Pergamon

Anxiety Disorders
15 (2001) 535–553

JOURNAL OF Anxiety Disorders

Heartbeat awareness and heart rate reactivity in anxiety sensitivity: A further investigation

Sherry H. Stewart*, Susan E. Buffett-Jerrott, Racqel Kokaram

Department of Psychology, Dalhousie University, Halifax, Nova Scotia, Canada B3H 4J1

Received 23 June 1999; received in revised form 24 September 1999; accepted 29 September 1999

Abstract

We examined the effects of anxiety sensitivity (AS) and arousal induction on heartbeat awareness and heart rate reactivity in a nonclinical undergraduate sample. Students were randomly selected from a larger screening sample to fill two groups (high and low AS; $n = 15$ per group) based on Anxiety Sensitivity Index (ASI) [Peterson, R. A., & Reiss, S. (1992). *Anxiety Sensitivity Index manual* (2nd ed. revised). Worthington, OH: International Diagnostic Systems] scores. Participants completed a mental arithmetic/spelling task to induce arousal. At two phases (i.e., baseline vs. stress), participants estimated their heart rates during specified intervals using a mental tracking paradigm. Actual heart rates were simultaneously measured. Although heart rate did increase significantly from baseline to stress phases, high and low AS groups did not differ in terms of heart rate reactivity to the stressor. As hypothesized, high AS individuals were more accurate in estimating their actual heart rate as compared to low AS individuals. Contrary to hypothesis, the AS group differences in accuracy of heartbeat estimations did not vary across baseline vs. stress phases. Interestingly, only low AS individuals provided heart rate estimates which were significantly lower than their actual heart rate readings. Although high and low AS individuals did not differ in actual heart rate, high AS individuals provided significantly higher heart rate estimates than low AS individuals. These results are consistent with the interoceptive sensitivity hypothesis. Implications of the greater heartbeat awareness of high AS individuals are discussed.

Keywords: Anxiety sensitivity; Anxiety Sensitivity Index; Arousal; Heart rate

* Corresponding author. Tel.: +1-902-494-3793; fax: +1-902-494-6585.
E-mail address: sstewart@is.dal.ca (S.H. Stewart).

0887-6185/01/$ – see front matter
PII: S0887-6185(01)00080-9

1. Introduction

Anxiety sensitivity (AS) is the fear of anxiety-related sensations (e.g., increased heart rate, breathlessness, dizziness, nausea) due to beliefs that such sensations have harmful consequences (Reiss, 1991). Feared consequences include concerns that arousal-related bodily sensations are signs of physical catastrophe, social embarrassment, or mental incapacitation (Stewart, Taylor, & Baker, 1997). High AS is associated with increased fearful responding to provocations of arousal (see review by Stein & Rapee, 1999). High levels of AS also distinguish patients with panic disorder from normal controls, as well as from patients with other anxiety disorders (see review by Cox, Borger, & Enns, 1999).

Reiss and McNally (1985) proposed an "enhanced reactivity" hypothesis as one potential explanation for the development of high AS. Specifically, they suggested that individuals who experience greater autonomic reactivity in response to stress would be more likely to develop concerns about arousal-related bodily sensations (i.e., high AS). Although some limited support for this notion has been obtained from studies of clinical panic disorder patients (see review by Stein & Rapee, 1999), studies with nonclinical high AS participants have generally failed to support the enhanced reactivity hypothesis. For example, Shostak and Peterson (1990) failed to find differences in muscle tension and systolic blood pressure across three nonclinical AS groups (low vs. moderate vs. high) in response to a mental arithmetic stressor. Furthermore, a study involving a hyperventilation challenge to induce arousal in a nonclinical sample also failed to find significant AS group (low vs. high) differences in heart rate reactivity (Asmundson, Norton, Wilson, & Sandler, 1994). Similarly, Stewart and Pihl (1994) failed to demonstrate significant AS group (low vs. moderate vs. high) differences in heart rate reactivity in response to a signaled loud noise burst stressor.

One study using nonclinical women does provide at least partial support for the enhanced reactivity hypothesis, however. Sturges and Goetsch (1996) investigated heart rate reactivity in response to two arousal-induction challenges: caffeine provocation and mental arithmetic. During the mental arithmetic stressor, nonclinical high AS women displayed a marginally greater heart rate response than low AS women ($P < .06$). However, during the caffeine provocation, there were no significant AS group differences in heart rate response. Studies that have failed to support the enhanced reactivity hypothesis among high AS nonclinical participants can be criticized on methodological grounds. Some have failed to use effective arousal induction procedures in terms of the magnitude of the heart rate response achieved (e.g., Asmundson et al., 1994; Sturges & Goetsch, 1996—caffeine challenge). Others have failed to measure heart rate (e.g., Shostak & Peterson, 1990). Heart rate reactivity in response to effective arousal-induction procedures appears to be a potentially promising area for further investigation of the enhanced reactivity hypothesis