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A CONTROLLED STUDY OF A BREATHING THERAPY FOR TREATMENT OF HYPERVENTILATION SYNDROME

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Abstract—A therapy directed toward slowing and regularizing the ventilatory pattern was compared with a partial-treatment, comparison procedure for individuals with somatic and psychological symptoms attributable to hyperventilation episodes (i.e. hyperventilation syndrome). Comparing repeated measures between a pretreatment baseline session and a post-treatment followup, we found that the experimental therapy, in contrast to the comparison procedure, produced a greater number of, and more extensive, improvements in psychological, symptom complaint and ventilatory dimensions. Results also suggest changes in central respiratory control mechanisms as a consequence of treatment.

HYPERVENTILATION syndrome (HVS) is a clinical disorder, with no known organic basis, which induces a host of variable somatic and psychological symptoms as an apparent consequence of episodes of hyperventilation (defined by reduced arterial and alveolar levels of carbon dioxide). A detailed description of the syndrome has been recently published elsewhere in this journal [1]. Diagnosis of the disorder is often made on the basis of recognition of presenting complaints during a brief period of voluntary hyperventilation and delayed recovery of CO_2 levels subsequent to instructions to terminate overbreathing. This diagnostic procedure is known as the provocation test (PT).

Although incidence estimates suggest that HVS is a frequently occurring problem in the general population [2–5], there have been few controlled investigations of potential therapies for the syndrome. Furthermore, the treatment studies which have been published have generally yielded equivocal findings. For example, two studies [6, 7] investigating the effects of a beta-adrenergic pharmacological blocker on HVS found no more improvement in CO_2 level or reduction of complaints with this treatment than with either a placebo treatment, other forms of therapy or no therapy whatsoever. Another investigation, comparing two behavioral therapies aimed at training patients to increase end-expiratory alveolar levels of CO_2, reported some benefit of treatment with regard to symptom reduction [8]. However, it is difficult to draw firm conclusions from this study, since patients came from a psychiatric clinic, groups were small, there were few criteria used to assess success of treatment, and diagnostic criteria employed to select HVS subjects were unclear. Several other publications [e.g. 9, 10] also claim significant benefits of ventilatory retraining for HVS but provide insufficient quantitative data to evaluate treatment efficacy.

The present study is a quantitative attempt to investigate the potential therapeutic effects of a ventilatory-training treatment among a group of stringently diagnosed HVS patients referred to us from general medical practices. We have utilized multiple ventilatory, symptomatic and psychological measures to evaluate effects of therapy and have also employed a partial-treatment group as a control procedure

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with which to compare our experimental treatment. The experimental therapy was aimed solely at modifying the sedentary breathing pattern with the aid of a ventilatory-feedback and training device. Subjects in this group were taught gradually to regularize and lengthen the time components of their breathing patterns, as well as to make inspiration more abdominally expensive.

MATERIALS AND METHODS

Subjects

Prospective subjects were referred to us by general medical practitioners on the basis of a suspected diagnosis of HVS. A preliminary physical examination excluded all patients with any serious physical ailment. For inclusion in the study, all subjects were required to meet unequivocally two major diagnostic criteria of HVS: (1) recognition of frequently occurring, chronic complaints during the provocation test, and (2) delayed recovery of end-expiratory (end-tidal) CO_2 concentration ($C_{et}CO_2$) at 5 min post-hyperventilation. Details of this procedure are later presented in the *Measurements* section.

Fifty-six subjects were initially included in the investigation and were assigned either to the experimental or the comparison treatment. Forty-seven subjects completed the entire study, with 25 in the experimental therapy group (10 males, 15 females) and 22 in the partial-treatment, comparison group (8 males, 14 females). The average age of the experimental group was 29.3 ± 9.7 (SD) yr and of the comparison group, 35.8 ± 11.4 yr ($p < 0.05$). At intake, half of the subjects in each group reported regularly taking medication (primarily benzodiazepines).

Experimental design

Figure 1 outlines the study design (for details, see later). Subjects were randomly assigned to one of the treatments. Half of these patients were assigned to each group. All subjects were administered the same diagnostic and assessment procedure at a pretreatment initial intake session and at a followup session four weeks after termination of treatment. All treatment and measurement sessions were carried out with single subjects and administered by the same experimenter. Data were scored and analyzed without awareness of the group membership of individual subjects.

Therapy for both groups occurred over a ten-week period. All patients were told they would receive an experimental treatment which was believed likely to be effective in alleviating symptoms related to HVS.

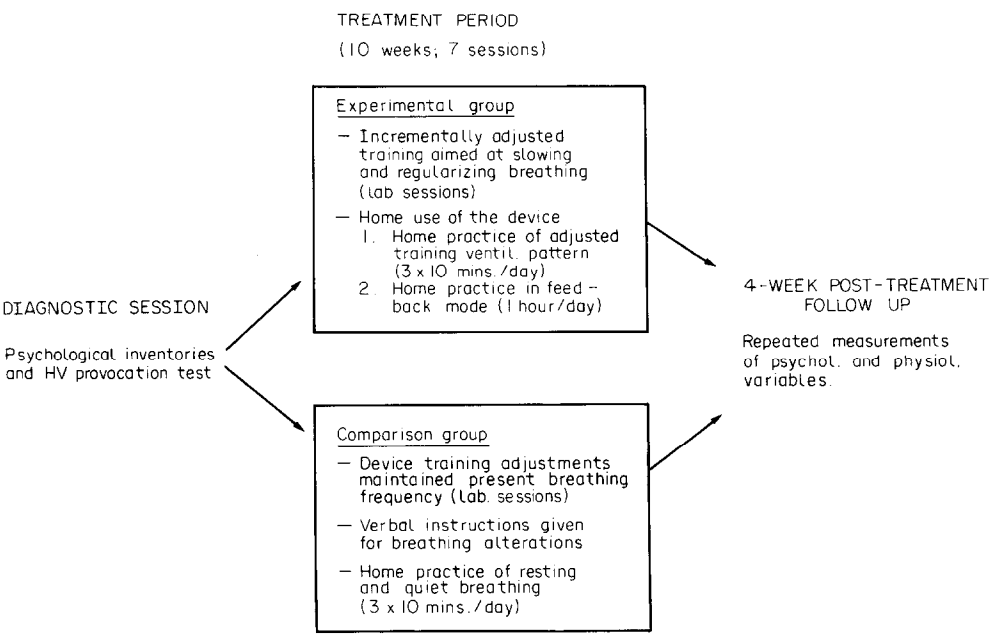

FIG. 1.—Outline of the experimental design of the present study.

Measurements and training: instruments

Physiological. Respiration was monitored by means of a CO_2 infrared gas analyzer (Beckman LB2) attached to a chart-paper recorder. This apparatus allows measurement of $C_{et}CO_2$ (percentage) and respiration rate (RR). Depending upon period of measurement, end-tidal gases were collected either via a light, comfortable face mask enclosing mouth and nose perimeters but with central opening to room air, or via a more intrusive, tight-fitting mouthpiece and noseclip, the end of mouthpiece also opening to room air. (See *Diagnostic* section for further details.)

For both treatment groups, a portable ventilatory training device, developed by Defares [11], was employed. This device measures $15 \times 7 \times 4$ cm and is powered by an internal 9V battery. The apparatus can be set to one of two different modes of operation: (1) The instrument generates auditory stimuli, via an earphone, which simulate a desired pattern of breathing (i.e. RR, inspiration time, expiration time and pause times between phases) adjustable between six and 30 respiratory cycles per minute. Inspiration is signalled by a continuous rising tone, expiration by a descending tone, and pauses by periods of silence. Hence in this mode, individuals are able to pace the time components of their own ventilatory pattern to the emitted tone pattern of the device. (2) The second mode of operation provides feedback information to patients concerning their ongoing, spontaneous ventilatory pattern and enables them to correct breathing pattern when it does not conform to preset criteria of time components. This is achieved by means of the apparatus monitoring RR via a mercury-in-silastic strain gauge transducer. When the adjustable, criterion RR level is continuously exceeded for longer than 30 sec, the above-described auditory tone pattern is generated, and the patient is required to pace his own ventilation accordingly. Once he successfully reduces RR below criterion level for a full minute, the auditory stimuli are automatically terminated, and no further feedback is given until the next violation.

Psychological and HVS complaint inventories. A 35-item HVS complaint checklist was used by subjects to rate frequency of occurrence of common HVS symptoms on a four-point scale (0 = never; 3 = often). Developed by a Dutch interuniversity working group on HVS, this questionnaire has recently been validated on a large group of patients [1]. It assesses self-reports of symptoms covering a wide range of systems (e.g. CNS, peripheral nervous, cardiovascular, respiratory, gastrointestinal and psychological).

Psychological inventories were all objective, paper-and-pencil questionnaires which have been validated on large populations in the Netherlands. Neuroticism and neurosomatic instability scales were taken from the Dutch modification of Eysenck's Maudley Personality Inventory [12]. Neurosomatic instability is defined as neurotic tendencies manifested by the presence of functional somatic complaints. State and trait anxiety measures were derived from translations of the Spielberger scales [13]. The self-esteem scale was from the widely used Netherlands Personality Inventory [14].

In order to evaluate effects of treatment, psychological tests and the HVS complaint checklist were administered twice during the study, at the initial intake session and at four weeks after the last treatment.

Diagnostic and post-treatment evaluation

The identical sequence of procedures was used for the initial diagnostic session and the evaluation followup occurring four weeks after treatment. This includes the following: (1) Completion of psychological inventories and HVS complaint checklist; (2) a ten-minute baseline, resting measurement of ventilatory activity using light facemask; (3) instructions for the PT, removal of facemask and attachment of mouthpiece and noseclip; (4) administration of the voluntary hyperventilation phase (approximately four minutes); (5) a 5 min recovery period following request to terminate hyperventilation.

Preceding attachment of mouthpiece and noseclip, subjects were solely informed that within a few minutes they would be asked to start breathing quickly and as deeply as possible. During the hyperventilation phase of the PT, patients were required to maintain $C_{et}CO_2$ below 2.5% for a 3 min period. Immediately after the hyperventilation period, subjects were told to resume their normal manner of breathing (five-minute recovery period). Subsequently they were asked to report any physical or psychological complaints experienced during the hyperventilation phase of the PT.

Ventilatory measures derived from this evaluation included the following: *baseline, resting RR* and $C_{et}CO_2$ based on the means of the last two minutes of the baseline period; CO_2 *recovery* in the first 30 sec following the 5 min post-hyperventilation period, expressed as percentage of mean prehyperventilation CO_2 level. These variables were used to assess effects of treatment.

Treatment procedures

Treatments *for both experimental (E) and comparison (C) groups* consisted of a total of seven half-hour laboratory sessions plus home assignments over a ten-week period. Laboratory sessions occurred at weeks 1, 2, 3, 5, 7, 9 and 11. During the beginning of the first treatment appointment, each subject was given an accurate account of the probable factors contributing to HVS and the likely consequences of hyperventilation for psychophysiological functioning. The first portion of all later sessions was reserved for discussion of home assignment difficulties and for providing general encouragement. During every treatment session, patients were subsequently attached to the CO_2 gas analyzer (via facemask), and their

resting RR was monitored for seven minutes. Immediately thereafter, the ventilatory training device was individually adjusted, placed in the pacing mode (mode 1 described in *measurement* section) and connected to the subject via earphones. Subjects were then requested to practice synchronizing their breathing patterns with the emitted tone pattern generated by the device, and verbal emphasis was placed upon the importance of expansion of the abdomen during inspiration. This pacing condition lasted 10 min. Sessions were concluded by repeating the importance of home practice of assigned exercises. No psychological counseling was offered to either group.

There were two major differences in procedure between treatment groups: (1) At each laboratory session, the training device for C patients was adjusted to produce a tone pattern with an identical RR to that which had just been previously measured during the preceding rest period of the same session. E subjects, on the other hand, always had the device tone pattern reset at each session so that it was slightly slower than their resting RR for that session. Hence, C subjects were maintained, via the device, at their normal resting RR for any session, whereas E subjects were trained progressively to slow their respiration rate. It should be noted that the actual adjustment of the device for E subjects was variable from subject to subject and across treatment sessions, typically one to three cycles per minute slower than their resting RR for a specific session.

(2) C group patients were given home assignments to relax three times a day (10 min each time) and to practice breathing slowly and abdominally during these periods. Hence, home assignments for comparison group subjects were rather globally instructed. Experimental group subjects, in contrast, were given an adjusted ventilatory training device to take home after each laboratory treatment session. In addition to instructions concerning abdominal breathing, E subjects were asked to train with the device at home three times daily (10 min each time) in the pacing mode (mode 1). They also were told to utilize the device separately in the feedback mode (mode 2, via strain gauge attached around the chest) for an additional period of one hour per day. The feedback mode was to be employed during the performance of some sedentary, but potentially stressful, daily activity. The criterion RR level of the feedback condition was successively lowered across weeks; in other words, a slower RR would be required across weeks in order to avoid triggering auditory feedback.

In sum, both treatment groups received a form of ventilatory therapy. However, C group subjects received primarily verbal, home assignment instructions to slow ventilation and to make inspiration more abdominally expansive. Utilization of the training device was restricted to the laboratory, the pacing mode was never adjusted below the present resting RR for a particular session, and ventilatory feedback (mode 2) was not employed. In contrast, E treatment subjects were trained, by means of both pacing and feedback modes of the device, to successively reduce RR both within and across sessions. In addition to having been given the same verbal instructions as the C group, E subjects were told to practice with the training apparatus daily at home.

Statistical analysis

One-way analyses of variance (ANOVA's) were initially performed to assess any baseline differences between treatment groups with regard to the dependent variables. Since none of these analyses approached significance, we assessed treatment effects between groups with one-way ANOVA's of ventilatory, psychological and symptom change scores, calculated as the difference between the initial, diagnostic intake values and those obtained at the four-week post-treatment, followup session. Within-group changes were evaluated using two-tailed *t*-tests. With respect to all variables, positive change scores indicate improvement and not necessarily the actual direction of change. Specifically, improvement for us implies the following changes from intake to followup measurements: reductions in resting RR, the HVS complaint checklist scores and all the psychological measures except self esteem and increases in self esteem, resting $C_{et}CO_2$ and PT CO_2 recovery.

RESULTS

Diagnostic intake values of dependent measures

Table I presents group means for all dependent measures at the initial diagnostic and evaluation intake sessions. Note that scores for each measure were similar between groups; none approached significance ($p > 0.10$). Regarding ventilatory parameters, both resting $C_{et}CO_2$ and CO_2 recovery means were well below normal values (5.0% and 90%, respectively; see 16 and 20). In fact, only a total of six subjects were within the normal range of resting CO_2 levels (5.0–6.3%), and all these patients were at the extreme low end of the range.

Examination of the psychological variable scores (Fig. 2) also revealed that our

TABLE I.—MEAN VALUES (± S.D.) OF PHYSIOLOGICAL AND PSYCHOLOGICAL MEASURES AT INITIAL DIAGNOSTIC SESSION

Variable	Experimental group $\bar{X}$	S.D.	Comparison group $\bar{X}$	S.D.
Physiological				
Resting RR (cycles/min)	16.9	6.8	16.4	4.4
Resting $C_{et}CO_2$ (%)	4.2	0.5	4.2	0.5
CO_2 recovery (%)	71.4	12.0	71.6	14.2
Self-report (pts.)				
State anxiety	48.2	12.7	50.2	11.7
Trait anxiety	46.8	8.7	50.8	10.8
Neuroticism	78.3	23.9	83.1	22.5
Neurosomatic instability	30.6	5.3	31.8	5.6
Self-esteem	23.2	5.8	20.2	6.7
Symptom complaints	47.6	14.7	47.2	17.8

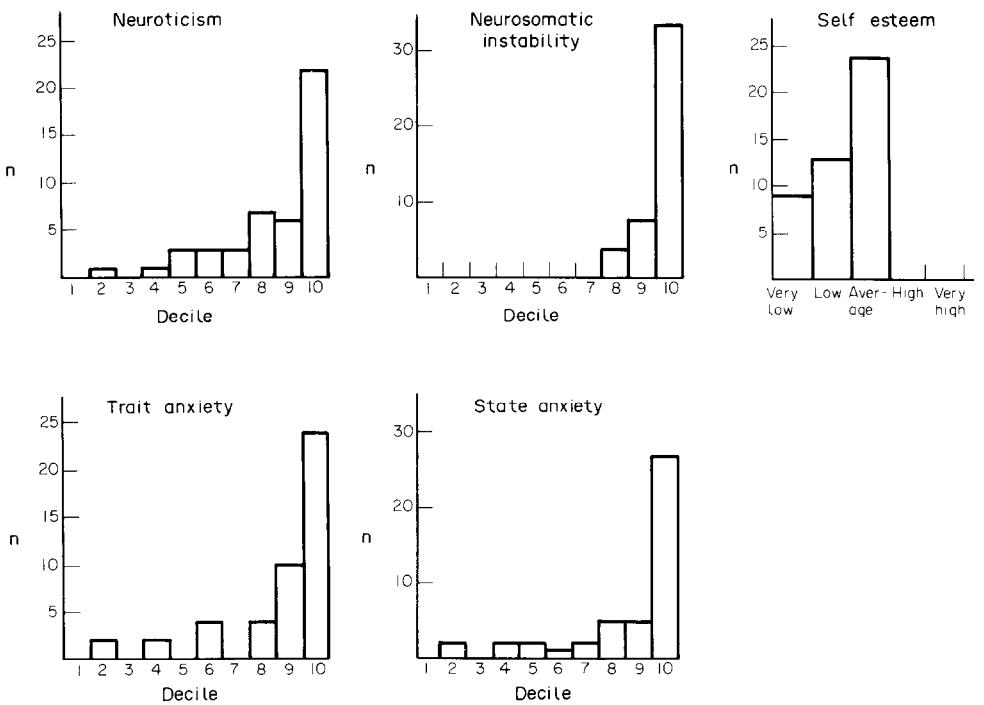

FIG. 2.—Distributions of all subjects among decile and category ranks of established population norms for normal adults (psychological variables). With regard to the self-esteem measure, very low, ≥ 5th percentile; low, 6th–19th percentiles; average, 20th–79th percentile; high, 80th–94th percentile; and very high, ≥ 95th percentile.

HVS patients deviated substantially from established population norms for the individual measures [12–14]. Both treatment groups are combined in Fig. 2. Subjects showed, in general, quite low scores on the self-esteem scale, and extremely elevated scores on measures of neuroticism, neurosomatic instability and state and trait anxiety. Hence most of our subjects, at diagnostic intake, could be characterized, on

the basis of the self-report inventories, as neurotic and anxious with relatively low self-esteem and marked tendency to express emotional problems by manifesting somatic complaints.

TABLE II.—VENTILATORY AND SELF-REPORT ALTERATIONS AS A FUNCTION OF TREATMENT MODALITY (CHANGE FROM BASELINE DIAGNOSTIC SESSION). POSITIVE SCORES INDICATE IMPROVEMENT AND NOT NECESSARILY DIRECTION OF CHANGE. EACH VALUE REPRESENTS THE MEAN ± S.E. NS, NOT SIGNIFICANT; *p*, PROBABILITY

Variable	Experimental therapy ($n=25$)	p*	Comparison treatment ($n=22$)	p*	F-ratio (between groups) (df = 1, 45)	
Physiological						
Resting RR (cycles/min)	+5.6 ± 0.9	0.001	+1.5 ± 1.1	NS	8.75	($p=0.005$)
Resting $C_{et}CO_2$ (%)	+0.5 ± 0.1	0.001	+0.4 ± 0.1	0.007	NS	
CO_2 Recovery (%)	+16.0 ± 2.9	0.001	+6.5 ± 2.2	0.008	6.41	($p=0.02$)
Self-Report (pts.)						
State anxiety	+8.8 ± 2.0	0.001	+2.9 ± 1.8	NS	4.52	($p=0.04$)
Trait anxiety	+6.1 ± 1.7	0.002	+1.3 ± 1.7	NS	3.91	($p=0.05$)
Neuroticism	+5.7 ± 3.0	0.08	−4.7 ± 3.0	NS	5.76	($p=0.02$)
Neurosomatic instability	+3.4 ± 0.9	0.001	+0.6 ± 1.0	NS	4.48	($p=0.04$)
Self-esteem	+1.7 ± 0.9	0.07	−0.3 ± 1.0	NS		
Symptom complaints	+13.6 ± 2.5	0.001	+1.9 ± 2.0	NS	13.29	($p=0.001$)

*Two-tailed *t*-tests for changes within groups.
†One-way ANOVA's of change scores for differences between groups.

Treatment effects

Ventilatory measures. ANOVA's of ventilatory change scores revealed differences between treatment groups for resting RR and CO_2 recovery (see Table II), E group subjects showing greater improvement for both variables. Paired *t*-tests for individual groups indicated that both groups improved with regard to $C_{et}CO_2$ at rest and PT CO_2 recovery, whereas only E group subjects additionally manifested a reduction in resting RR.

Symptom complaints and psychological inventories. ANOVA's of the self-report measures revealed a number of between-group differences, each of which was associated with a significant or near-significant improvement for only the experimental group. The E group showed improvement on the following scales: HVS complaints, trait anxiety, state anxiety and neurosomatic instability; neuroticism and self-esteem measures approached significance.

Individual effects of treatment. Since the above-mentioned findings may be of some clinical relevance, we examined the data with regard to the percentage of each group that exhibited a marked level of improvement. "Marked improvement" was arbitrarily defined as scores indicating improvement equal to or greater than two standard errors of the mean for a particular variable. Figure 3 indicates that, for each dependent variable, a higher proportion of substantial improvement was seen in the E group, discrepancies between treatment groups being largest among the self-report measures and CO_2 recovery. Hence, not only were the mean levels of improvement higher for subjects receiving the E therapy, but also there was a greater proportion of this group of subjects who showed substantial improvements in each measure.

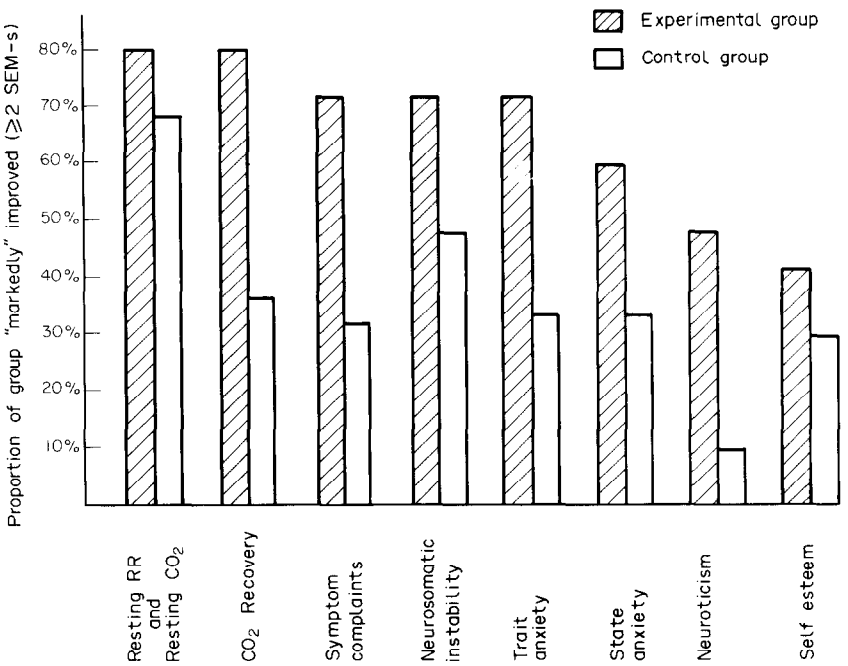

FIG. 3.—Percentage of each group displaying improvement greater than or equal to two standard errors of the baseline means for each of the dependent measures. Hatched bars, experimental group; white bars, comparison group.

Correlations between ventilatory and self-report change scores. Since both treatment groups demonstrated significant improvements in ventilatory parameters (albeit differentially), all subjects were combined into one group in order to evaluate correlations between ventilatory and self-report changes ($n = 47$). Table III indicates that alterations in the two CO_2 measures (resting CO_2 and PT CO_2 recovery),

TABLE III.—CORRELATIONS BETWEEN VENTILATORY AND SELF-REPORT CHANGES. POSITIVE COEFFICIENTS INDICATE CORRELATED IMPROVEMENTS BETWEEN VARIABLES. NS, NOT SIGNIFICANT

	Resting RR	Resting $C_{et}CO_2$	CO_2 Recovery
Symptom complaints	0.22 ($p = 0.06$)	0.35 ($p = 0.009$)	0.49 ($p = 0.001$)
State anxiety	0.20 ($p = 0.09$)	0.33 ($p = 0.01$)	0.38 ($p = 0.005$)
Trait anxiety	NS	0.25 ($p = 0.05$)	0.33 ($p = 0.01$)
Neuroticism	0.24 ($p = 0.06$)	NS	0.30 ($p = 0.02$)
Neurosomatic instability	NS	NS	0.27 ($p = 0.03$)
Self-esteem	NS	0.25 ($p = 0.05$)	NS

themselves not correlated with each other ($r = -0.09$), were positively associated with changes in each of the self-report variables. Resting respiration rate changes approached significance with three self-report measures. In cases where *within-group* ranges of variance were largely overlapping for correlated change scores, within-group correlations were similar to each other and to the combined-group correlation matrix.

DISCUSSION

The findings of this study indicate that a brief, seven-session therapy aimed at gradually slowing and regularizing respiratory phase durations, with an emphasis on abdominal breathing, was effective in ameliorating ventilatory, symptom complaint and psychological complexes associated with HVS. Both the experimental mode and the partial-treatment comparison procedure successfully achieved desired alterations in ventilatory characteristics, although effects of the E treatment were more extensive both with regard to the number of breathing parameters altered (three vs two) and the degree of improvement for any of these variables.

Results for the symptom complaint and psychological inventories were more pronounced: There were significant differences in amount of change between groups with regard to all self-report measures except self-esteem. Significant improvement or improvement approaching significance was found for each of these variables, including self-esteem, but only among the E group. Furthermore, the positive correlations between ventilatory changes, on the one hand, and complaint and psychological changes, on the other hand, indicate that long-term alterations in ventilatory parameters (the only direct goal of our therapy) were associated with both situational and dispositional changes in psychological characteristics and psychosomatic complaints.

There are, however, certain methodological issues that necessitate discussion and require us somewhat to temper our conclusions. First, since two of the three ventilatory measures used in this study (resting RR and resting CO_2) can, to a substantial extent, be temporarily altered by means of voluntary maneuvers, we cannot definitely rule out the possibility that E subjects merely voluntarily slowed down RR and raised CO_2 during laboratory sessions and their spontaneous ventilatory activity outside the laboratory remained unaffected by treatment. There are, nevertheless, several points that militate against this argument. In our experience, only certain limits of slowing RR and raising CO_2 can be tolerated for a period as long as 10 min. It seems unlikely that E subjects could maintain the altered levels of these variables which were exhibited during the post-treatment rest period without some underlying change in physiological ventilatory control mechanisms. Additionally, findings from our laboratory indicate that one-session voluntary slowing of RR was almost invariably accompanied by drops in end-tidal CO_2 (as a consequence of increased tidal volume), rather than increases [15]. Since CO_2 level and RR seem to be inversely related in studies of ventilatory control during spontaneous breathing [16], real alteration of ventilatory control is, once again, implied by our findings. Perhaps, the most persuasive evidence that long-term ventilatory control mechanisms have been altered derives from the large experimental-group changes with regard to the third respiratory parameter, post-hyperventilation CO_2 recovery. Several physiological investigations indicate that this measure reflects characteristics of central

ventilatory regulation mechanisms [17–19]. In view of the fact that subjects were provided with no information concerning this variable at any time in the experiment, the dramatic improvement in CO_2 recovery among E subjects rather convincingly argues for a long-term change in central control mechanisms. Hence, our results do, indeed, suggest that breathing therapy is capable of altering central control of respiration.

A second possible criticism of this study is that the therapist may have influenced the differential effects between groups by means other than the treatments themselves; in other words, experimenter bias may explain the differential effects. Within the experimental design, we attempted to address this issue by eliminating as much as possible any elements not related directly to the ventilatory training program and by comparing treatments differing in quantitative, rather than qualitative dimensions (i.e. extensiveness of ventilatory training). The fact that there was a parallel between degree of ventilatory improvement and extensiveness of training suggests a lack of significant experimenter bias, as do the correlations between ventilatory and psychological changes. Furthermore, it would seem that no bias could slip into the treatment of the data, since all our measures were either physiological variables or objective, subject-completed questionnaire data and were scored and analyzed blind to group membership of individual subjects.

A third, related question arises concerning the nonspecific, or placebo, effects of the experimental treatment. We dealt with this problem by presenting to each treatment group the same amounts of therapist contact and active home assignments. The major differences between treatments were that C subjects were not trained to lengthen their respiratory cycles, did not have a training device for home use and, consequently, had not the opportunity for feedback practice at home. We cannot, therefore, deny the possibility that there were some nonspecific benefits gained by E group subjects merely enjoying possession of the device and being assigned an extra hour's *passive* practice per day with the device in the feedback mode. However, it seem unlikely that these factors, alone, could account for the rather large discrepancies in treatment effects between groups.

Another issue requiring discussion pertains to the clinical implications of our research. Both the mean levels of improvement and the number of subjects 'markedly' improved within the E group, as well as the range of categories of change, imply that the E therapy may have some clinical relevance for treatment of HVS patients. However, the clinical significance of our investigation must be assessed with caution for a few reasons: (1) Although most E subjects showed 'marked' improvement according to our criteria, the majority of this group had not yet progressed sufficiently, at four weeks post-treatment, to be completely free of HVS-associated symptoms. (2) Due to experimental constraints, we were unable to follow-up subjects beyond one month post-treatment. Hence, we have no idea concerning how long the effects of therapy may last subsequent to the first month post-treatment. Until future research addresses these points, clinical applications of our therapy may be premature.

Despite the limitations of our study, we believe that the findings may have significance both specifically for HVS and more broadly for general psychophysiological matters. With regard to HVS, our investigation tends to confirm claims that alterations in ventilatory parameters are central to symptom formation and alleviation. Consequently, ventilatory-training therapies may eventually prove to be an effective

mode of HVS treatment. In a broader sense, the findings indicate that by means of direct voluntary training of respiration, it may be possible to effect long-term alterations in ventilatory control mechanisms. This may have implications for the behavioral treatment of other respiratory disorders (e.g. asthma, sleep apnea and emphysema). Since alterations in ventilatory parameters are known to induce substantial changes in a range of other physiological systems (e.g. cardiovascular and CNS; see [16]), long-term modification of ventilatory control, via breathing therapy, may also be useful in treating specific disorders of these other systems. Finally, the fact that the experimental therapy resulted in improved scores on both situational and dispositional psychological dimensions suggests that respiratory processes could be importantly related to general psychological functioning and supports other reports which provide evidence that variations in breathing pattern may modulate stress responses both over short- and long-term periods [16]. Taken together, these indications point to the possibility that respiratory processes may play an important role in the etiology and alleviation of psychosomatic disorders.

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