Figure A Averaged surface, rectified EMG from the first dorsal interosseus muscle, during weak voluntary contraction, in response to 50 magnetic stimuli at a strength (37% of output) below threshold for direct excitation. **B**: Transcranial magnetic stimulation (below) and csum (above) of the discharges of a single motor unit in the first dorsal interosseus during 100 magnetic stimuli at 37% output under identical conditions to those in A.

65. Thus the depression represents an 88% reduction in probability of discharge. The period of suppressed firing is followed by a pronounced increase in the probability of discharge lasting about 10–12 ms. There is no evidence in the peri-stimulus time histogram of a period of facilitation before the depression and this is emphasised by the flat time course of the csum.

The responses to transcranial magnetic stimulation of three other motor units were examined in the same subject. All showed the same qualitative pattern of response to transcranial magnetic stimulation at a strength (37%) just below threshold for exciting contraction of first dorsal interosseus. The periods of suppressed firing of the other three units started 31, 29, and 29 ms after the stimulus and lasted for 27, 23 and 21 ms respectively. On reducing the strength of stimulation to 35% of maximal output suppression of motor unit discharge was still evident starting at 29 ms after transcranial magnetic stimulation but lasting only 12 ms.

The results show that substantial suppression of motor unit discharge in normal humans can occur in response to transcranial magnetic stimulation in the absence of an earlier period of excitation. This was to be expected from our previous findings^{1–3} and shows that the suppression is not restricted to instances of neurological disorder.⁴ The finding expands the realm of transcranial magnetic stimulation in the investigation of cortical motor function in normal humans. It also means that abnormal responses to transcranial magnetic stimulation in cases of disordered brain function may reflect disturbance to either excitatory or inhibitory inputs to corticospinal neurons.

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Boniface and Mills reply:

We note with interest the results of Ellaway *et al* on the inhibition of motor unit discharge by transcranial magnetic stimulation in the intact human nervous system. This raises important issues related to the mechanisms involved and to our own findings in a patient with multiple sclerosis (MS).¹

We have studied five single motor units from the first dorsal interosseus muscle of four healthy subjects at stimulus intensities just below that required to produce a short latency excitatory response. A partial but significant reduction in firing probability was produced in three of these five motor units which was confirmed on csum analysis (Mills and Boniface, unpublished observations). Complete suppression of discharge as in the patient with MS, however, was not seen.

The prominent late peak evident in the peristimulus time histogram of Ellaway *et al* has a latency similar to the secondary peak described in histograms obtained at higher stimulus intensities.^{2,3} This may reflect not only an accumulation of delayed discharges from the inhibitory period but also a late excitatory event. This was not encountered in the three motor units from the patient with MS suggesting a different mechanism for the observed changes in firing probability.

High stimulus intensities are sometimes used when studying MS patients because of the high motor threshold, which is a common feature in this condition. The effects of transcranial magnetic stimulation on firing probability therefore are not necessarily directly comparable between patients and healthy subjects. Problems also arise from the selective nature of single motor unit recordings with transcranial magnetic stimulation which sample a small number of low threshold motor units. Despite these difficulties, it is clear that transcranial magnetic stimulation produces some suppressive effects in healthy subjects, but so far descriptions of the complete silencing of motor unit discharge in the absence of peaks in the peristimulus time histogram are confined to one patient with MS. It is conceivable that this phenomenon is a function of stimulus intensity, both in absolute terms and in relation to motor threshold. The evidence, however, suggests that changes in the balance of descending excitatory and inhibitory influences on the motor neuron by transcranial magnetic stimulation, whether by transient withdrawal of excitatory drive or by active inhibition, are different in the patient with MS when compared with healthy controls. These differences can be exploited to study the disordered central mechanisms of motor control in neurologi-

cal disease and the role of inhibitory processes.

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MRI in a case of rigid Huntington's disease

In a recent paper Savoiardo *et al*¹ report MRI findings of 18 patients with Huntington's disease (HD), seven of whom had the rigid variant. They found increased signal intensities of the caudate and putamen in all the rigid patients in intermediate weighted images and in all but one in T2 weighted images. All but one hyperkinetic patient had no signal abnormalities on MRI. Sethi² and Kang *et al*³ also report putamen hyperintensities in single cases of rigid HD. In contrast, Rutledge *et al*⁴ reported hypointensities of the caudate and putamen in four choreatic patients. Several authors either did not report on intensity changes or explicitly did not find any. We report a case of rigid HD with a pattern of MRI findings, which has not been described previously.

At the age of 18 our patient developed clumsiness of her hands. There was a rapid progression with development of gait disturbance, dysarthria, loss of spontaneous movements, urinary dysfunction and hyper-salivation. When referred to our hospital at the age of 26 she had orofacial apraxia, aphonia, spastic gait, severe bradykinesia with bamboo pole-like posture, rigidity of all extremities, dystonia of the upper extremities with hyperextension of the fingers, abducted arms and flexed elbows. Tendon reflexes were exaggerated with bilateral Babinski signs. Blood smears were negative for acanthocytes. Four other members of the family were also affected by either choreatic or rigid movement disorders. Pathological investigations were not carried out on any of these cases. Mode of inheritance seemed to be autosomal dominant. Our diagnosis was rigid HD.

MRI (1.5 Tesla) in our patient showed bilateral caudate atrophy (figure). Additionally, on T2-weighted images (SE 2.1/90) there was mild hypointensity of the putamen and the globus pallidus, which did not involve the caudate. The same constellation was found in one of the male relatives, who was hyperkinetic and the only clinically affected family member that we were able to study.

Our findings do not agree with Savoiardo's study in three ways: 1) instead of hyperintensities we found hypointensity in the basal ganglia; 2) in our case the puta-

Figure MRI showing bilateral caudate atrophy.

men and globus pallidus were involved with sparing of the caudate; 3) we found no differences between the MRIs of the rigid and the choreatic patient.

Signal hyperintensities are thought to represent increased water content due to cell loss or gliosis, for example. Whether hypointensities on T2-weighted images, as found by Rutledge *et al*⁴ and in our case, strictly reflect iron deposition is still a matter of controversy. However, the finding of neostriatal hypointensities parallels pathological reports of increased iron content in this region in HD. Thus both signal hyper- and hypointensity can be explained from the underlying pathology of HD. In a case of Hallervorden-Spatz disease⁵ it has been shown in repeated MRI scans that the resulting signal intensity can change with the course of the disease. It was suggested that this reflects two subsequently and partially simultaneously developing pathological changes (neuronal degeneration versus iron deposition), each leading to opposite changes. Furthermore, signal loss on T2-weighted images is strongly dependent on magnetic field strength (all but one of Savoiardo's hypokinetic patients were studied at 0.5 Tesla). To date, this has not been studied systematically in HD.

Our findings differ from both the studies of Savoiardo and of Rutledge in that the caudate is spared and the globus pallidus shows signal abnormalities. The significance of this finding is not clear. One might expect the caudate, as the prime location of degenerative changes to be most likely to show abnormalities. However, since the globus pallidus is the main output site of striatal structures, signal extinctions might reflect transsynaptic changes.

Recent studies suggest that the phenotype of HD depends on which subset of striatal neurons are affected. Involvement of neurons projecting to the internal pallidum seem to be the cause of rigidity. Thus not the degree, but primarily the selectivity of degeneration determines the clinical form. A different pattern of signal changes in both forms would therefore be expected, in contrast to what we found.

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BOOK REVIEWS

A Handbook of Neuropsychological Assessment. Edited by J R CRAWFORD, D M PARKER and W W MCKINLAY. (Pp 454; Price: £40.00). 1992. Hove (UK), Lawrence Erlbaum Associates. ISBN 0-96377-210-2.

'Despite having become unfashionable in some branches of clinical psychology, assessment is not an issue that will go away'. So writes Edgar Miller early in this book and what follows is a vindication of this statement. Psychometricians are often undervalued and if they need to boost their self-esteem, a journey through this book should do wonders. It is a well-edited compilation of chapters from UK-based contributors which is fairly priced for its 450 pages.

It is divided into four sections; basic principles and general issues (intelligence and laterality; assessment of major psychological functions (memory, language, frontal lobe and visuo-perceptual dysfunction, unilateral neglect, reading; specific clinical disorders (stroke, head-injured, drug abuse, dementia); and specialised assessment, including a challenging chapter on computer-based assessment by Wilson and McMillan, which in illustrating the value of a mechanical impartial assessor for routine work is surely anticipating important developments.

I would have preferred to see a separate summary of each of the main instruments described, but as this book is written primarily for psychologists one can perhaps understand this omission. There is an excellent index and extensive referencing. The editors and contributors deserve congratulations for putting together a sound treatise which should help to put neuropsychological assessment in its proper place at the heart of clinical psychology.

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Neurologic Clinics: The Neurology of Trauma. November 1992. Vol. 10 No. 4. Guest Editor: RANDOLPH W EVANS MD. (Pp 1092; Price Not Indicated). 1992. London, WB Saunders Co. ISBN 0733-8619.

This series enjoys an enviable reputation for up to date accounts of topical subjects. The Neurology of Trauma justifies this accolade. Dr Evans has collected proficient essays on major contemporary issues mainly relating to head injury; spinal injuries were dealt with in August 1991. However, whiplash injury: a favourite source of controversy to both editor and reviewer is surveyed in detail.

The selection of topics is uneven; major head injuries receive scant attention, and more importantly to the neurologist and neurosurgeon, the late sequelae, life expectancy and prospects of rehabilitation are not mentioned. Similarly, one seeks in vain for recent data about variables, risk factors and expectations for late traumatic epilepsy.

There are useful sections on the surgery of nerve and plexus injuries. A variety of movement disorders are assessed by Goetz and Pappert, most thought to be unrelated to antecedent trauma. The approach here considers the possibility that *post hoc ergo propter hoc* arguments may be fallacious in certain cases, and that latent disease or concealed symptoms may appear to emerge after head injury without being caused thereby. By contrast, the well-referenced descriptions of mild head injury, reflex sympathetic dystrophy, and whiplash are less critical. In a technology-ridden age it need not surprise that many measurable aberrations of nervous function are demonstrable after minor traumata, but the possibility of their being silent accompaniments rather than the cause of symptoms and disability is often overlooked, or worse, ignored.

Merskey provides a characteristically absorbing essay on psychiatric aspects, pointing out that physical illness can cause emotional disturbances which in turn can evoke hysterical conversion. Head injury can produce obsessional states, mania, organic depression as well as schizophrenic illness.

It is odd to find chapters on electric shock, mountain sickness, space travel, and an intriguing description of the neurologist as expert witness, in an excellent finale. But, well done as they are, many would prefer more information and counsel about the prognosis and management of the more severe injuries of the nervous system.

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