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The role of hyperventilation in panic disorder: a response to Ley (1991)

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Summary—Ley (*Behaviour Research and Therapy, 29*, 301–304, 1991) provided a reinterpretation of experimental findings on the efficacy of breathing retraining plus cognitive restructuring in reducing the symptomatology of patients with panic disorder with agoraphobia which were presented in a 1989 article in this journal. On the basis of his reinterpretation, they concluded that our findings supported the central role of hyperventilation in panic attacks. Ley's arguments are discussed and we conclude that his reinterpretation provides new arguments against a hyperventilation theory of panic. Furthermore, recent evidence from empirical studies does not support a central role for hyperventilation in panic attacks.

In a 1989 article in this journal by de Ruiter, Rijken, Garssen & Kraaimaat, we presented findings of a study comparing the effectiveness of three different treatment packages for panic disorder with agoraphobia: breathing retraining plus cognitive restructuring (BRCR), exposure *in vivo* (EXP) and a combination of both (BRCR + EXP). It was hypothesized that BRCR (i.e. training in slow breathing and reattribution of bodily symptoms to hyperventilation) would result in a significant decrease in panic frequency. Exposure *in vivo* was expected to lead to a reduction in avoidance behavior, and BRCR + EXP was expected to yield the largest reduction in panic frequency and agoraphobic avoidance. The results failed to show a differential effect of the different treatment methods, and BRCR had no significant effect on panic frequency. We thus concluded that the role of hyperventilation in panic attacks was perhaps less important than previously assumed.

Ley (1991) provided a reinterpretation of our results and concluded—contrary to our conclusion—that the findings of our study support the central role of hyperventilation in panic attacks. In this paper, we demonstrate that Ley's reinterpretation provides no support for his hyperventilation theory of panic. Furthermore, we show that the assumptions on which the hyperventilation theory rest are not supported by existing empirical evidence.

Discussion of Ley's Reinterpretation of our Findings

First, Ley indicated that we were mistaken in our application of a two-tailed t-test when testing the hypothesis that breathing retraining plus cognitive restructuring would lead to a decrease in frequency of panic attacks. Our two-tailed t of 1.82 (d.f. = 11) was nonsignificant at an α level of 0.05, but Ley's one-tailed t of 1.82 (d.f. = 11) did reach the 0.05 level of significance. His criticism on this point is valid—a directional hypothesis calls for a directional test. However, we wish to point out that the efficacy of BRCR is rather limited when the remaining self-report outcome measures are considered. Of 8 self-report scales measuring psychological distress (agoraphobic fear and avoidance, general anxiety, depression, somatic complaints), only the scale assessing fear of bodily sensations shows a significant decrease from pre- to posttherapy [$t(11) = 2.13$, $P < 0.05$, one-tailed; see Table 1 in de Ruiter *et al.*, 1989].

Second, Ley discusses our finding that the BRCR treatment has led to a decreased respiratory rate (RR) and a decline in end tidal pCO_2 ($petCO_2$). From the latter finding, he correctly deduces that minute volume (RR × tidal volume) must have increased. As RR decreased, tidal volume must also have increased (we only measured RR and $petCO_2$). So, patients apparently learned to breathe slower but also started to breathe deeper, resulting in a decreased $petCO_2$ posttherapy. The latter effect, an increase in ventilation, is contrary to the purpose of breathing retraining. Ley suggested that it may have been caused by exclusive attention to reducing the breathing rate. We will not argue this suggestion, but we would like to point out that Ley's reasoning is inconsistent with his hyperventilation theory of panic. First, he stresses that our BRCR treatment was successful, contrary to our conclusion, and that his reinterpretation supports his hyperventilation theory of panic. However, he subsequently concludes that we were unsuccessful in reducing our patients' tendency to hyperventilation. Ley overlooks the fact that the latter conclusion contradicts his hyperventilation theory. A cognitive interpretation provides a better explanation for our findings: the decrease in panic attack frequency was accompanied by a significant decrease in fear of bodily sensations (see above). It seems therefore to have been the cognitive component rather than the breathing component of the BRCR treatment that effected the change in panic frequency.

Since our study failed to provide support for the hyperventilation theory of panic attacks, we will examine the assumptions of this theory closer.

The Hyperventilation Theory of Panic

Ley (1991) summarizes the assumptions of his hyperventilation theory of panic attacks on pp. 302–303 of his paper. These assumptions are the following: (1) dyspnea and tachycardia, as the foremost symptoms during panic attacks, are the consequence of hyperventilatory hypocapnia; (2) panic fear is a consequence of dyspnea (i.e. the fear of suffocation), and (3) catastrophic thoughts during panic are the result of cerebral hypoxia due to hyperventilation.

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Table 1. Average percentage of symptoms associated with panic attacks in three studies

Study	Aronson*	Barlow†	Barlow†	Telch‡	Telch‡
sample	PD/PDA	PD	PDA	PD	PDA
n of sample	46	17	41	35	39
Dyspnea	71.7	82	90	85.7	94.9
Dizziness, faintness	82.6	100	95	85.7	92.1
Palpitations	84.8	88	98	97.1	97.4
Trembling	73.9	94	88	85.7	94.9
Sweating	82.6	94	93	88.2	92.1
Choking	54.4	54	73	68.6	71.1
Nausea				50.0	61.5
Derealization	73.9	94	68	82.9	87.2
Tingling	58.7	71	63	77.1	69.2
Flushes	65.2	82	85	68.6	87.2
Chest pain	54.4	71	76	74.3	82.1
Fear of dying	67.4			74.3	78.9
Fear of losing control	69.5	100	90	77.1	79.5

*Aronson and Logue (1988).
†Barlow *et al.* (1985).
‡Telch *et al.* (1989).
PD = panic disorder; PDA = panic disorder with agoraphobia.

We conducted a review of the literature to establish whether dyspnea and tachycardia are indeed the foremost (in terms of frequency and severity) symptoms during panic atttacks. Ley (1985) based the formulation of his theory on 10 *S*s who all experienced dyspnea during their panics. Since then, a number of studies on the phenomenology of panic have been conducted (Aronson & Logue, 1988; Barlow, Vermilyea, Blanchard, Vermilyea, DiNardo & Cerny, 1985; Norton, Dorward & Cox, 1986; Telch, Brouillard, Telch, Agras & Taylor, 1989). These studies provide information on the occurrence frequency of all DSM-III(-R) panic symptoms, and some also give severity ratings. This information is summarized in Tables 1 and 2.

From Table 1, it is readily apparent that dyspnea and palpitations are symptoms which are reported frequently during panic attacks. However, there are three other symptoms that are reported with similar frequency: trembling, sweating and derealization.

Three of the studies reported severity ratings of panic symptoms. Table 2 summarizes these findings. The symptom of dyspnea was not rated most severe in any of the studies. In general, the symptoms of dizziness, palpitations and trembling were rated as more severe than dyspnea. In terms of severity, dyspnea ranked 7th in the Norton *et al.* (1986) study, 4th in the Barlow *et al.* (1985) study, and 6th in the Telch *et al.* (1989) study. Thus, these data do not provide support for a central role for the symptom of dyspnea in panic attacks, neither in terms of frequency nor severity.

Assumption 1 also maintains that the symptoms of dyspnea and tachycardia are caused by hyperventilation. This part of the assumption can be questioned on the basis of physiological studies. Hyperventilation may lead to bronchoconstriction and dyspneic symptoms only in asthmatic patients. Experimental findings suggest, however, that these effects are not triggered by the hypocapnic state but are caused instead by cooling or water loss during overbreathing (Lockhart, Regnard, Dessanges, Florentin & Lurie, 1985). That hyperventilation causes dyspnea by means of a physiological mechanism has never been demonstrated in non-asthmatic *S*s. It is more plausible that dyspneic symptoms such as breathlessness, feelings of pressure in the chest or suffocation, lead to hyperventilation, rather than vice versa. We have suggested elsewhere (Garssen & Rijken, 1986) that such symptoms may be the consequence of long-standing excessive use of the upper chest muscles in breathing.

Tachycardia occurs in response to voluntary hyperventilation in nearly all *S*s, both patients and healthy *S*s. However, it is doubtful that this is caused by the hypocapnic state, since heart rate does not change with artificial overventilation, despite alterations in arterial pCO_2 and pH (Feinberg, Gerola & Katz, 1960; Wollman, Smith, Stephen, Colten, Gleaton & Alexander, 1968). Moreover, tachycardia is found during voluntary hyperventilation, even when hypocapnia is prevented

Table 2. Severity of symptoms reported by panic patients in three studies

Study	Norton*	Barlow†	Barlow†	Telch‡	Telch‡
sample	PD	PD	PDA	PD	PDA
n of sample	58	17	41	35	39
Dyspnea	1.03	1.75	1.67	1.69	1.95
Dizziness, faintness	1.55	1.76	1.99	1.63	2.29
Palpitations	2.36	2.21	2.29	2.29	2.46
Trembling	1.93	1.71	1.70	1.89	2.26
Sweating	1.69	1.44	1.48	1.47	1.89
Choking	0.55	1.06	1.32	1.17	1.53
Nausea			1.14	1.26	
Derealization	0.97	1.82	1.24	1.80	2.18
Tingling	0.74	1.00	0.74	1.41	1.49
Flushes	1.36	1.41	1.67	1.29	1.79
Chest pain	0.97	1.29	1.28	1.60	1.95
Fear of dying	1.32	1.83	2.03		
Fear of losing control		2.50	2.06	1.74	1.85

*Norton *et al.* (1986).
†Barlow *et al.* (1985).
‡Telch *et al.* (1989).
PD = panic disorder; PDA = panic disorder with agoraphobia.
Norton *et al.* used a 5-point scale of severity (0 = none, 4 = very severe). Barlow *et al.* and Telch *et al.* made use of 4-point scales (0 = none, 3 = severe).

by CO_2 inhalation (Burnum, Hickham & McIntosh, 1954; Hille, Hild, Mechelke & Barth, 1961). Thus, tachycardia seems to be induced by increased breathing effort and/or activation of the autonomic centers in the brainstem which accompany the respiratory activation, and not by hyperventilation itself.

Ley's second assumption states that panic fear is a result of the perception of uncontrollable dyspnea (i.e. the threat of suffocation). We examined studies in the literature that sought to determine the content of cognitions during panic attacks to ascertain whether the fear of suffocation was prominent among them. Telch *et al.* (1989) asked patients with panic disorder (PD) and panic disorder with agoraphobia (PDA) to rate the severity of a number of perceived consequences of panic (physical, social and loss of control consequences). Of five physical consequences (heart attack, fainting, stroke, suffocation and death), suffocation ranked 4th after fainting, heart attack and death. Rachman, Levitt and Lopatka (1987) asked 20 PD patients to enter a fearful situation in several standardized behavioral tests. They found that the fearful cognitions of passing out, acting foolishly, losing control and panic were the most frequent cognitions, i.e. occurring in 40% of panic attacks. The fearful cognition of suffocation was reported in $<10\%$ of panic attacks. Both the Telch *et al.* and the Rachman *et al.* studies point out that fear of suffocation is not very prominent among the fearful cognitions that PD patients experience during panic.

The third assumption of the hyperventilation theory of panic states that catastrophic thoughts during panic result from cerebral hypoxia. Elsewhere Ley (1989) has argued that this assumption derives support from studies documenting the effects of hyperventilation on mental functioning (e.g. reaction time, motor coordination, word-association tests). First, we would like to argue that it is a rather large extrapolation from studies of mental functioning during induced hyperventilation to the idea that certain specific catastrophic thoughts are caused by hyperventilation. Decreased cognitive performance is not the same as catastrophic cognitions. Secondly, it seems doubtful that the studies cited in Ley's 1989 article convincingly demonstrate a hyperventilation effect on cognitive functioning. Most of these studies did not use a control condition to monitor whether the effects are caused by hypocapnia or by the distraction of performing breathing manoeuvres. Moreover, in most of the tests used to measure cognitive performance, *S*s had to perform some kind of motor response. Muscle functioning may be hindered by the tetany induced by hyperventilation (Gibson, 1978), which lends uncertainty to whether cognitive or muscle deficits were demonstrated.

In summary, we would like to conclude that studies in the literature do not support the three assumptions underlying Ley's hyperventilation theory of panic.

Other Studies Offering Evidence Against the Hyperventilation Theory of Panic

Several recent studies provide results which are out of line with the idea of a causal role for hyperventilation in panic attacks. Hibbert and Pilsbury (1989) compared symptoms of panic attacks with and without hyperventilation, using long-term ambulatory monitoring of transcutaneous pCO_2. They found no difference between the two types of panic attacks in terms of types of symptoms. If hyperventilation is an important symptom-producing mechanism in panic attacks, one would expect to find a difference between panic attacks with and without hyperventilation. In a recent study, we gathered data on eight patients with panic disorder and agoraphobia who had at least one panic attack during ambulatory monitoring of their transcutaneous pCO_2. None of these patients showed hyperventilation during panic (Buikhuisen & Garssen, 1990).

Hibbert and Chan (1989) compared the efficacy of breathing retraining without cognitive interventions with a placebo treatment for panic disorder. The placebo treatment consisted of discussions with a therapist on the role of stress and personality traits in the onset of anxiety disorders, but no coping techniques were offered. Breathing retraining did not differ significantly from placebo treatment on any of the self-report measures (frequency and severity of panic attacks, Fear Questionnaire, symptoms during panic attacks, Beck Depression Inventory). There was a significant treatment effect on observer ratings of anxiety. However, when Hibbert and Chan compared the magnitude of their treatment effect to that of an earlier study using breathing retraining plus cognitive therapy (Clark, Salkovkis & Chalkley, 1985), the effect was about half that of this previous study. Thus, the cognitive component seems to be of crucial importance.

In a recent article (Garssen, de Ruiter & van Dyck, 1992), we provided a review of studies comparing the effectiveness of breathing retraining with other techniques not directly aimed at changes in breathing pattern (e.g. relaxation training, stress management) for patients with hyperventilation syndrome (HVS) and/or panic disorder. The review shows that breathing retraining has a therapeutic effect for patients suffering HVS and panic disorder. However, the techniques not aimed at altering breathing pattern were equally effective. Thus, the proven effectiveness of breathing retraining therapies cannot be used to support the central role of hyperventilation in panic attacks. In our paper (Garssen *et al.*, 1992), we termed breathing retraining 'a rational placebo', acknowledging its efficacy but also emphasizing the fact that the treatment does not work according to the principle of allegedly lowering the pCO_2.

The present paper pointed out that the evidence for a hyperventilation theory of panic is rather meager. We do not deny that hyperventilation may be present during some panic attacks, but the existing research literature does not support the notion of a central role for hyperventilation in causing panic attacks.

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