

# Effects of physical exercises on the ventricular myocardium

Pimenta, L.<sup>1</sup>, Gama, EF.<sup>1</sup>, Maifrino, LBM.<sup>1,2</sup> and Souza, RR.<sup>1\*</sup>

<sup>1</sup>Biodinamic and Morphology Laboratory, São Judas Tadeu University

<sup>2</sup>Dante Pazzanese Institute of Cardiology, São Paulo, SP, Brazil

\*E-mail: souzarrd@uol.com.br

## Abstract

It is known that people who exercise regularly are less likely to develop heart diseases such as hypertension, obesity, diabetes mellitus, and dyslipidemia decreasing the occurrence of atherosclerosis and its complications: heart failure, stroke, and peripheral vascular disease. In the presence of physical exercises, the myocardium adjusts to the work loads metabolically and mechanically. There are two kinds of physical exercises: aerobic exercise and strength exercise or resistance training. Nowadays, strength exercises have become very popular due to several different reasons. Strength exercises are those performed against a specific opposing gradual resistance to muscular contraction such as weights. According to the literature, both types of exercises have effects on different body tissues. In the present work, a literature review, including scientific articles since 1980, on the effects of aerobic and strength exercises on the ventricular myocardium was conducted. The low and high-intensity strength exercises have different effects, but none of them causes major effects on the cardiovascular function chronically. The most evident alteration of the myocardium subjected to strength exercises is the increase in the cross-sectional area of the cardiac myocytes leading to myocardial hypertrophy.

**Keywords:** myocardium, strength exercises, wistar rat, morphometry, stereology.

## 1 Introduction

According to the World Health Organization - WHO (OMS, 2002), cardiac diseases have been the major cause of death worldwide in the last years (Figure 1).

Corroborating these data, according to the Brazilian Health Ministry, the major cause of death in the year of 2000 in Brazil was cardiovascular diseases. The general consensus in the literature is that regular aerobic physical exercises help prevent and control cardiovascular risk factors besides reducing the morbimortality of those who already suffer from a cardiopathy. Aerobic exercises have been used in order to prevent heart diseases, and when regularly and properly performed they help the primary prevention of disorders such as hypertension, diabetes, hypercholesterolemia, and obesity through the control of the risk factors (BLAIR, GOODYEAR, GIBBONS et al., 1984; PAFFENBARGER, HYD, WING et al., 1993; MITTLEMAN, MACLURE, TOFLER et al., 1993; FORJAZ and NEGRÃO, 1999).

According to the National Institute of Health (NIH, 1995) and to Ciolac (2004), physically active populations are less likely to develop heart diseases such as hypertension, obesity, diabetes mellitus, and dyslipidemia decreasing the occurrence of atherosclerosis and its complications: heart failure, stroke, and peripheral vascular disease.

On the other hand, strength exercises have become increasingly popular. According to Santarém (2000), strength exercises are those performed against a specific opposing gradual resistance to muscular contraction. Most of the times such resistance is represented by weight training. Nevertheless, there are only a few studies reporting the effect of this kind of exercise on the myocardium either

as prevention or rehabilitation from cardiac diseases. These exercises have become widely popular in gyms and health clubs (or health/fitness facilities), and can be performed isolated or concomitant with other aerobic exercises, which stimulates carrying out studies on their effect on the myocardium.

Forjaz and Negrão (1999) argue that low and high-intensity strength exercises have different effects, but none of them causes major effects on the cardiovascular function chronically although having highly beneficial effects on the skeletal muscle. Nonetheless, Bjarnason-Wehrens, Mayer-Berger, Meister et al. (2004) state that moderate-load strength exercises with discrete isometric components have been used in cardiac rehabilitation associated with aerobic exercises.

There are numerous studies on the effects of aerobic exercises in the scientific literature, whereas studies focusing on strength exercises are still scarce. This is probably due to the difficulties related to protocols for animal and human experimental research.

## 2 Material and methods

The PubMed (www.pubmed.nl) and MEDLINE databases were used as search reference using keywords in English without time restrictions. In this systematization, papers were searched using the following keywords: myocardium, aerobic exercise, strength exercise, Wistar rat, Morphometry, and Stereology by randomized combination until these terms were included.

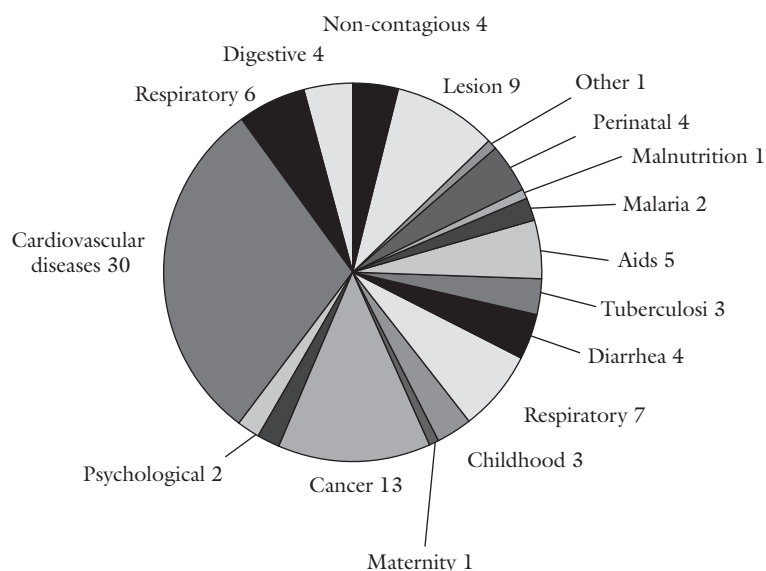


Figure 1. Cause of death worldwide % (OMS, 2002).

### 3 Results

#### 3.1 Functional myocardial structure

According to Hudlicka and Brown (1996), a normal adult myocardium consists of around 80% of myocytes and 20% of cellular matrix, which contains conjunctive tissue and blood vessels, among others. These proportions are different in distinct development stages and present an increased relative myocyte volume in the first years of life. In other cases, during pathological hypertrophy for example, the proportion of collagen and other cells rather than myocytes also increases in the cardiac interstitial.

The major function of myocardium muscle cells is to promote contraction- relaxation cycle in the heart. The myocytes contraction proteins account for half of the ventricular myocyte volume and are present in the myofibrils; while mitochondria occupy from one third to one fourth of the ventricular myocyte volume.

Reasonable amounts of collagen fibers among other elements are present in the myocardium interstitial, and they play important roles related to the ventricular function such as apoptosis regulation, deformation resistance, and structural alignment maintenance besides cardiac distension regulation, and force transmission during cardiac fiber shortening. Hence, they are considered important modulators for both diastolic and systolic cardiac function (CAULFIELD and WOLKOWICS, 1990). Nevertheless, an excess of collagen in the cardiac interstitial can cause ventricular diastolic dysfunction.

Besides the myocytes and collagen, the blood vessels also play an important role in the heart since they are responsible for the microcirculation and irrigation of this muscle.

#### 3.2 Effects of exercises on the myocardium

Effects on the myocytes. Aerobic exercises performed intensively and with an adequate duration can cause quantitative alterations of the cardiac myocytes, vessels,

and myocardium interstitial tissues both in humans and animals used for experimental research. Aerobic exercises are associated with eccentric cardiac physiological hypertrophy with predominant longitudinal myocyte growth (GROSSMAN, JONES and McLaurin, 1975; MOORE and KORZICK, 1995).

Studies with isolate intact myocytes indicate that long-duration continuous exercises can increase the length of left ventricular (LV) cells of rats (MOORE, 1993; PALMER, 1998; PALMER and MOORE, 1999) without altering the diameter although some studies have not find such results (LAUGHLIN, SCHAEFER and STUREK, 1992; DIFFEE, SEVERSEN and TITUS, 2001; DIFFEE and NAGLE, 2003).

Accordingly, other authors reported that intermittent exercises promote an increase in the width of ventricular myocytes of rats without increasing their length (NATALI, 2001, 2002). If low and moderate-intensity aerobic exercises can induce eccentric hypertrophy (elongated cells), some studies show that high-intensity exercises cause concentric hypertrophy (thick cells) (NATALI, 2001, 2002). The cardiac myocytes increase in area is a potential mechanism to explain the improvement in the contractile function of the myocardium induced by exercises (NATALI, 2004).

The transverse growth of the myocytes, with cardiac hypertrophy, is the response of the heart to the functional overload due to the low dividing capacity of cardiac cells (OLSON, 2004).

As proposed by Dorn, Robbins and Gugden (2003), the term hypertrophy should be used in the presence of cardiomyocytes increased in volume. Hypertrophy characterizes cell growth, which in the absence of cell division is used to differ hyperplastic and hypertrophic growth.

Effects on the collagen fibers. The myocardial collagen fibers are also influenced by physical exercises and the aerobic exercises attenuates the age-associated increase in the myocardial collagen (MAIFRINO and SOUZA, 2006).

Effects on blood vessels. Other components of the myocardium, such as arterioles, venules, and capillaries also undergo changes due to physical exercises. Aerobic exercises can cause quantitative changes in the myocardium vessels, increase the caliber of the coronary artery, increase the vessel lumen, and improve the blood flow systemically in the whole body.

Studies on the effects of aerobic exercises on the myocardium capillaries density or number indicate that quantitative alterations induced by the aerobic exercise in the cardiomyocytes can influence the coronary capillarization of the myocardium (SCHEUER, 1982; LAUGHLIN and McALLISTER, 1992; TOMANEK, 1994; BROWN and HUDLICKA, 1999). In addition to influencing the capillaries, aerobic exercises can increase the caliber of the coronary artery, increase the vessel lumen, and improve the blood flow systemically in the whole body (KRAMSCH, ASPEN, ABRAMOWITZ et al., 1981; GIANATTASIO, 2001).

Rakusan and Wicker (1990) carried out a study on swimming rats and verified that the number of capillaries and arterioles of the left ventricle increased significantly in relation to the sedentary control group enabling improved myocardium blood perfusion.

The increased number of myocardium vessels of the LV, which is due to aerobic physical exercises, results from a mechanism of equilibrium such as that of the skeletal muscle.

## 4 Conclusion

### 4.1 Cardiac myocytes (or cardiomyocytes)

Numerous studies have been carried out on physical activity as a requirement to benefit health in modern society (CIOLAC, 2004). The aerobic physical activity has become an important ally to prevent heart diseases, which account for 30% of mortality worldwide (MION Jr., MACHADO, GOMES, MAM et al., 2004). With regard to the myocardium, studies have shown that aerobic exercises do not promote myocytes hypertrophy (LAUGHLIN, SCHAEFER and STUREK, 1992; DIFEE, SEVERSEN and TITUS, 2001; DIFEE and NAGLE, 2003).

The aerobic training increases the blood return and consequently imposes volume overload (increased preload) upon the heart leading to an increase in the end systolic blood pressure, which is a stimulus to sarcomere replication and increase in the left ventricular cavity size. It has been demonstrated that such long-duration activity increases the cell length in the left ventricle (MOORE, 1993; PALMER, 1998; PALMER and MOORE, 1999) causing a possible remodeling of myocyte density per unit area.

Since gyms have become hugely popular, strength exercises performed isolated and or together with other aerobic activities have become a current mania, and so they should be studied more deeply as a possible way of preventing and rehabilitating coronary diseases, just like it has been done for aerobic exercises. The effects of aerobic and strength exercises on the various components of the myocardium were analyzed in the present work.

Several studies on strength exercises with experimental animals using the protocol proposed by Hornberger and

Farrar (2004) are reported in the literature because they resemble the protocol for humans considering the muscular-skeletal responses in relation to muscular hypertrophy. In the experiments, the animal has to climb the steps of an inclined ladder towards the top. During the experiment, weight load is added to the animal tail. This protocol does not include any kind of reward or help from the researchers to the animals during the climbing exercise (climb toward the top of the ladder with weight load attached to the tail), and it does not require electric stimulation; thus, climbing to the top is a natural exercise.

There are no studies that have shown specifically the effect of strength exercises on the myocytes in protocols similar to that of Hornberger and Farrar (2004). Nevertheless, the data obtained for the intermittent exercises indicate an increase in myocyte area (NATALI, 2001, 2002), showing the myocyte transverse growth.

In an earlier study, Li, Lincoln, Mendelowitz et al. (1986) observed cardiac hypertrophy due to physical exercises such as swimming proving the effects of this kind of exercises on the myocardium components.

The increase in the myocytes area due to strength exercises results from pressure overload (increased post-load) upon the heart leading to an increase in the end systolic blood pressure, which is a stimulus to parallel sarcomere replication and increase in the left ventricular parietal width causing concentric hypertrophy.

The increase in the cardiac myocytes area can contribute to the increase in the intrinsic contractibility of these cells since it is a potential mechanism to explain the improvement in the myocardium contractile function induced by physical exercises (NATALI, 2004).

The functional overload imposed upon the heart by physical exercises is one of the most important stimuli to cardiac cell alterations. Therefore, when the heart is subjected to volumetric overload, it alters its architecture, due to hypertrophy, promoting a ventricular remodeling in order to normalize the parietal pressure according to Laplace's law.

An increase in the myocytes number can also occur (hyperplasia) since studies have demonstrated an actual increase in the cardiac myocyte number under certain conditions without physical activities (ANVERSA, PALACKAL, SONNENBLICK et al., 1990). There are no studies demonstrating the cell division of cardiomyocytes (hyperplasia) related to physical exercises. Further research is needed to elucidate this topic.

### 4.2 Blood vessels and collagen fibers

Regarding other components of myocardium, there is an increase in the vessel density due to aerobic exercises. The increase in the vessel density in the heart area has been frequently reported in the literature, and the reason given is that it is due to the neoformation mechanism. Neoformation occurs in healthy animals and in animals with compromised myocardium function causing an increase in the number of vessels for better blood distribution in the myocardium (LAUGHLIN and McALLISTER, 1992; TOMANEK, 1994; BROWN and HUDLICKA, 1999).

This is essential to improve coronary microcirculation since it is known that perfusion deficit compromises the heart function in coronary diseases (ANVERSA, HILER, RICCI et al., 1986). Long ago, some authors demonstrated

that aerobic exercises increase the capillary/myocyte ratio favoring cardiac perfusion (BLOOR and LEON, 1970).

Studies show that the myocardial collagen fibers (the main constituent of the interstitial space) are also influenced by physical exercises. It has been proved that that aerobic physical exercises reduce the age-related myocardial collagen increase (MAIFRINO and SOUZA, 2006) reducing the risk of heart diseases associated with aging, which can occur due to an increase in the myocardial collagen.

## References

- ANVERSA, P., HILER, B., RICCI, R. et al. Myocyte cell loss and myocyte hypertrophy in the aging rat heart. *Journal of American College of Cardiology*. 1986, vol. 8, p. 1441-1448.
- ANVERSA, P., PALACKAL, T., SONNENBLICK, EH. et al. Myocyte cell loss and myocyte cellular hyperplasia in hypertrophic aging rat heart. *Circulation Research*. 1990, vol. 67, p. 871-885.
- BALADY, GJ., CHAITMAN, B., DRISCOLL, D. et al. Recommendations for cardiovascular screening, staffing, and emergency policies at health/fitness facilities. In American College of Sports Medicine - ACSM. *ACSM's health/fitness facility standards and guidelines*. ACSM, 1998. (appendix)
- BJARNASON-WEHRENS, B., MAYER-BERGER, W., MEISTER, ER. et al. German federation for cardiovascular prevention and rehabilitation: recommendations for resistance exercise in cardiac rehabilitation. *European Journal of Cardiovascular Prevention & Rehabilitation*. 2004, vol. 11, no. 4, p. 352-361.
- BLAIR, SN., GOODYEAR, NN., GIBBONS, LW. et al. Physical fitness and incidence of hypertension in healthy normotensive men. *Journal of the American Medical Association*. 1984, vol. 252, no. 4, p. 487-490.
- BLOOR, CM. and LEON, AS. Interaction of age and exercise on the heart and its blood supply. *Laboratory Investigation*. 1970, vol. 22, p. 160-165.
- BROWN, MD. and HUDLICKA, O. Exercise, training and coronary angiogenesis. *Advances in Organic Biology*. 1999, vol. 7, p. 155-196.
- CAULFIELD, JB. and WOLKOWICZ, PE. Myocardial connective tissue alterations. *Toxicologic Pathology*. 1990, vol. 18, no. 4, p. 488-496. (part 1).
- CIOLAC, EG. and GUIMARÃES, V. Exercício físico e síndrome metabólica. *Revista Brasileira de Medicina do Esporte*. 2004, vol. 10.
- DIFFEE, GM. and NAGLE, DF. Exercise training alters length-dependence of contractile properties in rat myocardium. *Journal of Applied Physiology*. 2003, vol. 94, p. 1137-1144.
- DIFFEE, GM., SEVERSEN, EA. and TITUS, MM. Exercise training increases the Ca<sup>2+</sup> sensitivity of tension in rat cardiac myocytes. *Journal of Applied Physiology*. 2001, vol. 91, p. 309-315.
- DORN, GW., ROBBINS, J. and SUGDEN, PH. Phenotyping hypertrophy: eschew obfuscation. *Circulation Research*. 2003, vol. 92, no. 11, p. 1171-1175.
- FORJAZ, CLM. and NEGRÃO, CE. Sedentarismo. In MION, DJ. and NOBRE, F. (Orgs.). *Risco Cardiovascular Global*. São Paulo: Lemos, 1999. p. 139-162.
- GIANATTASIO, E. Radial artery flow-mediated dilatation in heart failure patients: effects of pharmacological and nonpharmacological treatment. *Hypertension*. 2001, vol. 38, no. 6, p. 1451-1455.
- GROSSMAN, W., JONES, D. and MCLAURIN, LP. Wall stress and patterns of hypertrophy in the human left ventricle. *Journal of Clinical Investigation*. 1975, vol. 56, p. 56-64.
- HORNBERGER Jr., TA. and FARRAR, RP. Physiological hypertrophy of the FHL muscle following 8 weeks of progressive resistance exercise in the rat. *Canadian Journal of Applied Physiology*. 2004, vol. 29, no. 1, p. 16-31.
- HUDLICKA, O. and BROWN, MD. Postnatal growth of the heart and its blood vessels. *Journal of Vascular Research*. 1996, vol. 33, no. 4, p. 266-287.
- KRAMSCH, DM., ASPEN, AJ., ABRAMOWITZ, BM. et al. Reduction of coronary atherosclerosis by moderate conditioning exercise in monkeys on an atherogenic diet. *New England Journal of Medicine*. 1981, vol. 305, no. 25, p. 1483-1489.
- LAUGHLIN, MH. and McALLISTER, RM. Exercise training-induced coronary vascular adaptation. *Journal of Applied Physiology*. 1992, vol. 73, p. 2209-2225.
- LAUGHLIN, MH., SCHAEFER, ME. and STUREK, M. Effect of exercise training on intracellular free Ca transients in ventricular myocytes of rats. *Journal of Applied Physiology*. 1992, vol. 73, p. 1441-1448.
- LI, XX., LINCOLN, T., MENDELOWITZ, D. et al. Age-related differences in effect of exercise training on cardiac muscle function in rats. *American Journal of Physiology*. 1986, vol. 251, p. H12-H18.
- MAFRINO, LBM. and SOUZA, RRD. NADPH diaphorase positive cardiac neurons in the atria of mice: a morphoquantitative study. *BMC Neuroscience*. 2006, vol. 7, no. 1, p. 10.
- MION Jr. D., MACHADO, CA., GOMES, MAM. et al. *IV Diretrizes Brasileiras de Hipertensão Arterial*. Sociedade Brasileira de Cardiologia, 2004.
- MITTLEMAN, MA., MACLURE, M., TOFLER, GH. et al. Triggering of acute myocardial infarction by heavy physical exertion: protection against triggering by regular exertion. *New England Journal of Medicine*. 1993, vol. 329, no. 23, p. 1677-1683.
- MOORE, RL. and KORZICK, DH. Cellular adaptations of the myocardium to chronic exercise. *Progress in Cardiovascular Diseases*. 1995, vol. 37, p. 371-396.
- MOORE, RL. Chronic exercise alters contractility and morphology of isolated rat cardiac myocytes. *American Journal of Physiology*. 1993, vol. 264, p. C1180-C1189.
- NATALI, AJ. Different regional effects of voluntary exercise on mechanical and electrical properties of rat ventricular myocytes. *The Journal of Physiology*. 2002, vol. 541, p. 863-875.
- NATALI, AJ. Effects of chronic exercise on cardiac myocytes: a review about mechanical adaptations. *Revista Brasileira de Ciência e Movimento*. 2004, vol. 12, no. 1, p. 91-96.
- NATALI, AJ. Regional effects of voluntary exercise on cell size and contraction-frequency responses in rat cardiac myocytes. *Journal of Experimental Biology*. 2001, vol. 204, p. 1191-1199.
- NATIONAL INSTITUTES OF HEALTH - NIH. Physical activity and cardiovascular health. *The Journal of the American Medical Association*. 1996, vol. 276, p. 241-246.
- OLSON, MOJ. Sensing cellular stress: another new function for the nucleolus? *Science STKE*. 2004, vol. 2004, no. 224, p. 10.
- PAFFENBARGER Jr., RS., HYD, RT., WING, AL. et al. The association of changes in physical activity among men. *New England Journal of Medicine*. 1993, vol. 328, p. 538-45.
- PALMER, BM. and MOORE, RL. A model of isolated cardiac myocyte predicts an increase in myocyte compliance with exercise training. *The Physiologist*. 1999, vol. 39, p. A40.

PALMER, BM. Shortening and  $[Ca^{2+}]$  dynamics of left ventricular myocytes isolated from exercisettrained rats. *Journal of Applied Physiology*. 1998, vol. 85, p. 2159-2168.

RAKUSAN, K. and WICKER, P. Morphometry of the small arteries and arterioles in the rat heart: effects of chronic hypertension and exercise. *Cardiovascular Research*. 1990, vol. 24, no. 4, p. 278-284.

SANTARÉM, J. *Atualização em exercícios resistidos: adaptações cardiovasculares*. Available from: <www.saudetotal.com.br>.

SCHEUER, J. Effects of physical training on myocardial vascularity and perfusion. *Circulation*. 1982, vol. 66, p. 491-496.

TOMANEK, RJ. Exercise-induced coronary angiogenesis: a review. *Medicine and Science in Sports and Exercise*. 1994, vol. 26, p. 1245-1251.

WORLD HEALTH ORGANIZATION - WHO. *Definition, diagnosis and classification of diabetes mellitus and its complications*. WHO, 1999. p. 31-33. (part 2).

Received February 16, 2009

Accepted July 20, 2009