

Review

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Cardiac Adaptation in Performance Athletes: A Systematic Review

Cosmin-Gabriel Ursu , [Stefan Busnatu](#) , [Cristiana-Flavia Cristea](#) , [Obaida Makdah](#) , [Al Krayem Feras](#) , [Daniela Miricescu](#) ^{*} , Silvia Nica , [Silviu Dumitrescu](#) , [Constantin Stefani](#) , Maria Greabu , [Iulia-Ioana Stanescu-Spinu](#) , [Mariana Jinga](#) , [Silviu Stanciu](#) ^{*}

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Review

Cardiac Adaptation in Performance Athletes: A Systematic Review

Cosmin-Gabriel Ursu ^{1,†}, Stefan Busnatu ², Cristiana-Flavia Cristea ¹, Obaida Makdah ¹, Al Krayem Feras ¹, Daniela Miricescu ^{3,*}, Silvia Nica ^{4,5,†}, Silviu Dumitrescu ⁶, Constantin Stefani ⁷, Maria Greabu ³, Iulia-Ioana Stanescu-Spinu ⁸, Mariana Jinga ⁹ and Silviu Stanciu ^{9,*}

- ¹ Carol Davila University of Medicine and Pharmacy, Bucharest, Romania, cosmin.d.v.ursu@stud.umcf.ro (C.-G.U.), cristiana-flavia.cristea0721@stud.umfcd.ro (C.-F.C), obaida.makdah@stud.umfcd.ro (O.M.), firaskrayem2@stud.umfcd.ro (K.F.)
- ² Discipline of Cardiology, Carol Davila University of Medicine and Pharmacy, Bucharest, Romania, stefan.busnatu@umfcd.ro (S.B.)
- ³ Discipline of Biochemistry, Faculty of Dentistry, Carol Davila University of Medicine and Pharmacy, Bucharest, Romania, maria.greabu@umfcd.ro (M.G.)
- ⁴ Emergency Discipline, University Hospital of Bucharest, Romania, silvia.nica@umfcd.ro (S.N.)
- ⁵ Carol Davila University of Medicine and Pharmacy
- ⁶ Central Emergency University Military Hospital, 013058 Bucharest, Romania, dr.silviu.dumitrescu@gmail.com (S.D.)
- ⁷ Department I of Family Medicine and Clinical Base, "Dr. Carol Davila" Central Military Emergency University Hospital, Bucharest, Romania, constantin.stefani@umfcd.ro (C.S.)
- ⁸ Discipline of Physiology, Faculty of Dentistry, Carol Davila University of Medicine and Pharmacy, Bucharest, Romania, iulia.stanescu@umfcd.ro (I.-I.S.-S.)
- ⁹ Department of Internal Medicine and Gastroenterology, Carol Davila University of Medicine and Pharmacy, Central Military Emergency University Hospital, Bucharest, Romania
- * Correspondence: daniela.miricescu@umfcd.ro (D.M.); silviu.stanciu@umfcd.ro (S.S.)
- † These authors contributed equally to this work.

Abstract: Athlete's heart is characterised by structural, functional, and electrical remodelling. During an exercise session, the heart increases the use of oxygen, which is associated with decreased vascular resistance in the lungs' circulation. Left ventricular hypertrophy is an echocardiographic modification especially met in endurance athlete's heart. Moreover, left atrial dilatation represents another important characteristic of endurance athletes. Electrical modulation in the athletic heart induces neurological modifications leading to sinus arrhythmia, bradycardia, early repolarisation, and first-grade atrioventricular (AV) blocks. Athletes' nutritional status involves the assimilation of macronutrients such as proteins, carbohydrates, and fats. Depending on the duration of physical exercise, ATP can be obtained in aerobic or anaerobic conditions. This systematic review aims to find correlations between different evaluative methods of cardiac adaptation and present the metabolism in performance athletes.

Keywords: athlete's heart; electrical remodelling; ventricular hypertrophy; left atrial dilation; endurance athletes

1. Physiological Modifications in Athlete's Heart

The correlation between exercise and heart remodelling gives birth to the athlete's heart, which is characterised by structural, functional, and electrical remodelling; the intensity of exercise plays an important role in the remodelling limits [1,2]. Athletes detain specific heart characteristics regarding structural and physiological changes. The heart increases the use of oxygen (VO₂) and its output during an exercise session. These changes are associated with a lower vascular resistance compared to the untrained heart, especially in the lungs' circulation. These reasons presented as the increased perfusion alongside vasodilatation explain the increase in right ventricle afterload and pulmonary artery pressure [3].

Echocardiographic modifications in an athlete's heart include left ventricular hypertrophy, considered concentric in strength athletes and eccentric in endurance athletes. Through echocardiography, we can observe a reversible alteration in the structure of the heart, more precisely, an increase in the left ventricular mural thickness by 15-20% alongside an increase in the left ventricular size by 10-15% [4,5].

Another important element in athletic cardiac remodelling is left atrial dilation, mainly in endurance athletes. An increase in the atrial size of more than 50mm in men and 46mm in women is explained by the increased flow associated with the left ventricle's increased size. The ventricular alteration explains the supraventricular arrhythmic tendencies in this population. [6].

The left atrium pressure increases during exercise, leading to a charge in the pulmonary circulation, which increases the right ventricular filling. This leads to a dilated right ventricle [3]. The athletic heart also adapts functionally, which is observed by a higher capability during exercise to increase the stroke volume [7]. Especially in endurance exercise, we observe an improved left ventricle diastolic chamber distensibility and compliance [6].

Electrical modulation in the athletic heart comes hand in hand with the cardiac autonomic nervous system conditioning, including the withdrawal of sympathetic impulses and increased vagal tone. These neurological modifications lead to sinus arrhythmia, bradycardia, early repolarisation, and first-grade AV blocks. The ECG traces can be classified as follows: training-related changes (bradycardia, bundle branch blocks, AV blocks, QRS modifications correspondent to left ventricular hypertrophy) and changes unrelated to exercise (terminal phase alterations, ST depression, abnormal Q wave). All the modifications listed above should correlate with the patient's demographic characteristics [8].

This systematic review aims to find possible correlations between different evaluative methods of cardiac adaptation in performance athletes. In this regard, we have 3778 performance athletes split between 17 original studies published in the last ten years

2. Materials and Methods

2.1. Inclusion/Exclusion Criteria

Our eligibility criteria were:

- 1) Studies that have been published in the last ten years in peer-reviewed journals;
- 2) We only took into consideration the adult cohorts in studies
- 3) Mandatory mention of left ventricular parameters
- 4) Mandatory mention of ultrasonography parameters

Our search strategy was centered around the concepts of cardiac adaptation and athletes, using the following keywords: "myocardial adaptation", "athlete", "sport", and "cardiac remodelling". We had four independent reviewers search through the following databases: Pubmed, ResearchGate, Oalib, Base, Google Scholar, and ScienceDirect. The Interventional studies involving animals or humans, and other studies that search were conducted from 05.01.2024 to 20.03.2024 and found 2451 initial records from which we were left with 17 original studies (Figure 1). Clinical trial databases and grey literature were not included in the search protocol.

Our data was collected using Microsoft Excel 2023 V16 independently by four researchers, followed by cross-examination and verification among them, with any problems being brought up and solved via group decision.

Another important mention is that this review follows the PRISMA guidelines but was not registered in any database beforehand.

PRISMA 2020 flow diagram for new systematic reviews which included searches of databases and registers only

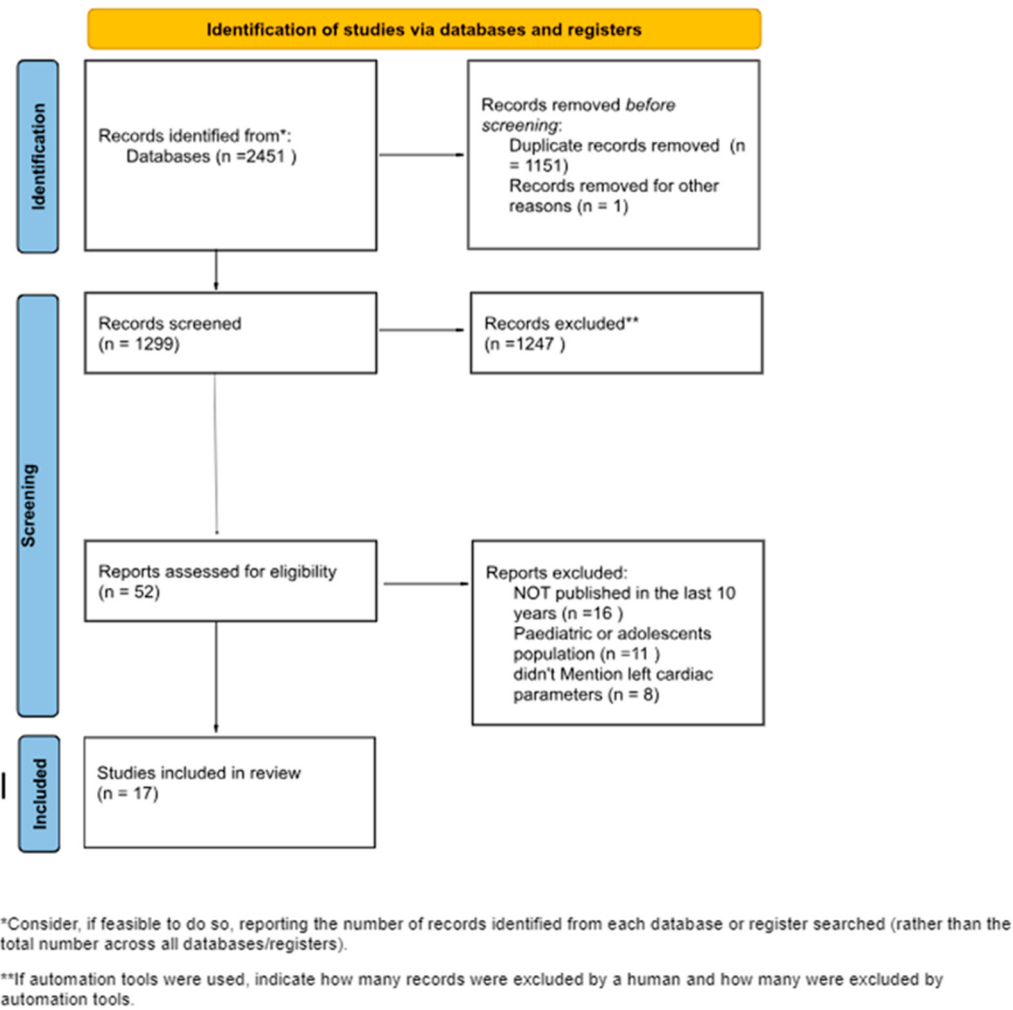


Figure 1. PRISMA flow diagram of our systematic review.

The main focus of our study was related to the type of sport, the ventricular mass, left ventricular volumes, left atrial volumes, and their respective indexes to the body surface area alongside the strain measurements of the left ventricles. We have also collected all the echocardiographic parameters measured by the 17 studies that were found to be statistically significant($p<0.05$).

2.2. Risk of Bias

The tool used when assessing the risk of bias was the Robins-E tool [9] due to the non-randomized nature of the studies analyzed in this systematic review. The choice for the tool was collectively made when taking into consideration the seven parameters (bias due to confounding, measurement of exposure, selection of the participants, post-exposure interventions, missing data, measurement of the outcome, and selection of the reported result) it evaluates as being the most probable to find any relevant source of bias (Figure 2). The traffic light plot used to show the assessment was made using the ROBVIS tool. Four reviewers independently assessed the risk of bias, with one of them formulating the results using the ROBINS-E tool, and all discrepancies were discussed among the team and solved accordingly. None of the authors of the studies included in the analysis have declared any conflicts of interest, and none of the 17 studies were financed by a pharmaceutical or biotechnological company.



Figure 2. Risk of bias domains.

3. Results

The results of the reviewed studies [10-26] are presented in Tables 1–3.

Table 1. Female Elite Athlete Echographic Findings.

Article	Patients	Type of sport / Intensity	LEFT VENTRICULAR MASS+ INDEX	VOLUMES	STRAIN	LEFT VENTRICULAR ECHO FINDINGS	RIGHT VENTRICULAR ECHO FINDINGS
						LVEF(%) (WD) 59,33 ± 7,23 (ITF) 63,06 ± 7,40	
						LVEDD (ml) (WD) 124,63 ± 32,70 (ITF) 117,76 ± 9,65	RVEDD (mm) (WD) 48,94 ± 4,35 (ITF) 47,59 ± 2,86
Hammami et al., 2021	20	STRENGTH				LVESD (ml) (WD) 51,55 ± 12,29 (ITF) 50,57 ± 10,26	RVESD (mm) (WD) 28,38 ± 3,39 (ITF) 30,00 ± 2,07
						IST (mm) (WD) 7,07 ± 0,62 (ITF) 8,19 ± 1,37 (p<0,05)	RVL (mm) (WD) 65,35 ± 8,51 (ITF) 64,37 ± 3,44
						PWIVS (mm) (WD) 6,85 ± 0,62 (ITF) 7,43 ± 0,74	
Finocchiario et al., 2017	1,083	ENDURAN CE+ MIXED	LVM/BSA (g/m2) 83 ± 17 (p=0.001)			LVEDD (mm) 49 ± 4 (p<0.001)	

		TYPE EXERCISE REGIMEN	LVEDD/BSA (mm/m^2) 28.6 ± 2.7(p<0.001) IVs (mm) 8.7 ± 1.2 (p<0.001) PW (mm) 8.4 ± 1.2 (p<0.001) RWT: 0.35 ± 0.04 (p<0.001) RWT >0.42: 34 (8) (p=0.04)			
Giraldeau et al., 2015	315	STRENGTH + MIXED TYPE EXERCISE REGIMEN	Stroke volume (ml) 75 +/- 12(0.001) LV end-diastolic volume absolute (ml) 142 +/- 23(0.001) LV end-diastolic volume indexed to BSA (ml/m2) 83 +/- 12(0.001) LV Mass-to-Volume ratio 0.79 +/- 0.09 (0.015) LV Mass-to-Volume ratio (Allometric correction) 0.24 +/- 0.03 (0.035) Left atrial volume absolute (ml) 47.5 +/- 11.7 (0.001) RA volume (ml) 41,2 +/- 10,9 (p<0,0001) RA volume indexed BSA (ml/m^2) 24,1 +/- 5,7 (p=0,02)			
			2D GLS manual strain (%) (F) - 21.6 +/- 1.9(0.02) ; LV GLS (%) (F) - 22.0 +/- 1.9(0.02); Diastolic early SR (%) (F) 1.81 +/- 0.40(<0.01); RV GLS (%) - 24,9 +/- 2,6 (p=0,76) LA LS contraction (%) - 11,2 +/- 4,4 (p=0,65) RA LS contraction (%) - 13,2 +/- 5,3 (p=0,056)			
			RV area (cm^2) 22,8 +/- 3,5 (p<0,0001) Interventricular sept thickness (mm) (F) 6,1 +/- 1,0 (0.001); Posterior wall thickness (mm) (F) 7,0 +/- 1,1(0.001) Left ventricular internal diameter (mm) (F) 47,1 +/- 4,2(0.001)			
			RV dimensions basal (cm) 3,7 +/- 0,5 (p<0,0001) RV dimensions mid (cm) 2,9 +/- 0,5 (p<0,0001) RV longitudinal (cm) 8,2 +/- 0,7 (p<0,0001)			
D'Ascenzi et al., 2020	720	ENDURAN CE+	LVM(g) 143.9±31.4 (96–202) (0.0001)			
		STRENGTH + MIXED TYPE	LVM index, g/m2 81.8±15.1 (60–111) (0.0001) LV ejection fraction, % 67±6 (57–77) (0.0001) LV end-diastolic diameter, mm (F) 48.8±3.3 (44–54) (0.0001) RV basal diameter (mm) 35.1±4.8 (27–44) (p<0.0001) RV midcavity diameter (mm) 23.9±4.2 (18–31) (p<0.0001)			

				LV end-diastolic diameter index, mm/m2 28.6±2.4 (25–33) (0.0001)	RV longitudinal diameter, (mm) 79.4±9.3 (65–95) (p<0.0001)
				LV end-systolic diameter, mm 30.2±3.3 (25–36) (0.0001)	RV diastolic area (cm^2) 19.5±5.2 (13–27) (p<0.0001)
					RV systolic area (cm^2) 9.1±2.7 (5–14) (p<0.0001)
				Posterior wall, mm 8.4±0.8 (7–10) (0.0001)	
				RWT 0.35±0.03 (0.31–0.39) (0.0001)	
				Left atrial diameter, mm 32.3±3.8 (26–38) (0.0001)	
Roslan et al., 2023	100	ENDURANCE+ MIXED TYPE EXERCISE REGIMEN	LVM index (g/m2) 71,57 +/- 28,21 (p<0.001)	IVSd (cm) 0,77 +/- 0,11 (p=0,11)	
				GLS (%) 21,91 +/- 2,54 (p=0,53)	PWTd (cm) 0,8 +/- 0,31 (p=0,035)
				LVEDV index (mls/m^2) 57,25 +/- 11,55 (p<0,001)	LVIDd (cm) 4,56 +/- 0,32 (p=0,009)
				LArS (%) 48,07 +/- 10,06 (p=0,004)	IVSs (cm) 1,07 +/- 0,14 (p=0,943)
				LVESV index (mls/m^2) 24,03 +/- 7,55 (p<0,001)	LAcS (%) 36,3 +/- 8,30 (p=0,232)
					PWTs (cm) 1,25 +/- 0,17 (p=0,095)
				RVGLS (%) 22,85 +/- 3,08 (p=0,34)	LVIDs (cm) 2,94 +/- 0,29 (p<0,001)
					RWT 0,35 +/- 0,13 (p=0,131)
Lakatos et al., 2020	138	MIXED TYPE EXERCISE REGIMEN	LVM index (g/m^2) 88.9 ± 9.5 (p<0.001)	LA GLS (%) 34.3 ± 7.8 (p<0.01)	
				LV GLS (%) -19.4 ± 1.9 (p<0.001)	LVEF (%) 57.9 ± 4.7 (p<0.01)
					LVMi (g/m^2) 88.9 ± 9.5 (p<0.001)
				LV GCS (%) -28.1 ± 3.4 (p<0.05)	

Huang et al., 2022	598	ENDURANCE CE+ STRENGTH + MIXED TYPE	LVEF (%) 58.5 ± 5.4 (p=0.06)				
			LV stroke volume index (mL/m ²) 39.3 ± 7.8 (p<0.001)		LVGLS (%) -23.7 ± 2.3 (p<0.001)	IVSd (mm) 8.6 ± 1.3 (p<0.001)	RVEDA index (cm ² /m ²) 11,3 +/- 1,9 (p<0,001)
			LVM index (g/m ²) 79.7±18.8 (p<0.001)	LVEDV index (mL/m ²) 67.3 ± 12.1 (p<0.001)	LVGCS (%) -28.5 ± 4.1 (p=0.778)	LVPWd (mm) 8.7 ± 1.3 (p<0.001)	
			LVESV index (mL/m ²) 28.0 ± 6.3 (p<0.001)		RVLS (%) -27,9 +/- 4,4 (p=0,358)	LVIDd index (mm/m ²) 27.9 ± 2.9 (p<0.001)	RVESA index (cm ² /m ²) 6,7 +/- 1,3 (p<0,001)
			LVIDs index (mm/m ²) 18.4 ± 2.5 (p<0.017)				

Table 2. Male Elite Athlete Echographic Findings.

Article	Patient s	Type of sport / Intensity	LEFT VENTRICULA R MASS+ INDEX	VOLUMES	STRAIN	LEFT VENTRICULAR ECHO FINDINGS	RIGHT VENTRICULAR ECHO FINDINGS
Eun et al., 2016	29	MIXED TYPE EXERCISE REGIMEN	LVM (g) 216.7±21.9 (p=0,001) LVM index (g/height ²) 110.2±9.4 (p=0,001)	Stroke volume (mL) 89.3±15.4 (p=0,01) End-diastolic volume (mL/m ²) 152.0±19.4 (p=0,001) End-systolic volume (mL/m ²) 62.7±12.7 (p=0,001)	Longitudinal strain: -17.6+/- 1.8 (p=ns)	Ejection Fraction (%) 62.3±4.2 (p=ns)	
						Interventricular septal thickness (mm) 10.4±0.8 (p=0,01)	
						Posterior wall thickness (mm) 10.6±0.7 (p=0,001)	
						End-diastolic diameter (mm) 53.1±0.7 (p=0,001)	RV Systolic Area (m ²) 14.9±3.7 (p=0,005)
						End-systolic diameter (mm) 36.5±1.8 (p=0,001)	RV Diastolic Area (m ²) 26.1±7.1 (p=0,002)
						Relative wall thickness 4.0±0.3 (p=ns)	
						Cardiac index (L/m ²) 2.5±0.6 (p=ns)	
						End-systolic WS (mmHg) 240.9±32.6 (p=0,001)	
						End-systolic MWS (g/cm ²) 21.5±1.5 (p=ns)	
Cooke et al., 2018	11	ENDURANCE CE	LVM(g) 181 +/- 27 (p= 0,001)	Stroke volume(ml) 87±14 (p< 0,01)		Peak twist(degrees) 13.4±6.6 (p=0,0001)	
			LVM index 95 +/- 12 (p=0,001)	Stroke volume index(ml m ⁻²) 46±9 (p<0,01)		Peak torsion(degrees cm ⁻¹) 1.5±0.8 (p=0,0001)	

				End-diastolic volume(ml) 148±20 (p<= 0,01)	LV shortening(Δcm) 1.9±0.5 (p=0,0001)
				End-diastolic volume index(ml m-2) 78±12 (p<= 0,05)	Sphericity index 1.7±0.1 (p=0,0001)
				End-systolic volume(ml)61±16 (p=0,0001)	End-diastolic LV length(cm) 9.3±0.7 (p=0,0001)
				End-systolic volume index(ml m-2) 32±8 (p=0,01)	Cardiac output(L min-1) 4.3±0.8 (p=0,0001)
					Cardiac index(L min-1 m-2) 2.3±0.5 (p=0,0001)
Major et al., 2014	52	ENDURAN CE	LVM(g/m^3) 99,4 +/- 17,3 (p,0,001)	LV rel. Stroke Volume (ml/m^3) 36,6 +/- 8,9 (p<0,01)	rel.RVLADd (mm/m) 63.4 ± 6.3 (p<0,001)
					RVLA.Fr.Sh. (%) 25.9 ± 6.3 (p= NS)
					rel.RVSADd (mm/m) 27.3 ± 3.6 (p<0,001)
					rel.LVLADd (mm/m) 63.8 ± 5.6 (p<0,05)
					RVSA.Fr.Sh. (%) 20.3 ± 8.4 (p= NS)
					RVAd (cm2) 28.0 ± 5.0 (p<0,001)
					RVFAC (%) 42.7 ± 6.8 (p= NS)
Miranda et al., 2014	40	ENDURAN CE+ STRENGT H	LVM index (g m^-2) (endurance) 79,25 +/- 20,75 (p=0,01)	LVM index (g m^-2) (strength) 88,74 +/- 28,71 (p<0,01)	Left ventricular ejection fraction (%) (endurance) 62,70 +/- 6,34 (p=0,628)
					Left ventricular ejection fraction (%) (strength) 61,35 +/- 4,69 (p=0,163)
					Septum thickness (mm) (endurance) 8,40 +/- 1,60 (p=0,041)
					Septum thickness (mm) (strength) 9,75 +/- 1,86 (p=0,01)
					Posterior wall thickness (mm) (endurance) 8,50 +/- 1,43 (p=0,01)

				Posterior wall thickness (mm) (strength) 10 +/- 2 (p=0,01)
				Left ventricular end diastolic dimension (mm) (endurance) 48,65 +/- 4,70 (p=0,055)
				Left ventricular end diastolic dimension (mm) (strength) 49,85 +/- 4,20 (p=0,01)
				Left ventricular end systolic dimension (mm) (endurance) 30,20 +/- 3,25 (p=0,042)
				Left ventricular end systolic dimension (mm) (strength) 31,25 +/- 3,66 (p=0,004)
Forsythe et al., 2018	139	MIXED TYPE EXERCISE REGIMEN	Stroke Volume (ml) 92 ± 16 (p<0,001)	Mean Wall Thickness (mm) 9 ± 1 (p<0,001)
			LVM (g) 191 ± 31 (p<0,001)	Maximum wall thickness (mm) 10 ± 1 (p<0,001)
			LVEDV (ml) 157 ± 25 (p<0,001)	LVIDd (mm) 56 ± 4 (p<0,001)
			LVM index (g/(m^2)^2.7) 38 ± 7 (p<0,001)	LVIDd index (mm/(m^2)^0.5) 37±2 (p<0,001)
			LVEDV (ml/(m^2)^1.5) 48 ± 7 (p<0,001)	LVIDs (mm) 38 ± 3 (p<0,001)
			LVM index (g/m2) 87 ± 13 (p<0,001)	LVIDs index (mm/(m2) 26 ± 2 (p=0,017)
			LVESV (ml) 65 ± 13 (p<0,001)	
			LVESV (ml/(m^2)^1.5) 20 ± 4 (p<0,001)	
Lin et al., 2016	87	MIXED TYPE EXERCISE REGIMEN	LVM (g) (lineman) 265 ± 43 (p<0.001)	PWT (mm) (lineman) 11.0 ± 1.2 (p<0.001)
			LVM (g) (non-lineman) 217 ± 35 (p<0.001)	PWT (mm) (non lineman) 10.4 ± 1.1 (p=0,01)
			LVM index (gm/m^2) (lineman) 109 ± 15 (p<0.001)	IVS thickness (mm) (lineman) 10.6 ± 1.2 (p<0.001)
				IVS thickness (mm) (non lineman) 9.8 ± 1.4 (p=0,004)

			LVM index (gm/m ²) (non- lineman) 101 ± 15 (p<0.001)		Relative wall thickness (lineman) 0.40 ± 0.06 (p<0.001)	
					Interventricular septum (mm) (runners) 7.7 ± 1	
					Interventricular septum (mm) (rowers) 9.4 ± 1 (p<0.001)	
					Posterior wall (mm) (runners) 8.4 ± 1	RV basal diameter (mm) (runners)
					Posterior wall (mm) (rowers) 9.7 ± 1 (p<0.001)	45.1 ± 6
			Stroke volume (mL) (runners) 102 ± 13		Wall thickness (average) (mm) (runners) 8.1 ± 1	RV basal diameter (mm) (rowers) 45.1 ± 4
			Stroke volume (mL) (rowers) 100 ± 15		Wall thickness (average) (mm) (rowers) 9.5 ± 1 (p<0.001)	RV diastolic area (cm ²) (runners)
			LVM (g) (runners) 159 ± 20	Stroke volume index (mL/m ²) (runners) 57 ± 6	Wall thickness/BSA (mm/m ²) (runners) 4.5 ± 1	27.4 ± 4
			LVM (g) (rowers) 209 ± 26 (p<0.001)	Stroke volume index (mL/m ²) (rowers) 52 ± 8 (p<0.001)	Wall thickness/BSA (mm/m ²) (rowers) 4.9 ± 1 (p<0.001)	RV diastolic area (cm ²) (rowers) 26.2 ± 3
						Diastolic area/BSA (cm ² /m ²) (runners)
						14.7 ± 3
			LVM index (g/m ²) (runners) 88 ± 11	LVEDV (mL) (runners) 182 ± 21	Relative wall thickness (runners) 0.32 ± 0.04	Diastolic area/BSA (cm ² /m ²) (rowers)
					Relative wall thickness (rowers) 0.37 ± 0.04	13.5 ± 2
			LVEDV (mL) (rowers) 172 ± 25 (p<0.05)		LVIDd (mm) (runners) 50.3 ± 3	RV systolic area (cm ²) (runners) 15.7 ± 3
			LVM index (g/m ²) (runners) 108 ± 13 (p<0.001)	LVEDV index (mL/m ²) (runners) 101 ± 10	LVIDd (mm) (rowers) 52.4 ± 3 (p<0.001)	RV systolic area (cm ²) (rowers) 15.8 ± 3
					LVIDd/BSA (mm/m ²) (runners) 27.9 ± 2	± 3
					LVIDd/BSA (mm/m ²) (rowers) 27.1 ± 2 (p<0.01)	Systolic area/BSA (cm ² /m ²) (runners)
						8.7 ± 1
					LVIDs (mm) (runners) 34.4 ± 3	Systolic area/BSA (cm ² /m ²) (rowers)
					LVIDs (mm) (rowers) 35.5 ± 2 (p<0.05)	8.1 ± 2 p=NS
					LVIDs/BSA (mm/m ²) (runners) 19.1 ± 6,2	
					LVIDs/BSA (mm/m ²) (rowers) 18.3 ± 6,1 (p<0.001)	

Weiner et al., 2015	12	ENDURANCE		Stroke volume (mL) 138.1±20.4 (p<0.001)	LV longitudinal strain (%) -19.9±2.5 (p=0.04)	Cardiac output (L/min) 7.0±1.1 (p=0.5) LV peak Sm (cm/s) 8.9±1.3 (p<0.001)
Finocchiaro et al., 2017	1,083	ENDURANCE+ MIXED TYPE EXERCISE REGIMEN	LVM index (g/m2) 101+/-21 (p<0.001)			LVEDD (mm) 54+/-5 (p<0.001)
						LVEDD/BSA (mm/m) 27.2+/-2.7 (p<0.001)
						LVEDD/BSA (mm/m) 27.2+/-2.7 (p<0.001)
						IVS (mm) 10.0+/-1.3 (p<0.001)
						Maximal LVWT >12 mm: 26 (40) (p<0.001)
Giraldeau et al., 2015	315	STRENGTH+ MIXED TYPE EXERCISE REGIMEN	LVM (g) 159 +/- 26 (p<0,001)	Stroke volume (mL) 98 +/- 20 (p<0,001)	LV global longitudinal strain (GLS) (%) - 20,6 +/- 2,0 (p=0,02)	
				LVEDV (ml) 193 +/- 33 (p<0,001)		
				LVEDV index to BSA (ml/m^2) 94 +/- 1(p<0,001)	LV systolic strain rate (%/s) -1,06 +/- 0,18 (p=0,67)	RV area (cm^2) 29,7 +/- 3,9 (p<0,0001)
				LVM index (g/m^2) 77 +/- 10 (p<0,001)	LV diastolic early strain rate (%/s) 1,56 +/- 0,43 (p<0,01)	RV dimensions basal (cm) 4,3 +/- 0,5 (p<0,0001)
				LA volume (ml) 59,1 +/- 16,8 (p<0,001)		
				LA volume index BSA: 28,6 +/- 7,5 (p=0,54)		RV dimensions mid (cm) 3,6 +/- 0,5 (p<0,0001)
				LVM index to LBM: 2,2 +/- 0,3 (p=0,63)	RV global longitudinal strain (%) -24,7 +/- 2,6 (p=0,76)	RV longitudinal (cm) 9,1 +/- 0,9 (p<0,0001)
				LV mass to volume ration 0,83 +/- 0,11 (p=0,015)		
				RA volume (mL) 56,1 +/- 15,3 (p<0,0001)	RV systolic strain rate (%/s) -1,5 +/- 0,3 (p=0,58)	

				RA volume index BSA 27,2 +/- 6,7 (p<0,02)	RV diastolic early strain rate (%/s) 1,78 +/- 0,60 (p=0,043)		
					LA LG contraction(%) 10,8 +/- 3,8 (p=0,65)		
					RA LG contraction (%) - 15,2 +/- 4,2 (p=0,056)		
Agrebi et al., 2015	36	MIXED TYPE EXERCISE REGIMEN	LVM (g) 238,73 +/- 29,57 LVM index (g/m^2) 119,66 +/- 12,67	LV end diastolic diameter (mm) 54,80 +/- 4,18			
				LV end systolic diameter (mm) 35,10 +/- 1,11			RV diameter (mm) 18,65 +/- 4,74
				Posterior wall thickness (mm) 8,10 +/- 1,10			
				LV ejection time (msec) 279±38,14			
Roslan et al., 2023	100	ENDURAN CE+ MIXED TYPE EXERCISE REGIMEN	LVM index (g/m^2) 93,53 +/- 15,85 (p<0,001)	IVSd (cm) 0,95 +/- 0,12 (p=0,003)			
				PWTd (cm) 0,91 +/- 0,11 (p<0,001)			RV diameter (mm) 18,65 +/- 4,74
				GLS (%) 19,96 +/- 2,37 (p=0,16)	LVIDd (cm) 5,05 +/- 0,36 (p<0,001)		
				LVEDV index (mls/m^2) 67,36 +/- 14,75 (p<0,001)	LArS (%) 44,12 +/- 9,55 (p<0,001)	IVSs (cm) 1,27 +/- 0,17 (p=0,473)	
				LVESV index (mls/m^2) 27,84 +/- 8,14 (p<0,001)	LAcS (%) 32,53 +/- 6,45 (p=0,038)	PWTs (cm) 1,42 +/- 0,16 (p=0,086)	
					RVGLS (%) 21,04 +/- 2,77 (p=0,43)	LVIDs (cm) 3,4 +/- 0,39 (p<0,001)	
				RWT 0,36 +/-0,05 (p=0,705)			
				Huang et al., 2022	598	ENDURAN CE+ STRENGT H+ MIXED TYPE	
LVEDV index (ml/m^2) 75,7 +/- 13,5 (p<0,001)	LVGCS (%) -28,4 +/- 4,5 (p=0,778)	LVPWd (mm) 9,9 +/- 1,5 (p<0,001)					
	RVLS (%) -27,5 +/- 4,7 (p=0,358)	LVIDd index (mm/m^2) 26,9 +/- 2,6 (p;0,001)	RVESA index (cm^2/m^2) 7,7 +/- 1,5 (p<0,001)				

LVESV index (ml/m ²) 32,0 +/- 6,6 (p<0,001)	LVIDs index (mm/m ²) 16,7 +/- 1,7 (p<0,017)
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Table 3. Elite Athlete Echographic Findings Based on the Type of Sport.

Title	Patient s	Type of sport / Intensity	LEFT VENTRICUL AR MASS+ INDEX	VOLUMES	STRAIN	LEFT VENTRICULAR ECHO FINDINGS	RIGHT VENTRICULAR ECHO FINDINGS
Eun et al., 2016	29	MIXED TYPE EXERCISE REGIMEN				Ejection Fraction (%) 62.3±4.2 (p=ns)	
						Interventricular septal thickness (mm) 10.4±0.8 (p=0,01)	
						Posterior wall thickness (mm) 10.6±0.7 (p=0,001)	
			LV Mass (g) 216.7±21.9 (p=0,001)	Stroke volume (mL) 89.3±15.4 (p=0,01)	Longitudinal strain: -17.6+1.8 (p=ns)	End-diastolic diameter (mm) 53.1±0.7 (p=0,001)	RV Systolic Area (m2) 14.9±3.7 (p=0,005)
			Mass index (g/height2) 110.2±9.4 (p=0,001)	End-diastolic volume (mL/m2) 152.0±19.4 (p=0,001)		End-systolic diameter (mm) 36.5±1.8 (p=0,001)	RV Diastolic Area (m2) 26.1±7.1 (p=0,002)
				End-systolic volume (mL/m2) 62.7±12.7 (p=0,001)		Relative wall thickness 4.0±0.3 (p=ns)	
						Cardiac index (L/m2) 2.5±0.6 (p=ns)	
						End-systolic WS (mmHg) 240.9±32.6 (p=0,001)	
						End-systolic MWS (g/cm2) 21.5±1.5 (p=ns)	
			Cooke et al., 2018	11	ENDURAN CE		Stroke volume(ml) 87±14 (p< 0,01)
LV mass (g) 181 +/- 27 (p= 0,001)	Stroke volume index(ml m-2) 46±9 (p<0,01)					Peak torsion(degrees cm-1) 1.5±0.8 (p=0,0001)	
LV mass index 95 +/- 12 (p=0,001)	End-diastolic volume(ml) 148±20 (p<= 0,01)					LV shortening(Δcm) 1.9±0.5 (p=0,0001)	
		Sphericity index 1.7±0.1 (p=0,0001)					
	End-diastolic volume index(ml m-2) 78±12 (p<= 0,05)					End-diastolic LV length(cm) 9.3±0.7 (p=0,0001)	

			End-systolic volume(ml)61±16 (p=0,0001)	Cardiac output(L min-1) 4.3±0.8 (p=0,0001)	
			End-systolic volume index(ml m-2) 32±8 (p=0,01)	Cardiac index(L min-1 m-2) 2.3±0.5 (p=0,0001)	
Major et al., 2014	52	ENDURAN ACE	LV mass (g/m^3) 99,4 +/- 17,3 (p,0,001)	LV rel. Stroke Volume (ml/m^3) 36,6 +/- 8,9 (p<0,01)	rel.RVLADd (mm/m) 63.4 ± 6.3 (p<0,001)
					RVLA.Fr.Sh. (%) 25.9 ± 6.3 (p= NS)
					rel.RVSADd (mm/m) 27.3 ± 3.6 (p<0,001)
					rel.LVLADd (mm/m) 63.8 ± 5.6 (p<0,05)
					RVSA.Fr.Sh. (%) 20.3 ± 8.4 (p= NS)
Miranda et al., 2014	40	ENDURAN CE+ STRENGT H	Left ventricular mass index (g m^-2) (endurance) 79,25 +/- 20,75 (p=0,01)	Left ventricular mass index (g m^-2) (strength) 88,74 +/- 28,71 (p<0,01)	Left ventricular ejection fraction (%) (endurance) 62,70 +/- 6,34 (p=0,628)
					Left ventricular ejection fraction (%) (strength) 61,35 +/- 4,69 (p=0,163)
					Septum thickness (mm) (endurance) 8,40 +/- 1,60 (p=0,041)
					Septum thickness (mm) (strength) 9,75 +/- 1,86 (p=0,01)
					Posterior wall thickness (mm) (endurance) 8,50 +/- 1,43 (p=0,01)

				Left ventricular end systolic dimension (mm) (endurance) 30,20 +/- 3,25 (p=0,042)			
				Left ventricular end systolic dimension (mm) (strength) 31,25 +/- 3,66 (p=0,004)			
Agrebi et al., 2015	36	MIXED TYPE EXERCISE REGIMEN	LV mass (g)	LV end diastolic diameter (mm) 54,80 +/- 4,18			
			238,73 +/- 29,57	LV end systolic diameter (mm) 35,10 +/- 1,11			
			RV diameter (mm)	18,65 +/- 4,74			
			LV mass index (g/m^2)	Posterior wall thickness (mm) 8,10 +/- 1,10			
			119,66 +/- 12,67	LV ejection time (msec) 279±38.14			
Forsythe et al., 2018	139	MIXED TYPE EXERCISE REGIMEN	Stroke Volume (ml)		Mean Wall Thickness (mm) 9 ± 1 (p<0,001)		
			LV Mass (g)	92 ± 16 (p<0,001)	Maximum wall thickness (mm) 10 ± 1 (p<0,001)		
			191 ± 31 (p<0,001)	LVEDV (ml) 157 ± 25 (p<0,001)	LVIDd (mm) 56 ± 4 (p<0,001)		
			LV Mass index	LVEDV	LVIDd index (mm/(m^2)^0.5) 37±2 (p<0,001)		
			(g/(m^2)^2.7)	(ml/(m^2)^1.5) 48 ± 7 (p<0,001)	LVIDd index (mm/(m^2)^0.5) 37±2 (p<0,001)		
			38 ± 7 (p<0,001)	LVESV (ml) 65 ± 13 (p<0,001)	LVIDs (mm) 38 ± 3 (p<0,001)		
			LV mass index (g/m2)	LVEDV	LVIDs index (mm/(m2) 26 ± 2 (p=0,017)		
			87 ± 13 (p<0,001)	(ml/(m^2)^1.5) 20 ± 4 (p<0,001)			
Lakatos et al., 2020	138	MIXED TYPE EXERCISE REGIMEN	Stroke volume index (mL/m2) 49.3 ± 7.8 (p<0.05)		LV global longitudinal strain (%) -18.3 ± 1.8 (p<0.001)		RVEF (%) 53.1 ± 4.2 (p<0.001)
			LV mass index (g/m2)	LVEDV index(mL/m2) 89.0 ± 13.1 (p<0.001)	LVEF (%) 55.4 ± 3.5 (p<0.01)		
			98.7 ± 15.6 (p<0.001)	LVESV index (mL/m2) 39.7 ± 6.8 (p<0.001)	LV global contraction strain (%) -26.9 ± 2.4 (p<0.05)		

			RV Stroke volume index (ml/m2) 49.1 ± 6.6 (p<0.01)			
			RVEDV index (ml/m2) 92.9 ± 13.7 (p<0.001)			
			RVESVi index (ml/m2) 43.8 ± 9.0 (p<0.001)			
Lin et al., 2016	87	MIXED TYPE EXERCISE REGIMEN	LV mass (g) (lineman) 265 ± 43 (p<0.001)		PWT (mm) (lineman) 11.0 ± 1.2 (p<0.001)	
			LV mass (g) (non-lineman) 217 ± 35 (p<0.001)		PWT (mm) (non lineman) 10.4 ± 1.1 (p=0,01)	
			LV mass index (gm/m^2) (lineman) 109 ± 15 (p<0.001)		IVS thickness (mm) (lineman) 10.6 ± 1.2 (p<0.001)	
					IVS thickness (mm) (non lineman) 9.8 ± 1.4 (p=0,004)	
			LV mass index (gm/m^2) (non-lineman) 101 ± 15 (p<0.001)		Relative wall thickness (lineman) 0.40 ± 0.06 (p<0.001)	
Hammami et al., 2021	20	STRENGT H			LVEF(%) (WD) 59,33 ± 7,23 (ITF) 63,06 ± 7,40	
					LVEDD (ml) (WD) 124,63 ± 32,70 (ITF) 117,76 ± 9,65	
					RVEDD (mm) (WD) 48,94 ± 4,35 (ITF) 47,59 ± 2,86	
					LVESD (ml) (WD) 51,55 ± 12,29 (ITF) 50,57 ± 10,26	
					RVESD (mm) (WD) 28,38 ± 3,39 (ITF) 30,00 ± 2,07	
Wasfy et al., 2014	71	ENDURAN CE			IST (mm) (WD) 7,07 ± 0,62 (ITF) 8,19 ± 1,37 (p<0,05)	
					RWL (mm) (WD) 65,35 ± 8,51 (ITF) 64,37 ± 3,44	
					PWIVS (mm) (WD) 6,85 ± 0,62 (ITF) 7,43 ± 0,74	
Wasfy et al., 2014	71	ENDURAN CE	LV mass (g) (rowers) 209 +/- 26 (p<0,001)		RV basal diameter (mm) (runners) 45.1+- 6	
			Stroke volume (mL) (runners) 102 +/- 13		RV basal diameter (mm) (rowers) 45.1 +- 4	

				Stroke volume (mL) (rowers) 100 +/- 15	Interventricular septum (mm) (rowers) 9.4 +_ 1 (p<0,001)	RV diastolic area (cm2) (runners) 27.4+- 4
				Stroke volume index (mL/m^2) (runners) 57 +/- 6	Posterior wall (mm) (runners) 8.4 +-1	RV diastolic area (cm2) (rowers) 26.2 +-3
				Stroke volume index (mL/m^2) (rowers) 52 +/- 8 (p<0,001)	Posterior wall (mm) (rowers) 9.7 +_1 (p<0,001)	Diastolic area/BSA (cm2/m2) (runners) 14.7 +-3
				Stroke volume index (mL/m^2) (rowers) 52 +/- 8 (p<0,001)	Wall thickness (average) (mm) (runners) 8.1 +- 1	Diastolic area/BSA (cm2/m2) (rowers) 13.5 +-2
				Stroke volume index (mL/m^2) (rowers) 52 +/- 8 (p<0,001)	Wall thickness (average) (mm) (rowers) 9.5 +-1 (p<0,001)	RV systolic area (cm2) (runners) 15.7 +_3
				Stroke volume index (mL/m^2) (rowers) 52 +/- 8 (p<0,001)	Wall thickness/BSA (mm/m2) (runners) 4.5 +_ 1	RV systolic area (cm2) (rowers) 15.8 +_3
				Stroke volume index (mL/m^2) (rowers) 52 +/- 8 (p<0,001)	Wall thickness/BSA (mm/m2) (rowers) 4.9 +- (p<0,001)	Systolic area/BSA (cm2/m2) (runners) 8.7 +_1
				Stroke volume index (mL/m2) (runners) 101 +- 10	Relative wall thickness (runners) 0.32 +_0.04	Systolic area/BSA (cm2/m2) (rowers) 8.1 +_2 p=NS
				Stroke volume index (mL/m2) (runners) 101 +- 10	Relative wall thickness (rowers) 0.37 +_0.04	
				Stroke volume index (mL/m2) (rowers) 89 +/- 13 (p<0,001)	LVIDd (mm) (runners) 50.3 +/- 3	
					LVIDd (mm) (rowers) 52.4 +/- 3 (p<0,001)	
					LVIDd/BSA (mm/m2) (runners) 27.9 +/- 2	
					LVIDd/BSA (mm/m2) (rowers) 27.1 +/- 2 (p<0,01)	
					LVIDs (mm) (runners) 34.4 +/- 3	
					LVIDs (mm) (rowers) 35.5 +/- 2 (p<0,05)	
					LVIDs/BSA (mm/m2) (runners)19.1 +/- 6,2	
					LVIDs/BSA (mm/m2) (rowers) 18.3 +/- 6,1 (p<0,001)	
					LV ejection fraction (%) 63.3±2.9 (p=0.23)	
Weiner et al., 2015	12	ENDURAN CE	Stroke volume (mL) 138.1±20.4 (p<0.001)	LV longitudinal strain (%) -19.9±2.5 (p=0.04)	Cardiac output (L/min) 7.0±1.1 (p=0.5)	
					LV peak Sm (cm/s) 8.9±1.3 (p<0.001)	

3.1. *Electrocardiographic Modifications*

ECG modifications appear in athletes due to the adaptive cardiac remodelling that happens as a response to chronic exposure to physical exercise. The benefit of this screening method is that it's widely available and cheap. Still, one of the big disadvantages is that the ECG gives us little information compared to other imaging techniques (unlike echocardiography or cardiac magnetic resonance - CMR)[27,28].

Out of the 17 original studies that were eligible for this review, four studies showed relevant data regarding ECG findings[12,14,16,17]. Two studies reported that male athletes met the diagnostic criteria for LVH more frequently[14,17]; one recorded RVH as less common than LVH[17]. One study showed that the duration of the PR interval increases with age but remains within normal limits[16]. Finocchiaro et al.[14] found that 4% of the participants presented a left-axis deviation, and 33% of the male athletes they studied had a QRS duration >100ms. In three of these studies, T wave inversion was recorded[12,14,17], and two of these studies showed that the female athletes had a T wave inversion in the anterior precordial leads more frequently than the male athletes, who usually had T wave inversion in the inferior leads[16,17]. In all studies, neither female nor male athletes presented depressed ST segments.

According to Drezner et al. 2017 [29], when it comes to athletes, there are normal ECG findings, borderline ECG findings (not necessarily pathological, but the presence of 2 or more borderline findings may suggest a pathological process), and abnormal ECG findings. Among the normal ECG findings in athletes, we mention increased QRS voltage, incomplete right bundle branch block, early repolarisation, first-degree atrioventricular block, and sinus bradycardia, all of which were present in the five studies. Borderline ECG findings, like left axis deviation, were rarely present. Abnormal ECG findings, from which we mention T wave inversion in the anterior and inferior leads, were present in some studies [29].

3.2. *Imagistic Findings*

CMR (cardiac magnetic resonance) is a non-invasive imaging technique designed to assess cardiovascular morphology, function, perfusion, and flow quantification [30]. CMR is considered the best method of evaluating the contractile function of the heart, with many well-known advantages, like excellent soft-tissue contrast, the use of a magnetic field and radio waves instead of ionising radiation, and a high spatial resolution being just a few of them. Like any imaging method, CMR presents a set of disadvantages, from which we take note of the high cost of the procedure, making it not nearly as accessible as other imaging techniques (like echocardiography) [31,32].

Out of the 17 eligible studies for this review, only one used CMR (cardiac magnetic resonance). The study compared CMR findings in both males and females, as well as differences between types of sport. Overall, all participants had a normal LVEF. Male athletes had higher LV and RV volumes compared to female athletes. Male athletes had lower LVEDV/LVM and RVEDV/RVM ratios (created specifically to measure the relation between ventricular hypertrophy and ventricular dilatation), suggesting a more noticeable hypertrophy. Male athletes had a higher RVEDV/LVEDV ratio compared to female athletes. Participants who practice endurance sports showed a lower LVEDV/LVM ratio than the rest of the groups [18].

3.3. *Differences Between the Type of Sport and Echocardiographic Findings*

Sports can be divided into two big categories: dynamic (isotonic) sports, with changes in muscle length and a small intramuscular force, and static (isometric) sports, with little to no change in muscle length but with a large intramuscular force [33]. Exercise can also be classified by the type of muscle metabolism in aerobic and anaerobic exercise. Usually, high-intensity static exercise is characterized by an anaerobic metabolism, while high-intensity dynamic exercise is characterized by an aerobic metabolism. Sports with a high dynamic demand are called endurance sports, and sports with a high static demand are called strength sports. Dynamic exercise will lead to greater oxygen consumption, higher systolic blood pressure, an increase in stroke volume, and a decrease in peripheral resistance. In contrast, static exercise won't present any changes in stroke volume but will present an increase in

oxygen consumption. Usually, athletes participating in high-intensity dynamic sports will have eccentric hypertrophy, and athletes training in high-intensity static sports will have a concentric type of ventricular hypertrophy [33].

In the 17 studies that have been selected, there were evaluated athletes from 3 categories of sports: endurance sports, mixed sports, and strength sports. From the studies we identified, endurance sports (swimming, triathlons, long-distance running, etc) were the most commonly practised among athletes. All studies showed that the resting heart rate for athletes who practised endurance sports was lower than their peers who practised strength-oriented sports, and only one study evaluated the systolic blood pressure, which was higher in the second group. Several studies proved that in endurance sports, the LV wall thickness and the LV mass are greater than in strength sports.

3.4. Racial Differences

Research showed an important difference in the heart's electrical function between races, more precisely between Caucasian and Black Athletes. The difference was evidenced using electrocardiography (ECG) [7]. One ECG parameter that stood out as an important modification between the two races was the T wave inversion (TWI). This phenomenon appeared more frequently in the African population, which was between 2.6% and 22.8% in black athletes, compared to 0% to 5% in Caucasian athletes [34-40]. T wave inversion occurred in multiple leads depending on the studies investigated, ranging from anterior (V2) to inferior (DII, aVF) and lateral (V5, V6, DI, aVL), all showing a higher prevalence in the African group compared to the Caucasian one [40]. Moreover, ST elevation was also a parameter more frequently present in Africans than in Caucasians. Ranging from 11% to 85% in Africans compared to 1% to 61% in Caucasians, being more present in the lateral and anterior leads [34-36,40-42]. Other ECG alterations were more readily found in the African population than the Caucasians, such as early repolarisation [40,43-46] and left ventricular hypertrophy [34-37,40].

With all these differences, it is important to conclusively note that cardiac adaptation can be evidenced through so-called ECG "normal findings" in African athletes. As per the Seattle criteria, these findings include convex ST segment elevation with T wave inversion in leads V1-V4 [40,47].

4. Where Adaptation Ends and Pathology Begins

Athlete's heart is a physiological cardiac adaptation characterised by cardiac hypertrophy, with normal cardiac morphology and a normal or enhanced cardiac function. Pathological cardiac hypertrophy is characterised by cardiac dysfunction that can progress toward heart failure, cellular death, and interstitial fibrosis [48,49]. Cardiac hypertrophy can be classified according to the geometry of the heart into eccentric and concentric hypertrophy, which can be either pathological or physiological. Concentric cardiac hypertrophy is characterized by increased cardiac mass and wall thickness, rarely with chamber volume changes. In contrast, eccentric cardiac hypertrophy is characterized by increased cardiac mass and chamber volumes, usually with normal wall thickness [48].

Even though the athlete's heart is an adaptive response to prolonged exercise, without pathological implications, there have been recorded some cases of sudden cardiac death (SCD) or sudden cardiac arrest (SCA), with an incidence of 21.7 per 1 million per year [50,51]. Many of the athletes under the age of 35 who suffered from SCD or SCA had an underlying congenital cardiac condition, the most common being hypertrophic cardiomyopathy (HCM) [51]. Therefore, this calls for a careful examination of athletes, starting from a simple ECG and looking into other imaging modalities depending on the presence of any abnormal findings. The first investigation should be a 12-lead ECG alongside a detailed personal and family history. If the ECG shows two or more abnormal ECG findings (T wave inversion, ST depression, pathological Q waves, complete left bundle branch block, third-degree atrioventricular block, etc.) [5], the next investigation should be a full standard echocardiography because even though ECG can sometimes detect an underlying pathology that might lead to SCD/SCA, it has a low sensitivity and specificity for this [29,30].

5. Brief Discussion on Doping and Effects of Anabolic Drugs and How They May Alter the Athlete's Heart

Doping refers to the use of certain substances to increase the athlete's performance, and it is a widespread problem in sports nowadays. The World Anti-Doping Agency (WADA) was founded as a way to reduce the use of banned substances, also called performance-enhancing drugs, in sports, as they would give the athlete unfair advantages during competitions and impose great health risks, especially cardiovascular risks. Even if these drugs have been strictly regulated over the years when it comes to performance athletes, it doesn't mean that they aren't still used by some performance athletes or by people practising casual sports [52].

To help athletes stay up to date with the banned substances, WADA created a list that's updated yearly, including what substances or methods are prohibited and when. The Prohibited List [53] comprises three big categories: substances and methods prohibited at all times, substances and methods banned only in competition, and substances and methods prohibited in certain sports. In the first category are included:

- 1) anabolic agents (anabolic androgenic steroids and other anabolic agents),
- 2) peptide factors, growth factors, and related substances (erythropoietin and agents affecting erythropoiesis, testosterone stimulating peptides, etc.),
- 3) beta-2 agonists (salbutamol, salmeterol, vilanterol, etc. but with some exceptions),
- 4) diuretics and masking agents, 5. hormone and metabolic modulators (anti-estrogenic substances, aromatase inhibitors, etc.),
- 5) gene and cell doping
- 6) manipulation of blood and blood components.

In competition, the following substances are prohibited: 1. stimulants (amfetamine, cocaine, etc), 2. narcotics (morphine, methadone, tramadol, etc.), 3. cannabinoids, 4. glucocorticoids, and 5. beta-blockers [53]. Anabolic androgenic steroids (AAS) are commonly used in sports to increase skeletal muscle mass, therefore boosting the athlete's performance [54]. AAS use has been associated with cardiovascular effects like hypertension, arrhythmias, pathological cardiac hypertrophy (concentric), cardiac fibrosis, dyslipidemia, and sudden cardiac death [55]. According to Hassan et al., 2009 in some cases of SCD/SCA, myocardial infarction occurs without any signs of atherosclerosis or thrombosis, which might mean that AAS can induce coronary vasospasm and cardiac arrhythmias, depending on the individual [54]. Another form of doping is represented by blood doping, which is defined as using substances or certain methods to increase oxygen delivery to muscles to achieve better athletic performance. Ways of blood doping include blood transfusions, administration of erythropoietin (rhEpo) or other erythropoiesis-stimulating agents (ESAs), and gene manipulation [56]. The main problem with this kind of doping is the rise of red blood cell (RBC) numbers, leading to a higher Hct and hyperviscosity syndrome [55,56]. RhEpo has no significant direct effects on the heart, but it seems like high blood pressure and thrombus formation are to blame for the negative effects of blood doping.

Exercise-induced cardiac remodelling seems to manifest slightly differently in female athletes than in male athletes. Recent research has shown that sex has an impact on cardiac adaptation to exercise, Finocchiaro et al. found in a study with more than 1000 highly trained athletes that the Sokolov-Lyon(SL) criteria for LVH were present in 14% of females in comparison to 42% of males, in addition, females had less left axis deviation [14], anterior T wave inversion (TWI) is more common in female [57], these interesting ECG findings highlight the need for further sex-based interpretation. Pelliccia et al. in one of the biggest studies on female athletes showed that females were rarely to have significant LV cavity enlargement (LVEDD >54mm) and LV wall thickness >12 mm [58]. Females develop eccentric hypertrophy more frequently than males [14].

6. Conclusions

The athlete's heart has a wide array of modifications resulting from many factors, such as race, gender, type of sport, and, in some cases, even subtypes of the same sport.

In terms of electrical changes, the most prevalent ones are seen amongst the African population vs. the Caucasian one, with the former having a higher frequency of inverted T waves and ST changes. The other notable changes are the "borderline" or "grey area" ones in which certain parameters such as PR duration or QRS duration are elongated but not to a clearly diagnosable value.

In terms of structural changes, the one that responds the most to all the factors that surmise the athlete's heart is the indexed left ventricular mass that differs between non-athletes and athletes and between types of sport and gender. Other relevant parameters, such as Left Ventricular Wall Thickness and Ventricular Chamber Diameters are predominantly seen in certain situations, such as wall thickness appearing more often in endurance athletes and ventricular chamber diameters changing almost exclusively in males.

7. Limitations

The primary limitations our systematic review has encountered are related to the type of studies included and the number of patients in said studies. Given the nature of the subject we reviewed, a prospective study would be highly impractical, as evidenced by the lack of such studies in the literature. Another significant limitation is the number of athletes included, which is once again constrained by the nature of the researched subject, as there is only a limited number of elite athletes in a country at any given time.

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