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

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ejection time ( $298.9 \pm 4.9$  ms vs.  $316.8 \pm 4.5$  ms) (t-test,  $p < 0.05$ ). In addition, the normotensives with a family history of hypertension have decreased latency of the baroreflex response ( $7.0 \pm 0.5$  s) compared to the control group ( $10.5 \pm 0.9$  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  $51.3 \pm 2.4$  years, BMI  $30.9 \pm 1.9$  kg/m<sup>2</sup>, duration of AH  $4.6 \pm 1.4$  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/m<sup>2</sup>) were assigned to group 1 and 53 obese patients ( $\text{BMI} > 30$  kg/m<sup>2</sup>) to group 2.

**Results:** The groups were comparable by the age, duration of AH, daily mean BP values. Mean values of IMT (gr1:  $1.02 \pm 0.03$  mm; gr2:  $1.08 \pm 0.04$  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:  $7.6 \pm 0.5$ ; gr2:  $7.01 \pm 0.3$ %) 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  $55.9 \pm 10.7$  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:  $\beta$ stiffness index, Ep-elastic modulus, AC-arterial compliance, PWV $\beta$ -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 $\beta$  ( $r = 0.25$ );  $p$  for all  $< 0.05$ ). However in multiple regression analysis the independent determinants were: age, BMI, mean BP, LVMI, PW, E/e' and PWV $\beta$ .

**Conclusion:** LAR is one of the earliest cardiac structural changes in H that precedes LVH and d.d. Local PWV $\beta$  is an independent determinant of LAR precedes 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 ( $127 \pm 14$  vs  $115 \pm 12$  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 ( $367 \pm 47$  vs  $317 \pm 60$  nM,  $p = 0.01$ , and  $2418 \pm 391$  vs  $2042 \pm 358$  nM/min,  $p < 0.01$ , respectively), but ANG did not affect CAT (peak or ETP). Baseline F1+2 was similar in FH and controls ( $189 \pm 41$  vs  $186 \pm 81$  pM) and unchanged by ANG. Baseline plasmin-antiplasmin complexes were similar in FH and controls ( $96 \pm 16$  vs  $93 \pm 27$   $\mu$ g/L) and increased ( $p < 0.001$ ) similarly by ANG in both groups. PAI-1 activity was similar in both groups at baseline ( $1.3 \pm 1.3$  vs  $1.1 \pm 1.2$  ng/L) and decreased ( $p < 0.001$ ) similarly in both groups, confirming the diurnal variation in fibrinolysis.