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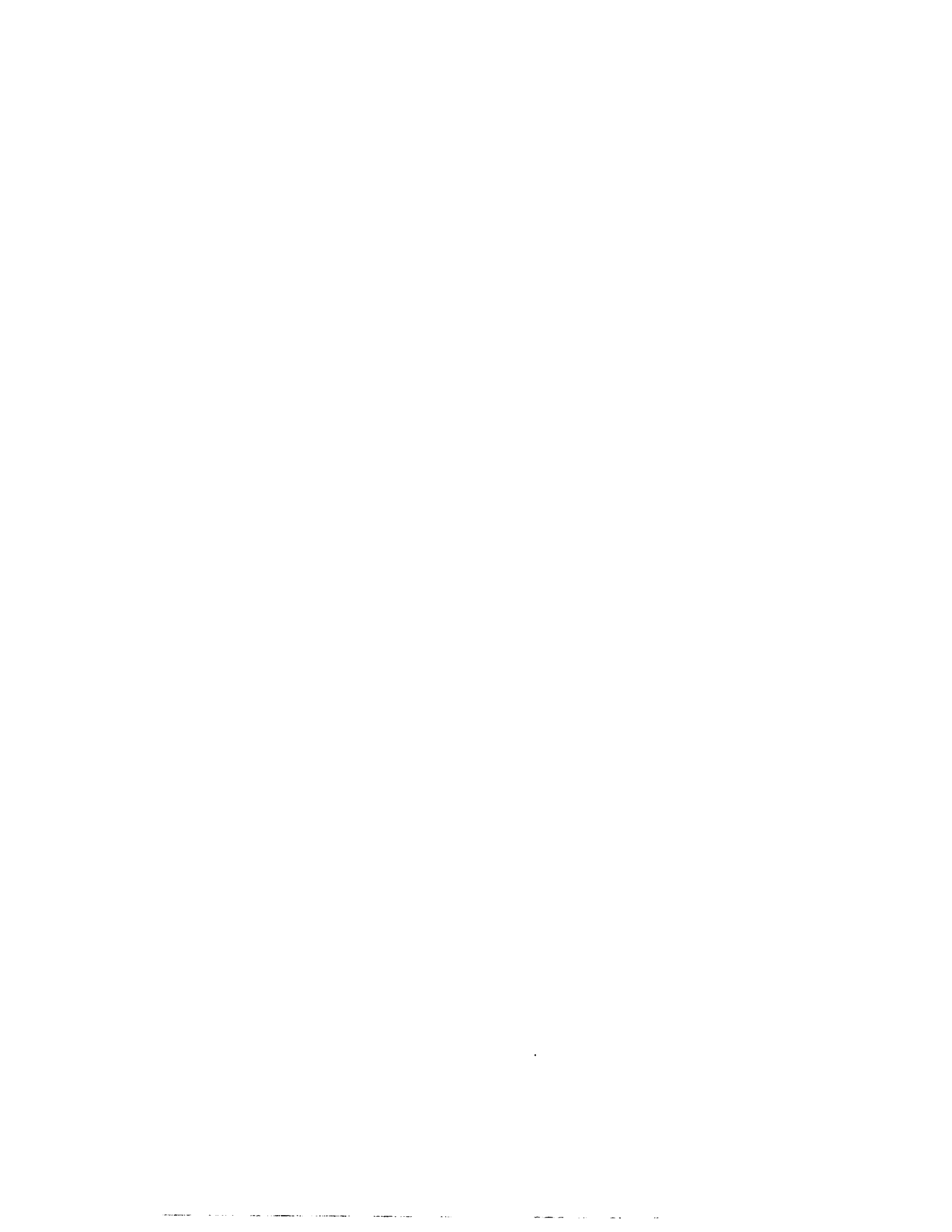
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Physiological Responses to Convective-air Blanket Warming of Women after 60  
Minutes of Cool Exposure

by

Diane Audiss

A dissertation submitted in partial fulfillment  
of the requirements for the degree of

Doctor of Philosophy

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Approved by

*Loren A. Thomas*  
(Chairperson of Supervisory Committee)

Program Authorized  
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## Doctoral Dissertation

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Abstract

Physiological Responses to Convective-air Blanket Warming of Women  
after 60 Minutes of Cool Exposure

by Diane Audiss

Chairperson of Supervisory Committee: Associate Professor Karen Thomas  
Department of Parent Child Nursing

Convective-air warming is frequently applied to postoperative patients, but the process of warming is not understood. Hence, the study purpose was to describe the rewarming process during convective-air warming in women, 40 to 60 years, after exposure.

Methods: Eight reclining women were exposed to 5 minutes of 35° C water, then 45 minutes of 30° C water, then three 5 minute incremental increases with the final 5 minutes at 35° C. The subjects transferred to a dry, reclining position. Warming consisted of a baseline interval with the subject covered by a deflated blanket and two cotton blankets, followed by a 50 minute active warming interval on high setting. The variables of interest were central temperature (esophageal temperature,  $T_{es}$ ) using a thermistor, mean skin temperature (Quartermaster weighted mean temperature,  $T_{qrec}$ ) using thermocouples, metabolic rate (oxygen consumption,  $VO_2$ ) using a metabolic cart, conductance (Thermal Circulation Index, TCI) calculated from environmental, central and skin temperatures, body size (body mass index, BMI, and skinfold thickness) using height, weight and a caliper (respectively), and vasoactivity (forearm blood flow, FBF) using plethysmograph, temperature gradient, and forearm temperature.

Results: During cooling, all subjects experienced a decreasing  $T_{es}$  (mean  $0.24 \pm 0.19^\circ \text{C}$ ). During warming, all subjects experienced afterdrop (mean  $0.18 \pm 0.10^\circ \text{C}$ ).  $T_{qrec}$  and FBF increased during baseline and active heating with the greatest rise during active warming.  $VO_2$  decreased during active warming. TCI, BMI, skinfold thickness, and FBF correlation with change in  $T_{es}$  were  $r = 0.85, -0.45, -0.50,$  and  $0.18$ , respectively. FBF to forearm-index finger correlation within subjects resulted in  $r$ -values of  $0.70$  to  $-0.90$  and to forearm temperature in  $r$ -values of  $0.60$  to  $0.90$ . Burton's three weighted skin sites are highly correlated with  $T_{qrec}$ .

Discussion: All individuals experienced afterdrop which took up to 60 minutes for recovery. The convective-air blanket was an effective therapy, since  $T_{es}$ ,  $T_{qrec}$  and FBF rose while  $VO_2$  decreased. BMI, skinfold thickness and duration of afterdrop are better predictors of change in  $T_{es}$  than FBF. Forearm temperature is a better predictor of FBF than forearm-index finger gradient.

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## ABBREVIATIONS

BMI	Body mass index
CAVR	Continuous arterio-venous rewarming
CPB	Cardio-pulmonary bypass
FBF	Forearm blood flow
ICU	Intensive Care Unit
PACU	Postanesthesia Care Unit
$T_{\text{abdomen}}$	Abdominal skin temperature
$T_{\text{amb}}$	Ambient temperature
$T_{\text{back}}$	Back skin temperature
$T_{\text{bl}}$	Bladder temperature
$T_{\text{burton}}$	Burton 3-site, weighted mean skin temperature
$T_{\text{calf}}$	Calf skin temperature
TCI	Thermal circulation index
$T_{\text{cheek}}$	Cheek skin temperature
$T_{\text{chest}}$	Chest skin temperature
$T_{\text{env}}$	Environment temperature
$T_{\text{es}}$	Esophageal temperature
$T_{\text{fa}}$	Forearm skin temperature
$T_{\text{fa}}$	Forearm skin temperature
$T_{\text{fa-if}}$	Forearm minus index finger skin temperatures
$T_{\text{foot}}$	Foot skin temperature
$T_{\text{forehead}}$	Forehead skin temperature
$T_{\text{front thigh}}$	Front thigh skin temperature
$T_{\text{h\&d7}}$	Hardy and Dubois 7-site, weighted mean skin temperature
$T_{\text{hand}}$	Hand skin temperature

$T_{if}$	Index finger skin temperature
$T_{\text{medial thigh}}$	Medial thigh skin temperature
$T_{or}$	Oral temperature
$T_{p\&p}$	Palms and Parker 6-site, weighted mean skin temperature
$T_{pa}$	Pulmonary artery blood temperature
$T_{pa-bl}$	Pulmonary artery minus bladder temperatures
$T_{pa-es}$	Pulmonary artery minus esophageal temperatures
$T_{pa-or}$	Pulmonary artery minus oral temperatures
$T_{pa-re}$	Pulmonary artery minus rectal temperatures
$T_{pa-ty}$	Pulmonary artery minus tympanic temperatures
$T_{qrec}$	Quartermaster Corps 10-site, weighted mean skin temperature
$T_{\text{Ramanathan}}$	Ramanathan 4-site, weighted mean skin temperature
$T_{re}$	Rectal temperature
$T_{sk}$	Skin temperature
$T_{\text{Teichner}}$	Teichner 6-site, weighted mean skin temperature
$T_{ty}$	Tympanic temperature
$T_{\text{Upper arm}}$	Upper arm skin temperature

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## CHAPTER I

### Statement of the Problem

Nurses prescribe whole body rewarming therapies for postoperative, hypothermic patients. The criteria for selection of rewarming therapies is severely lacking in the literature. Furthermore, the literature does not provide a description of the individual's physiological responses to rewarming. Theoretically, the rewarming process is affected by the metabolic rate and the ability to transfer heat from the applied therapy into the body. Furthermore, the rewarming process is affected by the therapy's effectiveness at eliminating or decreasing body heat loss to the environment.

The physics principle governing the ability to transfer heat from the exterior to the interior of the body is conductance. In the human body, conductance is affected by the transfer of heat across an insulation layer which is made up of subcutaneous fat and vasoconstricted skin. When searching the literature, no studies describing the rewarming process utilizing metabolic rate, conductance, subcutaneous body fat and vasoconstriction were identified. In hypothermic postoperative individuals, metabolic rate across age groups was studied and found to decrease across the adult life span (Roe, Goldberg, Blair, & Kinney, 1966). Hence, when studying rewarming, subject age must be considered along with the variables of metabolic rate, conductance, subcutaneous body fat and vasoconstriction.

The incidence of postoperative hypothermia in adults was 60 to 76% when the definition of hypothermia was a central temperature of 36<sup>o</sup> C or less (Erickson & Yount, 1991; Morris, 1971b; Slotman, Jed, & Burchard, 1985; Vaughan, Vaughan, & Cork, 1981). If hypothermia was defined as any decline in central temperature, 83 to 100% of adults who under went surgery

experienced hypothermia (Erickson & Yount, 1991; Morris, 1971b; Roe, et al., 1966). Approximately one-half of the postoperative hypothermic individuals are women, but there is virtually no research depicting postoperative rewarming in women.

The postoperative population who experienced hypothermia had an increase in morbidity and mortality rates which affected the individual's health outcome and health care costs. The morbidity indicators included physiological and psychological changes identified in hypothermic individuals. Physiological morbidity indicators were an increase in systemic vascular resistance (Hayward, Eckerson, & Kemna, 1984), the development of hypokalemia (Boelhouwer, Bruining, & Ong, 1987), prolonged prothrombin and partial thromboplastin times, and increased fibrin split products (Gentilello, Cobean, Offner, Soderberg, & Jurkovich, 1992). The aforementioned physiological morbidity indicators reverted to normal or more normal values with rewarming. Another physiological morbidity indicator, identified in elderly subjects, was a significant increase in catabolism of muscle protein measured by the excretion of 3-methylhistidine following a postoperative hypothermic event (Carli, & Itiaba, 1986). The aforementioned physiological morbidity indicators may manifest as a decrease in the individual's cardiac output, cardiac dysrhythmias, bleeding problems which may progress to disseminated intravascular coagulation, muscle wasting, or a decreased rate of healing postoperatively. The psychological morbidity indicator, thermal discomfort, was significantly higher in postoperative hypothermic individuals if no rewarming therapy was applied (Summers, Dudgeon, Byram, & Zingsheim, 1990). The postoperative hypothermic individual experienced

discomfort which may contribute to an increase in systemic vascular resistance.

Furthermore, individuals with a prolonged pattern of rewarming postoperatively had a significantly increased mortality rate when compared to individuals who had a shorter pattern of rewarming (Matthews, Meade, & Evans, 1974; Slotman, Jed, & Burchard, 1985). Slotman et al. (1985) reported a significant ( $p < 0.01$ ) increase in the percent mortality rate of postoperative individual's who had a central temperature of less than  $36.1^{\circ}\text{C}$  at 2, 4 and 8 hours postoperatively when compared to individual's with temperatures greater than  $36.1^{\circ}\text{C}$  at the same time intervals. The mortality rates at 2 hours were 25% compared to 4%, at 4 hours 47% and 9%, at 8 hours 79% and 13% (subjects with a central temperature less than  $36.1^{\circ}\text{C}$  compared to subjects with a central temperature greater than  $36.1^{\circ}\text{C}$ , respectively). Matthews et al. (1974) reported a 53% mortality rate in post-cardiac surgery patients who required more than 8 hours to reach a toe skin temperature of  $34^{\circ}\text{C}$  compared with a 6% mortality rate in those individuals who reached a toe skin temperature of  $34^{\circ}\text{C}$  in less than 8 hours postoperatively. Finally, Gentilello et al. (1992), studied postoperative individuals, with central temperatures of less than  $35^{\circ}\text{C}$ , who were rewarmed either entirely by self-rewarming or with continuous arterio-venous rewarming (CAVR). The study reported a nonsignificant difference in mortality rate between the self rewarmed and the CAVR groups (67% and 50% mortality rates, respectively). However, a 50% mortality rate is clinically important when compared to a 67% mortality rate. Hence, postoperative patients with prolonged hypothermia do have an increased mortality rate.

Increased health care costs for the hypothermic patient are incurred from the cost of the therapy utilized and from a prolonged length of stay. The cost of rewarming therapies varies. For example, the application of warmed cotton blankets costs \$39.62, based on an average of 6 blankets per patient, while the continuous heated air blanket (Bair Hugger) costs \$13.35 per hypothermic patient (Augustine, 1990). The described costs were based on data from telephone interviews with 100 hospitals across the United States. The cost was calculated including nursing time, initial-, overhead-, and equipment-costs.

The length of time the patient is maintained in an area of specialty care, such as the postanesthesia care unit (PACU) or an intensive care unit (ICU) because of unresolved hypothermia, also increases health care costs. In the PACU, one of the discharge criteria may be a minimum core temperature. If an ineffective therapy is utilized, the length of time in the PACU will increase which increases the costs. In the ICU, Gentilello et al. (1992) studied the length of stay in relation to rewarming with no- or an ineffective-therapy and an invasive rewarming therapy. Gentilello et al. reported the untreated or ineffectively treated hypothermia group's median length of stay in the ICU to be 22 days while the continuous arterio-venous rewarmed group had a median length of stay of 5 days. Therefore, the postoperative, hypothermic individual frequently experiences an increased morbidity and mortality as well as an increase in the health care costs.

To decrease the morbidity-, mortality-rate and the cost of health care of the postoperative hypothermic patient, nursing must prescribe effective rewarming therapies. However, to prescribe effective therapeutics, nursing must understand the rewarming process within the individual and the effect of the therapeutic upon the rewarming process within the individual. The

variables of interest in describing the rewarming process are conductance, subcutaneous fat, vasoactivity and metabolic rate. The variable measurements must be applicable to the clinical arena. Therefore, the purpose of this study was to describe the rewarming process prior to and during the application of a non-invasive rewarming therapy in women 40 to 60 years of age. The study questions were:

1. What were the patterns of rewarming defined by the trend in esophageal ( $T_{es}$ ) and mean skin ( $T_{sk}$ ) temperatures, forearm blood flow (FBF) and metabolic rate prior to and following the application of a warmed convective air blanket?
2. What were the relationships of conductance (thermal circulation index), vasoactivity (FBF), body fat (total mm of body fat from 4 sites) or body mass-to-surface area ( $\text{kg}/\text{m}^2$ ) with esophageal warming when the rewarming therapy is a warmed convective air blanket?
  - a. Within an individual and between individuals, how did the conductance measurement (thermal circulation index =  $T_{sk} - T_{environment} / T_{es} - T_{sk}$ ) prior to application of the warmed convective air blanket relate to the rate of rewarming ( $^{\circ}\text{C}/\text{min}$  rise in  $T_{es}$ ) across the warming period?
  - b. Within an individual and between individuals, how did vasoactivity (FBF) prior to application of the warmed convective air blanket relate to the rate of rewarming ( $^{\circ}\text{C}/\text{min}$  rise in  $T_{es}$ ) across the warming period?
  - c. Between individuals, how did body mass to surface area index ( $\text{kg}/\text{m}^2$ ) or to body fat (total mm of body fat from 4 sites) relate to the rate of rewarming ( $^{\circ}\text{C}/\text{min}$  rise in  $T_{es}$ ) across the warming period?

3. Using FBF as the criterion, what is the precision with which vasoactivity can be estimated with forearm - index finger temperature ( $T_{fa}-T_{if}$ ) and forearm temperature ( $T_{fa}$ ) during warming?
4. Using Quartermaster Corps 10-point weighted mean  $T_{sk}$  ( $T_{qrec}$ ) as the criterion, what is the precision with which the mean  $T_{sk}$  can be estimated with fewer sites during warming?

## CHAPTER II

### Review of Literature

The development of hypothermia and rewarming are processes affected by the movement of heat between the individual's body and the environment. This chapter will discuss heat transfer within the body and between the body and the environment, thermoregulation, hypothermia, and rewarming therapeutics.

#### Heat Transfer

Heat transfer is the process by which temperature equilibration occurs between areas or surfaces, e.g. within the body or between the body and the environment. Heat is transferred by four different principles which are conduction, convection, radiation and evaporation. Conduction is the movement of heat from one molecule to another molecule with no progressive movement of the masses or surfaces. Convection is the movement of heat in gas or liquid. Radiation is the movement of heat in rays. Evaporation is the movement of heat from an exposed surface by conversion of a liquid into a vapor.

All of the principles function based on a gradient. The greater the temperature gradient, the greater the rate of heat flow or transfer (Newton's Law of Cooling; Mott-Smith, 1962). The direction and the rate of heat flow is affected by the difference in temperature when discussing conduction, convection and radiation. The gradient of interest in evaporation is the amount of humidity surrounding the area in which the evaporation process occurs. If the air surrounding the surface is 100% saturated, the rate of movement from the liquid state to vapor is equal to the rate of movement from

the vapor state back to the liquid state resulting in no change in evaporation of liquid.

The principles of evaporation, radiation, convection and conduction affect heat transfer between the surface of the human body and the environment where the body is in contact with the environment. The principles of convection and conduction affect heat transfer within the body. Within the body, convective transfer of heat occurs as the blood flows through the vasculature of the body. The physiological phenomena of vaso-constriction or -dilation alters the blood flow which effectively alters the heat flow by convection. The conduction of heat through the body is affected by varying tissues, such as adipose and muscle, as well as varying thicknesses of tissues within an individual and between individuals. For example, the conduction rate of heat through muscle is approximately twice the rate of conduction through adipose tissue (Hatfield, 1953). Therefore, when discussing heat movement within the body, one must consider the physiological factors which affect the principles of heat flow. Hence, for the purposes of discussion, heat flow within the body will be referenced by individual factors (e.g. vasoconstriction or subcutaneous adipose thickness) while heat transfer between the body and the environment will be referenced by the heat transfer factors (e.g. conduction; Table 1).

The rate of heat transfer into or out of the body is affected by individual and environmental factors (Table 1). The individual factors which affect endogenous heat transfer are vasoactivity, conductance, and body surface body mass quotient (Brück, 1978). The individual factor which affects the rate of endogenous heat production is the metabolic rate. The environmental heat exchange factors of convection, conduction, and radiation affect exogenous

heat-gain or -loss depending upon the direction of the temperature gradient between the body and the environment. Evaporation facilitates exogenous heat loss. The mechanisms of heat transfer and heat production will be discussed further, but first the process of thermoregulation which is the means of controlling body heat will be introduced.

TABLE 1. Factors Affecting Maintenance of Normothermia.

	Heat Transfer	Heat Production
Individual	Vasoactivity (convection) Conductance Conductivity constant (conduction) Absolute tissue thickness (conduction) Blood flow rate to surface (convection) Body surface body mass quotient (area)	Metabolic rate
Environmental	Conduction Convection Radiation Evaporation	

#### Adult Human Thermoregulation

Thermoregulation is the process of balancing heat gain and heat loss in an individual to maintain normothermia. The heat balance is maintained by thermal sensory input which is integrated in the brain and results in effector output to conserve-, to dissipate-heat or to increase heat production. The thermal sensory input is from both cold and warm sensors which exist in various parts of the body including the skin, spinal cord and central organs of the body. The sensory information from the various areas of the body is integrated in the hypothalamus with the effector output delivered to the periphery via the sympathetic nervous system. The central thermal sensory input has a 10 to 20 times greater influence on vasoactivity than the

peripheral thermal sensory input (Rowell, 1986). Hence, in a cold environment, the two dynamic physiological mechanisms utilized to maintain body temperature are increasing vasoconstriction of vessels within the skin and increasing the metabolic rate via shivering. If the central temperature is below normal, the vasoconstriction will be greater than the amount of constriction seen if only the skin surface temperature were affected.

Rhythms. The body heat load does not remain constant through out a 24 hour period. The circadian rhythm is reflected in the body temperature with the highest temperature measured in the late afternoon or early evening, between 1600 and 2000, and the lowest temperature measured in the early morning, between 0400 and 0800 (Minors & Waterhouse, 1981). Therefore, the thermoregulatory process does not maintain a constant temperature through out the 24 hour period. Minors and Waterhouse hypothesized the set point, the temperature setting which the body regulates, exhibited a circadian rhythm. Although the body temperature does vary from  $0.5^{\circ}$  to  $1.0^{\circ}$  C during the circadian cycle (Minors & Waterhouse, 1981), the thermoregulatory process continues through out the 24 hour period.

Females between puberty and menopause experience a menstrual temperature rhythm. During the luteal phase of the menstrual cycle, progesterone levels increase. Progesterone acts as a thermogenic agent resulting in an increase in central temperature by approximately  $0.5^{\circ}$  C during the luteal phase (Goldfein & Monroe, 1986). In pre-menopausal women, the production of progesterone and estrogen begin decreasing. Following menopause, during the follicular phase, the progesterone levels are approximately 30% of the levels seen in young women (Goldfein & Monroe, 1986). Hence, the body does regulate temperature, but the temperature around

which the regulation occurs is dependent upon both the circadian rhythm and in females upon the menstrual rhythm. Furthermore, during pre-menopausal and menopausal phases of a woman's life, the menstrual rhythm effect decreases.

The body thermoregulates by manipulating vasoactivity which affects heat transfer. However, other individual and environmental factors affect heat transfer within the individual as well as between the individual and the environment. Hence, the individual factors, including vasoactivity, and the environmental factors will be discussed.

### Individual Factors

Vasoactivity. With a decline in the central body- or the skin-temperature, the hypothalamic effector output results in vasoconstriction of the skin vasculature which decreases the volume of warm blood carried to the skin surface. The decrease of blood flow to the skin surface decreases the rate of central cooling via a smaller volume of cooled, skin surface blood returning to the central body. The blood flow within the skin vasculature does vary greatly. For example, the forearm skin blood flow varies from 0.2 L of blood/min/100 grams of tissue with maximal vasoconstriction, to a resting state blood flow of 2 to 4 L of blood/min/100 grams of tissue, to 15 to 20 L of blood/min/100 grams with maximal vasodilation (Brenzelmann, 1989; Rowell, 1986). Hence, the volume of blood presented to the skin surface varies greatly yielding a fine tuned system with tremendous capacity to retain or loose heat.

Vasoactivity in the skin is controlled by three different mechanisms which are active vasoconstriction, active vasodilation and local vasodilation (Rowell, 1986). Active vasoconstriction is mediated by the hypothalamic output to the sympathetic nervous system, via the intermediolateral cell column, to alpha-

adrenergic receptors in both acral (vasculature with arteriovenous anastomoses such as fingers, toes, nose and ears) and nonacral skin (vascular system which feeds from arterioles to capillaries to veins such as in the limbs and the trunk) (Rowell, 1974). As core and skin temperatures increase, the neural activity to the receptors decreases, in order the vasoconstrictor tone decreases. In acral skin sites, such as the hand, the blood flow rapidly increases from 4 to 13 ml/100 ml/min with passive rewarming. However, in nonacral skin sites, such as the forearm, the blood flow increases from 3 to 5 ml/100 ml/min and plateaus with decrease of vasoconstrictor tone (Rowell, 1986).

The mechanism of control of active vasodilation is not understood, but it is known that active vasodilation is not present in acral skin (Barcroft, 1960; Rowell, 1974; Rowell, 1986). Active vasodilation is under the control of the sympathetic nervous system, which is demonstrated by blocking the sympathetic nerves supplying the lower arm and demonstrating that the blood flow to the forearm decreases back to the level where vasoconstrictor tone was removed. However, acral tissue of the hand displays no decrease in blood flow with nerve blockade (Rowell, 1986).

Finally, local vessel vasodilation in response to local skin heating is seen even in denervated skin. The mechanism of local response is not fully understood, but it is believed to be a response to heat within the vessel musculature (Rowell, 1986). Barcroft and Edholm (1943) studied the local vasodilation response to heat on the forearm and reported that a water bath temperature of 38<sup>o</sup> C was required before a local vasodilation was measured. In the water bath temperature range of 38 to 42.5<sup>o</sup> C, the peak vasodilation occurred between 40 and 60 minutes of exposure with a decline in vasodilation

following 60 minutes of exposure to the warm bath. In 45<sup>o</sup> C water bath temperature, vasodilation did not decline following 60 minutes of exposure but actually continued to display a gradual rise in vasodilation. Hence, the mechanisms of vasoactivity vary with the area of the body and probably are not under the same control mechanisms.

The degree of vasoconstriction or vasodilation varies within an individual at various sites under constant environmental conditions and within an individual at a given site under varying environmental conditions. For example, Rowell (1986) described the blood flow to acral skin, the hand, as 30 ml/100 ml/minute and to nonacral skin, the forearm, as 12 ml/100 ml/minute in the same environmental conditions with indirect heating (placing the legs in hot water). Furthermore, the blood flow to the same site varies with the environmental condition. Blair, Glover, and Roddie (1960) with indirect heating (Tamb 21 to 23<sup>o</sup> C with arms or legs placed in 44<sup>o</sup> C bath) described a forearm blood flow of 6.5 ml/100 ml/minute, an upper arm flow of 8 ml/100 ml/minute, a thigh flow of 4 ml/100 ml/minute and a calf flow of 5 ml/100 ml/minute. With indirect cooling (Tamb 16 to 18<sup>o</sup> C with arms or legs placed in 17<sup>o</sup> C bath), the forearm blood flow was 1.75 ml/100 ml/minute, the upper arm flow was 1.0 ml/100 ml/minute, the thigh flow was 1.0 ml/100 ml/minute and the calf flow was 0.75 ml/100 ml/minute (Blair et al., 1960). Hence, the mechanisms of control do not predict the actual flow values which will be seen within an individual in various areas of the body under similar or varying environmental conditions. Furthermore, a large variation in skin blood flow can occur in the attempt to conserve body heat.

The postoperative, hypothermic individual experiences vasoconstriction of the skin vasculature from active vasoconstriction in response to hypothermia.

The vasoactive state is also affected by maintenance of a normal vascular pressure (Rowell, 1986). Therefore, a state of skin vasoconstriction is very likely in postoperative hypothermic individuals due to both hypothermia and decreased vascular volumes. Since the central temperature has a 10 to 20 times greater influence on the vasoactive state than the skin temperature (Rowell, 1986), the local effect of vasodilation with heat application in a hypothermic person will be less than the vasodilation noted in normothermic individuals. Hence, in the postoperative hypothermic individual, active vasoconstriction will be in effect, and a lesser effect of local vasodilation will be seen with application of the rewarming therapy to the skin surface. Hence, the convective mechanism of heat transfer from the therapy into the body will be less effective until the central temperature reaches a more normal value.

Conductance. Conductance is the transfer of heat between two parallel surfaces. The rate of conductance is the rate at which heat is transferred between a given area of two parallel surfaces when the temperature difference is maintained between the surfaces (Bligh & Johnson, 1973). The three factors which affect the rate of conductance of a tissue are the conductivity constant of the given tissue, the absolute thickness of the tissue, and the blood flow rate through the tissue (Brück, 1978). The primary body tissues through which heat is conducted from the central mass to the body surface are muscle and adipose. Since the rate of conduction through adipose tissue is one half the rate through muscle tissue, adipose tissue will slow the rate of heat loss from the body as well as slow the rate of heat gain during rewarming.

The absolute thickness of the tissue also affects the rate of heat loss or gain. The greater the distance over which the exchange of heat must occur the

greater the time for equilibration of temperature. Within an individual, the amount of adipose and muscle tissue does not vary during short time frames of thermoregulation. However, when applying an external warming or cooling therapy the mass of tissue does affect the rate of heat gain or loss.

Finally, heat is transferred between the various tissues and the blood flowing through the tissues. The direction of heat transfer is down the temperature gradient between the blood and the tissues. Hence, via conduction heat moves from the warmer peripheral blood into the cooler peripheral tissue. Since, a temperature gradient effect exists between the peripheral tissue and the environment, the peripheral tissue may lose heat to the environment. When the cooler blood returns to the central tissues where the temperature gradient between blood and tissues is reversed, another exchange of heat down the gradient occurs. The transfer of heat in the blood between peripheral and central areas is convective transfer of heat. Hence, the body heat load decreases if the blood is exposed to tissues which are cooler and which are losing heat to the environment.

Hence, conductance is affected by the relative amount of adipose- and of muscle-tissue, the absolute thickness of the tissue and the blood flow rate of the tissue. Therefore, theoretically an individual with a large layer of subcutaneous adipose and postoperative vasoconstriction will have a large layer of insulation, which will decrease the rate of heat exchange between the environment and the skin surface. A decreased rate of heat exchange with the environment is an advantage when the body is attempting to prevent hypothermia. However, when an exogenous rewarming therapy is applied the large insulation layer is a disadvantage to rewarming. As the amount of vasoconstriction to the skin decreases, the insulation effect decreases.

Theoretically, increasing blood flow to the skin increases the movement of heat from the exogenous rewarming therapy into the body by convection. The overall rate of rewarming for this individual will be dependent upon both the thickness of the adipose layer and the vasoactive state. In comparison, an individual, with a small layer of subcutaneous adipose tissue, but the same blood flow to the skin as the person described above, will rewarm faster with an exogenous rewarming therapy because of a smaller insulation layer.

Body surface body mass quotient. The greater the body surface area for a given mass the greater the amount of heat loss (Brück, 1978). The equation for internal heat flow is  $H_{int} = A \times C (T_{re} - \text{mean } T_{sk})$  where A is surface area and C is thermal conductance (Brück, 1978). Hence, if mass, which is reflected by conductance, is held constant, but the area is increased the internal heat flow will increase.

Therefore, an individual with larger surface area per unit of mass will have a greater loss of body heat. For example, an individual of 5 foot and weighing 36 kg has a body surface area of 1.26 (Daily & Schroeder, 1981) with a body surface body mass quotient of 0.035. An individual of 5 foot and weighing 45 kg has a body surface area of 1.38 with a body surface mass quotient of 0.030. The individual of 5 foot and 36 kg has a greater body surface area per mass and would have a greater heat loss at a given temperature than the other individual of 5 foot but weighing 45 kg.

Metabolic rate. The aforementioned factors focused on mechanisms of heat transfer within the body. The final individual factor focuses on the rate of heat production which is determined by the metabolic rate. The metabolic rate or rate of heat production is a dynamic process which increases with central body or skin temperature cooling. Heat is produced as oxygen is consumed in

various chemical reactions within the body. For every liter of oxygen consumed, 4.8 kcal of heat are released (Brenzelmann, 1989). Oxygen consumption varies within an individual as well as between individuals with such items as increased epinephrine levels, increased thyroid hormone levels, physical activity, psychological state, food intake, and environmental temperature. Hence, to create a standardized measure between and within individuals the state of basal metabolic rate is measured. The basal metabolic rate is defined as the heat production or oxygen consumption in an organism in a rested, awake, fasting and thermoneutral state (Bligh and Johnson, 1973). The thermoneutral state is defined as the ambient temperature range within which the metabolic rate is at a minimum and temperature regulation is achieved by nonevaporative physical processes.

In young adult humans, the average basal metabolic rate is 3.5 ml oxygen consumed/kg/min (Brenzelmann, 1989). However, across the adult age span the basal metabolic rate declines. Shock, Watkin, Yiengst, Norris, Gaffney, Gregerman, and Falzone (1963) studied one hundred ninety-three male subjects from 21 to 95 years of age. From the third (20-29 years of age) through the sixth (50-59 years of age) decades of life, the basal metabolic rate gradually increased with the maximal basal metabolic rate occurring during the sixth decade of life. From the seventh (60-69 years of age) to the tenth (90-99 years of age) decades of life, the metabolic rate gradually decreased with a metabolic rate of 2.8 ml/kg/min in the ninth (80-89 years of age) and tenth (90-99 years of age) decades of life. Hence, basal metabolic rate (oxygen consumption or the rate of heat production) is not constant across the life span. Therefore, the likelihood of an older individual developing hypothermia in a cold environment with all else being equal is greater than a younger individual.

The change in metabolic rate across the life span may affect an individual's ability to increase metabolic rate once hypothermia occurs postoperatively. Roe et al. (1966) studied the metabolic rate in 24 post-operative, general anesthesia individuals. Those individuals with a temperature fall of  $<0.3^{\circ}\text{C}$  had a 7% increase in oxygen consumption. Those individuals with a temperature decline of  $0.3$  to  $1.2^{\circ}\text{C}$  had a 92% increase in oxygen consumption. Those individuals with a temperature decline of  $>1.2^{\circ}\text{C}$  had an oxygen consumption increase of only 40%. Roe et al. attributed the smaller increase in oxygen consumption to the fact that all subjects in the group with the temperature decline of  $>1.2^{\circ}\text{C}$  were in the age group of 61 to 80 years of age. Hence, older individuals have lower basal metabolic rates and have a smaller increase in metabolic rate with exposure to a surgical environment. Therefore, age must be considered when studying postoperative rewarming rate.

During exposure to a cool environment, the metabolic rate increases when a state of shivering or of non-shivering thermogenesis occurs. During the exposure, the hypothalamus integrates the information from the skin and the central temperature sensors. In shivering, the effector output to the muscle, via the sympathetic nervous system, increases tone or sets off oscillating rhythmical muscle tremors. The increased tone or muscle tremors result in an increase in oxygen consumption. Non-shivering thermogenesis is controversial in adult humans. In non-shivering thermogenesis there is an increase in the metabolic rate without an increase in muscle tone. An increase in secretion of epinephrine is thought to effect the increased metabolic rate.

Livingstone, Grayson, Frim, Allen & Limmer (1983) studied 5 young males sitting in a warm ( $24$  to  $26^{\circ}\text{C}$ ) and in a cold environment ( $-32^{\circ}\text{C}$  with  $11\text{ km/h}$  wind). The metabolic rate at 20 and 80 minutes during the warm exposure was

41.2 and 42.9 kcal/m<sup>2</sup>/hr, respectively, with a decline of esophageal temperature (T<sub>es</sub>) and rectal temperature (T<sub>re</sub>) of 0.15° C during the exposure period. When the subjects were exposed to the cold environment, the metabolic rate rose to 65.6 and 109.1 kcal/m<sup>2</sup>/hr at 20 and 80 minutes of exposure, respectively, with a decline of T<sub>es</sub> of 0.15° C and T<sub>re</sub> of 0.05° C during the exposure period. Livingstone et al. (1983) reported that the subjects shivered during the cold exposure.

Thermogenesis is also present in operative patients. Bay, Nunn, and Prys-Roberts (1968) studied 25 post-operative, general anesthesia subjects of whom 13 did not shiver, described as "lying quietly", but had an increase in the basal metabolic rate of 71 to 135%, while the 12 subjects who did shiver, described as "sharply different from those lying quietly, obvious shivering", had an increase in the basal metabolic rate of 135 to 486%. The absence of visible shivering does not preclude an increase in muscle tone. Therefore, it is impossible to determine whether the increase in metabolic rate is from non-shivering thermogenesis.

Hence, in a cool to cold environment, an individual may experience an increase in the metabolic rate in an attempt to maintain normothermia. All individuals are not capable of the same degree of elevation of the metabolic rate, such as the elderly. Other individuals, who may not be able to support an increase in oxygen consumption, are those individuals with oxygen delivery problems, such as individuals with limited cardiac outputs, with anemia, or with oxygenation problems. Hence, the development of hypothermia in some individuals will occur more rapidly.

Summary. A physiological or pathophysiological alteration of any of the individual factors will result in an alteration in the individual's ability to

thermoregulate. Chronic vasoactivity problems are seen in spinal cord injury victims, individuals with peripheral neuropathies, individuals with central brain injury or disease which affects the hypothalamus, and the aged. Acute vasoactivity changes are seen in individuals who have decreased cardiac outputs from decreased vascular volume. Individuals with a decrease in metabolic rate such as decreased thyroid function also have greater difficulty in maintaining a normal central temperature.

The individual factors of vasoactivity, conductance, body surface-to-body mass quotient and metabolic rate influence the individual's rate of hypothermia development when exposed to a cool or cold environment. The same individual factors are in effect when attempting to rewarm an individual. Hence, the individual factors must be considered in the choice of rewarming therapies if an effective rewarming process is desired. The environmental factors will be discussed in the next section.

### Environmental Factors

The environmental factors include convection, conduction, radiation and evaporation. All of the environmental factors may impact the body heat loss depending upon a temperature gradient in which the environment is cooler than the body. Convection, conduction and radiation may impact the body heat gain depending upon a temperature gradient in which the environment is warmer than the body.

Convection. Heat transfer based on convection is dependent upon the surface area exposed to the environment, the convection constant which varies with air movement, and the body surface to ambient temperature gradient (Convective heat transfer = Surface area exposed x Convection constant x Body surface to ambient temperature gradient; Bregelmann, 1989).

The convection constant is the amount of heat removed from an area, with a given rate of air movement, during a given time period. In a normal indoor environment, the convection constant is  $5.2 \text{ kcal/hr m}^2 \text{ }^\circ\text{C}$ . Tripling the air velocity, which simulates air movement while walking at a 4 miles/hr pace, approximately doubles the constant to  $9.8 \text{ kcal/hr m}^2 \text{ }^\circ\text{C}$ . Hence, the rate of heat loss is affected by the rate of air movement around the individual.

Therefore, both convection constant and temperature gradient have major effects on the rate of heat loss from the body.

In the surgical suite, the heat exchange from the body to the environment by convection is increased. Depending upon the type of surgery, an increased amount of the body surface and internal organs are exposed to ambient temperature ( $T_{amb}$ ). The surgical suite has a special air control system in which the room air is exchanged for fresh air which results in a higher than usual room air convection constant. Furthermore, the ambient temperature in surgical suites may be controlled within the range of  $17$  to  $24^\circ \text{C}$ . Hence, convection plays a role in the development of hypothermia in individuals undergoing surgery.

Conduction. Conductive heat transfer is based upon the thermal conductivity constant of the surfaces, the body surface area exposed and the temperature gradient of the two surfaces (Conductive heat transfer = Conductivity constant x Surface area exposed x Temperature gradient; Burton, 1934). Conduction generally does not have as great an effect upon heat exchange, because only a small amount of the body surface area is in direct contact with the conductive surface. For example, when an individual is reclining on a firm surface, less than one-third of the body surface, based on the Rule of Nines for calculating body surface area, is actually in contact with

the surface (Morris & Kumar, 1972). Nilsson (1987) and Sessler, Stoen and Glosten (1991) estimated that only 3 to 11% of the body heat lost in a reclining individual is lost via conduction.

In the operative suite, heat loss via conduction remains low since the individual is placed on a foam mattress which has a low conductive constant. Only a small area of the body surface is in contact with the foam mattress. Furthermore, the temperature gradient between the foam mattress and the body is decreased over time, since the mattress surface temperature will equilibrate with the skin temperature.

Radiation. Radiant heat transfer is dependent upon the radiant constant, the body surface area exposed and the temperature gradient between the skin and the radiant surfaces, such as walls, (Radiant heat transfer = Radiant constant x Body surface area exposed x Skin surface to radiant surface temperature gradient; Brengelmann, 1989). The radiant constant for ordinary indoor temperatures is  $5 \text{ kcal/hr/m}^2 \text{ }^\circ\text{C}$  (Brengelmann, 1989).

In the operating suite, the environmental surfaces, such as the walls, are cool. The body temperature is higher than the wall temperature resulting in a heat gradient from the body to the walls. Therefore, the individual with skin exposure in the operating suite will radiate heat from the body to the walls. If the wall temperature was the same as the body temperature, the radiant exchange would become neutral and the net heat flux would be zero. In environments, where there is not forced convective heat loss or intense radiant sources, approximately one-half of the non-evaporative heat loss is from radiant loss (Brengelmann, 1989).

Evaporation. In the thermoregulatory process, evaporative heat-loss occurs with the evaporation of water from the skin surface. Approximately 600 kcal

of heat are lost per liter of water which evaporates from the skin. In a normal individual, an elevation of the central temperature initiates sweating and depending upon the environmental conditions the sweat evaporates eliminating heat from the body. In an intraoperative situation, body heat is lost as the water evaporates from the moist body surfaces exposed to the environment. In a hypothermic postoperative individual, evaporation from the skin surface, assuming the skin is dry, is not of concern during rewarming.

Furthermore, evaporation is evidenced in normal insensible heat loss from the respiratory tract and through the skin. Approximately one liter of fluid is lost in a 24 hour period via insensible loss evaporation (Bregelmann, 1989), which amounts to 600 kcal of heat lost over a 24 hour period.

Summary. The individual and environmental factors affect the individual's ability to maintain normothermia. Table 2 demonstrates the effect of the factors on two different individuals in a 20<sup>o</sup> C environment. The larger individual (individual 1) has a greater heat production as well as a greater amount of heat loss. However, the smaller individual (individual 2) actually loses more heat than they are producing which would result in a decrease in central temperature over time. The example indicates the larger individual would actually have an increase in body temperature over time. Hence, both individual and environmental factors must be considered when normothermia is to be maintained. Furthermore, once hypothermia has occurred, both groups of factors must be considered when selecting a rewarming therapy. The following section will discuss hypothermia.

TABLE 2. Example of Individuals in a Cool Environment.

Factor	Individual 1	Individual 2
Weight	70 kg	50 kg
Height	1.78 m	1.68 m
Vasoconstriction	Equal	Equal
Tissue conductivity	Equal	Equal
Absolute thickness	Equal	Equal
Body surface to mass	0.027	0.031
Metabolic rate	3.5 ml O <sub>2</sub> /kg minute	3.5 ml O <sub>2</sub> /kg minute
Heat gain	70.6 kcal/hr	50.4 kcal/hr
BSA exposed	33%	33%
Core temperature	37 <sup>o</sup> C	37 <sup>o</sup> C
Skin temperature	30 <sup>o</sup> C	30 <sup>o</sup> C
Ambient temperature	20 <sup>o</sup> C	20 <sup>o</sup> C
Convective heat loss	32.76 kcal/hr	27.04 kcal/hr
Radiant heat loss	31.50 kcal/hr	26.00 kcal/hr
Heat loss (total)	64.26 kcal/hr	53.04 kcal/hr

### Hypothermia

In the literature, the definition of hypothermia varies from any amount of decrease in central temperature to a central temperature below a given degree of temperature. Gentilello, Jurkovich and Moujaes (1991) classified hypothermia as mild if the individual had a core temperature of 32<sup>o</sup> to 35<sup>o</sup> C, moderate with a core temperature of 28<sup>o</sup> to <32<sup>o</sup> C and severe if the core temperature was 20<sup>o</sup> to < 28<sup>o</sup> C. Gentilello et al. (1991) utilized 32<sup>o</sup> C as the beginning point of hypothermia, because cardiac conduction disturbances became evident at that temperature. However, other physiological changes are noted at temperatures greater than 32<sup>o</sup> C. For example, Hayward et al. (1984) reported an increase in systemic vascular resistance in a subject with a pulmonary artery blood temperature of 35.9<sup>o</sup> C; Boelhouwer et al. (1987)

reported the development of hypokalemia in individuals with rectal temperatures of  $36.5^{\circ}\text{C}$  or less; and Slotman et al. (1985) reported a significantly higher percent mortality rate in individuals who had a central temperature, specific site not identified, of less than  $36.1^{\circ}\text{C}$  for 2, 4 and 8 hours post-operatively. Since physiological changes have been identified at a central temperature of  $36.5^{\circ}$ , a central temperature of less than  $36.5^{\circ}\text{C}$  will be defined as hypothermia. The issue of the site of central temperature remains. Therefore, the next sections will discuss central temperature measurement sites.

### Central Temperature

Central or core temperature is the site which represents the heat balance of the entire body. The rate of heat production varies between the various organs of the body during rest (Graf, 1959; Brengelmann, 1989) and during exercise (Rowell, 1986). Hence, a site where the temperature is representative of the whole body heat load not the heat load of a specific organ is required. Furthermore, in the literature, the argument is posed that the central temperature site should be the temperature of the hypothalamus, the site of body temperature regulation. With the hypothalamus located within the bony structure of the cranium, the direct measurement of hypothalamic temperature is impossible on a routine basis. Hence, a site that is reflective of the hypothalamus, representative of the body heat load, but not overly influenced by a local organ, and accessible is needed. The pulmonary artery blood temperature ( $T_{pa}$ ) is a site in which the temperature is within  $0.01^{\circ}\text{C}$  of the hypothalamic temperature (Hayward and Baker, 1969; Eichna, Berger, Rader, & Becker, 1951; Mather, Nahas, & Hemingway, 1953). The pulmonary artery blood is a site which is representative of a mixing of the blood

temperatures from all organs of the body, which may have varying metabolic rates. Hence, the pulmonary artery blood temperature can be utilized as representative of the body heat load and similar to the temperature of the hypothalamus. Unfortunately, the pulmonary artery is not readily accessible for temperature measurement. Hence, other common sites of central temperature measurement are bladder (T<sub>bl</sub>), esophagus (T<sub>es</sub>), tympanic membrane (T<sub>ty</sub>), rectum (T<sub>re</sub>) and oral cavity (T<sub>or</sub>). These sites will be discussed in the following paragraphs.

Bladder temperature. The bladder temperature is similar to T<sub>pa</sub> in resting, normothermic, intensive care (ICU) patients. The bladder temperature was reported to vary from 0.26<sup>o</sup> C greater than to 0.22<sup>o</sup> C less than T<sub>pa</sub> (Tables 3, 4, and 5) in ICU patients.

TABLE 3. Temperature (<sup>o</sup> C) Sites in Non-Surgical Intensive Care Patients.

Nierman (1991) n=15	T <sub>pa</sub>	T <sub>pa</sub> -T <sub>bl</sub>	T <sub>pa</sub> -T <sub>ty</sub> <sup>a</sup>
ICU resting, 32 hours of measurement	37.69	-0.04	-0.38
		r <sup>2</sup> = 0.83	r <sup>2</sup> = 0.67

Note. T<sub>pa</sub> = pulmonary artery temperature; T<sub>pa</sub>-T<sub>bl</sub> = pulmonary artery - bladder temperature; T<sub>pa</sub>-T<sub>ty</sub> = pulmonary artery - tympanic temperature.

<sup>a</sup>Infrared auditory canal temperature

In the hypothermic (T<sub>pa</sub> < 36.5<sup>o</sup> C) intra-operative or ICU patient not on cardio-pulmonary bypass (CPB) or extracorporeal rewarming, the bladder temperature varied from 0.60<sup>o</sup> C greater than to 0.26<sup>o</sup> C less than T<sub>pa</sub> (Tables 4 and 5). However, the bladder temperature was generally no more than 0.25<sup>o</sup> C greater- or less-than pulmonary artery temperature. The difference of 0.6<sup>o</sup> C between the two sites was seen following removal from CPB and afterdrop occurred (Ramsay, Ralley, Whalley, DelliColli, & Wynands, 1985) resulting in a pulmonary artery temperature of 34.7<sup>o</sup> C (Table 4). The extracorporeal rewarming period is difficult to interpret since the studies do not indicate

which vessels were cannulated which may have a direct effect on the relationship of various temperature sites. Following extracorporeal rewarming, the rewarming process generally was not clearly described.

TABLE 4. Temperature ( $^{\circ}$  C) Sites in Intra-Operative Cardiac Surgery Patients.

Lilly et al. (1980) n=13	T <sub>pa</sub>	T <sub>pa-Tbl</sub>	T <sub>pa-Tes</sub>	T <sub>pa-Tre</sub>
Start of extracorporeal rewarming	29.7	0.20	-0.30	-0.80
Extracorporeal rewarming for 30 min	36.9	0.40	0.70	1.40
Correlation of temperatures during unassisted circulation	$r^2 = 0.91$			
Moorthy et al. (1985) n=12	T <sub>pa</sub>	T <sub>bl</sub>	T <sub>es</sub>	T <sub>re</sub>
Prior to cooling for surgery		35.6	35.9	35.4
Cooling for 45 min		27.4	24.0	28.1
Prior to intra-operative rewarming		26.3	24.7	26.0
Intra-operative rewarming for 75 min		34.8	36.9	33.1
Intensive care unit mean temperatures over 16 hours	37.2	37.1		37.5
Ramsay et al. (1985) n=29	T <sub>pa</sub>	T <sub>pa-Tbl</sub>	T <sub>pa-Tes</sub>	T <sub>pa-Tre</sub>
Start of intra-operative rewarming	26.8	-1.6		
Intra-operative rewarming for 50 min	36.3	2.1		
Removed from cardio-pulmonary bypass (CPB)	36.6	1.1		
20 min after removal from CPB	35.4	0.0		
60 min after removal from CPB	34.7	-0.6		
90 min after removal from CPB	34.9	-0.4		
150 min after removal from CPB	35.5	-0.1		
210 min after removal from CPB	36.5	0.1		
240 min after removal from CPB	36.8	-0.1		

Note. T<sub>pa</sub> = pulmonary artery temperature; T<sub>bl</sub> = bladder temperature; T<sub>es</sub> = esophageal temperature; T<sub>re</sub> = rectal temperature.

Upon review of the graphed temperatures of T<sub>pa</sub> and T<sub>bl</sub>, the two temperatures do track one another across the rewarming process (Earp & Finlayson, 1991; Isley, Rutten, & Runciman, 1983; Lilly, Boland, & Zekan, 1980; Ramsay, et al., 1985). Furthermore, T<sub>pa</sub> and T<sub>bl</sub> temperatures are highly correlated,  $r^2$  ranged from 0.83 to 0.98 (Table 3, 4 and 5). The bladder is an accessible site for temperature measurement in any individual who receives an indwelling catheter during the normal care process. Furthermore, T<sub>bl</sub> is

representative of T<sub>pa</sub>. Therefore, T<sub>bl</sub> is a valid site for central temperature measurement.

TABLE 5. Temperature ( $^{\circ}$  C) Sites in Postoperative ICU Cardiac Surgery Patients.

Mravinac et al. (1989) n=55	T <sub>pa</sub>	T <sub>pa</sub> -T <sub>bl</sub>	T <sub>pa</sub> -T <sub>re</sub>
Post-surgery admission to ICU and stable vital signs (VS) <sup>a</sup>	33.53	0.01	0.22
One hour after stable VS	33.91	0.11	0.30
Two hours after stable VS	34.80	0.26	0.36
Three hours after stable VS	35.81	0.24	0.31
Four hours after stable VS	36.63	0.17	0.15
Five hours after stable VS	37.00	0.06	0.09
Six hours after stable VS	37.23	-0.02	-0.09
Earp et al. (1991) n=14	T <sub>pa</sub>	T <sub>pa</sub> -T <sub>bl</sub>	T <sub>pa</sub> -T <sub>re</sub>
Post-surgery admission to ICU <sup>b</sup>	35.19	-0.22	
		r=0.98	
One hour after admission	35.51	-0.06	
		r=0.99	
Two hours after admission	36.03	0.04	
		r=0.99	
Three hours after admission	36.59	-0.04	
		r=0.99	
Four hours after admission	37.04	-0.09	
		r=0.97	
Five hours after admission	37.21	-0.17	
		r=0.96	
Six hours after admission	37.46	-0.14	
		r=0.95	

Note. T<sub>pa</sub> = pulmonary artery temperature; T<sub>bl</sub> = bladder temperature; T<sub>re</sub> = rectal temperature.

<sup>a</sup>T<sub>amb</sub> 22 to 22.7 $^{\circ}$  C, intubated receiving 32.76 to 35.40 $^{\circ}$  C humidified oxygen, 42 $^{\circ}$  C water filled warming blankets anterior and posterior of patient. <sup>b</sup>T<sub>amb</sub> 23 $\pm$ 1 $^{\circ}$  C, intubated receiving 70% oxygen, reflective thermal hat on head, two warmed blankets placed over the body upon admission to ICU.

Esophageal temperature. Esophageal temperature (T<sub>es</sub>) is similar to T<sub>pa</sub> in resting healthy individuals (Table 6) and patients with metastatic cancer (Table 7). The difference of temperature between T<sub>pa</sub> and T<sub>es</sub> remained within 0.25 $^{\circ}$  C in the healthy individual during cooling and rewarming (Hayward et al., 1984). However, during cooling and heating of the metastatic cancer

patients (Table 7), the temperature difference between the two sites was  $\pm 0.4^{\circ}$  C. The warming therapy was extracorporeal warming with the blood removed from the femoral vein and returned into the femoral artery at a temperature of  $43^{\circ}$  C. The authors estimated the warmed blood was delivered as high as the renal artery in the descending aorta, which negates the potential that the returned warmed blood was having an effect on one site, but not on the other. In both studies the esophageal thermistor was positioned at the cardiac level and verified by roentography.

TABLE 6. Temperature ( $^{\circ}$  C) Sites in Non-surgical Subjects.

Hayward et al. (1984) n=1	Tpa	Tpa-Tes	Tpa-Tre	Tpa-Tty <sup>a</sup>
Resting Tamb 21 to 22 $^{\circ}$ C	37.15	0.25	-0.15	0.30
10 $^{\circ}$ C bath for 100 min	35.90	0.20	-0.08	0.40
Self rewarming for 60 min	36.70	0.15	0.45	0.35
Inhalation of 43 to 45 $^{\circ}$ C saturated air for 60 min	37.30	0.10	0.40	0.20
38 to 39 $^{\circ}$ C bath for 60 min	37.80	-0.08	-0.60	-0.30

Note. Tpa = pulmonary artery temperature; Tes = esophageal temperature; Tre = rectal temperature; Tty = tympanic temperature.

<sup>a</sup>Thermocouple placed close to tympanum.

Hence, esophageal temperature is accessible, but for proper positioning of the probe at the cardiac level requires either roentography or electrocardiography. Furthermore, the variation of temperature seen in the active rewarming of the metastatic patients is concerning. Esophageal temperature is not used as a standard site of temperature measurement in the clinical area. Therefore, for clinical purposes, a site which has fewer requirements for proper positioning is appropriate. However, for laboratory purposes, T<sub>es</sub> is a valid measurement in non-metastatic cancer patients.

TABLE 7. Temperature ( $^{\circ}$  C) Sites in Intra-operative Hyperthermic Therapy in Patients with Metastatic Cancer.

Shiraki et al. (1986) n=3	T <sub>pa</sub>	T <sub>pa-Tes</sub>	T <sub>pa-Tre</sub>	T <sub>pa-Tty</sub> <sup>a</sup>
Temperature before therapy started	36.6	-0.1	0.0	0.3
20 min of warming <sup>b</sup>	39.2	0.4	-1.0	0.9
40 min of warming	40.0	0.1	-1.2	0.5
80 min of warming	41.1	-0.2	-0.6	0.5
600 min of warming	41.5	-0.2	-0.9	0.4
20 min of cooling <sup>c</sup>	38.1	-0.4	1.4	-0.4
20 min of warming <sup>d</sup>	38.0	-0.2	0.6	0.4
40 min of warming	38.4	-0.2	0.1	0.4

Note. T<sub>pa</sub> = pulmonary artery temperature; Tes = esophageal temperature; Tre = rectal temperature; T<sub>ty</sub> = tympanic temperature.

<sup>a</sup>Thermocouple in contact with tympanum. <sup>b</sup>Extracorporeal circuit of right femoral vein through heater to right femoral artery at 43 $^{\circ}$  C. <sup>c</sup>Extracorporeal circuit at 34 $^{\circ}$  C. <sup>d</sup>Extracorporeal circuit at 36 $^{\circ}$  C.

Tympanic temperature. Tympanic temperature (T<sub>ty</sub>) had variable results when compared with T<sub>pa</sub> in a resting healthy subject, ICU- and cancer-patients (Tables 3, 6 and 7). The difference between the temperatures with infrared technology was -0.38 $^{\circ}$  C in ICU patients and -0.30 to 0.40 $^{\circ}$  C in non-surgical subjects with thermocouple technology. The correlation of T<sub>ty</sub> and T<sub>pa</sub> in infrared technology was moderate ( $r^2=0.67$ ; Nierman, 1991). In part the difference between T<sub>pa</sub> and T<sub>ty</sub> may be caused by the thermistor not being in contact with the tympanic membrane (Benzinger, 1969). However, McCaffrey, McCook and Wurster (1975) demonstrated an effect on the tympanic temperature when local cooling and heating were applied to the head. Therefore, the T<sub>ty</sub> may be accessible, but the reliability and validity of this site as central temperature is questionable. Furthermore, positioning is vital for proper temperature measurement and the positioning technique is not a part of clinical practice. Therefore, T<sub>ty</sub> is not an acceptable site of temperature measurement in the clinical area.

Rectal temperature. Rectal temperature ( $T_{re}$ ), in the resting state of the healthy subject and the cancer patients (Tables 6 and 7), is similar to  $T_{pa}$ . However, with cooling and rewarming, the difference between the two temperatures ranged from  $-1.2$  to  $1.4^{\circ}\text{C}$  (Tables 5, 6 and 7). The great difference in rectal and pulmonary artery temperatures in Shiraki, Konda and Sagawa (1986) study may be related to the extracorporeal rewarming process. However, Hayward et al. (1984) also reported a difference of  $-0.60$  to  $0.45^{\circ}\text{C}$  between  $T_{pa}$  and  $T_{re}$  with non-invasive rewarming therapies. Furthermore, the rate of change of rectal temperature is known to be slower with cooling (Hayward et al., 1984; Table 8) and warming, as well as it may overshoot the actual central temperature. Cork, Vaughan and Humphrey (1983) monitored multiple temperature sites including  $T_{re}$  with anesthesia induction and following induction.  $T_{re}$  consistently lagged behind  $T_{bl}$  and  $T_{ty}$  across the 120 minute time period of the study (Table 8). Therefore, although rectal temperature is accessible,  $T_{re}$  is not a reliable and valid measurement of central temperature.

TABLE 8. Temperature ( $^{\circ}\text{C}$ ) Sites in Intra-Operative Patients (Non-Cardiac).

Cork et al. (1983) n=56	$T_{bl}$	$T_{re}$	$T_{ty}$
Temperature at time of induction	36.4	37.0	36.5
30 min after induction	36.3	36.4	36.2
60 min after induction	35.5	36.2	35.7
90 min after induction	35.1	36.0	35.4
120 min after induction	34.7	35.8	35.3

Note.  $T_{bl}$  = bladder temperature;  $T_{re}$  = rectal temperature;  $T_{ty}$  = tympanic temperature.

Oral temperature. Oral temperature, in resting ICU patients, varied from  $0.3$  to  $0.8^{\circ}\text{C}$  from  $T_{pa}$  (Table 9). McCaffrey et al. (1975) reported a change in oral temperature with the application of heat or cold to the head. The oral temperature change was not as great as the change seen in the tympanic

temperature. Furthermore, oral temperature is affected by thermistor positioning and ingestion of cold or warm fluids (Erickson, 1980; Brim & Chandler, 1948; Forster, Adler, & Davis, 1970). Therefore,  $T_{or}$  is accessible, but is not a reliable and valid measurement of central temperature. Furthermore, in the postoperative patient, the level of cooperation with oral temperature measurement may be limited.

TABLE 9. Temperature ( $^{\circ}$  C) Sites in Pre- and Post-Operative Patients in ICU.

Isley et al. (1983) n=5	$T_{pa}$	$T_{pa-Tbl}$	$T_{pa-Tor}^a$	$T_{pa-Tre}$
Pre-operative ICU measurements for patient #1	37.5		0.8	
for patient #2	36.8		0.3	
for patient #3	37.0		0.5	
for patient #4	36.8		0.3	
for patient #5	36.9		0.4	
Post-operative ICU measurements over 24 hours for patient #1		0.30		0.23
for patient #2		0.40		0.30
for patient #3		0.27		
for patient #4		0.14		
for patient #5		0.25		-0.07

Note.  $T_{pa}$  = pulmonary artery temperature;  $T_{bl}$  = bladder temperature;  $T_{or}$  = oral temperature;  $T_{re}$  = rectal temperature.

<sup>a</sup> $T_{or}$  measured with an electronic thermistor-type thermometer with digital display.

Hence, accessible temperature measurement sites which are estimates of central temperature are  $T_{bl}$  and  $T_{es}$ . In those patients, who have an indwelling bladder catheter for other reasons, the measurement of central temperature via the bladder is appropriate. However, an indwelling bladder catheter does increase the patient's risk of infection. Therefore, if the catheter is not required for other medical reasons, the bladder site would not be appropriate.  $T_{es}$  is not routinely monitored in clinical areas other than the operative and post anesthesia care units. Positioning the  $T_{es}$  probe does require roentography or use of an electrocardiogram. Therefore, both  $T_{bl}$  and  $T_{es}$  sites

have draw backs, but are the most accurate sites of central temperature measurement.

#### Operative Cooling and Rewarming Factors

The intra-operative, hypothermia literature identified age, the route of anesthesia administration, body fat, and body mass index as factors which had a statistically significant effect in individuals who developed hypothermia, compared to individuals who did not develop hypothermia. In the physiology literature, a difference in gender skin temperature was identified as significant. The factors which had a significant effect on postoperative rewarming were age, anesthesia route and amount of temperature decline. Table 10 identifies both statistically significant and nonsignificant factors, which have been studied in relation to the rate of cooling and of rewarming in the post-operative individual who develops hypothermia.

TABLE 10. Factors which Affect the Rate of Cooling and Rewarming.

<u>Factors Studied</u>	<u>Cooling</u>	<u>Rewarming</u>
Significant Effect	Age	Age
	Ambient temperature	Anesthesia route
	Anesthetic route	Lowest induced temperature
	Body fat	T <sub>co</sub> admission into recovery
	Body Mass Index	
	Duration of surgery	
	Gender	
Insignificant Effect	Operative site	
	Duration of fasting	Body fat
	Ethnic origin	Duration of surgery
	Volume of fluids infused	Operative site
	Circadian rhythm	Circadian rhythm

Note. T<sub>co</sub> = central body temperature.

The following section will discuss the significant factors and the circadian effect on rewarming.

### Age

The impact of age, on the decline of central temperature during operative procedures, has yielded varying results. Roe et al. (1966) and Goldberg and Roe (1966) in descriptive studies reported a greater decline in central temperature ( $T_{re}$ ) as the age of the subjects increased (24 and 101 subjects, respectively). Roe et al. reported a decline in  $T_{re}$  of  $0.6^{\circ}\text{C}$  in the 21 to 40 years old age group compared to a  $1.3^{\circ}\text{C}$  in the 61 to 80 years old age group. Closs, Macdonald, and Hawthorn (1986) reported a significant correlation between the magnitude of temperature decline and age ( $r = .5357$ ,  $p < 0.001$ ). However, Morris and Wilkey (1970) and Morris (1971b) reported no significant correlation between body temperature and the patient's age. Morris and Wilkey's and Morris' sample sizes were 17 and 21, respectively. Furthermore, in both studies the  $T_{amb}$  varied between  $17^{\circ}\text{C}$  and  $24^{\circ}\text{C}$  which would also have an effect on the body temperature. Hence, the small sample size as well as the lack of environmental control probably account for the insignificant findings. Therefore, the literature suggests that an age effect exists in relation to the decline in central temperature during surgery.

Age impacts the rate of rewarming in postoperative individuals. The rate of self rewarming, defined as  $^{\circ}\text{C/hr}$  central temperature change with no external heat source, was significantly ( $p < 0.05$ ) less in individuals over 60 years of age than individuals under 60 years of age (Carli, Gabrielczyk, Clark, & Aber, 1986). Shock et al. (1963) studied the basal metabolic rate across the life span and found a decreased metabolic rate in those individuals over 60 years of

age which would support the findings of Carli, Gabrielczyk et al. Therefore, age is a factor which affects both the rate of cooling and rewarming.

The effect of age on the development of hypothermia and rewarming may not be limited to a lower metabolic rate. In individuals older than 70 years of age, Braverman (1989) reported the vessel walls were thinner, 0.5 to 1.0 micrometer, than the expected adult vessel wall thickness, 2 to 3 micrometers. In preliminary studies of the local vasoactive response to heating, those individuals 60 years of age or older had a lower maximal vasodilation (12 to 15 ml/minute/100 grams of tissue) than younger individuals (15 to 30 ml/minute/100 grams of tissue; Audiss, Brengelmann & Savage, unpublished). The alteration in the vessel wall may impact the vasoactive portion of thermoregulation. Hence, both metabolic and vasoactivity changes may affect the ability of individuals over 60 years of age to remain normothermic or to rewarm to a normothermic state.

#### Ambient Temperature

Theoretically, if the temperature gradient between ambient temperature ( $T_{amb}$ ) and body temperature is great enough, the individual's heat production may be less than the rate of heat loss resulting in hypothermia. Newton's simplified Cooling Law is  $Heat\ loss = C (T_{sk} - T_{amb})$  where C is the thermal conductance of the air around the body,  $T_s$  is the temperature of the skin and  $T_{amb}$  is the temperature of the air (Hardy, 1961). Hence, with a larger temperature gradient between the skin and the air a greater amount of heat is lost.

Temperature gradient between  $T_{amb}$  and body temperature was found to have a significant effect ( $p < 0.007$ ) on the reduction in core temperature (Closs et al., 1986;  $n = 31$  operative patients). Morris (1971a) studied 45 surgical

patients with an age range of 20 to 85 years who underwent intra-abdominal and extra-abdominal surgeries and found a significant relationship ( $p < 0.001$ ) between the declining  $T_{es}$  and  $T_{amb}$ . The same effect, of  $T_{amb}$  and body temperature gradient, would be expected during the rewarming process. Hence, the rate of cooling and rewarming is affected by the temperature gradient between the environment and the body.

### Anesthesia

Anesthetic agents affect thermoregulation by altering vasoactivity and by altering mechanisms which increase the metabolic rate. Vasodilation of the skin has been reported in patients receiving combinations of volatile anesthetics as well as in individuals receiving epidural blocks (Table 11). Furthermore, the central vasoconstrictive response to hypothermia appears to be depressed in some individuals receiving general anesthetics. It is unknown whether the effect on vasoactivity is a result of a central effect or a more local effect. Other anesthetic agents, such as paralytics, actually prevent an increase in metabolic rate via prevention of shivering. However, all drugs within a category do not have the same effect upon thermoregulation.

Anesthesia route. Individuals receiving general anesthesia cool less intra-operatively over a longer period of time and rewarm quicker post-operatively than those receiving epidural anesthesia. Stjernstrom, Henneberg, Eklund, Tabow, Arturson, and Wiklund (1985) studied 25 transurethral prostate resection patients, who received general anesthesia (incremental doses of fentanyl 0.05 to 0.1 mg, pancuronium 1-2 mg and nitrous oxide) or epidural analgesia (bupivacaine 0.5%). The groups were similar for age and body weight. At 1 hour intra-operatively, the core temperature (aural external meatus, electronic thermometer) and the mean body temperature for both

groups had declined  $1^{\circ}\text{C}$  with no significant difference between the two groups. However, Holdcroft, Hall, and Cooper (1979) studied 52 patients, with lower abdominal operations for infertility, who received either general anesthesia (fentanyl 10 mcg/kg, fentanyl 50 mcg/kg, halothane 0.5%, or halothane 1%) or epidural analgesia (bupivacaine 0.5%). The five groups were similar in weight, height and age. At one hour of anesthesia, the central temperature (aural, methodology not described) declined  $0.3^{\circ}\text{C}$  in the fentanyl 10 mcg/kg group, and  $0.9^{\circ}\text{C}$  in the fentanyl 50 mcg/kg and epidural groups. However, the rate of decline in the epidural group remained at a faster rate than any of the general anesthesia groups, resulting in a significantly lower temperature in the epidural group between the second and third hours of anesthesia.

Stjernstrom et al. (1985) measured the oxygen consumption rate pre-, intra-, and post-operatively in the general anesthesia and the epidural groups. The pre-operative oxygen consumption rate was 200 ml/min in both groups. The general anesthesia group's oxygen consumption declined by 15% following induction of anesthesia and maintained at approximately the same decreased level through out the surgery. However, the epidural group did not have a decline in oxygen consumption during surgery. Furthermore, post-operatively, the general anesthesia group had a 40% increase in oxygen consumption, while the epidural group maintained the same rate of oxygen consumption through out the entire study.

Stjernstrom et al. (1985) reported a significant increase in mean skin temperature in the epidural group after the onset of the epidural block which did not occur in the general anesthesia group. Therefore, the greater decline

in central temperature, in the epidural group at 3 hours, is probably related to a greater heat loss via a greater vasodilation of the skin in the epidural group.

The post-operative rate of rewarming is 2 to 3 times faster in the general anesthesia patient than in the epidural analgesia patient at 1 hour post-operative (Stjernstrom et al., 1985; Vaughan et al., 1981; Carli, Gabrielczyk et al., 1986). Holdcroft et al. (1979) reported no statistical difference in the rate of rewarming of general and epidural groups at 30 minutes post-operatively. However, the actual rise in temperature of the epidural group was  $0.3^{\circ}\text{C}$  compared to  $0.7$  to  $1.2^{\circ}\text{C}$  in the four different general anesthesia groups. Furthermore, Vaughan et al. (1981) reported a rise in  $T_{\text{ty}}$  of  $0.4^{\circ}\text{C}$  at 30 minutes in the general anesthesia group (medication not reported) compared to a  $T_{\text{ty}}$  rise of  $0.1^{\circ}\text{C}$  in the epidural group (medication not reported). Hence, the statistical results reported by Holdcroft et al. may be an issue of sample size. Therefore, the literature supports a greater heat loss and a slower heat gain in the epidural analgesia operative patients when compared to general anesthesia operative patients. The cause of a greater heat loss in the epidural operative patients may be due to vasodilation with the epidural block. The postoperative, slower body heat gain may be related to no increase in metabolic rate post-operatively.

Anesthesia type. The effect of anesthetics upon an individual's ability to thermoregulate is difficult to study. However, Crocker, Okumura, McCuaig, and Denborough (1980) studied the effect of anesthesia on central temperature in 2410 patients (Table 11). Crocker et al. found that individuals receiving ketamine, suxamethonium and halothane, and halothane and nitrous oxide had significantly higher  $T_{\text{es}}$  temperatures than those individuals receiving other agents. Crocker et al. discussed the implication of suxamethonium and

halothane in malignant hyperthermia cases and suggested that the temperature elevations seen in subjects receiving ketamine may have been from a similar response.

TABLE 11. Anesthetic Effects on Thermoregulation.

Medication	Study findings
<b>Premedications</b>	
Hyosine Atropine Papaveretum Pethidine Morphine	<sup>a</sup> No significant difference in $T_{es}$ or $T_{re}$ between the subjects receiving and those not receiving belladonna agents. $T_{es}$ or $T_{re}$ declined by $0.3^{\circ}\text{C}$ over the first hour of surgery. <sup>b</sup> Postoperatively, significantly lower metabolic rate and rate of rewarming in individuals receiving continuous drip MS versus those receiving intermittent dose MS
<b>Non-inhalation anesthetics and adjuvants</b>	
Thiopentone Ketamine Propanidid Althesin Methohexidone Fentanyl Droperidol Chlorpromazine Diazepam	<sup>a</sup> Significantly higher $T_{es}$ and $T_{re}$ in subjects receiving ketamine as compared to subjects receiving thiopentone. With ketamine administration, $T_{es}$ increased during the first 30 minutes of anesthesia while $T_{re}$ increased through out the entire 60 minutes of observation. No abnormal temperature profiles were noted in patients receiving non-inhalation anesthetics other than ketamine.
<b>Neuromuscular blockers</b>	
<b>Depolarizing</b>	
Suxamethonium	<sup>a</sup> The patients receiving depolarizing blocker and halothane had significantly higher $T_{es}$ and $T_{re}$ in the first 30 to 45 minutes of surgery than the patients receiving non-depolarizing blockers, no neuromuscular blockers or suxamethonium without halothane.
<b>Non-depolarizing</b>	
Alcuronium Pancuronium Tubocurarine Gallamine Vecuronium	<sup>a</sup> No significant difference in $T_{es}$ and $T_{re}$ in those individuals receiving non-depolarizing blockers and those who received no neuromuscular blockers (Vecuronium not included in this study).

Table 11 (continued).

Inhalants	
Nitrous oxide Halothane Isoflurane	<p><sup>a</sup>Patients receiving halothane and nitrous oxide had significantly greater <math>T_{es}</math> at 20 and 30 minutes than those patients who received only halothane.</p> <p><sup>c</sup>Volunteers receiving isoflurane who were hypothermic as compared to those who were maintained in a state of normothermia did not have significantly different forearm - fingertip temperature gradients. During recovery, the hypothermic volunteers had a significantly higher forearm - fingertip temperature gradient than the normothermic anesthetic group at all levels of isoflurane.</p> <p><sup>d</sup>Volunteers received nitrous oxide and isoflurane for induction and immediately had a significant increase in finger blood flow and a decrease in the forearm - fingertip gradient.</p> <p><sup>e</sup>Patients receiving isoflurane anesthesia became hypothermic and developed significant vasoconstriction at central temperatures between 35.3 and 32.4<sup>o</sup> C as compared to volunteers exposed to the same environment without anesthesia who developed significant vasoconstriction at central temperatures between 36.8 and 37.6<sup>o</sup> C. The larger the end tidal concentration of isoflurane the greater length of time before the patient experienced significant vasoconstriction.</p> <p><sup>f,g</sup>Significant vasoconstriction was noted in individuals anesthetized with nitrous oxide and fentanyl or with halothane when the central temperature reached approximately 35<sup>o</sup> C or less.</p>

Note.  $T_{es}$  = esophageal temperature;  $T_{re}$  = rectal temperature.

<sup>a</sup>Crocker, Okumura, McCuaig, and Denborough, (1980). <sup>b</sup>Rodriguez, Weissman, Damask, Askanazi, Hyman, and Kinney, (1983). <sup>c</sup>Sessler, Rubinstein, and Moayeri, (1991). <sup>d</sup>Sessler, McGuire, Moayeri, and Hynson, (1991). <sup>e</sup>Stoen, and Sessler, (1990). <sup>f</sup>Sessler, Olofsson, and Rubinstein, (1988). <sup>g</sup>Sessler, Olofsson, Rubinstein, and Beebe, (1988).

Furthermore, Sessler, Rubinstein, and Moayeri, A. (1991), Sessler, McGuire, Moayeri, and Hynson (1991), Stoen, and Sessler (1990), Sessler, Olofsson, and Rubinstein (1988), Sessler, Olofsson, Rubinstein, and Beebe (1988) reported vasodilation of the skin vasculature with nitrous oxide and isoflurane

administration and a delayed onset of vasoconstriction in relation to central temperature decline. Hence, anesthetics tend to influence a vasodilatory effect until the central temperature is sufficiently low that vasoconstriction occurs. Furthermore, those individuals receiving a local anesthetic do not experience the post-operative increase in metabolic rate seen in individuals receiving a general anesthetic.

### Body Fat

Body fat acts as an insulator and slows the rate of body heat loss. Closs et al. (1986) studied various indicators of body fat (mid-arm circumference, triceps and biceps skinfold thickness, body density and percent body fat) and the magnitude of decline in body temperature. Simple regression of the body fat indicators with the magnitude of temperature decline resulted in all body fat indicators being significantly correlated with the magnitude of temperature decline ( $r$  ranged from - 0.45 to 0.45). The indicators with the greatest  $r$  value were the biceps skinfold thickness ( $r=-0.42$ ;  $p=0.005$ ), body density ( $r=0.45$ ;  $p=0.006$ ) and percent body fat ( $r=-0.45$ ;  $p=0.005$ ).

Carli, Gabrielczyk et al. (1986) studied self-rewarming rates and percent body fat and reported "no correlation" between percent body fat and rate of rewarming. Theoretically, the rise of body temperature from self-rewarming is from an increased metabolic rate and conservation of body heat. Therefore, if the subjects in the Carli, Gabrielczyk et al. (1986) study were in a neutral environment or covered with an adequate layer of exogenous insulation which decreased or removed the body heat loss, the percent of body fat should not have any effect on the rate of self-rewarming. However, if the rewarming had been from an exogenous source of heat applied to the exterior of the body, one would expect the body fat to have an effect on the rate of rewarming since

body fat has a lower rate of conductance than muscle. In conclusion, body fat does slow the rate of body heat loss, but does not directly affect self-rewarming if adequate insulation exists to decrease or prevent body heat loss.

#### Body Mass Index

Body mass index (BMI) is a calculation which takes into account the individual's height and body surface area, such that the individual with greater adiposity for the same height would have a greater BMI. Erickson and Yount (1991) reported BMI ( $\text{kg}/\text{m}^2$ ) accounted for 11% of the variance in  $T_{ty}$  from the time of entry into the operating suite until entry into the post-operative recovery area. Vaughan et al. (1981) reported that an elderly group, who had a significantly lower BMI than a younger group, had a significantly lower  $T_{ty}$  upon admission to the post-operative recovery area than the younger group. The difference in  $T_{ty}$  between the two groups remained significant during the 90 minutes of stay in the recovery area. Erickson and Yount (1991) did not find age to be a significant factor in the decline of  $T_{ty}$ . Theoretically, the individual with the larger amount of adipose tissue will have a smaller decline in body temperature. Also, the individual with the larger amount of adipose tissue will have a greater BMI for the same height. Hence, the findings of Erickson and Yount (1991) and Vaughan et al. (1981) do support BMI as an indicator which has predictive value in the decline of body temperature intra-operatively.

#### Gender

The effect of gender on the thermoregulatory process remains controversial. In clinical research literature, Closs et al. (1986) and Goldberg and Roe (1966) reported that the relationship of gender and the development of hypothermia was not statistically significant, but Roe et al. (1966) did report

a gender difference. Closs et al. study findings may have been affected by sample size and age. The older group (mean age of 72 years; range or standard deviation not reported) consisted of 12 females and 2 males while the younger group (mean age of 53.6 years; range or standard deviation not reported) consisted of 4 females and 4 males. Closs et al. did find age to be a significant factor in the thermoregulatory process, so the effect of gender may have been masked by the age effect. Goldberg and Roe (1966) also found the relationship of gender and development of hypothermia to be statistically nonsignificant, but reported a mean decrease in central temperature in the male group of  $0.62^{\circ}\text{C}$  ( $n=41$ ) and  $0.5^{\circ}\text{C}$  decline in the female group ( $n=60$ ). Furthermore, Goldberg and Roe (1966) reported age (18 to 87 years of age) had a significant effect on development of hypothermia. Also reported was that those individuals receiving d turbo-curare with the inhalation anesthesia had twice the degree of temperature decline as those individuals only receiving inhalation anesthesia. However, Goldberg and Roe do not discuss whether there was stratification of men and women based on age and anesthesia.

Roe et al. (1966) studied 24 adults of which 12 males and 8 females had a decline in central temperature during surgery. The male group had an average decline of  $0.8^{\circ}\text{C}$  with an increase in  $\text{O}_2$  consumption of 94% during and following the surgical procedure. The female group had an average decline in central temperature of  $1.4^{\circ}\text{C}$  with an increase in  $\text{O}_2$  consumption of only 43%. The Roe et al. study also found that the elderly (61 to 80 years of age) had the greatest decline in central temperature (mean of  $1.3^{\circ}\text{C}$ ). The study does not discuss whether the men and women were evenly distributed across the age span (18 to 79 years of age) studied. Hence, the effect of gender on thermoregulation remains questionable.

In the physiological research literature which focused on healthy, young (age range 20 to 42) subjects, the question of thermoregulation differences based on gender is not resolved. The central temperature gender difference reported varies from females having a significantly higher  $T_{ty}$  with cold air exposure (females mean  $T_{ty}$   $36.87^{\circ}$  C and males mean  $T_{ty}$   $36.57^{\circ}$  C after 1 hour of air exposure at  $16^{\circ}$  C) to no significant difference in  $T_{re}$  between the groups in either air or water exposure (Table 12; Cunningham, Stolwijk, & Wenger, 1978; McArdle, Magel, Gergley, Spina, & Toner, 1984; Walsh, & Graham, 1986; White, Ross & Mekjavic, 1992).

The skin temperature gender difference reported is also variable. Walsh and Graham (1986) and Cunningham et al. (1978) reported females to have significantly lower mean  $T_{sk}$  in neutral and in cold air environments than males. However, McArdle et al. (1984) reported the mean  $T_{sk}$  to be higher in females than males during water exposure of 20, 24 and  $28^{\circ}$  C. McArdle et al. do not report the mechanism utilized to measure  $T_{sk}$  or the calculation of mean skin temperature. Hence, the difference in skin temperature reported results may be a measurement issue or an issue of different effect based on different exposure mediums.

Furthermore, White, et al. (1992) reported an insignificant difference in the distribution of body fat based on gender. Although statistically nonsignificant, the females had greater skinfold thicknesses in the lower body while the males had greater skinfold thicknesses in the upper body. The difference in distribution of body fat (skinfold thickness) will affect the skin temperature depending upon whether the measurement sites are over areas with greater or lesser subcutaneous adipose. Therefore, gender temperature regulation issues may exist. Furthermore, females, from puberty through the

premenopausal period of the life cycle, experience an elevation of progesterone during the luteal phase of the menstrual cycle and an increase of central temperature of approximately  $0.5^{\circ}\text{C}$  (Goldfien & Monroe, 1986). The studies cited in Table 12 do not indicate the menstrual phase of the female subjects. Therefore, it is unknown whether the phase of the menstrual cycle had an effect on the findings.

TABLE 12. Gender, Skin and Central Temperatures in Cold Environments.

Study	Conditions	Female	Male
Cunningham, Stolwijk & Wenger (1978)	1 hour $16^{\circ}\text{C}$ Tamb exposure	$36.87^{\circ}\text{C}$ Tty $29.33^{\circ}\text{C}$ Tsk	$36.57^{\circ}\text{C}$ Tty $30.19^{\circ}\text{C}$ Tsk
	$27\text{-}30^{\circ}\text{C}$ Tamb exposure	$37.11^{\circ}\text{C}$ Tty $34.54^{\circ}\text{C}$ Tsk	$37.0^{\circ}\text{C}$ Tty $35.15^{\circ}\text{C}$ Tsk
McArdle, Magel, Gergley, Spina & Toner (1984)	1 hour $20^{\circ}\text{C}$ T <sub>bath</sub> exposure	$35.7^{\circ}\text{C}$ Tre $22.3^{\circ}\text{C}$ Tsk	$36.1^{\circ}\text{C}$ Tre $21.4^{\circ}\text{C}$ Tsk
	1 hour $24^{\circ}\text{C}$ T <sub>bath</sub> exposure	$36.2^{\circ}\text{C}$ Tre $25.2^{\circ}\text{C}$ Tsk	$36.6^{\circ}\text{C}$ Tre $24.7^{\circ}\text{C}$ Tsk
	1 hour $28^{\circ}\text{C}$ T <sub>bath</sub>	$36.6^{\circ}\text{C}$ Tre $28.8^{\circ}\text{C}$ Tsk	$36.5^{\circ}\text{C}$ Tre $28.3^{\circ}\text{C}$ Tsk
Walsh & Graham (1986)	10 minutes $21.7^{\circ}\text{C}$ Tamb	Tre NS difference Tsk Sign. lower	
	Recovery from 20 minutes exercise followed by 10 minute rest in $+10, +3.5, -3.5,$ and $-10^{\circ}\text{C}$	Tre NS difference Tsk Sign. lower	
White, Ross & Mekjavic (1992)	$40^{\circ}\text{C}$ T <sub>bath</sub> exposure until Tes reached $38.5^{\circ}\text{C}$		
	$30.6^{\circ}\text{C}$ T <sub>bath</sub> exposure until Tre decreased by $1.5^{\circ}\text{C}$ or O <sub>2</sub> Consumption above resting level		Tre NS correlation with gender

Note. Tamb = ambient temperature; Tty = tympanic temperature; Tsk = skin temperature; T<sub>bath</sub> = water bath temperature; Tre = rectal temperature; Tes = esophageal temperature.

### Operative Site

The intra-operative decline in body temperature is affected by the site of the surgery, such as abdominal cavity versus non-abdominal surgery (Goldberg & Roe, 1966; Babcock, Black, Chung, & O'Hara, 1979; Closs et al. 1986; Tollofsrud, Gundersen, & Andersen, 1984; Vaughan et al., 1981). However, the aforementioned studies do not address other factors, such as length of time exposed to the cold environment, amount of body and body tissues exposed to the cold environment, body fat or age of the subjects. Vaughan et al. (1981) studied individuals undergoing intra-abdominal surgical procedures, such as cholecystectomy, appendectomies and exploratory laparotomies, and individuals undergoing non-abdominal surgeries, such as eye, ear, nose, and rectal surgeries. The intra-abdominal surgeries may require a longer surgery time as well as require a greater amount of body surface and tissue exposure to the environment than the non-abdominal surgeries. Another example is the study by Babcock et al. (1979) in which patients undergoing inguinal hernia repair or aortic reconstruction were studied. The inguinal hernia repair group had a mean central temperature decline of  $0.6^{\circ}$  C, while the aortic reconstruction group had a mean decline on  $2.2^{\circ}$  C. The temperature of the patient in the inguinal hernia group with the most profound temperature drop had a smaller loss of body heat each hour as well as a smaller total body heat loss than the aortic reconstruction patient with the most profound temperature drop. Babcock et al. (1979) did not describe the amount of body or tissue exposure, however, inguinal hernia repair involves a small incision with minimal tissue exposure in the lower abdomen. Where as, aortic repair requires a large mid-line incision with exposure of the abdominal viscera.

Hence, the difference in central temperature with operative site is a function of multiple factors which include the length of exposure time, age, the amount of tissue or body exposure and even the temperature gradient that exists in the operating suite.

### Circadian Rhythm

The central body temperature varies during a 24 hour period (circadian rhythm) in normal healthy individuals. The normal circadian rhythm is interrupted postoperatively. A rise in central temperature in hypothermic individuals occurs postoperatively regardless of the time of the day in which the immediate postoperative phase occurs (Carli & Aber, 1987). The circadian temperature pattern postoperatively is altered such that a higher mesor occurs (Farr, Keene, Samson & Michael, 1984; Groza, Vrancianu, Filcescu, Dutu, Lemnete, Pana, Petrescu, Petersen, Kadefors, & Persson, 1982) which was evidenced from the first through the fifth postoperative days (Groza et. al, 1982). In humans, the amount of time for the mesor to return to the preoperative level is unknown while in rats Farr, Campbell-Grossman and Mack (1988) reported a normal mesor on the fifteenth post-operative day. The literature is in disagreement on a change in the acrophase post-operatively. Carli and Aber (1987) report a peak temperature approximately 12 to 14 hours post-operatively with a second temperature peak approximately 18 hours later. The surgeries occurred at different times of the day with the postoperative temperature change pattern remaining consistent among the subjects. Farr, Campbell-Grossman and Mack (1988) reported a shift in the acrophase in post-operative rats of 10 hours with a return to normal acrophase in 9 days post-operatively. However, Groza et. al (1982) indicate the acrophase was unchanged post-operatively in 10 women. Therefore, the circadian rhythm is

interrupted post-operatively, but the ability to rewarm appears to remain constant regardless of the time of day.

#### Whole Body Rewarming

Rewarming is the process of warming the individual with a hypothermic central temperature ( $<36.5^{\circ}\text{C}$ ) to a state of normothermia ( $\geq 36.5^{\circ}\text{C}$ ). Slotman et al. (1985) found those individuals with a central temperature of less than  $36.1^{\circ}\text{C}$  at 2 hours or greater post-operatively had a significantly higher mortality rate. Hence, rewarming is defined as returning the central body temperature to  $\geq 36.5^{\circ}\text{C}$  within 2 hours of the exposure. The rewarming process is affected by the endogenous heat supply (metabolic rate), the exogenous heat supply (therapy) and the rate of heat loss from the body.

#### Ideal Rewarming Therapy

An ideal therapy replaces the lost body heat without a rise in the metabolic rate. Minimally a rewarming therapy must not contribute to the further loss of over-all body heat and provides a consistent environment. In an individual who is in a compromised state from the hypothermia or from an incapacity to support an increased metabolic rate, the therapy decreases or eliminates further body heat loss as well as supplying a continuous exogenous heat source.

Rewarming therapeutics can be classified as invasive or non-invasive and as preventive or curative. An invasive therapy is a heat source which is applied to an internal membrane or body substance. A non-invasive therapy is applied to the skin surface. The non-invasive therapy alters the individual's environment, hence, affecting the environmental factors described in Table 1.

A preventive therapy is one which prevents or decreases the rate of body heat loss, while a curative therapy supplies a continuous exogenous heat supply to the body. Preventive or curative therapies may be utilized during

exposure to environments which cause an increased loss of body heat, such as in the operating suite, a non-invasive preventive or curative therapy may be applied prior to the development of hypothermia. Furthermore, if the individual is hypothermic, either a preventive or curative therapy may be selected. If a hypothermic individual is treated with a preventive therapy, the individual's rewarming process is from self-rewarming. In self-rewarming, the therapy prevents or decreases the body heat loss, while the increase in body heat is from heat production from metabolism. Table 13 lists therapies which are representative of the two different classifications of rewarming therapies.

The invasive, curative rewarming therapies provide an exogenous heat source which is applied via cannulation of some site or orifice of the body. The invasive therapies increase the individual's risk of infection, as well as require special skills for initiation of the therapy and a physician order. The non-invasive therapies are generally within the nursing realm of therapy decision and application.

TABLE 13. Rewarming Therapies.

	Preventive	Curative
Invasive		Heated, humidified gas Continuous arterio-venous rewarming Heart-lung bypass machine Peritoneal lavage Heated intravenous fluids
Non-invasive	Cotton blankets Plasticized aluminum blankets Plasticized aluminum head cover	Heated fluid blanket Heated convective air blanket Radiant warmer

The preventive, non-invasive rewarming therapies increase the exogenous insulation layer either via increasing the thickness of the material layer which surrounds the body or via creation of a micro-environment in which the environmental convective and radiant effects are decreased. The preventive non-invasive therapies do not provide a continuous heat source. The potential harm to an individual caused by these therapies is negligible.

The non-invasive, curative therapies provide an exogenous heat source which is applied to the skin surface of the body. Furthermore, the non-invasive, curative therapies alter the individual's environment. The skills to apply the therapy are minimal, however, the potential for harm does exist if the therapy is applied and managed incorrectly (Augustine Medical Bair Hugger, 1990; Scott, 1967). The following section will address the non-invasive therapies which are generally within the nursing realm of decision and application.

#### Non-invasive Preventive Therapies

Cotton blankets. Cotton blankets which cover a large portion of the body surface area decrease the convective and radiant effects of the surrounding environment. Warmed cotton blankets provide a temporary heat source. However, within minutes of application, the heat dissipates from the cotton blanket resulting in an increase in heat flux from the body (Sessler, McGuire, & Sessler, 1991). Sessler, McGuire and Sessler (1991) compared the heat flux of five reclining volunteers in a surgical suite in an ambient temperature (Tamb) of 20.6<sup>o</sup> C. Six different types of insulating materials were used. The heat flux from the uncovered volunteers, during the last 20 minutes of the 1 to 2 hour rest period in the described environment, was 100 Watts (W). With the application of the warmed cotton blanket (taken from 48<sup>o</sup> C blanket warmer),

the heat flux dropped to 58 W, but increased to a heat flux of 71 W by 40 minutes after application of the cotton blanket. The other insulators tested (plasticized aluminum cover, paper drape, cloth drape, and a Bair Hugger blanket partially expanded with room air) maintained a decreased, but constant, rate of heat flux from the body. The plasticized aluminum cover had the lowest rate of heat flux (61 W) throughout the hour of experimental observations.

Hence, warmed cotton blankets may serve as a preventive therapy, but do not provide a continuous source of heat. Warmed cotton blankets do decrease the rate of endogenous heat loss. However, one must question the efficacy of the application of warmed cotton blankets as a therapy since the rate of heat flux increases over time as the blankets cool, although the rate of heat flux did not return to the rate of flux measured prior to application of the warmed cotton blankets.

Plasticized aluminum blankets. The plasticized aluminum blanket is intended to reduce the amount of radiant heat loss via placement of the reflective side of the blanket towards the individual. However, the blanket probably decreases the convective air movement around the body which effectively increases the insulation layer. Sessler, McGuire and Sessler (1991) reported the plasticized aluminum drape to be the most effective insulator in decreasing the heat flux from the volunteers (heat flux of 61 W versus 100 W with no insulating cover) who reclined on a surgical table in an operative suite.

However, in individuals undergoing surgery, the effect of the aluminum blanket is controversial. Brunton, Thoms and Blair (1982) studied 28 neurosurgery patients over a 210 minute period and found no significant difference

in the  $T_{re}$  between the groups of patients with or without the aluminum blanket cover. However, when heat content was calculated, the control group had a significantly lower heat content than the experimental group. Brunton et al. (1982) measured  $T_{re}$  as central temperature in patients who ranged in age from 24 to 74 years of age.  $T_{re}$  is known to lag behind other central temperature sites. However, the measurement period was over 210 minutes which would allow enough time for rectal temperature changes. However, Brunton et al. did not consider the effect of age on thermoregulation. Hence, Brunton et al. results may have been influenced by not having comparable groups.

Other studies did report a significant difference in central temperature with the use of plasticized aluminum blankets. Bourke, Wurm, Rosenberg, and Russell (1984) studied 90 patients undergoing either an endarterectomy or neurosurgery and found a significant difference in the  $T_{es}$  of the experimental group, who were covered with the aluminum blanket. After statistically controlling for factors, such as age, body mass index, surgery time and the subjects temperature pretransport to surgery, Erickson and Yount (1991) in 60 surgical patients found a significant difference in the  $T_{ty}$  of patients who were covered with an aluminum blanket and those who were not covered with an aluminum blanket. Hence, the literature does support the plasticized aluminum blanket as an effective preventive therapy, in light of the blanket does decrease the body heat loss to the environment.

Plasticized aluminum head cover. The plasticized head cover decreases the radiant and convective heat loss from the head. Erickson and Yount (1991) studied the effect of an aluminum head cover without any other preventive therapies. Erickson and Yount found the central temperature of those studied

was not significantly different from the control group, with both groups experiencing approximately a  $2.0^{\circ}$  C decline in central temperature. Sessler, McGuire and Sessler (1991) point out the amount of skin surface covered is probably of more importance than which surface is covered when studying heat flux of volunteers in the operative environment. Hence, the potential of head covers serving as an effective preventive therapy, when utilized without any other preventive or curative therapies, is unlikely since only 5% of the body surface is covered with the head cover (Lund & Browder, 1944).

Summary. The noninvasive, preventive therapies do not provide a continuous heat source, but a decrease in loss of endogenous heat does occur. The plasticized aluminum blanket is the most effective in decreasing the rate of heat loss. However, with noninvasive, preventive therapies, the rate of rewarming is dependent upon the individual's metabolic rate which may not be sufficient to rewarm the individual within 2 hours post-operatively. Therefore, to provide exogenous heat, the therapy selection must be from the curative therapies. The following section will discuss non-invasive curative therapies.

#### Non-invasive Curative Therapies

Heated fluid blanket. The heated fluid blanket supplies heat to the body by conduction. The temperature of the blanket must be greater than the skin temperature to create a temperature gradient which results in transfer of heat into the body. If the heated fluid blanket is placed on the anterior surface, the blanket may also decrease the convective effects of the environment. However, the blanket is commonly placed only on the individual's posterior surface.

In clinical studies, the heated fluid blanket had no effect on maintaining the central temperature intra-operatively or elevating the central temperature during the rewarming process in the postoperative recovery area. Morris and Kumar (1972) studied the effect of a heated fluid blanket on the  $T_{es}$  in 50 operative patients in ambient temperatures of  $19.7^{\circ}\text{C}$  and  $22.3^{\circ}\text{C}$ . The patient was placed on top of the heated fluid blanket which was covered with two cotton blankets. The heated fluid blanket was turned on after the patient was placed on top of the blanket. The fluid temperature of the machine was  $21.5^{\circ}\text{C}$  prior to turning on the machine. With the machine control set at  $41^{\circ}\text{C}$ , the machine fluid temperature became  $41.3^{\circ}\text{C}$  after 11 minutes of heating. However, the temperature of the cotton blanket which was next to the patient reached a stable temperature of  $37.7^{\circ}\text{C}$  after 45 minutes of heating. Hence, the fluid blanket requires a long warm up period. Furthermore, Morris and Kumar (1972) reported that the  $T_{es}$  was not significantly different in those subjects warmed with the fluid blanket and those who were not warmed over a 3 hour period of anesthesia. However, Morris and Kumar (1972) did find a significant difference in  $T_{es}$  between those subjects in the warm ( $22.3^{\circ}\text{C}$ ) and the cold ( $19.7^{\circ}\text{C}$ ) environment at 2 and 3 hours intra-operatively. Therefore, the heated fluid blanket, placed under the patient, is not an effective therapy in maintaining the central temperature intra-operatively.

Hardy, Cirillo, and Gutzeit (1988) studied 45 subjects postoperatively who had an axillary temperature ( $T_{ax}$ )  $\leq 34.5^{\circ}\text{C}$  and were rewarmed with one of three different rewarming therapies (warmed cotton blankets placed on patient's anterior surface, warmed cotton blankets with heated fluid blanket placed on the patient's anterior surface, and warmed cotton blankets with heated aerosol). The  $T_{ax}$  of the three groups were not significantly different

upon entry into the postoperative care area. The rate of rewarming at 30 and 60 minutes of therapy were not significantly different for any of the three therapies. Therefore, the heated fluid blanket used intra-operatively or postoperatively has minimal effect on the rate of rewarming.

Sessler and Moayeri (1990) studied five volunteers who reclined in a 20.6<sup>o</sup> C operative suite. The volunteers reclined for 10 minutes prior to application of various therapies. The average heat loss was 130 W from the body to the environment during the first 10 minutes. After 10 minutes of warming with a heated fluid circulating blanket placed on the anterior surface, the average heat loss was 100 W. By approximately 20 minutes of heating with the heated fluid blanket, the average heat flux was approximately 15 W from the heated fluid blanket into the body. The heat flux into the body was maintained at approximately 15 W over the next 10 minute period. Hence, in healthy individuals the maximal heat flux into the body was 15 W when the only area of body surface exposed to the environment was the head and neck. Therefore, the effectiveness of the heated fluid blanket in the operative environment is minimal, because a greater amount of body surface area is exposed during an operative procedure and the potential for the blanket to be placed on the anterior surface during surgery is unlikely.

Heated convective air blanket. The heated air blanket provides a micro-environment, which covers the anterior portion of the body excluding the head. The mechanism of heat transfer from the micro-environment to the skin is convection. The heated air blanket (Bair Hugger, Augustine Medical Inc., Eden Prairie, Minnesota) provides three different temperature settings of low (approximately 33<sup>o</sup> C), medium (approximately 38<sup>o</sup> C) and high (approximately 43<sup>o</sup> C) (Sessler & Moayeri, 1990). When measuring the

effectiveness of this therapy the question is raised whether measurement of central temperature is the appropriate measurement or whether one should measure heat load via mean body temperature to get an accurate picture of what is occurring with rewarming (Sessler & Moayeri, 1990). The mean body temperature literature is fraught with argument of which weighting mechanism of central and mean skin temperatures result in the most valid measurement of mean body temperature. Hence, both central and mean skin temperature are probably needed to describe the rewarming process when the Bair Hugger rewarming therapy is utilized.

The effectiveness of the heated convective air blanket as a rewarming therapy appears to be controversial within the clinical literature. Summers, Dudgeon, Byram and Zingsheim (1990), in postoperative, hypothermic ( $T_{ty} \leq 36.0^{\circ} \text{C}$ ) patients, reported there was no significant difference in  $T_{ty}$  of those subjects who had been warmed for 1 hour with the heated air blanket and the subjects warmed with warmed cotton blankets. However,  $T_{sk}$  was significantly higher in those rewarmed with the heated air blanket at 1 hour of heating. Sessler and Moayeri (1990) studied five healthy volunteers, in  $T_{amb}$  of  $20.6^{\circ} \text{C}$ , with the heated air blanket set on low and on medium. Sessler and Moayeri (1990) monitored  $T_{ty}$  during the various therapies and reported a decline of  $0.3$  to  $0.2^{\circ} \text{C}$  with the heated air blanket, a heat flow into the body of approximately  $5 \text{ W}$  on a medium setting and a heat loss to the environment of approximately  $25 \text{ W}$  on a low setting. However, Sessler, Stoen and Glosten (1989) demonstrated in five healthy volunteers, in  $T_{amb}$  of  $28^{\circ} \text{C}$ , the heat flux from the body to the environment could be reversed to a negligible movement of heat (approximately  $4 \text{ W}$ ) from the body to the micro-environment with the heated air blanket set on low heat.

Sessler would argue that no significant difference between the effect of the therapies was because central temperature is not reflective of the heating process. However, the question must be raised regarding the effect of the air flow from the heated air blanket on the  $T_{ty}$ . McCaffrey et al. (1975) demonstrated that cooling of one side of the head did result in variation of the  $T_{ty}$  on the ipsilateral side. Furthermore, Conover, Lennon and Rose (1989) reported a significantly higher oral and axillary skin temperature in those individuals warmed with the Bair Hugger when compared to individuals warmed with cotton blankets at 30, 45, 60 and 75 minutes of therapy. Therefore, the effect of the air movement out the top of the blanket across the face may be the contributing factor in not finding a significant effect of rewarming when operationalizing central temperature as  $T_{ty}$ .

Furthermore, the temperature gradient between the room environment and the microenvironment may have an effect on the ability to rewarm an individual with the heated convective air blanket. The  $T_{amb}$  in the study by Sessler and Moayeri (1990) was  $20.6^{\circ}\text{C}$  compared to  $T_{amb} 28^{\circ}\text{C}$  in the study by Sessler, Stoen and Glosten (1989). In the  $T_{amb}$  of  $20.6^{\circ}\text{C}$  with the blanket set on low heat, the heat flux from the body was 25 W, while in  $T_{amb} 28^{\circ}\text{C}$  with a low heat setting, the heat flux was 4 W from the body.

Therefore, the heated convective air blanket does supply a continuous source of heat to the individual. However, the therapy may interfere with temperature measurement at  $T_{ty}$ . Furthermore, the effectiveness of the therapy does decline when the heat setting is low and the  $T_{amb}$  is low.

Radiant warmer. The radiant warmer consists of varying products which include shields that are permanently attached to the ceiling, portable shields or portable infrared lamps. The radiant warmer does not create a micro-

environment. The individual remains in contact with the ambient air movement which may negate the warming effect of the radiant ceiling.

The effectiveness of infrared bulbs was studied in 28, 20 to 55 year old, post-operative patients (Kucha, Nichols, Christ & Bynum, 1974). The radiant heat source was two 250 W infrared bulbs positioned 28 inches above the mattress over the abdomen which was covered with one sheet in  $T_{amb}$  of 23.5 to 26.88<sup>o</sup> C. When the rate of rise of  $T_{re}$  and  $T_{sk}$  (near the umbilicus) was compared in individuals who were covered with warmed cotton blankets and those who were warmed with infrared bulbs, there was no significant difference in the rate of rewarming. Sessler and Moayeri (1990) studied the average heat flux in five volunteers at rest and following exposure to two 250 W infrared bulbs positioned 28 inches above the mattress over the abdomen, in  $T_{amb}$  of 20.6<sup>o</sup> C. After a 10 minute rest period, the body to environment heat flux was 130 W. After 10 minutes of infrared bulb exposure, a 70 W flux of heat from the body to the environment existed. Measurements after 20 and 30 minutes of heating remained at 70 W of heat flux from the body to the environment. Hence, the infrared bulbs may reduce the rate of radiant heat loss, but do not provide large enough source of heat to reverse the trend of loss of body heat in the operative environment.

Henneberg, Eklund, Joachimsson, Stjernstorm and Wiklund (1985) studied the effects of a thermal ceiling (Aragona AB, Sweden) and no therapy (covered with hospital blankets) in 19, 21 to 33 year old, postoperative women. The group was split in half with each half receiving the thermal ceiling or no therapy for the first postoperative hour and then the groups were crossed over to the other therapy for the second hour.  $T_{re}$ ,  $T_{aural}$ , and  $T_{es}$  were "identical" through out the 2 hour period. However, when determining the

energy balance (increase in total body heat - energy production), the patients receiving the thermal ceiling therapy during the first hour had an increase in total body heat exceeding the energy production as determined by measuring the rate of oxygen consumption. The no therapy group had a higher metabolic rate than the therapy group and a smaller increase in total body heat which resulted in a negative heat balance. After the therapy cross over in the second hour, the increase in total body heat for the second hour was equal in both groups as well as the metabolic rates were similar (281 kJ and 301 kJ, no therapy and thermal ceiling, respectively). Hence, the thermal ceiling does provide a limited heat supply when applied within the first hour postoperatively.

Sessler and Moayeri (1990) studied five volunteer subjects exposed to a  $20.6^{\circ}\text{C}$  environment for a 10 minute rest period, followed by exposure to a thermal ceiling (Aragona Medical Inc., New Jersey) set on high (500 W) and mounted 56 cm above the volunteers. During the rest period, the average heat flux was 130 W from the body to the environment. At 10 minutes of thermal ceiling therapy, the heat flux was 105 W from the body to the environment. After 20 and 30 minutes, the heat flux was 80 W and 75 W from the body to the environment, respectively. The volunteers had a mean  $T_{ty}$  decline of  $0.05^{\circ}\text{C}$  during the thermal ceiling exposure. However, the mean  $T_{sk}$  (10 sites) rose approximately  $0.7^{\circ}\text{C}$  during the thermal ceiling exposure.

Hence, it remains unclear as to the overall effect of the thermal ceiling on rewarming. Minimally the rate of heat loss is decreased with either infrared bulbs or a thermal ceiling. One of the issues not addressed by radiant warmers is the convective heat loss to the environment which may have been at least part of the reason for the continued heat flux from the body.

Summary. The most effective noninvasive, curative therapy is the heated convective air blanket. The heated convective air blanket does provide a continuous heat source, but varying results have been reported when measuring effectiveness of therapy based on  $T_{ty}$  and  $T_{or}$  temperatures as central temperatures.  $T_{ty}$  may be affected by the flow of air which leaves the micro-environment through an opening which surrounds the neck.

The argument raised by Sessler and Moayeri (1990) of measuring mean body temperature instead of central temperature when evaluating the effectiveness of the convective-air blanket can be applied to all non-invasive warming therapies. Sessler and Moayeri (1990) reported an increase in mean skin temperature and a decrease in central temperature when the subjects were exposed to radiant thermal ceiling therapy. The heat loss measured in watts during convective-air blanket warming was terminated and a heat gain of 5 watts was attained on a medium setting. Whereas, the radiant thermal ceiling therapy resulted in a continual heat loss from the body of 75 watts after 30 minutes of therapy. Sessler and Moayeri did not calculate a mean body temperature in relation to either therapies. Hence, it is unknown whether a mean body temperature would reflect the actual heat load of the body or indicate the longer term effect on central temperature.

When the nurse is prescribing a therapy for a hypothermic, post-operative patient, the criteria for therapy selection are not clearly defined. For a non-invasive curative therapy to be effective, one must consider the ability to transfer heat from the therapy to the surface of the body and into the core of the body. Theoretically, the factors which affect the ability to transfer heat are an effective, continuous heat source, conductance, vasoactivity and body

surface to body mass ratio. However, no studies in the literature describe the aforementioned factors during the rewarming process.

To rewarm an individual with low conductance and vasoconstriction within 2 hours may require that an invasive mechanism of rewarming be utilized in place of a non-invasive therapy. Hence, a better understanding of the rewarming process is required for appropriate selection of rewarming therapies. However, the literature does not describe the physiological responses with rewarming or define which indicators are beneficial in selection of a rewarming therapy for the individual. Therefore, this study described central body and skin temperatures, vasoactivity, conductance, metabolic rate, and body mass to surface area during the rewarming process utilizing the most effective non-invasive curative therapy, a warmed convective-air blanket (Bair Hugger, Augustine Medical Inc., Eden Prairie, Minnesota).

The literature cites other factors which have a significant effect on the rate of whole body cooling (Table 10), such as age, ambient temperature, anesthesia route, duration of surgery or the site of surgery and gender. The effect of age on the rate of cooling and rewarming post-operatively is probably associated with a decreased metabolic rate in those individuals over the age of 60 which should be taken into consideration when selecting a therapy. The anesthesia route affects vasoactivity and may have some impact on the metabolic rate. The duration of surgery and the site of surgery may have similar effects since certain types of surgery require longer time to complete the procedure. Also, the site of surgery does have an impact on the amount of tissue exposed to the cool environment of the surgical area. The ambient temperature of the surgical suite affected the degree of hypothermia

experienced. Gender may also serve as a factor in the prediction of the ability to rewarm an individual based on different patterns of adipose distribution. Therefore, the study population will be limited based on age and gender. Furthermore, most of the cooling or warming studies include young subjects and particularly male subjects. Finally, the inability, to control for ambient temperature, the length of exposure, the amount of body tissue exposed, and the type and amount of anesthetics, precluded this initial study from occurring in the clinical arena. Hence, women between the ages of 40 and 60 years of age were brought into a laboratory setting, exposed to 60 minutes of controlled exposure, and 60 minutes of warming. The next chapter will describe the study procedure and measurements.

## CHAPTER III

### Methods

#### Design

This within-subject comparison study described the rewarming patterns in healthy, cooled nonoperative women, between 40 and 60 years of age, warmed with the Bair Hugger convective air blanket (Augustine Medical, Inc., Eden Prairie, Minnesota). Measurements of conductance, of metabolic rate, of vasoactivity, and of body mass were utilized to describe the factors within an individual which affected the pattern of rewarming. The phenomenon of rewarming is not well understood within an individual or between individuals. Furthermore, even less is known about rewarming in women. Conceptually, it is known that rewarming is dependent upon the balance between heat gain from the metabolic rate and the external heat sources and heat loss from exposure to a cool environment. Furthermore, it is known that the rate of central rewarming from an external heat source applied to the skin surface is affected by the thickness of the insulation layer of the body which consists of subcutaneous fat and vasoconstricted skin. However, the studies of rewarming have not taken into account the effect of the metabolic rate, the subcutaneous fat and the vasoactive state of the individual. Furthermore, the rewarming studies do not describe the pattern of rewarming within an individual and between individuals when the Bair Hugger was utilized. Therefore, the study design was a within-subject design to describe the pattern of rewarming within an individual and the relationships of factors which affected the rewarming rate. Furthermore, the study measurements which were utilized had not been studied together to describe the individual and the rewarming process within or between individuals.

### Setting

The subjects were studied in the Human Cardiovascular Functions Laboratory, Department of Physiology, University of Washington. The ambient temperature of the laboratory was controlled by a central heating plant, but was also somewhat influenced by the air temperature outdoors. The study of Subject 1 occurred in November, while the remaining experiments occurred in the following calendar year, between the last week of February and the third week of June. The room air movement was that of a typical indoor environment.

The cooling bed was a mesh fabric which was stretched over a metal frame and suspended approximately 12 inches at the lowest point from the floor. Spray nozzles were affixed below the mesh frame to direct water spray to the subject's posterior surface. The anterior spray nozzles were placed and supported over the anterior body surface after the subject reclined on the mesh frame. The subject and anterior spray nozzles were covered with plastic sheeting to contain the water spray. The water sprayed on the subject was collected in a plastic sheet below the subject and was then drained into a floor drain in a shower adjacent to the mesh frame. During cooling, the subject was supported in a semi-reclining position.

The warming bed was a 4 inch thick foam pad which was positioned on the floor of the laboratory. The subject reclined in a flat position, with elevation of the head on one pillow, during the warming period. Because of the physical set up of the laboratory, the cooling and warming beds were positioned in a parallel line, with the foot of the cooling bed within 3 feet of the head of the warming bed.

### Sample

A convenience sample of 8 females was studied.

#### Inclusion Criteria

The subjects were female, healthy, English speaking, between the ages of 40 and 60 years and within  $\pm$  20% of the ideal body weight for height as defined by the Metropolitan Height and Weight Tables. For this study, healthy was defined as having no diagnosed diseases which have an effect on cardio-neuro-endocrine-vascular systems which affect thermoregulation. The specific disease entities are listed under the exclusion criteria. For this study, obesity was defined as a weight of greater than 120% of the ideal body weight for height (Poleman & Peckenpaugh, 1991). Under weight was defined as less than 80% of ideal body weight (Appendix A).

#### Exclusion Criteria

Those individuals with known peripheral neuropathies, vasoactivity problems such as Raynaud's disease, thyroid problems, spinal cord injuries, or craniotomies were excluded. All pregnant individuals were excluded. All individuals on non-specific beta blocker medications were excluded to avoid a confounding effect of the medication on vasoactivity (Pescatello, Mack, Leach & Nadel, 1990). Based on a medication history, any individual who was currently taking a medication that would affect thermoregulation was excluded. All individuals with blood, saliva or air born communicable diseases were excluded.

#### Selection

The subjects were solicited by posting study notices on bulletin boards. The subjects were screened by phone for the inclusion and exclusion criteria (Appendix B). Those individuals who met the criteria were informed of the

study process. If the individual chose to participate in the study, the individual was brought into the laboratory where the study process was again explained. After touring the laboratory, written consent was obtained (Appendix C).

### Variables and Instrumentation

#### Demographic Information

Name, age, height, weight, and medical history were obtained during the telephone interview (Appendix B). During the telephone interview, all subjects were questioned about their menopausal status which was defined as pre-menopausal or menopausal. Menopausal was defined as no menses within the last 12 months. On the day of the study, all pre-menopausal subjects were interviewed for menstrual phase. The luteal menstrual phase was defined as the 14 days prior to onset of menses and the follicular phase as the remainder of the cycle (Goldfien & Monroe, 1986). The pre-menopausal changes to the menstrual cycle affect the follicular phase (Goldfien & Monroe, 1986). If the subject was unsure of the dates of the last occurrence of menses or the number of days since the end of menses, the woman was followed for up to 30 days to determine menstrual phase during the study.

The operationalized study variables are described in Table 14. The instrumentation used for the operationalized variables is described in the paragraphs following Table 14.

TABLE 14. Measurements.

Concept	Operationalized Measure
Rate of Rewarming	Change of $T_{es}$ in $^{\circ}\text{C}$
Conductance	
a) Thermal circulation index	$\frac{T_{qrec} - T_{blanket}}{T_{es} - T_{qrec}}$ $T_{blanket}$ = Air temperature within the blanket
Body Mass	
a) Skinfold thickness	Chest + Forearm + Thigh + Medial Calf skinfold
b) Body-mass to -surface index	$\text{kg}/\text{m}^2$
Vasoactivity	
a) Forearm blood flow	Venous occlusion plethysmography
b) Skin temperature gradient	$T_{fa} - T_{if}$
Metabolic rate	$\text{O}_2$ consumption
Weighted Mean Skin Temperatures	
a) Quartermaster Corps	$T_{qrec} = 0.15 T_{calf} + 0.125 T_{front\ thigh} + 0.125 T_{medial\ thigh} + 0.125 T_{back} + 0.125 T_{chest} + 0.10 T_{cheek} + 0.07 T_{upper\ arm} + 0.07 T_{fa} + 0.06 T_{hand} + 0.05 T_{foot}$
b) Hardy and Dubois	$T_{h\&d7} = 0.07 T_{forehead} + 0.14 T_{fa} + 0.05 T_{hand} + 0.07 T_{foot} + 0.13 T_{calf} + 0.19 T_{front\ thigh} + 0.35 T_{abdomen}$
c) Teichner	$T_{teichner} = 0.149 T_{cheek} + 0.186 T_{medial\ thigh} + 0.186 T_{back} + 0.186 T_{front\ thigh} + 0.186 T_{chest} + 0.107 T_{upper\ arm}$
d) Palmes and Parker	$T_{p\&p} = 0.14 T_{cheek} + 0.19 T_{chest} + 0.19 T_{back} + 0.32 T_{front\ thigh} + 0.11 T_{fa} + 0.05 T_{hand}$
e) Ramanathan	$T_{ramanathan} = 0.3 T_{chest} + 0.3 T_{upper\ arm} + 0.2 T_{front\ thigh} + 0.2 T_{calf}$
f) Burton	$T_{burton} = 0.5 T_{chest} + 0.36 T_{calf} + 0.14 T_{fa}$

Note.  $T_{es}$  = esophageal temperature;  $T_{qrec}$  = Quartermaster Research and Engineering Company weighted mean skin temperature;  $T_{blanket}$  = blanket temperature;  $T_{es}$  = esophageal temperature;  $T_{fa}$  = forearm temperature;  $T_{if}$  = index finger temperature;  $T_{calf}$  = calf temperature;  $T_{front\ thigh}$  = front thigh skin temperature;  $T_{medial\ thigh}$  = medial thigh temperature;  $T_{back}$  = back temperature;  $T_{chest}$  = chest temperature;  $T_{cheek}$  = cheek temperature;  $T_{upper\ arm}$  = upper arm temperature;  $T_{hand}$  = hand temperature;  $T_{foot}$  = foot temperature;  $T_{h\&d7}$  = Hardy and Dubois weighted mean temperature;  $T_{forehead}$  = forehead temperature;  $T_{abdomen}$  = abdomen temperature;  $T_{teichner}$  = Teichner weighted mean temperature;  $T_{p\&p}$  = Palmes and Parker weighted mean temperature;  $T_{ramanathan}$  = Ramanathan weighted mean temperature;  $T_{burton}$  = Burton weighted mean temperature.

### Rate of Rewarming

The rate of rewarming was calculated as the  $^{\circ}\text{C}$  change in central temperature per minute (Table 14). For description of the rewarming pattern within an individual, the change in temperature was then calculated over 5 minute periods. For description of the rewarming pattern between individuals, the change in temperature was described based on the direction of change of  $T_{es}$  noted in all individuals, such as decreasing-, plateau-, or increasing- $T_{es}$  phase. For comparison between individuals, the  $^{\circ}\text{C}$  per minute change during similar phases was compared.

Central temperature was operationalized as  $T_{es}$ . The subject placed the tip of the esophageal thermistor in the back of her oral cavity and then swallowed the probe tip to below the atrial level. With the thermistor tip in the esophagus, the atrial level was determined using electrocardiography. The esophageal thermistor was attached to the chest lead of a cardiac monitor (Spacelabs, Model 512, Redmond, Washington). The esophageal probe was repositioned until the cardiac waveform, obtained from within the esophagus, displayed a bi-phasic P-wave which indicated atrial level.

All temperatures, including central and skin, were measured utilizing resistance thermometry. A metal at a specific temperature has a given resistance. Since a relationship exists between resistance and temperature in a metal, the resistance can be measured and converted to degree temperature (Mueller, 1941). The precision of the measurement is dependent upon the properties of the metal utilized such as the diameter of the wire as well as the type of metal (Mueller, 1941).

$T_{es}$  was measured with a Yellow Springs Instruments thermistor, model 520 (Yellow Springs, Ohio). The range of temperature measurement for the model

520 thermistor is -40 to 150° C with a time constant of 0.2 seconds. The thermistor reaches approximately 63% of a new reading within 0.2 second and 100% of the new reading in 1.0 second. The esophageal thermistor was calibrated in a well stirred water bath against a National Bureau of Standards (NBS) calibrated thermistor. The level of accuracy of the model 520 thermistor was within thousandths of a °C of the NBS calibrated thermistor. Data was collected every 15 seconds by the Keithley data acquisition system.

#### Conductance Measurement

Conductance is the transfer of heat between two surfaces, which in the case of a human are the body core and the environment. A direct measurement of conductance would require measurement of the rate of heat flow from the environment to the center of the body, the temperatures of both surfaces, and the cross-sectional area (Burton, 1934). Burton theorized that the thermal circulation index, which is a ratio of the external drop of temperature ( $T_{sk} - T_{environment}$ ) to the internal drop of temperature ( $T_{internal} - T_{sk}$ ), gives an indication of the state of heat loss from an individual.

$$\text{Thermal Circulation Index} = \frac{T_{sk} - T_{environment}}{T_{internal} - T_{sk}}$$

The larger the thermal circulation index value the greater the amount of heat flux out of the body. Burton's theory was supported by Daniels and Baker's (1961) research which reported a progressively lower thermal circulation index as the percentage of body fat increased at various sites in 31 males exposed to an air temperature of 15.2° C for 2 hours. Finally, Burton theorized, if the thermal circulation index increases within an individual, the increase in heat flux had occurred from an increased peripheral circulation.

Two other methods of measuring conductance are needle thermistory (Greenfield, Whitney & Mowbray, 1963) and heat flux transducers (Sessler &

Moayeri, 1990). Needle thermistors require placement of a needle thermistor into a soft tissue and on the skin surface. The temperature within the soft tissue and on the skin surface are measured and the amount of heat transfer is calculated. Heat flux transducers require placement of the transducer on the surface of the body. The difference in transducer resistance on the skin and on the environmental surface of the transducer is measured. The resistance may then be converted into temperature and heat transfer.

Conductance measurement by needle thermocouples or heat flux transducers is not feasible in a clinical setting. The conductance measurement must be relative to the entire body since the entire body surface is involved in the transfer of heat. Therefore, multiple thermocouples or transducers must be placed in an attempt to measure the entire body surface effect. In the clinical setting, placement of needle thermocouples is not routine practice or currently under the nursing realm. Also, the instrumentation for both needle thermocouples and heat flux transducers are not available in clinical areas. Finally, the technology for validating the heat flux transducers is not available in the clinical setting. Hence, for the purpose of this study, conductance was operationalized as the thermal circulation index (Burton, 1934). Burton did not operationalize the temperature measurement sites. Hence, the next sections will address the operationalization of temperature sites used in calculating the thermal circulation index.

Skin temperature. Controversy exists in the literature over the measurement of skin temperature. The controversy centers around what is the appropriate number of skin sites and which sites to measure to obtain mean skin temperature. Therefore, to best represent the mean skin temperature

large numbers of sites have been studied with varying recommendations. However, clinically a minimal number of sites are required.

Since only the anterior body surface is exposed to the convective air blanket therapy, the  $T_{sk}$  measurement should represent an anterior site or an average temperature of the anterior skin surface which is exposed to the therapy. Furthermore, the heat loss from the posterior surface via conduction was minimal (3 and approximately 11%; Nilsson, 1987 and Sessler, Stoen & Glosten, 1989, respectively). Therefore, predominately anterior skin sites were utilized for a mean skin temperature measurement.

Furthermore, conductance is a measurement of the ability to move heat through the tissues both by conduction, movement through the solid mass, and convection, movement through the vasculature. Therefore, one must use skin sites over areas in which body fat are represented. The chest, the medial thigh and the upper arm skin temperatures had the greatest correlation with the percent of body fat measured with calipers ( $r=-0.60$ ,  $r=-0.58$ ,  $r=-0.57$  at  $p<0.05$ , respectively; Daniels and Baker, 1961). The chest accounts for approximately 6% of the BSA and 5% of the total blood flow. The anterior thigh accounts for approximately 9.5% of the total BSA and 5% of the total blood flow. The upper arms account for only 4-5% of the total BSA and receives approximately 2% of the total blood flow in a basal state (Table 15). Hence, the medial thigh temperature has a moderate correlation with percent body fat and represents a large portion of the anterior body surface. Therefore, a single site which may be representative of  $T_{sk}$  is the medial thigh.

However, in the literature, when medial thigh  $T_{sk}$  was compared to mean skin temperature measured at varying numbers of sites, the relationship varies. When comparing medial thigh  $T_{sk}$  to a 15 site mean weighted skin

temperature (Mitchell & Wyndham, 1969), the medial thigh temperature was within  $0.2^{\circ}$  C only 11% of the time (no correlation reported). When comparing medial thigh  $T_{sk}$  to Hardy-Dubois' system of 7 site, mean, weighted skin temperature (Ramanathan, 1963), an  $r=0.94$  with a "very high" significance was reported (no  $p$ -value reported). Hence, the use of only one skin temperature, medial thigh  $T_{sk}$ , may not be a valid reflection of mean skin temperature.

TABLE 15. Blood Flow, BSA and Body Fat-Skin Temperature for Various Sites.

	<sup>a</sup> %Total Basal Blood Flow	<sup>b</sup> % of BSA	<sup>c</sup> r % Body Fat and Lowest Skin Temperature $p < 0.05$
Hands	12	5	(-0.27 NS)
Palm	6	2.5	
Dorsum	3.5	2.5	
Forearms	7	6	(-0.19 NS)
Upper arms	4	8	-0.57
Feet	10	7	(-0.09 toe, -0.05 arch NS)
Lower legs	12	14	-0.42 calf
Thighs	10	19	-0.42 lateral thigh -0.58 medial thigh
Trunk & buttocks	29	31	
Buttocks	7	5	
Back	11	13	-0.54
Chest & abdomen	11	13	-0.60 chest
Head	14	7	(-0.22 forehead NS)

<sup>a</sup>Hertzman & Randall, 1948. <sup>b</sup>Lund & Browder, 1944. <sup>c</sup>Daniels & Baker, 1961.

Hence, 6 methods of mean skin temperature, which represent anterior skin sites and sites over adipose tissue, as well as medial thigh were used. The mean skin temperature measurements were Quartermaster Research and Engineering Center ( $T_{qrec}$ ) 10-point weighted mean (Mitchell & Wyndham, 1969; Teichner, 1958), Hardy and Dubois 7-point weighted mean (Mitchell & Wyndham, 1969; Teichner, 1958), Palmes and Parker 6-point weighted mean (Teichner, 1958), Teichner 6-point weighted mean (Mitchell & Wyndham, 1969; Teichner, 1958), Ramanathan's 4-point weighted mean temperature

(Ramanathan, 1963) and Burton's 3-point weighted mean (Mitchell & Wyndham, 1969; Teichner, 1958) (Table 14). Since  $T_{qrec}$  represents the largest number of sites,  $T_{qrec}$  was utilized in calculating the Thermal Circulation Index. The remaining mean skin temperature measurements were evaluated for applicability of use in the clinical area.

The skin temperatures were measured with copper-constantan thermocouples (manufactured by Dave Miller, Department of Physiology, University of Washington) which were accurate within  $\pm 0.1^{\circ}$  C of the NBS thermistor. The thermocouples were taped to the skin surface. The 13 sites were located on the left side of the body and consisted of index finger, hand, forearm, upper arm, forehead, cheek, chest, back, abdomen, front thigh, medial thigh, medial calf, and foot.

Environment temperature. The environment temperature ( $T_{environment}$ ) was measured within the convective air blanket. A copper-constantan thermocouple was inserted into a small pore of the blanket, at the midpoint of the length of the blanket, in the longitudinal tube, which was just left of the midline of the blanket. The ambient temperature at the head of the warming bed was also measured both during cooling and rewarming.

Internal temperature. The internal temperature ( $T_{internal}$ ) was operationalized as  $T_{es}$ .  $T_{es}$  was discussed under the rate of rewarming section. Therefore, the thermal circulation index calculation was  $T_{qrec} - T_{environment} / T_{es} - T_{qrec}$ .

The ability to compare the conductance measurement between subjects was questionable. Conceivably, an individual who was vasodilated with a large percentage of body fat could have a similar conductance measurement to an individual who was vasoconstricted and a smaller percentage of body fat. The

degree of effect of body fat or vasoactive state on the ability to rewarm was unknown. Hence, it was unknown whether a conductance measure would assist in the determination of the ability to rewarm an individual post operatively. Therefore, measurements of body mass and vasoactivity were used to clarify the rewarming process.

### Body Mass

Body mass affects the rate of heat flux into or out of the body based on the absolute thickness of tissue and upon the relationship of body mass to body surface area (body mass index). The next sections will address body fat and body mass to surface area.

Skinfold thickness. Body fat can be measured by dissection, densitometry, measurement of total body potassium with  $^{40}\text{K}$  isotope, total body water, or skin fold thickness (Martin & Drinkwater, 1991). The former four methods require invasive procedures or technology which are not readily available in a hospital setting. Skin fold thickness is utilized within the hospital setting. However, skin fold thickness measurement is an indirect measurement of body fat with questionable validity and reliability.

Martin, Ross, Drinkwater and Clarys (1985) compared the skinfold thickness caliper reading at 14 sites with the total subcutaneous adipose tissue mass of 13 cadavers (6 males and 7 females). The sites with a high correlation ( $p < 0.05$ ) between the caliper reading and the total subcutaneous adipose tissue are listed in rank order: front thigh ( $r = 0.89$ ), sup-patellar ( $r = 0.85$ ), medial calf ( $r = 0.84$ ), rear thigh ( $r = 0.82$ ), forearm ( $r = 0.80$ ), medial thigh ( $r = 0.76$ ), chest ( $r = 0.64$ ), suprascapular ( $r = 0.61$ ), biceps ( $r = 0.58$ ), and subscapular ( $r = 0.54$ ) (Martin et al., 1985). The correlation of the caliper reading to the total subcutaneous tissue mass was insignificant at the triceps, abdomen, waist, and

pectoral sites. Therefore, the validity of skinfold thickness as a measurement of subcutaneous fat is greater at certain sites.

The distribution of body fat varies between individuals. Martin et al. (1985) described the skinfold thickness in subject #18 (female) as 44 mm at the front thigh and 20 mm at the abdomen and in subject #19 (female) the front thigh skinfold thickness was 23 mm and 35 mm at the abdomen. Therefore, a single skin fold thickness is probably not the most valid measurement of body fat. However, use of multiple sites with high skinfold thickness to total subcutaneous fat correlations would increase the validity of the measurement.

Many sites for measurement of body fat have been utilized. Edwards (1950) utilized 53 sites when describing the contribution of subcutaneous fat to the body contours. Hammond (1955) advocated measurements from 1 or 2 sites to obtain a representative measure of total body fat. Martin et al. (1985) did not advocate any specific number of sites for measurement of subcutaneous fat. Therefore, for this study, the choice of sites was based on sites which had a high correlation of total body subcutaneous fat and skinfold thickness as well as sites on the anterior surface of the body. Hence, chest, forearm, front thigh and medial calf were selected because of anterior surface representation, distribution of sites across areas with high adipose tissue, and good correlation with total subcutaneous adipose tissue.

The chest measurement site was a vertical fold along the anterior axillary line at the level of the xyphoid process of the sternum (Drinkwater, 1984). The forearm measurement site was a fold on the anterior surface of the forearm parallel to the long axis at the level of maximum girth (Drinkwater, 1984). The front thigh site was a vertical fold on the anterior surface, midway between the projection at the skin surface of the ligamentum inguinale and the mid

point of the patella (Drinkwater, 1984). The medial calf site was the vertical fold on the medial surface at the level of maximum girth (Drinkwater, 1984).

The reliability of a skinfold site measurement was threatened by compressibility of fat, instrument error and inter-observer error. The compressibility of the fat varies with the measurement site, such as the compressibility of the skinfold at the biceps was reported to be 63.8% of the thickness and the front thigh skinfold had a compressibility of 33.6% (Martin et al., 1985). Edwards, Hammond, Healy, Tanner and Whitehouse (1955) addressed instrument design issues. For the best reproducibility of measurement, the caliper should have a spring pressure of between 9 and 15  $\text{g/mm}^2$  between the open caliper range of 2 to 40 mm and that the spring pressure should not vary more than 2  $\text{g/mm}^2$  between the 2 to 40 mm range. Edwards et al. (1955) further stated that the accuracy of repeated measures by an observer were "markedly better for an experienced observer." The standard deviation for skin fold thickness measurement between different observers at the biceps site (spring pressure 10  $\text{g/mm}^2$ ) was 22.84 (logarithmic scale) as compared to the same measurement repeated by an observer of 9.26. Therefore, the routine measurement of skin fold thickness in a clinical setting is inappropriate at the current time. However, the use of skinfold thickness to describe the study population was vital to evaluating patterns of rewarming which may be affected by the amount of body fat. Therefore, skin fold thicknesses were measured by a single observer with the measurement repeated three times.

Body mass index. The relationship of body mass to body surface area is measured by determining the mass in kilograms and the body surface area based on the height and weight nomogram described by Dubois and Dubois.

The body mass index ( $\text{kg}/\text{m}^2$ ) has been found to have a significant relationship to cooling and rewarming (Vaughan et al., 1981; Erickson & Yount, 1991, respectively) in postoperative patients, but no information was found in the literature for normal healthy individuals who were cooled. Hence, a prestudy measurement of weight in kilograms and height in centimeters was used. The individuals were weighed wearing only a cotton body suit. The height was determined with the individual standing in stocking feet with their back against a wall. The individual's heels were together and arms at sides. The individual was instructed to look straight forward and take in a deep breath. A perpendicular line was created between the top of the individual's head and the wall by placing a square on top of the head. The height measurement was taken from the point where the perpendicular line meets the wall to the floor.

#### Vasoactivity

A direct measurement of vasoactivity can not be made without exposing the vessel to the environment. Hence, in vivo measurement of vasoactivity utilizes measurement techniques which measure skin blood flow, indicative of a change in vasoactivity. Skin blood flow measurement techniques include vascular occlusion plethysmography (Greenfield, Whitney & Mowbray, 1963), laser doppler (Johnson, Taylor, Shepherd & Park, 1984; Haumschild, 1986), skin temperature (Felder, Russ, Montgomery & Horwitz, 1954) and skin temperature gradient (Rubinstein & Sessler, 1990). Vascular occlusion plethysmography, utilizing a Whitney strain gauge, is frequently utilized in laboratory studies of vasoactivity, but the equipment and the technical requirements make plethysmography unavailable for use in clinical practice. The laser doppler is more portable and requires less technical skill, but the laser doppler

measurement does not measure the actual blood flow. The laser doppler technique displays a waveform which can be quantified as a percentage of change in the waveform height during the measurement session, but no comparison between two different measurement sessions can be made. Thus, the laser doppler is unacceptable as a measurement of the vascular activity when attempting to determine the degree of vasoconstriction at a given moment.

Skin temperature of the toes has been found to be representative of blood flow to the toes particularly at low flows (Felder et al., 1954). Felder et al., studied 12 normal individuals in  $T_{amb} 20.5 \pm 2^{\circ} C$  and determined the relationship between toe  $T_{sk}$  and toe skin blood flow. At toe  $T_{sk}$  between 20 and 29 $^{\circ} C$ , a linear relationship was found. Between 29 and 31 $^{\circ} C$  toe  $T_{sk}$ , a curvilinear relationship was found returning to a linear relationship between 31 and 32 $^{\circ} C$  toe  $T_{sk}$ . Felder et al. did not report any statistical analysis of the relationships identified. Felder et al. pointed out that skin temperature does lag behind the change in flow, which requires obtaining a steady state of temperature before a measurement was reliable. Stabilized skin temperature was operationalized as an 18 minute period in which the temperature varied no more than  $\pm 0.5^{\circ} C$ .

Skin temperature gradient of the finger and forearm had a high correlation ( $r = 0.98$ ) with the total finger tip blood flow (Rubinstein & Sessler, 1990). Nineteen healthy volunteers between the ages of 25 and 57 years of age were studied. The group consisted of 15 male and 4 female subjects. A measurement of the total blood flow to the fingertip was made using occlusion plethysmography. The subject's skin temperature was influenced by two circulating water blankets which were placed anteriorly and posteriorly

(excluding the study arm from blanket coverage) with random settings between 30 and 40° C. The flows varied by 30 fold over a temperature gradient range of -1 to 8° C. Therefore, skin temperature gradient of the forearm - fingertip may serve as a clinical measurement which could be utilized as an indicator of vasoconstriction. However, skin temperature is not usually monitored in the clinical setting, but the instrumentation does exist within the clinical area for monitoring temperature.

For the purposes of this study, vasoactivity was measured by forearm vascular occlusion plethysmography, which measures forearm blood flow (FBF), and by forearm to index finger tip temperature gradient. Forearm blood flow was measured on the right arm, while forearm to index finger temperature gradient was measured on the left arm to avoid any effect from vascular occlusion for FBF measurement. The measurements were collected during a baseline interval (prior to inflation of the convective air blanket) and during the active warming interval (following inflation of the convective air blanket).

Vascular occlusion plethysmograph is a measurement of the resistance in a mercury in silastic strain gauge, which is positioned around the forearm at the point of greatest girth. As the forearm filled with blood, the silastic band stretched and the resistance was measured 20 times/second. During the measurement, the venous outflow from the arm was occluded by a cuff on the upper arm which was inflated to 10 mmHg above the diastolic pressure. The arterial and venous flow to the hand were restricted to a minimum by a second cuff which was placed at the wrist and inflated to 180 mmHg. The inflation of the cuffs were cycled with the upper arm cuff inflation and deflation twice a minute obtaining forearm blood flow measurements. The wrist cuff was

inflated for 4 minutes and deflated for 1 minute. During deflation of the wrist cuff, no forearm blood flow measurements were made and the right hand was gently massaged.

The strain gauge was mechanically and electrically calibrated prior to and following the experiment. The mechanical calibration involved stretching the gauge 2 mm and measuring the change in resistance. The electrical calibration involved changing the resistance by 1% and measuring the electrical output (Bregelmann & Savage, 1986). The calibration process insured that a 0.5% change in length of the gauge resulted in a 1% change in the resistance measurement. The maximal amount of difference in the pre- and post-experiment calibrations was 2.5% or 0.06 volts.

#### Metabolic Rate

Metabolic rate can be measured directly with calorimetry or indirectly by measuring the oxygen consumption ( $VO_2$ ) over a given time period. Calorimetry is the measurement of body heat production by placing the individual in an enclosure and measuring the temperature changes within the enclosure. Calorimetry is not available in most laboratory and clinical settings. Metabolic rate is more commonly measured utilizing the indirect method of measuring oxygen consumption. The fraction of oxygen inspired and expired over a given time period are measured to determine the amount of oxygen consumed during the time period (Luce, Tyler & Pierson, 1984). The metabolic rate is not constant. Therefore, continuous measurement of oxygen consumption over the rewarming process was required to determine whether rewarming was related to an increase in the metabolic rate or to the rewarming therapy. Various instruments are commercially manufactured which provide the capabilities of continuous measurement.

The continuous measurement of expired oxygen required a compliant subject to wear a tight-fitting, inflatable mask which covered the mouth and the nose. The mask was held in place by a 4 strap head-harness which fit around the back of the head and connected to the mask. All expired air passed through the circuitry of the instrument for measurement of expired oxygen. The oxygen consumption was measured with the Quinton, QPlex I (Seattle, Washington). The oxygen gas analyzer was a Zirconia Oxide sensor with a reported absolute oxygen measurement accuracy of  $\pm 0.05\%$  over the range of 10 to 35% oxygen. The recommended environment for instrument operation was 10 to 40° C. The measurement display rate was set for once every 20 seconds. However, the instrument analyzes data for every breath and computes an average value for the time frame selected.

The instrument was calibrated prior to use in each experiment. Across all experiments, the pre-experiment calibration results indicated no measurement error, between measured and expected values, when measuring percent of CO<sub>2</sub> and O<sub>2</sub> test gases, but a  $\pm 0.01$  volt maximum difference between measured and expected CO<sub>2</sub> (target values of 0 and 3.37 volts, low and high CO<sub>2</sub> calibration gases, respectively), and a  $\pm 0.01$  volt maximum difference between measured and expected O<sub>2</sub> (target values 1.00 and 2.50 volts, low and high O<sub>2</sub> calibration gases, respectively).

#### Procedure

The study protocol included a cooling period, a transfer period and a warming period which are depicted in Figure 1. The cooling and warming periods consisted of a set protocol for environmental temperature and minutes at that temperature (described in the following procedure). However, the minutes of the transfer period were dependent upon how smoothly the subject

with instrumentation was moved from one bed to the next which resulted in a different number of minutes for transfer time in each subject. Hence, Figure 1 does not include the minute value on the Time axis, because of variability between subjects, as well as ambient temperature during the transfer period is represented as an average temperature for all subjects. The actual procedure description follows.

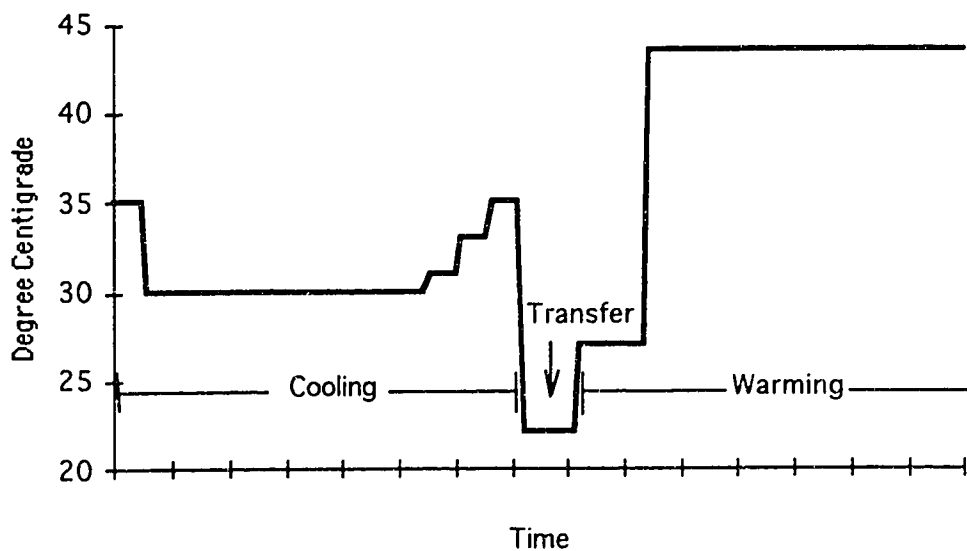


FIGURE 1. Study Periods: Cooling, Transfer and Warming.

1. The subjects were screened over the telephone for acceptance into the study. If the individual met the inclusion criteria, the study procedure was described. If the individual was interested in being a subject, demographic data were gathered at the time of the phone interview.
2. The subject was instructed to eat a small, low fat meal, at least 2 hours prior to coming to the laboratory, and no smoking, no caffeine intake or no heavy exercise for 4 hours prior to the study.
3. The subject was instructed to come to the laboratory at 1 PM on the day of the study.
4. On the day of the study, the metabolic cart was warmed up for at least 30 minutes prior to calibration. A low and a high gas calibration was performed. The low gas calibration consisted of 10% oxygen and 90% nitrogen. The high gas calibration consisted of 25% oxygen, 5% carbon dioxide and 70% nitrogen. The expected calibration measurement value

was within  $\pm 0.003$  volts of predicted when measuring oxygen and  $\pm 0.005$  volts of predicted when measuring carbon dioxide.

5. Upon arrival of the subject, the study procedure was explained, a tour of the laboratory was given, and a written consent was obtained.
6. The subject changed into a body suit with velcroed shoulder straps.
7. The subject was weighed and height measured.
8. The skinfold thickness was measured.
9. Instrumentation:
  - a. The appropriate size face mask was selected.
  - b. The skin thermocouples were taped to the skin.
  - c. The electrocardiogram skin patches were applied.
  - d. The esophageal thermistor was swallowed.
  - e. The subject reclined on the cooling bed and was covered with a cotton blanket.
  - f. The esophageal thermistor was repositioned observing the electrocardiogram waveform.
  - g. The Whitney strain-gauge was applied to the right arm which included suspending the right arm above atrial level.
  - h. The subject's blood pressure was measured on the left arm to determine the appropriate pressure for venous occlusion.
  - i. The spray nozzles were positioned over the subject with a separate spray system suspended over the right arm.
  - j. The cotton blanket was removed and the subject and spray nozzles were covered with a plastic wrap which assisted in directing the water spray over the subject.
  - k. The oral suction catheter was placed.
  - l. The face mask was placed.
10. Cooling:
  - a. The water was turned on at  $35^{\circ}\text{C}$ .
  - b. The metabolic cart tubing and valve were attached to the mask.
  - c. All measuring instruments were started with a synchronized start.

- d. After 5 minutes of measurements at 35<sup>o</sup> C water, the water temperature was turned down to 30<sup>o</sup> C (Figure 1).
  - e. If the metabolic rate rose and remained elevated and the subject was visibly shivering after 10 minutes of 30<sup>o</sup> C water temperature, the water temperature was adjusted to 30.5<sup>o</sup> C.
  - f. The right hand was massaged during the one minute of cuff deflation in each 5 minute cycle of forearm blood flow measurement.
  - g. After 45 minutes at 30<sup>o</sup> C water temperature, the water temperature was increased to 31<sup>o</sup> C for 5 minutes (Figure 1).
  - h. The Bair Hugger® machine was turned on with a heat setting of high and the hose directed into the air away from the warming and cooling beds.
  - i. After 5 minutes at 31<sup>o</sup> C water temperature, the water temperature was increased to 33<sup>o</sup> C for 5 minutes (Figure 1).
  - j. After 5 minutes at 33<sup>o</sup> C water temperature, the water temperature was increased to 35<sup>o</sup> C for 5 minutes (Figure 1).
  - k. The water was turned off, the instrument measurements stopped, and cables were prepared for the transfer of the subject from the cooling bed to the warming bed.
11. Transfer:
- a. The plastic cover and water spray apparatus were removed.
  - b. The subject stood and walked approximately 7 foot towards the warming bed with one person attending to cables in the transfer process.
  - c. The subject was enclosed in a terry cloth robe which velcroed at the shoulders and covered the entire anterior and posterior body, but remained open at the sides.
  - d. The investigator reached under the terry cloth robe and gently removed the wet body suit by unfastening the velcro at the shoulders and pulling the suit down off the subject's body.
  - e. The investigator patted the subject's skin dry.
  - f. The investigator reached under the terry cloth robe and placed a cotton gown over the subject's body. The terry cloth robe was removed.

- g. The subject then reclined on the foam mattress which served as the warming bed.
- h. The subject was covered with the full-body, deflated, convective air blanket (Bair Hugger®, Model 300 blanket, Augustine Medical, Inc., Eden Prairie, Minnesota) and two flannel blankets.
- i. The right arm was positioned using foam blocks under the upper arm to elevate the arm above atrial level. The arm was bent at the elbow and the wrist positioned across the subject's abdomen.
- j. All thermocouples and instruments were reconnected.
- k. The blankets were tucked in around the sides and the foot. The subject's head remained exposed with the top portion of the convective air blanket fitting up along side of the head.

## 12. Warming:

- a. All measuring instruments were started with a synchronized start.
- b. The forearm blood flow measurement was observed for the potential of repositioning the arm to optimize the flow measurements.
- c. A 10 minute baseline interval of measurement occurred in which the convective air warming blanket was maintained in the deflated state (Figure 1).
- d. After 10 minutes of baseline measurement, the heated Bair Hugger® machine was attached to the convective air blanket (Figure 1).
- e. A 50 minute, active heating interval occurred. At the end of the 50 minutes, a comparison of the pre-cooling and current esophageal temperatures was made. If the esophageal temperature was within  $0.2^{\circ}$  C of pre-cooling, the study protocol was stopped. If the temperature was below the desired value, the subject was informed and requested to remain under the warming blanket for additional time.
- f. Whitney strain-gauge circumference measurement was made with the gauge in place on the arm.
- g. All instrumentation was removed.
- i. The subject changed back into street clothing. A short discussion was held to determine the subject's current physical state and obtain final information for subject payment.

### Analysis

The following variables were described for each individual: age, height and weight, BMI, skinfold thickness, body fat distribution, and menstrual phase.

Esophageal temperature and oxygen consumption were revised using a locally weighted smoothing function (LOWESS) from SPlus (Seattle, Washington). The raw esophageal temperature data displayed spikes which were from the subject swallowing. To remove the swallowing artifact, smoothing using 5, 10 and 20% of the data points was calculated and graphed with the raw data. The graph was inspected for the % of smoothing that removed the swallowing artifact, but maintained the overall trend seen in the raw data. Hence, for analysis, the esophageal temperature was smoothed using 20% of the data points in which 10% of the data points in time were prior to the point of interest and 10% followed the data point of interest.

Oxygen consumption data were captured at a 20 second interval. To resolve some of the normal variation seen in oxygen consumption when a short measurement interval is utilized, oxygen consumption was smoothed using 10% of the data points in which 5% of the data points in time were prior to the point of interest and 5% followed the data point of interest. Oxygen consumption data were initially analyzed using 5, 10 and 20 % smoothing and graphed with the raw data. The graph was inspected for the % of smoothing which continued to represent the raw data pattern, but removed some of the measurement to measurement variation.

Changes in esophageal temperature were calculated over 1 minute and 5 minute intervals during the warming period. The smoothed  $T_{es}$  data were graphed for the first four subjects. The  $^{\circ}\text{C}$  change/ 5 minutes was then manually marked on the graphs. The graphs were visually inspected by 5 minute intervals for direction of the trend line (decreasing slope, flat line, increasing slope). Finally, the  $^{\circ}\text{C}$  change/ 5 minutes was compared with the direction of trend line. A decreasing or increasing slope was visible when the

$^{\circ}\text{C}$  change/ 5 minutes was greater than  $0.01^{\circ}\text{C}$ . Hence, the definition of esophageal temperature change was a difference in temperature greater than  $0.01^{\circ}\text{C}$  over a 5 minute period when defining the pattern of esophageal temperature. Three  $T_{\text{es}}$  phases were defined which were  $T_{\text{es}}$ -decreasing, -plateau and -increasing for all subjects.

For description of the actual minute at which change in direction of  $T_{\text{es}}$  began, the difference in esophageal temperature over a minute was calculated, such as the minute at which esophageal temperature began to rise. Finally, since the physiological changes (time of the beginning of the  $T_{\text{es}}$ -decreasing phase) did not occur in relation to the beginning of the active warming interval, the change in  $T_{\text{es}}$  during the baseline interval was also analyzed for all subjects.

The patterns for  $T_{\text{es}}$ ,  $T_{\text{qrec}}$ , FBF and metabolic rate were described utilizing the time increments associated with each subject's  $T_{\text{es}}$  phases. Temperature data was represented as change over time, while FBF and metabolic rate mean values were calculated.

Relationships were described in variables that were measured across time by describing the correlation between variables within the  $T_{\text{es}}$  phase. The relationships of episodic variables, such as thermal circulation index, body mass index or skinfold thickness, with across time variables, such as  $T_{\text{es}}$ , were compared by correlating the episodic measurement with a measure that described the across time variable. For example, body mass index was correlated with the rate of change in  $T_{\text{es}}$  across the entire warming period.

The least number of skin sites which best represent  $T_{\text{qrec}}$  were analyzed using multiple regression by the BMDP statistical package which loads all subjects data across time. The program looks for the best fit for all subjects

across all points in time to identify the one variable that best describes the relationship. The program then proceeds to identify the two variables that best describes the relationship, etc. With each step of variable entry into the model, a  $r^2$  and adjusted  $r^2$  value are reported.

Variables measured across time for each individual were graphed and are presented in Appendix D - K. Graphs of variables across subjects were also created and are presented within the body of Chapter IV.

## CHAPTER IV

### Results

The purpose of this study was to describe the rewarming process in women between 40 and 60 years of age. The variables of interest were esophageal temperature, skin temperature, forearm blood flow, oxygen consumption, body size and conductance. This study was conducted in a laboratory where there are fewer limits on instrumentation than in a clinical environment. In this study measurements which could be utilized in a clinical area were also tested.

#### Sample Description

The study included 8 healthy women between the ages of 40 and 56 (Table 16). Subject 1 served as the pilot subject for testing the study protocol which was revised following the experiment. However, when reviewing the data from all subjects, the response of esophageal temperature to the cooling and warming process of Subject 1 was similar to that of other subjects. Therefore, for the purposes of analysis Subject 1 was reported as one of eight subjects.

The cooling protocol for Subject 1 was a water temperature of 28° C during the 45 minute period with the 3 5-minute increments increase in water temperature of 31° C, 33° C and 35° C. The subject found the water temperature to be uncomfortable during the study, but agreed to continue. After the study was completed, discussion with the subject lead to the decision to change the cooling protocol such that the water temperature during the 45 minute period was set at 30° C (Figure 1) instead of 28° C.

During the warming period for Subject 1, the convective-air warming machine was set on high, but the temperature within the blanket averaged 39° C which was lower than the expected temperature based on the setting and on the blanket temperature seen in other subjects. Furthermore, the

thermocouple within the warming blanket of Subject 1 failed and was replaced during the study.

TABLE 16. Demographic Data.

Sub- ject	Age (yrs)	Height (cm)	Weight (kg)	BSA	BMI (kg/ m <sup>2</sup> )	Skin fold (mm)	Menstrual cycle (day)	Smoke (pack/ day)
1	44	165.1	51.8	1.56	33.30	40.0	18	none
2	40	170.2	80.9	1.93	42.01	87.0	7	none
3	49	162.6	59.1	1.63	36.25	65.0	ph <sup>a</sup>	none
4	41	163.8	60.5	1.66	36.53	69.5	6	none
5	44	166.4	65.0	1.73	37.65	56.0	7	none
6	42	168.9	52.7	1.60	33.02	47.5	7	none
7	49	170.2	64.1	1.75	36.74	50.0	ph <sup>b</sup>	1
8	56	160.7	67.7	1.71	39.53	74.0	ph <sup>c</sup>	none
Mean	45.6	166.0	62.7	1.70	36.88	61.3		
SD	5.37	3.57	9.26	0.11	2.98	15.59		

<sup>a</sup>partial hysterectomy, no medications. <sup>b</sup>partial hysterectomy, estrogen 0.156 qd for 25 days and no estrogen for remainder of month, study day = day 22 of medication cycle. <sup>c</sup>partial hysterectomy, estrogen 0.625 qd continuously.

All subjects met the inclusion criteria when surveyed over the phone. Subject 7 had a cough at the time of the telephone survey and described the cough as "having a cold". Approximately a week after the initial telephone survey, Subject 7 was contacted to discuss her health status and set up a study appointment. During the conversation, Subject 7 assured the investigator that she was fully recovered from the cold. On the day of study, Subject 7 did have an occasional cough which she dismissed as normal. Based on Subject 7's history of smoking and her physical presentation, the investigator and others present in the laboratory during the study suspect that Subject 7 had pulmonary changes which had not been diagnosed as a chronic problem.

All subjects met the  $\pm 20\%$  of ideal body weight criteria. The subjects' body weight ranged from 118% of ideal body weight in Subject 2 to 99.4% of ideal body weight in Subject 6 with skinfold thickness at four sites ranging from 87 mm in Subject 2 to 40 mm in Subject 1. The body mass index was largest in Subject 2 and smallest in Subject 6 (Table 16).

Normal physical activity level was not formally measured, but informal discussion was held to obtain some idea of the subject's physical activity. For Subject 1, no information was obtained. Subject 2 does not enjoy or include physical activity in her daily routine. Subjects 3, 4, 5 and 6 included walking, biking, hiking, cross country skiing and/or kayaking in their weekly activities. Subject 7 discussed physical activities while on vacation, but did not describe routine activities. Subject 8 was beginning a weight loss program which included walking.

#### Environmental Temperatures

The range of mean ambient temperatures within the laboratory during all the experiments was from 21.69<sup>o</sup> C for Subject 4 to 25.82<sup>o</sup> C for Subject 7 (Table 17). The experiment with the greatest change in ambient temperature was in Subject 3 in which the ambient temperature had a 2.6<sup>o</sup> C range during the warming period of the experiment. The experiments in which the ambient temperature was most stable were Subjects 5 and 6 where the ambient temperature displayed a 0.77<sup>o</sup> C range.

The warming blanket temperature measured with a thermocouple inserted inside the midpoint of the blanket was reported during the period of deflation, minutes 0 to 10, and during the period of inflation, minutes 12 to 60 (Table 18 and Figure 2). A 2 minute period for inflation of the blanket was not included in the mean temperature reported in Table 18. The convective air heat source

was prewarmed to avoid exposing the subject to cool air while the machine warmed. The convective air machine was turned on to warm at least 5 minutes prior to the end of the cooling period. When the convective air machine was connected to the warming blanket the temperature within the blanket immediately began a sharp rise in temperature over a 2 minute period with a more gradual rise over the next 1 to 2 minutes (Figure 2).

TABLE 17. Ambient Temperature During Warming.

Subject	Ambient Mean (°C)	Standard Deviation	Minimum	Maximum
1	22.21	0.14	21.87	22.68
2	22.97	0.16	22.60	23.68
3	23.84	0.37	23.31	25.89
4	21.69	0.28	21.04	22.35
5	22.84	0.16	22.49	23.26
6	23.57	0.13	23.29	24.06
7	25.82	0.22	25.33	26.44
8	24.01	0.17	23.57	24.55
Mean	23.37	1.27		

The mean blanket temperature for all experiments during baseline was 28.62° C. The mean blanket temperature during active warming was 44.22° C. The blanket temperature during the sixth experiment was higher than expected on a setting of high. During experiment six, the skin temperatures were observed closely and the subject was asked about her level of comfort. Subject 6's skin temperatures remained at or below 40° C, so the warming blanket setting was not reduced.

TABLE 18. Warming Blanket Temperature.

Sub- ject	Baseline <sup>a</sup>				Active Warming <sup>b</sup>			
	0 to 10 min		Min	Max	12 to 60 min		Min	Max
	Mean (°C)	SD			Mean (°C)	SD		
1	None <sup>c</sup>				39.00	0.20	38.23	39.27
2	29.37	0.21	28.84	29.63	44.09	0.71	40.22	44.87
3	28.07	0.26	27.46	28.33	44.57	0.80	41.20	45.91
4	26.06	0.18	25.65	26.29	43.16	0.49	41.00	43.76
5	28.41	0.16	28.15	28.74	44.71	0.36	43.24	45.22
6	29.62	0.22	29.19	29.94	47.22	0.25	46.37	47.76
7	29.58	0.46	28.34	30.06	45.20	0.49	43.37	45.83
8	29.22	0.12	28.98	29.41	45.78	0.36	44.13	46.32
Mean	28.62	1.27			44.22	2.42		

Note. SD = Standard Deviation; Min = Minimum; Max = Maximum.

<sup>a</sup>Temperature within deflated blanket, not connected to warming device.

<sup>b</sup>Temperature within inflated blanket, connected to warming device.

Measurements between 10 and 12 minutes were excluded to allow complete inflation of the blanket. <sup>c</sup>Blanket thermocouple failed, replaced after 30 minutes.

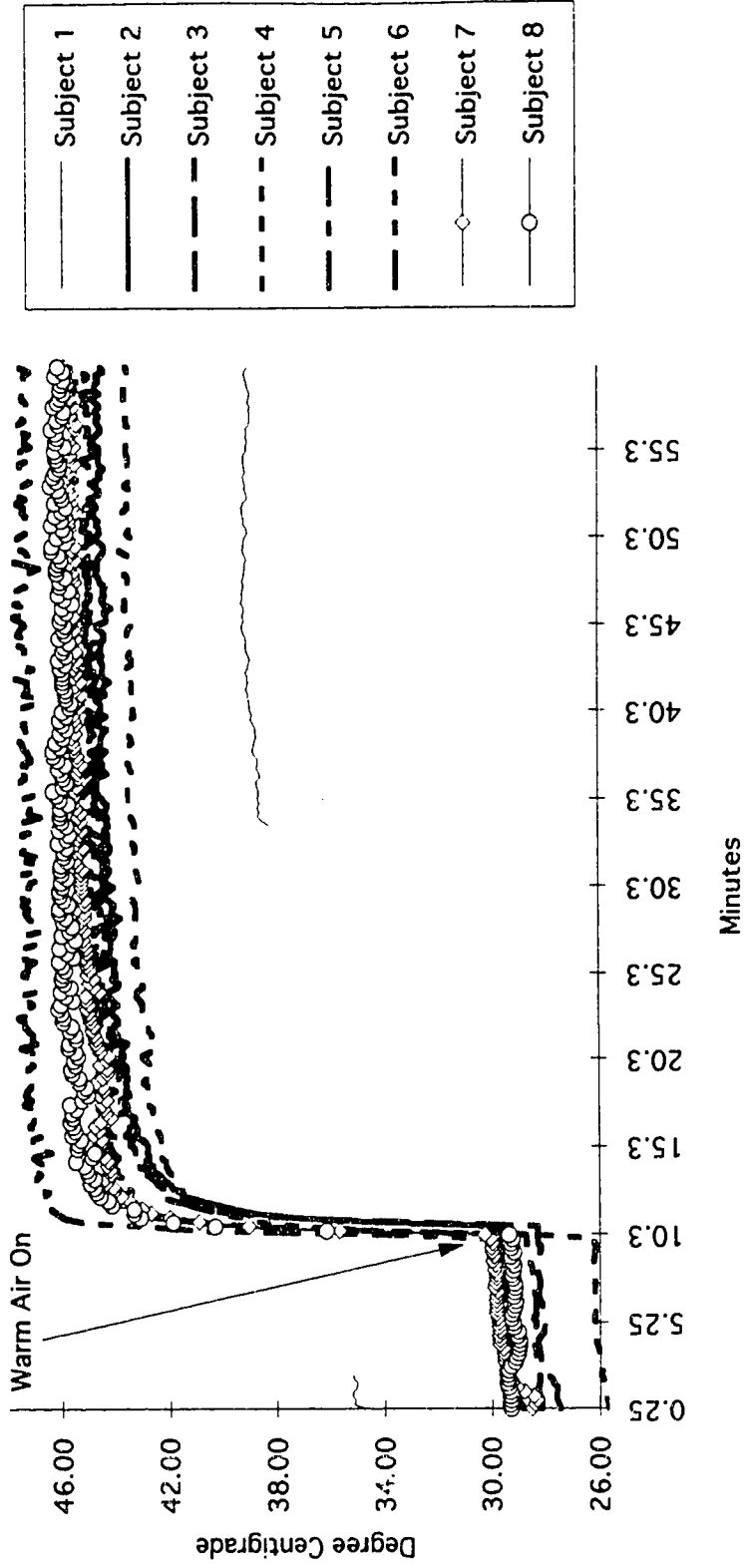


FIGURE 2. Warming Blanket Temperature.

### Patterns During the Warming Period

The following sections will address the first study question which was to describe the patterns of rewarming defined by the trend in esophageal and mean skin temperatures, forearm blood flow and metabolic rate prior to and following the application of the warmed convective air blanket.

#### Esophageal Temperature

The amount of drop in esophageal temperature ( $T_{es}$ ), during cooling, was separated into three groups (slight, mild and moderate) based on the natural split into categories created by the data (Table 19). Esophageal temperature decline during the cooling period was slight in Subjects 2, 5 and 8, mild in Subjects 1, 3 and 7 and moderate in Subjects 4 and 6 (Table 19). Subjects with a slight decrease in  $T_{es}$  had the greatest body mass index (BMI), but not necessarily the largest skinfold thickness (Table 16) which indicates a greater body size, but not necessarily a greater amount of body fat. Subjects with a mild to moderate decrease in  $T_{es}$  had smaller BMI than those subjects with a slight decrease in  $T_{es}$ , but between the subjects with the mild and moderate decrease there was not a distinction of BMI order. Menstrual cycle did not distinguish between subjects with a slight to a moderate decrease in  $T_{es}$ . Subjects on day 6 or 7 of their cycle experienced either a slight or a moderate decrease in  $T_{es}$  (Tables 16 and 19).

During cooling, all subjects experienced a rise in  $T_{es}$  following exposure to the 30° C water.  $T_{es}$  either plateaued or had a slight drop before the incremental increases in water temperature, but the greatest fall in  $T_{es}$  was noted during the last 3 5-minute increments in water temperature (Figure 3). At the beginning of the warming period, the subjects'  $T_{es}$  values were not consistently lower than their  $T_{es}$  values at the beginning of the cooling

period. The  $T_{es}$  value at the beginning of the warming period was either less than, equal to or greater than the  $T_{es}$  had been at the beginning of the cooling period (Table 19 and Figure 3).

TABLE 19. Esophageal Temperature During Cooling and Warming.

Subject	Cooling Period				Transfer Period (Minutes)	Warming Period		
	Begin (°C)	End (°C)	Drop (°C)	Drop		Begin (°C)	Afterdrop (°C)	End <sup>a</sup> (°C)
1	37.26	37.05	-0.21	Mild	35	37.11	-0.21	37.12
2	36.65	36.57	-0.08	Slight	25	36.89	-0.33	36.73
3	37.31	37.11	-0.20	Mild	13	37.14	-0.13	37.17
4	36.86	36.29	-0.57	Moderate	17	36.33	-0.06	36.54 <sup>b</sup>
5	36.88	36.86	-0.02	Slight	20	36.88	-0.06	37.11
6	36.82	36.37	-0.45	Moderate	25	36.53	-0.12	36.79
7	36.94	36.65	-0.29	Mild	18	36.99	-0.27	37.23
8	36.54	36.45	-0.09	Slight	27	36.62	-0.24	36.62
Mean	36.91	36.67	-0.24		22.5	36.81	-0.18	36.91
SD	0.27	0.31	0.19		6.93	0.29	0.10	0.27

<sup>a</sup>Temperature at the end of the 60 minute warming period. <sup>b</sup>Subject 4 warmed for a total of 75 min with final  $T_{es}$  of 36.77 °C

All subjects experienced the process of transfer from the cooling- to the warming-bed. The amount of time to transfer ranged from 13 to 35 minutes depending upon the amount of difficulty moving cables and equipment as the subject transferred and the amount of time to verify instrument connections once the subject was positioned on the warming bed (Table 19). Subjects 2 and 4 voided during the transfer period.

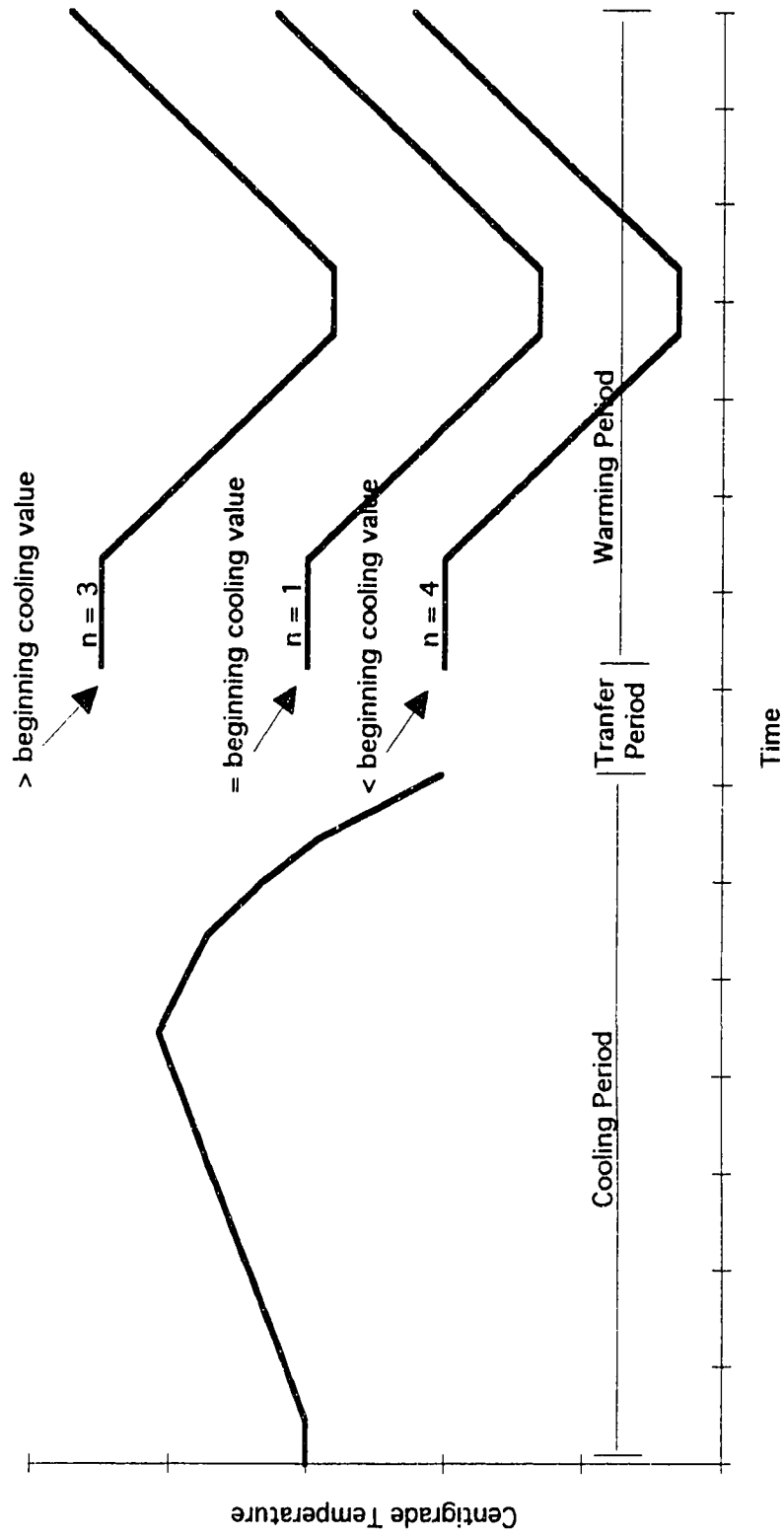


FIGURE 3. Esophageal Temperature Schema During Cooling and Warming.

The raw  $T_{es}$  data was first smoothed using SPlus (Seattle, Washington) to remove the spikes in the data which were from swallowing. To describe the pattern of change seen in  $T_{es}$ , the difference in  $T_{es}$  over 5 minute increments was calculated. On a graph of the smoothed data, the 5 minute time increments and the degree C change in  $T_{es}$  during the 5 minute increment were marked. The graph was then visually inspected for the time at which the direction of the slope of the line changed. When the change in  $T_{es}$  was equal to or less than  $0.01^{\circ}\text{C}$  over a 5 minute increment a horizontal line resulted, but when the change was greater than  $0.01^{\circ}\text{C}$  either an increasing or a decreasing slope in the line was visually evident. Hence, the definition of increasing or decreasing  $T_{es}$  over time was described as a change over a 5 minute period of greater than  $0.01^{\circ}\text{C}$ . Figure 4 demonstrates the esophageal temperature phases identified during the warming period.

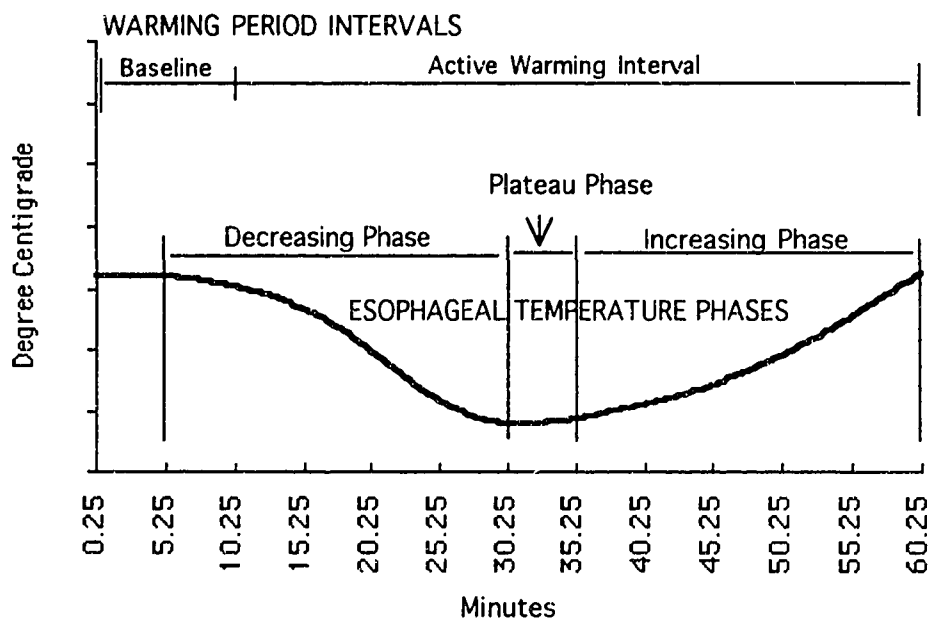


FIGURE 4. Esophageal Temperature Phases During Warming Period.

All subjects experienced a decline in  $T_{es}$  during the warming period (Table 19 and Figure 5). The beginning of the decrease in  $T_{es}$  varied from immediately upon entering the warming period up until 5 minutes after the active warming interval (warming blanket inflation) occurred (Table 20 and Figure 5). Following the decreasing  $T_{es}$  phase, all subjects, except Subject 6, experienced a 5 to 10 minute plateau phase of  $T_{es}$  where there was no change in  $T_{es}$ . All subjects experienced a period of increasing  $T_{es}$  which followed the plateau phase. All subjects at the end of warming experienced a  $T_{es}$  which was at least within  $0.15^{\circ}$  C of their  $T_{es}$  at the beginning of cooling.

TABLE 20. Esophageal Temperature Pattern During Warming Period.

Subject	Decreasing <sup>a</sup> (minute)	Plateau <sup>a</sup> (minute)	Increasing <sup>a</sup> (minute)
1	0.25 to 30.25	30.25 to 35.25	35.25 to 60.25
2	0.25 to 25.25	25.25 to 30.25	30.25 to 60.25
3	10.25 to 30.25	30.25 to 40.25	40.25 to 60.25
4	15.25 to 30.25	30.25 to 35.25	35.25 to 60.25
5	15.25 to 20.25	20.25 to 25.25	25.25 to 60.25
6	5.25 to 25.35	None	25.35 to 60.25
7	0.25 to 25.25	25.25 to 30.25	30.25 to 60.25
8	5.25 to 30.25	30.25 to 35.25	35.25 to 60.25

<sup>a</sup>Based on esophageal temperature change over 5 minute periods.

In the literature, the phenomenon of a decreasing central temperature during warming is identified as afterdrop. All subjects in this study experienced afterdrop (Neufer, Young, Sawka & Muza, 1988; Webb, 1986). For this study, afterdrop was the phenomena of decrease in  $T_{es}$ , during the warming period, which included the time from the beginning of  $T_{es}$  decreasing, until  $T_{es}$  returned to the  $T_{es}$  value at which the decrease started.

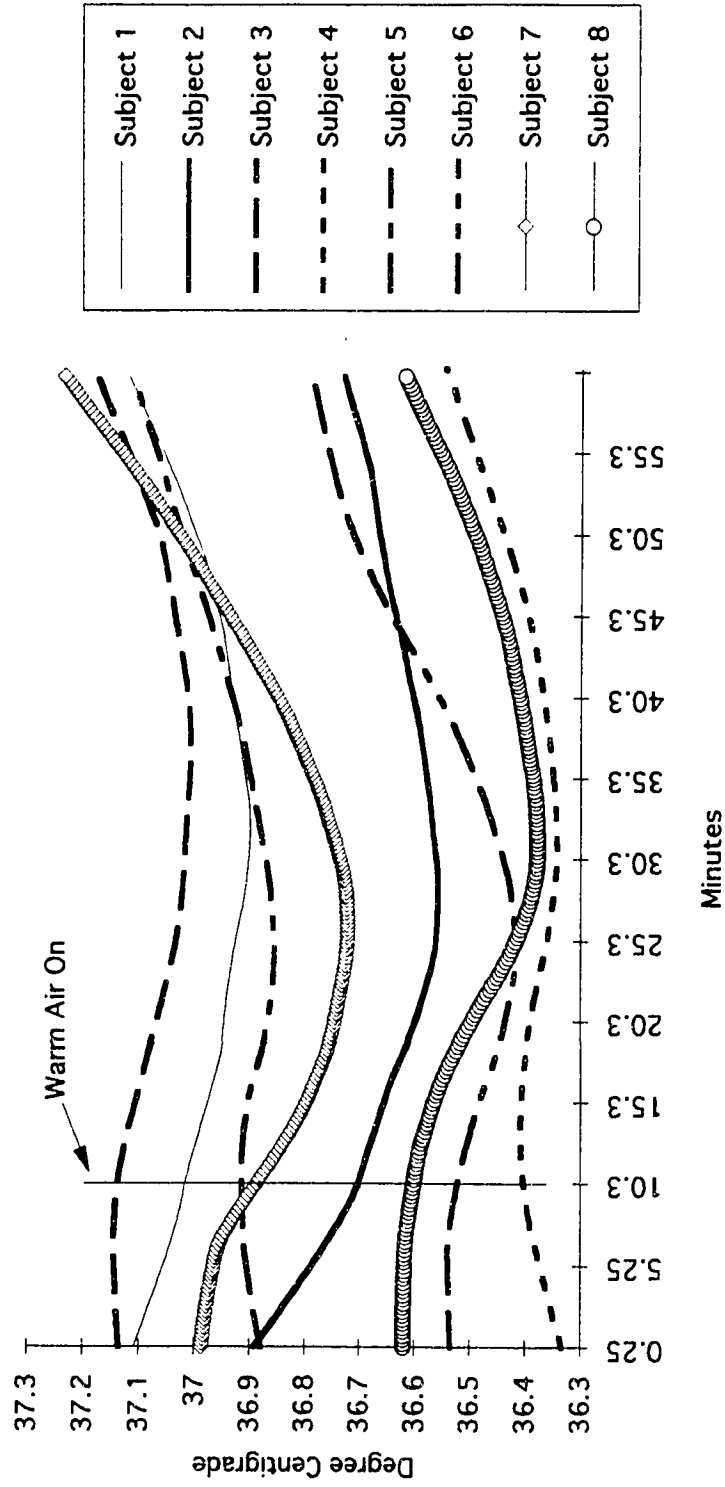


FIGURE 5. Esophageal Temperature for All Subjects During Warming.

Measurements of afterdrop included duration of condition as well as the amplitude of change in temperature (Figure 6). Onset of afterdrop was defined as the time from the beginning of the warming phase to the point where  $T_{es}$  began decreasing. In this study, onset of afterdrop varied from 0 to 15 minutes (Table 21). The duration of afterdrop, measured in minutes, was the time that included the decreasing phase and the portion of increasing phase until  $T_{es}$  value became equal to the  $T_{es}$  value at the beginning of the decreasing phase (Figure 6 and Table 21). The amplitude of afterdrop was the degree C difference between the  $T_{es}$  value at the beginning of afterdrop and the  $T_{es}$  value at the lowest measured  $T_{es}$ . The duration of afterdrop ranged from 28 minutes to 60 minutes. Subjects 3, 4, 5 and 6 had the shortest duration of afterdrop as well as the smallest amplitude of afterdrop (Table 21). However, Subjects 3, 4, 5 and 6 displayed from a slight to a moderate amount of drop in  $T_{es}$  value during the cooling phase (Table 19). Of note, subjects 3, 4, 5 and 6 were the individuals who described outdoor activities of biking, kayaking, etc. in their routine activities. The time to maximal afterdrop in  $T_{es}$  ranged from 24 to 38 minutes from the beginning of the warming period (14 and 28 minutes, respectively, from the beginning of the active warming interval). When reviewing all the subjects for time to maximal afterdrop and their BMIs, no consistent order of afterdrop and BMI were found (Tables 16 and 21). Furthermore, the ordering of subjects based on the smallest to largest drop in  $T_{es}$  during cooling was not the same ordering of subjects based on amplitude of afterdrop.

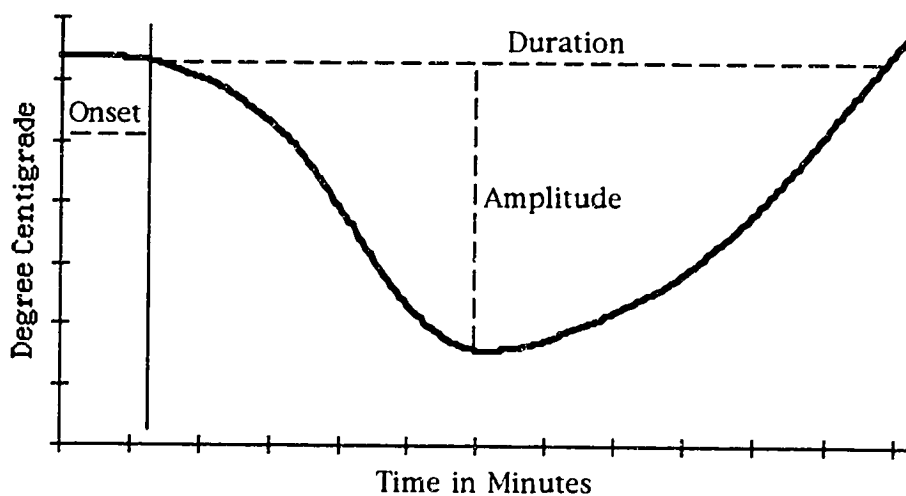


FIGURE 6. Esophageal Temperature Afterdrop During Warming Period.

TABLE 21. Afterdrop During Warming Period.

Subject	Onset <sup>a</sup> (minute)	Time to Maximal Drop <sup>b</sup> (minute)	Amplitude (°C)	Duration <sup>b</sup> (minutes)
1	0(-10) <sup>c</sup>	32(22) <sup>c</sup>	0.21	59
2	0(-10)	28(18)	0.33	60
3	10(0)	38(28)	0.13	47
4	15(5)	31(21)	0.06	37
5	15(5)	24(14)	0.06	28
6	5(-5)	25(15)	0.12	33
7	0(-10)	26(16)	0.27	49
8	5(-5)	31(21)	0.24	60

<sup>a</sup>Based on esophageal temperature change over 5 minute periods. <sup>b</sup>Based on esophageal temperature change over 1 minute period. <sup>c</sup>x(x) study time(time from warming device applied).

#### Mean Skin Temperature

Mean skin temperature was operationalized as the Quartermaster Corps 10-point weighted mean skin temperature ( $T_{qrec}$ ).  $T_{qrec}$  did not follow the same pattern as  $T_{es}$  during the warming period (Table 22 and Figure 7). All subjects experienced an increase in  $T_{qrec}$  during the first 10 minute (baseline interval)

of the warming period prior to the active warming interval. When the warmed, convective air machine was connected to the deflated blanket,  $T_{qrec}$  displayed a drop of up to  $0.5^{\circ}$  C. The trend in  $T_{qrec}$  returned to an increasing temperature trend within 2 minutes (Figure 7). Presumably, the drop in  $T_{qrec}$  was from evaporative heat loss from the skin surface which had remained slightly damp.

Utilizing the baseline interval and the  $T_{es}$  phases within each subject (e.g. decreasing, plateau and increasing), the  $^{\circ}$  C change per minute in  $T_{qrec}$  was calculated (Table 22). During the baseline interval (0 to 10 minute period prior to blanket inflation), all subjects experienced a rising  $T_{qrec}$  which was smaller than the rate of rise in  $T_{qrec}$  during the decreasing- $T_{es}$  phase.

TABLE 22. Quartermaster Corps Skin Temperature Pattern During Warming.

	$T_{qrec}$ in 0-10 min ( $^{\circ}$ C/min)	$T_{qrec}$ $T_{es}$ - Decrease ( $^{\circ}$ C/min)	$T_{qrec}$ $T_{es}$ - Plateau ( $^{\circ}$ C/min)	$T_{qrec}$ $T_{es}$ - Increase ( $^{\circ}$ C/min)	Time $T_{es}$ Began Rising (min)	$T_{qrec}$ @ Time $T_{es}$ Rising ( $^{\circ}$ C)	$T_{qrec}$ @ $35^{\circ}$ C (min)
1	0.04	0.15	0.06	0.06	33	35.42	28
2	0.13	0.17	0.14	0.04	29	35.32	27
3	0.12	0.16	0.10	0.08	38	34.87	40
4	0.13	0.21	0.09	0.05	32	35.11	31
5	0.11	0.29	0.17	0.08	25	34.05	33
6	0.08	0.19	None	0.06	26	35.66	23
7	0.09	0.18	0.12	0.04	27	36.24	21
8	0.15	0.22	0.09	0.04	32	35.83	26
Mean	0.11	0.20	0.11	0.06	30.25	35.31	28.6
SD	0.04	0.04	0.04	0.02	4.33	0.66	6.02

Note.  $T_{qrec}$  = Quartermaster weighted mean skin temperature;  $T_{es}$  = esophageal temperature; min = minute.

The rate of rise in  $T_{qrec}$  was greatest during the  $T_{es}$ -decreasing phase which includes the first 10 to 20 minutes of inflated blanket depending upon the

subject (Table 20).  $T_{qrec}$  continued to rise during the  $T_{es}$ -plateau and  $T_{es}$ -increasing phases, but the rate of rise decreased during each of those phases. Across all subjects, the mean time at which  $T_{es}$  began rising was at minute 30 ( $SD_{\pm}4.33$ ) when  $T_{qrec}$  averaged 35.31 ( $SD_{\pm}0.66$ ) (Table 22).

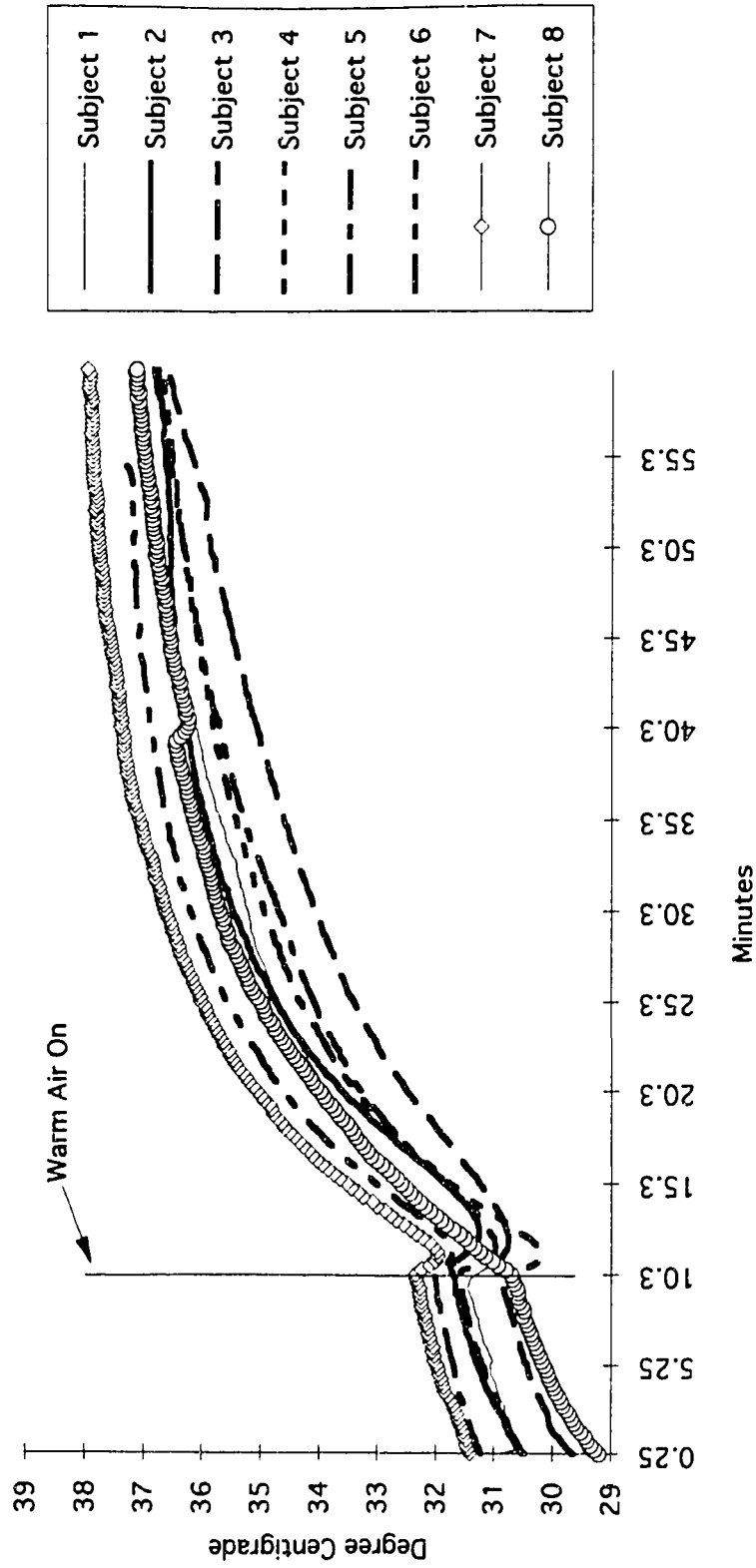


FIGURE 7. Quartermaster Corps Temperature for All Subjects During Warming.

### Forearm Blood Flow

Forearm blood flow (FBF) did not follow the same pattern as  $T_{es}$  (decreasing, plateau and increasing) during warming. Utilizing the same time intervals within the subjects as their  $T_{es}$  phases, the average FBF was calculated (Table 23). The average FBF increased in each phase across all subjects except in Subject 5. In Subject 5, a decrease in the mean FBF was seen during the plateau phase which may be an erroneous finding since the plateau phase was only 5 minutes long in this subject.

TABLE 23. Forearm Blood Flow During Warming.

Subject	Mean FBF $T_{es}$ -Decrease (ml/100ml/min)	Mean FBF $T_{es}$ -Plateau (ml/100ml/min)	Mean FBF $T_{es}$ -Increase (ml/100ml/min)
1	2.00	3.38	6.09
2	1.95	2.23	3.43
3	2.77	3.37	4.16
4	1.87	1.92	3.01
5	2.05	1.77	2.94
6	2.36	None <sup>a</sup>	5.95
7	2.32	3.12	4.12
8	2.01	3.06	4.12
Mean	2.17	2.69 <sup>b</sup>	4.23
SD	0.30	0.70 <sup>b</sup>	1.21

Note. FBF = forearm blood flow;  $T_{es}$  -Decrease = esophageal temperature decreasing phase;  $T_{es}$  -Plateau = esophageal temperature plateau phase;  $T_{es}$  -Increase = esophageal temperature increasing phase.

<sup>a</sup>No plateau phase in Subject 6's data. <sup>b</sup>Subject 6 omitted for calculation.

Subjects 1 and 6 experienced a large increase in FBF during the esophageal temperature increasing phase. Subjects 1 and 6 had the smallest body mass index (BMI) and skinfold thickness of all subjects (Table 16). During the last 20 minutes of the warming period, all subjects experienced an increase in the

rate of rise of FBF, but Subjects 1 and 6 demonstrated the greatest rate of rise (Figure 8).

The relationships of FBF with  $T_{es}$  and with  $T_{qrec}$  were analyzed during the esophageal temperature phases of the warming period (Table 24). During the esophageal temperature increasing phase all correlations of FBF to either esophageal or mean skin temperature were significant. However, during the decreasing and plateau esophageal phases the relationships were not always significant and both negative and positive  $r$  values were found.

TABLE 24. Correlation of Forearm Blood Flow to Esophageal Temperature and to Mean Skin Temperature During Warming.

Subject	FBF & $T_{es}$ Correlation			FBF & $T_{qrec}$ Correlation		
	$T_{es}$ - Decrease (r)	$T_{es}$ - Plateau (r)	$T_{es}$ - Increase (r)	$T_{es}$ - Decrease (r)	$T_{es}$ - Plateau (r)	$T_{es}$ - Increase (r)
1	-0.10	0.39**	0.91**	0.11	0.27**	0.89**
2	-0.10	0.28**	0.86**	0.17*	-0.17*	0.84**
3	-0.37**	0.38**	0.46**	0.37**	-0.13	0.42**
4	-0.56**	0.09	0.87**	0.46**	-0.05	0.82**
5	-0.20*	-0.14	0.79**	0.22**	0.16	0.73**
6	-0.84**	None <sup>a</sup>	0.97**	0.83**	None <sup>a</sup>	0.84**
7	-0.62**	-0.65**	0.90**	0.54**	-0.38**	0.84**
8	-0.65**	-0.44**	0.80**	0.56**	-0.42**	0.79**

Note. FBF = forearm blood flow;  $T_{es}$  = esophageal temperature;  $T_{qrec}$  = Quartermaster weighted mean skin temperature;  $T_{es}$  -Decrease = esophageal temperature decreasing phase;  $T_{es}$  -Plateau = esophageal temperature plateau phase;  $T_{es}$  -Increase = esophageal temperature increasing phase.

<sup>a</sup>No plateau phase in Subject 6's data.

\* $p = 0.05$ , two tailed. \*\* $p = 0.01$ , two tailed.

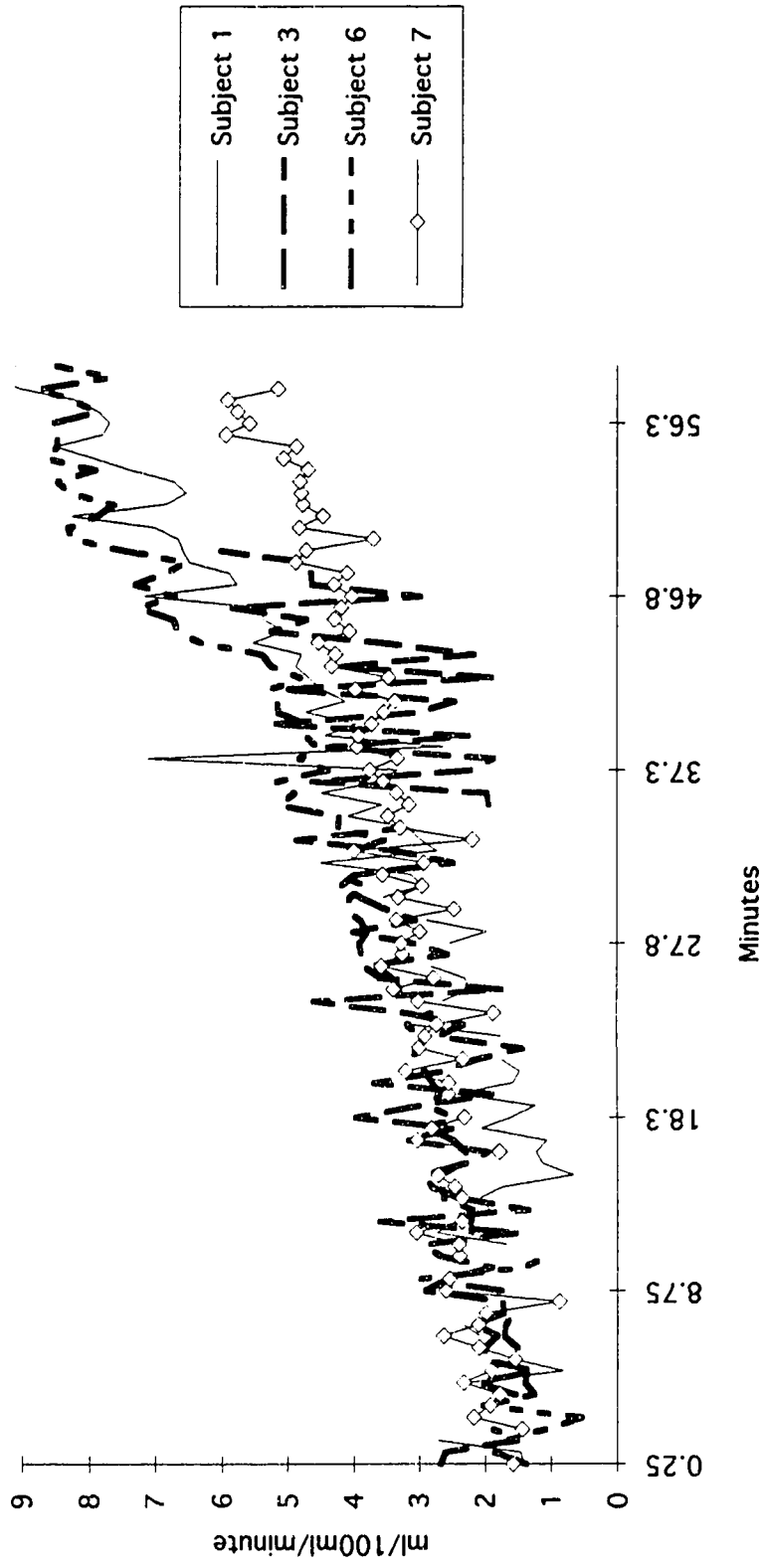


FIGURE 8. Forearm Blood Flow for All Subjects During Warming.

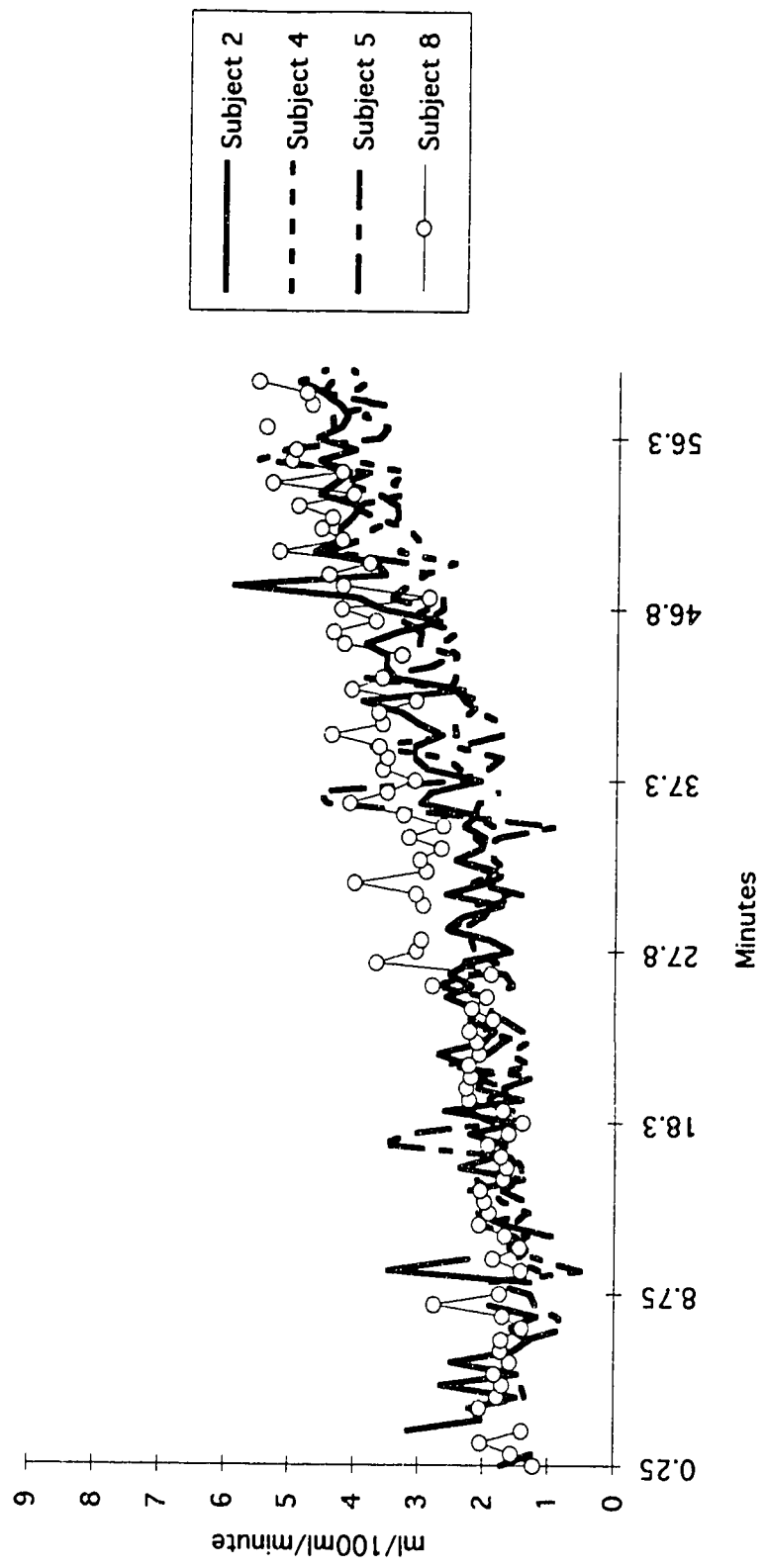


FIGURE 8. Forearm Blood Flow for All Subjects During Warming (Continued).

### Metabolic Rate

The metabolic rate data were measured at 20 second intervals. The oxygen consumption data were smoothed using SPlus (Seattle, Washington). Normally, the volume of expired air varies from minute to minute depending upon the individual's breathing pattern, e.g. sighing or being startled. Measuring the exhaled gas for metabolic rate measurement at 20 second intervals captured the normal variation seen from minute to minute (Figure 9). Because of the normal variation in metabolic rate which was still evident in the smoothed data, the analysis of the oxygen consumption data was conducted by calculating a mean oxygen consumption rate during each of the esophageal temperature phases.

Oxygen consumption had a small decrease across the esophageal temperature phases during the warming period in Subjects 4, 5, 6, and 7 (Table 25). Subject 3's oxygen consumption rate had a slight rise across the warming phases. Ten minutes prior to the end of the esophageal increasing phase, Subject 3 began coughing and the face mask was removed for a 5 minute period. Hence, the oxygen consumption data during Subject 3's final 10 minutes were discarded. However, it was unknown how long prior to the final 10 minutes the subject was suppressing the coughing which may have affected the data. Metabolic data were not obtained on Subject 1 because of equipment failure. Metabolic data were discarded on Subjects 2 and 8 because of erroneously low values when based on body size which potentially indicated a problem with obtaining a tight seal of the mask.

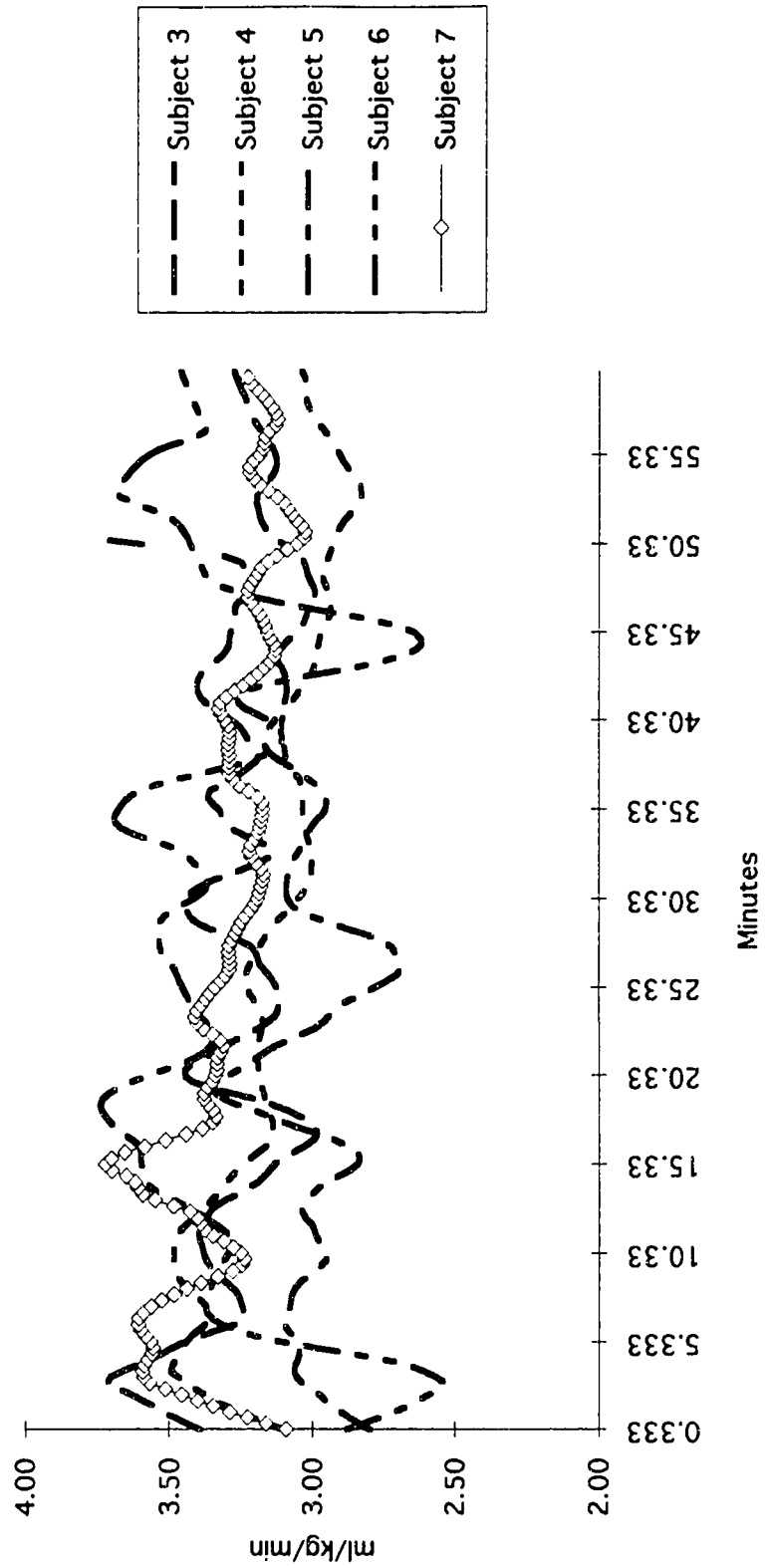


FIGURE 9. Oxygen Consumption for All Subjects During Warming.

TABLE 25. Metabolic Rate During Warming.

	Mean VO <sub>2</sub> Tes-Decrease (ml/kg/min)	Mean VO <sub>2</sub> Tes-Plateau (ml/kg/min)	Mean VO <sub>2</sub> Tes-Increase (ml/kg/min)
3	3.24	3.27	3.30
4	3.17	3.02	2.99
5	3.09	3.05	3.06
6	3.45	None	3.35
7	3.44	3.28	3.19

Note. VO<sub>2</sub> = oxygen consumption.

### Relationships During Warming

The following sections will address the second study question which was to describe the relationships of conductance, vasoactivity, body fat or body mass-to-surface area with esophageal warming.

#### Conductance, Body Size and Vasoactivity with the Rate of Rewarming

Conductance was operationalized as the thermal circulation index which is a ratio of external surface gradient to internal surface gradient ( $T_{qrec} - T_{blanket}/T_{es} - T_{qrec}$ ). Since TCI gives an indication of both the state of convection (vasoactivity) and of conduction (adipose tissue), the relationship of TCI prior to the active warming interval with the rate of rewarming, defined as the increase in  $T_{es}$ /minute, was analyzed. The relationship of TCI with the rate of rewarming was of interest to determine whether TCI may serve as a predictor of the ability to rewarm an individual with a convective-air warming therapy.

The calculation of the rate of rewarming based on esophageal temperature became clouded with the occurrence of afterdrop. The question of whether to exclude the afterdrop change in esophageal temperature or to calculate change in temperature from the lowest point of afterdrop to the end of the

warming period was addressed. In this study, the decision was made to address rewarming in relation to the intervals of warming (baseline interval which was 0 to 10 minutes and active warming interval which was 10 to 60 minutes) (Table 26). Since the time to onset of afterdrop among the subjects varied from the beginning of the baseline interval to 5 minutes into the active warming interval (Table 21), the rate of rewarming for the entire warming period was also calculated. The rate of rewarming was calculated by subtracting the beginning temperature from the ending temperature and dividing by the number of minutes during the rewarming interval being calculated.

The thermal circulation index value during the baseline interval of the warming period ranged between -0.01 and 0.77 (Table 26). As soon as the warmed, convective-air machine was connected to the warming blanket, the TCI values changed dramatically to values in the hundreds.

TCI was not calculated for Subject 1 because the blanket thermocouple failed during the first 30 minutes of warming. TCI increased in all subjects during the 10 minute baseline period, except Subject 7. Subject 7 was the only smoker in the group and was the person with the suspected undiagnosed pulmonary changes.

The correlation coefficient of the initial TCI, calculated at 0.25 minute of the warming period, with the rewarming rate during the active warming interval for the 7 subjects was moderately high ( $r=0.85$ ; Table 27). The correlation of the initial TCI measurement with the rewarming rate during active heating accounted for 72% of the variance when describing the rewarming rate. However, the TCI value calculated just prior to active warming interval accounts for only 46% of the variance of the rate of rewarming during the active warming interval.

TABLE 26. Thermal Circulation Index (TCI) and Esophageal Temperature Change ( $T_{es} \Delta$ ) During Warming.

Subject	TCI @ minute 0.25	TCI @ minute 10	$T_{es} \Delta$ 0 to 10 min ( $^{\circ}\text{C}/\text{min}$ )	$T_{es} \Delta$ 0 to 60 min ( $^{\circ}\text{C}/\text{min}$ )	$T_{es} \Delta$ 10 to 60 min ( $^{\circ}\text{C}/\text{min}$ )
1	No data <sup>a</sup>	No data <sup>a</sup>	- 0.0092	0.0002	0.0020
2	0.25	0.41	- 0.0190	-0.0027	0.0005
3	0.29	0.41	0.0001	0.0005	0.0007
4	0.53	0.77	0.0069	0.0035	0.0057
5	0.37	0.54	0.0034	0.0038	0.0040
6	0.38	0.45	-0.0013	0.0043	0.0053
7	0.52	0.50	-0.0100	0.0040	0.0069
8	-0.01	0.24	-0.0018	0.0000	0.0004

Note. min = minute.

<sup>a</sup>Blanket thermocouple broke

The decrease in  $r$  value from .85 to .68 (at minutes 0.25 and 10, respectively) may be related to 6 of the 7 subjects were experiencing a decreasing  $T_{es}$  by the measurement of TCI at 10 minutes compared with only 2 of 7 subjects experiencing a decreasing  $T_{es}$  at 0.25 minute.

TABLE 27. Correlation of Thermal Circulation Index (TCI) with Change in Esophageal Temperature ( $T_{es} \Delta$ ).

	$T_{es} \Delta$ 0 to 10 min ( $r$ )	$T_{es} \Delta$ 0 to 60 min ( $r$ )	$T_{es} \Delta$ 10 to 60 min ( $r$ )
TCI @ minute 0.25	-0.18	0.67	0.85
TCI @ minute 10			0.68

Autocorrelation of esophageal temperature measurements within subject remained significant at a lag of 40. The  $r$  value at lag 40 ranged between 0.23 to 0.35 across the subjects. An  $r$  value greater than 0.12 displayed significant autocorrelation with 240 repeated measures. Hence, correlating TCI and the

change in esophageal temperature violated the rule of independence of variables.

Duration of afterdrop was an alternate measure of rate of rewarming which met the requirement of independence of variables when correlated with TCI. The  $r$  values for the correlation of TCI with afterdrop duration was -0.61 and -0.63 (TCI measurement at time 0.25 and 10 minute, respectively). The relationship was not significant at  $p=0.05$ . The power of this test of significance was 0.37 (Cohen, 1969). Hence, Type II error may have been present.

Both body size and vasoactivity affect TCI. For this study, body size was operationalized as body mass index (BMI) and skinfold thickness. Vasoactivity was operationalized as forearm blood flow (FBF). During this study, body size remained constant, while vasoactivity (FBF) was dynamic (Figure 8). The desire was to analyze the FBF measurement made at the same time as the TCI measurement. However, during the first 5 minutes of the warming period, the subjects' right arms were repositioned to obtain valid FBF measurements, so the data during that time were not utilized. Furthermore, FBF displayed variation between measurements (Figure 8). Hence, a mean FBF over the last 5 minutes of baseline interval (minutes 5 to 10 of the warming period) was calculated for analysis along with the FBF measurement captured at minute 10.25 of the warming period (last measurement during baseline interval).

Across all subjects, the rate of rewarming was correlated with BMI, skinfold thickness and FBF (Table 28) for the warming period times of 0 to 10 minute, 0 to 60 minute and 10 to 60 minute (Table 29). There were no significant relationships between the rate of rewarming and FBF or body size (Table 29).

TABLE 28. Body Mass Index (BMI), Skinfold Thickness and Forearm Blood Flow (FBF).

Subject	BMI (kg/m <sup>2</sup> )	Skinfold (mm)	FBF Mean 5-10 min (ml/100ml/ min)	FBF @ 10.25 min (ml/100ml/ min)
1	33.3	40	2.158	3.630
2	42	87	1.592	1.287
3	36.3	65	2.012	2.540
4	36.5	69.5	1.614	1.145
5	37.7	56	1.227	1.876
6	33	47.5	1.794	2.969
7	36.7	50	2.029	2.543
8	39.5	74	1.812	1.425

Note. min = minute

TCI is a measurement which reflects the state of body size (skinfold thickness or BMI) and of vasoactivity (FBF). No significant correlations with the rate of rewarming (change in  $T_{es}$ /minute) were identified. However, duration of afterdrop, skinfold thickness, and body mass index displayed moderate correlations which require further study.

TABLE 29. Correlation of Body Mass Index (BMI), Skinfold Thickness and Forearm Blood Flow (FBF) with Change in Esophageal Temperature ( $T_{es}$   $\Delta$ ).

	$T_{es}$ $\Delta$ 0 to 10 min (r)	$T_{es}$ $\Delta$ 0 to 60 min (r)	$T_{es}$ $\Delta$ 10 to 60 min (r)
BMI	-0.36	-0.57	-0.45
Skinfold Thickness	-0.16	-0.59	-0.50
FBF Mean 5-10 minute		-0.18	-0.10
FBF at minute 10.25		0.18	0.13

Note. No correlations significant at 0.05. min = minute.

### Clinical Measurement of Criterion

The following sections will address the third study question which was to describe the precision with which vasoactivity can be estimated with forearm - index finger temperature and forearm temperature during warming.

#### Relationship of Forearm Blood Flow with Forearm-Index Finger Skin Temperature Gradient or Forearm Skin Temperature

The technology and expertise for measurement of forearm blood flow (FBF) is not available in the clinical setting. Hence, FBF was correlated with forearm-index skin temperature gradient ( $T_{fa-if}$ ) and forearm skin temperature ( $T_{fa}$ ). Across all subjects, the  $r$  value for the correlation of FBF with  $T_{fa-if}$  ranged from 0.70 to -0.90 when evaluated during the entire warming period which included the baseline interval and the active warming interval (Table 30).

Within subject correlation of FBF with  $T_{fa-if}$ , during the phases of decreasing-, plateau- and increasing- $T_{es}$ , resulted in variable  $r$  values. During the decreasing- $T_{es}$  phase, the  $r$  value was generally lower than the  $r$  value during the increasing- $T_{es}$  phase and was either a negative or a positive relationship. Hence,  $T_{fa-if}$  did not have a consistent relationship with FBF and would not be a good measurement of FBF in the clinical setting.

The relationship of FBF and  $T_{fa}$  during the entire warming period for all subjects was a positive value, significantly correlated and ranged between 0.60 and 0.90 (Table 30). When evaluating the relationship of FBF with  $T_{fa}$  during phases of decreasing-, plateau- and increasing- $T_{es}$ , the  $r$  value is greatest during the increasing- $T_{es}$  phase. During the decreasing- and plateau- $T_{es}$  phases, the  $r$  values were smaller than during increasing- $T_{es}$ . Hence,  $T_{fa}$  was a

better predictor of FBF than  $T_{fa-if}$ , but would require further evaluation in a clinical setting.

TABLE 30. Correlation of Forearm-Index Finger Temperature Gradient ( $T_{fa-if}$ ) and Forearm Temperature ( $T_{fa}$ ) with Forearm Blood Flow (FBF) During Warming.

	FBF & $T_{fa-if}$ Entire (r)	FBF & $T_{fa-if}$ $T_{es}$ -Decrease (r)	FBF & $T_{fa-if}$ $T_{es}$ -Plateau (r)	FBF & $T_{fa-if}$ $T_{es}$ -Increase (r)	FBF & $T_{fa}$ Entire (r)	FBF & $T_{fa}$ $T_{es}$ -Decrease (r)	FBF & $T_{fa}$ $T_{es}$ -Plateau (r)	FBF & $T_{fa}$ $T_{es}$ -Increase (r)
1	-0.86*	-0.11	-0.06	-0.89*	0.80*	0.06	0.08	0.88*
2	0.70*	0.04	-0.09	0.72*	0.75*	0.17	-0.04	0.87*
3	-0.45*	-0.25*	0.10	-0.48*	0.60*	0.36*	-0.15	0.43*
4	-0.31*	0.35*	-0.06	-0.85*	0.67*	0.46*	-0.07	0.84*
5	-0.75*	0.39*	-0.03	-0.79*	0.68*	0.25*	0.21	0.73*
6	-0.90*	-0.89*	None	-0.80*	0.90*	0.84*	None	0.93*
7	-0.31*	-0.61*	-0.39*	0.81*	0.84*	0.51*	-0.35*	0.85*
8	-0.80*	-0.65*	0.49*	-0.10	0.88*	0.61*	-0.34*	0.80*

Note.  $T_{es}$ -Decrease = decreasing esophageal temperature phase;  $T_{es}$ -Plateau = plateau esophageal temperature phase;  $T_{es}$ -Increase = increasing esophageal temperature phase.

\* $p = 0.01$ .

The following section will address the fourth study question which was to describe the precision with which Quartermaster Corps 10-point weighted mean skin temperature could be estimated by a mean skin temperature measured with fewer than 10 sites.

#### Quartermaster Corps 10-point Weighted Mean Skin Temperature and Other Measurements of Mean Skin Temperature

Weighted mean skin temperature was calculated with Quartermaster Corps 10-site, Hardy and Dubois 7-site, Teichner's 6-site, Palmes and Parker's 6-site, Ramanathan's 4-site and Burton's 3-site formulas.  $T_{qrec}$  was the criterion against which the other weighted mean skin temperatures were compared. In

four of the subjects,  $T_{qrec}$  value was less than the mean weighted skin temperature calculated by the other formulas across the warming period (Figure 10 and Appendices D through K). In the remaining four subjects,  $T_{qrec}$  value was greater than the  $T_{teichner}$  and  $T_{p\&p}$  values, but smaller than the other calculated weighted mean skin temperatures.

Teichner's weighted mean skin temperature correlated with  $T_{qrec}$  most consistently across the different phases of warming and with the highest  $r$  value in all subjects (Table 31). The time frames were held constant between subjects, e.g. temperature values for all subjects were used during the 15 to 30 minute time frame, to avoid a relationship difference which may have been related to time. Time frames for all subjects were selected which represented the decreasing and the increasing esophageal temperature phases (15 to 30 minutes and 30 to 60 minutes, respectively) as well as the baseline interval.

When analyzing the same relationships as described above, but within the individual subject, the correlation of  $T_{qrec}$  with Hardy and Dubois, with Teichner, with Palmes and Parker, and with Ramanathan resulted in an  $r$  value of 0.99 with only three exceptions and then the  $r$  value was 0.98 (Table 32). Burton's formula correlated with  $T_{qrec}$  with an  $r$  value of 0.98 or greater in all but three calculations. In subject 8, time period of 30 to 60 minutes, the  $r$  value was 0.80. Subject 8's raw data for skin temperature displayed a decrease in calf skin temperature at approximately 45 minutes of warming period. The time of drop in calf temperature was associated with the subject changing her leg position. The weighting value for calf temperature in  $T_{qrec}$  was 0.15 while the weighting for calf temperature in  $T_{burton}$  was 0.36. Hence, in quietly resting individuals  $T_{burton}$  serves as the formula with the least number of temperature sites to reliably represent  $T_{qrec}$ .

TABLE 31. Correlation of Quartermaster Corps Weighted Mean Skin Temperature ( $T_{qrec}$ ) with Other Weighted Mean Skin Temperature Calculations for All Subjects.

Weighted Mean	All Subjects 0 to 10 min (r)	All Subjects 15 to 30 min (r)	All Subjects 30 to 60 min (r)
$T_{h\&d7}$	0.9632	0.9456	0.8948
$T_{teichner}$	0.9782	0.9250	0.9407
$T_{p\&p}$	0.9777	0.8963	0.8389
$T_{ramanathan}$	0.9515	0.9569	0.8088
$T_{burton}$	0.9387	0.9505	0.8811

Note. min = minutes;  $T_{h\&d7}$  = Hardy and Dubois weighted mean skin temperature;  $T_{teichner}$  = Teichner weighted mean skin temperature;  $T_{p\&p}$  = Palmes and Parker weighted mean skin temperature;  $T_{ramanathan}$  = Ramanathan weighted mean skin temperature;  $T_{burton}$  = Burton weighted mean skin temperature.

In the clinical area, the fewer the number of sites used to calculate a mean skin temperature the more desirable the measurement. Hence, a forward stepwise regression, utilizing all subjects' data, was performed to identify the least number of sites required to reliably represent  $T_{qrec}$ . When all skin temperatures, from all subjects, across the entire warming period were entered into the forward stepwise regression, forearm skin temperature ( $T_{fa}$ ) accounted for 92.95% of the variance of  $T_{qrec}$  and  $T_{fa}$ . Five skin sites were loaded into the regression equation to account for 99% of the variance (Table 33). When the data were analyzed within the phases of  $T_{es}$  change, 6 skin sites were loaded in the forward stepwise regression to account for 99% of the variance (Tables 34, 35 and 36).

The skin sites identified as the best fit with  $T_{qrec}$  did not remain constant when the time frame of 0 to 60 minutes, 0 to 10 minutes, 15 to 30 minutes and 30 to 60 minutes (Tables 33, 34, 35 and 36) were analyzed. For example, the best fitting sites during the 0 to 60 minute time frame were forearm, calf, front

thigh, index finger and foot. However, the sites with the best fit during the 0 to 10 minute time frame were front thigh, index finger, medial thigh, upper arm, cheek and abdomen. Hence, a weighted mean skin temperature calculated with Burton's formula is preferable to 5 or 6 skin sites averaged to determine a mean skin temperature.

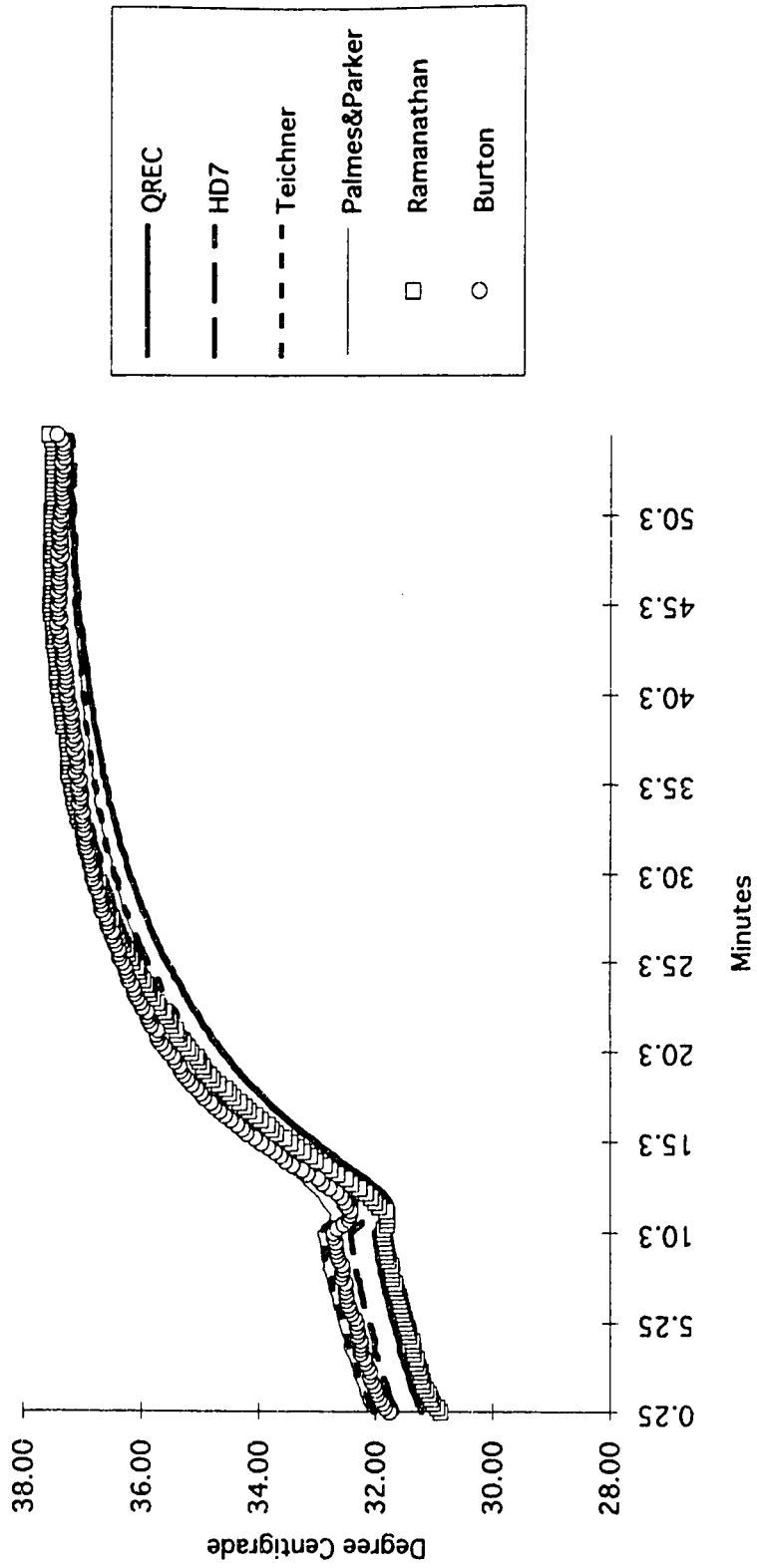


FIGURE 10. Weighted Mean Skin Temperature for a Subject

TABLE 32. Correlation of Quartermaster Corps Weighted Mean Skin Temperature ( $T_{qrec}$ ) with Other Weighted Mean Skin Temperature Calculations within Each Subject.

Weighted Mean	Subject 1	Subject 2	Subject 3	Subject 4	Subject 5	Subject 6	Subject 7	Subject 8
	0 to 60 min (r)	0 to 60 min (r)	0 to 60 min (r)	0 to 60 min (r)	0 to 60 min (r)	0 to 60 min (r)	0 to 60 min (r)	0 to 60 min (r)
$T_{h\&d7}$	0.9982	0.9998	0.9967	0.9997	0.9985	0.9983	0.9993	0.9975
$T_{teichner}$	0.9995	0.9989	0.9973	0.9973	0.9984	0.9995	0.9986	0.9920
$T_{p\&p}$	0.9978	0.9987	0.9936	0.9955	0.9987	0.9987	0.9991	0.9951
$T_{ramanathan}$	0.9983	0.9986	0.9947	0.9971	0.9944	0.9967	0.9986	0.9968
$T_{burton}$	0.9980	0.9985	0.9982	0.9922	0.9960	0.9954	0.9960	0.9805
	0 to 10 min	0 to 10 min	0 to 10 min	0 to 10 min	0 to 10 min	0 to 10 min	0 to 10 min	0 to 10 min
$T_{h\&d7}$	0.9949	0.9980	0.9973	0.9983	0.9988	0.9987	0.9985	0.9992
$T_{teichner}$	0.9957	0.9996	0.9988	0.9993	0.9990	0.9993	0.9985	0.9993
$T_{p\&p}$	0.9931	0.9995	0.9984	0.9989	0.9978	0.9989	0.9989	0.9998
$T_{ramanathan}$	0.9852	0.9993	0.9988	0.9989	0.9997	0.9979	0.9970	0.9996
$T_{burton}$	0.9535	0.9971	0.9961	0.9994	0.9971	0.9967	0.9977	0.9995
	15 to 30 min	15 to 30 min	15 to 30 min	15 to 30 min	15 to 30 min	15 to 30 min	15 to 30 min	15 to 30 min
$T_{h\&d7}$	0.9990	0.9999	0.9993	0.9996	0.9993	0.9997	0.9988	0.9978
$T_{teichner}$	0.9999	0.9999	0.9999	0.9968	0.9978	0.9999	0.9987	0.9997
$T_{p\&p}$	0.9990	0.9993	0.9992	0.9957	0.9994	0.9999	0.9994	0.9993
$T_{ramanathan}$	0.9997	0.9997	0.9998	0.9999	0.9996	0.9998	0.9988	0.9986
$T_{burton}$	0.9986	0.9998	0.9997	0.9990	0.9961	0.9983	0.9949	0.9955

TABLE 3.2. Correlation of Quartermaster Corps Weighted Mean Skin Temperature ( $T_{qrec}$ ) with Other Weighted Mean Skin Temperature Calculations within Each Subject (Continued).

Weighted Mean	Subject 1	Subject 2	Subject 3	Subject 4	Subject 5	Subject 6	Subject 7	Subject 8
	30 to 60 min (r)	30 to 60 min (r)	30 to 60 min (r)	30 to 60 min (r)	30 to 60 min (r)	30 to 60 min (r)	30 to 60 min (r)	30 to 60 min (r)
$T_{h\&d7}$	0.9986	0.9954	0.9967	0.9990	0.9987	0.9851	0.9958	0.9972
$T_{teichner}$	0.9988	0.9982	0.9970	0.9996	0.9982	0.9932	0.9971	0.9830
$T_{p\&p}$	0.9985	0.9984	0.9975	0.9995	0.9962	0.9939	0.9969	0.9902
$T_{ramanathan}$	0.9985	0.9968	0.9895	0.9866	0.9823	0.9764	0.9986	0.9920
$T_{burton}$	0.9930	0.9846	0.9957	0.9785	0.9913	0.9594	0.9971	0.7991

Note. min = minutes;  $T_{h\&d7}$  = Hardy and Dubois mean weighted skin temperature;  $T_{teichner}$  = Teichner mean weighted skin temperature;  $T_{p\&p}$  = Palms and Parker mean weighted skin temperature;  $T_{ramanathan}$  = Ramanathan mean weighted skin temperature;  $T_{burton}$  = Burton mean weighted skin temperature.

TABLE 33. Forward Stepwise Regression of Quartermaster Corps Weighted Mean Skin Temperature ( $T_{qrec}$ ) and Individual Skin Temperature Sites During Warming Period.

No. of Variables	Adjusted R Square	$T_{fa}$	$T_{calf}$	$T_{front\ thigh}$	$T_{if}$	$T_{foot}$	$T_{medial\ thigh}$	$T_{upper\ arm}$	$T_{back}$	$T_{cheek}$	$T_{abdomen}$	$T_{hand}$	$T_{chest}$	$T_{fore\ head}$
1	0.9295	x												
2	0.9686	x	x											
3	0.9806	x	x	x										
4	0.9877	x	x	x	x									
5	0.9907	x	x	x	x	x								
6	0.9931	x	x	x	x	x	x							
7	0.9973	x	x	x	x	x	x	x						
8	0.9986	x	x	x	x	x	x	x	x					
9	0.9990	x	x	x	x	x	x	x	x	x				
10	0.9993	x	x	x	x	x	x	x	x	x	x			
11	0.9996	x	x	x	x	x	x	x	x	x	x	x		
12	0.9996	x	x	x	x	x	x	x	x	x	x	x	x	
13	0.9997	x	x	x	x	x	x	x	x	x	x	x	x	x

Note. No. = number;  $T_{fa}$  = forearm skin temperature;  $T_{calf}$  = calf skin temperature;  $T_{front\ thigh}$  = front thigh skin temperature;  $T_{if}$  = index finger skin temperature;  $T_{foot}$  = foot skin temperature;  $T_{medial\ thigh}$  = medial thigh skin temperature;  $T_{upper\ arm}$  = upper arm skin temperature;  $T_{back}$  = back skin temperature;  $T_{cheek}$  = cheek skin temperature;  $T_{abdomen}$  = abdomen skin temperature;  $T_{hand}$  = hand skin temperature;  $T_{chest}$  = chest skin temperature;  $T_{forehead}$  = forehead skin temperature.

TABLE 3-4. Forward Stepwise Regression of Quartermaster Corps Weighted Mean Skin Temperature ( $T_{qrec}$ ) and Individual Skin Temperature Sites During 0 to 10 Minute Time Period.

Step Number	Adjusted R Square	$T_{fa}$	$T_{calf}$	$T_{front\ thigh}$	$T_{if}$	$T_{foot}$	$T_{medial\ thigh}$	$T_{upper\ arm}$	$T_{back}$	$T_{cheek}$	$T_{abdomen}$	$T_{hand}$	$T_{chest}$	$T_{fore\ head}$
1	0.7948										X			
2	0.9327	X									X			
3	0.9719	X			X						X			
4	0.9796	X			X	X					X			
5	0.9851	X			X	X	X				X			
6	0.9876	X	X		X	X	X				X			
7	0.9931	X	X	X	X	X	X	X			X			
8	0.9950	X	X	X	X	X	X	X	X		X			
9	0.9970	X	X	X	X	X	X	X	X	X	X			
10	0.9970	X	X	X	X	X	X	X	X	X	X			
11	0.9982	X	X	X	X	X	X	X	X	X	X			X
12	0.9982	X	X	X	-	X	X	X	X	X	X			X
13	0.9983	X	X	X	X	X	X	X	X	X	X			X

Note. No. = number;  $T_{fa}$  = forearm skin temperature;  $T_{calf}$  = calf skin temperature;  $T_{front\ thigh}$  = front thigh skin temperature;  $T_{if}$  = index finger skin temperature;  $T_{foot}$  = foot skin temperature;  $T_{medial\ thigh}$  = medial thigh skin temperature;  $T_{upper\ arm}$  = upper arm skin temperature;  $T_{back}$  = back skin temperature;  $T_{cheek}$  = cheek skin temperature;  $T_{abdomen}$  = abdomen skin temperature;  $T_{hand}$  = hand skin temperature;  $T_{chest}$  = chest skin temperature;  $T_{forehead}$  = forehead skin temperature.

TABLE 35. Forward Stepwise Regression of Quartermaster Corps Weighted Mean Skin Temperature ( $T_{qrec}$ ) and Individual Skin Temperature Sites During 15 to 30 Minute Time Period.

Step Number	Adjusted R Square	$T_{fa}$	$T_{calf}$	$T_{front\ thigh}$	$T_{if}$	$T_{foot}$	$T_{medial\ thigh}$	$T_{upper\ arm}$	$T_{back}$	$T_{cheek}$	$T_{abdomen}$	$T_{hand}$	$T_{chest}$	$T_{fore\ head}$
1	0.6198	x												
2	0.8826	x							x					
3	0.9305	x							x			x		
4	0.9646	x	x						x			x		
5	0.9822	x	x		x				x			x		
6	0.9933	x	x		x	x			x			x		
7	0.9955	x	x	x	x	x			x			x		
8	0.9966	x	x	x	x	x			x			x		x
9	0.9972	x	x	x	x	x		x	x			x		x
10	0.9991	x	x	x	x	x	x	x	x			x		x
11	0.9995	x	x	x	x	x	x	x	x	x		x		x
12	0.9995	x	x	x	-	x	x	x	x	x		x		x
13	0.9998	x	x	x	x	x	x	x	x	x	x	x	x	x

Note. No. = number;  $T_{fa}$  = forearm skin temperature;  $T_{calf}$  = calf skin temperature;  $T_{front\ thigh}$  = front thigh skin temperature;  $T_{if}$  = index finger skin temperature;  $T_{foot}$  = foot skin temperature;  $T_{medial\ thigh}$  = medial thigh skin temperature;  $T_{upper\ arm}$  = upper arm skin temperature;  $T_{back}$  = back skin temperature;  $T_{cheek}$  = cheek skin temperature;  $T_{abdomen}$  = abdomen skin temperature;  $T_{hand}$  = hand skin temperature;  $T_{chest}$  = chest skin temperature;  $T_{forehead}$  = forehead skin temperature.

TABLE 36. Forward Stepwise Regression of Quartermaster Corps Weighted Mean Skin Temperature ( $T_{rec}$ ) and Individual Skin Temperature Sites During 30 to 60 Minute Time Period.

Step Number	Adjusted R Square	T <sub>fa</sub>	T <sub>calf</sub>	T <sub>front thigh</sub>	T <sub>if</sub>	T <sub>foot</sub>	T <sub>medial thigh</sub>	T <sub>upper arm</sub>	T <sub>back</sub>	T <sub>cheek</sub>	T <sub>abdomen</sub>	T <sub>hand</sub>	T <sub>chest</sub>	T <sub>fore head</sub>
1	0.7589						x							
2	0.9197						x				x			
3	0.9541						x			x	x			
4	0.9807						x			x	x			
5	0.9871					x	x			x	x			
6	0.9891					x	x		x	x	x			
7	0.9944		x			x	x		x	x	x			
8	0.9971		x	x		x	x		x	x	x			
9	0.9996		x	x		x	x		x	x	x	x		
10	0.9997		x	x		x	x		x	x	x	x	x	
11	0.9997		x	x	x	x	x		x	x	x	x	x	x
12	0.9997	x	x	x	x	x	x	x	x	x	x	x	x	x

Note. No. = number; T<sub>fa</sub> = forearm skin temperature; T<sub>calf</sub> = calf skin temperature; T<sub>front thigh</sub> = front thigh skin temperature; T<sub>if</sub> = index finger skin temperature; T<sub>foot</sub> = foot skin temperature; T<sub>medial thigh</sub> = medial thigh skin temperature; T<sub>upper arm</sub> = upper arm skin temperature; T<sub>back</sub> = back skin temperature; T<sub>cheek</sub> = cheek skin temperature; T<sub>abdomen</sub> = abdomen skin temperature; T<sub>hand</sub> = hand skin temperature; T<sub>chest</sub> = chest skin temperature; T<sub>forehead</sub> = forehead skin temperature.

## CHAPTER V

### Discussion

### Conclusions

This study offers a comprehensive description of factors, both individual and environmental, which affect the warming process. Measuring the static (body mass) and the dynamic (vasoactivity and metabolic rate) individual factors which affect thermoregulation is unique in the rewarming literature.

In this study, all subjects experienced afterdrop, although the drop in  $T_{es}$  in a couple of subjects was only slight and probably would not have been detected in a clinical setting. Individuals with a larger BMI have a greater amount of afterdrop. The length of time for recovery from afterdrop is up to 60 minutes, which raises the issue of the length of time required to rewarm a hypothermic individual using a non-invasive curative therapy.

$T_{sk}$  and FBF patterns display increasing values across the phases of  $T_{es}$ -decrease, -plateau and -increase. The mean  $T_{sk}$  for all subjects is  $35.31 \pm 0.66^{\circ}C$  at the time  $T_{es}$  began to rise. The effect of a minimum skin temperature on central rewarming when using a non-invasive therapy has not previously been studied.

A moderately high correlation between TCI and the rate of change in  $T_{es}$  across the warming period was found, although the correlation must be viewed with the understanding that  $T_{es}$  did display significant autocorrelation. The components of conductance (TCI), vasoactivity (FBF) and body mass (BMI or skinfold thickness), have poor to moderate correlations, respectively, with the change in  $T_{es}$  across warming. Although, BMI has been reported in the rewarming literature, TCI has not been studied.

Vasoactivity operationalized as FBF can not be measured reliably using forearm - index finger temperature gradient. In the rewarming literature, forearm - index finger temperature gradient is being used as an indicator of forearm blood flow. The original study of forearm - index finger gradient with vasoactivity actually studied finger tip blood flow, not forearm blood flow. From the findings of this study, a more reliable measure of FBF is forearm skin temperature, if FBF measurement is not available.

Mean skin temperature across the warming phase can be reliably measured with fewer than the 10-weighted mean sites used when calculating Quartermaster Corps weighted mean skin temperature. However, 6-weighted mean sites described by Teichner is the least number of sites that consistently measured mean skin temperature with an  $r$  value of 0.99. However, if the criteria of acceptable  $r$  value is lowered to 0.95, Burton's 3-weighted mean sites reliably measured  $T_{qrec}$ .

### Pattern of Rewarming

#### Environmental Temperature

The ambient temperature in this study is consistent with the ambient temperature reported in other laboratory warming studies which utilized the Bair Hugger®, convective air, warming blanket. However, no other studies report the temperature within the environment of the blanket. In this study, the mean blanket temperature, within subjects, varies from a low of 39° C to a high of 47.22° C. Even excluding Subject 1's mean blanket temperature (39° C), the next lowest mean blanket temperature is 43.16° C which is a 4.06° C difference in mean blanket temperatures between the highest and second lowest mean blanket temperatures. The convective air, warming machine, with a setting of high, is to deliver an air temperature of  $43.3 \pm 2.8^{\circ}$  C. The

manufacturer does not describe the reason for the large standard deviation with air temperature, but potentially the effect is from varying ambient temperatures affecting the amount of heat being delivered by the heat source. In order, the heating element is set at a certain level, but the variation in ambient temperature is not controlled, so the warmed air delivered is not the same temperature as the heating element. Hence, in this study, the lowest ( $39^{\circ}$  C) and the highest ( $47.22^{\circ}$  C) blanket temperatures are outside the manufacturers specified ranges. The experiment with the lowest mean temperature (Subject 1) may be from an error in the machine setting of medium instead of high.

The adult, full body blanket (Model 300; Augustine Medical, Eden Prairie, Minnesota) is available as one size with no variation in length or width to accommodate individuals of different body shapes. In this study, all subjects were completely covered from side to side and foot to shoulders. Subject 2, who had the largest BMI, had a mean blanket temperature of  $44.09^{\circ}$  C. Subject 6, who had the smallest girth and body mass index, had the highest mean blanket temperature ( $47.22^{\circ}$  C). The potential of a poor blanket fit based on body size exists and may have had some influence on the mean blanket temperature. However, when the order of mean blanket temperatures and the order of BMIs are compared across subjects no pattern of body size to blanket temperature is noted.

Furthermore, one is concerned with the potential of tissue damage from the heat source. Subject 6 experienced the highest blanket temperature, but her skin temperatures did not rise above  $40^{\circ}$  C, except the index finger temperature which became greater than  $40^{\circ}$  C in the last 10 minutes of the study. In this study, no subjects experienced any tissue damage.

Hence, the warming instrument itself may affect the rate of rewarming between individuals. Potentially, the blanket temperature and the fit of the blanket affect the rate of rewarming. However, in this study, no consistent pattern of order of blanket temperature and order of warming was identified.

#### Esophageal Temperature

Both circadian and menstrual rhythms affect body temperature. In this study, the effect of circadian rhythm is controlled by studying all subjects at the same time of day and excluding potential subjects who work during the night time which will alter their normal circadian rhythm. The effect of menstrual rhythm is not controlled. However, the maximal luteal-follicular phase effect is during early morning hours with the least effect being at 3:30 p.m. (Schmidt, 1972, cited by Hessemer and Brück, 1985). Furthermore 4 of the 8 subjects were studied during the follicular phase, 1 during the luteal phase, and the remaining 3 had partial hysterectomies. The subject studied in luteal phase did have a higher  $T_{es}$  at the beginning of the study than the 4 subjects studied during the follicular phase, but the warming pattern for the subject in luteal phase was similar to the pattern of those individuals studied in the follicular phase.

During the cooling period, esophageal temperature initially rises with exposure to the 30° C water spray. In some subjects, after approximately 40 minutes a gradual decline in  $T_{es}$  was seen. In all subjects, when the water temperature was increased during the 5 minute incremental water temperature increases,  $T_{es}$  declined. The afore-described process has been seen in other studies in the laboratory where this study took place. The current understanding is as the skin is cooled, vasoconstriction increases which shunts the blood centrally. The heat produced within the body organs is

centrally-located and -shunted which results in an increase in central temperature. As the water spray temperature is increased, the temperature change is sensed by the neuroreceptors in the skin and the amount of vasoconstriction is lessened. With a lessening of the vasoconstriction, the blood is exposed to colder tissue and returns to the core cooler which results in a decrease in central temperature. In this study, the attempt was made to measure forearm blood flow (FBF) during cooling, however increased muscle tone and shivering interfere with valid measurement of FBF.

At the beginning of the warming period, three subjects displayed  $T_{es}$  that were greater than their  $T_{es}$  at the beginning of the cooling period. The phenomena described above of vasoconstriction and maintaining the centrally produced heat in the central aspect of the body may also explain the increased  $T_{es}$  at beginning of the warming period. Unfortunately, FBF measurement during the first 5 minutes of the baseline warming phase is unreliable because of the need to readjust arm position to obtain a valid measure. The three subjects were made up of the two individuals with the greatest BMI and the one individual who smoked. These three subjects did display the greatest amount of afterdrop during the warming period and had either a slight or moderate amount of decrease in  $T_{es}$  during cooling.

Afterdrop of esophageal temperature is identified in all subjects in this study. The group mean time to maximal afterdrop in this study is 19.4 minutes from the time of active warming. The time to maximal afterdrop may be related to the type of heat source which is applied to the body surface. Daanen and VanDeLinde (1992) and Savard, Cooper, Veale and Malkinson (1985) exposed laboratory cooled individuals to whole body immersion in a water bath of 42 or 40° C water temperature (respectively) and both reported 5 minutes time to

maximal afterdrop. Daanen and VanDeLinde's subjects had been cooled to 35° C esophageal temperature, while Savard et al. subjects were cooled until the esophageal temperature declined by 0.3 to 0.5° C. Daanen and VanDeLinde's self rewarming subjects displayed maximal afterdrop at 16.7 minutes. In both Daanen and VanDeLinde and Savard, et al. studies the subjects experienced only a 5 minute time period when transferring from the cooling to the warming portion of the study compared to a mean transfer time of 22.5 minutes in this study. The longer transfer time for this study may have affected the timing of the maximal afterdrop.

In this study, the greatest degree temperature of afterdrop was in Subject 2 who had the largest body mass index and skinfold thickness. Daanen and VanDeLinde (1992) also reported that the subject with the largest body fat had the greatest degree temperature afterdrop. Potentially, the greater body mass acts as insulation and protects the central temperature from decline during the cooling process. However, the insulation layer, shell portion of the mass, actually cools during the exposure. When the person with greater mass is exposed to the warming therapy, increasing vasodilatation supplies blood to the greater cold mass. Since there is greater mass, there is a greater amount of tissue with which heat can be exchanged as blood circulates to previously vasoconstricted tissue. Hence, a greater decline in central temperature occurs in the individual with either a greater BMI or a greater amount of body fat.

In this study, the mean duration of afterdrop was 47 minutes with a range of 28 to 60 minutes with a mean afterdrop amplitude of 0.18° C. In light of Slotman et al. findings (1985), of an increased mortality rate in individuals who remained hypothermic (central temperature < 36.1° C) after 2 or more hours postoperatively, the duration of afterdrop is concerning. A 47 minute

afterdrop duration is approximately 40% of the time of the 2 hour window. If the afterdrop duration found in this study is consistent with the afterdrop duration in postoperative patients, a large number of patients are at risk and do require effective rewarming methods.

#### Mean Skin Temperature and Forearm Blood Flow

Heat is transferred by convection between the environment of the warm air blanket and the subject's skin. Theoretically, the greater the temperature gradient is between the environment and the skin, the larger the heat transfer which is supported in this study. In the first minutes of active heating,  $T_{qrec}$  rises an average of  $0.20^{\circ}$  C/minute with a smaller rate of increase in  $T_{qrec}$  the longer the active heating interval progresses. The longer the active heating interval progresses the smaller the temperature gradient became between the environment and the subject's skin.

In the literature, an argument exists over the cause of afterdrop. The argument is whether the central temperature afterdrop is from convective or conductive heat movement from the body shell into the core. In order, is the afterdrop caused by vasodilation and cooling of the increased blood supply, which circulates through the cool shell tissue mass, which then returns to the core with a lower temperature resulting in a central temperature drop or is the core temperature decreased because of a conductive cooling of the mass which then affects the central temperature? Webb (1986) argued that afterdrop was from conduction of cold through the mass. However, in 1992, Webb's data revealed the gradient of tissue temperature at 40 mm within thigh muscle and thigh skin temperature, during 2 hours of hot exposure following cold exposure, reversed in degree of temperature as expected, but the subcutaneous thigh tissue temperature remained higher than either skin or

muscle temperature. For the subcutaneous tissue, which lies between muscle and thigh skin, to remain higher over a 2 hour period would require either a very slow rate of conduction or convective source of heat. Webb (1986) stated that peripheral blood flow remained low until afterdrop was completed.

In this study during warming, skin temperature rose rapidly during the initial 20 minutes with a continuing but lesser rate of rise during the following minutes, which is the same skin temperature pattern displayed by Webb's data (1992). Furthermore, in this study forearm blood flow did increase during the plateau and increasing esophageal temperature phases of afterdrop. Hence, the theory that afterdrop is from conductive heat transfer is not supported by this study.

In this study, all subjects experience afterdrop. The onset and course of afterdrop varies across subjects. The time at which esophageal temperature begins to rise is  $30.25 \pm 4.33$  minutes (20.25 minutes after initiation of active heating). Furthermore, the mean skin temperature, at the time esophageal temperature begins to rise, is  $35.31 \pm 0.66^{\circ}$  C. Other studies have not described the time to the beginning of esophageal temperature rise and the skin temperature at the time of esophageal temperature rise. Furthermore, in this study, the length of the transfer period (transfer from the cooling to the warming bed) is variable among subjects and the amount of time to transfer becomes quite long in some experiments which may effect both the time to rise in esophageal temperature and the mean skin temperature at which esophageal temperature begins to rise. Hence, the use of either time or skin temperature as a criterion in rewarming requires further study.

The argument of whether the skin thermocouples were measuring skin temperature or were overly affected by the environment temperature can be

raised. In this study, the thermocouples were taped to the skin with no further insulation or covering. Prior to beginning the experiments, a pilot study was performed in which thermocouples were attached to the forearm skin with tape over the thermocouple, with a surgical adhesive between the thermocouple and skin, and with tape and a small cup which protected the thermocouple from environmental air movement. Regardless of the method of attachment and insulation of the thermocouple, the resulting skin temperatures were very similar. Furthermore, during the warming period in experiment six, the chest thermocouple became untaped from the skin and was found lying loose between the skin and the cotton gown at the end of the experiment. The chest thermocouple temperature was 1 to 2° C higher than the abdominal skin temperature during active warming. In all other subjects where the thermocouples were firmly attached at the end of the experiment the chest and abdominal skin temperatures closely tracked one another. Hence, taping the thermocouples to the skin resulted in a temperature which was a measurement of skin temperature and not overly affected by the environment temperature.

FBF is affected by both central and local control mechanisms. According to Barcroft and Edholm (1943), the local control mechanism becomes evident after 40 to 60 minutes of warming in a water bath at 38 to 42.5° C. In this study, Subjects 1 and 6 displayed an increase in the trend of FBF after approximately 30 minutes of active warming which is from the local response. The remaining subjects did display an increase in FBF trend, but the amount of change was not as dramatic as Subjects 1 and 6.

Estrogen replacement therapy has been shown to decrease the  $T_{es}$  at which FBF increases in exercising women,  $48 \pm 2$  years of age (Tankersley, Nicholas,

Deaver, Mikita, & Kenney, 1992). Tankersley et al. studied six women pre- and post-estrogen therapy replacement (three who reported erratic intervals between menses and three with partial hysterectomies). In this study, afterdrop makes it difficult to analyze the effect that estrogen replacement may have on the temperature at which FBF begins to rise. However, when FBF is reviewed for each subject at 20.25 minutes, Subjects' 7 and 8, on estrogen replacement therapy, have higher FBF values (2.58 and 2.42 ml/100ml/min, respectively) than the other Subjects (1.42 to 2.42 ml/100ml/min range with all but one subject's FBF being less than 2.00). Furthermore, Subjects' 7 and 8 have a  $T_{qrec}$  of 35° C at minutes 21 and 26, respectively, which is a smaller number of minutes to reach  $T_{qrec}$  of 35° C than all but one of the remaining Subjects. In this study, the stimuli for change in FBF is an increase in skin temperature compared to an increase in central temperature in Tankersley et al. study. Hence, the findings of this study support Tankersley et al. findings of estrogen replacement therapy increasing the reactivity of FBF to either physiological or environmental stimuli.

#### Metabolic Rate

The majority of the rewarming studies do not describe the metabolic rate across the rewarming period. In this study, the data measurement interval for metabolic rate is set at every 20 seconds which results in variability in the data. The measurement interval captures the variation in respiration from a changing minute ventilation which is a normal phenomena. The data are smoothed using Splus, but the variability in the respiratory pattern is still evident. Hence, mean values over phases were used to describe the metabolic data.

To measure metabolic rate, one must capture all expired air. To capture the expired air, the subject must breathe through a mask or use a mouth piece with nasal clips. Because of the length of the study and for the subject's comfort, a mask was selected. Unfortunately, it is difficult to get a tight fit with a mask. For this study, a mask with an inflatable lip and a head strap mechanism which covers the occiput and wraps around the face to attach to the mask was used. A tight seal between the mask and face were visually observed as well as the actual oxygen consumption rate checked. Unfortunately, the metabolic cart displayed oxygen consumption data during measurement in ml/min instead of in ml/kg/min which would assist in alerting one to values which were artificially low for the subject under study. Hence, due to instrument failure, oxygen consumption data for Subjects 1, 2 and 8 are not available for analysis.

The metabolic rates in Subjects 4 through 7 demonstrated a slight decrease from the decreasing esophageal temperature to the increasing esophageal temperature phase (group mean difference of 0.14 ml/kg/min). In a 70 kg person, a 0.14 ml/kg/min difference in metabolic rate is a 40 kcal/kg/hr heat difference which equates with approximately 0.6° C difference in temperature if all else were equal. Subject 3 displays a slight increase in metabolic rate across time (0.06 ml/kg/min difference between decreasing esophageal and increasing esophageal temperature phases). The 0.06 ml/kg/min increase in metabolic rate equates with a 0.25° C increase in temperature in a 70 kg person over an hour, if all other sources of heat loss are eliminated. In the last 10 minutes of study, Subject 3 began to cough from irritation of the clear plastic tubing placed over the esophageal thermistor where the thermistor passes between the teeth. The plastic tubing slipped further into Subject 3's mouth

and was irritating the back of her mouth. Hence, the measurements for the final 10 minutes of the study are deleted. However, it is unknown whether the slight rise seen in oxygen consumption for Subject 3 is related to the plastic tube slipping into the mouth earlier and causing discomfort.

During the warming portion of the study, all subjects' metabolic rates are normal adult resting values. Hence, the rise in esophageal temperature during active warming is not from an increase in metabolic rate, but from heat transfer between the environment and the subject's core.

### Relationships During Warming

#### Rate of Rewarming and Conductance

Conductance was operationalized as thermal circulation index (TCI) in this study. Burton (1934) indicated that the greater the TCI is the greater the heat flux. Since TCI is the ratio of the skin-environment temperature gradient to the internal-skin temperature gradient, both convective and conductive heat transfer are taken into account. Hence, within an individual, in a constant environmental temperature, if the TCI changes, the effect is from a change in vasoactivity, because the body mass is static during a short period, e.g. a few minutes. However, when addressing rewarming across individuals at a moment in time, a measurement which incorporates both the body mass and the vasoactivity is needed. Hence, the relationship of TCI with the rate of rewarming was studied across subjects.

When TCI is correlated with the change in esophageal temperature over the warming period between all subjects, the correlation coefficient is moderate to moderately high ( $r = 0.67$  and  $0.85$ , TCI at minute 0.25 with change in  $T_{es}$  from 0 to 60 minutes and with change in  $T_{es}$  from 10 to 60 minutes,

respectively). Potentially, TCI may serve as a variable to predict warming rates of individuals, but further study is needed.

One of the variables used to calculate TCI is esophageal temperature, which raises the issue of the effect of esophageal temperature on the correlation coefficient value.  $T_{es}$  at a moment in time has a relationship with  $T_{es}$  at a time in the future in a constant environment. The autocorrelation of  $T_{es}$  was found to be significant at a lag of 40. Hence, TCI can not be treated as a variable independent of the rate of rewarming. However, the duration of afterdrop is an independent variable and has a moderate correlation with TCI. In this study the duration of afterdrop was as long as 60 minutes. It is unknown how the duration of afterdrop is affected by the actual degrees of decline in esophageal temperature from the cold exposure. It is also unknown what effect the duration of afterdrop may have on the overall recovery from hypothermia.

#### Relationships of the Rate of Rewarming with Vasoactivity and with Body Mass

TCI is a measurement of both body mass and vasoactivity. The question of the relationships of body mass and vasoactivity (the components of TCI) with the rate of rewarming was studied. In this study, when measurements of body mass (body mass index and skinfold thickness) and vasoactivity (FBF) are correlated with the change in esophageal temperature across the warming period, moderate ( $r = 0.59$ ) to poor ( $r = 0.09$ ) correlation coefficients are found, respectively. The static portion of TCI, body mass, is a better predictor of rewarming rate than the dynamic portion of TCI, FBF, in a female population which is within 20% of ideal body weight in an environment of convective air warming. In individuals, who weigh less than 80% of ideal body weight, the effect of FBF may become more evident since the static insulation layer is less.

In postoperative individuals, FBF is affected by a lower central temperature, by an altered fluid volume, and by medications. Hence, using FBF at the end of an operative procedure to predict the ability to rewarm over the next hour postoperatively will probably be even less reliable than the results of this study. In postoperative patients, Erickson and Yount (1991) reported BMI to be a reliable indicator of postoperative rewarming. Hence, body mass will be a better predictor of the rate of rewarming during the first hour of rewarming than FBF.

The preferred method of measuring body mass is BMI. The potential of measurement error is less when measuring BMI than when measuring skinfold thickness. Also, in the clinical setting, the instrumentation for measurement of BMI is readily available, while the instrumentation for skinfold thickness is not readily available. Hence, the relationship of BMI with the rate of rewarming warrants further study.

#### Relationships of Forearm Blood Flow with Temperature Gradient and with Forearm Skin Temperature

Rubinstein and Sessler (1990) studied the relationship of forearm - fingertip temperature gradient and finger blood flow in steady-state and reported an  $r = 0.98$ . Since that study, the forearm - fingertip temperature gradient has been used as a measurement of thermoregulatory vasoconstriction (Hynson, Sessler, Moayeri and McGuire, 1993). Since the control of acral vasculature (e.g. palmar hand vessels) is different from the control of non-acral vasculature (e.g. forearm), the question must be raised whether the forearm - fingertip temperature gradient correlates with blood flow of non-acral forearm skin. Furthermore, the measurement of FBF with occlusion plethysmography is unavailable in the clinical setting, because of

the lack of instrumentation and the high skill level required. Hence, the relationship of forearm - finger temperature gradient and FBF was studied.

The correlation coefficient for forearm - index finger temperature gradient with FBF across the entire warming period varies from -0.31 to -0.90 in seven of the subjects studied, while one subject's correlation coefficient is 0.70. When the correlation coefficient is calculated within the decreasing, plateau and increasing phases during warming, the  $r$  value is found to vary greatly, e.g. Subject 4  $r$  values are 0.35, -0.06 and -0.85 (decreasing, plateau and increasing phases, respectively). Hence, the temperature gradient of forearm - finger is not a valid measure of FBF during the warming period.

Forearm skin temperature is a more valid measure of FBF than the forearm - finger gradient. The correlation coefficient for  $T_{fa}$  with FBF, across the entire warming period, ranges from 0.60 to 0.90 in all subjects. However, during the decreasing esophageal temperature phase, the correlation coefficient varies from 0.06 in Subject 1 to 0.84 in Subject 6. The strength of the relationship increases during the increasing esophageal temperature phase ( $r = 0.43$  in one subject and from 0.73 to 0.93 in the remaining subjects). Hence, forearm skin temperature is a more valid measure of FBF than forearm - finger skin temperature gradient.

#### Mean Skin Temperature Measurement

Teichner (1958) investigated the reliability of weighted mean skin temperature formulas with Quartermaster Corps 10-point weighted mean skin temperature ( $T_{qrec}$ ) in 323 military male subjects with nude exposure to 15.6° C and 5 mph air movement. Teichner reported a 0.74, 0.71 and 0.60 correlation coefficient of  $T_{qrec}$  with  $T_{h\&d7}$ ,  $T_{p\&p}$  and  $T_{burton}$ , respectively. Utilizing the

data from the study, Teichner then developed a 6-point weighted mean skin temperature.

In this study, the correlation of  $T_{qrec}$  and other formulas for weighted mean skin temperature are also evaluated. In female, warming subjects,  $T_{h\&d7}$  across all subjects has a better correlation with  $T_{qrec}$  ( $r = 0.9632, 0.9456$  and  $0.8948$  during 0 to 10 minutes, 15 to 30 minutes and 30 + minutes of the warming period, respectively) than either  $T_{p\&p}$  and  $T_{burton}$  which agrees with Teichner's findings. Furthermore, when Teichner's 6-point weighted mean skin temperature ( $T_{teichner}$ ) is correlated with  $T_{qrec}$  across all subjects, a higher and more consistent correlation coefficient is found across the phases of the warming period ( $r = 0.9782, 0.9250$  and  $0.9407$  during 0 to 10 minutes, 15 to 30 minutes and 30 + minutes of the warming period, respectively).

When the correlation coefficient is calculated for  $T_{qrec}$  and  $T_{teichner}$  within each subject across different time periods of warming, the  $r$  value is 0.99, except in Subject 8 when the  $r$  value drops to 0.9830 during the 30 + minute period. The correlation of  $T_{qrec}$  with Ramanathan ( $T_{ramanathan}$ ) and with Burton ( $T_{burton}$ ) mean weighted skin temperature have greater variability of  $r$  values across all subjects and within subjects across the time periods than the other weighted mean skin temperature formulas. Hence, 6 or 7 weighted sites had the strongest and most consistent correlation with  $T_{qrec}$  within subjects and between subjects across the warming period.

The question must be raised as to the level of precision that is acceptable. An  $r$  value of 0.99 accounts for 98% of the variance, while an  $r$  value of 0.95 accounts for 90% of the variance. Both 0.99 and 0.95 are considered high correlations in the physiological literature. Hence, accepting a precision level of 0.95 is reasonable. Therefore, utilizing  $T_{burton}$ 's formula which displays an  $r$

value of 0.96 or greater except in Subject 8 is reasonable. Furthermore, when reviewing the raw skin temperature data it is evident that the subject moved resulting in a different orientation of her calf to the heat source. Since the weighting of calf temperature is 0.36 in Burton's formula but 0.15 in Quartermaster Corps' formula, the decrease in the correlation value for Subject 8 is understandable.

In the clinical setting, fewer skin sites for measurement of skin temperature is desirable. Hence, in this study analysis, forward stepwise regression is utilized to identify the least number of sites that can be used as a reliable measure of  $T_{qrec}$ . The stepwise regression for all subject temperature sites across the entire warming period results in an  $r^2$  value of 0.99 with 4 sites in the equation. However, when the esophageal phases of the warming period were analyzed 5 or 6 sites are in the equation to get an  $r^2$  value of 0.99. Unfortunately the sites vary immensely between the formulas. Hence, the use of fewer unweighted sites do not result in a reliable measure of  $T_{qrec}$  across the warming period in this study.

#### Limitations

The results of this study are not generalizable to other female populations, such as females of the same age group and body size who have undergone surgery. Also, since the decrease in central temperature across the subjects from the hour of cold exposure was small (group mean decrease in  $T_{es}$  was  $0.24^{\circ}$  C), the findings of this study can not be generalized to matching groups who have a greater drop in central temperature. However, this study does gain insights which other populations can be compared against.

The statistical analysis of data must be viewed with caution where the correlation or regression is of values which were repeated measures within a

subject, because the need for independent measures is violated. Since this study was descriptive, the correlation or regression values were used to describe the relationships, but the strength of the relationship must be viewed with caution. Furthermore, the small  $n$  increases the risk of creating a Type II error when parametric statistics are used.

Another limitation of this study is the missing metabolic data on three of the eight subjects. Other measurement difficulties included failure of the blanket thermocouple in Subject 1 and disconnection of the chest thermocouple in Subject 5, for which abdomen temperature was substituted in calculations where chest skin temperature was normally used.

The effect of the variable transfer time is unknown. It is unknown whether the duration of afterdrop or the rate of rewarming was affected by the transfer time.

#### Nursing Practice Implications

The findings of this study are preliminary and not generalizable. However, points of interest for nursing practice would include the frequency of afterdrop in individuals exposed to cool or cold environments for 1 or greater hours is probably quite high. The length of time for those individuals, who experienced  $0.2^{\circ}\text{C}$  or more of afterdrop, to return to the pre-afterdrop temperature with the convective air warming blanket on the high setting was 40 to 50 minutes of active warming, which does not include the time needed to recover from the decrease in central temperature from exposure. Hence, to recover from postoperative hypothermia and afterdrop within the 2 hour period described by Slotman et al. (1985) may require aggressive rewarming therapies such as applying a plasticized aluminum head cover to decrease further heat loss from the head and covering the convective-air warming

blanket with both cotton blankets and a plasticized aluminum blanket to optimize the patient's micro-environment. The appropriate therapy may include an invasive therapy along with a non-invasive therapy, but further study is required.

Also, those individuals who experienced the least afterdrop were the subjects who identified out doors activities as a part of their routine. The question of whether an individual's physical fitness level affects the amount of afterdrop must be raised.

A question must be raised whether there is a minimal  $T_{sk}$  before  $T_{es}$  will begin to rise. If a  $T_{sk}$  of  $35.3^{\circ}$  C is necessary before  $T_{es}$  will begin to rise, then application of the therapy may include setting the convective air warming machine on the high setting until the desired  $T_{sk}$  is reached, at which time, a re-evaluation of the therapeutic needs and potential contraindications may direct a reduction to a medium setting. Furthermore, the manufacturer recommends that no blankets be placed between the warming blanket and the patient's skin. Removing any insulating layer between the patient and therapy would assist in optimizing the rise in  $T_{sk}$ . Finally, the manufacturer recommends covering the convective air warming blanket with a flannel blanket. In this study, two flannel blankets were placed over top of the convective air blanket. The attempt is to create a micro-environment which excludes ambient effects, but does not cause deflation of the tubes within the convective air blanket.

Another question that needs more study is whether thermal circulation index is an effective indicator of the individual's rate of rewarming over a defined period of time. In this study, those individuals with a TCI of  $> 0.30$  at the beginning of the baseline interval had a  $T_{es}$  rate of rise of  $0.0040$  to  $0.0069^{\circ}$

C/minute during the warming interval. At the end of the 10 minute baseline interval, the TCI value separating those individuals with a more rapid rate of rewarming ( $T_{es}$  change of 0.0040 to 0.0069° C/minute) from those with a slower rate of rewarming ( $T_{es}$  rate of rise of 0.0004 to 0.0007° C/minute) increased to a TCI of > 0.41. However, the change in TCI values from the beginning of baseline to the end of baseline does not distinguish the subjects into two groups. Hence, further study is needed, but TCI at entry into postanesthesia recovery may predict an individual's rate of rewarming with a convective air warming therapy.

From the results of this study, one must question the use of forearm-finger temperature gradient as a measure of vasoactivity of the forearm. However, forearm skin temperature displays a more consistent relationship than forearm-finger temperature gradient, but the relationship is still variable. Hence, more study is needed to determine whether forearm temperature is an effective measure of FBF clinically. Furthermore, study is needed to validate the results of this study in postoperative patients.

#### Future Research

This study needs to be reproduced in a clinical setting to determine whether findings within the laboratory study are reproducible in populations which have greater decreases in central temperature, in populations who have undergone surgery and have both an uncontrolled amount of exposure to the environment as well as anesthetics. Further investigation is needed on the duration of afterdrop to determine whether various factors, such as amount of exposure, alters the duration of exposure. Further investigation is needed on the effect of physical fitness and the amount of afterdrop.

Further work is needed to determine whether a minimal  $T_{sk}$  is required before  $T_{es}$  will begin to rise with an external rewarming device. Must the  $T_{sk}$  be  $35^{\circ}$  C before enough vasodilatation can occur to actually result in convective heat transfer from the skin surface that is discernible in the core temperature. Does the body mass affect the  $T_{sk}$  level at which a discernible rise in core temperature occurs?

Also, study is needed to address environmental issues which affect the convective air warming blanket. Studies include addressing the variance in blanket temperature, attempting to improve the effectiveness of the microenvironment such as decreasing radiant and convective heat loss by covering the convective air warming blanket with a plasticized aluminum blanket, and evaluating issues of blanket to body fit.

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APPENDIX A

Women's Ideal Body Weight  $\pm$  20%  
(Ages 25-59 years)

Height**		Weight Ranges***	
Feet	Inches	Ideal Weight*	80-120% Ideal
4	10	102-131	82-157
4	11	103-134	82-161
5	0	104-137	83-164
5	1	106-140	85-168
5	2	108-143	86-172
5	3	111-147	89-176
5	4	114-151	91-181
5	5	117-155	94-186
5	6	120-159	96-191
5	7	123-163	98-196
5	8	126-167	101-200
5	9	129-170	103-204
5	10	132-173	106-208
5	11	135-176	108-211
6	0	138-179	110-215

\* Based on 1983 Metropolitan Height and Weight Tables

\*\* Height based on wearing shoes with 1 inch heels

\*\*\* Weight based on wearing indoor clothing estimated at 3 pounds.  
(Poleman & Peckenpaugh, 1991)

APPENDIX B

Study Screening Tool

Name \_\_\_\_\_ Interview Date \_\_\_\_\_

Phone \_\_\_\_\_ Address \_\_\_\_\_

Age \_\_\_\_\_ Height(stated) \_\_\_\_\_ ft \_\_\_\_\_ in Weight (stated) \_\_\_\_\_ lbs

Allergies \_\_\_\_\_

Tape Allergy \_\_\_\_\_ Heat Allergy \_\_\_\_\_

Medications (Prescription/Non-prescription) \_\_\_\_\_

**Medical History**

Neurological \_\_\_\_\_

\_\_\_Raynaud's \_\_\_Spinal cord injury \_\_\_Craniotomy \_\_\_Migraine \_\_\_Tremors

Cardiovascular \_\_\_\_\_

\_\_\_MI \_\_\_Hypertension \_\_\_Angina \_\_\_Valve dis. \_\_\_Stroke

Pulmonary \_\_\_\_\_

Mouth breather/Nose breather

\_\_\_Cigarette/cigar smoker \_\_\_Emphysema \_\_\_Asthma

Gastrointestinal \_\_\_\_\_

\_\_\_Difficulty swallowing pills

Genito-urinary \_\_\_\_\_

Premenopausal/Menopausal \_\_\_\_\_ Date of last menses

\_\_\_Pregnant \_\_\_Hysterectomy (date/therapy) \_\_\_\_\_

Integumentary \_\_\_\_\_

Endocrine \_\_\_\_\_

\_\_\_Diabetes \_\_\_Thyroid (hyper/hypo)

Communicable diseases \_\_\_\_\_

\_\_\_Hepatitis \_\_\_Tuberculosis \_\_\_HIV+

Surgery \_\_\_\_\_

**Study Data**

Date/time (study day) \_\_\_\_\_ Menstrual phase \_\_\_\_\_

Forearm circumference \_\_\_\_\_ Whitney gauge # \_\_\_\_\_

Height measurement \_\_\_\_\_ ft \_\_\_\_\_ in Weight measurement \_\_\_\_\_ lbs BMI \_\_\_\_\_

Skinfold thickness \_\_\_\_\_ Forearm \_\_\_\_\_ Chest \_\_\_\_\_ Thigh \_\_\_\_\_ Calf \_\_\_\_\_

## APPENDIX C

### UNIVERSITY OF WASHINGTON

#### CONSENT FORM

**TITLE: Interactions Between Cardiovascular and Thermoregulatory Effector Systems.**

Dr. George L. Brengelmann, Professor, Department of Physiology & Biophysics, telephone: 543-4038, 543-9094.

Ms. Margaret V. Savage, Visiting Scientist, Department of Physiology & Biophysics, telephone: 543-9094

Dr. Alec Rooke, Assistant Professor, Department of Anesthesiology, telephone: 764-2114, 543-2672.

Ms. Diane Audiss, Clinical Nurse Specialist, Harborview Hospital and Graduate Student, School of Nursing; telephone: 223-5889

#### Investigators' Statement

#### PURPOSE AND BENEFITS

In our research we try to learn more about how the cardiovascular system and the temperature regulation system work together in the human body. We ask questions like: How is the regulation of blood pressure affected when a person reacts to heat by developing a high rate of blood flow to the skin? How does an exercising person manage to supply enough blood flow to active muscles and, at the same time, supply enough blood flow to skin to get rid of the heat produced in the muscles? How important is skin temperature to control of the mechanisms that regulate body temperature compared to internal temperature? How is temperature control affected if only part of your skin is hot or cold and if you are only receiving information on the thermal condition of part of your body?

It may seem that questions like these could be answered in a few months and that we would quickly run out of work. The fact is, that our present research is the outgrowth of over 25 years of collaboration between physiologists and physicians at the University of Washington. We have worked with dozens of subjects who have gone through various stresses such as heating or exercise or combinations of stresses. We have gradually accumulated a store of knowledge. We have mapped out how skin blood flow and sweat rate change in relationship to internal temperature and at a couple of skin temperatures in humans lying down in the supine position and also in the upright posture. We have observed the changes in the cardiac output, i.e., the amount of blood the heart pumps per minute, under similar conditions. We also have made advances in understanding what happens to pressures in both the arteries and veins under these conditions, and how the body deals with the shifts in blood volume that occur. We see ahead a long path of more work along these lines. We haven't any idea how sweating and skin blood flow would be modified if internal temperature could be brought back down after a period of heating without changing skin temperature. We don't know whether the signals from the skin are weighted in proportion to how much of the skin is hot or cold. We don't know whether the temperature of the spinal cord is important in human

temperature regulation, as it is in animals. We don't know whether responses are modified by the rate at which internal temperature is changing and we don't know why skin blood flow and sweat rate lag behind the control signals issued by the brain. We don't know whether people of different ages respond differently to heat stress.

Our present experiments are part of a series designed to answer these questions. The procedures that subjects go through are of no direct benefit to them, but we hope that the contribution to knowledge that results will make it worth their efforts.

## PROCEDURES

1. Temperature Control. You will be asked to wear polypropylene long underwear that aids in the uniform distribution of water. To control body temperature, a fine mist of water is sprayed uniformly over the body surface from many tiny orifices in a system of plastic hoses. Water temperature is monitored continually and controlled manually.

In some instances we want to control body temperature with the use of a warm air blanket (Bair Hugger). Air circulated through the blanket will be distributed uniformly over the body surface from many tiny orifices in the blanket. The air temperature is monitored continually and is controlled manually.

2. Skin Temperature Measurement. Skin temperature is measured by small disks stuck directly to the skin with adhesive applied from a spray can. This material is similar to the adhesive on surgical tape. (You should let us know if you have had reactions to tape in the past--any rash or reddening after use of a band-aid, for example.) These temperature sensors are connected to our equipment through wires.

3. Internal Temperature Measurement. The best place for us to measure internal body temperature is inside the esophagus because it passes so close to the heart. The temperature sensor is inside a plastic tube that is soft and flexible so that it is not uncomfortable to have in the esophagus for long periods. Getting the probe into the esophagus can be uncomfortable for inexperienced subjects. It can be done two ways: through the mouth or through the nose.

Many of our subjects prefer to swallow the probe. To make this easier a small gelatin capsule filled with gelatin is attached to the end of the probe. You then proceed just the way you would if you were swallowing a vitamin pill for example. You simply put the capsule in your mouth and swallow it with sips of water. It pulls the probe down into the esophagus. Some people have a tendency to gag when they feel the capsule or probe at the back of the throat. Usually, this can be overcome with a little will power. From then on it is easy, because the probe can be passed back and forth in the esophagus without producing any gagging sensation. After a while, one is hardly aware of the tube in the back of the throat.

For long-term comfort, passing the tube through the nose is preferable. It helps to lubricate the tube with a surgical jelly that is water-soluble. The tube is inserted through a nostril and the subject advances it. It curls downward when it reaches the back of the throat. Then it is necessary to swallow or make a series of swallows so that the tube can be advanced down the esophagus. It helps to take sips of cold water.

To make sure that the probe tip is in the right place, we set up for recording an electrocardiogram from it. As the probe is advanced or retracted, we can

see a characteristic change in the shape of the electrocardiogram that we use to identify the location of the probe tip relative to the heart.

To get the best possible accuracy in our measurement of temperature in the esophagus, we need to avoid the cooling effect of swallowing saliva. The saliva is removed by a small plastic tube with tiny holes in the side (it is normally used as a feeding tube for infants). This is held in the mouth throughout the experiment. We have the subject control the suction with a valve held in one hand. If a sensation of dryness develops and the need to swallow is felt, the subject simply turns down the suction.

We also may record oral and/or rectal temperature. Sometimes these are necessary substitutes when subjects find that they cannot overcome an aversion to swallowing the probe, sometimes we want to get readings of core temperature from as many sites as possible. The procedures are practically identical to the procedures used in doctor's offices and clinics. Instead of thermometers, we use temperature sensors enclosed in flexible plastic tubing, about 1/8 inch in diameter.

For oral temperature, the subject simply holds the probe under the tongue, just as in the common method of taking a temperature with a clinical thermometer. With this method it is very important not to breathe through the mouth or to talk, since this temporarily cools the mouth. We recess from time to time to give the subject a chance to talk.

For rectal temperature, the tip of the tubing is lubricated with a sterile jelly and inserted in the rectum to a depth of about 5 inches. You can do this yourself, or one of the investigators will assist. Once the tubing is in place, you pass the free end under a belt to hold it.

We are also testing an alternative procedure for recording body temperature. This new instrument is already being used in some hospitals. We are testing it to see how well readings compare with other core temperatures, particularly esophageal. It is similar to otoscopes, which are used in routine physical exams to enable looking at the condition of your eardrum. It "looks" at your eardrum with a detector sensitive to the heat radiated from it. We can do this every few minutes with no discomfort other than what you may have felt in physical examinations that included looking into your ear. The part that is placed in your ear is shaped like a little plastic funnel. It fits snugly into the outer ear and is too big to get close to your eardrum. It is kept sterile by a cover that is used in only one subject.

4. Blood Pressure. We measure blood pressure regularly in a way similar to how it is done in doctors' offices, with an automated blood pressure cuff. The cuff is left loosely wrapped around the upper arm throughout the experiment. The cuff is cycled on automatically every 2 minutes; the pressure in the cuff builds up and is slowly released over the next few seconds.

5. Heart Rate. We need to have a continuous record of heart rate. This is obtained from an electrocardiogram that requires two electrodes on the chest. We use electrodes of the standard disposable type used in hospitals. We need to get an especially good record, so we take pains to make sure that the layer of dead skin is removed in the spots where the electrodes are attached. We rub the area with gauze pads saturated with the paste used to make good contact between electrodes and the skin. This is mildly uncomfortable because doing it right requires that the skin actually be reddened.

In some cases we may want to record your heart rate over a longer period of time. To do this we use a Holter monitor (a portable box 4"x6"x1" run by a 9 volt battery) with seven electrodes placed on the chest. We will ask you to wear

this during the experiment. If it is convenient for you we would also ask you to continue to wear the Holter monitor after the experiment for a total of 24 hours.

6. Forearm Blood Flow. We determine how much the blood flow to the skin changes by measuring the blood flow in the forearm where we can temporarily dam up the flow out of the arm for about 15 seconds without interfering with the flow entering the arm. This makes the arm expand slightly. A strand of tubing wrapped around the arm, filled with mercury, is used to measure the tiny change in circumference. This technique requires sensitive measurement apparatus and the cooperation of the subject -- the record can be thrown off just by wiggling a finger.

Two cuffs are placed on the arm: one on the upper arm around the bicep and the other around the wrist. The upper cuff is applied loosely. At intervals, we inflate it to apply a gentle pressure that squeezes the veins shut, but not the arteries. The wrist cuff is used to shut off the blood flow to the hand, which would otherwise interfere with our measurement. This is done by applying a pressure above arterial blood pressure. We also use the wrist cuff as a point of attachment for a sling used to hold the arm above heart level so that the veins will empty between cycles of cuff pressure. We cycle the wrist cuff pressure off for at least one minute out of five so that the hand can get a fresh supply of blood. The hand gets uncomfortable during the four minutes of pressure and tingles when the cuff is released.

We also measure skin blood flow with an instrument based on the Doppler effect. Light from a laser is directed through optical fibers in a thin cable that terminates in a head the size of a shirt button. The head is taped to the skin so that the laser light illuminates a tiny spot of skin. Light that reflects from blood vessels is changed slightly in frequency, depending on their velocity (Doppler shift). The instrument interprets this frequency shift as a measure of skin blood flow. The procedure involves no risk because the laser light is so feeble.

7. Sweat Rate. We measure the rate of sweating by measuring the amount of water picked up by a stream of dry gas passed over the skin surface. A chamber is attached to the skin to contain the gas stream. We use an adhesive patch to hold it down. The tubing attached to the chamber is flexible, but motion is restricted. Thus, when a chamber is mounted on the arm, it is important not to move the arm.

#### 8. Oxygen Consumption

Oxygen consumption is measured to determine the amount of heat being generated by the body. Determinations of oxygen consumption are based differences in the amount of coxygen in the inspired and expired air. So that we can analyze air you exhale, you will breathe into a mask placed over your mouth and nose during the experiment.

#### 9. Anthropometric Measures

The size of your body and the amount of fat affect the ability of your body to absorb and give up heat. Therefore, we measure your height and weight prior to the experiment. In addition, skin fold thickness is measured by gently grasping a double layer of skin and applying a caliper to the site. We will measure skin fold thickness at several sites on your body including the torso, arms and legs.

Risk, Stress, and Discomfort. Getting hot or cold is real stress. In the case of being heated you feel hot and uncomfortable, your heart pounds, you sweat profusely. In experiments where you are being cooled you will feel cold and

uncomfortable. You may even shiver a little. We limit the stress based on our observations: internal temperature is kept within safe limits, heart rate is monitored as a backup indication of condition, and we constantly monitor how you are feeling. After the experiment you will feel washed-out and you may find yourself more tired than usual that night. We do not go beyond limits that individuals experience when they take a long sauna or get soaking wet in a rain storm. The beauty of our situation is that we can give instant relief just by turning on the cold or warm water.

Our procedures cause additional stress. The experiment seems long and boring. The wrist cuff becomes annoying and the fingers tingle after the pressure in the wrist cuff is released. It may be annoying to have to hold so still during the 10-20 second periods during which the bicep cuff is inflated and also to have to keep from moving under the sweat capsule. You get tired of concentrating on not swallowing and having to control the saliva suction. Sweat may run into your eyes, despite our efforts to keep you face towed dry. You may feel uncomfortable in the cold. If you find yourself stressed to a point that you would not normally be willing to tolerate, say so and we will stop immediately. We don't want anyone to go away saying "I'll never do that again," and besides, measurements from an overly stressed subject would not be valid anyway.

Invasion of Privacy. The data from these experiments are published in scientific journals. They are never identified by the name of the subject except in our data books, which no one is permitted to take out of the laboratory. These data books are kept indefinitely because we may in the future be able to learn more from the data, such as some presently unexpected relationship between skin blood flow and sweat rate. In our publications, we refer to specific subjects by code numbers or letters, e.g., subject A weighed 150 pounds, was 34 years old, and was 175 cm tall. The information that is published is all quantitative data, such as the records of heart rate, sweating, etc.; nothing at all that relates to an individual's personality or individual characteristics as a person is included. We will take appropriate steps to assure your privacy during changing for the experiment. Ordinarily, the men and women of the laboratory personnel will work with a subject during these phases without regard to who is what sex. If your prefer not to have someone of the opposite sex around when your are dressing or undressing, make this known during the interview and we will quietly arrange it.

#### **OTHER INFORMATION**

You are free not to participate and to withdraw from the study at any time without penalty or loss of benefits to which you are otherwise entitled. We want to emphasize that we want you to feel free to stop at any point during your visit to this laboratory. If at any time you feel you are getting to the point of being over stressed, we want you to let us know immediately. We will stop immediately and reverse whatever is the source of the stress. Whether or not you have completed an experiment is not considered in the calculation of reimbursement.

Reimbursement. We want to compensate subjects for their time and any travel expenses. Our present rate is \$10.00 per hour, paid in the form of a check when you are ready to leave. The experiment will last a maximum of three hours. We don't want anyone to be induced to go through an experiment because of the financial reward. In addition to our moral convictions against that, it is true that we don't want data from someone who is covering up stress.

Arrangements for care. In the event of physical injury as a direct result of study procedures, subjects will be cared for on an immediate basis by Dr. Rooke, and if necessary, will be referred for treatment at no cost within the limits of the University's compensation plan.

## SIGNATURE PAGE

---

Investigator

---

Date

**Subject's Statement**

The study described above has been explained to me, and I voluntarily consent to participate in this research. I have had an opportunity to ask questions and understand that future questions I may have about the research or about the subjects' rights will be answered by one of the investigators listed above.

---

Signature of Subject

---

Date

Copies to: Subject  
File

**Emergency Phone Numbers**

Dr. Brengelmann: 543-4038, 632-6961

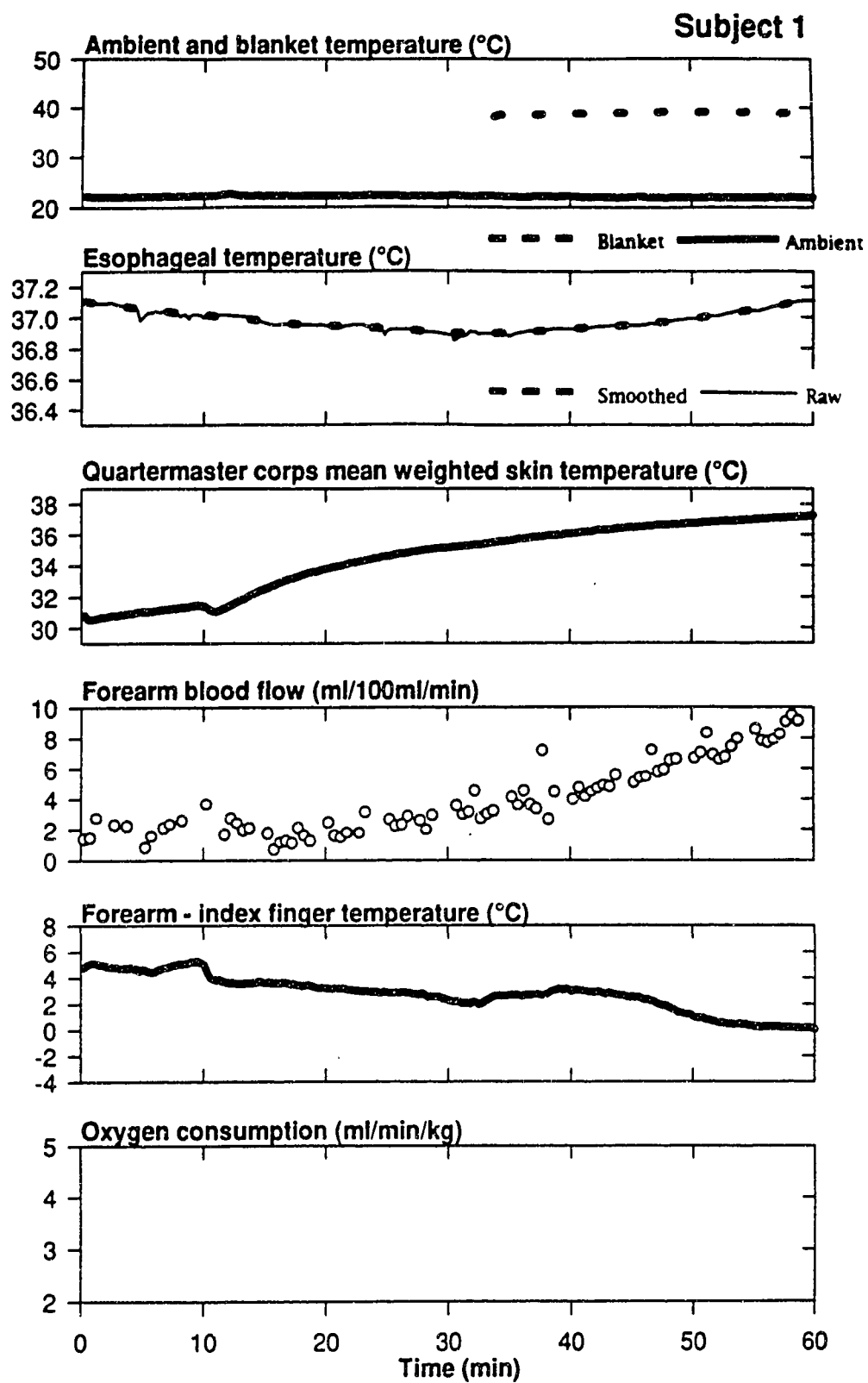
Dr. Rooke: 764-2114, 543-9094, 543-2672

Marg Savage: 543-9094, 545-9120

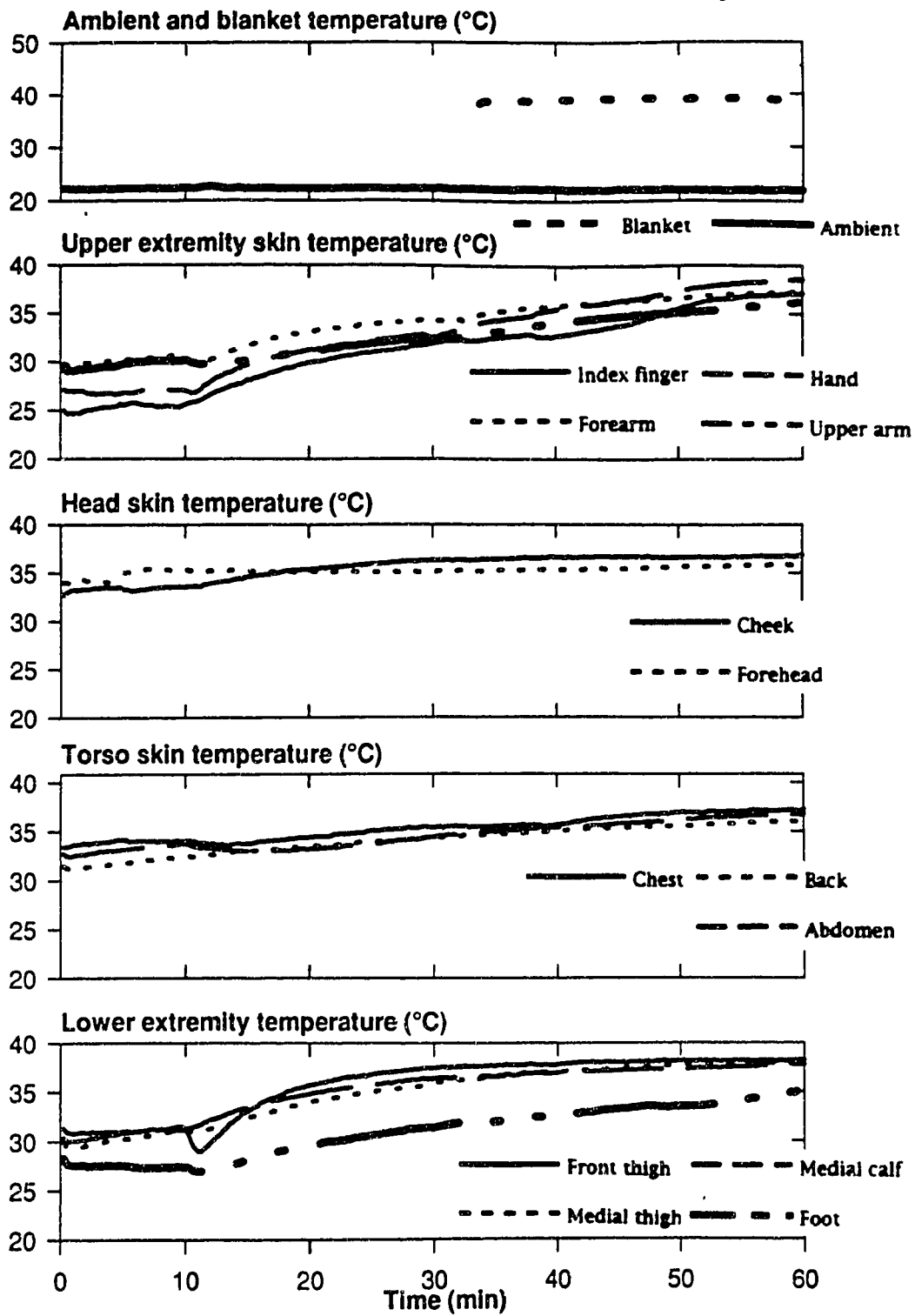
24-hour emergency telephone number: Dr. Alec Rooke, Anesthesiologist,  
Veteran's Administration Hospital 764-2114 or 762-1010.

APPENDIX D

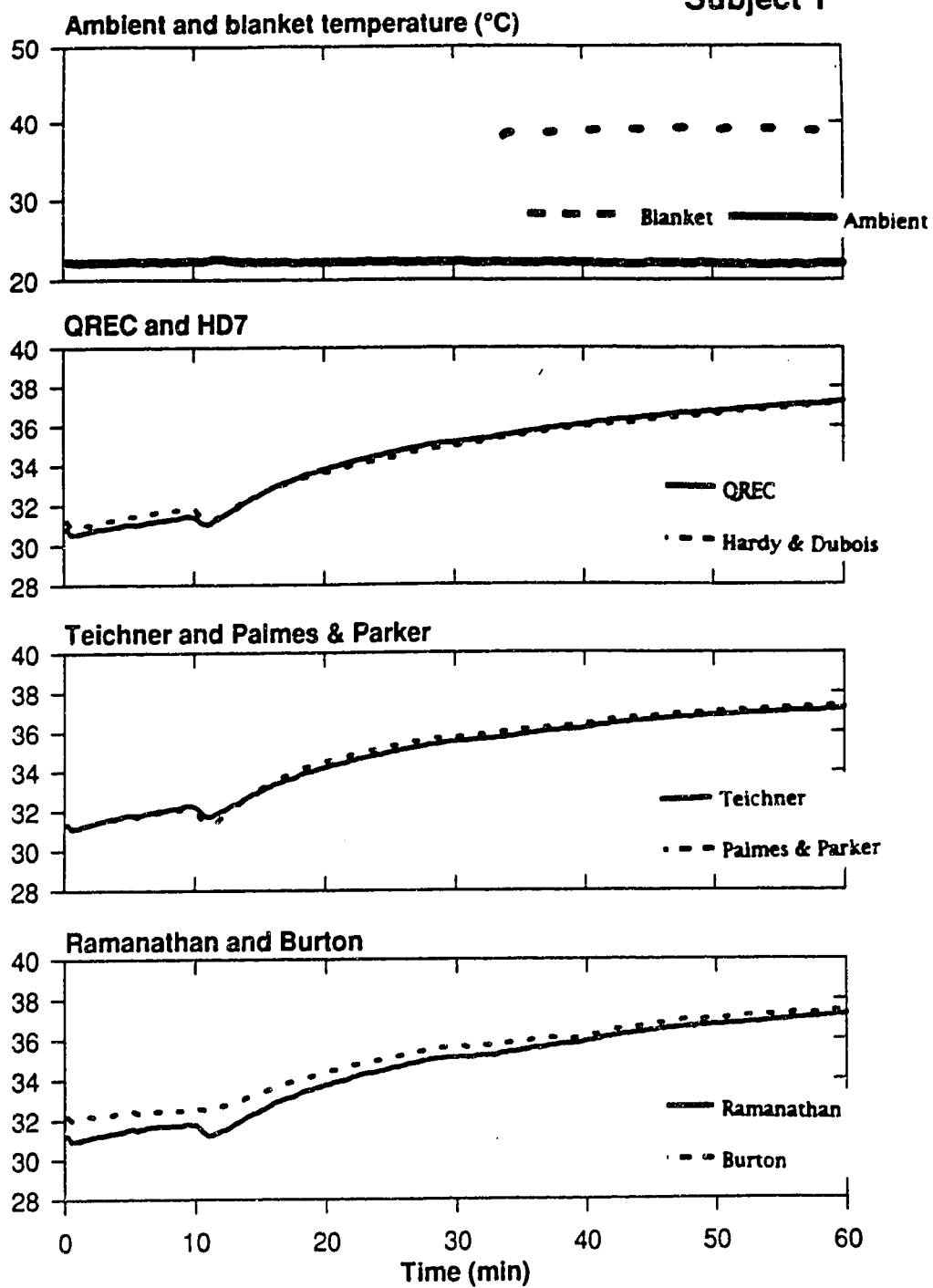
Subject 1's Variable Data



## Subject 1

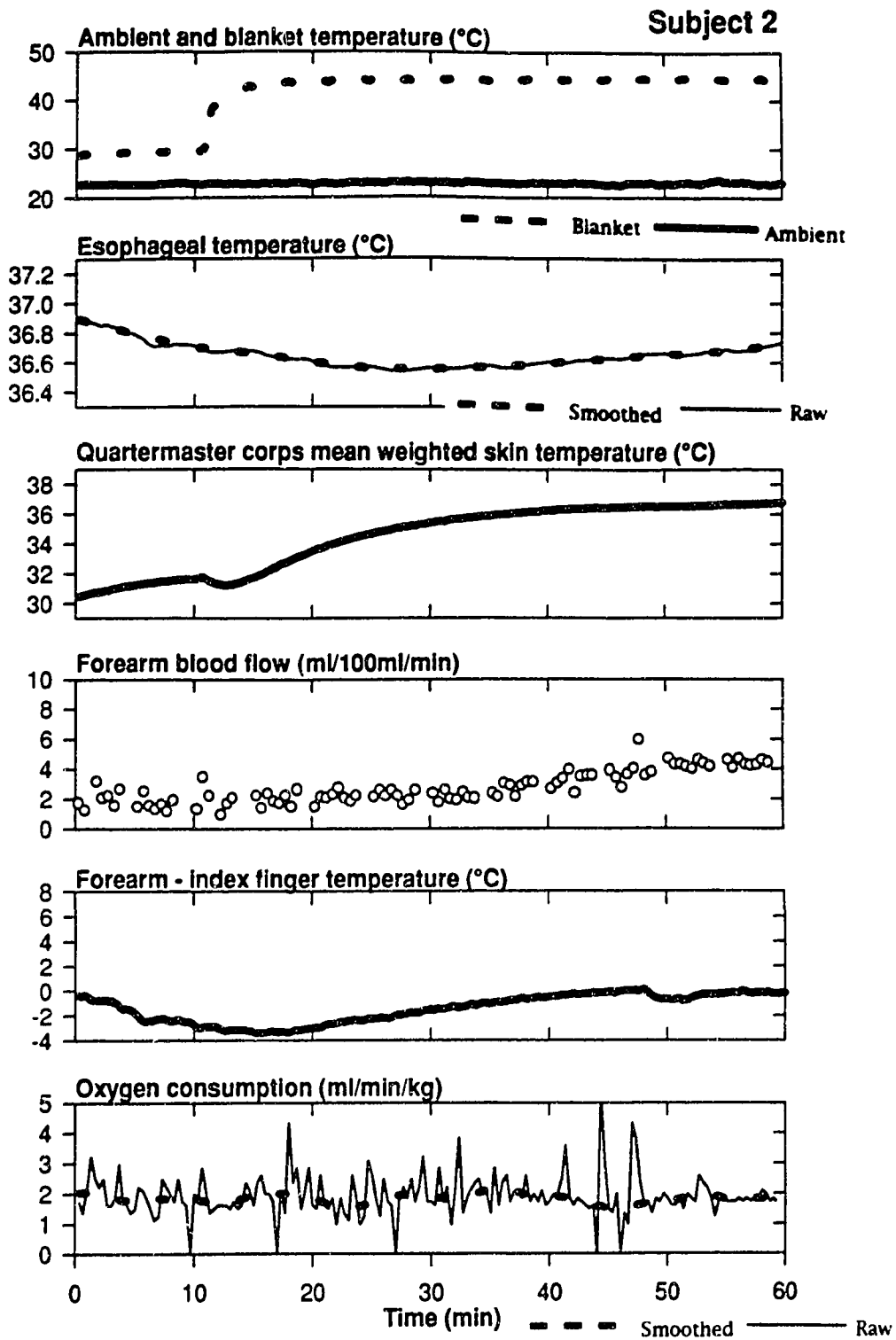


## Subject 1

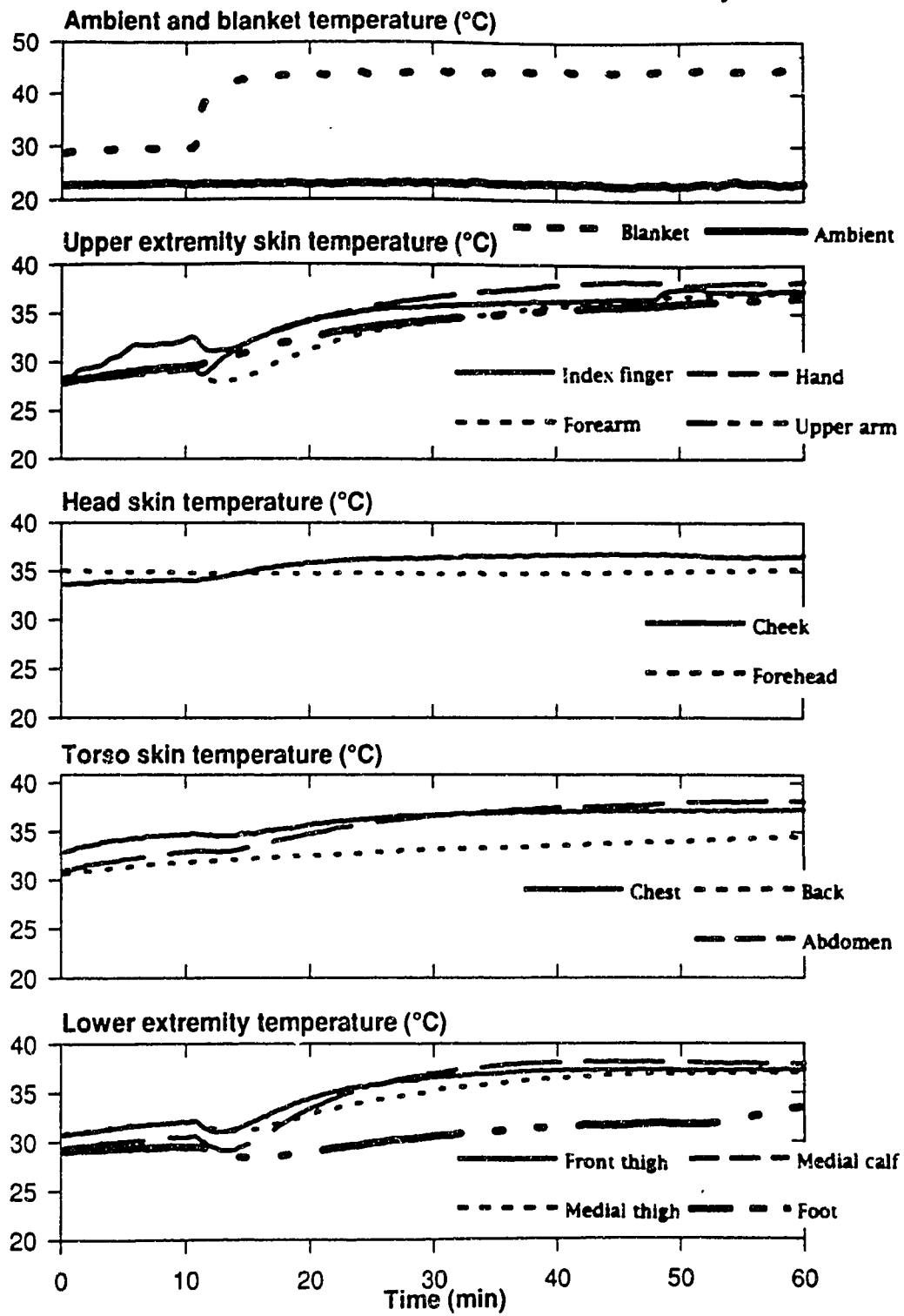


APPENDIX E

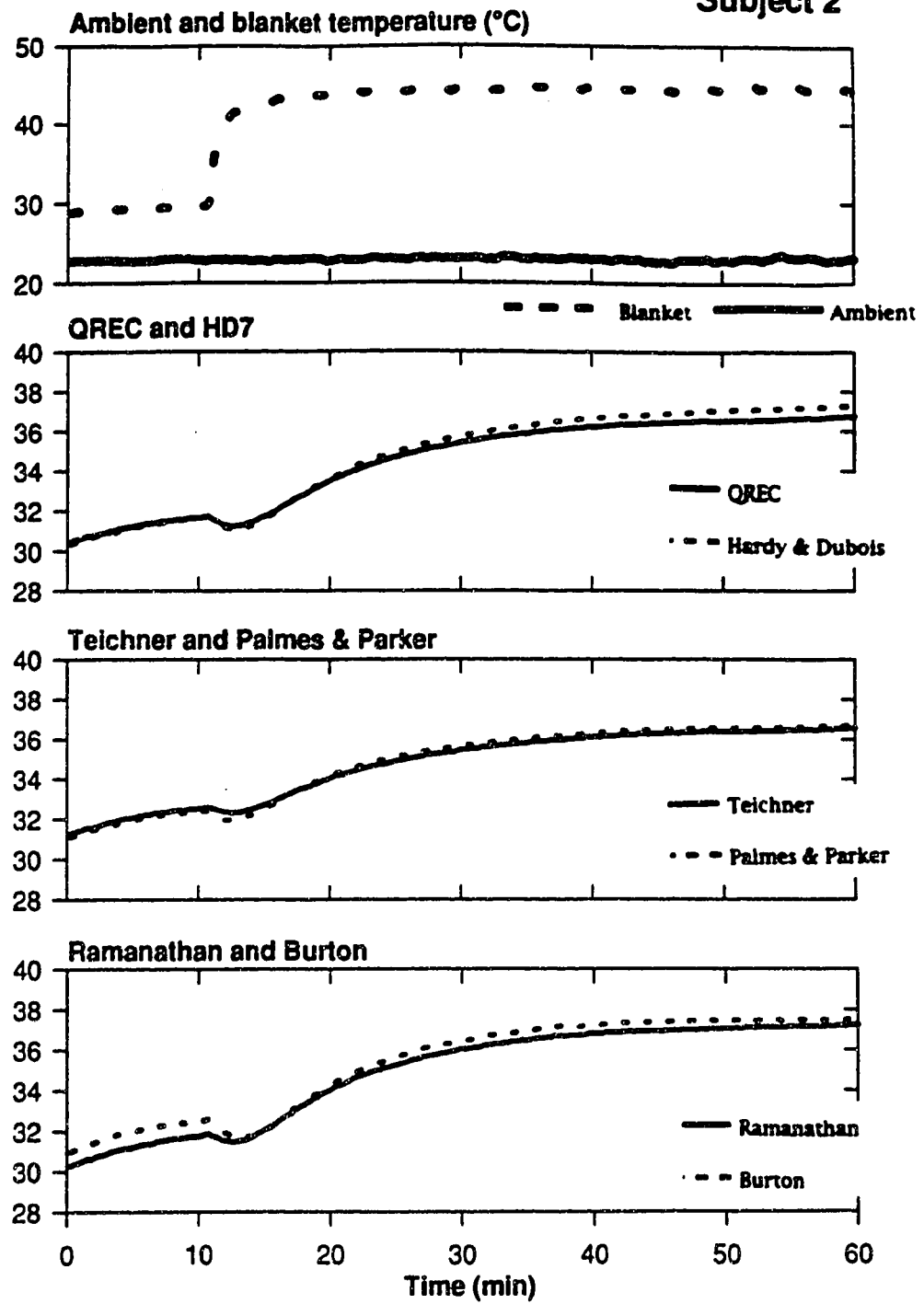
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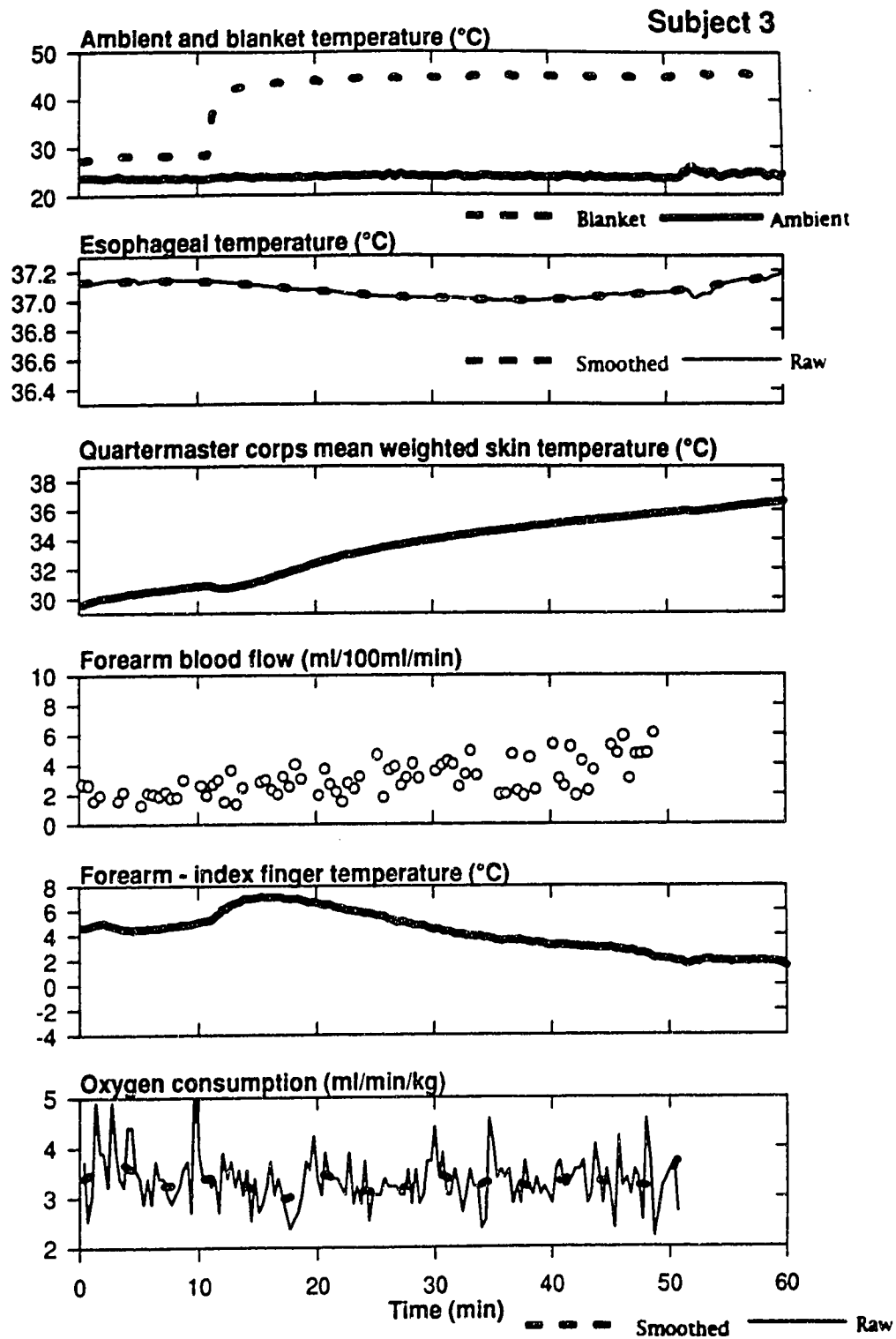


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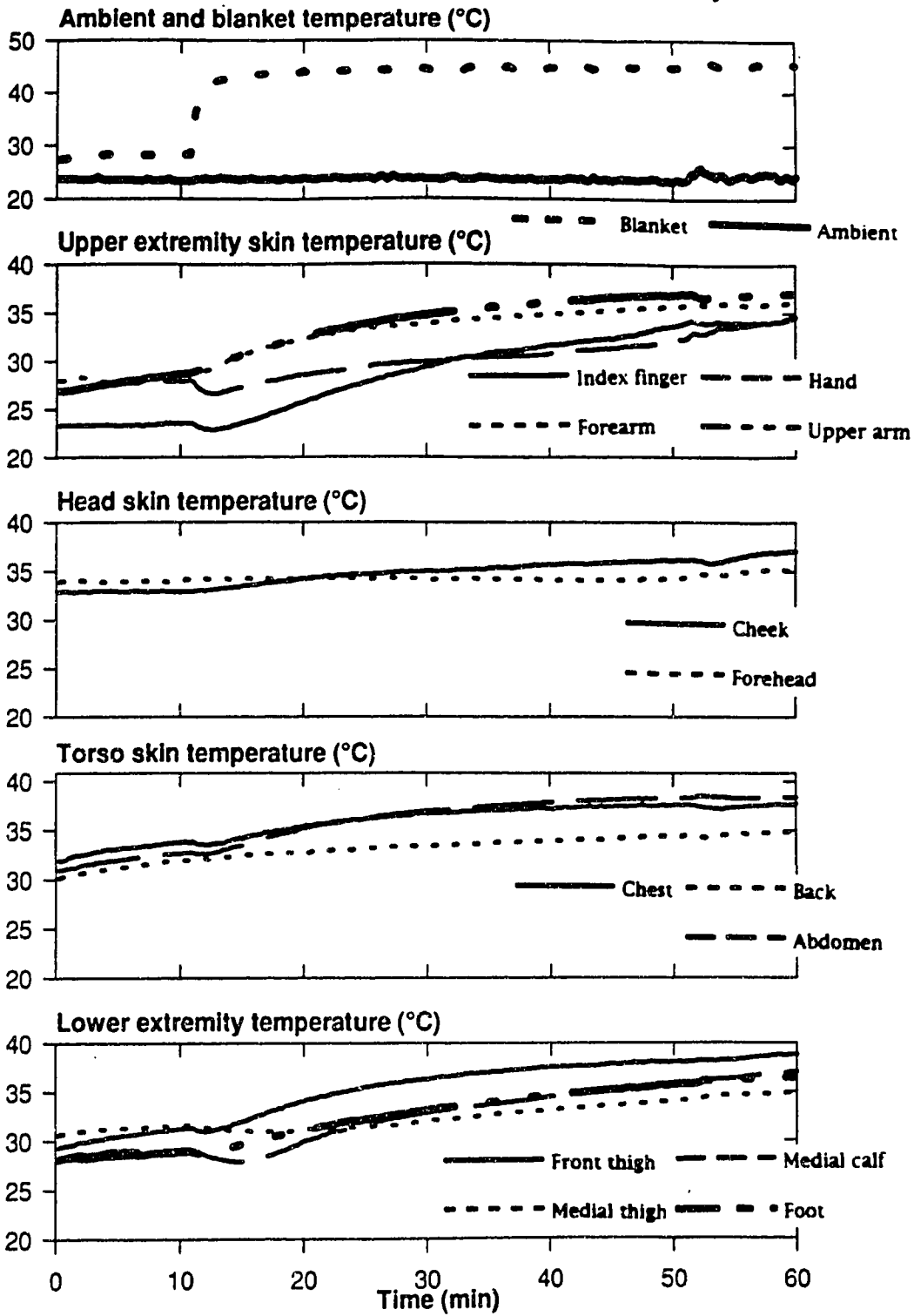


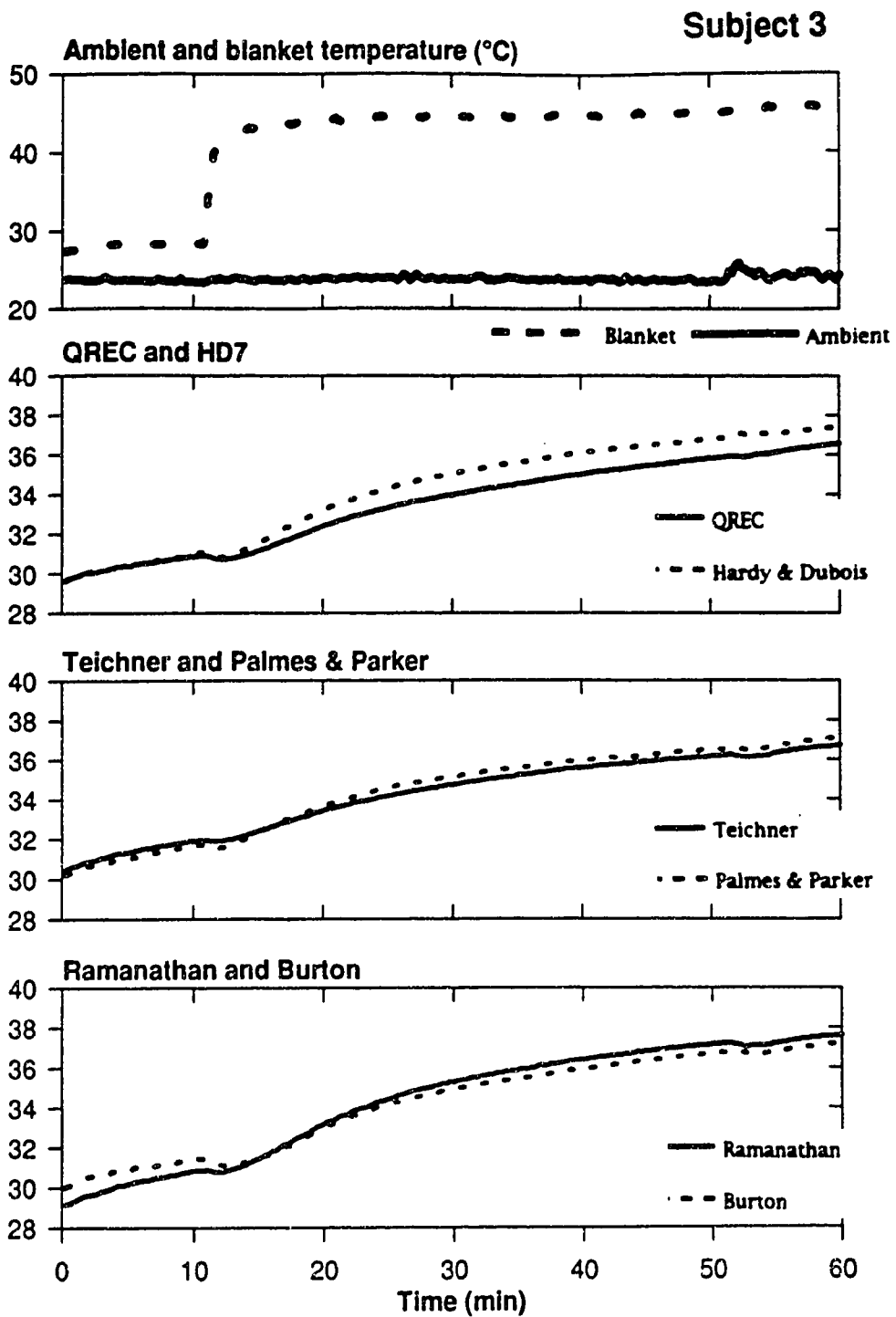
APPENDIX F

Subject 3's Variable Data



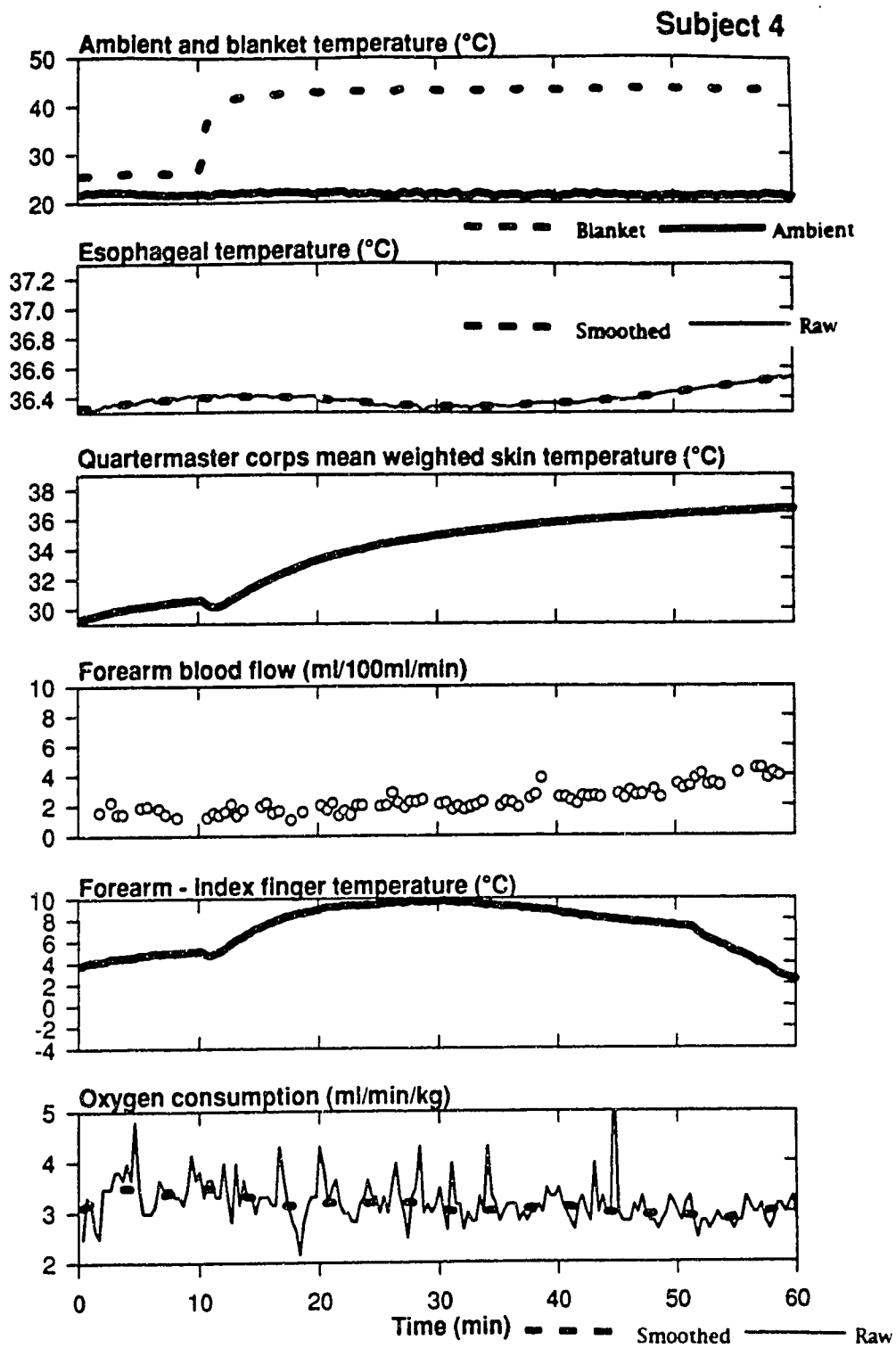
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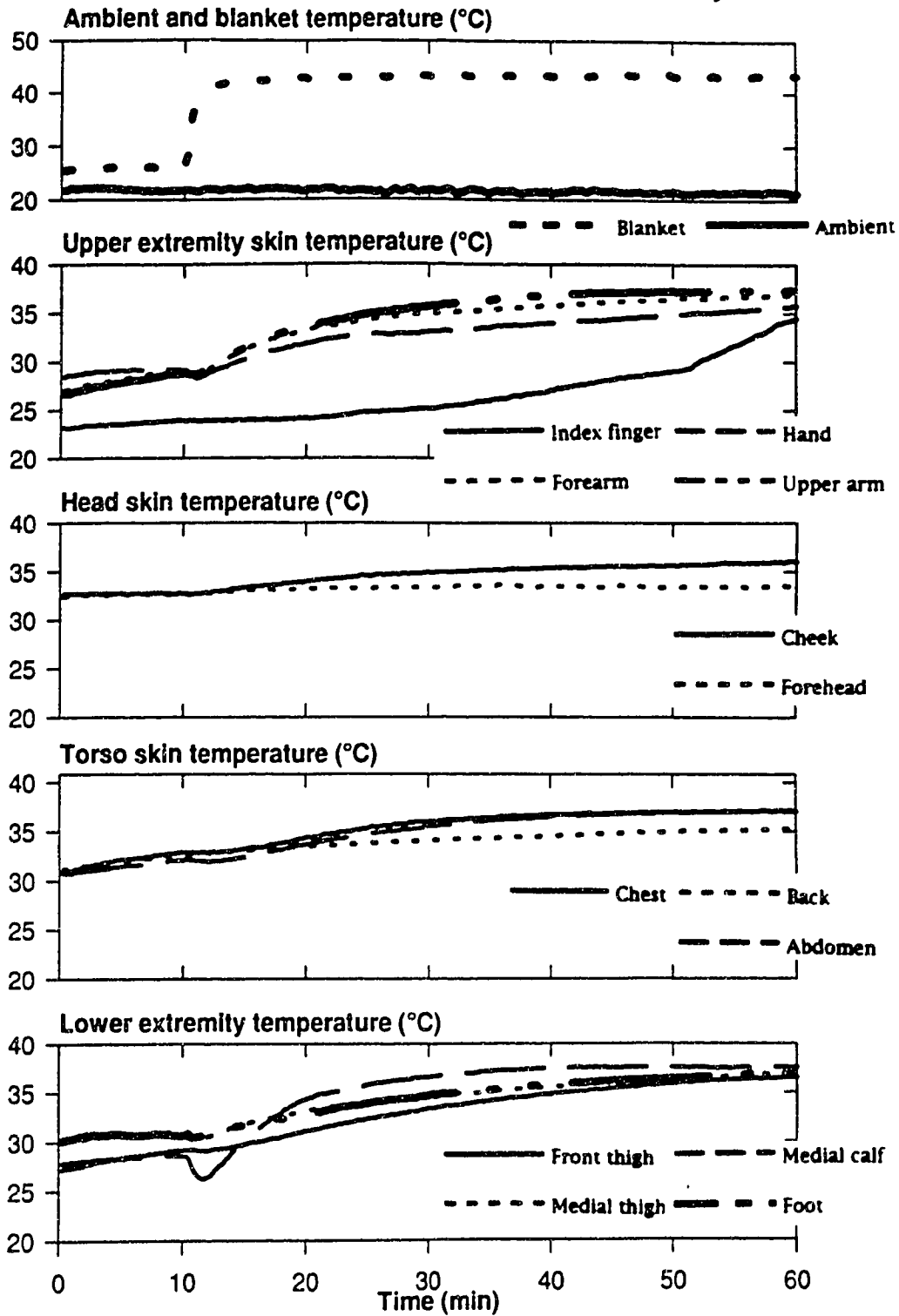


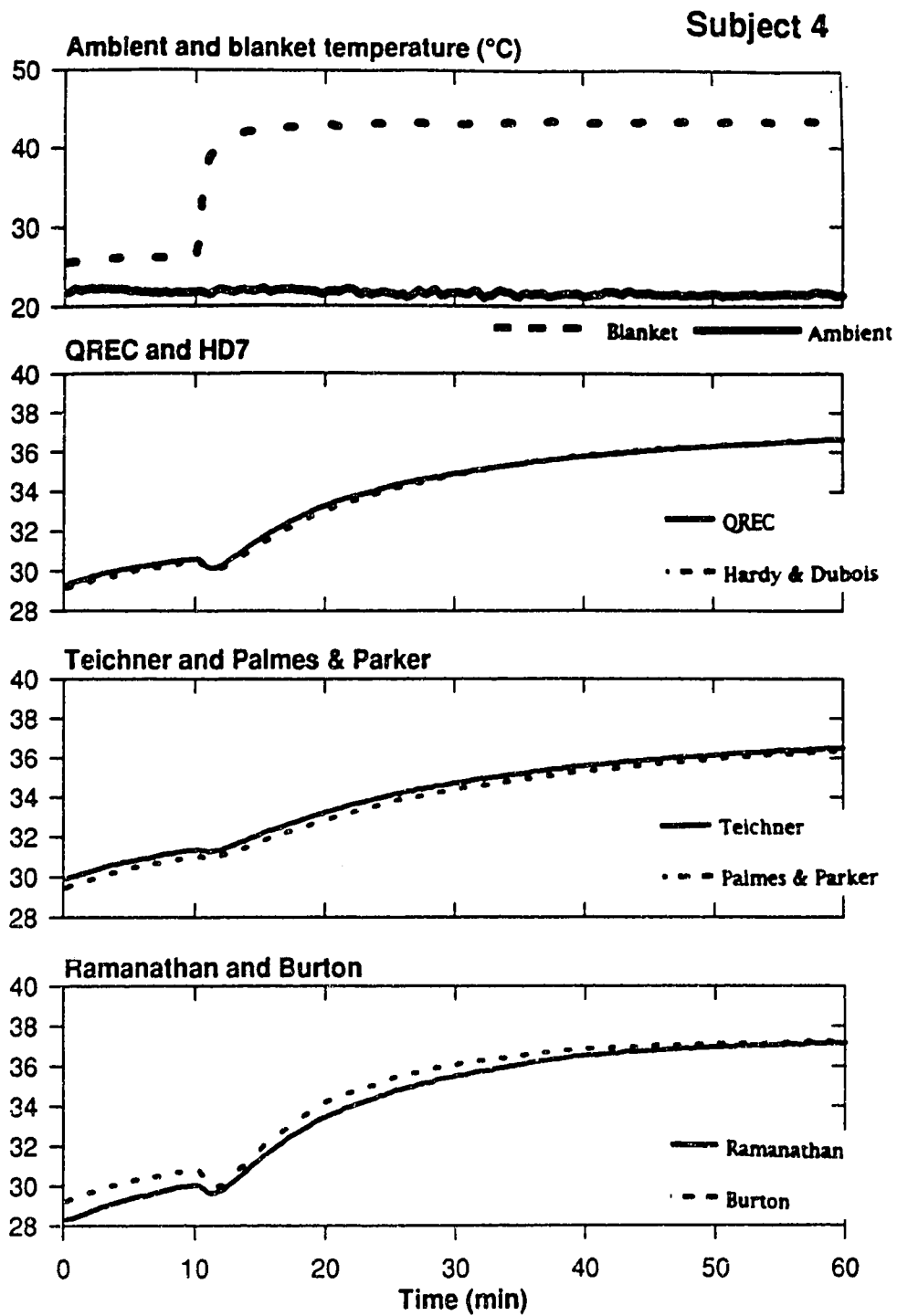
APPENDIX G

Subject 4's Variable Data



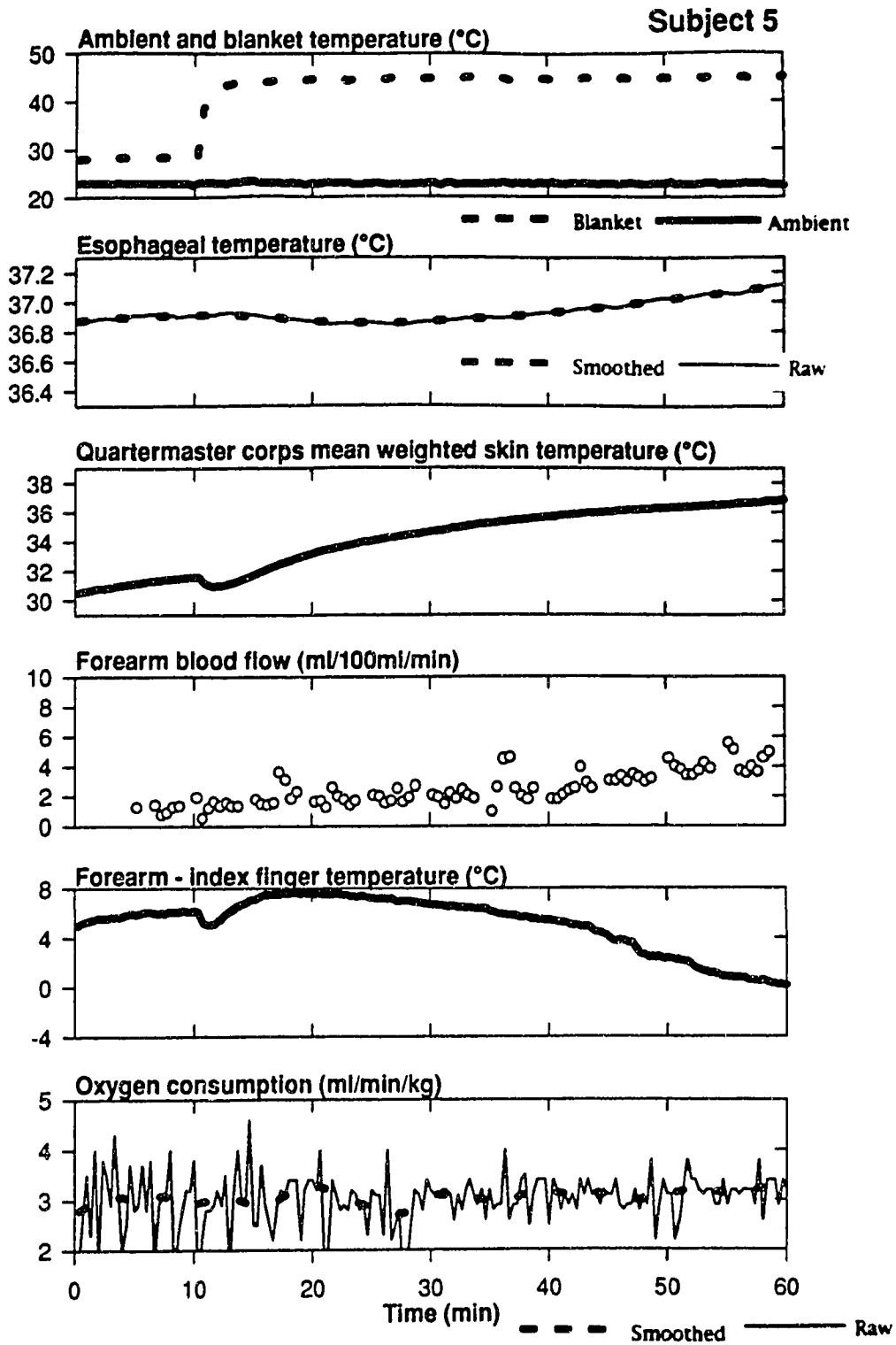
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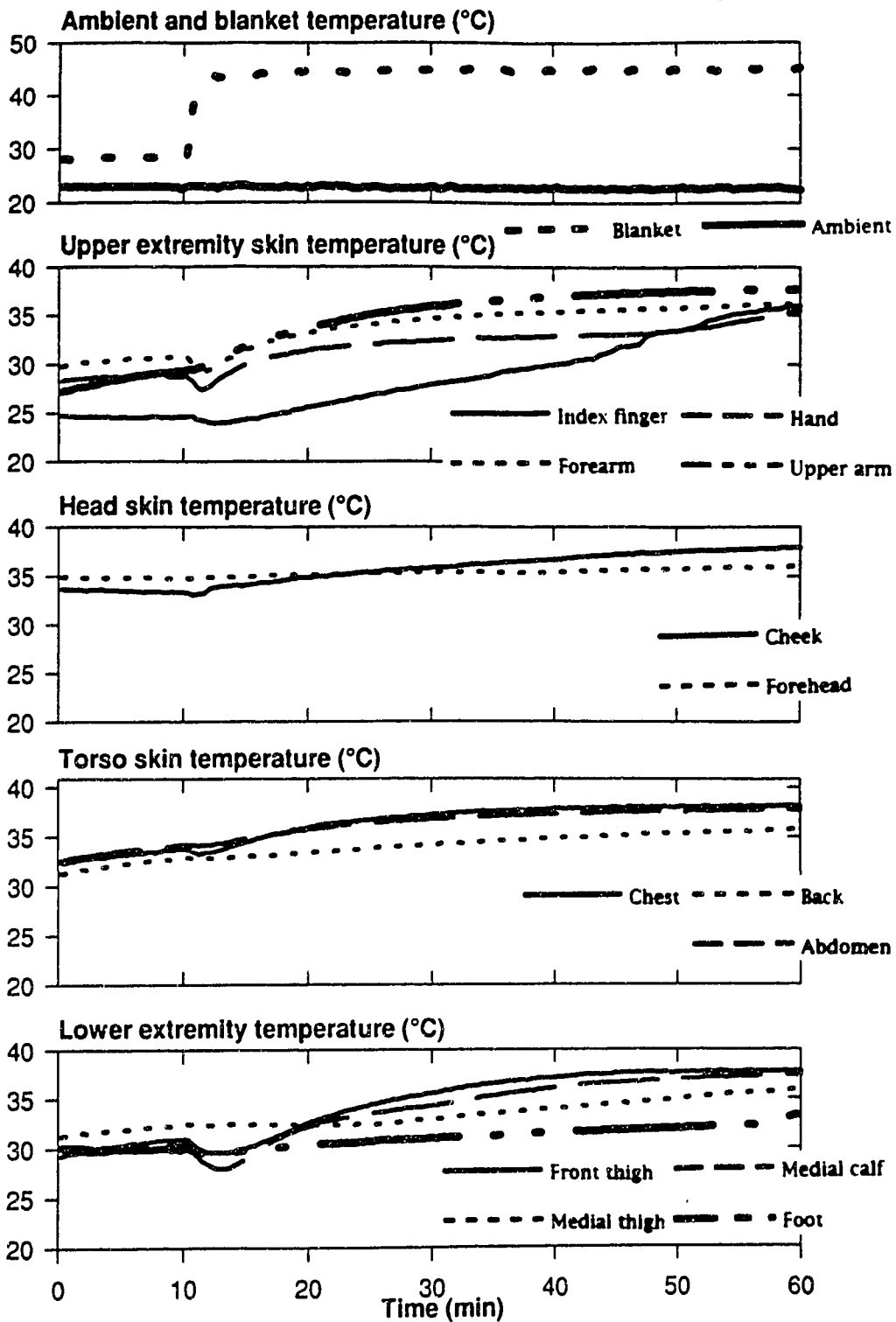


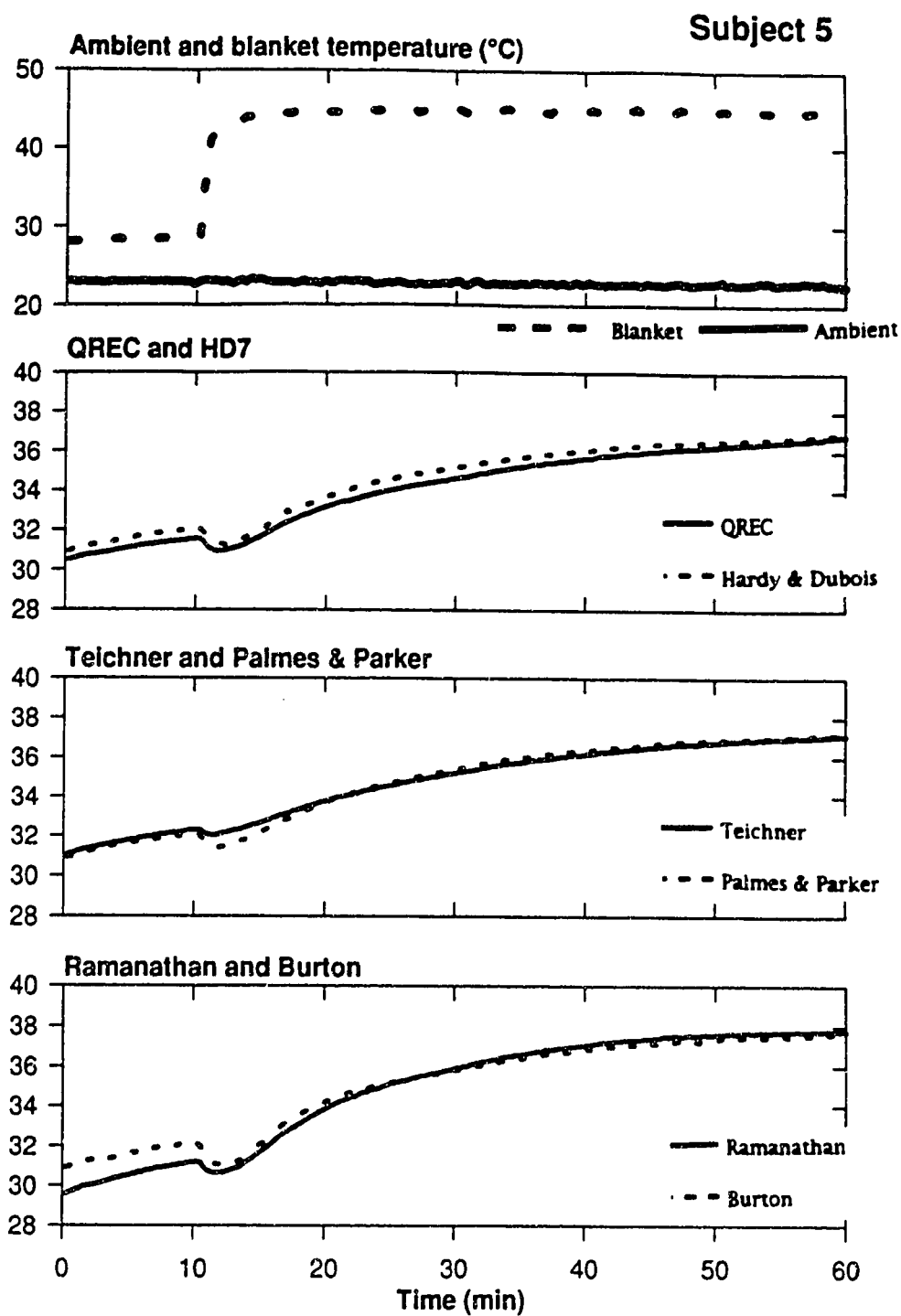
APPENDIX H

Subject 5's Variable Data



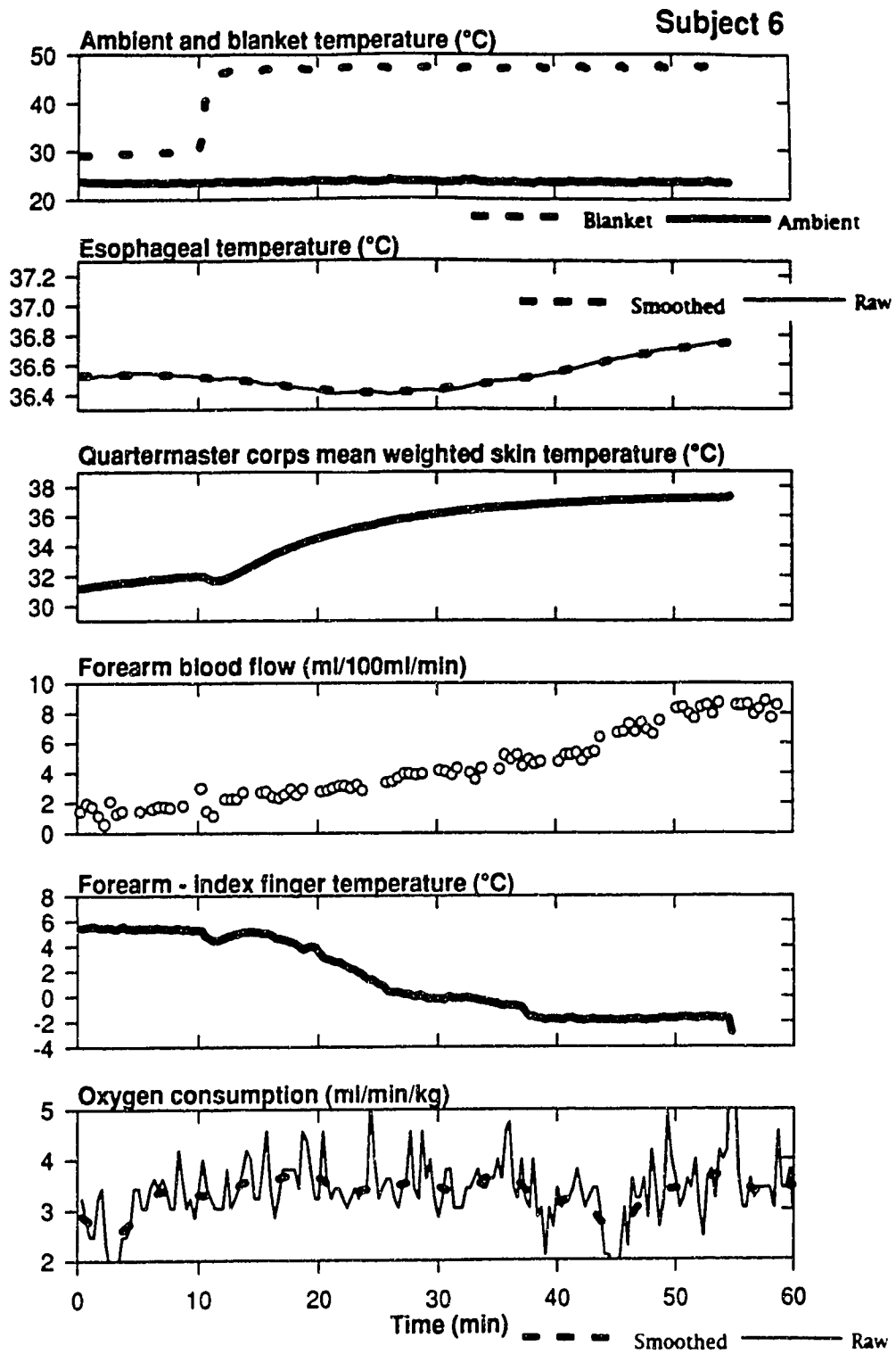
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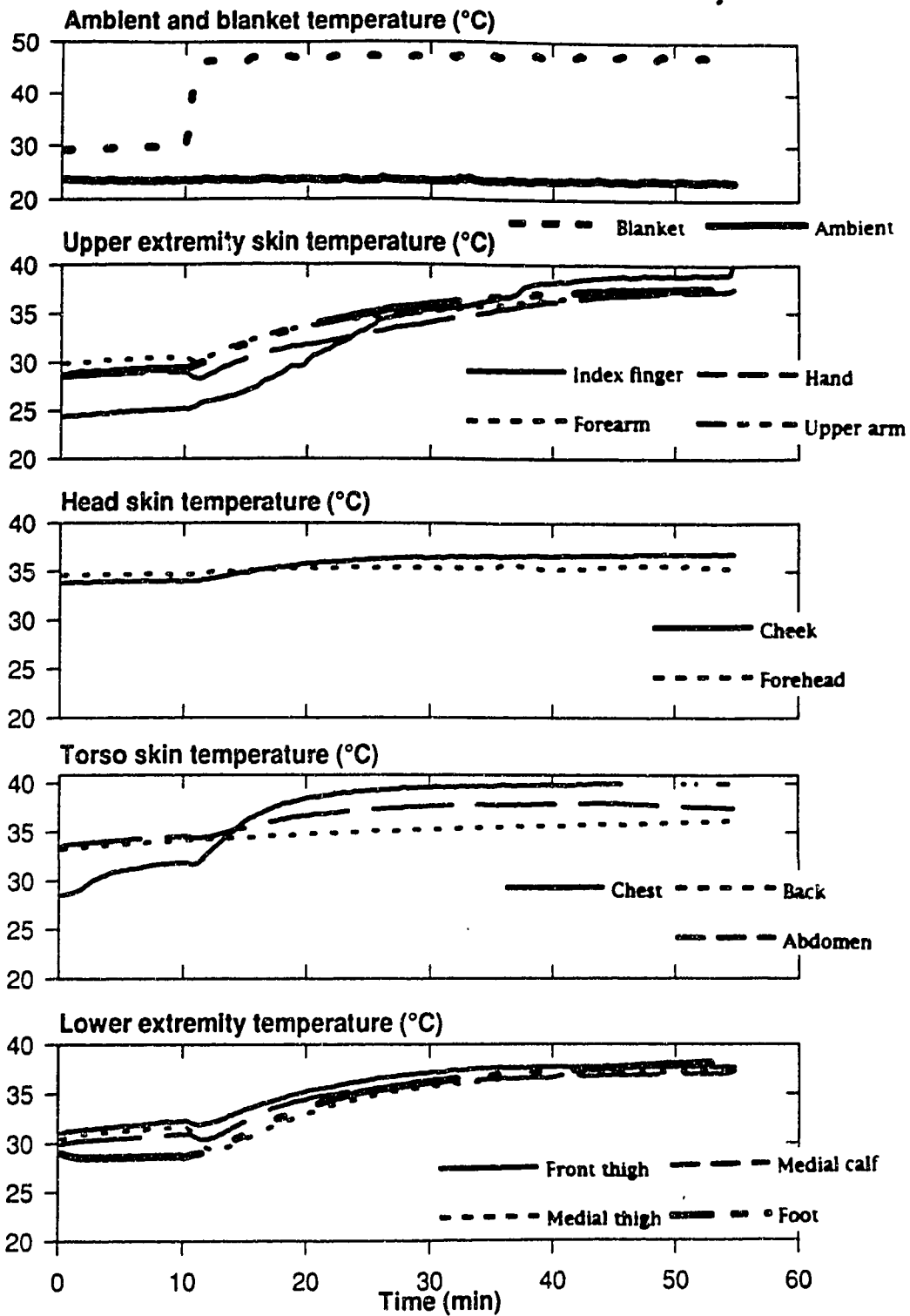


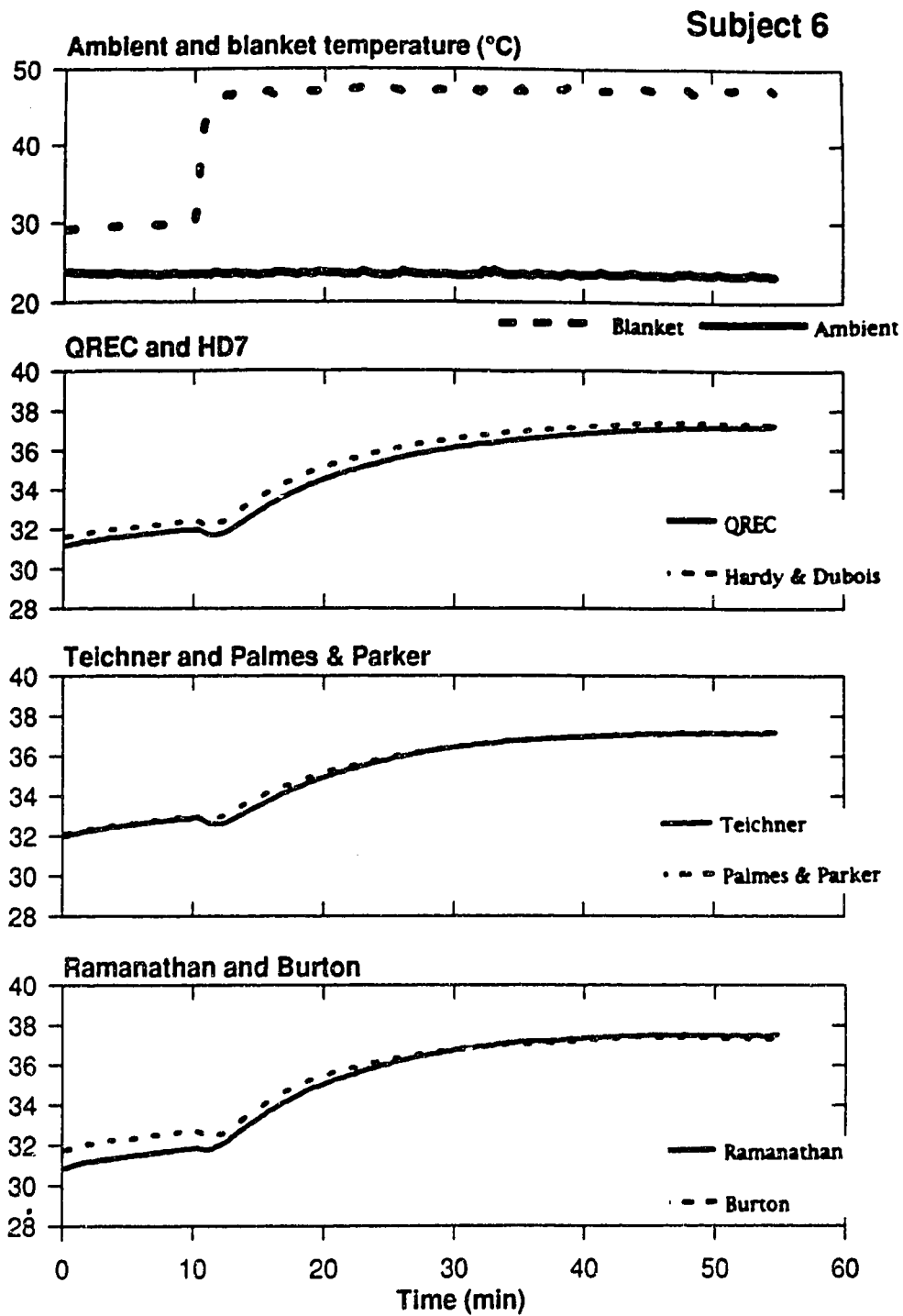
APPENDIX I

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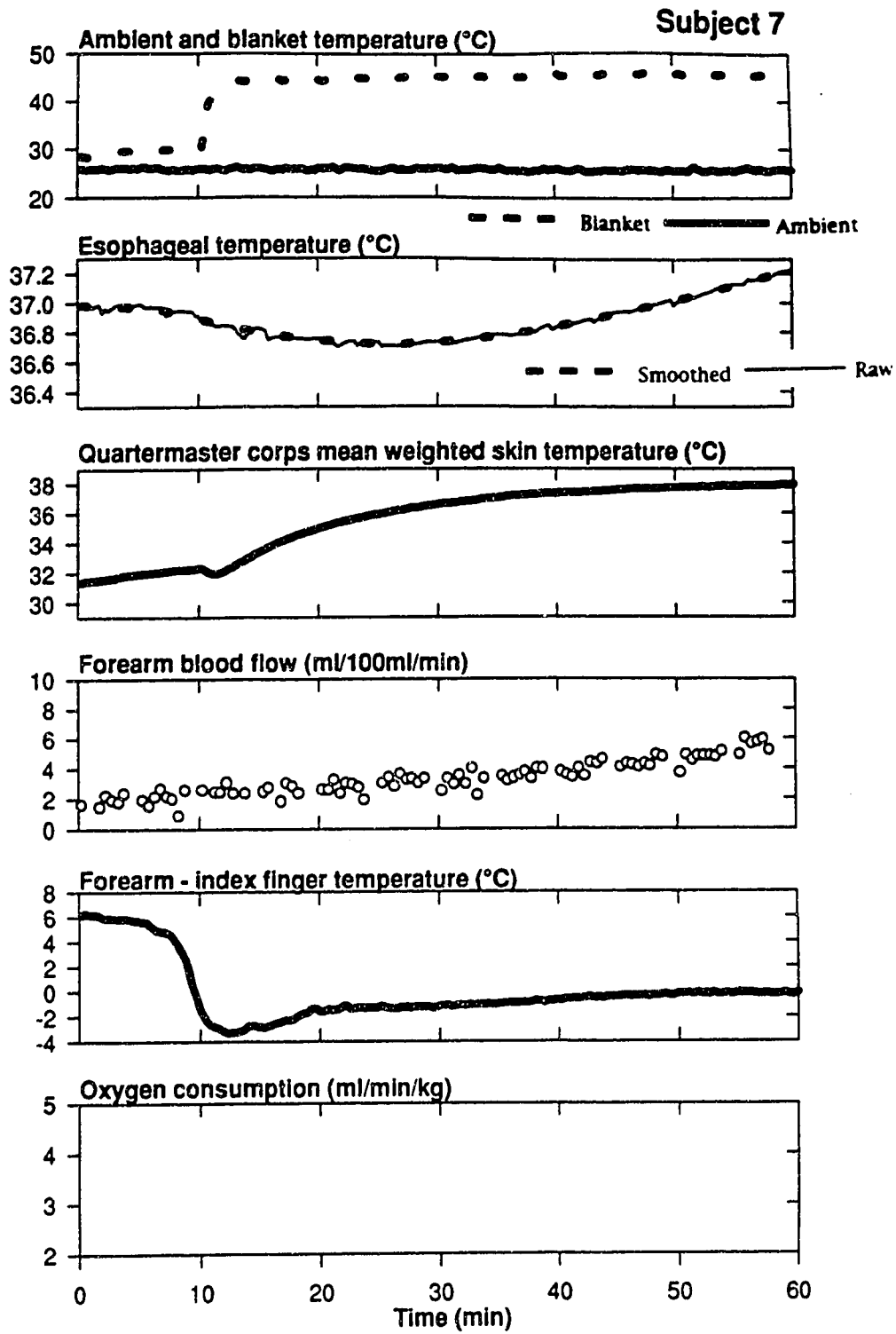
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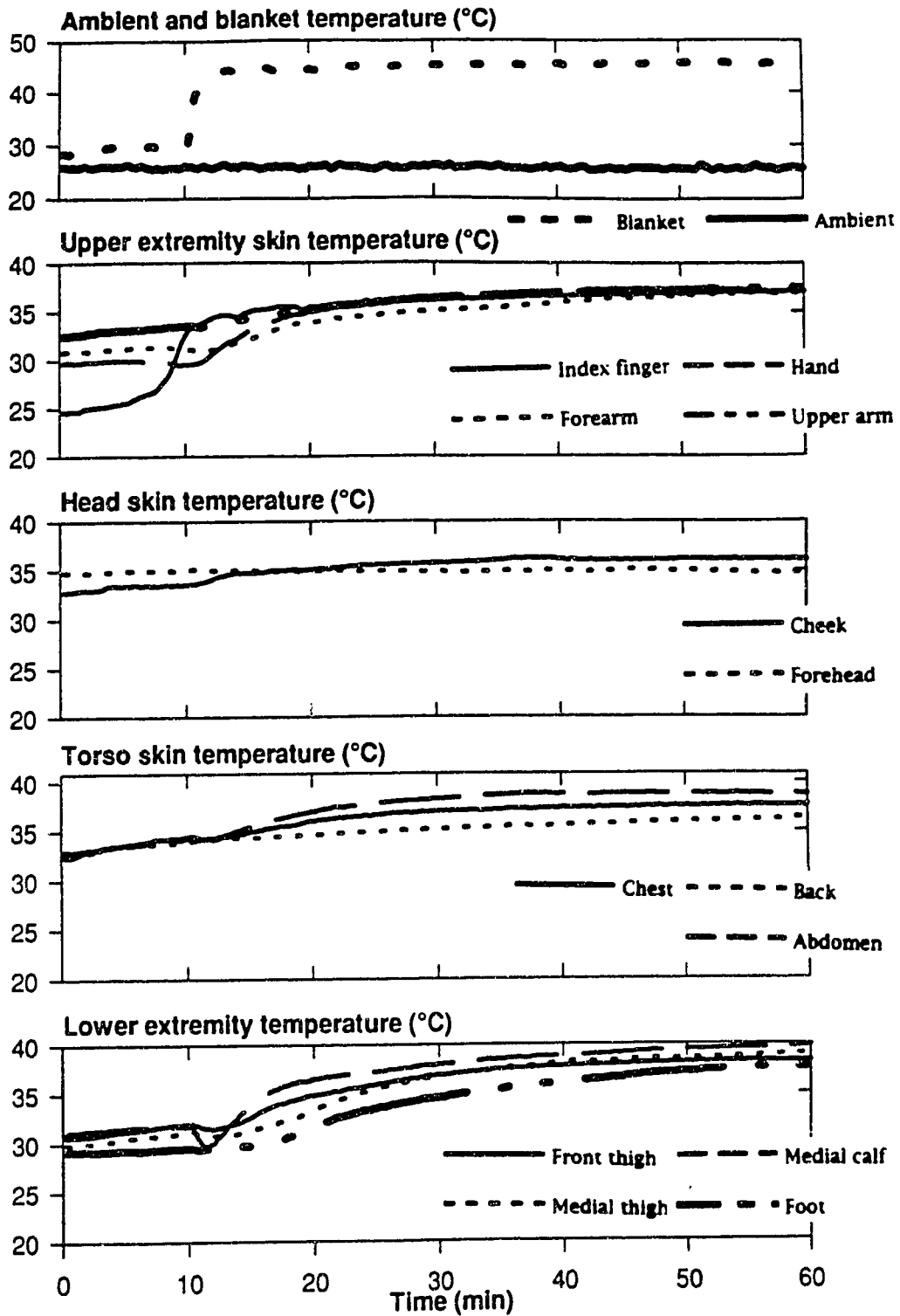


APPENDIX J

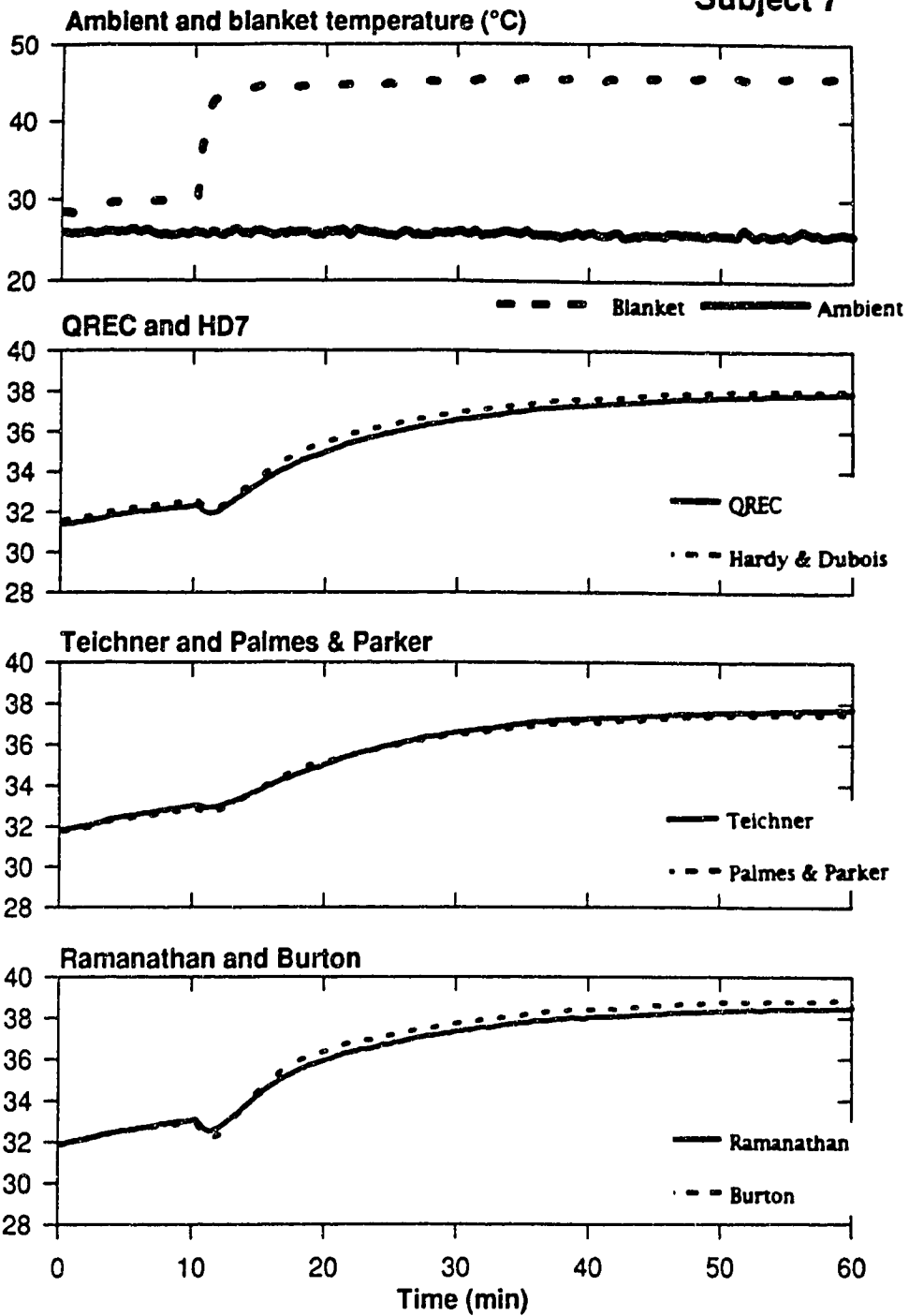
Subject 7's Variable Data



Subject 7

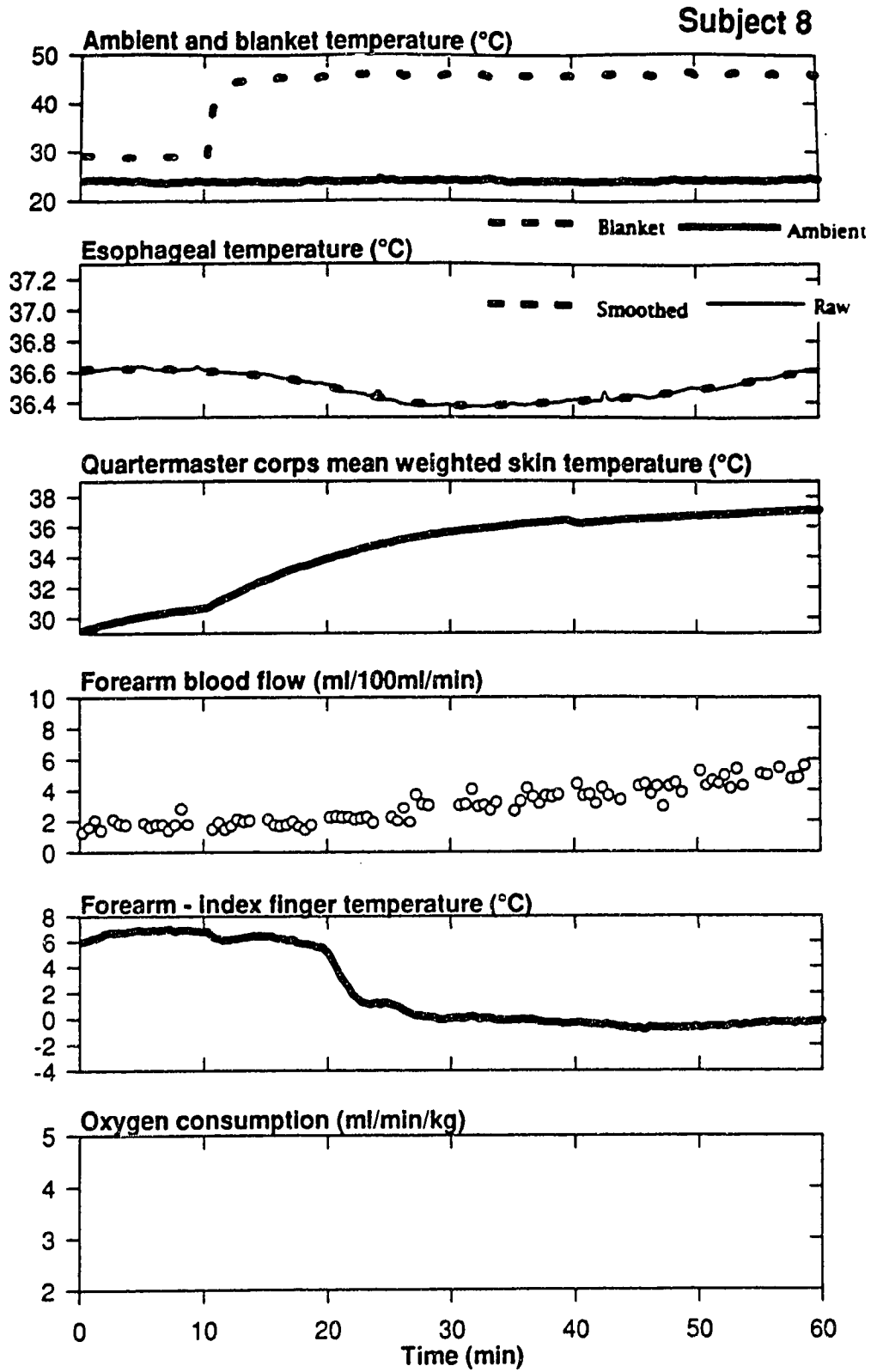


**Subject 7**

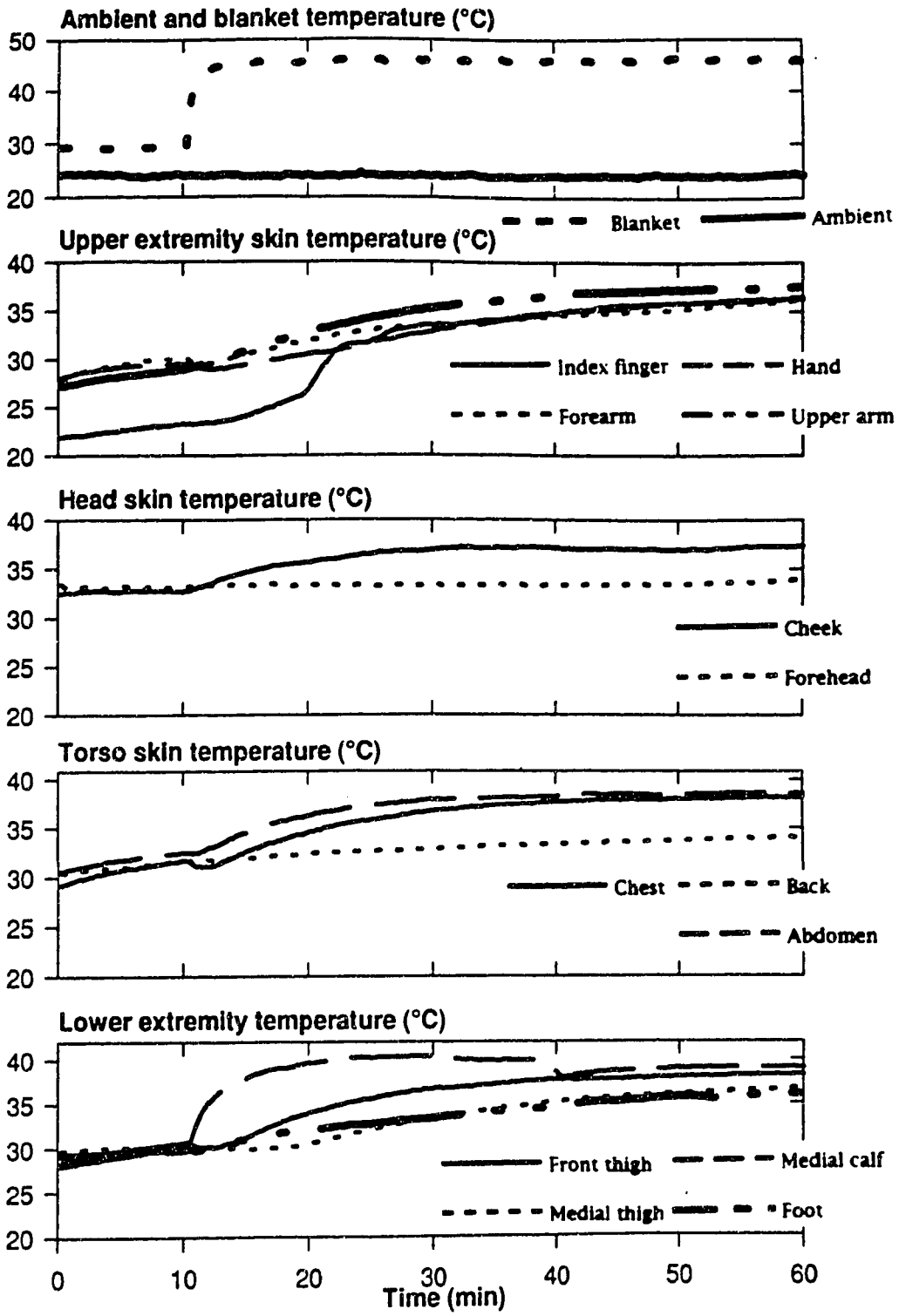


APPENDIX K

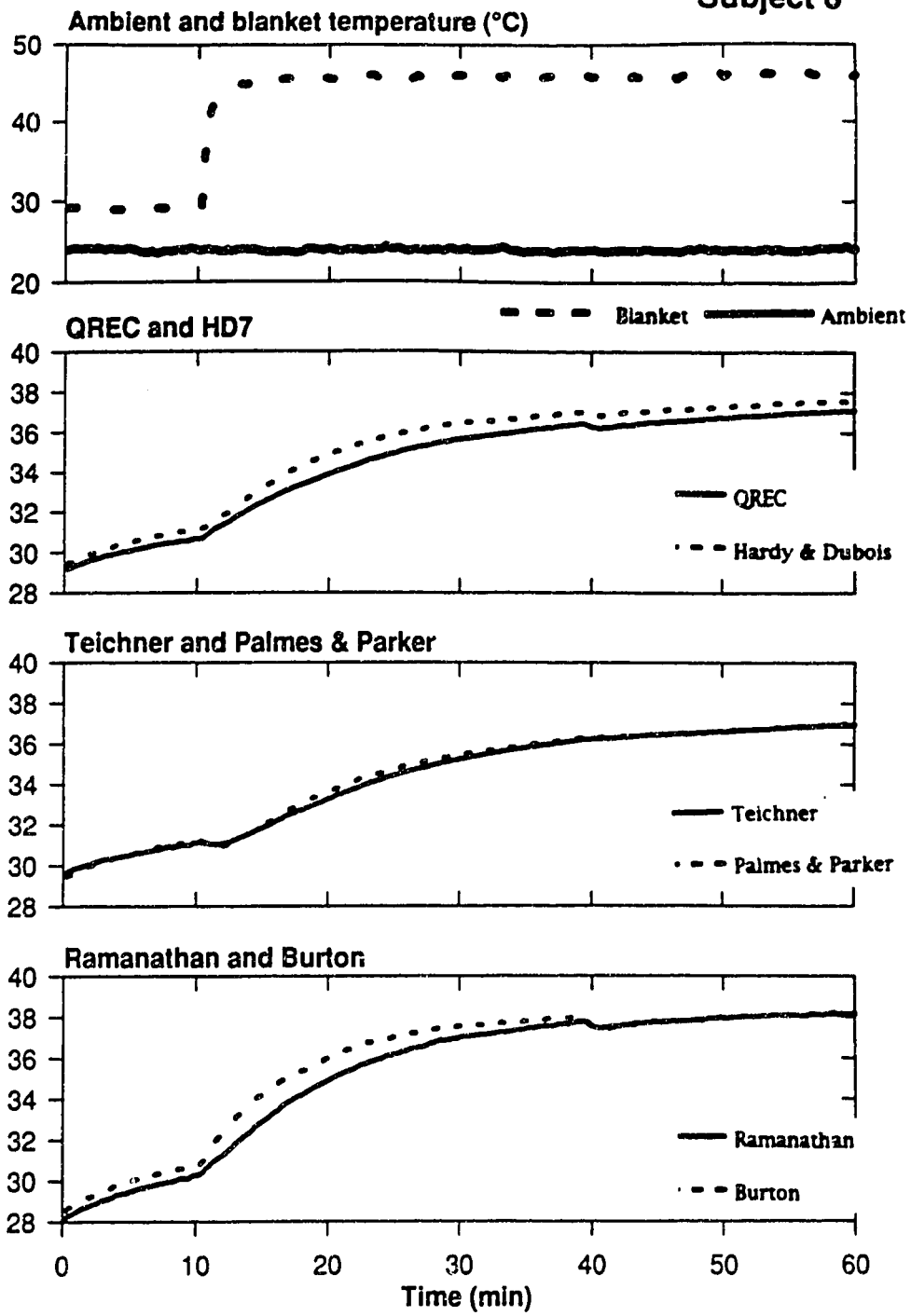
Subject 8's Variable Data



Subject 8



**Subject 8**



## VITA

### Diane L. Audiss' Advanced Degrees

Master of Nursing granted by University of Washington, Seattle,  
Washington, in 1988

Doctor of Philosophy granted by University of Washington, Seattle,  
Washington, in 1994