Table *Neopterin and biopterin levels in Down's subjects and healthy controls*

	N	Neopterin/ creatinine (mean, SD)	Biopterin/ creatinine (mean, SD)	Creatinine (mmol/l) (mean, SD)	N:B (mean, SD)
Downs syndrome	53	0·47, 0·32	0·17, 0·14	8·22, 5·3	3·44, 1·7
Control	32	0·14, 0·17	0·11, 0·1	12·3, 11·8	1·12, 0·55
p		<0·2%	NS	NS	<0·2%

Units μmol pteridine/mmol creatinine.

reduction in the baseline will be compensated for by reduced pterin excretion. This would also account for the elevated levels of biopterins in the plasma of such subjects.[10]

There is a greater incidence of Alzheimer type changes in Down's syndrome patients than in the normal population,[11] and Down's syndrome has been suggested as a model for accelerated ageing.[12] However, there is no correlation between age and urinary N/B ratio in Down's syndrome from birth and not one acquired with age.

RJ CATTELL*
CGB HAMON*
JA CORBETT†
J LEJEUNE‡
JA BLAIR*
*Pharmaceutical Sciences Institute, Aston University, Aston Triangle, Birmingham B4 7ET, UK
†Department of Mental Handicap, Lea Castle Hospital, Nr Kidderminster DY10 3PP, UK
‡Chaire de Genetique Fundamentale, Institut de Progenese, 15 Rue de 1 Ecole de Medecine, 75270 Paris Cedex 06, France

Correspondence to Dr CGB Hamon, Sherborne School, Abbey Road, Sherborne, Dorset DT9 3LF.

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Matters arising

Magnetic resonance imaging in patients with progressive myelopathy following spinal surgery

Sir: Post operative cervical myelopathy is a poorly recognised but important condition. The article by Avrahami *et al*[1] demonstrating by magnetic resonance imaging spinal cord cavitation and atrophy, is an excellent contribution to this subject. I suspect surgeons underdiagnose this condition preferring to blame the preoperative diagnosis rather than their operation. Bradshaw[2] was one of the first to describe this condition and Aboulker *et al*[3] opined that it was due to the development of progressive post-operative kyphosis.

Avrahami and colleagues pointed out that post-operative myelopathy develops more slowly after surgery for spinal stenosis or disc disease (15 months to 5 years) than after intra-medullary surgery but do not speculate on the mechanism. Eighteen years ago I published data on delayed post-operative myelopathy.[4] The onset of deterioration was, on average, 2·3 years after the operation. There was no statistically significant relationship between post-operative kyphosis (or lordosis) in this group compared with other groups; there was a highly significant relationship between the post-operative range of movement of the head and neck, and the development of post-operative deterioration. If the dura was opened, and especially if left open, then this condition occurred even in the presence of a lower range of movement of the head and neck, less than 40°.

I felt, and still feel, the mechanism is due to the disruption of the normal physiological movement of the cord and dura which has to adapt to this extremely mobile part of the spinal column.[5] After laminectomy the dura becomes adherent to the muscle (or after anterior surgery, to the posterior longitudinal ligament if severed). Normally the dural sheath is fixed at the foramen magnum. Hence this dual dural fixation produces during flexion a severe traction injury to the dura (and cord), if and when a high range of post operative movement develops. Obviously if the dura is opened, and especially if left open, the cord becomes directly adherent to the muscle with an even greater vulnerability to traction damage. Not surprisingly, Avrahami *et al* find that this condition develops more rapidly and seriously after intradural rather than extradural operations. This is my experience too.

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