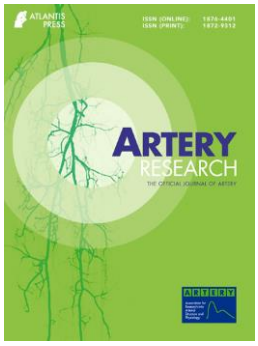

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15.8: AN EXTENDED ONE-DIMENSIONAL ARTERIAL NETWORK MODEL FOR THE SIMULATION OF PRESSURE AND FLOW IN UPPER AND LOWER LIMB EXTREMITIES

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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: β and γ. 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 β and γ 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. β and γ angles were systematically lower in the Vel/PRad compared to the Vel/PAoDesc loops (36 ° [34 ° − 40 °] vs. 43 ° [38 ° − 48 °] for β, 11 ° [3 ° -15 °] vs 25 ° [13 ° -30 °] for γ, $p < 0.0001$). The ARX model simulated PTFAoDesc with a NRMSE of 93% [77 − 96]. β and γ 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

the shape and wave details of the flow wave, where all features are reproduced in a rather faithful manner.
Conclusions: The extended model yields realistic pressure and flow waveforms in arteries of the hand and the foot. After full validation, this extended model will be used to assess the performance of diagnostic and screening devices relying on peripheral hemodynamics signals, such as the pOpmètre®.
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15.9
MODELLING ARTERIAL PULSE PRESSURE FROM HEART RATE DURING SYMPATHETIC ACTIVATION

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Background: The duration of the time segment between the systolic (SP) and post-dicrotic notch peak pressures (PDP) of the arterial pressure wave in humans has been proposed to be related to arterial pulse pressure (PP).[1,2] We considered an effect of RR-interval length on the diastolic pressure run-off and tested the hypothesis that heart rate (HR) affects the timing of systolic and post-dicrotic notch peak pressures.
Methods: We modelled the effects of sympathetic stimulation by progressive central hypovolemia on PP changes based on morphological features of a peripherally measured arterial blood pressure wave shape and HR, making use of linear mixed effect (LME) models. Changes of the arterial pulse wave were tracked from rest towards central hypovolemia in 44 subjects by exposing them to continuous −50 mmHg lower body negative pressure (LBNP). SP and PDP, and HR were extracted from arterial finger pressure and used as model input to predict PP.
Results: From rest to sympathetic stimulation, HR (30%) and thoracic impedance (15%) increased and systolic (SAP) fell by 10%. Model errors of PP (median, and 1st and 3rd quartiles) were 5.2 [3.3 8.9], 4.9 [3.8 7.7], and 4.9 [3.7 8.6] for LME models of, respectively, SP-PDP, HR and their combination.
Conclusion: Our study highlights that during sympathetic stimulation by progressive central hypervolaemia, HR affects arterial pressure wave characteristics and that linear models from both HR and SP-PDP duration allow for estimating PP.
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15.10
FORM FACTOR OF THE FEMORAL ARTERY: AN INVASIVE STUDY

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Background: There is a growing interest in precisely estimating mean blood pressure (MBP) in large arteries. The form factor (FF) is the fraction of pulse pressure that must be added to diastolic pressure to estimate the actual MBP, i.e., the pressure integrated (averaged) over the whole cycle. It is admitted that FF of the radial artery is 0.33, while FF of the aorta and carotid and brachial arteries is in the 0.40-0.45 range. The FF of the femoral artery remains to be determined.
Methods: Sixty-five hemodynamically stable intensive care unit patients equipped with an indwelling femoral catheter were prospectively studied (mean age+/-SD = 64+/-14 years). FF of the femoral artery was calculated as the time-averaged MBP minus diastolic blood pressure difference divided by pulse pressure (FF = (MBP − DBP) / PP).
Results: Form factor of the femoral artery was 0.35+/-0.04 (n=65 range 0.22-0.47). FF was similar in female (n=23) and male (n=42) patients (0.36+/-0.05 vs 0.34+/-0.04, respectively) and in patients receiving vasopressors (n=43) or not (n=22) (0.34+/-0.05 vs 0.35+/-0.03, respectively). FF of the femoral artery was positively related to MBP (r^2=0.11) and DBP (r^2=0.07) (each $P < 0.05$) while it was not related to patient's age, body height, body weight, heart rate, systolic pressure and PP.
Conclusions: The mean form factor of the femoral artery was 0.35, a value closer to the FF of radial artery than to the FF of central and brachial arteries. The implications for pressure wave transmission to the lower limbs remain to be studied.

15.11
TOWARDS NONINVASIVE CARDIAC CATHETERISATION

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Background: Doppler echocardiographic measures of diastolic function, such as E/e' are correlates of left ventricular (LV) end-diastolic pressure (p_{ed}) and diastolic compliance (C_d) [1]. We developed a noninvasive computational approach to obtain these essential markers of LV diastolic abnormalities and tested it against the invasive gold standard.
Methods: In patients undergoing coronary angiography (n=8, age 60+/-13yrs, with no atrial fibrillation or other dysrhythmia), we obtained mitral and aortic valve Doppler tracings, LV wall thickness and cavity volumes, brachial systolic and diastolic blood pressure (BP) and, for validation purposes, LV pressure and volume invasively by conductance catheter. Repeated echocardiography and BP measurements were performed at baseline conditions and averaged. Catheter measurements were performed during baseline and Valsalva manoeuvre. The latter causes a change in LV preload, enabling a robust estimation of C_d. We fitted a computational model describing the cardiovascular circulation (CircAdapt,www.circadapt.org) to the noninvasively measured data. Catheter measurements served as a reference to validate model-predicted p_{ed} and C_d.
Results: Catheter-measured p_{ed} was found to be 21+/-6mmHg (mean+/-SD, n=8) and C_d was 3.1+/-3.0ml/mmHg (n=6). The bias and limits of agreement between the model-estimated and catheter-measured p_{ed} and $C_d$were -0.9+/-7.5mmHg and 1.1+/-2.6ml/mmHg, respectively.
Conclusions: We found reasonable agreement between our noninvasive modelling-based method of estimating p_{ed} and C_d and catheter measurements. Due to its noninvasiveness, our method could be useful for detection of LV diastolic abnormalities in more patients and settings. Next, we will investigate how measurement errors propagate into the uncertainty of model predictions of p_{ed} and C_d.
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