

## Matters arising

### Visuospatial impairment in Parkinson's disease: does it exist?

Sir: Visuospatial function in Parkinson's disease has been the subject of several investigations. Conclusions, however, are contradictory. Della Sala *et al*<sup>1</sup> and Brown and Marsden<sup>2</sup> recently reported their findings: no visuospatial impairment could be demonstrated. One can raise objections to both studies, however.

Brown and Marsden<sup>2</sup> tested 15 Parkinsonians (14 taking levodopa) on a Spatial Choice Reaction Time task. This task tested visuospatial function, in our opinion, on two levels: on an elementary level by discrimination between left and right, and on a complex level by involving mental rotation. As the authors stated themselves, there is no direct proof that no verbal strategy was used, implying the possibility that something different from visuospatial function was tested. Admitting that spatial function is not a unitary entity, it seems fit to quote De Renzi *et al*:<sup>3</sup> "...space perception has been studied at a rather complex level, one at which it is difficult to disentangle the influence on performance of spatial as compared with praxic, intelligence and memory factors. It seems reasonable to expect that a more definite answer might be obtained by employing elementary tasks, which tap the basic mechanisms underlying spatial perception." Della Sala *et al*<sup>1</sup> employed such an elementary task, by asking the patient to forecast where a slanted line would intersect a horizontal line. Their test could be considered a sophisticated version of Benton's line orientation test.<sup>4</sup> An advantage is that results can be more readily quantified and that subjects have to indicate exactly where the line intersects instead of choosing from a fixed number of possibilities. The authors suggest that the absence of any visuospatial impairment in Parkinson's disease, in contrast to other studies, might be due to the fact that their sample of patients is highly selected. We should like to stress this point: all patients were younger than 70 years; all were on levodopa substitution therapy: all were in-patients and, most importantly: patients were excluded with "any sign of everyday impairment of their ability to cope socially". Thus patients who did show visuospatial impairment on a clinical level were effectively excluded from their study.

We also investigated visuospatial function in Parkinson's disease<sup>5</sup> and our findings are in contrast with those of Della Sala *et al*<sup>1</sup> and Brown and Marsden.<sup>2</sup> We used the rod

orientation test (ROT) as described by De Renzi *et al*<sup>3</sup> and, in addition, the line orientation test and the facial recognition test.

Forty three of 44 patients (all without levodopa substitution therapy) had abnormal results on the ROT, while on the orientation test and the facial recognition test respectively seven and 17 failed. The last mentioned tests and the test used by Della Sala *et al* might be specific for visuospatial function but are in our opinion less sensitive than the ROT. It has been admitted, however, that it is still an open question whether all these test do indeed explore identical aspects of visuospatial behaviour.

In our opinion, there does exist a visuospatial impairment in Parkinson's disease, although it might very well be that this cannot be demonstrated at more complex levels, or missed, when basic mechanisms are tested in a highly selected sample of patients. As Della Sala *et al* stated the problem consists of sample bias and test bias: Many tests probably screen different aspects of spatial behaviour.

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### Visuo-spatial impairment in Parkinson's disease. Does it exist?

Spinnler and Della Sala reply:

Hovestadt *et al*<sup>1</sup> published a paper supporting early visuo-spatial disorders in Parkin-

sonians. These conclusions are at variance with Brown and Marsden<sup>2</sup> and our own findings.<sup>3</sup> As Hovestadt *et al* state in their letter they agree that sample-biases (that is strict exclusion of "demented" Parkinsonians) and test-biases (that is elementary nature of the spatial task employed) actually made it unlikely to find poor performers in our study<sup>3</sup> and on the contrary with different constraints, to find them in their own.<sup>1</sup> We actually did exclude from our study all Parkinsonians with general coping inability and only included those patients whose spatial impairment under scrutiny could not be traced back to general cognitive deterioration, but would appear as "specific" feature of their nigro-striatal disorder. We take the present opportunity to outline our views beyond those listed in our paper.<sup>3</sup>

(1) *Sample-biases*. Parkinson's disease is a progressive degenerative condition for which the hypothesis might be put forward that in an unknown number of patients the degenerative process will sooner or later encroach also upon systems other than its nigro-striatal first target. This being the case (a worthwhile hypothesis suggested by clinical observation on unselected patients) longitudinal surveys on a carefully selected sample of early Parkinsonians would be likely to find, in time, an increasing number of patients adding to their motor problems also cognitive impairments. By means of such experiments sound answers could be gathered on the cognitive role specific to the striatum, possibly on its neuropsychological features as well as on its natural history. Longitudinal, PET and transmitter studies would provide the ideal correlative variables to the possible spreading of the damage from subcortical to neocortical (viz, prefrontal structures)<sup>4</sup> and their connections. Cross-sectional studies, on the other hand are in our opinion unable to give accurate results, essentially because they cannot avoid lumping together patients at poorly predictable degrees of their extra-striatal involvement (that is, patients with a more or less severe pre-frontal or even diffuse cortical encroachment). Put in other words, progressive diseases (such as Parkinson's or Alzheimer's disease) can supply information suited to reconstruct healthy functioning of the brain, only if the experimental designs take into account the progression of the deterioration, that is they are longitudinally structured starting from patients in early stage.

(2) *Test-biases*. The question of elementary vs complex spatial tasks plays its role at the moment of drawing a conclusion of experi-

mental data. Longitudinal surveys should allow us to find out, while the disease is running its course, whether the specific elementary defects appear in isolation first and more complex impairment only later on along with other cognitive defects (viz, impairment in attention and automatization, in behavioural planning, in intelligence, memory and even in some verbal abilities). By definition, complex behavioural disorders are multifactorial in nature and are poorly informative of the first hampered functions (such as Parkinson's spatial defects), being admittedly more representative of the everyday coping disorders.

Summing up, we presently share a wait-and-see position on the specification, if any, of the role in cognition of the basal ganglia, at least when conclusions stem from data collected from patients with progressive neurological "subcortical" disease; we are inclined to lend great credit to eventually forthcoming longitudinal studies, starting from reasonably homogeneous and "pure" samples (viz, employing very early patients). On the other hand, we feel rather sceptical on the general conclusions drawn from the current cross-sectional cognitive studies which yield ever increasing discrepancies. For instance, still unpublished findings of ours on the strategy-producing ability linked to the frontal network (that is, a simplified version of the "Towers of Hanoi" test) in strictly selected Parkinsonians failed to support neuropsychologically the specific frontal impairment in Parkinson's disease.<sup>5</sup>

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## Cerebral cysticercosis presenting with hemichorea

Sir: Your recent report of a patient with hemichorea due to metastatic carcinoma<sup>1</sup> prompts us to record a case of hemichorea which was also associated with focal lesions, in this case cerebral cysticercosis.

A 15 year old black girl presented in November 1982 with a two month history of involuntary movements of the left side of her body. The movements were gradual in onset and they were progressive. There was no recent or remote history of drug ingestion, oral contraceptives, exposure to toxins, head injury, sore throat or joint pains. She was admitted to a peripheral hospital in 1978 with severe headaches which subsided spontaneously. There was no family history of similar illness. On examination she was a mentally alert girl with left-sided facial distortions producing half smiles and half grimaces, and purposeless non-stereotyped jerks of the distal parts of her left arm and leg. Superimposed on these was intermittent flinging of the whole arm and leg. The tendon reflexes were slightly brisker on the left side but the plantar reflexes were flexor on both sides. Physical examination was otherwise normal.

The following investigations gave normal results: chest and skull radiographs, ESR, thyroid function tests, anti-streptolysin O titre, serum copper and caeruloplasmin levels, VDRL, auto-antibody screen and pregnancy test. The absolute eosinophil count in the peripheral blood was 770/mm<sup>3</sup>. The cysticercus haemagglutination test and the fluorescent antibody test were positive in the blood in titres of 1:640 and 1:10 respectively. The corresponding titres in the CSF were 1:16 and 1:1. There were no eosinophils in the CSF. EEG showed some excess of polymorphic slow activity bilaterally. The CT scan showed numerous cysts, with calcification and central or peripheral contrast enhancement, in both hemispheres (fig). The appearance is characteristic of parenchymatous cysticercosis in the late active stage. Of particular interest, in this clinical setting, were several cysts in the basal ganglia of the right hemisphere. One was situated in the head of the caudate nucleus, encroaching on the anterior horn. At least three more were situated in the region of the anterior limb of the internal capsule and the lenticular nucleus.

The patient was treated with haloperidol. There was some improvement in her involuntary movements. She had presented when the value of praziquantel in treating cerebral cysticercosis was not yet established. We have since lost contact with the

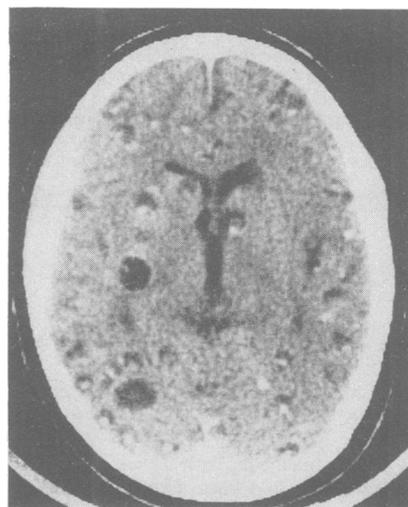


Fig Contrast enhanced CT scan showing multiple cysticerci, with some in the head of the right caudate nucleus and striatum.

patient.

There can be little doubt that the patient's neurological illness was the result of cerebral cysticercosis. While the lesions were diffuse and multiple, her left hemichorea could be explained by the cysts in, and adjacent to, the head of the right caudate nucleus. Some cases of chorea have been attributed to the neoplastic<sup>1</sup> and vascular<sup>2</sup> lesions in the contralateral caudate nucleus, putamen and other basal ganglia. Although cerebral cysticercosis has very protean manifestations, reviews of the field,<sup>3-4</sup> and a literature search retrospective to 1966, have yielded no reference to it as a cause of chorea. Greater availability of CT scanning may reveal this association more frequently in the Third World, where cerebral cysticercosis is a major cause of neurological illness.

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