Coll. Antropol. **35** (2011) 1: 167–172
Original scientific paper

Correlation of Cardio-Ankle Vascular Index, Ten-Year Risk Assessment and Other Atherosclerosis Risk Factors

Ljiljana Mayer[1], Vesna Bačić-Vrca[2,3], Pavao Šulentić[1], Ivan Sisić[1], Vlasta Marić-Miholić[1], Slobodan Romovski[3] and Draga Ljubić[1]

[1] »Sveti Rok« Polyclinic, Zagreb, Croatia
[2] University of Zagreb, Dubrava University Hospital, Zagreb, Croatia
[3] University of Zagreb, Faculty of Pharmacy and Biochemistry, Zagreb, Croatia

A B S T R A C T

The aim of the study was to assess correlation of atherosclerosis severity as determined by two different methods of screening for atherosclerosis: (A) measurement of the cardio-ankle vascular index-CAVI by use of the VaseraVS-1500 vascular screening device, and (B) Framingham scale scoring. 52 subjects (28 male and 24 female) were enrolled in the study. Classification of study subjects into four quartiles based on theoretically calculated 10-year risk according to Framingham scale (medians:1%, 3%, 4% and 15%) confirmed the risk increase to be associated with a statistically significant increase in CAVI, age and total cholesterol, and a statistically significant decrease in HDL-cholesterol (p<0.001 all). Spearman correlation coefficients showed a statistically significant correlation of 10-year risk with CAVI (p=0.0242; r=0.4494). Study results suggested that simultaneous determination of CAVI and 10-year risk might prove justified. They are not contradictory, the more so, these two parameters showed a significant positive correlation. This test panel yields comprehensible, implying all the possible consequences and highly motivating information that may stimulate the person for lifestyle modification.

Key words: *cardio-ankle vascular index CAVI, atherosclerosis, Framingham scale scoring, cholesterol*

Introduction

Atherosclerosis is known to be the key factor contributing significantly to the development of cardiovascular disease as the leading cause of premature mortality, morbidity and disability in the world as well as in Croatia[1–4]. In European Union countries, the relative percentage of cardiovascular disease in overall mortality is estimated to 42%, whereas in Croatia it is even higher, with a mean of 50.6% (female 57.9% and male 43.4%). In 2007, the most common postmortem diagnoses were ischemic heart disease (n=9767) and cerebrovascular disease (n=8323)[4,5].

The major risk factors for cardiovascular disease development include age, sex, elevated blood pressure, dyslipidemia and diabetes mellitus[6–8]. As some of these risk factors can be influenced by lifestyle modification or medicamentous therapy, timely recognition of the presence of atherosclerosis is of utmost importance[9,10]. This in turn requires identification and validation of appropriate screening methods. Noninvasive radiological methods such as ultrasonographic (US) measurement of arterial wall thickness are considered as good predictors of cardiovascular and total mortality in both hypertensive patients and general population[11,12].

In the last decade, efforts have been invested in search for a more appropriate, i.e. simpler and faster, yet reliable method of atherosclerotic screening. One of the attempts was measuring of brachial-ankle pulse wave velocity, which was indicated in the numerous studies as reliable and reproductible predictive mark in screening of atherosclerotic changes[13,14].

But, according to the results of the compared researches in the last few years, it seems that the CAVI – cardio-ankle vascular index – recent indicator is superior to brachial-ankle pulse wave velocity, probably primarily

Received for publication October 13, 2010

because of the fact that the latter one is subject to changes in blood pressure (BP) during measuring[15].

CAVI expressing the grade of aortic, femoral and tibial artery stiffness, seems quite promising. CAVI is obtained by mathematical calculation using a formula that includes systolic and diastolic pressure values, vascular diameter assessment and vascular diameter changes (elasticity). The result obtained simultaneously for the left and right side of the body, thus at the same time serving as control of the measuring procedure, may help motivate the patient for his/her lifestyle modification, while providing the physician with quantitative data on the effects of different atherosclerosis risk factors. Recent studies have confirmed statistically significant positive correlations of CAVI with age, hypertension, number of risk factors present, systolic blood pressure, and some US indicators[16–18].

According to the other model proposed, the risk of atherosclerosis is expressed as relative percentage, i.e. the probability of undesired cardiovascular event in the next ten years. The model has been derived from the wide Framingham study, as the result of systematic long-term follow up of a great number of subjects (more than 8000). Framingham scale scoring including age, sex, total and HDL-cholesterol concentration, systolic blood pressure, and data on antihypertensive medication[19]. The aim of this study is to compare the results obtained by the two models of atherosclerosis risk assessment, CAVI and Framingham scale scoring and to make correlation with other risk factors: total cholesterol and HDL-cholesterol concentration, systolic blood pressure and age. Such a study protocol implied indirect CAVI validation performed for the first time, to the best of our knowledge, in Europe. The search of the literature revealed it to be the first study assessing the correlation of CAVI with some of the panels assessing the risk of future cardiovascular events available.

Subjects and Methods

Subjects

The study included 52 subjects, 28 (54%) male and 24 (46%) female, age median 54 years. There were comprised persons with stable medical condition during their regular preventive systematic check-up. Exclusion criteria were history of atherosclerotic cardiovascular disease or stroke, of heart failure or obvious heart disease, and diabetes mellitus. This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving patients were approved by the Ethics Committee of Polyclinic Sveti Rok MD. Written informed consent was obtained from all persons.

Methods

Framingham scale scoring

We classified subjects by Framingham scale scoring according to the theoretical risk of coronary heart disease (CHD) during 10 year time. The factors used to calculate the score include age, sex, total cholestorol level, HDL cholesterol level, systolic blood pressure, and smoking habits. The final results are given in the percentage.

Determination of CAVI

CAVI was measured by the use of a Vasera VS-1500 (Fukuda Denshi, Tokyo, Japan) vascular screening device[20]. ECG leads are placed on the wrist; a microphone is placed on the sternum to record heart beats; and cuffs are placed on both arms and ankles. Data obtained by automated measurement are analyzed by the VSS-15 software (Fukuda Denshi, Tokyo, Japan) and results are calculated separately for the left and the right side of the body. Final result is has been obtained by calculating the mean of four consecutive measurements. It is expressed as the parameter of stiffness, β obtained by the following equation: $\beta=(\ln Ps/Pd)\cdot(D/\Delta D)$; in which ln Ps/Pd is natural logarithm of systolic-diastolic pressure ratio, while $\Delta D/D$ is the ratio of wall extensibility.

Determination of total cholesterol and HDL-cholesterol

Fasting blood samples were collected by use of disposable vacuum system without anticoagulant (Becton Dickinson, UK). Serum concentration of total cholesterol and HDL-cholesterol was measured on a Cobas Integra 400+ (Roche Diagnostics, Basel, Switzerland) autoanalyzer using the reagents, calibrators and control sera of the same manufacturer.

Statistics

Statistical data analysis was done by use of the MedCalc ver. 7.3.0.0 software (MedCalc Software, Mariakerke, Belgium). Results on the study parameters were expressed as absolute values, median and range (25^{th}–75^{th} percentile). Mann-Whitney U test was employed to compare the values of all parameters between male and female subjects and for testing the presence of statistically significant difference between L-CAVI and R-CAVI. Kruskal-Wallis test was used to determine the level of statistical significance all of the study parameters among the four groups according to the 10-year risk quartile. The correlation of 10-year risk and other parameters was tested by use of Spearman correlation.

Results

In Table 1 there are shown demographic data as well as values of the determined and measured parameters of all the examinees together and for male and female separately. The value of the level of statistic significance obtained by male *vs.* female is shown in the last right column. Fifty-two subjects (28 male and 24 female) were enrolled in the study. Median of all the examinees age was 54 (50 for male and 55 for female). Approximately 1/5 of the examinees of both sexes were smokers. Median

TABLE 1
DEMOGRAPHIC DATA AND STUDY PARAMETERS FOR ALL, MALE AND FEMALE WITH LEVEL OF STATISTICAL DIFFERENCE BEETWEN SEX

Parameter	All	Male	Female	p#
Number of subjects (%)	52 (100)	28 (54)	24 (46)	
*Age, yer	54 (48–52)	50 (44–56)	55 (37–58)	0.892
Number of smokers (%)	11 (21)	6 (21)	5 (21)	0.464
*Ten-year risk (%)	7 (2–11)	8 (3–15)	6 (2–9)	0.091
*L-CAVI	8.3 (7.4–8.9)	8.4 (7.6–8.8)	8.3 (7.4–8.8)	0.704
*R-CAVI	8.2 (7.4–8.9)	8.2 (7.5–8.9)	8.3 (7.3–8.9)	0.849
*Total cholesterol, mmol/L	5.9 (5.4–6.5)	5.7 (5.5–6.5)	6.1 (5.2–6.3)	0.957
*HDL-cholesterol, mmol/L	1.3 (1.0–1.7)	1.2 (1.0–1.5)	1.4 (1.0–1.7)	0.056
*Systolic blood pressure, mmHg	125 (120–140)	130 (120–140)	125 (120–135)	0.828
Number of subjects with antihypertensive therapy (%)	16 (31)	7 (25)	9 (37)	0.265
Number of subjects with statin therapy (%)	9 (17)	5 (18)	4 (17)	0.379

*results expressed as medians and ranges in parentheses (25^{th}–75^{th} percentile values); CAVI=cardio-ankle vascular index; L-CAVI= left-side CAVI; R-CAVI=right-side CAVI; #level of statistical significance between male and female subjects (Mann-Whitney test)

of 10 year risk according Framingham scale scoring of all the examinees was 7% (8 for male and 6 for female).

The above described parameters did not differ considering the sex (p values were greater than 0.05).

Numerical values of L-CAVI i D-CAVI of all the examinees from 8.3. and 8.2 also did not differ by sex (p= 0.704 and p=0.489).

The concentrations of total cholesterol and HDL-cholestrol (median) of all the examinees were 5.9 and 1.3 mmol/L. Statistically significant difference regarding the sex was also not proved for those two parameters. Median of the measured systolic pressure was 125 mm Hg for all the examinees (130 for male and 125 for female). There has not also been found any statistically significant difference regarding the sex.

From the total number of the examinees of both sexes 16 (31%) of them take the antihypertensive therapy (10-ACE inhibitors, 2-beta blockers, 4-combination of ACE inhibitors and beta blockers). Nine examinees (17%) take statines (4-atorvastatine, 3-simvastatine, 2-fluvastatine).

TABLE 2
TESTING FOR STATISTICALLY SIGNIFICANT DIFFERENCE BETWEEN L-CAVI AND R-CAVI

	L-CAVI	R-CAVI
Median	8.3	8.2
Minimum-maximum	6.4–15.4	6.3–11.1
Median (95% CI)	8.3 (7.4389–8.8900)	8.2 (7.4389–8.8611)
Interquartile range	7.375–8.852	7.575–8.930
Mann-Whitney test	p=0.8642	

CAVI – cardio-ankle vascular index; L-CAVI – left-side CAVI; R-CAVI – right-side CAVI

Table 2 shows results obtained by the methods of descriptive statistics: median with corresponding 95% confidence interval (CI), minimum, maximum and interquartile range for L-CAVI and R-CAVI. The values of the study parameters recorded were quite comparable, as confirmed by Mann-Whitney test. Comparison of L-CAVI and R-CAVI by Mann-Whitney test yielded no statistically significant difference (p=0.8642). In addition, testing for distribution normality produced a high p-value in

TABLE 3
MEDIAN (MIN-MAX) OF STUDY PARAMETERS GROUPED IN QUARTILES ACCORDING TO 10-YEAR RISK VALUE WITH CORRESPONDING LEVELS OF STATISTICAL SIGNIFICANCE AMONG GROUPS

	Main criteria 10-year risk				p#
	1st quartile	2nd quartile	3rd quartile	4th quartile	
Ten-year risk	1 (1–1)	3 (1–4)	4 (4–8)	15 (10–16)	<0.001
Age (yrs)	30 (26–59)	53 (42–58)	54 (38–65)	57 (55–61)	<0.01
L-CAVI	7.6 (6.3–8.3)	8.0 (7.1–9.2)	8.1 (6.4–9.2)	8.8 (7.5–9.8)	<0.01
Total cholesterol (mmol/L)	5.3 (3.9–6.9)	5.4 (4.7–6.1)	5.8 (5.5–8.9)	6.4 (6.0–8.1)	<0.001
HDL-cholesterol (mmol/L)	2.1 (0.8–2.3)	1.3 (1.0–2.0)	1.3 (0.8–2.3)	1.2 (1.0–1.7)	<0.001

#level of statistical significance among four quartiles (Kruskal-Wallis test)

TABLE 4
CORRELATION OF STUDY PARAMETERS WITH 10-YEAR RISK CALCULATED

	L-CAVI	HDL-cholesterol	Total cholesterol	Systolic blood pressure
Correlation coefficient (r)	0.4493	–0.3755	0.3799	0.4748
Level of significance (p)	0.0242	0.0643	0.0611	0.0165
95% Confidence interval	0.0659–0.7171	–0.6711–0.0230	–0.0180–0.6738	0.0980–0.7325

CAVI – cardio-ankle vascular index; L-CAVI – left-side CAVI; #level of statistical significance (Spearman correlation test)

both groups. Therefore, only results for L-CAVI are presented below, with due statistical correctness.

Table 3 shows median (range) of age, L-CAVI, total cholesterol and HDL-cholesterol of study subjects classified according to the theoretically calculated 10-year risk. Kruskal-Wallis test was used to test for statistically significant differences in study parameters among the four groups. The level of statistical significance, presented in the last column, shows the increase in the 10-year risk to be associated with a statistically significant increase in age, L-CAVI and total cholesterol, and a statistically significant decrease in HDL-cholesterol.

Table 4 shows statistical parameters (correlation coefficient, level of statistical significance, and 95% CI of correlation coefficient) obtained by correlation of 10-year risk and L-CAVI, total cholesterol and HDL-cholesterol, and systolic blood pressure. Ten-year risk showed significant positive correlation with L-CAVI ($r=0.4493$, $p=0.0242$) and systolic blood pressure ($r=0.4748$, $p=0.0165$); borderline positive correlation with total cholesterol ($p=0.0611$); and borderline negative correlation with HDL-cholesterol ($p=0.0643$).

Discussion

Recent data of the World Health Organization point to cardiovascular disease as the leading cause of death worldwide. In 2004, 17.1 million people died from cardiovascular disease in the world. The most common diagnoses were coronary (ischemic) heart disease and cerebrovascular disease. Unfortunately, the prognosis is not very promising, suggesting a continuous rise; it is estimated that as many as 23.6 million people will die from cardiovascular disease in 2030[21,22]. Such a scenario is anticipated to be mainly (up to 80%) due to the epidemic of cigarette smoking, inadequate physical activity, inappropriate dietary habits, obesity, elevated blood pressure left untreated, and diabetes mellitus[23]. As all these are significant but modifiable behavioral and environmental risk factors, the national health authorities have focused on their prevention. Efforts are invested to quantitatively reduce the unfavorable prognosis, as it is likely to grow into a huge health as well as political and socioeconomic problem[24].

It has been definitely established that atherosclerosis greatly contributes to the development of cardiovascular disease. The formation of atherosclerotic plaques containing LDL-lipoprotein particles ingested by tissue macrophages is favored by dyslipoproteinemia, i.e. excess of total cholesterol and LDL-cholesterol, and deficiency of atheroprotective HDL-cholesterol[25,26]. Irrespective of the primary cause of oxidation (e.g., by exposure to intensive free radical action, i.e. oxidative stress, or by the action of excess glycation endproducts), the LDL-particle modified by oxidation is even more prone to incorporate into the atherosclerotic plaque in the vascular subendothelial intima[27,28]. Vascular diameter and blood flow change with time, along with an increase in vascular resistance and blood pressure. The circulation hemodynamics is altered, and laminar blood flow turns inappropriately turbulent. These are preconditions for the formation of blood clots that can cause gradual (thrombosis) or abrupt (embolism) obstruction of the vessel and lead to undesired outcome[29]. Therefore, early recognition of atherosclerosis and large-scale population screening for atherosclerosis are probably crucial in the efforts to reduce its long-term sequels. The screening should ideally provide clear, reliable, widely understandable and motivating information that will also stimulate individuals for lifestyle modifications.

CAVI is a relatively novel method of atherosclerotic risk assessment[16,30]. It includes direct measurement of systolic and diastolic blood pressure, arterial diameter and changes in arterial diameter. CAVI result is obtained by entering these variables in the formula. Mathematically, CAVI is the product of the logarithm of systolic to diastolic blood pressure ratio and vessel diameter to vessel diameter change ratio. Final result (CAVI) is a dimensionless numerical index reflecting arterial condition (stiffness) from the heart to the ankle. CAVI is an example of simple, rapid and noninvasive atherosclerotic screening, especially since the instruments equipped with suitable and reliable software to calculate CAVI have become available on the market. However, these devices have only been available for several years now; therefore the relatively small number of related literature reports is quite understandable. In some studies, CAVI was found to be appropriate not only for screening of the general population but also in some target patient groups with high cardiovascular morbidity, such as those with diabetes mellitus and nephropathies[31,32].

A group of authors investigated correlation between CAVI and left ventricular (LV) diastolic dysfunction. The grade of diastolic dysfunction was assessed by US measurement of LV mass index, LV ejection fraction, left atrium diameter, etc. Higher CAVI values were recorded in the group of patients with reduced diastolic function.

Similar to our study, they classified study subjects according to CAVI into 4 quartiles. The percentage of patients with verified diastolic dysfunction was lowest in the first quartile and systematically increased in each of the next quartiles. The correlation of CAVI and grade of LV dysfunction was explained by the multifactorial synergistic effect of aging, diabetes mellitus and hypertension[18]. Exacerbation in terms of increased arterial stiffness and LV diastolic dysfunction is in part attributed to the glycation endproduct effect on collagen and elastin cross-linking, which results in structural histologic changes that may also entail functional consequences[33].

Determination of LV diastolic dysfunction by tissue Doppler US of the heart correlates better with CAVI than classic Doppler US of the heart[34].

Since CAVI as a marker of arterial stiffness is especially elevated in hypertensive patients, some authors propose CAVI to be used in the follow up of patients taking drugs with a marked hypertensive side effect (e.g., sunitinib, a thyrosine kinase inhibitor with anti-angiogenic and anti-tumor effects)[35]. Another study confirmed the statistically significant correlation of CAVI with HbA_{1c} and homocysteine-independent markers of atherosclerosis[16].

The Framingham study investigators determined the risk of cardiovascular disease by use of a multivariable model of logistic regression in a large number of subjects prospectively followed-up and classified according to sex (more than 4500 female and nearly 4000 male subjects). The following variables were used on risk calculation: age, sex, total cholesterol and HDL-cholesterol, smoking habit, systolic blood pressure, and antihypertensive medication[19]. These all are major cardiovascular risk components, along with the parameters recorded in the study. There are other models of risk assessment, e.g. Reynolds risk score for women (taking in account some other biomedical parameters such as hsCRP and HbA_{1c}), HEARTSCORE, QRISK and ASSIGN, the last two including additional family history data as compared with Framingham study[36–39]. We decided to use the Framingham model because the study was originally conducted in a cohort of Caucasian and Afro-American population, and other studies have confirmed it to be successfully extrapolated to the population of Europe, Mediterranean region in particular, and Asia[40].

Expressing the results of all measured variables as quartiles of the 10-year cardiovascular risk percentage may be perceived as the main conclusion of the present study. Categorizing the results in quartiles based on the key parameter value has lately been used in biomedical statistics and we decided to employ it in the present study[18,41,42]. It is an indirect method to confirm reliability of the parameter tested. Thus, study subjects were classified in quartiles (i.e. four groups of the same number) according to the 10-year risk percentage calculated. Categorized in this way, the results entered generated four groups of data for each parameter, in particular those referring to CAVI. It was quite conceivable that the values of total cholesterol, age and systolic blood pressure increased, and HDL-cholesterol decreased consistently in each of the next quartiles. This could be easily explained by the fact that these variables influence the 10-year cardiovascular risk in the same way since the first three parameters contribute positively and HDL-cholesterol negatively to the risk score. However, the statistically significant increase in CAVI in each of the next quartiles *versus* 10-year cardiovascular risk could be perceived as a result confirming the validity of the other method.

Conclusions

Accordingly, concurrent determination of CAVI and 10-year cardiovascular risk may be considered justified. They are not contradictory, the more so, they showed statistically significant positive correlation. This combined methodology provides literature search, mauscript preparation widely comprehensible (with all the possible consequences) and individually highly motivating information for reduce the rate off aggressive risk factors if present, in particular dyslipidemia, hypertension and cigarette smoking. The two methods, CAVI and 10-year cardiovascular risk assessment, offer dual monitoring in preliminary screening, therapy introduction or therapy modification.

Acknowledgements

The authors had no conflict of interest to declare. We had no funding sources for this study.

REFERENCES

1. HAFFNER SM, LEHTO S, RÖNNEMAA T, PYÖRÄLÄ K, LAAKSO M, N Engl J Med, 339 (1998) 229. — 2. LAKKA HM, LAAKSONEN DE, LAKKA TA, NISKANEN LK, KUMPUSALO E, TUOMILEHTO J, SALONEN JT, JAMA, 288 (2002) 2709. — 3. YUSUF S, REDDY S, OUNPUU S, ANAND S, Circulation, 104 (2001) 2746. — 4. DZAKULA A, SOGORIĆ S, POLASEK O, JURISA A, ANDRIĆ A, RADAKOVIĆ N, TODOROVIĆ G, Coll Antropol, 33 (2009) 87. — 5. KRALJ V, HRABAK-ZERJAVIC V, ERCEG M, TOMIĆ B, STEVANOVIĆ R, Croatian National Institute of Public Health, (2005) 59. — 6. MURRAY CJL, LOPEZ AD (Eds) The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries and Risk Factors in 1990 and Projected to 2020 (Boston, Mass: Harvard School of Public Health, 1996). — 7. LAW M, Lipids and cardiovascular disease. In: YUSUF S, CAIRNS JA, CAMM JA, FALLEN EL, GERSH BJ (Eds) Evidence Based Cardiology (London, UK: Br Med J Books, 1998). — 8. MACMAHON S, PETO R, CUTLER J, COLLINS R, SORLIE P, NEATON J, ABBOTT R, GODWIN J, DYER A, STAMLER J, Lancet, 335 (1990) 765. — 9. GRUNDY SM, PASTERNAK R, GREENLAND P, SMITH S JR, FUSTER V, Circulation, 100 (1999) 1481. — 10. STAMATELOPOULOS KS, PAPAMICHAEL CM, ZACHAROULIS A, PAPAIOANNOU T, KOLLIAS GE, KYRKOU K, CHRYSOCHOOU EE, VOIDONIKOLA P, ALEVIZAKI M, LEKAKIS JP, Eur J Cardiovasc Prev Rehabil, 15 (2008) 619. — 11. SRÁMEK A, BOSCH JG, REIBER JH, VAN OOSTAYEN JA, ROSENDAAL FR, Invest Radiol, 35 (2000) 699. — 12. BLACHER J, GUERIN AP, PANNIER B,

MARCHAIS SJ, LONDON GM, Hypertension, 38 (2001) 938. — 13. YAMASHINA A, TOMIYAMA H, ARAI T, HIROSE K, KOJI Y, HIRAYAMA Y, YAMAMOTO Y, HORI S, Hypertens Res, 26 (2003) 615. — 14. NAKANISHI N, SHIRAISHI T, WADA M, Hypertens Res, 28 (2005) 125. — 15. TAKAKI A, OGAWA H, WAKEYAMA T, IWAMI T, KIMURA M, HADANO Y, MATSUDA S,MIYZAKI Y, HIRATSUKA A, MATSUZAKI M, Hypertens Res, 31 (2008) 1347. — 16. KADOTA K, TAKAMURA N, AOYAGI K, YAMASAKI H, USA T, NAKAZATO M, MAEDA T, WADA M, NAKASHIMA K, ABE K, TAKESHIMA F, OZONO Y, Circ J, 72 (2008) 304. — 17. SATOH N, SHIMATSU A, KATO Y, ARAKI R, KOYAMA K, OKAJIMA T, TANABE M, OOISHI M, KOTANI K, OGAWA Y, Hypertens Res, 31 (2008) 1921. — 18. SAKANE K, MIYOSHI T, DOI M, HIROHATA S, KAJI Y, KAMIKAWA S, OGAWA H, HATANAKA K, KITAWAKI T, KUSACHI S, YAMAMOTO K, J Atheroscler Thromb, 15 (2008) 261. — 19. D'AGOSTINO RB SR, VASAN RS, PENCINA MJ, WOLF PA, COBAIN M, MASSARO JM, KANNEL WB, Circulation, 117 (2008) 743. — 20. YAMBE T, YOSHIZAWA M, SAIJO Y, YAMAGUCHI T, SHIBATA M, KONNO S, NITTA S, KUWAYAMA T, Biomed Pharmacother, 58 (2004) 95. — 21. World Health Organization. Atlas of Heart Disease and Stroke. Geneva, Avaliable from: URL: http://www.who.int/cardiovascular_diseases/resources/atlas/en/index. htlm; Accessing website: 25 Jul 2010. — 22. LINDHOLM LH, MENDIS S, Lancet, 370 (2007) 720. — 23. KOTSEVA K, WOOD D, DE BACKER G, DE BACQUER D, PYÖRÄLÄ K, KEIL U, Lancet, 373 (2009) 929. — 24. MACKENBACH JP, CAVELAARS AE, KUNST AE, GROENHOF F, Eur Heart J, 21 (2000) 1141. — 25. BERLINER JA, NAVAB M, FOGELMAN AM, FRANK JS, DEMER LL, EDWARDS PA, WATSON AD, LUSIS AJ, Circulation, 91 (1995) 2488. — 26. NAVAB M, FOGELMAN AM, BERLINER JA, TERRITO MC, DEMER LL, FRANK JS, WATSON AD, EDWARDS PA, LUSIS AJ, Am J Cardiol, 76 (1995) 18C. — 27. MADAMANCHI NR, VENDROV A, RUNGE MS, Arterioscler Thromb Vasc Biol, 25 (2005) 29. — 28. SINGH U, JIALAL I, Pathophysiology, 13 (2006) 129. — 29. WHEELER EC, BRENNER ZR, AACN Clin Issues, 6 (1995) 505. — 30. TAKAKI A, OGAWA H, WAKEYAMA T, IWAMI T, KIMURA M, HADANO Y, MATSUDA S, MIYAZAKI Y, MATSUDA T, HIRATSUKA A, MATSUZAKI M, Circ J, 71 (2007) 1710. — 31. WAKABAYASHI I, MASUDA H, Clin Chim Acta, 365 (2006) 230. — 32. TAKENAKA T, HOSHI H, KATO N, KOBAYASHI K, TAKANE H, SHODA J, SUZUKI H, J Atheroscler Thromb, 15 (2008) 339. — 33. ARONSON D, J Hypertens, 21 (2003) 3. — 34. MASUGATA H, SENDA S, GODA F, YAMAGAMI A, OKUYAMA H, KOHNO T, HOSOMI N, YUKIIRI K, NOMA T, KIYOMOTO H, MURAO K, NISHIYAMA A, KOHNO M, Tohoku J Exp Med, 217 (2009) 139. — 35. MASUGATA H, SENDA S, HIMOTO T, OKUYAMA H, INUKAI M, MURAO K, HOSOMI N, YUKIIRI K, KOHNO M, YAMAGAMI A, KOHNO T, GODA F, Tohoku J Exp Med, 218 (2009) 115. — 36. WENGER NK, Nat Clin Pract Cardiovasc Med, 4 (2007) 366. — 37. NICHOLLS M, Circulation, 114 (2006) 138. — 38. HIPPISLEY-COX J, COUPLAND C, VINOGRADOVA Y, ROBSON J, MAY M, BRINDLE P, BMJ, 335 (2007) 136. — 39. WOODWARD M, BRINDLE P, TUNSTALL-PEDOE H, Heart, 93 (2007) 172. — 40. MARRUGAT J, D'AGOSTINO R, SULLIVAN L, ELOSUA R, WILSON P, ORDOVAS J, SOLANAS P, CORDÓN F, RAMOS R, SALA J, MASIÁ R, KANNEL WB, J Epidemiol Community Health, 57 (2003) 634. — 41. KRAGELUND C, GRØNNING B, KØBER L, HILDEBRANDT P, STEFFENSEN R, N Engl J Med, 352 (2005) 666. — 42. SESSO HD, WANG L, BURING JE, RIDKER PM, GAZIANO JM, Hypertension, 49 (2007) 304.

Lj. Mayer

»Sv. Rok« Polyclinic, Department of Medical Biochemistry, Av. Vukovar 284, 10000 Zagreb, Croatia
e-mail: ljiljana.mayer@gmail.com

MEĐUOVISNOST CAVI, INDEKSA PROCJENE 10-GODIŠNJEG RIZIKA I DRUGIH RIZIČNIH ČIMBENIKA ATEROSKLEROZE

S A Ž E T A K

U ovom radu je ispitano postojanje povezanost intenziteta ateroskleroze određenog pomoću dvije različite metode aterosklerotskog screeninga: A) instrumentalnim mjerenjem cardio-ancle-vascular indeksa (CAVI) pomoću uređaja za vaskularni screening Vasera VS-1500 te B) bodovanjem temeljem Framighamske skale. Sveukupno je uključeno 52 ispitanika (24 žena i 28 muškaraca). Svrstavanjem ispitanika u četiri istobrojčana kvartila temeljem teoretski izračunatoga 10-godišnjeg rizika prema Framigham-skoj skali (medijani: 1, 3, 4 i 15%), potvrđeno je da je njegovo povećanje praćeno s statistički značajnim povećanjem CAVI, dobi, koncentracije ukupnog kolesterola, uz istodobno statistički značajno smanjenje HDL-kolesterola ($p<0{,}001$). Spearmanov-i koeficijenti korelacije su pokazali da 10-godišnjim rizik statistički značajno pozitivno korelira s CAVI ($p=0{,}0242$; $r=0{,}4494$). Temeljem dobivenih rezultata može se zaključiti da istovremeno određivanje CAVI indeksa te 10-godišnjeg rizika može biti opravdano. Ono nije proturječno, čak štoviše, oni značajno i pozitivno koreliraju. Na taj način se dobije lako razumljiva (sa svim svojim eventualnim posljedicama) i za pojedinca visoko-motivirajuća informacija koja će potaknuti na promjene štetnih ustaljenih životnih navika.