Neurological manifestations of the hyperventilation syndrome

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Summary

Seventy-eight patients with the hyperventilation syndrome are described, none of whom had been diagnosed prior to referral for neurological assessment. There is a constellation of symptoms associated with this condition, and attacks need to be reproduced by hyperventilation in order for the diagnosis to be established. Loss of consciousness and paraesthesiae were more frequent than in previously published series.

Introduction

At the beginning of the century, Sir William Gowers described patients with 'reversible vagal attacks', comprising pneumogastric symptoms, tetanoid spasms, an altered mental state and non-epileptic seizures[1]. There is little doubt that many of these patients had the hyperventilation syndrome (HVS) though this was not recognized as a clinical entity until 1929, when White and Hahn[2] described patients with 'sighing dyspnoea' who displayed similar clinical characteristics. Neurological manifestations of this condition include giddiness, paraesthesiae, visual disturbances and loss of consciousness. The symptoms are sometimes focal, and the condition frequently goes unrecognized. Patients have often had extensive investigation prior to diagnosis.

We describe here the manifestations of 78 patients with HVS who were referred to hospital for neurological assessment of their symptoms.

Patients and methods

During the period 1977–1983, 78 patients eventually diagnosed as having the hyperventilation syndrome were referred to GDP for outpatient neurological assessment. There were 52 women and 26 men (Table 1). The age at onset of symptoms ranged from 9 to 57 years, with 73.1% of women and 76.9% of men developing their symptoms between the ages of 10 and 39. The ratio of women to men was greatest in those with onset of symptoms in the sixth decade. When first seen, symptoms had been present for periods ranging from 2 months to 32 years. The basis for each patient's symptoms was confirmed by their reproduction during hyperventilation. All patients had a full neurological examination. Further investigations were performed in some, depending on the nature of their complaint.

Table 1. Patient details

Age at onset (years)	*No.*	*Sex distribution (M:F)*	*Duration of symptoms at presentation (mean)*	
			Male	*Female*
1–9	4	3: 1	2 mths	11 yrs
10–19	15	4:11	12 yrs	4 yrs
20–29	28	11:17	3 yrs	3 yrs
30–39	15	5:10	2 yrs	2 yrs
40–49	8	2: 6	2 yrs	3 yrs
50–59	8	1: 7	1 yrs	2 yrs
Total	78	26:52	3 yrs	4 yrs

Results

The major symptoms associated with attacks are summarized in Table 2.

Giddiness: This included dizziness, faintness, light headedness and vertigo. These were the commonest complaints and were present in 46 patients (59%).

Paraesthesiae: Twenty-eight patients (36%) had sensory symptoms, either numbness or paraesthesiae. Paraesthesiae were present in the upper limbs in 25, lower limbs in 10, face in 8 and on the trunk in 5 patients. Some patients had symptoms at more than one site. Paraesthesiae were unilateral in 7 patients (10%), 5 involving the left upper limb, and 2 the left side of the face.

Loss of consciousness: Twenty-four patients (31%) complained of loss of consciousness during, but not between, attacks. It was an occasional feature of severe attacks in 22 and a regular feature of all episodes in 2 patients. Though they occurred in

Table 2. Symptoms during attacks ●

	No.	*%*
Neurological		
Giddiness	46	59
Paraesthesiae	28	36
Loss of consciousness	24	31
Visual disturbance	22	28
Headache	17	22
Ataxia	14	18
Tremor	8	10
Tinnitus	2	3
Cardiorespiratory		
Dyspnoea	41	53
Palpitations	33	42
Chest discomfort	6	8
Gastrointestinal		
Nausea	15	19
Abdominal pain	1	1
Vomiting	1	1

● Most patients had more than one symptom

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Table 3. Referral diagnoses

	Number
Epilepsy	12
Vaso-vagal attacks	9
Multiple sclerosis	3
Functional	4
Migraine	3
Hypoglycaemia	1
Cerebral tumour	1
Vertebro-basilar insufficiency	1
No diagnosis	44
	78

any environment, attacks of loss of consciousness had been witnessed in only 8 patients. The exact duration of unconsciousness proved difficult to establish with certainty, most patients simply describing brief episodes. Convulsive movements or a bitten tongue did not occur and only one patient had had urinary incontinence. Loss of consciousness was unrelated to posture. Six patients had sustained head injuries from falls during their attacks. None had a past history of epilepsy, though one had received a trial of anticonvulsants.

Visual disturbance: Blurring of vision, loss of vision, photophobia or flashing lights occurred in 22 patients (28%). Momentary flashing lights occurred in 2 patients, but without the characteristics of the teichopsia associated with migraine.

Headache: Seventeen patients (22%) complained of nonspecific headache. The pain was generalized and, when associated with nausea, had often been diagnosed previously as migraine.

Ataxia was present in 14 (18%), tremulousness in 8 (10%) and tinnitus in 2 (3%).

Cardiorespiratory and gastrointestinal symptoms were frequent. Dyspnoea, typically described as an inability to get enough air into the chest, was present in 41 (53%). Palpitations occurred in 33 (42%), chest discomfort in 6 (8%) and nausea in 15 (19%). One patient vomited during attacks.

More chronic complaints were present in 63 patients (81%). Headaches of tension type occurred in 27, insomnia in 19, tiredness in 6, abdominal pain in 7 and chest discomfort in 9. No patient gave a history of migraine or ischaemic heart disease. Stress factors related to marital, domestic or financial problems were elicited in 40 patients.

Physical examination did not reveal any consistent finding. Global, fluctuating motor weakness involving one or more limbs was present in 5 patients, and 2 had unilateral equivocal plantar responses with no other pyramidal signs.

Electroencephalography (EEG) was performed in 17 patients, 2 of whom had unilateral or bilateral excess of theta activity. The recording was normal in the remainder.

In the referral letters to the clinic, a diagnosis had not been suggested in 44 (56%) whilst 12 patients were thought to have epilepsy (Table 3). One patient, diagnosed by a general physician as having complex partial seizures, had been hospitalized twice for 24-hour EEG and sphenoidal EEG recordings, both of which were normal. The diagnosis of HVS had not been considered in any patient.

Discussion

The hyperventilation syndrome appears to be common. Lum[3] has reported on more than 500 patients seen over a decade, concentrating on the cardiorespiratory manifestations of the condition. Amongst 500 successive referrals to an outpatient gastroenterology clinic, 29 (5.8%) were thought to have HVS[4]. In Lum's series the condition was most common between the ages of 15 and 30 in women, whilst in men it steadily climbed to a peak incidence in the sixth decade, where the incidence surpassed that of women. This trend has not been seen by others[5]. In our series the condition was twice as common in women; there were more women in all age groups except those whose symptoms had begun in the first decade (Table 1). The sex and age distribution was similar to that recorded by Ames[6]. All series have reported a female predominance.

Hyperventilation attacks occur mainly at rest. The rapid deep breathing which accompanies exercise is compensatory and hence does not lead to hypocapnia and the symptoms seen with spontaneous hyperventilation. The need to confirm the diagnosis by reproduction of attacks during voluntary hyperventilation has been emphasized[7]. However, the appearance of symptoms depends not only on hypocapnia but also on the accompanying psychological state which may be difficult to reproduce intentionally under observation. All our patients developed symptoms akin to their spontaneous attacks during hyperventilation, often requiring only 2 to 3 breaths to do so.

Episodes of loss of consciousness occurred in 24 patients. They were not accompanied by convulsive movements, incontinence (except in one) or tongue biting, but their presence inevitably suggested the alternative diagnosis of epilepsy, a diagnosis already considered in 12 patients by the referring physician. Electroencephalography, performed in the majority of these patients, failed to reveal epileptic activity. In Lum's experience[3], transient disturbances of consciousness with hyperventilation had frequently been attributed to petit mal (absence attacks). Other series have reported loss of consciousness in HVS with varying frequency, ranging from 6%[5], through 6.9%[4] and 12.5%[6] to 22%[8].

Hyperventilation is well recognized as a precipitant of some forms of seizure, particularly absence attacks. None of our patients showed altered awareness when typical attacks were induced in the clinic by hyperventilation. Patients with complex partial seizures or with attacks originating from the insular cortex may display hyperventilation during the ictus[7], though this is usually accompanied by motor or temporal lobe phenomena.

Descriptions in the literature of the episodes of loss of consciousness in HVS are generally inadequate. Attempts to render them coherent are frustrated by the differing types of attack which authors have included. Some have described, under the umbrella of HVS, attacks of loss of consciousness with tonic-clonic convulsions[9], which clearly would be more satisfactorily classified as epilepsy triggered by hyperventilation. In a proportion of patients, signs will develop during hyperventilation attacks that indicate conversion hysteria, including attacks of hysterical epilepsy. Typically, the patient will display rapid, and sometimes stertorous, respiration throughout[10]. In essence, loss of consciousness

during HVS is brief, with the patient lying quietly, though breathing deeply, and sometimes having a greater awareness of the environment than his appearance might suggest. If limb or truncal movement occurs, the features are either those of tetanic spasm or hysterical epilepsy. It has been suggested that HVS accompanied by loss of consciousness has a worse prognosis and indicates a greater degree of emotional instability[8].

Asymmetrical neurological symptoms, especially unilateral paraesthesiae, may occur in HVS[9,11] and are sometimes interpreted as focal epileptic seizures. Seven of our patients had focal paraesthesiae during attacks, all left-sided. This left-sided predominance has been reported previously[12], and is unexplained.

Hypocapnia consequent to hyperventilation has a number of effects, including cerebral vasoconstriction, alkalosis causing alteration of the ionic balance of calcium, and a shift of the haemoglobin oxygen dissociation curve, reducing oxygen delivery to the tissues. The cumulative effect of these changes impairs oxygenation in the central nervous system and increases the sensitivity of peripheral nerves to lowered ionized calcium.

Management of HVS has been discussed elsewhere[3,7]. Many patients whose attacks have developed recently respond to reassurance that there is no serious underlying neurological disorder. Attacks often respond to rebreathing from a paper bag, though the environment in which the attacks occur may inhibit the patient from practising this manoeuvre. Management of the chronic state of hyperventilation is more difficult. Patients need to be convinced that their symptoms are brought on by forced breathing before they are likely to respond to advice on breathing techniques. They can then be instructed to avoid mannerisms like sighing and deep breathing. Conscious attempts at reducing the respiratory rate and advice about diaphragmatic breathing are recommended. Recently beta-blockers have been shown to depress ventilation via central mechanisms and by suppression of peripheral chemoreceptor drive[13]. These effects may account for the increased $Paco_2$ observed in hyperventilating patients treated with beta-blockers, thereby reversing some of the clinical manifestations of hyperventilation. Where there is evidence of underlying anxiety or depression, psychiatric help may be of value.

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