Prevalence of Extracranial Carotid Artery Disease Detectable by Echo-Doppler in an Elderly Population

A. Pujia, MD; P. Rubba, MD; and M.P. Spencer, MD

Background and Purpose: Little information is available on extracranial carotid artery disease in free-living elderly individuals. We sought to evaluate the prevalence of carotid lesions in the elderly.

Methods: Using echo-Doppler, we assessed the prevalence of possible atherosclerotic lesions in the internal carotid arteries (n=478) and the external and common carotid arteries (n=956) of 239 subjects 65–94 years of age living in retirement homes in Seattle, Wash.

Results: We found that 152 (31.8%) internal carotid arteries were affected by nonstenosing plaque and 37 arteries (7.7%) had stenosis or occlusion. In addition, 193 (20.2%) external or common carotid arteries showed nonstenosing plaques. There were 128 subjects (53.6%) with internal carotid disease, 106 (44.3%) with evidence of external or common carotid disease, and 75 (31.4%) affected by disease in all three sites. There were 80 subjects (33.5%) with no ultrasound evidence of carotid disease. We found that the presence and severity of carotid disease increased between the decades 65–74 and 75–84. We also demonstrated a positive association between systolic blood pressure and ultrasound evidence of carotid disease that was independent of age.

Conclusions: The prevalence of extracranial artery disease in an apparently healthy population was high, although stenoses in most instances were not severe. We conclude that noninvasive ultrasound methods identify a relatively small fraction of individuals (5% of the total) at high risk for stroke or transient ischemic attack. Echo-Doppler might be used to monitor further disease progression and to evaluate the efficacy of different therapeutic or preventive interventions. (Stroke 1992;23:818–822)

KEY WORDS — aged • arteriosclerosis • carotid artery diseases • ultrasonics

Consistent evidence indicates that ultrasound evaluation of carotid disease is helpful for the prediction of subsequent cerebrovascular ischemia.1–3 Stroke is an important cause of invalidity or death in the elderly; recurrent cerebral ischemia might lead to multi-infarct dementia, another severely disabling condition of advanced age.

Most information on the prevalence of carotid abnormalities, as detected by ultrasound, refers to patient series, selected according to different criteria. In most instances these series included symptomatic vascular disease, with signs of coronary or peripheral ischemia4–6; an audible bruit over the neck in the absence of symptoms1,7–8; or high risk for atherosclerotic cardiovascular disease due to hypercholesterolemia,9,10 hypertension,1,12 or diabetes mellitus.13 Less information is available on unselected free-living individuals,14 especially if they are elderly.

We therefore conducted a survey, using noninvasive ultrasound methods, among elderly people (above age 65) living in retirement homes. We used echo-Doppler, which includes two different modalities of examination (namely, echo and Doppler) that can both be performed with a single means of instrumentation.15 Echo gives information on the arterial wall and is also sensitive to early lesions, including nonstenosing plaques. Doppler allows detection of flow disturbances with semiquantitative evaluation of the degree of stenosis. Both types of information were collected in this study.

Subjects and Methods

We conducted a study of 239 elderly people (57 male and 182 female) living in two retirement homes in Seattle. All subjects were over 65 years of age and were free living. All the 369 residents in retirement homes were invited to participate in our study during a preliminary meeting and with a letter. During the meeting, we explained the full procedures and the aims of our studies, and further explanation was given before the evaluation. A consent form was enclosed with the letter. The protocol and all procedures were approved by the Committee for Human Subjects Research of the Institute of Applied Physiology and Medicine of Seattle.

The full examination was performed after informed consent was obtained in 239 individuals (65% of those
invited). No subject who volunteered was excluded from the study.

A self-administered questionnaire was completed by the subjects to assess main cardiovascular risk factors such as hypertension, diabetes, hypercholesterolemia, smoking, family history of atherosclerotic accident, and any previous experience of cardiovascular disease symptoms. These included slurring of speech, numbness or tingling on one side of the body, monolateral visual disturbance, dizziness or loss of balance, weakness or paralysis in one arm or leg, confusion or temporary loss of memory; also included in this questionnaire was the Rose questionnaire to detect previous chest pain (myocardial infarction or angina pectoris) and intermittent claudication.16

Subjects with a history of serum cholesterol values above 200 mg/dl were considered to be hypercholesterolemic. The category of smokers included ex-smokers. Subjects with systolic pressure above 160 mm Hg or diastolic blood pressure above 95 mm Hg17 or those who were taking any antihypertensive medication were defined as hypertensive. Subjects with a history of diabetes or those who were taking any antidiabetic medication were considered diabetic. Subjects with parents or siblings who had had stroke, myocardial infarction, or who had died of sudden death before 50 years of age were considered to have a family history of cardiovascular disease. Those who had two or more symptoms of cardiovascular disease themselves were considered to have a history of cardiovascular disease, based on the criteria of a questionnaire for the investigation of cardiovascular symptoms.16 The other factors considered were age, gender, and obesity (defined as body mass index of greater than 29.9 kg/m²).18

The echo-Doppler evaluation was performed with an Advanced Technologies Laboratory Mark 600 duplex scanner using a probe at a frequency of 5 MHz for the pulsed Doppler and 7.5 MHz for the B-scan. The dynamic range was set at 50 dB. The echo-Doppler scanner combines a B-mode ultrasound imager with a pulsed Doppler and 7.5 MHz for the B-scan. The echo-Doppler scanning has a sensitivity of 80% for detecting flow reversal in the common carotid artery.

The carotid evaluation was performed in longitudinal and transverse planes with anterior, lateral, and posterior approaches. The low common carotid artery (CCA), the high CCA, the carotid bifurcation, the low internal carotid artery (ICA), the high ICA, and the external carotid artery (ECA) were studied in all subjects. The evaluations were recorded on videotape and later analyzed.

The diagnostic criteria for ICA stenosis (Table 1) were based on the interpretation of spectral analysis (peak systolic frequency and spectral broadening). B-mode imaging was used to detect the presence of abnormal echogenicity. The criteria for ICA disease were as follows: plaque, normal Doppler frequency (<4 kHz) and echogenicity along luminal wall or isolated bright spot; stenosis 1–49%, systolic frequency peak <4.0 kHz and spectral broadening; stenosis 50–99%, systolic frequency peak ≥4.0 kHz and spectral broadening; occlusion, no ICA signals and no ICA image. Abnormal echogenicity along the luminal wall of ECA or CCA was considered evidence of plaque.20–23

At the end of each day's examination, the videotapes were read and interpreted by a reader (the same for the entire study). We also recorded the peak maximum systolic velocity, the end-diastolic velocity, the spectral broadening, and the presence of possible plaques.

The statistical methods (χ² and nonpaired t test) were performed according to Snedecor and Cochran.24

Results

We found that 152 (31.8%) ICAs had nonstenosing plaque; 37 arteries (7.7%) had 1–99% stenosis or occlusion. In addition, we found 193 (20.2%) ECAs or CCAs with nonstenosing plaque. There were 128 subjects (53.6%) with ICA disease, 106 (44.3%) with evidence of ECA or CCA disease, and 75 (31.4%) affected by disease in all three sites. Eighty subjects (33.5%) had no ultrasound evidence of carotid artery disease.

Table 2 summarizes information on cardiovascular history and risk factors derived from a self-administered questionnaire that was completed by all the participants in the study. Approximately 20% of these elderly subjects had already experienced symptomatic cardiovascular disease. There was one smoker, and approximately 30% were ex-smokers. In 10% of the cases there was awareness of diabetes mellitus.

Figure 1 illustrates the distribution of plaques, as detected by high resolution echography, in the ICAs, CCAs, and ECAs of retired people. Prevalence of lesions was highest in the ICA and lowest in the ECA (p <0.01 versus CCA and ICA).

Table 1. Criteria for the Diagnosis of Internal Carotid Artery Stenoses Based on the Results of Doppler Spectrum Analysis

<table>
<thead>
<tr>
<th>Degree of stenosis</th>
<th>Spectrum</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–49%</td>
<td>Spectral broadening; systolic frequency peak &lt;4.0 kHz</td>
</tr>
<tr>
<td>50–99%</td>
<td>Spectral broadening; systolic frequency peak ≥4.0 kHz</td>
</tr>
<tr>
<td>Occlusion</td>
<td>No detectable signal; flow to zero reversed in the common carotid artery</td>
</tr>
</tbody>
</table>

Table 2. History and Clinical Data of 239 Elderly Subjects (57 Men and 182 Women) Living in Retirement Homes

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family history of cardiovascular disease</td>
<td>25</td>
<td>10</td>
</tr>
<tr>
<td>Myocardial infarction or angina</td>
<td>22</td>
<td>9</td>
</tr>
<tr>
<td>Stroke or transitory ischemic attack</td>
<td>29</td>
<td>12</td>
</tr>
<tr>
<td>Intermittent claudication</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Obesity (body mass index ≥30)</td>
<td>30</td>
<td>12</td>
</tr>
<tr>
<td>Known hypercholesterolemia</td>
<td>83</td>
<td>35</td>
</tr>
<tr>
<td>Known hypertension</td>
<td>91</td>
<td>38</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>73</td>
<td>30</td>
</tr>
<tr>
<td>Known diabetes mellitus</td>
<td>24</td>
<td>10</td>
</tr>
</tbody>
</table>
Figure 1. Graph of plaque distribution by arterial segment in elderly (65-94 years of age) subjects living in retirement homes in Seattle, Wash. ICA, internal carotid arteries; CCA, common carotid arteries; ECA, external carotid arteries.

Figure 2 shows the increase in the prevalence of plaques (at echo examination) and stenoses (according to Doppler criteria) with increasing subject age in the ICAs.

Increase in plaque prevalence (of approximately 60%) between 65-74 and 75-84 years of age was statistically significant (p<0.05), whereas there was a tendency to a plateau afterward (only a 15% increase in plaque prevalence between 75-84 and 85-94 years of age, which was not statistically significant).

The number of stenoses showed only a small rise with age, whereas the severity increased progressively: in particular, all flow-reducing lesions (lumen reduction ≥50%) were detected in the age range 75-94 years. We found flow-reducing stenoses in 11 individuals. One subject had experienced symptomatic brain ischemia (stroke). The other 10 were completely asymptomatic and did not report any previous attack of cerebral ischemia.

Table 3 demonstrates an age trend for plaques in the CCAs and ECAs similar to that observed in the ICAs, with an even steeper rise (by a factor of 3-4) between the age ranges 65-74 and 75-84 years (p<0.001). We then observed a tendency toward stabilization (however, for CCAs, the difference between the age ranges 75-84 and 85-94 years was statistically significant [p<0.05]).

Cardiovascular history or presence of risk factors (as evaluated by questionnaire) was not significantly related to the presence of carotid lesions as detected by ultrasound. However, systolic blood pressure (measured during the vascular examination) showed a positive association (p<0.05) with the presence of carotid lesions, which was independent of age (Table 4).

**Discussion**

This cross-sectional survey by noninvasive ultrasound methods in elderly people living in retirement homes...
has demonstrated a steep rise in the prevalence of arteriosclerotic plaques between ages 65−74 and 75−84 years, with a tendency to stabilization in the subsequent decade. No simple explanation is available for this finding. In an animal (Watanabe heritable hyperlipidemic rabbit) model of experimental atherosclerosis, intimal aortic lipid deposition increased linearly up to 57 months of age, whereas atheroma formation progressed up to 12 months of age and was markedly slowed down in older animals. It is thus unlikely, in Watanabe rabbits and possibly also in humans, that slow atherosclerosis progression in old age is due to less cholesterol deposition in the arterial wall.

In general, implementation of preventive efforts to retard arterial disease progression seems justified, especially in the first decade after retirement. After age 74, stenosis in some lesions was more severe (Figure 2), possibly as a result of thrombotic complications, which might lead to disturbed blood flow to the cerebral tissue. When this stage was reached, elderly people were at high risk for symptomatic ischemic attack (transient ischemic attack or stroke).

In the present study, 11 subjects with a flow-reducing stenosis, corresponding to 5% of the total group of individuals, were in this condition. Ten had never experienced any symptom of ischemia and were completely unaware that they were at high risk for vascular disease.

Numerous elderly subjects (over 50% of the total) had nonstenosing plaques, but without flow-reducing stenosis, in their carotid arteries. These patients should be reassured because, in the absence of symptoms of brain ischemia, no impending risk of cerebrovascular attack is associated with this finding. However, an intervention that was effective in slowing down the steep rise in the number of carotid plaques after age 65 would be expected to reduce the overall risk of stroke.

A more compelling problem is represented by the treatment of patients with flow-reducing stenoses. The well-known coronary risk factors do not seem to play a major role in the development of stroke. There are drawbacks to studying risk factors by questionnaire, but the conclusion of a limited role of hypercholesterolemia and cigarette smoking in relation to stroke is in agreement with other published reports, although for cigarette smoking, divergent data have also been published.

This survey confirms the relation between cerebrovascular disease and blood pressure consistently reported by others. In agreement with the view of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure, there is reason to think that elderly patients with an isolated systolic hypertension should have their blood pressure lowered cautiously toward a goal of 140−160 mm Hg.

Other candidate risk factors for stroke or transient ischemic attack in the elderly are high hematocrit and diabetes mellitus. By keeping the hematocrit low, optimizing diabetes control, and, possibly, using antithrombotic agents, the complication of preexisting plaques in flow-reducing stenoses or occlusion might possibly be prevented.

Screening for plaques and stenoses in the extracranial carotid arteries among elderly people living in retirement homes might be useful to identify those who are more likely to develop symptomatic cerebrovascular events in the following years. Noninvasive ultrasound methods might then be used to monitor disease progression and to evaluate the efficacy of different therapeutic or preventive interventions.

Acknowledgments

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References


Table 4. Blood Pressure in Elderly Subjects (n=239) With or Without Carotid Artery Disease (Plaque or Stenosis)

<table>
<thead>
<tr>
<th>Blood pressure (mm Hg)</th>
<th>Diastolic</th>
<th>Systolic</th>
</tr>
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<tbody>
<tr>
<td>No disease (n=80)</td>
<td>75±1</td>
<td>145±2</td>
</tr>
<tr>
<td>Carotid artery disease (n=159)</td>
<td>76±1</td>
<td>155±2*</td>
</tr>
<tr>
<td>After adjustment for age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No disease (n=80)</td>
<td>75±1</td>
<td>147±2</td>
</tr>
<tr>
<td>Carotid artery disease (n=159)</td>
<td>76±1</td>
<td>153±2*</td>
</tr>
</tbody>
</table>

*p<0.01, tp<0.05 by paired t test.
Values are mean±SEM.
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