Hyperventilation describes respiration that is in excess of metabolic needs. Commonly it is rapid and arrhythmic with prominent upper chest movement and obvious sighing, but it can present as an almost imperceptible exaggeration of normal movement and rate. The essence of it is the production of inappropriately low levels of carbon dioxide, termed hypocarbia or hypocapnia, and measured directly by sampling arterial blood or indirectly from the partial pressure of carbon dioxide in the expired air. This paper describes the aspects of hyperventilation that are important from the therapist’s point of view in everyday practice, namely its presentation as a primary disorder, but not as a product of organic disease, altitude or hysterical overbreathing.

The syndrome we now recognize as hyperventilation was described in the medical literature by Da Costa in 1871 from his study of soldiers who had been made incapable of further effort on the battlefield by the physiological consequences of disordered breathing. Many good descriptions have come from studies made in wartime where the causes are relatively violent and the responses acute and dramatic as compared with the more protracted and less intense conditions of peacetime. Apart from Da Costa’s syndrome, many other names have been given to the syndrome, e.g. soldier’s heart, neurocirculatory asthenia and effort syndrome. Lewis is to be credited with the discovery of the metabolic basis, an inadequate supply of buffer salts in the blood, and the recognition that the loss of performance and disordered breathing patterns were not solely a soldier’s malady but one of the commonest afflictions of sedentary town dwellers. It was assumed that these town dwellers were neurotic until Lumm made it clear that the so-called neurosis was usually iatrogenic, the consequence of failure of recognition and treatment. Many cases today are given labels such as postviral syndrome or myalgic encephalomyelitis.

The clinical outcome of hyperventilation does not depend solely upon the degree of hypocapnia produced at a given moment because it is also affected by the factors of chronicity and arousal.

Pathophysiology

Chronicity

The transient hypocapnia of short-lived hyperventilation is buffered by the body and respiratory alkalosis does not occur. When the overbreathing is continued, the kidneys compensate for the respiratory alkalosis by excreting bicarbonate and reducing the body’s alkaline reserves. Thus the chronic hyperventilator’s pH regulation is finely balanced: diminished acid (the consequence of hyperventilation) is balanced against the low level of blood bicarbonate maintained by renal excretion. In this equilibrium small amounts of over-breathing induced by emotion can cause large falls of carbon dioxide (PCO₂) and, consequently, more severe symptoms. In other words, the chronic hyperventilator lives closer to the threshold for hypocapnia than the normal person. The emotional arousal is not necessarily fear or anxiety. It is commonly anger, and it may be laughter, pleasurable excitement or watching television.

At the same time, the hypocapnia causes increased adrenergic activity and decreased vagal activity, and the subject’s arousal level is increased. A vicious circle is set up because increases of arousal stimulate the breathing. Thus the chronic hyperventilator is abnormally vulnerable to changes of arousal level.

Arousal

Arousal level expresses the position on a continuum from torpor or sleepiness at the lower end to conditions such as rage, terror, revulsion and ecstasy at the higher. Kahneman defines it as the general activation or drive state of the mind which results from the person’s interaction with his environment: what he is doing, the effort he is investing, the stress to which he is exposed and the way it affects him.

Figure 1, a performance/arousal curve, illustrates that there are levels of arousal (anabolic) which are consistent with healthy function and higher levels (catabolic) that exhaust, generate illness or even cause a breakdown when the individual loses the ability to continue to struggle with the demands made by his environment.

Health depends upon the ability of individuals to adapt and cope in such a way as to maintain a

---

**Figure 1. Human function curve. A model illustrating the stations of health as a continuum from healthy function and fatigue (upslope) to exhaustion and ill-health (downslope). P=point at which little extra arousal can precipitate a breakdown of health. The dotted line indicates the patient’s intended level of activity and the solid line his actual level of performance, i.e. the general ability to cope with activities of daily living.**
stable and orderly internal milieu irrespective of what is happening to him in his life.

The level of arousal generated by meeting and sustaining the challenges of everyday life varies from individual to individual and from time to time. The amount that can be tolerated without ill effect depends upon inheritance, education in its broadest sense, life events, psychological and social influences, the weight, rate and threat of challenges already presented by the environment and the way the individual perceives them - his self-talk.

Some individuals are handicapped by low curves and others, with higher curves, can cope better and keep going longer, but no matter what the height of the individual's curve, stamina and endurance remain finite qualities.

Healthy function
At rest in healthy function on the upslope of the curve (Figure 1) breathing is an automatic unconscious process, predominantly diaphragmatic, inspiration being the active phase and expiration a passive recoil. Its pattern follows the level of arousal in order to achieve proper oxygenation for the effort in hand, and a proper regulation of Pco₂ upon which the orderly function of the autonomic nervous system depends. In the face of danger, distress, excitement or being trapped for a moment by inability to see a satisfactory course to follow, the breathing of a healthy person may shift from the basic mammalian diaphragmatic pattern to include prominent movement of the upper chest as part of the fight, flight or amatory response, thus increasing the adrenergic drive and activating the sympathetic nervous system as vigilance rises with the prospect of a challenge. Once the action has been decided or the challenge has passed the prominent movement of the upper chest subsides. These transient changes of breathing pattern in response to effort, challenge and threat, the promise of reward or sexual activity are a part of healthy human function. However, if this pattern of breathing causes the Pco₂ level to fall too low for optimum autonomic function the individual is handicapped by loss of endurance through inadequately governed heart rate and blood pressure responses, unwanted or inappropriate emotional arousal, and loss of potential for effective action. For this reason students of the martial arts are taught to be aware and in control of their breathing in order to maintain a proper condition of readiness for challenges.

Healthy fatigue
If the demands of daily life are high the individual becomes reasonably tired, but, as can be seen on the human function curve (Figure 1) performance can continue to increase with arousal. More effort is required to cope than at lower levels of arousal, and stimulants such as alcohol, coffee and cigarettes may be increasingly used to enhance or sustain performance. The breathing is more easily thrown into disorder as the limits of physiological tolerance are approached and so relatively little extra effort on exertion can cause breathlessness or other symptoms of hypcapnia such as giddiness. One individual may recognize fatigue and arrange to overcome it. Another might use hyperventilation to 'psyche' himself up, generating an adrenergic overdrive which carries him over the top into an exhausted state. The individual who uses hyperventilation to over-ride fatigue signals is vulnerable to exhaustion and illness in ways that are not open to non-hyperventilators.

Exhaustion
The human function curve is drawn with a peak to emphasize the watershed between legitimate fatigue and the exhaustion which sets up vicious circles from which the individual can neither recover spontaneously nor keep up the level of performance needed. The vicious circles of exhaustion promote hyperventilation and sleep deprivation, loss of insight and judgement, social and behavioural deterioration, disorganization of surveillance and self-defence, and high levels of anger and despair.

On the downslope of the curve (Figure 1), the individual naturally struggles to close the gap between what he can do and what he thinks is intended of him, but the effort is self-defeating: performance decreases and arousal-induced metabolic disorders increase. The emotional changes are particularly important. Struggle with anger or anxiety is linked with activation of the sympathetic-adrenomedullary system, and the feelings of despair or the prospect of defeat with stimulation of the pituitary-adrenocortical system. Hyperventilation is commonly adopted as a displacement activity when the emotions are bottled up, and heightens adrenergic drive.

The nature and intensity of these products of high arousal, their aggravation by disordered breathing, and their persistence through failure of habituation and adaptation⁴,¹⁸ shift the individual downwards and to the right, widen the gap, reduce performance and further degrade the organization of the internal milieu. Loss of sleep is one of the most important consequences as it accelerates and aggravates the transition from an anabolic mode of metabolism to a catabolic mode.

Ill-health
In this catabolic mode the internal self-regulating homeostatic mechanisms lose their ability to maintain a stable and orderly internal milieu and the individual begins to experience symptoms of ill-health associated with the hyperventilation syndrome. Unfortunately the individual can rarely see the downhill course and take effective action to escape because denial removes insight.

The earlier symptoms are unspecific and distributed among all the body's systems. They have been documented by Lum⁶. Among the diagnoses commonly presented to us are angina pectoris with or without angiographic abnormality, vasomotor instability, chest pain, syncope and tachyarrhythmia, hypoglycaemia, hiatus hernia, irritable bowel, ill-defined disorders of immunity, migraine, multiple sclerosis, neurological disorders with paraesthesiae, tinnitus and hyperacussis and musculoskeletal aches and pains, anxiety states, phobic disorders, panic attacks, asthma, myalgia and non-specific arthralgia associated with sleeplessness.

The major cardiovascular consequences of hyperventilation and exhaustion of the buffer reserves have been described (Figure 2).

Breakdown
The concept of myocardial infarction and sudden coronary death as a sudden failure of homeostasis, triggered by hyperventilation-induced coronary arterial
Hyperventilation → hypocapnia
Extracellular alkalosis → catecholamine release
† sympathetic activity
† vagal activity
† oxygen uptake (Bohr)
† potassium

Intracellular alkalosis → Increased Ca**
myocardium
† systolic contraction
† diastolic relaxation
† compliance

coronary vessels
Constriction and spasm
E.C.G.
Arrhythmia
ST, T abnormality
Thromboxane A₂
Vasoconstriction and thrombosis.

Figure 2. Some effects of hyperventilation-induced respiratory alkalosis on the cardiovascular system

spasm, with or without atherosclerotic disease, after a long period of failure of adapting and coping 22,23 provides for creation of strategies for anticipation and prevention that are not conceivable if the death is thought to be from accident or chance.

Diagnosis
The diagnosis of hyperventilation-linked illness first depends upon the doctor’s being aware of hyperventilation and on the lookout for it, especially among patients who complain of fatigue, unexplained illness and failure of coping (effort syndrome). Masses of clinical case notes revealing a multitude of fruitless investigations for specific disease may provide advance warning. The simple questionnaire prepared by Grossman and de Swart 24 is a useful pointer to the diagnosis. In the interview, it is important to pay attention not only to what the patient says, but also the way in which the verbal message is delivered: changes of breathing patterns may provide the most useful information about the way a patient perceives and copes with his or her environment 25. Short periods of hyperventilation might highlight topics with a high but unacknowledged emotional charge. A mindless repetition of questions and answers with increasingly disordered breathing may indicate that the patient is trapped by inability to cope with problems but too exhausted to assimilate the information and answers given by the doctor.

The disorders of breathing include frequent breaks in the rhythm such as heaving sighs or inspirations deeper than required for the physical effort of the conversation. In the middle or end of a sentence the air is released as an expiratory sigh that is not readily noticed unless the clinician is on the look-out for it.

The $P_{CO_2}$ of the expired air can be recorded by means of a capnograph 26 which analyses the air drawn continuously through a fine bore plastic tube from the nostril. The end-tidal level ($P_{CO_2}$) closely reflects the arterial partial pressure when the peak expiratory flow rate is normal. A reading of 30 mmHg or less confirms the suspicion of significant hypocapnia at rest. This is especially useful where hypocapnia is not produced by the usual arrhythmic upper chest breathing but an undramatic, small and persistent increase of rate and depth of a normal rhythm of respiration.

When the $P_{CO_2}$ is normal at rest a challenge is required to test the patient’s ability to remain below the threshold for frank hypocapnia and symptom production.

The Hardonk and Beumier forced hyperventilation provocation test 27 is in standard use today. The patient overbreathes deeply and quickly for 3 min. Normally the $P_{CO_2}$ returns to a level above two-thirds of the resting level within 3 min of the end of the test, whereas there is a delay in the return among hyperventilators. This test may make the patient highly sensitive to emotional stimuli, and so the second challenge is to record the $P_{CO_2}$ during the recall of symptoms and circumstances of emotionally charged events such as admission to a coronary care unit 28. A variation of this test is to use the challenge of recall during hypnosis 29. The third challenge is exercise 30: in the normal case the $P_{CO_2}$ rises during exercise, but in the hyperventilator the effort is brought to an end by hypocapnia. The symptoms may suggest angina pectoris and the ECG may show ischaemic-looking ST depression.

Another useful diagnostic trait of chronic hyperventilators is their insensitivity to hypocapnia: levels of $P_{CO_2}$ less than 50% of normal may be regarded as normal (data to be published).

Management
Recovery from hyperventilation-induced symptoms cannot be achieved by tackling the breathing disorder alone; the hyperarousal, the failure of performance and the other downslope disorders must also be considered.

On the downslope the patients’ denial and information input overloading make them incapable of assimilating the information and executing the changes needed for recovery. Struggling fruitlessly to change a habit as deeply rooted as hyperventilation is likely to increase the arousal and move the patients further down the human function curve. They need to be led from illness to health through the two traditional phases of intervention 24-26 31-33.

The first phase is the one in which the patients rest and sleep well enough to recover from the exhaustion of the mind and the degradation of the homeostatic systems. In the second, patients learn to understand their condition, to recover healthy function and to reorganize their life against relapse.

Homeostatic recovery
Except for the mild and transient overbreathing attacks that can be overcome by rebreathing from a paper bag, the most important step towards homeostatic recovery is the speedy reduction of arousal to a level where the patient can take control and reduce the rate and volume of respiration to a proper level. This reduction can be achieved by the judicious use of diazepam 2-5 mg chewed and swallowed with a drink of water, but only while the patient is acquiring the confidence and skill to take charge, relax, and overcome the breathing problem.

After this the patient must rest and sleep well. A few days off work might be required for the recovery of healthy function. Sleep and the reduction of vigilance foster the recovery of autonomic stability, the reduction of hyperventilation, the recovery of the body’s buffering systems and the essential switch from a catabolic to an anabolic mode of metabolism 19.

In heavier cases, those who have shifted from exhaustion to ill-health or close to breakdown, a much longer period away from duty may be needed. The patient is usually reluctant to accept what is required: resenting imprisonment in inactivity, feeling angry and frustrated because the escape into exhausting
displacement activity is now denied, and feeling guilty because he is not working to close the gap between what he can achieve and what he sees as intended of him. If the exhaustion is generated at work, a period of leave may allow recovery to take place, but withdrawal is much harder to organize when the exhausting influences are at home.

In severe cases, where the patient appears to be heading towards a catastrophic breakdown such as unstable angina or a recurrence of myocardial infarction, it may be necessary to admit him to hospital in spite of the risk that admissions to hospital in general and coronary care units in particular can increase arousal by promoting anxiety, anger, helplessness, depersonalization and sleep-loss and so push the patient further down towards breakdown instead of rescuing him from it. Nurses who understand these factors provide comfort and take care of the needs for security, confidence and dignity. Their skills reduce arousal and lead the patient through denial, rage, bargaining and despair into acceptance and readiness for the second phase of training.

The first phase ends when the patient is well rested and calm, and able to accept the predicament instead of denying it. A useful clinical sign of this is the patient being seen to waken and face the world without hyperventilating.

**Second phase**

By the end of the first phase many patients, perhaps the majority, have reached a vantage point from which they can look back, see the psychosocial origins of their illness and correct them spontaneously. It is suggested that this is because they have recovered the proper balance of function of the cerebral hemispheres instead of being handicapped by a disproportionate output from one side. The patients who do not recover spontaneously need time and help in coming to terms with their illness: without accepting their predicament they cannot learn how to go back to their environment and adapt and cope in a healthy way. They appear to be handicapped by alexithymia, that is to say they seem insensitive to the emotional effects of exhaustion and distress: they tend to be unaware of their breathing patterns, oblivious of fatigue, incapable of choosing a tolerable balance of rest and effort. Repeated failure keeps them highly aroused, incompetent and low in self-esteem. The occupational therapist is trained to deal with these handicaps and to spot what the patient requires for healthy function. The therapist acts as a trainer, helping the patient to become aware of strengths and weaknesses and to outwit the problems that previously he allowed to overload and undermine him. The rate of progression is usually governed by the patient's ability to recognize and outwit the temptations to recreate exhaustion and hyperventilation by doing too much too soon.

In high arousal states and severe chronic hyperventilation, extreme care must be taken with breathing and relaxation exercises because the body's buffering reserves are diminished: reducing the respiratory rate and volume may cause distressing acidosis, and reducing the production of CO₂ by relaxing the limbs can trigger severe hypocapnia.

**Conclusion**

Hyperventilation causes hypocapnia which induces autonomic instability and a wide variety of biochemical and metabolic changes. These, in turn, produce fatigue, inability to sustain effort, unspecfic illness and symptoms mimicking a host of clinical disorders. The outcome of hyperventilation is governed by the individual's arousal level and his loss of performance at adapting and coping, and it is not usually possible for recovery to take place without alleviation of these factors. Although the condition is extremely common the diagnosis is rarely made except in the case of hysterical overbreathers who comprise about 1% of the total. Cases presenting to hospital commonly go undiagnosed for years on end (Fat Folder syndrome) and may be subjected to extravagant and prolonged investigations for non-existent specific disease. A negative coronary arteriogram is commonplace in cardiovascular practice. Large numbers grow frustrated and drift away from conventional medicine. They probably make up the bulk of alternative practice.

Diagnostic technology is unavailable to most practising clinicians and the general index of suspicion is low. The purpose of this paper is to increase awareness of the condition, to highlight the need for close clinical observations in diagnosis and to emphasize the fact that management requires a biopsychosocial approach: palliative treatment is ineffective because it neither addresses the problems of adapting and coping nor the disability caused by the inappropriate breathing. The occupational therapist is trained in the biopsychosocial approach. Her job is to work with these patients and teach them how to outwit the psychological and social origins of the hyperventilation, and the chronic, relapsing disability that it causes.

**Acknowledgments:** I would like to thank Dr Peter Nixon for the opportunity to work in a teaching hospital department where the biopsychosocial approach is employed. I also thank the Rayne Foundation and Smith's charity for their support.

**References**

4. Lewis T. The soldier’s heart and effort syndrome. London: Shaw and Sons, 1918


31 Weir Mitchell S. The treatment by rest, seclusion etc. in relation to psychotherapy. J A M A 1986;255:2033-7


33 Richardson FM. Fighting spirit. London: Leo Cooper, 1978


35 Lane RD, Schwartz GE. Induction of lateralised sympathetic input to the heart by the CNS during emotional arousal: A possible neurophysiologic trigger of sudden cardiac death. Psychosom Med 1987;49: 274-84


41 Reed K. Models of practice in occupational therapy. Baltimore: Williams and Wilkins, 1984

(Accepted 28 March 1988)