

WILL HYPERVENTILATION SYNDROME SURVIVE?: A REJOINDER TO LEY (1993)

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ABSTRACT. *In a reply to our review entitled "Breathing Retraining: A Rational Placebo?," Ley has raised a number of comments (Clinical Psychology Review, 13, pp. 393-408). We do not agree with most of his remarks. Where his comments may be right, his opinion does not contradict our general conclusion. We maintain our conclusion that (a) there are no good reasons for assuming that the effects of breathing retraining are caused by decreasing the tendency to hyperventilate and (b) experimental evidence does not support the hyperventilation syndrome (HVS) model for panic.*

In a recent article in this journal (Garssen, de Ruiter, & van Dyck, 1992), we reviewed 13 studies pertaining to the effects of breathing retraining in patients with panic disorder and/or hyperventilation syndrome (HVS). We discussed the questions of whether clinically relevant effects had been demonstrated in these studies and whether findings support

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the HVS model of panic. Half of the studies were difficult to evaluate because of methodological weaknesses such as small sample sizes and lack of control groups. Other studies incorporated both breathing retraining and cognitive reattribution of somatic symptoms to hyperventilation into a single treatment, so that it was impossible to determine the individual effect of either component. With these reservations, it was concluded that breathing retraining and cognitive reattribution are therapeutically effective. However, their efficacy is probably due to factors other than a decreased tendency to hyperventilate. Our reasoning is that other mechanisms possibly responsible for the therapeutic effects—such as offering plausible explanations for symptoms, relaxation, or increasing the feeling of self-control—could not be ruled out. The few studies that used adequate comparison groups (Hibbert & Chan, 1989; Vlaender-van der Giessen, 1986) did not support the HVS model of panic.

We considered the evidence from these few treatment studies insufficient by itself to dismiss the HVS model, and we reviewed findings from other lines of research to support our sceptical position. Studies pertaining to the occurrence of hyperventilation during panic and to the validity of the diagnostic test for HVS were discussed. These studies did not support the HVS model of panic (see Garssen et al., 1992, pp. 149–150).

Our article elicited a critical response from Ley (1993). He raises a number of arguments, which can be summarized as follows:

1. The cognitive HVS model for panic is contradicted by experimental evidence.
2. The division we made when discussing treatment studies between pure breathing techniques and cognitive interventions is inadequate.
3. Some treatment studies were inadequately described.
4. The role of hyperventilation in panic should be discussed in light of Ley's own recently proposed typology of panic attacks.
5. Our comment on the validity of the generally accepted procedure for diagnosing HVS—namely, recognition of symptoms induced by voluntary hyperventilation—is unfounded.

These five points of criticism are examined below. We do not agree with most of Ley's comments. Some of his comments give rise to interesting discussion. Because of the detailed nature of Ley's comments, we at times also go into details.

THE COGNITIVE HVS MODEL OF PANIC

We have described the HVS as a model because it concerns a suggested relationship, not an observable phenomenon—namely, the relationship between reported symptoms and (rarely observed and most often inferred) hyperventilation. We named two cognitive elements, which may be of importance in this relationship: (a) catastrophic interpretation of sensations and (b) anxious anticipation of new attacks. They form part of the circular model depicted in our article (p. 146). Ley questions the validity of this circular model, which he holds untenable. His criticism particularly focusses on the role of catastrophic interpretation of symptoms.

We made the following statement in our review article, which may further clarify our position: “The model has been extended to also include cognitive elements, which means that hyperventilation is seen as *a necessary factor for the development of the majority of somatic symptoms* [italics added], but not as a sufficient factor itself for developing panic” (p. 143). Discussion of the italicized portion of the statement was central in our 1992 article. Ley addressed his criticism to the remaining part (i.e., the cognitive elements). However,

even if all panic attacks developed without any preceding catastrophic cognitions, the (central) question of whether hyperventilation is an important factor in panic would still remain.

Ley cites the study of Rachman, Lopatka, and Levitt (1988) (actually cited incorrectly as Rachman, Levitt, and Lopatka, 1987), which seems to indicate that 27% of 30 panic disorder patients were unable to indicate catastrophic cognitions, leaving 73% who indicated fearful cognitions about somatic symptoms. Although expressing some doubts about the cognitive model, Rachman et al. concluded that their findings "are mainly consistent with Clark's (1986) cognitive theory of panic" (p. 39). Subsequently, Ley refers to his typology of panic attacks. One of Ley's three types of panic attack is "a Type III or cognitive panic attack." He indicated that "the cognitive attack seems to be growing disproportionately [in number] to the hyperventilatory attack . . ." (Ley, 1993, p. 396). Thus, the critic of the circular model himself introduces the cognitive panic attack. However, Ley ends his discussion by concluding that "the validity of the circular model of hyperventilatory panic attacks is highly questionable, if not completely untenable" (Ley, 1993, pp. 396-397). We fail to understand the justification of making such a sharp distinction between "pure" noncognitive hyperventilation (Type I) attacks and cognitive panic (Type III) attacks, as Ley proposes.

PURE BREATHING TECHNIQUES VERSUS COGNITIVE INTERVENTIONS

We made a division between pure breathing techniques and cognitive interventions on the basis of the procedures described in the articles reviewed. Ley rightly argued that breathing retraining always contains the cognitive element of suggesting to patients an understandable and manageable cause of their problems. Yet, we do not understand why this point is "critical." Ley's position makes the HVS model of panic (implying that hyperventilation is a necessary factor for symptom development) more problematic. According to his comment, the cognitive factor may be the effective element in all studies on the effect of breathing retraining.

SOME TREATMENT STUDIES INADEQUATELY DESCRIBED?

Ley's comment concerns four of the studies we described in our review. Regarding the first study, the study of Lum (1983), we mentioned only its therapeutic effects and remarked that details were lacking for an adequate evaluation. This rejoinder provides us with the opportunity to state that we consider Lum to be a highly skilled practitioner and scholar. However, this regard should not hinder an objective evaluation of his publications, and Lum himself might feel somewhat embarrassed by Ley's placing him in the ranks of figures of such magnitude as Darwin, Freud, James, Pavlov, Piaget, Sherrington, and Skinner.

Ley's second comment, concerning our description of the study of Bonn, Readhead, and Timmons (1984), is difficult to grasp. It seems that Ley's objections are that (a) we denied that the experimental and control groups in this study differed with regard to posttreatment assessment, (b) we did not mention the findings of the 6-month follow-up, and (c) we forgot to give the exact data on panic frequency.

Our summary of the study of Bonn et al. (1984) reads as follows:

Results showed no difference between the two therapy conditions at posttreatment. However, at 6-month follow-up, the respiratory control group showed further improvement, whereas the group that had received exposure alone had deteriorated. The respiratory control

group had significantly lower resting breathing rate and panic attack frequency, and scored significantly better on measures of somatic symptoms and agoraphobia. (p 148)

It is instructive to compare our summary with that of Bonn et al. (1984), which reads:

At discharge and at 1 month's follow-up the two groups of patients showed similar degrees of improvement in frequency of panic attacks and other psychophysiological scores. At 6 months' follow-up the patients treated with real-life exposure alone were beginning to show a fall-off in relearned adaptative behavior, whereas those given breathing retraining showed further improvement. (p. 665)

There are no differences between Bonn and colleagues' summary and our own, and we consider Ley's suggestion that we did not adequately describe this study unfounded.

Ley's third comment concerns our own treatment study (de Ruiter, Rijken, Garssen, & Kraaimaat, 1989), on which Ley had previously commented (1991). We published a rejoinder (de Ruiter, Garssen, Rijken, & Kraaimaat, 1992). Since Ley seems not to have taken the trouble to read our comment, we will reiterate some of the main issues of this discussion.

In our study, we compared three different treatment packages: breathing retraining plus cognitive restructuring (BRCR), exposure in vivo (EXP), and a combination of both (BRCR + EXP). Assessment included eight self-report measures, panic frequency, and two respiratory measures (respiratory rate and end-tidal PCO₂). Two types of statistical analyses were followed. The first was analysis of covariance (ANCOVA), using the baseline as a covariate, for all 11 measures. Most important were the interaction effects for Groups × Pre-Post, which were only significant for two avoidance measures, with α set at .05. However, these interaction effects were nonsignificant when using a Bonferroni correction for multiple tests. So, according to the first test, there were no differences in therapeutic outcome between the three groups.

Second, we tested the specific hypothesis that BRCR would result in a reduction in panic attack frequency. We performed a two-tailed test and found a nonsignificant result. Ley (1991) commented that a one-tailed test is indicated when testing a directional hypothesis; this one-tailed test yielded a significant result. We have already agreed that his criticism was valid on this point (de Ruiter et al., 1992). However, one must bear in mind that the statistical test criticized applied to changes in panic frequency *within* the BRCR group; the between-groups comparison of panic frequency using an ANCOVA was nonsignificant, as indicated above. We admit the validity of his remark that the power of our study was low with samples of only 12 subjects per group.

Next, Ley questions our statement that "combination therapy [in our 1989 study] was no more effective on any outcome variables at posttest than exposure alone, similar to the earlier findings of Bonn et al. (1984)" (p. 148). Bonn et al. did not find any intergroup differences at posttreatment, and neither did we. What is wrong with our conclusion?

In our previous comment (de Ruiter et al., 1992) we noted that Bonn et al. did find differences at follow-up, and we ended in announcing a follow-up study of our own patients. This study was published earlier this year (Rijken, Kraaimaat, de Ruiter, & Garssen, 1992) and revealed no differences in outcome measures between treatment packages.

Ley's next point concerns the respiratory data. We found a significant pre- to post-treatment reduction in respiratory frequency and also "a paradoxical *reduction* in end-tidal PCO₂ (the opposite of the intended purpose of breathing retraining) . . ." according to

Ley (1993, p. 399). We found, however, *no significant differences* between pre- and post-treatment values of PetCO₂.

In addition to the points above, we would like to emphasize that the most important argument of our 1992 rejoinder was that "Ley's reasoning is inconsistent with his [own] 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 hyperventilate. Ley overlooks the fact that the latter conclusion contradicts his hyperventilation theory" (de Ruiter et al., 1992, p. 643).

The fourth study is that of Hibbert and Chan (1989). In this study two treatment packages for panic disorder patients were compared. The first consisted of two sessions of breathing retraining (BR) and three sessions of BR and anxiety management (AM). The second group received two sessions of "placebo treatment" and three sessions of AM (without BR). Hibbert and Chan divided their patients into "hyperventilators" and "non-hyperventilators" on the basis of number of recognized symptoms during the hyperventilation provocation test, using a numerical criterion. Hibbert and Chan concluded that the hyperventilators did not benefit more from respiratory treatment than the nonhyperventilators.

Ley introduces the following example to counter the numerical criterion of Hibbert and Chan: "A problem with the definitions might be a patient who recognizes only the single symptom of tachycardia because the fear of an imminent heart attack is so intense that it overshadows all other symptoms experienced . . ." (Ley, 1993, p. 400). This condition might occur if the investigator only relied on spontaneous symptom reporting by the patient, but is highly improbable if a symptom checklist is used, as in the study of Hibbert and Chan.

Ley summarizes his criticism by stating "the most important point to be made here is that the patients in *both* [italics added] of Hibbert and Chan's (1989) groups . . . benefited from the breathing retraining" (p. 401). This assertion is false, first because *only one* group—not *both* groups—received breathing retraining, and second because there were no significant differences in self-report measures between the breathing retraining and placebo treatment groups at posttreatment.

THE ROLE OF HYPERVENTILATION IN PANIC

Ley suggests that three types of panic should be distinguished, and that a drop in PCO₂ is only expected in the first type. Ley argued that our conclusion that hyperventilation is an unimportant mechanism in producing panic attacks would be only correct for Type II and III panic attacks. We tried to find some indications of the prevalence of Type I attacks.

Ley presented a table showing the supposed differences between the three types. For the next discussion, we have chosen from his table the following differences between a Type I attack and the two other types of attack: (a) dyspneic fear, (b) unreal or bizarre thoughts, and (c) more intense symptoms of dyspnea and palpitations.

It is highly probable that Type I panic attacks, when based on these criteria, will occur frequently. In our earlier rejoinder, we presented data from five different studies on the frequency of occurrence of panic symptoms in panic disorder patients. Dyspnea is reported by 72–95% of the patients, palpitations by 85–98%, choking by 54–73%, and derealization (which may be equated with "unreal" thoughts) by 68–94%. As most panic attacks seem to fulfill the criteria for Type I attacks, the discussion about the role of hyperventilation in panic attacks in general is back to its starting point.

THE VALIDITY OF THE HYPERVENTILATION PROVOCATION TEST (HVPT)

The discussion here relates to two studies, that of Roll and Zetterquist (1990) and that of Hornsveld, Garssen, Fiedeldij Dop, and van Spiegel (1990). In both studies recognition of symptoms was compared in the HVPT and a nonhyperventilatory control test, the Stroop Color Word Test (ST). A comparable number of patients recognized symptoms during both tests. This finding seriously undermines the validity of the HVPT.

Roll's criterion was recognition of three or more symptoms. We performed two analyses using two different criteria—namely, four or more symptoms recognized and “overall recognition.” The findings were similar for both methods.

Ley disapproves of the use of the “arbitrary” numerical criterion for recognition in Roll's study. However, this criticism was already addressed by our application of the numerical criterion and the criterion of overall recognition, which led to similar results.

We divided symptoms into several categories, and used the following three categories in data analysis; (a) symptoms belonging to the HVS in the restricted sense (i.e., symptoms which may be pathophysiologically related to hyperventilation, such as paresthesias and dizziness); (b) general arousal symptoms (i.e., symptoms whose pathophysiological relationship to hyperventilation is less clear, such as rapid heartbeat and sweating); and (c) dummy symptoms which are unlikely to be produced by hyperventilation, such as low back pain and itching. The total symptom score, and the scores for the HVS in a restricted sense and for general arousal symptoms, were higher for the HVPT than for the ST, both with respect to induced and recognized symptoms. The absence of a difference in dummy symptoms was explained by the fact that they were scarcely experienced in both tests.

In his comment Ley mentions the outcome of a number of statistical tests. Ley's presentation may give the reader the impression that we overlooked an important issue. We wish to stress that these results were fully presented and discussed in our article.

To evaluate these findings one has to consider the following arguments: First, there is no arguing the potency of the HVPT in evoking physiological responses and somatic sensations. It is difficult to find a nonhyperventilatory control condition of comparable potency. This is not an argument in favor of the HVS model, but was a weakness in the experimental design. Second, it is probable that voluntary hyperventilation will induce a somewhat different pattern of physiological responses and somatic sensations than a nonhyperventilatory stressful task, which is also not an argument in favour of the HVS model.

Our findings are remarkable *despite* these expected differences. There was a significantly higher number of (induced and recognized) symptoms in the HVPT versus the ST, *both* in the category of symptoms probably related to hyperventilation and in general arousal symptoms. Moreover, the degrees of recognition for both symptom categories were virtually identical within both tests (the questionnaire contained 9 items of the first symptom category and 15 items of the second category; this difference was taken into account). The scores for recognized symptoms during the HVPT were .28 for symptoms probably related to hyperventilation and .21 for general arousal symptoms (range 0–1). The scores during the ST were .12 for symptoms possibly related to hyperventilation and .14 for general arousal symptoms.

Thus, Ley concluded on the basis of the same set of data that both tests differed in potency, which is true but irrelevant with respect to a discussion of the HVS model. We found it remarkable that, despite differences in potency, the degree of overall recognition was similar for both tests, and that patients seemed to base recognition on experiencing symptoms *irrespective* of their theoretical relationship to hyperventilation.

In our 1990 article we referred to the study of Svebak and Grossman (1985), which demonstrates that the potency of the test, and not so much its physiological character, seems to determine symptom reporting. Svebak and Grossman compared reactions to a HVPT and a video game, the latter with and without the threat of an aversive shock. The number of symptoms recognized was larger in the hyperventilation condition than in the control task. The number of symptoms was, however, also larger in the threat than in the no-threat condition of the video game (both without hypocapnia). This indicates that psychological manipulation induced differences similar to manipulation of PCO_2 .

Recently we replicated the Hornsveld et al. study using the more appropriate control condition of the "isocapnic overbreathing test" (IOT; Hornsveld, Garssen, & van Spiegel, 1992). During the IOT, the subject follows the instructions of overbreathing similar to the HVPT, but a drop in end-tidal PCO_2 is prevented by adding CO_2 to the inspiratory air. Sixteen patients with multiple unexplained symptoms performed both tests in random order. They were told that they were to perform the HVPT twice. Although the IOT induced significantly fewer symptoms than the HVPT, the number of patients who recognized their daily life symptoms was equal in both tests (about 70% of the patients).

Ley's last remark about the significant $PetCO_2$ decrease in our control condition is pointless. Ley knows that the reported decrease of 1.2 mmHg is too small to have any effect on the production of symptoms.

CONCLUSION

The HVS model of panic is an old idea. In the historical section of our review, we mentioned the study of Kerr, Dalton, and Gliebe (1937) as the starting point. The validity of this old idea is recently questioned, not only by us, but by several authors on the basis of a considerable amount of experimental data (Griez, Zandbergen, Lousberg, & van den Hout, 1988; Hibbert & Pilsbury, 1989; Spinhoven, Onstein, Sterk, & Le Haen-Versteijnen, 1992; van den Hout et al., 1992; Zandbergen, de Loof, Pols, & Griez, 1990). It is to be expected that our comment on an established theory will provoke response. Ley has established himself as a defender of the HVS model.

We do not agree with most of his remarks, as was explained above. Where his comments may be right, especially with respect to his second point, his opinion does not contradict our general conclusion. We maintain our conclusion that (a) there are no good reasons for assuming that the effects of breathing retraining are caused by decreasing the tendency to hyperventilate and (b) experimental evidence does not support the HVS model for panic, whether panic is defined as a Type I, Type II, or Type III attack.

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