Definition of Dementia

To the Editor:

In a recent editorial, Wells has rightfully questioned the role of stroke in dementia. He concludes that “cerebrovascular disease is not the major cause of the commonly encountered dementias of middle and late life.”

Classically, neurologists tend to define dementia as an impairment in higher intellectual function usually manifested by:

- difficulty with orientation to time, place, or person,
- diminished recall or memory (immediate more than remote),
- a decreased fund of general information,
- difficulty in performing such tasks of abstract reasoning as calculations, similarities and differences, and proverbs, and,
- grossly impaired judgment.

This definition of dementia, however, does not fully describe the spectrum of mental states resulting from focal disease of man’s cerebral hemispheres in adult life, particularly in stroke.

In reviewing our experience with over 1000 patients referred to a regional stroke rehabilitation center, we observed many patients “acting peculiarly” or doing “peculiar things” even though, in a classical sense, they were not demented and were often able to perform well on cognitive testing. These patients often suffered from:

- denial/neglect (hemi-inattention)
- apraxia (of many types)
- visual-spatial distortion
- right-left disorientation
- difficulty with sequencing activities
- difficulty in organizing concepts of body image and scheme
- difficulty in abstracting parts from a complex background (figure-ground deficits)
- difficulty with concepts of virtuality
- an inability to stop once started (perseveration)
- an inability to cope with simultaneously occurring events (cross-modal extinction)
- dramatic changes in concepts of time, and
- severe distortion of sleep-wake cycles.

Because these perceptually impaired patients function in a world within our world, their attempts to perform often seem bizarre to observers and often contribute to the patient’s frustration and anger. When the behavioral abnormalities appear in relatively quiet patients, they are often labelled as demented. When the behavioral abnormalities interfere with routines or bother others, the patient is often “treated” with psychoactive drugs which usually act to further distort an already distorted universe thus producing behavior which is even more bizarre and inappropriate.

There is some evidence that these perceptual problems can be improved with training. Since there are different approaches to treating cognitive and perceptual deficits it seems important to emphasize the need for an acceptable definition and classification of the spectrum of mental states resulting from focal disease of man’s cerebral hemispheres in adult life.

Finally, the statistics used to demonstrate a minor role for stroke in causing dementia may be spurious since they are based on populations presenting primarily with dementia, not stroke. Of 318 patients admitted to a regional stroke rehabilitation unit 27% had significant cognitive dysfunction or “dementia.” These statistics (also biased) show a more significant cause-effect relationship between stroke and dementia. Because 39% of this same population had significant perceptual deficits, one must assume that stroke is also a major cause of this type of “mental impairment.”

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References


Questions Arising in Carotid Bruit

To the Editor:

Dr. Fields’ article on the Asymptomatic Carotid Bruit — Operate or Not? (Current Concepts of Cerebrovascular Disease, Stroke 9: 195-293, 1978) is certain to generate varied responses. Certain basic questions must be answered before one can arrive at the decision regarding surgery. The asymptomatic carotid bruit generally indicates a stenotic lesion constricting the lumen more than 50%. The ultimate fate of this lesion often is occlusion. What may happen when this occurs is variable; there may be no symptoms, or the patient may have a stroke, (depending on the adequacy of the collateral flow). This indicates a need to know the status of the contralateral carotid in the person with carotid bruit. Cerebral angiography is needed for this evaluation before making a decision regarding the bruit and its underlying stenosis.

Fields states, “Unfortunately we have no evidence which enable us to state unequivocally which lesion is hazardous.” To adopt a wait-and-see attitude may be a game of Russian roulette. Aspirin is recommended for the asymptomatic bruit. In the absence of TIA’s, the value of aspirin seems doubtful since it has not been shown to retard the progress of the atheroma.

A prospective randomized study would appear to be the best means of resolving this continuing dilemma.

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The author replies:

I agree with Dr. Galbraith that certain basic questions must be answered before one can arrive at a decision regarding surgery. The presence of a carotid bruit suggests, but does not necessarily indicate, the constriction of the lumen of the internal carotid artery by a stenotic lesion of more than 50%. It is well known that bruits are seldom produced by stenosis of less than 50% and may be absent when stenosis is greater than 80%. Stenosis per se is probably important as an indicator that a lesion is present, but I still feel that the degeneration of an atheroma with subsequent embolization of atheromatous debris downstream is more critical.

Dr. Galbraith apparently is not aware of recent studies reported from the University of Washington and the University of Chicago which suggest that aspirin may have an important role in the prevention of atheroma formation on the one hand and in the
Questions arising in carotid bruit.
J G Galbraith

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