Parkinson's disease

Driving in Parkinson's disease

A Schrag

It is not possible for patients to predict their driving safety from a motor examination or the patient's own judgement

Patients with Parkinson's disease are usually advised to inform the driving licensing authority and their driving insurance company of the diagnosis. However, until a few years ago, Parkinson's disease was not considered a major obstacle to safe driving until the advanced stages. This changed when in 1999 sudden onset sleep attacks in patients with Parkinson's disease on dopaminergic drugs were first reported.[1] This not only led to considerable concern about the safe use of dopaminergic drugs in patients who drive, but also to a flurry of studies investigating the frequency of the problem, the contributing factors, and the mechanism and types of drugs associated with it. However, while this remains an important issue, relatively little attention has been paid to the overall ability and competence of patients with Parkinson's disease to drive a motor vehicle, unrelated to sleep attacks.

In this edition of JNNP (*see pp 176–80*), Wood *et al*[2] address this issue using structured qualitative and quantitative assessments of driving ability in patients with Parkinson's disease and age matched controls. Experienced, qualified driving assessors who were blinded to the diagnosis found that in a realistic, on road setting, overall driving safety in patients with Parkinson's disease was significantly impaired; worryingly, a high proportion (more than 50%) of patients would not have passed a state based driving test. The study included 25 patients who had volunteered for the study after attending lectures on the subject, and it is therefore not possible to conclude that such a high percentage of the overall population of patients with Parkinson's disease would fail a driving test. However, it highlights the potential impairment of driving competence that patients with Parkinson's disease may experience.

An important finding was that the driving examiner's assessment of driving ability correlated poorly with the drivers' own view of how well they were driving. Poor driving performance also did not correlate with disease severity as measured on clinical scales, although longer disease duration was associated with greater difficulties. No neuropsychological tests were included in the study, but the type of errors made related not only to difficulties in motor tasks but also appeared to be related to the neurocognitive deficits known to occur in Parkinson's disease.[3] Thus, while slowness of movement and reaction time are likely to play important roles in driving performance, other factors such as impaired visuo-spatial processing, planning, and sequencing are also likely to contribute to the difficulties.

While these results must be confirmed in larger, unselected samples of patients with Parkinson's disease who are currently driving, the present study not only draws attention to the impairment of driving ability in Parkinson's disease but also shows that it is not possible for patients to predict their driving safety or to deduce their driving ability from motor examination. While the recommendation to stop driving (or to have a driving assessment) is currently often based on physicians' overall clinical judgment, this study highlights the need to assess driving ability in Parkinson's disease regularly and in a standardised fashion. It also calls for studies to explore the motor and non-motor factors leading to impaired driving performance and whether compensatory strategies may help patients improve their driving ability and maintain independence for longer.

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Stroke

Mapping anterograde and retrograde degeneration after stroke

J-C Baron

Tools to quantify secondary degeneration after stroke and monitor effects of intervention

In this issue, two original articles from highly experienced groups report on the use of diffusion tensor imaging (DTI) to map the dynamics of secondary degeneration after stroke (***see pp 200–5 and 266–8***). Thomalla *et al*[1] monitored in two patients the time course of Wallerian degeneration (WD) of the pyramidal tract following striatocapsular stroke. DTI was obtained on three occasions from the subacute into the chronic stage. They found a progressive decrease of the fractional anisotropy (FA) with an increase in mean diffusivity (MD) in the pyramidal tract at the level of the cerebral peduncle, reflecting the changes expected in WD—a progressive disintegration of fibre structure. Hervé *et al*[2] serially studied nine patients from 1 week to 6 months following MCA territory stroke, focusing on the ipsilateral thalamus. They found significant increases in MD from 1 month onward, without parallel changes in FA, presumably reflecting a progressive loss of neurones and/or glial cells. These changes in thalamic MD without changes in FA indicate that within complex multi-nucleated grey matter structures devoid of large fibre bundles such as the thalamus, changes in FA should not be anticipated while loss of organised cellular arrangements will