HAM/TSP attributable to blood transfusion

A multi-centric case-control study was carried out to clarify possible environmental factors related to the onset of HTLV-I-associated myelopathy/tropical spastic paraparesis (HAM/TSP) in northern Kyushu, Japan, which comprises one of the most prevalent areas of HTLV-I in the world.1 The frequencies of blood transfusion before the onset of HAM/TSP were 33% (6/18) among male patients and 18% (12/67) among female patients, which were significantly higher than 8% in males and 9% in females in the general population. The age-adjusted summary odds ratios with 95% confidence intervals were 7.0 (2.9–17.0) for males and 2.4 (1.3–4.5) for females. The percentages of population attributable risk1 of HAM/TSP attributable to transfusion were estimated to be approximately 29% (5–52) for males and 11% (1–21) for females.

The fraction of HAM/TSP attributable to transfusion after the introduction of blood screening for HTLV-I, in effect since 1986 in Japan, was definitely smaller than that before the programme. Our observations seemed compatible with a marked decline in newly diagnosed HAM/TSP patients after its introduction,2 which may be part of the benefit of blood screening. Neither smoking nor drinking was related to the risk of HAM/TSP.

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Suppression of motor neuron firing by transcranial magnetic stimulation in a patient with multiple sclerosis

Transcranial magnetic stimulation produces a short latency excitatory response in tonically active single motor units in small hand muscles.3 This is shown by a peristimulus time histogram, which cross-correlates motor unit firing times with the time of the stimulus. The excitatory response in the first dorsal interosseous muscle (FDI) occurs at 20–30 ms and is termed the primary peak (PP); in some motor units a secondary peak at 50–90 ms is also seen.4 PP is characteristically followed by a compensatory period of zero firing probability, which reflects the advancement of discharges that would have occurred shortly after PP had no stimulus been applied. Complete suppression of firing in response to transcranial magnetic stimulation in the absence of an excitatory peak has not been reported. The response of 21 single motor units to transcranial magnetic stimulation has been recorded in nine patients with multiple sclerosis (MS), to study the neural mechanism of symptoms caused by upper motor neuron lesions.5 Findings have suggested that spatial and temporal summation at the spinal motoneuron is impaired by a reduction in the velocity and synchronisation of central transmission. For healthy subjects, however, complete suppression of tonic activity in single motor units by transcranial magnetic stimulation was not observed.

The patient, a 48 year old female, initially presented in 1986 with paraparesis, weakness affecting the left arm, and diplopia. The diagnosis of MS was supported by the finding of oligoclonal bands in the CSF, enhancing white matter lesions on CT and by delayed VERS. She was subsequently free of symptoms for four years. She then re-presented with pyramidal weakness of the right arm [reducing the power of the right FDI to 4/5, MRC scale], increased tone in the left leg, extensor plantar responses and impaired joint position sense in the toes. During this clinical episode the response to transcranial magnetic stimulation of three voluntarily activated low threshold motor units from the weakened right FDI was recorded.

The patient was right handed and gave her informed consent to the experiments which were performed with the approval of the local ethics committee. The inducing current flowed in an anticlockwise direction through a circular (Novametrix 200) positioned tangentially at the vertex. Single MU potentials were recorded using fine concentric needle electrodes (Dantec type 13L58). The patient was asked to maintain repetitive motor unit firing and was aided by auditory and visual feedback of the motor unit discharge. Signals were amplified with a band pass of 32 Hz to 16 kHz (Medelec type MS8) and epochs of 250 + 250 ms relative to the stimulus were digitised at 10 kHz (Cambridge Electronic Design 1401) for subsequent analysis. Up to 120 stimuli were given for each motor unit, delivered at random with respect to the

Figure Peristimulus time histogram constructed from the discharge of a single tonicially active motor unit from FDI, recorded with a concentric needle electrode. Magnetic stimuli were delivered at time = 0, on 120 trials. There is a distinct period of zero firing probability with an onset latency of 34 ms and a duration of 47 ms. No excitatory peak is seen.