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P4.54: SHORT-TERM HIGH SALT DIET REDUCES BRACHIAL ARTERY ENDOTHELIAL FUNCTION IN THE ABSENCE OF CHANGES IN BLOOD PRESSURE

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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 ²)	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).

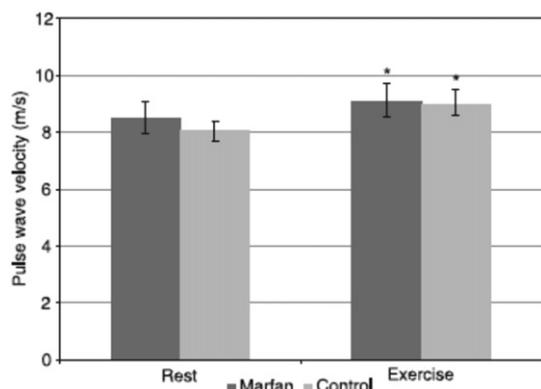


Figure 1 Pulse wave velocity values at rest and at the end of exercise for the patients with Marfan syndrome (N = 33) and for the control group (N = 18). Data are reported as absolute values. *P < 0.05 compared to rest (t-test).

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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.

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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.

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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 _{ee}	mmHg/ml	Lower E _{ee} (n=31)	higher E _{ee} (n=31)	p	Correlation with E _{ee}	
		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 _{se}	mmHg/ml	2.5 ± 0.8	3.3 ± 1.3	.006	0.41	.001
dp/dt _{ee}	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.