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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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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.

$30.6 \pm 0.6\%$, $P = 0.047$). 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 AWW. These results demonstrate that the endothelium contributes in vivo in humans to the regulation of AWW 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 mm²) 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

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 AWW 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 epoxigenases and L-NMMA associated with TEA or with fluconazole. AWW 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 AWW ($n = 5$: 27.6 ± 0.7 to $23.4 \pm 0.7\%$, $P = 0.053$). Conversely, AWW 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

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.