Original article

Electro-echocardiographic correlation in high-performance athletes

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Summary

Background: Functional and structural cardiac adaptations are generated by sustained physical training. The objective of our investigation was to evaluate the association in electrocardiographic and echocardiographic findings in a population of high-performance athletes.
Material and method: 30 male athletes (10 water polo players, 10 triathlonists and 10 swimmers), ages 18 to 40 years old, training 20 to 30 hours per week for at least one year, were evaluated. Clinical, electrocardiographic (ECG) and echocardiographic examination was performed on each of them at Instituto Vozzi.
Results: Echocardiographic results showed that the mean septal thickness, the mass index of the left ventricle (LV), the anteroposterior diameter and the area of the left atrium (LA), the area of the right atrium (RA) and the base of the right ventricle (RV) were found above normal values for the general population. None of the athletes ECGs presented LA, RA or RV enlargement. Nine of 30 (30%) presented signs of LV enlargement. After adjusting for age, weight, height, body surface area, and sport performed, LV diastolic diameter (LVDD) indexed to body surface area (BSA) was higher in athletes with LV enlargement on ECG (adjusted mean 28.94 ± 0.56 mm; 95% CI = 27.78-30.10) vs without (27.67 ± 0.36 mm; 95% CI = 26.93-28.41). More triathlonists presented LV enlargement signs on the ECG compared to the other groups.
Conclusions: Certain echocardiographic parameters in our population of athletes are above normal values for the general population. There was no relationship comparing electrocardiographic and echocardiographic signs of LA, RA and RV enlargement. An association was found between ECGs LV enlargement and increased LVDD indexed to BSA on the echocardiograms. LV enlargement on the ECGs was more frequent in the triathlon group.

Key words:
Sports. Electrocardiography. Echocardiography. Athletes. Hypertrophy.

Correlación electro-ecocardiográfica en deportistas de alto rendimiento

Resumen

Introducción: El entrenamiento físico sostenido genera adaptaciones cardíacas estructurales y funcionales. El objetivo de nuestro trabajo fue evaluar la correlación entre los hallazgos electro-ecocardiográficos en una población de deportistas de alto rendimiento.
Material y método: Se evaluaron 30 deportistas varones (10 waterpolistas, 10 triatletas y 10 nadadores), entre 18 y 40 años, con 20 a 30 horas semanales de entrenamiento por al menos un año. Se efectuó evaluación clínica, electrocardiográfica y ecocardiográfica a cada uno de ellos en el Instituto Vozzi.
Resultados: En la evaluación ecocardiográfica, se observó que la media del espesor septal, el índice de masa del ventrículo izquierdo (VI), el diámetro anteroposterior y el área de la aurícula izquierda (AI), el área de la aurícula derecha (AD) y la base del ventrículo derecho (VD) se hallaron por encima de los valores normales para la población general. En los ECG, ninguno de los deportistas presentó crecimiemto de AI, AD o VD. Nueve de los 30 (30%) presentaron signos de hipertrofia del VI. Luego de ajustar por edad, peso, talla, superficie corporal y deporte realizado, el diámetro diastólico del VI (DdVI) indexado a la superficie corporal (SC) fue mayor en los deportistas con hipertrofia del VI en el electrocardiograma (ECG) (media ajustada 28,94±0,56 mm; IC95%= 27,78-30,10) vs sin hipertrofia (27,67±0,36 mm; IC95%= 26,93-28,41). Los triatletas presentaron con mayor frecuencia hipertrofia del VI en el ECG respecto de los otros grupos.
Conclusiones: Ciertos parámetros ecocardiográficos en nuestra población de deportistas se hallan por encima de los valores normales para la población general. No se halló relación entre los signos electrocardiográficos y ecocardiográficos de crecimiento de la AI, la AD e hipertrofia del VD. Se halló relación entre hipertrofia del VI en el ECG y aumento del diámetro diastólico del VI indexado en el ecocardiograma. La hipertrofia del VI en el ECG fue más frecuente en el grupo de triatletas.

Palabras clave:
Deportes. Electrocardiografía. Ecocardiografía. Deportistas. Hipertrofia.

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Introduction

Studies conducted in the 1950s and 1960s, with angiographic correlation and by autopsy, established the limitations of the electrocardiogram (ECG) to detect ventricular hypertrophy and observed that the greatest accuracy in diagnosis was achieved in persons with hypertension and in patients with left-sided valvular heart disease, particularly when they exhibited moderate or acute hypertrophy[1]. Although new criteria were progressively developed over time, this did not improve the accuracy of the method.

It cannot be ignored that most of the electrocardiographic criteria for the detection of left ventricular hypertrophy (LVH) were validated in populations with a high prevalence of cardiovascular diseases (chronic kidney failures on haemodialysis[2], chronic arterial hypertension[3]). The obvious consequence is the low performance of these criteria when applied to populations with a low prevalence of these diseases. Athletes are one such population, in whom the interpretation of the ECGs is normally based on the traditional criteria used for the non-athletic population[4]. The validation of the hypertrophy criteria was made in principle by correlation with the anatomical piece, later through different imaging methods. In this respect, little information is available regarding the electro-echocardiographic correlation of the cardiac modifications that occur as an adaptive phenomenon to sustained physical training.

The aim of our study was to determine whether or not there is a relationship between the electrocardiographic and echocardiographic signs of growth of the cardiac chambers in high-performance athletes, understanding such an athlete to be one who systematically and regularly trains, with a high physical demand, for the purpose of being successful in a competition[5].

Material and method

Selection of subjects

We conducted a prospective, observational study between June and September 2018 on male athletes aged between 18 to 40 years, selected from high-performance centres of the city of Rosario and who were training from 20 to 30 hours per week in three types of sport: triathlon (T), swimming (S) and water polo (W) with a minimum training time of one year. The inclusion criteria also established that they must be free from cardiovascular or systemic diseases and that they were not taking any medication when joining the study. A medical, electrocardiographic and echocardiographic evaluation was made at the Vozzi Institute on each of the athletes on the same day. Their informed consent was requested for their participation in the study.

Medical examination

A physical examination was made, consisting in taking the vital signs and obtaining the anthropometric parameters (weight, height and body surface area).

Electrocardiogram

The 12-lead ECGs were performed in the supine position with a Fukuda Cardisuny model 501 B electrocardiogram machine with a recording speed of 25 mm/second with a standard of 1 mV/Cm. The tracings were interpreted by one of the authors, who was unrelated to the results of the echocardiograms, which were analysed in accordance with the guidelines issued by the European Society of Cardiology in 2010[6]. An evaluation was made of heart rhythm, heart rate, electrical axis, duration and voltage of the P wave, PR interval, QRS complex (duration and voltages), QT interval (its duration corrected using the Bazett formula), ST segment and the T wave. The left atrial enlargement (LAE) was defined as a duration of the P wave greater than 120 milliseconds in Lead II or a negative portion of the same ≥ 0.1 mV and ≥ 40 ms in V1[7]. Right atrial enlargement (RAE) was defined as a P wave with an amplitude greater than 0.25 mV at leads II and III or greater than 0.15 mV in V1 or V2[8]. The LVH and right ventricular hypertrophy (RVH) with the presence of at least 1 criterion with specificity greater than 85% (Tables 1 and 2) (Figure 1).

Table 1. Left ventricular overload criteria.

Left ventricular overload	Criterion N°
(R I) + (S III) ≥ 25 mm (Gubner 1943)	1
R of aVL ≥ 7.5 mm (Minnesota)	2
R of aVL > 11 mm (Sokolow 1949)	3
R of aVF > 20 mm (Golderberg 1949)	4
(S of V1) + (R of V5-6) > 35 mm (Sokolow 1949)	5
Product of Sokolow (Sokolow x duration QRS) >2880 mm/ ms (Molloy 1992)	6
R avL + S V3 >28 mm en ♂ (Cornell 1985)	7
Product of Cornell (Cornell x duration QRS) >2440 mm/ ms (Molloy 1992)	8
Maximum S + S V4 > 28 mm (Peguero Lo Presti 2017)	9
S (maximum) + R (maximum)= 45 mm (Friedman 1977)	10
R V5 or V6 > 26 mm (Sokolow 1949)	11

Table 2. Right ventricular overload criteria.

Right ventricular overload	Criterion N°
R/S at V1 ≥ 1 (Myers 1948)	1
R at V1 ≥ 7 mm (Sokolow 1949)	2
R/S at V5 or V6 ≤ 1 (Sokolow 1949)	3
S at V5 or V6 ≥ 7 mm (Sokolow 1949)	4
R at V1 + S V5/ V6 ≥ 10.5 mm (Sokolow 1949)	5
R V5/ V6 ≤ 5 mm (Sokolow 1949)	6
R of avR ≥ 5 mm (Sokolow 1949)	7
Electrical axis ≥ 110°	8

Figure 1. V1 and V6 leads for athlete with Sokolow positive for LVO.

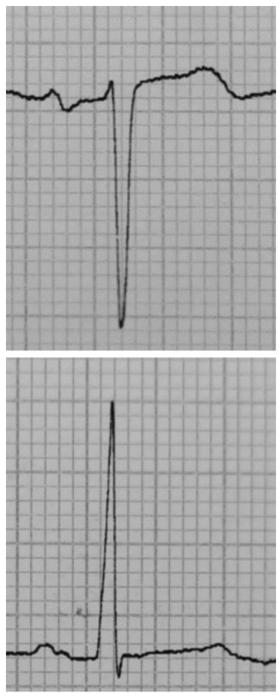
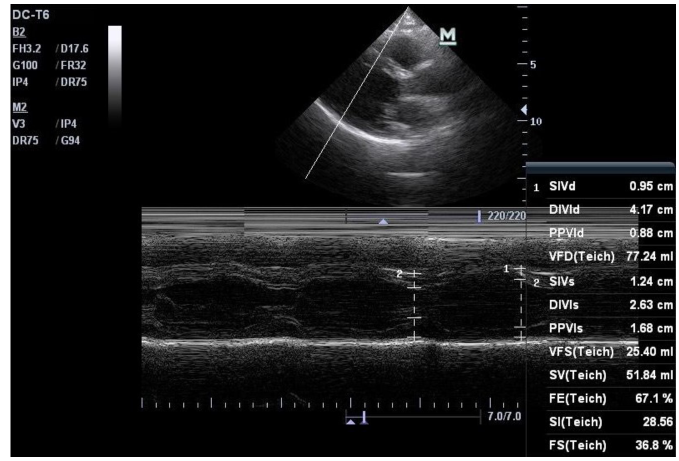


Figure 2. Measurement of size and thicknesses of LV.



Echocardiogram

The Doppler echocardiograms were performed by the same operator with a Mindray DC-T6 machine with a 2 to 4 MHz transducer. The operator was unrelated to the ECG results. The athletes were studied at rest, in a left lateral decubitus position. The dimensions and thickness measurements of the LV were obtained by M-mode two-dimensional images from the long-axis and short-axis parasternal views, based on the recommendations of the American Society of Echocardiography in effect at the time of the study[9] (Figure 2). The left ventricular mass was determined by the linear method with the cube formula (mass of VI = 0.8 x (1.04 x (diastolic diameter of LV (LVDd) + septal diastolic thickness + diastolic thickness of posterior wall)^3 - LVDd^3} + 0.6 g). The values were compared to those considered to be normal, adjusted for body surface area (BSA) (obtained according to the formula of Du Bois and Du Bois). A cutoff value was taken as an LV mass index of 15 g/m^2. The end-systole areas and volumes of both atria were obtained from an apical 4-chamber view. In the same view, but focused on the right chambers, measurements were made of the dimensions of the right ventricle (RV); the transverse diameters were obtained at the base level of the same (inflow tract) and in the middle third at the level of the papillary muscles, all at the end of the diastole, in accordance with the echocardiographic evaluation guidelines for the right chambers[10]. Measurements of the RV were also obtained on the short axis of the large vessels, where the diameter of the proximal and distal outflow tracts was measured. Finally, the right ventricular free wall thickness was measured from a subxiphoid view. The LV systolic function was evaluated following Simpson's biplane method, from apical 4- and 2-chamber views.

Atrial dilation, by echocardiography, was defined as an increase in atrial diameters, area and/or volume. With regard to the ventricles, dilation was defined as an increase in ventricular diameters and hypertrophy due to increased wall thickness (in the case of the RV) or of the indexed mass (for the LV). The measurements were valued as quantitative variables.

Statistical analysis

Descriptive statistics were calculated for all the variables studied.

The means obtained for each variable were compared by applying the ANOVA tests for independent samples, once compliance with the necessary assumptions had been evaluated. The Kolmogorov-Smirnov test was used to verify whether the data were normally distributed, while the homogeneity of variance was determined with the Levene test.

Furthermore, analyses of covariance (ANCOVA) were conducted in order to make adjustments for any possible confounding variables (age, weight, height, body surface area and sport practised) according to the parameters compared between athletes with and without hypertrophy. The adjusted means were as expressed as a ± standard error of the mean. By using the analysis of covariance , we considered the possibility of "correcting" or "adjusting" the difference found between the groups for the covariates with significant differences in order to make the study groups comparable.

An evaluation was made between the presence of LAE in the ECG and the presence of increased echocardiographic diameter, area or volume of the same cavity. Furthermore, an evaluation was made between the presence of RAE in the ECG and the presence of increased echocardiographic area or volume of the same.

An evaluation was also made of the relationship between the presence of LVH in the ECG and the mean values of the absolute and indexed LV diastolic diameter, posterior wall thickness, absolute mass and indexed mass of the LV.

To analyse the existence of differences in the presence of LVH in the ECG between the 3 groups of athletes, the absolute and relative frequencies were calculated and these were compared by applying the Freeman-Halton test (extension of Fisher's exact probability test for a 2-row by 3-column contingency table).

For all the LVO criteria that were positive, we determined sensitivity, specificity, positive and negative predictive value in relation to the presence of an increased LV mass index.

In all cases, the accepted significance cutoff value was $p < 0.05$.

Results

30 athletes (10 per group) were included in the study period with a mean age of 23.9 ±5 years (Table 3). A comparison of the mean age of the athletes in the three disciplines studies revealed statistically significant differences (W: 21.1 ± 2.1; S: 22.9 ± 4.4; T: 27.7 ± 5,5; p= 0.005). When performing post hoc multiple comparisons, it was found that the differences observed were attributed to the age differences of T (older) in relation to the W (p=0.005). The mean blood pressure was 117/69 mm Hg; the mean heart rate was 55 beats per minute. There were no differences in these parameters between the different groups. With regard to the anthropometric parameters, statistically significant differences were found in the mean height of the athletes of the three disciplines studied

(W: 183.70 ± 6.897; S: 177.90 ± 7.172; T: 172.90 ± 9.480 cm; p= 0.019). When performing post hoc multiple comparisons, it was found that the differences observed were attributed to the height difference between the W and T (greater in W, p=0.015). Likewise, a significant difference was found in the mean weight of the athletes in the three disciplines

(W: 88.0 ± 8.4; S: 73.0 ± 10.1; T: 69.5 ± 13.7 kg; p= 0.002). In this case, the differences are due to the fact that the W have a greater mean weight than the T (p=0.002) and the S (p=0.015). For this reason, the mean body surface area was 1.942 m^2, greater in W in relation to T (1.8±0.20, p=0.004) and S (1.9±0.16, p=0.003) (Table 3).

With regard to the echocardiographic evaluation, it was observed that the mean of the septal thickness, the LV mass index, the anteroposterior diameter and the area of the LA, the area of the RA, and the RV base were above the normal values for the general population (Table 4). The type of hypertrophy found was eccentric (16 athletes) and concentric (1 athlete); 9/10 athletes, 6/10 water polo players and 2/10 swimmers exhibited hypertrophy.

With regard to the ECG analysis, none of the athletes showed LA or RA enlargement. Nine of the 30 athletes (30%) exhibited signs of LVH (3W and 6T). None exhibited RVH. The evaluation of sensitivity, specificity, positive and negative predictive value for all the LVH criteria that showed positive results in relation to the increase of the LV mass index are shown in Table 5. Of particular note is the low sensitivity of the criteria, as well as a high specificity and positive predictive value.

As can be seen in Table 6, the LV diastolic diameter (LVDd) indexed to the body surface area (BSA) was greater in athletes with LVH in the ECG (29.68 vs 27.35 mm, p=0.015). The measurements adjusted by age, weight, height, body surface area and the sport practised (p*) continued to show the same behaviour (means adjusted for the LVDd indexed to the BSA of athletes with LVH in ECG: 28.4±0.6 mm; CI95%= 27.78-30.10; for athletes with no LVH in ECG: 27.67±0.36 mm; CI95%= 26.93-28.41). A significant difference was also found for the left ventricular mass index (LVMI), being greater (in the hypertrophy range) in athletes with LVH by ECG (126.89 vs 111.67 g/m^2; p= 0.049). When the ANCOVA was applied,

Table 3. Characteristics of the athletes.

Parameter	Range	Mean				p (between groups)*
		Overall	W	S	T	
Age (years)	19-39	23.9	21.1	22.9	27.7	T vs W: p=0.005 T vs N=NS
Systolic pressure (mm Hg)	100-138	117.2	120.0	118.0	113.5	NS (p=0.416)
Diastolic pressure (mm Hg)	60-80	69.7	68.0	69.0	72.0	NS (p=0.429)
Heart rate (beats per minute)	44-94	65.8	61.9	70.1	66.3	NS (p=0.290)
Height (cm)	162-193	178.16	183.7	177.9	172.9	W vs T: p=0.015 W vs N=NS
Weight (kg)	55-100	76.83	88.0	73.0	69.5	W vs N: p=0.015 W vs T: p=0.002
Body surface area (m^2)	1.58- 2.31	1.9	2.1	1.9	1.8	W vs N: p=0.003 W vs T: p=0.004

*Bonferroni.post-hoc tests

Table 4. Description of the echocardiographic values for the athletes studied.

Echocardiographic parameter	Mean ± SD	Minimum	Maximum	P50 (Mean)	% abnormal values
Diastolic diameter left ventricle (mm)	54.29 ± 3.80	48.50	64.70	54.40	10
LVDD indexed to body surface area (mm)	28.05 ± 2.47	24.15	32.71	27.56	20
Diastolic interventricular septum (mm)	11.22 ± 0.86	9.70	12.70	11.30	63
Diastolic posterior wall (mm)	10.27 ± 0,91	8.30	11.80	10.40	36
Left ventricular mass (grams)	229.00 ± 41.50	168.00	323.00	222.50	46
Left ventricular mass index (g/m^2)	116.23 ± 19.72	59.00	158.00	117.00	56
Relative parietal thickness	0.37 ± 0.04	0.30	0.45	0.38	13
Left atrial anteroposterior diameter (mm)	40.19 ± 3.52	35.00	48.50	40.10	36
Left atrial area (cm^2)	21.25 ± 3.10	15.00	26.85	21.28	60
Left atrial volume (ml/m^2)	33.91 ± 5.43	22.60	43.00	34.34	40
Right atrial area (cm^2)	19.84 ± 2.81	14.64	27.14	19.83	56
Right atrial volume (ml/m^2)	31.69 ± 5.88	21.90	46.00	30.85	33
Right ventricular base diameter (mm)	42.31 ± 4.00	35.00	50.00	42.00	56
Right ventricular mean diameter (mm)	31.60 ± 7.50	24.4	42.00	33.00	20
Proximal right ventricular outflow tract (mm)	33.36 ± 3.46	28.00	40.00	33.00	30
Distal right ventricular outflow tract (mm)	25.09 ± 5.60	20.00	30.00	26.00	33
Right ventricular thickness (mm)	4.45 ± 0.59	3.00	5.80	4.50	0

Note: In all cases, the data distribution normality and the variance homogeneity were verified.

Table 5. Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) of the different ECG criteria for LVO in relation to the presence of hypertrophy due to the increased LV mass index.

ECG criteria	Sensitivity %	Specificity %	PPV %	NPV %
R of aVF > 20 mm	6	100	100	47
(S of V1) + (R of V5-6) > 35 mm	35	95	86	63
Product of Sokolow >2880 mm/ms	35	95	86	64
R aVL + S V3 >28 mm	6	100	100	47
Maximum S + S V4 > 28 mm	24	100	100	57
S (maximum) + R (maximum)= 45 mm	24	100	100	57
R V5 or V6 > 26 mm	24	94	80	57

it was observed how the adjustment made the previously encountered significant differences disappear (LVMI adjusted means in athletes with LVH in ECG; 126.53±7.53 g/m^2; CI95% = 111.41-141.65; for athletes with no LVH in ECG: 111.37±4.93 g/ m^2; CI95%= 101.11-121.62). The remaining echocardiographic variables showed no statistically significant differences between athletes with or without LVH in the ECG.

The relationship between the sport practised and LVH in the ECG is shown in Figure 3. The proportion of athletes with LVH differs according to the sport practised (p=0.0155). The proportions between the S and W were compared by applying the Fisher test, obtaining a value of p=0.2105. Therefore, the proportion that differs corresponds to the T, who exhibited a greater LVH in the ECG than the athletes from the other disciplines.

Discussion

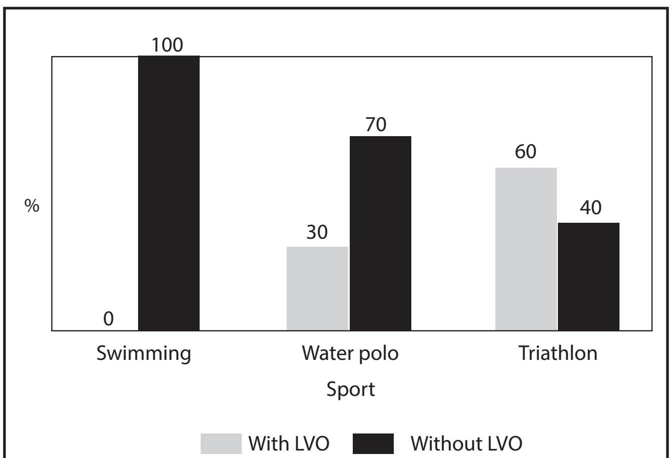
The principal electrocardiographic changes related to ventricular hypertrophy are shown in the QRS voltage and duration, the electrical axis and the ventricular repolarisation alterations. The increase in the voltage of the ventricular activation complex is due to the increase in the ventricular depolarization mass; the increase in the QRS duration is the product of the prolongation of the propagation of the impulse in the hypertrophic myocardium. The electrical axis deviation is a factor that supports the diagnosis of hypertrophy and is most significant in confirming the right hypertrophy criteria. The secondary ST-T alterations support the diagnosis of LVH in the presence of voltage criteria; by themselves they are insufficient to make a diagnosis. The atrial enlargements are reflected in the duration, morphology and voltage of the P wave[4,6].

Tabla 6. Comparison of the echocardiographic variables of the left ventricle in relation to the presence or absence of LV overload in the ECG.

	LVO in ECG	S	Mean ± SD	CI 95%	Minimum	Maximum	p	p*
Left ventricular diastolic diameter (mm)	No	21	53.86 ± 3.74	52.16-55.56	48.50	64.70	0.359	0.134
	Yes	9	55.28 ± 4.00	52.21-58.35	49.70	60.30		
	Total	30	54.29 ± 3.80	52.87-55.71	48.50	64.70		
LVDd indexed to the body surface area (mm/m²)	No	21	27.35 ± 1.98	26.45-28.25	24.20	32.30	0.015	0.001
	Yes	9	29.68 ± 2.84	27.50-31.87	24.75	32.71		
	Total	30	28.05 ± 2.47	27.13-28.98	24.15	32.71		
Diastolic interventricular septum (mm)	No	21	11.04 ± 0.89	10.64-11.45	9.70	12.50	0.090	0.645
	Yes	9	11.62 ± 0.66	11.12-12.13	10.80	12.70		
	Total	30	11.22 ± 0.86	10.90-11.54	9.70	12.70		
Diastolic posterior wall (mm)	No	21	10.29 ± 0.88	9.89-10.68	8.30	11.50	0.865	0.323
	Yes	9	10.22 ± 1.05	9.41-11.03	9.00	11.80		
	Total	30	10.27 ± 0.92	9.93-10.61	8.30	11.80		
Left ventricular mass (grams)	No	21	224.71 ± 38.62	207.13-242.30	168.00	323.00	0.397	0.287
	Yes	9	239.00 ± 48.52	201.71-276.29	186.00	321.00		
	Total	30	229.00 ± 41.50	213.50-244.50	168.00	323.00		
Left ventricular mass index (gr/m²)	No	21	111.67 ± 18.29	103.34-119.99	59.00	140.00	0.049	0.124
	Yes	9	126.89 ± 19.78	111.68-142.10	93.00	158.00		
	Total	30	116.23 ± 19.72	108.87-123.60	59.00	158.00		
Relative parietal thickness	No	21	0.38 ± 0.04	0.36-0.39	0.30	0.45	0.449	0.944
	Yes	9	0.37 ± 0.04	0.33-0.40	0.33	0.45		
	Total	30	0.37 ± 0.04	0.36-0.39	0.30	0.45		

p*: Analysis adjusted by age. weight, height, body surface area and sport.
Note: the calculations are made as if all the groups had the same covariate distribution.
In all cases, the data distribution normality and the variance homogeneity were verified.

Figure 3. Relationship between the sport practised and LVH in the ECG.



These changes were directly correlated (autopsy in which the LV was weighed following removal from the heart of the right chambers, left atrium, aorta and epicardial fat, or else the thickness of the RV wall was measured) or indirectly (through imaging methods such as radiography, ventriculography, echocardiography and nuclear magnetic resonance) with the dimensions and/or mass of the different cavities in order to establish the overload criteria by ECG[1,11-16].

The LV was the most-studied cavity and its electrocardiographic expression falls to the QRS voltage. This does not only depend on the presence of hypertrophy, but it is also influenced by age, sex, ethnicity, physical habits, the geometric pattern of the hypertrophy, its severity and the orientation of the heart in the thorax.

With regard to the athletes, the prevalence of morphological and electrical changes varies according to sex (predominantly male), ethnicity (black), type (greater in endurance sports such as rowing and athletics and lower in strength sports), the intensity and standard of competition, in response to physical training[17-20]. Most sports have mixed components, that is to say that they combine volume overload and pressure and, unlike other pathological processes, the overload of the cardiovascular system is of an intermittent nature. This leads to the physiological remodelling of the LV which, on an echocardiogram, is manifested as varying increases in its size, thickness and mass, the latter being either concentric or eccentric. With regard to the sports that we are evaluating, triathlon is considered to be a sport with high/high static and dynamic components, swimming moderate/high, while water polo does not appear in the classification of the last Bethesda conference [21], given that it is a little-studied sport.

The remodelling of the LV is expressed in the ECG as an increase in the QRS voltage as an isolated criterion of LVH, and is not accompanied by non-voltage criteria (LAE, electrical axis deviation, pathological Q waves, slow intrinsicoid deflection of the QRS, or repolarisation alterations)[17,22,23]. In this regard, Pelliccia *et al*[17] evaluated the ECGs of 1005 elite Italian athletes, observing 40% anomalies of which 60% had voltage criteria for hypertrophy with greater prevalence in endurance athletes. The voltage was associated with the male sex and the presence of an increased cavity and mass, on echocardiography. None of the athletes with isolated voltage criteria had structural abnormalities such as hypertrophic cardiomyopathy, as suggested by studies that observed this isolated alteration in less than 2% of patients with this cardiomyopathy[24-26]. Non-voltage criteria were not observed.

Most studies that have evaluated the ECG criteria of LVH were conducted on endurance athletes and taking the echocardiogram as a reference, however the results are not homogeneous. One fact that cannot be ignored is that the mass cut-off values when defining LVH by echo have varied over time (their value has decreased).

A study by Somauroo *et al*.[27] on adolescent soccer players observed that 50% exhibited Sokolow's positive LVH criteria, with poor echocardiographic correlation. Age appears not to be a determining factor in these findings, given that similar results were found in veteran long-distance runners with a mean age of 56 years[28]. Finally, a study by Douglas *et al* on marathon runners showed a 57% increased LV mass by echo. The Sokolow criteria had the most S (65%) with an E of 61%, while the most specific was Cornell (95%), but with an S wave of 8%[29]. No correlation was found between the LVH signs with the LV mass and size. There were no non-voltage signs of LVH in any of the mentioned tests.

In our study, we found a statistically significant correlation between the LVH by ECG and the LVDd indexed to the BSA as shown by earlier studies that have demonstrated that persons with increased LVDd, with no increase in thicknesses, may exhibit electrocardiographic criteria for LVH[30,31]. However, there are no criteria that make it possible to clearly distinguish between concentric or eccentric hypertrophy or dilation of the LV without hypertrophy[32]. It should be emphasised that in 8 out of 9 cases with ECG criteria of LVH, the athletes showed more than one criterion for hypertrophy in the ECG. The products of the Sokolow and Cornell criteria did not improve the S wave of the same, probably because there was no prolongation of the QRS in the athletes.

Other studies have found that the ECG criteria for LVH detection are more correlated with the increase of the LV mass than with the thickness and size of the same, by echo[33,34]. In our study, 56% of athletes had an increased LV mass index (94% eccentric type); Sokolow was the criteria with more S (35%); the E wasgreater than 90% for all criteria. However, there was no significant correlation between the LVMI and the LVH in the ECGs.

With regard to the sports assessed in our study, we found a greater frequency of LVH in the ECG of triathletes compared to the other groups, coinciding with the finding of LVH by echo in 90% of them. This could be related to the fact that this is the most demanding discipline, given that it combines three types of sport, added to the fact that their weight is lower than swimmers and water polo players, which facilitates detection of LVH by voltage criteria.

Worthy of note is the low voltage found in the avL lead, to such an extent that no athlete had this positive criterion and this also had a bearing on a low S wave for Cornell. Some of the causes that could explain this are the predominantly vertical electrical axes of the athletes or the neutralisation of the left electrical forces by the right ones, although these did not frequently appear in the ECGs, they did appear in the echocardiograms (with dilation of the right chambers).

In relation to the time that the cardiac adaptations take to appear, a study on pre-adolescents with a mean age of 12 years and with at least 3 years of training, showed significant differences in the precordial voltages and in certain echocardiographic parameters (diameter of the LA of the LV and mass index) compared to a control group[35].

With regard to the right chambers, Pellicia *et al*[17] reported a prevalence of 0.08% RAE and 0.6% 110º main axis in the same population of athletes. Another study showed 0.6% for Sokolow criteria for right VH in 172 adolescent soccer players[27]. Given the prevalence of the said alterations, the presence of RAE and RVH in the ECG should not be interpreted as physiological and it would be worth examining the images in order to rule out a pathology.

In our population, 56% of the athletes exhibited an increase in the size of the RV inflow tract, however this was not reflected in the ECGs.

With regard to the atria, dilation was observed, which varied according to the quantification method used (diameter, area, volume) from 36 to 60% for the LA and from 33 to 56% for the RA. Neither was this reflected in the ECGs, reflecting their low S wave.

The abnormal echocardiographic findings in our athletes were interpreted as adaptive given the normal biventricular Systo-diastolic function in all of them. In the ECG, we observed no pathological alterations of the ST and T wave, another indication of physiological changes in the athletes.

Based on our analysis, we could say that the S wave of the ECG for the detection of LVH in athletes is low and it is very unlikely that the refinement of the criteria may improve this relationship. However, we did find a correlation between the LVH by ECG with the diastolic dimensions of the LV by echo. The capacity of the ECG to detect physiological hypertrophy, dilation and thickness increases may differ from the pathologies and the different structural or geometrical characteristics of the myocardium between one pathology and another, may exert an influence on this.

Finally, although the number of athletes included in our study is small for the purpose of issuing valid conclusions, it should be emphasised that many studies that have evaluated the electro and echocardiographic manifestations of training, some of which we have cited herein, were based on a number of athletes that is similar to ours[28,29,33,36,37].

Limitations

A limitation of this study is the small number of athletes included to date. Even so, we consider that this limitation has been offset by a thorough collection of information on the variables evaluated and with the application of rigorous statistical analyses.

Another limitation was the fact that we had no precise information on how long the athletes had been training, which could have an influence on the adaptive phenomena observed.

It should be pointed out that the results of our study cannot be generalised to recreational sports. Furthermore, this evaluation only includes men.

Conclusions

Certain echocardiographic parameters in our population of athletes were above the normal values for the general population. No relationship was found between the electrocardiographic signs of the enlargement of the LA, RA or hypertrophy of the RV with the echocardiographic findings.

The ECG criteria for LVH voltage were correlated to a greater LVDd indexed by echocardiography and were most frequently observed in the triathlon group.

The morphological and functional adaptive changes to training on the cardiovascular system continue to be the subject of prospective investigations in order to provide new evidence with regard tot he best way to evaluate high performance athletes. Each and every one of the sports disciplines must be included and analysed in order to identify the cardiac impact generated by the different stimuli sustained, particularly those disciplines that are still little studied.

Conflict of interest

The authors do not declare a conflict of interest.

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