Incidence and Determinants of Poststroke Dementia as Defined by an Informant Interview Method in a Hospital-Based Stroke Registry

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Background and Purpose—Inconsistent information about incidence and determinants of poststroke dementia might be related to patient attrition, partly because of nonapplicability of formal neuropsychological testing to a large proportion of patients registered in a definite setting.

Methods—Using a proxy-informant interview based on ICD-10 criteria, we determined dementia at stroke onset and 1 year after stroke in the 339 patients who survived, were available for follow-up, and were not demented at stroke onset of 635 patients entered over a 1-year period in a stroke registry taken at 2 community hospitals in Florence, Italy.

Results—Of the 339 patients, 57 (16.8%) proved to have poststroke dementia. These patients were older, more frequently female, and more often (multivariate odds ratio, 2.35; 95% CI, 1.21 to 4.58) had atrial fibrillation than those without dementia. Aphasia and the clinical features expressing the severity of the stroke event in the acute phase predicted poststroke dementia.

Conclusions—In a hospital-based nonselected series of stroke survivors, despite the use of a method with low sensitivity for defining dementia, our study confirms that dementia is a frequent sequela of stroke and is mainly predicted by stroke severity. Certain determinants could be controlled in the prestroke phase, thus reducing its risk. (Stroke. 1998;29:2087-2093.)

Key Words: dementia ■ incidence ■ risk factors ■ stroke outcome

Information on the risk and determinants of poststroke dementia (PSD) is important in terms of cost evaluation and facility planning, as well as for indications about possible preventive intervention. This information, however, is still scanty and to some extent inconsistent. To our knowledge only a few prospective studies have reported data on this outcome. One is a population-based study performed in Rochester, Minn,1 whereas others are hospital-based studies, all reported very recently. 2–5 Among these studies, risk factors for PSD vary. In one study age, education, race, prior stroke, and diabetes mellitus were associated with PSD, while hypertension and cardiac disease were not.6 In another study atrial fibrillation significantly predicted PSD.7 In a recently reported study significant predictors of PSD were age, low educational level, and prior cerebrovascular events but not cardiac diseases or diabetes.7 Regarding the clinical presentation of stroke, pathological stroke subtypes (cortical, territorial, or lacunar stroke) have been found to be differently related to PSD.6,7 Most of the published surveys focused on first stroke or ischemic stroke only, excluding from the study sample patients with previous stroke or other stroke types. Moreover, because of the difficulties in applying a comprehensive, formal neuropsychological assessment to patients who are physically and neurologically impaired or present with aphasia or neglect, all of these studies examined only a subsample of the total patients registered in each setting. This selection may contribute to patient attrition, shown to have an impact on incidence and possibly risk factor data.8

We studied the incidence of PSD and its determinants (demographics, vascular risk factors, and clinical features in the acute phase), irrespective of the occurrence of a previous stroke and of stroke type, and using an informant interview method to establish whether patients were already demented before stroke onset or had become demented after the acute event. Patients were those surviving at 1 year of a total sample of 635 patients with acute stroke registered over a 1-year period in 2 community hospitals in the area of Florence, Italy.

Subjects and Methods

Setting and Patients

Index cases were all patients admitted for acute stroke to 2 general hospitals in the area of Florence, Italy, from October 1, 1993, to

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2087
September 30, 1994, and entered in a stroke registry. According to our institutional guidelines, all patients gave informed consent. This registry was implemented for evaluating costs of stroke care in relation to outcome within a project supported by the European Union (European Community Stroke Project).

The 2 hospitals are first-care community hospitals, since they admit the vast majority of patients presenting with stroke in the geographic area served by each one. The population in both areas is ~200,000. A collateral survey performed among general practitioners estimated that 90% of stroke cases occurring in these 2 areas are not hospitalized. Approximately half of these cases are mild strokes with quick recovery; the other half are severe strokes occurring in very old patients. Stroke was defined according to the World Health Organization criteria. Every stroke diagnosis was confirmed by a neurologist.

After registration, demographic and clinical information was gathered with the forms and the criteria specified by the European Community Stroke Project. In the present work, the possible predictors of PSD studied are as follows: (1) baseline characteristics, including age, sex, vascular risk factors, comorbid conditions (all listed below with related definitions), and prestroke level of handicap, as assessed by the Rankin Scale; (2) clinical state at time of maximum impairment: compromized level of consciousness, defined as follows: 1 = fully conscious, 2 = somnolent, 3 = semicomma, 4 = coma; site of motor deficit; severity of motor deficit, scored as follows: 1 = no deficit, 2 = weakness, 3 = paralysis in each of the 4 limbs, total severity score = sum of 4 limb scores; presence of speech or swallowing problems, urinary incontinence, confusion, or abnormal behavior; (3) specific stroke diagnosis: cerebral hemorrhage, subarachnoid hemorrhage, cerebral infarction, unclassified stroke; and (4) types of cerebral infarction, classified according to the criteria of Bamford et al into total anterior circulation infarction (TACI), partial anterior circulation infarction (PACI), posterior circulation infarction (POCI), and lacunar syndrome (LACI).

Vascular Risk Factors
Definitions for vascular risk factors were as follows: (1) atrial fibrillation: history of chronic atrial fibrillation, corroborated by ≥1 prior ECG and/or clinical evidence or ECG positive for atrial fibrillation on admission; (2) arterial hypertension: previous diagnosis or blood pressure values; (3) diabetes mellitus: previous diagnosis or blood glucose ≥11.1 mmol/L (≥200 mg/dL) after clinical stabilization; (4) smoking: previous smoking for duration of ≥24 hours’ duration; (5) hyperglycemia: blood glucose ≥200 mg/dL, in ≥2 recordings taken after clinical stabilization; (6) history of myocardial infarction: (a) previous diagnosis, or (b) history of typical symptoms with evidence of myocardial infarction on ECG, elevated serum enzymes levels, or atypical symptoms, unequivocal ECG signs of ischemia, or (c) typical symptoms, signs of myocardial infarction on ECG, and absence of normal serum enzymes level; (7) transient ischemic attack: history of focal neurological deficits of vascular origin of <24 hours’ duration.

Follow-Up
Three months and 1 year after the stroke event, trained personnel contacted all registered patients by telephone. In case of death, date and cause of death were collected and recorded. If the patient was alive, a follow-up visit was arranged either at the hospital or at home. All the visits were carried out by the attending physicians assisted by neurology residents.

Dementia Definition
Assessment was performed at admission and 1 year after the stroke event. To determine the presence of dementia we used an interview, based on the International Classification of Diseases, 10th Revision (ICD-10) criteria for dementia definition and administered to the proxy informant. Information was gathered by the interviewer (the physician performing the clinical assessment) with the aid of a checklist that followed the steps and the indications of ICD-10. The presence of dementia was established if, according to the informant, there were memory and intellectual deficits so severe as to interfere with everyday life activities and deterioration of both emotional control and behavior in the absence of clouding of consciousness. Memory and intellectual deficit had to be present for ≥6 months. This method was validated by the following studies. (1) The first study assessed sensitivity and specificity versus a standard: the subjects studied were 39 of the first 136 (mean age, 73.4 ± 5.2 years; mean education, 7.0 ± 3.8 years) screened for suspected dementia in a population study on aging (Italian Longitudinal Study on Aging) (ILSA), ongoing in the areas served by the study hospitals. First, the informant for each subject was interviewed by a physician following the method to be used in our study on PSD. The subjects were then assessed by a neurologist using the more extensive method adopted by ILSA for defining dementia. This method was based on criteria of the Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition (DSM-III-R) and also included criteria of the National Institute of Neurological Disorders and Stroke–Alzheimer’s Disease and Related Disorders Association, sections B and H of the Cambridge Mental Disorders of the Elderly Examination (CAMDEX), and the Pfeffer questionnaire on functional activities. The determination of dementia (whether present or absent) achieved by the ILSA method was taken as the gold standard for estimating sensitivity and specificity of the informant interview. Compared with the standard, our method proved to be 72.2% sensitive and 90.5% specific in defining dementia. (2) The second study assessed interobserver agreement: the informants for 48 patients (mean age, 68.6 ± 8.8 years; mean education, 6.9 ± 3.3 years) consecutively presenting for a visit at the Cerebrovascular Disease Clinic of our Neurology Department were independently interviewed by 2 observers, 1 senior neurologist and 1 young physician. The agreement, as measured by κ statistics, was substantial in rating memory deficit (weighted κ = 0.92, Z = 4.22) or intellectual decline (weighted κ = 0.94, Z = 4.16) or in the overall definition of dementia (weighted κ = 0.85, Z = 4.85).

Statistical Analyses
Differences in categorical variables among demented and non-demented patients were compared by means of odds ratios and related 95% CIs. P values of Pearson χ² test were also determined. To compare means of continuous variables we used the independent samples Student’s t test. A multiple logistic regression model with stepwise method for selection of variables was used to identify the best independent predictors of dementia among all the possible variables (either risk factors or clinical features in the acute phase). All calculations were done with SPSS for Windows version 7.5 (SPSS Inc.).

Results
Of the 635 patients with stroke registered in the 2 hospitals during the study period, 218 died between registration and 1-year follow-up assessment for dementia, and 39 were lost to follow-up. After exclusion of 39 patients who proved to be demented at entry, 339 patients remained and were assessed for PSD at 1 year. Compared with prestroke conditions, 57 patients (16.8%) were diagnosed as newly demented. This outcome is illustrated by the flowchart reported in the Figure. In each step patients with any type of stroke and those with ischemic stroke are indicated separately.

Table 1 reports the univariate comparison according to demographics, vascular risk factors, and prestroke handicap between the 57 patients with PSD and the 281 of 282 patients who at 1 year were not demented (1 patient was excluded from this analysis because of incomplete information about risk factors). Patients with PSD were older and more fre-
quently female than those without dementia. Atrial fibrillation and previous stroke were twice as frequent among demented compared with nondemented patients. No significant difference was observed for the other studied risk factors. A trend for smoking was observed more frequently among nondemented patients. Prestroke handicap, as assessed by the Rankin Scale (score >2), was 3 times more common among patients with PSD. All these differences were confirmed when patients with ischemic stroke only were considered.

Concerning stroke types and the risk of dementia, there were no significant differences between the 2 groups of demented versus nondemented patients in specific stroke diagnosis or in ischemic stroke syndromes (according to Bamford’s classification), although LACI was more common among nondemented patients (a difference of borderline significance) and TACI among demented patients (a nonsignificant trend).

Table 2 shows the differences between the 2 groups in clinical features at the time of maximum impairment in the acute phase. In this phase patients who were demented at 1-year follow-up were more often comatose, aphasic, and confused during the first week and more frequently had urine incontinence than patients who did not become demented. Moreover, patients who developed dementia had a higher degree of motor deficit. As opposed to aphasia, patients presenting with dysarthria had a lower probability of developing dementia (an effect of borderline significance).

When we examined by forward stepwise logistic regression analysis (Table 3) the net predictive effect of demographics, risk factors, and acute-phase variables, age, prestroke Rankin, atrial fibrillation, aphasia, and urine incontinence were all significantly and independently associated with PSD, while previous stroke had only a borderline effect.

Since aphasic patients might have been more prone to be classified as demented, we repeated this multivariate analysis using, instead of aphasia at stroke onset, the presence of aphasia as determined at 1 year, concomitantly with the assessment for dementia. Age, prestroke Rankin, atrial fibrillation, and urine incontinence were confirmed as independent predictors of PSD.

A variable proportion of patients who enter in a stroke registry die, and some may be lost to follow-up. This phenomenon may also contribute to patient attrition. Characteristics of patients who die may vary across different study settings. To evaluate the characteristics of this subsample of patients in our study, we examined the differences in demographics, vascular risk factors, and acute-phase clinical features between the patients who were seen at the 1-year follow-up and those who were not (dead or lost at follow-up) (Table 4). The latter were older, more frequently female, and more often had atrial fibrillation, aphasia or dysarthria, swallowing problems, and urinary incontinence. In contrast, hypertensives, diabetics, and smokers were less frequent.

**TABLE 1. Predictors of PSD: Demographics, Vascular Risk Factors, and Prestroke Rankin (Univariate Analysis)**

<table>
<thead>
<tr>
<th></th>
<th>Demented (n=57)</th>
<th>Nondemented (n=281)*</th>
<th>Odds Ratio (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean±SD age, y</td>
<td>76.2±9.4</td>
<td>70.0±11.5</td>
<td>1.78 (1.00–3.17)</td>
<td>0.049</td>
</tr>
<tr>
<td>Female sex</td>
<td>34 (59.6%)</td>
<td>128 (45.4%)</td>
<td>2.35 (1.21–4.58)</td>
<td>0.049</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>16 (28.1%)</td>
<td>40 (14.2%)</td>
<td>1.01 (0.57–1.79)</td>
<td>0.968</td>
</tr>
<tr>
<td>Hypertension</td>
<td>31 (54.4%)</td>
<td>152 (54.1%)</td>
<td>1.36 (0.73–2.54)</td>
<td>0.324</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>18 (31.6%)</td>
<td>71 (25.3%)</td>
<td>1.04 (0.51–2.15)</td>
<td>0.902</td>
</tr>
<tr>
<td>Smoking</td>
<td>5 (8.8%)</td>
<td>51 (18.1%)</td>
<td>0.43 (0.16–1.14)</td>
<td>0.082</td>
</tr>
<tr>
<td>Previous smoking</td>
<td>12 (21.1%)</td>
<td>64 (22.8%)</td>
<td>0.90 (0.45–1.81)</td>
<td>0.776</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>12 (21.1%)</td>
<td>26 (9.0%)</td>
<td>2.61 (1.23–5.56)</td>
<td>0.010</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>6 (10.5%)</td>
<td>27 (9.6%)</td>
<td>1.11 (0.43–2.82)</td>
<td>0.831</td>
</tr>
<tr>
<td>Transient ischemic attack</td>
<td>12 (21.1%)</td>
<td>49 (17.4%)</td>
<td>1.26 (0.62–2.56)</td>
<td>0.517</td>
</tr>
<tr>
<td>Prestroke Rankin</td>
<td>3 (7.0%)</td>
<td>6 (2.1%)</td>
<td>3.47 (0.95–12.72)</td>
<td>0.047</td>
</tr>
</tbody>
</table>

*One patient in the nondemented group was omitted from the analysis of baseline characteristics because of incomplete information about risk factors.
Discussion

Our results confirm previous indications that PSD is a frequent sequela of stroke. Age, prestroke disability, and certain clinical features expressing the severity of the event at stroke onset are major predictors of PSD. Among risk factors for stroke, atrial fibrillation proved an independent determinant of PSD.

We have previously indicated the main reasons for our choice of an informant interview for defining dementia. We further justify this choice as follows: (1) To establish the presence or absence of dementia before stroke in a hospital-based study of PSD, the use of historical information is the only possible method, and therefore an informant interview was chosen. For the sake of comparability, the same method should be used to determine the presence of dementia after stroke. (2) As in previous studies, formal neuropsychological testing is inapplicable in a number of patients (particularly those with severe neurological deficits); this implies, if only possible method, and therefore an informant interview based study of PSD, the use of historical information is the nant of PSD.

methods for defining dementia in selective settings. (4) Compared with a more formal method for determining the presence of dementia, our method proved highly specific and sufficiently sensitive. (5) This method was found to be reproducible in the setting of cerebrovascular disease patients. In contrast to the poor agreement in diagnosing dementia by clinical judgment in the first Stroke Data Bank Study \((\kappa=0.34)\), our method was definitely more reproducible \((\kappa=0.85)\).

The relatively low sensitivity of the method we used suggests that our incidence figure for PSD is underestimated. The low sensitivity might also be related to the use of ICD-10 criteria, which recently have been demonstrated to be more specific but less sensitive than other criteria for defining PSD.\(^24\) When dementia is associated with progressive decline, 2 assessments over time (soon after and 1 year after the stroke event, as in our study) may be not sufficient to define dementia. Therefore, we have possibly failed to count every patient with a progressive type of dementia. Additionally, this fact might have led to underestimation of the incidence of PSD.

**TABLE 3. Independent Predictors of PSD (Multiple Logistic Regression Model Including Both Risk Factors and Acute-Phase Clinical Features)**

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Odds Ratio (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;72 years*</td>
<td>2.78 (1.36–5.68)</td>
<td>0.005</td>
</tr>
<tr>
<td>Prestroke Rankin &gt;2</td>
<td>1.52 (1.03–2.24)</td>
<td>0.034</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>2.33 (1.09–4.98)</td>
<td>0.029</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>2.52 (0.99–6.40)</td>
<td>0.052</td>
</tr>
<tr>
<td>Aphasial</td>
<td>2.23 (1.13–4.40)</td>
<td>0.020</td>
</tr>
<tr>
<td>Urinary incontinence</td>
<td>3.28 (1.63–6.58)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*The variable age was dichotomized using as cutoff median age of the 339 study patients.
Compared with other hospital-based studies and owing to the free access and the nature of our study hospitals (first care, community-based), our cases are more representative of all acute stroke cases occurring in the population. To avoid further selections, all strokes listed in our registry were included in our study sample, independently of pathological type or first-ever/recurrent stroke. However, in a hospital-based study, data on incidence and determinants of PSD may vary because of the different number of patients who die in the postacute phase or are lost to follow-up. When we examined the effect of this selection on our study, we observed that, in the subsample of stroke patients who died or were lost, the predictors we have established in relation to PSD were even more present: the consequence of this selection may be an underestimation of our incidence figure. The determinants we found, however, seem unbiased. The patients we lost to follow-up were few and probably did not induce a relevant distortion.

Despite the use of a rather insensitive method for defining dementia, a considerable proportion of acute stroke patients still appear at risk of PSD. This risk seems higher among old and female patients. Of the preexisting vascular risk factors, only atrial fibrillation had an independent predictive effect on PSD. Almost all the clinical variables expressing stroke severity in the acute phase predicted PSD in the univariate analysis, although only aphasia and urine incontinence maintained an independent effect after multivariate analysis.

Data from our registry reported elsewhere showed that patients presenting with atrial fibrillation at onset, compared with those without, have a more severe stroke event. The results of the multivariate analysis, examining in the same model both prestroke risk factors and clinical features of the acute phase, seem to exclude that the effect of atrial fibrillation on PSD is dependent on stroke severity. Having suffered a previous stroke increases the risk of dementia, but in our study this factor was less strong than expected.

In a comparison of our results with those reported in the few previous studies, the risk of dementia at 1 year (17.7%) among our patients with ischemic stroke is remarkably lower than that (26.3%) reported at 3 months in the first series of Tatemichi et al of patients registered in the Stroke Data Bank. In this study only clinical judgment was used for defining dementia, but agreement among the observers was poor. In contrast, our figure is almost equal to the 1-year 17.9% figure (on an actuarial basis) reported by Tatemichi et al in a second study, in which a larger patient sample was followed up, and a more comprehensive neuropsychological examination was used to determine the presence of dementia. Our incidence was slightly higher than that estimated at 3 months by Censori et al (13.6%) in a cohort of patients with first-ever ischemic stroke. In that study neuropsychological testing was used for examining cognitive functions in the majority of patients, but an informant interview had to be used for defining dementia in aphasic patients. Our rate was

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### TABLE 5. Exclusions in Prior PSD Studies

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Age, y</th>
<th>Cause for Exclusion</th>
<th>Number Excluded/Total Stroke Patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tatemichi et al</td>
<td>1990</td>
<td>&gt;60</td>
<td>Impaired consciousness</td>
<td>159/925</td>
<td>17.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Aphasial</td>
<td>192/927</td>
<td>20.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hemineglect</td>
<td>81/927</td>
<td>8.7</td>
</tr>
<tr>
<td>Gorelick et al</td>
<td>1993</td>
<td>&gt;45</td>
<td>&lt;2 infarcts from history</td>
<td>316/782</td>
<td>40.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Aphasial</td>
<td>126/782</td>
<td>16.1</td>
</tr>
<tr>
<td>Moroney et al</td>
<td>1996</td>
<td>&gt;60</td>
<td>Prior cerebral ischemic events</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Severe aphasial</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Concomitant CNS disorder</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Kokmen et al</td>
<td>1996</td>
<td>NR</td>
<td>Neurological impairment/aphasia</td>
<td>71/1138</td>
<td>6.2</td>
</tr>
<tr>
<td>Censori et al</td>
<td>1996</td>
<td>40–80</td>
<td>Deficit &lt;24 h</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Alcohol &gt;600 g/wk</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Blindness, deafness</td>
<td>158/304</td>
<td>52.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Depression</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Lindsay et al</td>
<td>1997</td>
<td>&gt;65</td>
<td>Stroke &gt;3 y from admission</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Prencipe et al</td>
<td>1997</td>
<td>&gt;65</td>
<td>Severe aphasial</td>
<td>8/80</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Depression</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Pohjasvaara et al</td>
<td>1997</td>
<td>55–85</td>
<td>Intracerebral hemorrhage</td>
<td>229/1447</td>
<td>15.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Subarachnoid hemorrhage</td>
<td>69/1447</td>
<td>4.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>&lt;55 y</td>
<td>258/1147 (ischemic)</td>
<td>22.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>&gt;85 y</td>
<td>88/1147</td>
<td>7.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Aphasial</td>
<td>32/486</td>
<td>6.6</td>
</tr>
</tbody>
</table>

NR indicates not reported; CNS, central nervous system.

*Hospital-based study.

†Population-based study.
definitely lower than the 31.8% reported recently by Pohjasvaara et al., who used the DSM-III criteria and an extensive and detailed neuropsychological examination for diagnosing dementia. Desmond et al. obtained largely variable incidence rates using different criteria for defining dementia in a stroke patient cohort followed up for 3 months. Our figure is close to that estimated by these authors when the presence of dementia was established by clinical judgment or by clinical judgment combined with functional impairment evaluation (16.4% in both cases).

Regarding determinants of PSD, the results of our study confirm the effect of age consistently reported by all previous studies. Prior or recurrent stroke has been indicated as a risk factor for PSD by the studies of Tatemichi et al., Kokmen et al., and Pohjasvaara et al. Recurrent stroke in patients who survived at 1 year was infrequent in our registry. However, stroke occurred in 5.3% of patients in the group of demented patients and in 1.4% of patients in the nondemented group (odds ratio, 3.86; 95% CI, 0.84 to 17.74). Diabetes was an independent precursor in the studies of Tatemichi et al and Censori et al. However, in contrast with the other studies, we did not find lesion location in the dominant hemisphere to be an independent predictor of PSD. Regarding lesion type, our results would indirectly support the hypothesis that atrial fibrillation is more important for PSD than hypoperfusion due to cardiac pump deficit. Other vascular risk factors, such as hypertension and smoking seem irrelevant. In the study of Pohjasvaara et al., significant risk factors for PSD were age, low educational level, prior ischemic stroke, and any prior cerebrovascular disease. Among the clinical features of stroke, major dominant hemisphere syndrome, total score on the Scandinavian Stroke Scale, dysphasia, gait impairment, and urinary incontinence were all significantly associated with PSD. On logistic regression analysis odds ratios were increased by a factor of 5 for dysphasia and major dominant hemisphere syndrome.

Our results regarding the clinical predictors of PSD appear substantially consistent with those reported in the previous hospital-based studies, which used more sensitive methods for defining dementia. In our study, as in previous studies, PSD was primarily related to the clinical severity of the stroke event (also to CT volume of the infarct in the study of Censori et al). However, in contrast with the other studies, we did not find lesion location in the dominant hemisphere to be an independent predictor of PSD. Regarding lesion type, our data do not confirm that lacunar infarct or any other pathological stroke subtype contributes selectively to PSD.

In conclusion, even if a method with relatively low sensitivity was used for defining dementia, our study confirms that PSD is a frequent sequela of hospitalized stroke. Age, atrial fibrillation, and the severity of stroke predict PSD. PSD should be taken into account in evaluating the burden of care and social support of patients with stroke. Treatable factors are involved in the risk of PSD, and adequate prevention may reduce this burden. The observation that atrial fibrillation is a determinant of PSD further stresses the importance of using oral anticoagulants for stroke prevention in patients with atrial fibrillation.

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for the participants in the European Community Project Florence Stroke Registry

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