EFFECTS OF AGE, FITNESS LEVEL, AND EXERCISE TRAINING UPON AUTONOMIC CONTROL OF HEART RATE

THESIS

Presented to the Graduate Council of the North Texas State University in Partial Fulfillment of the Requirements

For the Degree of

MASTER OF SCIENCE

By

William Boyd Baun, B.S.
Denton, Texas
May, 1980
Baun, William B., Effects of Age, Fitness Level, and Exercise Training Upon Autonomic Control of Heart Rate. Master of Science (Physical Education), May, 1980, 75 pp., 6 tables, bibliography, 81 titles.

In this study the effects of age (18-55 years), differing levels of fitness (VO$_2$max ranging from 35.5 to 68.8 ml.kg.min$^{-1}$) and endurance training (10 weeks) on heart rate control were investigated. Fitness level was initially determined by a VO$_2$max stress test, succeeded by cold hand and cold face pressor test of autonomic activity. Following these baseline measurements, the subjects (32 nonsmoking male volunteers) were endurance-trained three to four times a week for a 10-week period. The baseline tests were readministered following the 10-week dynamic exercise training period. These data suggest that a natural consequence of aging is a diminishment of autonomic heart rate control; however, endurance training appears to interrupt the aging influence. Individuals of low fitness level appear to have heart rate control dominated by the sympathetic system, while individuals with high fitness levels have a vagally dominated heart rate control system.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>LIST OF TABLES</th>
<th>vi</th>
</tr>
</thead>
<tbody>
<tr>
<td>LIST OF ILLUSTRATIONS</td>
<td>vii</td>
</tr>
</tbody>
</table>

**Chapter**

<table>
<thead>
<tr>
<th>I. <strong>INTRODUCTION</strong></th>
<th>1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Statement of the Problem</td>
<td></td>
</tr>
<tr>
<td>Hypotheses</td>
<td></td>
</tr>
<tr>
<td>Limitations</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>II. <strong>REVIEW OF LITERATURE</strong></th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autonomic Control of Heart Rate</td>
<td></td>
</tr>
<tr>
<td>Effects of Endurance Training on Heart Rate</td>
<td></td>
</tr>
<tr>
<td>Effects of Age on Heart Rate</td>
<td></td>
</tr>
<tr>
<td>Non-Invasive Determination of Heart Rate Control</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>III. <strong>METHODS</strong></th>
<th>22</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects</td>
<td></td>
</tr>
<tr>
<td>Procedures</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>IV. <strong>RESULTS</strong></th>
<th>31</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic and Body Composition Measurements</td>
<td></td>
</tr>
<tr>
<td>Resting Heart Rate</td>
<td></td>
</tr>
<tr>
<td>Cold Hand Pressor Test Heart Rate Response</td>
<td></td>
</tr>
<tr>
<td>Cold Hand Pressor Test Blood Pressure Response</td>
<td></td>
</tr>
<tr>
<td>Cold Face Pressor Test Heart Rate Response</td>
<td></td>
</tr>
<tr>
<td>Cold Face Pressor Test Blood Pressure Response</td>
<td></td>
</tr>
<tr>
<td>Recovery from Cold Hand Pressor Test--Rebound</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>V. <strong>DISCUSSION</strong></th>
<th>48</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aging Effects on Autonomic Heart Rate Control</td>
<td></td>
</tr>
<tr>
<td>Fitness and Training Effects on Autonomic Heart Rate Control</td>
<td></td>
</tr>
<tr>
<td>Section</td>
<td>Page</td>
</tr>
<tr>
<td>------------------------------------------------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>Interaction Effects</td>
<td></td>
</tr>
<tr>
<td>Conclusions</td>
<td></td>
</tr>
<tr>
<td>Recommendations for Future Research</td>
<td></td>
</tr>
<tr>
<td>APPENDIX</td>
<td>62</td>
</tr>
<tr>
<td>BIBLIOGRAPHY</td>
<td>68</td>
</tr>
</tbody>
</table>
LIST OF TABLES

<table>
<thead>
<tr>
<th>Table</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. The Effect of Aging on the Adaptations of the Cardiovascular System to Endurance Training</td>
<td>16</td>
</tr>
<tr>
<td>II. Body Weight and Percent Fat Measurements Prior to and Following Training (Group Means ± SEM)</td>
<td>33</td>
</tr>
<tr>
<td>III. Heart Rates Obtained During the Cold Hand Test Prior to and Following Training (Group Means ± SEM)</td>
<td>39</td>
</tr>
<tr>
<td>IV. Relationship Between Cold Face Heart Rate Response Prior to and Following Training</td>
<td>44</td>
</tr>
<tr>
<td>V. Correlations Between Systolic Blood Pressure and Age for the Cold Face Test</td>
<td>45</td>
</tr>
<tr>
<td>VI. Correlation Coefficients and z Transformations Between VO₂max and Change in Blood Pressure During the Cold Face Test</td>
<td>45</td>
</tr>
</tbody>
</table>
LIST OF ILLUSTRATIONS

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Experimental Design</td>
<td>30</td>
</tr>
<tr>
<td>2</td>
<td>Group VO$_{2\text{max}}$ Increase After Training (Group Means ± SEM)</td>
<td>32</td>
</tr>
<tr>
<td>3</td>
<td>Alterations Observed in the Group Mean Heart Rate Response During the Cold Hand Test Before and Following Training</td>
<td>36</td>
</tr>
<tr>
<td>4</td>
<td>The Relationships Between Age and the Increase in Heart Rate During the Cold Hand Test Prior to and Following Training</td>
<td>38</td>
</tr>
<tr>
<td>5</td>
<td>Increases in Systolic Blood Pressure During the Stress Phase of the Cold Hand Tests (Group Means ± SEM)</td>
<td>41</td>
</tr>
<tr>
<td>6</td>
<td>Heart Rate Changes on the Cold Face Tests Prior to and Following Training (Group Means ± SEM)</td>
<td>43</td>
</tr>
<tr>
<td>7</td>
<td>Decrease in Heart Rate Recovery Phase of Cold Hand Tests Associated with Training (Group Means ± SEM)</td>
<td>47</td>
</tr>
</tbody>
</table>
ACKNOWLEDGMENTS

The investigation presented here has resulted in a number of presentations at national and regional meetings. A summary of these is presented below. The work involved in these investigations was supported in part by N.T.S.U. Faculty Grant #35649, and T.C.O.M. Faculty Grant #34340, and National Institute of Aging Special Initiatives Grant #AGO 1450-02.

Presentations and Publications


CHAPTER I

INTRODUCTION

The concept (homeostasis) that the internal environment of a multicellular organism must be stable regardless of the variability of the external environment was first formulated by Claude Bernard. Control of this internal stability is attained by the coordinated activity of both the endocrine and nervous system, the major regulators of homeostasis. Integration of these systems is achieved by the general principle of feedback control. This feedback can be either positive or negative. Unfortunately, with advancing age gradual alterations of homeostatic stability are observed in man. Shock (1961) reported a rise of blood cholesterol and lipoproteins, a fall in maximum ventilatory capacity (MVC), and a decreased inability of the cardiovascular system to adapt to exercise stress with advancing age. How and why gradual alterations of homeostatic stability occur with advancing age is presently only hypothesized in the literature. Descriptive documentation of the physiological deterioration of man during the aging process has been sufficiently detailed by Shock (1961), and by Baffitis and Sargent (1977). A general conclusion drawn from the literature is that the system becomes more susceptible to
age-related changes, the more complex the control of the system.

One of the simplest examples of physiological decrement with age is the decline of resting and maximal exercise heart rates (Åstrand & Rodahl, 1977, pp. 322-323). Yet the simple functional measurement of heart rate is in fact a reflection of complex integration of identifiable neural and hormonal feedback control mechanisms. Available information concerning the decline in resting and normal exercise heart rate with increased age does not clearly define the mechanisms of control.

Currently, exercise training prescriptions utilize the heart rate response at rest, at submaximal exercise, and at maximal exercise as indications of training and trainability. However, this concept is based upon data obtained from young or elite populations. The recent trend of increased participation of the middle-aged and elderly in exercise programs has raised questions concerning the validity of extrapolating exercise prescriptions from such a youthful data base in application to the elderly.

The classical response to endurance training is an increased maximal and submaximal stroke volume and a decreased maximal and submaximal heart rate as a result of the increased fitness level. This has also been observed following training in middle-aged and elderly individuals.
(Barry, Daly, Pruett, Steinmetz, Page, Birkhead, Rodahl, 1966; Hanson, Burton, Levy, & Nedde, 1968). However, others have reported the reduction in submaximal and maximal exercise heart rates without increases in stroke volume (Clausen & Trap-Jensen, 1970). This would suggest that the decreased heart rate response due to training is a reflection not merely of central adaptation but of peripheral involvement. Likewise, stroke volume changes may be a result of increased end-diastolic volume without alterations in end-systolic volume or a decrease in end-systolic volume with a constant end-diastolic volume.

The classical exercise training response in young or elite populations has been demonstrated to be partially affected by an alteration in the autonomic nervous system's control of heart rate. The interplay of sympathetic and parasympathetic activity appears to be an integral part of the mechanism which evokes training bradycardia. However, the age related changes of the autonomic nervous system's involvement in the mechanisms of training bradycardia have yet to be studied. The need for research investigating the effect of endurance training on the apparent alteration in autonomic heart rate control is imperative in determining the training response of the aged. Definitive determination of the mechanisms involved in heart rate alterations associated with aging and exercise training would provide insight.
for the establishment of exercise prescriptions for the elderly that are both effective and within safe upper limits of health tolerance. Unfortunately, evaluation of autonomic control of the heart is only one aspect of heart rate control and will not provide a complete explanation of the effects of aging on the overall training response of the elderly. However, such a study will provide a firm base upon which future work can be designed, and will provide for integrating myocardial mechanics, chronotropy, inotropy, and age related alterations into the elucidation of aging and exercise training.

Statement of the Problem

What effects do fitness level, age, and endurance training have upon the autonomic control of heart rate?

Hypotheses

1. There will be an influence of autonomic control of heart rate that can be attributed to the effect of age.

2. There will be an influence of autonomic control of heart rate that can be attributed to the effect of fitness level.

3. There will be an influence of autonomic control of heart rate that can be attributed to the effect of endurance training.

4. There will be an influence of autonomic control of heart rate that can be attributed to the interaction effects between age, fitness level, and endurance training.
The dependent variables used to test these hypotheses were heart rate response and blood pressure response to standardized cold pressor tests.

**Limitations**

The study was limited by the following factors:

1. Small sample sizes limited analysis of the parameters used to describe each condition.

2. Blood pressure was recorded using a noninvasive method which limited the frequency at which recordings were taken.

3. The endurance training program was a self-reporting system, which introduces recognized problems of control.

4. It was assumed that a 10-week training program would provide sufficient time for changes to occur within the autonomic nervous system's control of heart rate.

5. It was assumed that the cold pressor tests were valid means of evaluating the stability of the autonomic nervous system's control of heart rate.
CHAPTER II

REVIEW OF LITERATURE

Autonomic Control of Heart Rate

Principal extrinsic control of heart rate has been found to exist within the autonomic nervous system. However, the small size of the fibers and the frequent occurrence of mixed sympathetic and parasympathetic fibers in a single bundle has made anatomical and physiological study difficult. Previous work using dogs and baboons has determined that the autonomic system influences the inotropic and chronotropic functions of the heart (Randall, Armour, & Randall, 1971; Randall, Armour, Geis, & Lippincott, 1972). This work suggests that the autonomic neural mechanism has a fine degree of control over the myocardium.

Early observations established the action of the vagus as being a slowing of the heart rate. The use of micro-electrodes and radioactive tracers to record intracellular activity has defined the gross mechanisms involved. Alterations in the membrane potential of pacemaker fibers has indicated an increase in membrane permeability toward potassium ions after vagal stimulation (Mountcastle, 1968, pp. 54-58). This results in a hyperpolarization of the membrane, which means that a greater amount of depolarization in the pacemaker fibers is necessary to reach threshold.
Once threshold is reached, the fibers can discharge an action potential, which causes a heart beat. The exact manner in which these alterations are brought about remains unexplained.

Stimulation of the sympathetic nerve causes an increased rate of membrane polarization in the pacemaker cells of the sinus node during diastole (Mountcastle, 1968, pp. 54-58). The threshold at which action potential discharge occurs is reached sooner, thereby enabling the pacemaker cells to fire more often, thus increasing heart rate. The method by which the threshold for firing is reached earlier remains unclear.

A variety of pharmacological and chemical techniques have been used in studying the antagonism between the two divisions. However, these opposing influences are complicated by the interaction which exists between them. For example, Levy & Ziesk (1969) reported that vagal stimulation produced more depression of myocardial contractility against a background of tonic sympathetic activity than in the absence of sympathetic tone. This disagrees with the immense amount of information compiled by Gellhorn (1957, 1967) and Gellhorn & LoofBourrow (1963) on the integration of the autonomic nervous system. Their work suggests that the antagonism between the sympathetic and parasympathetic systems serves to balance autonomic control. Therefore, the system is constantly in a tuning process. Other investigators
have shown effects normally exerted by one division being evoked by the other division (Copen, Cirillo, & Vassalle, 1968).

Interaction between the two divisions is favored by their anatomical arrangement. The short distances between the nerve terminals allows the transmitters of one fiber to easily diffuse to the nerve terminals of the other division (Levy, 1971). Therefore, neural control of heart rate is complicated by the interaction of the two divisions. Although the basic actions of the two divisions of the autonomic nervous system have been clarified, much work is needed concerning the mechanisms behind these actions, and the interaction between these mechanisms.

Effects of Endurance Training on Heart Rate

Resting heart rate responses. Numerous studies involving animals and humans indicate sinus bradycardia can be evoked by endurance training (Marsland, 1968; Tipton, Carey, Eastin, & Erickson, 1974). Recent data (Scheuer, Tipton, 1977) indicate that alterations in the autonomic nervous system as a result of endurance training may be responsible for the decrease in resting heart rate regardless of alterations in venous return or inotropy.

Blocking the vagus nerve with atropine caused a greater acceleration of the resting heart in nontrained rats than in trained rats. These results suggest that training 1) increased the non-neural acetylcholine (Tipton & Taylor, 1965),
2) decreased the sensitivity of the heart to atropine (Lin & Horvath, 1972), or 3) altered the sympathetic/parasympathetic ratio. When Frick, Elovaino, & Somer (1967) administered atropine sulfate to both athletes and untrained subjects, resting heart rate was markedly accelerated by the blockade. However, the athletes' per cent increase in heart rate above resting levels were greater than the untrained, indicating that the resting bradycardia of the trained individual might be attributed to increased parasympathetic tone inhibiting sympathetic influences. In contrast, Lin et al. (1972), using control and trained rats, calculated the heart rate response to atropine sulfate and propranolol hydrochloride. The results indicated both systems were reduced by training, but the sympathetic tone had been reduced to a greater extent. It was concluded that resting bradycardia results from greater reduction of sympathetic tone than parasympathetic tone.

Other studies have used the simultaneous blockade of the parasympathetic and sympathetic systems (i.e., intrinsic heart rate) to study the effects of dynamic exercise training on resting heart rate. Studies involving human subjects (Sutton, Cole, Gunning, Hickie, & Seldon, 1967) and rats (Sivgardsson, Svanfeldt, & Kilbom, 1967) report an increased aerobic capacity accompanied by a decreased intrinsic heart rate following training. This lowering of intrinsic heart rate suggests that mechanisms other than changes in the sympathetic
and parasympathetic tone may likewise play a role in training bradycardia. However, when the intrinsic heart rate of rats was studied by Penpargkul and Scheuer (1970), no significant difference existed between the trained and untrained.

**Exercise heart rate responses.** Endurance training results in lower heart rates at both submaximal workloads and maximal workloads (Astrand *et al.*, 1977). This classical training response has been explained as an increase in stroke volume, improving the effectiveness of the oxygen transport system (Ekblom, 1969). This increased effectiveness has been attributed to an increased myocardial contractile force or an increase in ventricular cavity (Matthews & Fox, 1976, pp. 277-291). However, Clausen *et al.* (1970) found that after training, coronary patients showed a reduction in heart rates during moderate workloads, without increases in stroke volume. This finding suggests that the classical mechanism explaining changes in heart rate is not the predominant reason and that peripheral feedback factors may be involved in the training effect on heart rate.

Investigations comparing the heart rate response of trained and nontrained muscles during exercise (Clausen *et al.*, 1970); Clausen, Klausen, Rasmussen, & Trap-Jensen, 1973) suggest that two mechanisms contribute to reduced exercise heart rate after a training period. The first mechanism links the fall of exercise heart rate to the response of the
nontrained muscles to exercise. Heart rate reduction during exercise with nontrained muscles is likely to result from a direct training effect on the heart or other central adaptations. The extent of this effect apparently depends on the cardiac output, stroke volume, and absolute oxygen uptake attained during the training sessions (Clausen, 1977). The exact nature of this systemic contribution to the reduction in heart rate after training is unclear. At rest the balance between sympathetic and parasympathetic activity is dominated by the parasympathetic system (Tipton et al., 1965). During exercise this inhibitory effect is reduced as workloads are increased (Robinson, Epstein, Beiser, & Braunwald, 1966; Ekblom, Kilbom, & Soltysiak, 1973). Reduction in heart rate with nontrained skeletal muscles during exercise is less pronounced the heavier the workloads (Clausen et al., 1973), suggesting that the aspect of bradycardia induced by training is caused by an increased vagal tone on the sinus node (Clausen, 1977).

The second mechanism links the reduction of exercise heart rate during exercise of trained muscles to parallel proportional decrease in splanchnic-hepatic blood flow. This suggests that training reduces the sympathetic outflow to the heart and visceral organs by a common mechanism (Clausen et al., 1973). It is generally accepted that with increasing workloads, sympathetic drive predominates over
parasympathetic drive (Clausen, 1977). Pharmacological studies before and after training suggest reduced sympathetic drive after training (Frick et al., 1967; Brundin & Cernigliaro, 1975). Evidence for less sympathetic activation or reduction in the responsiveness of the beta-1 receptors of the heart is incomplete. A recent swimming and running study with rats by Harri & Narvola (1979) support both an increase and reduction of sympathetic influence after physical training. This suggests that the system is altered; however the final mechanisms have not been clarified. Research in this area is plagued by indirect assessment and studies in which dose-response principles have not been followed. However, it would appear that dynamic exercise training reduces the sympathetic outflow to the heart and visceral organs by a common mechanism.

Effects of Age on Heart Rate

In man and experimental animals heart rate at rest \( (HR_{\text{rest}}) \) and maximum exercise heart rate \( (HR_{\text{max}}) \) decrease with age (Corre, Cho, & Barnard, 1976; Astrand et al., 1976). At present it is not known whether the decrease is due to changes in neural influence or changes in the sino-atrial cells, altering the intrinsic heart rate. Several researchers have investigated structural changes within the heart occurring as a result of the aging process. The amount of heart collagen increases with age and alters the elastic
properties of the tissue (Lenkiewicz, Davies, & Rosen, 1972). This increase of collagen is paralleled by a decrease of muscle fiber (Davis & Pomerance, 1972), which in turn influences the swelling ability of the heart (Kohn & Rollerson, 1959). There is also an infiltration of fat about and in the sino-atrial node (Lev, 1954). These gross structural changes alter the myocardial cell contraction efficiency with age and therefore may alter sino-atrial cell function.

Jose (1966) provided researchers with a procedure for isolating the heart from the neural sympathetic and vagal stimuli (autonomic blockade). Propranolol and atropine were given together, producing a state closely resembling cardiac isolation by surgical methods. The heart rate during such pharmacological isolation was termed intrinsic heart rate, and Jose found it had a reproducible value. He found that with age the intrinsic heart rate declined. Corre et al. (1976), while investigating the mechanisms responsible for the decreased $HR_{max}$ due to training and age in rats, used Jose procedures. They found that during autonomic blockade, $HR_{rest}$ and $HR_{max}$ decreased with maturation of the rat. Both studies show that intrinsic changes in the heart cells occur with age. The mechanisms responsible for these changes remain unexplained, although it has been suggested that the ionic balance at the myocardial membrane
plays an important role (Jose & Stitt, 1969). However, as parasympathetic and sympathetic tone alter this membrane potential, the alterations in tone due to aging and exercise may likewise produce change.

In addition to structural changes in the intrinsic pacemaker, Adolph (1967) in studying the rat heart reported changes in the intrinsic pacemaker's sensitivity to pharmacological agents, and changes in tonic influences which affect its activity. He found that sympathetic prodding predominates over parasympathetic restraint in the rat fetus and infant, but not in the rat adult. Sympathetic impulses begin a day or two before birth, while parasympathetic do not start until the 16th to 20th day of postnatal age. Conway (1970) and Conway, Wheeler, & Sannerstedt (1971) found that propranolol (a beta adrenergic antagonist) reduced heart rate and cardiac output during exercise, but the effect was less in older subjects. This suggests that the sympathetic drive to the heart during exercise declines with age, which agrees with recent findings (Yin, Spurgeon, Greene, Lakatta, & Weisfeldt, 1979) that demonstrate an age-associated decline in the concentration of beta-receptors mediating heart rate. These studies suggest diminished sympathetic influence at rest and during exercise with increasing age. However, a recent study (Yin, Raizes, Guarnieri, Spurgeon, Lakatta, Fortuin, & Weisfeldt, 1978) involving humans has shown that the old heart is more dependent on the sympathetic system
during stress than the young heart. Therefore, although sympathetic activity may decline with age, its importance appears critical to the aging heart.

In addition to changes in the sympathetic system, the vagal affect on the cardiovascular system likewise appears to be altered with age. Frolkis, Bezrukov, & Shevchuk (1975) postulate that the cardiovascular system of old animals appears to be more sensitive to acetylcholine, possibly due to the decreased intensity of acetylcholine hydrolysis. However, the reactive capacity of the system to cholinergic stimulation declines with age, suggesting a decrease in the total number of cholinergic receptors or alteration of their properties, or both (Frolkis, Shevtchuk, Verkhratsky, Stupina, Karpova, & Lakiza, 1979). Respiratory sinus arrhythmia has been shown to be an excellent test of vagal tone (Katona & Jih, 1975), and its decrease with age is documented. This study indicates a possible decrease in vagal tone or diminishment of vagal control with increases of age.

Previous investigations of the aging affects on heart rate control have found many possible explanations for the decrements observed in $HR_{\text{rest}}$ and $HR_{\text{max}}$. Structural changes which alter cardiac efficiency and possibly reduce intrinsic heart rate, are complicated by alterations within the autonomic nervous system. It appears that aging modifies autonomic balance by decreasing vagal tone and the sympathetics' reactive capacity.
Non-Invasive Determination of Heart Rate Control

Although present research findings strongly suggest that endurance training has a positive effect on the middle-aged cardiovascular system, the mechanisms of these effects are unclear. There appears to be shifts in the aging cardiovascular system which alter the normal adaptability process to exercise. Therefore, the study of aging as it affects heart rate control as a factor in the cardiovascular adaptability process to endurance training, raises many questions (Table I).

### TABLE I

THE EFFECT OF AGING ON THE ADAPTATIONS OF THE CARDIOVASCULAR SYSTEM TO ENDURANCE TRAINING

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Young</th>
<th>Middle-Aged</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximal Oxygen Consumption</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Maximal Cardiac Output</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Maximal Heart Rate</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Maximal Stroke Volume</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Left Ventricle Contractility</td>
<td>++</td>
<td>??</td>
</tr>
<tr>
<td>Maximal Total Peripheral Resistance</td>
<td>--</td>
<td>+</td>
</tr>
<tr>
<td>Exercise Muscle Blood Flow</td>
<td>++</td>
<td>-</td>
</tr>
<tr>
<td>Resting Heart Rate</td>
<td>--</td>
<td>??</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>--</td>
<td>??</td>
</tr>
<tr>
<td>End Diastolic Volume</td>
<td>++</td>
<td>??</td>
</tr>
<tr>
<td>End Systolic Volume</td>
<td>--</td>
<td>??</td>
</tr>
<tr>
<td>Vasomotor Response</td>
<td>??</td>
<td>??</td>
</tr>
<tr>
<td>Parasympathetic Control</td>
<td>++</td>
<td>??</td>
</tr>
<tr>
<td>Sympathetic Control</td>
<td>--</td>
<td>??</td>
</tr>
</tbody>
</table>

+, Increase; ++, Large Increase; -, Decrease; --, Large Decrease; ??, Questionable or Unknown.
A large portion of the heart rate control research involves the use of autonomic drugs and animal models, both of which present interpretation problems. An autonomically induced response could reflect either alterations in the effector organs sensitivity, or changes in the nervous control. Standardization of pharmacological dosage to insure blockade is not presently available, nor is the assurance that total blockade occurs after injection of agents. Stroke volume does not increase with exercise in the rat (Popovic, Kent, Mojovic, Mojovic, & Hart, 1969) and it has been concluded (Barnard, Duncan, & Thorstensson, 1974) that the heart rate response to exercise of a rat is different from that found for humans. Therefore, it appears that research involving the animal models and pharmacological agents must be carefully evaluated.

There is no doubt that heart rate changes occur with increasing age and dynamic exercise training. However, present research does not clearly define the mechanisms involved, and unfortunately, the majority of the research involves techniques and methods presenting interpretation problems. As a result of the prevailing problems of interpretation recent studies have used the cardiovascular response to localized cold stressors as a non-invasive determinant of autonomic activity. Le Blanc (1966) suggests that these varied responses can be used to classify individuals according to their sympathetic or parasympathetic predominance.
A hand immersed in cold water will increase both arterial blood pressure and heart rate, indicating activation of the sympathetic nervous system (LeBlanc, Dulac, Côté, & Girard, 1975). Hines and Brown (1932) were the first to use the cold hand pressor test. They used it as an investigative tool in identifying patients who would probably suffer from hypertension. Systolic and diastolic blood pressure data were collected on over 2000 individuals, providing information which was transcribed into vascular reactivity charts (Hines, 1940). These studies were expanded by Thacker (1940), who added the dimension of time, suggesting that both the hypertensive and hypotensive subject had a slower reaction than that observed for a normatensive subject.

The face immersed in cold water will increase arterial blood pressure, but heart rate will decrease, indicating activation of the parasympathetic nervous system (LeBlanc, 1975). This striking phenomenon first observed in diving animals, has aroused the scientific curiosity of many investigators. The bradycardia response can also be elicited by simple apnea (Oldridge, Heigenhauser, Sutton, & Jones, 1978); however, it has been shown that the response is increased with facial immersion (Song, Lee, Chung, & Hong, 1969) and is inversely proportional to the water temperature (Kawakami, Natelson, & DuBois, 1967). Many theories have been
advanced to explain the bradycardia response, from neural reflex theories (Irving, 1963) to mechanical theories (Craig, 1963). Song et al. (1969) suggest a multifactor theory, combining aspects of both the mechanical and the neural theories. Recent work by Caputa and Cabanac (1979) suggests that the bradycardia is produced by selective brain cooling via the ophthalmic veins. Therefore, there is little agreement in the literature on the mechanisms causing the vagal response elicited by facial immersion.

**Fitness level and the cold pressor tests.** The relation of fitness to cold pressor test responses has been studied by several investigators. Le Blanc, Côté, Dular, & Dulong-Turcot (1978) found reduced increases in blood pressure during the cold hand test with increases in VO$_2$max $r = -.59$, $p < .01$. This suggests a reduced sympathetic drive, or a diminished response to cold stress due to decreased sensitivity in trained individuals. Baun and Raven (1979) observed a trend toward a lower heart rate response for high fit individuals, indicating greater vagal tone. However, similar investigations using the heart rate response to facial immersion produced conflicting results. Bove, Lynch, Connél, & Harding (1968) found diving bradycardia was enhanced following increases in aerobic capacity; however, the Harvard Step Test was used to measure physical improvement prompting questions concerning the actual degree of improvement. Many
other investigations (Stromme, Kerem, Elsner, 1970; Stromme, & Ingjer, 1978; Oldridge et al., 1978; Bergman, Campbell, & Wildenthal, 1972) have found no relation between facial immersion and fitness level.

**Aging and the cold pressor tests.** The effect of aging on the cold pressor response has only recently been investigated. Le Blanc et al. (1978) found age to be positively related, $r = .51$, $p < .01$, to the blood pressure response to the cold hand test. However, the heart rate response caused by the cold hand test was lessened with increases in age. These results indicate possible changes in the sympathetic branch of the autonomic nervous system. Lakatta (1979) suggests age-associated changes in the chronotropic responsiveness to catecholamines, possibly due to the decline in the concentration of beta-receptors (Yin et al., 1979).

In the cold face test Le Blanc et al. (1978) observed significantly lower heart rate responses in the older individuals. Frolikis et al. (1975) suggests that there is an increase of the sensitivity of the cardiovascular system to acetylcholine with increasing age, thus the lower responses to the cold face test. Therefore, aging appears to cause sensitivity changes within the autonomic nervous system, by diminishing receptor sites or altering the transmitters, or both.
The use of cold pressor tests to analyse the effects of age and fitness level on autonomic control of heart rate is in its infancy. LeBlanc's research (1978, 1979) has shown that the tests are credible as investigative non-invasive tools; although, presently only cross-sectional data has been collected providing a base for further investigations. This work needs to be replicated to insure credibility, and the experimental design needs to be altered to insure that the interacting effects of age and fitness level can be determined. Unfortunately, a cross-sectional design does not provide information concerning the effects of endurance training on autonomic control of heart rate. Therefore, the need for a longitudinal study using these non-invasive tests is imperative to our understanding of the effects and interactions of endurance training, age, and fitness level on autonomic control of heart rate.
Chapter III

Methods

The investigation was designed as a longitudinal study of the effects of endurance training, fitness level, and age on the autonomic control of heart rate. Subjects were divided into two distinct age groups, then further dichotomized into groups composed of high and low fitness levels. Fitness level was initially determined by a VO$_2$ max stress test. Fitness determinations were followed by the cold pressor tests, which served as determinants of autonomic activity. After these baseline measurements of fitness level and autonomic control of heart rate had been collected, each individual was given an exercise prescription based upon his initial fitness level. At the end of a 10 week endurance training period the initial tests were readministered. Data collected were analyzed by a three-way analysis of variance with repeated measures. The purpose of the analysis was to evaluate the possible changes in the main effects that could be attributed to the exercise training.

Subjects

Forty-one healthy non-smoking male volunteers between the ages of 18 and 55 years were recruited from the student and faculty population of North Texas State University and
the city of Denton, Texas. All volunteer subjects were informed of the purpose, protocol, and experimental procedure to be followed before acceptance as experimental subjects. Each subject signed an informed consent form which had been approved by the NTSU/TCOM Advisory Committee for Research Involving Human Subjects (Appendix 1). Before participation in the study potential subjects underwent a thorough medical screening examination, which included a medical history questionnaire (Appendix 2) and an interview examination with a consulting physician.

The clinical examination included a 7-lead resting electrocardiogram (ECG), and a Bruce symptom-limited maximal stress test, with continuously monitored blood pressure and V4 ECG. Subjects were only selected as participants if no evidence of clinical cardiovascular or pulmonary disease was found.

Thirty-two of the men completed the 10-week self report endurance training program. Two did not complete because of recurring injuries, and three moved before completing the program. The remaining four subjects were lost through normal attrition problems, with the greatest losses being noted in the middle-aged group. The remaining 32 male volunteers were divided into two distinct age groups. The young group (Y) consisted of 20 volunteers between the ages of 18 through 30 years. The middle-aged group (MA) consisted
of twelve volunteers between the ages of 35 to 55 years. These groups were further dichotomized into equal-sized fitness level groups, based upon the individual maximal oxygen consumptions within each age group. Individuals with a VO\textsubscript{2}max above 50 ml.kg.min\textsuperscript{-1} in the Y group were defined as H, with those below 50 ml.kg.min\textsuperscript{-1} being defined as L. In the MA group individuals with a VO\textsubscript{2}max above 43 ml.kg.min\textsuperscript{-1} were defined as H, and those below 43 ml.kg.min\textsuperscript{-1} as L.

The YH group consisted of 12 individuals with an average VO\textsubscript{2}max before training of 58.67 ml.kg.min\textsuperscript{-1} (SD ± 6.0) which was significantly greater than the eight YL with an average VO\textsubscript{2}max of 44.13 ml.kg.min\textsuperscript{-1} (SD ± 3.92). There were eight MAH individuals with an average VO\textsubscript{2}max of 48.33 ml.kg.min\textsuperscript{-1} (SD ± 5.15), and four MAL individuals with an average VO\textsubscript{2}max of 40.32 ml.kg.min\textsuperscript{-1} (SD ± 2.82).

Procedures

**Measurement of maximal aerobic capacity (VO\textsubscript{2}max).** Maximal oxygen uptake (VO\textsubscript{2}max) was determined by the Bruce protocol, as described earlier for the screening treadmill test (Åstrand, 1960). The session began with a five-minute rest period with the subject seated in a chair on the treadmill, breathing through a mouthpiece connected to an open-circuit calorimetry system. Resting oxygen uptake, carbon dioxide production, ventilation, and heart rate were recorded continuously. Following the rest period the
subject was instructed to stand and then start walking at a 1.7 mph pace with a 10% grade. At three-minute intervals the grade and speed were increased by 2% and 0.5 mph, respectively. The test was terminated when the subject was no longer motivated to continue, if any significant abnormal changes occurred in the ECG or blood pressure, or on the decision of the supervising staff physiologists or physician. A five-minute recovery period followed completion of the test.

Ventilation volumes were determined minute by minute by a Dry Gas Meter CD-4 (Parkinson-Cowan). A continuous recording of the oxygen and carbon dioxide content in the expired air was obtained with an oxygen analyzer (Beckman OM11) and a carbon dioxide analyzer (Beckman Model LB2). All parameters were recorded on a multichannel pen recorder (Soltec 3310), during rest, exercise, and recovery. All analyzers were calibrated against known standard gases (Haldane analysis). The electrocardiogram and heart rate were monitored continuously, as described earlier in the preliminary screening test. The appearance of the subject at the termination of the test, maximum heart rate ($HR_{max}$), and a plateauing of VO$_2$ during the final stages of the test were used as criteria for determining maximum effort (Åstrand, et al., 1976). If a test was considered sub-maximal, the subject was asked to repeat the test on
another day. Metabolic calculations were made according to formulae reported by Consolazio, Johnson, and Pecora (1963).

Measurement of heart rate, blood pressure, and body composition. Resting heart rate and blood pressure were measured after a rest period on a non-treadmill test day. Body weight was recorded to the nearest quarter of a pound. Anthropometric determination consisted of seven skinfold sites, measured with a Lange skinfold caliper. All skinfold measurements were taken on the right side of the body, and recorded to the nearest 0.5 mm. Jackson and Pollock's (1978) prediction equation was used to compute body fat.

Measurement of sympathetic and parasympathetic activity. The cold pressor tests were given on two separate days following the physician's examination and the VO$_2$max test. Both tests were given with the subject seated in a booth which separated him from the testing and recording equipment. An equipment cart containing the water baths was positioned according to the test and positioning needs of the subject. The cold water bath was kept between four and six degrees centigrade, and was monitored by a thermister probe (Yellow Springs Instruments). The second bath contained water at room temperature, and was used as a recovery medium to reduce the cold stress after completion of the hand test. Testing sessions consisted of rest, cold stress,
and recovery. With a Narco Physiograph, heart rate and ventilation were measured continuously during the last five minutes of rest, two minutes of stress, and three minutes of recovery. Because of the rapidity of heart rate changes, heart rate was averaged at six second intervals. Blood pressure was recorded automatically (Narco PE-300) every 30 seconds during the 10 minutes of recorded heart rate. The Narco PE-300 was calibrated before and after each test with a 100 mmHg signal produced by the PE-300.

**Cold hand pressor test.** During the cold hand pressor test (CH) the subject placed his hand (up to his wrist) into the cold bath for a period of two minutes. After completion of the two-minute stress period the subject was instructed to place his hand into the second bath for the three-minute recovery period.

**Cold face pressor test.** The cold face pressor test (CF) was executed with the aid of a nose plug, swimming goggles, and a snorkel. A pneumatachograph recording was used to monitor ventilation to insure apnea was not a confounding factor. A preparatory test using water at room temperature was conducted to introduce the subject to the equipment and help him gain confidence in his ability to complete the facial immersion test. During the rest, stress, and recovery periods the subject breathed through the snorkel. After sufficient rest following the preparatory test the subject was instructed to bend slightly
forward and place his face into the cold bath, completely submerging the swimming goggles to the level of the ears. After two minutes of cold stress, instructions to return to the original position were given.

**Endurance training program.** The endurance training program was a walk-jog program, consisting of a minimum of three 30-minute bouts a week for a 10-week period. A training intensity of 70% to 75% heart rate range (heart rate range = heart rate maximum - heart rate rest) above heart rate rest was used to determine the proper training intensity (Lamb, 1978, p. 243). Maximum heart rate was considered the highest value attained on the stress test. The initial training sessions were individualized and supervised, thereby insuring that each subject began his exercise prescription at the proper level. Each subject was instructed in the technique of determining appropriate training heart rate by the palpation technique. Exercise was monitored through a self-report card (Appendix 3) which was issued by and returned to the investigator each week. Throughout the individual's training program the self-report card was used to monitor the progress. As the degree of fitness improved, the subjects were encouraged to increase the intensity and duration of their work, so that a 70% to 75% heart rate range above heart rate rest was consistently reached during the endurance training. Thus, the combination
of the monitored self-report cards and bi-weekly subject-investigator phone conferences provided the control necessary to maximize the probability of a training effect.

Experimental design and statistical analysis. $\text{VO}_{2\text{max}}$ was evaluated before and after the 10-week endurance training program. The CH and CF tests succeeded both aerobic endurance evaluations. A three-way (2x2x2) factorial analysis of variance (ANOVA) with repeated measures (see Figure 1) was used to analyze the statistical differences from pre- to post-training across the independent variables of age and training level (Winer, 1971, p. 307). Pearson correlation was used to test the relationship between the responses to the CH and CF test, and the relationship of these pressor test responses to age, fitness level, and endurance training. Fisher's $z$ Transformation of $r$ was used to test the differences between the pre-and post-Pearson correlations (Glass & Stanley, 1970, pp. 308-309). Statistical analysis was performed on the North Texas State University IBM 360/50 computer system. North Texas State University Statistical Library Packages and the Statistical Packages for the Social Sciences (SPSS) were used for the individual programming. These statistical treatments were used to help make decisions regarding the rejection or acceptance of the hypotheses.
Age  Young = 18 - 30 years  
      Middle-Aged = 35-55 years  

Fitness Level  Young Low = <50 ml.kg.min⁻¹  
               Middle-Aged Low = <43 ml.kg.min⁻¹  
               Young High = >50 ml.kg.min⁻¹  
               Middle-Aged High = >43 ml.kg.min⁻¹  

Fig. 1--Experimental Design
CHAPTER IV

RESULTS

Metabolic and Body Composition Measurements

Training significantly increased the overall \( \text{VO}_2 \text{max} \) of the groups \( F(1,496) = 91.2, \ p < .001 \), eliciting a mean group improvement of 11.6%. The YL group experienced the largest improvement, while the YH group improved the least (see Figure 2). Overall increases in \( \text{VO}_2 \text{max} \) were negatively correlated to the initial \( \text{VO}_2 \text{max} \) testing results \( r = -0.41, \ p < .001 \) indicating that the least fit individuals improved the most as a result of training. In addition the differences noted between the \( \text{VO}_2 \text{max} \) of the Y and MA groups were significant \( F(1,672) = 13.06, \ p < .0001 \) as were those between the H and L groups \( F(1,1496) = 29.06, \ p < .0001 \).

A summary of the changes in total body weight and percentage of body fat is presented in Table II. After training, significant reductions in total body weight \( F(1,27) = 4.69, \ p < .03 \) and per cent fat \( F(1,45) = 17.03, \ p < .0003 \) were observed (see Table II).

Resting Heart Rate

Resting heart rate (RHR) measured in the sitting position during the initial phase of the cold hand and cold face test decreased an average of 5% after training. The changes in RHR are outlined in Figure 3, p. 36.
Fig. 2--Group VO$_2$$_{\text{max}}$ Increase After Training (Group Means $\pm$ SEM)
TABLE II

BODY WEIGHT AND PERCENT FAT MEASUREMENTS
PRIOR TO AND FOLLOWING TRAINING
(GROUP MEANS ± SEM)

<table>
<thead>
<tr>
<th>Group</th>
<th>Before Training</th>
<th>After Training</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Body Weight (kg)</td>
<td>Per Cent Fat (%)</td>
</tr>
<tr>
<td>Young</td>
<td>75.1±2.55</td>
<td>11.4±1.40</td>
</tr>
<tr>
<td>Middle-Aged</td>
<td>77.5±2.67</td>
<td>17.4±1.19</td>
</tr>
<tr>
<td>High</td>
<td>74.8±2.16</td>
<td>12.5±1.51</td>
</tr>
<tr>
<td>Low</td>
<td>77.9±3.90</td>
<td>16.3±1.62</td>
</tr>
<tr>
<td>Overall</td>
<td>76.0±2.01</td>
<td>14.0±1.17</td>
</tr>
</tbody>
</table>
Subjects had significantly \[ F(1,66) = 4.71, p < .03 \] lower RHR's in the post-training cold hand test.

The RHR of the Y group oscillated around a mean RHR value; however, this was not evident in the MA group. Using standard deviation (SD) of the mean RHR as a measure of the variance of this oscillation, a significant difference was found between the variance of the RHR of the Y and MA during the cold hand \[ F(1,10) = 5.86, p < .02 \] and cold face testing \[ F(1,10) = 4.59, p < .04 \]. Correlational analysis of age and the variance measurements showed that age is significantly related to the oscillation observed at rest during the cold hand \[ r = -.55, p < .001 \] and the cold face test \[ r = -.44, p < .006 \]. Following training, the degree of oscillation increased some 15% and 34% in the Y and MA respectively. Further analysis showed that there was no longer a significant difference between the variance of the RHR of the Y and MA during the cold hand test \[ F(1,11) = 3.41, p < .07 \] however, the differences were maintained in the cold face test \[ F(1,14) = 4.59, p < .04 \]. Furthermore, there were no significant changes from pre-to post-training observed in the relationship between age and variance measures. In order to determine the relationship of age and VO\(_2\)\(_{\text{max}}\) to the variance of RHR, partial correlational techniques were used. Holding VO\(_2\)\(_{\text{max}}\) constant, the relation between age and RHR variance was \( r = -.48, p < .01 \), which was only...
slight change from the original correlation $r = -0.55$, $p < 0.001$.

**Cold Hand Pressor Test Heart Rate Response**

The hand immersion test caused an expected increase in heart rate (HR) (see Figure 3). The increase (DMAX) was measured by subtracting the individual's average RHR observed during the initial four minutes of the resting phase, from the individual's maximal heart rate observed during the cold hand stress phase. It is evident from Table III (p.39) that there was a significant increase in heart rate when the hand was immersed in cold water. The overall group average increased 20% (or 14.1 beats/min), however there was a five beat/min difference between the Y and MA groups response and a 10 beat/min difference between the L and H groups heart rate response (see Table III). However, neither of these group differences in response were significant.

The overall correlation between DMAX and age obtained prior to training was weak [$r = 0.15$, $p < 0.05$], and was shown to reflect the equal but opposite relationship that existed between the heart rate response and age of the Y and MA groups. Before training analysis of the Y groups DMAX was significantly correlated with age [$r = +0.41$, $p < 0.03$], while the DMAX of the MA group was inversely correlated with age [$r = -0.76$, $p < 0.003$]. Following training the DMAX of the MA group was positively correlated with age (although
Fig. 3--Alterations observed in the group mean heart rate response during the cold hand test before and following training.
not significant), and the relationship was significantly
different from that attained prior to training [z = -2.45,
p < .05] (see Figure 4).

No significant differences existed between the response
(DMAX) of the L and H group; however, there was a trend
towards the high fit individuals having less of a response
than the low fit individuals (see Table III). The overall
correlation between DMAX and VO2max was r = .04, p < .10.

Average maximum heart rate was 83 beats/min., and was
reached at approximately 48 seconds into the stress phase
of the test. Further analysis revealed that there was a
significant difference between the time to peak heart rate
(TMAX) of the L and H groups, F(1, 98) = 4.92, p < .03, with
the L group responding faster (42 seconds) than the H group
(54 seconds). These results evidenced a positive correlation
[r = .55, p < .001] between TMAX and VO2max. Although there
were no differences between the Y and MA TMAX response, a
negative correlation was found between TMAX response and
age [r = -.41, p < .01], suggesting that with age the time
to peak heart rate decreased.

The rate of response (BTSECXM) was calculated by the
following equation (DMAX divided by TMAX/60) and had a
range from .0025 beats/sec/sec to .0075 beats/sec/sec.
There was no significant difference between the Y and MA
or the H and L groups rate of response. However, age was
found to be positively correlated with rate of response
Fig. 4--The relationships between age and the increase in heart rate during the cold hand test prior to and following training.
### TABLE III

HEART RATES OBTAINED DURING THE COLD HAND TEST
PRIOR TO AND FOLLOWING TRAINING
(GROUP MEANS ± SEM)

<table>
<thead>
<tr>
<th>Group</th>
<th>Before Training</th>
<th></th>
<th>After Training</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Resting HR</td>
<td>Max HR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diff</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Resting HR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Max HR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Diff</td>
</tr>
<tr>
<td>Young</td>
<td>71±2.75</td>
<td>85±2.98</td>
<td>13±1.61</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>70±2.06</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>78±4.36</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>12±1.61</td>
</tr>
<tr>
<td>Middle-Aged</td>
<td>72±3.18</td>
<td>81±3.18</td>
<td>18±3.75</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>66±2.02</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>83±3.75</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>14±2.31</td>
</tr>
<tr>
<td>Low</td>
<td>76±3.92</td>
<td>92±5.13</td>
<td>16±3.02</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>71±2.41</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>86±3.92</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>15±2.71</td>
</tr>
<tr>
<td>High</td>
<td>69±2.29</td>
<td>83±2.98</td>
<td>14±2.29</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>68±2.06</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>75±4.36</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>12±1.38</td>
</tr>
<tr>
<td>Overall</td>
<td>71±2.19</td>
<td>87±2.37</td>
<td>15±1.83</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>69±1.64</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>79±2.92</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>13±1.28</td>
</tr>
</tbody>
</table>
\( r = .36, \ p < .02, \) whereas the level of training \((VO_2\text{max})\) was not significantly correlated.

**Cold Hand Pressor Test Blood Pressure Response**

As shown in Figure 5, cold hand immersion causes systolic blood pressure to rise (DHBP). This increase was measured by subtracting the mean of the five minute resting systolic blood pressure from the peak systolic blood pressure observed during the cold stress phase. In general there was an overall increase of 5 mmHg in resting \(F(1,308) = 7.5, \ p < .01\), and peak systolic blood pressure \(F(1,775) = 11.3, \ p < .002\), following training. The MA peak systolic response was found to be significantly greater than the Y response \(F(1,1023) = 6.63, \ p < .01\) (see Figure 5). This difference was corroborated by the positive relationship between age and DHBP \(r = .42, \ p = < .01\). Age likewise was related to the rate of the response \((\text{rate} = \text{time to peak response/DHBP})\). Rate of response increased with increases of age \(r = .59, \ p < .001\), and this relationship indicated significant differences between the Y and MA rate response \(F(1, 08) = 4.26, \ p < .04\).

In contrast to the positive relationship of age on the systolic blood pressure response, \(VO_2\text{max}\) was found to be negatively related to DHBP \(r = -.43, \ p < .01\). Further correlational analysis revealed that the higher the \(VO_2\text{max}\), the greater was the time to peak systolic pressure response \(r = .30, \ p < .05\) and the slower the rate of increase in blood pressure \(r = -.58, \ p < .001\).
Fig. 5--Increases in systolic blood pressure during the stress phase of the cold hand test (Group Means ± SEM).
Cold Face Pressor Test Heart Rate Response

Immersion of the face into cold water elicits the diving reflex which results in bradycardia. The HR decrease (DMNF) was measured by subtracting the minimum HR observed during the stress phase, from the initial four minute average RHR observed prior to the cold face stress. No significant differences were found between the Y and MA, or the H and L groups; however, as shown in Figure 6, the MA group's response was greater than the Y group's response and the H responses were greater than the L responses. After training, the overall RHR decreased 4.75% (p > .05). The minimally attained HR during cold stress was further decreased 6% (p < .05). Correlations between DMNF and age indicated an inverse relationship between the YH and YL groups. It is evident from the data in Table IV that as age increases in the YH group, DMNF decreases, while in the YL group, an age increase produces a decreased response. Age does not significantly relate to the overall DMNF, nor were there significant correlations found between age within the MA group (see Table IV). When controlling for age, the variance in RHR was shown to be correlated with DMNF (r = .36, p < .04). However, DMNF was found to be negatively correlated with VO₂max (r = -.38, p < .02), with the strongest correlation existing in the YL group (r = -.84, p < .007). It was also observed that as RHR increased, DMNF was also increased (r = .48, p < .003).
Fig. 6—Heart rate changes on the cold face tests prior to and following training (Group Means ± SEM).
TABLE IV

RELATIONSHIP BETWEEN COLD FACE HEART RATE RESPONSE PRIOR TO AND FOLLOWING TRAINING

<table>
<thead>
<tr>
<th>Groups</th>
<th>Correlation Coefficients</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before Training</td>
<td></td>
<td>After Training</td>
</tr>
<tr>
<td>Young High</td>
<td>$r = -.44$, $p &lt; .07$</td>
<td></td>
<td>$r = -.64$, $p &lt; .01$</td>
</tr>
<tr>
<td>Young Low</td>
<td>$r = .68$, $p &lt; .04$</td>
<td></td>
<td>$r = .72$, $p &lt; .03$</td>
</tr>
<tr>
<td>Middle-Aged High</td>
<td>$r = .10$, $p &gt; .05$</td>
<td></td>
<td>$r = .35$, $p &gt; .05$</td>
</tr>
<tr>
<td>Middle-Aged Low</td>
<td>$r = .06$, $p &gt; .05$</td>
<td></td>
<td>$r = .32$, $p &gt; .05$</td>
</tr>
</tbody>
</table>

Cold Face Pressor Test Blood Pressure Response

Cold face immersion caused a significant increase in systolic blood pressure. The average group increase was 28 mmHg, which reflected a 23% increase above resting values. These values correspond to the cold hand mean group increase of 25 mmHg, or a 22% increase above resting values. The difference in increased systolic blood pressure (DFBP) between the Y and MA groups was significant [$F(1,816) = 7.62$, $p < .01$], with the MA response being 6% larger than the Y response. The effect of age was confirmed by the positive correlation that existed between age and DFBP [$r = .33$, $p < .04$]. Table V summarizes relationships between the groups and their maximum blood pressure responses.

Training significantly altered the relationship between $VO_2\text{max}$ and the MA and Y blood pressure response to cold face
TABLE V
CORRELATIONS BETWEEN SYSTOLIC BLOOD PRESSURE AND AGE FOR THE COLD FACE TEST

<table>
<thead>
<tr>
<th>Group</th>
<th>Correlational Coefficients of Systolic Blood Pressure With Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young</td>
<td>( r = .27 )</td>
</tr>
<tr>
<td>Middle-Aged</td>
<td>( r = .46, p &lt; .05 )</td>
</tr>
<tr>
<td>High</td>
<td>( r = -.23 )</td>
</tr>
<tr>
<td>Low</td>
<td>( r = .74, p &lt; .002 )</td>
</tr>
</tbody>
</table>

Prior to training, a positive relationship was observed between the Y DFBP response and \( VO_2^{max} \), while the MA DFBP response with \( VO_2^{max} \) was positive, yet insignificant. Following training both of the relationships became negative and non-insignificant; however, there existed a major alteration from the pre-training correlations (see Table VI).

TABLE VI
CORRELATION COEFFICIENTS AND \( z \)-TRANSFORMATIONS BETWEEN \( VO_2^{max} \) AND CHANGE IN BLOOD PRESSURE DURING THE COLD FACE TEST

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Training Correlations</th>
<th>Post-Training Correlations</th>
<th>( z )-Transformations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young</td>
<td>( r = .43, p &lt; .002 )</td>
<td>( r = -.05, p &gt; .05 )</td>
<td>( z = 2.13, p &lt; .04 )</td>
</tr>
<tr>
<td>Middle-Aged</td>
<td>( r = .21, p &gt; .05 )</td>
<td>( r = -.51, p &lt; .06 )</td>
<td>( z = 2.38, p &lt; .02 )</td>
</tr>
<tr>
<td>High</td>
<td>( r = .05, p &gt; .05 )</td>
<td>( r = .09, p &gt; .05 )</td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>( r = -.51, p &lt; .04 )</td>
<td>( r = -.76, p &lt; .005 )</td>
<td></td>
</tr>
</tbody>
</table>
The time to peak systolic blood pressure (TIMBPF) was significantly increased after training \( F(1, 816) = 7.62, p < .01 \), however, the rate of rise in blood pressure was unaltered.

**Recovery from Cold Hand Pressor Test--Rebound**

Immediately following the completion of cold hand immersion a bradycardia was observed in the recovery period. This rebound effect DMNH was measured by subtracting the minimum heart rate in recovery (MNHR) from the RHR. Figure 7 shows that training increased all of the DMNH responses, with an average increase of 44%.

The rebound effect after training was increased more in the L group than in the H group \( F(1, 273) = 6.56, p < .01 \), and the MA group increased more than the Y group \( F(1, 564) = 15.32, p < .001 \). Both the MA and the L groups rebound response was increased by 100% after training, while the Y group only increased 18% and the H group increased only 9%.

Rebound was found to be negatively related to age \( r = -0.41, p < .01 \); however, this was not related to \( VO_2^{\text{max}} \). In addition, increases in the variance of RHR were found to be positively related to the rebound response \( r = 0.47, p < .003 \).
Fig. 7--Decrease in heart rate recovery phase of cold hand tests associated with training (Group Means ± SEM).
CHAPTER V

DISCUSSION

The purpose of this discussion will be to elucidate findings of this study through an evaluation of data generated from the main effects and the subsequent interactions. Aging modifications of autonomic heart rate control will be presented first, followed by the effects of dynamic exercise training on the autonomic system. Then the interaction of age and dynamic exercise training will be discussed, concluding with the implications supported by the study.

Aging Effects on Autonomic Heart Rate Control

The data suggest that aging causes changes to occur within the autonomic nervous systems and the means by which heart rate is controlled. This alteration is reflected by an increased reactivity and a decreased overall responsiveness within the sympathetic system. Furthermore, vagal activity is markedly attenuated in the middle-aged. Therefore, it appears that a natural consequence of aging is a diminished autonomic control of heart rate.

Previous research (Le Blanc et al., 1978) has shown that age does not modify the response to cold pressor test except in subjects over 50 years of age. However, Le:Blanc's analysis was limited by his experimental design and chosen population, restricting closer examination of the aging
effect on heart rate control. Subjects in the aforementioned study were divided into two distinct age groups (young, 20 - 40 years, and middle-aged, 53 - 60 years); however, the arrangement removed a 13-year period from his analysis. Although he did find age to be a factor, accounting for the aging continuum would have better clarified the issue of aging effects (Le Blanc et al., 1978). The present investigation was designed to overcome this limitation through distinct, more inclusive age divisions, and through more sophisticated statistical analysis.

In contrast, the present investigation identified the effect of aging on the response to the cold hand test prior to 50 years of age. The absolute change in the heart rate response to the cold hand pressor test did not differ between the two age groups. However, further analysis showed that as age increased in the young group, the sympathetic response to the cold hand stress increased, whereas in the middle-aged group an increase of age decreased the sympathetic response to the cold hand stress. This reversal process appears to occur between the ages of 30 and 40 years, much earlier than LeBlanc postulated.

Yin et al. (1979) suggest that this age-associated decrease of the sympathetic chronotropic response is a result of a decline in the concentration of beta-receptors mediating heart rate response. While Corre and Barnard
(1976) proposed intrinsic alterations that have a chronotropic reducing effect on the SA node with maturation. Their work is supportive of the early work of Jose (1966), who after isolating the heart from its neural influences (intrinsic heart rate) found a progressive heart rate decline with increases of age.

Paradoxically, as the sympathetic response (measured as the absolute change in heart rate) is reduced with increases in age, the acceleration (a measure of reactivity) of the response appears to increase. This possible increased reactivity of the sympathetic system with age has not been reported in previous cold stress studies. Therefore, its physiological basis can only be conjectured upon, using basic principles from the autonomic nervous system literature. Gellhorn (1957), using humans, and Adolph (1967), employing rats, have shown that the tonic state of the autonomic nervous system influences heart rate response. During parasympathetic tuning the responsiveness of the sympathetic system is diminished. The degree of parasympathetic cardiac control can be measured by the presence of respiratory sinus arrhythmia, which is highly reflective of both the average respiratory rate interval and the resting heart rate (Katona et al., 1975). In this investigation the variance in RHR is an indication of respiratory sinus arrhythmia, and hence of degree of vagal tone. The middle-aged group's significantly
diminished variance in RHR reflects a decrease in vagal tone. Yin et al. (1978) have findings suggesting that the aged heart is more dependent on the sympathetic nervous activity than the young heart during hemodynamic stress. Combining the results of these studies, and recalling the arguments for a decrease of sympathetic responsiveness with age, it appears that the decreased vagal activity must have resulted in an increased reactivity of the middle-aged sympathetic system. However, the reported diminution of beta-receptors or intrinsic alterations, or both, may have resulted in the lessened sympathetic response that was observed. Early work by Gellhorn (1957) suggests that the action of sympathetic stimuli during a state of sympathetic tuning is to increase both the response and reactivity of the system. Finding increased reactivity in the sympathetic system of the middle-aged, concurrent with a reduced response, appears to indicate a state of autonomic imbalance, where the system is being dominated by a weakened sympathetic system.

In contrast to the results of Le Blanc et al. (1978), the data of this investigation suggest that age significantly affected increases in systolic blood pressure response due to the cold stress. Not only did the middle-aged group have larger increases in systolic blood pressure, but their rate to peak blood pressure was significantly faster. Gibbons, Pickering, & Sleight (1971) have shown that increasing age
reduces the gain (sensitivity) of the baroreceptor-heart rate reflex, diminishing the reciprocal alterations in vagal and sympathetic efferent activity which lower heart rate during gradual increases of blood pressure. Kroner (1977) suggests that there are three possible mechanisms involved in the reduction of baroreceptor-induced autonomic activity, these being 1) more difficult recruitment of baroreceptor units, 2) functional impairment of the baroreceptor, and 3) age-related degeneration of the receptor. Although direct observation of the baroreceptor-heart rate reflex was not evaluated in this study, it appears that aging effects on this mechanism are minimal.

The middle-aged groups’ bradycardia response elicited by the cold face test was insignificantly greater than the bradycardia response elicited by the young group. Le Blanc et al. (1978) found significant differences between the middle-aged and young bradycardia response, and suggest two possible mechanisms, these being 1) reduced sympathetic action, and 2) increased vagal activity or number of cholinergic receptor sites, or both. The latter possibility partially agrees with findings of Frolikis et al. (1975), who have shown an increase of the sensitivity of the cardiovascular system to acetylcholine with increasing age. However, as we age, the total number of cholinergic receptors is decreased, and the reactive capacity of the cardiovascular
system is decreased (Frolkis et al., 1979). Therefore, Le Blanc's (1978) increased bradycardia observed in the middle-aged during the cold face test is more likely a measure of increased sensitivity of the vagus to stress, and not a measure of enhanced vagal tone. Thus, aging appears to increase the reactivity of the autonomic nervous system to the cold pressor stress; however, autonomic control of heart rate diminishes with increasing age.

Fitness and Training Effects on Autonomic Heart Rate Control

High levels of fitness (VO\textsubscript{2max}) enhance vagal dominance. The theory of increased vagal tone and a reduction in sympathetic activity as a result of dynamic exercise training is expounded by several prominent researchers (Ekblom et al., 1973; Frick et al., 1967). An autonomic heart rate control system dominated by parasympathetic tone is characterized by lower resting heart rates and reduced responsiveness to sympathetic stimulus. After the endurance training period, resting heart rate was significantly reduced in the present investigation. In the middle-aged group, there was, furthermore, an increased variation of RHR, indicating increased vagal activity (Katona & Jih, 1975).

There were no statistically significant changes in heart rate response that could be attributed to fitness levels on the cold hand test; however, the low fitness group reacted significantly faster to the cold stress. This
increased reactivity of the low fitness group is likewise present in the blood pressure responses to the cold hand tests. Hence, it appears that the low fit group is sympathetically dominated (Gellhorn, 1957), whereas the high fitness group demonstrates vagal dominance, which reduces reactivity and the final response to the sympathetic stimuli. This is clearly evident in the significant changes which occur in the face test blood pressure responses after training, reducing the absolute increases and decreasing the reactivity. Le Blanc et al. (1978) suggest that the reduced responsiveness in the highly fit is probably due to reduced sympathetic activity. However, a recent study (Harri et al., 1979) into the effect of physical training on the sympathetic nervous system found support for both increased and decreased sympathetic influence. The effect of physical training on the sympathetic system is further clouded by research (Sutton et al., 1967; Sigvardsson et al., 1967) indicating diminishment of intrinsic heart rate with increasing levels of fitness. The various arguments for reduced sympathetic responsiveness in the highly fit indicate alterations occur, but the mechanisms producing these changes have not been clarified.

The degree of bradycardia observed in heart rate during the cold face tests was unchanged by the training period; however, the absolute heart rates obtained were significantly
lower. The lower heart rates were a reflection of the lower resting heart rates, not the consequence of an increased responsiveness to the cold pressor stress. Absolute responses to the cold face stress were nearly equal in the low and high fitness groups, although as fitness levels increased the responsiveness was less. These results disagree with Bove et al. (1968), who found increased bradycardia with increases in fitness level, and with other investigators (Oldridge et al., 1978; Stromme et al., 1970, 1978) who report that bradycardia response has no relation to fitness level. It has been consistently shown in previous research (Bergman et al., 1977; Stromme et al., 1978) that the diving reflex is increased during exercise, which supports Finley, Bonet, & Waxman's (1979) recent postulate that heart rate decrement during facial immersion increases with greater steady-state heart rates during exercise. The present investigation extends the findings of Finley et al. (1979), revealing that the pre-stress (resting heart rate) heart rate affects the responsiveness of the diving reflex. This explains the inverse relationships observed between age and the young high and young low heart-rate decrease data. The most fit and most unfit young individuals tended to be the elder of the young group of subjects. Therefore, their resting heart rates were the lowest and the highest. Individuals with high resting heart rates experienced a greater
absolute heart rate decrease in response to cold face stress than individuals with low resting heart rates.

A possible explanation for this increased heart rate decrement with increases of resting heart rate is that a background of sympathetic activity accentuates the vagal inhibition in the face test. Levy (1971) has found that in some instances vagal stimulation is more effective against a background of sympathetic activity than in the absence of sympathetic tone. The increased resting heart rates denote an enhanced sympathetic tone or a decreased vagal tone, or both, which when applying Levy's model would account for the increased heart rate decrements. Therefore, greater heart rate decrement in response to cold face stress does not indicate a more active parasympathetic system, as hypothesized by Le Blanc (1975), although it might provide useful information about the state of autonomic balance preceding the cold stress.

Gellhorn (1957) describes an excellent test to determine the state of autonomic balance. He calls it successive autonomic induction (rebound). It is simply the parasympathetic after-discharge which succeeds a sympathetic stimulation. Gellhorn (1957) found that during a pre-stress state of vagal tuning rebound is augmented; however, in a pre-existing sympathetic imbalanced system the rebound succeeding stress tends to be suppressed.
In the present investigation training enhanced the rebound phenomenon in all groups. The groups which experienced the largest fitness gains (VO₂ max) likewise had significantly increased rebounds. The positive relationship that exists between the rebound phenomenon and the variance in resting heart rate, indicates that it is, perhaps, another means of measuring vagal tone. Therefore, the increased rebound effect indicates that vagal tone has been enhanced through the endurance training program. This implies that high fitness levels are characterized by an autonomic heart rate control dominated by vagal tone. Modifications of the sympathetic response further suggest that alterations have occurred within the sympathetic system which may be a resultant of increased vagal tone weakening sympathetic activity. Therefore, endurance training providing increases in fitness level positively affects autonomic heart rate control and results in the classical reduction of heart rate at rest and during moderate stress.

Interaction Effects

The increased fitness levels attained in this investigation by the middle-aged, young, high fit, and low fit groups provoked alterations within the autonomic heart rate control system. Positive indications of these autonomic changes are seen in the modifications on resting heart rate and the changes which occur on the responses to the cold pressor
tests. After training, variation in resting heart rate was increased in the middle-aged group. This, coupled with the significant increases in the bradycardia observed during recovery of the middle-aged hand test, suggests that the vagal system is trainable in middle-age.

The sympathetic response to the cold hand test was significantly changed in the middle-aged group after training. Unlike the initial cold hand test responses, training appears to heighten the sympathetic response in the middle-aged group, indicating an increase in the responding capacity of the sympathetic system, possibly brought about through changes within the beta-receptors. Therefore, it appears that training restored some of the sympathetic tone which was naturally lost through the aging process.

Aging was positively related to the systolic blood pressure responses elicited by the cold pressor tests. It is generally agreed that increasing stiffness of the large arteries results in an increasing peripheral resistance and blood pressure in the aged. However, after training, the positive relationships observed in the young and middle-aged groups during the cold face test were significantly changed. These observed blood pressure decreases during the cold face pressor test after training reveal possible baroreceptor changes related to decreased peripheral sensitivity to cold stress (Baum, Bruck, & Schwennicke, 1976) or alteration at the baroreceptor.
This investigation has shown that aging diminishes the control which the autonomic nervous system has over heart rate. However, a dynamic exercise program which increases fitness level appears to interrupt this natural degeneration process. The apparent trainability of the vagal system provides a strong incentive for further research into autonomic heart rate control and endurance exercise training. Recent investigations (Kjekshus & Blix, 1977) suggest that patients suffering from acute myocardial ischaemia could be helped by an increased vagally induced bradycardia. Reduction of heart rate is the most effective way of reducing the myocardial oxygen requirements. Therefore, it becomes apparent that the trainability of the vagal system is an important finding and provides a base for future research.

Conclusions

Based upon the findings and limitations of this study, all of the hypotheses were accepted, and the following conclusions appear to be warranted:

1. Aging appears to increase the rate of reactivity, but decreases the magnitude of heart rate response to cold pressor stress. These data suggest that a natural consequence of aging is the diminishment of autonomic heart rate control.

2. Fitness level is negatively related to the rate of heart rate response and rate and magnitude of blood pressure response to sympathetic stimulation during cold hand tests.
This would suggest that individuals of low fitness level appear to have heart rate control dominated by the sympathetic system, while individuals with high fitness levels have a vagally dominated heart rate control system.

3. Endurance training while providing increases in fitness level, prolongs the time to peak blood pressure response in the cold face pressor tests. The vagally dominated responses to cold pressor stress are increased after endurance training. These data suggest that endurance training increases the vagal domination of heart rate control.

4. The interaction of aging, fitness level, and endurance training influences the rate and magnitude of reactivity of the heart rate response to the cold pressor tests. Increased fitness level through endurance training appears to increase the responding capacity of the middle-aged sympathetic system, while providing an increased vagal tone. This suggests that endurance training might alter the manifestation of aging on heart rate control.

Recommendations for Future Research

Future research needs to better clarify the actual mechanisms evoking the vagal bradycardia responses observed during stress phase of the cold face test and the recovery of the cold hand test. This will insure investigators that these responses are appropriate indicators of vagal tone, an unfortunate limitation of this investigation. Although
the noninvasive cold pressor tests are easily administered, researchers should be aware of the anticipatory heart rate problems which interrupt the normal tuning process. The vast amount of data which was collected during this study would have been better handled if it had been recorded initially by a computer storage system. This type of recording system would have allowed a closer look into the problems associated with anticipation. It also might provide a base from which theoretical autonomic control models could be drawn using the cold pressor data.

The increased reactivity of the aging sympathetic system observed in this study raises many interesting questions. Is the aging autonomic system sympathetically imbalanced as proposed by this investigator or is the response observed in the cold pressor stress only a specific case? Further research must first replicate and extend this investigation's findings before these questions can be answered. Utilization of tests specific for determining alteration of baroreceptor sensitivity and vasomotion is essential, while the correlation between the cold pressor test and pharmacoologically induced intrinsic heart rate, age, and fitness level needs to be evaluated. The present complexities and controversies of the effects of aging on heart rate control found within the autonomic literature challenges future researchers.
APPENDIX 1

CONSENT TO ACT AS HUMAN SUBJECT

SUBJECT'S NAME __________________________ Date: __________________

1. I AUTHORIZE Dr. Peter Raven and associates to perform the following procedures and investigations.

   Each subject will undergo an initial screening procedure requiring a resting 7-lead ECG, clinical lung function tests and then a maximum exercise test by walking on a treadmill. During this time expired air will be collected and monitored for volume, oxygen percentage and carbon dioxide production and used to calculate the subject's maximal aerobic power (VO₂ max). Electrocardiographic tracings will be obtained at intervals during the test and the ECG will be monitored continuously on an oscilloscope. At three minutes into the maximal test cardiac output will be measured by a CO₂ rebreathing method. In this technique each person breathes into and out of a bag containing a breathing mixture of oxygen, carbon dioxide and nitrogen at a specific rate. This breathing technique takes 10-30 seconds to complete. The method measures the amount of blood pumped by the heart. At the same time this technique is being performed ECG and blood pressure will also be determined. Following the test a 5-ml venous blood sample will be drawn from an arm-vein. Following the maximal test the heart-rate and blood pressure response to putting the hand in ice water and putting their face in cold water (4-5⁰) will be monitored for two minutes. In addition, on a separate day each subject will be asked to walk or run on a treadmill for 10 minutes at load equivalent to 35%, 50% and 75% of their maximal capacity. Between each load the subject will rest until baseline heart rate and blood pressure has been re-established. After this subjects will be asked to crank a bicycle using their arms at the same levels of work as outlined above. Similarly both the legs and arms will be tested with static (pushing against a resistance) at 35%, 50% and 70% of their maximum for two minutes or when fatigue sets in. During these tests, heart rate and blood pressure will be monitored continuously while measures of cardiac output and oxygen uptake will be made intermittently as described previously. After these initial tests the subjects will undergo a 10 week period of aerobic exercise training, three sessions per week for 30 minutes each session. The training will consist of riding a bicycle ergometer at an individual level based on 70% of their heart rate range (i.e. HR_{rest} + [HR_{max} - HR_{rest}] X 70/100.
Following the 10 weeks of the training the maximal and sub-maximal exercise tests and cold face and hand tests will be repeated. A further requirement is for each subject to allow physicians at Southwestern Medical School in Dallas to screen their heart function by a special technique--echocardiography. In this technique ultrasound is used. It entails no invasion of the body by needles, etc., but does require ECG paste to be placed on the chest. Many athletic trainers use ultrasound to treat deep muscle bruises or tears.

2. THE PROCEDURE(S) AND/OR INVESTIGATION(S) LISTED IN PARAGRAPH 1 HAVE BEEN EXPLAINED TO ME BY

3. I UNDERSTAND THAT THE PROCEDURE(S) AND/OR INVESTIGATION(S) DESCRIBED IN PARAGRAPH 1 INVOLVES (INVOLVE) THE FOLLOWING POSSIBLE RISKS AND DISCOMFORTS: (describe in detail)

   There exists the possibility that certain changes may occur during the progress of the stress test and exercise tests. These changes could include abnormal heart beats, abnormal blood pressure and in rare instances, a "heart attack." Professional care in selection and supervision of individuals provides appropriate precaution against such problems. The close monitoring of the subject by a licensed physician experienced in maximal stress test procedures reduces the risks involved in maximal testing. The cold pressor tests may produce transient localized pain in reactive subjects along with a feeling of discomfort associated with the application of cold water. A reflex slowing of heart rate is observed with the cold face test and may (in highly reactive subjects) transiently block electrical activity of the heart. However, this procedure has not been associated with any clinical symptoms or events. However, a physician will be present for all test procedures.

AND THAT THE FOLLOWING BENEFITS OF THE PROCEDURE(S) AND/OR INVESTIGATION(S) ARE AS FOLLOWS: (describe in detail)

The benefits included for the subject--a personal health and physical fitness evaluation, and medically supervised exercise training program to improve physical fitness. The data obtained will allow physicians to more easily judge an elderly patient's response to exercise training programs.

4. I understand that _____________________________________________ will answer any inquiries I may have at any time concerning the procedure(s) and/or investigations.
5. I understand that I may terminate my participation in the study at any time, and that, owing to the scientific nature of the study, the investigator may conclude my participation at any time.

SUBJECT'S SIGNATURE ____________________________________________________________________________

WITNESS ______________________________________________________________________________________

(If subject is a minor, or otherwise unable to sign, complete the following:)

Subject is a minor (Age __), or is unable to sign because:
____________________________________________________________________________________________
____________________________________________________________________________________________

Father ___________________________________________________________________________ Guardian __________
Mother ___________________________ Other person and relationship ____________________________
APPENDIX II

SELF-ADMINISTERED PRE-EXERCISE MEDICAL HISTORY FORM

Name __________________________ Number _____ Date _____

PAST HISTORY
(Have you ever had?)
- Rheumatic Fever ( )
- Heart Murmur ( )
- High Blood Pressure ( )
- Any Heart Trouble ( )
- Disease of Arteries ( )
- Varicose Veins ( )
- Lung Disease ( )
- Operations ( )
- Injuries to Back, etc. ( )
- Epilepsy ( )

FAMILY HISTORY
(Have any of your relatives had?)
- Heart Attacks ( )
- High Blood Pressure ( )
- Too Much Cholesterol ( )
- Diabetes ( )
- Congenital Heart Diseases ( )
- Heart Operations ( )
- Other ( )

PRESENT SYMPTOMS REVIEW
(Have you ever had?)
- Chest Pain ( )
- Shortness of Breath ( )
- Heart Palpitations ( )
- Cough on Exertion ( )

- Heart Attacks ( )
- Back Pain ( )
- Swollen, Stiff, or Painful Joints ( )
- Do you awaken at night to urinate? ( )
- Explain __________________________

RISK FACTORS

1. Smoking
   - Do you smoke ( ) ( )
   - How many? ___
   - How many years? ___
     Cigarettes ( ) ( )
     How many? ___
     How many years? ___
     Cigar ( ) ( )
     How many? ___
     How many years? ___
     Pipe ( ) ( )
     How many times a day? ___
     How many years?

   How old were you when you started? ___
   In case you have stopped, when did you? __________________________
   Why? __________________________

2. Diet
   - What is your weight now? __________ 1 year ago? __________
   - At age 21? _____
   - Are you dieting? _____
   - Why? __________________________
3. Exercise

Do you engage in sports? ________
What? ___________ How often? ______
How far do you think you walk each day?
Is your occupation: Sedentary ( ) Active ( )
( ) Inactive ( ) Heavy Work ( )
Do you have discomfort, shortness of breath, or pain with moderate exercise? ___ Specify _____________________
Were you a schoolboy or college athlete? ______
Specify ________________________________
APPENDIX 3

SELF REPORT CARD

<table>
<thead>
<tr>
<th>NAME</th>
<th>No.</th>
<th>Wk.</th>
</tr>
</thead>
<tbody>
<tr>
<td>J=Jogging  W=Walking  T=Treadmill  BE=Bicycle Ergometer</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>MODE</th>
<th>DISTANCE</th>
<th>TIME</th>
<th>HR</th>
<th>WT</th>
</tr>
</thead>
<tbody>
<tr>
<td>M</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>W</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Th</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Su</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
BIBLIOGRAPHY


Baun, W. B. & Raven, P. B. The effects of age and fitness level on the response to local cold pressor test. The Physiologist, 1979, 22(4), 8. (Abstract)


Stromme, S. B. & Ingjer, F. Comparison of diving bradycardia and maximal aerobic power. Aviation Space and Environmental Medicine. 1978, 49(11), 1267-1270.


