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Anxiety and hypervigilance to cardiopulmonary sensations in non-cardiac chest pain patients with and without psychiatric disorders

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ABSTRACT

We investigated body vigilance, cardiac anxiety, and the mediating role of interoceptive fear on pain in patients with non-cardiac chest pain (NCCP; a syndrome of chest pain in the absence of identifiable organic etiology). Patients were more attentive to cardiac-congruent sensations than cardiac-incongruent sensations (e.g., gastrointestinal, cognitive dyscontrol; p's $< .001$). Patients with a DSM-IV Axis I anxiety or mood disorder were more body vigilant compared to patients who did not have a disorder (p's $< .05$). Patients with anxiety disorders were particularly vigilant to and fearful of cardiac sensations relative to patients without anxiety disorders. Latent variable path models examined the extent that interoceptive fear mediated the association between body vigilance and cardiac anxiety on chest pain. Within each model, diagnostic status, body vigilance, and cardiac anxiety were exogenous and predicted interoceptive fear that in turn predicted pain. Separate models examined body vigilance and cardiac anxiety, and both models fit the data well. Findings showed partial mediation for the body vigilance factor, and full mediation for the cardiac anxiety factor. Interoceptive fear played a mediating role in both models. The syndrome of NCCP may persist partly due to conscious hypervigilance to and fear of cardiac-congruent body sensations, particularly among anxious patients.

Each year about 4.6 million people in the United States are evaluated by an urgent medical service because of symptoms suggestive of an acute cardiac syndrome (Burt, 1999), however, estimates suggest that only about one-quarter of these individuals receive a confirmed cardiac diagnosis (Pope, Ruthazer, Beshansky, Griffith, & Selker, 1998). Some individuals are relieved that their symptoms are not indicative of a medical or cardiac diagnosis. Yet, a significant number of these patients continue to experience chest pain, worry about their cardiac function, and experience disability similar to patients with coronary artery disease despite their apparently favorable prognosis (Eifert, Hodson, Tracey, Seville, & Gunsawardane, 1996). This syndrome of recurrent chest pain in the absence of identifiable organic etiology is non-cardiac chest pain (NCCP).

Data on the medical course of NCCP is mixed. Whereas some studies show a good prognosis for NCCP patients following a negative coronary angiography (Lichtlen, Bargheer, & Wenzlaff, 1995), other studies show a higher mortality rate than the normal population (Bodegard, Erikssen, Bjornholt, Thelle, & Erikssen, 2004) or a rate not different from those with cardiac chest pain (Eslick & Talley, 2008). Progress in understanding the syndrome of NCCP is slowed and complicated by the diagnostic classification of NCCP as a 'diagnosis' of exclusion (i.e., medical rule-outs) rather than diagnostic inclusion (White, 2007). Yet, longitudinal studies show that patients with NCCP experience chest pain from 1 to 11 years after the initial evaluation (Papanicolaou et al., 1986; Wielgosz et al., 1984), and nearly half of NCCP patients (44–50%) continue to believe that they have a cardiac condition up to one-year after negative evaluations (Ockene, Shay, Alpert, Weiner, & Dalen, 1980; Potts & Bass, 1993).

In the absence of wholly satisfactory medical explanations for NCCP, researchers have tended to conceptualize NCCP from a biopsychosocial perspective. Early pioneering researchers asserted the importance of studying both affective and pain qualities in this syndrome (Beitman et al., 1987; Eifert, 1991), and several modern theoretical models of NCCP (Eifert, Zvolensky, & Lejuez, 2000; Mayou, 1998; White & Raffa, 2004) have been adapted from or share common features with empirically supported models of panic disorder and health anxiety. Affective components, such as worry and rumination, link NCCP theories to broader theories of anxiety and health anxiety. The relevance of theories of panic disorder applied to NCCP is partly due to the central role physical sensations play in the development and maintenance of the problem. The most influential models of panic disorder implicate

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physical sensations in the etiology and maintenance of panic (Barlow, 1988, 2002; Clark, 1986; Reiss, 1991). In Barlow's model, for instance, individuals with panic disorder possess a biological vulnerability and a psychological vulnerability (i.e., a sense that events and emotions are uncontrollable and unpredictable). This vulnerability results in an inward shift of attention during arousal that contributes to developing anxious apprehension about experiencing additional attacks. Individuals with anxious apprehension about future attacks have a propensity to associate interoceptive cues with the first panic through conditioning (termed 'learned alarms'). Somatic and cognitive cues may trigger future panic attacks. This model and other influential models propose that panic is triggered by bodily sensations that may be misinterpreted and posits an important role (or essential role, in some cases; Clark, 1986) for conscious appraisal of the sensations as dangerous (Schmidt & Woolaway-Bickel, 2006). Similar hypotheses with adaptations have been extended to hypochondriasis (Barsky, Cleary, Sarnie, & Ruskin, 1994; Kellner, 1986; Warwick & Salkovskis, 1990) or cardiac neurosis (Liebhart, 1976). Moreover, in their model of hypochondriasis, Warwick and Salkovskis (1990) indicate that if a patient's anxiety about a disease elicits autonomic symptoms that coincide with the symptoms of the feared disease, then anxiety will escalate. In sum, theoretical models of NCCP are rooted in the broad, deep foundations of decades of research in anxiety and anxiety-related disorders and there is considerable clinical utility in applying these models to the study of the problem of NCCP.

Prevalence rates for anxiety disorders are elevated among patients who experience NCCP. DSM-IV anxiety and mood disorders are overrepresented among patients with NCCP (Mayou, 1998; White et al., 2008), particularly the occurrence of panic attacks (White, Ruhr, & Gervino, 2009). Although the occurrence of panic attacks alone does not indicate panic disorder, physical sensations appear play an essential role in the development and maintenance of this functional syndrome for some NCCP patients. Consistent with current theoretical models of NCCP, it may be that following an initial chest pain experience, patients with NCCP selectively monitor and are vigilant to their cardiac activity. If they perceive cardiac changes (e.g., acceleration, fluttering), they may misinterpret this as an indication of cardiac dysfunction (e.g., myocardial infarction, coronary heart disease). These cognitions elicit anxiety that may culminate in increased heart rate and possible panic. A similar type of feedback loop has been validated in the false heart rate paradigm as applied to panic (Ehlers, Margraf, Roth, Taylor, & Birbaumer, 1988). Although actual palpitations did not increase in the laboratory, patients with panic disorder who were provided with false feedback of heart rate acceleration reported greater subjective and physiological anxiety (Ehlers et al., 1988), with some exceptions (Antony et al., 1995).

Available research suggests that patients with NCCP are fearful of physical sensations, particularly cardiac sensations (Aikens, Michael, Levin, & Lowry, 1999). Aikens, Zvolensky, and Eifert (2001) found that NCCP patients report greater fear of cardiopulmonary symptoms compared to other physical sensations; as a result, these authors concluded support for their 'differential specificity hypothesis' that stated that NCCP patients were differentially fearful of cardiopulmonary sensations. Given the high rates of anxiety disorders in this population, however, it is unclear whether these results were perhaps due, in full or in part, to the high rates of anxiety disorders in this population rather than any differential specificity, per se. Research examining this competing hypothesis is needed. Moreover, fears for chest pain (compared to other sensations; e.g., stomach upset, dizziness) are heightened partly due to the public perception that chest pain is dangerous. Chest pain is often associated with coronary artery disease – the leading cause of death and disability in the United States (American Heart Association, 2006) – and public health education campaigns designed to increase public awareness of cardiac risk factors and the implications of chest pain may have increased anxiety among some individuals. Research examining whether NCCP patients are consciously more vigilant to cardiopulmonary sensations than other bodily sensations is needed. It may be that attentional focus on specific pain sensations (e.g., chest pain) is associated with increased pain. Patients with NCCP display low pain thresholds for cardiac-related stress tests (Bradley, Richter, Scarinci, Haile, & Schan, 1992), and NCCP patients' anxiety about pain may negatively impact peak metabolic output in the cardiac-related stress tests (Stein, White, Berman, Covino, & Gervino, under review). Research is needed to examine the possibility that patients with NCCP exhibit elevated cardiac hypervigilance compared to other physical sensations, and to examine whether this elevation is due partly to the association between NCCP and psychiatric disorders, particularly anxiety disorders.

We investigated body vigilance, cardiac anxiety, and the possible mediating role of interoceptive fear in patients with NCCP. First, we aimed to clarify the role of body vigilance – the extent that patients consciously attend to physical sensations – in this sample of NCCP patients. We hypothesized that patients would be more vigilant to cardiopulmonary sensations than other bodily sensations. Next, building on the work of Aikens et al. (1999), we examined the extent to which body vigilance may differ for patients with and without comorbid anxiety disorders. This study extends previous work by examining both conscious vigilance to and fear of physical sensations (i.e., cardiac-congruent and incongruent). We aimed to extend previous work to examine whether NCCP patients were both more vigilant to and more fearful of cardiopulmonary sensations compared to other physical sensations, and whether this difference was associated with the presence of a comorbid anxiety disorder. It was hypothesized that patients with comorbid anxiety disorders would show more body vigilance to cardiac sensations than patients without comorbid anxiety disorders. We theorized that the syndrome of NCCP might persist partly due to hypervigilance to cardiac-congruent body sensations increasing symptom recognition, particularly among anxious patients. Finally, consistent with theoretical models of NCCP, we sought to examine the extent that fear of anxiety-related physical sensations (i.e., interoceptive fear) mediated the impact of body vigilance and cardiac anxiety on pain. We theorized that the possible link between body vigilance and chest pain in the syndrome of NCCP might persist partly because of interoceptive fears, especially among anxious patients.

Method

Participants

Participants were 231 patients seeking evaluation in the cardiology department of a large, university-affiliated medical center. Patients were eligible based on these inclusion criteria: a) At least 18 years of age, b) Chief complaint of chest pain or discomfort, c) Completion of cardiac evaluation including general physical exam and exercise tolerance test, d) Tests indicated no abnormalities during an exercise tolerance test, and e) English language fluency. To enhance generalizability, patients were excluded only if they had a current or lifetime cardiac diagnosis (e.g., CHD, myocardial infarction), or current or recent (last 6 months) uncontrolled severe medical (e.g., diabetes) or psychiatric illness (e.g., active psychosis). Two patients were excluded from the study for failure to meet inclusion criteria at a follow-up data point. A final sample of 229 patients participated in this study.