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Effects of an Office-Based Carotid Ultrasound Screening Intervention

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Abstract

Background—Carotid ultrasound screening (CUS) has been recommended for cardiovascular disease (CVD) risk prediction; however, its effectiveness in clinical practice is unknown. The purpose of this study was to prospectively determine the effects of office-based CUS on physician decision-making and patient health-related behaviors (HRBs).

Methods—Physicians from 5 non-academic, community practices recruited patients ≥40 years old with ≥1 CVD risk factor. Abnormal carotid ultrasound screening (AbnlCUS) was defined as carotid intima-media thickness >75th percentile or carotid plaque presence. Subjects completed questionnaires before and immediately after CUS, then 30 days later to determine self-reported behavioral changes. Odds ratios (OR) for changes in physician management and patient HRBs were determined from multivariate hierarchical logistic regression models.

Results—There were 355 subjects (mean [standard deviation] 53.6 [7.9] years old, 2.3 [0.9] risk factors, 58% women); 266 (74.9%) had AbnlCUS. Presence of AbnlCUS altered physicians' prescription of aspirin (p<0.001) and cholesterol medications (p<0.001). Immediately after CUS, subjects reported increased ability to change HRBs (p=0.002), regardless of their test results. Subjects with AbnlCUS reported increased CVD risk perception (OR 4.14, p<0.001), intentions to exercise (OR 2.28, p=0.008), make dietary changes (OR 2.95, p<0.001), and quit smoking (OR 4.98, p=0.022). After 30 days, 34% increased exercise frequency and 37% reported weight loss; but these changes were not predicted by the CUS results. AbnlCUS modestly predicted reduced dietary sodium (OR 1.45, p=0.002) and increased fiber (OR 1.55, p=0.022) intake.

Conclusions—Finding abnormal results on CUS had major effects on physician but not patient behaviors.

Keywords

Atherosclerosis; Carotid arteries; Risk factors; Ultrasound

The limitations of current approaches to cardiovascular disease (CVD) risk prediction are well-known.[1–3] Because increased carotid intima-media thickness (CIMT) and carotid plaque presence are independent predictors of future CVD events, carotid ultrasound screening (CUS) has been recommended as a clinical tool to assist with CVD prediction in intermediate risk patients (*i.e.* patients with a 6%–20% risk of myocardial infarction or coronary heart disease death over 10 years).[4–6] The widespread availability of inexpensive, handheld ultrasound (HHU) systems, automated border detection programs for measurement of CIMT, and straightforward scanning protocols have facilitated transition of CUS from a

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research tool to a clinical test.[6,7–10] Furthermore, it has been demonstrated that non-sonographer clinicians (NSCs) can reliably use HHU devices to identify increased CIMT and carotid plaque.[7,11,12] However, there are limited data on the effectiveness of CUS in community medical practices and whether finding increased CIMT or carotid plaque improves patient outcomes.[6,13–19] The purpose of this study was to prospectively evaluate the effects of CUS findings on physician decision-making and 30-day patient health-related behaviors (HRBs).

Methods

Five community, non-academic, primary care medical practices participated in this study (Appendix 1). The institutional review boards at the University of Wisconsin School of Medicine and Public Health and each site approved this study. Participating health care providers and patient-subjects provided informed consent.

Training of Health Care Providers and NSCs

Health care providers (physicians, nurse practitioners and physician assistants) completed a 2-day training program that has been demonstrated to effectively train NSCs to accurately and reproducibly perform CUS studies for evaluation of CIMT and carotid plaque presence.[7] Training sessions included didactic lectures, hands-on ultrasound scanning, and image measurement practice. After training, each NSC was required to scan and measure paired mock studies at their clinic site, with overview of images and measurements by the core lab (Appendix 2). When the NSCs met standards for image quality, protocol adherence, measurement accuracy, and use of risk assessment tools, they were certified to enroll subjects.

Health care providers learned how to use information from the CUS scan for CVD risk stratification, treatment, and patient education. They received guideline-based training on lifestyle recommendations, smoking cessation, systolic blood pressure (SBP) and low-density lipoprotein cholesterol (LDL-C) targets, and recommendations on prescribing aspirin, antihypertensive and cholesterol-lowering medication. They were taught that a mean CIMT >75th percentile of the Atherosclerosis Risk in Communities Study (based on their age, sex, and race) or carotid plaque presence (Figure I) indicated the presence of an abnormal carotid ultrasound screening (AbnlCUS) and increased CVD risk, as per the recent consensus statement from the American Society of Echocardiography.[6,20]

Inclusion/Exclusion Criteria for Patient-Subjects

Subjects were patients presenting for routine office visits. Inclusion criteria were chosen to represent the types of patients referred for screening. They were 40–70 years old, asymptomatic, and had at least one CVD risk factor: recent cigarette smoking (in past year), hypertension (SBP ≥140 mmHg or taking antihypertensive medication), dyslipidemia (LDL-C ≥130 mg/dL or high-density lipoprotein cholesterol <40 mg/dL), family history of early CVD (first-degree relative male <55 or a female <65 years old), or diabetes mellitus (fasting glucose ≥126 mg/dL, hemoglobin A_1C >6.5%, or taking anti-glycemic medication). Exclusion criteria were use of cholesterol-lowering medications in the past year, active liver disease, active thyroid disease, creatinine >2.5 mg/dL, history of coronary artery disease, history of cerebrovascular disease, or history of peripheral artery disease.

Office Procedures

Subjects were approached for participation at their routine clinic visit by a member of the medical staff involved in their care. The visit proceeded according to usual procedures with standard office measurements, laboratory tests, history-taking, physical examination, and

physician recommendations prior to CUS. Immediately after the routine visit, an initial management plan (prescription of aspirin, lipid-lowering therapy, antihypertensive therapy, lifestyle changes, target LDL-C level, target SBP) was developed by the physician and recorded on the case report form. After the visit, but immediately before CUS, subjects completed a survey designed to assess motivation, perceived self-efficacy, and intention to change based on the Theory of Reasoned Action.[21,22] The survey included 14 questions presented on a 7-point bipolar Likert scale which assessed: 1) intention to exercise, 2) intention to make specific dietary changes (reducing saturated fat and sugar intake, increasing fiber intake), 3) intention to quit smoking (smokers only), 4) likelihood of using medications or lifestyle changes to reduce cholesterol and blood pressure, 5) perception of CVD risk associated with high cholesterol and blood pressure, 6) likelihood of having CVD currently and/or in the future, and 7) perceived ability to make lifestyle changes to reduce CVD risk. Response options ranged from strongly disagree (score = −3) to strongly agree (score = 3).

CUS was performed using a previously validated clinical scanning protocol recommended by the American Society of Echocardiography for assessing CVD risk (Appendix 2).[6,7,11,12] Subjects were not charged for the test. After CUS, the subject's physician could alter their management plan based on the ultrasound data. Next, subjects were informed of their results by their primary care provider. Subjects were shown pictures of their arteries and received structured and standardized education from trained medical staff about the association between AbnlCUS and CVD. Subjects then received CVD risk-reduction lifestyle recommendations and, if indicated, pharmacotherapy. The same survey was re-administered immediately after discussing their CUS results to re-evaluate the subjects' perceived CVD risk and perceived ability and intentions to change HRBs. Thirty days after CUS, subjects were mailed an additional survey to evaluate their self-reported behavioral changes.

Carotid Ultrasonography

CUS was performed by NSCs using a HHU system (Sonosite MicroMaxx™, Bothell, WA) with a linear array vascular ultrasound transducer (L38e). The abbreviated CIMT imaging protocol is consistent with recent clinical testing recommendations (Appendix 2 on-line video file 1).[6,7,11,12] The mean far wall CIMT of the distal 1 cm of each common carotid artery was measured in triplicate at the ECG R-wave using a semi-automated border detection program (SonoCalc IMT™, Bothell, WA). Images were obtained on each side from 3 angles of interrogation. Longitudinal and cross-sectional images of the common, bulb, and internal carotid artery segments were evaluated for plaque presence, defined as a focal intimal-medial thickness of ≥1.2 cm (Figure I).[23,24] All images, measurements, interpretations, and recommendations made for each subject were performed by NSCs and reviewed by the core lab. Corrections, modifications, and reporting of incidental findings were provided to the subject and their physician (Appendix 2).

Statistical Analysis

Data analysis was performed using Mplus software (Muthén and Muthén; Los Angeles, CA). Continuous variables are described as means (standard deviation, SD) and ranges. Categorical variables are described as percentages. Chi-square (χ^2) testing was performed to identify differences among categorical variables for the presence of AbnlCUS. Student's t-tests were used to identify differences in continuous variables among subjects with and without AbnlCUS, carotid plaque, or increased CIMT. Physician changes in CVD risk management decisions from pre- to post-CUS were examined by χ^2 analysis and multivariate hierarchical logistic regression models. Each model was nested by clinical site and corrected using the Holm-Sidak method for multiple comparisons. All regression analysis models were controlled for age, sex, and the presence of AbnlCUS. To this basic

model, additional variables were added including: body mass index, waist circumference, LDL-C, high-density lipoprotein cholesterol, triglycerides, SBP, diastolic blood pressure, history of hypertension, history of diabetes mellitus, cigarette smoking, and family history of early CVD. Baseline aspirin and antihypertensive use were added to the aspirin and antihypertensive models respectively. The beta (β), standard error, and 95% confidence intervals (CI) are reported. Odds ratios were not reported due to very small cell sizes for physicians prescribing preventive therapy for normal scan results, which significantly inflated the odds ratios of preventive prescriptions for abnormal scan results.

Patient survey results before and after CUS were evaluated as absolute differences (Δ) in survey scores for each question using paired t-tests with Bonferroni's correction for multiple comparisons. Changes in survey results were analyzed as continuous variables using least squares linear regression; all models included age, sex, and the presence of AbnlCUS. The same variables in the physician change models were added to this basic model, in addition to: educational level, exercise frequency (minutes/week), prescription medication coverage, and baseline survey response. Multivariable-adjusted odds ratios were calculated for changes in CVD risk perception and behavioral intentions in relation to the presence of AbnlCUS. Any increase in CVD risk perception or intention to change behavior was considered a positive response to CUS.

Results

Subject Characteristics (Table I)

Each clinical site recruited a mean of 70.6 (4.0) subjects, for a total of 355 subjects. Their mean age was 53.6 (7.9) years (range 40–70 years); 205 (57.7%) were women and 349 (98.6%) were Caucasian. Subjects had a mean Framingham risk score of 4.4 (5.0) %/10 years. At baseline, 119 (33.5%) were on aspirin, (39.2%) had a history of hypertension, and113 (81.2%) were on antihypertensive medication. Most subjects had a history of dyslipidemia (275 [77.9%]). Only 20 (5.6%) had a history of diabetes mellitus; 19 (95%) were on anti-glycemic medication. Less than half of the subjects graduated from college (n=167, 47.1%). Most had medical (n=347, 98.0%) and prescription (n=332, 93.4%) insurance. A composite Bland-Altman plot comparing the CIMT results of the NSCs with the core laboratory measurements is presented in Figure II. The NSCs' performance analysis is in Appendix 2.

Carotid plaques were detected in 125 (35.2%) subjects, 231 (65.1%) had increased CIMT (>75th percentile) and 266 subjects (74.5%) had AbnlCUS. The mean right and left CIMT values were 0.726 (0.134) and 0.737 (0.127) mm, respectively. In regard to AbnlCUS, only 9 subjects (2.5%) were incorrectly identified by the sites. Their office visit data were included in the analysis, but their 30-day data were excluded from the follow-up analysis because they received incorrect guidance (which was subsequently corrected after review by the ultrasound core lab). Subjects with AbnlCUS had higher systolic (125.2 vs. 120.6 mmHg, p=0.006) and diastolic blood pressures (77.1 vs. 73.9 mmHg, p=0.002) and were more likely to have dyslipidemia (p=0.002).

Physician Treatment Changes After Carotid Ultrasound Screening

The presence of AbnlCUS significantly altered physicians' target risk factor goals. In subjects with AbnlCUS, physicians lowered their LDL-C target (χ^2=182.1, p<0.001). The most frequent changes were decreasing target LDL-C from 130 to 100 mg/dL (n=105, 29.5%), or 100 to 70 mg/dL (n=76, 21.4%). Physicians did not significantly change their LDL-C target for patients with normal scans (χ^2=7.00, p=0.321). Only one subject's target was increased (from 70 to 100 mg/dL). When AbnlCUS was detected, physicians' target

SBP goal also changed (χ^2=72.0, p<0.001), most frequently a decrease from 140 to 130 mmHg (n=66, 18.6%). Physicians did not change their SBP target for patients with normal scans (χ^2=3.00, p=0.809).

AbnlCUS presence significantly altered physicians' prescription of aspirin and lipid-lowering medication (Table II). Physicians were more likely to prescribe aspirin to subjects with AbnlCUS (p<0.001). After CUS, aspirin was initiated for 92 (25.9%) subjects and 35 (9.9%) had an increase in their baseline aspirin dose. Only 3 (0.9%) subjects with normal scans were prescribed aspirin. In multivariate analysis, AbnlCUS presence independently predicted the prescription of aspirin (ϐ=6.59 [3.97–9.21], p<0.001), as did SBP (ϐ=0.07 [0.02–0.13], p=0.009), and diabetes mellitus (ϐ=3.81 [1.53–6.08], p=0.001).

Prior to CUS, 238 (67.0%) subjects were not at their LDL-C goal. Of these subjects, physicians planned to initiate or increase lipid-lowering medication in only 113 (47.4%) before CUS; however, these data do not reflect plans to initiate lifestyle modifications. After CUS, physicians initiated or increased lipid-lowering medication for all 238 (100%) subjects who previously were not at goal. Physicians did not alter therapy in subjects who were at LDL-C goal prior to screening. In multivariate analysis, AbnlCUS (ϐ=5.36 [4.31–6.41], p<0.001) and male sex (ϐ=0.79 [0.20–1.38], p=0.009) significantly predicted lipid-lowering medication prescription after CUS. Although all physicians received the same training, there were differences in prescription of cholesterol medication by site (p=0.006).

Prior to CUS, 40 (11.2%) subjects were not at their SBP goal; physicians planned to initiate or increase antihypertensive medication in 32 (80%) of these subjects prior to CUS. After CUS, antihypertensive medication changes were recommended for an additional 21 subjects, including 8 whose SBP already was at target. After CUS, additional diagnostic testing (usually stress tests) was ordered in only 22 subjects, 14 of which were ordered by one site. Referral for additional testing was not predicted by AbnlCUS (p=0.108).

Immediate Changes in Subject Intentions and Perceptions after CUS

All subjects completed surveys immediately before and after CUS. The absolute differences (Δ) in pre- and post-scan survey scores were statistically significant for all survey items, suggesting that the very act of screening increased perception of CVD risk and intentions to change HRBs (Table III). Immediately after CUS, subjects had increased perceived self-efficacy to change HRBs (p=0.002); however, this was not predicted by the presence of AbnlCUS (p=0.927). In multivariate analysis, having prescription medication insurance (OR 5.30 [1.08–25.96], p=0.040) was associated with increased perceived self-efficacy.

The presence of AbnlCUS predicted increased intentions to make some HRB changes (Table IV). AbnlCUS presence was associated with increased intentions to reach exercise goals (OR 2.28 [1.24–4.22], p=0.008) and to lower cholesterol with dietary changes (OR 2.95 [1.89–4.61], p<0.001); the latter was also influenced by body-mass index (OR 1.08 [1.01–1.17], p=0.027). Regarding specific dietary changes, AbnlCUS presence was associated with intentions to decrease saturated fat intake (OR 2.04 [1.54–2.70], p<0.001), but not changes in fiber, sugar, or salt intake. AbnlCUS presence also increased intentions to decrease cholesterol with medication (OR 19.70 [4.84–80.15], p<0.001), with significant influences from history of hypertension (OR 2.18 [1.48–3.21], p<0.001) and medication insurance (OR 2.14 [1.45–3.17], p<0.001). Although the total number of smokers was small (n=45), AbnlCUS presence (OR 4.98 [1.25–19.76], p=0.022) and a history of hypertension (OR 11.31 [4.78–26.86], p<0.001) predicted increased intentions to quit smoking after CUS. AbnlCUS presence also was associated with increased perceptions of having (OR 4.14 [1.99–8.62], p<0.001) or developing CVD (OR 2.75 [0.20–1.82], p=0.014).

30-Day Follow-Up

Only 28 subjects (7.9%) did not return their 30-day surveys. After 30 days, subjects' perceived risk of current or future CVD (p=0.467) and knowledge of the health benefits of lowering blood pressure (p=0.442) did not change significantly. At follow-up, 108 (34.1%) subjects reported increased exercise frequency with 62.9% exercising ≥30 minutes for 5 days/week, an increase from 43.7% at baseline (p<0.001). Weight loss was reported by 118 (37.2%) subjects. However, AbnlCUS presence did not predict the observed increase in exercise frequency (p=0.816) or weight loss (p=0.090). After 30 days, 197 (62.1%) subjects reported dietary changes. The only changes predicted by identifying AbnlCUS were a modest increase in dietary fiber intake (OR 1.55 [0.06–0.81], p=0.022) and changes to control blood pressure (OR 1.97 [1.53–2.54], p<0.001), specifically to decrease salt intake (OR 1.45 [1.15–1.83], p=0.002). Among the 45 smokers, 5 (11.1%) reported smoking cessation at 30 days; however, the sample size was too small for multivariate analysis.

Discussion

In this large, prospective, multi-center study, we demonstrated that when primary care providers identified increased subclinical atherosclerosis on an office-based CUS, they lowered their SBP and LDL-C targets and were more likely to prescribe preventive therapies such as aspirin and lipid-lowering medications. The vast majority of these subjects would not have qualified for LDL-C reduction or aspirin based on their CVD risk. Although the estimated CVD risk of the subjects in our study was low to moderate, the majority of our subjects had AbnlCUS, as previously described in populations with risk factors but low short-term CVD risk.[3, 25, 26] Despite identical training, differences in physicians' prescription of cholesterol medication were observed between the sites, most likely due to variations in baseline practices and disease severity. Similar practice variation in prescription of preventive therapies has been described in other studies.[27,28]

The most interesting and unique findings from this study are those related to patients' intentions and self-reported behaviors after 30 days. One of the most salient findings is that regardless of the results of the ultrasound, our patients reported an increased perceived ability to change HRBs after screening. Discovering AbnlCUS did not influence the patients' perceptions of their ability to change HRBs.

Therefore, it is likely that the very act of screening itself, not the results of the test, increased patients' perceived self-efficacy to make behavioral changes to decrease their CVD risk. This finding is consistent with previous studies by our research group that were performed in academic medical centers and research centers, showing that increased patient motivation for behavioral change is not related to having an abnormal CUS, but instead is related to the screening process.[11,18] It is possible that the observed increases in patients' perceived self-efficacy were related to having a more intensive clinical encounter with their primary provider and not the screening; however, previous research suggests that single CVD risk discussions are not very effective.[29]

Immediately after CUS, subjects with AbnlCUS perceived themselves to be at increased CVD risk. Previous studies have demonstrated that immediately after CUS, there is an increase in CVD risk perception.[11,18] Although risk perception is a key motivator for change, there have been conflicting results about risk perception as a predictor of behavioral change.[28,30–32] Patients with AbnlCUS were more likely to intend to reach exercise goals, make dietary changes and quit smoking, so finding AbnlCUS did affect some patients' intentions. Several models of behavioral change suggest that individuals' intentions are among the most important predictors of behavior.[30,31] Self-efficacy and outcome expectations are critical predictors of intentions; however, the relationship between these

processes is unclear.[32–34] Unfortunately, increased intention to make HRB changes does not consistently predict that an individual actually will make changes (the "intention-behavior gap").[22] Some behavioral models suggest that there is a major distinction between pre-intentional motivation processes that lead to behavioral intention and post-intentional processes that lead to documented behavioral change.[35] Thus, to help bridge the intention-behavior gap, pre-intentional factors such as threat perception and self-efficacy and post-intentional factors such as strategic planning on how to initiate and maintain behavioral changes should be addressed before assuming that intentions will translate into action [35] Planning should also include anticipation of barriers and alternatives to overcome them.[22, 35]

A strength of this study was the evaluation of 30-day outcomes. Behavioral changes reported after 30 days included increased exercise frequency, certain dietary changes, and weight loss. However, the presence of AbnlCUS only predicted specific dietary changes after 30 days, not changes in exercise frequency or reported weight loss. In spite of their intentions and the high percentage of subjects with AbnlCUS, a substantial portion of subjects did not report any HRB changes after 30 days. A separate survey to measure self-efficacy was not used and it is possible that differences in self-reported behavioral change in our study reflected differences in individuals' baseline levels of self-efficacy, since individuals with lower self-efficacy are less likely to transition from intention to action.[14] Previous smoking cessation studies demonstrated that individuals with higher self-efficacy were more likely to engage in cessation therapy.[14,36] In a small randomized study, the effect of the carotid ultrasound imaging on smoking cessation behaviors was mediated by self-efficacy.[14] In behavior prediction models, intention and perceived behavior control only account for 14–25% of population variance in documented behavior change, so large effects on intention can result in only small behavioral changes.[22,30,33] This supports the need for recurring CVD risk education and counseling and emphasizes the limitations of effectiveness of one-time interventions.[34] Because human behavior is so complex, additional research is needed to thoroughly evaluate the role of carotid ultrasound screening, or any atherosclerosis imaging technique, and the optimal frequency of feedback necessary to initiate and maintain behavioral change.[34]

Limitations

Although this study performed a prospective intervention and each physician and patient served as their own control, it was not a randomized clinical trial, so a strategy of CUS was not compared to standard care or to CVD risk counseling alone. This study design is susceptible to the Hawthorne Effect, whereby subjects - in this study, both doctors and patients - alter their behavior simply because they are being observed, and not due to an effect from the experimental intervention.[37] Although patients may have overestimated their intentions and reported 30-day outcomes differently because they were being studied, the presence of AbnlCUS had very little effect on self-reported behavioral changes after 30 days. Therefore, if significant behavioral changes were not noted in this type of study, it is unlikely that a randomized clinical trial would have a *greater* effect on patient behaviors. The Hawthorne Effect most likely biased the changes observed in physician behaviors. Because of the training the physicians received and their awareness of being observed, it is not surprising that finding AbnlCUS influenced their prescribing practices. A randomized clinical trial would be necessary to determine if the more aggressive treatment strategies adopted by physicians after finding AbnlCUS translate into CVD event reduction. Prescription of statins in patients with increased CIMT reduces CIMT progression,[38] but its effects on CVD events have not been studied. Although prescribing aspirin was considered a reasonable intervention for patients with subclinical atherosclerosis when this study was designed, recent studies suggest it might not be beneficial.[39]

We used surveys rather than direct measurements to describe some of the 30-day outcomes; however, direct measurement would have further reduced the modest effects at 30 days. The surveys have not been independently validated, but have been revised based on previous experience from our research group.[11,18] Although the 30-day follow-up period is relatively short, it is unlikely that we would have seen larger effects if the study was longer. Indeed, health behavior decay - a gradual migration away from newly adopted healthy behavior - is more likely.[40] Although imaging for CVD risk assessment is recommended for intermediate risk individuals, low to intermediate risk individuals, such as those recruited into this study, frequently are referred to such screening programs.[9,18,41] Finally, the lack of racial/ethnic diversity and limited geographical area of the clinical sites may limit the generalization of the findings.

Conclusions

When community physicians identify AbnlCUS in an office setting, they modify CVD risk factor targets and are more likely to prescribe aspirin and lipid-lowering therapy. These changes in response to the screening results may reduce their patients' long-term CVD risk; however, their effectiveness has not been proven. The effects of CUS on patients are much more complex. The very act of screening - not the results of the test - appears to increase patients' perceived ability to change behaviors. Screening also led to short-term improvements in exercise frequency, weight loss, and dietary changes; however, an abnormal CUS only predicted modest dietary changes - nothing else. Randomized trials are needed to determine the long-term clinical effects of atherosclerosis screening on health-related behaviors and clinical outcomes; however, our observations suggest that the results of screening on patient behaviors are modest, at best. Such trials should be guided by behavioral theory to best identify patients who are most likely to respond to imaging feedback.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

AbnlCUS	abnormal carotid ultrasound screening
CIMT	carotid intima-media thickness
CUS	carotid ultrasound screening
CVD	cardiovascular disease
HHU	handheld ultrasound system

HRBs	health-related behaviors
LDL-C	low-density lipoprotein cholesterol
NSC	non-sonographer clinician
SBP	systolic blood pressure

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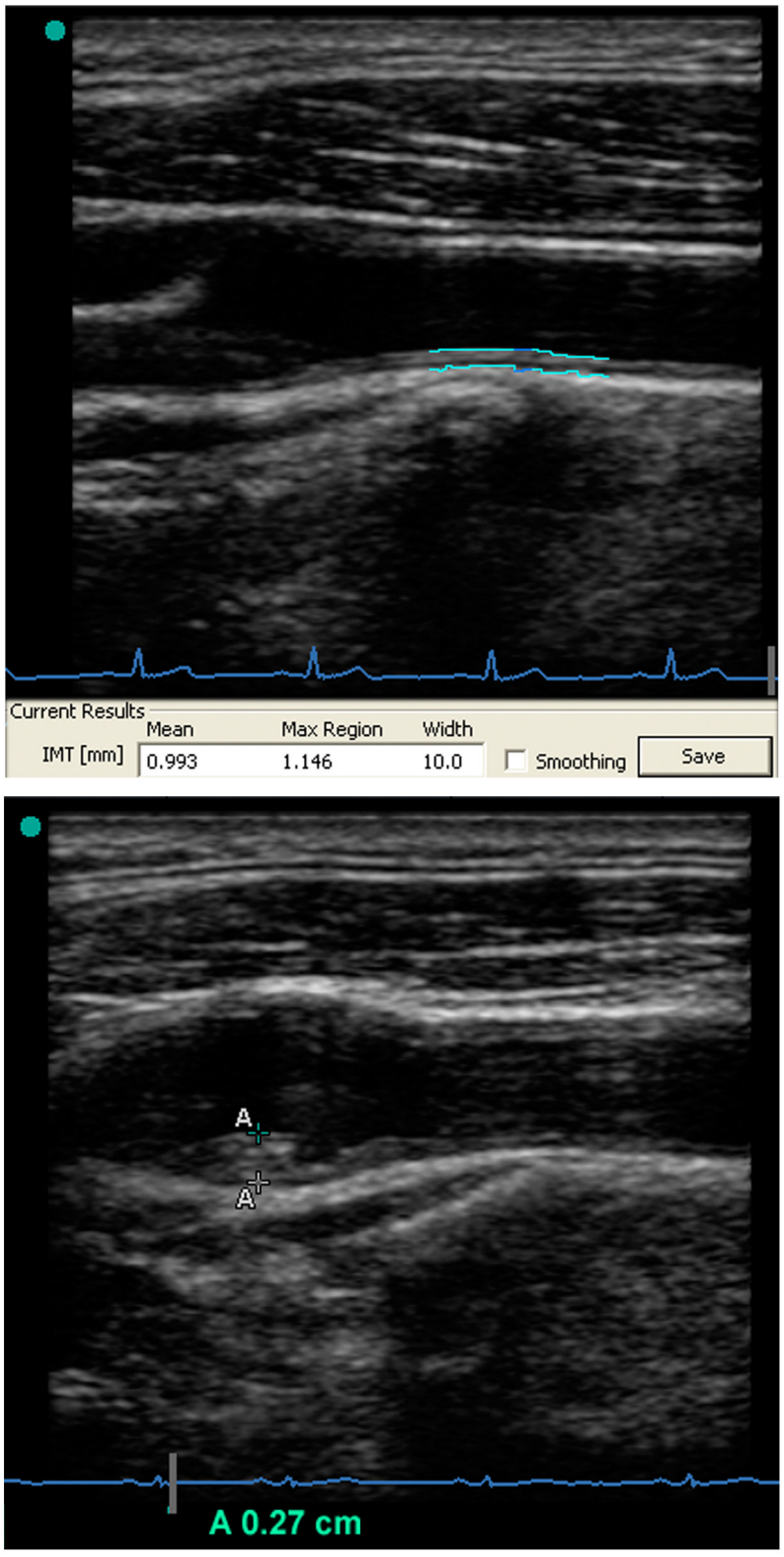

Figure I. Representative Carotid Ultrasound Images

IA. Right Common Carotid Artery Segment with Traced Far Wall Carotid Intima-Media Thickness.
IB. Plaque in Far Wall if Right Carotid Bulb
Note extent of plaque (27 mm) is denoted as the distance between the two "+" signs labeled by the letter "A.".

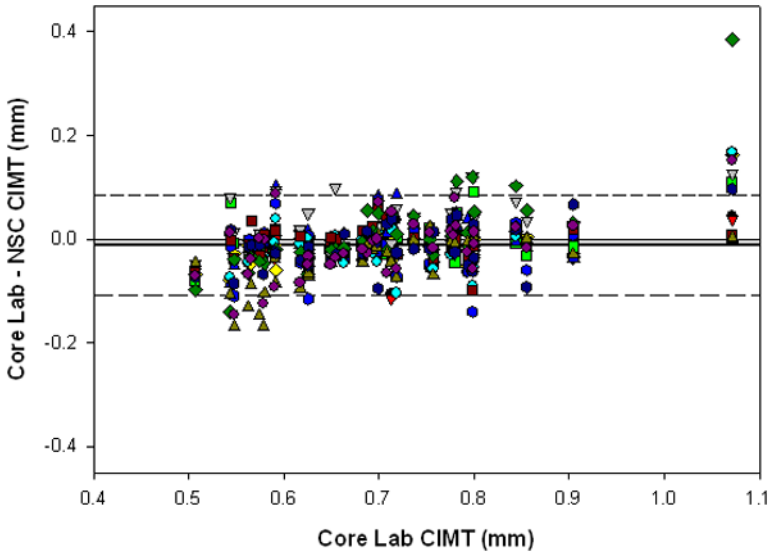

Figure II. Composite Bland-Altman Plot Comparing Non-Sonographer Clinicians with Core Laboratory CIMT Readings

IMT = carotid intima-media thickness

NSC = Non-sonographer clinician

Each discreet symbol (i.e. circle, square, diamond, etc.) represents a value from a single NSC

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Table I

Baseline Cardiovascular Disease Risk Factors by CIMT Tertiles Adjusted for Age and Sex (All Subjects, N = 355)

	Entire Group		CIMT Tertiles			P value
	Mean (SD)	Range	1	2	3	
Common carotid artery CIMT (mm)	0.731 (0.111)	0.489 – 1.069	0.489 – 0.673	0.674 – 0.760	0.761 – 1.069	-
Number of CVD risk factors	2.3 (0.9)	1–5	2.2 (0.9)	2.3 (0.8)	2.5 (0.8)	**0.008**
Waist circumference (cm)	96.9 (15.7)	58–151	95.2 (15.6)	96.1 (14.8)	100.9 (15.1)	**0.011**
Systolic blood pressure (mmHg)	124.1 (14.0)	90–172	120.3 (14.2)	125.8 (13.4)	126.8 (13.8)	**0.001**
Diastolic blood pressure (mmHg)	76.4 (9.23)	54–102	74.1 (9.5)	76.6 (8.9)	78.9 (9.2)	**0.001**
Total cholesterol (mg/dL)	223.4 (39.0)	102–359	217.9 (40.1)	220.5 (37.9)	229.1 (38.8)	0.091
Triglycerides (mg/dL)	146.9 (86.4)	40–584	140.0 (87.0)	143.1 (82.0)	162.1 (83.9)	0.118
High-density lipoprotein cholesterol (mg/dL)	54.3 (19.8)	17–154	53.7 (19.2)	55.7 (18.1)	50.4 (18.6)	0.091
Low-density lipoprotein cholesterol (mg/dL)	141.0 (35.7)	39–254	137.0 (37.1)	137.4 (34.9)	146.9 (35.8)	0.074
Non-HDL-C (mg/dL)	170.0 (38.3)	39–319	164.9 (40.6)	166.5 (38.3)	179.9 (39.3)	**0.009**

Data are expressed as mean (standard deviation)

P value = difference between CIMT tertiles

CIMT = carotid intima media thickness

CVD = cardiovascular disease

HDL-C = high-density lipoprotein cholesterol

Table II

Changes in Physician Management after Carotid Ultrasound Screening

	Presence of Advanced Subclinical Atherosclerosis		
Treatment	**θ**	**95% Confidence Interval**	**P value**
Add aspirin	6.59	3.97–9.21	**<0.001**
Add blood pressure medication	4.02	−0.37—8.42	0.073
Add lipid-lowering medication	5.36	4.31–6.41	**<0.001**
Refer for additional tests	1.26	−0.18—3.45	0.108

Table III

Changes in Subjects' Survey Responses Immediately after Carotid Ultrasound Screening

Survey Item	Δ	Standard Deviation	P Value
I will try to exercise 30 minutes, 5×/week	0.507	1.038	**<0.001**
I will try to lower my cholesterol by changing my diet	0.343	0.893	**<0.001**
I will try to reduce my cholesterol by taking medications	0.991	1.525	**<0.001**
I will try to eat at least 5 servings/day of high fiber foods	0.343	0.874	**<0.001**
I will try to eat less bad fats (saturated fat)	0.383	1.000	**<0.001**
I will try to eat less sugar	0.308	0.890	**<0.001**
I will try to eat less salt to help control my blood pressure	0.287	1.003	**<0.001**
I will try to lower my blood pressure by taking medications	0.343	1.303	**<0.001**
Family and friends think I should change my diet	0.429	1.100	**<0.001**
Family and friends think I should exercise more	0.276	1.034	**<0.001**
The likelihood I have heart disease is	−0.408*	1.642	**<0.001**
The likelihood I will develop heart disease is	−0.363*	1.699	**<0.001**
I will try to quit smoking	0.302	0.774	**0.006**
I know that I can change my lifestyle to reduce my risk	0.162	0.967	**0.002**

Δ = absolute difference of the mean survey scores (post-scan minus pre-scan survey results)

*Negative indicates an increase in likelihood (scale: extremely high → extremely low)

Table IV

Immediate Effects of Abnormal Carotid Ultrasound Screening on Patient Perceptions and Health-Related Behavior Intentions

	Odds Ratio	95% Confidence Interval	P value
Increased perception of CVD risk	4.14	1.99–8.62	**<0.001**
Plans to reach exercise goals	2.28	1.24–4.22	**0.008**
Plans to make healthy dietary changes	2.95	1.89–4.61	**<0.001**
Plans to quit smoking	4.98	1.25–19.76	**0.022**

CVD = cardiovascular disease