MATTERS ARISING

Thalamic stimulation for severe action tremor after lesion of the superior cerebellar peduncle

We read with interest the report by Geny *et al* concerning chronic high frequency thalamic stimulation in a patient with severe action tremor.¹ The postural and kinetic tremor was caused by a bleeding into a cavernous angioma located in the ipsilateral superior cerebellar peduncle. Although such tremors have been attributed repeatedly to cerebellar outflow lesions, neuropathological and neuroradiological confirmation has been limited. This case nicely demonstrates such a clinicopathological correlation. We recently identified lesions of the dentatothalamic pathways by MRI in 22 of 25 patients with severe kinetic post-traumatic tremor.²

We agree with the point that these tremors are particularly resistant to medical treatment and that functional stereotactic surgery should be considered for their treatment. We take issue, however, with the conclusion that thalamic stimulation "should be suggested to patients with this type of tremor".¹ So far, the question whether thalamotomy or thalamic stimulation is more advantageous in patients with unilateral tremor has not been settled. Disadvantages of deep brain stimulation include higher costs, limitations imposed by the hardware, the need of replacement of the batteries, and the natural resistance to implantation of a foreign body. We suggest refraining from decisive statements, particularly in patients with kinetic proximal tremors. Benabid *et al* have found satisfactory improvement after chronic thalamic stimulation only in 18% of their patients with proximal kinetic tremor.³ It is unclear whether differences in response of the tremors might be for technical reasons—that is, the use of a monopolar versus a quadripolar electrode for continuous high frequency stimulation. Only very few patients with severe proximal tremor have been reported to respond favourably to thalamic stimulation.^{4,5} The published findings on follow up do not exceed 2-5 years.

We still would consider stereotactic thalamotomy as a valuable choice of treatment. We recently published the long term results of a series of 35 patients with severe kinetic post-traumatic tremor who underwent stereotactic surgery in which lesions were placed in the basal oroventral thalamus and the zona incerta.⁶ On a 10-5 year mean long term follow up persistent improvement was noted in 88% of the patients with abolition of pronounced reduction of the tremor in 65%. It should be considered that thalamotomy in such patients may worsen pre-existing dysarthria. It is unclear as to whether chronic high frequency stimulation would be advantageous concerning this side effect. Dysarthria can also be induced by stimulation, and it has been seen in 15% of predisposed patients with different types of tremor in larger series during stimulation.³ Although this side effect is "reversible" by turning off the stimulator, it definitely interferes with possible improvement. We also want to mention that thalamic lesions to control kinetic tremor do not necessarily have to be large. The mean size of the coag-

ulative lesions in the zona incerta and the basal oroventral thalamus for kinetic post-traumatic tremor was 18 mm³ on long term MR examinations.⁶

Further controlled studies are needed before definite conclusions about the indications, merits, and drawbacks of alternative methods such as chronic stimulation and thalamotomy can be made, for patients with such rare conditions.

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Geny replies:

I appreciated the comments of Krauss and Mundinger. Their letter considers this important question: What are the respective places of chronic thalamic stimulation and thalamotomy in surgical treatment for movement disorders? In this case report, we did not have any ambition to show the superiority of thalamic stimulation over thalamotomy. The improvement in our patient only suggests that this technique is promising, all the more as no such results have been reported with thalamotomy in this kind of tremor. Krauss *et al* have recently reported excellent results of thalamotomy in a series of patients with post-traumatic tremor.¹ However, despite several convincing works published since the 1960s, thalamotomy has remained limited to rare expert centres. One of the main reasons for the limitation of the use of thalamotomy is the reluctance to cause permanent lesions of the CNS. Chronic stimulation offers the advantages of being reversible and adjustable if necessary. In a series of 13 patients with multiple sclerosis, we have shown that the possibility of modifying the electric variables and the site of stimulation after surgery was crucial for obtaining optimal results on tremor.² Those are the main reasons why chronic stimulation is particularly interesting in new indications similar to those in our patient but also for subthalamic nucleus or globus pallidum surgery in Parkinson's disease. Krauss and Mundinger raise the classic and important question of long term tolerance. This technique was developed seven years ago and no serious adverse effects leading to electrode removal have ever been reported to our

knowledge. The safety of this procedure has led to the stimulation of other sites in the brain in various diseases. We recently reported the spectacular effect of this motor cortex stimulation on severe action tremor.³ A multicentre study is in progress and should determine the tolerance and actual efficiency of chronic thalamic stimulation in parkinsonian and essential tremor. However, we agree with Krauss and Mundinger that it will be necessary to compare thalamotomy and chronic thalamic stimulation in a controlled study to specify the respective indications of these two types of surgery.

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Structural and functional changes in patients with motor neuron disease

I read with interest the letter of Cooper *et al* concerning structural or pathological changes in patients with motor neuron disease. However, I do not think that patients with this disease have normal frontal cortices.

I previously studied cases with motor neuron disease with dementia and without dementia^{2,3} and showed a correlation between reduced isotope uptake in the frontal lobe and neuropathological changes characterised by a moderate degree of neuron degeneration, invasions of reactive astrocytes, and mild spongiose states between the second and third layers in the frontal, temporal and central cortices. These neuropathological changes have been reported previously^{4,5} and our findings supported these from a neuroimaging point of view. These neuropathological changes were mild in patients with motor neuron disease without dementia and severe in patients with motor neuron disease with dementia. I, therefore, think that certain neuropathological and functional deficits exist even in the early stage of the disease process and these progress to neuropsychological deficits in patients with dementia.^{2,6}

Certainly, this should be prospectively tested, but I think that cognitive deficits in patients with motor neuron disease are caused by evident structural and pathological changes.

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