The finding by Kalra et al. that the physiotherapists in the stroke unit placed significantly greater emphasis on activities that addressed the specific functional needs of individual patients strongly suggests that an important difference in physiotherapy practices existed in the two treatment areas. The presence of significant differences between the two physiotherapy staffs could therefore explain (at least partly) the better outcome observed in the stroke unit.

Relevant information about the physiotherapy staff involved in each arm of the study would therefore be welcome, as it would enable further analysis of the interesting results of this trial.

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Reference

Response

Our study unequivocally showed that the outcome of stroke management was significantly better in stroke rehabilitation units compared with general wards. As discussed at length in the article, this difference was primarily due to better organization of services and targeting of therapy resources according to the ability and, more importantly, the needs of the patient rather than due to increased resource input in the stroke unit. We confirm that the therapists involved in both arms of the study were of equivalent grades (Senior Grade I) and that they were equally supported by physiotherapy and occupational therapy aids. None of the therapists involved in the study had formal specialist training in stroke management. The stroke unit was developed in a general medical ward using therapists already working on the ward rather than those specially recruited for their neurological interest.

We believe that the role of factors such as the time spent by physiotherapists with stroke patients, the grade of the therapists, and specialist training in stroke management (as mentioned by Dr Panayiotou and Ms Beeson) have received too much emphasis in the past, with little attention being paid to what the therapy actually achieves and its relevance to the patients' needs. The major point made by our article was that this emphasis was misplaced: the efficiency of stroke units depends upon directing therapy toward adapting the patients' residual abilities to their future needs. The role of nontargeted but prescribed remedial treatment often seen in general wards and some stroke units may be “professionally appropriate” (especially in the British setting), but it is inefficient and of little benefit to patients. We would agree with Dr Panayiotou and Ms Beeson that a responsive management philosophy contributed to the difference in outcome in our study. The objectives of stroke unit rehabilitation should include proactive targeting of therapy and better organization of resources, or they may fail to achieve their potential, as has been seen in a recent study from Nottingham.

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Migraine Equivalent and Hemorrhagic Infarction

The cause of stroke during migraine is unknown. We report here the very rare case of a patient who suffered a hemorrhagic cerebral infarction after scintillating scotoma.

In July 1980, acute left homonymous hemianopia and scintillating scotoma were experienced by a 70-year-old man, who had been suffering recurrent scintillating scotoma without migraine headache twice a month for about 30 years. The visual abnormality lasted approximately 30 minutes, which was longer than usual. He had a sensation of “lightning bolts,” left homonymous hemianopia, and what appeared to be glimmering lights. On neuroophthalmologic examination, visual field testing showed left homonymous superior quadrantanopia. Computed tomographic scanning and magnetic resonance imaging showed hemorrhagic infarction in the right occipital lobe. No abnormal findings were detected by electroencephalography. Angiography showed poor filling in the vicinity of the right posterior cerebral artery. The left homonymous hemianopia and scintillating scotoma changed into visual hallucinations. The visual hallucinations suddenly occurred following the scintillating scotoma, and the lesion was consistent with that of scintillating scotoma. Moreover, the hemorrhagic infarction lesion did not conflict with the focus of the symptom neurologically. The left homonymous hemianopia and visual hallucinations were thus considered to have a cause-and-effect relationship with the hemorrhagic infarction. Recurrent scintillating scotoma may be produced by a variety of causes. However, the scintillating scotoma and left homonymous hemianopic and visual hallucinations for 30 years may have been caused by transient ischemic attacks in the right occipital lobe followed by migraine equivalent.

In classical migraine headache, the neurological symptoms of the aura are generally attributed to focal ischemia. Migraine stroke is a complication of this headache. However, the mechanism of cerebral ischemia in migraine remains unknown. No cases of hemorrhagic infarction followed by migraine equivalent have been reported in the literature. This case should thus provide some clarification of the mechanism of cerebral ischemia in migraine.

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Reference

Major Cerebral Vessel Occlusion in SLE Due to Circulating Anticardiolipin Antibodies

Stroke due to major cerebral artery occlusion is a recognized but rare consequence of systemic lupus erythematosus (SLE). There are at least 30 reported cases; in all but one, the SLE was active at the time of the stroke. In that patient, the postulated mechanism was in situ thrombosis due to circulating anticardiolipin (aCL) antibodies, but the specific assay was not performed. We have studied a second such patient with an acute hemispheric stroke and angiographically proven large-vessel occlusion in association with inactive SLE, with aCL antibodies as the presumed mechanism.

At age 22, the patient had acute glomerulonephritis due to SLE. His condition improved, but there was residually elevated serum creatinine (2.8 mg/dL) and proteinuria (500 mg/24 h). He first developed hypertension and had a right frontal headache in June 1993. One month later he suddenly had left hemiplegia. At his local hospital, a brain computed tomographic (CT) scan showed a large superficial and deep infarct in the territory of the upper and lower
Migraine equivalent and hemorrhagic infarction.
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