Comparison of Three Field Treatments for Induced Mild ($33.0^{o}C$) Hypothermia

by

Gordon G. Giesbrecht

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ABSTRACT

COMPARISON OF THREE FIELD TREATMENTS FOR INDUCED MILD (33.0°C) HYPOTHERMIA.

Introduction. Three field applicable treatments for hypothermia were compared for their efficiency in rewarming the body. Methods. Six, fit healthy subjects were cooled in stirred cold water (< 8.0 °C) to a core temperature as low as 33°C. Each subject was then rewarmed in a random order by each of three techniques: shivering (SH), heating pad application (HP) where the STK HEATPAC was used and treadmill exercise (EX). In each case, the skin was insulated from warm ambient air. During cooling and rewarming, core temperature (Tc) was monitored continuously electronically with an esophageal thermistor probe at the level of the heart. Heart rate (HR), blood pressure (BP) and oxygen consumption were monitored intermittently. Treatment effectiveness was determined by calculating: rate of Tc increase (RI), amount of Tc afterdrop (ADA) and length of afterdrop period (ADL). Results. RI for EX ($4.9^{\circ}C \cdot hr^{-1}$) was significantly higher ($p=.05$) than SH ($3.5^{\circ}C \cdot hr^{-1}$) but not HP ($3.7^{\circ}C \cdot hr^{-1}$). Exercise ADA and ADL values (0.95°C and 24 min respectively) were significantly higher than both SH (0.33°C, 15 min) and HP (0.32°C, 14 min). Conclusion. In otherwise healthy young victims of mild immersion hypothermia, exercise is not only possible but may be, in certain situations, the preferred field treatment. Shivering pro-

duces a great amount of heat if skin is insulated and kept cooler longer. The heating pad seemed to inhibit shivering. This may explain why the RI for the heating pad was not significanly higher than for shivering thermogenesis.

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INTRODUCTION

Accidental hypothermia is generally defined as the unintentional lowering of body core temperature to below 35°C (Golden, 1972). This temperature has been determined by the Glossary Committee of the International Union of Physiological Sciences as being one standard deviation below the mean core temperatures of humans in a thermoneutral environment (Bligh & Johnson, 1973).

Hypothermia may occur in different groups of people for various reasons. Accidental hypothermia may afflict otherwise healthy victims such as athletes, recreationists or travellers (Pugh, 1966). Hypothermia also results from cold exposure precipitated by drug or alcohol abuse (Bristow, 1978). The elderly and those with various forms of metabolic disorders experience a high mortality rate with hypothermia (Lloyd, 1973). Finally, hypothermia is induced for some types of surgery in order to reduce metabolic requirements of the heart and brain (Keatinge, 1969).

History has recorded deaths from hypothermia of military men (Zingg, 1966), boaters, hikers and mountaineers (Pugh, 1966), accident victims, shipwrecked passengers (Keatinge, 1969), and the elderly (Bristow, 1986). It was estimated that between the years 1965 and 1975, Great Britain experi-

enced 700 cold water immersion deaths per year. This number was estimated to be as high as 6,000 in America (Golden & Rivers, 1975). Statistics from 1974-79 revealed that an average of 120 people died in Canada each year from hypothermia (Martyn, 1981).

There are various methods of treatment for hypothermia. Treatments are categorized as passive, active peripheral and active core (Martyn, 1981). Passive treatment generally consists of allowing the subject to rewarm via shivering thermogenesis in a dry warm environment (Conn, 1980). There are many active peripheral rewarming methods including hot water immersion, water heated blankets, hot water bottles, heating pads, and contact with human body warmth or sitting beside an open fire (Collis, Steinman & Chaney, 1977).

Active core rewarming can be subclassified as invasive or noninvasive. Inhalation of heated water saturated air is a noninvasive measure (Conn, 1980). Consumption of hot food or fluids may also fit this category. Invasive core rewarming methods occur in hospital treatment situations. These treatments include peritoneal lavage, extracorporial circulation, administration of warmed intravenous fluids (Bristow, 1986), gastric lavage (Bristow et al., 1977), thoracotomy, and pleural irrigation with warm fluids (Coughlin, 1973). These measures have been shown to facilitate substantial heat transfer, but are associated with increased morbidity due to their invasive nature (Bristow, 1986).

Although heat gain may also result from exercise metabolism, it is difficult to categorize the mechanism. Exercise is active but not exclusive to the periphery or the body core as the entire body warms up.

Rates of heat gain have been established experimentally or clinically for most treatments. Nontechnical methods such as exposure to heating pads or the containment of heat from exercise metabolism have not however, been examined thoroughly.

Knowledge of the effectiveness of these methods of treatment, although lacking ,is desirable as they may be the only methods available in locations far from rescue or emergency facilities.

STATEMENT OF THE PURPOSE

An experiment was designed to evaluate the safety and effectiveness of three rewarming procedures for use as treatment for hypothermia in a field setting.

The rewarming techniques were shivering thermogenesis (s), exercise induced metabolic heat (e), and the external application of heat (h). Evaluation of each technique will be directed to quantification of the afterdrop, subsequent rate of core temperature increase, and duration of the afterdrop period.

The following null hypotheses were tested:

(1) Ho: $\mu As = \mu Ae = \mu Ah$,

Ha: at least two μA's are different,

where A = amount of afterdrop.

(2) Ho: $\mu Ls = \mu Le = \mu Lh$,

Ha: at least two μL's are different,

where L = duration of the afterdrop period.

(3) Ho: $\mu Rs = \mu Re = \mu Rh$,

Ha: at least two μR's are different,

where R = rate of core temperature increase.

Subjects naturally fell into distinct categories according to sum of skinfolds: a 'low fat' subgroup with sum of skinfolds < 40 mm ; and a 'high fat' subgroup with sum of skinfolds > 50 mm. Some of the analyses carried out on the group as a whole were also carried out on these subgroups in order to see if any of the results were affected by amount of body fat.

DELIMITATIONS

Due to safety requirements, subjects were required to be physically fit with no drug, alcohol or disease influences existing. This experiment therefore pertained exclusively to immersion hypothermia experienced by otherwise healthy, fit persons.

LIMITATIONS

Due to the complexity and discomfort of the procedure the number of subjects may be less than optimal for statistical analysis.

It is recognized that external validity of results to all levels of hypothermia may be limited by 2 factors: appropriate treatment for mild hypothermia may be different than that required for moderate to severe cases; and physiological responses at Tc=33^{o}C may differ substantially from those at much lower temperatures.

DEFINITION OF TERMS

Afterdrop Amount

The drop in body core temperature (Tc), from the time of removal of the subject from the water, until it starts to increase.

Afterdrop Duration

The period of time, after removal from cold insult, for Tc to drop and regain its original removal value.

Cold Diuresis

A specific avenue of fluid loss where cold induced peripheral resistance increases central venous pressure and results in subsequent increased rate of fluid loss by the kidneys.

Dehydration (fluid loss)

The reduction of body fluid supplies via perspiration, respiration or excretion through the kidneys.

Fluid Shift

The movement of the salt and water portion of the plasma, to extracellular spaces, without movement of red blood cells. The resultant decrease in blood volume increases the hematocrit and blood viscosity.

Hydrostatic Squeeze

Increased atmoshperic pressure on the body caused by water during immersion resulting in substantially raised peripheral vasoconstriction.

Rate of Core Temperature Increase

The rate (^{0} C$\cdot$hr^{-1}) of increase in core temperature from the time of reversal of afterdrop until the conclusion of treatment.

REVIEW OF RELATED LITERATURE

INTRODUCTION

This experiment was directed towards understanding the effects of exposure to cold and the quantification of appropriate treatments to reverse these effects with minimum damage or risk to the individual. This review begins with a historical overview of observations and research into the area of cold exposure. Subsequent sections will elucidate classifications, causes, signs and symptoms of this condition. An in depth look at physiological and metabolic changes that occur during hypothermia will follow. Finally, various methods of treatment will be described, quantified and compared.

HISTORICAL OVERVIEW

Although literary recordings have only been common in the past few centuries, it is doubtless that death from exposure to cold has plagued mankind throughout history. This section reviews past research on effects of hypothermia and treatment procedures.

Records show that philosophies of rewarming have oscillated between rapid and slow heat transfer. Golden (1983)

reported several historical observations. Perhaps the first rewarming death in written history involved a soldier who died after being rapidly rewarmed in a tub of hot water in A.D. 320. Golden may have exposed the first recorded rewarming success witnessed by Naucler in 1757 when he saw a man already in a coffin come to life after being rewarmed by heated objects applied to his body. The father of maritime medicine, Sir James Lind of the Royal Navy described a regime for treatment of cold exposure in 1762. He suggested the victim be placed in a warmed bed with warmed clothes rubbed on his belly, breast and pit of the stomach. Warmed bricks, irons or water bottles were applied to the feet. Warmed air was blown by bellows into the anus and lungs. Immersion in luke-warm water was also suggested.

James Currie (1798) conducted the first experiments on the effects of cold water immersion and subsequent rapid or slow rewarming. He was able to show via oral thermometer readings, a representation of what happened following immersion in cold water. At first there was an increase in temperature which has since been explained by increased metabolism. Following this brief period the temperature began to drop and continued to drop (afterdrop) even after the volunteer was taken from the water. Currie proposed rapid rewarming in hot water as the appropriate treatment for cold water immersion.

The next documentated recovery from deep hypothermia came from Dr. John Laing (1815) who in 1806 resuscitated a sailor aboard the Endeavour who may have also suffered a cardiac arrest.

The first major research into effects of cooling came after Napoleon's army was almost destroyed by cold in 1812 (Richardson, 1974). Napoleon's army, originally 225,000 men , retreated from Moscow with 80,000 men of whom less than 45,000 returned safely. The majority of casualties were from cold exposure. The French chief surgeon Barron Larrey was probably better able than any other in history to observe the effects of cold exposure or hypothermia. He described such typical symptoms as lethargy, confusion, clumsiness, stiffness, weakness, blindness, staggering as though drunk, and quick coagulation of body fluid. It is noteworthy that these are the exact manifestations reported in the current literature.

Gaynard in 1828, focused experimentation on frogs. He froze them, finding that if they were frozen slowly and rewarmed gradually, they returned to normal temperature. An unfortunate misapplication of this knowledge occurred when an English naturalist named Hunter tried this procedure out on humans with fatal results (Golden, 1983).

Lapchinski, a Russian author was credited with the establishment of rapid rewarming as an accepted treatment following cold exposure (Alexander, 1945).

Perhaps one of the worst human disasters occurred on April 14, 1912 with the sinking of the Titanic. Within one hour and fifty minutes, all 1,489 of the people immersed in the 0^{0} C water were dead (Keatinge, 1969). It is unfortunate that medical science missed the opportunity to discern between drowning, the official cause given for all deaths, and the actual killer, cold water immersion hypothermia. Had authorities realized then the need for protection from the energy usurping cold water, thousands of lives may have been saved in later years.

A World War I disaster occured in 1914 when the Gneisenau sank near the Falkland Islands. Two hundred survivors were successfully rescued from the cold water but the majority died shortly thereafter from 'postrescue collapse' which is commonly associated with immersion hypothermia (Zingg, 1966). The physiological process of this phenomenon will be explained in a later section.

In 1941, a researcher named Talbot reported a fatality from rapid surface rewarming (Golden, 1983). This resulted in the choice of slow rewarming as the primary treatment for military men in World War II.

Another major impetus for research into treatment for cold exposure again resulted from heavy casualties related to warfare. In World War II 30,000 men of the Royal Navy died from drowning or exposure (Keatinge, 1969).

Alexander (1945) reported experiments carried out by Dr. Rascher and his associates in Dachau from 1941-43. It was a sad commentary of practices carried out in one of the lowest points of human history and scientific endeavor. At least 103 different prisoners were subjected to cold water immersion, often to the point of expiration. After the war, Alexander could only locate one survivor of the experiments, a Catholic priest from Poland. Although this practice was sadistic and totally unethical, much valuable information was gained and the approximately 100 men, to whom this work is dedicated, would have died in vain if results were not used to the best advantage for medical treatment of mankind. One major finding (others will be discussed later) was that rapid rewarming in hot water resulted in the best prognosis for recovery from cold water immersion.

Rapid rewarming was again touted as the treatment of choice by Burton and Edholm (1955). These results contradict the recommendations by Talbot. It must be pointed out that the Talbot fatality was an isolated incident and other mitigating factors may have been present which were not reported. The experiments conducted by Rascher's group as well as Burton and Edholm involved many subjects at various initial body core temperatures. These numerous experiments demonstrated a definite advantage for rapid rewarming.

A great deal of research has taken place in the last 30 years. New, more intricate treatments have been developed

but no single treatment has been established as a panacea. Treatment often depends on availability of equipment and other predisposing factors such as reasons for heat loss, rate of heat loss and core temperature at the commencement of treatment.

CLASSIFICATIONS OF HYPOTHERMIA

In order to facilitate the following discussion, a description of the various classifications of the hypothermic condition is required.

Victims may suffer from primary or secondary hypothermia. Primary hypothermia occurs as a direct result of overwhelming cold exposure in individuals with otherwise normal thermoregulatory function. If core temperature decreases as a result of impaired thermoregulation from metabolic disease, old age or alcohol and drug abuse, it is said to be secondary (Golden, 1983).

Terminology also describes the rate of heat loss. Acute hypothermia results from cold exposure of less than six hours. This usually occurs in cold water immersion. Subacute exposure lasts longer than six hours and is common in land based situations. It is also referred to as exhaustion hypothermia. Chronic hypothermia evolves slowly for periods of longer than 24 hours and is common in those with underlying metabolic disorders (Golden, 1983; Kuehn, 1983; Martyn, 1981).

The final classifications refer to the state of body core temperature. Hypothermic conditions are said to be mild, moderate and severe when core temperatures are 30-35° C, 25-29° C and below 25° C respectively (Bristow, 1986).

ETIOLOGY

The human body maintains homeostatic control of parameters such as hormone levels, energy stores, fluid balance and temperature. Thermoregulation is controlled by the anterior hypothalamus although lower centers such as the spinal cord exert a great deal of control as well (Keatinge, 1969). Under general conditions the body's core temperature is maintained at 37.0° C. Decreases in blood temperature are precipitated by one or a combination of three threats: inadequate heat production, increased heat loss, or impaired temperature regulation.

Inadequate Heat Production

Heat production is thwarted by various endocrine disorders, hypothyroidism, hypopituitarism and malnutrition (Bristow, 1986). In healthy individuals, fatigue causes decreased metabolic heat production. Exercise in the cold rapidly decreases available fuel stores for thermoregulation and energy demands (Golden, 1972). Shivering in a nonexercising individual produces metabolic heat at three (Keatinge, 1969) to six (Myers, Britten & Cowley, 1979) times the

resting rate. Glycogen stores are depleted after 6-8 hours of shivering (Kugelberg et al., 1967). These stores may diminish faster if insulin levels drop resulting in a decreased muscle uptake of blood glucose (Hamlet, 1985). Shivering also stops when muscle tissue reaches 20-25° C at which point muscles become rigid (Martyn, 1981) and contraction is no longer possible (Lloyd et al., 1972).

Increased Heat Loss

Several predisposing factors exist for increased heat loss. In cold air environments the cold, wet and wind triad increases heat loss dramatically as wetness decreases insulative values of clothing and shelter while wind increases convective as well as evaporative heat loss (Golden, 1972; Pugh, 1966). Conductive heat loss is the major component in cold water immersion as thermal conductivity of water is 25 times that of air at the same temperature (Golden, 1972). Experimental and clinical evidence has shown that heat loss may therefore be many times greater in water (Molnar, 1946).

Greater heat loss occurs in infancy, old age and in conjunction with various skin diseases and large areas of body burns (Bristow, 1986). Heat loss occurs faster in bodies with greater surface area/body mass ratios (Bristow, 1986). Lower levels of subcutaneous fat result in greater heat loss (Keatinge, 1969). Somatotype also has an effect, as an ectomorph weighing the same as a mesomorph will experience faster heat loss (Hayward, 1983).

Tissue conduction (circulation), is a source of heat loss via warm blood flow to the periphery. This effect is counteracted by peripheral vasoconstriction in cold exposure. Shivering may however decrease tissue insulation and increase heat loss by increasing muscle blood flow to meet metabolic demands. In cold water immersion tissue conductance is minimal due to marked vasoconstriction. Heat loss occurs mainly via physical conduction when heat is conducted to the surface from deep organs due to the substantial temperature gradient which exists in the short distance between these organs and the exterior (Keatinge, 1969).

One other major avenue of loss in cold air environments is through respiration. A small portion of this loss is conductive due to the heating of inspired air. The majority of heat is lost through evaporation as this air is saturated with water vapor in the lungs (Conn, 1980). Respiration may account for 30% of the total metabolic heat production in the hypothermic state compared to 10-12% in the normothermic condition.

Impairment of Temperature Regulation

The thermoregulation process mediated in the hypothalamus may be impaired by stroke, head injury (Bristow, 1986), or diabetes (Davidson & Grant, 1981). Old age increases risk due to substantially decreased subcutaneous fat stores (Keatinge, 1969). Some forms of drug ingestion may debili-

tate the thermoregulation process (Bristow, 1978) as will the use of anesthetics. The most common anesthetic effect is invoked by alcohol abuse which is the mitigating factor in the majority of all hypothermia cases.

Although hypothermia may result from factors in any one of these categories, a combination of factors from more than one category commonly occurs.

SIGNS AND SYMPTOMS

The cold induced symptoms observed by Napoleon's chief surgeon Dr. Barron Larrey are all now recognized as signs of hypothermia. The initial sign of onset of hypothermia is coldness of the skin (Golden, 1972). Shivering gradually increases in intensity to a maximum rate which occurs at a core temperature of 35° C (Golden, 1973). At this temperature a victim will show introversion, apathy (Pugh, 1966), confusion, disorientation, and hyperventilation (Bristow, 1986).

At or below a core temperature of 32-33° C shivering ceases, is replaced by muscular rigidity (Bristow, 1978) and retrograde amnesia may also occur (Golden, 1973). Pupils start to dilate at 33° C core temperature. When Tc drops to 29-31° C early hyperventilation is replaced by marked slow breathing, muscle rigidity gives way to muscle flacidity and the victim will experience stupor followed by unconsciousness (Bristow, 1986).

As core temperatures drop to 28°C, ventricular fibrillation may occur as a result of any mechanical stimulation such as rough handling. The possibility of ventricular fibrillation occuring spontaneously, rises considerably when core temperatures fall below 26° C (Paton, 1983). Death from hypothermia is generally a result of cardiac arrest.

The progression of hypothermia is insidious as victims are not aware of what is happening to them. Early impairment of cerebral function is what makes this affliction so dangerous. The condition deteriorates to an incapacitating level without the victim knowing that a problem exists.

PHYSIOLOGICAL RESPONSE

This analysis evaluates the effects of cold injury on cells and systems. A majority of these effects relate to a slowed metabolism, dehydration, fluid shift, and vasospasm (constriction) mediated by the hypothalamus (Martyn, 1981).

The extent of many physiological adjustments depends on the speed of onset, whether it be acute (<6 hours) or subacute (>6 hours). In acute hypothermia disturbances are exclusively due to a drop in core temperature whereas subacute situations present secondary problems, such as fluid shifts and dehydration which complicate treatment (Golden, 1972).

The majority of research has probed cardiovascular adaptations to hypothermia. A familiar reaction to cold exposure is a decreased peripheral blood flow, which is mediated by nervous stimulation, reflex reactions, A-V shunts and the 'hydrostatic squeeze', which will be described later.

Vasoconstriction in skin level blood vessels is mediated by sympathetic nerves as a release of norepinephrine causes excitation of smooth muscle cells lining the vessels. There is also a direct constriction reaction of the vessels to the cold, which is at least as important as nervous control (Keatinge, 1969). Constriction is not permanent however, as a cold induced vasodilation (CIVD) phenomenon has been described where vessels dilate following prolonged exposure to cold. This reaction is common in cold water immersion where dilation may be induced by cold paralysis of vascular smooth muscle. CIVD may also be a mediated response by the body in an effort to save tissue from freezing by cyclically injecting warm blood into the periphery. The effect on heat loss is not clear. Hayward (1983) stated that constriction of deep vessels in warmer tissues may compensate for this loss. Keatinge (1969), however, felt that the increased peripheral blood flow did increase heat loss.

The ultimate extension of CIVD occurs in the phenomenon of paridoxical undressing. Just before a victim becomes comatose the periphery may totally vasodilate as the body shunts heat to the periphery. The victims feel warm, and in

a stuporous state take their clothes off and die (Hamlet, 1985).

Arteriole-venule (A-V) shunts also decrease peripheral blood flow. As blood flows through the arterioles towards the capillary tree, precapillary sphincters are stimulated to shut off the capillaries. Blood is then redirected to the venules via deeper metarterioles reducing heat loss to the periphery (Kinnear & Rhine, 1982). Even though substantial heat loss from peripheral circulation is averted, blood is still cooled as it reaches distal cold tissue. The countercurrent heat exchange (Keatinge, 1969; Hayward, 1983) minimizes the overall cooling effect on the blood. As warm arterial blood flows outward, it cools by transferring its heat to returning blood in deep parallel veins instead of surrounding tissue. Returning blood is therefore warmed before reaching the core.

Several hemodynamic changes occur during hypothermia. Blood plasma volume diminishes from fluid shifts or actual loss (dehydration). Red blood cell (RBC) mass remains constant and the hematocrit rises. The method of plasma decrease is primarily dependent on the type of exposure encountered by the victim.

Fluid shifts occur in subacute hypothermia (>6 hours) and greatly complicate the condition. Intense vasoconstriction increases peripheral resistance and intravascular crystal-

loid moves to extracellular spaces (Bristow, 1986). It was reported by Keatinge (1969) that the sodium pump stops in endothelial cells at temperatures below 30° C. Membrane permeability is affected and sodium ions and water pass through to the extravascular space. The decrease in plasma volume has also been attributed to the sequestering of plasma in the periphery without trapping RBC (Keatinge, 1969). This explanation has been refuted by Paton (1983) who felt that the capillary beds were dry due to intense vasoconstriction. At core temperatures of 30° C blood volume has been measured at 68% of predicted values (Harari et al., 1975) but the number of RBC remain constant (Meriwether & Goodman, 1972). Viscosity increases and sludging occurs (Bristow, 1986) as there is a tendency for RBC aggregation at very low temperatures (Schmid-Schonbein et al., 1973). There is some experimental evidence however that the increased hematocrit is not solely due to decreased plasma volume. Kanter (1968) showed that at least 50% of the increase in hematocrit was attributable to increased splenic production of RBC. This has not been substantiated by other studies.

Fluid loss has a major effect on cardiovascular function and metabolism. These effects are greater in dry air subacute situations where more time is available for loss to occur. As mentioned earlier, the nonprotein portion of plasma is recompartmentalized from vascular to extracellular space

and may even shift intracellularly causing edema in the brain and lungs. Much of this fluid is rapidly mobilized during rewarming.

Both decreased fluid intake and increased fluid loss result in a negative fluid balance or dehydration. The thirst mechanism is depressed in the cold resulting in decreased intake. Respiratory evaporative water loss on a cold, dry day may account for 0.5 liters per day. Perspiration losses may reach 1-2 liters per day (Hamlet, 1983).

As mentioned earlier, cold increases peripheral resistance and central venous pressure (CVP) rises. This stimulates the baroreceptors in the carotid arteries to trigger lowered antidiuretic hormone (ADH) production (Golden, 1972; Hamlet, 1985), causing a reduction in the reabsorbtion of glomerular filtrate in the collecting ducts of the kidneys. 'Cold diuresis' results and water is excreted. This 'cold diuresis' is another process that reduces circulatory volume. Plasma reduction is an important consideration for treatment of subacute hypothermia, where vasodilation induced by peripheral rewarming without adequate fluid replacement, would result in a precipitous drop in blood pressure.

Vasoconstriction is even more pronounced in acute cold water immersion as water increases the atmospheric pressure on the body. The 'hydrostatic squeeze' increases peripheral

constriction, and therefore increasing cold diuresis, resulting in a greater decrease in plasma volume (Golden, 1983). Urine flow rate doubles in thermoneutral water and may rise three and a half fold in cold water (Hayward, 1983). Complications arise when the 'hydrostatic squeeze' is removed during rescue. Cold paralysis inhibits the vascular smooth muscles ability to constrict and maintain blood pressure. Vessels dilate and the ensuing severe drop in blood pressure may cause immediate death.

The cardiac response to initial cold exposure is sinus tachycardia with increased cardiac output mediated by sympathetic nerve stimulation (Bristow, 1986). As mentioned earlier, vasoconstriction and increased resistance raise blood pressure initially. As the body cools however, pacemaker cells and the Purkinje conduction system cool (Martyn, 1981), resulting in the slowing of the pacemaker and gradual bradycardia (Hunter, 1968).

Cardiac output is a function of core temperature. Total output declines to 70% (Keatinge, 1969) and 30% (Bristow, 1986) of initial output as core temperatures reach 31^{o}C and 25^{o}C, respectively.

The decreased cardiac index results from decreased heart rate (Bristow, 1986) as well as decreased stroke volume (Harari et al., 1978). Harari and his associates postulated

that hypovolemia, caused by extracellular fluid shift, may decrease ventricular filling and consequently decrease stroke volume. They felt the major hemodynamic change in cold exposure was a decrease in ventricular power. In any case, blood pressure decreases along with cardiac output.

Disturbances and damage affect the heart itself as it cools. ECG readings reveal prolonged PR, QRS, as well as QT intervals (Bristow, 1986). An Osborne J wave follows the QRS complex and T wave inversion occurs. Core rewarming quickly reverses these abnormalities (Hamlet, 1985).

At core temperatures below 30° C atrial fibrillation occurs and may decrease cardiac output by 20% simply by lessening the stroke volume. Cold decreases metabolic energy availability to the myocardium as glucose and oxygen are delivered in a lower quantity. Decreased temperature also results in a shorter refractory period of cardiac muscle which favors ventricular fibrillation. Ventricular fibrillation may result from mechanical stimulation at temperatures below 28° C and may occur spontaneously when temperature reaches 25° C.

As blood temperature decreases, the heart becomes hypodynamic (Alexander, 1945) and direct cold injury may result. Overload from increased resistance, viscosity and circulation time will eventually lead to cardiac arrest (Hunter, 1968).

The initial respiratory response to cold exposure is hyperventilation as minute ventilation ($\dot{V}E$) may increase up to fivefold in the first 1-2 minutes in direct response to metabolic requirements for shivering and the noxious stimulus. Although shivering results in an initial increased CO_2 production, hyperventilation caused by the noxious stimulus results in a decreased $PaCO_2$. Respiration rate and tidal volume decrease as cooler temperatures depress the respiratory centers in the medulla (Martyn, 1981). Respiratory requirements are also lower as decreased tissue temperature reduces the metabolic requirements at the rate of 50% for every drop of 10^{o} C (Karplus, 1966). Respiration becomes more difficult due to rigor of the respiratory musculature (Hunter, 1968).

Despite an increased CO_2 production, hyperventilation results in an initial respiratory alkalosis (Bristow, 1986). At low body temperatures respiratory alkalosis is replaced by metabolic (lactic) acidosis.

The avidity of hemoglobin for O_2 increases as temperature drops. The oxyhemoglobin dissociation curve shifts to the left inhibiting the release of O_2 at the cellular level for a given PaO_2 (Bristow, 1978). In cooling, metabolic demands of peripheral tissues diminish but O_2 delivery decreases at a faster rate than O_2 demand resulting in ischemic acidosis. During warming, peripheral tissue cells increase metabolic demands but cold blood provides insufficient O_2 delivery further contributing to metabolic acidosis (Paton, 1983).

As cerebral temperatures decrease, metabolic demands diminish according to the Van't Hoff-Arrhenius law which states that O_2 requirements are cut in half for every 10^0 C temperature drop (Karplus, 1966). This is a protective mechanism as cerebral blood flow will diminish with body temperature (Hernandez, 1983). Cerebration is delayed as temperature drops. Good mental capacity is maintained until temperatures reach 33^0 C at which time retrograde amnesia may occur (Hunter, 1968). At temperatures below 31^0 C, impending paralysis of the central nervous system (CNS) impairs speech, consciousness and reflexes. At lower levels, depression of the CNS decreases pulse and respiratory rates.

Impulse conduction time is increased as nerve tissue cools (Martyn, 1981), accounting for decreased strength, coordination and response time of cooling muscles and nerves (Hayward, 1983).

Immediately after cold insult the basal metabolic rate (BMR) increases. When core temperatures are above 34^0 C, shivering (involuntary contractions of skeletal muscle) increases O_2 consumption 5 times the control level (Bristow, 1978) therefore contributing to increased metabolic heat production.

The initial effects on BMR are soon reversed and shivering stops. Thereafter, $\dot{V}O_2$ drops 7% per 1^0 C reduction (Pa-

ton, 1983). According to the Van't Hoff-Arrhenius law, BMR slows to one half normal at a core temperature of 28° C.

The decreased O_2 requirements serve a protective function by increasing biological survival time (the ability of cerebral tissue to withstand O_2 deprivation). Irreversible brain damage commences after 5 minutes of O_2 deprivation at tissue temperatures of 37° C but is delayed to 10-15 and 20-30 minutes at temperatures of 27° C, and 17° C, respectively (Bristow, 1986).

The liver stops converting lactic acid to pyruvate when its' temperature is below 32° C, resulting in a metabolic acidosis. At less than 30° C the intracellular fluid is acidotic and potassuim is transported from the cells to the blood in exchange for hydrogen ions. The state of intracellular hypokalemia induces a hypotensive strain on the heart. Insulin may be administered at the beginning of rewarming to drive the potassium back into the cells. If too much is given however, potassium levels may drop to dangerous levels when the rewarmed pancreas starts to produce insulin (Hamlet, 1985).

In the kidneys, impaired enzymatic activity of the distal tubules decreases reabsorption of glucose and acids, resulting in hyperglycemia and acidosis (Hunter, 1968; Meriwether & Goodman, 1972). Hyperglycemia may also result from lower insulin production in the pancreas (Martyn, 1981).

As temperature reaches very low levels, decreased cardiac output precipitates tubular ischemia and subsequently highly concentrated urine and obliguria (Martyn, 1981).

TREATMENT

Research over the past 30 years has provided many unequivocal answers with regards to treatment of hypothermia yet left many questions unanswered.

One fact clearly stands out. Rewarming and resuscitation must be carried out on every hypothermic victim as the axiom "don't assume a patient is dead until he is warm and dead" holds true (Lilja, 1983, p. 144). Patients have been successfully resuscitated from core temperatures of 16° C (Bristow, 1986) and after 4 hours of asystole (Hamlet, 1985). In fact, in Sweden it is illegal to pronounce a victum dead until a rewarming attempt has been made (Barnes, 1980). It is important to understand the difference between clinical death (unconsciousness, pulselessness, apnea, dilated pupils) and biological death (irreversible inactivation of certain enzymes and cerebral function) (Karplus, 1966; Bristow, 1978). Although the victim shows signs of clinical death at extremely low Tc, these manifestations are often reversible as long as the body is not biologically dead.

Rescue and rewarming of a victim presents several major concerns such as core temperature afterdrop, rewarming shock and post rescue collapse.

Core Temperature Afterdrop

Victims on removal from cold insult have shown continual temperature drops of 0.5° C (Conn, 1980) to 4.0° C (Alexander, 1945). Although this phenomenon is well documented, a full understanding of its mechanisms and application to treatment are not as definite.

For years the accepted explanation has been that rewarming causes a surge of cold blood, previously sequestered in the capillary beds during vasoconstriction, to the core resulting in further cooling of the myocardium (Burton & Edholm, 1955; Collis et al., 1977). Hunter (1968) proposed that in actuality no blood was sequestered in the periphery as capillaries were severely constricted and empty. He felt that cooling resulted from blood flowing through newly dilated vessels into cold peripheral tissue. Paton (1983) presented a gradient model to show another proposed mechanism for cooled bloodflow. He stated that core-surface gradients change throughout the rewarming process as follows:

$Ts < Tsc < Tc$ (cold body) (1)

$Ts > Tsc < Tc$ (initial surface rewarming) (2)

$Ts > Tsc > Tc$ (protracted surface rewarming) (3)

where Ts = skin temperature, Tsc = subcutaneous tissue temperature and Tc = core temperature. This model shows that during initial rewarming, peripheral blood is cooled as it passes through subcutaneous tissues. He felt that longer exposure and corresponding smaller skin-core temperature gradients would likely result in less afterdrop effect.

Golden (1983) stated that the drop in core temperature was not physiologically related at all, but actually a physical response experienced by any object. Kaufman (1983) conducted experiments on inanimate objects by cooling and rewarming them, finding that a sphere of water experienced the same afterdrop. He attributed this solely to physical changes in temperature gradients. These results would indicate that since afterdrop has nothing to do with circulation, the rate of cooling or choice of treatment would have no effect on afterdrop.

Many authors disagree with Kugelberg and his associates (1967) who stated that the amplitude of temperature afterdrop was greater following rapid cooling in cold water immersion than slow cooling in dry air. This was supported by Paton's (1983) temperature gradient model as shorter exposure to cold water immersion would indeed create larger temperature gradients and corresponding afterdrop values.

Rewarming rates may also affect afterdrop. Golden (1983) felt that various methods altered the speed of afterdrop re-

versal but not its magnitude. Pugh (1966) showed that although the amount of afterdrop remained the same for slow or rapid cooling, the duration was shorter in rapid rewarming by hot water immersion. Burton and Edholm (1955) stated that quick surface rewarming, like hot water immersion, may actually minimize afterdrop to 1^{0} C or less. It is generally accepted that shell methods produce greater afterdrop than core methods.

Rewarming Shock

Rewarming shock has been documented mainly in subacute hypothermia as protracted exposure results in plasma volume loss via shifts and diuresis (Hayward & Steinman, 1975). Burton and Edholm (1955) presented a well accepted explanation that rewarming mediated vasodilation presented a need for increased cardiac output. As plasma volume had been substantially decreased, severe hypotension or hypovolemic shock resulted (Collis et al., 1977 ; Golden, 1983). Death may also occur as the return of cold acidotic blood carrying metabolites to the myocardium causes ventricular depression or fibrillation (Collis et al., 1977). Increased blood potassium concentrations may lead to dangerous cardiac excitation (Mills, 1983).

Postrescue Collapse

Postrescue collapse occurs in conjunction with cold water immersion and is somewhat parallel to rewarming shock. In this acute situation plasma loss results from diuresis rather than fluid shifts but the end result, hypovolemic shock, is the same but more pronounced. Cold diuresis however, causes much greater fluid loss than occurs from fluid shifts in subacute hypothermia. This may explain why death occurs much more commonly after cold water immersion.

Choice of Treatment

Some degree of controversy exists over choice of treatment regimes while other basic principles are generally accepted. Treatment differs in several ways. It can be slow or rapid, shell or core oriented, and result from an active application of exogenous heat or rely on passive endogenous heat production.

Hunter (1968) calculated that the maximum rate of restoration should be 1.1-1.6^{0} C per hour as a faster increase may lower systolic blood pressure below 100 mmHg or cause bradycardia below 50 beats per minute. Mechanisms for these responses have been discussed earlier. Hunter stated however that any means or rate of rapid rewarming may suffice for short duration hypothermia in young, otherwise healthy victims.

An effective guideline for rate of rewarming seems to relate to speed of onset. It has generally been excepted that slow rewarming is best for subacute cases while rapid rewarming serves best after acute onset (Golden, 1983). Zingg (1966), reported increased survival rates when victims of acute onset were rapidly rewarmed. It has been stated that moderate rewarming should be strictly avoided as the increased metabolic needs of newly rewarmed tissue is not supplied with sufficient O_2 from still cool blood (Kugelberg et al., 1967).

The choice between shell and core rewarming methods is dependent on initial core temperatures. Shell methods are recommended for core temperatures greater than 30-32^0 C. At lower temperatures these methods precipitate risks of afterdrop and rewarming shock. At initial core temperatures below 30^{0}C core rewarming should be initiated. As an added insurance against hypovolemic shock, fluid replacement should accompany treatment when temperatures fall below 33^0 C (Bristow, 1986).

Many factors influence the decision to allow passive reheating or to use active methods. Among these factors are, proximity to equipment, training level of rescue or medical personnel, previous success, desired rate of rewarming and the condition of the patient. Obviously if rapid rewarming is indicated, passive reheating will be inadequate. Seclusion in wilderness areas may also preclude the use of some active methods as group members will be unequipped.

Bristow (1986) stated that choice of method was not important to success of treatment if core temperatures were above 30° C. The method of treatment was important for moderately hypothermic victims above 25°C Tc. For cases below 25°C an immediate life threat existed and administration of core rewarming and perhaps drugs and CPR were critical to survival.

Moderate and severe situations are beyond the scope of this study which was limited to field treatment of mild hypothermia.

Several estimates have been made for the quantity of heat needed for treatment. Myers and his associates (1979) stated that if a person contains 60 kg water, 60 kcal are required to raise the body temperature 1° C. Metabolically, this would take two hours at an initial temperature of 30°C as BMR is reduced to 30 $kcal \cdot hr^{-1}$. They estimated the weight of the core; liver, heart, kidneys and brain, at 4.5 kg. The same BMR of 30 $kcal \cdot hr^{-1}$, would raise the core temperature 1° C in only 9 minutes if all the heat were supplied to those organs as only 4.5 kcal would be required. Roberts, Patton & Kerr (1983) also calculated energy requirements for rewarming. As the specific heat of the body is 0.8 $kcal \cdot hr^{-1} \cdot {}^{0}C^{-1}$ a 70 kg man would require an exogenous heat supply of 56 kcal to raise the entire body temperature 1° C. They estimated the core to comprise 60% body mass

therefore only 35.5 kcal would be required by core methods to produce the same 1^0 C increase. Their estimate of body core tissue was greater than that given by Meyers and his coworkers resulting in a larger energy demand for an increase of 1^0 C. Although it is true that all of the heat cannot be supplied to the small core area, this clearly illustrates the advantages of supplying heat through core methods over peripheral methods which must warm the entire body mass from the outside in.

Three other considerations relate to choice of heat replacement method. The first is that normally, the major source of heat is the body's own metabolism which is depressed (Lloyd et al., 1972), therefore treatment should consider restoring this ability as quickly as possible. It is also of great importance to quantify the ability of a given rewarming method to supply required heat. Finally, an understanding of how body functions assist (shivering) or hinder (respiratory heat loss) the rewarming process is essential.

External methods are capable of delivering large amounts of heat especially if a hot bath at $40-45^0$ C is used (Paton, 1983). These methods are good for mild cases especially if heat is applied to regions of high heat transfer such as the neck, groin and lateral chest (Kuehn, 1983).

For most external methods however, initial heat transfer is low. It occurs mainly through physical conduction as peripheral constriction inhibits blood and heat flow (Collis et al., 1977). A subsequent rapid decrease in constriction creates the need for increased cardiac output. Hypovolemia and relative hypoxia result as plasma volume cannot increase fast enough to meet the demand (Paton, 1983).

Heated peripheral receptors signal the hypothalamus to suppress shivering and consequently heat production (Kuehn, 1983). If the heat supplied is not at least equal to the subsequent loss of heat production from decreased shivering, a negative heat balance will ensue.

Core rewarming procedures have the advantage of supplying heat directly to the all important core circulation resulting in a faster rate of temperature increase (Martyn, 1981). All core methods other than ingestion of hot fluids and inhalation of heated water saturated air are invasive and result in increased morbidity. As they are also surgical procedures, the review will be limited to the two treatments cited above. Inhalation of heated water saturated air not only supplies heat to the core but also reduces heat loss through the nasopharynx and respiratory tree (Martyn, 1981). Core methods do not disturb peripheral constriction and allow gradual rewarming of noncore tissue from the inside out (Kuehn, 1983). There is a substantial reduction in risk of afterdrop and rewarming shock.

Kuehn (1983) listed rates of rewarming for various methods. They are as follows: hot bath immersion , 3-4$^{0}C \cdot hr^{-1}$; inhalation of heated saturated air , 0.5-2$^{0}C \cdot hr^{-1}$; and ingestion of hot food and drinks - negligable. It has been reported that shivering thermogenesis may yield 250 $kcal \cdot hr^{-1}$ and produce no afterdrop while inhalation of heated water saturated air also results in no afterdrop (Hayward et al., 1983). As yet little information is available relavent to effectiveness of heating pads in rewarming or limiting afterdrop. To be effective, they must transfer enough heat to more than compensate for the loss in shivering thermogenesis, which they have been show to induce, in order to be beneficial.

The following sections will describe three rewarming methods and attempt to quantify the amount of heat delivered by each one. These treatments represent a variety of method classifications. Shivering thermogenesis represents a passive endogenous heat source while an exercise regime will actively provide endogenous heat. The most effective shell rewarming method, hot water immersion, is not applicable in the field, however a heating pad will be introduced as a shell treatment method for rewarming.

SHIVERING THERMOGENESIS

Shivering, defined as involuntary oscillation of skeletal muscle (Pozos & Wittmers, 1983), is a good tool for the body to generate additional heat as ATP is converted to ADP, Pi and energy (Martyn, 1981). The production of heat from shivering thermogenesis may be substantial. As the BMR at core temperature of 30° C is merely 30 $kcal \cdot hr^{-1}$, 2 hours would be required to raise the temperature of a body containing 60 kg water 1° C. The generation of an extra 250 $kcal \cdot hr^{-1}$ via shivering would greatly increase the rate of heat gain (Myers et al., 1979).

Shivering progresses from the arms and legs to the chest and trunk. The rate of heat production varies between individuals, and it has been speculated that up to 10% of the population does not shiver noticeably in cold temperatures (Pozos & Wittmers, 1983).

Shivering is activated by cold receptors in the periphery and its' amplitude may be influenced by inspired air temperature (Pozos & Wittmers, 1983). Shivering is greatest with no other treatments as it is inhibited by warming of peripheral receptors (Collis et al., 1977). Inhalation of warm air has also been shown to inhibit shivering (Conn, 1980).

Burton and Edholm (1955) showed that shivering stopped during cooling at core temperatures between 30-33° C and commenced at 30° C during rewarming. If this process stops

at a Tc of 33° C it is likely due to exhaustion of glycogen stores while muscle rigidity would interfere at Tc < 30° C (Martyn, 1981).

There are some disadvantages to this method. Heat production is increased but the raised metabolic demand of the muscles causes peripheral vasodilation and subsequent cooling of blood before it returns to the core. This may induce ventricular fibrillation at lower Tc's. Heart rate is increased and glycogen reserves are depleted (Hunter, 1968; Martyn, 1981). Collis and his associates (1977) compared shivering to four other treatments and found that the recovery rate for shivering thermogenesis was the slowest of all treatments.

The measurement of O_2 consumption is used to quantify the metabolic heat production from shivering thermogenesis.

According to Nielsen (1969), the following equation describes heat production:

$$H = M - W \qquad (4)$$

where H = total heat production in $kcal \cdot hr^{-1}$, M = metabolic energy liberation in $kcal \cdot hr^{-1}$, and W = external work in $kcal \cdot hr^{-1}$.

When BMR is accounted for and W equals 0 $kcal \cdot hr^{-1}$ (no mechanical work is done as the subject lies motionless) this equation can be altered to calculate heat production from shivering (Hs):

$$Hs = M - BMR \quad (5)$$

where M and $BMR = \dot{V}O_2$ $l \cdot min^{-1} \times 4.9$ kcal $\cdot$ $l^{-1} \times 60$ $min \cdot hr^{-1}$ (6).

EXERCISE METABOLISM

Kaufman (1983) stated that external work was the only available mechanism to increase metabolism as modest exercise may double or triple heat production. Exercise is inappropriate for the severely hypothermic , as the lower limit of Tc compatible to continuous exercise may be 34-35^0 C (Freeman & Pugh, 1969). In mild situations, exercise may negate further heat loss if no other rewarming methods are available.

Alexander (1945) reported one set of Dachau experiments designed to evaluate body heat rewarming as male subjects were placed in full body contact with female prisoners. The general effect on recovery was negligable but a surprising phenomenon occurred in four subjects. While still at Tc 30-32^0 C these men engaged in sexual intercourse with the female prisoners and their subsequent recoveries were substantially faster than the other subjects. It seems logical to assume that exercise induced metabolic heat was responsible for these dramatic rises in Tc.

There is little experimental evidence of exercise benefits for hypothermia, but Kaufman (1983) referenced one experiment where afterdrop magnitude and rate of temperature

increase were comparable for inhalation rewarming and mild exercise.

Heat gain is easily explained as the body performs work at approximately 20% efficiency. Of total energy expended to perform exercise, 80% will be given off as heat while only 20% results in actual work. Nielsen (1969) proposed the following equation for walking on a treadmill:

$$H = M - W \qquad (4)$$

where H = total heat production in $kcal \cdot hr^{-1}$, M = metabolic energy liberation in $kcal \cdot hr^{-1}$, and W = external work in $kcal \cdot hr^{-1}$.

$$M = \dot{V}O2\ l \cdot min^{-1} \times 4.9\ kcal \cdot l^{-1} \times 60\ min \cdot hr^{-1} \qquad (6)$$

W was obtained by actual VO_2 measurement of normothermic subjects at the work rates they had experienced in the hypothermic conditions.

Nielsen found that core temperature was directly proportional to M while sweat secretion was proportional to H. This conclusion parallels findings by I. Astrand (1960) who found that increases in body temperatures were related to work load or % $\dot{V}O_2$ max.

It is hypothesized that metabolic heat from exercise is a viable source for rewarming and may be used to a victim's advantage. This method is not without its disadvantages

however. Energy expenditure is difficult to quantify or control and vasodilation in active muscles may add to cardiac stress (Kaufman, 1983). This vasodilation may also result in decreased blood pressure during the commencement of exercise. It is also likely that the afterdrop will increase as circulation to cold muscles increases.

EXTERNAL APPLICATION OF HEAT

Collis et al (1977) compared various practical rewarming methods including peripheral warming via heating pads. They found that although the rate of rewarming was slightly higher than that for shivering thermogenesis, the afterdrop was also greater. It was suggested that peripheral vasodilation enhanced central cooling and that shivering was subjectively decreased as peripheral receptors were warmed.

If external heat does inhibit shivering, it must produce at least enough heat to compensate for that loss from decreased shivering in order to be effective.

CONCLUSION

This chapter commenced with a historical overview of observations and research into the area of cold exposure. Hypothermia was classified as mild, moderate or severe. Acute exposure, usually involving cold water immersion, occurs for less than 6 hours while subacute exposure lasts longer than

6 hours. The drop in body core temperature results from one or a combination of the following causes: inadequate heat production; increased heat loss; and impairment of temperature regulation.

The physiological responses to hypothermia include decreased cardiovascular performance, vasoconstriction, fluid shifts, fluid loss, decreased metabolic demands and production, impaired neural, hepatic and renal function.

Methods for treatment of hypothermia were categorized as active or passive, rapid or slow, and shell or core. The three treatments described in detail were shivering thermogenesis (passive), exercise metabolism (active), and external heat application (active, shell).

Although there is disagreement concerning the optimal rate of rewarming for all victims, it is generally accepted that rewarming should be fast for acute victims and slow for subacute cases.

Many field and all emergency room treatment procedures have been quantified and analysed extensively. However, little or no scientific study has addressed two of the treatments in this study, exercise metabolism, and the application of external heat.

The location of hypothermic trauma is often far from medical help necessitating treatment in the field. The treat-

ment must be simple, noninvasive and require little equipment. It is with these needs in mind that an experiment was designed to compare the effectiveness of two unsubstantiated methods of treatment with a control (shivering thermogenesis) method.

METHODS AND PROCEDURES

An experiment was conducted to evaluate and compare the effectiveness of three rewarming procedures for the treatment of hypothermia in a field situation. The treatments consisted of shivering thermogenesis, exercise metabolism, and the application of external heat.

This chapter contains sections describing subjects, screening, experimental design, procedures, instrumentation and data analysis.

A pilot study was conducted to verify protocol and instrumentation procedures.

SUBJECTS

Subjects consisted of 7 healthy volunteers (6 male and 1 female) of average build and physical activity levels. In order to participate in the experiment subjects were required to meet stringent medical standards and fulfill average fitness requirements. They were informed of the details of the experimental protocol and the associated risks and discomforts. The protocol was approved by the Faculty's human ethics committee and written informed consent (Appendix 1) was obtained from all subjects.

SCREENING

Initial Screening Procedure

After reporting to the exercise physiology laboratory subjects were given a Physical Activity Readiness Questionnaire (Par Q) (British Columbia Ministry of Health, 1978) verbally in order to screen for any obvious contraindications to participation in the experiment. They were then asked to read and sign an informed consent form with the knowledge that even after signing they were not committed to completing the entire experiment as they were free to withdraw from this experiment at any time.

Age and anthropometric measures were then taken. Height, weight and sum of four skinfold thicknesses were recorded. Skinfolds were measured using fat calipers at sites on the biceps, triceps, subscapula and suprailiac.

Subjects performed a maximal exercise test on a treadmill (Bruce et al., 1963) to determine $\dot{V}O_2$ max. A minimum value of 40 $ml \cdot kg^{-1} \cdot min^{-1}$ was required for participation in the experiment.

Medical Examination

Subjects were asked to report to the attending physician for a medical examination. A medical history was taken and general medical suitability for this experiment was determined. Specifically, the physician was concerned with any

cardiovascular or pulmonary conditions which would have contraindicated continuous cold water immersion.

EXPERIMENTAL DESIGN

Each subject was asked to undergo the cooling procedure three times. One subject completed 4 procedures while one dropped out after only one trial because of inability to cool significantly due to high skinfold thickness. The latter subject's data was not analysed. Upon completion of each cooling phase, a different one of the three treatment procedures was conducted.

Treatments were administered in a randomized complete block design with subjects serving as their own controls.

DATA ANALYSIS

The data for subject GM was not used as only one treatment was completed.

Of the experimental parameters, only core tempreature was analysed as it best indicated the efficiency of the treatments.

The data for afterdrop amount, length of afterdrop period, and rate of Tc increase were subjected to a one way analysis of variance for repeated measures. The .05 level of significance was chosen. Post hoc analyses for signifi-

cant differences between treatments were accomplished by Tukey's post hoc test.

Afterdrop was calculated as the drop in Tc, after removal of the subject from the water, until it started to increase.

Length of the afterdrop period included the time from removal from the cold insult until Tc returned to the original removal Tc.

The rate of core temperature increase was estimated by linear regression using the sum of least squares method. This analysis did not include the entire afterdrop period as only data occuring after Tc started to rise was considered.

Subjects fell into two distinct subgroups according to skinfold thickness. Some of the analyses compared 'low fat' subgroups (sum of skinfolds < 40 mm) and 'high fat' subgroups (sum of skinfolds > 50 mm).

EXPERIMENTAL PROCEDURES

Intervals between experimental procedures were established at a minimum of one week for each subject in order to minimize possible residual effects from previous cold exposure. Subjects were asked to report at the same time of day to control for circadian effects.

Subjects were instructed to abstain from alcohol or medications for a period of 24 hours prior to each experiment.

They were also instructed not to exercise or eat within 6 hours of reporting. They then reported to the laboratory to prepare for the water immersion portion of the experiment. This procedure was identical for all visits.

Cooling Procedure

After subjects changed into a bathing suit, skin temperature thermistors were taped to the calf, thigh, chest and arm. Three ECG electrodes were also applied to the chest area. A physician, who was in attendance throughout each experiment, inserted a thermistor into the nasopharanx down the esophagus to the level of the heart. The esophageal temperature was used to indicate body core temperature (Tc). Subjects were then asked to lie motionless for a period of 10 minutes during which baseline values for HR, BP, Tc, and $\dot{V}\ O_2$ were established. They then entered the tank of water ($20^{o}C$) and remained submerged at approximately shoulder level. The temperature of the stirred water was lowered quickly to $8.0 \pm 0.5^{o}C$ as ice was added. Blood pressure and ECG were monitored every 15 minutes by a physician. $\dot{V}O_2$ was monitored until a steady state was reached. Monitoring recommenced just before exit from the tank. Subjects remained in the water until either they wished to terminate immersion, the physician advised exit for safety reasons, or body core temperature reached $33^{o}C$. Some of the subjects cooled very slowly as they had high sums of skinfolds. It was

clear that it would be too uncomfortable for them to stay immersed long enough to reach the initial target Tc. These subjects were removed at Tc ranging from 34-35^{o}C.

At this point subjects were helped out of the tank and one of three treatment procedures commenced.

Treatment Procedures

After each water immersion, subjects were lightly towel dried and insulated from ambient air temperature. For all treatments except exercise metabolism, insulation was accomplished by placing the subject in a vapour proof rescue bag. This insulation was approximated during exercise treatments by wearing a set of pile clothing. Insulation was necessary to prevent external heat effects as ambient temperature was greater than skin temperature at the commencement of treatment.

Treatment began within 2 minutes after the subject was removed from the cold insult. During treatments, temperature values were recorded at 1 minute intervals, while HR and BP were monitored intermittently. Metabolic parameters such as $\dot{V}O_2$ and $\dot{V}E$ were continuously monitored and recorded by the Sensormedics (Horizon) Metabolic Measurement Cart.

Treatment procedures for subjects who reached the target Tc of 33^{o}C, terminated when Tc reached 35.5^{o}C which was out of the hypothermic range. Subjects who exited at Tc > 33^{o}C

continued treatments until Tc increased to 36.0°C. Treatments were not continued past these points as enough data was available to establish linear rates of Tc increase.

Shivering Thermogenesis

Subjects were asked to lie on a mattress inside the insulated rescue bag. Body temperature increased as a result of metabolic heat produced by shivering thermogenesis.

Exercise Metabolism

After towel drying, subjects were fitted with one set of pile clothing (to approximate the insulation effect of the rescue bag) and asked to walk on a treadmill. The original workload was set at 1.1 kph and 4% grade. Subjects were asked to walk at the fastest pace they could comfortably maintain and the speed was increased, at their direction, gradually to 5.6 kph.

External Heat Application

A recent arrival on the cold related emergency response scene is the STK Heatpac. The Heatpac consists of a combustion chamber (charcoal fuel) and a branched heating duct. The heating duct is applied to areas of high heat transfer on the body and peripheral circulation is warmed resulting in eventual core rewarming.

At this time the Heatpac has not been used for treatment for hypothermic victims but for preventative temperature maintenance of injured persons during evacuation (Hamlet, 1986).

In this experiment the tube ducts from the Heatpac were placed around the lateral chest. Subjects were then instructed to lie motionless in the rescue bag until Tc increased sufficiently.

INSTRUMENTATION

Several paramaters were measured during the experimental process. These paramaters were categorized as a) safety monitoring, b) measurement of treatment effectiveness, c) quantification of heat supply by treatment modalities, and d) metabolism.

Safety Monitoring

Single channel ECG as well as heart rate were monitored on a Hewlitt-Packard DC powered monitor defibrilator at the modified chest lead (MCL 1) position. A sphygmomanometer measured blood pressure.

Measurement of Treatment Effectiveness and Quantification of Heat Supplied by Treatment Modalities

Body core temperature (Tc) was measured at a single site in the esophagus as Nielsen (1969) and Hayward (1979) stated that the best noninvasive representation of T blood in the heart could be gained from a thermistor inserted into the esophagus to the level of the heart. A thermistor was connected to a Yellow Springs Instruments telethermometer.

Mean skin temperature (MST) was established by four thermistors which conducted impulses to a Syborg telethermometer. A switching device controlled input from 4 skin locations to the telethermometer. MST was calculated according to Shanks (1975):

$$MST = 0.3(Tchest + Tarm) + 0.2(Tthigh + Tcalf) \quad (10).$$

Metabolism

A Critical Care/Nutritional Assessment Program was used on the Sensormedics (Horizon) Metabolic Measurement Cart (MMC) to analyse expired breath and record $\dot{V}O_2$, $\dot{V}E$, and $\dot{V}T$. Sampling took place during a baseline period prior to immersion, at the beginning and end of the immersion periods, and throughout each treatment procedure. During each collection period, parameters were averaged at one minute intervals and recorded. The MMC was calibrated before and after each test to ensure that no drift had taken place.

RESULTS AND DISCUSSION

SUBJECTS

Physical Characteristics

A physical description of each subject is presented in Table 1. There were 6 males and 1 female with the following characteristics ($\bar{x}$, SD): age (31.3 ± 5.6 yrs); height (179.2 ± 6.5 cm); weight (75.8 ± 18.2 kg); sum of 4 skinfolds (51.8 ± 19.5 mm); and $\dot{V}O_2$ max (49.5 ± 9.1 $ml \cdot kg^{-1}$).

When skinfold thickness and $\dot{V}O_2$ max were considered, the subjects fell into two distinct subgroups which were used for some of the analyses. A 'low fat' subgroup consisted of 3 subjects with sum of skinfolds < 40 mm and $\dot{V}O_2max$ > 50 $ml \cdot kg^{-1} \cdot min^{-1}$. 'High fat' subgroup members (4) had sum of skinfolds > 55 mm and $\dot{V}O_2$ max < 50 $ml \cdot kg^{-1} \cdot min^{-1}$.

TABLE 1

Physical descriptors of all subjects.

SUBJECT	SEX	AGE (yrs)	HEIGHT (cm)	WEIGHT (kg)	SUM OF SKINFOLDS (mm) <FATCLASS>	VO2 MAX (ml/kg/min)
PH	M	32	179.5	88.4	56.6 <high>	45.6
JH	F	22	170.0	75.2	80.8 <high>	45.0
RS	M	36	172.5	68.7	29.0 <low>	52.4
BT	M	36	178.0	76.8	61.6 <high>	42.0
TS	M	28	182.5	77.4	33.0 <low>	67.9
GG	M	28	183.0	77.1	36.0 <low>	51.3
MEAN		31.3	179.2	75.8	51.8	49.5
SD		5.6	6.5	18.2	19.5	9.1

POST IMMERSION PHASE

As this thesis focuses on variations between treatment types in the rewarming phase of the experiment, the cooling phase will not be reported in detail. However, immersion cooling rates, relative to post immersion afterdrop rates, will be analysed in a later section. Group data for the period of time after removal from cold insult (post immersion phase) will be analysed in the following 2 sections; afterdrop period, and period of core temperature increase. The first section will be directed towards hypotheses (1) and (2) which address afterdrop amount and length. Hypothesis (3) is conserned with rates of Tc increase and will be analysed in the second section.

In order to better explain the overall mechanisms for and effectiveness of the three treatments studied, the final two sections will present individual case studies. The 'individual treatments' section will analyse one trial of each treatment protocol in detail. The 'individual rewarming rates' section will compare effects of each treatment on Tc for individual subjects.

All parameters relating to Tc activity during all experiments are tabulated in Table 2. In subsequent sections, specific results will be depicted graphically for more complete analysis.

TABLE 2

Individual and group experimental parameters during trials with three treatment procedures.

SUBJECT	TREATMENT	COOLING RATE (°C/hr)	AD RATE	AD AMOUNT (°C)	AD LENGTH (min)	REWARMING RATE (°C/hr)
GG	EXERCISE	3.90	3.60	0.70	15	4.50
	SHIVERING	3.96	9.90	0.40	17	4.20
	EXT HEAT	3.00	3.75	0.25	15	4.15
RS	EXERCISE	4.02	4.14	0.90	23	5.50
	SHIVERING	3.78	8.52	0.70	9	5.13
	EXT HEAT	3.42	7.50	0.25	12	5.00
TS	EXERCISE	2.64	7.80	0.50	16	5.70
	SHIVERING	2.10	3.00	0.10	8	3.80
	EXT HEAT	1.86	1.86	0.20	10	5.52
BT	EXERCISE	2.88	7.10	1.05	35	3.80
	SHIVERING	2.10	6.00	0.10	18	3.10
	EXT HEAT	1.86	4.30	0.40	22	1.94
PH	EXERCISE	1.20	3.60	1.00	42	4.60
	SHIVERING	2.50	8.10	0.45	8	2.90
	EXT HEAT	3.06	2.70	0.30	9	3.57
JH	EXERCISE	1.30	6.36	1.30	30	5.80
	SHIVERING	2.49	1.02	0.20	24	1.60
	EXT HEAT	2.48	3.75	0.50	23	2.60
GROUP MEAN SD	EXERCISE	2.65	5.43¬	0.91***	26.8***	4.98*
		1.21	1.87	0.28	10.7	0.80
	SHIVERING	2.82	6.09¬	0.33	14.0	3.45
		0.83	3.45	0.24	6.7	1.20
	EXT HEAT	2.61	3.97	0.32	15.1	3.79
		0.67	1.93	0.11	6.0	1.40
LOWFAT MEAN SD	EXERCISE	3.52	5.18	0.70**	18.0 ns	5.23 ns
		0.76	2.28	0.20	4.3	0.64
	SHIVERING	3.28	7.14	0.40	11.3	4.37
		1.02	3.65	0.30	4.9	0.68
	EXT HEAT	2.76	4.37	0.23	12.3	4.89
		0.81	2.87	0.03	2.5	0.69
HIGHFAT MEAN SD	EXERCISE	1.79	5.68	1.11**	35.6 ns	4.73 ns
		0.94	1.84	0.16	6.0	1.01
	SHIVERING	2.37	5.04	0.25	16.7	2.53
		0.23	3.63	0.18	8.0	0.81
	EXT HEAT	2.46	3.58	0.40	18.0	2.70
		0.60	0.81	0.10	7.8	0.81

¬ AD Rate > Cooling Rate ** Ex > Ext Heat
*** Ex > Shiv & Ext Heat * Ex > Shiv

AFTERDROP PERIOD

Introduction

This is the period commencing at removal from cold insult during which Tc continues to drop to its nadir. Several parameters were studied. The rates of Tc decrease during immersion (rate of cooling) were compared to the rate of post immersion Tc decrease (rate of afterdrop). The absolute amount of afterdrop and length of afterdrop periods were also analysed. Analyses include the whole group as well as low fat and high fat subgroups. Calculations encompass all trials as well as differentiate between treatments. The general purpose of the analyses was to evaluate the effectiveness and safety of the treatments and to attempt to better understand the afterdrop phenomenon.

Results

Amount of Afterdrop

The amount of afterdrop for the entire group, as well as for the low fat and high fat subgroups, within three treatment protocols is summarized in Figure 1. Analylsis for the group as a whole indicated that the afterdrop during exercise (0.91 ± 0.28 ^{0}C) was significantly higher ($p<.05$) than for both shivering thermogenesis (0.33 ± 0.24^{0}C) or application of external heat (0.32 ± 0.11^{0}C). The difference between the latter two treatments was not significant. Based on these results, null hypothesis (1) was rejected and the

alternate hypothesis, that the afterdrop amount was different for at least two treatments, was accepted.

Results for the high fat subgroup were similar to the results of the whole group, but the difference between exercise and the other two treatments was greatly accentuated. The exercise protocol resulted in a higher ($p<.05$) afterdrop amount ($1.11 \pm 0.16^{o}C$) than in both shivering thermogenesis ($0.25 \pm 0.18^{o}C$) and application of external heat ($0.4 \pm 0.1\ ^{o}C$). Again the afterdrop amounts in the latter two treatments were not siginificantly different.

In the low fat group, the afterdrop amount during exercise ($0.7 \pm 0.2^{o}C$) was significantly higher than the application of external heat ($0.23 \pm 0.03^{o}C$), but not for shivering ($0.4 \pm 0.3^{o}C$).

Comparison of subgroups within treatments revealed that during both exercise and the application of external heat, the amount of afterdrop was highest in the high fat subgroup and lowest in the low fat subgroup. This trend was reversed in the shivering thermogenesis protocol where the low fat subgroup possesed the highest afterdrop amounts.

Figure 1: Amount of core temperature (Tc) afterdrop vs method of treatment for whole group, low fat and high fat subgroups.

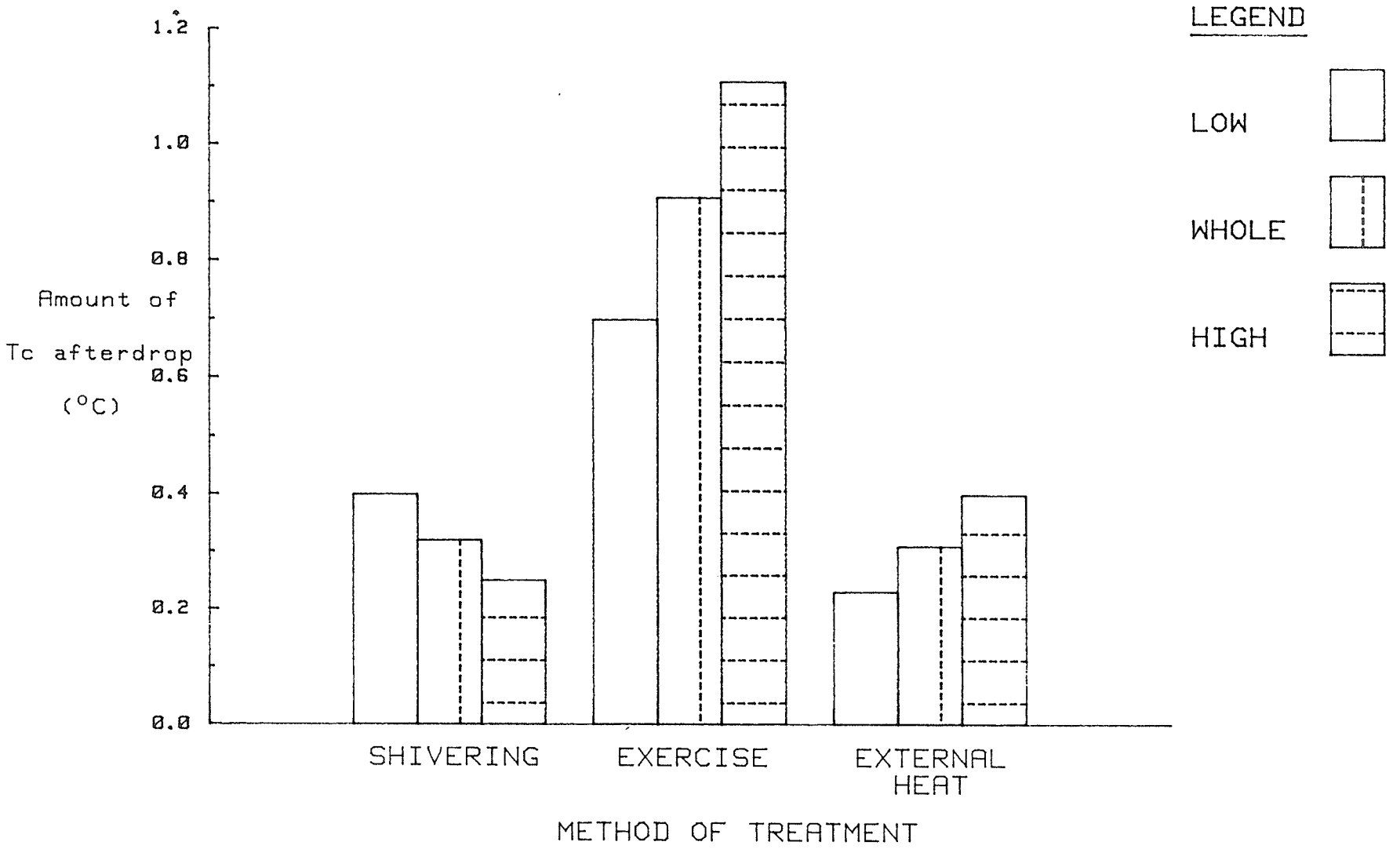
LEGEND
LOW
WHOLE
HIGH
1.2
1.0
0.8
0.6
0.4
0.2
0.0
Amount of
Tc afterdrop
(°C)
SHIVERING
EXERCISE
EXTERNAL
HEAT
METHOD OF TREATMENT

FIGURE 1

Based on these findings it appears that increased muscular circulation is probably the explanation for an increased afterdrop during exercise. The large muscles used for walking are idle and possess comparatively low blood flows during shivering and external heat application. In fact these two treatments would result in very little change in blood flow except where external heat would increase chest circulation. The chest, being a central area, would already have relatively higher blood flow than the legs and arms.

The different responses of high and low fat subgroups are a function of physical conduction by, and circulation through, fat tissue. A body with higher fat content would obviously possess more cold tissue upon removal from cold insult. The circulatory aspect for afterdrop would be increased as blood would be perfused through the many more capillaries exisiting in the extra fat tissue. The larger tissue mass itself would affect more cooling of inferior layers via physical conduction, also adding to the afterdrop effect. Where less fat exists the mechanisms contributing to afterdrop are less evident and differences between treatments would be predictably minimized. Greater fat mass would increase differences between treatments as afterdrop mechanisms would be increased.

The trend during exercise and external heat application is to be expected and is explained by a circulatory model. Both procedures would result in increased blood flow; exer-

cise through muscles in the legs and arms, and external heat through the chest area. A larger amount of fat would result in greater cooling of the blood in each case.

During shivering however, minimal changes occur in blood flow. Extra fat may be beneficial in this case as the extra insulation it provides would retain more heat produced from shivering. This would explain the lower amount of afterdrop compared to the whole group and low fat subgroup.

Figure 2 compares the amount of afterdrop over all trials between the low fat (0.44 ± 0.27°C) and the high fat (0.59 ± 0.42°C) groups. When an overall comparison was made, no significant difference was present partially due to the opposite trend in shivering compared to the other two treatments.

Although the difference between the subgroups is not significant at the .05 level, it certainly exists and a larger sample size may result in significance.

Figure 2: Comparison of afterdrop amount between low fat and high fat subgoups with all conditions combined.

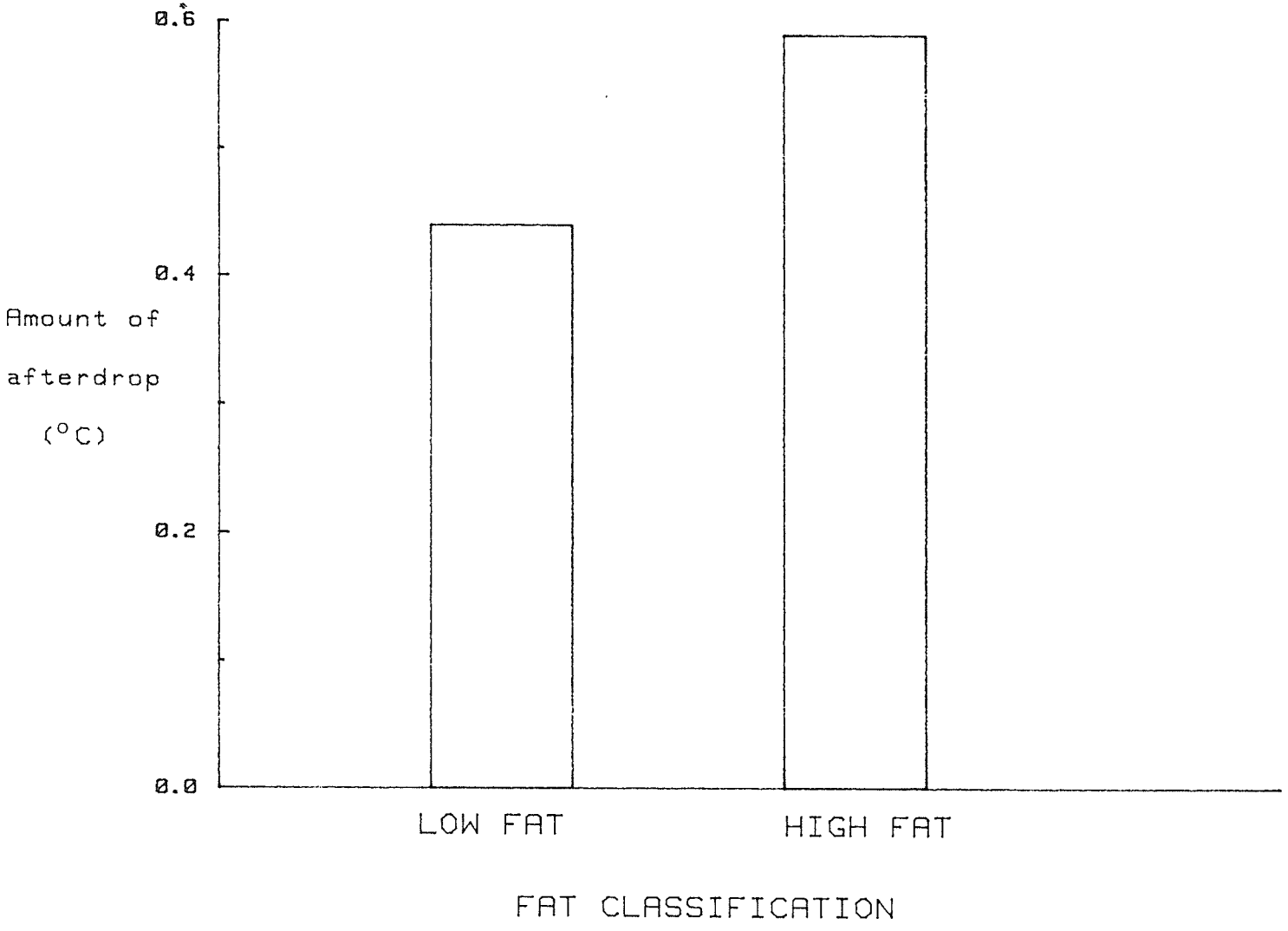
0.6
0.4
0.2
0.0
Amount of
afterdrop
(°C)
LOW FAT
HIGH FAT
FAT CLASSIFICATION

FIGURE 2

Length of Afterdrop

A comparison of length of afterdrop periods for whole, low fat and high fat subgroups in all three treatments is illustrated in Figure 3. For the whole group analysis, values for length of afterdrop period were higher ($p<.05$) in the exercise regime (26.8 ± 10.7 min) than in both application of external heat (15.1 ± 6.0 min) and shivering thermogenesis (14.0 ± 6.7 min). Results for shivering and external heat were not significantly different. Therefore null hypothesis (2) was rejected and the alternate hypothesis, that the length of afterdrop period was different for at least two treatments, was accepted.

The relationship for the high fat subgroup was similar but the difference between exercise and the other two treatments was much greater. Lengths of afterdrop period in the high fat subgroup were 35.6 ± 6.0, 18.0 ± 7.81, and 16.7 ± 8.0 min for exercise, external heat and shivering protocols respectively. Values for exercise were not significantly higher than for shivering or external heat, probably due to a small sample size (N=3).

Afterdrop periods were not significantly different between treatments in the low fat subgroup. These differences were much smaller than in the other two groups. Values for length of afterdrop period were 18.0 ± 4.35 min for exercise, 12.3 ± 2.5 min for application of external heat, and 11.3 ± 4.9 min for shivering thermogenesis.

Figure 3: Length of afterdrop period vs treatment method for whole group, low fat and high fat subgroups.

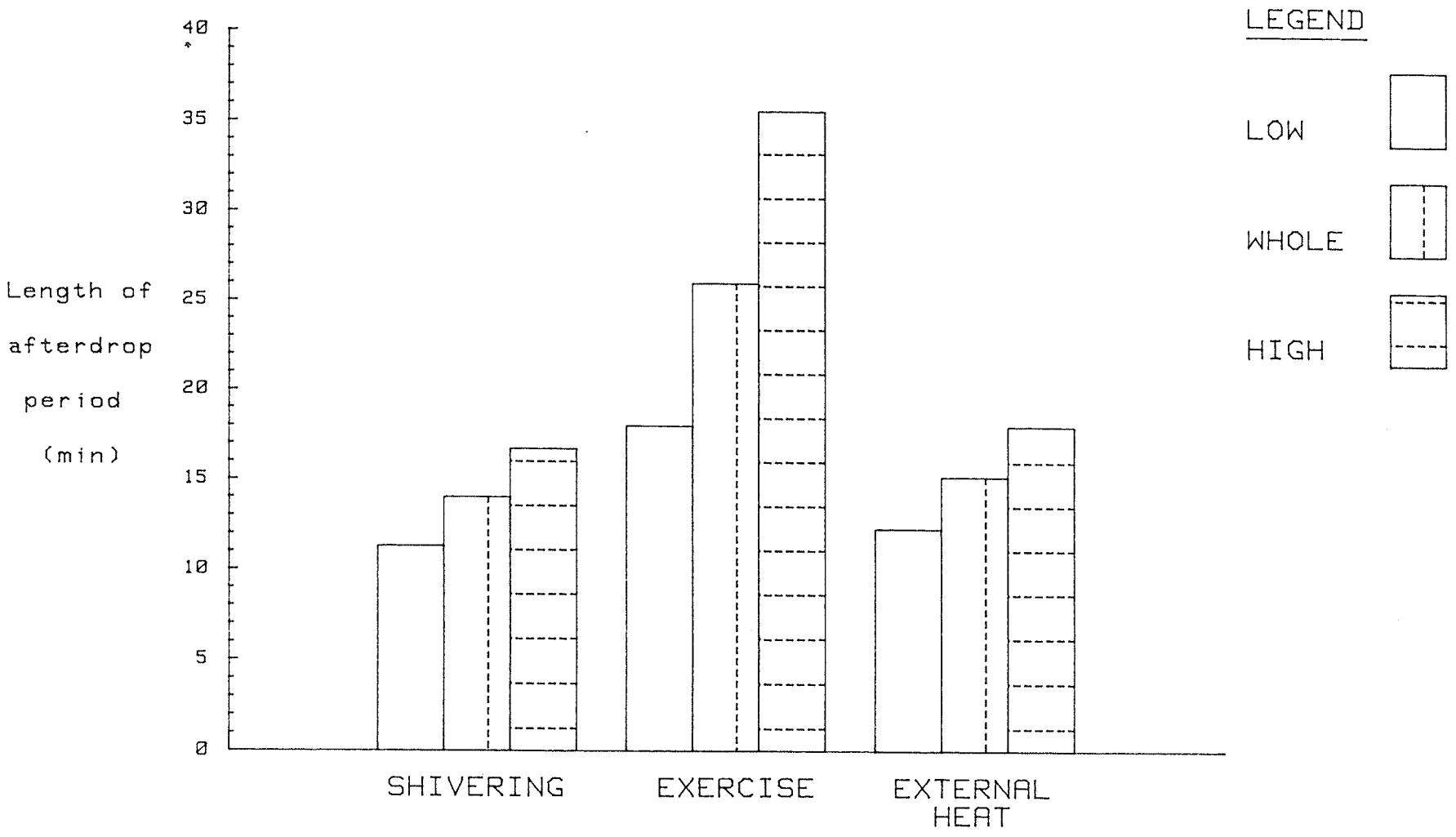
LEGEND
LOW
WHOLE
HIGH
Length of afterdrop period (min)
40
35
30
25
20
15
10
5
0
SHIVERING
EXERCISE
EXTERNAL HEAT
METHOD OF TREATMENT

FIGURE 3

The relationship between whole, low and high fat subgroups was the same in each treatment, as length of afterdrop period was longest in the high fat group and shortest in the low fat group.

Analysis of length of afterdrop among treatments revealed the same result in the whole group and both subgroups. Exercise produced the longest afterdrop periods. This would be expected as it produced the greatest amount of afterdrop therefore a longer period of time was necessary for recovery to initial core temperature upon removal.

The lengths of afterdrop period for the three groups in shivering and external heat were virtually equal. During external heat and exercise treatments, the relationship of body fat to length of afterdrop mirrored that found for amount of afterdrop in that the high fat subgroup had the longest period while the low fat subgroup had the shortest period. However these relationships were opposite during shivering. During shivering the insulation of higher fat mass may have retained more heat production resulting in lower afterdrop amount compared to the low fat group. However, the rewarming rate was slower for the high fat subgroup (see Figure 6) due to their larger mass, hence the longer afterdrop period.

The difference in length of afterdrop period between exercise and the other two treatments was accentuated in the

high fat subgroup compared to the low fat subgroup (very little difference) and even the whole group. Again this is explained by the combination of two effects of increased fat; higher levels of fat increase circulatory cooling, and there is now more mass which rewarms at a slower rate.

A comparison between length of afterdrop period for low fat (13.9 ± 4.7 min) and high fat (23.4 ± 11.2 min) subgroups during all treatments appears in Figure 4. The difference in length of afterdrop period, between the two subgroups was significant ($p<.05$).

Increased fat results in both a larger and longer afterdrop. The effect of body composition on length of afterdrop appears greater however as the observed difference is statistically significant. The increased cooling of blood in higher fat mass would naturally necessitate a larger period of time to reverse this phenomenon.

Figure 4: Comparison of length of afterdrop period between low fat and high fat subgroups for all conditions combined.

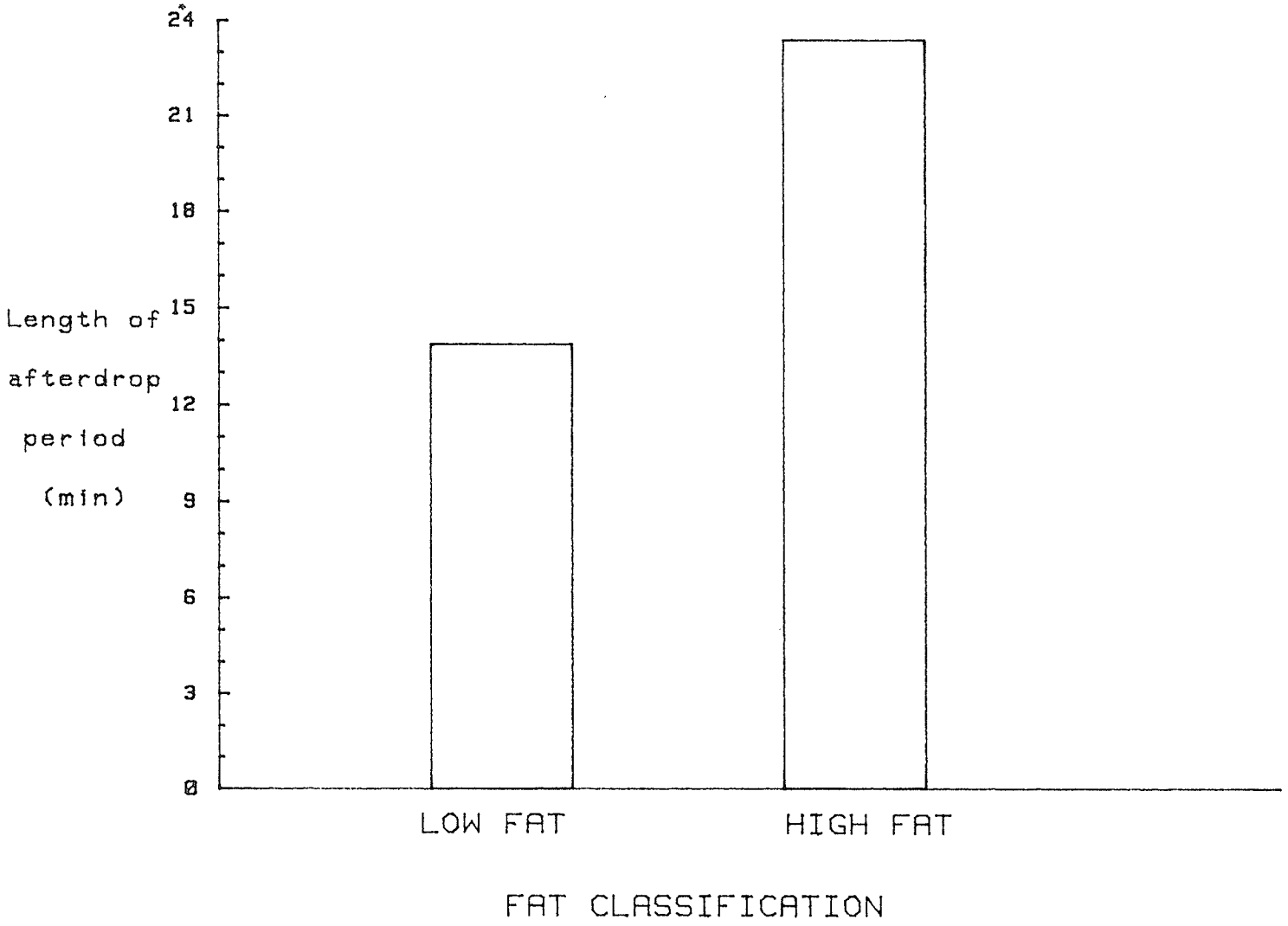
24
21
18
15
12
9
6
3
0
Length of afterdrop period (min)
LOW FAT
HIGH FAT
FAT CLASSIFICATION

FIGURE 4

Rate of Cooling vs Rate of Afterdrop

In order to evaluate whether or not Tc decreases at the same rate during cold water immersion and the afterdrop phase, these two rates were compared for each treatment. The relationship is illustrated in Figure 5. The afterdrop rate (6.09 ± 3.45 $^{0}C \cdot hr^{-1}$) was significantly ($p<.05$) higher than the immersion cooling rate (2.82 ± .83 $^{0}C \cdot hr^{-1}$) when shivering thermogenesis was studied. A significant ($p<.05$) difference also occurred between the afterdrop rate (5.43 ± 1.87 $^{0}C \cdot hr^{-1}$) and the immersion rate (2.65 ± 1.21 $^{0}C \cdot hr^{-1}$) during the exercise protocol. When the external heat was applied, no significant difference was found between the afterdrop rate and the immersion cooling rate which were 3.97 ± 1.93 $^{0}C \cdot hr^{-1}$ and 2.61 ± .66 $^{0}C \cdot hr^{-1}$ respectively.

Exercise and shivering produced rates of afterdrop higher ($p<.05$) than the immersion cooling rate. Although the afterdrop rate was greater than the cooling rate during the external heat treatment, the difference was not statistically significant. It is important that during shivering and external heat treatments, a large portion of the afterdrop took place in the two minute preparation interval between removal and commencement of treatments.

During this period the subjects were exercising as they moved from one location to another. Scrutinization of the data revealed that most of the afterdrop occured

Figure 5: Comparison of immersion cooling rate (CRT) and rate of afterdrop (ADR) for 3 treatments.

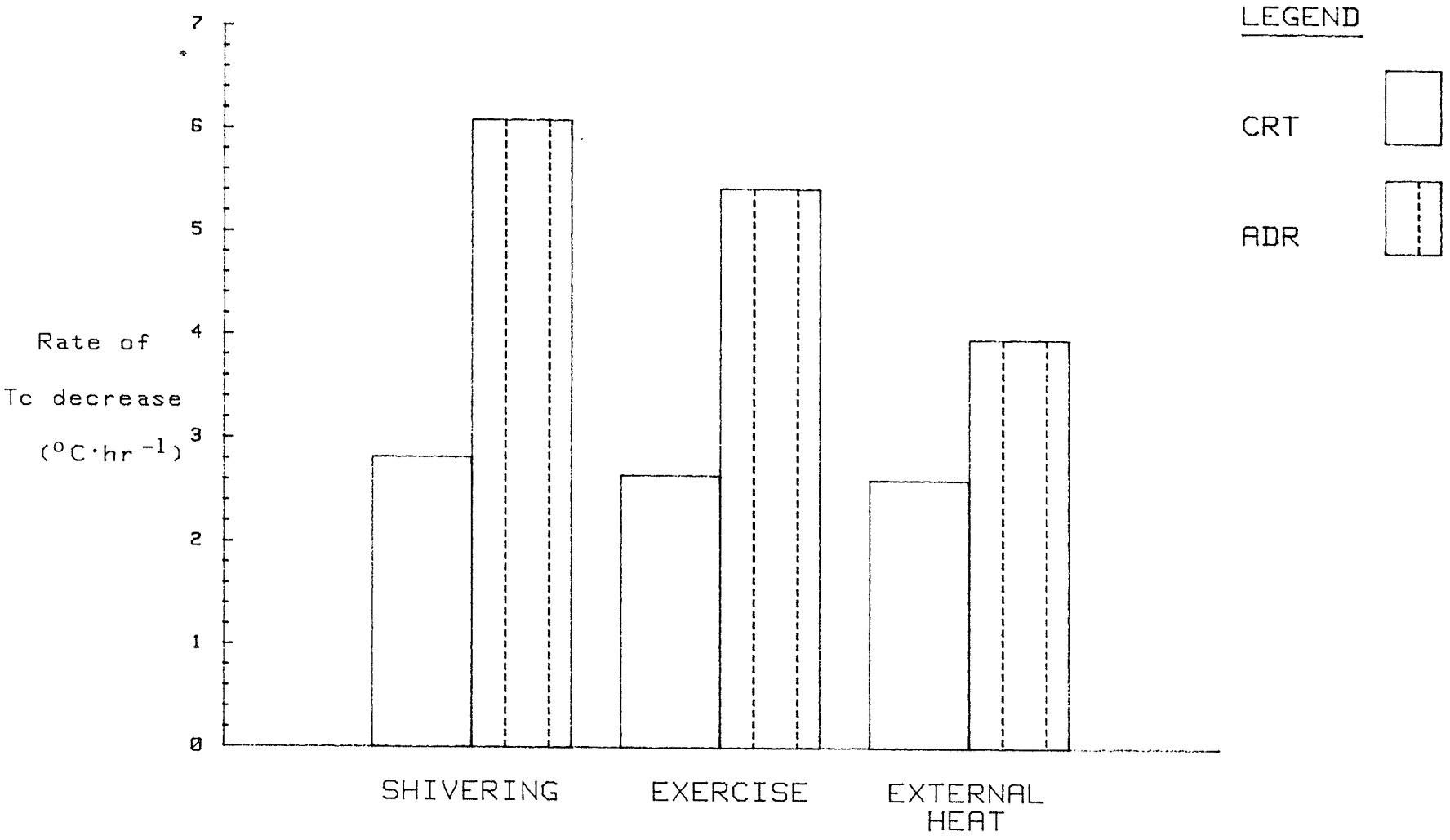
LEGEND
CRT
ADR
Rate of
Tc decrease
(°C·hr⁻¹)
7
6
5
4
3
2
1
0
SHIVERING
EXERCISE
EXTERNAL HEAT
METHOD OF TREATMENT

FIGURE 5

in the first 2 minutes of the afterdrop period during shivering and the application of external heat. It is unlikely that, in these two methods, the rates of afterdrop, excluding the preparation period, would be appreciably different from the immersion cooling rate.

It seems probable, that this increased rate of afterdrop is explained by the same mechanism which causes higher afterdrop rates during exercise. This afterdrop rate during exercise is valuable in explaining the mechanisms of afterdrop. There are several proposed mechanisms for the afterdrop phenomenon including: increased peripheral blood flow; physical conduction; and vasodilation in the cooler inner shell (muscles).

In resent years, it has been believed that afterdrop was caused by the return of cold blood previously trapped in the vasoconstricted periphery. This 'circulatory explanation' has been refuted in recent years. Savard and his coworkers (1985) showed that peripheral blood flow in the hand, forearm, calf and foot did not increase to a great extent until Tc had actually risen substantially during warm water immersion rewarming. They measured only one half the skin blood flow required to account for the entire afterdrop amount. Hamlet (1986), stated that if rates of afterdrop and immersion cooling are linear, the afterdrop is a function of mass action or physical conduction. It is proposed here however, that if the afterdrop cooling rate is significantly steeper

than in immersion cooling, circulatory effects must be prominent.

It was clear in this study that during exercise (and movement periods during shivering and external heat application), when muscle blood flow was increased to fuel activity, the afterdrop rate increased. This was especially evident in subject JH (Figure 11) where Tc dropped 1.3 ^{o}C after 9 minutes of exercise compared to a decrease of 1.7 ^{o}C in 87 minutes of immersion. Clearly, physical conduction could not account for such a drastic change. It is believed that core blood perfused cold muscles, fat and subcutaneous tissue, which previously had low blood flow, therefore cooling off before returning to the core.

On the other hand, shivering thermogenesis and application of external heat do not require movement or substantial increases in muscle blood flow. Therefore any afterdrop, exclusive of the two minute preparation period, would be more due to physical conduction and be expected to be minimal. This was supported in these results where afterdrop amounts ranged from 0.1 - 0.7 ^{o}C in the actual treatment phases of shivering and external heat procedures.

Golden and Hervey (1977) monitored afterdrop in live and dead pigs. They recorded similar afterdrops in both live and dead pigs and concluded that circulation was not needed to explain the drop in Tc but that a physical conduction

model was sufficient. They did not emphasize however that the afterdrop reaction was diminished and delayed slightly in the dead pigs. Webb (1986) also believed that no circulatory mechanism is needed to model afterdrop. If this is so, afterdrop rate and amount shouldn't be dependent on method of rewarming, but should be the same after all similar cooling experiences. From these results, it is believed that afterdrop is caused by a combination of mechanisms. If no increase in peripheral circulation occurs, afterdrop is cased mainly by physical conduction. If peripheral circulation does increase however, this will greatly affect the amount and rate of afterdrop.

PERIOD OF CORE TEMPERATURE INCREASE

Introduction

This phase encompases the period commencing when the afterdrop phenomenon reverses and Tc increases. A summary of rates of Tc increase between treatments for the whole group as well as low and high fat subgroups is presented in Figure 6.

Results

Whole group analysis revealed that rate of Tc increase for exercise ($4.98 \pm 0.8^{0}C \cdot hr^{-1}$) was significantly higher than for shivering thermogenesis ($3.45 \pm 1.2\ ^{0}C \cdot hr^{-1}$). Rate of Tc increase for application of external heat ($3.79 \pm 1.4\ ^{0}C \cdot hr^{-1}$) was similar to that for shivering and lower, although not at the .05 level of significance, than for exercise. It is probable that an increase in sample size (N) would result in significance at the .05 level. Null hypothesis (3) was rejected and the alternate hypothesis, that rates of Tc increase for at least two treatments are different, was accepted.

Rates of Tc increase were not significantly different between any treatment in either the low or high fat subgroups. In the high fat subgroup the difference in rate of Tc increase for exercise ($4.73 \pm 1.01^{0}C \cdot hr^{-1}$) was substantially higher, although not significantly (N=3), than that

Figure 6: Rate of Tc increase vs method of treatment.

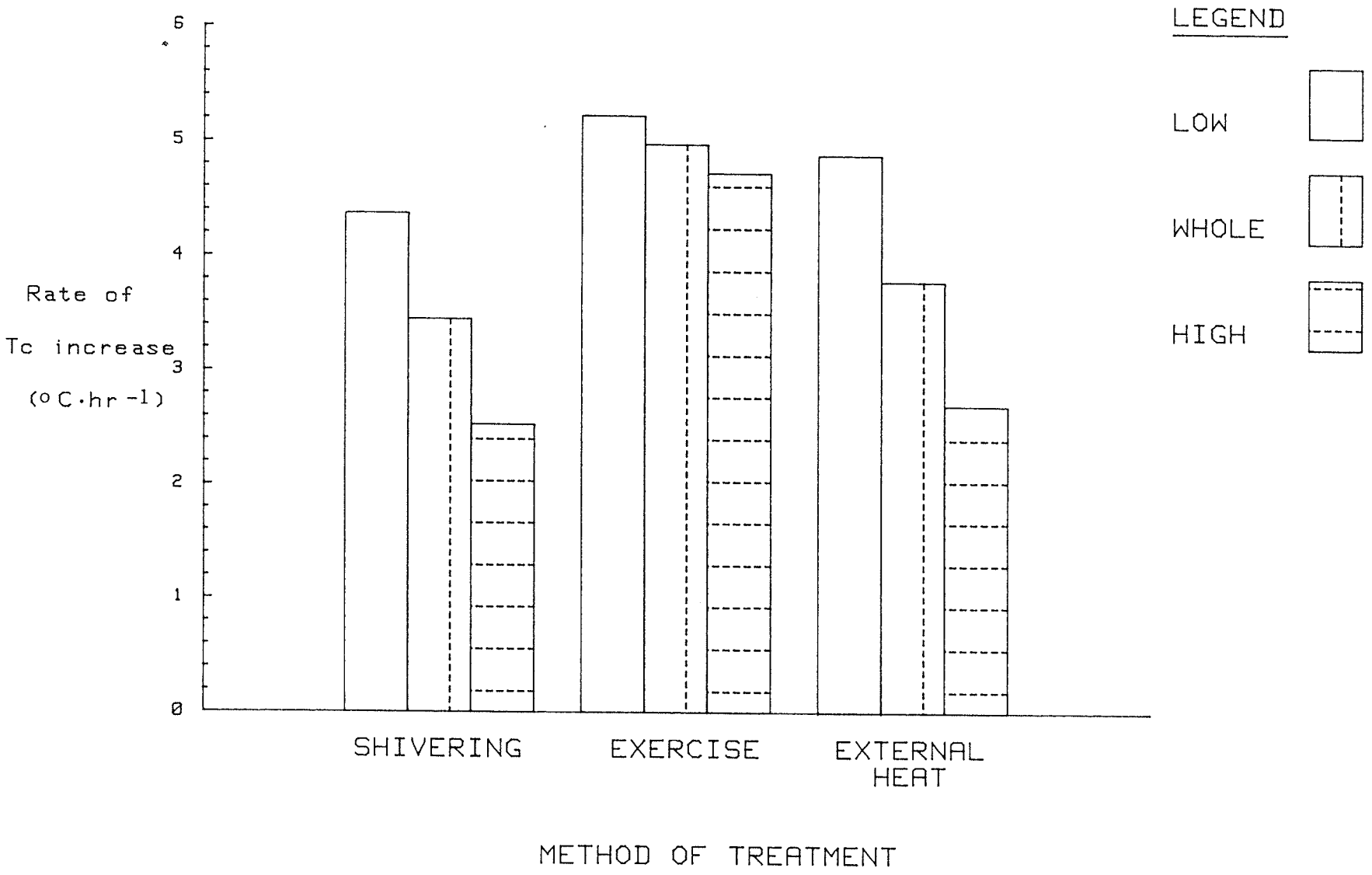
LEGEND
LOW
WHOLE
HIGH
Rate of
Tc increase
(°C.hr-1)
0
1
2
3
4
5
6
SHIVERING
EXERCISE
EXTERNAL HEAT
METHOD OF TREATMENT

FIGURE 6

for shivering (2.53 ± .81$^{0}C \cdot hr^{-1}$) or external heat (2.7 ± .81$^{0}C \cdot hr^{-1}$).

There was however, much less difference in the low fat subgroup. Rates of Tc increase were 5.23 ± .64, 4.89 ± .69 and 4.37 ± .68 $^{0}C \cdot hr^{-1}$ for exercise, external heat and shivering treatments respectively.

Within all groups, exercise produced a greater rate of Tc increase than either shivering or external heat, which were very similar. It was previously believed by this researcher that a great deal of heat was available from exercise using a major muscle mass. Results support this belief. As shivering may still occur during exercise, if the skin is kept cool, the additive effect would obviously produce a greater rewarming rate.

The rewarming rates for shivering thermogenesis were 2-4 times greater than previously reported (Pozos, 1986; Hayward et al., 1983; Collis et al, 1977). Conversation with these researchers revealed that subjects were not insulated from warm ambient air during shivering. In their studies it was observed that shivering decreased markedly and subjects rewarmed very slowly (approximately 1.5 $^{0}C \cdot hr^{-1}$). In those cases, warm laboratory air (22 ^{0}C) exerted a warming influence on the skin which initially was substantially cooler from immersion in cold water (7-10 ^{0}C). As the skin warmed up, the shivering stimulus was weakened and heat production,

and the rewarming rate decreased. In this experiment, subjects were placed in an insulated, vapour proof bag. It is true that the bag retained heat produced which would have been otherwise lost but the main advantage of this procedure was that warming of the skin by warm air was retarded. The cooler skin maintained the high shivering stimulus and a great deal more heat was produced for a longer period of time. This explains the surprisingly high rate of rewarming with this method.

External heat decreased shivering heat production by warming the skin and decreasing the shivering stimulus. However, it did not supply significantly more heat than was lost, from decreased shivering, resulting in similar rates of rewarming between these two treatments.

Upon removal from an acute exposure, a victim should have plenty of fuel for shivering and this method may be as effective, and possibly more practical, than application of external heat. Maximal results would occur only if the subject was removed from the cold insult, dried off, placed in dry clothing and an insulated vapour proof bag. This bag could be a sleeping bag placed inside two garbage bags, one pulled over the legs and one (with a hole cut for the face) pulled over the torso.

Accompanying the increased rate of rewarming during exercise are the larger afterdrop amount and longer afterdrop

period. If a victim was found at very low Tc of 30 - 32 °C, the resultant afterdrop of exercise (0.7 - 1.5 °C) may result in a new Tc at which shivering ability (heat production) and consciousness may be lost. In otherwise young, healthy adults rescued at higher core temperatures however, exercise may be quite beneficial as a larger initial afterdrop may not endanger survival.

Exercise during and after rescue, has been strongly contraindicated in the past (Bangs, 1979). The 'post rescue collapse' of many shipwrecked survivors has sometimes been attributed to exercise performed during rescue. It is noteworthy that there are many possible explanations for this phenomenon including the removal of the hydrostatic squeeze of water immersion. At this point, the precise cause for these deaths is uncertain and results from this study revealed no abnormal blood pressure or heart rate responses to exercise shortly after cold insult ended.

Practically, exercise could be used to treat cold water immersion hypothermia if a warm shelter were accessible or if air temperatures were warm. The victim should have wet clothes removed, be dried off, clothed in dry attire and allowed to perform a comfortable rate of some form of exercise involving a large amount of muscle mass. Suitable activities might be walking or, in confined quarters, stepping up and down on a step or chair.

Comparison of the difference in rate of rewarming between treatments across the three subgroups (Figure 6) revealed the same trend as results for afterdrop amount and length. While there is hardly any difference between treatments for the low fat subgroup, a great difference existed in the high fat subgroup. If results from these small subgroups (N=3) are representative of the general population, the practical ramifications of these findings are obvious. A thin victim could be rewarmed with any of these three treatments without much difference in rewarming rate. Choice of treatment for a victim with high fat skinfolds may be much more critical as the difference in rewarming rates are greatest in this group, exercise being the highest.

A final analysis of Figure 6 reveals that subgroup membership translates to large differences in rewarming rates for shivering and external heat while almost no difference exists in exercise. The same energy requirement exists for a certain work protocal by high or low fat subjects. Therefore the same amount of heat is produced for body rewarming. The slightly higher rate of rewarming for low fat subjects may be explained as they had less mass to rewarm. A given amount of energy would warm them faster than heavier high fat subjects.

Less mass to rewarm in low fat subjects also accounts for some of the increased rewarming rate over high fat counterparts during shivering and external heat. An added advan-

tage for low fat subjects in external heat may be that less insulation exists, allowing heat to penetrate via conduction more easily. Low fat subjects also have a decided advantage in shivering as they had greater muscle/fat mass ratios than the high fat subgroups. Therefore, a greater percentage of the body mass was able to produce heat for rewarming by shivering.

INDIVIDUAL TREATMENT CASE STUDIES

Introduction

Analysis of O_2 consumption ($\dot{V}O_2$) and mean skin temperature (MST) as they relate to Tc and activity level, provide many explanations for the efficacy of treatment procedures. Therefore, one such pictoral analysis is depicted for each treatment in Figures 7, 9 and 10. For a better understanding of the stimulus and mechanism of heat gain through the shivering thermogenesis method, an extra study is presented in Figure 8, in which the subject was not insulated from ambient room air during shivering thermogenesis. MST were calculated in only one of each of the treatment studies, in different subjects, therefore the same subject is not presented in each case. Time 0 represents removal time. A review of the heat production equation is necessary for a better comprehension of these figures. Nielsen (1969) proposed the following equation:

$$H = M - W \qquad (4)$$

where H = total heat production in $kcal \cdot hr^{-1}$, M = metabolic energy liberation in $kcal \cdot hr^{-1}$, and W = external work in $kcal \cdot hr^{-1}$.

$$M = \dot{V}O2 \ l \cdot min^{-1} \times 4.9 \ kcal \cdot l^{-1} \times 60 \ min \cdot hr^{-1} \qquad (6)$$

W was obtained by actual VO_2 measurement of normothermic subjects at the work rates they had experienced in the hypothermic conditions.

Shivering Thermogenesis (general)

Shivering is generally initiated by cooling of peripheral receptors in the skin. These receptors may stimulate shivering at a certain threshold in various conditions. Spur et al (1957) demonstrated commencement of shivering in subjects with the same MST in each of two cold exposures to 10 ^{o}C and -3^{o}C air.

Buguet et al (1976) demonstrated that shivering may be stimulated only by central receptors in the hypothalamous. Subjects in sleeping bags, with only facial areas cooled, shivered even though MST remained at 30 - 32 ^{o}C. They felt that a countercurrent exchange between the jugular veins and carotid arteries transferred a cold stimulus to the hypothalamus.

The stimulation to shiver may well be a combination of these two mechanisms as will be shown in the 'exercise metabolism' case study. Cort and McCance (1953) demonstrated peripheral stimulus for shivering by supplying cold air to the trachea in pigs. They found however that when cold air was later replaced by warm air (40 ^{o}C) shivering continued until internal temperatures had increased. It was postulat-

ed that the stimulus of cooled venous return in the hypothalamus maintained the shivering stimulus even though peripheral receptors had been warmed.

Shivering Thermogenesis (insulated skin)

Values for Tc, MST, metabolic energy liberation (M), basal metabolic rate (BMR), and heat production (Hsh) are plotted against treatment time for subject PH during the shivering thermogenesis protocol (Figure 7). Although BMR decreases minimally with lowered Tc, no correction was made, as only a small error was introduced. Heat production for shivering is the difference calculated between metabolic energy liberation and basal metabolic rate.

In this case, the afterdrop was minimal and reversed shortly after the two minute preparation period. M and Hsh decreased during this period as difference in warm air and cold water temperatures was drastic (15 ^{o}C). Once subject PH was insulated in the bag, M and Hsh increased while MST increased gradually. After 20 minutes, as MST approached normal temperatures, the shivering response decreased as indicated by lowered M and Hsh.

Horvath et al (1956) reported that generalized shivering occurred at MST ranging from 24 - 27 ^{o}C. The stimulus became greater as MST decreased. These results show that the inverse trend is also true as M decreased and Hsh reached near negligible levels as MST rose to approximately 29 ^{o}C.

Figure 7: Tc, MST, metabolic energy liberation (M), basal metabolic rate (BMR) and heat production for shivering (Hsh) vs treatment time for subject PH during shivering thermogenesis protocol (with skin insulation).

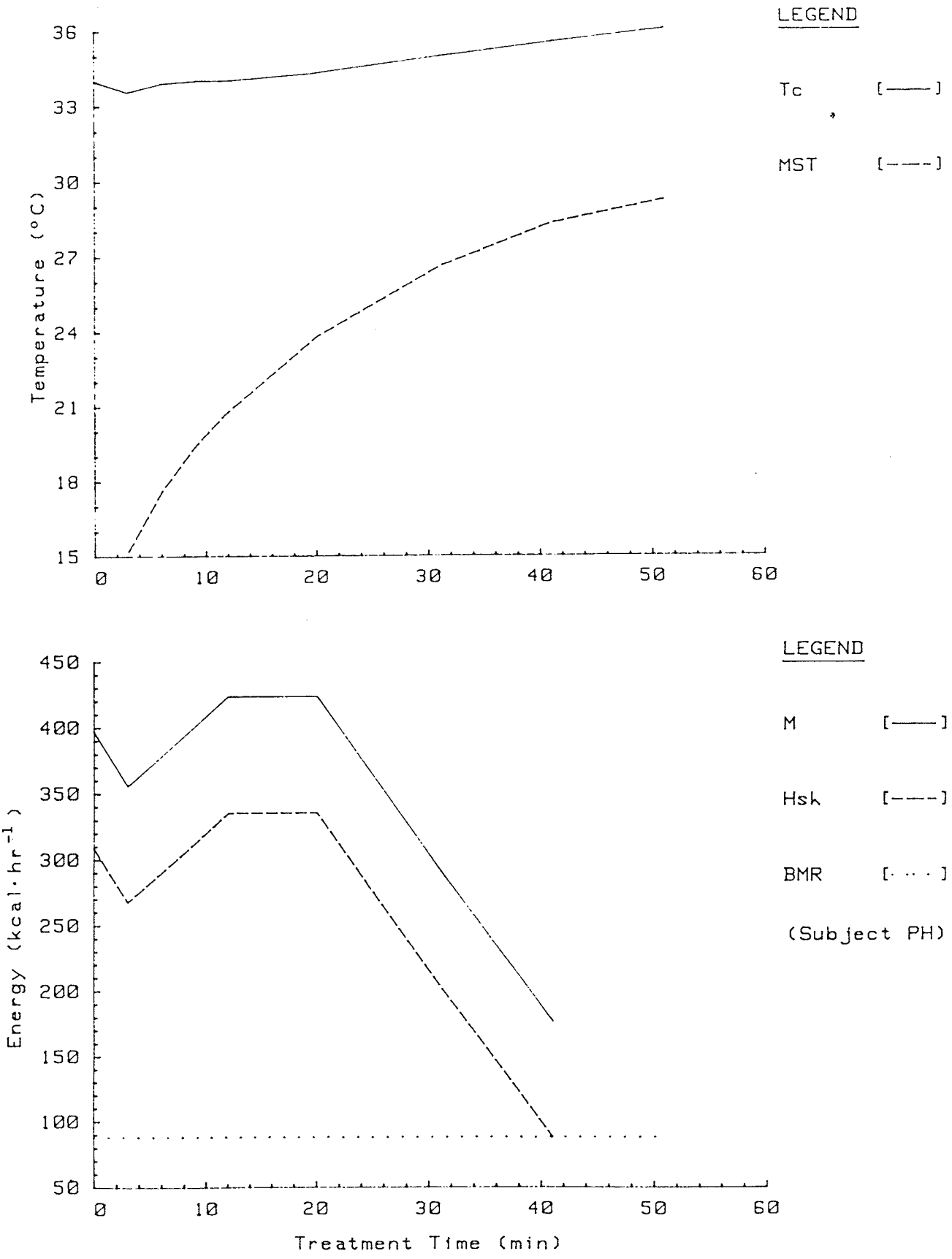
LEGEND
Tc [——]
MST [– – –]
Temperature (°C)
36
33
30
27
24
21
18
15
0
10
20
30
40
50
60
LEGEND
M [——]
Hsk [– – –]
BMR [· · ·]
(Subject PH)
Energy (kcal·hr⁻¹)
450
400
350
300
250
200
150
100
50
0
10
20
30
40
50
60
Treatment Time (min)

FIGURE 7

Shivering Thermogenesis (no skin insulation)

Figure 8 summarizes Tc, MST, metabolic energy liberation, basal metabolic rate and heat production as a function of treatment time for subject RS during the shivering thermogenesis protocol. This protocol did not include insulating the skin from the warm ambient air temperature.

This extra case study was performed in an attempt to explain why other researchers (Pozos, 1986, Collis et al., 1977) reported rates of rewarming for shivering (1.5 $^{0}C \cdot hr^{-1}$) so much lower than calculated in this experiment (3.45 $^{0}C \cdot hr^{-1}$). The only procedural difference was that subjects were insulated from room air in this experiment. Subject RS performed a fourth trial using shivering thermogenesis without skin insulation. MST in this case increased at a very fast rate compared to Tc rate of increase and leveled off while Tc was still increasing. MST in subject PH (Figure 6) was still rising when target Tc was reached. This leveling off of MST, at approximately 30 ^{0}C coincides with greatly reduced M and near negligible Hsh. It would seem that peripheral receptors were no longer stimulated and shivering was no longer stimulated. It is believed that this lack of insulation explains the drastic difference betweed rates of Tc increase reported in this and other studies.

In this case however, subject RS rewarmed at the same rate (5.1 $^{o}C \cdot hr^{-1}$) as in the noninsulated shivering treatments. This high rate continued until termination in the skin insulation protocol. In Figure 7 however, the rate of Tc increase droped from 5.1 to 1.65 $^{o}C \cdot hr^{-1}$ when MST leveled off.

Rates of recovery were consistantly high in this individual. He had the lowest sum of skinfolds (29 mm) and subjectively, the most muscle mass. It is significant that his maximal shivering $\dot{V}O_2$ was 50% of $\dot{V}O_2$ max on the treadmill. Other subjects only reached shivering $\dot{V}O_2$ levels ranging from 35.2 - 38.7% of their respective $\dot{V}O_2$ max. This subjects ability to commit such a large amount of energy to rewarming a light body containing minimal fat, may explain this abnormal rate of increase.

Figure 8: Tc, MST, metabolic energy liberation (M), basal metabolic rate (BMR), and heat production for shivering (Hsh) vs time for subject RS during shivering thermogenesis protocol (no skin insulation).

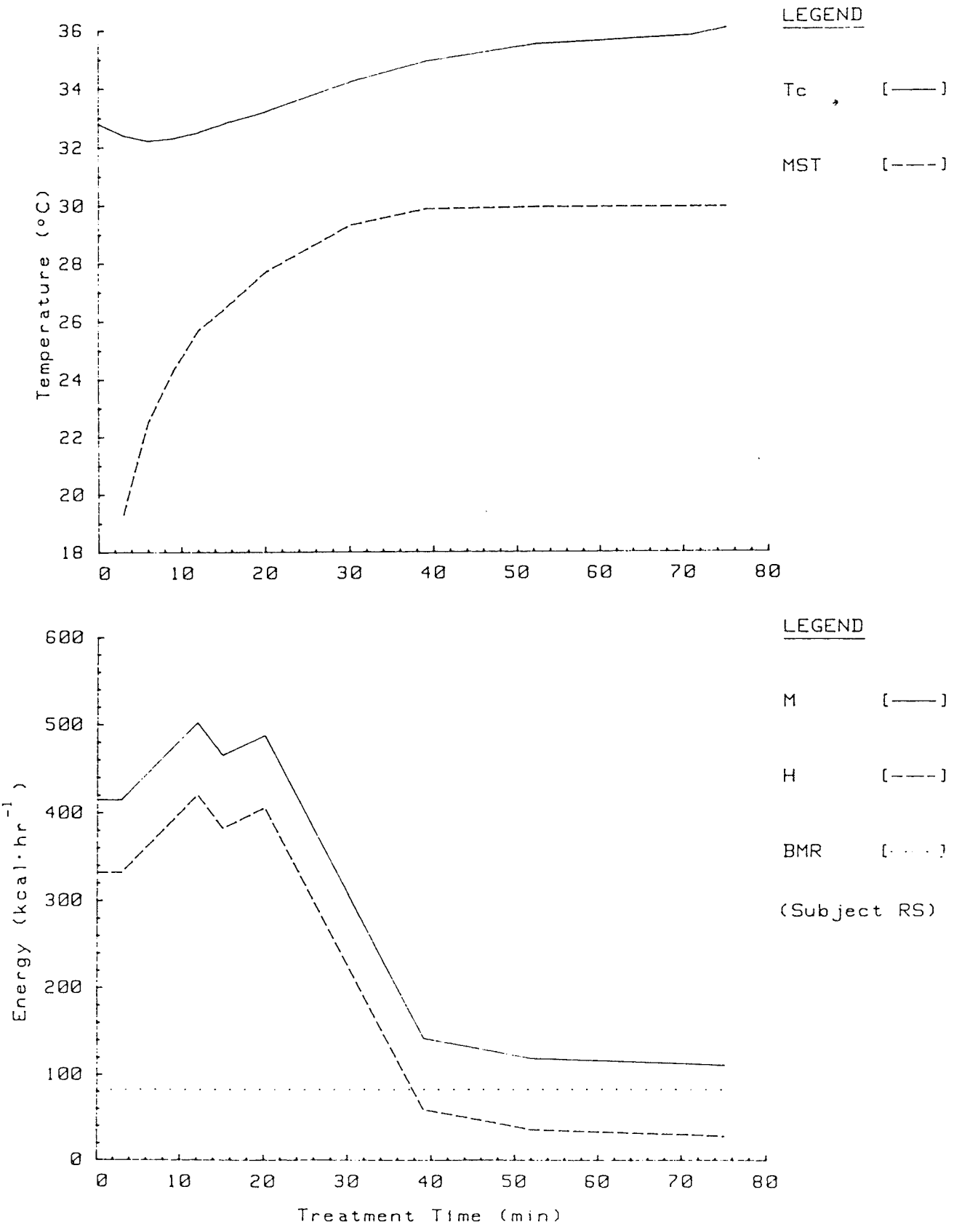
LEGEND
Tc [——]
MST [– – –]
Temperature (°C)
36
34
32
30
28
26
24
22
20
18
0
10
20
30
40
50
60
70
80
LEGEND
M [——]
H [– – –]
BMR [· · ·]
(Subject RS)
Energy (kcal·hr⁻¹)
600
500
400
300
200
100
0
Treatment Time (min)

FIGURE 8

Exercise Metabolism

Figure 9 presents Tc, MST, treadmill speed, metabolic energy liberation, external work and heat production (Hex) against treatment time for subject GG during exercise.

BMR, not seperately indicated in this graph, is included in the external work $\dot{V}O_2$. $\dot{V}O_2$ was directly measured by the Sensormedics MMC during the same work protocol in a normothermic state.

This figure is best analysed in two phases. In the first 25 minutes, the minimum treadmill speed is unaltered. As indicated by W, the energy requirements for this load are also unaltered, however M and Hex rise throughout the first nine minutes before decreasing. The peak in Hex concurs exactly with the lowest point of Tc. As Hex actually indicates energy production from shivering it is clear that, although MST rises from the onset of treatment, decreased Tc is actually the stimulus for shivering. M and Hex both decrease when Tc starts to increase. From this data the contribution of two shivering mechanisms may by understood. At higher Tc (35 - 37 ^{o}C) the main stimulus for shivering may be through peripheral receptors. As Tc decreases, Tc itself may control shivering stimulation through central receptors.

These results mirror those of Nadel et al (1973) who had subjects pedal a cycle ergometer at a constant workload after removal from cold water. They found the highest levels of shivering to occur at the lowest internal temperatures.

Figure 9: Tc, MST, treadmill speed, metabolic energy liberation (M) extermal work (W) and heat production during exercise (Hex) vs treatment time for subject GG during the exercise protocol.

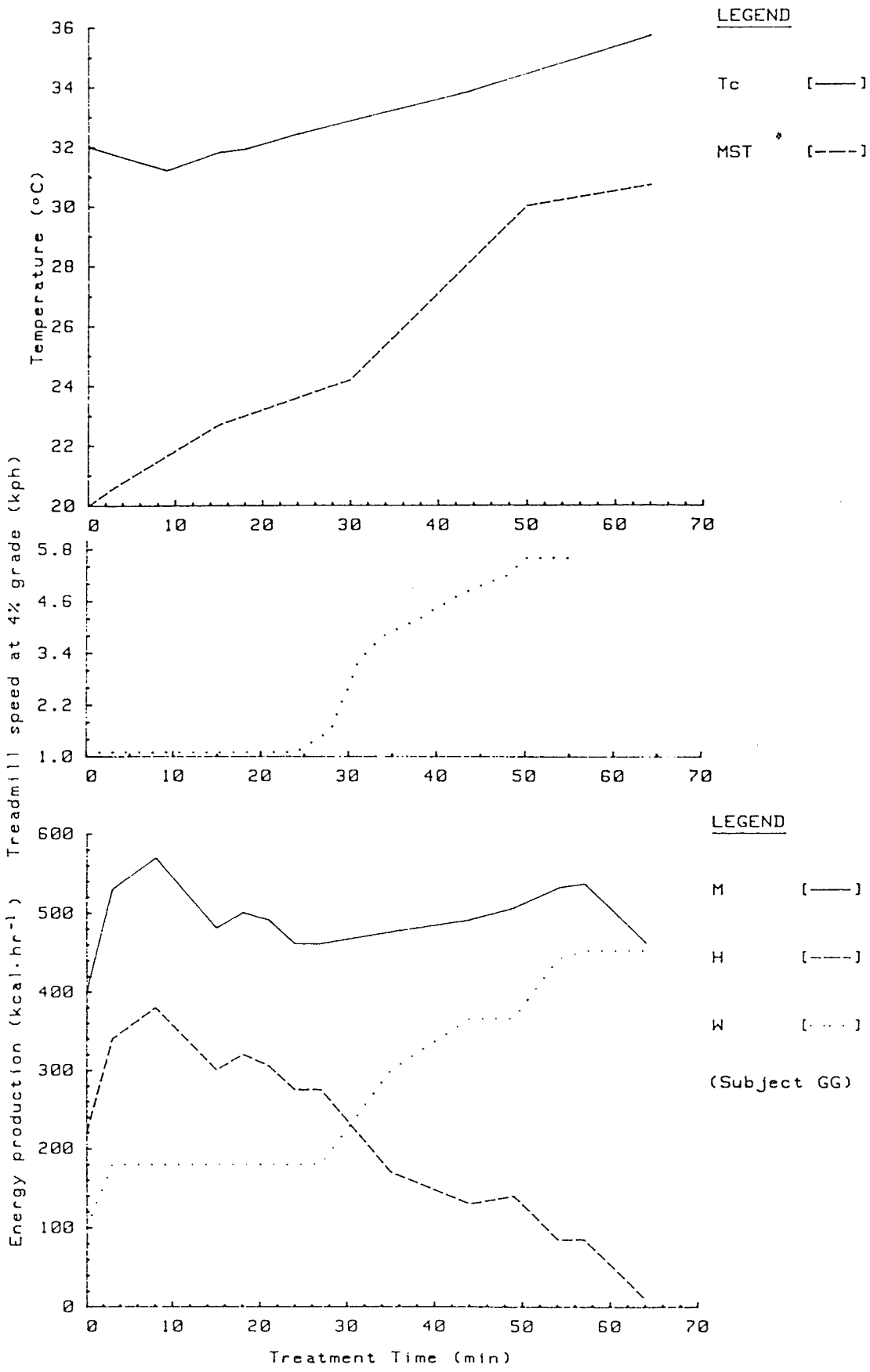
LEGEND
Tc [——]
MST [– – –]
Temperature (°C)
36
34
32
30
28
26
24
22
20
0 10 20 30 40 50 60 70
Treadmill speed at 4% grade (kph)
5.8
4.6
3.4
2.2
1.0
0 10 20 30 40 50 60 70
LEGEND
M [——]
H [– – –]
W [· · ·]
(Subject GG)
Energy production (kcal.hr⁻¹)
600
500
400
300
200
100
0
0 10 20 30 40 50 60 70
Treatment Time (min)

FIGURE 9

They also reported that metabolic energy liberation (M) was gradually reduced to the demands of the exercise at normal Tc. A review of the second phase (post - 25 minutes) shows a concurrance with Nadel's results. As Tc increased M and W converged toward a point where Hex was negligible. This occured when Tc was normothermic and MST was near its regular value.

Also note that MST rose at a higher rate during the period when treadmill speed was increasing. This rate decreased again when the maximal workload was reached. The surge in MST increase may have been due to corresponding increases in blood supply for increased contraction rates in the muscles.

Application of External Heat

Tc, MST and energy liberation as well as heat supplied from the STK Heatpac are presented against treatment time for subject JH in Figure 10. Manufacturer specifications state that the STK Heatpac produces 250 watts. This value (converted to 215 $kcal \cdot hr^{-1}$) was used in Figure 8 to indicate energy supplied from the heat source (HP). Heat production (Heh) in this case is again calculated as the difference between metabolic energy liberation (M) and basal metabolic rate (BMR).

During the initial nine minutes M and Heh decreased along with Tc as MST quickly increased. This decrease in M and

Heh is probably due to the great rewarming effect on peripheral receptors on the chest. M and Heh rise for a period of five minutes before a steady decrease is then experienced.

Figure 10: Tc, MST, M, BMR, heat production during application of external heat (Heh), and energy supplied from heat source (HP) vs treatment time for subject JH during external heat protocol.

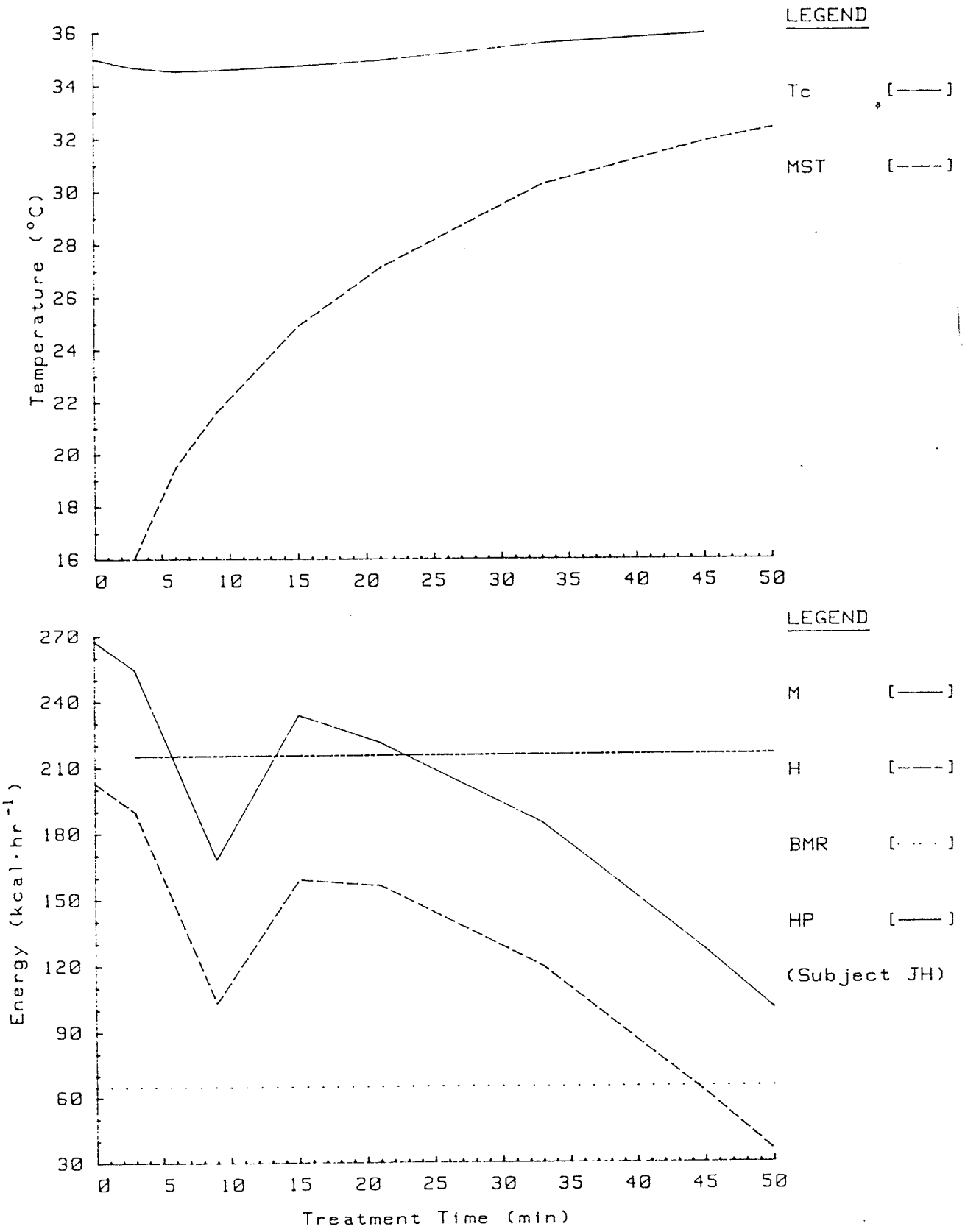
LEGEND
Tc [——]
MST [– – –]
Temperature (°C)
36
34
32
30
28
26
24
22
20
18
16
0 5 10 15 20 25 30 35 40 45 50
LEGEND
M [——]
H [– – –]
BMR [· · · ·]
HP [——]
(Subject JH)
Energy (kcal·hr⁻¹)
270
240
210
180
150
120
90
60
30
0 5 10 15 20 25 30 35 40 45 50
Treatment Time (min)

FIGURE 10

Subject JH had the highest sum of skinfolds (80.8 mm) and experienced the longest afterdrop period (23 minutes) for this method. It is possible that the continuing low Tc finally overrode the warm peripheral receptors and stimulated an increase in shivering in order to reverse the afterdrop phenomenon.

It is clear that the main effect of the heating pad is to decrease M therefore Heh. These results support those of Collis et al (1977) who found that heating pads warmed enough skin to inhibit shivering. They did not however deliver enough energy to alter the afterdrop. This has been shown in these results as all of the heat production, indicated by STK Heatpac manufacturer specifications, is not delivered to or utilized by the body for rewarming.

Kaughman (1983) reported that surface rewarming, unless in a hot bath (which would supply more than enough calories for significant rewarming), should be avoided. Results of this study may support this statement.

INDIVIDUAL RATES OF TC INCREASE

Introduction

Several parameters have been analysed (afterdrop amount and length, as well as rewarming rates) for various treatment methods. The conclusion regarding the safest and most effective treatment is still not clear. The best way to answer this question is to combine knowledge of previous analyses with individual summaries, including all three parameters together, comparing the three treatments.

Results

Figures 11-16 are graphical presentations of average cooling rates as well as Tc behavior during the 3 treatment procedures for each subject. In order to compare each treatment, the values were normalized so that the time of removal from the cold insult was set at $0^{o}C$ and 0 min. All other values were then presented relative to that value. These figures give an indication of another parameter, time of recovery to target Tc.

Figure 11: ΔTc vs time for subject JH during 3 treatment procedures.

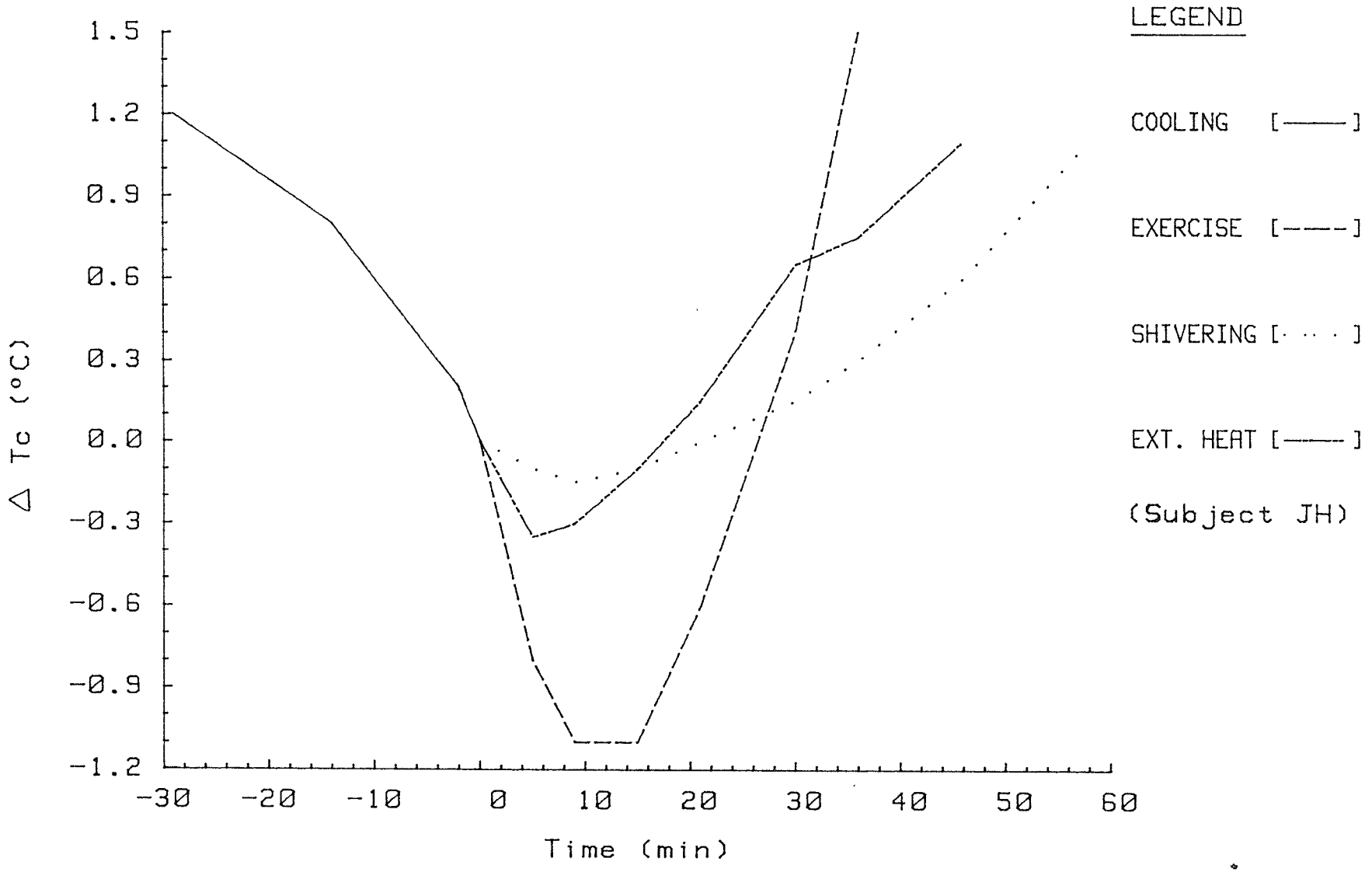
LEGEND
COOLING [———]
EXERCISE [– – –]
SHIVERING [· · · ·]
EXT. HEAT [—— —]
(Subject JH)
1.5
1.2
0.9
0.6
0.3
0.0
-0.3
-0.6
-0.9
-1.2
△ Tc (°C)
-30
-20
-10
0
10
20
30
40
50
60
Time (min)

FIGURE 11

Figure 12: ΔTc vs time for subject RS during 3 treatment procedures.

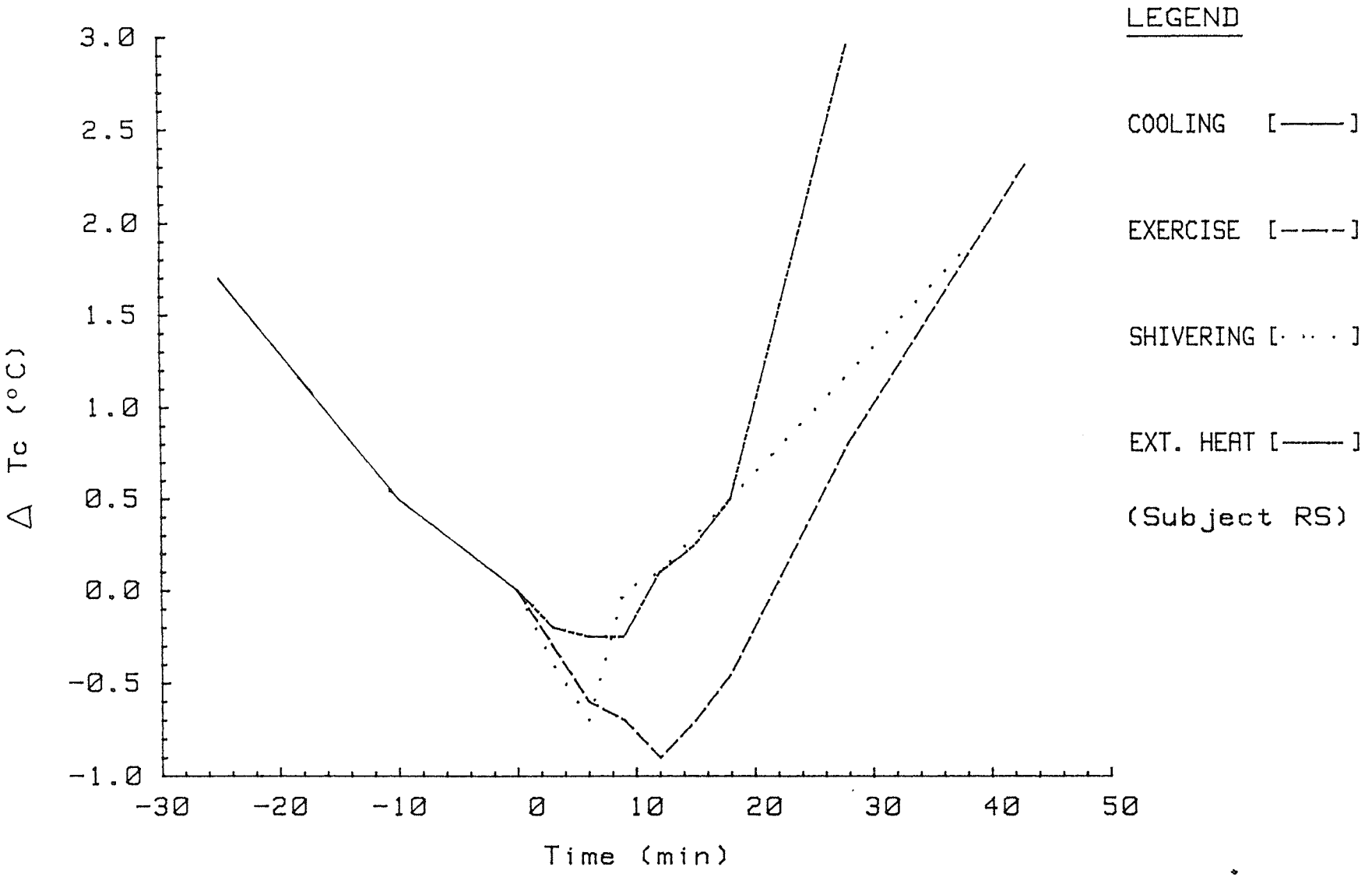
LEGEND
COOLING [———]
EXERCISE [– – –]
SHIVERING [· · · ·]
EXT. HEAT [———]
(Subject RS)
3.0
2.5
2.0
1.5
1.0
0.5
0.0
-0.5
-1.0
Δ Tc (°C)
-30
-20
-10
0
10
20
30
40
50
Time (min)

FIGURE 12

Figure 13: ΔTc vs time for subject GG during 3 treatment procedures.

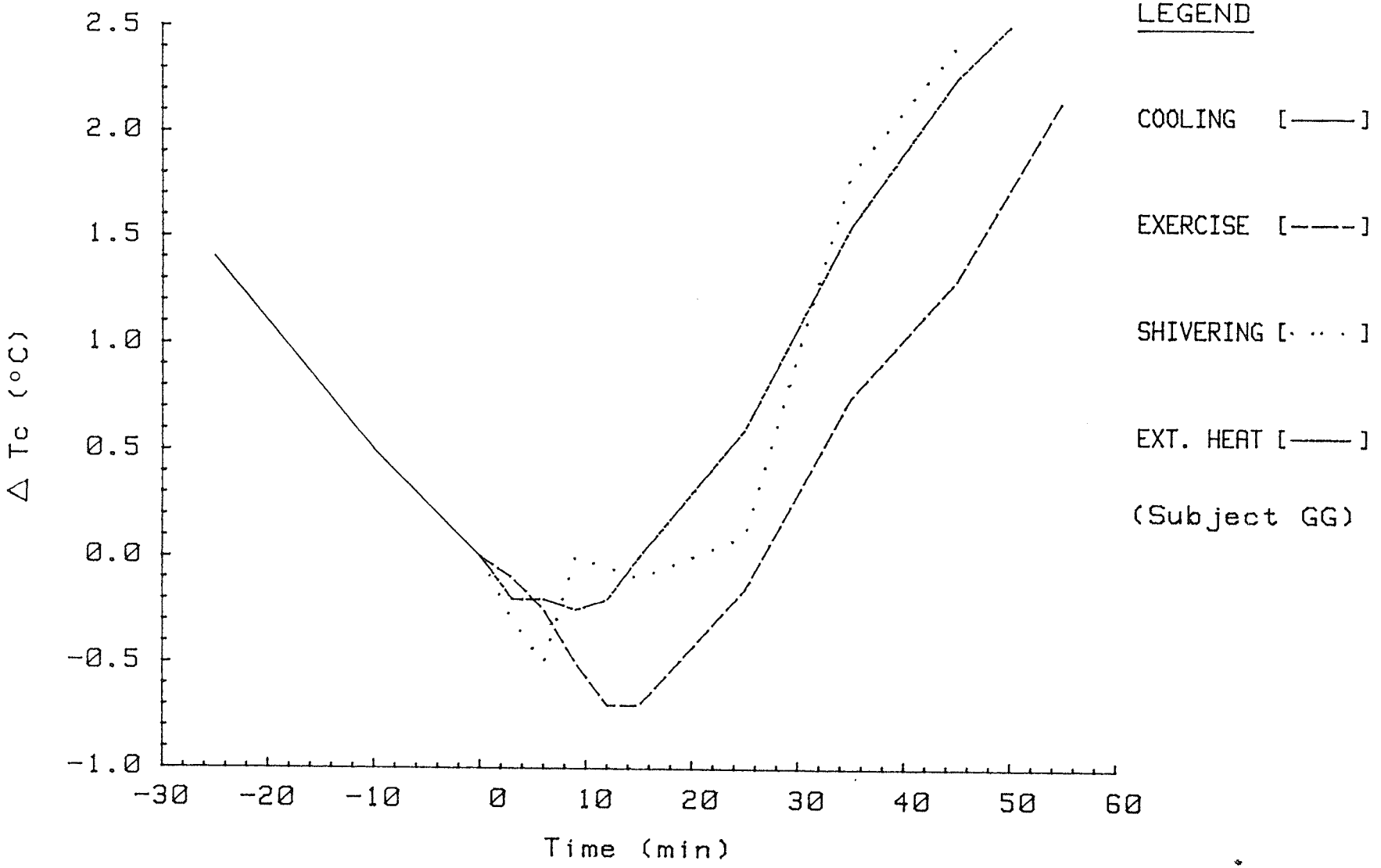
LEGEND
COOLING [——]
EXERCISE [– – –]
SHIVERING [· · · ·]
EXT. HEAT [——]
(Subject GG)
Δ Tc (°C)
2.5
2.0
1.5
1.0
0.5
0.0
-0.5
-1.0
-30
-20
-10
0
10
20
30
40
50
60
Time (min)

FIGURE 13

Figure 14: ΔTc vs time for subject PH during 3 treatment procedures.

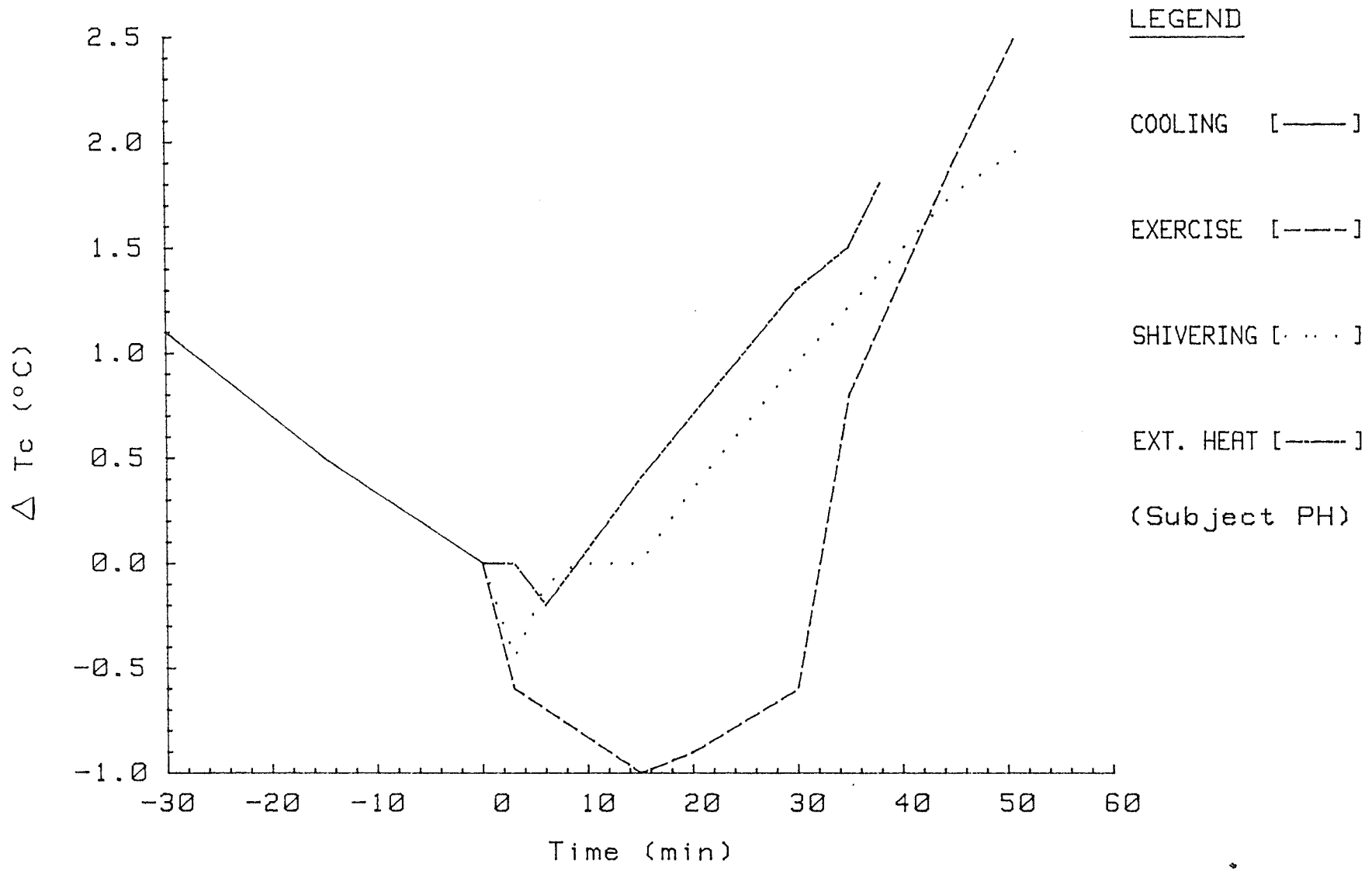
LEGEND
COOLING [———]
EXERCISE [– – –]
SHIVERING [· · ·]
EXT. HEAT [———]
(Subject PH)
2.5
2.0
1.5
1.0
0.5
0.0
-0.5
-1.0
Δ Tc (°C)
-30
-20
-10
0
10
20
30
40
50
60
Time (min)

FIGURE 14

Figure 15: ΔTc vs time for subject TS during 3 treatment procedures.

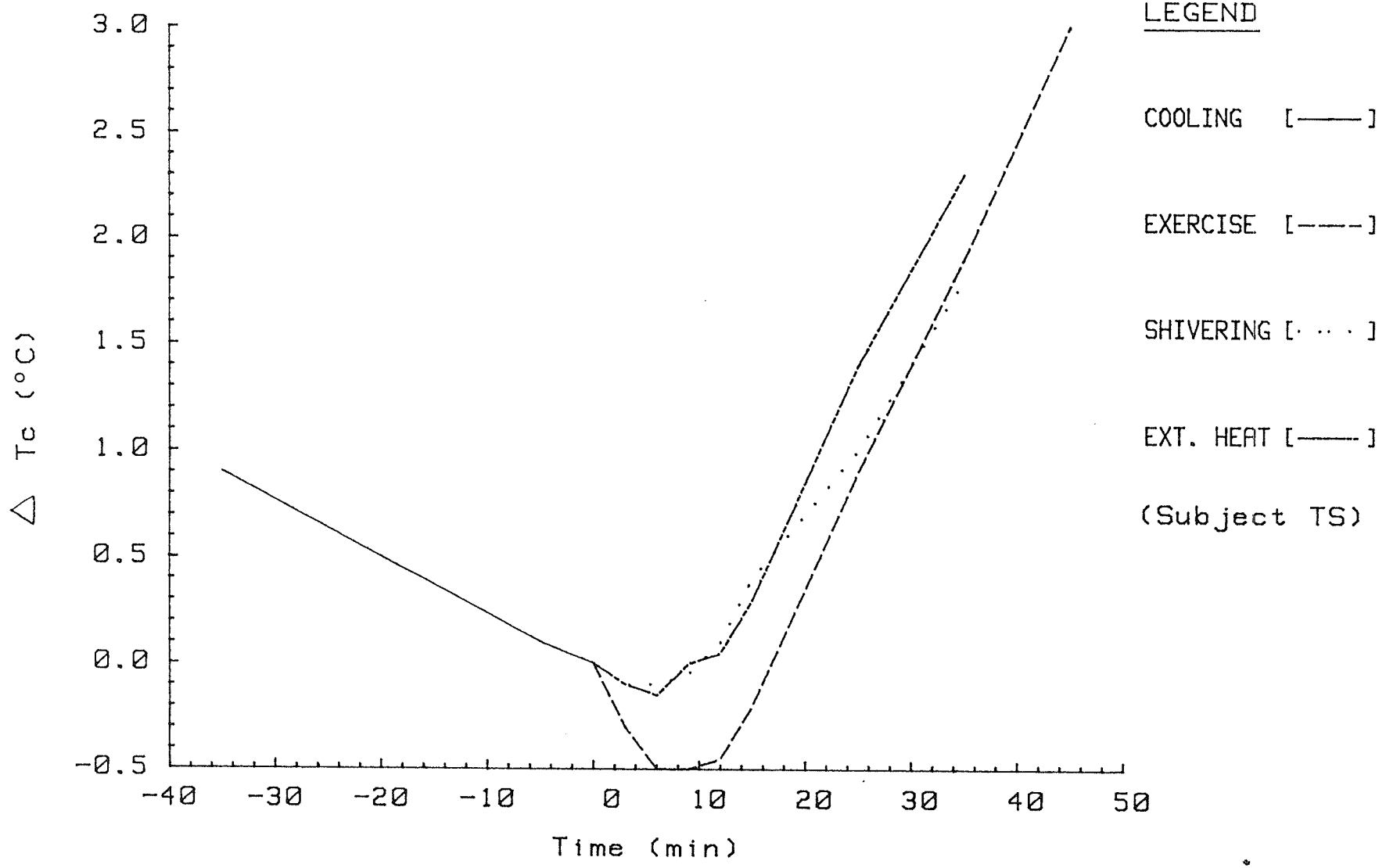
LEGEND
COOLING [———]
EXERCISE [– – –]
SHIVERING [· · · ·]
EXT. HEAT [——·——]
(Subject TS)
△ Tc (°C)
3.0
2.5
2.0
1.5
1.0
0.5
0.0
-0.5
-40
-30
-20
-10
0
10
20
30
40
50
Time (min)

FIGURE 15

Figure 16: ΔT_c vs time for subject BT during 3 treatment procedures.

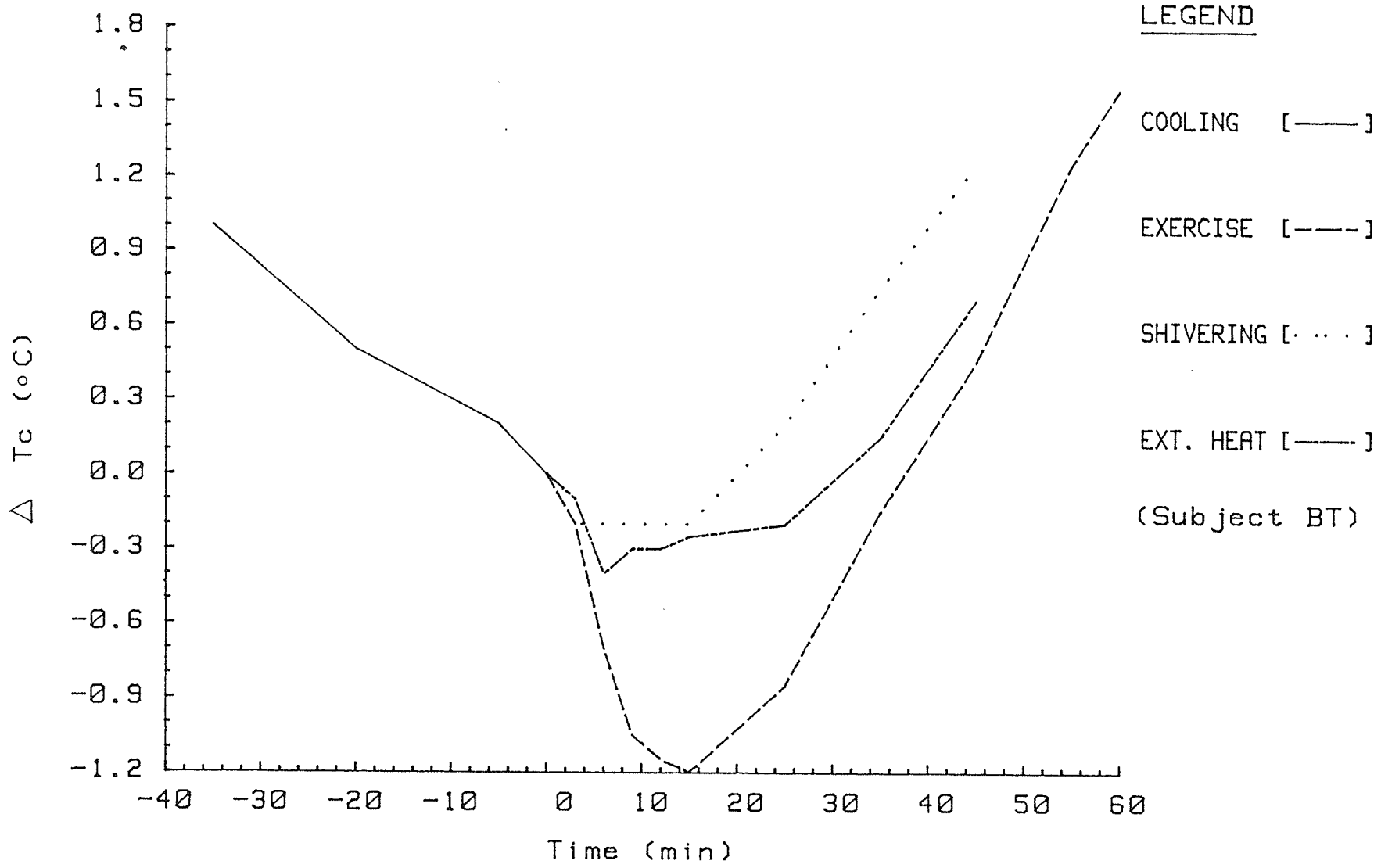
LEGEND
COOLING [———]
EXERCISE [– – –]
SHIVERING [· · · ·]
EXT. HEAT [——·——]
(Subject BT)
△ Tc (°C)
1.8
1.5
1.2
0.9
0.6
0.3
0.0
-0.3
-0.6
-0.9
-1.2
-40
-30
-20
-10
0
10
20
30
40
50
60
Time (min)

FIGURE 16

Figure 11 illustrates what may be expected from previous results with higher rates of Tc increase during exercise. Even though the afterdrop was precipitous in exercise the subsequent rate of increase for subject JH was much quicker than the other treatments and a 1.5 ^{o}C increase from removal Tc was accomplished much quicker (20 and 30 minutes) than with external heat and shivering respectively.

Figure 12 shows results for subject RS who had the lowest sum of skinfolds. After the two minute preparation phase afterdrop during both shivering and exercise continued. It was halted however in the external heat treatment. This higher nadir compared with a higher rewarming rate resulted in recovery, to 2 ^{o}C above removal Tc, 18 minutes before exercise or shivering. It is possible that the ability of the external heat to stop the afterdrop was due to the subjects very low sum of skinfolds (29 mm).

In Figure 13, subject GG experienced an extremely long afterdrop period during exercise compared to the other two treatments. Although rates of rewarming were similar for all treatments, a recovery of 2 ^{o}C from removal Tc took approximately 15 minutes longer than shivering or external heat.

Figures 14-16 indicate similar recovery times to target Tc, as the three rewarming rates are relatively parallel in each subject. Differences in recovery times for each subject are no more than ten minutes.

From these individual graphs it is clear that individual variability makes the choice of the most effective treatment difficult at best. The only discriminatory factor would be afterdrop amount and length during exercise. If afterdrop was to be kept at the absolute minimum, exercise would be contraindicated. Subjective analysis would indicate exercise may be one of the better treatments if victims were recovered at Tc > 33 - 34 ^{0}C when the added afterdrop may not be hazardous. Four of six subjects reported that exercise as a treatment provided the most positive experience during rewarming while only one disliked it the most. Positive reactions were based on the fact that subjects were able to do something for themselves. This activity helped take their mind off the discomfort of their condition. Generally, subjects felt very uncomfortable during shivering and did not feel the heat of external heat until well into that treatment.

The focus of this thesis has been to evaluate the effectiveness of three field treatments for hypothermia but not necessarily to establish the best of the three. Significant findings have been made regarding each treatment.

Exercise was found not only possible but beneficial at Tc much lower (31.2 ^{0}C) than has previously been believed. Associated with this method is a high rate of Tc increase but a larger and longer afterdrop.

The STK Heatpac may not be worth the expense as a hypothermic treatment aid as it provides very little advantage over shivering thermogenesis. It is only fair to mention that this device was not designed for treatment but as a preventative measure for evacuating injured normothermic victims. In victims with Tc low enough to result in termination of shivering, the Heatpac would obviously be beneficial as the body would not be producing heat on its own.

Results for shivering are significant also. One standard procedure used in field settings has been to reproduce our protocol by drying and covering victims. Yet outdoor enthusiasts have been looking from more effective methods because they have equated success of this field practice with the low rewarming rates of approximately 1.5 $^{0}C \cdot hr^{-1}$ reported in laboratory experiments. What has been unclear was that laboratory protocols in the past have exposed subjects to warm air thus reducing the shivering stimulus and therefore effectiveness. It is of great importance that the laboratory procedure for shivering in this experiment if used in a field setting will produce a rewarming rate of not 1.5 $^{0}C \cdot hr^{-1}$ but probably 3.5 $^{0}C \cdot hr^{-1}$. This treatment then, is very effective as it is safest and simplest, and the search for a better method need not be so intense.

It is very interesting to speculate on the effectiveness of a treatment protocol combining the safe small afterdrop of shivering thermogenesis and the significantly faster re-

warming rate of exercise. A future study should institute exercise once the afterdrop period during shivering is over. If exercise did not prolong or even reintroduce afterdrop, but result in an immediate rate of rewarming, this combination should provide a very quick recovery time.

SUMMARY AND CONCLUSIONS

Responses of 6 subjects to three rewarming methods for immersion hypothermia were analysed and compared. The three field applicable treatments were exercise, shivering thermogenesis and application of external heat via a STK Heatpac. Subjects were cooled in water (8.0 ± 0.5°C) until either their core temperature (Tc) decreased to 33°C, they decided to exit, or the attending physician advised them to do so. Esophageal temperature was used to indicate Tc, and mean skin temperature (MST) was derived from measurements taken from the arm, chest, thigh and calf. ECG, blood pressure and oxygen consumption were monitored at various times.

Within the limitations of the experimental protocol, the following conclusions were made:

1. Exercise resulted in approximately 3 times the amount of afterdrop compared to the other two treatments.
2. The duration of the afterdrop period was longer during exercise than in the other two treatments.
3. The rate of Tc increase was $1^{\circ}C \cdot hr^{-1}$ faster for exercise than for shivering thermogenesis and external heat.
4. Choice of the most efficient rewarming method, based on time of recovery from cold insult removal to a

target Tc, was not possible due to individual variability.

5. Exercise is possible, and may be beneficial for the rewarming process in otherwise healthy young adults, at a much lower Tc than has previously been believed possible.
6. Application of external heat via the STK Heatpac did not supply enough heat to result in a faster rate of Tc increase than shivering thermogenesis. This was likely because the Heatpac warmed peripheral receptors reducing the shivering stimulus.
7. Shivering thermogenesis may be the safest and easiest protocol as the afterdrop is minimal.
8. Shivering produces a much higher rate of Tc increase than previously reported if the skin is insulated from rewarming. As skin stays cool longer, the shivering response remains high.
9. Shivering is probably controlled by peripheral and central receptors. Low MST may stimulate shivering at higher Tc while a lower Tc itself may become the major shivering stimulus.

Based on these results, the following recommendations can be made for further rewarming research.

1. This study should be repeated with a larger subject pool which again would form two distinct groups according to sum of skinfolds. Choice of the most ef-

ficient treatment determined by time to recovery Tc may indeed be possible. Also, the effects of fat mass on the afterdrop and Tc rewarming phase may be substantiated.

2. An investigation should be carried out to assess the effectiveness of combining shivering thermogenesis and exercise. If exercise commenced once the afterdrop period of shivering was concluded, the recovery time may decrease substantially.
3. A study should be conducted to see if rates of Tc increase for shivering could be induced to even higher levels by applying a slight cold stimulus to the skin. This may stimulate an even higher more steady rate of shivering than seen in this study.

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Appendix A

INFORMED CONSENT FORM

EFFECTIVENESS OF VARIOUS FIELD TREATMENTS FOR INDUCED HYPOTHERMIA

THESIS EXPERIMENT

INFORMED CONSENT

Introductory Information

The following is a detailed description of the procedures to be undergone by subjects in a masters thesis study entitled "Effectiveness of various field treatments for induced mild hypothermia". Your time commitment as a subject will include this screening session, a medical examination by a physician and four experimental treatment sessions. Please feel free to ask any questions regarding the procedures at any time.

The screening and medical examination sessions are necessary for two reasons: to obtain anthropometric and fitness data for each subject; to determine if each subject is free of medical contraindications which may make the experimental procedures unsafe for him/her.

The experiment itself will compare the effectiveness of four treatment procedures for hypothermia. A different procedure will be applied to you after each of four sessions in which mild hypothermia will be induced by sitting in water cooled from an original temperature of 20 degrees C to below 10 degrees C. The drop in temperature will be gradual thereby saving you the subject the trauma of getting into cold water.

Screening Session

After reporting to the exercise physiology laboratory at the Max Bell Research Center you will be given a Par Q Questionaire verbally in order to screen for any obvious contraindications to participation in the experiment. You will then be asked to read and sign this informed consent. Remember that even after signing you are not committed to completing the entire experiment as you are free to withdraw from this experiment at any time.

Age and anthropometric measures will then be taken. Height and weight will be recorded and percent body fat will be estimated using tha sum of skinfolds method. Skinfolds will be measured using fat calipers at sites on the arm, back and hip.

You will then be asked to perform a PWC 170 test on a cycle ergometer. This test will require you to pedal the cycle ergometer at 50 rpm for six minutes while the testor adjusts the workload periodically. You will not become exhausted during this test. Data will be used to estimate your physical fitness level. Results will be compared to Canadian norms for your age and sex to indicate which level of fitness you are at within the population. You will be required to attain a score of at least average to be elegible for participation in the experiment.

Medical Examination

You will then be asked to see Dr. G. Bristow at the Emergency Department of the Health Sciences Center for a medical examination. A medical history will be taken and your general medical suitability for this experiment will be determined. A 12 lead electrocardiogram (ECG) will be performed on you and blood pressure taken to determine if any cardiovascular conditions may contraindicate continuous cold water immersion. If you have not had a chest x-ray in the past year, one may be taken at this time to screen for possible enlargement of the heart or any other cardiac problems. This checkup will not take long and will not result in any cost to you. Please have your Manitoba Medical number with you when you report to the emergency department.

Experimental Procedures

Upon receiving a physicians consent to participate, you will be asked to report again to the laboratory at the Max Bell Research Center. Here you will prepare for the water immersion portion of the experiment. This procedure is identical for all visits.

You will be asked to change into a bathing suit. Then skin temperature thermistors will be taped to your body on the calf, thigh, chest and arm. Also, 3 ECG electrodes will be applied to the chest area and forehead.

In order to measure your body core temperature an esophogeal thermister will record temperature in the esophagus at the level of the heart. A physician, who will be in attendance throughout each experiment, will apply a 10% xylocaine spray in one of your nostrils in order to anesthetize the area. This will decrease any discomfort caused by inserting the probe. The physician will also coat the end of the probe (approximately 1/8 inch in diameter) with a 2% xylocaine gel. He will then insert the probe through the nostril and pass it down the exophagus until it reaches the level of the heart. You will facilitate this action with swallowing motions as you sip a glass of water through a straw. It should be noted that this common procedure is repeated on patients daily in emergency wards, no anesthesia is used at all, and patients are not harmed in any way. You may encounter some discomfort at the time of insertion but this should subside quickly. You may also feel the probe in the back of the throat and it may take a few minutes to adjust to the sensation. You may also feel a slight discomfort in the nasal passage for a few days after the experiment but be assured no damage will be done.

You will then be asked to sit motionless on a chair for a period of 10 minutes during which baseline values for various experimental parameters will be established. Following this you will be asked to enter the tank of water, at 20 degrees C, and remain submerged at approximately neck level.

The temperature of the water will then be lowered gradually to below 10 degrees C. At this point your blood pressure will be determined every 15 minutes and ECG continuously monitored by a physician. You will remain in the water until either you wish to terminate your immersion, the physician advises you to exit for safety reasons, or your body core temperature reaches 33 degrees C. It should take approximately 1-2 hours of immersion to reach this core temperature. During this immersion you may be asked to perform various mental or physical tasks which will help to determine effects of lowered body core temperature.

At this point you will be helped out of the tank and one of four treatment procedures will commence. The order will be randomly determined.

The immersion experience will be uncomfortable as you will experience some of the symptoms of mild hypothermia. You will probably shiver a great deal and your fingers and toes may feel numb. Physically you may experience weakness and uncoordination. You may also become confused and feel lightheaded. As you will be supervised by a physician at all times, there is no danger of you experiencing the more serious symptoms of moderate or severe hypothermia such as gross muscle stiffness, unconsciousness, or cardiac dysfunction.

Treatment Procedures

Shivering Thermogenesis

You will be lightly dried with a towel and asked to lie on a mattress inside a sleeping bag. Your body temperature will rise as a result of metabolic heat produced by shivering thermogenesis. However, 10% of the population may not shiver to any substantial degree. If you should happen to be a nonshiverer, another treatment will be commenced immediately. During this and all other treatments you will be required to breath through a face mask connected to a metabolic measuring system. Each of the treatment procedures will be continued until your body core temperature increases to above 35.5 degrees C which is safely out of the hypothermic range. At this point you may sit in tub of water heated to 44 degrees C until you feel comfortable enough to leave. The treatment phase of the experiment will last approximately 1-2 hours.

Inhalation of heated water saturated air

This procedure is identical to the shivering procedure except that you will breath air saturated to 100% relative humidity and heated to 45 degrees C through the provided face mask.

Ingestion of hot fluids

You will be lightly towel dried and asked to sit, inside a sleeping bag, on a chair. At this point you will be given 1.5-2 liters of warm juice (approx 60 degrees C) and asked to drink it as quickly as is comfortable for you. When you are done, you will then lie down on the mattress until your core temperatures has risen sufficiently.

Exercise metabolism

After towel drying you will put on two sets of clothing (to insulate from exterior warming) and be asked to walk on a treadmill. The speed will be set to what you can comfortably maintain and you may increase or decrease it at your convenience.

CONSENT

I have read this entire 8 page 'informed consent' and agree to voluntarily take part in the experiments to be carried out for this masters thesis project and realize that I am free to withdraw from this study at any time.

I have answered the Par Q Questionaire truthfully and know of no physical or medical reason why I should not partake in this study. I agree to have a medical examination previously described. This will take place before I am allowed to undergo experimental treatments.

The information that is obtained during the experimental sessions will be treated as privileged and confidential. It may, however, be used for statistical or scientific purposes with my right to privacy retained. I realise that parts of this experiment may be video taped for educational or publicity reasons. If I do not want to be photographed my wish will be signified by not signing the second part of this document.

I have read the foregoing form and understand it. Any questions that have arisen or occurred to me have been answered to my satisfaction.

I consent to take part in this experiment.

Signature_____

Witness_____

Date_____

I consent to be photographed during the experiment.

Signature_____

Witness_____

Date_____

ADENDUM

In the time between signing of this form and the ommencement of trials, several changes in the experimental design were made. Since none of these changes required anything extra of the subjects, a new form was not written. Changes in this form were as follows:

1. VO2 max was measured on a maximal treadmill test (Bruce et al., 1963). A minimum score of 40 ml/kg/min was required to participate in the experiment.
2. 12 lead ECG and chest x-rays were generally not taken.
3. Manitoba Medical numbers were not required for medical examinations.
4. All three ECG electrodes were applied to the chest area.
5. The application of anesthetic to the nose prior to insertion of the

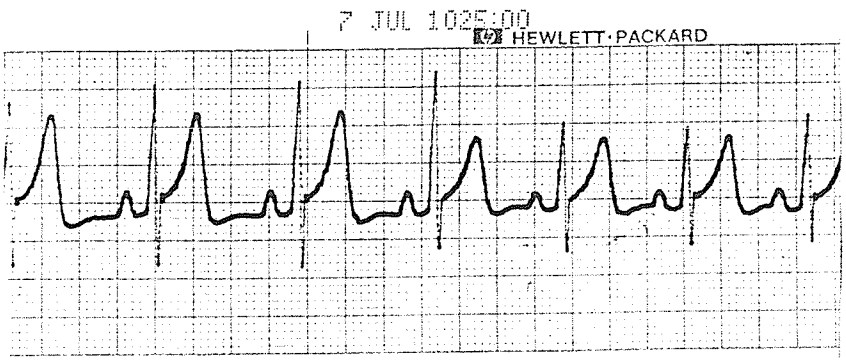
   esophageal probe was optional.
6. ECG was monitored at 15 minute intervals during cooling.
7. Subjects sat in water warmed to only 40 degrees C after treatments were completed.
8. 'Inhalation of heated water saturated air' and 'Ingestion of hot fluids' treatments were deleted from this experiment.

9. The Max Bell Researach Laboratory is actually the Sport & Exercise Research Institute located at the Max Bell Center.

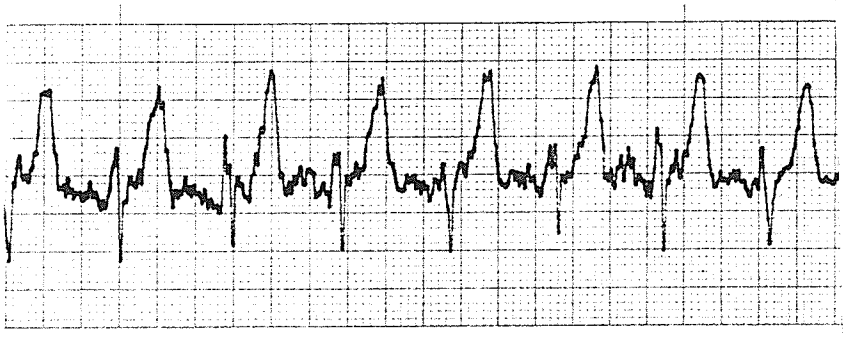
Appendix B

ECG RECORDINGS

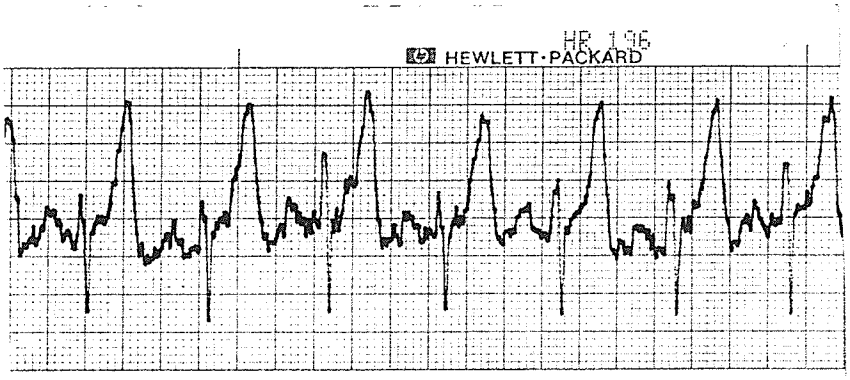
Subject GG, cooling phase.



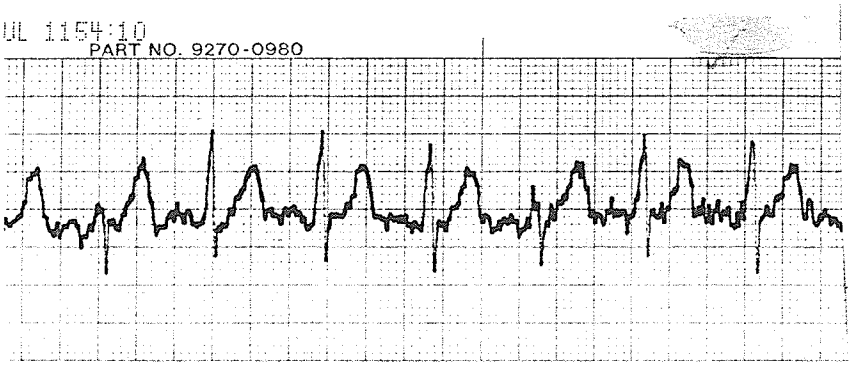
37.0°C



34.8°C

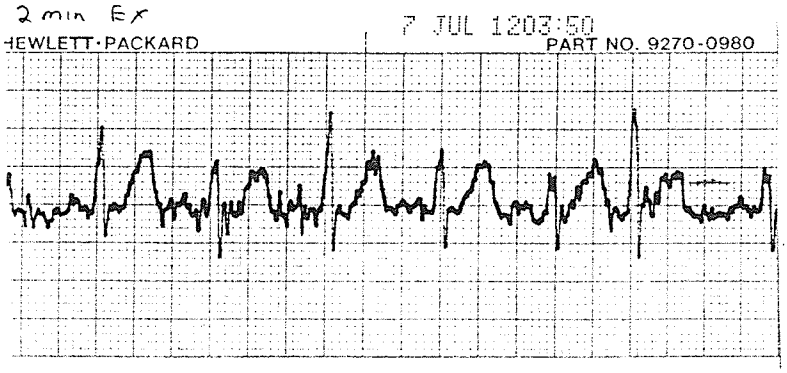


33.8°C

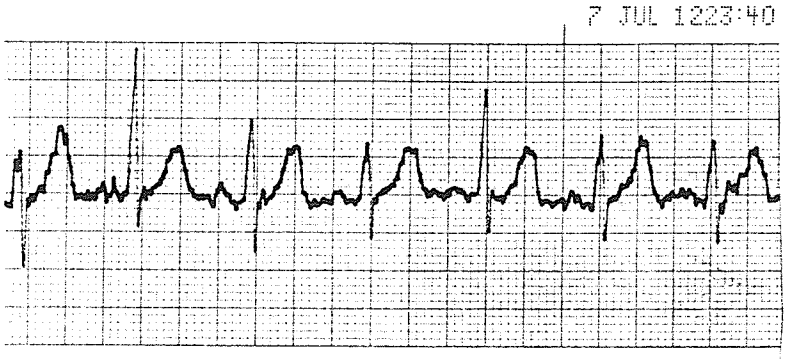


32.0°C

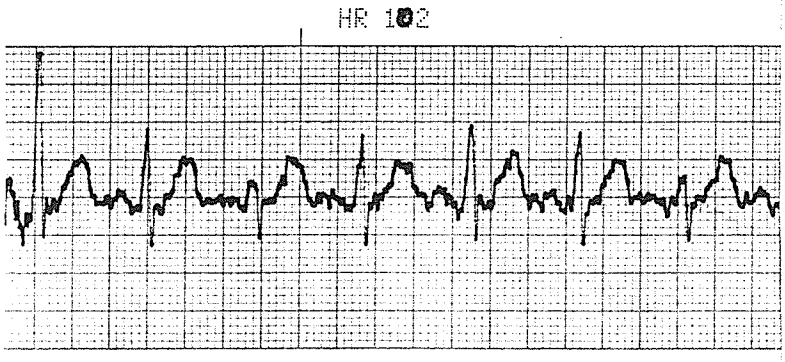
Subject GG, treatment-exercise, rewarming phase.



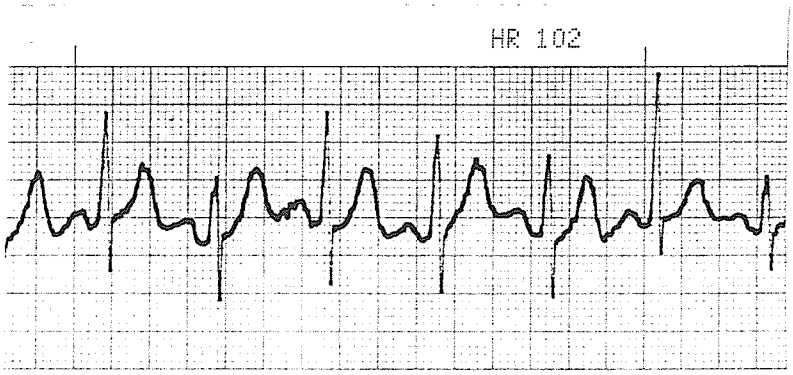
31.8°C



31.2°C

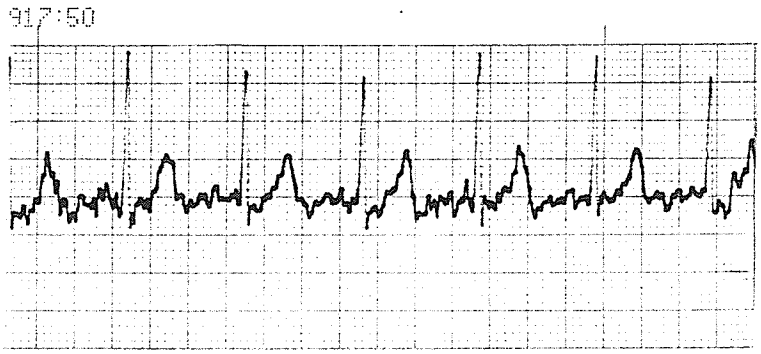


33.0°C

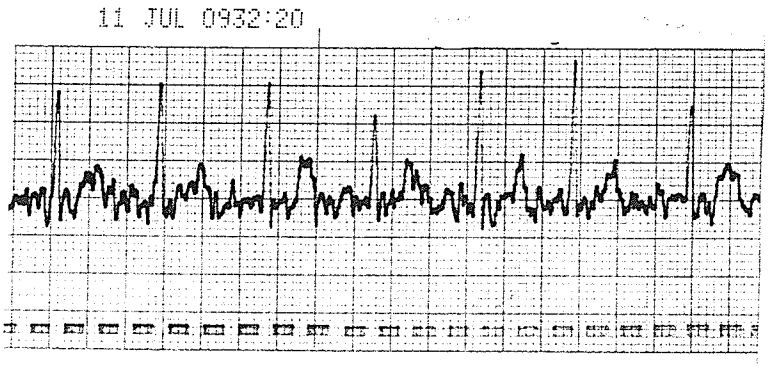


35.5°C

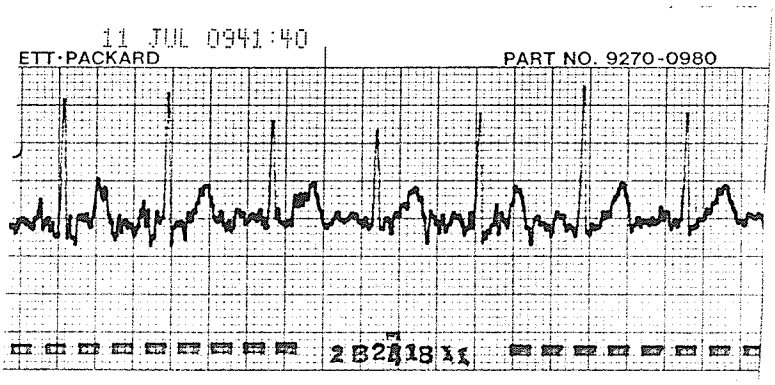
Subject RS, treatment-exercise.



End of cooling. 33.0°C

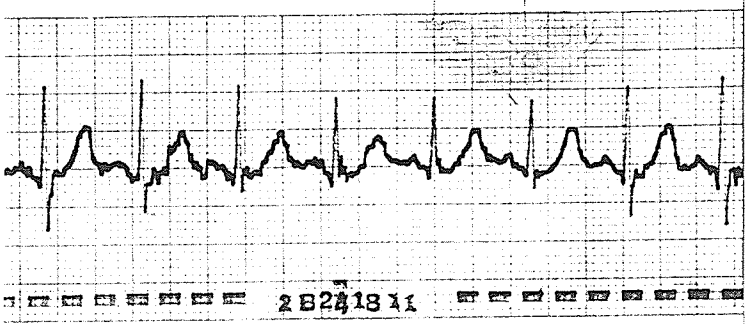


10 minutes of exercise. 32.3°C

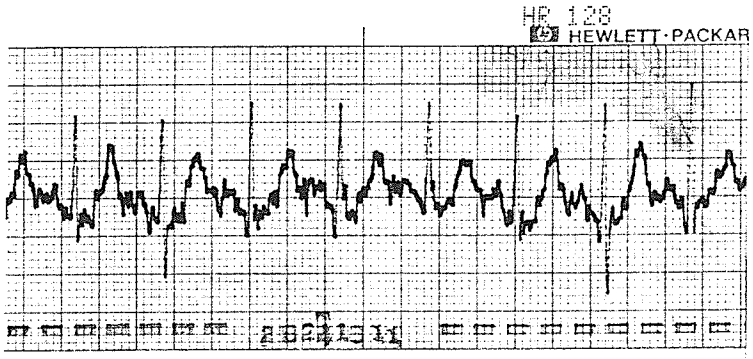


30 minutes of exercise. 34.4°C

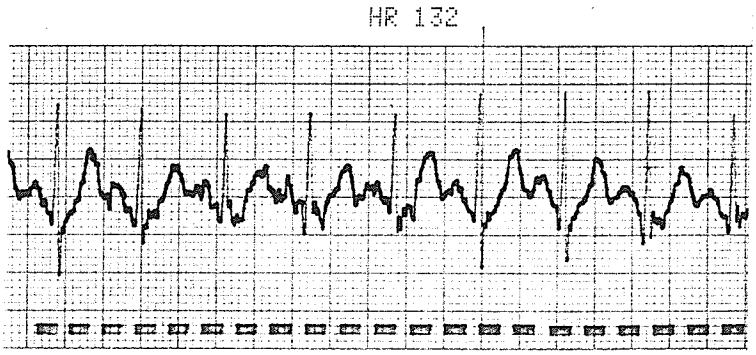
Subject BT, treatment-exercise, rewarming phase.



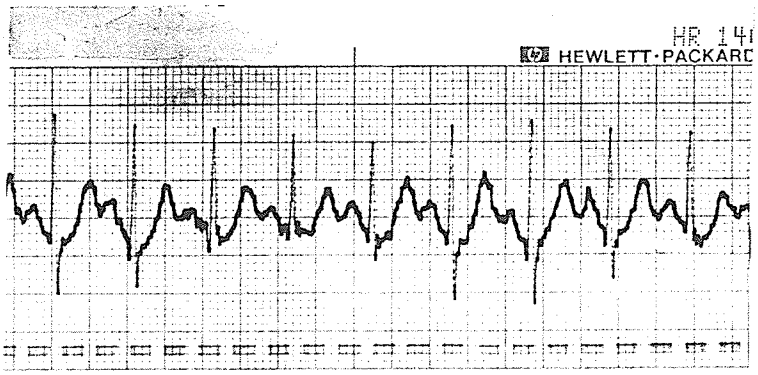
33.3°C



33.8°C

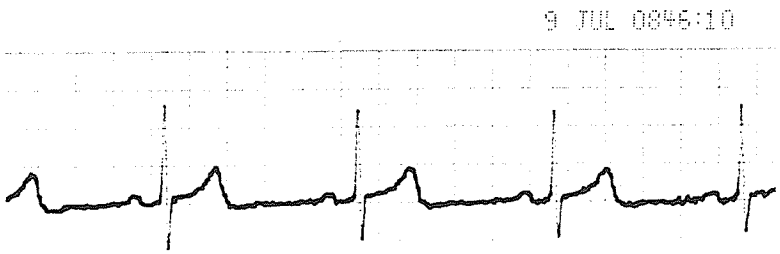


34.2°C

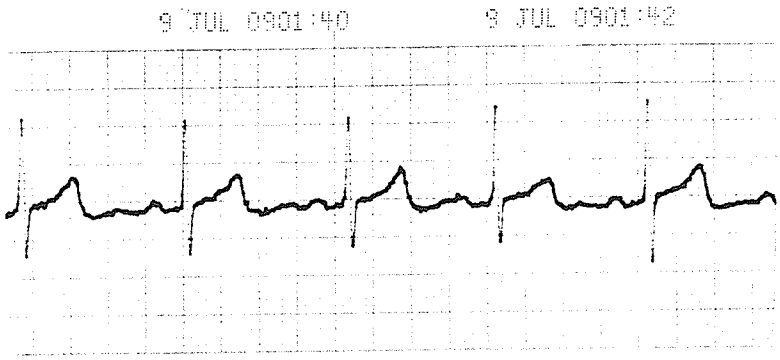


35.1°C

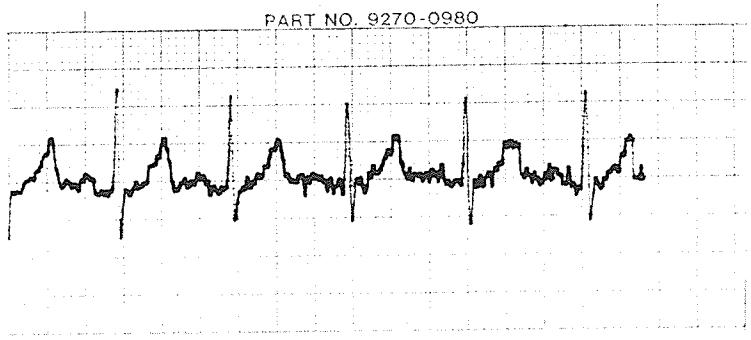
Subject PH, cooling phase,



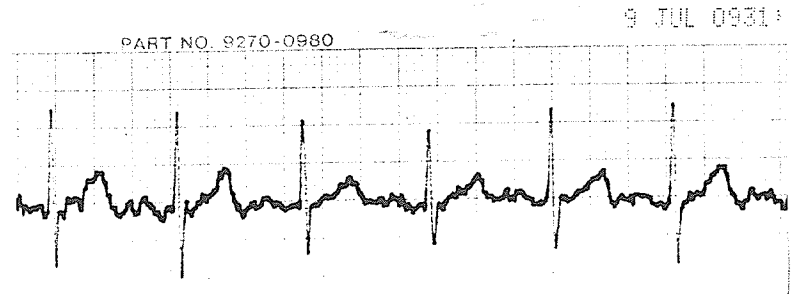
36.7°C



36.4°C



34.5°C



34.0°C