BREATHING RETRAINING:
A RATIONAL PLACEBO?

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ABSTRACT. *Breathing retraining of patients with Hyperventilation Syndrome (HVS) and/or panic disorder is discussed to evaluate its clinical effectiveness and to examine the mechanism that mediates its effect. In relation to this theoretical question, the validity of HVS as a scientific model is discussed and is deemed insufficient. It is concluded that breathing retraining and related procedures are therapeutically effective, but probably due to principles other than originally proposed, namely decreasing the tendency to hyperventilate. An alternative principle is the induction of a relaxation response, presenting a credible explanation for the threatening symptoms, giving a distracting task to practice when panic may occur, and promoting a feeling of control.*

In recent years, new attention has been given to the Hyperventilation Syndrome (HVS) as a scientific model that may explain some of the various somatic symptoms reported by anxiety disorder patients. Several authors have advanced the view that hyperventilation is the mechanism underlying some or most of the panic attacks experienced by patients with panic disorders or agoraphobia (Bass, Lelliot, & Marks, 1989; Cowley & Roy-Byrne, 1987; Garssen, van Veenendaal, & Bloemink, 1983; Holt & Andrews, 1989; Ley, 1985; Lum, 1981; Rapee, 1987; de Ruiter, Garssen, Rijken, & Kraaimaat, 1989).

Hyperventilation is defined as a higher degree of ventilation than is necessary to meet the demands of the body, caused by an increase in respiratory rate and/or depth. This

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excessive ventilation leads to a decrease in alveolar and arterial carbon dioxide pressure (PCO_2) and an increase of pH in the blood and the cerebrospinal fluid (respiratory alkalosis). Secondary physiological changes are induced if hyperventilation is strong enough and is sustained for some time (Garssen & Rijken, 1986), the most important ones being: constriction of the arteries in the brain and in the hands, increased neural excitability, increased production of lactic acid, and lowering of phosphate level in arterial blood. HVS is defined as a set of symptoms largely produced by a lowering of arterial PCO_2. The pathophysiological mechanism can be indicated for some of the HVS symptoms. For instance, dizziness and cold hands can be caused by the hyperventilation-induced vasoconstriction in the brain and the hands respectively, and tingling sensations can be produced by the increase in nervous excitability. However, it is incorrect to argue the other way around: dizziness, cold hands, and tingling may be produced by physiological factors other than hyperventilation.

Several treatment procedures have been developed based on this model. They are aimed at changing the breathing pattern to lower the chance of hyperventilation. Studies of breathing retraining therapies will be reviewed to evaluate their clinical effectiveness and to examine the mechanism that mediates success. We will focus on the question of whether therapeutic results are achieved by means of an inhibition of the tendency to hyperventilate or by mechanisms not specifically related to hyperventilation.

Furthermore, doubts have been raised about the significance of hyperventilation as a symptom producing mechanism in panic. Studies relevant to this topic will also be discussed. We start with a review of the development of the HVS model, in order to place these therapies in a historical context.

DEVELOPMENT OF THE HVS MODEL

In 1908, Haldane and Poulton extensively described hyperventilation and its physiological effects. They also mentioned the symptoms that may be induced: paraesthesia, muscle stiffness or cramps, and dizziness. Since then, numerous investigations have focused on the physiological consequences of hyperventilation (Brown, 1953; Weimann, 1968).

Some decades later, Kerr, Dalton, and Gliebe (1937) related the manifold somatic symptoms of anxious patients to hyperventilation. The variety of symptoms reported by these patients, such as breathlessness, tightness in the chest, pounding of the heart, tachycardia, precordial pain, dizziness, tingling, sweating, nausea, fatigue, nervousness, and anxiety, have always puzzled investigators. To ascribe them to a lowering of arterial PCO_2 seemed reasonable because of the abnormal breathing behavior often seen in these patients (White & Hahn, 1929) and because symptoms could be partly reproduced by voluntary hyperventilation (Kerr et al., 1937).

The clinical picture presented by patients reporting these symptoms had already been described by Da Costa in 1871. The syndrome has been given different names, such as Da Costa Syndrome, anxiety neurosis, effort syndrome, neurocirculatory asthenia, heart phobia, and irritable heart. These various labels reflect special interests in particular aspects of the clinical picture. Da Costa considered physical exhaustion a major cause of these symptoms. The interest in exhaustion, recurring in the name "effort syndrome," led to investigation of reactions to physical exercise in patients with anxiety neurosis. Accumulation of lactate in the blood is one of these physiological reactions, and Pitts and McLure (1967) supposed that the symptoms of anxiety neurosis were caused by an excessive lactate response or an abnormal reaction to lactate. A remnant of this line of research is the procedure of experimental induction of panic by means of sodium lactate infusion. However, lactate is no longer believed to play a role in producing spontaneously occurring symptoms.

Clinical reviews of HVS have been regularly published since 1937 (Garssen & Rijken, 1986; Lum, 1976; Magarian, 1982; Rice, 1950; Weimann, 1968). The physiological effects of hyperventilation are amply documented (Brown, 1953; Weimann, 1968). Some psychophysiological experiments have been carried out on normal subjects, indicating that hyperventilation often occurs when subjects are exposed to an activating situation, although changes in arterial PCO_2 appear to be modest (Garssen, 1980; Suess, Alexander, Smith, Sweeney, & Marion, 1980).

A theoretical refinement was the formulation of a circular model for the development of HVS (see Figure 1), which gives a prominent role to the patients' interpretation of the symptoms they experience (Clark, 1986). In this model physiological activation, particularly hyperventilation, leads to somatic symptoms. The patient will anxiously anticipate new attacks if these symptoms are interpreted as elusive and uncontrollable, attributed to a serious disease, or seen as evidence of inability to handle the situation. This anxious anticipation will involve physiological activation, thereby increasing the risk of new attacks.

The relation between HVS and anxiety disorders is a related topic. It has been suggested that at least some of the somatic symptoms of panic attacks are caused by hyperventilation. Though similar suggestions with respect to anxiety neurosis have been forwarded since the first publication about HVS (Kerr et al., 1937), only recently have studies been conducted. One line of evidence includes the large diagnostic overlap between HVS and panic disorder with agoraphobia (Garssen et al., 1983; de Ruiter, Garssen, Rijken, & Kraaimaat, 1989). This finding signifies that most agoraphobic patients recognize the somatic symptoms induced by voluntary hyperventilation. Subjective panic is rarely induced by this provocation test, which has been considered an argument against the hyperventilation model (Gorman et al., 1984, 1988; Griez, Zandbergen, Lousberg, & van den Hout, 1988). However, according to the circular model, anxiety or panic may or may not occur during hyperventilation, depending on how symptoms are interpreted. During the provocation test, the patients induce symptoms themselves by performing a respiratory maneuver. Having actual control over symptoms will generally inhibit anxiety or panic, as was experimentally demonstrated in a recent study using CO_2 inhalation to provoke panic (Sanderson, Rapee, & Barlow, 1989).

TREATMENT OF HYPERVENTILATION SYNDROME

The overall goal of treatment strategies for alleviating symptoms of HVS is to increase alveolar and arterial PCO_2 to normal levels. In line with the circular model for the development of HVS (see Figure 1), the positive feedback loop can be broken via two distinct treatment components: (1) reduction of respiratory rate, and (2) cognitive reattribution of physical symptoms to hyperventilation instead of other more catastrophic causes. Several intervention methods focusing on these components have been developed: general relaxation training, training in slow and/or abdominal breathing, biofeedback training, auditory regulation, voluntary hyperventilation, and several types of cognitive therapy. Most of these methods have been investigated in outcome studies. We will first review studies that investigated the efficacy of pure breathing techniques, (i.e., techniques utilizing only the first treatment component mentioned above). Subsequently we will describe research that also included cognitive interventions.

Lum (1983) reported the effectiveness of breathing retraining offered by physical therapists. In a group of more than 1000 HVS patients, 80% became symptom-free. The value of this study is limited, however, because no details are given on the exact nature of the treatment or the instruments used for evaluating therapeutic outcome.

TABLE 1. Overview of Treatment Evaluation Studies

| Study	Treatment group (N)	Control group (N)	Outcome measures	Findings
Lum (1983)	N = 1350		Clinical judgement	80% symptom-free
Grossman et al. (1985)	AR 7 sessions N = 25	BI 7 sessions N = 22	Self-report questionnaires (e.g., STAI, EPI[1]), respiratory measures	AR > BI[2], on psychological and physiological measures at 1 month post-treatment
Van Doorn et al. (1982)	BF 7 sessions N = 10	BI 4 sessions N = 10	Self-report HVS symptoms, Pet CO_2[3]	BF > BI, on HVS symptoms at post-treatment. BF = BI, on HVS symptoms, at 3-month follow-up. BF = BI, on $PetCO_2$ at post-treatment and follow-up.
Vlaander-van der Giessen & Lindeboom (1982)	1) R + C; N = 15, 2) R + C; N = 15	No treatment N = 15	Self-report anxiety, physical symptoms, panic attacks	Treatment > control on all measures at post-treatment. 1 = 2 on all measures, except for panic intensity (2 > 1)
Vlaander-van der Giessen (1986)	1) R + C; N = 23, 2) AR + C; N = 21, 3) B + C; N = 22, 4) R + AR + C; N = 27, 5) R + B + C; N = 26, 6) AR + B + C; N = 21, 7) R + AR + B + C; N = 27, all 12 sessions	No treatment N = 25	Self-report anxiety, depression, physical symptoms	Treatment > control on all measures at post-treatment and at 1-year follow-up. No differences between 7 therapy methods
Compernolle et al. (1979)	Voluntary hyperventiliation 2 or more sessions N = 106			Majority of patients were effectively treated; phobic behaviors disappeared. However, two thirds required additional family therapy.
Kraft & Hoogduin (1984)	1) Voluntary hyperventiliation; N = 6, 2) Relaxation-breathing retraining; N = 6, 3) Stress management; N = 7, all 6 sessions		Self-report HVS symptoms, HV attacks, depression	1 = 2 = 3, on psychological measures, at post-treatment and 1-month follow-up

Study	Treatment		Measures	Results
Rapee (1985)	B 3 sessions $N = 1$		Self-report panic attacks, anxiety, physical symptoms	Reduction in panic attacks, symptoms, and anxiety
Clark et al. (1985)	B + CT 2 sessions $N = 18$		Self-report panic attacks, anxiety, depression	Significant reduction in panic attacks, anxiety, and depression, at post-treatment, 6-month and 2-year follow-up
Salkovskis et al. (1986)	B + CT 4 sessions $N = 9$		Self-report panic attacks, anxiety, depression, PCO_2	Significant reduction in panic attacks, and anxiety. Significant increase in PCO_2 at post-treatment.
Bonn et al. (1984)	B + E 9 sessions $N = 7$	E 9 sessions $N = 5$	Self-report panic attacks, agoraphobia, physical symptoms, resting respiratory rate	B + E = E at post-treatment, B + E > E, at 6-month follow-up
De Ruiter et al. (1989)	B + C 8 sessions $N = 13$	1) E 8 sessions; $N = 13$ 2) B + C + E 8 sessions $N = 14$	Self-report panic attacks, agoraphobia PCO_2 respiratory rate	B + C = 1 = 2, on psychological and respiratory measures, at post-treatment
Hibbert & Chan (1989)	B + E 5 sessions $N = 21$	P + E 5 sessions $N = 19$	Self-report panic attacks, physical symptoms, anxiety, depression, observer-rated anxiety	B + E > P + E on observer-rated anxiety B + E = P + E, on self-report measures, at post-treatment

Note. AR = auditory regulation; BI = breathing instruction; BF = biofeedback; R = relaxation; C = cognitive restructuring; B = breathing retraining; CT = cognitive therapy; E = exposure; P = placebo treatment.
[1]STAI = State-Trait Anxiety Inventory; EPI = Eysenck Personality Inventory.
[2]AR > BI = Auditory regulation scored significantly better than breathing instructions.
[3]Pet CO_2 = End-tidal CO_2 pressure.
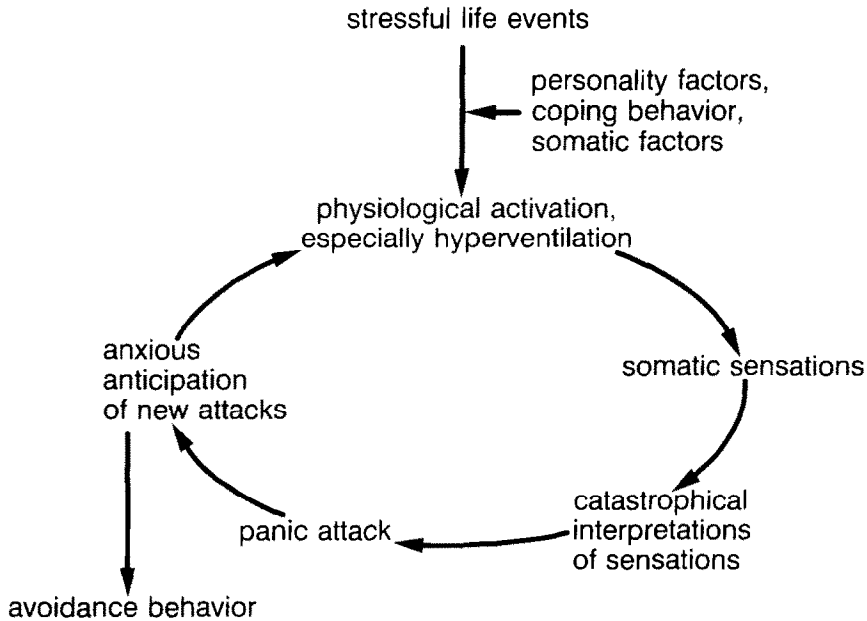
FIGURE 1. The Circular Model for the Development of the Hyperventiliation Syndrome.

Grossman, de Swart, and Defares (1985) used a combination of ambulatory auditory regulation and feedback with 25 HVS patients. The regulation instrument produced rhythmic sound signals which were timed at a slightly lower frequency than the patient's resting breathing frequency. Patients breathed along with the lower tempo three times a day for 10 minutes. The instrument could also serve as a biofeedback apparatus that produced the rhythm when breathing frequency exceeded a certain level. The biofeedback system was used for 1 hour each day. Results from this group were compared with results from a control group that received simple breathing instructions ("try to relax and breathe quietly"), to be practiced for 10 minutes three times a day. At post-treatment, the treatment group did significantly better on physiological and psychological outcome variables (PCO_2 at rest, state and trait anxiety, number of physical complaints).

Van Doorn, Folgering, and Colla (1982) gave 10 HVS patients biofeedback training (i.e., they received a maximum of seven weekly sessions of end-tidal PCO_2 feedback). A control group received simple breathing instructions to practice at home and four sessions with a therapist to discuss home practice. Both methods led to about equal increases in PCO_2 at post-treatment and at 3-month follow-up. The number of physical complaints was significantly lower in the biofeedback group at post-treatment, but this difference had disappeared at follow-up.

The studies of Grossman et al. (1985) and Van Doorn et al. (1982) point to the efficacy of breathing techniques aimed at increasing PCO_2 level for HVS patients. However, the majority of patients in both studies were not symptom-free at follow-up, causing some to seek additional treatment.

Vlaander-van der Giessen and Lindeboom (1982) evaluated the efficacy of breathing retraining and relaxation training by comparing both treatments with a control group receiving no treatment. Both treatments contained cognitive restructuring: that is, reattribution of physical symptoms to hyperventilation and explanation of the relationship

between stress and hyperventilation. Both treatments were more effective than no treatment. The only difference between treatment groups was the intensity of hyperventilation attacks (breathing retraining was more effective).

In her subsequent study, Vlaander-van der Giessen (1986) compared seven types of treatment for HVS. All treatments included cognitive restructuring and one or more types of treatments directed at changing breathing patterns: relaxation training, breathing retraining, and auditory feedback. Breathing retraining aimed at changing respiratory patterns to low-abdominal. Auditory feedback focused on reducing respiratory rate. If breathing was too fast, the patient heard the slow rate, which went off when the patient's breathing returned to the desired frequency. All seven therapies resulted in significant decreases in patients' pathology, compared to a wait-list control group. There were no significant differences among the therapies.

Compernolle, Hoogduin, and Joele (1979) applied repeated hyperventilation provocation as a therapeutic technique with 106 HVS patients. This technique can be considered a combination of exposure and a cognitive strategy; it focuses on reattribution of symptoms and lack of control over symptoms, which characterizes many HVS patients. Patients were instructed to induce a daily attack at home by voluntary hyperventilation. Subsequently, they had to rebreathe from a paper bag to let symptoms subside. The authors report that in 90% of their patients, attacks disappeared with just two sessions, followed by two or three maintenance sessions. The remaining 10% of the patients could handle their attacks without medical intervention. These findings seem impressive but are difficult to interpret because the authors provide no information on treatment compliance and the actual length of treatment, nor on the methods they employed for evaluating treatment outcome.

Finally, Kraft and Hoogduin (1984) investigated the effectiveness of four treatments for HVS patients: relaxation and breathing retraining, stress management, hyperventilation provocation, and benzodiazepines (lorazepam, chlordiazepoxide, flurazepam, and/or clopenthixal). The medication group was excluded from the analyses because of a large dropout rate. The three remaining treatments were equally effective in reducing hyperventilation attacks, physical symptoms, and depression. Unfortunately, the number of patients in each group was rather small ($N = 7$), which calls for cautious interpretation of these results. It should be noted that the rate of improvement in this small but controlled study was not as impressive as in the large uncontrolled study of Compernolle et al. (1979).

Treatment of Related Disorders

A number of studies have tested the efficacy of breathing retraining and reattribution of physical symptoms to hyperventilation in patients suffering panic disorder with or without agoraphobia (Bonn, Readhead, & Timmons, 1984; Clark, Salkovskis, & Chalkley, 1985; Hibbert & Chan, 1989; Rapee, 1985; de Ruiter, Rijken, Garssen, & Kraaimaat, 1989; Salkovskis, Jones, & Clark, 1986).

Rapee's (1985) paper presents a case study of a panic disorder patient treated with three sessions of breathing retraining, which resulted in a dramatic decrease in panic frequency from 22 to 2 attacks per 3 weeks at post-treatment.

Clark and colleagues (Clark et al., 1985; Salkovskis, Jones, & Clark, 1986) tested the effectiveness of respiratory control plus cognitive therapy techniques (e.g., Socratic questioning, altering catastrophic thinking) with panic patients in two uncontrolled studies. Their treatment significantly reduced panic attack frequency and significantly increased PCO_2. In fact, PCO_2 values at the end of treatment were similar to those found in nonpatient samples.

In the study by Bonn and colleagues (1984), 12 agoraphobic patients received either respiratory control plus exposure in vivo or exposure in vivo alone. Results showed no difference between the two therapy conditions at post-treatment. However, at the 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.

De Ruiter, Rijken, Garssen, and Kraaimaat (1989) compared the efficacy of three different treatment packages for panic disorder with agoraphobia: (1) breathing retraining plus cognitive restructuring, (2) exposure in vivo, and (3) a combination of these two. Breathing retraining and cognitive restructuring was highly similar to the respiratory control treatment of the Clark et al. (1985) and Salkovskis, Jones, and Clark (1986) studies. Quite unexpectedly, breathing retraining plus cognitive restructuring did not significantly reduce panic frequency. This is especially remarkable since treatment consisted of eight sessions, while the treatments in the earlier studies by Clark and Salkovskis comprised two and four sessions, respectively. De Ruiter, Rijken, Garssen, and Kraaimaat (1989) reviewed several explanations for their anomalous finding, but none of the alternative interpretations (e.g., different samples and settings) were deemed valid. Combination therapy was no more effective on any of the outcome variables at post-test than exposure alone, similar to the earlier finding of Bonn et al. (1984).

Hibbert and Chan (1989) compared the effectiveness of five sessions of pure breathing retraining with a placebo treatment in panic patients with or without agoraphobia. The placebo treatment consisted of discussion of the role of stress and personality characteristics in eliciting anxiety disorders, without use of any anxiety management techniques. Contrary to most previous studies, but somewhat in line with the study by De Ruiter, Rijken, Garssen, and Kraaimaat (1989), respiratory control did not differ significantly from placebo treatment on any of the self-report measures. The only difference that emerged was with regard to observer ratings of anxiety, which showed more improvement in the respiratory control group. Most importantly, patients who recognized symptoms during voluntary hyperventilation prior to therapy did not benefit more from respiratory treatment than patients who had not recognized symptoms induced by such provocation.

COMMENT

It should be emphasized that some of the studies contained very small samples (Bonn et al., 1984; Kraft & Hoogduin, 1984) or lacked control groups (Clark et al., 1985; Compernolle et al., 1979; Lum, 1983; Salkovskis, Jones, & Clark, 1986). Furthermore, some studies incorporated the two treatment components mentioned at the start of this review (breathing retraining and cognitive reattribution) into a single treatment, so that determining the individual effect of either component is impossible (e.g., Clark et al., 1985; de Ruiter, Rijken, Garssen, & Kraaimaat, 1989; Salkovskis, Jones, & Clark, 1986).

With these reservations, we think that the majority of the studies point to a therapeutic effect of breathing retraining and cognitive reattribution of physical symptoms to hyperventilation for patients suffering HVS and the closely related panic disorder with or without agoraphobia. This appears to support the model of symptom causation represented in Figure 1. However, the *specificity* of these techniques for HVS is questionable. Vlaander-van der Giessen (1986) found relaxation training just as effective as breathing retraining; and Hibbert and Chan (1989) found breathing retraining equally effective as a placebo treatment. Moreover, in the latter study, breathing retraining was not more

effective with patients who had recognized symptoms at a hyperventilation provocation test than with those who had not.

The conclusion seems warranted that both breathing retraining and cognitive reattribution help alleviate anxiety in patients with HVS or related disorders. The exact mechanism of their working remains elusive.

THE VALIDITY OF THE HVS MODEL

The model of symptom causation applied in the therapy studies was derived from a rather narrow basis: the similarity of symptoms produced by voluntary overbreathing and those occurring spontaneously during anxiety attacks. Only recently have efforts been made to test this model more stringently. First, studies explored whether overbreathing is the underlying mechanism of panic attacks (as defined in the DSM-III-R). Second, studies attempted to clarify whether the hyperventilation provocation test is a valid predictor of spontaneously occurring symptoms.

A direct proof of the validity of the HVS model would be the co-occurrence of spontaneous hyperventilation episodes and somatic symptoms. This is a serious technical problem, since patients are usually not available for measurement during spontaneous attacks. Baseline measurements are of limited value because normal breathing patterns may occur between hyperventilation episodes. Only chronic hyperventilation is relatively easy to determine. Gardner, Meah, and Bass (1986) found continuous hyperventilation in some patients during their 60 minutes protocol, using capnographic measurements. Chronic hyperventilation will also lead to a negative base excess in the blood, indicating long-term compensation for respiratory alkalosis. This has been demonstrated, but only in 14% of HVS patients (Folgering, 1986). On a few occasions, blood gas analyses have been performed during spontaneously occurring panic attacks in a hospital environment. These showed a decrease of PCO_2 during panic (Griez, Zandbergen, Lousberg, & van den Hout, 1988; Salkovskis, Warwick, Clark, & Wessels, 1986; Weimann, 1968). Recently, a method for long-term, ambulatory measurement of transcutaneous PCO_2 ($PtcCO_2$) has been developed (Pilsbury & Hibbert, 1987).[1] With this method, drops in $PtcCO_2$ were demonstrated during panic attacks in about half the patients with panic disorder (Hibbert & Pilsbury, 1989). Undoubtedly, spontaneous hyperventilation occurs in some patients. However, the simultaneous occurrence of hyperventilation and symptoms does not prove that a decrease of arterial PCO_2 *causes* the symptoms. Rather than being causal to panic attack, hyperventilation may be an accompanying symptom of panic. The studies of Gorman et al. (1988) and Hibbert and Pilsbury (1988, 1989) support this interpretation. The first study found that hyperventilation usually occurred during panic attacks provoked by lactate infusions, while voluntary hyperventilation by itself did not effectively provoke a clear panic attack. Hibbert and Pilsbury (1989) compared the symptoms of panic attacks with and without hyperventilation. If hyperventilation is an important symptom-producing mechanism, symptoms occurring during these two kinds of panic attacks should differ. However, such a difference was not found.

In summary, recent studies do not support the idea that hyperventilation is an important *causal* mechanism in producing panic attacks. Hyperventilation should rather be considered an accompanying phenomenon in some panic patients.

We will now turn to studies that attempt to validate the most widely used diagnostic test for HVS. This so-called "hyperventilation provocation test" (HVPT), with its criterion of

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[1]$PtcCO_2$ is closely related to arterial PCO_2, though changes in the blood are followed with some delay by changes in transcutaneous values (Pilsbury & Hibbert, 1987).

the patient's recognition of induced symptoms as being similar to symptoms experienced in spontaneous attacks, is still the most important element in the diagnosis of HVS (Lewis & Howell, 1986).

Studies to validate the recognition criterion of the HVPT have rarely been conducted. One type of control is to artificially prevent lowering of end-tidal CO_2 pressure ($PetCO_2$) during hyperventilation (isocapnic overventilation test; IOT). Weimann (1968) found that a considerable number of patients experienced and recognized symptoms during a HVPT, but not during this control test. Huey and West (1983) used the same control condition in a study of two groups of normal subjects, which were formed on the basis of the number of spontaneous symptoms. Using the number of symptoms induced during both tests as the dependent variable, they found an interaction between groups (low and high responders) and conditions (HVPT and IOT). Subjects with many spontaneous symptoms differed more from subjects with few symptoms during normal hyperventilation than during isocapnic overventilation. These two studies seem to validate the HVS model.

In a study by Griez et al. (1988), care was taken to avoid raising expectations of anxiety. The word hyperventilation was omitted when instructing patients and normals to overbreathe. Both patients and normals reported more somatic sensations during hyperventilation than during isocapnic overventilation. In contrast to the findings of Huey and West, there was no difference between groups. What remains is that in these studies hyperventilation led to more symptoms than the control condition. However, using another control condition, a similarity was found. Roll (1987) applied a stressful mental task as the control method in a study of patients with functional heart complaints. The same method was used in a replication study of patients suspected of having HVS (Hornsveld, Garssen, Fiedeldij Dop, & van Spiegel, 1990). The stressful mental load task consisted of performing the Stroop Color Word Task under time pressure. Capnographic measurements indicated no or minor changes in end-tidal PCO_2 during this task. During the HVPT, 44% of patients in the first study and 61% of patients in the second study recognized symptoms that are considered typical for hyperventilation. These patients would be labeled HVS patients on the basis of the recognition criterion. Despite nearly normal $PetCO_2$ levels, patients also experienced many symptoms during the stressful mental load task and recognized them as similar to symptoms in daily life. Symptoms were recognized during this control condition by 38% of the patients in the first study and 52% in the second study. These two studies invalidate the HVPT as a diagnostic tool by showing that the symptoms considered typical for hyperventilation can also be produced by a stressful task without decrease of the $PetCO_2$.

Using the method of ambulatory monitoring of $PtcCO_2$, Hibbert and Pilsbury (1989) also invalidated the recognition criterion. In their study, 10 patients recognized symptoms during the hyperventilation provocation test, but in 5 of these 10 patients $PtcCO_2$ did not decrease during panic. Five patients failed to recognize symptoms induced by voluntary hyperventilation, but two demonstrated considerable drops in $PtcCO_2$ during panic. Apparently, the response to the HVPT does not predict the occurrence of hyperventilation during panic attacks.

DISCUSSION

In this review, HVS is deliberately described as a scientific model, not as a term describing observable phenomena. The model implies that an important part of the patient's symptoms are caused by hyperventilation. 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, but not as a sufficient factor itself for developing panic. The principle of a vicious circle implied in this model gives an elegant explanation of the production of panic from the symptoms of hyperventilation. This model appears to be credible to therapists and patients, and has led to the development of therapeutic interventions aimed at breathing retraining, usually combined with cognitive restructuring. These therapies appear to be effective for patients diagnosed as having HVS, as well as for patients diagnosed as having panic disorder or agoraphobia.

Recent evidence, however, causes one to doubt the importance of hyperventilation as a necessary factor for panic attacks. No difference in symptoms was found between panic attacks with or without hyperventilation. Furthermore, the commonly used diagnostic test for HVS, the HVPT, can no longer be considered valid because symptoms assumed to be typical for HVS can be produced by a stressful mental task without the associated decrease of the alveolar CO_2 level. In the near future, HVS will probably join its predecessors (such as effort syndrome, irritable heart, or neurocirculatory asthenia) as an obsolete diagnostic label and a premature etiological explanation of the symptoms of panic attacks. Moreover, therapies derived from the hyperventilation model do not appear to be more effective than alternative treatments not based on the model. Furthermore, breathing retraining has been shown to work with anxious patients who show no signs of hyperventilating. Taken together, the therapy outcome studies suggest that methods of breathing retraining and attribution of symptoms to hyperventilation, although to some degree effective, are not specific therapies and do not work according to the principles upon which they are based.

In the history of medicine, successful cures that appeared rational but did not work according to the principles that were assumed to underlie them have by no means been exceptional. Wulff (1976) coined the term "rational placebo" for treatments that appear logical, but on closer inspection do not work as supposed. In psychotherapy, systematic desensitization is the best-known example of a procedure that is effective, but does not work according to the principles from which it was developed, as has been shown by an extensive literature (Emmelkamp, 1982; van Dyck, 1986).

How do breathing retraining and cognitive reattribution help anxious patients, if not through correction of a disturbed breathing pattern? As far as the cognitive element is concerned, authors such as Frank (1973) claim that any credible explanation may have a therapeutic effect, whether or not the rationale is correct. The HVS model seems to serve this function well, as it succeeds in reducing a wide range of disturbing symptoms to a rather simple and innocuous process. Furthermore, the element of breathing retraining will induce relaxation, which is an established method for reducing anxiety. Another effective element may be that patients are given a structured task in situations where panic may occur. This may distract them from feelings of anxiety and promote self-reliance. Such an effect has appeared in the literature under the labels of self-efficacy (Bandura, 1977) and sense of mastery (Frank, 1973). A recent study offers an experimental demonstration of the mechanism of mitigating anxiety in panic patients. Panic patients given the illusion that administration of an anxiety-provoking mixture of 5% CO_2 was under their control, reported less symptoms, less panic, and less recognition of anxious symptoms than patients who believed that they could not control the administration of the gas mixture (Sanderson, Rapee, & Barlow, 1989). This points to the conclusion that a sense of control, even if based on illusion, can be effective in reducing anxiety in panic patients. Any of the therapeutic interventions discussed here can generate such a sense of control. Because of these therapeutic qualities, breathing retraining and cognitive reattribution may in the future still be fruitfully applied to therapists as rational placebos, while more valid theories about the origins of anxiety attacks are being developed.

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