Resting Blood Pressure and Blood Pressure Reactivity: Contributions to Experimental Pain Report in Healthy Males

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Abstract: It is not known whether it is the resting blood pressure or the pain-induced blood pressure changes (blood pressure reactivity) that contributes to sex differences in nociception. The study investigated whether the relationship between sex and experimental pain report was explained by blood pressure at rest, or during pain task, or both in healthy, young adult males. Fifty-eight apparently healthy male subjects had their blood pressure, heart rate measured pre- and post- a cold pressor test. Pain threshold and tolerance were calculated. Univariate analyses indicated significant positive correlation between baseline systolic blood pressure, systolic blood pressure reactivity, heart rate, reactivity, but not baseline diastolic blood pressure, diastolic blood pressure reactivity, heart rate, weight, height and pain sensitivity. However none of the positively correlated parameters cloud significantly product pain threshold or pain tolerance. Both resting blood pressure and blood pressure reactivity contribute significantly to nociceptive processing in males, however they do not completely explain nociceptive behaviour in males.

Key words: Blood pressure · heart rate · pain · blood pressure reactivity

INTRODUCTION

Experimentally induced pain in healthy human subjects under controlled laboratory conditions often yields differentiated results, with women reporting more pain than men [1, 2]. Scientific efforts to explain sex-related difference in pain sensitivity between the sexes have focused on first order biological factors including genetic and anatomical differences. However, given the well-established differences between men and women in many cardiovascular parameters, blood pressure must be considered in any attempt to explain sex differences in pain sensitivity [3].

Fillingim and Mixner [4] assessed the impact of resting blood pressure on sex differences in pain reactivity in 23 female and 25 male subjects. Sex differences occurred only in the ratings of intensity for suprathreshold heat stimuli and in ischemic pain tolerance thresholds with women appearing more sensitive to pain. However the men had higher blood pressure then the women. Consequently, blood pressure could be a powerful influence on sex differences in pain sensitivity.

Women and men differ in their blood pressure responses to acute stress, with men generally showing greater blood pressure increases [5]. It is therefore conceivable that apart from resting blood pressure, blood pressure reactivity i.e. pain-induced changes in blood pressure may be positively associated with pain threshold and/or pain sensitivity. This was the hypothesis tested in the study using healthy male volunteers. Since there is a possible link between anthropometric indices and blood pressure, the study further examined the possible correlation between weight and height and pain sensitivity.

METHODS

Participants: Fifty-eight female undergraduate students aged 21 to 31 years (mean = 22.4, SD 2.78) participated in the study after informed consent was obtained in accordance with guidelines of the research ethics committee at the Ladoke Akintola University. Exclusion criteria were ill-health, smoking, recent caffeine intake and prior experience with the cold pressor a test.

Blood pressure measurement: Baseline blood pressure (BBP) and baseline heart rate (BHR) were calculated as the mean of three measures during the resting baseline period. The BP and HR readings taken after hand immersion...
served as task BP and task HR. BP reactivity (BPR) was calculated as task BP minus baseline BP. Heart rate reactivity (HRR) was similarly calculated.

**Pain assessment:** Pain was induced in a 2.5 cubic foot cold pressor bath fitted with a scream dividing ice from water and a rotor circulating water continuously through the ice, thereby maintaining water temperature of 1 to 3 degrees Celsius. Pain threshold was measured as the time in seconds to a participant is report of pain onset during the cold pressor task. Pain tolerance was measured as the total time that elapsed while a participant’s hand remained in the cold water.

**Procedure:** After the BP and HR had been obtained, the participants were asked to put their non-dominant hand, palm facing down, in the water. They were instructed to say ‘pain’ or their first experience of pain while still maintaining the hand in the water until they reached their tolerance level, at which time they should say ‘pain limit’ and withdraw the hand. A cut-off time of five minutes was set for the hand immersion. BP and HR readings were immediately taken after the cold pressor test.

**Statistical analysis:** We calculated Pearson’s product moment correlation to examine the relationships between SBP (Systolic blood pressure), DBP (Diastolic blood pressure), HR (Heart rate), height, weight, HRR, BPR, pain threshold and pain tolerance. Effect sizes (Cohen’s) were calculated to determine the magnitude of correlational relationships and sex differences. By Cohen’s convention [6], effect sizes of approximately 0.20 are small, 0.50 are moderate and 0.80 are large.

Two sets of regression analysis were performed. In the first set, SBP, DBP, HR, HRR, SBPR (Systolic blood pressure reactivity i.e. difference between pre- and post- immersion SBP), DBPR (Diastolic blood pressure reactivity, i.e. difference between pre- and post- immersion DBP) were regressed first on pain threshold. In the second set, these variables were regressed on pain tolerance. The t-test statistic was used to determine the significance of a predictor effect in both sets while a p<0.05 was the criterion for statistical significance.

**RESULTS**

Baseline SBP, SBPR and HR but not baseline DBP, HR, weight, height and DBPR positively correlated with pain threshold and tolerance (Table 1). This relationship was of medium size by Cohen’s convention.

**DISCUSSION**

We have investigated whether blood pressure at rest or blood-pressure changes induced by the pain task (blood pressure reactivity) could explain pain sensitivity in males. The cardinal findings of this study can be summarized as follows. 1) Resting SBP, SBPR, HRR showed positive correlation with pain sensitivity although none of them was a predictor of pain threshold or pain tolerance. 2) Resting SBP, SBPR and HRR alone can not explain pain sensitivity in males. Other factors must be involved. 3) The anthropometric measures height and weight did not correlate with pain sensitivity.

The significant positive correlation between resting SBP and pain tolerance/threshold coupled with several reports of close association between high blood pressure and diminished pain sensitivity [4] points to a high blood pressure- induced hypoalgesia. However, this must be interpreted with some caution against a backdrop of other reports to the contrary. For instance, Meller et al. [7] showed that Wistar- Kyoto rats (WKY) and spontaneously hypertensive rats (SHRs) did not differ in the tail-flick test, SHRs were found to exhibit normal [8] or exaggerated [9] nociceptive responses while Wong et al. [10] did not find any
difference in nocicception between WKY and SHR in the hot plate test. Could this disparity reflect a specie or test paradigm difference? In any case, it is obvious that resting SBP alone can not be responsible.

Although we did not do a comparative study it is noteworthy that our finding of a positive correlation between resting blood pressure and pain tolerance thresholds contradicts the findings of Fillingim [11] and Bragdon et al. [12] who found this to hold true only in women. They used contract heat to induce pain while we applied the cold pressor paradigm. Even then it must be noted that Bragdon et al. [12] measured nociceptive response by administering contact heat stimuli before and after the recall of a non-painful stressor i.e the recall of a stressful situation.

Although our study cannot compare perfectly with that of Bragdon et al. [12] for obvious methodological differences, our finding of a positive correlation between systolic blood pressure reactivity and pain tolerance and thresholds in males is partly in consonance with their finding of analgesia associated with the recall of a stressful situation only in women. Since pain is a stressor it is suggested that there is an interplay of stress-induced analgesia and resting blood pressure that modulates pain sensitivity in both sexes.

The coupling of diminished pain sensitivity and raised blood pressure has been attributed to many factors. Several lines of evidence point to a role of the baroreflex system. Reducing or interrupting sinoaortic afferent input by various techniques markedly attenuated or eliminated hypertension-associated hypoalgesia in several experimental models of acute and chronic BP elevation [13, 14]. Raised blood pressure is also known to attenuate nociceptive signal at the spinal level [14].

Finally, it is concluded that both resting blood pressure and blood pressure reactivity most likely with other unknown factors contribute to nociceptive behavioural profile in males. Even then, it might be scientifically premature to conclude that the relationship between blood pressure and pain sensitivity is causal or to speculate about its clinical relevance.

REFERENCES