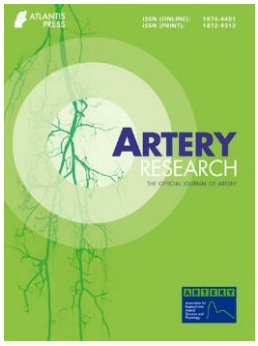

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10.8: SYSTOLIC AORTIC PRESSURE DERIVED FROM DIFFERENT CALIBRATION METHODS IN THE GENERAL POPULATION

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Methods: The cSBP was estimated from radial artery tonometry and a transfer function using a SphygmoCor® system (AtCor Medical, Australia) in 100 subjects (mean age+/-SD = 57+/-10 years). Pressure waveforms were calibrated from the brachial systolic and diastolic pressures, measured just prior to tonometric measurement with an oscillometric cuff system (Omron 705CP Omron, Japan). DCBP and cSBP were compared using the Bland and Altman method and subgroups were compared using unpaired Student's t test.
Results: The difference between DCBP (129.2+/-16.8 mmHg) and cSBP (129.4+/-16.4 mmHg) was -0.2+/-2.6 mmHg. The difference was not influenced by the mean (DCBP + cSBP / 2). Similar results were obtained in men (n=60) and women (n=40), and in subjects with/without hypertension (n=51/49), with/without diabetes (26/74), and with/without dyslipidemia (36/64).
Conclusions: The new DCBP® method and the transfer function applied to radial tonometry method were interchangeable in estimating central SBP. These results pertained strictly to the studied population (patients aged 57 years on average, and displaying a high percentage of cardiovascular risk factors). Further studies are needed to confirm our results.

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10.8
SYSTOLIC AORTIC PRESSURE DERIVED FROM DIFFERENT CALIBRATION METHODS IN THE GENERAL POPULATION

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Background: There is recent evidence from different research groups that accuracy [1] and prognostic value [2,3,4] of systolic aortic pressure significantly depends on the method of calibration. Although these results consistently show superiority of mean pressure calibration (aSBP2) over both, traditional calibrated aortic systolic (aSBP1) and brachial systolic pressure (bSBP), the investigated cohorts were relatively small and it is still unclear whether the observed associations between pressures are preserved in the general population.
Objective: Therefore the objective of this work is the investigation of associations between different methods of systolic pressure assessment it a large cohort and its comparison to reported outcome.
Methods: During a public health campaign cardiovascular hemodynamic data was assessed using the Mobil-O-Graph® device and ARCSolver® algorithms in a kiosk like setting. Systolic aortic pressure was derived from two different calibrations: systolic and diastolic pressure and mean and diastolic pressure. Furthermore brachial pressures, age, sex and anthropometric data were recorded and regression analysis was performed to investigate associations.
Results: Summary statistics of 7409 valid measurements are reported in Table 1. Systolic and subsequent pulse pressures significantly differed from bSPB for aSBP1 but not for aSBP2. Regression analysis unveiled that aSBP2 (R^2=0.853) is significantly ($p<0.00001$) less associated with bSBP than aSBP1 (R^2=0.937), see Figure 1.
Conclusions: Comparison of our data with literature suggests that unlike aSBP1 [5] the association between bSBP and aSBP2 is only slightly influenced by increased sample size [2] and therefore prognostic superiority over bSBP is likely to be sustainable and warrants further investigation.

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10.9
ARTERIAL STIFFNESS INDEX BETA AND CARDIO-ANKLE VASCULAR INDEX INHERENTLY DEPEND ON BLOOD PRESSURE, BUT CAN BE READILY CORRECTED

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Objectives: Arterial stiffness index β and cardio-ankle vascular index (CAVI) are widely accepted to quantify the blood pressure (BP)-independent, intrinsic exponent (β_0) of the BP-diameter relationship. CAVI and β assume an exponential relationship between pressure (P) and diameter (d). We aim (1) to demonstrate that, under this assumption, β and CAVI as currently implemented are inherently BP-dependent and (2) to provide corrected, BP-independent forms of CAVI and β.
Methods and results: In $P=P_{ref}*\exp[\beta_0(d/d_{ref}-1)]$, usually reference pressure (P_{ref}) and reference diameter (d_{ref}) are substituted with diastolic BP and diameter to accommodate measurements. Consequently, the resulting exponent is not equal to the pressure-independent β_0. CAVI does not only suffer from this reference pressure effect, but also from the approximation of dP/dd.
For example, assuming $\beta_0=7$, an increase of systolic/diastolic BP from 110/70 to 170/120 mmHg increased β by 8.1% and CAVI by 14.3%. We derived corrected forms of β and of CAVI ($CAVI_0$) that did not change with BP and represent the pressure-independent β_0.
To substantiate the BP effect on CAVI in a typical follow-up study, we realistically simulated patients (n=161) before and following BP-lowering treatment (assuming no follow-up change in intrinsic β_0 and therefore in actual P-d relationship). Lowering BP from 160±14/111±11 to 120±15/79±11 mmHg ($p<0.001$) resulted in a significant CAVI decrease (8.1±2.0 to 7.7±2.1, $p=0.008$) $CAVI_0$ did not change (9.8±2.4 and 9.9±2.6, $p=0.499$).
Conclusions: β and CAVI as currently implemented are inherently BP-dependent, potentially leading to erroneous conclusions in arterial stiffness research. BP-independent forms were derived to overcome this problem.

Summary statistics

	Male			Female		
	N	Median	2.5 — 97.5 P	N	Median	2.5 — 97.5 P
Age	2276	54,000	24,000 to 83,000	5133	54,000	20,000 to 81,000
aSBP1	2276	120,803	98,754 to 153,254	5133	114,872	91,723 to 152,578
aSBP2	2276	133,102	108,651 to 172,962	5133	124,914	100,692 to 167,257
SBP	2276	131,000	107,000 to 168,000	5133	124,000	100,000 to 165,000
MBP	2276	106,000	85,000 to 133,000	5133	99,000	79,000 to 129,000
DBP	2276	84,000	63,000 to 109,000	5133	77,000	58,000 to 102,175
BMI	2276	26,219	20,065 to 35,774	5133	23,875	18,218 to 36,634

Regression between bSBP, aSBP1 and aSBP2 respectively.