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 published the studied abstract of their work and it would seem that there are significant methodological differences between 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 Holshouser et al.3 They have published 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 and 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 in interpreting the metabolite ratios as quoted by Ray Chaudhuri et al., as it appears that at least one of these metabolites remains unchanged in the striatum.4,5 From spectra collected from a spectroscopic volume localised to the putamen and globus pallidus in only one of nine patients with idiopathic Parkinson's disease.6 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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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 Moult et al., 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.7,8 There were 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 two of these sources of potential embolism is very 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 ataxia with a potential