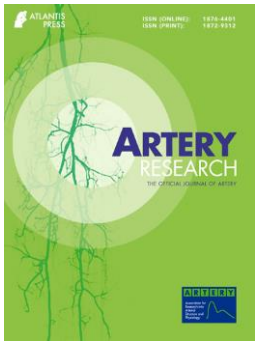

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P11.5: CARDIAC AND VASCULAR TISSUE PROPERTIES DETERMINE THE CENTRAL BLOOD PRESSURE WAVEFORM: CONSEQUENCES FOR PULSE WAVE ANALYSIS

M. Heusinkveld, B. Spronck, J. Lumens, T. Delhaas, A. Hughes, K. Reesink

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that married patients showed better levels of compliance 73.13% than single patients. patients originating from the north showed better compliance (70.45%) than those living in other regions of the Sudan. Disregard to origin most patients lived in desert environments 97.67% of those originating from the North and (66.66%)of these originating from the East).
Conclusion: Patients originating from the East showed lower compliance and therefore must be targeted for health to increase awareness

P11.1
THE PU AND QA LOOP METHODS OVER- AND UNDERESTIMATE LOCAL CAROTID WAVE SPEED: A CONSISTENT EXPLANATION AND SOLUTION TO THE PROBLEM

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Single-point methods such as the PU- and QA-loop methods are used to estimate local pulse wave velocity (PWV-PU and PWV-QA) in arteries from a combination of pressure (P), flow (Q), velocity (U) or cross-sectional area (A) waveforms. Available data indicate that the PU-loop method tends to overestimate PWV, while the QA-loop method tends to underestimate. Wave reflection has been suggested as a factor playing a role in the agreement between different methods. In this work, we (i) demonstrate the interference of wave reflection with the PU-loop method for both solitary sinusoidal waves as well as physiological waveforms; (ii) develop an operator-independent method to correct for the presence of reflections. Fluid-structure interaction simulations in a tube and carotid artery model with known mechanical properties confirm the theory. For the carotid artery model, PWV-PU severely overestimates PWV, while PWV-QA underestimates PWV. Correction (leading to an estimate termed PWV-corr) eliminates the impact of reflections. Finally, methods are applied in vivo in a subsample of the Asklepios population. Compared to PWV-PU and PWV-QA, PWV-corr leads to significantly better correlations of carotid PWV with PWV derived from carotid distensibility based on the Bramwell-Hill equation(with r^2 improving from about 0.25 to 0.91). Neither the PU-loop nor the QA-loop method provides reliable estimates of local PWV in settings where wave reflections are present - even when the PU- or QA-loops show a linear segment. They offer no alternative for the Bramwell-Hill based approach and their application should therefore be discouraged, especially for the carotid artery.

P11.2
A 1D-MODEL FOR THE SIMULATION OF THE ARTERIAL WALL DISPLACEMENT

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Background: Nowadays, a great emphasis has been placed on the modeling of the cardiovascular system. In 1d-models, the arterial diameter is generally deducted from the arterial pulse pressure by considering a stress-strain relationship. However, this assumption remains simplistic in nature since no interaction among elastic layers constituting the arterial wall is considered. Moreover, 3d-models offer generally a better description of the physiology of the arterial wall but are often too complex to be embedded in other 1d-arterial models.
Methods: In the present study, we propose a novel and simple 1d-model to simulate the arterial wall displacement in large arteries. This one relies on a system of coupled differential equations from the interactions among the elastic fibers of the arterial wall and the surrounding tissues. Thereafter, the common carotid arterial wall displacement is reproduced and compared to experimental data obtained from a high-resolution echo tracking ultrasound system in 10 patients.
Results: The model shows a distensibility of the carotid artery (5.6 10−3 mmHg−1 with the simulation) in the same range as observed for experimental data in 10 patients (4.5 10−3 mmHg−1). Moreover, the results suggest that the carotid diameter waveform cannot be directly substituted to the arterial pulse pressure as observed in other 1d-models and differs significantly during the systolic phase.
Conclusions: Subsequently, our model could give a reliable and useful tool for the simulation of the arterial wall displacement which could be easily embedded in other 1d-models treating of arterial system.

P11.3
DEVELOPMENT AND VALIDATION OF REALISTIC AORTIC PHANTOM TAILORED FOR EACH PATIENT

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The microstructure evolution of the aortic tissue in cardiovascular pathologies, such as aneurysm or arthrosclerosis, leads to an overall change of biomechanical properties. Successful treatment (e.g endovascular) of these pathologies depends on the comprehension of these properties and on the surgeon expertise. Many investigators have created general-purpose aortic replicas called "phantoms", for the preoperative training and/or the studies of surgical and radiological processes. However, the importance of the used material properties was generally neglected. Moreover, the specific shape and the mechanical behavior of each patient's aorta were not taken into account. Our work aims to create patient-specific phantoms able to accurately mimic each individual case.
We use a mechanical model comprising both hyperelastic and viscoelastic behaviors which can be scrutinized to predict aneurysm rupture and to diagnose the atherosclerosis, respectively. To identify the model parameters, we performed steady and dynamic ex-vivo experiments. Results were used to develop a large range of materials able to replicate real healthy and pathologic aortic mechanical behavior. For that purpose, different Bluesil® silicone materials from Bluestar Silicones Company were used and suitably formulated. After adjusting the material formulation, the specific aorta shape given by medical imaging is encoded in a finite element model in order to manufacture the specific phantom by 3D prototyping. The whole process results in a quick production of a specific phantom that can be positioned in a hydro-dynamic test bench, in which physiological hemodynamic conditions can be simulated and the model parameters can be verified from ultrasound images and pressure measurements.

P11.4
INVESTIGATION OF THE ARTERIAL AGEING AND ISOLATED SYSTOLIC HYPERTENSION BY FLUID DYNAMICS-BASED MODELLING

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Arterial and left-ventricular ageing strongly affects morbidity and mortality. It is characterized by stiffening, dilation and lengthening of large arteries, microcirculation changes, and alteration of heart contracting capacity and timing. The scientific community is debating the evaluation, impact, and interaction among these fundamental processes. As fluid dynamics play a key-role, our aim is to use a physically-based model of the heart-arterial tree hemodynamics to investigate quantitatively these processes.
Our multi-scale mathematical model considers lumped descriptions of left ventricle, aortic valve dynamics and microcirculatory distal volumes, and the 1D characterization of large-to-medium arteries. Notice that model has been validated in patient-specific settings against a population of six healthy young men. In the present work, starting from parameters statistically representing a healthy young man, the ageing of both heart and arterial tree is simulated by changing the diameter, length, wall thickness and mechanical properties of large arteries, and the left-ventricular force of contraction and its activation time.
Once the main features of the ageing heart-arterial interaction are simulated, our efforts are focused to reproduce the isolated systolic hypertension (ISH) coherently with the most advanced literature data about this pathology. With the aim to elucidate the links between ISH and "healthy" ageing, the key-role of aortic stiffening and remodeling as well as the consequent early-return of reflected pressure wave and the different ventricular ejection pattern are investigated and discussed, paying attention to the physical process identification and understanding.

P11.5
CARDIAC AND VASCULAR TISSUE PROPERTIES DETERMINE THE CENTRAL BLOOD PRESSURE WAVEFORM: CONSEQUENCES FOR PULSE WAVE ANALYSIS

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Various methods exist to estimate central blood pressure (BP) waveforms from noninvasive peripheral BP measurements. Most methods consider only

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 α, β and Υ 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 β ('stiffness index') quantifies the stiffness pressure-dependence and α 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 β and α were best-fitted to Eq.4 using symmetric regression.
Results: PWV^2 highly correlated with DBP for individuals (mean R=0.95). β 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, α (-3±57 mmHg) was -14±65 vs. 20±41 mmHg (P=0.01). β and α were highly correlated non-linearly (R=0.94).