TABLE 2 Ejection Fraction–Dependent Short Form 36 Scores

Scale	Ejection Fraction <40		Ejection Fraction ≥40		
	Mean ± SD	Median	Mean ± SD	Median	p Value
Pain index	51 ± 10	52	52 ± 9	52	0.57
General health perceptions	47 ± 8	45	49 ± 10	52	0.34
Mental health Index	49 ± 9	50	50 ± 10	54	0.46
Physical functioning	48 ± 8	48	48 ± 9	52	0.42
Emotional role	47 ± 10	47	49 ± 11	55	0.45
Physical role	48 ± 7	48	47 ± 11	54	0.78
Social functioning	52 ± 7	53	50 ± 11	57	0.75
Vitality	47 ± 11	46	45 ± 12	49	0.64

generic measures of health status (SF-36 and COOP charts). *Age Ageing* 1997; 26:7–13.

4. Ecochard R, Colin C, Rabilloud M, de Gevigney G, Cao D, Ducreux C, Delaheye F, PRIMA Group. Indicators of myocardial dysfunction and quality of life, one year after acute infarction. *Eur J Heart Fail* 2001;3:561–568.

5. AVID Investigators. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. The Antiarrhythmics versus Implantable Defibrillators (AVID) Investigators. *N Engl J Med* 1997;337:1576–1583.

6. Connolly SJ, Gent M, Roberts RS, Dorian P, Roy D, Sheldon RS. Canadian implantable defibrillator study (CIDS): a randomized trial of the implantable cardioverter defibrillator against amiodarone. *Circulation* 2000;101:1297–1302.

7. Kuck KH, Cappato R, Siebels J, Ruppel R. Randomized comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from cardiac arrest: the Cardiac Arrest Study Hamburg (CASH). *Circulation* 2000;102:748–754.

8. Juenger J, Schellberg D, Kraemer S, Haunstetter A, Zugck C, Herzog W, Haass M. Health related quality of life in patients with congestive heart failure: comparison with other chronic diseases and relation to functional variables. *Heart* 2002;87:235–241.

9. Cone DC, Niemann JT. What is the quality of life for survivors of cardiac arrest? A prospective study. *Ann Emerg Med* 2000;35:201–202.

10. Dracup K, Walden JA, Stevenson LW, Brecht ML. Quality of life in patients with advanced heart failure. *J Heart Lung Transplant* 1992;11:273–279.

11. Gorkin L, Norvell NK, Rosen RC, Charles E, Shumaker SA, McIntyre KM, Capone RJ, Kostis J, Niaura R, Woods P, et al. Assessment of quality of life as observed from the baseline data of the Studies of Left Ventricular Dysfunction (SOLVD) trial quality-of-life substudy. *Am J Cardiol* 1993;71:1069–1073.

12. Jennett BM. Assessment of outcome after severe brain damage: a practical scale. *Lancet* 1995;1:480–484.

13. Lombardi G, Gallagher EJ, Gennis P. Outcome of out-of-hospital cardiac arrest in New York City: the Pre-hospital arrest survival evaluation (Phase) study. *JAMA* 1994;271:678–683.

14. White RD, Hankins DG, Atkinson EJ. Patient outcomes following defibrillation with a low energy biphasic truncated exponential waveform in out-of-hospital cardiac arrest. *Resuscitation* 2001;49:9–14.

15. White RD, Hankins DG, Bugliosi TF. Seven years' experience with early defibrillation by police and paramedics in an emergency medical services system. *Resuscitation* 1998;39:145–151.

16. Eisenberg MS, Mengert TJ. Cardiac resuscitation. *N Engl J Med* 2001;344: 1304–1313.

17. Eisenberg MS, Horwood BT, Cummins RO, Reynolds-Haertle R, Hearne TR. Cardiac arrest and resuscitation: a tale of 29 cities. *Ann Emerg Med* 1990;19: 179–186.

18. Kellerman AL, Hackman BB, Somes G, Kreth TK, Nail L, Dobyns P. Impact of first-responder defibrillation in an urban emergency medical services system. *JAMA* 1993;270:1708–1713.

19. Myerburg RJ, Fenster J, Velez M, Rosenberg D, Lai S, Kurlansky P, Newton S, Knox M, Castellano A. Impact of community-wide police car deployment of automated external defibrillators on survival from out-of-hospital cardiac arrest. *Circulation* 2002;106:1058–1064.

Effect of Carotid Atherosclerosis Screening on Risk Stratification During Primary Cardiovascular Disease Prevention

Robert L. Bard, MA, Henna Kalsi, MD, Melvyn Rubenfire, MD, Thomas Wakefield, MD, Beverly Fex, RVT, Sanjay Rajagopalan, MD, and Robert D. Brook, MD

We investigated the effect that carotid plaque area (CPA) and intima media thickness (IMT) measurements have on risk stratification in 95 patients with intermediate Framingham scores (6% to 19%). The risk status of each patient was adjusted to be low, intermediate, or high based on the results of carotid ultrasound. After carotid testing, 44% (IMT) and 45% (CPA) of the intermediate-risk patients were stratified as low risk, and 22% (IMT) and 40% (CPA) were stratified as high risk. Using the threshold values derived from our laboratory, 28% (IMT) and 45% (CPA) of patients were stratified as low risk, and 35% (IMT) and 27% (CPA) were identified as high risk. These tests adjust the risk strata of ≥63% of patients deemed as having intermediate risk by Framingham scores.

(Am J Cardiol 2004;93:1030–1032)

From the Division of Cardiovascular Medicine and Section of Vascular Surgery, Department of Surgery, University of Michigan, Ann Arbor, Michigan. Dr. Brook's address is: 3918 Taubman Center, 1500 East Medical Center Drive, Ann Arbor, Michigan 48109. E-mail: robdbrok@umich.edu.

Manuscript received September 29, 2003; revised manuscript received and accepted December 24, 2003.

Measurement of carotid intima media thickness (IMT) improves cardiovascular risk assessment, particularly in patients with intermediate Framingham scores.[1,2] An abnormal IMT is an independent predictor of increased cardiovascular events,[3–8] and it has been suggested that patients with values >1.0 mm should be treated more aggressively than similar patients with a normal IMT.[1] It is unknown how many intermediate-risk patients' therapies would change based on carotid ultrasound results. Therefore, we investigated the effect of carotid IMT and carotid

0002-9149/04/$–see front matter
doi:10.1016/j.amjcard.2003.12.056

TABLE 1 Clinical Characteristics of the Intermediate-risk Patients (n = 95)

Age (yrs)	56 ± 9
Men/women	59/36
Positive cardiovascular family history*	32 (34%)
Current smoker	9 (9%)
Systemic hypertension†	52 (55%)
Hyperlipidemia (history)‡	19 (20%)
Systolic blood pressure (mm Hg)	124 ± 20
Diastolic blood pressure (mm Hg)	78 ± 10
Total cholesterol (mg/dl)	217 ± 60
High-density lipoprotein (mg/dl)	46 ± 15
Low-density lipoprotein (mg/dl)	132 ± 50
Triglycerides (mg/dl)	176 ± 120

*Positive family history is equal to first-degree relative with documented cardiovascular disease or event at <55 (male relative) or <65 (female relative) years of age.

†Hypertension denotes previous diagnosis of elevated blood pressure and/or currently on antihypertensive medication.

‡Hyperlipidemia history denotes previous diagnosis of elevated serum lipoproteins and/or currently on lipid-lowering medications.

TABLE 2 Risk Assessment Results from the 95 Intermediate-Risk Patients

Test	Mean ± SD
Framingham risk score (%)*	10.2 ± 3.6
Carotid IMT (mm)	0.82 ± 0.03
CPA (mm^2)	18.8 ± 3.8

*Framingham risk score is equal to the absolute risk of "hard" cardiovascular disease events (myocardial infarction, cardiovascular death, or new-onset unstable angina) within a 10-year period.

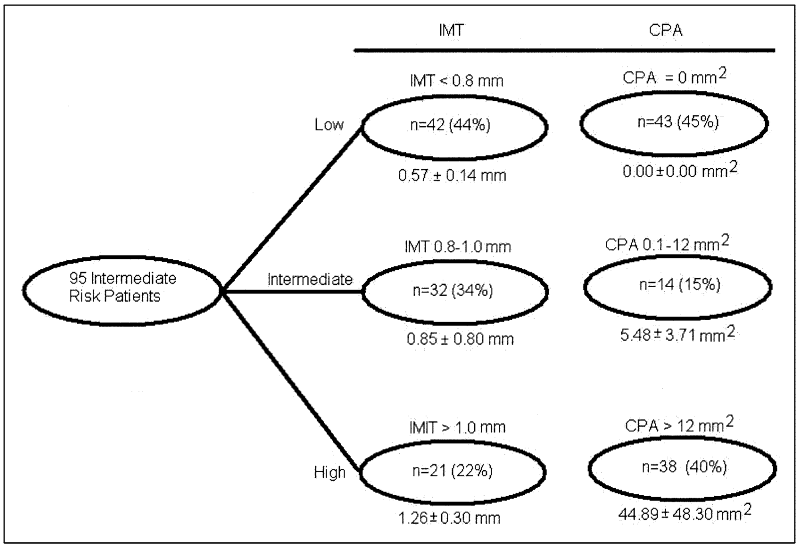

FIGURE 1. Effect of carotid IMT and CPA on subsequent risk stratification based on criteria from the research literature. Risk category criteria are listed above each bubble. Mean values for each category are listed below the bubbles in the flowchart. Absolute number and percentage of patients stratified by risk to each category are inside each bubble.

plaque area (CPA) measurements on the risk stratification of patients with intermediate Framingham risk scores.

• • •

This project was approved by the institutional review board of the University of Michigan Medical School. We performed a retrospective analysis of the first 200 consecutive patients who had carotid IMT and CPA tests performed clinically. Data were obtained from each patient to calculate a Framingham risk score.

Patients were risk stratified[1,2] as low (≤5%), intermediate (6% to 19%), or high (≥20%) risk by their Framingham risk scores. Only intermediate-risk patients (n = 95) without established cardiovascular disease or risk equivalents (peripheral vascular disease, diabetes mellitus, symptomatic carotid disease, and aortic disease) were considered for this study because this population's medical management is the most likely to be affected by the results of ultrasound.[1,2]

Risk stratification was adjusted using established IMT[3–8] and CPA[9] values from the literature (IMT risk strata: low <0.80 mm, intermediate 0.80 to 0.99 mm, high ≥1.0 mm; CPA risk strata: low 0 mm^2, intermediate 0.01 to 12 mm^2, high >12.0 mm^2) and the 25th and 75th percentile values from our laboratory. The population of 200 patients included 62 low-, 95 intermediate-, and 43 high-risk patients as defined by the Framingham risk score.

The carotid ultrasound tests were performed in an Intersocietal Commission for the Accreditation of Vascular Laboratories-approved diagnostic vascular unit using a 7.5-MHz linear array transducer connected to a Powervision ultrasound device (Toshiba, Inc., Tustin, California). On-screen measurements of IMT[6,7] and CPA[9] were determined as previously described in the literature.

The clinical characteristics and risk assessment results of the 95 intermediate-risk patients are displayed in Tables 1 and 2, respectively. The median Framingham risk score was 9, the range was 6 to 18, and the 25th and 75th percentiles were 7 and 13, respectively.

The cardiovascular risk status derived from IMT and CPA assessments (using either threshold criterion) differed substantially from the results of the clinical Framingham risk scores (Figures 1 and 2, respectively). The risk stratum was changed in most patients (≥63% of cases). IMT and CPA changed the risk stratum in at least 63% and 73% of cases, respectively (Figures 1 and 2). Further, the IMT and CPA risk assessments differed in most patients (Tables 3 and 4, respectively). IMT and CPA stratified patients identically in only 42% (literature review) and 38% (internal laboratory data) of cases. However, risk stratification differed by >1 level (e.g., low risk by IMT vs

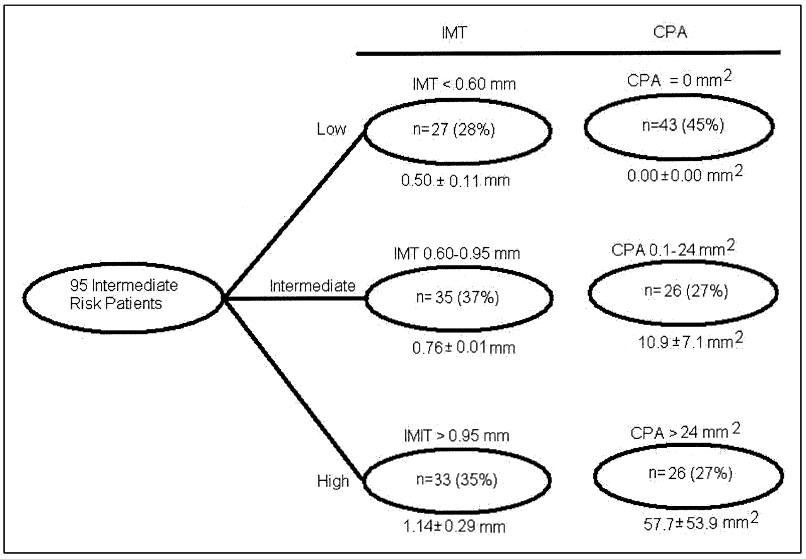

FIGURE 2. Effect of carotid IMT and CPA on subsequent risk stratification based on threshold values from our vascular laboratory. Risk category criteria are listed above each bubble. Mean values for each category are listed below the bubbles in the flowchart. Absolute number and percentage of patients stratified by risk to each category are inside each bubble.

TABLE 3 Agreement Between Carotid IMT and CPA in the Cardiovascular Risk Stratification of Intermediate-risk Patients According to Threshold Values Derived from the Medical Literature*

	IMT		
CPA	Low (<0.8 mm)	Intermediate (0.8–1.0 mm)	High (>1.0 mm)
0 mm^2	23 (24%)	15 (16%)	5 (5%)
0–12 mm^2	7 (7%)	4 (4%)	3 (3%)
>12 mm^2	12 (13%)	13 (14%)	13 (14%)

*Each value represents the number of patients (n = 95).

TABLE 4 Agreement Between Carotid IMT and CPA in the Cardiovascular Risk Stratification of Intermediate-risk Patients According to Threshold Values Derived from Our Vascular Laboratory*

	IMT		
CPA	Low (<0.6 mm)	Intermediate (0.60–0.95 mm)	High (>0.95 mm)
0 mm^2	14 (15%)	20 (21%)	9 (9%)
0–24 mm^2	8 (8%)	8 (8%)	10 (11%)
>24 mm^2	5 (5%)	7 (7%)	14 (15%)

*Each value represents the number of patients (n = 95).

high risk by CPA) in very few situations (14% to 17% of cases).

• • •

Atherosclerosis screening with IMT and CPA changed the risk stratum in most patients deemed as intermediate risk by clinical criteria alone. This result suggests that IMT and CPA may be useful modalities to enhance risk assessment beyond the Framingham risk score. Many patients without known atherosclerotic disease were found to have an abnormal IMT (22% to 35%) or CPA (27% to 40%), thus placing them in the highest-risk category (equivalent to the risk of coronary heart disease).

Established abnormal threshold values for IMT or CPA do not currently exist. Different threshold criteria would probably change the risk assessments. Nevertheless, criteria from 2 different sources produced very similar findings in our study. Additional research is necessary to better define clinically useful CPA and IMT threshold values (e.g., age and risk factor adjusted risk categories) and to determine the long-term clinical outcome of tailoring medical therapy based upon risk assessment modalities.

1. Greenland P, Smith SC, Grundy SM. Improving coronary heart disease risk assessment in asymptomatic people. Role of traditional risk factors and noninvasive cardiovascular tests. *Circulation* 2001;104:1863–1867.
2. Smith SC, Greenland P, Grundy SM. Prevention Conference V. Beyond secondary prevention: identifying the high-risk patient for primary prevention. Executive summary. *Circulation* 2000;101:111–1116.
3. Aminbakhsh A, Mancini GBJ. Carotid intima-media thickness measurements: what defines an abnormality? A systemic review. *Clin Invest Med* 1999;22:149–157.
4. Simon A, Gariepy J, Chironi G, Megnien JL, Levenson J. Intima-media thickness: a new tool for diagnosis and treatment of cardiovascular risk. *J Hypertens* 2002;20:159–169.
5. Mukherjee D, Yadav JS. Carotid artery intimal-medial thickness: indicator of atherosclerotic burden and response to risk factor modification. *Am Heart J* 2002;144:753–759.
6. O'Leary DH, Polak JF, Kronmal RA, Manolio TA, Burke GL, Wolfson SK. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. *N Engl J Med* 1999;340:14–22.
7. Hodis HN, Mack WJ, LaBree L, Selzer RH, Liu CR, Liu CL, Azen SP. The role of carotid arterial intima-media thickness in predicting clinical coronary events. *Ann Intern Med* 1998;128:262–269.
8. del Sol AI, Moons KGM, Hollander M, Hofman A, Koudstaal PJ, Grobbee DE, Breteler MMB, Witteman JCM, Bots ML. Is carotid intima-media thickness useful in cardiovascular disease risk assessment? The Rotterdam Study. *Stroke* 2001;32:1532–1538.
9. Spence JD. Ultrasound measurement of carotid plaque as a surrogate outcome for coronary artery disease. *Am J Cardiol* 2002;89(suppl):10B–16B.