



Voluntary hyperventilation in the treatment of panic disorder—functions of hyperventilation, their implications for breathing training, and recommendations for standardization

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Abstract

Hyperventilation has numerous theoretical and empirical links to anxiety and panic. Voluntary hyperventilation (VH) tests have been applied experimentally to understand psychological and physiological mechanisms that produce and maintain anxiety, and therapeutically in the treatment of anxiety disorders. From the theoretical perspective of hyperventilation theories of anxiety, VH is useful diagnostically to the clinician and educationally to the patient. From the theoretical perspective of cognitive–behavior therapy, VH is a way to expose patients with panic disorder to sensations associated with panic and to activate catastrophic cognitions that need restructuring. Here we review panic disorder treatment studies using breathing training that have included VH. We differentiate the roles of VH in diagnosis, education about symptoms, training of breathing strategies, interoceptive exposure, and outcome measurement—discussing methodological issues specific to these roles and VH test reliability and validity. We propose how VH procedures might be standardized in future studies.

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1. Introduction

Hyperventilation has a long history of being linked to anxiety. Breathing-related symptoms such as dyspnea,¹ shortness of breath, or feelings of suffocation are central features of the experience of panic. This is reflected in the criteria for panic disorder in the *Statistical Manual for Psychiatric Disorders* (DSM-IV; *American Psychiatric Association, 1994*). Additional symptoms on this list are dizziness and tingling sensations in the extremities, both of which are typically produced by hyperventilation-induced alkalosis.² Subjective anxiety has been associated with hyperventilation³ both in individuals suffering from anxiety disorders and in those who do not (e.g., *Rapee & Medoro, 1994; Rapee, Brown, Antony, & Barlow, 1992*). These links have motivated researchers to examine in a variety of experimental subjects what happens when hypocapnia⁴ is produced by voluntary hyperventilation (VH) (*Antony, Brown, & Barlow, 1997; Gorman et al., 1994; Holt & Andrews, 1989; Nardi, Valenca, Nascimento, & Zin, 2001; Papp et al., 1997; Spinhoven, Onstein, Sterk, & le Haen-Versteijnen, 1992; Wilhelm, Gerlach, & Roth, 2001*). Two prominent psychophysiological models of panic, the hyperventilation model and the false suffocation alarm model, have assigned to abnormalities in respiratory gas exchange a central role in the development of panic and its maintenance (see *Roth, Wilhelm & Pettit, in press; Smoller, Pollack, Otto, Rosenbaum, & Kradin, 1996*, for a review).

The *hyperventilation model* of panic disorder (PD) assumes that at some time in the learning history of the patient, symptoms elicited by hyperventilation led to fear that in turn fuelled further hyperventilation, creating a vicious circle spiraling upwards to panic (*Ley, 1985*). Although it has been difficult to prove the etiological role of hyperventilation in panic (*Roth, Wilhelm, Pettit & Meuret, in press*), a number of findings support its relevance. These include lower basal pCO₂⁵ levels (e.g., *Papp et al., 1997; Roth, Wilhelm, & Trabert, 1998; Salkovskis, Jones, & Clark, 1986*), stronger psychological response to VH (e.g., *Antony et al., 1997; Gorman et al., 1994; Holt & Andrews, 1989*), and slower recovery from VH (*Gorman et al., 1988; Maddock & Carter, 1991; Wilhelm, Alpers, Meuret, & Roth, 2001*) in patients suffering from PD compared to healthy controls. Respiratory abnormalities such as disorganized breathing patterns or frequent sighing have also been observed in individuals with panic disorder (*Abelson, Weg, Nesse, & Curtis, 2001; Martinez et al., 1996; Stein, Millar, Larsen, & Kryger, 1995*). A specificity of these abnormalities for panic and not other anxiety disorders was evidenced in some studies (e.g., *Wilhelm et al., 2001; Wilhelm, Trabert, & Roth, 2001a, 2001b*).

However, not all results support the hyperventilation model. Hypocapnia can be absent in individuals with panic disorder during baselines⁶ (*Holt & Andrews, 1989; Woods et al., 1986*), and even during naturally occurring panic attacks (*Garssen, Buikhuisen, & van Dyck, 1996; Hibbert & Pilsbury, 1988*). *Van den Hout et al. (1992)* observed hypocapnia at baseline with further decreases during an exciting

¹ Dyspnea is defined as abnormal or uncomfortable breathing in the context of what is normal for a person according to his or her level of fitness and exertional threshold for breathlessness.

² Respiratory alkalosis is a condition of excessive alkalinity of arterial blood, most often associated with respiratory (breathing) disorders. The pH (a measure of acidity or alkalinity) is high and carbon dioxide levels are low.

³ Hyperventilation (overbreathing) is defined as a combination of rate and depth of breathing that is too much for the body's needs at a particular point in time.

⁴ Acute decreases in arterial pCO₂.

⁵ Partial pressure of arterial blood carbon dioxide.

⁶ Quiet sitting periods.

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