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Accepted 10 February 1983

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.

Fig (a) Mentovertebral tomograph showing a large oval area of erosion with sclerosis in the margins. (arrows) (b) Left anteroposterior common carotid subtraction angiography demonstrating a giant aneurysm in the petrous bone. (c) Left common carotid angiography, mentovertebral view, showing the anterior extension of the aneurysm.

Under local anaesthesia the left common carotid artery first was clamped for 30 minutes without neurological deficits and then was double ligated. Recovery was uneventful and the patient was discharged pain free but still deaf.

Such aneurysms can present with spontaneous and repeated episodes of otorrhagia^{3,7,12,17,18} or epistaxis via the Eustachian tube.^{1,7,10} The Eustachian tube is located anteriorly and parallel to the horizontal portion of the carotid canal. In a microanatomic study Wayne *et al*²⁰ showed that the Eustachian tube was separated from the carotid artery by a thin layer of bone in 94% of the 50 cadavers examined and by mucosa only in the 6% remaining. In 56% the bone was very thin, measuring 0.1 mm. to 0.3 mm. the average thickness being 0.8 mm. Busby *et al*¹⁰ in a pathological study showed an aneurysm that perforated the bone septa and ruptured into the Eustachian tube. Such bleeding episodes often are dramatic and transfusion of several units of blood are required to reverse the hypotension and anaemia.^{1,3,7,10,18} Death due to exsanguination occurred in Busby's case.¹⁰ Massive bleeding can also be the result of attempted biopsy for presumed glomus tumours.^{9,11-13,19}

When the aneurysm is located medially it may cause only a Vth nerve palsy,^{4,15} or a combination of deafness and paralysis of the abducens.^{8,10} Guirguis and Tadros⁵ reported a patient with deafness and involvement of the 6th, 7th, 9th, 10th cranial nerves. Gupta and colleagues¹⁷ reported a 8-year-old girl with involvement

of the 6th, 7th, 9th, 10th cranial nerves. Both Harrison *et al*,⁶ and Drake¹⁵ presented cases with dysfunction of the trigeminal nerve alone. Ear pain and deafness as in our patient was reported by Allen.⁹

Several causes of aneurysms of the petrous portion of the carotid artery have been reported. Head trauma is the most common.^{4,7,10,14,19} Congenital aneurysms were reported by several authors.^{5,6,11,12,15,18} Others¹⁸ suggested that chronic middle ear infection may lead to aneurysm formation due to destruction of the external layers of the carotid artery. One case occurred after mastoidectomy.² In the present case there is no history of head trauma, temporal bone surgery or otitis. Probably the aneurysm was congenital.

Most of the reports cases were successfully treated by common carotid ligation.^{1,3,4,6,14,17,18} Harrison *et al*,⁶ and Sarwar¹⁵ reported gradual occlusion. Others preferred a one step ligation.^{1,3,4,18,19} Acute or gradual internal carotid ligation with an extracranial bypass procedure also has been suggested by Gelber & Sundt²¹ and Drake.¹⁶

We are grateful to Dan Marchesin Ph.D for editorial assistance.

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Accepted 18 December 1982

Sudden hearing loss and facial palsy at the contralateral side following acoustic tumour removal

Sir: We wish to report an unusual and unexpected complication following the removal of an acoustic neuroma.

A 37-year-old woman was admitted for excision of a large right sided acoustic neuroma. Computed tomographic (CT) scanning of the brain showed in addition to the cerebellopontine mass, three small right sided meningiomas, in the frontopolar, falcial and supratentorial regions, without any mass effect. The acoustic neuroma was completely removed through a suboccipital approach while the patient was in a left lateral recumbent position. After the operation, there was complete hearing loss on the right but preserved function of the facial nerve. The postoperative recovery was excellent. The patient received chloramphenicol and high doses of prednisolone. The second day after operation she was allowed to sit erect and suddenly developed complete deafness accompanied by vertigo and left sided peripheral facial palsy. Beside this, neurological examination disclosed horizontal nystagmus to the right and deviation to the left with the Romberg test. Audiometric testing showed a complete hearing loss on the right and an almost complete sensorineural hearing loss on the left. Brain stem auditory evoked responses were absent on both sides. The stapedius reflex was absent on the left. On electronystagmography a spontaneous nystagmus to the right was recorded and caloric testing revealed a marked diminished excitability of the left labyrinth. CT scanning of the brain, nine days after the operation, showed the operative changes and the three meningiomas, but no other abnormalities. Corticoid therapy was resumed for some days without any improvement.

During the following months there was no recovery of this left sided deficit.

The patient described here developed a sudden sensorineural hearing loss, loss of vestibular function and a peripheral facial paralysis on the non operated side, the second day after uncomplicated removal of an acoustic neuroma. This complication has to our knowledge not been reported previously. However, recently three cases of sudden contralateral hearing loss after operation in acoustic neuroma patients have been described.¹ In two of them, the hearing loss was of the perceptive type with the lesion situated respectively retrocochlear and cochlear, appearing the seventh and fourth day after the intervention. There were no other neurological signs. The deficit slowly improved, but the aetiology remained obscure. In the third case, the hearing loss resulted from a serious middle ear effusion. The first two cases show some similarity with our patient, except that they had no facial nerve involvement. The exact mechanism responsible for the event in our case could not be established. However, the lesion must be localised on both the left cochleovestibular nerves and/or inner ear and the left facial nerve. A possible explanation, in our opinion the most acceptable, is that there has been an occlusion of the left internal auditory artery. A postoperative shift of the brain stem to the right, possibly delayed by the presence of oedema at the operative site, could have been resulted in a stretching and subsequent thrombosis of this vessel. This theory is supported by the sudden onset of the deficit, and the fact that this artery supplies a part of both nerves and the inner ear. Occlusion of the internal auditory artery is indeed a well known cause of sudden deafness and loss of vestibular function,² but it is generally not considered as a cause of peripheral facial palsy. Nevertheless, the part of the facial nerve situated in the internal auditory canal is supplied by this artery.³ However, there exists a great variability in the vascularisation of this structures. Usually there is more than one internal auditory artery,⁴ and the facial nerve is for the most part supplied by branches of the occipital and middle meningeal arteries and by the anterior inferior cerebellar artery.³ Therefore, if the internal auditory artery (or arteries) is (are) occluded, the facial nerve function will be spared in most cases. Consequently, this vascular hypothesis offers also a possible explanation for the cases only affected by a sudden contralateral hearing loss following acoustic tumour

removal. These cases are probably not restricted to the two patients mentioned above. Our case was but a variation of a possible complication of cerebellopontine mass removal that, although rare, merits further attention. A better understanding of these events could lead to preventive measures to avoid such a dramatic complication.

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