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Demonstration and Treatment of Hyperventilation Causing Asthma

Ambulant, transcutaneous P_{CO_2} monitoring has been used to show that hyperventilation precedes exacerbation of asthma in a patient. Brief treatment was shown to give him greater control of his breathing and enable him to avoid attacks of asthma.

Attacks of asthma are frequently accompanied by hyperventilation of physiological origin (McFadden & Lyons, 1968). However, hyperventilation in asthmatic patients may also be caused by psychological states of extreme arousal or anxiety (Seuss *et al*, 1980; Hibbert & Pilsbury, in press). Hyperventilation, leading to airways cooling, will cause bronchoconstriction in vulnerable individuals (Deal *et al*, 1979; Lewis *et al*, 1984), which in turn will exacerbate the hyperventilation in a continuing vicious circle. A tendency to psychologically induced hyperventilation in an asthmatic patient may therefore be a cause of attacks of life-threatening severity. However, because attacks of asthma are accompanied by hyperventilation of physiological origin, the role of hyperventilation in causing asthma attacks may be overlooked.

The issue is further complicated by the substantial overlap between the symptoms of asthma, panic and the so-called 'hyperventilation syndrome'. Kinsman *et al* (1973), in a cluster analysis of symptoms associated with asthma attacks, found that a 'panic/fear' cluster occurred 'frequently' in 42% of asthmatic patients. Higenbotham & Dent (unpublished), investigating patients referred with a suspected diagnosis of 'hyperventilation syndrome', found that 41% had evidence of asthma. These observations suggest that a significant proportion of asthmatic patients may have attacks that are caused by hyperventilation of psychological origin. Simultaneous monitoring of changes in P_{CO_2} and airway obstruction would be of value in determining whether hyperventilation is causing attacks of asthma. If there is evidence that hyperventilation is causing asthma, teaching the patient to control his breathing may enable him to avoid some of his attacks.

We present the case of a man in whom an attack of asthma was shown to occur after a period of stress-induced hyperventilation, and which appears to have been provoked by it. He learnt to control his tendency to hyperventilate and thereby avoided exacerbations of his asthma.

Case report

The patient was a 20-year-old man with a lifelong history of asthma provoked by exercise or respiratory-tract infections. He was receiving treatment with salbutamol and beclomethasone rotacaps. He was prone to become breathless when aroused and his history suggested that, at times, arousal preceded attacks of asthma. He had suffered several severe attacks of asthma that required emergency admission over the previous year. As a consequence of his medical history he had been unable to obtain employment, and his avoidance of vigorous physical activity was severely disabling him. At assessment, he did not satisfy diagnostic criteria for an anxiety disorder or any other psychiatric diagnosis. He complained only of attacks of breathlessness, which were accompanied by an anxious mood and fears about his health.

Pre-treatment assessment

Before treatment, lung-function tests indicated mildly restrictive airways: forced expiratory volume in 1 s (FEV1), 3.0 litre (pred. 4.3 litre); forced vital capacity (FVC), 3.6 litre (pred. 5.1 litre); peak expiratory flow rate (PEFR), 470 litre/min (pred. 585 litre/min). Previous measures on different occasions had shown a variation of PEFR between 400 and 500 litre/min when he was well. Ambulatory, transcutaneous P_{CO_2} ($P_{tc}CO_2$) monitoring (Pilsbury & Hibbert, 1987) was undertaken, in combination with repeated measures of PEFR. Subjective anxiety was recorded using a visual analogue scale, anchored at 0 by "not at all anxious" and at 8 by "extremely anxious". The data are presented in Fig. 1. At the start of the assessment, his PEFR was 500 litre/min. Anxiety, persistent hyperventilation, and a small reduction in PEFR followed 2 min voluntary hyperventilation of room air, but not of 5% CO_2 + 95% O_2 mixture. Hyperventilation started while he was receiving instructions and preparing for exercise, although he was

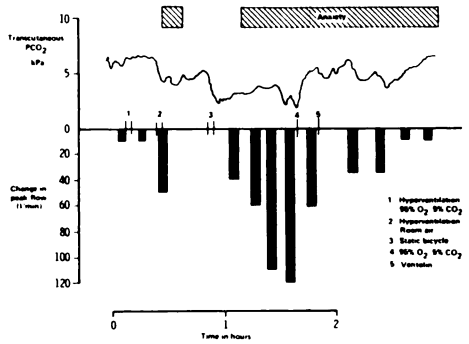

FIG. 1 Pre-treatment assessment of changes in $P_{tc}CO_2$, peak flow, and anxiety.

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not experiencing any anxiety. The period of exercise was accompanied by a steep fall in P_{CO_2} which preceded any evidence of an exacerbation of his asthma. Following exercise, PEFR gradually fell to 380 litre/min over a period of 40 min, noisy wheezing and marked expiratory effort accompanied the deterioration in peak flow measures, and the patient became increasingly anxious about his symptoms as the attack of asthma developed. After 40 min, 5% CO_2 was administered to reduce symptoms caused by respiratory alkalosis and thereby relieve some of his distress, and he was urged to control his breathing. Ventolin was administered to accelerate his recovery from the attack of asthma.

Treatment

The patient received five treatment sessions of 30 min each, over 2 months, in which he learned a technique for controlled breathing. He practised controlling his breathing after voluntary hyperventilation and after increasing degrees of exercise. In the course of treatment, the patient resumed his previous physical activities and became capable of performing levels of exercise never previously achieved.

Post-treatment assessment

The pre-treatment monitoring procedure was repeated and the results are shown in Fig. 2. At the start of assessment, his PEFR was 470 litre/min. During this post-treatment assessment, the patient was able to control his breathing after a period of voluntary hyperventilation, and did not suffer any anxiety or fall in PEFR. During the first 4 min of an incremental work test, his $P_{tc}CO_2$ levels remained stable. However, for the final minute of exercise, he was urged to make maximum effort, which precipitated hyperventilation. He was able to regain control of his breathing, but only with difficulty, as is apparent from the fluctuating return of his $P_{tc}CO_2$ to normal. Further demanding exercise was then undertaken (running). He was able to control his breathing without difficulty. During this procedure, there was no significant change in PEFR and he experienced no anxiety.

Discussion

Transcutaneous P_{CO_2} monitoring, combined with measures of airways obstruction and of anxiety, demonstrated that hyperventilation probably caused exacerbations of this patient's asthma. Hyperventilation preceded asthma, and when it was controlled, the asthma did not occur.

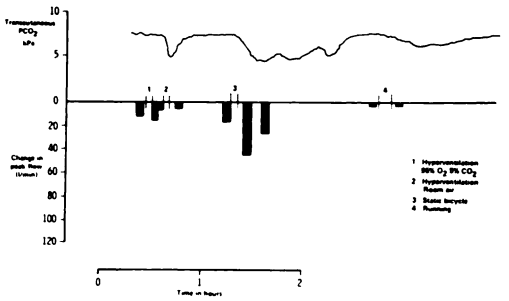

FIG. 2 Post-treatment assessment of changes in $P_{tc}CO_2$, peak flow, and anxiety.

PEFR is a crude measure of bronchoconstriction and, being effort-dependent, cannot alone provide conclusive evidence of asthma. However, during the first monitoring session, clinical observation confirmed that the patient experienced an attack of asthma after exercise. Fig. 1 demonstrates that hyperventilation started before exercise, making it unlikely that the hyperventilation was due to the onset of asthma. The rapid exacerbation of hyperventilation at the start of exercise suggests that it was not hyperventilation associated with a mild degree of asthma that was undetected by changes in PEFR.

During the post-treatment assessment, neither similar nor more vigorous exercise precipitated asthma. The possibility that the difference in response to exercise on the two occasions was due to spontaneous change in his condition is unlikely. Resting PEFR measures, though a crude measure of bronchoconstriction, were very similar at pre- and post-treatment assessments. Post-treatment assessment showed that he had learnt to control his breathing, although not completely. This enabled him to avoid an attack of asthma.

Ambulatory transcutaneous PCO_2 monitoring is of particular value in the assessment of asthmatic patients when psychologically induced hyperventilation is suspected, because it allows prolonged study of freely ambulant patients in naturalistic settings unencumbered by respiratory apparatus. It is therefore possible to study patients in the circumstances in which attacks occur, which are often outside the laboratory.

This case demonstrates that training in controlled breathing can help patients who hyperventilate to avoid some attacks of asthma. This is not to suggest that breathing exercises are an appropriate method for treating attacks of asthma, but rather that they can have a prophylactic effect, making attacks of asthma less frequent.

Acknowledgement

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Pre-Pubertal Depressive Stupor: A Case Report

A case of childhood affective disorder with episodes of depressive stupor in a 13-year-old pre-pubertal boy is described. Changes in the patient's clinical state were accompanied by changes in the dexamethasone suppression test. A family history of affective illness on the maternal side, with phenomenological similarities, is noted.

Manic–depressive psychosis occurs in adolescence (Anthony & Scott, 1960; Carlson & Strober, 1978) but has rarely been reported in the pre-pubertal stage. There are no published descriptions of stupor in this age group. We report a case of depressive stupor occurring on four occasions within 14 months, in a pre-pubertal 13-year-old boy. The patient's mother also suffered from similar but shorter episodes in her adolescence.

Case report

Family background

The patient, a short-statured boy of pyknic build, was the third of five children. His father, aged 45, was a bus driver who exhibited features suggestive of a personality disorder. His mother, aged 36, had a long-standing history of psychiatric illness. At age 14, she was referred to a child-guidance clinic because of her short self-limiting periods of withdrawal, mutism, and refusal to eat. At age 17, she was treated as an out-patient with imipramine for "cyclical depression of the manic–depressive type". Three months later, she was admitted to our hospital in a state identical to that of our patient on his first and subsequent admissions. She showed a gradual spontaneous recovery over a period of 10 days, but between the ages of 18 and 24 was admitted to hospital a further four times. Her hospital notes for this period show the diagnoses of schizophrenia, periodic catatonia, affective disorder (depression), and possible borderline intelligence. Treatments given included the use of ECT, major tranquillisers, and antidepressants. Five months after our patient's first admission to hospital, she once more required in-patient treatment for depression associated with marital and family problems, which led to divorce action, initiated during the course of our patient's third admission.

Physical history

Although our patient's birth was normal, a congenital cardiac valvular defect required his staying in hospital for some months after birth; this defect corrected spontaneously, and his development proceeded unimpaired. Two years prior to referral, he collapsed while playing football, but cardiac investigations at that time were normal. He followed the advice to give up strenuous exercise temporarily, which caused him annoyance, as football was important to him.

History of illness

The patient was described as a lively boy with normal peer relationships. His parents and school personnel had not