Seeing in Parkinson's disease

Although the motor disorders of Parkinson's disease (PD) produce the most obvious signs of the illness (tremor, rigidity, and akinesia), during the past 40 years evidence has been accumulating for more subtle disturbances of perception and cognition. Post-mortem studies of the eye, and imaging studies of the living brain, have revealed abnormalities of dopaminergic cells in the PD retina, and hypoactivity of metabolic processes in cortical regions at the rear of the PD brain (occipital, parietal and parts of the temporal lobes). These anatomical and physiological studies suggest that vision may be affected in PD.

Ganglion cells in the retina send information about the luminance contrast of local regions of the retinal image up to the brain. Work by others has demonstrated losses of contrast sensitivity for medium and high spatial frequencies in PD, and shown that the density of dopaminergic amacrine cells in the monkey parallels that of the rods (absent in the fovea with a peak 15 degrees or so into the periphery). It is in the peripheral retina that dopamine has its greatest effect on ganglion cells, which seems to be that of re-organising receptive field properties during light adaptation. As suggested by these anatomical and physiological findings, we found that losses of neurally-signalled contrast are greater in peripheral than in foveal vision (1) by getting patients to match the apparent contrast of a peripherally viewed grating to that of a foveally viewed grating. From similar studies in normal observers, either light or dark adapted, we found support for the idea that the Parkinsonian vision system behaves as though too dark-adapted for the prevailing luminance (2). Using the tilt aftereffect, whose magnitude is known to be contrast-dependent, we were able to show that losses of sensitivity contrast occur above as well as at threshold in PD (3).

The likely involvement of the right parietal lobe in PD suggests that visuo-spatial abilities may be impaired (though this has proved to be a controversial issue, with some investigators suggesting other reasons for errors made by PD patients on visuospatial tasks). In a study employing the mental rotation task developed by Shepard and Metzler, we showed that, although patients were relatively unimpaired (somewhat slower) when 2D rotation was required, they made systematic errors when large 3D rotations were needed, suggesting that they were using an inappropriate strategy (4). In subsequent work, we have investigated distortions of perceived visual space which may lead to difficulties in the control of movements such as reaching or avoiding objects, and which are greater in patients whose motor symptoms are worse on the left side of the body – LPD - (and so whose brains may be worse affected on the right side). Patients whose right parietal lobes have been damaged by e.g. a stroke often mis-bisect a horizontal line too far to the right, as though "neglecting" (failing to see or to attend to) the left end of the line. Lines were presented on a large screen to PD patients, who adjusted the position of a cursor with remote switches until it appeared to bisect the line. LPD but not RPD patients mis-bisected too far to the right (5). In a similar task, but with vertical lines, LPD patients set the cursor too low, suggesting that they neglect upper visual space (6). Consistent with the idea that visual space is compressed in LPD (especially on the left), LPD patients judge objects in left visual space to be smaller than those in right visual space (7). Such findings suggest that people with PD may have difficulties in judging spaces and spatial relationships in everyday life, and this is confirmed in a questionnaire study (8).

To analyse these potential problems in more detail, we asked patients to judge whether they would fit through a schematic doorway back-projected on a large screen without turning their shoulders. Control subjects judged that they would fit through a doorway which was 10% wider than their shoulder width. LPD patients required the doorway to be 150% of their body width before they felt that they would fit through it, in contrast to RPD, who, as a group, judged that they would fit through a doorway which was only 90% of their body width (9). These perceptual errors are

consistent with a compression of visual space in LPD, and a compression of one's own body-image in RPD.

In current work, in collaboration with Dr David Ewins (Centre for Biomedical Engineering, University of Surrey), we are studying how patients (whose perceptual difficulties are known) behave when walking through doorways and cluttered environments. Amongst other things, we are interested in how doorway widths and different decorative schemes affect freezing, veering, and other problems with gait in PD.

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