

The Effect of Swimming Training on Heart Structure and Function of Elite Athletes

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Abstract

Introduction: Swimming training fosters the heart performance. The aim of this study was to determine the effect of swimming training on heart structure and function of elite swimmers.

Methods: The study was a causal comparative. The statistical sample included swimmer and non-athlete groups. Athletes were the elite swimmers who participated voluntarily in this study. Inclusion criteria for athletes included a history of at least 5 years of regular swimming training. After sampling, all participants took part in the echo-heart test in which they were given Color Doppler M-Mode echocardiography. The functional and structural parameters included left ventricular internal diameter in systole (LVIDs), left ventricular internal diameter in diastole (LVIDd), interventricular septal end diastole (IVSd), left ventricular mass index (LVMI), left atrial dimensions (LAD), aortic root dimension (ARD), left ventricular posterior wall dimensions (LVPWD), heart rate (HR) and ejection fraction (EF). Shapiro-Wilk test was used to normalize the research data. Statistical analysis was used by independent samples t-test and Analysis of variance (ANOVA). The p-value was defined as $p \leq 0.05$. The hypothesis test was performed using SPSS software version 19.

Results: The independent sample t-test results showed that swimmers had a significant decrease in LVIDs ($p = 0.001$), LVPWD ($p = 0.001$) and HR ($p = 0.001$) compared to non-athletes. The results also revealed that swimmers had a significant increase in a LVIDd ($p = 0.001$), LAD ($p = 0.001$), ARD ($p = 0.001$) and EF ($p = 0.012$) compared to non-athletes. There was no significant difference between groups in IVSd ($p = 0.789$) and LVMI ($p = 0.931$). But the results were different when variables were adjusted to the participants' age, weight, height and body mass index (BMI) by ANOVA. The adjusted results showed that swimmers had a significant decrease in LVIDs ($p = 0.002$) and HR ($p = 0.019$) compared to non-athletes. The results also revealed that swimmers had a significant increase in a LAD ($p = 0.001$) and ARD ($p = 0.001$) compared to non-athletes. There was no significant difference between groups in LVIDd ($p = 0.266$), IVSd ($p = 0.255$), LVMI ($p = 0.984$), LVPWD ($p = 0.128$), and EF ($p = 0.063$).

Conclusion: Long-term swimming training can lead to some heart physiological changes in elite swimmers. It seems that these changes can improve heart performance in these athletes.

Keywords: Heart, Swimmers, Non-athletes

Introduction

Among all types of exercise, aerobic activities have favorable cardiovascular effects. Endurance exercise training has many positive effects on health. It also can improve metabolism, reduce cardiovascular risk and cardiovascular mortality (1). The studies also indicate that there is an inverse correlation

between the resting blood pressure and the amount of physical activity in both women and men. Regular exercise also prevents hypertension and decreases blood pressure in younger and older adults. There are many physiological changes induced by endurance exercise training. These changes include increase in heart size and volume, blood

volume and total hemoglobin, stroke volume, rest and exercise cardiac output and VO_{2max} (2). It is accepted that a high level of endurance exercise training increases parasympathetic tone and decreases sympathetic control of the heart. Endurance exercise training also increases plasma volume, which would increase ventricular preload and hypertrophy. These adaptations caused by endurance exercise training promote heart contract (3). Endurance is an necessary requirement for performance in swimming. So, endurance can effect heart hypertrophy. The result of this hypertrophy is the pumping of a great volume of nutritive blood to the arteries (4). Type of exercise is on of the most important factors that affects heart remodeling (5). For example, isotonic exercises include swimming, long distance running and cycling cause increase in cardiac output (1,6). While isometric exercises such as weightlifting cause peripheral vascular resistance (1,7). Increase in peripheral vascular resistance causes systolic hypertension (7). Cardiac remodeling has different implications. Hypertrophy of the heart can be physiologically or pathologically. Physiological changes enhance heart performance and output. Any change in cardiac output is determined by preload. Left ventricular internal diameter in diastole (LVIDd) is a indices of preload (8). Although physiological changes enhance heart ability but pathological changes attenuate cardiac output, enhance apoptosis and fibrosis (9). Physiological adaptations of the heart are essential for health. One of the major physiological adaptations of the heart is ejection fraction (EF). This variable is a good indicator that measures the amount of blood pumped out of the left ventricle (10). Decreased EF is associated with heart damage (10, 11). Although, LVIDd and EF are two important parameters of healthy heart but in order to accurately check the heart of swimmers and non-athletes it is necessary to measure all heart functional and structural parameters. Therefore, other functional and

structural parameters of the heart such as left ventricular mass index(LVMI), left ventricular internal diameter in systole (LVIDs), interventricular septal end diastole (IVSd), left atrial dimensions (LAD), aortic root dimension (ARD), left ventricular posterior wall dimensions (LVPWD) and heart rate (HR) will be checked. So this study aimed to investigate the effect of swimming training on heart structure and function of elite swimmers.

Methods

The study was a causal comparative that investigated the effect of swimming training on heart structure and function of elite swimmers. The research procedures had previously been approved by the Research Ethics Committee of Islamic Azad University, Omidiyeh Branch (IAUOB). The detailed information about the study was given to the volunteers during the first meeting. The statistical sample consisted of two groups of 10 including swimmers and non-athletes. Athlete group was the elite swimmers who participated voluntarily in this study. The selection was based on determined inclusion/exclusion criteria. The inclusion criteria were: (1) participation consent (2) age range 25 to 35(3) a history of at least 5 years of professional training. Exclusion criteria included (1) ages below 25 or above 35yrs (2) a history of below 5yrs of professional training (3) any cardiomyopathy disease. Athlete group was all elite national swimmers of Khuzestan province. After sampling and consultation with a cardiologist, the groups were called for echocardiography. For this aim, echocardiography Eco Color Doppler M-Mode was used. Statistical analysis of data was done through SPSS, Verssion 19. Significance level was defined as $p \leq 0.05$. Data normality was done by Shapiro-Wilk test. Differences between groups were assessed by independent samples t-test and ANOVA. Pearson correlation was used to describe the linear relationship between dependent variables. LVMI was calculated using the modified

Devereux formula (12). All variables were adjusted to the participants' age, weight, height and body mass index (BMI).

Results

Figure 1 shows descriptive data regarding some of the anthropometric and physiological characteristics of the subjects. Also figure 2 and 3 show heart structure and function results in swimmers and non-athletes.

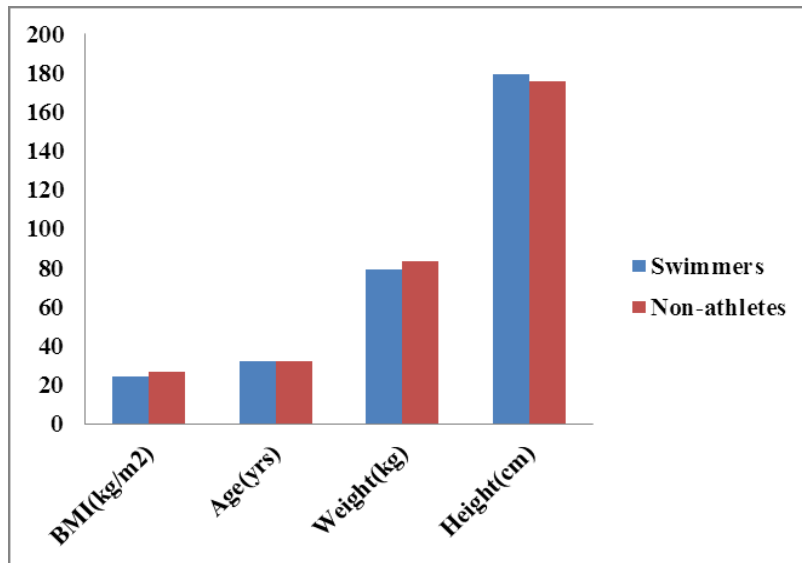


Figure 1. Descriptive result of anthropometric and physiological characteristics

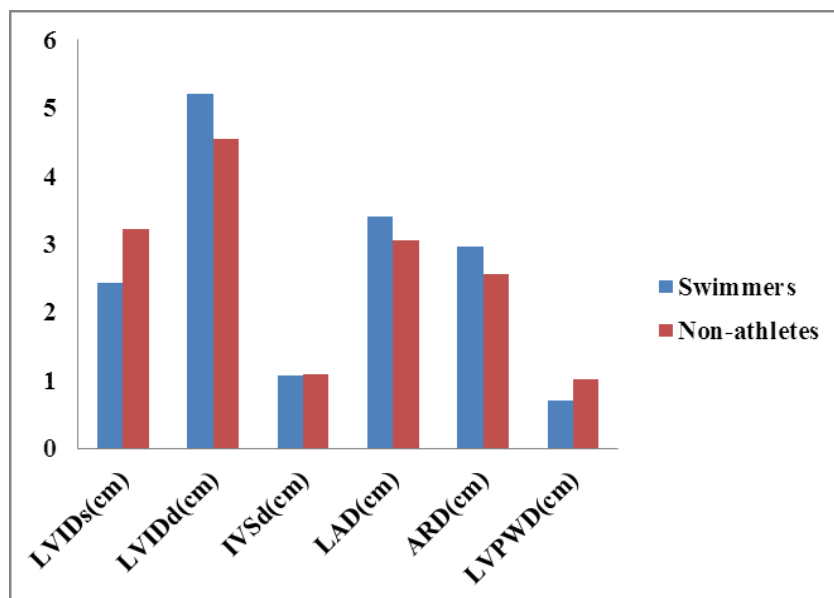


Figure 2. Heart structure results in swimmers and non-athletes

Table 1 shows independent samples t-test result of variables in swimmer and non-athlete groups. These results showed that swimmers had a significant decrease in LVIDs($p = 0.001$), LVPWD($p = 0.001$) and HR($p = 0.001$) compared to non-athletes. The results

also revealed that swimmers had a significant increase in a LVIDd ($p = 0.001$), LAD($p = 0.001$), ARD ($p = 0.001$) and EF ($p = 0.012$) compared to non-athletes. There was no significant difference between groups in IVSd ($p = 0.789$) and LVMI ($p = 0.931$).

Table 1. Independent samples t-test result of variables in swimmers and non-athletes groups

Variables	Groups	Number	t	df	Effect size	Sig
LVIDs (cm)	swimmers	10	-3.797	18	0.44	0.001*
	non-athletes	10				
LVIDd (cm)	swimmers	10	+3.859	18	0.45	0.001*
	non-athletes	10				
IVSd (cm)	swimmers	10	-0.271	18	0.01	0.789
	non-athletes	10				
LVMI (g/m ²)	swimmers	10	+0.087	18	0.01	0.931
	non-athletes	10				
LAD (cm)	swimmers	10	+3.826	18	0.44	0.001*
	non-athletes	10				
ARD (cm)	swimmers	10	+3.978	18	0.46	0.001*
	non-athletes	10				
LVPWD (cm)	swimmers	10	-6.249	18	0.68	0.001*
	non-athletes	10				
HR (bpm)	swimmers	10	-5.037	18	0.58	0.001*
	non-athletes	10				
EF (%)	swimmers	10	+2.777	18	0.30	0.012*
	non-athletes	10				

LVIDs: left ventricular internal diameter in systole, LVIDd: between left ventricular internal diameter in diastole, IVSd: interventricular septal end diastole, LVMI: left ventricular mass index, LAD: left atrial dimensions, ARD: left ventricular posterior wall dimensions, LVPWD: left ventricular posterior wall dimensions, HR: heart rate, EF: ejection fraction, * significant changes.

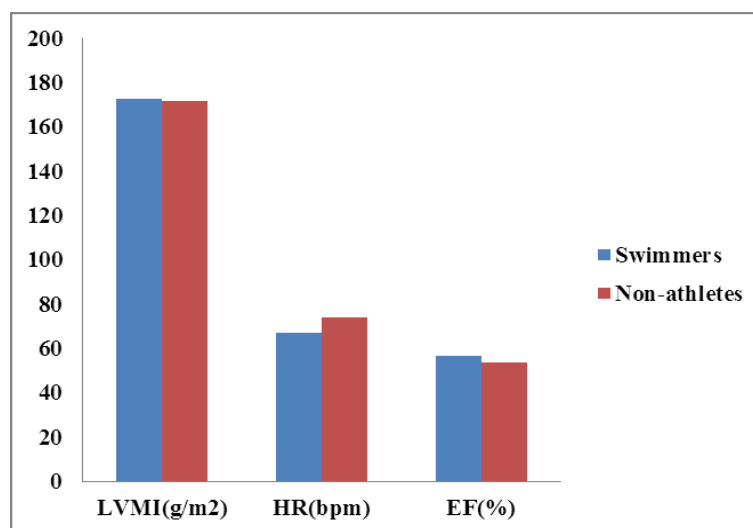


Figure 3. Heart structure and function results in swimmers and non-athletes

Table 2 presents data related to ANOVA result of variables in swimmer and non-athlete groups. The results were different from independent sample t-test findings when variables were adjusted to the participants' age, weight, height and BMI. The adjusted results showed that swimmers had a significant decrease in LVIDs ($p = 0.002$) and HR ($p = 0.019$) compared to non-athletes. The results also revealed that swimmers had a significant increase in a LAD ($p = 0.001$) and ARD ($p = 0.001$) compared to non-athletes. There was no significant difference between groups in IVSd ($p = 0.255$), LVPWD ($p = 0.128$), LVMI ($p = 0.984$), LVIDd ($p = 0.266$) and EF ($p = 0.063$). Table 3 presents data related to Pearson correlation on variables. This results show that there is the highest

correlations between weight and BMI ($r = +0.830$) and also between LVPWD and HR ($r = 0.667$).

Discussion

Physical inactivity can decrease cardiorespiratory fitness (13). In this research, the heart structural and functional parameters of elite athletes and non-athletes were investigated. The results regardless of anthropometric parameters show that there was a significant difference between the two groups in LVIDs, LVIDd, LAD, ARD, LVPWD, HR and EF. Swimmers had a significant decrease in LVIDs, LVPWD and HR compared to non-athletes. The results also revealed that swimmers had a significant increase in a LVIDd, LAD, ARD and EF compared to non-athletes.

Table 2. ANOVA result of heart function and structure variables

Variables	Sorce	Sum of Squares	df	F	Sig	Effect size
LVIDs(cm)	Groups	1.801	1			
	Error	1.653	14	15.248	0.002*	0.52
	Corrected total	4.750	19			
LVIDd(cm)	Groups	0.237	1			
	Error	2.465	14	1.345	0.266	0.08
	Corrected total	4.957	19			
IVSd(cm)	Groups	0.003	1			
	Error	0.026	14	1.411	0.255	0.09
	Corrected total	0.031	19			
LVMI(g/m ²)	Groups	0.331	1			
	Error	11124.074	14	0.001	0.984	0.01
	Corrected total	12078.233	19			
LAD(cm)	Groups	0.328	1			
	Error	0.435	14	10.559	0.006*	0.43
	Corrected total	1.366	19			
ARD(cm)	Groups	0.306	1			
	Error	1.718	14	5.968	0.028*	0.29
	Corrected total	1.626	19			
LVPWD(cm)	Groups	0.033	1			
	Error	0.175	14	2.616	0.128	0.15
	Corrected total	0.748	19			
HR(bpm)	Groups	59.069	1			
	Error	118.553	14	6.975	0.019*	0.33
	Corrected total	406.950	19			
EF(%)	Groups	21.962	1			
	Error	75.625	14	4.066	0.063	0.22
	Corrected total	150.000	19			

* Significant changes

Table 3. Relationship between variables

Variable	Variable	Number	r	r ²	Sig
Height(cm)	Weight(kg)	20	+ 0.533	0.284	0.016*
	HR(bpm)	20	- 0.464	0.215	0.039*
Weight(kg)	BMI(K/m ²)	20	+ 0.830	0.688	0.001*
BMI(K/m ²)	LVIDs(cm)	20	+ 0.489	0.243	0.028*
	LVPWD(cm)	20	+ 0.616	0.379	0.004*
	ARD(cm)	20	- 0.530	0.280	0.016*
LVIDs(cm)	LVPWD(cm)	20	+ 0.474	0.224	0.035*
	HR(bpm)	20	+ 0.600	.360	0.005*
	EF(%)	20	- 0.506	0.256	0.023*
	LVMI(g/m ²)	20	+ 0.652	0.425	0.002*
LVIDd(cm)	LAD(cm)	20	+ 0.649	0.421	0.002*
	LVPWD(cm)	20	- 0.582	0.338	0.007*
	HR(bpm)	20	- 0.526	0.276	0.017*
	EF(%)	20	+ .0568	0.322	0.009*
HR(bpm)	EF(%)	20	- 0.369	0.136	0.045*
LAD(cm)	LVPWD(cm)	20	- 0.606	0.367	0.005*
	HR(bpm)	20	- 0.496	0.246	0.026*
	LVPWD(cm)	20	- 0.488	0.238	0.029*
ARD(cm)	HR(bpm)	20	- 0.463	0.214	0.040*
	EF(%)	20	+ 0.512	0.262	0.021*
LVPWD(cm)	HR(bpm)	20	+ 0.667	0.451	0.001*
HR(bpm)	EF(%)	20	- 0.607	0.368	0.005*

* Significant relationship

There was no significant difference between groups in IVSd and LVMI. Base on theses results, it can be concluded that endurance exercise training can physiologically enhance cardiac structure and function. These adaptations in cardiac parameters of elite swimmers, especially the LVIDs, LVIDd and EF, can be caused by preload in left ventricle (14). Csajagi et al. showed significant

morphological adaptation of the left ventricular in swimmers (15). Some other studies have shown that swimming in endurance aerobic exercise form can increase cardiac output (1, 6). According to Franck Starling's law, increased preload caused by aerobic exercise can expand heart chambers internal dimensions. The left ventricle is one of the most affected chambers (16).The results

with consideration of anthropometric parameters show that there was a significant difference between swimmer and non-athlete groups in LVIDs, LAD, ARD and HR. In general, the results were completely different when variables were adjusted to the participants' age, weight, height and BMI by ANOVA. The adjusted results showed that swimmers had a significant decrease in LVIDs, and HR compared to non-athletes. The results also revealed that swimmers had a significant increase in a LAD and ARD compared to non-athletes. There was no significant difference between groups in LVIDd, IVSd, LVMI, LVPWD and EF. It seems that the anthropometric parameters play an important role in interpreting the results of heart structural and functional parameters. The LVIDd and EF are two parameters that have been affected by anthropometric parameters. Both independent t-test and ANOVA results showed that LAD in the swimmers group had a greater increase compared to the non-athlete group. Studies have shown that LAD has increased after training (17,18). Pelliccia et al. showed that LAD increased in 20% of athletes (19). It has also been reported that LAD has increased in highly trained athletes (20). It seems that cardiac preload caused by swimming training affects the LVIDs, LAD, ARD and HR more than LVIDd, IVSd, LVMI, LVPWD and EF. Despite these results, one of the most important limitations of a causal comparative study is the inability to control the training sessions. So to obtain the desired results in heart studies, other researchers

should consider this limitations and also adjust the variables to the participants' anthropometric parameters.

Conclusion

Swimming training can cause physiological adaptations in elite swimmers. These adaptations are different from cardiomyopathy changes. It seems that long-time regular swimming training can enhance heart health and reduce cardiovascular disorder.

Ethical issues

The study protocols and procedures had previously been approved by the Research Ethics Committee of Islamic Azad University Omidyeh Branch.

Authors' contributions

All authors contributed equally to the writing and revision of this paper.

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