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Dementia in Stroke Survivors in the Stroke Data Bank Cohort

Prevalence, Incidence, Risk Factors, and Computed Tomographic Findings

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We determined the prevalence of dementia in 927 patients with acute ischemic stroke aged ≥ 60 years in the Stroke Data Bank cohort based on the examining neurologist's best judgment. Diagnostic agreement among examiners was 68% ($\kappa=0.34$). Of 726 testable patients, 116 (16%) were demented. Prevalence of dementia was related to age but not to sex, race, handedness, educational level, or employment status before the stroke. Previous stroke and previous myocardial infarction were related to prevalence of dementia although hypertension, diabetes mellitus, atrial fibrillation, and previous use of antithrombotic drugs were not. Prevalence of dementia was most frequent in patients with infarcts due to large-artery atherosclerosis and in those with infarcts of unknown cause. Computed tomographic findings related to prevalence of dementia included infarct number, infarct site, and cortical atrophy. Among 610 patients who were not demented at stroke onset, we used methods of survival analysis to determine the incidence of dementia occurring during the 2-year follow-up. Incidence of dementia was related to age but not sex. Based on logistic regression analysis, the probability of new-onset dementia at 1 year was 5.4% for a patient aged 60 years and 10.4% for a patient aged 90 years. With a multivariate proportional hazards model, the most important predictors of incidence of dementia were a previous stroke and the presence of cortical atrophy at stroke onset. (*Stroke* 1990;21:858-866)

Cerebrovascular disease is considered to be the second most common cause of dementia; 20%–25% of cases of dementia are due to stroke, and another 10%–15% are attributed to a combination of vascular and Alzheimer's disease.¹

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Although cerebral arteriosclerosis does not play a causal role in Alzheimer's disease,² there is still controversy about the role of ischemic stroke in producing global cognitive disorders classifiable as dementia.^{3,4} The true risk of dementia caused by ischemic stroke is incompletely understood because most previous studies have focused on series of selected or consecutively encountered patients with dementia rather than assessing dementia in stroke cohorts.

To address this question properly, patients with stroke should be studied prospectively using neuropsychological and functional examinations combined with well-defined diagnostic criteria. In preparation for such a study, we turned to the Stroke Data Bank⁵ as a source of preliminary data. Because the study of dementia was not a major focus of the Stroke Data Bank, the diagnosis of dementia was based only on the best judgment of the examining neurologist. As a result, the validity and reliability of this source of information may be limited. Nonetheless, we believe that an exploratory effort using this data source could serve as a useful guide to future studies on this topic.

Our aims were to determine the prevalence of dementia after ischemic stroke in this cohort, to identify potential risk factors (including stroke mechanism and computed tomographic [CT] findings) related to prevalence of dementia at stroke onset, to determine the incidence of dementia in the remaining dementia-free cohort followed longitudinally, and to develop a predictor model for incidence of dementia using information about potential risk factors.

Subjects and Methods

The Stroke Data Bank procedures have been described.⁵ Neuropsychological tests were not given, and the diagnosis of dementia was based on the clinical judgment of the examining neurologist. The question was formulated as "Examiner believes patient is demented," with the possible responses being "yes," "no," and "untestable." Using this approach, we probably identified only patients with severe dementia. Patients with mild dementia require more detailed testing for proper diagnosis. The most likely cause of dementia, again based on the examiner's judgment, was designated as Alzheimer's disease, stroke, or other (including combined Alzheimer's disease and stroke). The cause of dementia was an opinion not subjected to further verification. Examinations were planned at 0–7 days, 7–10 days, and 3, 6, 12, and 24 months after stroke onset.

The design of the Stroke Data Bank included studies of interexaminer reliability for elements of the neurologic examination. Agreement on the dementia question was 68% with a significant κ of 0.34,⁶ reflecting fair to good agreement.⁷ This level of interexaminer variability was similar to that found for visual fields, level of alertness, and degree of hemiparesis.

The study population consisted of 927 patients ≥ 60 years of age with ischemic stroke. An elderly population was selected to focus on patients at the highest risk for dementia. Patients with parenchymatous or subarachnoid hemorrhage were excluded since they represent different pathophysiological groups. The diagnosis of dementia was based on findings from the 7–10 day examination where possible to permit the hyperacute effects of stroke to subside, thus maximizing the number of patients considered testable; when patients were discharged before day 7, the 0–7 day examination was used. For the incidence studies, we used the examinations at each planned follow-up whenever available up to 2 years.

Although the terms "prevalence" and "incidence" properly refer to unselected populations, we defined two cohorts using these terms to distinguish between findings at stroke onset and those at follow-up. The prevalence cohort was defined as patients whose dementia status was testable during the first 10 days after stroke onset. The incidence cohort was defined as testable patients who were free of dementia at admission and who were assessed neurologically during follow-up.

We determined prevalence rates (as a percentage) for each 5-year age group for the prevalence cohort.

Since many patients (21.7%) were untestable, we examined the features that predicted testability using a multiple logistic model⁸ to assess selection biases. We next examined the relation between prevalence of dementia and sociodemographic variables, vascular risk factors, functional history, ischemic stroke subtype, and CT findings. For functional history, we assessed the patient's activities during the month before the stroke including five domains from the Barthel Index (eating, dressing, transferring, walking, and continence). In this analysis we controlled for previous stroke since this factor alone might account for functional impairments. For ischemic stroke subtype, we used a diagnostic algorithm⁹ that classified patients as having infarction of unknown cause, infarction with normal angiogram, infarction with tandem arterial pathology, infarction due to cardiogenic embolism, infarction from thrombosis due to large-vessel atherosclerosis (atherothrombotic stroke), or infarction due to lacunes. For CT findings, we examined the first scan obtained in the first 10 days after stroke onset showing a new, related lesion; if only one scan was obtained, we used it for analysis whether or not a lesion was present. CT was examined in 426 of the 726 patients in the prevalence cohort. The methods for evaluating and recording CT findings⁵ and their interobserver reliabilities have been described.¹⁰ Although not a specified procedure in the Stroke Data Bank, we believe that the CT findings were recorded independently of the neurologic examination. Specific CT findings of interest were the number of new lesions related to the stroke, the number of old unrelated lesions, the presence of cortical atrophy or hydrocephalus, the volume of the infarct, and the presence of periventricular lucencies. The primary cerebral site of the infarction was defined by a combination of CT findings and the clinical syndrome. For these cross-sectional comparisons (except site), we used univariate tests of association, tests for trend with ordinal data,¹¹ and nonparametric tests as appropriate. Site was not subjected to statistical analysis since the cerebral sites examined represent a combination of multiple sites.

We determined incidence rates using Kaplan-Meier¹² product-limit estimates of the time to first evidence of dementia. We also used a logistic regression model⁸ to estimate the probability of incidence of dementia, using age as a prognostic indicator. In this model we controlled for the effects of previous stroke, cortical atrophy, and atherothrombotic stroke, which were the variables significantly related to prevalence of dementia. Cox's semiparametric proportional hazards model¹³ provided an estimate of the differences in time to incidence of dementia, and a stepwise elimination procedure identified potential predictors. Based on univariate analyses in the prevalence cohort, we considered the factors age, hypertension, previous myocardial infarction, atrial fibrillation, diabetes mellitus, previous stroke, previous use of antiplatelet or anticoagulant drugs, atherothrombotic and cardioembolic stroke subtypes,

TABLE 1. Testability and Prevalence of Dementia by Age at Stroke Onset in Stroke Data Bank Cohort ≥ 60 Years Old With Ischemic Stroke

Age (yr)	N	Testable		Demented		
		No.	%	No.	Yes	
					No.	%
60-64	158	130	82.3	117	13	10.0
65-69	199	163	81.9	145	18	11.0
70-74	191	163	85.3	139	24	14.7
75-79	155	118	76.1	93	25	21.2
80-84	132	89	67.4	67	22	24.7
>85	92	63	68.5	49	14	22.2
Total	927	726	78.3	610	116	16

$p < 0.001$ testability related to age ($\chi^2 = 23.43$, $df = 5$).

$p < 0.001$ demented related to age ($\chi^2 = 15.89$, $df = 5$).

and the CT findings of number of new related lesions, number of old unrelated lesions, cortical atrophy, hydrocephalus, and infarct volume.

Results

Among the study population of 927 patients with ischemic stroke aged ≥ 60 years, 726 (78.3%) were testable and comprised the prevalence cohort. Testability was related to age (Table 1) and to several clinical features that impair communication, including reduced alertness (occurring in 159 of 925 patients, 17.2%), aphasia (192 of 927 patients, 20.7%), and hemineglect (81 of 927 patients, 8.7%). Among these variables, logistic regression analysis showed that clinical features but not age were the main determinants of testability. Although older patients were underrepresented in the prevalence cohort, patients were selected out by the presence of these clinical features, which appeared to be more common in the elderly. This selection bias is unavoidable in any clinical study of dementia in stroke patients.

able in any clinical study of dementia in stroke patients.

Among the prevalence cohort of 726 testable patients, 116 (16%) were demented. Prevalence of dementia was significantly ($p < 0.001$) related to age (Figure 1). The largest increase in frequency occurred between the age groups 70-74 and 75-79 years. No difference in prevalence of dementia occurred between men and women. For 95 of the 116 demented patients, the examining neurologist estimated the probable cause of dementia to be stroke in 37 (38.9%), Alzheimer's disease in 34 (35.8%), and other (including mixed dementia) in 24 (25.3%).

The sociodemographic variables sex, race, handedness (in 685 patients), educational level (in 665), and employment status prior to the stroke (in 713) were not significantly associated with prevalence of dementia (Table 2). However, a trend was apparent between educational level and dementia; patients with < 8 years of education were more frequently demented than those with some college education. Among the vascular risk factors, both a history of stroke ($p < 0.001$) and a history of myocardial infarction ($p = 0.021$) were significantly related to prevalence of dementia (Table 3); hypertension, diabetes mellitus, atrial fibrillation, and previous antiplatelet or anticoagulant therapy showed no significant association with prevalence of dementia.

Functional history was related to prevalence of dementia, even after controlling for the effects of previous stroke (Table 4). In each domain of function, impairment was more frequent among demented patients than among nondemented ones; moreover, demented patients with a history of stroke showed the most impairment. Previous stroke alone was not significantly related to functional history.

Prevalence of dementia varied by ischemic stroke subtype (Table 5). In the entire prevalence cohort dementia was most frequent among patients with atherothrombotic infarction and those with infarction of unknown cause and was least frequent in those with strokes due to tandem arterial disease or lacunes. Since 18 of the 51 patients (35%) with atherothrombotic infarction had suffered a previous

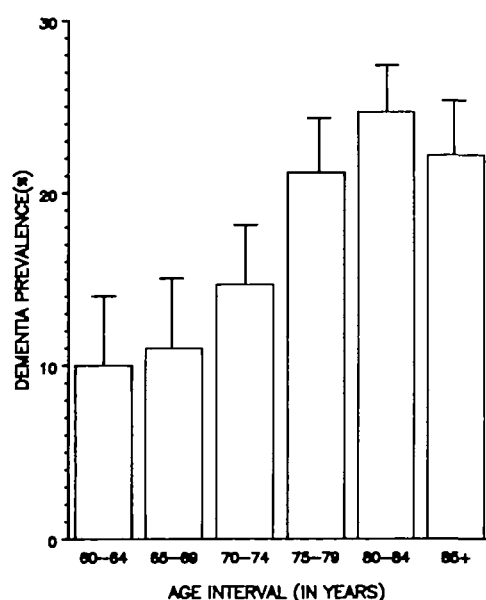


FIGURE 1. Bar graph. Mean \pm SEM prevalence of dementia by age at stroke onset among 726 testable patients ≥ 60 years old with ischemic stroke in Stroke Data Bank.

TABLE 2. Sociodemographic Variables and Prevalence of Dementia in 726 Testable Patients ≥ 60 Years Old With Ischemic Stroke

Variable	Total		Demented (%)	<i>p</i>
	No.	%		
Sex				
Female	392	54	17.3	0.274
Male	334	46	14.4	
Race				
White	244	33.6	16.8	0.066
Black	430	59.2	16.7	
Other (Hispanic)	52	7.2	5.8	
Handedness (<i>n</i> =685)				
Left	27	3.9	14.8	0.980
Right	651	95.1	16.0	
Ambidextrous	7	1.0	14.3	
Educational level (<i>n</i> =665)				
Grade 8 or less	284	42.7	20.4	0.090
Grade 9–11	133	20.0	15.0	
High school	159	23.9	13.2	
College or more	89	13.4	11.2	
Employment status (<i>n</i> =713)				
Retired	466	65.5	17.6	0.182
Not retired	247	34.5	13.8	

p determined using χ^2 test of association. Data not available on all variables for all patients.

stroke (Table 5, greatest frequency), we examined the possible interaction between stroke subtype and a history of stroke. Among the 558 patients without a known previous stroke, those with atherothrombotic infarction showed the highest frequency of prevalence of dementia ($p=0.046$). Among those with a history of stroke, the frequency of prevalence of

dementia varied nonsignificantly by subtype, with the highest frequency occurring among those with cardioembolic infarction.

CT findings in 426 patients were strongly associated with prevalence of dementia (Table 6). The number of new related lesions, the number of old unrelated lesions, and the presence of cortical atrophy (assessed

TABLE 3. Vascular Risk Factors and Prevalence of Dementia in Testable Patients ≥ 60 Years Old With Ischemic Stroke

Risk factor	Total		Demented (%)	<i>p</i>
	No.	%		
Hypertension (<i>n</i> =720)				
No	218	30.3	15.6	0.908
Yes	502	69.7	15.9	
Diabetes mellitus (<i>n</i> =719)				
No	530	73.7	17.0	0.111
Yes	189	26.3	12.2	
Atrial fibrillation (<i>n</i> =708)				
No	638	90.1	15.0	0.449
Yes	70	9.9	18.6	
Previous myocardial infarction (<i>n</i> =709)				
No	581	81.9	14.1	0.021
Yes	128	18.1	22.7	
Previous antiplatelet/anticoagulant drug use (<i>n</i> =718)				
No	625	87.0	16.0	0.815
Yes	93	13.0	15.1	
Previous stroke (<i>n</i> =702)				
No	534	76.1	9.4	<0.001
Yes	168	23.9	30.4	

p determined using χ^2 test of association. Data not available on all risk factors for all patients.

TABLE 4. Frequency of Functional Impairments in the Month Prior to Stroke in Relation to Prevalence of Dementia and a History of Previous Stroke in Testable Patients ≥ 60 Years Old

Demented	Previous stroke	n	Impairment (%)				
			Eating	Dressing	Transferring	Walking	Continence
Yes	Yes	48	18.8	29.2	25.0	22.4*	20.8
Yes	No	47	8.5	1.2	13.0†	13.0†	12.8
No	Yes	114	1.8	6.1	7.0	7.9	4.4
No	No	469	0.6	1.1	1.9	1.9	1.5‡

*% of 49.

†% of 46.

‡% of 470.

in 420 patients) or hydrocephalus (in 417) were significantly associated with prevalence of dementia; infarct volume (assessed in only 348 patients) showed no significant relation. When the lesions were classified by site, dementia was more frequent among patients with occipital, temporo-occipital, and temporoparietal lobe infarctions than among those with infarcts confined to the basal ganglia and capsule or brainstem and cerebellum (Table 7). Periventricular lucencies were rated in only 77 patients, limiting the usefulness of this information. However, the findings are worth noting. Thirty-two patients (41.6%) showed periventricular lucencies, and of these 32, 12 (37.5%) were demented. Among the 45 patients who had no periventricular lucencies, only two (4.4%) had dementia.

In the incidence cohort, Kaplan-Meier analysis revealed that the likelihood of survival free of dementia for up to 1 year was inversely related to age (Figure 2). The youngest four age groups clustered while the oldest two age groups diverged over time and showed the highest incidence rates. Mean \pm SEM 1-year point estimates for survival free of dementia for the six age groups were $93.3 \pm 2.7\%$, $95.8 \pm 1.9\%$, $91.2 \pm 2.9\%$, $91.9 \pm 3.6\%$, $81.6 \pm 6.9\%$, and $73.5 \pm 9.5\%$, respectively. Although the overall probability of incidence of dementia declined over time, the curves showed the sharpest decline early after stroke; thus, most examples of new-onset dementia in the incident cohort become apparent during the first 90 days following stroke onset. Using a similar Kaplan-Meier analysis, the probability of survival free of dementia

was also strongly related to the extent of cortical atrophy by CT at stroke onset (Figure 3). Incidence of dementia exceeded 30% at 3 months among patients with severe atrophy. Since none of these patients were followed for >3 months, their curve ends at that point. For atrophy rated as none, slight, moderate, and severe, mean \pm SEM 1-month point estimates of survival free of dementia were $98.7 \pm 1.2\%$, $94.4 \pm 2.5\%$, $91.9 \pm 3.1\%$, and $88.0 \pm 3.8\%$, respectively.

We estimated the independent effect of age on incidence of dementia from a logistic regression model, controlling for previous stroke, cortical atrophy, and atherothrombotic stroke subtype (Figure 4). Among patients with no previous stroke, no cortical atrophy, and a nonatherothrombotic stroke subtype, the predicted 1-year dementia rate was 5.4% for a person aged 60 years at stroke onset and 10.4% for one aged 90 years. These estimates can be compared with the Kaplan-Meier curves in Figure 2, which show that the probability of dementia-free survival at 1 year for those aged 60–64 years was approximately 93% (or 7% incidence); for those aged >85 years, this probability was 73% (or 27% incidence). The difference between the two models results from controlling for previous stroke, cortical atrophy, and stroke subtype in the logistic model but not in the Kaplan-Meier analysis and from using age groups in the Kaplan-Meier analysis but not in the logistic model.

Using Cox's proportional hazards regression procedures, we first developed a model that included age, previous stroke, atherothrombotic stroke subtype, and

TABLE 5. Ischemic Stroke Subtype and Prevalence of Dementia in 726 Testable Patients ≥ 60 Years Old

Subtype	Previous stroke				Total‡	
	Yes*		No†			
	No.	% demented	No.	% demented	No.	% demented
Unknown cause	61	36.1	190	16.3	251	21.1
With normal angiogram	8	12.5	19	10.5	27	11.1
With tandem arterial pathology	8	12.5	32	6.3	40	7.5
Cardiogenic embolism	33	39.4	97	9.3	130	16.9
Atherothrombotic	18	16.7	33	21.2	51	19.6
Lacunae	40	27.5	187	7.5	227	11.0
Total	168	30.4	558	11.6	726	16.0

* $p=0.227$ by χ^2 .† $p=0.046$ by χ^2 .‡ $p=0.064$ by Mantel-Haenszel χ^2 .

TABLE 6. Computed Tomographic Findings and Prevalence of Dementia in 426 Testable Patients ≥ 60 Years Old With Ischemic Stroke

Finding	Total		Demented (%)	<i>p</i>
	No.	%		
New related lesions				
None	163	38.3	28.2	0.037
1	249	58.4	14.9	
>1	14	3.3	35.7	
Old unrelated lesions				
None	262	61.5	15.3	<0.001
1	113	26.5	23.9	
>1	51	12.0	41.2	
Hydrocephalus (<i>n</i> =417)				
None	228	54.7	11.4	<0.001
Minimal	116	27.8	26.7	
Moderate	67	16.1	37.3	
Marked	6	1.4	66.7	
Cortical atrophy (<i>n</i> =420)				
None	106	25.2	4.7	<0.001
Slight	153	36.4	17.6	
Moderate	138	32.9	30.4	
Severe	23	5.5	52.2	
Volume (<i>n</i> =348)				
<15 ml	215	61.8	15.8	0.149
<50 ml	87	25.0	27.6	
>50 ml	46	13.2	19.6	

p determined using χ^2 test for trend.

cortical atrophy as the most significant predictors of incidence of dementia (Table 8, Model A). Further stepwise backward-elimination procedures led to a more parsimonious model that included only previous stroke and cortical atrophy (Model B).

Discussion

Our estimates of prevalence and incidence of dementia should be interpreted with caution in view of the limitations of the design of the Stroke Data Bank for this question. As a result, it is highly

TABLE 7. Cerebral Site of Lesion and Prevalence of Dementia in Testable Patients ≥ 60 Years Old With Ischemic Stroke

Site	Total		Demented (%)
	No.	%	
Anterior superficial (including perisylvian)			
Frontal	72	10.9	19.4
Frontoparietal	75	11.4	13.3
Insula-operculum	12	1.8	8.3
Frontotemporal	46	7.0	10.9
Posterior superficial			
Parietal	37	5.6	18.9
Occipital	24	3.6	25.0
Temporal	9	1.4	0
Parieto-occipital	6	0.9	16.7
Temporoparietal	33	5.0	24.2
Temporo-occipital	5	0.8	20.0
Deep			
Basal ganglia-capsule	196	29.7	10.7
Thalamus	21	3.2	14.3
Cerebellum—brain stem	124	18.8	9.7
Total	660	100.0	14.3

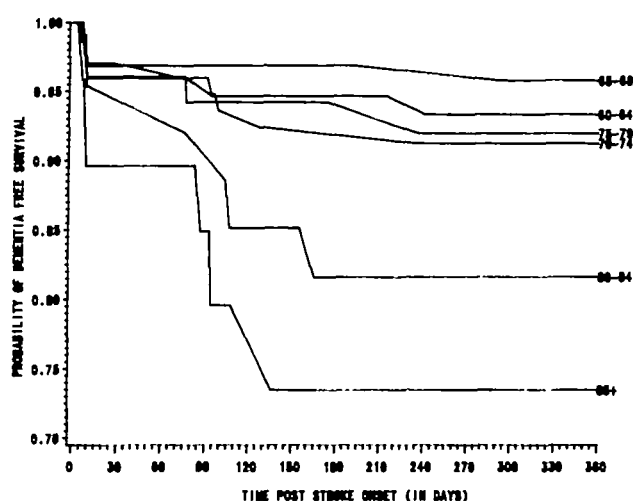


FIGURE 2. Kaplan-Meier estimate of survival free of dementia over 1 year by age at stroke onset.

probable that misclassification errors of undefinable magnitude occurred in both directions. Conceivably, dementia was underdiagnosed; the use of neuropsychological testing might uncover a larger number of clinically inobvious cases. If only the most overt cases were labeled demented, one interpretation is that our estimates describe a minimum frequency. In support of the validity of our findings, other studies have documented acceptable agreement between a clinician's impressionistic diagnosis of dementia and findings from formal mental function tests.¹⁴

Diagnostic misclassification in the opposite direction is equally conceivable. The possibility of overdiagnosis arises not only from the lack of specific operational definitions in the Stroke Data Bank but also because well-developed and validated criteria

for the diagnosis of vascular dementia do not currently exist for either clinical or research purposes.¹⁵ The disturbances in cognitive function that commonly occur from focal brain injury due to stroke may preclude dementia assessment in some patients (as in our study population) or could lead to overinterpretation of the clinical findings. The menu-driven approach used in the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III)¹⁶ is readily applicable to dementia due to diffuse brain disorders, but it may not adequately account for the focal effects of stroke.³ Finally, assuming that a syndrome classifiable as dementia can be operationally defined in stroke patients, the mere co-occurrence of dementia and stroke does not establish a causal relation. We can only argue that the internal consistency of our data, their agreement with the few previous studies, and the biologic plausibility all support the validity of our findings.

Both prevalence and incidence of dementia in the Stroke Data Bank cohorts were consistently and incrementally associated with advancing age. This expected, though striking, age dependency is consistent with findings from studies of dementia in other populations.¹⁷⁻²⁰ We are unaware of previous investigations of dementia in a large stroke cohort. Kotila et al²¹ studied a small group of stroke patients (mean age 49 years) who were examined for the occurrence of new-onset dementia defined by DSM-III criteria. Over 4 years, three of 37 patients with ischemic stroke became demented, giving a crude annual incidence rate of 2%. In several population studies,^{14,22,23} the annual incidence of dementia (chiefly Alzheimer's disease) ranged from 1.4% to 3.3% in the oldest age group. Thus, compared with these "baseline" rates in stroke-free populations, the

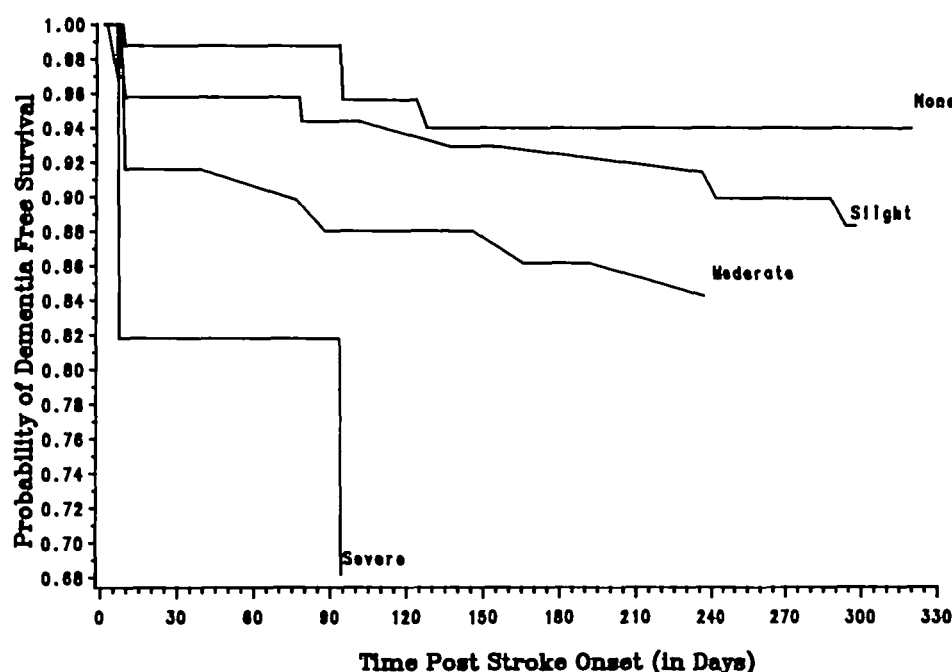


FIGURE 3. Kaplan-Meier estimate of survival free of dementia over 1 year by degree of cortical atrophy on computed tomogram at stroke onset.



FIGURE 4. Predicted incidence rate of dementia at 1 year by age at stroke onset, estimated by logistic regression model controlling for previous stroke, cortical atrophy, and stroke due to large-vessel atherosclerosis.

incidence rate that we observed in our non-population-based Stroke Data Bank cohort appears to be excessive.

The explanation for this elevated risk of dementia following stroke is not fully understood. One or more previous strokes evident clinically or on CT scan, whether symptomatic or not, were strongly associated with both prevalence and incidence of dementia. How multiple infarctions cause a dementia syndrome is yet to be determined. Injury of multiple brain regions, each specific for certain cognitive functions, might lead to dementia simply on an additive basis, a possibility encouraged by menu-driven diagnostic algorithms blinded to the expected clinical effects of focal lesions. Or, a multiplicative mechanism may apply, with the cumulative effect of several lesions, each individually innocuous or trivial in its clinical effects, leading to global mental decline classifiable as dementia. The latter effect, implied by the concept of "multi-infarct" dementia proposed by Hachinski et al²⁴ in 1974, still needs prospective validation.

The mechanism of ischemic stroke may be relevant to dementia. Hachinski et al²⁴ did not specify the size or aggregate volume of the multiple strokes leading to mental decline; however, the concept includes multiple lacunes, or "état lacunaire," which those authors considered "one of the commonest causes of vascular dementia." Yet, in the Stroke Data Bank prevalence of dementia was less commonly associated

with lacunar stroke than with other stroke subtypes; instead, dementia was most frequent in patients with large-vessel atherosclerosis, at least in the prevalence cohort among patients with no previous strokes. While dementia was also frequent with infarctions of unknown cause, this subtype probably includes both atherothrombotic and embolic mechanisms.²⁵ Thus, our studies do not support the relative importance of "lacunar dementia"²⁶ or "cardiogenic dementia"²⁷; rather, we propose that "distal-field dementia" may be more relevant in causing cognitive decline from stroke.

Carotid occlusive disease as a cause of dementia syndromes was proposed by Fisher²⁸ more than 35 years ago, although the mechanisms are still not clarified. Hemodynamic insufficiency has only rarely been demonstrated to cause mental incapacity.²⁹ Intellectual deterioration in the setting of carotid artery occlusion may be related to the focal topography of established infarction in the distal field territory, which includes the superior frontal, superior parietal, and the posterior temporo-occipital areas,³⁰ regions of the brain important for higher cerebral function. The role of brain location is suggested by the higher prevalence of dementia among patients with infarcts in the occipital, temporo-occipital, and temporoparietal regions, irrespective of stroke mechanism, in our prevalence cohort.

Cortical atrophy and accompanying hydrocephalus evident on CT scans were other strong determinants

TABLE 8. Predictors of Incidence of Dementia Based on Stepwise Proportional Hazards Model

Variable	β	SE	<i>p</i>
Model A			
Age	0.0232	0.0144	0.11
Previous stroke	0.8084	0.2174	<0.01
Atherothrombotic subtype	0.4075	0.3550	<0.25
Cortical atrophy	0.7134	0.1394	0.01
Model B			
Previous stroke	0.7999	0.2162	<0.01
Cortical atrophy	0.7667	0.1328	<0.01

$\chi^2=53.35$ for Model A, 49.84 for Model B.

of both prevalence and incidence of dementia in our Stroke Data Bank cohorts. Although the association between brain atrophy and dementia may be confounded in part by normal aging effects,³¹ the relation was prominent in our study, even after controlling for age. The simplest explanation might be that some patients had Alzheimer's disease prior to their stroke and presented with two disorders causally unrelated. Alternatively, preclinical Alzheimer's disease may have become apparent only when combined with the acute or chronic effects of the stroke, especially in the incidence cohort. The true proportion of our patients who had degenerative dementia complicated, aggravated, or unmasked by stroke effects in a synergistic way³² cannot be determined from our data.

The term "dementia" implies a change in state from one level of functioning to another over time. Thus, future studies of stroke cohorts should involve prospective, longitudinal observations using formal neuropsychological and functional assessments. Applying these methods, such studies should permit us to corroborate whether dementia was overdiagnosed or underdiagnosed in our Stroke Data Bank cohort and to verify or refute the risk factors identified in this study.

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