# Mechanisms Involved in the Effect of Head Cooling On the Rate of Decrease in Core Temperature

by

**Thea Pretorius**

A Thesis submitted to the Faculty of Graduate Studies of

The University of Manitoba

in partial fulfilment of the requirements of the degree of

**Doctor of Philosophy**

Individual Interdisciplinary Program

In the Departments of Kinesiology and Recreational Management, Physiology, and Community Health Sciences

University of Manitoba

Winnipeg, Manitoba, Canada

*Your file Votre référence*
*ISBN:* 978-0-494-64311-2
*Our file Notre référence*
*ISBN:* 978-0-494-64311-2

THE UNIVERSITY OF MANITOBA

FACULTY OF GRADUATE STUDIES

*****

COPYRIGHT PERMISSION

# Mechanisms Involved in the Effect of Head Cooling On the Rate of Decrease in Core Temperature

By

Thea Pretorius

A Thesis/Practicum submitted to the Faculty of Graduate Studies of The University of Manitoba in partial fulfillment of the requirement of the degree

Of

Doctor of Philosophy

Mamma, Ous, Dirk, Ei en Tommie. Julle hou die afstand altyd net 'n telefoonoproep ver en was dikwels my behoud in hierdie baie moeilike tyd. Ek is baie lief vir julle almal.

ACKNOWLEDGEMENTS

To be able do a PhD one has to rely on a team of experts to guide you with very little you can give in return. This leaves you in debt to many people at the end of that period. I hope that this one page will convey my sincere appreciation for my professors, friends and family.

To Dr. Lisa Lix, thanks for giving me an introduction to mathematical modeling and teaching me more about multi-variable analysis. I have enjoyed doing the course with you so much and having you and your expertise on my committee to explore the use of SEM in our field of research was an honor. Thanks for always being so friendly, supportive and prompt with you valuable responses, even after you have taken up the demanding job in Saskatchewan.

Dr. Newman Stephens, it was an honor to have someone with so much experience in English grammar, research, physiology and statistical analysis on the committee. Apart from your brilliance, you always made me feel good about my contribution to Science. Thank you so much.

Despite being 'thinly spread' in the department, I will always remember the way Dr. Phillip Gardiner helped students to make their effort count. In the past years, I often was one of those students. Thank you specifically for helping me rewrite chapter three of this thesis.

Being a student for the past many years in the Laboratory of Exercise and Environmental Medicine was equally difficult at times for both Dr. Gordon Giesbrecht and me. Despite of that he encouraged me to stay the two times I wanted to quit. Thanks Gord and thank you for allowing me to do a PhD in this facility. With many other thermo physiologists, I am happy you came back after three years of dedicating much of your time to a Bible College. There is essential work in the field of thermoregulation that could only be done in this laboratory, and I am proud to have been part of it for some time.

To my fellow students, Farrel, Gerren and Dominique I owe a great thanks. It was a challenging time for all of us and without the encouragement of each other we would not have gotten through. I am proud of you guys and will watch the stage of the world to see when it is your turn to appear.

To my friends, who without knowing, played crucial roles in encouraging me to keep on going until the end were Maggie, Cindy and Barry. Thank you so much for just being a friend.

And last but definitely not least, my family, who made it worth going home every day – thanks Jassie, Chrismarie and Marissa – I love you all so dearly.

INDEX

LIST OF TABLES

CHAPTER 1



CHAPTER 2



CHAPTER 4

LIST OF FIGURES

LIST OF ABREVIATIONS

| | |
|---|---|
| %BF | Percentage blood flow |
| ANOVA | Analysis of variance |
| AVA | Arterio-venous anastomoses |
| BF | Body fat |
| BSA | Body surface area |
| CI | Confidence interval |
| CMR | Cerebral requirements of oxygen |
| CWI | Cold water immersion |
| D | Disturbances |
| df | Degrees of freedom |
| DI | Dorsal head immersion |
| DROP | Drop in core temperature |
| FI | Facial immersion |
| HFT | Heat flux measured by thermocouple in $W/M^2$ |
| HL | Heat Loss |
| HP | Heat production |
| HR | Heart rate |
| ITS | Integrated thermal signal |
| M | Metabolic rate |
| MAP | mean arterial pressure |

| | |
|---|---|
| ML | Maximum likelihood |
| N.S. | Not significant |
| NFI | Normed fit index |
| $O_2$ | Oxygen |
| PA | Path analysis |
| PAR-Q | Medical questionnaire |
| PCO2 | Partial pressure of CO2 in the blood |
| Q | Cardiac output |
| RAM | Reticular action modeling |
| RER | Respiratory exchange rate |
| RHL | Respiratory heat loss |
| RMSEA | Root mean square error of approximation |
| SD | standard deviation |
| SEM | Structural equation modeling |
| SR | Structural regression |
| SRMR | Standardized root mean square residual |
| $T_a$ | Ambient temperature |
| $T_{co}$ | Core temperature |
| $T_{es}$ | Esophageal temperature |
| $T_{sk}$ | Skin temperature |
| $T_{skavg}$ | Average skin temperature |

| | |
|---|---|
| VAR | Variable |
| $V_E$ | Minute ventilation |
| $V_O^2$ | Oxygen consumption |
| WI | Whole head immersion |

# CHAPTER 1

# GENERAL INTRODUCTION

## INTRODUCTION

Many aquatic recreational, commercial and military activities present the risk of human exposure to cold water and the consequent development of accidental hypothermia. Recreational activities that typically have the potential to result in cold water emergencies include swimming, diving, fishing, sailing, water and snow skiing, dog sledding, diving, hunting in cold weather and even snowmobiling. Commercial activities such as fishing, diving, shipping, oil drilling and aquatic recreational activities in the tourist industry also pose a risk to water immersion and the consequent dangers that go along with that. History is full of evidence of hypothermic victims of military activities of not only the Coast Guard and Marine Corps but also the Army and Air Force.

Although drowning is relatively easy to prevent, hypothermia is not. According to Giesbrecht and Steinman (26) hypothermia is now more widely recognized than in the past, but prevention of hypothermia is still a difficult and often expensive proposition. Therefore, in cold regions, cold water safety, knowledge of cold water risks, the use of appropriate floatation and insulative clothing are essential.

Survival time prediction is critical in the efficient planning of any cold water rescue action. Predictions on survival time are often based on experience and case studies of cold water survivors and are limited to every unique situation.

Several studies on humans have been done to try to understand the effect of different levels of cold water immersion on the rate of body core cooling while only one study (64) has looked at the effect of submersion of the whole body and whole head in humans.

Factors that have been investigated include biophysical and physiological factors such as body composition (25,45,90), movement (33), body position (32), nutritional state (39,94), exhaustion (38,64,74), the effects of drugs and alcohol (40), shivering heat production (18,19,84,85), different levels of immersion (46,64,90), age and gender (83) and sea state (73). Although all of these factors have a significant effect on the development of hypothermia, no single factor investigated so far, could explain all inter- and intra- individual differences (90). The effect of blood flow has also been recognized by several studies (47,63,86,95) to play a role in thermoregulation. The study of this factor is complicated by the difficulty of measuring skin and muscle blood flow accurately (90). Increased muscle blood flow, and consequently the increased total perfused body mass (or thermal core), seems to contribute to core cooling rate during shivering in cold water (90). Contrary to this, it seems that reducing the thermal core increases core cooling rate during head immersion (46,82) and submersion (64).

Observations of controlled exposures to cold are limited and could only be measured at mild levels of hypothermia in healthy, breathing adult subjects, far from any risk of death. Controlled experiments in this field typically control for most of the above factors and try to isolate the effect of a single factor. It becomes clear, as more studies are done on this topic, that any attempt to predict the development of hypothermia has to take into account not only all the individual factors, but also the interaction between the factors, to fully understand the development of hypothermia. Because of the limitations of the practical and ethical implications of these studies, mathematical modeling is one approach that can be used to address these difficulties.

## STATEMENT OF THE PURPOSE OF THIS PROGRAM

Two more studies were conducted in an attempt to explain some of the mechanism(s) for the effect of whole head submersion in cold water on vasomotion and the rate of decrease of body core temperature ($T_{co}$) in shivering humans. Also, Structural Equation Modeling (SEM) was used to explain the role of the factors not controlled for in the experimental design.

In order to do this the following studies were done:

Study 1: Determine the effect of whole head cooling on core cooling rate in shivering humans in 17°C water.

Study 2: Determine the effect of face, dorsal head and whole head cooling on the level of vasoconstriction in humans over an extended period of time (one hour).

Study 3: Use Structural Equation Modeling to analyze the data of the head cooling studies to determine the integrated effect of factors which play a role in core cooling during whole head cooling as well as the magnitude of the effects.

## IMPORTANCE OF THE STUDY

This study has practical implications regarding the gradual onset of hypothermia during cold water exposure, whether accidental or during planned activities such as SCUBA diving, fishing and cold water swimming. Search and rescue personnel could benefit by understanding the effect of dorsal and whole head cooling and this could help in the survival time prediction for cold-water victims. Knowledge of the effect that dorsal and whole head exposure has on core cooling can help with the setting of protocols for designing of personal flotation devices and insulation for cold-water SCUBA divers and long distance swimmers in order to prevent hypothermia. It might also help to explain the mechanisms of

brain cooling in the event of cold-water near-drowning that serves as protection against ischemic brain damage.

Head cooling is also used for the induction of hypothermia for the benefits it has in some clinical conditions. Hypothermia is used for prevention of brain damage in patients suffering from stroke (85), brain injury (53) or cardiac arrest (3). The mechanism by which the brain is protected by cooling might be linked to a reduced metabolic rate (52) as well as reduction in biosynthesis, release and uptake of neurotransmitters (9).

Alexander (1) reported on unethical studies[1] that were done by two German investigators during the Second World War in concentration camps in Dachau. They found that the effect of cooling of the dorsal head in 1 – 2 °C water on rectal temperatures was minimal when the body was not exposed to the cold water, compared to the much greater effect when the whole body as well as the dorsal head was exposed. Although many are hesitant to use these data, it is referenced here only to provide a full review of studies done in this area. These

---

[1] These studies on prisoners of war in Dachau during World War II were grossly unethical, and the results are often considered invalid and unusable because of the emaciated condition of the prisoners as well as questions regarding the protocol and accuracy of the results.

studies were done by Lockhart et. al. (46) in 10°C water on shivering humans, and Giesbrecht et. al. (25) in 12°C water on non-shivering humans. It compared two personal flotation devices, which either kept the dorsal head in or out of the water. Comparison of the three studies showed that the effect of dorsal head cooling was augmented as water temperature decreased. The assumption was made that dorsal head cooling had a larger effect on a reduced thermal core (due to vasoconstriction), when the same amount of heat loss had a bigger effect on the core temperature.

Xu et al. (91) explained brain cooling using a 2-dimensional mathematical model that illustrated the avenues of heat loss through the human head during submersion. It showed that most heat was lost through convection by aspirated water in the lungs. Convection and conduction through the scalp had a minimal predicted effect on the rate of core cooling (91). Parts of this model were based on studies done on shaven, anesthetized dogs that were submerged in different conditions (15). This was verified by experiments on human cadavers (irrigating the nasopharyngeal area) and one human subject whose head was submerged in cold water (91).

A study on whole head cooling (64) with shivering suppressed was the first demonstration of a core cooling effect of whole head exposure when the body was both cold-stressed and insulated from the cold. In both these conditions, head

cooling increased core cooling with ~39%, while the heat loss from the head increased total heat loss with ~11%. This was in contrast with the dorsal head cooling studies where the increased core cooling effect was only seen when the body was exposed to the cold water (25). With facial submersion during whole head submersion, vasoconstriction would result from thermoregulatory control mechanisms. However, the human dive reflex, mediated by the trigeminal nerve, is known to also induce significant peripheral vasoconstriction as an oxygen sparing mechanism. This was considered the explanation for the increased core cooling rate even when the body was insulated from the cold (64).

The first two studies that will be done are designed so that the data analysis could be done with the use of repeated analysis of variance (ANOVA). This method allows for one variable to be changed while it is assumed that all other variables are controlled for by the comparing of each subject with him/herself. Any difference that would be detected in the outcome variable then is associated with the manipulation of the independent variable. It should be kept in mind that there are many variables affecting core cooling (see literature study) and although the ANOVA controls for 'between subject' differences, it does not necessarily control for 'between condition' differences. The use of a mathematical model will help to identify independent variables which are also

responsible for differences other than variables manipulated by the experimental conditions.

# REVIEW OF RELATED LITERATURE

## Introduction

This review gives a summary of the mechanisms through which humans exchange heat with the environment, the thermoregulatory control mechanisms as well as factors affecting core cooling. It also gives information on cold water immersion and submersion and results of related studies done on head cooling, with a special section on heat loss (HL) from the head. This part of the literature review overlaps with the literature study done by the applicant for fulfillment of a Masters degree (63). Finally, it includes a brief history on the importance and development of different mathematical models for thermoregulation and the potential of Structural Equation Modeling (SEM) for the purpose of analyzing data in this field.

## *Mechanisms of heat loss and gain*

Homeothermic species like humans require a relative constant core temperature ($T_{co}$) for optimal metabolic function. The human core temperature is

maintained within 0.2°C from the normal, which is generally considered to be 37°C (70).

The homeostasis in the human body depends on a balance between heat loss and heat gain. Physical mechanisms through which the body loses heat are radiation, conduction, convection and evaporation. Radiation from the skin surface happens through emission and absorption of heat energy, and is the largest source of heat loss. Conduction is the direct transfer of heat away from the body by substances cooler than the body surface, with which it is in direct contact. Evaporation, which is responsible for 20 to 30 percent of heat loss in temperate conditions, occurs when heat energy from the skin is used for the transformation of liquid (sweat) into a gas form. Convective heat loss occurs when air or water (that has a temperature below that of the temperature of the skin) ($T_{sk}$) comes in contact with the skin, is warmed and moves away so that the air (or water) that replaces the warmed air has to be warmed again (24).

Heat is gained through metabolic heat production (general metabolic function, shivering and exercise) and donation from the exterior (eg. warm air, water etc.). Metabolic heat is produced within the cells and then transferred to the surroundings. This process occurs passively through conduction, due to the thermal gradient between the cell and its surroundings and convectively through movement of blood throughout the body (60).

## Thermoregulation

Thermoregulation, necessary to maintain a body temperature favorable for metabolic function, occurs in three phases. First, afferent thermal information is received from peripheral sensors in the skin and central sensors in the deep central tissues (abdomen and thorax), the spinal cord, the hypothalamus and other parts of the brain. The peripheral sensors are particularly rate sensitive whereas the central response is a direct reflection of the absolute temperature of tissues as well as the returning blood from the peripheral area (70).

The hypothalamus is the main central regulator of body temperature with the preoptic posterior hypothalamus responsible for heat production and the preoptic anterior hypothalamus responsible for heat dissipation (49). It responds to an integrated thermal signal (ITS) from all afferent stimuli via response mechanisms that alter behavior and physiological responses that either alter HL to the environment or increase metabolic heat production.

Thermoregulatory models have originally strived to include regulation of body core temperature about a set point or an absolute value at which thermoregulation responses are triggered (Set point theory). Until the present moment, the mechanisms by which the set point is determined remains a mystery (17) and as a consequence, the definition of the set point has evolved over the past decades. Today it is accepted that the core temperature at which

thermoregulation responses are triggered does not exist as a fixed value, but rather a range of values.

An inter-threshold range exists in between the mean body temperature at which the ITS evokes either vasodilation or vasoconstriction and typically is 0.2°C (70). Some studies have clamped skin temperature to isolate the effect of core temperature on these responses (50) and defined a null zone or interthreshold zone (49) as a range of core temperatures at which no sweating or shivering responses are elicited.

These ranges depend on skin temperature and its rate of change (10,50) as well as other non thermal factors such as blood glucose, state of hydration, sleep, fever and environmental factors such as inert-gas narcosis (49). These and other non thermal factors not only have an effect on the interthreshold range, but also the magnitude in which the body responds to the cold or warm stimuli (49). This magnitude is also referred to as gain (70) and determined by the change in the response of the body to the magnitude of change in the stimuli.

The initial response to warm temperature is behavioral in nature and includes a body posture that allows for more surface heat loss, shedding of garments or moving to a cooler environment. Physiological responses to warm temperature include vasodilation which requires little resources, and sweating with a consequence of dehydration and loss of essential electrolytes.

As with heat, the initial responses to cold are behavioral and might include changed body position or the addition of clothing to increase insulation and decrease heat loss from the skin surface or avoiding the cold exposure.

Sympathetically mediated peripheral vasoconstriction is the first physiological response to a cold environment and, as with vasodilation, has minimal energy and resource requirements. It will decrease heat loss from the body surface through an increased insulation, decreasing radiative and convective heat loss (70). Cold induced vasoconstriction is limited to the distal extremities in the normal subject (29). Skin blood flow in the toes, fingers, nose, lips and ears are regulated through capillary and thermoregulatory arterio-venous shunt components (70), also referred to as arterio-venous anastomoses (AVAs), which are functionally different from capillaries which supply the remainder of the skin. These areas of the distal extremities also have a significant amount of innervated alpha receptors, which are believed to regulate the vasomotion of the AVAs (29). Cold-induced vasoconstriction is mediated primarily by the release of norepinephrine from presynaptic alpha adrenergic nerve terminals (29). This stimulus mainly affects the AVAs and has only a minimal effect on capillaries (70) which are more responsive to metabolic demands. Vasoconstriction is not a steady constriction of blood vessels but rather a dynamic state where AVAs constantly recycle from an open to a closed state while most of the AVAs are closed. This is

opposed to vasodilation where most of the AVAs are dilated. Vasomotion does not require much energy (60) and is most likely maximal in an environment sufficiently cold to cause central hypothermia (67). Blood flow in the fingers could be increased from 0.5 - 1 cc/min/cc tissue in a vasoconstricted state to 80 - 90 cc/min/cc tissue in a fully dilated state.

The neural response of vasoconstriction of the AVAs is also elicited by the human dive reflex, mediated by the trigeminal nerve, and is accompanied by bradycardia and breath hold - acting as an oxygen sparing mechanism (12,35,38). This mechanism occurs in thermoneutral water (i.e., 34°C) (12) and increases in magnitude at cooler water temperatures (i.e., 20-25°C) (35,38).

If the ITS increase further it will increase its metabolic heat production mainly through shivering which can increase heat production by 200-600% in adults (22,70) and requires higher oxygen consumption. This is the body's last defense against hypothermia. The net efficiency is lower than one would expect because muscle metabolism increases blood flow to the peripheral tissues and consequently increases heat loss to the environment (70).

The primary affect of cooling of the body is a decrease in metabolism according to the $Q_{10}$ principle. This is a change of physical and chemical rate of reactions with a 10°C change of temperature. The $Q_{10}$ of the whole body is about 2 (52), indicating that the metabolic rate of a body at body core temperature

($T_{co}$) of 37°C is twice the metabolic rate of a body with a $T_{co}$ of 27°C. Normally, this primary effect is overridden secondary to the increased metabolic requirements of shivering.

| Classification | Core Temperature | Symptoms and signs |
|---|---|---|
| Mild | 35 - 32°C | • intense shivering<br>• decrease in neural transmission<br>• muscular effectiveness<br>• increase in oxygen consumption ($V_{O2}$), minute ventilation ($V_E$), heart rate (HR), cardiac output (Q), and mean arterial pressure (MAP) |
| Moderate | 32 - 28°C | • shivering waxes and wanes, eventually ceases<br>• loss of consciousness |
| Severe | <28°C | • shivering is absent<br>• death occurs due to acid-base disturbances causing ventricular fibrillation and cardiac arrest. |

Table 1. The different stages of hypothermia.

## Measurement of core temperature

Core temperature is considered the temperature of the internal tissues of the body; this temperature stays stable, unlike the temperature of the outer body surfaces, which are more quickly affected by the environment. The non-invasive measurement of core temperature is a challenge and despite the development made in the medical world during the last century, the most common ways of measuring core temperature are still the mercury thermometer under the tongue (55) or infrared measurements in the ear canal. The measurement of core temperature during hypothermia has an added challenge due to the temperature gradient in the body that is increased under these conditions. The following cites for the measurement of temperature will be discussed: oral, tympanic membrane, rectal and esophageal temperatures.

The preferred placement of the thermometer in the mouth is in the sublingual pocket (i.e. under the tongue) due to the differences in temperature in different parts of the mouth. This method is popular in clinical environments, but is not feasible during long experiments due to the difficulty to keep the thermometer in place. Oral temperature is considered somewhat lower than core temperature and is influenced by any food, drinks and also breathing patterns (55) as well as local cooling of the face and head (5).

The accessibility of the tympanic canal makes this cite an obvious choice for some researchers although it could be uncomfortable and even result in perforation of the tympanic membrane (71). The tympanic membrane receives blood from the internal carotid artery and is considered by some (11) as a good indicator of brain temperature, although Nybo (59) showed that it is more responsive to face fanning than the temperature of jugular venous blood returning from the brain. Tympanic temperature has also been shown to be consistently lower than the blood temperature of the heart during steady state (72) as well as during exercise (59).

Rectal temperature is considered practical and accurate for measuring core temperature (75) especially in babies and small children (55). This method may have a prolonged response time compared to other methods as faeces could insulate the probe (71).

The esophagus is preferred by many as the site to measure (5,69) because it is located deep in the body close to the left ventricle, and its ability to respond quickly to changes in core temperature. In a study done in an intensive care unit it was found that esophageal temperature reflected quantitatively and more quickly the temperature changes in the pulmonary artery than tympanic membrane temperature (72). Therefore it is suggested that esophageal temperature is a preferable index of central blood temperature. The biggest disadvantage is the

difficulty to insert the probe, the discomfort, mainly to the nasal passages and throat (75) as well as the influence of any recent food or drink intake. This site is more appropriate for research than clinical conditions (55).

The measurement of the pulmonary artery is considered the most accurate since this artery shunts blood directly from the core to its surroundings (5). The invasive nature of it makes it an undesirable option in the research setting.

## Factors affecting core cooling

Several anthropometric factors have an effect on the rate of core cooling. Body composition (% body fat, height and weight) has been shown to have the greatest effect of these factors. At a given skin temperature shivering heat production will be less in humans with greater amounts of subcutaneous fat (25,41,45,90). Gender was not shown to have an effect on the decrease in $T_{co}$ due to cold water submersion of young humans. However when both age and gender were considered, older men were more susceptible to cold environments than women and younger men (27,84).

Vasomotion also appears to have an effect on the rate of core cooling. It is generally believed that vasoconstriction, as first response to an increased ITS, will decrease HL from the body surface and decrease the cooling rate of the body core. Vasomotion affects blood flow and consequently heat delivery to the

periphery. Normally cold stress would increase vasoconstriction, which reduces heat loss from the surface of the body, preserving $T_{co}$. Regional differences exist in the vasomotion response. An increased ITS will increase vasoconstriction of the AVAs, which reduces the blood flow to the distal limbs. However, a local cooling effect of the proximal limbs, the trunk and the head, will cause vasoconstriction of the capillaries of those regions. In four studies done on humans (1,22,47,63) it was shown that when the body was vasoconstricted, HL from the head increased core cooling significantly (250% in 1-2°C water to 40% in 17°C water). This was despite the fact that HL from the head did not appear to be disproportionally more than its proportional body surface (5 and 1 % increase in total heat loss with dorsal and whole head respectively). In contradiction to this, Xu (90) applied data from partially immersed, shivering subjects to a cold thermoregulatory model. Simulation analysis suggested that increased leg muscle blood flow (i.e. increased thermal core) due to increased shivering, may contribute significantly to the drop in core temperature in individuals with low % body fat (8.1 - 19%), and partly account for individual thermal responses to partial immersion.

Pugh (65) concluded that when cold stress, fatigue and discomfort are combined, the point of exhaustion is reached earlier than when the stresses operate in isolation. This would suggest that with the same cold stress, individuals

who are more fatigued would have less defense against core cooling. Jacobs (61) showed a negative relationship between blood glucose levels and rate of core cooling when humans are immersed in cold water. Young et al. (94) conducted a study on eight young males to determine the effect of exertional fatigue and chronic negative energy balance on thermoregulation. Military trainees were subjected to severe training while they were deprived from sleep and food. They had an average negative energy balance of ~ 850 kcal/day, lost 7.4 kg body weight and had an average of 12% body fat at the end of the training period. They were then subjected to cold exposure for four hours on three occasions: immediately after the course; 48 hours later, after a period of rest and energy repletion; and 109 days after the course. Immediately after the course no one could endure the cold for the entire 4 hours while only five of the eight subjects could complete the trial 48 hours later. On the third trial, when an average of 12.8 kg lean and fat body mass were restored (average body fat was 21%) everyone could complete the trial. This was likely because of the added metabolic capacity of regained muscle mass and the increased thermal insulation of the new fat mass.

External conditions affecting rate of core cooling are water temperature, clothing and insulation (77), body position and activity level (33). It was shown that minimizing both voluntary activity and the exposure of major heat loss areas of the skin to the cold water is the best way of minimizing the drop in core

temperature. Sagawa et al. (68) showed that the lowest water temperature in which humans could maintain normal core temperature through muscular activity was 25°C. Steinman et al. (73) reported faster cooling rates (50 – 100%) for subjects in rough water compared to calm water when loose fitting, wet garments (boat crew overalls) were worn The same effect was not seen in conditions where clothing had better insulation value.

## Water immersion and submersion

Core cooling in cold water is relevant to two serious emergencies – cold-water immersion where the head is either partially immersed or above the water and cold water submersion where the whole head is submersed in water.

- *Cold-water immersion*

Cold-water immersion (CWI) is generally equated with hypothermia and sudden death so that the general impression is that hypothermia occurs quickly after immersion. The majority of laboratory work in this field involves CWI and the responses to it can be divided into three phases, each with its own responses and risks – the immediate 'Cold Shock' response, cold- incapacitation after about ten minutes and hypothermia that sets in after 30 - 60 minutes of immersion.

When someone is immersed in cold water, the 'Cold Shock' response occurs immediately and it lasts up to 1-2 minutes (18). This is provoked by a massive

nervous stimulus (18) from the cold sensors in the skin and results in respiratory responses such as the gasp reflex, hyperventilation and a consequent reduction in partial pressure of $CO_2$ in the blood ($P_{CO2}$) as well as cardiovascular responses which includes vasoconstriction, tachycardia and hypertension (15,30,42,82). The gasp reflex may result in drowning while the hyperventilation could lead to fainting and consequent drowning. The cardiovascular responses significantly increase the workload of the myocardium, which in addition to a massive sympathetic discharge may lead to cardiac arrest and death, which is more likely in patients with underlying heart disease. The intensity and duration of these responses depend on the initial $T_{sk}$ and the rate of change of $T_{sk}$ (18) which will be affected by the temperature of the water, insulation and level and rapidity of entry (6). The response is greater in a high sea state (73).

The second phase starts when breathing gets under control and is known as 'Cold-Incapacitation'. Initially neural transmission ceases with consequent impairment of fine motor control. This is followed by impairment of voluntary muscle movement. If death occurs in this phase, it usually is because of drowning. Hypothermia, which is discussed in detail in another section, only sets in after about 30 – 60 minutes.

- *Cold-water submersion*

This refers to the condition where the whole body and head is submersed in the cold water and is relevant in two conditions, SCUBA diving and cold-water-near-drowning.

SCUBA diving in cold water sometimes results in the development of symptomless hypothermia with the risk of mental and physical cold-incapacitation that could lead to serious injury or death.

The remarkable survival of cold-water near-drowning victims (4) with intact neural outcome focused attention on cold-related mechanisms which protect the brain from ischemic injury. For a long time it has been attributed to the decreased cerebral metabolic requirements of oxygen ($CMR_{O2}$) according to the $Q_{10}$ principle. Mitchenfelder (52) has shown that the $Q_{10}$ of the brain increases from 3 between 37°C and 27°C to 4.8 between 27°C and 18°C. If the brain could survive an ischemic insult for 5 minutes at 37°C, it should survive 15 and 72 minutes respectively at 27°C and 17°C. The implication of this is that the brain had to cool at a very fast rate (more than 10°C in 5 minutes) or the victim had to be severely hypothermic at the point of submersion to explain these survival case studies.

However, several of these case reports (45,58) showed that the core temperatures of these patients were higher than what was required for the protection based on the decreased $CMR_{O2}$ alone. These results indicate that there were other mechanisms providing brain protection. Today it is known that even mild hypothermia protects the brain from ischemic injury (8,9,53,54). The mechanisms involve suppression of neurotransmitters (glutamate and dopamine) and free fatty acids (9), the protection of the blood brain barrier[2] (21) and reduction of hydroxyl radical production (28).

## Heat loss through the head

Xu et al. (91), developed a mathematical model to predict the rate of human brain cooling during cold-water submersion. This model was based on research done by Conn (15) on anesthetized, shaven dogs that were submersed in cold water under different conditions, human cadavers as well as one experiment on a human with the head submersed in cold water while the body was in thermal neutral water. The model distinguished between two boundaries through which heat was lost from the head - the outer boundary (skull and soft tissues) between the brain and the cold water, and the inner boundary, which is an interface between the brain and the upper airways. The model demonstrated the significance of each of these avenues

[2] The physiological mechanism that alters the permeability of brain capillaries to certain substances

during submersion when one or both of them were exposed to the cold water. Predicted conductive brain cooling through the outer boundary was minimal (< 1°C) within the first ten minutes of cooling. The effect of the inner boundary was greater – a drop of 4°C in 10 minutes was predicted when both the inner and outer boundaries were cooled. The contribution of the inner boundary was thus 3°C in the first 10 minutes.

Conn (15) found that the dogs were breathing water for several minutes after submersion. Flushing of the lungs with cold water cooled the pulmonary blood, which had a circulatory cooling effect on the whole body, including the brain. The application of these findings on the human head was validated with data obtained from human cadavers where the nasopharynx was irrigated with ice water and another subject that cooled his outer boundary with cold water of different temperatures (91).

According to the model of Xu et. al. (91), heat loss through the head during submersion in 2°C water occurs mainly through the convective heat loss to the circulating blood in the lungs. A drop of 7°C in 10 minutes was predicted if all the contributions to brain cooling were considered.

Predictions on the effect of cooling of the outer boundary are consistent with results of Mellergard (51) who observed changes in human brain temperature with different cooling methods. All the patients had severe brain injuries and were

unconscious. Brain temperature was measured directly with a thermocouple introduced through an intraventricular catheter that was used to monitor intracranial pressure.

Some patients were cooled with a gel cap with which blocks of frozen liquid were wrapped around the head. The temperature of the gel cap was 0°C and the temperature beneath the helmet was 10 - 14°C after 2 hours of cooling. These patients showed no significant decrease in brain or rectal temperatures over a period of 2.5 hours. Other patients were cooled with a cooling helmet covering the head and part of the neck. The helmet had a system of thin plastic channels on the inside, through which 5°C water was circulated. Cooling with this helmet caused very little decrease in brain and rectal temperature (0.5 - 0.6°C) over a period of more than 4 hours.

Lockhart et al. (46) tested two personal flotation devices and their effect on head exposure to the water and rate of core cooling in 10°C water. The flotation devices differed in the way that they held the body in the water. The one device kept the body vertical with the head out and the other device held the body in a recumbent position so that the dorsal head was immersed. A control condition where only the dorsal head was immersed resulted in a small decrease in $T_{co}$. After a period of 35 minutes an average decrease of 0.1°C was seen. When only the body was immersed, and the head was out, the decrease was 1.0°C over

the same time period. The combined dorsal head and body immersed caused a 1.5°C decrease in $T_{co}$, which was more than the sum of the isolated effects of the head and body exposure. This was explained by the different blood flow patterns in the different conditions. In the condition where only the dorsal head was in the cold water, there was a large volume of perfused tissue. Any cooled blood from the head would quickly be dissipated through the relatively large perfused tissue mass and have a small effect on the core temperature. In the condition where the whole body was in the water, the blood vessels of the extremities were maximally constricted, reducing the thermal core. The cold blood returning from the head was distributed into a smaller tissue volume, causing the greater drop in core temperature.

The conditions of the study by Lockhart (46) were repeated in a subsequent study by Giesbrecht et al. (25) where the thermal effects of dorsal head cold water immersion were studied. This time the confounding effect of shivering heat production was minimized by the use of meperidine and the water temperature was 12°C. The results in this study showed that when the body was insulated, dorsal head immersion did not result in a significant decrease in $T_{co}$ compared to the head out condition (0.2°C). However, addition of dorsal head immersion to whole body cooling resulted in additional decline in body core temperature of 0.7°C and 38% increase in core cooling rate. The results in this

study did not confirm that heat loss from the dorsal head was greater per surface area compared to the rest of the body (5% of total heat loss vs 3% of body surface area). The difference in the effect of dorsal head cooling on body core temperature in the different conditions was again explained by the difference in volume of perfused tissue.

These findings were consistent with reports from Alexander (1) who reported on studies that were done by two German investigators during the Second World War in concentration camps in Dachau. They found that the effect of cooling of the dorsal head in 1 - 2°C water on rectal temperatures was minimal when the body was not exposed, compared to the much greater effect when the whole body as well as the dorsal head was exposed.

In the only study done so far on whole head submersion (64) it was found that in contrast with dorsal head immersion, whole head submersion increased core cooling even when the body was insulated from the cold water (17°C). In both the body insulated and body exposed conditions, whole head cooling increased by 39 % after 45 minutes of immersion, while heat loss from the head increased total surface heat loss with only 11%.

Froese et al. (23) and Rasch et al. (66) studied heat loss from the head during rest and exercise respectively. These studies were done in air and although the studies used different techniques they were consistent in indicating

that a powerful heat sink exists on the head. These studies showed an inverse relation between ambient temperature ($T_a$) and heat loss through the head. Froese (23) concluded that heat loss from the head might be a large portion of total heat loss in a cold environment.

Based on$T_{sk}$ measurements he concluded that very little vasoconstriction exists with a decrease in $T_a$, even when the rest of the body is in a general state of vasoconstriction.

Hertzman and Roth (36) indicated that there was an absence of vasoconstriction reflexes in the forehead circulation. They used a plethysmograph to illustrate the selective character of vasomotor activity in the skin. This would suggest that the heat loss from the unprotected head might be excessive during exposure to low temperatures. This study does report a gradual onset of vasoconstriction in the forehead that could be due to direct effect of cold on the vessels and not from neural stimulation as seen in the fingers and toes.

Studies done in this laboratory (46,64,82) did not support these findings. Both head skin temperature and heat loss measurements of the head indicated vasoconstriction and heat loss calculations showed that heat loss from the head was not proportionally higher than from any other area of the body. This could be due to a local cooling effect of the head where $T_{sk}$ decreases from ~30°C to close to the water temperature of 17°C. According to the $Q_{10}$ principle, the metabolic

rate of these cells should decrease by more than half with a subsequent vasoconstriction of capillaries in the skin.

It is clear that the literature is diverse in its findings about heat loss through the head and its effect on core temperature as well as speculations about the mechanisms involved. One reason for these differences is the different protocols and conditions under which these experiments were done. It seems as if one should distinguish between head cooling effects during exercise and during cooling conditions as in our studies. Also, cooling in air and cooling in water are very different as heat is lost 25 times greater in water due to the higher density of the water compared to air.

- *Heat loss through the head and brain cooling*

Two of the limitations in our research setting are the inability to measure brain temperature directly in normal humans and the limited relevance of animal studies due to the large inter-species differences in brain size, cranial structure and vascular anatomy (56).

Convective heat exchange between brain and blood could either occur via arterial blood supply to the brain or venous blood return from the cooled face or scalp. Venous blood from the face and scalp and skull depends on local hydrostatic differences since no valves are present. Blood from the face and

scalp could either leave the scull directly through emissary veins or indirectly by filtering through the marrow and diploic veins. This latter blood will flow into the meningeal vessels and to the large venous pools or sinuses lying under the periosteum and will not enter the cerebral blood circulation, but drain through the base of the skull. A model was described by Nelson (56) to investigate the effectiveness by which the brain could be cooled with the blood returning from the face and scalp. They found that due to the large volume of the human brain and the low thermal conductivity of brain tissue, brain temperature mainly depended on arterial blood heat transfer and less on venous return from the face and scalp. This supports the findings of Xu (91) who also predicted minimal hat loss through the scalp.

Based on the findings of Nelson (56) and Xu (91), brain cooling depends on the arterial blood from the heart, even when the head is cooled. It would be logical to assume that the brain follows the temperature of the heart. It is of importance here to note that while the resting $O_2$ consumption of the adult body is 3.5 ml/kg/min, that of the brain is 3.5 - 4.5 ml/100g/min, requiring 55ml blood/100g of brain tissue per minute (48). To meet this requirement the brain, which accounts for 2% of the body weight, receives 15% of the cardiac output, making it a possibility that the brain will reflect heart and lung temperature more quickly than other organs (16).

- *Brain cooling and protection against ischemic damage*

The remarkable survival of cold-water near-drowning victims (4) with intact neural outcome focused attention on cold-related mechanisms which protect the brain from ischemic injury. For a long time it has been attributed to the decreased cerebral metabolic requirements ($CMR_{O2}$) according to the $Q_{10}$ principle[3]. Mitchenfelder (52) has shown that the $Q_{10}$ of the brain increases from 3 between 37°C and 27°C to 4.8 between 27°C and 17°C. If the brain could survive an ischemic insult for 5 minutes at 37°C, it would provide 15 and 72 minutes respectively at 27°C and 17°C. The implication of this is that the brain had to cool at a very fast rate (more than 10°C in 5 minutes) or the victim had to be severely hypothermic[4] at the point of submersion to explain these survival case studies.

However, several of these case reports (45,58) showed that the core temperatures of these patients were higher than what was required for the protection based on the decreased $CMR_{O2}$ alone, indicating that there were other mechanisms providing brain protection. Today it is known that even mild

[3] Change of physical and chemical rate of reactions with a 10°C change in temperature

[4] $T_{co} < 28°C$

hypothermia[5] protects the brain from ischemic injury (8,9,53,54). The mechanisms involve suppression of neurotransmitters (glutamate and dopamine) and free fatty acids (9), the protection of the blood brain barrier (21) reduction of hydroxyl radical production (28) as well as other factors such as intracellular signal conduction, ischemic depolarization and early gene expression. This protective mechanism is applied in the clinical setting after stroke (14) and cardiac arrest (44) with favorable outcome in animals and humans. Despite the protection of hypothermia against brain damage, dangerous side effects such as bleeding with impaired coagulation, electrolyte disturbances, sepsis and myocardial disturbances (20) also occur due to the suppression of cell metabolism.

## Mathematical modeling

Exposure to cold could be life-threatening depending on degree of cold intensity and the length and nature of exposure. Controlled experiments on humans are limited to mild levels of hypothermia in healthy, adult subjects, far from any degree of danger or the risk of death, and only provide information on the initial physiological reactions and factors that play a role in the development

[5] 35 >Tco > 32°C

of hypothermia. Limitations of these studies are numerous and range from ethical to financial issues. Thermal modeling is useful for understanding and predicting human responses to extreme conditions whether by degree of cold stimulus, heat transfer or duration of cold exposure. Their application is very broad based, from analyzing possible scenarios for rescue to assisting post mortem investigations. Such models then are validated with related data acquired from experimental or case studies from real life scenarios.

The first and simplest forms of thermoregulatory mathematical models were linear regression equations of measured phenomena. The simplest model consists of a single energy balance where the rate of energy storage of the body is expressed as the sum of heat gained or lost through metabolic rate and heat lost or gained through the skin and respiratory tract. Machte and Hatch (47) improved on this model introducing core and skin temperatures expressing the stored heat content of the body as a linear function of core and shell temperatures represented as rectal and mean skin temperature respectively.

Pennes (62) and Wyndham and Atkins (89) developed similar models in 1948 when they solved the steady state conduction equation for a cylindrical region with uniform perfusion and heat generation, modeling the temperature profiles of the forearm. This served as the basis for Wissler's first model in 1961 (86) and

an improved version in 1964 (87) consisting of a six cylinder model representing six cylindrical regions of the body – a head and trunk, two arms and two legs.

Wissler (88) described how mathematical modeling came to age during the Apollo space program when Stolwijk (74) and his associates developed a computer program capable of predicting the thermal response of astronauts during extravehicular missions. He further explained how their relatively simple model adequately analyzed changes resulting from heat stress, but due to the large internal temperature gradients resulting from cold stress, it did not have the same ability to predict cold stress responses. For a model to adequately describe thermal response during the kinds of exposures encountered in diving or accidental immersion, it had to deal with factors such as perfusion rate in muscle and the magnitude of countercurrent heat exchange between large arteries and veins (88). At that time Wissler (87) had the most complete model of thermoregulation which although it was primarily a thermal model it also contained mass balances for oxygen, carbon dioxide, and lactic acid in order to provide information required in the control equations for perfusion and ventilation.

In one of the first models to predict survival time in cold water Timbal et al. (80) used ambient temperature, morphological characteristics and environmental conditions to predict physiological responses and the thermal state of humans to cold air and water exposure. Hayward et al. (33) predicted survival

time with the aid of a model in terms of water temperature. Shortly after this he designed a new model using core and skin temperature to improve the first model (34). These models were based on regression equations.

In 1985 Wissler designed a general purpose model (88) consisting of a number of cylindrical elements representing longitudinal segments of the head, trunk, arms and legs. Each segment consisted of bone, fat, skin and had a vascular system composed of three parts representing the arteries, veins and capillaries. The circulatory path was faithfully reproduced in the sense of arterial blood flowing into capillaries of the same segment and into the arteries of the downstream segment. Blood from the capillaries flowed into the veins, mixing with venous blood from more distal segments. In the lungs, exchange of both heat and mass ($O_2$ and $CO_2$) occurred. Within a given segment, density and specific heat did not depend on time, but other variables such as temperature, oxygen ($O_2$) tension, metabolic and blood perfusion rate were defined by physiological control equations.

Tikuisis developed models to describe the shivering response (78) and the role of body fat in the prediction of the metabolic response (79). In 1989 Tikuisis (76) improved these previous models by including clothing properties. This model showed that clothed immersion in water introduces two important considerations: 1) Skin temperatures are not clamped near that of water and they can vary widely

among various body segments, and 2) Thermophysical properties of clothing must be taken into account when predicting thermal response to cold water immersion. He also demonstrated that a small proportion of heat is lost through respiration and that it increases with increased metabolic heat production as described by Burton (7).

A dynamic model of the human/clothing/environment-system was developed in 1997 (93). This model comprised of six segments consisting of the head, trunk, arms hands, legs and feet. Each segment is further divided into core, muscle, fat and skin. Thermoregulatory responses (vasomotion and metabolic heat production) were activated by the afferent signal composed of the weighted temperatures measured by thermal receptors distributed within the body. The model considers different blood flow patterns for skin and muscle and also cold-induced vasodilation. Additionally a combined model was created to investigate the interaction between the human body, clothing and the environment. A comparison with experimental data showed that this model could be applied in a wide range of environmental conditions.

Xu (91) estimated the contributions of different mechanisms of brain cooling during cold-water near-drowning using a two dimensional mathematical model. The mechanisms included conductive heat loss through tissue to the water at the head surface and in the upper airway and circulatory cooling to aspirated

water via the lung and via venous blood returning from the scalp. Results of this model showed that heat loss through the scull and upper airways are minimal, and that it's contribution to total heat loss decreases with age. He predicts that ventilation of the cold water may provide substantial brain cooling through circulation of the cold blood. It seems possible that peripheral cooling of the cortex and central cooling of the brainstem via the upper airway could also play a role. His model is verified by experiments on anesthetized dogs, cadavers and one study done on a human.

A multi segmental mathematical model was also developed to predict shivering and thermoregulatory responses during long-term cold exposure (92). It incorporated new knowledge on shivering thermogenesis including the control and maximal limits of its intensity, inhibition due to a low core temperature, and prediction of endurance time. The model also takes into account individual characteristics of age, height, weight, %body fat, and maximum aerobic capacity. The model was validated against three different cold conditions i.e. water immersion up to 38 h and air exposure.

Xu (90) designed another cold thermoregulatory model to examine mechanisms underlying the individual differences in response to cold exposure between partially immersed subjects. Subcutaneous fat percentage, shivering and muscle blood flow during two different immersion depths, were the mechanisms

under investigation. The model suggested that increased calf muscle blood flow for the fueling of shivering heat production, in low fat individuals could in part cause the individual differences in core temperature responses.

## Structural equation modeling in experimental research

Although the initial models described above were regression models, mathematical models in thermophysiology transformed into theoretical, deterministic models including as many of the biophysical and physiological characteristics that have been proven to affect changes in core temperature or shivering heat production (75,76,78,92,95-98). Verification for these models mainly comes from research studies attempting to isolate one of these factors or case studies of people in real life situations. Generally experimental studies on cold water immersion and submersion aim to control for physical factors such as water temperature and exposure time and even shivering heat production through the experimental design. The effect of other factors known to affect cooling rate such as body fat, the ability to shiver, age etc. are controlled for through the use of repeated measures analysis of variance (ANOVA). This approach controls for between-subject differences, but not necessarily for between-condition differences. The relatively small sample sizes of these studies limit the use of analytical methods that would allow for the studying of more variables. Another problem with this approach is that only measurable variables could be

studied. Several studies (1,22,47,63,86,95) have speculated about the significance of size of the thermal core in thermoregulation studies. Although it is possible to measure changes in the thermal core through the change in skin blood flow, the actual size is difficult to measure (90).

Structural Equation Modeling (SEM) as an analytic technique could possibly help to address the above mentioned difficulties. Presently SEM is commonly used in the social sciences (81) and Econometrics (2) while the technique is limited in applied physiology. In one of these applied physiology studies, SEM was used to study the stability and change in genetic and environmental influences on hand-grip strength in 152 pairs of older male twins. Measurement of handgrip strength 10 years apart were the only measured variables, while genetic, shared environmental and non-shared environmental influences were latent variables in the model (13). Another study (43) modeled perceived work demands and felt stress as latent variables as indicators of musculoskeletal neck/shoulder symptoms among 148 elderly female computer users. Only one study (57) in thermophysiology has used this analysis to demonstrate how additional information can be drawn from physiological ecology and general organismal studies by applying SEM when multiple variables are measured in the same individuals. In this study body mass, body length and foot length of the leaf-eared mouse was used as indicators of body size, a latent variable, and results

were compared between cold- and warm-acclimated animals. In all these studies, although quite different from each other, latent variables were included in their models, so that SEM would be a logic option to do the analysis.

## The structural equation model

Structural equation modeling (SEM) is a statistical technique for testing and estimating causal relationships, rather encouraging confirmatory than exploratory modeling. This view of SEM was articulated by the geneticist Sewall Wright (1921), the economists Trygve Haavelmo (1943) and Herbert Simon (1953).

Structural Equation Modeling is a very general analytic framework (81), allowing the researcher an interplay between theory and data (37) and a very powerful multivariate analysis technique that includes specialized versions of a number of other analysis methods as special cases such as:

1. Causal modeling involving either manifest variables, hypothetical or latent variables, or both (42);

2. Confirmatory factor analysis, an extension of factor analysis[6] in which specific hypotheses about the structure of the factor loadings and

[6] A technique to reduce the number of variables and to detect structure in the relationships between variables.

intercorrelations are tested (42);

3. Second order factor analysis, a variation of factor analysis;

4. Regression models, an extension of linear regression analysis[7] in which regression weights may be constrained to be equal to each other, or to specified numerical values;

5. Covariance structure models which hypothesize that a covariance matrix has a particular form and

6. Models errors in measurements for observed variables (37), reducing the error on the outcome variable.

7. Statistically test a priori and measurement assumptions against empirical data (37).

For the purpose of this study attention will be given to the combination of causal modeling and confirmatory factor analysis (structural regression model) as a method of testing the theory about factors affecting core temperature drop when the whole head is submersed in cold water.

---

[7] A term first used by Pearson, 1908, and the purpose was to learn more about the relationship between several independent or predictor variables and a dependent or criterion variable.

The experimental design is used by many researchers, including thermophysiologists, as an effective method to test theory (81) and more specifically causal relationships between variables as well as testing the experimental design (2). In the simplest case one would examine the effect of an independent variable, x, on a dependant variable y, where x and y could consist of qualitative variables (e.g. nominal or ordinal), quantitative variables (e.g. interval or ratio), or combinations of qualitative and quantitative variables (2). The relationship between the two variables is presented as: x —> y (2).

In some cases the independent variable would be a dichotomous nominal variable with a continuous dependant variable. Two groups of people will be tested, those affected by or treated with x, the independent variable and those not affected or treated with x. Depending on the study more groups could also be included e.g. a control without condition. The effect of x on y, the dependant variable, would be tested with the aid of the t- test or analysis of variance (ANOVA) where the means of y of the groups will be compared (2,30). For more than one dichotomous variable a multivariate ANOVA or repeated measures ANOVA would be the traditional choice of models. The difference between two groups will then be tested for significance using the F-test or p-values (30). In cases where one or more of the independent variables is continuous, when cell frequencies in a factorial design are unequal and disproportionate or when trends

in data are studied, multiple regression analysis is appropriate for determining whether a relation exists between x and y. R squared will determine the significance of the relation (2).

In addition to determining a significant relationship, one could also determine the strength or magnitude of a relationship using the omega ($\Omega$) squared index. This index indicates the amount of variance explained or accounted for by the experimental treatment (2).

Despite the valuable contribution of the above mentioned techniques in the process of determining causal effects of variables, there are some limitations which could be illustrated when we look at the equation representing the model above.

$$y = a + bx + e \qquad \text{(equation 1)}$$

where x and y respectively are the observed independent and dependent variables, a is the value when $x = 0$, b is the effect of x on y and e is an error term. The assumption is made that any change in y is due to the direct effect of a manipulation in x and it does not take in account the effect the change has on other variables also affecting y.

Although both the above mentioned models (ANOVA and regression analysis) also have the ability to model the effect of more than one variable as

well as interactions between these variables, there are still short comings that make these models ineffective to fully explain the change in the outcome variable.

The first reason is that although the researcher usually controls for as many factors possible, application of the ANOVA or regression model to experimental data only consisting of measured variables cannot determine whether (1) the significant F indicates that the manipulation alone produced the difference in the dependant variable, (2) other variables not measured or controlled for or artifacts produced the difference or (3) both these unaccounted factors and the manipulation produced the difference (2). Using the whole head cooling study (64) as an example, we see that cooling the head increases core cooling disproportionally to increased heat loss. Although the difference in means between the two groups is different, it does not explain the reason for the increased rate of core cooling. Only if the increased core cooling is due to measurable effects such as heat loss, water temperature etc. traditional methods could help explain the effect of an independent variable on core cooling. In this study however, it was speculated that a reduced thermal core was responsible for the increased cooling rate. The size of the thermal core was not measured, and therefore its contribution to the results could only be speculated and not modeled. The use of SEM for the analysis of the data would allow the researcher to include latent variables in the model to explain the role of unobserved factors.

The second problem with the traditional models is that the only error measurement is on the dependent variable and that the independent variables are presented as measured without error (2). This error is an indication of how well the independent variable(s) explain(s) the outcome variable. A large error might indicate the involvement of other variables in the outcome of a variable. For a full interpretation of the nature of the causal relationship between variables, it is necessary to model the degree of measurement error on all variables in a model (2). In a SEM model, we not only have the error on the outcome variable, but also on all other variables that have a causal relation with any other dependant variable. Modeling this error term, improves the overall fit of a model. It is not only in the sharing of the error that we reduce model error but also in the inclusion of explanatory variables to reduce the unexplained error variance.

A third problem applicable to experimental research in applied physiology is the effects dependant variables have on each other and how that affects the outcome or dependent variable. In the traditional model the only effect studied is the direct effect independent variables or an interaction of independent variables on the outcome variable. It does not explain the causal relationship between any of the independent variables, nor how that affects the outcome variable. To illustrate an example is taken from cooling studies. It is already known that both shivering and increased subcutaneous fat attenuate the rate of

core cooling. Although both these effects could be shown in a multiple regression model, the suppressing effect that increased subcutaneous fat has on the amount of shivering could not be shown. These independent variables, which is also affected by other independent variables (shivering in this case) as well as the outcome variable is now referred to as endogenous variables, opposed to the other independent variables which are considered exogenous variables (31,42).

## Structure of a SEM model

- *The Path Model:*

Path analysis (PA) from the SEM family is a possible technique when there is only a single measure of each theoretical variable and the researcher has prior hypotheses about causal relations among these variables. The starting point is the specification of a structural model that presents all causal hypotheses. When only observed variables are included in a model, the structural model is called a path model (42). The models used in for the purpose of this document will use the symbols from an approach to causal modeling called reticular action modeling (RAM) as used by (42).

Consider the path model as presented in Figure 1. All observed variables are presented with rectangles. An arrow represents a causal effect of the variable it is directed from on the variable it is directed to. A variable with no

arrows pointing toward it is an exogenous variable (e.g. VAR 1 and VAR 2) and their causes are either unknown and/or not of any concern in the study. Measured variables with arrows directing to them (e.g. VAR 3, VAR 4 and VAR 5) are considered endogenous variables. They too, as with variable VAR 3 and VAR 4 in Figure 1, could have arrows pointing away from them. This indicates a mediator effect of the endogenous variable affecting another endogenous variable (e.g. VAR 3 and VAR 4 both mediating the effect of VAR 1 on VAR 4). Circles or ellipses usually represent unmeasured or latent variables. In a path diagram these circles associated with endogenous variables model the error of the observed variable, and represent all unmeasured causes of that variable as well as measurement error. It is also referred to as disturbances (D) and considered exogenous variables.

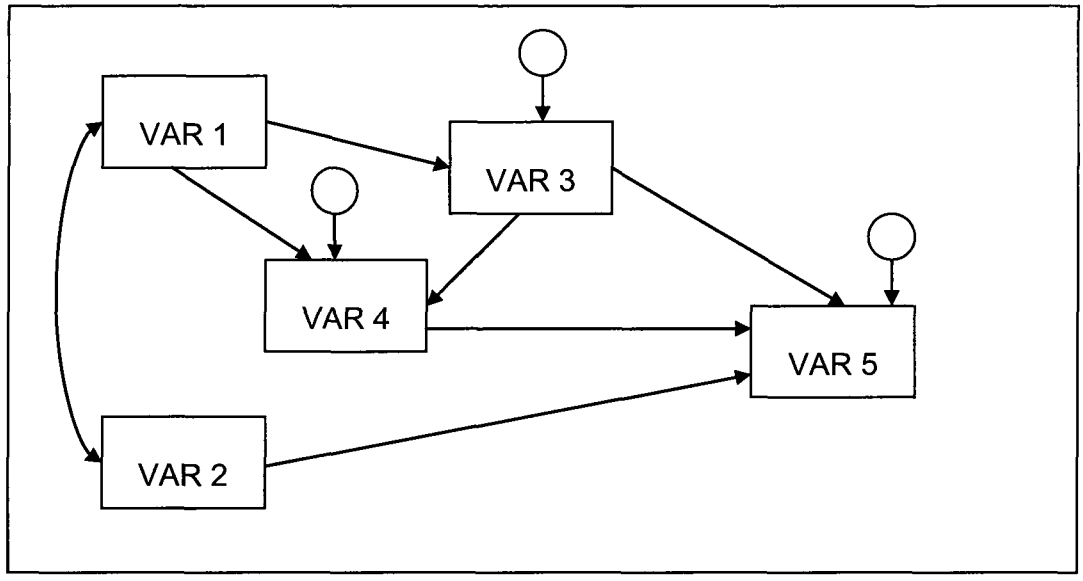


Figure 1. A path model

*Curved lines with two arrowheads, as the line connecting VAR 1 and VAR 2,* represents the covariance (or correlation in standardized form) between two variables and designates the unanalyzed association between two exogenous variables (42). The curved arrows exiting and entering the same exogenous variable or disturbances (unmeasured exogenous variables), represent the variance of these variables.

- *Confirmatory Factor Analysis:*

As in many other research areas, all biophysical and physiological variables affecting an outcome variable in thermal physiology, could not be measured, and therefore remain theoretical. In none of the traditional analysis techniques, such

as ANOVA and multiple regression could these factors be accommodated. However, in SEM, such variables could be included as latent or unobserved variables in a standard confirmatory factor analysis (CFA) measurement model (Figure 2). In these models the latent variables are considered exogenous variables, having a causal affect on measurable variables (indicators or endogenous variables).

Lines with single arrowheads point from the latent variables to the indicators are called factor loadings and are generally interpreted as regression coefficients that may be in the standardized or unstandardized form (42). Each indicator has an error term on it and the arrow pointing from the error to the indicator represents all other factors (not included in the model) having an effect on the latent variable. Each error term as well as the latent variable has a curved arrow, exiting and entering the same variable and is considered the variance on that variable. The measurement errors are independent of each other and of the factor (42).

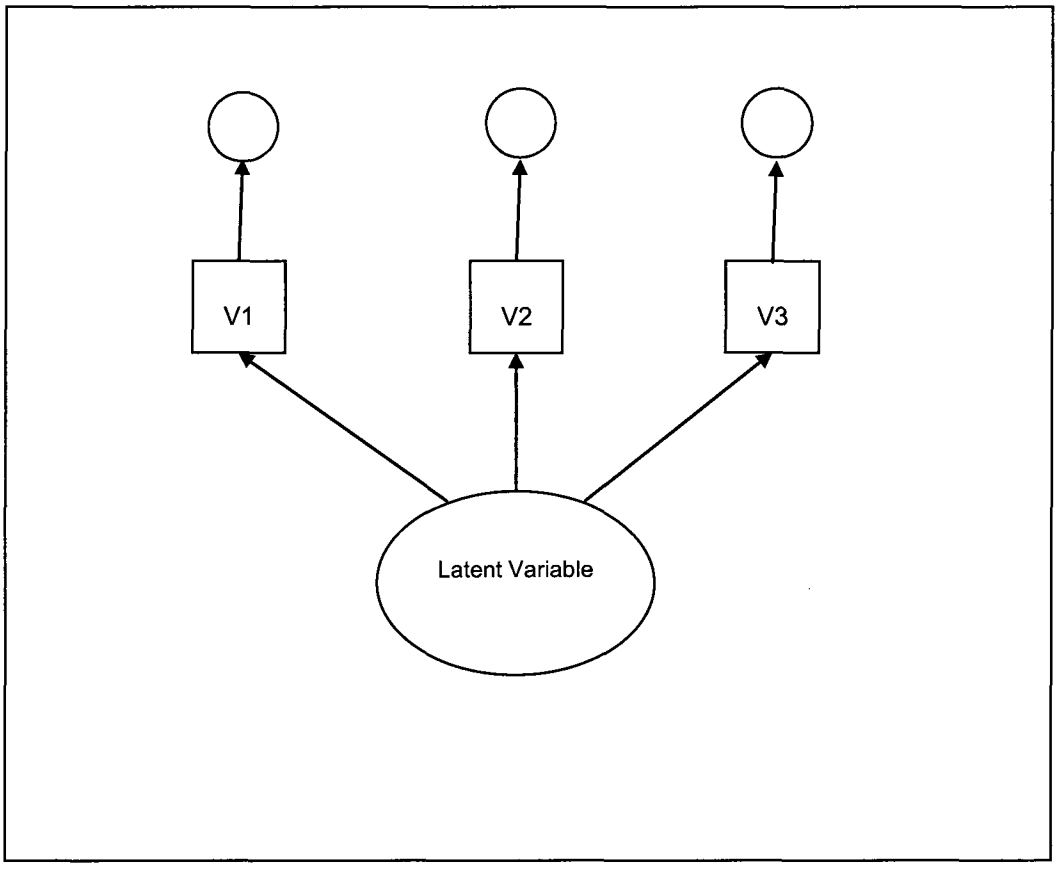


Figure 2. A Confirmatory Factor Analysis Model

- *Structural Regression Models:*

Models with both structural and measurement components are the most general of all SEM models (81) and also called structural regression (SR) models, hybrid or Lisrel models (42). These models as path models, allow for the testing of hypotheses of causal effects (42), and also help in diagnosing problems in the research design (2,19).

Necessary conditions for Path Analysis, Confirmatory Factor Analysis and hybrid models using covariance based SEM :

1. Latent variables should have at least three measured indicators. These indicators should be continuous (42) and should assume at least four values (31)

2. Normally distributed data (31,42).

3. Absence of multicollinearity.

4. Inclusion within the model of all nontrivial causal variables (31), and that all relations are linear.

5. Overidentified model (31). This requires that there should be at least as many observations as free model parameters so that the degrees of freedom (df) $\geq 0$ (42).

6. Minimal observations: Larger sample sizes are always preferable, and a sample size of 100 (42) or 5 observations per parameter to be estimated would be acceptable (31).

7. For more than one latent variable in one confirmatory model, the associations between the factors are unanalyzed and should be included as such (31,42).

8. Every latent variable should have a scale (metric) (42). This is ensured through assigning a value of one to one of the factor loadings of each latent variable (42).

## Small Sample Sizes in SEM.

One of the biggest challenges using a SEM model for the analysis of our data will be the small sample sizes typically used in this field to demonstrate differences between conditions. The covariance-based approach for SEM using maximum-likelihood (ML) function, involves amongst the other assumptions listed in the previous paragraph, bigger sample sizes (> 200) which is beyond the reach of our laboratory. Not having a sufficient number of subjects leads to poor parameter estimates, negative variances, out of range covariances and a potential for a Type II error. These tendencies increase as sample sizes decrease (37).

One way to address the problem of small sample size as we typically have in our area of research is with the use of bootstrapping (41). Bootstrapping was developed to provide standard errors and confidence intervals for regression coefficients and predicted values in situations in which the standard assumptions are questionable. Although the concept is simple, the procedure requires extensive computation time. In principle you assume that your sample is actually the population and you draw x number of samples (x should be more than 1000) from the original sample with replacement. With replacement indicates that each

observation may be selected more than once. For each bootstrap sample, the regression results are computed and stored.

Suppose that you want the standard error and a confidence interval of any correlation. The bootstrap sampling process has provided x estimates of all the correlations. The standard deviation of these x estimates of the correlation is the bootstrap estimate of the standard error. The bootstrap confidence interval is found by arranging the x values in sorted order and selecting the appropriate percentiles from the list. The main purpose when using the bootstrap method is to approximate the population (41).

## CONCLUSION

Cold water studies done on different levels of head immersion/submersion (with shivering suppressed or not) (1,25,46,64) all showed that heat loss from any part of the head has a significant effect on core cooling rate when the body is exposed to the cold water and supposedly vasoconstricted. This drop in core temperature is disproportional to the added heat loss through the head. In the only study done on whole head cooling, it was shown that this relatively small increase in heat loss also affected the core temperature drop when the body was insulated from the cold water. Consequent to submersion and cooling of the scalp

and face, thermal stimulation and the human dive response likely reduced the perfused body mass, thus a small increase in total heat loss caused a relatively large cooling of the body core.

Further work is warranted to study whole head cooling in shivering humans and also to quantify the effects of scalp and/or face immersion in cold water on regional blood flow and alterations in the mass of the thermal core.

It is also clear that research on cold water immersion and submersion is complicated in many ways. Firstly, the many factors that play a role in the development of hypothermia and the integration of their effects make it difficult to show the net effect of an isolated factor. Secondly these factors vary from factors which could easily be measured, such as skin temperature to factors which are difficult to measure such as the size of the thermal core. In traditional research analysis the role of these unmeasured factors could only be speculated. However, Structural Equation Modeling as a multivariate analysis technique has the potential to assist in both these problems. It has the ability to show the effect of all factors, not only on the outcome or studied variable, but also all other factors involved. It furthermore has the ability to model latent or unmeasured variables by means of measurable indicators through confirmatory factor analytic models. These latent variables can then be included as a variable in a structural regression model.

## REFERENCES

1. Alexander L. The Treatment of Shock from Prolonged Exposure to Cold, Especially in Water. Washington, DC: Office of the Publication Board, Department of Commerce. (Combined Intelligence Objectives Sub-committee), 1946, p. 1-228.

2. Bagozzi R. Structural Equation Models in Research. Journal of Marketing reasearch XIV: 209-226, 1977.

3. Bernard S, Gray T, Buist M, Jones B, Silvester W, Gutteridge G , and Smith K. Treatment of Comatose Survivors of Out-of-Hospital Cardiac Arrest with Induced Hypothermia. N. Engl. J. Med. 346: 557 - 563, 2002.

4. Bolte RG, Black PG, Bowers RS, Thorne JK and Corneli HM. The use of extracorporeal rewarming in a child submerged for 66 minutes. JAMA 260: 377-379, 1988.

5. Brengelmann GL. Dilemma of Body Temperature Measurement. In: Man in Stressful Environments: Thermal and Work Physiology, 1987, p. 5-22.

6. Burke WEA and Mekjavic IB. Estimation of regional cutaneous cold sensitivity by analysis of the gasping response. J. Appl. Physiol. 71: 1933-1940, 1991.

7. Burton AC. Man in a Cold Environment. London: Edward Arnold Ltd., 1955.

8. Busto R, Dietrich WD, Globus MY-T, Valdes I, Scheinberg P and Ginsberg MD. Small differences in intraischemic brain temperature critically determine the extent of ischemic neuronal injury. J. Cereb. Blood Flow Metab. 7: 729-738, 1987.

9. Busto R, Globus MY, Dietrich WD, Martinez E, Valdes I and Ginsberg MD. Effect of mild hypothermia on ischemia-induced release of neurotransmitters and free fatty acids in rate brain. Stroke 20: 904-910, 1989.

10. Cabanac M and Massonnet B. Thermoregulatory responses as a function of core temperature in humans. J. Physiol 265: 587-596, 1977.

11. Camboni D, Phillip A, Schebesch K and Schmid C. Accuracy of core temperature measurement in deep hypothermic circulatory arrest. Interactive CardioVascular and Thoracic Surgery 7: 922-924, 2008.

12. Campbell L. Simultaneous calf and forearm bloodflow during immersion in man. Austr. J. Exp. Biol. Med. Sci. 47: 747 - 754, 1969.

13. Carmelli D and Reed T. Stability and change in genetic and environmental influences on hand-grip strength in older male twins. J. App. Physiol. 89: 1879 - 1883, 2000.

14. Colbourne F, Sutherland G and Corbett D. Postischemic hypothermia: A critical appraisal with implications for clinical treatment. Mol Neurol. 14: 171-201, 1997.

15. Conn AW, Miyasaka K, Katayama M, Fujita M, Orima H, Barker G and Bohn D. A canine study of cold water drowning in fresh versus salt water. Crit. Care Med. 23: 2029-2037, 1995.

16. Cook D, Orszulak T, Daly R and Buda D. Cerebral hyperthermia during cardiopulmanary bypass in adults. J. of Thoracic and Cardiovascular Surgery 111: 672-676, 1996.

17. Cooper JO, McMurray S, Pavlin EG, Schoene RB and et.al. The effect of meperidine and naloxone on cold-induced shivering. Anesthes 73: A230, 1990.

18. Cooper KE, Martin S and Riben P. Respiratory and other responses in subjects immersed in cold water. J. Appl. Physiol. 40: 903-910, 1976.

19. Costner H. Utilizing Causal models to discover flaws in models. Sociometry 34: 398-410, 1971.

20. Dalton W and Kuluz J. New research in the field of stroke: Therapeutic hypothermia after cardiac arrest. Stroke 34: 1051-1053, 2003.

21. Dietrich WD, Busto R, Halley M and Valdes I. The importance of brain temperature in alterations of the blood-brain barrier following cerebral ischemia. J. Neuropathol. Exp. Neurol. 49: 486-497, 1990.

22. Eyolfson D, Tikuisis P and Giesbrecht GG. Measurement and prediction of maximal shivering capacity in humans. In: Proceedings of the 8th Int. Conf. on Environ. Ergonomics, edited by Hodgdon J and Heaney J. San Diego: NHRC, 1999.

23. Froese G and Burton AC. Heat losses from the human head. J. App. Physiol. 10: 235-241, 1957.

24. Giebrecht G and Wilkerson J. Hypothermia, Frostbite and other cold injuries. Seattle, WA: The Mountaineers Books, 2006.

25. Giesbrecht G, Lockhart T, Bristow G and Steinman A. Thermal effects of dorsal head immersion in cold water on nonshivering humans. J. Appl. Physiol. 99: 1958-1964, 2005.

26. Giesbrecht G and Steinman AM. Immersion in cold water. In: Wilderness Medicine (5th ed.), edited by Auerbach P. Philadelphia, PA: Mosby, 2007, p. 160-188.

27. Glickman-Weiss E, Cheatham C, Caine N, Blegen M, Marcinkiewicz J and Mittleman K. The Influence of Gender and Menstrual Phase on Thermosensitivity During Cold Water Immersion. Aviat. Space Environ. Med. 71: 715-722, 2000.

28. Globus MY, Busto R, Lin B, Schnippering H and Ginsberg MD. Detection of free radical activity during transient global ischemia and recirculation: effects of intraischemic brain temperature modulation. J. Neurochem. 65: 1250-1256, 1995.

29. Grayson J. Responses of the microcirculation to hot and cold environments. In: Thermoregulation: Physiology and Biochemistry, edited by Schönbaum E and Lomax P. New York: Pergamon Press Inc., 1990, p. 221-234.

30. Hassard T. Understanding Biostatistics. St Louis: Mosby - Year Book Inc., 1991.

31. Hatcher L. A step-bystep approach to using SAS for factor analysis and structural equation modeling. Cary, NC: SAS Institute Inc., 1994.

32. Hayward JS, Eckerson JD and Collis ML. Effect of behavioral variables on cooling rate of man in cold water. J. Appl. Physiol. 38: 1073-1077, 1975.

33. Hayward JS, Eckerson JD and Collis ML. Thermal balance and survival time prediction of man in cold water. Can. J. Physiol. Pharmacol. 53: 21-32, 1975.

34. Hayward JS, Eckerson JD and Collis ML. Thermoregulatory heat production in man: prediction equation based on skin and core temperatures. J. Appl. Physiol. 42: 377-384, 1977.

35. Heistad DD, Abboud F and Eckstein J. Vasoconstrictor response to simulated diving in man. J. Appl. Physiol. 25: 542-549, 1968.

36. Hertzman A and Roth L. The Absence of Vasoconstrictor Reflexes in the Forehead Blood Circulation. Effects of cold. Am J Physiol 136: 692-697, 1942.

37. Hoyle R. Statistical Strategies for small sample sizes. Thousand Oaks, California: Sage Publications, Inc., 1999.

38. Hurwitz B and Furedy J. The human dive reflex: An experimental, topographical and physiological analysis. Physiol. Behav. 36: 287-294, 1986.

39. Jacobs I, Martineau L and Vallerand AL. Thermoregulatory thermogenesis in humans during cold stress. In: Exercise and Sport Science Reviews, edited by Holloszy JO. Baltimore: Williams & Wilkins, 1994, p. 221-250.

40. Johnston CE, Bristow GK, Elias DA and Giesbrecht GG. Alcohol lowers the vasoconstriction threshold in humans without affecting core cooling rate during mild cold exposure. Eur. J. Appl. Physiol. 74: 293-295, 1996.

41. Joreskog K and Sorbom D. Lisrel 8: User's reference guide. Chicago, IL: Scientific Software International, 1996.

42. Kline R. Principles ad practice of structural equation modeling. New York: Guilford, 1998.

43. Larsman P, Sandsjo L, Klipstien A, Vollenbroek-Hutten M and Christensen H. Perceived work demands, felt stress, and musculoskeletal neck/shoulder symptoms among elderly female computor users. The NEW study. J App Physiol 96: 127-135, 2006.

44. Leonov Y, Stertz F and Safar P. Mild cerebral hypothermia during and after cardiac arrest improves neurologic outcome indogs. J. Cereb. Blood Flow Metab. 10: 57-70, 1990.

45. Letsou GV, Kopf GS, Elefteriades JA, Carter JE, Baldwin JC and Hammond GL. Is cardiopulmonary bypass effective for treatment of hypothermic arrest due to drowning or exposure? Arch. Surg. 127: 525-528, 1992.

46. Lockhart T, Jamieson C, Steinman A and Giesbrecht G. Life jacket design affects dorsal head and chest exposure, body core cooling and mental performance in 10°C water. Aviat. Space Environ. Med. 76: 954-962, 2005.

47. Machte W and Hatch TF. Mass exchanges in physiological responses. Physiol. Rev. 27: 200-227, 1947.

48. Mcclain C, Soranio S and Rockoff M. The brain and glands. In: A practice of anesthesia for infants and children, edited by Cote C, Lerman J and Todres D. Philadelphia, PA: Elsevier Inc., 2009, p. 491-557.

49. Mekjavic I and Eiken O. Contribution of thermal and nonthermal factors to the regulation of body temperature in human beings. J Appl Physiol 100: 2065-2072, 2006.

50. Mekjavic IB, Sundberg CJ and Linnarsson D. Core temperature "null zone". J. Appl. Physiol 71: 1289-1295, 1991.

51. Mellergard P. Changes in Human Intracerebral Temperature in Response to Different Methods of Brain Cooling. Neurosurgery 31: 671-677, 1992.

52. Michenfelder JD and Milde JH. The relationship among canine brain temperature, metabolism, and function during hypothermia. Anesthesiology 75: 130-136, 1991.

53. Minamisawa H, Nordstrom CH, Smith ML and Siesjo BK. The influence of mild body and brain hypothermia on ischemic brain damage. J. Cereb. Blood Flow Metab. 10: 365-374, 1990.

54. Minamisawa H, Smith ML and Siesjo BK. The effect of mild hyperthermia and hypothermia on brain damage following 5, 10, and 15 minutes of forebrain ischemia. Ann. Neurol. 28: 26-33, 1990.

55. Moran DS and Mendal L. Core temperature measurement; methods and current insights. Sports Med. 32: 879-885, 2002.

56. Nelson DA and Nunneley SA. Brain temperature and limits on transcranial cooling in humans: quantitative modeling results. Eur. J. Appl. Physiol. 78: 353-359, 1998.

57. Nespolo R, Arim M and Bozinovic F. Body size as a latent variable in a structural equation model: thermal acclimation and energetics of the leaf-eared mouse. J. Exp.Biol. 206: 2145-2157, 2003.

58. Norberg WJ, Agnew RF, Brunsvold R, Sivanna P, Browdie DA and Fisher D. Successful resuscitation of a cold water submersion victim with the use of cardiopulmonary bypass. Crit. Care Med. 20: 1355-1357, 1992.

59. Nybo L, Secher N , and Nielsen B. Inadequate heat resease from the human brain during prolonged exercise with hyperthermia. J Physiol 545: 697-704, 2002.

60. Parsons K. The Thermal Properties of Clothing. In: Human Thermal Environments. Bristol: Taylor & Francis Inc., 1993, p. 103-130.

61. Passias T, Meneilly G and Mekjavic I. Effect of Hypoglycimia on thermoregulatory responses. J App Physiol 80: 1021, 1996.

62. Pennes HH. Analysis of tissue and arterial blood temperatures in the resting human forearm. J. Appl. Physiol. 1: 93-122, 1948.

63. Pretorius T. Whole head exposure and its effect on the cooling rate of body core temperature. (Research). Winnipeg: University of Manitoba, 2005.

64. Pretorius T, Bristow G, Steinman A and Giesbrecht G. Thermal effects of whole head submersion in cold water on non-shivering humans. J. Appl. Physiol. 101: 669-675, 2006.

65. Pugh LGCE. Accidental hypothermia in walkers, climbers, and campers: Report to the medical commission on accident prevention. British Medical J. 1: 123-129, 1966.

66. Rasch W, Samson P, Cote J and Cabanac M. Heat loss from the human head during exercise. J. Appl. Phsyiol. 71: 590-595, 1991.

67. Rowell LB, Freund PR and Brengelmann GL. Cutaneous vascular response to exercise and acute hypoxia. J. Appl. Physiol. 53: 920-924, 1982.

68. Sagawa S, Shiraki K, Yousef MK and Konda N. Water temperature and intensity of exercise in maintenance of thermal equilibrium. J. Appl. Physiol. 65: 2413-2419, 1988.

69. Sawka M and Wenger C. Physiologic responses to acute exercise heat stress. In: Human performance physiology and environmental medicine at terrestrial extremes, edited by Pndolf K, Sawka M and Gonzalez R. Indianapolis (IN): Benchmark Press, 1998, p. 97-151.

70. Sessler DI. Thermoregulation and heat balance: general anesthesia. Advances in Pharmacological Sciences: Thermal Balance in Health and Disease: 251-265, 1994.

71. Shaaban Ali M, Harmer M and Kirkham F. Cardiopulmonary bypass temperature and brain function. Anaesthesia 60: 365-372, 2005.

72. Shiraki K, Konda N and Sagawa S. Esophageal and tympanic temperature responses to core blood temperature changes during hypothermia. Journal of Applied Physiology 61: 98-102, 1986.

73. Steinman AM, Hayward JS, Nemiroff MJ and Kubilis PS. Immersion hypothermia: comparative protection of anti-exposure garments in calm versus rough seas. Aviat. Space Environ. Med. 58: 550-558, 1987.

74. Stolwijk JA and Hardy JD. Temperature regulation in man- A theoretical study. Pflugers Arch. 291: 129-162, 1966.

75. Stuart M, Lee S and Williams W. Core temperature measurement during supine exercise: esophageal, rectal and intestinal temperature. Aviation Space Environ Med 71: 939-945, 2000.

76. Tikuisis P. Prediction of the thermoregulatory response for clothed immersion in cold water. Eur. J. Appl. Physiol 59: 334-341, 1989.

77. Tikuisis P. Prediction of survival time for cold exposure, 1995.

78. Tikuisis P, Gonzales RR and Pandolf KB. Thermoregulatory model for immersion of humans in cold water. J. Appl. Physiol. 64: 719-727, 1988.

79. Tikuisis P, Gonzalez RR, Oster and Pandolf KB. Role of body fat in the prediction of the metabolic response for immersion in cold water. Undersea Biomed. Res. 15: 123-134, 1988.

80. Timbal J, Boutelier C, Loncle M and Bougues L. Comparison of Shivering in Man Exposed to Cold in Water and in Air. Euro. J. of Phsyiol., 1976.

81. Tomarken A and Waller N. Structural Equation Modeling: Strenghts, Limitations and Misconceptions. Annu. Rev. Clin. Psychol. 1: 31-65, 2005.

82. Vanggaard L, Eyolfson D, Xu X, Weseen G and Giesbrecht GG. Immersion of distal arms and legs in warm water (AVA Rewarming) effectively rewarms hypothermic humans. Aviat. Space Environ. Med. 70: 1081-1088, 1999.

83. Wagner JA and Horvath SM. Cardiovascular reactions to cold exposures differ with age and gender. J. Appl. Physiol. 58: 187-192, 1985.

84. Wagner JA and Horvath SM. Influences of age and gender on human thermoregulatory responses to cold exposures. J. Appl. Physiol. 58: 180-186, 1985.

85. Wang H, Wang D, Olivero W, Lanzino G, Honings D, Rodde M, Burnham J, Milbrandt J and Rose J. Selective brain hypothermia can be achieved with a cooling helmet. J. Neurosurg. 100: 272-277, 2004.

86. Wissler EH. Steady state temperature distribution in man. J. Appl. Physiol. 16: 734-740, 1961.

87. Wissler EH. A mathhematical model of the human thermal system. Bull. Math. Biophys. 26: 147, 1964.

88. Wissler EH. Mathematical simulation of human thermal behavior using whole body models. In: Heat Transfer in Medicine and Biology, edited by Shitzer A and Eberhart RC. New York: Plenum Press, 1985, p. 325-373.

89. Wyndham CH and Atkins AR. An approach to the human biothermal problem with the aid of an analog computor. Conference on medical electronics, London, 1966.

90. Xu X, Castellani JW, Santee W and Kolka M. Thermal responses for men with different fat compositions during immersion in cold water at two depths: prediction vs observation. Eur. J. of Physiol. 100: 79-88, 2007.

91. Xu X, Tikuisis P and Giesbrecht G. A mathematical model for human brain cooling during cold-water near-drowning. J. Appl. Physiol. 86: 265-272, 1999.

92. Xu X, Tikuisis P, Gonzales L and Giesbrecht GG. Thermoregulatory model for prediction of long-term cold exposure. Comput. Biol. Med. 38: 287-298, 2004.

93. Xu X and Werner J. A dynamic model of the human/clothing/environment - system. Appl. Human Sci. 16: 61-75, 1997.

94. Young AJ, Castellani JW, O'Brien C, Shippee RL, Tikuisis P, Meyer LG, Blanchard LA, Kain JE, Cadarette BS and Sawka MN. Exertional fatigue, sleep loss, and negative energy balance invrease susceptibility to hypothermia. J. Appl. Physiol. 85: 1210-1217, 1998.

# CHAPTER 2

# SHIVERING HEAT PRODUCTION AND CORE COOLING DURING HEAD-IN AND HEAD-OUT IMMERSION IN 17°C WATER

Running title: Thermal Effects of Whole Head Cooling

KEYWORDS: Hypothermia, Cold-water Immersion, Perfused body mass, Symptomless hypothermia, Thermal core

Chapter 2 is reprinted here with permission as it appears in: Pretorius T, Cahill F, Kocay S , and Giesbrecht GG. Shivering heat production and core cooling during head-in and head-out immersion in 17°C water. Aviat. Space Environ. Med. 79: 495-499, 2008.

Aerospace Medical Association

President

*Robert W. Weien, M.D., M.P.H.*

*Colorado Springs, Colorado*

*320 South Henry Street*

*Alexandria, VA 22314-3579*

*Phone: 703-739-2240*

*Executive Director*

*Russell B. Rayman, M.D.*

*Association Home Office*

Dear Thea Pretorius,

You hereby have permission to include the following article in your thesis:

Pretorius, T. Shivering heat production does not prevent core cooling from whole head submersion in 17°C water. Aviat Space Environ Med 2008; 79:495-499.

This article was published by the Aerospace Medical Association. You may deposit the accepted manuscript in your institutional repository.

Pamela Day

Managing Editor

MY CONTRIBUTION TO THE PUBLICATION. This work was done in totality in the Laboratory of Exercise and Environmental Medicine as a follow-up study for my MSc research project. In the present study we looked at the effect of whole head cooling on core temperature when the shivering response was in tact. With the help of the head of this laboratory and my supervisor, Dr Gordon Giesbrecht, we planned this study based on the previous study. The set up for the previous study was used exactly as it was. Assisted by my fellow student, Farrell Cahill, and Sheila Kokay we conducted all the experiments at which time data were collected regarding temperature (skin and core) as well as metabolism and heart rate. It took team effort to prepare the laboratory and to assure that the water temperature was kept constant and all instruments were running smoothly during the hour and a half of recording of data.

After collecting the data I was responsible for the analysis. I have written the manuscript in consultation with Dr Giesbrecht and we agreed on a journal to send it to. At this time the manuscript was sent to all authors for their approval. After two revisions in consultation with Dr Giesbrecht, the manuscript was accepted and I was responsible for the final correction of the galley proofs.

ABSTRACT: This study isolated the effect of whole head submersion in cold water, on surface heat loss and body core cooling, when the protective shivering response was intact. METHODS: Eight healthy males were studied in 17°C water under four conditions – the body was either insulated or uninsulated, with the head either above the water or completely submersed in each body-insulation sub-condition. RESULTS: After 45 minutes, submersion of the head increased core cooling both in the body-insulated sub-condition (head-out: 0.13±0.2 °C, head-in: 0.47±0.3 °C) and the body-exposed sub-condition (head-out: 0.40±0.3 °C and head-in: 0.73±0.6 °C). Submersion of the head (7% of the body surface area) in the body-exposed condition, increased total heat loss by 11%. DISCUSSION: In both body-exposed and body-insulated sub-conditions, head submersion increased core cooling disproportionally more than the increased total heat loss. In 17 °C water the head does not contribute relatively more than the rest of the body to surface heat loss. However, a cold-induced reduction of perfused body mass - stimulated by thermosensitive and/or trigeminal receptors in the scalp, neck and face - may allow this small increase in heat loss to cause the relatively larger cooling of the body core. These cooling affects of head submersion are not offset by any increase in shivering heat production.

## INTRODUCTION

Many recreational, commercial and military activities involve exposure to cold water and the possible development of accidental hypothermia. Several studies have addressed the effect of cold water immersion on the rate of body core cooling (20,21,24,30). The initiation and degree of hypothermia are related to many variables, including water temperature, insulation, duration of exposure and the amount of body surface area exposed to the water. The effect of whole head cold-water submersion on core cooling has only been studied in non-shivering humans (27) and the effect on shivering humans is not known.

One hypothesis predicts a substantial heat loss through the head due to the great amount of surface blood flow in the scalp and because scalp blood vessels do not vasoconstrict in response to cold as do surface vessels in other body areas (7). An alternative hypothesis predicts minimal heat loss from the head because submersion of the head and neck would only involve 7-9% more of the body surface area (BSA) (19). As well, mathematical modeling predicts minimal conductive heat loss directly through the scalp and skull (32). Two studies done on non-shivering humans (8,27) did not show heat loss from the head to be proportionally more than from the rest of the body. This topic has important practical implications for activities where the whole head is completely

submersed in cold water such as SCUBA diving or recreational cold water swimming (e.g., adventure races).

Although a few studies on core cooling have addressed whole head cooling in animals exposed to cold water (4) and in humans exposed to cold air (7,28), only one study exposed the whole human head to cold water (27) while three human studies involved cold water immersion of the dorsal head (1,8,22). In general, dorsal head immersion in cold water has little effect on core temperature when the remainder of the body is not cold stressed. However, when the body is also exposed to cold water, additional immersion of the dorsal head has increased core cooling by 250% in 1-2°C water (1)[8], 87% in 10°C water when shivering was intact (22), and by 40% in 12°C water when shivering was suppressed by demerol (8).

The latter study (8) was the first head-immersion protocol to measure surface heat loss while eliminating the confounding effect of shivering heat production. Surprisingly, heat loss from the immersed dorsal head was not proportionately greater than for other body areas. Nevertheless, the 40% increase in core cooling rate during dorsal head immersion was proportionately much greater than the 10% increase in surface heat loss from the dorsal head and

---

[8] These studies on prisoners of war in Dachau during World War II were grossly unethical, and the results are often considered invalid and unusable because of the emaciated condition of the prisoners as well as questions regarding the protocol and accuracy of the results.

upper chest. This exaggerated core cooling rate was proposed to result from the increased heat loss affecting a smaller thermal core due to intense thermal stimulation of the dorsal head with resultant peripheral vasoconstriction and reduced perfused body mass (i.e., thermal core) (31).

In toto, results from dorsal head immersion studies support this explanation; as an increased thermal stimulus (i.e., water temperature decreasing from 12°C to 1°C) would further reduce the thermal core, thus increasing core-cooling effect of dorsal head immersion. The comparative effect of whole head submersion has only been investigated in non-shivering humans (27). This study done in 17°C water, showed that whether the body was exposed to the cold water or not, whole head cooling resulted in a significant drop in core temperature after 30 minutes of exposure. Heat loss from the head (11%) was not proportionally greater than the body surface area it represents (7%). This confirms the findings of Xu (32) who used a mathematical model to consider physical properties of the skull and soft tissue of the head in predicting head heat loss and body core cooling.

The results in the whole head cooling study (27) were again proposed to arise from a reduced thermal core due to increased vasoconstriction (3,16). It was speculated that the thermal core was even more reduced in the body-insulated, head-in condition due to the increased thermal stimulus from the head.

This supports the findings of Campbell (3) and Heistad (16) that increasing the stimulus by additional submersion of the face would not only increase the thermal stimulus for vasoconstriction to reduce heat loss but, through trigeminal nerve afferents, it would also stimulate the human dive response. This is a mechanism that conserves oxygen by inducing bradycardia and peripheral vasoconstriction, while making the body core more susceptible to the development of hypothermia (31).

While previous work on non-shivering humans provides valuable insights for situations like cold-water-near-drowning and symptomless hypothermia experienced by SCUBA divers, similar conditions in shivering humans provide additional insights in thermoregulation of SCUBA divers and cold water swimmers in adventure races. The purpose of this study was to determine whether intact shivering affected the relative impact of whole head submersion on core cooling under similar conditions (water temperature = 17°C) to our previous work on non-shivering humans (27).

We hypothesized that whole head submersion could still increase core cooling disproportionally more than the increase in surface heat loss, even when shivering heat production would tend to attenuate core cooling.

# METHODS

## Subjects

The experimental protocol was approved by the University of Manitoba Education / Nursing Research Ethics Board. The study was open to men and women however only men volunteered. Eight male subjects, each of whom provided written, informed consent and proof of SCUBA diving certification, were studied. These subjects were both mentally and physically healthy, had no significant medical history and none had male-pattern baldness. They completed a medical (PAR-Q) questionnaire to screen for cardio-respiratory disease and other conditions that could be exacerbated by exposure to cold water. They were studied on four separate occasions, at least 24 hours apart and at the same time each day to control for circadian effects. Abstinence from alcohol, tobacco and strenuous exercise for 12 hours prior to the study was requested. They were instructed to only consume a light meal before they arrived for the study.

Height, weight, and skin fold thickness at four sites were measured; percent body fat was calculated based on body density, estimated from the sum of four skinfolds (2). The mean age of the subjects was 32.4±12 years; they were 179.6±5.1 cm tall and weighed 88.3±16 kg on average. The mean % body fat was 20.24±6% and the mean body surface area was 2.07±0.1 $m^2$.

## Instrumentation

For each trial, subjects wore a swimsuit while being instrumented in a room at an ambient temperature of 22°C. Core temperature was measured using a thermocouple in the esophagus ($T_{es}$) at the level of the cardiac atria. This site has previously been shown to provide the closest correlation to intra-cardiac temperature (15). Single-channel electrocardiogram and heart rate were also monitored.

Cutaneous heat flux ($W \cdot m^{-2}$) and skin temperature (°C) were measured from 12 sites (listed below) using thermal flux transducers (Concept Engineering, Old Saybrook, CT) according to standard procedures (11). Body surface area (BSA) was calculated as follows: area ($m^2$) = $weight^{0.425}$ (kg) · $height^{0.725}$ (cm) · 0.007184 (5). The following regional percentages were assigned based on Layton et al. (19): forehead 4%, dorsum of the head 3%, chest 8.75%, abdomen 17.5%, scapula 8.75%, forearm 12%, posterior upper arm 7%, anterior thigh 9.5%, posterior thigh 9.5%, anterior calf 6.5%, posterior calf 6.5%, and the foot 7%. A light mesh hood was used to hold the dorsal head transducer snuggly against the hair on the back of the head. Flux was defined as positive when heat traversed the skin towards the environment (i.e. heat loss) and values for each transducer ($W \cdot m^{-2}$) were converted into $W \cdot region^{-1}$ as follows:

$Flux_{region}$ (W) = transducer flux ($W \cdot m^{-2}$) · body surface area ($m^2$) · regional percentage

Oxygen consumption ($\dot{V}O_2$) was measured with an open-circuit from expired minute volume and inspired and mixed-expired gas concentrations sampled from a mixing box ($V_{max}$ 229 by Sensormedics). Because subjects were completely submersed in half of the trials, they breathed compressed air through baseline and during immersion/submersion in all trials. The SCUBA tanks were kept at room temperature at all times before and during testing and the assumption was made that the temperature of the inspired gases was room temperature (~22°C). To facilitate metabolic measurements, a standard SCUBA regulator (Blizzard, Sherwood, Lockport, NY) was modified and connected to corrugated tubing so that all expiratory gas could be collected by the metabolic system. Metabolic measurements of respiratory activity conducted in hyperbaric conditions but analyzed at normobaric conditions will over-estimate ventilatory parameters but will accurately measure metabolic variables (i.e., oxygen consumption) (27). All data were recorded at 30 –second intervals.

In subsequent analysis, oxygen consumption and respiratory exchange rate (RER) were used to calculate metabolic rate (M) in Watts as follows:

$M (W) = \dot{V}O2 (l/min) \cdot 69.7(4.686 + [(RER - 0.707) \cdot 1.232])$ (31)

Respiratory heat loss (RHL) was calculated in dependence of metabolism (6):

$$RHL\ (W) = 0.09 \cdot M$$

Total energy production for the immersion/submersion was calculated by converting metabolic rate (W) to kilojoules (kJ). Total energy loss was calculated as the sum of total body cutaneous heat flux and respiratory heat loss. The net energy balance was determined by integrating the difference between total energy loss and production over the first 45 minutes of immersion. This method correlates well with direct measurements of tissue heat (18,23).

## Immersion conditions

Subjects were immersed/submersed four times in 17°C water. For each condition subjects were lowered with an electronically isolated hoist into the water. Two of the conditions involved complete submersion and required the breathing of compressed air. Thus, for all trials (baseline and immersion/submersion) subjects wore a nose clip and breathed compressed air as previously described. One weight belt (15 kg) was worn around the waist and another one (12 kg) was placed over the thighs, not covering the heat flux probes. In the head submersion trials a diving facemask was worn. The mask covered the nose, upper cheeks, the eyes and approximately 50% of the forehead, a total surface of approximately 160 $cm^2$. The average body surface of the subjects was

2.07 ± 0.1$m^2$. The face is considered to be 4% of the total BSA (19); this would imply that the average face surface of the subjects was 800$cm^2$ and that the mask covered 20% of this area.

In the body insulated conditions, the instrumentation wires exited the suit via the right wrist cuff. During these trials, the right hand was held just above the water surface to prevent water from leaking through the wrist seal.

Body-exposed, head-out. The subjects wore only a bathing suit and were immersed to the neck with the head positioned above the water. A mesh hood kept the posterior head heat flux disk in place.

Body-exposed, head-in. The subjects wore only a bathing suit and were lowered until the entire head was completely submersed. A mesh hood kept the posterior head heat flux disk in place while a diving mask covered the eyes and nose. The straps of the mask did not cover the posterior head probe.

Body-insulated, head-out. Subjects wore a 1.5 mm thick vulcanized rubber dry suit over thermal underwear (full body suit), a fleece suit, two pairs of socks and a wool glove on the left (immersed) hand, as well as a rubber glove over the wool glove. The insulative value of this ensemble was 6.3 clo. Subjects were immersed to the neck with the head positioned out of the water. A mesh hood kept the posterior head heat flux disk in place.

Body-insulated, head-in. Subjects wore the same insulation ensemble described above. They were lowered until the entire head was completely submersed. A mesh hood kept the posterior head heat flux disk in place while a diving mask covered the eyes and nose. The straps of the mask did not cover the posterior head probe.

## Protocol

After subjects were prepared, ten minutes of baseline measurements were recorded. In the body-insulated conditions only the thermal underwear as well as one pair of socks was worn during baseline. Before the subjects were immersed the rest of the insulative clothing were donned. This was done to prevent the subjects from overheating before the start of immersion/submersion. Subjects were lowered by an electronically-isolated hoist into the water.

In order to achieve an overall balanced design, the first 4 subjects were randomly assigned a different first condition. The remainder of the experiments of all subjects was then randomly assigned to achieve a balanced design.

The subjects remained immersed until one of four removal criteria was met: 1) immersion time of 60 minutes; 2) voluntary request by a subject for removal; 3) $T_{es}$ reached 34°C; or 4) termination of immersion by investigator for safety reasons. Upon removal from the cold water, subjects had a choice to be

placed in a 40°C stirred water bath until $T_{es}$ was > 36°C and they felt comfortably warm. This choice was made in all the body-exposed conditions. Only one of the subjects requested a warm bath after a body-insulated, head-in experiment.

## Data analysis

For different reasons, three subjects wished to exit after 45 minutes of submersion. Therefore all the analysis was done for the first 45 minutes of exposure.

The following calculations were made for each condition: 1) decrease in $T_{es}$ from baseline to 45 min; 2) rate of core cooling (calculated by linear regression for $T_{es}$ data from 10 to 45 min of immersion); 3) area-weighted mean skin temperature; 4) cutaneous heat loss; 5) metabolic heat production (kJ) over the first 45 min of immersion, and 6) net energy balance in the different conditions. Group results were calculated for each condition and were compared using repeated measures analysis of variance. Results are reported as means ± SD. $P < 0.05$ identified statistically significant differences. The Holm-Sidak test was used for post hoc analysis of significant differences. The Chi squared test was used to compare the observed heat loss from various body regions to the expected values (based on the assumption that regional heat loss is proportional to that region's relative surface area).

## RESULTS

All subjects remained immersed/submersed for 60 minutes in the two head-out conditions. In the body-exposed, head-in condition, two subjects exited the water at 45 minutes, while one subject requested to be taken out of the water at 45 minutes in the body-insulated, head-in condition . Although none of these subjects reached the cut-off value of 34°C, they requested to be taken out because of significant discomfort. In one body-exposed, head-in trial, ECG data were unusable due to shivering artifact. The missing value was replaced using all available data on HR, considering both the differences between conditions as well as between subjects (13).

### Core temperature responses

The average baseline core temperature for the four immersion/submersion conditions was 37.16±0.4. Figure 1 illustrates the change in core temperature following immersion/submersion (immersion = Time 0).

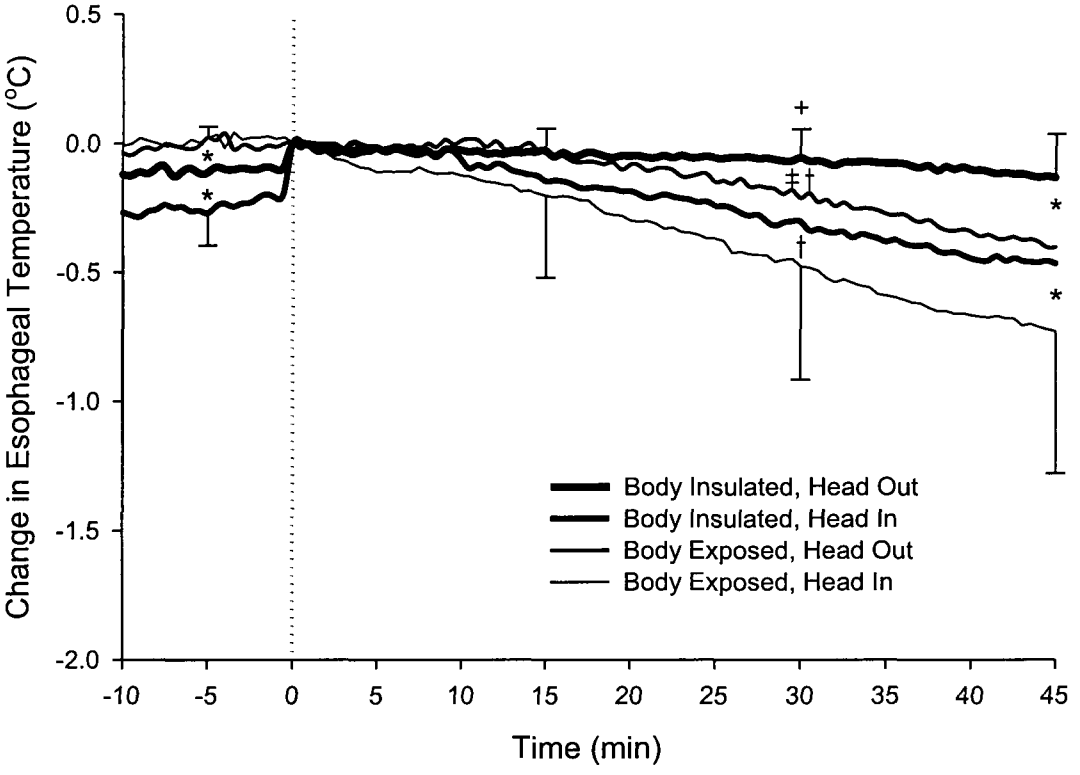


Figure 1. Change in esophageal temperature during ten minutes of baseline and 45 minutes of immersion/submersion (Start of immersion, Time = 0, $T_{es}$ = 0°C, n= 8). After the subjects were dressed for the body-insulated conditions core temperature rose slightly before immersion/submersion. + Body-insulated, head-out is higher than Body-exposed, head-in ($p < 0.001$). † Head-in conditions lower than head-out for the same body sub-condition ($p < 0.02$). ‡ Body-insulated, head-in and body-exposed conditions are lower than time 0 ($p < 0.05$). * Separates all conditions that are significantly different including time 0 ($p < 0.05$). Error bars represent SD.

Core temperature decreased significantly from baseline after 30 minutes in all conditions except the body-insulated, head-out condition, which never did drop below baseline. At this point of submersion/immersion, $T_{es}$ decreased more

in the head-in conditions when compared to the head-out conditions in both the body-exposed (0.47±0.4°C and 0.21±0.3°C respectively, $p < 0.02$) and body-insulated (0.30±0.2°C and 0.06±0.1°C respectively, $p = 0.02$) conditions. Alternatively, it took 45 minutes before body-exposure significantly decreased core temperature compared to body-insulation, within both the head-out (0.40±0.3°C and 0.13±0.2°C respectively, $p < 0.03$), and head-in (0.73±0.6°C and 47±0.3°C respectively, $p<0.05$) conditions. Throughout the 45 minutes of immersion/submersion there was no difference between $T_{es}$ in the body-insulated, head-in and body-exposed, head-out conditions. The relationship between the conditions are confirmed when data are presented as the rates of core cooling between 10 and 45 minutes of immersion/submersion (Table 1).

| | Metabolic Rate (W) | | Heart Rate (b/min) | | | |
|---|---|---|---|---|---|---|
| | Baseline | 45 minutes | Baseline | 45 minutes | Decrease in $T_{es}$ (°C) | Core Cooling Rate (°C/h) |
| Insulated Head-out | 137.5±31 | 139.2±32 | 79.2±9 | 69.5±7 | 0.13±0.2 * | 0.16±0.3 * |
| Insulated Head-in | 137.2±28 | 184.8±33 * | 85.9±7 | 73.1±6 * | 0.47±0.3 | 0.71±0.5 |
| Exposed Head-out | 140.1±41† | 325.4±97 | 82.2±9 | 79.1±7 | 0.40±0.4 * | 0.70±0.5 * |
| Exposed Head-in | 154.8±37† | 364.1±119 | 83.6±8 | 82.5±9 | 0.73±0.6 | 1.09±0.6 |

Table 1. Metabolic rate, heart rate and changes in esophageal temperature ($T_{es}$) before and during immersion in 17°C water. * Separates all conditions that are significantly different ($p \leq 0.05$). † Significant difference over time ($p \leq 0.05$)

## Mean skin temperature responses

During the baseline period, mean skin temperature ($T_{SKavg}$) was slightly, but significantly, higher during body-insulated conditions (31.1±0.9°C) than without insulation (29.4±1.0°C) ($p<0.03$). In the body-insulated sub-conditions, $T_{SKavg}$ decreased more with the head submersed (to 27.2±1.1°C) than in the head out (to 29.2±0.7°C) ($p=0.001$) after 45 minutes. Likewise, in the body-exposed conditions, $T_{SKavg}$ decreased more in the head-in condition (19.0±1.2°C) than in the head-out condition (20.5±1.3°C) ($p<0.01$).

## Metabolic responses

Baseline values for metabolic rate were similar for all conditions (Figure 2). Immediately after immersion in both the body-exposed conditions, metabolic rate increased above baseline values from 145 W to 250 W ($p < 0.001$). Metabolic rate increased over time to 345 W after 45 minutes of cold exposure. Total heat production was 80% more in the body-exposed conditions (808±244 kJ) than in the body-insulated (442±81 kJ) after 45 minutes of the experiment ($p< 0.001$, see Fig. 4). Throughout the experiment there were no differences between the head-out and head-in conditions in either of the body sub-conditions.

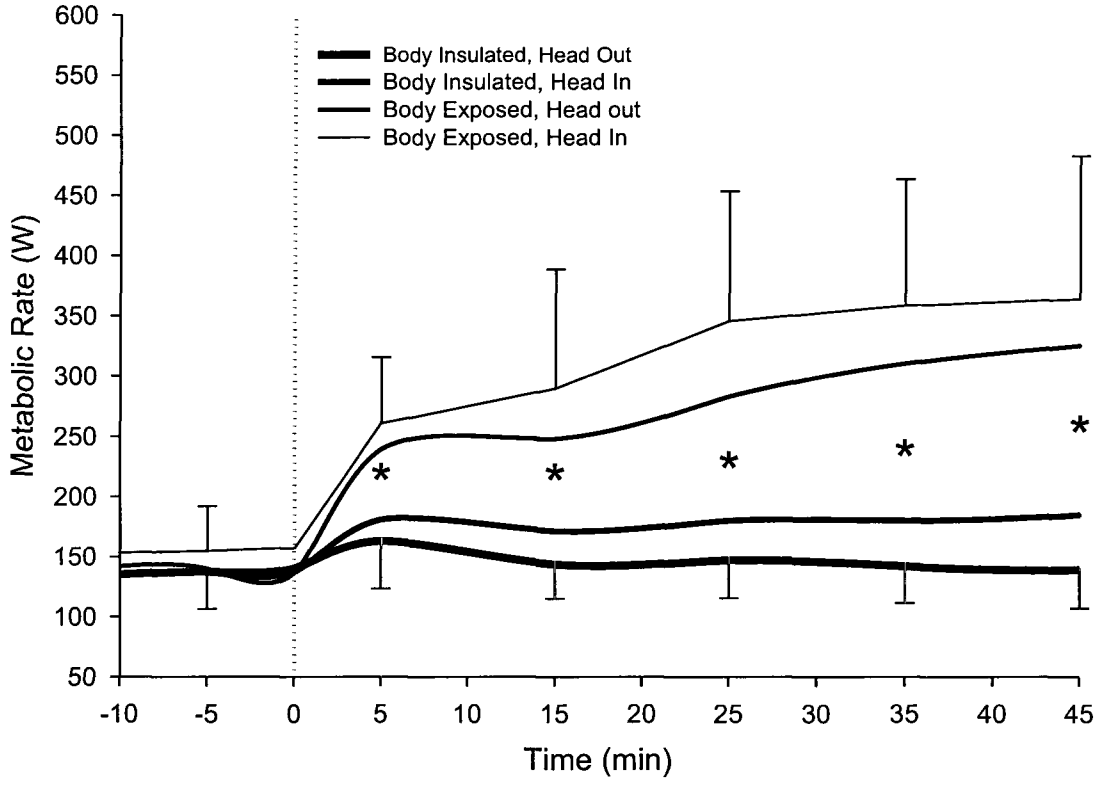


Figure 2. Metabolic rate for ten minutes of baseline and 45 minutes of immersion/submersion (Start of immersion, Time = 0, $T_{es}$ = 0°C, n= 8). * Separates all conditions that are significantly different ($p < 0.005$). Error bars represent SD.

## Cutaneous heat loss

The average rate of cutaneous heat loss during baseline was greater in the body-exposed (140.9±13 W) conditions than in the body-insulated (95.5±14 W) conditions (Figure 3). Heat loss increased markedly immediately upon immersion, with the effect in exposed areas gradually decreasing as skin cooled and the

temperature gradient between skin and water decreased. Head submersion resulted in a significantly higher rate of heat loss in both the body-insulated and body-exposed sub-conditions.

Except for the face, most of the head was covered with hair. The skin was not shaved in any area for comparison of heat loss from shaved and hair-covered areas of the immersed scalp. In this study heat loss was ~22% less from hair covered skin (251 $W \cdot m^{-1}$) than from the bare forehead (320 $W \cdot m^{-1}$). A light mesh hood was used to hold the heat flux transducer snuggly against the hair of the back of the head. This eliminated a layer of water between the hair and transducer, thus ensuring that heat loss from the skin, and through the hair, was channeled through the transducer. The mesh was light and provided negligible insulation when tested on the bare forehead (< 5% decrease in heat flux).

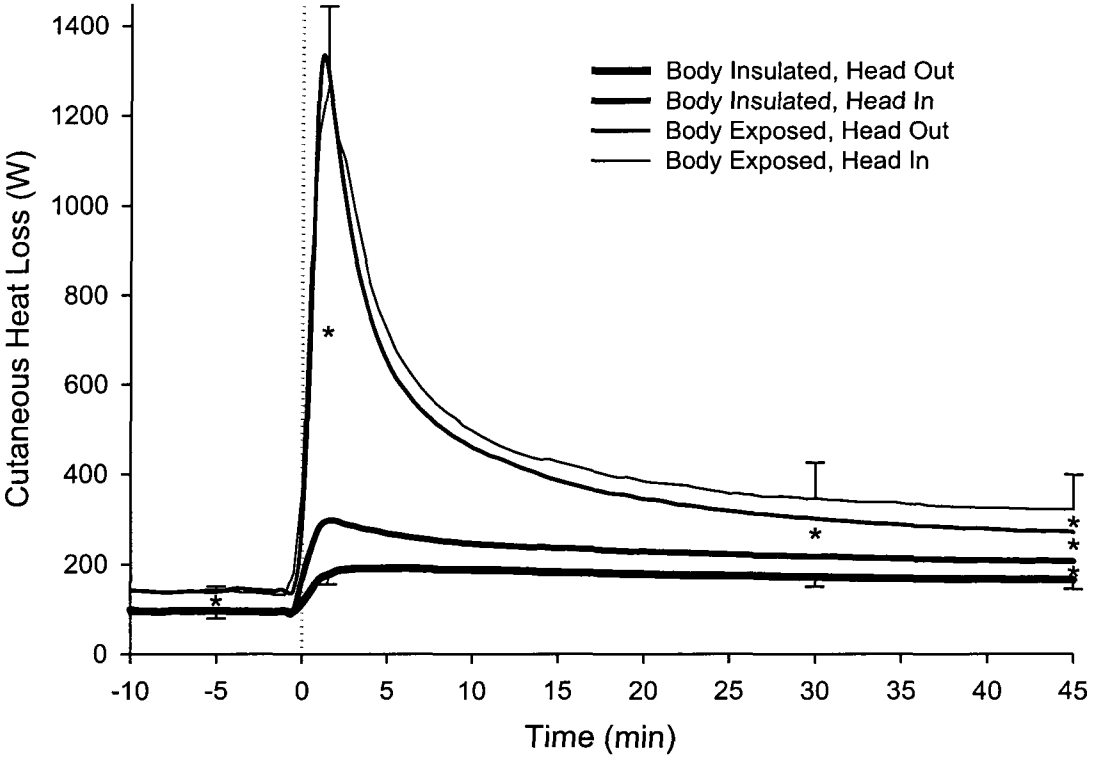


Figure 3. Average cutaneous heat loss values for ten minutes of baseline and 45 minutes of immersion/submersion (Start of immersion, Time = 0, $T_{es}$ = 0°C, n= 8). * Separates all conditions that are significantly different ($p < 0.001$). Error bars represent SD.

## Energy balance

Heat loss and energy production during the 45 minute period of cooling are presented in absolute terms in Figure 4; values are for total energy production, total body (including respiratory) heat loss, head heat loss as well as skin heat loss from the body excluding the head. Total body heat loss was more than twice the amount in the body-exposed (1239±187 kJ) than in the body-insulated (591±88 kJ)

conditions ($p<0.001$) with the greatest loss seen in the body-exposed, head-in condition (1303±223 kJ). In both the body-insulated and body-exposed sub-conditions, head submersion resulted in greater heat loss from the head ($p<0.001$).

During 10 minutes of baseline there was no difference in the mean heat loss (6.84±1.3 kJ) through the head between the four conditions. When the head was exposed to the cold water, head heat loss during the first 45 minutes of immersion was 137.17±15.3 kJ which was higher than the 32.12±5.12 kJ in the head-out conditions (<0.001).

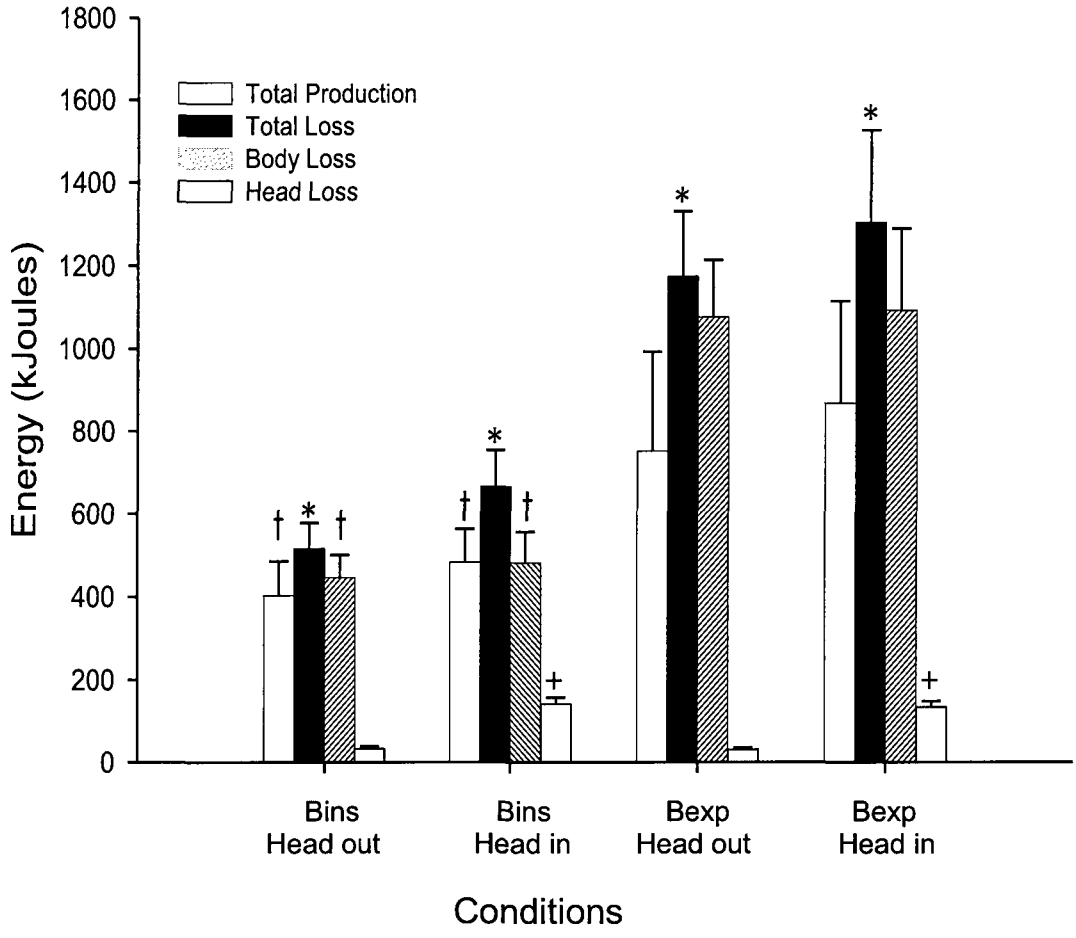


Figure 4. Energy production and loss during 45 minutes of immersion/submersion in 17°C water. Total Loss includes cutaneous and respiratory heat loss. Body Loss includes trunk, legs and arms. † Less than body-exposed conditions ($p < 0.001$). * Different from all other conditions ($p < 0.001$). + Greater than Head-out conditions ($p < 0.001$). Error bars represent SD.

Chi squared analysis indicated that there were no significant differences between observed and expected[9] regional heat losses when the body and head

[9] The expected values were based on regional percentages

were under similar thermal conditions (i.e., body-insulated, head-out and body-exposed, head-in). In the body-exposed, head-in condition, the head accounted for 7% of the total body surface area and 11% of the total skin heat loss (N.S.). Head heat loss was proportionally greater than its surface area in the body-insulated, head-in condition and proportionally lower in the body-exposed, head-out condition.

After 45 minutes the negative energy balance during immersion was significantly higher in the body-exposed conditions (-423.5 and -436.3 kJ with head-out and head-in respectively) than body-insulated conditions (-113.3 and -183.0 kJ with head-out and head-in respectively) ($p<0.001$).

## Heart rate responses

There were no inter-condition differences during baseline in heart rate (82.7±8 b/min). After a transient increase to 89±16 b/min during entry into the water, heart rate decreased gradually in all conditions. The average heart rate in the body exposed conditions between 15 and 45 minutes of immersion was higher (78±8 b/min) than in the body-insulated conditions (72±6 b/min) ($p=0.02$).

## DISCUSSION

This was the first study to evaluate the isolated contribution of whole head cooling to lowering of core temperature with shivering heat production intact. As expected, head submersion in 17°C water did not cause a disproportionately large increase in surface heat loss. Rather, the 7% increase in submersed surface area elicited a comparable 11% increase in heat loss with no increase in shivering heat production.

Compared to the results from the previous non-shivering study on whole head cooling which was also done in 17°C water (27), shivering attenuated the rate of core cooling in all conditions (ranging from 53% [body-exposed, head-in] to 83% [body-insulated, head-out]). However, several results were similar to the previous study (27). Head submersion increased the rate of core cooling both when the body was insulated and cold-exposed. Also, the core cooling rate was similar whether only the head or only the body was exposed to cold water, despite a large difference in total heat loss (665 vs 1174 kJ respectively).

Previous studies, with shivering intact, have reported that cooling only the dorsal head had little effect on core cooling whereas combined immersion of the dorsal head and body significantly increased core cooling (1,22). Compared to

body-only immersion, additional dorsal head immersion increased core cooling from 3.8 to 9.4°C·hr$^{-1}$ (250%) in 1-2°C water (1), and from 1.5 to 2.8°C·hr$^{-1}$ (87%) in 10°C water (22). When the study was repeated in 12°C water with shivering suppressed (8), dorsal head immersion increased core cooling rate by 39% (from 3.6°C·hr$^{-1}$ to 5.0°C·hr$^{-1}$). The previous whole head cooling study (27) reported an average of 41% increase in core cooling with 7% increase in exposed surface area. Hayward et al. (14) reported similar relative differences when subjects were physically active in 10°C water. They demonstrated that drownproofing (which intermittently submersed the whole head) increased core cooling by 36% to 4.6°C·hr$^{-1}$, compared to 3.4°C·hr$^{-1}$ while treading water with the head above water.

## Possible mechanisms for the results

The contribution of head submersion to core temperature cooling was of major interest in this study. As in previous studies (8,27), head cooling (in this case by total submersion) increased total heat loss in proportion to the increased surface area, yet core cooling increased by 343% in the body-insulated and 56% in the body-exposed conditions; the exaggerated effect of head submersion in the body-insulation condition is artifactually high because there was no significant core cooling in the body-insulated, head-out condition.

This increased core cooling rate likely results from the extra heat loss affecting a smaller thermal core due to peripheral vasoconstriction secondary to

intense cold stimulation of the body (31) as well as the dive response elicited by facial cooling (12). With facial submersion vasoconstriction would result from thermoregulatory control mechanisms and the human dive reflex, mediated by the trigeminal nerve as an oxygen sparing mechanism (3,16,17). This oxygen sparing mechanism occurs in thermoneutral water (i.e., 34°C) (3) and increases at cooler water temperatures (i.e., 20-25°C) (16,17). The dive reflex likely plays a significant role in the core cooling response even though the usual bradycardia (29) was not seen.

Since the face mask covered ~20% of the face and would attenuate the vasoconstriction response (12), the present results likely underestimate the total effects of whole head submersion.

Finally, it was interesting that core cooling was similar when either the head-only or body-only was cold-exposed, despite significantly greater heat loss by (509 kJ) during body-only immersion. Two opposing factors could explain these results. A reduced thermal core during head-submersion would enhance core cooling, while increased shivering heat production during body-immersion would tend to offset the core cooling effect. During body-immersion, shivering would not only increase heat production, but it could also increase the thermal core to fuel the muscles for shivering. A given heat loss will result in less core cooling when the thermal core increases in size (8,22,27). The sum of these factors

(shivering, surface heat loss and the size of the thermal core) will eventually determine the rate of core cooling. Future work clarifying the mechanisms of core cooling in these two conditions would aid the understanding of whole head cooling.

## Practical implications of results

This study has practical implications regarding the gradual onset of hypothermia in cold-water SCUBA divers and recreational swimmers. Scalp and facial cooling will tend to enhance core cooling even in cool water that only moderately increases the integrated thermal signal. The moderate thermal vasoconstriction may be synergistically augmented by the facially-stimulated dive response. Thus a moderate, but significant, increase in heat loss, without an increase in shivering heat production may result in significant core cooling. Cold-water SCUBA divers and recreational swimmers may be at risk if they experience hypothermia to the point of altered physical (10) and mental capacity (9,22).

According to Gooden et al. (12) the dive response is attenuated with a normal breathing pattern and when a face mask is worn with the head insulated. Thus, maintaining a slow deep breathing pattern (as is recommended for SCUBA diving) and insulating the head prophylactically should help prevent the development of hypothermia.

Since this study was dealing with head cooling, it would be of value to know how the temperature of the brain is affected by it. Our research is limited by the fact that we could not directly measure brain temperature. Based on the findings of Nelson (26) and Xu (32) that brain temperature mainly depends on heat transfer with arterial blood and less so on cooled blood from the face and scalp, it will be assumed brain temperature will drop due to the drop in core temperature as measured in the esophagus and not due to the conductive and/or convective heat loss through the skull and scalp. The assumption is that esophageal temperature represents the temperature of the heart (25) and thus arterial blood temperature to the brain and all other organs.

In conclusion, whole head submersion in 17°C water did not contribute relatively more than the rest of the body to total surface heat loss. Consequent to submersion and cooling of the scalp and face, thermal stimulation and the human dive response likely reduced the thermal core significantly. Thus a small increase in total heat loss, without the masking effect of increased shivering heat production, caused a relatively large cooling of the body core. Further work is warranted to quantify the effects of scalp and/or face immersion in colder water on regional blood flow and alterations in the mass of the thermal core.

## ACKNOWLEDGEMENTS

This study was supported by the Natural Science and Engineering Research Council of Canada. The authors wish to thank Doug Evans of One Stop Diving in Winnipeg, for providing the SCUBA regulator used in this study, and Jeremy Stewart of the Freshwater Institute, Winnipeg, for providing compressed air. We also wish to thank Kate McGarry and Dominique Gagnon for technical assistance during many of the trials.

REFERENCES

1. Alexander L. The Treatment of Shock From Prolonged Exposure to Cold, Especially in Water. Washington, DC: Office of the Publication Board, Department of Commerce. (Combined Intelligence Objectives Sub-committee), 1946, p. 1-228.

2. Brozek J. Densiometric analysis of body composition: revision of some quantitative assumption. Ann. N.Y. Acad. Sci. 110: 113-140, 1963.

3. Campbell L. Simultaneous calf and forearm blood flow during immersion in man. Austr. J. Exp. Biol. Med. Sci. 47: 747 - 754, 1969.

4. Conn AW, Miyasaka K, Katayama M, Fujita M, Orima H, Barker G and Bohn D. A canine study of cold water drowning in fresh versus salt water. Crit. Care Med. 23: 2029-2037, 1995.

5. DuBois D and Dubois E. A formula to estimate the approximate surface area if height and weight are known. The Archives of Internal Medicine 17: 863-871, 1916.

6. Fanger PO. Thermal Comfort: Analysis and Applications in Environmental Engineering. New York: McGraw-Hill, 1972.

7. Froese G and Burton AC. Heat losses from the human head. J. App. Physiol. 10: 235-241, 1957.

8. Giesbrecht G, Lockhart T, Bristow G and Steinman A. Thermal effects of dorsal head immersion in cold water on nonshivering humans. J. Appl. Physiol. 99: 1958-1964, 2005.

9. Giesbrecht GG, Arnett JL, Vela E and Bristow GK. Effect of task complexity on mental performance during immersion hypothermia. Aviat. Space Environ. Med. 64: 206-211, 1993.

10. Giesbrecht GG and Bristow GK. Decrement in manual arm performance during whole body cooling. Aviat. Space Environ. Med. 63: 1077-1081, 1992.

11. Giesbrecht GG, Goheen MSL, Johnston CE, Kenny GP, Bristow GK and Hayward JS. Inhibition of shivering increases core temperature afterdrop and attenuates rewarming in hypothermic humans. J. Appl. Physiol. 83: 1630-1634, 1997.

12. Gooden B, Lehman R and Pym J. Role if the face in the cardiovascular responses to total immersion. Austr. J. Exp. Biol. Med. Sci. 48: 687-690, 1970.

13. Hassard T. Understanding Biostatistics. St Louis: Mosby - Year Book Inc., 1991.

14. Hayward JS, Eckerson JD and Collis ML. Effect of behavioral variables on cooling rate of man in cold water. J. Appl. Physiol. 38: 1073-1077, 1975.

15. Hayward JS, Eckerson JD and Kemna D. Thermal and cardiovascular changes during three methods of resuscitation from mild hypothermia. J. resuscitation 11: 21-33, 1984.

16. Heistad DD, Abboud F and Eckstein J. Vasoconstrictor response to simulated diving in man. J. Appl. Physiol. 25: 542-549, 1968.

17. Hurwitz B and Furedy J. The human dive reflex: An experimental, topographical and physiological analysis. Physiol. Behav. 36: 287-294, 1986.

18. Kurz A, Sessler DI, Christensen R and Dechert M. Heat balance and distribution during the core-temperature plateau in anesthetized humans. Anesthesiology 83: 491-499, 1995.

19. Layton R, Mints WJ, Annis J, Rack M and Webb P. Calorimetry with heat flux transducers: comparison with a suit calorimeter. J. Appl. Physiol. 54: 1361-1367, 1983.

20. LeBlanc J. Subcutaneous fat and skin temperature. Candian Journal of Biochemistry and Physiology 32: 354-358, 1954.

21. LeBlanc J. Mechanisms of adaptation to cold. Int. J. Sports Med. 13: S169, 1992.

22. Lockhart T, Jamieson C, Steinman A and Giesbrecht G. Life jacket design affects dorsal head and chest exposure, body core cooling and mental performance in 10°C water. Aviat. Space Environ. Med. 76: 954-962, 2005.

23. Matsukawa T, Sessler DI, Sessler AM, Schroeder M, Ozaki M, Kurz A and Cheng C. Heat flow and distribution during induction of general anesthesia. Anesthesiology 82: 674-681, 1995.

24. Mekjavic IB and Morrison JB. A model of shivering thermogenesis based on the neurophysiology of thermoreception. IEEE Transactions on Biomedical Engineers BME-32: 407-417, 1985.

25. Moran DS and Mendal L. Core temperature measurement; methods and current insights. Sports Med. 32: 879-885, 2002.

26. Nelson DA and Nunneley SA. Brain temperature and limits on transcranial cooling in humans: quantitative modeling results. Eur. J. Appl. Physiol. 78: 353-359, 1998.

27. Pretorius T, Bristow G, Steinman A and Giesbrecht G. Thermal effects of whole head submersion in cold water on non-shivering humans. J. Appl. Physiol. 101: 669-675, 2006.

28. Rasch W, Samson P, Cote J and Cabanac M. Heat loss from the human head during exercise. J. Appl. Phsyiol. 71: 590-595, 1991.

29. Schagatay E and Holm B. Effects of water and ambient air temperatures on human diving bradycardia. Eur. J. Appl. Physiol. 73: 1-6, 1996.

30. Vanggaard L. Thermoregulation in the extreme cold environment. AGARD Conference Proceedings 540. The Support of Air Operations under Extreme Hot and Cold Weather Conditions, Victoria, Canada. North Atlantic Treaty Organization, 1993, p. 19.11-19.14.

31. Vanggaard L, Eyolfson D, Xu X, Weseen G and Giesbrecht GG. Immersion of distal arms and legs in warm water (AVA Rewarming) effectively rewarms hypothermic humans. Aviat. Space Environ. Med. 70: 1081-1088, 1999.

32. Xu X, Tikuisis P and Giesbrecht G. A mathematical model for human brain cooling during cold-water near-drowning. J. Appl. Physiol. 86: 265-272, 1999.

# CHAPTER 3

# CORE COOLING AND THERMAL RESPONSES DURING WHOLE HEAD, FACE AND DORSUM IMMERSION IN 17°C WATER

Running title: Thermal Effects of Whole Head Cooling

KEYWORDS: Hypothermia, Cold-water Immersion, Perfused body mass, Symptomless hypothermia, Effective perfused mass, Vasoconstriction

Chapter 3 appears here as it is sent in for publication to Applied Physiology, Nutrition and Metabolism by Thea Pretorius, Dominique Gagnon and Gordon G Giesbrecht

MY CONTRIBUTION TO THE PUBLICATION. This work was done in totality in the Laboratory of Exercise and Environmental Medicine as a follow-up study for the first manuscript of this thesis. In the present study we looked at the effect of face, dorsal head whole head cooling on core temperature and metabolism when the shivering response was intact and the body in thermal neutral air. With the help of the head of this laboratory and my supervisor, Dr Gordon Giesbrecht, and Dominique Gagnon, a fellow student, we planned the set up for the study. Dr Giesbrecht and Dominique were mainly involved in the building of the head submersion tank. Assisted by Dr Giesbrecht and my fellow students Dominique and Gerren McDonald, we conducted all the experiments at which time data were collected regarding temperature (skin and core) as well as metabolism, heart rate and data from the laser doppler. It took team effort to prepare the laboratory and to assure that the water temperature was kept constant and all instruments were running smoothly during the hour and a half of recording of data.

After collecting all data I was responsible for preparing and analyzing of the final figures. I have written the manuscript in consultation with Dr Giesbrecht and we agreed on a journal to send it to. At this time the manuscript was sent to all authors for their approval. We are still waiting for approval of the manuscript.

ABSTRACT: This study isolated the effects of dorsal, facial and whole head immersion in 17°C water on peripheral vasoconstriction and the rate of body core cooling. Seven subjects were studied in thermoneutral air (~28°C). They lay prone or supine on a bed with their heads inserted through the side of an adjustable immersion tank. Following 10 minutes of baseline measurements the water level was raised for 60 min such that the water immersed the dorsum (D), face (F) or whole head (W). During the first 30 minutes core (esophageal) cooling rate increased from D (0.29±0.2°C·h$^{-1}$) to F (0.47±0.1°C·h$^{-1}$) to W (0.69±0.2°C·h$^{-1}$) ($p<0.001$); cooling rates were similar during the final 30 minutes (mean; 0.16±0.1°C·h$^{-1}$). During the first 30 minutes fingertip blood flow (laser Doppler flux as % of baseline) decreased faster in W (114±52%·h$^{-1}$) than either F (51±47%·h$^{-1}$) or D (41±55%·h$^{-1}$) ($p<0.03$); rates of flow decrease were similar during minutes 30-60 (mean; 22.5±19%·h$^{-1}$). Total head heat loss over 60 minutes was significantly different between W (120.5±13 kJ), F (86.8±17 kJ) and D (46.0±11 kJ) ($p<0.001$). Rate of core cooling, relative to head heat loss, was similar in all conditions (mean; 0.0037±0.001°C/kJ). Although the whole head elicited a higher rate of vasoconstriction, the face did not elicit more vasoconstriction than the dorsum. Rather the progressive increase in core cooling from D to F to W correlates simply with increased heat loss.

# INTRODUCTION

Many marine activities involve, or pose the risk of, immersion in cold water with the consequent development of hypothermia. Given the fact that the head is often partially or fully immersed during voluntary activities (e.g., SCUBA diving and cold-water adventure swim racing) or emergency situations (e.g., ship wreck survival and cold-water drowning), an understanding of the specific effects of head cooling may help prevent hypothermia.

The rate of core cooling in cold water is affected by physiological factors (e.g., shivering, nutrition, exhaustion, pharmacology etc.) and biophysical factors (e.g., anthropometrics, insulation, body position and movement, and sea state and temperature) (9). One other important factor is the amount of body surface area exposed.

Until recently, most cold-immersion studies involved exposure of the body only, without addressing the effects of the head. Several studies from our laboratory were the first to isolate the effects of dorsal and whole head cold-water immersion. Consistently, if the body was already cold-exposed, additional immersion of the dorsum (8,16) or whole head (18,19) increased heat loss by 5% and 11% respectively, while causing a disproportional increase in core cooling rate of 40-87%. If the body was insulated, isolated whole head immersion still decreased core temperature (by 0.7-1.6°C·h$^{-1}$), however, dorsum immersion did not.

In fact, isolated whole head immersion resulted in the same core cooling rate as isolated body immersion (18,19).

Thus, two questions remain unanswered: 1) How can head cooling be so effective in decreasing core temperature? and 2) Why is whole head cooling so much more effective than dorsum cooling in decreasing core temperature, especially when the body is insulated and dorsum cooling has no effect? First, we have postulated that head cooling increases peripheral vasoconstriction, reducing the "effective perfused mass"; the mass of tissue perfused, and thermally affected by, blood flow (24). In this scenario the relative effect of extra heat loss from the head might be amplified.

Second, the exaggerated core cooling effect of the whole head (which includes the face) compared to that of the dorsum, seems to be greater than could be expected simply based on increased body surface area (BSA) for heat loss (i.e., the face is 4% BSA and the dorsum is 3% BSA). This phenomenon could be partially explained by a greater stimulation of peripheral vasoconstriction – and thus an even smaller effective perfused mass – when the face is also cooled. Facial immersion would increase vasoconstriction through normal thermoregulatory control mechanisms and the human dive reflex, which is mediated by the trigeminal nerve and elicits vasoconstriction, bradycardia and breath holding (4,12,13).

Although a few studies (14,21) have examined the immediate effect of facial cooling on peripheral vasoconstriction, none have investigated these effects over a longer period of time. In our previous work we have not measured peripheral blood flow nor have we compared facial cooling to dorsal cooling.

The purpose of this study was to isolate the effects of dorsal, facial and whole head cooling in 17°C water on peripheral blood flow and rate of core cooling. Based on a proposed additional effect of the facial-stimulated dive reflex, we hypothesized that facial cooling, in isolation and as part of whole head cooling, would increase peripheral vasoconstriction and core cooling rate compared to dorsal head cooling.

## METHODS

### Subjects

The experimental protocol was approved by the University of Manitoba Education / Nursing Research Ethics Board. Eight male subjects, each of whom provided written, informed consent, were studied. None of the subjects were engaged in any activity that would expose them to cold water on a regular basis immediately before or at the time of the study. Subjects completed a medical (PAR-Q) questionnaire to screen for cardio-respiratory disease. They were also

questioned about any other conditions that could be exacerbated by exposure to cold water. Subjects were studied on three separate occasions, at least 48 hours apart, and at the same time each day to control for circadian effects. The subjects were instructed to abstain from alcohol, tobacco and strenuous exercise for 12 hours prior to each trial and to consume only a light meal before they arrived at the laboratory. A familiarization session was held on a separate day before the first trial. During this session, the subject's head was partially and totally immersed in both the supine and prone positions. The purpose was to reduce potential stress for subsequent trials and evaluate the capability of the subject to participate in the study; subjects were comfortable after about one minute in each position.

## Anthropometric Data

Height, weight and skinfold thickness at four sites (biceps, triceps, subscapular and iliac) were measured; percent body fat was calculated based on body density, estimated from the sum of four skinfolds (3). Body surface area was calculated from height and weight measurements (6).

## Instrumentation

For each trial, subjects wore light clothing (t-shirt and shorts) and were instrumented while resting in an environmental chamber at ~28°C and 20% relative humidity.

Esophageal temperature ($T_{es}$) was measured using a thermocouple (Mon-a-therm - General Purpose, Mallinckrodt Medical Inc., St Louis, MO) in the esophagus at the level of the cardiac atria. This site has previously been shown to provide the closest non-invasive correlation to intra-cardiac temperature (10). Single-channel electrocardiogram (Lead II), heart rate (Hewlett Packard Co. McMinnville, OR) and blood pressure (AMG Medical Inc., Montreal, QC) were also monitored.

Thermal flux transducers (Concept Engineering, Old Saybrook, CT) measured heat flux (HFT) and skin temperature ($T_{sk}$) from the anterior head (forehead), cheek, dorsal head (neck) and superior surface of the torso (e.g. chest in the supine position and back in the prone position). (15)

Changes in fingertip skin blood flow were estimated with laser-Doppler perfusion measurements (PeriFlux 2B, Perimed, Stockholm, Sweden). A small angled probe was taped on the middle fingertip of the right hand. Perfusion was

defined as a product of the number of blood cells moving in the measured field and the mean velocity of these cells.

Skin temperatures (Tsk) were measured with thermocouples (Mon-a-therm - General Purpose, Mallinckrodt Medical Inc., St Louis, MO) on the ventral base and dorsal tip of the right index finger. Each trial was started only once fingertip vasodilation was indicated by a positive fingertip-to-finger base temperature gradient (see Protocol) (5,22). Fingertip skin temperature was also used as an adjunct to laser-Doppler flowmetry.

For the calculation of heat loss from the head the following regional percentages were assigned to the sites based on Layton et al. (15): forehead 2%, cheek 2% and dorsum of the head 3%. Heat flux was defined as positive when heat traversed the skin towards the environment (i.e. heat loss). To convert relative heat flux values ($W/m^2$) to an absolute rate (W) for each site ($HF_{site}$) the following equation was used:

$HF_{site}$ (W) = $HFT_{site}$ ($W/m^2$) x $BSA(m^2)$ x regional percentage of each site.

HF from the head ($HF_{head}$) = $HF_{dorsal\ head}$ + $HF_{forehead}$ + $HF_{cheek}$

To calculate absolute heat loss (kJ) over any given period, the average rate (W) was applied for the length of that period.

Anterior head heat loss was determined from the forehead and cheek because they represent most of the anterior head and these areas respond similar to the other non-acral area; the chin (2). Because the subject wore a nose clip, it was not possible to measure from the acral nose area.

Estimation of scalp heat loss was technically problematic because hair prevents direct contact of the heat flux transducers with the skin. In previous studies (16,18,19) dorsal head transducers were held in place over the hair with a light mesh hood. Although this was accepted as a viable solution under the circumstances, this arrangement potentially underestimates flux as some heat may flow from under the hair past the transducer, and the mesh - even though light - might restrict some flux. Similar practical limitations have been acknowledged in other studies quantifying head heat flow in which only the forehead and posterior neck were instrumented (15).

The current study configuration introduced another limitation - a practical one. Instrumentation of the head could only be achieved after it was placed inside the immersion tank since wires from the head would have compromised the neck seal if they were applied before head insertion. In the restricted tank area, it was impractical to apply the mesh hood technique while the head was inside the tank.

Since four of the seven subjects had medium length hair, it was not possible to attach the dorsal transducer to the scalp. This transducer could however, be applied to the posterior neck just below the hairline. Data from a previous study (19) indicated that, when the entire head was similarly exposed (i.e., cold immersion), HF from the hair-covered dorsum was consistently lower than on the bare forehead by 18%. Thus, for the three subjects with close shaved heads, neck HF values represented dorsal head heat loss. For the subjects with hair, neck HF values were corrected by an 18% reduction.

Oxygen consumption ($\dot{V}O_2$) was measured with an open-circuit from expired minute volume and inspired and mixed-expired gas concentrations sampled from a mixing box ($V_{max}$ 229 by Sensormedics). Subjects were required to breathe under water for two of the three conditions (facial and whole head immersion). Thus they breathed through a mouth piece attached to a snorkel in all trials to standardize protocols for all the conditions. The snorkel was extended by 10 cm with flexible tubing which connected to a Hans Rudolph 2-way valve (Hans Rudolph, inc. Kansas City, MO). The expiratory side of the valve was connected via corrugated tubing to the metabolic system.

Oxygen consumption and respiratory exchange rate (RER) were used to calculate metabolic rate (M) in Watts as follows:

$$M\ (W) = V_{O_2}\ (l/min) \cdot 69.7(4.686 + [(RER - 0.707) \cdot 1.232]) \quad (24)$$

All data were recorded at 30 –second intervals.

## Head Immersion Tank

A head immersion tank was designed such that only the subject's head was exposed to the water. The tank was constructed from a flexible scrim-reinforced vinyl cylinder (42 cm diameter, 88 cm tall) with a closed bottom end. A dry suit vulcanized rubber neck cuff was glued to a 24 cm diameter hole, which was cut into the side of the tank (the center was 27 cm from the top). This allowed head insertion through a waterproof neck seal. An internal frame (50 cm high) kept the top portion of the tank rigid (including the neck seal section). The top of this frame was securely suspended from an external support. The bottom of the tank sat on a platform which was suspended from an electrically-isolated hoist. Because the tank had flexible sides, elevating the platform – and thus the bottom of the tank - raised the water level within the tank relative to the head, thus allowing part, or all, of the head to be immersed. During immersion, the subjects were vigilantly observed at all times for any signs of distress as indicated by audible sounds, blood pressure, heart rate, respiration and hand signals or other body movements. If necessary, the platform could be quickly lowered. Water

temperature was controlled at 17±0.3°C with a pump which exchanged water between the immersion tank and a temperature controlled water bath (1250 L).

## Immersion Conditions

Each subject participated in three experimental trials involving either partial (dorsal and facial immersion) or total head immersion. The order of conditions was selected to achieve a balanced design.

*Dorsum Immersion* (D): Subjects lay supine on a mattress with the head through the neck cuff. The water level was raised until the entire ears were immersed (thus immersing most of the surface area innervated by the cervical nerves).

*Facial immersion* (F): Subjects lay prone on a mattress with the head through the neck cuff. The water level was raised to a point just anterior to the ear (thus immersing most of the surface area innervated by the trigeminal nerves)

*Whole Head immersion* (W): Subjects lay prone on a mattress with the head through the neck cuff. The water level was raised to completely immerse the whole head.

## Protocol

All trials were performed in a climatic chamber which was controlled at just above thermoneutral conditions (~28°C and at 20% relative humidity) (7) For each experimental trial the subject sat for a conditioning period of 20-40 min in the climatic chamber to ensure vasodilation as indicated by a skin temperature greater at the fingertip than the finger base (5,22). The subject lay supine on the mattress for instrumentation. He then either remained supine or assumed a prone position, depending on the condition, and inserted the head through the neck cuff. In order to support the head within the tank, the forehead or dorsum then rested on two styrofoam supports placed 6 cm apart, each with a surface area of 7.5 $cm^2$ (i.e., 1.5 cm laterally and 5 cm longitudinally). The two surfaces were slopped inward at 45°. Thus, <2% of the face or dorsum contacted the supports at any time. The subject then breathed through the snorkel for 10 minutes of baseline recording. At this point, the water was raised to the desired level for 60 minutes.

All subjects remained immersed for 60 minutes, the water was lowered and ten minutes of recovery data were collected.

## Data analysis

The following calculations were made for each condition: 1) rate of core cooling for first and second 30-minute periods of immersion calculated by linear regression from $T_{es}$ data; 2) rate of decreased peripheral perfusion for first and second 30-minute periods of immersion calculated by linear regression from data from laser-Doppler and fingertip temperature and 3) drop in core temperature relative to total amount of head heat loss was calculated after 30 and 60 minutes of immersion (°C/kJ).

Results for each condition were compared using repeated measures analysis of variance (ANOVA). As well, an ANOVA was also used to determine differences between mean values during baseline, immersion and recovery in the same condition for heart rate and mean arterial blood pressure. Tukey's multiple comparison test was used for post hoc analysis of significant differences. Results were reported as means ± SD. A p-value $< 0.05$ identified statistically significant differences.

## RESULTS

The subjects were 30.4±3 years old, 181.6±6 cm tall and weighed 90.9±16 kg. They also had 20.2±5% body fat, and a body surface area of 2.1±0.2 $m^2$. On average the area of the face was 0.08±0.002 $m^2$ and that of the dorsum 0.6±0.002 $m^2$. One subject dropped out after the first experiment because of discomfort during the whole head immersion trial. Therefore the analysis was only done on the data for the remaining seven subjects.

### Esophageal temperature

During the first 30 min, core (esophageal) cooling rate increased from D (0.29±0.2°C·$h^{-1}$) to F (0.47±0.1°C·$h^{-1}$) to W (0.69±0.2°C·$h^{-1}$) ($p<0.001$); cooling rates were similar during the final 30 minutes (mean; 0.16±0.1°C·$h^{-1}$). After 60 minutes the decrease in $T_{es}$ was greater in W (0.45±0.2) than F (0.25±0.1°C) and D (0.19±0.1°C), ($p\leq0.002$). During recovery $T_{es}$ did not drop any further in any of the three conditions.

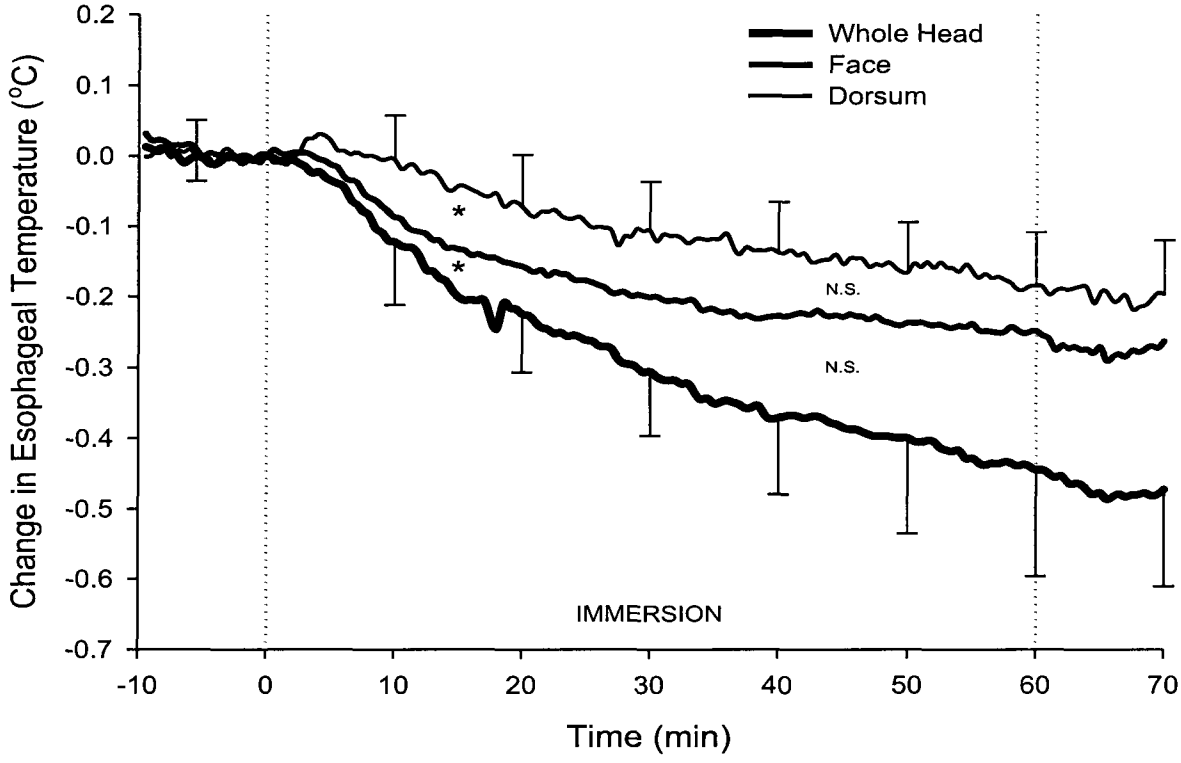


*Figure 1. Change in esophageal temperature (plotted every 30 seconds) during ten minutes of baseline, 60 minutes of immersion and 10 minutes of recovery (at start of immersion; time = 0 min, $T_{es}$ = 0 °C, n=7). *Separates conditions that have different rates of cooling from minute 0 to 30 ($p < 0.02$). N.S. Rates of cooling are similar for minutes 30 to 60. Error bars represent SD. Dotted lines indicate start and finish of immersion. Dashed line separates first and second 30 minute periods of analysis.*

## Fingertip blood flow and temperature

During the first 30 min fingertip blood flow (laser Doppler flux as % baseline) decreased faster in W (114±52%·h$^{-1}$) than either F (51±47%·h$^{-1}$) or D

(41±55%·$h^{-1}$) ($p<0.03$); rates of flow decrease were similar during min 30-60 (mean; 22.5±19%·$h^{-1}$).

Rate of change of fingertip temperature generally correlated to changes over time of Doppler flow and decreased faster in W (7.04±3.1°C·$h^{-1}$) than either F (2.84±2.2°C·$h^{-1}$) or D (1.87±2.7°C·$h^{-1}$) ($p<0.03$); rates fingertip temperature change were similar during min 30-60 (mean; 4.04±2.7°C·$h^{-1}$).

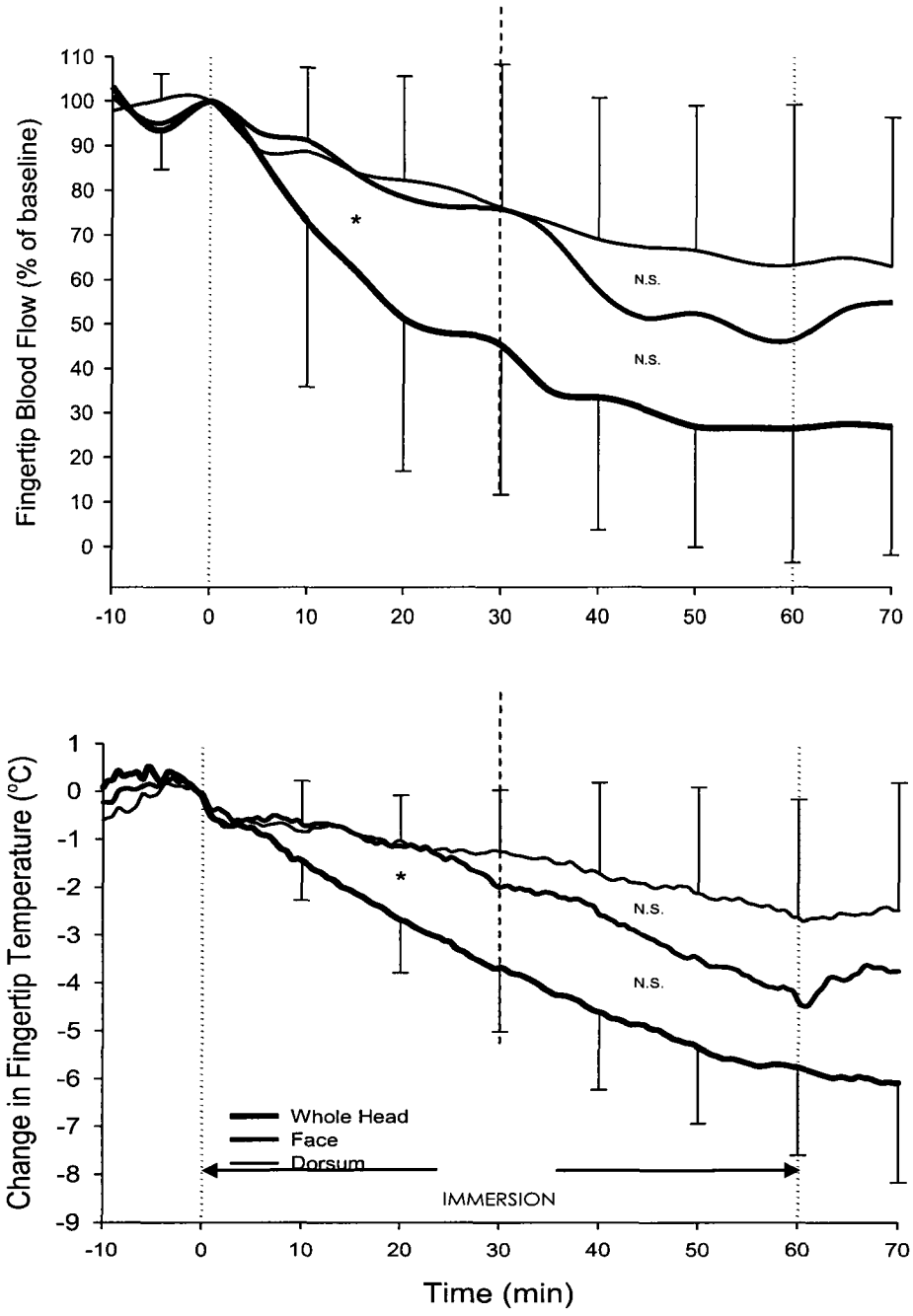


Figure 2. Fingertip bloodflow and temperature

Figure 2. Top. Laser Doppler fingertip skin blood flow during ten minutes of baseline, 60 minutes of immersion and 10 minutes of recovery (at start of immersion; time = 0 min). Data are averaged every 5 minutes and plotted as % of baseline. Bottom. Change in fingertip skin temperature. Data are plotted every 30 seconds.

*Separates conditions that have different rates of decrease from minute 0 to 30 ($p < 0.04$). N.S. Rates of cooling are similar for minutes 30 to 60. Error bars represent SD. Dotted lines indicate start and finish of immersion. Dashed line separates first and second 30 minute period of analysis.

## Heat production and loss

Metabolic rate was similar in all conditions and did not change significantly from baseline to immersion to recovery with mean values of 134.4±19 W, 127.3±17 W and 125.2±18 W respectively.

There were no differences between conditions in the amount of heat loss from the head during baseline or recovery. Figure 3 indicates that head heat loss significantly increased from dorsal to facial to whole head immersion.

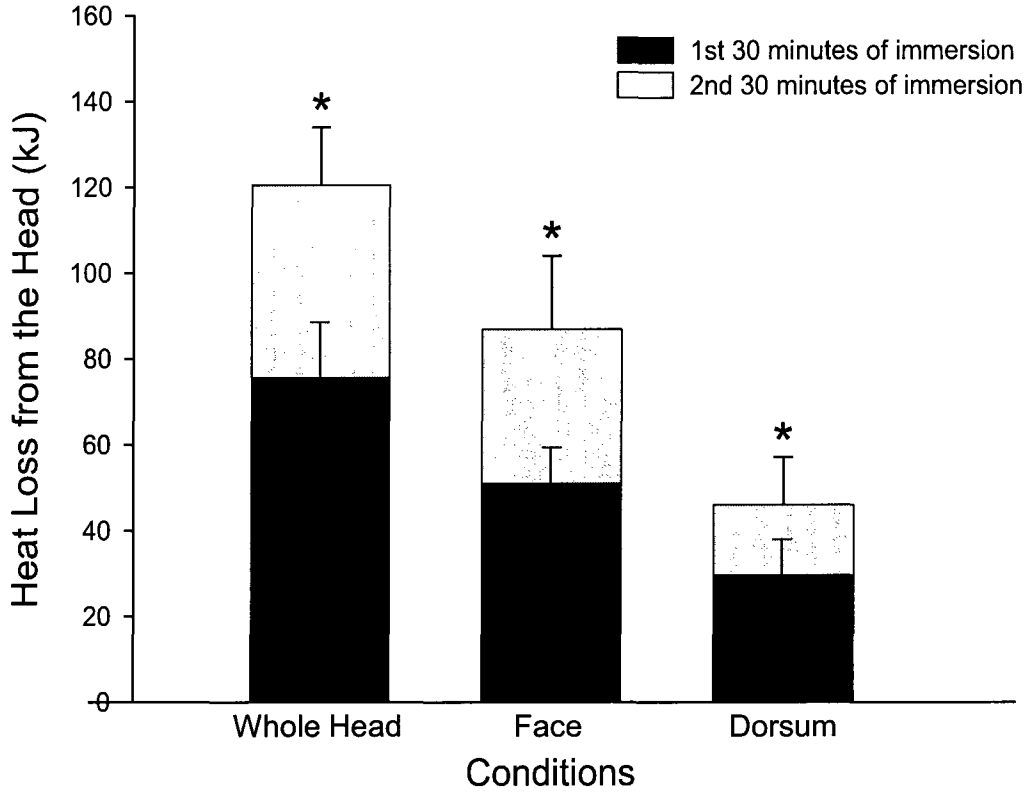


Figure 3. Head heat loss during the first and second 30-minute periods of immersion.*Different from all other conditions ($p<0.001$) during both time periods. Error bars represent SD.

## Change in $T_{es}$ relative to heat loss

In all three conditions the drop in Tes was similar relative to the amount of heat lost through the head after 30 minutes (0.0040±001°C/kJ, 0.0039±002 and 0.0038±002 respectively for W, F and D, $p>0.05$) and 60 minutes

(0.0037±001°C/kJ, 0.0030±001 and 0.0044±002 respectively for W, FI and D, $p>0.05$) of immersion.

### Heart rate and mean arterial blood pressure

Heart rate (HR) was similar in all conditions and did not change significantly from baseline to immersion to recovery with mean values of 72.1±9 $b \cdot min^{-1}$, 64.7±9 $b \cdot min^{-1}$ and 68.0±8 $b \cdot min^{-1}$ respectively.

In W and D, MAP increased during immersion [from 107.0±17 mmHg to 111.6±16 mmHg and 94.8±10 to 99.0±10 mmHg respectively, ($p<0.04$)]. These values remained elevated during recovery. MAP did not change throughout the F condition.

## DISCUSSION

This study was unique in examining and comparing the thermoregulatory and core cooling responses to long-term cold water (17°C) immersion of the face, whole head and dorsum. The hypothesis that core cooling would increase from dorsal to facial to whole head immersion was supported. However, the hypothesis that facial immersion would elicit greater vasoconstriction than dorsal immersion was not supported; although vasoconstriction was greater during whole head

immersion than both facial and dorsal immersion. Rather, core cooling correlated more to absolute heat loss during each condition. Subjects did not increase metabolic heat production despite core temperature decreases ranging from 0.2°C (D) to 0.45°C (W).

The core cooling effect of 60 minutes of isolated whole and dorsal head immersion was consistent with previous studies. Whole head immersion in 17°C water for 30 and 45 minutes decreased core temperature by 0.3 and 0.34°C respectively (18,19). Dorsal head immersion in 10°C water for 35 minutes, and 12°C water for 30 minutes, did not significantly affect core temperature (8,16). In the present study, although there was a significant decrease in core temperature after 60 minutes, the decrease was small (0.2°C).

We are unaware of other studies of long-term facial immersion. When our data were averaged over 5-minute periods, initial effects of face immersion seemed small. Fingertip blood flow decreased by only 7% after 5 minutes; eventually flow decreased by 54% after 60 minutes. When blood flow data were evaluated at 30-s intervals however, there was an initial transient decrease for all three conditions (ranging from 4-16% over 30-90 s) with subsequent return to near-baseline values within 5 min of immersion. Thereafter, blood flow progressively decreased for the remaining 55 min of immersion.

These transient results are consistent with other eupneic face immersions in ~10°C water where fingertip blood flow decreased by ~37% within 10 s; an effect that disappeared after 20 s (1). Apneic face immersion in 6°C water decreased fingertip blood flow by 77% after 30 s; this effect was likely larger because active breathing attenuates dive responses (14).

## Possible mechanisms for the results

As expected whole head immersion caused more vasoconstriction and core cooling than dorsal and facial immersion. Facial immersion did not however, augment vasoconstriction compared to dorsal immersion. First, trigeminal nerve stimulation was not completely limited to face immersion. The ear was immersed during dorsal immersion in order to expose a similar surface area during face (4% BSA) and dorsum (3% BSA) conditions. Thus some trigeminal nerve stimulation – from the anterior half of the external acoustic meatus and the external surface of the tympanic membrane – during dorsal immersion may have attenuated some of the expected differences between conditions. Second, prone and supine positions were used in this study. The prone position (in F and W) mechanically compresses the thorax and thus attenuates cardiac filling. This position also increases resting sympathetic nervous activity (20). However, other conditions that attenuate cardiac filling (e.g., lower body negative pressure and post-exercise hypotension), do not affect the sympathetic-mediated vasoconstriction response to face

immersion (14). Thus, body position was unlikely to have affected our results. Third, the initial dive responses were transient. Although the oxygen sparing mechanisms would only be teleologically advantageous for the duration of a breath hold, it is not confirmed if the dive response *per se* is active from 5 to 60 minutes of immersion in humans. Our data suggest this is not the case. Rather, continued decrease in fingertip blood flow and skin temperature is consistent with thermoregulatory control as core temperature continues to decrease.

At first, the hypothesis that increased vasoconstriction – a response that decreases heat loss - would increase core cooling, seems contradictory. It is important to note that this principle of core temperature protection relates primarily to the effect of exposure of the vasoconstricted areas. Our hypothesis however, relates to the effect of heat loss from other areas of the body. Specifically, a given amount of heat loss (i.e., from the head) would be expected to cause a greater temperature reduction on a smaller mass. We have previously referred to the "effective perfused mass" as the mass of tissue that is thermally affected by blood flow; vasoconstriction would decrease this mass (24). Thus, in response to a given heat loss, greater vasoconstriction would be expected to result in a lower temperature measured within the reduced mass.

Even though there was no difference in peripheral blood flow – nor presumably in effected perfused mass - between face and dorsum conditions, core

cooling was greater with face immersion. Differences in core cooling correlated simply to differences in amount of head heat loss. Thus, the 33% increase in exposed surface area during face immersion (e.g., 4 vs 3% BSA), explains at least some of the core cooling increase compared to dorsum immersion.

The amount of core cooling seems greater than would be expected from the observed heat loss, although these results are consistent with previous work (16,18,19). A full understanding of the mechanisms for this core cooling might require the use of similar immersion protocols while measuring regional heat contents and whole limb blood flow.

Finally, there was no increase in metabolic heat production despite decreases in core temperature. Certainly, the thermoregulatory thresholds for shivering in supine subjects (e.g., a core temperature decrease of 0.37°C) (17) was attained in this study. As well, a shivering response was expected based on a predictive equation using skin and core temperatures (23). First, predictive equations generally assume a relatively homogeneous skin temperature across the body, therefore this equation may not be valid for the present conditions where only a small area was cooled. For example in a previous study (19), two conditions resulting in similar decreases in core temperature of 0.4°C, evoked different metabolic results. When the body was immersed (with most of the skin homogeneously cooled), metabolic heat production doubled. However, when only

the head was immersed (as in the present study) there was no significant metabolic increase. Second, the lack of metabolic response when core cooling is very gradual is consistent with symptomless hypothermia experienced by some SCUBA divers (11).

## Practical implications of the study

The fact that a relatively small amount of heat loss from the small head surface can significantly decrease core temperature has important implications for many marine activities. Victims awaiting rescue at sea are often in a recumbent position with the head dorsum in the water. Our results confirm the importance of maximizing head insulation in sea survival ensembles.

Effective head thermal protection is also important for SCUBA divers. Since the onset of hypothermia can be symptomless – as confirmed by the lack of thermogenic response to core cooling in this study – prevention of core cooling is even more important. Diving hoods should have the maximum practical insulation and full facemask configurations might be considered for longer dives in cold water.

In conclusion, the isolated effect of head immersion, even with a mild cold stress of immersion in 17°C water, can significantly decrease core temperature. Although the face can exert a greater core cooling effect than the head dorsum

during the first 30 minutes of immersion, the mechanisms for this increase are not fully understood. Further studies measuring regional body heat contents and whole limb blood flow may help elucidate the mechanisms for our results.

## ACKNOWLEDGEMENTS

This study was funded in part by the Natural Science and Engineering Research Council (Canada) and Manitoba Health Research Council.

## REFERENCES

1. Andersson J, Schagatay E, Gislén A , and Holm B. Cardiovascular responses to cold-water immersions of the forearm and face, and their relationship to apnoea. *Eur. J. Appl. Physiol.* 83: 566-572, 2000.

2. Brajkovic D and Ducharme M. Facial cold-induced vasodilation and skin temperature during exposure to sold wind. *Eur. J. Appl. Physiol.* 96: 711-721, 2006.

3. Brozek J. Densiometric analysis of body composition: revision of some quantitative assumption. *Ann. N.Y. Acad. Sci.* 110: 113-140, 1963.

4. Campbell L. Simultaneous calf and forearm bloodflow during immersion in man. *Austr. J. Exp. Biol. Med. Sci.* 47: 747 - 754, 1969.

5. Clark S, Hollis S, Campbell F, Moore T, Jayson M , and Herrick A. The distal-dorsal difference as a possible predictor of secondary Raynauds Phenomenon. *J. of Rheumatol.* 26: 1125-1128, 1999.

6. DuBois D and Dubois E. A formula to estimate the approximate surface area if height and weight are known. *The Archives of Internal Medicine* 17: 863-871, 1916.

7. Fanger PO. *Thermal Comfort: Analysis and Applications in Environmental Engineering.* New York: McGraw-Hill, 1972.

8. Giesbrecht G, Lockhart T, Bristow G , and Steinman A. Thermal effects of dorsal head immersion in cold water on nonshivering humans. *J. Appl. Physiol.* 99: 1958-1964, 2005.

9. Giesbrecht G and Steinman AM. Immersion in cold water. In: *Wilderness Medicine* (5th ed.), edited by Auerbach P. Philadelphia, PA: Mosby, 2007, p. 160-188.

10. Hayward JS, Eckerson JD , and Kemna D. Thermal and cardiovascular changes during three methods of resuscitation from mild hypothermia. *j. resuscitation* 11: 21-33, 1984.

11. Hayward MG and Keatinge WR. Progressive symptomless hypothermia in water: possible cause of diving accidents. *Brit Med J*: 1182, 1979.

12. Heistad DD, Abboud F , and Eckstein J. Vasoconstrictor response to simulated diving in man. *J. Appl. Physiol.* 25: 542-549, 1968.

13. Hurwitz B and Furedy J. The human dive reflex: An experimental, topographical and physiological analysis. *Physiol. Behav.* 36: 287-294, 1986.

14. Journeay WS, Reardon FD , and Kenny GP. Cardiovascular responses to apneic facial immersion during altered cardiac filling. *J. of Appl. Physiol.* 94: 2249-2254, 2003.

15. Layton R, Mints WJ, Annis J, Rack M , and Webb P. Calorimetry with heat flux transducers: comparison with a suit calorimeter. *J. Appl. Physiol.* 54: 1361-1367, 1983.

16. Lockhart T, Jamieson C, Steinman A , and Giesbrecht G. Life jacket design affects dorsal head and chest exposure, body core cooling and mental performance in 10°C water. *Aviat. Space Environ. Med.* 76: 954-962, 2005.

17. Nakajima Y, Takamata A, Ito T, Sessler D, Kitamura Y, Shimosato G, Taniquchi S, Matsuyama H, Tanaka Y , and Mizobe T. upright posture reduces thermogenisis and augments core hypothermia. *Anesth. Analg.* 94: 1646-1651, 2002.

18. Pretorius T, Bristow G, Steinman A , and Giesbrecht G. Thermal effects of whole head submersion in cold water on non-shivering humans. *J. Appl. Physiol.* 101: 669-675, 2006.

19. Pretorius T, Cahill F, Kocay S , and Giesbrecht GG. Shivering heat production and core cooling during head-in and head-out immersion in 17°C water. *Aviat. Space Environ. Med.* 79: 495-499, 2008.

20. Pump B. Effects of supine, prone, and lateral positions on cardiovascular and renal variables in humans. *Am. J. Physiol. Regulatory Integrative Comp Physiol.* 283: R174-R180, 2002.

21. Schagatay E. *The Human Diving Response: Effects of Temperature and Training.* (Ph.D. Thesis). Lund: University of Lund, 1996.

22. Schuhfried O, Vacariu, G. Thermographic Parmeters in the Diagnosis of Secondary Raynaud's Phenomenon. *Arch. Phys. Med. Rehabil.* 81: 495-499, 2000.

23. Tikuisis P and Giesbrecht G. Prediction of shivering heat production from core and mean skin temperatures. *Eur. J. Appl. Physiol.* 79: 221-229, 1999.

24. Vanggaard L, Eyolfson D, Xu X, Weseen G , and Giesbrecht GG. Immersion of distal arms and legs in warm water (AVA Rewarming) effectively rewarms hypothermic humans. *Aviat. Space Environ. Med.* 70: 1081-1088, 1999.

# CHAPTER 4

## SHIVERING HEAT PRODUCTION AND BODY FAT PROTECT THE CORE FROM COOLING DURING BODY IMMERSION, BUT NOT DURING HEAD SUBMERSION.

## A STRUCTURAL EQUATION MODEL

Running title: Thermal Effects of Whole Head Cooling

KEYWORDS: Structural Equation Model, Core cooling, Surface heat loss, Cold sink, Metabolic heat production, Body fat

Chapter 4 appears here as it is sent in for publication to the Journal of Applied Physiology by Thea Pretorius, Lisa Lix and Gordon Giesbrecht

MY CONTRIBUTION TO THE PUBLICATION. The model in this manuscript originated from a trial and error model that was used as partial fulfillment for the reading course offered by Dr Lisa Lix during the fall of 2006. As a project I (the only student in the class) had to make a SEM model. I did not have to use my own data, but I had the data of the first whole head study and was convinced that despite the small sample size, this analysis technique will help us in our investigation of whole head cooling.

Although the initial model was only done on one of the two whole head studies, and some of the assumptions regarding sample size were somewhat violated, the potential of such a model became clear, and Dr Lix accepted it as a project for the completion of the course. In this model already, previous research was supported while the role of body fat became clear as an explanation of our results.

Supported by the enthusiasm of Dr Lix, I became more convinced that this is a model that should be incorporated in my PhD research. Dr Lix agreed to serve on my committee and the model was accepted as part of my PhD program.

After the whole head cooling study with spontaneous shivering, I made a second model, this time with shivering as one of the independent variables. I

submitted an abstract and presented it at an annual scientific conference of the Canadian Society of Exercise Physiology in 2007.

Over time many possibilities were explored to improve the model, and all attempts to put the data of the two studies together were unsuccessful. Using both studies would have the advantage of a bigger sample size as well as showing the effect of shivering over non-shivering. It was not until the past summer, that the idea came up to only use two of the four conditions of each study. This worked very well, and produced the final model in this manuscript. Dr Lix gave her expert advice on the two models and helped with the final preparation of this manuscript. We are still waiting for approval of the manuscript.

ABSTRACT: This study explored the factors which protect the body core from cooling when the body, but not the head, is exposed to cold water. Previous studies showed that core cooling rates are similar when only the head (with the body insulated from the cold) or only the body is cooled, despite twice as much heat loss from the exposed body. Structural Equation Modeling (SEM) was used to test the effects of surface heat loss (HL) from the body and the head as well as the interactive effects of percentage body fat (BF) and shivering heat production (HP) on core temperature during cooling in 17°C water. Data from two previous studies (14 male subjects) were used. In each experiment the body was exposed to 17°C water for 30 minutes. In 16 of these experiments the whole head was also exposed to the cold water. Core temperature drop (Drop), HP and HL from both the body and head (kJ) during this time was measured. Exposure of both the body and head increased Drop, while only the body elicited shivering to counteract this effect. BF attenuates both the shivering response as well as Drop. The bigger of these two effects is the attenuation of Drop. From our previous results it is postulated that this protection occurs mainly during body cooling where the fat on the body acts as a reservoir for cold through vasoconstriction. This explains why head cooling, despite an increase in HL of only 11%, increases core cooling rate by 39%.

## INTRODUCTION

Speculations on the effect of head heat loss on body core temperature, varying from being significant (8) to non-significant (41), inspired a series of controlled studies in our laboratory. Our initial results of dorsal head cooling studies (9,28) confirmed results from Alexander[10] (1); that dorsal head cooling increases body core cooling only when the body is also exposed to the cold water, but not when the body is insulated from the cold. One of the studies (28) measured the amount of surface heat loss (HL) and confirmed that HL from the dorsal head (~5% of total HL) is approximately proportional to the 3% body surface area (BSA) it represents (25), but disproportional to the increase in core cooling rates (250 - 39%) in these studies. Results of two studies on whole head cooling in 17°C water (30,31), showed a significantly greater increase in core cooling rate (~38%) when the whole head (7% of the BSA (25)) was exposed to the cold water. In both studies heat loss from the whole head was ~11% of total HL. This occurred whether the body was exposed to the cool water or not and whether the shivering mechanism was suppressed (30) or intact (31). What was

[10] These studies on prisoners of war in Dachau during World War II were grossly unethical, and the results are often considered invalid and unusable because of the emaciated condition of the prisoners as well as questions regarding the protocol and accuracy of the results.

particularly striking about both of these whole head cooling studies was the fact that core cooling rates were the same in the conditions where only the head or only the body was exposed to cold water, with a huge difference between the amount of HL between the two conditions. The results of these studies lead us to postulate that in the conditions where vasoconstriction is induced [i.e. in the body exposed conditions due to a cold stimulus (27) and the whole head submersion conditions due to the diving reflex (18)], core cooling rate was increased due to the effect of cooling a smaller effective perfused body mass. This speculation was not confirmed in a study (32) where the face, dorsum or whole head were cooled in 17°C water, each time with the body exposed to thermal neutral air. Although peripheral vasoconstriction increased as more of the head surface was exposed to the cold water, the core cooling rate relative to heat loss was similar in all conditions.

The finding that the increased core cooling rate due to the additional heat loss from the head is not explained by a reduced perfused mass, shifted our focus on the fact that the core cooling rate during both whole head cooling studies remained the same between conditions where only the head (with body insulated from cold water) or only the body was exposed to cold water (30,31). This phenomenon occurred despite the fact that the amount of HL from the exposed body was twice the amount of HL from the surface of the insulated body and the

exposed head. The initial question -'Why is rate of core cooling increased by exposure of the head?' now changed to: 'What protects the core from cooling when the body is exposed, but not when the head is exposed to cold water?'

While core cooling rate seems to be a function of surface heat loss from the head (32), previous studies on body-only cooling revealed that the core cooling rate is offset by a number of physiological and biophysical factors. These factors include shivering heat production (14), body composition (13,26,38), nutritional state (21,42), exhaustion (21,33,35), the effects of drugs and alcohol (22), age and gender, movement of the body (17) and water (34), body position (16), and different levels of immersion (28,30,40).

In the whole head studies done in this laboratory (30,31), the repeated measures design controlled for between subject differences, but not necessarily for between condition differences. Some factors [e.g. nutritional state, exhaustion, drugs (other than meperidine to suppress shivering), alcohol, body and water movement] were not measured but kept the same in each experiment, and the assumption was made that these effects did not differ between conditions.

Factors like body composition, age and shivering could have a more pronounced effect in the experiments where the body was subject to greater cold stress. Of all the body composition factors (height, weight, %body fat and muscle

mass) body fat is known to have the greatest effect in attenuating core cooling (13) through insulation (26).

The purpose of this study is to estimate the separate effects of surface heat loss from the body and the head on core temperature drop with the interactive effects of age, body composition (% body fat, height and weight) and shivering. This multivariable research question requires a statistical technique to not only estimate the effect of all variables on the outcome variable (core temperature), but also estimate the effect of the independent variables on each other (e.g. the effect of heat loss from the body on shivering). To accommodate these needs, data analysis was conducted using structural equation modeling (SEM) (24).

## METHODS

Data from the two previous whole head cooling studies (30,31) were used. In one of the studies (30) the shivering mechanism was suppressed with the use of buspirone and meperidine while spontaneous shivering occurred in the other study (31). Only the data obtained from the conditions where the body was exposed to the cool water (17°C) were used for the model. This was done in order to show the effect of shivering which differed significantly between the two

studies in these two conditions. In these experiments the head was either fully submersed or out of the water.

## Data

The following data were used from the previous studies: 1) Age; 2) Height 3) Weight; 4) % body fat; 5) Total heat loss (kJ) of head and body during 6) Total amount of metabolic heat production (kJ) over 30 minutes of immersion; 7) Drop in core temperature (°C) after 30 minutes of immersion.

## Data analysis.

Structural equation modeling is a statistical technique for testing and estimating hypothesized causal relationships among variables. A path model was specified to test the associations among the observed variables in this study. LISREL 8.80 software (23) was used to conduct the analyses.

In a path model, observed variables can either be endogenous or exogenous. Variables are defined as endogenous if the effect(s) of one or more variables on them are estimated and exogenous if no explicit causal effects are estimated by the model. Unanalyzed associations amongst exogenous variables could reduce some of the unexplained variation of a model (24).

All variables investigated in this study were measured on continuous scales. They included shivering heat production, percentage body fat, height, weight, age, heat loss from the body and heat loss from the head. The outcome was drop in core temperature. Factors which did not have any significant effect on any of the other variables, and neither improved model fit, were height, weight and age. They were not included in the final models.

The ability of SEM to test for effects of different variables, not only on the outcome variable, but also on each other, allowed us to do this analysis. Although, as with all multivariable techniques, SEM requires larger sample sizes (to reduce sampling error) than generally are used in our field of research. Kline (24) suggests at least ten observations for each parameter. Smaller sample sizes can lead to parameter estimates and fit indices with poor precision (4) and can result in Type II errors (29). To address this problem in our models, the bootstrap technique[11] was used to generate standard errors and 95% confidence intervals (CI) for regression coefficients in situations in which the standard assumptions are questionable e.g. small sample sizes. Three thousand samples were used to generate the CI and standard errors from the original observations. This is a well accepted number of repetitions to estimate standard error (5,19).

[11] The practice of estimating properties of a variable by measuring those properties in sub sets of the data set, which is obtained by random sampling from the original dataset.

In order to avoid problems associated with ill scaling (i.e., a ratio of greater than 10 of the largest to smallest variance for model variables) some of the variables had to be re-scaled, a process which does not affect the relationship among them. Univariate measures of skewness and kurtosis for all variables were close to zero indicating no serious departures from a normal distribution.

Three different models were considered. Model 1 was a path model with five observed variables; Total HL (kJ) from the body (Body HL), and the head (Head HL), percentage body fat (BF), shivering heat production (HP) and core temperature drop (Drop). Both HL and HP were calculated for the first 30 minutes of immersion/submersion. Drop was calculated as the difference in core temperature from baseline to the end of 30 minutes of immersion/submersion. All paths (causal effects) for this base model were based on the results of previous studies. It was hypothesized that only HL from the exposed body would increase heat production and not HL from the whole head (30-32). Body fat was expected to decrease both HP (12) and Drop (13,26), while HP was expected to delay core cooling (6,7). Both Body HL and Head HL were expected to increase Drop (30,31). Paths for all of these effects were included in all three models.

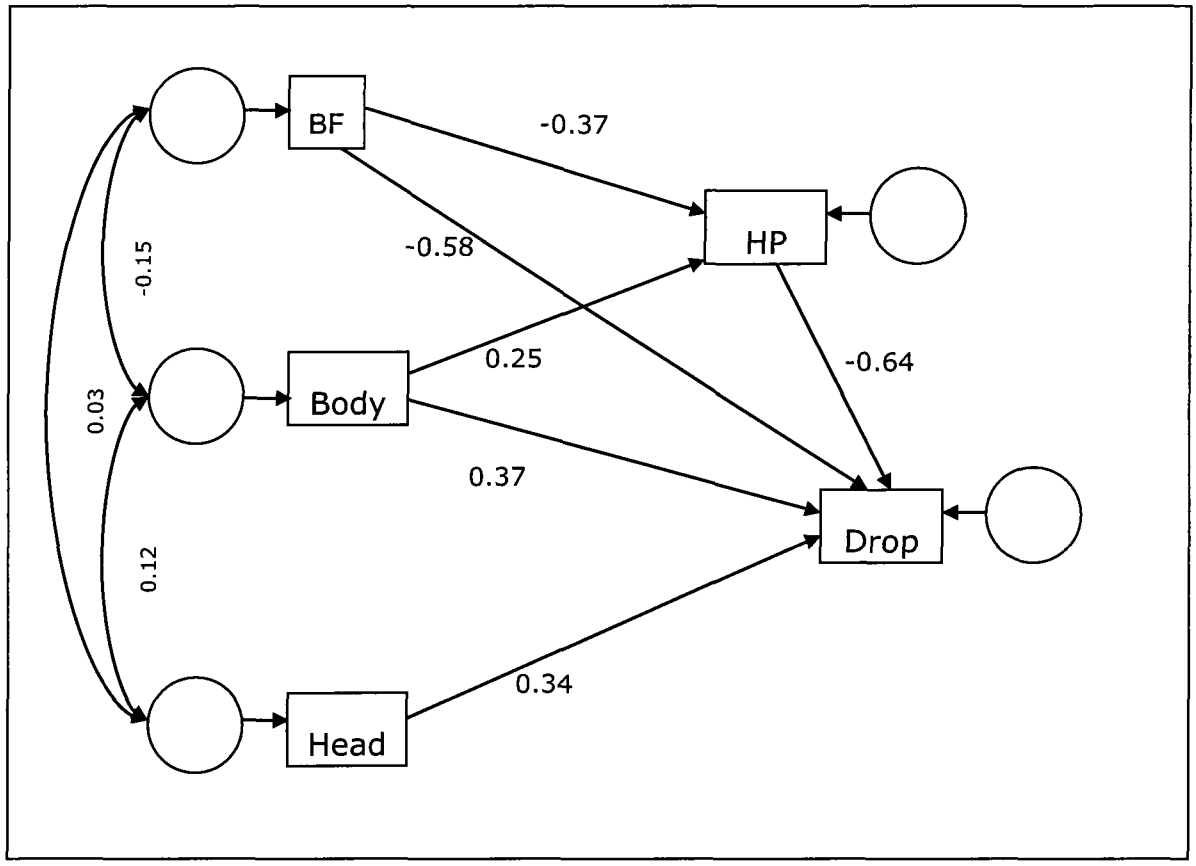


Figure 1. Model 1: Observed variables are represented as rectangles while error terms are represented by circles. A line with a single arrowhead represents a hypothetical direct causal effect of one variable on another and is also known as a path. Curved lines with two arrow heads represent unanalyzed associations. Three exogenous variables, percentage body fat (BF), body exposed to the cold water (Body) and the head in condition (Head) load on to two endogenous variables; shivering heat production (HP) the amount of core temperature drop during 30 minutes of exposure to 17°C water (Drop). Standardized estimates are given for each path and unanalyzed correlations.

Model 2 was used to test the hypothesis that subcutaneous fat reduced Body HL. The path model was the same as that of Model 1, except for inclusion of an additional path to test the effect of BF on Body HL (see Figure 2).

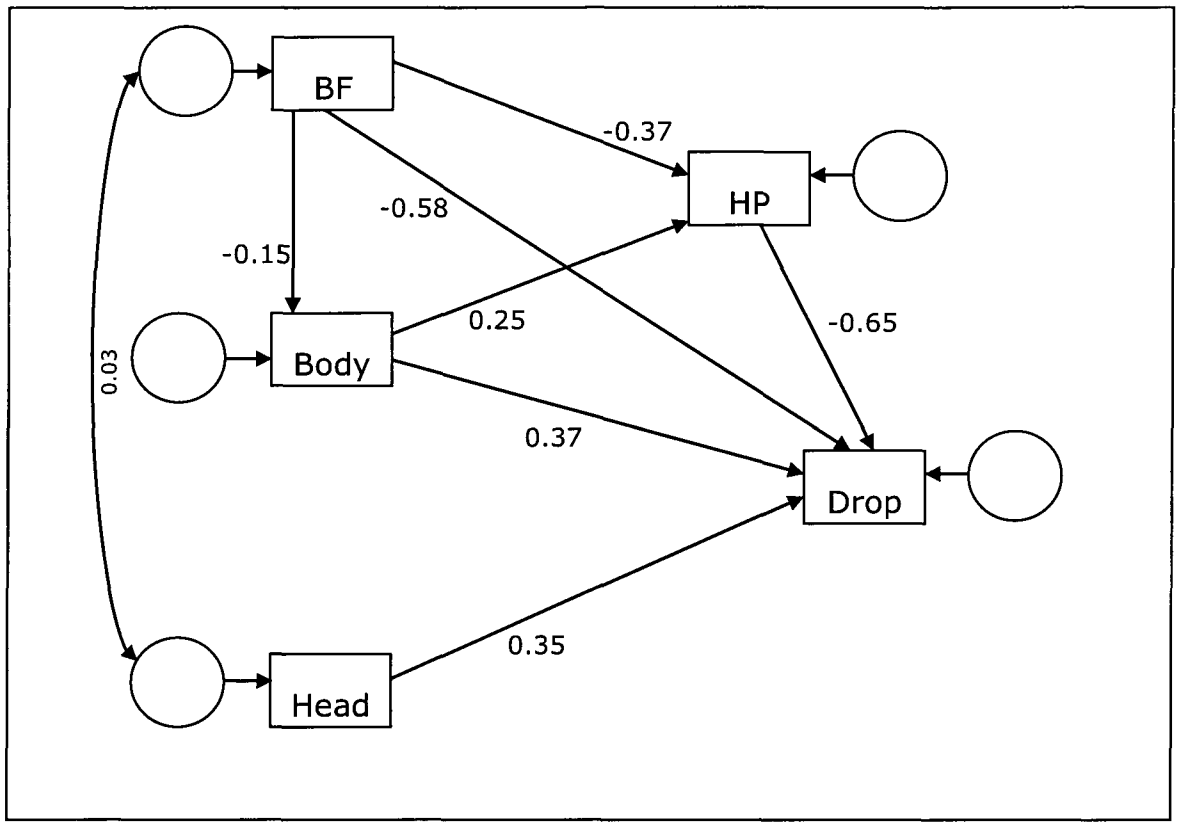


Figure 2. Model 2: Observed variables are represented as rectangles while error terms are represented by circles. A line with a single arrowhead represents a hypothetical direct causal effect of one variable on another and is also known as a path. Curved lines with two arrow heads represent unanalyzed associations. Two exogenous variables, percentage body fat (BF), and head heat loss (Head) load on to core temperature drop (Drop) during 30 minutes of exposure to 17°C water (Drop). BF also has an effect on body heat loss (Body) and shivering heat production (HP). Body heat loss has and effect on HP. The model has 2 degrees of freedom. An unanalyzed association exists between BF and Head. Standard estimates are given for each path and the unanalyzed association.

Model 3 (see Figure 3) was used to investigate the effect of BF on Head HL. Using the Model 2 as the base model, the path from BF to Head HL was specified.

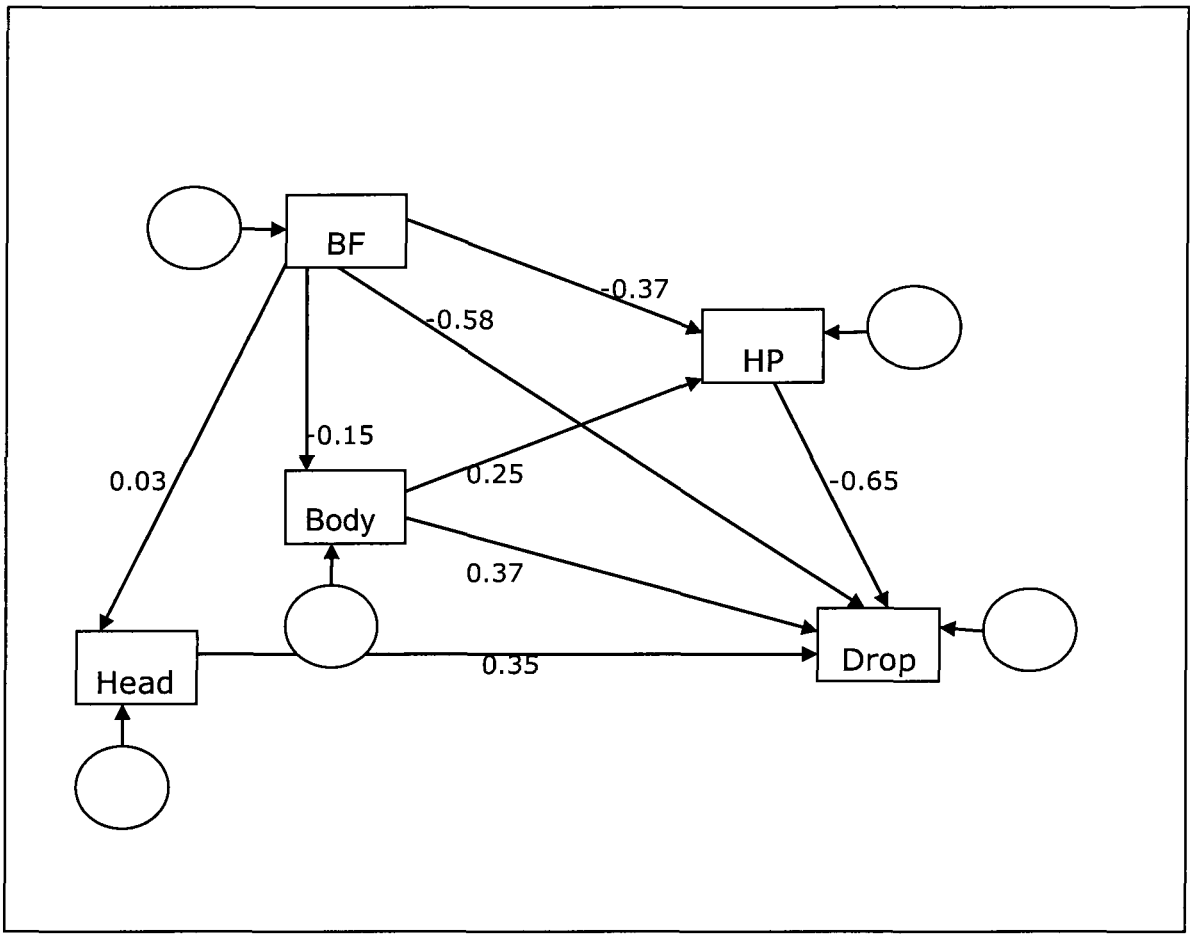


Figure 3. Model 3. Observed variables are represented as rectangles while error terms are represented by circles. A line with a single arrowhead represents a hypothetical direct causal effect of one variable on another and is also known as a path. Model 3: One exogenous variables percentage body fat (BF) loads onto all other variables; Body heat loss (Body) [with its effect on heat production (HP) and core temperature (Drop)], head heat loss (Head) as well as HP and the effect on Drop. The effects of HP and Head on Drop are also included. The model has 2 degrees of freedom. Standard estimates are given for each path.

## Evaluation of Models.

The $\chi^2$ statistic was used to measure overall fit and its significance was assessed at $\alpha = 0.05$. This likelihood ratio tests the null hypothesis that the model is correctly specified (39). A decrease or increase in the $\chi^2$ statistic was used to guide decisions regarding re-specification of the initial model. The likelihood ratio test based on the differences between the $\chi^2$ statistics and degrees of freedom of two models tested for significant difference between two models.

Model fit was also quantified using the root mean square error of approximation (RMSEA), which quantifies the lack of fit of the researcher's model to the population covariance matrix. It is typically reported along with its 90% confidence intervals. A common rule of thumb is that RMSEA $\leq$ 0.05 indicates close approximate fit; values between 0.06 and 0.10 indicate acceptable fit, and values greater than 0.10 indicate poor approximate fit (3). An incremental fit index, the normed fit index (NFI) (2), was also investigated. All incremental fit indices assess the relative improvement in model fit compared with the baseline or null model, which assumes zero population covariances among the observed variables. An accepted rule of thumb for this index is that a value greater than 0.90 indicate acceptable fit of the researcher's model (20).

Finally, the Standardized Root Mean Square Residual (SRMR) was selected as a measure of differences between observed and predicted covariances. The rule of thumb for this index is that a value of zero indicates perfect fit with any value < 0.05 acceptable while values > 1 indicate poor fit (24).

## RESULTS

The fit statistics for all models are presented in Table 1 and the correlation matrix is available as an appendix.

| | df | $\chi^2$ | p-value | RMSEA (90% CI) | SRMS | NFI |
|---|---|---|---|---|---|---|
| **MODEL 1** | 1 | 0.34 | 0.561 | 0 (0.0;0.70) | 0.048 | 0.96 |
| **MODEL 2** | 2 | 0.55 | 0.759 | 0 (0.0;0.40) | 0.062 | 0.94 |
| **MODEL 3** | 2 | 0.55 | 0.759 | 1 (0.0;0.39) | 0.062 | 0.94 |

Table 1. Fit Statistics for the three models includes degrees of freedom (df), Chi Squared values ($\chi^2$), p-values for $\chi^2$- statistics indicating how much of the variation is explained by the model, Root Mean Square Error of Approximation (RMSEA) as measure of parsimony, Standardized Root Mean Square residual (SRMS) measuring differences between observed and predicted covariances and Normed Fit Index (NFI), as an index of how well the model fits the data.

## Evaluation and interpretation of the models.

Model 1 fits the data well according to $\chi^2$ statistics (p=0.56) and normed fit index (0.96) while the residuals were small (SRMR = 0.048) (Table 2). This model confirms what we already know from previous experiments. Its significance lies in the fact that it is the first analysis showing the direct and interactive effects of more than one variable in a way that the effects could be compared with one another.

The overall fit of Model 2 was not significantly different than for Model 1 ($p > 0.05$). The effect of BF on Body HL (t-value = 0.51, $p > 0.05$) did not explain significantly more of the variation of the model. Model 3 included a path from BF on Head HL and included a path from BF to Head HL. This path was not significant ($t = 0.11$ , $p > 0.05$) and did not improve model fit when compared to Model 2, $p > 0.05$.

Figure 1 shows the standardized regression estimates[12] for Model 1. As expected, both HP (-0.64) and BF (0.37) had a negative direct effect on Drop. It was interesting that the direct effects of Body HL and Head HL on Drop were the

---

[12] The correlation between variables when all means equal zero and standard deviations equal one. This correlation (unlike unstandardized regression coefficients) is not affected by the metric of any of the variables 24. Kline R. *Principles and practice of structural equation modeling.* New York: Guilford, 2005.

same (~0.35). While this is the only effect of Head HL, the effect of Body HL on Drop is offset by its indirect effect through HP (-0.24). Although BF decreases HP and therefore indirectly increases Drop (0.24), its biggest effect is the direct attenuation of core temperature drop (-0.58).

| | Original Value | Lower CI | Upper CI | Bootstrap Mean |
|---|---|---|---|---|
| BF | -0.5003 | -0.7697 | 0.2129 | 0.4991 |
| Body HL | 0.1991 | 0.0502 | 0.3373 | 0.1994 |
| Head HL | 0.4499 | 0.1273 | 0.7646 | 0.4579 |
| Met Rate | -0.4055 | -0.5722 | -0.2616 | -0.4048 |

Table 2. Bootstrap results. Original values and 95% confidence intervals (CI) for regression coefficients for independent variables on the drop in core temperature (DROP) as calculated with the bootstrap method.

## DISCUSSION

This was the first time we used a multi variable analysis (SEM) to understand the effect of head cooling on core temperature. The model showed

that increased heat loss from both the head and body caused a drop in core temperature. This effect was attenuated by shivering only when the body was immersed in cold water. Body fat, although it suppressed the metabolic response, also attenuated core cooling.

While our previous experimental attempts focused on head cooling and possible mechanisms evoked from the dive response, this model focused on the differences between the protective responses of the cooled body versus the cooled head.

Data for the model were obtained from two previous whole head cooling studies (30,31). The paths of Model 1, the basic model, were based on results from a number of previous studies done on cooling of the body; both shivering heat production and percentage body fat delayed core cooling, while the shivering response was inversely related to the amount of body fat (36). Only heat loss from the body, not from the head, increased shivering heat production (30-32). Both heat loss from the body and the head increased core temperature drop. A second model tested weather the relationship between body fat and body heat loss would improve the effectiveness of the model to predict the effects of body and head heat loss on core temperature drop. Although there was an inverse relation between body fat and body heat loss, this model did not explain more of the variation on core temperature drop than Model 1. Similarly, Model 3 tested

weather the relationship between body fat and body heat loss would improve the effectiveness of the model to predict the effects body and head heat loss on core temperature drop. There was no significant relation between these two variables, and no improvement in model fit.

Allowing for all of these paths (effects) in the model, it was interesting to see that when controlled for other variables, the direct effect of HL from the body and head on core temperature drop was similar. It is important to note that while Head HL has only this one direct effect, Body HL also increases shivering heat production, and therefore indirectly counteracts cooling of the core.

An absence of increased heat production with head cooling, occurred despite a drop in skin temperature of the head (by ~15.0°C) and core temperature drop (of 0.3°C) [both skin and core temperatures are determinants of the shivering response (14,15,37)] and could be considered as one reason for increased core cooling rate during head submersion.

In the model there is a direct negative effect of BF on core temperature drop and our assumption is that this protection from body fat is mainly against body exposure and not head exposure. When heat is lost from the body surface, it affects the perfused mass (i.e. body core) as well as the non-perfused mass (i.e. subcutaneous fat).

When the head is also exposed to the cold water, the heat loss preferentially affects the body core - as a result of venous return from the cooled scalp and face to the heart - and not the non-perfused subcutaneous fat. This may, at least partially, explain why the 11% increase in total heat loss when the head is also immersed, causes a disproportional increase in core cooling of 38%, compared to body exposure only.

## Practical implications.

This study is an ongoing effort to try to explain and prevent symptomless hypothermia and consequent altered physical (11) and mental (10,28) capacity in scuba divers and recreational swimmers (e.g., adventure racers). As with our previous studies (9,28,30-32), the results indicate that the head should be kept out of the water as much as possible during cold water activities.

The authors of this paper are confident that the main factors playing a role in the effectiveness of head cooling over body cooling are increased HL, the absence of shivering heat production and the reduced protection provided by body fat. However, the mindful reader will notice that this still does not explain why dorsal head cooling in two previous studies (9,28) increased core cooling only when the body was exposed to cold. These studies were done in much colder water (10 - 12°C) than our whole head studies (17°C) which data were used for this model.

This still leaves the door open to the possibility that a smaller perfused mass could be responsible for the phenomenon found in dorsal head cooling studies only.

Further work is warranted to study core cooling rate and peripheral vasoconstriction during facial, dorsal and whole head and body cooling in colder water temperatures. The protective effect of body fat and muscle as a cold reservoir could be validated by comparing the amounts of these tissues with the afterdrop and amount of external heat donation required during rewarming. As well, from the results in our studies, it is not clear why head cooling, despite the lower head skin temperature and core temperature, does not elicit the shivering response. These values should be compared with models based on body-only immersion (36) to determine if the response is different between body and head cooling.

As a final note, the use of SEM for the analysis of this multivariable question seemed to work well, despite the small sample size. Because the studies used for the data in these models were designed for the use of an ANOVA, the number of subjects used in this study (14) is considered small for the use of SEM. While SEM is mainly used as a confirmatory approach, the models in this study are rather an exploratory approach to investigate the feasibility of using SEM in our research, despite smaller sample sizes. With the use of the bootstrap technique

we have confirmed that the regression coefficients of all independent variables fell within the 95% bootstrap confidence limits (see Table 1).

This model not only confirmed results from previous work, but also gave the opportunity for the researchers to compare the effectiveness of the variables (BF, Head HL, Body HL and HP) on the outcome variable (DROP) as well as determine the interactive effects of the independent variables. This model had the ability to show the significance of a variable (body fat) which was not possible with the use of the traditional repeated measures ANOVA.

REFERENCES

1. Alexander L. The Treatment of Shock from Prolonged Exposure to Cold, Especially in Water. Washington, DC: Office of the Publication Board, Department of Commerce. (Combined Intelligence Objectives Sub-committee), 1946, p. 1-228.

2. Bentler P. Comparitive fit indexes in structural models. Psychological Bulletin 107: 238-246, 1990.

3. Browne M and Cudeck R. Alternative ways of assessing model fit. . In: Testing structural equation models, edited by Bollen K and Long J. Newbury Park: Sage Publications, 1993, p. 136-162.

4. Chou C and Bentler P. Estimates and tests in structural equation modeling. In: Structural Equation Modeling: Concepts, issues and applications, edited by Hoyle R. Newbury Park, CA: Sage, 1995, p. 33-55.

5. Efron B. Better bootstrap conficence intervals. Journal of the American statistical society 82: 171-185, 1987.

6. Eyolfson D, Tikuisis P and Giesbrecht GG. Measurement and prediction of maximal shivering capacity in humans. In: Proceedings of the 8th Int. Conf.

on Environ. Ergonomics, edited by Hodgdon J and Heaney J. San Diego: NHRC, 1999.

7. Eyolfson D, Tikuisis P, Xu X, Weseen G and Giesbrecht G. Measurement and prediction of peak shivering intensity in humans. Eur. J. Appl. Physiol. 84: 100-106, 2001.

8. Froese G and Burton AC. Heat losses from the human head. J. App. Physiol. 10: 235-241, 1957.

9. Giesbrecht G, Lockhart T, Bristow G and Steinman A. Thermal effects of dorsal head immersion in cold water on nonshivering humans. J. Appl. Physiol. 99: 1958-1964, 2005.

10. Giesbrecht GG, Arnett JL, Vela E and Bristow GK. Effect of task complexity on mental performance during immersion hypothermia. Aviat. Space Environ. Med. 64: 206-211, 1993.

11. Giesbrecht GG and Bristow GK. Decrement in manual arm performance during whole body cooling. Aviat. Space Environ. Med. 63: 1077-1081, 1992.

12. Glickman-Weiss EL, Goss FL, Robertson RJ, Metz KF and Cassinelli DA. Physiological and thermal responses of males with varying body compositions

during immersion in moderately cold water. Aviat. Space Environ. Med. 62: 1063-1067, 1991.

13. Glickman-Weiss EL, Hearon CM, Prisby R and Caine N. The perceptual and physiological responses of high and low fat women exposed to 5°C air for 120 minutes. Wilderness Environ. Med. 9: 204-210, 1998.

14. Glickman N, Mitchell HH, Keeton RW and Lambert EH. Shivering and heat production in men exposed to intense cold. J. Appl. Physiol 22: 1-8, 1966.

15. Golden FSC, Hampton IFG, Hervey GR and Knibbs AV. Shivering intensity in humans during immersion in cold water. J. Physiol 290: 48p-49p, 1979.

16. Hayward JS, Eckerson JD , and Collis ML. Effect of behavioral variables on cooling rate of man in cold water. J. Appl. Physiol. 38: 1073-1077, 1975.

17. Hayward JS, Eckerson JD and Collis ML. Thermal balance and survival time prediction of man in cold water. Can. J. Physiol. Pharmacol. 53: 21-32, 1975.

18. Heistad DD, Abboud F and Eckstein J. Vasoconstrictor response to simulated diving in man. J. Appl. Physiol. 25: 542-549, 1968.

19. Hope A. A simplified Monte Carlo sig-nificance test procedure. Journal of the Royal Statistical Society, B30 3: 582-598. , 1968.

20. Hu L and Bentler P. Cutoff criteria for fit indices in covariance structure analysis: Conventional criteria versus new alternatives. Structural Equation Modeling 6: 1-55, 1999.

21. Jacobs I, Martineau L and Vallerand AL. Thermoregulatory thermogenesis in humans during cold stress. In: Exercise and Sport Science Reviews, edited by Holloszy JO. Baltimore: Williams & Wilkins, 1994, p. 221-250.

22. Johnston CE, Bristow GK, Elias DA and Giesbrecht GG. Alcohol lowers the vasoconstriction threshold in humans without affecting core cooling rate during mild cold exposure. Eur. J. Appl. Physiol. 74: 293-295, 1996.

23. Joreskog K and Sorbom D. Lisrel 8: User's reference guide. Chicago, IL: Scientific Software International, 1996.

24. Kline R. Principles and practice of structural equation modeling. New York: Guilford, 2005.

25. Layton R, Mints WJ, Annis J, Rack M and Webb P. Calorimetry with heat flux transducers: comparison with a suit calorimeter. J. Appl. Physiol. 54: 1361-1367, 1983.

26. LeBlanc J. Subcutaneous fat and skin temperature. Candian Journal of Biochemistry and Physiology 32: 354-358, 1954.

27. Lewis T. The blood vessels of the human skin and their responses: Shaw & Sons Ltd., 1927.

28. Lockhart T, Jamieson C, Steinman A and Giesbrecht G. Life jacket design affects dorsal head and chest exposure, body core cooling and mental performance in 10°C water. Aviat. Space Environ. Med. 76: 954-962, 2005.

29. MacCallum R, Browne M , and Sugawara H. Power analysis and the determination of sample size for covariance structure modeling. Psychological methods 1: 130-149, 1996.

30. Pretorius T, Bristow G, Steinman A and Giesbrecht G. Thermal effects of whole head submersion in cold water on non-shivering humans. J. Appl. Physiol. 101: 669-675, 2006.

31. Pretorius T, Cahill F, Kocay S and Giesbrecht GG. Shivering heat production and core cooling during head-in and head-out immersion in 17°C water. Aviat. Space Environ. Med. 79: 495-499, 2008.

32. Pretorius T, Gagnon D and Giesbrecht G. Head Immersion in Cold Water Elicits Vasoconstriction But no Shivering to Defend the Body Against Core Cooling. Submitted to Appl. Physiol. Nutr. Metab., 2008.

33. Pugh LGCE. Accidental hypothermia in walkers, climbers, and campers: Report to the medical commission on accident prevention. British Medical J. 1: 123-129, 1966.

34. Steinman AM, Hayward JS, Nemiroff MJ and Kubilis PS. Immersion hypothermia: comparative protection of anti-exposure garments in calm versus rough seas. Aviat. Space Environ. Med. 58: 550-558, 1987.

35. Thompson RL and Hayward JS. Wet-cold exposure and hypothermia: thermal and metabolic responses to prolonged exercise in rain. J. Appl. Physiol. 81: 1128-1137, 1996.

36. Tikuisis P. Prediction of the thermoregulatory response for clothed immersion in cold water. Eur. J. Appl. Physiol 59: 334-341, 1989.

37. Tikuisis P, Bell DG and Jacobs I. Shivering onset, metabolic response, and convective heat transfer during cold air exposure. J. Appl. Physiol 70: 1996-2002, 1991.

38. White MD, Ross WD and Mekjavic IG. Relationship between physique and rectal temperature cooling rate. Undersea Biomed. Res. 19: 121-130, 1992.

39. Widaman K and Thompson J. On specifying the null model for incremental fit indices in structural equation modeling. Psychological Methods 8: 16-37, 2003.

40. Xu X, Castellani JW, Santee W and Kolka M. Thermal responses for men with different fat compositions during immersion in cold water at two depths: prediction vs observation. Eur. J. of Physiol. 100: 79-88, 2007.

41. Xu X, Tikuisis P and Giesbrecht G. A mathematical model for human brain cooling during cold-water near-drowning. J. Appl. Physiol. 86: 265-272, 1999.

42. Young AJ, Castellani JW, O'Brien C, Shippee RL, Tikuisis P, Meyer LG, Blanchard LA, Kain JE, Cadarette BS , and Sawka MN. Exertional fatigue, sleep loss, and negative energy balance invrease susceptibility to hypothermia. J. Appl. Physiol. 85: 1210-1217, 1998.

# APPENDIX

## Correlation Matrix

| | BF | Body | Head | HP | Drop | Mean | SD |
|---|---|---|---|---|---|---|---|
| BF | **0.372** | | | | | 2.15 | 0.61 |
| Body | -0.152 | **0.960** | | | | 8.60 | 0.98 |
| Head | 0.031 | 0.124 | **0.160** | | | 0.58 | 0.40 |
| HP | -0.407 | 0.310 | 0.161 | **0.706** | | 2.28 | 0.84 |
| Drop | -0.372 | 0.312 | 0.277 | -0.247 | **0.260** | 0.68 | 0.51 |

The correlation matrix for all of the variables [percentage body fat (BF), heat loss from body (Body), heat loss from head (Head), heat production (HP) and drop in core temperature (Drop)] with means and standard deviations (SD). Variances are given on the diagonal (in bold).

# CHAPTER 5

# GENERAL DISCUSSION

# DISCUSSION

The purpose of this project was to determine if the body core cools faster when the head is exposed to cold water compared to body-only cooling when the body's shivering mechanism was intact. As well, the effect of facial immersion on the thermal core as a possible mechanism would also be determined. Other factors affecting the rate of core cooling such as body fat and shivering were also included in a structural equation model, (SEM) to compare the magnitude of these effects as well as the difference of these effects between head-only and body-only cooling.

The first study (described in chapter 2) confirmed an increased core cooling rate with head submersion that was disproportional to the increase in surface heat loss. The initial speculation of a decreased thermal core that was more likely to cool than when the body is less vasoconstricted (smaller thermal core) was not confirmed in the second study (described in chapter 3). It seems that it is rather increased heat loss from the head with no response of the body to counteract with increased metabolic heat production causing an increase in core cooling rate. A structural equation model (third study described in chapter 4) also estimated that body fat plays a major role in protection of the body core against cooling. Based on observations made during the head cooling studies and the fact that fat is more present on the rest of the body rather than the head, it was

assumed that this protective effect is mainly against body cooling and not head cooling.

Results from previous studies showed an increase in core cooling rates with dorsal head cooling only when the body too was cold stressed (1,2,10). Also, results of whole head cooling showed an increase in core cooling regardless whether the body was cooled or not (13). These results initially lead us to postulate that in the conditions where vasoconstriction is induced [i.e. in the body exposed conditions due to a cold stimulus (9) and the whole head submersion conditions due to the diving reflex (6)], core cooling rate was increased due to the effect of cooling a smaller thermal core (perfused body mass).

The first study (14) of this manuscript confirmed that head cooling increased core cooling rate even with the shivering mechanism intact. Shivering did not increase when the head was cooled and esophageal temperature consequently dropped due to increased heat loss. Head submersion increased the rate of core cooling both when the body was insulated from, and exposed to, 17°C water. The increase in core cooling was disproportionately greater than could be explained by the relative increase in heat loss alone (e.g., in the Exposed Head-in condition, head submersion accounted for an 11% increase in total heat loss, yet the rate of core cooling increased by more than 56% in both conditions).

An intact shivering mechanism seemed to attenuate core cooling (14) in all comparable conditions when compared to the previous head cooling study with shivering inhibited (13)s, but did not alter the effect of head cooling. Core cooling rates for shivering-inhibited and shivering-intact trials respectively, were: Insulated Head-out, 1.1 *vs* 0.2°C/h; Insulated Head-in, 1.6 *vs* 0.7°C/h; Exposed Head-out, 1.8 *vs* 0.7°C/h; and Exposed Head-in, 2.5 *vs* 1.1°C/h).

What was striking in both these studies whether shivering was inhibited or not, was that core cooling rates were similar whether the head only (Insulated Head-in) or the body only (Exposed Head-out) was exposed to cold water, despite a large difference in total heat loss (665±88 *vs* 1174±156 kJ respectively).

The concept of a smaller thermal core affected more than a larger thermal core by equal amounts of heat loss which was inherited from a previous dorsal head studies (2,10) still seems to work as an explanation for the current results. The speculation was that facial cooling would induce the diving reflex (an oxygen sparing mechanism eliciting breath hold, vasoconstriction and bradycardia) (17) and consequently reduces the thermal core.

The second study (15) was done to confirm the role of vasoconstriction as elicited by the diving reflex (an oxygen sparing mechanism eliciting breath hold, vasoconstriction and bradycardia) on the rate of core cooling when the face is cooled. The effect of dorsal, facial and whole head cooling (in 17°C water) on

peripheral vasomotion and core cooling was compared. In all conditions the body was in thermoneutral air (~28°C air). One limitation of this study was the difficulty to estimate the size of the thermal core as no direct measurement of it is possible. Vasoconstriction was assumed as a function of decreased finger tip blood flow which was measured as finger tip temperature and also moving red blood cells in the finger tip. A decrease in both of these measurements would imply peripheral vasoconstriction.

Vasoconstriction increased from dorsal to facial cooling and was even more increased when the whole head was cooled. However, the rate of core cooling, which increased form dorsal to facial to whole head cooling, was not affected by this and seemed to be only a function of heat loss from the head (0.0038°C/kJ). This finding is different from body cooling which is offset by a number of factors, one which is the onset of shivering (7).

The most striking result from this study was that there was no increase in metabolic heat production in any of the conditions, despite a drop in core temperature as well as head skin temperature, both which are considered determents of increased metabolism (19). Metabolic rate was predicted using a model (18) based on core and skin temperatures to test whether shivering was expected with this small drop in core temperature. This model over predicted

metabolic rate in all three conditions for comparable core and skin temperature reductions.

With these results known, a different approach to the results of the first study could be considered. The decrease in core temperature was calculated in terms of heat loss from the body and the head respectively. This implies that the increased heat loss from the head could be responsible for the increased core temperature drop, as seen in the second study. When this is considered, it shows that cooling the body is responsible for a drop of 0.0003±0.0003°C/kJ of heat lost from the body (insulated or exposed), while there is a drop of 0.0023±0.0004°C/kJ of heat lost from the head with the body insulated and 0.0022±0.0004°C/kJ of heat lost from the head when the body is exposed to cold water. This reduction in core temperature is almost 800% more when the head is cooled compared with when only the body is cooled per unit of heat lost, while increased heat loss was only 11%.

As in the first study limitations included a small sample size as well as the fact that only men volunteered. This limits generalizability to only men and only to the age group they belonged to (20 – 50 years of age).

From the results of the first two studies it was reasonable to conclude that the increase in heat loss from the head, with no defense from the body to increase metabolic heat production, could explain the increased cooling rate as

seen in both the head cooling studies. However, it still did not explain why in both head cooling studies [with (13) or without (14) the use of meperidine to suppress shivering], core cooling rates were the same when only the head or only the body was exposed to the cold water, despite the fact that the amount of HL from the exposed body was twice the amount of HL from the surface of the insulated body and the exposed head. The initial question -'Why is rate of core cooling increased by exposure of the head?' now changed to: 'What protects the core from cooling when the body is exposed, but not when the head is exposed to cold water?'. While core cooling rate seems to be a function of surface heat loss from the head (15), previous studies on body-only cooling revealed that the core cooling rate is offset by a number of physiological and biophysical factors.

The small sample size in the first two studies had the limitation of only allowing for the use of a one way ANOVA so that it would be difficult to test any other factors such as body fat or shivering on the effect of core cooling.

The purpose of the third study (16) was to estimate the separate effects of surface heat loss from the body and the head on core temperature drop with the interactive effects of age, body composition (% body fat, height and weight) and shivering. This multivariable research question required a statistical technique to not only estimate the effect of the all the variables on the outcome variable (core temperature), but also estimate the effect of the independent variables on

each other (e.g. the effect of heat loss from the body on shivering). To accommodate these needs, data analysis was done using SEM (8).

While our previous experimental attempts focused on head cooling and possible mechanisms evoked from the diving response, this model focused on the differences between the protective responses of the cooled body versus the cooled head. Data for the model were obtained from two previous whole head cooling studies (13,14). The paths of Model 1 (see figure 1 (16)) were based on results from a number of previous studies done on cooling of the body; Both shivering heat production and percentage body fat delayed core cooling, while the shivering response was inversely related to the amount of body fat (20). Only heat loss from the body, not from the head, increased shivering heat production (14,15). It is important to note that while head heat loss has only this one direct effect, heat loss from the body also increases shivering heat production, and therefore indirectly counteract cooling of the core.

There are two effects of subcutaneous body fat (BF); the greater of the two is the direct protection against core cooling, with a smaller, indirect effect suppressing the shivering response. In order to explain the protection of the core through BF two models were tested. In model 2 (see figure 2 (16)) an extra path was constructed from BF to Body HL, testing whether BF would reduce the amount of heat loss from the body. This effect was not significant (t = 0.51,

p>0.05), and the model did not explain more of the variation than Model 1 (p>0.05). Model 3 (see figure 3 (16)) tested this protective effect of fat on heat loss from the head, but it too proved to be insignificant.

## Limitations

1. Limitations of these studies include the testing of only men, which might limit findings to men only, as well as the relative small sample size which is common in this field of research but could potentially reduce the power of this study. As well the generalizability of studies with small sample sizes is limited to people with the same characteristics such as body fat, age etc.
2. The small sample size in the first two studies had the limitation of only allowing for the use of a one way ANOVA so that it would be difficult to test any other factors such as body fat or shivering on the effect of core cooling.
3. A limitation in the first study, because we did not expect these results, neither the amount of external heat donated nor the time it took to recover, were recorded. From records kept in the lab and memory, it seems as if it took at least 20 minutes for subjects to recover and during that time core temperature dropped (a.k.a. afterdrop) before it increased to normal levels.
4. As mentioned in the literature study (chapter 1) our research is limited by the fact that we could not directly measure brain temperature. Based on

the findings of Nelson (12) and Xu (23) that brain temperature mainly depends on heat transfer with arterial blood and less so on cooled blood from the face and scalp, it will be assumed that in all studies where the head is cooled, brain temperature will follow core temperature as measured in the esophagus and not change due to the conductive and/or convective heat loss through the skull and scalp. The assumption is that esophageal temperature represents the temperature of the heart (11) and thus arterial blood temperature to the brain and all other organs.

## Possible explanation for the results

It appears as if the increase in core cooling rate seen when the head is exposed to cold water, is mainly determined by the amount of heat loss from the head with no defense from the body to prevent this drop with increased metabolic heat production. The significance of this observation lies in the effectiveness with which the body could prevent, or at least delay, drop in core temperature during cold exposure with increased shivering heat production.

Studies done on cooling of the body surface show that the drop in core temperature for a given amount of heat loss could be offset by the initial responses of the body to cold. Both vasoconstriction and increased metabolic heat production have proved to reduce the effect of heat loss from the body surface on core cooling, while body composition factors such as fat and muscle

mass could also delay body core cooling (22). In the second study (15) increased peripheral vasoconstriction was seen as more of the head was exposed to the cold water, but it did not affect drop in core temperature in any of the conditions in any way. No metabolic heat production was elicited to defend the core from cooling in any condition and this supported results from our previous whole head cooling studies (13,14). This was seen despite a significant drop in core temperature as well as head skin temperatures - both determinants of the shivering response. Predicted metabolic responses based on core and head skin temperatures (19) over predicted the response in all conditions with 50 - 60W (table1 (15)).

The first two studies showed that the increased core cooling rate seen in the initial two head cooling studies could be explained by in an increase in heat loss through the head (11% of total heat loss) and the absence of metabolic heat production. This phenomenon only explained the increase in cooling rates when the head is exposed to the cold water, and not why the core cooling rates are similar when only the body or only the head (with body insulated from cold) is exposed to cold water despite a large difference in surface heat loss. To answer this question, a mathematical multivariate model was used to consider factors known to offset core cooling during body immersion. This SEM model showed that body fat had a significant negative effect on the core cooling rate as well as a smaller

negative effect on shivering. We made the assumption that the protection from body fat is mainly protection from surface heat loss from the body and not the head. This is based on the fact that there is more fat deposition on the rest of the body than the head. Also, our results (15) showed that core cooling is linearly related to heat loss from the head, and not offset by other factors. Further support for the assumption is based on results from the whole head cooling study (14) with shivering intact. This study showed a similar drop in core cooling when only the head was exposed with the body insulated from the cold or the whole body was exposed to the cold water. The researchers observed that in all the head submersion trials, although subjects were offered a warm bath afterwards, no one needed external heat donation to recover from the cold exposure. This was different from the body exposure experiments, where all subjects were so cold after the experiments and it seemed impossible for them to recover without the warm bath.

The assumption is made that the heat loss from the body surface causes vasoconstriction so that the heat from the core of the body could not be exchanged with blood flow to the vasoconstricted soft tissue (fat, muscles etc.) preventing the core from cooling. Only when the body is warmed and blood flow to the cold tissue restored, heat from the core is exchanged with the fat mass. From our observations we also know that fatter subjects spend more time to

recover from the experiments despite the fact that their core temperatures do not drop as much during the cold immersion compared to leaner subjects.

Since all of these studies were dealing with head cooling, it would be of value to know how the temperature of the brain is affected by it. As mentioned in the literature study (chapter 1) our research is limited by the fact that we could not directly measure brain temperature. Based on the findings of Nelson (12) and Xu (23) that brain temperature mainly depends on heat transfer with arterial blood and less so on cooled blood from the face and scalp, it will be assumed that in all studies where the head is cooled, brain temperature will drop due to the drop in core temperature as measured in the esophagus and not due to the conductive and/or convective heat loss through the skull and scalp. The assumption is that esophageal temperature represents the temperature of the heart (11) and thus arterial blood temperature to the brain and all other organs.

## Practical implications

These studies are part of an ongoing effort to try to explain and prevent symptomless hypothermia and consequent altered physical (5) and mental (4,10) capacity in scuba divers and recreational swimmers (e.g., adventure racers). As with our previous studies (2,10,13,15), the results indicate that the head should be kept out of the water as much as possible during cold water activities.

Although the practical implications of these studies are not different from our previous research, these studies serve as an ongoing attempt to explain the greater effect of head cooling on core temperature. The authors of this paper are confident that the main factors playing a role in the effectiveness of head cooling over body cooling are increased HL, the absence of shivering heat production and the reduced protection provided by body fat. However, the mindful reader will notice that this still does not explain why dorsal head cooling in two previous studies (2,10) increased core cooling only when the body was exposed to cold. These studies were done in much colder water (10 - 12°C) than our present whole head studies (17°C), the data from which, were used for this model. This still leaves the possibility that a smaller effective perfused mass could still be responsible for the phenomenon found in dorsal head cooling studies only.

The isolated effect of head immersion, even with a mild cold stress of immersion in 17°C water, can significantly decrease core temperature. Although the face can exert a greater core cooling effect than the head dorsum during the first 30 minutes of immersion, the mechanisms for this increase are not fully understood. Further studies measuring regional body heat contents and whole limb blood flow may help elucidate the mechanisms for our results.

This is the not the first time that body fat is described as a 'cold sink' protecting the core from cooling during body immersion in cold water (21). These

speculations should be validated in studies where amount of body fat and muscle are plotted against measurements of exterior heat donation and afterdrop in the core temperature during recovery from cold exposure. This would give an idea of the amount of 'heat debt' of the body. It almost looks as if one should distinguish between external and internal insulation factors. External insulation such as a dry suit with insulative undergarments protects the body against heat loss and core cooling as seen in the Insulated conditions in the first study (14), while internal insulation such as muscle and fat tissue would lose some heat to the environment but because of vasoconstriction, this cold tissue does not exchange heat with the core. This mechanism remains effective until blood flow to the tissue is restored either because of increased metabolic requirements (shivering) (22) or warming of the skin (warm bath) resulting in further cooling of the core.

As a final comment, the use of SEM for the analysis of this multivariable question seemed to work well, despite the small sample size. This model not only confirmed results from previous work, but also gave the opportunity for the researchers to compare the effectiveness of the variables (BF, Head HL, Body HL and HP) on the outcome variable (DROP) as well as determine the interactive effects of these independent variables. This model had the ability to show the significance of a variable (body fat) which was not possible with the use of the traditional repeated measures ANOVA. For the future it would be of value to

redo this model with a bigger sample, where the subjects are only exposed to one of the conditions and compare the results to the present model. This approach will give the opportunity to include a larger range of subjects regarding sex, body fat and muscle to further strengthen the validity and application of the model to a larger part of the community.

## REFERENCES

1. Alexander L. The Treatment of Shock from Prolonged Exposure to Cold, Especially in Water. Washington, DC: Office of the Publication Board, Department of Commerce. (Combined Intelligence Objectives Sub-committee), 1946, p. 1-228.

2. Giesbrecht G, Lockhart T, Bristow G , and Steinman A. Thermal effects of dorsal head immersion in cold water on nonshivering humans. J. Appl. Physiol. 99: 1958-1964, 2005.

3. Giesbrecht G and Pretorius T. Survey of Public Knowledge and Responses to Educational Slogans Regarding Cold Water Immersion. Wilderness Medicine Newsletter Awaiting Approval, 2008.

4. Giesbrecht GG, Arnett JL, Vela E and Bristow GK. Effect of task complexity on mental performance during immersion hypothermia. Aviat. Space Environ. Med. 64: 206-211, 1993.

5. Giesbrecht GG and Bristow GK. Decrement in manual arm performance during whole body cooling. Aviat. Space Environ. Med. 63: 1077-1081, 1992.

6. Heistad DD, Abboud F and Eckstein J. Vasoconstrictor response to simulated diving in man. J. Appl. Physiol. 25: 542-549, 1968.

7. Hemingway A. Shivering. Physiol. Rev. 43: 397-442, 1963.

8. Kline R. Principles and practice of structural equation modeling. New York: Guilford, 2005.

9. Lewis T. The blood vessels of the human skin and their responses: Shaw & Sons Ltd., 1927.

10. Lockhart T, Jamieson C, Steinman A and Giesbrecht G. Life jacket design affects dorsal head and chest exposure, body core cooling and mental performance in 10°C water. Aviat. Space Environ. Med. 76: 954-962, 2005.

11. Moran DS and Mendal L. Core temperature measurement; methods and current insights. Sports Med. 32: 879-885, 2002.

12. Nelson DA and Nunneley SA. Brain temperature and limits on transcranial cooling in humans: quantitative modeling results. Eur. J. Appl. Physiol. 78: 353-359, 1998.

13. Pretorius T, Bristow G, Steinman A and Giesbrecht G. Thermal effects of whole head submersion in cold water on non-shivering humans. J. Appl. Physiol. 101: 669-675, 2006.

14. Pretorius T, Cahill F, Kocay S and Giesbrecht GG. Shivering heat production and core cooling during head-in and head-out immersion in 17°C water. Aviat. Space Environ. Med. 79: 495-499, 2008.

15. Pretorius T, Gagnon D and Giesbrecht G. Head Immersion in Cold Water Elicits Vasoconstriction But no Shivering to Defend the Body Against Core Cooling. Submitted to Appl. Physiol. Nutr. Metab., 2009.

16. Pretorius T, Lix L and Giesbrecht G. Shivering Heat Production and Body Fat Protects the Core from Cooling During Body Immersion, but not During Head Submersion, a Structural Equation Model. J. Appl. Physiol., submitted for review, 2009.

17. Schagatay E and Holm B. Effects of water and ambient air temperatures on human diving bradycardia. Eur. J. Appl. Physiol. 73: 1-6, 1996.

18. Tikuisis P and Giesbrecht G. Prediction of shivering heat production from core and mean skin temperatures. Eur J Appl Physiol 79: 221-229, 1998.

19. Tikuisis P and Giesbrecht G. Prediction of shivering heat production from core and mean skin temperatures. Eur. J. Appl. Physiol. 79: 221-229, 1999.

20. Tikuisis P, Gonzalez RR, Oster RA and Pandolf KB. Role of body fat in the prediction of the metabolic response for immersion in cold water. Undersea Biomed. Res. 15: 123-134, 1988.

21. Vanggaard L, Eyolfson D, Xu X, Weseen G and Giesbrecht GG. Immersion of distal arms and legs in warm water (AVA Rewarming) effectively rewarms hypothermic humans. Aviat. Space Environ. Med. 70: 1081-1088, 1999.

22. Xu X, Castellani JW, Santee W and Kolka M. Thermal responses for men with different fat compositions during immersion in cold water at two depths: prediction vs observation. Eur. J. of Physiol. 100: 79-88, 2007.

23. Xu X, Tikuisis P , and Giesbrecht G. A mathematical model for human brain cooling during cold-water near-drowning. J. Appl. Physiol. 86: 265-272, 1999.