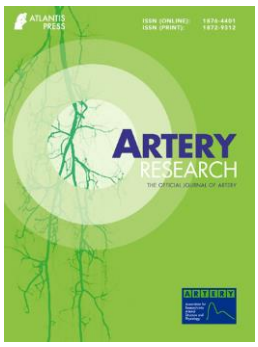

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09.04: A UNIFYING EXPLANATION OF THE AORTIC PULSE WAVEFORM IN HUMANS

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with rheumatoid arthritis (RA). The aim of this study was to investigate the effect of simvastatin and ezetimibe on inflammation, disease activity, arterial stiffness and endothelial function in patients with RA and to test our hypothesis that cholesterol lowering per se can improve arterial stiffness and reduce inflammation.
Methods: 20 RA patients received simvastatin 20 mg and ezetimibe 10 mg in a double-blind cross over study. Blood pressure, aortic pulse wave velocity (PWV) and flow mediated dilatation response (FMD) were measured before and after each treatment. Serum inflammatory markers and disease activity were also determined. Data are mean changes±SEM, and significance was determined using 2-way repeated measures ANOVA.
Results: As expected both ezetimibe and simvastatin significantly reduce total cholesterol (-0.62±0.12 and -1.28±0.11 mmol/L, respectively; $P < 0.0001$). Both drugs significantly reduced CRP (-5.35±2.07 and -5.05±1.41 mg/L; $P = 0.0002$); disease activity (-0.74±0.24 and -0.50±0.18; $P < 0.0001$); aortic PWV (-0.69±0.26 and -0.71±0.16 m/s; $P = 0.0012$) and concomitantly, FMD was significantly improved (1.37±0.26 and 2.51±0.48%; $P = 0.0001$). Importantly, only the effect on total cholesterol differed significantly between the drugs ($P < 0.001$).
Conclusion: The present study shows, that both ezetimibe and simvastatin reduce inflammatory markers and disease activity to a similar extend in patients with RA. Moreover, aortic PWV was reduced with both drugs and concomitantly, endothelial function was improved. This suggests that cholesterol lowering per se has anti-inflammatory effects and improves vascular function.

09.02
RELATIONSHIP BETWEEN GROWTH AND AORTIC STIFFNESS IN EARLY YEARS OF LIFE

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Introduction: The exact course of aortic stiffness in early years of life is not known. This study was designed to test the relationship between aortic pulse wave velocity (aPWV) and the parameters of growth amongst children aged between 0 and 2 years. Our hypothesis was that aPWV is influenced by growth velocity.
Methods: Data was obtained from 517 baby-visits between 0 to 24 months of age, and included measurement of weight, length, blood pressure (BP) and aPWV.
Results: The weight, BMI and rate of BMI change are associated with aPWV and BP at birth, 1 and 2 years of age as shown in the table. aPWV

	Weight	At birth		1 year		2 years	
		Weight	BMI	BMI rate 0-1	Weight	BMI	BMI rate 0-2
aPWV	r = 0.29, p = 0.001						
SBP		r = 0.30, p < 0.001	r = 0.24, p = 0.002	r = 0.24, p = 0.006	r = 0.46, p < 0.001	r = 0.36, p < 0.001	r = 0.27, p = 0.013
DBP			r = 0.19, p = 0.017	r = 0.241, p = 0.007			

increased by 6% from birth to 12 months and 37% from 12 to 24 months with an overall increase from birth to 24 months of 45%. Adjusting for gender, ethnicity, weight-rate and height, pulse pressure was found to be independently influenced by pulse pressure at age of 1 year (Beta = -0.027, p = 0.030; 95% CI -0.05 to -0.003), but this association was lost at age of 2 years.
Conclusions: Aortic stiffness is associated with increasing age, weight and BMI, as well as the rate of change of the latter two. All these variables are recognised cardiovascular risk factors and should be controlled from an early age.

09.03
MULTI-AXIAL MECHANICAL CHARACTERISTICS OF CAROTID PLAQUE IN HYPERTENSIVES ASSESSED BY MULTI-ARRAY ECHOTRACKING SYSTEM

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The vulnerability to rupture of carotid plaque depends on the various types of mechanical stress including higher circumferential wall stress (CWS) in hypertensives and histological characteristics of plaque.
Objective: determine the multiaxial mechanical deformations of the common carotid artery (CCA) with an echotracking system allowing measurement of thickness, diameter, strain, distensibility, elastic modulus (Einc) and CWS on 4 cm long CCA segments including plaque. This allowed us to determine a longitudinal bending stress (BS) equal to the ratio of strain at the level of plaque to strain of adjacent CCA.
Patients: we included 25 patients with a recent cerebrovascular ischemic event and a plaque on CCA homolateral to stroke territory. We divided patients into two groups according to BS behaviour: pattern A (outward BS, larger strain at plaque site than on CCA), pattern B (inward BS).
Results: 16 patients belonged to pattern A and 8 patients to pattern B. Prevalence of dysplipidemia and diabetic were higher in pattern B (100% *vs* 56%, $p = 0.03$ and 63% *vs* 12%, $p = 0.04$). In pattern B distensibility was significantly lower at the level of plaque than in CCA it was the converse in A patients (13.1±6.5 *vs* 18.2±3.9, $p < 0.003$ and 22.3±11.2 *vs* 16.6±12.4 $kPa^{-1}10^{-3}$, $p < 0.001$). Pattern A patients had lower Einc at the level of the plaque than of CCA (374±173 *vs* 802±669 kPa, $p < 0.01$) the opposite was observed in B patients (739±497 *vs* 543±146 kPa, $p < 0.01$). CWS in CCA was higher in B than in A patients (83±16 *vs* 65±15 kPa, $p < 0.01$), plaque CWS was similar in the two groups (60±7 vs 53±13 kPa, NS).
Conclusion: type 2 diabetes and dyslipidemia were associated with a stiffer plaque than adjacent CCA. These results suggest that the higher risk of plaque complication, reported in patients with diabetes and hypercholesterolemia, may be due to a specific pattern of strain gradient between plaque and adjacent CCA.

09.04
A UNIFYING EXPLANATION OF THE AORTIC PULSE WAVEFORM IN HUMANS

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Introduction: Despite more than 200 years of research, no model has been able to fit all the aortic pressure waveform with physiologically interpretable parameters. We propose that the arterial waveform is composed of two components: (1) an arterial windkessel which stores ejected blood during systole and discharges it during diastole and (2) waves originating from the left ventricle and distal reflection sites.
Method: In 19 subjects (age 54±13 years) we measured simultaneous pressure and velocity in the aorta. The windkessel component of the pressure wave was calculated, and forward and backward waves were identified as previously described [1]. The peak contribution of each component was calculated after subtraction of the diastolic pressure.
Result: In the human aorta, the initial rise in pressure was due to a wave arising from the left ventricle (Figure 1). This wave was responsible for 20 mmHg (29%) of the total rise in pressure. Windkessel pressure was responsible for 40 mmHg (57%) of the total pressure rise. Reflected waves were responsible for 10 mmHg (14%) of the total rise in pressure.

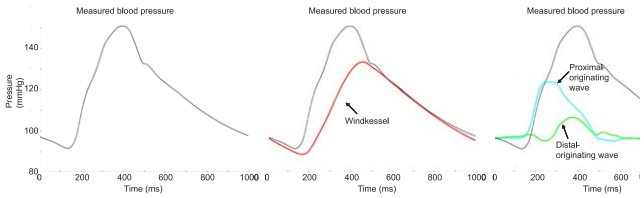

Fig. 1.

Conclusions: Using this new approach we have shown that the aortic pressure wave consists of three principal components. The systolic rise in pressure in the aorta is largely determined by a windkessel and waves arising from the left ventricle. Reflected waves make only a minor contribution. Waves do not contribute to the pressure and flow in diastole. Diastolic pressure is due to capacitative discharge of pressure from the Windkessel.

References

[1] Wang JJ et al., Am J Physiol Heart Circ Physiol 2003; 284: H1358-H1368.