Letters to the Editor

1885

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 I).

Dr Bruno agrees with the general conclusion of our study: the rule "anterior choroidal artery territory infarction = 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 most finding important to determining the best therapeutic strategy for the secondary prevention of stroke in these patients.

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References

Thrombosis and Endothelial Injury

To the Editor:
The recent article by Rote et al1 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 their exposure of GPIIb/IIIa. In the accompanying editorial comment by Feuerstein,2 the importance of endothelial breakdown and exposure of platelets to collagen is again discussed.

I wish to point out that the traditional emphasis on denudation as the starting point for platelet adhesion/aggregation continues to detract attention from the fact that endothelial cell dysfunction can lead to platelet adhesion and subsequent aggregation and degradation without any breakdown of the endothelial cell barrier to the basal lamina. In fact, adhesion/aggregation may lead to rather than result from denudation.3-5 In models with denudation as the cause of adhesion/aggregation, results of treatment may not parallel results in certain other models or in human disease because in the latter cases endothelial denudation is not the sole or even the primary cause of adhesion/aggregation. 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.

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References

Stroke Scale Comparisons

To the Editor:
de Haan and coworkers1 evaluated 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).2,3 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 their Table I the mean value of the prognostic score of the SSS is 50.94,1 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 score 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 al1 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 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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