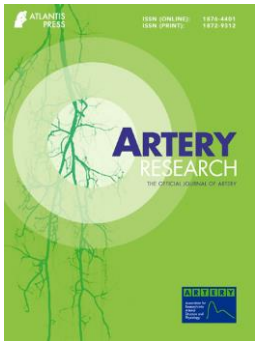

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P4.27: AUTONOMIC NERVOUS SYSTEM REACTIVITY IN NORMOTENSIVE SUBJECTS WITH A FAMILY HISTORY OF HYPERTENSION DURING VALSALVA MANOEUVRE

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Table 2 Associations between carotid plaque mobility (S and SR values), echogenicity and degree of stenosis.

	Echogenicity (Spearmen correlation analysis) r*	Degree of Stenosis (Univariant analysis) r
cap		
S	**-0.24 p=0.00045**	**-0.12 p=0.049**
SR	**-0.13 p=0.029**	**-0.17 p=0.004**
core		
S	**-0.29 p=0.0004**	-0.07 p=0.39
SR	**-0.21 p=0.001**	**-0.20 p=0.15**
base		
S	**-0.24 p=0.0043**	-0.14 p=0.086
SR	**-0.18 p=0.035**	**-0.22 p=0.008**

* negative correlation implies that hypo- and moderate echogenic plaques have lower S and SR values

P4.24

COMPLIANCE AND DISTENSIBILITY OF DIFFERENT SEGMENTS OF AORTA AND RELATIONSHIPS BETWEEN AORTA DISTENSIBILITY AND SYSTOLIC AND DIASTOLIC FUNCTIONS OF THE LEFT VENTRICULAR

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Objective: to investigate compliance and distensibility of different segments of aorta and to assess the influence of systolic and diastolic function on distensibility.
Methods: Study population consisted of 33 healthy subjects (aged 16-26, median-18). Compliance and Distensibility were calculated using M-mode ultrasound measurements at five locations: the sinuses of Valsalva, the proximal ascending aorta, the aortic arch, the infrarenal aorta, the abdominal aorta before bifurcation and also in the left common carotid artery (CCA) using wall-tracking software. All subjects underwent comprehensive transthoracic echocardiography and Central_BP measurement. Left ventricular peak wall stress (LVPS) was calculated as follows: LVPS=0.86x (0.334xSAPxEDD)/[PWTdx(1+(PWTd/EDD))]-2, where SAP- systolic blood pressure, PWTd- end-diastolic posterior-lateral wall thickness, EDD- end-diastolic diameter.
Results: Compliance gradually decreased from the proximal to the distal segments of aorta (table 1), whereas distensibility coefficient (DC) and β-stiffness index did not significantly differ in various aorta segments. By univariant analysis negative direct associations were found between DC and LVPS (r=-0,48 p=0,005 for DC in proximal ascending aorta). This relation was tested by multivariate linear regression after adjusting for potential confounders (age, male sex, weight). The model demonstrated that LVPS and E/A ratio are the only independent determinant of aorta distensibility (β=-0.41 and -0.43 for LVPS and E/A accordingly $p<0.03$, DC in the proximal ascending aorta).
Conclusions: Compliance gradually decreased from the proximal to the distal aorta segments (from the sinuses of Valsalva to the bifurcation of abdominal aorta) whereas stiffness does not differ significantly. Both systolic and diastolic parameters of left ventricular influence the distensibility.

P4.26

ADIPOCYTOKINES LEVELS MARK ENDOTHELIAL FUNCTION IN NORMOTENSIVE INDIVIDUALS

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Endothelial dysfunction is an independent risk factor for cardiovascular events. Inflammatory mediators released by the adipose tissue can lead to local insulin resistance and endothelial dysfunction. This study addressed the relationship of adipocytokines with endothelial function and blood pressure. In 92 newly diagnosed, drug-naïve essential hypertensive patients (HT) without organ damage and 66 normotensive subjects (NT), by an automated system, we measured endothelium-dependent and -independent vasodilation as brachial artery flow mediated dilation (FMD) before and after administration of glyceryl-trinitrate (GTN). Retinol binding protein-4 (RBP4) and resistin levels were determined by ELISA and RIA, respectively. Oxidative stress was evaluated by measuring serum malondyaldeide (MDA). FMD was significantly (p=0.03) lower in HT (5.3±2.6%) than NT (6.1±3.1%), while response to GTN (7.5±3.7% vs 7.9±3.4%) was similar. RBP4 (60.6±25.1 vs 61.3±25.9 μg/ml), resistin (18.8±5.3 vs 19.9±6.1 ng/ml) and MDA levels (2.39±1.26 vs 2.08±1.17 nmol/ml) were not different in HT and NT.
RBP4 (r=-0.25; p=0.04) and resistin levels (r=-0.29; p=0.03) were related to FMD in NT, but not in HT (r=-0.03 and r=-0.10, respectively). In NT multivariate analysis, including RBP4 and confounders showed that only body mass index (BMI) or waist circumference remained related to FMD. In the multivariate model including resistin and confounders, BMI, age and resistin were significantly related to FMD, while only age remained a significant correlate of FMD when BMI was replaced by waist circumference.
In conclusion, adipocytokine levels, particularly resistin, are independent predictors of endothelial dysfunction in the peripheral circulation of healthy subjects, providing a pathophysiological link between inflammation from adipose tissue and early vascular alterations.

P4.27

AUTONOMIC NERVOUS SYSTEM REACTIVITY IN NORMOTENSIVE SUBJECTS WITH A FAMILY HISTORY OF HYPERTENSION DURING VALSALVA MANOEUVRE

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Introduction: This study was designed to address alterations in autonomic nervous system activity in normotensive subjects with a family history of hypertension. We compared the autonomic nervous system activity in 11 normotensives with a family history of hypertension (age 23.3 ± 0.4) and 14 normotensives with no family history of hypertension (age 22.9 ± 0.3).
Methods: In all of the participants their cardiovascular parameters, including impedance cardiography, were measured at rest. In addition, the Valsalva manoeuvre was performed and Valsalva Index was obtained. On the basis of the arterial blood pressure change, provoked by the Valsalva manoeuvre, the latency of baroreflex response was determined.
Results: Normotensives subjects with a family history of hypertension, compared to the control group, showed significantly higher heart rate (75.0 ± 3.4 vs. 62.4 ± 1.8 beats/min), cardiac output (7.6 ± 0.4 vs. 6.7 ± 0.3 L/min), left ventricular weight index (4.6 ± 0.3 vs. 3.9 ± 0.1) and shorter

Table 1 Compliance, Distensibility and Stiffness in the Different Aorta Segments and in the left CCA

	Compliance C=ΔD/(SBP-DBP) mm × mm Hg^{-1}	Distensibility Coeffieicient DC= 2×ΔD/[Ddx(SBP-DBP)] mm Hg^{-1}	Stiffness index β=[ln(SBP/DBP)×Dd]/ ΔD	Strain CS=ΔD/D d × 100%
Sinuses of Valsalve	0.16±0.1	0.015±0.009	2.62±2.17	21±7
Proximal Ascending Aorta	0.13±0.09	0.012±0.008	2.88±2.08	17±7
Aortic Arch	0.11±0.08 *	0.013±0.008	2.53±1.32	20±8
Infrarenal Aorta	0.09±0.04 *,**	0.015±0.007	2.34±1.2	23±5
Abdominal Aorta before Bifurcation	0.08±0.04 *,**	0.015±0.009	2.43±0.92	21±5
left CCA	0.016±0.005 ***	0.005±0.002 ***	6.51±3.94 ***	7.7±2 ***

Data are expressed as mean ± SD, * significant difference ($p<0.05$) with Sinuses of Valsalve, **-significant difference ($p<0.05$) with Proximal Ascending Aorta, *** - significant difference between CCA and all aorta segments, ΔD = Ds-Dd, where Ds and Dd — systolic and diastolic diameters, SBP and DBP — central systolic and diastolic blood pressure.

ejection time (298.9 ± 4.9ms vs. 316.8 ± 4.5ms) (t-test, $p < 0.05$). In addition, the normotensives with a family history of hypertension have decreased latency of the baroreflex response (7.0 ± 0.5 s) compared to the control group (10.5 ± 0.9 s) ($p<0.001$).
Conclusions: Our results indicate that even normotensives with a family history of hypertension exhibit changes of some cardiovascular parameters at early age. The changes in Valsalva manoeuvre response also show alteration of the autonomic nervous system reactivity.

P4.28
IMPACT OF WEIGHT CHANGE ON INTIMA MEDIA THICKNESS OF CAROTID ARTERIES AND ENDOTHELIAL FUNCTION IN GEORGIAN OBESE AND OVERWEIGHT HYPERTENSIVE SUBJECTS

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Objectives: Taking into consideration that obesity and AH are the most important related risk-factors of CVD we examined differences in carotid artery intima-media thickness (IMT) and endothelial function between obese and overweight hypertensive individuals.
Methods: We studied 102 patients with mild to moderate AH (67males/35females, mean age 51,3±2,4years, BMI 30,9±1,9kg/m^2, duration of AH 4,6±1,4years). Examination included: color triplex carotid artery scanning; assessment of endothelial function of brachial artery; 24-hour BP monitoring. 49 overweight patients (25<BMI< 29,9kg/m^2) were assigned to group 1 and 53 obese patients (BMI>30kg/m^2) to group 2.
Results: The groups were comparable by the age, duration of AH, daily mean BP values. Mean values of IMT (gr1:1,02±0,03mm;gr2: 1,08±0,04mm) were certainly increased in obese patients compared with overweight ones ($p<$ 0,001). Prevalence of carotid atherosclerosis was higher in gr2 (79%vs67%). Endothelium - dependent vasodilatation (EDVD) (gr1:7,6±0,5;gr2:7,01±0,3%) was significantly reduced in obese patients ($p<0,01$), but occurrence of endothelial dysfunction was almost equal (gr1:59;gr2:60%). BMI positively correlated with IMT ($r = 0,25$, $p<0,02$) and negatively with EDVD ($r = -0,4$, $p<0,05$).
Conclusions: Thus, in obese hypertensive subjects we detected more pronounced and frequent carotid artery affection and endothelial dysfunction comparing with overweight ones. Data of our study demonstrate importance more profound examination of cardiovascular system in obese hypertensive patients with subsequent more aggressive blood pressure and weight reduction.

P4.29
LEFT ATRIAL REMODELLING IS AN EARLY CARDIAC STRUCTURAL CHANGE IN HYPERTENSION

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Background: The interest in left atrial remodelling (LAR) as a TOD in hypertension (H) has been growing recently. Little is known on the role of arterial stiffness (a.s) in the pathophysiology of LAR in H. We hypothesized that LAR precedes LVH and diastolic dysfunction (d.d.) in H and is associated with carotid a.s. independently of other possible confouders.
Methods: 85 patients—65 with H,31male and 34female,mean age55,9±10,7 years and 20 control matched subjects(C). From echocardiography: left atrial volume normalized to BSA (LA vol/BSA;ellipsoid method), LVMI, RWT, IVS, PW; from conventional and Tissue Doppler: early(E), late(A) mitral flow velocities, E/A ratio,early (e'),late(a') diastolic mitral annular velocities, e'/a' ratio;E/e'ratio were calculated. From carotid arteries ultrasound—IMT and high-resolution echo-tracking method a.s. parameters were evaluated: βstiffness index, Ep-elastic modulus, AC-arterial compliance, PWVβ-one-point pulse wave velocity.
Results: LAvol/BSA was the highest in H with LVH(24,9ml±6,1) and in H with d.d.(23,5ml±6). However,already in H without LVH,LA vol/BSA was significantly higher than in C (21,1ml±4,9vs18,3ml±4,8;$p=0,05$) and also in H without d.d. LA vol/BSA was significantly higher than in C (20,5ml±5,5vs18,3ml±4,8,$p=0,048$). Linear regression analysis revealed the following significant correlations between LA vol/BSA and age($r=0,3$), BMI($r=0,38$), mean BP($r=0,25$), preload($r=0,27$), afterload($r=0,24$), LVMI($r=0,59$), RWT($r=0,23$), IVS($r=0,5$), PW($r=0,42$), e'($r=-0,3$), E/e'($r=0,46$), BNP($r=0,73$), Ep($r=0,25$) and PWVβ($r=0,25$);p for all <0,05). However in multiple regression analysis the independent determinants were: age,BMI,mean BP,LVMI,PW,E/e' and PWVβ.
Conclusion: LAR is one of the earliest cardiac structural changes in H that precedes LVH and d.d.Local PWVβ is an independent determinant of LAR beyond:BP components, LVH, d.d.indices.It supports the hypothesis on the contribution of arterial stiffness to LAR.

P4.30
ALCOHOL EXERTS A SHIFTED U-SHAPED EFFECT ON CENTRAL AND PERIPHERAL BLOOD PRESSURE IN YOUNG ADULTS

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Consumption of 1-2 alcoholic beverages daily has been associated with a lower risk of cardiovascular disease and all-cause mortality in middle-aged and older adults. Recent studies suggest that central blood pressure (BP) is a better predictor of cardiovascular risk than peripheral BP. However, potential effects of habitual alcohol consumption on central BP particularly in young adults, the primary consumers of alcohol in North America, have yet to be investigated. Therefore, we aimed to study the effect of alcohol consumption on central and peripheral BP, pulse pressure amplification, and arterial stiffness specifically in young adults.
We recruited 130 healthy, non-smoking, non-obese individuals. Using a standardized questionnaire, alcohol consumption (drinks/week) was used to classify participants into non-, (<2), light (2-6), moderate (women: 7-9, men: 7-14), and heavy drinkers (women: >9, men: >14). Central BP and arterial stiffness measurements were obtained using applanation tonometry. We found a U-shaped effect of alcohol consumption on both central and peripheral BP. Light drinkers had significantly lower central and peripheral systolic, and mean arterial BPs when compared to non- and moderate drinkers ($P<0.05$). No significant associations with arterial stiffness parameters were noted.
A U-shaped relationship was found between alcohol consumption and both central and peripheral BP in young individuals, which importantly, was shifted towards lower levels of alcohol consumption than currently suggested. This is the first study, to our knowledge, that examines the effect of alcohol consumption on central BP and arterial stiffness exclusively in young individuals. Prospective studies are needed to confirm the relationships observed herein.

P4.31
ALTERED THROMBIN GENERATION IN SUBJECTS WITH FAMILIAL HYPERCHOLESTEROLEMIA

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Purpose: The effects of angiotensin II (ANG) on inflammation and haemostasis were examined in 16 otherwise healthy patients with familial hypercholesterolemia (FH) and in 16 healthy controls.
Methods: Plasma markers of inflammation (hs-CRP, IL-6, fibrinogen, leukocyte counts (Lct)), coagulation (thrombin generation: F1+2, Calibrated Automated Thrombogram (CAT), fibrinolysis (plasmin-antiplasmin complexes, PAI-1 activity) were assessed in conjunction to iv ANG infusion (10 ng/kg/min for 3 h). Means ± SD; repeated measures ANOVA, log transformation when appropriate.
Results: Baseline systolic blood pressure was higher in FH than in controls (127±14 vs 115±12 mm Hg, $p<0.05$), while responses to ANG were similar (+24±10 and +21±7 mm Hg). Baseline hs-CRP, IL-6, Lct, and fibrinogen were similar in FH and controls, and all increased similarly in both groups ($p<0.05$) during ANG. Baseline CAT (peak and ETP) was higher in FH (367±47 vs 317±60 nM, $p=0.01$, and 2418±391 vs 2042±358 nM/min, $p<0.01$, respectively), but ANG did not affect CAT (peak or ETP). Baseline F1+2 was similar in FH and controls (189±41 vs 186±81 pM) and unchanged by ANG. Baseline plasmin-antiplasmin complexes were similar in FH and controls (96±16 vs 93±27 μg/L) and increased ($p<0.001$) similarly by ANG in both groups. PAI-1 activity was similar in both groups at baseline (1.3±1.3 vs 1.1±1.2 ng/L) and decreased ($p<0.001$) similarly in both groups, confirming the diurnal variation in fibrinolysis.