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### 15.6: INFLUENCE OF THE PRESSURE MEASURING SITE FOR VELOCITY/PRESSURE LOOPS

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that  $C_T$  may be better related with LVM compared to the gold-standard regional aortic stiffness.

**Methods:** Two hundred twenty six subjects with established hypertension (untreated or treated with antihypertensive drugs) or with suspected hypertension underwent blood pressure (BP) assessment, carotid-to-femoral pulse wave velocity (cf-PWV) and echocardiographic measurement of LVM. LVM index (LVMI) was calculated by the ratio of LVM to body surface area.  $C_T$  was estimated by a previously proposed and validated formula:  $C_T = 36.7 / PWV^2$  [ml/mmHg], which is based on Bramwell-Hill equation.

**Results:** LVMI was significantly associated with age ( $r=0.207$ ,  $p=0.002$ ), systolic BP ( $r=0.248$ ,  $p<0.001$ ), diastolic BP ( $r=0.139$ ,  $p=0.04$ ), mean BP ( $r=0.212$ ,  $p=0.002$ ), pulse pressure ( $r=0.212$ ,  $p=0.002$ ), heart rate ( $r=-0.172$ ,  $p=0.011$ ), cf-PWV ( $r=0.268$ ,  $p<0.001$ ) and  $C_T$  ( $r=-0.317$ ,  $p<0.001$ ). The highest correlation was observed for  $C_T$  which was significantly stronger than the respective correlation of cf-PWV ( $p<0.001$ ). Multivariate analysis showed that  $C_T$  was a stronger determinant, compared to cf-PWV, of LVMI and LVH.

**Conclusion:** Total (systemic) arterial compliance is better associated with left ventricular mass and hypertrophy than the cf-PWV. It remains to be further explored whether  $C_T$  has also a superior prognostic value beyond and above local or regional (segmental) estimates of pulse wave velocity.

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#### 15.6

##### INFLUENCE OF THE PRESSURE MEASURING SITE FOR VELOCITY/PRESSURE LOOPS

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**Background:** Velocity/pressure (Vel/P) loops are obtained by combining aortic blood velocity (measured by esophageal Doppler-ED-, CombiQ™, Deltex Medical, Chichester, UK) and arterial pressure signals. They represent a tool to estimate afterload of the heart and arterial stiffness with at least two remarkable angles:  $\beta$  and  $\gamma$ . Pressure is usually measured in the radial artery (PRad) rather than in the descending thoracic aorta (PAoDesc) where ED measures blood flow. Our aims were to assess the influence of the site of pressure recording on the values of  $\beta$  and  $\gamma$  and to develop a mathematical transfer function (TF) to estimate PAoDesc from PRad and then reconstruct Vel/PTFAoDesc loops.

**Methods:** After institutional review board approval (CE SRLF n#17611-356), 15 patients scheduled for elective endovascular neuroradiology were included. Pressures were recorded simultaneously in the radial artery and in the aorta. Vel/PRad and Vel/PAoDesc loops were constructed and compared. A transfer function was estimated using an autoregressive-exogenous (ARX)[1] model to obtain a simulated descending thoracic aorta pressure waveform (PTFAoDesc). The estimation was quantified by the normalized root mean squared error (NRMSE). Vel/PTFAoDesc loops were constructed and compared to Vel/PAoDesc loops.

**Results:** 153 loops were analysed.  $\beta$  and  $\gamma$  angles were systematically lower in the Vel/PRad compared to the Vel/PAoDesc loops ( $36^\circ$  [ $34^\circ - 40^\circ$ ] vs.  $43^\circ$  [ $38^\circ - 48^\circ$ ] for  $\beta$ ,  $11^\circ$  [ $3^\circ - 15^\circ$ ] vs  $25^\circ$  [ $13^\circ - 30^\circ$ ] for  $\gamma$ ,  $p < 0.0001$ ). The ARX model simulated PTFAoDesc with a NRMSE of 93% [ $77 - 96$ ].  $\beta$  and  $\gamma$  obtained with Vel/PAoDesc and Vel/PTFAoDesc were similar and strongly correlated  $\rho = 0.96$ ,  $p < 0.0001$ ) (Fig 1&2)

**Conclusions:** The location where the arterial pressure is monitored has a huge influence on the Vel/P loop parameters. Using a transfer function improves the estimation of the pressure waveform at the site of the Doppler signal.

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#### 15.7

##### STUDY OF WAVE DYNAMICS OF AN EXTRA-AORTIC COUNTERPULSATION DEVICE IN A ONE-DIMENSIONAL COMPUTER MODEL OF THE ARTERIAL SYSTEM

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**Background:** The C-Pulse heart assist system (Sunshine Heart, Inc., Eden Prairie, Minnesota) is a novel extra-aortic counterpulsation device to unload the heart in heart failure patients. Its impact on overall hemodynamics, however, is not fully understood.

**Methods:** The function of the C-Pulse device was implemented in a previously published and validated one-dimensional model of the arterial tree (1). Central and peripheral pressure and flow waveforms with the C-Pulse disabled and activated were simulated for different settings. The results were studied using wave intensity analysis and compared with in-vivo data measured non-invasively in three heart failure patients and with invasive data measured in a pig.

**Results:** In all cases the activation of the C-Pulse showed a diastolic augmentation in the pressure and flow waveforms. The device activation initiates a forward compression wave, whereas a forward expansion wave is associated to the device relaxation, with waves exerting an action in the coronary and the carotid vascular beds. In settings with reduced arterial compliance, the same level of aortic compression demands higher values of external pressure, leading to stronger hemodynamic effects and enhanced perfusion. Computer simulations were in good qualitative agreement with in-vivo observations, but in-vivo effects of the device were stronger. We speculate that besides a direct hemodynamic effect, the C-Pulse action might also induce other adaptive (neuromodulated) mechanisms, not captured by the model.

**Conclusions:** The one-dimensional model may be used as an efficient tool for predicting the hemodynamic impact of the C-Pulse system in the entire arterial tree, complementing in-vivo observations.

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#### 15.8

##### AN EXTENDED ONE-DIMENSIONAL ARTERIAL NETWORK MODEL FOR THE SIMULATION OF PRESSURE AND FLOW IN UPPER AND LOWER LIMB EXTREMITIES

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**Background:** Arterial pulse wave velocity and pulse waveform analysis have become an established component of cardiovascular research. As validation and assessment of devices is not always trivial in an in vivo setting, arterial network computer models may be useful for that purpose. It is, however, mandatory that the model includes sufficient detail, especially when analysing peripheral waveforms.

**Objectives:** To extend the existing 1D arterial network model (103 segments) of Reymond et al. to a more detailed model (143 segments) including the foot and hand circulation (radial and tibial arteries). The arterial tree dimensions and properties were taken from the literature and completed with data from patient scans. The model solves the one-dimensional form of the Navier-Stokes equations over each arterial segment. A non-linear viscoelastic constitutive law for the arterial wall was considered.

**Results:** Comparison of simulations with and without detailed hand and foot circulation demonstrate important differences in waveform morphology in the distal beds. The completed model predicts pressure and flow waves in the hand and foot arteries which are in good qualitative agreement with the published in-vivo measurements. The agreement is especially good for