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. We have published the studied abstraction of their work and it would seem that there are significant methodological differences between their 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 studies of Hollmayer et al.1,2 We are aware of 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.3 They found that their findings do not exclude the possibility of neuronal loss and 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.4

C A DAVIE
D H MILLER
G J BARKER
A J LEES


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, we studied the frequency of a potential cardioembolic source, and internal carotid artery stenosis > 50% ipsilateral to a previous 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.1 We found an embolic source in 47 (95%) 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. Forty 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

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

Not only does this treatment have a use in early spasticity 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 fingermarks 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 hallucis, 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 increases 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,2 we agree with the opinion of Gadjoisk and Bohnannon3 that this threshold is too low. Even well-trained observers, with or without observer errors up to 5° have been reported for hip goniometry.4

RIPON B BHATKA
J ALASTAIR COZENS
M ANNE CHAMBERLAIN
Rheumatology and Rehabilitation Research Unit
University of Leeds, UK
JOHN M BAMFORD
Department of Neurology
St James University Hospital
Leeds, UK

Dunne replies:
Bhatka and his colleagues, in accord with our report, have successively reported 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 use EMG guidance and believe 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 (0-4 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 significant 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 muscles.

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.

1 J W DUNNE Neurophysiology Laboratory, Royal Perth Hospital PO Box X215, GPO Perth, Western Australia 6001, Australia
source of embolism is substantial. However, a potential cardioembolic source, large vessel disease, is an absence of carotid compression does not exclude the presence of small vessel disease as the cause of a lacunar infarct presenting with AH/DCHS. Patients could hernie the stroke, which is often seen with small vessel disease, one of which becomes symptomatic first. The fact that most silent brain infarcts in patients with AH/DCHS with a small deep hemispheric infarct on brain imaging and a >70% ipsilateral internal carotid artery stenosis. The absence of carotid disease is not a sufficient factor. However, drug use can be a cause of small vessel disease, and the absence of carotid disease is not a sufficient factor. However, drug use can be a cause of small vessel disease, and the presence of another factor is necessary.3

Wodarz et al.16171920 present a case of musical hallucinations attributed to basal ganglia calcifications. The patient, however, satisfies only one of the four criteria for determining a neurological as opposed to epileptic or otological cause for musical hallucinations.21 There was no evidence for epileptic activity, but there was deafness and ataxia. The patient had no cerebellar symptoms, but no clear cerebellar symptoms, with apparently no check if this ataxia was partly or wholly of vestibular origin. She had chronic hypoaeropharyngitis, yet no mention was made of any drugs she was taking.

These criteria were set up on the general scientific principle that if most cases of a phenomenon are caused by a known factor (in this case drug use), one should be very cautious before concluding that the remaining cases are due to a second, quite different factor (brain disease), rather than being variants of the first cause.

Wodarz et al. offer a simplistic version of the ototogic theory, which, not surprisingly, they then dismiss. It is clear that hearing loss itself is not a sufficient factor, and indeed drugs can induce musical hallucinations in patients with no underlying neurology. It seems that the extra factor is an endolymphatic hydrops, as seen in incipient Meniere's disease. This can cause fluctuating or progressive hearing loss, hyperacusis, or no deafness but with auditory ataxia, or even tinnitus. This seems to be the mechanism whereby a wide range of drugs induce musical hallucinations in normal subjects; deaf ears are even more suggestive of cochlear hydrops (slight or unilateral or asymmetric or low tone losses, pancochlear losses, etc.). Other factors which could trigger an acute hydrops in an ear include anything likely to reduce perilymphatic pressure, such as dehydration, hypotension, weight loss, or, in this case, electrolyte imbalance.

Wodarz et al.16171920 claim that musical hallucinations have not been reported before in post-surgical hypoaeropharyngitis. There is, however, a case3 distinguished by the richness and intensity of hallucinations, which is very instructive. As neoplasia showed absolutely no brain pathology. (There are many other reports that I have not checked.) She heard music and bells ringing and talked of composing a symphony! In other hallucinations she felt herself being thrown through the air or down a hole. Her sight was very poor and she had vivid visions thought to have been of retinal origin. In other patients sheionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsionsion