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### **P4.53: IMMEDIATE EFFECTS OF SUBMAXIMAL EFFORT ON PULSE WAVE VELOCITY IN PATIENTS WITH MARFAN SYNDROME**

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bisoprolol alone. **PARICIPANTS.** 50 patients with stable angina and COPD were included (88% men, mean age 62.8±7.2 years). All patients received bisoprolol, the dose of which was titrated until the clinical signs of intolerance. Then, the patients were randomized into two groups: patients of the first group continued to take bisoprolol and patients of the second group were added ivabradine (5–15 mg).

**Results:** Combination therapy resulted in further decrease of heart rate (HR) to an average of 62±4 bpm over 6 month of follow-up. This was associated with additional decrease of the number of angina attacks (by 4.7±4.4 per week vs. 2.5±4.7,  $p<0.05$ ) and increase of quality of life ( $p<0.05$ ). Only in combination therapy group there was also the decrease of broncholytics consumption (from 2.9±3.2 to 1.9±2.7 per week,  $p<0.05$ ). Pulse wave velocity (PWV) estimated by Arteriograph (TensioClinic «Meditech») decrease significant in both groups, but no difference delta PWV between groups.

**Conclusion:** The combination of tolerable doses of bisoprolol and ivabradine is safe and allows to achieve adequate HR decrease. This is associated with maximal antianginal effect, decrease in the need for broncholytic therapy, improvement of the quality of life compared with the treatment with bisoprolol alone. We did not find the influences of ivabradine on the arterial stiffness.

#### P4.51

##### NONINVASIVE EVALUATION OF STRUCTURAL AND FUNCTIONAL CHANGES IN THE HEART AND LARGE ARTERIES IN PATIENTS WITH PRIMARY ALDOSTERONISM

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**Background** Only few studies, in small groups of patients, have evaluated large arteries alterations in primary aldosteronism(PA). In addition, the assessment of cardiac and vascular involvement has not been simultaneously performed.

**Aim** of the study was to evaluate cardiac, vascular and renal complications in patients with PA.

**Patients and Methods:** 70 patients with PA(age 49±6years,27 F) and 70 essential hypertensives (EH) matched for age, sex, BMI and BP, underwent laboratory examinations, cardiac and carotid ultrasound and PWV.

**Results:** no differences in age, sex, BMI, BP and HR were observed. Cholesterol was lower in PA (195±37 vs 215±33 mg/dl,  $p<0.05$ ); no difference in tryglicerides, glucose and uric acid was observed. In PA eGFR(MDRD) was greater in comparison to EH(96±22 vs 89±17mL/min/1.73m<sup>2</sup>,  $p=0.05$ ). LV mass index(LVMI) and relative wall thickness(RWT) were significantly greater in PA vs EH(LVMI 42±13 vs34±7 g/m<sup>2</sup>.7,  $p<0.001$  and RWT 0.34±0.08 vs 0.31±0.04,  $p<0.001$ ). At TDI Emvel was significantly lower, and Evel/Em vel was significantly higher in PA (9.5±2.9 vs 10.5±2.7,  $p<0.05$  and 9.2±4.4 vs 7.6±2.7,  $p<0.05$ , respectively). No difference was observed in carotid prevalence of thickening or plaques and in IMT (Meanmax 1.02±0.25 vs 0.98±0.18,  $p$  ns, CBMMax 1.05±0.26 vs 1.01±0.16,  $p$  ns), as well as in PWV(10.8±2.1 vs 10.8±1.6 m/sec,  $p$  ns).

**Conclusions:** In this large group of patients with PA a significant increase in LVMI and concentric geometry, associated with a worse diastolic function were observed. Opposite to previous findings, no difference in aortic stiffness and carotid structure was observed.

#### P4.52

##### CORONARY ARTERY DISEASE AND STROKE IN TYPE 2 DIABETIC PATIENTS: POSSIBLE ROLE OF A RAISED CENTRAL PULSE PRESSURE

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**Background:** Whether large artery static and dynamic properties are different in type 2 diabetics with coronary or cerebrovascular disease (DM-CVD) as compared to patients free of clinical complications (DM) is debated. **Aim:** To evaluate common carotid (CCA) IMT, local and aortic stiffness in DM-CVD as compared to DM and controls (CTRL).

**Methods:** thirty-five nondiabetic normotensive CTRL, 130 diabetics without complications (DM) and 25 diabetics with CAD and/or previous stroke/TIA (DM-CVD) (Tab 1) underwent radiofrequency (RF)-based ultrasound (QIMT® and QAS®, Esaote) of CCA, that provides automatic measurement of far-wall IMT, local stiffness ( $\beta$ -index) and contour wave analysis yielding local pulse pressure (PPc). Carotid-femoral pulse-wave velocity (PWV) was also measured (Complior).

**Results:** Adjusting for sex, age and smoking habit, CTRL and diabetics differed significantly ( $p<0.05$ ) in PWV (9.0±1.9 vs. 11.4±2.5 m/s), CCA IMT (619±146 vs. 736±169  $\mu$ m) and PPc (37±9 vs. 46±14 mmHg), but not in  $\beta$ -index (10.1±2.9 vs. 12.5±4.9;  $p=0.23$ ). Within diabetics, after adjustment for confounders, DM-CVD showed higher ( $p<$ at least 0.05) PPc but not CCA IMT,  $\beta$ -index and brachial PP (PPb) than DM (Tab 2).

Table 1.

| Groups | Age   | BMI   | Glycemia | SBPb    | PPb   |
|--------|-------|-------|----------|---------|-------|
| CTRL   | 56±8* | 26±4* | 88±10*   | 123±12* | 46±8* |
| DM     | 62±8  | 29±5  | 146±42   | 136±18  | 57±16 |
| DM-CVD | 66±7  | 29±4  | 138±42   | 136±23  | 62±18 |

Table 2.

| Groups | PWV      | IMT      | $\beta$ -index | SBPc    | PPc    |
|--------|----------|----------|----------------|---------|--------|
| CTRL   | 9.0±1.9* | 619±146* | 10.1±2.9*      | 115±12* | 37±9*  |
| DM     | 11.3±2.4 | 726±173  | 12.4±4.3       | 123±16  | 44±13° |
| DM-CVD | 12.0±3.5 | 794±129  | 14.6±6.8       | 127±19  | 52±16  |

\*:  $p<0.05$  in CTRL vs DM and DM-CVD; °:  $p<0.05$  in DM vs DM-CVD

**Conclusions:** central PP appears to be the only structural/functional large artery measure significantly different in diabetic patients with or without cardiovascular complications, despite similar glucose levels and BMI. Contour wave analysis by ultrasound provides additional information beyond IMT and local stiffness indices.

#### P4.53

##### IMMEDIATE EFFECTS OF SUBMAXIMAL EFFORT ON PULSE WAVE VELOCITY IN PATIENTS WITH MARFAN SYNDROME

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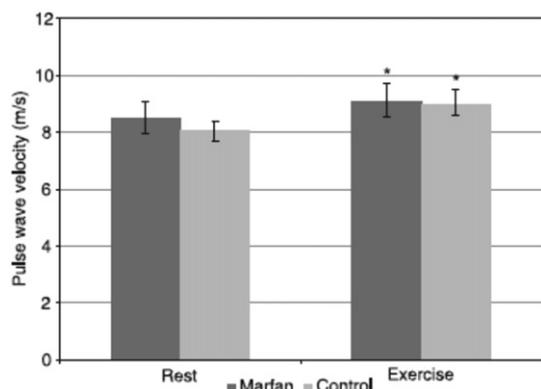
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Marfan syndrome (MS) is a dominant autosomal disease caused by mutations in chromosome 15, the locus controlling fibrillin 1 synthesis, and may exhibit skeletal, ocular, cardiovascular, and other manifestations. Pulse wave velocity (PWV) is used to measure arterial elasticity and stiffness and is related to the elastic properties of the vascular wall. Since the practice of exercise is limited in MS patients, it was of interest to analyze the acute effect of submaximal exercise on aortic distensibility using PWV and other hemodynamic variables in patients with MS with either mild or no aortic dilatation. PWV and physiological variables were evaluated before and after submaximal exercise in 33 patients with MS and 18 controls. PWV was 8.51 ± 0.58 at rest and 9.10 ± 0.63 m/s at the end of exercise ( $P = 0.002$ ) in the group with MS and 8.07 ± 0.35 and 8.98 ± 0.56 m/s in the control group, respectively ( $P = 0.004$ ). Comparative group analysis regarding PWV at rest and at the end of exercise revealed no statistically significant differences. The same was true for the group that used  $\beta$ -blockers and the one that did not. The final heart rate was 10% higher in the control group than in the MS group ( $P = 0.01$ ). Final systolic arterial pressure was higher in the control group ( $P = 0.02$ ). PWV in MS patients with mild or no aortic dilatation did not differ from the control group after submaximal effort.

**Table 1.** Anthropometric and echocardiographic data of patients with Marfan syndrome and controls.

|                          | Marfan (N = 33) | Control (N = 18) |
|--------------------------|-----------------|------------------|
| Age (years)              | 19 ± 8          | 20 ± 5           |
| Weight (kg)              | 60.9 ± 12.6     | 62.4 ± 11.9      |
| Height (m)               | 1.78 ± 0.10*    | 1.71 ± 0.10      |
| BMI (kg/m <sup>2</sup> ) | 19.32 ± 3.93    | 21.28 ± 2.63     |
| ECHO (mm)                | 33 ± 7*         | 28 ± 2           |

Data are reported as means ± SD. BMI = body mass index; ECHO = echocardiogram. \*P ≤ 0.05 compared to control group (t-test).



**Figure 1** Pulse wave velocity values at rest and at the end of exercise for the patients with Marfan syndrome (N = 33) and for the control group (N = 18). Data are reported as absolute values. \*P < 0.05 compared to rest (t-test).

#### P4.54

##### SHORT-TERM HIGH SALT DIET REDUCES BRACHIAL ARTERY ENDOTHELIAL FUNCTION IN THE ABSENCE OF CHANGES IN BLOOD PRESSURE

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High salt diets are associated with impaired vascular relaxation, hypertension, and cardiovascular disease. We hypothesized that 1) short-term high salt intake impairs brachial artery endothelial function in the absence of changes in blood pressure or vascular stiffness and 2) acute exercise reverses endothelial function after elevations in salt. Healthy, inactive subjects (n=11) were fed 6 mg of sodium chloride for 7 days or normal diet and then underwent a single progressive 15 minute leg press WL session. Brachial artery flow-mediated dilation (FMD) and nitroglycerin (NTG; 0.4 mg) dilations were measured with ultrasound at baseline, after 7 days of high salt or normal salt intake, and before and after WL. Pulse wave velocity was determined before and after high salt. All subjects had normal blood pressures (mean SBP: 117±12 mmHg) before and after high salt and exercise.

Circulating plasma renin was reduced after high salt. Brachial artery FMD was reduced after high salt (12±0.7% vs. 7.5±0.9; p=0.003). Acute exercise reduced brachial FMD on normal salt (9.6±0.9% vs. 6.6±1; p=0.03) and there was no effect of acute exercise on FMD after high salt (7.1±0.2%; p=0.6 vs. pre exercise). Endothelium-independent responses to NTG (mean: 29±2%) and pulse wave velocities were similar before and after high salt and between groups. These data indicate 1) Elevated salt intake for 7 days impairs brachial artery endothelial function in the absence of changes in blood pressure or vascular stiffness and 2) acute resistance exercise does not restore arterial function after high salt intake.

#### P4.55

##### CHANGES IN CAROTID-RADIAL PULSE WAVE VELOCITY AND RADIAL-FINGER TIP SKIN VASCULAR BED TRANSIT TIME DURING AND AFTER GRADED AEROBIC EXERCISE

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Physical activity is known to have beneficial effects on prevention of cardiovascular disease and on microcirculation. The aim of our study was to measure the carotid-radial pulse wave velocity and the transit time for blood to reach the finger tip skin capillary bed in middle aged healthy subjects before, during and 20 minutes after aerobic exercise.

Experiments were performed on 9 males, 47±7 years old. We measured ECG, arterial blood pressure, skin blood flow (SkBF) and carotid or radial pulse with a tonometer. After 5 minutes rest subjects started a graded exercise at the workload of 40 W in steps of 50W lasting 3 minutes each until 85% of the estimated maximal heart rate was reached. After ceasing exercising, the parameters were measured for subsequent 25 minutes. Carotid-radial pulse wave velocity (c-rPWV) and transit time for the pulse to propagate from the radial artery to finger tip skin capillary bed (r-sk tT) were calculated.

Our results revealed that c-rPWV was significantly smaller and r-sk tT significantly longer 20 minutes after exercise compared to control values while the heart rate was still significantly elevated (65,4±5,2 and 77,5±8,2). A linear correlation between c-rPWV and corresponding RR duration during exercise was found (p<0.05) but no correlation between r-sk tT and RR.

We conclude that during exercise increased sympathetic tone is the main reason for increasing c-rPWV, but other mechanisms should contribute to the regulation of the finger tip skin microcirculation, where termoregulation plays a major role.

#### P4.56

##### INCREASED LEFT VENTRICULAR ELASTANCE AT END-EJECTION IS ASSOCIATED WITH LOWER ARTERIAL COMPLIANCE AND REDUCED VENTRICULAR RELAXATION

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**Background:** The response of a normal left ventricle (LV) to an increase in afterload is to increase its rate of relaxation. In diastolic dysfunction LV relaxation is impaired and passive chamber stiffness is increased. We investigated the potential of incremental LV elastance measures, as obtainable by non-invasive means, to assess LV relaxation performance.

**Methods and Results:** We obtained paired central arterial pressure and LV volume curves from PulseCor and 3D-echo recordings in 62 consecutive

| E <sub>ee</sub>     | mmHg/ml | Lower E <sub>ee</sub> (n=31) | higher E <sub>ee</sub> (n=31) | p    | Correlation with E <sub>ee</sub> |         |
|---------------------|---------|------------------------------|-------------------------------|------|----------------------------------|---------|
|                     |         | 8 ± 2                        | 17 ± 5                        |      | Pearson's r                      | p       |
| HR                  | 1/min   | 59 ± 9                       | 64 ± 11                       | .054 | 0.26                             | .04     |
| SBP                 | mmHg    | 135 ± 13                     | 144 ± 17                      | .026 | 0.42                             | .001    |
| E <sub>se</sub>     | mmHg/ml | 2.5 ± 0.8                    | 3.3 ± 1.3                     | .006 | 0.41                             | .001    |
| dp/dt <sub>ee</sub> | mmHg/s  | -389 ± 122                   | -525 ± 188                    | .001 | -0.63                            | <.0001  |
| SV/PP               | ml/mmHg | 0.88 ± 0.26                  | 0.69 ± 0.23                   | .004 | -0.50                            | <0.0001 |
| e'                  | cm/s    | 7.5 ± 1.9                    | 6.4 ± 1.3                     | .009 | -0.33                            | .008    |
| E/e'                | -       | 8.4 ± 2.4                    | 10.0 ± 3.5                    | .025 | 0.26                             | .04     |
| EF                  | -       | 0.63 ± 0.06                  | 0.65 ± 0.06                   | .4   | 0.07                             | .59     |
| Ees/Ea              | -       | 1.80 ± 0.42                  | 1.89 ± 0.43                   | .4   | 0.06                             | .67     |

Data given as mean±SD.