HHS Public Access

Author manuscript

J Am Soc Echocardiogr. Author manuscript; available in PMC 2015 October 02.

Published in final edited form as:

J Am Soc Echocardiogr. 2008 October ; 21(10): 1156–1162. doi:10.1016/j.echo.2008.05.001.

Ultrasound Detection of Increased Carotid Intima-Media Thickness and Carotid Plaque in an Office Practice Setting: Does It Affect Physician Behavior or Patient Motivation?

Claudia E. Korcarz, DVM, FASE, **Jeanne M. DeCara, MD**, **Alan T. Hirsch, MD**, **Emile R. Mohler, MD**, **Bryan Pogue, MD**, **John Postley, MD**, **Wendy S. Tzou, MD**, and **James H. Stein, MD, FASE**

University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin (C.E.K., J.H.S.); the University of Chicago Pritzker School of Medicine, Chicago, Illinois (J.M.D.); the University of Minnesota School of Public Health and Minneapolis Heart Institute Foundation, Minneapolis, Minnesota (A.T.H.); the University of Pennsylvania Medical School, Philadelphia, Pennsylvania (E.R.M., W.S.T.); The Heart Prevention Clinic of Idaho, Boise, Idaho (B.P.); and New York Physicians, New York, New York (J.P.)

Abstract

Background—The aim of this multicenter study was to determine if identifying increased carotid intima-media thickness (CIMT) or carotid plaque during office-based ultrasound screening examinations could alter physicians' treatment plans and patients' motivation regarding health-related behaviors.

Methods—Carotid ultrasound studies were performed by a nonsonographer clinician using a handheld system. Changes in physicians' treatment plans and patients' motivation on the basis of scan results were analyzed using multivariate regression.

Results—There were 253 subjects (mean age, 58.1 ± 6.6 years). When increased CIMT or carotid plaque was detected, physicians were more likely to prescribe aspirin and lipid-lowering therapy ($P < .001$). Subjects were more likely to report increases in plans to take cholesterol-lowering medication ($P = .002$) and the perceived likelihood of having or developing heart disease ($P = .004$).

Conclusions—Findings from office-based carotid ultrasound studies can influence physicians' prescriptions of evidence-based interventions. Patients with abnormal ultrasound findings recognize their increased cardiovascular risk and plan to take cholesterol-lowering medication.

Keywords

Atherosclerosis; Carotid arteries; Risk factors; Ultrasound

Although the vast majority of patients who develop cardiovascular disease (CVD) have traditional risk factors, many patients with risk factors do not develop CVD, so decisions

Reprint requests: James H. Stein, MD, G7/341 CSC, MC 3248, 600 Highland Avenue, Madison, WI 53792 jhs@medicine.wisc.edu.
0894-7317/$34.00.

about the aggressiveness of risk factor intervention are not always straightforward.[1] Indeed, adults who are at "intermediate risk" represent a subgroup of patients for whom treatment decisions may not be clear, and they are the individuals most likely to benefit from screening tests that independently predict future CVD events.[2-5] On the basis of these observations, a simple, noninvasive, and inexpensive screening tool to improve CVD risk prediction and to help target the aggressiveness of preventive therapies toward those at greatest risk would be a useful clinical tool.[2,3]

Increased carotid intima-media thickness (CIMT) is a validated marker of subclinical vascular disease that predicts future risk for cardiac death, myocardial infarction, and stroke, independent of traditional CVD risk factors.[3-6] Also, the presence of carotid plaques independently predicts adverse CVD outcomes and total mortality.[7,8] These markers of increased CVD risk can be detected noninvasively by ultrasound, but their use in clinical practice has been limited. Some of the barriers to the more widespread use of these methodologies include the expense of the instrumentation, technical challenges with image acquisition and interpretation that require highly trained and expensive sonographic technicians, and the absence of evidence that patient outcomes are improved by the use of these tests. The Office Practice Assessment of Carotid Atherosclerosis (OPACA) study demonstrated that some of these barriers could be overcome by training nonsonographer clinicians (NSCs) to obtain images of the carotid arteries using handheld ultrasound (HHU) systems, measure CIMT, and identify findings indicating increased CVD risk.[9] It is not known, however, if patient outcomes are improved when subclinical vascular disease is identified. This phase of the OPACA study investigated whether finding increased CIMT or carotid plaque during office-based ultrasound screening could alter physicians' treatment plans and patients' motivation regarding health-related behaviors.

Methods

Study Protocol

The institutional review boards at the University of Wisconsin School of Medicine and Public Health and each participating site approved this study. Each NSC and each research subject provided informed consent. This study was performed at 5 clinical sites. Ultrasound data were collected by 8 NSCs who completed a 2-day training program taught by University of Wisconsin Atherosclerosis Imaging Research Program, the core laboratory and coordinating center for this study.[9]

All participants were men aged ≥ 45 years or women aged ≥ 55 years with ≥ 1 additional CVD risk factor, including current cigarette smoking, diabetes mellitus, hypertension (systolic blood pressure ≥ 140 mm Hg or taking antihypertensive medication), dyslipidemia (low-density lipoprotein [LDL] cholesterol ≥ 130 mg/dL or high-density lipoprotein cholesterol < 40 mg/dL), or a family history of premature CVD (male first-degree relative aged < 55 years or female first-degree relative aged < 65 years). Women aged 45 to 54 years could participate if they had family histories of CVD and ≥ 1 additional CVD risk factor.

Exclusion criteria were age >70 years, the use of cholesterol-lowering medications, uncontrolled hypertension, coronary artery disease, cerebrovascular disease, and peripheral

arterial disease. Subjects with kidney, liver, or thyroid disease were also excluded. Fasting laboratory tests were performed in local laboratories.

Potential subjects were invited to participate after routine office visits. Some sites advertised for the study. Subjects completed a pretest survey to assess motivation and intention to change lifestyle behavior. Their physicians' initial plans of action regarding the use of antiplatelet, lipid-lowering, and antihypertensive medications, as well as need for further testing, on the basis of their standard medical practice, were recorded. After the subjects completed the pretest survey, they underwent carotid ultrasound studies to identify carotid plaque and measure CIMT. Physicians then had the opportunity to modify their treatment plans on the basis of the scan results. The physicians' scan-based plans of action were recorded, and the subjects were informed of the test results and their physicians' recommendations. Subjects were told that there is a strong association between carotid artery disease and coronary artery disease and that if they had plaque or increased CIMT, their risks for heart attack, stroke, and death were increased. After this discussion, subjects completed a posttest survey that was identical to the pretest survey.

Carotid Ultrasound Imaging

Imaging was performed by NSCs using an HHU device (MicroMaxx; SonoSite, Inc., Bothell, WA) with an L38 linear-array transducer, as previously described.[9] The NSCs included 3 physicians, 2 registered nurses, 2 medical assistants, and 1 emergency medical technician.[9] Intrascanner and interscanner variability for the NSCs in this study were low and have been described previously.[9] Carotid plaque was defined as a focal echogenic thickening of the intimal reflection that encroached on the arterial lumen, with a minimal intimal plus medial thickness ≥ 1.2 mm, or an area of focal thickening at least twice the adjacent region.[8,10]

Electrocardiographically gated end-diastolic images of the distal 1 cm of the far wall of each common carotid artery were obtained in triplicate from 3 different angles of incidence and stored digitally. These images were used for the offline measurement of CIMT using a semiautomated border-detection program (SonoCalc version 3.0; SonoSite, Inc.).[11] The scanning procedure took, on average, 20 minutes to perform, ranging from 15 to 30 minutes depending on the NSC. All scanners and readers were certified; the reproducibility of CIMT measurements and plaque detection in this study has been previously reported.[9] Physicians were informed that the presence of carotid plaque or a mean common carotid artery CIMT ≥ 75th percentile for age, sex, and race signified increased CVD risk.[12]

Assessment of Patient Motivation

Subjects completed identical surveys before and after the ultrasound examinations. The surveys were designed using the theory of planned behavior, that an individual's intention to perform a behavior is determined by the attitude toward the behavior, the subjective norm, and the individual's perception of control he or she has over his or her behavior.[13,14] The survey instrument used a 7-point, bipolar Likert-type scale and contained 15 questions assessing subjects' (1) motivation to exercise, (2) motivation to make specific dietary changes, (3) motivation to quit smoking (smokers only), (4) likelihood that they would use

medications or lifestyle changes to reduce cholesterol and blood pressure, (5) perceptions of the impacts that high cholesterol and blood pressure have on their health, and (6) likelihood that they had or would develop CVD.[15] Survey response options ranged from “unlikely” or “strongly disagree” (score = 1) to “highly likely” or “strongly agree” (score = 7).

Statistical Techniques

SigmaStat version 3.0.1 (SPSS, Inc., Chicago, IL) and SAS version 8.0 software (SAS Institute Inc., Cary, NC) were used for descriptive and comparative statistics. Continuous variables are described as mean ± SD or as median (range). Student's *t* test was used to identify differences in continuous variables among subjects with and without carotid plaque or with CIMT below or above the 75th percentile. The χ^2 test was used to identify differences among categorical variables.

Changes in clinical management were analyzed using a hierarchical logit model with treatment change as a binary response, comparing postscan treatment (1 = increase over prescan, 0 = no change or negative change) nested by site. Differences among the sites were tested by evaluating the variability (σ^2) in management changes among sites. To this model, we added age, sex, race, body mass index, LDL cholesterol, systolic blood pressure, diastolic blood pressure, and ultrasound findings of carotid plaque presence and/or increased CIMT. A 2-way analysis of variance was used to evaluate differences in referral for additional testing. Each model was corrected using the Holm-Sidak method for multiple comparisons.

Survey results before and after testing were compared as posttest minus pretest Δ scores for each survey item using paired *t* tests with Bonferroni's correction for multiple testing ($P = .05$/n questions). Only comparisons with *P* values ≤ .004 were considered to indicate rejection of the null hypothesis that the Δ score with normal scan results was equal to the Δ score with abnormal scan results. Changes in survey results were analyzed as continuous variables in a multiple linear regression model with terms that included carotid plaque presence and/or increased CIMT, as well as age, sex, race, body mass index, LDL cholesterol, systolic blood pressure, diastolic blood pressure, and study site. Interaction terms between the carotid ultrasound results and age, sex, body mass index, and smoking status were introduced one at a time. Survey responses to each item showed significant variability among subjects but equal variance among sites.

Results

Subject Characteristics

There were 268 subjects, but data from 5 subjects were eliminated prospectively because of abnormal thyroid function (n = 2) and not meeting entry criteria for age or risk factors (n = 3). The 263 subjects are described in Table 1. Their mean age was 58.1 ± 6.6 years, and 128 of the subjects were men (49%). The mean 10-year Framingham CVD risk was 6.1 ± 5.2%. There were 158 subjects (60%) who reported histories of hyperlipidemia, but none were receiving lipid-lowering therapy. Histories of hypertension were reported in 44% of the

subjects, of whom 35% were being treated. Only 24 subjects (9%) had diabetes mellitus, and 18 (7%) were receiving diabetes medications.

There were 154 subjects (58.6%) with CIMT ≥ 75th percentile for age, sex, and race. Patients with increased CIMT were younger (56.7 vs 60.2 years; $P < .001$) and had higher systolic blood pressure (131.3 vs 126.4 mm Hg; $P = .030$). They also tended to be more likely to report histories of hypertension ($P = .059$), family histories of coronary heart disease ($P = .081$), and histories of dyslipidemia ($P = .096$) and to have higher waist circumferences ($P = .073$) and body mass index values ($P = .076$). Carotid plaque was present in 153 (58%) subjects. Patients with carotid plaque were older (59.3 vs 56.5 years; $P < .001$) and had higher total cholesterol (214.5 vs 202.2 mg/dL; $P = .016$) and LDL cholesterol (130.0 vs 119.7 mg/dL; $P = .027$) values. Accordingly, they had higher Framingham CVD risk scores (7.3% vs 4.7%; $P < .001$). Subjects with carotid plaque also had a higher mean right and left CIMT ($P < .001$). Differences in other variables in Table 1 were not observed.

Impact of Screening Results on Physicians' Treatment Plans

Changes in physicians' treatment recommendations are described in Table 2. When increased CIMT or carotid plaque was detected, physicians were more likely to prescribe aspirin (odds ratio, 6.34 and 4.84, respectively; $P < .001$ for each) and lipid-lowering therapy (odds ratio, 2.93 and 7.40; $P < .001$ for each). The prescription of antihypertensive medication did not change significantly ($P > .40$ for each).

In multivariate models, increased CIMT ($P < .001$) and carotid plaque presence ($P < .001$) predicted the prescription of antiplatelet therapy, with significant contributions of systolic ($P = .005$) and diastolic ($P = .003$) blood pressure. When increased CIMT and carotid plaque were present, only systolic blood pressure significantly predicted the addition of antihypertensive therapy ($P = .040$ and $P = .024$, respectively). Increased CIMT and carotid plaque presence both predicted the prescription of lipid-lowering medication ($P < .001$ for both). When increased CIMT and carotid plaque were present, only LDL cholesterol significantly predicted the addition of lipid-lowering medication ($P < .001$ for both). There was no heterogeneity across sites, indicating that the relationships between the carotid ultrasound findings and prescription of these therapies were not affected by study site.

Further testing was recommended in 37 subjects (14.1%). Recommended studies included treadmill stress testing (n = 22 [8.4%]), carotid duplex ultrasonography (n = 10 [3.8%]), glucose tolerance testing (n = 2 [< 1%]), ambulatory blood pressure monitoring (n = 1 [<1%]), ankle-brachial index (n = 1 [<1%]), and cardiac catheterization (n = 2 [<1%], both in patients with cardiomyopathy). Recommendations for further testing were predicted by increased CIMT ($P = .044$) and LDL cholesterol ($P = .045$). Although there only was a trend for differences among the sites in the prevalence of abnormal ultrasound findings ($F = 3.40$; $P = .053$), there was a significant difference among the sites in the frequency at which additional tests were ordered ($F = 5.46$; $P = .010$). Differences were attributable to 2 sites that, after the carotid ultrasound studies, ordered more stress tests than the other 3 sites ($P = .01$).

Impact of Plaque Screening on Patients' Motivation

As shown in Table 3, the process of screening appeared to increase subjects' motivation to exercise and make dietary changes, with significant ($P = .002$) changes noted on all 6 questions addressing lifestyle changes. Additionally, screening was associated with subjects' planning to take cholesterol medications ($P < .001$) and having an increased perceived likelihood of both having and developing heart disease ($P = .005$). When the subjects with abnormal scan results were compared with those who did not have plaque or increased CIMT, however, differences were identified. Even subjects without ultrasound abnormalities reported increased motivation to exercise ($P = .003$), and they tended to be more motivated to eat more fiber-containing foods ($P = .051$); however, they did not report an increased perceived likelihood of having and developing heart disease, indicating that they understood the meaning of the carotid ultrasound findings. Subjects with increased CIMT or plaque were more likely than individuals without abnormalities to report increases in plans to take cholesterol-lowering medication ($P = .002$), increased perceived likelihood of having heart disease ($P = .004$), and increased perceived likelihood of developing heart disease ($P < .001$).

Subjects with increased body mass index values and carotid plaque reported increased motivation to limit their dietary intake of sugars, sweetened drinks, juices, and starches. Subjects with increased body mass index values and ultrasound abnormalities reported increased motivation to reduce their dietary intake of saturated and trans fats. The effect of having carotid plaque on the perception of having heart disease was somewhat stronger in men ($P = .090$) and in younger subjects ($P = .071$). Because there were so few smokers, questions regarding motivation to stop smoking could not be evaluated meaningfully. Only 40 of 45 smokers completed the surveys, and none of the smoking-related questions showed significant changes after the scans.

Discussion

A previous publication from the OPACA study demonstrated that NSCs could be trained to use an HHU device and a border detection program to accurately identify carotid plaque and increased CIMT.[9] The associations between these markers of increased CVD risk are independent and strong, with relative risks for future CVD events in the range of 2 to 4 when carotid plaque is present[7,16-18] or when CIMT is elevated.[16,19-22] A recent clinical trial demonstrated that middle-aged adults at apparently low CVD risk who had increased CIMT had reduced rates of atherosclerosis progression from statin therapy that they otherwise would not have qualified for on the basis of current treatment guidelines.[23] The magnitude of the difference in CIMT progression rates (–0.145 mm/yr) was similar to that observed in secondary prevention trials that were associated with a 52% reduction in the odds of future CVD events.[24] These data and the consensus of several expert panels support the use of carotid ultrasound to assist with risk prediction in appropriately selected patients.[2-5]

This study adds to the growing body of literature demonstrating that carotid ultrasound is a feasible screening test and that it can be performed in an office setting.[9,15] This study was the first to assess the effects of carotid ultrasound screening on patients' and physicians'

behavior in a multicenter setting. It was unique in that it was performed in an office practice setting, with images obtained by NSCs. Its relatively large size permitted the more detailed evaluation of subjects' responses to survey questions.

This study demonstrated that after detecting increased CIMT or carotid plaque, physicians were more likely to prescribe evidence-based, CVD risk-reducing interventions such as aspirin and lipid-lowering therapy. These findings are concordant with those of a small study of plaque screening in a cardiology clinic[15] and 2 studies in which patients with higher coronary artery calcium scores were more likely to begin taking aspirin, lipid-lowering medications, or antihypertensive therapy.[25,26] This is an important finding given the well-documented undertreatment of risk factors in the United States.[27,28]

Interestingly, the very act of screening increased patients' stated motivation to exercise and make dietary changes. Although a small study suggested that carotid plaque screening improved smoking cessation rates,[29] evidence that atherosclerosis screening affects patients' motivation or reduces long-term CVD risk is lacking. Indeed, a recent study demonstrated that long-term changes in health-related behaviors were related to case management, not the results of coronary calcium screening.[30] In our study, even subjects without abnormalities on their scans reported increased motivation to exercise and to eat more fiber. Those with ultrasound abnormalities were more likely to report increases in plans to take lipid-lowering medication, as well as increased perceived likelihood of having and developing heart disease. These findings suggest that screening per se affected the reported motivation, but more important, they suggest that both physicians and patients understand the implications of the findings of carotid ultrasound studies with regard to cardiovascular risk and the importance of risk-reducing interventions. If patients are more likely to take cholesterol-lowering medications and physicians are more likely to prescribe them when subclinical vascular disease is identified, presumably CVD risk will be reduced; however, a long-term outcomes study is needed to test this hypothesis. Several studies have demonstrated that nonadherence to prescribed preventive therapies is prevalent and associated with adverse outcomes.[31-33]

Limitations

We have demonstrated that the use of ultrasound to screen for carotid plaque and increased CIMT changed physicians' treatment plans and patients' reported motivation regarding health-related behaviors, but we have not shown that carotid screening and the changes in patients' and physicians' behavior that may result from it reduce long-term CVD risk. Indeed, although changes in physicians' practice were observed in the office encounters, this study evaluated only patients' reported motivations, not their actions. It is unlikely that screening alone will lead to sustained improvements in patients' compliance with medication and lifestyle recommendations, but if longer term studies show improvements in health-related behaviors, office-based carotid ultrasound may be a useful part of a comprehensive case identification and risk management program. Because this study did not have a control group, it is possible that the observed changes in physicians' behavior and subjects' motivation in this study may be different when applied to clinical practice, in which patients and health care professionals are not formally being observed and evaluated.

It must be emphasized that the NSCs in this study underwent a rigorous training and certification program with ongoing quality control and feedback. The high degree of skill achieved by the NSCs in this study was the result of intensive training and feedback from an experienced core ultrasound laboratory that is very experienced at performing and teaching CIMT scanning and measurement.[9] The widespread use of suboptimally trained NSCs in office practices may lead to significant errors in test ordering and patient treatment. Standards for training and quality control for the clinical performance of CIMT studies have recently been described.[34] Some practices used participants recruited by advertising. Although individuals who respond to advertisements may be more motivated to change behavior, we did not find any heterogeneity in carotid ultrasound findings or outcomes between sites that advertised and those that did not.

There was heterogeneity among sites with regard to ordering additional testing, with 2 sites appearing to be more likely to order stress tests when carotid ultrasound findings were abnormal. Although the prescription of risk-reducing lifestyle and medication changes after the discovery of subclinical vascular disease may be justifiable, ordering stress tests in asymptomatic patients is more controversial, and the downstream costs of additional diagnostic testing after screening tests may not be justifiable.[35] Indeed, the incremental value and cost-effectiveness of additional diagnostic testing have not been proved. Unnecessary testing results from carotid ultrasound studies may lead to adverse medical outcomes and additional costs.

Conclusions

This was the first multicenter study to demonstrate that ultrasound screening for the presence of carotid plaques and increased CIMT can be integrated into an office visit and that the findings of office-based carotid ultrasound studies can influence physicians' prescriptions of evidence-based interventions such as antiplatelet and lipid-lowering therapy. Screening also increased patients' motivation regarding several health-related behaviors. Patients with abnormal carotid ultrasound findings recognized their increased cardiovascular risk and planned to take lipid-lowering medication. Long-term studies that evaluate CVD screening programs are needed to determine if the short-term changes in physicians' behavior and patients' motivation observed after screening translate into a long-term, cost-effective reduction in CVD risk.

Acknowledgments

This study was funded by SonoSite, Inc. (Bothell, WA). SonoSite also provided ultrasound equipment and the semiautomated border detection software used in this study, as well as technical support for its use. SonoSite had no role in the design or analysis of the data from the study and did not contribute to the content of this report.

Dr Korcarz was supported by a Ruth L. Kirschstein National Research Service Award from the National Heart, Lung, and Blood Institute (Bethesda, MD) to the University of Wisconsin–Madison Cardiovascular Research Center (T32 HL 07936).

Participating institutions and individuals and steering committee members are listed in the Appendix.

Appendix

The core laboratory and coordinating center for this study was the University of Wisconsin Atherosclerosis Imaging Research Program, University of Wisconsin School of Medicine and Public Health, Madison, WI (James H. Stein, MD, principal investigator [PI]; Claudia E. Korcarz, DVM, RDCS). The following individuals and institutions participated in this study: Heart Prevention Clinic of Idaho, Boise, ID (Bryan Pogue, MD, site PI; Kelli Sizemore; Amy Webb); New York Physicians, New York, NY (John Postley, MD, site PI; Maria Chan); University of Chicago Pritzker School of Medicine, Chicago, IL (Jeanne M. DeCara, MD, site PI; Kathy Furlong, RN); University of Minnesota School of Public Health and Minneapolis Heart Institute Foundation, Minneapolis, MN (Alan T. Hirsch, MD, site PI; Faye Imker-Witte; Kristi Jacobson); and University of Pennsylvania Medical School, Philadelphia, PA (Emile R. Mohler, MD, site PI; Wendy S. Tzou, MD).

The steering committee included Dr Stein (study PI); Dr Hirsch (study co-PI); Dr DeCara (University of Chicago Pritzker School of Medicine); Patrick E. McBride, MD, MPH (University of Wisconsin School of Medicine and Public Health, Madison, WI); Christopher M. Rembold, MD (University of Virginia Medical School, Charlottesville, VA); Neil Stone, MD (Northwestern University Feinberg School of Medicine, Chicago, IL); and Dr Tzou.

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Table 1

Subject characteristics (n = 263)

Characteristic	Mean	SD	Range
Descriptive			
Age (yr)	58.1	6.6	45-70
Men (%)	48.7	—	—
White race (%)	78%	—	—
Cigarette smoking (%)	17.1	—	—
Family history of premature cardiovascular disease (%)	46.8	—	—
History of hypertension (%)	44.5	—	—
Receiving blood pressure medication (%)	35.0	—	—
History of dyslipidemia (%)	60.1	—	—
Diabetes mellitus (%)	9.1	—	—
Receiving glycemic control medication (%)	6.8	—	—
Measured			
Systolic blood pressure (mm Hg)	129.3	18.1	86-180
Diastolic blood pressure (mm Hg)	75.9	10.1	52-100
Waist circumference (cm)	36.8	5.6	24.0-58.0
Body mass index (kg/m^2)	27.9	5.6	17.8-56.0
Fasting glucose (mg/dL)	100.0	26.8	60.0-351.0
Total cholesterol (mg/dL)	209.3	41.4	114.0-319.0
Triglycerides (mg/dL)	143.6	122.3	30.0-1,182.0
High-density lipoprotein cholesterol (mg/dL)	56.1	17.7	21.0-131.0
Low-density lipoprotein cholesterol (mg/dL)	125.6	36.8	33.4-236.0
Total/high-density lipoprotein cholesterol (mg/dL)	4.1	1.4	1.8-10.0
Framingham 10-year cardiovascular risk (%)	6.1	5.2	<1.0-24.3
Right common carotid artery CIMT (mm)	0.769	0.135	0.460-1.241
Left common carotid artery CIMT (mm)	0.776	0.141	0.466-1.357
left or right CIMT ≥ 75th percentile (%)	59.0	—	—
Carotid plaque present (%)	58.2	—	—

CIMT, Carotid intima-media thickness.

Table 2

Physicians' treatment changes after carotid ultrasound studies

	In creased CIMT				Carotid plaque present			
Treatment	***β***	**OR**	**95% CI**	***P* value**	***β***	**OR**	**95% CI**	***P* value**
Add antiplatelet medication	1.85	6.34	3.31-12.16	<.001	1.58	4.84	2.60-9.00	β.001
Add blood pressure medication	0.41	1.50	0.55-4.06	.423	0.42	1.51	0.55-4.13	4.412
Add lipid-lowering medication	1.08	2.93	1.59-5.40	<.001	2.00	7.40	3.62-15.11	β.001
Refer for another test	1.07	2.91	0.96-8.74	.057	1.32	3.75	1.02-13.75	0.046

CI, Confidence interval; *CIMT*, carotid intima-media thickness; *OR*, odds ratio.

Multinomial logistic regression models included site, age, sex, race, body mass index, systolic blood pressure, diastolic blood pressure, and low-density lipoprotein cholesterol.

Table 3

Changes in responses to survey questions before and after carotid ultrasound scans

		All subjects		Carotid scan results not abnormal		Increased CIMT		Carotid plaque present		Increased CIMT or carotid plaque present	
	Survey item	Δ	*P* value	Δ	*P* value	Δ	*P* value	Δ	*P* value	Δ	*P* value
1.	I plan to exercise 30 minutes, 5 times/week.	0.682	<.001	0.543	.003	0.741	<.001	0.705	<.001	0.714	<.001
2.	I plan to lower my cholesterol by changing my diet.	0.529	<.001	0.333	.123	0.600	<.001	0.592	<.001	0.574	<.001
3.	I plan to reduce my cholesterol by taking medications.	0.521	<.001	−0.225	.275	0.806	<.001*	0.701	<.001*	0.687	<.001*
4.	Lowering cholesterol is … (harmful – beneficial)	0.049	.209	0.149	.206	0.000	.889	0.041	.357	0.026	.494
5.	I will try to eat at least 5 servings each day of high fiber foods such as: vegetables, oatmeal, whole grain breads and cereals.	0.398	<.001	0.304	.051	0.378	<.001	0.430	<.001	0.420	<.001
6.	I intend to limit saturated and trans fats such as butter, cheese, ice cream, fatty meats, and deep-fried foods in my diet.	0.494	<.001	0.333	.137	0.550	<.001	0.570	<.001	0.533	<.001
7.	I will try to limit sugars, sweetened drinks, juices and starches in my diet.	0.219	.002	0.000	.942	0.364	<.001	0.267	.005	0.272	<.001
8.	I plan to lower my blood pressure by changing my diet.	0.407	<.001	0.200	.652	0.547	<.001	0.496	<.001	0.453	<.001
9.	I plan to reduce my blood pressure by taking medications.	0.229	.076	0.122	.761	0.386	.018	0.209	.095	0.254	.048
10.	Lowering blood pressure is … (harmful – beneficial).	-0.008	.893	0.000	1.000	−0.015	.966	0.041	.369	−0.010	.889
11.	The likelihood I have heart disease is … (extremely high – extremely low).	0.454	<.001	−0.205	.614	0.585	<.001*	0.717	<.001*	0.612	<.001*
12.	The likelihood I will develop heart disease is … (extremely high – extremely low).	0.274	.005	−0.442	.103	0.396	.005	0.593	<.001*	0.439	<.001*

Δ, Absolute difference, postscan minus prescan.

*Significant difference compared with the group with normal results ($P < .004$, corrected for multiple testing).