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Comments

THE HYPERVENTILATION SYNDROME

The tension/hyperventilation state is a common cause of psychosomatic symptoms particularly in general hospital practice. This physiological response is the normal accompaniment of states of arousal, particularly those that may lead to intense muscular activity; but, given that modern man rarely resolves his passions by intense muscular activity, he may persist in a physiologically inappropriate state for long periods of time.

The ache and discomfort associated with tense muscles is a commonplace, well understood experience. Although the clinical importance of hyperventilation has been known for many decades, doctors have been very resistant to incorporating the knowledge into their clinical practice. Rice (1950) found that 10.7 per cent of the patients whom he saw in a general medical clinic were suffering predominantly from a hyperventilation syndrome. McKell and Sullivan (1947) in a gastroenterological practice found the syndrome in 5.8 per cent of their patients. Most doctors associate hyperventilation with carpopedal spasm, paraesthesiae and disturbances of consciousness, all manifestations of gross hypocapnia. There is, however, a multitude of symptoms associated with moderate hypocapnia, amongst which the most common are excessive fatigue, palpitations, chest pains, sweating and 'lightheadedness'.

Hypocapnia leads to a reduction of the cerebral circulation and this produces impairment of cerebral function. The effect is compounded by the reduced availability of the oxygen of oxyhaemoglobin in regions of low pCO_2 (Bohr effect). Cellular function is also disturbed by intracellular changes in pH due to reduced pCO_2. Muscular tension is heightened by the generally increased irritability of nerve and muscle consequent upon the reduced ionization of calcium with raised pH. Hyperventilation increases sympathetic arousal which in turn adds to the state of distress.

The individuals who readily produce symptoms with hyperventilation, usually have a low basal pCO_2 due to a habit of hyperventilation even under normal unstressed circumstances. In whatever way the habit becomes established, after a while the individual adapts to lower levels of pCO_2 which will then become the norm for him. At these lower levels the buffering systems of the body are not working at their optimum and cannot cope with a relatively minor decrease in pCO_2, which can then give rise to symptoms.

There are two main components in the aetiology of a hyperventilation syndrome, the persistent bad habit of hyperventilation, and any underlying psychological distress which promotes episodic hyperventilation. The extent of either component in individual series can be partly determined by referral bias and the orientation of the receiving doctor. Thus a respiratory physiologist (Lum, 1975) sees mainly patients who have developed a bad habit of breathing and a psychiatrist (Burns in Burns and Howell, 1969) sees many more patients with underlying depressive and other illnesses.

Diagnosis is based on the clinical history, informed by a knowledge of the manifestations of mild hypocapnia. A relative may have observed frequent sighing of which the patient is unaware, and at interview the patient will often hyperventilate in an obvious fashion. In cases of doubt, voluntary hyperventilation will usually reproduce the patient's physical syndrome.

Treatment is based on the use of a relaxation technique, either through progressive muscular relaxation or with hypnosis/autohypnosis. Both techniques pay attention to breathing, and this approach may be all that is needed to help the patient to breathe normally. In a resistant case specific attention to breathing is necessary. A useful approach is to slow respiration by training the patient to breathe out passively without muscular effort and to insert a short rest period after each respiratory cycle. Another complementary technique is to train the patient to breathe more from the abdomen, thus avoiding the use of the accessory muscles of respiration around the thorax. A simple feedback system that can be very helpful is provided by placing one of the patient's hands on his chest and the other on his abdomen and to instruct him to move the thoracic less than the abdominal hand.

Where a cause for psychological distress is apparent this needs to be tackled with the usual psychiatric armamentarium. If the syndrome is a reflection of an

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underlying depression it is very unlikely that the therapist will be able to relax the patient by the above techniques. (In a distressed patient, the ability to relax can provide a helpful diagnostic test to distinguish the more depressed from the more anxious). Even when considerable psychological distress is apparent this often proves to be secondary to the hyperventilation state and the distress dissipates with attention to the breathing. The relaxation process also contains a number of powerful psychological factors in addition to the retraining process. The patient relaxes in the presence of the therapist, which may help him to overcome a degree of social phobia commonly present in these patients. The patient is allowed to be dependent in the therapeutic situation and often carries with him a powerful fantasy of the therapist's presence which may assist him outside the therapeutic situation.

The patient must be encouraged to practise relaxation every day and warned that it may be some months before his respiratory apparatus adjusts to functioning at normal levels of pCO_2.

When the syndrome is largely due to a bad habit the great majority of patients become asymptomatic after treatment, often feeling dramatically better after years of suffering (Lum, 1975). When the syndrome is secondary to some other primary disturbance the prognosis is more related to that of the primary disturbance.

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