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THE POST-EXERCISE CARDIOVASCULAR RESPONSE OF POST MYOCARDIAL INFARCTION MALES TO VARYING WATER TEMPERATURES IN A HOT TUB

by

Janeen S. Docsa

A Thesis
Submitted to the
Faculty of The Graduate College
in partial fulfillment of the requirements for the
Degree of Master of Arts
Department of Health, Physical Education and Recreation

Western Michigan University
Kalamazoo, Michigan
August 1986
THE POST-EXERCISE CARDIOVASCULAR RESPONSE OF POST MYOCARDIAL INFARCTION MALES TO VARYING WATER TEMPERATURES IN A HOT TUB

Janeen S. Docsa, M.A.
Western Michigan University, 1986

This study compared heart rate, blood pressure, and electrocardiographic response of post myocardial infarction males taking either vasodilator and beta blocker or just beta blocker medications, and a control, to different water temperatures in a hot tub. Twenty-six adult males were observed. Each subject engaged in exercise, then sat in a hot tub for 12 minutes. Each subject repeated this procedure three times, once at each water temperature (36.6°, 38.3°, and 40°C).

The data during immersion indicated: (a) heart rate was affected by increased temperature and duration of exposure; (b) blood pressure was affected by length of exposure; (c) frequency of arrhythmias was influenced by group differences; and (d) immediately following immersion, heart rate was influenced by temperature, blood pressure was influenced by interaction of temperature and group differences. It was concluded that immersion in water up to 40°C by post myocardial infarction males taking vasodilator and/or beta blocker medications should not be restricted.
ACKNOWLEDGEMENTS

I would like to dedicate this thesis to all the clients and staff at the Institute for Cardiovascular Health. It is through their inspiration that this study was conducted; and I am indebted to all of them for their cooperation in its completion.

I would like to recognize the members of my thesis advisory committee, Dr. Roger Zabik, Dr. Mary Dawson, and Dr. Harold Ray. Their assistance in preparing this study is greatly appreciated.

My special appreciation is also extended to Jeff Morgan, Paul Visich, Vickie Schmidt and Anne Smith of the institute staff. The use of their facilities, as well as their patience, understanding and words of encouragement were invaluable.

I am deeply indebted to the participants of this study for volunteering their time and cooperation. Without those unique individuals, this study could not have taken place.

Lastly, I would like to express my sincere gratitude to the members of my family, especially my husband Steve. Their assistance and constant encouragement made the completion of this study possible.

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CHAPTER I
INTRODUCTION

A form of recreation, of therapeutic value, that is gaining popularity in this country, is the hot tub. Locally there are commercially available public hot tub facilities and increasing numbers of tubs installed in private homes. As with any body of water there exists the potential for injury and possibly even death. This potential is compounded in the hot tub due to the high water temperature.

Heat can cause drowsiness, lethargy, vasodilation, and increased core temperature. These effects can cause fatal consequences in even the healthy individual. There is special concern therefore, for higher risk populations. One such group of people is cardiac patients, especially those taking medications that suppress blood pressure and/or heart rate response.

Two categories of cardiac medications that pose this concern are vasodilating agents and beta adrenergic blocking agents. The vasodilators act to dilate the walls of the blood vessels. When this occurs under resting conditions, the result is a decrease in blood pressure. Since this is also a result of hyperthermia, fear is that when the two are combined, the blood pressure may fall dangerously low.

Beta blockers act by blocking the beta receptor sites of the sympathetic nervous system. As a result heart rate and blood pressure are suppressed and attempts by the central nervous system to
increase them are only partially achieved. It is possible that individuals taking this type of medication, exposed to hyperthermic environments, may also experience severe hypotension.

Statement of the Problem

This study compared the heart rate, blood pressure and electrocardiographic response of post myocardial infarction males, using two different classes of cardiac medications, to three different water temperatures in a hot tub. Investigation of this problem involved concurrent examination of the following subproblems:

1. To compare the effect of increasing water temperature on each subgroup.

2. To compare the response of the subgroups at each water temperature.

Purpose of the Study

The purpose of this study was to determine if current recommendations on water temperature for cardiac populations are justified.

Need for the Study

This study was conducted to determine safe water temperature levels, for cardiac diseased populations, in a hot tub. The use of hot tubs by persons with cardiac disease poses extraordinary risk. Frequently the capability of the cardiovascular system in this type of individual is compromised by the disease. It may be further limited by the use of medications that suppress the action of the
cardiovascular system.

The United States Consumer Product Safety Commission keeps statistics on fatalities involving hot tubs. The cause for these fatalities ranges from electrocution, to drowning after hair was caught in the drain, to the combined effects of heat and alcohol leading to drowning. The latter is the largest cause of hot tub related fatalities. To date there are no reported fatalities attributed to the compromised cardiovascular response of a coronary artery diseased person (United States Consumer Product Safety Commission, 1986). It should be kept in mind however, that public facilities are required to display warning statements that "persons suffering from heart disease, diabetes, high or low blood pressure should not enter the hot tub without prior medical consultation and permission from their doctor" (National Spa and Pool Institute, 1981, p. 16). This warning may have prevented persons from using the hot tub, and thus prevented potentially dangerous situations from becoming statistics.

Hot tubs are increasing in popularity for private and public use. One is available for use in a local cardiac rehabilitation program. This cardiac program offers hot tub experiences at water temperatures well below the guidelines established for the general population (P. S. Visich, personal communication, March 17, 1986). However, these guidelines were established by default. Since no fatalities were reported in temperatures at or below the recommended maximum, it was therefore assumed to be safe for the general population (Brown, 1981). Despite the increasing availability of hot tubs, no reported investigation of the cardiovascular response of a
diseased nor even a healthy population could be found.

Delimitations

This study was delimited to the following characteristics.

1. Participants were males between the ages of 31 and 74 years of age.

2. All subjects had participated in the supervised aerobic exercise program at Borgess Medical Center's Institute for Cardiovascular Health for at least three months.

3. Participants were divided into three subgroups: (a) myocardial infarction, taking beta blocker and vasodilator medications, (b) myocardial infarction, taking beta blocker but not vasodilator medications, (c) no known history of coronary artery disease, not taking vasodilator or beta blocker medications.

4. There were four measures of cardiovascular response: heart rate, systolic blood pressure, diastolic blood pressure, and frequency of arrhythmias.

5. The water temperatures 36.6°, 38.8°, and 40° C were observed for 12 minutes.

Limitations

This investigation was limited to the following:

1. A narrow range of water temperatures was studied.

2. Subjects in all groups may have taken other medications that affected their cardiovascular response.

3. Water was circulated by hydrojets which may affect the trans-
fer of heat compared with other types of water circulation.

Assumptions

The study was conducted under the following assumptions:

1. Participants in the medicated groups were compliant with the prescribed regime.
2. Participants accurately reported their smoking status.
3. Participants reported recent illness and injury.
4. The sphygmomanometer and telemetry equipment remained accurate to their original calibration throughout the data collection.

Hypotheses

This study was conducted to test the following hypotheses:

1. At any given water temperature, the heart rate response during and immediately following immersion in a hot tub would be higher for the post myocardial infarction group taking vasodilator and beta blocker medications than the post myocardial infarction group without vasodilator medications.

2. At any given water temperature, the heart rate response during and immediately following immersion in a hot tub would be higher for the post myocardial infarction group taking beta blocker medications than for the healthy, non-medicated group.

3. At any given water temperature, the blood pressure response during and immediately following immersion in a hot tub would be lower for the post myocardial infarction group taking vasodilator and beta blocker medications than the post myocardial infarction group
without vasodilator medications.

4. At any given water temperature, the blood pressure response during and immediately following immersion in a hot tub would be lower for the post myocardial infarction group taking beta blocker medications than for the healthy, non-medicated group.

5. At any given water temperature, the frequency of arrhythmias during immersion in a hot tub would be greater for the post myocardial infarction group taking vasodilator and beta blocker medications than the post myocardial infarction group without vasodilator medications.

6. At any given water temperature, the frequency of arrhythmias during immersion in a hot tub would be greater for the post myocardial infarction group taking beta blocker medications than for the healthy, non-medicated group.

7. Within each subgroup of subjects, the heart rate response during and immediately following immersion in a hot tub would become greater as the temperature of the water increased.

8. Within each subgroup of subjects, the blood pressure response during and immediately following immersion in a hot tub would become lower as the temperature of the water increased.

9. Within each subgroup of subjects, the frequency of arrhythmias during immersion in a hot tub would become greater as the temperature of the water increased.

10. Within each subgroup the heart rate and blood pressure response would increase as the duration of immersion increased.
Definition of Terms

The following terms and their definitions are important to the understanding of this study:

1. Hemodynamics - the forces generated by the heart and resulting motion of blood through the cardiovascular system (Astrand and Rodahl, 1977).

2. Hyperthermia - an elevated body core temperature, higher than the thermoregulatory "setpoint" (Brown, 1981).

3. Tachycardia - a rapid heart rate, greater than 100 beats per minute (Phillips and Feeney, 1980).

4. Cardiac output - the volume of blood ejected by the heart per minute. It is the product of heart rate and stroke volume (Smith and Kampine, 1984).

5. Stroke volume - the volume of blood ejected per contraction of the heart (Shepherd and Vanhoutte, 1979).

6. Ectopic beat - a contraction of the myocardium that originates from any focus other than the sinus node (Marriott, 1977).

7. Ventricular ectopic beat - a contraction of the myocardium that originates from a ventricle (Marriott, 1977).

8. Supraventricular ectopic beat - a contraction of the myocardium that originates from above the ventricle (Marriott, 1977).

9. Ischemia - a temporary insufficient blood supply to a tissue (Goldman, 1979).

10. Sphygmomanometer - a device used to measure blood pressure consisting of an inelastic inflatable cuff attached to a column of mercury that gauges the pressure (Smith and Kampine, 1984).
11. Myocardial infarction - a sudden, irreversible, ischemic injury due to coronary arterial narrowing or occlusion with sustained damage to a segment of the myocardium (Smith and Kampine, 1984).

12. Vasodilating agent - a medication that relaxes the smooth muscle of vascular walls and dilates the vessel which acts to decrease blood pressure (Liss, 1980).

13. Beta adrenergic blocking agent - a medication that blocks the beta-receptor sites of the sympathetic nervous system. This decreases heart rate, contractility and blood pressure (Liss, 1980).

14. Arrhythmia - any variation from the normal electrical rate and sequences of the cardiac activity. This term has come to include abnormalities of impulse formation and conduction (Phillips and Feeney, 1980).
CHAPTER II

REVIEW OF LITERATURE

When confronted with a hyperthermic situation, the response of the human body is to cool itself. It does this by increasing blood flow to the surface of the body where heat can be dissipated by convection and evaporation. The increased blood flow to the surface is achieved because of cardiovascular changes. The cardiovascular response is the prime concern when considering the appropriateness of individual use of the hot tub. Although considerable study has been made of the effects of various forms of heat stress dating back to 1889, little information is available specifically on immersion in a hot tub. The available literature suggests that the response is in general, similar to the response to heat from other sources such as saunas and ambient temperatures.

This review of literature will discuss the heart rate, heart rhythm, blood pressure, cardiac output, vascular and coronary ischemic response to hyperthermia. The discussion will pertain to hyperthermia in general as reports are from both warm air and warm water immersion. When possible, any distinction between coronary disease populations and normals will be considered, as well as current guidelines. Since subjects will be exercising prior to immersion, a brief review of physiological response to aerobic exercise will be made.
Blood Pressure

One of the primary cardiovascular responses to hyperthermia is vasodilation as this allows more blood to flow through the vasculature. Besides allowing for greater blood flow, vasodilation also results in a decrease in blood pressure. The available literature ranges from studies in controlled atmospheres, to saunas, to water baths. Temperatures range from 0°C to 45°C. Regardless of the variety of methodology, all reported a decrease in blood pressure due to vasodilation in warm temperatures (Bazett, Scott, Maxfield, and Blithe, 1937; Grollman, 1930; Sancetta, Hackel, Traks, and Wittels, 1964; Sancetta, Kramer, and Husni, 1958; Sherif, Shahwan, and Sorour, 1970; Taggert, Parkinson, and Carruthers, 1972; Turner, 1980).

Taggart et al. (1972) studied the heat stress of a 32.2-43.3°C (dry bulb) sauna versus exercise for 35 males, 18 of whom had a previous history of myocardial infarction. They found that for both groups, systolic and diastolic pressures were lower during the sauna.

Sherif et al. (1970) looked at 3 groups of cardiac patients, one with rheumatic mitral stenosis, another with emphysema cor pulmonale, another with biharzial cor pulmonale and a control. They were monitored for two hours in a 40°C catheterization room. The systemic circulation showed marked changes in both control and cardiac patients after the acute thermal stress. There was a consistent marked drop in the total peripheral vascular resistance. Exposure to heat resulted in marked vasodilation involving both primary release of the vasoconstrictor tone and active vasodilation. The percentage of drop
of the total peripheral vascular resistance was comparable in the normal group and in the three cardiac groups. No mention was made, however, of amount or type of medications, if any, that subjects took.

Many of the authors looked at exposure to heat for extended periods of time. Sancetta et al. (1958) observed 16 patients with diseased left ventricles in an ambient temperature of 36.6°C for two hours and noticed a linear relationship between decrease in blood pressure and length of exposure to the heat. Koroxenidis, Shepherd and Marshall (1961) studied eight healthy males who were wrapped in blankets with feet and legs immersed in a stirred 43.8°C bath. The systolic pressure dropped on the average about 15 mm Hg while the diastolic decreased about 5 mm Hg. Goldschmidt and Light (1925), after observing subjects whose arms were immersed to their elbows in 43.8-46.1°C water, noted "that the veins became extremely dilated as the temperature of the water rose" (p. 157).

Heart Rate

Another major cardiovascular response to hyperthermia is an increased heart rate. In order for more blood to be circulated, the heart must pump faster. An increase in heart rate in response to hyperthermal stress has been observed repeatedly in the literature.

Bazett et al. (1937) found that as the temperature of a bath rose from a sub-body temperature of 32°C to 38.6°C, the heart rate rose progressively from a mean of 57 to 91 bpm. Koroxenidis et al. (1961) observed that after 50 minutes of immersion of the feet and
legs in 43.8°C water and the body wrapped in blankets, the heart rate increased by 49%. Sancetta et al. (1958) looked at 15 patients with hypertrophied left ventricles, half of whom were considered to be in heart failure. They found that all 15 subjects when exposed to 36.6°C, 40% humidity for two hours, showed increased heart rates. Taggart et al. (1972), studied post myocardial infarction and normal subjects. They were impressed by "the degree of tachycardia in the sauna bath and the short time required for very fast heart rates to be attained" (p. 74). In the only report of observations in a hot tub, all three subjects developed tachycardia during immersion in 41°C water (Turner 1980).

Cardiac Output

Since cardiac output is the product of heart rate and stroke volume, it follows that if the heart rate increases in response to hyperthermia, then so should cardiac output. Before making this conclusion, the response of stroke volume must be explored.

Burch and Hyman (1957), studied patients with chronic congestive heart failure and subjects without cardiovascular disease, exposed to room atmosphere of 43.8°C and 86% humidity. They found that cardiac output and stroke volume increased in every subject. There was a threefold increase in the control and a twofold increase in the cardiac group.

Slightly different findings were reported by several other researchers who attributed the increase in cardiac output primarily to an increase in heart rate. Sherif et al. (1970) observed three
different cardiac groups and a control group. They found an almost twofold increase in cardiac output in the normal group and slightly less in the cardiac groups, crediting the increase in cardiac output to increased heart rate. A slight increase in stroke volume was reported in the normal group and even less in the cardials.

Similarly, a study of eight healthy subjects with feet and legs immersed in 43.8°C water and bodies wrapped in blankets, found a 60% increase in cardiac output from the control values (Koroxenidis et al. 1961). The heart rate showed a 49% increase, whereas the stroke volume only increased by 9%. A fairly constant stroke volume in the face of increased cardiac output was also reported by Grollman (1930). It would appear from these reports, that a change in the contractility of the heart does not play a major role in the response to hyperthermia.

Ectopic Heart Rhythm

Although not a consistent response, there are reports of increased ectopic rhythms with hyperthermia. Taggart et al. (1972) observed ventricular ectopic beats that were prominent and at times recurrent in four of their 17 normal subjects during the sauna. Six out of 18 of their post myocardial infarction subjects developed ectopic beats during the sauna, ventricular in five and supraventricular in one.

Turner (1980) also observed multiple ventricular ectopic beats in one out of three of his healthy subjects while immersed in a hot tub. It would thus appear that ectopic beats are a common but not
universal response to hyperthermia.

Myocardial Ischemia

Since hyperthermia is a stress to the body, it is conceivable that it could cause ischemia in the myocardium. Taggart et al. (1972) reported observing signs of ischemia. Many of their cardiac as well as normal subjects rapidly developed ST-T changes resembling ischemia in response to heat. The only other study to report electrocardiographic data did not mention any observations of ischemia, however, the sample size was limited to three healthy subjects between 20 and 36 years of age.

Air versus Water as a Surrounding Medium

The available literature pertaining to the cardiovascular response to immersion in warm water is extremely limited. Therefore, to understand the anticipated response one must consider research pertaining to heated environments in general. These range from water, to moist air, to dry air, to a combination of water and conservation of body heat with blankets. However, Grollman (1930) pointed out that "due to the greater conductivity of water, as compared to air, the temperature effect of a water bath at any given temperature on the circulation, will differ markedly from the results obtained when air at the same temperature is used as the surrounding medium" (p. 271). To be more specific, one might speculate that just as cold water cools the body better than cold air, warm water might warm the body better (Goldschmidt and Light, 1925). Therefore, some
of the effects reported from ambient temperatures may be more profound in water of equal temperatures. For this reason only general trends should be compared from one medium to another.

**Acute Effects of Aerobic Exercise**

With the onset of rhythmic muscular exercise, a complex series of cardiovascular adjustments occur to ensure that: (a) the active muscles receive a blood supply appropriate to their increased metabolic needs; (b) the heat generated by the active muscles is eliminated; and (c) the blood supply to the brain and the heart is maintained (Shepherd and Vanhoutte, 1979). To accomplish these needs, exercise invokes a sympathetic response of tachycardia, increased myocardial contractility, hypertension, peripheral vasoconstriction and dilation, (Smith and Kampine, 1984). The heart rate increases almost immediately in response to exercise (Lamb, 1978). It is stimulated to do so because of the increased demand for oxygen by the working muscles, need to dissipate heat, and increased venous return to the right atrium. If all the blood vessels in the body were dilated, the blood supply would be insufficient to fill them. Therefore, during exercise, blood is shifted to the working areas by way of vasoconstriction in nonworking areas and vasodilation in working areas. Simultaneously, however, cardiac output is increasing, due to increased contractility of the myocardium, which causes a greater systolic blood pressure that more than counteracts the tendency toward vasodilation in the working muscles. The net effect of exercise on blood pressure therefore, is an increase in systolic
pressure, primarily because of increased cardiac output (Lamb, 1978).

One very important function of blood circulation is to transport heat. As one exercises, heat is generated by the working muscles. Excess heat is carried from the interior of the body to the body surface by the blood (Astrand and Rodahl, 1977). This is accomplished through the vasodilation and increased circulation to the skin, so that heat is lost through convection (Lamb, 1978).

As the heart muscle works harder during exercise, its demand for oxygen increases. This demand is met by a greater blood flow through the coronary arteries due to coronary vasodilation. This may increase up to five times the resting value (Lamb, 1978). However, if the elasticity of the arteries are compromised due to atherosclerotic disease, this demand may not be met and a state of ischemia develops.

Similar to the heart, blood flow to the lungs is increased as the output of the right ventricle increases (Lamb, 1978). The overall supply of blood to the brain during exercise remains constant (Milnor, 1980).

Following exercise, for a period of time that will be affected by physical condition, some effects will remain. The heart rate slows rapidly when exercise stops. However, it does not immediately return to pre-exercise levels. The accelerator effect of increased levels of hormones, such as adrenalin and nor-adrenalin, and of increased temperature of the heart, may continue to operate until the body cools and the hormones are metabolized (Lamb, 1978). Recovery from the vasodilation effect of exercise does not take place as fast as the recovery of cardiac output. Therefore, following exercise

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blood pressure, especially systolic, may fall below its pre-exercise level. The effect of ischemia, if not too severe, is relieved by the decreased demand on the myocardium.

Response to Hyperthermia

The protected human being may tolerate great variations in environmental temperatures. But a person can tolerate a variation of only about 4°C in deep body temperature without impairment of physical and mental work capacity (Astrand and Rodahl, 1977). Temperature regulation, especially against overheating, is of high priority.

Exposure to a hot environment has a number of effects on the circulation, most of them traceable to the cutaneous vasodilation that constitutes one of the mechanisms for heat regulation. Along with a greater blood flow to the skin, the cardiac output and resting pulse rate increase, systolic arterial pressure rises moderately and diastolic pressure falls. The intensity of the initial adjustment to high temperatures varies greatly in different individuals (Milnor, 1980).

Stress of Hyperthermia versus Exercise

Since hyperthermia and exercise are both stressors on the body, some researchers have attempted to compare the two. Taggart et al. (1972) found that for both their normal and post myocardial infarction groups, both systolic and diastolic pressures were lower during the sauna, whereas during exercise, there was an increase in systolic pressure with small variable changes in diastolic pressure. They
also found that of their four normal subjects that exhibited ven­
cular ectopic beats during the sauna, none exhibited any during exer­
cise. Of the six post myocardial infarction subjects that experi­
cenced ectopic beats in the sauna, only one did so during exercise and

to a lesser extent than in the sauna.

While some of the responses to these two stressors are similar
such as increased heart rate, cardiac output and vasodilation; others
are opposite such as blood pressure response. Turner (1980) observed
this when the effects of exercise following hypothermic immersion
maintained the tachycardia, yet reverted the hypotension. It there­
fore should not be assumed that just because an individual responds
without problems to one stressor, that the same will be true for a
different stressor.

Postural Hypotension and Hyperthermia

When man rises from a supine position and stands erect, the
weight of intravascular columns of blood in vessels above and below
the heart places stresses on the circulation that are absent in the
supine position. This alters local transmural pressures, so that the
larger veins, which are readily distended, tend to enlarge in the
legs and collapse in the upper portions of the body (Milnor, 1980).
Decreased tolerance to the upright posture will occur if the skeletal
muscle pump effect on venous return is inadequate or if the
baroceptor reflexes are interrupted. Baroreceptors sense pressure
against arterial walls and stimulate vasodilation (depressor reflex)
or vasoconstriction (pressor reflex) to maintain adequate blood
pressure levels. Partial or complete loss of sympathetic vasoconstrictor responses may occur after administration of beta adrenergic blocking agents (Smith and Kampine, 1984).

The vasodilatory effects of hyperthermia may interfere with the pressor reflex in response to sudden standing. It was reported by Turner (1980) that on standing after hyperthermic immersion, each subject exhibited considerable accentuation of tachycardia and hypotension. The diastolic blood pressure became unrecordable in two of the three subjects, and each developed severe syncopal symptoms. These were healthy, non-medicated subjects. No information was found on the response to standing following hyperthermia, after the administration of beta adrenergic blocking or vasodilating agents.

Effect of Hyperthermal Stress on Right versus Left Ventricle

The effect of the hemodynamic burden of acute thermal stress on the work of the right and left ventricle is quite different. Although both pump a higher output per minute, "the left ventricle does so in the face of a reduced mean systemic pressure, while the right ventricle has to overcome the added factor of a raised mean arterial blood pressure" (Sherif et al. 1970, p. 313). In a study of cardiac patients, they found this to be the main factor responsible for the eventual (two hour exposure) right ventricular decompensation and the appearance of signs of right-sided heart failure. Hyperthermal stress would thus appear to be contraindicated for populations of rheumatic mitral stenosis, emphysema cor pulmonale and bilharzial such as theirs. By the same token, other researchers have suggested
that hyperthermia might be of benefit to certain patients with left ventricular failure, at least on a short term basis (Sancetta et al. 1958; Sancetta et al. 1964).

Recommended Water Temperatures

The National Spa and Pool Institute (1981) and the U.S. Consumer Product Safety Commission have set standards for water temperature in public and residential hot tubs (Brown, 1981). They both recommended that the temperature not exceed 40°C. The basis for this recommendation was that as of 1981 there were no known cases of death "in which the water temperature was 104°F (40°C) or lower, therefore it is presumed that this recommended maximum water setting is relatively safe for most at risk populations" (Brown, 1981, p. 13). Three exceptions were given: pregnant women, children, and adults with certain health problems such as hypertension or circulatory impairment. For these populations a maximum water temperature was recommended to be 100 - 102°F (37.7 - 38.8°C). No basis for the recommendation of these exact temperatures was given.

Grollman (1930), after observing responses to temperatures from 0 - 38.3°C, found that at elevated temperatures, increasing temperature exerted a progressively greater effect on the heart rate. This, in theory, would support the above recommendations.

Duration of Exposure

Specific recommendations in the literature for duration of exposure to the hyperthermic environment of a hot tub were vague.
The United States Consumer Product Safety Commission stated: "There is some indication that a range of between 20 to 30 minutes would be appropriate for most users if the water temperature does not exceed 104°F (40°C) (Brown, 1981, p. 12). They further stated that it should be less for the special populations described above under recommended water temperature. Bauer (1984) set a time limit of ten minutes but there was no rationale stated.

There is support for the idea that the exposure be brief. Many of the investigators in this review of literature have studied exposure to heat for extended time periods. Sancetta et al. (1958) noticed a linear relationship between decrease in blood pressure and length of exposure to the heat. Similarly, Taggart et al. (1972) reported that heart rates showed a progressive increase during the sauna, being roughly related to the time in the sauna. It would therefore appear desirable to curtail the length of time spent in the heat, especially for persons in whom the combination of fast heart rates and hypotension are undesirable.
CHAPTER III

DESIGN AND METHODOLOGY

The purpose of this study was first to compare the cardiovascular effect of increasing water temperature on each subgroup. Second, to compare responses of the subgroups at each water temperature. Third, to determine if current recommendations on water temperature for cardiac populations are valid.

This chapter includes four sections: (a) Subject Selection, (b) Data Collecting Procedures, (c) Instrumentation, and (d) Statistical Analysis.

Subject Selection

The subjects in this study were chosen from the client population of the Institute for Cardiovascular Health at Borgess Medical Center in Kalamazoo, Michigan. As such, they engaged in any combination of walking, jogging, rowing, stationary bicycling and/or simulated Nordic skiing, for 30 to 40 minutes roughly, three times per week. Only those clients who had attended regularly for the past three months were eligible. There were 26 males ranging in age from 31 to 72 years. They were classified according to medical history and current medication regime. Males from each appropriate category were then randomly selected until nine for each group were found willing to participate.

The first group, referred to as Vasodilator/Beta Blocker Group,
consisted of males with a history of myocardial infarction documented by electrocardiography and/or coronary angiography. They were asymptomatic at the time of the study and had no history of heart failure. Their most recent maximal graded exercise test was negative for ischemia. They had been prescribed to take a form of beta blocker medication on a daily basis and a form of vasodilator medication on a daily basis and/or before exercise. The second group, referred to as Beta Blocker Group, was identical in criteria except that they did not take any vasodilator medication. The final group, referred to as the Control Group, was selected from the disease prevention population. They were males with no known history of coronary artery disease who were found to be at risk for its development. Their most recent maximal graded exercise test was negative for ischemia, or they had coronary angiography which ruled out coronary artery disease. They were not taking a vasodilator nor beta blocker medication at the time of the study. The general characteristics of the subjects are presented in Table 1.

Table 1
Descriptive Analysis by Weight, Height and Age for Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Weight</th>
<th>Height</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>X</td>
<td>s</td>
<td>X</td>
</tr>
<tr>
<td>Vasodilator/</td>
<td>9</td>
<td>76.5</td>
<td>4.4</td>
<td>56.4</td>
</tr>
<tr>
<td>Beta Blocker</td>
<td></td>
<td></td>
<td></td>
<td>6.0</td>
</tr>
<tr>
<td>Beta Blocker</td>
<td>8</td>
<td>84.9</td>
<td>13.7</td>
<td>63.9</td>
</tr>
<tr>
<td>Control</td>
<td>9</td>
<td>80.5</td>
<td>13.9</td>
<td>47.1</td>
</tr>
</tbody>
</table>

Note. Weight is in kg. Height is in cm. Age is in years.

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Data Collection Procedures

Subjects

Data collection took place at the Institute for Cardiovascular Health in Kalamazoo, Michigan. Each subject was observed three times during a six-week period, never more than once on any given day. A consent form which explained the nature of the study and the possible risks involved was signed by each subject at the beginning of the data collection. The consent form appears in Appendix A.

Subjects reported for observation wearing their regular loose, comfortable clothing and exercise shoes. Resting measures of heart rate, blood pressure and heart rhythm were taken. A Recent History Screening form, available in Appendix B, was completed. Subjects then participated in their regular exercise session which included ten minutes of warm-up, 30 minutes of aerobic exercise at an intensity of 70 to 85% maximal heart rate reserve, ten minutes of one bounce volleyball or light walking, and five minutes of cool-down stretches. The subjects then sat quietly until one of the following occurred: (a) their heart rates returned to within twelve beats per minute of their original resting heart rate; or (b) they were asymptomatic after ten minutes of sitting. They then prepared for immersion in the hot tub. This preparation involved toweling off any perspiration and changing into swim trunks.

Hot Tub Observation

Following the exercise session, toweling off, and change into
swim trunks, the subjects appeared in the hot tub room for preparation and observation. Two adhesive electrodes were applied and connected to the ECG transmitter unit. Heart rate, blood pressure and heart rhythm were again recorded, then the subjects entered and seated themselves in the hot tub. The electrocardiogram (EKG) was monitored throughout the 12 minute immersion while heart rate and blood pressure were recorded every 3 minutes. At the end of the 12 minutes, subjects exited the tub and, if possible, remained standing while a heart rate and blood pressure were recorded. They were then assisted to a seated position in a chair where the ECG, heart rate and blood pressure were monitored an additional ten minutes.

Electrocardiogram Monitoring

Upon arrival in the hot tub room, the subjects were prepared for electrocardiogram (EKG) monitoring. A General Electric telemetry device was used to monitor the EKG. Lead wires, seven feet in length, were used so that the General Electric's nine-volt telemetry transmitter could be kept well away from the water. A twelve-lead EKG was not used for the following reasons: (a) one lead was sufficient to record arrhythmia and heart rate as demonstrated by the literature review (Billone, 1985; Eckberg, Drabinsky and Braunwald, 1971; Irving, 1963; Olsen, Fanestil and Scholander, 1962; Ryan, Hollenberg, Harvey and Gwynn, 1976; Whayne and Killip, 1967); and (b) the twelve-lead EKG monitored underwater through AC current carried an undetermined hazard.

Electrodes were applied for a bipolar lead which approximated a
Standard Lead I: negative terminal right arm, 3 cm below mid clavicle and positive terminal left arm, 3 cm below mid clavicle. Lead I was used as electrodes in this position were primarily out of the water and conducted less artifact. The sites were cleansed with alcohol and abraded with gauze and fine emery paper. Dyna-trace electrodes by Andover Medical were used.

One technician, certified by the American College of Sports Medicine as a Preventive and Rehabilitative Exercise Test Technologist, collected all data. Because of this, while measuring blood pressure, a continuous hard copy of the ECG was allowed to run to include all arrhythmias. The remainder of the time the oscilloscope was observed and any arrhythmias tallied as they occurred. Each deviation from normal sinus rhythm, PAC, PNC, PVC, etc., was tallied as one arrhythmia. Heart rates were measured by averaging rates on a six second strip.

**Water Temperature Range**

Observations were made at the following water temperatures: 36.6°C (98°F); 38.3°C (101°F); and 40°C (104°F). The rationale for these temperatures was based on the recommendations of the U.S. Consumer Product Safety Commission (Brown, 1981) and the National Spa and Pool Institute (1981). The temperature 40°C was selected for observation as this was the recommended water temperature limit for the general population. A range of 37.7 - 38.8°C was recommended as the maximum temperature for cardiac populations. For this reason a mean of 38.3°C was selected to be observed. The temperature 36.6°C
was chosen as a temperature below the recommendations, and at an increment equal to that between the other two temperatures.

Due to the use of a repeated measures statistical design, subjects were randomly assigned an order of temperatures observed to minimize the effect of an ordered pattern in data collection procedures. The subjects were unaware of their temperature order until all data were collected.

Instrumentation

**EKG Telemetry Monitor**

A General Electric ECG Telemetry Monitor, model number 11KH1A1 was used. The unit was inspected by the medical electronics department at Borgess Medical Center prior to use. Paper speed was measured to be 25mm per second at the start of data collection.

**Thermometer**

An L & M Mercury Thermometer was used to measure water temperature. The thermometer measured to the nearest 0.2°C. The water temperature was measured ten minutes before observation, adjusted and measured again just prior to each observation until the temperature was within 0.1°C of the specified temperature for that observation.

**Sphygmomanometer**

A Tycos anaeroid sphygmomanometer, model number 509003 was used. This model was used for ease in reading while kneeling on the floor, and its portability during the subject's move out of the tub. The
instrument was calibrated prior to the start of data collection by the maintenance department at Borgess Medical Center according to the recommendations of the American Heart Association (1967).

Stethoscope

A Littman Dual-headed Stethoscope was used throughout the study. Only the diaphragm head was used for auscultation of Korotkoff sounds to determine blood pressure.

Statistical Analysis

Raw data for the four three-minute water intervals and immediately post immersion were coded to reflect their deviation from their respective measurements immediately prior to entering the water. This information was then analyzed at Western Michigan University, using the Computer Program BMDP. An Analysis of Variance (ANOVA), Split Plot Factorial statistical design with fixed effects was used to determine the effects of the independent variables on the dependent variables. Where the variance proved significant, Tukey's Honestly Significant Difference (HSD) test for multiple comparisons was employed to determine at what point the means became significantly different (Hopkins and Glass, 1978).

Two different designs were used to analyze the data. The first design consisted of three independent variables. The first was group with three levels. The second was water temperature, also with three levels. The final independent variable was duration of immersion, with four levels. The first design was repeated three times, once
for each dependent variable: (a) heart rate, (b) systolic blood pressure, and (c) diastolic blood pressure.

The second design involved only two independent variables: group with three levels, and temperature with three levels. This design was repeated four times, once for each of the dependent variables: (a) frequency of arrhythmias, (b) heart rate immediately post immersion, (c) systolic blood pressure immediately post immersion, and (d) diastolic blood pressure immediately post immersion.
CHAPTER IV

RESULTS AND DISCUSSION

This chapter includes the results and discussion of the cardiovascular responses obtained from three specific groups to three different water temperatures in a hot tub. The purposes of this study were to compare the post exercise cardiovascular effects of increasing water temperature on each subgroup, to compare responses of the subgroups at each water temperature, and to determine if current recommendations on water temperature for cardiac populations are justified.

Raw data for the four three-minute water intervals and immediately post immersion were coded to reflect their deviation from their respective measurements immediately prior to entering the water. This information was then analyzed at Western Michigan University, using the Computer Program BMDP. An Analysis of Variance (ANOVA), Split Plot Factorial Design with fixed effects was used to determine the effects of the independent variables on the dependent variables. Where the variance proved significant the Tukey Honestly Significant Difference (HSD) test for multiple comparisons was employed to determine at what point the group means became significantly different (Hopkins and Glass, 1978). The independent variables included: (a) groups with three levels, vasodilator and beta blocker medications, beta blocker medications only, and no medication; and (b) water temperature with three levels, 36.6°, 38.4°, and 40.0°C. The
dependent variables of this study, measured over time at each temperature, were: (a) heart rate, (b) systolic blood pressure, (c) diastolic blood pressure, and (d) frequency of arrhythmias. This chapter is presented in two sections: (a) results, and (b) discussion.

Results

Heart Rate During Water Immersion

Descriptive data for the dependent variable, change in heart rate, were divided into cells, each representing a group and a water temperature. Each water temperature was further divided into duration of immersion. These descriptive data are presented in Appendix C. An ANOVA was calculated using group, water temperature, and time as the independent variables. The difference the heart rate during immersion deviated from the heart rate immediately prior to entering the water served as the dependent variable. The ANOVA (see Table 3) indicated the following:

1. No difference in mean heart rate change, $F = 0.64$, was found between groups ($F(2, 23) = 3.44, p < .05$).

2. There was a significant difference, $F = 17.45$, between mean heart rate change dependent on water temperature ($F(2, 46) = 3.23, p < .05$).

3. No difference in mean heart rate change, $F = 1.10$, was found for the first order interaction of temperature X group ($F(4, 46) = 2.61, p < .05$).

4. There was a significant difference, $F = 27.54$, between mean
5. The first order interaction of time X group showed a significant difference between mean heart rate change, $F = 2.48$ ($F(6, 69) = 2.25, p < .05$).

6. Another first order interaction, temperature X time, also proved a significant difference in mean heart rate change, $F = 11.65$ ($F(6, 138) = 2.17, p < .05$).

7. For mean heart rate change, the second order interaction of group X temperature X time, $F = 0.96$, was not found to be significant ($F(12, 138) = 1.83, p < .05$).
Table 2
ANOVA Summary Table for Heart Rate During Water Immersion

<table>
<thead>
<tr>
<th>Source</th>
<th>S.S.</th>
<th>df</th>
<th>M.S.</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Between Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group (G)</td>
<td>216.01</td>
<td>2</td>
<td>108.00</td>
<td>0.64</td>
</tr>
<tr>
<td>Error</td>
<td>3904.00</td>
<td>23</td>
<td>169.74</td>
<td></td>
</tr>
<tr>
<td><strong>Within Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature (TP)</td>
<td>1729.21</td>
<td>2</td>
<td>864.61</td>
<td>17.45*</td>
</tr>
<tr>
<td>T X G</td>
<td>218.67</td>
<td>4</td>
<td>54.67</td>
<td>1.10</td>
</tr>
<tr>
<td>Error</td>
<td>2278.62</td>
<td>46</td>
<td>49.54</td>
<td></td>
</tr>
<tr>
<td>Time (TI)</td>
<td>462.73</td>
<td>3</td>
<td>154.24</td>
<td>27.54**</td>
</tr>
<tr>
<td>TI X G</td>
<td>83.42</td>
<td>6</td>
<td>13.90</td>
<td>2.48***</td>
</tr>
<tr>
<td>Error</td>
<td>386.44</td>
<td>69</td>
<td>5.60</td>
<td></td>
</tr>
<tr>
<td>TP X TI</td>
<td>268.39</td>
<td>6</td>
<td>44.73</td>
<td>11.65****</td>
</tr>
<tr>
<td>TP X TI X G</td>
<td>44.27</td>
<td>12</td>
<td>3.69</td>
<td>0.96</td>
</tr>
<tr>
<td>Error</td>
<td>529.69</td>
<td>138</td>
<td>3.84</td>
<td></td>
</tr>
</tbody>
</table>

*F(2, 46) = 3.23, p < .05  
**F(3, 69) = 2.76, p < .05  
***F(6, 69) = 2.25, p < .05  
****F(6, 138) = 2.17, p < .05

For the comparison of the heart rate means associated with temperature and time, Tukey's HSD test for multiple comparisons was used to locate significant differences between the three temperatures (see Table 3) and between the four times (see Table 4). The analysis of mean differences for temperature indicated that:

1. No significant difference, q = 1.16, existed between 36.6°
and 38.3° ($q(3, 101) = 3.40, p < .05$).

2. A significant difference, $q = 7.86$, existed between 36.6° and 40° ($q(3, 101) = 3.40, p < .05$).

3. A significant difference, $q = 6.74$, also existed between 38.3° and 40° ($q(3, 101) = 3.40, p < .05$).

### Table 3
Multiple Comparison of Heart Rate Means During Immersion by Temperature

<table>
<thead>
<tr>
<th>Temperature</th>
<th>36.6°C</th>
<th>38.3°C</th>
<th>40.0°C</th>
</tr>
</thead>
<tbody>
<tr>
<td>36.6°C</td>
<td>-</td>
<td>1.16</td>
<td>7.86*</td>
</tr>
<tr>
<td>38.3°C</td>
<td>1.16</td>
<td>-</td>
<td>6.74*</td>
</tr>
<tr>
<td>40.0°C</td>
<td>7.86*</td>
<td>6.74*</td>
<td>-</td>
</tr>
</tbody>
</table>

*$q(3, 101) = 3.40, p < .05$

The analysis of mean differences for time of immersion indicated that:

1. No significant difference, $q = 2.15$, existed between 3 and 6 minutes ($q(4, 74) = 3.74, p < .05$).

2. A significant difference, $q = 7.70$, existed between 3 and 9 minutes ($q(4, 74) = 3.74, p < .05$).

3. A significant difference, $q = 11.82$, existed between 3 and 12 minutes ($q(4, 74) = 3.74, p < .05$).

4. A significant difference, $q = 5.55$, existed between 6 and 9 minutes ($q(4, 74) = 3.74, p < .05$).

5. A significant difference, $q = 9.66$, existed between 6 and 12
minutes \( q(4, 74) = 3.74, p < .05 \).

6. A significant difference, \( q = 4.11 \), existed between 9 and 12 minutes \( q(4, 74) = 3.74, p < .05 \).

Table 4

Multiple Comparison of Heart Rate Means
During Immersion by Time

<table>
<thead>
<tr>
<th>Time</th>
<th>3 Minutes</th>
<th>6 Minutes</th>
<th>9 Minutes</th>
<th>12 Minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 Minutes</td>
<td>-</td>
<td>2.15</td>
<td>7.70*</td>
<td>11.82*</td>
</tr>
<tr>
<td>6 Minutes</td>
<td>2.15</td>
<td>-</td>
<td>5.55*</td>
<td>9.66*</td>
</tr>
<tr>
<td>9 Minutes</td>
<td>7.70*</td>
<td>5.55*</td>
<td>-</td>
<td>4.11*</td>
</tr>
<tr>
<td>12 Minutes</td>
<td>11.82*</td>
<td>9.66*</td>
<td>4.11*</td>
<td>-</td>
</tr>
</tbody>
</table>

\( q(4, 74) = 3.79, p < .05 \)

Two second order interactions were found to be significant. First, there was an interaction between times and groups. This interaction is described in Figure 1. All groups had an increase in heart rate over time, however, after six minutes the magnitude of that increase was greater for the Vasodilator/Beta Blocker and Control Groups than for the Beta Blocker Group.
Figure 1. Time X Group Interaction for Heart Rate During Immersion

Second, there was an interaction between temperatures and times. This interaction is described in Figure 2. Overall, the longer the exposure and the higher the temperature, the greater the change in heart rate. However, for the first six minutes, 38.3°C elicited a greater decrease in heart rate than 36.6°C; but after that, the heart rate increased in response to 38.3°C.
Figure 2. Temperature X Time Interaction for Heart Rate During Immersion

Systolic Blood Pressure During Water Immersion

Descriptive data for the dependent variable, change in systolic blood pressure, were divided into cells, each representing a group and a water temperature. Each water temperature was further divided into duration of immersion. These descriptive data are presented in Appendix D. An ANOVA was calculated using group, water temperature, and time as the independent variables. The difference the systolic blood pressure during immersion deviated from the systolic pressure immediately prior to entering the water served as the dependent variable. The ANOVA (see Table 5) indicated the following:

1. No difference in mean systolic blood pressure change, $F = 1.18$, was found between groups ($F(2, 23) = 3.44$, $p < .05$).

2. No difference in mean systolic blood pressure change, $F =$
1.10, was found between different water temperatures ($F(2, 46) = 3.23, p < .05$).

3. No difference in mean systolic blood pressure change, $F = 1.16$, was found for the first order interaction of temperature X group ($F(4, 46) = 2.61, p < .05$).

4. A significant difference in mean systolic blood pressure change, $F = 8.79$, was found between time of immersion ($F(3, 69) = 2.76, p < .05$).

5. No difference in mean systolic blood pressure change, $F = 0.99$, was found between the first order interaction of time X group ($F(6, 69) = 2.25, p < .05$).

6. No difference in mean systolic blood pressure change, $F = 0.80$, was found between the first order interaction of temperature X time ($F(6, 138) = 2.17, p < .05$).

7. No difference in mean systolic blood pressure change, $F = 0.64$, was found for the second order interaction of group X temperature X time ($F(12, 138) = 1.83, p < .05$).
Table 5
ANOVA Summary Table for Systolic Blood Pressure During Water Immersion

<table>
<thead>
<tr>
<th>Source</th>
<th>S.S.</th>
<th>df</th>
<th>M.S.</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Between Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group (G)</td>
<td>709.82</td>
<td>2</td>
<td>354.90</td>
<td>1.18</td>
</tr>
<tr>
<td>Error</td>
<td>6894.46</td>
<td>23</td>
<td>299.76</td>
<td></td>
</tr>
<tr>
<td><strong>Within Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature (TP)</td>
<td>601.35</td>
<td>2</td>
<td>300.67</td>
<td>1.10</td>
</tr>
<tr>
<td>T X G</td>
<td>1160.77</td>
<td>4</td>
<td>290.19</td>
<td>1.06</td>
</tr>
<tr>
<td>Error</td>
<td>12604.18</td>
<td>46</td>
<td>274.00</td>
<td></td>
</tr>
<tr>
<td>Time (TI)</td>
<td>459.14</td>
<td>3</td>
<td>153.05</td>
<td>8.79*</td>
</tr>
<tr>
<td>TI X G</td>
<td>103.87</td>
<td>6</td>
<td>17.31</td>
<td>0.99</td>
</tr>
<tr>
<td>Error</td>
<td>1201.46</td>
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<td>17.41</td>
<td></td>
</tr>
<tr>
<td>TP X TI</td>
<td>131.72</td>
<td>6</td>
<td>21.95</td>
<td>0.80</td>
</tr>
<tr>
<td>TP X TI X G</td>
<td>210.08</td>
<td>12</td>
<td>17.51</td>
<td>0.64</td>
</tr>
<tr>
<td>Error</td>
<td>3803.12</td>
<td>138</td>
<td>27.56</td>
<td></td>
</tr>
</tbody>
</table>

*F(3, 69) = 2.76, p < .05

For the comparison of systolic means associated with time of immersion, Tukey's HSD test for multiple comparisons was used to locate significant differences between the four times (see Table 6). The analysis of mean differences for time indicated that:

1. A significant difference, q = 6.27, existed between 3 and 6 minutes (F(4, 74) = 3.74, p < .05).
2. A significant difference, q = 6.21, existed between 3 and 9
2. A significant difference, $q = 6.21$, existed between 3 and 9 minutes ($q(4, 74) = 3.74, p < .05$).

3. A significant difference, $q = 4.18$, existed between 3 and 12 minutes ($q(4, 74) = 3.74, p < .05$).

4. No significant difference, $q = 0.05$, existed between 6 and 9 minutes ($q(4, 74) = 3.74, p < .05$).

5. No significant difference, $q = 2.09$, existed between 6 and 12 minutes ($q(4, 74) = 3.74, p < .05$).

6. No significant difference, $q = 2.04$, existed between 9 and 12 minutes ($q(4, 74) = 3.74, p < .05$).

Table 6
Multiple Comparison of Systolic Blood Pressure Means During Immersion by Time

<table>
<thead>
<tr>
<th>Time</th>
<th>3 Minutes</th>
<th>6 Minutes</th>
<th>9 Minutes</th>
<th>12 Minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 Minutes</td>
<td>-</td>
<td>6.27*</td>
<td>6.21*</td>
<td>4.18*</td>
</tr>
<tr>
<td>6 Minutes</td>
<td>6.27*</td>
<td>-</td>
<td>0.05</td>
<td>2.09</td>
</tr>
<tr>
<td>9 Minutes</td>
<td>6.21*</td>
<td>0.05</td>
<td>-</td>
<td>2.04</td>
</tr>
<tr>
<td>12 Minutes</td>
<td>4.18*</td>
<td>2.09</td>
<td>2.04</td>
<td>-</td>
</tr>
</tbody>
</table>

*$q(4, 74) = 3.74, p < .05$

Diastolic Blood Pressure During Water Immersion

Descriptive data for the dependent variable, change in diastolic blood pressure, were divided into cells, each representing a group and a water temperature. Each water temperature was further divided
into duration of immersion. These descriptive data are presented in Appendix E. An ANOVA was calculated using group, water temperature, and time as the independent variables. The amount the diastolic blood pressure during immersion deviated from the diastolic pressure immediately prior to entering the water served as the dependent variable. The ANOVA (see Table 7) indicated the following:

1. No difference in mean diastolic blood pressure change, $F = 2.63$, was found between groups ($F(2, 23) = 3.44, p < .05$).

2. No difference in mean diastolic blood pressure change, $F = 0.45$, was found between different water temperatures ($F(2, 46) = 3.23, p < .05$).

3. No difference in mean diastolic blood pressure change, $F = 1.75$, was found for the first order interaction of temperature X group ($F(4, 46) = 2.61, p < .05$).

4. A significant difference in mean diastolic blood pressure change, $F = 8.49$, was found between time of immersion ($F(3, 69) = 2.76, p < .05$).

5. No difference in mean diastolic blood pressure change, $F = 0.34$, was found between the first order interaction of time X group ($F(6, 69) = 2.25, p < .05$).

6. No difference in mean diastolic blood pressure change, $F = 0.92$, was found between the first order interaction of temperature X time ($F(6, 138) = 2.17, p < .05$).

7. No difference in mean diastolic blood pressure change, $F = 0.92$, was found for the second order interaction of group X temperature X time ($F(12, 138) = 1.83, p < .05$).
Table 7
ANOVA Summary Table for Diastolic Blood Pressure
During Water Immersion

<table>
<thead>
<tr>
<th>Source</th>
<th>S.S.</th>
<th>df</th>
<th>M.S.</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Between Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group (G)</td>
<td>587.34</td>
<td>2</td>
<td>293.67</td>
<td>2.63</td>
</tr>
<tr>
<td>Error</td>
<td>2563.70</td>
<td>23</td>
<td>111.47</td>
<td></td>
</tr>
<tr>
<td><strong>Within Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature (TP)</td>
<td>124.32</td>
<td>2</td>
<td>62.16</td>
<td>0.45</td>
</tr>
<tr>
<td>T X G</td>
<td>966.85</td>
<td>4</td>
<td>241.71</td>
<td>1.75</td>
</tr>
<tr>
<td>Error</td>
<td>6343.15</td>
<td>46</td>
<td>137.89</td>
<td></td>
</tr>
<tr>
<td>Time (TI)</td>
<td>718.96</td>
<td>3</td>
<td>239.65</td>
<td>8.49*</td>
</tr>
<tr>
<td>TI X G</td>
<td>57.66</td>
<td>6</td>
<td>9.61</td>
<td>0.34</td>
</tr>
<tr>
<td>Error</td>
<td>1947.36</td>
<td>69</td>
<td>28.22</td>
<td></td>
</tr>
<tr>
<td>TP X TI</td>
<td>125.85</td>
<td>6</td>
<td>20.98</td>
<td>1.45</td>
</tr>
<tr>
<td>TP X TI X G</td>
<td>160.08</td>
<td>12</td>
<td>13.34</td>
<td>0.92</td>
</tr>
<tr>
<td>Error</td>
<td>1997.41</td>
<td>138</td>
<td>14.47</td>
<td></td>
</tr>
</tbody>
</table>

*F(3, 69) = 2.76, p < .05

For the comparison of diastolic means associated with time of immersion, Tukey's HSD test for multiple comparisons was used to locate significant differences between the four times (see Table 8).

The analysis of mean differences for time indicated that:

1. No significant difference, q = 2.86, existed between 3 and 6 minutes (F(4, 74) = 3.74, p < .05).
2. A significant difference, $q = 4.73$, existed between 3 and 9 minutes ($q(4, 74) = 3.74$, $p < .05$).

3. A significant difference, $q = 6.86$, existed between 3 and 12 minutes ($q(4, 74) = 3.74$, $p < .05$).

4. No significant difference, $q = 1.88$, existed between 6 and 9 minutes ($q(4, 74) = 3.74$, $p < .05$).

5. A significant difference, $q = 4.00$, existed between 6 and 12 minutes ($q(4, 74) = 3.74$, $p < .05$).

6. No significant difference, $q = 2.13$, existed between 9 and 12 minutes ($q(4, 74) = 3.74$, $p < .05$).

Table 8

Multiple Comparison of Diastolic Blood Pressure During Immersion by Time Means

<table>
<thead>
<tr>
<th>Time</th>
<th>3 Minutes</th>
<th>6 Minutes</th>
<th>9 Minutes</th>
<th>12 Minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 Minutes</td>
<td>-</td>
<td>2.86</td>
<td>4.73*</td>
<td>6.86*</td>
</tr>
<tr>
<td>6 Minutes</td>
<td>2.86</td>
<td>-</td>
<td>1.88</td>
<td>4.00*</td>
</tr>
<tr>
<td>9 Minutes</td>
<td>4.73*</td>
<td>1.88</td>
<td>-</td>
<td>2.13</td>
</tr>
<tr>
<td>12 Minutes</td>
<td>6.86*</td>
<td>4.00*</td>
<td>2.13</td>
<td>-</td>
</tr>
</tbody>
</table>

$q(4, 74) = 3.74$, $p < .05$

Frequency of Arrhythmias During Water Immersion

The frequency of arrhythmias was recorded for the total duration of water immersion. Descriptive data for this dependent variable were divided into cells, each representing a medicated group and a
water temperature. These data are presented in Appendix F. An ANOVA was calculated using group and water temperature as the independent variables. Frequency of arrhythmias served as the dependent variable. The ANOVA (see Table 9) indicated the following:

1. A significant difference in frequency of arrhythmias, $F = 7.11$, was found between groups ($F(2, 23) = 3.44, p < .05$).

2. No significant difference in frequency of arrhythmias, $F = 1.09$, was found between different water temperatures ($F(2, 46) = 3.23, p < .05$).

3. No significant difference in frequency of arrhythmias, $F = 0.75$, was found for the interaction of temperature X group ($F(4, 46) = 2.61, p < .05$).

Table 9

<table>
<thead>
<tr>
<th>Source</th>
<th>S.S.</th>
<th>df</th>
<th>M.S.</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group (G)</td>
<td>22.19</td>
<td>2</td>
<td>11.10</td>
<td>7.11*</td>
</tr>
<tr>
<td>Error</td>
<td>35.92</td>
<td>23</td>
<td>1.56</td>
<td></td>
</tr>
<tr>
<td>Within Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature (TP)</td>
<td>1.94</td>
<td>2</td>
<td>0.97</td>
<td>1.09</td>
</tr>
<tr>
<td>T X G</td>
<td>2.66</td>
<td>4</td>
<td>0.67</td>
<td>0.75</td>
</tr>
<tr>
<td>Error</td>
<td>40.95</td>
<td>46</td>
<td>0.89</td>
<td></td>
</tr>
</tbody>
</table>

$*F(2, 23) = 3.44, p < .05$
For the comparison of the means associated with groups, Tukey's HSD test for multiple comparisons was used to locate significant differences between the three groups (see Table 10). The analysis of mean differences indicated that:

1. A significant difference, $q = 3.72$, existed between the Vasodilator/Beta Blocker and the Beta Blocker Groups ($q(3, 75) = 3.40, p < .05$).

2. No significant difference, $q = 1.54$, existed between the Vasodilator/Beta Blocker and Control Groups ($q(3, 75) = 3.40, p < .05$).

3. A significant difference, $q = 5.21$, existed between the Beta Blocker and Control Groups ($q(3, 75) = 3.40, p < .05$).

<table>
<thead>
<tr>
<th>Group</th>
<th>V/BB</th>
<th>BB</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasodilator/Beta Blocker (V/BB)</td>
<td>-</td>
<td>3.72*</td>
<td>1.54</td>
</tr>
<tr>
<td>Beta Blocker (BB)</td>
<td>3.72*</td>
<td>-</td>
<td>5.21*</td>
</tr>
<tr>
<td>Control (C)</td>
<td>1.54</td>
<td>5.21*</td>
<td>-</td>
</tr>
</tbody>
</table>

$q(3, 73) = 3.40, p < .05$
Heart Rate Immediately Post Immersion

The heart rate response immediately following water immersion was termed heart rate immediately post immersion. Descriptive data for this dependent variable were divided into cells, each representing a group and a water temperature. These data are presented in Appendix G. An ANOVA was calculated using group and water temperature as the independent variables. Change in heart rate served as the dependent variable. The ANOVA (see Table 11) indicated the following:

1. No significant difference in heart rate change, $F = 1.45$, was found between groups ($F(2, 23) = 3.44, p < .05$).

2. A significant difference in heart rate change, $F = 8.45$, was found between different water temperatures ($F(2, 46) = 3.23, p < .05$).

3. No significant difference in heart rate change, $F = 0.58$, was found for the interaction of temperature X group ($F(4, 46) = 2.61, p < .05$).
Table 11
ANOVA Summary Table for Heart Rate Immediately Post Immersion

<table>
<thead>
<tr>
<th>Source</th>
<th>S.S.</th>
<th>df</th>
<th>M.S.</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Between Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group (G)</td>
<td>377.74</td>
<td>2</td>
<td>188.87</td>
<td>1.45</td>
</tr>
<tr>
<td>Error</td>
<td>2995.76</td>
<td>23</td>
<td>130.25</td>
<td></td>
</tr>
<tr>
<td><strong>Within Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature (TP)</td>
<td>1456.73</td>
<td>2</td>
<td>728.36</td>
<td>8.45*</td>
</tr>
<tr>
<td>T X G</td>
<td>201.50</td>
<td>4</td>
<td>50.38</td>
<td>0.58</td>
</tr>
<tr>
<td>Error</td>
<td>3964.27</td>
<td>46</td>
<td>86.18</td>
<td></td>
</tr>
</tbody>
</table>

*F(2, 46) = 3.23, p < .05

For the comparison of the means associated with water temperature, Tukey's HSD test for Multiple comparisons was used to locate significant differences between the three temperatures (see Table 12). The analysis of mean differences indicated that:

1. No significant difference, $q = 2.56$, existed between 36.6° and 38.3°C ($q(3, 101) = 3.40, p < .05$).

2. A significant difference, $q = 9.84$, existed between 36.6° and 40.0°C ($q(3, 101) = 3.40, p < .05$).

3. A significant difference, $q = 7.28$, existed between 38.3° and 40.0°C ($q(3, 101) = 3.40, p < .05$).
Table 12
Multiple Comparison of Immediately Post Immersion Heart Rate Means by Temperature

<table>
<thead>
<tr>
<th>Temperature</th>
<th>36.6°C</th>
<th>38.3°C</th>
<th>40.0°C</th>
</tr>
</thead>
<tbody>
<tr>
<td>36.6°C</td>
<td>-</td>
<td>2.56</td>
<td>9.84*</td>
</tr>
<tr>
<td>38.3°C</td>
<td>2.56</td>
<td>-</td>
<td>7.28*</td>
</tr>
<tr>
<td>40.0°C</td>
<td>9.84*</td>
<td>7.28*</td>
<td>-</td>
</tr>
</tbody>
</table>

*q(3, 101) = 3.40, p < .05

Systolic Blood Pressure Immediately Post Immersion

The systolic blood pressure response immediately following water immersion was termed systolic blood pressure immediately post immersion. Descriptive data for this dependent variable were divided into cells, each representing a group and a water temperature. These data are presented in Appendix H. An ANOVA was calculated using group and water temperature as the independent variables. Change in systolic blood pressure served as the dependent variable. The ANOVA (see Table 13) indicated the following:

1. No significant difference in systolic blood pressure change, \( F = 1.82 \), was found between groups (\( F(2, 23) = 3.44, p < .05 \)).

2. No significant difference in systolic blood pressure change, \( F = 0.05 \), was found between different water temperatures (\( F(2, 46) = 3.23, p < .05 \)).

3. No significant difference in systolic blood pressure change, \( F = 0.24 \), was found for the interaction of temperature X group (\( F(4, 46) = 2.61, p < .05 \)).
Table 13
ANOVA Summary Table for Systolic Blood Pressure
Immediately Post Immersion

<table>
<thead>
<tr>
<th>Source</th>
<th>S.S.</th>
<th>df</th>
<th>M.S.</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group (G)</td>
<td>1987.37</td>
<td>2</td>
<td>993.69</td>
<td>1.82</td>
</tr>
<tr>
<td>Error</td>
<td>12533.91</td>
<td>23</td>
<td>544.95</td>
<td></td>
</tr>
<tr>
<td>Within Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature (TP)</td>
<td>32.89</td>
<td>2</td>
<td>16.45</td>
<td>0.05</td>
</tr>
<tr>
<td>T X G</td>
<td>336.13</td>
<td>4</td>
<td>84.03</td>
<td>0.24</td>
</tr>
<tr>
<td>Error</td>
<td>16068.59</td>
<td>46</td>
<td>349.32</td>
<td></td>
</tr>
</tbody>
</table>

Diastolic Blood Pressure Immediately Post Immersion

The diastolic blood pressure response immediately following water immersion was termed diastolic blood pressure immediately post immersion. Descriptive data for this dependent variable were divided into cells, each representing a medicated group and a water temperature. These data are presented in Appendix I. An ANOVA was calculated using group and water temperature as the independent variables. Change in diastolic blood pressure served as the dependent variable. The ANOVA (see Table 14) indicated the following:

1. No significant difference in diastolic blood pressure change, \( F = 3.14 \), was found between groups \( (F(2, 23) = 3.44, p < .05) \).

2. A significant difference in diastolic blood pressure change, \( F = 5.04 \), was found between different water temperatures \( (F(2, 46) = \)
3.23, $p < .05$).

3. A significant difference in systolic blood pressure change, $F = 5.17$, was found for the interaction of temperature X group ($F(4, 46) = 2.61, p < .05$).

Table 14
ANOVA Summary Table for Diastolic Blood Pressure Immediately Post Immersion

<table>
<thead>
<tr>
<th>Source</th>
<th>S.S.</th>
<th>df</th>
<th>M.S.</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group (G)</td>
<td>643.36</td>
<td>2</td>
<td>321.68</td>
<td>3.14</td>
</tr>
<tr>
<td>Error</td>
<td>2354.80</td>
<td>23</td>
<td>102.38</td>
<td></td>
</tr>
<tr>
<td>Within Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature (TP)</td>
<td>847.37</td>
<td>2</td>
<td>423.68</td>
<td>5.04*</td>
</tr>
<tr>
<td>T X G</td>
<td>1739.65</td>
<td>4</td>
<td>434.91</td>
<td>5.17**</td>
</tr>
<tr>
<td>Error</td>
<td>3866.81</td>
<td>46</td>
<td>84.06</td>
<td></td>
</tr>
</tbody>
</table>

*F(2, 46) = 3.23, $p < .05$  **F(4, 46) = 2.61, $p < .05$

For the comparison of the means associated with water temperature, Tukey's HSD test for multiple comparisons was used to locate significant differences between the three temperatures (see Table 15). The analysis of mean differences indicated that:

1. A significant difference, $q = 5.11$, existed between 36.6°C and 38.8°C ($q(3, 75) = 3.40, p < .05$).

2. A significant difference, $q = 8.15$, existed between 36.6°C and 40.0°C ($q(3, 75), = 3.40, p < .05$).

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3. No significant difference, $q = 3.04$, existed between $38.3^\circ$ and $40.0^\circ C$ ($q(3, 75) = 3.40$, $p < .05$).

Table 15

### Multiple Comparison of Immediately Post Immersion Systolic Blood Pressure Means by Temperature

<table>
<thead>
<tr>
<th>Temperature</th>
<th>$36.6^\circ C$</th>
<th>$38.3^\circ C$</th>
<th>$40.0^\circ C$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$36.6^\circ C$</td>
<td>-</td>
<td>$5.10^*$</td>
<td>$8.15^*$</td>
</tr>
<tr>
<td>$38.3^\circ C$</td>
<td>$5.10^*$</td>
<td>-</td>
<td>$3.03$</td>
</tr>
<tr>
<td>$40.0^\circ C$</td>
<td>$8.15^*$</td>
<td>$3.03$</td>
<td>-</td>
</tr>
</tbody>
</table>

*$q(3, 75) = 3.40$, $p < .05$

The interaction of water temperature X group was found to have a significant affect on diastolic pressure. This interaction is described in Figure 3. There was an inconsistent response between the groups to the different water temperatures. The control group had a consistent drop in pressure as the temperature increased. The Beta Blocker Group had a consistent increase in pressure as the temperature increased. The Vasodilator/Beta Blocker Group on the other hand, exhibited the lowest pressure from the middle temperature.
Discussion

The subjects of this study were prescribed varying dosages of medications that suppress the heart rate and blood pressure. Likewise, the individual heart rate and blood pressure response to exercise varied. In light of the possible medication effects and the fact that this study was conducted in a post exercise situation, all heart rate and blood pressure measurements were coded to reflect the amount of deviation from their respective post exercise measure, that was prior to entering the water, for that particular day.

Heart Rate During Water Immersion

The amount of change in heart rate across all temperatures did not differ significantly between the medicated and the Control
Groups. Means for the three groups were as follows: (a) Vasodilator/Beta Blocker, 2.66; (b) Beta Blocker, 0.98; and (c) Control, 0.85. This was not expected because the nature of the medication was to suppress heart rate. It could be that the temperature of the water was not hot enough to elicit a response great enough to distinguish between the groups.

Across all groups there were no significant differences between the response to 36.6°C and 38.3°C (\( \bar{X} = -0.55 \) and 0.22 respectively). However, 40°C (\( \bar{X} = 4.87 \)) did bring about a significantly greater mean increase in heart rates. This was consistent with findings reported by Bazett et al. (1937), Koroxenidis et al. (1961), Sancetta et al. (1958), Taggart et al. (1972), and Grollman (1930). The temperatures and temperature mediums varied in these four studies; however, they all reported an increase in heart rate with an increase in temperature. In this study the mean change in heart rate at 36.6°C was a decrease of .55 beats per minute (bpm). When the temperature was increased to 38.3°C, the heart rate increased by .77 bpm. It would appear from this study that it takes a temperature of at least 38.3°C to cause an increase in heart rate.

Taggart et al. (1972) reported that heart rates showed a progressive increase during the sauna, being roughly related to the time in the sauna. The significant difference in increased heart rate, across all groups over time in this study, supports those findings. There was a significant difference between all times except between three and six minutes (3 min. \( \bar{X} = 0.06 \), 6 min. \( \bar{X} = 0.64 \), 9 min. \( \bar{X} = 2.13 \), 12 min. \( \bar{X} = 3.23 \)). Although there was no significant differ-
ence between measures at the first two intervals, a review of the mean changes by temperature shows a steady increase in heart rate over time.

All groups showed an increase in heart rate over time across all temperatures (see Figure 1). As expected, the Control Group, unrestrained by medications, exhibited the greatest response over time, increasing a mean 5.44 bpm over the 12 minute exposure. The reason the Vasodilator/Beta Blocker Group showed a greater response (+3.29 bpm) than the strictly Beta Blocker Group (+1.17 bpm) is unclear. Of the vasodilating medications taken by the Vasodilator/Beta Blocker Group, only nitroglycerin could have had an effect on heart rate. All of these subjects were administered nitroglycerin at least one hour prior to observation. Any reflex tachycardia due to the nitroglycerin should have ceased by that point. It is possible that the sample size of this study was not large enough to elicit a true estimate of the population parameter.

Brown (1981) alluded to an interaction between temperature and duration of exposure in her recommendations on those topics. This is supported by a significant interaction of temperature and time of exposure affecting the heart rate response in this study. Although each temperature produced an increase in heart rate over time, the higher the temperature, the greater the magnitude of that increase (see Figure 2).

The investigator feels it is important to note that the greatest mean increase for any combination of medicated group or temperature was less than ten bpm (see Appendix C). It is her opinion that from
a clinical standpoint, a ten bpm increase over a resting heart rate, as a response to a stressor, has few practical implications.

**Blood Pressure During Water Immersion**

Bazett et al. (1937), Grollman (1930), Sancetta et al. (1958), Sherif et al. (1964), Taggert et al. (1972), and Turner (1980) all reported a decrease in blood pressure as a result of exposure to hyperthermia. This study, resulted in a mean decreased systolic pressure of 6.4 mm Hg and mean decreased diastolic pressure of 12.12 mm Hg, was in agreement with those findings. Taggert et al. found no difference in the response of the post myocardial infarction and the Control Groups. It was therefore not expected that infarction would be a factor in blood pressure response; however, it was expected that due to the nature of the medications separating the groups, there would be a significant difference between them. This was not the case (Vasodilator/Beta Blocker Group $\bar{X} = -4.73/-10.44$, Beta Blocker Group $\bar{X} = -8.45/-12.75$, and Control Group $\bar{X} = -6.40/-13.74$ mm Hg). Some possible reasons this did not occur are as follows:

1. Vasodilator and beta blocker medications do not significantly potentiate the blood pressure response to hyperthermia.

2. The sample size was insufficient to adequately estimate the true population parameter.

3. Subjects in this study were not taking sufficient doses of these medications to elicit a significant difference.

4. The temperatures in this study were not high enough to elicit a significant difference.
Because vasodilation reduces blood pressure and vasodilation is a general response to heat, an inverse relationship was expected to exist between blood pressure response and water temperature. Turner (1980) reported that upon standing after hyperthermic immersion, his three healthy, non-medicated subjects exhibited considerable hypotension. The diastolic blood pressure became unrecordable in two of the three subjects. This study failed to demonstrate any relationship between water temperature and blood pressure response \( (36.6^\circ C \bar{X} = -8.29/-11.71, 38.3^\circ C \bar{X} = -5.25/-11.56, 40^\circ C \bar{X} = -5.67/-13.09 \text{ mm Hg}) \). Possible explanations for this occurrence are as follows:

1. The sample size was insufficient to elicit a significant difference between temperatures.
2. The narrow range of temperatures used was not large enough to elicit a significant difference between temperatures.
3. The temperatures in this study were not high enough to elicit a significant difference between temperatures.

Both systolic and diastolic blood pressures showed a significant response over time \( (\bar{X} \text{ systolic decreased a } -4.43 \text{ to } -7.40 \text{ to } -7.37 \text{ to } -6.41 \text{ over the 12 minutes, diastolic } \bar{X} \text{ decreased a } -9.95 \text{ to } -11.67 \text{ to } -12.79 \text{ to } -14.08 \text{ over the 12 minutes}) \). The systolic response, however, contradicted that reported in the literature. Sancetta et al. (1958) reported a linear relationship between decrease in blood pressure and length of exposure to the heat. The results of this study indicated a significant difference between each three minute interval only when it is compared with the first three minute interval. Consequently it would appear that the systolic blood pressure
response occurred primarily in the first three minutes and was insignificant after that.

The diastolic blood pressure response on the other hand, was more in keeping with that reported by Sancetta et al. (1958). There were significant progressive decreases over the duration of exposure across all temperatures. It is interesting to note however, all significant decreases were between intervals of six or more minutes. This suggests that it takes more than three minutes for a response in the diastolic blood pressure to become significant.

From a clinical standpoint, it is the opinion of the investigator that the diastolic blood pressure responses demonstrated in this study may have practical implications. The largest mean decrease in diastolic blood pressure, seen in the Control Group at 40°C, dropped almost 20 mm Hg. A decrease of that magnitude could potentially produce symptoms in the individual, although none were reported in this study.

Frequency of Arrhythmias During Water Immersion

Taggart et al. (1972) reported ectopic beats in both post myocardial infarction and control subjects. Turner (1980) also observed ventricular ectopic beats in one of his healthy subjects. These findings, occurring across both healthy and diseased groups, might suggest that the arrhythmias were a response to the heat rather than a difference between groups. Contrary to those findings, this study showed a significant difference between medicated groups. The Control Group did not exhibit a single arrhythmia. The Vasodilator/
Beta Blocker Group did not differ significantly from the control ($\bar{X} = 0.37$). The Beta Blocker Group however, was significantly different from both other groups ($\bar{X} = 1.29$). It was expected that the two post infarction groups might exhibit a higher frequency of arrhythmias. The fact that the Vasodilator/Beta Blocker Group did not exhibit such a frequency could be explained by a decrease in coronary ischemia due to the vasodilating medications.

Another contradiction to the findings of Taggart et al. (1972) and Turner (1980) was the lack of a significant difference between water temperatures in this study. Though insignificant, a review of the marginal means for water temperatures also show an inverse relationship between frequency of arrhythmias and water temperature ($36.6^\circ \bar{X} = 0.73, 38.3^\circ \bar{X} = 0.46, 40^\circ \bar{X} = 0.38$). This could again be due to the vasodilating effect of heat reducing the demand on the heart and thus reducing the frequency of arrhythmias. The arrhythmias observed would appear to be an inherent nature of the groups and unrelated to immersion in water up to $40^\circ$C.

**Heart Rate Immediately Post Immersion**

An increase in heart rate upon rising, during a hyperthermic state, was the expected response. The hyperthermia was expected to cause vasodilation. The vasodilation would cause a drop in blood pressure and a decreased venous return. This in turn would cause a decrease in cardiac output which should elicit a compensatory increase in heart rate. Similar to the results of heart rate during water immersion, $40^\circ$C ($\bar{X} = 24.5$) caused a significantly greater
increase in heart rate than 36.6° or 38.3°C ($\bar{x} = 14.15$ and 16.85 respectively). There was no significant difference between the effects of 36.6° and 38.3°C. Although neither the drop in blood pressure nor the increase in heart rate was enough to be declared statistically significant between temperatures during water immersion, those indications of vasodilation did occur. Thus, it would appear that increased vasodilation during immersion at 40°C was probably responsible for the significant increase in heart rate at that temperature.

Although the heart rate response was significant from a statistical standpoint, it should be further evaluated from a clinical view. None of the subjects achieved a heart rate equal to or greater than the minimum of their individually prescribed exercise training range (60% maximum heart rate reserve). Therefore, in the opinion of the investigator, the heart rate response does not contraindicate a 12 minute immersion in 40°C water for any of the groups observed.

**Blood Pressure Immediately Post Immersion**

Systolic blood pressure was not significantly different between groups nor between temperatures, upon rising from a hyperthermic environment. Likewise, this variable was not significant during immersion. In both cases the increased heart rate could explain the maintenance of systolic pressure.

Diastolic blood pressure on the other hand, demonstrated a significantly greater decrease as the temperature rose (36.6° $\bar{x} = -1.55$, 38.3° $\bar{x} = -3.58$, 40° $\bar{x} = -8.30$). That effect, however, was
confounded by a significant interaction between temperature and medicated groups (see Figure 3). The three groups showed an inconsistent response to increasing temperature. The Vasodilator/Beta Blocker Group was expected to show the greatest decrease because the vasodilating medications were expected to compound the effects of the hyperthermic vasodilation. However, the greatest decrease for that group was observed at 38.3°C ($\bar{X} = -5.33$) with a decrease of only half that at 40°C ($\bar{X} = -2.44$). It was the Control Group that experienced the greatest drop at the highest temperature ($\bar{X} = -20.22$). Similarly, the only two subjects to complain of lightheadedness after exiting the hot tub were from the Beta Blocker and the Control Groups. It should also be noted that the Beta Blocker Group showed an inverse relationship to temperature ($36.6^\circ\bar{X} = -5.75$, $38.3^\circ\bar{X} = -3.25$, $40^\circ\bar{X} = -1.75$).
CHAPTER V

SUMMARY, FINDINGS, CONCLUSIONS AND RECOMMENDATIONS

Summary

This study was conducted to compare the heart rate, blood pressure, and electrocardiographic response of post myocardial infarction males using two different classes of cardiac medications, to three different water temperatures in a hot tub. The comparison of both the effect of increasing water temperature on each subgroup, and the response of the subgroups at each water temperature were simultaneously investigated. Twenty-six adult males were divided into three groups based on cardiovascular disease status and prescribed medications. There were nine in the Vasodilator/Beta Blocker Group, eight in the Beta Blocker Group, and nine in the Control Group. Each subject engaged in 30 minutes of aerobic exercise, cooled down, and then sat in a hot tub for 12 minutes. Each subject repeated this procedure three times, once at each of the following water temperatures: (a) 36.6°C, (b) 38.3°C, and (c) 40°C. Heart rate and blood pressure were determined just prior to entering the water, at three minute intervals during the immersion, and immediately upon exiting the tub. A frequency of arrhythmias was determined for the duration of immersion at each temperature.

Raw data were coded and analyzed using the Computer Program BMDP. An Analysis of Variance, Split Plot Factorial design with fixed effects was used. Tukey's HSD Multiple Comparison Test was
used to determine significant differences between means (Hopkins and Glass, 1978). The independent variables included: (a) subject groups with three levels, Vasodilator/Beta Blocker, Beta Blocker, and Control; (b) water temperature with three levels, 36.6°, 38.8°, and 40°C; and (c) duration of exposure with four levels, 3, 6, 9, and 12 minutes. This study included four dependent variables, heart rate, systolic blood pressure, diastolic blood pressure, and frequency of arrhythmia.

Findings

Significance for the findings of this study was determined at the .05 level. The ANOVA and multiple comparison tests used to determine the effects of the independent variables indicated the following:

1. Changes in heart rate during immersion were related to increases in temperature and duration of exposure, but not to the groups.

2. Changes in blood pressure during immersion, both systolic and diastolic, were related to length of exposure only.

3. The only factor to influence frequency of arrhythmias was the difference in groups.

4. Increases in heart rate immediately post immersion were consistent across all groups and related only to differences in temperature.

5. There were no significant differences in systolic blood pressure immediately post immersion as a result of any of the
variables.

6. The effects of temperature on the diastolic blood pressure immediately post immersion were confounded by an interaction with group differences.

Conclusions

The effects of water temperature, duration of immersion in a hot tub, and group differences on heart rate, blood pressure, and frequency of arrhythmias led the investigator to suggest the following conclusions:

1. There was no evidence to suggest that the groups responded any differently, in terms of heart rate or blood pressure.

2. There was evidence to suggest that water temperature was a factor in elevating the heart rate.

3. There was no evidence to suggest that the temperature of the water had any effect on the blood pressure response or frequency of arrhythmias.

4. There was evidence to suggest a difference between groups based on frequency of arrhythmias.

5. There was evidence to suggest a relationship between duration of immersion and heart rate and blood pressure response.

Recommendations

Based on the results of this study, a 12 minute immersion in water up to 40°C by post myocardial infarction males taking vasodilator and/or beta blocker medications should not be restricted.
Further studies could investigate the following:

1. A larger sample size could be observed.
2. The duration of exposure could be observed over a longer period of time.
3. The effects of temperatures greater than 40°C could be explored.
4. The cardiovascular response could be compared between groups that had exercised prior to use of the hot tub and those who did not.
5. Body temperature could be measured both pre and post immersion.
Appendix A

Consent Form
APPENDIX A

Consent Form

Purpose:
The purpose of this investigation is to compare the cardiovascular response after exercise to three different water temperatures in a hot tub. The information obtained will be used by Janeen Docsa with similar information from 35 other participants to complete a thesis. This thesis is a partial requirement toward the completion of a master's degree from Western Michigan University.

Explanation:
I will need to complete three immersions in the hot tub, one with the water at 98°F, another at 101°F and another at 104°F. I will not know the order of the water temperatures experienced until the study is complete. Prior to each hot tub immersion I will exercise at my prescribed exercise intensity for 30 minutes. To begin each session, I will have two adhesive electrodes applied to my chest and connected to a EKG telemetry unit. A resting heart rate and blood pressure will be recorded. I will then sit myself in the hot tub. While sitting, my EKG, heart rate and blood pressure will be monitored for twelve minutes. Following this, I will exit the tub, be seated and again be monitored for ten minutes. The period in the water may be cut short if a problem develops.

Average changes in heart rate, blood pressure and heart rhythm for all participants will be determined. I will receive a summary of the results when all data is in and results have been compiled.

Risks:
There is a possibility of certain changes occurring during the exposure to hot water. They include: abnormal blood pressure, dizziness, fainting, irregular heartbeats and the remote possibility of a heart attack. Every effort will be made to minimize any risks by preliminary questioning and observation during the session. All monitoring personnel are trained in basic cardiac life support and emergency equipment is available in the facility should a problem arise. An on-call emergency team will also be available from Borgess Medical Center. Any emergency assistance which may be needed that is not covered by my medical insurance, will be provided at no expense to me.

There also exists the risk of skin infection. To minimize this possibility, the chlorine and pH levels will be closely monitored and maintained.
Inquiries:
Any question about the procedures are welcome. I will be given information throughout the session and all procedures will be explained.

Confidentiality:
I understand that the information obtained from the three hot tub immersions I will undergo will be kept confidential. The information will be coded to protect individual identity. My name will not appear in any publication related to this study. Following the completion of the study, all individual data will be destroyed or returned to me at my request.

Freedom of Consent:
I understand that participation in this study is voluntary and refusal to participate will involve no penalty or loss of benefits to me. I also understand that I may discontinue participation at any time without penalty. I will inform the investigator if I do not wish to participate.

I HAVE READ THIS FORM AND UNDERSTAND THE PROCEDURE I WILL BE INVOLVED IN. I AGREE TO PARTICIPATE IN THIS STUDY.

Date:___________ Signed:________________________

Witnessed:__________________
Appendix B

Data Collection Worksheet

68
Medications: ____________________

70 - 85% Max HR Reserve: ______ to ______
Intensity today: ______ to ______
Duration of exercise: ______ minutes
Hours of sleep last night? ______
Hours since last meal? ______
Smoker? ______
Time of last meds.? ______

NTG taken during exercise? ______
Caffeine today? ______
Unusual chest pain in last 24 hrs? ______

Unusual dizziness or lightheadedness in last month? ______
Illness or injury in last 2 weeks? ______

Any discomforts right now? ______

Height ______ in ______ cm
Weight ______ lb ______ kg

<table>
<thead>
<tr>
<th></th>
<th>HR</th>
<th>BP</th>
<th>Arrhythmias</th>
<th>Comments</th>
</tr>
</thead>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post Ex.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre Water</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12 min</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I.P.W.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 min.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Group ______ Code ______ Name ____________

APPENDIX B
Appendix C

Table of Cell Means for Heart Rate During Water Immersion
### APPENDIX C

Cell Means for Heart Rate During Water Immersion

<table>
<thead>
<tr>
<th>Temp./Time</th>
<th>Vasodilator/</th>
<th>Beta Blocker Beta Blocker Control Marginal</th>
</tr>
</thead>
<tbody>
<tr>
<td>36.6°C/ 3 min.</td>
<td>0.78</td>
<td>-0.50</td>
</tr>
<tr>
<td>6 min.</td>
<td>0.66</td>
<td>-0.38</td>
</tr>
<tr>
<td>9 min.</td>
<td>1.11</td>
<td>-0.63</td>
</tr>
<tr>
<td>12 min.</td>
<td>1.67</td>
<td>-1.38</td>
</tr>
<tr>
<td>38.3°C/ 3 min.</td>
<td>-1.11</td>
<td>0.50</td>
</tr>
<tr>
<td>6 min.</td>
<td>-0.11</td>
<td>0.00</td>
</tr>
<tr>
<td>9 min.</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>12 min.</td>
<td>2.11</td>
<td>1.88</td>
</tr>
<tr>
<td>40.0°C/ 3 min.</td>
<td>4.11</td>
<td>0.13</td>
</tr>
<tr>
<td>6 min.</td>
<td>4.67</td>
<td>3.00</td>
</tr>
<tr>
<td>9 min.</td>
<td>7.11</td>
<td>3.38</td>
</tr>
<tr>
<td>12 min.</td>
<td>9.89</td>
<td>4.75</td>
</tr>
<tr>
<td>Marginal</td>
<td>2.66</td>
<td>0.98</td>
</tr>
</tbody>
</table>

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Appendix D

Table of Cell Means for Systolic Blood Pressure During Water Immersion
### APPENDIX D

**Cell Means for Systolic Blood Pressure During Immersion**

<table>
<thead>
<tr>
<th>Temp./Time</th>
<th>Vasodilator/</th>
<th>Beta Blocker</th>
<th>Beta Blocker</th>
<th>Control</th>
<th>Marginal</th>
</tr>
</thead>
<tbody>
<tr>
<td>36.6°C/ 3 min.</td>
<td>-4.67</td>
<td>-8.00</td>
<td>-6.89</td>
<td>-6.46</td>
<td></td>
</tr>
<tr>
<td>6 min.</td>
<td>-6.44</td>
<td>-15.00</td>
<td>-10.67</td>
<td>-10.54</td>
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</tr>
<tr>
<td>9 min.</td>
<td>-5.78</td>
<td>-12.25</td>
<td>-7.56</td>
<td>-8.38</td>
<td></td>
</tr>
<tr>
<td>12 min.</td>
<td>-4.44</td>
<td>-12.50</td>
<td>-6.89</td>
<td>-7.77</td>
<td></td>
</tr>
<tr>
<td>38.3°C/ 3 min.</td>
<td>-4.44</td>
<td>-1.00</td>
<td>-2.89</td>
<td>-2.85</td>
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<tr>
<td>6 min.</td>
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<td>-5.25</td>
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<td>9 min.</td>
<td>-8.56</td>
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<td>-4.22</td>
<td>-6.65</td>
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</tr>
<tr>
<td>12 min.</td>
<td>-8.22</td>
<td>-7.50</td>
<td>-2.67</td>
<td>-6.08</td>
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</tr>
<tr>
<td>40.0°C/ 3 min.</td>
<td>1.78</td>
<td>-8.25</td>
<td>-6.00</td>
<td>-4.00</td>
<td></td>
</tr>
<tr>
<td>6 min.</td>
<td>-2.22</td>
<td>-9.00</td>
<td>-7.78</td>
<td>-6.23</td>
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<td>9 min.</td>
<td>-3.77</td>
<td>-7.75</td>
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<td>-7.08</td>
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</tr>
<tr>
<td>12 min.</td>
<td>-2.67</td>
<td>-7.75</td>
<td>-6.00</td>
<td>-5.38</td>
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<tr>
<td>Marginal</td>
<td>-4.73</td>
<td>-8.46</td>
<td>-6.25</td>
<td>-6.40</td>
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Appendix E

Table of Cell Means for Diastolic Blood Pressure During Water Immersion
## APPENDIX E

### Cell Means for Diastolic Blood Pressure During Immersion

<table>
<thead>
<tr>
<th>Temp./Time</th>
<th>Vasodilator/</th>
<th>Beta Blocker</th>
<th>Beta Blocker</th>
<th>Control</th>
<th>Marginal</th>
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</thead>
<tbody>
<tr>
<td>36.6°C</td>
<td>3 min.</td>
<td>-10.67</td>
<td>-10.50</td>
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<tr>
<td>6 min.</td>
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<td>-10.00</td>
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<td>9 min.</td>
<td>-11.33</td>
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<td>-10.00</td>
<td>-11.54</td>
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</tr>
<tr>
<td>12 min.</td>
<td>-11.78</td>
<td>-16.50</td>
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</tr>
<tr>
<td>38.3°C</td>
<td>3 min.</td>
<td>-6.00</td>
<td>-9.50</td>
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<td>-8.23</td>
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<td>6 min.</td>
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<td>9 min.</td>
<td>-12.44</td>
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<td>-10.22</td>
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<td>-18.22</td>
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<tr>
<td>12 min.</td>
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<td>-12.75</td>
<td>-19.11</td>
<td>-15.15</td>
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Appendix F

Table of Cell Means for Frequency of Arrhythmias During Water Immersion
# APPENDIX F

**Cell Means for Frequency of Arhythmias**

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<th>Temp.</th>
<th>Vasodilator/</th>
<th>Beta Blocker</th>
<th>Beta Blocker</th>
<th>Control</th>
<th>Marginal</th>
</tr>
</thead>
<tbody>
<tr>
<td>36.6°C</td>
<td>0.44</td>
<td>1.88</td>
<td>0.00</td>
<td></td>
<td>0.73</td>
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<td>38.3°C</td>
<td>0.44</td>
<td>1.00</td>
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Appendix G

Table of Cell Means for Heart Rate Immediately Post Immersion
## APPENDIX G

Cell Means for Heart Rate Immediately Post Immersion

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<th>Vasodilator/ Beta Blocker</th>
<th>Beta Blocker</th>
<th>Control</th>
<th>Marginal</th>
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<tr>
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<td>16.11</td>
<td>11.75</td>
<td>14.33</td>
<td>14.15</td>
</tr>
<tr>
<td>38.3°C</td>
<td>15.00</td>
<td>15.38</td>
<td>20.00</td>
<td>16.85</td>
</tr>
<tr>
<td>40.0°C</td>
<td>25.67</td>
<td>19.13</td>
<td>28.11</td>
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<td>Marginal</td>
<td>18.93</td>
<td>15.41</td>
<td>20.81</td>
<td>18.50</td>
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Appendix H

Table of Cell Means for Systolic Blood Pressure
Immediately Post Immersion
### APPENDIX H

#### Cell Means for Systolic Blood Pressure
##### Immediately Post Immersion

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<th>Temp.</th>
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<th>Beta Blocker</th>
<th>Control</th>
<th>Marginal</th>
</tr>
</thead>
<tbody>
<tr>
<td>36.6°C</td>
<td>1.11</td>
<td>-10.25</td>
<td>-4.22</td>
<td>-4.23</td>
</tr>
<tr>
<td>38.3°C</td>
<td>2.00</td>
<td>-5.25</td>
<td>-6.00</td>
<td>-3.00</td>
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<tr>
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<td>7.33</td>
<td>-8.75</td>
<td>-7.78</td>
<td>-2.85</td>
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<tr>
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<td>-8.08</td>
<td>-6.00</td>
<td>-3.36</td>
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</table>

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Appendix I

Table of Cell Means for Diastolic Blood Pressure
Immediately Post Immersion

<p>| | | | |</p>
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<tr>
<td>82</td>
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<td></td>
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</tbody>
</table>
### APPENDIX I

**Cell Means for Diastolic Blood Pressure Immediately Post Immersion**

<table>
<thead>
<tr>
<th>Temp.</th>
<th>Vasodilator/</th>
<th>Beta Blocker</th>
<th>Beta Blocker</th>
<th>Control</th>
<th>Marginal</th>
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<td>-5.8</td>
<td>2.22</td>
<td>2.22</td>
<td>0.08</td>
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<tr>
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<td>-1.75</td>
<td>-20.22</td>
<td>-8.39</td>
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<tr>
<td>Marginal</td>
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<td>-3.58</td>
<td>-8.30</td>
<td>-4.52</td>
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</table>
BIBLIOGRAPHY


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