admission he was noted to have poor memory, fine horizontal nystagmus, bilateral restriction of upward gaze and an inability to walk heel to toe. Fundoscopy was normal as was the rest of his neurological and general examination. A repeat of CT revealed a midline mass lesion obliterating most of the third ventricle and growing into the left lateral ventricle. The lateral ventricles were more prominent than on the previous scan. Histological examination of a small fragment of the tumour following burr-hole biopsy revealed the features of an astrocytoma. The fragment was considered too small for grading.

The patient initially responded to dexamethasone and local radiotherapy and in August 1987 a further CT head scan again revealed the midline mass lesion but indicated some shrinkage. Following this, his condition gradually deteriorated and he died in November 1988.

There seems little doubt that this man's exercise induced diplopia resulted from his midline astrocytoma. At presentation this tumour was presumably producing a small elevation of the cerebrospinal fluid pressure as demonstrated by the possible enlargement of the ventricular system seen on CT scan, and the pressure recorded at lumber puncture. As the illness progressed, raised intracranial pressure became more pronounced. It seems likely that the onset of diplopia during exercise was due to a further elevation of intracranial pressure produced by exertion.

Exercise induced diplopia is not a well recognised presenting feature of midline cerebral tumours although disturbances of gaze are not unusual. A "Medline" computer search back to 1966 using "exercise," "sports" and "diplopia" as key words revealed no publications on the association.

The case does provide circumstantial evidence that, in humans, exercise tends to elevate intracranial pressure. It is possible, however, that this pressure elevation only occurs when pathological abnormalities exist.

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Reference

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Effect of tropatepine, an anticholinergic drug, on regional cerebral blood flow in patients with Parkinson's disease.

Sir: Despite the recent discovery of alterations in numerous central neurotransmitters and peptides in Parkinson's disease, only dopaminergic or anticholinergic agents have been found to be active therapeutic agents. Previous studies have shown that acute administration of dopaminergic drugs (levodopa3 or bromocriptine4) induced a significant increase in rCBF in Parkinson's disease. However, as far as we know, no study has investigated the effects of anticholinergic drugs on rCBF in Parkinsonians. In this study, we investigated the influence of tropatepine, an anticholinergic agent commonly used in the treatment of Parkinson's disease.

Fifteen patients with idiopathic Parkinson's disease (10 men and five women, mean age: 63.5, SD 8 1 years), stage II to IV on the modified Hoehn and Yahr's staging were included in this study. All these patients were regularly treated with levodopa (plus dopa decarboxylase inhibitor). Tropatepine hydrochloride was injected acutely by intramuscular route at a dose of 10 mg, which is the dose commonly used in clinical practice to counteract the extrapyramidal effects of neuroleptic drugs. A first rCBF measurement was made with SPECT (Tomatic 64, Medimatic, Copenhagen) (for description see2) at time 0 (that is, at 11 a.m. before drug injection). The second measurement was performed at time 90 min, that is at the mean maximal peak plasma level of tropatepine. PCO2 was measured and different regions of interest (ROIs) were determined as previously described.1 The changes were evaluated before and after tropatepine by paired t test and the level of significance was p < 0.05.

There was no significant difference in PCO2 before and after tropatepine (39.2, SD 3.1 versus 39.9, SD 3.1 mmHg). Under basal conditions in Parkinsonians, mean rCBF was 50.6, SD 11.4 ml/100 g/min which is a normal value for such a population under our experimental conditions.2

Acute administration of tropatepine did not significantly change total CBF (50.7, SD 10.7 ml/100 g/min) or rCBF in any ROI. The effect on extrapyramidal symptoms were not investigated and no side effect was observed except in one patient in stage II who developed 60 min after tropatepine a confusional state which spontaneously disappeared after 4 hours.

The study allows two conclusions to be made. First, it confirms our previous findings4 that, under our experimental conditions, the total CBF of non-demented Parkinsonians (especially regularly treated ones) does not exhibit major changes in comparison with normal subjects. We failed to find any decrease in the frontal pattern, unlike Bes et al.1

Secondly, our results demonstrate that an anticholinergic drug, in contrast to levodopa3 or bromocriptine4, failed to change total or rCBF in Parkinsonians. In fact a physiological role for cholinergic mechanisms in the regulation of CBF has been suggested: binding studies showed the presence of muscarinic cholinergic receptor sites in cerebral blood vessels5 and cholinergic agents (like acetylcholine or physostigmine) were found to increase CBF. Sorennin et al.6 suggested that, in rabbits, the cholinergic cerebral vasodilatation does not depend on cerebral metabolic activation and involved muscarinic receptors located beyond the blood brain barrier. Few studies have investigated the effects of anticholinergic drugs in man: using individual detectors and the [133Xe] inhalation technique, Honer et al.7 recently found that high doses of another anticholinergic drug, scopolamine reduced global CBF and especially frontal cortex perfusion in normal subjects. Although we used a therapeutic and effective dose (see the patient with the induced side effect of tropatepine, we were unable to find similar results in Parkinsonians.

References
Benign recurrent multiple mononeuropathy in Wegener's granulomatosis

Sir: Neurological involvement in Wegener’s granulomatosis is common, occurring in 26% of patients in a review of 374 cases. In the more localised form, midline granuloma, 12% of 125 patients had neurological involvement but only one had a peripheral neuropathy. We wish to report a patient with a 5 year history of recurrent multiple mononeuropathy prior to histological diagnosis of Wegener’s granulomatosis.

A 62 year old woman presented in March 1983 with a two month history of severe right fronto-temporal and temporal headache. Sinus radiographs were consistent with sinusitis. She did not improve with antibiotics and ENT opinion was that there was no significant sinus disease. ESR was 100 mm in 1 hour, temporal artery biopsy specimen was normal; she was referred to the neurology department and treated with prednisolone with resolution of her headache.

Four months later on prednisolone 20 mgs per day, she developed double vision due to an almost complete right orbital ophthalmoplegia without ptosis. ESR was 46 mm in 1 hour; and her signs resolved after 5 days of prednisolone 100 mgs per day which subsequently was gradually reduced.

In November 1983, while on prednisolone 12.5 mgs per day, she developed a left vocal cord paralysis and a chest radiograph showed elevation of the left hemidiaphragm. ESR was 20 mm in 1 hour: mediastinal tomography, CT thorax and neck, bronchography, sputum cytology, thyroid and bone isotope scans were all normal. The gradual reduction in prednisolone dosage was continued and her voice was much better one year later. She remained on 2.5 mgs prednisolone daily until March 1986 when she developed numbness and nagging pain in the left side of her face and forehead. Sensation over the left infraorbital nerve was impaired, ESR was 40 mm in 1 hour and sinus radiographs showed an opaque left anterolateral maxillary antrum. Left maxillary anterolateral examination revealed an absent medial nasal wall which was attributed to previous surgery; antral washings contained polymorphs only with no malignant cells. Her symptoms improved with prednisolone 15 mgs per day. CT of her naso-pharynx revealed no other abnormality. In January 1987 on 7.5 mgs of prednisolone she developed sudden visual impairment of the left eye with pain around the orbit. Examination revealed constriction peripheral vision in the left eye with visual acuity of N48, and a left relative afferent pupillary defect. There was full recovery after one week on 30 mgs prednisolone per day, and the dose was gradually reduced.

In June 1987 because of osteoporosis the steroids were gradually reduced and discontinued six months later. In August 1988 she complained of left maxillary pain and an oro-antral fistula with erosion of all the surfaces of the maxillary antrum was found. Histology of the fistula lining showed multinucleated giant cells and necrosis surrounded by pallisading of histiocytes with necrotising vasculitis of small arteries, characteristics of Wegener’s granulomatosis. She was started on cyclophosphamide 75 mgs per day and reducing doses of prednisolone, and remains well. At no stage has there been any renal or respiratory system disorder.

This woman had a 5 year history of recurrent cranial and peripheral nerve lesions including the left optic, left infraorbital, left phrenic and left recurrent laryngeal nerves before the diagnosis of Wegener’s granulomatosis was made when an oro-antral fistula developed. The initial diagnosis was based on the severe headache, high ESR and the response to steroids was giant cell arteritis and the subsequent external ophthalmoplegia was attributed to this disorder also.

The correct diagnosis probably could have been made by biopsy of antral mucosa in March 1986. Any cranial nerve may be affected by Wegener’s granulomatosis but ocular involvement is frequent; the optic neuropathy was unusual in the rapid response to a modest dose of steroids and was presumably due to compression by contiguous inflammatory granulomas. The phrenic and recurrent laryngeal nerves were presumably affected by a vasculitis of the vasa nervorum and the external ophthalmoplegia may be explained by orbital muscle rather than third nerve involvement since ptosis and pupilary abnormality were absent.

This case report serves to emphasise that Wegener’s granulomatosis may be a rather indolent process and that, in the presence of any cranial or peripheral neuropathy, symptoms of sinus disease should be fully investigated.

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References

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Progressive systemic sclerosis presenting as a case of trigeminal neuropathy

Sir: A 58 year old shipyard welder first noticed numbness over the right lower lip in...