CENTRAL THERMOSENSITIVITY OF METABOLIC HEAT PRODUCTION DURING COLD WATER IMMERSION

by

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CENTRAL THERMOSENSITIVITY OF METABOLIC HEAT PRODUCTION DURING COLD WATER IMMERSION

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ABSTRACT

The importance of both central and peripheral thermoreception in the determination of metabolic heat production (shivering thermogenesis) during exposure to cold environments has been well established. This knowledge has led to the development of predictive equations for heat production based on the displacement of both skin and core temperatures from their respective threshold or "set-point" values. Although contributions from dynamic peripheral inputs have been elucidated and included in a number of mathematical models, the effect of cooling rate on the central drive to increase heat metabolism has been difficult to evaluate. In addition, predictive equations and thermoregulatory models suggest that for a range of subjects the thermogenic response will be identical, assuming an identical integration of afferent information emanating from the peripheral and central thermoreceptors.

In the present study cold water immersion (15° C) trials were conducted to quantify central thermosensitivity to shivering thermogenesis for a similar combination of core and peripheral thermal inputs for all subjects. Skin temperature was clamped close to water bath temperature while core temperature, measured in the esophagus (T_{es}), was altered following pressure cuff occlusion of extremity blood flow. Occlusion was introduced for a ten minute period during which the temperature of the trapped extremity blood volume and surrounding tissues cooled toward the temperature of the external water medium. Upon deflation of the cuff pressure, the cooled blood was recirculated to the core region and a rapid drop in T_{es} occurred. Concomitant with the decline in core temperature was an increased thermogenic response. Although core temperature was measured at both rectal and esophageal sites, results indicated the temperature in the esophagus, representative of cardiac temperature, was affected by the recirculating cooled blood to a greater extent than rectal temperature ($p < 0.05$). Utilizing the occlusion technique, several hypotheses were investigated.

The thermogenic response to a similar peripheral thermal input and absolute core temperature input was compared during natural cooling (slow and variable cooling rate) with the

response obtained when cooling rate was enhanced by the sudden return to the core of cooled extremity blood. The relationship (β) between metabolic heat production (H) and T_{es}, used to evaluate central thermosensitivity, was significantly greater ($p < 0.05$) when core cooling rate was increased. Inter-subject differences in the thermogenic response to similar peripheral and central thermal stimuli were evident during both immersions.

Evaluation of metabolic heat production induced by a similar thermal drive enabled the assessment of the contribution of morphology and aerobic fitness to the variability observed in central thermosensitivity of young, healthy males. Results indicated that when subjects ($n = 13$) of diverse morphologies and fitness levels experienced a similar peripheral and central thermal stimulus, the resulting thermogenic response was independent of these factors. Inclusion of somatotype and tissue compartments (muscle and adipose) in the assessments did not improve the relationship between central thermosensitivity and morphology.

The effect of short-term adaptation on central thermosensitivity following cold water immersion on five consecutive days was studied in five subjects. All subjects exhibited a reduction in heat production prior to pressure cuff occlusion of blood flow during repeated immersions. The thermogenic response following the occlusion was extremely labile over the 5 days, however a significant reduction ($p < 0.05$) in β was observed in the 4 subjects who completed all 5 immersions. Aerobic fitness and morphological characteristics of the subjects did not appear to influence the variability observed in central thermosensitivity after the short-term adaptation. The ventilatory and oxygen uptake responses at the onset of immersion were significantly reduced during the final immersion when compared with the initial immersion ($p < 0.05$). This suggests short-term adaptation may occur in the integrated thermoafferent drive from peripheral receptors.

ACKNOWLEDGMENTS

The completion of this thesis would not have been possible without the guidance and support of many people.

I am deeply indebted to my supervisor, Dr. Igor Mekjavić, whose continual support, enthusiasm and friendship kept me from joining the ranks of the A.B.D.'s. Special thanks are offerred to my committee members, Doctors Eric Banister, Bill Ross, and John Hayward, for their recommendations and editorial comments as well as providing valuable input during Dr. Mekjavić's absence. I am also extremely grateful for the priviledge of meeting and working with Dr. John Bligh whose wisdom and distinguished character exemplifies a role model which I will strive to emulate.

Collection of data was expedited through the assistance of Wendy Burke, Nickos Geladas, Gavril Morariu, Vic Stobbs, Pat Sullivan, and Matt White. I thank you all for your help and friendship. I am also grateful for the computer programming provided by Naoshi Kakitsuba during his post-doctoral tenure and for the technical support of Henry Michna.

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My heartfelt thanks to my supportive family for never doubting my abilities, and to Neil whose patience and love helped make my dream a reality.

Finally, funding for this research was provided in part from the Natural Sciences and Engineering Research Council of Canada. I am also grateful for the financial support provided by Simon Fraser University throughout my tenure as a graduate student.

DEDICATION

To the 20 subjects who unselfishly gave themselves and their time so that this research could come to fruition

and

To the thousands of A.B.D.'s:

"No retreat, no surrender" —*Bruce Springsteen*

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

During conditions of thermoneutrality in homeotherms,[1] core temperature is regulated within very narrow limits and opposing thermal-correction effectors (e.g. heat production (H or HP), heat loss (HL)) are in equilibrium (Benzinger et al., 1963; Cabanac and Massonnet, 1977). These effector mechanisms are responsible for the maintenance of core temperature via the neuronal integration of peripheral and central, warm and cold thermoreceptor activity as shown in Figure 1.1 by the Wyndham-Atkins/Bligh model (Bligh, 1984).

Upon exposure to cold ambient conditions, thermogenesis as a result of shivering is elicited by enhanced activity of the peripheral cold sensors in an attempt to prevent a reduction in core temperature. When the external environment is extreme, as is the case during cold water immersion, core temperature can no longer be maintained and shivering is enhanced by the increased excitation of central cold receptors. As shown in Figure 1.1, the integrated effector response will also involve the cross-inhibition from cold and warm receptors (Benzinger et al., 1963; Bligh, 1984).

Several stimulus-reponse equations have been proposed in an attempt to predict heat production on the basis of skin and core temperatures during cold water immersion (Brown and Brengelmann, 1970; Timbal et al., 1976; Hayward et al., 1977). Mekjavić and Morrison (1984; 1986) demonstrated that the predictive power of these models may be improved with the inclusion of rate constant terms as well as individual-specific threshold coefficients. The latter authors incorporated the Wyndham-Atkins/Bligh neural model (Figure 1.1) into a model for heat production based on thermoreceptor characteristics (Mekjavić and Morrison, 1985), shown in Figure 1.2. Although this model enabled a better prediction of heat production than previous equations

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[1] Definitions of terms, abbreviations and units of measurement used throughout this thesis conform to the guidelines of the International Union of Physiological Sciences (IUPS), proposed by Bligh and Johnson (1973, "Glossary of terms for thermal physiology", J. Appl. Physiol. 35: 941-961) and Bartels et al. (1973, "Glossary on respiration and gas exchange", J. Appl. Physiol. 34: 549-558).

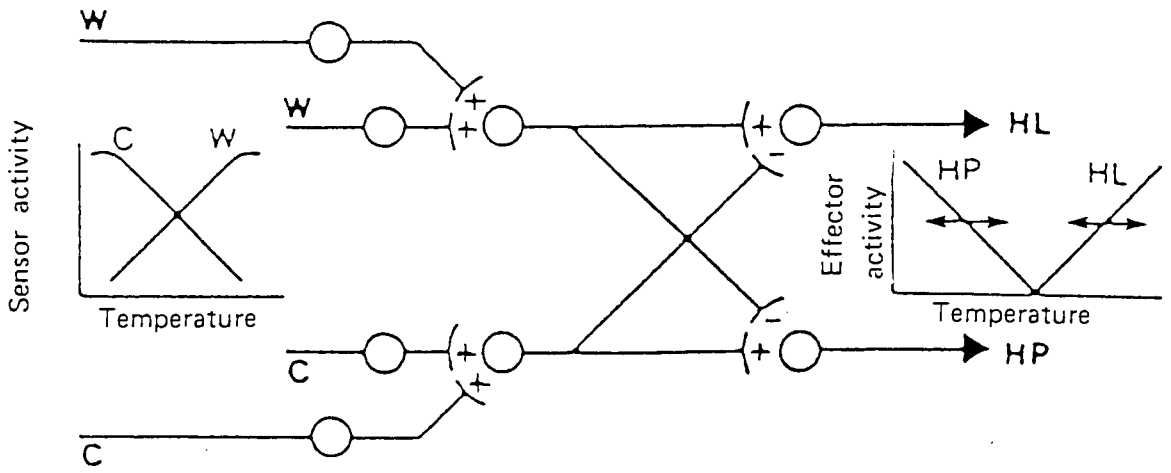

Figure 1.1: The Wyndham and Atkins/Bligh neuronal model of temperature regulation adapted from Bligh (1984). The model suggests the heat production (HP) and heat loss (HL) effector responses (right graph) will be activated as a result of the integration of firing activities of the peripheral and central warm (w) and cold (c) sensors (left graph) as temperature is altered. The integrated effector response is modified in the hypothalamus via crossing inhibitory (−) influences based on the Sherringtonian principle of cross-inhibition.

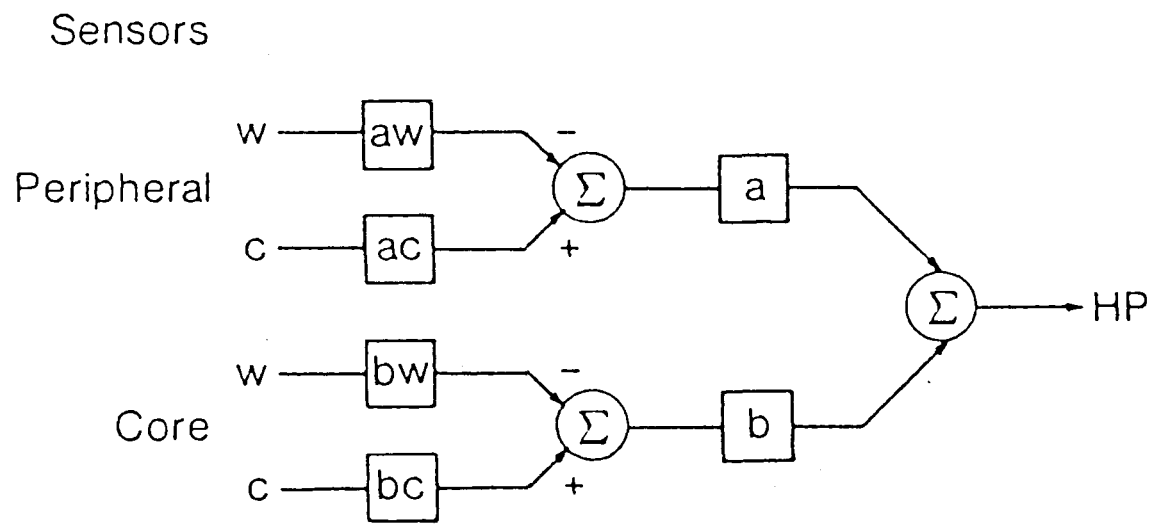

Figure 1.2: Proposed model for predicting metabolic heat production (HP) based on the concepts of Bligh (1984) and Mekjavić and Morrison (1985). Overall peripheral (a) and central (b) thermal drives to heat production are based on integrated excitatory (+) and inhibitory (−) information emanating from warm (w) and cold (c) receptors in the peripheral (aw, ac) and core (bw, bc) regions, respectively. The observed HP is a result of the incorporation of both "a" and "b" thermal drives.

(Mekjavić and Morrison, 1986), it did not address the problem of individual variability in thermogenic responses to similar thermoafferent information. This variability may be a result of individual-specific gain of the peripheral ("a") and central ("b") thermal drive (Figure 1.2) to metabolic heat production during cold water immersion (Mekjavić et al., 1986). Differential thermogenic responses which have been observed for similar thermoafferent information have been attributed to differences in body composition and shape, as well as the degree of adaptation of the individual (Bazett, 1949; Scholander et al., 1958; Hammel, 1964; Brück, 1976; Baum et al., 1976; Hayward and Keatinge, 1981).

Recently, individual differences in respiratory drive ($P_{0.1}$) resulting from cutaneous thermoreceptor activity during the onset of cold water immersion were reported (Mekjavić et al., 1987). This variability, attributed to differences in central integration of thermoafferent stimulation, was shown to be independent of subcutaneous adiposity. Determination of central thermosensitivity during cold water immersion has previously been limited to the investigation of the relationship between metabolic heat production and core temperature during natural body cooling which results in different rates of core cooling among individuals. For individuals who cool at a relatively slow rate, the static discharge of the central receptors will predominate rather than their dynamic response (Hensel, 1982). Thus, the sensitivity of thermoregulatory heat production to similar combinations of peripheral and core thermal drive4 has not been compared between individuals.

Definition of Central Thermosensitivity

The Mekjavić-Morrison model, summarized in Figure 1.2, suggests that neural coded temperature information emanating from warm and cold receptors in peripheral ("aw" and "ac", respectively) and core ("bw" and "bc", respectively) regions are integrated to form a total peripheral ("a") and core ("b") drive to shivering thermogenesis. Both "a" and "b" will depend upon the absolute levels of the peripheral and core temperatures and their respective rates of

change, since both warm and cold, cutaneous and central receptors exhibit static and dynamic response characteristics (Hensel, 1970; Hensel, 1982). The mode of integration of "aw" and "ac" for the total peripheral drive "a" and of "bw" and "bc" for the total central drive "b" incorporates the Sherringtonian principles of cross-inhibition demonstrated in Figure 1.1 (Bligh, 1984).

Metabolic heat production, H ($W \cdot kg^{-1}$), during a given environmental stress is thus a result of the integration of peripheral "a" and central "b" drives. The degree of modulation/integration occurring between the onset of stimulation of thermoreceptors and the observation of effector response, resulting from "a" and "b", will depend upon both individual thermosensitivity and responsiveness of the thermoregulatory control system to a given drive from peripheral and core receptors. Thus determination of the thermosensitivity of H to a given "a" and "b" would involve a complex array of experiments designed to elucidate the effect of various combinations of skin temperature (T_{sk}), rate of change of skin temperature ($\dot{T}_{sk}$), core temperature (T_c), and the rate of change of core temperature ($\dot{T}_c$) upon this parameter.

In the present thesis, the complexity of the above task was simplified by examining central thermosensitivity of shivering thermogenesis, the latter which was determined from oxygen uptake ($\dot{V}O_2$, $L \cdot min^{-1}$) for a limited number of combinations of T_c and $\dot{T}_c$ while T_{sk} was held constant and $\dot{T}_{sk}$ could be neglected. The thermosensitivity of H to a central drive, "b" in Figure 1.2, would encompass the entire physiological range of thermoreceptor combinations. Throughout this thesis however, thermosensitivity of shivering thermogenesis for a specific combination of T_{sk}, $\dot{T}_{sk}$, T_c, and $\dot{T}_c$ is denoted by the Greek letter, β, representing the slope of the relationship between H and T_c at a constant T_{sk}. It is assumed that thermosensitivity determined at a given combination of core and skin temperature displacements (static and dynamic) in any individual will be indicative of his/her overall thermosensitivity. As the definition of central drive to shivering thermogenesis or central thermosensitivity (β) are not standard nomenclature, clarification of their usage pertaining to the present thesis is necessary, thus:

1. Central drive for shivering thermogenesis is a result of integration of thermoafferent information emanating from the body core for a particular combination of decreasing body temperatures (T_{sk}, $\dot{T}_{sk}$, T_c, $\dot{T}_c$). Neural coded information from thermal stimuli is integrated then transmitted to the effector mechanisms resulting in an elevation in metabolic heat production (H).
2. Central thermosensitivity of shivering thermogenesis is determined from the relationship between the effector response (H) and stimulus (T_c) at a specific skin temperature. In the present thesis, central thermosensitivity, β ($W \cdot kg^{-1} \cdot {}^{\circ}C^{-1}$), is assessed as the relationship between the increase in metabolic heat production, H, ($W \cdot kg^{-1}$) and the decrease in core temperature, measured in the esophagus (T_{es}), as a result of a central perturbation with skin temperature clamped ($\dot{T}_{sk} = 0$).

Hypotheses

In the present thesis, several research questions pertaining to central thermosensitivity of metabolic heat production during cold water immersion were investigated:

1. **Chapter II**: Does central thermosensitivity of metabolic heat production for a specific combination of skin and core temperatures differ between healthy, active males? Will an increased rate of core cooling, resulting from the cooled venous return from the limbs following occlusion of extremity blood, alter central thermosensitivity of metabolic heat production?

2. **Chapter III**: Is central thermosensitivity of heat production to a given thermal drive a function of morphology thus accounting for differences in thermogenic responses observed during cold water immersion? Will an individual's somatotype and relevant tissue volumes (muscle and adipose), in addition to classical physique (surface area to mass ratio) and body composition (skinfold thickness), be related to central thermosensitivity of heat production as

they include anthropometric determinants of heat production as well as heat loss? Does aerobic fitness as determined from maximal oxygen consumption influence individual differences in central thermosensitivity?

3. **Chapter IV**: Does an individual's central thermosensitivity of heat production to a core thermal stimulus alter following immersion in cold water on five consecutive days? Are differences in short-term adaptation influenced by morphological characteristics, as morphology itself may provide a form of central adaptation? Will aerobic fitness alter short-term adaptation of central thermosensitivity of metabolic heat production?

The remaining chapters do not address central thermosensitivity *per se* but investigate thermoregulatory responses imposed by the experimental procedures. The following hypotheses were investigated:

1. **Chapter V**: Is the elevation in ventilation and oxygen uptake that is elicited by the sudden immersion in cold water modulated by the short-term adaptation obtained during repeated immersions on five consecutive days?

2. **Chapter VI**: Is the alteration in core temperature, resulting from the venous return following occlusion of blood within extremities during cold water immerison, differentially reflected in esophageal and rectal temperature measurements?

Preliminary results from both these chapters have been reported (Mittleman and Mekjavić, 1987a; Mittleman and Mekjavić, 1987b).

1. **Chapter VII**: Summary and recommendations based on the thesis results.

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CHAPTER II

DETERMINATION OF CENTRAL THERMOSENSITIVITY OF SHIVERING THERMOGENESIS DURING COLD WATER IMMERSION

Introduction

In homeotherms exposed to a cold environment, metabolic heat production is a major effector response elicited by peripheral and core thermoreceptor stimulation (Lim, 1960; Fusco et al., 1961; Benzinger et al., 1963; Craig and Dvorak, 1966; Benzinger, 1970; Brown and Brengelmann, 1970; Nadel et al., 1970). The thermogenic response in man has been characterized by: the critical core temperature (also referred to as the lower critical temperature, LCT) at which the rate of heat production is elevated above resting levels; the relative increase in thermogenesis with decreasing core temperature; and the enhancement of shivering thermogenesis with decreasing skin temperature (Benzinger et al., 1963; Craig and Dvorak, 1966; Stolwijk and Hardy, 1966; Benzinger, 1970; Brown and Brengelmann, 1970; Nadel et al., 1970; Hayward et al., 1975, 1977; Timbal et al., 1976; Cabanac and Massonnet, 1977).

The relationship between thermogenesis and core temperature for a range of skin temperatures is often represented by the classical diagram introduced by Benzinger et al. (1963) and shown in Figure 2.1. Protocols used to derive such relationships clamp skin temperature at a constant level and observe metabolic heat production as core temperature (measured in the rectum, esophagus or aural canal) is varied, with no attempt to standardize the rate at which core temperature changes. Discrepancies exist in the literature concerning the nature of the relationship shown in Figure 2.1, i.e. whether it is curvilinear or linear, or the magnitude of the thermogenic response at any given combination of skin and core temperature (Benzinger et al., 1963; Craig and Dvorak, 1966; Benzinger, 1970). Mekjavić (1983) has suggested that these differences may be due in part to the variation in core temperature cooling rate between individuals exposed to similar peripheral and central temperature inputs. However, the difficulty in manipulating core

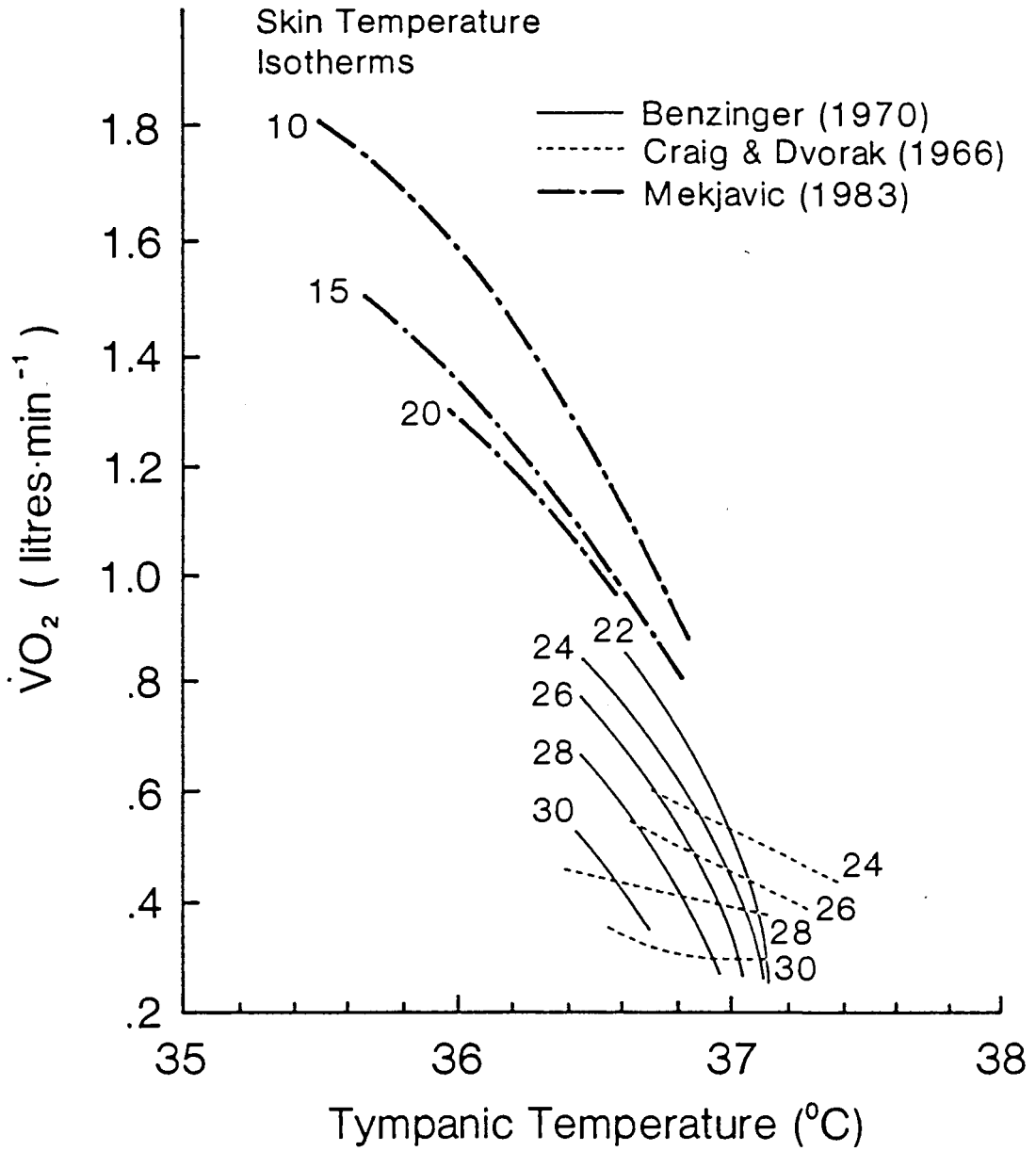

Figure 2.1: The relationship between tympanic temperature (T_{ty}, °C) and oxygen uptake ($\dot{V}O_2$, L·min^{-1}) for a range of skin temperatures. Although observations made by Craig and Dvorak (1966) are over a similar range of core and skin temperatures to those of Benzinger (1970), they suggest a linear $\dot{V}O_2 - T_{ty}$ relationship in contrast to the curvilinear relationship proposed by Benzinger. The results of Mekjavić (1983) extend these findings to a lower skin temperature zone and suggest a curvilinear $\dot{V}O_2 - T_{ty}$ relationship. It is suggested that differences in core cooling rate may give rise to discrepancies observed between results of Benzinger and Craig and Dvorak, and should be accounted for when such comparisons are made.

temperature cooling in humans (Roos and Jessen, 1987) has left this question unresolved. Nadel et al. (1970) utilized the ingestion of ice cream to elicit a thermogenic response to local esophageal cooling. Their results suggested a rate of change component of core temperature contributed to response in metabolic heat production. Hong and Nadel (1979) studied the effect of cooled venous blood on the relationship between the thermogenic response and esophageal temperature at the onset of exercise, following skin temperature cooling in room air maintained at 10° C. Although induced shivering thermogenesis in this case was proportional to the decrease in internal temperature, the suppression of shivering by exercise due to an integrated arousal response (Stitt, 1976; Hong and Nadel, 1979), may have influenced the relationship between heat production and core temperature.

In the present study the selective manipulation of esophageal temperature (T_{es}, °C) at a constant skin temperature (T_{sk}, °C) during cold water immersion was produced by the alternate occlusion/release of extremity blood flow using pneumatic pressure cuffs. The transient decrease in T_{es}, resulting from the cooled venous blood returning to the core upon release of cuff pressure, was reflected in an increased thermogenic response (H, $W \cdot kg^{-1}$). Although reactive hyperemia may contribute to the initial post-occlusion responses, its influence in the cold is lessened as will be discussed in further detail below. Thus the slope of the $H - T_{es}$ relationship (β) observed during a control immersion compared with the β resulting from the increased rate of cooling, induced by the occlusion technique, might determine the influence of esophageal temperature cooling rate ($\dot{T}_{es}$, $°C \cdot min^{-1}$) *per se* (T_{es} was similar between conditions) on the thermogenic response during cold water immersion. In addition, evaluation of the thermogenic response at a similar T_{es}, $\dot{T}_{es}$, and constant skin temperature ($\dot{T}_{sk} = 0$) for all subjects should demonstrate individual variability in central thermosensitivity of metabolic heat production.

Methods

Pressure Cuff Occlusion of Blood Flow

Cold water immersion has been used to isolate the contribution of both skin and core temperature input to thermoregulatory heat production (Benzinger et al., 1963; Craig and Dvorak, 1966; Brown and Brengelmann, 1970; Hayward et al., 1975, 1977; Mekjavić and Morrison, 1985). Although skin temperature may be modulated with relative ease by altering water temperature, the precise manipulation of core temperature at a constant skin temperature has been difficult. A decrease in rectal temperature accompanied by an increase in shivering was observed in a subject upon release of tourniquets applied to the legs which were immersed in 12° C water (Glaser and Holmes Jones, 1951). These researchers concluded that shivering depended on the cooling of central receptors as a result of the increased venous return from muscles of a cooled limb. More recently, Knudsen (1985) observed a significant elevation in oxygen consumption when core temperature was reduced in cold water (17° C) following the release of pressure cuff occlusion of extremity blood flow in both arms and legs. A control immersion in 35° C water was also conducted to verify the rise in oxygen consumption and carbon dioxide production following release of cuff pressure was due to heat production and not to build up of metabolites during the occlusion period.

In the present study an occlusion cuff procedure similar to that described by Knudsen (1985) was utilized in an attempt to standardize the rate of core temperature cooling at a constant skin temperature. The inflation and deflation of the four cuffs, positioned at the proximal one-third of each upper arm and thigh, was controlled by a solenoid valve (Figure 2.2). Upon activation of the valve, the arm and thigh cuffs were inflated by a high pressure air source to 170 mmHg and 240 mmHg, respectively. The pressure in each cuff was regulated manually by properly adjusting a relief valve. Throughout the ten-minute occlusion period, cuff pressures were continuously monitored by pressure gauges connected to each cuff and mounted outside the immersion tank. Inflation of the cuff traps blood within the limb. As a result of the diminished

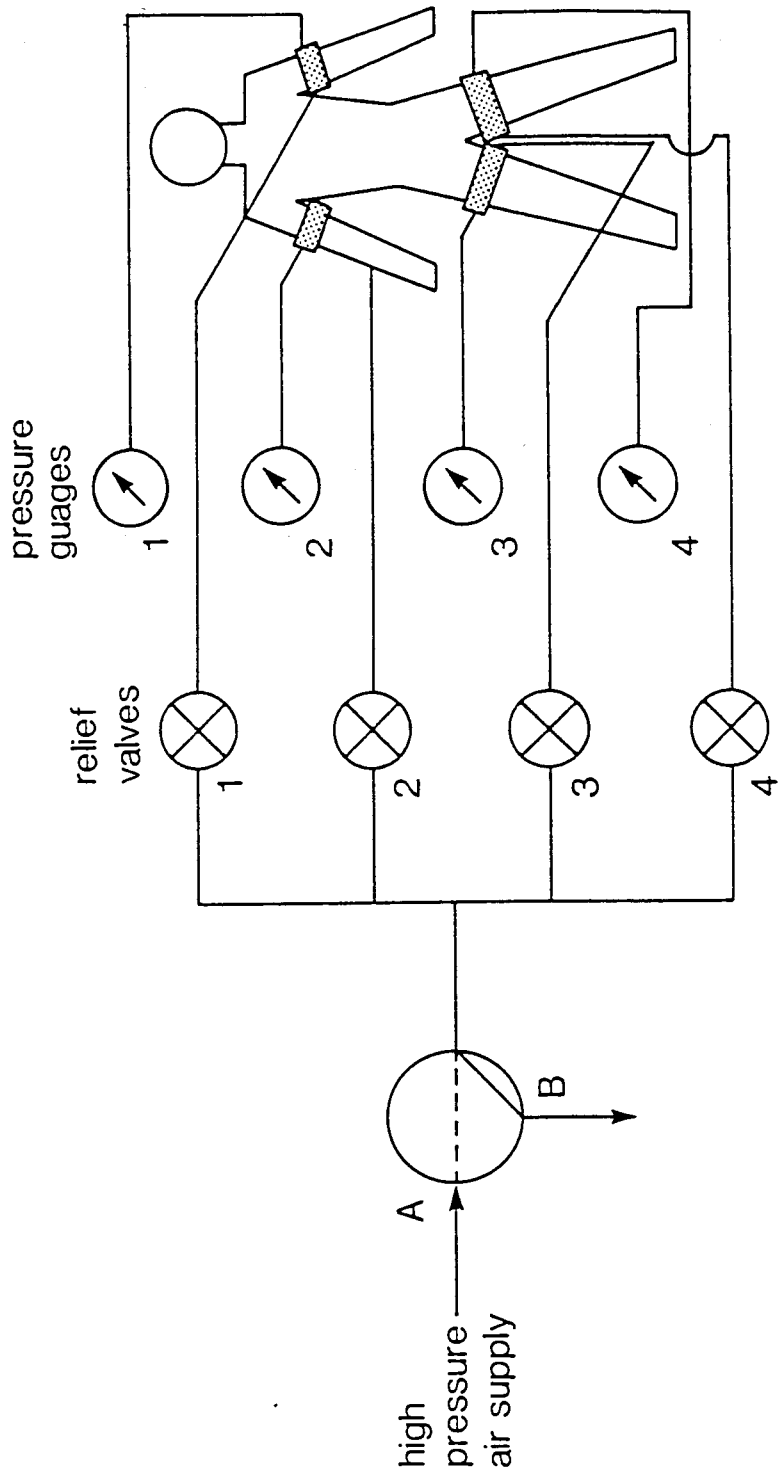

Figure 2.2: The arrangement for the pressure cuff occlusion of extremity blood flow. Pressure cuffs, positioned at the proximal one-third of each extremity (shaded area), were inflated from a constant high pressure air supply (A) which was controlled by a solenoid valve (B). The pressure within the cuffs was monitored visually with a series of pressure gauges and adjusted appropriately (see text) with relief valves.

delivery of warm arterial blood as well as the ongoing reduction in temperature of the limb tissues immersed in the cold water (Coles and Cooper, 1959), the venous blood draining the occluded limb will cool. Upon release of cuff pressure the cooled venous blood is returned to the core region instigating a transient decrease in core temperature.

Subjects

Seven male subjects consented to participate in this study after undergoing medical evaluation and being informed of the procedures and potential risks. All experimental procedures were approved by the SFU Ethics Committee. Skinfold thicknesses were measured with a Harpenden Caliper (John Bull, England) at six sites (triceps, subscapular, suprailiac, abdominal, front thigh, and medial calf) following the guidelines established by the International Working Group on Kinanthropometry (Ross and Marfell-Jones, 1983). Height and weight were also assessed for determination of surface area (A_D) using the formula proposed by DuBois and DuBois (1916). Physical characteristics of the participants are shown in Table 2.1.

Immersion Protocol

Each subject participated in two water immersion trials commencing at the same time of day but spaced at least one week apart in order to eliminate circadian rhythm and acclimation effects, respectively. Both trials consisted of immersion in cold water (15.82 ± 1.03° C; Mean ± SD for all 14 trials), with one trial serving as a control immersion (I_{con}). In the second trial (I_{exp}) extremity blood flow was occluded during the immersion for a ten-minute period and then released using the cuff occlusion method described previously. The thermogenic response as a result of the decrease in core temperature following the recirculation of the cooled blood was used to evaluate central thermosensitivity. The order of the trials was counterbalanced so that four subjects underwent I_{con} first, and three underwent I_{exp} first. Data collected from one subject who underwent I_{exp} first were not included due to equipment malfunction.

Table 2.1: Physical characteristics of subjects

Subject No.	Age (yr)	Height (cm)	Weight (kg)	A_D (m^2)	Σ 6 Skinfolds (mm)
1	33.0	177.0	72.5	1.89	50.5
2	35.6	170.1	76.8	1.88	97.0
3	24.1	186.0	81.9	2.06	40.5
4	26.8	176.9	69.9	1.86	31.8
5	26.0	164.3	62.7	1.68	69.7
6	25.0	189.2	79.2	2.06	52.6
7	27.5	171.3	74.2	1.87	114.5
Mean	28.3	176.4	73.9	1.90	65.2
S.D.	4.3	8.8	6.4	.13	30.4

Prior to each immersion, subjects were instructed to avoid physical exertion and report to the laboratory at least four hours postprandial. Following insertion of the core temperature probes, subjects relaxed in a thermoneutral room (26.05 ± 1.40° C) while the remaining transducers were positioned. After 30 minutes subjects were assisted into a mesh chair suspended from the ceiling above the immersion tank where pre-immersion recordings were obtained for 5 minutes. At the end of the baseline period subjects were immediately lowered into the tank until the water level reached the sternal notch.

During the I_{con}, subjects remained in the tank for one hour or until T_{es} reached 35° C. During the I_{exp}, blood flow to the extremities was occluded after skin temperature stabilized, approximately 5-10 minutes into the immersion. An attempt was made to initiate the occlusion at a similar core temperature for all subjects, thus cuffs were inflated at a T_{es} of 36.5° C, whenever possible. In those subjects who maintained their T_{es} above 36.5° C, occlusion was introduced at minute 40 of the immersion. Following a ten minute occlusion period, the cuffs were deflated and the subjects remained immersed in the bath until T_{es} reached 35° C, or upon completion of one hour of immersion. Immediately post-immersion for both conditions, the subject was removed from the tank and placed in a well-insulated bed for rewarming via endogenous heat production. The subject was allowed to voluntarily terminate the experiment at any time.

In order to assess the effect of occlusion on the enhanced rate of heat production following the release of cuff pressure, a thermoneutral immersion (35° C) was conducted on 7 male subjects, 3 of whom participated in the present cold water immersions. A similar protocol to that described above was utilized, with the exception that occlusion of extremity blood flow was initiated following the 10th minute of immersion and all trials were completed after 30 minutes of immersion.

Instrumentation

The immersion tank (88 x 86 x 90.5 cm) was constructed from plywood and encased in a steel frame. A custom-made vinyl liner was designed to hold a total volume of .685 m^3 of water. Throughout the immersion water was continously stirred (maximum flow rate = 75 $L \cdot min^{-1}$) by a spa support system (Swimquip, Wicor Canada Ltd., Missassauga, Ont.) and temperature maintained at the desired level by a constant flow portable cooling unit (Blue M Electric Co., Blue Island, Ill.).

Core temperature changes were assessed from both the rectal (T_{re}) and esophageal (T_{es}) sites, utilizing YSI thermistor probes (No. 701 and 702A, Yellow Springs Instrument Co., Yellow Springs, Ohio). The rectal thermistor was inserted 15 cm beyond the anus and the esophageal probe was inserted through the nasal passage to a point 37 cm beyond the external nares, the maximum length of the probe.

Skin temperature and heat flux were evaluated by thermistor gradient heat flux transducers with imbedded thermistors (Model HA 13-18-10P(C), Thermonetics Corp., San Diego, Ca.). Transducers were positioned and secured in place with waterproof tape (Elastoplast, Smith and Nephew, Inc., Lachine, Que.) at the following locations on the subject's right side: 1) Triceps—back of the arm, 5 cm below acromiale (just above occlusion cuff); 2) Chest—5 cm above nipple; 3) Back—2.5 cm lateral to vertebral column between L2 and L4; 4) Thigh—10 cm above patella (just below occlusion cuff); and 5) Calf—lateral portion of leg at level of maximum girth. Mean skin temperature ($\bar{T}_{sk}$) and mean heat flux ($\dot{Q}_{mean}$, $W \cdot m^{-2}$) were calculated from the unweighted average of these sites.

Prior to each trial, ambient temperature (T_a) was recorded from a YSI 701 thermistor located above the immersion tank. Water temperature (T_w) was measured by a similar thermistor placed at the side of the tank at midlevel. Care was taken to ensure the thermistor did not come in contact with the subject or the side of the tank. All temperatures were evaluated to the nearest .01°C. Calibration of thermistors and transducers were conducted according to the

procedures outlined in Appendix A.

Metabolic heat production (H, $W \cdot kg^{-1}$) was calculated from oxygen uptake ($\dot{V}O_2$)[1] which was determined from the analyses of mixed expired gases and inspired minute volumes. Oxygen and carbon dioxide contents of the expired gas were measured with an Applied Electrochemistry S-3A Oxygen Analyzer and a Godart Capnograph, respectively. Gas analyzers were calibrated prior to each immersion with the use of 2 standard gas mixtures and room air.

A pneumotachometer (Hans Rudolph, Kansas City, Mo.) was attached to a pressure transducer (Vacumed, Ventura, Ca.) driven by a carrier oscillator with the subsequent output voltage demodulated and amplified to determine flow rate of the inspired gas. Ventilatory volumes were determined by integrating the inspiratory flow rate using a precision low drift integrator. Volume calibration was performed prior to each immersion by administration of a standard syringe volume (5.92 L). Ventilation ($\dot{V}_E$) and $\dot{V}O_2$ were measured in $L \cdot min^{-1}$, corrected to standard temperature and pressure, dry (STPD). Barometric pressure was recorded from a mercury barometer (Fischer Scientific, Ottawa, Ont.) and relative humidity determined by a sling psychrometer (Taylor Instrument Co., Rochester, N.Y.).

Heart rates (HR, $b \cdot min^{-1}$) were recorded with an electrocardiograph (Physio-Control Systems, Seattle, Wa.) located five feet from the immersion tank. Disposable silver/silver chloride electrodes were placed on the subject's chest in a modified Lead I (CM_5) position and were connected to the ECG by an extended, shielded patient cable. The electrodes and cable were secured in place by waterproof tape.

Skin temperatures, T_{es}, T_w, $\dot{V}O_2$, and heat flux data were collected on-line by a Hewlett-Packard 3497A data acquisition system, controlled by a Hewlett-Packard 9817 minicomputer. Values were recorded every 10 seconds during the first 40 seconds of each minute, and averaged for printout at the end of each minute. Heart rate and T_{re}, measured from a

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[1] Conversion Factor : 1 Liter $O_2 \cdot min^{-1}$ = 352.015 W; based on the energy equivalent of 5 kcal for each liter O_2 consumed.

digital multimeter (Model 5000, Dana Laboratories Inc., Irvine, Ca.), were recorded manually at the end of each minute.

Analysis of Central Thermosensitivity

The return of cooled blood to the core region upon release of cuff pressure results in a rapid nonlinear decrease in T_{es}. This decay in T_{es} is matched with a similar rapid and nonlinear increase in the rate of heat production (H) as determined indirectly by oxygen consumption. The stimulus (T_{es}) - response (H) relationship was assessed over a similar range of T_{es} for the control (no occlusion) trial and during the transient decrease in T_{es} at the initial post-occlusion phase of the experimental trial. By comparing the slope (β) of the $H - T_{es}$ relationship during the control and experimental immersions, contribution of $\dot{T}_{es}$ to the rate of heat production may be assessed. The slope of this relationship was selected over the analysis of the comparison of the absolute changes in T_{es} and H for the determination of β as the latter analysis does not take into account the time course of these variables.

Although both T_{es} and T_{re} were assessed, only the T_{es} response is presented in detail as it has been shown to be representative of cardiac temperature (Cooper and Kenyon, 1957; Wyss et al., 1974; Hayward et al., 1984). Brengelmann (1987), in a recent review of the measurement of body temperature, concluded that esophageal temperature provided the best measurement of either hypothalamic or core temperature.

Statistical Analyses

Differences between parameters measured during the control and experimental trials were evaluated for statistical significance ($p < 0.05$) by multivariate analysis of variance (MANOVA) for repeated measures (Dixon, 1983). A univariate analysis of variance (ANOVA) for repeated measures was used to compare variables in which missing data occurred. Cooling rate and the slope of the $H - T_{es}$ relationship were derived from a least squares linear regression analysis. Mean values and standard deviations are presented for group data unless noted otherwise.

Results

Physiological Responses During Cold Water Immersions

1. Pre-Immersion and Pre-Occlusion

Comparison of the pre-immersion values for T_{es}, T_{re}, H, $\bar{T}_{sk}$, $\dot{Q}_{mean}$, and HR between the control and experimental condition, presented in Table 2.2, revealed no statistical significance ($p > 0.05$). During the immersion, the pattern of change in the physiological variables evaluated in the experimental trial was similar to that observed in the control trial, until the occlusion period. As shown in Figure 2.3, a rapid decrease in $\bar{T}_{sk}$ to a constant level (18.04 ± .97° C) occurred within 5 to 10 minutes of the onset of immersion. A concomitant transient elevation in $\dot{Q}_{mean}$, to a peak value of 879.42 ± 155.04 $W \cdot m^{-2}$, was observed within the first minute of immersion which diminished thereafter, so that 80% of its total decrease was attained during the first 10 minutes of immersion. Oxygen consumption ($\dot{V}O_2$, $L \cdot min^{-1}$ STPD) and ventilation ($\dot{V}_E$, $L \cdot min^{-1}$ STPD) responses exhibited a characteristic elevation during the onset of immersion, which decreased to a level at or slightly above pre-immersion, and finally increased at a slower rate until the initiation of occlusion. The values for $\dot{Q}_{mean}$, T_{es}, T_{re}, H, $\bar{T}_{sk}$, and HR, determined during the period just prior to occlusion in the experimental condition and over a corresponding time period during the control condition, were similar.

Inter-subject differences in the time course of T_{es} were evident during the pre-occlusion phase. The elapsed time from initial immersion until T_{es} began to decline continuously, ranged from 9 to 32 minutes. $\dot{T}_{es}$, determined from the time derivative during the steady decline in T_{es}, varied from -.075 to -.014° $C \cdot min^{-1}$ ($\bar{x}$: -.042 ± .020° $C \cdot min^{-1}$). In those subjects whose T_{es} was maintained near its resting value, the elevation in H was minimal (Figure 2.3, Table 2.2). When $\dot{T}_{es}$ was increased, H was sustained at an elevated level. The increase in H from its resting value (ΔH) ranged from .82 to 5.23 $W \cdot kg^{-1}$ ($\bar{x}$: 2.98 ± 1.73 $W \cdot kg^{-1}$) during the final minutes of the control immersion.

Table 2.2: Pre-immersion and pre-occlusion values for variables measured during experimental and control immersions.

		Experimental						Control					
Subject No.	Exper. Order	T_{es} (°C)	T_{re} (°C)	H ($W \cdot kg^{-1}$)	T_{sk} (°C)	$\dot{Q}$ ($W \cdot m^{-2}$)	HR (min^{-1})	T_{es} (°C)	T_{re} (°C)	H ($W \cdot kg^{-1}$)	T_{sk} (°C)	$\dot{Q}$ ($W \cdot m^{-2}$)	HR (min^{-1})
I. Pre-Immersion													
1	2	36.99	37.31	—	32.31	65.23	70	36.92	37.41	2.91	32.73	58.69	58
2	1	37.05	37.28	2.01	32.92	47.99	76	37.04	37.31	1.88	32.53	55.10	71
3	2	36.84	37.21	2.28	33.64	47.77	69	36.53	36.92	1.14	32.86	51.05	60
4	2	37.32	37.53	2.42	33.69	59.82	72	37.12	37.54	2.28	33.27	77.60	68
5	1	36.60	36.98	1.47	33.21	50.94	72	37.02	37.32	1.66	31.40	48.12	72
6	2	36.83	37.12	2.59	34.38	54.42	79	36.88	—	3.26	33.79	56.54	70
7	1	37.18	37.27	1.95	31.78	54.63	68	36.89	37.00	1.96	32.58	55.73	78
II. Pre-Occlusion													
1	2	36.62	37.15	3.04	16.52	263.92	69	36.68	37.10	5.12	18.88	246.18	71
2	1	36.86	37.15	1.74	16.93	215.26	—	37.00	37.31	1.52	16.99	224.68	55
3	2	36.55	36.68	3.38	18.73	323.92	74	36.14	36.74	3.56	18.50	439.46	74
4	2	36.68	37.26	3.82	19.43	324.88	74	36.65	37.33	3.53	18.80	330.08	70
5	1	36.55	36.77	2.32	19.22	293.73	71	36.93	36.99	1.43	16.73	199.76	64
6	2	36.82	36.73	2.50	18.40	368.24	54	36.53	—	2.98	18.15	321.24	84
7	1	37.50	37.33	2.31	17.73	142.88	55	37.00	—	2.39	17.55	154.40	—

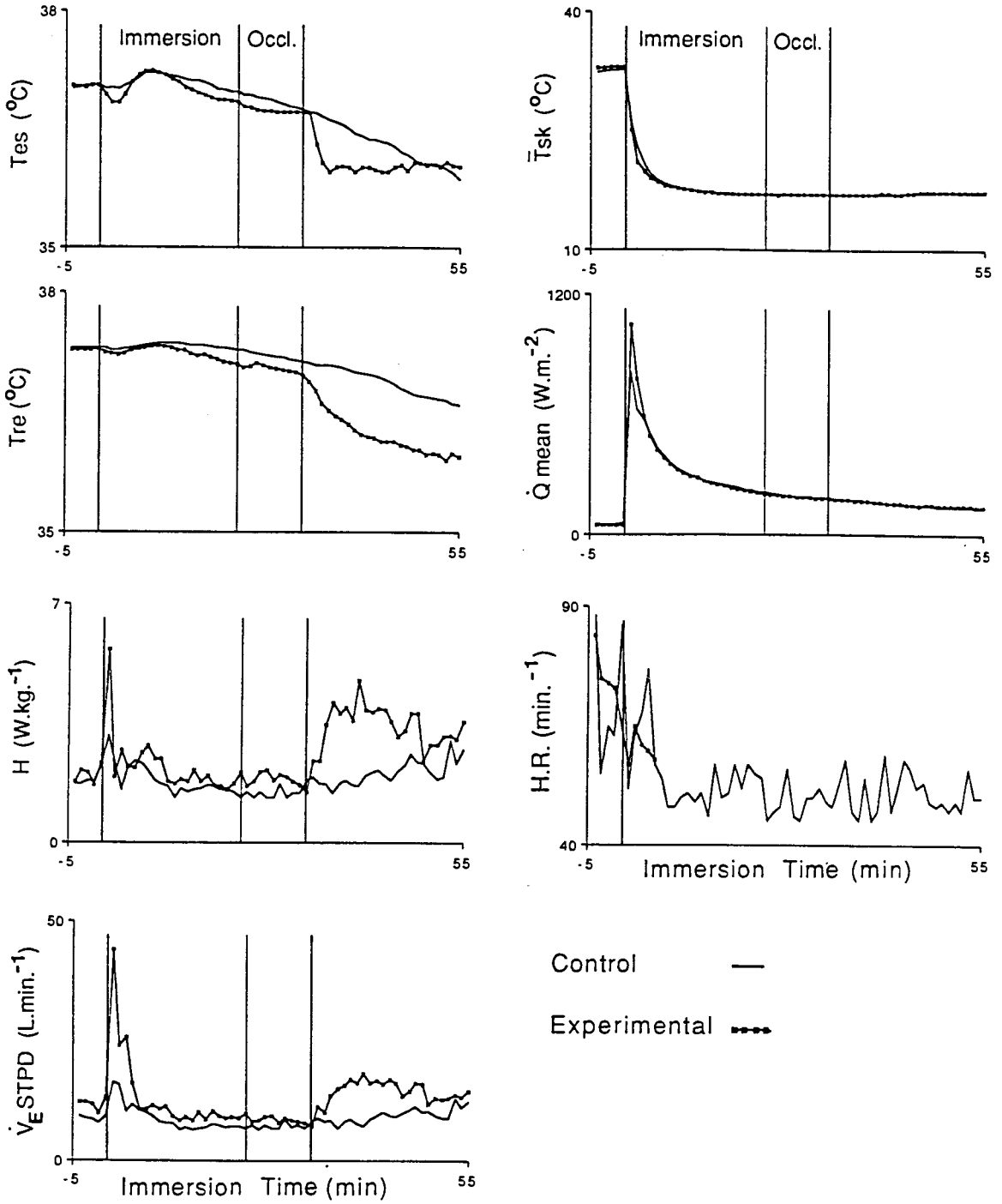

Figure 2.3: Physiological responses of subject 2 during cold water immersions. The figure indicates the similarity in the responses in the control (I_{con}) and experimental (I_{exp}) trials during the pre-occlusion period. Release of the cuff occlusion instigates a rapid decrease in esophageal temperature T_{es} accompanied by an elevation in heat production (H). Following an initial transient phase, at the onset of immersion $\dot{Q}_{mean}$ and $\bar{T}_{sk}$ (calculated from unweighted average of 5 sites) remain fairly stable. Heart rates were not able to be determined following the initial five minutes of the experimental trial for this subject.

2. Occlusion

At the onset of occlusion a transient elevation in H was followed by a reduction to, or below its pre-occlusion level. A concomitant decrease in $\dot{T}_{es}$ from $-.023 \pm .023$ to $-.019 \pm .009^{\circ}C \cdot min^{-1}$ was observed, although the reduction in T_{es} became nonlinear in most subjects. During this phase, shivering was visibly reduced and subjects stated they felt warmer. $\dot{Q}_{mean}$ was minimally altered by the occlusion of limb blood flow (Figure 2.3). Heart rates were also reduced during the occlusion period as shown in Figure 2.4.

3. Post-Occlusion

Upon release of cuff pressure, T_{es} decreased rapidly to a constant level $.76 \pm .17^{\circ}C$ below its occlusion value. As seen in Table 2.3 the initial linear rate of cooling of esophageal temperature ($\dot{T}_{es}$, $^{\circ}C \cdot min^{-1}$) was similar for all subjects, with the exception of subject 3. Although $\dot{T}_{es}$ was similar, the magnitude of decrease in T_{es} from onset of cuff release to the achievement of a stable value varied twofold among subjects (range : .53 to $1.05^{\circ}C$). T_{es} generally stabilized within 5 minutes. The decline in T_{es} for subject 7 was immediately reversed without stabilizing.

Accompanying the decrease in T_{es} was a parallel rise in H. A constant level, 3.15 ± 1.09 $W \cdot kg^{-1}$ above its occlusion value (Table 2.3), was achieved within 5 minutes with the exception of subject 6 whose H continued to rise until the 8th minute following release of cuff pressure. The elevated metabolic heat production was maintained for the remainder of immersion which ranged from 10 to 35 minutes. A visible increase in shivering was also noted during this phase. HR during post-occlusion was transiently elevated.

Effect of $\dot{T}_{es}$ on the $H - T_{es}$ Relationship

In Figure 2.5 the values for β observed during the control immersion (reduced $\dot{T}_{es}$) and during the post-occlusion phase of experimental trial (increased $\dot{T}_{es}$) are shown. Within each subject, the $H - T_{es}$ relationship was determined over a similar temperature range for both

Table 2.3: Experimental and control values for core temperature and thermogenic response during post-occlusion phase

Subject No.	Initial T_{es} (°C)		Final T_{es} (°C)		$\dot{T}_{es}$ (°C·min^{-1})		ΔH (W.kg-1)		ß (W·kg^{-1}·°C^{-1})	
	Exp.	Con.	Exp.	Con.	Exp.	Con.	Exp.	Con.	Exp.	Con.
1	36.26	36.47	35.21	35.51	-.261	-.052	3.69	2.02	-2.255	-1.955
2	36.70	36.71	35.95	35.95	-.243	-.035	2.30	1.11	-2.205	-1.026
3	36.36	36.21	35.70	35.54	-.141	-.049	4.70	1.91	-4.375	-2.273
4	36.51	36.50	35.64	35.59	-.238	-.072	4.01	1.78	-2.448	-1.751
5	36.14	36.06	35.55	35.50	-.224	-.061	1.76	1.31	-2.402	-1.556
6	36.77	36.77	35.92	35.95	-.260	-.064	3.45	2.17	-3.308	-3.017
7	37.26	37.25	36.63	36.67	-.260	-.011	2.17	1.18	-2.236	-1.787
Mean	36.57	36.57	35.80	35.82	-.232*	-.049	3.15*	1.64	-2.747*	-1.909
S.D.	.38	.39	.44	.42	.043	.021	1.09	0.43	0.813	0.620

* Significantly greater than control value ($p < .05$).

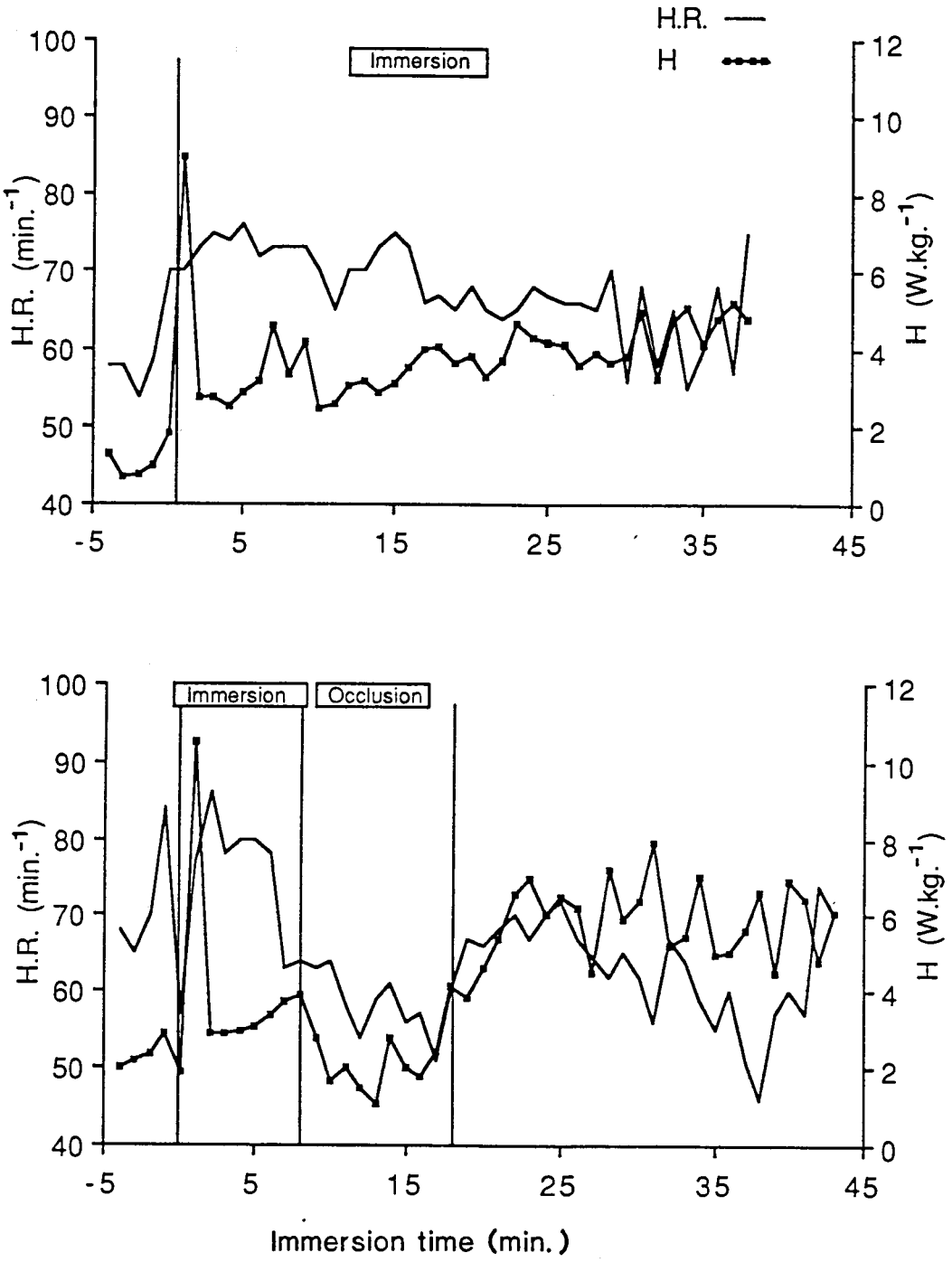

Figure 2.4: The rate of metabolic heat production (H, $W \cdot kg^{-1}$) and heart rate (HR, min^{-1}) during control (top) and experimental (bottom) trials for subject 3. Both HR and H were reduced during the occlusion phase. Following release of cuff pressure HR transiently increased while the elevation in H was sustained.

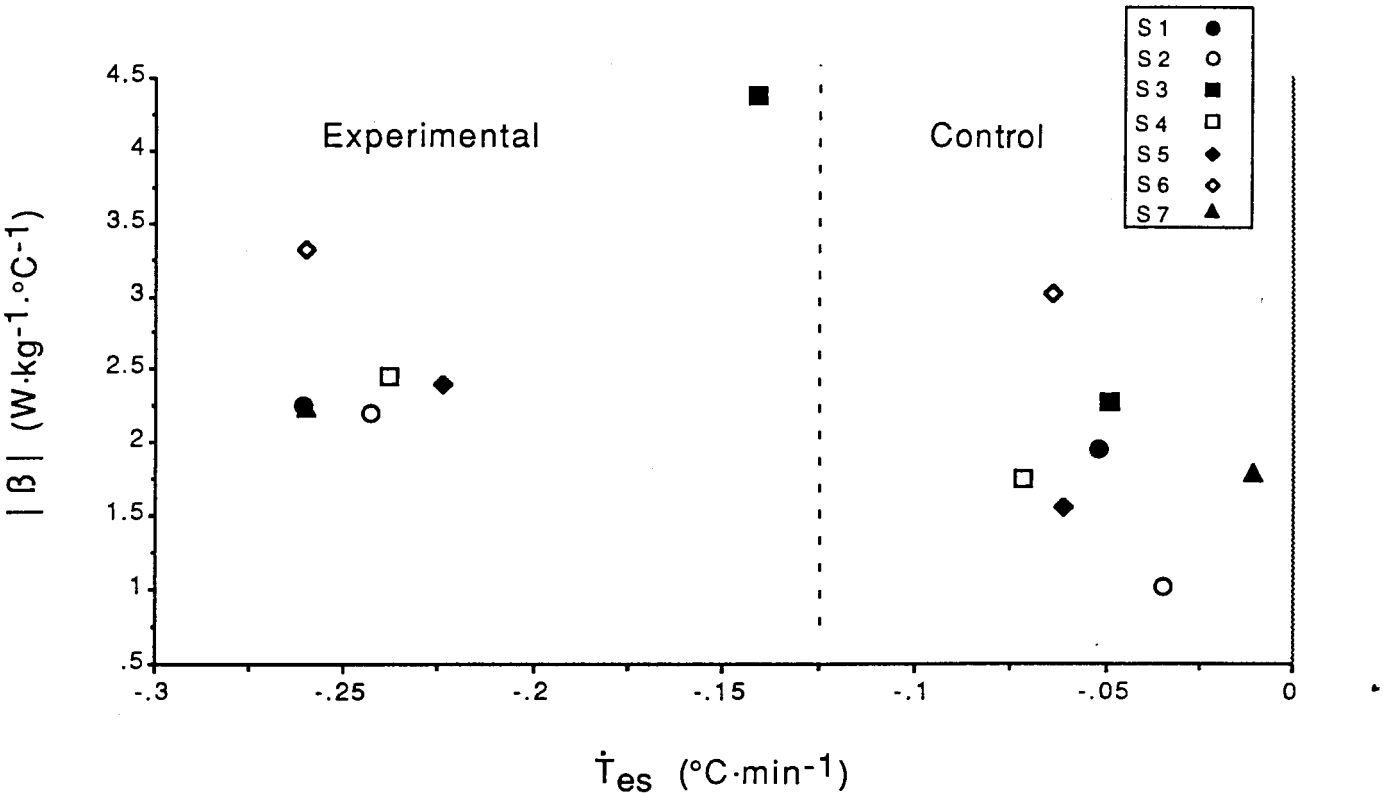

Figure 2.5: Differences in the slope of H – T_{es} relationship (β) during control immersion (reduced $\dot{T}_{es}$) and immediately following the release of occlusion during experimental trial (increased $\dot{T}_{es}$). Symbols for each of the 7 subjects are shown in the legend. All subjects exhibited an elevated β with an enhanced $\dot{T}_{es}$.

immersions. As evident from Table 2.3, $\dot{T}_{es}$ was significantly higher in the experimental compared with the control trial which suggests the increased negative slope (β) of the H – T_{es} relationship observed during the experimental trial (Figure 2.5, Table 2.3) is a result of the increase in $\dot{T}_{es}$. Although the cooling rate for subject 3 during the post-occlusion phase was lowest among the group, his central drive to shivering thermogenesis increased 92.5% above that evaluated during the control immersion. In contrast, the increase in β observed for subject 6 during the experimental trial was only 9.6% greater than the control trial value, while his cooling rate following the occlusion was among the highest.

Physiological Responses During Thermoneutral Immersion

The group mean time course of H during the thermoneutral (35° C) water immersion is shown in Figure 2.6. The peak increase in H immediately following the release of cuff pressure was 1.54 ± .59 $W \cdot kg^{-1}$ and occurred during the second minute of the post-occlusion phase for all subjects. The peak ΔH post-occlusion was similar to the ΔH observed during the initial minute of immersion (1.23 ± .51 $W \cdot kg^{-1}$).

Discussion

The experimental manipulation of core temperature by pressure cuff occlusion and subsequent release of cooled blood trapped in the extremities, resulted in an increased shivering thermogenesis. Similar results have been reported after pressure cuff occlusion of limbs immersed in cold water (Glaser and Holmes Jones, 1951; Knudsen, 1985). The observed increase in esophageal temperature cooling rate is a result of the return to the core region of the trapped extremity blood, the flow of which may be enhanced by reactive hyperemia associated with the occlusion of blood flow (Lewis and Grant, 1925; Abramson et al., 1941, 1958, 1961; Coles and Cooper, 1959). Mechanisms of reactive hyperemia proposed include a reduction in intramural pressure in the arterial vessels distal to the site of occlusion (Folkow, 1949; Patterson, 1956), an

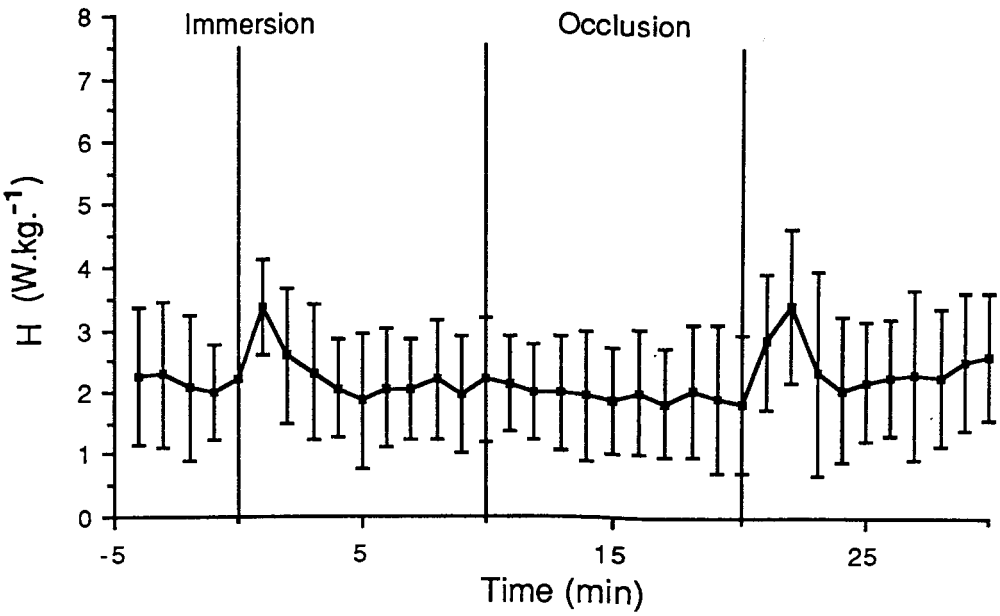

Figure 2.6: Group mean heat production (H) response during immersion in thermoneutral water for 7 subjects. Peak values following release of cuff occlusion are similar to those observed during the first minute of immersion.

increase in vasodilator metabolites (Lewis and Grant, 1925; Coles and Cooper, 1959) and local tissue anoxia (Crawford et al., 1959). Concurrent with the reactive hyperemia following arterial occlusion under normal ambient conditions, a transient increase in oxygen uptake (associated with the repayment of oxygen debt) has been reported (McNeill, 1956; Abramson et al., 1958, 1961; Coles and Cooper, 1959). However, several factors suggest that this increased tissue metabolism may play a minor role in the enhancement of metabolic heat production observed following the release of occlusion in the present study. First, lowering the tissue temperature of a resting human limb prior to arterial occlusion has been shown to reduce the duration and magnitude of reactive hyperemia (Lewis and Grant, 1925; Freeman, 1935; Abramson et al., 1941, 1958; Coles and Cooper, 1959) as well as decrease the average excess oxygen uptake both during and following the period of occlusion (Abramson et al., 1958). Although shivering activity was observed in most subjects prior to the occlusion phase of the immersion, a marked reduction in shivering was evidenced with the onset of extremity blood flow occlusion, thereby reducing the oxygen deficit incurred. Abramson et al. (1958) also reported that lower tissue temperatures resulted in a less effective removal of oxygen from the blood. More recently, Blomstrand et al. (1986) suggested that delivered oxygen is less effectively utilized at subnormal than at normal tissue temperature during intensive dynamic exercise. Secondly, when extremity blood flow was occluded during immersion in thermoneutral water (35° C), the rate of heat production upon deflation of the cuffs transiently increased to a level similar to that observed during the initial minute of immersion. This suggests that the increase in central blood volume observed immediately upon immerison (Arborelius et al., 1972) may also be the major contributing factor in the heat production response following release of occluded limbs in thermoneutral water. Thus the enhanced post-occlusion thermogenic response during the cold water immersion may contain both a thermoregulatory and hemodynamic component. Knudsen (1985) conducted a thermoneutral immersion in addition to immersion in 17° C water in male divers and suggested the increased total oxygen consumption following release of occluded limb blood during cold water immersion was not due to build up of metabolites during the occlusion period.

The reduction of shivering during the occlusion period may be a result of a number of influences. Several subjects reported feeling pain at the onset of inflation of the pressure cuff. The stimulation of nociceptors may result in an integrated arousal response which has been shown to inhibit shivering in both rabbits (Stitt, 1976) and humans (Hong and Nadel, 1979). Alternatively the occlusion may have blocked the afferent activity of the thermoreceptors as has been reported by Fruhstorfer et al. (1974) following injection of a local anaesthetic. However, Marsden et al. (1977) observed that complete ischemic block of muscle spindle afferents does not occur until about 1 hour following pressure cuff occlusion. In the present study inflation pressures were not great enough to completely occlude arterial blood flow which further suggests the blockage of afferent activity is not a primary factor in reducing shivering activity. The reduction in esophageal temperature cooling rate during the occlusion period may have also contributed to the decreased heat production, although the cessation of shivering occurred almost immediately while cooling rates were reduced after a few minutes of occlusion. The continued heat flux from the skin during occlusion suggests a continuation of conductive heat loss from the underlying muscle tissue in the occluded limb to the surface for exchange with the surrounding water. Following the release of occlusion and recirculation of extremity blood skin heat flux was minimally altered (Figure 2.3). This would support the results of Coles and Cooper (1959) that an increased blood flow following release of cuff occlusion occurs predominantly in muscle tissue when skin blood vessels are vasoconstricted, as occurs during cold water immersion. The transient increase in heart rate after release of occluded blood may be attributed to the enhanced venous return to the right atrium, or Bainbridge reflex (Little, 1985). It is clear that further research investigating the cuff occlusion technique during whole body immersion in cold water is necessary.

The sensitivity of thermogenic heat production to a decreasing esophageal temperature has previously been reported in humans during the initiation of exercise in 10° C air (Hong and Nadel, 1979), and during immersion in 28° C water (Cabanac and Massonnet, 1977). The mean β observed in the present study ($-2.747 \pm .813$ $W \cdot kg^{-1} \cdot {}^{\circ}C^{-1}$) is similar to the β calculated from the data reported by Hong and Nadel (1979) during exercise at 30 W (-2.853 $W \cdot kg^{-1} \cdot {}^{\circ}C^{-1}$, using

body mass of present subjects as values were not reported). Inter-subject variability was also observed in their study. In contrast, Cabanac and Massonnet (1977) reported a mean value for β (-1182.8 ± 148 ($W \cdot {}^{\circ}C^{-1}$) which is over 5 times greater than the value recorded in this study when present mean body mass was incorporated. This discrepancy may be explained by the effect of an increased metabolism of tissues at higher temperatures, or the Q_{10} effect. In the study by Cabanac and Massonnet (1977) the subjects were hyperthermic (T_{es} = 38°C) prior to immersion in 28°C water. An elevation in internal temperature would increase metabolic activity of the tissues preserving a greater overall rate of heat production as esophageal temperature decreases. Immersion in cold water will lower muscle temperature which will result in a diminished blood flow (Lewis and Grant, 1925; Coles and Cooper, 1959) as well as a less effective utilization of the delivered oxygen (Abramson et al., 1958; Blomstrand et al., 1986). Thus the increased shivering thermogenesis associated with a decrease in core temperature at the lower tissue temperatures obtained in the present study would be lower in magnitude than if the tissues were hyperthermic prior to a reduction in core temperature as was the case with the subjects studied by Cabanac and Massonnet (1977).

The positive effect of an increased cooling rate on the central thermal drive to increased heat production is contrary to previous results observed in humans (Cabanac and Massonnet, 1977) and recently in goats (Roos and Jessen, 1987). However, Hammel (1973) proposed a time derivative component of hypothalamic temperature contributing to the thermoregulatory heat production response in dogs. In man, the presence of a rate component in central drive to increase heat production was suggested by the results reported by Nadel et al. (1970) following ingestion of ice cream. Equivocal results may be due in part to species differences as well as different techniques used to effect an isolated core perturbation. Although animal studies have provided invaluable information pertaining to thermoregulatory mechanisms, the conscious control of human subjects provides an additional input to this already complex system. In studies with human subjects the manipulation of esophageal temperature by limb occlusion/release in cold water (Knudsen, 1985; present study), ice cream ingestion in room at air temperature (Nadel et

al., 1970), exercise in a cold room (Hong and Nadel, 1979), and warm then cool water immersion (Cabanac and Massonnet, 1977) has been shown to result in an enhanced heat production. However, the status of the subject prior to core temperature manipulation (whether hypothermic, euthermic or hyperthermic) will alter the effector response as will the influence of exercise as discussed above. Thus comparison of data between studies is difficult.

From the present results, the individual variability in β at a standard rate of core cooling, as well as in the increased β associated with a greater cooling rate, may provide some insight into the errors in in prediction of thermoregulatory heat production with contemporary models as reported by Mekjavić and Morrison (1984; 1986). Most of the existing equations used to predict thermoregulatory heat production are based on absolute displacement of mean skin and core temperatures from their respective threshold values without accounting for the rate of change of these temperatures. Brown and Brengelmann (1970) and Timbal et al. (1976) were among the first to stress the need for the inclusion of time derivative terms in thermoregulatory models. Although models developed by these investigators incorporate both static and dynamic firing rate activity of peripheral thermoreceptors, only the static component of central thermoreception is considered, suggesting a negligible contribution of the dynamic core component. The influence of the rate of core cooling on the thermogenic response for any given skin temperature may explain the difference between the data obtained by Benzinger et al. (1963) and those reported by Craig and Dvorak (1966). As shown in Figure 2.1, a curvilinear relationship between tympanic temperature cooling and metabolic heat production was observed in the subject evaluated by Benzinger et al. (1963) and again presented by Benzinger (1970), while the mean $H - T_c$ relationship of eight subjects studied by Craig and Dvorak (1966) was linear. Mekjavić (1983) extended the skin temperature isotherms and reported a curvilinear relationship for one subject. These discrepancies as well as the differences in the magnitude of heat production may have been influenced by variations in core cooling rates.

The importance of evaluating individual thermoregulatory responses at similar core cooling rates is emphasized by the comparison of two subjects in Figure 2.5. These data suggest that subject 6 is thermosensitive to a slow rate of core cooling while subject 3 is responsive to a higher rate of core cooling. Present thermoregulatory models do not account for these inter-individual differences in the integration of thermoafferent information or the subsequent sensitivity of the effector response. Differential central thermosensitivities may account for the differences in the thermogenic response observed in two subjects whose core and skin temperature inputs were similar during immersion in 10° C water (Mekjavić et al., 1986).

The occlusion of extremity blood flow during cold water immersion appears to be a useful technique for evaluating the thermoregulatory heat production response to a dynamic change in core temperature. However, empirical verification that the enhanced metabolic heat production following the occlusion release is primarily a result of the reduction in core temperature (measured within the esophagus), with minimal contribution of the repayment of oxygen debt during reactive hyperemia, is desirable. As β may be taken as an index of central thermosensitivity for a specific $\dot{T}_{es}$ and at a constant skin temperature of $\simeq$18° C, this method may be used to evaluate individual variability in thermogenic responses further.

Potential factors which contribute to the differences in the thermogenic response to thermal stimulation during cold water immersion have been previously suggested. These include body composition and physique (Hayward and Keatinge, 1981; Strong et al., 1985), previous exposure (Hayward and Keatinge, 1981), sex differences (McArdle et al., 1984) and psychological predisposition (Brück et al., 1976; Radomski and Boutelier, 1982). Evidence of genetic influences on the thermogenic response to cold air have also been reported (Scholander et al., 1958; Hammel, 1964). Further research is necessary to quantify the effect of these factors on the central thermosensitivity of metabolic heat production during cold water exposures.

In conclusion, the results of this study have indicated that central thermosensitivity of metabolic heat production during cold water immersion exhibits inter-subject specificity among a

group of active, healthy young males. The enhanced rate of core cooling resulting from the occlusion/release of extremity blood contributed to a greater central thermosensitivity of heat production than that observed during a slower core cooling rate.

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CHAPTER III

THE ROLE OF MORPHOLOGY IN THE CENTRAL THERMOSENSITIVITY OF SHIVERING THERMOGENESIS DURING COLD WATER IMMERSION

Introduction

The magnitude of heat loss from the body during cold water immersion, evidenced by a decline in core temperature, has led to numerous investigations which have attempted to identify factors that limit core temperature cooling. A large emphasis has been placed on the role of subcutaneous adipose tissue acting as a peripheral insulator to diminish the heat flux between a subject's core and the surrounding environment (Pugh and Edholm, 1955; Carlson et al., 1958; Cannon and Keatinge, 1960; Keatinge, 1960; Rennie et al., 1962; Buskirk and Kollias, 1969; Hanna and Hong, 1972; Kollias et al., 1974; Timbal et al., 1976; Hayward and Eckerson, 1984; McDonald et al., 1984; Nunneley et al., 1985) as well as the effective area available for heat transfer, normalized to body mass (surface area to mass ratio) (Buskirk and Kollias, 1969; Sloan and Keatinge, 1973; Kollias et al., 1974; Hayward and Keatinge, 1981; McArdle et al., 1984; Strong et al., 1985). However, cooling rate is ultimately determined by the imbalance between heat loss and heat production mechanisms. Thus it has been suggested that the relationship between morphology and core cooling should account for individual differences in metabolic heat production (Strong et al., 1985; Mekjavić et al., 1986; Mekjavić et al., 1987b).

An inverse relationship between subcutaneous adiposity and the metabolic response (shivering thermogenesis) during cold water immersion has been reported (Carlson et al., 1958; Cannon and Keatinge, 1960; Keatinge, 1960; Keatinge and Evans, 1961; Buskirk et al., 1963; Buskirk and Kollias, 1969; Kollias et al., 1974; McArdle et al., 1976, Strong et al., 1985). This has led to the proposal that a large subcutaneous adipose layer may be associated with a lack of responsiveness and an alteration in the interaction between peripheral and central temperature and heat production shown in Figure 2.1 in Chapter II (Buskirk and Kollias, 1969). Alternatively, the

thermogenic response elicited during cold water exposure is an autonomic effector mechanism resulting from a combination of peripheral and central thermoreceptor stimulation (Benzinger et al., 1963; Craig and Dvorak, 1966; Benzinger, 1970; Brown and Brengelmann, 1970; Hayward et al., 1975, 1977; Timbal et al., 1976; Cabanac and Massonnet, 1977; Mekjavić and Morrison, 1985), thus the lack of responsiveness observed in subjects with larger adipose tissue or smaller surface area to mass ratios may be due to a diminished central thermal drive (Buskirk et al., 1963; Strong et al., 1985). When subjects were immersed in the coldest water temperature at which they were able to maintain a stable core temperature (core temperatures were relatively similar and rate of core cooling was nil), a linear relationship between metabolic rate and subcutaneous fat thickness was not evident as the differences in the thermogenic response of subjects with similar skinfold thicknesses were large (Hayward and Keatinge, 1981). However, water temperatures ranged from 12 to 32° C, thus differences in the peripheral thermogenic drive may have contributed to the variability observed.

In the present study, the blood occlusion method detailed in Chapter II was conducted on a larger group of subjects with a wide range of morphological characteristics. Thus the central thermosensitivity of metabolic heat production of the volunteers, who were immersed in cold water (≃15° C) with their skin temperature clamped at a level a few degrees above bath temperature, was evaluated and inter-subject comparisons made with respect to morphological characteristics.

Methods

Seventeen males, 29.0 ± 3.3 (SD) years of age, consented to participate in the investigation following medical evaluation and information of procedures and potential risks. All procedures were approved through the SFU Research Ethics Review Committee. Aerobic fitness has been implicated as a potential factor altering the thermogenic response to cold water immersion (Buskirk and Kollias, 1969; Hayward and Keatinge, 1981; Jacobs et al., 1984), thus maximal oxygen uptake ($\dot{V}O_2max$) was determined in each subject. Aerobic fitness, evaluated from maximal aerobic

power, and anthropometric measurements were obtained a minimum of one week prior to each cold water immersion.

Maximal Aerobic Power

A graded maximal exercise test, performed on an electrically-braked cycle ergometer (Lode, Groningen, Holland) was used to evaluate maximal aerobic power. A 25 W load was introduced every 30 seconds following four minutes of unloaded pedalling at 90 RPM. When the subject could no longer maintain a speed of 80 RPM, the test was terminated.

Throughout the exercise test, oxygen uptake ($\dot{V}O_2$, $L \cdot min^{-1}$ STPD) was determined from the analyses of inspired gas volumes and mixed expired gases. Inspired volumes were determined each breath using a turbine volumeter (Alpha Technology, Laguna Hills, Ca.). Expired oxygen and carbon dioxide gas contents were sampled from a mixing chamber with an Applied Electrochemistry S-3A O_2 analyzer and an Applied Electrochemistry CO_2 analyzer (Sunnyvale, Ca.). Calibration of gas analyzers was conducted prior to each test with room air and a standard calibration gas. Barometric pressure was recorded from a mercury barometer (Fischer Scientific, Ottawa, Ont.), and relative humidity determined with a sling psychrometer (Taylor Instrument Co., Rochester, N.Y.).

Data were collected on-line with a Hewlett-Packard 3497A data acquisition system driven by a Hewlett-Packard (HP85) minicomputer. Values were recorded at the end of each breath and stored for printout upon completion of the test. $\dot{V}O_2$ for each breath was displayed graphically throughout the exercise. Maximal oxygen consumption ($\dot{V}O_2max$) was calculated by averaging the $\dot{V}O_2$ over 5 breaths every 10 seconds during the final minute of work. In all cases a plateau was achieved and $\dot{V}O_2max$ was determined as the average of the two highest consecutive values obtained during the plateau phase.

Heart rates (HR, $b \cdot min^{-1}$) were recorded from an electrocardiograph (Fukuda-Denshi, Tokyo, Japan) at the end of each work increment. Disposable silver/silver chloride electrodes

(Medi-Trace, Graphic Controls Canada Limited, Ont.) were placed on the subject's chest in a modified Lead I (CM_5) position. A visual display of the ECG trace was monitored continuously during the exercise with an oscilloscope (Physio-Control Systems, Seattle, Wa.).

Body composition and Physique

Complete anthropometric profiles, including 12 girths, 8 skinfold thicknesses, 6 breadths, 9 segmental lengths, height and body mass were obtained following the guidelines established by the International Working Group on Kinanthropometry (Ross and Marfell-Jones, 1983). Skinfold thicknesses were measured with a Harpenden Caliper (John Bull, England). Girths were assessed by a flexible steel tape (Keuffel and Esser Co., West Germany). An anthropometer and sliding caliper (GPM Co., Switzerland) were utilized for the measurement of lengths and breadths. Girths, lengths and breadths were measured to the nearest 1 mm while skinfolds were assessed to the nearest .1 mm. The measurements were incorporated into a geometrically-based model for the anthropometric estimation of tissue masses (skin, adipose, muscle, bone and organs plus viscera) proposed by Drinkwater (1984), as well as used to calculate the Heath-Carter anthropometric somatotype (Carter, 1980). These methods will be discussed in detail in the "Data Analyses" section below.

Immersion Protocol

Subjects reported to the laboratory in a rested, post-absorptive state, having refrained from food consumption and physical exertion a minimum of 4 hours. Insertion of the esophageal temperature probe and placement of transducers and electrodes were carried out in a thermoneutral room (24.8 ± 1.40 (SD) $^{\circ}$C). After 30 minutes the subject was assisted into a mesh chair suspended from the ceiling above the immersion tank. After positioning the mouthpiece for respiratory gas collection, pre-immersion recordings were obtained for 5 minutes. At the end of the baseline period the subject was immediately lowered into the tank until the water level reached the sternal notch. The water was well-stirred and the temperature maintained

at a constant level as outlined in the previous chapter. The mean ± standard deviation for water temperature (T_w) over all 17 trials was 15.65 ± 1.50 °C.

During immersion, blood flow to the extremities was occluded utilizing the apparatus and procedures detailed in Chapter II. Inflation of the pressure cuffs was initiated following the stabilization of skin temperature (within 5–10 minutes). An attempt was made to initiate the occlusion at a common T_{es} of 36.5° C, when possible. For those subjects whose T_{es} was maintained above 36.5° C, occlusion was introduced at minute 40 of the immersion. Following the ten minute occlusion period, cuff pressure was automatically released and the subject remained immersed until T_{es} approached 35° C, or upon completion of one hour of the immersion procedure. The subject was then removed from the tank and placed in a well–insulated bed for 30 minutes while rewarming occurred via endogenous heat production.

Instrumentation

Core temperature was assessed within the esophagus with a YSI 702A thermistor probe (Yellow Springs Instrument, Yellow Springs, Ohio). For 13 subjects, the esophageal probe was inserted 37 cm beyond the external nares to its maximal length. In four subjects an extended probe enabled the depth of insertion to be individually estimated from anthropometric measurements. Insertion depth was calculated from the distance between the external nares and mandibular angle plus the distance between the nares and 2 cm above the junction of the xyphoid process and costal cartilage. The depth ranged from 43 to 46 cm for the four subjects. Mean skin temperature $\overline{T}_{sk}$ and mean heat flux $\dot{Q}_{mean}$ were calculated from the unweighted averages of five sites as described in Chapter II.

Metabolic heat production (H, $W \cdot kg^{-1}$), calculated from oxygen uptake[1], and heart rates (HR, $b \cdot min^{-1}$) were measured with instrumentation and procedures reported in the previous chapter. All data were collected as documented in Chapter II.

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[1] Conversion Factor : 1 Liter $O_2 \cdot min^{-1}$ = 352.015 W; based on the energy equivalent of 5 kcal for each liter O_2 consumed.

Data Analyses – Body Composition and Physique

1. Estimation of Tissue Compartments

In addition to the importance of adipose tissue as insulation surrounding the internal core, unperfused muscle tissue has been recognized as a major component of the peripheral insulative shell (Veicsteinas et al., 1982; Park et al., 1984). During colder water immersion muscle mass may contribute to shivering thermogenesis in an attempt to limit the reduction in core temperature (Carlson et al., 1958; Hayward and Keatinge, 1981). As the thermogenic response is influenced by both heat loss and heat production, the estimation of both muscle and adipose compartments was determined. A geometric model of human body composition (Drinkwater, 1984) was used to estimate adipose and muscle tissue components. This technique was recently incorporated in the determination of the rate of body heat storage during cold water immersion (Kakitsuba and Mekjavić, 1987). The model describes the body as 10 truncated cones, each comprised of concentric shells of tissues (skin, adipose, muscle, bone and organs plus viscera). The volume of each segment was determined from the geometrical dimensions obtained by surface anthropometry. Cadaver data (Clarys et al., 1984) were utilized to determine relationships between measured volumes and anthropometrically-derived volumes, thus a scaling factor for the "true" volume was included in the model. For the purpose of the present study total body muscle volume (MV), adipose volume (AV) and their ratio (MV:AV) were included in the evaluation of the effect of morphology on central thermosensitivity.

2. Somatotype and Somatotype Attitudinal Distance

The Heath-Carter anthropometric somatotype (Carter, 1980) was calculated for the determination of physique. Initially conceptualized by Sheldon et al. (1940), the somatotype is comprised of three components – endomorphy, mesomorphy and ectomorphy. Endomorphy represents relative fatness, mesomorphy expresses relative musculoskeletal development per unit height and ectomorphy represents relative linearity (Carter, 1980). The individual somatotypes of the subjects are shown in the somatochart in Figure 3.1.

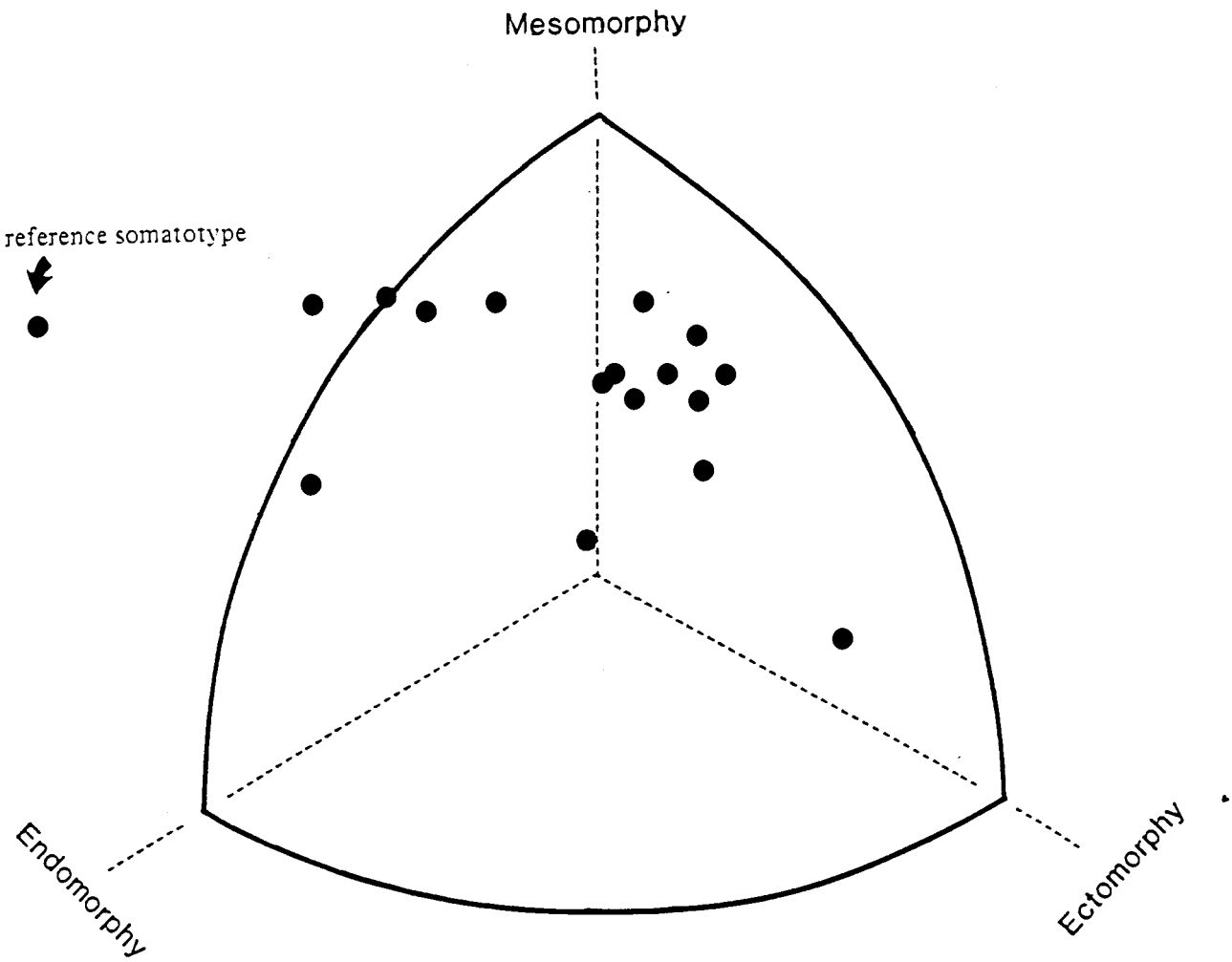

Figure 3.1: Somatochart of individual somatotypes. The somatopoint for Subject 16 is the reference somatotype for calculation of SAD.

The separate physique components of the somatotype have been used to evaluate thermoregulatory responses during exposure to heat (Docherty et al., 1986; Hayward et al., 1986) and cold water immersion (Morrison et al., 1980; Hayward and Eckerson, 1984). Mesomorphy was related to the development of hyperthermia during exercise in a warm, humid environment (Hayward et al., 1986), while ectomorphy was shown to influence the loss of body heat during cold water exposure (Morrison et al., 1980; Hayward and Eckerson, 1984). Although the comparison of individual somatotype components in evaluating thermoregulatory responses has proved fruitful, the importance of the relationship of the ratings to each other, the "true" meaning of the somatotype, may have been overlooked (Carter et al., 1983). By comparing the overall somatotype, the morphological characteristics which influence both heat loss and heat production may be evaluated simultaneously. The comparison of the total somatotype between individuals is based on the somatotype attitudinal distance (SAD) which is a quantification of the deviation of the somatotype components for each individual from those of a reference somatotype. The SAD was calculated according to the following procedure outlined by Carter (1980):

$$SAD = \sqrt{(I_A - I_B)^2 + (II_A - II_B)^2 + (III_A - III_B)^2} \qquad (1)$$

where: I = Endomorphy; II = Mesomorphy; III = Ectomorphy;
A = Individual's somatotype; B = Reference somatotype

In the present study, the somatotype of subject 16, an extreme mesomorphic-endomorph, was used as the reference somatotype to which all other subjects were compared. Although theoretically any somatotype may be used as a reference somatotype, this subject was selected on the basis of his extreme value (Figure 3.1) as well as his "typical" endomorphic response during the cold water immersion (see "B" in Figure 3.2). An application of SAD to performance was reported by

Araujo (1979, cited by Carter et al., 1983) who compared the somatotypes of Brazilian swimmers to that of a reference Olympic-caliber swimmer.

3. Surface Area to Mass Ratio and Skinfold Thickness

The influence of both surface area to mass ratio (A_D:M) and subcutaneous adiposity, determined from skinfold thickness, on heat transfer during cold water immersion have been documented. Therefore, these variables were also included in the evaluation of the relationship between morphology and central thermosensitivity. Surface area (A_D) was determined with the formula proposed by DuBois and DuBois (1916). The sum of 8 skinfold thicknesses (skin plus adipose tissue; ΣSKF) was used to estimate subcutaneous adiposity.

Data Analyses – Core Thermogenic Drive

Upon deflation of the pressure cuff, a decrease in esophageal temperature (T_{es}) was elicited by the cooled venous return from the limbs to the core region. This decrease in T_{es} was accompanied by an increase in heat production (H), measured by $\dot{V}O_2$, which was sustained well above its pre-occlusion value for the remainder of the immersion (see Figure 3.2). As described in Chapter II, the relationship between the increased H and decreased T_{es} was evaluated from the slope of the H – T_{es} relationship during the transient decrease in T_{es} following the release of occluded blood flow. The slope of the H – T_{es} relationship (β) may be indicative of the sensitivity of the controlling system to a central thermal input at a constant skin temperature ($\simeq 18^\circ$C).

In order to make a comparison of β between subjects, as it was measured in the present study, several criteria were established to ensure that the central thermal drive following release of occluded blood was similar for all subjects. Therefore subjects whose T_{es} did not decrease below 37°C at the initiation of cuff pressure were excluded from the group comparison. The rationale for this exclusion was based on the confounding influence of an enhanced metabolism of higher tissue temperatures on overall heat production as well as the reduction of blood flow in muscle

tissue at lower temperatures (Coles and Cooper, 1959), as previously discussed in Chapter II. A second criterion for exclusion from the group comparison was that the decrease in T_{es} instigated by the venous return of cooled blood following release of cuff pressure, was greater than one standard deviation of the group mean decrease. This criterion was established because of the effect of the magnitude of change in T_{es} on the thermogenic response reported by Nadel et al. (1970).

Statistical Analyses

The relationship between the anthropometric variables and the β response was determined by an "all possible subsets" regression analysis (Dixon, 1983). The adjusted r^2 value was chosen as the criterion for selection of the best possible subset. Correlation matrices were generated to assess the relationships among variables with statistical significance set at $p < 0.05$. The slope of $H - T_{es}$ were determined from least squares linear regression analyses. Mean values and standard deviations are reported for group data.

Results

Physical Characteristics

Anthropometric and aerobic fitness data are shown for all subjects in Table 3.1. The coefficient of variation (C) for ΣSKF (64%), SAD (35%) and MV:AV (47%) suggests a wide distribution between subjects for these variables. In contrast, MV and A_D:M are relatively homogeneous as C for these variables was 11% and 7%, respectively. The volume of the limbs (42.051 ± 6.385 L) accounted for 52.4 ± 3.2 % of total body volume as the trunk and head volume combined was 38.628 ± 9.071 L. A wide dispersion in somatotypes among the subjects is indicated by the group somatochart in Figure 3.1, however the mesomorphic component was dominant as might be expected for the healthy, active male volunteers in the present study. The mean somatotype (Table 3.1) is similar to that reported previously for male Canadian students

Table 3.1: Morphological characteristics and aerobic fitness of subjects

Subject No.	Height (cm)	Mass (kg)	Σ 8 Skf (mm)	Muscle Vol. (L)	Musc/Adip (N.D.)	A_D (m^2)	A_D:M ($m^2 \cdot kg^{-1}$)	Somatotype (N.D.)	SAD (N.D.)	$\dot{V}O_{2max}$ ($L \cdot min^{-1}$)
1	169.7	63.6	59.1	29.128	2.687	1.74	.0274	1.8-5.7-2.5	7.76	3.50
2	199.3	79.5	54.3	38.765	3.263	2.14	.0269	1.6-2.6-5.3	10.32	4.22
3	183.9	76.3	43.1	39.483	4.344	1.99	.0261	1.2-4.7-3.1	8.81	4.95
4	188.5	87.0	95.0	35.589	1.617	2.14	.0246	2.2-4.9-2.5	7.69	4.30
5	175.2	80.2	133.7	34.461	1.633	1.96	.0244	3.8-6.0-1.2	5.56	3.84
6	189.4	86.6	113.6	37.398	1.767	2.14	.0247	2.9-3.3-2.7	7.99	4.15
7	170.1	76.8	135.4	30.537	1.442	1.88	.0245	4.1-6.3-0.9	5.14	3.92
8	186.4	81.9	55.7	40.427	3.532	2.06	.0252	1.6-5.0-2.7	8.23	5.41
9	164.3	62.7	99.2	28.453	2.036	1.68	.0268	3.2-6.1-1.7	6.16	4.08
10	189.2	79.2	75.5	37.513	2.621	2.06	.0260	2.0-4.2-3.6	8.51	4.40
11	171.3	74.2	160.9	30.056	1.240	1.87	.0252	5.4-4.6-1.2	5.00	3.45
12	176.9	69.9	44.9	33.326	4.042	1.86	.0266	1.3-4.3-2.8	8.80	4.67
13	177.0	72.5	70.2	32.213	2.461	1.89	.0261	1.9-4.4-2.5	8.17	3.58
14	178.5	90.0	190.0	30.539	.851	2.09	.0232	5.1-6.6-0.9	4.10	3.78
15	183.9	77.2	50.9	37.827	3.697	2.00	.0259	1.5-5.3-3.0	8.30	4.68
16	175.1	120.4	301.8	34.631	.606	2.33	.0194	8.9-8.1-0.5	0.00	3.90
17	176.9	71.1	77.5	32.114	2.235	1.88	.0264	2.4-5.0-2.6	7.50	5.05
Mean	179.7	79.4	103.6	34.262	2.357	1.98	.0250	3.0-5.1-2.3	6.94	4.23
S.D.	9.0	13.0	66.8	3.839	1.116	.16	.0020	2.0-1.3-1.2	2.43	.57

(2.8–4.9–2.8) (Carter et al., 1982; Carter, 1984). The SAD value for the reference somatotype is 0, thus the largest SAD value (10.3, subject 2) corresponds to the somatopoint furthest from the reference somatopoint (Figure 3.1).

Although none of the subjects were members of an athletic team, most were physically active. The average value for $\dot{V}O_2max$ ($L \cdot min^{-1}$) in the present group (Table 3.1) was greater than that reported for trained physical education students of a similar mean age (3.0 ± .50 $L \cdot min^{-1}$) (Åstrand and Rodahl, 1986).

Physiological Responses

1. Pre–Occlusion Phase

Time course changes in T_{es}, H, $\bar{T}_{sk}$, and $\dot{Q}_{mean}$ are shown for subjects 5 and 16 in Figure 3.2. Upon immersion $\bar{T}_{sk}$ decreased rapidly to a constant value within 5 minutes. The mean value and standard deviation for the group was 17.95 ± 1.03° C. $\dot{Q}_{mean}$ reached a peak value of 873.49 ± 110.01 $W \cdot m^{-2}$ during the first minute of immersion and continued to decline until approximately the 25th minute of immersion. The value for $\dot{Q}_{mean}$ observed during the 5 minutes prior to occlusion of extremity blood flow was 246.48 ± 85.16 $W \cdot m^{-2}$. As shown in Figure 3.2, those subjects higher in subcutaneous adiposity exhibited lower $\dot{Q}_{mean}$ values.

During the first minute of immersion all subjects exhibited a transient elevation in heat production from its resting level (4.39 ± 1.90 $W \cdot kg^{-1}$). In several subjects H remained elevated during the first 10 minutes (Figure 3.2A) followed by a more gradual increase with the onset of decline in T_{es}. The average rate of cooling of T_{es} ($\dot{T}_{es}$) was −.024 ± .021° $C \cdot min^{-1}$ with a range between +.005 and −.064° $C \cdot min^{-1}$. Subject 16 (Figure 3.2B) showed a minimal thermogenic response throughout the immersion as T_{es} remained at or above its pre–immersion value, thus his data was excluded from the comparison of central thermosensitivity between subjects. Two additional subjects (11 and 14) were also excluded from the group comparison as T_{es} remained above 37° C when occlusion was initiated. These three subjects had the largest ΣSKF values which provides further support for an insulative effect of subcutaneous adiposity. However, aside

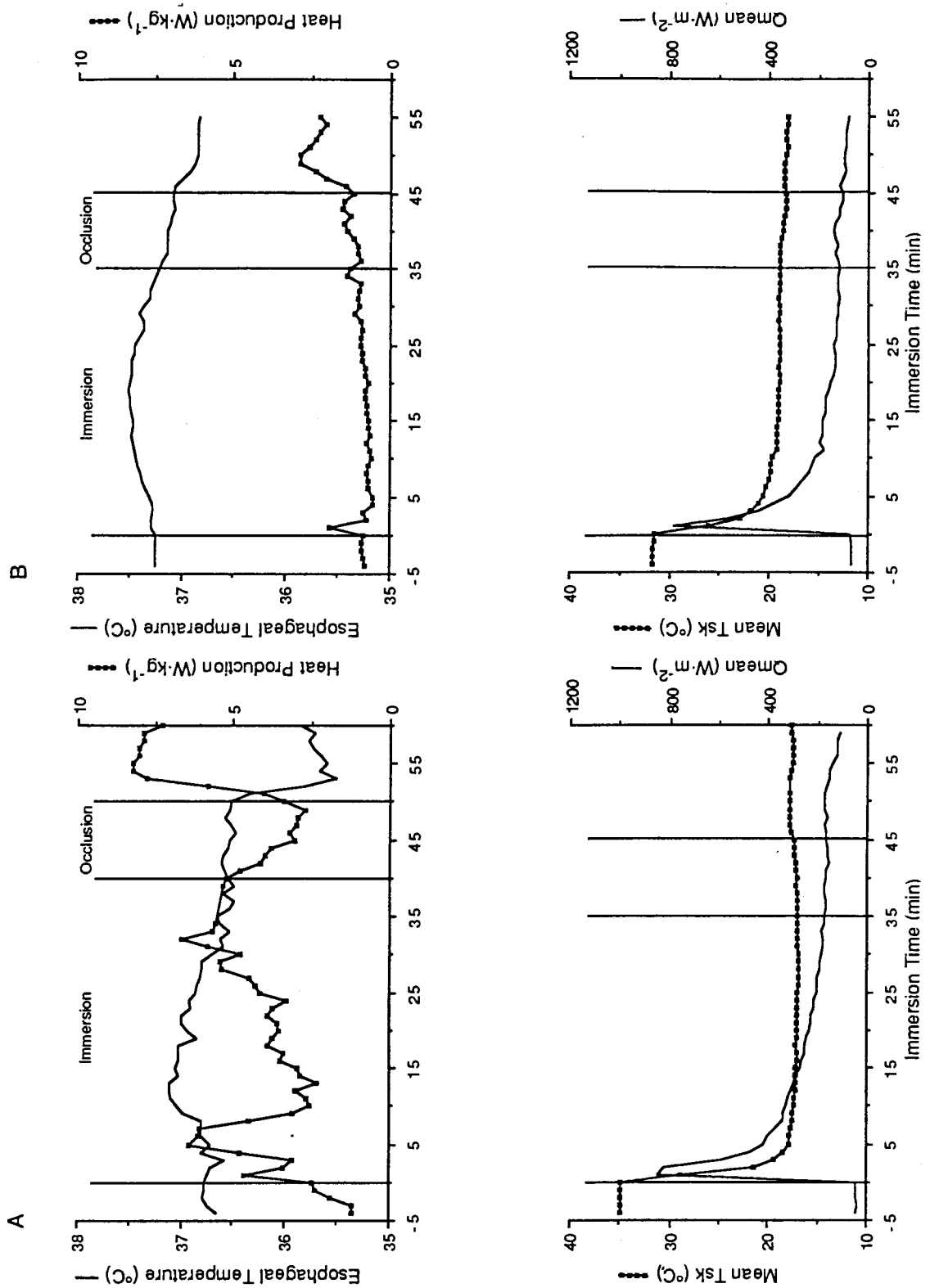

Figure 3.2: Time course of T_{es}, H, $\overline{T}_{sk}$, and $\dot{Q}_{mean}$ for Subjects 5 (A) and 16 (B) during 15°C water immersion.

from the three extreme values, a larger ΣSKF thickness was not always associated with a low thermogenic response, as evidenced by the data of subject 5 in Figure 3.2A (ΣSKF = 133.7 mm; mean skinfold thickness = 16.7 mm).

2. Occlusion Phase

During the 10 minutes of pressure cuff occlusion of extremity blood flow, T_{es} generally stabilized. The pre-occlusion $\dot{T}_{es}$ (-.018 ± .029 $^{\circ}C \cdot min^{-1}$) was reduced to -.007 ± .025 $^{\circ}C \cdot min^{-1}$ during occlusion. Individual variability in the rate of heat production in response to the occlusion of extremity blood flow was exhibited (Figure 3.2). In general there was a slight reduction in H at the end of the occlusion phase from its pre-occlusion value (3.59 ± 1.49 $W \cdot kg^{-1}$ to 2.66 ± 1.17 $W \cdot kg^{-1}$) and visible shivering was markedly reduced. $\dot{Q}_{mean}$ was minimally altered by the occlusion of extremity blood flow which suggests the return of cooled blood was mainly confined to the deeper tissues as has been reported previously for forearm blood flow (Coles and Cooper, 1959).

3. Post-Occlusion Phase

Upon deflation of the cuffs, the venous blood returning from from the cooled occluded limbs to the core region resulted in a rapid nonlinear decrease in T_{es} and a concomitant rise in H which stabilized at an elevated level, usually within 5 minutes. The decrease in T_{es} for subject 3 was 1.82° C which was well above 1 SD greater than the the group mean decrease of .82 ± .30° C, thus this subject was not included in the comparative analysis of central thermosensitivity. The decrease in T_{es} and increase in H (ΔH) following the release of occlusion and subsequent recirculation of the cooled extremity blood are reported for the 13 remaining subjects in Table 3.2. The ΔH ranged from 1.76 to 5.54 $W \cdot kg^{-1}$ although the decrease in T_{es} was similar (.88 ± .24° C). As seen in Figure 3.3, the decrease in T_{es} following the cuff occlusion method was homogeneous among the subjects (C = 0.5 to 0.7%). In contrast thermogenic response data exhibited a larger dispersion (C = 29.3% to 35.1%).

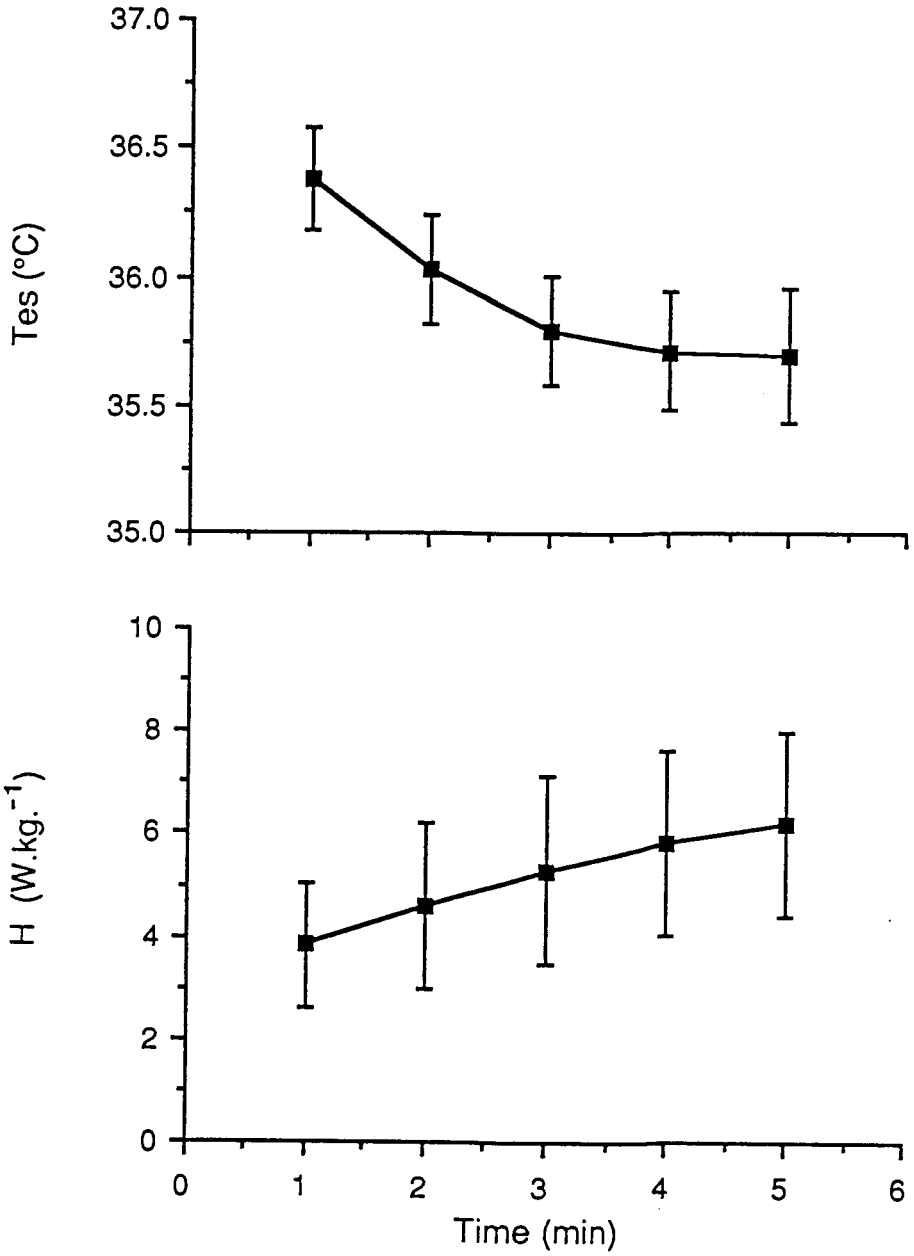

Figure 3.3: Mean ± SD for T_{es} and H responses for 13 young male subjects during the first 5 minutes following release of occlusion and subsequent recirculation of cooled extremity blood flow.

Central Thermosensitivity

The H – T_{es} relationship (β) determined during the transient decay in T_{es} is shown for the individual subjects in Table 3.2 along with the r^2 value for the equation and number of data points included. In general, T_{es} attained its steady state value within 5 minutes following the release of the cuff.

Relationship Between Morphology and Central Thermosensitivity

Table 3.3 shows the correlation coefficients among variables included in the regression analysis to evaluate the relationship between morphology, maximal aerobic power ($\dot{V}O_2max$, $L \cdot min^{-1}$) and central thermosensitivity (β). The relationship between variables was significant at the .05 level when $r \geq .553$ (df = 11). A significant negative correlation was observed between ΣSKF and SAD, A_D:M, and MV:AV, while muscle volume was positively related to SAD. Aerobic power was not significantly related to the morphological characteristics, however there was a tendency for subjects having a larger volume of muscle to have a greater aerobic power (r = .547, $p < 0.10$). The correlation matrix showed no significant relationship between central thermosensitivity (β) and morphology or aerobic power.

An "all possible subsets" multiple linear regression analysis of the relationship between morphological characteristics and $\dot{V}O_2max$ to central thermosensitivity was calculated, however the final best fit equation selected was not statistically significant (F = 2.41, p = .149).

Discussion

In 13 of the 17 subjects participating in the present study, the cuff occlusion method provided a similar peripheral (18° C) and central (36.5° C) thermal drive to metabolic heat production during cold water immersion. This allowed the resultant effector (H) – stimulus (T_{es}, $\dot{T}_{es}$) relationship, or central thermosensitivity (β) to be quantified and compared between individuals representing a range of morphological characteristics and aerobic fitness. Further

Table 3.2: T_{es}, ΔH and ß responses following release of occluded extremity blood

Subject No.	Initial T_{es} (°C)	Final T_{es} (°C)	ΔH ($W \cdot kg^{-1}$)	ß ($W \cdot kg^{-1} \cdot °C^{-1}$)	n	r^2
1	36.32	35.56	2.96	-4.353	4	.998
2	36.16	35.04	3.54	-2.585	10	.786
4	36.75	35.65	5.38	-2.373	8	.528
5	36.52	35.52	5.20	-4.353	3	.956
6	36.53	35.56	2.92	-3.121	4	.788
7	36.70	35.95	2.30	-2.205	4	.731
8	36.36	35.70	4.70	-4.375	7	.712
9	36.14	35.55	1.76	-2.402	10	.440
10	36.77	35.92	3.45	-3.308	10	.412
12	36.51	35.64	4.01	-2.448	4	.501
13	36.26	35.21	3.69	-2.255	5	.950
15	36.23	35.33	3.89	-4.245	10	.703
17	36.56	35.79	5.54	-5.702	7	.958
Mean	36.45	35.57	3.79	-3.363	6	.728
S.D.	.22	.26	1.17	1.126	3	.206

n = number of data points in regression analysis

r^2 = coefficient of determination for regression equation to determine ß

Table 3.3: Correlation coefficients between ß and morphological/fitness characteristics

Variable†	ΣSKF	Muscle Vol.	SAD	A_D:M	MV:AV	$\dot{V}O_{2max}$	ß
ΣSKF	--------						
Muscle Vol.	-.284	--------					
SAD	-.816*	.577*	-------				
A_D:M	-.701*	-.317	.483	-------			
MV:AV	-.903*	.392	.706*	.538	-------		
$\dot{V}O_{2max}$	-.400	.547	.304	-.053	.491	-------	
ß	-.158	.130	-.037	.083	.134	.424	------

† See text for abbreviations

* $p < .05$.

discussion pertaining to the determination of central thermosensitivity was presented in Chapter II. Inter-subject differences in central thermosensitivity was evident across the present group of subjects. However, neither the morphological variables nor aerobic fitness level were significantly related to the thermogenic response following a similar thermoafferent input.

In most previous studies which have evaluated the relationship between morphology and thermoregulatory responses during cold water immersion, the insulation provided by subcutaneous adipose tissue resulted in a diminished central drive to shivering thermogenesis as evidenced by a smaller decline in core temperature or a slower rate of core cooling (Pugh and Edholm, 1955; Carlson et al., 1958; Keatinge, 1960; Sloan and Keatinge, 1973; Kollias et al., 1974; Nadel et al., 1974; Timbal et al., 1976; Hayward and Eckerson, 1984; McDonald et al., 1984; Nunneley et al., 1985). As a result of the reduced central thermal input, shivering thermogenesis will also be reduced suggesting an inverse relationship between subcutaneous adiposity and metabolic heat production (Carlson et al., 1958; Cannon and Keatinge, 1960; Keatinge, 1960; Keatinge and Evans, 1961; Buskirk et al., 1963; Buskirk and Kollias, 1969; Kollias et al., 1974; McArdle et al., 1976, Strong et al., 1985). For those subjects in whom core temperature is decreased, a large variability in the metabolic response to cold water immersion has been reported (Buskirk et al., 1963; Buskirk and Kollias, 1969; Hayward and Keatinge, 1981; McArdle et al., 1984; Strong et al., 1985). These differences will influence the rate of decline of core temperature as the heat generated may be stored (Strong et al., 1985). This may be seen in the thermoregulatory responses to 10°C water immersion for two subjects reported by Mekjavić et al. (1986). A large initial thermogenic response observed in the leaner subject in their experiment reduced his core temperature cooling rate such that he cooled as slowly as a subject whose sum of 6 skinfolds was over three times greater. These results suggest that the variability in heat production for a given combination of peripheral and central temperature inputs may be influenced by morphological factors. As it is unlikely that thermoreceptor characteristics will be altered by morphology, a possible mechanism may be the modification of nonthermosensitive central neurons responsible for the integration of thermoafferent stimuli (Brück, 1976). Thus a certain physique and/or body

composition may be thought of as contributing to acclimatization.

In a study by Hayward and Keatinge (1981), subjects were immersed in the coldest water in which they were able to maintain core temperature. When core thermal drive was similar (T_{re} = 35.85 ± .36°C (SD); $\dot{T}_{re}$ = 0), a strong inverse relationship between metabolic heat production and subcutaneous adiposity was not observed as the variability in thermoregulatory heat production was high at any given level of adiposity. The peripheral thermal drive varied in these experiments since water temperature ranged from 12 to 32°C, thus the integrated thermoafferent drive was not similar. However, similar results are seen in the present study when both the peripheral and central thermal drives to metabolic heat production were standardized. Thus the hypothesis that morphology alters the central integration of thermoafferent stimuli, in a mannner similar to that expected from long-term cold or heat exposure, appears unfounded.

Buskirk and Kollias (1969) stated that knowing body fatness alone does not enable the precise prediction of the metabolic response to cold. This has led to the inclusion of surface area to mass ratio (A_D:M) in the evaluation of the thermogenic response during cold water immersion (Buskirk and Kollias, 1969; Sloan and Keatinge, 1973; Kollias et al., 1974; Hayward and Keatinge, 1981; McArdle et al., 1984; Strong et al., 1985). The importance of this geometric component in addition to subcutaneous adiposity in reducing body heat loss is evident when comparing males and females, as the effective area available for heat loss is greater in females at any given level of adiposity (Kollias et al., 1974; McArdle et al., 1984). This was also shown in core cooling of children (Sloan and Keatinge; 1973). The inverse relationship between surface area to mass ratio and subcutaneous adiposity observed in the present study was also reported by Kollias et al. (1974). Hayward and Keatinge (1981) have suggested that a greater A_D:M is associated with a smaller muscle mass and therefore the ability to produce less heat. In the present study a low correlation was obtained between muscle volume and A_D:M (r = −.317) and neither were related to the thermogenic response following the increased core thermal drive. However, muscle volume was relatively homogeneous in the present group of subjects which may account for the lack of

relationship.

Aerobic fitness has also been proposed as a potential factor altering the thermogenic response during cold water immersion (Buskirk and Kollias, 1969; Hayward and Keatinge, 1981; Jacobs et al., 1984). Although the correlation coefficient for the relationship between aerobic fitness and central thermosensitivity was the highest among all the predictor variables in the present study (r = .424), this relationship was not statistically significant. Hayward et al. (1975) studied the thermogenic response in males and females exposed to water temperatures between 5 and 18° C and found no relationship between fitness and the level of thermogenesis. Jacobs et al. (1984) have suggested the method of determining aerobic fitness may partially explain these equivocal results. They measured the level of blood lactate accumulation following a submaximal exercise test which they proposed to be the most sensitive indicator of aerobic fitness. However utilizing a similar workrate (200 W) for all subjects may place the smaller subjects, who may be extremely fit, at a disadvantage as they are working at a greater relative aerobic power. In the present study post-exercise blood lactates were not evaluated, however the use of maximal aerobic power as an indication of training and hence relative fitness is well accepted (Åstrand and Rodahl, 1986).

In addition to the morphological characteristics classically evaluated during cold water exposure (e.g., subcutaneous adipose tissue, A_D:M), the Heath-Carter anthropometric somatotype was assessed. Logically, all three components of the somatotype may play a role in the thermoregulatory response during cold water immersion. Endomorphy refers to relative fatness thus would provide a means of assessing insulation. Mesomorphy or relative muscularity may be important in determining both insulation and the metabolically active tissue available for increasing shivering thermogenesis. Ectomorphy or relative linearity indicates the effective surface area available for heat transfer. In the past, researchers have utilized the components separately in evaluating core temperature responses during cold water immersion. Morrison et al. (1980) reported that subjects with high ectomorphy ratings had lower core temperatures prior to

rewarming following immersion in 5°C water. Hayward and Eckerson (1984) observed a significant effect of endomorphy on reducing the rate of core cooling in males and females immersed in ice water. Although this approach is valid and offers a technique for assessing morphological characteristics, the somatotype is intended as a technique to assess the relationship of the three components to each other. In the present study the somatotype attitudinal distance was evaluated in an attempt to determine whether the interrelationship between the three components may provide a means for predicting central thermosensitivity. The correlation matrix (Table 3.2) showed a poor correlation between SAD and central thermosensitivity.

As suggested previously, both muscle and adiposity may play important roles in the thermogenic response to a reduction in peripheral and central temperature. It was hypothesized that the ratio of these two tissue compartments may provide a better indicator of central thermosensitivity than the individual tissues. The geometric model proposed by Drinkwater (1984) provided a technique for assessing body composition and has been used to determine the rate of storage of body heat during cold water immersion (Kakitsuba and Mekjavić, 1987). However, the present results suggest that body composition is not an important determinant of central thermosensitivity for the range of thermal inputs evaluated.

Although the influence of morphology and fitness on the thermogenic response during cold water immersion has been reported, present data suggests that when individuals are given a similar thermal drive the large variability observed in their thermogenic responses is not related to differences in morphology or fitness. Additional factors cited as influencing thermogenesis during cooling include dietary status, psychological profile, genetic background and previous exposure (Bazett, 1949; Scholander et al., 1958; Hammel, 1964; Buskirk and Kollias, 1969; Brück, 1976; Baum et al., 1976; Hayward and Keatinge, 1981). In the following chapter the effect of short-term adaptation on central thermosensitivity will be investigated.

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CHAPTER IV

SHORT-TERM ADAPTATION IN CENTRAL THERMOSENSITIVITY OF METABOLIC HEAT PRODUCTION DURING COLD WATER IMMERSION

Introduction

Alterations in man's thermoregulatory heat production response to repeated cold water exposures have been evidenced in both long-term acclimatization (Rennie et al., 1962; Hong, 1973) and short-term acclimation studies (Lapp and Gee, 1967; Skreslet and Aarefjord, 1968; Radomski and Boutelier, 1982; Young et al., 1986). Such modifications include a decreased core temperature at which shivering is initiated (Rennie et al., 1962; Hong, 1973; Young et al., 1986) and a diminished shivering or metabolic response (Lapp and Gee, 1967; Skreslet and Aarefjord, 1968). The decrease in thermogenesis has been attributed to a reduction in sympathetic activity (Radomski and Boutelier, 1982) however evidence of an increased sympathetic response, which would lead to a greater peripheral vasoconstriction thereby diminishing heat flux from the core to the surrounding environment, has been documented (Paik et al., 1972; Hong, 1973; Young et al., 1986). Differences in methods of cold adaptation (intermittent vs. continuous) and assessment of sympathetic activity (urinary vs. plasma norepinephrine) have been cited as potential factors influencing these equivocal results (Radomski and Boutelier, 1982; Young et al., 1986). Alternatively, it has been postulated that the process of adapting to repeated cold water exposure occurs along a continuum which includes first, a "hypothermic" adaptation (e.g., decreased shivering threshold temperature, reduced metabolic response), followed by an "insulative" adaptation (e.g., increased vasoconstriction) (Skreslet and Aarefjord, 1967; Young et al., 1986).

Evidence of an initial hypothermic adaptation to cold air in man has been reported to occur within hours (Brück and Hinckel, 1984) and within 4–7 exposures over a 2-week period (Brück et al., 1976). Modifications reported in these studies were a decreased mean body temperature at which shivering was initiated, a lower esophageal temperature at any given ambient temperature

and a reduction in the rate of oxygen uptake. As no evidence exists for functional alterations in either peripheral or central cold thermoreceptors following short-term cold exposure (Hensel and Banet, 1978; Brück, 1986), modification in the neuronal structures involved in the central integration of thermoregulation, primarily the subcoeruleus region (SC) and nucleus raphe magnus (NRM) of the lower brain stem, has been postulated (Brück and Hinckel, 1982; Brück and Hinckel, 1984; Brück, 1986). In addition to the adaptation observed in shivering threshold, exposure to cold ambient temperatures (4° C) for 5 weeks has resulted in a reduction of the slope of heat production vs. body temperature curve in young guinea pigs (Brück and Hinckel, 1984; Hinckel et al., 1986). These changes in thermoregulatory heat production have been attributed to an increased inhibitory input from warm-responsive NRM units resulting from the repeated cold air exposures (Hinckel and Perschel, 1987).

In the present study, the effect of repeated cold water exposures on short-term adaptation of the metabolic thermoregulatory response following a sudden reduction in core temperature was investigated. Utilizing the pressure cuff occlusion of limb blood flow to effect a decrease in esophageal temperature, the central thermosensitivity to shivering thermogenesis was assessed during cold water immersions on 5 consecutive days. In addition, subjects with diverse morphological characteristics were selected in an attempt to determine the role of morphology in the variability observed in short-term adaptation of the thermogenic response to repeated cold exposures (Brück et al., 1976).

Methods

Five healthy males, with an average age of 29.2 years (range 24.7-33.8 years), consented to participate in the study after undergoing a medical examination. The subjects were informed of the experimental procedures as well as potential risks. All procedures were previously approved by the SFU Research Ethics Review Committee.

Anthropometry and Aerobic Fitness Assessment

Prior to the cold water immersions, complete anthropometric measurements were obtained and aerobic fitness was evaluated following the procedures outlined in Chapter 3. Assessment of aerobic fitness was conducted due to the possible influence of a "positive cross adaptation" between physical exercise and cold exposures reported by Baum et al. (1976). Physical characteristics of the subjects are shown in Table 4.1.

Water Immersions

The instrumentation and equipment described in Chapter 2 were utilized in the present study. Esophageal temperature (T_{es}) was measured with a YSI thermistor (Probe No. 702A, Yellow Springs Instrument Co., Yellow Springs, Ohio) inserted to a depth determined anthropometrically for each subject (see Chapter 3).

Five cold water immersions (15.80 ± 1.05° C, mean ± SD for all 25 trials) were conducted on consecutive days, with each trial carried out at the same time of day in each subject. The within-subject variation in water temperature during the experiment ranged from .13 to .56° C (SD). Subjects were asked to refrain from physical activity and food consumption for 4 hours prior to each immersion.

The occlusion of limb blood flow was carried out with the pneumatic device described in Chapter 2. When possible, pressure was applied to the limbs when T_{es} had decreased to 36.5° C. In those subjects whose T_{es} remained above 36.5° C (n = 3), the occlusion of limb blood flow was initiated after minute 40 of the experimental procedure. This time frame was selected to ensure that thermoregulatory responses following the release of the cooled limb blood flow could be evaluated over a sufficient period of time.

Subjects were removed from the water if T_{es} decreased below 35° C or upon completion of 1 hour of immersion. Rewarming was achieved by endogenous heat production in an insulated bed. The subject was allowed to voluntarily terminate the experimental procedure at any time.

Table 4.1: Physical characteristics of subjects

Subject No.	Height (cm)	Mass (kg)	Σ 8 Skf (mm)	A_D (m^2)	A_D:Mass ($m^2 \cdot kg^{-1}$)	Somatotype (N.D.)	$\dot{V}O_{2max}$ ($L \cdot min^{-1}$)	$\dot{V}O_{2max}$ ($ml \cdot kg \cdot min^{-1}$)
1	176.9	71.1	77.5	1.88	.0264	2.4-5.0-2.6	5.05	71.03
2	183.9	77.2	50.9	2.00	.0259	1.5-5.3-3.0	4.68	60.58
3	178.5	90.0	190.0	2.09	.0232	5.1-6.6-0.9	3.78	41.96
4	175.1	120.4	301.8	2.33	.0194	8.9-8.1-0.5	3.90	32.25
5	190.5	82.2	83.4	2.10	.0255	2.3-3.8-3.5	5.23	63.62

Data Analysis

Central thermosensitivity of metabolic heat production for a given thermoafferent drive was determined in the manner detailed in Chapter II. The slope of the H – T_{es} relationship (β) was evaluated following the return of the cooled limb blood flow to the core region during each immersion. A repeated measures ANOVA was used to compare β over the 5 immersions. Significance was established at the .05 level. Tukey's HSD test for comparison between daily mean values was used if an overall treatment effect was present.

Results

Repeated Immersion Responses-- Pre-Occlusion

Time course changes in T_{es}, heat production (H), and the unweighted averages of skin temperature ($\overline{T}_{sk}$) and heat flux ($\dot{Q}_{mean}$) are shown for each subject during the 5 immersions (Figures 4.1 to 4.5). Within each subject the pattern of change for each parameter was surprisingly uniform over all trials.

Data for pre-occlusion measurements obtained during the first and last immersions are shown in Table 4.2. Values represent the average of data collected over the same 5-minute period within each subject, just prior to the occlusion of limb blood flow. For all subjects $\overline{T}_{sk}$ and $\dot{Q}_{mean}$ were altered minimally by the repeated cold water exposures. The difference between the initial and final immersion values for $\overline{T}_{sk}$ was within .4° C while the difference in $\dot{Q}_{mean}$ was within 15 $W \cdot m^{-2}$.

Consistent data were also observed for the metabolic response. The elevation in H above its pre-immersion value was diminished between 30 and 60% by the final immersion for all subjects. Heart rate decreased between 4 and 25 beats per minute between days 1 and 5. By the final immersion for all subjects, the pre-occlusion value for HR was below its pre-immersion value. Although an elevation in the pre-immersion HR (> 5 $b \cdot min^{-1}$) between the first and final

Table 4.2: Comparison between initial and final values for pre-immersion and pre-occlusion responses

		Pre-Immersion			Pre-occlusion				
Subject No.	Immersion No.	T_{es} (°C)	H ($W.kg^{-1}$)	HR ($b.min^{-1}$)	T_{sk} (°C)	Q_{mean} ($W \cdot m^{-2}$)	$\Delta H^{\dagger}$ ($W.kg^{-1}$)	$\Delta T_{es}^{\dagger}$ (°C)	$\Delta HR^{\dagger}$ ($b.min^{-1}$)
1	1	36.57	1.71	56	16.99	197.43	2.77	+.21	+2
	5	36.77	2.39	57	16.61	212.68	1.10	+.34	-10
	Difference	+.20	+.68	+1	-.38	+15.25	-1.67	+.13	-12
2	1	37.12	2.23	69	16.97	211.01	2.34	-.53	-3
	5	37.38	2.69	83	17.23	208.40	1.40	-.92	-28
	Difference	+.26	+.46	+14	+.26	-2.61	-.94	-.39	-25
3	1	37.31	1.63	101	16.32	169.30	1.28	-.21	-14
	5	37.33	2.28	94	16.33	171.89	.90	-.06	-18
	Difference	+.02	+.65	-7	+.01	+2.59	-.38	+.15	-4
4	1	37.25	.85	61	18.95*	121.56	.28	+.03	+5
	5	37.20	1.71	62	18.86	132.27	.19	+.06	-6
	Difference	-.05	+.86	+1	-.09	+10.71	-.09	+.03	-11
5	2	36.47	2.19	66	17.79	387.87§	1.66	-.41	+8
	5	36.56	1.94	71	18.02	373.83	1.12	-.21	-7
	Difference	+.09	-.25	+5	+.23	-14.04	-.54	+.20	-15

† Δ = Pre-occlusion immersion value minus pre-immersion value.

* Mean water temperature for this subject was 17.73 (SD .34) °C.

§ Occlusion was initiated prior to stabilization of value.

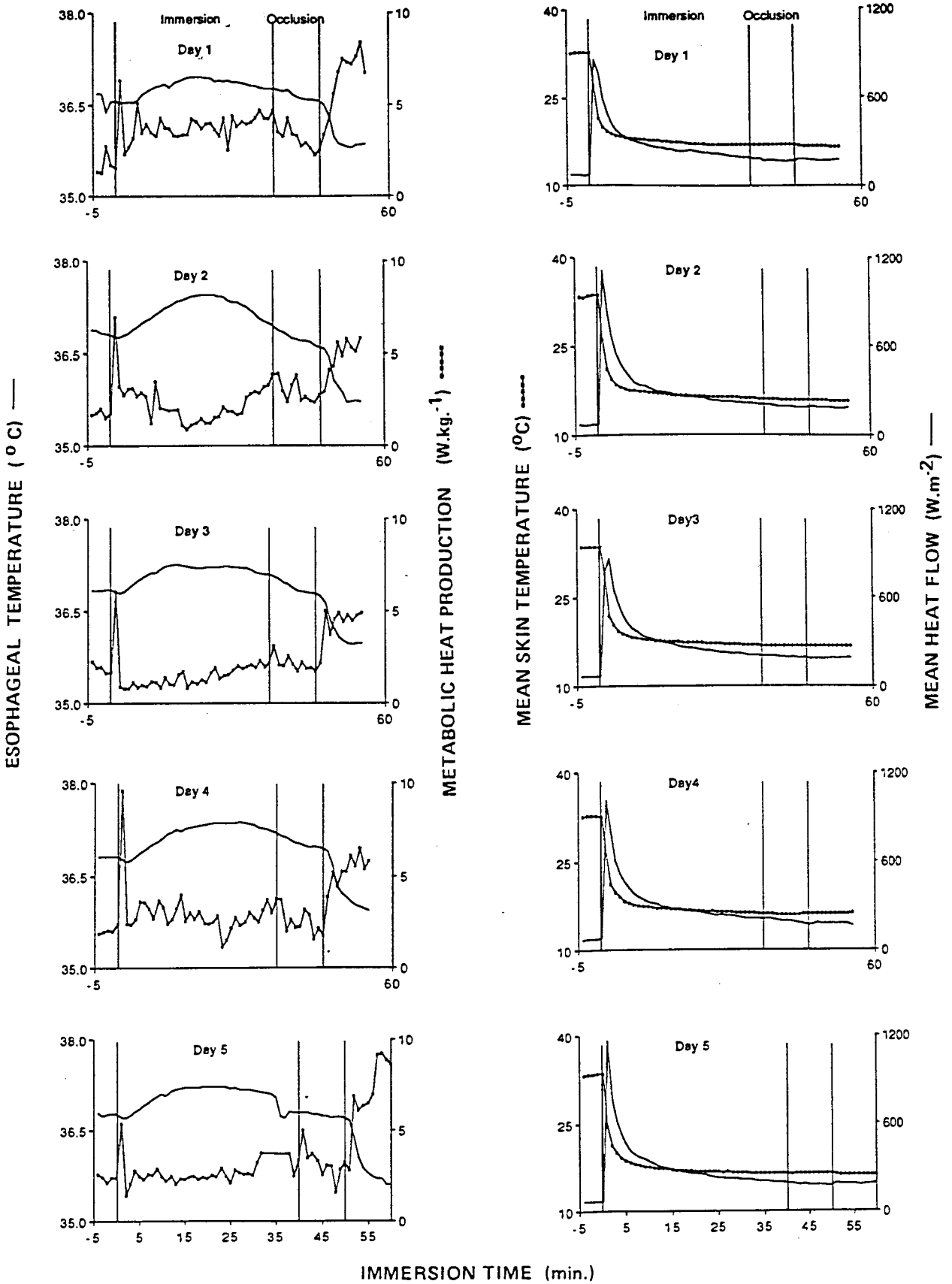

Figure 4.1: Responses of T_{es}, H, $\overline{T}_{sk}$, and $\dot{Q}_{mean}$ observed for subject 1 during 5 consecutive immersions.

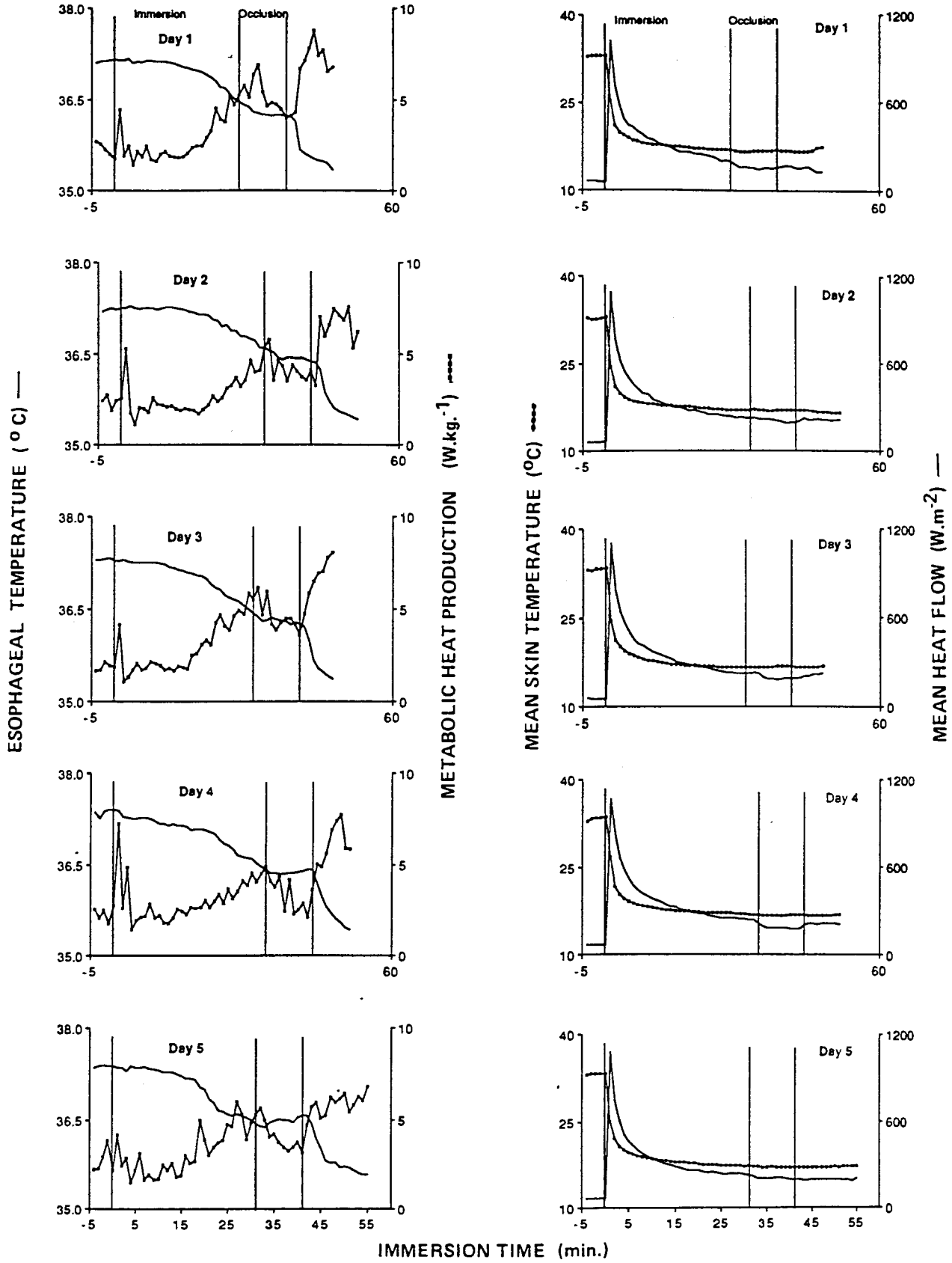

Figure 4.2: Responses of T_{es}, H, $\bar{T}_{sk}$, and $\dot{Q}_{mean}$ observed for subject 2 during 5 consecutive immersions.

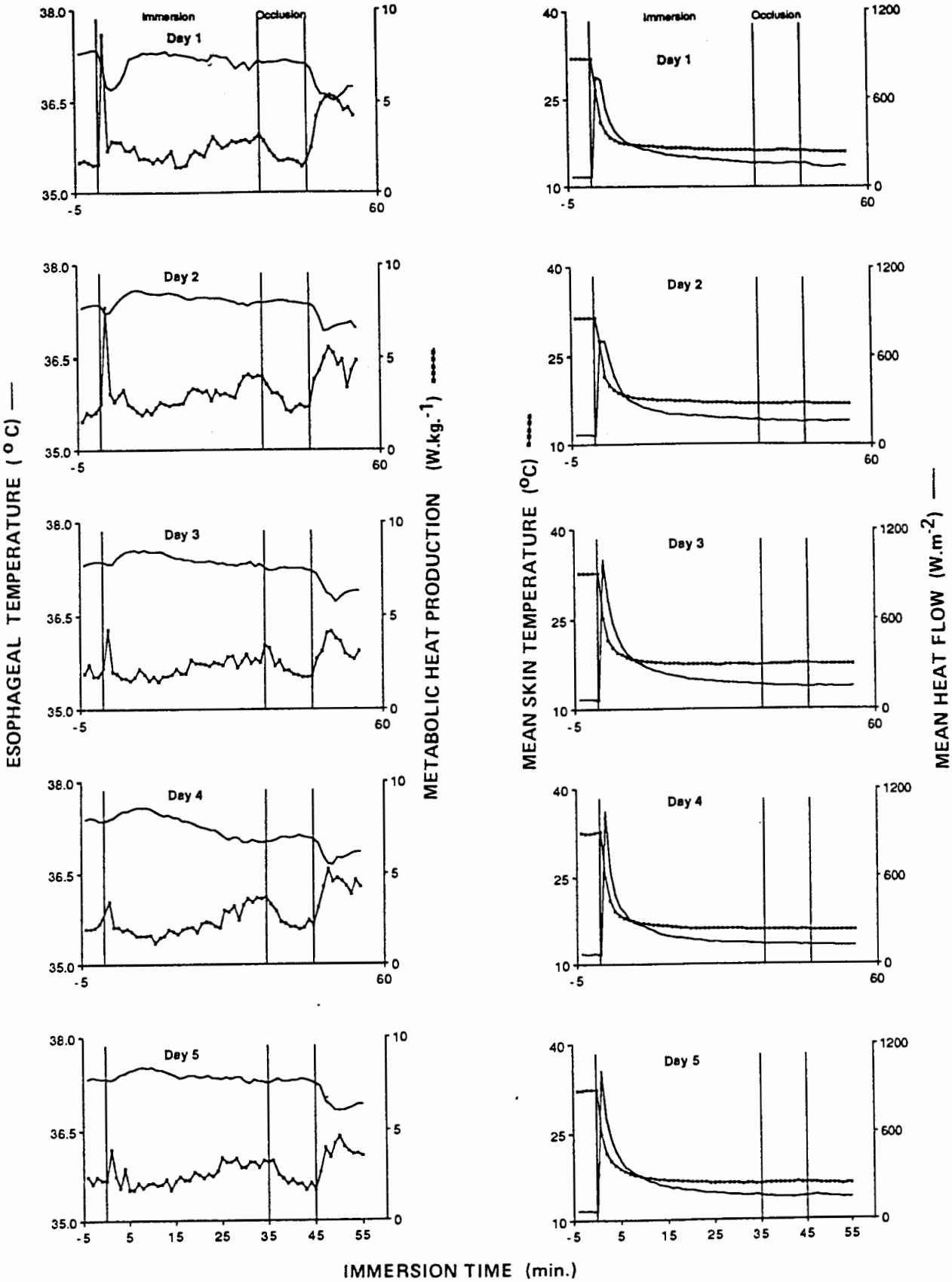

Figure 4.3: Responses of T_{es}, H, $\bar{T}_{sk}$, and $\dot{Q}_{mean}$ observed for subject 3 during 5 consecutive immersions.

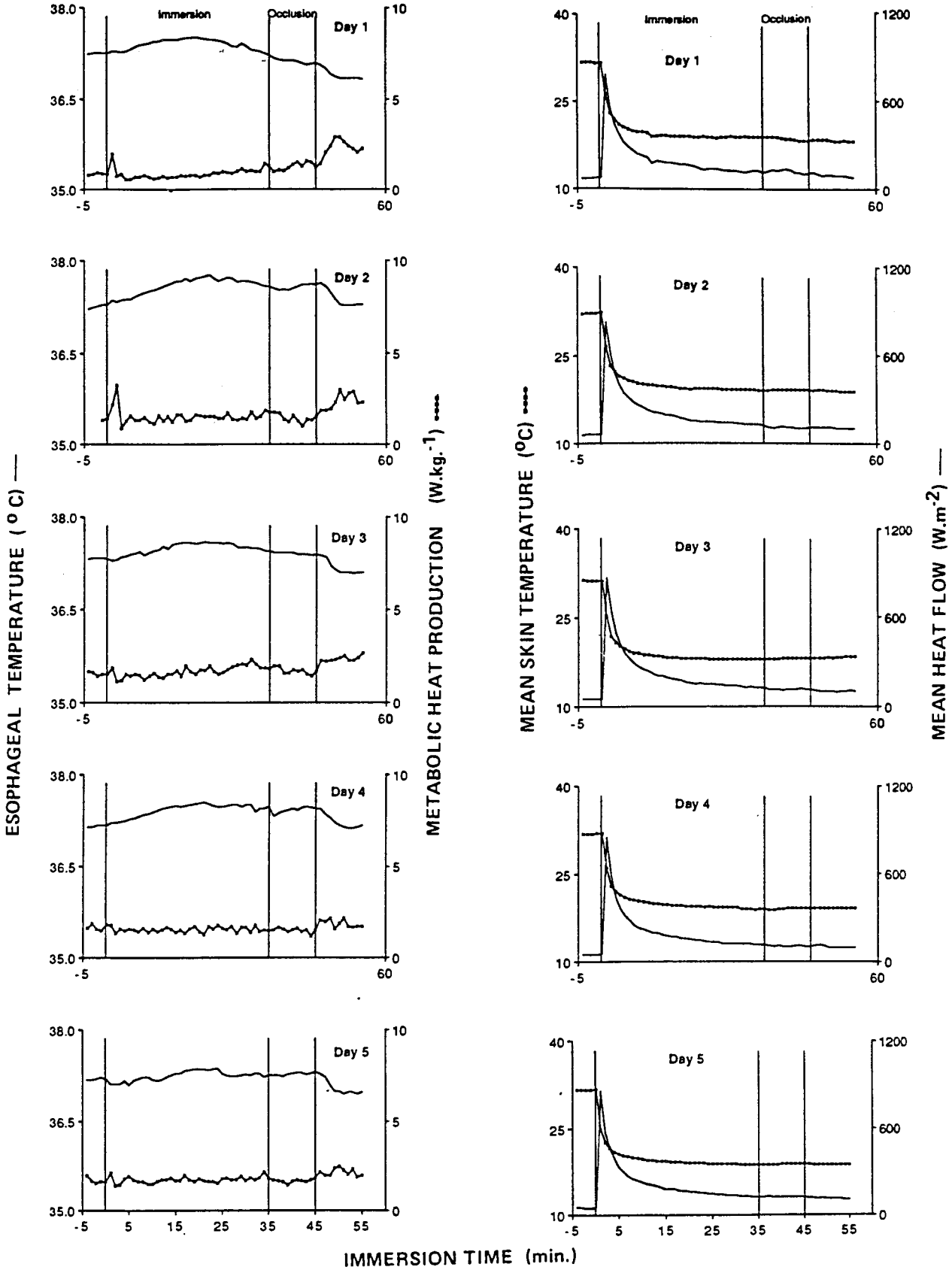

Figure 4.4: Responses of T_{es}, H, $\overline{T}_{sk}$, and $\dot{Q}_{mean}$ observed for subject 4 during 5 consecutive immersions.

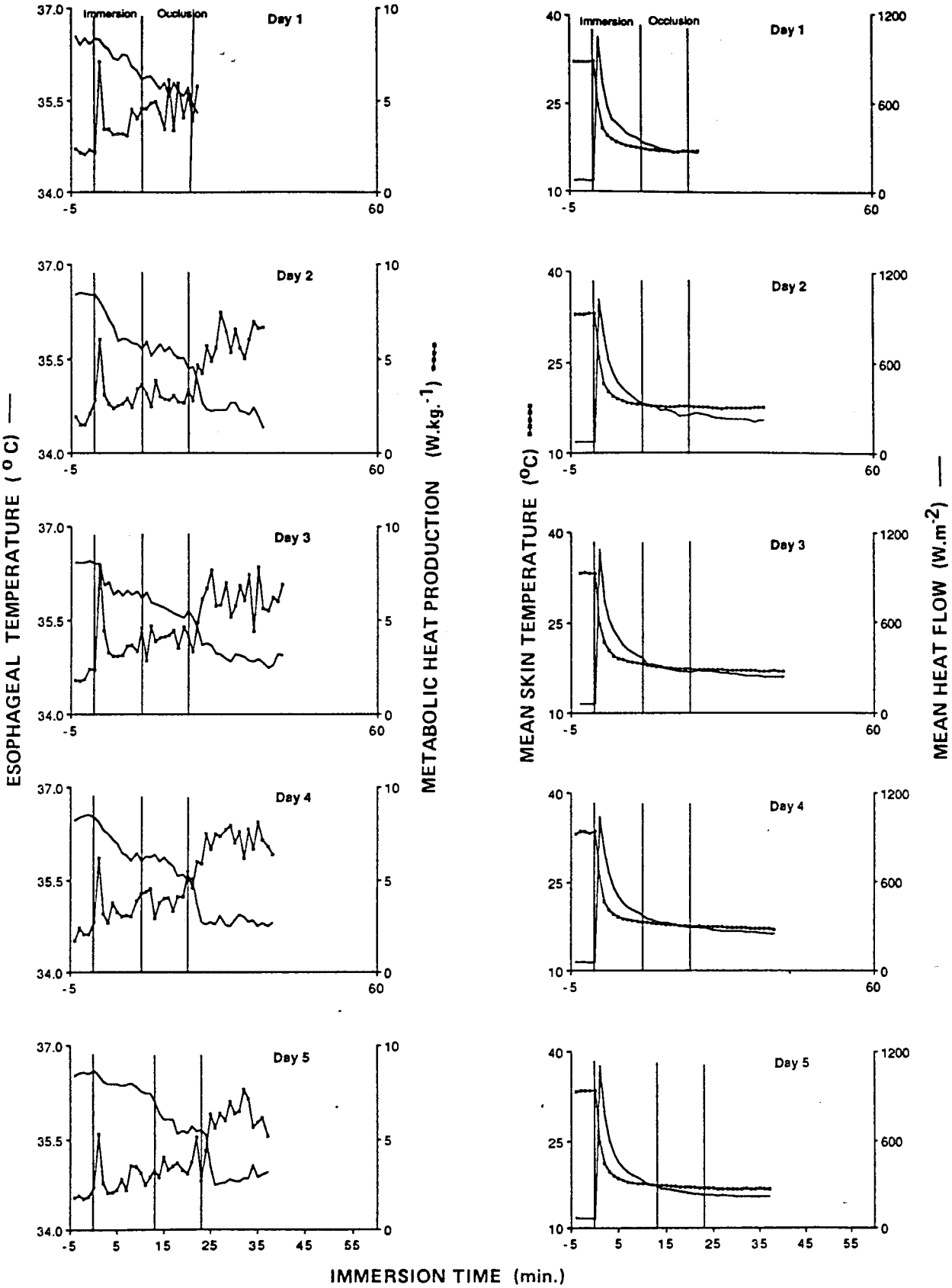

Figure 4.5: Responses of T_{es}, H, $\bar{T}_{sk}$, and $\dot{Q}_{mean}$ observed for subject 5 during 5 consecutive immersions.

immersion was observed in only 1 subject, pre-immersion metabolic rate increased in 4 of the 5 subjects. Subject 4 increased his pre-immersion metabolic rate 100% by the fifth exposure, however the depressed response observed during the initial immersion was increased 60% by the second measurement occasion (.85 vs. 1.37 $W \cdot kg^{-1}$).

Inter-subject variation in the T_{es} response was noted. Only subject 2 exhibited a greater decrease in T_{es} (.39° C) during the final trial. For 2 subjects the decrease in T_{es} was diminished (.15 and .2° C for subjects 3 and 5, respectively) while the remaining 2 subjects continued to exhibit a slight increase in T_{es} from its resting value. This decrease in thermal stress was likely a contributing factor in the reduction of the metabolic response cited above.

The consistency among subjects seen in the alteration (or lack of alteration) of thermoregulatory responses to repeated cold water exposures suggests a minimal role for morphological and physical fitness differences in modifying these changes. One possible exception was the lower T_{es} observed in the leanest subject.

Repeated Immersion Responses--Occlusion and Post-Occlusion

The pressure cuff occlusion of extremity blood flow resulted in variable responses between subjects although intra-subject differences during the 5 cold water immersions were relatively small (Figures 4.1 to 4.5). For 4 of the subjects T_{es} stabilized during the final 5 minutes of occlusion while a continuous decrease was noted for subject 5. Heat production during the blood flow occlusion either decreased or remained at its pre-occlusion value in 4 of the 5 subjects. The thermogenic response in subject 5 was increased as T_{es} decreased.

Following the release of cuff pressure, the return of the cooled venous blood from the extremities to the core region resulted in a dramatic reduction in T_{es} and a concomitant rise in H. However, the post-occlusion responses showed less uniformity within subjects than the pre-occlusion responses discussed above. Table 4.3 shows the changes in the post-occlusion T_{es}

over the five cold water exposures.[1] A greater decrease in T_{es} following the return of the occluded extremity blood flow was observed during the final immersion in 4 subjects. However, the rate at which T_{es} decreased ($\dot{T}_{es}$) was faster in only subjects 4 and 5. In 4 of the subjects the magnitude of the rise in H above its occlusion level was diminished between 25 and 56% whereas the final value for subject 5 was unchanged.

The slope of the H – T_{es} relationship (β) during the transient decrease in T_{es} following the return of cooled extremity blood to the core region is shown in Table 4.3 and Figure 4.6 for all immersions. The overall effect of the 5 repeated immersions on β was significant (F = 4.23 (4,12), $p < 0.05$). When differences between daily mean values were compared, only β observed on Day 3 was reduced significantly from Day 1. No other mean comparisons were significant. The response of subject 3 on Day 4 and subject 1 on Day 5 indicates lability in central thermosensitivity of metabolic heat production. A continuous decrease across the daily immersions was only observed for subject 4. Subject 5 exhibited no alteration in the β between days 2 and 5. The relationship between T_{es} and H observed during the initial and final immersion is shown in Figure 4.7 for all subjects. The Day 2 immersion for subject 5 was again used for comparison with the β evaluated on Day 5. Although a linear regression analysis was used for the determination of β, these graphs suggest a nonlinear relationship may also be present (S4, S5 in Figure 4.7). H on Day 5 in subject 4 was elevated almost 60% above the value for H observed on Day 1 which may reflect the pre–immersion increase discussed above (Table 4.2).

In terms of morphology, the subjects with the lowest and highest subcutaneous adiposity (subjects 2 and 4, respectively), determined from the sum of skinfold thickness, showed a reduction in the slope of β during the final immersion. However, in the present sample of young, healthy males neither fitness level nor morphological characteristics appeared to contribute to the variability in the short–term adaptation of central thermosensitivity of metabolic heat

[1] The day 1 immersion for subject 5 was terminated within two minutes of the release of cuff pressure, therefore day 2 immersion data are used for comparison with the final immersion data for this subject.

Table 4.3: Alteration in T_{cs}, H, and central thermosensitivity with repeated immersions

Subject No.	Immersion No.	Initial T_{cs} (°C)	ΔT_{cs} (°C)	$\dot{T}_{es}$ (°C·min^{-1})	ΔH (W·kg^{-1})	ß (W·kg^{-1}·°C^{-1})	r	n
1	1	36.56	-.77	-.265	4.96	-5.702	.979	7
	2	36.63	-.93	-.174	3.03	-2.826	.907	6
	3	36.78	-.83	-.177	2.91	-1.817	.589	8
	4	36.94	-.95	-.290	3.81	-1.837	.782	10
	5	36.70	-1.00	.267	3.71	-4.602	.822	9
2	1	36.23	-.70	-.270	3.49	-4.245	.838	10
	2	36.37	-.82	-.203	3.50	-1.960	.515	10
	3	36.26	-.90	-.234	2.91	-3.497	.973	7
	4	36.42	-.88	-.186	5.06	-2.945	.780	9
	5	36.57	-.87	-.191	2.62	-0.965	.565	8
3	1	37.11	-.45	-.204	3.61	-5.721	.987	6
	2	37.35	-.44	-.199	3.07	-3.053	1.000	3
	3	37.21	-.39	-.158	2.37	-2.976	.852	5
	4	37.06	-.44	-.168	2.77	-5.104	.935	4
	5	37.26	-.45	-.125	2.59	-3.659	.908	4
4	1	37.09	-.25	-.076	1.55	-6.282	.985	5
	2	37.63	-.36	-.089	1.43	-3.070	.897	5
	3	37.39	-.30	-.088	.99	-0.773	.860	6
	4	37.44	-.16	-.067	.70	-0.739	.411	6
	5	37.31	-.32	-.103	.68	-0.685	.511	4
5	2	35.35	-.68	-.234	3.48	-3.214	.828	4
	3	35.66	-.56	-.224	2.79	-3.275	.596	9
	4	35.50	-.75	-.361	3.58	-2.905	.860	4
	5	35.66	-.91	-.407	3.42	-3.006	.817	4

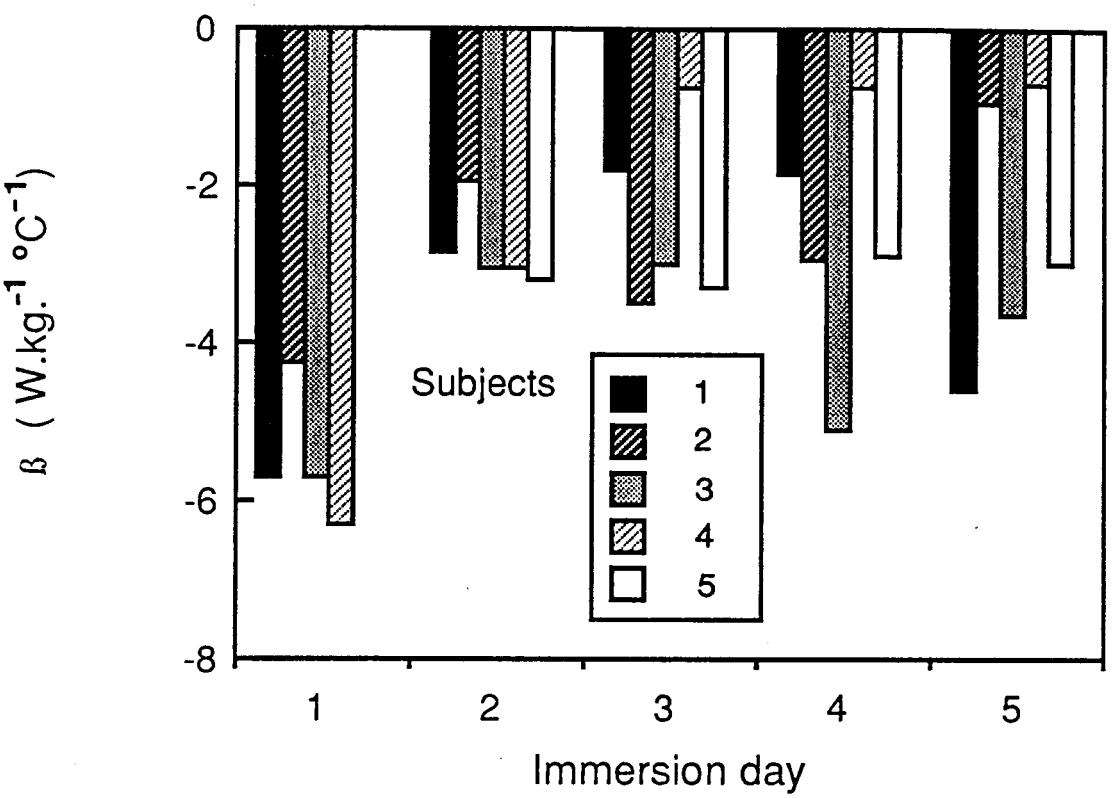

Figure 4.6: Individual changes in β during the five immersions. A significant decrease in β was observed over the 5 days in the 4 subjects evaluated on all occasions.

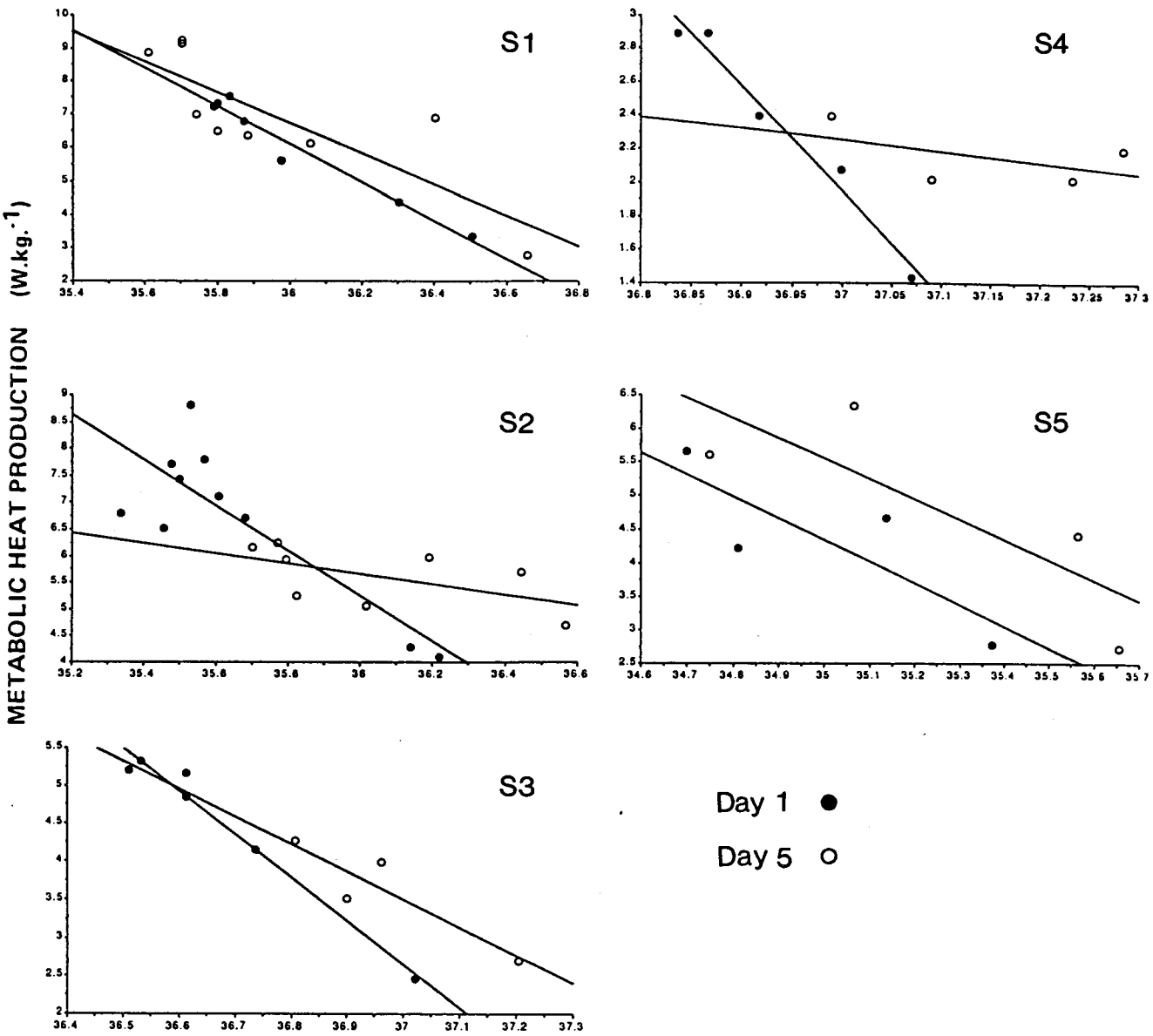

Figure 4.7: Comparison of β during the initial (Day 1) and final (Day 5) immersions for each subject. The Day 2 response of subject 5 was used as his Day 1 data were incomplete.

production during cold water immersion.

Discussion

The results of the present study indicate that repeated cold water exposures over 5 consecutive days does not produce an "insulative" adaptation as skin temperature and heat flux between the body surface and surrounding environment were unaltered between the initial and final immersions for all subjects. This is in agreement with previous literature suggesting that this type of response occurs after longer exposure periods (Skreslet and Aarefjord, 1967; Hong, 1973; Brück et al., 1976; Young et al., 1986).

Short-term adaptation in thermoregulatory responses following cold air exposures have included a reduction in shivering threshold as well as in oxygen uptake (Brück et al., 1976; Brück and Hinckel, 1984). A diminished sympathetic response to cold air exposure, determined from overnight urinary norepinephrine values, has also been observed following acclimation to 9 consecutive days of cold water immersion (Radomski and Boutelier, 1982). These alterations have been classified as "hypothermic" adaptation involving a habituation of the central integration of thermoregulation (LeBlanc, 1956; Hammel, 1964; Lange Andersen, 1965; Brück, 1986; Hinckel and Perschel, 1987). A reduction in the metabolic response, prior to the initiation of limb blood flow occlusion, was observed among all subjects in the present study (Table 4.2). However, only 1 subject exhibited a reduced T_{es} which is characteristic of hypothermic adaptation (LeBlanc, 1956; Lange Andersen, 1965; Baum et al., 1976; Brück, 1976). In 3 of the 5 subjects a slight reduction in the decline in T_{es} was evident. This alteration may have contributed to the diminished metabolic response, however it is unlikely that the 60% reduction in heat production from day 1 to day 5 in subject 1 was a result of the .13°C greater increase in T_{es} from its pre-immersion value. Somewhat surprising was an increase in metabolic rate prior to immersion which was evidenced in 4 of the 5 subjects. This response has been attributed to cold acclimatization in the Alacaluf Indians (Hammel, 1964) and in Korean ama (Hong, 1973), populations acclimatized to

cold ambient environments over long periods of time. In the present subjects, it is possible the increase in thermogenesis prior to immersion may reflect an anticipatory preparation of the ensuing cold stress which enabled the subject to increase heat storage, however esophageal temperature prior to immersion was increased greater than .10°C in only two subjects. Alternatively, Brück (1986) has reported that heavy physical training may result in an elevated resting metabolic rate for approximately 20 hours. The participants in this study were requested to refrain from heavy physical activity for four hours prior to the immersions, however activity level beyond the four hour period was not monitored. An additional non-thermoregulatory explanation for the increased pre-immersion metabolic rate may have been the stressful nature of the experiment which combined cold water exposure with the occlusion of extremity blood flow, both of which induced some degree of nociceptor activity based on the subjective response of the subjects. As epinephrine levels were not measured as an indication of enhanced anxiety (Gale, 1973), this explanation remains speculative.

In addition to the effect of repeated cold water immersion on the thermoregulatory responses observed prior to extremity blood occlusion, the effect of short-term adaptation on central thermosensitivity of metabolic heat production was also evaluated. Occlusion of limb blood flow for ten minutes during the immersion resulted in a decrease in T_{es} when the cooled trapped blood was released. The corresponding increase in heat production was analyzed in an attempt to quantify central thermosensitivity to shivering thermogenesis. Further discussion of the technique is presented in Chapter II. Immersion over 5 days resulted in a significant reduction in β for the 4 subjects evaluated on all occasions. This suggests the central integration of thermoafferent stimuli is easily adjusted in an attempt to diminish the effect of an environmental stressor. However, additional inputs enter into the integral process as can be observed in the β values determined on Day 4 for subject 3 and Day 5 for subject 1 (Figure 4.6). Each of these subjects expressed considerable feelings of anxiety on the immersion day on which the extreme values were observed.

The individual variability in short-term adaptation to repeated cold water immersions observed in the present experiments has also been observed in subjects exposed to cold air exposures (Brück et al., 1976; Radomski and Boutelier, 1982) and in divers exposed to sea temperatures as low as 2.5°C for 45 days (Skreslet and Aarefjord, 1967). A continuous decrease in the central thermogenic response during the course of the experiment was only observed in the subject with the greatest subcutaneous adiposity, however the overall results do not suggest morphological characteristics have a major influence on short-term adaptation. The effect of aerobic fitness on the adaptive response to short-term cold exposure also was not apparent. The importance of an individual's psychological profile has been suggested in the variability observed in the adaptation to cold air exposure (Brück et al., 1976; Thompson and Hayward, unpublished data). In addition genotypic factors may also play a major role in man's ability to adapt to cold stress (Hammel, 1964; Hong, 1973).

Results of the present study suggest that immersion in cold water (≃15°C) over 5 continuous days may lead to a diminished metabolic response during natural cooling conditions. In addition, the thermogenic response following the increased esophageal cooling was also reduced, however both inter- and intra-subject variability was apparent. Evidence of a hypothermic adaptation was not observed as a lower core temperature during a similar thermal perturbation was evident for only one subject. Individual variability in the adaptation to repeated cold water exposures does not appear related to aerobic fitness or morphological characteristics in the young, healthy males assessed in the present study. Future studies should focus on psychological and genetic factors governing the adaptive response.

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CHAPTER V

DAILY COLD WATER IMMERSION REDUCES THE VENTILATION AND OXYGEN UPTAKE RESPONSES AT ONSET OF EXPOSURE

Introduction

Sudden immersion in cold water elicits elevations in oxygen uptake ($\dot{V}O_2$, L·min^{-1}) and ventilation ($\dot{V}_E$, L·min^{-1}), which can be separated into two phases, as depicted in Figure 5.1. Coincident with a very rapid decline in mean skin temperature ($\bar{T}_{sk}$, °C) at onset of immersion is a marked transient elevation in $\dot{V}O_2$ and $\dot{V}_E$ reaching a peak within the first minute. This first phase response occurs in the absence of core temperature perturbations, and has therefore been attributed primarily to the stimulation of cutaneous cold receptors (Benzinger, 1976; Hayward and Eckerson, 1984; Mekjavić and Morrison, 1984; Mekjavić et al., 1987). Following the first phase response, $\dot{V}O_2$ and $\dot{V}_E$ decrease toward resting levels, followed by a second slower increase. This second phase increase in $\dot{V}O_2$ and $\dot{V}_E$ commences with the onset of core temperature cooling rate. As evident from Figure 5.1, $\bar{T}_{sk}$ is held fairly constant at a level several degrees higher than that of the water temperature during the second phase of the thermogenic response. Thus the elevation in $\dot{V}O_2$ in this region is attributed mainly to stimulation of core cold receptors (Benzinger, 1976; Mekjavić and Morrison, 1983). As the process of acclimation includes modulation of the thermogenic response, repeated immersion in cold water should alter the responses observed both in phase 1 and phase 2. Evidence presented in Chapter IV suggests that the central thermosensitivity of shivering thermogenesis is reduced, as a result of daily immersion in 15°C water. The present report focuses on the first phase responses of cold water immersion and assesses the effect of short-term adaptation on specifically the $\dot{V}_E$ and $\dot{V}O_2$ responses. Preliminary results have previously been reported (Mittleman and Mekjavić, 1987).

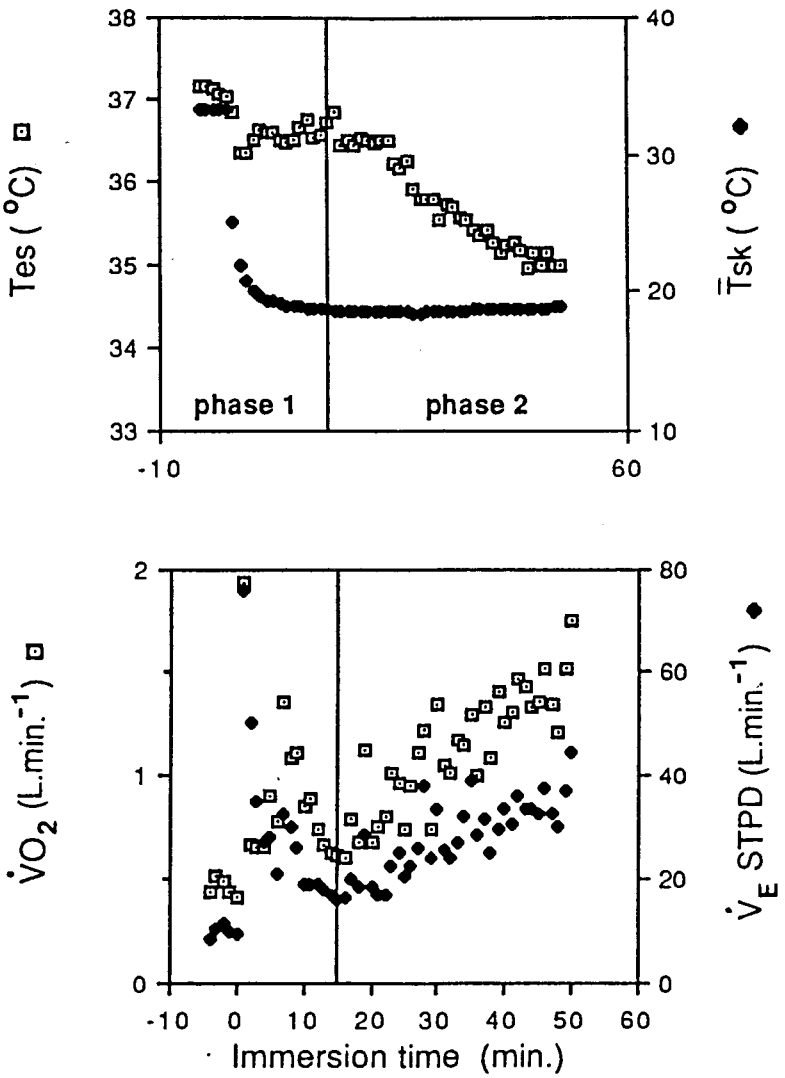

Figure 5.1: The oxygen uptake ($\dot{V}O_2$, L·min^{-1}) and ventilation ($\dot{V}_E$, L·min^{-1}) responses during cold water immersion for subject 1 in Chapter II. Two phases may be differentiated. Phase 1 is the response occurring immediately upon immersion and is associated with a rapidly decreasing skin temperature. Phase 2 occurs when skin temperature is stable and core temperature begins to decrease.

Methods

The data analyzed in the present paper were collected during the study described in Chapter IV. Group data are expressed as mean and standard deviation. As the homogeneity of variance assumption for parametric statistics was not met, a Wilcoxon Signed Rank Test was used to compare the onset $\dot{V}_E$ and $\dot{V}O_2$ values assessed during the Day 1 immersion with those evaluated on Day 5. Significance was established at the .05 level.

Results

Immediately upon immersion, skin temperature decreased to levels several degrees Celsius above water temperature and stabilized within five minutes. During this period core temperature did not exhibit any significant rate of cooling.

As evidenced from Figure 5.2, all subjects exhibited an instantaneous rise in $\dot{V}O_2$ and $\dot{V}_E$ at onset of immersion, indicated by the dashed line. Both $\dot{V}O_2$ and $\dot{V}_E$ decreased towards pre-immersion values, following the peak response attained during the first minute of immersion. As the present investigation focuses on the phase 1 responses, data from only the initial five minutes of immersion are presented in Figure 5.2.

Although the pattern of the $\dot{V}O_2$ and $\dot{V}_E$ response was similar for all immersions, the magnitude of the responses differed among the five subjects and also varied with successive immersions. Subject PL was the only subject that exhibited a decrease in the phase 1 responses with successive immersions. The remaining four of the five subjects demonstrate an enhancement in both $\dot{V}_E$ and $\dot{V}O_2$ responses following the immersion during day 1. The pattern of the acclimation of these responses varied among subjects, thus RW exhibits the highest peak values during immersion on the second day, subject NM during the third immersion, while both subjects DD and NT attained highest peak values during the penultimate trial.

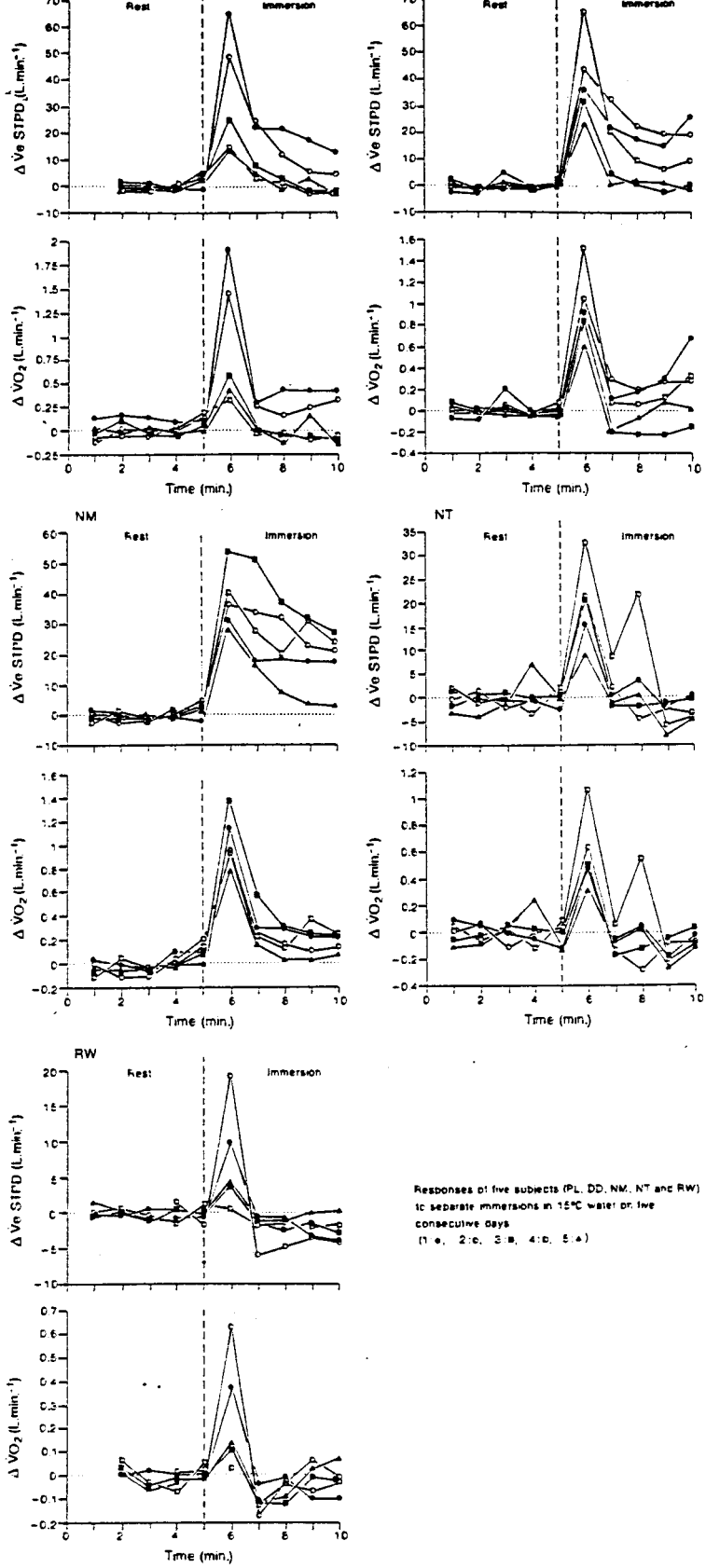

Figure 5.2: The relative elevations in oxygen uptake ($\Delta \dot{V}O_2$, L·min^{-1}) and ventilation ($\Delta \dot{V}_E$, L·min^{-1}) above pre-immersion level during the initial five minutes of immersion for five subjects (DD, PL, NM, NT, and RW). Each subject participated in five immersions conducted on consecutive days.

Regardless of the pattern of acclimation of the $\dot{V}_E$ and $\dot{V}O_2$ responses, there was an overall reduction in both responses over the five immersions. Comparison of the relative increases in $\dot{V}_E$ and $\dot{V}O_2$ above resting values between the first and last (fifth) immersion revealed a significant reduction (Wilcoxon Signed Rank Test, $p < 0.05$) of the $\Delta\dot{V}_E$ (from 31.7 ± 21.7 L·min^{-1} to 15.9 ± 9.9 L·min^{-1}) and $\Delta\dot{V}O_2$ (from 0.97 ± 0.62 L·min^{-1} to 0.46 ± 0.25 L·min^{-1}).

Discussion

Previously, Benzinger (1976), Hayward and Eckerson (1984) and Mekjavić and Morrison (1984) suggested that the responses of $\dot{V}O_2$ and $\dot{V}_E$ observed at the onset of immersion are thermogenic in nature. Results from a recent study by Mekjavić and Bligh (unpublished) support this hypothesis, but also indicates that ventilatory drive and oxygen consumption upon immersion are significantly altered by the pressure exerted over the body by the water itself, i.e. a hydrostatic component. Figure 5.3 depicts their observations of $\Delta\dot{V}O_2$ and $\Delta\dot{V}_E$ for a range of water temperatures (10°, 15°, 20°, 28° and 40° C) and indicates that the phase 1 $\dot{V}O_2$ and $\dot{V}_E$ responses are present during immersion in thermoneutral (28° C) and hot (40° C) water, thus under conditions of minimal cold receptor stimulation. They suggest that 40% of the $\Delta\dot{V}O_2$ response is attributable to a hydrostatic component (Arborelius et al., 1972) and the remainder to a reflexive component associated with the elevation in $\Delta\dot{V}_E$. Thus repeated immersion in 15° C water affects primarily the reflexive component reducing its magnitude towards the level of the hydrostatic component, as reflected in the responses to immersion in 28° and 40° C water. Further evidenced for this hypothesis is evidenced in the data from the present 5 subjects incorporated in Figure 5.3.

Observations from the present study do not support the suggestions of Cabanac et al. (1964), namely that the increase in $\dot{V}O_2$ is a result of the increased utilization of oxygen by the respiratory muscles, as the response is too rapid. Neither do the present results lend support to the suggestion that the response is mediated by shivering as shivering tremor is not evident

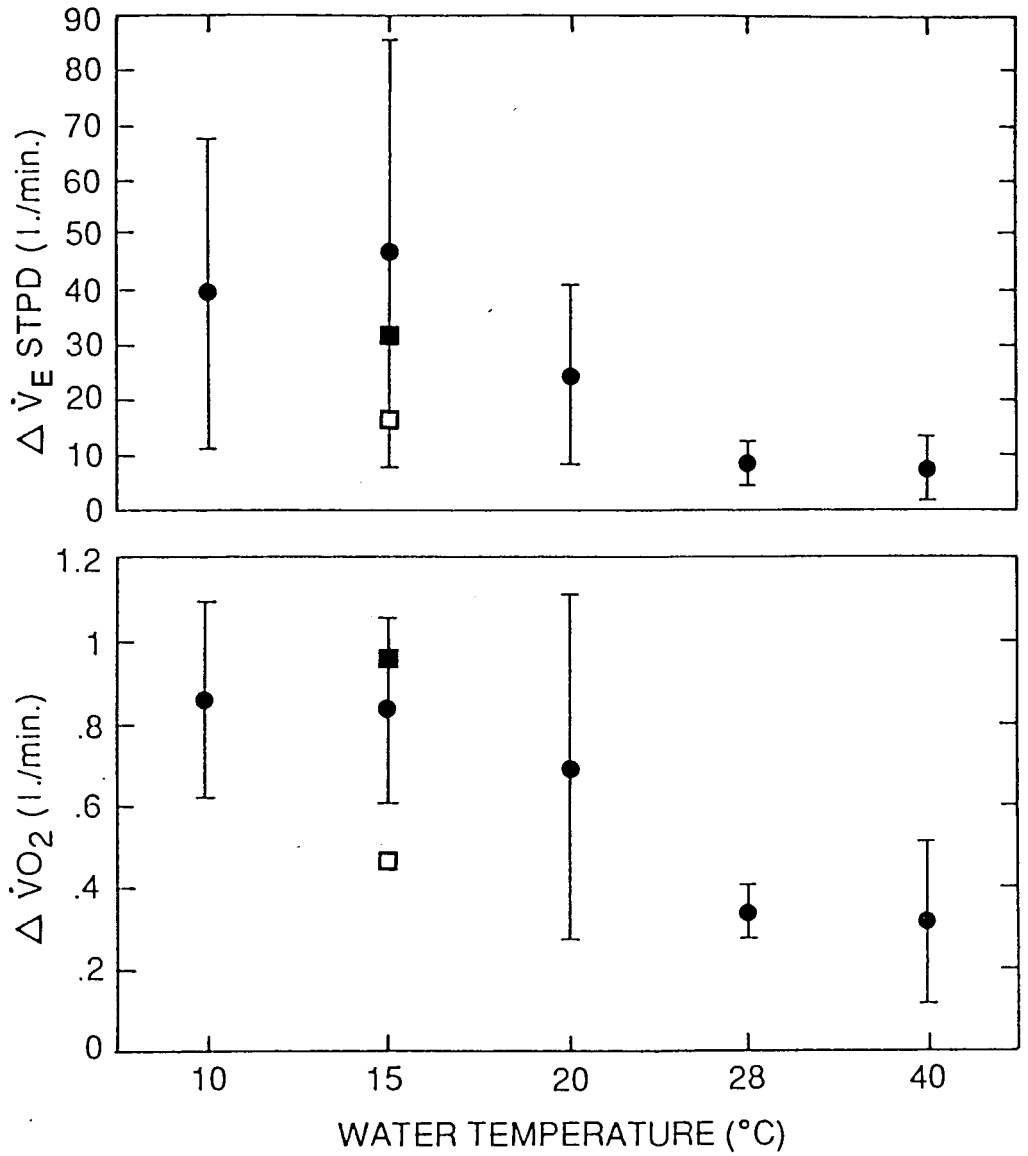

Figure 5.3: The relative elevations in oxygen uptake ($\Delta\dot{V}O_2$, L·min^{-1}) and ventilation ($\Delta\dot{V}_E$, L·min^{-1}) for immersions in 15°, 20°, 28°, and 40°C water, adapted from Mekjavić and Bligh (1987). Group data from the present study for the Day 1 immersion (solid square) and Day 5 immersion (open square) are also shown.

within the first minute of immersion. In part it is the momentary, but transient increase in venous return to the right side of the heart, combined with the increased ventilation that gives rise to a transient increase in oxygen uptake. Further evidence which contradicts support for an increased metabolic response at the onset of immersion is the transient increase in the respiratory quotient (R) above 1.0 during the initial phase of immersion seen in the present subjects and previously reported for subjects immersed in ice water (Hayward and Eckerson, 1984). Visual observation of subjects entering cold water suggests the presence of isometric muscle contractions upon water entry. It is perhaps this type of muscular activity, which may have a contributory role in the $\dot{V}O_2$ increases observed.

Several patterns of short-term acclimation were observed among the five subjects participating in the present study. The majority of the subjects exhibited a slight enhancement of the responses with repeated daily immersions in 15° C water. However, all subjects demonstrated a general diminution of the responses by the fifth immersion (day 5). Based on previous observations regarding the individual variability in $\dot{V}O_2$ (Mekjavić and Morrison, 1984) and respiratory (Mekjavić et al., 1987) responses at the onset of cold water immersion, the observed range of patterns of acclimation, reflected by the changes in the magnitudes of the $\dot{V}O_2$ and $\dot{V}_E$ responses during phase 1, may indicate individual variability thermoresponsiveness to peripheral cold stimulation.

The increase in $\dot{V}O_2$ response following the first trial is contrary to the decreased metabolic response following short-term adaptation to cold air exposure (Brück and Hinckel, 1984), however the increased stress of cold water immersion may alter this mechanism to some extent. All subjects had reduced responses by the 5th day of immersion which suggests short-term adaptation in the peripheral thermoafferent drive as the hydrostatic drive, a purely physical phenomenon, should not be altered (Figure 5.3).

Researchers conducting thermoregulatory studies, which require repeated immersions of each subject, are often faced with the dilemma of selecting the optimal time delay between exposures.

Although this study does not directly address this concern, it does indicate the degree of variability that may be expected with repeated immersions, in the responses observed during phase 1 of immersion.

Finally, as the "gasp" response ($\Delta\dot{V}_E$) has been implicated in drownings that have occurred during sudden cold water immersion (Cooper et al., 1976; Keatinge et al., 1969), the alteration in the peripheral thermal drive, as shown in present study, may be beneficial for workers constantly exposed to the threat of sudden accidental cold water immersion. A reduction of respiratory drive during initial immersion in cold water may enhance survival time during the phase 1 of the immersion.

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CHAPTER VI

EFFECT OF OCCLUDED VENOUS RETURN ON CORE TEMPERATURE DURING COLD WATER IMMERSION

Introduction

The potentially fatal consequence of hypothermia induced by cold water immersion has long been recognized (for reviews, see Golden and Hervey, 1981; Harnett et al., 1983). A continuous drop in core temperature following removal from cold water, commonly referred to as the "afterdrop", has been implicated as a major contributor to hypothermia-induced deaths which occur during cold water rescue (Burton and Edholm, 1955; Golden, 1973; Hervey, 1973; Harnett et al., 1983). Consequently, various rewarming methods have been investigated with respect to their ability to reduce the magnitude of the afterdrop (Morrison et al., 1982; Light et al., 1983; Hayward et al., 1984; Hoskin et al., 1986). Although the quantitative assessment of rewarming is paramount in determining the effectiveness of a given method, the physiological principles governing the afterdrop and its reversal must also be considered.

Based on evidence from early studies, a circulatory mechanism for the afterdrop phenomenon was proposed (Burton and Edholm, 1955). This theory postulates that upon removal of the victim from cold water, an increase in blood flow to the limbs, caused by physical movement or removal of thermally-induced peripheral vasoconstrictor activity, will cool the blood perfusing the cold peripheral tissues. The cooled returning blood will inevitably lead to cooling of the body's internal regions through the mechanisms of counter-current (blood to blood), convective (blood to tissue), and conductive (tissue to tissue) heat exchanges. Empirical evidence of diminished arterial pressure and total peripheral resistance during warm bath immersion following mild hypothermia, has led to the discouragement of rapid rewarming of the body surface to above 30°C and the promotion of active rewarming of the core region, using techniques such as inhalation rewarming (Hayward et al., 1984).

In recent years, the circulatory mechanism for the continued decline in core temperature has been challenged. Webb (1986), based on earlier evidence from an animal model (Golden and Hervey, 1977; Golden et al., 1977), demonstrated the physical principle of "thermal inertia", whereby an inanimate object's core region will continue to cool after it is removed from cold water, in a manner similar to the drop in core temperature observed in humans removed from cold water. This led the author to propose that the contribution of circulation in effecting the decrement in core temperature during rewarming was negligible, and that the afterdrop may be explained solely on the basis of conductive heat exchange. Although several researchers have reported data which supports a major role of conduction in contributing to the afterdrop in humans (Savard et al., 1985; Hoskin et al., 1986), these authors were unable to rule out conclusively the contribution of circulation to the afterdrop phenomenon.

In the investigation of thermoregulatory responses during cold water immersion, a series of experiments were conducted in which peripheral circulation, minimized by the combined effects of hydrostatic pressure (Arborelius et al., 1972) and intense cutaneous and skeletal muscle vasoconstriction (Barcroft and Edholm, 1943; Glaser and Holmes Jones, 1951; Keatinge and McCance, 1957; Keatinge et al., 1964; Major et al., 1981; Savard et al., 1985), was occluded during the immersion. The response of core temperature following the return of the cooled limb blood to the core region, suggests that the circulatory component was effective in reducing esophageal temperature, a measurement representative of cardiac temperature (Cooper and Kenyon, 1957; Wyss et al., 1974; Hayward et al., 1984). Preliminary results have previously been reported (Mittleman and Mekjavić, 1987).

Methods

The data analyzed in the present paper were collected during the studies described in detail in Chapters II and III. Physical characteristics of the subjects are shown in Table 6.1.

Data Analyses

Cooling rates ($^{\circ}C \cdot min^{-1}$) for esophageal ($\dot{T}_{es}$) and rectal ($\dot{T}_{re}$) temperature were determined during three phases of the immersion: 1) 5 minutes just prior to application of cuff pressure ($Pre\dot{T}_{es}$, $Pre\dot{T}_{re}$); 2) 10 minutes of blood flow occlusion ($Occ\dot{T}_{es}$, $Occ\dot{T}_{re}$); and 3) Initial 5 minutes following the return of the trapped blood to the core region ($Post\dot{T}_{es}$, $Post\dot{T}_{re}$). Nonlinear core temperature responses were observed during the occlusion and post-occlusion periods, thus cooling rate was calculated from the average change in temperature determined each minute over the 3 immersion phases. During the pre-occlusion phase in which cooling rate was linear, the averaged cooling rate was similar to the slope of core temperature with respect to time as determined by least squares linear regression analysis ($\dot{T}_{es}$: $-.017^{\circ}C \cdot min^{-1}$ (average) vs. $-.018^{\circ}C \cdot min^{-1}$ (slope); $\dot{T}_{re}$: $-.028^{\circ}C \cdot min^{-1}$ (average) vs. $-.027^{\circ}C \cdot min^{-1}$ (slope)).

Differences in the average rates of cooling between the core temperature sites ($\dot{T}_{es}$ vs. $\dot{T}_{re}$) and over the 3 immersion phases (Pre, Occ, Post) were analyzed for statistical significance ($p < 0.05$) using a multifactor repeated-measures analysis of variance program (ANOVA, Dixon, 1983). Linear trend analysis (Winer, 1971) was used to evaluate the relationship between levels when a significant main effect or interaction was observed. Mean values and standard deviations are reported for group data.

Table 6.1 Physical characteristics of subjects

Variable	Mean ± SD
Age (yrs)	29 ± 3.3
Height (cm)	179.7 ± 9.0
Mass (kg)	79.4 ± 13.0
A_D (m^2)	1.98 ± 0.16
Σ 6 SF (mm)	75.6 ± 49.9

Results

Immersion Responses — Pre-Occlusion

The response of T_{es}, T_{re}, skin temperature and heat flux from the lower back (T_{back} and $\dot{Q}_{back}$) and medial calf (T_{calf} and $\dot{Q}_{calf}$) are shown in Figure 6.1 for one subject. At the onset of immersion for all subjects, unweighted mean skin temperature ($\overline{T}_{sk}$) decreased in a nonlinear manner from 32.95 ± .94 °C to a constant value of 17.96 ± 1.03 °C. In contrast, unweighted mean heat flux ($\dot{Q}_{mean}$) exhibited a marked transient increase to over 1000 $W \cdot m^{-2}$ from its pre-immersion value of 58.51 ± 8.50 $W \cdot m^{-2}$ and eventually decayed with increasing levels of vasoconstriction to 246.48 ± 85.16 $W \cdot m^{-2}$. Whereas skin temperatures stabilized within five to ten minutes of immersion, heat flux attained relative stability around twenty minutes of immersion, although 80% of its decay occurred within 10 minutes.

Common in all subjects was a higher pre-immersion value for T_{re} compared with T_{es} (37.21 ± .24 vs. 36.92 ± .28 °C). Prior to the initiation of extremity blood flow occlusion, both T_{es} and T_{re} decreased as a result of the extreme cold stress. Although the onset of a continuous rate of cooling was delayed in T_{re} (Figure 6.1), $Pre\dot{T}_{es}$ was lower than $Pre\dot{T}_{re}$ just before occlusion (Table 6.2). The variation in pre-occlusion cooling rates among subjects is attributed to differences in morphology and thermogenic drives as discussed in Chapters II and III.

Immersion Responses — Occlusion and Post-Occlusion

Ten minutes of occlusion of blood flow to the extremities and subsequent release did not result in any substantial alteration in the $\dot{Q}_{mean}$ response or the regional heat flux as seen in Figure 6.1. In subjects in whom intense shivering was observed prior to inflation of the pressure cuffs, a slight reduction in $\dot{Q}_{mean}$ was evident during occlusion which returned to its pre-occlusion level upon deflation of the cuffs.

Extremity blood flow occlusion resulted in a 48% decrease in $\dot{T}_{es}$ while $\dot{T}_{re}$ decreased 18% (Table 6.2). The rate of change in core temperature at both sites increased substantially when the

Table 6.2 : Comparison of cooling rates (mean ± SD)

Cooling Phase	$\dot{T}_{es}$ ($°C \cdot min^{-1}$)	$\dot{T}_{re}$ ($°C \cdot min^{-1}$)
Pre-Occlusion	-.017 ± .033	-.028 ± .019
Occlusion	-.009 ± .022	-.023 ± .014
Post-Occlusion	-.149 ± .052	-.050 ± .026

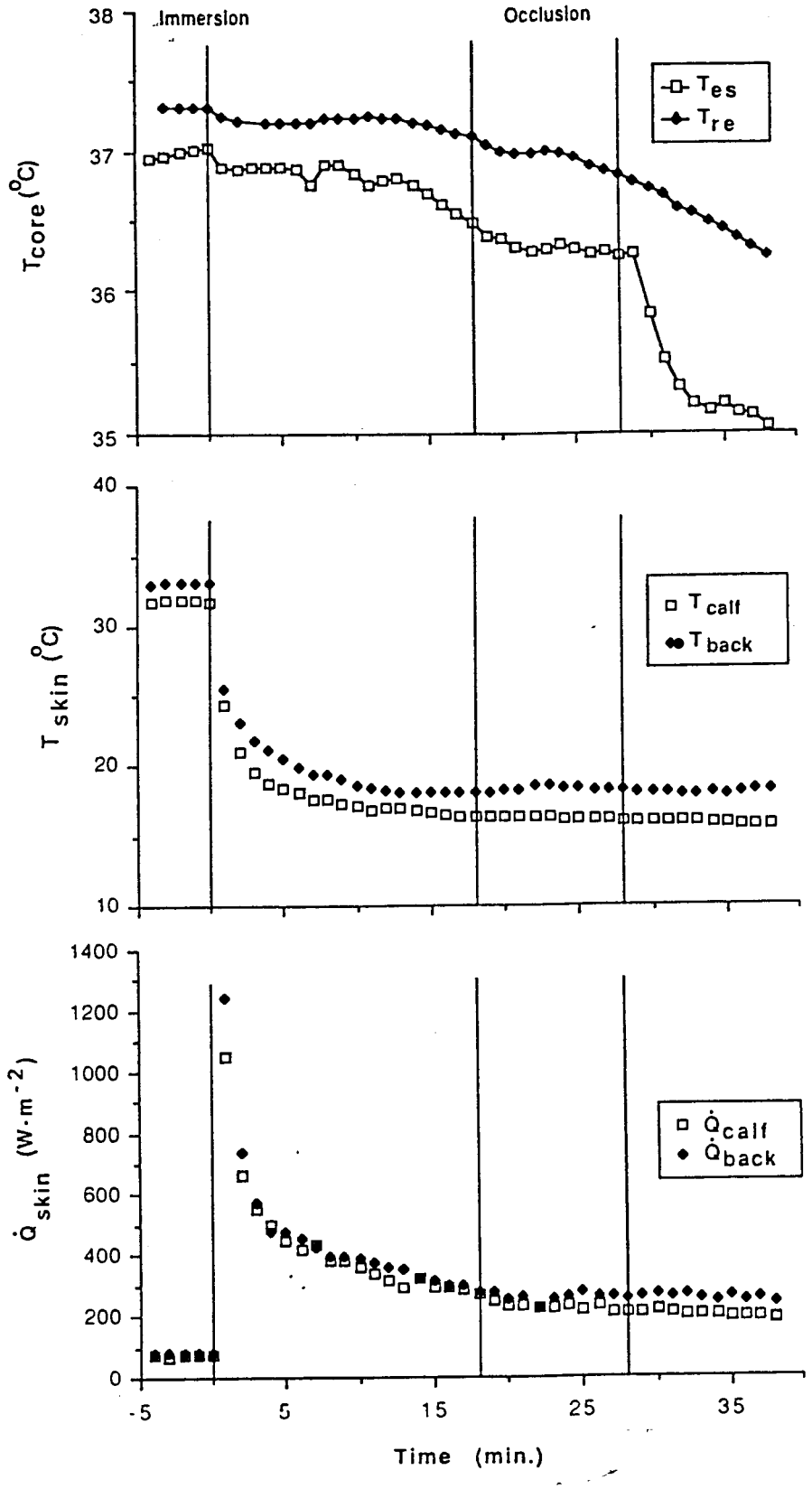

Figure 6.1: Physiolgical responses observed during cold water immersion in subject 1 from Chapter II.

cooled extremity blood was recirculated to the core region as shown in Table 6.2. The change in T_{es} and T_{re} from its pre-occlusion value is depicted in Figure 6.2.

A significant difference was evaluated between $\dot{T}_{es}$ and $\dot{T}_{re}$ (F = 25.34 (1,16), p <.001) as well as between the assessment periods (F = 77.57 (2,34), p <.001). An interaction between the two effects was also significant (F = 45.36 (2,34), p <.001). Linear trend analysis indicated that a significant portion of the total variance could be explained by both a linear (46%) and a quadratic component (21%) for the effect of assessment period. A significant contribution of a quadratic component in the interaction between the temperature measurement site and the assessment period (9%) was also determined. This diversion from linearity as well as the interaction between the two measurements of core temperature can be seen in Figure 6.2. The cooling rate for both T_{es} and T_{re} was significantly increased during the post-occlusion phase with the increase in $\dot{T}_{es}$ significantly greater than the increase measured in $\dot{T}_{re}$.

Discussion

In the present study, core temperature responses were evaluated during cold water immersion following the alteration of blood circulation by pressure cuff occlusion of peripheral blood flow and subsequent release of occluded cooled, extremity blood back to the general circulation. Although the "afterdrop" associated with rewarming was not directly studied, these results provide insight into the mechanisms contributing to core temperature cooling.

Immersion in cold water (approximately 15° C) resulted in a reduction in both esophageal and rectal temperature. Based on the large temperature gradient between the skin and the core, conduction would provide a primary avenue of heat exchange. However, with an alteration in circulation during cooling caused by blood flow occlusion to the limbs in the present study, a modified picture emerges. Esophageal temperature, representative of cardiac temperature (Cooper and Kenyon, 1957; Wyss et al., 1974; Hayward et al., 1984; Brengelmann, 1986), was affected by the occlusion procedure to a greater extent than rectal temperature. During the ten minutes of

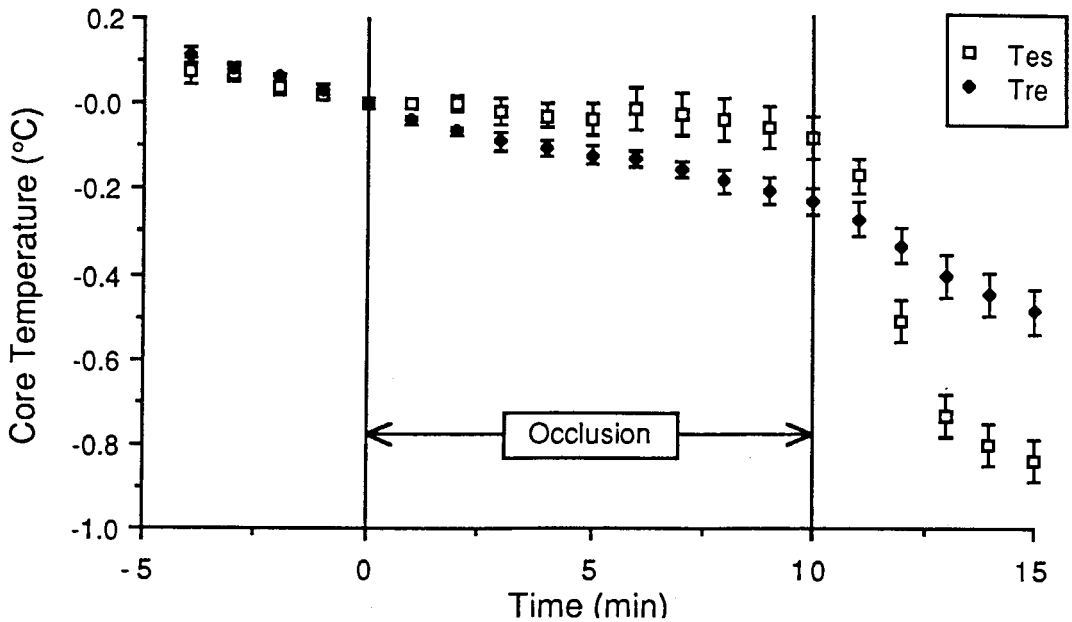

Figure 6.2: A comparison of the Mean ± SD responses of esophageal (T_{es}, °C) and rectal (T_{re}, °C) temperatures observed prior to, during, and following occlusion.

extremity blood flow occlusion, the rate of cooling observed for T_{es} was significantly arrested ($Occ\dot{T}_{es}$ = −.009 °C·min^{-1}). In contrast, rectal temperature was reduced, but not arrested ($Occ\dot{T}_{re}$ = −.023 °C·min^{-1}). Upon deflation of the cuff pressure, the cooled, trapped blood returning to the core region resulted in a large decrease in esophageal temperature and a smaller decrease in rectal temperature.

The differential response observed for post-occlusion rectal and esophageal cooling rates may be explained by the disparity in the direct effect of perfusion in these core regions which has been reported to occur during exercise (Nielsen and Nielsen, 1962). The large heat capacity of the tissues surrounding the rectum, combined with the shunting of blood from the lower body due to the hydrostatic effect of water immersion (Arborelius et al., 1972), will emphasize the reduced role of circulation in determination of rectal temperature. This supports a previous suggestion that rectal temperature is not a relevant measurement of core temperature during rewarming from hypothermia (Hayward et al., 1984). A diminished transient response and increased lag time observed in rectal temperature to changes in core temperature (Carlsten and Grimby, 1958; Cooper and Kenyon, 1957; Cranston et al., 1954; Gerbrandy et al., 1954; Stupfel and Severinghaus, 1956) is also evident from the present results.

Although the importance of circulating blood in determining the esophageal temperature response to cooling is evident from the above discussion, the importance of conduction has been demonstrated in the afterdrop phenomenon (Golden and Hervey, 1977; Savard et al., 1985; Hoskin et al., 1986; Webb, 1986). Savard et al. (1985) measured forearm blood flow during cold water immersion and rewarming and noted that the continued drop in core temperature following immersion was evident prior to any increase in blood flow measured in the distal limb. However, their results do not rule out an effect mediated by circulating blood for several reasons. First, the technique for measuring forearm blood flow is extremely sensitive to movement (e.g. shivering, voluntary muscle contractions) or to changes in the forearm circumference (Brengelmann and Savage, 1986) which may occur as a result of an alteration in the peripheral tissues by immersion

in cold water. In addition, Johnson et al. (1986) have recently shown a very small change in skin blood flow may greatly increase heat exchange capacity over the entire body. A slight increase in forearm blood flow was observed by Savard et al. (1985) upon initiation of rewarming, but these authors suggested it was minimal and could therefore not account for the afterdrop observed. Finally, accounting for the afterdrop by a purely conductive mechanism could not explain the dramatic increase in cooling rate observed when subjects were removed from the cold water and placed in a warm bath as reported by Savard et al. (1985, Figure 2).

Although conduction has been proposed as the primary factor contributing to the afterdrop, Savard et al. (1985) and Hoskin et al. (1986) have suggested that the continued drop in core temperature following cold water immersion may be partly explained by returning blood from skeletal muscle initiated by movement from the water to the location for rewarming. Evidence for the contribution of both conduction and circulation to the afterdrop has been reported in a study of older subjects (Collins et al., 1982). These authors observed a conductive process of heat exchange during rewarming in subjects diagnosed with poor peripheral vascular control, while subjects with normal vasomotor responses had an increased rate of cooling due to the contribution of the returning circulation.

The present results confirm the contribution of cooled limb blood flow to a decrease in core temperature, especially within the esophagus. As esophageal temperature has been shown to closely follow both pulmonary arterial and hypothalamic temperatures (Brengelmann, 1987), its measurement is extremely important and useful in characterizing thermoregulatory responses.

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CHAPTER VII

SUMMARY

A series of 45 cold water immersions were designed to elucidate the human thermogenic response to core temperature cooling at a constant peripheral temperature and the influences of cooling rate, morphology and short-term adaptation on this response.

In order to ensure a common peripheral and central thermal drive condition, limbs were occluded during immmersion to allow the trapped blood to cool toward the temperature of the surrounding tissues. Upon release of the occlusion, return of the extremity blood flow back to the core region introduced a controlled decrease of temperature measured in the esophagus and rectum. The concomitant rise in shivering thermogenesis, measured indirectly from oxygen uptake, was evaluated. This technique provided a basis for monitoring individual differences in the thermogenic response to similar peripheral and core temperature stimuli.

Based on the results in the previous chapters the following conclusions appear warranted:

Instrumentation

1. The use of the pressure cuff occlusion technique was successful in creating a similar condition of a rapid decrease in core temperature measured by esophageal and rectal temperatures. Thus the thermogenic response concomitant with the decrease in core temperature allowed the quantification and comparison of central thermosensitivity of metabolic heat production among individuals.

2. Esophageal temperature showed a more dramatic response to the occlusion technique than rectal temperature. This result has implications in monitoring the effects of hypothermia as esophageal temperature may be more sensitive to changes in blood temperature and ultimately heart temperature. The effect of the recirculation of the cooled extremity blood

flow following the release of occlusion on esophageal temperature suggests that this region is sensitive to dynamic changes in circulation which may also be operative during rewarming from hypothermia.

Core Cooling Rate

3. The thermogenic response to a decrease in core temperature is a function of both the absolute temperature and its rate of change. Central thermosensitivity of metabolic heat production showed a intra-subject specificity.

4. In comparing the thermogenic response to cooling among different subjects or between repeated trials of the same subject, monitoring peripheral and core temperatures and their derivatives (T_{sk}, $\dot{T}_{sk}$, T_c, $\dot{T}_c$) are necessary to verify experimentally controlled conditions are met or to account for their uncontrolled effects.

Morphology

5. While it is well-established that morphological characteristics may effect body heat loss during cold water exposures, this thesis demonstrates that the thermogenic response to a common peripheral and central thermal drive, induced by the occlusion technique, is independent of these characteristics within the range of subjects examined. Furthermore, central thermosensitivity does not appear to be related to maximal aerobic power.

6. A corollary of the above observation is that studies addressing thermoregulatory responses to cold water immersion must take into account a morphologically-independent, individual thermogenic sensitivity.

Short-term Adaptation

7. The experience of replicated exposure (5 consecutive days) to conditions of cold water immersion was shown to reduce oxygen uptake and ventilatory responses at the onset of immersion by the final day. To this extent, integration of cutaneous thermoafferent stimuli may be thought of as having some aspects of a learned behavior. However only 1 subject exhibited continuous daily reduction in these onset responses.

8. During immersion prior to occlusion, all subjects showed a reduction in heat production as a result of 5 days of repeated cold exposure. This suggests an increased efficiency in maintaining core temperature as the metabolic cost was reduced.

9. Central thermosensitivity of metabolic heat production, assessed following the occlusion technique, was shown to be reduced following immersion in 15° C water on 5 consecutive days. Emotional factors were shown to influence the thermogenic response to a repeated temperature stimulus.

10. The pattern of adaptation of the peripheral and central thermosensitivities exhibited a great deal of variation among individuals.

Individual differences

11. While contemporary thermoregulatory models may provide some indication of cold-induced metabolic heat production, they do not obviate the need for comprehensive assessment of individual central thermosensitivity.

Recommendations

As often occurs during the investigation of a specific problem, a number of related questions arise. The following ideas for future research are suggested:

1. The present study has demonstrated the variability of central, and to a smaller degree peripheral thermosensitivity among a group of morphologically different people. Individual variability might be demonstrated more clearly by evaluating central and peripheral thermosensitivity in a number of subjects with very similar anthropometric characterisitics rather than very different ones.

2. The cuff occlusion technique was successful in introducing a rapid, quantitative, standard decrease in core temperature. The variability in the physiological responses observed during occlusion as well as following the release of cuff pressure suggests however that different mechanisms may have contributed to the ultimate reduction in esophageal temperature. Further investigation of this technique is warranted.

3. Kollias et al. (1974) investigated thermoregulatory responses in 5 obese subjects following a weight reduction program. Although the thermoregulatory heat production and core cooling was not altered as a group, the body composition changes were not dramatic. The one subject who lost 12.3 kg showed a greater thermogenic response and an increased tissue conductance. Whether central or peripheral thermosensitivity may be altered by such modification is open to evaluation.

4. The question of whether peripheral and central thermosensitivity are inherited characteristics has been raised in the present thesis. Evidence to address this query is as yet unavailable. The present study has not observed a large influence of morphology on central

thermosensitivity of shivering thermogenesis. This suggests that the morphology acquired by the subject during his lifetime has not had a strong influence on peripheral and central thermogenic responses (in the present group of subjects), assuming no previous acclimatory stresses. Further work is necessary to elucidate the interaction between morphology and acclimation on peripheral and central thermosensitivity.

5. Within subjects (Chapter II), β has been determined for natural cooling and within a similar core temperature range, where a rapid core cooling was instigated with the cuff occlusion method. Core sensitivity should be examined for several rates of cooling, perhaps by decreasing occlusion time. The effect of skin temperature could also be assessed by conducting the trials in different water bath temperatures.

6. Analysis of the onset (phase 1) response of $\dot{V}O_2$ and $\dot{V}_E$ supports previous work suggesting that there may exist a hydrostatic component to the responses. Thus further analysis is required to clearly define the method of determining α. It is suggested that in addition to an immersion in cold water for the determination of α and β, subjects be immersed (on a separate occasion) in a bath at core temperature (37° C) to assess the influence of the hydrostatic force of the water on the skin during the phase 1 response and to quantify the contribution of hyperemia to the phase 2 response.

7. The short-term adaptation observed in the $\dot{V}O_2$ and $\dot{V}_E$ responses upon initiation of cold water immersion suggests that individuals at risk of accidental immersion may benefit from the "learning effect" of short-term repeated exposures.

8. The relative importance of limb vs. trunk insulation during cold water immersion as well as limb vs. trunk muscle in shivering thermogenesis should be evaluated in greater detail.

APPENDIX A: CALIBRATION PROCEDURES

Thermistors

Thermistors were calibrated using a 6 liter refrigerating circulator (Lauda (Model RMT-6), Brinkmann Instruments Co., Rexdale, Ont.). Temperatures of 10, 20, 30 and 40°C were selected to include the entire range of temperature values encountered during the experimental procedures. The set temperatures were controlled within ± .1°C and were checked against a standard reference thermometer. Thermistors remained at each temperature level for thirty minutes to ensure equilibration was achieved. Output voltages (to .1 mV) were recorded from the HP3497A data acquisition system, except for the four rectal probes which were measured by a portable voltmeter (Model 5000, Dana Laboratories Inc., Irvine, Ca.).

Heat Flux Transducers

The use of heat flux transducers to measure the rate of heat transfer across a given area of the body surface during water immersion has been subject to extensive investigation (Hody and Kacirk, 1972; Kuehn, 1978; Wissler and Ketch, 1982; Bell et al., 1985). Although caution has been expressed in extrapolating the heat flux observed at one site to adjacent sites, especially in water where a low thermal resistance occurs (Wissler and Ketch, 1982), Bell et al. (1985) reported good agreement between heat flux measured from 3 to 15 sites over the entire body.

One potential source of error has been shown to be the calibration constants supplied by the manufacturer. Bell et al. (1985) reported an increased error with increasing rates of heat transfer (i.e., at initial stages of cold water immersion), however linearity was observed until heat flux approached $\simeq$ 400 $W \cdot m^{-2}$.

In the present study, the manufacturer's calibration constants were checked using a copper encased water bath designed for measuring the thermal properties of clothing material (Figure

A.1). A similar device was used by Nuckels and Piantadosi (1980) for calibration of heat flux transducers used in hyperbaric helium.

Apparatus

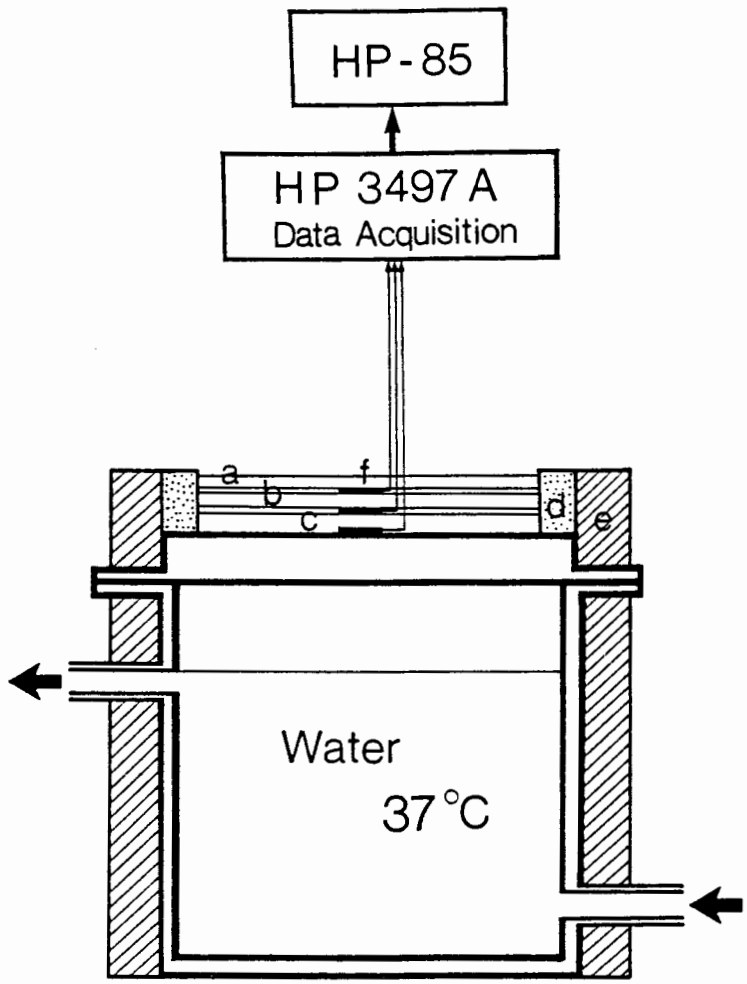
Water contained in a 31 x 36 cm rectangular container was heated and circulated through the copper cylinder (diameter = 15.4 cm) by a dynamic flow heating system (Heto, Denmark). Six rubber hoses (diameter = 4 cm) circulated water continuously between the supply container and the cylinder. Two hoses supplied water to inlet tubes located at opposite ends of the cylinder, 3 cm from its bottom. Four outlet tubes, situated at equidistant points around the cylinder, 5 cm from the material–cylinder interface, were connected by hoses to the water supply for recirculation. Four incremental bath temperatures (20, 30, 45 and 55° C) were selected to provide a wide range of calibration heat flow values.

Three slabs of 5 mm neoprene (diam = 15.35 cm) were situated above the copper cylinder (a, b, c in Figure A.1) with thermocouples placed centrally between each interface. Each heat flux transducer was placed adjacent to the middle thermistor. Output voltages (to .1 mV) were recorded after 90 minutes to ensure equilibration. Calibrations were conducted with the apparatus *in situ* to the experimental situation so that output voltages were collected on–line through the data acquisition system (HP3497).

Calculation of Heat Flux for Calibration

Heat flow across the surface of a material is influenced by a combination of factors including the thermal conductivity, thickness and area of the surface over which the heat is transferred. Analagous to Ohm's Law (Voltage = Current x Resistance), heat flow ($\dot{Q}$) may be expressed as:

$$\dot{Q} = \frac{\text{thermal potential difference}}{\text{thermal resistance}} \qquad (1)$$



a Neoprene
b fabric
c standard fabric
d cork
e insulative material
f thermocouple

Figure A.1: Calibration device for heat flux transducers. For the purpose of the present thesis, the three fabric slabs ("a", "b", "c") were all neoprene and the water bath temperature was altered to determine heat flux at three bath temperatures (see text).

The thermal potential difference is the temperature gradient (ΔT) across the material and thermal resistance (R_{TH}) may be calculated as:

$$R_{TH} = \frac{\Delta X}{kA} \tag{2}$$

where, ΔX = thickness of material

k = thermal conductivity of material

A = surface area of material

From a prior investigation, the thermal resistance of neoprene, 5 mm thick and 15.35 cm in diameter, was established as .116 $m^{2}\cdot^{\circ} K\cdot W^{-1}$.

In addition to the influence of thermal resistance on the heat flow measured across a surface, the present apparatus involved the measurement of heat flow across two solid slabs of identical material (Figure A.2). Thus it was also necessary to account for the thermal contact resistance between the neoprene slabs, which was previously determined as 6.6 x 10^{-3} $m^{2}\cdot^{\circ} K\cdot W^{-1}$. Taking into consideration both the thermal resistance and contact resistance of the neoprene, $\dot{Q}$ was calculated using the following equation:

$$\dot{Q} = k_A A \frac{T_1 - T_{2A}}{\Delta X_A} = \frac{T_{2A} - T_{2B}}{\frac{1}{h_c A}} = k_B A \frac{T_{2B} - T_3}{\Delta X_B} \tag{3}$$

This equation may then be rewritten as:

$$\dot{Q} = \frac{T_1 - T_3}{\frac{\Delta X_A}{k_A A} + \frac{1}{h_c A} + \frac{\Delta X_B}{k_B A}} \tag{4}$$

Table A.1: Regression equations for calibration corrections for heat flux transducers

	Temperature Factors			Heat Flux Factors		
Transducer No..	slope	intercept	r^2	slope	intercept	r^2
1280 (Chest)	1.081	.986	.998	.715	2.725	.986
1313 (Arm)	1.080	1.006	.998	.700	2.050	.996
1320 (Thigh)	1.077	1.609	.998	.902	-2.817	.967
1314 (Calf)	1.076	1.176	.998	.801	.389	.994
1319 (Back)	1.085	2.885	.961	.929	.539	.991

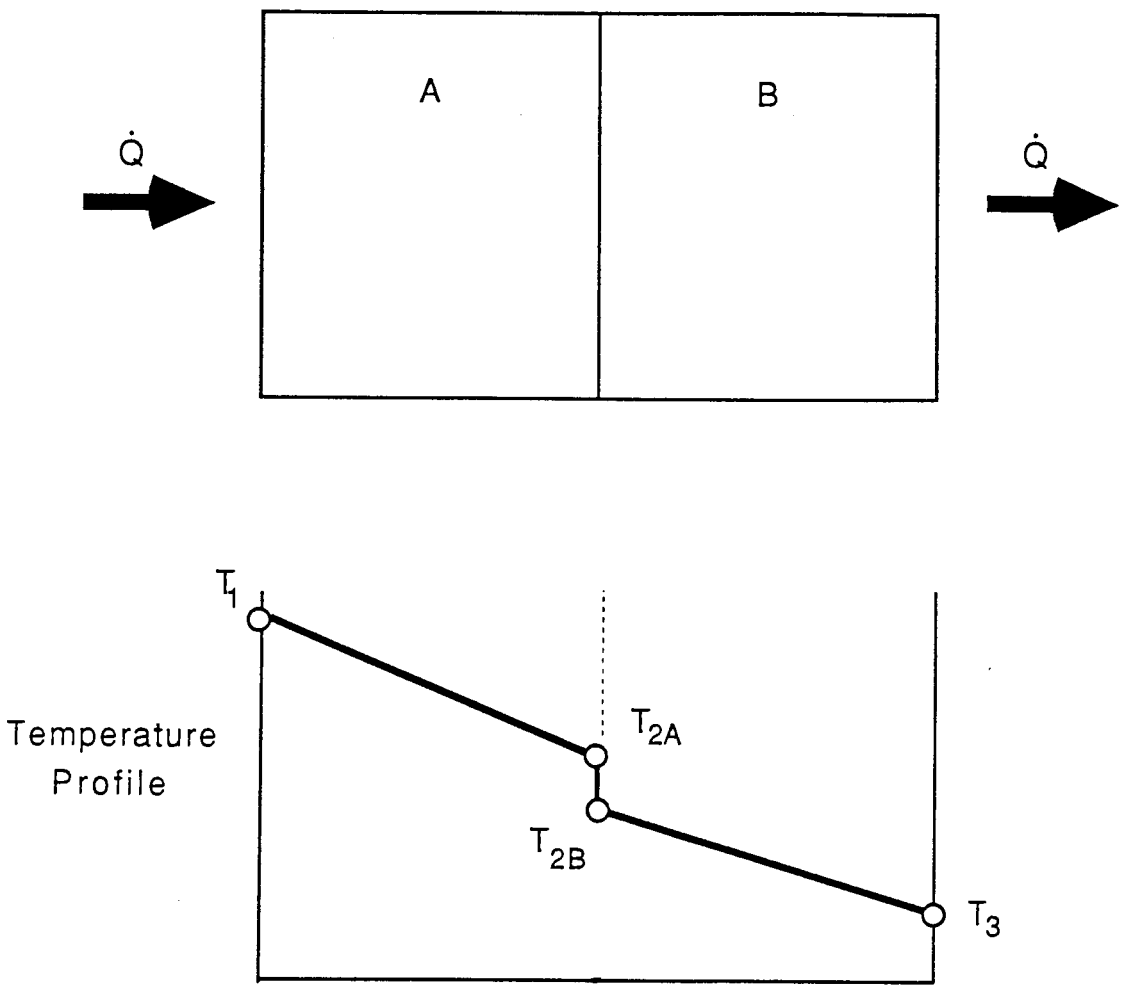


Figure A.2: Heat flux and thermal contact resistance between two neoprene slabs, "A" and "B". The heat flux transducer was placed between the neoprene and the observed heat flux was calibrated against the calculated heat flux as described by the equations in the text.

Regression coefficients and intercepts for the correction equations applied to the observed values of the heat flux transducers are shown in Table A.1.

Limitations

Although the intention was to calibrate the transducers over a range potentially observed during cold water immersion experiments (50 to 1500 $W \cdot m^{-2}$), the maximum temperature which could be maintained by the heating system was 55° C. Due to the high insulative quality of the neoprene, this translated into a calculated heat flow of 105 $W \cdot m^{-2}$. As the response of the transducers has been reported to be essentially linear up to ≃400 $W \cdot m^{-2}$ (Bell et al., 1985), these calibration constants were utilized. However, the accuracy of the values observed beyond this linear range cannot be validated.

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