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Cardiovascular Adaptation to Self-Reported Physical Exercise Among Subjects Without Known Cardiovascular Disease: The Contribution of the Right Ventricle

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Abstract

Aim: Assess the effects of mild leisure-time versus vigorous long-term physical activity (PA) on left (LV) and right ventricle (RV) function and structure in healthy subject.

Methods: The study was conducted on 360 subjects, 120 sedentary, 120 leisure-time mild exercisers and 120 recreational athletes. They underwent standardized transthoracic echocardiography and carotid artery stiffness assessment with an echo-tracking ultrasound system.

Results: Left ventricular (LV) and RV dimensions were significantly higher in the athletes group. Left and right ventricular diastolic function significantly improved in the leisure-time mild PA group (LV: sedentary group vs leisure-time mild PA $p<0.0001$. RV: sedentary group vs leisure-time mild PA $p=0.025$). There were no differences among groups for LV systolic function, while tricuspid annular plane systolic excursion (TAPSE) increased according to PA intensity after adjustment by age and sex ($p=0.004$).Pulmonary artery systolic pressure (PAPS) was similar among groups. When indexed by stroke volume (SV), PAPS significantly decreased in the recreational athletes (recreational athletes vs. leisure-time mild exercisers and control $p<0.005$). Carotid stiffness and HDL-cholesterol improved according to PA ($p<0.001$).

Conclusion: In normal subjects, PA has a positive effect on cardiac adaptation driven mainly through higher levels of exercise; even mild levels of PA can improve RV longitudinal contraction (TAPSE) and diastolic function.

Keywords: Sport Activity; Transthoracic Echocardiography; Carotid Ultrasound; Cardiovascular Adaptation; Right Ventricle

Abbreviations: PA: Physical Activity; CO: Cardiac Output; RV: Right Ventricular; LV: Left Ventricle; CMR: Cardiac Magnetic Resonance; BP: Blood Pressure; HR: Heart Rate; MAP: Mean Arterial Pressure; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; EACVI: European Association of Cardiovascular Imaging; TDI: Tissue Doppler Imaging; TRV: Tricuspid Regurgitation Velocity; PASP: Pulmonary Artery Systolic Pressure; RAP: Right Atrial Pressure; TAPSE: Tricuspid Annular Plane Systolic Excursion; PVR: Pulmonary Vascular Resistance; PWV: Pulse Wave Velocity; BMI: Body Mass Index; HDL: High Density Lipoprotein; LVMI: Left Ventricular Mass Index; LVIDD: Left Ventricular Internal Diameter in Diastole; SV: Stroke Volume; LVEF: Left Ventricular Ejection Fraction; EES: Ventricular Elastance; EA: Arterial Elastance; SRV: Lateral Tricuspid Annulus Peak Systolic Velocity; RA: Right Atrium.

Introduction

The effect of physical activity (PA) on cardiovascular structure and function has been extensively studied [1-3]. The impact of PA on the heart is characterised by biventricular and atrial enlargement to increase the cardiac output (CO) and meet the muscle's oxygen request. Because of the low resistance and high compliance of the pulmonary circulation, the right ventricle (RV)

has an excellent adaptability to physical stress, with RV mass and wall thickness approximately one-quarter that of the left ventricle (LV). The RV adaptation during short-time intense exercise is characterised by a proportional increase in RV contractility and pulmonary pressure that results in adequate ventricular-arterial coupling. However, prolonged intense exercise causes extreme RV wall stress there and RV dysfunction, increasing pulmonary pressure due to raised left atrial and left ventricular diastolic pressure. Regular vigorous exercise causes cardiac remodeling, with the RV being more affected than the LV.

Endurance athletes showed greater RV than LV volume, highlighting that RV loading may cause greater RV remodeling [4,5]. Moreover, chronic high-intensity training may induce permanent RV damage with reduced systolic function and/or precipitate subclinical RV diseases, such as arrhythmogenic RV cardiomyopathy (ARVC) in predisposed subjects [6]**.** All these scenarios have been described in elite athletes, but less is known about the effect of physical exercise on the general population who practice mild to moderate PA. The MESA study shed some light on this topic [7]. In this community-based population study, the ventricle dimensions were studied using cardiac magnetic resonance (CMR), which showed that the RV mass size was proportional to the amount of reported exercise and independent of LV adaptation. The present study aimed to evaluate the effect of self-reported PA on cardiovascular changes focusing on RV adaptation in a group of subjects without known cardiovascular disease, studied by conventional transthoracic echocardiography and local carotid arterial stiffness.

Study Population

Participants were selected from healthy subjects such as blood donors, third age university members and subjects who underwent cardiology evaluation, ECG, and standard echocardiogram for eligibility for sports activity. Subjects diagnosed with diabetes mellitus, hypertension, nephropathy, cardiovascular disease including valve diseases or any other condition requiring chronic medications were excluded. From the original dataset of 900 healthy subjects, PA was assessed using a standardized questionnaire [8,9]. Among these, 120 recreational athletes engaged in aerobic competitive sports, 120 subjects of similar age who performed leisure-time mild aerobic PA for at least two years and 120 sedentary subjects were considered.

Subjects were categorized as sedentary (class 0) if they did not perform any PA; leisure-time mild exercisers (class 1) if they performed leisure physical activities (running, jogging, cycling, swimming, soccer, tennis, etc.) at least once a week without participating in competitions and exercisers or recreational athletes (class 2) if they performed aerobic sports and participated in competitions requiring a sports activity eligibility check-up. Office brachial blood pressure (BP) and heart rate (HR) were measured twice in the supine position, 5 minutes apart, in a quiet room using a semi-automatic sphygmomanometer [10,11]. Mean arterial pressure (MAP) was obtained by adding the systolic blood pressure (SBP) to the doubled diastolic blood pressure (DBP) and dividing the composite sum by three.

Transthoracic Echocardiogram (TTE)

A standardized TTE examination under continuous ECG recording was performed (Alfa 10; Aloka Co, Ltd, Tokyo, Japan), according to the American Society of Echocardiography/ European Association of Cardiovascular Imaging (EACVI) recommendations [12,13]. All studies were reviewed and analyzed offline with an image processing workstation implemented by the software COMPACS (Rev. 10.5.8, Mediatic, Genoa, Italy). Each parameter was assessed in 3 to 5 consecutive cardiac cycles, and the corresponding mean values were recorded.

Left Ventricle

The LV mass was calculated according to the Penn convention [14]. From the apical four-chamber pulsed Doppler LV mitral valve, the diastolic inflow was recorded to measure peak E-wave velocity, A-wave velocity and E/A ratio was derived. Pulse tissue Doppler imaging (TDI) was performed in four-chamber view at the septal and lateral mitral annular level and at the lateral tricuspid annulus. Peak myocardial wave velocity during systole (Sm), early diastole (Em), and late diastole (Am) (cm/s) were measured. E/Em was the ratio between transmittal E-wave and early diastole wave velocity Em on TDI [15]. Arterial elastance (Ea) and left ventricular end-systolic elastance (Ees) were used to evaluate the left ventricular-arterial coupling or cardiac length–force relationship, as the interaction between the contractile state of the LV (represented by Eas) and the systemic arterial system (Ea). Ea was calculated as [(SBP × 0.9)/SV], where SBP is brachial systolic BP, and SV is stroke volume [16]. Ees was derived from the so-called single-beat method developed by Chen et al. [17].

Right Ventricle

The basal, mid, and longitudinal RV diameters (mm) at the end diastole were measured from an RV – focused four-chamber apical view. Peak tricuspid regurgitation velocity (TRV) was calculated from multiple views, and the highest TRV was used to calculate RV systolic pressure. The pulmonary artery systolic pressure (PASP) estimation was based on the simplified Bernoulli equation, with the addition of estimated right atrial pressure (RAP) based on IVC size and collapse [18]. The PAPS was indexed by SV to remove the effect of RV pre-load. Tricuspid annular plane systolic excursion (TAPSE) was measured from the four-chamber views by placing M-mode through the tricuspid annulus, measuring the excursion distance between end-diastole and end-systole (in mm) with optimal image orientation and alignment to avoid underestimation.

The TAPSE/PAPS ratio describes the cardiac length–force relationship and gives insight into the contractile state of the RV and its interaction with the pulmonary circulation [19-21]. RV diastolic function was obtained from the apical 4-chamber view, with the Doppler beam aligned parallel to the RV inflow. The

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sample volume was placed at the tips of the tricuspid leaflets, and measures were taken at held end-expiration. Peak E wave, A wave and E/A ratio were obtained [22]. Pulmonary vascular resistance (PVR) was measured as [(TRV/RVOTVTI) *10)0.16] [23]. The average of five consecutive beats was considered.

Local Arterial Stiffness

The procedure used to assess local carotid stiffness was previously published [24,25] and local carotid pulse wave velocity (PWV) was considered.

Statistical Analysis

Within the 900 healthy subjects, 360 were selected and divided into three groups of 120 subjects each, best matched by sex and age. Since the group of leisure time and mild physical activity were similar regarding CV adaptation, they were combined. Data are expressed as mean ± SD. ANOVA was used to compare continuous variables within the groups, and post hoc analysis was used to test the differences between groups. Differences were adjusted for age and sex). PASP was adjusted by LV E/E`. Univariate and multivariate regression analyses were performed to test the independent association between the dependent variables and age, sex, MBP, local carotid PVW, LVEF, SV and sports activity. p values < 0.05 were considered statistically significant. Data were analyzed using SPSS (Rel. 11.0, 2002, SPSS Inc., Chicago, IL, USA).

Results

General characteristics. The general characteristics of the 360 healthy subjects studied (80% males, 89% non-smokers), divided into three groups, are reported in (Table 1). Specifically, waistline (p=0.0001), DBP (p=0.002) and HR (p<0.0001), systolic BP (p=0.023), HDL-cholesterol (p<0.001) and triglycerides (p<0.0001) were inversely related to the intensity of PA also after adjustment for age and sex. HDL-cholesterol increased according to the intensity of PA. Leisure-time mild aerobic exercise significantly affected waistline, triglycerides, and HR (Table 1). Echocardiography. LV parameters are reported in (Table 2) and (Figure 1). All the parameters considered showed a significant positive relation with physical exercise intensity, driven by group 2, except LFEF, ventricular and arterial elastance (Ees) and ventricular-arterial coupling (Ees/Ea), which did not change significantly among groups.

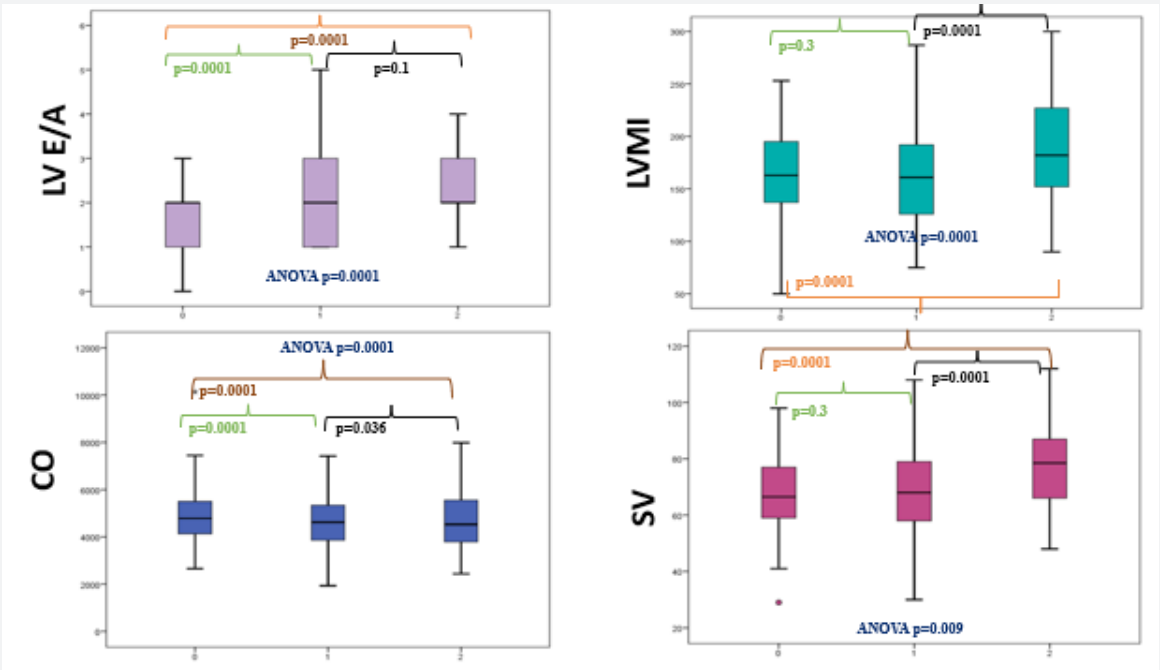

Figure 1: Dark blue: ANOVA between the 3 groups of sport, orange: group 0 (sedentary) vs group 2 (recreational athletes), green: group 0 vs group 1 (leisure-time exerciser), black: group (leisure-time exerciser) 1 vs group 2 (recreational athletes).

LV E/A: Left Ventricle Doppler Inflow Across the Mitral Valve.

LVMI: Left Ventricle Mass Indexed to Body Surface Area.

CO: Cardiac Output.

SV: Stroke Volume.

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Table 1: Baseline Characteristics of the Population Studied.

Variable (mean±SE)	Physical activity group 0 =120	Physical activity group 1=120	Physical activity group 2 =120	ANOVA	ANOVA (age and sex)	Gr 0 vs 1 (Adj age, sex)	Gr 1 vs 2 (Adj age, sex)	Gr 0 vs 2 Adj age, sex)
Age	33.9±13.1	31.8±12.7	29.6±14.1	0.042	-	0.2*	0.1*	0.01*
Weight (Kg)	74.5±15.9	71.8±14.0	71.2±14.1	0.16	0.0001	0.058	0.7	0.023
BMI	24.3±3.7	23.7±3.3	22.8±2.7	0.002	0.0001	0.1	0.01	0.0001
Waist line (cm)	89.5±11.7	86.1±10.0	81.4±7.9	0.0001	0.0001	0.002	0.0001	0.0001
SBP (mmHg)	123.4±12.6	124.4±13.5	122.9±12.9	0.6	0.015	0.5	0.3	0.7
DBP (mmHg)	75.5±10.2	73.8±10.3	72.2±9.8	0.05	0.002	0.1	0.1	0.007
HR (bpm)	73.5±12.5	68.4±11.0	61.2±13.9	0.0001	0.0001	0.001	0.0001	0.0001
Triglic (mg/dl)	116.9±69.1	102.9±50.5	65.7±23.8	0.0001	0.0001	0.09	0.0001	0.0001
Chol-HDL (mg/dl)	51.4±13.0	58.4±17.9	61.1±13.0	0.001	0.001	0.008	0.2	0.001

BMI: Body Mass Index

SBP: Systolic Blood Pressure

DBP: Diastolic Blood Pressure

HR: Heart Rate

HDL: High-Density Lipoprotein

*No adjusted by age.

Table 2: Left Atrium and Ventricle Dimension and Function.

Variable (mean±SE)	Physical activity group 0 =120	Physical activity group 1=120	Physical activity group 2 =120	ANOVA	ANOVA (age, sex)	Gr 0 vs 1 (age, sex)	Gr 1 vs 2 (age, sex)	Gr 0 vs 2 (age, sex)
Left atrium (mm)	33.3±5.8	34.6±5.0	34.8±5.0	0.09	0.009	0.09	0.4	0.015
Left atrium volume (ml)	42.5±13.8	45.2±15.3	49.1±17.7	0.005	0.001	0.1	0.045	0.001
LVMi (g/m2)	86.1±16.8	88.7±20.8	103.3±29.5	0.0001	0.0001	0.3	0.0001	0.0001
LVIDD (mm)	50.3±4.9	50.5±4.6	52.8±4.7	0.0001	0.008	0.6	0.0001	0.0001
IVSD (mm)	8.06±1.08	8.0±1.1	8.5±1.3	0.005	0.0001	0.6	0.001	0.004
E/A	1.79±0.8	2.10±0.9	2.2±0.9	0.0001	0.0001	0.0001	0.1	0.0001
E/E`	5.4±1.4	5.7±1.5	6.1±1.6	0.004	0.15	0.3	0.09	0.002
SV (ml)	67.7±12.8	69.1±16.3	77.7±14.8	0.0001	0.009	0.3	0.0001	0.0001
CO (ml/min)	4948±1192	4714±1335	4742±1383	0.3	0.0001	0.0001	0.036	0.0001
LVEF (%)	63.3±6.8	62.9±5.9	61.7±6.5	0.1	0.15	0.8	0.2	0.1
Ees (mmHg/ml)	1.98±0.79	2.08±0.72	1.90±0.55	0.3	0.08	0.3	0.09	0.4
Ea (mmHg/ml)	1.70±0.47	1.73±0.54	1.38±0.49	0.0001	0.0001	0.5	0.0001	0.0001
Ea/Ees	1.01±0.09	0.97±0.23	0.99±0.12	0.1	0.86	0.1	0.4	0.3

LVMI: Left Ventricular Mass Index.

LVIDD: Left Ventricular Internal Diameter in Diastole.

E/A: Early Filling/ atrial contraction ratio from mitral inflow doppler signal.

SV: Stroke Volume.

CO: Cardiac Output.

LVEF: Left Ventricular Ejection Fraction.

EES: Ventricular Elastance.

EA: Arterial Elastance.

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LV diastolic function, represented by E/A ratio (p<0.0001), was statistically different between sedentary and leisure-time mild activity groups but similar between groups 1 and 2. Right ventricular diameters in diastole were proportional to PA intensity, driven by the group with higher exercise intensity (Table 3 and Figure 2). TAPSE, a surrogate of RV free wall longitudinal contraction (p<0.0001), was positively related to PA. However, the RV S' wave, a parameter that represents the longitudinal contraction of the basal segment of the RV, did not differ among groups. PAPS did not differ across the three groups, but when it was indexed by SV (indexed by flow), there was a significant decrease in group 2.

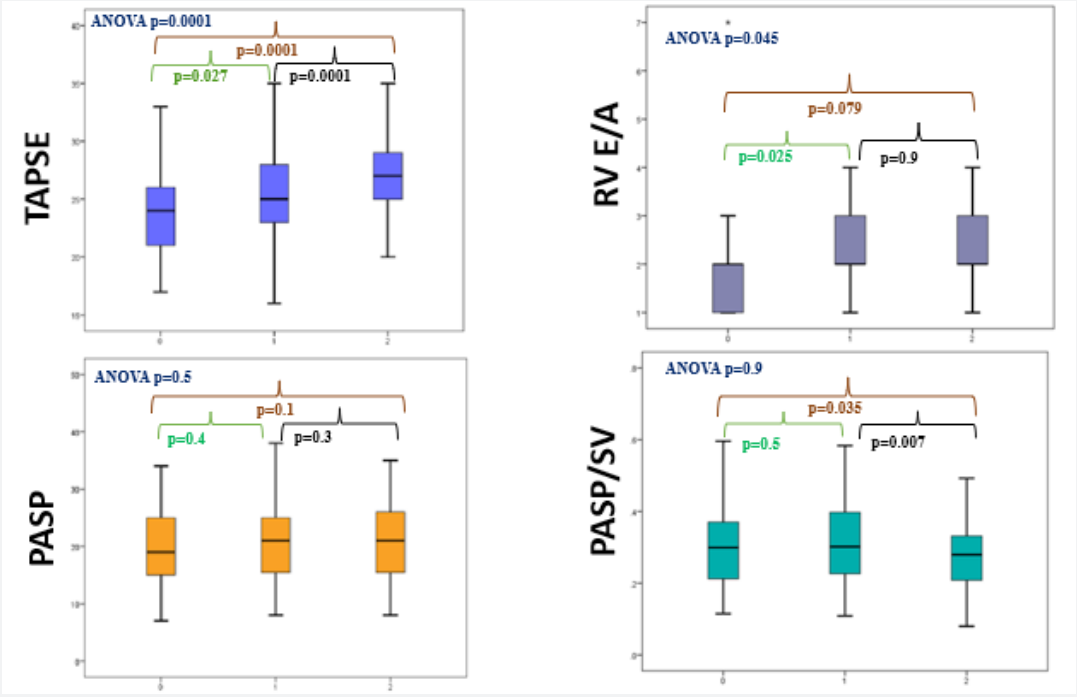

Figure 2: Dark blue: ANOVA between the 3 groups of sport, orange: group 0 (sedentary) vs group 2 (recreational athletes), green: group 0 vs group 1 (leisure-time exerciser), black: group (leisure-time exerciser) 1 vs group 2 (recreational athletes).

TAPSE: Tricuspid Annular Plane Systolic Excursion.

RV E/A: Right Ventricle Inflow Across the Tricuspid Valve.

PASP: Pulmonary Artery Systolic Pressure.

SV: Stroke Volume.

No differences in PVR were present among the groups. RV diastolic function (represented by E/A ratio) was positively related to the intensity of physical exercise (p<0.01). RV diastolic function differed significantly between sedentary and leisure-time mild PA groups. Univariate and multivariate analyses were performed for those dependent variables that showed the most considerable differences between-groups (Table 4). In the multiple-regression analysis, PA was an independent predictor of TAPSE, along with SV. At the same time, RV longitudinal diameter was affected by age, sex, and SV (similar results for the other RV diameters). The variables that explained the RV A/E ratio were sex, carotid PWV and HR. The independent variables associated with TAPSE/SV were age, sex, MAP, and PA. Physical activity, in addition to age, sex, SV and HR, were independent predictors of LVM, while age, sex, LVEF and HR were predictor of LV E/A ratio.

Discussion

The effects of PA on cardiac structure [26,27] and cardiovascular risk factors [28,29] are well known. Regular exercise reduces the risk of cardiovascular events and mortality [30,31] although some studies reported a U-shaped association between PA and mortality [32]. High intensity physical exercise leads to heart remodeling commonly referred to as athlete's heart [33]. Also, less intense PA can increase biventricular volumes and determine vascular changes [34] but less is known about heart remodeling in mild PA, particularly the effects on the RV.

The main findings of our study about the effects of PA in a cohort of apparently healthy subjects are:

1) The RV structural changes were proportional to the

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amount of PA mainly driven by the high intensity group. TAPSE was positively associated with the amount of exercise. PAPS and RV-arterial coupling did not change across groups while PAPS/SV was significantly lower in the most active group.

Table 3: Right Ventricular parameters.

Variable (mean±SE)	Physical activity group 0 =120	Physical activity group 1=120	Physical activity group 2 =120	ANOVA	ANOVA (age and sex)	Gr 0 vs 1 (Adj age, sex)	Gr 1 vs 2 (Adj age, sex)	Gr 0 vs 2 (Adj age, sex)
TAPSE (mm)	23.8±3.8	25.0±4.3	27.0±3.7	0.0001	0.004	0.027	0.0001	0.0001
S` RV (cm/sec)	0.134±0.002	0.140±0.002	0.136±0.002	0.1	0.1	0.1	0.1	0.1
E/A RV	1.86±0.87	2.21±0.90	2.17±0.87	0.01	0.045	0.025	0.9	0.079
E/E` RV	3±1	3.1±0.9	3.2±1	0.4	0.5	0.3	0.9	0.02
RV length (mm)	62.1±7.1	61.8±7.4	64.9±9.0	0.017	0.018	0.9	0.019	0.027
RV basal diameter (mm)	32.7±4.9	33.7±4.9	35.2±3.9	0.002	0.001	0.1	0.6	0.0001
RV mid diameter (mm)	24.3 ±4.3	23.9±3.7	26.0±3.7	0.001	0.02	0.4	0.0001	0.003
RA volume (ml)	33.5±12.4	35.5±14.0	39.7±14.8	0.003	0.001	0.3	0.006	0.0001
PASP (mmHg)	20.2±6.0	20.6±6.4	21.3±6.6	0.2	0.5	0.4	0.3	0.1
PASP/SV	0.31±0.01	0.31±0.12	0.28±0.09	0.051	0.9	0.5	0.007	0.035
TAPSE/PASP	1.32±0.50	1.35±0.56	1.40±0.53	0.4	0.5	0.6	0.4	0.2
PWV (m/s)	5.17±1.01	4.82±0.92	4.51±0.95	0.0001	0.0001*	0.017*	0.029*	0.0001*

TAPSE: Tricuspid Annular Plane Systolic Excursion.

S`RV: Lateral Tricuspid Annulus Peak Systolic Velocity.

RV: Right Ventricle.

RA: Right Atrium.

PASP: Pulmonary Artery Systolic Pressure.

CO: Cardiac Output.

SV: Stroke Volume.

PVR: Pulmonary Vascular Resistance.

PWV: Pulse Wave Velocity.

HR: Heart Rate.

*In the adjustment HR was included.

Table 4: Uni and Multiregression analysis among groups.

Dependent Variable: PASP		
Univariate Model	**β Coefficient**	**p**
Constant	4.558	0.0001
Age	-0.069	0.41
Sex	-0.056	0.44
BMI	-0.004	0.95
MAP	-0.002	0.98
HR	-0.095	0.166
Local PWV	-0.131	0.148
E/E`	0.103	0.107

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LVEF	-0.052	0.4
SV	0.143	0.054
Sport Intensity	0.061	0.378
Backward Multiregression Model		
Constant	8.263	0.0001
SV	0.155	0.014
HR	-0.121	0.051
PWV	-0.174	0.006
Dependent Variable: RV Longitudinal Diameter		
Univariate Model	β Coefficient	p
Constant	59.287	0.0001
Age	-0.092	0.092
Sex	-2.644	0.037
MAP	0.024	0.647
HR	-0.033	0.413
Local PWV	0.362	0.573
LVEF	0.018	0.795
SV	0.088	0.011
Sport Intensity	0.44	0.289
Backward Multiregression Model		
Constant	61.357	0.0001
Age	-0.075	0.049
Sex	-2.729	0.028
SV	0.101	0.003
Dependent Variable: PASP/CO Ratio		
Univariate Model	β Coefficient	p
Constant	7445	0.0001
Age	0.008	0.431
Sex	0.548	0.039
MAP	-0.036	0.001
Local PWV	-0.15	0.257
LVEF	0.0001	0.998
Sport Intensity	0.146	0.074
Backward Multiregression Model		
Constant	7.158	0.0001
Sex	0.522	0.038
MAP	-0.38	0.0001
Sport Intensity	0.165	0.036
Dependent Variable: LVM		
Univariate Model	β Coefficient	p
Constant	156.333	0.0001
Age	0.373	0.089
Sex	-41.624	0.0001
MAP	0.203	0.361

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HR	-0.383	0.002
Local PWV	0.721	0.789
LVEF	-0.251	0.416
SV	0.959	0.0001
Sport Intensity	3.89	0.031
Backward Multiregression Model		
Constant	157.494	0.0001
Age	0.461	0.005
sex	-42.867	0.0001
SV	0.973	0.0001
HR	-0.344	0.032
Sport Intensity	3.884	0.029

MAP: Mean Arterial Pressure.

HR: Heart Rate.

LVEF: Left Ventricle Ejection Fraction.

SV: Stroke Volume.

PWV: Pulse Wave Velocity.

TAPSE: Tricuspid Annular Plane Systolic Excursion.

PASP: Pulmonary Artery Systolic Pressure.

SV: Stroke Volume.

RV: Right Ventricle.

LV: Left Ventricle.

2) The LV remodeling was proportional to PA also driven by the group with more intense activity while LV systolic function and LV-arterial coupling was similar among groups. 3) LV E/A ratio and RV E/A ratio were significantly higher in the leisure-time mild PA group compared to the control group but like those in the athletes.

Regular aerobic exercise is associated with increased LV internal dimensions, eccentric LV hypertrophy, and improved LV diastolic function [35,36]. Intense prolonged aerobic exercise is also associated with dilated RV chambers and is accompanied by changes in RV systolic function. In a recent metanalysis [37] that included athletes engaging in highly dynamic sports, PASP, TAPSE and S' were higher compared to sedentary controls. However, RV global (6-segments) and free-wall (3-segments) longitudinal strain were similar between athletes and controls. An echocardiographic study on elite pre-adolescent soccer players showed that they had greater RV cavities, higher RV fractional shortening and similar longitudinal strain compared to a control group [38]. Lakatos et al. [39,40] used 3D echocardiography to study RV mechanics in young top-level water polo athletes.

A new software program able to decompose the model's motion of the RV combined with the three orthogonal anatomically relevant axes as longitudinal, radial, and anteroposterior wall motions was used. The authors found that the relative contribution of RV longitudinal motion and longitudinal EF were increased while radial motion and radial EF were reduced in the athletes compared to the control group. Physical exercise also affects the pulmonary circulation which in the athletes is characterized by high flow, low pressure, and low resistance. D`Andrea et al [41] reported the range of TVR and derived PASP at rest in highly trained athletes. They found that PASP can reach the upper physiologic limit of 40 mmHg in line with the increase of blood flow, and it was higher in endurance than in strength athletes.

Much less is known about the effect of physical exercise in the general population on the RV. One of the largest studies on this topic using CMR, was done from the MESA-Right Ventricle Study, an ancillary study of the Multi-Ethnic Study of Atherosclerosis (MESA). They found that RV mass and volumes were directly related to the intensity of physical activity, but RV function was not evaluated. Dawes et al [42], also using CMR, studied a group of 1096 adults without cardiovascular disease or cardiomyopathy-associated genetic variants. The population was divided into 5 PA-intensity groups, from no exercise (level I) to high exercise level (level V). Also, in this case there was a correlation between LV, RV volumes and PA. They also found a positive association between LV indexed by stroke volume (SVi, LV-SVi) and RV indexed by SV or RV-SVi, with PA. In this study, the difference in cardiac adaptation

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among groups was driven by the groups that exercised more while no difference was found between sedentary and light physical activity groups. CMR is the gold standard for the evaluation of the RV but is not the first approach for cardiac evaluation in clinical routine [43].

Our study based on TTE cardiac evaluation confirmed that RV and RA adaptation is characterized by a progressive increase in diameters from the sedentary to the most active group [44]. However, the increase of RV longitudinal diameter was not directly dependent on PA but instead on SV, representing the RV preload. Intense physical activity changed RV volumes more than LV ones, and an increased PAPS at rest has been observed in highly trained athletes rather than in recreational sports people. In our study, PASP was within normal values at rest, and did not increase across PA groups as did PAPS/SV highlighting the compliance of the pulmonary vasculature even when the SV increased, in athletes' group. We also found a progressive increase in myocardial longitudinal displacement (TAPSE) according to the amount of exercise but not changes in myocardial velocity (S` wave) in agreement with previous data.

The apparent discrepancy between TAPSE and S` can be explained by the fact that TAPSE represents all free wall movement and is affected by myocardial contractility and RV load, while the S` waveis more representative of local basal contraction. The increase in TAPSE across groups was not only dependent on pre-load but also on PA per se. Importantly, RV-arterial coupling (TAPSE/PASP) did not change across the groups suggesting a proportional adaptation of the RV function to pulmonary pressure. RV E/A ratio improved across exercise groups, but the maximum effect was seen in the leisure-time mild exercisers. However, the improved diastolic function was not independently related to PA but to carotid PWV in a negative fashion. Explaining the effect of carotid PWV on RV diastolic function is challenging but, as Lam et al hypothesized 45, systemic arterial stiffness could be a marker of overall stiffness, included the pulmonary vasculature. Regular PA can delay the age-related increase in arterial rigidity either of the pulmonary and the systemic circulation.

Our results confirm that an increase in SV characterizes LV adaptation to PA and, consequently, in LV and LA dimensions, being more evident in the active group. The improvement of LV diastolic function was similar in leisure-time mild exercise group and the recreational athletes suggesting that a beneficial effect of exercise on LV diastolic properties can be achieved with less exercise intensity.

Limitations

There are several limitations to be listed. First, the classification of the amount of PA was self-reported and was not precise, although supported by a significant inverse correlation with HR. Considering the cardiac structure and function parameters recorded by echocardiography, none of the new indexes, such as longitudinal strain and 3D volumes for both ventricles, were available when the study started. Another major limitation is our study design's cross-sectional nature, which precludes us from making any longitudinal observation.

Conclusions

In conclusion, we found that regular PA in healthy subjects benefits the heart in different ways. Intense PA affects cardiac structure adaptation while leisure- time mild PA is more beneficial on the diastolic function. Even within normal limits, right ventricular adaptation to regular PA is indicated by a gradual rise in TAPSE and PASP. Despite the considerable rise in SV, the pulmonary vasculature was likely compliant enough to handle the higher RV-preload. Future research is needed to provide a more complete description of RV adaptation to chronic PA in non-professional athletes, as well as its impact on cardiovascular outcomes.

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