Relation of Extent of Extracranial Carotid Artery Atherosclerosis as Measured by B-Mode Ultrasound to the Extent of Coronary Atherosclerosis

James L. Wofford, Frederic R. Kahl, George R. Howard, William M. McKinney, James F. Toole, and John R. Crouse III

The extent of carotid artery atherosclerosis as measured by B-mode ultrasound has been shown to be strongly and independently correlated with the presence or absence of coronary atherosclerotic disease (CAD), but no studies to date have used carotid B-mode ultrasound to compare the extent of atherosclerotic disease in the two arterial circulations. We used data from a registry of patients undergoing cardiac catheterization and B-mode ultrasound of the carotid arteries to compare the extent of CAD (number of major coronary vessels with 50% or greater stenosis as judged by a consensus interpretation) with the extent of extracranial carotid atherosclerosis. Four hundred thirty-four patients (234 men, 200 women) greater than 40 years of age were stratified by gender and then divided into quartiles on the basis of a B-mode score that was derived by summing arterial wall thickness at nine sites in the left and nine sites in the right carotid arteries. Evaluation of extent of CAD for the four B-mode quartiles showed that men in the lowest B-mode quartile were over six times more likely to have normal coronary arteries than three- to four-vessel CAD, while men in the highest B-mode quartile were over 10 times more likely to have three- to four-vessel CAD than normal coronary arteries. The findings were similar for women but not as dramatic. Gender-specific discriminant function models using traditional risk factors alone or in combination with B-mode score were developed to predict the extent of CAD. Discriminant models containing traditional risk factors alone performed only slightly better than a model that contained only the B-mode score. The addition of the B-mode score to models of traditional risk factors added little to the predictive ability for CAD extent. (Arteriosclerosis and Thrombosis 1991;11:1786-1794)
CAD may differ from those related to extent of CAD, we evaluated B-mode ultrasound for its ability to predict the extent of CAD.

Methods

Patient Population

Patients for this study were selected from an existing registry of patients undergoing cardiac catheterization at North Carolina Baptist Hospital, as has been described previously. All patients admitted from December 1982 to February 1987 for cardiac catheterization to evaluate chest pain, arrhythmias, valvular heart disease, myocardial infarction, positive treadmill tests, and heart failure were considered eligible for this registry. Patients were excluded if they were less than 25 years of age; if they had a history of liver, kidney, or thyroid disease, alcohol abuse, recent myocardial infarction, previous coronary bypass grafting, or angioplasty; if they were treated with heparin, cortisone, or lipid-lowering drugs; or if they were clinically unstable. Patients were then selected for inclusion in the registry on the basis of a stratified random sampling strategy designed to admit men and women above and below the age of 50 with and without CAD comparably. Additional exclusion criteria for the present study were history of significant aortic valvular disease or previous endarterectomy. Exclusion of patients with aortic valvular disease was based on our earlier findings that suggested an independent association of aortic valve disease with carotid disease (unpublished results from our laboratory).

Interview of Patients and Risk Factor Determination

Informed consent was obtained for each patient before they were interviewed. On the night before cardiac catheterization, each subject underwent a standardized interview that emphasized cardiovascular risk factors. The presence of hypertension was defined by history of the diagnosis or a systolic blood pressure greater than 150 mm Hg or diastolic blood pressure greater than 90 mm Hg. The presence of diabetes mellitus was defined by history of the diagnosis or a fasting glucose level greater than 140 mg/dl. Smoking status was recorded as the number of pack-years smoked. A family history of CAD was defined as a history of myocardial infarction or CAD in any first-degree relative less than 65 years old.

Plasma was collected from subjects on the morning before cardiac catheterization. Lipid profile determinations were performed in a Centers for Disease Control standardized laboratory. Total cholesterol and triglycerides were measured according to Lipid Research Clinics procedures using the Technicon Auto Analyzer. High density lipoprotein (HDL) cholesterol was measured as described in the manual of operations of the Lipid Research Clinics Program after precipitation with heparin-manganese. Plasma low density lipoprotein (LDL) cholesterol was measured using Lipid Research Clinics methodology after ultracentrifugation of plasma at $d=1.006 \text{ g/ml}$ to float very low density lipoproteins (VLDLs) and chylomicrons. LDL cholesterol was calculated as the difference between the total $d=1.006 \text{ g/ml}$ infranate cholesterol and infranate HDL cholesterol.

Measurement of Extent of Coronary Atherosclerosis

Coronary angiography was performed by the percutaneous technique using either Judkins or multipurpose catheters. The extent of CAD was coded as 0, 1, 2, 3, or 4 according to the number of major coronary vessels (left anterior descending, left circumflex, right coronary artery, or left main coronary) with an obstruction of 50% or greater. Patients with nonobstructive CAD (percent stenosis less than 50%) were excluded from analysis because Fried and Pearson have shown that such patients in fact have extensive disease and are difficult to categorize. In addition, Brown et al have shown that assessment of coronary stenosis of 30-50% is highly variable and poorly reproducible among observers. Patients were considered to have normal coronary anatomy if no evidence of luminal irregularities was noted by the angiographer. A 50% or greater obstruction of vessels other than the four major epicardial arteries was counted as obstruction of the major vessel from which it arose.

For each patient, the hospital chart provided two independent interpretations of the cardiac catheterization, one by the patient's attending cardiologist and one by an experienced radiologist. For cases in which there was disagreement between the two readers on the number or location of stenosed vessels, a third blinded reading of the coronary angiogram was performed by a different staff cardiologist. Agreement between two of the three readings was accepted as the true measure of CAD extent. Cases for which there was no agreement among the three interpretations were excluded from further analysis.

Measurement of Extent of Carotid Atherosclerosis

B-mode ultrasound of the carotid arteries was performed by trained technicians in the clinical ultrasound laboratory, as previously described. With the patient in a sitting position, echogenic lesions at six sites in both carotid arteries (low common carotid, below bifurcation, and above bifurcation) were quantified in the anterior oblique, lateral, and posterior planes using a Biosound compact real-time (B-mode) imager with an 8-MHz sector scanner probe and digital scan converter. Ultrasound technicians were unaware of the results of the cardiac catheterization. A scoring system derived from summing the maximal thicknesses of echogenic lesions at the near and far walls of each of the three imaging sites in each carotid system yielded a measure of the extent of carotid atherosclerosis in millimeters. The reliability of this scoring system has been documented, with a correlation coefficient for repeat readings of $r=0.80$. Recently 128 randomly selected videotapes from this project have been reread according to the
protocol developed for the Arteriosclerosis Risk in Communities Study (ARIC, Drs. Ralph Barnes and Ward Riley). The correlation coefficient for our “summary” score versus the ARIC “sum” was \( r=0.82 \), and the coefficient for our score versus the ARIC “mean” was \( r=0.79 \) (unpublished observations from our laboratory).

Statistical Analysis

Because few patients had four-vessel CAD, three- and four-vessel disease were combined for the purposes of analysis. This provided a four-classification CAD score for comparison: normal, one-vessel disease, two-vessel disease, and three- and four-vessel disease. All analyses were performed for men and women separately. Proportions and means were calculated for the dichotomous variables (hypertension, diabetes mellitus, family history of premature CAD, and race) and continuous variables (total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, pack-years smoked, age, and B-mode score) to describe each CAD group.

The univariate relation between B-mode score and extent of CAD was assessed by dividing patients into quartiles on the basis of B-mode score. Frequency distributions of the CAD score were compared for each of the four B-mode quartiles. Analysis of covariance was used to test for the association of B-mode score with CAD score controlling for age and was used to calculate age-adjusted CAD scores for each quartile.

To establish the usefulness of the traditional risk factors and B-mode score in predicting the extent of CAD, discriminant function models were developed for each gender using CAD score as the outcome variable and the following variables as predictors: 1) B-mode score alone, 2) risk factors alone, and 3) B-mode score plus risk factors. Multivariate analysis was performed with the STEPDISC and DISC procedure in SAS. A random two-thirds subset of the gender-specific sample (learning set) was then tested on the remaining one-third of the gender-specific sample (validation set). The unweighted percent correctly classified was computed for each of the six models against that of chance at one.

To obtain a consensus agreement on the extent of CAD, chart review of these 320 patients was performed. Either one or both of the two interpretations of the angiogram was not obtainable from hospital records of 14 patients. Thirty-four percent (105 of 306) of patient charts showed a discrepancy in interpretation between the first cardiologist and radiologist as regards presence or absence of 50% or greater obstruction disease or vessel involved. For nine patients with discrepant first readings, the angiogram was not available for a third interpretation. For 10 patients (10 of 297, or 3.4%), there was no agreement on CAD extent despite three independent readings.

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Results

Of the 843 patients entered in the registry, 73% underwent coronary angiography for evaluation of chest pain; 20% for valvular heart disease; and 7% for arrhythmias, heart failure, and other problems. Figure 1 shows the schema for patient selection. B-mode ultrasound scans of the carotid arteries were available for 566 patients whose hospital stay permitted the investigators to obtain this test. Forty-seven patients were excluded because of aortic valve disease and two because of previous carotid endarterectomy. Of the remaining 517 patients, 468 were older than 40 years of age. Of these patients over 40 years of age who had undergone cardiac catheterization and B-mode ultrasound of the carotid arteries, 320 had at least one 50% or greater stenosis of a major coronary artery, as previously documented in the registry.

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In one case rereading of the angiogram resulted in classification of the angiogram as one without any significant stenoses in any major vessel. After exclusion of the above, the remaining 286 patients were compared with 148 registry patients over 40 years old who had normal coronary anatomy.

Dichotomous variables for the 234 men and 200 women stratified according to CAD extent are presented in Table 1. Mean values (±1 SD) are shown in Table 2. In general, levels of risk factors increased (decreased in the case of HDL cholesterol) with increasing CAD extent. Extent of coronary atherosclerosis increased with increasing CAD extent for both men and women.

B-mode scores ranged from 0 to 25.6 mm for men and 0 to 21.3 mm for women. B-mode quartiles were established, and mean CAD scores (±1 SD) for each quartile were computed (Table 3). Analysis of covariance showed that B-mode score was still significantly correlated with CAD extent after controlling for age (\( p<0.001 \)). Age adjustment did not change the mean number of vessels with stenosis for each B-mode quartile.

Figures 2 and 3 show the distribution of CAD extent for each B-mode quartile. Men with B-mode scores in the lowest quartile were over six times more likely to have normal coronary arteries than three- or four-vessel disease. Men with B-mode scores in the highest quartile were more than 10 times more likely to have three- or four-vessel disease than normal coronary arteries. The trends of decreasing likelihood of normal coronary status and increasing likelihood of three- or four-vessel disease with increasing B-mode score are present in both sexes but are less dramatic in women than in men.
The results of the forward selection stepwise discriminant function procedures presented in Tables 4 and 5 show univariate (step one) and multivariate (last step) relations between risk factors and CAD extent. B-mode score shows the strongest univariate predictive value for CAD extent for both genders.

When B-mode score is not included as a predictor variable, stepwise selection shows age to be a significant predictor of CAD extent for both genders. Total and HDL cholesterol remain in the model for men, whereas smoking and diabetes remain in the model for women. The addition of B-mode score to the group of predictor variables in the stepwise selection procedure removes the predictive value of age in both genders. For women, the predictive value of diabetes and smoking was reduced with addition of B-mode score, whereas LDL cholesterol gained predictive value. For men, total cholesterol and HDL cholesterol retained predictive value, although slightly reduced.

Discriminant analysis performed on the validation set showed significant evidence \( (p < 0.001) \) of heterogeneity in the covariance matrixes across the four groups. As such, classifications were based on the relative heights of the four density functions, each estimated using within-groups covariance matrixes. Because the pooled covariance matrix was not used, it was not possible to estimate the standard discriminant function coefficients.

Classification matrixes generated by discriminant function applied to the gender-specific validation sets are shown in Tables 6 and 7. The percentage accurately classified and \( \kappa \) statistic for models derived from the learning set but tested on the validation set are compared in Table 8. B-mode score alone performs almost as well as models with

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**TABLE 1. Characteristics of Coronary Artery Disease Extent Groups by Gender (Percentages for Dichotomous Variables)**

<table>
<thead>
<tr>
<th>CAD extent*</th>
<th>Women</th>
<th>Men</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>87</td>
<td>61</td>
</tr>
<tr>
<td>1</td>
<td>48</td>
<td>36</td>
</tr>
<tr>
<td>2</td>
<td>24</td>
<td>20</td>
</tr>
<tr>
<td>3</td>
<td>21</td>
<td>17</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>200</td>
<td>234</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DM</th>
<th>HTN</th>
<th>Family history</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonwhite</td>
<td>5.7</td>
<td>8.2</td>
</tr>
<tr>
<td>DM</td>
<td>5.8</td>
<td>16.7</td>
</tr>
<tr>
<td>HTN</td>
<td>51.7</td>
<td>41.7</td>
</tr>
<tr>
<td>Family history</td>
<td>36.8</td>
<td>36.1</td>
</tr>
</tbody>
</table>

*Coronary artery disease

†Aortic valve disease, Status post carotid endarterectomy

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**FIGURE 1. Schematic diagram of patient selection.**

CAD, coronary artery disease; YO, years old.
### Table 2. Characteristics of Coronary Artery Disease Extent Groups by Gender (Means for Continuous Variables)

<table>
<thead>
<tr>
<th>CAD extent</th>
<th>Women</th>
<th>Men</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Patients</td>
<td>Age (yr)</td>
</tr>
<tr>
<td>0</td>
<td>87</td>
<td>55.7±8.01</td>
</tr>
<tr>
<td>1</td>
<td>48</td>
<td>55.6±10.1</td>
</tr>
<tr>
<td>2</td>
<td>24</td>
<td>58.7±10.3</td>
</tr>
<tr>
<td>3</td>
<td>41</td>
<td>58.4±9.7</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>...</td>
</tr>
</tbody>
</table>

**CAD**, coronary artery disease; **HDL**, high density lipoprotein; **LDL**, low density lipoprotein; **TG**, triglycerides. Values for continuous variables are mean±1 SD.

### Discussion

B-mode ultrasound has proven to be a useful noninvasive means of quantitatively assessing the amount of atherosclerosis in the carotid arterial system. The advantage of B-mode ultrasound lies in its ability to image atherosclerosis within the arterial wall rather than the lumen of the artery. Its acceptance by medical researchers as a continuous measure of localized atherosclerosis is reflected in its use in ongoing cohort studies and clinical trials.

Although systemic atherosclerosis may have serious clinical effects in the carotid circulation, its effects on the coronary circulation are unquestionably more prevalent and life threatening. The mortality from cardiac disease, the vast majority of which results from coronary atherosclerosis, is over five times that from cerebrovascular disease. In addition, patients with carotid artery disease more often die from coronary atherosclerosis than from complications of carotid atherosclerosis. Thus, the relation of measurements of carotid atherosclerosis to the extent of disease in the coronary circulation may be more important than its use in measuring local disease.

We have previously shown a significant independent association between the extent of carotid atherosclerosis measured by B-mode ultrasound and the presence or absence of coronary atherosclerosis documented by coronary angiography. In multivariate modeling that included age, hypertension, smoking, lipids, body mass index, family history, and left ventricular hypertrophy as predictor variables, B-mode score was the variable most predictive of the presence of CAD. The present study further examined the relation between carotid and coronary disease in this same population, now characterized according to extent of CAD rather than its presence or absence.

Our previous study showed a strong relation between extent of carotid disease and presence or absence of CAD in both men and women. The results of the present study suggest that extent of carotid disease is related to CAD extent for both genders, but the relation is more pronounced in men than in women. In contrast, the predictive power of B-mode score for CAD extent is comparable to that of men.

### Table 3. Range of B-Mode Score and Mean Coronary Artery Disease Score for B-Mode Quartiles

<table>
<thead>
<tr>
<th>Quartiles</th>
<th>Women</th>
<th>Men</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Range of B-mode score (mm)</td>
<td>0–1.40</td>
</tr>
<tr>
<td></td>
<td>CAD score* (mean±1 SD)</td>
<td>1.5±0.9</td>
</tr>
<tr>
<td>2</td>
<td>Range of B-mode score (mm)</td>
<td>1.41–3.95</td>
</tr>
<tr>
<td></td>
<td>CAD score* (mean±1 SD)</td>
<td>1.9±1.1</td>
</tr>
<tr>
<td>3</td>
<td>Range of B-mode score (mm)</td>
<td>3.96–8.45</td>
</tr>
<tr>
<td></td>
<td>CAD score* (mean±1 SD)</td>
<td>2.1±1.1</td>
</tr>
<tr>
<td>4</td>
<td>Range of B-mode score (mm)</td>
<td>8.46–21.30</td>
</tr>
<tr>
<td></td>
<td>CAD score* (mean±1 SD)</td>
<td>2.8±1.1</td>
</tr>
</tbody>
</table>

**CAD**, coronary artery disease.

*Number of major coronary vessels with 50% or greater stenosis.
of traditional risk factors for women but less than that of traditional risk factors for men.

In both univariate and multivariate analysis B-mode score has stronger predictive value for CAD extent than any of the risk factors examined. Risk factors collected at the time of catheterization represent a single, static view of a constantly changing exposure of the coronary arteries to those factors and are poor predictors of CAD extent. Duration of the exposure to risk factors is generally difficult to estimate. B-mode score may estimate the aggregate effect of a multitude of risk factors over time.

In approximately one half of the cases, the models were able to predict accurately the number of coronary arteries with 50% or greater stenosis of lumen diameter in this population of patients with symptomatic cardiac disease. However, the lack of improvement in predictive accuracy when B-mode score was added to the risk factor models was disappointing. As strong as B-mode ultrasound appeared in the stepwise analyses, it actually added little predictive value in the final models. Thus, B-mode score alone appears no better and no worse than an aggregate of several factors.

In autopsy studies, the relation between the amount of atherosclerosis in the cerebral and the coronary circulations has been suggestive but not convincing. However, in vivo evidence points to the association of atherosclerotic involvement in the two arterial circulations. First, most patients with measurable carotid disease or symptoms of carotid disease die of complications of coronary atherosclerosis, not of stroke. Second, there are shared risk factors for development of disease in the two arterial locations. Studies by Crouse et al and Salonen have demonstrated that several established coronary risk factors (age, HDL cholesterol, hypertension, LDL cholesterol, cigarette smoking) are associated with carotid disease as well. Third, patients with documented cerebrovascular disease have measurable disease in the coronary circulation. Hertzer and Young showed that of 506 patients with cerebrovascular symptoms or carotid bruits who were undergoing elective carotid endarterectomy, 35% had at least a 70% obstruction of at least one coronary artery by angiography. Our study offers yet another line of evidence of an in vivo relation between the amount of disease in the two circulations.

Limitations of our study include potential selection bias, the use of two imperfect technologies for measuring extent of atherosclerotic disease, and the use of discriminant analysis in a nonnormal (Gaussian) environment. In addition to the well-documented selection bias inherent in studies of patients under-
TABLE 6. Classification Matrix for Women Using B-Mode Score, Diabetes Mellitus, and Low Density Lipoprotein Cholesterol

<table>
<thead>
<tr>
<th>Actual CAD extent</th>
<th>Predicted CAD extent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>0</td>
<td>26</td>
</tr>
<tr>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>3-4</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>34</td>
</tr>
</tbody>
</table>

Percent correctly classified, 37 of 69, or 54%. \( \kappa = 0.28 \) \( (p=0.0004) \), \( n=69 \). CAD, coronary artery disease.

13% of the population. The use of a consensus reading to determine the number of vessels with a greater than 50% stenosis similar to the method in our study has achieved a reliability of as high as 90%.

Comparison of multiple interpretations of the coronary angiogram eliminated clerical errors and resolved disagreement on borderline lesions. However, in 3.4% of the patients no consensus could be reached despite three interpretations of the angiogram, thus emphasizing the interobserver variability in interpreting the angiogram.

Computer-assisted angiography may improve reproducibility of estimates of lumen obstruction, but more serious concerns arise with this method. Fried and Pearson have explored the implications, from the standpoint of epidemiology, of inclusion of patients with nonobstructive disease within the control group of case-control studies that have arteriosclerosis as the outcome variable. In addition, autopsy studies have called into question the validity of the method, noting consistent underestimation of atherosclerosis from lumen dimensions.

Despite the validation of B-mode ultrasound against tissue specimens, its demonstrated reliability, and its growing use in clinical trials and large epidemiological studies, the approach to quantification of disease extent is evolving. The scoring system on which this study is based is now being used in many years and was modeled after scoring systems used to quantify extent of coronary disease.

This system has been evaluated for its reliability. This approach emphasizes quantification...
of diseased segments over normal segments. More recently, investigators have relied on indexes of disease extent that included normal segments. Measures of normal walls and wall thickening either at a single site or at multiple sites ultimately may better reflect the extent of local disease or the total atherosclerotic burden for an individual. Our approach correlates well with one such measure of the extent of local disease that includes normal walls, but final adjudication as to which approach is best will await further experience with B-mode ultrasound.

Finally, the use of discriminant analysis for evaluating the use of multiple predictor variables with a polychotomous outcome variable was not without problems. Notably, discriminant analysis assumes a multivariate normal distribution among the independent (risk factors) variables, which is clearly not met in our case where some variables are dichotomous. However, the use of a stepwise procedure to identify the optimal subset of predictor variables in a learning set and the subsequent evaluation of accuracy in an independent validation set allows for the most conservative evaluation of the performance in the presence of the violations of the statistical assumptions.

We used the \( \kappa \) statistic to evaluate agreement between the predicted and true CAD categories. The \( \kappa \) statistic has frequently been used to evaluate observer agreement where a \( \kappa \) statistic of less than 0.40 typically reflects poor interrater agreement. That our \( \kappa \) values are less than this level should not be interpreted with the same strict criteria, since we are using \( \kappa \) to 1) describe the relative level of predictive power in risk factor–B-mode score models and 2) test if there is statistical evidence of predictive power (test the null hypothesis that there is no agreement between predicted and observed CAD classes beyond chance alone (\( H_0: \kappa = 0 \)).

The net effect of all the factors described above would be to weaken any real associations and to bias the observed relations toward the null. That strong correlations exist between extent of extracranial carotid atherosclerosis and extent of coronary disease in spite of these limitations attests to the validity of the associations.

In summary, our study suggests a strong relation between the extent of carotid atherosclerosis, as measured by B-mode ultrasound, and the extent of coronary atherosclerosis, as measured by coronary angiography. Although B-mode ultrasound appears to be as useful as traditional risk factors in predicting the extent of CAD, the addition of B-mode ultrasound of the carotid adds little to the predictive ability of already recognized risk factors for the extent of CAD.

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KEY WORDS • carotid atherosclerosis • coronary atherosclerosis • B-mode ultrasound
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