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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- 2 Krauss JK, Wakhloo AK, Nobbe F, Tränkle R, Mundinger F, Seeger W. MR pathological correlations of severe posttraumatic tremor. *Neurol Res* 1995;17:409-16.
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- 4 Broggi G, Brock S, Franzini A, Geminiani G. A case of posttraumatic tremor treated by chronic stimulation of the thalamus. *Mov Disord* 1993;8:206-8.
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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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- 1 Cooper PN, Siddons M, Mann DMA. Normal frontal cortex histology and immunohistochemistry in patients with motor neuron disease. *J Neurol Neurosurg Psychiatry* 1995;59:644.
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- 4 Davidson C. Amyotrophic lateral sclerosis:

original extent of the upper motor neuron lesion. *Archives of Neurology and Psychiatry* 1941;46:1039-56.

5 Brownwell B, Oppenheimer DR, Hughes JT. The central nervous system in motor neuron disease. *J Neurol Neurosurg Psychiatry* 1970;33:338-57.

6 Talbot PR, Goulding PJ, Lloyd JJ, Snowden JS, Neary D, Testa H. Inter-relation between "classic" motor neuron disease and frontotemporal dementia: neuropsychological and single photon emission computed tomography study. *J Neurol Neurosurg Psychiatry* 1995;58:541-7.

Cooper et al reply:

Dr Abe is making essentially the same point as we do. In those papers that he cites in which the pathological findings are described, the presence of extramotor cortical pathology in motor neuron disease is confined to patients in whom dementia had been clinically evident during life. It is well recognised that a proportion of patients with motor neuron disease without clinically evident dementia will show deficits in "frontal lobe" tasks when subjected to detailed neuropsychological tests, and may also demonstrate frontal cortical hypometabolism on functional imaging. If prospective studies confirm that the extramotor frontal cortex is normal in such patients, then this would suggest that the observed functional deficits are secondary to disease elsewhere.

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NOTICE

World Federation of Neurosurgical Societies: awards to young neurosurgeons

The World Federation of Neurosurgical Societies will give five awards to young neurosurgeons for the best papers submitted for presentation at the XIth International Congress of Neurosurgical Surgery to be held in Amsterdam, The Netherlands on 6-11 July 1997. This will be open to all neurosurgeons born after 31 December 1961. Each award will consist of an honorarium of US \$1500, a certificate, and complete waiver of registration fees along with accommodation for the Congress. The papers will be judged by a committee and must contain original, unpublished work on basic research or clinical studies related to neurosurgery.

Young neurosurgeons should submit eight copies of the manuscript (not more than 10 double spaced typewritten pages exclusive of figures and tables) to:

Albert L Rhoton Jr, MD, Chairman
WFNS Young Neurosurgeons
Committee, Department of Neurological
Surgery, University of Florida Medical
Center, PO Box 100265; 1600 SW
Archer Road, Gainesville, Florida
32610-0265.

The submission should be accompanied

by a supporting letter from the head of the candidate's neurosurgical department. The last date for submission is 1 October 1996.

BOOK REVIEWS

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the United Kingdom and for members of the British Forces Overseas, but overseas customers should add £2 per item for postage and packing. Payment can be made by cheque in sterling drawn on a United Kingdom bank, or by credit card (Mastercard, Visa or American Express) stating card number, expiry date, and your full name.

Negative Motor Phenomena. (Advances in Neurology Vol. 67). Edited by STANLEY FAHN, MARK HALLETT, HANS O LUDERS and C DAVID MARSDEN. (Pp 416; £88.50). Published by Lippincott-Raven, New York 1995. ISBN 0-7817-0264-X.

Positive motor phenomena are well known to all practising physicians—the Jacksonian fit, chorea and indeed most movement disorders are examples of such phenomena. However, fewer clinicians will be aware of negative motor phenomena, which form the theme of this book, although most of us will have experience, when entering the twilight zone of lecture induced somnolence!

Perhaps the most classic examples of negative motor phenomena are asterixis and postanoxic myoclonus—both of which are characterised by periods of CNS inhibition of muscular activity. In addition there are other conditions that clearly belong under this rubric of negative motor phenomena because of this inhibitory CNS outflow, including cataplexy and sleep paralysis and atonic seizures, although other conditions discussed in this book are slightly more difficult to accommodate in this definition, for example hyperekplexia and freezing in Parkinson's disease.

This book approaches the subject of negative motor phenomena via five main sections, beginning with clinical syndromes and then moving through the possible mechanisms according to anatomical locus (cortex, brainstem, and spinal cord) before finally discussing the pharmacology and treatment of atonic seizures and myoclonus. The book then concludes with a final chapter by James Lance which is a superb précis of all these sections. Thus the book starts and finishes in the clinical domain having travelled up and down the pathways of clinical research, much of which depends on the use of magnetic stimulation and the back averaging of EEGs relative to the abnormal movement. Although this approach with these sectional topics is clearly useful, an alternative and perhaps more preferable format would have been to start with anatomy before discussing the physiology and the clinical pathology of the relevant motor systems. This approach has the merit of defining the anatomical substrate which facilitates our understanding of

the normal physiology of the motor networks that ultimately underlie the CNS driven negative motor phenomena. Thus having defined the anatomy and physiology, the clinical conditions characteristic of negative motor phenomena can be discussed and the unnecessary separation of topics, as occurs in this book, can be avoided.

However, despite my reservations on the structure of the book, the content is quite outstanding, with many chapters serving to summarise complex topics with great clarity. Such examples include the chapters on startle phenomena (Peter Brown) and possible spinal cord mechanisms of negative motor phenomena (Peter Ashby). These two chapters further highlight another strength of this book, namely its ability to discuss clinical neurology and experimental neuroscience in equal depth and with equal authority. Occasionally chapters end in a disappointing fashion when no summary is given whilst some are rather too speculative and others tend to be repetitive.

Overall this book is to be recommended, as it is well-written with much to excite and interest the neurologist and neuroscientist with an interest in movement disorders. For example neurologists may be surprised to know that asterixis, whilst being seen most commonly in metabolic encephalopathies, can also be seen with focal lesions in a number of CNS sites, whilst neuroscientists may be disappointed to hear that there are at least 26 distinct reticulospinal nuclei or sub-nuclei that project to the spinal cord. Finally though I come back to what I really admire about this book and that is its unashamed use of experimental data and clinical findings in a combined effort to understand not only how the CNS can produce negative motor phenomena in the pathological state, but how this is achieved or not achieved in the normal physiological situation.

ROGER BARKER

Brain Ischemia. Basic Concepts and Clinical Relevance. Edited by LOUIS R CAPLAN. (Pp 380; price DM270.00.) 1995. London: Springer Verlag. ISBN 3-540-19850-4/0-387-19850-4.

Balliere's Clinical Neurology: Cerebrovascular Disease. Edited by V HACHINSKI. (Pp 381; price £30.00.) 1995. London: Baillière Tindall. ISBN 0-7020-2005-2.

Stroke Therapy. Edited by MARC FISHER. (Pp 490; price £70.00.) 1995. Oxford: Butterworth Heinemann. ISBN 0-7506-9575-7.

We all know that strokes are common, even those working in neurology units where they are a relative rarity. We all know that serious treatments for stroke are on the way but the cry of those looking after stroke patients is "When, oh when, is all this fancy science going to make the slightest difference to our patients?" Well, it must be agreed that the problem is considerably more difficult than finding a cure for tuberculosis, which took half a century, and the clinicians need to be patient. We can still be optimistic after reading these three books that the considerable knowledge being acquired about the processes of stroke, since it was recognised that cerebral infarction is a process and not an event, will bring forth clinical fruit. In the United Kingdom the big hurdle after that will be the logistics to bring the resultant