Institute for
the Awakened Mind

Controlling the Breath to Counteract the Diverse and Problematic Effects of Hyperventilation

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Definition and Symptoms * Misdiagnoses * Anxiety, Stress and Life Changes * Hypocarbia * Exercise

Hyperventilation, or over-breathing, is a very common phenomenon, but its range and the variety of physiological effects are rarely appreciated; all kinds of physical and mental disorders can be initiated by simply over-breathing.

Medical awareness generally stops short at the 'hysterical fit', with extreme over-breathing, tetanus and sometimes coma – which may cause the whole syndrome to be mistaken for epilepsy. Tetanus is one of the least common manifestations in which few doctors recognize the more subtle, but infinitely more common syndromes of chronic habitual hyperventilation.

These may show symptoms relating to virtually any organ or system of the body – suggesting, for example, thyroid, cardiac, gastrointestinal, respiratory or central nervous system disease. In fact, hyperventilation can fairly claim to have replaced syphilis as the great mimic. The archetypal syndrome is Da Costa's, which currently enjoys a revival as 'mitral valve prolapse syndrome ' – the latter in a long line of attempts to attribute a common physiological dysfunction – over-breathing – to an anatomical variant which is rarely significant.

Patients, who are generally regarded as neurotic, may present with symptoms suggesting dysfunction or disease of any organ or any part of the body. 'Cardiac' manifestations are particularly common; as are neurological disturbances, dizziness, faintness, or blackouts, a great variety of visual disturbances and paraethesiae affecting many parts of the body, but particularly the hands, feet and face.

Respiratory complaints are surprisingly less common, shortness of breath tending to be overshadowed by general fatigue; but irritable unproductive cough is frequent, as are gastrointestinal symptoms, dysphagic, globus, burping, oesophageal reflux and heartburn. Muscular cramps, 'fibrosistis' of the neck, shoulders and back are very often troublesome, but tetanus itself is very rare.

Most disturbing of all are the psychic disturbances, strange sensations and ideas, ranging through tension and free-floating anxiety to 'unreal' feelings, depersonalization and even hallucinations. The latter two symptoms are seldom admitted, for there is very often an unspoken fear of madness. General exhaustion, lack of concentration and diminished performance are almost universal, while sleep disturbances, nightmares and night-terrors, and emotional sweating of the armpits and palms of the hands are very often to be found. And all of this is simply the result of taking in too much air.

Several factors foster the neglect of hyperventilation as a positive diagnosis, an example being the absence of any conspicuous overbreathing. Shortness of breath is very seldom the primary complaint, but most important is the too ready acceptance of the blanket diagnosis 'neurosis' or 'anxiety state' to cover the doctor's inability to explain multiple symptoms without overt pathology providing an adequate cause.

Modern neurophysiology, and latterly the use of microelectrodes, can now supply biofeedback answers to most of the problems which have previously hindered appreciation of the true role of hyperventilation in anxiety states. The time has therefore come to attempt to answer the important question "Does anxiety cause the symptoms, or do the symptoms cause the anxiety?

Hyperventilation acts by blowing off excessive quantities of carbon dioxide. It might seem logical to expect that in chronic hyperventilators, one finds an abnormally low level of carbon dioxide in the arterial blood – hypocarbia, as it is called. Values below normal are in fact found in about two-thirds of cases, but in more than one-third, that is to say in a substantial minority, the level is either equivocal or in the low-normal range.

The diagnosis can, however, be proved by a simple test: forced voluntary overbreathing will almost always reproduce symptoms which the patient can recognize within 2-3 minutes. This manoeuvre, besides its diagnostic value, underlines the fact that it is the change in arterial CO_2 rather than the prevailing level which is important.

The symptomatic response is evoked just as readily when the baseline arterial CO_2 is normal as when it is low. So try this for yourself; just try the effect of deliberately overbreathing for 2-3 minutes – what effects does this produce in you? Are they effects which you very readily recognize, or are they strange effects, ones which you don't really recognize at all?

A low value of carbon dioxide in itself does not necessarily cause symptoms. The body can learn to adapt itself quite readily without symptoms to very low levels of arterial CO_2 as in high altitude adaptation. At 14,000 feet the average arterial CO_2 in normal subjects is 27mm HG, as against 40mm Hg at sea level, which is a pretty big change. But it is, however, a long-term adaptation; full acclimatization takes days or even weeks.

Over the last thirty years neurophysiological research has demonstrated the all-pervasive influence of changes in carbon dioxide level on the function of the nervous system. The infra-red analyser, or skin-conductance measurement at the palms of the hands, now provides a ready means of monitoring variations in arterial CO_2.
In the normal person, during quiet breathing continuous monitoring of end-tidal arterial CO_2, shows only minor fluctuations from the average value. A single, deep breath, may lower this by 10mm Hg or more and this may take 20-30 seconds to recover. Voluntary overbreathing for three minutes produces a sharp fall to a low value which is maintained during the overbreathing period, but recovers to normal within a few minutes.

The behavior of hyperventilators is markedly different. At rest, considerable fluctuations in end-tidal CO_2 occur, mirroring the fluctuations in tidal volumes; a marked fall occurs with each sigh and recovery takes longer than in the normal. Voluntary overbreathing produces a sharp fall, maintained during the overbreathing period, but on stopping overbreathing the CO_2 tends to remain low for many minutes; once started, they find it difficult to stop overbreathing.

The symptoms attributed variously to anxiety states or to hyperventilation, according to the fancy of the doctor, embrace a wide variety of bodily symptoms, organ dysfunction, particularly cardiac or gastrointestinal, anxiety state and disturbance of mental function. The common pathway leading to this plethora of symptoms is the nervous system, which, as mentioned above, is very susceptible to changes in the CO_2 level in the blood.

Synaptic transmission is accelerated and recovery times are shortened, in fact there is a general speeding up of automatic processes. In the autonomic system there is a selective depression of parasympathetic activity – the sympathetic system is also depressed, but to a lesser degree. Hence the subject tends to show a picture of sympathetic dominance: there are dilated pupils of the eyes, cold extremities, palmar and axillary sweating, and tachycardia. With even less carbon dioxide in the blood, these disappear in a similar fashion to the extinction of the stimulation response when hypocarbia proceeds to severe.

So moderate degrees of hyperventilation – classically in the ‘fight or flight’ situation – cause increased motor excitability and also increased sensitivity to sensory stimuli: lights appear to be brighter, sounds seem louder; photophobia and hyperacusis (that is fear of bright lights and loud sounds) are common.

The survival value of this response is obvious; heightened sensory perception, the muscular system tense and alert, the reflexes quickened. In severe grades of hypocarbia many of these responses become extinguished – therefore we have the phenomenon of paralysis by fright. Moreover, carbon dioxide controls the caliber of cerebral arteries. Hypocarbia causes vasoconstriction and hence cuts down the oxygen supply to the brain. Many of these clinical effects stem from too little oxygen in the brain supply.

The foregoing provides only a brief account of some of the extremely diverse effects of the breathing out of excessive amounts of carbon dioxide. There is great individual variation in response, particularly in the cerebral vascular reaction. Dizziness and faintness are almost universal in the young but much less so in the elderly. Some tolerate levels of CO_2 which would produce fainting in others.

Dizziness, faintness, visual disturbance and impaired mental performance are all closely relaxed to too little oxygen in the brain supply. The compounding of sensory and motor disturbance with too little oxygen in the brain supply has been aptly termed “a welter of most unpleasant bodily sensations’, and it is the cause of curious mental states. Depersonalization is frequent; occasionally hallucinations are experienced, but these are seldom divulged spontaneously lest they should confirm the subject’s frequent fear of madness.
A few other clinical effects of hyperventilation should be mentioned. Firstly it markedly potentiates the effects of alcohol, producing extraordinary symptoms. Hyperventilation is very common in aircraft. The passengers who get quite out of control on a couple of whiskies are a problem familiar to air hostesses. Hyperventilating motorists should therefore be mindful of their condition, and double careful not to drink and drive.

Although most forms of perception are heightened, there is one important exception – pain. The sensation which we recognize as pain is dulled or abolished. This is exemplified in tribal initiation or voodoo rites during ritual mutilation ceremonies. The excitement, singing, dancing, and beating of drums induce a crescendo of hyperventilation, during which initiates are enabled stoically to endure otherwise extremely painful procedures. And finally, of course, hyperventilation powerfully affects the induction of hypnosis. Most standard procedures for hypnotic induction employ deliberately induced hyperventilation. In the light of the known neurological effects of too little oxygen in the brain, many of the bizarre manifestations of hypnosis can be readily understood.

It remains to consider why certain individuals become prone to hyperventilate, which is a perfectly normal reaction under normal circumstances, to a degree which causes recurrent and persistent symptoms. Neurological examination can now leave little doubt that habitually unstable breathing is the prime cause of the symptoms. Why they breathe in this way must be a matter of speculation, but manifestly the salient characteristics are pure habit – excessive sighing, sniffing, nervous coughing; and such habits are very often family characteristics.

Exaggerated thoracic breathing is encouraged by cultural influences such as physical education and sexual display, such as the chesty he-man and the bosomy lady. Singers, actors, and athletes are trained to make maximal inspiratory efforts with the thorax as well as with the diaphragm – hyperventilation is an occupational hazard with opera singers, as well as with others.

The vast majority of clinical cases tend to be perfectionist or mildly obsessional. The Type A personality (Friedman & Rosenman, 1959) is particularly prone in men. These are people who tend to drive themselves too hard in setting themselves goals at the very limit of their reach.

It is often assumed that anxiety is the chief or the only cause of hyperventilation. On the contrary, any change of mood – happiness, laughter, relief, animated conversation, and even watching television can frequently be the cause. The first attacks commonly follow a purely physical illness. General anaesthesia and operations are potent triggers. The driving personality, addicted to his work, often develops the first attack at weekends or on holidays. Anxiety then develops out of the persistent symptoms, and with repetition, the response takes on the character of a conditioned reflex.

Although it was R. L. Rice (American Journal of Medicine, 8, 1950) who first pointed out that the anxiety was produced by the symptoms, and not the other way around, and that sufferers could be cured by curing their faulty breathing habits, it was B. I. Lewis (Biochemical Clinics, 4, 1964) who identified the role of anxiety as a trigger, rather than the prime cause. Given habitual hyperventilation, a variety of triggers, psychic or somatic, can initiate the vicious cycle of increased breathing and symptoms; anxiety arising from the symptoms exacerbating the over-breathing and thus generating more symptoms and more anxiety.

At Cambridge, more than 1000 patients have received a course of breathing retraining and relaxation in the physiotherapy department of Papworth Hospital. Symptoms are usually abolished within one to six months. Some young patients require only a few weeks while older or more severe cases may take many months: 75% are completely free of all symptoms at 12 months; 20% are left with occasional mild symptoms only, and these do no trouble them. Most of these become free from symptoms later, and they lose their anxiety.

Now I want you all to try and produce symptoms again. You should all find this quite easy now that you know more about it – but please do not try too hard. First of all, it will be necessary to attach the skin resistance and temperature meters. This time I want you to make written notes so that you can see quite definitely the effects upon yourself of breathing too much or too little.

Breathing Exercise

A – Awareness of Normal Breath
B – Deliberately overbreathe for 2-3 minutes. Note the changes of skin-resistance, and also the changes in temperature. Note the time taken (in seconds) for the meter to return to this starting position.
C – Deliberately under-breathe for 2-3 minutes. Again note the meter changes and the time taken for the meters to return.
D – Mantra Breathing. Using AUM (Aaa-h, Ooo, Mmm) draw out the sound for about 30 seconds. Keep this going for at least 5 minutes.
E – SO-HAN. Very quietly, very slowly, so as just to produce hypoventilation.