

ORIGINAL ARTICLE

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Relationship between blood pressure obtained from the upper arm with a cuff-type sphygmomanometer and central blood pressure measured with a catheter-tipped micromanometer

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Abstract Recently, the importance of central blood pressure for cardiovascular risk stratification has been emphasized. Accordingly, the differences in peak systolic and bottom diastolic pressures between the ascending aorta and the brachial artery should be clarified. Study subjects consisted of 82 consecutive patients with suspected coronary artery disease who underwent cardiac catheterization, and in whom ascending aortic pressure waveform was obtained using a catheter-tipped micromanometer, and at the same time systolic and diastolic pressures were measured (single measurement) from the right upper arm with a cuff-type sphygmomanometer based on the oscillometric technique. No significant systematic difference (bias) was found between the peak pressure obtained in the ascending aorta and the systolic pressure from the right upper arm (133.6 ± 25.1 vs 131.8 ± 21.5 mmHg, not significant). Bland–Altman analysis showed only a small bias of +1.8 mmHg, and the limits of agreement were 25.4 mmHg and –21.8 mmHg. In contrast, the bottom pressure in the ascending aorta was significantly lower compared with the diastolic pressure from the upper arm (68.5 ± 10.7 vs 73.0 ± 12.4 mmHg, $P < 0.0001$). Bland–Altman analysis showed a small but significant bias of –4.5 mmHg, and the limits of agreement were 14.1 mmHg and –23.1 mmHg. The observed biases seemed to remain within practical range. However, random variation in the two measurements was rather large. This is considered to be caused by the random error in the single measurement with the cuff-type sphygmomanometer.

Key words Augmentation index · Brachial pressure · Central pressure · Coronary artery disease

Introduction

Recently, the importance of central blood pressure evaluation for cardiovascular risk stratification has been emphasized.¹ There are previous studies which show that central (aortic) peak systolic pressure is lower than brachial systolic pressure, while mean and bottom diastolic pressures are generally constant across different sites of the arterial tree, bringing lower pulse pressure in the center than in the periphery.^{2–4}

Several investigators have reported that the central pressure waveform can be precisely generated from the radial pressure waveform obtained by applanation tonometry with the use of general transfer function.^{5–7} However, in the clinical setting, radial pressure waveforms are calibrated by noninvasively obtained brachial systolic and diastolic pressures. It is reported that significant underestimation of central peak and pulse pressures is caused by this procedure,^{8–11} and hence brachial systolic and diastolic pressures measured with a cuff-type sphygmomanometer may give better estimates of central peak and bottom pressures.^{8–10} Accordingly, we assessed the relationship between blood pressure in the upper arm obtained with a cuff-type sphygmomanometer and central blood pressure with a catheter-tipped micromanometer in patients who underwent cardiac catheterization for the evaluation of coronary artery disease (CAD), and in whom precise blood pressure measurements were required for cardiovascular risk management.

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Patients and methods

Study subjects consisted of 82 consecutive patients (65 men and 17 women; age 64.3 ± 9.4 years) with suspected CAD who underwent cardiac catheterization. Patients with an acute coronary syndrome, primary valvular heart disease, or

atrial fibrillation were excluded from the study. According to the findings of cardiac catheterization, 69 patients had CAD, 46 patients with prior myocardial infarction and 23 without prior myocardial infarction. The remaining 13 patients had neither significant coronary stenosis nor left ventricular wall motion abnormality but had atypical chest pain. In all patients, pulsation of the right brachial artery was overt at their antecubital fossae. All studies were performed while patients were receiving cardiac and/or antihypertensive medications. All subjects gave written informed consent to participate in the study, and the study was performed according to the regulations proposed by the Ethical Guidelines Committee of the Nagoya City University Graduate School of Medical Sciences.

Blood pressure measurements

During diagnostic cardiac catheterization, before contrast material was injected into the left ventricle or coronary artery, aortic pressure waveforms were obtained using a catheter-tipped micromanometer (SPC-454D, Millar Instrument, Houston, TX, USA) at approximately 10 cm above the aortic valve in the ascending aorta and recorded on a polygraph system (RMC-2000, Nihon Kohden, Tokyo, Japan). The micromanometer was electrically calibrated at the two pressure levels, i.e., 0 and 100 mmHg, using a control unit (TC-510, Millar Instrument). The frequency response of the micromanometer used was flat to 10 kHz. Before the catheter-tipped micromanometer inserted into the aorta, the pressure zero was adjusted electrically just beneath the silent surface of saline. The offset of the pressure waveform was again adjusted to that obtained with a fluid-filled system at the bottom diastolic pressure level when it was in the ascending aorta. The details of our pressure measurement during cardiac catheterization were reported elsewhere.¹² Patients kept breathing naturally during the measurement. From the recorded aortic pressure waveforms, peak and bottom pressures in the ascending aorta were measured during five cardiac cycles and the average values were used for statistical analysis. We also calculated the augmentation index of waveform, as shown in Fig. 1.¹³

While obtaining ascending aortic pressure waveforms, blood pressure in the right upper arm was simultaneously measured with a validated automated cuff-type sphygmomanometer (BP-8800, Omron Colin, Tokyo, Japan), which uses the oscillometric method. The measurement of upper arm pressure was made only once while obtaining aortic pressure.

Statistical analysis

Data are presented as means \pm SD. Differences between pressures obtained with the catheter-tipped micromanometer and with the automated sphygmomanometer were tested using the paired Student *t*-test. Bland–Altman analysis¹⁴ was applied to the evaluation of agreement between the two methods of pressure measurement. Differences with *P* values less than 0.05 were considered significant.

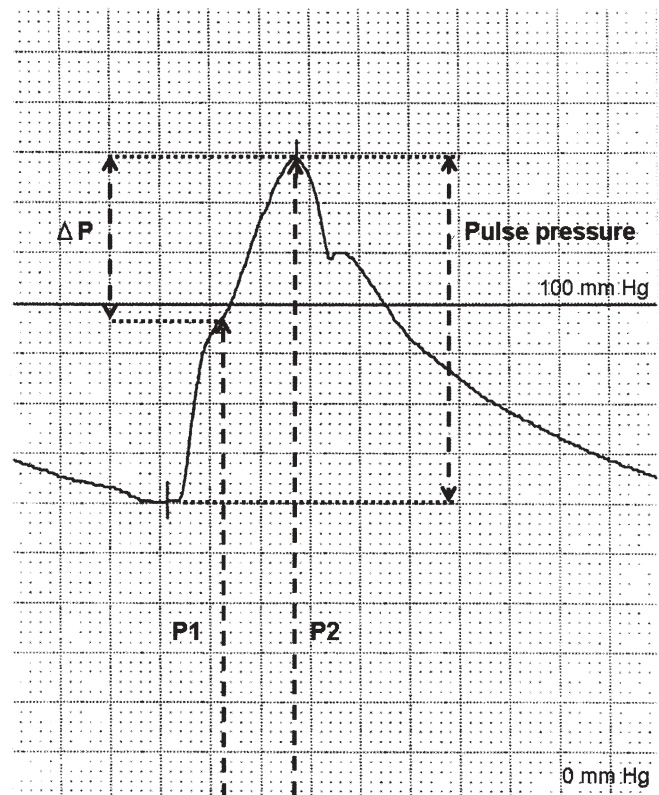


Fig. 1. Representation of pressure waveform obtained in the ascending aorta using a catheter-tipped micromanometer. The augmentation index was calculated as the difference between P2 and P1 (ΔP), expressed as a percentage of the pulse pressure

Results

No significant difference was found between the peak pressure obtained in the ascending aorta and the systolic pressure obtained from the right upper arm (133.6 ± 25.1 vs 131.8 ± 21.5 mmHg, not significant). The relationship between the two parameters was expressed as the following equation: $Y = 1.03X - 2.58$ ($r = 0.88$, $P < 0.0001$) (X = systolic pressure from the right upper arm in mmHg; Y = peak pressure in the ascending aorta in mmHg). Bland–Altman analysis showed only a small bias of +1.8 mmHg, and the limits of agreement were 25.4 mmHg and -21.8 mmHg (Fig. 2). In contrast, the bottom pressure in the ascending aorta was significantly lower compared with the diastolic pressure from the right upper arm (68.5 ± 10.7 vs 73.0 ± 12.4 mmHg, $P < 0.0001$). The regression line of the bottom pressure in the ascending aorta (Y mmHg) on the diastolic pressure from the right upper arm (X mmHg) was $Y = 0.59X + 25.4$ ($r = 0.69$, $P < 0.0001$). Bland–Altman analysis showed a small but significant bias of -4.5 mmHg, and the limits of agreement were 14.1 mmHg and -23.1 mmHg (Fig. 3). The pulse pressure in the ascending aorta was significantly greater compared with that from the right upper arm (65.1 ± 21.2 vs 58.9 ± 17.2 mmHg, $P < 0.0001$). The regression of the pulse pressure in the ascending aorta (Y mmHg) against the pulse pressure from the right upper arm (X mmHg) was $Y = 1.02X + 5.12$ ($r = 0.83$, $P < 0.0001$). Bland–Altman analy-

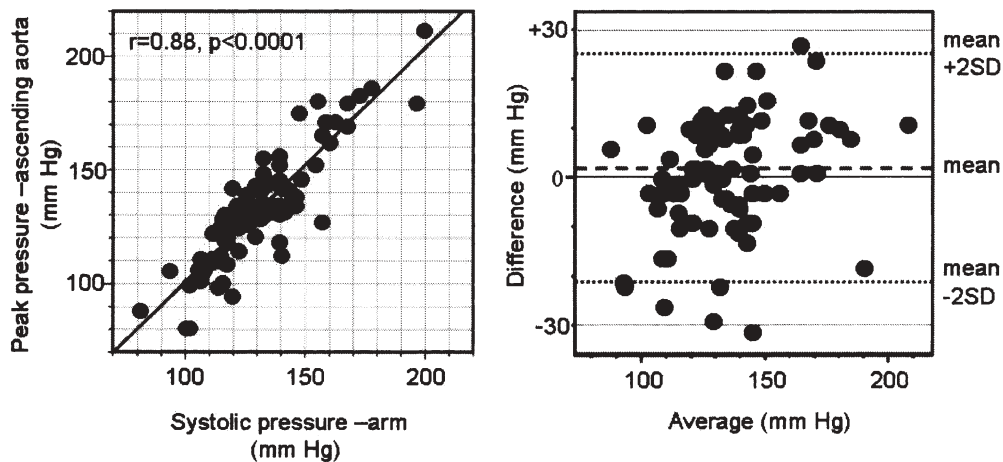


Fig. 2. **Left** Relationship between the peak systolic pressures measured at the ascending aorta with a catheter-tipped manometer and from the right upper arm with an oscillometric sphygmomanometer. A significant and close correlation was observed. **Right** Bland–Altman plot for peak systolic pressures measured by the both sites. The analysis showed

only a small bias of +1.8 mmHg, and the limits of agreement were 25.4 mmHg and –21.8 mmHg.

Difference = peak aortic pressure – brachial systolic pressure
Average = (peak aortic pressure – brachial systolic pressure)/2

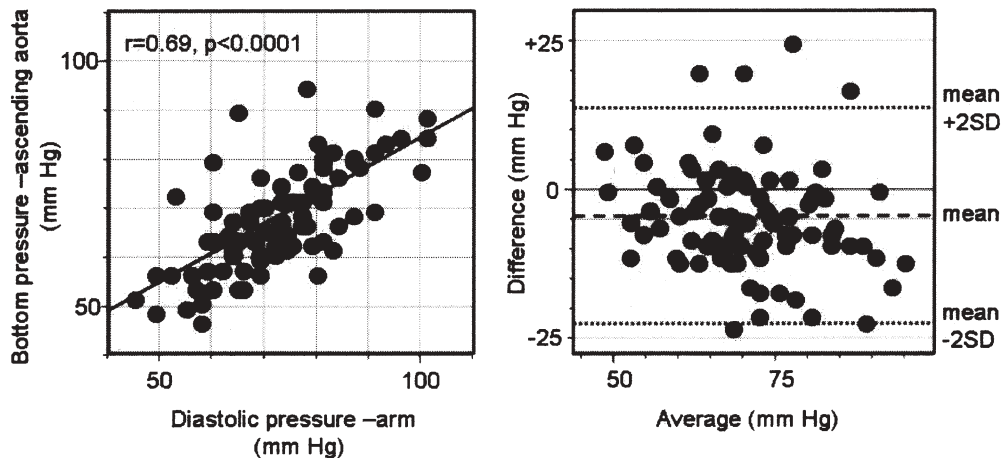


Fig. 3. **Left** Relationship between bottom diastolic pressures measured at the both sites. A significant but relatively weak correlation was found. **Right** Bland–Altman plot of bottom diastolic pressures at the both sites. The analysis showed a small but significant bias of –4.5 mmHg, and the limits of agreement were 14.1 mmHg and –23.1 mmHg.

Difference = bottom aortic pressure – brachial diastolic pressure
Average = (bottom aortic pressure – brachial diastolic pressure)/2

sis also showed a small but significant bias of +6.3 mmHg, and the limits of agreement were 29.1 mmHg and –16.5 mmHg (Fig. 4).

The augmentation index obtained in the ascending aorta was significantly correlated with aortic peak pressure ($r = 0.64$, $P < 0.0001$) and also with aortic pulse pressure ($r = 0.71$, $P < 0.0001$), indicating that late systolic peaking caused by pulse wave reflection from the lower body affected the both pressures (Fig. 5).

Discussion

The oscillometric method is based on detecting the oscillations on the lateral walls of the occluded artery during

deflation of the cuff. The oscillations begin at approximately the level of systolic pressure and reach their maximum amplitude at the level of mean arterial pressure. Systolic pressure measurement by this method is accurate, but diastolic pressure, which is derived from the systolic and mean pressures, may not be accurate.¹⁵ Each company has its own secretive algorithm for calculating diastolic blood pressure. Generally speaking, oscillometric devices are designed to give the same systolic and diastolic pressures as the auscultatory technique using Korotkoff sounds. The auscultatory method tends to give diastolic pressures that are slightly higher than direct intra-arterial measurements.^{16,17} Since the bottom diastolic pressures in the aorta and in the brachial artery are nearly equal, the difference between the former obtained by direct intra-aortic measurements and the latter obtained by oscillometric measurements shows that the

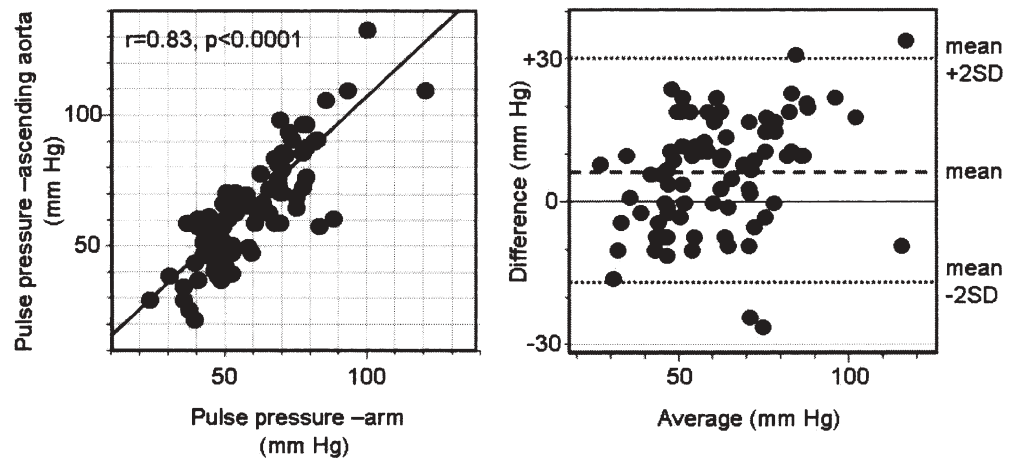
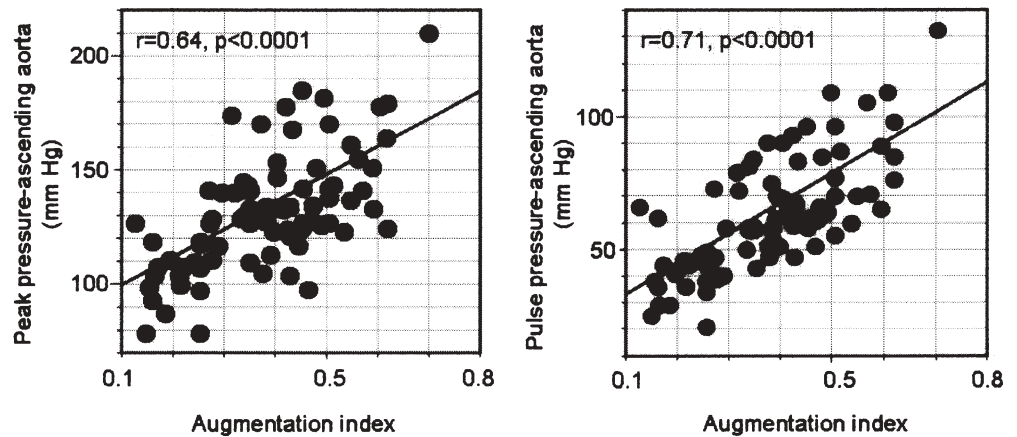


Fig. 4. **Left** Relationship between pulse pressures measured at the both sites. A significant and close correlation was observed. **Right** Bland–Altman plot of pulse pressures at the both sites. The analysis showed a relatively small but significant bias of +6.3mmHg, and the limits of agreement were 29.1 mmHg and –16.5 mmHg.

Difference = aortic pulse pressure – brachial pulse pressure
Average = (aortic pulse pressure – brachial pulse pressure)/2

Fig. 5. Relationships between augmentation index and aortic peak systolic and pulse pressures. Significant positive correlations were found between the parameters



oscillometric device which we used had the above-mentioned tendency (bias).

Even if there is no difference in peak systolic pressure between the direct intra-arterial measurement and the non-invasive oscillometric measurement, the peak systolic pressure in the ascending aorta and that obtained from the upper arm can differ because of the amplification of the pressure pulse with transmission. However, the present study demonstrates that there was no significant systematic difference (bias) between the peak pressure measured in the ascending aorta and the systolic pressure obtained from the right upper arm in our study group of patients with suspected coronary artery disease. It is reported that amplification is greatest when pulse wave velocity is low and ejection duration short, and where wave reflection has the greatest positive effect on the peripheral pressure wave and the least positive effect on the central pressure wave.¹⁸ In relatively younger subjects, reflected waves return from the lower body to the ascending aorta in diastole because of low pulse wave velocity in the arterial tree without arteriosclerosis. In such a case, augmentation of aortic systolic pressure

does not occur. On the other hand, in the elderly, reflected waves return as early as late systole because of higher pulse wave velocity in the stiffer arterial tree, which augments late systolic pressure (late systolic peaking). Therefore, amplification with transmission to the lower body (ratio of peak peripheral pressure to peak aortic pressure) is greater for younger subjects. The process of formation of the peak of pressure wave in the upper arm is different from that in the aorta. The peak is formed due to summation of reflected waves returning from the upper limb (mainly from the hand). Since reflection sites in the upper arm are anatomically closer, the timing of the formation of pressure peak is considerably earlier than that in the aorta. In the upper arm, the peak appears in very early systole. In contrast to pulse wave transmission along the aorta to and from the lower body, aging has little effect on pulse wave velocity in the upper limb.¹⁸ Therefore, aging has little effect on the timing of the formation of pressure peak in the upper arm. In young adults, this peak in the upper arm is considerably higher than the aortic pressure peak. Therefore, amplification of pressure in the upper arm in young adults is consid-

erably large. However, in the elderly, augmentation of aortic pressure caused by early return of reflected waves from the lower body becomes prominent, which increases aortic peak pressure (late systolic peaking) (Fig. 5), and eventually makes the difference between aortic peak pressure and upper arm peak pressure less significant.

Our study group was a particular group of patients with suspected coronary disease aged 64.3 ± 9.4 years, and showed prominent augmentation. Such a group is typical of those in which pressure waves are less amplified. Indeed, there was no significant systematic difference between the peak pressure obtained in the ascending aorta and the systolic pressure obtained from the right upper arm.

Although pressure measurements were made at two different sites in the present study, an assessment according to the USA Association for the Advancement of Medical Instrumentation (AAMI) SP10 criteria¹⁹ for arterial pressure measurements with different methods at the same location may provide a standard for the quality of pressure measurement. The AAMI SP10 criteria for acceptability of blood pressure measurement devices requires that blood pressures measured with an automated sphygmomanometer should achieve a mean difference of ± 5 mmHg and SD of ± 8 mmHg (2SD = ± 16 mmHg) against a reference standard. In our study, the mean differences in peak systolic and bottom diastolic pressures between the ascending aorta and the right upper arm remained less than ± 5 mmHg. However, the standard deviations of the differences between paired measurements exceeded the above criteria. Indeed, the limits of agreement for both systolic and diastolic pressure measurements would not be acceptable for clinical purposes.

The relatively large standard deviations of the differences between paired measurements of aortic pressure and upper arm pressure are considered to be caused by the poor reproducibility of the single measurement from the upper arm using a cuff-type sphygmomanometer.^{11,20} Rushing procedure of invasive measurement of ascending aortic pressure did not allow us to measure systolic and diastolic pressures from the upper arm more than one time in each patient. In any case, single measurement of blood pressure by sphygmomanometer causes a large error.¹⁷ In addition, the upper arm pressure measurements were made in unusual circumstances in the catheter laboratory. This may also have brought such relatively large random variations. However, if we made measurements from the upper arm by sphygmomanometer as many times as practically possible to obtain the average values, the peak and bottom pressures in the ascending aorta could be replaced by the systolic and diastolic pressures obtained from the upper arm in a study group with less pressure amplification.

Several investigators reported that ascending aortic pressure waveform could be precisely generated from radial pressure waveform, obtained using applanation tonometry with the use of general transfer function.⁵⁻⁷ In the generation of ascending aortic pressure waveform using the transfer function, radial pressure waveform should be calibrated using systolic and diastolic pressures in the radial artery. If the pressure waveform is calibrated by pressures invasively

obtained in the radial artery, aortic systolic and diastolic pressures may be precisely estimated using the transfer function.⁵⁻⁷ However, in the clinical setting, systolic and diastolic pressures in the radial artery are replaced by those in the brachial artery measured using a cuff-type sphygmomanometer.⁵⁻⁷ Smulyan et al.¹¹ reported that an inaccuracy of the oscillometric cuff method for measuring pressures from the upper arm appeared to be a limiting factor for the use of applanation tonometry on the radial artery. O'Rourke and Adji²⁰ also addressed that there was a debate on the biases of accuracy of the cuff-type sphygmomanometer to describe systolic and diastolic brachial pressures. Furthermore, several investigators reported that an amplification of systolic pressure between the brachial artery and the radial artery exists, which causes underestimation of central pressures when the tonometry method calibrated by cuff-derived brachial artery pressures is applied.^{21,22}

Davies et al.^{8,9} demonstrated that central pressure waveform generated using applanation tonometry from the radial artery with the transfer function and calibration using noninvasively obtained brachial pressures significantly underestimated systolic and overestimated diastolic blood pressures compared with invasively measured pressures in the ascending aorta. They also reported important findings showing that peripheral systolic blood pressure measurements using a cuff-type automated sphygmomanometer from the left upper arm overestimated invasive measurements of systolic blood pressure in the ascending aorta, but only by a nonsignificant value of 3.36 ± 10.47 mmHg, and peripheral blood pressure measurements significantly overestimated diastolic blood pressure measured invasively, with a mean of 11.7 ± 7.18 mmHg; they concluded that peripheral blood pressure gave a better estimate of aortic peak pressure than did the tonometry method. Cloud et al.¹⁰ also reported a similar finding. They presented the data that noninvasive brachial systolic pressure underestimated the catheter-measured peak aortic pressure by only 1.9 mmHg and brachial diastolic pressure overestimated the bottom aortic pressure by 11.5 mmHg. Our results are compatible with their results, although the range of overestimation of bottom aortic pressure was relatively smaller in our study. The difference between their studies and ours is considered to be caused by the difference in the algorithm for deriving diastolic pressure.

The present study may provide comprehensive understanding of central blood pressure and arterial wave reflection physiology in the arterial tree. This may contribute to precise evaluation of the effect of antiatherosclerotic therapy, considering the influence of blood pressure on arterial stiffness and intima-media thickening.^{23,24}

Conclusions

These findings indicated that peak systolic and bottom diastolic pressures in the ascending aorta can be directly estimated from systolic and diastolic pressures measured from the upper arm using an automated cuff-type sphygmoma-

nometer in an elderly study group. The observed biases seem to remain within practical range. However, random variation in the single measurement of upper arm pressure was large and unacceptable for clinical purposes. Measurement by sphygmomanometer should be repeated as many times as practically possible.

References

- Williams B, Lacy PS, Thom SM, Cruickshank K, Stanton A, Collier D, Hughes AD, Thurston H, O'Rourke M; CAFE Investigators; Anglo-Scandinavian Cardiac Outcomes Trial Investigators; CAFE Steering Committee and Writing Committee (2006) Differential impact of blood pressure-lowering drugs on central aortic pressure and clinical outcomes: principal results of the Conduit Artery Function Evaluation (CAFE) study. *Circulation* 113:1213–1225
- Rowell LB, Brengelmann GL, Blackmon JR, Bruce RA, Murray JA (1968) Disparities between aortic and peripheral pulse pressure induced by upright exercise and vasomotor changes in man. *Circulation* 37:954–964
- Pauca AL, Wallenhaupt SL, Kon ND, Tucker WY (1992) Does radial artery pressure accurately reflect aortic pressure? *Chest* 102:1193–1198
- Kelly R, Fitchett (1992) Noninvasive determination of aortic input impedance and external left ventricular power output: a validation and reproducibility study of a new technique. *J Am Coll Cardiol* 20:952–963
- Chen CH, Nevo E, Fetcs B, Pak PH, Yin FCP, Maughan WL, Kass DA (1997) Estimation of central aortic pressure waveform by mathematical transformation of radial tonometry pressure. Validation of generalized transfer function. *Circulation* 95:1827–1836
- Pauca AL, O'Rourke MF, Kon ND (2001) Prospective evaluation of a method for estimating ascending aortic pressure from the radial artery pressure waveform. *Hypertension* 38:932–937
- Adji A, O'Rourke MF (2004) Determination of central aortic systolic and pulse pressure from the radial artery pressure waveform. *Blood Pressure Monitor* 9:115–121
- Davies JI, Band MM, Pringle S, Ogston S, Struthers AD (2003) Peripheral blood pressure measurement is as good as applanation tonometry at predicting aortic blood pressure. *J Hypertens* 21:571–576
- Davies J, Struthers A (2003) Assessment of central arterial pressure? Authors' reply. *J Hypertens* 21:4126
- Cloud GC, Rajkumar C, Kooner J, Cooke J, Bulpitt CJ (2003) Estimation of central aortic pressure by SphygmoCor[®] requires intra-arterial peripheral pressures. *Clin Sci* 105:219–225
- Smulyan H, Siddiqui DS, Carlson RJ, London GM, Safar ME (2003) Clinical utility of aortic pulses and pressures calculated from applanated radial-artery pulses. *Hypertension* 42:150–155
- Ohte N, Narita H, Hashimoto T, Akita S, Kurokawa K, Fujinami T (1998) Evaluation of left ventricular early diastolic performance by color tissue Doppler imaging of the mitral annulus. *Am J Cardiol* 82:1414–1417
- O'Rourke MF, Kelly PP (1993) Wave reflection in the systemic circulation and its implications in ventricular function. *J Hypertens* 11:327–337
- Bland JM, Altman DG (1986) Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1:307–310
- Perloff D, Grim C, Flack J, Frohlich ED, Hill M, McDonald M, Morgenstern BZ (1993) Human blood pressure determination by sphygmomanometry. *Circulation* 88:2460–2470
- Jones DW, Appel LJ, Sheps SG, Roccella E, Lenfant C (2003) Measuring blood pressure accurately: new and persistent challenges. *JAMA* 289:1027–1030
- Goh R, Saito T, Arase T, Higuchi S (1988) A comparison of intra-arterial, oscillometric and auscultatory measurements of blood pressure – influence of blood pressure level and arteriosclerosis (in Japanese). *Anesthesia (Masui)* 37:189–196
- Nichols WW, O'Rourke MF (2005) McDonald's blood flow in arteries, 5th edn. Hodder Arnold, Oxon, UK, pp 360–363
- White WB, Berson AS, Robbins C, Jamieson MJ, Prisant M, Roccella E, Sheps SG (1993) National standard for measurement of resting and ambulatory blood pressures with automated sphygmomanometers. *Hypertension* 21:504–509
- O'Rourke MF, Adji A (2004) An updated clinical primer on large artery mechanics: implications of pulse waveform analysis and arterial tonometry. *Curr Opin Cardiol* 20:275–281
- Nichols WW, O'Rourke MF (2005) McDonald's blood flow in arteries, 5th edn. Hodder Arnold, Oxon, UK, pp 178–180
- Verbeke F, Segers P, Heireman S, Vanholder R, Verdonck P, Van Bortel LM (2005) Noninvasive assessment of local pulse pressure. Importance of brachial-to-radial pressure amplification. *Hypertension* 46:244–248
- Matsuo T, Iwade K, Hirata N, Yamashita M, Ikegami H, Tanaka N, Aosaki M, Kasanuki H (2005) Improvement of arterial stiffness by the antioxidant and anti-inflammatory effects of short-term statin therapy in patients with hypercholesterolemia. *Hear Vessels* 20:8–12
- Sugiyama M, Ohashi M, Takase H, Sato K, Ueda R, Dohi Y (2005) Effects of atorvastatin on inflammation and oxidative stress. *Heart Vessels* 20:133–136