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THE EFFECTS OF HYPERVENTILATION; INDIVIDUAL VARIABILITY AND ITS RELATION TO PERSONALITY

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Summary—Self-reported affective and somatic disturbances and heart rate changes resulting from a brief period of voluntary hyperventilation are presented and related to individuals' Eysenck Personality Questionnaire (E.P.Q.) scores. Considerable individual variability was observed in the effects of hyperventilation. Neuroticism was significantly correlated with affective but not self-reported somatic or objectively measured heart rate changes. Other possible determinants of observed variability and its possible relevance to the etiology of panic attacks are discussed.

INTRODUCTION

It has been known for many years that there is considerable individual variability in the effects of hyperventilation (*cf.* Kerr, Dalton and Gliebe, 1937). However, systematic investigation of this and its correlates is lacking. This is unfortunate as there is currently a resurgence of interest in the possible role of hyperventilation in the aetiology and/or maintenance of a variety of neurotic disorders (Lum, 1975, 1976; Gibson, 1978; Pfeffer, 1978; Clark, 1979) and information on individual differences in its effects could be helpful in clarifying this role.

Personality is likely to be one of the variables mediating response to hyperventilation. Two personality dimensions, neuroticism and extraversion, as measured by the Eysenck Personality Questionnaire (Eysenck and Eysenck, 1975), have been most consistently shown to mediate emotional responsiveness; it would therefore be of particular interest to assess how the effects of hyperventilation relate to these. None of the stimuli discussed by the Eysencks in theoretical formulations of their personality dimensions, or used in experimental investigations of them, are obviously similar to overbreathing, and so firm predictions relating them to the effects of hyperventilation cannot be made. However, neuroticism has been shown to mediate magnitude of response to a wide range of stressful stimuli (*cf.* Eysenck, 1967; Sipprelle *et al.*, 1977). In addition, the dysthymic group of disorders are characterised by high N (neuroticism) scores. Agoraphobia and anxiety states belong to this group and Clark (1979) has argued that both may be partially caused by hyperventilation. On these grounds a positive correlation between neuroticism and the effects of hyperventilation might be expected. Studies of the relationship between extraversion and response magnitude have tended to find that introverts respond more strongly at low stimulus intensities and extroverts more strongly at high stimulus intensities. Unfortunately we have no grounds for categorizing a particular degree of hyperventilation as a high or low intensity stimulus in this context, and so predictions concerning the relationship between extraversion and the effects of hyperventilation cannot be derived from these studies. However, as dysthymics have lower E (extraversion) scores than normals (Eysenck, 1967) there is some

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reason for anticipating a negative correlation. Current theorising on the nature of psychoticism, the Eysencks' third personality dimension, and experimental investigations of it, fail to suggest a hypothesis linking it with the effects of hyperventilation.

The present study had two aims (i) to collect information on the effects of hyperventilation in a standardized fashion, (ii) to relate individual differences in these effects to E.P.Q. scores.

METHOD

Subjects

Subjects were 27 female students (16 clinical psychology graduate students and 11 nursing students). Means and standard deviations for age, E.P.Q. and resting arterial $paCO_2$ are shown in Table 1. The importance of arterial $paCO_2$ levels and the way they were obtained is described in the measures section.

Table 1

	Mean	S.D.
Age	25.20	4.20
Psychotisism	3.41	2.69
Extraversion	13.48	3.90
Neuroticism	11.34	5.90
Lie	3.44	2.90
Resting arterial $paCO_2$	38.35	3.00

Measures

Arterial $paCO_2$. The most accurate indicant of alveolar ventilation is the partial pressure of carbon dioxide in arterial blood. This was determined non-invasively by a much shortened version of Campbell and Howell's (1962) rebreathing method. One, 30 sec, period of rebreathing gas initially of 5% CO_2/95% O_2 was used in the shortened version, details of which are given in Clark (1979), where a demonstration of its high reliability and validity is also given. The air samples obtained by the rebreathing method were analysed using a Godard Capnograph type 146s.

Respiration. This was recorded with a nasal thermistor suspended immediately in front of one nostril, using a bent paper clip taped to the bridge of the nose. The signal was recorded on paper by a Grass Polygraph (model 7, D.C. preamplifier 71A, time constant of one second). Respiration rate was determined by counting full inspiration-expiration cycles in 30 sec epochs.

Heart rate. An electrocardiogram signal was obtained with standard plate electrodes placed on the outside of each wrist and a 10 mm cup electrode placed, as earth, on the collarbone. The signal was again recorded on paper by a Grass Polygraph (model 7, D.C. preamplifier 71A, P.G.R. input setting). Heart rate was calculated from number of QRS Complexes in 30 sec epochs. Filtering and amplification were such that these were very visible and movement artifacts negligible.

Self-report. Symptoms Rating sheet; this consisted of most of the body sensations and affective changes which have previously been reported as effects of hyperventilation, five items not usually associated with it and intended as response bias items, and a space in which to write any sensations experienced but not listed. Increases occurring in each item during and/or just after overbreathing were rated on a five-point severity scale ranging from "not at all" to "very severe".

Procedure

On arrival at the laboratory each subject was seated in a comfortable, reclining chair, and told that she would be participating in a study of the effects on the body of 2 min fast, deep breathing. Respiration and heart rate transducers were then attached and instruction in the rebreathing technique for estimating arterial $paCO_2$ given. A practice $paCO_2$ measurement was taken to familiarise the subject with the technique. Next, the subject was asked to sit quietly for 3 min during which time baseline measures of respiration rate and heart rate were obtained. The last 2 min intervals of this period were used in the heart rate change calculations reported in the results section. At the end of the 3 min, a baseline measure of ventilation (arterial $paCO_2$) was obtained and the subject was then asked to follow a tape which paced respiration at 30 breaths per minute and lasted 2 min. The subject was instructed to breathe in as deeply as possible, through the mouth as well as the nose, each time the voice on the tape said IN, and to breathe out, completely emptying her lungs, each time it said OUT. The respiration trace on the polygraph was used to check that the tape had been followed. Immediately after the 2 minutes' hyperventilation the subject was asked to, "Relax and take note of any sensations you might be experiencing". Forty-five seconds later arterial $paCO_2$ was again measured so that the change in ventilation which had occurred as a result of following the pacing tape could be calculated. The 45 sec delay between the end of the tape and the $paCO_2$ measurement was intended to allow subjects some time to introspect on the effects of hyperventilation while undisturbed. Previous investigation had shown that $paCO_2$ did not rise appreciably during this delay although it did if the delay was 1½ min or longer. Finally, each subject completed the Symptoms Rating Sheet.

RESULTS

All subjects were able to follow the pacing tape and increased their ventilation while doing so (mean arterial $paCO_2$ decrease, 10.91 mm.Hg. S.D., 2.69 mm.Hg). No subject had an average score on the response bias items greater than 50% of their average score on those relating to hyperventilation. Hence the level of indiscriminate responding was sufficiently low to

allow all subjects to be retained for subsequent analysis.

The mean increase in heart rate during hyperventilation was 31.6 beats (S.D. = 12.9, range = 10–50).

Symptoms Rating Sheet data is presented in Tables 2 and 3. From Table 2 it can be seen that only one item—faintness, was reported by everyone as an effect of hyperventilation. Over 90% reported increases in dizziness and weakness; over 80% increases in apprehension and tingling; over 70% increases in anxiousness, feelings of unreality, a dry mouth, shakiness, heart pounding, heart racing and sweating. The three least frequently reported items listed on the sheet and not response bias items were nausea (29%), headache (29%) and muscle pain (18%). Inspection of the range column in Table 2 indicates that there was considerable variability in the severity with which many items were experienced.

Fifty-two per cent of subjects reported some

Table 2

Item	Slight	Moderate	Severe	Very severe	Mean	S.D.	Range
Central neurological							
Unreality	79	61	32	14	1.8	(1.4)	0–4
Dizziness	93	75	43	11	2.3	(1.1)	0–3
Faintness	100	64	32	11	2.1	(1.0)	1–4
Peripheral neurological							
Numbness	61	50	18	0	1.4	(1.2)	0–3
Coldness	36	0	18	0	0.8	(1.2)	0–3
Tingling	86	75	54	25	2.5	(1.3)	0–4
Pins and needles	64	57	32	7	1.7	(1.4)	0–4
Muscular							
Tremor	54	11	4	0	0.7	(0.8)	0–3
Shakiness	75	46	14	4	1.4	(1.1)	0–4
Tight muscles	46	25	4	0	0.8	(0.9)	0–3
Muscle pain	18	11	0	0	0.2	(0.6)	0–2
Cardiovascular							
Heart racing	75	39	14	7	1.3	(1.1)	0–4
Heart pounding	71	39	11	0	1.2	(1.0)	0–3
Gastro-intestinal							
Nausea	29	7	0	0	0.4	(0.6)	0–2
Other somatic							
Dry mouth	75	57	25	7	1.6	(1.2)	0–4
Headache	29	11	0	4	0.4	(0.9)	0–4
Sweating	75	32	14	4	1.2	(1.0)	0–4
Weakness	96	50	25	0	1.7	(0.9)	0–3
Affective							
Apprehension	82	36	14	7	1.4	(1.1)	0–4
Anxiousness	79	29	11	0	1.2	(0.9)	0–3
Tension	64	29	11	0	1.0	(0.9)	0–3
Unhappiness	39	11	4	0	0.6	(0.8)	0–3
Response bias							
Catarrh	21	4	0	0	0.3	(0.5)	0–2
Drowsiness	43	14	7	0	0.7	(0.9)	0–3
Stinging	4	0	0	0	0.04	(0.1)	0–1
Choking	18	0	4	0	0.2	(0.6)	0–3
Earache	4	0	0	0	0.04	(0.1)	0–1

Columns 1–4 show the percentage of subjects reporting an increase in each item on the Symptoms Rating Sheet during and/or just after hyperventilation. Figures in the "slight" column are percentages of people reporting an increase of that magnitude or greater, in the "moderate" column of that magnitude or greater and so on. To derive the means, standard deviations and ranges given in columns 5 to 7 "not at all" was scored as 0, "slight" as 1, "moderate" as 2, "severe" as 3 and "very severe" as 4.

sensations which had not been listed on the Symptoms Rating Sheet. These were—Panic (1 subject), Fear (1), Pleasure (1), Vision Blurred (2), Vision Sharpened (1), Sounds Altered (1), Stomach Churning (1), Stomach Spasm (1), Hot (2), Floating Sensation (1), Lightness (2), Clamminess (1), Painful Throat (1), Head Throbbing (1), Drumming in Arms (1), Creeping Sensations in Arms (1), Brief Paralysis (1), Breathlessness (2).

Inspection of the polygraph respiration trace for the two individuals reporting breathlessness indicated that after hyperventilating they were breathing at a much faster rate than they had been before. For some other subjects the polygraph record suggested that they had hardly breathed at all for 10–20 sec after hyperventilating.

Symptoms Rating Sheet items varied from individual to individual in their relative intensity. For example one subject reported a headache as one of the two most intense effects of hyperventilation that she experienced even though in the group as a whole headaches were one of the least frequently experienced effects. Table 3 illustrates individual variability in response patterning. In addition it shows that tingling was reported more frequently in the highest intensity category subjects used than any other items.

Intercorrelations between the Symptoms Rating Sheet, E.P.Q., heart rate increase and arterial

Table 3

Subject	Symptoms Rating Sheet items																					Subject's highest intensity category
	Unreality	Dizziness	Faintness	Numbness	Coldness	Tingling	Pins & needles	Tremor	Shakiness	Tight muscles	Muscle pain	Heart racing	Heart pounding	Nausea	Dry mouth	Headache	Sweating	Weakness	Apprehension	Anxiousness	Tension	Unhappiness	
1	×					×	×												×				4
2		×							×						×								2
3		×	×			×	×								×								2
4		×		×		×	×					×	×			×							2
5		×	×	×		×	×								×				×				2
6	×			×		×									×								3
7				×			×			×					×		×						2
8						×									×				×	×			2
9							×																3
10						×																	4
11		×	×			×									×								4
12	×	×				×			×														4
13	×	×	×									×	×		×			×					2
14		×				×	×					×											4
15	×					×																	3
16			×																				4
17						×	×																3
18	×	×		×		×	×					×	×			×							3
19		×				×																	3
20			×													×							4
21						×									×								4
22						×																	4
23	×	×	×									×	×					×	×	×			3
24	×		×	×															×				3
25																			×				4
26		×	×												×								3
27			×																			×	2

Crosses indicate items endorsed in the highest intensity category a subject used. Scoring of intensity categories is the same as that used in Table 2.

Table 4

	P	E	N	Aff	Som	A/S	↓paCO$_2$	↑H.R.
P		−0.19	0.04	−0.20	0.20	−0.29	0.04	0.11
E			0.07	0.10	0.16	−0.06	0.24	0.31
N				0.37*	0.19	0.36*	0.02	0.10
Aff					0.51**	—	0.29	0.30
Som						—	0.41**	0.45**
A/S							0.16	0.06
↓paCO$_2$								0.67**

*$P < 0.05$.
**$P < 0.01$ (1-tailed t-tests).

paCO$_2$ decrease were computed and the Pearson product moment correlation coefficients obtained are presented in Table 4. Linearity was verified for all significant correlations by visual inspection of scattergrams. For the purpose of computing correlations individuals' Symptom Rating Sheet responses were reduced to three numbers—affect score, somatic score and the ratio of these two (A/S). These were derived by scoring an item for which "not at all" was endorsed = 0, "slight" = 1, "moderate" = 2, "severe" = 3 and "very severe" = 4 and adding up the scores on apprehension, anxiousness, tension and unhappiness for the affect score and on the remaining items, excluding the response bias ones, for the somatic score.

Somatic score and increase in heart rate were both significantly correlated with arterial paCO$_2$ decrease and also with each other. Computation of the first-order partial correlation coefficient between these two variables, controlling for the effects of paCO$_2$ decrease, suggested that the latter correlation was a spurious one largely accounted for by the positive correlation of both variables with paCO$_2$ decrease (in the partialling procedure r dropped from 0.45 to 0.25). Clearly the effects of paCO$_2$ decrease on somatic score and heart rate increase are largely independent.

A/S—the relative intensity of affective and somatic disturbances, was unrelated to heart rate increase ($r = 0.06$).

The only significant correlations involving the E.P.Q. were between neuroticism and affective score ($r = 0.37$, $P < 0.05$) and neuroticism and A/S ($r = 0.36$, $P < 0.05$) though a trend for psychoticism to correlate negatively with A/S ($r = -0.29$, $P < 0.14$, 2-tailed t-test) and extraversion to correlate with heart rate increase ($r = 0.31$, $P < 0.12$, 2-tailed t-test) was apparent. First-order partial correlation coefficients were computed to determine whether the latter two relationships were being masked in this sample by correlations with other variables. The correlation of psychoticism with A/S increased but remained non-significant ($r = -0.33$, $P < 0.10$) when the effects of neuroticism were controlled for in this manner while other correlation coefficients were largely unaffected. The correlation between extraversion and heart rate increase was substantially reduced ($r = 0.31$ to $r = 0.20$) when paCO$_2$ decrease was controlled for. Other correlations were again unaffected.

DISCUSSION

Considerable individual variability, both in the magnitude and type of response to 2 min hyperventilation was observed. To some extent this variability can be accounted for by differences in the degree to which subjects increased their ventilation as paCO$_2$ decrease correlated 0.67 with heart rate increase, 0.41 with somatic score and 0.29 with affective score. However, this still leaves a large amount of variance unexplained, particularly in affective response. Neuroticism correlated most highly with this and also A/S—but these correlations account for only about 13% of the variance. Variability which the aforementioned conspicuously fail to account for is the pleasure that one subject experienced just after hyperventilation and the

visual sharpening (in contrast to the more usually reported blurring) that another experienced. The latter seems most likely to be due to vasomotor idiosyncrasies while the former could be due to the subject's previous experience of many of the sensations induced by hyperventilation in a pleasant context (taking drugs with friends). Mood state at the start of the experiment and confidence in the competence of the experimenter might have been other determinants of variability, particularly in affective response. Sargant (1973) points out that hyperventilation has been reported in a variety of emotions, ranging from orgasm and trance states, to panic. Its effects on affect are no doubt partially determined by the psychological context in which it occurs in the same way as the effects of mescalin (Huxley, 1956) and noradrenalin (Schacter and Singer, 1962) are.

It was hypothesized that the effects of hyperventilation would correlate positively with neuroticism and negatively with extraversion. Results supporting the neuroticism hypothesis were obtained; however, no support was obtained for that relating to extraversion. Although the latter might be a genuine finding, it is possible that it is a range effect as the variability of E in this sample was small and clearly less than that of N. The trend for A/S to correlate negatively with psychoticism was not predicted, and warrants further investigation as it occurred despite very small variability in *P*. If confirmed, an extension of Eysenck and Eysenck's (1975) theorizing on the nature of psychoticism will be required. They describe high *P* scores as "lacking in feeling" (1975, p. 11), but are referring to responsiveness to social cues and distress in others, not reactivity to somatic disturbances.

Patients who suffer from panic attacks are more disturbed by a brief period of voluntary hyperventilation (Lewis, 1959) or the chemical induction of plasma alkalosis* (*cf.* Pitts and McClure, 1967; Grosz and Farmer, 1972) than are normals. This could simply be a function of having experienced panic attacks, however, it could also be a premorbid personality characteristic. The correlation of N with affective response to hyperventilation observed in the present study is consistent with the latter but the failure to obtain a correlation with E is not, as Eysenck (1967) argues that it is the combination of both a high N and a low E score which predisposes individuals to dysthymic disorders.

Several authors have pointed out that panic attacks can be caused by a vicious circle of apprehension leading to increased ventilation, the effects of which lead to increased apprehension which further increases ventilation and so on. Whether or not this occurs clearly depends, among other things, on individual differences in the extent to which people respond to stressors with an increase in ventilation, the extent to which this is self-maintaining by inducing a state of breathlessness and the extent to which it induces apprehension. Only the latter two sources of variability have been investigated in this study and work on the former is clearly required.

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*Hyperventilation induces plasma alkalosis by "blowing off" alveolar (and hence arterial) $paCO_2$.

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