SHORT REPORT

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 thamic 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 or the neuronal targets of these inputs in either Vop or Vim. The observation of postoperative ataxia led to the prospective examination of a series of patients undergoing Vim thalamotomy to see if this was a consistent finding and whether it correlated in any way with the surgical outcome.

METHODS

Subjects

We studied six consecutive patients undergoing Vim stereotaxic thalamotomy for severe Parkinsonian tremor refractory to medical treatment. The subjects (three men and three women) were aged between 55 and 75 years.

Thalamotomy

After attachment of the Leksell stereotaxic frame, computed tomography of the brain was undertaken to measure the three dimensional coordinates of the anterior and posterior commissures (AC, PC). The Vim nucleus was located using the construct of Guiot, 6 mm anterior to the PC, 2 mm above the plane of the AC–PC line, and approximately 11 mm lateral to the wall of the third ventricle. A monopolar electrode with a 2 mm long exposed tip was introduced into the target under local anaesthetic. The target was confirmed by noting the responses to electrical stimulation through the electrode. Pacring and disruption of tremor were observed at low frequency stimulation, and asterixis of the contralateral arm, paraesthesiae of the contralateral arm and face, and reduction of tremor were observed at high frequency stimulation. Lesions were then made by heating the electrode tip to between 70°C and 76°C. Postoperative confirmation of lesion location was obtained in all cases.

Analysis of unrestrained arm movements

Subjects underwent routine neurological examination and were videotaped performing a “finger–nose” arm movement before and after thalamotomy. The arm movement was unrestrained and based on the standard neurological examination of placing the index finger on the nose, then moving to the target and back to the nose. Frame by frame analysis of the videotapes was undertaken to trace the trajectory of the index finger during this task. Subjects were examined on three occasions: preoperatively, one or two days postoperatively, and between one and two months postoperatively.

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
be abolished after ipsilateral cerebellar stroke or injury.14 Cer- 
bebral synaptic activity on positron emission tomography (PET) is increased in subjects with essential tremor compared 
with normal subjects.2 PET is increased in subjects with essential tremor compared 
with normal subjects.2 Moreover, in patients with Parkinson’s 
disease, stimulation within the Vim nucleus at intensities suf- 
ficient to abolish tremor causes a reduction in cerebellar syn- 
aptic activity as measured by PET, whereas there was no 
change following stimulation at intensities that do not abolish 
tremor.15 This effect was observed in both medial cerebellar 
structures, and so could not be attributed to a reduction of 
proprioceptive input to one cerebellar hemisphere following 
cessation of tremor. The investigators concluded that high fre- 
quency Vim stimulation led to inhibition of a central oscillatory mechanism.16 To these observations we add the 
finding that successful abolition of tremor by Vim thalamot- 
omy is accompanied by transient cerebellar ataxia, suggesting 
that the surgical lesion interrupts cerebellar projections to 
Vim.

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 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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