Dementia After Stroke

To the Editor:

Two recent stimulating and important studies published in this journal address the problem of dementia after stroke.1,2 We offer some comments on the concept of vascular dementia that we believe have general importance.

Both studies use poor diagnostic criteria for dementia. Tatemichi et al acknowledge this and leave the diagnosis to the discretion of each physician participating in the Data Bank project, without giving explicit criteria. They base their estimation of prevalence on a diagnosis made 7-10 days after the stroke, even though the acute phase of any brain disease, when arousal and attention may both be disturbed, may not be an appropriate time to formulate the diagnosis.4

In their estimate of incidence, the first diagnosis of dementia was made within 90 days after the stroke, a period still within the recovery phase.5 It seems unlikely that as many as 5-30% of patients (depending on age and amount of cerebral atrophy) in this 3-month time period after the stroke would further deteriorate from their level of function at 7-10 days unless they had had a new stroke. Perhaps many of these newly-demented patients were "untestable" at the first poststroke assessment and, if so, should be part of the prevalence estimation instead. Another possible explanation of this high incidence may be that because the physical disability is subsiding and no longer the principal focus of attention, the severity of the cognitive disability becomes more recognized and assumes a new and important feature.

Babikian et al use a Mini-Mental State Examination (MMSE) score of <24 as the criterion for dementia. Since focal lesions, especially in the left hemisphere, can lead to low MMSE scores in the absence of more global cognitive decline, the MMSE is inappropriate for a diagnosis of dementia.4 Furthermore, patients with a subcortical type of cognitive decline and dementia according to the DMS-III-R-criteria5 may score above the cutoff value of such simple bedside tests because no time limits are applied.9 Since demented patients with lacunar infarcts were found to have a subcortical type of cognitive disturbance,9 this may be the explanation for the authors' finding that patients with lacunar strokes were less often demented, according to the MMSE criterion, than were patients of the "mixed" group, even though cognitive disturbances were not significantly different between the groups.

A related problem is that different types of assessment of severity of dementia, such as measures of cognitive dysfunction, activities of daily living, and burden experienced by primary caregivers, are not necessarily strongly correlated.8 As a result, the diagnosis of dementia should be made independently of any measures of severity. These comments all lead to a forceful conclusion that for a diagnosis of dementia in patients with vascular disease, both specific criteria and the timing of assessment are critically important.

Definition of dementia in patients with vascular disease is complicated. Many of these patients do not present with the problem of cognitive decline but with a stroke syndrome. The sudden and severe physical disability becomes the most prominent feature. In these patients the presence of stable cognitive deficits can only be accurately judged after a time period of at least half a year, and even then it may be difficult to justify the diagnostic criteria for dementia. A single lesion may lead to more than one type of cognitive deficit, and even when a single cognitive deficit seems to be present, its presence may seriously affect the assessment of other cognitive functions. With single lesions, personality changes and depression are both likely to occur. Furthermore, it cannot always be accurately estimated whether the handicap experienced by patients and caregivers is determined mainly by the cognitive changes or by the physical disability.

When such patients are eventually judged to be demented, we would be inclined to label the problem as "stroke-with-dementia." These patients are more easily differentiated from patients with dementia caused by degenerative disease. Nonetheless, in patients with both cerebrovascular disease and a progressive cognitive decline and personality change without significant physical disability, the clinical presentation may be appropriate for other diseases leading to dementia, of which Alzheimer's disease is the prototype. In this category of patients, the problem is that of a "vascular dementia" caused by many infarcts (multi-infarct dementia) or a dementia with no identifiable focal cerebrovascular lesions at all (nonstroke vascular dementia).

Lesion type and location of lesions, as well as the presence of noninfarct vascular brain damage,9 may account for the difference in clinical presentation between these groups of patients. Epidemiological differences may also be of importance. Patients with "stroke-with-dementia" and those with "vascular dementia" will probably be seen by different kinds of doctors and be diagnosed and treated in different sectors of health care systems, which may also contribute to conceptual confusion.

Both types of dementia that we identify for patients with vascular disease need to be studied more intensively, as Tatemichi et al and Babikian et al have done for "stroke-with-dementia." At the same time, we need continued investigation and elucidation of the conceptual differences between this clinical problem and that of the other vascular dementias more like Alzheimer's disease.

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