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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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 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. 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.

<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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Objectives: To assess the agreement of two radiofrequency (RF)-based ultrasound systems for evaluating carotid stiffness using different RF protocols.

Methods: A total of 40 healthy volunteers, 20 of whom were smokers and 20 non-smokers, were included in the study. The RF-based ultrasound systems were QAS (Esaote) and E-Track (Aloka). The carotid artery was scanned in the longitudinal axis, and the stiffness index (SI) was calculated as the ratio of the forward pressure wave to the reflected pressure wave.

Results: The mean SI values were 0.25±0.09 for QAS and 0.31±0.11 for E-Track. The Bland-Altman analysis showed a significant difference between the two systems with a limit of agreement of ±0.15. The correlation coefficient (r) was 0.85 for QAS and 0.70 for E-Track.

Conclusions: The two systems have comparable performance for assessing local carotid stiffness, but the E-Track system provides slightly higher SI values and a lower correlation coefficient compared to QAS.

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. Pulse pressure 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 46%. 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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