and appraisal of pain. Forty-two unmedicated hypertensives and 21 normotensive controls of both genders were exposed to an electric current stimulus, while various cardiovascular parameters and pre-stressor anxiety were measured. In general, hypertensive women, but not men, showed diminished pain sensitivity compared to their normotensive counterparts. When the analyses were repeated with controlling for cardiovascular reactivity, the between-group effects were no longer significant. The results indicate that (i) profound gender differences exist in hypertension-related pain sensitivity and (ii) these effects seem to be mediated, at least partly, by cardiovascular reactivity.

Keywords: Baroreceptor; Gender; Cardiovascular reactivity; Hypertension; Pain sensitivity

1. Introduction

Repeatedly, elevated blood pressure has been found to be associated with diminished sensitivity to painful physical stimulation. This has been demonstrated for electrical (Zamir et al., 1980), thermal (Sheps et al., 1992), and finger pressure pain stimulation techniques (Bruehl et al., 1992). Moreover, also in normotensive

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samples, an inverse relationship between blood pressure and perceived painfulness of physical stressors has been obtained (Bruehl et al., 1992). This inverse relationship has been found in both between-subjects (Zamir and Shuber, 1980) and within-subjects designs (Dworkin et al., 1979), in animal (Randich and Maixner, 1984) as well as in human studies (Sheps et al., 1992).

However, until now, most studies on the relationship between cardiovascular activity and pain sensitivity have been conducted on male subjects (Zamir and Shuber, 1980; Elbert et al., 1988; Bruehl et al., 1992; Sheps et al., 1992). The few studies conducted on both genders have revealed conflicting results. For instance, Fillingim and Maixner (1996) found an inverse association between resting systolic blood pressure (SBP) and pain sensitivity only in men, not in women. In another study, partially a reverse effect was found: resting SBP and blood pressure reactivity to a speech task were associated with lower pain sensitivity to a thermal stimulus only in women (Bragdon et al., 1994). The latter outcome is in agreement with an investigation showing that parental history of hypertension was related to lower retrospective pain ratings after venipuncture in women, not in men (France et al., 1994). In light of these discrepancies, in the present study, the primary aim was to examine gender differences in various pain conditions more systematically.

In most studies showing diminished pain sensitivity in hypertensives, subjects had little control over the physically aversive stimuli (Zamir and Shuber, 1980; Bruehl et al., 1992; Sheps et al., 1992). Therefore, it may be hypothesized that having little control over a stressor moderates the relationship between hypertension and pain sensitivity. For instance, Rau et al. (1994) argued that their failure to find differences in pain threshold between hypertensives and normotensives may be attributed to the fact that the participants had some feeling of control over the stimulus: as soon as the pain threshold was reached, the stimulation was stopped by the participants. Therefore, in the present study, electric stimulation was used both in an externally controlled condition, in which stimulus intensity was controlled automatically by a computer and a self-controlled condition, in which the participants themselves had full control over stimulus intensity. Furthermore, it has been proposed that stimulus duration and frequency may be important variables influencing the pathways involved in antinociception (Terman et al., 1984). For example, it has been suggested that in exposure to short-duration aversive stimuli, an opioid system may be involved, whereas non-opioid mechanisms may predominate when the organism is exposed to stressors of long-duration (Terman et al., 1984). It is conceivable that involvement of qualitatively different systems might also influence the degree of antinociception (Randich and Maixner, 1984). In the present study, the externally controlled condition included two subconditions: one with only a few slow, relatively long stimuli and one in which more frequent, but short stimuli were presented.

Furthermore, it was examined whether appraisal of pain would be related to indices of cardiovascular *reactivity*, a putative risk factor or marker for hypertension (Manuck et al., 1990). Bruehl et al. (1992) demonstrated that SBP reactivity during a finger pressure stimulus trial was positively related to pain ratings, in contrast to negative correlations with baseline SBP. Similar results were obtained in