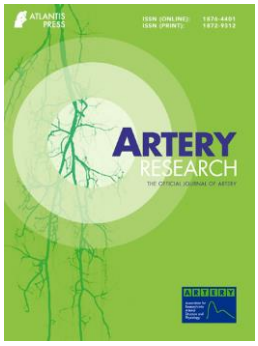

Artery Research

ISSN (Online): 1876-4401 **ISSN (Print): 1872-9312**
Journal Home Page: https://www.atlantis-press.com/journals/artres

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

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To cite this article: B. Gavish, G. Pucci, F. Battista, G. Schillaci (2014) P11.9: AGE- AND PRESSURE-DEPENDENCE OF PULSE WAVE VELOCITY (PWV): MODEL PREDICTION AND OBSERVATIONS, Artery Research 8:4, 162–163, DOI: https://doi.org/10.1016/j.artres.2014.09.227

To link to this article: https://doi.org/10.1016/j.artres.2014.09.227

Published online: 7 December 2019

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).

Conclusions: PWV^2 is superior to PWV for data analysis for its direct association with stiffness and linear dependence on DBP with age- and disease-dependent but pressure-independent coefficients β and α.

P11.10
CAN THE BEHAVIOR OF LIQUIDS UNDER HIGH PRESSURES HELP INTERPRETING STIFFNESS-RELATED MEASURES IN ARTERIES?

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One expression of the fact that real arteries, in the physiological range, are not simple elastic tubes is expressed by increase of its stiffness at greater pressures. Let stiffness G be defined as rate of change of arterial pressure P with size V, i.e. dP/dV, where V stands for volume or cross-section area or diameter, relatively to a reference value. It has been shown that $G(P) = \beta(P+\alpha)$, where β and α are pressure-independent constants and β ('stiffness index') measures the rate of stiffness increase per one unit of pressure change. Both parameters can be determined by measuring stiffness at different pressures. This equation is identical to the so-called Tait equation (Tait, 1888) that describes the 'equation of state' liquids (P-V relationship over thousands of atmospheres) with remarkable accuracy. Although at elevated pressures liquids are compressed while arteries are stretched, in both cases the constituting components change its packing under pressure. This suggests using the knowledge accumulated in high-pressure physics for interpreting stiffness-related measures in arteries. Following this approach it can be shown that arteries behave as elastic tubes only for size changes $\Delta V << 1/\beta$ -a condition that is violated frequently during the systole; the known increase of β with age and vascular pathology may reflect 'enhanced structuring' of the wall components; The parameter α may stand for 'internal pressure' contributed, in part, by the net attraction/repulsion of wall elements, independently of the applied pressure. In conclusion, stiffness-related measures may probe the physical state of the arterial wall microstructure.

P11.11
VENOUS VALVES DYNAMICS AND THE HEMODYNAMICS OF THE MUSCLE PUMP EFFECT: A MODELING APPROACH

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Orthostatic intolerance is observed in astronauts after spaceflight, in patients with spinal cord injury and in the elderly. Their inability to compensate for the gravity-induced blood volume shift towards the legs in upright position can result in critical events as syncope. Normally, the muscle pump effect plays a crucial role in proper regulation of the fluid distribution. Leg muscle contraction increases venous return by collapsing deep veins while distal valves close. During muscle relaxation venous refilling is fastened as perfusion pressure is increased due to pressure shielding by the proximal valves. Furthermore, the connected superficial veins, which are less affected by muscle contraction, serve as an extra reservoir during venous refilling. Unfortunately, this complex physiological mechanism, in particular the contribution of deep and superficial veins in blood volume shift, remains poorly understood.
Therefore, the objective of this study is to characterise the muscle pump effect using a 1D pulse wave propagation model of the venous system including venous collapsibility, hydrostatic pressure and venous valves. A four-second muscle contraction has been simulated in a configuration connecting a deep to a superficial vein via four perforating veins.
Muscle contraction resulted in increased venous return and distal valve closure. Furthermore, increased perfusion was observed during relaxation and the superficial veins contributed to venous refilling.
In summary, the model can qualitatively reproduce the local muscle pump effect. Future work will focus on extending the model with regulation mechanisms and a closed loop circulation, which can ultimately result in increased insight in orthostatic intolerance.

P11.12
SIMPLIFICATION OF A NON-LINEAR MECHANICAL MODEL OF HUMAN COMMON CAROTID ARTERY WITH SENSITIVITY ANALYSIS

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Background: We described a model allowing in vivo non-linear mechanical characterization of human common carotid arteries (CCAs) (Masson 2011). It relied on 14 different parameters corresponding to geometric properties (number 1 to 3), fibrillar constituents (n. 4 to 9), perivascular parameters (n. 10 and 11) and contractile properties (n. 12 to 14). Because of the non-linearity and the high number of parameters of the objective function, convergence towards an optimal solution is difficult. We propose here to quantify the contribution of each parameter for optimizing the model.
Methods: We studied 58 subjects. The 14 parameters were first determined using Masson's method, then each parameter was independently changed by +/- 1, 5 and 15%. Changes in objective function were computed and compared. Parameters were ranked.
Results: Geometric parameters contributed the most (rank 1, 2 and 3), followed by contractile components. The elastic component parameters contributed the least to the energy function. The ranking depended on the amplitude of imposed variation, especially for geometrical parameters. Some fibrillar parameters (7 and 9) had marked contribution for negative imposed change (rank 2 and 4) but not for positive one (rank 13 and 14). Convergence of modelling was more reliably obtained when using stepwise procedures based on the ranking than with standard least-square procedures.
Conclusion: The contribution of parameters in large artery energy function is unequal and stepwise introduction of parameters improves the optimization procedure. Reduction in the number of parameters might be made possible by a smart selection of the parameters.