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Hypertension and other risk factors of atrial fibrillation in athletes

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Abstract

Cardiovascular mortality in athletes in 50% of cases is due to cardiovascular diseases (CVD). Among them heart arrhythmias, in particular atrial fibrillation (AF) comprise a significant part. Therefore, the assessment of risk factors for AF is extremely important in athletes. We summarize the data of recent experimental and clinical studies on the relationship of physical activity, hypertension, obesity, carbohydrate metabolism disorders, genetic aspects and AF. The diagnostic approaches to heart rhythm disorders in athletes are discussed. We also indicate the directions of further research. The pathogenetic rationale for diagnostic strategies for AF in athletes is developed.

Key words: atrial fibrillation, athletes, risk factors, heart rhythm disorders

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Влияние артериальной гипертензии и других факторов риска на развитие фибрилляции предсердий у спортсменов

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Резюме

Сердечно-сосудистая летальность у спортсменов в 50 % случаев обусловлена сердечно-сосудистыми заболеваниями, среди которых значимую роль занимают нарушения сердечного ритма (НРС), в частности фибрилляция предсердий (ФП). В связи с этим особую значимость приобретает изучение факторов риска ФП в этой группе пациентов. На основе анализа экспериментальных и клинических исследований последних лет суммированы данные о взаимосвязи физических нагрузок, артериальной гипертензии, ожирения, нарушений углеводного обмена, генетических аспектов и ФП. Рассмотрены особенности диагностики данного НРС у спортсменов. Обосновано и сформулировано с позиций клинической значимости направление дальнейших исследований. Усовершенствовано патогенетическое обоснование данной проблемы, что открывает новые направления поиска диагностических стратегий ФП у лиц, профессионально занимающихся спортом.

Ключевые слова: фибрилляция предсердий, спортсмены, факторы риска, нарушения ритма сердца

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Introduction

The problem of reducing of premature mortality and identifying the compliance of the possibilities of the cardiovascular system with the level of training and competitive loads are the main tasks of modern sports medicine. However, the question of the biological feasibility of certain changes in the heart in the process of adaptation to muscular activity has not yet been resolved. Research studies show that exercises in moderate dosage help to normalize the function of the organs of athletes, while overtraining can lead to serious consequences [1].

Most scientific papers indicate that, compared to the general population, professional athletes have double higher risk of sudden death, in 50 % of cases due cardiovascular diseases (CVD) significant cardiac rhythm disorders in particular atrial fibrillation (AF) [1–3].

Literature data confirms that AF in professional athletes are diagnosed 2 to 10 times more frequently than patients with normal physical activity. In addition, mortality after thromboembolic complications due to AF in athletes reaches 6–7 % [4, 5].

The contribution of this tachyarrhythmia to cardiovascular mortality is beyond doubts. However, diagnostic approaches and risk factors for AF in athletes have not been studied enough.

The pathogenesis of AF in athletes

The most important factor influencing on the development of AF is physical activity. The long-term adaptation of athletes to motor activity of varying intensity is accompanied by specific morphological and functional alterations of the heart and blood vessels. In addition, parasympathicotonia is a feature associated with AF in this group of patients. In more than 68 % of cases of AF in athletes associated with hypertonus of the vagus nerve [6–9]. The sensitivity of baroreceptors and sensitivity of acetylcholine receptors increase in the athletes as a result of inhibition of regulation of G-signal proteins [10]. L. Mont and co-authors in their work (2008), conducted on the basis of a survey of 1160 men, showed that active athletes developed AF 5 times more often than people of the sedentary lifestyle. Scientists explained this circumstance by higher values of the mass of the myocardium, the size of the left atrium, the thickness of the myocardium of the left ventricle and vagotonia [11]. These data are the same data in the prospective Kasper study conducted in 52755 athletes [12]. Many researchers have noted the high prevalence of AF in individuals who train endurance quality in high-dynamic, high-intensity sports, such as marathon running, biathlon, orienteering, and cross-country skiing. In this group of patients, AF is detected in 5–10 % of cases [13–15].

Experimental confirmation of the relationship of AF and exercise intensity is the work of S. Baldesberger et all (2008). Cardiac arrhythmias in cyclists were diagnosed in 10 % of cases, whereas golfers did not have any paroxysms [12].

Most authors note a certain link between the development of AF and the duration of physical exertion. Athletes training more than 10 hours a week for more than 10 years have a significant high risk for the development of arrhythmias. A reliable relationship was established between the formation of AF, the degree of dilatation of the left atrium and the number of training hours during the period of a sports career. The increase in the volume of the left atrium depends on the duration of sports in life. An increase in the volume of the left atrium is detected in 24 % of athletes engaged in less than 1500 hours, in 40 % of athletes engaged in from 1500 to 4500 hours, and in 83 % of athletes engaged in more than 4500 hours [4, 5, 11, 16]. This fact is explained by vagotonia, volume loading with the development of hypertrophy and dilation of the heart chambers. These changes are the basis of LDCs [12, 17]. However, ambiguous results were obtained in a prospective analysis of 4 thousand people with a sedentary lifestyle and athletes with AF performed by L. Mont et al. (2008). There were no differences in the degree of hypertrophy and dilatation of the left ventricle, dilatation of the left atrium [11]. This fact can be explained by the presence of diseases associated with hypodynamia and their influence on the morphofunctional parameters of the heart. The development of paroxysmal form of AF, characteristic feature in athletes, was described in these studies. This fact requires additional large-scale research. Persons with a permanent form of AF are not able to perform prolonged physical exertion.

Genetic determinism of AF in athletes

Genetic determinism is the most important factor among non-modifiable risk factors for AF [17, 20]. One of the recently discovered causes of sudden death was the presence of mutations in the triadin gene. The pathology of triadin receptors responsible for calcium currents is associated with the development of life-threatening ventricular LDCs. Data obtained by M. J. Ackermann, showed that RyR 2 mutation is the main cause of catecholaminergic ventricular tachycardias in young people against the background of physical or psycho-emotional stress. It is known that physical exertion can be a trigger for the development of ventricular arrhythmias in the syndrome of prolonged QT interval. Mutations in the triadine gene are considered as a new genetic basis for the formation of a recessively inherited syndrome of an extended QT type 1 interval [21]. The obtained data require further research and search for relationships in the formation of ventricular and atrial arrhythmias due to the lack of clear information on the effect of physical exertion on the athlete's genotype.

The role of arterial hypertension in the development of AF of athletes

One of the reasons predisposing to the development of AF is arterial hypertension. Intensive sports exercises do not exclude the likelihood of

hypertension in an athlete, despite the fact that high physical activity is considered a factor in the primary prevention of this disease. According to research conducted by A. V. Smolensky (2015), the prevalence of hypertension among young athletes (under 35 years old) is 5.6% [22]. The high level of sodium consumption, frequent stress, alcohol abuse, the use of doping drugs play a huge role in the development of hypertension in this group of patients. Similar data were obtained in studies of the status of CVD in groups of teenagers, athletes and non-athletes [24, 26]. The formation of hypertension in athletes is due to the effects of tachycardia, daytime sympathicotonia, volume overload, and development of ventricular diastolic dysfunction. [6, 9, 27, 28]. Today there is no definitive answer on the question of whether hypertension, which develops directly during a sports load, is a risk factor of hypertension [29].

Fibrous myocardial degeneration in athletes

It has been experimentally proven that cardiac fibroblasts play a key role in myocardial restructuring. The function of these cellular structures is modulated by various factors, such as mechanical stretching, oxygen levels, changes in the metabolism of the extracellular matrix, cell proliferation and migration processes, high values of proinflammatory cytokines, growth factors, vasoactive peptides and hormones [30, 31]. Activated cardiac fibroblasts are transformed into myofibroblasts, which synthesize collagen and other proteins of the extracellular matrix, precipitated in the interstitial space. All these processes lead to the formation of fibrosis [32]. Recent studies have demonstrated that cardiac fibroblasts undergo remodeling during tachyarrhythmias [33]. An increase of atrial fibrosis and fibrosis of pulmonary veins contributes to the blocking of conductivity, recurrent excitation and the occurrence of trigger activity [34–36]. B. Benito (2011) confirms in his research that athletes of all age groups are characterized by the formation of atrial and ventricular myocardial degeneration against the background of increased production of profibrotic markers: fibronectin 1, procollagen 1 and 3, transforming growth factor B 1, matrix metalloproteinase 2, tissue inhibitor metalloproteinases 1. According to some authors, the process of fibrosis in miocardium in athletes is enhanced and accelerated [28, 37, 38]. The level of profibrotic factors increases with the time of the sporting experience and the age of the athlete [39]. Similar data were obtained by A. Wolk and co-authors (2014) during a survey of 45 elite athletes with AF. In these patients, pronounced myocardial fibrosis was confirmed by to echocardiography and magnetic resonance imaging, and an increased level of the profibrotic metalloelastase enzyme. The comparison was made with an identical group of people not involved in sports [5]. Studies of J. Drezner (2016), S. Bondarev et al. (2017) also indicated a high degree of fibrous degeneration of the myocardium of the ventricles and atria in athletes on magnetic resonance imaging with gadolinium [40, 41].

Disorders in the exchange of athletes with AF

The problem of overweight for athletes is sometimes no less relevant than for people with a sedentary lifestyle. An imbalance in nutrition, the use of high-calorie foods and supplements, taking a lot of medicines, the formation of certain eating habits, the absence of a decrease in calories after the end of a career contribute to the obesity formation. According to large-scale prophylactic examinations in the USA, the prevalence of this pathology among young athletes is 23.5% [42, 43]. This problem is most relevant for athletes who have completed their careers. In these individuals, obesity is detected 3 times more often than in the population. Overweight causes chronic systemic inflammation, which leads to the formation of structural remodeling and interstitial fibrosis of atrial myocardium and changes in the electrophysiological properties of cardiomyocytes [44, 45]. Abdominal fat is metabolically active due to free fatty acids and numerous cytokines. Cytokines are the triggers of activation of profibrotic components, both directly and indirectly due to the development of insulin resistance.

The important risk factor of AF in athletes is carbohydrate metabolism disorders. This is facilitated by the use of high-energy nutrition and the use of steroids [24, 46 48]. Hyperglycemia and hyperinsulinemia increase the proliferation of fibroblasts and vascular smooth muscle cells, contribute the restructuring of the myocardium and vessels. Hyperinsulinemia also activates the sympathoadrenal system, leading to vasoconstriction and an increase in cardiac output, increases the synthesis of triglycerides, cholesterol, very low density lipoproteins in the liver, damaging the intima of the arteries, activates the renin-angiotensin system. Such changes increase the total peripheral and renal vascular resistance, violate transmembrane ion-exchange mechanisms, cause electrolyte imbalance, and increase the reabsorption

Table

RESULTS ECHOCG STUDIES IN HEALTHY ATHLETES ACCORDING TO VARIOUS AUTHORS

Author	Year	N	Type of sport	Parameter	Gender	Mean Value	Cut-off Value
Peliccia	1999	1309	M	LV End diastolic diametr, мм	M	55	70
Whyte	2004	442	P	LV End diastolic diametr, мм	F	49	65
Pelliccia	1996	600	P, M	LV End diastolic diametr, мм	F	49	66
Makan	2005	900	E	LV End diastolic diametr, мм	F, M, teenagers	51	60
Spirito	1994	947	P, M, E, S	LV whol thickness, мм	M	10	16
Rawlins	2010	440	E	LV whol thickness, мм	M, blacks	9,5	13
Sharma	2001	720	E, P	LV whol thickness, мм	F, M, teenagers	9,5	12
Basavarajaiah	2008	300	E, P	LV whol thickness, мм	M, blacks	11,5	16
Caselli	2015	1145	M, P, E, S	LV mass/bsa, g/m^2	M, F	103	146
Finocchiaro	2016	1083	M, P	LV mass/bsa, g/m^2	M F	101 83	143 117
Pelliccia	2005	1777	E, P, M, S	LA antero/ posterior diametr, mm	M F	37 32	50 45
D'Andrea	2010	650	P, E	La volum index, ml/m^2	M F	28 26	36 33
Pelliccia	2010	2317	P, E	Aortic toot diametr, mm	M F	32 28	40 34
D'Andrea	2010	615	P, E	Proximal ascending aorta, mm	M	28	34
Caselli	2015	1145	P, S, E	LV ejection fraction, % E/A TDI e' septal, sm/s TDI e'/a' septal, sm/s E/e' septal, sm/s TDI e' later sm/s TDI e'/a' later sm/s E/e' later sm/s	M & F M & F M & F M & F M & F M & F M & F	64 1.93 13.8 2.04 6.4 15 1.45 5.8	55 1.3 10.3 1.23 8.5 9 1.2 8.0
D'Andrea	2013	650	P, E	RV diastolic basal, mm RV diastolic median diameter, mm RV longitudinal diastolic diameter, mm	M F M F M F	43.5 39 34 32 89 82	55 49 47 43 109 100
D'Ascenzi	2016	1009	P, S, E, M	RV diastolic area, sm^2 RV systolic area, sm^2 RA area, sм^2	M F M F M F	25.1 19.3 12.1 9 18.9 14.8	33 27 18 14 18 14

Note: BSA — body surface area; TDI — Tissue Doppler imaging; LV — left ventricle; RV — right ventricle; LVM — left ventricular myocardium mass; RV — right ventricular myocardial mass; LA — left atrium; P — power; E — endurance; S — skillet; M — mixed.

of sodium and water [49]. The above changes may potentiate the arrhythmogenic effect.

Athletes are characterized by modifiable and non-modifiable risk factors for AF. The modifiable factors include excessive physical activity, hypertension, obesity, and disorders of carbohydrate metabolism. Non-modifiable factors include genetic predisposition. According to recent studies, such conditions are associated with autonomic dysfunction, volume loading with the development of hypertrophy and dilatation of the heart chambers. The insufficiency and ambiguity of the experimental data require further large-scale research.

Examination of athletes with AF

For the diagnosis of AF in athletes, a thorough history and complaints are taken, vegetative balance and risk factors are assessed, an electrocardiogram (ECG), daily monitoring ECG, echocardiography with an assessment of the ventricular diastolic function, stress tests, magnetic resonance imaging of the heart with degree analysis fibrous myocardial degeneration, medical and genetic studies are held. Despite the great potential of clinical and laboratory and instrumental methods of research, the identification of AF in professional sports is associated with great difficulties. In some cases, this is due to the asymptomatic course of this tachyarrhythmia, late examination, psychological characteristics of the personality of athletes. However, the keystone is the problem of the "norm" of the morphofunctional parameters of the cardiovascular system. According to the opinion of a number of authors in sportsmen, it is advisable today to consider echocardiography indicators at the level of the reference values indicated in the table [50].

The appearance of signs of impaired myocardial relaxation, registered by the method of constant-wave Doppler research, tissue Doppler sonography, is considered as a component of atrial remodeling [6, 40, 41].

Athletes with high normal pressure (less than 140/90 mm Hg) can be admitted to competitions without restrictions, while observing general dietary and lifestyle recommendations and/or receiving antihypertensive drugs [50].

Clinically significant paroxysmal AF in athletes are AF episodes lasting ≥5 minutes [9, 43]. The main methods of registration of AF are a 12-lead ECG and 24-hour ECG monitoring. A feature of AF in athletes is the high likelihood of paroxysm development directly during training or after training.

Conclusions

The registration of tachyarrhythmias and their interpretation by a specialist can be ensured only with the help of telemedicine technologies. These diagnostic measures are included in the American and European guidelines for the conduct of athletes [40, 51]. The experts of the American Society of Sports Medicine (2016) recommended to make an individual decision regarding each athlete at any age (including those under 25) in order to prevent sudden arrhythmic death.

Thus, excessive physical activity, hypertension, obesity, carbohydrate metabolism disorders and genetic predisposition through various points of application contribute to the formation of AF in athletes. However, the lack of a complete understanding of the mechanisms by which the proarrhythmic effect is realized, as well as the difficulties in diagnosing of AF, contribute to cardiovascular mortality. Problems of athletes with AF demand the future researches.

Conflict of interest / Конфликт интересов

The authors declare no conflict of interest. / Авторы заявили об отсутствии конфликта интересов.

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