*Davie et al reply:*

We thank Ray Chaudhuri and colleagues for their comments regarding our recent publication in this *Journal*.[1] They cite preliminary results from a spectroscopic study localised to the putamen, carried out in a group of patients with idiopathic Parkinson's disease.[2] We have studied the published abstract of their work and it would seem that there are significant methodological differences between their study and our own in terms of data acquisition, spectroscopic localisation, and methods of measurement which make direct comparison difficult.

Ray Chaudhuri *et al* are correct to quote the recent paper by Holhouser *et al*.[3] This is 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, 80–90% of the volume having been localised to the striatum. 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*,[2] as it assumes that at least one of these metabolites remains unchanged in concentration (a hypothesis as yet unproved in idiopathic 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.[4] 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.

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## Treatment of chronic limb spasticity with botulinum toxin A

We read with interest the short report by Dunne *et al*[1] regarding the use of botulinum toxin (BTX-A) for limb spasticity. Our experience also suggests that botulinum toxin can be a safe and effective treatment for spasticity in selected patients. We would, however, like to raise the following points:

Not only does this treatment have a use in early spasticity but also where the limb is held in a flexed posture for many months or even years. In these patients one might be justified in thinking that immobility of a spastic limb is due to irreversible changes in soft tissue and that inappropriate muscle activity is no longer a relevant contributing factor. We have treated 15 such patients after hemiplegic stroke in whom severe flexor spasticity of the forearm caused difficulty with hand hygiene (in some cases fingernails traumatising the palmar skin). These patients could be deemed to have major contractures, yet 80% responded to botulinum toxin. This raises the interesting question of how to identify patients in this category who might benefit from treatment.

We note that needle EMG, was used to confirm the degree of spasticity in a muscle. We are interested as to which EMG criteria were used to assess spasticity?

In our experience of injecting biceps brachii, flexor digitorum superficialis and profundus, flexor carpi ulnaris, hamstrings, hip adductors, and gastrocnemius, EMG localisation is not required to achieve a satisfactory therapeutic effect. In vitro studies of rabbit longissimus dorsi have shown that diffusion of botulinum toxin occurs up to 45 mm from the injection site and that the toxin can cross fascial planes.[2] In the light of this we are interested in the authors' assertion that needle electromyographic guidance for BTX-A injection "enhances the accuracy of injections" and wondered whether EMG was used only to locate deep muscles, or was it also used to define the injection site within a given muscle.

We appreciate that change in range of motion of joints after treatment may be relatively large but we would suggest a higher threshold be used for difference in goniometer measurements as an indicator for real change. Although a change of 5° has previously been suggested as a criterion of improvement,[3] we agree with the opinion of Gajdosik and Bohannon[4] that this threshold is too low. Even in healthy subjects, within observer errors up to 5% have been reported for hip goniometry.[5]

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*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 0·5–45) 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 we have found that 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 effect; 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 active 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.

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## 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 Moulin *et al*,[1] we studied the frequency of a potential cardioembolic source, and internal carotid artery stenosis > 50% ipsilateral to a presumed hemispheric infarct, in patients presenting with the syndrome of ataxic hemiparesis (AH) or dysarthria-clumsy hand syndrome (DCHS). Patients had been registered as described in an earlier report.[2] Among our first 859 patients we registered 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.[2] 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. Considered 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*,[1] in that among patients presenting with a syndrome of "cerebellar type" ataxia the number with a potential