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CORRESPONDENCE

Neurovisual rehabilitation in Balint's syndrome

Further to the excellent review of neurovisual rehabilitation by Kerkhoff¹, we think that it is prudent to communicate our experience in the management of a patient with Balint's syndrome after traumatic brain injury. This was seen in a 41 year old right handed manual worker whose initial cranial CT showed right extradural haematoma. Subsequent scans demonstrated left posterior occipital infarct. Brain MRI 3 months after the injury showed high signal in the right occipitoparietal and left occipitotemporal regions. His physical recovery was satisfactory in that he was fully mobile unaided. However, he presented with simultanagnosia, optic ataxia, and psychic paralysis of gaze.² This had an adverse impact on his functional independence; he had difficulty in route finding—crashing into furniture and walls—and other activities of daily living including dressing and toileting. He failed most subtests in the Rivermead perceptual assessment battery (RPAB).

We agree with the author that effective treatment strategies are poorly developed and evaluated. We have identified three approaches for the rehabilitation of the perceptual deficits including those seen in Balint's syndrome.

- The adaptive (functional) approach,³ which involves functional tasks utilising the person's strengths and abilities, helping them to compensate for problems or altering the environment to lessen their disabilities
- The remedial approach,⁴ which involves restoration of the damaged CNS by training in the perceptual skills, which may be generalised across all activities of daily living. This could be achieved by tabletop activities or sensorimotor exercises
- The multicontext approach,⁵ which is based on the fact that learning is not automatically transferred from one situation to another. This involves practising of a targeted strategy in a multiple environment with varied tasks and movement demands, and it incorporates self awareness tasks.

In this patient, we used the adaptive approach, practising functional tasks repeatedly with increasing complexity of the tasks as the sessions continued. This approach assumes that treatment has little effect on impairment and that generalisation to other tasks is unlikely. It also assumes that the brain has limited ability to improve and restore itself after injury.³ The remaining abilities are used to offset the deficits. This patient was able to develop his own compensatory strategies, learned to use his hands to acquire tactile feedback, and managed to direct his

gaze to visually locate objects when required. His performance on the RPAB was improved and he was successfully discharged home with little support.

As Balint's syndrome is likely to be seen in clinical conditions such as Alzheimer's disease, multiple strokes, intracranial tumours, brain injury, and CNS complications of HIV infection, we thought it important to outline the possible options for the management of this condition. Further work is required on a larger series of patients.

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Kerkhoff replies:

Al-Khawaja and Haboubi have reported successful neurovisual rehabilitation in a patient with Balint's syndrome due to a right occipitoparietal and a left occipitotemporal lesion, using adaptive practising of functional tasks with increasing complexity. This case shows, together with some of the other published cases,¹ that individually tailored rehabilitation strategies can be adapted successfully for some severely disabled patients. Due to the various aetiologies and subsequent lesion localisations it is likely that most patients with Balint's syndrome will have some outstanding deficits, but also some spared abilities, which can be used for compensatory purposes (for example, intact tactile feedback¹).

In two patients with Balint's syndrome treated for several months in our department, significant improvements could be achieved by systematic treatment so that both patients could live independently at home with only minimal assistance. One patient, who had received traumatic, uraemic, and hypoxic brain damage at the age of 27 years initially was nearly blind. Two years later, when treatment started in our unit, he couldn't perceive more than two visual stimuli simultaneously (simultanagnosia), was almost unable to read, showed optic ataxia, and had severely impaired spatial-perceptual functions in the visual and tactile modality. However, his memory, intelligence, and executive functions were largely preserved, so that he relearned reading partially, learnt to dress himself partially, and was finally able to travel by train. He managed to live alone in his flat, with only minor assistance from others.

The second patient, a 60 year old physician, had bilateral vascular parieto-occipital lesions. She was initially (falsely) considered as blind, although she could well see and describe faces and correctly identify the colour of one's eyes. She presented with severely disturbed depth and horizontal distance perception, simultanagnosia, and optic ataxia as well as a peculiar deficit in identifying spatial directions and locating sound sources. For instance, it proved difficult for her to identify the direction in which someone pointed when describing a particular route, or to decide in which direction a train would move when looking at the railway track. However, as in the first patient, she had some spared abilities—that is, excellent introspection and awareness of her disorder, preserved cognitive abilities, and she was highly motivated to relearn route finding in her town district. After intensive training for reading and route finding she could be discharged, living independently at home. She continues to use public transport to go shopping, visit friends, see her neurologist, the pharmacist, or going to a concert hall.

I think that systematic treatment in both cases helped to improve basic visual abilities and activities of daily living so that both patients could live independently at home—which was hardly expected when seeing them at the onset of treatment. To conclude, I am convinced that many patients with Balint's syndrome can learn to compensate for at least some of their visual deficits by systematic and individualised treatment. The search for spared functions will undoubtedly disclose multiple ways for compensation and will increase our understanding of some unresolved aspects of this fascinating syndrome—for example, the tactile or auditory-spatial abilities of patients with Balint's syndrome.

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Arnold Chiari malformation and nystagmus of skew

I enjoyed Pieh and Gottlob's article¹ pointing out the association of a Chiari malformation with a "unique" form of nystagmus that they call "the nystagmus of skew." The distinctive feature of this nystagmus is a disjunctive vertical oscillation in which the fast phase of one eye moves upward while, at the same time, the other eye moves downward.

The authors¹ state that their second patient had a "rotatory component" by which I assume they mean torsional; this pattern of nystagmus is already established in the literature and is known as "jerk-waveform see-saw nystagmus".^{2,3} In their first patient they point out that the amplitude of the vertical nystagmus was so small that they were unable to confidently exclude a torsional component. It would have been most interesting to obtain recordings looking for a torsional component using the modified