Blood Pressure

ISSN: 0803-7051 (Print) 1651-1999 (Online) Journal homepage: informahealthcare.com/journals/iblo20

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To cite this article: Jan Filipovský, Milena Tichá, Renata Cífková, Věra Lánská, Vlasta Šťastná & Patrik Roučka (2005) Large artery stiffness and pulse wave reflection: Results of a population-based study, Blood Pressure, 14:1, 45-52, DOI: 10.1080/08037050510008814

To link to this article: https://doi.org/10.1080/08037050510008814

Published online: 08 Jul 2009.

Blood Pressure. 2005; 14: 45–52

Taylor & Francis
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ORIGINAL ARTICLE

Large artery stiffness and pulse wave reflection: Results of a population-based study

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Abstract

Objective. To assess the determinants of large artery stiffness and pulse wave reflection in a population sample. *Methods.* A 1% random population sample aged 25–65 years was selected in nine districts of the Czech Republic for a survey of cardiovascular risk factors (Czech post-MONICA). Of 891 individuals screened in the Pilsen centre in the year 2000, arterial properties were studied in 291 (143 males and 148 females) using the Sphygmocor device. Pulse wave velocity (PWV) in the aorta and in the lower limbs was measured to assess large artery stiffness. Wave reflection was assessed from radial pulse wave analysis; the main estimated parameter was peripheral augmentation index (PAI) defined as P2/P1=ratio of pulse pressures measured at the peaks of secondary and primary waves. *Results.* Aortic PWV increased with age ($p<0.001$) and was similar in both sexes. Lower extremity PWV increased with age in women, but not in men, and its mean value was higher in men ($p<0.01$). PAI was higher in females in all age groups ($p<0.001$) and increased steeply with age in both sexes ($p<0.001$). PAI was increased in current smokers ($p<0.01$ in both sexes) and in male smokers, the reflected wave returned earlier than in male non-smokers ($p<0.05$). Correlation coefficient of PAI with aortic PWV was 0.22 ($p<0.01$), and with central augmentation index (CAI), derived from PAI by mathematical transformation, was 0.94 ($p<0.001$). Multiple regression analyses, where age, sex, systolic blood pressure (SBP), total cholesterol level, smoking, glucose level and body mass index were included as independent variables, were performed. PAI was better predicted than aortic or lower extremity PWV in these models (41%, 14% and 10% of variance explained, respectively). Age, female sex, smoking, SBP and total cholesterol predicted PAI level whereas age, SBP and glucose level were the main determinants of aortic PWV. *Conclusions.* Of the studied arterial parameters, PAI showed the closest association with cardiovascular risk factors. The correlation between PAI and aortic PWV was loose, and both parameters had partially different determinants. PAI, which is obtained by direct measurement above radial artery, was practically identical with the mathematically derived CAI in the studied population sample, and therefore, it is a suitable parameter for studying the phenomenon of wave reflection.

Key Words: *Aortic stiffness, blood pressure, cardiovascular risk assessment, smoking, wave reflection*

Introduction

The structure and function of central large arteries change with increasing age. The aorta and its main branches, where elastin compounds in the vessel wall predominate over collagen, become stiff as the amount of collagen fibres increases. The underlying changes responsible for the process of stiffening are diffuse and occur mainly in the media of the arterial wall. They lead to enlargement of the central arteries and result in impairment of their buffering function (1). Thus, arterial stiffening, caused by the aging process and accelerated by high blood pressure (BP), is different from atherosclerosis in many aspects. Atherosclerosis develops above all at predisposed

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(Received 20 July 2004; accepted 27 September 2004)

ISSN 0803-7051 print/ISSN 1651-1999 online
DOI: 10.1080/08037050510008814

sites, it is localized in the intima and leads usually to the narrowing of the vessel lumen; therefore, the conduit function is predominantly impaired. Besides the stiffening of elastic-type large arteries, the pulse waveform changes with age. The primary pressure wave, generated by contraction of the left ventricle, propagates towards the periphery where, probably at the level of smallest arteries, it is reflected and runs back. The reflected wave interferes with the primary one, and the definite pulse waveform is therefore composed of primary and secondary wave (the higher-order waves seem to play a minor role). In young individuals, the reflected wave falls typically into early diastole and diminishes the diastolic BP (DBP) drop. As age increases, the wave is higher and tends to return earlier for complex reasons. It produces a late systolic peak, which may be as high as 30–40 mmHg. Arterial stiffness can be assessed non-invasively by measuring the velocity of pulse wave propagation between two sites (pulse wave velocity, PWV), and wave reflection is estimated from pulse waveform analysis using most often augmentation index (AI).

Both large artery stiffness and wave reflection contribute to the increase of systolic BP (SBP) and widening of pulse pressure with age (2). This type of BP is associated with a high risk of cardiovascular events, and SBP has been shown to be associated with risk of cardiovascular events more closely than DBP (3). Therefore, it is important to study the pathogenesis and the risk factors for the SBP increase. Whereas atherosclerosis and its risk factors have been explored in detail, little is known about factors determining the mechanical properties of the arteries. As their changes usually precede the development of atherosclerosis, a detailed knowledge of mechanisms could allow early prevention of vascular changes. This paper presents an analysis of several vascular indices showing arterial stiffness and wave reflection, measured in healthy subjects selected randomly from a general population. Whereas there are some data about aortic stiffness in epidemiological studies (4,5), data about stiffness of peripheral arteries and about wave reflection in normal subjects are rare (6,7). PWV and AI are considered by some authors to give similar information about large artery properties. This presumption need not be correct, as wave reflection is a complex phenomenon, depending not only on large artery stiffness. Therefore, we focused in the present analysis on the relationships between the parameters of stiffness and wave reflection. We further studied the relationships of these parameters with age, sex and classical cardiovascular risk factors.

Population and methods

The present study was performed as part of the Czech post-MONICA study as an extension of the WHO MONICA (Multinational MONItoring of trends and determinants in CArdiovascular disease) study. In 2000/2001, a survey for cardiovascular risk factors was conducted in nine districts of the Czech Republic, involving a 1% population random sample aged 25–65 years in each district. Selection (stratified by age, sex and community size) was made from the General Health Insurance Registry keeping, by law, a list of all those insured. The examination included patient's medical history, physical examination, three measurements of BP to the nearest 2 mmHg and taking a fasting blood sample.

In the Pilsen centre, the overall response rate was 67.5%. Besides the screening for cardiovascular risk factors, vascular properties were examined in about one half of the randomly chosen subjects (Table I) using the Sphygmocor device (AtCor Medical Ltd., Australia). The first part of this examination was the registration of radial pulse wave. Radial pulse wave analysis (PWA) was used primarily to evaluate wave reflection (Fig. 1). The parameter showing wave reflection is AI, defined as the ratio of pulse pressure at the peaks of secondary to primary wave (AI=P2/P1). The radial waveform can be mathematically transformed to the form in central circulation (8) and thus, CAI and several other central haemodynamic parameters are estimated. The mathematical transformation using the general transfer function was validated towards invasive measurements by some authors (9); however, others cast doubts on its precision (10). We focused therefore on peripheral parameters, which are directly measured. Immediately before the radial pulse wave registration, an additional BP reading was obtained using the Omron 705CP oscillometric device, validated by an independent centre (11) and recommended by the European Society of Hypertension (12). The probability of observer bias is minimized with this type of device. The second part of the examination was PWV measurement assessing arterial stiffness (7,13). We used the same device and measured it with the patient in supine position in the aorta, i.e. between carotid and femoral arteries, and in the lower extremity, i.e. between femoral and dorsalis pedis/tibialis posterior arteries. Consecutive registrations of the pulse waves are ECG gated and thus, the time shift between the appearance of wave at the first and the second sites can be calculated. The distance between the two sites was measured on the body surface; to determine aortic PWV, we measured the distance from the jugular fossa to the pulsation of the

Table I. Population characteristics.

	Males	Females	*p*
Subjects (n)			
Age 25–34	29	26	
Age 35–44	29	30	
Age 45–54	49	47	
Age 55–65	36	45	
Total number	143	148	0.768
Indices of vascular properties[a]			
Aortic PWV (m/s)	7.8±2.8	7.4±2.3	0.171
Lower extremity PWV (m/s)	13.0±5.2	11.2±3.6	<0.001
PAI (%)	66.2±20.2	77.4±20.1	<0.001
TP1 (ms)	101.3±14.8	102.5±17.9	0.546
TP2(ms)	227.0±23.0	222.2±30.2	0.136
CAP (mmHg)	6.8±6.8	9.6±6.9	<0.001
CAI (%)	123.8±21.4	137.4±22.8	<0.001
Cardiovascular risk factors			
Systolic blood pressure (mmHg)	128.3±15.5	123.0±17.2	0.01
Diastolic blood pressure (mmHg)	82.8±9.8	78.0±9.0	<0.001
Hypertension (%)[b]	39.9	28.6	<0.05
Total cholesterol (mmol/l)	5.9±1.2	5.8±1.1	0.339
HDL-cholesterol (mmol/l)	1.3±0.4	1.6±0.3	<0.001
LDL-cholesterol (mmol/l)	3.7±0.9	3.5±1.0	0.19
Triglycerides (mmol/l)	2.0±1.2	1.5±0.7	<0.001
Hypercholesterolaemia (%)[c]	80.3	71.2	0.105
Glucose (mmol/l)	5.7±1.3	5.5±1.5	0.231
Diabetes (%)[d]	5.3	5.2	0.975
Current smoking (%)	40.6	28.6	0.032
Height (cm)	177.3±7.0	163.6±6.8	<0.001
Weight (kg)	85.8±13.3	70.0±11.5	<0.001
BMI (kg/m^2)	27.3±4.0	26.2±4.4	0.027

Peripheral parameters are those measured on radial artery, central ones are estimated by mathematical transformation in the aorta. [a]Mean ± SD is given. [b]Mean of three measurements ⩾140/90 mmHg and/or on antihypertensive treatment. [c]total cholesterol level ⩾5 mmol/l and/or on antihyperlipidemic treatment. [d]glucose level ⩾7 mmol/l and/or on antidiabetic treatment. PWV, pulse wave velocity; PAI, peripheral augmentation index; CAP, CAI, central augmentation pressure and augmentation index, respectively; TP1, TP2, time to peaks of primary and secondary wave, respectively, measured on radial artery; HDL, high-density lipoprotein; LDL, low-density lipoprotein; BMI, body mass index.

femoral artery in the groin, subtracting from this distance the distance from the jugular fossa to carotid pulsation, since the pulse runs here in the direction opposite to that in the aorta. The average of measurements over a period of 8 s (9–10 cardiac cycles) was calculated after the exclusion of extreme values. Both PWA and PWV were shown to have good reproducibility (14,15). Wilkinson et al. found that within-observer variability was 0.49±5.37% for AI and 0.07±1.17 m/s for aortic PWV (14). In our previous study performed in healthy subjects, AI was found less variable than BP values when several measurements of these parameters were compared (15).

Statistical analysis was performed using Pearson's correlation coefficients for normally distributed variables (normality was tested by χ^2 goodness-of-fit test), partial correlation coefficients for elimination of the age effects and Spearman's correlation coefficients for highly skewed variables. Analysis of variance was applied to calculate age trends and sex differences. The indices of vascular properties were further correlated with the 10-year risk of coronary heart disease calculated according to the equation derived from the Framingham Study; the methodology published by Anderson et al. was used (16). All calculations were done by statistical SYSTAT 10 software.

Results

Basic characteristics of the sample are given in Table I. Mean aortic PWV was much lower than lower extremity PWV (aortic PWV: 7.8 and 7.4 m/s in males and females, respectively, lower extremity PWV: 13.0 and 11.2 m/s in males and females, respectively). Mean peripheral augmentation index (PAI), showing wave reflection on the radial artery, was 66% in males and 77% in females, i.e. the

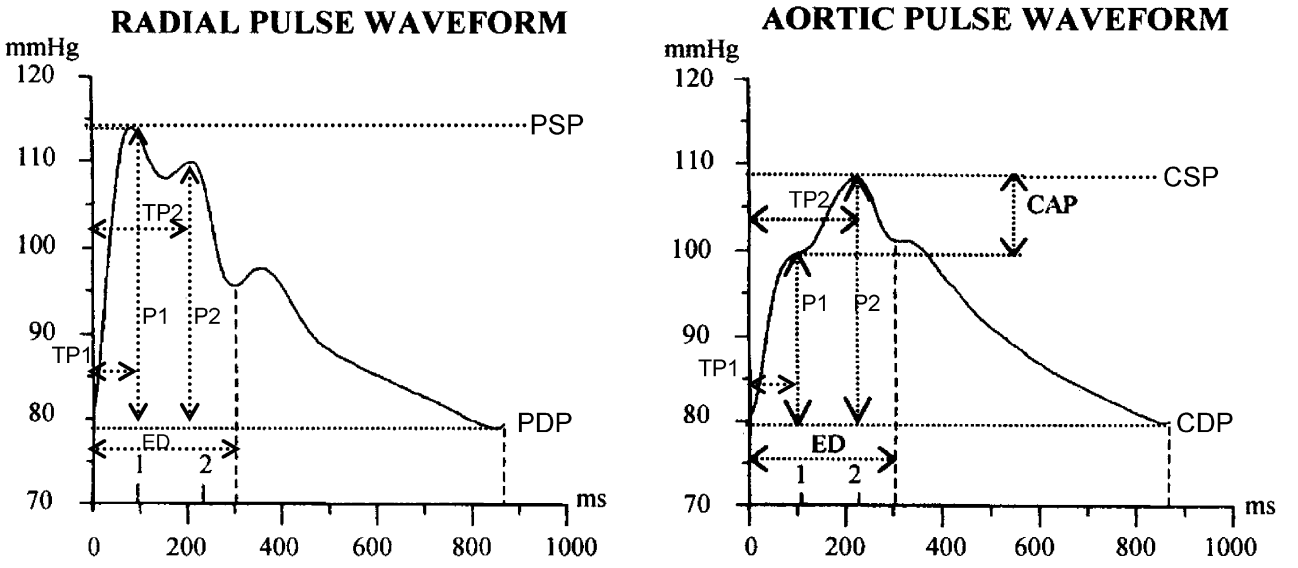

Figure 1. Pulse waveform registered on radial artery (left) and derived by means of mathematical transformation in the aorta (right). Radial pulse wave is calibrated on blood pressure measured conventionally on brachial artery (peripheral systolic and diastolic blood pressure, PSP, PDP). Aortic pulse wave is calibrated on mean pressure, obtained by integration of radial pulse wave, assuming that mean pressure is identical at the two sites. P1, P2, first and second pulse pressure peak, respectively; TP1, TP2, time to the peak of primary and reflected wave, respectively; ED, ejection duration; CSP, CDP, central systolic and diastolic pressure; CAP, central augmentation pressure – increase of pressure over P1 due to wave reflection. Augmentation index is defined as P2/P1 (peripheral, PAI, or central, CAI, measured on the radial or aortic pulse wave, respectively).

secondary wave did not exceed the primary wave in a typical case, whereas, in the aorta, the mean values were 123.8% in males and 137.4% in females (=central augmentation index, CAI). Compared to females, males had significantly higher lower extremity PWV, lower PAI, CAI and central augmentation pressure, higher SBP and DBP, body mass index, triglycerides level and lower high-density lipoprotein cholesterol level. Males were also more often current smokers (40.6% vs 28.6% in females, $p<0.032$).

In Fig. 2, the changes in vascular indices with age are shown separately for males and females. Aortic PWV (panel A) increased significantly with age ($p<0.001$). Lower extremity PWV was found to increase with age in females ($p<0.01$), but not in males (panel B); except for the highest age group (55–65 years), it was lower in females than in males. PAI (panel C) increased with age ($p<0.001$) and was consistently higher in women in all age groups ($p<0.001$). The timing of waves on the radial artery changed significantly with age (panel D): the primary peak was reached later with increasing age ($p<0.001$), whereas the reflected wave arrived earlier ($p<0.05$).

Table II shows associations of vascular indices with age and cardiovascular risk factors. Age correlated most closely with PAI, and further with aortic PWV and time to the primary pressure wave peak. BP values showed the closest age-adjusted correlations with aortic PWV in both sexes. Body height correlated inversely with PAI. Current smoking was associated with higher PAI in both sexes ($p<0.01$ for both sexes) whereas PWV, measured either in the aorta or in lower extremity, was not related to smoking status, and the reflected wave occurred earlier in male, but not female, smokers ($p<0.05$).

Table III shows mutual correlations of vascular indices. PAI and the derived CAI were closely correlated ($r=0.94$, $p<0.001$): this indicates that these two parameters were nearly identical in our setting. On the contrary, the correlations of PWV with parameters of wave reflection were fairly loose.

Multiple linear regressions were further calculated (Table IV). In order to increase statistical power, both sexes were analysed together. Of the vascular parameters tested as dependent variables, PAI was best predicted by the included independent variables (41% of variance explained). PAI was significantly associated with age, sex (higher in females), smoking status, SBP and total cholesterol level and aortic PWV was significantly associated with age, SBP and glucose level.

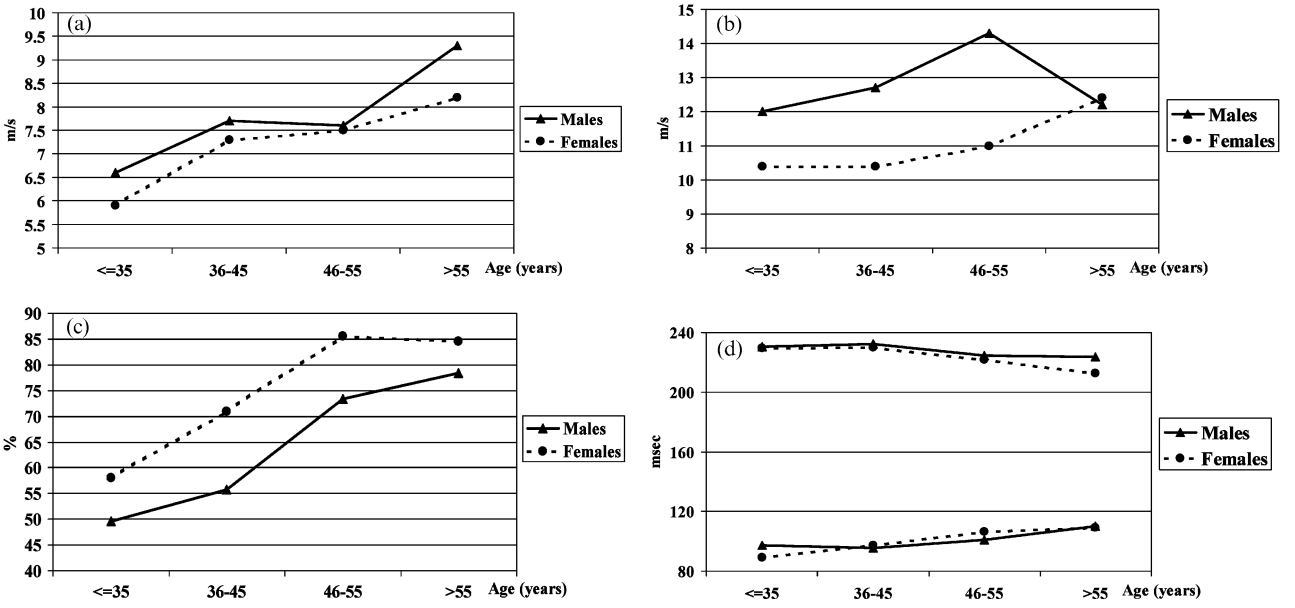

Figure 2. Analysis of vascular indices according to age and sex. Panel A: aortic pulse wave velocity. Age trend: $p<0.001$, sex difference: NS. Panel B: Pulse wave velocity on arteries of lower extremity. Age trend: NS (correlation coefficient of PWV with age NS for males, 0.24 for females, $p<0.01$). Sex difference: $p<0.01$. Panel C: Augmentation index assessed on radial pulse waveform. Age trend: $p<0.001$, sex difference: $p<0.001$. Panel D: Times to reach primary peak (lower curves) and secondary peak (upper curves) on radial pulse wave. Age trends: $p<0.001$ for first peak and $p<0.05$ for second peak, sex difference: NS for both peaks.

Table II. The age-adjusted associations of vascular indices to cardiovascular risk factors and anthropometric measurements.

	Aortic PWV(m/s)		Lower extremity PWV(m/s)		PAI(%)		TP1(ms)		TP2(ms)	
	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females
1. Correlation analysis of continuous variables										
Age[a]	0.30***	0.38***	0.06	0.24**	0.57***	0.52***	0.34***	0.40***	−0.12	−0.22**
Systolic blood pressure[b]	0.29***	0.27***	0.12	0.14	0.16	0.16	0.16	0.13	0.08	−0.17*
Diastolic blood pressure	0.28***	0.24**	0.25**	0.09	0.28**	0.11	0.05	0.01	−0.10	−0.17*
Total cholesterol	−0.12	0.11	0.10	−0.07	0.18	0.17	−0.02	−0.03	−0.21*	−0.03
HDL-cholesterol	−0.04	−0.06	0.16	−0.20	0.19	−0.06	−0.09	−0.10	−0.10	0.14
LDL-cholesterol	−0.14	0.12	0.07	−0.01	0.08	0.23*	−0.03	−0.01	−0.10	−0.07
Triglycerides	−0.02	0.08	0.02	0.02	0.21*	−0.05	0.05	0.04	−0.15	−0.04
Glucose	0.17	0.10	−0.15	−0.09	0.14	−0.11	−0.02	0.02	−0.32**	−0.01
Height	0.05	−0.12	−0.13	−0.07	−0.29***	−0.22*	0.08	−0.15	0.10	0.08
Weight	0.08	0.06	0.02	−0.12	−0.18*	−0.03	0.11	0.01	0.08	−0.11
Body mass index	0.07	0.13	0.09	−0.09	−0.04	0.07	0.08	0.08	0.04	−0.16
2. Analysis of smoking (yes/no)										
Smokers[c]	7.8	7.8	12.5	11.7	71.1**	84.0**	101.5	103.4	220.8*	219.3
Non-smokers	7.9	7.3	13.4	11.0	62.9	74.4	101.2	102.1	231.1	223.7

* $p<0.05$, ** $p<0.01$, *** $p<0.001$ [a]Pearson corelation coefficients are given. [b]Age-adjusted partial corelation coefficients are given. [c]Age-adjusted means are given. PWV, pulse wave velocity; PAI, peripheral augmentation index; TP1, TP2, time to peaks of primary and secondary wave, respectively, measured on radial artery; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

The vascular indices were further related to the 10-year risk of coronary heart disease as predicted by the Framingham equation (Fig. 3). Mean risk was 11.0% (range 0.35–36.1%) in males and 5.2% (range 0.02–26.5%) in females. Both aortic PWV (Panel A) and PAI (Panel C) were significantly associated with the risk; the closest relationship was that of PAI in males ($r=0.51$, $p<0.001$). The correlation of lower extremity PWV with the risk was not significant in males, and low in females.

Table III. The interrelationships of pulse wave velocities and parameters of pulse wave reflection.

	Aortic PWV	Lower extremity PWV	PAI
Aortic PWV			
Lower extremity PWV	0.18**		
PAI	0.22**	0.04	
CAI	0.13*	0.02	0.94***

* $p<0.05$, ** $p<0.01$, *** $p<0.001$. Spearman correlation coefficients are given. PWV, pulse wave velocity; PAI, CAI, peripheral and central augmentation index.

Table IV. Multiple regression analysis of vascular parameters.

	Aortic PWV	Lower extremity PWV	PAI	TP1	TP2
R^2 (%)	14	10	41	16	5
Age	0.20**	0.09	0.44**	0.31**	−0.06
Sex	−0.06	−0.19**	0.33**	0.09	−0.12
Systolic blood pressure	0.16*	0.15	0.14*	0.11	−0.05
Total cholesterol	−0.06	0.05	0.11*	−0.05	−0.09
Glucose	0.14*	−0.15*	−0.03	−0.01	−0.12
Smoking	0.06	0.06	0.18**	0.04	−0.06
Body mass index	−0.01	−0.01	−0.03	0.10	0.01

R^2, percentage of variance explained. Standardized regression coefficients are given; * $p<0.05$, ** $p<0.01$. Sex: 0 – male, 1 – female. Smoking: 0 – nonsmoker, 1 – exsmoker (no smoking for a period of more than one year), 2 – current smoker. Other variables are continuous. PWV: pulse wave velocity, PAI: peripheral augmentation index; TP1, TP2, time to peaks of primary and secondary wave, respectively, measured on radial artery.

Discussion

In our study, we analysed the relationships of parameters characterizing arterial properties with age, sex and classical cardiovascular risk factors in a sample of general population. PAI was best predicted in multiple regression analysis where cardiovascular risk factors were included as independent variables (41% of total variance explained; Table IV), mainly through its association with age, smoking and to lesser extent through BP and total cholesterol level. Other vascular parameters, including aortic PWV, were much less predicted by the independent variables. We further correlated the arterial parameters with the Framingham score separately in both sexes and the findings were compatible with the previous ones: the score was most closely associated with PAI in males (Fig. 3). Smoking was significantly associated with higher reflected wave in both sexes and with its earlier return in males (Table II), whereas no association with PWV, measured either above aorta or on lower extremity, was found. The fact that the timing of reflected wave between female smokers and non-smokers was not different (power of the test less than 20%), whereas it differed between male smokers and non-smokers (power of the test 66%), could be probably ascribed to the fact that females smokers were less numerous in our sample and generally have lower cigarette consumption than males.

Our results show, in accordance with the recently published findings of Millasseau et al. (17), a very close relationship between the parameters of wave reflection measured in peripheral circulation on the radial artery and derived in central circulation (correlation coefficient 0.94 between PAI and CAI). This indicates that peripheral, directly measured parameters can be used for studying the phenomenon of wave reflection. Wave reflection is influenced by several factors: (i) contractility of the heart determines the magnitude and duration of the primary wave; (ii) large artery stiffness determines the velocity of primary as well as secondary wave and, therefore, influences the timing of reflected wave; (iii) the status of resistance vessels determines the degree of wave reflection because wave reflection occurs there (with peripheral vasodilation, e.g. by nitrates, there is less reflection); and (iv) the length of the arterial bed from the heart to the reflection sites influences timing – this is why smaller individuals have higher reflected wave as it returns earlier (see also our results in Table II). Wave reflection is therefore a complex phenomenon determined by heart function and the status of the arterial tree as a whole rather than by local properties

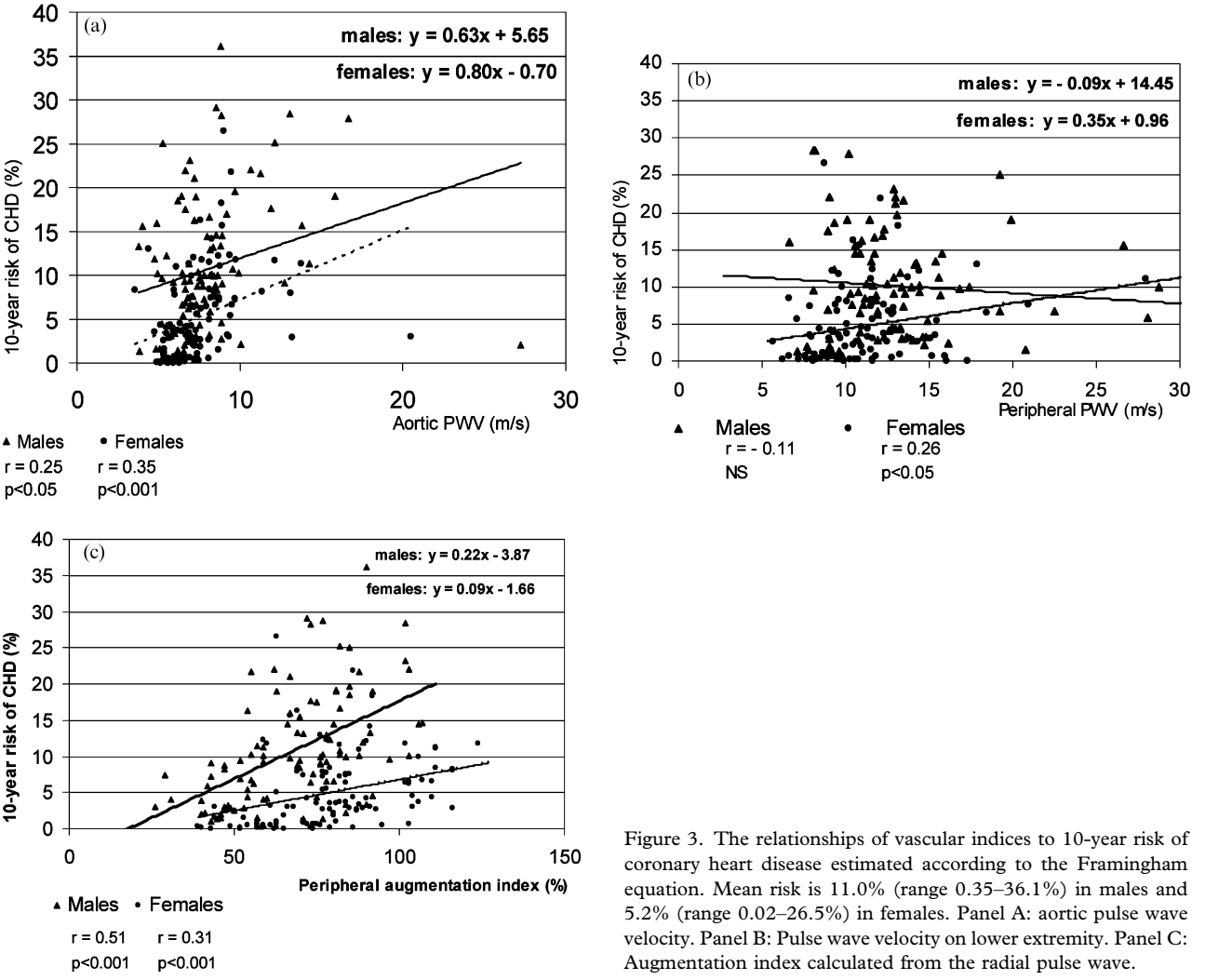

Figure 3. The relationships of vascular indices to 10-year risk of coronary heart disease estimated according to the Framingham equation. Mean risk is 11.0% (range 0.35–36.1%) in males and 5.2% (range 0.02–26.5%) in females. Panel A: aortic pulse wave velocity. Panel B: Pulse wave velocity on lower extremity. Panel C: Augmentation index calculated from the radial pulse wave.

of the vascular bed where reflection is measured. In addition, we found rather low associations between augmentation indices and pulse wave velocities (Table III). These findings confirm the fact that large artery stiffness is only one factor contributing to wave reflection.

PAI was significantly higher in females in all age groups (see panel C of Fig. 2). The same applied for CAI and absolute height of reflected wave – augmentation pressure (data not shown in detail, see also Table I). Similar findings were reported by other authors and they were ascribed to the fact that women have smaller body height. If this were indeed the reason then there would be a difference in timing between males and females, which was not the case in our material (see panel D of Fig. 2). This issue was studied in detail by Gatzka et al. (18) in 104 pairs of elderly males and females of identical height. Females also had higher AI in this setting, and they had a smaller aortic diameter and longer duration of systole. Both these phenomena are supposed to account for the gender differences in wave reflection.

Arterial stiffness as assessed by PWV is different in the aorta and lower limb arteries. Mean values were much higher on the lower limb in our material (Table I): this reflects higher stiffness due to the higher content of collagen fibres. Stiffness of these two arterial beds was not closely correlated (r=0.18; Table III). Thus, stiffness of elastic-type and muscular-type arteries is probably regulated by different mechanisms and may have different risk factors. The relationships with age and sex also differ (panels A and B of Fig. 2): whereas aortic PWV increased with age and there was no difference between sexes, lower extremity PWV was higher in men and did not increase with age, though it did increase in women and reached the same value as in men in the oldest age group (55–65 years). The reason for the

difference between males and females is not known and cannot be explained from our data; we can only hypothesize that the change in hormonal status after the menopause could increase the stiffness of distal large arteries in women.

In conclusion, the parameters under study are non-invasive and well reproducible. PAI gives practically the same information about wave reflection as CAI. The phenomenon of wave reflection should always be studied separately in males and females, because it is systematically higher in the latter, because of anatomic conditions and probably related to different mechanic properties of arterial system. The associations of pulse wave velocities and indices of wave reflection are loose; this indicates that the two sorts of parameters show different aspects of arterial properties. Aortic stiffness was shown to predict subsequent morbidity and mortality (19–21) and the same was found for wave reflection in patients with end-stage renal disease (22); these relationships existed in most studies independently of classical risk factors. Therefore, aortic PWV and AI could be used for better stratification of cardiovascular risk, and interventions that would be successful in influencing positively these parameters could represent a progress in cardiovascular disease prevention and treatment.

Acknowledgement

The study was supported by grant No. NG/6757-4, awarded by the Czech Ministry of Health.

References

1. Safar M. Pulsatile pressure and hypertension in large arteries. In: Safar M, editor. Arteries in clinical hypertension. Philadelphia: Lippincott-Raven, 1996, 21–30.
2. Wilson PWF, Kannel WB. Hypertension, other risk factors and the risk of cardiovascular disease. In: Laragh JH, Brenner BM, editors. Hypertension: pathophysiology, diagnosis, and management. New York: Raven Press, 1995: 99–114.
3. Stamler J, Stamler R, Neaton JD. Blood pressure, systolic and diastolic, and cardiovascular risks. Arch Intern Med. 1993;153:598–615.
4. Avolio AP, Fa-Quan D, Wei-Qiang L, et al. Effects of aging on arterial distensibility in populations with high and low prevalence of hypertension: comparison between urban and rural communities in China. Circulation. 1985;71:202–10.
5. Ho KL, Avolio AP, O'Rourke MF. Aortic pulse wave velocity, a noninvasive index of left ventricular afterload and aortic stiffening in an Australian community. Fed Proc. 1983;42:1128.
6. Nurnberger J, Keflioglu-Schreiber A, Saez AMO, Wenzel RR, Philipp T, Schafers RF. Augmentation index is associated with cardiovascular risk. J Hypertens. 2002;20:2407–14.
7. Davies JI, Struthers AD. Pulse wave analysis and pulse wave velocity: a critical review of their strengths and weaknesses. J Hypertens. 2003;21:463–72.
8. Nichols WW, O'Rourke MF. Ascending aortic pressure waves. In: Nichols WW, O'Rourke MF, editors. McDonald's blood flow in arteries. Theoretical, experimental and clinical principles. London: Edward Arnold, 1998: 457–6.
9. Chen CH, Nevo E, Fetics B, et al. Estimation of central aortic pressure waveform by mathematical transformation of radial tonometry pressure. Validation of generalized transfer function. Circulation. 1997;95:1827–36.
10. Hope SA, Tay DB, Meredith IT, Cameron JD. Use of arterial transfer functions for the derivation of aortic waveform characteristics. J Hypertens. 2003;21:1299–305.
11. O'Brien E, Mee F, Atkins N, Thomas M. Evaluation of three devices for self-measurement of blood pressure according to the revised British Hypertension Society Protocol: the Omron HEM-705CP, Philips HP5332, and Nissei DS-175. Blood Press Monit. 1996;1:55–61.
12. Reims H, Kjeldsen SE, Mancia G, et al. Home blood pressure monitoring. Eur Soc Hypertens Scient Newsl. 2002;3:No. 12.
13. Blacher J, Asmar R, Djane S, London G, Safar M. Aortic pulse wave velocity as a marker of cardiovascular risk in hypertensive patients. Hypertension. 1999;33:11–117.
14. Wilkinson IB, Fuchs SA, Jansen IM, et al. Reproducibility of pulse wave velocity and augmentation index measured by pulse wave analysis. J Hypertens. 1998;16:2079–84.
15. Filipovský J, Svobodová V, Pecen L. Reproducibility of radial pulse wave analysis in healthy subjects. J Hypertens. 2000;18:1033–40.
16. Anderson KM, Odell PM, Wilson PWF, Kannel WB. Cardiovascular disease risk profiles. Am Heart J. 1990;121:293–8.
17. Millasseau SC, Patel SJ, Redwood SR, Ritter JM, Chowienczyk PJ. Pressure wave reflection assessed from the peripheral pulse: is a transfer function necessary? Hypertension. 2003;41:1016–20.
18. Gatzka CD, Kingwell BA, Cameron JD, et al. Gender differences in the timing of arterial wave reflection beyond differences in body height. J. Hypertension. 2001;19: 2197–203.
19. Blacher J, Guerin A, Marchais SJ, Safar M, London G. Impact of aortic stiffness on survival in end-stage renal disease. Circulation. 1999;99:2434–9.
20. Laurent S, Boutouyrie P, Asmar R, et al. Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. Hypertension. 2001;37:1236.
21. Meaume S, Benetos A, Henry OF, Rudnichi A, Safar ME. Aortic pulse wave velocity predicts cardiovascular mortality in subjects >70 years of age. Arterioscler Thromb. 2001;21:2046–50.
22. London GM, Blacher J, Pannier B, Guerin AP, Marchais SJ, Safar ME. Arterial wave reflections and survival in end-stage renal failure. Hypertension. 2001;38:434–8.