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


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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 dilation response (FMD) were measured before and after each treatment. Serum inflammatory markers and disease activity were also determined. Data are mean ± SD and significance was determined using 2-way repeated measures ANOVA.

Results: As expected both ezetimibe and simvastatin significantly reduced 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); 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).

Conclusions: The present study shows, that both ezetimibe and simvastatin reduce total cholesterol and markers of disease activity and improve arterial stiffness. 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 to 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.

<table>
<thead>
<tr>
<th>aPWV</th>
<th>0.29</th>
<th>0.001</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>0.30</td>
<td>0.001</td>
</tr>
<tr>
<td>DBP</td>
<td>0.24</td>
<td>0.001</td>
</tr>
</tbody>
</table>

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 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.

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 is reflected blood pressure; 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 arriving 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.

References