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Blood pressure and geometry of the left ventricle in power sports athletes of heavy weight categories

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***Purpose*—To research blood pressure and heart geometry in power sports athletes of heavy weight categories.**

***Material and methods*—645 representatives of the power sports (weightlifting, powerlifting, bodybuilding) having sports qualification of CMS, MS, IMS with the average body weight of 102.7±6.4 kg were examined. Everything to the examined athletes along with survey carried out the standard electrocardiogram of rest, double measurement of the ABP, a transthoracic echocardiography.**

***Results*—A survey of 645 athletes of power sports of heavy weight categories showed that 238 (37%) athletes have high blood pressure (systolic blood pressure - 157.4 ± 5.6, diastolic blood pressure - 91.2 ± 5.3) and violation of left ventricular geometry. Reliably in hypertensive athletes, compared with normotensive athletes, the following heart parameters were increased: TIS by 0.1mm (p <0.01), TPWLV by 0.2mm <0.01), DRV by 4.2 mm (p <0.01), LVMM by 32.2g (p <0.01), LVMI by 17.8 g/m^2 (p <0.01), RWTLV by 0.08 mm (p <0.01). And also in the group of hypertensive athletes there was a significantly lower EDD by 0.2mm (p <0.05).**

***Conclusion*—Thus, the patterns obtained in relation to associations of blood pressure and disorders of heart geometry of athletes of power sports, heavy weight categories, it may be implemented in prevention programs in future with priority focus on the "risk-bearing" groups of men.**

***Keywords*—Blood pressure, arterial hypertension, power sports, sports heart, sudden cardiac death, myocardium hypertrophy, heart remodeling.**

I. INTRODUCTION

In the majority of the population, exercise and sports are associated with health and increased life expectancy. Therefore cases of sudden cardiac death in highly qualified athletes always attract a lot of attention.

Cardiovascular diseases are the leading cause of death in sport, and the sudden cardiac death meets at men more often (from 0.46 to 0.75 cases on 100. 000 athletes a year), than at women [1]. Pioneers of monitoring of cardiovascular system of athletes at power sports were John Longhurst and co-authors [2], who was among the first to point out the increased blood pressure and vulnerability of the cardiovascular system of athletes of power sports, and a little later, other researchers began to note that high blood pressure levels are the most common abnormal diagnosis during pre-screening of the cardiovascular system in such athletes [3, 4]. Perhaps because of the large static component in the training program [5] or the high blood pressure [6] during exercise, the heart is subjected to an additional hemodynamic loading. It increases the formation of transverse bridges, according to the Frank-Starling law, and activates neurohormonal mechanisms to enhance concavity, leading to its compensatory hypertrophy. According to Laplace's law, the stress on the wall of the left ventricle (LV) is proportional to the production of the pressure and LV-radius and inversely proportional to the thickness of its wall. In order to maintain normal values of intra-myocardial stress under conditions of blood pressure increase and LV size increase, it is necessary to increase myocardial thickness. Therefore, the increased pressure as a result of the power operation can be compensated by the increase in the wall thickness of the LV. Because concentric hypertrophy develops under the influence of pressure overload, peak systolic stress in the wall has been proposed as a stimulus for parallel sarcomer replication, causing concentric hypertrophy [7].

Unlike isokinetic, isometric exercises, referred to as power training, are characterized by increased peripheral vascular resistance and normal or slightly elevated cardiac discharge. This increase in peripheral vascular resistance causes transient conditions with a potential risk of hypertension and an increase in post-loading [8]. The increase in tension of a left ventricle's wall, for example, caused by the hypertension induced by increase in an afterload will stimulate a hypertrophy of myocytes, formation of collagen and fibroblasts, and, thus, to lead to remodeling of a myocardium with disproportionate increase in fibrous tissue. These changes subsequently reduce left ventricle compliance, leading to diastolic dysfunction. The increase in left ventricle wall stress is a major mechanical factor in the development of left ventricle hypertrophy, and arterial pressure is the most powerful determinant of left ventricle mass. However, some additional hemodynamics factors play an important role in the development and maintenance of left ventricle hypertrophy, thus bulk overload also makes an important contribution to the development of cardiac hypertrophy. In addition, an increase in total vascular resistance may be due to an increase in the rigidity of the arterial system. The increase in systolic blood pressure contributes to the development of myocardial hypertrophy, whereas diastolic blood pressure is more closely related to the increase in left ventricle wall thickness [9].

Though the Working Group "Function of a Myocardium" of the European Society of Cardiologists recommends to use the term "hypertrophy" only in the context of the size of myocytes of heart, and the term "remodeling" for definition of a regrouping of various elements of heart fabric - process by means of which heart changes the size, geometry and function [10] eventually, however terms remodeling and a hypertrophy are often used in clinical practice is interchangeable.

Knowledge and understanding of how power load can affect the geometry of the left ventricle and blood pressure of athletes of power sports is important, considering that the relative risk of sudden cardiological death in athletes is higher than in non-athletes [11]. Based on the analysis of the problem situation, data of literary sources and requests of sports biologists and doctors, the purpose of the study was set.

II. MATERIAL AND METHODS

The research took place on the basis of the Sports Medicine's Department of the Russian State University of Physical Education, Sports, Youth and Tourism (SCOLIPE) from January 2017 to May 2019. The research was attended by 645 representatives of power sports (weightlifting, power lifting, bodybuilding), having sports qualifications CMS, MS, WCMS. The average age of male athletes was 31.0 ± 5.2 years. The average body weight of athletes was 102.7 ± 6.4 kg. All participants of the research gave voluntary informed consent to participate in the experiment according to the Helsinki Declaration [12]. The following methods were used to achieve the objectives. All examined athletes were subjected to standard rest electrocardiogram, double blood pressure measurement, transthoracic echocardiography on Aloka 3500 (Japan), cardiac sector sensor with frequency 3.5 MHz using B- and M-modes, pulse wave, color and tissue doppler. The weight of the left ventricle myocardium was calculated by the modified formula ASE: WMLV= 0.8 x [1.04 x (FDD+TVSD+TPWLV) 3-FDD3)] + 0.6, where the FDD is the final diastolic diameter of the LV; TVSD - thickness of the ventricle septum in diastole; TPWLV is the thickness of the posterior wall of the left ventricle in diastole. The left ventricle myocardium weight index (LVMWI) was calculated to the body surface area determined by the formula Dubois and Dubois. Left ventricle myocardium hypertrophy (LVMH) included men with LVMWI of 116 g/m2 and above. And the LVMH type was determined by the formula according to the recommendations of Lang and co-authors [13] LVRWT = 2 TPWLV/FDD, where LVRWT is the relative wall thickness of LV in diastole. After passing the examination, athletes were divided into the following cohorts: group-1 (athletes with optimal and normal blood pressure (n = 407)); Group-2 (athletes with elevated blood pressure level (n = 238)). In athletes with increased levels of blood pressure, including increased normal blood pressure, an additional survey was conducted with assessment of specific risk factors of arterial hypertension (AH) in athletes. Mathematical statistics methods were also used in the processing of the obtained data.

III. RESULTS AND DISCUSSION

The examination of 645 power sports athletes of heavy weight categories showed that 238 (37%) athletes have increased blood pressure (systolic BP - 157.4 ± 5.6, diastolic BP - 91.2 ± 5.3) and violation of heart geometry (tables 1 and 2). Although there are serious discrepancies regarding the prevalence of arterial hypertension in different countries and age groups, there is an increase in its recurrence worldwide, as evidenced by the prospective research "MONICA/KORA (2018)" which revealed an increase in the prevalence of hypertension from 34% to 63% [14]. However, the prevalence of AH among young people in Italy (aged 18 to 35) is 11% [15], and in economically developed countries the burden of hypertension in young men was reported as 14% and 21% at the age from 20 to 29 and from 30 to 39 [16]. As for athletes, the research of Caselli and co-authors (2.040 athletes aged 25 ± 6 years, 64% of men, Olympic sports) registered only 3% of the prevalence of AH [17]. Apparently, there are sports in which the recurrence of the AH spread is quite high, for example, in the research by Karpinos and co-authors [18] it was shown that the prevalence of AH was higher among athletes involved in American football (19.2%) than among athletes of other sports (7%).

This data is supplemented by Weiner and co-authors, claiming that 47% of athletes engaged in American football have pre-hypertension and 14% - first stage hypertension. At the same time, among football players there was a significant increase in the prevalence of concentric hypertrophy of LV (31%) and the change in weight of LV correlated with the seasonal change in systolic BP [19]. Concerning athletes of power sports (excluding weight category), the frequency of recurrence of increased BP is found in 21.2% [20], and the total prevalence of hypertension among athletes of power sports of heavy weight category (115-120kg) of China (weightlifting, judo, wrestling, and also athletics throwing (Spear throwing, disk throwing and core pushing)) was 55.4% (49.5% of athletes had mild and moderate hypertension, and 5.9% had severe AH) [21].

In our research, in a comparative analysis in hypertensive athletes, the thickness of the LV myocardium was significantly higher than normal values and averaged 1.2 ± 0.1 mm, which may indicate the presence of minor hypertrophy. The difference between the groups was 0.1 mm and was statistically significant ($p < 0.01$). Athletes also have proportionally larger left atrium sizes and right ventricle diameter, and it reflects a balanced process of heart remodeling [22]. Reliably in hypertensive athletes, compared to normotensive athletes, the following heart parameters were increased: TPWLV on 0,2mm ($p<0,01$), RWD on 4,2mm ($p<0,01$), WMLV on 32,2g ($p<0,01$), LVMWI on 17,8 g/m^2 ($p<0,01$), LVRWT on 0,08mm ($p<0,01$).

Also in the group of hypertensive athletes was significantly less FDD by 0.2 mm ($p < 0.05$). Depending on the value of LVRWT 4 types of LV geometry are identified, the most unfavorable variants of LV heart remodeling are concentric remodeling and concentric hypertrophy, since the formation of these variants of LV heart remodeling involves the development of the most severe disorders of heart diastolic function, increased diastolic and systolic vascular resistance, left atrium overload, hypertrophy of the right ventricle's wall [23].

TABLE I. COMPARATIVE ANALYSIS OF THE HEART GEOMETRY OF HYPERTENSIVE AND NON-HYPERTENSIVE POWER SPORTS ATHLETES (N=645)

Group (N=645)	FDD (mm)	Δ	TIVS (mm)	Δ	TPWLV (mm)	Δ
group-1 (n=404)	5,6±0,4	0,2*	1,1±0,1	0,1**	1,0±0,1	0,2**
group-2 (n=238)	5,4±0,3		1,2±0,1		1,2±0,1	

Notification: FDD – final diastolic diameter; TIVS - thickness of the interventricular septum; TPWLV - thickness of the posterior wall of the left ventricle.
*Asterisk (*) on the right are indicated statistically significant differences between the groups – p<0,05; * – p<0,01; ***

TABLE II. COMPARATIVE ANALYSIS OF THE HEART GEOMETRY OF HYPERTENSIVE AND NON-HYPERTENSIVE POWER SPORTS ATHLETES (N=645)

Group (N=645)	DRV (mm)	Δ	WMLV (g)	Δ	WMLVI (g/m²)	Δ	LVRWT (mm)	Δ
group-1 (n=404)	22,8±3,5	4,2**	239,3±40,4	32,2**	107,4±17,5	17,8**	0,38±0,03	0,08**
group-2 (n=238)	27,0±4,3		271,5±32,3		125,2±9,8		0,46±0,02	

Notification: DRV - diameter of the right ventricle; WMLV - weight of myocardium of the left ventricle; WMLVI - left ventricle myocardium weight index; LVRWT - relative wall thickness of the left ventricle. Asterisk (*) on the right are indicated statistically significant differences between the groups. – p<0,05; – p<0,01;***

According to the manual published by Leng and the co-authors [24], HLV is defined as a significant increase in the weight of the myocardium of LV, expressed as the weight of LV indexed to the body surface area (> 115 g/m² for men). In addition, by calculating the relative wall thickness, it is possible to determine the geometric difference of the LV weight and classify the HLV. It is well known that if the LVRWT is more than 0.42, the hypertrophy is concentric [25]. In the analysis of 13 researches of Haykowsky and co-authors of power sports athletes (excluding weight category) the most common patterns of LV were normal geometry (37.5%) and concentric hypertrophy (37.5%), and only 25% of athletes observed eccentric hypertrophy. And most of the aggravated athletes showing this pattern were weightlifters, and a smaller number (< 20%) were powerlifters [26].

IV. CONCLUSION

Examination of power sports athletes (bodybuilding, powerlifting and weightlifting) of heavy weight categories revealed quite high percentage (37%) of athletes having increased BP. Considering that in general in the Olympic population AH is quite rare (3%), it is worth paying close attention to this cohort of athletes. Also in hypertensive athletes there was revealed violation of geometry of LV, and the most common pattern of geometry matched to concentric hypertrophy of LV diagnosis. Further researches are required that will help to find out what leads to impaired LV geometry, arterial hypertension, isometric loading or increased body weight.

Conflict of interest: not declared.

REFERENCES

[1] Lauschke J, Maisch B. Athlete's heart or hypertrophic cardiomyopathy? Clin Res Cardiol. 2009; 98:80-8.

[2] Longhurst J, Charles S. The power athlete. Cardiology clinics. 1997; 15:413–29. doi:10.1016/s0733-8651(05)70349-0.

[3] Berge H, Isern C, Berge E. Blood pressure and hypertension in athletes: a systematic review. British Journal of Sports Medicine. 2015; 49:716–23. doi:10.1136/bjsports-2014-093976.

[4] Schleich K., Smoot M., Ernst M. Hypertension in Athletes and Active Populations. Curr Hypertens Rep. 2016; 18:1–8. doi:10.1007/s11906-016-0685-y.

[5] De Freitas Brito A., Brasileiro-Santos M., Coutinho de Oliveira C. et al. Post exercise Hypotension Is Volume-Dependent in Hypertensives: Autonomic and Forearm Blood Responses. J Strength Cond Res. 2019; 33:234–41. doi:10.1519/jsc.0000000000001735.

[6] Gjovaag T, Hjelmeland A, Oygard J. et al. Resistance exercise and acute blood pressure responses. J Sports Med Phys Fitness. 2015:1–21.

[7] Krysztofiak H, Petkow Dimitrow P. Differentiating physiology from pathology in elite athletes. Left ventricular hypertrophy versus hypertrophic cardiomyopathy. Kardiol Pol. 2016; 74:705-16. doi:10.5603/KP.a2016.0084.

[8] Leddy J, Izzo J. Hypertension in athletes. J Clin Hypertens. 2009; 11:226–33. doi:10.1111/j.1751-7176.2009.00100.x.

[9] Kahan T, Bergfeldt L. Left ventricular hypertrophy in hypertension: its arrhythmogenic potential. Heart. 2005; 91:250-6. doi:10.1136/hrt.2004.042473.

[10] Knöll R, Iaccarino G, Tarone G. et al. European Society of Cardiology. Towards a re-definition of 'cardiac hypertrophy' through a rational characterization of left ventricular phenotypes: a position paper of the Working Group 'Myocardial Function' of the ESC. Eur J Heart Fail. 2011; 13:811-9. doi:10.1093/eurjhf/hfr071.

[11] Corrado D, Basso C, Rizzoli G. et al. Does sports activity enhance the risk of sudden death in adolescents and young adults? J Am Coll Cardiol. 2003; 42:1959–63. doi:10.1016/j.jacc.2003.03.002.

[12] Harriss D, Atkinson G. Ethical Standards in Sport and Exercise Science Research: 2016 Update. Int J Sports Med. 2015; 36:1121–4. doi:10.1055/s-0035-1565186.

[13] Lang R, Bierig M, Devereux R. et al. Recom mendations for chamber quantification. Eur. J. Echocardiogr. 2006; 7:79–108. doi:10.1016/j.euje.2005.12.014.

[14] Atasoy S, Johar H, Peters A. et al. Association of hypertension cut-off values with 10-year cardiovascular mortality and clinical consequences: a real-world perspective from the prospective MONICA/KORA study. Eur Heart J. 2019;1; 40:732-38. doi:10.1093/eurheartj/ehy694.

[15] Bruno R, Pucci G, Rosticci M. et al. Association between lifestyle and systemic arterial hypertension in young adults: a national, survey-based, cross-sectional study. High Blood Press Cardiovasc Prev. 2016; 23:31-40. doi:10.1007/s40292-016-0135-6.

[16] Kearney P, Whelton M, Reynolds K. et al. Global burden of hypertension: analysis of worldwide data. Lancet. 2005; 365:217-23. doi:10.1016/s0140-6736(05)17741-1.

[17] Caselli S, Vaquer Sequì A, Lemme E. et al. Prevalence and Management of Systemic Hypertension in Athletes. Am J Cardiol. 2017; 119:1616-22. doi:10.1016/j.amjcard.2017.02.011.

[18] Karpinos A, Roumie C, Nian H. et al. High prevalence of hypertension among collegiate football athletes. Circ Cardiovasc Qual Outcomes. 2013; 6:716-23. doi:10.1161/circoutcomes.113.000463.

[19] Weiner R, Wang F, Isaacs S. et al. Blood pressure and left ventricular hypertrophy during American-style football participation. Circulation. 2013; 128:524-31. doi:10.1161/circulationaha.113.003522.

[20] Volnov N.I. Blood pressure at athletes: Abstract of thesis … for PhD. – 1958.

[21] Guo J, Zhang X, Wang L. et al. Prevalence of metabolic syndrome and its components among Chinese professional athletes of strength sports with different body weight categories. PLoS One. 2013;8: e79758. doi:10.1371/journal.pone.0079758.

[22] D'Ascenzi F, Pelliccia A, Corrado D et al. Right ventricular remodelling induced by exercise training in competitive athletes. Eur Hear J Cardiovasc Imaging. 2015; 17:301–7. doi:10.1093/ehjci/jev155.

[23] Devereux R, Wachtell K., Gerdts E. et al. Prognostic significance of left ventricular mass change during treatment of hypertension. JAMA. 2004; 292:2350–56. doi:10.1001/jama.292.19.2350.

[24] Lang R, Badano L, Mor-Avi V. et al. Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging. 2015; 16:233–71. doi:10.1093/ehjci/jev014.

[25] Ganau A, Devereux R, Roman M. et al. Patterns of left ventricular hypertrophy and geometric remodeling in essential hypertension. J Am Coll Cardiol. 1992; 19:1550–8. doi:10.1016/0735-1097(92)90617-v.

[26] Haykowsky M, Dressendorfer R, Taylor D. et al. Resistance training and cardiac hypertrophy: unravelling the training effect. Sports Med. 2002; 32:837-49. doi:10.2165/00007256-200232130-00003.