vasomotor function and lipid metabolism of the arterial wall. It is our hope that a genetic profile of critical mutations may provide a portraiture of stroke-prone subjects for whom precise interventions to prevent strokes can be designed. Studies we are pursuing involve the gene defect in the angiotensin-converting enzyme, specifically the double alu-sequence deletion in the 16th intron; lipoprotein (a) analyses; apoprotein E_; and factors influencing the proliferation of cerebral arterial smooth muscle and endothelial cells.

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Cerebral Blood Flow and Poststroke Depression

I read with interest the article by Yamaguchi et al concerning cerebral blood flow (CBF) and poststroke depression. The authors demonstrated an inverse relation between depressive scores and regional cerebral blood flow (rCBF) in patients with poststroke depression. Furthermore, they have shown improvement in scores of depression that correspond with increases in rCBF, raising the possibility that CBF could be etiologically linked with poststroke depression. However, I would like to raise three pertinent issues that deserve to be addressed before validation of the association between impaired CBF and poststroke depression.

First, alterations in CBF in distant areas of the brain have been reported in patients after focal cerebrovascular lesions (diaschisis), which suggests that changes in CBF are not specific to poststroke depression. Second, CBF tends to improve over a period of time beginning with the second week, with a gradual return to normal or near normal several months after vascular injury. This improvement indicates that a time factor may affect cerebral circulation and depressive scores. Third, it is difficult to conclude from the present study whether circulatory disturbance associated with poststroke depression is the cause or the effect. In light of the above-mentioned limitations of the present study, I would like to suggest a few methodological approaches that might help to establish etiologic significance between CBF and poststroke depression.

To control nonspecific changes in CBF and time factor, a comparative study of rCBF in depressed and nondepressed poststroke patients would be in order. A prospective follow-up study with initial assessment of rCBF in all stroke patients and comparison between the rCBF values of stroke patients who develop depression and those who do not would help to establish the role of rCBF in the pathogenesis of poststroke depression. Furthermore, whether the circulatory disturbance is a trait or state marker could be demonstrated by whether abnormal circulation is reversible with antidepressant treatment. A third approach would be to compare the efficacy of vasodilators with antidepressants and placebos. Finally, a fourth strategy would be to examine whether the prophylactic use of vasodilators in poststroke patients would decrease the incidence of poststroke depression.

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Response

We appreciate the opportunity to respond to Dr. Ramasubbu regarding our recent report.1 Diaschisis is a well-known phenomenon in stroke patients, although the mechanism is still controversial.2 We did not mention that the reduction of cerebral blood flow is due to specific or poststroke depression. Rather, our argument is that depression may be based on the reduction of cerebral blood flow (CBF) whatever the reason for CBF reduction. Diaschisis may be one of the major reasons for the reduction.

We agree with his point that a time factor contributed to changes in both CBF and depression score. However, our point is whether two variables were independently or interactively affected by the time factor. Our longitudinal data suggest that the latter is the case.

It is a statistical rule that data from correlational research can be interpreted only in causal terms based on some theories that we have, and correlational data cannot conclusively prove causality. We noted a caution in drawing conclusions from our correlational data, but in conjunction with the data from other lesion studies,3 our data suggest that decrease of regional CBF in certain brain area contributes to depression.

His proposals should be appreciated for future studies, but at least some of his suggestions have been already considered in our report. First, we did not exclude nondepressed patients from the study. We had criteria for selecting subjects, but those were for avoiding effects of medication or dementia and not for excluding nondepressed patients. Second, we used a type of prospective follow-up in our study. Some patients developed depression and some showed alleviation of depression during the follow-up period in association with the changes in regional CBF, which were shown in Figures 2 and 3 in our article. Experimental studies using medications such as antidepressants and vasodilators would provide further evidence for elucidating the mechanisms underlying poststroke depression.

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