LEFT VENTRICULAR MASS AND LEFT VENTRICULAR DIASTOLIC FUNCTION IN TRAINED ATHLETES

Thesis
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The Master Degree In Cardiology

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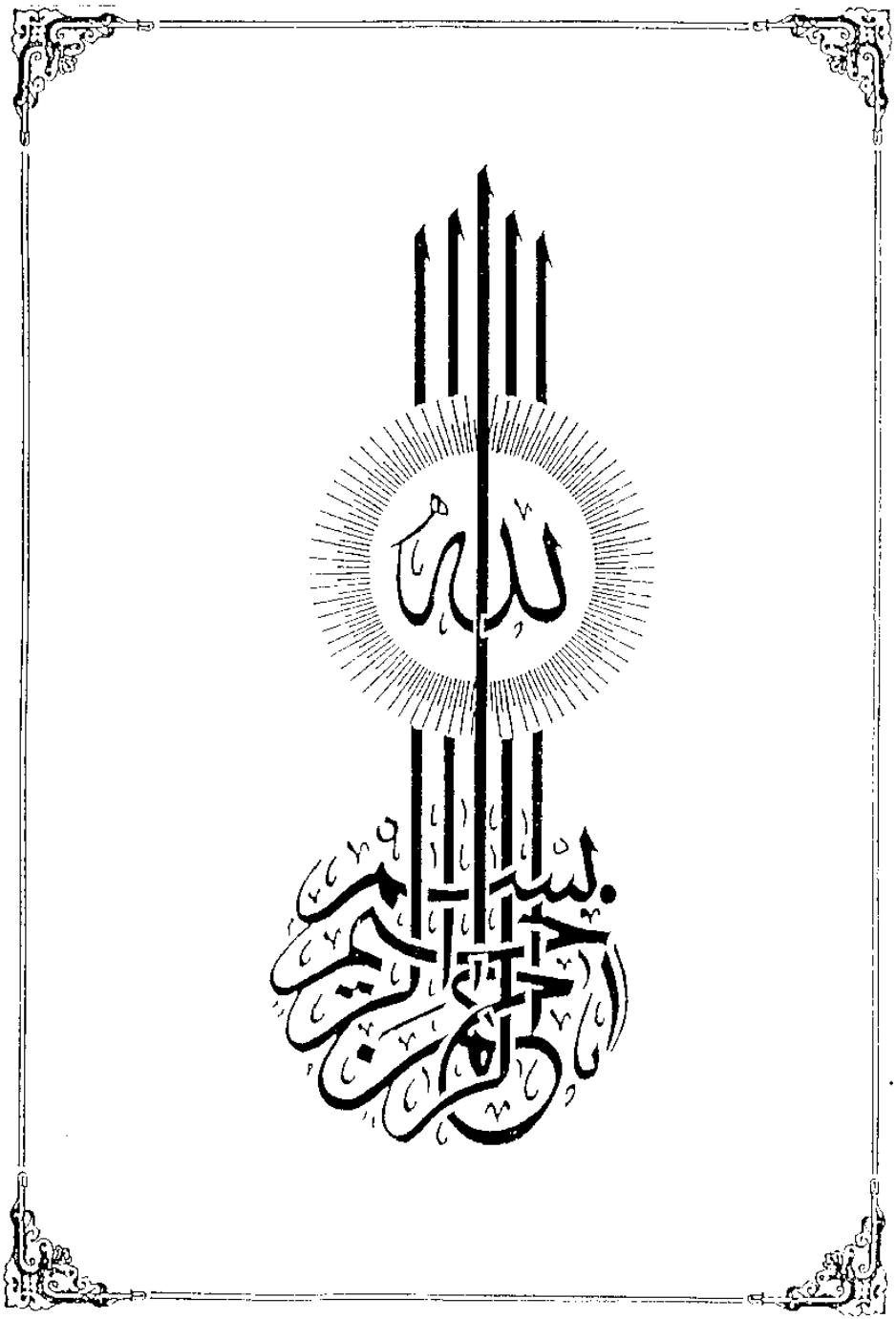

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CONTENTS

Introduction and Aim of the Work

Cardiovascular adaptation to exercise can occur with chronic repetitive exercise. This adaptation occurs with any of the two types of exercise, the isometric and the dynamic exercise, although there is some difference in the form of adaptation between the two types of exercise.

Physiologic hypertrophy had been known to occur in the athlete's heart. With the advent of echocardiography as an easy and non-invasive technique, it has provided us with a more refined and precise concept of the normal athlete's heart.

It is well known that the pathological hypertrophy causes diastolic dysfunction of the heart. So, it is of great importance to study the diastolic function of the athlete's heart. Studying the diastolic function of the heart by Doppler echocardiography is an easy, non-invasive and reliable method. This study may explain whether the diastolic dysfunction of the pathological hypertrophy is due to hypertrophy itself or the disease which causes it.

Aim of the Work

The aim of this work was to study the left ventricular dimensions, mass, and mass index in dynamically trained athletes by echocardiography and to assess their left ventricular diastolic function during rest and exercise by Doppler echocardiography.

Physiologic Consequences of Systematic Training

Exercise training results in physiologic changes that affect the entire body. There are several aspects of exercise training that influence the physiologic response. The type of training is important as well as intensity and duration, and the frequency with which it is performed. The end result of training is an increased ability of the human body to perform muscular activity. It is of great importance for the cardiologist to recognize the physiologic consequences of training and to distinguish them from apparently similar changes that occur in some disease states ***(Crawford, 1992)***.

The most important determinant of cardiovascular response to exercise is the type of exercise performed. In general, exercise can be divided into dynamic and static exercise ***(Asmussen, 1981)***. Dynamic exercise is when there is a change in muscle length, with little or no change in tension. This is also referred to as isotonic exercise. An excellent example of which is running. With static exercise, there is little or no change in muscle length, but a marked increase in tension. This is referred to as isometric exercise and an excellent example of this would be hand grip. Most exercise represents some combination of these two forms. At one extreme, would be running, which is mainly isotonic but does require some static muscular activity to maintain the upright posture, weight lifting is a good example of largely isometric exercise. Although there are differences in the response of these two types of exercise, there are similarities as well.

Acute Response to Dynamic Exercise:

In order to appreciate the effects of habitual training, it is important to understand the acute physiologic response to exercise. Increased muscular activity requires oxygen delivery to support the demands of aerobic metabolism. Thus, a major characteristic of the acute response to exercise is an increase in oxygen consumption (VO_2). $VO_2 = CO \times AVO_2$ where CO is cardiac output and AVO_2 is the arteriovenous oxygen difference. Thus VO_2 can increase by increasing cardiac output, AVO_2 or both. CO = HR X SV, where HR is heart rate and SV is stroke volume. Consequently, cardiac output can increase by either an increase in heart rate, an increase in stroke volume or both.

AVO_2 is determined by the oxygen extraction across the capillary bed and the distribution of blood flow to the exercising muscle bed. Thus, AVO_2 can increase by increasing muscle oxygen extraction or increased blood flow to muscles. The physiologic response to exercise results in changes in all these parameters, which ultimately result in an increase in VO_2. There is a linear relationship between VO_2 and workload during dynamic exercise until maximum VO_2 is reached . Maximum VO_2 is a measure of aerobic capacity and is determined by the maximal oxygen delivery and extraction by working muscles. Maximal VO_2 is often used as a measure of maximal exercise performance capabilities because it is independent of the type of exercise being performed. Maximal VO_2 is often expressed as milliliters per kilogram per second to adjust for body size and is highly reproducible from day to day in a given individual *(Crawfard, 1992)*.

In addition to training, maximal VO_2 varies with lean body mass, age, gender, and genetic characteristics ***(Klissouras, 1971)***. In children, there is no gender differences in maximal VO_2 until puberty, when values in girls become 20% to 30% less than those in boys. This is partly due to the fact that girls on the average weigh less and have a lower lean body mass than boys but also may be related to relative decrease in hemoglobin content and less activity in girls. Maximal VO 2 also decrease with age.

Cardiac output is linearly related to VO_2. Cardiac output during exercise is largely determined by increases in heart rate ***(Stone et al., 1981)***. Stroke volume does contribute to the increase in cardiac output, especially early during dynamic exercise due to relative increase in venous return caused by the pumping action of the exercising muscles.

Maximal dynamic exercise results in a fourfold increase in cardiac output, a threefold increase in heart rate and a twofold increase in stroke volume. A VO_2 increases threefold due to both a redistribution of cardiac output to the active muscle tissue and an increase in oxygen extraction across the exercising muscle capillary beds. Resting heart rate is determined largely by vagal tone.

Heart rate is higher in the upright as compared with the supine position because the reduction in venous return in the upright position results in sympathetic reflex activation. Standing results in lower stroke volume and hence aortic pressure. Baroreceptor reflexes are activated which increase sympathetic tone mildly . Once exercise is initiated, there

is an initial rapid increase in heart rate due to the withdrawal of vagal tone mediated by changes in central nervous system mechanisms. These centrally mediated changes are due to cerebral mechanisms and reflex changes caused by activation of mechanical receptors in the skeletal muscles that are being activated. As exercise progresses subsequent increases in heart rate are mediated by increases in sympathetic nervous system activity and increases in circulating catecholamines. In cardiac transplant patients these neurally mediated mechanisms are impaired and cardiac output does not increase until late in exercise when circulating levels of epinephrine are high ***(Savin et al., 1980)***.

The left ventricular stroke volume response depends on the exercise position. In the supine position at rest, stroke volume is 80% of maximally obtainable values. In the upright position, stroke volume is 65% of maximal obtainable values because of reduced venous return. Consequently, the magnitude of change during exercise varies between exercise in the upright and supine position ***(Poliner et al., 1980)***. With the initiation of exercise , venous return is increased due to the pumping action of skeletal muscles. Intrathoracic pressure is decreased so that left ventricular filling pressures are increased, the mitral valve orifice area enlarges and diastolic volume increases progressively early during exercise and then levels off during the later stages of exercise ***(Rassi et al., 1988 and Thadani et al., 1978)***.

Thus, the early increases in stroke volume are due to the activation of Frank Starling mechanism when end diastolic volume is increased or maintained despite increase in heart rate that shortens

diastolic filling time. As exercise progresses, this mechanism is insufficient to further augment the stroke volume in the face of a progressively shorter diastolic filling time, and sympathetic nervous stimulation and circulating catecholamines increase stroke volume further by means of increased contractility and reduced end systolic volume.

During muscular exercise, cardiac out is redistributed such that flow is reduced to the abdominal organs and increased to the exercising muscles and coronary circulation. Blood flow to the brain remains the same. Thus, proportionally more oxygen is being delivered to the metabolically active organs. This is especially critical for the coronary circulation because the heart extracts 70% to 80% of available oxygen at rest. Hence, increase in oxygen delivery to the heart muscle must be accompanied by increase in coronary blood flow. During exercise, coronary blood flow increases four times, mainly due to vasodilatation and reduction in coronary vascular resistance because the diastolic driving pressure to coronary blood flow is actually reduced during exercise. There is also evidence that extraction of oxygen is increased in the metabolically more active organs. The net result is that the arterio-venous oxygen difference increases during exercise.

Systemic systolic blood pressure increases during exercise and diastolic pressure decreases mainly but the mean pressure is little changed despite the widening pulse pressure. Changes in blood pressure are influenced by the magnitude of the muscle mass being exercised. The influence of vasodilatation in a big muscle mass during

exercise is different than that of a small muscle mass during exercise on a total vascular conductance. Thus, the same increase in cardiac out put is achieved with a higher blood pressure during arm exercise as compared with leg exercise because leg exercise would tend to decrease total systemic vascular conductance and reduce blood pressure.

Pulmonary artery pressure increases more than systemic pressure because there is less of a decrease in pulmonary vascular resistance during exercise as compared with systemic vascular resistance, which decreases markedly, especially during large muscle mass exercise. The increases in pulmonary pressure and tidal lung volume augment pulmonary oxygen transport during exercise ***(Ehsani, et al 1991)***. There is no evidence that diffusing capacity across the lung alveoli and capillaries changes during exercise.

Acute Response to Static Exercise:

Although the general physiologic response to static exercise is similar to dynamic exercise, there are unique differences. Intramuscular pressure increase markedly during the contractile effort at the same muscle length. This results in a marked decrease or even cessation of muscle blood flow. Hand grip exercise at greater than 70% of maximum voluntary contraction completely ceases forward blood flow. Leg blood flow is even more sensitive to contractile effort and contraction greater than 20% of maximum voluntary contraction in the leg at a fixed muscle length completely ceases muscle blood flow ***(Shepherd et al., 1981)***. Consequently, prolonged isometric exercise depends on anaerobic

mechanism for providing the energy for muscle contraction and the increase in VO_2 and cardiac output is modest.

Once isometric exercise is stopped, VO_2 and cardiac output transiently increases before decreasing. The explanation for this phenomena is that during isometric contraction, muscle ischaemia provokes local mechanism that increase vasodilatation of the muscle bed. When flow is returned, local oxygen demand and blood flow increase causing the transient rise in cardiac output and VO_2. This post-ischaemic hyperemic response presumably pays back the oxygen debt that was incurred during isometric exercise.

Increase in cardiac output during isometric exercise is largely a result of increases in heart rate because stroke volume does not increase appreciably until the immediate recovery period. Nevertheless, stroke volume is maintained despite the increase in heart rate, because the initial contractile effort probably squeezes blood out of the exercising muscles and at least transiently, increases venous return. Also, activation of the sympathetic nervous system increases the contractility of the heart during isometric exercise.

Arterio-venous oxygen difference is unchanged during isometric exercise because of the contracted muscles block access of blood. After the release of isometric tension, AVO_2 difference increases due to increased flow to the muscles and increases in muscles extraction in the post exercise bed. Systemic arterial pressure increases during isometric exercise. Stroke volume and total systemic vascular resistance

are not increased enough to appreciably affect the blood pressure. Thus, changes in reflected waves due to muscle bed constriction probably increase the measured blood pressure *(Mitchel et al., 1981)*.