

Chronic fatigue syndrome--aetiological aspects.

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The chronic fatigue syndrome (CFS) has been intensively studied over the last 40 years, but no conclusions have yet been agreed as to its cause. Most cases nowadays are sporadic. In the established chronic condition there are no consistently abnormal physical signs or abnormalities on laboratory investigation. Many physicians remain convinced that the symptoms are psychological rather than physical in origin. This view is reinforced by the emotional way in which many patients present themselves. The overlap of symptoms between CFS and depression remains a source of confusion and difficulty. But even if all CFS patients were rediagnosed as depressives, this would not negate the possibility of an underlying organic cause for the condition, in view of the growing evidence that depression itself has a physical cause and responds best to physical treatments. There is some evidence both for active viral infection and for an immunological disorder in the CFS. Many observations suggest that the syndrome could derive from residual damage to the reticular activating system (RAS) of the upper brain stem and/or to its cortical projections. Such damage could be produced by a previous viral infection, leaving functional defects unaccompanied by any gross histological changes. In animal experiments activation of the RAS can change sleep state and activate or stimulate cortical functions. RAS lesions can produce somnolence and apathy. Studies by modern imaging techniques have not been entirely consistent, but many magnetic resonance imaging (MRI) studies already suggest that small discrete patchy brain stem and subcortical lesions can often be seen in CFS. Regional blood flow studies by single photon-emission computerized tomography (SPECT) have been more consistent. They have revealed blood flow reductions in many regions, especially in the hind brain. Similar lesions have been reported after poliomyelitis and in multiple sclerosis--in both of which conditions chronic fatigue is characteristically present. In the well-known post-polio fatigue syndrome, lesions predominate in the RAS of the brain stem. If similar underlying lesions in the RAS can eventually be identified in CFS, the therapeutic target for CFS would be better defined than it is at present. A number of logical approaches to treatment can already be envisaged.