Original Article

Ability of Ambulatory Blood Pressure Monitoring and Myocardial/Carotid Ultrasound to Predict the Location and the Severity of Coronary Artery Lesions in Normotensive Patients: A Clinical Study

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Pulse pressure has been recognized as a marker of cardiovascular disease in normotensives. Moreover, internal carotid artery intima-media thickness (IMT) has been proposed to reflect coronary artery lesions. The aim of the present study was to evaluate the predictive value of other parameters derived from ambulatory blood pressure monitoring (ABPM), myocardial ultrasound, and carotid ultrasound to predict the location and the severity of coronary artery disease in normotensives. One hundred and thirteen patients with suspected coronary artery disease underwent coronary angiography, 24-h ABPM and myocardial/carotid ultrasound. Multivariate analysis was applied and equations were extrapolated based on independent variables derived from ABPM and ultrasound. The Gensini score was independently correlated with male gender, pulse pressure, average heart rate for both 24-h (p=0.001) and night (p=0.006) values, as well as percentage of high systolic blood pressure (BP), average diastolic BP, average mean BP, and heart rate concerning daily mesurements (p=0.001). Moreover, the Gensini score was independently correlated with end-systolic volume, posterior wall thickness during systole and intraventricular septum thickness during diastole, along with male gender and age (p=0.001), as well as mean internal and right common carotid artery IMT (p=0.002). Similar mathematical formulas have been calculated separately for the coronary arteries and their main branches. In conclusion, the location and the severity of coronary disease can be effectively evaluated by ABPM and myocardial/carotid ultrasound in normotensives. This approach could be useful for determining atypical patients at risk and/or for treating patients with suspected coronary disease who refuse coronary angiography. (*Hypertens Res* 2007; 30: 741–749)

***Key Words*: coronary artery disease, ambulatory blood pressure monitoring, intima-media thickness, Gensini score**

Introduction

Pulse pressure has been recognized as a marker of cardiovascular disease in normotensives, as it has been independently correlated with coronary artery lesions, thickening of the intima-media wall of the internal carotid artery and increase in left ventricle mass in normotensives (*1*). Moreover, carotid

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Table 1. Baseline Patient Characteristics: Demographic, Biochemical, ECG, Rx and Coronary Angiography Variables

Variables	Mean (95% CI)	Mean	
	Total	Men	Women
Age (years)	60.7 (58.9–62.5)	60.99	59.55
Height (cm)	169.3 (162.7–175.9)	171.6	160.3
Weight (kg)	78.4 (68.6–88.2)	79.5	74.3
BSA (m^2)	1.88 (1.85–1.91)	1.90	1.81
BMI (kg/m^2)	27.4 (24.3–30.5)	26.9	28.9
Blood glucose (mg/dL)	111.8 (104.7–118.9)	111.6	112.6
Blood creatinine (mg/dL)	1.19 (1.11–1.28)	1.24	0.99
Blood uric acid (mg/dL)	6.28 (5.93–6.63)	6.62	4.95
Total cholesterol (mg/dL)	228.2 (219.4–237.6)	222.3	251.3
Triglycerides (mg/dL)	166.0 (152.3–179.7)	165.3	168.8
High-density lipoprotein cholesterol (mg/dL)	48.1 (46.6–49.6)	47.7	49.8
Low-density lipoprotein cholesterol (mg/dL)	149.8 (140.3–159.3)	141.5	167.7
Lp(a) (mg/dL)	28.4 (27.2–29.5)	28.9	26.6
Apo A (mg/dL)	127.2 (121.6–132.8)	126.2	131.3
Apo B (mg/dL)	135.8 (125.6–146.0)	134.8	139.8
Sokolow index	18.0 (16.5–19.4)	18.3	16.8
CT index (%)	46.7 (45.4–48.0)	47.1	45.1
Smoking habits (%)	39.8	36.6	52.2
LM (%)	2.0 (−0.2–4.2)	2.5	0.0
LAD (%)	63.2 (56.1–70.3)	71.7	29.8
D1 (%)	9.1 (4.3–13.9)	9.3	8.5
LCX (%)	40.9 (32.9–49.0)	45.2	23.9
RCA (%)	35.1 (27.1–43.1)	40.7	13.0
AD (Gensini)	38.9 (33.0–44.7)	44.3	17.9

Rx, chest X-ray; CI, confidence interval; BSA, body surface area; BMI, body mass index; Lp(a), lipoprotein(a); Apo, apolipoprotein; LM, left cornary artery stem; LAD, left anterior descending ramus; D1, diagonal artery; LCX, left circumflex artery; RCA, right coronary artery.

artery intima-media thickness (IMT) has been proposed to reflect the severity of coronary artery lesions (*2*, *3*). However, other parameters from ambulatory blood pressure monitoring (ABPM), myocardial ultrasound and carotid ultrasound have not been evaluated in terms of their ability to predict the location and the severity of coronary artery disease in normotensives.

Much effort has been concentrated on the diagnosis of coronary artery disease by noninvasive methods. The efficacy of 16- and 64-multislice spiral computed tomography (MSCT) for such diagnosis has been extensively evaluated. The available results show that MSCT is acceptable as a first-step assessment in suspected coronary artery disease (*4*, *5*).

In the present study we constructed mathematical models that use data from three different noninvasive methods, ABPM, myocardial ultrasound and carotid ultrasound, in order to estimate the location and the severity of stenotic lesions in the left coronary artery stem (LM), left anterior descending ramus (LAD), left circumflex artery (LCX), diagonal artery (D1) and right coronary artery (RCA) as well as to estimate the Gensini score (AD) in normotensive patients with suspected coronary disease.

Methods

One hundred and thirteen consecutive patients (90 men and 23 women) with normal blood pressure (systolic blood pressure [SBP] <130 mmHg and diastolic blood pressure [DBP] <80 mmHg) (*6*), who were hospitalized for suspected coronary artery disease, were included in the study. One hundred and seven of these patients had already undergone 24-h ABPM and 88 had already undergone carotid and myocardial ultrasound on an outpatient basis on the same day, 3–7 days prior to coronary angiography. Age, body surface area (BSA), blood biochemistry, Sokolow index and cardiothoracic index were recorded for each patient at admission.

Inclusion criteria were a history of normal blood pressure, a normal 24-h ABPM, symptoms indicative of angina pectoris, and a positive exercise stress test. Exclusion criteria were a previous myocardial infarction history and the need for nitrates, β-blockers, Ca-antagonists or other antihypertensive regimens due to instability. All patients received aspirin 100 mg daily and statins if their low-density liporotein (LDL) cholesterol was greater than 100 mg/dL.

Table 2. Ambulatory Blood Pressure Variables (Abbreviations and Mean Values with 95% Confidence Intervals)

Variables	24-h	Daytime (6:00–22:00)	Nighttime (22:00–6:00)
Average systolic BP	S_MEA_24	S_MEA_D	S_MEA_N
	119.9 (117.3–122.4)	121.6 (119.1–124.1)	116.7 (113.9–119.5)
SD of systolic BP	S_SD_24	S_SD_D	S_SD_N
	10.6 (10.0–11.2)	10.5 (10.0–11.1)	8.8 (8.2–9.4)
Average diastolic BP	D_MEA_24	D_MEA_D	D_MEA_N
	72.2 (70.6–73.9)	73.8 (72.2–75.4)	69.6 (67.7–71.5)
SD of diastolic BP	D_SD_24	D_SD_D	D_SD_N
	8.5 (8.0–9.0)	8.3 (7.9–8.8)	7.1 (6.5–7.6)
Average mean BP	M_MEA_24	M_MEA_D	M_MEA_N
	87.9 (86.0–89.9)	89.5 (87.6–91.4)	85.1 (83.0–87.3)
Average pulse pressure	PP_24	PP_D	PP_N
	47.6 (46.0–49.3)	47.8 (46.1–49.5)	47.1 (45.3–48.9)
% measurements of systolic BP >140 mmHg	S_140_24	S_140_D	S_140_N
	10.2 (6.6–13.7)	11.8 (8.1–15.5)	7.2 (3.6–10.8)
% measurements of diastolic BP >90 mmHg	D_90_24	D_90_D	D_90_N
	7.8 (4.8–10.7)	8.3 (5.5–11.0)	5.6 (2.6–8.6)
Average heart rate	R_MEA_24	R_MEA_D	R_MEA_N
	68.9 (66.8–71.0)	70.6 (68.4–72.8)	66.3 (64.2–68.4)

BP, blood pressure.

Table 3. Myocardial and Carotid Ultrasound Parameters (Abbreviations and Mean Values with 95% Confidence Intervals)

Variables	Abbreviation explanation	Mean (95% CI)	Mean	
			Men	Women
IVSD (cm)	Intraventricular septum–diastole	1.17 (0.97–1.37)	1.18	1.12
IVSS (cm)	Intraventricular septum–systole	1.57 (1.52–1.62)	1.58	1.54
PWD (cm)	Posterior wall thickness–diastole	0.91 (0.88–0.94)	0.92	0.88
PWS (cm)	Posterior wall thickness–systole	1.72 (1.67–1.76)	1.74	1.66
LVEDD (cm)	Left ventricle end-diastolic diameter	5.27 (5.14–5.40)	5.31	5.12
LVESD (cm)	Left ventricle end-systolic diameter	3.27 (3.13–3.42)	3.29	3.19
EDV_TEIC (mL)	End-diastolic volume	135.8 (127.7–143.9)	137.0	131.0
ESV_TEIC (mL)	End-systolic volume	47.5 (42.5–52.6)	47.7	46.6
LVmass/BSA	Left ventricular mass/BSA	121.6 (89.6–153.6)	124.2	111.4
RICA (mm)	Right internal carotid artery IMT	0.81 (0.73–0.90)	0.81	0.81
RCCA (mm)	Right common carotid artery IMT	0.74 (0.69–0.78)	0.76	0.66
LICA (mm)	Left internal carotid artery IMT	0.79 (0.72–0.85)	0.82	0.69
LCCA (mm)	Left common carotid artery IMT	0.81 (0.75–0.87)	0.83	0.72
MICA (mm)	Mean internal carotid artery IMT	0.80 (0.73–0.87)	0.82	0.75
MCCA (mm)	Mean common carotid artery IMT	0.77 (0.72–0.82)	0.80	0.69

CI, confidence interval; BSA, body surface area; IMT, intima-media thickness.

All patients offered their informed consent to participate in the study. The study was approved by the University of Athens and conformed to all ethical issues included in the Helsinki declaration.

Coronary artery disease was established by coronary angiography, and luminal narrowing in LM, LAD, D1, LCX and RCA were recorded. The severity of coronary lesions was evaluated by the Gensini score (AD), which depends on the degree of luminal narrowing and the geographic importance of each stenosis (*7*). The Gensini scoring system incorporates data on the geometrically increasing severity of lesions, the cumulative effects of multiple obstructions, the significance of their locations, the modifying influence of the collaterals, the size and quality of the distal vessels and the status of the myocardial function (*8*). The Gensini score is commonly used for assessing coronary artery disease severity (*9*).

For ABPM, patients were instructed to act and work as normal between 6:00 AM and 10:00 PM and rest or sleep

Table 4. Multiple Regression Analysis (Stepwise Method) of ABPM (a: 24-h Data, b: Daytime Data, c: Nighttime Data) against Stenotic Lesions (Expressed as % Luminal Narrowing) in LM, LAD, LCX, RCA and D1 as well as Gensini Score

Dependent variable	*N*	*F*	*p*	Intercept	Variables in equation	β	Equation
a: 24-h data							
LM	107	4.14	0.004	−12.927	S_SD_24	0.518	LM = 1.810 × S_SD_24 − 1.432 × D_SD_24 + 0.115 × R_MEA_24 − 3.001 × SEX − 12.927
					D_SD_24	−0.370	
					R_MEA_24	0.127	
					SEX	−0.120	
LAD	107	4.68	0.004	64.201	SEX	−0.230	LAD = −22.092 × SEX + 0.616 × AGE − 0.511 × R_MEA_24 + 64.201
					AGE	0.155	
					R_MEA_24	−0.150	
D1	107	3.68	0.008	24.512	S_140_24	0.615	D1 = 0.806 × S_140_24 − 3.801 × S_SD_24 − 0.613 × D_90_24 + 2.427 × D_SD_24 + 24.512
					S_SD_24	−0.440	
					D_90_24	−0.390	
					D_SD_24	0.256	
LCX	107	1.96	0.165	43.847	SEX	−0.140	LCX = −14.302 × SEX + 43.847
RCA	107	2.67	0.026	93.952	SEX	−0.250	RCA = −26.152 × SEX + 1.492 × S_SD_24 − 1.515 × D_MEA_24 + 0.485 × S_140_24 + 0.506 × R_MEA_24 + 93.952
					S_SD_24	0.100	
					D_MEA_24	−0.310	
					S_140_24	0.213	
					R_MEA_24	0.131	
AD (Gensini)	107	5.61	0.001	−31.273	SEX	−0.290	AD = −21.809 × SEX + 0.999 × PP_24 + 0.379 × R_MEA_24 − 31.273
					PP_24	0.288	
					R_MEA_24	0.136	
b: Daytime data							
LM	107	3.47	0.035	0.358	S_140_D	0.346	LM = 0.177 × S_140_D − 0.116 × D_90_D + 0.358
					D_90_D	−0.170	
LAD	107	5.10	0.002	72.381	SEX	−0.230	LAD = −21.993 × SEX − 0.582 × R_MEA_D + 0.578 × AGE + 72.381
					R_MEA_D	−0.180	
					AGE	0.146	
D1	107	2.10	0.071	53.055	S_140_D	0.619	D1 = 0.773 × S_140_D − 0.301 × S_MEA_D − 2.660 × S_SD_D − 0.451 × D_90_D + 1.698 × D_SD_D + 53.055
					S_MEA_D	−0.160	
					S_SD_D	−0.320	
					D_90_D	−0.270	
					D_SD_D	0.176	
LCX	107	1.96	0.165	43.847	SEX	−0.140	LCX = −14.302 × SEX + 43.847
RCA	107	3.91	0.003	180.637	SEX	−0.220	RCA = −23.642 × SEX + 2.764 × S_SD_D − 1.762 × D_MEA_D + 0.830 × S_140_D − 1.041 × PP_D + 180.637
					S_SD_D	0.191	
					D_MEA_D	−0.350	
					S_140_D	0.382	
					PP_D	−0.220	
AD (Gensini)	107	5.61	0.001	29.263	S_140_D	0.209	AD = 0.326 × S_140_D − 21.908 × SEX − 2.698 × D_MEA_D + 2.035 × M_MEA_D + 0.370 × R_MEA_D + 29.263
					SEX	−0.290	
					D_MEA_D	−0.740	
					M_MEA_D	0.664	
					R_MEA_D	0.141	
c: Nighttime data							
LM	106	1.54	0.210	−18.373	S_SD_N	0.100	LM = 0.336 × S_SD_N + 0.124 × R_MEA_N + 0.144 × AGE − 18.373
					R_MEA_N	0.138	
					AGE	0.136	
LAD	106	4.43	0.006	64.439	SEX	−0.230	LAD = −22.602 × SEX − 0.464 × R_MEA_N + 0.549 × AGE + 64.439
					R_MEA_N	−0.140	
					AGE	0.139	

Table 4. Continued

Dependent variable	*N*	*F*	*p*	Intercept	Variables in equation	β	Equation
D1	106	3.56	0.032	6.736	S_140_N	0.372	D1 = 0.482 × S_140_N − 0.351 × D_90_N + 6.736
					D_90_N	−0.220	
LCX	106	1.84	0.100	1.958	S_SD_N	0.181	LCX = 2.566 × S_SD_N − 20.126 × SEX − 0.631 × S_140_N
					SEX	−0.190	+ 2.479 × M_MEA_N − 2.072 × D_MEA_N − 0.696
					S_140_N	−0.280	× AGE + 1.958
					M_MEA_N	0.667	
					D_MEA_N	−0.480	
					AGE	−0.160	
RCA	106	3.06	0.051	9.185	SEX	−0.200	RCA = −21.242 × SEX + 0.500 × AGE + 9.185
					AGE	0.112	
AD (Gensini)	106	4.41	0.006	−15.914	SEX	−0.280	AD = −21.608 × SEX + 0.741 × PP_N + 0.358 × R_MEA_N
					PP_N	0.225	− 15.914
					R_MEA_N	0.130	

LM, left cornary artery stem; LAD, left anterior descending ramus; D1, diagonal artery; LCX, left circumflex artery; RCA, right coronary artery.

between 10:00 PM and 6:00 AM. They had not received any antihypertensive drug treatment at any time. They had not been given any other type of drug that might affect their blood pressure level for at least 2 weeks before entering the study. The Spacelabs 90209 ambulatory blood pressure monitor (Spacelabs Inc., Redmond, USA) was used. Readings were obtained automatically at 15-min intervals throughout a 24-h study period. All patients evaluated had at least three valid readings per hour. As a result, 80 to 96 pairs of SBP and DBP readings per recording were collected. Separate averages were obtained for the 24-h, daytime (6:00 AM–10:00 PM) and nighttime (10:00 PM–6:00 AM) values. The accuracy of the automatic blood pressure readings was checked against manual readings taken using a standard mercury sphygmomanometer, twice for each ABPM. Blood pressure was measured with the patient sitting, before the beginning of the ABPM, and after a 5-min rest period. Three readings were obtained and averaged. The accuracy test was repeated after the end of each 24-h ABPM. Patients with a more than 5 mmHg difference in SBP between the manual and automatic reading were to be excluded from further analysis, but there were no such patients in this study.

All patients were examined in the supine position with the head slightly elevated. The scans were performed with a high resolution ultrasound Doppler system (Acuson 128 XP; Acuson, Mountain View, USA) using a 7-MHz linear transducer. Both carotid arteries were scanned longitudinally to visualize the IMT in the far wall of the artery. The best images of the far wall that could be obtained were used to determine the IMT values of the common carotid and internal carotid arteries.

Measurements were made on frozen images, magnified to standard size, on-line. The IMT of the common carotid artery (MCCA) was defined as the mean of the IMT of the right (RCCA) and left common carotid artery (LCCA), calculated from 10 measurements on each side, taken within 10 mm proximal to the carotid bifurcation. The lumen/intima leading edge (I line) to media/adventitia leading edge (M line) method was used, which has been previously anatomically validated (*10*, *11*). The proximal 1.0 cm of the internal carotid artery was used for the calculation of the IMT (MICA), again as the mean of the right (RICA) and left internal carotid artery (LICA).

All echocardiograms were obtained by a skilled sonographer using a Sigma-1C echocardiograph (Kontron Instruments Inc., Everett, USA) and a 3.5-MHz transducer. Two-dimensional guided M-mode echocardiograms at the level of the chordae tendineae were recorded. Numbers were randomly assigned to all echocardiograms, blinding all patient identification and time sequences. Each echocardiogram was read by two expert echocardiographers. Four to six cycles of septal and posterior wall thickness and left ventricular (LV) internal diastolic and systolic dimensions were measured by each reader, using the guidelines of the American Society of Echocardiography. The American Society of Echocardiography convention marks diastole as the onset of the QRS complex and measures wall thickness and chamber dimensions from leading edge to leading edge. The average of mean measurements provided by the two investigators for each echocardiogram were used in all calculations (*12*).

Left ventricular mass (LVmass) was calculated using the following equation, based on necropsy validation studies (*13*):

$$\text{LVmass (g)} = 0.8 \times [1.04 \times (\text{IVSd} + \text{LVDd} + \text{PWTd})^3 - (\text{LVDd})^3] + 0.6;$$

$$\text{LVmass index} = \text{LVmass/BSA},$$

where IVSd is interventricular septal thickness at end-dias-

tole, LVDd is LV internal dimension at end-diastole, PWDd is posterior wall thickness at the end-diastole, and BSA is the body surface area.

All data were analyzed using SPSS for Windows 9.5 (SPSS Inc. Chicago, USA). Standard descriptive statistics, simple correlation analyses and multivariate linear regression analyses (stepwise entry technique) were performed where appropriate. The level of statistical significance was set to $p<0.05$. All mean values are accompanied by their 95% confidence intervals.

Results

Data concerning baseline patient characteristics are presented in Table 1. Specifically, age, anthropometric indices, basic biochemistry, Sokolow index, cardiothoracic index (CT index), and the percent luminal narrowing of LM, LAD, LCX, RCA, D1 and AD are included (*14*, *15*).

Ambulatory blood pressure variables (24-h, daytime and nighttime) are presented in Table 2, and data derived from myocardial and carotid ultrasound are presented in Table 3.

All data included in Tables 2 and 3 were used for multivariate linear regression analyses (stepwise method) against luminal narrowing of LM, LAD, LCX, RCA, D1, and AD (expressed as a percentage of luminal narrowing).

The results of the multiple regression analysis of ABPM parameters against stenotic lesions (expressed as a percentage of luminal narrowing) in LM, LAD, LCX, RCA, D1 and AD are shown in Table 4. Similarly, results from the analysis of the myocardial and carotid ultrasound parameters are presented in Table 5.

Discussion

In the present study, we constructed mathematical models that use data from noninvasive techniques to evaluate the possibility of coronary disease in normotensive patients. In our study, we used three different noninvasive models, ABPM, myocardial ultrasound and carotid ultrasound in order to mathematically assess the possibility of stenotic lesions in LM, LAD, LCX, RCA and D1 (expressed as a percentage of luminal narrowing) as well as to estimate AD. This procedure involved the use of all raw data from ABPM, myocardial ultrasound and coronary ultrasound of the study patients as independent variables in multiple regression analysis against the data obtained from coronary angiography of the same patients as dependent variables. Thus, several parameters from both ABPM as well as myocardial and carotid ultrasound parameters were independently correlated with the location and the severity of coronary artery disease as expressed by the Gensini score. This implies that these parameters would be very potent as independent predictors of coronary artery disease, in cases where invasive methods, such as coronary angiography, cannot be performed.

Our results underscore the clinical significance of several ABPM parameters in the assessment of coronary artery disease in normotensive patients. The Gensini score has been found to be independently correlated with male gender as well as average pulse pressure and average heart rate for both 24-h ($AD=-21.809\times SEX+0.999\times PP\_24+0.379\times R\_MEA\_24-31.273$) and nighttime ($AD=-21.608\times SEX+0.741\times PP\_N+0.358\times R\_MEA\_N-15.914$) values. As far as daytime ABPM values are concerned, the Gensini score is influenced by the percentage of high SBP measurements as well as the average diastolic and mean blood pressure and heart rate ($AD=0.326\times S\_140\_D-21.908\times SEX-2.698\times D\_MEA\_D+2.035\times M\_MEA\_D+0.370\times R\_MEA\_D+29.263$). It is interesting that the overall influence of gender was similar in the three equations that showed the dependence of AD on 24-h, daytime, and nighttime ABPM values. Moreover, increased blood pressure variability might play a crucial role in the formation of coronary artery lesions, as suggested by the above equation.

Moreover, as far as myocardial ultrasound parameters are concerned, the Gensini score has been demonstrated to be independently correlated with end-systolic volume, posterior wall thickness during systole (PWS), and intraventricular septum thickness during diastole (IVSD), along with male sex and age ($AD=0.410\times ESV\_TEIC+28.135\times PWS+4.703\times IVSD-8.883\times SEX+0.342\times AGE-54.248$). This evidence is supported by the fact that increased ESV_TEIC has been correlated with heart failure (*16*), and both increased PWS and IVSD have been correlated with diastolic dysfunction of the left ventricle, all of which might have a common denominator, *i.e.*, carotid artery disease (*17*).

The possible efficacy of carotid ultrasound for the assessment of coronary artery disease is reflected in the suggested independent correlations of Gensini score with mean internal carotid artery IMT (positive) and right common carotid artery (negative), as described in the equation

$$AD=-29.324\times SEX+36.778\times MICA-26.934\times RCCA+35.231.$$

This finding suggests that lesions in the left carotid arteries (both common and internal) might be better correlated with coronary artery disease than lesions in the right carotid arteries. Again, the influence of male gender is detrimental.

It is of great interest that similar formulas have been drawn for the LM, LAD, LCX, RCA and D1, thus linking the topography of the suspected coronary stenotic lesions with data derived from the above mentioned noninvasive methods and providing a more detailed tool.

Much effort has been concentrated on the diagnosis of coronary artery disease by noninvasive methods. The efficacy of 16- and 64-MSCT for such diagnosis has been extensively evaluated. A recent study comparing 16-MSCT with coronary angiography in patients suspected of having coronary artery disease suggests that the method has 81% accuracy, 99% specificity and 96% sensitivity. Nevertheless, despite its documented value and its promising future, the usefulness of

Table 5. Multiple Regression Analysis (Stepwise Method) of Ultrasound Parameters (a: Myocardium, b: Carotid) against Stenotic Lesions (Expressed as % Luminal Narrowing) in LAD, LCX, RCA and D1 as well as Gensini Score

Dependent variable	*N*	*F*	*p*	Intercept	Variables in equation	β	Equation
a: Myocardium							
LM	104	2.39	0.056	−37.924	BSA	0.170	LM = 10.371 × BSA + 0.156 × AGE + 0.059 × ESV_TEIC
					AGE	0.146	+ 4.507 × PWS − 37.934
					ESV_TEIC	0.144	
					PWS	0.107	
LAD	104	3.60	0.003	−46.963	SEX	−0.140	LAD = − 13.066 × SEX + 33.813 × PWS + 0.614 × AGE + 0.188
					PWS	0.211	× ESV_TEIC + 50.009 × PWD − 25.581 × IVSS − 46.963
					AGE	0.151	
					ESV_TEIC	0.121	
					PWD	0.211	
					IVSS	−0.170	
LCX	104	6.17	0.001	−138.931	LVEDD	0.638	LCX = 39.865 × LVEDD − 0.424 × EDV_TEIC + 8.242
					EDV_TEIC	−0.410	× LVESD − 138.931
					LVESD	−0.145	
RCA	104	2.60	0.079	9.391	SEX	−0.190	RCA = − 19.779 × SEX + 0.489 × AGE + 9.391
					AGE	0.109	
AD (Gensini)	104	4.70	0.001	−54.248	ESV_TEIC	0.323	AD = 0.410 × ESV_TEIC + 28.135 × PWS + 4.703 × IVSD
					PWS	0.215	− 8.883 × SEX + 0.342 × AGE − 54.248
					IVSD	0.156	
					SEX	−0.110	
					AGE	0.103	
b: Carotid							
LAD	88	3.24	0.016	44.713	SEX	−0.340	LAD = − 32.646 × SEX + 21.331 × LICA − 39.806 × MCCA
					LICA	0.178	+ 0.579 × AGE + 44.713
					MCCA	−0.210	
					AGE	0.136	
D1	88	2.48	0.050	−19.816	MICA	0.118	LCX = 9.388 × MICA − 17.754 × RCCA + 14.857 × LICA
					RCCA	−0.180	+ 0.350 × AGE − 19.816
					LICA	0.205	
					AGE	0.136	
LCX	88	3.14	0.048	22.662	LICA	0.200	LCX = 25.858 × LICA − 16.357 × SEX + 22.662
					SEX	−0.160	
RCA	88	10.96	0.000	−8.933	MICA	0.425	RCA = 60.841 × MICA − 22.646 × SEX − 8.933
					SEX	−0.210	
AD (Gensini)	88	5.40	0.002	35.231	SEX	−0.350	AD = − 29.324 × SEX + 36.778 × MICA − 26.934 × RCCA
					MICA	0.326	+ 35.231
					RCCA	−0.200	

LM, left cornary artery stem; LAD, left anterior descending ramus; D1, diagonal artery; LCX, left circumflex artery; RCA, right coronary artery.

MSCT has certain limitations, mainly due to artifacts from calcium, as well as the small size of the most distal coronary branches (*4*). These limitations are underscored by the findings of a very recent study concerning the application of MSCT for the evaluation of stent patency after left main coronary artery stenting: the positive predictive value of the method was found to be only 67%, as 5 of 15 patients were erroneously misclassified as having in-stent restenosis (*5*). In a very recent study, MSCT was proposed to be more effective than MRI in detecting lesions in large coronary arteries (*18*).

As far as ultrasound in large arteries is concerned, a potent correlation between carotid artery IMT and coronary artery disease was proposed some time ago (*19*). In a clinical study evaluating acute chest pain, the combination of stenotic lesions in carotid arteries (>50%) and low ejection fraction was demonstrated to effectively predict coronary artery disease (*16*). Increased IMT of the internal and common carotid artery has also been shown to predict coronary artery stenotic

lesions (*3*). A recent study indicates that the IMT score, a scoring system derived from data of the internal carotid artery, common carotid artery, carotid bifurcation and femoral artery, is much better correlated with the extent of coronary atherosclerosis than with individual IMT. This approach also has a documented predictive value as based on multiple regression analysis and is in keeping with our own results concerning carotid ultrasound (*20*). The above results are supported by a recently reformed autopsy study, in which internal and common carotid IMT measurements were demonstrated to be good predictors of coronary artery disease, but not of collateral circulation (*21*).

Concerning ABPM, it has been demonstrated that increased pulse pressure in normotensive patients, attributed to reduced arterial compliance, is a good predictor of cardiovascular disease, as it has been documented to have independent correlation with coronary artery disease as expressed by the Gensini score. Nevertheless, this study failed to demonstrate a statistically significant independent correlation of other ABPM parameters with the Gensini score (*1*). Additionally, pulse pressure has been shown to correlate with atherosclerosis in the large Rotterdam Study (*22*). This phenomenon has been clearly shown to have clinical implications for the long-term treatment of stroke patients (*23*), and might have even greater implications for the elderly (*24*).

The mathematical formulas proposed in the present study are believed to be of value in the clinical treatment of normotensive patients with suspected coronary disease. Because there may be important limitations to this model, especially in regard to the estimated location of lesions, a clinical assessment using an ROC analysis, compared with the gold standard technique of classical coronary angiography, would be very helpful in further testing its predictive value. Nevertheless, a high estimated value for the Gensini score, along with the presumed topography, could suggest the need for invasive methods of assessment of carotid artery disease in patients who show atypical symptoms or are unwilling to undergo coronary angiography.

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