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RESEARCH ARTICLE

PARASYMPATHETIC INFLUENCE ON THE MYOCARDIUM TO THE ONSET OF EXERCISE AND IMMEDIATE POST EXERCISE RECOVERY

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Abstract

Background: The purpose of this study was to investigate the influence of parasympathetic nervous system on heart rate after the onset of exercise and immediate post exercise recovery. The heart rate is one of the most important cardiovascular parameter, which is very commonly used in sports to measure athletic performance. The parasympathetic nervous system is one of the major factors controlling the heart rate response due to exercise or any other stressful activity. It is also found that parasympathetic nervous system has a trainable characteristic. Hence the study focused on relating the heart rate response to the parasympathetic nervous system during the onset of exercise and immediate post exercise recovery. Methodology: This study followed an experimental approach to the problem through the analysis of archived data in the form of heart rate response to incremental workloads to volitional fatigue. The subjects in the archived data comprised of past research data measured during cycle ergometry testing to volitional fatigue. The demography of the subjects was determined via the archived data. The data consisted of 33 healthy male subjects between 19 and 47 years of age. The subjects were divided into two groups: very fit and moderately fit group, based on their VO_2 max. The 50^{th} percentile of the $MaxVO_2$ was determined by using the median for the group. The median value obtained was 57.2 ml/kg/min. The subjects with the $MaxVO_2$ above 57.2 ml/kg/min were grouped into a very fit category of the individuals, and the subjects with $MaxVO_2$ below 57.2 ml/kg/min were grouped into moderately fit category. There were sixteen (n=16) subjects in the very fit group and seventeen (n=17) subjects in the moderately fit group. The outcome of the test was measured in heart rate. The parasympathetic withdrawal includes the heart rate data collected during the first three minutes of exercise. The parasympathetic reinfusion includes the heart rate data collected during the last three minutes of exercise. The statistical analysis comprised of correlation analysis and the t-test for independent samples. The results were: the correlation coefficient for very fit group parasympathetic withdrawal versus parasympathetic reinfusion is -0.81 at $p < 0.05$ and was found to be significant. The correlation coefficient for moderately fit group parasympathetic withdrawal versus parasympathetic reinfusion is 0.65 at $p < 0.05$ and was found to be non significant. A t-test for independent samples was used to determine the

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difference between the heart rate response due to parasympathetic withdrawal and heart rate response due to parasympathetic reinfusion for the very fit group of subjects, and the difference was found to be statistically significant at p value of 0.002. A t-test for independent samples was used to determine the difference between the heart rate response due to parasympathetic withdrawal and the heart rate response due to parasympathetic reinfusion for the moderately fit group of subjects, and the difference was found to be statistically significant at p value of 0.001. The conclusion is that there is significant association between; heart rate response due parasympathetic withdrawal and heart rate response due to parasympathetic reinfusion for very fit group of subjects. But there is no significant association between heart rate response due to parasympathetic withdrawal and heart rate response due to parasympathetic reinfusion for moderately fit group of subjects. There exists a significant difference between the heart rate response due to parasympathetic withdrawal and heart rate response due to parasympathetic reinfusion in both very fit and moderately fit group of subjects.

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Introduction:-

Cardiovascular fitness has become an important component of training programs of various sports. Heart rate is one of the most important cardiovascular parameters used in sports measuring athletic performance. Heart rate response to exercise has always been a field of argument for many sports scientists and athletic trainers. The heart rate is variable, and the measures of heart rate variability are used to investigate the dynamics of the cardiovascular control system. The heart rate is directly proportional to intensity of exercise (Hoffman, 2002). There are many factors which cause heart rate variability. During certain activities, an increase in heart rate can be seen before the onset of exercise. This anticipatory rise in heart rate is primarily related to sprinting or anaerobic activity (McArle, Katch, & Katch, 1996). Just before the start of exercise, the heart rate usually increases above normal resting values. This is called a normal anticipatory response. This response is mediated through the release of neurotransmitter: nor epinephrine from the sympathetic nervous system and the hormone epinephrine from the adrenal medulla (Wilmore, Costill, & Kenney, 2008).Heart rate is affected by many extrinsic factors. The main extrinsic factors responsible are the Autonomic nervous system and the Endocrine system (Wilmore, Costill, & Kenney, 2008). The Parasympathetic nervous system is a branch of the autonomic nervous system, and reaches the heart through the vagus nerve. The vagus nerve has a depressant effect on the heart and it results in a decrease in heart rate (Wilmore, Costill, & Kenney, 2008). A study was performed to associate cardiac parasympathetic regulation with cardio respiratory fitness and training load. The study's objective was to establish the separate associations between parasympathetic modulations of the heart through heart rate variability indexes and post exercise heart rate recovery indexes with cardio respiratory fitness and training load (Buchheit & Gindre, 2006). The results indicated that vagal related heart rate variability indexes are more related to cardio respiratory fitness whereas heart rate recovery appears to be better associated with training load (Buchheit & Gindre, 2006). The Sympathetic nervous system is another branch of the autonomic nervous system. It has an opposite effect in comparison to the parasympathetic nervous system (Wilmore, Costill, & Kenney, 2008). After the onset of exercise the heart rate first increases due to withdrawal of vagal tone, with further increase if necessary due to sympathetic activation (Wilmore, Costill, & Kenney, 2008).

Statement of Purpose

The purpose of this study is to investigate the influence of parasympathetic system on heart rate after the onset of exercise and immediate post exercise recovery.

Hypothesis

H_1- There is a significant association between the heart rate response due to parasympathetic withdrawal at the onset of exercise to the heart rate response due to parasympathetic re-infusion at immediate post exercise.

H_{01}- There is not a significant association between the heart rate response due to parasympathetic withdrawal at the onset of exercise to the heart rate response due to parasympathetic re-infusion at immediate post exercise.

H_2- There is a significant difference between the heart rate response between fitness groups at parasympathetic withdrawal at the onset of exercise and the heart rate response at parasympathetic re-infusion during immediate post exercise.
H_{02}- There is a not a significant difference between the heart rate response between fitness groups at parasympathetic withdrawal at the onset of exercise and the heart rate response at parasympathetic re-infusion during immediate post exercise.

Limitations of the study
If the subjects did not follow the exercise protocol thoroughly at the time of testing, then the results might have been affected.

Delimitations of the study
Factors affecting the performance: diet, sleep and nutrition. Sympathetic neural factors affecting the heart rate response. The archived data only includes male subjects.

Assumptions
It will be assumed that all the participants did put forth their sincere efforts and followed the instructions when they were tested. There were no extraneous factors which can influence the results.

Independent variables
Exercise protocol on cycle ergometer is the independent variable.

Dependent variables
The heart rate recorded during the study is the dependent variable of this study.

Significance of this study
Myocardial response to the onset of exercise and the post exercise recovery has always been the area of interest for many exercise physiologists and athletic trainers. The purpose of this study is to establish the association between the heart rate response due to parasympathetic withdrawals at the onset of exercise and the heart rate response due to parasympathetic re-infusion at immediate post exercise. The heart rate response is the most important parameter used in measuring the magnitude of training level or performance of the individual. In this study heart rate is used as the parameter to measure the myocardial response to the exercise. Consequently, it is very important to find the possible reasons for the difference in the heart rate response in the athletic population and nonathletic population. The parasympathetic nervous system is one of the major factors controlling the heart rate response due to exercise or any activity. This study can be a small contribution to one of the aspects of heart rate response to exercise and will help in expanding our knowledge in this field of sports science. This will help future sports scientists and athletic trainers to better plan their training programs and improve the performance of athletes.

Literature Review:-

Background
Myocardial response to exercise has always been a major topic of interest for many sport scientists. Exercise physiologists have always tried their best to understand athletic performance and find possible ways to improve it. However, many questions still remain unanswered making research in this field necessary. This is applicable to myocardial response to exercise as well.

The purpose of this literature review is twofold. First, it will establish myocardial response to exercise in untrained and trained subjects. Second, it will find the statistical relationship of the data between trained and untrained subjects. This will help to establish current trends in cardiovascular training and the limiting factors affecting it. Therefore, future research can be directed at overcoming some of these limitations.

Myocardium
Myocardium is the cardiac muscle tissue. The heart is the primary pumping organ in the human body. Its function is to pump blood to all of the organ systems through the vascular system. The heart is made of contractile tissue. Cardiac muscle is collectively called myocardium or myocardial muscle (Wilmore, Costill, & Kenney, 2008). It is the main tissue constituting the wall of the heart. It is a voluntary muscle tissue which is striated in appearance like the skeletal muscle tissue. The individual fibers are multinucleated cells interconnected in latticework fashion

(McArle, Katch, & Katch, 1996). Consequently, when one cell is stimulated or depolarized, the action potential spreads through the myocardium to all the cells. This causes the heart to function as a single functioning unit (McArle, Katch, & Katch, 1996). The myocardial muscle tissue can generate an electrical signal on its own, which allows it to contract without any external stimulation. The impulse for a normal heart contraction is initiated in the Sino atrial (SA) node. The SA node is known as the heart's pacemaker. The thickness of the myocardial tissue varies at different locations depending upon the stress put upon it (Wilmore, Costill, & Kenney, 2008). The left ventricle is the most powerful of the four heart chambers. The function of this chamber is to pump blood through the entire body (Wilmore, Costill, & Kenney, 2008). A considerable amount of force is generated by the left ventricle in order to pump blood to the systemic circulation. The rate and strength of contraction is modified by the autonomic nervous system (Wilmore, Costill, & Kenney, 2008).

Myocardial functions

Cardiac output

Cardiac output (Q) is the total volume of blood pumped by the heart in one minute. It is the product of stroke volume and heart rate. Cardiac output depends upon the workload put upon the body, and it varies from person to person. The cardiac output is higher in a trained individual as compared to a sedentary individual (Hoffman, 2002).

Cardiac Output (Q) = Heart rate × Stroke volume

At rest, cardiac output is approximately 5 liters. During the maximal exercise, the cardiac output may increase up to 20 liters in young sedentary males. It may increase up to 40 liters in young endurance male athletes (Hoffman, 2002).

Stroke volume

Stroke volume is the amount of blood ejected from ventricles during systole. It is the volume of blood pumped per beat or contraction (Wilmore, Costill, & Kenney, 2008). The volume of the blood in the ventricles at the end of the diastolic phase, when the ventricular filling is completed, is called end diastolic volume (EDV). The volume of the blood left in the ventricles at the end of the systolic contraction is the end systolic volume (ESV). The stroke volume is the difference between end diastolic volume and end systolic volume (Wilmore, Costill, & Kenney, 2008).

Stroke Volume = End Diastolic Volume – End Systolic Volume

Stroke volume is affected by many factors like blood volume, body position, venous return, venous tone, cardiac muscle pump and heart chamber volume (Wilmore, Costill, & Kenney, 2008).

Blood Volume

Blood Volume in the body varies considerably with individual's size and state of training (Wilmore, Costill, & Kenney, 2008). Larger blood volumes are associated with greater lean body mass and higher level of endurance training. The blood volume of people with average body size and normal physical activity generally ranges from 5 to 6 liters in men and 4 to 5 liters in women. Blood is composed of plasma and formed elements like red blood cells, white blood cells and platelets. Endurance training increases blood volume, and this effect is larger as training intensity increases (Wilmore, Costill, & Kenney, 2008). This increase in blood volume results from an increase in plasma volume and an increase in red blood cell count. Although the actual number of red blood cells may increase, the ratio of the red blood cell volume to total blood volume may actually decrease. A trained athlete's hematocrit can decrease to a level where the athlete appears to be anemic on the basis of a low concentration of red blood cells and hemoglobin. This can reduce the viscosity of blood. Reduced viscosity can enhance the oxygen delivery to the active muscle mass. The turnover rate of red blood cells also may increase with intense training (Wilmore, Costill, & Kenney, 2008; Silverthorn, 2007).

Body position

Body position can result in changes in stroke volume in many ways. A study was performed on regulation of stroke volume during sub maximal and maximal upright bicycle exercise in normal male individuals. Twenty four asymptomatic male volunteers were evaluated during maximal upright bicycle exercise. Increase in heart rate from 73 to 167 beats per min, and stroke volume from 41 to 58 ml was recorded after the test. This increase in left ventricular stroke volume during upright exercise in the men was dependent upon the changing relationship among heart rate, left ventricular filling, and left ventricular contractility (Higginbotham, Morris, Coleman , & Cobb, 1984;

Higginbotham, Morris, Williams, McHale, Coleman, & Cobb, 1986). At low levels of exertion, an elevated left ventricular filling pressure and end diastolic volume are important determinants of the stroke volume response through the Frank Starling's mechanism (Wilmore, Costill, & Kenney, 2008). At high levels of exertion, the heart rate is elevated and is accompanied by a decrease in end diastolic volume, but there is a progressive increase in left ventricular filling pressure. The stroke volume must be maintained by a decrease in end systolic volume (Higginbotham, Morris, Coleman , & Cobb, 1984; Higginbotham, Morris, Williams, McHale, Coleman, & Cobb, 1986). The circulatory responses to mild and maximal upright exercise were studied in 6 normal subjects and 21 cardiac patients (Epstein, Beiser, Stampfer, Brain, & Braunwald, 1967). It was found that the usual hemodynamic indices employed to evaluate cardiac performance during exertion were unreliable in separating patients from normal individuals (Epstein, Beiser, Stampfer, Brain, & Braunwald, 1967). In contrast, the cardiac index achieved at a pulmonary arterial oxygen saturation of 30% was found to be highly reliable and sensitive for distinguishing the normal from the abnormal response to exercise (Epstein, Beiser, Stampfer, Brain, & Braunwald, 1967). There is an age associated decline in heart rate, ejection fraction, and cardiac output responses to supine exercise in healthy men (Stratton, Levy, Cerqueira, & Schwart, 1994). Increase in peak heart rate was observed in the patients who were on prolonged bed rest during upright exercise (Convertino , Hung , Goldwater, & DeBusk, 1982).

Venous return

Venous return is also termed as preload. Venous return describes the amount of blood returning to the heart through the veins (Guyton & Hall, 2006). Increase in venous return or preload elevates the stroke volume. When the ventricle stretches more during filling, it subsequently contracts more forcefully. When the larger volume of blood enters and fills the ventricles during diastole, the ventricular wall stretches (Wilmore, Costill, & Kenney, 2008). To eject this greater volume of blood, the ventricle responds by contracting more forcefully. This is referred to as Frank Starling's mechanism (Wilmore, Costill, & Kenney, 2008). A study on the role of Frank Starling's mechanism during exercise concluded that, in spite of their high heart rates, the stroke volume increased during the exercise. The augmentation in stroke volume was due to the combined effect of sympathetic nervous system activity and Frank Starling's mechanism (Horwitz, Atkins, & Leshin, 1972).

Venous tone

Venous tone refers to the capacitance in the venous vessels (Klabunde, 2012). Veins are a part of our circulatory system. Their function is to carry the deoxygenated blood from the body tissues back to the heart, except for the pulmonary veins and the umbilical veins, which carry oxygenated blood to the heart. The veins are less elastic in nature as compared to arteries (Guyton & Hall, 2006). Veins have valves to prevent back flow of blood and to maintain the blood flowing to the heart. The main function of the veins is to carry deoxygenated blood from the body tissues to the heart (Klabunde, 2012). Like other blood vessels, veins are under the influence of the sympathetic nervous system that will lead to vasoconstriction. Circulating levels of epinephrine and angiotensin II will have the same effect. The heart itself provides some suction when right atrial pressure fluctuates below zero at specific points in the cardiac cycle (Hoffman, 2002). A respiratory pump also promotes venous return, as the cycle of inspiration and expiration results in periods of negative pressure within the thoracic cavity. This results in a moderate increase in venous blood flow during inspiration (Guyton & Hall, 2006; Hoffman, 2002; Wilmore, Costill, & Kenney, 2008)

Heart chamber volume

Heart chamber volume refers to the morphological changes that take place in the heart due to prolonged exercise training (Hoffman, 2002). An increase in left ventricular mass has been observed after short duration endurance and resistance training (Ricci et al., 1982). During exercise, the hemodynamic demands on the heart are predominantly pressure and volume. During prolonged training, the heart will adapt to match the work load that is placed on the left ventricle in order to maintain a constant relationship between the systolic cavity pressure and the ratio of wall thickness to the ventricular radius (Ricci et al., 1982; Shapiro, 1997). Adaptations to the morphology of the heart are governed by the law of La Place, which states that wall tension is proportional to pressure and the radius of curvature (Ford, 1976). During a pressure overload, which are common in resistance exercise programs, the septum and posterior wall of the left ventricle increase in size to normalize the stress put on the myocardial wall. During a volume overload, which is common in endurance training programs, the increase is predominantly in the internal diameter of the left ventricle, with a proportional increase in both the septum and the posterior wall of the ventricle (Ford, 1976). Endurance training and resistance training are at either ends of the spectrum concerning the volume and pressure stresses placed on the heart. However, most of the sport activities have a parallel impact on both cavity dimension and wall thickness (Spirito et al., 1994). In these sports, athletes perform some combination of aerobic

and anaerobic training, resulting in cardiovascular adaptations associated with both enlarged diastolic cavity dimension and a larger wall thickness. In a sport which is limited to a single form of training, the morphological changes in the heart may be more extreme. The left ventricular mass is, on average, forty five percent greater in highly trained athletes than in age-matched control subjects (Hoffman, 2002). This increase in mass is related to the increase in left ventricular internal diameter and the thickness of the ventricular wall. When examined relative to changes in body mass or body surface area, the significantly greater ventricular mass is still present. Some studies also suggest that differences are more prevalent in elite athletes than in athletes of lesser caliber (Fleck, 1988; Hoffman, 2002).

Heart rate

Heart rate (HR) is one of the simplest yet most informative of the cardiovascular parameters (Wilmore, Costill, & Kenney, 2008). Measuring heart rate involves taking the subject's pulse, usually at the radial or carotid artery (Guyton & Hall, 2006). Heart rate is a good indicator of the intensity of the exercise (Wilmore, Costill, & Kenney, 2008). It is one of the most important cardiovascular parameters used in the sports science. It is the measurement of the number of heart beats per unit time, and is expressed as beats per minute (Smith & Fernhall, 2011). The heart rate of an individual keeps varying depending upon the body's demands (Hughson & Tschakovsky, 1999). Measuring heart rate helps the medical professional to detect any underlying pathology in the cardiovascular system. It is also used by the athletic trainers and sports scientists to measure the performance or training level of the athletes. Heart rate is measured by finding the pulse of the body. This pulse rate can be measured at any point on the body where the artery's pulsation is obvious and can be measured with the index and middle fingers (Spirito et al., 1994). Electronic devices are currently being used to take an accurate measurement of the heart rate. Auscultation at the apex of the heart in the chest region gives a more accurate measurement of the heart rate. Commercial heart rate monitors with chest straps are also available these days and are more commonly preferred in the field of athletic training. A resting Heart rate below 60 beats per minute is considered bradycardia and a resting Heart rate above 100 beats per minute is tachycardia (Wilmore, Costill, & Kenney, 2008). Another variable of Heart rate is maximal heart rate or Heart rate max (Tanaka, Monahan, & Seals, 2001). Heart rate max is the highest value of heart rate achieved during any stressful activity until the point of complete exhaustion (Tanaka, Monahan, & Seals, 2001). This is a more reliable value because it remains constant from day to day. However, this value may change slightly each year due to normal age related decline in heart rate max, which shows a slight but steady decrease of about one beat per year beginning at 10 to 15 years of age. Average maximal heart rate (HRmax) is obtained by subtracting one's age from 220. However, this is just an estimate, and individual value may vary from this average value (Tanaka, Monahan, & Seals, 2001). Heart rate is affected by many extrinsic factors like the Autonomic nervous system and the endocrine system (Wilmore, Costill, & Kenney, 2008).

Autonomic nervous system

The Autonomic nervous system is the part of the nervous system which controls most of the visceral functions of the body (Guyton & Hall, 2006). This system helps to control arterial pressure, gastrointestinal motility, gastrointestinal secretion, urinary bladder emptying, sweating, body temperature and many other activities, some of which are controlled almost entirely, and some only partially, by the autonomic nervous system (Guyton & Hall, 2006).The Autonomic nervous system is broadly divided into the sympathetic nervous system and the parasympathetic nervous system.

Sympathetic nervous system

The Sympathetic nervous system has an excitatory effect on the heart rate. Sympathetic stimulation causes a release of epinephrine and norepinephrine from the sympathetic neural fibers and elevates the heart rate by increasing Sino atrial node activity (Smith & Fernhall, 2011). Sympathetic stimulation increases the rate of impulse generation and speed of conduction and thereby elevates the heart rate. Maximal sympathetic stimulation can increase the heart rate up to 250 beats per minute (Wilmore, Costill, & Kenney, 2008). Heart rate elevation during exercise is primarily controlled by the sympathetic stimulation (Hoffman, 2002). During certain activities, an increase in heart rate can be seen before the onset of the exercise. This is an anticipatory response, and it is seen in sprint and other anaerobic types of events (McArle, Katch, & Katch, 1996).

Parasympathetic nervous system

The Parasympathetic nervous system is another branch of the Autonomic nervous system, which originates centrally in the region of the brain stem called the medulla oblongata and reaches the heart through the vagus nerve (Guyton & Hall, 2006; Silverthorn, 2007). The vagus nerve carries the impulses to the Sino atrial and Atrio ventricular, and,

when stimulated, causes a release of acetylcholine, which causes hyperpolarization of the conduction cells. This has a withdrawal effect and results in a decrease in heart rate (Klabunde, 2012). At rest, activity of the parasympathetic system predominates, and the heart is said to have vagal tone. In absence of vagal tone, resting heart rate would be approximately 100 beats per minute (Wilmore, Costill, & Kenney, 2008). Maximal vagal stimulation can decrease the heart rate to as low as 20 to 30 beats per minute. The vagal stimulation also decreases the force of cardiac muscle contraction (Wilmore, Costill, & Kenney, 2008). A study on parasympathetic effects on cardiac electrophysiology during exercise illustrated that moderate intensity exercise is associated with a shortening of the sinus cycle length, Atrio Ventricular block cycle length, Atrio Ventricular interval and ventricular effective refractory period (Kannankeril & Goldberger, 2002). Although there is a parasympathetic withdrawal at the onset of exercise, there are still measurable parasympathetic effects at the Sino atrial node, Atrio Ventricular node and ventricles during the moderate exercise and recovery (Kannankeril & Goldberger, 2002). However, the parasympathetic influence is less during the exercise as compared to when the individual is at rest (Kannankeril & Goldberger, 2002). A test was performed to associate cardiac parasympathetic regulation with cardio respiratory fitness and training load (Buchheit & Gindre, 2006; Buchheit , Haddad, Millet, Lepretre, Newton, & Ahmadi, 2009). The objective was to establish the separate associations between parasympathetic modulations of the heart through heart rate variability indexes and post exercise heart rate recovery indexes with cardio respiratory fitness and training load (Buchheit & Gindre, 2006; Buchheit , Haddad, Millet, Lepretre, Newton, & Ahmadi, 2009). The results indicated that vagal related heart rate variability indexes are more related to cardio respiratory fitness, whereas heart rate recovery appears to be better associated with training load (Buchheit & Gindre, 2006; Buchheit , Haddad, Millet, Lepretre, Newton, & Ahmadi, 2009).

Endocrine system

The Endocrine system is one of the other factors which influence the heart rate. It exerts its effect through norepinephrine and epinephrine, which are released by the adrenal medulla and stimulate the heart, increasing its rate and contractility (Guyton & Hall, 2006). In fact, the release of these hormones from the adrenal medulla is triggered by sympathetic stimulation during times of stress, and their action prolongs the sympathetic response (Hoffman, 2002).Stimulation of the sympathetic nerves to the adrenal medulla causes large quantities of epinephrine and norepinephrine to be released into the circulating blood, and these two hormones in turn are carried in the blood to all the tissues of the body (Hoffman, 2002). On average, about 80 % of the secretion is epinephrine and 20 % is norepinephrine, although the relative proportions can change considerably under different physiologic conditions. A study on feedback effects of circulating norepinephrine on the sympathetic outflow in healthy subjects indicated that elevated levels of circulating norepinephrine causes a feedback inhibition on sympathetic outflow in healthy subjects (Tulppo et al., 2005). According to the study, this inhibitory mechanism is caused by the pre synaptic auto regulatory feedback mechanism or can be due to some other mechanism that is not prevented by a nonselective α-adrenergic blockade (Tulppo et al., 2005).

Heart rate

Heart rate will affect the power generated by cardiac muscle tissue. Cardiac muscle power describes the force generated by the cardiac muscle (Smith & Fernhall, 2011). Many theories have been presented to understand the cardiac muscle's power. Force produced by the muscle is directly proportional to the muscular strength (Khurana, 2008). The amount of blood pumped out of the ventricles during systole depends upon the amount of force generated by cardiac muscle tissue during contraction (Wilmore, Costill, & Kenney, 2008). The amount of contraction is governed by Frank-Starling's mechanism. The Frank-Starling's mechanism describes the intrinsic ability of the heart to adapt to increasing volumes of in flowing blood during ventricular filling (Hoffman, 2002). The increasing volume load of the blood on the ventricular tissue leads to stretch. The greater the heart muscle is stretched during filing, the greater is the force of contraction, and the greater the quantity of blood pumped.

Length-tension relationship

Length-tension relationship is the relation between initial fiber length and total tension in the cardiac muscle, which is basically similar to that in skeletal muscle (Khurana, 2008). The length tension relationship explains how the changes in preload or the end diastolic volume effect the development of isometric force in the cardiac muscle tissue (Khurana, 2008).

Force-velocity relationship

The force-velocity relationship is very important to understanding the functioning of the cardiac muscle (Khurana, 2008). The cardiac muscle can alter its work and power (rate of working) at any given load and muscle length by

nature of its changing force-velocity relationship in different conditions. When the pressure against which the heart is pumping the blood is elevated, the heart strokes out less blood than it receive through the venous return. Consequently, more blood accumulates in the ventricles, which elevates the end diastolic volume and increases the initial length of the muscle fibers (i.e. size of the heart) (Khurana, 2008). The distended heart beats more forcefully, and output returns to its previous level by an increase in the initial length of the cardiac muscle (Khurana, 2008). Conversely, when the pressure against which the heart is pumping the blood is reduced, the stroke output rises transiently, but the size of the heart decreases, and the stroke output falls to the previously constant level (Khurana, 2008)

Myocardial response to the onset of exercise

Myocardial response to the onset of exercise depends upon several factors: the type of exercise (Aerobic/Endurance training or Anaerobic/Resistance training), intensity of exercise (Sub maximal intensity or Maximal intensity), duration of exercise(short or long), mode of exercise(running or cycling for Aerobic/Endurance training, and sprinting or weight training for Anaerobic/Resistance training), and the training status of the subject(trained individual or untrained individual).

Cardiac output

Cardiac output at rest is approximately 5 liters (Hoffman, 2002). However, during the maximal exercise, cardiac output may increase up to 20 liters in young sedentary males and may increase up to 40 liters in young endurance male athletes (Hoffman, 2002). The importance of a large cardiac output for endurance athletes is reflected by the linear relationship seen between cardiac output and oxygen consumption (Lewis, Taylor, Graham, Pettinger, Schutte, & Blomqvist, 1983). This relation has also been observed in children and adolescents (Cunningham, Paterson, Blimkie, & Donner, 1984). A similar relationship has also been observed between trained and untrained subjects (Hermansen & Saltin, 1969).

Stroke volume and response to endurance training

Increase in stroke volume is observed during the acute phase of exercise (Hoffman, 2002; Alonso, Rodríguez , & Coyle , 2000). This is accomplished through an increase in end diastolic volume or preload. This rapid elevation in stroke volume is due to Frank Starling's Mechanism, which is related to the increased volume of blood that returns to the heart through venous return during the onset of exercise (Hoffman, 2002). With a greater volume of blood returning to the heart during exercise, the ventricles become stretched to a greater extent than normal and respond with more forceful contraction. This stronger contraction results in a greater volume of blood entering the systemic circulation with each heartbeat. This mechanism appears to occur during the acute phase of exercise and at a relatively low level of exercise intensity. The Frank Starling's mechanism may cause an approximate 30-50% increase in stroke volume (Bonow, 1994). As the exercise continues, increase in end diastolic volume reaches a plateau while exercise intensity is still sub maximal (Hoffman, 2002). A further increase in stroke volume is due to left ventricular contractile function, resulting in a greater decrease in end systolic volume. There are two mechanisms that appear to be responsible for the increase in end diastolic volume during the exercise. The first mechanism is the use of exercising muscles as pumps to increase the rate of return of the blood to heart. This would lead to an increase in left ventricular pressure and thereby increase the diastolic pressure. However, this is not the case in a healthy heart where the relaxation is seen in the left ventricle, which reduces the ventricular pressure below the left atrium (Hoffman, 2002). This causes the mitral valve to open and begins the ventricular filling. As mentioned earlier, the elevated sympathetic response during the exercise increases the relation period during the diastole (Hoffman, 2002). During this time, the increase in size of the left ventricle causes further reduction in pressure, creating a suctioning effect that draws additional blood into the chamber. This process of facilitation by sympathetic drive is the secondary mechanism that contributes to the increased stroke volume and plays an important role in Frank Starling's mechanism (Bonow, 1994). In a study, it was hypothesized that 10 days of training on a cycle ergometer would enhance cardiac output (Q) and stroke volume during peak exercise and increases the inotropic response to β-adrenergic stimulation (Mier, Turner , Ehsani , & Spina , 1997; Constance , Michael , & Ali , 1997). Ten subjects aging between 24 to 28 years were trained on a cycle ergometer for ten days. At peak exercises, training increased oxygen uptake, cardiac output and stroke volume. Left ventricular size and function at rest were assessed with two-dimensional echocardiography before and after the atropine (1 milligram) injection and during the four graded doses of dobutamine. Left Ventricular contractile performance was assessed by relating fractional shortening to the estimated end systolic wall stress. Training increased the slope of the force-stress relationship indicating the enhanced systolic function. The increase in slope correlated with increase in cardiac output and stroke volume. The increase in blood volume also correlated with increase in cardiac output and stroke

volume (Mier, Turner , Ehsani , & Spina , 1997). This data showed that 10 days of training enhanced the inotropic response to β-adrenergic stimulation associated with increase in cardiac output and stroke volume during peak exercise (Mier, Turner , Ehsani , & Spina , 1997; Constance , Michael , & Ali , 1997). Endurance training has consistently been found to be potent stimuli for increasing stroke volume at rest and during maximal exercise (Hoffman, 2002). Athletes going through long term endurance training have been observed with 60% greater stroke volume as compared to a sedentary individual (McArle, Katch, & Katch, 1996). An enlarged ventricular chamber or eccentric hypertrophy can also be a causative factor for improved stroke volume. The possible mechanism behind this is the increased ventricular filling which occurs after long term endurance training. This increased preload is thought to be related to expanded blood volume after long term endurance training (Carrol, Convertino, Graves, Lowenthal, & Pollack, 1995; Convertino, Rickards, & Ryan, 2012). A comparison between cardiorespiratory response to an underwater treadmill and a land treadmill was used to derive an equation to estimate VO_2 during underwater treadmill exercise (Greene, Greene, Carbuhn, Green, & Crouse, 2011). Fifty five men and women completed one land treadmill and five underwater treadmill exercise sessions on separate days. The underwater treadmill session consisted of chest deep immersion with 0, 25, 50, 75 and 100% water-jet resistance (Greene, Greene, Carbuhn, Green, & Crouse, 2011). In each session, the treadmill velocities increased every three minutes from 53.6 to 187.8 meters per min. The cardiorespiratory responses were similar between land treadmill exercise and underwater treadmill exercise when jet resistance for underwater treadmill training was more than 50%. This data indicates that similar land treadmill and underwater treadmill data are achievable (Greene, Greene , Carbuhn , Green, & Crouse, 2011). A study on cardiovascular response to repeated treadmill exercise soon after Myocardial Infarction involved 24 male subjects with ages between 48 years and 62 years (Haskell & DeBusk, 1979). They were made to perform two symptom-limited tests several days apart from 3, 7 and 11 weeks after the acute event. Significant differences in these variables were not noted in systolic blood pressure or heart rate and systolic blood pressure product for any of the three test periods (Haskell & DeBusk, 1979). No significant differences were noted for any variable recorded at a sub maximal work load of 4 metabolic equivalents (Haskell & DeBusk, 1979). The frequency of exercise induced ischemic ST segment depression; angina pectoris and premature ventricular complexes did not change from visit to visit and was highly reproducible in the 3 months after uncomplicated myocardial infarction. Changes in the response to treadmill exercise tests performed several weeks apart reflect an alteration in the cardiovascular response (Haskell & DeBusk, 1979).

Stroke volume and response to resistance training

Changes in stroke volume due to resistance training are debatable. Stroke volume is influenced by preload, after load, cardiac contractility and heart rate (Hoffman, 2002). Sometimes the extrinsic factors like the catecholamine release may affect the inotropic state of the heart. However, increase in after load has been observed during resistance training which produces only minimal variations in stroke volume. In general, no changes in stroke volume have been observed during low intensity resistance training but may decrease during high intensity resistance training (Sullivan, Knowlton, DeVita, & Brown, 1996). High intensity muscle contraction leads to reduction in preload and increase in after load which eventually reduces the stroke volume (Stone, Wilson, Blessing, & Rozenek, 1983). The intra thoracic pressure is accentuated due to increased abdominal pressure by the Valsalva maneuver, which further compromises the stroke volume. However, a significant increase in stroke volume has been observed in elite level weight lifters compared to recreational weight lifters (Stone, Wilson, Blessing, & Rozenek, 1983). A comparison of left ventricular diastolic function was done between a group of 16 elite male weight lifters and a group of 10 recreational weight lifters of the same age (Pearson, Schiff, Mrosek, Labovitz, & Williams, 1986). The Pulsed Doppler examination was used to measure left ventricular inflow. They found that the elite weight lifters had a greater stroke volume than recreational weight lifters (Pearson, Schiff, Mrosek, Labovitz, & Williams, 1986). Another study was done on cardiovascular response to isometric exercises (Alexander, 1970; Laird, Fixler, & Huffines, 1979). According to the study, stroke volume remains constant at lower intensity isometric exercises. However, a decrease in stroke volume was observed at a higher intensity of isometric exercises (Alexander, 1970; Laird, Fixler, & Huffines, 1979). This suggests that the increase in cardiac output is due to elevated heart rate (Alexander, 1970). Lower limb strength training improves the left ventricular performance by increasing the stroke volume (Kanakis & Hickson, 1980).

Heart rate and response to exercise

Heart rate elevation is seen during acute phases of exercise (Hoffman, 2002). This is primarily controlled by sympathetic stimulation from the higher centers of the brain. Heart rate response is directly proportional and linear to the intensity of exercise (Hoffman, 2002). Heart rate will continuously keep increasing as the intensity of exercise increases until it reaches the maximum level. At maximal intensity, the heart rate will plateau, indicating that the

individual is reaching his/her maximal extent. The initial increase in heart rate on the onset of exercise is attributed to parasympathetic withdrawal (Hoffman, 2002; McArle, Katch, & Katch, 1996). This occurs during low intensity exercise. As the exercise intensity or duration increases, the sympathetic stimulation becomes more effective and the heart rate is elevated. Sympathetic activation occurs through feedback mechanisms from peripheral mechanical and chemical receptors (Hoffman, 2002). During certain activities like sprinting or weight lifting, an anticipatory increase in heart rate can be seen before the onset of exercise (Hoffman, 2002). An increase in heart rate results in a greater volume of blood being pumped into the circulatory system. However, there is no limit to this effect. As the heart rate rises above a certain level, the strength of each contraction may decrease due to metabolic overload. The greater the rate of contraction, the less time spent in diastole. The time interval between subsequent contractions is reduced which leads to incomplete ventricular filling thereby decreasing the circulating blood flow (Hoffman, 2002; Sitkowski, Starczewska, & Burkhard, 2004).

Endurance training and heart rate response

Resting and sub maximal heart rates decrease during aerobic training. This response is very much apparent in individuals who were previously sedentary. This reduction in heart rate can be used to measure the magnitude of training improvement (Lamberts, Lemmnik, Durandt, & Lambert, 2004; McArle, Katch, & Katch, 1996). A decrease in resting heart rate and a relative heart rate at any particular stage of exercise at any given sub maximal VO_2 is a commonly found adaptation in long term endurance training (Blomqvist & Saltin , 1983; Charlton & Crawford, 1997). However, the magnitude in the heart rate reduction during endurance training may be much smaller than that reflected in some of the cross sectional studies comparing elite endurance athletes with sedentary controls (Wilmore, Costill, & Kenney, 2008). The effect of resistance training on heart rate is debatable. Several studies have reported a significant decrease in resting heart rate after long term resistance training programs (Goldberg, Elliot, & Kuehl, 1988). However, some of the studies failed to see any significant changes (Lusiani et al., 1986). The mechanism behind reduced heart rate can be attributed to change in autonomic response. In addition, a decrease in the intrinsic Sino Atrial node activity after long term training may lead to bradycardia (Schaefer, Allert, Adams, & Laughlin, 1992). Another study was done on heart rate variability after endurance training by Carter and colleagues. They found that after 12 weeks of endurance training, there was a significant decrease in resting heart rate as well as heart rate at submaximal intensity of exercise (Carter, Banister, & Blaber, 2003). This was applicable to the middle age group as well as the younger age group (Carter, Banister, & Blaber, 2003; Ogawa et al., 1992). There is a difference in heart rate response to exercise testing between females and males working at the same intensity (Gulati , Shaw, Thisted, Black , Bairey, & Arnsdorf, 2010; Ogawa et al., 1992). Impaired heart rate response to exercise manifests chronotropic incompetence which can predict the increased mortality rate and development of coronary heart disease (Lauer, Okin, Larson, Evans, & Levy, 1996). The patients with ischemic myocardial disease responded to the stress of exercise with cardiac dilatation when tested on the bicycle ergometer(Rerych, Scholz, Newman, Sabiston, & Jones, 1977). This is more acute in patients with left main coronary artery stenosis (Rerych, Scholz, Newman, Sabiston, & Jones, 1977). A spectral analysis of heart rate was performed to establish an index of relative sympathetic nervous system and parasympathetic nervous system activity during exercise(Yamamoto, Hughson, & Peterson, 1991).Eight subjects completed six repetitions of seventeen-minute sub maximal exercise tests and one resting measurement in the upright sitting position(Yamamoto, Hughson, & Peterson, 1991).The results suggested that parasympathetic nervous system activity decreased progressively from rest to a work rate equivalent to 60% ventilatory threshold, and sympathetic nervous system activity increased only when exercise intensity exceeded ventilator threshold(Yamamoto, Hughson, & Peterson, 1991).Heart rate makes little contribution to maximal oxygen uptake because maximal heart rate remains the same since it is not altered by endurance training. Resting heart rate, however, is decreased, and the importance of this is that it allows a larger range over which it can increase during exercise (Bunca, Hellera, & Lesoa, 1988). This, combined with a larger stroke volume, enables the same cardiac output to be achieved at a lower heart rate, thus enabling the very high values of cardiac output to be reached without compromising filling time by much higher heart rates. An added benefit is that oxygen delivery to the heart muscle is greater because the longer diastole increases cardiac perfusion for a given workload (Bunca, Hellera, & Lesoa, 1988). A lower resting heart rate may have other benefits, such as reducing adverse events due to coronary artery disease or heart failure (Buch , Coote, & Townend, 2002). The rate of perceived exertion when incorporated with heart rate in professional cycling races has shown positive results (Rodríguez-Marroyo, Villa, García-López , & Foster, 2012).

Resistance training and heart rate response

Anaerobic training consists of high intensity, intermittent bouts of exercise such as weight training, resistance exercises, sprinting, plyometric drills, speed, agility and interval training (Alcaraz, Sánchez, & Blazevich , 2008).

Anaerobic metabolism is primarily involved in anaerobic exercises. However, in certain anaerobic exercises aerobic metabolism is also highly involved. Anaerobic exercise also results in many physiological changes and adaptations in the body. These changes and adaptations depend upon the type of anaerobic exercise, intensity and the duration of exercise training (Baechle, Earle, & Ratamess, 2008; Olson, Williford, & Wang, 1997). Heart rate increases as the intensity of the anaerobic exercise increases (Mayo & Kravitz, 1999). Anticipatory increase in heart rate before the onset of exercise has also been observed during sprinting and heavy weight lifting exercises (McArle, Katch, & Katch, 1996). The heart rate response depends upon the type, duration and intensity of the anaerobic exercise. An acute bout of resistance training or sprinting can result in a significant increase in heart rate, which can be more prominent if the individual uses the Valsalva maneuver (Lepley & Hatzel, 2010). An acute bout of anaerobic exercise significantly increases the cardiovascular response, especially if the individual uses the Valsalva maneuver. Heart rate and stroke volume increases significantly during resistance exercises. Peak blood pressures of 250 to 320 millimeters of mercury and a heart rate of 170 beats per minute have been reported during a high intensity leg press exercise (Hill, Collins, Cureton, & DeMello, 1989). Generally, the blood pressure response increases nonlinearly with the magnitude of active muscle mass and is higher during the concentric phase of each repetition than during the eccentric phase. In addition, intra thoracic pressure increases and plasma volume reductions of up to 22% have been reported (Hill, Collins, Cureton, & DeMello, 1989). Acute anaerobic exercise results in increased heart rate and stroke volume. During a set of resistance exercises, stroke volume and cardiac output increases mostly during the eccentric phase of each repetition, especially when the Valsalva maneuver is used (Baechle, Earle, & Ratamess, 2008).The concentric phase of a repetition is much more difficult and elevations in intra-thoracic and intra-abdominal pressures are more prominent. This becomes more prominent when the Valsalva maneuver is used (Miller, Hall, Chmelo, Morrison, Dewitt , & Kostura , 2009). This limits the venous return which results in reduction of end diastolic volume. The hemodynamic response of resistance exercise is delayed such that cardiac output increases more during the eccentric phase or during the rest period between sets. This is also true for the individual heart rate response during the first five seconds after completion of a set. The heart rate is higher than it was during the set. The degree to which the blood flow is increased in the working muscles during the resistance training depends upon the amount of resistance, duration of effort or number of repetitions and sets performed, and the size of the muscle mass (Rozenek, Rosenau, Pat, & Stone, 1993). Lower resistance performed for many repetitions produces responses similar to aerobic exercises. However, heavy resistance exercises decrease blood flow to the working muscles. Muscular contractions greater than 20% of maximal voluntary contraction impede blood flow during a set, but the blood flow increases during the rest period after the set (Alcaraz, Sánchez, & Blazevich , 2008; Hill, Collins, Cureton, & DeMello, 1989). The concentric phase of exercise elevates the heart rate more than the eccentric phase of exercise (Miller, Hall, Chmelo, Morrison, Dewitt , & Kostura , 2009). A study was performed to establish relationship between heart rate and rate of percieved exertion during cardiovascular training (Glass & Holcomb, 1997). They found that using the rate of percieved exertion scale helps to take the individual to a higher intensity level (Glass & Holcomb, 1997). Higher heart rate was recorded during circuit resistance training as compared to treadmill training at the same amount of VO_2(Gotshalk , Berger , & Kraemer , 2004; Murray, Michael, & McClellan, 1995). The increase in heart rate is observed more during the concentric phase of the resistance exercise as compared to eccentric exercise due to increased intra-thoracic and intra-abdominal pressure (Lepley & Hatzel, 2010). Heart rate depends upon the amount of resistance used during the exercise. Lower resistance exercise with more repetition may have similar patterns like the aerobic exercise training (Fry & Parks, 2001). A study evaluated the acute cardiovascular responses to fatiguing resistance exercises with accommodating variables, and fixed resistances as well as with a graded exercise test (Kleiner, Blessing, Davis, & Mitchell, 1996). Heart rate, systolic blood pressure, diastolic blood pressure, and rate-pressure product were directly and continuously recorded during all exercises (Kleiner, Blessing, Davis, & Mitchell, 1996). Peak heart rates and blood pressures were highest and lowest, respectively, during the graded exercise test. For the resistance exercises, peak cardiovascular values were highest with accommodating resistance exercises, followed by fixed and then variable resistance exercises (Mayo & Kravitz, 1999; Pichon, Hunter, Morris, Bond, & Metz, 1996). There were significant differences between graded and resistance exercises for heart rate, systolic blood pressure, and diastolic blood pressure, but not for rate pressure product. There were also significant differences between accommodating versus fixed and variable resistance for systolic blood pressure and rate pressure product. This data shows that cardiovascular stress is increased during resistance exercises and those responses may differ between the various forms of resistance (Kleiner, Blessing, Davis, & Mitchell, 1996). Highly strength-trained athletes are observed with average values of resting systolic and diastolic blood pressure (Pearson, Schiff, Mrosek, Labovitz, & Williams, 1986; Queiroz, Gagliardi, Forjaz, & Rezk, 2009). A study was performed to check cardiovascular response to Punching Tempo. Eighteen trained volunteers: twelve men and six women, aging between 19 and 25 participated in two-minute, randomized fitness boxing trials. During each trial, heart rates were measured continuously (Kravitz, Greene,

Burkett, & Wongsathikun, 2003). The heart rate responses yielded results ranging from 167.4 to 182.2 beats per minute, or 85 to 93% of Heart Rate max. These results suggest that fitness boxing programs compare favorably with other exercise modalities in cardiovascular response and caloric expenditure (Kravitz, Greene, Burkett, & Wongsathikun, 2003).

Summary
An increase in stroke volume is observed at the acute onset of exercise due to increased end diastolic volume or preload. The stroke volume continues to increase as the exercise continues. Endurance athletes have been recorded with higher stroke volumes as compared to sedentary individuals.

Resistance training
No significant changes in stroke volume have been recorded at low intensity resistance training exercises. A decrease in stroke volume has been found during high intensity resistance training like power weight lifting. An increase in stroke volumes has been found with long term resistance training exercises.

Heart rate
Endurance training
An increase in heart rate has been found at the onset of exercise. Progressive increase in heart rate is seen as the exercise intensity increases and at certain intensity level, it plateaus. Decreased resting heart rate has been recorded in long term endurance training athletes.

Resistance training
Anticipatory increase in heart rate has been found before the onset of resistance exercises like power weight lifting and sprinting. Heart rate increases as the exercise begins. Changes in resting heart rate after long term resistance training are debatable.

Post exercise heart rate recovery
A study performed by Katsuji et al. (1994) to find the vagal mediated heart rate recovery after exercise in patients with chronic heart failure and in well trained athletes by analyzing the post exercise heart rate decay. They concluded that heart rate measured at 30 seconds could be specific index for vagal mediated heart rate recovery. The vagal mediated heart rate recovery after exercise is accelerated in well trained, but blunted in patients with chronic heart failure (Katsuj et al., 1994). Lamberts et al. (2009) performed a study on changes in heart rate recovery after high intensity training in well trained cyclists. They found that heart rate recovery was significantly improved in well trained cyclists after high intensity training sessions (Lamberts, Swart, Noake, & Lambert, 2009). They also concluded that heart rate recovery is a sensitive marker which tracks changes in training status in already well trained cyclists and has the potential to have an important role in monitoring and prescribing training (Lamberts, Swart, Noake, & Lambert, 2009). Kannankeril et al. (2004) performed a study on parasympathetic effect on heart rate recovery after exercise. They concluded that in normal subjects, parasympathetic effects persist during high-intensity exercise and are prominent in the early phases of recovery (Kannankeril, Francis, Kadish, & Goldburger, 2004). These parasympathetic effects may play an important role in prevention of sudden cardiac death during these periods of increased risk (Kannankeril, Francis, Kadish, & Goldburger, 2004). A study was performed to assess the contribution of the autonomic nervous system to heart rate recovery following exertion, heart rate was observed after peak treadmill exercise in six men following parasympathetic blockade with atropine sulfate, sympathetic blockade with propranolol hydrochloride, double blockade with both drugs, and no drugs(Savin, Savin, Davidson, & Haskell, 1982). At the cessation of exercise the decreases in venous return and the systemic need for cardiac output are accompanied by an exponential HR decline (Savin, Savin, Davidson, & Haskell, 1982). The exponential character of the cardio deceleration seen after peak exercise appears to be an intrinsic property of the circulation because it occurred under each experimental condition (Savin, Savin, Davidson, & Haskell, 1982). A study performed by Gordon and Eric (2004) on assessing autonomic function by analysis of heart rate recovery from exercise in healthy subjects. This study presents a unique method for quantitatively testing theories on the relative roles of sympathetic withdrawal and parasympathetic reactivation during recovery from exercise. It provides indexes of dynamic sympathetic and parasympathetic functions, with the parasympathetic system having a faster response time (Gordon & Eric, 2004). It supports theories of coordinated interaction of parasympathetic reactivation and sympathetic withdrawal during exercise recovery and does not support using simple measures of exercise HR recovery as indexes of vagal function alone (Gordon & Eric, 2004). Another study was conducted on modulation of cardiac autonomic activity during and immediately after exercise (Arai et al., 1989). They found that during recovery, heart

rate power increased in normal subjects but remained significantly below the base line (Arai et al., 1989). The findings demonstrate a marked reduction of autonomic modulation of heart rate in patients with heart failure and after cardiac transplant and support a progressive withdrawal of vagal activity during exercise with a gradual increase during recovery in normal subjects (Arai et al., 1989). A study was performed by Yamamoto et al. (2000) on effects of endurance training on resting and post-exercise cardiac autonomic control. They found that with endurance training, changes in cardiac ANS modulation partly contribute to a decrease in HR measured at rest and during post exercise recovery periods (Yamamoto, Miyachi, Saitoh, Yoshioka, & Onodera, 2000). The effects of adaptability of the cardiac autonomic control to endurance training occur sooner in immediate post exercise recovery periods than at rest (Yamamoto, Miyachi, Saitoh, Yoshioka, & Onodera, 2000).

Implications for future research

There is an abundance of literature available on myocardial response to exercise. However, there are many gaps in this literature which can be filled through further research. Many questions can be raised upon the previous literature based on this topic. The physiological basis of the heart rate variability is still not discovered to the extent it should be. More over the difference in the heart rate recovery patterns of a trained and a sedentary individual is still questionable. There are not many studies about parasympathetic withdrawal at the onset of exercise. The increase in heart rate during exercise is also significant. Is there any specific pattern of increase in the heart rate of trained individuals? Is there any difference in the heart rate pattern of a trained and untrained individual? What are the possible reasons for this change in pattern and recovery pattern? What role does parasympathetic nervous system play in heart rate response during the exercise? Can we modulate heart rate response in any individual through specific training program? This gap in the literature can be filled through efficient research methods, and new findings can be established which will promote future research as well as the field of cardiovascular training in sports and sedentary population.

Methodology:-

Purpose

The purpose of this study was to investigate the influence of parasympathetic system on heart rate after the onset of exercise and immediate post exercise recovery.

Experimental approach to the problem

This study followed an experimental approach to the problem through the analysis of archived data in the form of heart rate response to incremental workloads to volitional fatigue. It was a comparative study. The testing protocol was the active independent variable involved in the study. It has two levels: the heart rate response at the onset of exercise and the heart rate response at immediate post exercise. The demography of the subjects was determined via the archived data. It includes age (y), gender (m/f), body weight (kg), height (cm), and maximal oxygen consumption (MVO_2, ml $kg^{-1}min^{-1}$). The outcome of the test was measured in heart rate, which is the dependent variable of the study. Heart rate was measured in beats per minute. Subjects

The subjects comprised of past research data during cycle ergometry testing to volitional fatigue. The data was archived for further analysis. Ethical approval for the investigation was obtained from the Institutional Review Board for Humans as Subjects at Midwestern State University.

Procedures:-

Beat by beat heart rate recorded and averaged over 20 seconds was obtained for analysis.

Statistical Analysis

Descriptive analysis included mean and standard deviation (SD) of the subject's height, weight, age and Max VO_2. Heart rate data for the completed tests utilized a linear-logarithmic cross over design to determine the end point of parasympathetic withdrawal. Heart rate (HR) data from peak heart rate at volitional fatigue to the first minute of immediate post exercise was analyzed for parasympathetic re-infusion. The parasympathetic withdrawal included the heart rate data collected during the first three minutes of exercise. The parasympathetic reinfusion included the heart rate data collected during the last three minutes of exercise.

Slope for heart rate analysis was established through a linear regression analysis. A Pearson Product R correlation coefficient was run to determine associations between heart rate response due to parasympathetic withdrawal at the

onset of exercise vs. heart rate response due to parasympathetic re-infusion at immediate post exercise. Statistical significance was set at priori $p< 0.05$.

Results:-

The archived data included only male subjects. The mean and standard deviation of age, height, weight, and maximal oxygen consumption ($MaxVO_2$) is shown in table 1. Post hoc analysis determined two groups of four comparisons utilizing $MaxVO_2$ as the criterion measure to establish fitness level. The 50^{th} percentile of the $MaxVO_2$ was determined by using the median for the group. The median value obtained was 57.2 ml/kg/min. The subjects with the $MaxVO_2$ above 57.2 ml/kg/min were grouped into a very fit category of the individuals, and the subjects with $MaxVO_2$ below 57.2 ml/kg/min were grouped into moderately fit category. There were sixteen (n=16) subjects in the very fit group and seventeen (n=17) subjects in the moderately fit group. The difference between the two groups fitness status was found to be statistically significant by using a t-test, independent by groups with t-score of 7.7 and p value of 0.00. The mean $MaxVO_2$ for the very fit group was 66.53 and for the moderate fit group was 53.50, with a p-value of 0.001. The standard deviation for the very fit group was 6.50 and for the moderately fit group was 2.47. A 2-D graph (Figure 1) was plotted using mean with error plot to present the difference between two groups graphically. Each group had two comparisons: parasympathetic withdrawal and parasympathetic reinfusion. Parasympathetic withdrawal consisted of the first three minute heart rate measures of the subject. Parasympathetic reinfusion consisted of the last three minute heart rate measures of the subject. A graph (Figure 2) was plotted using a scatter plot, keeping time on the X-axis and the four variables: very fit parasympathetic withdrawal, very fit parasympathetic reinfusion, moderately fit parasympathetic withdrawal, and moderately fit parasympathetic reinfusion on the Y-axis. The slope for very fit parasympathetic withdrawal was 0.0982; the slope for moderately fit parasympathetic withdrawal was 0.079; the slope for very fit parasympathetic reinfusion was -0.217; the slope for moderately fit parasympathetic reinfusion was -0.135. Further, the t-test for independent samples was run between all four groups. The difference between heart rate response due to parasympathetic withdrawal in very fit group and heart rate response due to parasympathetic withdrawal in moderately fit group was found to be statistically significant with t-score of 2.5 and p-value of 0.02. The difference between heart rate response due to parasympathetic reinfusion in very fit group and heart rate response due to parasympathetic reinfusion in moderately fit group was found to be statistically significant with t-score of -2.35 and p-value of 0.03. A post hoc correlation analysis was run to determine if the heart rate response due to parasympathetic withdrawal was associated with heart rate response due to parasympathetic reinfusion, in both moderately fit and very fit groups of subjects. The correlation coefficient for very fit group parasympathetic withdrawal versus parasympathetic reinfusion was -0.81 at $p < 0.05$ and was found to be significant. The correlation coefficient for moderately fit group parasympathetic withdrawal versus parasympathetic reinfusion was 0.65 at $p < 0.05$ and was found to be non significant. A t-test for independent samples was used to determine the difference between the heart rate response due parasympathetic withdrawal and the heart rate response due to parasympathetic reinfusion for very fit group of subjects. The difference was found to be statistically significant with t-score of -7.24 and p-value of 0.002. A t-test for independent samples was used to determine the difference between the heart rate response due to parasympathetic withdrawal and the heart rate response due to parasympathetic reinfusion for the moderately fit group of subjects. The difference was found to be statistically significant with t-score of -16.8 and p-value of 0.001.

Table 1:- Mean and standard deviation of demographic data.

	Age (y)	Height (cm)	Weight (KG)	Max VO2 (L/min)	Max VO2 (ml/kg/min)
Mean	25.74	173.09	74.52	4.53	59.82
Standard deviation	8.05	29.69	14.05	0.52	8.16

Figure 1:- Graph depicting the difference between very fit and moderately fit group *VF=very fit; MF=moderately fit.

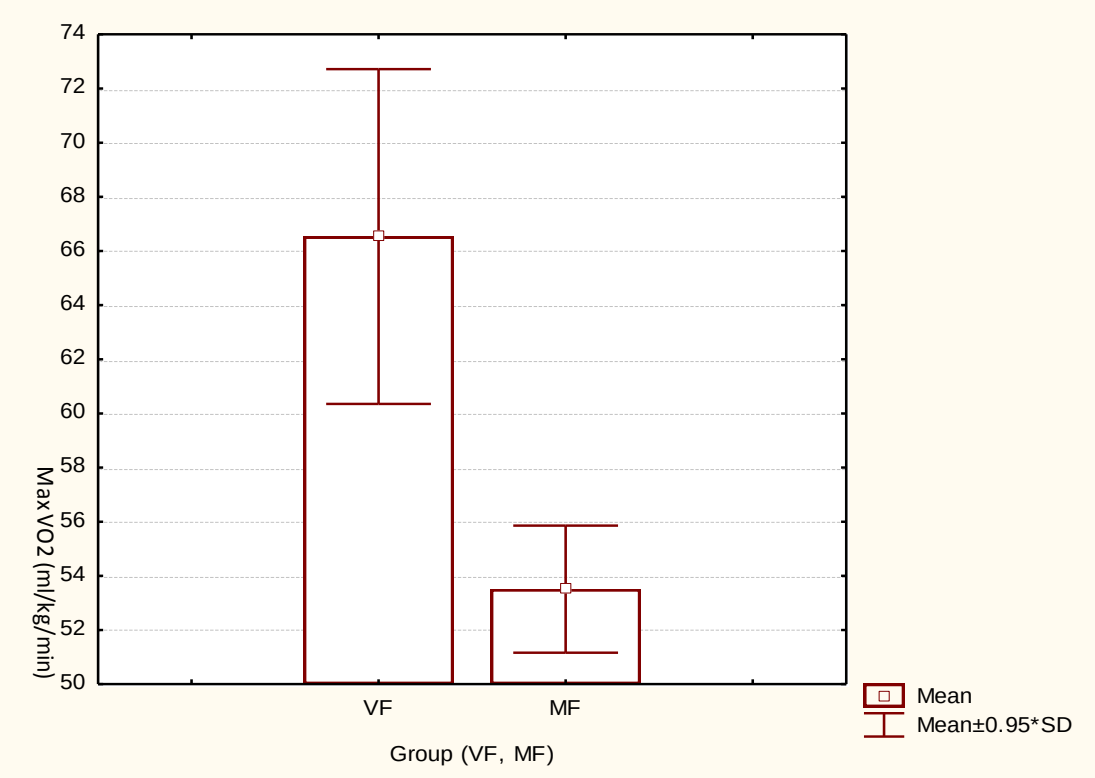
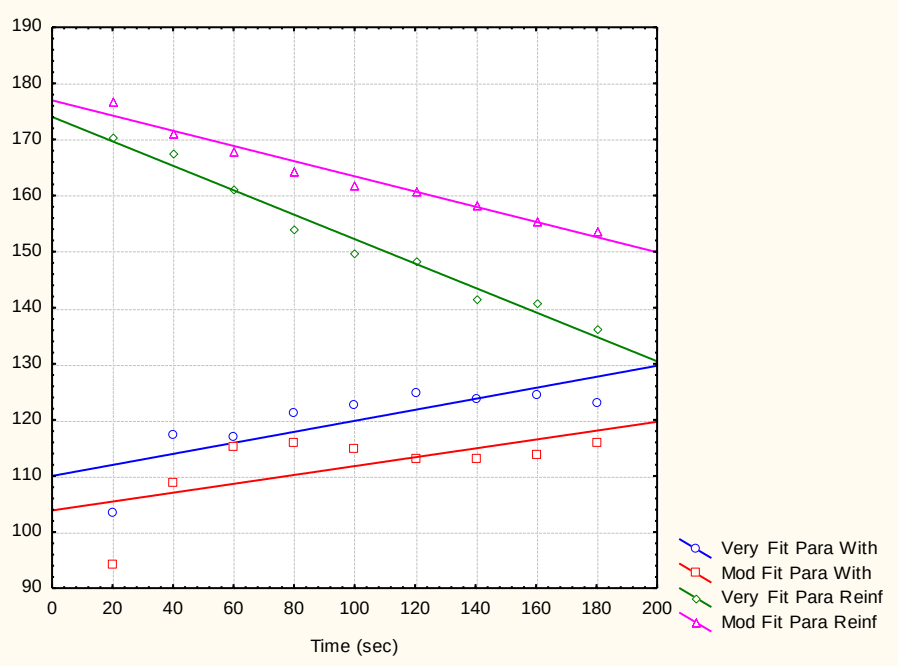


Figure 2:- Scatter plot depicting slope.



*Very Fit Para With=very fit parasympathetic withdrawal; Mod Fit Par With=moderately fit parasympathetic withdrawal; Very Fit Para Reinfusion = very fit parasympathetic reinfusion; Mod Fit Para Reinfusion = moderately fit parasympathetic reinfusion.

Discussion:-

The purpose of this study was to investigate the influence of the parasympathetic system on the heart rate after the onset of exercise and the immediate post exercise recovery. The research literature suggests that changes in cardiac autonomic nervous system modulation partly contribute to a decrease in heart rate measured at rest and during post exercise recovery periods (Yamamoto, Miyachi, Saitoh, Yoshate oka, & Onodera, 2000). Current study included 33 male subjects with high aerobic capacity which is different as compared to the previous studies. The subjects were divided into two broad categories on the basis of their Max VO_2, which is the measure of the aerobic capacity of the individual. Figure 1 suggests the difference in Max VO_2 of the very fit and the moderately fit subjects included in the study. This difference in Max VO_2 between the two groups suggests that very fit group subjects have higher aerobic capacity as compared to the moderately fit group subjects. The 50^{th} percentile of the MaxVO_2 was determined by using the median for the group. The median value obtained was 57.2 ml/kg/min. The subjects with the MaxVO_2 above 57.2 ml/kg/min were grouped into a very fit category of the individuals, and the subjects with MaxVO_2 below 57.2 ml/kg/min were grouped into the moderately fit category. There were sixteen (n=16) subjects in the very fit group and seventeen (n=17) subjects in the moderately fit group. The difference between the two groups fitness status was found to be statistically significant by using a t-test, independent by groups with t-score of 7.7 and p value of 0.00. The mean MaxVO_2 for the very fit group was 66.53 and for the moderate fit group were 53.50, with a p-value of 0.001. The standard deviation for the very fit group was 6.50 and for the moderately fit group were 2.47. This difference in Max VO_2 between the subjects can be explained by the duration of their endurance training and their fitness status at the time of testing. There is a linear increase in aerobic power after a prolonged duration of endurance training (Hickson, Bomze, & Holloszy, 1977). There are multiple adaptations occurring in the body due to endurance training stimulus. Some adaptations occur within the muscles themselves, promoting more efficient transport and utilization of oxygen and fuel substrates (Wilmore, Costill, & Kenney, 2008). There are also cardiovascular and pulmonary adaptations taking place which help in improving the overall aerobic capacity (Wilmore, Costill, & Kenney, 2008). Each group had two comparisons: parasympathetic withdrawal and parasympathetic reinfusion. At rest, activity of the parasympathetic system predominates, and the heart is said to have vagal tone (Wilmore, Costill, & Kenney, 2008). Physical exercise is associated with parasympathetic withdrawal and increased sympathetic activity resulting in heart rate increase (Javorka, Zila, Balharek, & Javorka, 2002). After the onset of exercise the heart rate first increases due to withdrawal of vagal tone, with further increase if necessary due to sympathetic activation (Wilmore, Costill, & Kenney, 2008). Sympathetic activation occurs through feedback mechanisms from peripheral mechanical and chemical receptors (Hoffman, 2002). This is to meet the increased demand of the body after the onset of exercise. In current study the parasympathetic withdrawal consists of the first three minute heart rate measures of the subject. After the exercise is terminated post exercise recovery period starts. The parasympathetic reinfusion occurs in the post exercise recovery period. Parasympathetic reinfusion consisted of last three minute heart rate measures of the subject. Heart rate recovery after exercise involves a coordinated interaction of parasympathetic re-activation and sympathetic withdrawal (Borresen & Lambert, 2008). The parasympathetic reinfusion during the post exercise recovery period plays a very important role in bringing the heart rate back to normal and establishing the homeostasis in the body. The endurance-trained athletes have an accelerated heart rate recovery after exercise (Borresen & Lambert, 2008). Since the autonomic nervous system is interlinked with many other physiological systems, the responsiveness of the autonomic nervous system in maintaining homeostasis may provide useful information about the functional adaptations of the body (Borresen & Lambert, 2008). Figure 2 was plotted using a scatter plot, keeping time on the X-axis and the four variables: very fit parasympathetic withdrawal, very fit parasympathetic reinfusion, moderately fit parasympathetic withdrawal, and moderately fit parasympathetic reinfusion on the Y-axis. The slope for very fit parasympathetic withdrawal was 0.0982, and the slope for moderately fit parasympathetic withdrawal was 0.079. The slope for very fit parasympathetic withdrawal was steeper as compared to the slope for moderately parasympathetic withdrawal. This finding suggests that subjects in the very fit group have faster parasympathetic withdrawal as compared to subjects in the moderately fit group. During peak exercise older subjects with moderate fitness had less parasympathetic withdrawal as compared to young very fit subjects (Levy et al., 1998). The slope for very fit parasympathetic reinfusion was -0.217, and the slope for moderately fit parasympathetic reinfusion was -0.135. The slope for very fit parasympathetic reinfusion was steeper as compared to the slope for moderately parasympathetic reinfusion. This finding suggests that subjects in the very fit group have faster recovery as compared to subjects in the moderately fit group. The vagal mediated heart rate recovery after exercise is accelerated in well trained, but blunted in patients with chronic heart failure (Katsuj et al., 1994). Lambert and colleagues performed a study on changes in heart rate recovery after high intensity training in well trained cyclists. They found that heart rate recovery was significantly improved in well trained cyclists after high intensity training sessions (Lamberts, Swart, Noake, & Lambert, 2009). They also concluded that heart rate recovery is a sensitive marker which tracks changes

in training status in already well trained cyclists and has the potential to have an important role in monitoring and prescribing training (Lamberts, Swart, Noake, & Lambert, 2009). A decrease in resting heart rate and a relative heart rate at any particular stage of exercise at any given sub maximal VO_2 is a commonly found adaptation in long term endurance training (Blomqvist & Saltin , 1983; Charlton & Crawford, 1997). However, the magnitude in the heart rate reduction during endurance training may be much smaller than that reflected in some of the cross sectional studies comparing elite endurance athletes with sedentary controls (Wilmore, Costill, & Kenney, 2008). Further, the t-test for independent samples was run between all four groups. The difference between heart rate response due to parasympathetic withdrawal in very fit group and heart rate response due to parasympathetic withdrawal in moderately fit group was found to be statistically significant with t-score of 2.5 and p-value of 0.02. This suggests that fitness status affects the parasympathetic withdrawal in the subjects. The difference between heart rate response due to parasympathetic reinfusion in very fit group and heart rate response due to parasympathetic reinfusion in moderately fit group was also found to be statistically significant with t-score of -2.35 and p-value of 0.03. This finding suggests that fitness status affects the parasympathetic reinfusion in the subjects. A post hoc correlation analysis was run to determine if heart rate response due to parasympathetic withdrawal was associated with heart rate response due to parasympathetic reinfusion in both moderately fit and very fit groups of subjects. The correlation coefficient for very fit group parasympathetic withdrawal versus parasympathetic reinfusion is -0.81 at $p < 0.05$ and is found to be significant. This finding suggests that there is significant association between heart rate response due to parasympathetic withdrawal and heart rate response due to parasympathetic reinfusion in the very fit group of subjects. The correlation coefficient for moderately fit group parasympathetic withdrawal versus parasympathetic reinfusion is 0.65 at $p < 0.05$ and is found to be non significant. This finding suggests that there is no significant association between heart rate response due to parasympathetic withdrawal and heart rate response due to parasympathetic reinfusion in moderately fit group of subjects. A t-test for independent samples was used to determine the difference between the heart rate response due to parasympathetic withdrawal and heart rate response due to parasympathetic reinfusion for very fit group of subjects, and the difference was found to be statistically significant with t-score of -7.24 and p-value of 0.002. This finding suggests that there is significant difference between heart rate response due to parasympathetic withdrawal and heart rate response due to parasympathetic reinfusion for the very fit group of subjects. A t-test for independent samples was used to determine the difference between the heart rate response due to parasympathetic withdrawal and heart rate response due to parasympathetic reinfusion for the moderately fit group of subjects, and the difference was found to be statistically significant with t-score of -16.8 and p-value of 0.001. This finding suggests that there is significant difference between heart rate response due to parasympathetic withdrawal and heart rate response due to parasympathetic reinfusion for the moderately fit group of subjects.

Conclusion:-

The current study suggests that there is a significant difference in Max VO_2 between the very fit and the moderately fit groups. There is a significant difference in heart rate response due to parasympathetic withdrawal between subjects of very fit and moderately fit group. It is also found that a significant difference also exists for heart rate response due to parasympathetic reinfusion between subjects of very fit and moderately fit groups. Correlation study also suggests that there is significant association between heart rate response due to parasympathetic withdrawal and parasympathetic reinfusion for the very fit group of subjects. Hence the H_0 hypothesis is rejected and the alternative hypothesis H_1 is accepted. But there was no significant association found between heart rate response due to parasympathetic withdrawal and parasympathetic reinfusion for moderately fit group of subjects. Hence the H_0 hypothesis is accepted and the alternative hypothesis H_1 is rejected. Further analysis suggests that there is a significant difference found between heart rate response due to parasympathetic withdrawal and parasympathetic reinfusion for the very fit group of subjects. Hence the H_{02} hypothesis is rejected and the alternative hypothesis H_2 is accepted. Also there exists a significant difference between heart rate response due to parasympathetic withdrawal and parasympathetic reinfusion for moderately fit group of subjects. Hence the H_{02} hypothesis is rejected and the alternative hypothesis H_2 is accepted.

References:-

1. Alcaraz, P. E., Sánchez, L. J., & Blazevich , A. J. (2008). Physical Performance and Cardiovascular Responses to an Acute Bout of Heavy Resistance Circuit Training versus Traditional Strength Training. Journal of Strength & Conditioning Research, 667-671.
2. Alexander, L. R. (1970). Cardiovascular Responses to Static Exercise. Journal of American Heart Association, 173-176.

3. Alonso, J. G., Rodríguez , R. M., & Coyle , E. F. (2000). Stroke volume during exercise: interaction of environment and hydration. American Journal of Physiology, 321-330.
4. Arai, Y., Saul, J. P., Albrecht, P., Hartley, L. H., Lilly, L. S., Cohen , R. J., et al. (1989). Modulation of cardiac autonomic activity during and immediately after exercise. American Journal of Physiology, 132-141.
5. Baechle, T. R., Earle, R. W., & Ratamess, N. A. (2008). Essentials of Strength Training and Conditioning. Champaign IL: Human Kinetics.
6. Blomqvist, C. G., & Saltin , B. (1983). Cardiovascular adaptations to training. Annual review of physiology, 169-189.
7. Bonow, R. O. (1994). Cardiovascular response to exercise. Newyork: Futura.
8. Borresen, J., & Lambert, M. I. (2008). Autonomic Control of Heart Rate during and after Exercise. Sports Medicine , 633-646.
9. Buch , A. N., Coote, J. H., & Townend, J. N. (2002). Cardiac vagal control and physical training. Journal of Exercise Physiology, 423-435.
10. Buchheit , M., Haddad, H. A., Millet, G. P., Lepretre, P. M., Newton, M., & Ahmadi, S. (2009). Cardiorespiratory and Cardiac Autonomic Responses to 30-15 Intermittent Fitness Test in Team Sport Players. Journal of Strength & Conditioning Research, 93-100.
11. Buchheit, M., & Gindre, C. (2006). Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load. American Journal of Physiology, 451-458.
12. Bunca, V., Hellera, J., & Lesoa, J. (1988). Kinetics of heart rate responses to exercise. Journal of Sports Sciences, 39-48.
13. Carrol, J. F., Convertino, V. A., Graves, W. J., Lowenthal, D. T., & Pollack, M. L. (1995). Effects of training on blood volume and plasma hormone concentrations in the elderly. Medicine and Science in sports and exercise, 79-84.
14. Carter, J. B., Banister, E. W., & Blaber, A. P. (2003). The effect of age and gender on heart rate variability after endurance training. Medicine and Science in Sports and Exercise, 1333-1340.
15. Charlton , G. A., & Crawford, M. H. (1997). Physiological consequences of training . Cardiology clinics, 345-354.
16. Constance , M. M., Michael , J. T., & Ali , A. E. (1997). Cardiovascular adaptations to 10 days of cycle exercise. Journal of Applied Physiology, 1900-1906.
17. Convertino , V. A., Rickards , C. A., & Ryan, K. L. (2012). Autonomic mechanisms associated with heart rate and vasoconstrictor reserves. Official Journal of the Clinical Autonomic Research Society, 123-130.
18. Convertino , V., Hung , J., Goldwater , D., & DeBusk, R. F. (1982). Cardiovascular responses to exercise in middle-aged men after 10 days of bedrest. Journal of American Heart Association, 134-140.
19. Convertino, V. A. (1991). Blood volume: Its adapattion to endurance training. Medicine and Science in Sports and Exercise, 1338-1348.
20. Cunningham, D. A., Paterson, D. H., Blimkie, C. J., & Donner, A. P. (1984). Development of cardiorespiratory function in circumbertal boys. Journal of Applied Physiology, 302-307.
21. Epstein, S. E., Beiser, D. G., Stampfer, M., Brain, F. R., & Braunwald, E. (1967). Characterization of the Circulatory Response to Maximal Upright Exercise in Normal Subjects and Patients with Heart Disease. Journal of American Heart Association, 1049-1062.
22. Fleck, S. J. (1988). Cardiovascular adaptations to resistence training. Medicine and science in sports and exercise, 146-151.
23. Ford, L. E. (1976). Heart size. Circulatory Research, 299-303.
24. Fry, A. C., & Parks, M. J. (2001). Tachycardia During Resistance Exercise: A Case Study. Journal of Strength & Conditioning Research, 401-529.
25. Glass, S. C., & Holcomb, R. R. (1997). Heart Rate Response Associated With Non-Paced Exercise Prescription Using Ratings of Perceived Exertion. Journal of Strength & Conditioning Research, 211-288.
26. Goldberg, L., Elliot, D. L., & Kuehl, K. S. (1988). Cardiovascular Changes at Rest and During Mixed Static and Dynamic Exercise after Weight Training. Journal of Strength & Conditioning Research, 39-59.
27. Goldberg, L., Elliot, D. L., & Kuehl, K. S. (1994). A Comparison of the Cardiovascular Effects of Running and Weight Training. Journal of Strength & Conditioning Research, 209-275.
28. Gordon, P. L., & Eric, J. V. (2004). Assessing autonomic function by analysis of heart rate recovery from exercise in healthy subjects. The American Journal of Cardiology, 64-68.
29. Gotshalk , L. A., Berger , R. A., & Kraemer , W. J. (2004). Cardiovascular Responses to A High-Volume Continuous Circuit Resistance Training Protocol. Journal of Strength & Conditioning Research, 689-923.

30. Greene, N. P., Greene , E. S., Carbuhn , A. F., Green, J. S., & Crouse, S. F. (2011). VO2 Prediction and Cardiorespiratory responses during Underwater Treadmill Exercises. Research quarterly for exercise and sport, 264-273.
31. Gulati , M., Shaw, L. J., Thisted, R. A., Black , H. R., Bairey, M. C., & Arnsdorf , M. F. (2010). Heart Rate Response to Exercise Stress Testing in Asymptomatic Women : The St. JamesWomen Take Heart Project. Journal of American Heart Association, 130-137.
32. Guyton, A. C., & Hall, J. E. (2006). Text book of Medical Physiology. Pennsylvania: Elsevier Saunders.
33. Haskell, W. L., & DeBusk, R. (1979). Cardiovascular responses to repeated treadmill exercise testing soon after myocardial infarction. Journal of American Heart Association, 1247-1251.
34. Hermansen, L., & Saltin, B. (1969). Oxygen uptake during maximal treadmill and bicycle exercise. Journal of Applied Physiology, 31-37.
35. Hickson, R. C., Bomze, H. A., & Holloszy, J. O. (1977). Linear increase in aerobic power induced by a strenuous program of endurance exercise. Journal of Applied Physiology , 372-376.
36. Higginbotham, M. B., Morris, K. G., Coleman , R. E., & Cobb, F. R. (1984). Sex-related differences in the normal cardiac response to upright exercise. Journal of American Heart Association, 357-366.
37. Higginbotham, M. B., Morris, K. G., Williams, R. S., McHale,, P. A., Coleman, R. E., & Cobb, F. R. (1986). Regulation of stroke volume during submaximal and maximal upright exercise in normal man. Journal of American Heart Association, 281-291.
38. Hill, D. W., Collins, M. A., Cureton, K. J., & DeMello, J. J. (1989). Blood Pressure Response After Weight Training Exercise. Journal of Strength & Conditioning Research, 22-50.
39. Hoffman, J. (2002). Physiological Aspects of Sport Training and Performance. Champaign: Human Kinetics.
40. Horwitz, L. D., Atkins, J. M., & Leshin, S. J. (1972). Role of the Frank-Starling Mechanism In Exercise. The Journal of American Heart Association, 868-875.
41. Hughson, R. L., & Tschakovsky, M. E. (1999). Cardiovascular dynamics at the onset of exercise. Medicine & Science in Sports & Exercise, 1005-1010.
42. Javorka, M., Zila, I., Balharek, T., & Javorka, K. (2002). Heart rate recovery after exercise:relations to heart rate variability and complexity. Brazilian journal of Medical and Biological research , 991-1000.
43. Kanakis, C., & Hickson, R. C. (1980). Left ventricular responses to a program of lower-limb strength training. Chest physiology, 618-621.
44. Kannankeril, P. J., & Goldberger, J. J. (2002). Parasympathetic effects on cardiac electrophysiology during exercise and recovery. American Journal of Physiology, 2091-2098.
45. Kannankeril, P. J., Francis, L. K., Kadish, A., & Goldburger, J. (2004). Parasympathetic effects on heart rate recovery after exercise. Journal of investigative medicine , 394-401.
46. Katsuj, I., Hideyuki , S., Masatsugu, H., Hideo , K., Hitoshi , O., Yokoyama, H., et al. (1994). Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patients with chronic heart failure. Journal of the American College of Cardiology, 1529-1535.
47. Khurana, I. (2008). Essentials of Medical Physiology. Noida: Elsevier.
48. Klabunde, R. E. (2012). Cardiovascular Physiology Concepts. Baltimore: Lippincott William & Wilkins.
49. Kleiner, D. M., Blessing, D. L., Davis, W. R., & Mitchell, J. W. (1996). Acute Cardiovascular Responses to Various Forms of Resistance Exercise. Journal of Strength & Conditioning Research, 63-64.
50. Kravitz, L., Greene, L., Burkett, Z., & Wongsathikun, J. (2003). Cardiovascular Response to Punching Tempo. Journal of Strength & Conditioning Research, 205-208.
51. Laird, W. P., Fixler, D. E., & Huffines, F. D. (1979). Cardiovascular response to isometric exercise in normal adolescents. Journal of American Heart Association, 651-654.
52. Lamberts, R. P., Lemmnik, K. A., Durandt, J. J., & Lambert, M. I. (2004). Variation in Heart Rate During Submaximal Exercise: Implications for Monitoring Training. Journal of Strength & Conditioning Research, 401-684.
53. Lamberts, R. P., Swart, J., Noake, T. D., & Lambert, M. I. (2009). Changes in heart rate recovery after high-intensity training in well-trained cyclists. European Journal of Applied Physiology , 705-713.
54. Lauer, M. S., Okin, P. M., Larson, M. G., Evans, J. C., & Levy, D. (1996). Impaired Heart Rate Response to Graded Exercise. Journal of American Heart Association, 1520-1526.
55. Lepley, A. S., & Hatzel, B. M. (2010). Effects of Weightlifting and Breathing Technique on Blood Pressure and Heart Rate. Journal of Strength & Conditioning Research, 2179-2183.
56. Levy, W. C., Cerqueira, M. D., Harp, G. D., Johannessen, K.-A., Abrass, I. B., Schwartz, R. S., et al. (1998). Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. The American Journal of Cardiology , 1236-1241.

57. Lewis, S. F., Taylor, W. F., Graham, R. M., Pettinger, W. A., Schutte, J. E., & Blomqvist, C. G. (1983). Cardiovascular responses to exercise as functions of absolute and realtive workload. Journal of Applied Physiology, 1314-1323.
58. Lusiani , L., Ronsisvalle , G., Bonanome, A., Visona, A., Castellani , V., Macchia , C., et al. (1986). Echocardiographic evaluation of the dimensions and systolic properties of the left ventricle in freshman athletes during physical training. European Heart Journal, 196-203.
59. Maron, B. J. (1986). Structural features of the athletic heart as defined by echocardiography. Journal of the American College of Cardiology, 190-203.
60. Mayo, J. J., & Kravitz, L. A. (1999). Review of the Acute Cardiovascular Responses to Resistance Exercise of Healthy Young and Older Adults. Journal of Strength & Conditioning Research, 3-96.
61. McArle, W. D., Katch, F. I., & Katch, V. L. (1996). Exercise Physiology Energy Nutrition and Human Performance. Pennsylvania: Williams & Willkins.
62. Mier, C. M., Turner , M. J., Ehsani , A. A., & Spina , R. J. (1997). Cardiovascular adaptations to 10 days of cycle exercise. Journal of Applied Physiology, 1900-1906.
63. Miller, P. C., Hall, E. E., Chmelo, E. A., Morrison, J. M., Dewitt , R. E., & Kostura , C. M. (2009). The Influence of Muscle Action on Heart Rate, RPE, and Affective Responses After Upper-Body Resistance Exercise. Journal of Strength & Conditioning Research, 366-372.
64. Murray, S. R., Michael, T. J., & McClellan, P. D. (1995). The Influence of Fluid Replacement Rate on Heart Rate and RPE During Exercise in a Hot, Humid Environment. Journal of Strength & Conditioning Research, 211-274.
65. Ogawa, T., Spina, R. J., Martin, W. H., Kohrt, W. M., Schechtman, K. B., Holloszy, J. O., et al. (1992). Effects of aging, sex, and physical training on cardiovascular responses to exercise. Journal of American Heart Association, 494-503.
66. Olson, M. S., Williford, H. N., & Wang, N. (1997). Cardiorespiratory Responses to Lateral Motion Exercise With and Without Ankle Weights. Journal of Strength & Conditioning Research, 211-288.
67. Pearson, A. C., Schiff, M., Mrosek, D., Labovitz, A. J., & Williams, G. A. (1986). Left ventricular diastolic function in weight lifters . American Journal of Cardiology, 1254-1259.
68. Pichon, C. E., Hunter, G. R., Morris, M., Bond, R. L., & Metz, J. (1996). Blood Pressure and Heart Rate Response and Metabolic Cost of Circuit Versus Traditional Weight Training. Journal of Strength & Conditioning Research, 139-210.
69. Queiroz, A. C., Gagliardi, J. F., Forjaz, C. L., & Rezk, C. C. (2009). Clinic and Ambulatory Blood Pressure Responses After Resistance Exercise. Journal of Strength & Conditioning Research, 571-578.
70. Rerych, S. K., Scholz, P. M., Newman, G. E., Sabiston, D. C., & Jones, R. H. (1977). 6Cardiac Function at Rest and During Exercise in Normals and in Patients with Coronary Heart Disease. Annals of Surgery, 449-463.
71. Ricci, G., Lajoie, D., Petitclerc, R., Peronnet, F., Ferguson, R. J., Fournier, M., et al. (1982). Left ventricular size following endurance, sprint, and strength training . Medicine and Science in Sports and Exercise, 344-347.
72. Rodríguez-Marroyo, J. A., Villa, J. G., García-López , J., & Foster, C. (2012). Comparison of Heart Rate and Session Rating of Perceived Exertion Methods of Defining Exercise Load in Cyclists. Journal of Strength & Conditioning Research, 2249–2257.
73. Rozenek, R., Rosenau, L., Pat, R., & Stone, M. H. (1993). The Effect of Intensity on Heart Rate and Blood Lactate Response to Resistance Exercise. Journal of Strength & Conditioning Research, 51-61.
74. Savin, Savin, W. M., Davidson, D. M., & Haskell, W. L. (1982). Autonomic contribution to heart rate recovery from exercise in humans. The journal of applied physiology, 1572-1575.
75. Schaefer, M. E., Allert, J. A., Adams, H. R., & Laughlin, M. H. (1992). Adregenic responsiveness and intrinsic sinoatrial automaticity of exercise trained rats. Medicine and Science in Sports and exercise, 887-894.
76. Shapiro, L. (1997). The morphological consequencies of systemic training . Cardiology clinics, 373-379.
77. Silverthorn, D. U. (2007). Human Physiology: An integrated approach. San Francisco: Benjamin Cummings.
78. Sitkowski, D., Starczewska, J. C., & Burkhard, K. J. (2004). Determination of Anaerobic Threshold Based On The Dynamics of the Heart rate and Stroke volume estimated in the upper body progressive test. Biology of Sport Poland, 337-350.
79. Smith, D., & Fernhall, B. (2011). Advance Cardiovascular Exercise Physiology. Illinois: Human Kinetics.
80. Spirito, P., Pelliccia, A., Proschan, M., Granata, M., Spataro, A., Bellone, P., et al. (1994). Morphoplogy of Athletes heart assessed by echocardiography . American Journal of cardiology, 802-806.
81. Stone, M. H., Wilson, G. D., Blessing, D., & Rozenek, R. (1983). Cardiovascular response to short term Olympic style weight training in young men. Canadian Journal of Applied Sports Science, 134-139.

82. Stratton, J. R., Levy, W. C., Cerqueira, M. D., & Schwart, R. S. (1994). Cardiovascular responses to exercise. Effects of aging and exercise training in healthy men. Journal of American Heart Association, 1648-1655.
83. Sullivan, J. J., Knowlton, R. G., DeVita, P., & Brown, D. D. (1996). Cardiovascular Response to Restricted Range of Motion Resistance Exercise. Journal of Strength & Conditioning Research, 53-64.
84. Tanaka, H., Monahan, K. D., & Seals, D. R. (2001). Age-Predicted Maximal Heart Rate Revisited. Journal of the American College of Cardiology, 0735-1097.
85. Tulppo, M. P., Huikuri, H. V., Tutungi, E., Kimmerly, D. S., Gelb, A. W., Hughson, R. L., et al. (2005). Feedback effects of circulating norepinephrine on sympathetic outflow in healthy subjects. American Journal of physiology, 710-715.
86. Wilmore, J. H., Costill, D. L., & Kenney, L. W. (2008). Physiology of Sport and Exercise. Champaign: Human Kinetics.
87. Yamamoto, Y., Hughson, R. L., & Peterson, J. C. (1991). Autonomic control of heart rate during exercise studied by heart rate variability spectral analysis. The Journal of Applied Physiology, 1136-1142.
88. Yamamoto, K., Miyachi, M., Saitoh, T., Yoshioka, A., & Onodera, S. (2000). Effects of endurance training on resting and post-exercise cardiac autonomic control. Official Journal of the American College of Sports Medicine, 1496-1502.