This case raises the more general issue of the frequency, characteristics, and mechanisms of transient episodes preceding an hemorrhagic stroke. Indeed, transient neurological deficits, possibly resulting from a concurrent cerebral ischemia related to the underlying vascular disease, have been reported to herald an ICH in about 7% of patients. Taking for granted that a subclinical cerebral ischemia can trigger epileptic seizures, we suggest that the same determinants might be responsible for both paralytic and irritative manifestations, although seizures are less common than transient neurological deficits, and both are more likely to occur before a cerebral infarction than before a cerebral hemorrhage. As to the latter, the role of amyloid angiopathy should be emphasized, as well as that of atherosclerotic disease, because amyloid angiopathy may be responsible for both ischemic and hemorrhagic damage in the same patient.

In fact, our patient bears some similarities to another patient, described by Smith et al., in whom surgery revealed pathological features of amyloid angiopathy, and even more with patient 3 in the report by Greenberg et al. The latter patient, who was clinically diagnosed as having amyloid angiopathy, presented with transient visual spells regarded as epileptic seizures possibly due to a small hemorrhage that may have heralded a larger one; this hypothesis, however, was not verified because neuroimaging was available only after the stroke. In our patient there was no CT or MRI evidence of hemorrhage at the time of seizures, which indirectly favors the role of an ischemic mechanism rather than that of petechial hemorrhages. He may have amyloid angiopathy, although a 5-year follow-up indicated neither recurrences of ICH nor dementia. The existence of a cryptogenic haemartoma too small for angiographic detection, though unlikely, cannot be definitely ruled out since pathological findings were not available.

In conclusion, the occurrence of seizures prior to a stroke does not necessarily imply that a cerebral infarction has taken place. In fact, seizures heralding an ICH, though uncommon, do occur. Although the mechanism is still obscure, the triggering role of a subclinical ischemia related to the underlying vascular disease should be considered, at least in principle.

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Silent Infarctions in First-Ever Stroke Patients

To the Editor:

We read with interest the article by Jorgensen et al about silent infarction in acute stroke patients and wish to congratulate the authors for this very important and stimulating contribution. They are kind enough to quote our own work on the same topic, making interesting comparisons; however, they make two small mistakes that we would like to correct.

(1) They write that we defined a poor outcome as “death or persistence of physical handicap.” Actually, we defined handicap (both physical and functional) in terms of the Rankin Scale, a validated method for measuring the outcome of stroke. We agree that “smaller differences in outcome may not be recognized with these end points,” but this is probably also a problem with the Barthel Index, which Jorgensen et al used.

(2) They write that “only 56% of the patients had CT [computed tomographic] scan,” but in fact all 209 patients we studied were scanned within 30 days after stroke. They represent 56% of the registered stroke cases in the Studio Epidemiologico sull’ Incidenza delle Vascularopatie Acute Cerebrali (SEPIVAC) study, and 96% of the patients with a definite cerebral infarction; these figures are available in our paper. We could not have discussed silent infarctions in patients without a CT scan.

Apart from these two points, we fully agree with the conclusion by Jorgensen et al: in community-based studies (ie, in the “real world”) there is no evidence at all that the presence of these silent lesions contribute to a worse prognosis of patients with a first-ever ischemic stroke.

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As to the concluding remark by Ricci et al in their letter, we agree that firm evidence supports the conclusion that the presence of a silent infarction does not influence the prognosis of the stroke patient in terms of mortality from the stroke, rate of discharge to nursing home, speed of recovery, and neurological as well as functional ADL (activities of daily living) outcome. This lack of influence is noted regardless of the age of the patient, his or her prestroke condition, and the severity of the stroke. Whether a silent infarction has a subtle, negative influence on the intellectual performance of the stroke patient is, however, still unsettled.

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Functional Recovery on Stroke Units

To the Editor:

I wish to comment on three aspects of the randomized trial by Kalra.1

Although the two groups of elderly subjects with an intermediate prognosis after stroke, treated in a stroke rehabilitation unit or in medical wards, had the same median initial Barthel score, the group treated in the stroke unit had a higher initial range of Barthel scores (0 to 12) than the group treated in medical wards (0 to 8). Because initial Barthel score is a powerful predictor of later scores,2 it is possible that a difference in functional status at baseline contributed to the difference between the groups' subsequent scores. Kalra could dispel this concern by showing that mean initial Barthel scores were similar in the two groups and that on multivariate analysis the variable treatment group (stroke unit or medical ward) remained a significant predictor of later scores when initial score was included as a dependent variable.

Kalra1 demonstrated that the stroke unit was associated with a significantly shorter mean length of stay than the medical wards (48.7 days and 104.6 days, respectively). Efforts to improve the efficiency of stroke management are meritorious. However, lengths of stay in both groups are greater than in a group of elderly subjects admitted to our hospital with stroke in 1993. Because I do not use Orpington scores the cohort could not be matched according to such scores. The subjects had survived for more than 30 days and had a Barthel score of 0 to 9 at 7 days after stroke; their median Barthel score of 4 was the same as the mean score of Kalra's subjects, and their mean age (77.8 years) was similar. Ninety percent were transferred to our stroke rehabilitation unit and the rest were treated exclusively on medical wards. The mean length of stay in the hospital was 32.5 days, 16 days shorter than that of Kalra's group treated in a stroke rehabilitation unit.

Kalra stated that mean length of stay could be "artificially shortened by discharging stroke patients before they achieve their

Response

We thank Dr Ricci and his colleagues for their kind letter and for this opportunity to discuss their objections.

Regarding their first point: In a proper evaluation of the possible impact of silent infarctions on the outcome of stroke, several conditions have to be fulfilled: First, are the patients with and without silent infarctions otherwise comparable, particularly in terms of prestroke condition and initial stroke severity? If not, a bias will be introduced when outcomes are compared. In the SEPIVAC study possible differences in initial stroke severity between the groups did not seem to be considered.1 This introduces a bias to the outcome evaluation of the study because patients with silent infarctions tend to have smaller symptomatic infarctions (and less severe strokes) than stroke patients without silent infarctions.2 Second, outcome measures should be calibrated to what one expects to find. The nature of silent infarctions is that they are indeed silent, probably because the brain volume lost is too small to produce symptoms. If silent infarctions influence the stroke patient's recovery, this influence should be expected to be small. Nonetheless, in the SEPIVAC study, a subject's death or dependence at 3 months, the latter as defined by the Rankin Scale, was used to evaluate a possible impact of silent infarctions on stroke outcome. However, few people would suspect that mortality from stroke is influenced by the presence of a silent infarction, or that a silent infarction would make the difference between being dependent or independent after stroke. The results of the SEPIVAC study must therefore be interpreted with caution. In the Copenhagen Stroke Study3 we analyzed both neurological (using the Scandinavian Neurological Stroke Scale) and functional (using the Barthel Index) outcomes quantitatively, so a much more detailed analysis could be performed. Furthermore, we used multivariate analysis to eliminate bias from possible confounding factors such as differences between the groups in initial stroke severity.

Regarding the second point made by Ricci et al: The SEPIVAC study is a community-based study comprising 375 stroke patients, and an interesting paper has been published about the stroke incidence in the Umbria area.3 However, only 209 (56%) of these patients were included in the SEPIVAC study of silent infarctions.1 One hundred and sixty-four of the 375 were excluded because they did not have a CT scan. This large number of excluded patients raises serious doubt about whether the use of the label "community-based" to the SEPIVAC study is justified. The low number of patients scanned introduces a selection bias that makes it difficult to interpret results as they may or may not apply to the whole population. Therefore, we feel that the authors should have avoided the use of the term "community-based" about this part of their study.

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Silent infarctions in first-ever stroke patients.
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