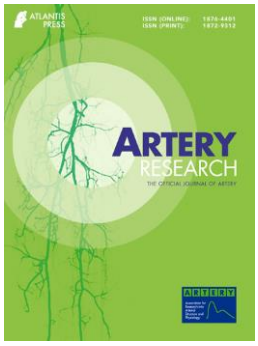

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04.02: EXAMINATION OF EFFECTS OF TNF-ALPHA ANTAGONISTS ON ARTERIAL STIFFNESS IN PATIENTS WITH RHEUMATOID ARTHRITIS AND RELATED ARTHROPATHIES: A CONTROLLED STUDY

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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 $1/3^{rd}$ 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

differences for AIx were not observed (change -0.62% and +0.44%, p=0.48). As expected, a significant reduction in CRP (-9.2 mg/l, p=0.011) and DAS28 for the RA patients (-0.73, p=0.002) was observed in the treatment group, but we did not find significant correlations between change in aPWV and CRP in the entire treatment group (r=0.055, p=0.785) and between change in aPWV and DAS28 in the RA group (r=0.091, p=0.737).
Conclusion: These findings indicate that anti-TNF-alpha therapy ameliorates functional parameters of early atherosclerosis. However, changes in aPWV were not correlated to improvement in markers of inflammatory activity.

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04.03
LIFE-COURSE HABITUAL PHYSICAL ACTIVITY AND ITS IMPACT ON ARTERIAL STIFFNESS: THE AMSTERDAM GROWTH AND HEALTH LONGITUDINAL STUDY (AGAHLS)

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Purpose: To examine how the development over time (i.e. from adolescence to adulthood) of habitual physical activity (HPA) impacts on arterial stiffness (AS) of both the elastic carotid (CCA) and the muscular femoral (CFA) arteries in adulthood.
Methods: Longitudinal data on HPA (expressed in metabolic equivalents/week − METs/wk) were retrieved from the AGAHLS (*n=373*, 196 women, 8 follow-up measures between the ages of 13 and 36 yrs). AS (i.e. CCA and CFA distensibility and compliance coefficients and CCA's Young's elastic modulus) was assessed by non-invasive ultrasonography when subjects were 36 yrs old; a sex-specific AS score for each artery was calculated by averaging the height and MAP-adjusted z-scores of each of these estimates. Generalized estimating equations were used to compare the mean levels of HPA throughout the 24-yr follow-up period between those subjects with 'stiffer' (i.e. lowest quartile) *vs.* 'normal' (highest 3 quartiles of AS score) arteries at the age of 36.
Results: Compared to subjects with 'normal', those with 'stiffer' CCA and CFA arteries had spent, on average and throughout the longitudinal period, 376 and 500 less METs/week on HPA (corresponding to @ 9 to 12 min/day of light-to-moderate intensity bicycling), respectively (Table). Adjustments for other risk factors (RFs), in particular cardiopulmonary fitness, explained these differences to a large extent for the CCA, but other RFs may also explain the association between HPA and CFA stiffness.

Conclusion: Promoting increases in HPA during adolescence and throughout the course of life may prevent the development of AS, partially due to its beneficial effects on fitness and other cardiovascular RFs.

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06.01
LIFE-COURSE OF MEAN ARTERIAL PRESSURE AND ITS IMPACT ON ARTERIAL STIFFNESS: THE AMSTERDAM GROWTH AND HEALTH LONGITUDINAL STUDY (AGAHLS)

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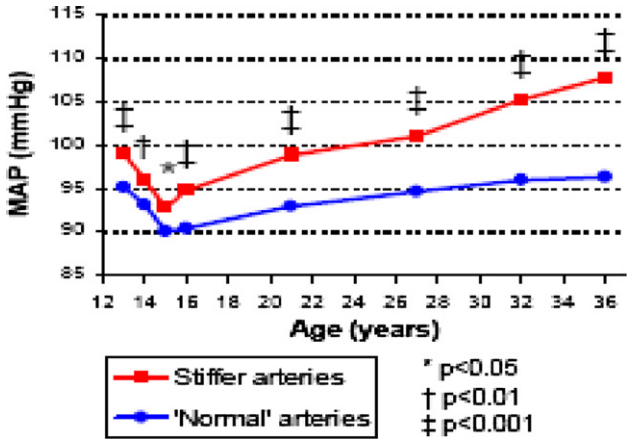
Model: adjustments	Stiff vs. normal CCA		Stiff vs. normal CFA	
	β	95% CI	β	95% CI
1. time, sex, height, smoking, alcohol, energy intake	−376	−724; −27	−500	−839; −161
2. + body fatness (sum of 4 skinfolds)	−317	−668; 34	−471	−810; −132
3. + cardiopulmonary fitness (VO_2max)	−172	−510; 166	−386	−712; −60
4. + blood lipids (total−to−HDL cholesterol ratio)	−234	−585; 117	−489	−826; −152
5. + resting heart rate	−328	−670; 15	−461	−794; −128
6. + systolic blood pressure	−350	−709; 8	−482	−828; −136
7. + all variables in models 2 to 6	−145	−490; 201	−422	−750; −93

Purpose: To investigate how the development over time (i.e. from adolescence to adulthood) of mean arterial pressure (MAP) impacts on arterial stiffness in adulthood.
Methods: Longitudinal data on systolic (SP) and diastolic (DP) blood pressure were retrieved from the AGAHLS (*n=373*, 196 women; 8 follow-up measures between the ages of 13 and 36 yrs). MAP was calculated as [(2*DP)+SP]/3. Arterial stiffness (i.e. carotid, brachial and femoral distensibility and compliance coefficients) was assessed by non-invasive ultrasonography when subjects were 36 yrs old; a sex-specific total stiffness score was calculated by averaging the height and local MAP-adjusted z-scores of each of these estimates. Generalized estimating equations were used to compare the mean levels (and the patterns of development) of MAP throughout the 24-yr follow-up period between subjects with 'stiffer' (i.e. lowest quartile) *vs.* 'normal' arteries (highest 3 quartiles of the total stiffness score) at the age of 36.
Results: Compared to subjects with 'normal', those with 'stiffer' arteries had, on average, 6.36 mmHg (95%CI: 5.04; 7.67) greater levels of MAP throughout the longitudinal period. These differences were already present in adolescence and were further amplified thereafter with subjects with stiffer arteries showing a steeper increase in MAP between adolescence and age 36 (Figure). Adjustments for other risk factors (i.e. smoking behaviour, energy and alcohol intake, physical activity, body fatness, blood lipids and heart rate) only slightly attenuated these differences.



Conclusion: Blood pressure monitoring should start already in early age in order to avoid/delay arterial stiffening and related cardiovascular complications.

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06.02
RELATION OF AORTIC STIFFNESS WITH ECHOCARDIOGRAPHIC INDICES OF LEFT VENTRICULAR DIASTOLIC FILLING AND LONGITUDINAL VELOCITIES IN SUBJECTS FREE OF CLINICAL CARDIOVASCULAR DISEASE

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Introduction: a relation between aortic stiffening (AS) and LV systolic dysfunction and hypertrophy is established in the elderly. A relation between AS and LV diastolic function can be hypothesized, mediated by age and increased LV mass (LVM).
Aim: to verify whether AS may affect LV diastolic function independently of LVM and age in subjects with preserved systolic function.
Methods: 144 subjects below 65 years, (59 controls: age 40±12, MBP 84±7 mmHg; and 85 patients with at least one major risk factor, free of CV disease, age 42±16, mean BP 96±12 mmHg). LV mass, systolic function, diastolic filling