vious data, we have performed a Cox multivariate analysis of the probability of survival without stroke in 92 TIA patients with normal CT scan and in 24 TIA patients with cerebral infarction followed during an average period of 21 months. Age, carotid lesions detected by ultrasonographic examination or angiography, and glycaemia level higher than 100 mg/dl were used as covariates since in a previous study we observed that these variables were related independently with the prognosis. Ninety nine per cent of the TIAS without infarct and 79% of the TIAS with infarct survived without suffering severe functional damage (Mantel-Cox test p = 0.0001). The probability of survival, free of stroke, was related with the presence of carotid atherosclerosis (odds ratio = 3.07, 95% confidence intervals = 1.27-7.40). Ischaemic lesions in the CT scan, age, and glycaemia levels had no independent predictive value.

Our results suggest TIAS with cerebral infarction as a poorer outcome. There is a higher frequency of atherosclerosis of the neck arteries.

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Koudstaal and van Gijn reply:
We would like to thank Dr Dávalos et al for their comments on our study.1 Their observation of a higher risk of stroke in TIA patients with a cerebral infarct on CT in a univariate, but not in a multivariate analysis, is very interesting.2 We have recently completed a study of predictors of major vascular events in 3150 patients with a TIA or minor stroke who were entered into the Dutch TIA Study.3 In contrast to the findings of Drs Dávalos et al, we found that ischaemic abnormalities on CT scan were an independent risk factor for stroke and other vascular events, irrespective of the duration of the symptoms. In this multicentre study, however, it was impossible to collect objective information on the presence and degree of atherothrombotic abnormalities of the extracranial bloodvessels, which may explain the discrepancy between our findings and those of our Spanish colleagues.

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Matters arising


Kiers, Davics, Larkins et al reply:
We thank Drs Murros and Fogelholm for their comments. The relationships between diabetes, stress hyperglycaemia and stroke outcome are indeed complex. In our study design, we attempted to examine separately the effects of diabetes and stress hyperglycaemia on stroke outcome, compared with the hyperglycaemia non-diabetic group, using the four groups categorised on the basis of history, fasting glucose and glycosylated haemoglobin.1 We acknowledge that stress hyperglycaemia was also likely to be present in the non-diabetic patients, but these subjects could not be identified.

Our study demonstrated significantly higher mortality in the combined diabetes groups (as well as the stress hyperglycaemia group) compared with the diabetes, non-diabetic subjects, but we would agree that this adverse effect could have been due to stress hyperglycaemia in a proportion of the diabetic patients.

We are aware that there have been animal studies of cerebral ischaemia which have suggested that hyperglycaemia may be protective,4,5 but there is also substantial evidence to the contrary. This appears clearly shown by the results of our study, and previous human investigations,6 is that elevated blood glucose is associated with a worse outcome after stroke. This association was even present within our euglycaemic group. As discussed in our paper, it is not possible to conclude whether this is a causal relationship or whether the degree of hyperglycaemia reflects the severity of the acute event. Until the mechanism of the association can be resolved, however, glucose infusions should be avoided in acute stroke.

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