Silent Infarction in Acute Stroke Patients
Prevalence, Localization, Risk Factors, and Clinical Significance: The Copenhagen Stroke Study

Henrik Stig Jørgensen, MD; Hirofumi Nakayama, MD; Hans Otto Raaschou, MD; Jørgen Gam, MD; Tom Skyhøj Olsen, MD, PhD

Background and Purpose Our objective was to study age-specific prevalence, computed tomographic (CT) characteristics, risk factors, and the prognostic influence on stroke outcome of silent infarction in acute stroke patients.

Methods The study was prospective and community-based and included 801 acute stroke patients, of whom 587 had first-ever stroke. A CT scan was performed in 500 (85%) of the 587 patients with first-ever stroke. CT was reviewed blindly, and infarcts were classified according to patient history as silent or symptomatic. Patients were evaluated initially with the Mini-Mental State Examination (MMSE) and weekly with both the Scandinavian Stroke Scale (SSS) and the Barthel Index (BI) from the onset of stroke to completion of rehabilitation. CT characteristics, risk factors, and stroke outcome were compared in stroke patients with and without silent infarction.

Results The prevalence of silent infarction in patients with first-ever stroke and recurrent strokes was similar, at 29% (group aged 0 to 54 years, 16%; 55 to 64 years, 22%; 65 to 74 years, 30%; 75 years or older, 33%). Silent infarcts were small and subcortical. Independent risk factors were increasing age (odds ratio [OR], 1.95 per 25 years; confidence interval [CI], 1.19 to 3.15), hypertension (OR, 1.75; CI, 1.13 to 2.70), claudication (OR, 1.74; CI, 1.01 to 3.00), and male sex (OR, 1.72; CI, 1.12 to 2.64); other stroke risk factors such as atrial fibrillation and former transient ischemic attack were not independent risk factors. Patients with and without silent infarction did not differ in frequency of prestroke home care (P=0.7), MMSE (P=0.56), initial BI (P=0.62) and SSS score (P=0.08), BI (P=0.85) and SSS score (P=0.75) after completion of rehabilitation, or in the speed of recovery (P=0.85). Length of hospital stay, mortality rate, and discharge rate to nursing home also did not differ between the two groups.

Conclusions This community-based study shows that silent infarction in stroke patients is more related to certain stroke risk factors than others and that silent infarction does not seem to influence the prognosis of stroke.
TABLE 1. Number of Patients, Age, and Sex in the Copenhagen Stroke Study

<table>
<thead>
<tr>
<th>Category</th>
<th>n</th>
<th>Mean±SD Age, y</th>
<th>Sex, M/F</th>
</tr>
</thead>
<tbody>
<tr>
<td>All stroke patients</td>
<td>801</td>
<td>74.3±11.5</td>
<td>371/430</td>
</tr>
<tr>
<td>Former stroke unknown</td>
<td>43</td>
<td>80.7±7.8</td>
<td>11/32</td>
</tr>
<tr>
<td>Recurrent stroke</td>
<td>171</td>
<td>74.7±10.4</td>
<td>89/82</td>
</tr>
<tr>
<td>First-ever stroke</td>
<td>587</td>
<td>73.7±11.8</td>
<td>271/316</td>
</tr>
<tr>
<td>CT in first-ever stroke</td>
<td>500</td>
<td>73.0±12.0</td>
<td>236/264</td>
</tr>
</tbody>
</table>

CT indicates computed tomography.

To elucidate some of these problems and controversies, we present our data from the Copenhagen Stroke Study.

Subjects and Methods

Information from all patients admitted with an acute stroke to the Neurological Department of Bispebjerg Hospital, Copenhagen, has been collected prospectively and entered into a computerized data bank since September 1, 1991 (the Copenhagen Stroke Study). The study population is community-based. First, Bispebjerg Hospital serves a well-defined community with 239,886 inhabitants within the city of Copenhagen. Second, hospital care is free, and a very high proportion of stroke patients are admitted to the hospital; in a neighboring area within Greater Copenhagen it was recently shown that 88% of all stroke patients are hospitalized. Third, Bispebjerg Hospital is the only hospital serving the region. Finally, all persons from the community who have an acute cerebrovascular disease that requires admission are referred to the neurological department. Not only are initial diagnostic procedures and treatment performed at the neurological department, but also all stages of rehabilitation, regardless of the age of the patient, the severity of stroke, and the condition of the patient before the stroke.

Exclusion Criteria

A history of former stroke was not available in 43 patients (30 patients were unconscious, and 13 were deceased) (Table 1). These patients were significantly older than the rest of the patients (mean±SD age, 80.7±7.8 years versus 74.3±11.5 years; P<.001).

Patients were divided according to whether they had first-ever-in-a-lifetime stroke (hereafter referred to as first-ever stroke) or if they had a history of former stroke (hereafter referred to as recurrent stroke). Patients with recurrent stroke (n=171) were excluded from the study (Table 1), except that the prevalence of silent infarction was also studied in this group. Mean age was not significantly different between patients with recurrent stroke (74.7±10.4 years) and patients with first-ever stroke (73.7±11.8 years) (P=.32).

Patients in whom a CT scan was not performed were also excluded from the study. This was the case in 87 (15%) of the 587 patients with first-ever stroke; either the patient was too ill to be transferred to the CT department (n=27), the patient died before a planned CT scan could be performed (n=51), or the patient refused further investigation (n=9). Mean age of these patients was significantly higher (78.1±9.3 years) than mean age of the patients in whom a CT scan was performed (73.0±12.0 years). However, in a multiple logistic regression analysis of risk factors in patients with and without CT, only age differed between the two groups.

Consequently, a total of 500 patients with first-ever stroke were included in the study.

The Neurological Department of Bispebjerg Hospital

The Neurological Department of Bispebjerg Hospital is specially designed and equipped to handle stroke patients from the time of admission to the end of rehabilitation. It is the largest department for stroke patients in Denmark, with 74 beds, of which approximately 60 are used for stroke patients. The nursing and training staff (including physiotherapists, occupational therapists, speech therapists, neuropsychologists, and social workers) are educated in the handling of stroke patients. Rehabilitation is performed according to the principles of Bobath and offered to all patients. Rehabilitation continues until no further improvement is reached. The patient is then discharged either to home or, if the patient is unable to carry on an independent life at home, to a nursing home.

Definition of Acute Stroke

Stroke was defined according to World Health Organization criteria: rapidly developed clinical signs of focal disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than vascular origin. Subarachnoidal bleeding was not included.

Information Regarding Former Stroke and Transient Ischemic Attack

A history of former stroke or transient ischemic attack (TIA) including a description of side localization and initial symptoms was obtained on admission. The hospital register containing information on diagnosis from former admissions was also studied.

Computed Tomographic Measurements

The CT scan was performed with a Siemens Somatom DR scanner. Contrast was not given routinely. The time from onset of stroke to CT examination was dependent on the accessibility of the scanner, which varied during the study period. In most cases it was made within the first 2 weeks (mean±SD, 11.9±12.7 days). Usually it was not repeated. All scans were described by the same neuroradiologist with no knowledge of clinical data. Description included type, size, age, and region of lesion(s).

Size was measured in millimeters as the largest visible diameter of the infarct on CT. An estimation of the age of the infarct as new, old, or unknown was attempted. Infarction was divided according to whether or not cortical structures were involved. The term cortical was applied if infarction involved the term subcortical was used if no cortical structures were affected on CT.

Only distinct circumscribed hypodensities were regarded as sign of cerebral infarction. This excluded white matter hypodensities such as leuoencephalopathy and local atrophy.

Definition and Classification of Silent Infarction

Silent infarction was diagnosed if CT examination revealed an infarct that could not be related to either the present stroke or to a former stroke. This evaluation was performed by a neurologist who simultaneously reviewed patient history and CT description.

Risk Factors

The following risk factors were considered: age, sex, AF, intermittent claudication, daily alcohol consumption, current smoking, hypertension, diastolic and systolic blood pressure on admission, diabetes, ischemic heart disease (IHD), former
myocardial infarction (MI), former TIA, and ischemic changes on ECG. These risk factors were defined as follows: AF: if present on ECG obtained on admission; intermittent claudication: a history of intermittent claudication; current smoking: daily smoking of any kind of tobacco; daily alcohol consumption: patients were divided into the three groups of (1) no daily alcohol consumption; (2) moderate daily alcohol consumption (corresponding to the alcohol content of from one to five bottles of ordinary beer, glasses of wine, etc); and (3) high daily alcohol consumption (equivalent to more than five beers or glasses of wine daily); diabetes: a history of diabetes or diabetes diagnosed during the hospital stay; former MI: a clinical event diagnosed as an MI confirmed by hospital records and either ECG and/or enzymes; IHD: a history of IHD or IHD diagnosed during the hospital stay; ischemic changes on ECG: ECG showing signs of former MI; and hypertension: either (1) in treatment with antihypertensive drugs at the time of admission or (2) hypertension diagnosed during the hospital stay.

Clinical Assessment

The Scandinavian Stroke Scale (SSS) was used to assess neurological deficits.13,14 The SSS evaluates level of consciousness, eye movement, power in arm, hand, and leg(s); hemiplegia; aphasia; facial paresis; and gait. The score ranges from 0 to 58 points. It was performed on admission, the day after admission, every week during the hospital stay, and at discharge. Gain was calculated by subtraction of admission score from discharge score. To assess the speed of recovery, time in weeks from admission to achievement of highest SSS score was calculated for each patient. The weekly assessment was performed by the same neurologist (H.S.J.) in all patients. Subscores in the SSS were used for assessment of level of consciousness, orientation, and presence of aphasia on admission.

The Barthel Index (BI) score was used for the assessment of functional ability.15 It evaluates 10 different abilities and ranges in score from 0 to 100 points. It was measured by the nursing and training staff during the first week after admission, each week during hospital stay, and at discharge. Gain was calculated by subtraction of admission score from discharge score. To assess the speed of recovery, time in weeks from admission to achievement of highest Barthel score was calculated for each patient.

The Mini-Mental State Examination (MMSE)16 was performed on admission to test the mental abilities of the patient. The score ranges from 0 to 30 points.

Statistical Analysis

Statistics were performed using the SPSS package. In univariate analysis of risk factors and the influence of silent infarction on outcome, the Student’s t test was used for continuous data and the x² test for noncontinuous data. A logistic multiple regression model was used to calculate independent risk factors for silent infarction. All variables of interest were tested using the backward procedure, and variables that had a value of P<.2 were then entered using the forward procedure to attain as much information as possible. Linear multiple regression analysis was used to determine the possible clinical significance of silent infarction. The level of significance was chosen to be P<.05.

Ethics

The study was approved by the Ethics Committee of Copenhagen, approval No. V. 100.2263/91.

Results

Prevalence

Silent infarction was present in 147 (29.4%) of the 500 patients with first-ever stroke. One hundred thir-teen (22.6%) patients had 1 silent infarct, 28 (5.6%) patients had 2 silent infarcts, and 6 (1.2%) patients had 3 silent infarcts. None had more than 3 silent infarcts. In total, 186 silent infarcts were detected in the 147 patients. The age-specific prevalence rates are given in Fig 1. In patients with recurrent stroke, the prevalence of silent infarction was 28.5%. This prevalence is not different from the prevalence found in first-ever stroke patients (P=.83).

In 322 (64.4%) of the 500 first-ever stroke patients, CT showed an infarct to be the cause of the present stroke. In 39 (7.8%) patients the cause was an intracerebral hemorrhage, and in the remaining 139 (27.8%) patients CT revealed no focal abnormality connected to the present stroke. Only 4 (10.3%) of the 39 patients with a hemorrhage had a silent infarct, compared with 105 (32.5%) of the 322 patients with a symptomatic infarct and 38 (27.4%) of the 139 patients with no CT abnormality in relation to the present stroke. The low prevalence of silent infarction in patients with intracerebral hemorrhage was significant (P=.006).

Size and Location

Table 2 shows data regarding size, cortical/subcortical location, side, and regional involvement in both silent and symptomatic infarcts. Silent infarcts were significantly smaller (15.5 mm versus 35.4 mm) than symptomatic infarcts (P<.0001), and 60% of the silent infarcts were smaller than 15 mm. Silent infarcts were more frequently subcortical (88% versus 58%; P<.0001) and more often located in the basal ganglia or internal capsule (68% versus 37%; P<.0001). The symptomatic infarct in patients with silent infarction was significantly smaller than the symptomatic infarct in patients without silent infarction (29.5 mm versus 38.1 mm; P=.005), was less often cortically placed (30% versus 51%; P<.0005), and more often involved the basal ganglia or internal capsule (P=.0001) (Table 3).

Risk Factors

Results from the univariate analysis of risk factors for silent infarction in patients with acute stroke are presented in Table 4. Both increasing age and hypertension were significantly related to the presence of silent infarction (P=.001), whereas AF, former MI, ischemic changes on ECG, current smoking, daily alcohol consumption, diabetes, and former TIA were not.
Using a logistic regression model to search out independent risk factors, we found age (odds ratio [OR], 1.95 per 25 years; confidence interval [CI], 1.19 to 3.15), male sex (OR, 1.72; CI, 1.12 to 2.64), hypertension (OR, 1.75; CI, 1.13 to 2.70), and intermittent claudication (OR, 1.74; CI, 1.01 to 3.00) to be independent risk factors for silent infarction (Fig 2).

Daily alcohol consumption, AF, former MI, and former TIA tended to reduce the risk of silent infarction, but this did not reach statistical significance.

Clinical Significance

The results of a univariate analysis of outcome variables comparing outcome of acute stroke in patients with and without silent infarction are shown in Table 5. Home care was equally common in the two groups before the stroke. Patients with silent infarction were significantly older than patients without silent infarction. A poorer outcome should therefore be expected, but this difference in age was counterbalanced by the size of the symptomatic infarct in patients with silent infarction, which was significantly smaller than the symptomatic infarct in patients without silent infarction. The frequency of aphasia, disorientation, lowered level of consciousness, and score obtained in the MMSE on admission were not different in patients with and without silent infarction. There was no significant difference in neurological score or in BI score between the two groups, either on admission or at discharge. Mortality rate during hospital stay and discharge rate to nursing home were similar in patients with and without silent infarction. Regarding the speed of recovery, the highest scores on both the SSS and BI were reached at the same time in both groups, and the length of hospital stay did not differ between the two groups.

Finally, in a linear multiple regression model, we tested the independent influence of the presence of silent infarction on both SSS and BI scores at discharge, together with SSS and BI scores obtained on admission, patient age, sex, and size of infarct. In accordance with the univariate analysis, we found no significant influence of the presence of silent infarction on either SSS ($P=.47$) or BI ($P=.70$) scores at discharge. Logistic regression was used to test if the presence of silent infarction was significantly associated with higher risk of in-hospital mortality in patients with silent infarction.

### Table 2. Silent and Symptomatic Infarcts: Total Number, Size, Side, and Location

<table>
<thead>
<tr>
<th></th>
<th>Silent Infarcts</th>
<th>Symptomatic Infarcts</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of infarcts</td>
<td>186</td>
<td>322</td>
<td></td>
</tr>
<tr>
<td>Diameter of infarct, mm (SD)</td>
<td>15.5 (8.7)</td>
<td>35.4 (25.7)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Cortical/subcortical</td>
<td>23/163</td>
<td>134/188</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Side location, L/R</td>
<td>88/98</td>
<td>159/163</td>
<td>.65</td>
</tr>
<tr>
<td>Frontal lobe</td>
<td>19</td>
<td>90</td>
<td>28</td>
</tr>
<tr>
<td>Temporal lobe</td>
<td>8</td>
<td>62</td>
<td>19</td>
</tr>
<tr>
<td>Parietal lobe</td>
<td>19</td>
<td>93</td>
<td>29</td>
</tr>
<tr>
<td>Occipital lobe</td>
<td>6</td>
<td>34</td>
<td>11</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>10</td>
<td>14</td>
<td>4</td>
</tr>
<tr>
<td>Brain stem</td>
<td>2</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Basal ganglia or internal capsule</td>
<td>127</td>
<td>119</td>
<td>37</td>
</tr>
</tbody>
</table>

### Table 3. Size, Location, and Regional Involvement of Symptomatic Infarcts in First-Ever Stroke Patients With and Without Silent Infarction

<table>
<thead>
<tr>
<th></th>
<th>With Silent Infarction</th>
<th>No Silent Infarction</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>105</td>
<td>217</td>
<td></td>
</tr>
<tr>
<td>Diameter of infarct, mm (SD)</td>
<td>29.5 (23.0)</td>
<td>38.1 (26.5)</td>
<td>.005</td>
</tr>
<tr>
<td>Side, L/R</td>
<td>55/50</td>
<td>135</td>
<td>.45</td>
</tr>
<tr>
<td>Cortical/subcortical</td>
<td>29/76</td>
<td>105/111</td>
<td>.0004</td>
</tr>
<tr>
<td>Frontal lobe</td>
<td>18</td>
<td>72</td>
<td>33</td>
</tr>
<tr>
<td>Temporal lobe</td>
<td>10</td>
<td>52</td>
<td>24</td>
</tr>
<tr>
<td>Parietal lobe</td>
<td>23</td>
<td>70</td>
<td>32</td>
</tr>
<tr>
<td>Occipital lobe</td>
<td>11</td>
<td>23</td>
<td>11</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>4</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Brain stem</td>
<td>0</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Basal ganglia + internal capsule</td>
<td>54</td>
<td>65</td>
<td>30</td>
</tr>
</tbody>
</table>
infarction had any independent influence on mortality and the discharge rate to nursing home. The test was performed with the same independent variables as stated above. Silent infarction had no influence on these outcome measures \(P=0.39\).

**Discussion**

In this study we included all hospitalized acute stroke patients from an area with a population of 239,886 inhabitants in the city of Copenhagen. Approximately 90% of all strokes are referred to the hospital in this area.\(^1\) The study can therefore be regarded as a prospective and community-based study of silent infarction in stroke patients. A CT scan was performed in a very high proportion of patients (85%), and risk factors except age did not differ between stroke patients with and without CT. The study is therefore representative of the total stroke population.

The care of stroke patients in hospitals is often organized in a way that makes it difficult to obtain detailed and reliable information regarding the whole course of stroke for all stroke patients. Handling of the patient in the acute stage is frequently performed at one department and rehabilitation at another department, or even at different hospitals. Furthermore, patients are often treated at different departments according to age or stroke severity. In this study we were able to monitor the whole course of stroke closely in each patient because all patients from the community with an acute stroke were referred to, treated, and rehabilitated at the same department.

An ideal study of the prevalence of silent infarction should include all stroke patients in a community-based stroke population and not only first strokes. The definition of silent infarction in stroke patients has varied in former studies of the frequency of silent infarction. Herderschee et al\(^3\) assumed in a study of patients with TIA and minor stroke that patients with normal or almost normal function in regard to activities of daily living (ADL) had no former stroke(s) and that infarcts in these patients were then silent if they did not relate to the present stroke or TIA. Strokes with full remission might then have been classified as silent. In other studies,\(^1,2,4\) patients with previous stroke were excluded. Dealing only with first-ever stroke patients is methodologically an advantage because an infarct visualized by CT is then either (1) the cause of the patient’s present symptoms or (2) a silent infarct. The risk of "false"
silent infarcts, ie, symptomatic infarcts believed to be silent, is greater if patients with recurrent strokes are included because information regarding symptoms and side of former strokes becomes less accurate as time goes by. Exclusion of patients with previous stroke thereby diminishes the bias of false silent infarcts. On the other hand, information about silent infarction in recurrent stroke patients will be lost. We have therefore attempted to also classify infarcts in recurrent stroke patients as symptomatic or silent, but only to determine the prevalence in patients with recurrent stroke.

### Prevalence

Silent infarction was present in approximately 30% of all acute stroke patients in this study. This figure was similar in first-ever stroke patients as well as in recurrent stroke patients. The prevalence is lower than in an MRI study of stroke patients in which a frequency of 47% was found,1 but it is higher than that reported in most other studies using CT, in which frequencies ranged between 10% and 38%.1-4 However, these studies are not directly comparable to the present study. MRI is more sensitive than CT in detecting small infarcts, mainly because of the higher resolution of this method. The specificity of MRI as well as of CT in diagnosing silent infarcts is, however, not certain: changes on MRI scans considered to be silent infarcts were found in as many as 47% of 101 normal volunteers,17 and the study failed to uncover any statistically significant association of white matter changes with the diagnosis of stroke. Likewise, changes on CT scans considered to be silent infarcts were found in 10% of 30 normal volunteers.18

Chodosh et al1 found in 1203 selected first-stroke patients that 13% had silent infarction, but the study was not community-based. In the Framingham Study,2 the prevalence of silent infarction was 10%, but the study was based on small numbers, ie, 13 silent infarcts in 124 first-ever stroke patients. Herderschee et al3 found silent infarction in 11% of 2329 patients participating in a secondary prevention trial, but only patients with TIA and minor stroke were included, and the study was not community-based. Ricci et al4 found in a community-based study silent infarction to be present in 80 (38%) of 209 patients with first-ever stroke. However, only 56% of the patients had CT scan, and silent infarcts were diagnosed just as frequently as symptomatic infarcts. Furthermore, age-specific prevalence rates are needed to compare rates between studies, because age (see “Risk Factors”) is a strong independent risk factor for silent infarction. Mean age was higher in the present study compared with other studies, and the finding of a higher prevalence of silent infarction in the present study than found in most other studies was therefore expected. However, it is not possible to give a safe estimate of the influence of age on the variation in frequency of silent infarction reported in the previous studies,1-4 because age-specific prevalence rates were not calculated. In the present study the age-specific prevalence rate of silent infarction rose from 16% in stroke patients aged younger than 55 years to 33% in patients aged older than 75 years.

### Characteristics of Silent Infarction on Computed Tomography

The general characteristics of the silent infarct in regard to size, side, location, and topography (cortical
Silent infarctions are in general found to be small and subcortical. The subcortical nature of the silent infarction was even more pronounced in the present study, in which 88% of the silent infarcts were subcortical compared with 53% in Reference 2, 59% in Reference 1, and 79% in Reference 3. The mean diameter of the silent infarcts was 15.5 mm compared with 35.4 mm in symptomatic infarcts; 60% of the silent infarcts were smaller than 15 mm, confirming that silent infarcts are in general small. The silent infarct was located in the basal ganglia or the internal capsule in 68% of cases, as also found by Haderschöe et al. Small infarcts are probably easier to recognize when they are subcortically located because small infarcts in the cortical regions could be difficult to distinguish from atrophy. We believe, however, that this is only a minor bias in the general picture of silent infarcts being small and subcortical. We compared the CT characteristics of symptomatic infarcts in patients with and in patients without silent infarction and found that the symptomatic infarcts were significantly smaller (mean, 29.5 mm versus 38.1 mm) and more often subcortically located (72% versus 52%) in patients with than in those without silent infarction. This might suggest that infarcts in patients with and in patients without silent stroke have a different pathophysiology.

**Risk Factors**

Contradictory findings exist concerning risk factors for silent infarction. In the study of Chodosh et al,1 risk factors for stroke and silent infarction were not found to be different. Only crude rates were compared. In contrast, Haderschöe et al3 found age, hypertension, and current cigarette smoking to be independent risk factors for silent infarction in a group of patients with TIA and minor stroke included in a secondary prevention trial. Diabetes, cardiovascular disease, hematocrit, and blood fibrinogen were not found to be independent risk factors. AF was not investigated because patients with AF were excluded. In patients with exclusively minor stroke (n=507), only current cigarette smoking remained significantly related to the occurrence of silent infarction. In the Framingham Study,4 glucose intolerance was more common in stroke patients with silent infarction but not hypertension, cigarette smoking, cardiovascular disease, and AF. However, a multivariate analysis was not performed, and numbers were small. Ricci et al4 found hypertension, male sex, and ischemic changes on ECG to be independent risk factors. Norris and Chu5 reported in a retrospective study of selected patients that silent infarction was more frequent in patients with both TIA and carotid stenosis than in patients with either TIA or carotid stenosis. The influence of other risk factors was, however, not eliminated. A high frequency of silent infarction has been reported in patients with AF. However, these studies were retrospective and included few cases, and the influence of other risk factors was not eliminated. In a recent Danish community-based study of 340 stroke patients, AF was not found to be a risk factor for silent infarction. In the present study age, male sex, hypertension, and a history of intermittent claudication proved to be independent risk factors for silent infarction, although male sex and intermittent claudication did not show significance in the univariate analysis. Interaction between age and sex explains the different findings for male sex in the two analyses, because women were older than men in the study. In the univariate analysis this age difference concealed a true higher incidence for silent infarction in men. The multivariate model does not, however, readily explain the interaction between claudication and covariates. Diabetes just failed to reach statistical significance (OR, 1.70; CI, 0.89 to 3.26) as an independent risk factor for silent infarction.

In contrast, AF, daily alcohol consumption, a history of former MI, and TIA had no independent influence on the risk of silent infarction in stroke patients; they tended to reduce the risk of silent infarction, but this did not reach statistical significance. Consequently, it seems probable that risk factors are shared for stroke and silent infarction, but some risk factors such as age, male sex, hypertension, and intermittent claudication are more likely to be associated with silent infarction than with other risk factors such as AF. This theory is supported by Shimada et al,20 who in a magnetic resonance study of healthy individuals found silent lacunar infarction to be more frequent in hypertensive than in normotensive individuals. Thus, a possible difference in stroke pathophysiology between stroke patients with and without silent infarction might be explained by this difference in the distribution of risk factors.

**Clinical Significance of Silent Infarction**

Knowledge of the influence of silent infarction on the outcome of stroke is very limited. In the study by Chodosh et al,1 case-fatality rate was reported similar for stroke patients with and without silent infarction. Ricci et al4 compared outcome measured as death or persistent functional deficits and found no difference between patients with and without silent infarction. These outcome measures might not recognize smaller differences in outcome, however, and if silent infarction does influence the outcome of stroke, only small changes should be expected. In the present study we found that need for prestroke home care and mental abilities on admission were similar in stroke patients with and without silent infarction. The presence of silent infarction did not influence any outcome measure. No differences were found in BI score on admission or at discharge, recovery in ADL and neurological scores, time needed for recovery, length of hospital stay, mortality rate, or rate of discharge to a nursing home. In view of the fact that stroke in itself9 and even the presence of stroke risk factors in stroke-free volunteers5,6 affect cognitive and intellectual abilities, it is surprising that silent infarction did not affect outcome. Two possible explanations could account for this: either silent infarcts are indeed silent in all aspects, or the different outcome measures we used, namely, the BI, SSS, and MMSE, were too insensitive to detect the impact of small impairments of brain function on stroke outcome. The last explanation seems in our view the most likely. The MMSE detects only larger neuropsychological deficits; the BI evaluates the ability to perform daily life activities such as eating, dressing, and walking; and the SSS measures only neurological deficits. None of these scales are designed to measure in detail functions specifically dependent on high intellectual performance. Further studies are therefore needed.
to determine the clinical impact of silent infarction in detail. However, for the patient, the patient’s relatives, and the clinician, the outcome measurements used in this study should be fully informative. Silent infarction does not worsen the prognosis for the recovery in ADL and neurological functions and does not retard recovery from stroke.

Acknowledgments
This study was supported by grants from the Danish Health Foundation, the Danish Heart Foundation, Ebba Celinders Foundation, and the Gangsted Foundation.

References
H S Jørgensen, H Nakayama, H O Raaschou, J Gam and T S Olsen

Stroke. 1994;25:97-104
doi: 10.1161/01.STR.25.1.97

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/25/1/97