from Drs Swanson and Sharp points out, our equation (Leach et al) will provide a better correction if edema occurs outside the infarcted region. Data calculated using both equations and either area-by-area correction or total volume figures still demonstrate the efficacy of BW619C89 in this rat model of focal ischemia.

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References

Risk Factors for Cervical Atherosclerosis in Patients With Transient Ischemic Attack or Minor Ischemic Stroke

Palomaki et al report a relationship between symptomatic carotid stenosis and traditional vascular risk factors. Age, smoking, hypertension, serum triglycerides, regular light alcohol consumption (inverse association), and body mass index (marginal inverse association) were independent determinants of the presence of atherosclerosis. On the other hand, age and the ratio of high-density lipoprotein to total cholesterol (inverse association) were associated with the severity of extracranial carotid stenosis. Current smoking and female sex were predictors only of the percent stenosis and the length of the lesions, whereas hypertension showed a significant association only with the length of lesions. Such a risk-factor profile discrepancy between the presence and the severity of extracranial carotid stenosis has been observed previously in the literature.2-3 The inconsistent association of traditional risk factors to the severity of extracranial carotid stenosis may indicate the presence of additional factors (eg, hemodynamic factors) that could contribute to the severity of carotid stenosis. In the North American Symptomatic Carotid Endarterectomy Trial (NASCET), 387 of 1360 patients (28.5%) had severe (70% to 99%) angiographically defined extracranial carotid stenosis on one side with none-to-mild (<30%) on the contralateral side. If traditional risk factors are associated with atherosclerosis at the carotid bifurcation, how can one explain the asymmetric nature of carotid disease?

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References

Response
To find out the potential determinants of atherosclerosis in our study, extracranial parts of all 11 major cervical arteries were evaluated. Among those who had atherosclerosis (180 of 294 patients), the severity of the disease was assessed by using three indexes. These were computed separately for the total length, total thickness, and percent stenosis of the plaques, accounting for all visible atherosclerotic lesions in all 11 arteries, and we did not analyze the plaques at or near carotid bifurcations separately. However, five traditional risk factors showed a significant association with the total length of the plaques, percent stenosis and the thickness of the plaques were explained by four and two risk factors, respectively. According to this variability, traditional risk factors seemed to predict in particular the overall dissemination of atherosclerotic disease instead of being strong determinants of the grade of stenotic plaques. Among other factors, hemodynamic forces could have a role, and platelets may have an influence on the development of atherosclerotic lesions.1 In early atherosclerosis, the sites of predilection are vessel orifices and bifurcations; here the flow patterns may be complicated, possibly augmenting platelet adhesion to the vascular endothelium at these sites. Platelets, in turn, could contribute to the development of stenotic lesions in at least two ways: by stimulating the migration and proliferation of vascular smooth muscle cells and by formation of thrombi that become consolidated and incorporated into the vessel wall.3 In general, the presence of relatively few (and partly inconsistent) associations between traditional risk factors and the severity of atherosclerosis in our study suggests that other factors not included (and perhaps not identified at all) may also be involved.

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Reference

Denial of Illness and Depression in Stroke

The extensive review on denial of illness in stroke by Ellis and Small highlighted phenomenological and etiological aspects of denial of illness pertaining to physical disability in stroke patients. However, authors have failed to discuss the relationship between poststroke depression and denial of illness. This letter is intended to focus on the prevalence of denial of depression in poststroke depression and its correlation to lesion location; in addition, the relationship between denial of illness concerning physical disability and poststroke depression will be discussed.

Gainotti suggested that the depression in patients with right hemispheric lesion may be ameliorated due to their tendency to deny depression and also by their failure to express the affect. Fedoroff et al3 in their study of acute stroke patients, found that 5% of poststroke patients deny depressed mood although they fulfill the criteria for depression. Among patients with depression, approximately 10% presented with denial of depressed mood. Of patients with denial of depression, 60% had right hemispheric...
lesions and 10% had left hemispheric lesions. This finding confirms Gainsotti’s view that denial of depressed mood is common in right-sided lesions. On further analysis of the data, it was found that only 6 among 21 depressed patients with right hemispheric lesions present with denial of depressed mood. Although the reasons for rarity of this symptom remain largely undetermined, this could be explained in the context of intrahemispheric lesions and its correlations with depression and denial of illness in right-sided lesions. The literature suggests that right posterior lesions are correlated with depression, whereas right frontotemporal lesions are correlated with a reaction of indifference that has been associated with denial of illness.5 Based on this evidence, one may assume that denial of illness is less common among depressed patients with right hemispheric lesions, as the sites of lesions producing depression and those producing denial of illness vary significantly.

The second issue is related to anosognosia and its association with poststroke depression. Psychological theories indicate that anosognosia protects the organism from realizing the full extent of deficits and anosognosia may not coexist. However, evidence against the psychological hypothesis of anosognosia came from a study that demonstrated that depression was equally frequent among patients with and without anosognosia. Hence, studying the phenomenon of anosognosia in poststroke depression highlighted many facts that include anosognosia or denial of illness is not only concerned to physical disability but also to depression. Second, the reactive theory of poststroke depression has been challenged with the finding that anosognosia and depression coexist, which suggests that denial of physical disability does not protect patients from developing poststroke depression. Third, denial of depression may be associated with right anterior frontal lesions, whereas poststroke depression with depressed mood may be associated with right posterior lesions. Fourth, identification of depression in the absence of depressed mood may be a difficult task in clinical practice. However, the presence of cognitive and vegetative symptoms that fulfill the criteria for depression is sufficient to make the diagnosis of depression even in the absence of depressed mood.

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References

Response
We appreciate the chance to respond to the comment of Dr Ramasubbu that we did not discuss poststroke depression and denial of illness. As they rightly point out, our article focused on physical disability, with particular emphasis on denial of hemiplegia. The literature concerning the investigation of mood disorders after stroke is extensive and, indeed, in itself deserving of a comprehensive review. We therefore welcome a letter that highlights this aspect of denial.

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Leukocytes in Acute Stroke
Wang and coworkers have described a useful method for imaging leukocyte infiltration in acute ischemic stroke. In reviewing the literature concerning the role of leukocytes in acute stroke, they failed to mention studies linking leukocytosis with reduced survival after stroke. Among 283 subjects with acute stroke, elevated leukocyte count proved to be a significant univariate predictor of short- and long-term mortality.6 Among 492 elderly subjects with acute stroke followed up for a mean of 18 months, elevated leukocyte count was a strong univariate predictor of mortality; it remained a significant predictor of mortality in multivariate models containing initial level of consciousness, age, serum creatinine, and Mini-Mental State score.7 Further studies are required to determine the role of leukocytes in acute stroke.

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References

Response
Dr Friedman’s study showed that an elevated leukocyte count was the third-best univariate predictor of mortality and the fifth-best multivariate predictor of mortality in acute stroke patients. Our study focused on the dynamics of leukocyte infiltration in acute stroke. A previous study with the same methodology did show poor prognosis with high leukocyte infiltration in acute stroke patients. The postulated effects of leukocytes to the pathogenesis of cerebral stroke include (1) limitation of cerebral blood flow by vessel plugging or vasoconstrictive mediator release; (2) exacerbation of blood-brain barrier or parenchymal injury through hydrolytic enzyme release, lipid mediator production, or oxygen radical production; and (3) initiation of thrombois.

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Denial of illness and depression in stroke.
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