International Heart and Vascular Disease Journal
Volume 5, Number 14, March 2017

Journal of the Cardioprogress Foundation

Arterial hypertension and heart remodeling in athletes

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Summary

Objective

To investigate left ventricle's hemodynamic characteristics and morphofunctional condition in different types of athletes.

Materials and methods

This study included 231 athletes aged 18-32 years working in 8 different sport types: boxing, wrestling, weight lifting, track and field athletics (middle-distance running), cycle racing, bicycle motocross, yachting, and pentathlon. Analysis of the character of physical exercise was performed using J.H. Mitchell classification (2005) that classified sports according with the combination of dynamic and static loads. All athletes underwent examination, standard resting electrocardiogram, repeated blood pressure (BP) measurement, and transthoracic echocardiography. Athletes with high normal and elevated BP underwent 24-hours BP monitoring. Athletes with elevated BP underwent additional questioning of specific arterial hypertension (AH) risk factors.

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Results

Elevated BP was detected in 5.6% of examined athletes. Elevated BP was presented mostly in athletes specializing on static loads of high intensity combined with aerobic loads of moderate or high intensity. According with the results of echocardiography study, 19 men had mild increase of left ventricular myocardial mass, and 10 men had left ventricular hypertrophy. Specific AH risk factors in athletes include increased consumption of sodium, alcohol, caffeine and prohibited medications like erythropoietin, growth hormone and oral contraceptives in females.

Conclusion

Patterns of athletic heart development may be explained by the orientation of training and competition activity; normally they do not lead to abnormal systolic and diastolic function, but BP elevation in athletes increases the risk of myocardial hypertrophy and remodeling that may be a possible contraindication for sports requiring high intensity static loads.

Key words:

Arterial hypertension, myocardial remodeling, isokinetic and isometric exercise, athletic heart.

Introduction

Treatment and prevention of arterial hypertension (AH) is one of the most important healthcare objectives in developed countries. Increased AH prevalence goes along with proportional growth of morbidity and mortality due to myocardial infarction and brain stroke that are currently considered to be associated with AH. Distinct correlation between blood pressure (BP) levels and cardiovascular mortality risk indicate that BP reduction is the most effective method of cardiovascular mortality prevention not only in patients with elevated BP, but also in patients with normal BP. According with epidemiological studies, in the Russian Federation AH occurrence in persons above 15 years reaches 39.5%, that is equivalent to approximately 40 mln people suffering from AH. AH is detected more often in females comparing to males (40.4% and 37.2%, respectively) [1].

AH often starts in early age. Swift increase of number of children and adolescents with AH causes concerns. AH occurrence in school students is around 12-18% [2.3]. Epidemiological studies demonstrated that physical activity and cardiorespiratory training have inverse relation with BP levels and AH occurrence. These trends have been proved in randomized trial that demonstrated that physical activity can reduce BP in both people with normal and increased BP. In general, aerobic exercise lowers systolic BP (SBP) approximately by 2 mm Hg (up to 7 mm Hg) [4, 5]. At the same time, patients with AH demonstrate the most significant BP reduction. General prevalence of AH in physically active people is 50% lower than in general population [4]. But one study that included 467 adolescents making sports demonstrated that 57 young people (12.2%) had elevated BP, and 43 of them (79.6%) had persistently elevated BP for one year. Another important observation of this study was related to the fact that speed-power loads prevailed in the group of young athletes. Secondary AH occurrence in athletes is the same as in general population. Together with the characteristics of training, prevalence of isometric and speed-power loads possibly promoting BP increase, other risk factors could influence AH in athletes: increased alcohol consumption, illegal use of drugs (cocaine, etc), anabolic steroids, stimulators use (for example, in supplementaries increasing organism's energetic resources and body weight controllers), high stress levels, male gender (it occurs twice as more often in African race patients comparing with the European ones, and Asians have lower morbidity comparing with Europeans), several hereditary factors: family history of AH and cardiac diseases in males above 55 years and females above 65 years, diabetes mellitus or impaired glucose tolerance, smoking and chewing tobacco, obesity. Secondary AH causes in athletes include administration of oral contraceptives (women), growth hormone, erythropoietin, non-steroidal anti-inflammatory drugs [6, 7, 8]. Unlike isokinetic training, isometric exercises, called strength training, are characterized with increased peripheral vascular resistance and normal or slightly increased cardiac output. This increase of peripheral vascular resistance leads to development of transitional conditions with increased post-load and potential risk of hypertension [6, 7, 8]. It is worth to notice that AH induced by physical exercise, apart from myocardial remodeling, may lead to myocardial fibrosis that can further provoke development of malignant arrhythmias [9, 10, 11]. More than that, several researchers think that

high-intense aerobic training often causes elevation of blood levels of myocardial damage markers (troponin I and B-type natriuretic peptide) correlating with reduced right ventricular ejection fraction [12].

Speed-sthrength training is the dominating form of activity in such sports like weight lifting, wrestling, throwing sports, American football, rugby. Many sports like playing sports (football, lacrosse, basketball, hockey, field hockey) include significant loads aiming to develop endurance and strength exercises.

Long intensive physical exercise causes physiological adaptation of heart resulting in its structural and functional changes like physiological hypertrophy of myocardial walls and moderate dilatation of its cavities. These physiological changes depend on such factors like age, gender, constitution, sport's type, and normally they do not exceed reference values. Athletes develop different kinds of physiological heart adaptation depending on particular features of their sport's type.

Sportsmen training in cycling, mostly aerobic sports (long-distance running, skiing, and swimming) develop mostly left ventricle (LV) cavity's dilatation with proportional increase of its walls' thickness. It is caused by increased cardiac output during exercise, so LV overload by volume, and increased systemic BP. It leads to development of eccentric LV hypertrophy without changing ratio between LV wall thickness and its diameter.

Sports requiring static or isometric loads (weight lifting, wrestling, throwing sports) lead to development of concentric myocardial hypertrophy, increased LV wall thickness without changing its cavity size because of increased systemic BP and increased heart post-load during exercising.

Results of echocardiography (EchoCG) studies demonstrate that LV posterior wall thickness (LVPWT) in athletes is increased by 15-20% comparing with untrained people. LV end diastolic size (LVEDS) of the majority of athletes is 10% bigger comparing with untrained people, still remaining in the range of reference values.

At the same time, A. Pelliccia and coauthors identified LVEDS increase up to 60 mm in sportsmen (reference value – up to 70 mm) and enlarged interventricular septum thickness (IVST) (>12mm) that were not associated with the signs of dilatation cardiomyopathy (DC) [13, 14]. The study of Basavarajaiah and coauthors found out increased LVPWT up to 13mm (maximal value 16 mm) in 1.5% of observed athletes [15].

Another sign of the "athletic heart" is increased volume and mass of the right ventricle. Systolic and diastolic function of both ventricles remains normal during rest and physical exercise. Usually dimensions of ventricular cavities and walls come back to normal values with termination of intensive training. Reversibility of these changes is considered to be one of key signs of the "athletic heart".

At the same time, there is a small group of athletes with evident myocardial hypertrophy and/or dilatation of cardiac cavities who have phenotype similar with hypertrophic cardiomyopathy (HC) and DC. There is a crossing area between different cardiomyopathy types and "athletic heart", so-called "grey zone" consisted of sportsmen with evident EchoCG signs of LV hypertrophy (13-16mm) and/or dilatation of LV cavity (LVEDS 55-60 mm) [16, 17.18.19.20.21].

. It is possible that myocardial hypertrophy in athletes could be caused by increased BP.It has been proved by several studies demonstrating that sportsmen with hypertensive reaction to load have significantly higher LV myocardial mass comparing with the athletes with normotensive reaction.

It's possible to consider elevated BP in athletes as one of the forms of overloaded cardiovascular system's reaction that may lead to "athletic heart" remodeling. One of the key differences between "athletic heart" and myocardial hypertrophy in patients with cardiovascular disease is the absence of LV diastolic dysfunction in sportsmen with increased heart mass.

Materials and methods

This study included 231 athletes aged 18-32 years (average age 22 years) working in 8 different sport types: boxing, wrestling, weight lifting, track and field athletics (middle-distance running), cycle racing, bicycle motocross, yachting, and pentathlon and having qualification from I adult class to the master of sports.

Analysis of the character of physical exercise was performed using J.H. Mitchell classification (2005) [22] that classified sports according with the combination of dynamic and static loads (Table 1). The classification is based on peak static and dynamic load. Degree of dynamic component increase is determined by maximal oxygen uptake (max O_2) and degree of cardiac output increase; degree of static component increase is identified using the percentage of maximal voluntary contraction (MVC) increase.

Table 1. **Classification of sports depending on combination of static and dynamic loads**

Characteristic of physical exercise	A. Low intensity dynamic exercise (<40% max O_2)	B. Moderate intensity dynamic exercise (40-70% max O_2)	C. High intensity dynamic exercise (>70% max O_2)
I. Low demand static exercise(<20% MVC)	Billiards, bowling, cricket, curling, golf, riflery	Baseball/softball, table tennis, volleyball	Badminton, cross-country skiing (classic technique), race walking, running (long distance), squash, orienteering, tennis
II. Moderate demand static exercise (20-50% MVC)	Archery, auto racing[1, 2], diving[2], motorcycling[1, 2], gymnastics[1, 2], karate[1, 2], judo[1, 2], equestrian[1, 2], yachting	American football, field events (jumping), figure skating (pair skating)[1], rugby[1], cross, running (sprint), synchronized swimming^	Basketball[1], ice hockey[1], biathlon, cross-country skiing (skating technique), lacross[1], running (middle and long distance), figure skating (single), swimming[2], handball, football[1]
III. High demand static exercise (>50% MVC)	Bobsledding/luge[1, 2], martial arts[1], field events (throwing), gymnastics[1, 2], sport climbing, water skiing[1, 2], weight lifting[1, 2], windsurfing[1, 2]	Body building[1, 2], downhill skiing[1, 2], skateboarding[1, 2], snowboarding[1, 2], wrestling[1, 2]	Boxing[1], canoe/kayaking, cycling[1, 2], decathlon, rowing, speed-skating[1, 2], triathlon[1, 2], mountain skiing, water polo

Comment: [1] - risk of injury, [2] - high risk of syncope

All observed athletes underwent standard ECG at rest, repeated BP measurement, transthoracic echocardiography using Aloka 3500 (Japan), Vivid 7 GE (USA), Philips IE 33 HP (Netherlands) apparatus and cardiac sector transducer with 3.5 mHz frequency in B- and M- modes, impulse-wave, color and tissue Doppler-echocardiography (TD-EchoCG). 24-hours BP monitoring (Astrocard® Holtersystem) was performed in all athletes with high normal and elevated BP.

Sportsmen with elevated BP including high normal BP underwent addtional questioning in order to evaluate specific AH risk factors.

Left ventricular myocardium mass (LVMM) was quantified using modified ASE formula: LVMM=0.8*[1.04*((EDS+IVST+LVPWT)^3-EDS^3)]+0.6. LV myocardial mass index (LVMMI) was estimated using body surface area (BSA) quantified with Dubois formula. Males with LVMMI >116g/m^2 and females with LVMMI> 109g/m^2 were considered to have LV myocardial hypertrophy (LVH).

LV diastolic function (DF) was evaluated using characteristics of trans-mitral flow (TMF) measured in impulse-wave mode of Doppler-EchoCG (peak velocity of early diastolic filling of LV – E, cm/sec, peak velocity of late diastolic filling – A, cm/sec, E-wave deceleration time (Dt, msec) were measured, then the E/A ratio was quantified), and mitral fibrous ring displacement (lateral part, interventricular septum, LV anterior and posterior walls) in impulse-wave TD-EchoCG mode. During TD-EchoCG we measured the following parameters of LV systolic and diastolic function: maximal velocity of main "peaks" of myocardial movements (Sa – systolic movement of myocardium, two diastolic movements: e and a, cm/sec), then we quantified the ratio of main peak velocities of early TMF, diastolic myocardial movement (E/e), and ratio of diastolic myocardial movements peaks (e/a).

Systolic myocardial stress (SMS, din/cm^2) was quantified using the following formula:

SMS=(SBP*LVESS/4*systolicLVPWT)*(1+systolicLVPWT/LVESS), where LVESS –left ventricular end systolic size.

Diastolic myocardial stress (DMS, din/cm^2) was quantified using the following formula:

DMS=(SBP*LVEDS/4*diastolicLVPWT)*(1+diastolicLVPWT/LVEDS), where LVEDS –left ventricular end diastolic size.

Statistical analysis of results was performed using Excel 2007 and STATISTICA 8.0 (StatSoft Inc., USA) software. Before choosing the method of data comparison we performed normality tests. To test the hypothesis of two average values equation in two groups we used Student's t-test or non-parametric Mann-Whitney test, to disprove the null hypothesis we applied Student's t-test. Probability of differences was quantified accurate within 0.0001. P-value <0.05 was considered significant.

Results and discussion

5.6%(13 persons) of 231 athletes involved in this study had elevated BP, increased BP was present mostly in sportsmen practicing static exercises of high demand combined with aerobic loads of moderate or high intensity.

According with the results of EchoCG study, sportsmen were divided into following groups: females without LVH (n=81), males without LVH (n=103), males with insignificant increase (border-line) of LVMMI (n=19), males with LVH (LVMMI 132-148 g/m^2,

n=10). Enlargement of one of LV walls during diastole (up to 1.2 cm) or LVMMI 116-131 g/m^2 were considered as border-line LVMM.

Results of comparative analysis of myocardial DF in relation to LVMMI are present in the Table 2.

TD-EchoCG results demonstrated no abnormal LV myocardium DF in all groups of athletes; this fact goes along with the majority of known studies [23, 24]. There were no statistically significant differences in DF char-

Table 2. **Morphofunctional condition of LV (according with TD-EchoCG results)**

Characteristic		Women with normal LVMMI, N=81	Men with normal LVMMI, N=103	Men with insignificantly increased (borderline) LVMMI, N=19	Men with LVH, N=10
MMI, g/m^2		76.5±15	92±16	106±12	139±16
Relative wall thickness (RWT)		0.38±0.06	0.40±0.05	0.46±0.04	0.43±0.04
Dt, msec		188±29	194±31	200±17	195±30
E/A		2.0±0.4	1.82±0.4	2.0±0.5	2.2±0.6
Lateral wall	Sa, cm/sec	12.9±2.5	12±2.5	11.3±3.5	10.1±2.8
	e, cm/sec	18±3.4	18±3.7	17±3.2	16±3.5
	E/e	5.1±0.8	4.9±1.2	5.1±1.1	5.5±1.2
	E/a	2.7±0.7	2.8±0.8	2.9±1.1	3.0±1.1
Interventricular septum (IVS)	Sa, cm/sec	9.3±1.9	9.2±1.2	8.9±1.4	8.6±1
	e, cm/sec	14.1±2.3	13.2±2.2	12.6±2.0	12.1±1.7
	E/e	6.7±1.3	6.6±1.3	6.3±1.6	6.9±1.8
	E/a	2.2±0.6	2.0±0.5	2.0±0.6	2.2±0.6
Anterior wall	Sa, cm/sec	11.9±2.7	11±2.4	10±2.4	9.7±2.1
	e, cm/sec	17.8±3.2	17.3±3.8	16.8±3.2	18.7±3.1
	E/e	5.3±0.9	5.2±1.2	4.8±1.3	4.5±0.7
	E/a	2.7±0.8	2.7±1	2.8±1.2	3.2±1
Posterior wall	Sa, cm/sec	9.5±0.9	9.7±1.5	9.3±1.25	9.3±1.7
	e, cm/sec	15±2.3	14±2.5	13.2±2.0	13.9±2.7
	E/e	6.3±1.2	6.1±1.2	6±1.5	6.1±1.6
	E/a	2.3±0.6	2.2±0.6	1.8±0.6	2.2±0.9

Table 3. **Morphofunctional condition of LV (according with TD-EchoCG results)**

Characteristic		Men with normal LVMM, (n=103)	Men with LVH and border-line LVMM (n=29)	p-value
MMI, g/m^2		92±16	117±20	0.0001
SMS		190±36	236±32	0.0001
DMS		177±31	224±29	0.0001
RWT		0.40±0.05	0.45±0.04	0.0001
Dt, msec		194±33	198±22	0.607
E/A		1.8±0.4	2.1±0.5	0.006
Lateral wall	s, cm/sec	11.5±2.5	10.9±3.3	0.346
	e, cm/sec	17.7±3.7	13.3±3.3	0.115
	E/e	4.9±1.2	5.2±1.1	0.387
	e/a	2.7±0.8	2.9±1.1	0.356
IVS	s, cm/sec	9.2±1.2	8.8±1.2	0.204
	e, cm/sec	13.2±2.2	12.4±2.1	0.189
	E/e	6.6±1.3	6.3±2.1	0.470
	e/a	2.1±0.5	2.2±0.9	0.531
Anterior wall	s, cm/sec	11.2±2.4	10.0±2.3	0.064
	e, cm/sec	17.3±3.8	17.5±3.2	0.804
	E/e	5.2±1.2	4.8±1.6	0.351
	e/a	2.7±1.0	3.1±1.1	0.276
Posterior wall	s, cm/sec	9.7±1.5	9.3±1.4	0.263
	e, cm/sec	14±2.5	13.5±2.3	0.154
	E/e	6.1±1.2	6.0±2.0	0.853
	e/a	2.2±0.6	2.1±0.8	0.698
Shortening of anterior-posterior dimensions	%	31.2±4.1	30.6±5.4	0.543
Ejection fraction (EF) (Simpson)	%	60.9±4.7	58.3±5.1	0.013
Cardiac output	L	4.6±1.2	4.8±1.3	0.288

acteristics between the groups. But Sa and diastolic e peak in the lateral area of mitral valve fibrous ring (MVFR), IVS, and to less extent – posterior wall, tended to decrease with the growth of myocardial mass.

In order to perform more obvious comparative analysis, we divided the athletes into two groups: males without LVH (n=103) and males with insignificant increase of LVMM and LVH (n=29). The results of this comparative analysis are demonstrated in Table 3.

Correlation analysis did not reveal correlation between LVMM characteristics and E/A peaks ratio (r=0.022, p=0.741). There were no significant differences in parameters characterizing LV DF. We identified weak but statistically significant correlation of LVMM characteristics and Sa velocity of MVFR lateral part (r=-0.174, p=0.013). Comparative analysis of LV EF measured using Simpson's approach demonstrated that this value was significantly lower in athletes with increased LVMM. Sa values in sportsmen with borderline LVH did not differ significantly from the same value in the group of patients with normal LVMM.

According with the results of Vinereanu D et al., 2001 [24], average systolic speed of fibrous ring movements less than 9 cm/sec should be considered as the most significant criteria of pathological LVH (sensitivity 87%, specificity 97%). In our study the average systolic speed of mitral valve fibrous ring movements in athletes with LVH was 10.1±2.8 cm/sec that can be possibly caused by initial steps of hypertensive LV myocardial remodeling. Other parameters of LV myocardium DF function in the group of sportsmen with LVH were normal.

7 out of 13 athletes with elevated BP verified with 24h BP monitoring had border-line LVH or LVH that allowed prescribing them antihypertensive therapy with angiotensin converting enzyme (ACE) inhibitor enalapril (5 mg/day). Evaluation of 3-month therapy efficacy is present at Figure 1.

According with the results of our previous studies that had been performed in 47 boat racing athletes (aged 17-19 years) high normal BP was detected in 8.5% of cases, and AH 1 stage was found in 25.6% of cases. Apart from it, these sportsmen with elevated BP had LVMMI=89.07 g/m^2 comparing with 74.6 g/m^2 in sportsmen with normal BP [25]. At the same time, there was no correlation between LVMM and the level of physical functionality in athletes with elevated BP (r = 0.17).

Results of our current study suggest that athletes with AH, apart from particular exercise program, had

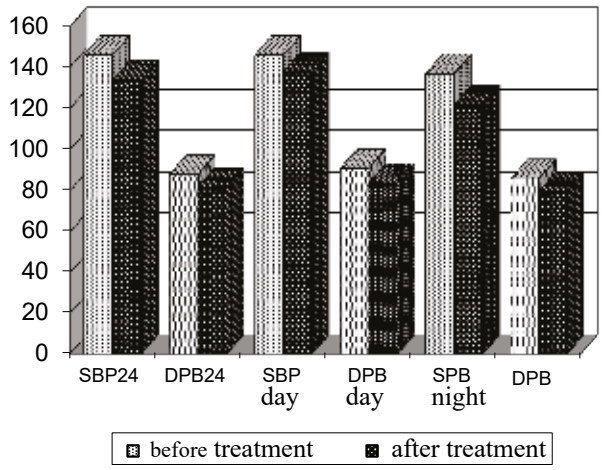

Figure 1. Dynamics of BP parameters in athletes before and after treatment

more isometric and speed-strength loads that could have influenced AH development, and myocardial hypertrophy in sportsmen with AH could be caused by increased post-load. The presence of elevated BP and increased LVMM evaluated as target organ lesion allowed us using ACE inhibitor enalapril as the drug of choice. Results of 3-month treatment have led to significant reduction of 24h-BP monitoring parameters, but they did not favour regression of myocardial hypertrophy.

Standard criteria of the Russian Society of Cardiology (RSC) should be used for estimation of BP levels and AH stage in athletes; apart from it, RSC guidelines discuss the tactics of observation of sportsmen with elevated BP [26].

Before starting training and competition activity athletes should undergo routine BP measurement, and in case of elevated BP (>140/90 mm Hg) it is reasonable to measure "not-office" BP in order to exclude white coat hypertension. Sportsmen with BP in the range of 120-139/80-89 mm Hg are advised to change the lifestyle and minimize possible AH risk factors without changing physical activity. In case of resistant BP elevation it is recommended to perform EchoCG for differential diagnostics of athletic heart and LVH. The presence of LVH is an indication for restriction of physical training and possible pharmacological correction.

The presence of AH 1 stage in case of no target organs' lesions like LVH or concomitant cardiac disorders do not restrict training and competition activity of sportsmen, but it is recommended to measure BP every 2-4 month to control the influence of physical exercise on BP.

Athletes with more severe AH (II-III stage), even if they have no evident target organs' lesions like LVH,

should be kept away from static exercises of high intensity (IIIA, IIIB, IIIC classes of sports) unless lifestyle change or pharmacological treatment would help to normalize BP levels.

When prescribing antihypertensive therapy it is necessary to take into account the fact that athletes belonging to registered pool of international federation or participating in international competitions can receive medications only according with the rules of their international federation.

Talking about young athletes, American Academy of Pediatrics recommends to admit children and adolescents with AH to training and competitions if the absence of target organs lesions or concomitant cardiac pathology is proved; it is advised to perform control ECG every two months. Sports with high static (isometric) loads are not recommended to young athletes with severe AH, even if there are no evidences of target organs' lesions [27].

In conclusion it is worth to notice that AH prevalence in athletes is enough low and is 50% less than in general population, but it increases significantly with age. At the same time, it does not mean that the problem AH is not relevant for the athletic population, since 8% of sudden cardiac death cases of sportsmen younger than 35 years are caused by non-differentiated myocardial hypertrophy (according with autopsy results), and in fact it is the only proved cause of sudden death in this population. Major epidemiologic studies demonstrated that LVMH is an independent risk factor of cardiovascular disease development. According with the results of the Framingham study, LVMM increase by 50 g/m^2 correlates with 2.21 and 1.73 times increase of 4-years risk of cardiovascular disease in females and males, respectively, LVH is an independent risk factor for heart failure, coronary heart disease (CHD), ventricular arrhythmias and sudden death. LVH presence is responsible for 5-fold increase of congestive heart failure risk. The presence of concomitant CHD increases three times the risk of lethality, and myocardial infarction increases this risk four times. CHD is the main cause of sudden death of sportsmen above 35 years old; it is very likely that development of coronary atherosclerosis in athletes is caused by the presence of LVH and AH that had not been diagnosed in time. Whereas the only morphological substrate for the main cause of sudden death in athletes above 35 years old is CHD, it is impossible to exclude such direct cause of it like malignant arrhythmias possibly caused by myocardial fibrosis; it requires further studies of electric remodeling of sportive heart [9, 10, 11]. Taking into account all the mentioned above facts, it is necessary to control precisely BP in athletes including children and adolescents especially in sports requiring intense speed-strength and isometric loads. Diagnostic procedures should include EchoCG diagnostics and stress tests. Sportive activity can be allowed only in case of well-controlled BP and low risk of cardiovascular complications. Nowadays there is a hypothesis that moderate aerobic loads after intensive speed-strength training may be one of approaches for AH prevention in athletes.

The question of AH pharmacological therapy in athletes is the subject of further investigation, since there are not enough evidences. According with several researchers [28], it is more relevant to prescribe ACE inhibitors, angiotensin-1 receptor blockers and dihydropyridine calcium channel blockers, because the use of other drugs like diuretics and beta-blockers is restricted by the World Anti-Doping Agency.

Conflict of interest: None declared.

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