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 Holzheimer *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.

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

1 Davie CA, Pirtosek Z, Barker GJ, Kingsley DPE, Miller DH, Lees AJ. Magnetic resonance spectroscopic study of parkinsonism related to boxing. *J Neurol Neurosurg Psychiatry* 1995;58:688–91.

2 Ray Chaudhuri K, Lemmens G, Williams S, Ellis C, Leigh PN. Proton magnetic resonance spectroscopy study of striatal changes in Parkinson's disease. *J Neurol* 1995;242 (suppl 2):S15.

3 Holzheimer BA, Komu M, Moller HE, Zijlmans J, Kolem H, Hinshaw Jr DB, *et al*. Localised proton NMR spectroscopy in the striatum of patients with idiopathic Parkinson's disease: a multicentre pilot study. *Magn Reson Med* 1995;33:589–94.

4 Davie CA, Wenning GK, Barker GJ, Tofts PS, Kendall BE, Quinn N, *et al*. Differentiation of multiple system atrophy from idiopathic Parkinson's disease using proton magnetic resonance spectroscopy. *Ann Neurol* 1995; 37:204–10.

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 Bohannon⁴ that this threshold is too low. Even in healthy subjects, within observer errors up to 5° have been reported for hip goniometry.⁵

BIPIN B BHAKTA
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*

1 Dunne JW, Heye N, Dunne SL. Treatment of chronic limb spasticity with botulinum toxin A. *J Neurol Neurosurg Psychiatry* 1995;58:232–5.

2 Borodic GE, Pearce LB, Ferrante R. Therapeutic botulinum toxin: histologic effects and diffusion properties. In: DasGupta BR, ed. *Botulinum and tetanus neurotoxins*. New York: Plenum Press, 1993:623–45.

3 Boone DC, Azen SP, Lin C-M, Spence C, Baron C, Lee L. Reliability of goniometric measurements. *Phys Ther* 1978;58:1355–60.

4 Gajdosik RL, Bohannon RW. Clinical measurement of range of motion. Review of goniometry emphasising reliability and validity. *Phys Ther* 1987;67:1867–72.

5 Ellis MI, Stowe J. The hip. *Clin Rheum Dis* 1982;8:655–75.

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.

J W DUNNE
*Neurophysiology Laboratory,
Royal Perth Hospital,
PO Box X2213 GPO Perth,
Western Australia 6001, Australia*

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

source of embolism is substantial. However, a potential cardioembolic source, large vessel disease, or even the absence of hypertension do not exclude the presence of small vessel disease as the cause of a lacunar infarct presenting with AH/DCHS. Patients could have more than one type of vascular disease, one of which becomes symptomatic first. The fact that most silent brain infarcts in patients with a cardioembolic territorial infarct are small lesions also point at this possibility.³ I wonder whether Moulin *et al*¹ would recommend carotid endarterectomy in patients with AH/DCHS with a small deep hemispheric infarct on brain imaging and a >70% ipsilateral internal carotid artery stenosis? I know neurologists who, considering a carotid lesion a coincidental feature, don't even perform carotid ultrasound in patients with lacunar stroke.

J LODDER
Department of Neurology,
University Hospital Maastricht,
PO Box 5800,
NL-6202 AZ Maastricht,
The Netherlands

- 1 Moulin Th, Bogousslavsky J, Chopard JL, Ghika J, Crépin-Leblond Th, Martin V, Maeder P. Vascular ataxic hemiparesis: a re-evaluation. *J Neurol Neurosurg Psychiatry* 1995;58:422-7.
- 2 Lodder J, Bamford J, Kappelle J, Boiten J. What causes false clinical prediction of small deep infarcts? *Stroke* 1994;25:86-91.
- 3 Boon A, Lodder J, Heuts-van Raak L, Kessels F. Silent brain infarcts in 755 consecutive patients with a first-ever supratentorial ischemic stroke. Relationship with index-stroke subtype, vascular risk factors, and mortality. *Stroke* 1994;25:23-84.

Moulin and Bogousslavsky reply:

We are delighted that the data from Maastricht confirm our study—that is, that potential sources of embolism to the brain are not uncommon in patients with acute stroke presenting as ataxic hemiparesis.

We also agree with the point that the exact aetiology of brain infarct is presumed rather than proven in most instances, but it may be presented also from another perspective: the presence of a small deep infarct is commonly compatible with underlying small vessel disease, although there is no test which can show this *in vivo*; however, this assumption does not rule out the possibility that it may also be embolic, especially if a source of embolism is indeed shown. The argument of the coexistence of cardioembolic territorial infarct with silent small deep infarcts¹ (Lodder's ref³), could also be presented the other way around to support the concept that small deep infarcts may often be embolic in origin.

The answer to the question asked by Lodder about the performance of carotid endarterectomy in patients with deep small infarct and ipsilateral >70% carotid stenosis seems to lie in the randomised trials of carotid endarterectomy, in which these patients were included and contributed to the global superiority of surgery over medical treatment alone. Thus the neurologists mentioned by Lodder are not following the scientific data, which showed the usefulness of carotid surgery in symptomatic patients with >70% carotid stenosis.

- 1 Boon A, Lodder J, Heuts-van Raak L, Kessels F. Silent brain infarcts in 755 consecutive patients with a first-ever supratentorial ischemic stroke. Relationship with index-stroke subtype, vascular risk factors, and mortality. *Stroke* 1994;25:23-84.

Do musical hallucinations have a neurological cause?

Wodarz *et al*¹ 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.² There was no evidence for epileptic activity, but there was deafness and tinnitus. The patient had "cerebellar" ataxia, but no other cerebellar symptoms, with apparently no check if this ataxia was partly or wholly of vestibular origin. She had chronic hypoparathyroidism, yet no mention was made of any drugs she was taking.

These criteria² were set up on the general scientific principle that if most cases of a phenomenon are caused by a known factor (in this case ear disease), 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*¹ offer a simplistic version of the otogenic 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 people with normal hearing.³ 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 audiosensitivity, or tinnitus, or vertigo. This seems to be the mechanism whereby a wide range of drugs induce musical hallucinations in normal subjects; deaf ears are even more susceptible to drugs.² In their own case Wodarz *et al*¹ attribute deafness to presbycusis. This is an overused default diagnosis, which should be confined to patients with smooth high tone symmetric losses. Their patient had mild pancochlear perceptive hearing loss, worse on the left, far more consistent with at least some hydrops component. A third of patients with hypoparathyroidism⁴ had inner ear hearing loss, not presbycusis but 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 a deaf or normal ear include anything likely to reduce perilymphatic pressure, such as dehydration, hypotension, weight loss,⁵ or, as in this case,¹ electrolyte imbalance.

Wodarz *et al*¹ claim that musical hallucinations have not been reported before in postsurgical hypoparathyroidism. There is, however, a case⁶ distinguished by the richness and intensity of hallucinations, which is very instructive as necropsy showed absolutely no brain pathology. (There are many other reports that I have not checked.) She⁶ 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 another patient the main hallucinations were a feeling of flying through the air and oscillations in the head. No mention was made of otological examinations. An autobiographical account, however, by a social worker⁶ confirms that vestibular and auditory hallucinations are prominent features of psychosis. She felt herself suspended in mid-air or upside down, or suddenly being moved.

These hallucinations could be checked by having a light on after dark and by opening her eyes. Her balance was very poor. She had brief Menieriform attacks comprising nausea, a whirling vibration in her head like an egg beater, deafness, and mental confusion. She could tolerate the cramps and other symptoms, but most of all she was in terror of these vibrations, later described as loud buzzing, roaring in the ears, noises in the head, or pulsations. The auditory sensation depended on the rapidity of the vibration.

It only needs a couple of cases with consistent neurological lesions but no deafness to completely sink the otogenic theory^{2,3} and so reduce the causal possibilities for musical hallucinations. My previous appeal² for such a case has been unsuccessful, so it is reasonable to assume that there is no case in the medical literature. Wodarz *et al*¹ state that musical hallucinations can occur with brain-stem lesions, but give no reference. Please could they cite one which includes patients without cochlear or neural deafness? I appeal again to neurologists to publish new non-deaf cases of musical hallucinations.

A G GORDON
32 Love Walk,
London SE5 8AD, UK

- 1 Wodarz N, Becker T, Deckert J. Musical hallucinations associated with post-thyroidectomy hypoparathyroidism and symmetric basal ganglia calcifications. *J Neurol Neurosurg Psychiatry* 1995;58:763-4.
- 2 Gordon AG. Musical hallucinations. *Neurology* 1994;44:986-7.
- 3 Gordon AG. Benzodiazepines and the ear—tinnitus, hallucinations and schizophrenia. *Can J Psychiatry* 1993;38:156-7.
- 4 Ikeda K, Kobiyashi T, Kusakari J, Takasaka T, Yumita S, Furukawa Y. Sensorineural hearing loss associated with hypoparathyroidism. *Laryngoscope* 1987;97:1075-9.
- 5 Schoelly ML, Heuscher JE. Contribution à l'étude des psychoses tétaniques. *Monatsschrift für Psychiatrie und Neurologie* 1950;119:141-55.
- 6 Dda CF. Psychological states resulting from parathyroid deficiency. *Journal of Abnormal and Social Psychology* 1939;34:481-96.

Wodarz *et al* reply:

Gordon's interesting comments and the additional three patients with postsurgical hypoparathyroidism and associated psychosis are very much appreciated. The musical hallucinations in these patients, however, were associated with various other psychiatric symptoms such as delirious state, epileptic seizures, and paranoid ideation. On the contrary, our patient, in terms of psychopathological syndromes, presented with isolated musical hallucinosis. As in his previous comments to other papers Gordon attributes musical hallucinations to a peripheral = otogenic mechanism.¹ His support for this hypothesis in our patient is, however, based on some misunderstandings.²

(1) We did, in fact, report the drugs given to our patient during inpatient treatment. In the six months preceding the hallucinosis she received the equivalent of 2.3 mmol Ca²⁺ intravenously only when symptoms of tetany occurred (once or twice per month). This might well result in an acute ear hydrops, as suggested by Gordon. As the musical hallucinosis disappeared, however, after addition of oral dihydroxycholesterol plus oral and intravenous Ca²⁺, a drug induced