There are two important corollories that surgeons ignore at their patients' peril; first, movement is an essential factor in the pathogenesis of cervical spondylotic myelopathy[6 7] 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,[6] 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.

CBT ADAMS
The Radcliffe Infirmary, Oxford, OX2 6HE, UK

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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, Swingler and Compston[1] 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 event. 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, Swingler and Compston's estimate of the mean time from onset of multiple sclerosis to death is likely to be considerably too long.

CN MARTYN
C OSMOND
MRC Environmental Epidemiology Unit, Southampton General Hospital, Southampton SO9 4XY, UK

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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.[1] 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 achlorhydria

and a Schilling test revealed an absorption in part I of less than 5%, rising to 19·5% after addition of intrinsic factor. Cerebrospinal fluid examination was normal.

Motor and sensory conduction studies in the upper and lower limbs were normal apart from an absent right sural sensory action potential. Somatosensory evoked responses (SSEP) were measured following median nerve stimulation. N9, N13 and N20 responses were assessed on admission, and after two, five and twelve months of replacement therapy. Treatment with hydroxycobalamin produced symptomatic improvement. His sensory symptoms disappeared and by three months he was walking unaided, though his gait remained spastic and ataxic. The spasticity lessened thereafter and by six months he had returned to work. After one year, joint position sense loss in the feet was minimal and both ankle jerks were obtainable.

Studies of both peripheral and central conduction studies have been performed in patients with pernicious anaemia. There have been four previous analyses of somatosensory evoked potentials in sub acute combined degeneration of the spinal cord.[1-4] In the first, three patients were studied.[2] In addition to measurement of peripheral nerve conduction, and F responses, N9 and N1 (N20) were recorded. The F responses and the N9 latencies were either slightly prolonged or at the upper limit of normal. N1 (N20) latencies, on the other hand, were significantly delayed with values between 22 and 23 ms, compared to values of less than 19 ms in control subjects. The findings were interpreted as representing the consequent of severe demyelination in the posterior columns of the spinal cord with slight axonal loss in the peripheral nerves. The second paper on the subject assessed seven patients, though only two of these had substantial neurological involvement.[3] Median somatosensory responses were normal in all of them but two (those neurologically affected) had absent peroneal responses which reappeared on testing. No values for somatosensory responses at this time were given. In the third paper,[4] concerning two patients, somatosensory evoked potentials with median nerve stimulation were normal, but those elicited by peroneal nerve stimulation revealed prolonged central conduction times. Peripheral sensory and motor nerve action potentials were reduced with normal or slightly reduced conduction velocities. The authors postulated that the main pathological change in the central nervous system might be demyelination in the posterior columns in addition to axonal degeneration in the peripheral nerve. In the fourth paper on the subject,[1] which reports similar results to our own, electrophysiological testing was performed on a 72 year old lady with pernicious anaemia 6 weeks after the onset of symptoms and subsequently after 9 months treatment. Peripheral nerve conduction studies were normal throughout. The somatosensory cortical responses N20, P25 and P40, following right median nerve stimulation, were markedly delayed but shortened in latency after nine months Vitamin B12 therapy.

In our patient, there were abnormalities of central conduction (absent N13 and delayed N20) following stimulation of the median nerve, tests of peripheral conduction in which had been normal. The initial improvement in the SSEPs, the reappearance of N13, was noted five months after starting therapy. The N20 potential remained delayed with the retest seven months later. The further shortening of these latencies, between five and twelve months after the initiation of therapy, was mirrored by improvement in the patients' neurological status.

Our findings and those of others indicate that in sub acute combined degeneration, altered somatosensory responses are a sensitive index of the presence of pathological changes in the sensory pathway. The predominant abnormality and later recovery, of the N20 responses reflect the known tendency for pathological change to centre on the dorsal columns.

GD PERKIN
SW ROCHE
R ABRAHAM
Regional Neurosciences Centre, Charing Cross Hospital, London
Address for correspondence:
DR S ROCHE
Department of Geriatrics, Central Middlesex Hospital, Acton Lane, London NW10 7NS, UK

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Elastic properties of muscle measured at the elbow in man. Defining velocity sensitive resistance to passive movement in a clinically measurable manner.

Sir: Normal muscle "tone" does not normally depend on neural mechanisms. The resistance to passive movement should theoretically represent mechanical factors only,[1] and those factors should not change even with anaesthesia.[2] Yet velocity controlled movement does have a threshold where neural effects can be seen in normals and these effects together with complex resistances cloud the separation of the normal and spastic individual.

Wiegner *et al* have solved a number of problems defining the elastic or position sensitive component of the resistance to passive motion.[3] The elastic components can be demonstrated by observing the forces at slow velocities well below the threshold of reflex activation, thus providing important information about intrinsic changes within the morphology of the muscle. While the authors appropriately apply the method to the rigidity of Parkinsonism,[4] they also recommend this tool for objective assessment of spasticity.[3] These authors report 0·014, SD 0·0294 NM/deg. compliance for the elbow in the mid-range of movement,[3] almost identical with that found by others.[5] As a practical matter, with certain assumptions, the effect of spasticity may be objectively demonstrated in a far simpler manner. Spasticity can be defined as the velocity sensitive resistance to passive movement.[6] If the elbow is passively oscillated at faster and faster velocities, the elastic components (even in normal subjects) are relatively insignificant compared with the viscous and inertial effects. A passive constant-velocity dynamometer that is available in the clinic that includes a built-in dual channel EMG can be used as an effective tool.[7] Hill has suggested that taking velocity as the independent variable has the advantage of "better definition of length-tension curve particularly at the end of the curve with the higher velocities".[8] Also if the muscle fibre were excited, "the fibre would not have to move anything except itself".[8]

Relationship between area of passive length-tension curve and velocity. The velocity sen-