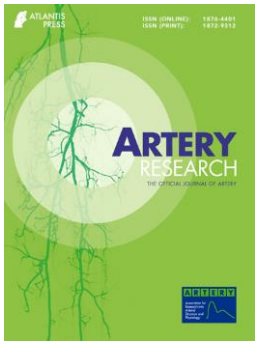

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3.8: IMPLEMENTING FLUID-STRUCTURE INTERACTION COMPUTATIONAL AND EMPIRICAL TECHNIQUES TO ASSESS HEMODYNAMICS OF ABDOMINAL AORTIC ANEURYSMS

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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%.

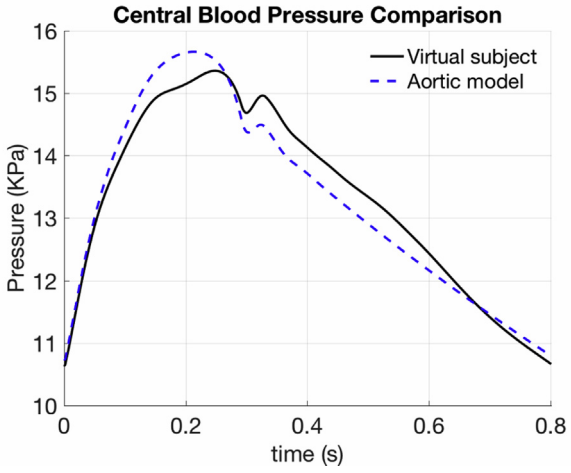

Figure 1 CBP estimation using the aortic 1-D model for a given virtual patient.

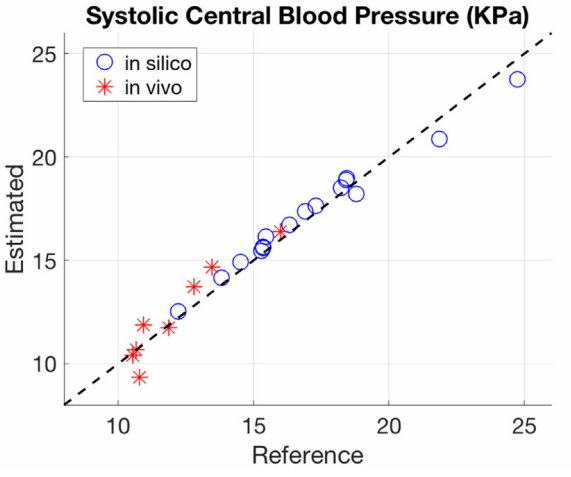

Figure 2 Systolic CBP estimated using the aortic 1-D model against reference systolic CBP values from *in silico* and *in vivo* data.

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

validation prior to their implementation in clinics. For this purpose, silicone replicas of anatomically realistic geometries of AAAs are fabricated and the flow field in the aneurysmal region is experimentally measured *in vitro*, using time-resolved volumetric Particle Image Velocimetry (PIV) [3–4]. Furthermore, the experimental setup allows for strain measurements of the aneurysmal wall to be taken simultaneously using Digital Image Correlation (DIC). These data are used to validate concurrent computational simulation results and FSI analyses. The results demonstrate that the FSI computational approach can predict the patterns of flow from the PIV measurements, which arise from the geometry of the AAA. This work highlights that empirical and computational modelling can complement each other to investigate AAA development towards our goal of producing validated computational simulations that can be used for diagnostic purposes.

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Oral session IV — Clinical Aspects

4.1
PILOT STUDY ON THE PRECLINICAL VASCULAR DAMAGE IN BOLIVIAN PATIENTS WITH CHAGAS INDETERMINATE CHRONIC PHASE

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Background: In Italy, the prevalence of seropositivity for *Trypanosoma cruzi* in immigrants from endemic countries is about 11.3% (30.7% for Bolivian immigrants).
The disease acute phase is usually asymptomatic, often leading to chronic infection that may remain silent for life (chronic indeterminate phase). Chagas heart disease is the most severe and frequent (20–30%) form of chronic phase; its pathophysiology shares similar mechanisms with the arterial impairment associated to other diseases (diabetes, hypertension, aging), leading to chronic inflammation and stiffening. In literature there are no data about the possible elastic arteries deterioration in Chagas disease. Our hypothesis was that early arterial compliance modifications might be found in the chronic indeterminate phase of Chagas disease.
Methods: 35 consecutive Bolivian subjects (21 with indeterminate Chagas disease, mean age [SD] 44.2 [8.2], 5 women, and 14 controls, mean age 40.2 [8.2], 5 women) accessing the service of Tropical Medicine were enrolled. Staging of the disease, laboratory assay, and hemodynamics (central and peripheral blood pressure [BP], aortic pulse wave velocity [PWV], carotid intima media thickness, cardiac ultrasound) were assessed.
Results: No clinical nor laboratory differences were found between the cases and controls. Peripheral and central BPs components were similar. Chagas patients presented higher PWV than controls (7.87 ± 1.29 vs 6.43 ± 1.12 m/s, $p = 0.002$), even when adjusting for age, mean BP, heart rate, body mass index, smoking status ($p = 0.001$).
Conclusion: Patients with Chagas indeterminate chronic phase presented higher arterial stiffness than controls, pointing out an early arterial involvement as the possible etiological mechanism underlying the increased cardiovascular risk in these patients.

4.2
SEX-DEPENDENT EFFECTS OF PERIVASCULAR ADIPOSE TISSUE ON VASCULAR FUNCTION

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Background: Premenopausal women are relatively protected against hypertension compared to males. Estrogen levels have been identified as a potential underlying cause, but the pathophysiological mechanisms remain incompletely understood. We hypothesised that sex-dependent effects of perivascular adipose tissue PVAT mediate altered vascular function in hypertension.
Methods: The effect of PVAT was investigated on resistance vessels of 16 week old male and female stroke-prone spontaneously hypertensive rats (SHRSP).
Results: Wire-myography was used on 3rd-order mesenteric vessels (maximum contraction: male +PVAT 113.3 ± 1.1 vs. female +PVAT 91.4 ± 11.36 %). Noradrenaline mediated vasoconstriction was increased in SHRSP males compared to females. K_{ATP} channel-mediated vasorelaxation by cromakalim was impaired in males compared to females (maximum relaxation: male +PVAT 46.9 ± 3.9 % vs. female +PVAT 97.3 ± 2.7 %) A cross-over study assessing function of male PVAT on female vessels and vice versa confirmed the reduced K_{ATP} mediated vasorelaxation induced by male PVAT (maximum relaxation: female $+PVAT_{female}$ 90.6 ± 1.4 % vs. female $+PVAT_{male}$ 65.8 ± 3.5 %). An adipokine array with subsequent western blot validation identified resistin as a potential modifier of vascular reactivity. Resistin was increased by approximately 2-fold in SHRSP male PVAT. Male and female vessels pretreated with resistin (40 ng/ml) showed no difference in response to noradrenaline. However, vasorelaxation in response to cromakalim was significantly impaired in resistin treated female vessels, similar to levels observed in male vessels (maximum relaxation: female +PVAT 97.3 ± 0.9 % vs. female +PVAT +resistin 36.8 ± 2.3 %).
Conclusion: We identified a novel role for resistin in sex-dependent PVAT mediated vascular function in hypertension through a K_{ATP} channel mediated mechanism.

4.3
ABNORMAL PRESSURE WAVE REFLECTION ACCELERATES THE DEVELOPMENT OF HYPERTENSION VIA THE INCREASE OF ARTERIAL STIFFNESS

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Objectives: It is noted that not only arterial stiffness but also abnormal pressure wave reflection are risks for the development of hypertension. However, the association between arterial stiffness and pressure wave reflection in the development of hypertension has not been fully clarified. The present study was conducted to examine whether the abnormal pressure wave reflection accelerates the development of hypertension via the increase of arterial stiffness.
Methods: In 3102 middle-aged healthy Japanese men without hypertension at baseline, systolic/diastolic blood pressures, brachial-ankle pulse wave velocity (baPWV), and radial augmentation index (rAI) were annually measured during a 9-year study period.
Results: In multivariate linear regression analysis and in mixed model linear regression analysis, baPWV was not longitudinally associated with rAI. Linear regression analysis demonstrated that the higher rAI at the baseline was associated with the larger longitudinal increase of baPWV ($beta = 0.17$, $p < 0.01$). At the end of study period, 404 subjects were developed to hypertension. The prevalence rate of the development of hypertension during the study period was higher in subjects with higher baPWV and higher rAI at the baseline (220 in 939 subjects: 23%) than that in other 3 groups classified by the status of baPWV and rAI at the baseline (e.g. 52 in 942 subjects with low baPWV combined with low rAI: 6%, $p < 0.01$).
Conclusion: The abnormal pressure wave reflection, which may be derived from both arterial stiffness and peripheral vascular damages, may be an accelerator for the development of hypertension via the increase of arterial stiffness.