Carotid Artery and Heart Disease in Subtypes of Cerebral Infarction

Arne Lindgren, MD; Anders Roijer, MD; Bo Norrving, MD, PhD; Lena Wallin, MD; Jan Eskilsson, MD, PhD; Barbro B. Johansson, MD, PhD

Background and Purpose The aim of the study was to determine the prevalences of carotid artery disease and major and minor potential cardioembolic sources (1) in patients with cerebral infarction and age-matched control subjects and (2) in different clinical subtypes of cerebral infarction.

Methods A series of 166 consecutive patients with cerebral infarction and 59 control subjects was examined. The study protocol included clinical subtyping of the cerebral infarctions, ultrasonography of the carotid arteries, transthoracic echocardiography (TTE), transesophageal echocardiography (TEE), ECG, and examination of the brain with computed tomography, magnetic resonance imaging, or autopsy.

Results Carotid artery stenosis ≥80% or occlusion was present in 35 (21%) patients but in no control subjects (P<.001; χ² test). A major potential cardioembolic source was detected in 65 (39%) patients and 3 (5%) control subjects. Atrial fibrillation was present in 35 (21%) patients and 3 (5%) control subjects at initial ECG (P<.01) and in 47 (28%) patients at repeat examination; 17 patients had paroxysmal atrial fibrillation. Sinus rhythm and a major potential cardioembolic source were detected in 18 (11%) patients but in no control subjects (P<.01) at TTE (all patients and control subjects examined) or TEE (118 patients and 52 control subjects examined). The frequency of a minor potential cardioembolic source detectable at TTE or TEE was similar in the patient and control groups (51% and 53%, respectively [NS]) and increased significantly with age. A finding of carotid artery stenosis ≥80% or occlusion, atrial fibrillation, or a major cardioembolic source detected at TTE or TEE was more frequent among patients with cortical symptoms from anterior or middle cerebral artery territories than among those with lacunar syndromes (66% versus 22%, respectively). The probable source of cerebral infarction was identified in most of the 166 patients: cardiac embolism in 28% of cases (n=46), carotid artery disease in 8% (n=14), both cardiac embolism and carotid artery disease in 7% (n=11), and lacunar infarction in 23% (n=38). In 37 (34%) of the patients no unequivocal cause of the cerebral infarction was found.

Conclusions The prevalences of carotid artery and heart disease differ significantly between clinical subtypes of cerebral infarction. The cause of cerebral infarction remains uncertain in one third of patients. Because a minor potential cardioembolic source occurs in about 50% of both patients and control subjects, this finding is of questionable value as a risk factor for stroke in the elderly. (Stroke. 1994;25:2356-2362.)

Key Words: • carotid artery disease • cerebral infarction • echocardiography • stroke classification

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creased level of consciousness (n=2), severe concomitant disease (eg, psychiatric disorders, cardiogenic shock, etc; n=4), or refusal to undergo complete examination (n=10).

The patients were compared with a control group consisting of 59 age- and sex-matched subjects from a nonhospitalized control series without stroke or transient ischemic attacks, randomly selected from the population register of the same catchment area as the stroke patients. All patients and control subjects were examined with CT or MRI of the brain, sonography of the carotid arteries, and echocardiography. Details of recruitment of the control series have been reported earlier. The study was approved by the ethics committee of the University of Lund, and informed consent was obtained from all participants (or relatives in the case of patients unable to communicate).

Clinical Evaluation

Based on clinical features only (for details, see Reference 11) and with knowledge of findings at brain imaging, echocardiography, and sonography of carotid arteries, each of the 166 patients was assigned to one of four clinical subgroups according to the OCSP criteria: (1) total anterior circulation infarcts (TACI), both cortical and subcortical symptoms from anterior and middle cerebral arterial territory; (2) partial anterior circulation infarcts (PACI), more restricted and predominately cortical symptoms from the same arterial territories; (3) lacunar infarcts (LACI), lacunar syndromes in anterior, middle, or posterior cerebral or vertebrobasilar arterial territories, including sensorimotor lacunar syndrome; and (4) posterior circulation infarcts (POCI), vertebrobasilar or posterior cerebral arterial symptoms.

Hypertension, diabetes mellitus, and heart disease were considered to be present if the subjects were receiving medical treatment for these diseases at the time of investigation. Heart disease was also considered to be present if the subjects had previously received medical or surgical treatment for heart disease.

Heart Examinations

All subjects underwent 12-lead ECG. For patients with cerebral infarction, the study protocol included daily ECG recordings on days 0 through 2 after stroke onset when possible.

Ultrasonography of the heart was performed with Hewlett-Packard Sonos 500 equipment. All patients and control subjects underwent TEE with a 2.5- or 3.5-MHz transducer. One hundred eighteen patients and 52 control subjects underwent TEE with a 5-MHz single-plane transducer. Echocardiography findings were categorized as representing major or minor PCSs according to criteria similar to those described by Hart. Major PCSs included atrial fibrillation (AF), prosthetic valve, mitral stenosis, recent myocardial infarction, left atrial or ventricular thrombus, atrial myxoma, infective or marantic endocarditis, and dilated cardiomyopathy (ejection fraction < 35%). Minor sources included mitral valve prolapse, severe mitral annulus calcification, patent foramen ovale (diagnosed with color Doppler contrast echocardiography with intravenous administration of agitated sodium chloride), atrial septal aneurysm (defined as bulging ≥ 15 mm with a base of ≥ 15 mm of the interatrial septum into the left or right atrium or both atria), calcific aortic stenosis and slight dysfunction of the left ventricle (ejection fraction, 35% to 50%). The same categories of major and minor PCSs were used at TTE and TEE, although some of the abnormalities are detectable with one of the methods only. Because TEE was performed in addition to TTE, the TEE findings are presented together with the TTE findings. Spontaneous echo contrast in the left atrium is often related to embolism and AF and was registered separately. We also noted the presence of any large (≥ 5 mm) protruding aortic plaques. AF was not included among the ultrasonographic findings because it is normally diagnosed with ECG.

Carotid Artery Examination

All control subjects and 165 patients underwent ultrasonography of the carotid arteries performed with SC 6100 continuous-wave equipment (Carolina Medical Electronics Inc), using a 5-MHz probe. Detection criteria for 50% to 99% stenosis have been reported earlier. A Doppler shift frequency of 8.0 kHz or more over the common or internal carotid artery was considered to represent stenosis of 80% or more. Absence of any Doppler signal from the internal carotid artery was considered to represent occlusion.

All control subjects and 137 patients underwent ultrasonography of the carotid arteries performed with the duplex technique using Diasonic CV400 or Acuson 128XP equipment. The criteria for 50% to 99% stenosis and for occlusion have been reported earlier. Stenosis of 80% to 99% was considered present if two or more of the following criteria were met: (1) peak flow velocity > 4.0 m/s at the stenosis; (2) ratio of peak flow velocities in internal and common carotid arteries > 3.4; (3) Doppler shift frequency > 9.1 kHz at peak flow, with a pulsed Doppler frequency of 3 MHz; (4) internal or common carotid artery diameter reduction of 80% or more, as measured on the real-time image; (5) internal or common carotid artery cross-sectional area reduction of 96% or more, as measured on the real-time image; and (6) diastolic flow velocity > 1.4 m/s at the stenosis. Carotid stenosis of 50% to 99% or 80% to 99%, or occlusion, was considered to be present if the respective criteria were fulfilled at either of the ultrasound examinations.

Probable Cause of Cerebral Infarction

On the basis of clinical evaluation, we assessed the probable cause of cerebral infarction in each patient and the presence of carotid artery and heart disease. In patients with TACI or PACI, the presence of any of the following was considered the probable cause of stroke: carotid stenosis of 80% to 99%, or occlusion on the symptomatic side, or a major PCS at ECG, TTE, or TEE. In patients with the LACI syndrome, lacunar infarction was considered the probable cause only in the absence of both (1) carotid artery stenosis of 80% to 99% or occlusion on the symptomatic side and (2) a major PCS. In patients with POCI, only a major PCS was considered the probable cause of stroke.

Statistics

Means and 95% confidence intervals were calculated where appropriate. Differences among clinical subgroups of stroke were assessed with the χ² test for nominal scale variables but with one-factor ANOVA for continuous variables. The unpaired two-tailed Student's t test was used to compare age in groups with versus those without carotid artery disease and heart disease. A value of P < .05 was considered significant. Stepwise logistic regression analysis (forward logistic regression, using the SPSS package) was performed to elicit possible correlations between carotid artery stenosis, AF at ECG, findings at echocardiography, clinical stroke subgroups, age, hypertension, history of heart disease, and smoking (previous smokers who had quit were classified as nonsmokers). Advice on statistical analysis was given by Eva Kelty of Clinical Data Care AB.

Results

Cerebral Infarction Versus Control Subjects

One hundred sixty-six patients (mean age, 73 years; range, 38 to 103 years; 92 men) with first-ever stroke caused by cerebral infarction and 59 control subjects (mean age, 72 years; range, 51 to 95 years; 30 men) were included in this study. Of the 166 patients, 131 were 65 years or older. Demographic data and some cardiovascular risk factors are shown in Table 1. Known heart
The prevalence of carotid artery stenosis was used: carotid artery stenosis of 50% to 99% or occlusion was seen in 35 (21%) patients versus none of the control subjects (95% CI, 14% to 28%) versus none of the control subjects (5%) control subjects (P<.01). In follow-up examinations, AF was detected in 12 other patients with sinus rhythm at initial ECG, whereas sinus rhythm was detected in 5 of the patients with AF at initial ECG. Thus, AF was present in a total of 47 patients (28%), of whom 17 had paroxysmal AF. The prevalence of AF at initial ECG increased with age: the mean age of subjects without AF was 71 years and of those with AF, 81 years (P=.0001, t test). Of the 44 patients with AF examined with ECG before stroke onset, 34 (77%) already had AF at that time.

Sinus rhythm and a major PCS were detected at TTE or TEE in 11% (18/166) of the patients but in no control subjects (P<.01, χ² test). The frequency of a major PCS other than AF (shown in Table 2 for patients and control subjects) did not increase with age. The most frequent major PCSs were AF (47 patients, 3 control subjects), prostatic heart valve (2 patients, no control subjects), left atrium or ventricle thrombus (13 patients, no control subjects), and dilated cardiomyopathy (12 patients, 1 control subject). A detailed account of major and minor PCSs found at echocardiography will be reported separately (A.R., unpublished data, 1994).

A minor PCS was a frequent finding, both in control subjects (47% at TTE, 53% at TTE and TEE) and in patients (43% at TTE, 51% at TTE and TEE) (ie, after exclusion of those with a major PCS at echocardiography); the difference between the patients and control subjects was nonsignificant. Even when subjects with AF were also excluded, patients and control subjects did not differ in the frequency of minor PCSs. In contrast to that of major PCSs (other than AF), the overall frequency of minor PCSs increased with age, with the mean age being 77 years among subjects (patients and control subjects) with a minor PCS and 70 years among subjects (patients and control subjects) without a minor PCS (P<.01). Age and the frequency of minor PCSs were

### Table 1. Demographic Data and Selected Cardiovascular Risk Factors

<table>
<thead>
<tr>
<th></th>
<th>Infarction (n=166)</th>
<th>Control Subjects (n=59)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, y</td>
<td>73±3</td>
<td>71±6</td>
<td>NS*</td>
</tr>
<tr>
<td>Men/women, n</td>
<td>92/74</td>
<td>30/29</td>
<td>NS†</td>
</tr>
<tr>
<td>Hypertension‡</td>
<td>44 (27)</td>
<td>12 (20)</td>
<td>NS†</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>29 (17)</td>
<td>1 (2)</td>
<td>.003†</td>
</tr>
<tr>
<td>Heart disease</td>
<td>81 (49)</td>
<td>12 (20)</td>
<td>.001†</td>
</tr>
<tr>
<td>Current smoking</td>
<td>33 (20)</td>
<td>11 (19)</td>
<td>NS†</td>
</tr>
</tbody>
</table>

Numbers in parentheses indicate percentage.

* t test.
† χ² test.
‡ Hypertension defined as receiving medical treatment for hypertension.

### Table 2. Findings at ECG and Echocardiography in Patients With Cerebral Infarction and Control Subjects

<table>
<thead>
<tr>
<th>Finding</th>
<th>Infarction (n=166)</th>
<th>Control Subjects (n=59)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major PCS at TTE or AF</td>
<td>55</td>
<td>3</td>
<td>.0001</td>
</tr>
<tr>
<td>TTE</td>
<td>14 §</td>
<td>8 (4-13)</td>
<td></td>
</tr>
<tr>
<td>Minor PCS†</td>
<td>65</td>
<td>43 (34-51)</td>
<td></td>
</tr>
<tr>
<td>TEE and TTE combined</td>
<td>25 §</td>
<td>15 (9-21)</td>
<td>.006</td>
</tr>
<tr>
<td>Minor PCS†</td>
<td>72</td>
<td>51 (42-60)</td>
<td></td>
</tr>
</tbody>
</table>

P indicates probability value (χ² test) for difference between patients and control subjects; CI, confidence interval; PCS, potential cardioembolic source; TTE, transthoracic echocardiography; TEE, transesophageal echocardiography; and AF, atrial fibrillation at initial ECG.

* Other than AF.
† Patients with major PCSs (other than AF) excluded.
‡ TEE performed in 118 patients and in 52 control subjects.
§ Two subjects also had AF.
 ¶ Five subjects also had AF.
correlated, both among patients and among control subjects. With TEE findings included, the mean age of subjects (patients and control subjects) with a minor PCS was 75 years, and that of those without was 70 years (P<.001; t test). The most frequent minor PCSs were severe mitral annulus calcification (49 patients, 16 control subjects), patent foramen ovale (20 patients, 15 control subjects), atrial septal aneurysm (24 patients, 9 control subjects), and calcific aortic stenosis (5 patients, 1 control subject).

Spontaneous echo contrast was seen in 19 subjects (16 patients and 3 control subjects), all but one of whom had AF. Large (≥5 mm) plaques in the aortic arch were seen at TEE in 8 patients and in 3 control subjects.

Subtypes of Cerebral Infarction

Mean age was significantly higher in the TACI subgroup (79.6 years) than in the other subgroups (P=.0001; ANOVA). The prevalence of male sex, hypertension, diabetes mellitus, history of heart disease, and current smoking did not differ significantly among subgroups (P=.0001; ANOVA). The most frequent minor PCSs were severe mitral annulus calcification (49 patients, 16 control subjects), patent foramen ovale (20 patients, 15 control subjects), atrial septal aneurysm (24 patients, 9 control subjects), and calcific aortic stenosis (5 patients, 1 control subject).

Discussion

To the best of our knowledge, this is the first population-based study in which consecutive patients with cerebral infarction have been examined with brain imaging, TTE, TEE, and transesophageal echocardiography; and AF, atrial fibrillation at initial or subsequent ECG. Numbers in parentheses indicate percentage.

Prognosis

Based on the combined results of clinical classification, ECG, carotid artery sonography, and echocardiography, the probable cause of cerebral infarction was identified in 66% (109/166) of the series as a whole (Table 4): cardiac embolism in 28% of patients (n=46), carotid artery disease in 8% (n=14), both cardiac embolism and carotid artery disease in 7% (n=11), and lacunar infarction in 23% (n=38). The proportion of probable identifications differed among subgroups (TACI, 80%; PACI, 54%; LACI, 78%; POCI, 38%). In 57 (34%) of the patients no unequivocal cause of the cerebral infarction was found.

Table 3. Findings at Echocardiography in Subgroups of Cerebral Infarction

<table>
<thead>
<tr>
<th>Finding</th>
<th>TACI (n=44)</th>
<th>PACI (n=52)</th>
<th>LACI (n=49)</th>
<th>POCI (n=21)</th>
<th>All (n=166)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major PCS at TTE/TEE or AF</td>
<td>25 (57)</td>
<td>24 (46)</td>
<td>8 (16)</td>
<td>8 (38)</td>
<td>65 (39)</td>
<td>.0005</td>
</tr>
<tr>
<td>TTE</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major PCS*</td>
<td>4 (9)</td>
<td>4 (8)</td>
<td>1 (2)</td>
<td>5 (24)</td>
<td>14 (8)†</td>
<td>.03</td>
</tr>
<tr>
<td>Minor PCS†</td>
<td>17 (42)</td>
<td>22 (46)</td>
<td>19 (40)</td>
<td>7 (44)</td>
<td>65 (43)</td>
<td>NS</td>
</tr>
<tr>
<td>TEE§ and TTE combined</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major PCS*</td>
<td>6 (14)</td>
<td>9 (17)</td>
<td>5 (10)</td>
<td>5 (24)</td>
<td>25 (15)†</td>
<td>NS</td>
</tr>
<tr>
<td>Minor PCS†</td>
<td>16 (42)</td>
<td>24 (56)</td>
<td>24 (55)</td>
<td>8 (50)</td>
<td>72 (51)</td>
<td>NS</td>
</tr>
</tbody>
</table>

TACI indicates total anterior circulation infarction; PACI, partial anterior circulation infarction; LACI, lacunar infarction; POCI, posterior circulation infarction; P, probability value for χ² test for difference between patients and control subjects; PCS, potential cardioembolic source; TTE, transthoracic echocardiography; TEE, transesophageal echocardiography; and AF, atrial fibrillation at initial or subsequent ECG. Numbers in parentheses indicate percentage.

*Other than AF.
†Patients with major PCS (other than AF) excluded.
‡Four also had AF.
§TEE performed in 118 patients.
||Seven also had AF.
level of consciousness, severe concomitant systemic disease, death soon after admission, or technical reasons. The exclusion of patients may affect the implications of our results with regard to the ischemic stroke population in general. However, we found the prevalences of carotid artery disease and potential major (but not minor) cardiac embolic sources to differ significantly, both between patients and control subjects and between clinical subtypes of cerebral infarction. The proportion of stroke patients aged 65 years or more (79%) is in accord with earlier figures for the same catchment area.14

There were more patients than control subjects with hypertension (27% versus 20%), although the difference was not significant. Because blood pressure often increases in the acute phase after stroke, we only registered patients already receiving medical treatment for hypertension as hypertensive. Probably several other patients had hypertension that had not been diagnosed or medically treated.

Carotid Artery Disease

Our finding that carotid artery disease is more common in patients with cerebral infarction than in control subjects is in accord with earlier reports.1 We could not confirm the relation between carotid artery disease and age reported earlier,1,15,16 perhaps because of the small number of younger subjects in our study. In stepwise logistic regression analysis (with age included), there was an independent correlation between carotid artery stenosis of 80% to 99% or occlusion and clinical subtype of cerebral infarction. Carotid artery disease was most frequent in patients with TACI, a finding consistent with the view that this clinical syndrome is often caused by atherosclerosis of large extracranial arteries.

Atrial Fibrillation

In the OCSP,17 AF was present in 18% of patients with cerebral infarction and was most common in patients with TACI and PACI, which is in accord with our findings. We found AF in 21% of the patients at initial ECG and in 28% at repeat ECG. In our study, 10 (23%) of 44 stroke patients with AF were found to have had sinus rhythms at ECG performed before stroke onset. It has recently been suggested that AF may sometimes be the consequence and not the cause of stroke.18

In our study, paroxysmal AF was present in no less than 17 (36%) of the 47 patients with AF. It has been suggested that paroxysmal AF is associated with a lower risk of embolization than chronic AF.19 However, in a recent study of patients with AF, paroxysmal AF was not an independent predictor of increased or decreased risk of thromboembolism.20 It is noteworthy that in our study 12 subjects with sinus rhythm at the first ECG examination after stroke onset developed AF later on. It is suggested that ECG should be repeated in the first days after stroke in patients with no clear cause of their cerebral infarction. In patients with paroxysmal AF, increased risk of embolism has been related to high age, hypertension, and increased left atrial diameter.21

Minor Potential Cardiac Embolic Sources

The detection of minor PCSs has increased with the introduction of TEE.2,7 We found the prevalence of minor PCSs to increase with age in both control subjects and patients with stroke. Similar results were recently reported from a stroke population study.7 Even though minor PCSs are often found in patients with stroke, it is also a common finding in control subjects and was seen in about 50% of both patients and control subjects in our study. Therefore, the finding of a minor PCS should be used with caution—if at all—as an explanation of ischemic stroke in elderly patients. The situation in younger stroke patients may be different.5-7

Different Risk Factors in Clinical Subgroups of Stroke

As earlier reported by us,11 the OCSP clinical subgroups of cerebral infarction3 manifest significant correlation to several features in CT and MRI imaging of the brain, such as mean lesion volume, and to the proportion of patients with cortical or posterior circulation territory involvement. The present results suggest that the OCSP subgroups also differ as to findings of ultrasonography of the heart and carotid arteries. It is therefore probable that the clinical subgroups represent different pathogenetic mechanisms and thus can be used for predicting prognosis, selecting patients for specific treatments, and selecting patients who will most probably benefit from such ancillary investigations as

### Table 4. Distribution of Carotid Artery Disease and Major Cardioembolic Sources in Stroke Subgroups and Overall, Compared With Control Subjects

<table>
<thead>
<tr>
<th>Source</th>
<th>TACI (n=44)</th>
<th>PACI (n=52)</th>
<th>LACI (n=49)</th>
<th>POCI (n=21)</th>
<th>All (n=166)</th>
<th>Control Subjects (n=59)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid artery disease ≥80%*</td>
<td>10 (23)</td>
<td>4 (8)</td>
<td>3 (6)</td>
<td>0 (0)</td>
<td>17 (10)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Major cardiac embolic source†</td>
<td>18 (41)</td>
<td>20 (38)</td>
<td>7 (14)</td>
<td>7 (33)</td>
<td>52 (31)</td>
<td>3 (5)</td>
</tr>
<tr>
<td>Both</td>
<td>7 (16)</td>
<td>4 (8)</td>
<td>1 (2)</td>
<td>1 (5)</td>
<td>13 (8)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Neither</td>
<td>9 (20)</td>
<td>24 (46)</td>
<td>38 (78)</td>
<td>13 (62)</td>
<td>84 (51)</td>
<td>56 (95)</td>
</tr>
</tbody>
</table>

TACI indicates total anterior circulation infarction; PACI, partial anterior circulation infarction; LACI, lacunar infarction; and POCI, posterior circulation infarction. Numbers in bold type (10+4+18+20+7+7+4+1+38=109 [66% of patients]) indicate patients with a probable cause of cerebral infarction. In the remaining 57 patients, the cause of cerebral infarction was uncertain. Numbers in parentheses indicate percentage.

*Or occlusion at ultrasonography of carotid arteries. Only the symptomatic side is included (except for POCI and control subjects for whom both sides are included).

†At echocardiography. Patients with atrial fibrillation at ECG are also included.
CT or MRI of the brain and examination of heart and carotid arteries.

Our results support the assumption that embolism is a common cause of cerebral infarction in patients with TACI, as 35 (80%; 95% CI, 67% to 92%) of 44 patients with TACI had carotid artery stenosis of 80% to 99% or occlusion, a major PCS, or both. In patients with PACI, sometimes proposed to be caused by the same mechanisms as TACI, the corresponding figure was 28 (54%; 95% CI, 40% to 68%) of 52 patients. Thus, the cause of cerebral infarction more often remains uncertain in patients with PACI (46%) than in patients with TACI (20%).

In the TACI and PACI groups, there were 34 patients with carotid artery stenosis of 50% to 99% or occlusion on the symptomatic side. Of these patients, only 3 (15%) with TACI had stenosis of 50% to 79% compared with 6 (43%) of those with PACI. Although based on small samples, the proportion of patients with carotid artery stenosis of 50% to 79% seems to be higher in PACI than in TACI groups, which may suggest that severe carotid artery stenosis is more often associated with severe neurological deficits.

Several pathogenetic mechanisms have been suggested to cause subcortical infarcts. Patients with LACI may have small subcortical infarcts due to penetrating artery disease. Cardiac embolism has been reported to be less frequent in patients with lacunar infarction than in patients with other types of cerebral infarction, which is consistent with our finding that 38 (78%) patients with LACI had neither carotid artery stenosis of 80% to 99% or occlusion nor a major PCS. However, an embolic etiology of lacunar infarction cannot be excluded, and a potential cardiac source of embolism, severe stenosis, or occlusion of the carotid artery has been reported in more than one quarter of patients with an infarction limited to the territory of deep perforators. Patients with subcortical infarcts may have cortical signs (e.g., dysphasia in patients with the PACI syndrome). Therefore, a separate, detailed comparison between patients with PACI and LACI needs to be performed.

Stroke of Uncertain Cause

The cause of cerebral infarction remained uncertain in no less than one third (34%) of patients, a finding in accord with earlier results. There are several possible reasons why the cause of stroke could not be determined in so many patients in our study. (1) Twenty-nine percent of the patients were not examined with TEE. (2) The relation between ischemic stroke and atheromatous plaques in the aortic arch detected at TEE has been reported previously. The importance of the aortic arch as a potential embolic source could not be precisely determined in our study because a single-plane transducer was used for TEE. With a biplane or multiplane TEE transducer, more plaques in the aorta would probably have been detected. In a recent autopsy study, the prevalence of ulcerated plaques in the aortic arch was no less than 61% among patients with cerebral infarction of undetermined cause. Some patients with LACI manifested signs of heart or carotid artery disease, perhaps indicating generalized atherosclerosis and thus making the diagnosis of lacunar infarction uncertain. (4) The vertebral and basilar arteries were not examined with ultrasonography, and thus the contribution of large-vessel atherosclerosis to PCSI was not determined.

Conclusions

Carotid artery disease and major PCSs differ in prevalence between the clinical OCSP subgroups of cerebral infarction. This classification may therefore be useful clinically to assess possible pathophysiological mechanisms in patients with ischemic stroke. In one third of patients with cerebral infarction, the cause of stroke remains uncertain. Both among patients and control subjects, the proportion of those with a minor PCS increased with age. It is questionable whether a minor PCS should be regarded as a risk factor for stroke in the elderly.

Acknowledgments

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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/25/12/2356