

LETTERS TO THE EDITOR

CHRONIC FATIGUE SYNDROME IN ARMY GENERAL PRACTICE

From Dr PGF Nixon

Sir, It may be unfortunate that Dr JH Johnston (J R Med Corps 1994: 140: 59-60) takes the Editorial from the British Medical Journal as his reference point because military medicine is more flexible, and can adopt a systems approach to a problem where the conventional binary system of triage (disease or nor disease) does not provide useful answers. The systems approach accommodates the grey area of reduction of performance and deterioration of health which follows over-use of the individual: Swank and Marchand's model illustrates the point (Fig 1) (1).

Performance deteriorates when we go 'over the top'. Early homeostatic responses include vagal inhibition; sympathetic overactivity; surges of adrenal cortical secretion; and overbreathing. In this hyper-reactive phase we see the picture of the acute Da Costa or effort syndrome.

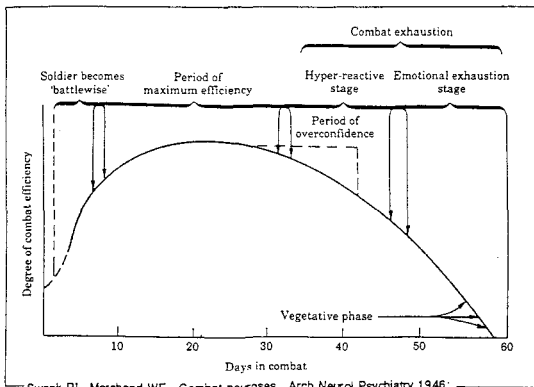


Fig 1. Performance-arousal curve of human function in battle: a model for the systems approach to performance, stress and health. The lower-level stressors of civilian life appear to take about two years to produce effort syndrome.

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If the stressors continue to overwhelm the anabolic processes, the individual is carried down to a level of emotional exhaustion and may breakdown. It is probable that Swank & Marchand's phases of emotional exhaustion and vegetation correspond physiologically with chronic fatigue syndrome even though the time base is different: some thirty days for combat and about two years for the lower but longer drawn out stresses of everyday life.

The evidence suggests that these lower phases have an important metabolic element, namely, depletion of the body's alkaline buffering system brought about by the

overbreathing that characterises the upper phases of the downslope. The overbreathing discharges excessive quantities of carbon dioxide from the body, a process countered by the body's dumping alkalies into the urine. When the body's alkaline buffering systems are depleted the individual is unable to make and sustain effort as he should: the lactic acid produced by exercising muscle cannot be buffered properly *in situ*. It reaches the central circulation and the brain responds by increasing the ventilation. The end-tidal, end expiratory or alveolar $p\text{CO}_2$ levels fall. The point at which they began to fall is called the respiratory response to the anaerobic threshold. In a study of anaerobic threshold in chronic fatigue syndrome this respiratory marker occurred at a very low level, below the requirements of everyday activity. In other words, one of the clinical problems of the chronic fatigue patient is 'living above the anaerobic threshold'.

The clinical consequences include overwhelming fatigue; inability to make and sustain effort; musculo-skeletal aches and pains; sleep disturbance; difficulty of resting or being still; and increased neuronal sensitivity and reactivity.

The appropriate tests are not described in the British Medical Journal. They assess the competence of the body's alkaline buffering systems, and include breath-holding time and measurement of the respiratory response to rapidly-incremental exercise.

Lewis was the first to discover the metabolic disorder of buffering that characterises his effort syndrome. He pointed out that those who ignored this aspect of the syndrome would find themselves creating fanciful diagnosis and taking pejorative views of the subject's moral worth and keenness for work. His system of rehabilitation was more effective than ours: six weeks was the period usually required (2).

It is unfortunate that his work on the pathogenic effects of buffer depletion was discounted when normal or near-normal levels of plasma bicarbonate were found: it was not realised in those days that the plasma bicarbonate is a poor indicator of the body's ability to buffer the acidic products of exercise. The major contributors to buffering are contained in the intracellular compartment (75%), the phosphates and the plasma proteins. As with potassium, the plasma level tends to remain normal until the cellular compartment is severely depleted.

The work discussed here, its historical aspects and references have been published recently (3-5).

I am etc
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REFERENCES

1. Swank RL, Marchand WE. Combat Neuroses. *Arch Neurol Psychiatry* 1946; 55: 236-47.

2. MEDICAL RESEARCH COMMITTEE. Reports upon soldiers returned as cases of 'disordered action of the heart' (DAH) or 'valvular disease of the heart' (VDH). London. Her Majesty's Stationery Office 1917:13.
3. NIXON PGF. The grey area of effort syndrome and hyperventilation: from Thomas Lewis to today. *J R Coll Physicians Lond* 1993; **27**:377-83.
4. NIXON PGF. Effort syndrome: hyperventilation and reduction of anaerobic threshold. *Biofeedback Self Regul* 1994; **19**:155-69.
5. NIXON PGF. Breathing: physiological reasons for loss of self-control. In: Carlson JG, Seifert AR, Birbaumer N, eds. *Clinical applied psychophysiology*. New York, Plenum, 1994: 157-67.

PARADOXICAL BRONCHOCONSTRICTION WITH SALBUTAMOL

From Maj G Wheatley, RAMC and Capt S Walden, RAMC

Sir, the recent published interest in Paradoxical Bronchoconstriction in B2-agonists (1) has coincided with two recent experiences of this phenomenon in our practice.

The first concerned an 11 month old boy who was a known asthmatic and had been seemingly stabilised on Becotide twice daily and nebulised Salbutamol on a required basis. Although initially his mother felt that the Salbutamol had helped, she had gradually found that his coughing and wheezing was worse after taking the drug. He and his mother were brought in to be supervised with the usage of the nebuliser. After 2.5mg Salbutamol his

chest was noticeably more wheezy and he had an increased respiratory rate. Nebulised Ipratropium Bromide was tried, which made a considerable improvement to his wheeziness. Subsequently he was admitted to hospital where a repeat of the problem occurred and he has now been stabilised on Ipratropium Bromide.

The second concerned a two year old boy with persistent night coughing for ten weeks. He had started with some other URTI symptoms (fever, running nose), but only the cough had persisted. During the day he was comfortable, had a normal appetite and seemed happy, with only an occasional cough. At night, he had regular coughing paroxysms that kept him and his parents awake and sometimes ended in vomiting. A trial of Salbutamol Syrup 2mg four times daily was stopped after four days at the parents request as his nightly coughing became markedly worse.

The existence of paradoxical bronchostriction in B2-agonists is established (1): Measuring its prevalence and severity in Primary Care presents a problem - its characteristics of worsening wheeze or cough are subjective, particularly so in young children who are unable to use a peak flow meter. We would be interested to hear of other cases.

We are etc
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REFERENCES

1. Tattersfield AE Use of Bs agonists in asthma much ado about nothing; *Br Med J* 1994; **309**: 794-795.