Correspondence

Da Costa’s syndrome or neurocirculatory asthenia

Sir,

Da Costa’s syndrome or its major physiological elements are common in patients with normal coronary arteries and in coronary arterial disease,1-3 and their management lies in the province of cardiac rehabilitation. From the therapeutic point of view it is surprising to find Dr Paul (1987;58:306–15) omitting some of the most important considerations. For example, he does not refer to the extensive work that has given a leading aetiological role to hypocapnia and respiratory alkalosis.4,5 Failure to recognise this role of hyperventilation is an error: its neglect prevents a rational approach to the dynamic factors that are capable of reducing the coronary circulation,1-3 it invites a needlessly high surgical referral rate,2 and takes away the possibility of dealing with a major cause of illness behaviour after myocardial infarction.1-3

The exhaustion factor so well known to Thomas Lewis and James Mackenzie requires treatment because it promotes high levels of arousal and sympathetic nervous activity that are aggravated by hyperventilation, together with habituation defects, limbic dysfunction, and disturbed activity of the left cerebral hemisphere.6,7 The high levels of arousal interfere with sleep, reduce anabolic opportunities,8 and lower the threshold for angina pectoris.2 Defective habituation encourages the heart rate, the blood pressure, and other neuroendocrine responses to stimuli to become “undamped” and excessive, and thereby reduces the capacity for effort. The patient’s furious struggling to keep up a customary range of activities perpetuates the arousal and increases the disabilities.8

We believe that the cardiac rehabilitation programme should accommodate these physiological factors and pay close attention to sleep, arousal, the breathing, the balance of rest and effort, and the self-esteem of the patient (acronym, SÀBRES).29

Reassurance does not have a longlasting effect upon the physiological degradation caused by hyperventilation and disordered habituation, nor can physical training restore high level fitness and stamina where performance is undermined by sleep loss, hyperarousal, hyperventilation, exhaustion, and loss of self esteem from seemingly endless cycles of frustration and defeat.

Dr Paul’s recommendations sound uncomfortably like those that obtained no more than a 15% recovery rate in the era before we learned about hyperventilation and habituation.

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References


This letter was shown to the author, who replies as follows:

Sir,

I thank Miss King and Dr Nixon for their comments on my review of Da Costa’s syndrome (1987;58:306–15).
Their first concern is to emphasise the "leading aetiological role" of hyperventilation with hypoxia and respiratory alkalosis in this syndrome. I agree that most patients with this condition complain of breathlessness, some may sigh intermittently, and a few may hyperventilate chronically. (Comroe defines hyperventilation as "alveolar ventilation in excess of that needed to eliminate metabolically formed CO₂".) Unfortunately, as the reports cited both in my review and in the above letter indicate, although there have been excellent descriptions of physiological and clinical abnormalities resulting from hyperventilation (as defined by Comroe), it has not been possible to equate these with Da Costa's syndrome. Most patients with the syndrome do not hyperventilate chronically, their blood studies do not reveal consistent alkalosis, and essentially they never show the signal features of full-blown hyperventilation—that is numbness and tingling of the hands, feet, mouth, and tongue, spasms of the hands and feet, twitching, and finally convulsions. The whole issue was discussed by Paul Wood who wrote "the total evidence indicates that hyperventilation cannot be held responsible for the symptoms and signs of Da Costa's syndrome, neither by causing tissue alkalosis nor by interfering with the circulation; but it does occur in some, and when present must aggravate certain of the symptoms." I find no reason with the passage of 47 years to differ with these conclusions.

Their second comment calls attention to exhaustion and its complications. I agree that there has been solid support for the role of exhaustion in certain patients, especially during wartime conditions.

Their third group of remarks relates to rehabilitation and treatment and in general I also concur with these, although I have found reassurance to be extremely helpful, and indeed critical.

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References

Notices

British Cardiac Society

The Autumn Meeting will be held at the Wembley Conference Centre, London, on 22 to 24 November 1988. The closing date for receipt of abstracts has been changed to 24 June 1988 and is not as indicated in an earlier notice.

The Annual General Meeting for 1989 will take place in Oxford on 6 and 7 April 1989, and the closing date for receipt of abstracts will be 6 January 1989.

Lasers in cardiovascular diseases

The 2nd International Symposium on Lasers in Cardiovascular Diseases will take place in Vienna on 3 and 4 October 1988. Inquiries to Congress Secretariat: Cosmos Convention Service, PO Box 141, Vienna, Austria.