Echographic Assessment of Carotid and Femoral Arterial Structure in Men With Essential Hypertension

Jérôme Ganey, Alain Simon, Marc Massonneau, Ales Linhart, Patrick Segond, Jaime Levenson, and Groupe PCVMETRA

Extracoronary in vivo structural arterial changes were studied in asymptomatic essential hypertension. Carotid and femoral arteries were examined with B-mode echography for the presence or absence of plaque (the whole vascular segments of each vessel in the both sides) and for automated measurement of the far wall intima-media thickness (the vascular segment of each vessel proximal to the bifurcation in the right side) in 53 never treated hypertensive men and 133 normotensive men similar with regard to age, serum cholesterol levels, and smoking history. In the hypertensive group carotid plaque was more frequent \( (P < .05) \) and carotid and femoral intima-media thicknesses were greater \( (P < .001) \) than in the normotensive group. In the overall normotensive and hypertensive population intima-media thickness was independently associated with age and systolic pressure in both arteries \( (P < .001) \) and with cholesterol in the femoral artery \( (P < .05) \) while plaque was associated with systolic pressure \( (P < .01) \), and cholesterol \( (P < .01) \) in the carotid arteries and with age \( (P < .01) \), cholesterol \( (P < .05) \), and smoking \( (P < .001) \) in the femoral arteries. No significant difference in intima-media thickness in both arteries existed between hypertensive subjects with plaque and those without.

Wall thickening and plaque were more frequent in hypertensive patients. Thickening was distributed homogeneously to both arteries, while plaque affected preferentially the femoral bed. The influence of age and pressure was more marked on intima-media thickness than on plaque. The lack of association between wall thickening and plaque suggested that vascular hypertrophy and early atherosclerosis might be two different structural changes. Am J Hypertens 1996;9:126–136

KEY WORDS: Extracoronary vessels, arterial structure, wall thickness, arterial plaque, ultrasonography, hypertension.

Arterial damage in extracoronary vessels represents a major cause of complications in hypertension, but relatively little information exists about the clinically silent phase of its development many years before symp-toms occur. B-Mode ultrasonography is increasingly used to detect early structural changes in superficial peripheral arteries, such as early atherosclerosis (nonstenotic plaque) or increased intima-media thickness.\(^1\) The prevalence and the distribution of extracoronary plaques at different vascular sites remain unclear in hypertension,\(^5\) unlike hypercholesterolemia,\(^3\) but an increased wall thickness has been recently documented in peripheral arteries of asymptomatic hypertensive patients.\(^6\) Nevertheless the nature and the mechanisms of this thickening process are still unresolved. Some studies in subjects with risk
factors other than hypertension have suggested that increased wall thickness in the carotid artery can be an expression of early atherosclerosis. On the other hand, increased wall thickness does not necessarily mean atherosclerosis and may be compatible with other structural changes, such as vascular growth. Thus, the aim of the present investigation was to evaluate the presence of two structural changes (wall thickening and plaque) and their potential relations within the carotid and femoral arteries in men with essential hypertension never treated with antihypertensive treatment and without clinical evidence of cardiovascular disease.

METHODS

Study Population One hundred and thirty three control normotensive men (32 to 72 years) with a supine diastolic blood pressure <90 mm Hg (Korotkoff phase V) and 53 ambulatory male patients (25 to 75 years) with sustained hypertension, defined as a supine diastolic blood pressure >95 mm Hg on an average of three outpatient visits, entered the study. The study population was selected at their worksites, by a group of occupational health physicians known as Groupe PCVMETRA, who conducted a cardiovascular risk factor screening program for employees of several companies within the Paris area. All patients had essential hypertension documented by appropriate laboratory tests, and none had ever taken any antihypertensive treatment. Hypertension was uncomplicated in all patients, and none had neurological, cardiac, or renal involvement, or arteriopathy of the legs. Systolic blood pressure was determined at the time of investigation as the mean of at least three measurements by standard sphygmomanometric procedure in the arm with the patient in supine position after 10 min of rest. Systolic blood pressure did not exceed 150 mm Hg in the normotensive group and only nine normotensive subjects had systolic pressure between 140 and 150 mm Hg. Body mass index (weight/height²) was used to quantify excess of weight. Total cholesterol and triglycerides were measured by classical enzymatic methods after subjects had fasted for 14 h. A dyslipidemia was considered as present if total cholesterol was at 6.5 mmol/L or above or if triglycerides were at 2.3 mmol/L or above. Blood glucose was also measured after an overnight fast. The smoking status was expressed as current daily smoker or as lifelong dose in pack-years. After giving informed consent, subjects were referred to the vascular laboratory for arterial investigations.

Arterial Investigations Investigations were performed with a real-time, B-mode ultrasound imager (Ultramark 4, Advanced Technologies Laboratories, Les Ulis, France) by using a 7.5 MHz probe. The same physician (J.G.) made all investigations throughout the study.

Detection of Plaque The detection of plaque was performed in the extracranial carotid arteries and in the femoral arteries in the upper part of the thigh according to a procedure described in detail elsewhere. Briefly, the carotid scanning was performed on both sides and included the common carotid artery, the carotid bifurcation, the carotid bulb, and the internal carotid artery. The femoral scanning was also performed on both sides and included the common femoral artery, the femoral bifurcation, the superficial femoral artery, and the profunda femoral artery in the upper part of the thigh. The ultrasonic images were magnified and projected in real time on a television monitor. Arterial plaque was defined as an echogenic structure encroaching into the vessel lumen with a distinct area at least 50% thicker than the surrounding wall. During the scanning, the sonographic physician classified findings into two categories at the carotid and femoral sites: absence of any plaque, or presence of one or more plaques regardless of their location and number. The same physician checked the original classification in one session from the hard copies of real-time images made from longitudinal and axial sections of arteries. The whole examination of both sides of the two arteries took approximately 20 min.

Imaging of Intima-Media Thickness The measurement of intima-media thickness was performed in the far wall of the common carotid artery and of the common femoral artery 3 cm proximal to the bifurcation of each artery. Figure 1. To avoid prolonging the examination time, only the right side of the common carotid and femoral arteries far walls were examined, especially since this area is reported to have the least measurement variability for the carotid intima-media thickness. Scanning of the common carotid artery was performed in the anteroposterior projection, with the patient lying on his back with the head in axis. Scanning of the common femoral artery was also performed in the anteroposterior projection, with the patient lying on his back with the lower limb in slight external rotation. The ultrasonic images were projected in real time on a television monitor. During the scanning, the sonographic physician adjusted the sound beam perpendicularly to the arterial surface of the far wall of the vessel to obtain the two parallel echogenic lines corresponding to the lumen-intima and media-adventitia interfaces. If a plaque was present in the segment of measure, the subject was excluded from the study, because the plaque encroachment disrupted the double line arterial pattern of the intima-media complex. Once the two parallel
lines of the far wall were clearly visible on the television monitor along at least 1 cm of longitudinal length of the vessel, the frozen end-diastolic image (electrocardiographic R-triggering) of the double line arterial pattern was transferred on a computer (Apple Macintosh, Cupertino, CA), digitized into 640 x 580 peak cells with 256 gray levels, stored in a memory mass system, and restored on request for off-line analysis. The whole investigation of the two arterial sites, exclusive of the off-line analysis, took approximately 20 min.

**Right Common Carotid Artery**

**Right Common Femoral Artery**

**FIGURE 1.** Computer off-line visualization of the ultrasound image of the intima-media complex (double continuous white lines) in the far wall of the right common carotid artery (left panel) and of the right common femoral artery (right panel) 3 cm proximal to the bifurcation of each vessel. The white rectangles drawn by the reader around the area of interest in the far wall of the vessels determined the segment (1 cm of length) of automatic measurement of the intima-media thickness (distance between the two parallel lines).

**Automated Intima-Media Thickness Measurement**

The off-line analysis of image, was performed automatically according to an automated computerized procedure previously validated (lotec System, Ío Data Processing Co., Paris, France). The image was transferred from the storage memory mass system to the television monitor of the computer without the investigator knowing the blood pressure status of the subject. The reader drew a rectangular field of measure (of at least 1 cm long) around the area of interest (including the double line arterial pattern) using a mouse (Figure 1). A four-step detection process was then used. For the first step, the computer analyzed globally the statistical distribution of the pixel gray densities and identified automatically the locations of the blood-intima and media-adventitia interfaces by computing the rate of intensity change in gray level. For the second step, the computer validated the points consistent with the expected behavior at the two interface levels and substituted the points of discontinuity or the aberrant points (parasites or ultrasonic reading) for the use of appropriate algorithms. For the third step, the vertical and horizontal coherence of each pixel labelled as an interface point was performed. For the fourth step, the whole set of interface points were visualized on the television monitor of the computer by two continuous lines (Figure 1) and the computer concomitantly determined by the intima-media thickness as the distance of the two lines calculated by their average difference at 100 successive points. The total time for each automated image analysis was about 0.15 sec.

**Data Analysis**

The statistical analysis was carried out on a computer (Apple Macintosh, Cupertino, CA) with the use of Statsview (Abacus Concepts Corp.,...
Berkeley, CA) JMP (SAS Institute, Cary, NC, and Excel (Microsoft, Les Ulis, France) software. Mean values ± standard deviation (SD) in the normotensive and hypertensive groups were calculated and compared by Student’s t test. Differences in prevalences between the two groups were compared by a χ² test. The relation between continuous variables was evaluated by linear regression and that of dichotomous variable (plaque: presence/absence) by logistic regression. The independence of associations was assessed by multivariate analysis.

**RESULTS**

**Study Population** Table 1 shows that normotensive and hypertensive groups were similar with regard to age, cholesterol, triglycerides, prevalence of dyslipidemia, and smoking exposure. Nevertheless, body mass index and blood glucose were significantly higher (P < .001) in the hypertensive group than in the normotensive group (Table 1), but diabetes mellitus (fasting blood glucose at 7.7 mmol/L or above) did not exist in either group.

**Wall Thickness and Arterial Plaque** Figure 2 shows the distribution of intima-media thickness values in both arteries and in the two study groups. The proportion of hypertensive patients with intima-media thickness above the 90th percentile of the normotensive group was 30% for the carotid artery and 25% for the femoral artery.
the femoral artery. The mean value of intima-media thickness was significantly higher in the normotensive group than in the hypertensive group in both carotid (P < .001) and femoral (P < .001) arteries (Figure 2).

Carotid and femoral intima-media thicknesses were correlated together in the normotensive group (r = 0.47, P < .001, Figure 3), in the hypertensive group (r = 0.33, P < .01, Figure 3), and in the pooled normotensive and hypertensive groups (r = 0.46, P < .001).

Figure 4 shows the distribution of plaque in both arteries in the two study groups. Plaque in the carotid arteries was more frequent in the hypertensive group than in the normotensive group (20% vs 10%, P < .05). Plaque in the femoral arteries was nonsignificantly more frequent in the hypertensive group than in the normotensive group (39% vs 27%, P = .07). Plaque was more frequent in the femoral artery than in the carotid artery in the normotensive group (P < .05) as well as in the hypertensive group (P < .01).

Relation of Risk Factors With Arterial Structure
Univariate relations between risk factors and arterial structure are shown in Tables 2 and 3. Age was correlated with carotid and femoral intima-media thickness in the normotensive group (r = 0.43, r = 0.45, P < .001) and in the hypertensive group (r = 0.29, P < .05; r = 0.51, P < .001) (Table 2 and Figure 5). The slope of the regression line relating age (in years) to carotid intima-media thickness (in millimeters) was 0.0034 mm/year in the hypertensive group and 0.0045 mm/year in the normotensive group. The slope of the regression line relating age to femoral intima-media thickness was 0.010 mm/year in the hypertensive group and 0.0071 mm/year in the normotensive group. No significant difference in the slopes corresponding to each vessel existed between the normotensive and hypertensive groups. Age was correlated with carotid and femoral plaque in the normotensive group (r = 0.23, P < .05; r = 0.25, P < .01) in the hypertensive group (r = 0.23, P = NS; r = 0.36, P < .01) (Table 3). Blood pressure was correlated to carotid intima-media thickness (significantly for systolic and diastolic: r = 0.27, r = 0.24, P < .01) and to femoral intima-media thickness systolic r = 0.17, P = NS, diastolic r = 0.19, P < .05 in the normotensive group (Table 2). Blood pressure was slightly correlated to carotid intima-media thickness: (r = 0.26, P = .05 for systolic and diastolic) and to femoral intima-media thickness nonsignificantly for systolic (r = 0.25) and just significantly for diastolic (r = 0.26, P = .05) in the hypertensive group (Table 2). Blood pressure was only correlated to carotid plaque in the normotensive group (r = 0.39, P < .001 for systolic and r = 0.28, P < .05 for diastolic) (Table 3).

Total cholesterol was correlated to carotid and femoral intima-media thickness (r = 0.20, P < .05 and r = 0.33, P < .001) in the normotensive group, but not in the hypertensive group (Table 2). Total cholesterol was correlated with carotid and femoral plaque in the normotensive group (r = 0.44, P < .001 and r = 0.21, P < .05) and with femoral plaque only in the hypertensive group (r = 0.27, P < .05) (Table 3).

Lifelong smoking dose was correlated to carotid and femoral intima-media thickness (r = 0.21, P < .05; r = 0.28, P < .01) in the normotensive group but not in the normotensive group.
the hypertensive group (Table 2). Lifelong smoking dose was correlated with femoral plaque in the normotensive group \( (r = 0.57, P < .001) \) and in the hypertensive group \( (r = 0.33, P < .01) \) but such correlation did not exist with carotid plaque in any group (Table 3).

In multivariate analyses of the overall normotensive and hypertensive population (including age, systolic pressure, cholesterol, and smoking) carotid intima-media thickness was independently associated with age \( (P < .001) \) and systolic pressure \( (P < .001) \) and femoral intima-media thickness was independently associated with age \( (P < .001) \), systolic pressure \( (P < .01) \), and cholesterol \( (P < .05) \) (Table 4).

The similar multivariate analyses for plaque showed that carotid plaque was independently associated with systolic pressure \( (P < .01) \) and cholesterol \( (P < .01) \) and that femoral plaque was associated with age \( (P < .01) \), cholesterol \( (P < .05) \) and smoking \( (P < .001) \) (Table 4).

Relation Between Wall Thickens and Plaque The carotid intima-media thickness was not significantly different between subjects with carotid plaque and those without, in the normotensive as well as in the hypertensive group (Figure 6). The same nonsignificant difference in intima-media thickness was observed when the comparison of subjects with and without carotid plaque was restricted to the presence or absence of plaque in the right carotid side ipsilateral to the side of intima-media thickness measurement \( (0.56 \pm 0.14 \text{ or } 0.53 \pm 0.08 \text{ mm in the normotensive group}, \text{ and } 0.60 \pm 0.13 \text{ or } 0.59 \pm 0.10 \text{ mm in the hypertensive group}) \).

The femoral intima-media thickness was higher in normotensive subjects with femoral plaque than in those without \( (P < .01) \) (Figure 7), but this difference disappeared when the comparison of subjects with and without femoral plaque was adjusted for age by covariance analysis. The femoral intima-media thickness was not different in hypertensive subjects with femoral plaque than in those without (Figure 7). The same findings were observed when the comparison of subjects with and without femoral plaque was restricted to the presence or absence of plaque in the right femoral side \( (0.62 \pm 0.13 \text{ or } 0.55 \pm 0.12 \text{ mm}, P < .01) \), but nonsignificant for the age adjusted comparison in the normotensive group, and \( 0.69 \pm 0.21 \text{ or } 0.62 \pm 0.17 \text{ mm}, \text{nonsignificant in the hypertensive group}) \).

**DISCUSSION**

The objective of the study was to detect early structural changes in carotid and femoral arteries in asymptomatic hypertensive men compared with normotensive male controls. The value of carotid intima-media thickness found with our automatic procedure especially in control subjects was, on average, lower than most values obtained with other methods in the literature. Nevertheless intima-media thickness increases with age and in a number of literature studies the subjects investigated were older than in the present work.\(^2,6,8,13,15\) In the few reports including subjects with ages similar to ours,\(^10,16,17\) only slight differences, in intima-media thickness existed compared to our values and these differences could be attributed to the geographic difference of the populations studied. Furthermore, the accuracy of our method of measurement of intima-media thickness has been previously tested by comparing in vitro ultrasonographic and histological measures in a small series of human arterial specimens.\(^10\) Both measures were strongly correlated, but the histological measures overestimated the ultrasound measure, probably because our histological technique included dehydration and staining, which might have artifactually thickened the intima-media complex.\(^10\) We have also verified that the automatic

**TABLE 2. CORRELATIONS BETWEEN CAROTID AND FEMORAL INTIMA-MEDIA THICKNESS AND THE RISK FACTORS IN THE TWO STUDY GROUPS**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normotensive Group</th>
<th>Hypertensive Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Carotid Thickness</td>
<td>Femoral Thickness</td>
</tr>
<tr>
<td>Age</td>
<td>0.43*</td>
<td>0.45†</td>
</tr>
<tr>
<td>Body mass index</td>
<td>0.16</td>
<td>0.07</td>
</tr>
<tr>
<td>Body surface area</td>
<td>0.00</td>
<td>0.02</td>
</tr>
<tr>
<td>Blood pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>0.27†</td>
<td>0.17</td>
</tr>
<tr>
<td>Diastolic</td>
<td>0.24†</td>
<td>0.19*</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>0.20*</td>
<td>0.33†</td>
</tr>
<tr>
<td>Ln (triglycerides)</td>
<td>0.00</td>
<td>0.15</td>
</tr>
<tr>
<td>Blood glucose</td>
<td>0.01</td>
<td>0.06</td>
</tr>
<tr>
<td>Ln (lifelong smoking)</td>
<td>0.21*</td>
<td>0.28†</td>
</tr>
</tbody>
</table>

\( \text{Ln, Napierian logarithm. Values are correlation coefficients.} \)

\( ^*P < .05, ^†P < .01, ^‡P < .001. \)
TABLE 3. UNIVARIATE LOGISTIC REGRESSIONS BETWEEN PLAQUE (PRESENCE/ABSENCE) AND RISK FACTORS

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normotensive Group</th>
<th>Hypertensive Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Carotid Plaque</td>
<td>Femoral Plaque</td>
</tr>
<tr>
<td>Age</td>
<td>0.23*</td>
<td>0.25†</td>
</tr>
<tr>
<td>Body mass index</td>
<td>0.11</td>
<td>0.06</td>
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<tr>
<td>Blood pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>0.39†</td>
<td>0.04</td>
</tr>
<tr>
<td>Diastolic</td>
<td>0.28*</td>
<td>0.13</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>0.44†</td>
<td>0.21*</td>
</tr>
<tr>
<td>Ln (triglycerides)</td>
<td>0.24*</td>
<td>0.15</td>
</tr>
<tr>
<td>Blood glucose</td>
<td>0.02</td>
<td>0.15</td>
</tr>
<tr>
<td>Ln (lifelong smoking)</td>
<td>0.13</td>
<td>0.57‡</td>
</tr>
</tbody>
</table>

Ln. Neperian logarithm. Values are correlation coefficients.

*P < .05, †P < .01, ‡P < .001.

As previously reported, we found that untreated method gave close values of intima-media thickness compared to the classical operator-assisted computer measurement technique. The automatic method has the advantage of being more precise as a consequence of the great number of measurements performed (100 successive points of measure) and more reproducible by minimizing observer bias.

FIGURE 5. Correlations between common carotid and femoral artery intima-media thickness and age in the two study groups. The continuous lines indicate the linear regression lines. For the values of the slopes see results.
essential hypertension was associated with an increased intima-media thickness in both arteries. The proportion of hypertensive subjects with intima-media thickness value above the 90th percentile of the normotensive group was close in the carotid artery (30%) and in the femoral artery (25%). The independent influence of hypertension on intima-media thickening was attested by significant multivariate associations between systolic pressure and intima-media thickness in the normotensive and hypertensive population as a whole. Moreover, univariate regressions between blood pressure and wall thickness showed similar correlation coefficients in separate normotensive and hypertensive groups, but the correlation values were not significant in hypertensives since the sample size was smaller than in normotensives. Thus, the increased wall thickness may be a response to the hemodynamic stimulus of increased pressure, but it also may be related to other mechanisms associated with hypertension, such as vascular growth. We have previously reported that intima-media wall thickening was associated with normal lumen diameter in hypertensive subjects, which implied a greater cross-sectional area of the intima-media thickness. This phenomenon is similar to the compensatory enlargement of arteries associated with atherosclerosis, but the mechanisms are probably quite different between hypertension and atherosclerosis. Furthermore, many risk factors other than hypertension were associated with intima-media thickness. Age was strongly correlated to carotid and femoral intima-media thickness, emphasizing the need for comparing groups of similar age in studies of wall thickness measure-

### TABLE 4. MULTIVARIATE ANALYSES OF RISK FACTORS INFLUENCING ARTERIAL STRUCTURE IN THE NORMOTENSIVE AND HYPERTENSIVE GROUPS

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>Carotid Site</th>
<th>Femoral Site</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Thickness</td>
<td>Plaque</td>
</tr>
<tr>
<td>Age</td>
<td>&lt;.001</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic pressure</td>
<td>&lt;.001</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>NS</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Lifelong smoking</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Data are P values.
NS, nonsignificant.

**FIGURE 6.** Common carotid artery intima-media thickness and age in subjects with carotid plaque and in those without in the two study groups. Data are mean values ± SD. NS = nonsignificant.
ments. Cholesterol and smoking exposure were correlated to carotid and femoral intima-media thickness in the normotensive group but not in the hypertensive group, and such associations disappeared in the multivariate analysis of the overall groups except the association between cholesterol and femoral thickness. These observations support the possibility that the effect of hypertension on intima-media thickness might outweigh the effects of certain other associated risk factors.

Another finding of the study was the greater prevalence of carotid plaque in the hypertensive group than in the normotensive group, as previously reported. In contrast, femoral plaque was nonsignificantly more frequent in the normotensive group than in the femoral thickness. This may be explained by the fact that the femoral artery is exposed to higher hydrostatic pressure and to different flow patterns than the carotid artery, especially in the hypertensive group. This may be explained by the fact that the femoral artery is exposed to higher hydrostatic pressure and to different flow patterns than the carotid artery, and may have greater susceptibility to the atherogenic influence of risk factors, as supported by the associations found between femoral plaque and age, cholesterol, and smoking.

When analyzing the association between wall thickening and the prevalence of plaque in the two vessels, we found no greater intima-media thickness when plaque was present in the same vascular bed, except in the normotensive group in whom a greater femoral thickness existed in subjects with femoral plaque. Nevertheless, subjects with plaque were 5 years older than those without and the femoral thickness differences, according to the presence or absence of femoral plaque disappeared when the analysis was adjusted to age. The lack of association between wall thickening and plaque did not support the common idea that the increase in intima-media thickness can be considered as an obligatory indicator of atherosclerotic process. It is somewhat at odds with reports in which the subdivision of normotensive and hypertensive subjects according to the presence or absence of carotid atherosclerotic plaque revealed that the subjects with atherosclerosis had increased carotid wall thickness. Nevertheless, such an increase in wall thickness could be due to the fact the subjects with plaque were older than those without plaque. Therefore, the intima-media thickening in hypertension is compatible with a nonatherosclerotic process such as intimal or medial hypertrophy. Unfortunately, our current ultrasonic imaging, even with the assis-

**Figure 7.** Common femoral artery intima-media thickness and age in subjects with femoral plaque and in those without in the two study groups. Data are mean values ± SD. NS = nonsignificant. *P < .01.
tance of computerized image analysis, could not discriminate the part of the media from that of the intima in the thickening of the intima-media complex.

In conclusion, the prevalence of wall thickening and plaque was increased in hypertension but the thickening process was distributed more homogeneously than plaque to carotid and femoral vessels. Age and blood pressure influenced wall thickness in both arteries only, whereas multiple risk factors influenced the plaque prevalence to different degrees according to the site. The lack of association between wall thickening and plaque suggested that vascular hypertrophy and early atherosclerosis may be two different structural changes. However, these findings have been obtained in men and therefore they cannot be extrapolated to hypertensive women.

ACKNOWLEDGMENTS


We also thank Mrs Isabelle d’Argenlé for her excellent secretarial assistance.

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