Aortic properties and atrial electrophysiology in the young and old football players

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Summary

Objective: The purpose of this study was to investigate the differences between P wave dispersion, aortic elastic properties and transthoracic echocardiographic findings in the young and old football players compared to control groups in order to asses the influence of regular sportive activity on aortic distensibility and its potential effect on atrial electrophysiology. **Methods**: We recruited 42 young football players with a training history of many years. The control group was formed by 27 healthy sedentary men. Twenty-three healthy retired football players of a professional football club aged over 50 years were included in the study as old group and 18 subjects over 50 year old who did not perform regular exercise when they were young were included in the control group of old subjects. **Results:** The heart rate and ejection fraction were decreased in the young football players. There were no significant differences in the aortic elastic parameters and P wave dispersion between young football players and control group. But in old subjects with sustained participation in regular sportive activity, the significant difference of left ventricular dimension, wall thickness and systolic functions detected in the young group disappeared while increase in the left atrial diameter became significant. **Conclusion:** Potential effect of aortic elastic properties which changes with age, on atrial electrophysiology through increasing P wave dispersion was shown.

Keywords: Sports medicine; atrial fibrillation; cardiac electrophysiology.

Resumo

Propriedades da aorta e electrofisiologia atrial em futebolistas profissionais ativos e aposentados

Objetivo: Com este estudo pretendeu-se levar a cabo um ensaio clínico que permitisse investigar as diferenças entre a dispersão da onda P (DOP), as propriedades elásticas da aorta e os resultados da ecocardiografia transtorácica em futebolistas profissionais ativos e reformados, face a grupos de controle, de modo a avaliar a influência da atividade desportiva regular na distensibilidade aórtica e o seu potencial efeito na electrofisiologia atrial. **Métodos:** Para este estudo foram recrutados 42 jovens futebolistas profissionais com um histórico de treino de vários anos. O grupo-controle foi constituído por 27 homens saudáveis e sedentários. No grupo composto por indivíduos mais velhos, foram incluídos 23 futebolistas profissionais já aposentados, saudáveis e com mais de 50 anos de idade e, como grupo-controle, 18 indivíduos com mais de 50 anos de idade que nunca fizeram qualquer tipo de exercício físico regular quando eram mais novos. **Resultados:** A frequência cardíaca e fração de ejeção eram menores nos jovens futebolistas profissionais. Não se verificaram diferenças significativas nos parâmetros de elasticidade da aorta e a dispersão da onda P entre os jovens futebolistas profissionais e o grupo-controle. Mas, já no caso do grupo dos indivíduos mais velhos com uma atividade desportiva regular, a diferença significativa na dimensão ventricular esquerda, espessura das paredes e funções sistólicas detectada no grupo jovem e ativo desapareceu, enquanto o aumento no diâmetro atrial esquerdo tornou-se expressivo. **Conclusão:** Demonstrou-se com este estudo o efeito potencial das propriedades elásticas da aorta, que se alteram com a idade, na electrofisiologia atrial por meio do aumento da dispersão da onda P.

Unitermos: Medicina esportiva; fibrilação atrial; eletrofisiologia cardíaca.

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Submitted on: 01/20/2011
Approved on: 03/02/2011

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Conflict of interest: None.

Introduction

Football is one of the most popular and widely viewed sports in the world. Executing such movements during performance, both aerobic and anaerobic metabolic systems appear to be involved throughout a game[1]. Widely used in sports medicine, the transthoracic echocardiography allows quantitative assessment of cardiac structure and function for the athletes. The elite athletes often exhibit some changes in the heart, called athlete's heart[2,3]. Athlete's heart is an enlarged heart related to repeated strenuous exercise. As a result of exercise, the heart will expand physiologically by enlarging chambers, increasing muscle mass and increasing the volume of blood pumped per stroke. The athletes heart is associated with some types of electrocardiogram (ECG) abnormalities, such as sinus bradycardia, sinus arrhythmia, early repolarization, first-degree atrioventricular block and left atrial enlargement[4].

Atrial fibrillation is the most frequent cause of prolonged palpitations in young competitive athletes, even including those performing elite sport activity[5]. Endurance sport practice increases the probability of suffering of atrial fibrillation, after adjusting for other risk factors[6]. The possible mechanisms explaining the association remain speculative. Atrial ectopic beats, inflammatory changes, and atrial size have been suggested[6]. P wave dispersion, detected from the surface ECG, have been though to reflect left atrial enlargement and altered conduction[7,8]. P wave dispersion and P wave maximal duration, that reflect the activation of atrial muscle and may depend primarily upon the mass of tissue excited, have been used in the assessment of the risk for atrial fibrillation which is characterized by nonhomogeneous and discontinuous atrial conduction[7,8]. P wave dispersion was defined as the difference between the longest and the shortest P wave duration recorded from multiple different surface ECG leads[7]. The clinical significance of P wave duration has been demonstrated in many clinical conditions, especially in paroxysmal atrial fibrillation[8]. P wave dispersion has been shown to be influenced by the autonomic nervous system activation, which induces changes in left atrial size and the velocity of impulse propagation[9].

Arterial compliance plays a role in determining both arterial systolic and diastolic pressure and therefore, in a clinical context, influences left ventricular size and function and coronary blood flow[10-12]. Aerobic exercise has well-documented efficacy for cardiovascular risk reduction, and it appears that at least part of its benefit derives from modification of arterial properties[13]. Non-invasive ultrasound techniques such as echocardiography and aortic pulse wave velocity are used to evaluate vascular system and cardiovascular condition[11,12].

No studies cited in the literature suggest the young and old football players are at increased risk for atrial fibrillation and arterial stiffness. The purpose of this study was to investigate and compare P wave duration, P wave dispersion, aortic elastic properties (aortic strain, aortic distensibility) and transthoracic echocardiographic findings in the young and old football players.

Methods

Subject's population

We recruited 42 young football players with a training history of many years. They were the members of a local football team and they were regularly maintaining sportive activities and training programs. Twenty-seven healthy sedentary men formed the control group. Healthy and over 50 years old retired 23 football players of a professional football club were included in the study as old group and 18 subjects over 50 years old who did not perform regular exercise when they were young were included in the control group of old subjects. A detailed history was taken and each participant underwent a systemic physical examination to exclude cardiovascular or other relevant disease before attending to the study. Any subjects who had a history of cardiovascular or any other systemic disorders such as hyperlipidemia, hyperglycemia, anemia and those on medications known to alter cardiac conduction were excluded from the study. All subjects were non smokers and non alcoholics. All subjects gave their consent for inclusion in the study. The investigation conforms to the principles outlined in the Declaration of Helsinki.

Body mass index measurement

Body mass index (kg/m²) was calculated dividing body weight in kilograms by square of body height in meters.

Blood pressure measurement

The arterial blood pressure of the subjects was measured by the same clinician. The subjects were in the supine position and had rested at least 20 minutes before the measurement. The blood pressure was measured, using a mercury sphygmomanometer with a cuff appropriate to the arm circumference (Korotkoff phase I for systolic blood pressure and V for diastolic blood pressure). Blood pressure measurements were performed twice for each subject and their mean was used for statistical analysis.

Pulse pressure = systolic blood pressure - diastolic blood pressure

Mean blood pressure = [systolic blood pressure + 2 x diastolic blood pressure] / 3

P wave dispersion measurement

P wave duration was measured in all simultaneously recorded 12 leads of the surface ECG. All recordings were performed in the same quiet room during spontaneous breathing, following 20 minute of adjustment in the supine position. P wave duration measurements were obtained manually by two of the investigators using calipers and magnifying lens for accurate definition of the ECG deflection as defined in previous study[7,8]. The onset of the P wave was defined as the point of the first visible upward departure of the trace from the bottom of the baseline. The return to the baseline of the bottom of the trace in wave was considered to be the end of the P wave. P maximum in any of the 12 lead surface ECGs was measured and used as a marker of prolonged atrial conduction time. The difference between P wave maximum and P wave minimum durations was defined as P wave dispersion.

Transthoracic echocardiography

A Vivid 3 cardiovascular ultrasound system [3S sector probe (1.5 - 3.6 MHz), GE] was used for transthoracic echocardiographic evaluation[14]. Echocardiography was performed with the subject in the lateral decubitus position. Interventricular septal thickness (IVS), left ventricle posterior wall thickness (LVPW), left ventricle end-diastolic (LVED) and end-systolic diameters (LVES), left atrial diameter (LAD), aortic root diameter and aortic valve openness were measured in the parasternal long-axis view. The measurements were obtained from two-dimensional guided M-mode recordings. The pulsed Doppler sample volume was positioned at the mitral leaflet tips. Early diastolic peak flow velocity (E) (m/s) and late diastolic peak flow velocity (A) (m/s) were measured by transmitral Doppler imaging. After the routine conventional echocardiographic examination was performed, subjects were placed in a mild recumbent position, and the ascending aorta was recorded in the two-dimensional guided M-mode tracings. Aortic diameters were recorded 3 cm above the aortic valve by M-mode echocardiography. Aortic systolic diameter was determined at the time of the full opening of the aortic valve, and aortic diastolic diameter was determined at peak QRS. The same blinded investigator performed the echocardiography and the echocardiograms were analyzed by two blinded cardiologists.

Aortic strain was calculated as follows:
Aortic strain = (AoS - AoD) / AoD
(AoS, systolic aortic diameter, AoD, diastolic aortic diameter)

Aortic distensibility was calculated as follows:
Aortic distensibility = 2 x (AoS - AoD) / (AoD x PP)
(PP, pulse pressure)[15,16]

Statistical analysis

The statistical analysis was done using the SPSS (version 16.0) ready-to-use programme. All values were expressed as mean ± standard deviation. Mann-Whitney U non-parametric tests were done. Significance limit was accepted as $p \leq 0.05$.

Results

In the young football players group, LVED, LVES, LVPW, IVS and LAD were increased in compared to healthy sedentary subjects ($p < 0.001$; $p < 0.001$; $p < 0.001$; $p = 0.004$; $p = 0.003$; respectively). On the contrary; the heart rate and ejection fraction were decreased in the young football players group ($p < 0.001$; $p = 0.005$; respectively). There were no significant differences in the aortic elastic parameters and P wave dispersion between two groups ($p > 0.05$) (Table 1). In the old football players group while LVED, LVES, LVPW, IVS were not significantly different from their control group, LAD was significantly increased ($p < 0.001$). Furthermore, the difference in heart rate and ejection fraction disappeared along with aging. Although a significant difference in aortic elastic properties and P wave dispersion was not revealed in young groups, P wave dispersion was significantly higher in old football players in compared to old control group ($p = 0.004$). Whereas, aortic strain was significantly lower in old football players group ($p < 0.001$), aortic distensibility was significantly higher in compared to old control group ($p < 0.001$) (Table 2).

Discussion

High intensity dynamic endurance exercise is associated with cardiac morphologic changes, including increased left ventricle systolic and diastolic diameter, and wall thickness as in our study[2,3]. An increased ejection fraction on dynamic exercise was not different from control in elite athletes[17,18]. However, fractional shortening or ejection fraction at rest was significantly higher or depressed (within normal limits) in athletes[19,20], as we found in our study. Long-term dynamic exercise is usually associated with the rhythm and conduction abnormalities such as sinus arrhythmia, early repolarization and first-degree atrioventricular block[4]. Among these are sinus bradycardia, as we found in our study, due to high resting vagal tone[19,20]. It has also been suggested that there are intrinsic changes within the sinoatrial and atrioventricular nodes, including prolonged sinus node recovery time. It is thought that athletes are vulnerable to atrial fibrillation because of enhanced parasympathetic activity and consequent bradycardia is like by to cause dispersion of atrial repolarization[21]. This dispersion of atrial repolarization can facilitate atrial fibrillation. P wave dispersion is associated with inhomogeneous and discontinuous propagation of sinus impulses[22]. The non-homogeneous propagation of sinus impulses and the prolongation of atrial conduction time are electrophysiologic characteristics in patients with atrial fibrillation[22]. Some studies showed that endurance-trained subjects present higher values of power spectral heart rate variability, indicating increasing parasympathetic activity[22,23]. Chen *et al.*[24], showed that the patients with atrial fibrillation have a greater increase in atrial size than

Table 1 – Anthropometric, hemodynamic, laboratory, echocardiographic and electrocardiographic values in the young groups

	Football players (mean ± SD)	Control (mean ± SD)	p
Age (years)	22.5 ± 4.5	21.8 ± 3.36	0.62
Height (cm)	175.76 ± 5.96	175.14 ± 6.31	0.72
Weight (kg)	71.09 ± 8.85	72.48 ± 11.11	0.79
Waist (cm)	80.90 ± 5.08	77.14 ± 6.96	0.17
Hip (cm)	97.33 ± 5.61	96.85 ± 6.46	0.97
Body mass index (kg/m^2)	23.00 ± 2.52	23.58 ± 3.04	0.69
Systolic blood pressure (mmHg)	107.14 ± 9.43	110.55 ± 9.83	0.25
Diastolic blood pressure (mmHg)	69.04 ± 9.95	68.88 ± 6.25	0.88
Pulse pressure (mmHg)	38.09 ± 6.22	41.66 ± 7.20	0.06
Mean blood pressure (mmHg)	82.85 ± 9.66	82.77 ± 6.83	0.67
Heart rate (beats/min)	62.04 ± 8.48	75.66 ± 9.62	< 0.001
LVED (mm)	52.47 ± 3.12	48.18 ± 2.70	< 0.001
LVES (mm)	32.95 ± 3.02	29.00 ± 3.17	< 0.001
Ejection fraction (EF) %	65.71 ± 4.25	70.07 ± 4.89	0.005
LVPW (mm)	11.33 ± 0.73	9.33 ± 1.46	< 0.001
IVS (mm)	11.23 ± 0.83	10.51 ± 0.70	0.004
Left atrium (mm)	35.66 ± 2.05	32.92 ± 3.22	0.003
Aortic root (mm)	27.76 ± 1.84	28.03 ± 2.90	0.72
Aortic valve openness (mm)	18.66 ± 1.98	19.48 ± 2.13	0.15
Mitral E velocity (cm/s)	1.27 ± 0.17	1.27 ± 0.14	0.98
Mitral A velocity (cm/s)	1.01 ± 0.13	0.92 ± 0.15	0.05
Pulmonary artery velocity (cm/s)	1.08 ± 0.17	1.03 ± 0.12	0.50
Aortic strain (%)	5.02 ± 2.08	5.34 ± 2.25	0.62
Aortic dist ($cm^2.dynes^{-1}.10^{-6}$)	0.27 ± 0.13	0.26 ± 0.12	0.86
Maximum P wave duration (ms)	86.66 ± 13.16	83.70 ± 9.66	0.42
Minumum P wave duration (ms)	41.90 ± 6.01	40.37± 7.06	0.29
P wave dispersion (ms)	44.76 ± 10.77	43.33 ± 11.43	0.90
Hemoglobin (g/dl)	15.11 ± 0.87	14.85 ± 0.74	0.30
Hematocrit (%)	43.98 ± 2.95	44.92 ± 3.13	0.18

SD, standard deviation; LVED, left ventricle diastolic diameter; LVES, left ventricle systolic diameter; LVPW, left ventricle posterior wall thickness in diastole; IVS, interventricular septum thickness in diastole; Dist, distensibility

those without atrial fibrillation. It was also shown that elite junior athletes have a significantly greater left atrial diameter as in our study[4,25]. Therefore, it would be expected that P wave dispersion and P wave duration could be increased in the young football players. In contrast, we found no significant differences in P wave duration and P wave dispersion between young football players and control group. However, some studies showed that maximal left atrial diameter is not a significant predictor of atrial fibrillation and there is no correlation between filtered P wave duration and left atrial enlargement[8,26]. But the significant association which was shown in between P wave dispersion and LAD in old group in this study proved the influence of aging on atrial electrophysiology, independent of increase in LAD.

The aorta is a complex organ with multiple functions. It acts as both a conduit and an elastic buffering chamber. By virtue of its elastic properties, this vessel influences left ventricular functions and coronary blood flow[10,11]. It has been reported that aortic distensibility is decreased in patients with coronary heart disease[10,11]. On the other hand, some studies have shown an increase or decrease in these properties in trained athletes[27,28]. Dynamic exercise may play a critical role in aortic elastic properties and its caliper of the arterial lumen[27]. Altered arterial elastical responses to cardiac cycle may be due to changes of hemodynamic parameters and arterial wall properties[27]. The changed vascular response (increased arterial distensibility) might continue at rest period because of adaptation to regular exercise. In contrast, Bertovic *et al.*[28] showed that whole body arterial compliance is lower in strength-trained men than in age-matched sedentary controls. The lower arterial compliance of the athletic group was the higher pulse pressure (systolic blood pressure - diastolic blood pressure), measured both peripherally at the brachial artery and centrally at the com-

Table 2 – Anthropometric, hemodynamic, laboratory, echocardiographic and electrocardiographic values in the old groups

	Football players (mean ± SD)	Control (mean ± SD)	p
Age (years)	56..4± 2.5	58 ± 1.16	0.87
Height (cm)	165.77± 2.96	164.14 ± 2.01	0.42
Weight (kg)	78.08 ± 3.85	81.48 ± 1.4	0.12
Waist (cm)	87.90 ± 5.9	89.14 ± 2.76	0.89
Hip (cm)	105.33 ± 2.61	109.85 ± 5.0	0.90
Body mass index (kg/m^2)	29.00 ± 2.02	33.18 ± 7.04	0.86
Systolic blood pressure (mmHg)	128.19 ± 5.83	134.55 ± 8.86	0.88
Diastolic blood pressure (mmHg)	79.045± 9.85	83.55 ± 3.21	0.76
Pulse pressure (mmHg)	49.14 ± 4.21	51.00± 8.20	0.54
Mean blood pressure (mmHg)	88.35 ± 5.76	89.79 ± 9.66	0.71
Heart rate (beats/min)	75.96 ± 0.44	75.66 ± 8.62	0.97
LVED (mm)	47.77 ± 5.13	48.10 ± 6.90	0.23
LVES (mm)	30.55 ± 7.70	28.80 ± 0.15	0.13
Ejection fraction (EF) %	68.33 ± 2.85	69.07 ± 4.29	0.66
LVPW (mm)	10.33 ± 0.03	9.63 ± 9.12	0.48
IVS (mm)	10.11 ± 0.66	10.21 ± 0.65	0.33
Left atrium (mm)	39.09 ± 4.55	31.66 ± 4.20	< 0.001
Aortic root (mm)	33.36 ± 1.16	29.05 ± 4.87	0.28
Aortic valve openness (mm)	19.36 ± 5.97	18.58 ± 0.56	0.98
Mitral E velocity (cm/s)	1.07 ± 1.24	1.37 ± 1.12	0.58
Mitral A velocity (cm/s)	1.47 ± 0.67	1.86 ± 0.98	0.48
Pulmonary artery velocity (cm/s)	1.18 ± 0.67	1.11 ± 0.13	0.79
Aortic strain (%)	4.77 ± 3.758	5.21 ± 1.19	< 0.001
Aortic dist (cm^2.dynes^{-1}.10^{-6})	0.25 ± 0.09	0.21 ± 0.26	< 0.001
Maximum P wave duration (ms)	91.23 ± 11.45	85.70 ± 4.45	0.90
Minumum P wave duration (ms)	39.54 ± 5.09	40.22± 8.08	0.79
P wave dispersion (ms)	47.51 ± 80.11	44.31 ± 90.27	0.004
Hemoglobin (g/dl)	14.16± 0.66	14.57 ± 1.98	0.98
Hematocrit (%)	40.67 ± 4.76	38.33 ± 8.69	0.75

SD, standard deviation; LVED, left ventricle diastolic diameter; LVES, left ventricle systolic diameter; LVPW, left ventricle posterior wall thickness in diastole; IVS, interventricular septum thickness in diastole; Dist, distensibility

mon carotid artery. The difference in pulse pressure was attributable to both greater systolic and lower diastolic arterial blood pressure in the athletes. The higher systolic pressure of the athletic group was maintained at maximal exercise, indicating a greater afterload during aerobic exercise in the strength-trained group. However, the muscular strength-trained athletes have similar (as in our study) or lower pressures than the sedentary population[29,30].

Endurance exercise-trained middle-aged/older adults demonstrate lower large elastic artery stiffness and greater endothelium-dependent dilatation (EDD) than their sedentary peers. With daily brisk walking, previously sedentary middle-aged/older adults show reduced stiffness and improved EDD. The mechanisms underlying the effects of regular aerobic exercise on large elastic artery stiffness with aging are mostly unknown, but are likely to include changes of the composition of the arterial wall. Enhanced EDD in older adults who exercise is mediated by increased nitric oxide bioavailability associated with reduced oxidative stress. Aerobic exercise also may protect arteries in advanced ageing by increasing resistance to the effects of other cardiovascular diseases risk factors like LDL-cholesterol.

In conclusion, while the beneficial effects of exercise to aortic compliance were not evident in young footballers yet, they were evidently maintained in old football players group.

Study limitations

There might be some limitations in this study. Firstly, we did not measure sympathetic and parasympathetic system activation. Secondly, P wave dispersion measurement errors done with manual evaluation may be potential bias for observed results. However, manual measurement of P wave dispersion has been well accepted and used in several studies[7,8].

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