



## Artery Research

ISSN (Online): 1876-4401

ISSN (Print): 1872-9312

Journal Home Page: <https://www.atlantis-press.com/journals/artres>

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### **12.06: 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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**To cite this article:** A. Lin, A. Lowe, K. Sidhu, W. Harrison, P. Ruygrok, R. Stewart (2011) 12.06: COMPARISON OF SIMULTANEOUS INVASIVE CENTRAL ARTERIAL PRESSURE MEASUREMENTS WITH NON-INVASIVE ARTERIAL PRESSURE ESTIMATES BY SUPRASYSTOLIC OSCILLOMETRY USING PULSECOR R6.5, Artery Research 5:4, 202–202, DOI: <https://doi.org/10.1016/j.artres.2011.10.191>

**To link to this article:** <https://doi.org/10.1016/j.artres.2011.10.191>

Published online: 14 December 2019

**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, there 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.

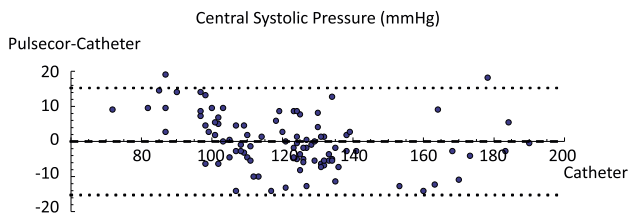
**Methods:** 94 central pressure waveforms were recorded by catheter in 37 subjects (61±12yrs) undergoing diagnostic coronary angiography. Pulsecor central pressures were compared using Bland-Altman analysis with ensemble-averaged catheter pressures obtained simultaneously over the period of the Pulsecor 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 RADIOFREQUENCY-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 ( $7.58\pm1.07$  vs.  $7.35\pm1.00$  mm;  $378\pm146$  vs.  $447\pm154$   $\mu\text{m}$ ; and  $0.35\pm0.17$  vs.  $0.44\pm0.19$  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  $7.5\pm4.6\%$  and  $9.0\pm6.9\%$  with QAS and  $9.8\pm8.5\%$  and  $12.4\pm6.49\%$  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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