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, 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.

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 contralateral arm and face, and reduction of tremor persisted. 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.

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 involve the cerebellar cortex and dentate nucleus.
be abolished after ipsilateral cerebellar stroke or injury.\(^1\) Cerebellar synaptic activity on positron emission tomography (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 sufficient to abolish tremor causes a reduction in cerebellar synaptic activity as measured by PET, whereas there was no change following stimulation at intensities that do not abolish tremor.\(^3\) 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 frequency Vim stimulation led to inhibition of a central oscillatory mechanism.\(^4\) To these observations we add the finding that successful abolition of tremor by Vim thalamotomy is accompanied by transient cerebellar ataxia, suggesting that the surgical lesion interrupts cerebellar projections to Vim.

We cannot exclude the possibility that abolition of tremor is related to inactivation of Wop neurons or pallidal afferents adjacent to Vim, and that transient ataxia is caused by interruption of cerebellar afferents to Vim, either directly or by perioperative oedema. These explanations, however, would not account for the physiological studies discussed above implicating the cerebellum in the genesis of tremor. Resolution of the limb ataxia over the weeks following thalamotomy is consistent with adaptation and compensation for the acute cerebellar deficit, as is well described after cerebellar injury.\(^5\)

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.

Authors’ affiliations

T E Kimber, P D Thompson, University Department of Medicine and Department of Neurology, Royal Adelaide Hospital and University of Adelaide, Adelaide, Australia

B P Brophy, Department of Neurosurgery, Royal Adelaide Hospital

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Correspondence to: Professor P D Thompson, University Department of Medicine, Royal Adelaide Hospital, North Terrace, Adelaide, South Australia 5000; philip.thompson@adelaide.edu.au

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