Effect of Hyperglycemia on Infarct Size After Cerebrovascular Occlusion in Cats

To the Editor:

The recent paper, "Hyperglycemia Decreases Acute Neuronal Ischemic Changes After Middle Cerebral Artery Occlusion in Cats," merits discussion since its results directly contradict the findings of our previously reported study. The findings of the two studies were opposite, that is, hyperglycemic compared to normoglycemic middle cerebral artery (MCA) occlusion resulted in significantly less damage in Zasslow's study, while in ours it resulted in more.

The morphometric methods used by the two studies to assess ischemic tissue damage differed. Thus, Zasslow et al determined the percentage of acute (6-hour) cortical ischemic tissue changes as evaluated from a single coronal brain section, while de Courten-Myers et al determined the percentage of the MCA territory that was infarcted in animals after 2 weeks of survival as assessed from six representative coronal sections throughout the brain. Although the former method ignores white matter and basal ganglia involvement and depicts only two-dimensional cortex damage, our testing of the relation between extents of cortical infarct at the midchiasma level (as percentage of cortex present at that level) and three-dimensional infarct measurements (as percentage of hemispheric volume) in 25 cats shows the two to be highly correlated (r=0.93, p<0.001). Thus, differences in morphometric methods seem not to explain the differences in results.

Perhaps the reason for the discrepancy in results between the two studies is brain pathologic assessment following markedly different durations of survival after blood vessel occlusion. Zasslow et al sacrificed their animals after 6 hours compared to our 2 weeks. However, another study has demonstrated that restoring blood flow by clip release after 4 hours of MCA occlusion leads to a markedly different outcome from permanent occlusion. About one half of hyperglycemic and 92% of normoglycemic cats showed no or minimal infarcts (<1/30 the mean infarct size after permanent occlusion), while 54% and 8%, respectively, died acutely after clip release from total territory infarction, hemispheric edema, and brain stem compression. The death rate from edema increased significantly over that following permanent occlusion. Thus, defining cortical morphologic changes as early as 6 hours after occlusion seems not to differentiate reliably reversible from irreversible damage. It follows that Zasslow's study fails to demonstrate that elevated serum glucose concentrations confer a lasting protective effect in focal ischemia.

Yet even if not indicative of permanent tissue damage, Zasslow's study demonstrated less acute morphologic alterations in animals receiving 50% glucose infusion throughout the 6 hours of MCA occlusion compared to animals receiving hypotonic (0.45%) saline. Because a 50% glucose solution is about 9X higher in its osmolality (2,780 mosm/l) than plasma, the infusion of such a hypertonic solution may have temporarily reduced brain edema—a major component of acute tissue change.

When comparing osmotic stimulation, the two studies differ markedly. Zasslow et al administered 155 mosm/kg body wt over 6 hours and 5 minutes, while we administered only 23 mosm/kg over 7 hours. Because hyperglycemia's detrimental effects on markedly ischemic brains can be identified only after more prolonged survival, the extent of acute morphologic changes, the appearance of which may be reduced osmotically, are probably irrele-
References

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