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CLINICAL RESEARCH

Athlete's heart patterns in elite rugby players: Effects of training specificities

Le cœur d'athlète du joueur de rugby professionnel : effets des spécificités de l'entraînement

Laurent Chevalier[a], Gaëlle Kervio[b,*], Luc Corneloup[a],
Marie-Pierre Vincent[a], Christophe Baudot[a],
Jean-Louis Rebeyrol[c], Francis Merle[a],
Laurent Gencel[a], François Carré[b,d]

[a] Sport Clinic, Bordeaux, France
[b] Inserm, CIC-IT 804, Rennes, France
[c] Biarritz Olympic Basque Country, Biarritz, France
[d] Inserm, UMR1099, Rennes, France

Summary
Background. — Athlete's heart patterns have been widely described. However, to our knowledge, few studies have focused on professional rugby players, who train differently according to their field position.
Aim. — To describe electrocardiographic and echocardiographic patterns observed in elite rugby players according to their field position.
Methods. — One hundred and thirty-five professional rugby players at the end of the competitive season were included.
Results. — According to a modified Pelliccia's classification, 68.1% of electrocardiograms were normal or had minor abnormalities, 27.2% were mildly abnormal and 3.7% were distinctly abnormal. Heart rate was higher in scrum first-row players ($P < 0.05$). Absolute and indexed left ventricular end-diastolic internal diameters (LVIDd; absolute value 59.3 ± 4.7 mm) exceeded

65 mm and 32 mm/m^2 in 13% and 1.5% of players, respectively. Indexed LVIDd values were higher in back players ($P < 0.001$). Left ventricular interventricular septum and posterior wall thicknesses (absolute values 9.4 ± 1.7 mm and 9.2 ± 1.6 mm, respectively) exceeded 13 mm in 3.7% of players. Concentric cardiac hypertrophy was noted in 3.7% of players. Except for one Wolff—Parkinson—White pattern, players with significant ECG or echocardiographic abnormalities showed no cardiovascular event or disease during follow-up.

Conclusion. — Thus, elite rugby players present similar heart patterns to elite athletes in other sports. Major electrocardiographic and echocardiographic abnormalities are quite rare. Eccentric cardiac remodelling is more frequent in back players.

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Résumé

Contexte. — Les caractéristiques du cœur d'athlète ont largement été décrites dans la littérature. À notre connaissance, trop peu d'études se sont cependant intéressées aux joueurs professionnels de rugby, qui s'entraînent différemment selon leur position occupée sur le terrain.

Objectifs. — Étudier les paramètres électrocardiographiques et échocardiographiques de joueurs professionnels de rugby selon leur position sur le terrain.

Méthodes. — Cent trente-cinq joueurs à la fin de la période de compétition (de la saison sportive) ont été inclus.

Résultats. — En accord avec la classification de Pelliccia, 68,1 % des ECG étaient normaux ou présentaient des anomalies mineures, 27,2 % des ECG présentaient des anomalies modérées et 3,7 % des ECG étaient anormaux. Les valeurs de fréquence cardiaque sont plus élevées chez les joueurs de première ligne ($p < 0,05$). Les diamètres télédiastoliques du ventricule gauche (DTDVG), absolus ($59,3 \pm 4,7$ mm) et indexés, sont supérieurs à 65 mm et 32 mm/m^2 chez respectivement 13 % et 1,5 % des joueurs. Les valeurs DTDVG indexées sont plus élevées chez les arrières ($p < 0,001$). Les épaisseurs du septum interventriculaire ($9,4 \pm 1,7$ mm) et de la paroi postérieure du VG ($9,2 \pm 1,6$ mm) sont supérieures à 13 mm chez 3,7 % des joueurs. Une hypertrophie cardiaque concentrique a été observée chez 3,7 % des joueurs. À l'exception d'un Wolff—Parkinson—White, aucun évènement ou pathologie cardiovasculaire n'a été observé chez les joueurs présentant des anomalies majeures sur l'électrocardiogramme et/ou sur l'échocardiographie.

Conclusion. — Les joueurs professionnels de rugby présentent les mêmes caractéristiques du cœur d'athlète que les autres sportifs. Les évènements cardiaques majeurs sur l'électrocardiogramme et sur l'échocardiographie sont assez rares. Le remodelage cardiaque excentrique est plus fréquent chez les arrières.

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Background

A high level of intensive physical training can be associated with electrical, structural and functional myocardial adaptations — the so-called athlete's heart [1,2]. It is well known that athlete's heart patterns depend on age, sex, body size and race [3—8]. However, the sport's specificities are accepted as the main determinant of cardiac adaptations in athletes [2,4,8].

Rugby is described as a sport with moderate isometric components and moderate or high dynamic components [9,10]. However, these components vary in accordance with the player's position on the field. The main static component concerns scrum first-row players, equal dynamic and static stresses are imposed on scrum second- and third-row players and the higher dynamic component concerns back players [11]. To the best of our knowledge, only one purely descriptive study specifically concerning a small population of rugby players has been published [12]. Thus, the aims of this study were to describe electrocardiography and echocardiography patterns in high-level rugby players and to look for specific cardiac adaptations in accordance with the player's position on the field.

Methods

Population

This prospective study consecutively included 135 healthy rugby players (116 Caucasians, 10 Polynesians and nine Africans) from the French first league from 1998 to 2008. All players were selected from only two professional clubs. Players were distributed according to their field position: group 1, scrum first-row ($n = 34$); group 2, scrum second- and third-row ($n = 46$); and group 3, backs ($n = 55$). Each player had a cardiovascular examination performed in a blinded manner by the same cardiologist (L.C.) between July and

August, which included personal and family history, physical examination, twelve-lead electrocardiogram (ECG) and echocardiography at rest.

All experimental procedures were performed in accordance with the ethical standards of the Helsinki Declaration and were approved by the university's human subjects review board. All players gave written informed consent to the investigators before participating.

Data recording

Electrocardiogram

Standard twelve-lead ECGs were recorded at rest (AT-5, Schiller, Baar, Switzerland, 1998—2003; Mac 1200, Marquette/GE, Milwaukee, WI, USA, 2003—2010). All ECGs were interpreted by the same experienced observer (G.K.) who was blinded to the players' clinical data. Heart rate, QRS duration, and QT and PR intervals were measured with a tracer table (Graph'Image, Gennevilliers, France).

ECG data were classified as normal/minor, mild or major abnormalities according to a modified Pelliccia's classification [7] and current recommendations [13].

Echocardiography

Two-dimensional echocardiograms were registered with three successive ultrasound systems from the same company (Vivid 5, Vivid 3 and Vivid I systems; General Electric, Milwaukee, WI, USA). Standard views of the heart were obtained with a 3.5 MHz frequency phased-array transducer and analysed according to the protocol recommended by the American Society of Echocardiography [14]. M-mode measurements were obtained from the two-dimensional parasternal short axis; they concerned left atrial diameter (LAd), aortic diameter (Aod), left ventricular (LV) end-diastolic and end-systolic internal diameters (LVIDd and LVIDs, respectively) and left ventricular end-diastolic interventricular septum and posterior wall thicknesses (IVSd and PWd, respectively). The Devereux formula was used for calculation of left ventricular mass (LVM). As recommended [14], all these absolute values were indexed to body surface area (BSA) and LVM was also indexed to height$^{2.7}$. Left ventricular hypertrophy (LVH) was defined as LVM/BSA > 116 g/m^2 [14] or LVM/height$^{2.7}$ > 49 g/m$^{2.7}$ (mildly abnormal 49—55; moderately abnormal 56—63; severely abnormal ≥ 64) [14]. LVH index was calculated with the IVSd + PWd/LVIDd ratio. Concentric and eccentric myocardial remodelling were defined as values higher than 0.44 and below 0.30, respectively, as proposed in sedentary people [14].

Two-dimensional echocardiograms were recorded from the apical four-chamber view. End-systolic LA and right atrial (RA) areas were measured in their largest area to avoid foreshortening, excluding the appendages and pulmonary vein confluence [14]. Early (E) and atrial (A) peak velocities were measured with pulsed Doppler and the E/A ratio was then calculated [14].

All scans were reviewed by a cardiologist (L.C.) blinded to the athletes' identities.

A preliminary intraobserver analysis carried out on a sample of 30 subjects showed a variation of 2.8% for M-mode and two-dimensional variables and of 2.0% for the Doppler criteria.

Clinical findings and follow-up in athletes with abnormalities

Athletes showing a distinctly abnormal ECG pattern and/or increased cardiac dimensions above the normal limits (defined arbitrarily here as LVIDd > 65 mm and/or left ventricular wall thickness > 13 mm) underwent recommended further investigations (48-hour Holter ECG, maximal exercise test and cardiac magnetic resonance imaging [MRI] with gadolinium intravenous administration) to exclude a pathological cardiac condition [5,7,15].

Data analysis

Continuous data are expressed as means ± standard deviations. The training specificity's effect was analysed with a comparison between the three groups using a Kruskall—Wallis test, followed by a Mann—Whitney test when appropriate. A Chi^2 test was used for qualitative data comparisons. The significance threshold was always set at 0.05.

Results

Demographic data

All players were of similar age (25.8 ± 4.0 years for group 1; 25.6 ± 4.4 years for group 2; and 24.1 ± 3.7 years for group 3). All were asymptomatic with no personal or family history of cardiovascular disease. Concerning height, players were taller in group 2 (194.3 ± 4.7 cm) than in group 1 (182.3 ± 4.5 cm) and group 3 (181.9 ± 5.9 cm) (P < 0.001); no difference was noted between groups 1 and 3. Concerning weight, players were lighter in group 3 (87.1 ± 8.8 kg) compared with group 1 (113.0 ± 12.0 kg) and group 2 (109.0 ± 10.1 kg) (P < 0.001); no difference was observed between groups 1 and 2. The BSA range was 2.2—2.7 m^2, with the smallest BSA in group 3 (P < 0.001).

Electrocardiographic patterns

Global population

Most ECGs were normal (16%) or had minor abnormalities (52%). Minor abnormalities constituted mostly of bradycardia (61%) and incomplete right bundle branch block (27%) (Table 1). The ECG mild abnormalities (27%) were mainly flat, particularly tall or minimally inverted T waves (13%). Biphasic T waves in lead V2 were noted in one player. LA enlargement was noted in 12% of players. Distinctly abnormal ECGs were seen in five (4%) players: one Caucasian player with Wolff—Parkinson—White pattern; one Polynesian player and one African player with right QRS axis deviation; and one Polynesian player and one African player with deeply inverted T waves.

Table 1 Electrocardiographic variables in all rugby players and in each group (group 1, scrum first-row players; group 2, scrum second- and third-row players; group 3, back players).

Variables	Total ($n = 135$)	Group 1 ($n = 34$)	Group 2 ($n = 46$)	Group 3 ($n = 55$)
Quantitative variables				
Heart rate (beats/min)	58.6 ± 11.5	$61.1 \pm 11.8^*$	55.4 ± 10.0	59.9 ± 12.1
PR duration (ms)	171.4 ± 23.4	174.8 ± 19.6	173.5 ± 22.0	167.6 ± 26.4
QRS duration (ms)	89.5 ± 9.9	89.1 ± 9.4	90.9 ± 10.5	88.7 ± 9.9
QT duration (ms)	391.8 ± 30.6	386.9 ± 33.1	399.9 ± 26.6	388.0 ± 31.3
QTc duration (ms)	383.4 ± 26.5	385.8 ± 22.1	381.0 ± 25.2	383.9 ± 30.1
Qualitative variables				
Normal or minor alterations	92 (68.1)	22 (64.7)	34 (73.9)	36 (65.4)
Normal	22 (16.3)	8 (23.5)	6 (13.0)	8 (14.5)
Sinus bradycardia (< 60 beats/min)	82 (60.7)	16 (47.0)	32 (69.5)	34 (61.8)
PR interval (> 0.20 s)	13 (9.6)	3 (8.8)	6 (13.0)	4 (7.3)
R or S wave (25—29 mm) in any lead	17 (12.6)	5 (14.7)	5 (10.8)	7 (12.7)
Early repolarization in ≥ 2 leads	15 (11.1)	2 (5.9)	4 (8.7)	9 (16.4)
IRBBB	37 (27.4)	8 (23.5)	14 (30.4)	15 (27.3)
Mildly abnormal ECG	37 (27.4)	11 (32.3)	10 (21.7)	16 (29.1)
R or S wave (30—34 mm) in any lead	1 (0.7)	0 (0)	1 (2.2)	0 (0)
Q waves (2—3 mm) in ≥ 2 leads	2 (1.5)	0 (0)	0 (0)	2 (3.6)
Mild repolarization peculiarity in ≥ 2 leads	17 (12.6)	5 (14.7)	3 (6.5)	9 (16.4)
Abnormal R wave progression (V1 to V3)	10 (7.4)	5 (14.7)	0 (0)	5 (9.1)
CRBBB	3 (2.2)	0 (0)	2 (4.3)	1 (1.8)
LA enlargement	16 (11.8)	5 (14.7)	8 (17.4)	3 (5.4)
PR interval (≤ 0.12 s)	2 (1.5)	0 (0)	1 (2.2)	1 (1.8)
Distinctly abnormal ECG	5 (3.7)	1 (2.9)	1 (2.2)	3 (5.4)
Inverted T wave > 2 mm in ≥ 2 leads	2 (1.5)	1 (2.9)	0 (0)	1 (1.8)
QRS axis deviation ($\leq -30°$ or $\geq 110°$)	2 (1.5)	0 (0)	1 (2.2)	1 (1.8)
WPW pattern	1 (0.7)	0 (0)	0 (0)	1 (1.8)

Data are number (%) for categorical variables and mean $\pm$ standard deviation for continuous variables. BBB: bundle branch block; ECG: electrocardiogram; IRBBB: incomplete right BBB (RSR' in lead V1 < 0.12 s); CRBBB: complete right BBB: CLBBB: complete left BBB; LA: left atrial; WPW: Wolff—Parkinson—White. Early repolarization is defined as ST-segment elevation > 2 mm in ≥ 2 leads; mild repolarization peculiarity is defined as flat, particularly tall, minimally inverted T waves or J-point upward/domed convexity ST-segment elevation with inverted or biphasic T waves in ≥ 2 leads. Note that no rugby player had right atrial hypertrophy (mildly abnormal ECG) and R or S waves (≥ 35 mm) in any lead, Q waves (≥ 4 mm) in ≥ 2 leads, LBBB, ST-segment depression (> 1 mm), premature ventricular beats and long QT duration (distinctly abnormal ECG).
* $P < 0.05$, comparison between groups 1 and 2.

Intergroup comparison

Resting heart rate was higher in group 1 versus group 2 ($P < 0.05$) (Table 2). No difference was noted concerning distinctly abnormal ECGs.

Echocardiographic patterns

Mild mitral, tricuspid and aortic insufficiencies were noted in 12 (9%), three (2%) and two (1.5%) players, respectively.

Global population

The LVIDd, which exceeded 65 mm in 18 (13.5%) players, was > 32 mm/m^2 in two (1.5%) players; both were Caucasian back players without increased wall thickness and with ECG minor alterations. A wall thickness > 13 mm was observed in five (3.7%) players (IVSd, $n = 2$; PWd, $n = 1$; IVSd; PWd, $n = 2$). Five (3.7%) players presented a LVH index > 0.44 and 67 (49.6%) players presented a LVH index < 0.30. LVM exceeded 116 g/m^2 in 21 (15.5%) players and LVM/height$^{2.7}$ exceeded 49 g/m$^{2.7}$ in 28 (20.5%) players (mildly abnormal $n = 22$, 16%;

moderately abnormal $n = 4$, 3%; severely abnormal $n = 2$, 1.5%). Among them, a LVM > 116 g/m^2 was associated with a LVH index < 0.30 or > 0.44 in five players and one player, respectively. Twelve (9%) players had a LAd > 45 mm. No player had a LAd/BSA > 22 mm/m^2. Concerning the association between ECG and echocardiographic abnormalities, two players with a LAd > 45 mm had an ECG LA enlargement. In both cases, familial and personal history and physical examination were strictly normal. Lastly, one player with inverted T waves had an IVSd thickness of 12 mm with a LVIDd of 59 mm. His follow-up is described below.

Intergroup comparison

Group 2 showed the highest absolute LVIDd and LVIDs values ($P < 0.05$) (Table 2). Five (15%) players in group 1, 11 (24%) players in group 2 and two (3%) players in group 3 had a LVIDd > 65 mm. The highest indexed LVIDd was noted in group 3 ($P < 0.001$); among this group, two players had a value > 32 mm/m^2. The thinnest absolute IVSd and PWd values were noted in group 3 ($P < 0.05$). No difference was noted

Table 2 Absolute and indexed echocardiographic variables in all rugby players and in each group (group 1, scrum first-row players; group 2, scrum second- and third-row players; group 3, back players).

Variables	Total (n = 135)		Group 1 (n = 34)		Group 2 (n = 46)		Group 3 (n = 55)	
	Absolute	Indexed to BSA ($/m^2$)	Absolute	Indexed to BSA ($/m^2$)	Absolute	Indexed to BSA ($/m^2$)	Absolute	Indexed to BSA ($/m^2$)
LVIDd (mm)	59.3±4.7 (51.0–72.0)	25.9±2.2 (20.7–32.3)	59.4±4.6[*] (51.0–70.0)	24.5±1.8 (20.7–28.0)	61.7±4.8[c] (51.0–72.0)	25.3±1.7[c] (21.2–29.4)	57.3±3.8[d] (51.0–68.5)	27.2±2.1[f] (23.1-32.3)
LVIDs (mm)	37.5±4.3 (24.0–49.0)	16.4±2.0 (10.1–21.5)	37.8±4.4 (25.0–46.0)	15.6±1.9 (10.1–19.3)	38.7±4.6[b] (24.0–49.0)	15.9±1.8[c] (10.4–19.4)	36.5±3.7 (27.9–45.5)	17.3±1.8[f] (13.0-21.5)
IVSd (mm)	9.4±1.7 (5.0–15.5)	4.1±0.7 (2.3–6.6)	9.8±1.4 (7.0–13.1)	4.1±0.5 (2.9–5.2)	9.7±1.7[a] (6.0–13.9)	3.9±0.8 (2.3–5.8)	9.0±1.7[e] (5.0–15.5)	4.3±0.7 (2.4-6.6)
PWd (mm)	9.2±1.6 (6.0–15.3)	4.0±0.6 (2.8–6.4)	9.4±1.5 (7.5–13.8)	3.9±0.6 (2.9–5.2)	9.7±1.7[b] (7.2–15.3)	4.0±0.7 (2.9–6.4)	8.7±1.4[d] (6.0–12.0)	4.1±0.6 (2.8-5.7)
LVH index	0.31±0.06 (0.21–0.55)		0.32±0.06 (0.23–0.45)		0.32±0.06 (0.22–0.55)		0.30±0.05 (0.21–0.45)	
LVM (g)	224.7±54.4 (108.1–378.7)	97.1±19.3 (51.5–161.5)	233.4±48.7 (157.4–370.8)	95.9±16.4 (67.0–134.6)	250.0±52.1[c] (146.0–363.9)	102.4±20.5 (55.4–152.4)	198.1±48.1[f] (108.1–378.7)	93.3±19.4 (51.5-161.5)
LVM ($g/m^{2.7}$)		41.7±9.0 (22.1–75.2)		46.5±8.6[*] (29.9–63.2)		41.6±8.6 (24.0–66.2)		39.1±8.6[f] (22.1-75.2)
Aod (mm)	32.7±3.1 (25.0–38.8)	14.3±1.4 (11.1–17.7)	33.8±3.2 (25.0–38.8)	13.9±1.3 (11.5–17.2)	33.6±3.2[c] (26.7–38.5)	13.8±1.4[c] (11.1–16.8)	31.2±2.4[f] (26.5–35.8)	14.8±1.3[e] (12.8-17.7)
LAd (mm)	39.8±4.0 (33.3–48.9)	17.3±1.8 (13.3–21.9)	42.2±2.4 (37.5–46.6)	17.4±1.4 (15.0–20.4)	41.1±3.7[c] (33.3–48.9)	16.9±1.6 (13.3–20.6)	37.2±3.7[f] (28.0–46.5)	17.7±2.1 (13.3-21.9)
LA area (cm^2)	19.7±3.2 (12.6–30.2)	8.6±1.3 (5.8–11.7)	19.9±2.6 (15.2–24.6)	8.2±1.2 (6.0–10.4)	21.1±3.3[c] (14.2–30.2)	8.6±1.3 (5.9–11.2)	18.3±3.1[d] (12.5–24.2)	8.7±1.5 (5.8-11.7)
RA area (cm^2)	19.1±4.4 (8.0–28.3)	8.3±1.9 (4.1–12.4)	19.8±4.1 (13.3–28.3)	8.2±1.8 (5.3–11.1)	20.1±4.4[a] (12.4–26.1)	8.2±1.8 (5.0–11.7)	17.9±4.4 (8.0–25.8)	8.5±2.0 (4.1-12.4)
LVEF (%)	65.3±6.4 (49.0–78.0)		64.9±6.4 (49.0–78.0)		65.7±6.6 (52.0–78.0)		65.2±6.1 (52.0–78.0)	
E wave (m/s)	0.86±0.2 (0.52–1.35)		0.90±0.2 (0.55–1.23)		0.84±0.2 (0.52–1.34)		0.86±0.2 (0.55–1.35)	
A wave (m/s)	0.48±0.1 (0.22–0.97)		0.50±0.1 (0.24–0.71)		0.48±0.1 (0.25–0.97)		0.48±0.1 (0.22–0.89)	
E/A ratio	1.9±0.5 (0.7–3.7)		1.8±0.4 (1.0–2.8)		1.9±0.5 (0.7–3.7)		1.9±0.5 (1.0–3.61)	

Data are mean ± standard deviation (min-max). Aod: end-diastolic aortic diameter; IVSd: end-diastolic interventricular septum thickness; LAd: end-systolic left atrial diameter; LA: left atrial; LVEF: left ventricular ejection fraction; LVH index: left ventricular hypertrophy index (2 × PWd/LVIDd); LVIDd: left ventricular end-diastolic internal diameter; LVIDs: left ventricular end-systolic internal diameter; LVM: left ventricular mass; PWd: end-diastolic posterior wall thickness; RA: right atrial.

[*] $P < 0.05$, comparison between groups 1 and 2.

[a] $P < 0.05$, comparison between groups 2 and 3.

[b] $P < 0.01$, comparison between groups 2 and 3.

[c] $P < 0.001$, comparison between groups 2 and 3.

[d] $P < 0.05$, comparison between groups 1 and 3.

[e] $P < 0.01$, comparison between groups 1 and 3.

[f] $P < 0.001$, comparison between groups 1 and 3.

after BSA indexation. The absolute LVM value was lower in group 3 ($P < 0.001$). No difference was noted between the two groups after indexation to BSA, but after indexation to height$^{2.7}$, LVM was higher in group 1 in comparison with groups 2 and 3 ($P < 0.05$). No difference was noted between groups 2 and 3. Aod, LAd and LA area were smaller in group 3 ($P < 0.01$). LVH index, left ventricular ejection fraction (LVEF), E and A waves and E/A ratio were not different between groups.

Clinical findings and follow-up in athletes with abnormalities

Twenty-eight (21%) players presented at least one cardiovascular abnormality; they all had further cardiovascular investigations. Concerning ECG alterations, the player with the Wolff—Parkinson—White pattern had successful ablation therapy and returned to competition. Players with right QRS axis deviation ($n = 2$) or deeply inverted T waves ($n = 2$) were asymptomatic, with normal familial and personal histories, and presented no echocardiographic or other cardiovascular abnormality. The players with inverted T waves were Polynesian and African, and their echocardiography variables were in accordance with physiological left ventricular remodelling. Resting inverted T waves (leads V2 and V3) disappeared during maximal exercise testing in both players and a 48-hour Holter recording showed no arrhythmias. Cardiac MRI was performed only in one player (2008) and was normal. The second player (2000) could not have cardiac MRI. During follow-up (4.2 ± 2.6 years), none presented cardiovascular symptoms or events. Concerning the five players (two Caucasians, two Polynesians, one African) with a wall thickness > 13 mm, all were asymptomatic, none presented a distinctly abnormal ECG and all presented a LVIDd > 45 mm with normal diastolic and systolic functions. All had a 48-hour Holter recording and a maximal exercise test without any abnormality. Among the five players, three (1999—2000) could not have cardiac MRI and two players (2006 for both) had cardiac MRI. In both cases, the examination was strictly normal and confirmed the quantitative echocardiographic data. During follow-up (5.0 ± 2.0 years), none presented cardiovascular symptoms or events. Lastly, the 18 players with a LVIDd > 65 mm had a normal resting LVEF and only two Caucasians had a BSA indexed value > 32 mm/m^2. None presented a distinctly abnormal ECG and all performed a normal exercise test with a peak oxygen uptake adapted to their training level. The two players with LVIDd/BSA > 32 mm/m^2 had normal exercise echocardiography during follow-up. None of the 18 players presented cardiovascular symptoms or events during follow-up (5.3 ± 2.1 years).

Discussion

This prospective study performed in a large population of professional rugby players describes the ECG and echocardiographic traits observed in these athletes, proposes echocardiographic standards indexed to BSA in this population — characterized by the large size of its athletes — and, for the first time, compares these variables in accordance with the player's position on the field.

Electrocardiographic patterns

In accordance with the slightly modified Pelliccia's classification used in this study [7], resting ECG was considered as strictly normal in only 16% of our population. Most players presented minor alterations and the prevalence of mild abnormalities observed is similar to that reported in American football players and lower than that observed in endurance athletes [1,7,16]. We observed less than 4% of distinctly abnormal ECGs, which is concordant with previous studies [5,9,17]. All five players with distinctly abnormal ECGs were asymptomatic and had a normal cardiovascular examination. The player with Wolff—Parkinson—White pattern had successful ablation and returned to playing rugby [9,10]. As recommended, all have an annual cardiovascular follow-up [18]. In American football players, marked ECG differences in relation to the field player's position have been reported [16]. We observed only a higher resting heart rate in the scrum first-row players, which may be due to the high level of static activity performed in this group [5,7].

Echocardiographic patterns

All indexed values obtained for aortic diameter are in accordance with the nomogram previously proposed in other athletes [19]. Absolute LAd mean value is in accordance with previous research, but is lower than that reported in professional cyclists [4]. The prevalence of LAd > 45 mm is similar to that noted in soccer players [20]. No rugby player exceeded the proposed upper normal limit of 22 mm/m^2 [14]. Our results confirm the weak correlation between LAd and LVIDd and the preservation of the LA/RA ratio [18,21]. LVIDd and LVIDs absolute values observed are similar to those reported in endurance athletes [21]. Absolute LVIDd values are larger than in soccer or American football players [1,16,18,20]. However, only two players exceeded the proposed upper indexed-to-BSA normal limit of 32 mm/m^2 [14]. Left ventricular walls in our rugby players appear thinner than those reported in endurance and power-trained athletes, but similar to those described in other team sports [1,2,4,6,12,18,20—22]. Less than 4% of players presented a left ventricular wall > 13 mm and only 1.5% presented a left ventricular wall > 15 mm. These results are in accordance with the upper limits proposed in Caucasian athletes [5,15,22]. All indexed values appear normal [5,15,22]. Concerning the LVM, the mean absolute and indexed values observed can be considered as normal [2,5,6,8]. The upper normal limit of LVM proposed in sedentary people was exceeded in 15% of players [14]. Our results are similar to those published [2,5,21]. However, data concerning both left ventricular wall thickness and LVM are different from those reported in American football players [1,16]. The ethnic influence that is reported in left ventricular remodelling in athletes may partially explain this discrepancy [3]. We observed 49% and 4% of eccentric and concentric myocardial remodelling, respectively. These data are quite similar to those reported [4,9,15]. The LVEF, E and A waves and E/A were normal in all players. The mean E and A values are similar to those previously reported [1]. The intergroup comparison showed that most differences were linked to BSA. However, back players presented higher LAd/BSA and

LVIDd/BSA ratios than scrum players but similar left ventricular wall thickness/BSA, LVM/BSA and LVH indices.

Study limitations

Because of several echocardiographic limitations, we chose not to assess the right ventricle [2,8]. We were unable to index echocardiographic data to fat-free mass as has been sometimes recommended [6]. Athlete's heart in relation to ethnicity has been previously reported [7]; however, because of the small samples of Polynesian ($n = 10$) and African ($n = 9$) players, we could not discuss the effect of ethnicity on our results.

Conclusion

In elite rugby players, we conclude that ECG and echocardiographic major abnormalities seem scarce and in no case were complicated by adverse developments during the follow-up period. Echocardiographic data must be normalized for individual BSA because of the large size of rugby players. The player's position on the field seems not to be related to ECG abnormalities, whereas a larger indexed left ventricular dilatation was observed in back players.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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