8.4: DIURNAL CHANGES IN CENTRAL PRESSURE AND PULSE WAVE PARAMETERS IN HEALTHY SUBJECTS

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8.1 ARTERIAL STIFFNESS, BLOOD PRESSURE AND CARDIAC OUTPUT STUDY

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We are planning a prospective study in 200 patients with an abdominal artery aneurysm (AAA). Non-invasive measurements will be performed including tonometry-based pulse wave analysis (PWA) and pulse wave velocity (PWV), echocardiography, and 24-hour blood pressure measurements.

This study will provide insight in how PWV/PWA-parameters can help identify characteristics of prostheses used to treat AAA that best match native arterial characteristics and will lead to the best long-term outcome after aneurysm repair. Also the interaction between blood pressure (and control) and cardiac output will be evaluated. These results will form the basis for evidence-based practice for stent choice and lead to better outcomes after AAA treatment.

First we will validate non-invasive against invasive central pressure in 20 patients treated with endovascular aneurysm repair (EVAR).

This study will provide insight if arterial stiffness parameters change over time after treatment of AAA and the possible role of PWV/PWA for the surveillance after treatment. We expect to provide insight in the various determinants of the PWV/PWA-parameters pre- and post-repair of AAA.

We will study whether the different PWV/PWA parameters predict outcome after AAA repair for different prostheses.

Finally, this study will reveal whether parameters of cardiac output obtained by tonometry correspond with parameters obtained by echocardiography in AAA patients. If so, the PWV/PWA measurement can detect cardiovascular problems at an early stage during follow-up. By early treatment, the development of heart failure can be delayed or even prevented. We look forward to input on our study-plan.

8.3 QUANTIFYING HEART AND ARTERIAL CONTRIBUTIONS TO CENTRAL BLOOD PRESSURE IN SYSTOLE

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Background: A recent study has shown that the central pressure waveform could be determined by a very small set of parameters accounting for the physical properties of the heart and the arteries [1]. Particularly, main pressure features like first systolic shoulder (P1) and systolic (P2) pressures were estimated accurately.

Methods: By combining a numerical virtual population (n = 3,325) similar to [2] and experimental data acquired from a pressure/Doppler flow velocity transducer place in the ascending aorta in 18 patients (mean±SD: age 63±11 yr, aortic BP 136±23/73±13 mmHg) at the time of cardiac catheterization, we assessed the accuracy of those predictions for magP1 (P1-DBP) and P2 using respectively a water hammer [3] and a 3-element Windkessel models [4]. Contributions of the heart and arterial properties to these estimates though respectively blood velocity, volume and pulse wave velocity, compliance, resistance were then derived from the theoretical models used.

Results: P1 and P2 estimates agreed well with theoretical pressure both in the ascending aorta in 18 patients (mean±SD: age 63±11 yr, aortic BP 136±23/73±13 mmHg)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>20–29 years</th>
<th>30–49 years</th>
<th>50–69 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>pSBP (mmHg)</td>
<td>118.8 *</td>
<td>103.4</td>
<td>124.8</td>
</tr>
<tr>
<td>pDBP (mmHg)</td>
<td>75.7 *</td>
<td>59.5</td>
<td>81.4</td>
</tr>
<tr>
<td>cSBP (mmHg)</td>
<td>106.3 *</td>
<td>97.1</td>
<td>114.8</td>
</tr>
<tr>
<td>cDBP (mmHg)</td>
<td>77.4 *</td>
<td>60.6</td>
<td>83.1</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>76.0 *</td>
<td>59.6</td>
<td>77.5</td>
</tr>
<tr>
<td>AIx</td>
<td>18.2</td>
<td>19.0</td>
<td>19.7</td>
</tr>
<tr>
<td>AIx75</td>
<td>19.3</td>
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<td>20.6</td>
</tr>
<tr>
<td>RM</td>
<td>55.2</td>
<td>63.2</td>
<td>59.7</td>
</tr>
</tbody>
</table>

Table: Mean day and night values for peripheral systolic blood pressure (pSBP), peripheral diastolic blood pressure (pDBP), central systolic blood pressure (cSBP), central diastolic blood pressure (cDBP), heart rate (HR), augmentation index (AIx, AIx75) and reflection magnitude (RM) * marks a significant difference between day and night (t-test, p<0.05).

Conclusions: Arteries and heart contribute as much to rise in P1. More clinical data are being collected to quantify the contributions of the heart and arteries to P2.

References

References