EDITORIAL COMMENTARY

Cognitive function and pallidotomy

The paper by Perrine et al (this issue, pp 150–4) is one in a series of recent reports of cognitive outcome after unilateral pallidotomy for patients with Parkinson’s disease.1,4 These studies have taken advantage of sophisticated and selective surgical techniques (and outcome measures) unavailable to earlier, poorly controlled, studies in the pre-levodopa era. More reliable targeting of globus pallidus interna lesions is likely to have reduced cognitive complications, although potentially, the margin for error remains small and there has been at least one report of hemisphere specific postoperative cognitive changes.4 Nevertheless, despite a considerable degree of individual variability (postoperative gains and deficits), experienced centres have not generally found clinically significant cognitive sequelae (in terms of statistically significant changes in group mean scores) associated with good neurological outcomes (at 3 to 12 month follow ups). Similar findings have been reported after bilateral pallidotomy, although postoperative deterioration in verbal fluency, speech-motor apparatus, and possibly “executive” functions, remains a concern.3

This individual variability in outcome has yet to be explained and it may require larger sample sizes for any consistent pattern of group cognitive changes to emerge. However, refinements are also needed in the methodology of plotting the volume, contour, and location of pallidal lesions relative to individual neuroanatomy, before any exploration of associations between lesions and selective postoperative cognitive change can proceed. Such studies might advance mapping the functional architecture of the globus pallidus. Until more is known about the role of the globus pallidus interna in cognition (or the reliability of surgical targeting), psychometric test batteries should arguably be as comprehensive as is clinically practicable (rather than selective in terms of cognitive domains known to be impaired in Parkinson’s disease), if they are to represent an adequate audit.

Careful consideration needs to be given to the design of a neuropsychological audit, and the interpretation of any postoperative changes. For example, when tests are repeatedly administered, performance may improve due to practice and/or familiarity effects. With a comprehensive battery of tests, many comparisons will need to be made from baseline to follow up(s), and a careful balance therefore struck between the clinical significance of making type I or type II errors in statistical analyses. The recruitment of appropriately matched control groups or employing tests with parallel forms partially addresses the problem of test-retest effects, but it is not known if these are different for control and pallidotomy groups and postoperative scores may also be indirectly influenced by other factors. For example, a reduction in dyskinesias will improve dexterity and may also have a secondary, beneficial effect on attention; further, the effects of any postoperative changes in parkinsonian medications are largely unknown.

Despite these difficulties, adequate neuropsychological audit of ablative neurosurgery (or the implantation of stimulators) in consecutive series is important for several reasons, not the least of which is to enable meaningful comparison of the safety of different procedures—for example, surgery guided by microelectrorecording as opposed to (various methods of) image guidance with macrostimulation. Clinicians may also find baseline psychometric measures of cognition helpful in interpreting any subjective reports of postoperative change; “effort after meaning” can be a problem after any neurosurgical procedure. In addition, baseline assessment is an important screening device. Dementia is widely accepted as a contraindication to pallidotomy, but as many patients present with advanced disease and complex symptomatology, brief screening tests such as the mini mental state examination are often too crude to adequately identify suitable surgical candidates. Retrospective evaluation of baseline assessments (in the light of subsequent outcome) may ultimately help establish empirically, more selective cognitive contraindications.

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When evaluating patients with epilepsy, the integration of data from different modalities is the key to understanding the localisation of onset of seizures, the epilepsy syndrome, and the aetiology. This process is particularly important when patients have refractory partial seizures and neuro-surgical treatment is an option; the best results from surgery being obtained when the operative procedure removes the area of brain that gives rise to the seizures but is confined to cerebral tissue, the loss of which does not give rise to significant cognitive or neurological disturbance, with the remaining brain functioning normally. In this issue, the paper by Koutroumanidis et al (pp 170) describes 28 patients with temporal lobe epilepsy that was not due to foreign tissue lesions, in whom surgical treatment was being considered. The emphasis of the paper was the EEG finding of interictal regionalslowwave activity and the relation of this to data that infer functional abnormality—clinical features, regional cerebral glucose metabolism; and with structural data—MRI and the pathology of the subsequently resected temporal lobes.

The other functional data that may be integrated into the overall picture include the clinical description of seizures, neuropsychological tests, functional brain imaging (functional MRI and single photon emission computed tomography), and functional electrophysiological data (interictal and ictal EEG and magnetoencephalograms (MEG)).

In presurgical evaluation of the EEG, it is the distribution of ictal and interictal epileptiform activity that generally receives the most attention and the paper by Koutroumanidis et al serves as a timely reminder to pay due attention to abnormalities of the background EEG. A striking finding was the concordance between regional slow activity recorded from anterior, mid-temporal, and posterior temporal electrodes with hypometabolism in the posterior lateral temporal neocortex.

Brain PET with 18F-deoxyglucose (18FDG) commonly shows hypometabolism in a region that includes the site of onset of seizures. Regional diachisis and a functional inhibition are the likely causes for the area of hypometabolism being larger than the pathological abnormality. The use of 18FDG PET may show abnormalities when MRI is unremarkable. Comparisons of this type, however, depend crucially on the relative sophistication of the MRI and PET equipment and protocols being used. 11C-flumazenil binds to the central benzodiazepine receptor that is part of most cerebral GABA, receptors. It has been known for a decade that epileptic foci are commonly associated with an area of reduced flumazenil binding and for 5 years that this area is usually more restricted than the area of hypometabolism.

The mechanisms giving rise to regional slow activity in this setting, in the absence of an underlying neocortical or foreign tissue lesion, are not clearly understood. The parallel with the finding of regional hypometabolism suggests that a reversible diachisis and inhibitory processes, which result in neuronal dysfunction, may be the underlying causes.

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J Neurol Neurosurg Psychiatry 1998 65: 148-149
doi: 10.1136/jnnp.65.2.148

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