

## MATTERS ARISING

### CSF diversion in CSF fistulae

I read the article on benign intracranial hypertension as a cause of CSF rhinorrhoea, by Clark *et al.*<sup>1</sup> with great interest. It highlighted some of the difficulties associated with CSF diversion as the sole treatment of CSF dural fistulae. The medical literature shows that the incidence of symptomatic pneumocephalus in these patients is 17% and the rate of postshunt meningitis is 11%.<sup>2</sup> Cases 1 and 4 of Clark *et al.* showed that stoppage of CSF rhinorrhoea is not a guarantee of healing of the underlying dural rent, because the dura in these patients is often very thin and the underlying bone is eroded. The recurrent rate of CSF rhinorrhoea after CSF diversion without dural repair is 42% (table); however, CSF diversion as a secondary procedure for recurrent CSF leakage has been advocated for many years<sup>3</sup> and seems to be very successful in allowing healing of the dural repair; it carries very few complications compared to shunting alone (table).

It seems, therefore, that the most appropriate course of action in patients with CSF fistulae associated with benign intracranial hypertension is to drain the CSF perioperatively to facilitate intracranial dural repair of the cribriform plate and to continue the CSF drainage through a lumbar drain postoperatively for a few days. If the high CSF

pressure persists after operation, a permanent CSF shunt should be inserted before the CSF leakage recurs.

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- 1 Clark D, Bullock P, Hui T, Firth J. Benign intracranial hypertension: a cause of CSF rhinorrhoea. *J Neurol Neurosurg Psychiatry* 1994;51:847-9.
- 2 Eljamel MS. The role of surgery and B2-transferrin in the management of CSF fistulae (thesis). Liverpool: University of Liverpool, 1993.
- 3 Spetzler RF, Wilson CB. Management of recurrent CSF rhinorrhoea. *J Neurosurg* 1978;49:393-7.

#### P Bullock replies:

I thank Eljamel for his comments on our paper *Benign intracranial hypertension: a cause of CSF rhinorrhoea*. He has in the past reviewed patients with a CSF leak from a wide range of aetiologies, and recognised the difficulties that are often experienced with lumboperitoneal shunts.

The purpose of presenting our group of patients with malignant intracranial hypertension who had already undergone a lumboperitoneal shunt was to highlight the difficulties that are unique to a high pressure CSF leak, and warn of the hazards that may be encountered at craniotomy. To the best of our knowledge this is the first time that this group of patients has been reported, and one would recommend a revision of Ommaya's original classification of high pressure CSF leaks to include patients with benign intracranial hypertension.

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#### Summary of pooled data on CSF diversion in patients with CSF fistulae

CSF diversion	Primary treatment	Secondary to dural repair
No of patients	36	35
Recurrent CSF leakage	42%	8.6%
Symptomatic pneumocephalus	17%	8.6%
Postshunt meningitis	11%	2.8%

### Hemiballism in Parkinson's disease

Two patients with Parkinson's disease who developed severe and persistent hemiballism with improvement of parkinsonian signs were reported independently by Vidaković *et al.*<sup>1</sup> and Inzelberg and Korczyn<sup>2</sup> in the August issue of the Journal.<sup>1,2</sup> They add to two previously published cases showing the same combination.<sup>3,4</sup> Transient hemichorea-hemiballism was found in up to 5% of patients with Parkinson's disease treated with stereotaxic surgery and its severity occasionally gave rise to serious complications.<sup>5</sup> Our own experience with a large number of monkeys treated with 1,2,3,6-tetrahydropyridine to induce parkinsonism and submitted to subthalamotomy indicates that hemichorea-ballism is actually a common manifestation, despite which all animals showed considerable improvement in motor performance in the dyskinetic arm, confirming the previous experiences of Bergman *et al.*<sup>6</sup> and of Aziz *et al.*<sup>7</sup> The occurrence of persistent and severe hemichorea-ballism after placing a lesion in the subthalamic nucleus (STN) is not, how-

ever, a necessary requirement to achieve improvement in parkinsonian signs as shown by the patient reported by Sellal *et al.*<sup>8</sup> Chorea-ballism results when neuronal activity in the sensorimotor region of the globus pallidus medialis (GPM) falls below a given threshold due to reduction in the excitatory STN drives. In the parkinsonian state, the STN-GPM pathway is hyperactive and the inhibitory afferent activity to the GPM from the external pallidum and the striatum is reduced, resulting in exaggerated and abnormal neuronal firing in GPM. It is very likely that similar changes occur in the substantia nigra reticulata. Whether or not severe and permanent dyskinesia will result after an STN lesion probably depends on the degree of GPM neuronal activity shifting from a hyperactive (parkinsonism) to a near normal or hypoactive (chorea-ballism) state. According to experience with monkeys, the possibility of obtaining a favourable equilibrium and a good therapeutic response by subthalamotomy depends on two major factors: firstly, the degree of dopaminergic depletion that conditions the severity of parkinsonism and

therefore, the level of GPM hyperactivity; secondly, the extent of the subthalamic lesion, which will determine the reduction in GPM neuronal firing.

The STN is probably the basal ganglia structure with the greatest capacity to modify basal ganglia output through its connections with the GPM, substantia nigra reticulata, and brain stem reticular formation (pedunculopontine nucleus etc). The variety of symptoms and signs of Parkinson's disease suggests the participation of many basal ganglia output pathways in the pathophysiology of parkinsonism. On balance, we think that the data now available are encouraging for further consideration of the STN as a surgical target in Parkinson's disease.<sup>9</sup>

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- 1 Vidaković A, Dragašević N, Kostić VS. Hemiballism: report of 25 cases. *J Neurol Neurosurg Psychiatry* 1994;58:945-9.
- 2 Inzelberg R, Korczyn AD. Persistent hemiballism in Parkinson's disease [letter]. *J Neurol Neurosurg Psychiatry* 1994;58:1013.
- 3 Scoditti U, Rustichelli P, Calsetti S. Spontaneous hemiballism and disappearance of parkinsonism following contralateral lenticular lacunar infarct. *Int J Neurol* 1989;10:575-7.
- 4 Burnett L, Jankovic J. Subthalamotomy and Parkinson's disease. *Mov Disord* 1992; 7(suppl 1):160.
- 5 Dierssen G, Bergmann L, Gioino G, Cooper IS. Hemiballism following surgery for Parkinson's disease. *Arch Neurol* 1961;5:63-73.
- 6 Bergman H, Wichmann T, DeLong MR. Reversal of experimental parkinsonism by lesions of the subthalamic nucleus. *Science* 1990;249:1436-8.
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- 8 Sellal F, Hirsch E, Lisovski F, Mutschler V, Collard M, Marescaux C. Contralateral disappearance of parkinsonian signs after subthalamic hematoma. *Neurology* 1992;42: 255-6.
- 9 Guridi J, Luquin MR, Herrero MT, Obeso JA. The Subthalamic nucleus: A possible target for stereotaxic surgery in Parkinson's disease. *Mov Disord* 1993;8:421-9.

#### Inzelberg and Korczyn reply:

We thank Obeso *et al.* for their comments on our communication. The patient described by us<sup>1</sup> had been parkinsonian for several years before developing a spontaneous haemorrhage into the subthalamic nucleus. Figure 1 shows the CT demonstrating the lesion (because of a printing error, a different figure appeared with our report).

Obeso *et al.* correctly emphasise the central role of the subthalamic nucleus, which is strategically placed to modulate the output of the basal ganglia. The subthalamic nucleus is hyperactive in Parkinson's disease, thus increasing the inhibitory output from the globus pallidus. Levodopa reverses this situation, resulting in dyskinesia<sup>2</sup> (fig 2). Lesions of the subthalamic nucleus, as in our patient and several others,<sup>3-6</sup> abolish the excitatory drive from the subthalamic nucleus to the globus pallidus.