CHAPTER I
INTRODUCTION

Due to the high degree of automatization, modern man is experiencing a high degree of inactivity which is becoming an increasingly significant factor in the appearance of a great number of illness. In these current living conditions where technological development has directed man's activities from physical to intellectual labour, modern man is increasingly susceptible to a sedentary lifestyle. This brings about a decrease in physical activity, and thus leads to the endangerment of the health and normal functioning of organs and systems of organs (Hollman, 1992; Hollman & Hettinger, 2000; Weineck, 2000). The threat to the health of sedentary individuals is conditioned by a decrease in the functioning of the locomotor, cardio-vascular, and respiratory system, as well as other organs and systems of organs. Physical inactivity and a sedentary lifestyle have a very negative effect on almost all of the systems of the human body, and especially on cardiovascular functions (Fentem, 1992). The decrease in the functional abilities of the human body in the modern world, the development of hypertension and obesity are just some of the problems which can be solved by regular physical activity.

Differing exercise modalities may have unique effects on exercise response variables eliciting different physiologic and metabolic responses.
The common psychological responses that are used to evaluate effort during maximal and sub-maximal exercise are maximum oxygen consumption, heart rate, respiratory exchange ratio and blood lactate.

In clinical and sport science, various tests have been introduced to determine the fitness level of individuals. The measurement of maximal oxygen consumption (VO2 peak) through open circuit spirometry and the accumulation of lactic acid during exercise are vital through which an individual's cardiovascular fitness may be evaluated (American College of Sports Medicine, 1991).

In many cases, maximal exercise testing is not a feasible method for assessing maximal oxygen consumption (VO2 max). Such exercise testing not only requires a maximal effort, but specialised equipment and additional personnel are needed which can be costly. Sub-maximal tests were developed as a result. Not only are sub-maximal test cost effective, but they are highly practical in that they mimic a typical exercise session. While maximal tests allow for the direct measurement of VO2 max, sub-maximal tests can be used to estimate VO2 max (ACSM, 1991). An incremental exercise protocol is typically employed in order to estimate VO2 max. During each subsequent stage of exercise, HR response is measured. The test is stopped once the subject reaches a predetermined percentage of their age predicted maximum.
Heart rate can then be plotted against exercise intensity allowing for the estimate of VO2 max (ACSM, 1991).

During exercise, the point of transition from aerobic to anaerobic metabolism, which is known as anaerobic threshold (AT) Wasserman, 1994), has been used as an index to assess aerobic fitness (Wasserman, 1994), to establish an appropriate level of exercise training intensity (Casaburi et al., 1995), and to preoperatively evaluate a patient undergoing major surgery. The most accurate way to measure AT is based on the determination of the concentration of lactic acid, which requires frequent blood draws (Yoshida et al., 1981). Despite some controversy, may investigators demonstrated that AT can be accurately estimated, non-invasively, with methods based on ventilator and pulmonary gas exchange indices in laboratory conditions (Whipp, Ward and Wasserman, 1986). However, estimation of AT in subjects exercising outside of laboratory conditions is not easy and requires specialised equipment. Conconi et al., (1982) introduced a simple, non-invasive method to estimate AT that is based upon the heart rate-work rate relationship. A deflection point in the heart rate-work rate relationship occurs when exercise intensity increases from aerobic to anaerobic intensity. It has been proposed that an observed deviation from linearity in the heart rate-work rate relationship during exercise coincided with AT (Conconi et al., 1982). This test has achieved much popularity due to its relative simplicity and non-invasive nature.
There is a large body of literature concerning the heart rate deflection point at AT estimation. The physiological mechanisms of the deflection point in the heart rate-work rate relationship have yet to be completely understood. The results of the studies of the heart rate-work rate relationship are controversial; some studies showed a good relationship between the heart rate deflection point and AT (Bellarin et al., 1996; Bodner et al., 2002), while others observed no significant relationship between the two (Jones and Doust, 1997; Ozcelik and Kelestimur, 2004). However, a limited number of studies have been performed to assess the physiological effect of aerobic fitness on the heart rate work rate relationship at AT estimation. Variablity in heart rate among trained athletes and sedentary individuals is another vital parameter indicating fitness levels of individuals.

A cardinal feature of the exercise intensities on physiological response is the relationship of exercise intensity with the blood lactate profile (Poole, 1994). Constant-load exercise intensities below the LT result in a steady state VO2 profile whereas intensities above the LT are associated with "excess" VO2 consumption. The LT occurs when the accumulation of lactic acid exceeds the removal and resultant increases in blood lactate are found in the body. At low exercise intensities (below 40% of the VO2max) there may be little or no change in the lactate concentrations. As exercise intensity increases, a point is reached at which an increase in the concentration of lactate in the blood becomes evident (Gollnick, Bayly & Hodgson, 1986). It
has been suggested by Gollnick et al., (1986) that lactate production occurs at all intensities of exercise and that the difference between its production and clearance determine whether or not there is an accumulation in the blood. The accumulation of blood above that of baseline values has been typified as the lactate threshold (Steed, Gaesser & Weltman, 1994). The lactate threshold is dependent on the individual and their fitness level. At the same exercise intensity trained individuals have a higher LT than the untrained and therefore, the onset of fatigue is delayed. By determining LT, work intensities below the LT can be established so as to avoid any drift in VO2 (SC) and subsequently increase the time to exhaustion. Roston, Whipp, Davis, Cunningham, Effros, and Wasserman (1987) demonstrated a correlation between end exercise lactate levels and the increase in VO2 occurring after the 3rd minute of exercise.

The classical concept of lactate metabolism during exercise suggests that a deficit in oxygen uptake and delivery result in muscle anaerobiosis. In turn, this stimulates muscle glycogenolysis and glycolysis and the production of lactic acid. However, this view is changing rapidly because of the findings of tracer kinetic experiments. Lactate production is an ongoing process which takes place in the resting individual; moreover, during exercise, lactate production is highly correlated to metabolic rate. Moreover, not only lactate production the inevitable result of muscle glycogenolysis and glycolysis, but lactate production during exercise appears to serve at least two purposes: the
maintenance of blood glucose through gluconeogenesis and more importantly, the shuttling of oxidisable substrate, in the form of lactate, from the areas of glycogenolysis (production) to areas of high cellular respiration (removal).

Normally there is a low concentration of lactate in muscle and blood at rest – approximately 1m.Mol./kg. wet muscle or litre of blood. The source of this lactate is probably the low resting metabolic rate of muscle that occurs with a low blood flow and the fact that erythrocytes have a low and constant metabolism, the metabolic end product of which is lactate. At low exercise intensities (below about 40% of the VO₂max) there may be little or no change in these lactate concentration. As exercise intensity increases, a point is reached at which an increase in the concentration of lactate in muscle and blood become evident. The intensity of exercise that elicits the rise in lactate concentration in muscle and blood is highly variable and is highly influenced by numerous factors. Exercise at 50% or 60% of the VO₂max usually elicits a rise in the blood and muscle lactate only in relatively unfit individuals. As the exercise intensity increases, there is an exponential rise in lactate concentration in blood and muscle.

The role of lactate production is to release some of the energy contained in the glucose molecule and to transfer it to ADP for the production of ATP. The simplest explanation for this process is that lactate production is an emergency method for ATP production or, in the case in which the
oxygen uptake of the body is high or near maximal, a supplement to the maximal aerobic production of ATP.

The net production of ATP from the degradation of glycosyl units to lactate releases only about ten percent of the total energy stored in the glucose molecule. Though small, under some circumstances this ATP produces the difference between mediocre and high level performance. Lactate formation can be important when on Oxygen is available, or as is more frequently the case, to supplement the energy produces by the terminal oxidative system.

Lactic acid production, thus, is a metabolic phenomenon inherently related to any sort of muscular effort, maximal or sub-maximal. After lactate production, a part of the lactate remains in the muscle and is utilized while the remainder diffuses in to blood. Lactate of the muscle is absorbed by a number of organs such as the liver and the inactive muscle where it is metabolized. In any case, the presence of lactate cause metabolic acidosis and muscle fatigue. The amount of lactate produced is a trade-off between the intensity of exercise, number of repetitions of the exercise, bouts and time duration of the exercises.

The increase in lactate above the baseline during high intensity workload, depends on the balance between the rate lactate is produced (Ra) and the rate lactate is catabolised (Rd). During exercise Ra must depend entirely on the rate of lactate is produced in the contracting muscles, while Rd
depends on the rate lactate is utilized for gluconeogenesis or cellular respiration in the liver and tissues which are not in a lactate producing state.

Increased lactic acid concentration is usually indicative of anaerobic metabolism but studies have shown that lactate production is not controlled by a single factor but that many others contribute. Controversy still exists over the means to explain why lactic acid levels suddenly rise during incremental exercise. It is possible that this rise is partially due to the lack of oxygen available in the exercising muscle. However, the formation of lactic acid could still occur independent of the muscle cell's oxygen availability because of the rate of glycolysis, carbohydrate metabolism. The type of muscle fibres recruited also contributes. Faster twitch fibres, predominately anaerobic, tend to result in higher lactate accumulation where as slower twitch fibres, predominately aerobic, tend to result in less lactate buildup. Finally, the rate of lactate removal by responsible tissues also contributes to the explanation of lactate threshold; when more is produced the result is a buildup of lactic acid. Regardless of physiological means to explain lactate threshold, the point of concern is that accumulation occurs and that this accumulation has important implications for predicting sports performance and planning training for athletes, particularly endurance athletes. Studying factors that effect lactate threshold is important for this reason; one important factor is studying the effect the environment has on an athlete, particularly lactate threshold.
The anaerobic energy system allows energy to be produced quickly without the use of oxygen. A by-product of the anaerobic energy system is lactic acid (lactate), and an accumulation of lactic acid can cause the muscle to slow down or to stop working. The measurement of blood lactic acid concentration during a graded exercise test gives an indication of the extent to which the muscle has to rely upon the anaerobic energy system. The muscle constantly produces lactic acid; therefore even at rest lactic acid is present in the blood. Blood lactate concentration, and changes in it, result from the balance between adding lactic acid to the blood, and the clearance of lactate from the blood by muscle and the heart for metabolic purposes. At rest and low exercise intensities, lactic acid's rate of removal matches its rate of formation, resulting in a stable blood lactate concentration. At higher workloads, the muscle produces a greater proportion of energy anaerobically, therefore more lactic acid is produced. This leads to an accumulation of lactate in the blood. It has been shown that the greater the exercise intensity an individual can reach before there is an accumulation of lactate in the blood, the better their endurance performance.

There are at least two apparent discontinuities or thresholds in blood lactate response to graded exercise. The first of these thresholds, Lactate Threshold (LT), is associated with the first workload at which there is a sustained increase in blood lactate above resting levels. The second of these discontinuities, Anaerobic Threshold (AT) is marked by a very rapid rise in
blood lactate concentration. This second point represents a shift from aerobic to partly anaerobic energy metabolism.

During the demands of high-intensity exercise, the cell is utilizing a lot of glucose (from glycolysis) and muscle glycogen (stored from of glucose). The final step of glucose breakdown results in the production of two molecules of pyruvate. The pyruvate molecules begin to accumulate in the cell, as well as the protons (from the splitting of ATP) from the vigorous exercise. In order to neutralize the soaring accumulation of pyruvate and protons (from the splitting of ATP), each pyruvate molecule absorbs two protons into its structure, converting to lactate. Thus, lactate production is actually a consequence of cellular acidosis and not the cause of the acidosis. More blatantly, lactate production actually retards acidosis. Lactate is a temporary 'neutralizer' or 'buffer' to the cells elevated accumulation of protons during high intensity exercise. Since increased lactate production coincides with acidosis, lactate measurement is an excellent 'indirect' market for the metabolic condition of the cell.

During high-intensity resistance training, the body's more explosive fast-twitch motor units are recruited to overcome the resistance loads. The fuel for this energy demands comes primarily from the anaerobic metabolism (phosphagen and glycolytic energy systems). Fast-twitch muscle fibres have fewer mitochondria (where cell respiration occurs as well as the uptake of
protons) than slow-twitch, or aerobic endurance fibres. Thus, during high-intensity resistance training, because of the extensive use of the fast-twitch fibres (with few mitochondria and less uptake of protons) there is a greater accumulation of protons, causing acidosis.

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Changes in specific blood lactate thresholds can also serve as distinct indicators of change in an athlete's training status. Increases in the intensity at LT (shift in the curve to the right and/or downward at lower intensities) reflect an improvement in base aerobic condition. This delayed lactate production could be due to enhanced fat oxidation and enhanced aerobic mechanisms. Increased in exercise intensity at AT, represented by a graphical shift down and/or to the right at higher workloads, may be indicative of an improvement in higher-level aerobic endurance. Possible causes may be improved lactate clearance. The blood lactate response to incremental exercise should be evaluated periodically and the training intensity adjusted as aerobic fitness improves.

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Blood lactate accumulation during exercise and the heart rate response and the recovery pattern following exercise are vital indicators of exercise intensity and fitness adaptations of individuals. Though studies investigating
exercise intensity and lactate formation have been widely studied, very few studies have attempted to study the same in relation to different age groups.

A recovery heart rate is a measurement taken to determine how well your heart to functioning. It refers to the heart’s ability to return itself to a normal rhythm after being elevated during exercise. The term heart rate refers to the number of times the heart beats per minute. As the heart muscle pumps blood out to the body, it first contract to push the blood out and then relaxes. The heart rate measures how many times per minutes that heart contracts. The average healthy heart rate around 70 to 80 beats per minute. It can go as low as 40 to 60 in athletes.

There is the target heart rate which is the rate to which you want to raise the pulse inorder to make the work out effective. There is a resting heart rate, which refers to how hard the heart works while doing everyday activities. Measuring the recovery heart rate tests the hearts ability to return to the normal resting heart rate after reaching the target heart rate. The more efficient and healthy the heart is, the quicker the return to the resting heart rate will be.

To find out the recovery heart rate, first take and record the pre-exercise heart rate. Then exercise on a treatment or go up and down on an aerobic step for a given period of time. The heart will be monitored during the exercise session, after stopping the exercise, sit and take the heart rate to see
how high it was elevated. Then take the recovery heart rate every 15 seconds for the first minute. Then once every minutes until it returns to the pre exercise level. This is one’s recovery heat rate; the amount of time it took for the heart rate the recover from exercise. If some one is fit and in good shape, the heart rate should recover quickly. The heart rate should return to pre exercise level with in 10 to 15 minutes after exercise. Normal heart rate recovery is defined a decrease in the pulse of 15 to 25 beats per minute.

Researchers at the Cleveland clinic exercised patients on a treadmill, then measured their heart rate one minute after running stopped and compared it to their peak heart rate. The failure of heart rate to fall rapidly after exercise stopped was associated with increased over all mortality Cole CR et al. (1999).

To Measure recovery heart rate, exercise on a treadmill until the breadth hard, record the heart rate, and hold that pace for at least a minute. Then cool down and measure the pulse rate exactly one minute after stopping. If the heart does not slow down at heart 12 beats in the first minute you are poor shape and at increased risk for a heart attack. If the heart rate slows down more than fifty beats in the fist minutes, you are in excellent shape. Cole CR et al. (1999).

A unique feature of the heart is its ability to contract rhythmically without either neural or hormonal stimulation. Oxygen consumption is elevated
during acute exercise to meet the higher energy needs of the exercising muscle. As exercise intensity increases, a greater demand for energy to met by an increase in the cardiac output or by a greater oxygen extraction from the vasculature.

Heart rate elevation during exercise is primarily controlled by the stimulation from the higher somatomotor centres of the brian. The heart rate response is directly proportional to linear to the intensity of exercise. As intensity of exercise increases, the heart rate will continue to increase until exercise reaches maximal intensity. A decrease in resting heart rate and a relative decrease in heart rate at any given sub-maximal exercise VO\textsubscript{2} is a commonly found adaptation in endurance training programmes (Blomquist and Saltin 1983; Charlton and Crawford 1997).

The present study attempts to study the variations in blood lactate levels and recovery heart rate among three different age groups after a sub-maximal exercise.

**STATEMENT OF THE PROBLEM**

The present study was undertaken to find out the effect of sub-maximal exercise on Blood lactate and Recovery heart rate in three specified age groups.

The present study was undertaken with the following objectives:
1. To find out whether sub-maximal exercise brings about any changes in Blood lactate concentration among the different age group of subjects.

2. To find out whether sub-maximal exercise bring about any changes in Recovery heart rate among the different age group of subjects.

**HYPOTHESES**

In the present study, the investigator makes an attempt to observe the changes in Blood lactate and Recovery heart rate following similar intensity of exercise (sub-maximal) for all the different age groups. Therefore the present study was undertaken with the following hypotheses:

1. There would not be any significant differences among the three groups in Blood lactate formation after sub-maximal exercise.

2. There would not be any significant differences among the three groups in Recovery heart rate after sub-maximal exercise.

**DELIMITATIONS**

1. The study was confined to 60 healthy men with 20 each in the following age groups: (1) between 18 and 22 years; (b) 28 – 32 years; (c) between 38 to 42 years.

2. The study was delimited to subjects belonging to Ernakulam district of Kerala.

3. The study was further delimited to the following variables only: (a) blood lactate (b) recovery heart rate.
LIMITATIONS

The study being an experimental one, the investigator has taken every step to control the factors that might affect the results of the study. However, the factors like food habits, lifestyle and daily routine of the subjects which might affect the results of the study was considered as a limitation of the study.

DEFINITIONS AND EXPLANATION OF KEY TERMS

Blood Lactate (Lactic Acid)

Lactic acid is a by-product of anaerobic glycolysis, the high accumulation of which in the muscle and blood causes fatigue (Fox and Mathews, 1976).

Recovery Heart Rate

The time required for the maximal heart rate to come back to normal heart rate. The hearts ability to return itself to a normal rhythm after being elevated during exercise Col C. Blackstone (1999).

Sub-maximal Exercise

According to Rating of perceived exertion (RPE) term coined by Borg (1982) the level of exercise at which 70% of HR max co-incided with the
exercise that feels “Some what hard” exercises will be called as sub-maximal exercise.

**SIGNIFICANCE OF THE STUDY**

A preponderance of research in recent years has been dedicated to the investigation of the effect of exercise on physiological responses. However, the physiological responses to similar sub-maximal exercise in different age groups has not been investigated. The present study undertaken with a probe into the effects of sub-maximal exercise on Blood lactate and Recovery heart rate will be of significance in the following ways:

1. The study will add to the knowledge of metabolic and physiological responses of sub-maximal exercise.

2. The study will reveal the differences in physiological response, especially Blood lactate and Recovery heart rate among different age categories of men.

3. The study will be helpful to coaches and Physical Education teachers in understanding the dynamics of recovery process after sub-maximal workloads.

4. The study will provide specific knowledge regarding lactate formation during graded sub-maximal exercise which will be beneficial in planning training schedules.
Statistical Techniques Used

The data collected from the three age groups before and after the exercise were statistically examined or significant difference by using analysis of co-variance. Whenever the F-ratio was found significant LSD post-hoc test was used to determine which of the paired mean differed significantly. In all cases the criteria for statistical significance was set at 0.05 level of confidence (p < 0.05).