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Aerobic Training Induced Structural Changes of the Heart

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ABSTRACT

Exercise may cause structural changes, which may increase cardiac functions such as enhanced pumping mechanism due to increased heart mass and volume. These changes may contribute to better performance. According to several studies, endurance training causes changes in structure of the heart since endurance trained athletes had larger LV dimensions, and mass. For instance, both cycling and rowing studies indicated positive structural adaptations of the heart; in addition, elite rowers had higher values than sub-elite rowers; this result suggests that elite athletes have higher level of adaptations supporting. Also, In all rat studies supported that endurance training increases LV mass, wall thickness and dimensions. Endurance training causes structural adaptations such as increased cardiac muscle mass, left ventricular wall thickness and chamber size. It is recommended that structural changes in the heart because of endurance training enhance the heart's ability to pump blood mainly by increasing its stroke volume. Training for adequate duration and time also should be taken in consideration to improve cardiac function because of the changes in the structure of the heart.

Keywords: Heart, cardiovascular system, training, structural changes.

INTRODUCTION

Exercise has several effects on cardiovascular mechanisms, including physiological hypertrophy and structural changes, which may improve cardiac functions such as better pumping mechanism because of increased heart mass and volume. Physical activity affects the loading conditions of the heart (3). In dynamic exercise, the force development is very low compared to static exercise, which results in improved cardiac output by increasing heart rate and stroke volume. In dynamic exercise, the load on the heart is mainly of the volume type. However, in static exercise, force development caused by my minimal movement resulted in slight elevation of cardiac output, and a more pronounced rise of blood pressure. This causes a pressure load on the heart. The alterations on the structure of the heart because of different loads seem to be normal and beneficial for cardiac functions; however, in disease states, volume load may cause an enlargement the left ventricular internal diameter and proportional increase of wall thickness; on the other hand, pressure work may cause thickening of the ventricular wall and unaltered internal dimension. These changes are assumed to operate to control systolic wall stress. These typical overload types can impact the cardiac structure in different ways (9). Exercise training has been linked with structural changes of cardiac muscle mass, left ventricular wall thickness and chamber size. It has been found that structural changes in the heart may cause increased stroke volume. A higher stroke volume helps the heart work more efficiently because more blood is being ejected during each heartbeat. Higher stroke volume is very important to determine performance capability. The structural changes of the heart may contribute to better performance; therefore, it is essential to know how endurance training contributes to the changes in cardiac structure. Several articles were reviewed to investigate and analyze structural adaptations of the heart through endurance training models.

Review

In a comparison study of strength and endurance trained versus control subjects, Baggish et al. (2) investigated the influence of training on competitive athletes, after 90 days of strength and endurance training. They predicted that significant structural changes would occur depending on the training discipline. They assessed competitive university athletes including endurance (EA, n = 40 long-distance male rowers) and strength athletes (SA, n = 24 football players). EAs had increased LV dilation, improved LV diastolic function, biatrial growth, and RV dilation with improved systolic and diastolic function. On the other hand, even SAs had concentric LV hypertrophy, they did not experience changes in atrial dimensions or RV parameters but their LV diastolic function were reduced. They questioned if the subjects used anabolic steroids or not instead of performing serum assessment; thus, they see this limitation for the study. They noted that there was not a previous study that found that strength training because significant concentric LV hypertrophy with decreased diastolic function.

This is one of the unique studies that compared the functional and structural changes of both strength and endurance trained athletes, and adequately supports previous studies on improvements of LV dimensions through aerobic training. However, the athletes had no previous competitive athletic experience other than training in their current university clubs, and 90 days of training may not be

enough to see the possible changes that might result at the end of the study such as improved RV parameters and atrial dimensions. Also, endurance trained group had both female and male subjects but the strength trained group did not include female subjects.

In a similar study, Venckunas et al. (12) also studied the degree and type of cardiac hypertrophic response to diverse athletic training in females. The subjects were Caucasian female sprinters (n = 10) and long-distance runners (n = 10) of similar age (range 16–34 years), training experience (5–18 years) in age-matched healthy female sedentary controls (n = 10).). Long distance runners had thicker interventricular septum and bigger LV wall, and larger LV mass as compared to sprinters or sedentary controls. Relative wall thickness was higher in long-distance runners. Female long-distance runners had larger cardiac mass primarily due to myocardial wall thickening.

The findings of this study are consistent with the Baggish et al. (2). Endurance training created have better adaptations both function and structure of the heart. Since the study only have female subjects, it is hard to compare the possible changes in both genders that might have created different structural changes.

Combined Activity Studies

Cycling or rowing represents endurance type of exercises on heart loads. In a study to assess cardiac structural and functional changes induced by competitive amateur cycling, Kuchynka et al. (5) assessed 51 competitive amateur cyclists and 47 medical students of the same age who served as the control group. According to results, the cyclists had higher LVM values than controls because of larger LV wall thickness and LVEDD. Cyclists had larger left and right atrial sizes than in controls. There was a correlation between the left atrial diameter and LVEDD. Athletes' RVEDD was slightly larger after adjustment for BSA. They stated that, in a previous study, elite cyclists had 15 mm wall thickness implying that they were be danger of hypertrophic cardiomyopathy. Even though wall thickness was higher in cyclists in this study, since none of the study subjects had above 13 mm wall thickness suggesting that the subjects are not considered as clinically at risk condition.

Functional and structural changes of cyclists' hearts were very well tested, it seems that amateur cyclists' hearts' dimensions were increased as endurance trained athletes did as stated in previous studies noted in the introduction section, and also elite athletes' wall thickness were higher than amateur cyclists' hearts' wall thickness noted in this study. There were only male athletes included in this study; thus, it would be better to include female subjects in the study.

Baggish et al. (1) also examined elite competitive rowers (ER), subelite competitive rowers (SR), and sedentary controls (C) to determine differences of cardiac structure and function. They used two-dimensional, tissue Doppler, and speckled-tracking echocardiography to measure the parameters among ER (n = 20), SR (n = 20), and C (n = 20). Rowers in both study groups (ER and SR) had significantly greater cardiac structural dimensions than controls in all parameters assessed. Both groups had larger LV wall and chamber than controls. ER had larger unadjusted LV wall and chamber dimensions, LV mass, left atrial volumes, and RV dimensions than SR. After adjustment for BSA, LV and RV chamber dimensions and LV mass was larger in ER than that in SR. However, there were no left atrial size and LV wall thickness after BSA adjustment. They discussed that ER had more training due to the long-term rowing experience (years) than SR; therefore, it was hard to define relative contributions of experience or skill type.

The methods and the development of the purpose of the study were explained very clearly and explicitly. It is a unique study testing the relative affects of skill or training levels in the same sports. This study was done in 2010, and there are new instruments to measure the structural changes of the heart such as three-dimensional CMR instead using echocardiography; thus, it would be better to use CMR. Both cycling and rowing studies have similar results of structural adaptations of the heart; however, it is very clear that elite rowers had higher values than sub-elite rowers; this result suggests that elite athletes have higher level of adaptations even the rowing study implied that the contribution of training time should be investigated because elite athletes might have trained longer time than amateur athletes.

Sex Specific Endurance Training Studies

Steding et al. (7) aimed to test hypothesis if the left and right ventricles expand in the same order of extent in males and females. There were 71 athletes (30 female) and 60 healthy controls (20 female) in the study. Cardiovascular Magnetic Resonance was used to measure the cardiac structures. As a result, as LVEDV increased, RVEDV increased in the same order of magnitude in both males and females ($R2 = 0.87$, $p < 0.005$. All male subject groups had a significantly higher THV/BSA and LVM/THV when compared to females, except for THV/BSA in triathletes because female triathletes had 4 hours more endurance training than males. Cardiac dimensions were increased with endurance training supporting Petersen et al. (8) study. The left and right ventricles were proportionally increased with increment of endurance training. They suggested that cardiac pumping in athletes can be further investigated with velocity encoded CMR.

The methods of the study were explained explicitly and the results of the study were very well discussed and analyzed. However, the sports such as handball and soccer were different than triathlon training. The heart load in these sports may be different, soccer and handball sometimes can be considered as strength training activities. They should be carefully reviewed before counting them as strength training. When it was carefully looked at the results, it is obvious that each sport had different amount of contribution on the structural change of the subject's heart.

In a similar study, Petersen et al. (8) aimed to investigate male and female young adult elite athletes with age-and sex-matched sedentary controls to assess sex-specific differences for cardiac structures hypothesizing those structural changes to exercise training would be sex-specific. Twenty-three male athletes in rowing, swimming, or triathlon and 20 female athletes in rowing, swimming, or triathlon) and age- and sex-matched sedentary controls (21 male/17 female) were assessed with (CMR) imaging. The LV/RV ratio for mass index was considerably enlarged in athletes compared to controls. The study indicated an unbalanced LV and RV hypertrophy in those sports. The study suggested that this disagreement might reflect differential effects to exercise type or severity as well as duration of training. They concluded that adolescent elite athletes do not confirm sex-specific adaptive structural to exercise training.

The strength of both sex-specific studies is that they used elite athletes as subjects in the study and CMR was used to measure the cardiac dimensions; however, the results conflict with each other when the sex specific structural changes were considered. As it was noted in the discussion section, conflict results might be because of differential effects to exercise type or severity as well as duration of training

Rat Studies

Kemia et al. (4) tested relative efficiency of 10-week HIGH versus moderate (MOD) exercise amount on cardiac structure. Sprague–Dawley rats performed Treadmill-running at either 85%–90% (HIGH) or 65%–70% (MOD) of VO2max. Randomized three groups, high (HIGH) and moderate intensity (MOD), and sedentary control rats consisted total of 24 female adult rats. The study suggested that exercise intensity is serious factor on beneficial effects of regular exercise on cardiac adaptations since left ventricular cardiomyocytes were 14% longer in HIGH, versus 5% in MOD and had significantly larger Width and volume in HIGH, whereas there was only a trend in MOD. In addition, intra- ventricular septum, posterior wall thickness and chamber size was increased significantly.

There was a small sample size to test the different intensities. Also there were only female subjects in the study and 10 weeks of total training time might change the results of the study. The technique used to measure the cardiac dimensions was different than the other studies' techniques. It should be taken into consideration.

Medeiros et al. (6) investigated the effect of swimming exercise (ST) on cardiac hypertrophy by measuring cardiac weight and myocyte morphometry in sedentary (S, N = 12) and trained (T, N = 12) male Wistar rats. Training consisted of 1 hour training 5 times a week for 8 weeks. In T rats, the study suggested there was a cardiac hypertrophy because of the increases in left ventricle weight (13%) and myocyte dimension (21%). In addition, both right ventricle (18%) and total heart weight (13%) were significantly greater than before. LV myocyte diameter was also 21% increased in swimming-trained rats. The study discussed volume versus pressure overload by mentioning that strength-trained subjects showed increased LV wall thickness with a pattern of concentric geometry caused by pressure overload during exercise because they predominantly performed in static isometric anaerobic exercise and swimming training is mainly associated to volume overload-induced cardiac eccentric hypertrophy with prevalently longitudinal myocyte growth. Therefore, they concluded in discussion section that the cardiac hypertrophy seen in swimming-trained rats is commonly related with increased cardiac performance because of physiological and favorable cardiac modification.

The study examined of male rats: The functional and structural adaptations were divided in sections in both results and methods; thus, the study is very understandable and clear. It would be hard to know the long-term adaptations of cardiac structure according to this study. Therefore, the subjects should have been tested after termination of the study. In addition, it is also not clear if the procedures of rats' training can be applied to humans or not; that's why the results of the study cannot be generalized even though it supports previous studies that used human subjects. They also used only male subjects in the study.

Rodrigues et al. (10) studied the effects of 6 months of moderate-intensity aerobic exercise training on normal hearts. Septal and posterior wall thicknesses were increased with a 15% increase in LV mass index. LV diameters were not changed. It is noteworthy to mention that they stated the structural adaptations were not observed in previous studies probably because of the training programs used in the other studies may not have provided adequately intense or longer training period.

Using three-dimensional CMR to measure structural changes and training over the six-month period are important to have accurate results even though they are not regular athletes. It would be better to include athletes in the study.

Shi and Selig (11) also compared exercise responses and cardiac structure and at rest in endurance-trained (EX) and untrained X youngsters volunteered from an elite swimming team and an athletics organization. The participants trained in swimming or running three times per week for a year. Work intensity was self-rated using a scale of 1–5, Most of the trained adolescents in this study had a work intensity score was above 3. To improve aerobic capacity, 30 minutes of aerobic activity at least 70% of V02max is required. So, the score 3 meets the criteria. Males aged 14–16 years. After the assessment, there were no group differences between the two groups for LVDd, LVDs, and LVPW. Thus, the study suggests that if long-term training is responsible for cardiac hypertrophy, the alteration is slow and modest in adolescent people.

It is logical to assume that structural adaptations occur over a time and training period. Self-reported intensity may be a limitation for the study. Also, the subjects might have been trained in higher intensities to have possible adaptations; thus, the intensity of training may have been underestimated.

DISCUSSION

In comparison studies among endurance, strength and control subjects, the findings of Venckunas et al. (12) study are consistent with Baggish et al. (2) study. Endurance training have better adaptations both function and structure of the heart since endurance trained athletes had larger LV dimensions, and mass. Also, both cycling and rowing studies have similar results of structural adaptations of the heart; however, it is very clear that elite rowers had higher values than sub-elite rowers; this result suggests that elite athletes have higher level of adaptations supporting previous study that elite cyclists" wall thickness were higher than amateur cyclist's heart's wall thickness suggesting that the elite athletes may have better chance to have hypertrophy. In sex specific studies, cardiac dimensions were increased with endurance training in Steding et al. (7) and Petersen et al. (8) study. However, major contradiction of both studies is that even the left and right ventricles were proportionally increased with increment of endurance training in Steding et al. (7) study, right and left ventricle were not increased in the same magnitude of order in Petersen et al. (8) study. The conflicting result might be because of the differential effects of exercise type or severity as well as duration of training. In all rat studies discussed in the review also supported the human studies that endurance training increases LV mass, wall thickness and dimensions. The rat studies suggested that adequate intensity or longer training period is important to alter cardiac structure of the subjects. Rodrigues et al. (10) study also supports the concept that endurance training increases cardiac structures, whereas Shi and Selig (11) found no significant changes suggesting that long term training is necessary to have adaptive changes.

To conclude, endurance training causes structural adaptations such as increased cardiac muscle mass, left ventricular wall thickness and chamber size. It is suggested that structural changes help increase the heart capacity to pump blood primarily by increasing its SV. If there is no adaptation through endurance training, it should be because of not training for adequate duration and time. However, several factors should be considered to further elucidate the structural adaptations of endurance training. First, there should be more studies investigating sex-specific changes since two sex-specific studies contradict each other; one study suggests that right and left side of the heart does not enlarge in the same order of magnitude. This was a major contradiction. It was told that it might be because of different intensity, duration, or the type of the exercise. Second, most studies only include only male or female subjects; thus, it would be better to include both sexes in the studies. Finally, long-term training and intensity of the training are major factors affecting the structural changes.

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