were obtained in five of the six patients. In particular, in two patients (2 and 5) the reduction in spasticity led to a significant improvement in fine movements of the dominant hand, so that writing became possible. Reduced spasticity in one patient (4) enabled hand held utilisation of walking devices; in another two patients (3 and 6), it made for independence in dressing and eating. Clinical benefit started after two to three days and peaked in about three weeks. Three patients did not need a second injection; the other three patients were reinjected after varying times. Patient 1 had right biceps and brachioradialis muscle injection in the first session and a reinjection of the biceps muscle after 1.5 months; spasticity was reduced, mainly in the wrist and finger joints. The follow up of this patient was interrupted after four months. Patient 1 is still in a vegetative state. Three patients (1, 2, and 5) had pain arising from muscle spasms, which was relieved by BTX-A treatment.

These preliminary data show that BTX-A treatment is effective in reducing spasticity in selected patients with focal upper limb muscular tone disorders secondary to traumatic brain injuries. Spasticity, as measured by means of the modified Ashworth scale, was reduced and range of movement increased in five of the six patients. Greater facility in nursing care and reduced disability were found in all the subjects. The effects of a single injection were long lasting and only three patients needed a second injection. The treatment in the early phases of the muscular tone disorders seemed to be a critical factor. The pain secondary to muscle spasms was relieved by BTX-A treatment in our patients; the pain arising from capsular and synovial tissues, mainly at the elbow joint, was unaffected.

Previous studies have shown comparable results in spasticity secondary to stroke, multiple sclerosis, degenerative diseases, and cerebral palsy. Few reports are available for patients with traumatic brain injury, dealing only with isolated cases or small samples of patients with lower limb spasticity.²⁻³ To our knowledge, only one paper deals with a large population of patients with traumatic brain injuries⁴; most of these patients (12 out of 21) were chronically affected, (that is, spasticity had lasted for more than one year), and one of the acute patients had experienced a penetrating brain injury. The results obtained in the remaining eight acute patients were similar to ours.

In our patients, the changes were not strictly limited to the muscles injected; the treatment of proximal arm muscles invariably had a positive effect also on finger movement. A local toxin diffusion or an indirect permissive action could be hypothesised; however, action mechanisms other than the peripheral one for BTX-A cannot be ruled out.⁵

The treatment was completely safe and none of the patients complained of weakness due to the BTX-A injection.

Several points deserve further study: the choice of muscles, the number of injections, the dilution of the drug, and, in particular, the selection of patients. Furthermore, it is very important to define a clear goal for therapy for each patient. Double blind, placebo controlled, multicentre trials are warranted to provide answers for the aforementioned questions.

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CORRESPONDENCE

Erdheim-Chester disease and slowly progressive cerebellar dysfunction

In February 1995, Fukuzawa *et al* described a woman with Erdheim-Chester disease, a rare histiocytic disorder characterised by lipid laden cells infiltrating long bones, who presented with ataxia and diabetes insipidus.¹ They thought that this was the first reported case of Erdheim-Chester disease showing cerebellar symptoms and deep cerebellar lesions on MRI. We recently encountered a similar case, but on reviewing the literature we found that a few cases with ataxia and neurological signs had been reported previously. Erdheim and Chester's first case was thought to have had multiple sclerosis for seven years because of ataxia and ocular signs.² Subsequently a case was described in the German language by Kujat *et al*: a 62 year old man who presented with ataxia and bulbar symptoms.³ Brain MRI showed multiple brain stem, diencephalon, and cerebellar lesions. Another case was described by Smith *et al*: a 58 year old man with brain stem signs had a biopsy taken from the tibia which showed the characteristic lipid laden cells, and MRI of the brain showed multiple lesions.⁴

So, by contrast with the recognised pattern of skeletal involvement in Erdheim-Chester disease, the involvement of the CNS has variable presentation.

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- 1 Fukuzawa T, Tsukishima E, Sasake H, et al. Erdheim-Chester disease and slowly progressive cerebellar dysfunction. *J Neurol Neurosurg Psychiatry* 1995;58:238-40.
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Fuzakawa replies:

We appreciate the comments made by Bohlega. We agree that Erdheim-Chester disease can show variable CNS involvement,¹⁻³ and we also suspect that the involvement of the CNS may be relatively common during the entire clinical course of the disease. However, we had not found the case report in German by Kujat *et al*³ when we submitted our case report. In our article, we described a 59 year old woman with ECD who showed a four year clinical course of slowly progressive cerebellar symptoms, the clinical features of which suggested cerebellar degeneration and were different from those of previously reported cases of Erdheim-Chester disease showing ataxia.¹⁻⁴ The symptoms of diabetes insipidus had disappeared spontaneously when we first saw her, although they reappeared three years later. There was no definitive abnormality on CT, whereas MRI showed abnormal hyperintensity on T2 weighted studies in both dentate nuclear regions. Conversely, the brain MRIs of the previously reported patients with ECD who showed neurological signs disclosed multiple enhanced lesions in the CNS.²⁻⁴ We recently found another case report of a slowly progressive cerebellar syndrome associated with Erdheim-Chester disease, a 69 year old man presented with a one year history of progressive gait ataxia, dysarthria, dysphagia, fatigue, and weight loss.⁵ Unlike our patient, however, his brain MRI disclosed multiple white matter enhanced lesions in both cerebral hemispheres, pons, medulla, and brachium pontis. Furthermore, some cases with another form of histiocytosis—multifocal eosinophilic granuloma (MEG)—were reported to have presented as progressive cerebellar syndrome,⁶⁻⁸ and we previously reported a case with MEG showing progressive cerebellar and brainstem symptoms and multiple enhanced lesions on MRI.⁹ Therefore, these enhanced lesions on MRI may be a common finding of CNS infiltration of histiocytes. From the view of neuroimaging, our patient with ECD presented in this *Journal* is unique, although we could not clarify whether the MRI appearances were due to histiocytic infiltration. However, Erdheim-Chester disease should be included in the differential diagnosis of patients with otherwise unexplained slowly progressive cerebellar symptoms, and skeletal surveys are also indicated for these patients.

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Histological surprise: callosal tuberculoma presenting as malignant glioma

I read with great interest the recent case report by Fath-Ordoubadi *et al.*¹ This is an excellent report and the authors are to be congratulated for their good work. They have clearly demonstrated their clinical wisdom. They confirmed the diagnosis through a biopsy as there is no specific clinical or radiological criterion for the diagnosis of tuberculoma of the brain.

These patients usually present with symptoms and signs suggesting an intracranial space occupying lesion. There is generally no extracranial clinical features to suggest the diagnosis of active pulmonary or extrapulmonary tuberculosis nor any definite history of previous pulmonary infection.² Biopsy of the mass lesion remains the scientific ideal for establishing the diagnosis as in the reported case. This should and must be the approach in parts of the world where tuberculosis is rare, although the procedure is invasive and is not without risk.^{3,4}

We treated a group of 24 consecutive patients including 14 reported during a 10 year period without resorting to biopsy. The treatment was based on a strict selection criteria and the response to a 12 week therapeutic test. Cerebral angiography was done only in the first 10 patients who were admitted to hospital for this purpose. It disclosed avascularity of the mass lesion in all of them. Treatment was generally carried out as outpatients. All patients responded to the therapeutic test and all except one were treated successfully with triple antituberculous drug therapy, for a continued total period of 18 months. A failure of medical treatment occurred in one patient due to non-compliance. In this patient the lesion became smaller and more dense, turning into a calcified 4x2 cm sized irregular mass which needed excision for intractable epilepsy. Another patient needed a ventriculoperitoneal shunt for the treatment of an associated obstructive hydrocephalus. There were 18 patients with solitary and six with multiple lesions, the size of the lesions varying from 3x2 cm to 7.5x6 cm. Even the largest tuberculoma resolved to a tiny calcified nodule needing no further treatment after 18 months.

It is our practice to perform serial CT in these patients. Each of our patients underwent an average of five scans during the course of the investigation (plain and contrast scans), treatment (contrast scan only, after three, six, and 18 months of treatment), and follow up (contrast scan only, one year after treatment was discontinued). No patient needed treatment beyond 18 months due to the resolution of the tuberculomas as shown in the serial CT. Complete resolution of the lesion(s) or its resolution to a tiny calcified nodule was taken as the criterion to stop therapy. Follow up scans showed no recurrence. Response to antituberculous therapy can be assessed clinically but the resolution of a tuberculoma can be judged only by CT. No scan was done in this reported patient during the course of the treatment. I would like to know the reason for continuing antitubercu-

lous treatment for 27 months and the criteria used to stop therapy.

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Lane replies:

We thank Choudhury for his kind comments. Our patient also underwent serial CT during the course of his antituberculous treatment. The lesion appeared quiescent some time before therapy was finally stopped; we erred on the side of caution. The final decision to stop treatment was empirical and we would generally support Choudhury's view that 18 months of treatment should be sufficient.

NOTICE

Second Advanced Course

The Use of Magnetic Resonance Techniques in Multiple Sclerosis

22-23 May 1998

Ospedale San Raffaele, Milan, Italy

Aim: to provide updated lectures from experienced researchers on the use of conventional and non-conventional MR techniques to diagnose multiple sclerosis and to monitor its evolution, either natural or modified by treatment

Main topics:

- 1 MR techniques to monitor clinical trials
- 2 Magnetisation transfer imaging

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BOOK REVIEW

Minimally Invasive Therapy of The Brain. Edited by ANTONIO DE SALLES and ROBERT LUFKIN. (Pp368; DM 375.00.) Published by Thieme Medical Publishers, New York. 1997. ISBN 3-13-106541-9.

The ability of the healthcare systems in the United States to generate new ideas, new technology, and new resources for clinical care never fails to astound those of us who have spent most of our working lives in a cash strapped British system, underresourced and overwhelmed with patients and pathology. The innovative potential of the American system is partly a reflection of sheer numbers of doctors involved in medical care in the United States. In neurosurgery alone there are more neurosurgeons in New York City than there are in the whole of the United Kingdom. However, another factor, and perhaps a more important one in this context, is that this large concentration of medical talent operates in a true market and must compete for market share. One of the effects of the application of market forces to the practice of neurology and neurosurgery in America is the proliferation of books of this type. It is perhaps symptomatic that in the introduction the authors, who are both members of the UCLA Medical School, advertise the courses which they run and on which the material for this book is based. The introduction also contains an email address, a web site address, and the names and addresses of suppliers of CD roms of the content of the book. As practitioners on both sides of the Atlantic have discovered, one of the best methods of advertising in medicine is to publish. As in other markets, for such medical advertising to be successful it is usually necessary for the advertising material to be obviously "state of the art", "glossy", and the title and content should be appropriately "sexy". This book fulfils these criteria on all counts. Minimally invasive therapy is currently "sexy" and is thus a field of endeavour ripe for "niche" marketing. However, despite its "state of the art" pretensions, in the neurosciences minimally invasive therapy has a long and illustrious history. The stereotaxic technique was first described by Horsley and Clarke in 1908 and was first applied to patients in 1947 by Spiegel and Wycis. Similarly, neuroendoscopy was first performed by Dandy in 1922. Although minimally invasive techniques in neurosciences have been around for a very long time indeed; what is new is the marriage of these techniques to modern imaging and computing technology. If the application of information technology in other fields is anything to go by an explosion of new developments in this field can be expected. Although there is little which is completely new in the book, it does provide a useful overview of the various areas where such techniques are beginning to be applied and it will be interesting to see how a second