Ataxic arm movements after thalamotomy for Parkinsonian tremor

T E Kimber, B P Brophy, P D Thompson

Parkinsonian tremor is thought to be sustained by oscillatory activity in neuronal circuits linking the ventral lateral thalamic nuclei and the motor cortex. Some neurones in ventralis oralis posterior (Vop), which receives input from the pallidum, and ventralis intermedius (Vim), which receives input from the cerebellum, display rhythmic activity related to tremor at frequencies of 4–6 Hz, corresponding to the frequency of parkinsonian tremor. The precise relation of the rhythmic thalamic discharges and central oscillatory circuits remains unclear, though rhythmic thalamic activity does not appear to be driven by peripheral sensory feedback or efferent copy from the motor cortex. Stereotaxic lesions of Vim and Vop abolish Parkinsonian tremor in the contralateral arm in 80–90% of cases. The precise mechanisms responsible for tremor and the abolition of tremor after Vim thalamotomy remain the subject of debate. In particular, it is not known whether the critical lesion for abolition of tremor interrupts cerebellar or pallidal inputs to the thalamus, and supports the concept that abnormal cerebellar activity is an important contributor to the generation of tremor in Parkinson’s disease.

**RESULTS**

All six subjects had complete abolition of Parkinsonian tremor in the contralateral arm after Vim thalamotomy. In the first few postoperative days, all showed signs of past pointing and terminal intention tremor of the arm during the finger–nose movement (fig 1). Mild hypotonia of the limb was also evident on clinical examination. Two patients had mild ataxia of gait and the contralateral leg. The upper limb ataxia was asymptomatic in five of the six patients. No weakness, reflex change, or sensory impairment were evident in the limb contralateral to the thalamotomy, and no visual impairment occurred postoperatively. By the one to two months follow up, the ataxia had resolved in all but one patient. In that case, the ataxia in the immediate postoperative period was symptomatic.

**DISCUSSION**

Limb ataxia with past pointing and intention tremor, of varying degree, was a consistent postoperative finding after stereotaxic Vim thalamotomy for Parkinsonian tremor in the patients in this series. In all patients, ataxia accompanied successful abolition of limb tremor. The ataxia could not be accounted for by weakness, deafferentation or sensory loss, or visual field defects. The ataxic arm movements and hypotonia had the characteristics of cerebellar ataxia after an acute cerebellar injury. In the majority of patients in our study, contralateral limb ataxia resolved over the month following surgery. Transient limb ataxia after thalamotomy has been noted infrequently in previous studies of stereotaxic surgery for Parkinsonian tremor.

There is evidence implicating the cerebellum in the pathogenesis of tremor of various types. Essential tremor may...
Figure 1 Trace of finger trajectory during a movement of the arm from the nose to a target in a patient with severe parkinsonian tremor before thalamotomy (upper panel), the day following surgery (middle panel), and one month after surgery. In the upper panel a terminal postural tremor is seen in both directions of movement. In the immediate postoperative period there was past pointing with terminal corrective movements; this had settled when the patient was re-examined one month later.

Conclusions
The most parsimonious explanation for post-thalamotomy ataxia is interruption of cerebellothalamic connections. The implications of this finding fit well with recent observations on the pathophysiological basis of tremor and reinforce the notion that the cerebellum and cerebellothalamic connections are critical links in the oscillatory motor circuits responsible for Parkinsonian tremor.

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