Risk Factors and Raised Atherosclerotic Lesions in Coronary and Cerebral Arteries

Statistical Analysis from the Oslo Study

Ingar Holme, Sven C. Enger, Anders Helgeland, Ingvar Hjermann, Paul Leren, Per G. Lund-Larsen, Lars A. Solberg, and Jack P. Strong

In 1972-1973, about 16,200 men living in Oslo, aged 40 to 49 years, were examined for cardiovascular disease, and had a number of coronary risk factors measured. This report gives the results of 129 autopsied cases with regard to the association between raised atherosclerotic lesions in coronary and cerebral arteries and various coronary risk factors. For coronary raised lesions, the high density lipoprotein (HDL) cholesterol ratio was the most significant risk factor. Systolic blood pressure and total serum cholesterol were also significantly associated. Physical activity at work and at leisure, nonfasting triglycerides, and cigarette smoking did not show a significant association with coronary artery raised lesions. The association between total serum cholesterol and systolic blood pressure indicates that total serum cholesterol may be more important than systolic blood pressure in the synergism affecting the development of coronary atherosclerosis. For cerebral artery raised lesions, blood pressure was the most important risk factor, even though serum cholesterol was highly associated with the lesions. The interaction analysis also suggested that blood pressure was more important than serum cholesterol in the synergism.

Numerous epidemiological reports have demonstrated an association between certain risk factors and coronary heart disease (CHD). Since coronary atherosclerosis as a rule forms the basis for the development of CHD, the assumption has been made that risk factors for CHD are identical with those for coronary atherosclerosis. This assumption may be true for some risk factors but not necessarily for all of them. Risk factors could also act directly on the myocardium and its conduction system and thus contribute to the development of clinical disease.

Since quantitative estimates of atherosclerosis are difficult to obtain in living persons, most information about risk factors has been obtained from retrospective autopsy studies. More recently, several prospective studies have been initiated that are characterized by careful documentation of certain risk factors during life and standardized evaluation of atherosclerotic lesions at autopsy. These studies have shown significant relationships between levels of serum cholesterol, blood pressure (BP), other selected risk factors, and the extent of coronary atherosclerosis. This report analyzes in more detail the multivariate relationship between risk factors and raised atherosclerotic lesions in the coronary and cerebral arteries of the population in the Oslo study.
Methods

The Oslo Study has been described elsewhere. Briefly, in 1972–1973 all men living in Oslo, aged 40 to 49 years were asked to undergo a medical examination that included BP measurement and blood samples for lipid analysis. A questionnaire on previous cardiovascular disease, diabetes mellitus, physical activity at work and at leisure, and smoking habits was also administered. The details of the screening and laboratory and postmortem procedures have already been reported.

A coronary risk score was computed for each man, based on values for serum cholesterol, systolic BP, and the number of cigarettes smoked daily. Of this group, autopsies were performed on 150 men who had attended the health screening. This study deals with the 129 men whose data were complete except for a lack of HDL cholesterol measurement in some cases.

The dependent variable in the analyses performed was the percentage of the intimal surface covered with raised atherosclerotic lesions in either the coronary arteries or in the main intracranial arteries. Different measures of risk were used as independent variables, including serum cholesterol, HDL cholesterol, cholesterol ratio (HDL cholesterol/[total cholesterol − HDL cholesterol]), nonfasting serum triglycerides, systolic and diastolic BP, number of cigarettes smoked daily, coronary risk score, and degree of physical activity at work and at leisure.

Stepwise linear multiple regression analyses were performed on these data using different sets of the risk factors as independent variables. Transformations of the dependent variables were not used, although raised lesions (RL) in the intracranial arteries appeared to have a slightly skewed distribution. The effect of moderate skewness on p values, however, is known to be small.

Differences in the RL-risk factor correlations in the coronary and cerebral arteries were tested using the t statistic and standard Z transforms of the calculated product moment correlations.

Analysis of Risk Factors

The mean value and standard deviation (sd) for measures of selected risk factors in the autopsy series and in those men who died but were not autopsied are presented in Table 1. Approximately 46% of all men in the total cohort who died underwent autopsy. While there were some significant differences between the autopsied men and those who were not autopsied, most of the differences were moderate. An exception was in the number of cigarettes smoked, for which the autopsied men had considerably greater values. Thus, there seems to have been considerable selectivity in the autopsy series in regard to cigarette smoking.

The simple correlation coefficients between dependent and independent variables are presented in Table 2. As expected, correlations between systolic and diastolic BP, and between coronary risk score and its three components, serum cholesterol, systolic BP, and number of cigarettes smoked, were highly significant (p < 0.01). Cholesterol ratio and total serum cholesterol were also highly correlated (p < 0.01). The correlation between RL in coronary and cerebral arteries was high (p < 0.01) but it should be noted that this correlation indicates "only" about 20% common lesion variation in the two arterial segments. Cholesterol ratio, serum cholesterol, and systolic and diastolic BP were the independent variables most highly correlated with coronary RL, while coronary risk score was only of borderline significance. The absolute value of HDL cholesterol was a weaker associate of RL than the cholesterol ratio in both the coronary and cerebral arteries. Such factors as triglycerides, physical activity at work and leisure, and cigarette smoking were not significantly associated with coronary RL.

In the case of raised lesions in the cerebral arteries, the highest correlation coefficients corresponded to diastolic and systolic BP, followed by coronary risk score, serum cholesterol, and

### Table 1. Mean Value and Standard Deviation of Selected Risk Factors in the Autopsy Series from a Cohort of Deaths Among Oslo Men Aged 40–49 Years

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>Autopsy series (n = 146)</th>
<th>Not autopsied (178)</th>
<th>Total cohort (324)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum cholesterol (mg/dl)</td>
<td>272.8 ± 44.7</td>
<td>287.5 ± 56.57</td>
<td>280.9 ± 52.0</td>
</tr>
<tr>
<td>Serum triglycerides (mmole/liter)</td>
<td>2.73 ± 1.74</td>
<td>2.27 ± 1.07</td>
<td>2.48 ± 1.43</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>145.2 ± 20.3</td>
<td>139.6 ± 17.52</td>
<td>142.1 ± 19.0</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>92.2 ± 12.5</td>
<td>89.5 ± 13.31</td>
<td>90.7 ± 13.0</td>
</tr>
<tr>
<td>No. cigarettes</td>
<td>11.4 ± 8.9</td>
<td>7.0 ± 5.38</td>
<td>9.0 ± 7.5</td>
</tr>
<tr>
<td>Risk score</td>
<td>23.2 ± 30.6</td>
<td>15.2 ± 24.37</td>
<td>18.8 ± 27.6</td>
</tr>
</tbody>
</table>

*p < 0.01 for differences between autopsied and not autopsied cases.
chol: sterol ratio. Again, triglycerides, physical activity, and cigarette smoking showed no signifi-
cant correlations with raised lesions in the arter-
ies. Use of the $t$ statistic and the $Z$ transform for testing differences between correlation coeffi-
cients of corresponding risk factors with RL in
the coronary and cerebral arteries did not show
any of the observed differences to be significant
statistically.

Tables 3 and 4 present results of forward step-
wise linear multiple regression analyses. Be-
cause of a high degree of correlation between
systolic and diastolic BP, only one of these two
factors was used in any one regression model.
This was done to facilitate the interpretation of
BP results, although there was a risk of not de-
tecting some interactions. Similar techniques
were used for the HDL cholesterol and choles-
terol ratio.

The stepwise multiple regression analysis also
suggested that the cholesterol ratio was the risk
factor that correlates best with coronary RL (table 3). The reason that it "lost its power" in
Step 3 of the analysis is that the simple correla-
tion between serum cholesterol and cholesterol
ratio is high ($r = 0.441$), and, therefore, some of

Table 3. Partial and Multiple Correlation Coefficients and Degree of Explanation ($R^2$) of Coronary Raised Lesions by Several Coronary Risk Factors in a Stepwise Regression Analysis

<table>
<thead>
<tr>
<th>Step</th>
<th>Cholesterol ratio</th>
<th>Systolic BP</th>
<th>Serum cholesterol</th>
<th>Physical activity at work</th>
<th>Multiple correlation coefficient</th>
<th>Degree of &quot;explanation&quot; (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-0.352</td>
<td></td>
<td></td>
<td></td>
<td>0.352</td>
<td>12.4</td>
</tr>
<tr>
<td>2</td>
<td>-0.341</td>
<td>0.313</td>
<td></td>
<td></td>
<td>0.458</td>
<td>21.0</td>
</tr>
<tr>
<td>3</td>
<td>-0.241</td>
<td>0.309</td>
<td>0.188</td>
<td></td>
<td>0.488</td>
<td>23.8</td>
</tr>
<tr>
<td>4</td>
<td>-0.237</td>
<td>0.307</td>
<td>0.192</td>
<td>-0.152</td>
<td>0.505</td>
<td>25.5</td>
</tr>
</tbody>
</table>

*Full regression model with all risk variables included (except coronary risk score).
Model applied to data obtained from 129 autopsied Oslo men aged 40–49 years.

Table 4. Partial and Multiple Correlation Coefficients and Degree of Explanation ($R^2$) of Cerebral Raised Lesions by Several Coronary Risk Factors in a Stepwise Regression Analysis

<table>
<thead>
<tr>
<th>Step</th>
<th>Systolic BP</th>
<th>Serum cholesterol</th>
<th>Multiple correlation coefficient</th>
<th>Degree of &quot;explanation&quot; (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.397</td>
<td></td>
<td>0.397</td>
<td>14.4</td>
</tr>
<tr>
<td>2</td>
<td>0.370</td>
<td>0.292</td>
<td>0.465</td>
<td>21.6</td>
</tr>
</tbody>
</table>

*Full regression model with all risk variables included (except coronary risk score).
Model applied to data obtained from 129 autopsied Oslo men aged 40–49 years.
Table 2 (continued)

<table>
<thead>
<tr>
<th>Systolic BP</th>
<th>No. of cigarettes</th>
<th>Cholesterol ratio</th>
<th>Diastolic pressure</th>
<th>Risk score</th>
<th>HDL chol.</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.326†</td>
<td>-0.039</td>
<td>-0.352†</td>
<td>0.275†</td>
<td>0.181†</td>
<td>-0.251†</td>
</tr>
<tr>
<td>-0.043</td>
<td>-0.068</td>
<td>0.052</td>
<td>-0.008</td>
<td>-0.036</td>
<td>0.046</td>
</tr>
<tr>
<td>-0.068</td>
<td>-0.263†</td>
<td>0.074</td>
<td>0.152</td>
<td>-0.134</td>
<td>0.139</td>
</tr>
<tr>
<td>-0.040</td>
<td>0.068</td>
<td>0.332†</td>
<td>0.005</td>
<td>0.208†</td>
<td>-0.271†</td>
</tr>
<tr>
<td>0.093</td>
<td>0.090</td>
<td>-0.441†</td>
<td>0.084</td>
<td>0.547†</td>
<td>-0.113</td>
</tr>
<tr>
<td>1</td>
<td>0.049</td>
<td>-0.097</td>
<td>0.723†</td>
<td>0.496†</td>
<td>-0.082</td>
</tr>
<tr>
<td>0.049</td>
<td>1</td>
<td>-0.029</td>
<td>0.072</td>
<td>0.361†</td>
<td>-0.026</td>
</tr>
<tr>
<td>-0.097</td>
<td>-0.029</td>
<td>1</td>
<td>-0.155</td>
<td>-0.242†</td>
<td>0.915†</td>
</tr>
<tr>
<td>0.273</td>
<td>0.072</td>
<td>-0.155</td>
<td>1</td>
<td>0.370†</td>
<td>-0.150</td>
</tr>
<tr>
<td>0.496</td>
<td>0.351</td>
<td>-0.242</td>
<td>0.370</td>
<td>1</td>
<td>-0.143</td>
</tr>
<tr>
<td>-0.082</td>
<td>-0.026</td>
<td>0.915</td>
<td>-0.150</td>
<td>-0.143</td>
<td>1</td>
</tr>
</tbody>
</table>

*p ≤ 0.05.
†p ≤ 0.01.

the information in the latter is included in the partial coefficient for the cholesterol value. The degree of explanation of RL variability is only moderately increased by including factors other than cholesterol ratio and systolic BP. Including diastolic instead of systolic BP in the model induced some inconsequential changes in the pattern described. The only difference was that the partial correlation coefficient between coronary RL and diastolic BP, after accounting for cholesterol ratio, was somewhat lower than for systolic BP (r = 0.240 vs 0.313).

In the case of cerebral RL, systolic BP was a better predictor than serum cholesterol, the other significant risk factor identified in the stepwise regression analysis (see table 4). All other risk factors did not substantially reduce the residual RL variance. Diastolic BP correlated as well or better with cerebral RL than systolic BP (r = 0.390).

To test the hypothesis that BP adds significantly to RL after adjusting for the lipid factors (total serum cholesterol and HDL cholesterol), we calculated the partial correlation coefficients of coronary and cerebral RL with systolic BP before and after adjusting for lipid factors in the stepwise regression analysis (table 5). While BP, total serum cholesterol, and HDL cholesterol were almost orthogonal (all intercorrelations 0.113 or less in table 2), adjustment by the lipid factors produced only small changes in the correlation between BP and RL. On the other hand, the multiple correlations between coronary and cerebral RL with lipid factors (cholesterol ratio and serum cholesterol) were 0.397 and 0.311, giving a 15.7% and 9.7% degree of explanation, whereas the multiple correlation coefficients amounted to 0.488 and 0.476, giving a 23.8% and 22.7% degree of explanation of RL variability when systolic BP was added to the model. Although the data are not presented, analogous results were obtained using diastolic BP instead of systolic BP in the multiple regression models. Thus, BP was still a significant risk factor for cerebral RL after adjusting for lipid factors.

When we replaced serum cholesterol, systolic BP, and the number of cigarettes smoked daily by their composite — the coronary risk score — in the stepwise regression model, the predictions of coronary atherosclerosis were poor; the composite score did not even enter as an independent risk factor in the stepwise procedure. In the forced full regression model, the partial correlation coefficient between coronary RL and coronary risk score was only 0.100 (p > 0.10), suggesting that the composite risk score was not significantly associated with coronary RL when the cholesterol ratio was included among the independent variables in the stepwise adjustment. On the other hand, the only significant risk factor for cerebral RL in the modified model was the composite coronary risk score. However, this had considerably lower predictive power in comparison with the factors presented in table 4.

Table 5. Partial Correlation Coefficients between Coronary and Cerebral Raised Lesions and Systolic Blood Pressure, Adjusting for Total Serum Cholesterol and then for HDL-cholesterol

<table>
<thead>
<tr>
<th>Adjusting factor</th>
<th>Coronary</th>
<th>Cerebral</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>0.326†</td>
<td>0.397†</td>
</tr>
<tr>
<td>Serum cholesterol</td>
<td>0.313†</td>
<td>0.389†</td>
</tr>
<tr>
<td>Serum cholesterol and HDL cholesterol</td>
<td>0.305*</td>
<td>0.386*</td>
</tr>
</tbody>
</table>

*p < 0.05.
Table 6. Product Moment Correlations of Raised Lesions With Measures of Selected Risk Factors in Different Tertile Groupings

<table>
<thead>
<tr>
<th>Correlated Variable</th>
<th>Arterial segment</th>
<th>( T_1 )</th>
<th>( T_2 )</th>
<th>( T_3 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum cholesterol</td>
<td>Coronary</td>
<td>0.485</td>
<td>0.353</td>
<td>0.185</td>
</tr>
<tr>
<td></td>
<td>Cerebral</td>
<td>0.327</td>
<td>0.259</td>
<td>0.292</td>
</tr>
<tr>
<td>Cholesterol ratio</td>
<td>Coronary</td>
<td>-0.432</td>
<td>-0.418</td>
<td>-0.292</td>
</tr>
<tr>
<td></td>
<td>Cerebral</td>
<td>-0.140</td>
<td>-0.309</td>
<td>-0.053</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>Coronary</td>
<td>0.359</td>
<td>0.531</td>
<td>-0.076</td>
</tr>
<tr>
<td></td>
<td>Cerebral</td>
<td>0.228</td>
<td>0.547</td>
<td>0.400</td>
</tr>
<tr>
<td>Serum triglycerides</td>
<td>Coronary</td>
<td>-0.085</td>
<td>0.099</td>
<td>0.029</td>
</tr>
<tr>
<td></td>
<td>Cerebral</td>
<td>-0.140</td>
<td>0.136</td>
<td>-0.219</td>
</tr>
</tbody>
</table>

\( T_1 = \) lower tertile; \( T_2 = \) middle tertile; \( T_3 = \) upper tertile.

Data obtained from 129 autopsied Oslo men aged 40–49 years.

Analysis of Synergistic Effects

To examine possible multiplicative (synergistic) effects of BP and serum lipids on RL, we divided the cases into strata (tertiles) based on serum cholesterol and systolic BP values and then computed the simple correlation coefficients between RL and these independent variables within each stratum (table 6). The correlation coefficients between serum cholesterol and coronary RL differed in the BP tertiles. In the lower BP tertile, there was a strong correlation between serum cholesterol and coronary RL (\( r = 0.485 \)), but in the upper tertile, the correlation was only of borderline significance (\( r = 0.185 \)). In the middle BP tertile, the correlation of serum cholesterol with RL was moderate. Thus, there was a marked interaction between BP and serum cholesterol with regard to association with coronary RL. This tendency, however, did not seem to hold in the cerebral arteries, where the correlation coefficients were about the same in the three BP strata. Conversely, the correlations between systolic BP and coronary RL within tertiles of serum cholesterol (table 6) were not as systematic, suggesting that serum cholesterol may be a more important contributor to the interactive synergism of the three factors in the coronary arteries. The correlation with systolic BP within tertiles of serum cholesterol appeared to be more systematic for cerebral RL than for coronary RL, perhaps indicating that in the cerebral arteries BP may be more important in the synergistic effects of the factors under consideration.

We also observed that the interactive effect of the cholesterol ratio with systolic BP was similar to that of serum cholesterol in terms of coronary RL. The correlation coefficients of RL with serum triglycerides seemed to vary unsystematically with the systolic BP and were not suggestive of interactive effects between triglycerides, BP, and raised lesions in either coronary or cerebral arteries.

Discussion

Some highly significant relationships between suspected coronary risk factors and the extent of raised atherosclerotic lesions are shown in this epidemiologic study with autopsy follow-up. There is a significant positive relationship between the level of total serum cholesterol or BP and the extent of coronary and cerebral raised atherosclerotic lesions. There is a significant negative relationship between HDL cholesterol or the HDL cholesterol to non-HDL cholesterol ratio and coronary raised lesions. These statistically significant relationships between risk factors measured in living persons and the extent of atherosclerotic lesions measured after death and autopsy were detected despite recognized methodological limitations. For example, the method of evaluating atherosclerotic lesions was based on standardized visual estimation rather than on an objective direct measurement of the extent of involvement. The analyses were based on only a single measurement of any one risk factor and the measurement was made at variable times, from a few weeks to 6 years before death and autopsy. Therefore, nothing is known about the time course of the risk factors during the life of the individual. The fact that this study demonstrates significant relationships despite these limitations suggests that the associations are indeed strong.
Several other concurrent epidemiologic studies with autopsy follow-up show similar relationships between total serum cholesterol and BP levels with the extent of atherosclerotic lesions. A unique feature of our present study was the measurement of serum LDL cholesterol levels in many of the autopsied subjects. We believe that this is the first study to examine the relationship between HDL cholesterol or the HDL cholesterol to non-HDL cholesterol ratio and the extent of atherosclerotic lesions. The fact that the ratio showed the strongest association with coronary raised lesions is an important finding, particularly since the relationship observed is in keeping with the experience that HDL cholesterol is a negative risk factor for coronary heart disease mortality and morbidity. Thus, the role of HDL cholesterol would seem to be acting at the level of the formation of atherosclerotic lesions rather than at some other point in the pathogenesis of coronary heart disease (CHD).

The absolute value of HDL cholesterol had a poorer predictive value than cholesterol ratio, serum cholesterol, or BP (simple correlation coefficient to RL = 0.25). The reason that the HDL cholesterol ratio appeared as a better predictor of RL than absolute HDL was probably a consequence of its high degree of association with total serum cholesterol. The negative correlation of HDL cholesterol to coronary RL is interesting in view of the hypothesis that HDL may remove cholesterol from the intima and thus decrease the risk of developing atherosclerotic lesions. Clinical studies have also shown negative correlations between HDL and CHD.

Serum cholesterol and systolic BP have about the same predictive value for the development of coronary RL. Our analysis of the multiplicative effects of these two factors, however, suggests that serum cholesterol may be the more important in the synergistic effect. The correlation between coronary RL and systolic BP was clearly and systematically dependent on the level of serum cholesterol; there was no clear trend in the other direction. Total serum cholesterol showed clearer synergistic effects on BP than did cholesterol ratio. Serum triglycerides had little, if any, synergistic effect with BP.

The risk factors measured in this study explain at best only about 25% of the variation in lesions. This leaves a great deal of unexplained residual variation, as has been indicated in previous studies. Better methods of evaluating atherosclerotic lesions and more information on the time course of suspected risk factors might help explain some of this residual variation.

There may be several explanations for those coronary heart disease risk factors that did not show significant correlation with the extent of coronary atherosclerosis in this study. Considering all the limitations of the study, we wonder if the lack of significant correlations may reflect the fact that these are weaker atherogenic risk factors than serum cholesterol levels and BP, and could not be detected as significant because of the small sample size. A larger study might show significant correlations for some of these factors. On the other hand, these risk factors might be related to the development of clinical CHD by mechanisms other than the atherosclerotic lesion, i.e., the coagulation mechanism for the formation of thrombi, or myocardial metabolism.

The lack of association in this study between cigarette smoking and the extent of atherosclerosis is somewhat surprising, since several previous studies have shown a relationship between cigarette smoking and the extent of atherosclerotic lesions in the coronary arteries and aorta. Several epidemiologic studies with autopsy follow-up also have shown a significant relationship between cigarette smoking and aortic atherosclerotic or coronary atherosclerosis. Clinical studies have also shown a relationship between cigarette smoking and aortic, cerebral, and femoral artery atherosclerosis. Garcia-Palmieri et al. found a significant relationship between smoking and aortic, but not coronary, atherosclerosis.

Is there a weak relationship between cigarette smoking and coronary atherosclerosis that this study has failed to detect? Are smoking habits different in Norway than in other parts of the world where a positive relationship has been shown? Or is there really no true relationship between smoking and the basic lesions of atherosclerosis? If cigarette smoking has no real effect on atherosclerotic lesions, the possibility exists that the strong clinical association between cigarette smoking and CHD may be explained by the effect of smoking on the myocardium and its conduction system rather than its effect on basic atherosclerotic lesions. Another possibility is that cigarette smoking may involve the coagulation system and enhance the formation of thrombi or preexisting atherosclerotic lesions. Thus, smoking could interact with coronary raised lesions in the development of CHD in persons who have extensive coronary raised lesions from causes other than cigarette smoking.

Serum triglycerides are less well correlated with atherosclerosis than with clinical CHD. The explanation may be that triglycerides are posi-
tively associated with some coagulation factors, and may influence the formation of thrombi rather than the progression of atherosclerosis.

The degree of physical activity at work and at leisure may appear as significant risk factors for CHD mortality and morbidity. In a multivariate analysis, however, physical activity at leisure was not significant for CHD mortality when taking into account coronary risk score and social class as explanatory variables (Holme et al., unpublished observation). Therefore, the poor partial association between coronary atherosclerosis and physical activity does have its parallel in some clinical data.

Coronary risk score, which has proved to be a most useful “risk factor” for CHD morbidity and mortality, shows only a marginal association to coronary RL. The most obvious reason seems to be that one of its components, cigarette smoking, is not significantly associated with coronary RL in this study. For cerebral atherosclerosis, the association with risk score is highly significant, probably due to the fact that cigarette smoking has a positive (though not significant, $r = 0.09$) association with cerebral RL.

The epidemiology of cerebral atherosclerosis may seem somewhat different from that of coronary atherosclerosis. Blood pressure is slightly, but not significantly, more predictive of cerebral RL than serum cholesterol and other lipid risk factors. This agrees with clinical experience showing that BP is more predictive for stroke than for CHD and that serum cholesterol is less predictive than BP for the development of stroke.

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References


Index Terms: raised atherosclerotic lesions • cerebral arteries • coronary arteries • coronary atherosclerosis • cerebral atherosclerosis • high density lipoprotein cholesterol
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