

Matters arising

Behavioural manifestations of third ventricular colloid cysts

Sir: When reading Dr Robert Winer's comment on the interesting topic on the symptomatology of third ventricular colloid cysts,¹ my attention was directed in particular to the statement that "neurosurgical consultation was obtained with three different neurosurgeons, all of whom declined to pursue an operative course". It may be easy to understand the hesitancy to surgery, if only the traditional transventricular removal is considered. However, I would like to point out that stereotactic aspiration of colloid cysts is established therapy.² The results after this alternative approach are promising. The first case mentioned has now been followed for 16 years, without recurrent symptoms. The aspiration technique has been employed by others, with rewarding results reported in a significant number of patients.^{3,4}

With regard to the innocent character of a colloid cyst in the third ventricle, a conventional transventricular removal may be too mutilating a procedure. Any experienced brain surgeon may recall frustrating episodes from such surgery, which no longer seems to be necessary. A common indication for surgical treatment has been signs of impaired CSF circulation, with ventricular dilatation. As a wider spectrum of the clinical picture has been elegantly described by Dr Winer as well as by Lobosky *et al.*,⁵ more patients than those with ventricular dilatation should probably be candidates for surgery, especially as a less traumatic stereotactic aspiration technique is well established management.

ERIK-OLOF BACKLUND
Department of Neurosurgery,
University of Bergen,
School of Medicine,
5016 Haukeland Hospital,
Bergen, Norway

References

- 1 Winer RI. Behavioural manifestations of third ventricular colloid cysts. *J Neurol Neurosurg Psychiatry* 1985;48:847.
- 2 Bosch DA, Ræhn T, Backlund EO. Treatment of colloid cysts of the third ventricle by stereotactic aspiration. *Surg Neurol* 1978;9:15-8.
- 3 Lunsford LD, Martinez AJ. Stereotactic exploration of the brain in the area of computed tomography. *Surg Neurol* 1984;22:222-30.
- 4 Rivas JJ, Lobato RD. CT-assisted stereotactic aspiration of colloid cysts of the third ventricle. *J Neurosurg* 1985;62:238-42.

- 5 Lobosky JM, Vangilder JC, Damasio AR. Behavioural manifestations of third ventricular colloid cysts. *J Neurol Neurosurg Psychiatry* 1984;47:1075-80.

Winer replies:

I am pleased to respond to Dr Backlund's letter concerning treatment of third ventricular colloid cysts. In relation to the use of stereotactic aspiration of the cysts, I would suggest that the morbidity involved in this procedure without the use of a CT-guided stereotactic system is unacceptably high. Experience in the use of CT-guided biopsy has clearly shown its superiority to non-CT-guided biopsy. Unfortunately, in the United States there is difficulty finding a neurosurgical centre which performs CT-guided biopsy.

Apart from the choice of surgical approach to these lesions are the problems that a clinician encounters when trying to ascertain an objective approach to the management of these lesions. The difficulty that I encountered in my case,¹ revolved around three issues: (1) the absence of hydrocephalus, (2) the operative morbidity in a 67-year-old patient, (3) the lack of ability to predict that an operative procedure would improve cognitive symptoms. In reviewing several series of colloid cysts of the third ventricle, the mean age of the patients were as follows: Antunes *et al.*:² 37 years in a series of 33 cases,² 31 years of age in six patients reported by Michels,³ 37 years of age in a series of 14 patients reported by Ganti *et al.*⁴ Clearly, there is need for objective and age-related criteria for management of these tumours.

Broadly speaking, these tumours should be divided into those with hydrocephalus and those without. Once this differentiation has been made symptom complexes such as headache, syncope, visual symptomatology, and cognitive and neuropsychiatric symptomatology should be factored in. In addition, objective findings on neurological examination must be considered in any analysis of case material.

I believe that it is incumbent upon all neuroscientists treating such patients to develop, either through correspondence or symposia, the above-mentioned data. Unless this is accomplished, management of these tumours will be steeped in the tradition of the treatment evolved in each particular geographical location.

I thank Dr Backlund for his timely mention of alternative surgical approach to this problem.

References

- 1 Winer RI. Behavioural manifestations of third ventricular colloid cysts. *J Neurol Neurosurg Psychiatry* 1985;48:847.
- 2 Antunes JL, *et al.* Colloid cysts of the third ventricle. *Neurosurgery* 1980;7:450-5.
- 3 Michels LG, *et al.* Colloid cysts of the third ventricle. *Arch Neurology* 1982;39:640-3.
- 4 Ganti SR, *et al.* CT of colloid cysts of the third ventricle. *Radiology* 1981;138:385-91.

Disappearing focal CT scan abnormalities in epilepsy

Sir: I read with interest the paper by Sethi *et al.*,¹ reporting a group of patients presenting with seizures and abnormal CT scan who showed complete resolution of the CT scan changes on follow up, without any treatment other than anticonvulsants. To explain the hyperdense enhancing lesions, pathologies other than these considered by the authors should also be considered. Theoretically intracranial sarcoidosis could be a possible explanation. In fact, it is well known that in this disease CT scan shows lesions with slightly increased attenuation, homogeneous enhancement, and little surrounding low attenuation. The lesions are usually multiple but a case of sarcoidosis mimicking malignant glioma on CT scan and producing partial seizures has been reported.² Nevertheless, the complete disappearance of the lesions in the cases of Sethi without any steroid treatment makes this hypothesis unlikely. Also multiple sclerosis cannot be excluded *a priori*. It is known that 4% of the patients affected by this disorder can present epileptic seizures during the exacerbations of the disease.³ Drake and Macrae reported 13 patients with a definite diagnosis of multiple sclerosis who presented with epilepsy.³ The authors found a good correlation between clinical deficits due to the disease, EEG, and seizures.³ Thus multiple sclerosis could possibly explain the clinical and CT features in the patients of Sethi even if the seizures only exceptionally are the onset symptom of the disease.

In 1984 Rougier *et al.* reported four adolescent patients with focal seizures and appearing and disappearing CT enhancing lesions.⁴ In three cases stereotactic biopsy revealed astrocytic proliferation. In the authors' opinion whether this "gliosis" was the cause or an effect of the seizures is questionable. The most probable explanation of the phenomenon described by Sethi *et al.* is the alteration of the blood-brain barrier dur-