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Sustained levodopa therapy in tardive dyskinesia

Sir: The pathophysiology of tardive dyskinesias is still poorly understood, but striatal post-synaptic dopamine receptor hypersensitivity may be implicated.¹ Permanent discontinuation of the offending neuroleptic offers the best hope of relief, but the patients' mental state often precludes this. Tetrabenazine is the most effective drug treatment, but side-effects including sedation, depression, Parkinson's syndrome and akathisia are common. The longterm administration of levodopa to rodents has recently been shown to attenuate the behavioural and biochemical features of dopaminergic hypersensitivity.^{2,3} Promising results have also been reported in tardive dyskinesia giving sustained levodopa treatment² or small doses of dopamine receptor agonists.⁴ In view of these findings we have been encouraged to extend this approach to the treatment of patients with irreversible, persistent dyskinesias no longer receiving neuroleptics.

Seven patients (six female, one male) with moderate or severe tardive dyskinesia agreed to participate. Their mean age was 69 years (range 53-93) and the mean duration of involuntary movements was 7 years (range 3-12). Neuroleptics had been given for a mean period of 9.2 years (range 3-30) for schizophrenia, save for three with chronic dyspepsia, agoraphobia and depression respectively. Their conditions were static and their movement disorders comprised a bucco-linguo-masticatory syndrome in seven, additional limb chorea in five and torticollis in one. Two had co-existent akathisia, but none had Parkin-

son's disease. With one exception all had discontinued neuroleptics for at least one year (mean 4 yr) before the trial. Baseline clinical assessments were made by two independent observers using the AIM scale⁵ and dyskinesia was recorded simultaneously on video tape. Levodopa 300 mg daily in combination with benserazide was then gradually introduced (Madopar 125, 1 capsule 8 hourly) and the patients assessed at 14 day intervals by the same observers. After a minimum of 12 weeks sustained therapy, patients were re-filmed and the levodopa then discontinued abruptly. Follow-up observation continued for six months with AIM scale scoring.

An initial aggravation of the dyskinesias was seen in one patient following levodopa introduction, but otherwise no significant changes in dyskinesia severity occurred at any stage of the trial.

These disappointing results do not compare favourably with those obtained by Bjørndal and colleagues who reported modest improvement in drug-free patients following one month's levodopa therapy.⁶ Benefit has also been claimed with chronic levodopa in patients still receiving neuroleptics⁷ or in those who have just stopped them.⁸ Casey *et al.*⁹ however, using very large doses of levodopa in combination with benserazide for treatment periods of 8 weeks failed to produce benefit in five neuroleptic treated schizophrenics with tardive dyskinesia. In contrast to other studies we also failed to demonstrate an initial increase in dyskinesia following levodopa introduction.⁷ Further studies using dopamine receptor agonists and large doses of levodopa in this refractory group of incapacities are now under way.

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Giant aneurysm of the petrous portion of the carotid artery

Sir: Aneurysms of the internal carotid artery extracranially are rare; those arising from the petrous segment are particularly unusual. A survey of the literature showed approximately 30 reported cases.¹⁻¹⁹

A 28-year-old woman had been complaining of severe left ear pain, tinnitus and hearing loss for approximately six months. The pain was continuous and irradiated towards the face, the retromastoid region and sometimes down to her neck. At ENT consultation a vascular mass was found in the left middle ear. An attempted biopsy resulted in massive bleeding controlled by packing the ear. She was then referred to the neurosurgical department with the diagnosis of glomus tumour. Besides a left sensorineural deafness, neurological examination was normal. Both carotid pulsates normally and no bruit was heard about the head and neck. Skull radiographs demonstrated a large area of bone erosion in the left petrous pyramid extending anteriorly into the sphenoid sinus. A sclerotic ring could be noticed around the lytic lesion, suggesting a chronic process. Left carotid arteriography revealed a large (1.5 × 2.5 × 5 cm.) aneurysm located in the petrous segment of the carotid artery projecting laterally into the middle ear, with normal intracranial arborisation. Right carotid arteriography with left carotid compression showed excellent cross filling of the left anterior cerebral and middle cerebral arteries, without aneurysm opacification.