Original Article

Relationship between Radial and Central Arterial Pulse Wave and Evaluation of Central Aortic Pressure Using the Radial Arterial Pulse Wave

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Since a decrease of central aortic pressure contributes to the prevention of cardiovascular events, simple measurement of not only brachial blood pressure but also central aortic pressure may be useful in the prevention and treatment of cardiovascular diseases. In this study, we simultaneously measured radial artery pulse waves non-invasively and ascending aortic pressure invasively, before and after the administration of nicorandil. We then compared changes in central aortic pressure and radial arterial blood pressure calibrated with brachial blood pressure in addition to calculating the augmentation index (AI) at the aorta and radial artery. After nicorandil administration, the reduction in maximal systolic blood pressure in the aorta (*Δ*a-SBP) was –14 ± 15 mmHg, significantly larger than that in early systolic pressure in the radial artery (*Δ*r-SBP) (–9 ± 12 mmHg). The reduction in late systolic blood pressure in the radial artery (*Δ*r-SBP2) was –15 ± 14 mmHg, significantly larger than *Δ*r-SBP, but not significantly different from *Δ*a-SBP. There were significant relationships between *Δ*a-SBP and *Δ*r-SBP (r=0.81, p<0.001), and between *Δ*a-SBP and *Δ*r-SBP2 (r=0.91, p<0.001). The slope of the correlation regression line with *Δ*r-SBP2 (0.83) was larger and closer to 1 than that with *Δ*r-SBP (0.63), showing that the relationship was close to 1:1. Significant correlations were obtained between aortic AI (a-AI) and radial AI (r-AI) (before nicorandil administration: r=0.91, p<0.001; after administration: r=0.70, p<0.001). These data suggest that the measurement of radial artery pulse wave and observation of changes in the late systolic blood pressure in the radial artery (r-SBP2) in addition to the ordinary measurement of brachial blood pressure may enable a more accurate evaluation of changes in maximal systolic blood pressure in the aorta (a-SBP). (*Hypertens Res* 2007; 30: 219–228)

***Key Words*: central aortic pressure, late systolic blood pressure, pulse, vasodilator agents, augmentation index**

Introduction

It is well known that blood pressure management is important for the prevention of cardiovascular events (*1*, *2*). Elevation of the central aortic blood pressure induces coronary arteriosclerosis, which easily causes various adverse events such as stenosis and myocardial infarction (*3*–*6*). Brachial blood pressure, which is usually measured in clinical settings, is an essential parameter for the evaluation and management of central aortic pressure. Since the observation and reduction of central aortic pressure contribute to the prevention of cardiovascular events, simple measurement of not only brachial blood pressure but also central aortic pressure may be useful in the prevention and treatment of cardiovascular diseases. The large-scale ASCOT-CAFE study reported that central

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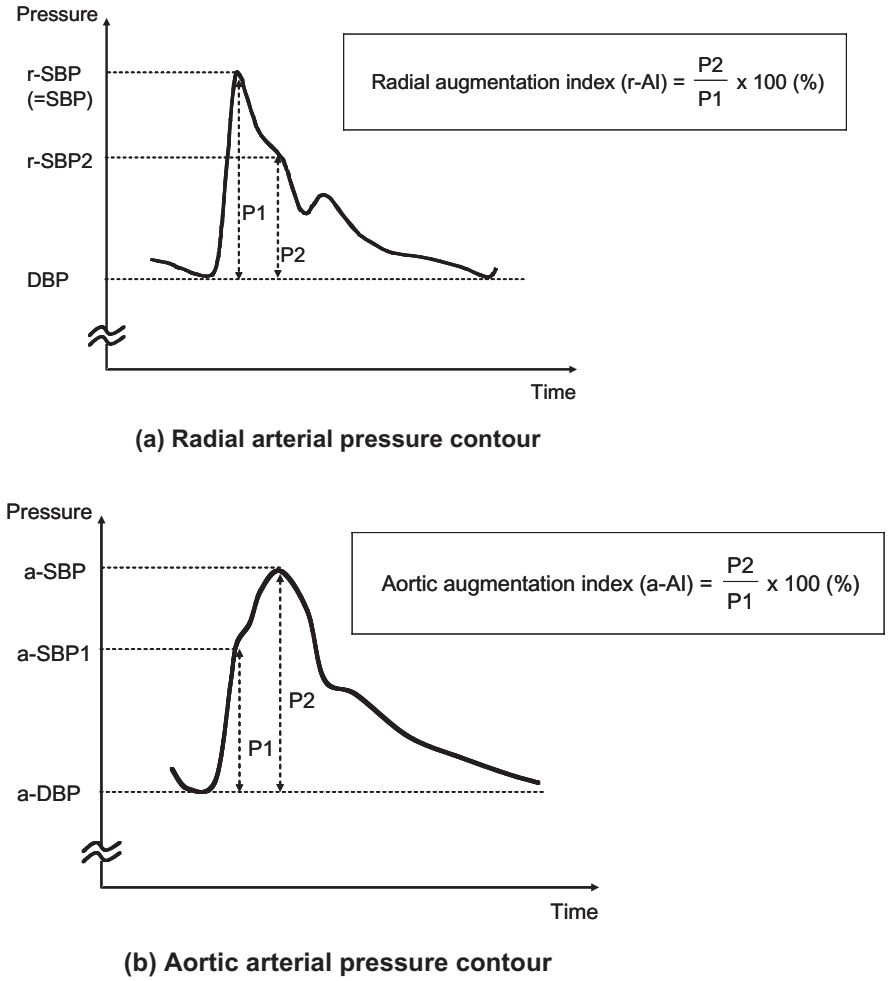


Fig. 1. *Schema of the radial (a) and aortic (b) arterial pressure contours. P1 and P2 indicate the height of the early systolic shoulder/peak pressure and that of the late systolic shoulder/peak pressure, respectively.*

aortic pulse pressure may be a determinant of clinical outcomes and that brachial blood pressure is not always a good indicator of the effect of blood pressure–lowering drugs on arterial hemodynamics (*7*). Wilkinson *et al*. (*8*) also insisted that the CAFE study would seem to support the view that blood pressure lowering *per se* matters, but that it is central and not brachial pressure they should be interested in, and predicted that the importance of central aortic pressure management will increase.

Practically, however, central aortic pressure measurement requires invasive catheterization, and thus, brachial blood pressure, which can be easily measured using a manchette, is measured as a substitute. Central aortic pressure has been measured invasively by inserting a catheter into the heart, but is estimated by non-invasive methods in most cases. In addition to the manchette method described above, arterial blood pressure pulse wave measurement by tonometry is also available as a non-invasive method. Measurement of pressure pulse waves of the carotid artery (*9*, *10*), which is near the heart, and estimation of central pulse waves from radial arterial pulse waves using transfer functions (*7*, *11*, *12*) (one of these functions (*7*, *12*) has been adopted in a commercially available system [SphygmoCor]) are used substitutionally for central aortic pressure information. The relations of carotid arterial pulse wave or central pulse wave information estimated from the radial artery to cardiovascular events and diseases have been reported (*3*), and a close relation to invasive central pulse wave information has also been reported (*13*).

Each method described above seems to have some problems. For example, breathing movements interfere with carotid arterial pulse waves, and this measurement requires an extended period of time, while estimation of central pulse waves from the radial artery provokes concern about applying only one transfer function to all patients. Millasseau *et al*. (*14*) questioned the use of a transfer function and suggested that similar information on central pressure wave reflection can be obtained directly from the radial pulse.

It has been reported that ordinary brachial blood pressure

Table 1. Baseline Characteristics of Patients

Number of patients	18
Age (years old)	61±10 (47–78)
Sex (M/F)	15/3
Height (cm)	162±10
Weight (kg)	65±15
Hypertension	10
Hyperlipidemia	10
Diabetes mellitus	7
Angina pectoris	10
Myocardial infarction	4
Left ventricular dysfunction	4
Left ventricular hypertrophy	3
Valvular dysfunction	1
Medications	
Nitrates	6
β-Blockers	4
ACE inhibitors	3
Angiotensin recepter blockers	3
Calcium channel blockers	7
Statins	5

M, male; F, female; ACE, angiotensin II converting enzyme.

measurement alone without observation of pressure pulse waves causes an underestimation of the effect of vasodilators on the ascending aorta. Also, the possibility of the concomitant measurement of radial arterial pulse waves to evaluate the drug-induced reduction of ascending aortic blood pressure has been reported (*15–18*). However, there have been few reports on the estimation and evaluation of central aortic pressure using non-invasive radial arterial pulse wave measurements (*13*). In this study, we simultaneously measured radial arterial pulse waves non-invasively by a tonometry method using a newly developed radial arterial pulse wave measurement system, and ascending aortic pressure using a catheter before and after the administration of a vasodilator, and compared changes in central aortic pressure and radial arterial blood pressure corrected by brachial blood pressure to investigate the possibility of evaluating central aortic pressure using radial arterial pulse waves. In addition, pressure pulse wave information of the aorta and radial artery were compared.

Methods

Subjects

Twenty patients underwent cardiac catheterization at our institution between October 2003 and December 2003. Of these patients 18 (15 males and 3 females) were found to be eligible for this study, with 2 patients in whom a stable pulse wave could not be measured due to arrhythmia being excluded. All procedures were approved by the ethics committee of Tokyo Medical University, and informed consent was obtained from all patients.

Study Protocol

Cardiac catheterization was performed by brachial arterial or femoral arterial puncture. Ascending aortic pressure was measured using a pressure guide wire (PRESSURE WIRE; RADI Medical Systems, Gothenburg, Sweden), and recorded on a laptop personal computer through a SEIREG polygraph (MICOR; Siemens, Solna, Sweden). Radial arterial pulse waves were simultaneously measured non-invasively by tonometry (HEM-9000AI prototype; Omron Healthcare Co., Ltd., Kyoto, Japan), and recorded on the same laptop computer. Radial arterial blood pressure was calibrated with brachial blood pressure automatically measured by oscillometry (TM2740; Colin Medical Technology Co., Komaki, Japan). Administration of antihypertensive drugs and oral vasodilators was avoided for 24 h before cardiac catheterization. After measurement of both aortic and radial artery pressure pulse waves for 40 s and brachial blood pressure under the control condition, 8 mg nicorandil was injected intravenously for 2 min. After the completion of intravenous injection, aortic and radial artery pressure pulse waves were again measured for 40 s, as well as brachial blood pressure. Both before and after administration, brachial blood pressure was measured immediately after the pulse wave measurements.

Pulse Wave Analysis

The non-invasive radial arterial pulse wave measurement system consists of a tonometry sensor unit, radial pulse measurement unit, and laptop personal computer. The sensor unit has a pressure sensor consisting of an array of 40 microtransducer elements. As one of these 40 sensor elements is automatically selected to obtain optimal radial arterial pressure waveforms, this method is thought to be a more objective approach. Signals of the ascending aortic and radial arterial pulse waves were low pass-filtered at a cut-off frequency of about 25 Hz and 105 Hz, respectively. Then, both waves were simultaneously recorded on the laptop personal computer at a sampling frequency of 500 Hz. The radial arterial pulse waveforms obtained with this device are reported to be identical to the simultaneously and invasively measured intra-arterial pulse waveforms of the opposite radial artery (*19*). Radial arterial pressure pulse waves were also accurately measured in our study. The coefficients of variation of the intra- and inter-observer measurement of radial augmentation index (AI) by this device were 3.6% and 2.4%, respectively, showing that it has a good reproducibility.

Inflection points or peaks that corresponded to early and late systolic blood pressure were obtained by multidimensional derivatives of the original pressure pulse waveforms. As an index of wave reflection (*12*, *16*, *20*, *21*), AI was defined as the ratio of the height (P2/P1) of the late systolic

Table 2. Differences of the Parameters before and after the Injection of Nicorandil

Parameter	Before	After	Differences	*p* value
Aortic				
SBP (mmHg)	115±22	101±19	−14±15	<0.001
DBP (mmHg)	64±9	59±12	−5±7	<0.005
PP (mmHg)	51±20	42±14	9±9	<0.001
AI (%)	154±40	131±31	−23±20	<0.001
Radial				
SBP (mmHg)	119±17	110±18	−9±12	<0.005
DBP (mmHg)	75±8	68±12	−6±8	<0.005
SBP2 (mmHg)	104±21	89±22	−15±14	<0.001
PP (mmHg)	45±13	42±10	3±8	n.s.
PP2 (mmHg)	29±16	20±13	8±8	<0.001
AI (%)	63±22	48±21	−15±15	<0.001
Pulse rate (bpm)	61±8	66±8	5±3	<0.001

SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; AI, augmentation index; SBP2, late SBP; PP2, late PP; n.s., not significant.

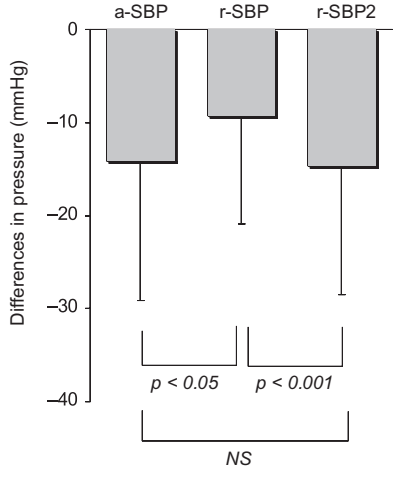


Fig. 2. *Comparison of the changes in a-SBP, r-SBP, and r-SBP2.*

shoulder/peak (P2) to that of the early systolic shoulder/peak (P1) (Fig. 1). The maximal systolic blood pressure (r-SBP) and diastolic pressure in the radial artery were corrected to the brachial systolic blood pressure and brachial diastolic blood pressure, respectively. The late systolic blood pressure in the radial artery (r-SBP2) was calculated by the following equation:

$$\text{r-SBP2 [mmHg]} = (\text{P2/PPh}) \times (\text{SBP [mmHg]} - \text{DBP [mmHg]}) + \text{DBP [mmHg]}$$

where PPh, wave height corresponding to radial arterial pulse pressure; SBP and DBP, brachial systolic and diastolic blood pressures, respectively.

For early and late systolic blood pressure and the AI of the aorta and radial artery before and after nicorandil administration, the means of 10 stable consecutive pulses immediately prior to brachial blood pressure measurement were used. The absence of a 10-mmHg or larger variation of blood pressure was confirmed during pulse wave measurement.

Statistical Analysis

All results are expressed as the means±SD unless otherwise specified. The Wilcoxon signed rank test was used for analysis of differences between before and after drug administration, and a *p*-value less than 0.05 was considered statistically significant. In the correlation analysis among the parameters, Pearson's correlation coefficients were calculated.

Results

The baseline characteristics of the patients are shown in Table 1. The medical history of the patients revealed previous or current hypertension in 10 patients, hyperlipidemia in 10, diabetes mellitus in 7, angina pectoris in 10, and myocardial infarction in 4. From observations of ultrasonic echocardiography and electrocardiography, 4 patients with left ventricular dysfunction (ejection fraction < 55), 3 patients with left ventricular hypertrophy, and 1 patient with valvular dysfunction (aortic and mitral insufficiency) were found. The numbers of patients taking vasoactive drugs are also shown in Table 1.

Change in Central Aortic Pressure

The parameters before and after nicorandil administration, and their changes are shown in Table 2. The central aortic pressure (a-SBP) decreased from 115±22 to 101±19 mmHg, and r-SBP decreased from 119±17 to 110±18 mmHg. The reduction in a-SBP (*Δ*a-SBP) was −14±15 mmHg, signifi-

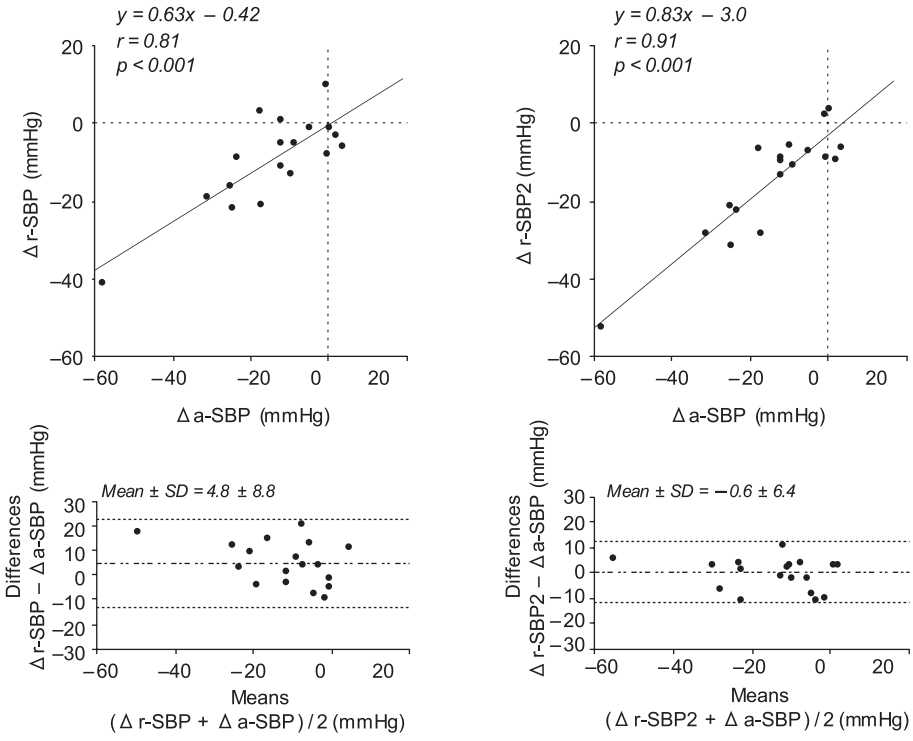


Fig. 3. *Top: Correlation between Δa-SBP and Δr-SBP or Δr-SBP2. Bottom: Bland-Altman plot of two parameters: Δa-SBP and Δr-SBP; Δa-SBP and Δr-SBP2.*

cantly larger than that in r-SBP (*Δ*r-SBP) (−9±12 mmHg, $p<0.05$). On the other hand, r-SBP2 decreased from 104±21 to 89±22 mmHg, and the reduction in r-SBP2 (*Δ*r-SBP2) was −15±14 mmHg, which was significantly larger than *Δ*r-SBP ($p<0.001$), but not significantly different from *Δ*a-SBP (Fig. 2). Significant positive relationships between *Δ*a-SBP and *Δ*r-SBP or *Δ*r-SBP2 ($r=0.81$, $p<0.001$ and $r=0.91$, $p<0.001$, respectively) were identified (Fig. 3, top). The slope of the correlation regression line with *Δ*r-SBP2 (0.83) was larger ($p=0.186$, not significant) and closer to 1 than that with *Δ*r-SBP (0.63), showing that the relationship was close to 1:1. The mean error and standard deviation of the Bland-Altman plot were 4.8±8.8 mmHg and −0.6±6.4 mmHg, respectively, showing that the standard deviations of the error between *Δ*a-SBP and *Δ*r-SBP2 were smaller (Fig. 3, bottom).

Change in Central Aortic Pulse Pressure

Changes in pulse pressure after nicorandil administration were compared between the central and peripheral arteries. There was a significant correlation ($r=0.59$, $p<0.01$) between the changes in central aortic pulse pressure (*Δ*a-PP) and the changes in radial arterial pulse pressure (*Δ*r-PP) after nicorandil administration. The changes in radial arterial late systolic pulse pressure (*Δ*r-PP2), calculated by subtracting radial arterial DBP from r-SBP2, were significantly and positively correlated with *Δ*a-PP ($r=0.92$, $p<0.001$). The slope of the correlation regression line with *Δ*r-PP2 (0.83) was larger ($p=0.113$, not significant) than that with *Δ*r-PP (0.51) and close to 1, showing that the relationship was close to 1:1 (Fig. 4, top). The mean error and standard deviation of the Bland-Altman plot were 6.4±7.8 mmHg and 1.0±3.7 mmHg, respectively, showing that the standard deviations between *Δ*a-PP and *Δ*r-PP2 were smaller (Fig. 4, bottom).

Central Aortic Pressure

The relationships between a-SBP and r-SBP or r-SBP2 are shown in Fig. 5 (a: before nicorandil administration; b: after administration). There were significant positive correlations between a-SBP and r-SBP (before: $r=0.88$, $p<0.001$; after: $r=0.85$, $p<0.001$) and between a-SBP and r-SBP2 (before: $r=0.95$, $p<0.001$; after: $r=0.93$, $p<0.001$). The slopes of the correlation regression lines between a-SBP and r-SBP were 0.67 (before) and 0.80 (after). The slopes between a-SBP and r-SBP2 were 0.91 (before) and 1.06 (after). The slopes between a-SBP and r-SBP2 were larger (before: $p=0.052$, not significant; after: $p=0.128$, not significant) than those with r-SBP and close to 1, showing that the relationship between a-SBP and r-SBP2 was close to 1:1. The mean error and standard deviation of the Bland-Altman plot between a-SBP and r-SBP were 4.0±10.6 mmHg (before) and 8.8±10.1 mmHg (after). Those between a-SBP and r-SBP2 were −11.7±7.1 mmHg (before) and −12.3±8.4 mmHg (after). These results

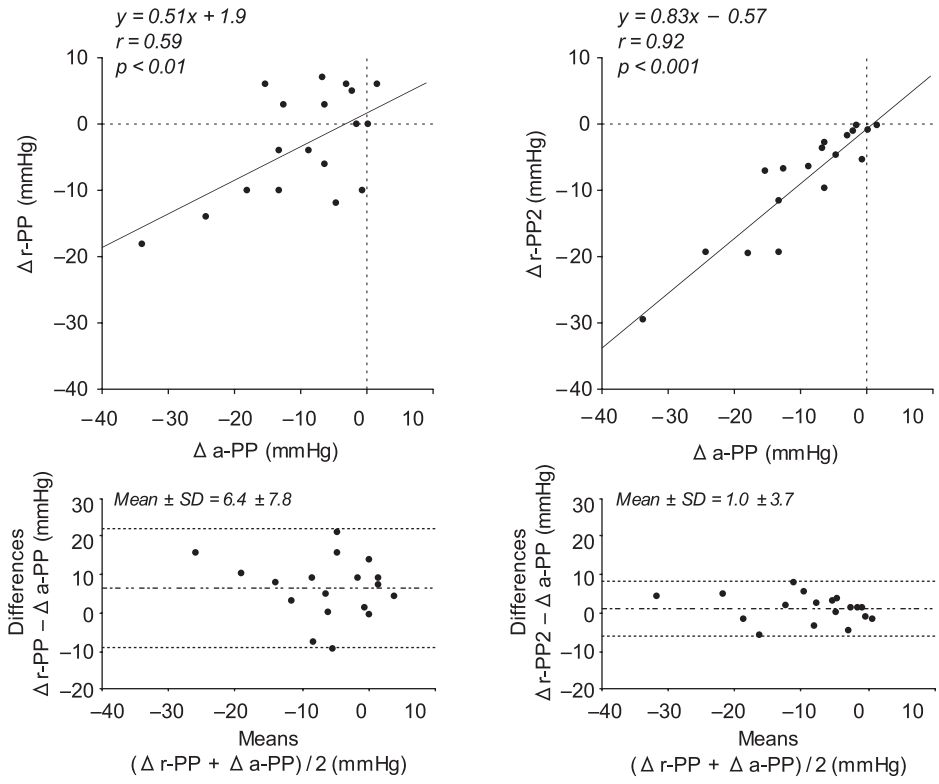


Fig. 4. *Top: Correlation between Δa-PP and Δr-PP or Δr-PP2. Δr-PP2: the changes of [r-SBP2 − diastolic blood pressure] after the injection of nicorandil. Bottom: Bland-Altman plot of two parameters: Δa-PP and Δr-PP; Δa-PP and Δr-PP2.*

show that the standard deviations were smaller in the relationship between a-SBP and r-SBP2.

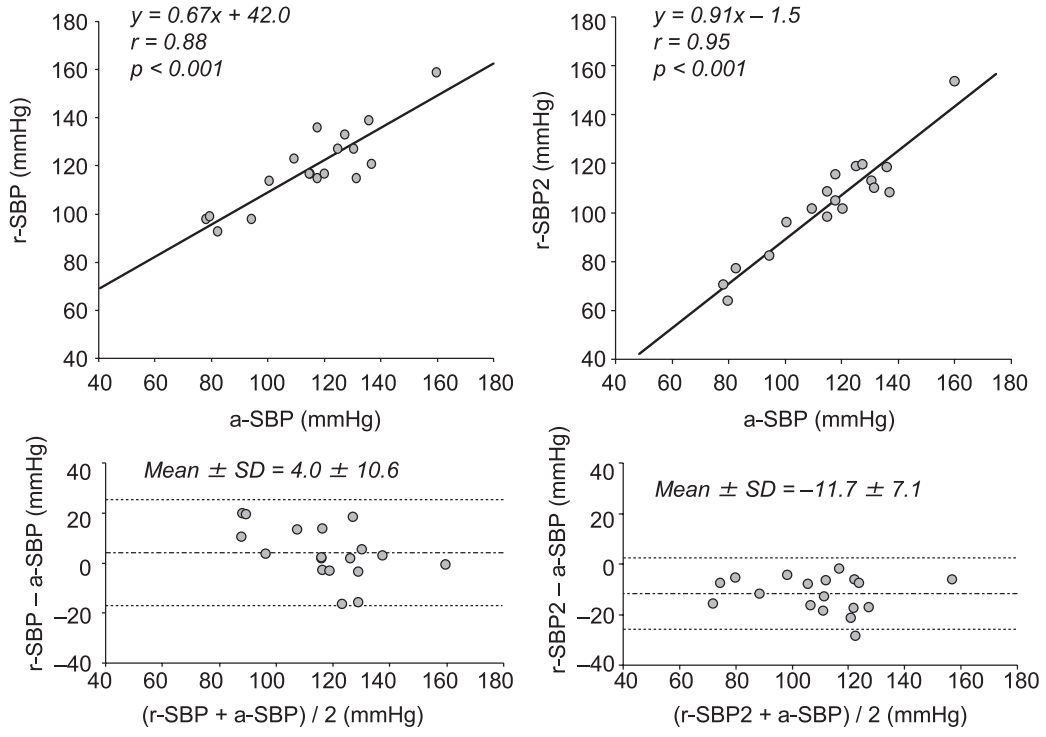
AI in the Aorta and Radial Artery

On comparison of the AI between the central and peripheral arteries, there was a significant positive correlation between central aortic AI (a-AI) and peripheral radial AI (r-AI) (before nicorandil administration: $r=0.91$, $p<0.001$; after administration: $r=0.70$, $p<0.001$), as shown in Fig. 6.

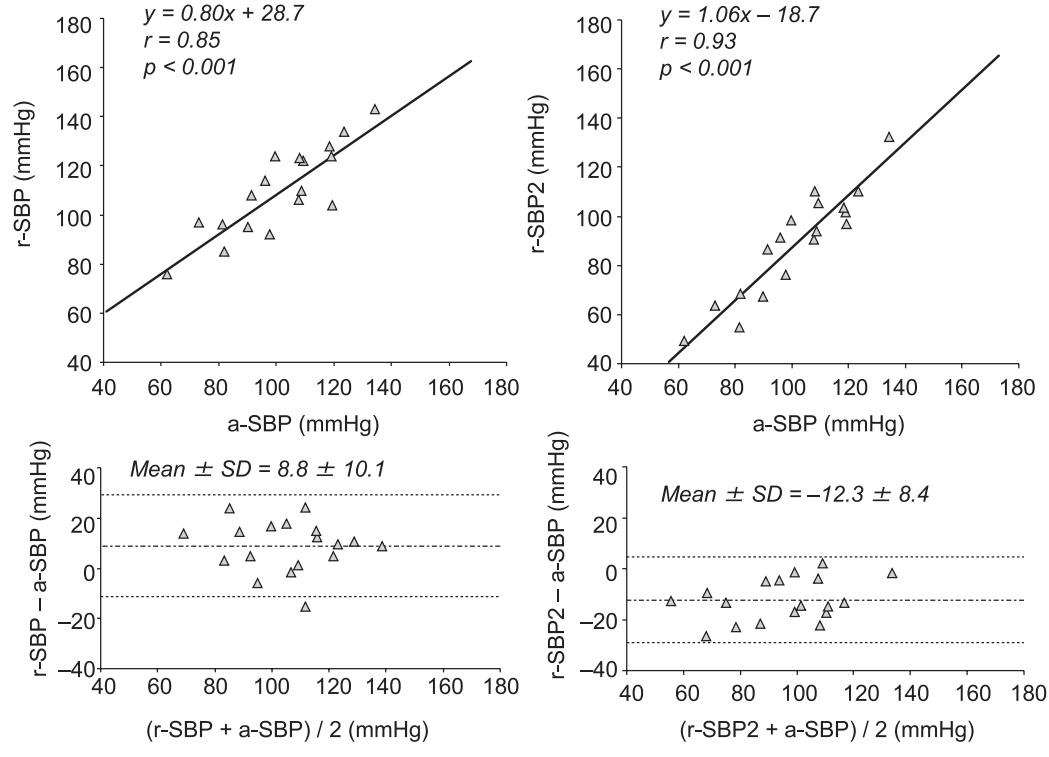
Discussion

We investigated whether central aortic pressure can be evaluated by the non-invasive measurement of not only brachial blood pressure but also radial arterial pulse waves, in addition to observing the relationship between peripheral and central pulse wave information. When a vasodilator, nicorandil, was intravenously injected, the decrease in a-SBP was significantly larger than the decrease in r-SBP (SBP: brachial systolic blood pressure), and similar to the decrease in r-SBP2. These findings were consistent with previous studies by Kelly *et al*. (*15*) and Takazawa *et al*. (*16*), who reported that ordinary peripheral artery blood pressure measurement underestimates the vasodilative effects on the ascending aorta. This may be due to marked differences in the pressure pulse waveforms at the aorta and radial artery, as previously reported. Although, in the ascending aorta, maximal systolic pressure usually coincides with the late systolic peak, in the radial arteries it usually coincides with the first systolic peak. In this study, since nicorandil reduces late systolic pressure in the ascending aorta by reducing the reflection wave (as does nitroglycerin (*22*)), the reduction in late systolic pressure in the radial artery was observed with the same reduction in maximal systolic pressure in the ascending aorta. We also investigated the relationship between aortic AI and radial AI because not only central aortic pressure but also aortic AI are important clinically. For example, Ueda *et al*. (*23*) reported that central aortic AI is related to restenosis after percutaneous coronary stenting.

As shown in Fig. 3, the correlation coefficient between Δa-SBP and Δr-SBP2 (0.91) was higher than that between Δa-SBP and Δr-SBP (0.81). The slope of the correlation regression line between Δa-SBP and Δr-SBP was 0.83 and was closer to 1. Based on these findings, observation of r-SBP alone may underestimate changes in a-SBP, but additional observation of r-SBP2 may enable us to more accurately evaluate the changes in a-SBP. These results suggest that the observation of SBP2 reductions, thought to represent decreases in both brachial blood pressure and the reflected wave, may be useful in evaluating the reduction of blood pressure, particularly changes in central aortic pressure



(a) Before nicorandil administration



(b) After nicorandil administration

Fig. 5. *Top: Correlation between a-SBP and r-SBP or r-SBP2. Bottom: Bland-Altman plot of two parameters: a-SBP and r-SBP; a-SBP and r-SBP2.*

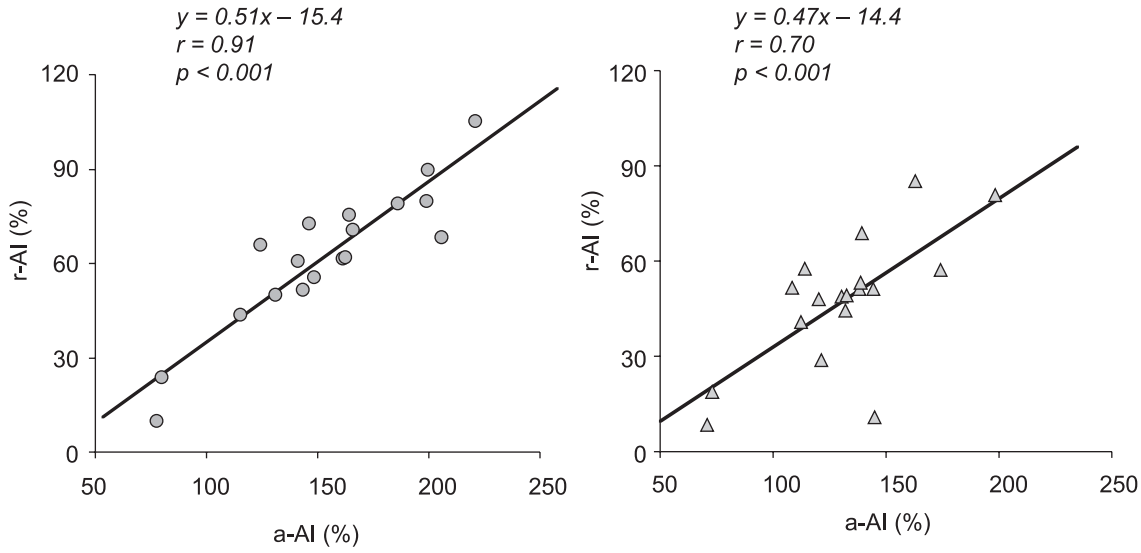


Fig. 6. *Correlation between aortic AI (a-AI) and radial AI (r-AI). Left: before nicorandil administration. Right: after nicorandil administration.*

caused by vasodilator effects.

In the large-scale ASCOT-CAFE study (*7*), conventional atenolol (β-blocker)-based therapy and contemporary amlodipine (calcium channel blocker)-based therapy were performed in patients with hypertension, and changes in brachial blood pressure, central aortic pressure, and other hemodynamic parameters were followed for 4 years. Decreased brachial blood pressure after 3 years was not significantly different between the 2 groups, but the incidence of cardiovascular events was significantly lower in the amlodipine-based therapy group. Central aortic pressure obtained by estimation of the central pulse wave from the radial artery was significantly lower in the amlodipine-based therapy group than in the atenolol-based therapy group, and this may explain the difference in the incidence of cardiovascular events. On analysis using the Cox proportional hazards model adjusted for age and baseline risk factors, central aortic pulse pressure was significantly correlated with a post-hoc–defined composite outcome of total cardiovascular events. In this study, we also focused on pulse pressure. As shown in Fig. 4 (left), Δa-PP and Δr-PP before and after nicorandil administration were significantly correlated ($r=0.59$, $p<0.01$). Since maximal systolic pressure in the aorta is proportional to late systolic pressure in the radial artery, the difference between r-SBP2 and DBP (r-PP2) was calculated and compared with central aortic pulse pressure (a-PP). As shown in Fig. 4 (right), the correlation coefficient between Δa-PP and Δr-PP2 was 0.92 ($p<0.001$) and much larger than that with Δr-PP. The slope of the correlation regression line was 0.83, close to 1. As for the relationship between Δa-PP and Δr-PP2, the correlation coefficient was higher, and the slope was larger and closer to 1 compared to those between Δr-PP and Δa-PP, suggesting that a-PP can be approximated by monitoring r-PP2.

Regarding the relationship with a-SBP, as shown in Fig. 5, the correlation coefficients between a-SBP and r-SBP were 0.88 (before nicorandil administration, $p<0.001$) and 0.85 (after administration, $p<0.001$), and those between a-SBP and r-SBP2 were 0.95 (before, $p<0.001$) and 0.93 (after, $p<0.001$). These results show a higher correlation with r-SBP2. The slopes of the correlation regression lines between a-SBP and r-SBP were 0.67 (before) and 0.80 (after). The slopes between a-SBP and r-SBP2 were 0.91 (before) and 1.06 (after), and thus both values were close to 1. On the Bland-Altman plot, the standard deviations of errors were 10.6 mmHg (before) and 10.1 mmHg (after) between a-SBP and r-SBP, and 7.1 mmHg (before) and 8.4 mmHg (after) between a-SBP and r-SBP2. The standard deviations in the latter relationship were smaller, suggesting that, for the estimation of a-SBP using r-SBP or r-SBP2, r-SBP2 may provide more accurate values with less variation. Smulyan *et al*. (*13*) reported that the correlation coefficient and standard deviation of the error between the measured central aortic pressure and aortic pressure estimated from the radial artery pulse wave using the transfer function of the SphygmoCor were 0.89 and 11.13 mmHg, respectively. Similarly, Hope *et al*. (*11*) reported that the correlation coefficient and standard deviation of the error between aortic pressure estimated from the radial artery using their transfer function and measured central aortic pressure were 0.94 and 7.3 mmHg, respectively. Although a simple comparison with these reports is not possible, partly because of the differences in the methods of aortic pressure measurement (in the former study: a micromanometer-tipped catheter; in the latter: a fluid-filled catheter), based on the comparison of correlation coefficients or standard deviations of the error, r-SBP2 may enable us to evaluate central aortic pressure as precisely as the transfer functions do.

There was a significant correlation between a-AI and r-AI (before nicorandil administration: $r=0.91$, $p<0.001$; after administration: $r=0.70$, $p<0.001$), as shown in Fig. 6. In previous studies, Takazawa *et al*. (*16*) reported that the correlation coefficient between a-AI using a catheter and r-AI using tonometry was 0.74. In comparison with their study, the correlation coefficient of 0.91 (before nicorandil administration) between the measured r-AI and a-AI in the current study was sufficiently high, and the radial artery may have reflected the central pulse wave information.

On the other hand, with regard to carotid AI, Chen *et al*. (*10*) reported that the correlation coefficient between the measured a-AI using a catheter and carotid AI using tonometry was 0.78. A high correlation between a-AI and carotid AI has been considered to demonstrate that carotid arterial pulse wave information reflects central aortic pulse wave information. The correlation coefficient of 0.91 (before nicorandil administration) between a-AI and r-AI in our study was higher than that reported by Chen *et al*. (*10*), suggesting that r-AI may well reflect central pulse wave information just as well as carotid AI. The report by Millasseau *et al*. (*14*), which discusses whether or not a transfer function is necessary and which suggests that similar information on central pressure wave reflection can be obtained directly from the radial pulse, supports our data.

As shown in Fig. 6, there were 2 subjects whose a-AI values were under 100%. In these subjects, a-SBP corresponded to the early systolic blood pressure in the aorta, and r-SBP2 corresponded not to a-SBP but to the late systolic blood pressure in the aorta. In this study, we generally included these 2 subjects in our investigation of the relationships between a-SBP and r-SBP2 ($n=18$), but we also investigated these relationships after excluding the 2 subjects ($n=16$), as described below. a-SBP decreased from 120 ± 18 to 104 ± 18 mmHg, and r-SBP decreased from 122 ± 16 to 112 ± 18 mmHg. The reduction in a-SBP (Δa-SBP) was -16 ± 15 mmHg, significantly larger than that in r-SBP (Δr-SBP) (-10 ± 12 mmHg, $p<0.05$). On the other hand r-SBP2 decreased from 108 ± 17 to 93 ± 20 mmHg and the reduction in r-SBP2 (Δr-SBP2) was -16 ± 14 mmHg, which was significantly larger than Δr-SBP ($p<0.005$), but not significantly different from Δa-SBP. The other relationships between Δa-SBP and Δr-SBP2 were as follows: the correlation coefficient was 0.92 ($p<0.001$); the slope of the correlation regression line was 0.87; and the mean error and standard deviation of the Bland-Altman plot were 0.2 ± 6.2 mmHg. The relationships between Δa-PP and Δr-PP2 were as follows: the correlation coefficient was 0.91 ($p<0.001$); the slope of the correlation regression line was 0.83; and the mean error and standard deviation of the Bland-Altman plot were 1.2 ± 3.9 mmHg. The relationships between a-SBP and r-SBP2 were as follows: the correlation coefficients were 0.92 (before nicorandil administration, $p<0.001$) and 0.92 (after administration, $p<0.001$); the slopes of the correlation regression lines were 0.86 (before) and 1.03 (after); and the mean error and standard deviation of the Bland-Altman plot were -11.7 ± 7.4 mmHg (before) and -11.6 ± 8.1 (after). These values obtained with the $n=16$ subjects were close to the values obtained with the $n=18$ subjects. So, in this study, the 2 subjects whose a-AI values were under 100% had no effect on the relationship between a-SBP and r-SBP2. However, in further investigations with more subjects, it should be clarified how common such subjects are and how effective the evaluation of a-SBP by r-SBP2 is when an evaluation method established for total subjects is applied to subjects whose a-AI values are under 100%.

Although there were large differences in the absolute values between a-SBP and r-SBP2, the correlation coefficients between them were high and the standard deviations of the differences were small both before and after nicorandil administration. Thus r-SBP2, compared with the ordinary measurement of brachial blood pressure, may facilitate a more accurate evaluation of a-SBP when using, for example, linear regression equations. Although the changes of linear regression equations by nicorandil administration were small in this study, further investigations may clarify cases in which the linear regression equations change remarkably. In such cases, the factors causing these changes should be identified, and should be considered when determining the equations for evaluation.

In this study, the maximal systolic blood pressure and diastolic blood pressure in the radial artery were corrected to the brachial systolic blood pressure and brachial diastolic blood pressure measured by an upper arm-cuff device based on the cuff oscillometric principle, respectively. Wrist-type devices based on the cuff oscillometric principle may be more appropriate for the correction, but they are not regarded as standard devices at present (*24*). Further investigations will be needed to clarify the best way to correct the tonometrically measured radial arterial blood pressure in order to evaluate the maximal systolic blood pressure in the aorta.

In conclusion, the observation of changes in late systolic blood pressure in the radial artery by measuring not only the ordinary brachial blood pressure but also the radial arterial pulse wave may enable a more accurate evaluation of changes in central aortic pressure during vasodilator therapy. Further investigations of central aortic pressure using late systolic blood pressure in the radial artery will be needed, including studies with a larger number of patients, a consideration of chronic and acute effects, and a prognostic cohort study.

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