Changes in Cardiac Geometry Due to Hypertrophy

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Introduction

For any given body size, men have larger hearts than women, athletes have larger hearts than nonathletes, and often times, an enlarged heart is a symptom of an underlying disorder that is causing the heart to work harder than normal. This review will emphasize the differences between a pathologically enlarged heart and an athletically enlarged heart.

Pathologically induced hypertrophy (myopathy)

When heart cells get bigger, (often is the case when heart disease is present) the total heart works less efficiently. Some people suffer from conditions like hypertrophic cardiomyopathy, which includes significant heart muscle enlargement, and can be genetic or caused by high blood pressure. Cardiomyopathy decreases the size of the heart's chambers, reducing blood flow. Hypertrophy, or thickening, of the heart muscle can occur in response to increased stress on the heart. The most common causes of Cardiomyopathy are related to increased blood pressure. The extra work of pumping blood against the increased pressure causes the ventricle to thicken over time, the same way a body muscle increases in mass in response to weightlifting.

Cardiomyopathy can occur in both the right and left atrium and the right and left ventricles. Blood travels through the right ventricle to the lungs. If conditions occur that decrease pulmonary circulation, extra stress can be placed on the right ventricle, and can lead to right ventricular myopathy. The most common causes of right ventricular myopathy are diseases that damage the lungs like emphysema and cystic fibrosis. These diseases destroy blood vessels in the lungs, causing increased pressure in the remaining vessels. Conditions that decrease oxygen levels, such as chronic bronchitis and sleep apnea, also lead to right

ventricular myopathy. Mild forms of right ventricular myopathy may not show any symptoms at all and in the severe form, patients will experience shortness of breath along with chest pains and pressures. Additionally, the sensation that the heart is beating rapidly and forcefully is also felt.

Left ventricular myopathy

When the heart has trouble pumping blood, it might start beating more rapidly or at irregular intervals. Drugs like beta-blockers, ACE inhibitors, angiotensin receptor blockers, diuretics, and digoxin may be prescribed. If drugs can not treat the cause, there may be a need to implant a defibrillator or a pacemaker in the patient. The way that the heart pumps blood depends on its shape and contractility. If these changes are due to cardiomyopathy and the thickening of the heart walls, then the heart's left ventricle (LV) will not pump blood as efficiently. This can lead to a reduced ejection fraction which does not allow enough blood to be pumped throughout the body. As the ventricle becomes thicker, it starts to lose its elasticity. Thus, filling during diastole becomes more difficult and depends on atrial systole (26). As a consequence, LV end-diastolic pressure and left atrial pressure increases, resulting in enlargement of the left atrium. Oxygen demand and basal coronary blood flow increases as myocardial myopathy increases. Only with exercise or severe left ventricular hypertrophy (LVH) will subendocardial blood flow be jeopardized, potentially causing myocardial infarction (MI) and fibrosis. Left ventricular hypertrophy is the most common type of hypertrophy seen in both pathologically and athletically enlarged hearts. Pathological left ventricular hypertrophy develops in response to some factors, such as high blood pressure and obesity, which requires the left ventricle to work harder. Left ventricular myopathy usually develops gradually and as the workload increases, the walls of the chamber grow thicker, lose elasticity and

eventually may fail to pump with as much force as that of a healthy heart. A person may experience no signs or symptoms, especially during the early stages of the condition. As left ventricular myopathy progresses and complications develop, symptoms such as shortness of breath, chest pain, rapid, fluttering or pounding heartbeats may occur. Left ventricular myopathy changes both the structure and function of the chamber: The enlarged muscle loses elasticity and stiffens, preventing the chamber from filling properly and leading to increased pressure in the heart (29). There are many complications that can occur in those with left ventricular myopathy. These may include an increased inability of the heart to pump enough blood through the body, which is considered heart failure.

Left ventricular diastolic function has been studied by use of mechanocardiography, radionuclide techniques, imaging echocardiography, and Doppler velocimetry. Mechanography is the indirect recording of the pulsatile movements of arteries, veins, cardiac apex and pericardium. The changes in contour of these pulsations as well as the measurement of the duration of the various phases during the cardiac cycle can provide information about cardiovascular dynamics and myocardial performance (28). Radionuclide ventriculography provides quantitative measures of biventricular function and regional wall motion. Radionuclide imaging is used for analyzing diastolic function, parametric imaging and uses methods for calculating absolute ventricular volumes. Radionuclide imaging uses a gamma camera to create an image following injection of radioactive material. This test is done to evaluate coronary artery disease (CAD), valvular or congenital cardiac disorders, cardiomyopathy, and other cardiac disorders (24). Echocardiography is the principal method used to diagnose LVH (33). Echocardiography is a complex, noninvasive imaging technique that creates ultrasound images of heart structures and produces accurate assessment of the blood flowing through the heart, using pulsed or continuous wave doppler ultrasound. This allows assessment of both

normal and abnormal blood flow through the heart. Color Doppler as well as spectral Doppler is used to visualize any abnormal communications between the left and right side of the heart, any leaking of blood through the valves, and can estimate ejection fraction and how well the valves open and close (3, 34). The use of Stress Echocardiography may also help determine whether any chest pain or associated symptoms are related to heart disease. The electrocardiogram (ECG) can show signs of increased voltage from the heart in individuals with LVH, so this type of test is often used as a screening test to determine if the patient should undergo further testing (30). A 12 lead electrocardiogram showing hypertrophy of the ventricles, will produces QRS complexes that have exaggerated amplitude. With left ventricular hypertrophy, there is a very tall R wave in lead V5, which is the lead over the left ventricle. The second type of test used in determination of ventricular hypertrophy is if the V1 electrode produces a deep S wave. The formula for checking for ventricular hypertrophy is simply to add the depth of the S wave in V1and V2 to the height of the R wave in V5 and 6. If the sum of the two numbers is greater than 35, LVH is present. The determination of the heart's electrical axis is an important part of the electrocardiogram diagnosis. The direction of the depolarization waves depends on the orientation of the heart during the particular instant of the cardiac cycle being considered. This can be done by observing the voltages of the QRS complex from two different perspectives using two different leads. Lead I provides a horizontal axis (from left arm to right arm). Lead III has an axis of about 120 degrees (from left arm to left leg). Using the recordings from leads I and III, the normal mean electrical axis of the ventricles is about 59 degrees. The axis of the heart shifts toward the hypertrophied ventricle for two reasons: 1. More muscles exist on the hypertrophied side, which allows excess generation of electrical potentials on this side. 2. More time is required for the depolarization to travel to the hypertrophied ventricle.

Normal electrical axis is classified as -30 degrees to 90 degrees. Right ventricular hypertrophy is associated with right axis deviation, which is 90 degrees to 180 degrees (19). Right axis deviation may be normal in the young and thin individuals. Right axis deviation is common in taller individuals, because the apex of the heart generally moves lower into the thorax. Other causes of right axis deviation include chronic obstructive pulmonary disease, pulmonary embolism, and Wolf- Parkinson- White syndrome. Left ventricular hypertrophy is associated with left axis deviation, which is classified as 30 degrees to -90 degrees (19). Left ventricular hypertrophy, where the left ventricle is enlarged, generates more electrical activity, so the heart axis is pulled to the left. Left axis deviation is usually not problematic unless there are other diseases present, like a left anterior block. Left axis deviation can be seen in older individuals and those with obesity. In obese people, the diaphragm is often positioned high, pushing the heart upwards and rotating its electrical axis to the left. A study conducted by Peter M. Okin et al. (20) examined gender differences, electrocardiographic voltages, and QRS duration to differences in cardiac dimensions and body size between men and women and gender differences in test performance of ECG criteria for the detection of echocardiographic left ventricular hypertrophy. 389 subjects (112 women and 277 men) were chosen to take part in the study. ECG voltage-duration was calculated as the product of QRS duration and voltages. Some subjects had normal left ventricular mass and some with left ventricular hypertrophy. While men had longer QRS duration, higher Cornell voltage (a calculation method for LVH using an ECG test (RaVL + S-wave amplitude in V(3) [SV(3)]), higher 12-lead sum of QRS voltage, and higher Cornell and 12-lead voltage-duration products than did women (27). Standard 12-lead ECG measurements indicate that men have significantly longer QRS durations and greater ECG voltages than do women. Although these differences in ECG findings have been attributed to gender differences in body size and left

ventricular mass, it remains uncertain whether gender differences in QRS durations and voltages can be fully attributed to differences in cardiac dimensions and body size between men and women. Men and women with and without a cardiomyopathy were similar with respect to age, but men had a significantly higher mean body surface area, greater left ventricular wall thickness, greater left ventricular internal dimension during diastole, and higher left ventricular mass. Gender-specific indexed left ventricular mass partitions showed a higher prevalence of left ventricular hypertrophy in women than men.

When analyzing the results of an ECG, conducted on a static or weight lifting/ power athlete, you may find an athlete's heart to have some abnormal rhythms, abnormal conduction alterations, changes of the QRS complex, and repolarization abnormalities (14). ECG tracings conducted on an athlete may show very large S-T segment elevation with upward convexity and upright diphasic T waves. The most common training-induced ECG changes in athletes are first-degree AV block, second-degree AV block, QRS duration <0.12 s, early repolarization, and criteria for left ventricular hypertrophy. Another study conducted by Toufan et al. (31), assessed the ECG, echocardiography, and heart rate variability(HRV) in a group of dynamic and static type athletes. Fifty professional athletes (20 static and 30 dynamic exercise athletes) and 50 healthy nonathletes (control group) were recruited. Standard 12-lead ECG and transthoracic echocardiography was performed on all athletes and the control group. Through echocardiography, variables including left ventricular (LV) end-diastolic/systolic diameter, LV mass, and left atrial volume index were measured. In addition, both the athletes and the control group underwent ECG monitoring for 15 minutes and several parameters related to HRV (time and frequency domain) were recorded. The most common ECG abnormalities among the athletes were sinus bradycardia and incomplete right bundle branch

block. LV end-diastolic diameter and left atrial volume index were significantly greater in the dynamic athletes. LV end-systolic diameter was significantly lower in the static group. LV mass of the dynamic and static athletes was significantly greater than that of the controls. Among the ECG monitoring findings, the dynamic athletes had lower systolic blood pressure than the controls and heart rate was lowest in the control group. These findings are suggestive of cardiac adaptation of the static athletes for their particular type of exercise, ie, strength training exercises. It also indicates that during dynamic exercise, the LV withstands repetitive stress, which leads to an increase in LV mass.

Athletically enlarged hearts

Athletic heart syndrome (also known as athlete's heart, athletic bradycardia and/ or exercise-induced cardiomegaly) is a non-pathological condition, in which the human heart is enlarged, and the resting heart rate is lower than normal (10). If expansion happens uniformly, and the chamber thickness and chamber size keep the same relative proportions, it can be a healthy adaption. However, if the walls thicken and the chamber size becomes smaller relative to the expanding walls, it is more likely a sign of disease (7). It typically takes more than three hours of exercise per week to observe changes in heart rate, aerobic power, and left ventricular mass (5). Enhancing cardiac output allows one to maintain lower heart rates during physical activity. After three or more months of training, increased cardiac output, increases in maximum oxygen consumption, stroke volume, and systolic blood pressure, are associated with decreased peripheral vascular resistance. This enables the athlete to sustain the same exercise intensity at a lower heart rate. Athlete's heart is common in athletes who routinely exercise more than an hour a day, and occurs primarily in endurance athletes. Athlete's heart is generally believed to be benign and most often does not have any physical symptoms, although an indicator would be a consistently low resting heart rate. Athletes often do not

realize they have the symptoms of athlete's heart unless they undergo certain medical tests because athlete's heart is a normal, physiological adaptation of the body to the stresses of physical conditioning and aerobic exercise. This can make it difficult to determine if the cause of cardiac enlargement is due to a hypertrophic cardiomyopathy, or if it is due to a healthy adaptation from intensive training over a long period of time (2). Echocardiography is the preferred imagistic technique to differentiate between physiological and cardiomyopathy (12, 13, 33). The thickness of the left ventricular wall is usually less than 1.3 cm in thickness. Well-trained athletes may have a left ventricular wall thickness of up to 1.5 cm (22). This adaptation helps with the generation of additional cardiac output during exercise. A larger heart results in higher stroke volume and cardiac output, as more blood is pumped out with each beat. The raised cardiac output allows the heart to beat more slowly, resulting in bradycardia. Athlete's heart does not pose any health threats to the athlete (8). It is recommended that the athlete see a physician to be sure that the symptoms are due to athlete's heart and not another heart disease, such as cardiomyopathy.

Changes in the structure of the heart may not be the same for every athlete. Different types of training, such as aerobic and anaerobic exercise can result in a variety of changes in the heart (32). Endurance training exerts a volume overload on the left ventricle and increases filling pressures that can lead to increased stretching force on the myocardium. Due to the Frank-Starling mechanism, the heart muscle is able to increase contractile force when the ventricular wall is stretched. The Frank–Starling law of the heart states that the stroke volume of the heart increases in response to an increase in the volume of blood filling the heart, (end diastolic volume) when all other factors remain constant. The increased volume of blood stretches the ventricular wall, causing cardiac muscle to contract more forcefully (Frank-Starling mechanisms). The stroke volume may also increase as a result of greater contractility

of the cardiac muscle during exercise, independent of the end-diastolic volume (18). The main features that aerobic exercise has on the heart are increased ventricular stroke volume and cardiac output which improves aerobic capacity. A routine of regular exercise is highly effective for prevention and treatment of many common chronic diseases and improves cardiovascular health. Chronic training and competing in excessive endurance events such as marathons, ultramarathons, ironman distance triathlons, and very long distance bicycle races, may cause transient acute volume overload of the atria and right ventricle, with transient reductions in right ventricular ejection fraction, all of which return to normal within one week. Over months to years of repetitive injury, this process, in some individuals, may lead to patchy myocardial fibrosis, particularly in the atria, interventricular septum, and right ventricle, creating a substrate for atrial and ventricular arrhythmias. Additionally, long-term excessive sustained exercise may be associated with coronary artery calcification, diastolic dysfunction, and large-artery wall stiffening. Adverse effects of both short-term intense physical activity and cumulative endurance exercise are most apparent in the right sided cardiac chambers. Cardiac output at rest is approximately 5 L/min but typically increases 5-fold to about 25 L/min during vigorous physical activity (4). A high degree of endurance training increases parasympathetic tone and decreases sympathetic tone of the heart, which is associated with greater aerobic power.

With anaerobic training, skeletal and cardiac muscles adapt themselves in response to the type of training. Resistance training results in an alteration with elevated of blood pressure, leading to pressure overload in the heart. This leads to an increase in left ventricular wall thickness (15). In anaerobic exercise, like weight lifting, the force generated on the heart is different than with aerobic activity (11). Weight lifting is a pressure overload that forces Valsalva while aerobic exercise is a low pressure volume overload. At the beginning of a

weight lifting movement, pressure in the chest cavity increases due to the compression of air. The heart rate does not increase, but blood pressure increases due to intrathoracic pressure as it is forced from the lungs into the heart. The amount of blood coming into the heart (venous return) decreases. Less blood then goes out of the heart. When the breath is released, chest cavity pressure decreases, and blood flows normally from the lungs into the heart. Blood pressure may rise slightly above normal before returning to baseline. Heart rate also returns to normal (25, 6, 9). A study by Abinader et al. (1) explored the effects of isometric stress on left ventricular filling in athletes with isometric or isotonic training and then compared them to both hypertensive and normal controls. Thirty-eight males, comprising 13 long-distance runners, eight weight-lifters, eight hypertensive patients and nine age-matched healthy male controls were studied. All the athletes were national competitive sportsmen engaged in periodic sporting events and were examined to exclude usage of performance-enhancing drugs. Long-distance runners were at national level actively engaged in running at least 60 km per week and weight-lifters had been training for at least 4 years for several hours per session at least three times a week, and each could bench-press at least 100 kg. Blood pressures in the hypertensive patients had been elevated for 3-5 years before the study, and were found repeatedly to have systolic and diastolic pressures greater than 140 and 90mmHg, respectively. Normal subjects were sedentary healthy age-matched males. The isometric handgrip test was performed in the study and control groups. The resting blood pressure was recorded before exercise and afterwards. To determine maximal hand-grip force, at least 3 min intervals three brief less than 10 second intervals. Maximal voluntary right hand contractions were performed. Then the subjects maintained 50% of the maximal force for 90 seconds. Electrocardiographic monitoring was performed during the study. Measurement of heart rate, cuff arm blood pressure, and heart rate systolic blood pressure product were also

obtained. Using two dimensionally directed M-mode echocardiographic recordings, the diameters of the left ventricle at end-diastole and end-systole were measured while the subject was at rest. Left ventricular septal and posterior wall end-systolic and end-diastolic thickness was measured from the parasternal short axial views just below the level of the mitral valve. Left ventricular mass and mass index were also calculated while at rest. The results found that left ventricular end-systolic diameters were smaller in weight-lifters than in the other groups, while left ventricular end-diastolic diameter and fractional shortening, in runners, were higher than in the other groups. The end-diastolic thickness of the interventricular septum and left ventricular posterior wall were similar in the runners, weight-lifters and hypertensive groups and were higher than in normal subjects. Left ventricular mass and mass index were highest in runners and weight-lifters. Thus, the runners had left ventricular hypertrophy with a larger end diastolic diameter and when corrected for body surface area, while weight-lifters had ventricular hypertrophy and a smaller end-diastolic diameter. Resting peak wall stress was highest in hypertensive patients and lowest in weight-lifters. Heart rates in runners at rest and during isometric stress were significantly lower than in the other groups. Blood pressure was highest at rest in hypertensive patients, while during isometric stress weight-lifters achieved similar blood pressures as in the hypertensive group. Thus runners had left ventricular hypertrophy associated with larger end-diastolic diameters and low resting peak meridional wall stress while weight-lifters had left ventricular hypertrophy, normal end-diastolic diameters and the lowest peak meridional wall stress. The current study was important because the hemodynamic effects in athletes usually happens during strenuous exercise, cardiac output increases fourfold; mean arterial blood pressure and left ventricular filling pressure increase. Since diastolic dysfunction often precedes systolic dysfunction, the present study assessed left ventricular filling parameters both at rest and during isometric stress, which predominantly

increases left ventricular afterload in runners. Left ventricular hypertrophy was associated with larger end-diastolic diameters, while in weight-lifters; the hypertrophy was associated with normal end-diastolic diameters. Left ventricular masses and mass indices were similar in runners and weight-lifters and were significantly higher in athletic subjects than in hypertensive patients and normal subjects. Peak meridional wall stress was lowest in weight-lifters, while in hypertensive patients it was similar to normal controls. Systolic blood pressure was the parameter which showed the biggest difference and it seems to be the factor to explain the differences in ventricular filling in weight-lifters and runners.

There is a growing urgency to make it mandatory for both professional and school based athletes to be tested for any cardiac abnormalities (4). Enlarged pathological hearts, (and less frequently,) idiopathic dilated cardiomyopathy represent pathologic causes responsible for sudden, unexpected death in young athletes (17). Therefore, extreme alterations in left ventricular hypertrophy observed in highly trained athletes' raises several critical issues, including the clinical significance and long-term consequences of the hypertrophy induced by training and the likelihood of complete reversibility after deconditioning. There have been several well-known athletes experiencing sudden unexpected death due to cardiac arrest, such as U.S. Olympic volleyball player Flo Hyman in 1986; college basketball player Hank Gathers in 1990; and professional basketball players Pete Maravich in 1988 and Reggie Lewis in 1993. In 2007, during the US Olympic marathon trials in New York City, Ryan Shay collapsed approximately 5 ½ miles (8.9 km) into the race. Doctors at the hospital who examined him reported that he died of a massive heart attack, due to a pre–existing enlarged heart. Sudden cardiac death among marathoners is very rare, with 1 event per 100,000 participants (16). Further investigation needs to be done to identify at-risk individuals and design physical fitness regimens for cardiovascular health in athletes. Most

school systems require athletes to see a doctor and go through a basic physical before they can play school sports, but it is still not required for most athletes to participate in extreme sports. Screening athletes for heart problems is essential to diagnose specific heart conditions and prevent sudden cardiac death. These conditions can be distinguished with an echocardiogram, but sometimes other investigations might be required such as exercise stress testing. Screening athletes for heart problems is essential to diagnose specific heart conditions Some of the causes of sudden cardiac death associated with athletic activity include cardiomyopathy, aortic stenosis, arrhythmia, coronary artery disease, aortic aneurysm and congenital heart disease. The distinction between athlete's heart and cardiomyopathy can sometimes be difficult even with the use of an echocardiogram but a detailed assessment including family history, electrocardiogram, exercise stress testing should be able to distinguish between the two.

Treatment for cardiomyopothy

Treatment for pathological left ventricular hypertrophy (cardiomyopothy) depends on the overall health of the patient as well as the symptoms the patient shows. Medical tests and medications can be used to successfully improve heart size and function. Prevention or regression of LVH is a major therapeutic target whether achieved by pharmacological, mechanical, surgical, or genetic means (9). There has been clinical evidence that regression of LVH reduces morbidity and mortality and improves prognosis. Some of the most common medical treatments for cardiomyopathy include the use of prescription medications or surgical intervention. Prescription medications are often the first line of medical treatment for cardiomyopathy. These medications are designed to relax the heart muscle and slow the heart rate so that the heart can pump blood throughout the body in a more efficient manner. Some

high blood pressure drugs may prevent further enlargement of left ventricle muscle tissue. One blood pressure medication that is often used is Angiotensin-converting enzyme (ACE) inhibitors. These drugs widen, or dilate blood vessels to lower blood pressure, improve blood flow and decrease the workload on the heart. Angiotensin II receptor blockers (ARBs), have many of the beneficial effects of ACE inhibitors, but they do not cause a persistent cough. They may be an alternative for people who cannot tolerate ACE inhibitors. Thiazide diuretics act on the kidneys to help the body eliminate sodium and water, thereby reducing blood volume. Examples include enalapril (Vasotec), lisinopril (Prinivil, Zestril) and captopril (Capoten). Beta blockers are used to slow the heart rate, reduce blood pressure and prevent some of the harmful effects of stress hormones. Surgical procedures are frequently a necessary part of treatment for cardiomopathy. Devices such as a pacemaker or defibrillator may be surgically implanted into the chest cavity in order to regulate the heartbeat. Other surgical procedures may involve the removal of excess cardiac tissue or the destruction of tissue through the use of injections. For those with cardiomyopathy, there are procedures to reduce sections of heart muscle and improve heart function. One of these requires open heart surgery. A pacemaker may be implanted to relieve symptoms and improve hemodynamics in patients. As reported in a study conducted by Robert A. O' Rourke et al. (21), dual-chamber pacemakers (VDD) were implanted in 9 patients with exertional dyspnea due to hypertensive left ventricular hypertrophy with supranormal systolic ejection (mean ejection fraction 85%) and distal cavity obliteration (hypertensive hypertrophy with cavity obliteration. In this small number of patients, intrinsic atrial rate was sensed and ventricular pre-activation achieved by shortening the atrial-ventricular delay. All patients had concentric hypertrophy with increased septal and left ventricular wall thickness. VDD pacing was randomized to "on" or "off" for 3-month periods followed by 6 additional pacing "on" months. With pacing on, exercise duration

rose by an average of 82% and maximum oxygen consumption increased 24% above that measured when pacing was off. Improved exercise capacity persisted after 1 year (pacer on). Clinical symptoms of stimulated systemic reserve (stroke volume and cardiac output) increased when pacing was on. The study concluded that pacing should never be considered for a primary form of therapy in patients with cardiomyopathies. The appropriate AV interval, which is usually short, is crucial to the success of dual chamber pacing in the myopathic heart. Using pace making for those with severe cardiomyopathies is not a useful form of treatment.

Cardiac rehabilitation in the form of a structured exercise program can help reduce symptoms of cardiac enlargement. Cardiac rehabilitation is a professionally supervised program to help people recover from heart attacks, heart surgery and percutaneous coronary intervention procedures such as stenting and angioplasty. Cardiac rehab programs usually provide education and counseling services to help heart patients increase physical fitness, reduce cardiac symptoms, improve health and reduce the risk of future heart problems, including heart attack. Treatment measures that are often recommended for use at home include healthy dietary habits, including a low-sodium, low fat diet, adequate amounts of rest, not smoking, and the avoidance of alcohol.

Treatment is usually not necessary for those with non pathological, athletically induced LVH. Deconditioning from exercise for a period of approximately three to nine months, followed by a gradual return to athletic training will allow the heart to return to its regular size (21, 23). Pelliccia et al. (21) evaluated 40 elite male athletes who had shown marked LV cavity enlargement. The 40 subjects had been formerly engaged in rowing, canoeing, cycling, and tennis and middle-distance running. All were elite competitors, including 21 finalists or medalists at Olympic Games or World Championships. Electrocardiograms and

echocardiograms were obtained to evaluate LV cavity dimensions and anterior ventricular septal and posterior free wall thickness. LV ejection fraction was calculated from end-diastolic and end-systolic volumes in the apical 4-chamber view. Parameters of LV filling were obtained with pulsed Doppler echocardiography. Over the follow-up period, they found that LV end-diastolic cavity dimension decreased by 7%. Individual subject analysis showed most of these athletes (92%) had reduction in cavity dimension, whereas the remaining 3 subjects showed little or no change, no athlete showed an increase in cavity size with detraining. However, absolute cavity dimensions remained enlarged in most of the deconditioned athletes, including 22% with more substantial dilatation, consistent in absolute dimensional terms with dilated cardiomyopathy. The study concluded that substantial LV cavity dilatation was only partially reversible after long periods of deconditioning. Although reduction in cavity dimensions occurred in 90% of the former athletes, the final dimensions remained markedly enlarged in 20%. LV mass remained increased in about half of the former athletes. Therefore, because the athletes had been exposed to particularly intense and prolonged athletic training at an elite level, they could not exclude the possibility that the incomplete LV remodeling may represent a long-term, irreversible consequence of extreme athletic conditioning. Although they found no clinical or echocardiographic evidence of systolic or diastolic LV dysfunction, cardiac symptoms, or impaired physical performance in the former athletes, the possibility that this marked residual chamber enlargement may represent a subtle cardiomyopathic process that could ultimately lead to clinical consequences later in life cannot be excluded. During the post-exercise period, the cardiac geometric dimensions can, in some cases be restored, but with this stretch of the chambers and reestablishment of the chamber geometry, some individuals may be prone to the development of chronic structural changes including chronic dilatation of the right ventricle and right atrium, with patchy myocardial scarring in response to the recurrent

volume over- load and excessive cardiac strain. These abnormalities are often asymptomatic and probably develop over many years, but they may predispose to serious arrhythmias such as atrial fibrillation and/or ventricular arrhythmias (16).

There is no limited life expectance for a person living with an athletically enlarged heart or a pathological condition. A person can live with an enlarged heart or a cardiomyopathy from weeks, to months, to years. It all depends on how well the heart functions. Some patients with left ventricular hypertrophy may have a decreased longevity due to complications like congestive heart failure, heart attack, ischemic heart disease, sudden cardiac arrest, arrhythmia and heart failure. A patient may also have to live with symptoms including chest pain, shortness of breath, dizziness, palpitations, and fainting. Even though the heart may be enlarged, if it is well taken care of, a person can still live a normal healthy life. The heart weakens as it enlarges, but if it is detected early enough; deconditioning or treatment interventions can be done to reduce the chance of the condition to worsen.

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