Matters arising

Spatial disorientation in right hemisphere infarction

Sir: Personal involvement in the care of a close relative can make reading a related paper especially interesting for a physician. It was in this sense that I read JD Meerwaldt’s article about the prognosis of spatial disorientation due to right hemisphere infarction with particular interest.1 In his study only one of 16 patients did not recover completely within six months after an ischaemic stroke. I have had the unfortunate opportunity to follow my father’s illness with the same syndrome and my comments seem relevant.

He is presently 82 years old, and was in excellent health until three years ago. He had a few brief episodes of dizziness prior to May 1981, when he fell off his bicycle as a result of one such episode. On initial neurological examination he had mild left sided hemiparesis, left homonymous hemianopia in addition to the usual right parietal signs: constructional and dressing apraxia, prosopagnosia and profound spatial disorientation. This was so severe that he could not find his way in and around the house that he had lived in for over 15 years. CT scan and angiograms were not performed, but he had no midline shift on his echoencephalogram and the EEG showed right temporoparietal slowing only. He was discharged from hospital with the diagnosis of an ischaemic stroke due to probable middle cerebral artery occlusion. I first had the opportunity to examine him about two months after the initial event. By then the hemiparesis had almost completely disappeared, and there was only visual extinction, but no hemianopia, on the left side. He still had difficulties with dressing himself and was unable to “build” a house from matches. He was unable to name directions, could not describe how to get to the houses of his children, how to find the local railway station, market place etc. He recovered from some of his symptoms by July 1983, when last examined. He still complained of stiffness in his left arm, made frequent mistakes with naming directions and was unable to get home from a distance of a few hundred yards. The disorientation was especially noticeable in darkness, and a light had to be left on in his bedroom to help his orientation in the evening and at night.

It would not be prudent to compare the results of a test done on 16 patients with the clinical findings on one patient. The discrepancy between the quick recovery in Meerwaldt’s 15 cases and my father’s slow and incomplete recovery, however, is quite noticeable. Age difference might have been one of the reasons (my father was 80, the oldest in Meerwaldt’s study 70). Meerwaldt commented on the difference between his and Benton’s2 findings, and explained it on the basis of different aetiologies and presence or absence of cerebral oedema. My father in all probability, although there was no CT scan or angiographic proof, had an ischaemic infarct, and should be comparable to Meerwaldt’s cases. I am inclined to believe that the discrepancy is basically artificial and related to the unnatural examination technique used by Meerwaldt. Neither the rod, nor the line orientation test can be used as substitutes for testing in real life situations and for inquiring from relatives about the patients’ behaviour. This, of course, is difficult to quantify and difficult to use in scientific research. This method alone, however, is unlikely to provide us with the information about prognosis in this syndrome that practising neurologists are looking for. It would be advisable to complement it with corresponding clinical information in similar future studies.

Matters arising


References


Notice

European Society for Neurochemistry, 5th General Meeting will be held in Budapest, 21–26 August 1984.

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