receiving therapy). This suggests that diabetes may contribute to mortality in the acute stroke period but not to that remote-from-stroke occurrence when other factors may be operating. This effect was unrelated to stroke severity on admission, as this did not differ between the diabetic and nondiabetic groups.

The disparities between the two studies may well represent methodological differences in the assay or possibly a demographic difference. Findings very similar to ours have been reported in a group of acute myocardial infarction patients who were drawn from the same population as that from which this stroke group came.\(^\text{3}\) Woo et al\(^\text{4}\) failed to show an association between mortality and HbAlc levels. However, their study referred largely to a Chinese population in which intracerebral hemorrhage was over-represented compared with an occidental population. Diabetes does not appear to be a risk factor for this phenomenon.\(^\text{5}\) Finally, we excluded patients receiving intravenous glucose therapy, I am unaware of the status of such patients in Murros and Fogelholm's study.

In conclusion, our results support the suggestion that those patients whose admission glucose levels are above 11 mEq/L are likely to be diabetic, either known or hitherto undiagnosed, and that these values are not due to a stress response. Few patients with normal HbAlc values after stroke will have even modestly elevated blood sugar levels (between 7–11 mEq/L). Mortality is significantly correlated with elevated glycosylated HbAlc values and thus with the presence of an antecedent state of glucose intolerance.

Stephen Oppenheimer, MB, MRCR, FRCP(C)
The John P. Robarts Research Institute
London, Ontario
Canada

References

Silent Infarcts on Computed Tomography Scans
Are More Common in Cardiogenic Stroke

To the Editor:

Although some 30% of patients with ischemic stroke have a possible cardiac source of embolism,\(^\text{1}\) it remains very difficult in an individual case to decide whether cardiogenic embolism or arterio-artery thromboembolism is responsible for a cerebral infarct. A sudden onset, loss of consciousness, and lack of a preceding transient ischemic attack are all more frequent in cases of cardiogenic embolism,\(^\text{2}\) as are some computed tomographic scan appearances. Hemorrhagic infarcts and cortical lesions are more frequent in cardiac cases,\(^\text{3}\) with the finding of lacunar infarcts making such a source of embolism less likely.\(^\text{4}\)

It is conventional teaching that the diagnosis of cardiogenic embolism is also supported by the development of symptomatic infarcts in multiple sites. In theory, the computed tomography finding of silent cerebral infarcts in nonsymptomatic territories should also be more frequent in those with a central (cardiac) source of embolism.

We therefore reviewed the computed tomography findings in 85 patients examined after their first clinical episode of cerebrovascular disease and related the findings to the clinical evidence of a possible cardiac embolic stroke. Of the 85 patients, 75 had presented with a completed stroke, and 10 had had a transient ischemic attack. Twenty patients had nonhemorrhagic atrial fibrillation and 11 had rheumatic heart disease or recent myocardial infarction (myocardial infarction, \(n=2\)). Fifty-four patients had no evidence of atrial fibrillation, valvular heart disease, cardiomyopathy, or myocardial infarction within the preceding month.

Multiple infarcts affecting the other hemisphere, or the cerebellum in the case of carotid territory strokes, were found in only one of the 54 patients with no clinical evidence of a cardiac source (1.9%), compared with eight of the 31 with such evidence (25.8%). This difference is significant (\(x^2\), 11.8; \(p<0.001\); odds ratio, 0.08; confidence interval, 0.02–0.34).

The data included in the report on silent infarcts published by Chodosh et al\(^\text{5}\) support the findings of this small personal series studied retrospectively. That paper\(^\text{6}\) describes a prevalence of infarcts in nonsymptomatic territories of 34.3% in 70 patients diagnosed clinically as having a cardioembolic stroke compared with a prevalence of 11.4% in 887 patients in all other stroke categories. This difference is highly significant (\(x^2\), 29.9; \(p<0.001\); odds ratio, 0.13; confidence interval, 0.06–0.27).

These data support the view that the diagnosis of a cardiac source of ischemic stroke is supported by the finding of cerebral infarcts in asymptomatic territories. The increased sensitivity of magnetic resonance imaging may be even more helpful.

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M.J.G. Harrison, DM, FRCP
The National Hospital
Queen Square
London, UK

Serum Concentrations of Glyceral With Continuous or Discontinuous Intravenous Administration

To the Editor:

Intravenous glyceral has been studied in controlled trials in patients with acute stroke.\(^\text{1–10}\) A 10% solution of glyceral was administered either continuously (500 mL within 24 hours)\(^\text{1}\) or discontinuously (once daily, 500 mL over 4 hours, \(500\) mL over 1–2 hours, \(500\) mL over 6 hours, \(500\) mL over 2 hours, \(500\) mL over 4 hours)\(^\text{2–4}\) or 1.2 mg/kg body wt over 6 hours\(^\text{5}\). Many trials have yielded favorable results in some or all of the patients studied,\(^\text{1–5,7,9,10}\) while others have shown no benefit.\(^\text{1,2,6,8}\) Glyceral was given to reduce edema formation\(^\text{1–10}\) or to affect cerebral metabolism,\(^\text{1–3,7,9}\) since glyceral favors coupling of oxidative phosphorylation.\(^\text{11}\)

In patients suffering from brain edema, 10% glyceral was infused either continuously (20–60 mL/hr, 28 patients) or 500 mL was administered within 4 hours once daily (15 patients). After at least 24 hours of continuous infusion and at the end of the 4-hour period, we determined serum glyceral concentrations. Serum glyceral concentrations differed among individuals and were not solely related to body weight. The highest concentrations obtained in one patient during continuous administration of 60 mL/hr were 208–303 mg/L (Figure 1). At the end of an infusion of 500 mL glyceral over 4 hours, serum concentrations were 191–923 mg/L (Figure 1).
Silent infarcts on computed tomography scans are more common in cardiogenic stroke.

M J Harrison

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