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Influence of physical effort on aortic stiffness in young male athletes

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Abstract. – OBJECTIVE: The progression of cardiovascular disease is blunted by regular exercise training as a common non-pharmacological treatment. Recent findings have confirmed that central aortic pressure is more strongly related to cardiovascular events than brachial blood pressure. The aim of the study was to evaluate the influence of a single bout of significant physical effort on central aortic pressure and pulse wave velocity in young male athletes.

PATIENTS AND METHODS: The study group consisted of 16 healthy male athletes undergoing regular endurance training. The subjects of the study (21.6 ± 2.85 years of age) underwent a submaximal exercise test consisting of cycling for 30 minutes. Aortic pulse wave velocity (PWV) and derivatives (augmentation index set to 75 heart beats, AIx75; pulse pressure amplification, PPA), ejection duration (ED), subendocardial viability ratio (SEVR) and central blood pressure were examined before and after the exercise test Blood pressure and pulse waveform were evaluated in the supine position after a 15-minute rest by means of the oscillometric method the oscillometric method.

RESULTS: Comparing the rest condition to the period immediately following the exercise test, athletes showed lower central than peripheral systolic blood pressure both before (129 ± 11 mmHg and 112 ± 8 mmHg, respectively, $p < 0.001$) and after (130 ± 10 mmHg to 112 ± 8 mmHg, respectively, $p < 0.001$) the test. They also showed a decrease of ED from 339.7 ± 44.4 ms to 326.9 ± 41.4 ms ($p < 0.02$) and an increase of PPA from 136.2 ± 5.4% to 140.3 ± 5.0% ($p < 0.02$), whereas PWV, AIx75 and SEVR changed insignificantly.

CONCLUSIONS: We confirm that PPA is sensitive to an instant change in aortic elasticity. Furthermore, the hemodynamic response to a single physical effort composed of shorter ejection time and increased relative elasticity of the aorta prevents impairment of oxygen supply to the heart musculature.

Key Words
Pulse wave velocity, Pulse pressure amplification, Central blood pressure, Cardiovascular outcomes.

Introduction

Agreat emphasis has been placed on the role of arterial stiffness in the development of cardiovascular (CV) diseases[1,2]. A loss of distensibility in the central elastic arteries, particularly the aorta, compromises their ability to act as a buffer to the blood volume ejected by the left ventricle and raises systolic blood pressure[3]. This affects coronary blood flow in particular, and increases the risk of cardio- and cerebrovascular events[4-6]. It has been suggested that measurement of the central aortic pressure has greater prognostic capability than that of conventional brachial blood pressure (BP)[7-10]. Some findings have confirmed that central aortic pressure is more strongly related to vascular hypertrophy, the extent of atherosclerosis and cardiovascular events than brachial BP[7,8,11].

The progress of civilization has a tendency to alter the lifestyle. A decreased exercise capacity has been associated with an increased risk of all-cause mortality and major adverse cardiovascular events[12,13]. Physiological adaptation to exercise includes structural, neurohumoral, autonomic, metabolic and regulatory mechanisms, which lead to an increase in cardiac output[14]. Moreover, coronary perfusion improves and myocardial oxygen demand decreases[15]. It has been confirmed that aerobic training significantly reduces CV risk[14,16]. Furthermore, regular exercise slows the progression of cardiovascular disease as a common non-pharmacological treatment[17]. However, even among well-trained athletes CV events do occur[18].

In light of this, we decided to evaluate the instant hemodynamic response of the cardiovascular system to a single bout of significant physical effort, in particular for estimating central aortic pressure and cardiovascular risk factors such as arterial stiffness and parameters describing pulse waveform during recovery in young male athletes.

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Patients and Methods

Study Procedures

The study group consisted of 16 healthy male athletes undergoing regular endurance training recruited among football and water polo players (clinical characteristics of the study group are presented in Table I). To participate in the study, athletes filled in a medical history questionnaire (according to the guidelines of the American College of Sports Medicine – ACSM) and underwent a medical examination together with an ECG assessment to reduce the risk of sudden cardiac events (according to guidelines for pre-participation screening[19]).

Exclusion criteria were: age ≥ 25 years, presence of known CV risk factors, hypertension, diabetes mellitus, peripheral arterial disease, alcohol, obesity, smoking, history of vascular surgery, arrhythmia, cardiac valvulopathy or myocardial ischemia, any medical drug treatment or any disorders in ECG.

The athletes included underwent an exercise test. Participants did not eat or drink for two hours prior to the test. The exercise test was based on cycling with the parallel registration of respiratory system parameters employing a cycle ergometer and K4b2 spirometer (Cosmed, Pavona di Albano, RM, Italy). The exercise test protocol started with a warm-up for three minutes at a load level of 50 watts. The load level increased gradually to 150 watts. The athletes continued with this load for 30 minutes. Next, the load was gradually decreased to zero watts over three minutes. The participants underwent the exercise test at a submaximal load level confirmed by the metabolic equivalent of oxygen (METs) reached by the subjects in the test (mean 8.18 ± 0.02 MET, range from 6.6 to 9.8 MET).

Non-invasive Assessment of Peripheral Blood Pressure and the Pressure Waveform

Blood pressure and pulse waveform were evaluated in the supine position after a 15-minute rest by means of the oscillometric method employing Vasotens® technology (BP Lab, Nizhny Novogorod, Russia) with a conventional cuff adapted to the arm circumference[20,21]. The measurements took place at a comfortable room temperature, while avoiding external stress influences. Examination of the blood pressure and pulse waveform of the participants continued until repetitive results were achieved. The measurements were taken on the left arm before and 15 minutes after the physical test. The peripheral pulse pressure curve was registered at the brachial artery during a step-by-step deflation.

Table I. Characteristic of study group.

Variables	Mean	±SD
Age (years)	22	3
Height (cm)	180.0	7.5
Weight (kg)	81.0	9.0
BMI (kg/m^2)	25.0	2.3

Note: Values for n = 16.

Blood Pressure and Pulse Waveform Analysis

The analysis included heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and pulse pressure (PP). Assessment of the central aortic pressure parameters using the "transfer function" was based on analysis of the peripheral arterial waveform obtained by oscillometry[22]. In particular, we analyzed mean blood pressure (MBP), aortic systolic blood pressure (SBPao), aortic diastolic blood pressure (DBPao), aortic mean blood pressure (MBPao) and aortic pulse pressure (PPao).

Pulse waveform analysis was applied to assess peripheral and central hemodynamics employing pulse wave velocity (PWV), reflected wave transit time (RWTT), ejection duration (ED) and the artery stiffness index (ASI). On the basis of the pulse waveform, the augmentation index set to 75 heart beats per minute (AIx75), pulse pressure amplification (PPA) and subendocardial viability ratio (SEVR) were calculated[23].

Statistical Analysis

Results are presented as mean ± standard deviation (SD). The Shapiro-Wilk test was performed to confirm the normal distribution of the data. Parameters before and after the physical test were compared using a dependent Student's t-test. For parameters with non-normal distribution, the Wilcoxon signed-rank test for small groups was applied. Results were regarded as statistically significant at $p < 0.05$.

Ethics Statement

The study was approved by the Medical Ethics Committee of the Pomeranian Medical University in Szczecin, Poland (nr KB-0012/55/13), and all participants gave written informed consent.

Results

After the physical test, the athletes revealed a non-significant increase in heart rate compared with the resting period before the test (60 ± 8 bpm to 68

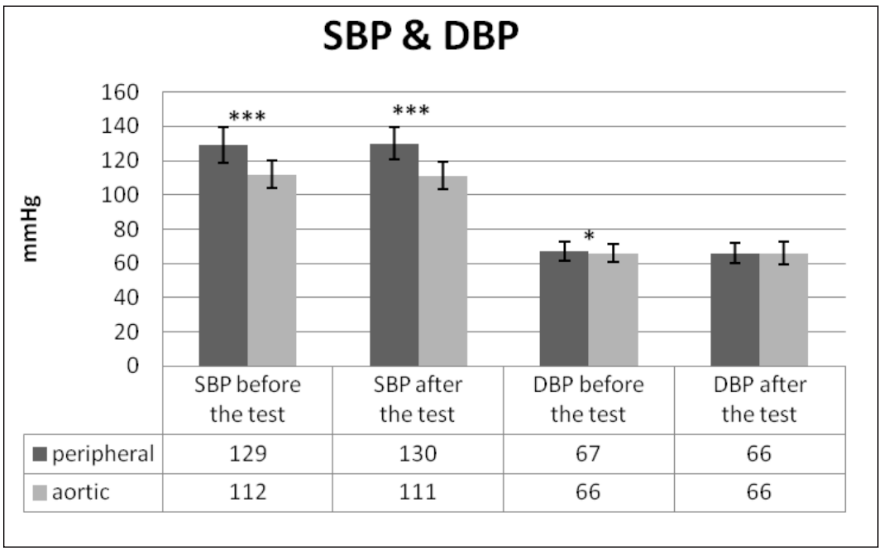

Figure 1. Analysis of blood pressure. Comparison of peripheral and central systolic (SBP) and diastolic blood pressure (DBP). Asterisks denote significant differences (*$p < 0.05$; ***$p < 0.001$).

± 18 bpm, respectively). Nevertheless, we noted a significantly decreased ED after the physical test.

Comparison of the brachial artery and aortic blood pressure values presented in Figure 1, shows higher peripheral than aortic pressures. Before the test, systolic pressure was 129 ± 11 mmHg in the brachial artery and 112 ± 8 mmHg in the aorta (significance $p < 0.001$), and after the test these values were 130 ± 10 mmHg and 111 ± 8 mmHg, respectively (significance $p < 0.001$). At the same time, before test diastolic blood pressure was significantly higher in the brachial artery than in the aorta (67 ± 5 mmHg and 66 ± 6 mmHg, respectively; significance $p < 0.05$). There was no significant difference between peripheral and central diastolic blood pressure evaluated after the test (66 ± 6 mmHg and 66 ± 6 mmHg, respectively).

Regarding the hemodynamic response, especially with respect to arterial stiffness, comparing the resting condition with the recovery period following the exercise test (data presented in Table II) athletes revealed a decrease of RWTT parallel to an increase of PWV, but both changed insignificantly.

Central pulse waveform analysis revealed an insignificant increase in AIx75, but significant increase of PPA after the physical test. Nevertheless, we observed an insignificant change of SEVR.

Discussion

The level of systolic pressure modifies pulse pressure, which in turn affects microvascular function and, when increased, it may lead to tissue dysfunction, particularly of highly sensitive organs such as the heart, brain and kidneys[24,25]. The athletes examined here exhibited significantly higher peripheral than aortic systolic blood pressure. It

Table II. Parameters of aortic pulse wave.

Variables	Before exercise test		After exercise test		Significance
	mean	±SD	mean	±SD	*p*-value
PWV, m/s	8.98	0.52	8.31	0.52	N.S.
RWTT, ms	167.7	8.3	163.4	9.0	N.S.
AIx75, %	-11.0	5.3	-7.1	10.9	N.S.
PPA, %	136.2	5.4	140.3	5.0	0.019
ED, ms	339.7	44.4	326.9	41.4	0.013
SEVR, %	134.4	42.9	135.3	53.9	N.S.
ASI, mmHg	156.7	33.5	163.2	25.4	N.S.

Note: Values for n = 16; PWV, pulse wave velocity; RWTT, reflected wave transit time; AIx75, augmentation index set to 75 heart beats per minute; PPA, pulse pressure amplification; ED, ejection duration; SEVR, subendocardial viability ratio; ASI, aortic size index; N.S, not significant.

is believed that the high compliance of the arterial system connected with the young age of the subjects specifically amplifies the pulse pressure wave and thus central and peripheral pressure differ from one another[26]. Contraction of the left ventricle accompanied by ejection of blood creates a pulse wave. This wave travels along the aorta and is reflected by resistance vessels. PWV directly describes the stiffness of the aortic wall and influences the returning time of the reflected wave[27]. In healthy young men with compliant arteries, the reflected pressure wave returns to the heart during diastole and raises diastolic pressure[28]. This phenomenon protects the microvascular circulation from damage and, moreover, improves blood flow through the coronary system, carried out mainly during diastole. This effect is much more clearly seen in a high heart rate[23]. Nevertheless, it should be emphasized that the aortic blood pressure opposes ejection and affects myocardial tension during contraction, thus creating the condition of blood flow through the coronary system[24]. Therefore, significantly lower aortic than brachial artery pressure improves the conditions for coronary flow. However, in our work independent of pressure type and evaluation region, a single significant physical effort had no significant affect on pressure.

Elastic properties of arteries are evaluated by employing the phenomenon of pulse wave reflection and are expressed as aortic augmentation index (AIx) and pulse pressure amplification (PPA). These parameters indirectly reflect the degree of arterial stiffness and basically describe the pulse waveform. The new method for quantifying indicators of cardiovascular risk like pulse wave velocity and independent indicators of central aortic pressure uses the oscillometric methodology[29]. Both PWV and AIx increase with cardiovascular risk factors[30]. A strong positive correlation between AIx and PWV has been observed since a higher PWV will result in earlier arrival of reflected waves that thereby augment central systolic pressure during early systole[31]. In the ACCT trial[32] the absence of a linear relationship between particular parameters describing arterial stiffness has been demonstrated. The study revealed an age-related change in AIx and PWV. The young individuals examined exhibited a faster increase in AIx than of PWV. This could mean that young individuals with an elastic arterial system require more sensitive indicators than PWV.

Our research showed that a single significant physical effort increases PPA without change of PWV and AIx75. PPA shows the relation between central and peripheral arterial stiffness, expressed as a percentage increase in pulse pressure value size dependent on pulse wave reflection. The PARTAGE study[33] revealed a significant relation between PPA and the prevalence of cardiovascular diseases. Ribeiro et al[34] observed that a single physical effort, consisting of treadmill walking, does not change AIx75 after the test in healthy young males. Thus, PPA seems to be a very sensitive index evaluating relative arterial stiffness. This application confirms Lemogoum's study[30]. The author recorded that some drugs may change central pulse pressure and AIx without influence on aortic PWV, and suggested they had a greater effect on wave reflection than on aortic stiffness.

In our findings, parallel to the increase of PPA, ED decreased. This phenomenon could be explained by the increased contractility of the heart in the post-exercise period[35]. Usually, increased contractility of the heart leads to increased systolic pressure[36]. However, increased PPA related to the increase of elastic properties of the aorta seems to compensate for the possible increase in systolic pressure in our study. This may explain why differences in aortic systolic blood pressure before and after the exercise test do not occur. Nevertheless, a high inotropic state increases oxygen demand and need for blood of the heart[27]. Especially bad conditions occur in the subendocardial layer due to the transmitted high pressure that develops in the heart ventricles and among the contracting heart muscle fibers[23]. This leads to almost complete occlusion of the vessels and stops the blood flow in the microcirculation during ventricular contraction. Thus, the blood and oxygen supply is maintained mainly in the diastolic phase. The index reflecting the relationship between blood supply and oxygen requirement is SEVR[23]. In our study, the SEVR value did not deteriorate. This may suggest that the blood supply to the heart is not impaired, even if the inotropic state of the heart is increased. Our findings are in line with the hypothesis that a highly endurance-trained heart could demonstrate higher oxygen extraction from blood and greater myocardial efficiency[15].

It has been suggested that regular exercise delays or prevents the age-related increase in arterial stiffness[12,37]. Aerobic exercise is associated with a decrease in large elastic artery stiffness. Kakiyama et al[38] observed a decrease in the magnitude of PWV after a course of endurance training in young sedentary men. This

effect cannot be maintained without continuing physical exercise. In addition to its preventive action, aerobic exercise can also be viewed as a therapy for destiffening large elastic arteries with age[39].

Endurance athletes adapt to an almost constant influence of stress factors connected with physical effort[15]. This adaptation is particularly visible in the cardiovascular system. Laurent et al[40] observed lower PWV in endurance athletes than in sedentary young men. Tanaka et al[37] revealed the absence of an age-related increase in arterial stiffness, evaluated by PWV and AIx, in highly physically active women, in contrast to their sedentary counterparts.

Although the beneficial effects of physical activity have been well documented, sudden cardiac death (SCD) among athletes is still a reality. It has been pointed out that the reasons for SCD are closely related to age. Ferreira et al[18] reported cardiac congenital abnormalities characteristic of athletes younger than 35, in particular. For older athletes, coronary heart disease and impaired coronary flow are the main reasons for SCD. This conflicts with the benefits of physical activity and requires further investigation in order to clarify the relationship between them.

Conclusions

A single significant physical effort in young male athletes increases relative aortic elasticity depicted by pulse pressure amplification. Especially for young male athletes, PPA is a parameter sensitive to instant change in aortic elasticity in comparison with pulse wave velocity and an augmentation index set to 75 heart beats per minute. The hemodynamic reaction to a single physical effort compares shorter ejection time with increased relative elasticity of the aorta and thus impairment of oxygen supply to the heart musculature does not occur.

Acknowledgements

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Conflict of Interests

No conflicts of interest, financial or otherwise, are declared by the authors.

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