THE NOTION THAT SLOWLY progressive demen-
tia in the elderly is the result of chronic diffuse brain
hypoxia is largely discredited — Alzheimer disease
has nothing to do with cerebral arteriosclerosis. Nonetheless, it is still widely believed that many elderly
people are demented on a cerebrovascular basis, most
often in the form of "multi-infarct dementia." Such a
term does not refer to the circumscribed intellectual
turbances which follow single large infarcts in the
territories of major cerebral vessels. Rather it de-
notes more global cognitive loss in the presumed pres-
ence of several lesions, often deep lacunes, none of
which would alone be expected to cause intellectual
impairment. In fact, many clinicians believe that when
a demented patient has symptoms or signs of stroke (as
reflected, for example, in the Hachinski Ischemic
Score), cerebrovascular disease can be presumed to be
either causing or contributing to the dementia, and on
such an assumption rest a number of well-known clin-
cal studies. Perhaps, therefore, one should ask upon
what basis such an assumption is made.

Tomlinson, Blessed, and Roth have provided the
most persuasive pathological evidence to date that
stroke does cause dementia in the elderly. Comparing
50 brains of demented old people to brains of unde-
mented controls, they concluded that half had what
today is called senile dementia of Alzheimer type
(SDAT), 17% had either definite or probable arterio-
sclerotic dementia, and 18% had a combination of the
two diseases. The authors stressed a number of im-
portant details, however. First, of the 9 patients consid-
ered to have arteriosclerotic dementia, three had be-
tween 60 and 82 ml volume of infarction, and the other
six had volumes of 101 to 412 ml; only one of the
controls had more than 60 ml of infarction, and none
had more than 100 ml. Second, the authors did not deal
with "the possibility of ischaemic lesions affecting
areas of brain likely to be particularly important in
relation to producing features of dementia" — possi-
ble relevant to the fact that three patients with lesions
of 60 to 82 ml had dementia, whereas the single control
with such a degree of infarction did not. Third, four of
the nine cases of mixed arteriosclerotic dementia and
SDAT also had more than 100 ml of softening; the
other five did have volumes of infarction (mean 38.8
ml) encountered in several controls, suggesting to the
authors that the presence of either disorder might lower
the threshold for clinical expression of the other.
Moreover, softening of mild to moderate degree was
present in 19 of the 25 patients considered to have
SDAT of sufficient severity to explain the dementia
alone. (How many of these patients, or those whose
dementia was of "mixed" type, had had symptoms or
signs of stroke — i.e., a high "Ischemic Score" —
was not determined.) Fourth, multiple small dience-
phalic infarcts were seen in both demented and non-
demented subjects, and among those with arterioscle-
rotic dementia were never the only or even the major
lesions. Fifth, "The sample was not a truly random
one." Thirty-four of the 50 demented patients, includ-
ing eight of the nine with arteriosclerotic dementia and
all 9 with mixed arteriosclerotic dementia and SDAT,
were male, yet 15 of the 25 with SDAT were female.
Since, in another survey, the authors had found de-
mentia to be twice as frequent among women as among
men, arteriosclerotic dementia was probably overrep-
resented in their series. As they noted, "... conclu-
sions could not be drawn from this sample about the
prevalence of the various types of dementing process
in old age."

Thus, the frequency of arteriosclerotic dementia
among old people cannot be determined from the data
of Tomlinson, Blessed, and Roth. Their major finding,
however, is one that seems to have been ignored by
other investigators, namely that arteriosclerosis causes
dementia only when there are large total volumes of
brain infarction, usually of more than 100 ml. Smaller
lesions, including multiple small subcortical infarcts,
are unlikely to cause dementia. Such a conclusion
would have come as no surprise to C. Miller Fisher,
who two years earlier had expressed his view that
lacunes were overrated as a cause of progressive de-
mentia, and that when they occurred in large enough
numbers (Marie's "l'état lacunaire") to cause cogni-
tive loss, there was usually pseudobulbar palsy, with
dysarthria, dysphagia, bradykinesis, and gait distur-
bance. Paraphrasing Boyd, Fisher commented that
"... lacunes lick the psyche and bite the soma, just
opposite to senile dementia." Moreover, for them to
appear in large numbers is unusual; of Fisher's 114
consecutive patients with lacunes at autopsy, only nine
had more than 10, and dementia, with or without pseud-
obulbar palsy, was rare.19 Fisher recently has reiter-
ated this point: "... one rarely (if ever) encounters the
lacunar clinical state as described [by Marie]."

Clearly, then, pathological evidence of stroke
doesn't necessarily mean that cerebrovascular disease
has anything to do with a patient's dementia, and in
most instances it probably doesn't. Yet many cli-
icians and pathologists have ignored the above data and
assumed that if a demented patient has symptoms or
signs of stroke, or autopsy evidence of cerebral infar-
cion, then the dementia is at least partly vascular in
origin. Such an inference is frequently made from patho-
logical reports even when the size or location of the
lesion(s) is not described. Often-cited studies of this
nature include those of Corsellis,20 who found "organ-
ic psychosis" among 167 elderly patients to be "asso-

The author is Director of Neurology, Harlem Hospital Center, and
Professor of Clinical Neurology, Columbia University College of Phy-
sicians and Surgeons.

Address correspondence to: John C. M. Brust, M.D., Department of
Neurology, Harlem Hospital Center, 506 Lenox Avenue, New York,
New York 10037.

Received June 21, 1982; revision accepted September 28, 1982.
associated with cerebral vascular changes" in 46 (28%) (of whom 20% had had focal neurological signs) and "associated with mixed vascular and senile changes" in 20 (12%) (of whom 25% had had focal neurological signs); Delay et al., who attributed dementia in old age to cerebrovascular disease in a quarter of cases and to mixed senile and arteriosclerotic disease in half; Sourander and Sjogren, who classified as "cerebrovascular dementia" 72 (23%) of their 318 cases (none of whom, interestingly, was considered to have a mixture of SDAT and cerebrovascular dementia); Malamud, who found "arteriosclerotic brain disease" in 356 (29%) of 1225 cases and "mixed senile-arteriosclerotic disease" in 283 (23%); Todorov et al., who diagnosed "vascular dementia" in 132 (17%) of 776 cases and "combined senile-vascular dementia" in 250 (32%) (criteria for "vascular" in this study were simply "parenchymatous lesions of vascular origin"); and Jellinger, who attributed dementia in the elderly to cerebrovascular disease in 225 (22%) of 1009 cases and to "mixed senile and vascular origin" in 136 (13%). (In this last study, "lacunar state" was the most frequently encountered change in patients with vascular dementia. There was also a high frequency of cerebrovascular lesions in patients classified as having "senile dementia" (i.e., SDAT) and of "senile parenchymal changes" in patients considered to have vascular dementia.)

Even the absence of pathologic Alzheimer disease or other identifiable dementing illness may not warrant blaming dementia on vascular disease when only small infarcts are found at autopsy, for the cause of dementia is not always clear pathologically. Some of Marie’s patients considered to have "l’etat lacunaire" may well have had normal pressure hydrocephalus; alcoholic cerebral atrophy was not recognized until the past decade; and demented patients have been found at autopsy to have lesions which defy classification. However, most patients considered demented on a vascular basis probably aren’t. The conclusion of Tomlinson, Blessed, and Roth is as valid today as it was a dozen years ago: "Arteriosclerotic dementia is almost certainly overdiagnosed clinically."

References


Vascular dementia--still overdiagnosed.

J C Brust

Stroke. 1983;14:298-300
doi: 10.1161/01.STR.14.2.298

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/14/2/298.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/