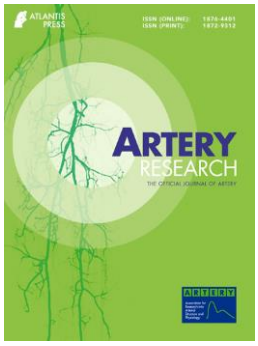

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# P11.6: TOWARDS AORTIC PRESSURE AND FLOW WAVES MODELLING IN THE CLINIC

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vascular effects and are based on population reference data and over-simplified boundary conditions. Because contractile properties of the heart may play a role as well, we investigated by means of a computational model the isolated and combined influences of cardiac properties as well as vascular stiffening on the central BP waveform.
A model of the circulation (Arts et al.2005, AJP-Heart) was used to simulate central and peripheral BP waveforms from the left ventricle (LV) to femoral and radial arteries. We investigated the effect on BP waveforms of 1) a 50% reduction in the shortening velocity (v-s) of LV sarcomeres and 2) a vascular stiffness increase, corresponding to an increase in carotid-femoral pulse wave velocity from 8.6m/s to 10.2m/s. Central BP waveforms were characterized using augmentation index (AIx, based on the 2nd derivative) and pulse pressure (PP).
We obtained realistic BP waveforms for LV, central and peripheral vessels. Reducing v-s (all else equal) caused AIx to increase from respectively 16% (PP=60mmHg) to 30% (PP=60mmHg). Vascular stiffening (all else equal) resulted in an AIx increase from 16% to 36% and an increase in PP from 60 to 100mmHg. Combined reduced v and vascular stiffening resulted in an AIx of 42% with a PP of 80mmHg.
Not only vascular, but also cardiac properties influence the central BP waveform. We conclude that heart-vessel interaction should be considered in pulse wave analysis.

### P11.6
### TOWARDS AORTIC PRESSURE AND FLOW WAVES MODELLING IN THE CLINIC

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Combinations of three-dimensional (3-D), one dimensional (1-D) and lumped parameter (0-D) models have been proposed to model blood flow in vessels. Within the field of 1-D modeling there has been an upward trend in the total number of arterial vessels computed. However, as we increase the spatial dimensions of our models we require larger amounts of clinical data to determine all the model parameters for patient-specific simulations in the clinical setting.
Using a verified 55-vessel, nonlinear, 1-D model of pulse wave propagation in elastic vessels we systematically reduced the number of generations of bifurcations, while preserving the total compliance and net peripheral resistance of the system, to better understand the contributions of multiple reflections at each branching site to the pressure waveform measured along the upper aorta. This was achieved by reducing systematically 1-D model peripheral vessels to three-element 0-D Windkessel models that account for vessel tapering. When applied to the baseline 55-artery model we observed that a reduction in the generations of bifurcations from 5 to 1 resulted in a root-mean-square difference of aortic pressure and flow waveform shape of 0.3% and 17.9% respectively. We further assessed the methodology applied to four adaptations of the baseline model using generalised arterial stiffening, an iliac stenosis, carotid stent or abdominal aortic aneurysm.
Our study shows that a 1-D model can efficiently simulate the aortic pressure and flow waveforms with less than 20 arterial segments.

### P11.7
### THE DECAY OF AORTIC BLOOD PRESSURE DURING DIASTOLE: INFLUENCE OF AN ASYMPTOTIC PRESSURE LEVEL ON THE EXPONENTIAL FIT

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Aortic blood pressure is decreasing approximately exponentially during diastole and the time constant of the decay is supposed to hold information about total arterial compliance and peripheral resistance. However, it is unclear if the pressure would drop to zero without further excitations from the heart or if it would reach an asymptotic pressure level Pinf. The aim of this work was to examine the fitting performance of an exponential decay with and without Pinf in invasive aortic pressure readings with prolonged diastoles caused by missing heartbeats.
A total number of 35 pressure signals (5F Millar SPC-454D catheters) from 5 different subjects were examined. For the fitting procedure, the squared error between measured data and analytical function was minimized with Pinf ranging from 0 to 100% (5% steps) of the diastolic blood pressure DBP. The data was fitted over the duration of both, the regular and the prolonged diastole.
The irregular heartbeats were on average 1.7 (0.3 SD) times longer than the preceding beats. In all settings, mean root mean squared error RMSE between measured and calculated pressure drop was lowest for Pinf=0.7*DBP. For Pinf=0, the deviation was more than two times higher than for Pinf=0.7*DBP, regardless of the chosen part for fitting (mean RMSE: 1.8 (0.6 SD) and 5.2 (2.6 SD) with and without Pinf respectively when fitted to the first part).
The results indicate that an asymptotic pressure level exists, which is, at least for the observed timescales, maintained by the vascular system even without ejection from the heart.

### P11.8
### ARTERIAL ELASTICITY DETERMINATION BY PPG SIGNALS PROCESSING USING PULSE WAVEFORM DECOMPOSITION AND SECOND ORDER DERIVATIVE

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Arterial stiffness is a disease caused by various risk factors and processes. Easy measurements of arterial stiffness may provide information about these processes, but also information regarding the cumulative history of risk factor exposure. To determine the arterial elasticity index we use a combination of red and infrared emitting LED lights which are laid on the surface of the finger/toe nail to measure arterial pulses as light intensity variations. Pulse wave analysis and its decomposition evaluation could be a method for elasticity screening. The pulse wave comprises five different wave components, the incident wave $f_1(t)$ and the reflected waves $f_i(t)$ (i=2-5). The arterial pulse is a envelope which morphology has waves appearing as the four peaks. All the peaks together are the percussion, tidal, dicrotic, repercussion and retidal wave. Of these waves the percussion wave travels from the heart and the other waves travel to the heart. The amplitude and the velocity of the waves also change markedly depending of the arterial elasticity. The second derivative (SD) of the arterial pulse waveform is also a valuable tool in the parallel analysis of pulse waves. Many noninvasive methods for extracting the reflected components from a pulse wave have been proposed in addition to the SD. A new approach is used to estimate the pulse waveform and logarithmic transform of time axis to decompose the waveform into its Gaussian components. This method was applied to subjects aged from their 20s to 69s. The results indicate moderate correlation between age and elasticity index.

### P11.9
### AGE- AND PRESSURE-DEPENDENCE OF PULSE WAVE VELOCITY (PWV): MODEL PREDICTION AND OBSERVATIONS

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**Objective:** PWV(m/s), a gold-standard measure of arterial stiffness, depends on both age and arterial pressure P(mmHg). We evaluated a model-derived expression that enables to separate between these factors.
**Design and method:** A previously reported model assumed that P varies exponentially with the arterial volume V (in relative units): $P=-\alpha+\Upsilon\exp(\beta V)$ [Eq.1], where $\alpha$, $\beta$ and $\Upsilon$ are pressure-independent constants that may depend on age. Arterial stiffness (in mmHg) is defined as the local slope of the P-V curve, i.e. dP/dV [Eq.2] and can be expressed by the Bramwell-Hill equation $7.85\bullet PWV^2$ [Eq.3]. Eqs.1-3 provide the model prediction $PWV^2=0.127\beta(P+\alpha)$ [Eq.4], for which $\beta$ ('stiffness index') quantifies the stiffness pressure-dependence and $\alpha$ is the model-based internal pressure. In 68 healthy subjects (59% men, age 54±17 years, BP 133±20 /76±11 mmHg), we measured brachial BP and carotid-radial PWV in supine position with arm supported at 3 postures: below-, at- and above the heart level to obtain PWV at different DBP levels (the relevant pressure for foot-by-foot analysis method). Parameters $\beta$ and $\alpha$ were best-fitted to Eq.4 using symmetric regression.
**Results:** $PWV^2$ highly correlated with DBP for individuals (mean R=0.95). $\beta$ was 10.6±7.8 (Mean±SD), and was greater for age ≥53 yr (median) than youngers: 14.0±9.0 vs. 7.3±4.3 (P=0.0002). Similarly, $\alpha$ (-3±57 mmHg) was -14±65 vs. 20±41 mmHg (P=0.01). $\beta$ and $\alpha$ were highly correlated non-linearly (R=0.94).