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 1) 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 glittering 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 neuroradiologically. 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 hemianopia 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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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
Left, Lateral subtraction cerebral arteriogram of right internal carotid artery shows absence of Sylvian branches of middle cerebral artery (MCA). Right, Frontal view shows complete occlusion (arrow) of proximal MCA at its origin.

divisions of the right middle cerebral artery (MCA). A CT scan 1 week later showed spontaneous hemorrhagic transformation in the absence of clinical worsening. Magnetic resonance angiography revealed a right MCA stem occlusion. The patient was transferred to Columbia-Presbyterian Medical Center, where neurological examination showed a left homonymous hemianopia, left central facial plegia, dysarthria, and left hemiplegia. There was left-sided sensory loss, extinction to double simultaneous stimulation, and asterognosis. Deep tendon reflexes were hyperactive on the left side. Hoffmann’s and Babinski’s signs and ankle clonus were present on the left side.

The aCL-IgG isotype titer was 60 GPL (normal range, 0 to 10). The serum antinuclear antibody titer was 1:320 (homogenous), and the anti-DNA antibody titer was 371 U/mL (normal positive active range, 0 to 249), with normal total hemolytic complement, Westergren erythrocyte sedimentation rate, partial thromboplastin time, and cerebrospinal fluid.

Selective cerebral angiography performed 1 month after the stroke showed total occlusion of the right proximal MCA at its origin (Figure), with retrograde filling of distal branches. There was no angiographic evidence of vasculitis or abnormalities of any other blood vessel. A standard resting electrocardiogram (ECG) and transthoracic and transesophageal echocardiogram, including color-flow Doppler and saline contrast studies, were normal. The patient was treated with intravenous heparin after effective control of his blood pressure. He was then given warfarin for long-term (and probably life-long) oral anticoagulation.

Elevated aCL titers are an independent risk factor for stroke.4 The frequency of aCL was 18% in unselected young stroke patients.5 As many as two thirds of patients with SLE show aCL even in the absence of lupus anticoagulant.6 Further, the tendency for vascular thrombosis may be independent of the disease activity of SLE,7 making screening for aCL important in all patients with known SLE.

Oclusive disease of the large cerebral vasculature in active SLE is most often due to cardiogenic embolism from a left-sided valvular disease, inflammatory or noninflammatory vasculopathy, cervical arterial dissection, and accelerated atherosclerosis.5 The mechanism of vascular occlusion in our patient cannot be known with absolute certainty; however, the absence of a cardiac abnor-
mality clinically (by ECG and echocardiography), with normal ESR, prodromal headache, hypertension, and proximal location and persistence of the lesion, made in situ thrombosis most likely.

The factors responsible for the mechanism of thrombosis in aCL have been recently reviewed.9 β-2-Glycoprotein I (β2GPI) seems to be an immunogen for the production of antibodies to cardiolipin. The immune response directed against β-2-GPI likely results from increased binding of the protein to anionic phospholipid surfaces; that process is amplified by local vascular endothelial cell damage.

There is uncertainty about the best therapy for stroke associated with aCL antibodies. Unanswered questions include the role of antiplatelet agents either with or without associated SLE, as well as the intensity and duration of anticoagulation. A prospective collaborative study is underway between the Antiphospholipid Antibodies in Stroke Study (APASS) and the Warfarin Antiplatelet Recurrent Stroke Study (WARSS) to determine the outcome of patients with aCL and stroke who are treated with aspirin or warfarin. Until these results are known, treatment decisions will continue to be empiric, based on limited retrospective12 or prospective13 data and incomplete understanding of the pathophysiology of this condition.

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was to "classify headache appearing in stroke patients ... using

6.2.1.2 indicates "headache associated with intracerebral he-

6.1.2.1 indicates "headache associated with thromboembolic
disease" and "intracranial hematoma" are coded to groups 6.1 and

vascular disorder are coded to group 6," termed "headache

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Headache in Stroke: Use of the International
Headache Society Classification

I wish to comment on certain aspects of the article "Headache
in Stroke" by Vestergaard et al.1 According to the classification
of the International Headache Society (IHS),2 "patients who
develop a new form of headache (including migraine, tension-type
headache, and cluster headache) in close temporal relation to a
vascular disorder are coded to group 6," termed "headache
associated with vascular disorders." Because the aim of this article
was to "classify headache appearing in stroke patients ... using
the IHS criteria," so the IHS criteria were indeed
followed.

1. Concerning the IHS classification of headache in stroke, we
are well aware that headache with migraine or tension-type
features are coded in different groups regarding if the headache
related to the vascular episode appears for the first time or is a
worsening of a preexisting headache. Patients were classified
(Table 3) according to this, so the IHS criteria were indeed
followed.

2. We looked at headache from 3 days before to 3 days after the
stroke for two main reasons. (a) A small time span is, of course,
necessary to be sure that the headache was indeed related to the
vascular disorder and did not occur for any other reason. (b) Had
a longer time span been chosen (eg, 14 days, which may rarely
occur in stroke patients according to the IHS criteria), patients
may have had difficulty remembering whether or not the headache
was associated with the vascular accident.

3. It would, of course, be interesting to know the frequency of
carotid dissection in the present patient material, but for practical
reasons it was not possible to perform an ultrasonographic
examination in all patients. Such a measure is necessary to establish the
diagnosis reliably. We would like to add that although interesting,
carotid dissection was not an aim of our study.

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Lobar Hemorrhages

In his editorial in the April issue of Stroke,1 Dr Molinari
discussed some controversies about the mechanisms and causes of
lobar intracerebral hemorrhage (ICH). I would like to comment on
two issues raised by the author. (1) The relationship between

matoma presenting with tension-type headache features." Wors-
ening of preexisting headache is coded to preexisting headache
form.

The possibility of adding the fourth digit to specify the type of
headache was mentioned by the authors in a misleading way. Indeed,
the diagnostic criteria provided by the IHS were not followed. Headache associated with acute ischemic cerebrovascular
disease may "begin as long as two weeks after the stroke." However, in the present study the patients were interviewed about
headache occurring by up to 3 days after the acute event. No
information on the status of carotid arteries was given. This would
have been particularly important in patients with lateralized
headache and ipsilateral infarct to exclude carotid dissection
(coded to group 6.4.2).

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Response

It is a pleasure for us to have the opportunity to answer the
questions raised by Dr De Marinis. We do agree with some of the
points raised by the author but would like to add the following
about the use of the IHS classification of headache.

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Major cerebral vessel occlusion in SLE due to circulating anticardiolipin antibodies.
D S Younger, R L Sacco, A G Khandji, G B Appel, I A Jaffe, S R Levine and P Mitsias

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