Age and Sex Differences in the Distribution and Ultrasound Morphology of Carotid Atherosclerosis

The Tromsø Study

Oddmund Joakimsen, Kaare H. Bønaa, Eva Stensland-Bugge, Bjarne Koster Jacobsen

Abstract—Atherosclerosis begins early in life and is the major underlying cause of cardiovascular morbidity and death. Yet, population-based information on age and sex differences in the extent and morphology of atherosclerosis throughout life is scarce. Carotid atherosclerosis can be visualized with B-mode ultrasound and is a marker of atherosclerosis elsewhere in the circulation. We assessed both the prevalence and the morphology of carotid atherosclerosis by B-mode ultrasound in 3016 men and 3404 women, 25 to 84 years old, who participated in a population health survey. The participation rate was 88%. Plaque morphology was graded according to whether a plaque was predominantly soft (echolucent) or hard (echogenic). Atherosclerotic plaques were found in 55.4% of the men and 45.8% of the women. In men, there was a linear increase with age in the prevalence of carotid atherosclerosis, whereas in women, there was a curvilinear age trend, with an inflection in the prevalence rate of women at ~50 years of age. The male predominance in atherosclerosis declined after the age of 50 years, the plaque prevalence being similar in elderly men and women. Men had softer plaques than women; this sex difference in plaque morphology increased significantly (P=0.005) with age. The sex difference in the prevalence of atherosclerosis and the female age trend in atherosclerosis show significant changes at the age of ~50 years, suggesting an adverse effect of menopause on atherosclerosis. The higher proportion of soft plaques in men compared with women increases with age and may partly account for the prevailing male excess risk of coronary heart disease in the elderly despite a similar prevalence of atherosclerosis in elderly men and women. (Arterioscler Thromb Vasc Biol. 1999;19:3007-3013.)

Key Words: ultrasonography • carotid arteries • atherosclerosis • sex

High-resolution B-mode ultrasound is a valid and reproducible method to visualize and quantify carotid atherosclerosis noninvasively. Both ultrasound and autopsy studies have found that carotid atherosclerosis correlates well with atherosclerosis elsewhere in the circulation and can be used as a marker of general atherosclerosis.1–4 Previous population-based studies on ultrasound-detected atherosclerosis are small or did not examine sex differences in the prevalence and extent of atherosclerosis from young adulthood to old age.5–9

Not only the extent but also the morphology of atherosclerosis is important for the development of clinical vascular disease. Soft and lipid-rich plaques in the coronary arteries seem to be particularly prone to rupture and cause occlusive thrombosis and acute coronary syndromes,10 and ultrasound morphology of stenotic carotid plaque is an independent risk factor for stroke.11,12 Dark and low-echogenicity (echolucent) plaques on ultrasound correspond with soft and lipid-rich plaques at autopsy, supporting the validity of the ultrasound method.13–15 The reproducibility of carotid plaque morphology is acceptable.16 No studies on age and sex differences in ultrasound-assessed plaque morphology have been published.

In this population-based ultrasound study, we examined the prevalence and morphology of carotid atherosclerosis in 6420 men and women 25 to 84 years old.

Methods

Subjects

The Tromsø Study was started in 1974 and is a single-center study of inhabitants in the municipality of Tromsø, Norway. The aims of the study are to investigate, by means of epidemiological and clinical research, determinants of chronic diseases to assess etiological significance and to identify potentially modifiable determinants that may be developed into preventive or therapeutic strategies. The main focus is on cardiovascular diseases. The study design includes repeated population health surveys to which total birth cohorts and random samples of other age groups are invited. The fourth survey of the Tromsø Study started in September 1994 and was completed in October 1995. The survey was conducted by the University of Tromsø in cooperation with the National Health Screening Service and comprised 2 screening visits 4 to 12 weeks apart. All inhabitants >24 years old were invited to the first visit, and all subjects 55 to 74 years old and random 5% to 10% samples in the other 3-year age groups >24 years old were invited to both visits. A total of 6891 subjects, 88% of the eligible population, attended both visits. The protocol for the first visit was similar to the previous surveys in this population17 and included standardized measurements.
of height, weight, blood pressure, and nonfasting serum lipids. The second visit also included ultrasonographic examination of the right carotid artery. The study was approved by the regional ethical committee.

Cardiovascular Risk Factors

Height and weight were measured with the subject in light clothing without shoes; body mass index was calculated as weight divided by the square of height (kg/m²). The letter of invitation gave information about the survey and also included questions on previous myocardial infarction or stroke, prevalent angina pectoris or diabetes mellitus (all yes/no), treated hypertension (never/previously/currently), and smoking habits. The questionnaire was checked for logical inconsistencies.

Ultrasonography

High-resolution B-mode ultrasonography of the right carotid artery was performed on 6420 persons with an ultrasound scanner (Acuson Xp10 128 ART-upgraded) equipped with a linear-array 5-MHz color Doppler/pulsed-wave Doppler and 7-MHz B-mode transducer. The subjects were examined in the supine position with the head turned slightly to the left. The common, internal, and external carotid arteries were identified by combined B-mode and color Doppler/pulsed-wave Doppler ultrasound. We attempted to identify and record atherosclerotic plaques from 6 sites of the carotid artery: the near and far walls of the internal carotid artery as far upstream as technically possible; the bifurcation of the common carotid artery, ie, the distal part of the common carotid artery from the point at which the 2 parallel near and far walls start to deviate, up to the tip of the flow divider that separates the external carotid artery from the internal carotid artery; and the common carotid artery from the bifurcation segment and as far downstream of the supraclavicular region as technically possible. Frozen ultrasound images were stored on sVHS videotapes, and a 1-minute live recording of the carotid artery from different transducer positions and angles was also stored to document representative recordings of plaque thickness and morphology.

Instrument imaging adjustments (preprocession and postprocession, persistence, transmit zones, log compression, image depth, transmit power) were set at fixed values. The gain setting (including the depth gain compensation curve), however, was adjusted according to such factors as neck thickness, subcutaneous fat, and echogenicity of the near artery wall structures to obtain optimal visualization of arterial wall morphology. The gain setting was also changed continuously during the scanning procedure on the same individual to enhance plaque detection and characterization. The gain was not set so high that structural details of the high-echogenicity far-wall media-adventitia interface were concealed.

A plaque was defined as a localized protrusion of the vessel wall into the lumen. The maximum plaque thickness was measured online on frozen B-mode images marked with electronic calipers with measurement readout in tenths of a millimeter. The measures were recorded on videotapes and on written forms by the sonographers. In the far wall, the plaque thickness was defined as the distance between the lumen-plaque interface and the media-adventitia interface. Plaques in the near wall were measured from the far edge of the periadventitia-adventitia interface to the far edge of the intima-lumen interface. According to the protocol, plaques were to be visualized in the full diameter of the vessel, ie, both the proximal and the distal parts of the plaque should be “attached” to the typical double-lined intima-media structure, and the double lines should also be visible on the opposite wall of the vessel lumen. Focal calcification within the vessel wall (causing echo shadowing distally) without protrusion into the lumen was not considered to indicate atherosclerotic lesions.

Plaque morphology, in terms of ultrasound echogenicity, was graded from 1 to 4, where grade 1 denotes low echogenicity, or echolucency (defined as a plaque appearing black or almost black, like flowing blood), and grade 4 denotes strong echogenicity (defined as a plaque appearing white or almost white, similar to the far-wall highly echogenic media-adventitia interface) (Figure 1).
Agreement on classification of plaque echogenicity in the 2 categories used in the analysis was also substantial, with \( \kappa \) values (95% CI) of 0.80 (0.61 to 0.99) and 0.79 (0.61 to 0.97) between and within sonographers, respectively.16 A \( \kappa \) value of 0 means no agreement beyond chance, and a \( \kappa \) value of 1 means total agreement.

**Statistical Analysis**

Means were compared by 2-sample Student’s \( t \) test. Logistic regression was used to calculate age-adjusted odds ratios of having soft plaques according to sex and age. Descriptive statistics, \( t \) tests, and regression analyses were performed with the SAS software package.20 The probability values are 2-sided, and a value of \( P<0.05 \) was considered statistically significant.

**Results**

Selected characteristics of the 3016 men and 3404 women are presented in Table 1. Men were slightly younger than women and had a higher prevalence of carotid plaque (\( P<0.001 \)). Approximately 32% of both men and women were smoking cigarettes daily. The mean cholesterol level was relatively high, females having significantly higher levels than men (\( P<0.001 \)).

A total of 1670 men (55.4%) and 1558 women (45.8%) had carotid plaques. The prevalence of plaque increased with age in both men and women (Table 2). The age-adjusted odds ratios (95% CI) for plaque prevalence in men compared with women were 1.85 (1.24, 2.76), 1.65 (1.41, 1.91), 1.46 (1.24, 1.73), 0.76 (0.38, 1.55), and 1.56 (1.41, 1.74) among subjects <55 years old, 55 to 64 years, 65 to 74 years, >74 years, and for all subjects, respectively. The odds ratios did not change notably with control for age, body mass index, total cholesterol, HDL cholesterol, current smoking, treated hypertension, and diabetes mellitus: 1.77 (1.10, 2.84), 1.70 (1.44, 2.00), 1.65 (1.41, 1.91), 1.46 (1.24, 1.73), 0.76 (0.38, 1.55), and 1.56 (1.41, 1.74) among subjects <55 years old, 55 to 64 years, 65 to 74 years, >74 years, and for all subjects, respectively. The odds ratios did not change notably with control for age, body mass index, total cholesterol, HDL cholesterol, current smoking, treated hypertension, and diabetes mellitus: 1.77 (1.10, 2.84), 1.70 (1.44, 2.00), 1.65 (1.41, 1.91), 1.46 (1.24, 1.73), 0.76 (0.38, 1.55), and 1.56 (1.41, 1.74) among subjects <55 years old, 55 to 64 years, 65 to 74 years, >74 years, and for all subjects, respectively. The odds ratios did not change notably with control for age, body mass index, total cholesterol, HDL cholesterol, current smoking, treated hypertension, and diabetes mellitus: 1.77 (1.10, 2.84), 1.70 (1.44, 2.00), 1.65 (1.41, 1.91), 1.46 (1.24, 1.73), 0.76 (0.38, 1.55), and 1.56 (1.41, 1.74) among subjects <55 years old, 55 to 64 years, 65 to 74 years, >74 years, and for all subjects, respectively.

**Table 2. Plaque Prevalence, Mean Number of Plaques Among Subjects With Plaques, Mean Plaque Thickness, and Plaque Localization in the Carotid Artery by Age and Sex**

<table>
<thead>
<tr>
<th>Sex/Age, y</th>
<th>No. of Subjects Examined</th>
<th>Percentage (No.) With Plaque</th>
<th>Mean No. of Plaques*</th>
<th>Mean Plaque Thickness, mm†</th>
<th>Percentages of Subjects With Plaques in Different Locations of the Carotid Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Male</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25–34</td>
<td>99</td>
<td>3.0 (3)</td>
<td>1.0</td>
<td>1.1</td>
<td>0</td>
</tr>
<tr>
<td>35–44</td>
<td>114</td>
<td>14.9 (17)</td>
<td>1.2</td>
<td>1.6</td>
<td>0</td>
</tr>
<tr>
<td>45–54</td>
<td>150</td>
<td>32.0 (48)</td>
<td>1.3</td>
<td>1.9</td>
<td>9.3</td>
</tr>
<tr>
<td>55–64</td>
<td>1422</td>
<td>52.2 (742)</td>
<td>1.5</td>
<td>2.1</td>
<td>14.3</td>
</tr>
<tr>
<td>65–74</td>
<td>1146</td>
<td>69.4 (795)</td>
<td>1.9</td>
<td>2.3</td>
<td>26.0</td>
</tr>
<tr>
<td>75–84</td>
<td>85</td>
<td>76.5 (65)</td>
<td>2.1</td>
<td>2.5</td>
<td>25.9</td>
</tr>
<tr>
<td>Total</td>
<td>3016</td>
<td>55.4 (1670)</td>
<td>1.7</td>
<td>2.2</td>
<td>17.0</td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25–34</td>
<td>118</td>
<td>1.7 (2)</td>
<td>1.0</td>
<td>1.1</td>
<td>0</td>
</tr>
<tr>
<td>35–44</td>
<td>166</td>
<td>10.8 (18)</td>
<td>1.1</td>
<td>1.5</td>
<td>0.6</td>
</tr>
<tr>
<td>45–54</td>
<td>198</td>
<td>18.2 (36)</td>
<td>1.1</td>
<td>1.6</td>
<td>2.5</td>
</tr>
<tr>
<td>55–64</td>
<td>1453</td>
<td>40.3 (586)</td>
<td>1.3</td>
<td>1.9</td>
<td>9.2</td>
</tr>
<tr>
<td>65–74</td>
<td>1368</td>
<td>61.0 (834)</td>
<td>1.6</td>
<td>2.1</td>
<td>20.5</td>
</tr>
<tr>
<td>75–84</td>
<td>101</td>
<td>81.2 (82)</td>
<td>1.8</td>
<td>2.2</td>
<td>31.7</td>
</tr>
<tr>
<td>Total</td>
<td>3404</td>
<td>45.8 (1558)</td>
<td>1.5</td>
<td>2.0</td>
<td>13.3</td>
</tr>
</tbody>
</table>

FWI indicates far wall of internal carotid artery; NWI, near wall of internal carotid artery; FWB, far wall of the bifurcation part of the common carotid artery; NWB, near wall of the bifurcation part of the common carotid artery; FWC, far wall of common carotid artery; and NWC, near wall of common carotid artery.

*Among persons who had \( \geq 1 \) plaques in the carotid artery ( \( n = 1670 \) men and \( n = 1558 \) women).

†The mean value of the thickest plaque when \( >1 \) plaque was present.
2.00), 1.59 (1.32, 1.91), 0.87 (0.37, 1.22), and 1.66 (1.47, 1.86) among the same subject groups, respectively. In men, there was a nearly linear increase until the age of 65 years; thereafter, the age-related increase leveled off (Figure 2). Compared with men, women had a less steep increase in plaque prevalence between the ages of 35 and 49 years. After this age, atherosclerosis accelerated more rapidly in women than in men. In the age group of 75 to 84 years, more women (81.2%) than men (76.5%) had carotid atherosclerosis. The difference, however, was not statistically significant. The male-to-female ratio of plaque prevalence was highest in the age group of 45 to 49 years (Figure 3) and declined thereafter. Table 2 also shows that the number of atherosclerotic lesions and the thickness of plaques increased with age in both sexes. The predilection site of atherosclerosis seems to be in the bifurcation segment of the carotid artery, where the number of plaques is highest for both sexes at any age. Figure 4 shows that only 15% of the plaques were located entirely outside the bifurcation segment. There was no sex difference in the within-artery distribution of carotid atherosclerosis.

Soft carotid plaques were present in 37.7% of the 3100 subjects with morphologically classifiable plaques. In all age groups, there was a greater proportion of soft plaques in men than in women (Table 3). The proportion of soft plaques declined with age in both sexes, but more in women than in men; the odds ratio for soft plaques in men compared with women increased by age (Table 3). In a multiple logistic regression analysis with age and sex, there was a statistically significant age-by-sex interaction \((P=0.005)\). The prevalence of soft plaques increased with age until 60 years for both sexes (Figure 5). After this age, there was no further increase in prevalence of soft plaques for either sex. In all age groups, more men than women had soft plaques. This sex difference in soft plaque prevalence remained fairly constant from the age of 40 years and throughout old age (Figure 5). The odds ratios did not change notably with control for age, body mass index, total cholesterol, HDL cholesterol, current smoking, treated hypertension, and diabetes mellitus: 1.17 (0.50, 2.74), 1.00 (0.79, 1.26), 1.35 (1.07, 1.71), 2.26 (0.90, 5.65), and 1.14 (0.97, 1.34) among subjects <55 years old, 55 to 64 years, 65 to 74 years, >74 years, and for all subjects, respectively. The interaction between age and sex was now nonsignificant \((P=0.21)\). When the 153 women who had ever used postmenopausal hormone replacement therapy were excluded from the analysis, the risk estimates for plaque prevalence and plaque morphology did not change notably even after adjustment for cardiovascular risk factors (data not shown).

## Discussion

This population-based ultrasound study demonstrates a strong relationship between age and prevalence of carotid atherosclerosis in both sexes. Similar findings have been reported from other population-based studies (Table 4), but previous studies were small or did not include subjects throughout a broad age range.\(^5\)-\(^9\) Differences in study designs, including various ultrasound imaging methods, inclusion criteria, and definition of atherosclerosis, make direct comparisons between studies difficult. However, the prevalence of ultrasound-detected carotid atherosclerosis, with a few exceptions, seems to be fairly similar in studies from Europe and the United States (Table 4).

Atherosclerosis occurs more frequently in men than in women. The present study shows, however, that the sex gap in plaque prevalence is strongly influenced by age. The male-to-female ratio in prevalence peaks at age 45 to 49 years and then declines steadily (Figure 2). For subjects >75 years old, the total plaque prevalences in men and women do not differ. The curvilinear shape of the atherosclerosis prevalence curve for women suggests that the incidence of new athero-

![Figure 2](http://atvb.ahajournals.org/)

**Figure 2.** Prevalence of carotid atherosclerosis by age and sex. The vertical bars denote the 95% confidence intervals of proportions.

![Figure 3](http://atvb.ahajournals.org/)

**Figure 3.** Ratio of male-to-female prevalence of carotid atherosclerosis by age.

![Figure 4](http://atvb.ahajournals.org/)

**Figure 4.** Distribution of plaques among the 3228 participants who had ≥1 carotid plaques.
sclerotic plaques is lower for women than for men before the age at which the majority of women experience menopause and higher thereafter. Some of the declining prevalence of atherosclerosis with age among older male subjects may be due to a survival selection bias causing an overrepresentation of atherosclerosis-free male survivors.

The overall rate of attendance in our study was high (88%), but lower (≈70%) among the youngest (<35 years old) and oldest (>80 years old) participants. It is likely that this has resulted in a lower prevalence of plaques in the oldest age group, because one must expect subjects with the lowest burden of atherosclerosis-related disease to be overrepresented among the attenders (and survivors). The effect of the lower attendance rate is more unpredictable among younger participants but is probably marginal. The ultrasound examination was conducted without any clinical interview and was blinded with regard to symptoms of atherosclerosis. Some subjects, however, revealed symptoms and fragments of their medical history to the sonographers during the examination. We find it unlikely that this information has influenced our results with regard to plaque prevalence to any significant degree.

No previous large, population-based study has compared the prevalence of carotid plaque in men and women from early adulthood through old age. Prati and colleagues examined a total of 1348 subjects between the ages of 18 and 99 years and did not find plaque before the age of 40 years in either sex. In the MONICA Project Augsburg, a curvilinear relationship between age and carotid atherosclerosis in women seemed to appear but was not discussed by the authors. A direct comparison with our results is difficult, because the analysis in that study was done in 10-year age groups and none of the participants were >65 years old. The Bruneck Ischemic Heart Disease and Stroke Prevention Study, which included 909 subjects >40 years old, also performed analyses on 10-year age groups, and their data on prevalence of atherosclerosis are therefore difficult to compare with our findings. That study showed a declining sex difference in plaque prevalence at high ages similar to those we found. Apart from ultrasound-based studies, information on the prevalence of atherosclerosis and sex differences in the prevalence of atherosclerosis in the general population is scarce. In an autopsy survey, Sternby found age-related prevalence curves for coronary atherosclerosis in men and women similar to those we found for carotid atherosclerosis, with convergence of the prevalence of atherosclerosis in all 3 main coronary arteries in the youngest (<35 years) and oldest (>75 years) age groups.

It has been discussed whether coronary morbidity and mortality in women is affected by menopause and whether there is an acceleration in the risk of coronary disease and death after menopause. Recently, Tunstall-Pedoe described it as a myth of menopause that risk in women is held low until menopause, when it rebounds, becomes equal, and later surpasses that in men. He showed that the male excess in risk of coronary death continues to rise with age and that the sex gap never closes. Similar findings were reported by Barrett-Conner. Our findings of a premenopausally increasing sex gap in the prevalence of atherosclerosis and a postmenopausal decrease and ultimately closure of the gap at higher ages are not at variance with their findings, because clinical manifestations of vessel wall atherosclerotic lesions may be delayed by many years.

In addition, sex differences in plaque morphology may account for the prevailing male excess risk of coronary death in older age, despite a similar prevalence of atherosclerosis in elderly men and women. Our study shows, to the best of our knowledge for the first time in a general population, that men have softer plaques than women and that the male excess prevalence of soft plaques remains high in old age. One previous clinical study on ultrasound-assessed plaque morphology of stenotic plaques also found that women had harder plaques than men, but that study has limitations.

### Table 3

<table>
<thead>
<tr>
<th>Age, y</th>
<th>Men</th>
<th>Women</th>
<th>Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. With Plaques</td>
<td>Percentage With Soft Plaques</td>
<td>No. With Plaques</td>
</tr>
<tr>
<td>&lt;55</td>
<td>67</td>
<td>56.7</td>
<td>53</td>
</tr>
<tr>
<td>55–64</td>
<td>723</td>
<td>43.7</td>
<td>559</td>
</tr>
<tr>
<td>65–74</td>
<td>771</td>
<td>36.3</td>
<td>787</td>
</tr>
<tr>
<td>&gt;75</td>
<td>64</td>
<td>34.4</td>
<td>76</td>
</tr>
<tr>
<td>Total</td>
<td>1625</td>
<td>40.4</td>
<td>1475</td>
</tr>
</tbody>
</table>

Nonclassifiable plaques are not included in the analyses. Interaction was significant (P=0.005) between age and sex.
because the subjects were selected for carotid endarterectomy. Most myocardial infarctions and sudden coronary deaths are caused by the rupture of soft, lipid-rich plaques.\(^{10,24}\) If carotid and coronary plaques share common morphological characteristics within individuals, our finding may provide for the continued male excess risk of coronary death in older age. Sex differences in plaque morphology may also partly account for the substantially greater male-to-female ratio for atherosclerosis in elderly men and women. These findings may thereafter declines so that the prevalence of atherosclerosis is similar in elderly men and women. This present study indicates that men have softer plaques than women. We hypothesize that sex differences in plaque morphology may partly account for the prevailing male excess risk of coronary heart disease in the elderly.

**Acknowledgments**

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**References**


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**TABLE 4. Prevalence of Ultrasound-Assessed Carotid Atherosclerotic Lesions in Population-Based Studies of Men and Women**

<table>
<thead>
<tr>
<th>Study (n=No. of Participants)</th>
<th>Response Rate, %</th>
<th>Age Range Examined, y</th>
<th>Qualifying Lesion</th>
<th>Age, y</th>
<th>No. of Subjects</th>
<th>Prevalence of Plaque, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular Health Study (CHS), USA (n=5201)(^{16})</td>
<td>58</td>
<td>≥65</td>
<td>Focal protrusion</td>
<td>65–69</td>
<td>688</td>
<td>1126</td>
</tr>
<tr>
<td>Atherosclerosis Risk in Communities (ARIC), USA (n=14046)(^{17})</td>
<td>46–67</td>
<td>45–64</td>
<td>Two of 3 conditions: (1) Focal protrusion, or (2) High echo-brightness, or (3) IMT=1.5 mm</td>
<td>60–64</td>
<td>639</td>
<td>463</td>
</tr>
<tr>
<td>San Diele Project, Italy (n=1348)(^{18})</td>
<td>75</td>
<td>18–99</td>
<td>Focal protrusion or wall mineralization</td>
<td>60–69</td>
<td>90</td>
<td>104</td>
</tr>
<tr>
<td>MONICA Project Augsburg, Germany (n=1388)(^{19})</td>
<td>Not specified</td>
<td>25–65</td>
<td>Focal protrusion</td>
<td>55–64</td>
<td>214</td>
<td>185</td>
</tr>
<tr>
<td>Brunec Ischemic Heart Disease and Stroke Prevention Study, Italy (n=909)(^{20})</td>
<td>94</td>
<td>40–79</td>
<td>Focal protrusion</td>
<td>60–69</td>
<td>119</td>
<td>113</td>
</tr>
<tr>
<td>Present study, Tromsø, Norway (n=6727)</td>
<td>88</td>
<td>25–84</td>
<td>Focal protrusion</td>
<td>55–64</td>
<td>1422</td>
<td>1453</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>60–64</td>
<td>686</td>
<td>703</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>60–69</td>
<td>1308</td>
<td>1453</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>65–69</td>
<td>622</td>
<td>750</td>
</tr>
</tbody>
</table>

IMT denotes intima media thickness. Prevalence of plaque in comparable (overlap or adjacent) age groups is shown. There were small between-study differences in the ultrasound imaging procedures. In the ARIC study, only far-wall lesions were recorded. In the MONICA Project Augsburg, lesions in the external carotid artery were recorded. In the other studies, lesions from the bifurcation and from the common and internal carotid arteries were recorded. In the Tromsø Study, only the right carotid arteries were examined.
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