12.09: PULSE PRESSURE AMPLIFICATION, PRESSURE WAVEFORM CALIBRATION AND TARGET ORGAN DAMAGE

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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 OSCILLOSCOPY USING PULSECOR R6.5
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Background: Many devices estimate central aortic blood pressure (BP) from non-invasive measurements. Most need careful 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. The ensemble-averaged catheter pressures obtained simultaneously over the period of the Pulsecor measurement.

Results: Cohort mean central BP's 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 BP's calibrated using built-in oscillometric BP measurement.

<table>
<thead>
<tr>
<th>Central BP</th>
<th>Catheter</th>
<th>Pulsecor</th>
<th>Difference</th>
<th>P-value</th>
<th>Pearson's r</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic</td>
<td>122.3 ± 23.6</td>
<td>122.3 ± 22.3</td>
<td>0.0 ± 7.7</td>
<td>1.00</td>
<td>0.95</td>
</tr>
<tr>
<td>Diastolic</td>
<td>68.7 ± 8.6</td>
<td>68.8 ± 6.0</td>
<td>0.1 ± 5.1</td>
<td>0.9372</td>
<td>0.81</td>
</tr>
<tr>
<td>MAP</td>
<td>91.3 ± 11.5</td>
<td>91.3 ± 10.0</td>
<td>0.0 ± 4.8</td>
<td>0.9785</td>
<td>0.91</td>
</tr>
</tbody>
</table>

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 (6 groups: 21 controls (NL), 35 prehypertensives (PHTP), 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 cm vs. 7.35 ± 1.00 cm; 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 ± 6.4% 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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Background: Obtaining pulse pressure non-invasively from applanation tonometry requires the calibration of pressure waveform with brachial systolic and diastolic blood pressure. In literature, several calibration methodologies are applied and clinical studies do not agree about the predictive value of central hemodynamic parameters.

Objective: To compare 4 calibration methodologies and assess the usefulness of pulse pressure amplification as an index independent of calibration.

Methods: We investigated 108 subjects with tonometry in carotid, femoral, brachial, radial and dorsalis pedis arteries; pulse pressure amplification between arterial waveforms was calculated. Four methods to calibrate the waveforms were compared: the 1/3 rule, the 40% rule, the integral of radial and brachial waveforms. Pulse pressure amplification in 5 arterial territories was studied (carotid-femoral, carotid-brachial, carotid-radial, and carotid-pedis amplifications; femoral-pedis amplification).

Results: Pulse pressure can non-invasively be measured in 5 arteries. Pulse pressure strictly depends on calibration, with differences up to 18 mmHg between methodologies. When pulse pressure amplification was calculated, calibration method effect disappeared. Furthermore, pulse pressure amplifications in 5 arterial territories presented a peculiar pattern of clinical/biological determinants: heart rate and body height were common determinants of carotid to brachial, radial and femoral amplifications; diabetes was related to carotid to brachial amplification and pulse wave velocity to femoral to pedis amplification.

Conclusion: The calibration method can influence the results from clinical trials and that pulse pressure amplification can evade this issue. We also suggest that the alteration of amplification in each arterial territory might be considered as a signal for the discovery of clinical/subclinical damage.

P12.10
TWO NEW INDICES FOR A MORE ACCURATE ASSESSMENT OF THE LOCAL AORTIC STIFFNESS

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Purpose: Recently, the local aortic stiffness (AoStiff) can be evaluated using a non-invasive bioelectrical impedance (BI) technique. Herein, AoStiff is estimated from the measurement of two new BI variables: 1) The local aortic flow resistance (AoRes) exerted by the drag forces onto the local aortic wall (2) The local aortic wall distensibility (AoDist). We propose to compare these two indices with the reference pulse wave velocity (PWV) measurement and the direct assessment of the aortic drag forces (DF) and distensibility (DS) obtained by the magnetic resonance imaging technique (MRI).

Methods: PWV was measured by tonometry (Pulse Pen, Diatrace) on 98 patients (mean age ± years). Local aortic properties (DF and DS indices) were estimated from phase-contrast MRI data on 9 patients (mean age ± 14 years). In both studies, AoRes and AoDist indices were evaluated by BI technique on patients at rest.

Results: A significant correlation was found between PWV and AoRes, AoDist respectively (r = 0.58, p < 0.0001, 95% confidence interval: 0.43 to 0.70; r = -0.45, p < 0.0001, 95% confidence interval: -0.60 to -0.27) as well as between AoRes and DF (r = 0.96, p < 0.0002, spearman coefficient) and between AoDist and DF (r = 0.95, p < 0.0004, spearman coefficient).

Conclusion: The local BI method relies on a differential analysis of AoStiff leading to two new indices (AoRes and AoDist) which show a higher and significant correlation with the parameters obtained with MRI technique than PWV method. Furthermore, a larger scale validation should be performed before considering these indices as a suitable alternative to standard methods.

P12.11
THE RETROGRADE FLOW A NOVEL PARAMETER OF AORTIC STIFFNESS

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Aim: The aim of this prospective study was, to quantifies parameters of the retrograde flow (volume, flow rate) using magnetic resonance phase shift velocity and to study there relationships with aortic stiffness parameters calculated with both MRI and tonometry and waves reflections parameters.

Materials: Eighty healthy subjects without history of cardiac disease were studied (50 men and 30 women). The median age of the subjects was 42 years, ranging from 13 to 79

Results: The mean value of the retrograde volume mean was 13.2 ml (range from 2.2 to 46 ml), or expressed as a percentage of antegrade flow, it was 2.1% (range from 0.31% to 9.5%). All parameters of the retrograde flow were correlated significantly with age in univariate analysis.

The rate of the pic of retrograde flow rate to pic of the antegrade flow rate was more closely correlated to age (r = 0.753, P < 0.05) compared to CF PWV (r = 0.8008, P < 0.05). All parameters of the retrograde flow correlates closely to the parameters of waves reflectin calculated (TI and AI).

Conclusion: The retrograde flow appear to be a novel parameter of aortic stiffness which correlates significantly with age and parameters of aortic stiffness and waves reflection.