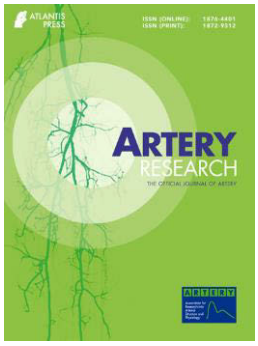

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3.6: NON-INVASIVE, MRI-BASED ESTIMATION OF PATIENT-SPECIFIC AORTIC BLOOD PRESSURE USING ONE-DIMENSIONAL BLOOD FLOW MODELLING

Jorge Mariscal Harana, Arna van Engelen, Torben Schneider, Mateusz Florkow, Peter Charlton, Bram Ruijsink, Hubrecht De Bliek, Israel Valverde, Marietta Carakida, Kuberan Pushparajah, Spencer Sherwin, Rene Botnar, Jordi Alastruey

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(n = 30) was also associated with gothic arch (p = 0.01), and dilated ascending aorta but with no aortic root dilation (p = 0.02).
On multivariate regression analysis, gothic arch was indeed associated with coarctation and stenosis, and also with non-coronary valve fusion pattern (p = 0.03). Patients with aortic regurgitation tended to have larger aortas (p = 0.005).
Conclusion: The presence of aortic coarctation and stenosis may influence the amount of dilation and the overall arch architecture in BAV patients. Patients with BAV present profoundly different morphological phenotypes depending on the presence/absence of aortic coarctation (Fig. 1).

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3.4
RESERVOIR PRESSURE SEPARATION AT BRACHIAL, CAROTID AND RADIAL ARTERIES: A QUANTITATIVE COMPARISON AND EVALUATION

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Background: At present, reservoir pressure parameters are derived from arterial pressure waveforms regardless of the location of measurement. However, a comparison between sites has not been made, and site-related differences may affect interpretation. In this study, we computed reservoir pressure waveform separations on hypertensive individuals where brachial, carotid and radial pressure measurements were available and quantitatively assessed their results.
Methods: 95 participants in the Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT) had sequential measurements of pressure and flow velocity waveforms from carotid, brachial and radial arteries [1]. Pre-processing was performed to impose identical diastolic and mean blood pressures at all three arterial locations. Using pressure information only, reservoir pressure separation was performed [2, 3]. Systolic durations were estimated based on minimum pressure waveform derivatives.
Reservoir curves characterized by physiologically implausible parameters, i.e. a rate constant $b < 0$ or an asymptotic pressure $P\infty < 0$, were discarded, leaving 74 subjects with valid reservoir pressure waveforms at all three arterial locations.
Results: Estimated reservoir parameters are shown in Table 1. We observed significant differences between arteries in almost all parameters. A high correlation was observed between reservoir pulse pressure and reservoir pressure area at all locations, and the correlation between brachial and radial arteries was stronger for all parameter.
Conclusions: The results of this study indicate differences in parameters derived from reservoir pressure separation at different arterial locations. This suggests that interpretations cannot be made agnostic to the location of measurement.

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Table 1 Quantification of reservoir pressures at three arterial locations in the format of mean ± standard deviation based on 74 subjects whereby PP denotes the reservoir pulse pressure, A_p the area of reservoir pressure above diastolic blood pressure, P_∞ the asymptotic blood pressure and a, $b = 1/\tau$ the rate constants with the time constant τ describing the diastolic pressure decay. The correlation coefficient r is computer between relevant arterial locations. The statistical significance of the differences between locations was based on a paired t-test with * indicating $p < 0.05$.

Reservoir	Brachial Artery(B)	Carotid Artery(C)	Radial Artery(R)	*r*(B,C)	*r*(B,R)	*r*(C,R)
PP [mmHg]	37.1 ± 8.6	41.6 ± 9.0	36.1 ± 8.4	0.84*	0.95*	0.84*
A_p [mmHg s]	16.7 ± 5.0	19.0 ± 4.4	16.0 ± 4.3	0.91*	0.96*	0.91*
P_∞ [mmHg]	61.6 ± 14.2	66.6 ± 12.8	66.2 ± 11.2	0.50*	0.51*	0.46*
A [1/s]	8.3 ± 3.7	11.4 ± 2.7	7.0 ± 2.7	0.11*	0.91*	0.18*
B [1/s]	1.8 ± 0.6	2.2 ± 0.9	2.1 ± z0.7	0.30*	0.62*	0.40*

3.5
HEART RATE DEPENDENCE OF REGIONAL AND LOCAL AORTIC PULSE WAVE VELOCITY IN RATS AS A FUNCTION OF BLOOD PRESSURE

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Background: Pulse wave velocity (PWV) is quantified by time difference of arrival of the blood pressure (BP) wave at two sites along the arterial bed (transit time; TT-PWV), or by combining measured arterial pressure and diameter using the Bramwell-Hill equation (BH-PWV). Besides the dependence of PWV on BP, TT-PWV also depends on heart rate (HR). The present study aimed to also quantify the dependence of BH-PWV's on HR, as a function of diastolic BP (DBP).
Methods: Adult anaesthetised rats (n = 24) were randomly paced at 300–500 bpm, at 50-bpm steps. At each step, aortic TT-PWV (two pressure-tip catheters) and BH-PWV (pressure-tip catheter and ultrasound wall-tracking; abdominal aorta) were measured simultaneously, across a pharmacologically induced DBP range of 60–110 mmHg.
Data from 9142 heart beats was analysed using mixed-effects modelling.
Results: HR dependence of TT-PWV increased from 0.03 m/s/100 bpm at DBP = 60 mmHg to 0.06 m/s/100 bpm at DBP = 110 mmHg (both $p \leq 0.023$). HR dependence of BH-PWV was 0.11 m/s/100 bpm at DBP = 60 and 85 mmHg, but paradoxically decreased to 0 at DBP = 110 mmHg ($p = 0.686$). This decrease in dependence is explicable in that standard BH-PWV uses an approximate derivative of pressure to diameter, which overestimates PWV with increasing pulse pressure (PP). PP decreases as HR increases, potentially causing a BH-PWV decrease with HR. This effect can be overcome by estimating the full pressure-diameter curve for each HR, and calculating the true derivative at DBP, yielding a BH-PWV that no longer shows significant HR dependence ($p \geq 0.076$ at all DBPs).
Conclusions: BH-PWV and TT-PWV show a different HR dependence, affected by DBP.

3.6
NON-INVASIVE, MRI-BASED ESTIMATION OF PATIENT-SPECIFIC AORTIC BLOOD PRESSURE USING ONE-DIMENSIONAL BLOOD FLOW MODELLING

Jorge Mariscal Harana [1], Arna van Engelen [1], Torben Schneider [2], Mateusz Florkow [1,2], Peter Charlton [1], Bram Ruijsink [1], Hubrecht De Bliek [3], Israel Valverde [1], Marietta Carakida [1], Kuberan Pushparajah [1],

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Background and objectives: Clinical evidence shows that central (aortic) blood pressure (CBP) is a better marker of cardiovascular risk than brachial pressure [1]. However, CBP can only be accurately measured invasively, through catheterisation. We propose a novel approach to estimate CBP non-invasively from aortic MRI data and a non-invasive peripheral (brachial) pressure measurement, using a one-dimensional (1-D) model of aortic blood flow.
Methods: We created a population of virtual (computed) subjects, each with distinctive arterial pulse waveforms available at multiple arterial locations, to assess our approach. This was achieved by varying cardiac (stroke volume, cardiac period, time of systole) and arterial (pulse wave velocity, peripheral vascular resistance) parameters of a distributed 1-D model of the larger systemic arteries [2] within a wide range of physiologically plausible values. After optimising our algorithm for the aortic 1-D model in silico, we tested its accuracy in a clinical population of 8 post-coarctation repair patients.
Results: Results from our in silico study, after varying cardiac and arterial parameters by ±30%, showed maximum relative errors for systolic, mean and diastolic CBP of 4.5%, 3.6% and 4.2%, respectively. Average relative errors for systolic, mean and diastolic CBP were 2.7%, 0.9% and 1.2%, respectively. Corresponding average relative errors from our clinical study were 5.4%, 1.5% and 8.0%.

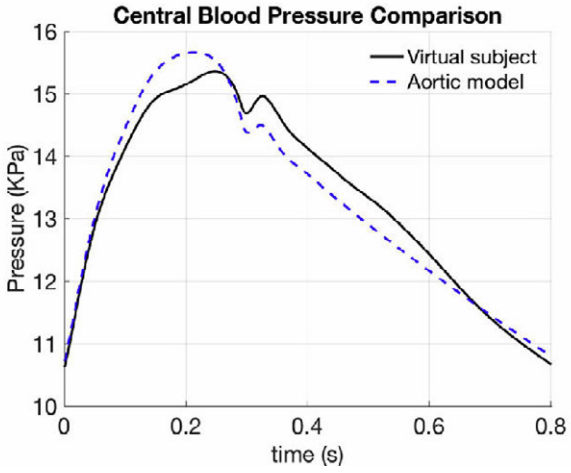

Figure 1 CBP estimation using the aortic 1-D model for a given virtual patient.

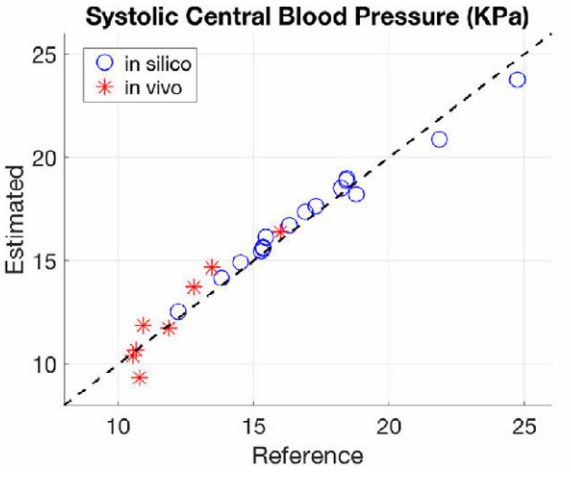

Figure 2 Systolic CBP estimated using the aortic 1-D model against reference systolic CBP values from *in silico* and *in vivo* data.

Conclusions: We have provided a proof of concept for the non-invasive estimation of patient-specific central blood pressure using computational aortic blood flow modelling in combination with MRI data and a non-invasive peripheral pressure measurement.
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3.7
CHANGES OF INTRINSIC STIFFNESS OF THE CAROTID ARTERIAL WALL DURING THE CARDIAC CYCLE MEASURED BY SHEAR WAVE ELASTOGRAPHY IN HYPERTENSIVES COMPARED TO NORMOTENSIVES

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Objective: Because measurement of arterial stiffness is highly dependent on blood pressure (BP), methods independent of BP are required. Shear wave elastography (SWE, Supersonic Imagine, Aix-en-Provence, France) enables to assess local tissue stiffness by tracking the propagation of shear waves generated into the tissue using ultrafast imaging. This method has never been tested against classical Echotracking (Artlab, Esaote, Maastricht, NL) and carotid to femoral pulse wave velocity (cf-PWV, Sphygmocor, AtCor, Sydney, Australia).
Methods: We included 25 subjects, 14 normotensives (NT) and 11 essential hypertensives (HT), matched for age and sex. We optimized SWE algorithms for carotid wall tracking and shear wave group velocity calculation for the anterior (a-SWV) and posterior wall (p-SWV). 8 ultrasonic pushes were triggered at intervals of 200 ms to study the variations of stiffness during the cardiac cycle.
Results: p-SWV showed no association with carotid PWV, cf-PWV nor BP. Mean a-SWV over the cardiac cycle was strongly associated with carotid PWV measured by Echotracking ($r = 0.56$, $p = 0.003$) and cf·PWV ($r = 0.66$, $p < 0.001$). a-SWV strongly increased with BP level during the cardiac cycle ($p < 10^{-6}$). Similar associations between a-SWV and BP were found in NT and HT although HT had higher values of a-SWV throughout all BP levels. However, when a common BP value (100 mmHg) was considered, no significant difference was found between NT and HT.
Conclusion: We have demonstrated with a method independent of BP that the increased arterial stiffness in HT is entirely due to the BP increase. SWE seams a promising technique for assessing arterial stiffness.

3.8
IMPLEMENTING FLUID-STRUCTURE INTERACTION COMPUTATIONAL AND EMPIRICAL TECHNIQUES TO ASSESS HEMODYNAMICS OF ABDOMINAL AORTIC ANEURYSMS

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An Abdominal Aortic Aneurysm (AAA) represents a degenerative disease process of the abdominal aorta that leads to a focal dilation and irreversible remodeling of the arterial wall [1].
The reliable assessment of AAA rupture risk in a clinical setting is crucial in decreasing related mortality without needlessly increasing the rate of surgical repair. Currently there is no accepted technique to quantify the risk of rupture for individual AAAs. Elective repair decisions are generally founded on the "maximum diameter criterion" [2].
A multi-disciplinary approach including constitutive modeling and vascular biomechanics is required to increase the effectiveness in assessing and treating the disease.
Guidelines for treatment of AAAs from the Society for Vascular Surgery indicate computationally acquired rupture predictors need additional