ORIGINAL ARTICLE

Significance of the second peak of systolic blood pressure for identifying both high and low cardiovascular risk states

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This study was conducted to clarify whether the second peak of the systolic blood pressure (SBP2) has significant information about cardiovascular (CV) risk state, independent of the brachial BP. SBP2 was measured by radial pressure wave analysis in 7847 Japanese subjects (50 ± 10 years old), and the Framingham risk score (FRS) and general cardiovascular disease risk score were calculated (FRSgen). The results of multivariate analysis revealed that the SBP2 showed a significant correlation with the FRS ($\beta=0.04$, t -value=3.92, $P<0.01$) and FRSgen ($\beta=0.05$, t -value=6.55, $P<0.01$), independent of the brachial SBP. The non-standardization coefficient of SBP2 was smaller than that of brachial SBP. The logistic regression analysis showed that SBP2 (2 mm Hg per increase) had a significant odds ratio to identify not only subjects with a high risk for coronary heart disease (CHD) and general CV disease (CVDgen), but also subjects with a low risk for CHD and CVDgen, independent of the brachial SBP. However, when the analysis was limited to subjects of ≥ 49 years of age, SBP2 could not identify either high or low CV risk subjects. Thus, in middle-aged Japanese subjects, SBP2 may provide little, yet significant, information reflecting both high and low CV risk states, independent of the brachial BP. SBP2 seems to be more applicable for CV risk stratification in younger subjects than in older subjects.

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INTRODUCTION

Pressure wave reflection in the aorta is an independent marker of cardiovascular (CV) risk,^{1,2} and the blood pressure measured in the aorta by non-invasive pressure wave analysis, the so-called central blood pressure (central BP), has been proposed to be a predictor of future CV events, independent of brachial blood pressure (brachial BP).^{3–6} The second peak of radial systolic blood pressure (SBP2) is a non-invasive surrogate marker of central BP, and this value is closely correlated with the aortic systolic BP values obtained by invasive methods in both high- and low-aortic systolic BP ranges.⁷ Brachial BP is a continuous variable reflecting the CV risk state, with CV risk increasing with increasing brachial BP.^{8,9} Furthermore, even in normal brachial BP ranges, low brachial BP reflects a low CV risk state.¹⁰ However, it is not yet clear whether low SBP2 would provide a significant additive predictive value of a low CV risk state, independent of brachial BP.

We have recently succeeded in measuring SBP2 in a large number of Japanese subjects undergoing health screenings.¹¹ This cross-sectional study was conducted to clarify whether SBP2 might provide significant information for CV risk stratification not only for high CV risk state,

but also for low CV risk state, independent of the brachial BP, in middle-aged Japanese subjects. The CV risks were assessed by the Framingham risk score (FRS), a risk marker for coronary heart disease (CHD),¹² and general CV disease (CVDgen) risk scores in the Framingham heart study (a risk marker for all initial atherosclerotic CV events).¹³ Both of these are applicable for the Japanese population.¹⁴

METHODS

Study cohort

An invitation to participate in this study was circulated among Japanese subjects undergoing health screening examinations at the Health Care Center of Tokyo Medical University between June 2006 and November 2007. This cross-sectional study was meant to establish normal values of the radial augmentation index (radial AI) and examine the clinical significance of the radial AI as a marker of CV risk in Japanese subjects. More than 90% of the subjects visiting the health care center agreed to participate in this study. In this study protocol, radial AI was measured in addition to the routine health checkup, which included evaluation of the CV risk factors. Written informed consent was obtained from each of the participants before measurement. The study was conducted with the approval of the ethical guidelines committee of Tokyo Medical University.

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The exclusion criteria included in this study protocol were as follows: (1) inaccurate measurement of the radial AI (that is, cases in which the s.d. of the radial AI was $\geq 6.0\%$), (2) patients with a permanent pacemaker and/or atrial fibrillation and (3) patients undergoing treatment for heart disease and/or cerebral disease.

Measurements

Second peak of radial SBP. The blood pressure and SBP2 were measured after the subjects had rested for 5 min in the sitting position in an air-conditioned room reserved exclusively for this purpose and kept at 24–26 °C. SBP and diastolic BP was measured in the right upper arm using an oscillometric method (HEM-907; Omron Healthcare, Kyoto, Japan). Immediately after this measurement, the left radial arterial waveform was recorded using an arterial applanation tonometry probe equipped with an array of 40 micropiezoresistive transducers (HEM-9000AI; Omron Healthcare). The HEM-9000AI device is programmed to automatically determine the pressure against the radial artery to yield the optimal radial arterial waveform, and 10 radial arterial pressure waveforms were recorded.^{15,16} This technique has been shown to be reproducible in our institute in another study.¹¹ The first and second peaks of the radial systolic pressure (SBP1 and SBP2) and radial diastolic pressure were then automatically detected, using fourth derivatives for each radial arterial waveform, and then averaged.

Laboratory measurements. The serum levels of total cholesterol, high-density lipoprotein cholesterol, triglyceride, creatinine and the fasting plasma glucose were measured using enzymatic methods. All of the blood samples were obtained in the morning after the subjects had fasted overnight.

The FRS was then calculated for each subject.¹¹ Subjects with a 10-year risk of CHD of $\geq 10\%$ as predicted by the FRS were defined as having a high CHD risk. Those with total cholesterol from 160 to 199 mg per 100 ml, high-density lipoprotein cholesterol ≥ 45 mg per 100 ml in men and ≥ 55 mg per 100 ml in women, BP of <120 mm Hg systolic and <80 mm Hg diastolic, nonsmoking and free of diabetes mellitus, were defined as having a low CHD risk.¹² In addition, the CVDgen risk score was also calculated as described in the Framingham heart study (FRSgen).¹³ The subjects with a 10-year risk of CVDgen of $\geq 20\%$ as predicted by the FRSgen were defined as having a high CVDgen risk, and subjects with a 10-year risk of CVDgen of <1% as predicted by the FRSgen were defined as having a low CVDgen risk.

Statistical analysis

Data are expressed as means $\pm$ s.d. Univariate linear regression analysis and multivariate linear regression analysis with adjustments were applied to assess the relationships among SBP2, brachial SBP, FRS and FRSgen. Logistic regression analysis was applied to assess the significance of the SBP2 for identifying subjects with a high CHD risk, high CVDgen risk, low CHD risk and low CVDgen risk. In multivariate linear regression analysis and logistic regression analysis, two adjustment models were applied: model 1=adjustment of the covariates (height, pulse rate determined using radial arterial pressure waveform analysis and conventional risk factors for CVD (body mass index, smoking status, total cholesterol, high-density lipoprotein cholesterol, triglyceride and fasting plasma glucose) and medication for hypertension, diabetes mellitus and/or dyslipidemia) and model 2, in which SBP 2, brachial SBP and the covariate in model 1 were entered simultaneously into the model. The receiver operating characteristic curve analysis was applied to obtain the area under the curve of the SBP2 and brachial SBP to identify these subjects. All analyses were conducted using SPSS software for Windows, version 11.0J (SPSS, Chicago, IL, USA); *P*-values of <0.05 were considered statistically significant.

RESULTS

Of the 8940 subjects who agreed to participate in this study (age 20–86 years old), 655 subjects were excluded because of at least one exclusion criterion (that is, inaccurate measurements of radial pressure waveform ($n=363$), permanent pacemaker and/or atrial fibrillation ($n=78$), undergoing treatment for heart disease ($n=148$) and cerebral disease ($n=92$)). In addition, 438 subjects who were under 30 years of

Table 1 Clinical characteristics of the study subjects

Age (years)	50 $\pm$ 10
Gender (men/women)	4503/3344
Height (cm)	165 $\pm$ 8
BMI (kg m ⁻²)	22.6 $\pm$ 3.3
WC (cm)	83 $\pm$ 9
Smoking status (Nev/Fo ≥ 5 /Fo <5/Curr)	3630/1739/655/1823
rAI (%)	80.2 $\pm$ 13.6
SBP1 (mm Hg)	116 $\pm$ 16
SBP2 (mm Hg)	107 $\pm$ 17
Pulse rate	68 $\pm$ 10
brSBP (mm Hg)	119 $\pm$ 17
brDBP (mm Hg)	72 $\pm$ 12
TC (mmol l ⁻¹)	5.5 $\pm$ 0.9
HDL (mmol l ⁻¹)	1.6 $\pm$ 0.4
TG (mmol l ⁻¹)	1.3 $\pm$ 1.0
FPG (mmol l ⁻¹)	5.4 $\pm$ 1.0
Crnn (μ mol l ⁻¹)	55 $\pm$ 12
FRS	0.4 $\pm$ 5.2
FRSgen	7.9 $\pm$ 5.4
Low CHD risk (%)	516 (6.6)
High CHD risk (%)	619 (7.9)
Low CVDgen risk (%)	161 (3.3)
High CVDgen risk (%)	782 (10.0)
Subjects receiving medication (%)	
For hypertension	754 (10)
For diabetes mellitus	173 (2)
For dyslipidemia	443 (6)

Abbreviations: BMI, body mass index; brDBP, brachial diastolic blood pressure in the left upper arm determined using an oscillometric method; brSBP, brachial systolic blood pressure in the left upper arm determined using an oscillometric method; Crnn, serum creatinine; Curr, current smoking; Fo ≥ 5 , former smoker who quit smoking over 5 years ago; Fo <5, former smoker who quit smoking <5 years ago; FPG, fasting plasma glucose; FRS, Framingham risk score; FRSgen, general cardiovascular disease risk scores in Framingham heart study; HDL, serum high-density lipoprotein cholesterol; High CHD risk, number of subjects with a high risk for coronary heart disease (a 10-year risk of coronary heart disease of $\geq 10\%$ as predicted by the Framingham risk score); High CVDgen risk, number of subjects with a 10-year risk of general cardiovascular disease of $\geq 20\%$ as predicted by the general cardiovascular disease risk scores in Framingham heart study; Low CHD risk, number of subjects with a low risk for coronary heart disease predicted by the Framingham risk score; Low CVDgen risk, number of subjects with a 10-year risk of general cardiovascular disease of <1% as predicted by the general cardiovascular disease risk scores in Framingham heart study; Nev, never smoking; pulse rate, pulse rate determined using radial arterial pressure waveform analysis; rAI, radial augmentation index; SBP1, first peak of the radial systolic blood pressure; SBP2, second peak of the radial systolic blood pressure; TC, serum total cholesterol; TG, serum triglycerides; WC, waist circumference.

age were excluded because of the difficulty in calculating the FRS. The data of the remaining 7847 subjects were entered into the analysis.

Table 1 shows the clinical characteristics of the study subjects. The SBP2 and brachial SBP values were divided into quintile ranges (SBP2: quintile 1=63–92 mm Hg ($n=1512$), quintile 2=93–100 mm Hg ($n=1506$), quintile 3=101–108 mm Hg ($n=1538$), quintile 4=109–119 mm Hg ($n=1611$) and quintile 5=120–183 mm Hg ($n=1680$); brachial SBP: quintile 1=73–104 mm Hg ($n=1518$), quintile 2=105–112 mm Hg ($n=1509$), quintile 3=113–121 mm Hg ($n=1638$), quintile 4=122–132 mm Hg ($n=1570$) and quintile 5=133–200 mm Hg ($n=1612$)).

Heart rate is one of the major determinants of the AI,¹¹ therefore, heart rate is thought to affect SBP2. As shown in Figure 1, the pulse rate showed a significant negative correlation with SBP2, whereas it was significantly positively associated with the brachial SBP. Figure 2 shows that a significant number of the study subjects had different BP profiles when evaluated by their distribution in the SBP2 and brachial SBP quintiles (that is, 66% of the subjects with SBP2 in quintile 1

Figure 1 The relationship of pulse rate to the second peak of the radial systolic pressure waveform and to brachial systolic blood pressure. brSBP, brachial systolic blood pressure; SBP2, second peak of radial systolic pressure waveform.

Figure 2 Variations in the second peak of the radial systolic pressure waveform with increasing levels of brachial systolic blood pressure. brSQs, quintile ranges of the brachial systolic blood pressure; S2Qs, quintile ranges of the second peak of the radial systolic pressure waveform.

range were in quintile 1 of the brachial SBP, and 74% of the subjects with SBP2 in quintile 5 range were in quintile 5 of the brachial SBP).

Univariate linear regression analysis showed a significant correlation of the FRS with the brachial SBP and SBP2 and that of the FRSgen with the brachial SBP and SBP2 (Table 2), although brachial SBP is a component of FRS and FRSgen. Multivariate linear regression analysis in model 1 showed that not only brachial SBP but also SBP2 had a significant relationship with both the FRS and the FRSgen, independent of height, pulse rate determined using radial arterial pressure waveform analysis, conventional risk factors for CVD and medications (Table 2). In the multivariate linear regression analysis in model 2, SBP2 and brachial SBP had a significant independent relationship with the FRS and the FRSgen. In these analyses, the changes in R^2 in multivariate linear regression analysis were smaller in cases when SBP2 was entered as an additional variable in model 1 for assessment of the independence of the relationship of brachial SBP with FRS and FRSgen (from 0.548 to 0.555). This is in contrast with the case in

which brachial SBP was entered as an additional variable in model 1 for assessment of the independence of the relationship of SBP2 with FRS and FRSgen (from 0.536 to 0.555; Table 2). In addition, in model 2, the non-standardization coefficient of SBP2 was smaller than that of brachial SBP (Table 2).

As shown in Table 1, 619 subjects were shown to have a high CHD risk, 516 subjects had a low CHD risk, 782 subjects had a high CVDgen risk and 161 subjects had a low CVDgen risk. Logistic regression analysis in model 1 showed that SBP2 (per 2 mmHg increase) has a significant odds ratio in identifying not only subjects with a high CHD risk and high CVDgen risk but also those with a low CHD risk and low CVDgen risk, independent of height, pulse rate determined using radial arterial pressure waveform analysis, conventional risk factors for CVD and medications (Table 3). Furthermore, logistic regression analysis in model 2 showed that SBP2 has a significant odds ratio in identifying these subjects, independent of brachial SBP (Table 3). As shown in Figure 3, the areas under the

Table 2 Results of univariate linear regression analysis and those of multivariate linear regression analysis with adjustments to assess the independency of the relationship of the second peak of the radial systolic pressure waveform with the FRS and the FRSgen

For FRS										
Univariate analysis			Multivariate analysis							
			Model 1 ($R^2=0.536$)				Model 2 ($R^2=0.555$)			
	β	P-value	Non-stand B (95%CI)	β	t-value	P-value	Non-stand B (95% CI)	β	t-value	P-value
SBP2	0.55	<0.01	0.129 (0.124–0.134)	0.42	49.94	<0.01	5.351×10^{-2} (0.044–0.063)	0.17	11.13	<0.01
Model 1 ($R^2=0.548$)										
	β	P-value	Non-stand B (95% CI)	β	t-value	P-value				
brSBP	0.56	<0.01	0.138 (0.132–0.143)	0.45	52.67	<0.01	9.088×10^{-2} (0.081–0.101)	0.30	18.47	<0.01
For FRSgen										
Univariate analysis			Multivariate analysis							
			Model 1 ($R^2=0.673$)				Model 2 ($R^2=0.699$)			
	β	P-value	Non-stand B (95% CI)	β	t-value	P-value	Non-stand B (95% CI)	β	t-value	P-value
SBP2	0.62	<0.01	0.147 (0.143–0.152)	0.46	65.73	<0.01	5.674×10^{-2} (0.049–0.065)	0.18	13.86	<0.01
Model 1 ($R^2=0.692$)										
	β	P-value	Non-stand B (95% CI)	β	t-value	P-value				
brSBP	0.65	<0.01	0.159 (0.154–0.163)	0.50	71.12	<0.01	0.109 (0.101–0.118)	0.35	26.06	<0.01

Abbreviations: β , standardization coefficient; brSBP, brachial systolic blood pressure in the left upper arm determined using an oscillometric method; CI, confidence interval; FRS, Framingham risk score; FRSgen, general cardiovascular disease risk scores in Framingham heart study; Model 1, adjusted of the covariates (height, pulse rate determined using radial arterial pressure waveform analysis, conventional risk factors for cardiovascular disease (body mass index, smoking status, serum total cholesterol, serum high-density lipoprotein cholesterol, serum triglycerides and fasting plasma glucose) and medication for hypertension, diabetes mellitus and/or dyslipidemia); Model 2, the second peak of the radial systolic pressure waveform, the brachial systolic blood pressure and the covariates in model 1 were entered simultaneously into the multivariate linear regression model; Non-stand B, non-standardization coefficient; SBP2, second peak of the radial systolic pressure waveform.

receiver operating characteristic curves for SBP2 and brachial SBP were comparable for the identification of not only subjects with a high CHD risk and high CVDgen risk but also subjects with a low CHD risk and low CVDgen risk. In model 2, however, the adjusted odds ratio of SBP2 was smaller than those of brachial SBP except to identify low CVDgen risk state.

The median age of the subjects in this study was 49 years. In subjects of <49 years of age, logistic regression analysis using model 2 showed a significant independent adjusted odds ratio of SBP2 to identify subjects with a high CHD risk, low CHD risk and low CVDgen risk, but not those with a high CVDgen risk (Table 4). In subjects aged ≥ 49 years (none of the subjects ≥ 49 years old was categorized as having a low CVDgen risk), logistic regression analysis using model 2 showed no significant independent adjusted odds ratio of SBP2 to identify either high or low CV risk subjects (Table 4).

DISCUSSION

Consistent with a previous report,¹⁷ this study confirmed substantial inter-individual variations of the SBP2 through all quintile ranges of brachial SBP. Thus, SBP2 and brachial SBP may partially reflect different hemodynamic conditions.

Guidelines for the management of high blood pressure classify the brachial BP into various categories of severity: optimal, prehypertension (normal and high normal) and stage I–III hypertension, and the

CV risk is known to increase with increasing brachial BP levels.^{8,9} On the other hand, in the treatment of high blood pressure, the concept that lower blood pressure is better is well recognized.^{8,9} Thus, brachial BP is an established continuous variable reflecting the CV risk state. In this study, SBP2 had a significant relationship with FRS as well as FRSgen, independent of brachial SBP and covariate, including height and pulse rate. However, the strength of the effect of SBP2 on FRS and FRSgen might be small because the changes in the R^2 value in multivariate linear regression analysis were small when SBP2 was entered as an additional variable into model 1 for assessment of the independence of the relationship of brachial SBP with FRS and FRSgen. In model 2, the non-standardization coefficient of SBP2 was smaller than that of brachial SBP. In addition, we showed a significant adjusted odds ratio of the SBP2 for identifying not only subjects with a high CHD risk and high CVDgen risk, but also subjects with a low CHD risk and low CVDgen risk, independent of brachial SBP. However, the value of the adjusted odds ratio of SBP2 for identifying these subjects was smaller than those of brachial SBP, except for identifying subjects with low CVDgen risk. Thus, the SBP2 may be a continuous variable that provides additional information reflecting the cardiovascular risk states, including low and high CV risk states, to the brachial BP. However, the value of this information might be small.

Table 3 Results of logistic regression analysis without and with adjustments to assess the significance of the second peak of the radial systolic pressure waveform for the identification of subjects with high and low CHD risk and high and low CVDgen risk

Variable	Crude odds (95% CI)	P-value	Model 1 AdOdds (95% CI)	P-value	Model 2 AdOdds (95% CI)	P-value
<i>To identify subjects with a high CHD risk</i>						
SBP2 (per 2 mm Hg increase)	1.061 (1.056–1.066)	<0.01	1.075 (1.068–1.083)	<0.01	1.016 (1.004–1.028)	<0.01
brSBP (per 2 mm Hg increase)	1.068 (1.062–1.073)	<0.01	1.086 (1.079–1.094)	<0.01	1.072 (1.059–1.086)	<0.01
<i>To identify subjects with a low CHD risk</i>						
SBP2 (per 2 mm Hg increase)	0.940 (0.933–0.947)	<0.01	0.956 (0.948–0.964)	<0.01	0.988 (0.975–1.000)	0.04
brSBP (per 2 mm Hg increase)	0.941 (0.934–0.948)	<0.01	0.946 (0.938–0.955)	<0.01	0.956 (0.942–0.969)	<0.01
<i>To identify subjects with a high CVDgen risk</i>						
SBP2 (per 2 mm Hg increase)	1.064 (1.059–1.069)	<0.01	1.075 (1.068–1.081)	<0.01	1.018 (1.007–1.029)	<0.01
brSBP (per 2 mm Hg increase)	1.072 (1.067–1.077)	<0.01	1.085 (1.077–1.092)	<0.01	1.069 (1.057–1.080)	<0.01
<i>To identify subjects with a low CVDgen risk</i>						
SBP2 (per 2 mm Hg increase)	0.906 (0.892–0.920)	<0.01	0.947 (0.931–0.964)	<0.01	0.963 (0.938–0.988)	<0.01
brSBP (per 2 mm Hg increase)	0.910 (0.897–0.924)	<0.01	0.948 (0.931–0.965)	<0.01	0.978 (0.951–1.000)	0.04

Abbreviations: brSBP, brachial systolic blood pressure in the left upper arm determined using an oscillometric method; CHD, coronary heart disease; CI, confidence interval; CVDgen, general cardiovascular disease; Model 1 adOdds, odds ratio adjusted of the covariates in model 1 (the details are described in the footnote of Table 2); Model 2 adOdds, odds ratio obtained by the second peak of the radial systolic pressure waveform, the brachial systolic blood pressure and the covariates in model 1 were entered simultaneously into the model; SBP2, second peak of the radial systolic pressure waveform.

Figure 3 Receiver operator characteristic curves for the second peak of the radial systolic pressure waveform and brachial systolic blood pressure to identify subjects at high risk for coronary heart disease, low risk for coronary heart disease, low general cardiovascular disease risk and high general cardiovascular disease risk. AUC, area under the curve and 95% confidence intervals; brSBP, brachial systolic blood pressure (dotted line); high CHD risk, high risk for coronary heart disease (a 10-year risk of coronary heart disease of $\geq 10\%$ as predicted by the Framingham risk score); high CVDgen risk, 10-year risk of general cardiovascular disease of $\geq 20\%$ as predicted by the general cardiovascular disease risk scores in the Framingham heart study; low CHD risk, low risk for coronary heart disease predicted by the Framingham risk score; low CVDgen risk, 10-year risk of general cardiovascular disease of $< 1\%$ as predicted by the general cardiovascular disease risk scores in the Framingham heart study; SBP2, second peak of radial systolic pressure waveform (solid line).

Central BP obtained by invasive methods has been shown to be a marker of the severity of coronary atherosclerosis and/or a predictor of the prognosis of subjects with coronary artery disease.^{18,19} Furthermore, central BP estimated by non-invasive methods has also been shown to be a predictor of future CV events in hypertensive and elderly subjects, as well as in subjects with a high risk for CV disease.^{4–6} The conventional risk factors for CV disease cause macro- and/or microvascular dysfunction.^{20,21} The FRS and FRSgen are calculated based on these risk factors.^{12,13} SBP2 is estimated by the pressure wave reflection, and the aforementioned vascular dysfunction is a major determinant of the elevation of SBP2 and mediates CV events.^{1,2,20,21}

Thus, such dysfunction might contribute, at least in part, to the independent relationship of SBP2 to FRS and FRSgen.

This study used FRS and FRSgen as a marker of CV risk, and age is one of the components used in the FRS and FRSgen calculations.^{12,13} McEniery *et al.*²² suggested that pressure wave analysis might be a more sensitive marker of CV risk in younger subjects than in older subjects, and the ANBP2 study reported that central BP is not an independent variable to predict future CV events in elderly women.²³ The results of the present study were consistent with the previous findings, indicating that SBP2 is useful in identifying not only high CV risk state but also low CV risk state in younger subjects.

Table 4 Results of logistic regression analysis with the adjustment (model 2) to assess the significance of the second peak of the radial systolic pressure waveform for the identification of subjects with high and low CHD risk and high and low CVDgen risk in subgroups divided by the median of age

Variable	Aged < 49 years		Aged ≥ 49 years	
	Model 2 AdOdds (95% CI)	P-value	Model 2 AdOdds (95% CI)	P-value
<i>To identify subjects with a high CHD risk</i>				
SBP2 (per 2 mm Hg increase)	1.051 (1.000–1.100)	0.04	1.009 (0.996–1.022)	0.18
brSBP (per 2 mm Hg increase)	1.068 (1.009–1.130)	0.02	1.074 (1.059–1.088)	<0.01
<i>To identify subjects with a low CHD risk</i>				
SBP2 (per 2 mm Hg increase)	0.978 (0.962–0.994)	<0.01	1.022 (0.996–1.048)	0.11
brSBP (per 2 mm Hg increase)	0.982 (0.966–0.999)	0.04	0.894 (0.869–0.920)	<0.01
<i>To identify subjects with a high CVDgen risk</i>				
SBP2 (per 2 mm Hg increase)	1.047 (0.979–1.118)	0.18	1.008 (0.996–1.021)	0.18
brSBP (per 2 mm Hg increase)	1.075 (0.997–1.160)	0.06	1.078 (1.064–1.091)	<0.01
<i>To identify subjects with a low CVDgen risk^a</i>				
SBP2 (per 2 mm Hg increase)	0.971 (0.946–0.996)	0.02	—	—
brSBP (per 2 mm Hg increase)	0.983 (0.956–1.011)	0.24	—	—

Abbreviations: brSBP, brachial systolic blood pressure in the left upper arm determined using an oscillometric method; CHD, coronary heart disease; CI, confidence interval; CVDgen, general cardiovascular disease; Model 2 adOdds, odds ratio obtained by the second peak of the radial systolic pressure waveform, the brachial systolic blood pressure and the covariates in model 1 were entered simultaneously into the model; SBP2, second peak of the radial systolic pressure waveform.

^aNone of the subjects ≥49 years old was categorized as having a low CVDgen risk.

The study limitations were as follows. First, the Strong Heart Study has already shown that central BP is more important than brachial BP in predicting cardiovascular outcomes.⁶ However, the present study could not confirm the strength of SBP2 as an independent marker of CV risk stratification. In addition, whereas some studies have shown that radial AI serves as a reasonably accurate non-invasive estimate of the pressure wave reflection in the aorta,^{15,16} the accuracy of SBP2 as a surrogate marker of central SBP is still discussed.²⁴ Confirmation of the present results using carotid arterial pressure wave analysis is recommended for future study.

CONCLUSION

In middle-aged Japanese subjects, the SBP2 may provide little, yet significant, information reflecting both high and low CV risk states independent of brachial BP. SBP2 seems to be more applicable for CV risk stratification in younger subjects than in older subjects.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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