International Heart and Vascular Disease Journal • Volume 7, № 23, September 2019

Journal of the Cardioprogress Foundation

LEADING ARTICLE

Arterial pressure and left ventricular geometry in heavyweight athletes

A. B. Miroshnikov, A. B. Smolensky, A. D. Formenov, S. U. Zolicheva

Russian State University of Physical Education, Sport and Tourism, Moscow, Russia.

Authors

Alexander B. Miroshnikov*, Ph.D., assistant professor of the Department of Sports Medicine of Russian State University of Physical Education, Sport and Tourism, Moscow, Russia.

Andrey V. Smolensky, M.D., Ph.D., doctor of sciences, professor, head of the Department of Sports Medicine of Russian State University of Physical Education, Sport and Tourism, Moscow, Russia.

Alexander D. Formenov, master of the Department of Philosophy and Social Sciences of Sports Medicine of Russian State University of Physical Education, Sport and Tourism, Moscow, Russia.

Sventlana U. Zolicheva, M.D., Ph.D., assistant professor of the Department of Sports Medicine of Russian State University of Physical Education, Sport and Tourism, Moscow, Russia.

Objective. *To study arterial pressure and left ventricular geometry in heavyweight athletes.*

Materials and methods. *We examined 645 heavyweight athletes (weightlifters, powerlifters, bodybuilders) with candidate to master of sports, master of sports and international master of sports qualifications with average body weight of 102.7±6.4 kg. All patients underwent general examination, standard electrocardiography, double BP measurement and transthoracic echocardiography.*

Results. *The results of investigation showed that 248 (37%) athletes had increased BP (systolic BP — 157,4±5,6, diastolic BP– 91,2±5,3) and left ventricular (LV) geometry impairment. The following heart parameters increased in athletes with hypertension compared with normotensive athletes: diastolic interventricular thickness by 0,1 mm (p<0,01), left ventricular posterior wall thickness by 0,2 mm (p<0,01), right ventricular diameter by 4.2 mm (p<0.01), LV myocardial mass by 32, 2g (p<0,01), LV myocardial mass index by 17,8 g/m^2, LV relative wall thickness by 0,08 mm (p<0,01). LV end-diastolic volume was 0,2 mm (p<0,05) lower in hypertensive athletes.*

Conclusion. *Thus, the results of the study on the association between BP and heart geometry disturbances in heavyweight athletes may be used as the scientific basis for organizing preventive programs with the main focus on risk groups.*

Key words: *arterial pressure, arterial hypertension, sport, athlete heart, sudden cardiac death, myocardial hypertrophy, heart remodeling.*

Conflict of interests: none declared.

* Corresponding author. Tel.: 8(985)-125-12-23. E-mail: benedikt116@mail.ru

Received: 16.07.2019 **Accepted:** 07.08.2019

Introduction

Most people associate physical exercises and sports with health and increased life expectancy. Therefore, cases of sudden cardiac death (SCD) in highly-qualified athletes always attract great attention. Cardiovascular diseases (CVD) are the leading cause of death among athletes. It is also remarkable that SCD is more frequent in men (from 0.46 to 0.75 cases for 100000 athletes per year) compared with women [1]. John Longhurst et al. [2] were the first researches of cardiovascular system (CVS) in heavyweight athletes, who observed increased arterial pressure (AP) and CVS vulnerability among them, that were later supported by other researches [3, 4]. This can be associated with additional heart hemodynamic stress during exercises due to larger static component [5] or higher AP [6]. It increases the development of cross-bridges according to Frank-Starling law and activates the neurohormonal mechanisms to enhance heart contractility that leads to its compensatory hypertrophy. According to Laplace's law, the voltage on the wall of the left ventricle (LV) is proportional to the multiplication of the pressure and LV radius and inversely proportional to the thickness of LV wall. In order to maintain normal values of intramyocardial tension during AP and LV size increase, it is necessary to rise myocardial wall thickness. Therefore, an pressure due to increased power load can be compensated by rise of LV wall thickness. Since concentric hypertrophy develops during pressure overload, peak systolic wall tension is thought to be its stimulus, because it causes parallel replication of sarcomeres [7].

Unlike isokinetic, isometric exercises, that we call strength training, are characterized by increased peripheral vascular resistance and normal, or slightly increased cardiac output. This increase of peripheral vascular resistance causes transient conditions with potential risk of hypertension and increased afterload [8]. LV wall tension increase, for example, caused by hypertension induced by increased afterload, stimulate myocyte hypertrophy, the formation of collagen and fibroblasts, and, thus, leads to myocardium remodeling with disproportionate increase of fibrous tissue. These changes subsequently decrease LV compliance, that leads to diastolic dysfunction. Increased LV wall tension is the main mechanical factor of the LV hypertrophy (LVH) development, and BP — of LV mass. But some additional hemodynamic factors also play an important role in the development and maintenance of LVH, for example, volume overload also contribute to the development of heart hypertrophy. Total vascular resistance can also contribute to the increase of arterial stiffness. Systolic blood pressure (SBP) contributes to the development of myocardial hypertrophy, while diastolic blood pressure (DBP) is associated with LV wall thickness [9].

Although the Working Group «Myocardial Function» of the European Society of Cardiology recommends to use the term «hypertrophy» as the size of heart myocytes, and the term «remodeling» to define rearrangement of cardiac tissue — the process when heart changes its size, geometry and function over time [10], however, these terms are often used interchangeably in clinical practice. Knowledge and understanding of how power load can affect LV geometry and BP in athletes are important, because relative risk of SCD is higher among athletes [11]. The study objective was set based on the analysis of problem relevance, literature sources and the data of requests of sports biologists and doctors.

Materials and methods

The study was conducted at the Department of Sports Medicine of Russian State University of Physical Education, Sport and Tourism from January 2017 to March 2019. We examined 645 heavyweight athletes (weightlifters, powerlifters, bodybuilders) with candidate to master of sports, master of sports and international master of sports qualifications. Average age was 102,7±6,4 kg. Average body weight — 102.7±6.4 kg. All study participants signed informed consent to participate in the study according to the Helsinki Declaration [12]. All athletes underwent general examination, standard resting electrocardiogram, two separate blood pressure measurements, transthoracic echocardiography using Aloka 3500 devices (Japan) with cardiological sector probe with 3.5 Mhz frequency using B- and M-modes, pulse-wave, color and tissue doppler. Left Ventricular mass (LVM) was calculated using modified ASE formula:

$$LVM = 0,8\times[1,04\times((LV\ EDV + IVSTd + LVPWd)\ 3 - LV\ EDV)] + 0,6,$$

where LV EDV — left ventricular end diastolic volume; IVSTd — intraventricular septum thickness during diastole; LVPWTd — left ventricular posterior wall thickness during diastole.

Left ventricular mass index (LVMI) was calculated to body surface area found using Du Bois method. Men with LVMI over 116 g/m^2 had left ventricular hypertrophy (LVH). The type of LVH was estimated according to guidelines of Lung et al. [13] — RLVWTd =2LVPWd/LV EDV, where RLVWTd — relative left ventricular wall thickness during diastole. After the examination athletes were divided into the following cohorts: group-1 (athletes with optimal and normal BP (n=407)); group-2 (athletes with increased BP (n=238)). Athletes with increased or normal increased BP, were additionally interviewed to assess specific arterial hypertension (AH) risk factors. We also used mathematical statistic methods to process obtained data.

Results and discussion

The investigation of 645 heavyweight athletes showed that 238 (37%) had increased BP (SBP — 157,4±5,6, DBP — 91,2±5,3) and heart geometry impairment (table 1, 2). According to the prospective «MONICA / KORA (2018)» study, the prevalence of AH increased from 34% to 63% [14], although there are serious differences on AH prevalence in different countries and age groups. However, the prevalence of AH among young people in Italy (aged from 18 to 35 years) is 11% [15], and in developed countries AH was recorded in 14% and 21% of patients aged from 20 to 29 and from 30 to 39 years, respectively [16]. According to Kazelli et al. study (2,040 athletes aged 25±6 years, 64% — men, Olympic sports), the prevalence of AH among sportsmen was only 3% [17]. Apparently, there are sports with higher rate of AH, for example, Karpinos et al. [18] showed, that the prevalence of AH was higher among American football (19.2%) players compared with other sports (7%). Weiner et al. confirmed these data and showed, that 47% of American football athletes had prehypertension and 14% — stage 1 hypertension. It is also remarkable that football players had significant increase of LV concentric hypertrophy prevalence (31%), and LV mass changes correlated with seasonal SBP changes [19]. The prevalence of increased blood pressure was 21.2% [20] in powersport athletes (excluding weight category), and general prevalence of hypertension among heavyweight athletes (115–120 kg) in China (heavy athletics, judo, wrestling), as well as athletics throwing (javelin, disk throwing and shot put) was 55.4% (49.5% had mild or moderate hypertension, and 5.9% — severe hypertension) [21].

Our comparative analysis of hypertensive athletes showed that LV myocardium wall thickness significantly exceeded normal values (1.2±0.1 mm on average) that may indicate slight hypertrophy. The difference between groups was statistically significant — 0.1 mm ($p<0.01$). Athletes also had proportionally larger size of left atrium and right ventricle diameter (RVD), that reflected balanced process of heart remodeling [22].

The following heart parameters significantly increased in hypertensive athletes compared with normotensive: LVPWT by 0.2 mm ($p<0.01$), RVD by 4.2 mm ($p<0.01$), LVM by 32,2 g ($p<0.01$), LVMI on 17.8 g/m^2 ($p<0.01$), RLVWT by 0.08 mm ($p<0.01$). Hypertensive athletes also had significantly lower EDV — by 0,2 mm ($p<0,05$). We identified 4 types of LV geometry according to RLVWT. The most unfavorable types of heart remodeling are concentric remodeling and concentric hypertrophy, since they are associated with the development of the most severe heart diastolic function disorders, increased diastolic and systolic vascular resistance, left atrium overload, and right ventricle wall hypertrophy [23].

Conflict of interest: None declared.

Table 1. **Comparative analysis of heart geometry in hypertensive and normotensive athletes (N=645)**

Group (N=645)	EDV (mm)	Δ	IVST (mm)	Δ	LVPWT (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	

* $p<0,05$; ** $p<0,01$ the differences between groups are statistically significant.

Table 2. **Comparative analysis of heart geometry in hypertensive and normotensive athletes (N=645)**

Group (N=645)	EDV (mm)	Δ	LVM (g)	Δ	LVMI (g/m^2)	Δ	RLVWT (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	

* $p<0,05$; ** $p<0,01$ the differences between groups are statistically significant.

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.
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.
4. Schleich K., Smoot M., Ernst M. Hypertension in Athletes and Active Populations. Curr Hypertens Rep. 2016; 18:1–8.
5. De Freitas Brito A., Brasileiro-Santos M., Coutinho de Oliveira C. et al. Postexercise Hypotension Is Volume-Dependent in Hypertensives: Autonomic and Forearm Blood Responses. J Strength Cond Res. 2019; 33:234–41.
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.
8. Leddy J., Izzo J. Hypertension in athletes. J Clin Hypertens. 2009; 11:226–33.
9. Kahan T., Bergfeldt L. Left ventricular hypertrophy in hypertension: its arrhythmogenic potential. Heart. 2005; 91:250–6.
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.
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.
12. Harriss D., Atkinson G. Ethical Standards in Sport and Exercise Science Research: 2016 Update. Int J Sports Med. 2015; 36:1121–4.
13. Lang R., Bierig M., Devereux R. et al. Recom mendations for chamber quantification. Eur. J. Echocardiogr. 2006; 7:79–108.
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;40:732–38.
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.
16. Kearney P., Whelton M., Reynolds K. et al. Global burden of hypertension: analysis of worldwide data. Lancet. 2005; 365:217–23.
17. Caselli S., Vaquer Sequì Aю, Lemme E. et al. Prevalence and Management of Systemic Hypertension in Athletes. Am J Cardiol. 2017; 119:1616–22.
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.
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.
20. Volnov N.I. Blood pressure in athletes: Abstract. Diss. cand. med. sciences, 1958. — 12 p. Russian.
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.
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.
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.
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.
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.
26. Haykowsky M., Dressendorfer R., Taylor D. et al. Resistance training and cardiac hypertrophy: unravelling the training effect. Sports Med. 2002; 32:837–49.