Matters arising

There are two important corollaries that surgeons ignore at their patients' peril; first, movement is an essential factor in the pathogenesis of cervical spondylotic myelopathy\(^6\) and so full flexion-extension lateral radiographs of the cervical spine are an essential preoperative investigation. Second, decompressive laminectomy should be avoided in those patients with cervical spondylotic myelopathy with a high range of movement. These patients are liable to develop delayed postoperative myelopathy. Decompressive laminectomy should be reserved only for those patients with diffuse spondylosis causing a relatively immobile but narrow cervical canal; as Barnes pointed out these patients tend not to deteriorate neurologically because of the low range of movement,\(^4\) but if they do, then decompressive laminectomy is indicated.

I welcome Avrahami and colleagues' article and also the opportunity to stress the importance of assessing the biomechanics of cervical spondylotic myelopathy. Surgeons should not advocate one particular operation for this condition. Each patient requires the appropriate operation for the particular biomechanical factors present in that patient.

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The prevalence of multiple sclerosis in south east Wales

SIR: In a recent report of a prevalence survey of multiple sclerosis in south east Wales. Swincer and Compston\(^7\) estimate the mean duration of disease to be 33 years. This estimate was derived using a method first described by Poskanzer et al.\(^2\) They proposed that "... assuming that there is no change in the basic pattern of the disease its average duration may be calculated as twice the average period from onset to prevalence day." The idea is that, for any individual patient, prevalence day is a random time. On average therefore the time from onset of disease to prevalence day will be half the duration of the disease.

Leaving aside the questionable assumptions that must be made about the constancy of the underlying rates of incidence and survival, this method is almost certain seriously to overestimate the true mean duration of disease. At any given time, such as prevalence day, the probability of a patient with disease of long duration being alive is greater than the probability of a patient with disease of short duration being alive. Indeed, the probability of an individual patient being alive on prevalence day is directly proportional to the duration of his disease. Asking patients how long they have had the disease on prevalence day will therefore give a biased estimate of the mean duration of disease. Only in the extremely unlikely event that all patients survive for exactly the same length of time after the onset of the disease would the method be valid.

The size of the error depends on the shape of the patient survival curve. As an example, if this curve is exponential, the estimated duration of disease will be twice the true duration. Mortality in multiple sclerosis increases with time and the survival curve cannot be characterised by an exponential function. This has the effect of reducing the size of the error. Even so, Swincer and Compston's estimate of the mean time from onset of multiple sclerosis to death is likely to be considerably too long.

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References

Delayed somatosensory evoked potentials in pernicious anaemia with intact peripheral nerves

SIR: We read with interest the communication on delayed somatosensory evoked potentials in pernicious anaemia with intact peripheral nerves.\(^3\) We would like to report the case of a 31 year old man with sub acute combined degeneration of the spinal cord in whom peripheral nerve conduction studies were essentially normal but whose initially abnormal somatosensory evoked responses (SSEP) recovered substantially over a one year period following the initiation of treatment.

He presented in October 1986 complaining of ataxia and lower limb weakness. A year before, he had developed tenderness of the thighs associated with tight sensations in the legs and a disturbance of gait. Over the next few months the symptoms improved but failed to resolve completely. In August 1986, he had an illness associated with vomiting following which his neurological status deteriorated. He used a stick when walking and noticed stiffness and weakness of his legs. He became aware of parasthesiae in his hands. His speech and thought processes had slowed. His tongue felt sore and sensitive. His grand mother had had pernicious anaemia and his father had vitiligo. His diet was unremarkable and he was not taking medication. On examination he was sallow and mentally slow. The cranial nerves were normal as were the upper limbs apart from depression of the supinator reflexes. The lower limbs were spastic with weakness of hip and knee flexion. The ankle reflexes were absent with flexor plantar responses. He had marked heel shin ataxia and an unsteady gait. Joint position sense was depressed in the feet and vibration sense absent to the costal margins. Cutaneous sensation was normal.

Investigative findings included a haemoglobin of 12·8 g/dl with an MCV of 110 fl. Vitamin B12 concentration was 60 ng/l and serum folate 11·0 ng/l. The bone marrow was megaloblastic. A standard biochemical screen was negative. An auto antibody screen, including tests for parietal cell and intrinsic factor antibodies was negative. He was found to have achlorhydra