

Letter

SIR,—We read with great interest the article "Dementia paralytica: deterioration from communicating hydrocephalus".¹ I made a similar observation in 1976,² on the basis of which I stated that communicating hydrocephalus in patients with dementia paralytica may not be a rare event and can be an important factor in deterioration of the patient.

We studied a 62 year old man with progressive mental deterioration of about eight months duration, dysarthric speech, pyramidal tract signs and gait apraxia. He had positive fluorescent treponemal absorption test (FTA-ABS) and inflammatory changes in the cerebrospinal fluid with opening pressure of 37 cm H₂O. Pneumoencephalography revealed a pattern of low pressure hydrocephalus with convexity block, gross ventricular dilatation and a corpus callosum angle of 111°. Isotope cisternography showed early ventricular entry with subsequent intraventricular stasis for 48 hours and absence of parasagittal radioactivity.

The patient was treated with ampicillin 500 mg three times a day by mouth for a period of three months to no avail. Four months prior to his death an atrio-ventricular shunt was performed and the patient improved slightly for a short while, but rapid deterioration and death followed.

At autopsy typical gross and microscopic changes of dementia paralytica was found. The leptomeninges over both cerebral convexities were thickened and adherent to the surface of the brain. The cerebral ventricles were mildly dilated, but less so than expected from the enlargement of the ventricles at the time of pneumoencephalography. There was moderate loss of the nerve cells in the cerebral cortex accompanied by moderate astrogliosis and microgliosis. Numerous lymphocytes and few plasma cells infiltrated the leptomeninges and Virchow-Robin perivascular spaces. Ependymitis granularis of the cerebral ventricles was prominent, and cervical hypertrophic pachymeningitis also was found.

We interpreted the failure of surgery

as due to insufficient treatment with penicillin, and our case shows that communicating hydrocephalus in dementia paralytica does not rule out persistence of active parenchymal inflammation.

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References

- 1 Giménez-Roldán S, Benito C, Martín M. Dementia paralytica: deterioration from communicating hydrocephalus. *J Neurol Neurosurg Psychiatry* 1979; 42:501-8.
- 2 Tavčar D, Pogačar S, eds. Clinical pathological conference. Insufficient doses of penicillin as a cause of unsuccessful treatment. *Zdrav Vestn* 1976; 45:477-81.

Book reviews

Optic Neuritis and its Differential Diagnosis by GD Perkin and F Clifford Rose (pp 283; £20) Oxford University Press: 1979.

There is an absolute flood of monographs of varying standard at the moment on a variety of neurological topics, but this one on optic neuritis is well worth reading. The book in effect comprises three parts. The first and perhaps the most important is a review of the authors' considerable experience of patients with optic neuritis. They have assembled 170 consecutive cases from their practice and extensively analysed the data with respect to the entire clinical picture and prognosis, paying some attention to the ultimate development of multiple sclerosis. The second part comprises a review of the literature and is overall a valuable résumé. The authors are refreshingly honest in indicating which of the numerous references they have not sighted, a feature that some of the authors of the larger American

neuro-ophthalmological texts should adopt if they are not to misquote European literature.

The third part deals with differential diagnosis in four separate chapters. It is this section that is a little weak and unbalanced. For example there is almost as much on mycotic and parasitic affections simulating optic neuritis as on vascular disease and some sections are deficient in references, although this latter criticism cannot be made of an excellent and exhaustive, if not exhausting, chapter on toxic involvement of the optic nerve. Other minor criticisms are the lack of illustrations of investigative methods, again chiefly in the sections on differential diagnosis. There are no computerised tomogram or fluorescein angiogram illustrations, surely a "must" in 1979, and the evoked potential section is a little thin. Finally the numerous visual field illustrations of the authors' patients have been assessed by the method of Friedmann while those from the literature are the more conventional type. This is a pity as it makes comparison a little difficult.

Overall this book is a useful addition to the library of the neurologist, ophthalmologist and general physician and it contains the best review of a large series of patients with optic neuritis in the current literature.

PETER RUDGE

Sleep Research Edited by RG Priest, A Pletscher and J Ward (pp 224 £7.95) MTP Press: Lancaster 1979.

This book results from an international symposium in September 1978 sponsored by a drug company, bringing together pharmacologists and clinicians interested in sleep research. The book is mainly about benzodiazepines and although these have come a long way in the last two decades, their mode of action remains obscure despite improved methods of drug testing in sleep disturbances and much greater knowledge of the biochemistry, neurophysiology and anatomy of neurohumoral transmitters.