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

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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

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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 1/3rd 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 1/3rd 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 ± 12 years, 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 ($54.7\pm 6.0\%$), but in the AS patients this rose to $74.1\pm 10.7\%$, $p<0.001$. This resulted from larger absolute microcirculatory originating waves, both during systolic microvascular compression (no valve disease: $1.4 [0.6-3.2] \times 10^3 \text{Wm}^{-2}\text{s}^{-1}$ versus AS: $11.7 [5.4-25.5] \times 10^3 \text{Wm}^{-2}\text{s}^{-1}$, $p<0.001$) and during diastolic microvascular relaxation (no valve disease: $14.0 [6.6-18.0] \times 10^3 \text{Wm}^{-2}\text{s}^{-1}$ versus AS: $31.1 [20.4-47.4] \times 10^3 \text{Wm}^{-2}\text{s}^{-1}$, $p<0.001$). 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