originally reported in the Hoehn and Yahr study in the prelevodopa area. Does this mean that dopaminergic replacement with levodopa, dopamine agonists, or combinations of both has not significantly altered the longterm outlook for people with Parkinson's disease? Probably not. As the authors admit, their patients may have been undertreated due to the initial design of the study as a comparative trial of low dose levodopa versus low dose bromocriptine. Their outcome may not be representative for the treated parkinsonian population at large. By contrast the recent 9 year follow up results of the DATATOP cohort of patients showed supernormal life expectancy with a standard mortality ratio of 0.9.[5] Such discrepancies in outcome between prospective follow up studies over similar time periods are likely to reflect differences in baseline severity and comorbidity and possibly treatment strategies. Idiopathic Parkinson's disease is not a prognostically uniform entity; elderly patients with comorbid dementia and cerebrovascular and heart disease face a high risk of significant disability or death after 10 years, contrasting with a near normal life expectancy in the younger onset patient without dementia or other significant comorbidity and optimal treatment under specialist supervision.

W POEWE

Universitätsklinik für Neurologie, Universität Innsbruck, Anickstrasse 35, A–6020 Innsbruck, Austria

1 Poewe WH, Wenning GK. The natural history of Parkinson's disease. *Ann Neurol* 1998;**44**(suppl 1):S1–9.
2 Hoehn MM, Yahr MD. Parkinsonism: onset, progression and mortality. *Neurology* 1967;**17**:427–2.
3 Clarke CE. Does levodopa therapy delay death in Parkinson's disease? A review of the evidence. *Mov Disord* 1995;**10**:250–6.
4 Olanow CW. Attempts to obtain neuroprotection in Parkinson's dusease. *Neurology* 1997;**49**(suppl 1):26–33.
5 The Parkinson's Study Group. Mortality in DATATOP: a multicenter trial in early Parkinson's disease. *Ann Neurol* 1998;**43**:318–25.

EDITORIAL COMMENTARY

Deep brain stimulation in Parkinson's disease

This issue of *the Journal* sees the publication of two papers that increase our knowledge of the functions of the internal architecture of the thalamus and globus pallidus—an important achievement given the existing literature on stereotactic functional surgery for Parkinson's disease.

The paper by Caparros-Lefebvre *et al*[1] (pp 308–14) is fascinating, because one would have expected that after nearly 50 years of thalamic surgery every possible internal thalamic target would have been explored. However, the surgical outcomes have not always been studied carefully, or published for others to share. Caparros-Lefebvre *et al* compared the functional results and electrode positions obtained by two teams performing thalamic stimulation for parkinsonism. Anatomical comparisons were possible because ventriculography had been performed by both groups. The two teams used similar techniques for the implantation of electrodes into the ventralis intermedius nucleus of the thalamus (VIM), although there were minor differences in the approach trajectory which led to team A's electrodes being placed an average of 2.9 mm posteromedial to those of team B. The result of this slight positional difference was that both tremor and drug induced choreic dyskinesias were abolished by the more posteromedial target, whereas only tremor was relieved by the more anterolateral electrode position. Evidence for this antichoreic dyskinetic effect being secondary to involvement of the centre median and parafascicularis complex (CM-Pf) nucleus is provided. It is noteworthy that no effect on dystonic dyskinesias was found, suggesting a segregation of the pathways involved in these two forms of dyskinesias. However, the clinical importance of this paper lies in the demonstration that surgery to a single posteromedial VIM target can achieve the same functional outcome as that involving both VIM and ventralis oralis posterior—a finding that may translate into a reduced risk of side effects.[1]

The paper by Durif *et al*[2] (pp 315–22) considers the possible causes for the variability in clinical outcome obtained after pallidal surgery. The study focuses on the precise target site which in most series, including this one, lies within the posterior half of the pallidum. Durif *et al* report that within their pallidal target, ventral stimulation is more effective than dorsal stimulation for alleviating rigidity, bradykinesia, and drug induced dyskinesias, a finding that concurs with a recent study of pallidotomy and clinical outcome, but differs from the findings obtained by Krack *et al* who noted that ventral stimulation within GPi caused improvement in rigidity and alleviation of levodopa induced dyskinesias but caused severe akinesia and blocked the antiakinetic effect of levodopa.[4 5] There are two possible reasons for this discord: firstly, the target chosen by Krack *et al* is posterolateral to that selected by Durif *et al*, and secondly the approach angle may matter.

These studies show that from detailed assessments of the relation between surgical target and clinical outcome important clinical and physiological questions may be answered about the function of specific areas within the thalamus and globus pallidus.

T Z AZIZ
P G BAIN
Department of Neurosciences, Charing Cross Hospital, London, UK

T Z AZIZ
Department of Neurosurgery, Radcliffe Infirmary, Oxford, UK

1 Caparros-Lefebvre D, Blond S, Feltin M-P, *et al*. Improvement of levodopa induced dyskinesias by thalamic deep brain stimulation is related to slight variation in electrode placement: possible involvement of CM Pf. *J Neurol Neurosurg Psychiatry* 1999;**67**:308–14.
2 Durif F, Lemaire J-J, Debilly B, *et al*. Acute and chronic effects of anteromedial globus pallidus stimulation in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1999;**67**:315–22.
3 Narabayashi H. In: Gildenberg PL, Tasker RR, eds. *Textbook of stereotactic and functional neurosurgery*. New York: McGraw-Hill, 1998:1033–8.
4 Gross RE, Lombardi WJ, Lang AE, *et al*. Relationship of lesion location to clinical outcome following microelectrode-guided pallidotomy for Parkinson's disease. *Brain* 1999;**122**:405–16.
5 Krack P, Pollack P, Limousin P, *et al*. Opposite motor effects of pallidal stimulation in Parkinson's disease. *Ann Neurol* 1998;**43**:180–92.