

AUTONOMIC RESPONSE TO STRESS IN INTERSTITIAL CYSTITIS

Aims of Study

The hypothesis has been advanced that interstitial cystitis (IC) patients have greater sympathetic tone and greater reactivity to stress than healthy individuals. Sixty percent of IC patients report symptom exacerbation by stress [1]. The stress response involves physiological adaptation to stimuli perceived as physically or emotionally threatening [2] with activation of the sympathetic nervous system, hypothalamic-pituitary-adrenal axis, immune system and various other physiologic and psychological effects. Findings consistent with the sympathetic effects on the inflammatory processes in IC patients have been documented. Increased symptoms of pain and urgency with voiding were evoked by an acute mental stressor in IC patients but not controls [3]. This study examines effects of a laboratory mental stress challenge on blood pressure and heart rate in IC patients and in healthy controls.

Methods

Fourteen female IC patients and fourteen age-matched female controls participated in a laboratory session, including a 60-minute baseline, 25-minutes of mental stress tasks, and 75-minutes of recovery. The mental stress challenge involved a 5-minute videotaped speech task, a 14-minute computerized Stroop word color test [4] and 6-minutes of mental arithmetic performed aloud. Systolic (SBP) and diastolic (DBP) blood pressure and heart rate (HR) were measured at regular intervals (every 2 minutes) beginning 15-minutes before the stressor, during the stress challenge, and until 100 minutes following the onset of the stressor. Level of chronic stress, symptom severity and pain at voiding were also assessed during the session (NIDDK Interstitial Cystitis Data Base symptom questionnaire) [5].

Results

The mean age of participants was 49 years (range, 32-66). There was no difference in average reports of chronic stress during the week prior to the study session in patients or controls. In both groups, the laboratory stressor was effective in eliciting a stress response characterized by autonomic arousal, as measured by increased SBP ($p < 0.001$), DBP and HR. Both groups perceived the task as equally stressful. HR of IC patients at baseline (82.02 beats per minute) was significantly higher than that of controls (63.31 bpm; $p = 0.0001$). IC patients had significantly elevated HR at each time-point measured as compared with controls (all p values < 0.0001) (Figure 1), with a relatively constant average mean difference between groups of 19.5 bpm (S.D. 4.0; main effect for group $p < 0.0001$). This suggests that although there was a consistent elevation in HR among IC patients, there was not a difference in HR reactivity to stress between groups. Although both SBP and DBP were consistently elevated in patients as compared to controls, these elevations were not significant ($p = 0.12$) (Figure 2), and there was not a difference in blood pressure reactivity to stress between groups. Assessments of HR change in conjunction with pain over time indicated that in IC patients: for a 1-unit increase in pain score, there was a 0.9 unit increase in HR. This relationship was not evident in controls. Pain level did not change in controls.

Conclusions

Female IC patients have elevated HR at baseline and throughout a laboratory mental stress challenge compared to healthy age-matched women. Differences in HR and blood pressure reactivity between the two groups were not evident. This physiologic evidence adds support to the hypothesis that patients with IC have greater sympathetic tone at baseline than healthy women but do not have greater sympathetic reactivity to stress. These results are consistent with reports of stress related exacerbation of clinical symptoms and parameters in other neuroinflammatory conditions (rheumatoid arthritis, psoriasis, irritable bowel disease). Further evaluation of the physiologic mechanisms underlying stress related Interstitial Cystitis symptom exacerbation is needed.

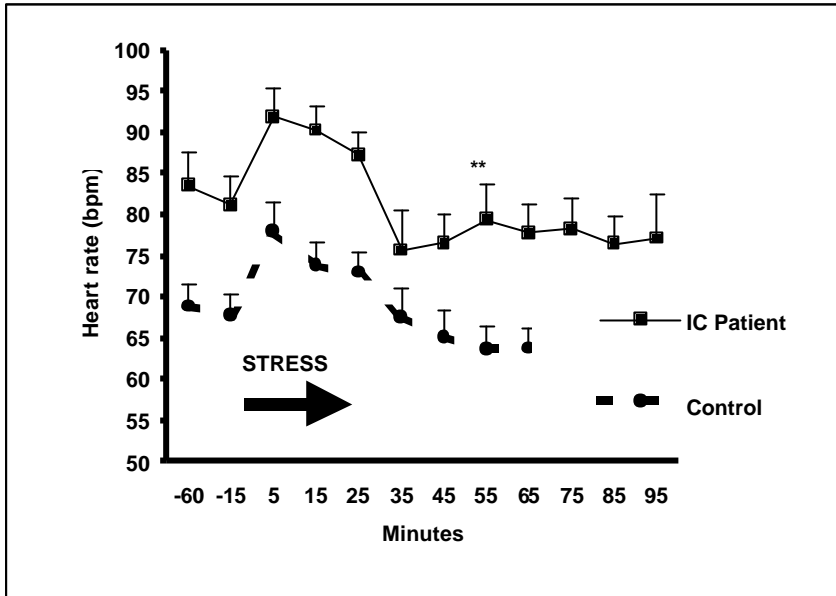


Figure 1: Heart rate (HR) in patients with interstitial cystitis and controls with time. Stress challenge occurred between minutes 0 and +25 (arrow). HR was significantly greater in patients than in controls at baseline and at all time points (all p values < 0.0001).

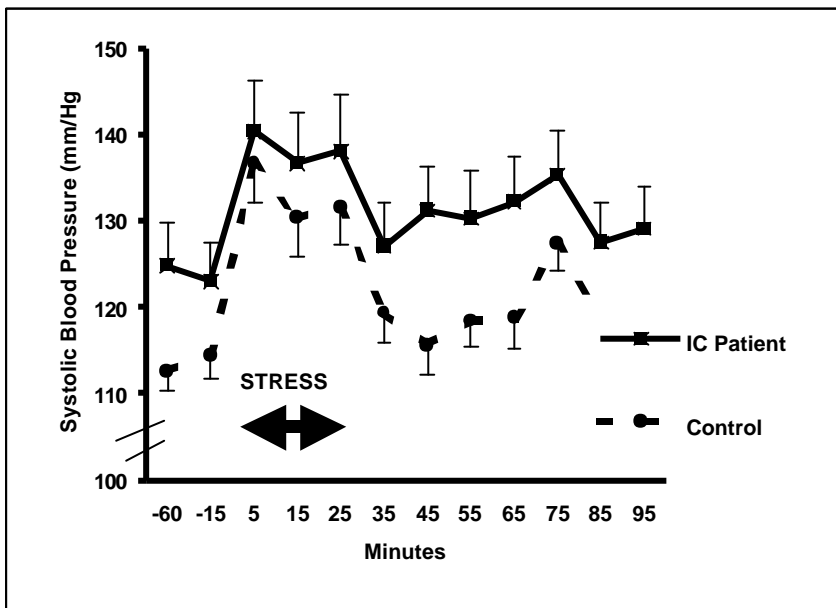


Figure 2: Systolic blood pressure (SBP) in patients with interstitial cystitis and controls with time. SBP was consistently elevated in patients compared to controls at all time points. These elevations were not significant (p=0.12).

References

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5. Simon LJ, Landis JR, Erikson DR, et al. The Interstitial Cystitis Data Base Study: concepts and preliminary baseline descriptive statistics. *Urology* 1997;49(5A suppl):64-75.