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Atrial function is altered in lone paroxysmal atrial fibrillation in male endurance veteran athletes

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Aims Intensive endurance sport practice is associated with an increased risk of atrial fibrillation (AF) in male veteran athletes. Paroxysmal AF (PAF) is the very beginning step of this disease. The description of atrial remodelling occurring at this early stage might enable to depict predictive factors of AF in veteran athletes in order to give them personalized recommendation according to their sport practice.

Methods and results Twenty-seven male endurance veteran athletes with documented PAF were retrospectively enrolled and compared with 30 control endurance athletes without documented AF, with similar training level, age, and cardiovascular risk factors. All subjects underwent a resting-electrocardiogram (ECG) to assess the electric remodelling of P-waves as well as an echocardiography, to evaluate the left and right atrial (LA, RA) anatomical and functional (assessed by 2D strain) remodelling. No difference was noted between groups for the ECG P-wave parameters. Atrial function was decreased in the PAF group, particularly the peak atrial longitudinal strain (L-ε-Max) of LA (29.3 ± 7.9% vs. 49.1 ± 7.8% respectively in the PAF group and in controls, $P < 0.0001$) and RA (36.5 ± 7.0% vs. 50.6 ± 10.2%, $P < 0.0001$). LA and RA volumes were also larger in the PAF group. Receiver operating characteristic analysis demonstrated that L-ε-Max of LA [area under curve (AUC): 0.957 ± 0.023] and RA (AUC: 0.901 ± 0.042) had the best ability to identify the athletes with PAF, far better than the anatomical parameters (AUC < 0.75 in all).

Conclusion Atrial function analysed by strain in echocardiography is strongly associated with PAF and might enable to identify male endurance veteran athletes at risk to develop AF.

Keywords atrial fibrillation • athlete • strain • atria

Introduction

Atrial fibrillation (AF) is the most common arrhythmia and has a major impact on morbidity and mortality in the general population.[1] The beneficial influence of regular sport practice to reduce cardiovascular risk is well accepted, and low-to-moderate intensity endurance exercise also decrease the risk of AF.[2] In contrast to the benefits of regular sport practice, a link between competitive sport practice, especially strenuous endurance exercise, and AF has been described.[3] Whereas AF occurs generally in the setting of structural heart disease or cardiovascular risk factors as hypertension or diabetes,[4] the pathophysiological mechanisms responsible for the onset of lone AF in athletes are not clearly defined. Intense sports practice, mainly endurance, is related to a morphological remodelling which involves the atria, as well as alterations in the autonomic system,[5] which can result in the initiation and maintenance of AF. After the onset of AF, AF will further promote an adverse atrial remodelling which will contribute to increase AF burden.[6] Advising endurance athletes to reduce their sport practice at an early stage of this mal-adaptative atrial remodelling might ensure them not to enter in this vicious circle of

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AF.[7] Therefore, it might be of interest to comprehensively describe the atrial remodelling occurring in endurance athletes with lone paroxysmal AF (PAF) in order to be able to predict the athletes at risk to develop this pathology.

In sedentary patients, 12-lead electrocardiogram (ECG) is able to depict P-wave abnormalities related to changes in atrial histology,[8] but echocardiography seems to be a better tool to assess adverse atrial remodelling, characterized by an increase in atrial volumes[9] and a decrease in atrial function.[10] Indeed, it was previously published that the three components of atrial function (i.e. reservoir function, conduit and pump function) assessed by speckle tracking echocardiography were altered in sedentary patients with lone PAF without overt cardiovascular disease.[9]

To the best of our knowledge, this was never assessed in the specific population of male veteran athletes with lone AF, in whom the limits between adaptative[11] and mal-adaptative atrial remodelling might be tight. Thus, the aim of this study was to investigate in male endurance veteran athletes the relationship between atrial remodelling assessed by ECG and echocardiography and the occurrence of AF by comparing a group of athletes with lone PAF and a control group of healthy athletes. We hypothesized that atrial function or deformation might better differentiate athletes with and without PAF than ECG and atrial volumes.

Methods

Study population

We retrospectively reviewed a database of athletes referred to the department of sport medicine of our institution from January 2010 to September 2015. From this database, we selected male endurance veteran athletes (age >35 years) with a documented PAF and who had undergone a comprehensive evaluation including ECG, exercise test and echocardiography, all performed in sinus rhythm. PAF was defined by at least one episode of AF documented by an ECG or a Holter 24 h-recording according to ESC guidelines.[12] Athletes were included only if they had performed lifelong competitive endurance sport. Exclusion criteria were any overt cardiovascular disease or a previous catheter ablation of AF. Thirty-six male veteran athletes with PAF were identified; 27 of them were included in the lone PAF group. One athlete was excluded because of AF during echocardiography; 8 athletes were excluded because of the quality of the echocardiography (which did not permit a complete tracking of all the atrial segments in four-chamber view). This first group of veteran athletes was compared with a second group of thirty male endurance veteran athletes without AF, with similar age, training (type of endurance sport, time per week of practice) and cardiovascular risk factors (especially hypertension and body mass index).

The study was approved by the hospital ethics committee and conducted in accordance with the Declaration of Helsinki. All participants gave informed consent.

Study protocol

Medical history and physical examination were collected from the medical file of the athletes. A single experienced cardiologist, blinded as regards to the inclusion group, retrospectively reviewed all 12-lead resting ECG, exercise tests and echocardiographic recordings.

Electrocardiogram

A resting 12-lead ECG was recorded in supine position after 5 min of rest. ECG assessment included heart rate, P-wave amplitude (the highest value observed in lead II, III, or aVF), P-wave morphology (i.e. biphasic aspect) and PR duration (the highest value in lead I or II).

Exercise test

All subjects performed a progressive maximal exercise test on ergocycle (ERG 900, Jaeger, Hochberg, Germany) according to Wasserman recommendations.[13] Exercise was symptom limited or stopped at exhaustion. ECG was continuously monitored (CardioSys, Marquette-Hellige, Freiburg, Germany), blood pressure was measured every 2 min. Maximal predicted power was calculated using the following formula: maximal predicted power (watts) = [[(50.02 - 0.394 × A) × W] - (5.8 × W + 151)]/10.3, A: age in years; W: weight in kilograms.[14]

Transthoracic echocardiography

Despite the retrospective nature of the study, there was a standardization in the performance of the echocardiography. They were performed on a Vivid 7 ultrasound system (GE Healthcare, Horten, Norway), using standard acquisitions in the parasternal, apical, and subcostal views. In order to assess 2D strain, 2D grayscale images were acquired in the four-, three-, and two-chamber views at a frame rate ≥60 frame/s. Off-line analyses were performed on digitally stored images (BT 12-EchoPAC, GE Healthcare, Horten, Norway). Cardiac chamber size, left ventricular ejection fraction (LVEF) were evaluated according to the recommendations of the European Society of Echocardiography.[15] To assess left ventricular (LV) global longitudinal strain (GLS), a line was traced along the LV endocardium's inner border in each of the three apical views, and a region of interest was automatically defined between the endocardial and epicardial borders; GLS was then automatically calculated from the strain in the three apical views. Pulsed-wave Doppler at the tip of mitral valve leaflets was used to measure early (*E*) and late (*A*) diastolic filling velocities, *E*/*A* ratio and *E* deceleration time. Peak early and peak atrial diastolic mitral annular velocities were measured, and then the average values of septal and lateral velocities were used as e' and *a'*. *E*/e' was calculated and used to evaluate LV filling pressure.

Left atrial (LA) and right atrial (RA) size and function: 2D LA and RA acquisitions were obtained from the four-chamber view. LA and RA area were assessed in end systole. LA and RA volumes were measured using the modified Simpson's method[9,16] and were indexed to the body surface area. Maximal LA and RA volume index (MaxLAVi and MaxRAVi) were measured at the ventricular end-systolic frame just before the atrioventricular valve opening. Pre-atrial contraction volumes (PaLAVi and PaRAVi) were obtained from the last frame before atrial contraction. Minimal atrial volume (MinLAVi and MinRAVi) was measured at the end of LV diastole, just before the closure of the atrioventricular valve. LA function parameters were calculated with the following formulas: LA ejection fraction (LAEF) [reservoir function]: (MaxLAVi - MinLAVi)/MaxLAVi ×100; passive emptying fraction (p-LAEF) [conduit function]: (MaxLAVi - PaLAVi)/MaxLAVi ×100; Active LA emptying fraction (a-LAEF) [pump function]: (PaLAVi - MinLAVi)/PaLAVi ×100. The same formulas were used for the RA.[9] Left atrial conduit function was also assessed by the following formula: [LV stroke volume - LA reservoir volume].[17]

To assess atrial longitudinal strain, a line was traced along atria endocardium's inner border in the four-chamber view, and a region of interest was automatically defined between the endocardial and epicardial borders, delineating a region of interest composed by six segments. Manual modifications were made to correct the automatic tracing. LA appendage and pulmonary veins were excluded. The ECG-derived R-wave was taken as the first reference frame. Peak and pre-atrial contraction values of LA longitudinal strain [L-ε-Max (reservoir function) and L-ε-PA (pump function), respectively] were calculated by averaging the values observed in

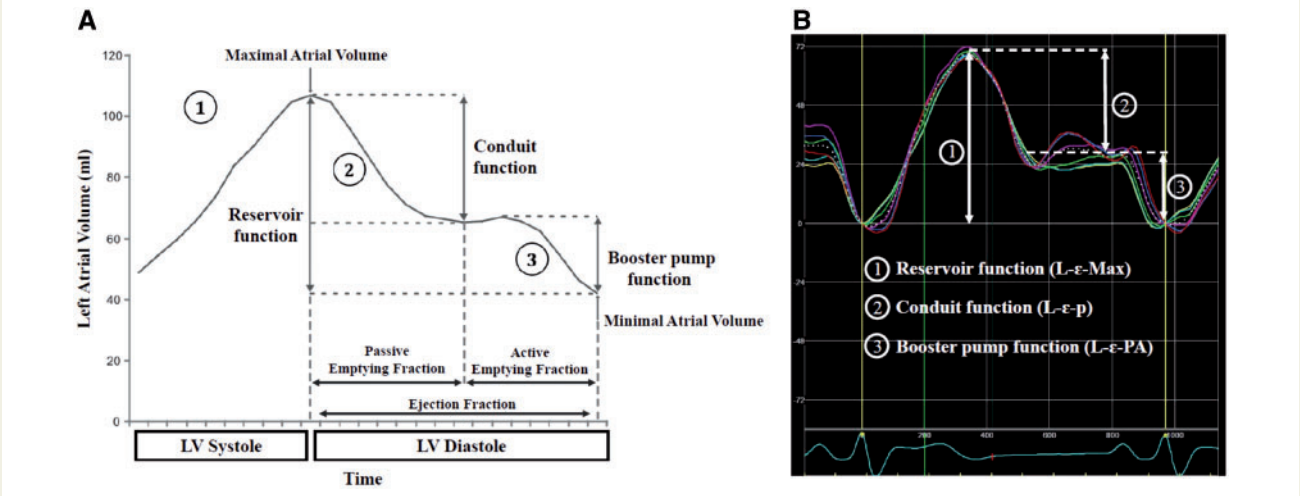

Figure 1 Atrial anatomical and functional parameters according to the cardiac cycle. (*A*) Components of atrial functions assessed by volumes during a cardiac cycle. (*B*) Components of atrial functions assessed by strain during a cardiac cycle.

the LA segments. Difference between peak and pre-atrial contraction values of LA longitudinal strain was calculated [Lε-p = L-ε-Max - L-ε-PA; (conduit function)]. The same analysis was done with the RA strain[9] (*Figure 1*).

Statistical analysis

Gaussian distribution of all continuous variables was confirmed by a Kolmogorov-Smirnov test, and values are reported as mean ± standard deviation. Comparisons between PAF and control groups were performed using unpaired *t*-test for continuous variables and χ^2 test for categorical variables, as appropriate. To study the confounding effect of anti-arrhythmic drugs, a subgroup analysis was performed on the therapy-naïve subjects.

Receiver operating characteristic (ROC) curves were created and areas under curves (AUC) were calculated for the ability of atrial volumes and functions to identify PAF patients. Tests with *P*-values <0.05 were considered to be statistically significant.

Inter- and intraobserver variability of atrial strain and volumes were expressed by intraclass correlation coefficients in three subjects randomly selected from each group ($n = 6$) (*Figure 2*). As each patient underwent only one echocardiography, the reproducibility was assessed from this single echocardiographic examination, the reader blinded as regards to the loop which was used previously, previous interpretations and inclusion group.

Statistical analysis was performed using SPSS (v.20 SPSS Inc.; Chicago, IL, USA). ROC curves analysis and pairwise comparison using the Delong method were performed using MedCalc version 15.2.2 (MedCalc Software, Ostend, Belgium).

Results

As requested by inclusion criteria, there was no difference between the PAF and control group in age (59.9 ± 7.4 vs. 61.8 ± 6.4 years, respectively in the PAF and in the control group, $P = 0.285$), cardiovascular risk factors and sport practice. Indeed, the weekly training duration was not different (6.4 ± 2.6 h/week vs. 6.4 ± 1.5 in the PAF and in the control group respectively, $P = 0.869$) and all practiced endurance sports [running (36.7% vs. 37% in PAF and controls respectively), or cycling/triathlon (63.3 vs. 63%), $P = 0.977$] (*Table 1*).

As expected, all athletes had an exercise capacity above the predicted value. But, PAF athletes achieved a lower maximal power than controls (174.4 ± 28.0% vs. 212.2 ± 47.4% of predicted value; respectively in the PAF and control group, $P = 0.004$). Considering heart rate, no difference was observed considering both resting (57.4 9.0 vs. 62.1 ± 10.2 beat per minute (bpm), respectively, $P = 0.078$) and maximal values (167.8 ± 17.7 vs. 167.3 ± 11.1 bpm, respectively, $P = 0.903$). Thirteen PAF patients were treated with anti-arrhythmic medication [beta-blockers ($n = 6$), flecainide ($n = 6$), amiodarone ($n = 4$)].

ECG characteristics

P-wave characteristics, i.e. duration, amplitude, and percentage of biphasic P-wave, were not different in both groups (99.2 ± 17.5 vs. 93.7 14.5 ms, $P = 0.206$; 0.13 ± 0.06 vs. 0.14 ± 0.04 mV, $P = 0.357$; 44% vs. 33%, $P = 0.125$, respectively in the PAF and control group). There was also no difference in the PR interval duration (192 ± 49.4 vs. 177.2 ± 22.5 ms, $P = 0.147$, respectively) (*Table 1*).

Echocardiographic characteristics

Ventricular parameters

There was no difference between both groups in LV anatomy (LV diameters and volumes, LV thickness), LV systolic function assessed by LVEF (63.5 ± 8.3% vs. 64.1 ± 6.4%, respectively in the PAF and control group, $P = 0.759$) and global longitudinal strain (-21.0 ± 2.3% vs. -20.8 ± 1.8%, $P = 0.724$) (*Table 2*). There was no difference in diastolic function (*E*, *E/A*, *E* deceleration time, e' and *E/e'*) (*Table 2*).

Atrial parameters

Atrial anatomy: left and right atrial minimal and maximal indexed volumes were larger in the PAF group (*Table 3*).

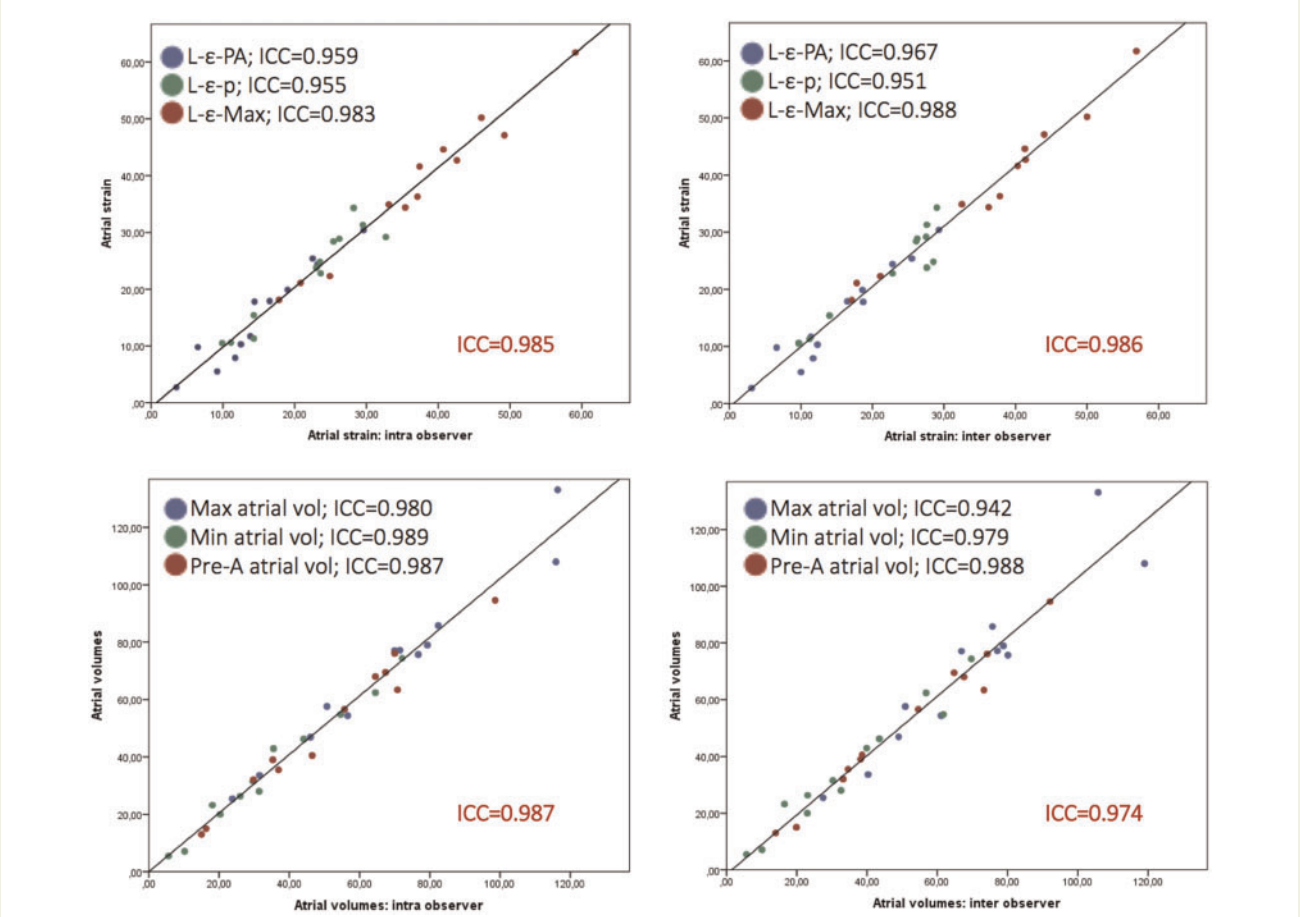

Figure 2 Intra and inter observer variability for atrial strain and atrial volumes. Linear regressions with intra-class correlation coefficients. L-ε-Max, peak atrial longitudinal strain; L-ε-PA, pre-contraction atrial longitudinal strain; L-ε-p, difference between peak and pre-contraction of atrial longitudinal strain; Max atrial volume, Maximal atrial volume; Min atrial volume, minimal atrial volume.

Atrial function: The three functions of both atria assessed by myocardial deformation analysis were significantly worse in the PAF group. Reservoir function (LA: L-ε-Max 29.3 ± 7.9% vs. 49.1 ± 7.8%, $P < 0.0001$; RA: L-ε-Max 36.5 ± 7.0% vs. 50.6 ± 10.2%, $p < 0.0001$, respectively), conduit function (LA: L-ε-p 17.0 ± 6.2% vs. 27.0 ± 6.7%, p 0.0001; RA: L-ε-p 21.6 ± 6.9% vs. 28.5 ± 9.3%, $P = 0.003$, respectively) and booster pump function (LA: L-ε-PA 12.3 ± 6.4% vs. 22.0 ± 5.6%, $P < 0.0001$; RA: L-ε-PA 14.9 ± 5.9% vs. 22.1 ± 5.6%, $p < 0.0001$ respectively) were all lower in the PAF group.

With volumetric methods, similar results were observed in the atrial reservoir function (LA ejection fraction: 59.3 ± 12.9% vs. 67.6 ± 10.0%, $P = 0.010$; RA ejection fraction: 51.9 ± 16.5% vs. 59.6 ± 11.4%, $P = 0.044$, respectively) and pump function (LA active emptying fraction: 23.4 ± 9.6% vs. 29.6 ± 9.0%, $P = 0.014$; RA active emptying fraction: 26.0 ± 11.3% vs. 33.8 ± 10.8%, $P = 0.010$, respectively) which were decreased in the PAF group. No difference was observed in conduit function [LA passive emptying fraction: 36.0 ± 16.2% vs. 37.9 10.1%, $P = 0.591$; (LV stroke volume - LA reservoir volume): 43.6 ± 20.9 vs. 47.1 ± 16.1 mL, $P = 0.479$; RA passive emptying fraction: 24.9 13.6% vs. 25.7 ± 13.9%, $P = 0.822$, respectively] between both groups.

The subgroup analysis with therapy-naïve athletes confirmed the decrease in atrial function assessed by 2D strain in the PAF group (see Supplementary data online, *Table S3 bis*).

ROC curve analysis (*Table 4* and *Figure 3*) showed that atrial function measured by deformation technic was superior to volumetric method to detect PAF athletes [three components of LA function; RA reservoir and conduit function ($P < 0.05$)]. Especially LA and RA L-ε-Max were the best parameters to detect athletes with AF ($P < 0.05$ for all; except vs. L-ε-PA; $P = 0.061$ for LA; $P = 0.128$ for RA), with a low overlap between both groups (*Figure 4*), as compared with atrial volumes.

Discussion

The main finding of this study is that in male endurance veteran athletes a decrease of atrial function is strongly associated with lone PAF. In this population, this parameter seems more robust than the assessment of atrial volumes, as there is lower overlap between athletes with and without PAF according to these functional parameters.

Table 1 Demographic, ECG, and exercise test characteristics of the subjects

Characteristics	PAF group ($n = 27$)	Control group ($n = 30$)	*P*-value
Demographic characteristics			
Age (years)	59.9 ± 7.4	61.8 ± 6.4	0.285
Body mass index (kg/m^2)	24.1 ± 2.9	24.2 ± 2.4	0.912
Hypertension (%)	29.6	33.0	0.976
SBP (mmHg)	123 ± 11	126 ± 10	0.291
DBP (mmHg)	74 ± 8	78 ± 8	0.147
Dyslipidaemia (%)	11.1	3.3	0.253
Smoking (%)	0.0	0.0	—
Diabetes (%)	0.0	0.0	—
Training quantity (h/week)	6.4 ± 2.6	6.4 ± 1.5	0.869
ECG			
P-wave duration (ms)	99.2 ± 17.5	93.7 ± 14.5	0.206
P-wave amplitude (mV)	0.13 ± 0.06	0.14 ± 0.04	0.357
Biphasic P-wave (%)	44.4	30.0	0.125
PR duration (ms)	192.0 ± 49.4	177.2 ± 22.5	0.147
Resting heart rate (bpm)	57.4 ± 9.0	62.1 ± 10.2	0.078
Exercise test			
Maximal power (W)	220.6 ± 34.2	258.2 ± 41.4	0.002
Percentage of maximal predicted power (%)	174.4 ± 28.0	212.2 ± 47.4	0.004
Maximal heart rate (bpm)	167.8 ± 17.7	167.3 ± 11.1	0.903

PAF, paroxysmal atrial fibrillation; bpm, beat per minute; SBP, systolic blood pressure; DBP, diastolic blood pressure; W, watts.

Table 2 Left ventricular echocardiographic parameters

Characteristics	PAF group ($n = 27$)	Control group ($n = 30$)	*P*-value
LVEF (%)	63.5 ± 8.3	64.1 ± 6.4	0.759
Global longitudinal strain (%)	-21.0 ± 2.3	-20.8 ± 1.8	0.724
LV end-diastolic diameter (mm)	52.2 ± 4.8	51.2 ± 4.7	0.435
LV end-diastolic indexed volume (mL/m^2)	71.1 ± 14.5	68.1 ± 15.6	0.448
LV end-systolic indexed volume (mL/m^2)	26.5 ± 8.8	24.3 ± 8.5	0.342
Interventricular septum end-diastolic (mm)	9.7 ± 2.0	9.6 ± 1.4	0.847
Peak *E* mitral velocity (cm/s)	0.67 ± 0.18	0.72 ± 0.14	0.299
Peak *A* mitral velocity (cm/s)	0.54 ± 0.21	0.57 ± 0.14	0.617
E/*A*	1.35 ± 0.48	1.34 ± 0.44	0.920
E-wave deceleration time (ms)	215.5 ± 57.4	201.8 ± 55.0	0.365
e'(cm/s)	13.4 ± 3.6	11.7 ± 2.6	0.060
a' (cm/s)	10.4 ± 1.7	8.3 ± 2.6	0.037
E/e'	5.6 ± 2.0	6.4 ± 1.4	0.079

PAF, paroxysmal atrial fibrillation; LVEF, left ventricular ejection fraction; LV, left ventricle.

Moreover, there was no difference in ECG P-waves parameters between both groups.

Resting ECG is not efficient to depict athletes at risk of AF

In sedentary patients, a link between prolonged P-waves, PR duration, and occurrence of AF was demonstrated,[18,19] nevertheless this was not the case in the present population. Indeed, the P-wave pattern and PR duration of the PAF group were within the normal range.[17] This is consistent with previous findings in healthy athletes, which demonstrated the absence of correlation between P-wave morphology and atrial enlargement.[20] We can suppose that this differences between athletes and sedentary subjects might be related to a different physiopathology of AF.

Table 3 Left and right atrial parameters

Characteristics	PAF group ($n = 27$)	Control group ($n = 30$)	*P*-value
Left and right atrial size			
Left atrial area (cm^2)	22.0 ± 6.2	17.8 ± 3.8	0.004
Max LAVi (mL/m^2)	37.6 ± 14.9	27.5 ± 8.6	0.004
PA LAVi (mL/m^2)	27.9 ± 10.0	23.6 ± 7.1	0.066
Min LAVi (mL/m^2)	16.1 ± 9.9	9.1 ± 4.2	0.002
Right atrial area (cm^2)	21.8 ± 4.2	19.6 ± 3.2	0.032
Max RAVi (mL/m^2)	37.0 ± 10.6	32.0 ± 8.6	0.054
PA RAVi (mL/m^2)	27.9 ± 10.1	23.6 ± 7.1	0.066
Min RAVi (mL/m^2)	18.5 ± 9.1	13.0 ± 5.3	0.008
Left and right atrial reservoir function			
LAEF (%)	59.3 ± 12.9	67.6 ± 10.0	0.010
LA L-ε-Max (%)	29.3 ± 7.9	49.1 ± 7.8	<0.0001
RAEF (%)	51.9 ± 16.5	59.6 ± 11.4	0.044
RA L-ε-Max (%)	36.5 ± 7.0	50.6 ± 10.2	<0.0001
Left and right atrial conduit function			
LA passive emptying fraction (%)	36.0 ± 16.2	37.9 ± 10.1	0.591
LA L-ε-p (%)	17.0 ± 6.2	27.0 ± 6.7	<0.0001
RA passive emptying fraction (%)	24.9 ± 13.6	25.7 ± 13.9	0.822
RA L-ε-p (%)	21.6 ± 6.9	28.5 ± 9.3	0.003
Left and right atrial pump function			
LA active emptying fraction (%)	23.4 ± 9.6	29.6 ± 9.0	0.014
LA L-ε-PA (%)	12.3 ± 6.4	22.0 ± 5.6	<0.0001
RA active emptying fraction (%)	26.0 ± 11.3	33.8 ± 10.8	0.010
RA L-ε-PA (%)	14.9 ± 5.9	22.1 ± 5.6	<0.0001

PAF, paroxysmal atrial fibrillation; Max L(R)AVi, maximal left (right) atrial volume index; PA L(R)AVi, pre-atrial contraction left (right) atrial volume index; Min L(R)AVi, minimal left (right) atrial volume index; L(R)AEF, left (right) atrial ejection fraction; L-ε-Max, peak atrial longitudinal strain; L-ε-PA, pre-contraction atrial longitudinal strain; L-ε-p, difference between peak and pre-contraction of atrial longitudinal strain.

Table 4 ROC curves: accuracy of atrial echocardiographic parameters to identify athletes with AF

		AUC	*P*-value	Cut-off value	Se (%)	Sp (%)
LA anatomy	Max LAVi	0.700 ± 0.072	0.005	32.5	59.3	80.0
	PA LAVi	0.680 ± 0.072	0.002	22.9	77.8	60.0
	Min LAVi	0.731 ± 0.067	0.003	7.6	85.2	50.0
LA function	Ejection fraction	0.688 ± 0.073	0.0015	61.9	59.3	80.0
	Passive emptying fraction	0.575 ± 0.082	0.330	26.7	37	90.0
	Active emptying fraction	0.699 ± 0.073	0.010	28.0	77.8	70.0
	L-ε-Max	0.957 ± 0.023	<0.0001	39	92.6	90.0
	L-ε-p	0.875 ± 0.047	0.0005	20.4	81.5	86.7
	L-ε-PA	0.870 ± 0.050	<0.0001	17.9	88.9	80.0
RA anatomy	Max RAVi	0.665 ± 0.074	0.032	32.9	77.8	60.0
	PA RAVi	0.680 ± 0.072	0.020	21.7	81.5	53.5
	Min RAVi	0.719 ± 0.069	0.005	13.8	70.4	67.0
RA function	Ejection fraction	0.632 ± 0.075	0.0097	61.6	81.5	46.7
	Passive emptying fraction	0.512 ± 0.079	0.360	22.1	51.9	63.3
	Active emptying fraction	0.654 ± 0.073	0.0061	21.3	40.7	86.7
	L-ε-Max	0.901 ± 0.042	<0.0001	41.4	81.5	90.0
	L-ε-p	0.737 ± 0.068	<0.0001	22.7	66.7	80.0
	L-ε-PA	0.806 ± 0.057	<0.0001	16.5	63.0	86.7

Max L(R)AVi, maximal left (right) atrial volume index; PA L(R)AVi, pre-atrial contraction left (right) atrial volume index; Min L(R)AVi, minimal left (right) atrial volume index; L-ε-Max, peak atrial longitudinal strain; L-ε-PA, pre-contraction atrial longitudinal strain; L-ε-p, difference between peak and pre-contraction of atrial longitudinal strain.

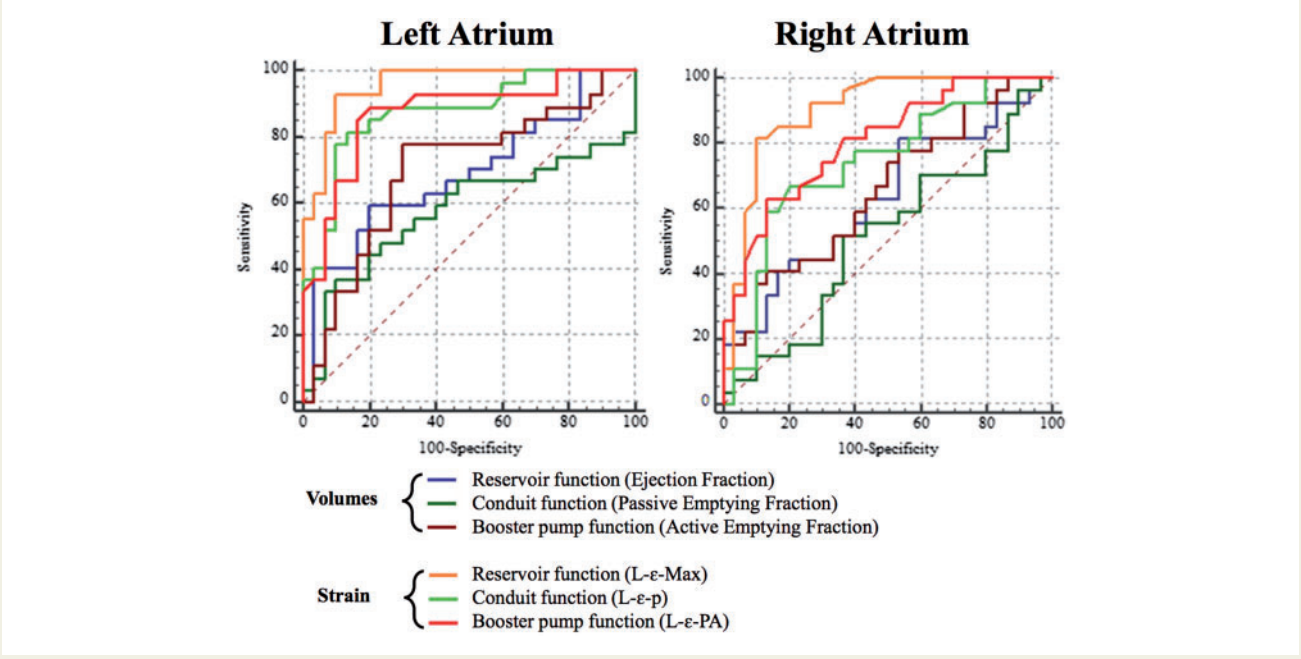

Figure 3 Receiver operating characteristic curves of atrial functions assessed by volumes and strain.

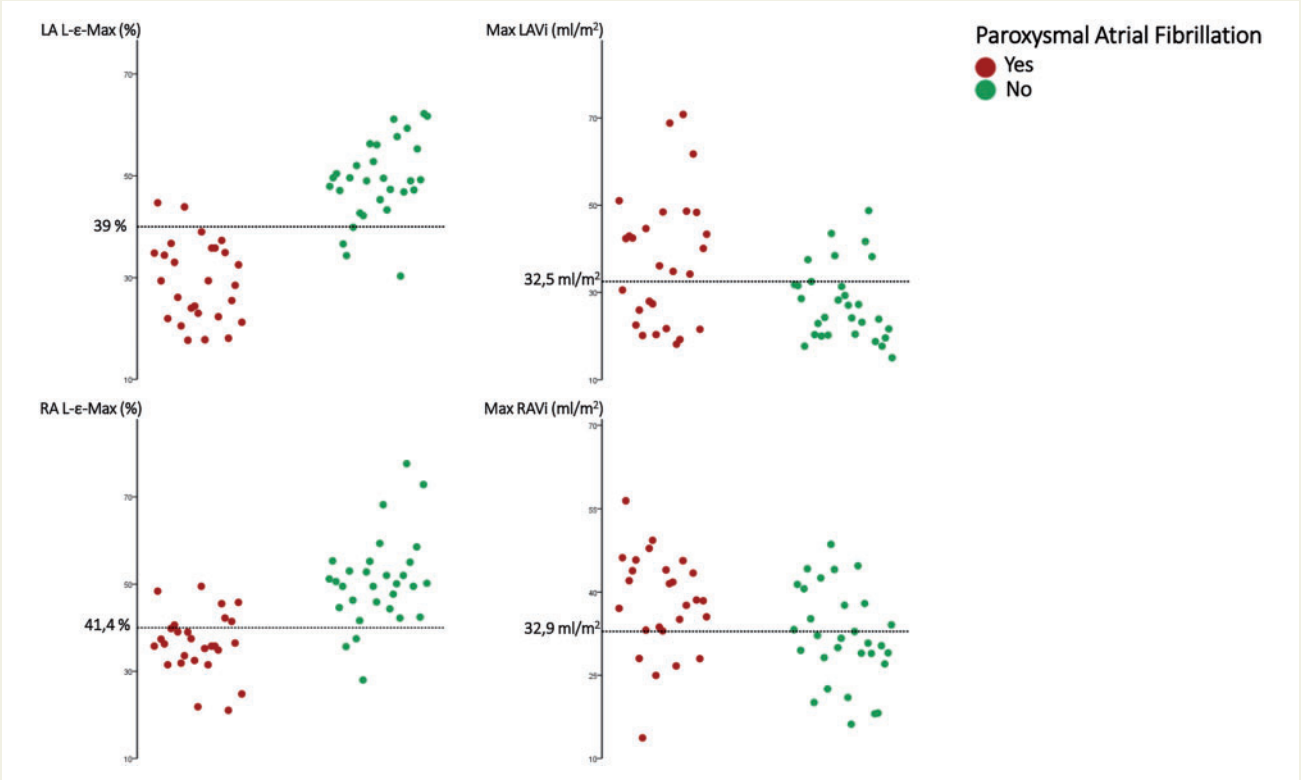

Figure 4 Scatter plots of anatomical and functional parameters. Scatter plots of L(R)A L-ε-Max (peak of left (right) atrial longitudinal strain) and Max L(R)AVi (maximal left (right) atrial volume index). Dotted lines represent the best cut-offs obtained with ROC curves analysis.

Dilation of atria can be a physiological remodelling; decrease of function is always abnormal

The results of this study are consistent with previous publications on PAF in sedentary patients. Indeed, Schaaf *et al.*[9] also reported that patients with PAF had dilated left atria with an altered atrial function. Nevertheless, in their study, LA minimal volume was more accurate than functional parameters to predict AF. Whereas in our study the parameters of atrial function, especially LA and RA L-ε-Max, had a higher sensitivity and specificity to identify PAF as compared with atrial volumes; with a substantial lower overlap between both groups.

The overlap observed in atrial volumes between the two groups might by due to the long-life sport practice of our athletes. Indeed, in athletes atrial enlargement can be regarded as a physiologic adaptation, with values of atrial volumes above the cut-off value proposed in sedentary population[11] leading to a more difficult interpretation of this parameter. In the general population, the cut-off value to consider a LA as dilated is a max LAVi >34 mL/m^2.[16] In our population of healthy control athletes, the mean max LAVi was in the normal range (27.5 mL/m^2), but with 20% of values above this cut-off. On the opposite, there is evidence that the physiological adaptation in athletes is associated with an increased atrial function.[21] In the general population, a LA L-ε-Max <30% is considered as a significant alteration of LA reservoir function.[16] None of our control athletes had a significant alteration of LA L-ε-Max. But, it was altered in PAF athletes (mean LA L-ε-Max: 29.3%), with 56% of values under this cut-off. The higher cut-off value of 39% found to distinguish the two groups studied might be explained by the healthy lifestyle of our athletes, with a low burden of cardiovascular risk factors.

Pathophysiology of atrial dysfunction in endurance athletes

The subgroup analysis demonstrated that atrial dysfunction in PAF athletes was not due to the effect of anti-arrhythmic drugs. We could speculate that the alteration and dilation of atria may be a consequence of AF rather than a predictive factor of AF. Indeed, PAF can result in a stunning of the atria after spontaneous cardioversion.[22] But we might also speculate that atrial dilation and decrease of function might be an early stage of mal-adaptative atrial remodelling, which might be used to predict AF in this population. Indeed, our athletes only suffered from PAF, which is the very early stage of the disease.[23,24]

Our study was not designed to prove the link between endurance exercise and occurrence of AF, indeed both group practiced the same training level. Nevertheless, they had none of the classical risk factor of AF,[5] and it has to be emphasized that diastolic function was not different in in both groups. Because of this absence of usual AF risk factors the mal-adaptative atrial remodelling in the PAF group is likely due to intense exercise. Indeed, our athletes had a lifelong history of competitive endurance sport practice, as demonstrated by their high level of exercise capacity.

We could assume that there is a personal pre-disposition to develop AF in some subject which might be enhanced by intense endurance sport practice, such as previously described in arrhythmogenic right ventricular cardiomyopathy.[25] Intense endurance sport practice might in some predisposed athletes lead to atrial fibrosis, which is the main substrate of AF[26] by creating conduction heterogeneity in the atrial tissue.[27] In animal models it has been demonstrated that strenuous endurance training induces atrial fibrosis and arrhythmias.[28] The decrease in atrial function in our AF athletes might be related to atrial fibrosis as previously demonstrated in patients undergoing mitral valve surgery.[29]

We propose two hypotheses to explain the superiority of longitudinal deformation over volumetric methods to detect PAF athletes. First, the circumferential and longitudinal orientation of atrial fibres;[30] we might speculate that longitudinal fibres are preferentially altered in PAF athletes. Secondly, the lower load dependence of 2D strain method,[31] which could explain the discrepancy observed for conduit function evaluation, which is markedly influenced by LV suction and diastolic function, that was normal in both groups studied.

Clinical implications

Our results suggest that mal-adaptative atrial anatomical and functional remodelling assessed by 2D strain are risk factors of AF in male veteran endurance athletes. A decrease in atrial function, especially in LA and RA L-ε-Max, might be used to select veteran athletes at risk to further develop PAF. It might be considered to advise them to reduce their sport's practice or at least to increase their medical follow-up. Another potential implication could be to orient clinician towards the diagnosis of AF in an athlete with palpitations associated with a decreased atrial function.

Limitations

This study presents some limitations. First, it is a retrospective study; a prospective cohort study on healthy athletes before any occurrence of PAF would be more robust to depict risk factors for PAF. Second, the sample size of PAF athletes was quite small. However, these two limits can be explained by the low reported prevalence of PAF in veteran athletes, which is only 1.3%.[32] Moreover, we chose to focus on a selected group of PAF athletes with no cardiac disease at risk of AF, which is even more difficult to recruit. Third, atrial strain was only measured in four-chamber view because of the poor quality of the atrial images in two-chamber view.

Conclusions

LA and RA dilation in male veteran endurance athletes can be as well an adaptative or a mal-adaptative remodelling, but decrease of atrial function appears to be always a sign of mal-adaptation which can promote AF.

Supplementary data

Supplementary data are available at *European Heart Journal—Cardiovascular Imaging* online.

Conflict of interest: None declared.

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