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-