THE EFFECTS OF TRANSCENDENTAL MEDITATION ON SELECTED PHYSIOLOGICAL PARAMETERS DURING REST, EXERCISE, AND RECOVERY FROM EXERCISE

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THESIS

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CHAPTER I

THE PROBLEM

Introduction

In a technological world that is constantly changing man must seek new ways to adapt to his environment. The process of adaptation is comprised of psychological, physiological, and behavioral changes that allow man to cope with the stress of daily life. It is therefore not surprising that many researchers in a variety of fields are seeking the factor or combination of factors that might lead to a better understanding of the nature of stress and a more complete adaptation to the stressors.

Similarly, it is understandable that extensive research has been done to define the ways in which stress can be avoided or at least better tolerated. In this context, techniques of relaxation have been used clinically for more than forty years in the western world for the alleviation of stress. Relaxation does seem to relieve stress but the physiological mechanisms or changes involved in this process are still inadequately studied. (Martin 1961, Selye 1956, and Wolf 1968). One researcher in the field of relaxation stated:

> How badly we need isolation and meditation occasionally. They provide psychological relaxation and refreshment. Would that more sophisticated individuals would learn of simple people and philosophers to put some period aside each day for meditation and calm reasoning.
>
> (Rathbone, 1969)

Such comments related to the improvement of general mental and physical health lead one to ask; what might be the implications of

actively reducing stress prior to athletic performance? Although numerous researchers have reported that increased stress or anxiety will act as a deterrent to motor performance (Spence 1958, Harleston 1965, Martin 1961, Wilson 1970, Pitts 1967, Demartini 1965 and Jones 1946), the effects of reduced stress on physical performance have not been studied.

Statement of the Problem

This study was designed to examine the effects of an active technique of relaxation called transcendental meditation (TM) on selected physiological parameters associated with rest, exercise and recovery.

Subproblems

1. To examine to degree of active relaxation via the technique of transcendental meditation, as shown by changes in oxygen consumption, carbon dioxide elimination, minute ventilation, heart rate, respiratory rate, blood lactate and galvanic skin resistance.

2. To examine the effects of transcendental meditation on exercise performance as measured by the same physiological variables which were mentioned in Subproblem 1, with the exception of galvanic skin resistance.

3. To examine the effects of transcendental meditation on recovery from exercise again measured by the same physiological variables, with the exception of galvanic skin resistance.

Definition and Abbreviation of Terminology

Transcendental Meditation (TM)

Transcendental meditation is defined as a technique used to actively induce a relaxed state. It is characterized by decreased oxygen consumption ($\dot{V}o_2$), carbon dioxide elimination ($\dot{V}_E co_2$), minute ventilation ($\dot{V}_E$), heart rate (HR), respiratory rate ($\mathcal{F}$), and blood lactate, with an increase in galvanic skin resistance (GSR). (Benson 1974 and Wallace 1971).

Relaxation

Relaxation is defined as the diminution of muscular and mental tension, which is characterized by decreased $\dot{V}o_2$, $\dot{V}_E co_2$, $\dot{V}_E$, HR, respiratory rate and blood lactate with an increase in GSR. (Rathbone 1969 and Jacobson 1936, 1938).

Stress

Stress is defined as a dynamic state which fluctuates within every individual. It is characterized by an increase in $\dot{V}o_2$, $\dot{V}_E co_2$, $\dot{V}_E$, HR, respiratory rate, blood lactate, blood pressure, muscular tension, organ function, blood catecholamines and other hormones which are above normal resting levels, as well as a decrease in GSR (Wolf 1968).

Anxiety

Anxiety is defined as a response or a learned response to non-specific stimuli and is characterized by behavioral, physiological, and self-assessed measures (Martin 1968). For the purpose of this study anxiety is physiologically defined by an increase in the same parameters

described under the definition of stress.

Blood Lactate (lactacidosis)

Lactic acid or the lactate ion is produced mainly via glycolytic metabolism and is measured in the blood in milligrams per 100 millilitres (mg.%) (Astrand 1970). Lactacidosis is defined as a metabolic form of acidosis resulting from a higher than normal level (10-20 mg.%) of lactic acid in the blood (Karlsson 1971A).

Galvanic Skin Resistance (GSR)

Galvanic skin resistance is defined as a measure of electrical resistance between two points on the surface of the skin. GSR is an indice of stress, and is measured directly in kilohms (Benson 1974, Orme Johnson 1973, and Hori 1970).

Oxygen Consumption ($\dot{V}o_2$)

Oxygen consumption is the volume of oxygen taken from inspired air measured in millilitres or litres per minute (ml./min.).

Carbon Dioxide Elimination ($\dot{V}_E co_2$)

Carbon dioxide elimination is the volume of carbon dioxide expired measured in millilitres or litres per minute (ml./min.).

Minute Ventilation ($\dot{V}_E$)

Minute ventilation is the volume of expired air measured in millilitres or litres per minute (ml./min.).

Fraction of Expired Oxygen ($F_{E}O_{2}$)

$F_{E}O_{2}$ is the fraction of expired oxygen in expired air measured as a percent (%).

Heart Rate (HR)

Heart rate is the number of contractions of the heart measured in beats per minute (b.p.m.).

Respiratory Rate ($\mathcal{f}$)

Respiratory rate is the number of breaths per minute measured by the same (breaths/min.).

(Consolazio 1963 and Astrand 1971)

Rationale for the Study

Stress has been defined in this study, as a dynamic state which is unique to the individual and which can fluctuate moment to moment. Stress is also thought of as a continuum above and below normal levels. These normal levels of stress are a result of the individuals daily routine, while high levels of stress are caused by a change or an upset of this routine.

Accumulation of stress over an extended period of time drives an individual to the "hypertensive" end of the stress continuum (Selye 1956). Selye the originator of the term stress as it applies to the biological function conceptualized that with an accumulation of stress man maladapts. This maladaption to stress is revealed by abnormal behavioral changes which he called the "General Adaptation Syndrome" (Selye 1956).

A number of investigators have reported that abnormally high levels of stress or induced stress caused decreases in physiological, intellectual and motor performance (Wilson 1970, Carron 1968, Pitts 1967, Demartini 1965, Denny 1965, Harleston 1965, Ryan 1962, Martin 1961, Spence 1958, Wolf 1946, and Jones 1946).

Abnormally low levels of stress which cause a decrease in $\dot{V}o_2$, $\dot{V}_E co_2$, $\dot{V}_E$, HR, and respiratory rate have been shown to result from relaxation (Benson 1974, Wallace 1971, 1970, Lader 1970, Rathbone 1969, Tart 1967, and Gellhorn 1953). Relaxation is also accompanied by a large rise in GSR which indicates low stress levels (Orme Johnson 1973, Wallace 1971, 1970, Lader 1970, Hori 1970, and Tart 1967). Benson (1974) stated that these physiological changes brought about by specific techniques of relaxation are a result of a generalized decreased sympathetic nervous sytem activity.

Jacobson in his early work on relaxation noted that although athletes could achieve greater relaxation than normal subjects they could not relax to the same extent as individuals trained in a technique of relaxation (Jacobson 1936). It has also been found that motor and intellectual performance may be improved when subjects are trained in a technique of relaxation (Abrams 1971, Blasdell 1971, Shaw 1971, Nakamura 1965A, 1965B, and Haverland 1953).

Transcendental meditation is a technique of relaxation which reduces stress and results in the decrease in a number of metabolic variables (Benson 1974, Orme Johnson 1973, Wallace 1972, 1971, 1970, and Allison 1970).

As previously mentioned stress can act as an inhibitor of physiological performance and intellectual and motor tasks may be improved with relaxation. It therefore could be rationalized that techniques of relaxation such as transcendental meditation might also improve exercise performance and recovery. Thus if athletic performance could derive benefits from transcendental meditation techniques of relaxation may have practical implications for physical educators and coaches.

Limitations of the Study

1. The effects of "history" (Campbell 1963), the specific events occurring between test sessions which could have affected the results, were not controlled in this study. However most test sessions were conducted within a period of two weeks so as to minimize the effects of this confounding variable.

2. The statistical procedures used for the analysis of the data was a limitation of the study. Multiple t tests were used because there was no computer program available for multivariate analysis of variance with repeated measures. Because multiple t tests increase the probability of commiting a type I error the alpha level was appropriately adjusted.

3. The effects of the individual subject's motivation was not controlled in this investigation except by a standard form of laboratory protocol. The protocol attempted to standardize motivation in each testing session.

4. The effects of temperature, relative humidity, barometric pressure and extraneous interference in the laboratory was not controlled

in this experiment. However all respiratory data was corrected for standard temperature and pressure.

5. It was not possible to control for a fixed time of day for testing due to laboratory time tabling. Each subject was tested at approximately the same time of day so that metabolic rhythms were constant within individuals.

6. Due to the limited number of subjects that were tested (12), there was very little external validity or generalizability of the findings in this study (Campbell 1963). The conclusions drawn from the data taken in this study gave only an indication of the effects of transcendental meditation on exercise and recovery for the twelve subjects tested. It was therefore proposed that the results would serve only as a pilot study or a basis for further investigation.

CHAPTER II

REVIEW OF THE LITERATURE

Introduction

The Review of Literature is a systematic presentation of the existing state of knowledge on transcendental meditation and related areas as they affected this study. The material is presented and briefly summarized under the following headings: Transcendental Meditation; Relaxation; Anxiety and Stress; Normal Metabolic Response to Rest, Exercise and Recovery; and Performance - Stress and Relaxation.

Transcendental Meditation

TM was founded in North America by Marharishi Mahesh Yogi who brought this technique of relaxation from India where for centuries it has been an important part of the Asian religion called Buddhism. Marharishi defines TM as an altered state of consciousness or an awakening of the mind:

> . . . turning the attention inwards toward the subtler levels of thought until the mind transcends the experience of the subtlest state of the thought and derives the source of the thought.
>
> (Marharishi 1969)

Scientists may never be able to answer the question whether or not TM brings an individual to a state where he might experience finer levels of thought as this seems to be a subjective experience. On the other hand there is physiological evidence to show that transcendental meditation exists as a deep from of relaxation (Benson 1974, Orme

Johnson 1973, Gellhorn 1972, Wallace 1971, Wallace 1970, Wenger 1961, and Bagchi 1957). Such identification is made as a function of changes in $\dot{V}o_2$, $\dot{V}_E co_2$, $\dot{V}_E$, HR, GSR, respiratory rate and blood lactate.

Transcendental meditation has attracted a wide variety of people and only became popular in the western world in the last five years. It is now predicted that there are 225,000 individuals meditating in North America, 25,000 of which are Canadian. These include businessmen from IBM and Shell Oil Company, housewives, students, doctors and other professionals (List 1972).

TM is very easily learned and involves no religious beliefs, autosuggestion or commitment to an organization although there is a world organization called the International Meditation Society (Marharishi 1966). It is usually taught on a weekend course consisting of three two hour sessions. During the first session the student is given personal instruction with a teacher who has studied under Marharishi Yogi. The individual is at this time given a "Mantra" which is a sound repeated to one's self over and over again while meditating. There is no focus of attention on any specific thoughts as the mantra is said but while meditating such thoughts do creep in. As soon as the individual becomes aware of these thoughts he goes back to repeating the mantra in his mind. In this way the individual meditates or redirects his attentions away from active thought for fifteen to twenty minutes twice a day. This period of relaxation prepares or rejuvenates him for his daily activities.

Meditation and control of automatic functions of the body have been of interest to researchers for the past forty years. The

interpretation of their investigative results has been difficult due to problems in catagorizing the subject types and the selection of testing procedures and measuring equioment (Wallace 1971, Bagchi 1957, and Sugi 1968). The responses of different subjects seems to be dependent upon their experience, the types of tests, and availability of testing equipment. However consistent physiological changes have been observed during the use of transcendental meditation under relatively standardized conditions (Akishige 1968, Anand 1961, Kasamatsu 1966, Reichert 1967, Sugi 1968, Wallace 1970, Wallace 1971, and Orme Johnson 1974).

Wallace gave the following reasons for studying TM:

1. Consistent physiological changes characteristic of a rapidly produced wakeful hypometabolic state were noted during this practice (Allison 1970, Riechert 1967, and Wallace 1970).

2. The subjects found little difficulty in meditating under experimental conditions.

3. A large number of subjects were readily available who had received uniform instruction . . .

(Wallace 1971)

In his second study (1971) the most extensive investigation in the area of meditation Wallace confirmed and extended the types of physiological changes other researchers had earlier attributed to meditation. Wallace tested thirty-six subjects each of whom acted as his own control. The testing of these subjects was done in one session which was divided into three sections. The subjects sat in a chair for a ten to twenty minute period which was called premeditation control, meditated for twenty to thirty mintues and then rested for ten to twenty minutes in a post meditation control period. Physiological

variables were monitored before, during, and after meditation and the results are shown with significant changes in Table 1.

As is shown in the aforementioned table, transcendental meditation produced significant changes in $\dot{V}o_2$ which verified the work of other researchers (Allison 1970, Wallace 1970, Rao 1968, Sugi 1968, Riechert 1967, Milles 1964, Wenger 1961, and Bagchi 1957).

A significant reduction was also seen in $\dot{V}_E co_2$ and as a result of the decreases in $\dot{V}o_2$ and $\dot{V}_E co_2$ there was no change in the respiratory quotient (RQ).

The most significant change as shown by Wallace and others was the rise in GSR (Orme Johnson 1974, Wallace 1970, Riechert 1967, Wenger 1961, and Bagchi 1957).

Blood lactate values reflected a significant decrease during meditation and Wallace suggested that this could be explained in increased muscle blood flow with a consequent increase in aerobic metabolism (Wallace 1971). This hypothesis is in accordance with the work of Bolme (1969), who reported that with induced vasodialation in the canine gastronemius an increase in blood flow as well as oxygen uptake or aerobic metabolism was found. Similar findings were obtained by Whalen (1969).

Wallace also demonstrated decreases in heart rate, respiratory rate, base excess, and arterial pH.

Although it is not shown in Table 1, Wallace also observed distinct changes in electroencephalographic (EEG) recordings. Increased intensity but persistently slow alpha waves of eight to nine cycles per second were obtained.

TABLE 1 - PHYSIOLOGICAL CHANGES BEFORE, DURING, AND AFTER MEDITATION (Wallace 1971)

Variable	Number of Subjects	Pre Control Period (means)	Meditation Period (means)	Post Control Period (means)
$\dot{V}o_2$ (ml./min.)	20	251.2	211.4**	242.1
$\dot{V}_E co_2$ (ml./min.)	15	218.7	186.8**	217.9
$\dot{V}_E$ (l./min.)	4	6.08	5.14*	5.94
f (breaths/min.)	5	13	11**	11
Blood press. systolic	6	106	108	111
diastolic	6	57	59	60
RQ	15	0.85	0.87	0.86
pH	10	7.421	7.413*	7.429
Pco_2 (mm.hg.)	10	35.7	35.3	34.0
Po_2 (mm.hg.)	10	103.9	102.8	105.3
Base excess	10	- 0.5	- 1.3**	- 1.0
HR (b.p.m.)	13	70	67*	70
Blood lactate (mg.%)	8	11.4	8.0**	7.3
GSR (kilohms)	15	90.5	234.6**	120.5
Rectal temp. (^{o}C)	5	37.5	37.4	37.3

P is the probability of the mean value of pre control period being identical to the mean of the meditation period.

** $P < 0.005$ * $P < 0.05$

Although transcendental meditation elicits the same relaxation response as hypnosis, autoregulation, sleep, zen and yoga, progressive relaxation, biofeedback, and operant conditioning (Benson 1974) both Gellhorn and Wallace stated that the physiological changes noted during TM show marked differences from these altered states of consciousness (Gellhorn 1972 and Wallace 1971). These investigators concluded this based mainly upon the differences in EEG patterns found in these various states.

Wallace also stated, "Physiological changes during TM occur simultaneously and without the use of specific feedback procedures." He suggested that this fact marked a difference between TM and operant conditioning and biofeedback. Gellhorn (1972) supported this statement when he reported that proprioceptive impulses are not necessary for the production and maintenance of tropotropic and ergotropic cognitive and emotional states. Proprioceptive feedback does seem to play an important role in modifying and facilitating these states (Gellhorn 1972).

There are a number of other benefits related to motor performance, psychology, and clinical therapy, which have been attributed to transcnedental meditation.

Shaw and Kolb (1971) found that TM speeds up reaction time. Meditators showed faster reaction times before meditation and a further improvement after meditation when they were compared with normal controls. Subjects who practice TM also performed faster and more accurately in a complex perceptual motor test (Blasdell 1971). Blasdell attributed this to better coordination, flexibility, perceptual awareness, and

neuromuscular integration.

In a study at the University of California Abrams observed that meditators increased short term and long term recall when they practiced TM on a regular basis. He concluded that transcendental meditation improves memory and learning ability (Abrams 1972).

Studies of 570 and 1,862 subjects lead Otis and Benson to conclude that the practice of transcendental meditation decreases drug abuse of non prescribed and prescribed drugs (Otis 1972, Benson 1972, 1969). Brautigam (1971) reported similar findings.

Benson in a study of thirty hypertensive patients also concluded that with practice of TM these patients could significantly decrease high systolic blood pressure. He stated that this data was quantitatively comparable to that obtained employing operant conditioning techniques (Benson 1973).

There is evidence in the literature which indicates that significant physiological and psychological changes occur with the use of transcendental meditation. These changes are summarized as follows:

1. The respiratory changes are decreases in $\dot{V}o_2$, $\dot{V}co_2$, $\dot{V}_E$, and respiratory rate with no change in RQ.
2. There is a very significant rise in GSR as well as a decrease in blood lactate.
3. Slight decreases have been found in base excess, pH, and heart rate.
4. TM produces changes that differ from those found in sleep, hypnosis, autosuggestion, progressive relaxation, biofeedback, and operant conditioning.

5. Increases have been reported in reaction time, motor performance, learning, and recall ability, with the practice of TM.
6. TM is also effective in reducing drug abuse and systolic blood pressure in hypertensive patients.

Relaxation

The review of the literature on transcendental meditation described a hypometabolic state characteristic of relaxation. What have other investigators found in their studies of relaxation? Secondly, how do these studies support the assumption that TM represents a state of deep relaxation?

It has been theorized that the relaxation response which was first called the "trophotropic" response by Hess (1957), is an integrated hypothalamic response resulting in generalized decreased sympathetic nervous system activity. This response may also be associated with increased parasympathetic discharges (Benson 1974, Gellhorn 1972, and Hess 1957).

The relaxation response which is modulated by the parasympathetic nervous system results in the relaxation of muscles, lessened cortical excitation, decreased blood pressure and respiratory rate, and pupil constriction (Benson 1974, Gellhorn 1972). Hess stated, "Let us repeat at this point that we are actually dealing with a protective mechanism against overstress belonging to the trophotropic-endophylactic system and promoting restorative processes." (Hess 1957, p. 40).

The counterpart of the relaxation response is what Hess termed the "ergotropic reaction" which corresponds to the "emergency reaction"

first described by Cannon (Hess 1957 and Cannon 1941). This reaction is now popularly referred to as the "fight or flight response" and consists of an increased sympathetic nervous system activity (Benson 1974 and Gellhorn 1972).

Benson described seven mental techniques that produce or are associated with the relaxation response (Benson 1974). These techniques are: transcendental meditation, autogenic training, hypnosis, zen and yoga, cotention, sentic cycles, and progressive relaxation. He reviewed the changes in the physiological parameters that have been studied on these techniques and this review is shown in Table 2 (Benson 1974).

Both Benson and Gellhorn concluded that physiological changes which accompany altered states of consciousness represent a shift in the trophotropic - ergotrophic balance to the trophotropic side or the central nervous system reaction called the relaxation response (Benson 1974 and Gellhorn 1972).

Gellhorn reported that a reduction in $\dot{V}_E$ is observed in states of relaxation as a result of diminished responsiveness of the somatic nervous system (Gellhorn 1953). When discussing transcendental meditation he stated that decreases seen in heart rate, respiratory rate and $\dot{V}o_2$ confirm the interpretation that trophotropic system dominance is involved. He also proposed that reduced $\dot{V}o_2$ results from relaxation of skeletal musculature (Gellhorn 1972). This same observation was made by Jones (1946) when he found that $\dot{V}_E$, $\dot{V}o_2$ and $\dot{V}_Eco_2$ were reduced with relaxation or decreased muscle tension.

A number of investigators have found that states of relaxation are accompanied by high levels of skin resistance (Lader 1970, Hori 1970,

TABLE 2 - PHYSIOLOGIC PARAMETERS SUPPORTING THE EXISTENCE OF THE RELAXATION RESPONSE DURING THE PRACTICE OF VARIOUS MENTAL TECHNIQUES (BENSON 1974)

Parameters	TM	Autogenic Training	Hypnosis	Zen and Yoga	Contention	Sentic Cycles	Progressive Relaxation
Oxygen consumption	↓	N.M.	↓	↓	N.M.	↓	N.M.
Respiratory rate	↓	↓	↓	↓	↓	↓	N.M.
Heart rate	↓	↓	↓	↓	N.M.	↓	N.M.
EEG alpha waves	↑	↑	N.M.	↑	present	N.M.	N.M.
Skin resistance	↑	↑	↑	↑	N.M.	N.M.	N.M.
Blood pressure	no change	?**	?	no change	N.M.	N.M.	N.M.
Muscle tension	N.M.*	↓	N.M.	N.M.	N.M.	N.M.	↓

* N.M. = not measured
** ? = inconclusive results
↑ = increased
↓ = decreased

Mathews 1969, Tart 1967, Hawkins 1962 and Carron 1959). It has been proposed that increased skin resistance found during meditation (Wallace 1970, 1971) and other states of relaxation reflect a lessened ergotrophic tone. Sympathetic discharges to the sweat glands responsible for skin resistance appear to be diminished (Gellhorn 1972).

Systolic blood pressure and heart rate are significantly lower in completely relaxed subjects as compared to normal subjects as a result of decreased synpathetic activity. Several researchers have reported that this reduction in sympathetic discharge lowers the heart rate and reduces peripheral resistance which in turn lowers blood pressure (Rathbone 1969, Selye 1956, Jacobson 1938 and King 1953).

Although Wallace (1970) has shown reduced cardiac output and heart rate during meditation he was unable to demonstrate any change in systolic blood pressure (Wallace 1971). He suggested that this may have been a result of the abnormally low resting blood pressures found in the meditators he tested.

In summary:

1. The relaxation response has been explained as an integrated hypothalmic-cortical response with dominance of the trophotropic system. This results in decreased sympathetic nervous system activity.
2. There are various mental techniques that produce the relaxation response including transcendental meditation.
3. Relaxation is accompanied by reduced $\dot{V}_E$, $\dot{V}_E co_2$, $\dot{V}o_2$, heart rate, respiratory rate, muscle tension, and systolic blood pressure with an increase in Galvanic skin resistance.

Anxiety and Stress

The changes in circulation, respiration, metabolism, body temperature and blood chemistry that manifest themselves in stressful situations are attributable to an overall sympathetic response of the central nervous system and perhaps more importantly to the adaptation of the endocrine system. These two systems are so complexly integrated that it is often difficult to distinguish between them in their effort to maintain homeostatic conditions in the body (Guyton 1971).

The sympathetic response that Guyton described is the same response that Hess termed "ergotropic" or Cannon first described as the "emergency reaction" (Hess 1957 and Cannon 1941). These two terms are given to the acute response to stress. Selye's theory of the "general adaptation syndrome" which was mentioned in the introduction represents a chronic behavioral response to overstress (Selye 1956).

Gellhorn stated that the ergotropic response results in increased skeletal muscle tone and diffuse cortical excitation (Gellhorn 1972). Benson adds that this response also produces dilation of pupils, increased blood pressure, and respiratory rate, and heightened motor excitability. Various researchers have made similar observations in their studies of the effects of stress and these findings are reviewed in the material that follows.

Several investigators have reported that there was a significant rise in resting $\dot{V}o_2$ in cases of induced anxiety (Pitts 1967, Schachter 1957 and Ax 1953). This increase in oxygen uptake was explained by Gellhorn as a result of abnormally high levels of muscular tension found in subjects with anxiety symptoms (Gellhorn 1953).

A number of researchers have shown that subjects with high anxiety levels have abnormally high resting concentrations of blood lactate (Pitts 1967, Demartini 1965, Cohen 1950, Wallas 1949, and Jones 1946). Demartini stated that this increase in venous blood lactate may have been due to peripheral vasoconstriction which is characteristic of hypertension. He hypothesized that vasoconstriction could cause tissue anoxia and therefore could increase cellular anaerobic metabolism. He further theorized that the energy required by vascular smooth muscle cells to sustain vascular constriction exceeded the supply of oxygen to these blood vessels and resulted in a greater formation of lactate (Demartini 1965).

Pitts offered another explanation when he suggested that anxiety symptoms might occur in normal individuals under stress as a result of an increase in lactate production in response to increased epinephrine release (Pitts 1967).

It is commonly accepted that one of the best indicies of the level of stress in an individual is galvanic skin resistance. With increased levels of stress there is increased sympathetic nervous system activity which activates the sweat glands via sympathetic discharge (Hori 1970 and Lader 1970). The production of perspiration by the sweat glands causes a decreased resistance between two points on the skin. Many researchers have shown in various studies of stress that with induced stress the skin resistance of an individual falls significantly. As previously mentioned this appeared to be due to increased stimulation of the sweat glands (Hori 1970, Lader 1970, Wilson 1970, Liederman 1964, Martin 1961, and Carron 1959).

In a review of the assessment of anxiety of physiological and behavioral measures Martin reported that diastolic and systolic blood pressures, heart rate, cardiac output and respiratory rate all increased during stressful situations (Martin 1961). Martin also found a marked decrease in GSR with induced stress. These physiological changes in human function which Martin described as an adaptation to stress is in accordance with the findings of other researchers (Wolf 1968, 1952, 1946, Harleston 1965, Selye 1956, and Jones 1946).

In summary:

1. High levels of stress in an individual are depicted by increases in heart rate, cardiac output, blood pressure, respiratory rate, blood lactate, $\dot{V}_E$, and $\dot{V}o_2$ as well as a decrease in GSR.
2. These physiological changes have been linked with an overall sympathetic response of the central nervous and endocrine systems.

Normal Metabolic Response to Rest, Exercise and Recovery

The increase in metabolism during exercise is dependent upon the intensity and duration of the exercise (Keele 1965, Astrand 1970). There are three broad classifications of the intensity of exercise found in the literature - supermaximal, maximal, and moderate exercise. The latter leads to a "steady state" (Astrand 1970). A steady state condition denotes an exercise situation where the oxygen uptake equals the oxygen requirement of the tissues. Consequently there is little or no accumulation of lactic acid in the body (Astrand 1970). During steady

state exercise, heart rate, cardiac output, $\dot{V}_E$ and $\dot{V}o_2$ remain fairly constant (Astrand 1970).

Table 3 represents normal values found by Astrand and Keele for $\dot{V}o_2$, $\dot{V}_E$, $\dot{V}_E co_2$, respiratory rate, heart rate and blood lactate during rest, moderate exercise, and maximal exercise in untrained subjects. It can be seen from this table that there is a proportional increase in these variables during moderate exercise and again during maximal exercise.

During severe exercise anaerobic processes must supply part of the energy during the early phase of exercise and lactic acid is produced. In work loads of some duration that demand more than fifty percent of an untrained individual maximum $\dot{V}o_2$, the anaerobic breakdown of glucose, called glycolysis, results in a measurable concentration of lactate in the blood (Hermansen 1972, 1967, Karlson 1971, and Nagle 1970).

It has been shown that there is a proportional increase in $\dot{V}o_2$, $\dot{V}_E$, $\dot{V}_E co_2$, and heart rate with increasing intensities of exercise (Table 3). Also there is a linear increase in $\dot{V}o_2$ measured during five minutes of exercise at increasing work loads (Astrand 1970, Keele 1965).

A linear relationship exists between $\dot{V}o_2$ and heart rate for a given exercise load but at heart rates above 170 beats per minute $\dot{V}o_2$ increases relatively more than the heart rate (Astrand 1970, 1961).

Pulmonary or minute ventilation at rest and during moderately heavy work was found to be 20 to 25 liters per liter of $\dot{V}o_2$ and 30 to 35 liters per liter of $\dot{V}o_2$ during maximal work (Saltin 1967). Astrand reported that there is a exponential increase in $\dot{V}_E$ with increased intensities of work with a relatively greater increase at heavier work

TABLE 3 - NORMAL METABOLIC RESPONSE TO REST, MODERATE EXERCISE AND MAXIMAL EXERCISE FOR UNTRAINED SUBJECTS
(ASTRAND 1970, KEELE 1965)

Variables	Rest	Moderate Exercise (900 kpm)	Maximal Exercise
Oxygen consumption (l./min.)	0.350	2.50	3.0-4.0
Carbon dioxide elimination (l./min.)	0.278	2.0-2.5	3.0-4.0
Minute ventilation (l./min.)	8.250	55-60	100-120
Respiratory rate (breaths/min.)	15.0	not given	40-45
Heart rate (beats/min.)	72.0	155-165	190-200
Blood lactate (mg.%)	10-20	50-55	120-150

loads (Saltin and Astrand 1967). The same relationship exists for respiratory rate as both respiratory rate and V_E are under the same controlling mechanisms.

Regardless of work intensity all exercise results in some degree of oxygen indebtedness to working muscle. The persistence of increased metabolic rate during recovery from exercise is due to the repayment of this "oxygen debt" which is the amount of oxygen consumed during recovery in excess of normal resting levels (Dempsey 1967).

Two portions of oxygen debt were first hypothesized by Margaria et al.(1933). Oxygen debt is still not perfectly understood and there has been much controversy in the literature about it. It is thought that the "alactacid" debt is the part of the total excess oxygen consumed during recovery resulting from repletion of the body's oxygen stores and the rapid synthesis of high energy phosphates. "Lactacid" debt is thought of as the restoration of normla chemical balance in blood and skeletal muscle mainly via the oxidation of lactate to pyruvate. Pyruvate can then be converted to stored glycogen through the process of glycogenesis (Hermansen 1972, Astrand 1970, Dempsey 1967, Huckabee 1957, and Margaria 1933).

In recent studies the examination of lactacid debt has led to the discovery that cardiac muscle, skeletal muscle as well as the liver have the capability of removing lactate from the blood and using it as a substrate during prolonged steady state exercise and recovery (Hermansen 1972, Karlson 1972, 1971A, 1971B, Rowell 1971, and Jorfelt 1970). Increased lactate removal seems to be a result of increased blood flow during exercise and recovery. It has also been shown that aerobic training results in a rise in the activity of the lactate dehydorgenase

(LDH) system which converts lactate to pyruvate (Hermansen 1972).

The exponential drop in cardiorespiratory metabolism normally observed during recovery from exercise reveals the functional adaptability of the human system in restoring homeostasis.

The regulation of respiration during rest, exercise, and recovery is extremely important in the determination of physiological responses to these three conditions. This regulatory system involves the integration of a large number of physical, chemical, and electrical or nervous variables. The partial pressure of oxygen, carbon dioxide, and the hydrogen ion concentration in the blood are the most predominant chemcial variables. Physical factors are represented by temperature changes in the body and feedback from stretch receptors in the lungs and respiratory muscles. Emotional states and stress which cause an increase in circulating adrenalin also affect respiration (Astrand 1970, Sørensen 1971, Keele 1965, Dejours 1964, and Kellogg 1964). A comprehensive summary of the regulation of respiration during exercise is presented in Figure 1 (Astrand 1970). Astrand's summary introduces several factors which are not necessary in the review of literature in the present study. It does though represent a good general outline.

Two chemosensitive areas for the control and regulation of ventilation have been demonstrated. They are as follows: 1. peripheral chemorceptors in the carotid and aortic bodies and 2. central chemoreceptors located in the brain stem (Sørensen 1971, Astrand 1970, and Kellogg 1964). The role of peripheral chemoreceptors in the control of ventilation has been shown to be related mainly with the sensitivity to changes in arterial Po_2 (Comroe 1964). The central chemoreceptive areas

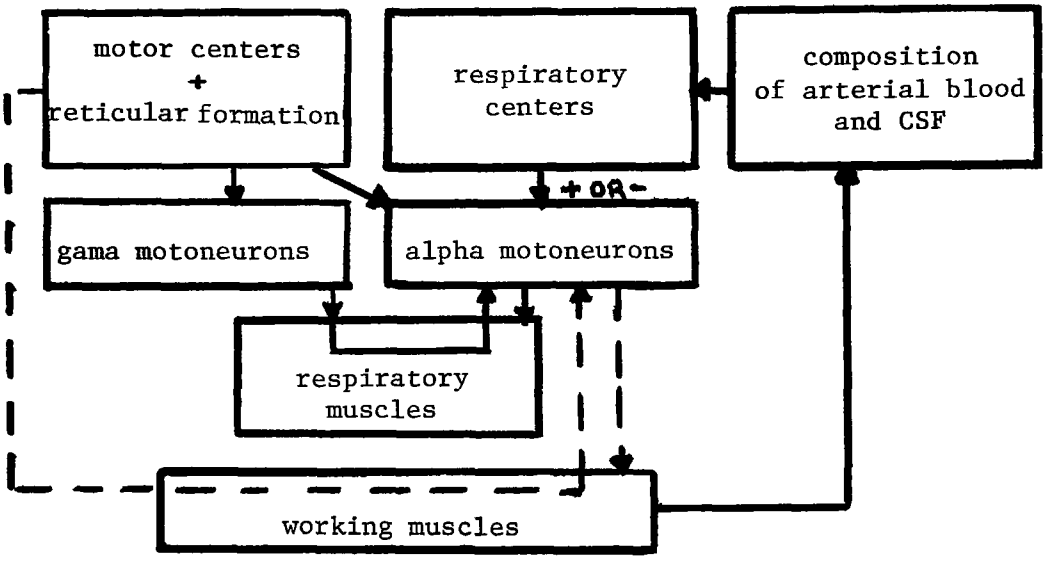

Figure 1 Schematic Summary of the Regulation of Breathing During Exercise

(Astrand 1970, p. 242)

The respiratory muscles are activated via their gama alpha motoneurons (filled-line arrows). In a similar way the other working muscles are activated (dotted-line arrows). The different respiratory centers are influenced directly by the chemical composition of the arterial blood and cerebro spinal fluid (CSF), mainly their Pco_2, Po_2 and pH. These centers can then facilitate or inhibit the motoneurons of the respiratory muscles, depending on the effectiveness of the gas exchange in the lungs. Particularly critical is the co_2 refill from the muscles and co_2 output from the lungs. (The afferent nerve impulses to motor centers include impulses from receptors located in tendons and joints.)

are sensitive to pH changes and because they react slowly to metabolic acid-base changes in the blood, function mainly as Pco_2 sensors (Sørensen 1971, Kellogg 1964).

Pulmonary ventilation at rest is mainly regulated by the central chemoreceptors and their sensitivity to the partial pressure of carbon dioxide in the blood (Sørensen 1971). An increase in H^+ and co_2 concentration (hypercapnia) in the arterial blood causes an increase in ventilation via "hypercapnic drive".

The regulation of respiration during work or exercise is most probably activated by what Dejours (1964) called the neurogenic factor. The chemoreceptor systems then act as a secondary feedback mechanism which modulate and regulate respiration according to the chemical composition of the arterial blood. Neurogenic respiratory drive is responsible for the initiation of ventilatory response during exercise. It has been postulated that the afferent impulses from exercising limbs and the stimulation form various parts of the brain due to increased motor activity are the activators of respiratory muscles (Sørensen 1971, Astrand 1970, and Dejours (1964).

The stimulation of respiration after the onset of exercise is also due to both an increase in arterial Pco_2 and a reduction of Po_2. The lowering of the partial pressure of oxygen as a result of increased utilization of oxygen causes a stimulation of the peripheral chemoreceptors which in turn signal the respiratory center in the medulla to increase respiration. This stimulation of respiration due to a lack of oxygen is called "hypoxic drive" (Sørensen 1971).

The central and peripheral chemoreceptor systems are integrated with the inspiratory and expiratory respiration centers located in the medulla oblongata. These two respiration centers are inherently rhythmic and reciprocally inervate the inspiratory and expiratory muscles via the alpha and gama motoneuron systems (Astrand 1970). This regulatory system varies the rate and depth of breathing. Euler has suggested that the switch from inspiration to expiration is controlled by additional proprioceptors in the lungs and respiratory muscle spindles (Euler 1966).

During exercise increased sympathetic nervous system activity raises the level of blood catecholamines (Biscoe 1965, Lee 1964). It has been hypothesized that vasoconstrictor fibers in the peripheral and central chemoreceptors are activated by this increased sympathetic activity. Consequently there is enough of a reduction in blood flow to produce an excitation of these chemoreceptors despite little change in arterial Po_2. This excitation of the carotid and aortic bodies stimulates respiration in the same way as hypoxia and offers a plausible explanation for the increased ventilation seen with increased emotional and exercise stress (Lee 1964, Biscoe 1965).

Another explanation of the large increase in ventilation during muscular work as compared to the relatively small changes seen in Po_2, Pco_2, and H^+ concentration is found in the examination of proprioceptive feedback (Astrand 1970, Defares 1964). Astrand stated:

> It is suggested as a hypothesis, that muscular work as such, through afferent impulses from the engaged muscle spindles and or from the central nervous system, increases the activity of the gama and alpha motoneurons of the respiratory muscles and through spinal and supraspinal reflex centers, and thus in a closely coordinated manner produces an increase in frequency and depth of respiration in pace with muscular movements.
>
> (Astrand 1970)

In summary:

1. The normal metabolic responses to rest, moderate exercise, and maximal exercise are shown in Table 3.
2. There is a proportional increase in metabolism related to the six variables shown in this table and this increase is dependent upon intensity and duration of exercise.
3. The continued increased metabolic rate found during recovery reflects the repayment of the lactacid and alatacid portions of oxygen debt.
4. A schematic summary of the regulation of respiration during exercise is shown in Figure 1. It involves the central nervous system integration of physical, chemical, and nervous inputs into the respiratory control centers.

Performance - Stress and Relaxation

The review of normal metabolism in rest, exercise, and recovery indicated that there are a number of physiological changes that occur with exercise and recovery as compared with resting levels. It is now necessary to relate the normal exercise and recovery responses to the changes that occur in performance as affected by stress and relaxation.

There is conflicting opinion as to whether the effects of low levels of stress increase or decrease motor performance. There is good evidence for both. Beam (1955) studied the effects of "real life stress" on serial learning and conditioning. He found that competitive stress facilitated conditioning although it hampered serial learning. He arrived at this conclusion when he found that the stressed subjects made significantly more conditioned responses than the control group but they also made more errors. This indicated that competitive stress inhibited learning.

In a task consisting of hitting a ball with simple arm movement Howell used electrical shock as a stress motivator. The results demonstrated that stress of this nature increases the speed of motor performance when comparisons are made with control subjects. Howell (1953) suggested that performance of simple motor tasks is facilitated by motivational stress.

Another investigation of speed stress verified the results of Howell and Beam. The effects of stress were studied on a paired associate motor learning task in which pre-experimentally acquired positions were arbitrarily designed as correct or incorrect. The stress group made significantly more responses while the nonstress group had better results (Castanda 1965). Again the implication was made that motor performance can be positively or negatively affected by stress depending upon the difficulty of the task and the degree of learning involved.

Further evidence has suggested that subjects who score high on anxiety inventories exhibit impaired performance in complex learning, problem solving, and motor performance tasks (Wilson 1970, Carron 1968,

Spielberger 1966, Denny 1965, Harleston 1965, Ryan 1962, Martin 1961 and Spence 1958). Wilson (1970) commented that this decrease in performance is particularly true if the tests are administered under stressful conditions.

From the review of literature concerning motor performance and learning it may be concluded that stress decreases or increases performance depending on the difficulty of the task involved and the anxiety state of the subjects. Ryan (1962) explained the situation this way: "In simple tasks stress may facilitate performance while in gross motor skills and difficult tasks involving learning, performance may be impaired."

The effects of stress on motor performance have been studied in some detail but this is not true of physiological performance. Physiological evidence reported by Pitts and others indicates that there is excessive lactate production with standard exercise in patients with anxiety neurosis (Pitts 1967, Linko 1950, and Holmgren 1959). Pitts also found that the rise in concentration of lactate per second of maximal work was significantly greater in these anxiety patients. He theorized that the mechanism for increased lactate production could be explained as follows:

> . . . that anxiety symptoms may have a common determining biochemical end mechanism involving the complexing of ionized calcium at the surface of excitable membranes (in the interstitial fluid) by the lactate ion produced intercellularly.

He adds that:

> There may be something specific about the lactate ion in producing the naturally occurring hypocalcemic anxiety symptoms.
>
> (Pitts 1967)

The validity of these hypotheses has yet to be examined in further investigation.

The literature is even more limited concerning the effects of psychological stress on carido respiratory response to exercise. One study made on a patient with severe anxiety showed a doubling of $\dot{V}_E$ as the utilization coefficient of oxygen decreased by half therefore resulting in no net change in $\dot{V}o_2$. This study was done by comparing the responses of a standard two step exercise test in a normal and anxiety induced states (Wolf 1946). Contrary to these findings Jones showed a significant increase in $\dot{V}o_2$ in anxiety patients (Jones 1946). In a study of the response to exercise using twenty subjects Jones reported that anxious subjects manifested greater oxygen uptakes for standardized work than normal controls. He also showed that heart rate and $\dot{V}_E$ increased significantly with stress during standardized work (Jones 1946).

In light of this scanty evidence, it seems unclear what the exercise response is under conditions of psyhological stress, although the work of Jones might indicate that excessive anxiety inhibits normal performance.

There have been several investigations of the effects of relaxation training upon selected motor performance tasks but again very little work has been done on the effects of relaxation upon physiological responses to exercise.

Haverland studied the influence of relaxation on some aspects of motor performance, i.e. coordination, steadiness and reaction time. He found that subjects showed faster or improved reaction time, greater

accuracy, and greater steadiness of movement when using Jacobson's progressive relaxation training prior to testing (Haverland 1953, Jacobson 1938). These findings are in accordance with those of, Shaw 1971, Blasdell 1971, and Abrama 1972. These researchers reported that transcendental meditation decreases reaction time and improves coordination, flexibility, and perceptual and learning ability.

Relaxed subjects with "subnormal drive conditions", or abnormally low levels of arousal also tended to give more complete responses in a discriminant learning task. This result was reported in two studies done by Nakamura (1965A, 1965B) involving 52 and 40 experimental subjects half of which were given training in progressive relaxation (Jacobson 1938).

Evans (1954) electromyographically measured the level of muscular tension or stress in college women playing basketball. The experimental subjects were given a course in relaxation training and later in a game situation were compared with normal controls. It was noted that the relaxed subjects had lower levels of tension or stress which according to Evans suggested the value of relaxation training as a tension-reducer in athletic competition. These results were verified by Lyons (1967) who conducted virtually the same study.

In summary:

1. Stress can impair or improve motor performance and learning depending upon the nature of the stress and the complexity of the task.
2. Due to the limited amount of research dealing with stress and physiological performance, the effects of psychological

stress on exercise and recovery is open to question. It does seem that anxiety patients demonstrate impaired exercise performance.

3. Relaxation techniques do appear to enhance the learning and performance of motor tasks while the effects of relaxation on exercise and recovery performance remains to be examined.

CHAPTER III

METHOD OF RESEARCH

Introduction

The methodology is presented under the following headings: Subjects, Laboratory Method and Equipment, Test Protocol, Calculation, Analysis and Processing of Data, and Research Design and Statistical Analysis.

Subjects

The selection of twelve (12) volunteer subjects was fixed. All subjects were untrained, male, and between the ages of 18 and 25 years ($\bar{x}$ = 21.3). Anthropometric data is shown in Table 4. The subjects were trained in the technique of transcendental meditation as stipulated by the International Meditation Society and were practicing meditation on a regular daily basis. The subjects' experience in meditation ranged from 8 to 35 months ($\bar{x}$ = 23). One subject did not want to continue in the experiment after the first test session and was substituted by another to maintain the experimental N at 12.

Laboratory Method and Equipment

The respiratory variables, $\dot{V}o_2$, $\dot{V}_E$, $\dot{V}_Eco_2$, and respiratory rate, were measured by the modified open circuit method (Consolazio 1963) which is schematically represented in Figure 2. The open circuit method for the determination of $\dot{V}o_2$ and other respiratory variables is a modification of the Douglas Bag method for gas collection. As shown in

TABLE 4 - ANTHROPOMETRIC DATA FOR EXPERIMENTAL SUBJECTS

Subjects	Age (years)	Weight (pounds)	Height (ft.-inches)	Experience in Meditation (months)
R.V.S.	25	151	5-10	8
P.M.	20	144	5-9	33
D.B.	21	164	5-9	16
C.G.	23	190	5-11	23
D.Y.	19	165	6-1	25
R.G.	18	130	5-7	30
C.P.	18	172	6-2	26
P.P.	22	146	6-0	32
M.R.	23	168	6-0	9
P.B.	22	170	6-0	27
G.L.	20	146	6-3	12
R.B.	24	126	5-6	35
Means →	21.3	156	5-10.8	23

KEY TO FIGURE 2 SCHEMIC DIAGRAM OF EQUIPMENT

A.	Treadmill Control Amplifier Model No. 642	Quinton Ltd., Seatle, Wash.
B.	Programmed Exercise Control Amplifier Model No. 642	" "
C.	Exercise Cardiotachometer Model No. 609	" "
D.	ECG Isolation Amplifier Model No. 620	" "
E.	Treadmill	" "
F.	ECG Preamplifier Model No. 607	" "
G.	Monoghan Neoprene Cushion Oxygen mask	Monoghan Co., Denver, Colorado
H.	Triple J value	Warren E. Collins Inc.
I.	Mixing box	" "
J.	Rampox O_2 Analyser	N.V. Godart Co., Holland
K.	Capnograph Co_2 Analyser	" "
L.	Grass 5D Polygraph	Grass Instr. Co.
LL.	AC/DC Psycogalvanometer	Lafayette Instr. Co., Lafayette, Indiana
M.	Standard Hospital Bed	
N.	Larger Tissot Gasometer	Warren E. Collins Inc.
O.	Gas Collection Valve	" "
P.	Potentiometer	" "
Q.	Two way shut-off Valve	" "
R.	Small Tissot Gasometer	" "
S.	DC/AC Coupler Type 7170	Narco Bio Systems Inc., Houston, Texas
T.	DC/AC Coupler Type 7170	" "
U.	High Gain Coupler Type 7170	" "
V.	Servo Writer	" "
W.	Four B Polygraph	" "
X.	Event Marker	" "
Y.	Foot Switch for the Event Marker	" "
Z.	Switch for Solinoid Valve	" "

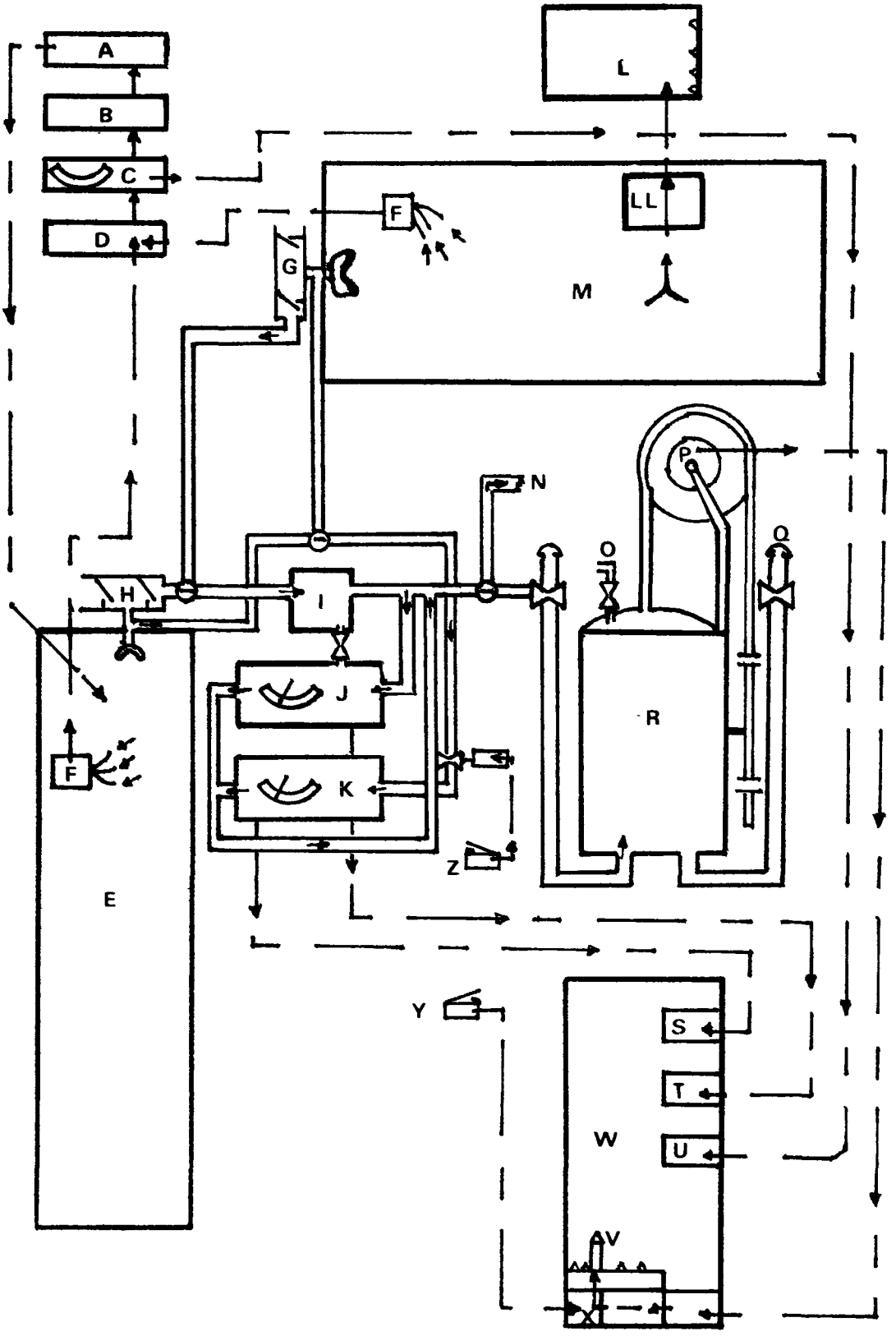

Figure 2 Schematic Diagram of the Equipment

Figure 2 expired oxygen and carbon dioxide were analysed by the Rapox and Capnograph gas analysers respectively. Minute ventilation was calculated from the collection of expired gas in a large and small Tissot Gasometer while respiratory rate was measured by a potentiometer attached to the indicator mechanism of the Gasometer. The output for these four variables, $\dot{V}o_2$, $\dot{V}_E co_2$, $\dot{V}_E$ and respiratory rate was then coupled to separate channels of the Narco Bio Systems Four B Polygraph which gave separate recordings for each variable.

Heart rate was measured via an electrocardiogram (ECG). A Quinton ECG Isolation Amplifier was also coupled to the Four B Polygraph to give continuous heart rate recordings.

Galvanic skin resistance was measured with a Lafayette AC/DC Psychogalvanometer on a DC mode which when coupled to a Grass 5D Polygraph gave a continuous GSR recording.

Venous blood samples for lactate were taken from the brachial vein via a no. 20-gl Becton, Dickinson & Co. Vacutainer seringe. All blood samples were taken by a qualified laboratory technician or the researcher who qualified himself for taking blood through a medical physician. The procedures for taking blood was followed after the method of Becton, Dickinson & Co.

Treadmill exercise was chosen in this experiment for two reasons: 1. A number of investigators have found that greater values for $\dot{V}o_2$ can be shown in standard treadmill exercise tests (Astrand 1971, Kasch 1966, and Sheppard 1966). 2. The treadmill could be programmed to control exercise in relation to heart rate.

Exercise was performed on a Quinton treadmill which was programmed to increase and then vary the speed according to the subject's heart rate. In this way the exercise heart rate was elevated and then maintained at 165-170 beats per minutes. Exercise began at 3.5 miles per hour with 8.6 percent grade which was constant throughout exercise. The Programmed Exercise Control Amplifier (Fig. 2) was set at a constant heart rate acceleration, treadmill acceleration, and a maximum heart rate of 170 beats per minute. Treadmill exercise was therefore standard for each subject in each experimental testing session. The exercise heart rate was set at 170 beats per minute according to the findings of Astrand. Astrand (1971) has shown that at work loads requiring a heart rate over 170 b.p.m. Vo_2 tends to increase relatively more than the heart rate.

The following laboratory procedure for the preparation of the experimental subjects was applicable to all testing sessions. As previously mentioned an attempt was made to test each subject within a period of two weeks to minimize the duration of time between tests. The subjects arrived at the laboratory in a post-absorptive state and were instructed to sit comfortably on a standard hospital bed. ECG chest leads were fixed to the chest so that heart rate could be monitored (Buyton 1971, Goldman 1970). Silver silver GSR electrodes were placed on the middle and little fingers of the subjects' right hands so that GSR could also be monitored. The subjects then began breathing on the respiratory apparatus (Fig. 2) in order to become acclimatized to the respiratory equipment, while final calibration of the Psychogalvonmeter and Rapox and Capnograph gas analysers was made.

Test Protocol

Testing consisted of three separate sessions each of which was conducted on a different day. These sessions were: 1. control for meditation, 2. control for exercise and recovery, and 3. treatment for exercise and recovery. The variables that were measured in each test session and the time of collection are presented in Table 5. The protocol for each experimental test session is outlined below.

Session 1. Control for Meditation

This session was designed to acquaint the subjects with laboratory procedures and to act as a control for observations on resting metabolism and metabolism associated with transcendental meditation.

(A) The subjects rested for a minimum of fifteen minutes prior to meditation and the physiological variables except blood lactate were measured continuously in the last 5 minutes of rest. Blood lactate was taken in the minute following rest.

(B) The subjects then meditated for twenty minutes and the same dependent variables were monitored continuously. The blood sample was taken in the minute following this meditation.

(C) All subjects were then instructed in the proper technique of getting on and off the treadmill which acquainted with treadmill exercise. It was thought that this instruction would relieve anxiety caused by unfamiliarity with this piece of equipment. Anxiety could have affected the subjects' exercise performances or meditations in a later test session (The total time for this session was approximately one hour).

TABLE 5 - MEASURED PHYSIOLOGICAL VARIABLES AND TIME OF COLLECTION FOR TEST SESSIONS 1-3

Variable	Session No. 1 A. Rest B. Meditation	Session No. 2 A. Rest B. Exercise C. Recovery	Session No. 3 A. Rest B. Meditation C. Exercise D. Recovery + (TM)
$\dot{V}O_2$ $\dot{V}_E CO_2$ $\dot{V}_E$ f	Measured continuously for 5 minutes of rest and 20 minutes of meditation.	Measured for 5 minutes of rest, the last minute of exercise and continuously for 20 minutes of recovery.	Measured continuously for 5 minutes of rest, 20 minutes of meditation, the last minute of exercise and for 20 minutes of recovery.
H.R.	Measured continuously for all test conditions		
Blood lactate	Measured 1 minute after rest and 1 minute after meditation.	Measured 1 minute after rest and recovery and 5 minutes after exercise.	Measured 1 minute after rest, meditation, and recovery and 5 minutes after exercise.
G.S.R.	Measured continuously for rest and meditation.	Not measured.	Measured continuously for rest and meditation.

Session 2. Control for Exercise and Recovery

This session acted as a control for exercise and recovery and was used as a comparison for the treatment of session 3.

(A) The subjects rested for a minimum of 15 minutes as in session 1A, (one) and the same variables were measured at the same time with the exception of GSR which was not measured.

(B) The experimental subjects then exercised for five minutes at a work load that maintained their heart rates between 165-170 beats per minute. The physiological variables were measured in the last minute of exercise and a blood sample was taken 5 minutes post exercise.

(C) The same variables were again measured continuously for a twenty minute recovery period following exercise and a blood sample was taken during the minute after recovery (The total time for this session was approximately one hour).

Session 3. Treatment for Exercise and Recovery

This session was designed to be the treatment for exercise and recovery and it combined testing sessions one and two (i.e. rest, meditation, exercise, and recovery with meditation). The meditation during recovery began 6 minutes post exercise after the exercise blood sample was taken. The dependent variables (Table 5) were measured at the same time as was done in the controls for meditation, exercise and recovery (The total time for this session was approximately one hour and a half).

Calculation, Analysis, and Processing of Data

Calculation of Data

The respiratory variables of $\dot{V}o_2$, $\dot{V}_E co_2$, and $\dot{V}_E$ were corrected for standard pressure and temperature and calculated with an automatic program on a Wang 6,000 calculator. This program was developed in the Exercise Laboratory, Department of Kinathropology, at the University of Ottawa using a modified method described by Consolazio (1963). An example of this method is shown in Appendix 1A.

The data for heart rate, respiratory rate, and galvanic skin response was taken directly from the respective polygraph recordings as this data represented actual values.

Blood Analysis

The venous blood samples were deprotenized immediately after collection according to the Biochemica Test Combination, Boehringer Mannheim GmbH, Mannheim, Germany (see Appendix B). The deprotenized blood was then frozen for subsequent analysis.

Blood samples were enzymatically assayed for lactate using the lactate kit, cat. no. 15972TLAA, Biochemica Test Combination (Appendix B), and analysed spectrophotometrically with a Precision Spectrophotometer, model no. 505, Bausch & Lomb Co., Rochester, New York (Bergmeyer 1962, and Laudahn 1959).

Processing of Data

In every test session the data collected for blood lactate was immediately ready for statistical analysis.

The data for GSR for each subject was taken from absolute values at the end of rest and meditation. This data was also immediately ready for statistical analysis. Mean values for five minutes of resting data were calculated for the following variables: $\dot{V}o_2$, $\dot{V}_E co_2$, $\dot{V}_E$, HR and respiratory rate. In the two meditating conditions control and treatment means were calculated for each of the variables measured. These mean values were calculated on the lowest metabolic steady state response of a three minute duration during the twenty minutes of meditation. These three minutes were considered to be a steady state condition when the values for $\dot{V}o_2$, $\dot{V}co_2$ and $\dot{V}_E$ varied less than ten per cent.

Recovery data was calculated for each subject for the first five minutes and for every second minute in the last fifteen minutes of recovery (eg. 1,2,3,4,5,7,9,11---19th min.). Means were then calculated for each of these minutes. This was done for the individual subject's data for each of the five variables. Subsequently, means were calculated for each subject and each variable for the seventh, ninth, eleventh, ---, and nineteenth minutes of recovery. This represented the meditation portion of recovery.

Research Design and Statistical Analysis

The research design was crossed, meaning that all of the dependent variables with the exception of GSR (Fig. 3) were measured in each of the testing sessions (Keith 1972, Scott 1965, and Weber 1970). Repeated measures were also used with each of the subjects acting as his own control. There were seven dependent variables, twelve replications,

two treatments and four conditions. Half of the subjects were randomly assigned to be tested for treatment for exercise first while the other half were tested for control for exercise first. This meant that the design "blocked" or cancelled any learning effect that might have occurred due to one of the treatments (Campbell 1963).

As mentioned in the limitations it was not possible to statistically analyse the data with the ideal method (i.e. Multivariate Analysis of Variance with repeated measures).

Twenty six multiple t tests for correlated samples were computed with the use of an IBM, APL computer terminal, CAN program - "Two Groups", Faculty of Education, University of Ottawa. An example of this statistical procedure is given in Appendix 2A.

When multiple t tests are used for statistical analysis, the t tests cannot be regarded as independent and the various tests themselves refer to redundant, overlapping aspects of the data (Hays 1963, Keith 1972 and Scott 1965). Also, when multiple t tests are used the probability of committing a type I error or rejecting the null hypothesis incorrectly is increased (Keith 1972). Instead of using the significance level $P < 0.05$ it was determined that the alpha level would be set a $P < 0.01$. In this way the probability of committing a type I error was decreased (Weber 1970 and Scott 1965).

Simple comparisions were made on all the dependent variables between the control and the treatment data. These comparisions were made on the four conditions of rest, meditation, exercise and recovery to bring the total number of t tests up to twenty-six. No attempt was made to compare data within treatments as it was assumed that there

would be significant difference between resting, exercise, and recovery values (See Table 1, Astrand 1970, Keele 1965).

CHAPTER IV

RESULTS

Introduction

This chapter presents the results under three major headings: Rest and Meditation, Exercise, and Recovery. Group data is presented in tabular and graphical forms which show means, standard deviations and t values. Two levels of statistical significance are reported and $P < 0.01$ is the accepted level because multiple t tests were used.

Tables 6 and 7 along with Figure 3 serve as illustrations of the data collected on a single subject. Complete data collected on the twelve subjects may be seen in Tables C1, C2, C3 and C4 (Appendix C).

Rest and Meditation

Group data for rest and meditation is shown in Table 8 and Figures 4 through 9. In the control for meditation, test session one, there was a significant difference found in $\dot{V}_E$, $\dot{V}o_2$, $\dot{V}_E co_2$ and GSR when a comparision was made between rest and meditation. In test session three, treatment for exercise and recovery, there was a significant difference in $\dot{V}_E$ and GSR when a similar comparison was made between rest and meditation.

Exercise

The group data for exercise in test sessions two and three is shown in Table 9 and Figures 10 through 15. There were no significant differences found in any of the dependent variables when control and

TABLE 6 - COMPLETE DATA FOR REST, MEDITATION, AND EXERCISE FOR A SINGLE SUBJECT

Variables	Session No. One		Session No. Two		Session No. Three		
	Rest	Meditation	Rest	Exercise	Rest	Meditation	Exercise
Blood lactate mg%	11.3	10.2	14.6	31.8	10.2	9.4	38.1
Heart rate (b.p.m.)	82.4	78.6	83.6	167	72.2	65.7	167
Respiratory rate (breaths/min.)	16.2	15.3	16.0	31	15.4	14.3	32
Minute ventilation l./min.	9.630	8.463	10.149	75.158	8.766	7.915	76.633
Oxygen consumption ml./min.	282	246	292	2530	259	230	2498
Carbon dioxide elimination (ml./min.)	208	182	266	2780	203	178	2414
Galvanic skin resistance (kilohms)	135	275	NM	NM	120	245	NM

NM = Not measured

TABLE 7 - COMPLETE RECOVERY DATA FOR A SINGLE SUBJECT

Variables	1	2	3	4	5	7	9	11	13	15	17	19
	← TIME OF RECOVERY IN MINUTES →											
Blood lactate (mg.%)	—	—	—	—	31.4[1] 38.1[3]	—	—	—	—	—	—	19.0 17.8
Heart rate (b.p.m.)	136 126	99 92	86 77	89 83	88 76	83 74	88 74	88 75	90 75	89 74	85 73	90 76
Respiratory rate (breaths/min.)	28 28	26 25	21 22	17 21	21 22	16 20	18 19	18 19	15 19	16 17	16 18	17 17
Minute (l./min.) ventilation	47.8 36.3	25.4 35.6	15.6 20.0	16.0 17.6	16.5 16.7	12.5 15.2	12.0 11.5	12.0 10.0	10.0 10.3	9.4 10.0	10.4 9.4	10.6 9.0
Oxygen (l./min.) consumption	1659 1211	892 1151	432 577	445 491	446 412	306 381	317 325	336 287	275 310	277 279	327 273	325 244
Carbon dioxide elimination (l./min.)	1818 1125	913 1157	485 618	478 528	480 468	324 365	281 374	315 219	258 227	231 205	270 197	260 178

[1] Top value is the control recovery [3] Bottom value is recovery with meditation

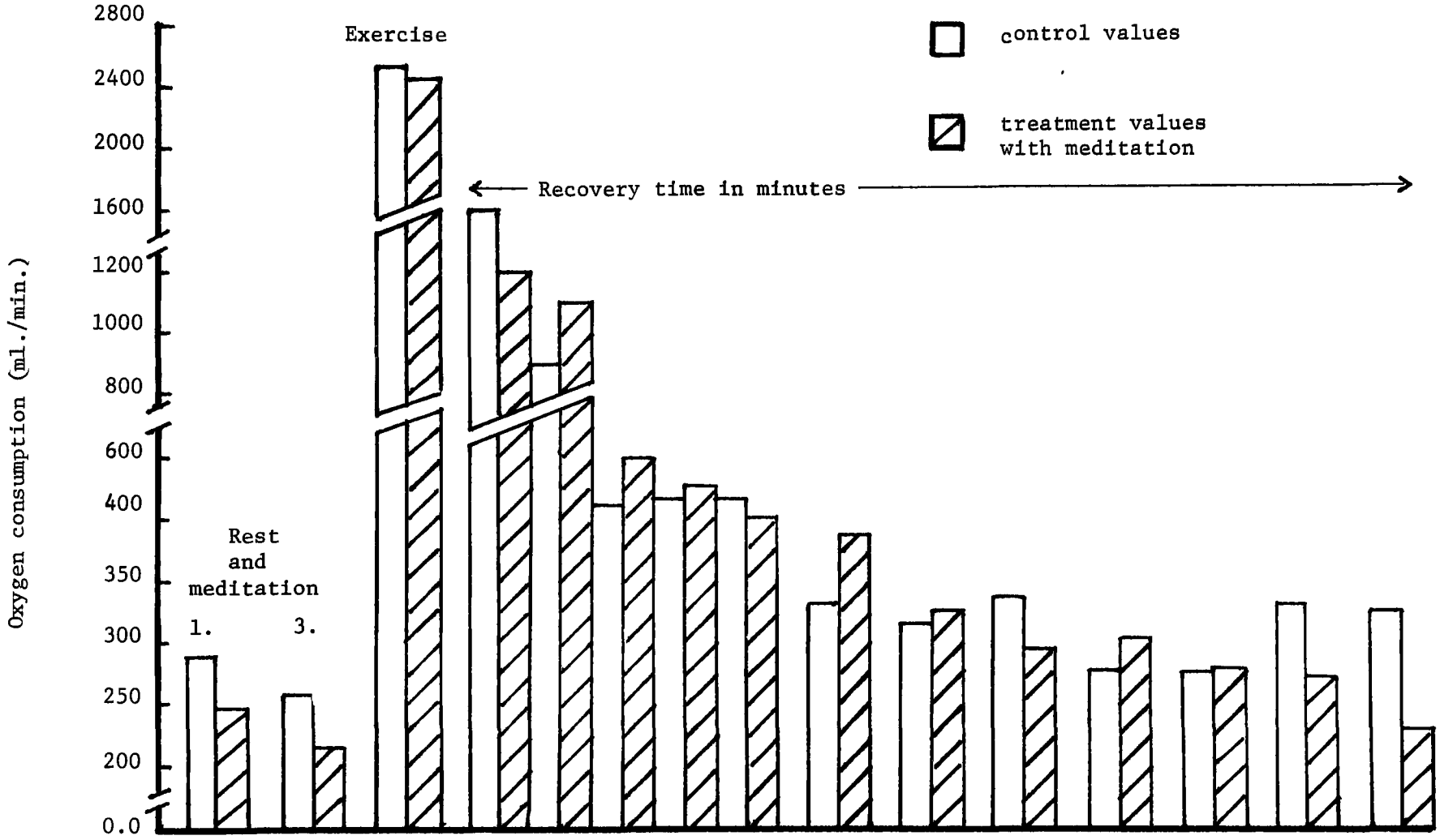

Figure 3 Complete Data for Oxygen Consumption on a Single Subject

TABLE 8 - GROUP MEANS, STANDARD DEVIATIONS, AND t VALUES FOR REST AND MEDITATION

Variables	Session Number One			Session Number Three		
	Rest	Meditation	t Value	Rest	Meditation	t Value
Blood lactate (mg.%)	10.8 ±3.1SD	9.3 ±3.0	2.437 *	10.0 ±3.1	9.4 ±2.8	0.789
Heart rate (b.p.m.)	76.8 ±7.1	74.4 ±7.8	1.893	71.0 ±5.8	69.2 ±5.8	0.991
Respiratory rate (breaths/min.)	12.5 ±2.6	11.2 ±2.9	1.663	11.6 ±2.9	11.3 ±3.2	0.254
Minute ventilation (l./min.)	8.159 ±1.066	6.750 ±0.917	5.206**	8.131 ±1.02	7.018 ±0.559	3.248**
Oxygen consumption (ml./min.)	270 ±45.9	235 ±40.4	4.132**	271 ±45.5	255 ±37.1	1.832
Carbon dioxide elimination (ml./min.)	218 ±30.7	179 ±27.8	5.294**	214 ±32.0	190 ±26.5	2.595*
Galvanic skin resistance (kilohms)	102 ± 25	210 ±29	-14.246**	111 ±26	215 ±24	-13.827**

P is the probability that the mean values for rest and meditation are equal.
Degrees of freedom = 11.

* $P < 0.05$ ** $P < 0.01$ SD = standard deviation

Figure 4

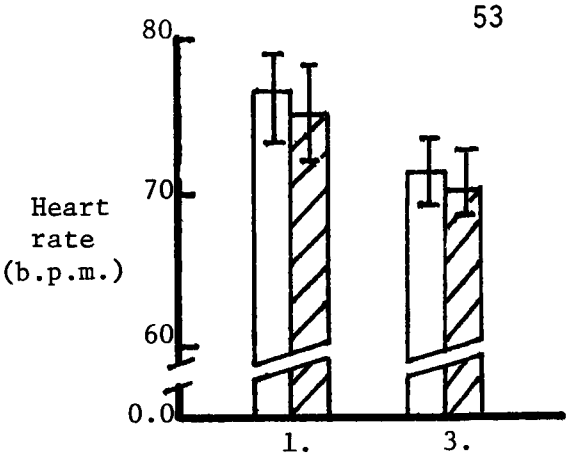

Figure 5

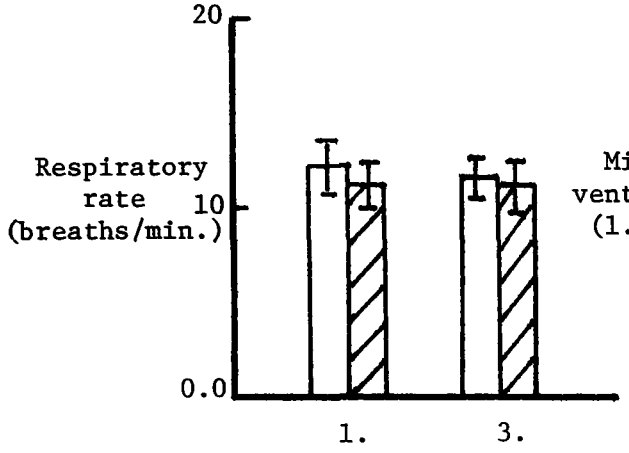

Figure 6

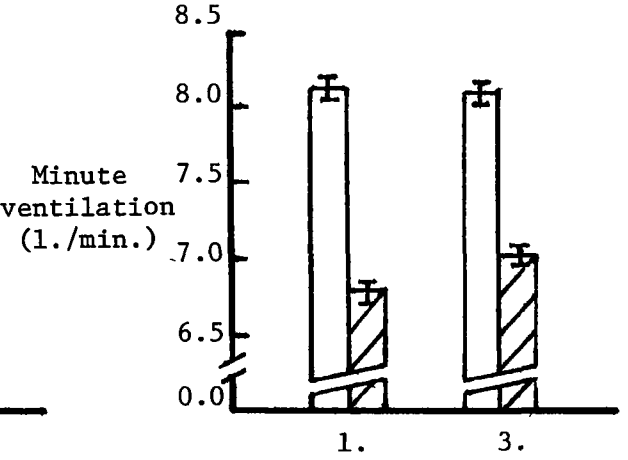

Figure 7

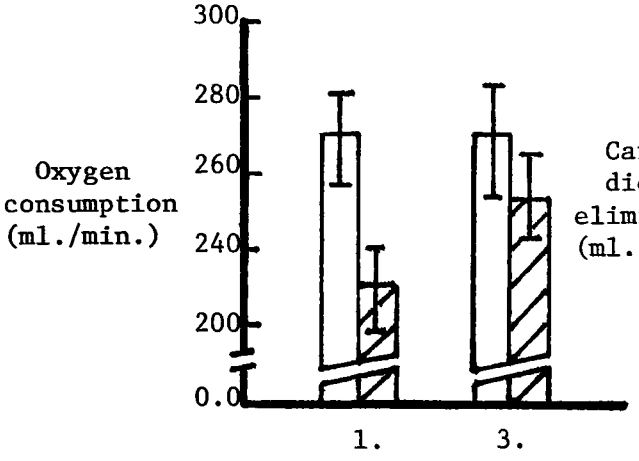

Figure 8

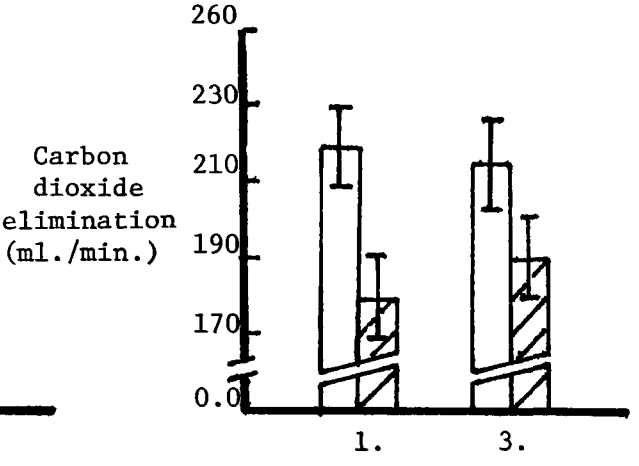

Figure 9

Figures 4-9 Means and Standard Deviations for Rest and Meditation in Control for Meditation and Treatment for Exercise and Recovery

TABLE 9 - GROUP MEANS, STANDARD DEVIATIONS, AND t VALUES FOR EXERCISE WITH AND WITHOUT MEDITATION

Variables	Session No. Two Control Exercise	Session No. Three Exercise and Meditation	t Value *
Blood lactate (mg.%)	32.2 ±10.3 SD	33.3 ±13.4	-0.392
Heart rate (b.p.m.)	168.7 ±3.2	168.1 ±3.0	1.205
Respiratory rate (breaths/min.)	25.6 ±3.6	24.6 ±3.8	1.980
Minute ventilation (l./min.)	61.7 ±8.3	62.5 ±9.3	-0.380
Oxygen consumption (l./min.)	2.660 ±0.402	2.583 ±0.355	0.802
Carbon dioxide elimination (l./min.)	2.470 ±0.365	2.417 ±0.276	0.718

* The t values required for significance at the 0.05 and 0.01 levels, with 11 degrees of freedom, were 2.201 and 3.106 respectively.

SD = standard deviation

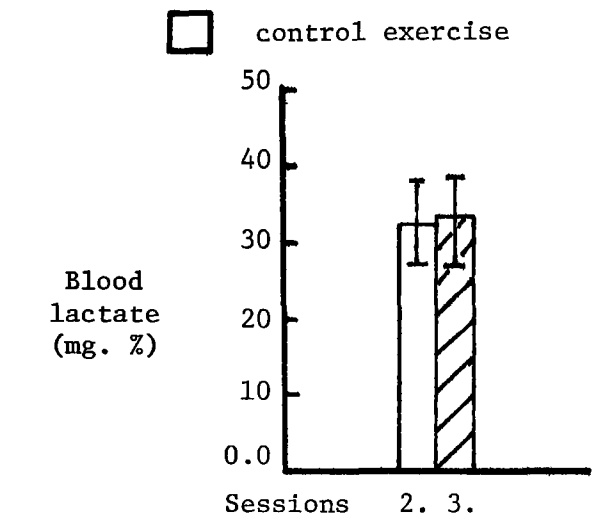

Figure 10

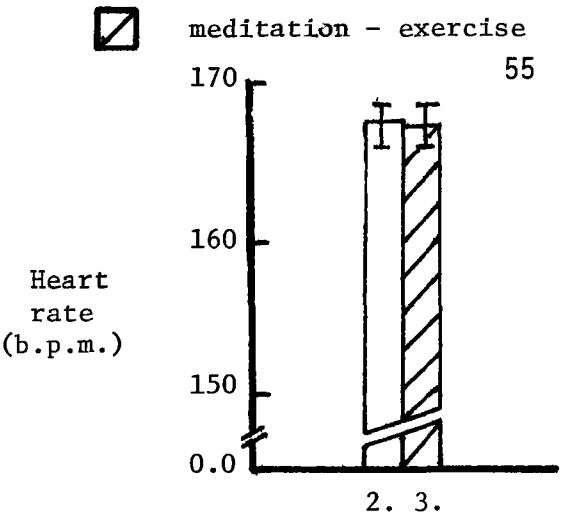

Figure 11

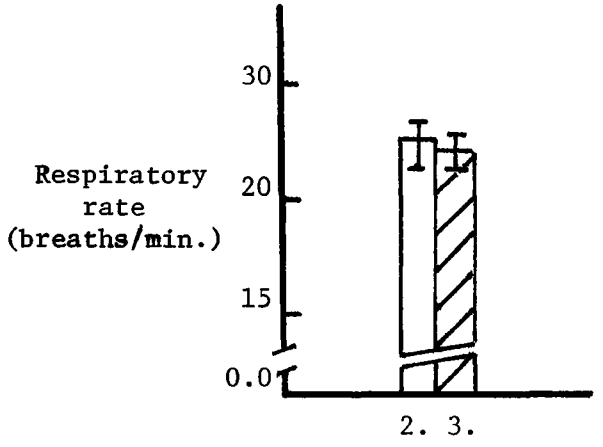

Figure 12

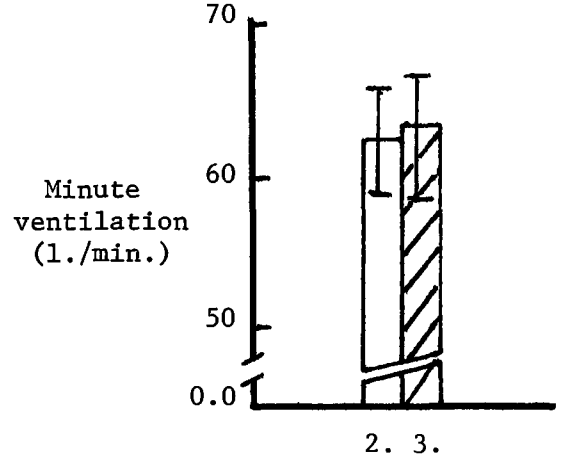

Figure 13

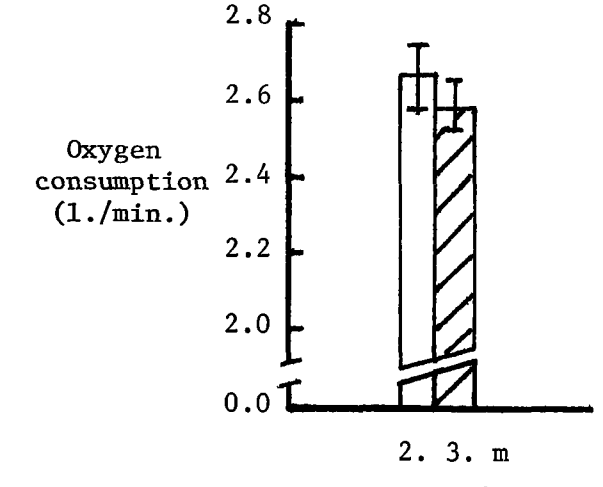

Figure 14

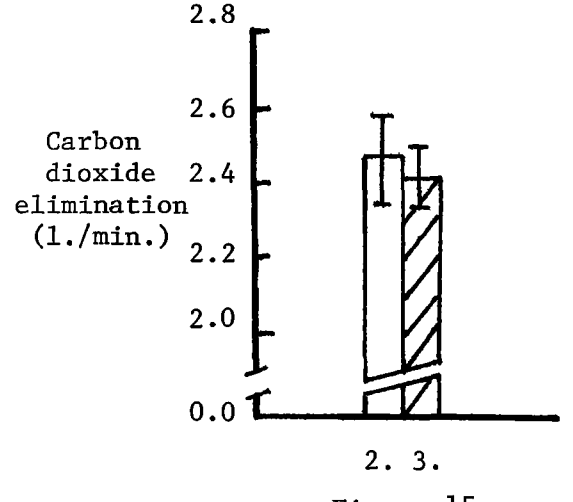

Figure 15

Figures 10-15 Means and Standard Deviations in Control and Treatment Sessions for Exercise

treatment sessions for exercise were compared.

Recovery

The group data for recovery is graphically shown in Figures 16 through 21. The data for the last twelve minutes of recovery is reported in Table 10. In this meditating portion of recovery there was a significant difference in $\dot{V}_E$ (Table 10) when a comparison was made between the control and treatment sessions.

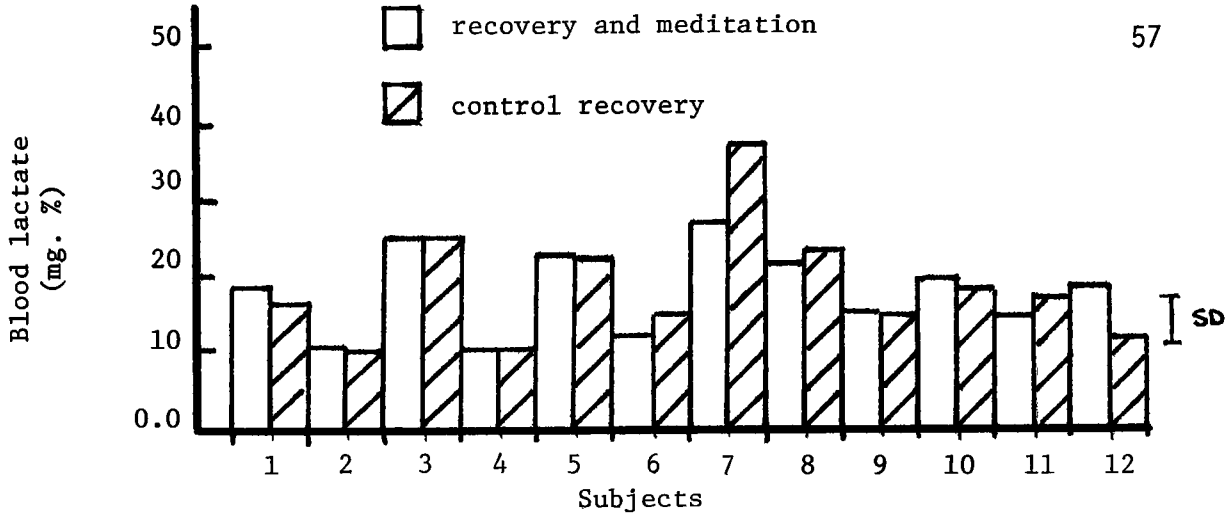

Figure 16 Recovery Blood Lactate, mg. %

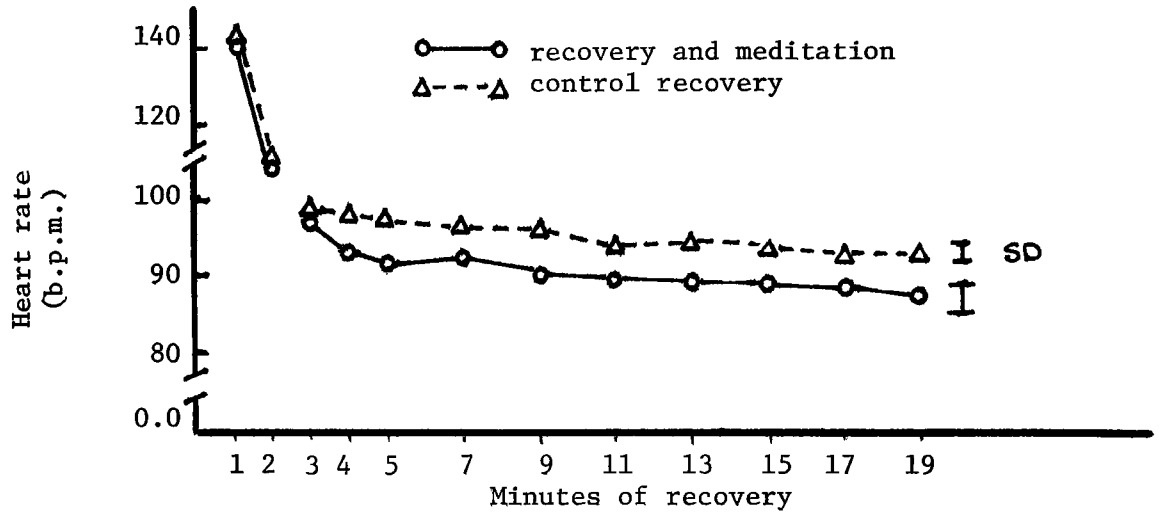

Figure 17 Recovery Heart Rate, Beats/min.

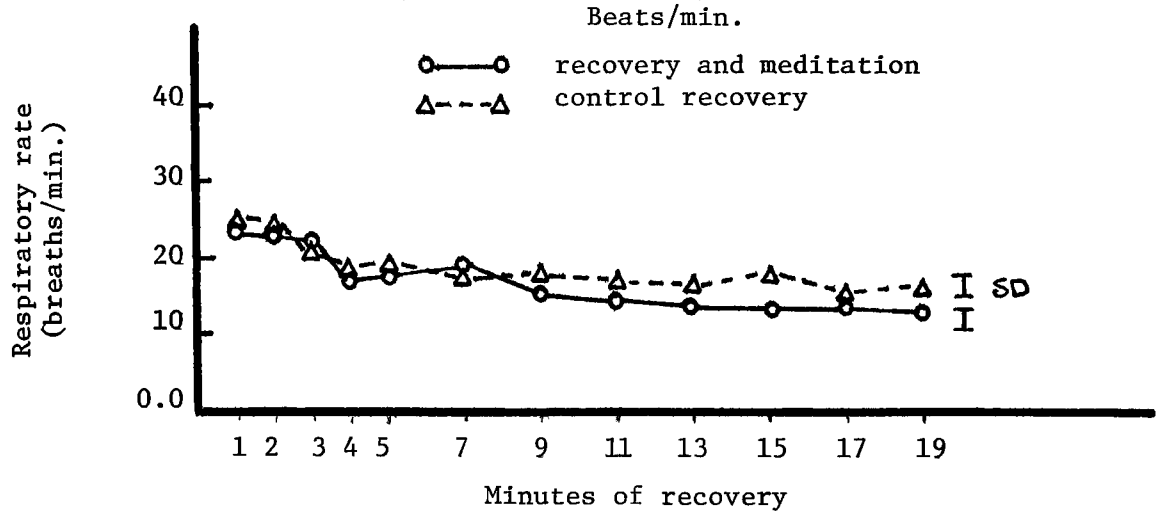

Figure 18 Recovery Respiratory Rate, Breaths/min.

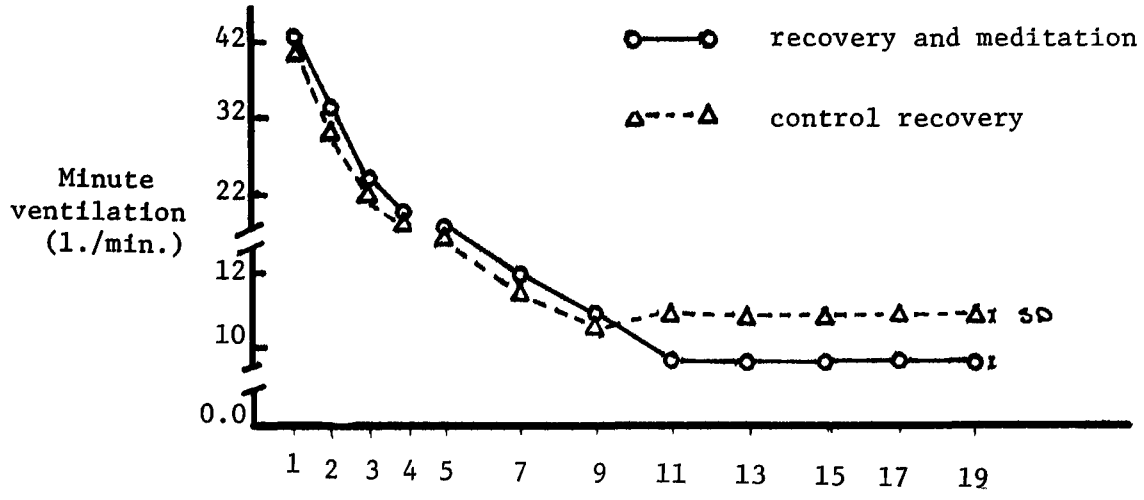

Figure 19 Recovery Minute Ventilation, l./min.

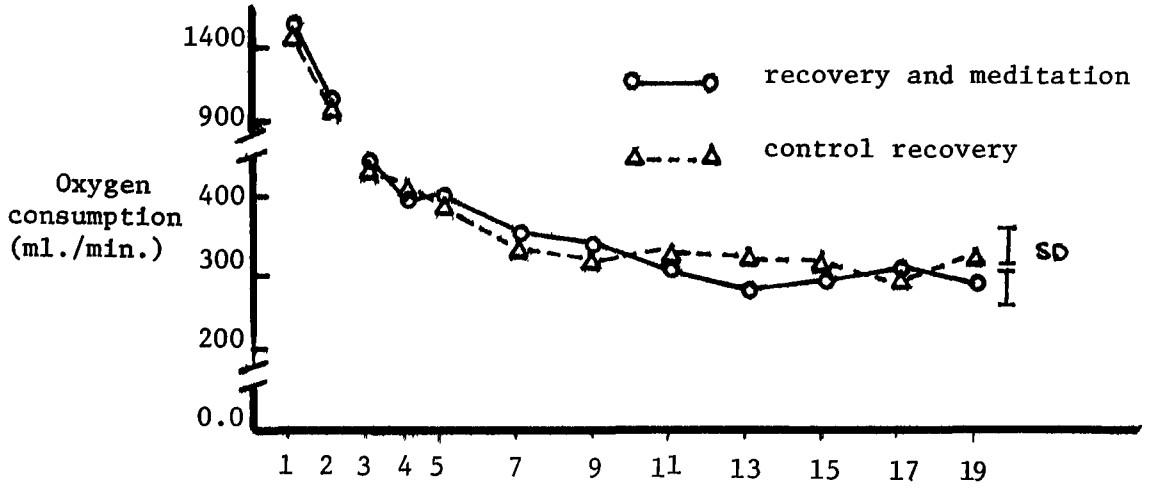

Figure 20 Recovery Oxygen Consumption, l./min.

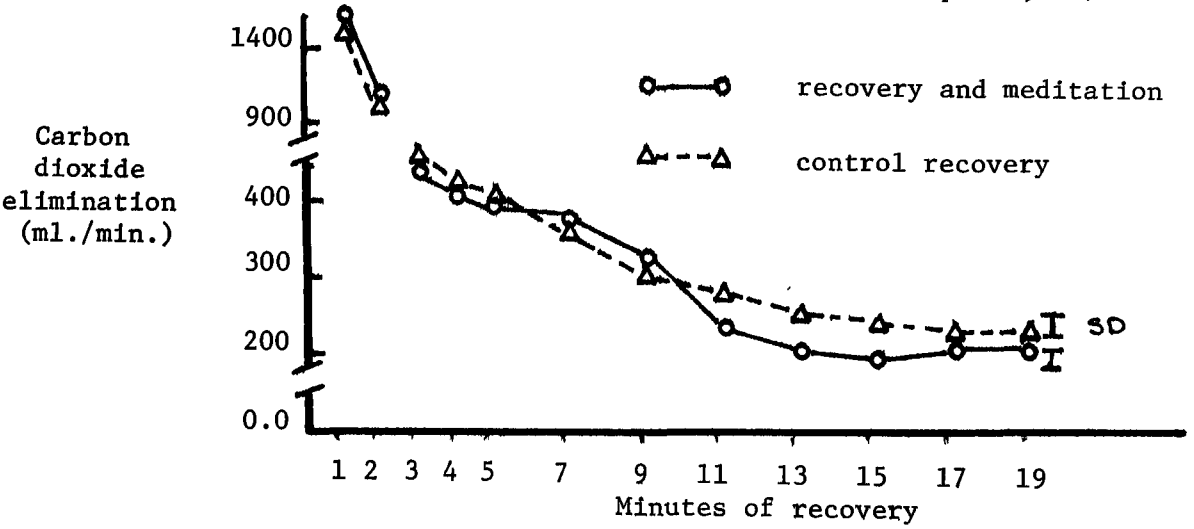

Figure 21 Recovery Carbon Dioxide Elimination, ml./min.

TABLE 10 - GROUP MEANS, STANDARD DEVIATIONS, AND t VALUES FOR THE LAST TWELVE MINUTES OF RECOVERY WITH AND WITHOUT MEDITATION

Variables	Session No. Two Control Recovery	Session No. Three Recovery with Meditation	t Value
Blood lactate (mg.%)	17.7 ±5.4 SD	18.6 ±7.1	-0.875
Heart rate (b.p.m.)	91.5 ±3.6	86.9 ±6.4	2.928*
Respiratory rate (breaths/min.)	15.3 ±4.3	14.6 ±3.8	1.461
Minute ventilation (l./min.)	9.755 ±1.281	8.873 ±1.059	3.140**
Oxygen consumption (ml./min.)	365 ±63	359 ±54	0.347
Carbon dioxide elimination (ml./min.)	250 ±36	234 ±36	2.509*

P is the probability that the mean values for recovery with and without meditation are equal.
Degrees of freedom = 11.

* $P < 0.05$ ** $P < 0.01$ SD = standard deviation

CHAPTER V

DISCUSSION

Introduction

The discussion of the results is presented under the major headings that were presented in the previous chapter: Rest and Meditation, Exercise, and Recovery. An explanation of the results and the mechanisms involved with the changes that were observed, is discussed in conjunction with related literature.

Rest and Meditation

The findings of the present investigation during the control for meditation are in general agreement with those of Wallace (1971) and others. The results that were observed during rest and meditation in the treatment condition for exercise and recovery are not as clear and shall be discussed in the later half of this section.

Control for Meditation (Subproblem 1)

The greatest physiological change shown in the present study was the increase in GSR (Table 8). This change during meditation indicates a low state of arousal or a reduced level of stress. Comparable results have been reported by Benson (1974), Orme Johnson (1973), Wallace (1972, 1971, 1970), Wenger (1961), and Bagchi (1957).

Significant ($P < 0.01$) decreases in $\dot{V}_E$, $\dot{V}o_2$ and $\dot{V}_E co_2$ were found during meditation (Table 8) which was in accordance with other investigators (Wallace 1972, 1971, 1970, Wenger 1961, and Bagchi 1957).

Wallace (1971) did not report as significant a change in $\dot{V}_E$ as was found in the present study but this was probably due to the small experimental sample he tested (N = 4). It might be expected that the same magnitude of change in $\dot{V}o_2$, $\dot{V}_Eco_2$ and $\dot{V}_E$ would occur with relaxation because these variables are under the same controlling mechanisms.

Although there was no significant decrease in blood lactate found in this study a definite trend ($P < 0.05$) is indicated in Table 8. There are several plausible explanations for the difference in the results reported in this investigation and those of Wallace (1971).

The subjects in Wallace's study meditated twenty to thirty minutes and then rested another twenty minutes post meditation. He reported that blood lactate continued to decrease for ten minutes following meditation (Wallace 1971). This suggests that blood lactate may drop as a function of time spent in meditation. The subjects who participated in this experiment only meditated for twenty minutes which represented the normal length of time of a daily meditation (Marharishi 1966). It might be therefore suggested that the subjects in this investigation did not produce the same decrease in blood lactate concentrations because they did not meditate as long as Wallace's subjects.

Secondly Wallace (1971) took arterial blood samples for lactate determinations while venous blood samples were taken in this experiment. Forester (1972) has demonstrated that slightly lower levels of lactate are found when arterial blood samples are taken. He also reported an increased lability of arterial lactate as compared to venous lactate, depending on the anatomical site from which the blood sample is taken. This difference in sampling method offers another explanation for the

discrepancy between the results found in this study and those of Wallace (1971).

It has been reported that significant decreases in respiratory rate accompany the meditating state (Wallace 1970, 1971, Allison 1970, Rao 1967, Wenger 1961, Bagchi 1957). Allison (1970) reported that the respiratory rate fell from a resting average of thirteen breaths per minute to six and later four breaths per minute during meditation. He stated his reason for using a thermistor for the measurement of respiratory rate as follows:

> . . ., it was necessary to use a method that would distract the attention as little as possible from the mental practice. Masks and flow meters proved too intrusive, and also presented another serious difficulty: when the tidal volume becomes very small, the small increase in dead space involved in breathing through a mask and flow meter becomes proportionately more significant.
>
> (Allison 1970)

A face mask was used in the present study because $\dot{V}o_2$, $\dot{V}_E co_2$ and $\dot{V}_E$ were measured. Hence, it is proposed that the non-significant decrease in respiratory rate (Table 8) reflected the imposition of this mask and the open circuit used for the collection of expired gas.

The slight decrease in heart rate of 2.4 beats per minute shown in Table 8 and the three beat per minute change found by Wallace (1971) are in accordance with Wallace's previous findings (1970). Wenger (1961) and Bagchi (1957) also reported that practitioners of yoga were capable of lowering their heart rates.

The physiological changes that occurred during the control for meditation support the hypothesis that the technqiue of transcendental meditation elecits a trophotoropic or relaxation response (Benson 1974,

Gellhorn 1972). These changes appear to be caused by a decreased sympathetic nervous system activity and are modulated by increased discharge from the parasympathetic nervous system (Benson 1974, Gellhorn 1972).

It might also be suggested that decreased sympathetic discharge found during relaxation could lower the level of circulating catecholamines (Selye 1956). Although this has not been studied in the case of transcendental meditation, lower concentrations of catecholamine may have a regulatory effect on the metabolism associated with meditation.

The chemoreceptors, respiratory control centers and vasomotor centers in the medulla may be directly affected by sympathetic discharge as well a circulating catecholamines (Sørensen 1971, Astrand 1970, Dejours 1964). Therefore it could be hypothesized that the hypometabolic cardio-respiratory response found in meditation is due to the nervous and hormonal effect upon these centers.

Galvanic skin response was likely affected in much the same way. Gellhorn stated, "The increase in skin resistance noted during meditation shows that in fact the 'ergotropic' tone is lessened: the sympathetic discharges to the sweat glands are diminished" (Gellhorn 1972).

Several explanations have been made for the reduction in blood lactate observed during meditation (Wallace 1971). Riechert (1967) reported a 300% increase in forearm muscle blood flow during meditation. Wallace explained that the decrease in blood lactate during meditation may be a result of this increase in muscle blood flow and increased aerobic metabolism. The work of Bolme (1969) and Whalen (1969) supports Wallace's hypothesis. Bolme and Whalen both reported the occurrence of hypoxia in

resting muscle. Bolme found that increased blood flow caused by induced vasodialation produce an elevation in oxygen uptake by the muscle.

The occurrence of hypoxia in resting muscle provides a satisfactory explanation of lactate formation and release in resting muscle. The increased blood flow with a consequent increased aerobic metabolism found during transcendental meditation provides a possible explanation for the reduced blood lactate concentration observed in this relaxed state.

The research of Hermansen (1972), Jorfeldt (1970), Karlsson (1971, 1972), and Rowell (1971) could provide yet another explanation for the decreased blood lactate concentrations reported during meditation. They have reported that with increased muscle blood flow during exercise and recovery, cardiac muscle, (Rowell 1971) skeletal muscle and the liver are capable of removing excess lactate from the blood and using it as a substrate. It is therefore conceivable that with the increased muscle blood flow found during meditation the decrease in blood lactate concentration is a result of an increased uptake of lactate by cardiac and skeletal muscle. This concept is certainly inadequately studied and therefore it is not possible to make any concluding statements until further investigation is carried out.

Rest and Meditation (Treatment for Exercise and Recovery)

The proceeding paragraphs give an explanation of the physiological changes shown in the control for meditation but they did not explain the difference in the results reported for the treatment session (Table 8).

Session number three was designed as the treatment condition of rest and meditation prior to exercise and recovery. Significant reductions ($P < 0.01$) between rest and meditation were observed in $\dot{V}_E$ and GSR while a definite trend ($P < 0.05$) was seen in carbon dioxide elimination. There was no difference in blood lactate and only a slight decrease in oxygen consumption.

In view of these findings it seems that the subjects were not capable of producing the same degree of relaxation through meditation as was observed in the previous experimental condition. The reason for these results is unknown but it might be conceptualized that the prospect of exercise following meditation may have inhibited a full relaxation response.

Gellhorn (1972) stated, "to maintain this state requires a conscious effort. . . ." If the subjects were contemplating exercise while they were meditating this "conscious effort" may have been interrupted and the normal reduction in sympathetic nervous system activity may not have occurred, although this was not reflected in the GSR measurements. GSR was increased significantly in both control and treatment conditions.

Exercise (Subproblem 2)

There were no significant changes in exercise when control and treatment conditions were compared (Table 9). Any of the slight changes demonstrated by a comparison of means can be attributed to normal variation in response to exercise. Keele (1966) and Astrand (1970) have both stated that the normal response to standard exercise varies between 3 and 5 percent.

There are two possible reasons why no changes were seen in exercise. Either the relaxation response prior to exercise in test session

three was not "strong" enough to affect changes in the response to exercise or transcendental meditation has no effect upon the response to exercise. From the results of the present study it must be concluded that the latter is most probable. However it cannot be determined what effect transcendental meditation would have on exercise if experimental subjects were habituated to the experience of meditation followed by exercise. This consideration might be the basis for further investigation.

Recovery (Subproblem 3)

Figures 16 through 21 show that there was very little difference in the recovery response from the two different exercises, when observations were made in the first five mintues of recovery. However when the results of the last twelve minutes or the meditating portion of recovery were analysed a significant decrease in the minute ventilation was found (Table 10). It should be noted that a trend ($P < 0.05$) of decreased heart rate and carbon dioxide elimination was also observed.

It is not surprising that little difference was found in the initial five minutes of recovery. Since meditation had no significant treatment effect on exercise it would not be expected that the non meditating recovery response from this exercise would differ from the control recovery. The first five minutes of recovery therefore represented a normal response to standard exercise in both conditions.

In the treatment condition the reduction in $\dot{V}_E$ and $\dot{V}_E co_2$ found in the meditating portion of recovery (Table 10) coincided with the decreases in these variables shown for rest and meditation (Table 8). Meditation during recovery seemed to elicit the same hypometabolic

cardiorespiratory response as found in normal meditation, which may be a result of reduced sympathetic discharge to the chemoreceptors and the respiratory control center in the medulla (Benson 1974, and Gellhorn 1972). The reduced heart rate during meditation and recovery could also be explained in terms of a decreased sympathetic discharge to the vasomotor control center in the medulla although the decrease in heart rate during recovery (Figure 17) began before the meditating portion of recovery. Therefore improvement in recovery heart rate was not necessarily a function of meditation.

An explanation for the lack of change in $\dot{V}o_2$ during recovery (Table 10) could be obtained from previous research related to repayment of oxygen debt and control of respiration during exercise and recovery.

Although there has been much controversy in the literature concerning oxygen debt and repayment of it, the classical position taken by Margaria, et al., (1933) has stood the test of time. Margaria was the originator of the terms "alactacid" and "lactacid". The alactacid debt is assumed to be due to the replacement of the body's oxygen stores and high energy phosphates. The lactacid portion of oxygen debt is related to the restoration of homeostasis in blood and skeletal muscle primarily via the lactate dehydrogenase system which oxidizes lactate to pyrvuate (Astrand 1970, Dempsey 1967, Knuttgen 1961).

During normal recovery from exercise oxygen debt results in part from lactacidosis and a certain degree of tissue hypoxia. In the arterial blood these two conditions are evidenced by low Po_2 and high Pco_2, which in turn causes increased activity of the central and peripheral chemoreceptors (Sørensen 1971). Hypercapnic and hypoxic drive are

therefore thought to be the primary regulatory mechanisms for increased $\dot{V}o_2$ and ventilation observed during recovery from exercise (Sørensen 1971, Dempsey 1967, and Dejours 1964).

Although in the present study $\dot{V}_E$ and $\dot{V}_Eco_2$ decreased during recovery with meditation there was an increased uptake of oxygen as evidenced by F_Eo_2. This resulted in no net change in $\dot{V}o_2$. It might be conceptualized that the respiratory control center was receiving conflicting inputs. On one hand meditation caused decreased sympathetic activity which called for a reduction in ventilation while simultaneously the alactacid and lactacid portions of oxygen debt demanded that the oxygen supply be maintained as in normal recovery.

The above concept becomes more appealing upon the realization that there was no significant difference in the concentration of blood lactates found in exercise (Table 9). In addition no significant difference in the removal of lactate was shown in the recovery data (Table 10). Hence it may be concluded that there was no difference in the excess oxygen consumed during recovery associated with lactacid oxygen debt.

The fact that there was no change in the removal of lactate during recovery with meditation (Table 10) could be explained in light of the proposed mechanisms for reduction in lactate concentration found duirng normal meditation. The explanations of increased aerobic metabolism given by Wallace (1971) as well as increased uptake of lactate by cardiac and skeletal muscle (Karlsson 1972, Hermansen 1972 and Rowell 1971), are both based upon the assumption of increased muscle blood flow. Exercise itself causes a redistribution of, and an increase in blood

flow to working muscle and this condition continues into recovery (Astrand 1970, p. 147-150). It therefore might be anticipated that no further increase in muscle blood flow would occur as a result of transcendental meditation. If such was the case the explanations of reduced blood lactate during normal meditation could not be applied to recovery and meditation. Hence, it is not surprising that meditation had no effect on the removal of lactate during recovery.

The significant decrease in $\dot{V}_E$ and the reduction in $\dot{V}_E co_2$ and heart rate (Table 10) found during recovery points to the fact that transcendental meditation does effect those variables which are under the control of the autonomic nervous system during recovery. The lack of change in $\dot{V}o_2$ and blood lactate suggests that relaxation techniques have no effect on energy metabolism during recovery. Exercise produces a stress which is physiologically related to the energy cost of the exercise and mental techniques do not seem to alliviated this stress.

CHAPTER VI

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Summary

This study was designed to examine the effects of the technique of relaxation called transcendental meditation, on resting, exercise, and recovery metabolism. There were three questions or subproblems investigated in this study: 1. Is there a difference between the resting and meditating metabolism? 2. Does transcendental meditation affect exercise when it is used prior to exercise? 3. What is the effect of transcendental meditation on recovery when it is practiced during recovery? It was suggested that if meditation could improve the physiological responses to exercise and recovery, it could have practical applications for physical educators and coaches.

Twelve volunteer subjects were studied, each of whom acted as his own control. All subjects were untrained males and had a minimum experience in meditation of eight months ($\bar{x}$ = 23). Each subject was tested on three separate occasions. The three testing sessions were called: 1. control for meditation, 2. control for exercise and recovery, and 3. treatment for exercise and recovery. In the control for meditation resting and meditating data was taken for the following variables: $\dot{V}o_2$, $\dot{V}_E co_2$, $\dot{V}_E$, HR, respiratory rate, blood lactate and GSR. In the control and treatment sessions for exercise and recovery the same physiological parameters were examined with the exception of GSR.

Group means and standard deviations were calculated for each variable in each condition of the three testing sessions. In every condition simple comparisons were made for each variable via a t test for paired groups. These comparisons were: 1. rest and meditation in control for meditation, 2. rest and meditation in treatment for exercise and recovery, 3. exercise in both control and treatment for exercise, and 4. recovery in both control and treatment for recovery.

In the control for meditation significant decreases in $\dot{V}o_2$, $\dot{V}_E co_2$, and $\dot{V}_E$ with an increase in GSR were found during meditation. These results were in accordance with the findings of Wallace (1971) and others. In the treatment session for exercise and recovery the same degree of relaxation through meditation was not found. Only a significant decrease in $\dot{V}_E$ with a large rise in GSR was observed during meditation. It was suggested that this difference in meditation may have been a result of the subjects thinking about exercise. If the subjects were nervous about the exercise this may have interrupted their meditations and caused a less profound "relaxation response", although this was not reflected by GSR.

There were no changes in any of the variables during the first five minutes of recovery; and therefore any slight changes that occurred were a result of the normal variability in recovery from standard exercise. There was a significant decrease in $\dot{V}_E$ and a definite trend of decreased HR and $\dot{V}_E co_2$ during the last twelve minutes of recovery with meditation. It was suggested that meditation during recovery resulted in a certain degree of decreased sympathetic activity, but no change in $\dot{V}o_2$ was observed because of the need for the repayment of alactacid and

lactacid portions of oxygen debt. The changes found in recovery with meditation seem to indicate that autonomic function can be controlled during recovery stress as has been found in normal meditation. Meditation however does not seem to affect energy turnover and metabolism during recovery.

Conclusions

Within the scope and limitations of this study, the following may be concluded.

1. Consistent physiological changes, which are indicative of a hypometabolic state, are found during the technique of relaxation called transcendental meditation.

2. When transcendental meditation is used prior to exercise, experimental subjects do not seem capable of a normal meditation, although the decrease in $\dot{V}_E$ with an increase in GSR, mark the presence of a "relaxation response".

3. Transcendental meditation has no effect upon the normal physiological response to exercise when it is practiced prior to exercise.

4. The practice of transcendental meditation during recovery can cause a improvement in physiological variables controlled by the automatic nervous system; $\dot{V}_E$, $\dot{V}_E co_2$, and HR. Relaxation techniques such as meditation seem to have little affect on those parameters which reflect energy metabolism such as $\dot{V}o_2$ and blood lactate.

Recommendations

There is a need for further investigation of the effects of transcendental meditation. The use of a larger experimental sample and a

separate control sample would enhance further research in this area. Particular emphasis might be directed to the study of the effects of relaxation techniques on exercise and recovery, when the use of these techniques prior to exercise and during recovery becomes an habituated experience.

BIBLIOGRAPHY

BIBLIOGRAPHY

ABRAMS. "Increased Learning Ability" 1972, Scientific Research on Transcendental Meditation. Los Angeles: MIU Press, 1972.

AKISHIGE, Y. "A Historical Survey of the Psychological Studies in Zen", Kyushu Psychol. Studies. V, Bull. Fac. Lit. Kyushu Univ. 11: 1-56, 1968.

ALLISON, J. "Respiration Changes during Transcendental Meditation", Lancet. 1: 833-834, 1970.

ANAND, B.K., G.S. CHHINA, and B. SINGH. "Some aspects of Electroencephalographic Studies in Yogis", Electroencephalog. Clin. Neurophysiol. 13: 452-456, 1961.

ASTRAND, P.O. Textbook of Work Physiology. New York: McGraw-Hill Book Co., 1970.

ASTRAND, P.O. and B. SALTIN. "Maximal Oxygen Uptake and Heart Rate in Various Types of Muscular Activity", J. Appl. Physiol. 16: 977, 1961.

AX, A.F. "The Physiological Differentiation between fear and anger in Humans", Psychosom. Med. 15: 433-442, 1953.

BAGCHI, B.K. and M.A. WENGER. "Electrophysiological Correlates of some Yogi Exercises", Electroencephalog. Clin. Neurophysiol. Suppl. 7: 132-149, 1957.

BEAM, J.C. "Serial learning and conditioning under real Life Stress", J. of Abnorm. and Soc. Psych. 51: 543-551, 1955.

BENSON, H. N. Engl. J. Med. 281: 1133, 1971.

BENSON, H. "Yoga for drug abuse", N. Engl. J. Med. 281: 20, 1969.

BENSON, H., J.F. BEARY and M.P. CAROL. "The Relaxation Response", Psychiatry. 37: 37-45, February, 1974.

BENSON, H., B.A. ROSNER, and B.R. MARZETTA. "Decreased Systolic Blood Pressure in Hypertensive Subjects Who Practiced Meditation", J. Clini. Invest. 52: 8A, 1973.

BENSON, H. and R.K. WALLACE. "Decreased drug abuse with Transcendental Meditation - a study of 1,862 subjects, in Drug Abuse", Proceedings of the International Conference. Philadelphia: Lea & Febiger, 1972, pp. 369-376.

BISCOE, T.J. and M.J. PURVES. "Carotid Chemoreceptor and Cervical Sympathetic Activity during Passive Third Limb Exercise in the Anaesthetized Cat", J. Physiol (London). 178: 43P, 1965.

BLASDELL. "Superior Perceptual-Motor Performance" 1971, Scientific Research on Transcendental Meditation. Los Angeles: MIU Press, 1972.

BOLME, P. and J. NOVOTNY. "Oxygen Uptake in skeletal muscle of the anaesthetized Dog during Sympathetic Vasodilation", Acta Physiol. Scand. 77: 333-343, 1969.

CAMPBELL, D.T. and J.C. STANLEY. Experimental and Quasi-Experimental Designs for Research. Chicago: Rand McNally Co., 1963.

CANNON, W.B. "The Emergency Function of the Adrenal Medulla in Pain and the Major Emotions", Amer. J. Physiol. 33: 356, 1941.

CARRON, A.B. "Reliability of Activation Level during Adaption to Stress", Science. 129: 784, 1959.

CARRON, A.V. "Motor Performance under Stress", Res. Quart. 39: 463, 1968.

CASTANEDA and LIPSITT. "Relation of Stress and differential position habits to motor performance in Motor Learning", J. of Exper. Psych. 57: 25-30, 1965.

COHEN, M.E. and P.D. WHITE. "Life situation, emotions and neurocirculatory asthenia (anxiety neurosis, neurasthenia, effort syndrome)", Res. Publ. Assoc. Res. Nervous Mental Disease. 29: 832-896, 1950.

COMROE, J. Jr. "The peripheral chemoreceptors", Handbook of Physiology, sect. 3, Respiration, Washington, D.C.: American Physiological Society, 1964.

CONSOLAZIO, F., R.E. JOHNSON, and L.J. PECORA. Physiological Measurements of Metabolic Functions in Man. New York: McGraw-Hill, 1963.

DATEY, K.K., DESHMUKH, DALVI and VINEKAR. "'Shavasan': a Yogi Exercise in the Management of Hypertension", Angiology. 20: 325-333, 1969.

DECKER, D.G. and J.D. ROSENGAUM. "The distribution of lactic acid in human blood", Am. J. Physiol. 138: 7-11, 1942-43.

DEFARES, J.G. "Principles of feedback control and their application to the respiratory control system", Handbook of Physiology, sect. 3, Respiration, Washington, D.C.: American Physiological Society, 1964.

DEJOURS, Pierre. "Control of respiration in muscular exercise", Handbook of Physiology, sect. 3, Respiration, Washington, D.C.: American Physiological Society, 1964.

DEMARTINI, F.E., CANNON, STASON, and LARAGH. "Lactic Acid Metabolism in Hypertensive Patients", Science. 148: 1482-1484, 1965.

DEMPSEY, J.A. and J. RANKIN. "Physiological Adaptations of Gas transport Systems to muscular work in Health and Disease", Am. J. Physic. Med. 46: 582-633, 1967.

DENNY, P.J. "Effects of Anxiety and Intelligence on Concept Formation", J. of Exper. Psych. 72: 596-602, 1966.

EULER, C.V. "Proprioceptive Control of Respiration", in R. Granit (Ed.), Muscular Afferents and Motor Control, Nobel Symposium 1, New York: John Wiley and Sons, Inc., 197, 1966.

EVANS, Laura. "The Influence of Relaxation Techniques on the varying level of Tension in College Women", PH. D. Dissertation, Iowa State University, 1954.

FORESTER, H.V., J.A. DEMPSEY, J. THOMPSON, E. VIDRUK, and G.A. DoPICO. "Estimation of arterial Po_2, Pco_2, pH and lactate from Arterialized Venous Blood", J. Appl. Physiol. 32: 134-137, 1972.

GELLHORN, E. "Physiological Foundations of Neurology and Psychiatry", Minneapolis: University of Minnesota Press, 1953.

GELLHORN, E. and W.F. KIELY. "Mystical States of Consciousness: Neurophysiological and Clinical Aspects", The Journal of Nervous and Mental Disease. 154: 399-405, 1972.

GOLDMAN, M.J. Principles of Clincial Electrocardiography. Los Altos, California: Lange Medical Pub., 1970.

GUYTON, A.C. Textbook of Medical Physiology. London: W.B. Saunders Co., 1971.

HARLESTON, B.W., G.M. SMITH, and D. AREY. "Test-anxiety level, Heart Rate, and Anagram Problem Solving", J. of Person. and Soc. Psych. 1: 551-557, 1965.

HAVERLAND, L.E. "The Effects of Relaxation Training on certain aspects of Motor Skills", PH. D. Dissertation, University of Illinois, 1953.

HAWKINS, D.R., PURYEUR, WALLACE, DEAL and THOMAS. "Basal Skin Resistance during Sleep and Dreaming", Science. 136: 321-322, 1962.

HAYS, W.L. Statistics for Psychologists. New York: Holt, Rinehart and Winston, Inc., 1963.

HERMANSEN, L. and B. SALTIN. "Blood lactate concentration during exercise at acute exposure to altitude", Exercise at Altitude. Excerpta Medica Foundation. Amsterdam, 1967, p. 48-53.

HERMANSEN, L. and I. STENSVOLD. "Production and removal of Lactate during Exercise in man", Acta Physiol. Scand. 86: 191-201, 1972.

HESS, W.R. Functional Organization of the Diencephalon. Grune and Strattan, 1957.

HOLMGREN, A. and G. STROM. "Blood Lactate Concentration in relation to absolute and relative work load in Normal Men and in Mitral Stenosis, Atrial Septal defect, and Vaso Regulatory Asthenia", Acta. Med. Scandinav. 163: 185-193, 1959.

HORI, T., MIYASITA, and NIIMI. "Skin potential Activity and their Regional Differences during Normal Sleep in Humans", Jap. J. Physiol. 20: 657-671, 1970.

HOWELL, M.L. "Influences of Emotional Tension on speed of Reaction and Movement", Res. Quart. 24: 22-32, 1953.

HUCKABEE, W.E. "Relationship of Pyruvate and Lactate during Anaerobic Metabolism. II. Exercise and formation of O_2 - Debt", Clin. Invest. 37: 255-262, 1958.

JACOBSON, E. "The Course of Relaxation in Muscles of Athletes", Am. J. of Psych. 48: 98-108, 1936.

JACOBSON, Edmund. Progressive Relaxation. Chicago: University of Chicago Press, 1938.

JONES, M. and V. MELLERSH. "Comparison of Exercise Response in Anxiety States and Normal Controls", Psychosomat. Med. 8: 180-187, 1946.

JORFELDT, L. "Metabolism of L (+) - Lactate in Human Skeletal during Exercise", Acta Physiol. Scand. Suppl. 338, 1970.

KARLSSON, J. "Lactate and phosphogen concentration in working muscle of man", Acta Physiol. Scand. Suppl. 358, 1971A.

KARLSSON, J. "Lactate in working muscle after prolonged exercise at standardized work loads", Acta Physiol. Scand. 82: 123-130, 1971B.

KARLSSON, J., L. NORDESJÖ, L. JORFELDT and B. SALTIN. "Muscle lactate ATP and CP levels during exercise after physical training in man", J. of Appl. Physiol. 33: 199-203, 1972.

KASAMATSU, A. and T. HIRAI. "An Electroencephalographic Study on the Zen Mediation (Zazen)", Folia Psychiat. Neurol. Japon. 20: 315-336, 1966.

KASCH, F.W., W.H. PHILLIPS, W.D. ROSS, J.E.L. CARTER, and J.L. BOYER. "A Comparison of Maximal oxygen uptake by Treadmill and Step Test Procedures", J. Appl. Physiol. 21: 1387, 1966.

KEELE, C.A. and E. NEIL (editors). Samson Wright's Applied Physiology. London: Oxford University Press, 1965.

KEITH, V. Design and Analysis in Experimentation. Ottawa: University of Ottawa Press, 1972.

KELLOGG, Ralph H. "Central chemical regulation of respiration", Handbook of Physiology, sect. 3, Respiration, Washington, D.C.: American Physiological Society, 1964.

KING, Shirley. "Relaxation and Stress", M.ED. Thesis, University of North Carolina, 1958.

LADER, M.H. and A.M. MATHEWS. "Comparison of Methods of Relaxation using Physiological Measures", 1970.

LEE, K.D., R.A. MAYON and R.W. TORRANCE. "The Effect of Blood Pressure upon Chemoreceptor Discharge to Hypoxia and Modifications of this effect by the Sympathetic - adrenal system", J. Quart. Exp. Physiol. 49: 171, 1964.

LIEDERMAN, P.H. and D. SHAPIRO. "Studies on the Galvanic Skin Potential Level: some Behavioral correlates", J. Psychosom: Res. 7: 277-281, 1964.

LINKO, E. "Lactic acid Response to Muscular Exercise in Neurocirculatory Asthenia", Ann. Med. Int. Fenniae. 39: 161-176, 1950.

LIST, W. "Meditators extol the Maharishi's Transcendental Techniques", Globe and Mail (Toronto), November 21, 1972.

LYONS, M.D. and B. LUFKIN. "Evaluation of tension control courses for college Women", Research Quart. 38: 663-70, 1967.

MARGARIA, R., H.T. EDWARDS and D.B. DILL. "The possible mechanism of Contracting and paying the oxygen debt and the role of lactic acid in muscular Contraction", Am. J. Physiol. 106: 689, 1933.

MARHARISHI MAHESH, Yogi. Maharishi Mahesh Yogi on the Bhagavad Gita: A New Translation and Commentary. Baltimore: Penguin, 1969, p. 10-17.

MAHARISHI MAHESH, Yogi. The Science of Being and the Art of Living. London: Intern. SRM Publishers, 1966, p. 50-59.

MARTIN, B. "The Accessment of Anxiety by Physiological Behavioral Measures", Psych. Bull. 58: 234-255, 1961.

MATHEWS, A.M. and M.G. GELDER. "Psycho-physiological investigations of brief relaxation training", J. Psychosom. Res. 13: 1-12, 1969.

MILES, W.R. "Oxygen consumption during three yoga-type breathing patterns", J. Appl. Physiol. 19: 75-82, 1964.

NAGLE, F., D. ROBINHOLD, E. HOWLEY, J. DANIELS, G. BAPTISTA and K. STOEDEFALKE. "Lactic acid accumulation during running at submaximal aerobic demands", Medicine and Science in Sports. 2: 182-186, 1970.

NAKAMURA, C.Y. and W.E. BROEN, Jr. "Facilitation of competing responses as a function of 'Subnormal' drive conditions", Journal of Experimental Psychology. 69: 180-185, 1965A.

NAKAMURA, C.Y. and W.E. BROEN, Jr. "Further study of effects of low drive states on competing responses", Journal of Experimental Psychology. 70: 434-436, 1965B.

ORME-JOHNSON, D.W. "Autonomic Stability and Transcendental Meditation", Psychosomatic Medicine. 35: 341-348, July-Aug. 1973.

OTIS, L. "Reduced use of Prescribed and Non-prescribed Drugs" 1972, Scientific Research on Transcendental Meditation. Los Angeles: MIU Press, 1972.

PITTS, F.N. and J.N. McCLURE. "Lactate Metabolism in Anxiety Neurosis", New Engl. J. Med. 277: 1329-1336, 1967.

RAO, S. "Oxygen consumption during yoga-type breathing at altitudes of 520 m and 3800m.", Indian J. Med. Res. 56: 701-705, 1968.

RATHBONE, J.L. Relaxation. Philadelphia: Lea and Febiger, 1969.

RIECHERT, H. "Plethysmograpische Untersuchengen bei Konxentrations-und Meditationsubungen", Arztliche Forsch. (English Abstract), 21: 61-65, 1967.

ROWELL, L.B. "The liver as an energy source in man during exercise", Muscle Metabolism during Exercise. New York: Plenum Press, 1971, p. 127-141.

RYAN, E.D. "Effects of Stress on Motor Performance and Learning", Res. Quart. 33: 111, 1962.

SALTIN, B. and P.O. ASTRAND. "Maximal oxygen uptake in Athletes", J. Appl. Physiol. 23: 353, 1967.

SCOTT, M.G. Research Methods. American Assoc. for Health Physical Education and Recreation, Washington, 1959.

SELYE, H. The Stress of Life. New York: McGraw-Hill Co., 1956.

SHAW and KOLB. "Faster Reaction Time" 1971, Scientific Research on Transcendental Meditation. Los Angeles: MIU Press, 1972.

SHEPPARD, R.J. "The Relative Merits of the step test, bicycle ergometer and treadmill in assessment of Cardiovascular Fitness", Intern. Z. Angew. Physiol. 23: 219, 1966.

SØRENSEN, S.C. "The Chemical Control of Ventilation", Acta Physiol. Scand. Suppl. 361, 1971.

SPENCE, K.W. "A Theory of emotionally based drive (D) and its relation to performance in simple learning situations", American Psychologist. 13: 131-141, 1958.

SPIELBERGER, C.D. and L.H. SMITH. "Anxiety (drive), stress, and serial-position effects in serial verbal learning", Journal of Experimental Psychology. 72: 589-595, 1966.

SUGI, Y. and K. AKUTSU. "Studies on Respiration and Energy Metabolism during setting in Zazen", Res. J. Phys. Ed. 12: 190-206, 1968.

TART, C.T. "Patterns of Basal Skin Resistance during Sleep", Psychophysiol. 4: 35-39, 1967.

VAN de VALK, J.M. and J. GROEN. "An Investigation of the Electrical Resistance of the skin during induced emotional stress in normal individuals and in patients with internal disease", Proc. Ass. Res. Nerv. Ment. Dis. 29: 279, 1950.

WALLACE, R.K. "Change in Cardiac Output" 1970, Scientific Research on Transcendental Meditation. Los Angeles: MIU Press, 1972.

WALLACE, R.K. "Physiological Effects of Transcendental Meditation", Science. 167: 1751-1754, 1970.

WALLACE, R.K. and H. BENSON. "The Physiology of Meditation", Sci. Amer. 226: 84-90, 1972.

WALLACE, R.K., H. BENSON and A.F. WILSON. "A Wakeful Hypometabolic State", American Journal of Physiology. 221: 795-799, 1971.

WALLAS, R.H. "Physiological Variations and Normal Values of the Total Bases in Blood Serum", Acta. Physiologia. Scandinav. 17: 235-254, 1949.

WEBER, J.C. and D.R. LAMB. Statistics and Research in Physical Education. St. Louis: Mosby Co., 1970.

WENGER, M.A. and B.K. BAGCHI. "Electrophysiological correlates of some Yogi exercises", Electroencephalog. Clin. Neurophysiol. Suppl. 7: 132-149, 1957.

WENGER, M.A. and B.K. BAGCHI. "Studies of autonomic functions in practitioners of Yoga in India", Behavioral Sci. 6: 312-323, 1961.

WHALEN, W.J. and P. NAIR. "Po_2 in Skeletal Muscle", Federation Proceedings. 28. comm. 1442, 519. 1969.

WILSON, A. and A.S. WILSON. "Psychophysiological and Learning Correlates of Anxiety and induced muscle Relaxation", Psychophysiol. 6: 740-8, 1970.

WOLFF, H.G. Stress and Disease. Springfield: Charles C. Thomas Pub., 1968.

WOLFF, H.G. "Life Stress and Bodily Disease", Bull. New York Acad. Med. 52: 1680188, 1952.

WOLF, G.A. and H.G. WOLFF. "Study on the nature of certain symptoms associated with Cardiovascular Disorders", Psychosom. Med. 8: 293, 1946.

APPENDIX A

1A RESPIRATORY METABOLISM: CALCULATIONS

2A EXAMPLE OF A t TEST FOR CORRELATED OR PAIRED GROUPS

1A Respiratory Metabolism: Calculations

(Consolazio 1963)

Pulmonary Ventilation

Pulmonary or minute ventilation is expressed as liters of air expired per minute, the volume of gas being reduced to standard temperature and pressure 0°C and 760 mm Hg, dry. The formula is

$$\frac{P_B - P_{H_2O}}{760\ (1 + 0.00367T)}$$

where P_B = ambient barometric pressure
P_{H_2O} = the vapor tension of water, mm Hg, at the temperature of the gasometer
T = the temperature of the gasometer °C

Oxygen Consumption and Carbon Dioxide Elimination

The volume of inspired air may be calculated from the expired air by the formula

(1) $\dot{V}$ inspired = V expired x $\frac{\%\ N_2 \text{ in expired air}}{79.04}$

The total volume of oxygen inspired (not all consumed) is then

(2) $\dot{V}o_2$ inspired = V air inspired $\frac{\%\ O_2 \text{ of inspired air}}{100}$

The percentage of oxygen in outdoor air is 20.93; hence,

$$(3)\quad \dot{V}o_2 \text{ inspired} = \dot{V} \text{ air inspired} \times \frac{20.93}{100}$$

The volume of oxygen expired (amount not consumed) is

$$(4)\quad \dot{V}o_2 \text{ expired} = \frac{\%\ O_2 \text{ in expired air}}{100} \times V \text{ air expired}$$

The amount of oxygen consumed is

$$(5)\quad \dot{V}o_2 \text{ consumed} = \dot{V}o_2 \text{ inspired} - \dot{V}o_2 \text{ expired}$$

Substituting values from Eqs. (3) and (4),

$$\dot{V}o_2 \text{ consumed} = \dot{V} \text{ air inspired} \times \frac{20.93}{100} - V \text{ air expired} \times \frac{\%\ O_2 \text{ in expired air}}{100}$$

Substituting values from Eqs. (1),

$$\dot{V}o_2 \text{ consumed} = \dot{V} \text{ air expired} \times \frac{\%\ N_2 \text{ in expired air}}{79.04} \times \frac{20.93}{100} - \dot{V} \text{ air expired} \times \frac{\%\ O_2 \text{ in inspired air}}{100}$$

Simplifying,

$$(6)\quad \dot{V}o_2 \text{ consumed} = \frac{\dot{V} \text{ air expired}}{100} \left(N_2 \text{ in expired air} \times 0.265 - \%\ O_2 \text{ in expired air}\right)$$

The factor (% N_2 in expired air x 0.265 - % O_2 in expired air) is the true oxygen, the number by which the volume of expired air (divided by 100) is multiplied to give the oxygen consumption. Using a formula similar to that for the derivation of oxygen consumption, the carbon dioxide expired becomes

$$\dot{V}co_2 \text{ expired} = \frac{\dot{V} \text{ air expired}}{100} \times \left(\%\ Co_2 \text{ in expired air} - \frac{\%\ N_2 \text{ in expired air}}{79.04} \times \%\ Co_2 \text{ in inspired air}\right)$$

When one uses outdoor air, this becomes

$$\dot{V}co_2 \text{ expired} = \frac{\dot{V} \text{ air expired}}{100} \left(\%\ Co_2 \text{ in expired air} - 0.03\right)$$

The factor (% Co_2 in expired air - 0.03) is the true carbon dioxide, the number by which the volume of expired air (divided by 100) is multiplied to give the carbon dioxide production.

Hence, the oxygen consumption in liters per minute is expressed as

$$\dot{V}o_2 \text{ (l./min.)} = \frac{\dot{V} \text{ gas (l./min.)}}{100} \times \text{true } O_2$$

The carbon dioxide elimination in liters per minute is expressed as

$$\dot{V}_E co_2 \text{ (l./min.)} = \frac{\dot{V} \text{ gas (l./min.)}}{100} \times \text{true } Co_2$$

2A Example of a t Test for Correlated or Paired Groups

Scores:

Pretest	8	3	7	2	4	6	6	8	2	4
Posttest	10	6	11	4	5	4	6	12	6	6

$m_1 = 5 \quad m_2 = 7 \quad S_1 = 2.19 \quad S_2 = 2.76 \quad r = 0.75$

When group measurements are correlated the standard error is decreased. As a result,

$$S_{m_1 - m_2} = \sqrt{S_{m_1}{}^2 + S_{m_2}{}^2 - 2rS_{m_1}S_{m_2}}$$

The standard error of $m_1 - m_2$ must be calculated

$$S_{m_1} = \frac{2.19}{\sqrt{9}} = 7.3$$

$$S_{m_2} = \frac{2.76}{\sqrt{9}} = 0.92$$

Therefore,

$$S_{m_1 - m_2} = \sqrt{.5329 + .8464 - 2\,(.75)\,(.6716)}$$

$$= \sqrt{0.3719}$$

$$= 0.61$$

$$t = \frac{D}{S_D} = \frac{2}{0.61} = 3.28$$

$$0.95t_9 = \pm 2.26$$

Since $t = 3.28$ excceds $0.95t_9$, the $m_2 - m_1$ difference is significant. In other words, the null hypothesis that there is no difference between the means is rejected with 95% confidence.

APPENDIX B

METHOD OF LACTATE ANALYSIS

Method of Lactate Analysis

Biochemica Test Combination
Cat. No.: 15972 TLAA

Preparation of solutions

1. Dilute solution in bottle 1 with redist. water and make up to 75 ml. Stable for one year at room temperature.

2. Dissolve contents of bottle 2 in 7 ml. redist. water. Stable for four weeks at approx. + 4°C.

3. Use suspension in bottle 3 undiluted. Stable for one year at approx. + 4°C.

In addition

Perchloric acid, approx. 0.6 N, Cat.-No.: 15902 TYAG or dilute 5.15 ml. 70% (6.5 ml. 60%) perchloric acid with redist. water and make up to 100 ml.

Concentration of solutions:

1. 0.5 M glycine buffer, pH 9.0; 0.4 M hydrazine

2. 27 mM NAD

3. 2 mg. LDH/ml.

Deproteinisation

Pipette into 10 ml. centrifuge tube:

perchloric acid, ice-cold	1.0 ml.
blood (taken from the vein without stasis)	0.5 ml.

Mix well, centrifuge for 10 min. at approx. 3000 r.p.m.

Spectrophotometric measurements

Wavelength: 366, 340 or 334 nm
Glass cuvette: 1 cm. light path
Temperature: 25°C.

Measure against air. One blank is sufficient for each series of determinations.

Pipette into test tubes:

	blank	sample
solution 1	2.00 ml.	2.00 ml.
supernatant fluid	--	0.20 ml.
perchloric acid	0.20 ml.	--
solution 2	0.20 ml.	0.20 ml.
suspension 3	0.02 ml.	0.02 ml.

Mix, incubate in water bath at 25°C for exactly 1 hour. Pour into cuvette, read optical densities of the sample (E_s) and the blank (E_b) against air.

$E_s - E_b = \Delta E$.

Please note

By using a quarter or half of the volumes given in the pipetting scheme, micro assays can be performed. The calculation factor remains the same.

In case the final volume used in the macro assay is not sufficient to fill the cuvette, take 3.0 ml. of solution 1, 0.3 ml. of the supernatant fluid (or perchloric acid), 0.3 ml. of solution 2, and 0.03 ml. of suspension 3.

Controls can be prepared with 1 N l-lactate (Lactate calibration solution, Cat. No.: 15672 TYAK which is available together with instructions on request, free of charge). A 1:2000 dilution of this 1 N solution will give a solution of 9 µg. l-lactate/0.2 ml.

APPENDIX C

INDIVIDUAL AND GROUP DATA FOR REST,
MEDITATION, EXERCISE, AND RECOVERY.

TABLE C1 - INDIVIDUAL SUBJECTS' DATA FOR REST, DURING CONTROL FOR MEDITATION AND TREATMENT FOR EXERCISE AND RECOVERY

Variables	Subjects 1	2	3	4	5	6	7	8	9	10	11	12
Blood lactate (mg.%)	17.2[1]	8.4	12.8	7.4	12.2	10.7	12.3	10.8	8.4	11.3	5.7	13.4
	10.8[2]	8.4	10.8	15.4	8.8	6.6	15.4	10.8	10.6	10.2	5.4	6.7
Heart rate (b.p.m.)	82.4	75.4	79.2	85.4	67.2	78.8	68.4	80.8	101	82.4	64.2	80.8
	63.2	72.2	73.4	63.4	70.0	69.8	71.6	81.0	92.2	72.2	64.8	79.4
Respiratory rate (breaths/min.)	17.6	12.4	10.6	11.0	11.1	13.6	10	13.8	18.4	16.2	9.6	11.2
	15.6	12.2	8.6	10.4	9.2	12.4	9.8	15.2	18.4	15.4	11.6	7.4
Minute ventilation (l./min.)	9.15	7.93	7.51	9.38	8.27	8.39	7.46	6.09	5.99	9.63	7.16	8.81
	7.48	8.02	8.04	7.28	8.44	7.42	7.24	7.26	5.72	8.77	8.92	10.6
Oxygen consumption (ml./min.)	224	281	298	344	314	247	266	172	174	282	264	286
	205	236	331	285	318	241	279	206	146	259	303	322
Carbon dioxide elimination (ml./min.)	200	240	216	268	238	209	216	151	127	208	209	250
	169	184	240	230	239	183	217	166	113	203	227	220
GSR (kilohms)	127	135	85	72.5	80	80	115	135	95	135	80	85
	135	125	120	120	110	130	95	135	120	120	50	75

[1] Top value is resting data in control for meditation

[2] Bottom value is resting data in treatment for exercise and recovery

TABLE C2 - INDIVIDUAL SUBJECTS' DATA FOR MEDITATION, DURING CONTROL FOR MEDITATION AND TREATMENT FOR EXERCISE AND RECOVERY

	Subjects											
Variables	1	2	3	4	5	6	7	8	9	10	11	12
Blood lactate (mg.%)	16.2[1]	10.5	6.9	5.6	8.7	8.4	12.4	10.4	--	10.2	5.3	9.6
	7.7[2]	12.9	10.0	9.2	7.7	7.2	16.0	9.0	11.0	9.4	6.5	6.3
Heart rate (b.p.m.)	71	74	81.7	89.3	63.7	76.3	67.7	79	93.5	78.6	63.7	73
	75	77.7	72.3	63	62.6	68	65.7	77.7	88.3	65.7	63	71.7
Respiratory rate (breaths/min.)	12.7	11.3	8.7	6	13.7	14	8.3	13.3	19.5	15.3	11.7	8.7
	13.3	10.3	8.3	11	15.3	14.3	9.0	15	18.2	14.3	6.7	7.7
Minute ventilation (l./min.)	7.20	68.6	7.28	5.70	7.50	6.89	5.98	5.20	8.87	8.46	6.25	6.99
	7.01	7.30	7.51	7.17	7.54	7.02	5.96	6.58	7.31	7.92	6.71	6.49
Oxygen consumption (ml./min.)	199	261	296	233	285	212	229	157	286	246	207	259
	198	281	279	273	294	225	223	217	212	230	288	303
Carbon dioxide elimination (ml./min.)	163	198	218	169	216	163	173	124	197	182	162	202
	155	215	214	234	218	168	168	164	159	178	193	182
GSR (kilohms)	220	250	185	215	175	225	185	200	195	275	195	205
	225	235	200	225	200	225	235	185	245	245	190	175

[1] Top value is meditating data in control for meditation

[2] Bottom value is meditating data in treatment for exercise and recovery

TABLE C3 - INDIVIDUAL SUBJECTS' DATA FOR EXERCISE, DURING CONTROL AND TREATMENT SESSIONS

Variables	Subjects 1	2	3	4	5	6	7	8	9	10	11	12
Blood lactate (mg.%)	25.5[1]	19	48.7	--	43.5	27.7	44.6	40.8	28.6	31.4	31.9	30.0
	24.9[2]	24.9	56.4	15.7	21.1	28.1	53.7	44.8	31.3	38.1	41.3	19.6
Heart rate (b.p.m.)	168	172	165	169	169	171	172	171	171	167	170	165
	166	170	168	166	167	171	170	171	171	167	171	165
Respiratory rate (breaths/min.)	23	24	26	20	26	26	25	32	28	31	26	21
	22.4	26	24	18	26	28	23	28	28	30	24	18
Minute ventilation (l./min.)	51.8	47.1	69.9	63.0	64.9	56.3	60.2	58.5	73.2	75.2	62.2	58.9
	61.2	53.0	74.0	53.1	59.6	57.8	61.6	60.5	67.8	76.6	76.5	49.1
Oxygen consumption (ml./min.)	2.23	2.27	2.76	3.07	2.97	2.45	3.18	1.91	3.17	2.53	2.80	2.60
	2.08	2.47	2.96	2.71	2.74	2.46	2.85	1.96	2.51	2.50	3.23	2.48
Carbon dioxide elimination (l./min.)	2.07	1.98	2.73	2.73	2.53	2.14	2.77	1.81	2.85	2.78	2.61	2.65
	2.32	2.17	2.74	2.55	2.56	2.14	2.59	1.93	2.47	2.41	2.91	2.21

[1] Top value is control for recovery

[2] Bottom value is treatment for recovery

TABLE C4 - GROUP DATA FOR RECOVERY, DURING CONTROL AND TREATMENT SESSIONS

Variables	Time of Recovery in Minutes											
	1	2	3	4	5	7	9	11	13	15	17	19
Heart rate (b.p.m.)	145[1]	112	99	98	97	96	95	92	92	91	89	89
	143[2]	110	98	94	92	93	91	89	89	87	87	86
Respiratory rate (breaths/min.)	23	22	20	18	19	17	17	16	15	16	14	15
	23	22	22	17	18	18	16	15	14	14	14	14
Minute ventilation (l./min.)	38.18	26.51	19.91	17.37	15.29	12.36	10.88	10.25	9.75	9.50	8.90	8.76
	43.14	29.98	20.07	16.10	15.43	12.89	10.91	9.00	8.25	8.38	8.40	8.30
Oxygen consumption (ml./min.)	1563	1008	643	500	402	338	323	321	321	308	293	309
	1801	1144	619	451	427	361	334	305	281	299	302	296
Carbon dioxide elimination (ml./min.)	1525	1050	726	587	482	357	297	267	251	235	217	224
	1722	1165	697	525	482	371	320	244	214	192	213	211

[1] Top value is the control recovery [2] Bottom value is recovery with meditation

ABSTRACT

This study investigated the effects of "transcendental meditation" on resting, exercise, and recovery metabolism. Twelve untrained male subjects were studied, each of whom served as his own control. Vo_2, V_Eco_2, V_E, HR, , GSR, and blood lactate were measured during each condition. Significant changes in Vo_2, V_Eco_2, V_E and GSR were found during meditation. There was no change in the response to exercise, when the subjects meditated prior to exercise. A significant change in V_E was observed when the subjects meditated during recovery from exercise, and a slight decrease was found in V_Eco_2 and heart rate. The physiological changes that occurred with the practice of meditation indicated that transcendental meditation produces a hypometabolic state. Meditation had no effect on exercise performance, but it may have practical application during recovery from exercise.