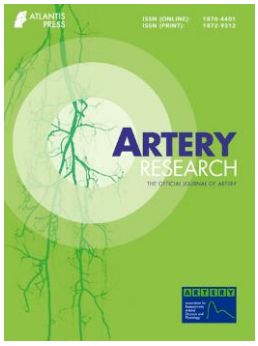

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12.05: AMBULATORY ARTERIAL STIFFNESS INDEX: ANOTHER AMBIGUOUS STIFFNESS INDEX?

J.G. Kips, S.J. Vermeersch, P. Reymond, P. Boutouyrie, N. Stergiopulos, S. Laurent, L. Van Bortel, P. Segers

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P12.02
VARIATIONS OF WAVE REFLECTION INDEXES INDUCED BY ACUTE BLOOD PRESSURE CHANGES AT DIFFERENT ARM HEIGHTS

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Acute blood pressure (BP) changes might influence augmentation index (AIx), an integrated dimensionless measure of reflected wave timing and amplitude. In 30 healthy subjects (49±16 years, 43% men), supine brachial BP and radial-artery waveform (applanation tonometry, SphygmoCor) were obtained with the right arm supported in 3 different positions: at the heart level (0°), raised by 30° (+30°), and lowered by 30° (-30°). BP and tonometric measures were also obtained on the contralateral arm, which was held at the heart level during the examination.
Brachial systolic/diastolic BP was 121/67±18/8 mmHg. Radial AIx was 84±19%, and estimated central AIx 27±14%. As expected, changes in arm position modified substantially mean BP (96±12 mmHg at -30°, 85±11 mmHg at 0°, 74±11 mmHg at +30°, all $p<0.001$).
Radial and central AIx were both reduced at -30° (71±22% and 17±17%), and increased at +30° (97±21% and 30±14%, all $p<0.001$) vs corresponding values at 0°. Heart rate and contralateral BP and AIx did not change. Changes in radial and aortic AIx were strongly related each other ($r=0.76$, $p<0.001$). Percent variation in radial AIx (highest minus lowest, divided by AIx at heart level) had a strong inverse relationship with age ($r=-0.43$, $p<0.001$) and systolic BP ($r=-0.37$, $p<0.001$).
In conclusion, acute gravitational upper-limb BP changes generate opposite changes in radial AIx. Acute changes in radial Aix decrease with age and BP levels, and might represent a novel index of vascular aging. Artifactual changes in aortic AIx may arise in the presence of radial-aortic distending pressure gradient.

P12.03
VALIDATION OF A BRACHIAL CUFF-BASED METHOD FOR ASSESSING CENTRAL BLOOD PRESSURE AT REST AND DURING LIGHT EXERCISE

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Background: Central blood pressure (BP) may be more predictive of cardiovascular events than brachial BP. A cuff-based ambulatory central BP monitor is now available; the aim of this study was to compare values of central BP between this device and the SphygmoCor device.
Methods: Two studies were conducted. Study 1: We compared seated central systolic BP (cSBP) and pulse pressure (cPP) between the Mobil-o-graph and SphygmoCor devices. Study 2: We compared cSBP and cPP between the Mobil-o-graph and SphygmoCor devices at rest and during light bicycle exercise, corresponding to approximately 12 and 25 watts.
Results: Study 1 contained 51 healthy subjects (mean age 51±20yrs, 31 females) and study 2 contained 20 subjects (mean age 43±11yrs, 9 females). Study 1:The mean difference between devices was 1±5mmHg, $P=0.18$ (cSBP) and 0±4mmHg, $P=0.54$ (cPP). There was a strong correlation between devices for cSBP ($r=0.94$, $P<0.0001$) and cPP ($r=0.92$,$P<0.0001$). Study 2:The mean difference in cSBP between devices was 1±3mmHg at rest and 1±6mmHg at the highest workload. The mean difference in cPP between devices was 0±3mmHg at rest and 0±6mmHg at the highest workload. The devices were strongly correlated at rest ($r=1.00$, $P<0.0001$, cSBP) and ($r=0.87$, $P<0.0001$, CPP) and at the highest workload for ($r=0.94$, $P<0.0001$, cSBP) and ($r=0.85$, $P<0.0001$, cPP).
Conclusion: Non-invasive measurement of central BP by the mobilograph device is in good agreement and highly correlated with the widely used SphygmoCor device, both at rest, and in response to light exercise simulating everyday activities during which ambulatory BP measurements might be made.

P12.04
HOW MUCH DOES PRESSURE WAVE REFLECTION CONTRIBUTE TO AUGMENTATION INDEX?

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Background: Aortic pulse pressure can be partitioned into the height of the first systolic shoulder (P1) and augmentation pressure AP. P1 is thought to be determined by an outgoing pressure wave generated by ventricular contraction and AP by a backward wave "reflected" from the distal circulation. Augmentation index (AIx = AP / cPP) is commonly used to quantify wave reflection. Nitroglycerin (NTG) has a powerful effect to reduce AIx which has been attributed to a reduction in wave reflection. The objectives of this study were to examine the contribution of forward and backward waves to AIx at rest and after administration of NTG.
Methods: A ComboWire 9500 catheter (VolcanoCorp, USA) with a Doppler probe and a pressure sensor at the tip was placed in the aortic root in 21 subjects (11 men, aged 45-81). Simultaneous measurements of aortic blood flow velocity and blood pressure were made at baseline and after the admission of sublingual NTG (400 μg). Using wave decomposition, AIx was expressed as the summation of forward and backward components, F_{AIx} and B_{AIx} respectively.
Results: AIx decreased by 17.3% (from 39.5±3.6 to 22.2±5.0%, $P<0.001$) after NTG. The decrease in AIx was attributable to a similar decrease in both forward and backward components (decreases in F_{AIx} and B_{AIx} by 8.7% and 8.6% respectively ($P<0.05$).
Conclusions: These results suggest the forward wave is a major determinant of AIx and that the role of reflection in mediating effects of NTG may be less than previously thought.

P12.05
AMBULATORY ARTERIAL STIFFNESS INDEX: ANOTHER AMBIGUOUS STIFFNESS INDEX?

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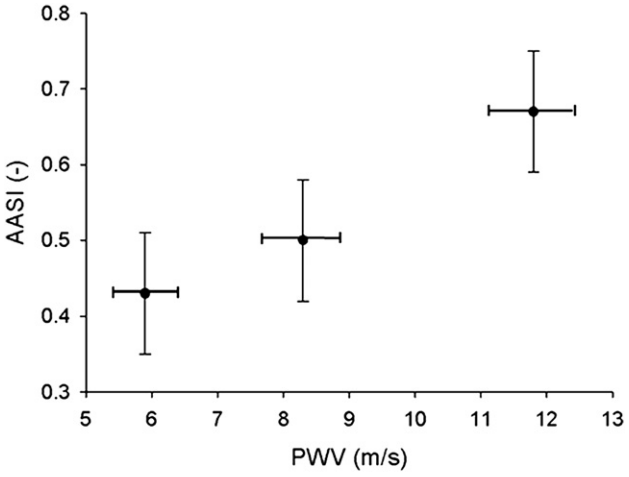
Introduction: The Ambulatory Arterial Stiffness Index (AASI), derived from ambulatory blood pressure (ABPM) recordings, has been proposed as a surrogate marker of arterial stiffness. However, there is controversy to which extent it reflects stiffness or is affected by other parameters. Using a computer model of the arterial circulation, the relative importance of the different determinants of the AASI was explored.
Methods: Arterial distensibility (inverse of stiffness), peripheral resistance, heart rate, maximal cardiac elastance and venous filling pressure were varied from 80 to 120% of their initial value in steps of 10% to generate 3125 BP-values, mimicking the daily fluctuations in one theoretical subject. From this dataset, we assessed the confidence with which AASI can be derived in this subject, as well as the influence of different individual parameters on AASI. To assess the ability of AASI to detect large changes in arterial stiffness, two additional subjects were simulated with a distensibility of 50% and 25% of the default distensibility, respectively.



Range of AASI- and PWV-values associated with each of the three theoretical subjects

Results: The distribution of AASI-values, obtained from 10000 ABPM simulations (each using 72 BP-values randomly selected among 3125) was normal (AASI=0.43±0.04 (SD)).
An increase in heart rate, distensibility or resistance from 80 to 120% of its default value caused the AASI to decrease by 37, 21 or 9%, respectively. Whereas there was no overlap in the distensibility ranges for the three theoretical subjects, the was considerable overlap between the AASI distributions.
Conclusion: The confounding effects of resistance and heart rate limit the use of AASI as a marker of stiffness.

P12.06
COMPARISON OF SIMULTANEOUS INVASIVE CENTRAL ARTERIAL PRESSURE MEASUREMENTS WITH NON-INVASIVE ARTERIAL PRESSURE ESTIMATES BY SUPRASYSTOLIC OSCILLOMETRY USING PULSECOR R6.5

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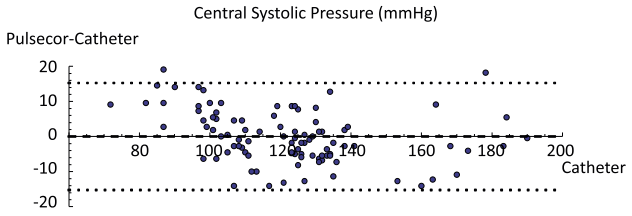
Background: Many devices estimate central aortic blood pressure (BP) from non-invasive measurements. Most need calibration using separately measured BP, which introduces unquantified inaccuracies. The Pulsecor R6.5 device estimates central BP using a suprasystolic brachial cuff and built-in oscillometric BP unit in approximately 60 seconds. We compared central BP estimated using the Pulsecor device and those obtained by catheter during coronary angiography.
Methods: 94 central pressure waveforms were recorded by catheter in 37 subjects (61±12yrs) undergoing diagnostic coronary angiography. Pulsecor central pressures were compared using Bland-Altman analysis with ensemble-averaged catheter pressures obtained simultaneously over the period of the Pulsecor measurement.
Results: Cohort mean central BPs estimated by Pulsecor and invasively were very similar. The spread of differences was larger for systolic than diastolic and mean pressures, although all were within Association for the Advancement of Medical Instrumentation (AAMI) standards.
Conclusions: Pulsecor R6.5 accurately estimates central aortic BPs calibrated using built-in oscillometric BP measurement.

Central BP	Catheter	Pulsecor	Difference	P-value	Pearson's r
Systolic	122.3 ± 23.6	122.3 ± 22.3	0.0 ± 7.7	1.00	0.95
Diastolic	68.7 ± 8.6	68.8 ± 6.00	0.1 ± 5.1	0.9372	0.81
MAP	91.3 ± 11.5	91.3 ± 10.0	0.0 ± 4.8	0.9785	0.91

Data reported as mean±sd, mmHg



P12.07
COMPARISON OF TWO RADIOFRAQUENCY-BASED SYSTEMS FOR ASSESSMENT OF LOCAL CAROTID STIFFNESS

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Objective: two ultrasound systems (QAS, Esaote; and E-Track, Aloka) provide radio-frequency (RF)-based tracking of carotid wall, allowing real-time determination of vessel diameter, distension, and stiffness (CS). Measurement is performed in a single line by E-Track and in 16 equidistant lines by QAS.
Aim: to evaluate whether measures of CS with the two systems are comparable and to assess intra- and interoperator variability.
Methods: MyLab 70 (Esaote) and Alpha 7 (Aloka) were used in random order to measure right CCA diameter and distension, and to calculate distensibility coefficient (DC) in 173 subjects (5 groups: 21 controls (NL), 35 prehypertensives (PHBP), 23 hypertensives (HBP), 27 type 2 diabetics (DM) and 67 HIV-positive patients. In 30 subjects, the study was repeated after 60-min, both by the same and by another operator.
Results: correlation coefficients between the two systems for CCA diameter, distension and DC were high (r=0.84, 0.90 and 0.87, p<0.0001). QAS provided significantly (P<0.001) higher CCA diameter and lower distension and DC than E-track (7.58±1.07 vs. 7.35±1.00 mm; 378±146 vs. 447±154 μm; and 0.35±0.17 vs. 0.44±0.19 kPa). In the 5 study groups, DC obtained with QAS and E-Track discriminated among them with similar statistical significance. Intra- and inter-operator variability for CCA distension was 7.5±4.6% and 9.0±6.9% with QAS and 9.8±8.5% and 12.4±649% with E-Track.
Conclusions: measures of CS with QAS and E-Track are correlated and equally effective to discriminate diseased populations. Yet, CS values are higher with QAS, and therefore the systems are not interchangeable

P12.08
SYSTOLIC HYPERTENSION MECHANISMS: EFFECT OF GLOBAL AND LOCAL PROXIMAL AORTA STIFFENING ON PULSE PRESSURE

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Decrease in arterial compliance leads to an increased pulse pressure. Pressure waveform is the sum of a forward and a reflected wave, which are altered when the arterial system stiffens. Two mechanisms have been proposed in the literature to explain systolic hypertension upon arterial stiffening. One is based on the augmentation and earlier arrival of reflected waves. The second is based on the augmentation of the forward wave due to increased characteristic impedance of the ascending aorta.
A validated 1-D model of the systemic arterial tree was used to analyze the aforementioned mechanisms. The arterial tree was stiffened by decreasing compliance either locally in the aortic arch, or globally in all arteries.
The pulse pressure increased by 58% when proximal aorta was stiffened and the compliance decreased by 43%. Same pulse pressure increase was achieved when compliance of the globally stiffened arterial tree decreased by 47%. In presence of local stiffening in the aortic arch, characteristic impedance increased by 3 times and led to a substantial increase in the amplitude of the forward wave. Under global stiffening, the pulse pressure of the forward wave increased by 41% and the amplitude of the reflected wave by 83%.
Local stiffening in the proximal aorta increases systolic pressure mainly through the augmentation of the forward wave, whereas global stiffening augments systolic pressure principally through the increase in wave reflections. The relative contribution of the two mechanisms depends on the topology of arterial stiffening and geometrical alterations taking place in aging or in disease.

P12.09
PULSE PRESSURE AMPLIFICATION, PRESSURE WAVEFORM CALIBRATION AND TARGET ORGAN DAMAGE

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