

HYPERVENTILATION: THE TIP AND THE ICEBERG

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OVER THE PAST decade, some 700 cases of the hyperventilation syndrome have been studied in the respiratory physiology department of Papworth Hospital. 82 per cent had no detectable pathology. The remainder had well documented pathological states, but their symptoms were largely or entirely related to overbreathing. This survey does not include a very considerable number of patients in whom hyperventilation was present, but who also had organic disease of sufficient severity to produce symptoms; e.g. cardiac infarction.

We have paid particular attention to the cardiological aspects of this syndrome, which shows up in medical clinics under many other guises; largely because these cases have been through the double sieve of cardiological and respiratory physiological investigation.

Under the heading of "hyperventilation syndrome" standard English texts mention only the classic triad of massive overbreathing, paresthesiae and tetany: a syndrome well known to any recent graduate. But spontaneous tetany is one of the rarest manifestations of hyperventilation and in my experience occurs in about one per cent of cases. This is merely the tip of the iceberg; the body of the iceberg, the ninety nine per cent who do not present in this fashion (and are not accorded the dignity of a mention in any standard English text), presents a collection of bizarre and often apparently unrelated symptoms, which may affect any part of the body, and any organ or any system. The many organs involved are often reflected in the number of specialists to whom the patient gets referred, and my colleagues have variously dubbed this the "multiple doctor" or the "fat folder syndrome". Indeed the thickness of the case file is often an important diagnostic clue.

Table 1 lists the main symptoms observed by a general physician in a series of 270 cases. Symptoms may show up anywhere, in any organ, in any system; for we are dealing with a profound biochemical disturbance, which is as real as hypoglycemia, and more far-reaching in its effects. Such patients are often pursued relentlessly With every investigative device known to modern science, and end up with the label of "anxiety state" and the implication that they are inadequate or in some way inferior. They may be advised: "pull yourself together, it's only your nerves" or possibly a more sympathetic surgeon may be persuaded to tinker with or remove the complaining organ--an organ, which, I may say, is merely protesting against an unbalanced diet deficient in carbon dioxide, bicarbonate, oxygen, and calcium ions: to name but a few of the well-known biochemical disturbances which accompany acute hypocapnia.

TABLE 1.

HYPERVENTILATION

CARDIOVASCULAR:

palpitations, tachycardia, precordial pain Raynaud's phenomenon

NEUROLOGICAL:

Central: dizziness, disturbance of consciousness/vision Peripheral: paresthesiae, tetany (rare)

RESPIRATORY:

shortness of breath. "asthma" chest pain

GASTROINTESTINAL:

globus, dysphagia, epigastric pain aerophagy

MUSCULOSKELETAL:

muscle pains, tremors, tetany

PSYCHIC:

tension, anxiety

GENERAL:

fatigability, weakness, exhaustion, sleep disturbance nightmares

TABLE 2

NON-ORGANIC DISEASE IN MEDICAL OUTPATIENTS PRESENTING SYMPTOMS

GASTROINTESTINAL:

Abdominal distension, constipation, diarrhea, vomiting, burping [44]

C.N.S:

Twitching eyelids, headache, giddiness, fainting, diplopia [21]

RESPIRATORY:

Difficulty in breathing, cough [19]

CARDIOVASCULAR:

Precordial discomfort, palpitating missed beats [15]

SKELETAL SYSTEM:

Weak limbs, painful limbs, vague pains [6]

GENERAL SYMPTOMS ONLY:

Weakness, irritability, insomnia [10]

MISCELLANEOUS: [13]

{Gottlieb 1969}

Some forty years ago Kerr, Dalton and Gliebe wrote [1] "Patients presenting the well known pattern of symptoms haunt the offices of physicians and specialists in every field of medical practice. They are often shunted from one physician to another, and the sins of commission inflicted upon them fill many black pages in our book of achievement."

Unfortunately I believe this to be still true today, despite the many and excellent reviews which have appeared in the intervening years [2-5]. Among the sins of commission are fruitless operations on the abdomen, the spine, and other organs-- invasive investigations which are not without risk--and, even worse, damaging diagnoses like epilepsy and cardiac infarction which may create chronic iatrogenic invalidism.

The high incidence of these cases in various clinics has been reported many times, varying from six per cent in specialist clinics such as gastro-enterology [6] and cardiology [7] and in otherwise healthy wives of service personnel [8], to ten per cent in the office of a general physician [9]. Burns and Howell [10], in a study of disproportionate dyspnea, found hyperventilation in ten per cent of controls.

Gottlieb [11] in 1969 reported that forty per cent of patients presenting at his London medical outpatients had no detectable organic disease. His table of symptoms (Table 2) is almost identical with the symptoms of hyperventilation, but he failed to mention this possibility at all. He called them all "anxiety states". This I believe to be the commonest diagnosis under which hyperventilation is mislabeled, and it is especially damaging. It implies inadequacy or constitutional inferiority; it tempts the doctor to dismiss the very real and often painful symptoms of a biochemical disturbance as imaginary or due to a low pain threshold, and it swells the tide of tranquillizer consumption which threatens to inundate the health service.

Figure 1, which reflects our experience at Papworth, illustrates graphically what happens when physicians and surgeons become alerted to the wide ranging manifestations of hyperventilation.

The cases shown on this histogram were all those in which the patient's typical symptoms could be reproduced by voluntary hyperventilation, and in which appropriate tests showed either the absence of significant disease or, if some disease was present, it was not sufficient to cause the symptoms. The numbers seen in each year are indicated above the open columns, and the cross-hatching indicates those in whom the diagnosis was suggested by the referring physician. Until the early part of 1968, only two cases had the diagnosis suggested before referral, and these were cases presenting with tetany. You will note that the crucial year was 1968, which was the year in which the furor about the Pill and thromboembolism was at its height. Cardiologists and thoracic surgeons were bombarded with young ladies suffering from syncopal attacks or other possible manifestations of embolism. These were subjected to the most sophisticated investigations available to modern technology: lung scans, cardiac catheters, pulmonary angiography, and even in one young man, who had severe precordial pain, coronary angiography. Investigations were negative.

In a period of one month, five such patients were found in various departments of the hospital. In each case investigations were negative. In each case symptoms could be fully reproduced by voluntary hyperventilation. These cases excited a heightened awareness of the syndrome, with the results seen in the figures for subsequent years.

The diagnosis, as I have said, rests on reproducing the patient's symptoms by voluntary hyperventilation in a form which the patient recognizes, and on taking reasonable steps to exclude significant pathology. Voluntary overbreathing usually accomplishes reduplication of symptoms within a few minutes; often within thirty seconds, with the exception of skeletal and particularly precordial pain. Persistent overbreathing for up to twenty or thirty minutes will usually bring on pain, but I do not now attempt to push patients with anginal type pain to this extent because I believe it may be dangerous. I will return to this point.

A brief review of the physiology of hyperventilation and hypocapnoe is apposite at this point. Overbreathing can rapidly blow off enormous amounts of carbon dioxide, and produce a precipitous fall in PCO₂ levels i.e. respiratory alkalosis. Bicarbonate and carbonic acid are, quantitatively, the most important buffer system in the extracellular fluid. The body seems to care deeply about its pH and has three major systems to maintain it: 1. Buffers: bicarbonate/carbon dioxide. (Hemoglobin and protein buffers). 2. Renal regulation of H⁺ excretion. 3. Respiratory regulation of carbon dioxide excretion. Our first concern is with the interlocking relationship between pH, carbon dioxide, and bicarbonate. (Fig. 2).

If you offload carbon dioxide (which can be done in vast quantities and in a short time by overbreathing), you are left with an excess of bicarbonate ion and a deficiency of hydrogen ion. Bicarbonate excess is compensated by renal excretion and tissue storage. The important point is: hypocapnia and pH shift are almost immediate; adjustment of bicarbonate, like Eartha Kitt's Englishman, takes time.

First consider the immediate effects of hypocapnia. The most striking direct effect is on the cerebral circulation. Carbon dioxide is the most important regulator of cerebral vascular tone. Hypocapnia causes immediate vasoconstriction leading to cerebral hypoxia.

Hypoxic effects are potentiated by the effect of alkalosis on the hemoglobin dissociation curve, which is shifted to the left. (Bohr effect). The effect of the shift with hypocapnia is both to decrease the amount of oxygen available from hemoglobin, and to slow down its release. Hence the net result on the brain is less blood, delivering less oxygen, and delivering it more slowly. These effects are immediate, hence the most frequent, as well

as the most rapidly produced manifestations of hypocapnia are: faintness, dizziness, and visual disturbance, which may vary from simple clouding of vision to tunnel vision, total loss of vision or flashing lights like fortification spectra. Blackouts are common, and particularly dangerous if you also have aortic stenosis, for syncope is then a prime indication for open-heart surgery. One such case had been given an aortic homo-graft (elsewhere). This gave him a normal circulation, but left him with his blackouts which continued with unabated severity until abolished by our physiotherapists. Transient disturbances of consciousness run the risk of confusion with petit mal. Grand mal may be triggered by hyperventilation.

Returning to the ionic shifts, which attempt to restore hydrogen ion balance, we have a complex series of events, of which I mention only the most significant.

1. Offloading bicarbonate via the kidney. Thinking back to the pair of scales (Fig.2): pH is returned to normal by lowering the bicarbonate level of the plasma. Thus the hallmark of chronic hypocapnia is a low plasma bicarbonate. This was noted as far back as 1916 by Sir Thomas Lewis. He found deficient bicarbonate buffer as a constant feature of the effort syndrome: Other features are: suppression of hydrochloric acid formation by the stomach, retention of H⁺ by the kidney, retention of phosphate and chloride, lactic acid production.

2. Reduction of available calcium ion which has been thought to play some part in the production of tetany, although this is uncertain. Tetany is probably caused by the direct action of alkalosis on peripheral nerves. Tetany does not imply the full blown main d'accoucheur; it may be manifest as muscle hyperirritability, or painful local spasm of viscera as well as skeletal muscle.

Hyperventilation has often been labeled--stigmatized is perhaps a better term--as anxiety state. I would emphatically disagree with this. Anxiety, in my experience, has usually been the product, not the prime cause. Emotional upset has been the most frequent trigger which has set off the chain of symptoms; the anxiety state seems to have most frequently been engendered by doctors who have failed to recognize the profound biochemical disturbance just outlined. Unfortunately when his many investigations prove negative the patient is left with the belief that he is suffering from something which is beyond modern medical science, or he may begin to question his own sanity. Are these not sufficient grounds for chronic anxiety?

It has always seemed to me that hyperventilation is essentially a bad habit; a habit of breathing in such a way that the day to day level of P_{CO2} is relatively low. Given this basic bad habit, any physical or emotional disturbance may trigger off a chain reaction of increased ventilation, rapidly producing hypocapnic symptoms, alarm engendered by the symptoms, consequent sympathetic arousal resulting in increased ventilation and increased symptoms. (Fig. 3).

The type of breathing adopted by hyperventilators is quite characteristic and best illustrated by a cine film:

[a film was shown of the typical breathing]

You will remark the effortless heaving of the upper sternum and the lack of lateral costal expansion. The movement is the same as that employed in sighing, and indeed frequent sighs are a hallmark of hyperventilation. This type of breathing becomes habitual and the patient is usually unconscious of his sighs, although friends and family often comment.

This is obviously the breathing of stress or emotion. You find references to it everywhere (except in the case notes). The "heaving bosom" of the Victorian heroine (who also had the habit of frequent swooning, and attacks of the vapors). "Love", in the words of W. S. Gilbert [12], "nightmare like, lies heavy on my chest".

May I commend this breathing habit as an important physical sign. It turns bosom watching from a furtive masculine diversion into a positive scientific study--particularly in

tube trains, for hyperventilators are frequently claustrophobic. But lest you think it is a disease of weak women, please note the sex incidence: it is almost exactly 50-50 in my series (Fig. 4). The difference is in age incidence, reflecting the times of maximum emotional stress. The incidence is higher in the younger woman, but a steadily mounting incidence in middle-aged men. The factors commonly are Romance or Finance. Nor are these the weaklings. The most prone are the meticulous, hard-working perfectionists, prone to make excessive demands on themselves.

Hyperventilators frequently present with symptoms suggestive of heart disease and indeed well marked physiological changes in heart action are produced. T wave changes resembling ischaemia are well documented and may be taken as confirmatory evidence when the pains resemble cardiac infarction (Fig. 5). Alarming dysrhythmias may occur and for this reason I do not now push too hard in attempting to reproduce precordial pain, (Fig. 6) since I believe that it could be dangerous. Time does not allow a discussion of the details of treatment, beyond saying that we try to make patients aware of their disordered breathing habits and convert to a slow diaphragmatic type of breathing. In the older patients this often requires much patient work by the physiotherapist. No patient under 25 has failed to be completely cured. Two thirds of all patients so treated are completely relieved of symptoms. It is very uncommon to find a patient who does not experience marked improvement. To recapitulate:

1. We each probably see at least one of these patients a week.
2. The type of breathing is utterly characteristic, virtually diagnostic, and seldom recorded in the notes.
3. The size of the case folder is a frequent pointer to the diagnosis.

REFERENCES

1. Kerr W. J., DALTON, J. W. and GLIEBE P. A. Some physical phenomena associated with the anxiety states and their relation to hyperventilation. *Ann. Int. Med.* 11, 962-992 (1937)
2. SLEY M. H. and Shock N. W. The etiology of effort syndrome. *Am J. Med.* 196, 840 (1938).
3. ENGEL G. L., FERRIS E. B. and LOGAN. Hyperventilation: Analysis of clinical symptomatology. *Annals Int. Med.* 27, 683 (1947).
4. LEWIS B. I. Hyperventilation Syndrome. A clinical and physiological evaluation. *California Med.* 91, 121 (1959).
5. TUCKER W. I. Hyperventilation in differential diagnosis. *M. Clin. N. Am.* 47, 491 (1963).
6. MCKELL T. E. and SULLIVAN A. J. The hyperventilation syndrome in gastroenterology. *Gastroenterology.* 9, 6-16 (1947).
7. Yu P. N., YIM B. J. and STANFIELD C. A. Hyperventilation Syndrome. Changes in the electrocardiogram, blood gases and electrolytes during voluntary hyperventilation. *AMA Archive. Int. Med.* 103 (Jan.), 70/902 (1959).
8. SINGER E. P. The hyperventilation syndrome in clinical medicine. *New York State J. Med.* 1 st May, 1494 (1958).
9. RICE R. L. Symptom patterns of the hyperventilation syndrome. *Am. J. Med.* 8, 691 (1950).
10. BURNS B. H. and HOWELL J. B. Disproportionately severe breathlessness in chronic bronchitis. *Quarterly Med. J.* 38 (151), 277 (1969).
11. GOTTLIEB. Non-organic disease in medical out-patients. *Medical Update* 1, 917-922 (1969).
12. GILBERT W. S, and SULLIVAN A. *Iolanthe: Chancellor's Song.*