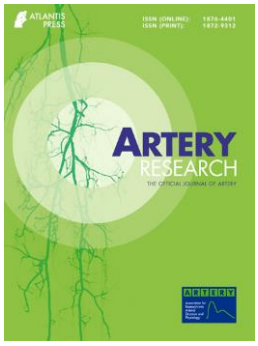

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P6.14: WHICH ESTIMATE OF MEAN ARTERIAL PRESSURE IS TO BE USED FOR ADJUSTMENT FOR PULSE WAVE VELOCITY?

T.K. Soender, L.M. Van Bortel, J. Lambrechtsen, J. Hangaard, J. Moeller, K. Egstrup

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Parameter	P value	Odds ratio
PWV	0.024	1.329
Use of BAB	0.560	0.671
Use of ACE-I	0.186	0.428
Use of CCB	0.027	4.131
Use of nitrates	0.570	1.555
Use of statins	0.854	0.857

Conclusions. Increased aortic PWV correlates significantly with the presence of significant CAS and may be important integrative marker for CAD.

P6.13
IMPACT OF CALIBRATION ON ESTIMATES OF CENTRAL BLOOD PRESSURE

T. K. Soender [1], L. M. Van Bortel [2], J. Lambrechtsen [3], J. Hangaard [4], J. Moeller [5], K. Egstrup [1]
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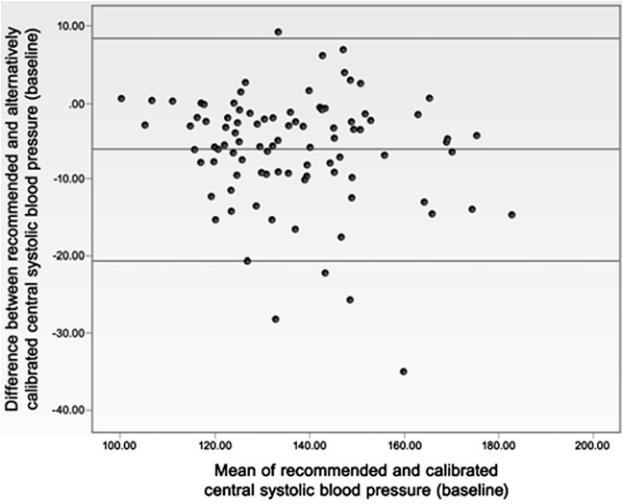
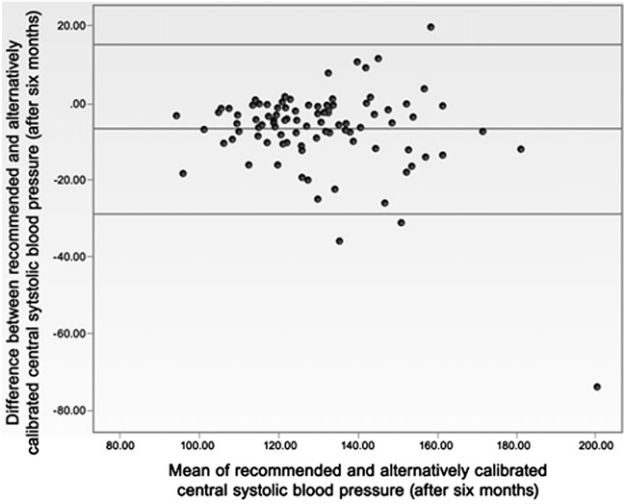
[2]*Heymans Institute of Pharmacology, Gent University, Gent, Belgium*
[3]*Department of Cardiology, University Hospital of Odense, Svendborg, Denmark*
[4]*Department of Endocrinology, University Hospital of Odense, Svendborg, Denmark*
[5]*Department of Cardiology, University Hospital of Copenhagen, Rigshopsitalet, Copenhagen, Denmark Objective: To examine effects of calibration on estimates of central blood pressure (CBP)*

Methods: We included 122 patients with type-II-diabetes and hypertension. They were characterized as having controlled (CH), uncontrolled (UH) or resistant (RH) hypertension based on ambulatory blood pressure measurement and number of antihypertensive agents. CBP was estimated using Sphygmocor. We calibrated the radial pressure wave as recommended by the manufacturer using brachial systolic (BSBP) and diastolic (BDBP) BP and used the generalized transfer function for estimation of the aortic pressure waveform. Afterwards we recalibrated the radial pressure wave using BDBP and mean arterial pressure (BMAP). Data were analyzed offline in customized software.
Results: Estimates of CSBP were significantly correlated ($R2=0.83$, $P<0.0001$ at baseline and $R2=0.71$, $P<0.0001$ after six months). As shown in figures 1 and 2 CSBP was 6.1 mmHg to 6.6 mmHg lower when calibrated with BSBP and BDBP and variation of difference ranged from 14.6 mmHg to 22 mmHg.
Differences between BSBP and CSBP lost significance in patients with RH after six months when alternatively calibrated with BDBP and BMAP as did reduction in CSBP.
Conclusion: Although we found good correlation between estimates of CBP using either calibration, variation of difference was high suggesting that the difference between the 2 calibration methods is more than a systematic error. And our results show that it could be important to take into account measures used for calibration when interpreting clinical effect on non-invasive estimates of CBP.





P6.14
WHICH ESTIMATE OF MEAN ARTERIAL PRESSURE IS TO BE USED FOR ADJUSTMENT FOR PULSE WAVE VELOCITY?

T. K. Soender [1], L. M. Van Bortel [2], J. Lambrechtsen [3], J. Hangaard [4], J. Moeller [5], K. Egstrup [1]
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[4]*Department of Endocrinology, University Hospital of Odense, Svendborg, Denmark*
[5]*Department of Cardiology, University Hospital of Copenhagen, Rigshospitalet, Copenhagen, Denmark Objective: To examine correlation of different estimates of mean arterial pressure (MAP) to pulse wave velocity (PWV) and their effect on statistical adjustment*

Methods: Patients with hypertension and type-II-DM were included. They were characterized as having controlled (CH), uncontrolled (UH) or resistant hypertension (RH). Patients with UH and RH received intensified antihypertensive treatment for six months.
We measured clinic BP using Omron HEM 757, ABPM rmed using Kivex TM 2430 and Spacelab 90217devices and PWV using Sphygmocor.
Results: We included 108 patients.
MAP measured immediately before measurement of PWV correlated to PWV ($R^2=0.1$, $P=0.001$ at baseline and $R^2=0.16$, $P<0.0001$ after six months) whereas ABPM MAP did not ($R^2=0.01$, $P=0.31$ at baseline and $R^2=0.01$, $P=0.25$ after six months).
PWV was significantly higher in patients with RH than in patients with CH ($P=0.001$) but we found no significant reduction in PWV after six months ($P=0.16$). Reduction in PWV in patients with UH was significant ($P=0.02$). PWV remained significantly higher in patients with RH ($P=0.05$) as did reduction in PWV in patients with UH ($P=0.05$) when including ABPM MAP in the statistical model. However when including clinic MAP, PWV was no longer significantly higher in patients with RH ($P=0.08$) and reduction in PWV in patients with UH lost significance ($P=0.33$).
Conclusion: Clinic MAP correlates best with PWV and using clinic MAP for statistical adjustment produces different results from using ABPM MAP. As PWV has a diurnal variation, MAP taken at the same time as PWV is likely the better to correct for.

Variable	Baseline			After six months		
	CH (N=33)	UH (N=33)	RH (N=42)	CH	UH	RH
SBP	122 [118;125]	142 [138;150]	138 [132;147]	126 [117;135]	133 [123;143]	136 [126;143]
PP	52 [45;56]	65 [58;68]	66 [62;70]	55 [43;63]	57 [49;62]	64 [57;70]
PWV	8.3 [7.3;10.6]	9.6 [8.3;11.1]	10.9 [8.4;12.8]	8.8 [7.3;10.1]	8.9 [8;10]	10.3 [8.4;12.8]

P6.15
EFFECT OF BODY POSITION ON THE MEASUREMENTS OF CENTRAL HEMODYNAMIC PARAMETERS: "PLEASE HAVE A SIT?" OR "PLEASE LIE DOWN?"

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Background: Estimation of aortic pressure waveform provides clinical information regarding BP cardiovascular risk additional to the brachial blood pressure (BP). The effect of body position on central haemodynamics (BP, pulse pressure (PP) amplification (amp), augmentation index (AIx), augmentation pressure (AP), subendocardial viability ratio (SVR) have never been investigated. Aim/design: to investigate in a randomized cross over study changes in both the peripheral and central haemodynamics in supine and sitting position. Methods: Sixty one subjects referred for BP assessment were examined (36 males, mean age 50±12 yrs). Brachial and aortic waveforms were assessed in sitting and supine position. In each position: triplicate brachial BP measurements were performed; then 2 consecutive aortic pressure waveforms were estimated by applanation tonometry of the radial artery - pulse wave analysis and the use of transfer functions (Sphygmocor). The average of the last 2 brachial BP recordings was used in statistical analysis and for peripheral waveforms calibration. Results: Mean arterial BP did not differ significantly between the sitting and supine position (table). Brachial and aortic SBP, PP, AP, AIx were significantly higher in the supine position whereas DBP and PP amplification (ratio: brachial/aortic PP) significantly smaller. Moreover, significant alterations were observed in heart rate, ejection duration and SVR. Conclusions: Mean BP remained unchanged but the pulsatile BP component was higher in the supine position. This was more pronounced in the aorta, as shown by PP amplification, in part due to alterations in heart rate, wave reflections leading to alterations in coronary perfusion.

Parameter	Sitting position	Supine position	p-value
Mean BP (mmHg)	110.8 ± 13.7	110.9 ± 14.9	0.945
Brachial SBP (mmHg)	140.1 ± 17.4	142.7 ± 18.5	0.022
Brachial DBP (mmHg)	94.2 ± 14.4	90.1 ± 14.4	<0.001
Brachial PP (mmHg)	45.9 ± 16.0	52.6 ± 15.6	<0.001
Aortic SBP (mmHg)	131.7 ± 16.9	134.4 ± 18.6	<0.001
Aortic DBP (mmHg)	95.0 ± 14.4	91.3 ± 14.5	<0.001
Aortic PP (mmHg)	36.7 ± 15.2	43.1 ± 13.9	<0.001
AP (mmHg)	10.8 ± 7.7	13.9 ± 7.3	<0.001
AIx (%)	26.9 ± 11.9	31.1 ± 10.2	<0.001
PP amplification (ratio)	1.3 ± 0.2	1.2 ± 0.1	<0.001
Heart rate (bpm)	67.2 ± 8.7	64.5 ± 7.4	<0.001
Ejection duration (msec)	297.2 ± 22.7	327.6 ± 17.6	<0.001
SVR	179.6 ± 25.7	161.2 ± 25.8	<0.001

P6.16
DETERMINATION OF CAROTID AND FEMORAL WAVE SPEED AND DISTENSIBILITY IN A HEALTHY POPULATION USING A NEW NON-INVASIVE TECHNIQUE

A. Borlotti [1], S. Vermeersch [2], E. Rietzschel [3], P. Segers [2], A. W. Khir [1]
[1]*Brunel University, Brunel Institute for Bioengineering, Uxbridge, London, United Kingdom*
[2]*Ghent University, IBiTech-bioMMeda, Gent, Belgium*
[3]*Ghent University Hospital, Gent, Belgium Local wave speed (c) is a predictor of cardiovascular diseases because is related to arterial distensibility*

In this work, carotid and femoral distensibility were assessed in the Asklepios study population. Local wave speed was determined with a new non-invasive technique based on velocity (U) and diameter (D) measurements (lnDU-loop) [1]. Distensibility was calculated using c and the Bramwell-Hill equation, and changes were studied with respect to age and gender.
Figure 1 shows changes in carotid and femoral wave speed (a) and distensibility (b) with age and gender. Carotid wave speed increases and distensibility decreases with age (a part from male aged 40-45 and 45-50) and there is no difference between males and females. In the femoral artery, these parameters do not change with age and wave speed is lower and distensibility is higher in females.
The mechanical properties of elastic (carotid) and muscular (femoral) arteries change differently with age, which is in line with results of other investigators. The new technique provides a means for the determination of arterial distensibility using non-invasive measurements of D and U, which can potentially be clinically useful as they could be taken using Doppler ultrasound.

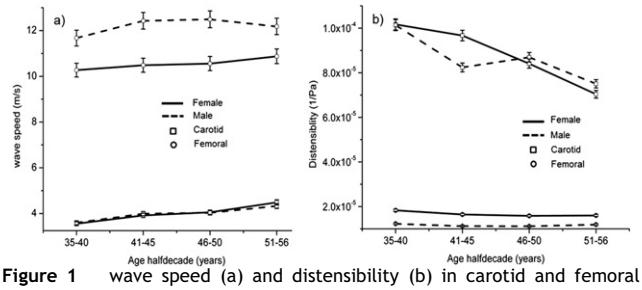

Figure 1 wave speed (a) and distensibility (b) in carotid and femoral arteries

1. Feng and Khir Determination of wave speed and wave separation in the arteries using diameter and velocity. J.Biomech. 43: 3: 455-462, 2010.

P6.17
CAROTID ARTERY CROSS-SECTIONAL AREA AND STIFFNESS NON-LINEARITY AS MARKERS OF VASCULAR AGEING

K. D. Reesink, E. Hermeling, L. Veenstra, J. Op't Roodt, M. Daemen, J. Waltenberger, A. P. G. Hoeks, A. A. Kroon
Maastricht University Medical Centre, Maastricht, Netherlands

Background: Artery wall ageing involves elastic fibre loss and increased fibrosis, leading to dilatation and increased artery wall stiffness. Notably, the associated gradual transfer of tensile stresses from elastic to stiffer components in the wall will likely increase non-linear elastic behaviour of the vessel wall. We investigated whether age is indeed a determinant of carotid artery cross-sectional area (CSA) and stiffness non-linearity; the latter being quantified as the difference between systolic and diastolic pulse wave velocity: ΔPWV, in m/s (Figure).