interaction, the presence of important additional non-HLA genetic factors. All these possibilities need to be investigated. Thus, we think that HLA studies in “ophthalmoplegia plus” should be usefully extended to test, in families, the linkage with genes on chromosome 6 other than HLA ones (that is Bf, glyoxalase, phosphoglucomutase polymorphism).

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Post-traumatic tremor and myoclonic jerking

Sir: Andrew et al.1 described myoclonic jerking in most of their cases of severe post-traumatic tremor. Patients with severe essential tremor, multiple sclerosis, head trauma and, more rarely, cases of hereditary neuropathy may show what clinically appears to be a combination of tremor and myoclonus. However, we believe that careful analysis may indicate that, in many instances, the jerks are actually an exaggeration of a beat of the on-going tremor. Such occurrence is illustrated by the following patient: a 13-year-old girl who had a head injury two years ago. She was in coma for a few days, but recovered over the following weeks. One year after the accident, neurological examination showed mild mental impairment, truncal ataxia, signs of precocious puberty and gross incoordination of the right arm. A severe, regular tremor of the right upper limb was present upon maintaining the arms outstretched, but sudden and large displacement of the arm, resembling myoclonic jerking often interrupted the rhythm of the tremor. Voluntary movements were greatly hampered by this dyskinesia. Electromyographic recording from the affected muscles revealed that the jerks were due to sudden increments in the amplitude of the EMG bursts, producing the tremor (fig). The patient was treated with propranolol (50 mg per/day) added to valproic acid (800 mg per/day). A marked improvement of the tremor and of motor control of the right arm was observed two weeks after reaching the optimal dosage of propranolol (80 mg daily). Placebo controlled studies showed the real efficacy of the treatment. Interestingly, both drugs were required to maintain control of the tremor.

A certain degree of confusion exists when referring to coarse, severe tremor affecting the limbs, which worsens during action. While English speaking authors often employ the term “red nucleus tremor”, Garcin2 introduced in France the name “hyperkinesie volitionelle” for this movement disorder. However, either term fails to portray any useful clue as to the aetiology, pathophysiology, pathology or treatment of this abnormal movement and probably should be discarded. Like Andrew et al.,1 one could refer to the type of dyskinesia, that is action tremor, action myoclonus and to the aetiology when this is known, until more precise information about the pathological basis of this tremor is obtained. Pharmacological treatment may obviate the need for stereotactic surgery in some patients. The combination of drugs acting at different levels of the peripheral and central nervous system seems a promising approach for the treatment of severe postural and action tremor.

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Figure EMG recording by surface electrodes of the right triceps and biceps muscles while the patient was keeping both arms outstretched. Rhythmic alternating EMG activity at a frequency of 4.5–5 Hz was seen. The arrows point to the bigger EMG bursts which coincided with larger myoclonic jerks observed clinically.

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