

## Anxiety and autonomic flexibility: a cardiovascular approach<sup>1</sup>

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### Abstract

Autonomic characteristics of panickers, blood phobics, and nonanxious controls were compared with a variety of cardiovascular measures, including spectral analysis of the cardiac inter-beat interval time series (derived from the electrocardiogram). Responses to laboratory stressors (shock avoidance and cold face stress) of 16 participants who reported recent occurrences of frequent severe panic attacks, 15 participants who reported strong somatic reactions and fainting to the sight of blood, and 15 controls, were recorded. Results suggested distinct autonomic patterns among the three groups. Across conditions, panickers displayed the highest heart rates (HR) coupled with the least HR variability, which indicates low levels of cardiac vagal tone. Blood phobics showed more vagally mediated HR variability than panickers, with a significant association between cardiac rate and mean arterial pressure. Controls generally showed the most HR variability and ‘spectral reserve’ (a quality that indicates flexible responsiveness). Results are discussed in the context of traditional models of anxiety and autonomic activity in contrast to contemporary notions of stability and change in biological systems. © 1998 Elsevier Science B.V. All rights reserved.

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## 1. Anxiety and autonomic dysregulation

Anxiety is often accompanied by somatic manifestations that suggest marked changes in autonomic nervous system (ANS) activity, such as rapid heart rate (HR), shortness of breath, and sweating. These symptoms have frequently been viewed as signs of increased sympathetic (SNS) activation. This interpretation is in accord with Cannon's 'flight-or-fight' model of fear (Cannon, 1929) that is based upon the assumption of global states of SNS activation.

Among the various forms of anxiety, *panic* is singularly marked by the rapid onset of intense somatic symptoms and subjective reports of terror (Barlow, 1988). The presence of recurrent uncued panic attacks is the primary defining feature of panic disorder (PD) (American Psychiatric Association, 1994). Descriptions of this syndrome have an extended history in the medical literature and were often subsumed under the generic label of 'neurocirculatory asthenia' (NCA, see Caranosos, 1974, for review). This diagnosis was given to patients who experienced bouts of severe anxiety accompanied by pronounced somatic disturbances without apparent organic cause. The most frequently reported symptom in these cases was tachycardia. Similarities in symptomology suggest a substantial diagnostic overlap between NCA and PD (Stamper, 1982).

The scope of biological models of panic has been exceedingly broad. However, the salience of reports of tachycardia have implicated SNS disturbances since early investigations of panic syndromes (e.g. Fraser and Wilson, 1918). More contemporary research has been focused at the level of adrenergic neurotransmitter dysfunctions, but has not yielded definitive results (e.g. Grunhaus et al., 1981). More global characterizations have invoked the concept of an autonomic 'imbalance' in the direction of sympathetic dominance in pathological anxiety (e.g. Reich, 1982 (original work published in 1939)). Other broad ANS representations include slow habituation (Lader, 1980), high tonic activation (Roth et al., 1986), and other sundry ANS dysfunctions (for reviews of ANS models of panic, see Mitchell and Shapiro, 1991; Friedman and Thayer, in press).

Of particular bearing on the present study are models which have linked anxiety with excess autonomic lability and reactivity (Eysenck, 1970; Costello, 1971). In such depictions, the ANS is seen as hyperreactive with chronic fluctuations from a steady state. These representations are in need of reconsideration in view of the present state of knowledge of the specificity of ANS regulation of cardiovascular (CV) function, as is detailed below.

### 1.1. Anxiety and autonomic specificity

There are several overarching concerns with the research on the autonomic underpinnings of panic anxiety. One issue is the prevailing emphasis on the SNS and the relative neglect of parasympathetic (PNS) activity. Recent advances in both cardiology and psychophysiology have stressed the importance of vagal influences on CV regulation (e.g. Saul, 1990; Porges, 1995). Furthermore, studies of adrenergic

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