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CENTRAL OR PERIPHERAL SYSTOLIC OR PULSE PRESSURE: WHICH BEST RELATES TO TARGET-ORGANS AND FUTURE MORTALITY?

Kang-Ling Wang^a, Hao-Min Cheng^b, Shao-Yuan Chuang^c, Harold A. Spurgeon^d, Chih-Tai Ting^e, Edward G. Lakatta^d, Frank C.P. Yin^f, Pesus Chou^h, and Chen-Huan Chen^{b,g,h,i}

^aDepartment of Medicine, Taipei Veterans General Hospital, Taipei, Taiwan

^bDepartment of Research and Education, Taipei Veterans General Hospital, Taipei, Taiwan

^cInstitute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan

^dthe Laboratory of Cardiovascular Science in the National Institute on Aging in Baltimore, MD, USA

^eCardiovascular Center, Taichung Veterans General Hospital, Taichung, Taiwan

^fDepartment of Biomedical Engineering, Washington University, St Louis, MO, USA

^gCardiovascular Research Center, National Yang-Ming University, Taipei, Taiwan

^hDepartment of Public Health, National Yang-Ming University, Taipei, Taiwan

ⁱI-Lan University Hospital, National Yang-Ming University, Taipei, Taiwan

Abstract

OBJECTIVE—We examined the relationship between brachial and central carotid pressures and target organ indices at baseline, and their association with future mortality.

METHODS—We examined cross-sectionally and longitudinally the relations of baseline systolic and pulse pressures in central (calibrated tonometric carotid pulse) and peripheral (brachial, mercury sphygmomanometer) arteries to baseline left ventricular mass, carotid intima-media thickness, estimated glomerular filtration rate, and 10-year all-cause and cardiovascular mortality in 1272 participants (47% women, aged 30–79 years) from a community of homogeneous Chinese.

RESULTS—Left ventricular mass was more strongly related to central and peripheral systolic pressures than pulse pressures. Intima-media thickness and glomerular filtration rate were more strongly related to central pressures than peripheral pressures. A total of 130 participants died, 37 from cardiovascular causes. In univariate analysis, all four blood pressure variables significantly predicted all-cause and cardiovascular mortality. Each blood pressure variable was entered into the multivariate models, both individually and jointly with another blood pressure variable. With adjustment for age, sex, heart rate, body mass index, current smoking, glucose, total cholesterol/high-density-lipoprotein-cholesterol ratio, carotid-femoral pulse wave velocity, left ventricular mass, intima-media thickness, and glomerular filtration rate, only central systolic pressure

Correspondence and reprint request to: Chen-Huan Chen, MD, No. 201, Sec. 2, Shih-Pai Road, Division of Cardiology, Taipei Veterans General Hospital, Taipei, Taiwan, Telephone: (866)-2-28712121, ext. 2073, Fax: (886)-2-28717431, chench@vghtpe.gov.tw.

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consistently independently predicted cardiovascular mortality (Hazards ratio=1.30 per 10 mmHg). No significant sex interactions were observed in all analyses.

CONCLUSION—Systolic and pulse pressures relate differently to different target organs. Central systolic pressure is more valuable than other blood pressure variables in predicting cardiovascular mortality.

Keywords

Pulse pressure; Central blood pressure; Glomerular filtration rate; Mortality

INTRODUCTION

Sustained elevation of systolic (SBP), diastolic (DBP), and pulse (PP) pressure leads to target organ damage and cardiovascular events.[1–3] SBP and PP are superior predictors of coronary heart disease than DBP in patients ≥ 50 years of age,[1] and also confer greater risk for congestive heart failure than DBP.[2] SBP integrates both steady and pulsatile loads whereas PP is mainly a measure of pulsatile load.[4, 5] Although an elevated PP is increasingly being recognized as a risk factor for cardiovascular disease,[1, 5] its importance relative to SBP for various target organs damage may vary and needs to be clarified.[3, 6]

Blood pressure in central arteries, such as the ascending aorta and common carotid arteries, is directly related to the loads imposed on the heart.[7] The branching structure and mechanical properties of the arterial system cause blood pressure in peripheral arteries, such as the brachial and radial arteries, to be amplified and thus peripheral pressure does not necessarily accurately reflect the central pressure.[8] Since central blood pressure can be estimated noninvasively,[9, 10] it is reasonable to presume, and it has been shown in population-based study[7] and in hypertension trials,[11] that the noninvasively estimated central blood pressure may be superior to brachial blood pressure in predicting cardiovascular outcomes. However, a recent study reported that brachial PP but not noninvasively measured central pressures, predicted outcomes in older female hypertensive patients.[12] This unexpected finding underscores the need to better assess the prognostic role of central as opposed to peripheral blood pressure in more large-scale observational and interventional studies.[10] Therefore, the purpose of the present study was to investigate the relations of central and brachial arteries to left ventricular mass (LVM), carotid intima-media thickness (IMT), estimated glomerular filtration rate (eGFR), and 10-year all-cause and cardiovascular mortality in a community-based population.

METHODS

Study population

The study cohort of 1272 normotensive and untreated hypertensive ($SBP \geq 140$ or $DBP \geq 90$ mmHg)[13] Taiwanese participants (598 women, aged 30–79 years) was drawn from a previous community-based survey conducted in 1992 to 1993.[14] Reasons for 43 of the original participants not being included in the present analysis included insufficient data for the calculation of central blood pressure or eGFR.[15] Baseline comprehensive cardiovascular evaluation performed in non-fasting state included complete medical history and physical examination, arterial tonometry and ultrasonography, and echocardiography as previously described.[14] None of the participants had a previous history of diabetes mellitus, angina pectoris, or peripheral vascular disease, and none had clinical or echocardiographic evidence of other significant cardiac diseases. All participants gave informed consent and the study was approved by the institutional review board at Johns Hopkins University.

Definition of variables

Anthropometric variables—Body mass index (BMI) and body surface area (BSA) were calculated from measurements of weight and height.[14]

Blood pressure variables—Blood pressure was measured manually using a mercury sphygmomanometer and a standard-sized cuff (13 cm × 50 cm) by senior cardiologists. Two or more measurements separated by at least five minutes were taken from the right arm of subjects after they were seated for at least 5 minutes. Reported blood pressures represent the average of at least two consecutive measurements. Brachial PP was calculated as [SBP – DBP] and brachial mean blood pressure was calculated as [DBP + (PP/3)]. Carotid SBP and PP, which closely reflect central aortic SBP and PP,[16] were derived by calibrating the right common carotid artery pressure waveforms with brachial DBP and mean blood pressure. [9] Carotid artery pressure waveforms were registered noninvasively with a tonometer. [7, 14]

Biochemical variables—Overnight fasting serum and plasma samples were drawn for glucose, lipid, and other biochemical measurements. Serum cholesterol, blood urea nitrogen, albumin and creatinine (using the Jaffe's kinetic method) were measured with a Hitachi auto-analyzer. Serum high-density lipoprotein cholesterol (HDL) was measured using a precipitation method (Kodak Ektachem HDL Kit). Plasma glucose concentration was determined by a hexokinase/glucose-6-phosphate dehydrogenase method [Glucose (HK) Reagent Kit, Gilford system, Oberlin, OH].

Arterial function variables—Stiffness of the aortic trunk was indexed by the carotid-femoral pulse wave velocity, which was calculated from sequential nondirectional Doppler (Parks model 802) flow velocity at the right carotid artery and femoral artery and a simultaneous electrocardiogram.[14] The extent of arterial wave reflections was indexed by the carotid augmentation index analyzed from the right common carotid arterial pressure waveforms.[17]

Variables of target organs—LVM was obtained from the 2-D guided M-mode echocardiography performed by the same experienced sonographer using a Hewlett-Packard SONOS 500 echocardiographic unit with a 2.5 MHz transducer.[14] LVM index was calculated by correcting LVM with BSA.

IMT of the posterior wall of the right common carotid artery was measured on-line from frozen, digitized images obtained with a 7-MHz vascular probe incorporated in the echocardiographic unit.

eGFR was calculated using age, sex, and serum levels of blood urea nitrogen, creatinine, and albumin according to the modified glomerular filtration rate estimating equations for Chinese patients.[18]

Follow-up

The date and causes of death for those who had deceased within follow-up period after the baseline survey were collected in all of the 1272 participants by linking our database with the National Death Registry through a unique, life-long personal identification number given to every Taiwan citizen. Subjects not appearing on the National Death Registry on December 31, 2003 were considered surviving. Subjects were not re-evaluated for the central and peripheral blood pressure during the 10 years. The National Death Registry database registers valid information based on the certified death certificates. The death certificates were coded according to the International Classification of Disease, Ninth

Revision (ICD-9). The ICD-9 codes used for cardiovascular death were 390–459. The accuracy of cause-of-death coding in Taiwan's National Death Registry database has been validated.[19]

Statistical Analysis

Data are presented as percents or mean $\pm$ standard deviation. Student's *t* test and Chi-square test were used for between-group comparisons where appropriate. Correlations between the blood pressure variables and the three target organ indices were assessed with Pearson's correlation coefficients. Partial correlation coefficients with adjustment of age, sex, heart rate, body mass index, current smoking, glucose levels, ratio of total cholesterol to HDL, and carotid-femoral pulse wave velocity were also provided. Comparisons between two correlation coefficients from paired measurements were carried out using the formula created by Olkin.[20] The period of survival was defined as the interval between the baseline survey and the date of death or the end of follow-up. The Cox proportional hazards model was used to estimate the relative risks of all-cause and cardiovascular mortality. Each blood pressure variable was entered into the multivariate models, both individually and jointly with another blood pressure variable.[21] Potential sex-interactions were examined by entering the corresponding interaction term of sex*blood pressure variable into the models. Two-tailed $P < 0.05$ was considered statistically significant. Statistical analyses were performed using the statistical package SPSS 15.0 (SPSS Inc).

RESULTS

Baseline characteristics of the participants are presented in Table 1. Women had significantly higher BMI, levels of fasting plasma glucose, brachial PP, central SBP and PP, AI, and eGFR, and lower prevalence of smoking, cholesterol/HDL ratio, brachial DBP, LVM, LVMI, and IMT, than men.

Relations of central aortic and brachial blood pressures to target-organ indices

Table 2 presents the relations of central and brachial blood pressures to LVM, IMT and eGFR. All central and peripheral SBP and PP correlated significantly with LVM, IMT, and eGFR.

For LVM, the ranks of correlation coefficients in descending order were central SBP, brachial SBP, central PP, and brachial PP. The correlation coefficients were significantly greater for central SBP than for brachial SBP, brachial SBP than central PP, and central PP than brachial PP (all $P < 0.05$). All blood pressure variables remained significantly positively related to LVM after adjustment for age, sex, heart rate, body mass index, current smoking, glucose, total cholesterol/HDL ratio, and carotid-femoral pulse wave velocity.

For IMT, the ranks were central PP, central SBP, brachial SBP, and brachial PP. The correlation coefficients were significantly greater for central PP than for brachial PP, and central SBP than brachial PP (all $P < 0.05$). All blood pressure variables remained significantly positively related to IMT, after adjustment for age, sex, heart rate, body mass index, current smoking, glucose, total cholesterol/HDL ratio, and carotid-femoral pulse wave velocity.

For eGFR, the ranks were central PP, central SBP, brachial SBP, and brachial PP. The correlation coefficients were significantly greater for central PP than for brachial PP, central SBP than brachial PP, and brachial SBP than brachial PP (all $P < 0.05$). After adjustment for age, sex, heart rate, body mass index, current smoking, glucose, total cholesterol/HDL ratio, and carotid-femoral pulse wave velocity, only central SBP remained significantly negatively related to eGFR.

Relations of central aortic and brachial blood pressures to mortality

After an average of 10.8 ± 1.70 years of follow-up, 130 (10.2%) deaths occurred, including 37 cardiovascular deaths. The causes for cardiovascular deaths included myocardial infarction or coronary artery disease ($n = 9$), aortic dissection or rupture of aortic aneurysm ($n = 1$), heart failure ($n = 7$), hypertension ($n = 1$) and stroke (either ischemic or hemorrhagic) ($n = 19$).

Table 3 shows the sex-stratified hazards ratios for all-cause and cardiovascular mortality by univariate analysis. Age, carotid-femoral pulse wave velocity, LVM, LVMI, IMT, eGFR, and all blood pressure variables were significant determinants of all-cause and cardiovascular mortality in both men and women. Additionally, smoking significantly predicted all-cause mortality in women, low body mass index significantly predicted all-cause mortality in men, high fasting plasma glucose levels significantly predicted all-cause and cardiovascular mortality in women, high total cholesterol/HDL ratio significantly predicted all-cause mortality in women and cardiovascular mortality in men.

Multivariate Cox proportional hazards regression models with adjustment for age, sex, heart rate, body mass index, current smoking, fasting plasma glucose levels, cholesterol/HDL ratio, carotid-femoral pulse wave velocity, LVM, IMT, and eGFR were performed with each blood pressure variable entering into the multivariate models individually (with checking of sex-interactions) and then jointly with another blood pressure variable.[21] No significant sex-interactions were found. None of the blood pressure variables independently contributed to all-cause mortality (Figure 1A). Only central SBP and PP independently contributed to cardiovascular mortality with hazards ratios of 1.303 (95% confidence interval 1.121–1.515) per increment of 10 mmHg SBP-C, and 1.257 (95% confidence interval 1.016–1.556) per increment of 10 mmHg PP-C (Figure 1B). With two blood pressure variables jointly entering the multivariate models, only central SBP was consistently the significant independent predictor of cardiovascular mortality (Table 4).

DISCUSSION

In this homogeneous, Chinese population without previous history of diabetes, angina pectoris, peripheral vascular disease, or other significant cardiac diseases, we found that central blood pressures had stronger associations with target organ indices than brachial blood pressures. Among the four blood pressure variables, central SBP was the best correlate for LVM, while both central SBP and PP were the best correlates for IMT and eGFR. In addition, central blood pressures but not brachial blood pressures predicted cardiovascular mortality independently of the conventional risk factors, arterial stiffness, LVM, IMT, and eGFR. Central SBP but not PP was the independent predictor of cardiovascular mortality when both central SBP and PP were entered into the multivariate model.

The superiority of central blood pressures over brachial blood pressures in the prediction of cardiovascular mortality was not sex-dependent. This may contradict the negative results from a subset of female participants in the Second Australian National Blood Pressure Study.[12] That study enrolled a small fraction (15.6%) of all female participants, who had high central SBP (162 ± 30 mmHg) and PP (84 ± 26 mmHg) yet small number of events (less than half of the main study cardiovascular events rate).[12] More importantly, the impact of antihypertensive medications (angiotensin converting enzyme inhibitors versus diuretics with or without additions of beta-blockers, calcium-channel blockers, and alpha-blockers) on the outcomes was not accounted for. Therefore, the study might be limited by a type 2 error and the interpretation and generalization of the results should be cautious.

SBP is determined mainly by arterial compliance and total peripheral resistance, [4, 22] while PP is determined by arterial compliance and stroke volume.[5] Both SBP and PP can be augmented by arterial wave reflections, especially with advancing age and increasing arterial stiffening.[4, 5] Epidemiological studies have documented a linear relation with age for brachial and central SBP and central PP, and a non-linear relation for brachial PP.[5, 23, 24] Although some studies have shown slightly greater importance of brachial PP over brachial SBP in predicting cardiovascular outcome,[7, 12, 25] the association of brachial PP with mortality may be complex and subject to ethnic differences.[3, 26] High brachial PP due to high brachial SBP is associated with increased risk for target organ damage, while high brachial PP due to low brachial diastolic blood pressure can be associated with increased risk, decreased risk, or no change in risk depending on age and blood pressure level.[26] In the present study, the correlation coefficients for LVM, IMT, and eGFR were greater for brachial SBP than for brachial PP and the difference was significant for LVM and eGFR. Therefore, our results support the perspective that the cardiovascular risk for brachial SBP and central SBP and PP is consistently strong but is complex for brachial PP. [26]

Brachial SBP has consistently been shown as a major hemodynamic predictor of LVM,[14, 27, 28] whereas brachial PP is a relatively weak determinant.[28] On the other hand, brachial PP but not brachial SBP was related to LVM in 87 older adults with borderline isolated systolic hypertension by a multivariate regression model that included all blood pressure variables (SBP, DBP, mean blood pressure, and PP).[29] However, this result may have been subject to problems of co-linearity. We demonstrated in a previous analysis of a very large number of subjects that the effects of the derived measures of arterial stiffness on LVM were smaller than brachial SBP.[14] The present study further demonstrates a stronger relation of central SBP than central PP with LVM. The result supports that central SBP integrates both pulsatile and nonpulsatile loads, while central PP and other indices of arterial stiffness mainly represent pulsatile load to the left ventricle. The superiority of central SBP over brachial SBP as a major determinant of LVM regression has recently been shown in 52 hypertensive patients treated with low-dose perindopril/indapamide and atenolol.[30]

Elevated PP may damage the microcirculation and increase resistance to mean flow, as illustrated in the Framingham Heart Study that abnormal aortic stiffness and increased pressure pulsatility were associated with blunted microvascular reactivity to ischemic stress. [31, 32] Indeed, increased aortic pulse wave velocity is associated with reduced eGFR in patients with type 2 diabetes without overt renal impairment,[33] and in patients with chronic kidney disease.[34] Central (aortic) pulse wave velocity appears to be more closely associated with eGFR than peripheral (upper-limb) pulse wave velocity in hypertensive patients with normal renal function.[35] Therefore, central PP and SBP may be more representative of the local pressure pulsatility for kidneys than brachial pressures, due to their closer relation with central arterial stiffness. The present study demonstrates a stronger relation of central blood pressures than peripheral blood pressures with eGFR. The result supports the importance of local pressure pulsatility in causing microvascular damage.[32]

The prominent influence of local pulsatile mechanical load on IMT has long been recognized.[36] In the Strong Heart Study, central PP was more strongly related to IMT than brachial PP.[7] That study also showed a slight but significant difference of correlation coefficients for IMT (0.293 versus 0.257, $P<0.001$) in favor of central PP over central SBP. [7] Our study shows similar magnitude and trend in correlation coefficients (0.265 versus 0.252, P nonsignificant) and further confirms that central blood pressures are more relevant to IMT than peripheral blood pressures. Reduction of IMT is also more related to central PP reduction than lowering of mean blood pressure after treatment in 98 hypertensive patients. [37]

The present study reveals that in multivariate analyses, none of the blood pressure variables contributed to all-cause mortality, while only central SBP predicted cardiovascular mortality, even in the presence of central PP. This conclusion differs from those of the Conduit Artery Function Evaluation (CAFÉ) study of the Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT) hypertension trial,[11] in which central PP was a determinant of clinical outcome; the Strong Heart Study,[7] in which central PP was a determinant of cardiovascular events; and the Second Australian National Blood Pressure Study, in which brachial PP was independently related to cardiovascular events.[12] The major distinction from other studies is that our results further emphasize the value of central SBP as a predictor of cardiovascular risk beyond other well-known strong predictors, such as LVMI, IMT, eGFR, and indices of arterial stiffness.

Limitations of the present study

The event rate for cardiovascular mortality was low in this relative low risk study population. The low cardiovascular mortality rate might reduce the power for central PP as an independent predictor in the multivariate analyses with inclusion of two blood pressure variables. The vital status of the study population was determined from the National Death Registry database without further confirmation. However, because Taiwan's regulations for house-hold registration, personal identification registration, and death registration are strict and robust,[19] very few participants might have uncertain vital status and therefore the results of the present study would not significantly be affected. Except for the outcomes of all-cause and cardiovascular mortality, other follow-up data including the incidence of new hypertension and the status of drugs therapies in this cohort of normotensive and untreated hypertensive subjects were not available. The potential impact of blood pressure lowering or other drugs therapies on the differential effects between central and peripheral blood pressures on mortalities could not be adjusted.

In conclusion, SBP and PP relate differently to different target organs. Non-invasively determined central SBP is more valuable than other blood pressure variables in the prediction of cardiovascular mortality. The present study supports that measuring central blood pressures is of value in the management of hypertension.

Abbreviation Definitions

AI	carotid augmentation index
BMI	Body mass index
BSA	body surface area
eGFR	estimated glomerular filtration rate
HDL	high-density lipoprotein cholesterol
IMT	intima-media thickness
LVM	left ventricular mass
PP	pulse pressure
SBP	systolic blood pressure
DBP	diastolic blood pressure

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Figure 1.

Hazards ratios of the individual blood pressure variables per 10 mmHg increment for all-cause (A) and cardiovascular (B) mortality, accounting for age, sex, heart rate, BMI, current smoking, fasting plasma glucose levels, cholesterol/HDL ratio, carotid-femoral pulse wave velocity, LVM, IMT, and eGFR.

PP-B = brachial pulse pressure; PP-C = central pulse pressure; SBP-B = brachial systolic blood pressure; SBP-C = central systolic blood pressure.

Table 1

Baseline characteristics of the study population.

Variables	Women (n = 598)	Men (n = 674)	P values
Age, years	52.0 ± 12.7	52.4 ± 12.9	0.599
Hypertensive*, n (%)	212 (35.5)	217 (32.2)	0.235
Current smoking, n (%)	14 (2.3)	315 (46.7)	<0.001
Body mass index, kg/m ²	25.2 ± 4.0	24.2 ± 3.2	<0.001
Fasting Plasma Glucose, mmol/l	5.6 ± 1.4	5.4 ± 0.8	0.005
Cholesterol/HDL ratio	4.0 ± 1.2	4.3 ± 1.4	<0.001
Heart rate, bpm	73.7 ± 9.5	73.5 ± 10.4	0.629
SBP-B, mm Hg	139.4 ± 25.2	138.5 ± 22.3	0.524
DBP-B, mm Hg	86.9 ± 14.8	88.9 ± 14.3	0.019
PP-B, mm Hg	52.4 ± 17.7	49.7 ± 15.3	0.003
SBP-C, mm Hg	128.6 ± 26.2	125.9 ± 21.7	0.048
DBP-C, mm Hg	85.1 ± 14.5	86.7 ± 14.1	0.050
PP-C, mm Hg	43.9 ± 17.2	39.2 ± 13.9	<0.001
PWV, m/s	9.5 ± 2.5	9.5 ± 2.3	0.771
AI, %	19.5 ± 13.9	7.4 ± 14.5	<0.001
LVM, g	153.8 ± 44.7	174.5 ± 42.1	<0.001
LVMI, g/m ²	98.5 ± 26.4	101.4 ± 23.2	0.043
IMT, mm	1.01 ± 0.25	1.04 ± 0.28	0.012
eGFR, ml/min/1.73 m ²	100.5 ± 25.2	86.7 ± 19.8	<0.001

Hypertension was defined as SBP≥140 or DBP≥90 mmHg.[13]

AI = carotid augmentation index; eGFR = estimated glomerular filtration rate; HDL = high-density lipoprotein cholesterol; IMT = intima-media thickness; LVM = left ventricular mass; LVMI = left ventricular mass index; PP-B = brachial pulse pressure; PP-C = central pulse pressure; PWV = carotid-femoral pulse wave velocity; SBP-B = brachial systolic blood pressure; SBP-C = central systolic blood pressure.

Table 2

Relations of central and brachial blood pressure values to target organ indexes.

Variables	LVM			IMT			GFR		
	r	Partial r [†]	r [‡]	r	Partial r [†]	r [‡]	r	Partial r [†]	r [‡]
SBP-B	0.370**	0.231**	0.225**	0.101**	-0.170**	-0.041			
PP-B	0.219**	0.112**	0.204**	0.072*	-0.131**	0.032			
SBP-C	0.410**	0.270**	0.252**	0.137**	-0.179**	-0.058*			
PP-C	0.286**	0.194**	0.265**	0.127**	-0.187**	-0.012			

* P value<0.05
** P value<0.001

[†] adjusted for age, sex, heart rate, body mass index, current smoking, fasting plasma glucose, total cholesterol/high-density lipoprotein cholesterol, and carotid-femoral pulse wave velocity.

eGFR = estimated glomerular filtration rate; IMT = intima-media thickness; LVM = left ventricular mass; PP-B = brachial pulse pressure; SBP-B = brachial systolic blood pressure; SBP-C = central systolic blood pressure.

Table 3

Hazards ratios and 95% confidence intervals for all-cause and cardiovascular mortality by univariate analysis.

Variables	All-cause mortality		Cardiovascular mortality	
	Women	Men	Women	Men
Age, years	1.111 (1.082–1.142)	1.098 (1.074–1.123)	1.114 (1.060–1.170)	1.100 (1.054–1.148)
Current smoking	4.172 (1.505–11.563)	1.297 (0.828–2.030)	0.048 (0.000–>10)	1.535 (0.647–3.642)
Body mass index, kg/m ²	1.014 (0.949–1.083)	0.919 (0.854–0.989)	0.995 (0.879–1.127)	1.036 (0.908–1.182)
Fasting Plasma Glucose, mmol/l	1.140 (1.051–1.236)	0.752 (0.549–1.030)	1.183 (1.058–1.322)	0.607 (0.324–1.138)
Cholesterol/HDL ratio	1.262 (1.042–1.529)	1.070 (0.917–1.247)	1.380 (0.994–1.916)	1.289 (1.068–1.557)
PWV, m/s	1.243 (1.167–1.324)	1.182 (1.103–1.268)	1.306 (1.176–1.451)	1.223 (1.084–1.381)
AI, %	1.010 (0.990–1.030)	1.033 (1.017–1.050)	1.014 (0.979–1.051)	1.061 (1.029–1.094)
LVM, g	1.008 (1.003–1.014)	1.006 (1.002–1.011)	1.013 (1.004–1.022)	1.011 (1.003–1.018)
LVMI, g/m ²	1.018 (1.009–1.027)	1.018 (1.010–1.026)	1.028 (1.013–1.043)	1.023 (1.010–1.037)
IMT, mm	5.996 (2.432–14.781)	3.276 (1.690–6.349)	10.717 (2.510–45.761)	4.044 (1.224–13.360)
eGFR, ml/min/1.73 m ²	0.971 (0.961–0.980)	0.973 (0.962–0.983)	0.961 (0.945–0.977)	0.971 (0.951–0.991)
SBP-B, 10 mmHg	1.151 (1.040–1.273)	1.108 (1.006–1.221)	1.295 (1.083–1.547)	1.332 (1.111–1.597)
PP-B, 10 mmHg	1.250 (1.095–1.426)	1.357 (1.204–1.530)	1.408 (1.124–1.765)	1.540 (1.244–1.906)
SBP-C, 10 mmHg	1.234 (1.122–1.356)	1.114 (1.012–1.226)	1.510 (1.271–1.795)	1.353 (1.149–1.594)
PP-C, 10 mmHg	1.500 (1.318–1.708)	1.359 (1.206–1.531)	1.767 (1.413–2.210)	1.432 (1.153–1.778)

Numbers in bold letters indicate statistical significance.

AI = carotid augmentation index; eGFR = estimated glomerular filtration rate; HDL = high-density lipoprotein cholesterol; IMT = intima-media thickness; LVM = left ventricular mass; LVMI = left ventricular mass index; PP-B = brachial pulse pressure; PP-C = central pulse pressure; PWV = carotid-femoral pulse wave velocity; SBP-B = brachial systolic blood pressure; SBP-C = central systolic blood pressure.

Table 4

Multivariate Cox proportional hazards regression models relating incidence of cardiovascular mortality to dual blood pressure components of SBP-B, PP-B, SBP-C, and PP-C

	Hazards Ratio/10 mmHg (CI)	Hazards Ratio/SD increment (CI)
Model 1		
SBP-B	1.049 (0.800–1.374)	1.119 (0.590–2.122)
PP-B	1.138 (0.802–1.616)	1.238 (0.694–2.209)
Model 2		
SBP-B	0.957 (0.787–1.164)	0.902 (0.567–1.434)
SBP-C	1.336 (1.107–1.612)	2.002 (1.276–3.140)
Model 3		
SBP-B	1.076 (0.906–1.278)	1.189 (0.791–1.789)
PP-C	1.199 (0.941–1.530)	1.330 (0.908–1.947)
Model 4		
PP-B	1.042 (0.835–1.299)	1.070 (0.743–1.540)
SBP-C	1.286 (1.089–1.520)	1.828 (1.226–2.726)
Model 5		
PP-B	1.116 (0.895–1.393)	1.199 (0.832–1.728)
PP-C	1.190 (0.933–1.518)	1.314 (0.898–1.923)
Model 6		
SBP-C	1.491 (1.154–1.928)	2.606 (1.408–4.821)
PP-C	0.786 (0.541–1.141)	0.685 (0.381–1.230)

Numbers in bold letters indicate statistical significance.

All adjusted for age, sex, heart rate, BMI, current smoking, fasting plasma glucose levels, cholesterol/HDL ratio, carotid-femoral pulse wave velocity, LVM, IMT, and eGFR. PP-B = brachial pulse pressure; PP-C = central pulse pressure; SBP-B = brachial systolic blood pressure; SBP-C = central systolic blood pressure.