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P4.31: ALTERED THROMBIN GENERATION IN SUBJECTS WITH FAMILIAL HYPERCHOLESTEROLEMIA

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ejection time (298.9 ± 4.9 ms vs. 316.8 ± 4.5 ms) (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 ± 2.4 years, BMI 30.9 ± 1.9 kg/m², duration of AH 4.6 ± 1.4 years). Examination included: color triplex carotid artery scanning; assessment of endothelial function of brachial artery; 24-hour BP monitoring. 49 overweight patients ($25 < \text{BMI} < 29.9$ kg/m²) were assigned to group 1 and 53 obese patients ($\text{BMI} > 30$ kg/m²) 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 ± 0.03 mm; gr2: 1.08 ± 0.04 mm) were certainly increased in obese patients compared with overweight ones ($p < 0.001$). Prevalence of carotid atherosclerosis was higher in gr2 (79% vs 67%). 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 confounders.

Methods: 85 patients—65 with H, 31 male and 34 female, mean age 55.9 ± 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: β stiffness index, Ep-elastic modulus, AC-arterial compliance, PWV β -one-point pulse wave velocity.

Results: LA vol/BSA was the highest in H with LVH ($24.9 \text{ ml} \pm 6.1$) and in H with d.d. ($23.5 \text{ ml} \pm 6$). However, already in H without LVH, LA vol/BSA was significantly higher than in C ($21.1 \text{ ml} \pm 4.9$ vs $18.3 \text{ ml} \pm 4.8$; $p = 0.05$) and also in H without d.d. LA vol/BSA was significantly higher than in C ($20.5 \text{ ml} \pm 5.5$ vs $18.3 \text{ ml} \pm 4.8$; $p = 0.048$). 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 PWV β ($r = 0.25$); p for all < 0.05). However in multiple regression analysis the independent determinants were: age, BMI, mean BP, LVMI, PW, E/e' and PWV β .

Conclusion: LAR is one of the earliest cardiac structural changes in H that precedes LVH and d.d. Local PWV β 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 \pm 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 ($+24 \pm 10$ and $+21 \pm 7$ 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 $\mu\text{g/L}$) 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.

Conclusions: Subjects with FH have an increased thrombin generation potential, while an intact fibrinolysis. ANG has proinflammatory effects, similar in FH and healthy controls, but does not affect coagulation or fibrinolysis.

P4.32

HAEMODYNAMIC PATTERN OF OBESE PATIENTS ON PRIMARY CARDIOVASCULAR PREVENTION

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Background: Impedance Cardiography (ICG) is a non-invasive method to assess the main haemodynamic parameters: cardiac output, peripheral resistance, cardiac work, and thoracic fluid content. There is no agreement about the haemodynamic pattern in obese patients and it is very important at the time to decide adequate therapeutic interventions.

Objective: To study the haemodynamic pattern in obese patients

Methods: We compared 95 male obese patients with 212 matched male lean controls between DEC2010 and JAN2011 derived for routine cardiovascular evaluation without history of CV disease, for Primary CV Prevention. We used an Impedance Cardiograph (Z Logic (R)) following standard procedures. **Results:** (only signif.) Obese patients presented higher BP, HR, BMI and BSA. They had increased Thoracic Fluid Content and higher Peripheral Resistance Index and reduced Aortic compliance.

Cardiac Index and cardiac acceleration index were lower and pre-ejective period was longer in the obese group. (see table attached)

Conclusion: Obese patients compared with matched controls present increased thoracic fluid content, peripheral vascular resistance and aortic stiffness associated with depression of cardiac function. This pattern may be associated to early stages of ventriculo arterial uncoupling and increased CV risk.

PARAMETERS	LEAN (n=212)	OBESE (n= 95)	SIGNIF
AGE (yrs)	50,1 ± 5,7	50,1 ± 5,8	NS
WEIGHT (kgs)	82,1 ± 8,3	102,2 ± 11,9	<0.001
HEIGHT (cms)	176,6 ± 6,1	175,9 ± 6,9	NS
BMI (Kg/m2)	26,3 ± 1,8	33 ± 3,1	<0.001
BSA (m2)	2 ± 0,1	2,2 ± 0,2	<0.001
SBP (mmHg)	132 ± 17,5	140 ± 16,4	<0.001
DBP (mmHg)	82,3 ± 11,1	88 ± 9,3	<0.001
HR (bpm)	59,2 ± 10,1	63 ± 11,2	.003
THOR IMP (Z0) (Ohms)	23,9 ± 2,2	22,6 ± 2,5	<0.001
TFC (kohms. (-1))	42,1 ± 3,9	44,7 ± 5	<0.001
CO (l/min)	4,8 ± 1,3	4,7 ± 1	NS
CI (l/min /m2)	2,5 ± 1	2,2 ± 0,6	0.001
PVR (dyn.seg.cm-5)	1683,9 ± 528,3	1855,2 ± 643,3	0.024
PVRI (dyn.seg.cm-5.m2)	3341,8 ± 1074,3	4016 ± 1482,1	<0.001
PREEJECT P (mseg)	119 ± 21,6	127,6 ± 18	<0.001
EJECTIVE P (mseg)	324,4 ± 33,3	312,4 ± 42,5	0.016
CARD ACC IND (1000.seg)	68,3 ± 15,3	59,8 ± 14,1	<0.001
AO COMPL (ml/mmHg)	1,8 ± 0,7	1,6 ± 0,6	0.011

Ref: Thor Imp: Thoracic Impedance TFC: Thoracic Fluid Content CO: Cardiac Output CI: Cardiac Index PVR: Peripheral Vascular Resistance PVRI: Peripheral Vascular Resistance Index Preeject P: Preejective Period Ejective P: Ejective Period Card Acc Ind: Systolic Acceleration Index Ao Compl: Aortic Compliance (Zc)

P4.33

ASSOCIATION BETWEEN DEPRESSION, ANXIETY AND INFLAMMATION PROCESS IN POSTOPERATIVE PERIODS OF CORONARY ANGIOPLASTY AND AORTO-CORONARY BYPASS GRAFT SURGERY

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Purpose: Depression and anxiety has been related to a higher risk of developing coronary heart disease, but the mechanism that accounts for this association is unclear. The aim of our study was to investigate the association between history of depressive episode and anxiety and presence of low-grade systemic inflammation as measured by serum C-reactive protein in postoperative period of coronary angioplasty (PCA) and aorto-coronary bypass graft surgery (CABG).

Methods: The research was performed in 80 patients (n=80), mean age 60±15 years. These patients have no high cholesterol level, high body mass index and n= 64 (80%) of them are no smoker. To evaluate depression we used Beck depression scale. Anxiety was assessed by the Spilberger State-trait anxiety scale. CRP was measured in venous blood.

Results: In angioplasty group patients who had increased level of CRP had high degree of depression and trait anxiety p=0.001; p<0.001. In aorto-coronary bypass graft surgery group elevated level of CRP was also associated with high degree of depression p=0.001. In PCA and CABG groups patients who were rehospitalized with signs and symptoms of heart failure (NYHA II-III) during 2 year follow up period had high and moderate degree of depression p = 0.003 and anxiety p=0.001 (state anxiety p = 0.001; trait anxiety p = 0.001).

Conclusions: Our study demonstrated association between depression, anxiety and increased c-reactive protein level. These results may have important implications in explaining the pathophysiological mechanisms linking depression and anxiety to cardiovascular disease.

P4.34

ARTERIAL STIFFNESS CHANGES DURING ACUTE EXERCISE IN PATIENTS WITH UNTREATED STAGE I ESSENTIAL HYPERTENSION

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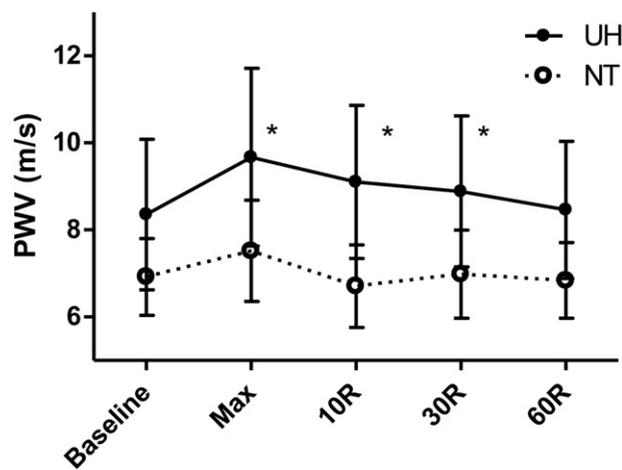
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Objective: Acute exercise exerts potentially harmful effects on the cardiovascular system. However, underlying mechanisms, especially regarding the role of arterial stiffness, remain largely understudied. Thus, we sought to investigate arterial stiffness changes after acute exercise in young patients with untreated, recently diagnosed stage I essential hypertension (UH) compared to healthy individuals (NT).

Design and Methods: We studied 25 consecutive UH (Blood Pressure, (BP): 147.2±6.2/93.5±8.1 mmHg) and 15 age- and sex-matched NT volunteers (BP: 118.2±10.9/75.8±10.3 mmHg). All subjects underwent a treadmill exercise test (Bruce protocol), up to 85% of the maximal heart-rate according to age and gender. Aortic PWV was performed by applanation tonometry (Sphygmocor device) at baseline, at the end of the test (max) and at 10, 30 and 60 minutes later (10R, 30R and 60R respectively).

Results: At all time-points UH exhibited significantly higher aortic PWV and BP levels than NT. Systolic BP rose significantly at max and subsequently fell in levels similar to baseline, in both groups. Aortic PWV increased significantly at max (p<0.001), 10R (p=0.001), and 30R (p=0.003) compared to baseline in UH (Figure). In contrast, no significant changes were observed after exercise in NT.

Conclusions: Arterial stiffness is impaired following high-intensity acute exercise in UH and PWV remains increased despite BP fall in pre-exercise levels. These prolonged effects on arterial stiffness indicate that high-intensity acute exercise is potentially harmful, even in the early stages of essential hypertension.



*, p<0.01 for PWV values compared to baseline in UH