We read Treatment by the reduction of neuronal findings only in our localisation 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., as it appears that at least one of these metabolites remains unchanged in the cerebrospinal fluid (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 the 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 A DAVIE DH MILL 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 Moulton et al., we studied the frequency of a potential cardioembolic source, and internal carotid artery stenosis >50% ipsilateral to a prerolandic 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. We studied 30 of the first 35 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. Twenty four (51%) of our cases had hypertension, whereas six (13%) had a potential cardioembolic stroke source. Forty five (9%1%) patients who had carotid ultrasound studies had an ipsilateral stenosis > 50%. Percentages were similar for patients with or without lesions on CT. Considering Positron Emission Tomography, the high prevalence 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 Moulton et al., 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, is the absence of carotid stenosis and ipsilateral >70% carotid stenosis does not exclude the presence of small vessel disease as the cause of a lacunar infarct presenting with AH/DCHS. Patients could harbor both, and the relationship of either 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 to this possibility. I wonder whether Moulin et al1 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. McElduff et al3 made the assumption that it may be a coincidence that in Lodder’s ref3, in 3 of the coexistence of carotid stenosis and ipsilateral >70% carotid stenosis, there is no evidence for epileptic activity, but there was deafness and uninnutition. The patient had bilateral alexia, but no other symptoms, with apparently no check if this alexia was partly or wholly of vestibular origin. She had chronic hypoparathyroidism, 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 (or syndrome), this case could be very cautiously before concluding that the remaining cases are due to a second, quite different factor (brain disease), rather than being variants of the first cause.

Moulin et al1 presented 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 individuals with multiple sclerosis. 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 deafness but with auditory hallucinations, or both.

Wodarz et al5 presented a study of 70% ipsilateral carotid artery stenosis? Stroke 1994;25:86-91. In 70% of these patients, with a first-ever supratentorial ischemic stroke, the association with ischemic stroke subtype, vascular risk factors, and mortality. Stroke 1994;25:23-84.

Do musical hallucinations have a neuro- logical cause?

This question has been asked by Fox and Saberi in their 1993 paper.1 This question has been posed by the neurologists who, considering a carotid lesion a coincidental feature, don’t even perform carotid ultrasound in patients with lacunar stroke.

Wodarz et al1 present a case of musical hallucinations attributed to basal ganglia calcifications. The patient, however, satisfies only one of the 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 uninnutition. The patient had bilateral alexia, but no other cerebellar symptoms, with apparently no check if this alexia 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 (or syndrome), this case could be very cautiously 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 al5 presented 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 individuals with multiple sclerosis. 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 deafness but with auditory hallucinations, or both. This seems to be the mechanism whereby a wide range of drugs induce musical hallucinations in normal subjects; deaf ears are even more responsive to drugs.2

If we assume a selective loss of the basal ganglia, this would explain the symptoms such as sudden deafness, orotogenic source, Deafness and tinnitus. (brain hydrops, which musical hallucinations.

My previous paper1 for such a case has been unsuccessful, so it is reasonable to assume that there is no case in the medical literature. Wodarz et al5 state that musical hallucinations can occur with brainstem lesions, but give no reference. Please could they cite one which includes patients without cochlear or neural deafness? I appeal again to neurologists but would like to see new notes of cases of musical hallucinations. A G GORDON J L, London S5 5AD, UK

Wodarz et al reply: Gordon’s interesting comments and the additional three patients with posturgical hypoparathyroidism and associated psychosis are very much appreciated. The musical hallucinations in these patients, however, were associated with various other psychiatric symptoms such as delusional 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 comment to other papers Gordon attributes musical hallucinations to a peripheral otogenic mechanism.1 His support for this hypothesis is not surprising as, 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 hallucinosis, as confirmed by Gordon. As the musical hallucinosis disappeared, however, after addition of oral dihydroxyatsterol plus oral and intravenous Ca2+, a drug induced...
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