

THE EFFICACY OF BREATHING RETRAINING AND THE CENTRALITY OF HYPERVENTILATION IN PANIC DISORDER: A REINTERPRETATION OF EXPERIMENTAL FINDINGS

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Summary—The present paper addresses de Ruiter, Rijken, Garssen, and Kraaimaat's (*Behaviour Research and Therapy*, 27, 647–655, 1989) interpretation of data pertaining to the efficacy of breathing retraining in the treatment of panic disorder. The proffered reinterpretation of these data makes it clear that breathing retraining led to a significant reduction in the frequency of panic attacks. These findings thus lend additional support to the central role of hyperventilation in primary panic attacks.

The purpose of the present paper is two-fold. The first purpose is to provide a reinterpretation of de Ruiter, Rijken, Garssen and Kraaimaat's (1989) experimentally derived findings pertaining to the efficacy of breathing retraining in the treatment of panic attacks. The second purpose is to explain how this reinterpretation supports the central role of hyperventilation in primary panic attacks.

Reinterpretation of findings

The de Ruiter *et al.* (1989) study was designed to compare the relative effectiveness of three therapeutic approaches to the treatment of panic disorder with agoraphobia: (a) breathing retraining with cognitive restructuring (eight sessions), (b) exposure therapy (eight sessions), and (c) abbreviated breathing retraining with cognitive restructuring (four sessions) plus abbreviated exposure therapy (four sessions). The experimental design employed by de Ruiter *et al.* consisted of repeated measures (pre- and post-treatment) for each of the three treatment groups.

The central criterion variable upon which de Ruiter *et al.* based their primary conclusions was 'mean panic attack frequency/day'. Those data relevant to this criterion variable reported by de Ruiter *et al.* are given in Table 1. From these data, it is clear that the Breathing Retraining Group was the only treatment group that showed a reduction in the frequency of panic attacks following treatment. The issue at this point is the reliability of this improved change. That is, does this mean reduction of 0.46 panic attacks per day (i.e. 3.22 attacks per week) represent a statistically significant difference or is it more reasonable to attribute the reduction to sampling error? de Ruiter *et al.* chose, appropriately, to base their decision and *interpretation* on a *t*-test for repeated measures:

To test the hypothesis that BRCR (Breathing Retraining with Cognitive Restructuring) would result in a significant reduction in panic attack frequency, a paired *t*-test was performed, revealing no significant improvement [$t(11) = 1.82$. NS]. (de Ruiter *et al.*, 1989, p. 652.)

Table 1. Mean number of panic attacks per day before treatment (pre-test) and after treatment (post-test) for each of the three treatment groups

Treatment group	N	Pre-test		Post-test		Mean difference
		Mean	SD	Mean	SD	
Breathing Retraining	12	1.89	2.18	1.43	1.68	0.46
Exposure	12	1.36	1.77	1.36	1.98	0.00
Abbreviated Breathing Retraining and Exposure	13	0.64	0.41	0.63	0.66	0.01

While there is no argument with the statistical test chosen, there is serious reason to question their interpretation that the t of 1.82 (d.f. = 11) indicates 'no significant improvement'. The tabled values of t for 11 d.f. indicate that the t corresponding to an α level of 5% (one-tailed test) is 1.7959, a value less than the 1.82 reported by de Ruiter *et al.* Since the statement of their hypothesis (*viz.* "To test the hypothesis that BRCR would result in a significant reduction of panic attack frequency") makes it explicit that the expected direction of the change would be a decrease in panic attack frequency, a one-tailed t -test is clearly called for.

In an effort to check the appropriateness of a one-tailed test in the present situation, the writer conducted a quasi-random survey of 13 recently published text books in statistics (Arney, 1990; Bohrnstedt & Knoke, 1988; Darlington & Carlson, 1987; Dougherty, 1990; Groninger, 1990; Kenny, 1987; Kirk, 1990; May, Masson & Hunter, 1990; Papoulis, 1990; Philips, 1988; Spence, Cotton, Underwood & Duncan, 1990; Sprinthall, 1990; Witte, 1989). While Spence *et al.* mentioned a controversy concerning one-tailed tests, all 13 confirmed the contention that a one-tailed test is appropriate under conditions in which the alternative to the null hypothesis clearly predicts the direction of the expected outcome. (The exact probability of 13 confirmations in a sample of 13 books is given by the binomial expansion as $1/8192$, i.e. $P = 0.00012$.) It may be of further interest to note that had de Ruiter *et al.* elected to use an analysis of variance solution, the same directional-hypothesis issue would apply (see Ley, 1979).

Since it was conceivable that the published report of $t(11) = 1.82$ might be in error, the present writer requested, and received, the pertinent raw data (de Ruiter, personal communication, 1990). A statistical analysis revealed the same outcome. However, a perusal of the individual panic-attack frequencies for each of the 12 Ss provided additional data to support the significant decrease in frequency of panic attacks following the Breathing Retraining treatment. Table 2 lists the frequency of panic attacks before and after the Breathing Retraining treatment for each of the 12 patients. From this table, it can be seen that of the 12 patients, only two (S6 and S8) reported an increase in the frequency of panic attacks following breathing retraining. The exact probability of two or fewer negative results (the only data that run counter to predictions from a hyperventilation theory) out of 12 is given by the binomial expansion as $13/4096$, i.e. $P = 0.003$. The significant disproportionality indicated by this statistic provides additional evidence to substantiate the significant decrease in the frequency of panic attacks following breathing retraining suggested by the t -test regardless of considerations concerning one-tailed vs two-tailed tests of statistical significance.

The central role of hyperventilation in panic attacks

The theoretical ramifications of the reinterpretation of the t -test that correctly indicates a decrease in panic attacks following breathing retraining are critical. The reason for this lies in basic assumptions of a hyperventilation theory of primary panic attacks (Ley, 1985, 1987, 1989). This theory proposes that dyspnea and tachycardia (the foremost symptoms of panic attacks) and other somatic complaints are the consequence of hyperventilatory hypocapnea, that the panic fear is a consequence of the perception of uncontrollable dyspnea (i.e. the threat of suffocation), and that

Table 2. Mean number of panic attacks per day before treatment (pre-test) and after treatment (post-test) for each of the 12 patients who received the Breathing Retraining treatment

S	Pre-test	Post-test	Difference
1	7.43	5.43	-2.00
2	0.71	0.43	-0.28
3	2.00	1.14	-0.86
4	3.29	2.71	-0.58
5	0.43	0.00	-0.43
6	1.00	2.43	+1.43
7	4.43	3.14	-1.29
8	0.00	0.57	+0.57
9	1.29	1.00	-0.29
10	1.00	0.00	-1.00
11	1.14	0.29	-0.85
12	0.00	0.00	0.00
Mean	1.89	1.43	-0.46
SD	2.18	1.68	0.88

Table 3. Mean end-tidal $p\text{CO}_2$ and mean respiration frequency for baseline, pre-test, and post-test assessments

Respiration measure	Assessment phase					
	Baseline		Pre-set		Post-test	
	Mean	SD	Mean	SD	Mean	SD
End-tidal $p\text{CO}_2$ (mm Hg)	37.7	2.4	39.8	2.5	37.1	4.7
Respiration frequency (bpm)	15.1	3.4	15.1	3.2	12.7	2.3

the frequently reported catastrophic thoughts are the consequence of cerebral hypoxia induced by hyperventilation. Thus, a program of treatment that helps panic patients to exercise voluntary control to reduce ventilation should be effective as a prophylaxis in the prevention of panic attacks or as an ameliorative in reducing the intensity and duration of attacks.

In their Discussion, de Ruiter *et al.* state "the present study found breathing retraining plus cognitive restructuring ineffective in reducing panic" (p. 654). On the basis of their questionable interpretation (whatever the outcome of the *t*-test) they overlooked the discordance between their interpretation and the findings of studies that have demonstrated positive effects of breathing retraining with panic and agoraphobic patients (e.g. Bonn, Readhead & Timmons, 1984; Clark, Salkovskis & Chalkley, 1985; Rapee, 1985; Salkovskis, Jones & Clark, 1986) and hastily dismissed hyperventilation as a central theoretical phenomenon. In the light of these collateral studies and the fact that only two of 12 patients (S6 and S8) reported an increase in the frequency of panic attacks following breathing retraining, any observed effect in the predicted direction should receive cautious consideration before dismissing hyperventilation as an 'epiphenomenon of panic'.

From the point of view derived from a reinterpretation of those data which indicate a significant reduction in the frequency of panic attacks among the disproportionately large number of Ss who experienced a decrease following breathing retraining, the central role of hyperventilation in panic disorder is once again elevated to its position of primacy. The reinterpreted findings of de Ruiter *et al.* can now be added to those of Bonn *et al.* (1984), Clark *et al.* (1985), Rapee (1985), and Salkovskis *et al.* (1986), findings that offer clear support for a hyperventilation theory of panic disorder (Ley, 1985, 1987, 1989).

Breathing retraining

Statistical considerations aside, the relatively small reduction in the frequency of panic attacks following breathing retraining may lie in a faulty procedure. Table 3 gives the means and standard deviations for $p\text{CO}_2$ (end-tidal CO_2 pressure) and respiration frequency reported by de Ruiter *et al.* for the Breathing Retraining group during the assessment phase (baseline, pre-test, and post-test). In keeping with the expected reduction in ventilation that marks the goal of breathing retraining, the mean respiration rate (breaths per minute) shows a significant decline from 15.1 b/min pre-test (before breathing retraining) to 12.7 b/min post-test (after breathing retraining). However, mean $p\text{CO}_2$, which should increase if ventilation is reduced, decreases from 39.8 mm Hg pre-test to 37.1 mm Hg post-test.

Table 4. End-tidal $p\text{CO}_2$ during pre-test (before treatment) and post-test (after treatment) assessments for each of the 12 Ss who receive the Breathing Retraining treatment

S	Pre-test	Post-test	Difference
1	40.1	37.8	-2.3
2	36.0	41.8	+5.8
3	42.0	43.2	+1.2
4	41.0	28.2	-12.8
5	42.4	41.3	-1.1
6	39.1	31.0	-8.1
7	40.2	38.8	-1.4
8	38.0	35.8	-2.2
9	37.9	40.3	+2.4
10	43.3	35.9	-7.4
11	44.2	35.7	-8.5
12	36.8	34.0	-2.8
Mean	40.08	36.98	-3.1
SD	2.61	4.49	5.25

Since ventilation is measured in terms of minute volume (the sum of the tidal volumes of each breath for 1 min), a decline in $p\text{CO}_2$ accompanied by a decrease in respiration rate can only be accomplished by means of an increase in minute volume. In other words, while the patients appear to have learned to reduce their respiration rates, they apparently increased the tidal volumes of their breaths. Thus, excessive concentration of respiration frequency without sufficient attention to tidal volume appears to have resulted in a paradoxical increase in ventilation.

In order to verify this hypothesis, the present writer requested the individual pre- and post-test measures for the Breathing Retraining Group (de Ruiter, personal communication, 1990). These data (see Table 4) reveal a decrease in $p\text{CO}_2$ from pre- to post-test for all but three (S2, S3 and S9) of the 12 patients (binomial probability is 79/4096, i.e. $P = 0.0193$). This reduction in $p\text{CO}_2$ accompanied by a significant decrease in respiration rate makes it clear that the breathing retraining treatment led to an increase, rather than a decrease, in ventilation, at least on the occasion of the assessment trial. [While the overall increase was small, one patient (S4) showed a seriously large decline in $p\text{CO}_2$ from a normal 41.0 mm Hg to an abnormally low hypocapnic level of 28.2.] None the less, the respiratory control learned during the Breathing Retraining treatment helped all but two of the 12 patients to reduce significantly the frequency of panic attacks suffered subsequent to treatment.

These data and the analysis presented here point to the complications of breathing retraining (see Timmons & Ley, 1991). Efforts to reduce ventilation through exclusive attention to a reduction in respiration frequency may not only be unsuccessful in reducing ventilation, but may, as in the study in question, produce a paradoxical increase in ventilation, an effect opposite to the express purpose of breathing retraining. The results presented here indicate that $p\text{CO}_2$ monitoring should be an integral part of breathing retraining programs.

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