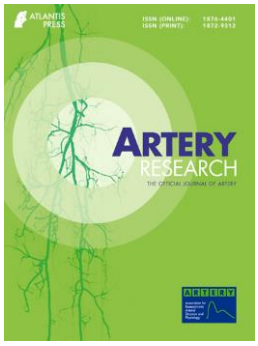

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P9.08: ASSESSMENT OF PLAQUE FORMATION IS A PRE-REQUISITE TO PREDICT CARDIOVASCULAR COMPLICATIONS USING CAROTID INTIMA MEDIA THICKNESS

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P9.05
INFLUENCE OF PERIPHERAL BLOOD PRESSURE CALIBRATION ON THE ESTIMATION OF CENTRAL BLOOD PRESSURE

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Objective: To examine the influence of calibration of peripheral blood pressure on the estimation of central systolic blood pressure (cSBP) from peripheral arterial waveforms using a transfer function.

Methods: Central aortic pressure was measured with a pressure tipped catheter (Millar, Houston, Texas) placed in the proximal aortic root in 30 subjects at the time of cardiac catheterisation. Digital pressure waveforms were acquired using a Finometer (Finapres, Netherlands). Non-invasive brachial artery pressure was measured oscillometrically (Omron 705IT, Omron, Japan). Measurements were obtained at baseline and after nitroglycerin (500 μg sublingual). Digital arterial waveforms were calibrated using 1) oscillometric systolic and diastolic pressures 2) oscillometric mean and diastolic pressures 3) invasive aortic mean and diastolic pressures. The same transfer function was applied to these waveforms to estimate cSBP and estimated cSBP compared with measured values. Accuracy of peripheral oscillometric blood pressure was assessed by comparison with the invasively calibrated digital waveform.

Results: Oscillometric values of systolic, diastolic and mean pressures were 1.1 ± 14.5 lower, 11.9 ± 7.6 higher and 7.9 ± 6.8 mmHg higher (means $\pm$ SD) respectively than values obtained by invasive calibration. When digital waveforms were calibrated from oscillometric systolic and diastolic pressures estimated cSBP was 3.3 ± 11.2 mmHg higher than measured cSBP and when calibrated using oscillometric mean and diastolic pressures estimated cSBP was 3.7 ± 10.7 mmHg higher than measured cSBP.

Conclusion: Although systematic errors in estimation of peripheral blood pressure are high, they may compensate so that cSBP can be estimated without much systematic error.

P9.06
EVIDENCE FOR A ROLE OF THE VASCULAR ENDOTHELIUM IN THE REGULATION OF ARTERIAL WALL VISCOSITY IN VIVO IN HUMANS

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Although the viscoelasticity of large arteries has been extensively investigated, few studies have focused on arterial wall viscosity (AWV) itself and its regulation by the endothelium in vivo. This is of particular importance since AWV is a major source of energy dissipation through the vascular system reducing cardiovascular coupling efficiency. We simultaneously measured radial artery diameter and arterial pressure (NIUS02) in healthy volunteers before and after local infusion of L-NMMA (8 μmol/min) as NO-synthase inhibitor, tetraethylammonium (TEA: 9 μmol/min), as blocker of calcium-activated potassium channels, the target of endothelium-derived hyperpolarizing factors (EDHF), fluconazole (0.4 mmol/min), as inhibitor of EDHF synthesis by cytochrome epoxygenases and L-NMMA associated with TEA or with fluconazole. AWV was estimated from the ratio of the area of the hysteresis loop of the pressure-diameter relationship to the area representing the whole energy exchanged during each cardiac cycle. L-NMMA paradoxically reduced AWV ($n = 5$: 27.6 ± 0.7 to $23.4 \pm 0.7\%$, $P = 0.053$). Conversely, AWV was increased by TEA ($n = 6$: 25.5 ± 0.5 to $31.3 \pm 0.7\%$, $P = 0.040$) and fluconazole ($n = 5$: 26.6 ± 0.6 to $30.6 \pm 0.6\%$, $P = 0.047$). This increase was more marked with the association of L-NMMA + TEA ($n = 6$: 27.6 ± 0.9 to $41.0 \pm 0.7\%$, $P = 0.002$) and L-NMMA+-fluconazole ($n = 6$: 26.1 ± 0.7 to $36.3 \pm 0.3\%$, $P = 0.001$) showing a synergistic effect of both combinations on AWV. These results demonstrate that the endothelium contributes in vivo in humans to the regulation of AWV through an interaction between NO and cytochrome-related EDHF release. Therefore, the prevention of endothelial dysfunction appears a critical target to improve cardiovascular coupling and thus may help to limit the development of complications in cardiovascular diseases.

P9.07
ULTRASOUND MAPPING OF THE SUPERFICIAL VEINS IN HEALTHY SUBJECTS

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Objectives: Anatomical and physiological data on the forearm venous vascular bed is needed to (i) gain insight into the complex arterial and venous remodeling processes after creation of an arterio-venous fistula, and (ii) provide input data for computer models of the forearm vasculature.

Methods: Ultrasound measurements were performed in 12 healthy volunteers (age 23-31; 11 men) along both arms during control conditions and with application of a proximal tourniquet. The elliptical small and large diameter (d1 and d2) of the basilica and cephalic veins were measured. Cross sectional areas (CSA; in mm^2) and the eccentricity ratios (ER = d2/d1) were derived.

Results and discussion: Data are presented as mean values $\pm$ standard errors (table), N is the total number of measurements. ER values show an expected decrease when the tourniquet is used. Unlike ER, CSA showed large scatter as anticipated. Measurements did not indicate a defined tendency in CSA values after applying the tourniquet.

		Basilic		Cephalic	
		Control	Tourniquet	Control	Tourniquet
Upper arm	CSA	19.58 ± 2.12;N = 38	18.37 ± 1.96;N = 41	7.63 ± 1.23;N = 13	5.79 ± 0.75;N = 13
	ER	1.29 ± 0.03;N = 38	1.21 ± 0.03;N = 41	1.29 ± 0.05;N = 13	1.25 ± 0.03;N = 13
Elbow	CSA	15.10 ± 1.47;N = 31	14.48 ± 1.62;N = 31	17.75 ± 2.56;N = 23	19.14 ± 2.37;N = 23
	ER	1.39 ± 0.04;N = 31	1.39 ± 0.03;N = 31	1.35 ± 0.04;N = 23	1.32 ± 0.04;N = 23
Lower arm	CSA	10.68 ± 1.08;N = 16	10.17 ± 1.41;N = 16	6.47 ± 0.66;N = 73	6.63 ± 0.61;N = 75
	ER	1.40 ± 0.07;N = 16	1.23 ± 0.06;N = 16	1.58 ± 0.04;N = 73	1.46 ± 0.04;N = 75
Wrist	CSA	6.69 ± 3.43;N = 3	8.53 ± 3.51;N = 3	9.94 ± 1.17;N = 24	9.52 ± 1.11;N = 24
	ER	1.84 ± 0.16;N = 3	1.51 ± 0.09;N = 3	1.68 ± 0.06;N = 24	1.46 ± 0.05;N = 24

Conclusion: The ultrasound mapping protocol allowed to generate valuable data from healthy volunteers which will be useful in future patient studies. The large variability in venous topology suggests that an individualized, patient-specific modeling approach will be required.

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P9.08
ASSESSMENT OF PLAQUE FORMATION IS A PRE-REQUISITE TO PREDICT CARDIOVASCULAR COMPLICATIONS USING CAROTID INTIMA MEDIA THICKNESS

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Role and function of the Carotid Intima Media Thickness (CIMT) and atherosclerotic Plaque (**P**) in clinical management of cardiovascular risk factors is yet unclear. Cardiovascular Risk Factor Stratification (SCORE) and CIMT with **P** typing and visualization were assessed in 269 asymptomatic individuals over 45 years (45-65 y), undergoing an annual health check. Cases ***with* P** (n = 38) were compared to cases ***without* P** (n = 231). The CIMT was measured using a fully automated quantitative algorithm that assesses the average, minimum and maximal thickness during several seconds, as single frames sometimes did not show either a quality CIMT or Plaque formation.

Results:	CIMT with Plaque	CIMT without Plaque	*p* value
	N = 38 (age 59)	N = 231(age 58)	
CIMT (mm)	0.78 ± 0.12	0.74 ± 0.09	NS
BMI (>30 kg/m^2)	16%	10%	<0.01
Smoking	20%	15%	<0.01
Systolic BP >135 mmHg	62%	44%	<0.05
Diastolic BP > 85 mmHg	40%	21 %	<0.01
LDL-c mmol/l	3.9	3.6	NS
HDL-c mmol/l	0.95	1.05	NS

These findings indicate that normal/high CIMT values were present in both populations. Individual risk factors were only significantly correlated to risk factors if **P** was present.

Conclusion: assessment of only a CIMT in a population group above 45 years old does not add much to the standardized SCORE risk factor stratification. If a **P** is present, the addition of a measurement of CIMT plus **P** has incremental value to manage the subclinical disease in clinical practice. These findings underscore that in subclinical disease several frames should be assessed to maximize quality. CIMT alone adds little value to a SCORE measurement procedure.

Summary:

1.Plaque assessment seems a critical component to manage disease in clinical practice to provide incremental value in addition to SCORE and CIMT alone.

2. Multiple frames should be assessed to maximize yield on image quality.

P9.09
LARGE ARTERY FUNCTION AND VENTRICULAR ARTERIAL COUPLING DURING PROLONGED BED REST

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Background: prolonged circulatory unloading associated with head-down tilt bed rest (HDTBR) is followed by cardiovascular deconditioning.

Aim of the study was to investigate to what extent large artery function and arterial-ventricular coupling (VA) are involved.

Methods: ten healthy male volunteers (age 23 ± 2) were studied before and after a 35-day HDTBR. Left ventricular (LV) volumes were investigated by echocardiography; carotid diameter and intima media thickness were assessed by high resolution ultrasound (Q-IMT, Esaote Europe). Contour Wave Analysis, performed by tonometer (PulsePen, DiaTecne, Milan Italy), was used to explore large artery function. Carotid-femoral pulse wave velocity (PWV) was also estimated (Complior, Alam, Paris).

Results: no changes were observed for systolic and diastolic blood pressure, PWV and QIMT vs baseline, while LV volumes showed a significant reduction ($p < 0.05$). Arterial Elastance (Ea= end systolic pressure/stroke volume) and LV Elastance (Elv= end systolic pressure (ESP)/end systolic volume (ESV)) increased after HDTBR (for Ea: 1.08 ± 0.198 vs 1.31 ± 0.21, $p = 0.01$; for Elv 1.478 ± 0.32 vs 1.765 ± 0.42, $p = 0.04$) with unchanged Ea/Elv (0.74 ± 0.09 vs 0.76 ± 0.1). Contour wave analysis showed no significant changes for Augmentation Index (Aix), a reduction for PPI (Pulse Pressure Index: pulse pressure/mean arterial pressure, from 0.55 ± 0.14 to 0.45 ± 0.09, $p < 0.05$), SEVR (subendocardial variability ratio: 1.84 ± 0.33 vs 1.55 ± 0.25, $p = 0.008$) and LVET (left ventricular ejection time: 304.6 ± 19.8 vs 291.5 ± 11.2 ms, $p = 0.05$), and an increase in heart rate (from 58 ± 2 to 73 ± 6, $p < 0.05$).

Conclusions: no significant alterations in intrinsic arterial stiffness and structure were detected after HDTBR. The observed changes in large arteries function appear secondary to changes in LV performance.

P9.10
PULSE WAVE REFLECTION IN SUPINE AND STANDING HEALTHY YOUNG AND ELDERLY

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Objective. Pressure wave reflection poses a load on the heart often augmenting pressure and adversely affecting cardiac output. Reflection effects are age dependent and may be altered by sympathetic activation by postural stress. We sought to determine the influence of standing on wave reflection and total peripheral resistance (TPR), in healthy elderly and young individuals. Wave reflection is believed to be increased by higher TPR.

Design. Analysis of aortic pressure reconstructed from noninvasive finger arterial pressure and calculated aortic flow.

Subjects. Healthy elderly (n = 15) and healthy young subjects (n = 15).

Interventions. At least 5 minutes supine followed by 5 minutes standing.

Measurements. Calculated aortic pressure and flow were used to derive forward (Pf) and backward (Pb) pressure waves, Reflection Magnitude (RM, amplitude ratio of Pb and Pf), Augmentation Index (AI), and TPR in supine position, and after 60 s and 300 s of standing.

Results. RM and AI were higher in the elderly group as expected. Upon standing, in both groups, diastolic pressure and heart rate increased while pulse pressure, stroke volume and cardiac output decreased; RM and AI decreased but TPR increased. Pb decreased in both groups, but Pf remained the same in the elderly group whereas it decreased in the young group.

Conclusions. With standing, hemodynamic variables change similarly in elderly and young healthy subjects. The opposite changes in RM and TPR during postural stress in both young and elderly suggest that wave reflections do not solely originate from the periphery.