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THE EFFECTS OF ICED WATER INGESTION ON HEART RATE, ELECTROCARDIOGRAM, AND BLOOD PRESSURE

The University of Arizona

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THE EFFECTS OF ICED WATER INGESTION ON HEART RATE,
ELECTROCARDIOGRAM, AND BLOOD PRESSURE

by
Kathryn Lea Kerr

A Thesis Submitted to the Faculty of the
COLLEGE OF NURSING
In Partial Fulfillment of the Requirements
For the Degree of
MASTER OF SCIENCE
In the Graduate College
THE UNIVERSITY OF ARIZONA
1987
STATEMENT BY AUTHOR

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Date: 1/13/87
ACKNOWLEDGEMENTS

The author wishes to express sincere appreciation to the dear family and friends whose encouragement made possible the completion of this project. Most especially the author thanks Mom, Bogie and John Przybyla.
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ABSTRACT

An experimental design was used to determine the effects of 240 ml of 0-5°C water on heart rate, ST segments and T waves of the 12-lead electrocardiogram, and systolic and diastolic blood pressures. The study sample consisted of 20 normal subjects recruited from a southwestern town. Each subject was tested under both experimental and control conditions. Baseline electrocardiograms and blood pressures were recorded on all subjects. Under the experimental condition, subjects drank 240 ml of 0-5°C water. Under the control condition, they drank 240 ml of 22-26°C water. Repeat electrocardiograms were obtained at 2, 5, 10, and 30 minutes after the water was consumed.

Heart rate per minute and ST segment and T wave changes were measured by the electrocardiogram. Analysis of variance for repeated measures revealed a statistically significant, through clinically insignificant, increase in systolic blood pressure in the experimental group over time. No significant difference was noted in heart rate or diastolic blood pressure between groups. Data were insufficient to analyze ST segment and T wave changes by repeated measures ANOVA.
CHAPTER 1

INTRODUCTION

For many years, cold water has been withheld from patients suspected of having a recent myocardial infarction. It was believed that cold water had a deleterious effect on the recently injured heart, or upon its function (Caplin, 1984; Pratte, Padilla, & Baker, 1973).

The distended fundus of the stomach, cooled as a result of ice water ingestion, comes in direct contact with the posterio-inferior surface of the left ventricle. The epicardium of the left ventricle, thus cooled, was believed to retard the ventricular repolarization process of the heart (Dowling & Hellerstien, 1951). Cooling of the heart has also been theorized to extend an area of myocardial ischemia (Dowling & Hellerstein, 1951; Houser, 1976), or to change the cardiac rhythm (Kirchhoff, 1981).

Recent medical practices, however, such as the thermodilution measurement of cardiac output and cardioplegia during open heart surgery, have not resulted in the complications of retarded ventricular repolarization, increased myocardial ischemia, or changes in cardiac rhythm. In the technique of thermodilution, 10 ml of 0.5° to 5° C isotonic dextrose solution is injected directly into the right atrium. The change in blood temperature which follows is measured in the pulmonary artery. Cardiac input is in inverse proportion to the fall in
temperature (Ganz, Donoso, Marcus, Forrester, & Swan, 1971). Cardioplegia is the use of a cold solution which contains an arresting agent at the time of ischemic arrest during open heart surgery. This solution cools the myocardium to approximately 15° C, thus reducing the metabolic requirements of the cells. Not only does this cooling not result in ischemia, but in fact, cardioplegia reduces the rate of perioperative myocardial infarction (Sanderson & Kurth, 1983). Despite the uses of cold solutions in cardiac procedures with patients having coronary artery disease, a significant number of coronary care units continue to deny patients cold beverages (Cohen, Alpert, Francis, Vieweg, & Hagan, 1977; Andreoli, Powkes, Zipes, & Wallace, 1983).

The effect of cold water ingestion on cardiac function has been studied extensively. In previous studies, the drinking of iced water was associated with increased T wave negativity in various leads of the electrocardiogram (Wilson & Finch, 1923; Dowling & Hellerstein, 1951). Prolongation of the ST segment, decrease in heart rate, and increase in systolic blood pressure were also associated with ingestion of iced water (Pratte, et al., 1973). These reported changes in cardiac function followed intake of 600 to 800 ml of cold water. When smaller amounts of iced water had been consumed, changes in cardiac function did not follow. Significant changes in electrocardiogram (EKG), heart rate, and blood pressure were not reported after drinking 150 to 240 ml of cold water (Pratte, et al., 1973; Fitzmaurice & Simon, 1974; Houser, 1976; Cohen, et al., 1977; Siegal & Sparks, 1980).
The relationship between iced water consumption and cardiac function remains unclear. Conflicting results of studies provide the rationale for investigating the effects of 240 ml of iced water on heart rate, EKG, and blood pressure. The purpose of this investigation is outlined.

**Study Purpose**

The purpose of this study is to investigate the effects of iced water ingestion on heart rate, electrocardiogram, and blood pressure.

**Significance of the Problem**

Iced water is often withheld from clients who have recently sustained a myocardial infarction, although the rationale for this nursing practice does not seem based on sufficient scientific evidence (Siegal & Sparks, 1980; Houser, 1976). Denial of moderate amounts of cold water may be a needless frustration to individuals already faced with an abrupt change in diet and activity while hospitalized following a heart attack.

Research is necessary to determine the efficacy of this nursing intervention in assisting an individual to increase his level of cardiac wellness. With increasing health care costs, nurses are expected to justify the expense of their time in delivering services. Documented effectiveness of specific nursing actions is the means to defend nursing behavior, and thus, time spent with the client. Lack of demonstrated value for a nursing intervention is cause for its modification or abandonment (Pilot & Hungler, 1983).
Summary

A review of the studies relating oral intake of cold fluids to cardiac function indicated a need for further investigation. This study was intended to determine the effects of a single ingestion of 240 ml of 0° to 5° C water on heart rate, electrocardiogram, and blood pressure.
CHAPTER 2

THE THEORETICAL FRAMEWORK

The purpose of this study was to investigate the effects of ingestion of a single glass of iced water on heart rate, electrocardiogram, and blood pressure. In order to accomplish this purpose, the concepts and their postulated relationships must be clearly delineated. A theoretical framework describing the possible relationship between oral iced water ingestion and cardiovascular changes is presented in this chapter. Previously conducted studies describing associations between these concepts are reviewed.

The theoretical framework (see Figure 1) is derived from the postulated physiological actions of temperature upon the cardiovascular system. Temperature is believed to affect cardiac function by changing the self-excitation process of the myocardial cells. Self-excitation is the rhythmic ion exchange across the cardiac cell membrane, an exchange called the action potential. The action potential is followed by the contraction of the atrial, and then ventricular, syncytia, resulting in the pumping of blood by the heart. Heat, it is believed, causes an increased permeability of the myocardial cell membrane to ions, thus accelerating the ion exchange process. Therefore, increased temperature increases the heart rate, and decreased temperature causes a decrease in heart rate (Guyton, 1986). An increase in temperature has also been
Figure 1. Model of Temperature and Cardiovascular Responsiveness
found to transiently increase contractile strength of the heart in vitro, though after a period of time, the metabolic systems of the heart become exhausted and weakness ensues (Guyton, 1986). The effects of temperature on heart function are ultimately explained at the cellular level by what effects temperature may have on the ionic exchange which is responsible for initiation of the heart beat. The results of studies investigating the relationship between temperature and alterations in cardiac function are discussed as follows.

Temperature and Cardiovascular Changes

The action of temperature upon cardiac function have been studied by cooling and heating myocardial cells both directly and indirectly. In 1941, Hoff and Nahum placed a hollow tin chamber directly over canine hearts. Water of 55°C, and alternately, 5°C was circulated through the chamber while electrocardiograms of both right and left ventricles were recorded. In all of the seven canine subjects, the R-R intervals of the electrocardiograms were shortened by the warm water and lengthened after application of cold water. Vectors of the T waves were altered also; these changes in magnitude and direction of the T wave could be predicted and explained by the effects of heat and cold on the action potential of the myocardial cells.

Action potentials are recordings of the rapid reversals of the voltage difference between the inside and outside of the cell membrane (Vander, Sherman & Luciano, 1980). These action potentials determine the duration of cardiac contraction (Guyton, 1986). In its resting
state, the myocardial cell membrane is positively charged on the outside and negatively charged on the inside. The action potential is a rapid reversal of the resting negative value to positive intracellularly, and an extracellular reversal to negative. Abruptly, after 0.2 seconds in atrial muscle and 0.3 seconds in ventricular muscle, the cell membranes return to the resting potential in a process called repolarization (Guyton, 1986).

Hoff and Nahum found, for example, that cooling of the left ventricle or heating of the right ventricle increased the negativity of the T waves of the left ventricular EKG, or levocardiogram. This increased T wave negativity could be explained by prolonged repolarization of the left ventricle relative to the right (Hoff & Nahum, 1941).

In a follow-up study, using the same method of circulating 5°C and 55°C water over canine ventricles, Nahum, Hoff and Kaufman (1941) recorded not only electrocardiograms, but also monophasic action potentials while altering the temperature of the myocardium. Two electrocardiogram leads were recorded: one from the anterior surface of the right ventricle and the other from the anterior surface of the left ventricle. As expected, the T wave from the right ventricular lead, normally negative, became increasingly negative after application of 55°C water and became upright after application of 5°C water. Also normally having a negative deflection, the T wave of the left ventricular lead became upright after 55°C water was circulated over it, and more negative after circulation of 5°C water. Again, these T
wave changes could be explained by the prolongation or acceleration of ion exchange due to cold and heat, respectively.

In this experiment by Nahum, et al. (1941), monophasic action potentials were recorded from the same locations on the myocardium as were the electrocardiograms, enabling the investigators to demonstrate that indeed, cooling of the myocardium with 5°C water prolonged the action potential of that area of muscle, while warming with 55°C water was followed by a shortened action potential. Findings of this experiment in five canine hearts thus demonstrated that "alterations in the rate of recovery at an isolated region of the surface of the heart result from changes in temperature" (Nahum, et al., 1941, p. 399).

Hellerstein and Liebow (1950) produced electrocardiogram changes by cooling and heating the epicardium of 10 anesthetized dogs. The temperature of the heart's subendocardial surface was changed by injecting normal saline directly into the heart through intracavitary catheter electrodes. Electrocardiograms from both epicardial and intracavitary leads were recorded before, during, and after saline injection. Twenty ml of 5° to 8°C normal saline was used to cool the endocardium of the left ventricular apex, the endocardium of the right ventricular apex, and the endocardial surface of the posterior basal portion of the left ventricle. After cooling the endocardia in this fashion, there were consistent electrocardiographic changes. In both epicardial and intracavitary leads, QT was prolonged in every experiment; R-R interval slowed immediately when the right ventricle
was cooled. In the intracavitary leads, T waves became wider and more deeply negative with cooling of the apical and posterior endocardium.

When the left apical endocardium was cooled, the epicardial lead immediately above this area recorded wider, higher T waves than the controls. With cooling of the right ventricular apical endocardium, precordial leads located over the intraventricular septum also showed an increase in amplitude and duration of the T wave. After cooling of the endocardium of the basal, posterior, left ventricle, T waves in the anterior epicardial lead became markedly inverted.

Heating the endocardium of the left ventricular apex consistently resulted in T waves, previously negative to the isoelectric line, becoming upright in the intracavitary leads. In the epicardial leads, with control T waves being upright, intracavitary injection of 20 ml 45° to 60°C normal saline was followed by markedly inverted T waves. Effects of cardiac temperature changes from the intracavitary injections were immediate, and resolved within 10 seconds to two minutes.

Hellerstein and Liebow (1950) also produced temperature changes on canine epicardia by two methods. In dogs with intact chests, the intact epicardium was sprayed with 20 ml of hot or cold saline through a needle inserted into the fourth or fifth intercostal space near the left parasternal line. Epicardial temperature was changed in open chest preparations by local application of ice cubes, or by a stream of hot or cold solutions. The temperatures of the solutions and ice cubes were not stated.
Cooling of the left ventricular epicardium prolonged the QT interval in all leads. In the intracavitary leads, normally negative T waves became upright and broad. The epicardial leads, whose control T waves were upright, became negative and broad. These electrocardiographic changes were transient, resolving in less than three minutes.

Heating the left ventricular epicardium was followed by T wave changes in all experiments. Heating any part of the left ventricular epicardium produced large, negative T waves in the intracavitary lead directly beneath that area.

According to Hellerstein and Liebow (1950), the T wave changes produced by heating or cooling areas of the heart resulted from changes in the electrical state of the myocardium. Ventricular repolarization, the return of the muscle to its resting electrical state after an action potential, is recorded as the T wave of the electrocardiogram (Guyton, 1986). The normal direction of repolarization is always from the epicardium to the endocardium. T waves in an EKG lead are negative whenever the myocardium nearest the electrode recovers later than other areas (Hellerstein & Liebow, 1950). Thus, negative waves were seen in the control intracavitary leads while positive T waves were seen in the control epicardial leads.

T wave negativity was increased in these experiments where the electrode was in the middle of an area with retarded repolarization. Positivity was increased in leads where the electrode was distant to an area of slower repolarization and separated from this area by muscle of relatively faster repolarization.
Once again, T wave changes can be explained by the effect of heating and cooling on the cardiac cells' action potential. For example, more negative T waves were produced in the endocardial EKG lead when the endocardium was cooled, or when the endocardium was not treated and the epicardium was heated. In these instances, the exploring electrode was within an area of retarded repolarization due to local cooling or due to relative coolness in relationship to the epicardium.

As in previous studies, the experimental findings of Hellerstein and Liebow (1950) illustrated that heating an area of heart muscle increases the intensity and rate of its repolarization, while cooling an area retards the return of the electrical resting state of the myocardium.

Bender, Austen, Tsunekawa and Ebert (1963) investigated the site of action of hypothermia on myocardial contraction in dogs by employing separate cardiopulmonary and systemic perfusion systems. In nine of the dogs, the systemic blood was cooled until a core temperature of 15°C was reached, while the heart was kept normothermic. With four dogs, systemic blood was kept normothermic while the heart and lungs were cooled. In three animals, the pulmonary circulation was cooled to 15°C while the heart and systemic circulation were kept at normal temperature.

In these experiments, no change in heart rate occurred when the entire body was cooled while the heart and lungs were kept at normal temperature, or when the lungs alone were cooled. However, when the
heart and lungs were cooled until the cardiac temperature was 25°C, the heart rate decreased by 67% from its normothermic rate. The findings of these experiments demonstrated that the decrease in heart rate which occurs with hypothermia is from the direct effect of cold on the heart (Bender, et al., 1963).

In another attempt to study the effect of temperature on heart muscle devoid of extracardial influence, Moore, Marable and Ogden (1966) experimented with contracting strips of rat ventricle. Ventricle strips were immersed in a isotonic bath between two electrodes. Contractions were elicited every second, while force of contraction was recorded. Each strip was stretched by increments of 0.1mm until it was 18% more than original length. At the same time, the bath temperature was varied over a range of 24° to 41°C. Little temperature influence upon contractility was observed until the bath reached the temperature of 40°C. At this point, a marked decrease in contractile force and cardiac output were demonstrated. On the basis of these findings, Moore, et al. (1966) believed that the decreased cardiac output resulting from lower temperatures in intact heart preparations was due to "some extracardiac factor" (Moore, et al., 1966, p. 450).

Oral Cold or Hot Fluids and Heart Rate, Ischemia, and Blood Pressure

The influence of orally ingested cold or hot fluids upon cardiac function has been studied by several researchers. The majority of these experiments used human beings as subjects, and used, of course, intact chest preparations.
In 1923, Wilson and Finch conducted five experiments with normal adult men as subjects. Subjects drank 600 ml of 40°F (14.5°C) water while lying on a couch. Limb leads I, II, and III of the electrocardiogram were recorded before and after the water was consumed. Immediately following ingestion of the cold water, in all experiments, T waves in limb leads II and III were changed in form, though returned to their control state in 10 to 15 minutes. The T wave in lead II decreased in height, and the T wave in lead III became inverted, or more negative, if it had been negative in the control electrocardiogram. Following this experiment, each subject drank 600 ml of hot lemonade. No resultant alterations in the EKGs were observed.

One of the research subjects drank barium while lying supine in the same experimental position. Through fluoroscopy, it was determined that the fundus of the stomach, thus distended, came in contact with the "postero-inferior aspect of the apex of the left ventricle" (Wilson & Finch, 1923, p. 276). Therefore, these scientists believed that cooling of this portion of the ventricle, by cold water ingestion, was responsible for a retarded rate of repolarization of the left ventricle, reflected by T wave changes in leads II and III.

Dowling and Hellerstein, in 1951, wished to repeat the experiment of Wilson and Finch while recording not only the bipolar limb leads, but also unipolar limb and several precordial leads. Not all of the research subjects were in good cardiac health: six subjects were normal resident physicians, 11 had a history of hypertension and signs of left ventricular hypertrophy, nine had a history of old
anterior myocardial infarction (MI), and eight had a history of old posterior MI. Subjects were in the supine, recumbent position and drank 800 ml iced water, temperature undisclosed. Before, immediately after, and at several five minute intervals following water consumption, EKG leads I, II, III, aVR, aVL, V1, V2, V5, and V7 or V8 were recorded. Primary T wave changes were seen in five of the six normals, four of the 11 hypertensive patients with left ventricular hypertrophy, five of seven with old anterior MI, and four of seven with old posterior infarctions. Maximal T wave changes occurred within five minutes after iced water ingestion and were completely resolved within 25 minutes. The T wave changes were as follows: increased negativity or decreased positivity of the T wave in leads aVF, II, III, V1, and V2; increased positivity or decreased negativity in aVR, aVL, and the right-sided precordial leads.

Dowling and Hellerstein believed these T wave changes resulted from delayed repolarization of the heart's posterior wall. No evidence of acute coronary insufficiency was seen in any subject; heart rate was not slowed after drinking iced water. In control experiments, 800 ml of tea at body temperature was consumed without significant EKG changes (Dowling & Hellerstein, 1951).

Attempting to discover whether iced water ingestion was a means to detect early coronary artery disease, Kuaity, Wexler, & Simonson (1969) studied 21 normal persons and 26 individuals who had had a documented myocardial infarction with the past five years. Subjects drank 600 ml iced water, temperature 0° to 2°C, over three minutes,
Clients' physical position was not reported. Before, immediately after, and at 5, 10 and 20 minutes following ingestion, electrocardiograms (leads undisclosed) were recorded.

T waves in lead III became significantly more negative in both normals and the persons having a history of myocardial infarction. However, there was not statistically significant difference between the T wave changes in the two groups (Kuaity, et al., 1969).

Pratte, et al. (1973) used 20 volunteer males to study cardiac activity during and after ingestion of water, under several different conditions. Two different amounts of water, 200 and 600 ml; two different temperatures, 38° to 45°F (3° to 7°C) and 98° to 105°F (22° to 26°C); and two different body positions, Fowler's and horizontal, were used. Subjects ranged in age from 30 to 65 years and had no history of cardiac problems. After a baseline 13-lead EKG was made, each subject was randomly assigned to one of four experimental conditions possible in the Fowler's position: 200 ml of 38° to 45°F water, 200 ml of 98° to 105°F water, 600 ml of 38° to 45°F water, or 600 ml of 98° to 105°F water. EKG leads II, V5, and aVF were recorded during, and at 3 minutes after water ingestion. Then, these same three leads were recorded at 10 minutes after the second measurement and at 20 minutes after the third measurement. Three to five days later, each subject was retested using the same amount and temperature of water in the horizontal position.

Pratte, et al. found that ST segments were more prolonged with the greater volume of water, the lower temperature of water, and with
the supine position while drinking. Greatest ST changes were recorded during and at three minutes after ingestion. T wave direction changed most with the greater volume of water in the supine (horizontal) position. Heart rate decreased most with the greater volume ingested, regardless of water temperature or body position (Pratte, et al., 1973).

In 1974, Fitzmaurice and Simon used 28 patients within seven days of suffering an acute myocardial infarction as research subjects. In semi-Fowler’s position, 150 ml of 4°C water was consumed. Limb lead II of the electrocardiogram was recorded just before, during and for 30 seconds after ingestion. Lead II was also recorded at these intervals when the subject ingested 150 ml of 15°C water. No significant differences were observed in heart rate changes, PR interval, T wave voltage, or QT interval between the lead II electrocardiograms at the different temperatures (Fitzmaurice & Simon, 1974).

Also using subjects post myocardial infarction (these 3 and 4 days post myocardial infarction), Houser (1976) studied the effects of iced water ingestion on 10 people. Iced water, 240 ml of 0° to 10°C, was administered to each subject one day, and 240 ml of 36° to 37°C water was administered on the following day. Subjects' physical position was not reported. Before drinking the water, a 12-lead EKG, blood pressure, an heart rate were measured. Twelve-lead EKGs were again recorded at 5 and 30 minutes post ingestion. Blood pressure and heart rate were measured at five minute intervals after the water was consumed. No significant differences occurred between subjects' EKGs,
blood pressure, or heart rate following iced water or tap water ingestion. Additionally, no abnormalities in 12-lead EKG, blood pressure, or heart rate were seen after iced water was consumed (Houser, 1976).

Patients with suspected myocardial infarction were research subjects in another study by Cohen, et al. (1977). This experiment was designed to discover what effects the drinking of hot and cold fluids had upon heart rate, blood pressure, and cardiac rhythm in post MI patients. Within six hours of hospital admission, patients ingested 6 ounces (180 ml) of Sanka at a temperature greater than 70°C over less than five minutes. Heart rate and rhythm were monitored continuously; blood pressure was taken near the end of drinking the fluid, and five minutes before and after the fluid was drunk. These measurements were repeated when the same patient later drank 6 oz. iced water of average temperature of 7°C (also over less than 5 minutes). Twenty of the experimental subjects were later found to have suffered documented myocardial infarction. These individuals had no significant change in cardiac rhythm, frequency of premature ventricular contractions (PVCs), heart rate, or blood pressure as a result of drinking hot and cold liquids (Cohen, et al., 1977).

In 1980, Siegel and Sparks studied effects of iced water drinking on the blood pressure and heart rate of 26 normal subjects. Two different volumes (240 ml and 720 ml) and two different temperatures (0° to 3°C and 23° to 26°C) of water were used. In a sitting position, subjects drank the lesser volume of water over 5 minutes, and the greater over 10 minutes. Blood pressure and heart rate
were recorded immediately after water ingestion, and at 2, 5, 10, 15, and 30 minutes after the first reading. Significant effects were noted on the systolic blood pressure which increased 10 mmHg by the larger volume (720 ml) of both iced and tap water. Heart rate decreased significantly with 720 ml of both iced and tap water. However, the greatest decrease in heart rate (10 beats/minute) occurred after ingestion of 720 ml iced water (Siegel & Sparks, 1980).

The results of the studies described do not provide evidence for a consistent relationship between temperature and cardiovascular changes. Experiments with human subjects used different amounts and temperatures of water; subjects drank the water in different positions over varying time spans; subjects were in diverse states of cardiovascular health. Parameters used to measure the dependent variable, cardiovascular changes, varied from one investigation to another. The present study investigated the effects of a single volume of iced water on heart rate, EKG, and blood pressure.

**Operational Definitions**

The operational definitions are as follows:

**Glass of Iced Water**: 240 ml water at a temperature of 0° to 5°C. 240 ml is equivalent to the standard 8 ounces which most water glasses contain.

**Glass of Tap Water**: 240 ml water at a temperature of 22° to 26°C.

**Heart Rate**: The number of QRS complexes in a one-minute, lead II electrocardiogram. With a person in sinus rhythm, heart rate
can be extrapolated from the 3-second intervals of the 12 lead EKG.

**ST Segment:** The interval from the end of the QRS complex to the beginning of the T wave. Normally the ST segment is isoelectric and is on the same horizontal level as the flat line before the P wave. An ST segment 1mm or more below the isoelectric line indicates ischemia of the left ventricle (Davis, 1985).

**Direction of the T wave:** T waves, which represent ventricular repolarization, are normally positive in all of the 12 standard electrocardiogram leads except III, aVR, and V₁. Inverted T waves in leads II, III, aVL, aVF, and V₂ through V₆ reflect retarded repolarization, related to ischemia of the left ventricle (Davis, 1985).

**Blood Pressure:** The resistance of the brachial artery to pressure exerted by a sphygmomanometer heard via stethoscope and expressed from the number where sound is first heard to the number at which sound is last heard.

**Hypotheses**

The hypotheses to be tested were:

1. Heart rate measured in normal subjects following ingestion of a glass of iced water will differ significantly from heart rate measured in healthy subjects following ingestion of a glass of tap water.
2. ST segment depression in EKGs of normal subjects following ingestion of a glass of iced water will differ significantly from ST segment depression in EKGs of normal subjects following ingestion of a glass of tap water.

3. T wave direction in EKGs of subjects following ingestion of a glass of iced water will differ significantly from T wave direction of EKGs of normal subjects following ingestion of a glass of tap water.

4. Blood pressure measured in subjects following ingestion of a glass of iced water will differ significantly from blood pressure measured in normal subjects following ingestion of a glass of tap water.

Summary

This chapter has described the development of a theoretical framework to describe the possible relationship between oral ingestion of cold water and changes in heart rate, ST segments, T waves, and blood pressure. The framework was derived from a review of the literature describing the action of temperature upon the mechanisms responsible for the initiation of the normal heart beat. The results of studies which attempted to relate iced water ingestion to cardiovascular changes were also discussed.
CHAPTER 3

METHODOLOGY

This study was designed to investigate the effects of iced water ingestion on heart rate, electrocardiogram, and blood pressure in normal subjects. The data collection and analysis protocols used to accomplish this purpose are described in detail in this chapter.

Study Design

An experimental, cross-over design, using a convenience sample, was constructed to investigate the effects of a single volume of iced water on heart rate, EKG, and blood pressure. Because of nonprobability sampling, the adequacy of these subjects in representing the population under study is indeterminable. In a cross-over design, the subjects receive both treatments in a randomly assigned order.

Study Setting

The study was conducted in a simulated laboratory in an empty room in a southwestern hospital. Subjects were received into the room and were encouraged to ask any questions they might still have regarding the experiment. Heart rate, EKG, and blood pressure were recorded in the room.
Study Sample

A convenience sample of twenty volunteers was recruited from a southwestern city. Criteria for inclusion were:

1. Age 25-40
2. Able to read and speak English
3. No history of chronic or acute physical illness
4. Non-smoker
5. No habitual use of any medication
6. Normal baseline heart rate of 60-100 beats/minutes (Andreoli, 1983)
7. Normal baseline 12-lead EKG, i.e., sinus rhythm or sinus arrhythmia without ectopic heart beats or signs of myocardial ischemia, injury, or infarction (Andreoli, 1983)
8. Normal baseline blood pressure; systolic 100 to 140 mmHg, and diastolic 60 to 90 mmHg (Andreoli, 1983)

Rationale for Selection Criteria

Criteria were formulated for the volunteer sample which were intended to limit extraneous variables that could affect the outcome of the study. Factors known to alter an individual's cardiovascular function are such extraneous variables.

Coronary artery disease, which causes impairment of coronary circulation, is the most frequent cause of heart disease in adults (Andreoli, 1983; Guzzetta & Dossey, 1984). To reduce the likelihood that subjects' possible coronary artery disease would affect the
parameters of cardiovascular function being measured, the sample was kept homogeneous with respect to the primary risk factors of this disease.

The age criterion of 25 to 40 years for inclusion in the study was based on the fact that persons of this age are unlikely to suffer from coronary artery disease (Andreoli, 1983; Guzzetta & Dossey, 1984). Persons who had such primary risk factors as smoking, hypertension, and diabetes mellitus were excluded by the criteria of non-smoker and no history of chronic or current acute illness.

Possible effects of medication on cardiovascular function were eliminated as extraneous variables by exclusion of subjects taking medication. Subjects had to be able to read and speak English so that anxiety related to a lack of knowledge of the experimental procedure would not impinge upon the measured dependent variables.

Any individual with abnormal baseline heart rate, EKG, and/or blood pressure was excluded. Endogenous factors already affecting his cardiovascular function would have altered experimental measurements.

Sample selection criteria were explained to potential subjects at the time of recruitment. Compliance with selection criteria was determined by self-report of each potential subject. No physical examination was performed on these subjects.

Protection of Human Subjects

The purpose and design of this study was presented to the University of Arizona Human Subjects Committee. After approval of the
project was obtained, recruitment of subjects was begun (see Appendix A). Each subject was required to read a disclaimer which explained the purpose and methodology of the study (see Appendix B). In the disclaimer, subjects were informed of their right to withdraw from the project at any time, without incurring ill will. Subjects were encouraged to ask questions at the time of recruitment and at the initiation of data collection.

Each subject was given a code number and data were coded according to the number to assure subject anonymity and confidentiality. Only the investigator had access to the key which showed the numbers assigned to subjects.

**Data Collection Procedure**

Each subject was given a copy of the disclaimer. An appointment time was agreed upon for appearance at the simulated laboratory. Every person taking part in the study was provided with the investigator's phone number so that any questions could be answered. Questions were answered by the investigator in the laboratory as well.

All members of the sample were asked to fast, without food for four hours preceding data collection, and without fluids for two hours preceding the test. This fasting period, similar to that used in previous studies (Siegel & Sparks, 1980; Pratte, et al., 1973), was intended to decrease any influence food or fluid might have had on absorption of the experimental volume of water.
Age was recorded for all subjects upon arrival at the lab. Then, for fifteen minutes, each subject remained at chair rest. This interval was provided to allow subjects to return to the resting state after the activity involved in travel to the lab. When this rest period had elapsed, a baseline, 12 second rhythm strip, 12-lead EKG, and blood pressure were recorded. Those subjects with abnormal baseline measurements were excluded from the study, viz., those individuals with a heart rate less than 60 or greater than 100 beats per minute, those persons with cardiac dysrhythmias, those persons whose EKG indicated myocardial ischemia, injury, or past infarction, and those individuals whose blood pressure was less than 100/60 or greater than 140/90 mmHg.

Immediately after the baseline measurements, the subjects received a 240 ml glass of either tap or iced water. The ordering of the water, whether or tap or iced first, was determined by flipping a coin. Tap water was 240 ml of water between 22° and 26°C. Iced water, the independent variable, was maintained from 0° to 5°C.

Subjects were allowed one minute to drink the 240 ml of water. Two minutes after completely drinking the water, a 12-lead EKG and blood pressure were recorded. The measurements were repeated at five minutes, ten minutes, and thirty minutes after completely drinking the 240 ml of water.

Ten minutes after the last measurement (40 minutes after drinking the first glass of water), 240 ml of either the tap or iced water was given to the subject. The temperature of water given the second time was dependent upon which the subject had received during
the first trial. Had he received tap water the first time, he was given iced water the second, and vice versa. Subjects were allotted one minute in which to drink the water. Once again, blood pressure and 12-lead EKG were recorded at 2, 5, 10, and 30 minutes after water consumption.

Data were collected between the hours of 10 a.m. and 9 p.m. Total time commitment required from each subject was approximately 1.5 hours.

Electrocardiograms were obtained with a 12-lead EKG machine, and were recorded with the subject sitting upright in a chair. Blood pressures were obtained with a mercury column sphygmomanometer, also with the subject sitting upright in a chair.

Study Instruments

Electrocardiograph

The reliability of physical measurements obtained from instruments such as the electrocardiograph are affected by imperfections in the tools themselves, inaccuracy in their operation, and by random error. Calibration reduced instrumental error (Skoog, 1985). Therefore, the 12-lead EKG was calibrated before each electrocardiogram to increase reliability of this measurement. Machine calibration marks were consistent between recordings.

Depressed ST segments and inverted T waves of the EKG in leads I, II, aVL, aVF, and V₂ through V₆ have criterion-related validity as indicators of myocardial ischemia. Criterion-related validity is the
correspondence between a measurement and a certain criterion (Carmines & Zeller, 1979). Depressed ST segments and inverted T waves have long been established by authorities as reflections of myocardial ischemia (Davis, 1985; Guyton, 1986).

Sphygmomanometer

Reliability of blood pressure measurements taken with a sphygmomanometer is also affected by instrument error, personal error, and random error. Calibration of the sphygmomanometer was ascertained between subjects by noting the position of the column of mercury at zero. The same sphygmomanometer was used for all measurements, and was left in the same position for all measurements on each subject. Blood pressure measurements made with a sphygmomanometer have criterion-related validity in reflecting arterial pressure, as established by the ubiquitous use of the sphygmomanometer for blood pressure measurement.

Data Analysis Plan

A repeated measures analysis of variance was used to test for significant differences in heart rate, ST segment depression, T wave direction, and blood pressure between subjects at 2, 5, 10, and 30 minutes after ingestion of iced water, and at 2, 5, 10, and 30 minutes after ingestion of tap water. ST depression and T wave direction will be treated as dichotomous data.
Hypothesis One

Repeated measures ANOVA was used to assess if heart rate significantly differed in a group receiving iced water when compared to a group receiving tap water. Groups were compared at 2, 5, 10, and 30 minutes after completely drinking the water.

Hypothesis Two

Repeated measures ANOVA was used to assess whether ST segment depression (of 1 mm or more below the isoelectric line in the 12-lead EKG) significantly differed in a group receiving iced water when compared to a group receiving tap water. Groups were compared at 2, 5, 10, and 30 minutes after completely drinking the water.

Hypothesis Three

Repeated measures ANOVA was used to assess whether T wave direction differed significantly in leads I, II, aVL, aVF, and/or V2 through V6 of the 12-lead EKG in a group receiving iced water when compared to a group receiving tap water. Groups were compared at 2, 5, 10, and 30 minutes after completely drinking the water.

Hypothesis Four

Repeated measures ANOVA was used to assess whether systolic and diastolic blood pressure differed significantly in a group receiving iced water when compared to a group receiving tap water. Groups were compared at 2, 5, 10, and 30 minutes after completely drinking the water.
Limitations

1. The study was primarily limited by the small sample size (n=20).

2. Self-reports of physical health of the subjects may have further limited the study. It is possible that a subject may unknowingly have had an endogenous variable which affected his cardiovascular function.

3. Unconscious personal bias of the investigator in making measurements may have affected the results of this experiment.

Summary

This chapter has described the development of a quasi-experimental design to assess the relationship between iced water and heart rate, EKG, and blood pressure. An explanation of the study setting, data collection protocol, and data analysis plan was given. The study instruments, potential sources of bias, and limitations were described.
CHAPTER 4

RESULTS OF DATA ANALYSIS

The results of data analysis are presented in this chapter. Characteristics of the research sample are described. The analyses of variance in heart rate and blood pressure between experimental and control groups are reported. Electrocardiograms made before and after oral water ingestion are described.

Description of the Sample

The convenience sample consisted of eleven females and nine males. Each subject was tested under control and experimental conditions, after drinking tap water and iced water, respectively. Data were collected on eight separate days on a two-week interval.

Subjects' ages ranged from 25 to 40 years. The mean age was 33.6 years, with a standard deviation of 4.73 years.

Heart Rate

Nine 12-lead electrocardiograms were obtained on each subject. Calibration was consistent among all electrocardiograms. The mean baseline heart rate for all subjects was 74.5 beats/minutes, with a standard deviation of 11.26 beats/minute. In Table 1 are displayed the mean heart rates at baseline, and at 2, 5, 10, and 30 minutes after drinking iced and tap water. Sixteen of the twenty subjects had a sinus
Table 1. Description of Heart Rate Under Experimental and Control Conditions: Range, Means, and Standard Deviations (n = 40)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Experimental Condition* n = 20</th>
<th>Control Condition** n = 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate (Beats per minutes)</td>
<td>Range</td>
<td>(\bar{x})</td>
</tr>
<tr>
<td>Baseline</td>
<td>60-100</td>
<td>74.50</td>
</tr>
<tr>
<td>2 minutes after water ingestion</td>
<td>55-100</td>
<td>74.70</td>
</tr>
<tr>
<td>5 minutes after water ingestion</td>
<td>50-93</td>
<td>70.85</td>
</tr>
<tr>
<td>10 minutes after water ingestion</td>
<td>54-97</td>
<td>73.30</td>
</tr>
<tr>
<td>30 minutes after water ingestion</td>
<td>54-100</td>
<td>76.60</td>
</tr>
</tbody>
</table>

* 240 ml 0-5° C water  
** 240 ml 22-26° C water
arrhythmia, a normal variant, in which the heart rate varies slightly with ventilation.

Analysis of Heart Rate Differences Between Groups

For the purpose of analysis, the twenty individuals tested under experimental and control conditions were treated as two separate groups of twenty people, one of which ingested iced water, while the other drank tap water. To reduce a possible treatment order effect, the treatment a subject received first, either iced or tap water, was decided by random selection. In the sample of twenty, seven received iced water first, and 13 received tap water as the primary treatment. Subjects' baseline heart rate, measured before either of the treatments, was used as a covariant to adjust for differences in heart rates between subjects.

Hypothesis One

The first null hypothesis states that heart rate measured following ingestion of iced water will not differ significantly from heart rate measured following ingestion of tap water. All measured heart rates were adjusted, using subjects' resting, baseline heart rate as a covariant (see Table 2). The difference in mean heart rate between groups receiving the two temperatures of water, independent of time, was not significant. Subjects' heart rates across time for the combined groups did not vary significantly. Heart rates analyzed to assess
Table 2. Comparison of Groups on Heart Rate Adjusted by Baseline Rate Analysis of Variance (n = 40)

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group (Iced water vs. tap water)</td>
<td>1</td>
<td>4.56</td>
<td>.02</td>
</tr>
<tr>
<td>Error</td>
<td>37</td>
<td>247.88</td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>3</td>
<td>75.32</td>
<td>2.52</td>
</tr>
<tr>
<td>Iced Water/Time Interaction</td>
<td>3</td>
<td>57.36</td>
<td>1.92</td>
</tr>
</tbody>
</table>

whether the iced water plus time interaction had an effect did not vary significantly.

In reviewing the mean heart rates for the experimental and control groups at the four times following water ingestion (2, 5, 10, and 30), no significant differences were observed. Therefore, the first null hypothesis failed to be rejected.

Hypothesis Two

The second null hypothesis states that ST segment depression in EKGs of subjects following ingestion of a glass of water will not differ significantly from ST segment depression in EKGs on any of the twenty subjects included in the study. Data were insufficient to be analyzed by repeated measures ANOVA. Thus, the second null hypothesis was not tested statistically.

Hypothesis Three

The third null thesis states that T wave direction in EKGs of subjects following ingestion of a glass of iced water will not differ significantly from T wave direction in EKGs of subjects following ingestion of a glass of tap water. In order to participate in the study, subjects had to have a normal baseline EKG. T waves could only be inverted in leads III, aVR, and/or V_1. Only a single subject had a change of T wave direction. Following iced water ingestion, one subject's T wave, previously upright in lead III, became negatively deflected in this lead at the two minute and five minute measurements. By ten minutes after iced water ingestion, the T wave was again upright
in limb lead III. As only one change in T wave direction was observed, data were insufficient to be analyzed by repeated measures ANOVA. Therefore, the third null hypothesis was not tested statistically.

Hypothesis Four

The fourth null hypothesis states that blood pressure measured in subjects following ingestion of a glass of iced water will not differ significantly from blood pressures measured in subjects following ingestion of a glass of tap water.

Systolic and diastolic blood pressures were analyzed separately, using repeated measures ANOVA (see Table 3 for ranges, means, and standard deviations of systolic blood pressures). The difference in mean systolic blood pressure between groups receiving the two temperatures of water, independent of time was not significant. Systolic blood pressure in the combined groups across time did not vary significantly. Analysis of variance for repeated measures measures did reveal a significant difference in systolic blood pressure following the iced water by time interaction, $p=0.01$. Following iced water ingestion, mean systolic blood pressure increased by 3.3 mm Hg from two minutes after water consumption to 30 minutes afterwards (see Table 4).

On Table 5 are shown descriptive measures for diastolic blood pressure. Analysis of variance for repeated measures revealed no significant difference in diastolic blood pressure between experimental and control groups, across time, or for the iced water by time interaction (see Table 6). Because of the significant value of the iced water
Table 3. Description of Systolic Blood Pressure Under Experimental and Control Conditions: Range, Means, and Standard Deviations (n = 40)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Experimental Condition* n = 20</th>
<th></th>
<th>Control Condition** n = 20</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>( \bar{x} )</td>
<td>S.D.</td>
<td>Range</td>
</tr>
<tr>
<td>Baseline</td>
<td>100-130</td>
<td>11.40</td>
<td>9.74</td>
<td>100-130</td>
</tr>
<tr>
<td>2 minutes after water ingestion</td>
<td>94-130</td>
<td>109.50</td>
<td>9.38</td>
<td>96-134</td>
</tr>
<tr>
<td>5 minutes after water ingestion</td>
<td>94-132</td>
<td>111.20</td>
<td>9.23</td>
<td>94-128</td>
</tr>
<tr>
<td>10 minutes after water ingestion</td>
<td>100-124</td>
<td>109.70</td>
<td>7.12</td>
<td>100-132</td>
</tr>
<tr>
<td>30 minutes after water ingestion</td>
<td>96-130</td>
<td>112.80</td>
<td>10.14</td>
<td>90-130</td>
</tr>
</tbody>
</table>

* 240 ml 0-5° C  
** 240 ml 22-26° C
Table 4. Comparison of Groups on Systolic Blood Pressure Adjusted by Baseline Systolic Analysis of Variance (n = 40)

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group (Iced water vs. tap water)</td>
<td>1</td>
<td>4.23</td>
<td>.03</td>
</tr>
<tr>
<td>Error</td>
<td>37</td>
<td>140.43</td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>3</td>
<td>7.69</td>
<td>.36</td>
</tr>
<tr>
<td>Iced Water/Time Interaction</td>
<td>3</td>
<td>80.76</td>
<td>3.82*</td>
</tr>
</tbody>
</table>

* p = .012
Table 5. Description of Diastolic Blood Pressure Under Experimental and Control Conditions: Range, Means, and Standard Deviations (n = 40)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Experimental Condition* n = 20</th>
<th>Control Condition** n = 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diastolic Blood Pressure (mm Hg)</td>
<td>Range</td>
<td>$\bar{x}$</td>
</tr>
<tr>
<td>Baseline</td>
<td>60-90</td>
<td>79.20</td>
</tr>
<tr>
<td>2 minutes after water ingestion</td>
<td>64-90</td>
<td>79.40</td>
</tr>
<tr>
<td>5 minutes after water ingestion</td>
<td>64-96</td>
<td>81.20</td>
</tr>
<tr>
<td>10 minutes after water ingestion</td>
<td>60-94</td>
<td>80.20</td>
</tr>
<tr>
<td>30 minutes after water ingestion</td>
<td>64-100</td>
<td>80.50</td>
</tr>
</tbody>
</table>

* 240 ml 0-5°C water
** 240 ml 22-26°C water
Table 6. Comparison of Groups on Diastolic Blood Pressure Adjusted by Baseline Diastolic Analysis of Variance (n = 40)

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group (Iced water vs. tap water)</td>
<td>1</td>
<td>.03</td>
<td>.00</td>
</tr>
<tr>
<td>Error</td>
<td>37</td>
<td>127.24</td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>3</td>
<td>29.69</td>
<td>1.84</td>
</tr>
<tr>
<td>Iced Water/Time Interaction</td>
<td>3</td>
<td>15.69</td>
<td>.97</td>
</tr>
</tbody>
</table>
by time interaction upon systolic blood pressure, the fourth null hypothesis was rejected.

**Summary**

Oral iced water ingestion was associated with a rise in systolic blood pressure over time. No significant differences in heart rate, ST segment, T wave direction, or diastolic blood pressure were observed between experimental and control groups.
CHAPTER 5

DISCUSSION OF RESULTS

Results of the data analysis, conclusions, and implications for nursing practice are discussed in this chapter. Design changes for future research are suggested.

Heart Rate Following Iced Water Ingestion

No significant change in heart rate was noted in any subject during the experiment. This finding supports the work of other researchers who found no changes in heart rate after iced water consumption of 240 ml or less (Pratte, et al., 1973; Fitzmaurice & Simon, 1974; Houser, 1976; Cohen, et al., 1977; Siegel & Sparks, 1980).

ST Segment Depression Following Iced Water Ingestion

ST segment depression, an index of left ventricular ischemia (Davis, 1985), was neither observed in this experiment, nor reported in any of the previous studies reviewed. However, prevention of increased myocardial ischemia resultant to drinking iced water continues to be a rationale for its denial to cardiac patients (Houser, 1976). It was for this reason that ST segments were measured in the present experiment.

42
Change in T Wave Direction
After Iced Water Ingestion

Inversion of a T wave that had been upright during the baseline EKG was observed in the EKG of one subject. The T wave in lead III was inverted at two minutes and five minutes after iced water ingestion. By the 10 minute measurement, the T wave was again upright.

Transient T wave directional changes have been observed by other researchers following subjects' consumption of iced water (Wilson & Finch, 1923; Dowling & Hellerstein, 1951; Kuaity, et al., 1969; and Pratte, et al., 1973). However, these changes were resultant to large (> 600 ml) quantities of water drunk in the recumbent position.

T waves may normally be negative to the isoelectric line in lead III (Davis, 1985). Therefore, temporary inversion of the T wave in lead III following the drinking of iced water is not believed to have clinical significance.

Systolic Blood Pressure Change
Following Iced Water Ingestion

The combined effect of iced water and time resulted in an increase in mean systolic blood pressure of 3.3 mm Hg from two minutes after ingestion to 30 minutes after ingestion. The mean systolic blood pressure of the control group decreased 1.9 mm Hg from two minutes after tap water consumption to 30 minutes after consumption. Other researchers did not show significant blood pressure changes after 240 ml, or less, of iced water (Houser, 1976; Cohen, et al., 1977; Siegel & Sparks, 1980). Siegel and Sparks (1980) observed an increase in
systolic blood pressure subsequent to the consumption of 720 ml of water; this increase was unrelated to the temperature of the water.

In this investigation, the interaction between iced water and time for systolic blood pressure, without other concomitant cardiovascular changes, cannot be easily explained. For thirteen of the twenty subjects, participation in the data collection procedure was coming to an end. One would expect relaxation, and with it, a drop in blood pressure. Replication of the study with a larger sample may clarify the interaction between 240 ml of iced water and time for systolic blood pressure.

**Diastolic Blood Pressure**

*After Iced Water Ingestion*

Analysis of variance for repeated measures did not reveal a significant difference between the diastolic blood pressures of the iced water and the tap water groups. The absence of change was also noted in former studies (Houser, 1976; Cohen, et al., 1977; Siegel & Sparks, 1980).

**Implications for Nursing Practice and Suggestions for Further Study**

Ingestion of 240 ml 0-5°C water was related to a significant change in systolic blood pressure over the 30 following minutes. Whether or not the increase would produce ill effects in healthy subjects is doubtful. An increase of 3 mm Hg in systolic blood pressure in persons having sustained recent myocardial injury would not likely be deleterious, either. However, findings obtained from 20 healthy,
young individuals cannot be generalized to those persons with myocardial damage. A larger sample which included subjects having cardiac illness would extend the applicability of the data.

The effect of iced water ingestion on blood pressure remains unclear. Elucidation of the effect with larger and more heterogeneous samples is necessary to determine whether or not iced water should be denied to cardiac patients.

In summary, clarification and generalizability of research findings may result from replication of the study with two changes in design:

1. increase the sample size.
2. include persons with known cardiac disease in the sample.

Summary

In contrast to previous similar experiments, this investigation found a significant difference in the systolic blood pressure over time in subjects when they consumed iced water as opposed to when they drank tap water. Whether or not such an increase would harm cardiac patients, or that they would even have such an increase, must be investigated before iced water can be given to these individuals.

There was no significant interaction between iced water, time, or iced water over time, upon heart rate, ST segment depression, T wave direction, or diastolic blood pressure in the present study. A significant effect may be discovered through replication of this study using larger and more varied samples.
APPENDIX A

HUMAN SUBJECTS APPROVAL LETTER
TO: Kathryn Kerr, BS, RN
Graduate Student
College of Nursing

FROM: Ada Sue Hinshaw, PhD, RN
Linda R. Phillips, PhD, RN
Director of Research
Chairman, Research Committee

DATE: October 21, 1986

RE: Human Subjects Review: The Effects of Iced Water Ingestion on Heart Rate, Electrocardiogram, and Blood Pressure

Your project has been reviewed and approved as exempt from University review by the College of Nursing Ethical Review Subcommittee of the Research Committee and the Director of Research. A consent form with subject signature is not required for projects exempt from full University review. Please use only a disclaimer format for subjects to read before giving their oral consent to the research. The Human Subjects Project Approval Form is filed in the office of the Director of Research if you need access to it.

We wish you a valuable and stimulating experience with your research.

ASH/fp
APPENDIX B

DISCLAIMER
The Effects of Iced Water Ingestion on Heart Rate, Electrocardiogram, and Blood Pressure

By arriving at the laboratory for drinking of water and blood pressure and electrocardiogram measurement, you will be giving your consent to participate in the study. This experiment is designed to discover possible relationships between drinking iced water and normal heart function. Participation is completely voluntary. You may withdraw from the project at any time without incurring ill will and you are free to ask questions at any time. Your name will be known only to the primary investigator. All information will be kept confidential and reported using only an identification number. Data may be used for further publication. There are no known risks to participation in the study.
REFERENCES


Wilson, F.N., & Finch, R. (1923). The effect of drinking iced-water upon the form of the T deflection of the electrocardiogram. *Heart, 10*, 275-278.