

# Chronic hyperventilation and its treatment by physiotherapy: discussion paper<sup>1</sup>

**R A Cluff MCSP SRP**

*Papworth Hospital, Papworth Everard, Cambridge CB3 8RE*

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## Introduction

Hyperventilation (or overbreathing) is a perfectly normal reaction to stress, and a large proportion of people have at some time experienced some of its effects, e.g. the racing heart, weak knees and 'butterflies' in the stomach so often felt before examinations. But these symptoms normally disappear once the stressful situation is over.

Her Majesty the Queen described a classic example during the television series 'Royal Heritage':

'The whole Cullinan Diamond, as it came to be known, was given to my great-grandfather, King Edward VII, on his birthday. The Dutch jeweller who had to split it before it could be polished, was so overcome by the responsibility that he fainted as he struck the blow, but he did it perfectly.'

Fainting or 'blacking out' are common symptoms of overbreathing. A certain proportion of people, however, develop the habit of hyperventilating all the time, while others react excessively in their breathing whenever exposed to emotional stimuli. This type of person is commonly a perfectionist, meticulous in his work, and setting himself very high standards. He becomes tense and easily irritated if he thinks he is falling short of these standards. Then, commonly following a prolonged period of stress or physical illness, he begins to develop symptoms (Table 1) and, because of their strangeness and diversity, he begins to worry that he may be developing some life-threatening disease that doctors do not understand (Lum 1976). Thus he easily gets into the vicious cycle of increased hyperventilation, increased anxiety and increased symptoms, as illustrated in Figure 1 (Lewis 1959). As this over-reaction develops, symptoms may tend to recur in certain situations; the symptoms are then attributed to the situations, and so phobic reactions develop.

## Physiology

Breathing eliminates carbon dioxide ( $\text{CO}_2$ ) and overbreathing eliminates excessive amounts. A single deep breath may cause a momentary fall of 20–25% in the level of carbon dioxide in the blood, while sustained overbreathing can rapidly drop it to half and maintain it at this level. In the normal person the rate and depth of breathing are markedly sensitive to physical and emotional stimuli such as heat, cold, fear, excitement, happiness and pain. Such variation causes changes in the blood carbon dioxide (expressed as  $\text{PCO}_2$ ). The erratic breathing characteristic of hyperventilation (*see* Figure 2B,C) causes a wildly fluctuating  $\text{PCO}_2$ . Between one-half and two-thirds of patients overbreathe consistently so that the  $\text{PCO}_2$  is continuously below the normal range (36–44 mmHg = 4.8–6.0 kPa), a condition of respiratory alkalosis, or hypocarbia. However, even more important is the continued fluctuation in  $\text{PCO}_2$  produced by the erratic breathing.

Carbon dioxide is one of the most important factors which govern activity of nervous tissue. Moreover it diffuses in and out of nerve cells faster even than water, so that changes in blood  $\text{CO}_2$  are quickly followed by changes within nerve cells. Consequently, when arterial  $\text{CO}_2$  drops, carbon dioxide rapidly leaves nerve cells; they become more alkaline and their activity increases, in both sensory and motor neurones. Hence there is motor and sensory hyperirritability, shown by tenseness, tremors or occasionally tetany on the one hand, and

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*Table 1. Symptoms commonly encountered in chronic and acute hyperventilation*

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*Cardiac:* Palpitations, missed beats, tachycardia, 'angina', atypical chest pain, dull precordial or lower costal ache, vasomotor instability

*Neurological:* Dizziness, faintness, visual disturbance, migrainous headache, numbness, paraesthesiae of limbs, face, or elsewhere, intolerance of bright lights or loud noise

*Respiratory:* Irritable cough, 'asthma', tight chest, excessive sighing or yawning

*Gastrointestinal:* Dysphagia, dry throat, flatulence and belching, aerophagy, upper abdominal distress, globus

*Muscular:* Cramps, diffuse or localized myalgia, tremors or course twitches, rarely tetany

*Psychic:* Tension, anxiety, 'unreal' feelings, depersonalization, hallucination, fear of insanity, panic attacks, phobic states

*General:* Weakness, exhaustion, lack of concentration and memory, sleep disturbance, nightmares, emotional sweating (axillae and palms)

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abnormal sensations such as numbness, deadness or 'pins and needles' on the other. The speed of somatic motor reflexes (e.g. pupillary and knee jerks) is increased and there is a selective depression of the parasympathetic system so that the clinical state of the patient reflects sympathetic dominance, i.e. hyperactivity, tension, emotional sweating and instability, and various visceral disturbances of heart, gut and bladder (Wyke 1963, Lum 1981).

The blood flow through the brain is, to a large extent, controlled by carbon dioxide (Lennox *et al.* 1938). When CO<sub>2</sub> falls, cerebral blood flow decreases. In addition, the haemoglobin dissociation curve shifts to the left and so can give up less oxygen (Bohr effect); the combined effect is of cerebral hypoxia. Hypoxia leads to dizziness, faintness, visual disturbances and altered states of consciousness – some of the commonest symptoms of hyperventilation.

**Breathing pattern**

The chronic hyperventilator almost invariably has a characteristic way of breathing. In the normal person the greater part of the air intake is caused by movement of the diaphragm; there is very little movement of the upper chest. Inspiration is the active phase, and expiration is a passive relaxation of the inspiratory muscles. The hyperventilator, on the other hand, tends to exaggerate upper thoracic movement and to use the diaphragm hardly at all. The breathing is also highly erratic and irregular, with wide variations in rate and

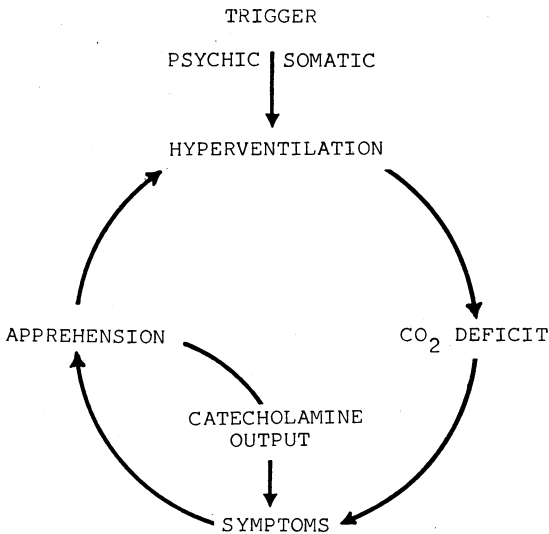


Figure 1. The vicious cycle of events triggered by psychological or physical stress in hyperventilation subjects (modified from Lewis 1959)

rhythm of breathing, frequent sighs, and often a forced, audible expiration. The respiration rate is usually above 15 breaths per minute (average normal 12–14) and is often more than 20; during acute attacks it may reach 30 or more.

These characteristics can be recorded electronically by impedance spirometry (Hamilton *et al.* 1965). Pairs of electrodes placed strategically below the clavicles and in the mid axillae record respirations in the upper and lower chest respectively (Figure 2). Variations in alveolar (and hence arterial)  $PCO_2$  are shown by the percentage of carbon dioxide reached at the end of expiration (normally 5–6%) (Figures 2, 3, 4, 5). Basic lung function tests and chest X-ray are usually done to exclude other lung disease which might cause hyperventilation. Where indicated, an electrocardiogram may be done. The diagnostic test is that of voluntary hyperventilation. Deep breathing at about 40 breaths per minute for up to three minutes will usually produce sufficient symptoms for the patient to recognize.

### **Principles of treatment**

Physiotherapy aims to treat these patients by correcting the breathing pattern and slowing the rate of respiration so as to maintain a constant level of  $CO_2$ , and by teaching general relaxation of the mind and body.

#### *Initial record*

At the first session the patient is made comfortable by sitting in an armchair or in the half-lying position supported by pillows, to help him relax. A record is made of his symptoms, when they started, whether they are continuous and, if not, how often they occur, and whether there is a trigger factor. Note is made of the duration of the symptoms and whether the patient has any way of sending them off. Observations of the breathing pattern are made, whether upper thoracic or diaphragmatic, and the rate and depth noted, including sighs and sounds. The patient is asked if he is aware of his breathing, and in some this will bring on overbreathing, while others are unaware of their breathing pattern. Note is made of any signs of tension – whether the patient is fidgety, sitting on the edge of his chair with clenched fists and tense face and shoulder muscles. He is asked if he is aware of tension and whether he has any methods to help him relax.

#### *Breathing exercises*

The patient is given a simple explanation of the cause of his symptoms and made aware of his breathing pattern by resting one hand on the upper chest and the other on the upper abdominal wall. He is told to let the upper chest relax down and with the next breath in allow the upper abdomen to swell forwards on inspiration, and relax back gently on expiration, so that his breathing becomes silent and effortless. It is often useful for him to observe his breathing in a mirror. Once the rhythm is established, a pause is put in at the end of the expiratory phase in order to slow the rate down to 8 breaths a minute. Patients often feel breathless at first, but they must be discouraged from taking big sighs to compensate. Regular practice sessions are necessary to establish a normal breathing pattern, which must carry on for 24 hours a day to prevent the fluctuations in  $CO_2$  levels which cause the symptoms. The patient should practise at least twice a day for 20–30 minutes and think about his breathing for a few minutes each hour in between. He should time himself occasionally to check that he has a slow steady rate, but not become obsessional about it.

#### *Relaxation*

This should be taught with the patient fully supported in the lying position. The contrast method is used first – the patient is asked to tighten up each group of muscles, e.g. the arms, as much as possible, and then let them relax completely. This shows him the difference between tension and relaxation and should give him some idea how tense he has become. This leads on to general relaxation, where the patient is asked to close his eyes and concentrate on relaxing each muscle group in turn, starting at the feet and working all the way up the body to the neck and face muscles until the whole body feels comfortably warm

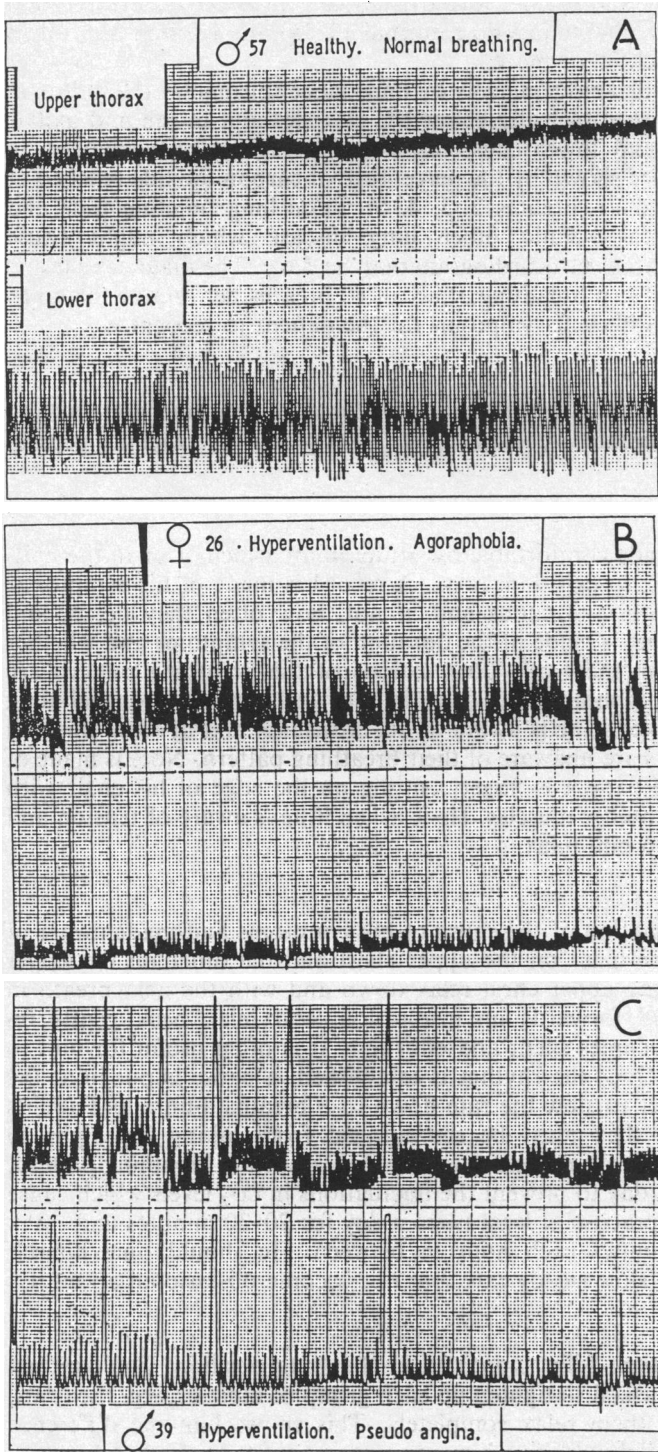


Figure 2. Impedance spirometer records. A, Normal subject: respiratory excursions in upper and lower lungs. Note the predominance of the lower (diaphragmatic) breathing. B, Hyperventilator: note the predominance of upper thoracic breathing and gross irregularity with sighs (large spikes). C, Hyperventilator: excessive upper thoracic movement and frequent sighs. (Reproduced from Lum 1977, with kind permission)

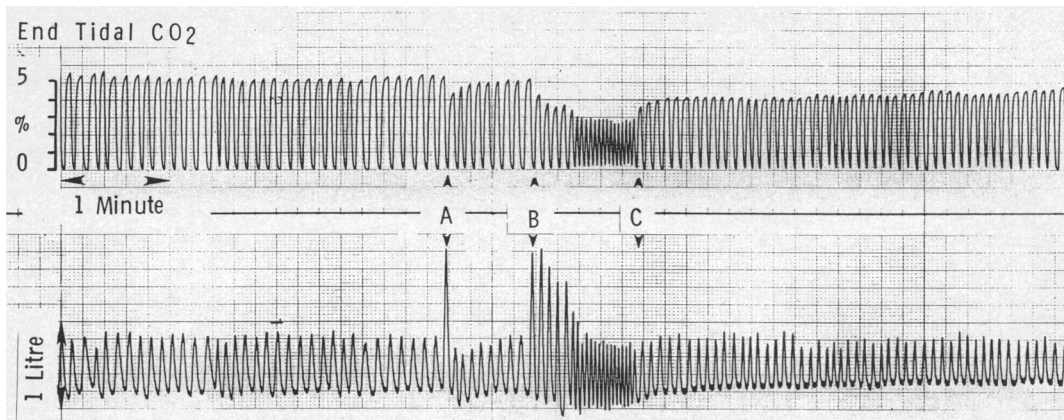


Figure 3. Normal subject: note stable end-tidal  $\text{CO}_2$  during normal breathing; a single deep breath (A) causes a fall from 5.3% to 4.3%; a fall to 3% and below is produced by a few deep breaths between B and C, and recovery time is prolonged – it has not returned to normal after four minutes

and relaxed. The patient should be warned that he is not aiming to fall asleep but should listen to the suggestions and be aware of the feelings of peace and calm that total relaxation brings. The suggestions are kept simple and repeated many times to aid concentration so that they are absorbed by the mind.

Eventually the patient associates the relaxation session with pleasant feelings of warmth and calmness, which in turn build up his self-confidence. This is similar to a light hypnotic trance. A patient who finds concentration difficult may prefer visual relaxation, where he can imagine himself sitting in a quiet place, such as a garden, and associate these feelings with those of calm and peacefulness.

#### *Co-ordination of breathing and relaxation*

Some patients find the breathing control relatively easy, while others master the relaxation first; it is important to combine the two as soon as possible. It can be useful to record a

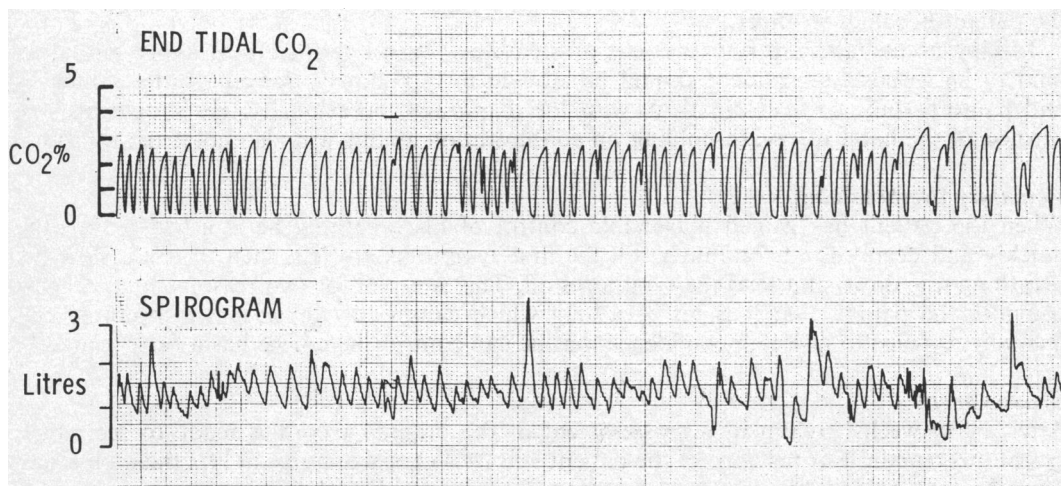


Figure 4. Note low resting end-tidal  $\text{CO}_2$  and gross fluctuations produced by erratic breathing pattern. The patient suffered from dyspnoea and chest pain

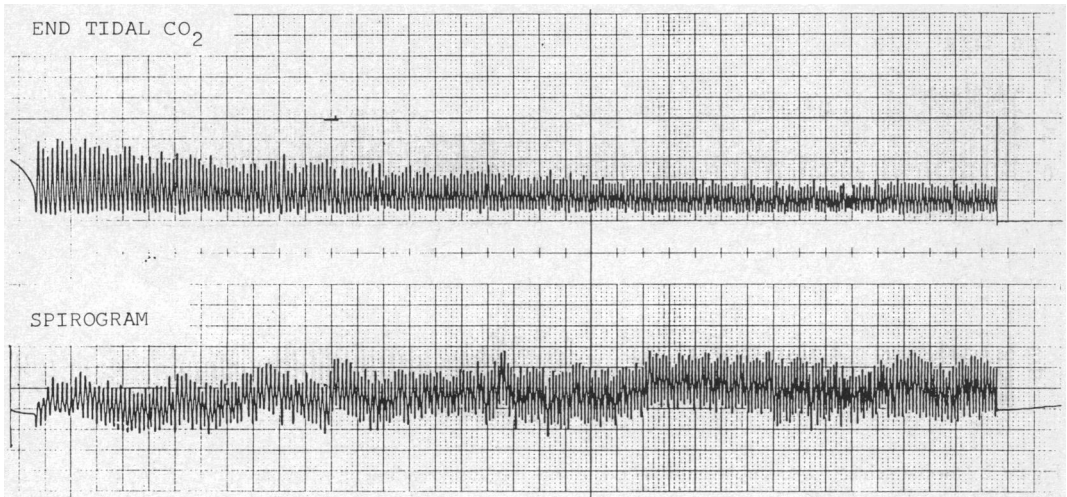


Figure 5. Hyperventilator with agoraphobia: note rapid rate (30 rpm) and severe hyperventilation provoked by breathing through a mouth-piece

cassette for the patient to play back at home, as this aids concentration and reinforces the commands until the correct pattern is established.

#### *Breathing control on exercise*

The patient should progress to practising the breathing exercises sitting, standing and walking, keeping a slow steady rhythm. He is warned that on more vigorous exercise there will be a natural increase in the rate and depth of breathing, but that a certain amount of control is needed to prevent overbreathing and a recurrence of the symptoms, particularly after exercise stops. Many patients have become unfit through lack of exercise as it caused frightening symptoms, so a simple exercise regimen can be given to increase fitness gradually. This may start with simple exercise such as running on the spot, or using a static bicycle for one minute, followed by a pause in which the patient is given time to relax and steady his breathing rate down again. The time and resistance can be gradually increased as the patient becomes stronger.

Talking necessitates a certain amount of breathing control and, as most hyperventilators tend to be verbose, the patient should be advised to talk slowly, deliver shorter sentences and pause to take a small breath in with the diaphragm occasionally. He should also be warned of the habit of breath-holding on concentration as this will also upset the rhythm.

#### *Voluntary overbreathing*

When the patient has gained reasonable control of his breathing he is asked to breathe quickly and deeply for 1–2 minutes, till the first symptoms are felt, then to relax, slow his breathing rate down and send the symptoms off. This is useful for two reasons: first, it helps convince the patient that it is his breathing which causes the unpleasant symptoms, and secondly, it helps to give him confidence that he can control them if he has a further attack.

#### *Reassessment*

After a few weeks' treatment a reassessment of the original record is made to see which symptoms remain. For instance, if the patient still feels claustrophobic in lifts then, on a day when he feels reasonably calm and symptom-free, he can be taken up and down in a lift while being given reassurance and encouragement to concentrate on keeping a steady breathing rhythm.

### *Advice on adjustment of life style*

The patient must train himself to take things more slowly, allowing himself time to relax and enjoy life. He should be reminded that working too quickly will only produce symptoms. He must not set himself such high standards and must learn to be satisfied with less than perfection.

### *Treatment time*

It must be emphasized that treatment may take many months, as the breathing habits of a lifetime cannot be changed overnight. Much time and patience are needed for a complete cure. Patients with severe symptoms benefit from being admitted to hospital, where they can receive treatment sessions, for about 30 minutes, twice a day, staying on average 2–3 weeks before being followed up as outpatients. These and others who come only as outpatients will be seen once or twice a week for the first month and then, if they are making reasonable progress, they will be seen fortnightly and then monthly for six months to a year, till symptom-free. On discharge they are reassured that, should any symptoms recur, they can make an appointment for a few refresher sessions.

### **Results**

At Papworth Hospital the number of chronic hyperventilators seen increased dramatically in the early 1970s and between 1965 and 1981 approximately 1500 patients received treatment by physiotherapy.

The patients were a heterogeneous group generally classified as neurotic, i.e. with anxiety states, anxiety-depression, Da Costa's syndrome, effort syndrome, panic disorders, etc., in which complete remission, whether spontaneous or under treatment, is relatively uncommon. Moreover, diagnostic classification is so imprecise that psychiatrists, even with the same training background, seldom achieve agreement in more than 20% of cases when presented with the same group of patients (Eysenck 1977). The common feature is a background of physical and psychological symptoms which can be shown to be associated with hyperventilation.

The regimen described above was based on the hypothesis that an acquired habit of hyperventilation was the prime physiological mechanism which perpetuated symptoms and psychological disturbance. It was found empirically that patients, irrespective of diagnostic category, could be taught not to hyperventilate and thereafter lost their somatic and psychological symptoms in the vast majority of cases. Lum (1976) reported the status at one year of the first 320 cases treated under his supervision by Papworth physiotherapists: 70% were completely symptom-free, 25% had only minor symptoms, and only 5% failed to respond. Evans & Lum (1977) reported longer-term results in cases of 'cardiac neurosis' (in our experience, and that of others, the most refractory group to treat) and found that 76% remained asymptomatic at 11–68 months. On subsequent follow up at 6 to 11 years the percentage of total cures remained the same (Evans & Lum 1981). This must be contrasted with studies of similar patients treated by regimens which do not correct hyperventilation, where around 90% have residual symptoms, and 22–51% remain unable to work (Brandon 1983).

After the first detailed follow up, this treatment was adopted as a routine in the physiotherapy department for this type of patient. Patients were kept under supervision until either asymptomatic for 6 months, or else having attained maximum benefit.

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