Letters to the Editor

Proposed Method for Analyzing Carotid Endarterectomy Results

To the Editor:

The recent series of letters to the Editor published in Stroke regarding the proposals by Jonas1-2 included a suggestion that caused Dr. Jonas to refer in his response3 to an article of mine.4 In reviewing this article, Dr. Jonas states that "The assumption least favorable for surgery would be that 10 operative strokes occurred in the 146 relevant carotid endarterectomy patients, a 6.8% operative stroke plus death (S+D) rate."

In reviewing our article, I do not know how Dr. Jonas could have obtained these figures since data for the mortality and morbidity in patients with transient ischemic attacks (TIAs) was not included. However, our data indicate that for the 152 patients operated on for anterior hemispheric TIAs (including amaurosis fugax), there were no deaths and there was a permanent neurologic deficit rate of 2.6%. Thus, the late stroke-free survival rate for this group of patients is significantly better than that calculated by Jonas using his method of intact months of patient survival (IMPS).

I believe that our results are typical of those obtained by many other surgeons in good centers. In addition, Dr. Jonas has not dealt with our data objectively but must have made some assumptions highly prejudicial against surgery in obtaining a presumed morbidity and mortality rate that was three times our actual experience. Thus, I would caution Dr. Jonas and your readers to use extreme care in evaluating the available data before coming to such important and damaging conclusions regarding the results of carotid surgery.

Rather than argue that the average results of carotid surgeons in this country do not demonstrate a significant advantage over the natural history of the disease for patients with TIA, it may well be that we should determine the boundary line of acceptable morbidity and mortality, below which surgery would be inappropriate. Statistics for both the surgeon and the patient survival (IMPS).

I do hope that the forthcoming randomized prospective studies will address this issue with sufficient power to permit arriving at such a conclusion.

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References


Significance of Age-Related White Matter Lesions

To the Editor:

Our studies of Alzheimer's disease have recently linked the presence of periventricular white matter lesions (PWMls) with both an increased prevalence of motor and gait deficits on neurologic examination1 and altered patterns of glucose utilization as measured by positron emission tomography.2 Several reports, including our own, have also confirmed the association of PWMls on computed tomograms (CTs) demonstrating small vessel disease in the brain.3-4 The hyalinosis and white matter rarefaction are consistent with hypertensive encephalopathy.1-3 In recent years, numerous CT and magnetic resonance imaging reports have indicated that PWMls affect as many as 10-30% of cognitively normal elderly study subjects. The prognostic and clinical significance of these lesions is largely unknown in otherwise normal individuals. There has been a failure to find differences in the prevalence of gait impairments in elderly normal subjects as assessed on comprehensive and standardized neurologic examination.1-4 We now report that PWMls in normal subjects are associated with subtle deficits in motor control.

We analyzed the performance of 17 cognitively normal elderly subjects (six with CT evidence for PWMls in their frontal lobes and 11 without such lesions) on cognitive and motor tests. All subjects were research controls falling within accepted ranges of normality based on extensive medical, neurologic, psychiatric, and cognitive examinations (e.g., Global Deterioration Scale1 scores of 1 or 2). Details of our clinical evaluations can be found elsewhere.5-6 The group with frontal PWMls was equivalent to the group without such lesions in age (range 52-80 years) and performance on nonmotor, cognitive evaluations of immediate memory (digit span), recent memory (paragraph recall), and language function (vocabulary).

Our results show that the group with frontal PWMls had significant (p<0.05) deficits on psychometric motor tests, including digit symbol substitution and choice reaction time, and on a computerized motor tracking task. The motor tracking task required the execution of a head-tilt maneuver to reach and fix onto a stationary visual target. The scores from the head-tracking test were the most accurate indexes of group member-
ship. The head-position error score, reflecting distance from the target over time, achieved an overall accuracy of group prediction of 94% ($\chi^2 = 13.2, p<0.001$) with a sensitivity of 100%.

Our results suggest that the presence of frontal PWMLs in otherwise cognitively normal elderly subjects is associated with subclinical motor dysfunction. Of course, performance on tests of psychomotor function (such as digit symbol substitution and motor tracking) can also be affected by deficits in other functions, especially attention. However, subjects with PWMLs performed as well as those without PWMLs on nonmotor evaluations (e.g., digit span backward and paragraph recall), which also require attention for their execution. Thus, we believe that these frontal PWMLs are primarily interfering with motor/psychomotor activity.

Our findings raise a number of questions. Is the presence of frontal PWMLs partly responsible for the often-reported observation of psychomotor slowing with advancing age? Are PWMLs related to "failing" in the elderly? Do frontal PWMLs herald the subsequent development of a more widespread cognitive decline? A recent report linking the presence of PWMLs with both mild cognitive impairment and motor dysfunction in elderly subjects implies that the answer to this last question may be affirmative. Our findings point to a relation between the presence of PWMLs and motor deficits and suggest that patients with PWMLs be followed longitudinally with more comprehensive neuropsychological and neuroimaging evaluations to determine if they are at increased risk for the development of clinically significant motor dysfunctions or other disorders.

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Letters to the Editor

Disability and Rehabilitation After Stroke

To the Editor:

We read with great interest the recent articles by Dombovy et al on disability and rehabilitation after stroke and by Bernspång et al on the impact of motor and perceptual impairments on self-care ability in stroke patients. The two articles are important contributions in predicting the pattern of recovery from stroke as well as in health care and rehabilitation planning. While the authors have identified factors such as age, presence of comorbidities,1 and motor and perceptual impairments2 as indicative of worsening disability in stroke victims, we wish to draw attention to the presence of poststroke depression as an additional factor determining rehabilitation outcome after stroke.

In a recent study, we found that in-hospital depression was an important prognostic factor for physical impairment 2 years after stroke.3 In another study, we found that there was a significant positive correlation between depression and physical impairment 3 and 6 months and 1 and 2 years after stroke.4 In a 5-year study, Berrios et al found that long-term recovery in general neurologic patients (including those with strokes) who were depressed was significantly worse than in similar patients who were not depressed. Sinyor et al also noted that nondepressed patients showed a slight increase or no change in physical impairment with time whereas depressed patients had more functional impairment during the first 6 months after stroke. It is interesting to note that Dombovy et al found a stabilization of functional status only 6 months after the stroke. Since these early months seem critical to physical recovery, early-onset poststroke depressions may, by bringing about a lack of motivation to participate actively in rehabilitation therapy, exert a negative influence on physical recovery that extends beyond the duration of the depression itself.

We have also documented the existence of a dementia of poststroke depression that is manifested by poorer scores on many perceptual tasks.7 These tasks are quite similar to those used by Bernspång et al. Patients who are affected by poststroke depression exhibit a greater degree of cognitive impairment than do nondepressed stroke patients with similar lesions. Thus, although dementia following stroke can result from multiple etiologies such as lesion location, number, and volume, the presence of depression further amplifies the degree of dementia in these patients. Since approximately 30–40% of patients suffer from poststroke depression immediately after stroke, the effect of depression may have significantly influenced the findings in both articles.

We recently compared the recovery in activities of daily living in patients with an in-hospital diagnosis of major depression, minor depression, and no depression using the Johns Hopkins Functioning Inventory (JHFI) as a measure of impairment. Patients were matched across the three diagnostic groups for degree of in-hospital impairment on the JHFI. At 2 years’ follow-up, nondepressed patients were significantly better than patients from either depressed group. These findings emphasize the need to evaluate stroke patients for depression as well as other variables before assessing the relative impact of these variables on long-term outcome and rehabilitation.

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