Davie et al reply:

We thank Ray Chaudhuri and colleagues for their comments regarding our recent publication in this Journal. They cite preliminary results from a spectroscopic study localised to the putamen, carried out in a group of patients with idiopathic Parkinson's disease. We have published the abstract of their work and it would seem that there are significant methodological differences between our in vivo study of spectroscopic localisation, and methods of measurement which make direct comparison difficult.

Ray Chaudhuri et al are correct to quote the study of Holthouse et al. They have used the only large spectroscopic study of idiopathic Parkinson's disease to date, in which spectra were collected from the basal ganglia in 151 patients with idiopathic Parkinson's disease. Their findings do not exclude the possibility of localised pathology. They found no significant reduction in the NAA/creatine ratio compared with controls. They noted a decrease in the NAA/choline ratio in the older patients with idiopathic Parkinson's disease. They concluded that their findings may indicate a slight decrease in NAA or alternatively increased concentrations of choline and creatine in this subgroup.

This highlights the difficulty of interpreting the metabolite ratios as quoted by Ray Chaudhuri et al, as it assumes that at least one of these metabolites remains unchanged in controls and is present in reduced levels in Parkinson's disease.

In our recent study we have used a semi-quantitative method to overcome this problem. To date we have found a significant reduction in apparent NAA concentration collected from a spectroscopic volume localised to the putamen and globus pallidus in only one of nine patients with idiopathic Parkinson's disease. Whereas we agree that our findings do not exclude the possibility of neuronal loss or dysfunction occurring within the putamen alone in idiopathic Parkinson's disease, this needs to be confirmed by the demonstration of an absolute reduction of NAA from this structure.

C DAVID W BUSST


Dunne replies:

Bhakta and his colleagues, in accord with our report, have successfully treated patients with chronic spasticity. The mean duration of spasticity for our patients was 10 (range 5-45) years, and the degree of benefit from botulinum toxin A treatment did not correlate with the duration of spasticity. We agree that in some patients clinical differentiation between active and fixed or passive contracture can be difficult. In this situation the use of needle EMG is extremely useful, as prominent activation is present if muscles are contributing substantially to the abnormal posture. We grade the degree of motor unit potential activation with a five point ordinal scale (0 = no activation to 4 = full interference pattern), correlating this with limb posture and resistance to passive stretch.

We agree that EMG localisation may not be required to achieve a satisfactory result, but we are inclined to agree with Holthouse et al, however, the optimal delivery technique is unknown and will require randomised trials. We find that EMG is a useful adjunct to physical examination by assisting in the accurate localisation of the palpable muscle.

We have found a mean improvement of range of passive joint movement of 28° (95% confidence intervals 21°-36°), and applying a threshold change of 10° or 15° does not alter our results.

J W DUNNE

Vascular ataxic hemiparesis: a re-evaluation

Prompted by the unexpectedly high rate of a potential embolic source in patients with the clinical syndrome of ataxic hemiparesis in the recent study by Moultin et al, we studied the frequency of a potential cardioembolic source, and internal carotid artery stenosis >50% ipsilateral to a pre-existing hemispheric infarct, in patients presenting with the syndrome of vascular ataxic hemiparesis (AH) or dysarthria-clumsy hand syndrome (DCHS). Patients had been registered as described in an earlier report. We reviewed 47 (5%) cases of AH/DCHS; 27 had a lacunar infarct on CT, two a territorial infarct, whereas 16 had no specific CT lesion. There were no patients with other specific lesions on CT, such as haemorrhage. Obviously, the chance of a specific lesion other than a small deep infarct was low in our series. In a prior analysis of the first 350 patients AH/DCHS was a more accurate predictor of a small deep infarct than pure motor syndrome or sensory motor syndrome. Twenty four (51%) of our cases had hypertension, whereas six (13%) had a potential cardioembolic stroke source. Four of 35 (11%) patients who had carotid ultrasound studies had an ipsilateral stenosis > 50%. Percentages were similar for patients with or without lesions on CT. Considering separately, the frequency of these two sources of potential embolism are rather low; however, almost a quarter of our 47 cases had either of these two features. Our data, therefore, concur with those of Moulin et al, in that among patients presenting with a syndrome of “cerebellar type” ataxia the number with a potential
Treatment of chronic limb spasticity with botulinum toxin A.

B B Bhakta, J A Cozens, M A Chamberlain and J M Bamford

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