


Response

We would like to thank Dr Alpert for his comment on our article, in which we demonstrated that extracranial carotid atherosclerosis is increasing in Japanese patients with brain ischemia.2 We also reviewed some vascular risk factors possibly related to our findings; these include hypertension, diabetes mellitus, ischemic heart disease, and hypercholesterolemia. This list of risk factors is rather incomplete because of the failure to obtain thorough medical histories from the early patients (1963 to 1965). We are aware that some important risk factors, including smoking, are missing from our analysis, and hence we do not conclude that diabetes mellitus is the only risk factor responsible for the change in the rate of extracranial cerebral atherosclerosis in the Japanese population. Accordingly, we respect the comment made by Dr Alpert, and do not exclude the possibility that smoking played an important role in the increase in extracranial carotid atherosclerosis revealed by our analysis.

Nonetheless, there is some evidence against the involvement of smoking in the increase in extracranial carotid lesions in Japan. First of all, the proportion of smokers in the Japanese population overall has been decreasing during the past 30 years.22 Although we do not know the exact proportion of cigarette smokers among our patients, there is no reason to believe that smokers were preferentially incorporated into the recent group of subjects or were excluded from the early group. Second, in Japan approximately five times as many men as women smoke,2 and the early group consisted largely of male patients. These factors, taken together, lead us to suppose that the proportion of smokers was higher in the early group than in the recent group. Finally, the frequency of extracranial carotid lesions was similar in men and women in our analysis, despite the difference in smoking rates between the sexes. These lines of evidence apparently contradict the notion that smoking is responsible for the increase in extracranial carotid lesions in our study.

According to the results of our population-based epidemiological study in a Japanese rural community, Hisayama, rates of hypertension and smoking have decreased in recent years whereas those of hypercholesterolemia, obesity, and glucose intolerance have significantly increased.2 Such metabolic disorders appear to play more important roles than ever in the development of ischemic stroke in Japan. There may be subtle differences in the risk factor profiles for stroke between different races.

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References


Anterior Choroidal Artery Territory Infarcts

To the Editor:

I read with interest the study on anterior choroidal artery territory infarcts by Leys et al1 in the April 1994 issue of Stroke. The authors studied the causes of anterior choroidal artery territory infarcts in 16 patients and came to a different conclusion than my colleagues and I did in a study of 31 patients.2 We concluded that anterior choroidal artery territory infarcts are usually caused by small-vessel disease, while Leys et al concluded that infarcts in this territory are rarely related to small-vessel occlusion. This disparity in findings can be explained largely by differences between the patients in these two studies. We included only patients with a solitary infarct in the anterior choroidal artery territory, whereas 6 of the 16 patients Leys et al included had associated infarcts in other vascular territories. Without these 6 patients, the results of the studies would be similar. Of the 10 remaining patients in the study by Leys et al, there would be 7 with small-vessel disease and 3 with carotid artery disease as the most likely cause of anterior choroidal artery territory infarction. In none would cardioembolism be the most likely cause.

I agree with Leys et al1 that patients with an anterior choroidal artery territory infarct, including those without an associated infarct in another vascular territory, should be evaluated to determine any possible causes of the infarct.

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References


Response

We are grateful to Dr Bruno for his interesting remarks about our study.1 He and his coworkers came to the conclusions in their study that most anterior choroidal artery territory infarcts are due to small-vessel disease, and that associated large-vessel atherosclerosis and potential cardiac sources of emboli are rare and may be coincidental findings.2 Our study led us to draw the conclusion that small-vessel occlusion probably accounts for a minority of anterior choroidal artery infarcts. Dr Bruno points out that the discrepancy between the two studies can be explained largely by differences between the inclusion criteria. We entirely agree with him that including 6 patients with associated infarcts in other vascular territories may have skewed our results towards a higher rate of large-vessel atherosclerosis or cardioembolism. Our criteria for small-vessel occlusion differed from those of Bruno et al, who considered as having “small-vessel disease” patients who had no recognized cause of stroke; this definition probably led to an overrepresentation of small-vessel occlusion in their study population. Moreover, Bruno et al did not perform an echocardiography in all patients, and therefore may have underestimated the prevalence of cardiac sources of stroke. To define “small-vessel occlusion” we used criteria defined by two of Bruno’s coauthors.3 These criteria may be too strict and may have led to an underestimation of the prevalence of small-vessel occlusion. However, as we explained in the last part of our discussion, no more than 1 of the 7 patients in the “undetermined cause of stroke” group might actually have had small-vessel occlusion, although this patient

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did not meet the criteria: this point was detailed in the fifth paragraph of the discussion. According to our criteria it is not true that after exclusion of 6 patients with associated infarcts, 7 of the 10 remaining subjects fulfilled criteria for small-vessel occlusion: none of these 10 did. Only 1 patient (of 16) met criteria for small-vessel occlusion in our study; this patient had an associated ipsilateral thalamic infarct (see Table 1).

Dr Bruno agrees with the general conclusion of our study: the rule “anterior choroidal artery territory infarct = small-vessel occlusion” is not true for all patients, and patients with such infarcts should be systematically evaluated for possible large-vessel disease and cardioembolism. This remains the finding most important to determining the best therapeutic strategy for the secondary prevention of stroke in these patients.

**References**


**Thrombosis and Endothelial Injury**

**To the Editor:**

The recent article by Rote et al. is extremely important. In it the authors discuss possible reasons for discrepancies between ex vivo or in vitro data and in vivo results using models of thrombosis. They also discuss differences between human drug trials and the data from animal models, including the model they used. A keynote of these comparisons was the effect, or lack thereof, of treatments designed to inhibit platelets, with the focus on inhibition of platelet GPIIb/IIIa. Platelets were activated by endothelial denudation, with exposure to subendothelial collagen (or collagen-bond von Willebrand factor) as the activating step leading to platelet aggregation without endothelial denudation. Additional useful information may be gained by studying models of platelet aggregation that depend on endothelial dysfunction rather than denudation to initiate the thrombotic event.

**Stroke Scale Comparisons**

**To the Editor:**

de Haan and coworkers analyzed five stroke scales and correlated them with the Barthel Index, the Rankin scale, and the Sickness Impact Profile in 87 patients examined 6 months after stroke. One of the scales evaluated was the Scandinavian Stroke Scale (SSS). However, in their use of this scale certain infelicities have crept in. Contrary to what de Haan et al state, the prognostic score of the SSS does not include gait, but only consciousness, gaze, and arm and leg strength. The total obtainable score is 22. In Table 1 the mean value of the prognostic score of the SSS is 50.94, indicating that they must have misunderstood the design of the scale. In addition, the prognostic score is meant to be used for stratification in the acute phase of stroke and not at follow-up. At 6 months consciousness is usually normal and there is no gaze palsy. Therefore, evaluation of consciousness and gaze palsy does not contribute any additional information at follow-up, as is indeed demonstrated in the article: the correlation coefficients for the comparisons of the Barthel Index score with the SSS prognostic score and long-term scale are identical. The authors question the value of the prognostic score on the basis of use of the scale at a point in time not appropriate for prognosis.

The long-term part of the SSS includes gait, as well as several other items, and has a maximal score of 48 points. de Haan et al analyzed the long-term score with and without what they call “functional items,” which they do not explicitly describe. From the context we infer that one of them must be gait, but it is unclear whether other items were also excluded. In our opinion a stroke scale should be used according to its original design. If certain items are left out, it is another scale and can no longer be called by its original name. Gait is included in the SSS because it is an integral part of the neurological examination and because it is of utmost importance to the patient.

Opinions may differ as to the relevance of stroke scales as such. The relevance of comparing stroke scales with the Barthel Index can also be questioned. The SSS is meant to measure neurological restitution or deterioration from the acute phase until the end of some observation period, and is not to be used solely at the end of that period. It was not intended to be a replacement for the Barthel Index, which is has to do with activities of daily living and which measures the ability of the patient to cope with neurological deficits. Rather, the two scales were intended to evaluate different aspects of brain damage. However, we thank our colleagues for demonstrating an excellent correlation between the Barthel Index and the SSS 6 months after stroke.

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