Correspondence

Brainstem perfusion in CFS

Sir,

Differences of opinion uncover opportunities for research, and so I do not regret my inability to accept the letter from Dr Costa and his colleagues as an answer to my comments.

I agree that mild and moderate hyperventilation can produce vasoconstriction, whereas severe and prolonged hypocapnia does not. Lum has given the reason: in minor respiratory alkalosis the neurone is stimulated, but in major and prolonged hypocapnia the accumulation of lactic acid reduces neuronal activity even to the point of extinction.¹

As Costa et al. point out, people exposed to high altitudes for 3–5 days have higher cerebral blood flow than at sea level, but this is the period required for acclimatization, and it is unsurprising that the processes of adaptation can be generous.

Rosen and I have already commented on the tests that purport to tell us whether important acts of hyperventilation have occurred: as yet without agreement.²⁻⁵

On the other hand, it is possible to diagnose gross depletion of the body's alkaline buffering systems by identifying the respiratory response to the anaerobic threshold using capnography during rapidly incremental exercise.⁵ This buffer depletion is usually due to overbreathing in response to effort and distress, and its symptoms are indistinguishable from ME/CFS.⁵

Finally, the Costa team argue that reduction of cerebral blood flow in hyperventilation, in contrast to their findings in CFS, is global or generalized, and assert 'there is, so far, no evidence to suggest that regional differences are likely to exist'. This is a bold claim when Wyke has already demonstrated it,⁶ and Terada et al. have reported that Tc-99 HMPAO brain SPECT studies of hyperventilation 'have greatly contributed to the quantitative evaluation of focal perfusion decrease'.⁷

I hope that others will investigate depletion of the alkaline buffering systems before concluding that there is no known cause of ME/CFS symptoms. I shall continue to rely on Wyke and Terada's work, because their findings provide an explanation for the repeated focal and transient ischaemic attacks that can be found in exhaustion and hyperventilation where there is no evidence of organic arterial disease or embolism.

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References


Diagnosis, disease and illness

Sir,

Drs Mayou and Sharpe make some helpful points in their article on 'Diagnosis, disease and illness' (QJM November 1995), but as psychiatrists they should be aware of the beam in their own eye before endeavouring to remove the mote from ours. Psychiatric diagnoses are based on pattern recognition rather than the understanding of a process as implied by the word 'diagnosis' and seem to be at the same stage as medicine was when its categories were such entities as fever, phthisis, etc. The writers should surely also have mentioned Himsworth's concept of a syndrome not as a set of associations—things that experience shows tend to run together—but as the...