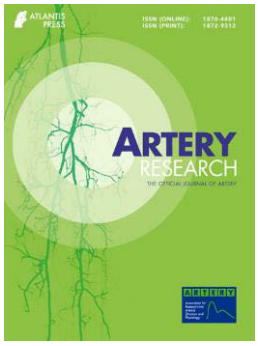

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03.03: VALIDATION OF A 1D MODEL OF THE SYSTEMIC ARTERIAL TREE INCLUDING THE CEREBRAL CIRCULATION

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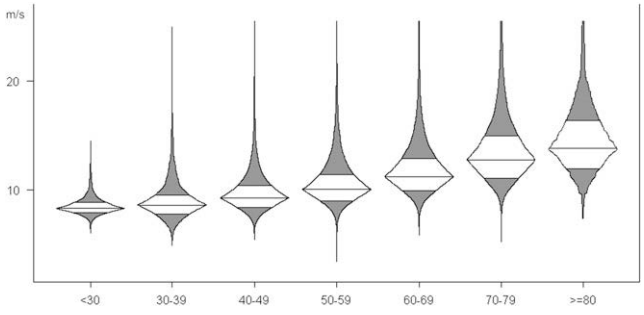

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03.01

ANALYSIS OF ARTERIAL WAVE REFLECTION PATTERNS IN A PATIENT-SPECIFIC HYDRAULIC BENCH MODEL OF THE HUMAN FOREARM

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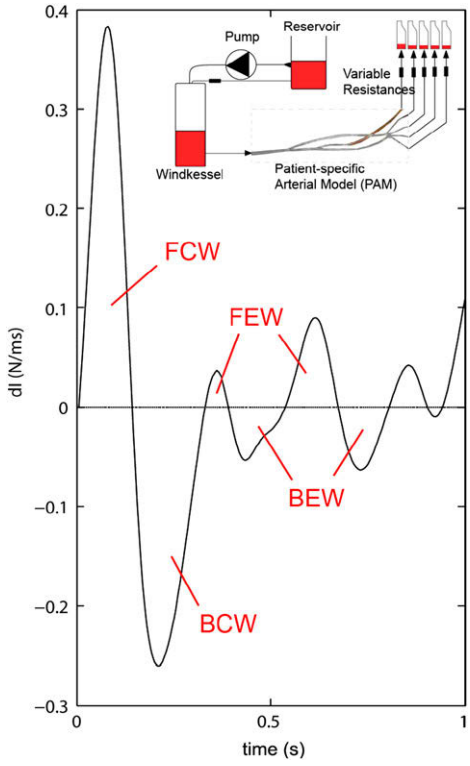

The complex branching topology of the local vascular bed contributes to the complex nature of the blood pressure and flow in the human forearm. The aim of this work is to develop a full scale hydraulic bench model within the framework of our research into arterio-venous fistula creation and the relationship between blood pressure in the upper arm and the wrist. A silicon 3D-model of the brachial, ulnar, radial and anterior interosseous artery completed with the palmar arch was constructed in full scale. The geometry was based on patient-specific functional measurements and MR-data. The Patient-specific Arterial Model (PAM) was built in a mock loop consisting of an upstream reservoir, a pulsatile pump, a windkessel and variable resistances downstream (Figure). 7.5% of the blood mimicking fluid (water-glycerine mixture) was flowing to the interosseous, while the remaining was split equally over the four outflow paths in the palmar arch. Wave Intensity Analysis (WIA) was performed to assess wave reflection patterns in the model. A typical WIA pattern at the brachial inlet is shown in the figure. The initial forward compression wave (FCW) generated by the heart is distally reflected and returns as a backward compression wave (BCW), whereas the subsequent forward expansion wave (FEW) can be interpreted as the open end-type reflection of this BCW. The FEW is on his turn reflected downstream in the backward expansion wave (BEW). The in-vitro PAM shows the complexity of the wave reflections and will be used to study more specific flow problems in the forearm.

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03.02

EVIDENCE OF A ''COMMON'' RESERVOIR PRESSURE TRANSMITTED ALONG THE LENGTH OF THE AORTA WHICH IS THE PREDOMINATE DETERMINATE OF ARTERIAL PRESSURE IN HUMANS

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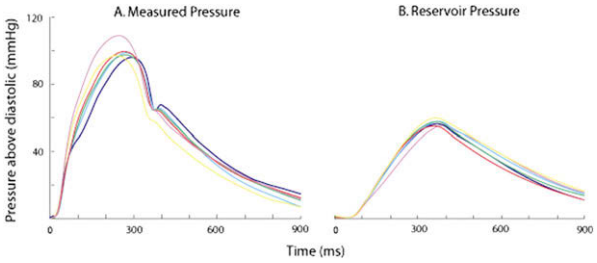

Background: Despite the large variation in pulse pressure waveform throughout the aorta, the diastolic decay of the pressure waveform is almost identical. We hypothesise that this is because there is a common reservoir pressure along the entire aorta, principally determined by the highly elastic proximal aortic root. We apply a new technique to calculate this reservoir pressure along the aorta to test this hypothesis.
Method and Results: Using intra-arterial wires, pressure and Doppler velocity were measured at 10cm intervals along the aorta in 16 patients (aged 55±11 years). Pressure was separated into reservoir and wave components using the new wave-reservoir technique. In all patients, the intra-subject reservoir pressure waveforms were almost identical (mean correlation coefficient 0.99±0.01) regardless of the marked changes in the measured pressure waveform (systolic pressure $p=0.020$ and pulse pressure $p=0.001$). Significant variation in reservoir pressure was seen between subjects (peak reservoir = 63.4 − 21.4 mmHg). The reservoir pressure was the predominate determinant of the pressure waveform and accounted for 67.0 ± 8.8% of the total integrated pulse pressure across all aortic sites.
Conclusions: The aortic pressure waveform is predominately determined by the reservoir pressure. This reservoir pressure is similar along the length of the aorta despite marked changes in the shape of the measured pressure waveform. Manipulation of the arterial reservoir, rather than wave-reflection sites may be more important in regulation of blood pressure control.

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03.03

VALIDATION OF A 1D MODEL OF THE SYSTEMIC ARTERIAL TREE INCLUDING THE CEREBRAL CIRCULATION

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The aim of this study is to develop and validate a distributed model of the systemic arterial tree, coupled to a heart model and including a detailed description of the cerebral arteries. No model has been developed so far that offers a physiologically relevant coupling to the heart and includes the entire cerebral arterial tree.
The 1D forms of the continuity and momentum equations are applied over tapered arterial segments. The intimal shear stress is modeled using the Womersley theory. A non-linear viscoelastic constitutive law for the arterial wall is considered. The arterial tree is coupled to the heart, which is modeled using the time varying elastance model. All distal vessels are terminated with three-element windkessel. Coronary arteries are modeled assuming a systolic flow impediment dependent on the varying elastance of the ventricles. The systemic

arterial tree dimensions and properties were taken from literature and extended to include the cerebral arterial tree obtained from real patient scans. To validate model predictions, we performed noninvasive measurements of pressure (applanation tonometry) and flow (ultrasound and MRI) waves in volunteers.
The model predicts pressure and flow waves which are in good qualitative agreement with in-vivo measurements, especially for the shape and wave details, where all features are reproduced in a rather faithful manner. The results obtained validate the model predictions of pressure and flow in central arteries as well as in major arteries of the brain, reinforcing thus the general applicability of the 1D model to the entire systemic and cerebral circulation.

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03.04
MECHANICAL AND STRUCTURAL CHARACTERISTICS OF CAROTID PLAQUES: ANALYSIS BY MULTI-ARRAY ECHOTRACKING SYSTEM AND MRI

H. Beaussier [1], O. Naggara [2], D. Calvet [2], R. Joannides [3], M. Iacob [3], B. Laloux [1], E. Bozec [1], E. Guegan-Massardier [3], A. Triquenot-Bagan [3], I. Masson [1], C. Oppenheim [2], P. Boutouyrie [1], S. Laurent [1].
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Background: Combining functional and structural approaches may improve the predictive value for plaque rupture and ischemic events. Two distinct patterns were previously determined along the common carotid artery (CCA) (Paini et al. Stroke 2007): Pattern A (larger radial strain at the plaque level than at adjacent CCA) and its opposite, Pattern B.
Aim: To correlate arterial mechanics and composition of an atherosclerotic plaque at the site of the CCA.
Method: 27 patients with carotid stenosis and an atherosclerotic plaque on the ipsilateral CCA were included: 18 asymptomatics (AS) and 9 symptomatics (S, i.e. with previous ischemic stroke). Mechanical parameters were measured at 127 sites on a 4 cm long CCA segment by a novel non-invasive echotracking system (ArtLab®) and plaque composition was determined by non invasive magnetic resonance imaging (MRI).
Results: There was a trend for pattern A (21 patients) being more often associated with ''simple'' plaque (i.e. AHA stage I-III) than complex plaque (AHA stage IV-VII), by contrast to pattern B (25 patients) (chi square $P=0.054$). Pattern B was more frequently observed in S than AS patients (75% vs 43%, $P<0.04$). In S patients, plaques were characterized by an outward remodeling (increased external diameter and no change in internal diameter) whereas AS plaques grew according to an inward remodeling.
Conclusion: Patients with previous ischemic stroke had a stiffer carotid at the level of the plaque and present a more ''complex'' plaque composition than asymptomatic patients. Pattern B and complex plaque composition may lead to a higher risk of rupture.

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03.05
ESTIMATED CENTRAL BLOOD PRESSURE: IMPORTANCE OF RADIAL ARTERY PRESSURE WAVEFORM CALIBRATION

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Background: Non-invasive estimation of central blood pressure from radial artery (RA) pressure waveforms is increasingly applied. We investigated the impact of RA waveform calibration on central blood pressure assessment, with focus on the one-third rule used to estimate mean arterial blood pressure (MAP).
Methods: Pressure waveforms were non-invasively measured at the radial (RA), brachial (BA), and carotid (CA) artery in 1899 apparently healthy subjects (age 45.8±6.1 yr). RA and CA waveforms were calibrated using DBP_{BA} and (i) SBP_{BA}; (ii) MAP estimated with the one-third rule; (iii) MAP estimated as DBP_{BA} + 40% of BA pulse pressure (PP_{BA}), and (iv) MAP from the scaled BA pressure waveform (MAP_{ref}). Central SBP was obtained via a transfer function (SBP_{TF}).
Results: SBP_{TF} calculated by assuming $SBP_{BA}=SBP_{RA}$ (i), with the one-third rule (ii) and 40% rule (iii) calibration was respectively 6.2±4.8, 11.9±5.5 and 3.7±5.3 mmHg ($p<0.001$) lower than SBP_{CA} calibrated with method (iv), considered as the reference value. Applying the $1/3^{rd}$ rule, brachial-to-radial amplification was negative (-6.3±4.5 mmHg), while positive (6.5±4.9 mmHg) as expected with reference method (iv). PP_{BA} and brachial-to-radial amplification were main determinants of the difference between SBP_{CA} and SBP_{TF}.
Conclusions: SBP_{TF} is highly sensitive to the RA calibration procedure which determines the extent of brachial-to-radial pressure amplification accounted for. The $1/3^{rd}$ rule should be avoided to calibrate radial artery pressure waveforms. We therefore advise to use 40% of the PP to assess MAP as advocated by Bos et al. when brachial tonometry measurements are not available.

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04.01
EFFECT OF EXTRAVASCULAR COMPRESSION AND RELAXATION ON CORONARY HAEMODYNAMICS

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Background: Different haemodynamics are present in left ventricular hypertrophy (LVH) due to arterial hypertension and aortic stenosis (AS) and these can have different effects on the microvasculature. We explored this by applying wave intensity analysis to (1) identify the proximal- and distal-originating intracoronary waves determining the flow velocity waveform and (2) investigate the extravascular influences on these waves.
Methods: Thirty-one patients (mean age 63±12years, 18 female) with unobstructed coronary arteries, ten of whom had severe aortic stenosis, underwent simultaneous pressure and Doppler velocity measurements with sensor-tipped intra-arterial wires in each of the left coronary arteries to derive wave intensity.
Results: In subjects with normal valves, the microcirculatory waves already accounted for the majority of the intra-coronary wave energy (54.7±6.0%), but in the AS patients this rose to 74.1±10.7%, $p<0.001$. This resulted from larger absolute microcirculatory originating waves, both during systolic microvascular compression (no valve disease: 1.4 [0.6-3.2] $x10^3Wm^{-2}s^{-1}$ versus AS: 11.7 [5.4-25.5] $x10^3Wm^{-2}s^{-1}$, $p<0.001$) and during diastolic microvascular relaxation (no valve disease: 14.0 [6.6-18.0] $x10^3Wm^{-2}s^{-1}$ versus AS: 31.1 [20.4-47.4] $x10^3Wm^{-2}s^{-1}$, $p<0.001$). Haemodynamic loading of the left ventricle accounted for the extent of the compression wave ($r=0.79$, $p<0.001$) and the diastolic microvascular relaxation wave was accounted for by reduced diastolic time ($r=-0.62$, $p<0.001$).
Conclusion: Coronary circulation in aortic stenosis is even more dependent on distal-originating waves than it is in normals and this is in contrast to what is seen in LVH due to arterial hypertension. This is because the enhanced extravascular force overwhelms any local impairment within the microvasculature.

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04.02
EXAMINATION OF EFFECTS OF TNF-ALPHA ANTAGONISTS ON ARTERIAL STIFFNESS IN PATIENTS WITH RHEUMATOID ARTHRITIS AND RELATED ARTHROPATHIES: A CONTROLLED STUDY

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Objective: It has been suggested that the chronic inflammatory state of rheumatoid arthritis (RA), ankylosing spondylitis (AS) and psoriatic arthritis (PsA) contributes to accelerated atherosclerosis. The aim of this study was to evaluate the effect of anti-TNF-α therapy on arterial stiffness in patients with RA, AS and PsA.
Methods: 35 patients (RA=17, AS=12 and PsA=6) who started with anti-TNF-α therapy (adalimumab=15, etanercept=12, infliximab=8) and a non-treatment group of 25 patients (RA=12, AS=9 and PsA=4) underwent measurements of aortic Pulse Wave Velocity (aPWV) and Augmentation Index (AIx) at baseline and after 3 months (Sphygmocor). Patients in the non-treatment group had the same indications for anti-TNF-α therapy, but had to postpone their initiation due to positive Mantoux-test or planned operations.
Results: Patients who started anti-TNF-α therapy had a significant decrease in aPWV (-0.465 m/s) whereas the patients in the control group had no change (+0.061 m/s, $p=0.002$ for between group changes). Between group