Eur J Vasc Endovasc Surg **22**, 355–360 (2001)
doi:10.1053/ejvs.2001.1465, available online at http://www.idealibrary.com on IDEAL®

Comparison of Brachial Artery Pressure and Derived Central Pressure in the Measurement of Abdominal Aortic Aneurysm Distensibility

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Objective: *AAA distensibility (Ep, β) may predict growth and risk of rupture. However, distensibility measurements based on brachial rather than central pressure may be inaccurate. Our aim was to compare AAA distensibility using non-invasive brachial and derived central aortic pressure.*

Design: *brachial and central pressures were measured prospectively by automated sphygmomanometry (Omron) and pulse wave analysis (SphygmoCor) respectively. AAA distensibility was calculated using brachial (Ep^b, β^b) and central (Ep^c, β^c) pressures by ultrasonic echo-tracking (Diamove). Twenty-eight patients (18 males) were selected on a first come basis from a larger study of AAA patients. There were no exclusion criteria, so 54% had cardiac dysfunction (MI, angina) and 14% were hypertensive (BP>140/90 mmHg).*

Results: *median (IQR) age was 74 (70–77) years, median AAA (IQR) diameter was 44 (40–51) mm. Central and brachial systolic pressures were significantly different, [140 (121–153) vs 144 (130–164) mmHg respectively, $p \leq 0.01$]. Central and brachial diastolic pressures were not significantly different [76 (72–86) vs 76 (71–86) mmHg respectively, $p = 0.5$]. Ep^c (3.0, [2.2–4.9]) and β^c (22.2 [15.5–33.2]) were significantly lower than Ep^b (3.6, [2.4–5.1] $10^5 Nm^{-2}$) and β^b (24.7 [17.1–33.0] a.u., all $p<0.001$. Brachial and central derived distensibility remained significantly different after adjusting for age and diameter ($p<0.001$).*

Conclusion: *the use of brachial pressure leads to a small, systematic overestimate of Ep (18%) and β (11%) independent of age and AAA diameter. This systematic error will not bias follow-up of changes in distensibility.*

Key Words: Abdominal aortic aneurysm; Blood pressure; Distensibility.

Introduction

The decision to operate on a patient with an asymptomatic abdominal aortic aneurysm (AAA) involves weighing the risks of rupture against those of operative repair. Although cohort studies indicate that rupture is related to maximum AAA diameter (Dmax), growth rate and blood pressure (BP), none of these variables reliably predict the behaviour of individual aneurysms.[1] As no AAA is entirely free from risk of rupture, a variable that provides a more precise quantification of risk is required.

Previous work has suggested that, in addition to maximal diameter, AAA wall distensibility, expressed as pressure-strain elastic modulus (Ep) and stiffness (β), measured by means of a commercially available ultrasound echo-tracking system (Diamove), may be related to future growth rate and risk of rupture.[2] We[2] have previously shown that when AAA diameter and aortic stiffness increase concomitantly (decreasing distensibility), aneurysm rupture is less likely than when AAA diameter increases but stiffness decreases (increasing distensibility). We[3] also reported that AAA wall distensibility might indicate matrix degeneration in terms of collagen and elastin integrity, since distensibility decreases with elastin degeneration and collagen deposition, but in the final stages of collagen breakdown distensibility increases.[3] These findings suggest that serial simultaneous measurement of diameter and distensibility might provide a better understanding of the degeneration occurring in the aortic wall matrix than simply assessing diameter. They also suggest that the absolute value of AAA distensibility is less important than its change over time.

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1078–5884/01/100355+06 $35.00/0

Arterial wall compliance describes the change in volume of a segment of artery, in relation to pulsatile change in BP.[4] However, measurement of change in wall thickness in response to change in pressure and vessel volume is necessary to calculate true vessel compliance.[4] At present, neither variable can be reliably measured in the aorta *in vivo*. Arterial wall distensibility, which describes the relationship between relative diameter change and pressure, has been used by a number of workers[5–7] as a "surrogate" measure of compliance.

Ep[4] is a measure of the structural distensibility of the artery, rather than a measure of the elasticity of the arterial wall material,[7] where:

$$\text{Ep} = \text{K (P systolic} - \text{P diastolic)/}$$
$$\text{[(D systolic} - \text{D diastolic)/D diastolic]}$$
$$\text{and K} = 133.3, \text{ P} = \text{pressure and D} = \text{aortic diameter.}$$

Stiffness index (β)[8] also describes the visco-elastic behaviour of arteries within the physiological pressure range, where:

$$\beta = \ln(\text{P systolic/P diastolic)/}$$
$$\text{[(D systolic} - \text{D diastolic)/D diastolic]}$$

β is less pressure dependent than Ep,[8] both are inversely related to distensibility and compliance. These concepts are discussed more fully in two reviews.[9,10] Both can be measured using ultrasonic echo-tracking equipment described in the Methods section.

Poiseuille's law describes the flow of fluids and shows that blood "flow is directly proportional to the difference between inflow (aortic) and outflow (peripheral) pressures".[11] Further studies have shown that systolic pressure increases along the arterial tree from the aorta to the peripheral arteries by 10–35 mmHg[12–14] due to differences in vessel stiffness and wave reflections. In contrast diastolic pressure and median arterial pressure (MAP) fall only slightly – which provides the pressure gradient for forward flow of blood along a pressure gradient.[12–14] The net result is an increase in pulse pressure peripherally.[12–14]

The aim of this study, therefore, was to compare AAA distensibility (Ep and β) calculated using brachial BP with that calculated from derived central BP (estimated by pulse wave analysis) using two non-invasive methods of BP measurement, as would be the case in a clinical setting. Thus any possible underestimation of BP due to sphygmomanometry technique would be constant between the techniques.

Methods

Central blood pressure can now be assessed non-invasively using pulse wave analysis (PWA). PWA allows accurate recording of peripheral arterial pressure waveforms, and construction of the corresponding central pressure waveform and augmentation index. The technique uses applanation tonometry, which is based on the principle that when opposing curved surfaces of a vessel are flattened until parallel with each other, circumferential pressures are equalised. In other words, when an arterial wall is flattened (applanated) by the tip of the tonometer, the contact pressure between the transducer and the wall equals the intra-arterial pressure. This technique can be accurately applied to peripheral arteries such as the radial or the carotid, and can also be used on the femoral artery to derive aortic pulse wave velocity. The peripheral waveform is recorded and transformed into the corresponding central waveform using an integral transfer function, which has previously been validated using invasive recordings.[15–17] Both waveforms can then be analysed and a number of variables measured including central systolic, diastolic, mean arterial and pulse pressures.

Twenty-eight subjects (18 male) were studied. These subjects had known AAA and were recruited on a "first-come" basis from a larger prospective study investigating AAA distensibility and rupture. In order to truly replicate the normal clinical setting there were no exclusion criteria and, as a result, 54% of these patients had some cardiac dysfunction (angina or MI). Only 14% had hypertension according to the British Hypertension Society guidelines (pressure >140/90 mmHg).[18]

PWA was used to determine central pressure non-invasively (Sphygmocor, SCOR; PWV Medical, Sydney, Australia).[19] Pressure waveforms were recorded from the radial artery using a high fidelity micromanometer (SPC-301, Millar Instruments, Texas, U.S.A.) and fed directly into a portable microcomputer.[20] The integral system software allowed online recording of the radial waveform and, after 20 sequential waveforms were collected, an averaged peripheral and corresponding central waveform was generated. Central aortic pressure was then calculated from the waveform[21] using a validated transfer function.[15–17,20] To evaluate the quality of the recorded wave, the software calculates two parameters of the wave variability allowing the observer to accept the waveform according to pre-stated levels of acceptable variability; namely wave amplitude >100 mV, standard deviation of systolic and diastolic peak <5%. The validation and reproducibility of the SphygmoCor tech-

nique in pulse wave analysis and the measurement of central pressure has been discussed previously and found to be acceptable.[20,22] There was a short time delay between tonometric and brachial pressure measurements; however, both were carried out alternately first or second to avoid a time-dependent bias.

BP was measured from the brachial artery in the right arm using an oscillometric sphygmomanometer (model 711, Omron, Japan); and phase locked loop echo-tracking (Diamove, Teltec, Sweden) was used to measure aortic Ep and β. The echo-tracking ultrasound system has been described in detail previously.[9,10] Briefly, a 3.5 MHz linear array transducer was used to provide a standard real time longitudinal B-scan image of the AAA at the point of maximal antero-posterior (AP) diameter. The vessel walls were tracked after initial placement of a cursor within the vessel. A phase-locked loop restored the position of an electronic gate relative to the moving echo while the compensatory movement of the gate yielded the movement of the echo.

Data acquisition and analysis were carried out on a Pentium computer (DCS, Edinburgh). The pressure-diameter curve was registered on the computer in real time and at least three consecutive waves were analysed. The Diamove software automatically identified the start and end of each cardiac cycle. The operator manually selected the waveforms of interest and an average wave was produced. Brachial artery pressures were entered and the calculated variables, including Ep and β, were then displayed on the screen. Distensibility calculated using derived central pressure is referred to as Ep^c and β^c, whereas distensibility calculated using brachial pressure is referred to as Ep^b and β^b.

Statistical analysis was carried out using SPSS Base 8.0.[23] The data were skewed so median and interquartile ranges (IQR) were calculated. Spearman's rank correlation was used to examine the correlation between brachial and central variables, Wilcoxon signed rank test was used to evaluate the differences between central and peripheral derived variables. In order to examine whether age and diameter confounded the observed relationships, the data were first logarithmically transformed to normality. Linear regression was then used to calculate predicted (log) distensibility adjusted for the effect of age and diameter.

Results

The mean (range) age of the subjects was 74 (63–84) years and the median (interquartile range (IQR)] AP diameter was 44 (40–51) mm. The median (IQR) brachial pressures were systolic 144 (130–164) mmHg, diastolic 76 (71–86) mmHg, and the median (IQR) central pressures were systolic 140 (121–153) mmHg, diastolic 76 (72–86) mmHg. The amplification ratio (peripheral PP:central PP) was 1.1.

There was a significant positive correlation between the central and brachial pressures (Table 1). Derived central systolic pressure, pulse pressure and MAP were significantly higher than the brachial equivalents (Table 1). There were no differences with regard to diastolic pressure ($p = 0.5$).

There was a significant correlation between Ep^c and brachial Ep^b ($r = 0.90$, $p \leq 0.001$) (Fig. 1) and between β^c and β^b ($r = 0.90$, $p \leq 0.001$) (Fig. 2). However, the Wilcoxon signed rank test showed that median Ep and β were significantly higher ($p \leq 0.01$) when using brachial pressure rather than central pressure: [Ep^b 3.6 (2.4–5.1) vs Ep^c 3.0 (2.2–4.9) $10^5 Nm^{-2}$, $p \leq 0.001$]; [β^b 24.7 (17.1–33.0) vs β^c 22.2 (15.5–33.2) a.u, $p \leq 0.01$].

In order to examine whether the difference in distensibility derived from central and brachial pressures were confounded by age or AAA diameter, predicted log values for central and brachial-derived distensibility adjusted for age and diameter were calculated and compared. The differences between distensibility calculated using brachial and derived central pressures remained significant (both $p \leq 0.001$). Median (IQR) Ep^b predicted from brachial pressure was 1.22 (1.08–1.45) $10^5 Nm^{-2}$, and Ep^c predicted from central pressure was 1.11 (0.94–1.35) $10^5 Nm^{-2}$. Similarly, β^b predicted from brachial pressure was 3.18 (3.05–3.37), and β^c predicted from central pressure was 3.07 (2.94–3.32).

Discussion

This study compares, for the first time, the use of non-invasive brachial artery pressure and derived central aortic pressure in the measurement of AAA distensibility.

Previous work using invasive intra-aortic pressure measurement and non-invasive assessment of aortic distensibility (ultrasonic echo-tracking) suggested that using peripheral blood pressure to calculate distensibility underestimates Ep and β by 25–30%.[24,25] However, in one of these studies[24] systolic pressure was the same or lower in the brachial artery than in the aorta, diastolic pressure higher in the brachial artery, and consequently pulse pressure was lower in the brachial artery. In the second study,[25] systolic and diastolic pressures were higher in the brachial artery

Table 1. Wilcoxon signed rank test comparing brachial and central pressures and pressure-strain elastic modulus (Ep) and stiffness (β) derived from brachial and central pressures.

	Brachial Median (IQR)	Central Median (IQR)	% Differences	Significance (Two tailed)
Systolic (mmHg)	144 (130–164)	140 (121–153)	+ 3	0.001
Diastolic (mmHg)	76 (71–86)	76 (72–85)	0	0.5
Pulse pressure (mmHg)	65 (50–79)	60 (44–75)	+ 8	0.001
Mean arterial pressure (mmHg)	100 (89–110)	99 (89–106)	+ 1	0.003
Ep ($10^5 Nm^{-2}$)	3.6 (2.4–5.1)	3.0 (2.2–4.9)	+18	0.001
β (a.u.)	24.7 (17.4–33.0)	22.2 (15.5–33.2)	+11	0.010

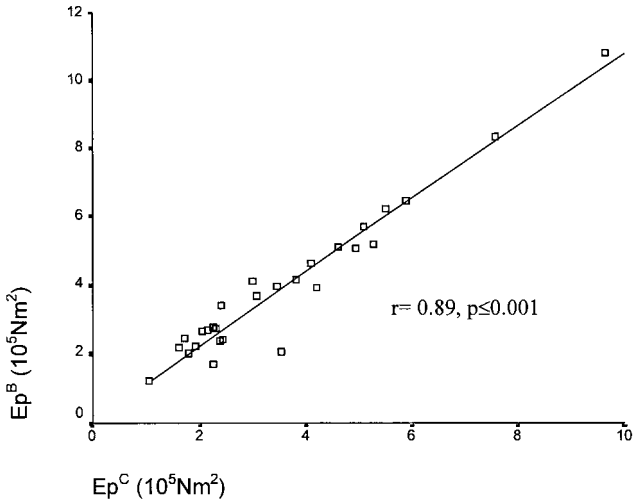

Fig. 1. Scatter plot of correlation between Ep calculated using brachial and central aortic pressures (r = Spearman's rank correlation).

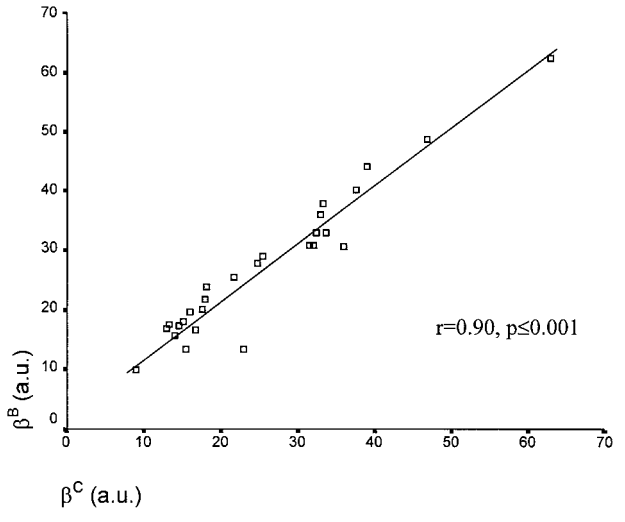

Fig. 2. Scatter plot of Spearman's rank correlation between β calculated using brachial and central aortic pressures.

than in the aorta, and pulse pressure was lower in the brachial artery. Not only are these findings difficult to explain physiologically, but they are also in contrast to the majority of other data.[11–14] The most likely explanation may be that because the authors compared invasive aortic pressure measurement with sphygmomanometrically determined brachial artery pressure, the error was dependant on the method of measurement rather than the site of measurement. Indeed, the inaccuracy of sphygmomanometric blood pressure measurements has been previously reported.[26]

As the pressure wave travels through the arterial tree from the large, elastic arteries to the smaller, muscular vessels, the speed and amplitude of the wave increase because of decreasing vessel compliance. The pressure contour also becomes distorted along the arterial tree: the systolic portion becomes narrowed and elevated; the incisura is damped and eventually disappears: a hump appears in its place in the diastolic portion. This damping of the high frequency components of the pressure wave is attributed to the viscoelastic properties of the arterial wall. Reflection, vascular tapering and transmission velocity enhance the peaking of the pressure wave. The result is that in the young there is a pronounced difference in central and peripheral pressures, systolic pressure increasing distally whilst diastolic pressure remains essentially unchanged,[27] i.e. there is amplification of the waveform (Fig. 3).

Ageing of the arterial tree reduces vessel distensibility (increases stiffness) and markedly reduces the difference between central and peripheral systolic pressure while increasing pulse pressure, especially in the aorta. This is because stiffer arteries transmit the pressure wave at a higher velocity, i.e. pulse wave velocity is increased. The result is that a larger than normal reflected pressure wave returns to the heart earlier, augmenting late systolic peak pressure.[12] Thus, whilst age increases aortic systolic pressure, peripheral systolic pressure is much less affected, so the gradient between central and peripheral systolic pressure is reduced.[14] Pauca *et al.*[14] examined a group of subjects aged 48–77 (median 61) years and found the ascending aortic systolic pressure to be 12 mmHg lower than

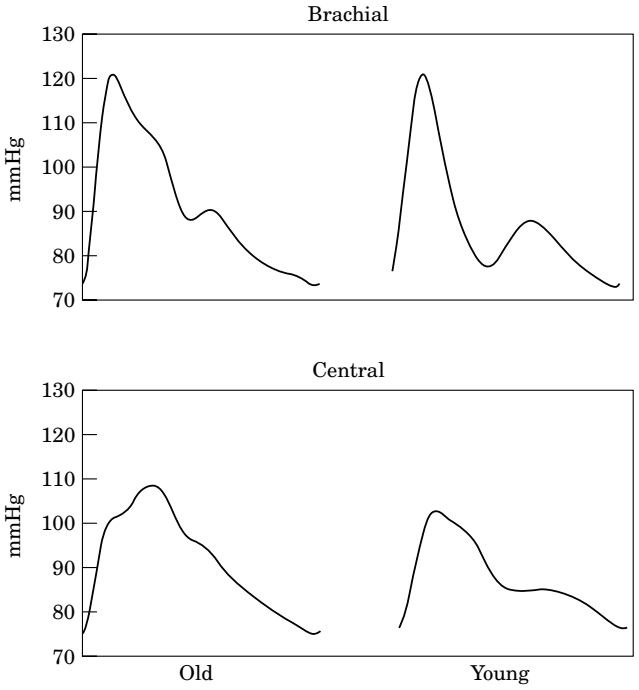

Fig. 3. Central arterial waveform (lower panel) and peripheral waveform (upper panel) in a young (right) and an elderly (left) subject. Reproduced from Wilkinson *et al.* 1998.[17]

radial systolic pressure and ascending aortic diastolic pressure to be 1 mmHg higher than radial pressure. In the present study, the median central-brachial pressure difference was 6 mmHg for systolic pressure but there was no difference in diastolic pressure. The amplification ratio (peripheral pulse pressure:central pulse pressure) of 1.1 reflects the older age of our study population (68–84, median 74 years).

Aortic pressure was that in the aortic arch and not at the site of the AAA. Abdominal aortic pressure remains extremely difficult to measure non-invasively at present. However, the PWA-derived ascending aortic pressure is the closest approximation available and is considerably closer than the brachial artery. Previous work has suggested that in evaluating ascending aortic dilation and distensibility in Marfan's syndrome, the pulse pressure in the carotid artery may be more useful than that from the brachial artery.[28] It may also be the case that aortic arch pressure is more representative of pressure at the site of the AAA than that at the brachial artery. However, this requires further study and a comparison of distensibility calculated using invasive aortic pressure at the site of the AAA with that using derived central pressures.

The principle finding of the study was that use of brachial pressure as opposed to central aortic pressure significantly increased Ep by 18% and β by 11%. This is of a similar magnitude to that of the previous authors.[10,15] The error was a systematic overestimate of Ep and β; however, the margin of error in calculation of β was relatively smaller because it is less pressure dependent than Ep. The importance of these findings is that this non-invasive method of aortic wall distensibility measurement can be used successfully in the clinical setting. Use of non-invasive, derived aortic pressure would enhance the accuracy of distensibility measurement; however, the error caused by using non-invasive brachial pressure is deemed small and systematic. Previous findings of this group[2,3] suggest that routine follow-up of distensibility and diameter could provide a greater understanding of AAA wall degeneration than diameter alone. If this is the case then the systematic nature of the error should not bias the measurements because it is the change in the measurements over time that provides the important information and not the absolute values.

In conclusion, we view the overestimate of Ep and β (by 18% and 11% respectively) to be small and, therefore, acceptable clinically. The linearity of the relationship between central and peripherally derived distensibility shows that the error is a small and systematic overestimate and would not bias follow-up comparison of changes in distensibility within each patient. However, since the discrepancy between central and peripheral systolic pressure (i.e. pressure amplification) is age-dependent, greater differences between Ep^b/β^b and Ep^c/β^c may occur in younger individuals, and care must be exercised when comparing measures of distensibility based on peripheral blood pressure measurements between age groups.

Acknowledgements

Funding was provided by the British Heart Foundation.

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Accepted 13 July 2001