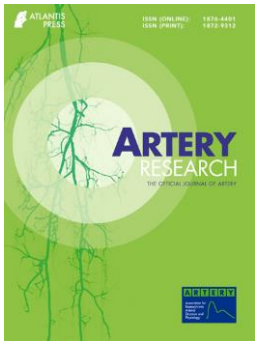

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P4.56: INCREASED LEFT VENTRICULAR ELASTANCE AT END-EJECTION IS ASSOCIATED WITH LOWER ARTERIAL COMPLIANCE AND REDUCED VENTRICULAR RELAXATION

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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).

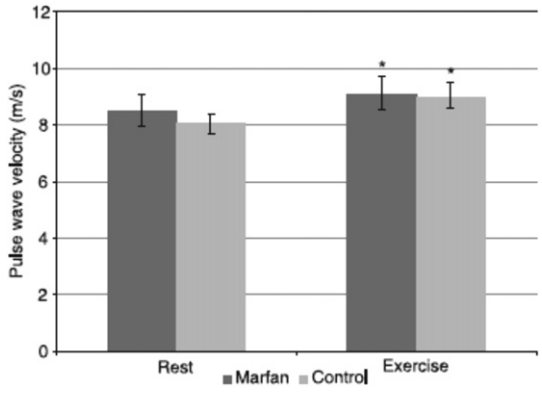

Figure 1 Pulse wave velocity values at rest and at the end of exercise for the patients with Marfan syndrome (N = 33) and for the control group (N = 18). Data are reported as absolute vales. *P < 0.05 compared to rest (*t*-test).

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_{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.

subjects aged 71±6 yrs (mean±SD) from an existing community study. Incremental LV elastances at the start (E_{se}) and end of ejection (E_{ee}) were calculated as the ratio of dp/dt and dV/dt at corresponding positions in time. In our analysis we considered the inter-relationships of E_{ee} with heart rate (HR), systolic blood pressure (SBP), pressure relaxation rate (dp/dt_{ee}), arterial compliance (SV/PP), mitral annulus velocity e', E/e', ejection fraction (EF) and the classic non-invasive ventricular-vascular coupling index (Ees/Ea). Univariate correlations (Table) as well as stratification according to lower and higher E_{ee} groups showed that a higher E_{ee} was associated with a higher E_{se} and with lower arterial compliance and reduced ventricular relaxation rate (e'), despite increased dp/dt_{ee}. EF and Ees/Ea were not associated with any of these measures.
Conclusions: An increased E_{ee} reflects slowed ventricular relaxation, which may be due to the impact of reduced arterial compliance on LV diastolic performance. The classic non-invasive ventricular-vascular coupling index Ees/Ea did not reveal such a relationship.

P4.57
STIFFNESS OF THE LARGE ARTERIES IN INDIVIDUALS WITH AND WITHOUT DOWN SYNDROME

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Background: Down syndrome (DS) is known to cause premature aging in several organ systems[1]. In this controlled study, the possibility of changes in the large arteries due to aging was evaluated in patients with DS[1].
Methods: Eighty-two subjects of both genders were selected. The DS group had 41 active subjects. The control group was consisted of 41 healthy matched for age and gender. Carotid–femoral pulse wave velocity was obtained as an index of aortic stiffness using an automatic noninvasive method[2].
Results: The general characteristics of the groups and the main results are shown in Table 1 and Figure.
Conclusion: Despite evidence in the literature that patients with DS undergo early aging[1], this process does not seem to affect the large arterial trunks[3]. Considering that DS presents with chronic hypotension, it is reasonable to propose that the prolonged reduction of arterial distending pressure may contribute to functional preservation of the arteries in patients with Down syndrome
Keywords: aging, Down syndrome, pulse wave velocity, arterial stiffness

References

1 Nakamura E, Tanaka S. Biological ages of adult men and women with Down's syndrome and its changes with aging. *Mech Ageing Dev.* 1998; 105:89–103.
2 Asmar R, Benetos A, Topouchian J, et al. Assessment of arterial distensibility by automatic pulse wave velocity measurement. *Hypertension.* 1995; 26:485–490.

Table 1 Anthropometric and hemodynamic characteristics in the experimental groups.

	DOWN SYNDROME (n=41)	CONTROL (n=41)	*P* value
Age (years) Variance	21 ± 1 (13-42)	21 ± 1 (13-42)	-
Weight (kg)	55 ± 2	61 ± 2	< 0.05
Height (m)	1.47 ± 0.01	1.64 ± 0.01	< 0.001
BMI ($kg.m^{-2}$)	25 ± 1	22 ± 1	< 0.01
WHR	0.88 ± 0.01	0.80 ± 0.01	< 0.001
SBP (mmHg)	106 ± 2	117 ± 2	< 0.001
DBP (mmHg)	66 ± 2	77 ± 2	< 0.001
MAP (mmHg)	80 ± 1	90 ± 1	< 0.001
HR(bpm)	74 ± 2	76 ± 2	NS
PWV (m/s)	7.51 ± 0.14	7.84 ± 0.12	< 0.05

Data are expressed as mean ± standard error (SEM).
Abbreviations: DS = Down syndrome; BMI = body mass index; WHR = waist-hip ratio; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = heart rate; PWV= pulse wave velocity; NS = not significant.

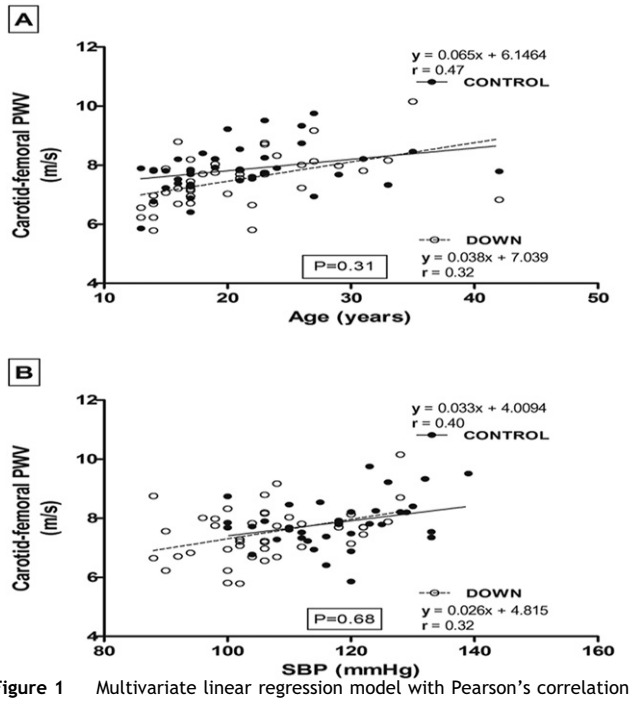

Figure 1 Multivariate linear regression model with Pearson's correlation coefficient between age, systolic blood pressure, and carotid–femoral pulse wave velocity in controls and subjects with Down syndrome.

3 Rodrigues AN, Coelho LC, Gonçalves WLS, at al. Stiffness of the large arteries in individuals with and without Down syndrome. *Vascular Health and Risk Management* 2011; 7: 375–381

P4.59
ASSOCIATION BETWEEN ENDOTHELIAL NO SYNTHASE POLYMORPHISM AND AORTIC STIFFNESS

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Background: Recently, rs3918226 polymorphism in the promoter region of endothelial NO synthase (NOS3) was strongly associated with arterial hypertension in a large genome-wide association study*. We investigated whether this polymorphism is associated with arterial phenotypes in a Czech general population.
Methods: In a pilot study, we genotyped 101 untreated subjects (age, 54.0 years; 51.5% women, 30.7% smokers). Arterial properties were measured using SphygmoCor. In multivariate-adjusted analyses, we assessed effect of **rs3918226 on** aortic pulse wave velocity (aPWV) and augmentation index (AIx). As independent covariates we considered sex, age, MAP, heart rate and smoking.
Results: Frequency of rs3918226 genotypes were CC 85.2%, CT 14.8%, and TT 0%. Carriers of mutated T allele tended to have higher both aPWV (8.59±0.45 vs. 7.77±0.18 m/s; P=0.098) and AIx (91.77±3.56 vs. 85.89±1.45%; P=0.13) compared to CC homozygotes. These associations were modified by smoking. In smokers we observed similar trend as in the whole population ($0.067<P<0.19$), while in nonsmokers we did not find any association ($P\geq0.50$). We did not observe any association between blood pressure and the polymorphism under study ($P\geq0.67$).
Conclusion: This is first study to explore the association of rs3918226 polymorphism in NOS3 gene with arterial properties. We found marginally higher aPWV and AIx in carriers of mutated T allele in this pilot study. We hypothesize that genetic modulation of intermediate arterial phenotypes might lead to higher blood pressure. As the prevalence of