The following is in reply:

To the Editor:

In response to the comment from J. Vazquez-Cruz, MD, regarding the paper entitled "Effect of Insulin on Acute Experimental Cerebral Ischemia in Gerbils," in our study, every gerbil had blood glucose levels measured at the beginning, immediately before surgery, and on the day of sacrifice. We presented our results in Table 2 of the paper. We do not believe that it is necessary to repeat blood glucose testing within 1 hour after surgery. Rather, it is more important to know the blood glucose level at the time of acute cerebral ischemia since these levels are usually temporarily elevated with surgical stress. This may not have a significant effect on the results of the study anyway because the experimental conditions were the same throughout. It is well known that hyperglycemia and hypoglycemia both have an adverse effect on the ischemic brain. The poor outcome of the gerbils in Group D in our study was apparently due to the adverse effects of hypoglycemia.

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References

Plasma Cortisol as a Measure of Stress
Response in Acute Stroke

To the Editor:

Several studies have suggested that elevated blood glucose levels in acute stroke are related to clinical outcome. This could be a cause-and-effect relationship since there is evidence of aggravated tissue lactacidosis and a poor outcome in hyperglycemic animals subjected to acute cerebral ischemia. The hyperglycemia could also be a reflection of a stress response only coincidentally related to prognosis. Support for the concept of stress changes being linked with outcome results from studies of blood cortisol levels.

In the double-blind randomized trial of dexamethasone carried out in Nottingham, UK, blood cortisol levels were recorded on admission. The relationship between cortisol levels and survival is shown in Figure 1. There was also a relationship between plasma cortisol and the level of consciousness. Thus the 12 deeply unconscious patients had a mean plasma cortisol level of 1,750 nmol/1, in contrast to a mean level of 800 nmol/1 in 36 alert subjects.

These data show that patients with severe strokes who are in coma on admission and destined to die within 1 month show a high plasma cortisol, providing evidence of an increased stress response in such patients. Two previous studies also showed evidence of a greater stress response in stroke patients with poor outcome. Oka showed a higher plasma cortisol in stroke patients who died and a return to normal levels by the second week in those who recovered. Feibel et al found a higher mortality in stroke patients with high 24-hour urinary catecholamine levels, and their cortisols were also elevated when compared with those with low catecholamine excretion rates. Woo et al also noted that fructosamine and glycosylated hemoglobin levels, which reflect prestroke glucose levels, did not correlate with outcome.

Only interventional studies will reveal whether a cyclical process could occur with stress hyperglycemia due to severe ischemic insult that aggravates the tissue metabolic damage, thereby increasing tissue destruction. In the absence of such information, epidemiological evidence that hyperglycemia is linked to outcome should not yet be interpreted as proof of a cause-and-effect relationship.

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