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Patients With Acute Hyperventilation Presenting to an Inner-City Emergency Department*

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We studied 23 consecutive patients with acute hyperventilation presenting to an inner-city emergency department, diagnosed on clinical grounds by the attending physician and confirmed by arterial blood gas values in 5 patients. An organic basis for the presenting complaints was excluded and chest radiograph, serum biochemistry, blood cell count, and thyroid function test results were normal. The male to female ratio was 12:11. Presenting complaints were dyspnea (61%), paresthesia (35%), chest pain or tightness (43%), muscle spasm (9%), dizziness (13%), palpitations (13%), and panic (30%). Similar previous episodes were reported in 74%. Misattribution of the presenting complaints to a cardiac or other life-threatening disorder was reported in 20 patients (87%) and was the main reason for their presentation to the hospital. Although no patients presented with clinical features of asthma, 7 (30%) were known asthmatics receiving treatment and another 10 (44%) had a history and investigation results suggestive of asthma. Only 2 had a history of anxiety or depression, but 17 (78%) patients exceeded the threshold for anxiety or panic on Clinical Interview Schedule (CIS-R) interview (score ≥12). Marihuana or alcohol abuse were involved in 17% with a history of past abuse in 26%. When assessed 2 months after the attack, 13 (57%) had resting or stressor-induced hyperventilation with a significant ($p<0.05$) association with asthma but not with a positive CIS-R score. These results illustrate the multifactorial basis of acute hyperventilation, the importance of misattribution, and the danger of using the term "hyperventilation syndrome" in the emergency department. ***(CHEST 1996; 110:952-57)***

Key words: anxiety; carbon dioxide; hyperventilation; hypocapnia; misattribution; panic; psychiatric disorder; respiration

Abbreviations: CIS-R=Clinical Interview Schedule; $P_{ET}CO_2$=end-tidal P_{CO_2}; VHV=voluntary overbreathing (voluntary hyperventilation)

There is uncertainty about the definition of "hyperventilation syndrome" and the boundaries of hyperventilation-related disorders.[1-11] Chronic hyperventilation has been documented extensively,[3,6,7] but there is little information about acute and subacute hyperventilation and about the clinical spectrum of patients presenting to emergency departments with apparent acute hyperventilation. Nevertheless, such patients are frequently encountered, and further information is of importance, both to prevent misdiagnosis of serious organic disease, and to initiate the correct course of treatment for these difficult patients.

This lack of information is highlighted by anecdotal reports and descriptions in the literature of patients with severe and potentially life-threatening organic disorders such as myocardial infarction, asthma,[12] and diabetic ketoacidosis[13] being misdiagnosed in emergency departments as hyperventilation syndrome, often with disastrous consequences. Patients with hyperventilation are usually regarded by physicians as either "hysterical" or anxious and within the remit of the psychiatrist. However, hyperventilation implies excessive respiratory drive that can be due to a wide range of organic and psychogenic conditions.[11] Not only does the labeling of a patient as anxious or neurotic tend to impede searching for organic abnormalities, but the commonly applied treatment of rebreathing from a paper bag, for which there is little literature or proof of efficacy, can lead to potentially fatal hypoxia in the presence of lung disorders such as asthma.[14]

We wished to study a consecutive series of patients presenting to a typical busy emergency department, labeled by emergency department physicians on clinical grounds as having "hyperventilation" or "hyperventilation syndrome." We wished to know the cause of the hyperventilation in these patients, and in particular, the relative roles of both acute and chronic psychogenic factors such as anxiety, panic and misattribution, and organic disorders such as

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asthma not apparent on clinical grounds at the time of presentation.

Materials and Methods

Patients

We studied 23 consecutive patients presenting with an episode of acute hyperventilation over a period of 1 year. The patient register was checked to ensure that no patients had been missed over the study period. Diagnosis of acute hyperventilation was made on clinical grounds by the emergency department physicians on the basis of history, observation, and examination, and in five patients arterial blood gas determinations, taken within the first 2 h of presentation to the hospital. In all cases, the primary diagnosis was of hyperventilation or hyperventilation syndrome and other causes of the presenting symptoms were excluded. No patient was referred with any clinical evidence of asthma or other organic disease or any abnormality of chest radiograph, ECG, blood urea, electrolytes, or hemoglobin. No record was available of patients not referred.

Assessment

Patients were referred from the emergency department to our chest clinic where a more detailed history was taken and examination performed some weeks after the initial presentation. Further investigation included, where necessary, a repeat of the emergency department investigation, a more complete biochemical profile, and thyroid function tests. We performed full lung function testing, including measurement of spirometry with reversibility, and single breath carbon monoxide gas transfer without and with correction for lung volume. In some patients, we performed a histamine bronchial provocation test using a technique employing 2-min continuous inhalation of doubling concentrations of histamine via a nebulizer (Wright; Abbey Instruments; Mitcham, Surrey, UK).[15] Psychiatric assessment was performed by a structured psychiatric interview, Clinical Interview Schedule (CIS-R).[16]

Hyperventilation Screen

To detect hyperventilation, end-tidal P_{CO_2} (P_{ETCO_2}) was measured uninvasively for every breath by rapid-response infrared CO_2 analyzer (Gould Capnograph IV; Bilthoven, The Netherlands), sampling via a moisture-permeable fine-bore catheter taped a few millimeters inside one nostril.[7,17] Data were stored and displayed digitally using a data acquisition system (Codas II; Dataq Instruments Inc; Ohio) and analyzed off-line for breath-by-breath measurement of P_{ETCO_2} using an electronic cursor.

P_{ETCO_2} was measured over an approximately 45-min period during a protocol involving the application of various stressors.[7] Rest in a comfortable chair for 10 min was followed by exercise on an electrically braked cycle ergometer at a work load chosen by the subject roughly to simulate the level of activity outside the laboratory. A further 10-min rest was followed by 3 min of voluntary overbreathing (VHV) to a P_{ETCO_2} of about 20 mm Hg with measurement of rate of recovery over the subsequent 10 min. VHV could be regarded as simulating in the laboratory everyday activities such as talking, which might precipitate hyperventilation in real life. P_{ETCO_2} was averaged over every 2 min of the protocol except during VHV when it was averaged every minute. Our criteria for hypocapnia[7,11] were more stringent than is usual. P_{ETCO_2} above 30 mm Hg at rest, at all times during and after exercise, and at 5 min after the end of VHV, was considered normal.

Results

Our patients were almost equally divided between men (12) and women (11). The presenting complaints are listed in Table 1 and were elicited by the emergency department physicians at the time of initial presentation. No patient with an organic cause for their complaints was referred to us, and in all cases, chest radiograph, blood biochemistry, and full blood cell count measured at the time of presentation were normal. No patient presented with any symptoms or signs suggestive of asthma, and results of peak flow measurements were normal.

Table 1—*Complaints at Time of Initial Presentation*

Complaint	Men (%)	Women (%)	All (%)
Anxiety/panic	4/12 (33)	3/11 (9)	7/23 (30)
Palpitations	3/12 (25)	0/11 (0)	3/23 (13)
Dyspnea	7/12 (58)	7/11 (64)	14/23 (61)
Paresthesia	7/12 (58)	1/11 (9)	8/23 (35)
Chest tightness	5/12 (42)	2/11 (18)	7/23 (30)
Chest pain	1/12 (8)	2/11 (18)	3/23 (13)
Faintness/dizziness	2/12 (17)	1/11 (9)	3/23 (13)
Muscle spasm	1/12 (8)	1/11 (9)	2/23 (9)
Hyperventilation	2/12 (17)	2/11 (18)	4/23 (17)

Fourteen of 23 patients (61%) complained of shortness of breath (Table 1). Seven (30%) reported panic and anxiety, associated with palpitations in 13%. Symptoms associated with hypocapnia included paresthesia (35%), chest tightness (30%), chest pain (13%), faintness and dizziness (13%), and muscle spasm (9%). Only 17% recognized that they were hyperventilating. Arterial blood gas measurements were made in 5 patients (Table 2) at various times up to 2 h after the initial presentation. These confirmed severe hypocapnia with associated increase in P_{aO_2} in four subjects, and mild hypocapnia in the fifth. Bicarbonate levels were variably reduced.

When interrogated at the subsequent interview, on average, 7.9±6.2 (mean±SD) weeks (range, 1 to 15 weeks) after the initial presentation, 10 male and 7 female (74%) patients admitted to previous but usually less severe episodes of hyperventilation. In all but 3 patients, the presenting symptoms were misattributed by the patient at the time of presentation to serious or life-threatening disease (Table 3). The most common misattribution, particularly in men, was to heart disease or a myocardial infarction in 11 of 23 (48%), and the next most frequent was to a life-threatening consequence of drug or alcohol abuse in 4 of 23 (17%) with a wide variety of individual misattributions in other patients. Table 4 presents pooled data of past medical and psychiatric history, and Table 5 shows the respiratory history, pulmonary function data, chest radiographic findings, result of the hyperventilation screen, and psychiatric evaluation by CIS-R for individual patients.

Only one patient had no relevant medical history. Of the nonrespiratory disorders, 6 patients (26%) reported a significant history of drug or alcohol abuse. One patient had a prior diagnosis of depression and

Table 2—*Arterial Blood Gas Data Within 2 h of Initial Presentation for Five Subjects*

Patient	$PaCO_2$, mm Hg	PaO_2, mm Hg	HCO_3^-, mEq/L	pH	H^+, nmol/L
1	24.8	108.0	24		25
11	31.5	107.3	20	7.38	
14	20.3	116.3	19		26
20	8.3	129	5.2	7.43	
21	16.5	136.5 (28% O_2)	16		25

another of chronic anxiety with panic. The other nonrespiratory disorders were probably of no relevance to the present episode.

Seven of 23 patients (30%) were known asthmatics currently receiving inhaled treatment for asthma (Table 5). Two more patients, one with a history of hay fever and one with a positive family history of asthma, had a positive histamine challenge. Five patients had a negative histamine challenge but four of these had a history of chestiness and chest tightness strongly suggestive of asthma. In 9 patients in whom a histamine challenge was not performed, 2 had a current history of wheeze and cough with some spirometric values more than 20% below the predicted, and 2 had a history of childhood asthma with normal lung function but hyperinflation on the chest radiograph. Only 5 of 23 (22%) patients showed no evidence of airflow obstruction on history, chest radiograph, or laboratory investigation. Twelve patients were current smokers and two had stopped smoking within the last year.

Only two (9%) patients admitted to a past psychiatric history at the time of the initial history (Table 4). However, on the basis of clinical and the CIS-R interviews (Table 5), 17 (78%) patients exceeded the threshold for psychiatric disorder (score ≥12) with panic in seven (30%) and anxiety in 13 (52%). Both conditions coexisted in 4 (17%) patients.

Ten patients (7 men, 3 women) had a normal hyperventilation screen (Table 5). Thirteen patients (57%; 5 men and 8 women) had positive hyperventilation screens with $PETCO_2$ levels at or less than 30 mm Hg at rest, during or after exercise, or at or beyond 4 min following VHV.

The association among asthma, a positive hyperventilation screen, anxiety, and panic was assessed. In the 9 patients with clear evidence of asthma (known asthmatics or with a positive histamine challenge), all but 1 (89%) had an abnormal hyperventilation screen. This association was significant ($p<0.05$) as assessed by a nonparametric sign test. There was no significant association between a positive hyperventilation screen and anxiety or panic. Of the 13 patients with chronic anxiety, 9 (69%, NS) had an abnormal hyperventilation screen but 6 of these also had asthma. Of the 6 patients with chronic anxiety who did not have asthma, only 2 (33%) had a positive hyperventilation screen. Two of 7 (29%) patients with recurrent panic attacks had a positive hyperventilation screen. Of those, one had asthma.

The results of other routine investigations, including a full blood cell count, biochemical profile, thyroid function, and ECG were all within the normal range.

Table 3—*Patient Attributions at Time of Presentation*

Attribution	Men (%)	Women (%)	All (%)
Heart attack/disease	7/12 (58)	4/11 (36)	11/23 (48)
Drug/alcohol abuse	2/12 (17)	2/11 (18)	4/23 (17)
Stroke/epilepsy	1/12 (8)		1/23 (4)
Lung cancer		1/12 (9)	1/23 (4)
Hypoglycemia		1/11 (9)	1/23 (4)
Fever	1/12 (8)		1/23 (4)
Near death	1/12 (8)		1/23 (4)
No attribution		3/11 (27)	3/23 (13)

Table 4—*Past Medical Problems*

History	Men (%)	Women (%)	All (%)
Current asthma	1/12 (8)	6/11 (55)	7/23 (30)
Childhood asthma	3/12 (25)	0	3/23 (13)
Recurrent bronchitis	1/12 (8)	0	1/23 (4)
Alcohol abuse	4/12 (33)	0	4/23 (17)
Drug abuse	1/12 (8)	1/11 (9)	2/23 (9)
Anxiety/panic	1/12 (8)	0	1/23 (4)
Depression	0	1/11 (9)	1/23 (4)
Diabetes	0	1/11 (9)	1/23 (4)
Menorrhagia	0	1/11 (9)	1/23 (4)
Rheumatoid arthritis	0	1/11 (9)	1/23 (4)
Uterine cancer	0	1/11 (9)	1/23 (4)
No past problems	1/12 (8)	0	1/23 (4)

Discussion

Patients with acute hyperventilation are well known to all emergency departments, but to our knowledge, this study is probably the first to attempt to study these difficult patients at and soon after the time of initial presentation. In common with most studies that rely on busy emergency department staff for patient selection, the selection criteria could be criticized in that arterial blood gas data were available only in about one fourth of the patients referred, and this blood was sometimes not taken until some time after the initial presentation to the hospital, almost certainly the reason for the 1 value of $PaCO_2$ above 30 mm Hg. The blood gas data confirmed the clinical assessment of extreme hypocapnia in the patients in whom these measurements were made and helped to validate the clinical assessment of hyperventilation in the remainder of the patients in whom there was a high incidence of presenting complaints consistent with severe hypocapnia. Hyperventilation was clearly the predominant and most dramatic symptom and, had the study not been in progress, these patients would probably have been labeled as "hysterical" and dismissed, labeled as hav-

Table 5—*Respiratory Symptoms, Psychiatric Evaluation, and Lung Function Data for Each Individual Subject***

Age, yr/ Sex	Respiratory History	FEV_1, %	VC, %	PEF, %	Histamine Challenge	TCO, %	KCO, %	Chest Radiograph	Hyperventilation Screen, mm Hg	Psychiatric Evaluation Results
25/M	Night cough, sputum, wheeze	78/78	85/89	97/100		96	111	n	Normal	Normal
23/M	Nil	98/105	102/104	90/97		128	133	n	Normal	Panic
49/M	Nil	114/114	127/122	88/91		146	119	n	29 post-VHV	Panic
36/M	Childhood asthma	109/112	107/107	83/85		108	113	HI	Normal	Normal
30/M	Childhood asthma	107/107	98/100	99/100		103	106	HI	Normal	Normal
28/M	Chest tightness, bronchitis	96/102	95/95	92/102	Normal	125	128	n	Normal	Hypochondriasis
37/M	Nil	92/92	82/87	88/93	Normal	127	165	HI	Normal	Anxiety disorder with panic
26/M	Known asthmatic	65/88	81/85	60/80		105	121		27 rest, post-ex, 26 post-VHV	Normal
32/M	Hayfever	97	98	86	Positive	134	150		21 at rest	Anxiety disorder with panic
22/M	Occasional chest tightness	107/109	102/100	93/94	Normal	135	132	n	Normal	Anxiety disorder
35/M	Chronic chestiness	125/128	127/127	107/109	Normal	116	112	n	30 post-ex	Anixety disorder, depression
30/M	Chestiness, childhood asthma	92/97	87/85	100/106	Normal	118	133	n	30 rest and post-ex	Anxiety disorder with panic
51/F	Nil	123/136	121/118	121/128		165	153	n	Normal	Anxiety disorder, depression
28/F	Known asthmatic	103/109	110/120	100/99		101	89	n	29 rest and post-ex, 20 post-VHV	Anxiety disorder
25/F	Nil	106/110	106/109	99/111		108	111	n	Normal	Panic
18/F	Known asthmatic	104/104	103/97	93/99		138	110	n	25 rest, 28 ex, 26 post-VHV	Normal
31/F	Family history asthma	100	103	111	Positive	84	89	n	30 rest, 29 post-VHV	Anxiety disorder, depression
24/F	Known asthmatic	100/104	111/111	108/108		138	163	n	Normal	Anxiety disorder with panic
57/F	Wheeze and cough	88/92	89/91	74/81		74	82	n	27 rest	Anxiety disorder
36/F	Known asthmatic	89/95	94/102	93/99	Positive			HI	21 rest	Anxiety disorder
42/F	Known asthmatic	77/85	84/87	92/105		84	94	n	22 rest	Anxiety disorder, agoraphobia
57/F	Nil	114/119	89/96	118/122		104	112		29 post-VHV	Normal
61/F	Known asthmatic	79/83	80/84	77/79				n	26 rest	Anxiety disorder

*M=male; F=female; VC=vital capacity; PEF=peak expiratory flow; TCO and KCO=single breath carbon monoxide transfer factors, without and with correction for lung volume; n=normal; HI=hyperinflation; ex=exercise. All are expressed as percent of predicted normal, and FEV_1, VC, and PF are shown with values before and after inhaled albuterol.

ing anxiety states with eventual referral to a psychiatrist, or misdiagnosed as having an unknown cardiac disorder with inappropriate admission to a coronary or other high dependency unit. It is the appropriateness of these outcomes that this article addresses. The subsequent analysis of our patients supported the emergency department physicians' assessment that there was no clinically obvious organic cause for the hyperventilation other than the factors discussed below. We were gratified that perusal of the records showed that virtually all patients presenting to the emergency department during the year in which data had been collected had been referred.

The main findings of this study were the extremely

high prevalence of certain and probable asthma in these patients, the equally high prevalence of anxiety as detected by CIS-R scoring, the moderately high incidence of drug and alcohol abuse associated with the initial presentation, the almost universal misattribution of the presenting attack to a range of serious diseases, and the relatively low incidence of reported panic as a presenting symptom. Most attacks had occurred previously but only the patients with asthma had any evidence of hypocapnia some weeks after the attack.

As discussed in a recent review by Gardner,[11] hyperventilation implies excessive drive to breathe, which can have a wide range of physiologic, organic, and psychogenic causes. Nevertheless, the high incidence of asthma in our patients was surprising. To diagnose asthma, we chose to use a combination of history, lung function assessment, and histamine challenge. We believed that any one of these tests alone would not give a true picture, and histamine challenge may be negative in known asthmatics. Asthma, especially when mild, is a potent cause of hypocapnia with reduction of $PaCO_2$ to below 25 mm Hg in association with only mild or moderate reduction of FEV_1.[5,12,18,19] Results of standard lung function tests are often normal or are difficult to interpret due to anxiety, panic, or dyspnea. In the present study, while the initial clinical assessment and the relatively high PaO_2 values in the patients in whom blood gas samples were taken does not suggest the presence of severe asthma at the time of presentation, the persistently low P_{CO_2} in our asthmatic patients suggests that asthma made a significant contribution to the initiating attack, probably by long-term lowering the resting $PaCO_2$ closer to the threshold for symptom production. We have previously found that in normal subjects, symptoms of hypocapnia occur at a mean $P_{ET}CO_2$ of 20 mm Hg with an outside range of 14 to 29 mm Hg[20] and with no relation to the rate of fall of $P_{ET}CO_2$. It is not difficult for a number of factors to conspire to lower the P_{CO_2} below this symptom threshold.

There was no clear association between mild anxiety and a low $P_{ET}CO_2$ and the role of anxiety in the initiation of the attack is less clear. Anxiety was only usually detectable by careful assessment and very few patients had a previous psychiatric history. Nevertheless, it would not be unreasonable to suggest that anxiety contributed to the initiating attack, but not to the extent that is commonly supposed or as was implied in the initial descriptions of hyperventilation syndrome.[1,4] Panic disorder consists of a broad range of symptoms, including dyspnea, extreme fear, tachycardia, and palpitations. The mechanisms of panic are uncertain and there is a complex relationship among anxiety, hyperventilation, and panic.[21] In our patients, only 30% reported panic as a symptom during the attack, suggesting that acute hyperventilation is a separate disorder from panic disorder and that panic was not the major component of the presentation of our patients.

One theory about the etiology of panic suggests that it may be due to the cognitive misinterpretation or misattribution of somatic symptoms to a life-threatening illness such as a myocardial infarction or a stroke.[22] The role of misattribution in the etiology of hyperventilation in the absence of panic is less clear, but this was a constant feature of many of the original descriptions of hyperventilation syndrome.[2] In our study, 87% of patients misattributed their attack to serious disease. This was not surprising in that hypocapnia can induce a range of alarming symptoms,[11] including paresthesia and tetany, altered consciousness and cold extremities due to reduction in cerebral and peripheral blood flow, respectively, chest pain, and even unilateral weakness. Most patients (and many physicians) are unfamiliar with the symptoms of hypocapnia. In our patients, this misattribution, especially to heart disease, almost certainly led to a vicious circle of increasing anxiety and hyperventilation, and was the main reason for the patients' presentation to the emergency department.

Twenty-six percent of these patients reported a history of alcohol and marihuana abuse, and 17% believed that abuse of these substances contributed to the presenting attack. A variety of drugs, including caffeine, alcohol, nicotine, cannabis, and cocaine have been described as associated with panic and hyperventilation,[23] and dependent alcohol consumption is associated with increased trait anxiety and hyperventilation symptoms.[24]

Although most of our patients had previously presented with acute hyperventilation attacks, in general, only the patients with evidence of asthma had a positive hyperventilation screening test when assessed some weeks after the index episode. This suggests that in the remainder, the acute attacks did not occur on the basis of preexisting subacute or chronic hyperventilation and that there were no other chronic causes of hypocapnia. In that female subjects have a lower P_{CO_2} than male subjects, especially in the second half of the menstrual cycle, it is of interest that the gender division of our patients was roughly equal. We might have expected a female preponderance.

In summary, this study suggests that not one factor but a number of factors and especially mild asthma, chronic anxiety, and drug/alcohol abuse combine to lower the $PaCO_2$ in these patients to the point at which symptoms of hypocapnia occur. Misattribution then leads to a vicious circle of increasing anxiety and panic which results in presentation to the emergency department. In the context described in this article, not only is the term hyperventilation syndrome of dubious value scientifically, but its use can be dangerous in that

it distracts from seeking the true causes of the increased respiratory drive.

These patients should be referred initially to a physician although specialist psychiatric assessment may ultimately also be required in a percentage. Careful assessment for mild asthma, advice about drug and alcohol abuse, explanation about the possible presenting sequence of events and the symptoms of hypocapnia, and full organic investigation to reassure about misattributed disorders provide the best therapeutic approach. In the emergency department organic respiratory causes of hyperventilation can best be detected by insisting that arterial blood gas analysis and a chest radiograph be performed in all cases of hyperventilation. Even with a normal chest radiograph, a psychogenic etiology should not be considered as the sole diagnosis unless the PaO_2 is raised proportionately to the reduction in $PaCO_2$.

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