Changes in insular infarcts

Cardiovascular and metabolic changes in insular infarcts: a delayed phenomenon?

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Aggressive monitoring of electrocardiogram, blood pressure and blood glucose levels after 72 h in patients with insular infarcts is crucial in identifying whether they are prone to develop arrhythmias, hyperglycaemia and hypertension after the onset of stroke.

Infarction of the insular cortex may be associated with autonomic sympathetic activation, leading to repolarisation changes and arrhythmias.¹ Moreover, the involvement of the insular cortex in the infarct area has been reported to be associated with a higher frequency of hyperglycaemia, and this association does not depend on the volume of the lesion, or pre-existing diabetes mellitus.² Patients with insular infarcts also show a marked increase in nocturnal blood pressure, and higher serum norepinephrine concentrations.³ By contrast, in the article by Pettersen *et al*⁴ (see p 885), insular infarcts are not associated with higher levels of blood pressure or serum glucose. How may one reconcile these differences?

The Pettersen study does have a methodological limitation: as all the patients underwent thrombolytic treatment, patients with systolic blood pressure higher than 180 mm Hg or blood glucose levels higher than 8.88 mmol/l were excluded. This limitation, however, is unlikely to explain the differences between the results of the Pettersen study and those of previous studies.

A major strength of this study is that all the patients were assessed within 3 h

after the onset of stroke, but if autonomic changes are the consequence of the insular lesion, they are likely to appear with some delay after the onset of stroke, and not immediately. The lack of association between insular infarcts and high blood pressure or serum glucose levels may be because of including patients within 3 h of the onset of stroke—that is, before the changes can occur. Similar results have been found in the Prevention of Ischemic Events by Early Treatment of Cerivastatin Study,⁵ in which an analysis of the electrocardiogram (ECG) recorded at admission, with a median delay of 3 h after the onset of stroke, did not show any association between the presence of insular infarcts and ECG changes.¹ The results of Prevention of Ischemic Events by Early Treatment of Cerivastatin Study, compared with those of previous studies, in which ECG changes were recorded up to 72 h after the onset of stroke, suggest that ECG changes have a delayed appearance.

Thus, the study by Pettersen *et al*⁴ and the Prevention of Ischemic Events by Early Treatment of Cerivastatin Study⁵ share the same potential methodological weakness: in both studies, patients were

not re-evaluated during the 72 h after admission. This would have been necessary to prove the delayed occurrence of ECG, blood pressure and blood glucose level changes in patients with insular infarcts. Further studies are needed to confirm the hypothesis that autonomic sympathetic changes, not yet present in the first 3 h after stroke, occur later. This is crucial to help identify patients who are prone to develop arrhythmias, hyperglycaemia and hypertension after stroke. Hence, a more aggressive monitoring of these variables should be undertaken. This is an important issue, because these complications are likely to worsen cerebral ischaemia in the penumbra area.

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