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Physiological and psychological effects of acute intentional hyperventilation*

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Summary—Research is reviewed which suggests that hyperventilation syndrome is an underdiagnosed disorder for the presentation of many patients experiencing apparent anxiety states. In a test of this hypothesis, 21 normal individuals (9 female) underwent a 2 min period of intentional hyperventilation following a 10 min baseline phase. Hyperventilation was accompanied by increased subjective anxiety and tachycardia, and indications of peripheral vasoconstriction. Following hyperventilation, *S*s experienced increased levels of state anxiety and perceived autonomic arousal, as indexed by self-report instruments. These results support the hypothesis that undiagnosed hyperventilatory phenomena may be etiologically implicated in states of pathologic anxiety.

INTRODUCTION

In recent years, it has become increasingly evident that patients apparently suffering from anxiety states are actually experiencing symptoms secondary to hyperventilation (Lum, 1975; Campernolle, Hoogduin and Joelle, 1979). The reported incidence of hyperventilation syndrome has been estimated to range from 6 to 10% of patients seen by medical specialists (Lum, 1977) and the syndrome is characterized by a broad spectrum of physiological and psychological effects similar to those experienced by patients with Generalized Anxiety Disorder, Panic Disorder or Agoraphobia (American Psychiatric Association, 1980). Apart from general practice, patients with hyperventilation syndrome are often initially seen in emergency rooms, cardiology departments and neurology clinics, only to be eventually referred to psychiatrists or psychologists when no organic basis can be determined for the patient's myriad and complex array of symptoms.

Hyperventilation can be simply defined as the maintenance of ventilatory efforts in excess of metabolic needs, and is not usually detectable through behavioral observation of the patient's breathing (Magarian, 1982). The presence of frequent sighing, complaints of a dry mouth, and observing the patient licking his lips are suggestive indications for hyperventilation and the diagnosis can be more definitely arrived at through a hyperventilation challenge test. If acute intentional hyperventilation for several minutes reproduces many of the patient's symptoms, then the behavioral treatment should be directed toward the modification of breathing patterns (Lum, 1976; Cooke, 1979). Such simple procedures as bag-rebreathing or having the patient hold several breaths can produce dramatic reductions in symptomatology (Kerr, Dalton and Gliebe, 1937). Hyperventilation syndrome is known to occur in children and adolescents (Enzer and Walker, 1967) and long-term follow-up studies have shown that a large proportion of child hyperventilators retain the habit as adults and often present as suffering from chronic anxiety (Herman, Stickler and Lucas, 1981). When correctly diagnosed, the prognosis for patients with hyperventilation syndrome is quite good, however, with 80–90% of patients significantly improving (Campernolle *et al.*, 1979; Lum, 1976).

Much of what is known about hyperventilation syndrome is based upon practitioners' clinical experience with patients suffering from the disorder. Hyperventilation rapidly produces respiratory alkalosis which may lead to cerebral vasoconstriction, a wide range of neurological symptoms such as syncope, dizziness, tingling in the extremities and numerous other complaints, such as shortness of breath, tremors, weakness, subjective fear and chest pain (Lum, 1975). The similarity of this symptomatic picture to that seen in the anxiety disorders in obvious. Somewhat surprisingly, hyperventilation syndrome is not listed as a differential diagnosis for any of the anxiety disorders (American Psychiatric Association, 1980).

Although it is known that hyperventilation and anxiety are intimately related, the directionality of this relationship remains unclear. Most research on the effects of hyperventilation in normal *S*s concerns the areas of blood chemistry changes and hemodynamics (see review by Magarian, 1982). It is known that acute anxiety may lead to hyperventilation in both patients and normal *S*s (Magarian, 1982), and that hyperventilation reportedly leads to anxiety symptomatology in patients (Stead and Warren, 1973; Lum, 1975). Relatively little work, however, has been conducted on the psychological and physiological effects of hyperventilation in normal *S*s. The present study was designed to determine if acute, intentional hyperventilation in normal individuals would result in increases in subjective and autonomic measures of anxiety. Failure to find such a directional relationship would weaken the hypothesis that hyperventilation syndrome is an etiology for apparent anxiety states, as postulated by Lum (1975) and others (Campernolle *et al.*, 1979; Cooke, 1979; Tucker, 1963).

METHOD

Subjects

The *S*s were 21 adults (9 female) with a mean age of 21 yr. All *S*s were undergraduate students and in good health.

Measures

Anxiety was measured using the Subjective Units of Distress Scale (SUDS), a 100-point analogue rating scale, with 0

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representing complete relaxation and 100 maximum anxiety or panic (Wolpe, 1973). A further self-report index of anxiety was the State–Trait Anxiety Inventory (STAI; Spielberger, Gorsuch and Lushene, 1970), a 20-item rapidly completed pencil-and-paper measure. The STAI has a range of scores from 20 to 80, with higher rating reflective of increased acute anxiety.

Autonomic measures of anxiety included heart rate (HR), peripheral vasoconstriction (finger temperature, FT) and Borkovec's (1976) Autonomic Perception Questionnaire (APQ). HR was monitored with a Gulf and Western Cardiotach (Model 4600), with the photoplethysmograph attached to the index finger of the *S*'s left hand. The *S*'s HR in beats per minute (bpm) was displayed based upon the average beat-to-beat interval every 4 sec. Peripheral vasoconstriction was monitored with an Autogen 2000B feed back thermometer. The thermister was attached to the middle finger of the *S*'s left hand. Both HR (Obrist, 1976) and peripheral vasoconstriction (Thyer, Papsdorf, Davis and Vallecorsa, 1984) are known to be associated with autonomic arousal and are commonly employed in research on anxiety. The APQ is a 37-item pencil-and-paper measure which has the *S* report the extent (s)he is presently experiencing a number of autonomic symptoms. The APQ has a range of scores from 0–138, with higher scores reflective of increased perceived autonomic arousal.

Procedure

After the *S* arrived at the laboratory, (s)he was seated facing the experimenter at a table in a sound-attenuated chamber and had the sensors attached to his/her left hand. All *S*s were then given the following instructions:

> This is a study of the physiological effects of a brief period of rapid breathing. I will record data from your sensors for 10 min, then ask you to breathe rapidly for 2 min, to be followed by a second 10 min period of normal breathing. During the experiment, please remain still and try not to talk. Do you have any questions?

Any questions were answered and the SUDS scale explained to the *S*. The first baseline was begun and measures recorded of the *S*'s SUDS, HR and FT every 30 sec. At the end of 10 min, the experimenter administered the STAI and the APQ. When the pencil-and-paper measures were complete the experimenter stated, "Alright, I'd like you to begin breathing

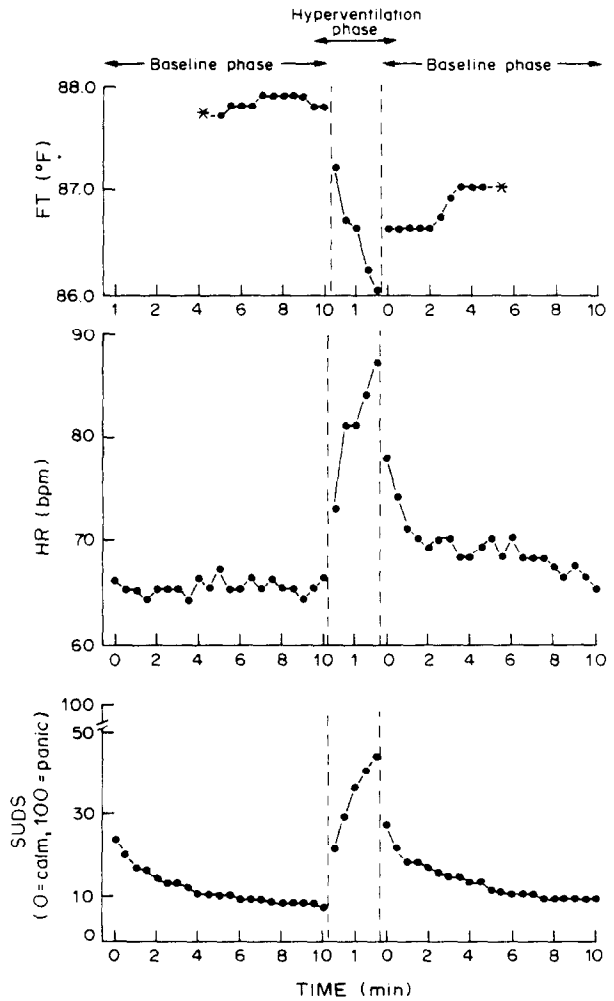

Fig. 1. Mean FT, HR and SUDS values during each time Interval. *Data not available.

Table 1. Mean SUDS, HR and FT scores during each experimental period

Dependent measure	Time period: First baseline $\bar{X}$	First baseline SD	Hyperventilation phase $\bar{X}$	Hyperventilation phase SD	Second baseline $\bar{X}$	Second baseline SD
SUDS	10.77	5.86	33.86	19.17	12.60	8.44
HR (bpm)	65.59	11.66	81.04	14.58	68.91	14.09
FT (°F)	87.20	7.49	86.55	7.25	86.40	7.51

Table 2. Mean STAI and APQ scores pre- and post-hyperventilation

Dependent measure	Time period: Immediately before hyperventilation $\bar{X}$	Immediately before hyperventilation SD	Immediately after hyperventilation $\bar{X}$	Immediately after hyperventilation SD
STAI	33.33	7.25	44.57	9.12
APQ	12.57	7.54	27.66	14.65

rapidly at about one breath per second like this". The experimenter demonstrated the proper breathing rate and volume and then asked the *S* to begin. HR, SUDS and FT were recorded at 30-sec intervals during the 2 min of hyperventilation. The STAI and APQ were administered immediately after the cessation of rapid breathing. Measures were recorded during the second baseline phase as in the initial period.

RESULTS

The SUDS, HR and FT data were averaged for all *S*s at each 30-sec interval of the experiment. These values are displayed in Fig. 1 and indicates that acute, intentional hyperventilation was associated with increased subjective anxiety, mild tachycardia and an apparent decrease in FT. The data for each separate phase of the experiment were averaged and these mean values and their standard deviations are presented in Table 1. A one-way ANOVA for repeated measures was conducted on the mean scores for each dependent variable across the three phases and revealed a significant effect for subjective anxiety changes [$F(2,18) = 20.47$, $P < 0.0001$] and for HR [$F(2, 18) = 22.47$, $P < 0.0001$]. A similar analysis performed on FT scores failed to reach significance [$F(2, 18) = 1.18$, $P = 0.32$]. The mean data for STAI and APQ scores taken pre- and post-hyperventilation is displayed in Table 2. As assessed by pairwise t-tests, both state anxiety ($t = 6.36$, $P < 0.001$) and perceived autonomic arousal ($t = -5.86$, $P < 0.001$) were observed to significantly increase following hyperventilation. These data support the hypothesis that brief periods of hyperventilation can produce acute and significant changes in an individual's level of anxiety, as assessed by a number of self-report measures of subjective discomfort (SUDS, STAI scores) and autonomic arousal (APQ scores), and by HR. Changes in FT observed during hyperventilation also suggested that increased peripheral vasoconstriction, a well-established index of anxiety, was also a sequelae to excessive ventilatory efforts.

DISCUSSION

Previous studies have found that various stress-induction procedures can produce mild hyperventilation (Garssen, 1980; Suess, Alexander, Smith, Sweeney and Marion, 1980). The present results clearly document the converse, acute hyperventilation can result in fairly dramatic and pervasive physiological and psychological effects. The immediate consequences of hyperventilation include clinically significant elevations in subjective anxiety, moderate tachycardia, mild peripheral vasoconstriction (cold hands?) and a much greater sensitivity to autonomic arousal. These documented effects of hyperventilation bear a striking resemblance to the symptomatic profile presented in Generalized Anxiety Disorder and the apparently 'spontaneous' attacks of acute anxiety characteristic of Panic Disorder and Agoraphobia (American Psychiatric Association, 1980).

There is some additional evidence which bears on the contingency relationships between hyperventilatory phenomena and states of pathologic anxiety. A number of clinical reports have documented the efficacy of CO_2 therapy in reducing neurotic anxiety (Wolpe, 1973; Latimer, 1977). Somewhat better controlled investigations have also shown that brief inhalations of CO_2 decrease the subjective anxiety in both neurotic patients (van den Hout and Griez, 1982; Slater and Leavy, 1966) and normal *S*s (Ley and Walker, 1973), as well as reducing chronic anxiety in patients with lactate-inducable panic attacks (Haslam, 1974).

The present data documenting the anxiety-inducing effects of acute rapid breathing and previous research on the anxiolytic effects of CO_2 therapy, suggest that more careful consideration be given to hyperventilatory phenomena as a differential diagnosis for apparent anxiety disorders than the current diagnostic criteria indicate. A hyperventilation challenge may be given to patients not currently anxious, or brief breath-holding or a bag-rebreathing test conducted on individuals acutely anxious, in order to test the hypothesis that the patient is suffering from hyperventilation syndrome. If the diagnosis is supported, relatively simple behavioral treatments may provide the patient with significant relief (Campernolle *et al.*, 1979; Lum, 1976; Cooke, 1979).

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