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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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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 $\pm 30\%$, 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%.

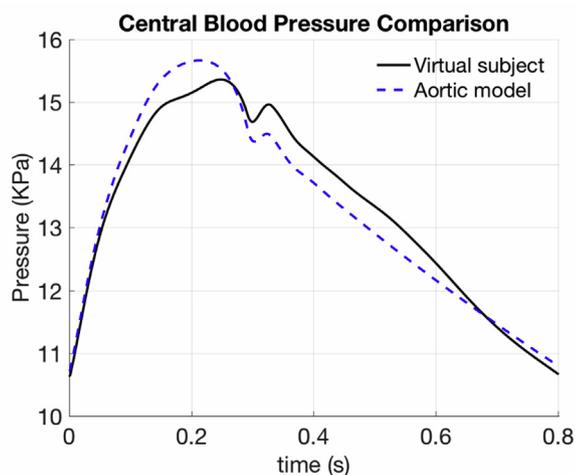


Figure 1 CBP estimation using the aortic 1-D model for a given virtual patient.

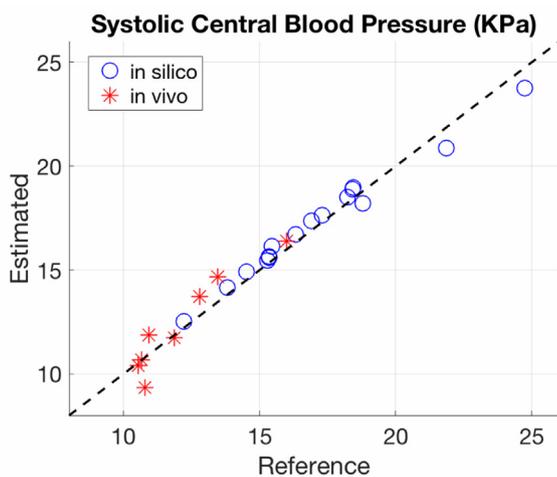


Figure 2 Systolic CBP estimated using the aortic 1-D model against reference systolic CBP values from *in silico* and *in vivo* data.

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.

References

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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 seems a promising technique for assessing arterial stiffness.

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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