THE REWARMING EFFECTS OF RADIANT HEAT ON THE BLUSH AREA IN

POSTOPERATIVE CARDIAC SURGICAL PATIENTS.

by

Edward V. Cushing

B.Sc., Simon Fraser University, 1994

THESIS SUBMITTED IN PARTIAL FULFILLMENT OF

THE REQUIREMENT FOR THE DEGREE OF

MASTER OF SCIENCE

in the School

of

Kinesiology

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SIMON FRASER UNIVERSITY

June 1997

0-612-24115-7

APPROVAL

NAME: Edward V. Cushing

DEGREE: Master of Science

TITLE OF THESIS: THE REWARMING EFFECTS OF RADIANT HEAT ON THE BLUSH AREA IN POSTOPERATIVE CARDIAC SURGICAL PATIENTS

EXAMINING COMMITTEE:

Chair: Dr. T. Milner

_____

Dr. Tom Calvert
Senior Supervisor
Professor, School of Kinesiology

_____

Dr. Keith Mills
Clinical Assistant Professor
Department of Anaesthesiology
Faculty of Medicine
Vancouver Hospital and Health Science Centre
The University of British Columbia

_____

Dr. John Dickinson
Professor, School of Kinesiology

_____

Dr. Glen Tibbits
External Examiner
Professor, School of Kinesiology

Date Approved: June 5, 1997

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ABSTRACT

Postoperative shivering and hypothermia are common problems seen in patients recovering from anaesthesia. Both of these post-operative conditions are dangerous to the recovering patient and require intervention. Although there are various techniques available to treat postoperative shivering and the underlying hypothermia, the incidence of occurrence is still unacceptable. The aim of this study was to test the hypotheses that applying radiant heat on the blush area inhibits shivering, as well as inhibiting peripheral vasoconstriction, thereby increasing the effectiveness of the convective rewarming technique. In a vasodilated individual more heat energy is transferred to the venous blood in the periphery, and therefore, to the patient's core. Applying radiant heat to the blush area activates the trigeminal nerve heat sensors. This heat stimulation overrides the cold sensors' input to the hypothalamus, and thereby lowers the vasoconstriction and shivering thresholds. This mechanism thus helps to prevent vasoconstriction of the periphery, as well as inhibiting shivering. Post-operative cardiac surgical patients were divided into two rewarming groups consisting of: 1. the convective rewarming technique (BH) (n=16), 2. the combination of convective rewarming technique and radiant heat (41°C) on the blush area (RH) (n=17). Both groups were investigated from entry into the postanaesthetic recovery room to normothermia, by continually measuring core temperature (Tc, °C), mean skin temperature (Tsk, °C), forearm-fingertip temperature gradient (Tsk-gr), and shivering by the VOSS scale and electromyography (EMG). Patients with radiant heat required significantly less rewarming time (p=0.0016), with the rewarming rate (°C/min) being significantly higher (p=0.004). There was no difference between groups in mean skin temperatures at minute 1, but the radiant heat group was significantly warmer (p<0.0001) at minutes 26, 52, and 72. The forearm-fingertip temperature gradient measure which reflects peripheral vasoconstriction indicated there was no statistical difference between groups at minute 1, but the radiant heat group was significantly less

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vasoconstricted (p=0.0072) at minutes 25, 50, and 70. The radiant heat group shivered significantly (p=0.000023) less often and less severely than the convective heat group by the VOSS measure. Collectively these results strongly support using radiant heat as an integral rewarming technique for postoperative cardiac surgical patients. This study found that applying radiant heat on the blush area inhibits shivering and peripheral vasoconstriction, thereby leading to a more effective postoperative rewarming technique. Clinically these findings translate into a healthier postoperative recovery for patients.

DEDICATION

In memory of Dr. Tom Richardson. My mentor, teacher, supervisor, colleague, and friend; whose favorite question was
"Why do you think that is?"

You inspired all those around you to strive for excellence, and make the most out of every day.

Tom, You are dearly missed.

ACKNOWLEDGMENTS

I would like to thank Pam Merrick from the Department of Anaesthesiology at Vancouver Hospital and Rob Balshaw at Simon Fraser University's Statistical Consulting Service for their assistance with experimental design and statistical advice.

I would also like to thank the Vancouver Foundation for the generous grant; Innovative Industries Incorporated for engineering and constructing the prototype radiant heater; Teri Lydiard at the Industrial Liaison Office at Simon Fraser University for her substantial aid in the business aspects of this research; Rob Taylor and Mel Frank for their technical support from SFU; and Quest Scientific for the use of the HumBug. My friend and business partner, Doug Bourne, for his work and energy in helping initiate this project. All my G.P. friends who generously volunteered their time and bodies *in the Name of Science*.

I would especially like to thank my family and friends for the love, unlimited support and understanding they gave me throughout my Master's degree.

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1.1 Thermoregulation in Humans

1.1.a Introduction

Thermoregulation in humans includes two intertwined forms of control. One form is the conscious control over behavioral thermoregulation. This includes putting on a jacket, going inside to a warmer environment, curling up, having a hot or cool drink, shedding clothes, sitting in the shade, and so forth. The other is the autonomic control over shivering, the metabolic system, the sweat glands, and the vasomotor system.

Human physiology functions efficiently in a very narrow temperature range, thus the regulation of body temperature necessitates extreme efficiency. When body temperature does move from its normal range, which is around 37°C, it is referred to as hyperthermia or hypothermia. The occurrence of hyperthermia exists when the mean body temperature exceeds its normal temperature range. Hypothermia is a decrease in the mean body temperature, with 36.5°C being considered the beginning of mild hypothermia. The actual range of normothermia where no active thermoregulation by sweating or shivering occurs is usually around 0.4- 0.6°C in width around 37°C.[1,2,3] This is known as the thermoneutral zone (TNZ). The thermoregulatory system in mammals, and specifically humans, is very complex. "The active components of the thermoregulatory system form a highly redundant network of negative-feedback loops. The central nervous

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structures subserving controller functions are driven by inputs from multiple signal generators and act on multiple and variable input-output connections."[4]

The thermoregulation system in man has been described in engineering terms with inputs, integrators, outputs, and feedback loops. For temperature regulation to occur, "there must be some form of comparison or counteraction between two quantitatively different functions related to (temperature), and this will be somewhere on the feedforward pathway from disturbance-sensor(s)-to-correction-effector(s); and there must be the feedback of the influence of the correction effector(s) upon the disturbance sensor(s)."[5] This control system functions to maintain homeostatic systems in a very precise physiological temperature range. The body attempts to maintain this temperature range by reacting to feedback and feedforward input, error signals which indicate there are or will be thermal deviations. The input is from various thermosensors around the body, for instance areas in the central nervous system (CNS), skin and viscera.[2] The areas in the CNS, consisting of the brain stem and the spinal cord, integrate and regulate the information from the thermosensors. The effectors that react to compensate for any thermal stress include the metabolic rate, muscular system, sweat glands, and the blood vessels which together act to balance heat gain with heat loss.[2]

The autonomic system essentially uses only one mechanism for heat gain, - metabolism. The metabolic process gives off heat energy as a waste product. The higher the metabolic rate, the higher the heat gain. The thermoregulatory mechanism of shivering uses the musculature to drastically increase metabolism and heat production.

In studying thermoregulation the body is often modelled as a cylinder within a cylinder to describe the control of heat loss. The inner cylinder represents the core, (T_{co})

for example the brain, spinal cord, heart, lungs, and abdomen. The outer cylinder represents the periphery, (T_{sk}) which includes the skin, subcutaneous blood vessels and tissues, fingers, toes, ears and the nose. The body is modelled in this way to take account of the temperature gradient between the two cylinders, the core and the periphery. Brengelmann (1989) critiques the model suggesting that the true complexity of body temperatures are concealed behind the simplistic concepts of T_{co} and T_{sk}.[6] He does concede, however, that the model is useful for the study of changes in skin blood flow and clothing insulation properties.[6] These two cylinders are constantly shifting volumes between each other to maintain the mean body temperature within its temperature range by controlling the body's rate of heat loss.[6] Heat loss is accomplished by taking advantage of four basic mechanisms; conduction, convection, radiation, and evaporation.[7] Heat transfer by conduction occurs in solids due to internal temperature gradients and gradients between two or more solid bodies in close contact. Convection involves heat transfer by bulk movement of a fluid or gas down a temperature gradient. Heat transfer by radiation occurs since all bodies emit electromagnetic radiation, varying with the quantity of thermal energy transported. Another mechanism that can account for a large amount of heat loss from a body is evaporation. The evaporation of a fluid from a body surface consumes its latent heat of vaporization and significant heat exchange occurs. The larger the blood volume exposed to the ambient temperature through peripheral vasodilation, the greater the effect the environment can have on the body temperature.

1.1.b Thermoregulation

Today's commonly held theories rely on current neurophysiology and not on speculative engineering or neurophysiological hypotheses.[5,2] In the following, Bligh (1990) discusses what he considers the skeleton framework for a model of thermoregulation in mammals, structured only on current neurophysiology.

> A personal preference for the proffered model rests on (1) the existence of two populations of thermosensors with reciprocal activities over the same range of local temperature; (2) the common occurrence in the CNS of the reciprocal inhibition first postulated by Sherrington (1906) to account for the non-overlapping activities of counteracting flexor and extensor musculatures; and (3) the inevitability of the massive convergence of excitatory and inhibitory influences at each synapse of the thermosensor to thermoregulatory effector pathways, such occurrences forming the basis of the integrative role of the CNS.

This basic model of thermoregulation is the most current and widely accepted due to its simplicity.

1.1.b.i Mean Body Temperature

Previous theories of the thermoregulation of humans included identification of the hypothalamic temperature as the regulated variable. This notion has been extensively modified to support the theory that the regulated variable is a weighted mean body temperature.[4,5,6] Some factors that contest the older theory that the hypothalamus is the sole regulated variable is its anatomical location,[5] and researcher's findings of many other

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highly thermosensitive tissues.[5,4] It is believed that the hypothalamus is more

representative of mean brain temperature than mean body temperature since it is

influenced by contrasting heat exchange between arterial and venous blood supply to the

brain.[5] In finding the existence of various thermosensitive tissues around the body,

researchers endeavoured to identify their function. They found that they do in fact

contribute to the regulation of body temperature. The studies suggest that

thermoneutrality is maintained by the processing of afferent information from

thermoreceptors everywhere around the body, such as skin, internal organs, and the

brain.[8,4] All the thermosensitive tissues must contribute to create a "multiple input

system, in which the controlled variable is a function of several local temperatures rather

than a single area within the body."[9] Bligh (1990) stated that "the prevailing view is that

there may be no such thing as one regulated temperature, and that a weighted mean body

temperature is as near as one can get to expressing the regulated variable."[5]

1.1.b.ii The Regulatory System

    Another previously held theory in thermoregulation included the hypothalamus as

the sole thermoregulating center in the body. There is much evidence supporting the

belief that the posterior hypothalamus is very important as an integrator in

thermoregulation.[9] Studies have shown that even though the hypothalamus is most

probably the most significant area involved in thermoregulation, it is probably not the

only one.[5,9,10,4,8] Researchers have presented evidence of integration occurring throughout

the CNS, from medullary to spinal cord levels.[5,9,10,4,8] For example, studies on rats with

pre-optic lesions measured three major responses, shivering, nonshivering thermogenesis,

and vasoconstriction.[10] If the hypothalamus were to be the only structure within the CNS

capable of thermoregulation, then no appropriate responses to thermal insults would be

possible. These researchers found that over the course of many months the autonomic

responses to cold recovered independently.[10] It is clear that the hypothalamic area is not

the exclusive thermoregulatory center as previously believed. This theory has been

modified to include other areas within the CNS as integrators.

Although the hypothalamus is not the sole integrator of the thermoregulatory

system, it still plays an important role. It is believed that the multiple integrators

throughout the CNS can function alone, as seen, but act together in a hierarchical system.

A hierarchical system acts with many possible levels of individual control, but normally

each level is regulated by a single dominant drive. It is believed that the hypothalamus

represents the highest level in this hierarchy of CNS thermal integration.[4,8,5,10] The

hypothalamus does not function alone.[8,10,4] There is evidence or thermoregulatory

control throughout the brain stem, including the spinal cord.[8,10,4] Thermoregulation

consists thus of a hierarchical control system with interrelated yet individually functioning

parts.[8,10,4] It appears that the lower thermoregulatory structures are modified and

inhibited by those above, up to the highest level of the hypothalamus.[10] Evidence

supports the theory that thermoregulation in humans consists of a very complex

hierarchical multi-structural and interactive system.

1.1.b.iii Lateral Inhibition and the Thermoneutral Zone

Experimental evidence has shown that the various multiple integrators throughout the CNS transform the afferent thermal information into efferent signals that conduct the thermoregulatory effectors.[4] (For a description of the thermosensors, see below and 1.1.d Thermosensitivity.) The most obvious question to this description of regulation is *How?* The general tenet of regulation is that there be two quantitatively differing responses to the variable being regulated.[5] This is the basis for the explanation suggesting paired sensor activity may be responsible for thermal integration. Vendrik (1959) proposed that the set-point determinant could be the reciprocal activity of two populations of thermosensors.[11] Since there are two populations of thermosensors, warm and cold, the requirements are met. More specifically, there are two sensor-effector pathways in the system and they regulate by interacting.[5] Bligh (1990) suggest these pathways consist of warm sensors to heat loss effectors, and cold sensors to heat production effectors, with reciprocal inhibition between them.[5] It has been proposed that the warm sensors from all over the body, such as the hypothalamus, skin, and gut, converge on a common pathway before reaching the point of crossover inhibition, as do the cold sensors.[5,3] Thermoregulation is thought to be controlled by lateral inhibition between the cold and warm thermosensors.[5,2,9,3] Lateral inhibition is common in neural control systems. As with other functions, lateral inhibition is dependent on the intensity of each input. If one input is much greater than the other, lateral inhibition will enhance the dominant signal, and abolish that of the weak signal. The dominant signal's effectors will then be active. If the signals are equal, or very similar, neither signal will be

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represented. This appears to be true in thermoregulation. As the temperature increases higher than normothermia, the warm sensors' neural activity will increase, and the cold thermosensors' activity will decrease. Through integration via lateral inhibition of the two pathways, this relationship will cause the heat loss effectors' activity to increase. As the temperature decreases lower than normothermia, the cold sensors' activity will increase, and warm sensors' activity will decrease. Through integration, this relationship will cause the heat production effectors' activity to increase. In normal thermoregulation, there is a narrow range (0.4-0.6ºC) in which neither the sweating or shivering effector is active.[1,2,3] This range has been referred to as the null zone, the neutral range, dead band, thermoneutral zone, and many others.[5,2,12,13,4] The thermoneutral zone is quite narrow, magnifying the incredible control that occurs in thermoregulation when considering how rarely one's average body temperature actually shifts far from the normothermic range.

Figure 1. The Thermoneutral Zone Theory

fig 1.[13]
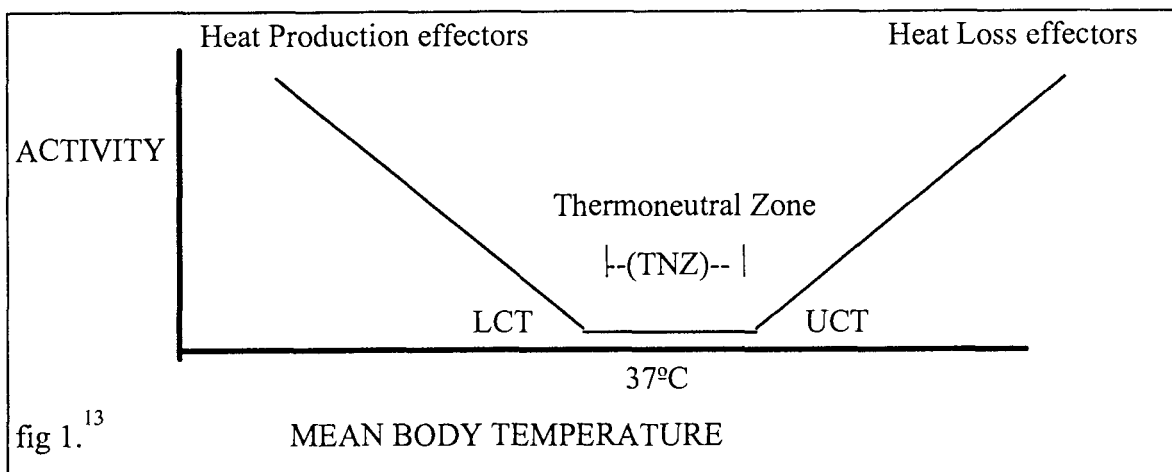
Within the thermoneutral zone heat production by shivering and heat loss by evaporation of sweat are inoperative, which means that heat loss and heat production are balanced and maintain a stable body temperature.[5,2] The thermoneutral zone's boundaries

are the lower critical threshold (LCT) or the shivering threshold, and the upper critical threshold (UCT) or the sweating threshold.[5,2,13] The LCT is the warmest mean body temperature at which shivering is initiated, and the UCT is the coldest mean body temperature at which sweating is initiated.[2,13] It is important to understand that the thermoneutral zone is dynamic; it does shift slightly. The thermoneutral zone will shift daily, monthly, seasonally, between activities, and with individual differences between persons.[5,2,13,4] The zone therefore shifts via intrinsic and extrinsic influence. Mekjavic, Banister, and Morrison (1988) suggested that these varying internal and external factors, including behavioural means of thermoregulation, make it impossible to label absolute values to the ambient temperatures defining the TNZ boundaries.[13] Also, no absolute values can be attributed because of intrinsic variations that cause shifts in the TNZ.[2] Some factors that affect the thermoneutral zone are exercise, fever, warm and cold environment adaptation, drugs and anaesthesia.[9,2,13,3]

One type of effector that is active in the thermoneutral zone is the vasomotor mechanism. The vasomotor mechanism is thought to have its own null zone within the thermoneutral zone, where there is neither active vasodilation or vasoconstriction in response to thermoregulative drives. There are vasodilation and vasoconstriction temperature thresholds, which when reached trigger active vasodilation or vasoconstriction, respectively.[14] Our core temperatures rarely shift far from the thermoneutral zone because of the subtle actions of the vasomotor system and behavioral adjustments.[13]

1.1.c Nervous control of effectors and their actions

There are a small number of autonomic mechanisms that actively work to either increase heat loss, or, decrease heat loss and increase heat gain. These include shivering, nonshivering thermogenesis, vasomotor control, and sweating. These are controlled through the neural system; the hormonal system acts primarily for long term adaptations.[9]

1.1.c.i Shivering

Shivering effectors are innervated by motor neurons descending from the hypothalamus.[15,9] The axons join the supraspinal motor pathways making contact with motoneurons in the anterior horns of the spinal cord.[15,9] The motoneuron axons then make their way to the various muscles involved in shivering. Normal thermoregulatory shivering is characterized by overt tremorlike movements[16] with contraction of antagonistic muscles[17], and in electromyography (EMG) as being oscillatory, within the 5-7 Hz frequency range.[18,3] The movements of shivering "are believed to be generated at the segmental level by proprioceptive feedback loops, the inherent rythmicity of the spinal cord, or some combination of both."[16] The mechanism of shivering can include, when at maximal shivering, a great majority of the skeletal muscles in the body. Shivering intensity occurs on a continuum up to a maximum, and has been observed to 'wax and wane' in intensity.[16] At the beginning of a shivering epoch, muscles of the head and neck, such as the masseter, sternocleidomastoid, and trapezius, are the first to contract.[19,20] The

response of shivering is proportional to the insult, so the colder the insult, the greater the shivering response with more and larger muscles joining in at greater intensities.[4] The occurrence of shivering in an individual produces a relatively small amount of heat energy compared to the huge amount of energy stores utilized.[21] The cost of shivering to the individual can be quite high. Oxygen consumption (VO_2) has been observed to increase three to five times in shivering individuals.[21,22,23,24,25,26] In a healthy individual these requirements may not be significant, but in individuals with compromised myocardial function, shivering can be very demanding and dangerous. Shivering leads to an increase in oxygen consumption,[24,23,21,27,25,22,28,29,26] carbon dioxide production,[24,23,25,22,26] heart rate,[23,21,25,22] rate pressure product,[23,25,22] and mean arterial pressure.[23,21,25,22] The increase in rate pressure product and heart rate suggests an increase in myocardial oxygen consumption (MVO_2)[23,25,22,30,31,32] and myocardial work.[23,25,22,30,30,31,32] An increase in myocardial work in a normal healthy individual is generally a minor stress, but to an individual with compromised myocardial function, it can be severely detrimental to their health.

1.1.c.ii Vasomotor thermoregulation

The vasomotor system has a very large role in control of body temperature. The blood vessels dictate the peripheral surface area available to the blood, which essentially directs the rate of heat loss. The greater the volume of blood allowed into the periphery, the greater the rate of heat loss through the heat loss mechanisms previously described. Controlling the rate of heat loss is important considering thermal balance equals heat gain minus heat loss. The effects of vasomotor control are particularly felt in the thermoneutral zone, where active thermoregulation by shivering or sweating does not occur. The vasomotor control of thermoregulation also has a null zone where there is essentially no activity. When mean body temperature reaches these thresholds, active vasomotor thermoregulation occurs.[14,33]

The characteristics of skin microanatomy over the entire body are not necessarily uniform. There are different characteristics of cutaneous microcirculation depending on the area of the body. The entire surface area of the skin has capillary, or 'nutritional,' blood flow. The fingers, hands, and toes, on the other hand, also have arteriovenous anastomoses, or A-V shunts.[34,35,36,37,38,39] This dual circulation is unique to the above mentioned areas in cutaneous tissue. Grant and Bland (1931) dissected fresh cadavers and found a large number of A-V shunts in the nail beds and tips of the fingers, less numerous in the palm of the phalanges, and none in the dorsum of the hand or flexor surface of the forearm.[35] This variation in microcirculatory anatomy has exhibited different physiological responses, with respect to magnitude, than capillary vasculature. Lewis (1930-31) and Grant and Bland (1931) observed significant differences in local responses

to cooling between tissues of the fingertips and the forearm.[40,35] The significance of A-V

shunts is that their anatomy suggests a thermoregulatory function.[35,41,39] Hales (1986)[41]

discusses the possible functions of the arteriovenous anastomoses (AVAs) found in the

hands and feet. Upon measuring cutaneous heat exchange in sheep, Hales found that heat

loss is very high when AVAs are dilated, irrespective of capillary blood flows.[41] Hales

(1986) suggested that the functioning of AVAs is related to heat dissipation.[41] This

suggests that the existence of A-V shunts in the hands and feet represent a specialized

thermoregulatory effector beyond which is seen in capillary vasculature. Coffman (1972)

observed that sympathetic nervous system activity exerts a greater effect on finger

arteriovenous shunt flow than on nutritional flow.[38] Also, Hales (1986) observed that in

anaesthetized rabbits' tissue containing A-V shunts, while nutritional blood flow is

responsive to local temperature effects yet independent of sympathetic nerve activity,

"indirect heating reflexly evoked AVA dilation, apparently by withdrawal of constrictor

tone."[41] These observations support the hypothesis that arteriovenous anastomoses appear

to have a role as thermoregulatory effectors, specifically heat loss effectors.

1.1.d Thermosensitivity

Inputs to the thermoregulatory system consist of neural temperature sensors from

around the body.[8] The temperature information is from cutaneous thermosensors and

deep body thermosensors.[2] Researchers found that there are many sources from which

thermal information originates. They found thermosensory inputs originating in the spinal

cord, the midbrain, and the lower brain stem.[4,8] These discoveries helped form the

foundation for the multiple-input concept of thermoregulation.[4] The multiple-input

concept of thermoregulation is simply that there are many sources of input from all over

the body that affect the regulation of body temperature, not merely one source. Also,

tissues that show thermosensitive properties are not limited to the neural system.

Thermosensitive tissues have been identified in the skin, gut, and muscular system.[9,4]

There are two different types of thermosensors, warm and cold.[4] At constant

temperatures the thermosensor's discharge frequencies are at steady state. When there is

a change in temperature, the appropriate type of thermosensor increases its neural

discharge frequency proportionally to the amplitude of the temperature change, while the

opposing thermosensor decreases its neural discharge frequency. If the temperature

increases, the warm thermosensor's discharge frequency increases; while if the

temperature decreases, cold thermosensors increase their discharge frequency.

The cutaneous thermosensors are very active as feedforward indicators. If the

environment suddenly becomes cool, as when one walks out from a warm house on a cold

and snowy winter day, the skin's cold thermosensors will increase their activity, and

thereby send this change in temperature information to the CNS. The CNS will cause the

effectors, such as peripheral vasoconstriction, to act *before* the body actually becomes

cold, to prevent heat loss. This is an example of the neural feedforward system in

thermoregulation.

Bligh (1990) suggests that the primary feedback mechanism in thermoregulation is

not directly due to neural processes, but is by the circulating blood's thermal influences

on the thermosensors.[5] This essentially means that the neural sensors throughout the

body, such as at the hypothalamus, brain stem, spinal cord, gut, and muscles, are effected by the circulating blood. The arterial blood "is rapidly influenced by every change in heat production and heat loss, and the body-wide distribution of the arterial blood means that changes in arterial blood temperature could act upon all the thermosensors of the body, wherever they are located."[5] Since there are temperature gradients throughout the body, arterial blood is modified to variable extents before reaching the capillaries.[5] Upon reaching the capillaries, the variable blood temperatures, therefore, cause equally variable afferent feedback from the widely distributed thermosensors.[5]

1.1.d.i Deep Body Thermoreception

There are a few general areas deep in the body that have been positively identified to have thermoreceptors. The preoptic area of the hypothalamus contains both warm and cold sensors.[9,10,4,8] Appropriate thermoregulatory responses have been elicited by locally cooling and warming this area; the responses include metabolic reactions and vasoconstriction, and vasodilation and panting/sweating, respectively.[9,10] Interestingly, it has been found that there is a larger population of warm sensors than cold in the hypothalamus.[9,10,4] The spinal cord is another deep area which has thermosensors. Both types of sensors have been found, but the cold sensors are rarely activated in natural conditions.[9] Researchers have found local thermoresponsiveness in the lower brain stem levels, the anterior hypothalamus, the pre-optic region, and the spinal cord, including the anterolateral tracts.[4] Warm and cold sensitive thermosensors have been identified outside

the CNS, such as vagal and splanchnic afferents.[4] Thermosensors were also found deep in the skeletomuscular system.[9,4]

1.1.d.ii Cutaneous Thermoreception

Cutaneous thermoreceptors respond to various stimuli, such as the skin temperature's rate and direction of change, and the size of the stimulated area.[9,42] Both types of sensors have dramatic responses to sudden temperature change, regardless of initial skin temperature.[42] There is a temperature range in which each type of cutaneous thermosensor responds maximally. The cold thermosensor's range of maximum activation is between 16° and 32°C, which has a gentle slope throughout most of its range.[9] On the other hand, the warm thermosensors have a peak maximum activation near 45°C. This curve has a large spike immediately around that temperature, while having quite low activity much above or below the peak temperature.[9] The relationship between warm and cold thermosensors and temperature in the range of 30°-47°C can be described as the warm sensors having a positive coefficient and cold sensors having a negative coefficient.[9] The activation curves of warm and cold sensors overlap, or intercept, somewhere between 35° and 40°C.[9] The intercept is approximately at normothermia, about 37°C, at which the different sensors have very similar activation intensities, as well as their activation curves having very similar approaching slopes. This lends itself to the theory of a thermoneutral zone, in which the neural activity of both of the opposing thermoreceptors are negated by equal intensity of lateral inhibition resulting in net zero neural activity.

The distribution of cold and warm thermosensors is uneven. The cold receptors are quite evenly distributed over the entire surface of the body, while that of the warm receptors is very variable.[9,4] There is a high density of warm sensors on the hands and face.[9,4,42] Hissa (1990) indicated there is a relatively large number of cold sensors in the skin as compared with warm sensors; while in the internal organs the situation is opposite.[8] There is also variability in depth of skin at which the cutaneous thermosensors are located. Some lie superficially in the skin, and others are located deep next to the subcutaneous fat layer.[43] The deeper thermosensors are not as easily stimulated as the more superficial ones. Different forms of heat energy, such as radiant heat, penetrate deeper through the skin, therefore, stimulating the deep thermosensors more intensely than other forms of heat energy.

1.1.d.iii Weighting and its effect on Thermoregulatory Effectors

The regulated temperature variable of the body has been generally termed the weighted mean body temperature. Since it is weighted, this suggests that there are areas throughout the body that have more dominant effects on the process of regulation.[44] Various studies have looked at thermosensitivities throughout the body and their subsequent effect on thermoeffectors. Simon, Pierau, and Taylor (1986) reviewed findings of different weighting of thermal stimuli applied to the hypothalamus, spinal cord, and facial skin; with spinal thermal inputs more influential in controlling autonomic than behavioral thermoregulation.[4] It is important to note that it is impossible to quantify

accurately what contribution different sensors have on the absolute input in a thermosensory region,[4] but qualitative assessment is possible. Many studies have been able to observe trends, as well as reproducible phenomena. Researchers found that they could suppress muscular shivering in guinea pigs by locally heating the pre-optic area.[8] They also recognized that information from other areas had significant effects, and suggested that muscular shivering were dependent on stimuli from the skin and the spinal cord.[8]

Simon et al (1986) suggested that thermal stability is achieved by the concerted action of all thermosensors in the body core and shell rather than by the hypothalamus and skin alone.[4] The weight of specific local thermal inputs have been qualitatively assessed. Hypothalamic studies showed dominating influences from its area. There are other areas that have very powerful influences on the thermoregulatory system. Simon et al (1986) have found that information from different skin regions is processed quite differently.[4,44,42] These researchers reviewed discoveries that there are two highly specialized regions of the skin, the trigeminal and the inguinal region.[4] It was observed that facial thermoreception seems to be particularly important in the perception of ambient temperatures and in the control of behavioral or discriminative processes.[4] The facial area, or blush area, is a specialized area in thermoreception and its input has been studied extensively. The trigeminal nerve, represented in the blush area, inputs to the trigeminal nucleus caudalis, and is mostly relayed to other structures except that there "is an accentuation of dynamic and static thermoresponsiveness. It has ... been shown that there is a close agreement between the responses of trigeminal nucleus caudalis neurons in the cat to static and changing facial skin temperature and the reports of human

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volunteers in a psychophysical study."[4] This suggests that the blush area is a highly

sensitive area to ambient temperatures.[44,42] Mekjavic and Eiken (1985) found that radiant

heat on the blush area of the body inhibited shivering in cooled subjects,[45] while Murphy

et al (1985) found similar results in squirrel monkeys.[46] Since the trigeminal nerve has a

powerful influence on shivering, they hypothesized that the mechanism inhibiting

shivering is that radiant heat on the innervated area causes a withdrawal of cold-receptor

stimulation.[45] Other studies using radiant heat were performed by Sharkey, Lipton,

Murphy and Giesecke (1987,) and Sharkey, Gulden, Lipton and Giesecke (1993,) who

studied hypothermic patients post-operatively to determine its effects. Both studies found

that VO_2 significantly decreased and that shivering was successfully inhibited.[47,48] As

seen in the above examples, an important observed phenomenon is the ability of some

areas to dominate the thermoregulatory mechanisms. "In cases of large and rapid skin

temperature changes, dynamic components in the thermosensory input from the skin may

temporarily dominate the activities of autonomic cold- and heat-defense effectors and,

especially, thermally induced operant behavior."[4] Although, usually the relative

importance or weight of thermal afferent inputs from the skin is minor."[4,44] "However, at

mean skin temperatures greater than 39ºC, a 40% reduction was observed in the gain of

the metabolic response to core temperature changes, which suggests that stimulation of

warm receptors in the skin exerted a strong inhibitory effect on the activation of metabolic

cold defense by the central cold-signal input."[4] This suggests that by giving a high

temperature stimulation on the skin, one could essentially inhibit cold effectors, such as

metabolic rate, shivering, and peripheral vasoconstriction.

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Wyss et al (1974) examined the effects of core and skin temperature on control of sweat rate and skin blood flow in heated, at rest human subjects.[49] The subjects were heated to just beyond the sweating threshold to a maximum skin temperature of 39ºC to try to assess the relative roles of core and skin temperatures. They found that skin temperature had an insignificant effect on sweating rate and the core temperature had a significantly greater effect (twenty times greater) on forearm blood flow than skin temperature.[49] Cheng at al (1995) found that mean skin and core temperatures are linearly related at the vasoconstriction and shivering thresholds in men.[14] The researchers found a linear relationship between core and mean skin temperatures for vasoconstriction and shivering, with a contribution ratio of approximately five to one (5:1), respectively.[14] These experiments exemplify the variation in relative contributions between core and skin temperatures, different areas of skin, and which thermoregulatory effector system is being effected, heat gain or loss.

1.2 Anaesthetics

There is a wide variety of drugs used in surgical, cardiac surgical and post-operative situations. These agents have diverse mechanisms of action, therapeutic indications, cardiovascular effects, pharmacokinetics, adverse reactions, and potential for toxicity; these are some characteristics that medical personnel must consider when caring for patients. Some briefly discussed drugs include general anaesthetics, analgesic narcotics, and muscle relaxants.

1.2.a General Anaesthesia

General anaesthesia has been described as being at the far end on a continuum of CNS depression. The continuum has three stages - starting from sedation, to hypnosis, and finally anaesthesia.[50] Palfai et al describe the anaesthetic state and administration in the following passage.

> Anaesthesia is a sleep state accompanied by muscle relaxation, loss of reflexes, and insensitivity to pain. The progressive stages of anaesthesia represent a progressive depression of the CNS produced by drugs. General anaesthetics are delivered as inhalants, gases and vaporized liquids, and as intravenous solutions. Inhaled forms of anaesthetics are immediately voided unchanged from the lungs, therefore, administration must be continued as long as the effect is desired.

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There are many types of anaesthetics available to clinicians today. Their choice of which

anaesthetic to use depends on the type of surgery, personal preferences, trends in the

profession, and pathophysiological factors specific to the patient.

1.2.a.i Inhalation Anaesthetics

Inhalation anaesthetics have a brief duration, and are used as general anaesthetics.

This means that there is no specificity in their actions and they depress the activity of all

neurons in the CNS. Inhalation anaesthetics such as isoflurane, halothane, enflurane, and

as an adjunctive anaesthetic agent, nitrous oxide, are the most commonly used

anaesthetics in cardiac surgeries today.

The effects of inhalation anaesthetics on cardiovascular performance is broad

ranging. Curling and Kaplan (1983) indicated that induction of general anaesthesia with

the volatile agents currently in use is known to depress whole-body oxygen consumption

(VO_2) as well as the VO_2 in individual tissues.[51] Myocardial tissue has been recognized

to have a disproportionately larger decrease in VO_2 than other organ tissues.[51] These

metabolic decreases are seen with halothane, enflurane and isoflurane. It has been

suggested that inhaled anaesthetics may act as general metabolic depressants. This finding

has important ramifications in many surgical situations, particularly cardiac surgery,

where a low tissue metabolic rate is ideal. Halothane, enflurane and isoflurane decreased

cardiac output, VO_2, mean arterial pressure, and heart rate from awake values.[51] Other

effects are on the systemic vascular resistance, which also decreases.[51,52] This effectively

means a decreased blood pressure, total peripheral resistance (TPR), afterload, and preload.

1.2.b Other Drugs Associated with Surgery and Post-Operative Care

1.2.b.i Narcotic Analgesics (opioids)

Narcotics are defined as drugs that cause sedation and drowsiness.[50] Narcotics are often used in surgery and postoperatively for deep sedation and analgesia. Narcotics are primarily painkillers and are the most effective class of drugs used for analgesia. The different forms are analogs of morphine, heroin, and cocaine. Some clinical narcotics used include morphine, codeine, meperidine, fentanyl, and sufentanil.

1.2.b.ii Vasodilators

Another important drug class is the vasodilators. Vasodilators such as sodium nitroprusside, and nitroglycerin are used to cause deliberate hypotension. This reduces afterload, preload, myocardial wall tension, and myocardial oxygen consumption.[53]

1.2.b.iii Muscle Relaxants

Muscle relaxants, also known as neuromuscular blocking agents, act to inhibit muscular activity. At higher doses these muscle relaxants essentially paralyze the patient. A popular muscle relaxants for cardiac surgery is pancuronium.[54]

1.3 Effects of Anaesthesia on Thermoregulation in Humans

The effects of anaesthetics on the human body are very diverse. Anaesthetics alter the homeostatic mechanisms in all the major systems, including the thermoregulatory system. The effects of anaesthetics on thermoregulation are drastic, but the majority of studies have focused on one particular observed phenomenon, a shift in the thermoneutral zone (TNZ.) The shift and expansion of the thermoneutral zone is commonly found in anaesthetic situations.

Isoflurane is an inhalational anaesthetic commonly used in cardiac surgery. The effect of isoflurane is very similar to that of others within this class. During anaesthesia, many of the thermoregulatory effectors are inhibited. The effectors that are not inhibited, but are altered, are the nonshivering thermogenesis and the vasomotor system.[3,18,55,56,57,58,59,60,61] The actual mechanisms of the anaesthetics are as yet, unknown. Sessler et al (1988) suggested that although suppression of thermoregulatory mechanisms by anaesthetics is generally assumed, the extent to which thermoregulation is active during general anaesthesia is not known.[58] This states that due to the paralyzing

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and general suppression effect of general anaesthetics, it is unknown if thermoregulatory effectors are actually trying to respond, but are simply not expressed. The responses, such as shivering, sweating, and behavioral mechanisms, are all inhibited throughout anaesthesia. Nonshivering thermogenesis continues to function, but the general metabolic rate is depressed from the anaesthetic.[62] The last, and most commonly reported occurrence is that of decreased peripheral vasomotor tone.[52] The peripheral vasoconstriction threshold has been noted to shift left, and expand in width.[3,18,55,57,58,59,60,61]

There are many studies detailing the shift of the thermoneutral zone (TNZ) to about 34.4°C when under the effects of isoflurane, enflurane, halothane, and nitrous oxide -fentanyl anaesthesia.[3,18,55,56,57,58,59,63,60,61] This is a threshold shift of about 2.5°C to the left, while the TNZ essentially expands with it. This means that the vasoconstriction threshold is lower. Accompanying the decrease in the vasoconstriction threshold is an increase in the vasodilation threshold. The degree of shift to the right of the sweating threshold is not as great, but it does occur. With both the vasoconstriction threshold's significant decrease, and the vasodilation threshold's increase, the vasomotor TNZ grows in width. The width grows from approximately 0.5° at normal thermoregulation, to approximately 4°C when under anaesthesia.[3,18,55,57,58,59,60,61,64,58]

Researchers believe that the effects from isoflurane on vasoconstriction is centrally mediated, since once vasoconstriction is triggered, the response is normal.[3,18,55] This means that the actual thermoregulatory response of vasoconstriction may not be affected, while the central nervous system regulating this response has been affected.

The general effect of shifting the vasomotor TNZ left approximately 2.5ºC is seen in all of the mentioned anaesthetics, isoflurane, enflurane, halothane, and nitrous oxide - fentanyl anaesthesia.[3,18,55,57,58,59,63,60,61,64] The effect of the inhibition of tonic thermoregulatory vasoconstriction is what has been described as redistribution hypothermia.[3,18,55] Redistribution hypothermia is when the warmer blood in the core redistributes to the periphery since the thermoregulatory mechanisms are not effective. The body rapidly loses heat energy due to this fluid shift which increases the peripheral blood volume. The cooler blood in the periphery will return to the core and result in hypothermia.

Thermoregulatory shivering is inhibited during surgery by neuromuscular block. There appears to be another form of 'shivering.' A common occurrence for postoperative hypothermic patients, although this phenomenon has been observed in many patients whose core temperatures have remained fairly stable, is that of shivering. The term attributed to it, *postoperative shivering*, is not very accurate for the actual muscle activity that occurs. One simple reason that the term postoperative shivering is misleading is that it has actually been observed in animals *during* surgery.[65] A term that is more accurate in its description is postanaesthetic muscle activity, or tremor. Postoperative muscle activity has been observed for years, but only recently have researchers defined that what is observed is not always representative of thermoregulatory shivering. Normal thermoregulatory shivering is oscillatory, and its EMG activity occurs in a frequency range of 5-7 Hz.[65] This type of shivering has been observed, but along with other more prominent types during postanaesthetic recovery. "The EMG characteristics of post-anaesthetic tremor generally resembles those produced by pathological ankle clonus in

patients with spinal cord transections (which cannot be a centrally modulated thermoregulatory response)."[18] Sessler et al's (1988) results suggest that spontaneous postanaesthetic tremor is caused by spinal reflex hyperactivity that results when descending cortical control is inhibited by residual anaesthetic, rather than by a thermoregulatory mechanism.[18] The EMG characteristics found in postanaesthetic muscular activity include tonic bursts of 5-7 Hz, clonic bursts of 5-7 Hz, tonic bursts of 5-15 Hz, and clonic bursts of 5-15 Hz.[18] These characteristics are quite different from normal thermoregulatory shivering. Also, the finding concerning lack of descending cortical control of shivering suggests that thermoregulatory shivering may play only a small role, if any, during initial postanaesthetic recovery. As the anaesthetic concentration decreases, the TNZ returns to its normal range, the body senses the cold, and thermoeffectors such as vasoconstriction and shivering are initiated.[18] The anaesthetic wears off slowly. This means that the disinhibition of shivering will not occur suddenly or homologously throughout the body. As the anaesthetic wears off, shivering becomes more controlled and effective. The initial aspects of the return of thermoregulatory shivering is unhomologous since different muscles are still affected by the anaesthetic while others have regained control. Sessler et al (1988) have suggested three stages of recovery when referring to muscle activity, or 'shivering'.[18] These include 1) early recovery ... when little muscular activity occurs; 2) middle recovery ... during which thermoregulatory responses to cold are inhibited but spinal reflex activation causes a clonic spontaneous tremor (spontaneous EMG clonus and tonic EMG activity with underlying clonus); 3) late recovery ... during which thermoregulatory responses are no longer inhibited and spinal reflexes are no longer activated. If these patients remain

hypothermic, they may demonstrate normal shivering.[18] Sessler et al (1988) also found examples of when shivering intensities did not respond in proportion to body temperatures.[18] They observed severe shivering when near normothermia, as well as no shivering epochs at subnormal temperatures.[18,66] This further supports the notion that the observed muscular activity in immediate postanaesthetic recovery may not be thermoregulatory in nature. These studies have shown that anaesthetics have various significant effects on thermoregulation.

1.4 Surgery and Cardiac Surgery

Patients who undergo surgery and cardiac surgery passively and actively lose heat energy and become hypothermic. The patient passively loses heat through different heat transfer mechanisms; conduction, convection, evaporation, and radiation. The operating room is cool in temperature for the benefit of the surgical team. The patient loses heat to the cold operating table by conduction, the ambient air by convection and to the room by radiation. When the patient's thoracic cavity is opened for surgery, heat loss by evaporation occurs. The anaesthetic inhibits the body's ability for thermoregulation, thereby allowing it to lose heat through redistribution of fluid without its effectors trying to compensate for this heat loss.[3,67] Since the blood vessels are not constricting to prevent heat loss,[52] there is a greater blood volume in the periphery, therefore, a larger surface area over which heat loss occurs. This essentially results in heat transfer from the core to the periphery to the environment.[3] As heat loss is greater than metabolic heat production,

hypothermia occurs. Some non-cardiac procedures during which patients become moderately hypothermic include radical cystectomies, thoracic aneurysms, and abdominal aortic aneurysms.

Another major cause of hypothermia in cardiac surgeries is from active cooling of the heart and the body in general. A cold cardioplegic solution is diverted into the coronary arteries which produces immediate cardiac arrest, minimizes its metabolic rate, supplies substrates for energy metabolism, maintains an appropriate pH, as well as other functions to protect the cardiac tissue.[68,69,70,71] This procedure combined with cold cardiopulmonary bypass promotes hypothermia in the cardiac patient. Cold cardiopulmonary bypass (CPB) cools and maintains the blood at 20-30°C when it bypasses the heart and lungs and courses through the oxygenator- heat exchanger to the rest of the body.[72,68,62,71,22]

When the patient is brought to the post-anaesthetic care unit, he or she is emerging from the effects of the anaesthetic and can be in various stages of hypothermia, depending on the length of the surgical procedure, the size of the surgical incision, the temperature and duration of cold cardiopulmonary bypass, temperature of the cardioplegic solution, the temperature and volume of the injected fluids and blood components, the ambient temperature in the operating room and the post-anaesthetic recovery room, the extent of rewarming by the warm cardiopulmonary bypass, and individual etiologies such as body surface area-to-mass ratio, and insulative layer (subcutaneous fat) thickness. Since the patient's effectors gradually become uninhibited as the anaesthetic is metabolized, and the thermoregulatory system senses the body is cold, the thermogenic response of shivering

and vasoconstriction is initiated. The degree of hypothermia is important for the patient's recovery since hypothermia tends to prolong the duration of the actions of some drugs.[50,][54,3] An example of this is with the muscle relaxant, pancuronium. Studies have found that hypothermia increases the duration of induced neuromuscular blockade.[54] This occurs because of the hypothermic effect of delaying the urinary and biliary excretion, and the decreased metabolism of active pancuronium to inactive metabolites.[54] Studies have also found that the mechanical recovery of the neuromuscular blockade is prolonged.[54]

The recovery from anaesthesia occurs on a continuum, from fully anesthetized, to the patient becoming conscious. If one follows a patient's postanaesthetic recovery, a profile such as the following may be seen. It is important to remember that no one profile could cover all the possible variable situations. The following profile suggests a fairly general, and common cardiac surgical recovery.

> After cardiac surgery is finished, the patient is rewarmed on cardiopulmonary bypass, which generally warms the core temperature up to 38ºC. "The extremities, because of cooling during bypass, are slower to warm and remain vasoconstricted, less well perfused, and hypothermic."[73] When the patient is taken off the bypass, there is a large gradient, and the core acts as a 'heat sink.'[73] The cool blood from the extremities and other body compartments will quickly cool the warm core.[72] When the body reaches equilibrium, the core will usually be about 34-35ºC. This drop is referred to as 'afterdrop.'[72] On arrival in the ICU, patients will generally be hypothermic and peripherally vasoconstricted.[73] Since there is an increased arterial tone and reduced venous capacitance, there is an increased left ventricular afterload and an increased preload, respectively. The increased muscular activity from postanaesthetic tremor and thermoregulatory shivering causes an increased myocardial work, and

therefore, increased oxygen demand.[73,22,21,23,66] "Treatment is thus directed at reduction of high systemic vascular resistance and decreasing peripheral oxygen requirements."[73] Systemic vasodilators are often given to reduce high systemic vascular resistance. An example of one of these is nitroglycerin. Active rewarming also helps to reduce the hypothermia and the shivering. Hemodynamic changes occurring during the rewarming period are generally due to vasodilation.[73] When body temperature approaches normothermia, and blood pressure characteristics are stable, the patient is considered to be in a much safer situation.[73]

This profile is general, with no emphasis on the hundreds of variables that could change an uncomplicated recovery into an unstable one, such as with hemodynamic instability. The cardiac patient is very sensitive postoperatively, and must be treated as such. The above profile is very similar to the non-cardiac surgeries mentioned above with exception of the warm cardiopulmonary bypass and the resulting afterdrop. These patients arrive in post-operative care in very similar conditions, particularly the degree of hypothermia.

1.5 Dangers to the Post-operative Patient and Rewarming Techniques

The hypothermic post-operative patient is extremely vulnerable within the first few hours after leaving surgery. Maintaining physiological stability while assisting recovery from the diseased state is the caregiver's primary goal. The condition of hypothermia and its related shivering is dangerous. Effective rewarming is required to decrease the profound negative effects hypothermia has on all the systems of the body. Howell et al (1992) suggest that all body systems, including an already compromised cardiovascular system, are stressed by the lower body temperatures; indeed, the literature emphasizes a diminution in myocardial and cerebral functions, the development of respiratory acidosis, an impairment of hematologic and immunologic functions, and the onset of cold diuresis.[74,75,76] Specifically, hypothermia causes platelet dysfunction,[77] therefore increasing coagulation time, as well as tending to prolong the duration of some drugs' actions.[50,54,3,78]

A related and very important concern to caregivers is the occurrence of post-operative shivering. The incidence of post-operative shivering has been reported to range from 5 to 65%.[79] Some of the dangers of shivering are as follows: an increase in oxygen consumption of up to five hundred percent which could cause a hypoxic episode in the tissues of patients who are incapable of maintaining the high oxygen demand,[18,48,74,75,80] an increased heart rate and mean arterial pressure,[74,80,18,75] increased respiratory demand and carbon dioxide production,[74,80,18,48,81] decreased blood oxygen saturation,[80,18,48,81] depletion of glycogen stores,[80,18] metabolic acidosis from increased levels of lactic acid,

carbon dioxide and other acids[80,74,18,76] wound dehiscence,[18] dental damage,[18] and disruption of delicate surgical repairs.[18]

Some of the more common current techniques and devices in rewarming post-operative patients are either passive or active. Active rewarming can be defined as methods that actively donate heat energy to the patient. Passive techniques include warmed blankets and head coverings, while active techniques include circulating warm water mattresses, inhalative rewarming, convective rewarming, and radiant heat. The current methods used to help rewarm post-operative patients and protect them from the dangers of hypothermia and shivering each have their are benefits, but are still generally ineffective[82,80,23]

Head coverings and blankets, even warmed blankets, are passive in that they simply reduce the rate of heat loss by supplying some insulation. Howell at al (1992) studied the benefits of two different kinds of head coverings on post-operative cardiac patients.[74] They found no significant difference in the length of time for patients to reach normothermia between the control group without head coverings, and either of the groups with head coverings.[74]

Experiments using convective warm air rewarming techniques have had some promising results. A product that is currently on the market that uses convective rewarming principles is the Bair Hugger®. The Bair Hugger® has been marketed to be used intraoperatively as a preventative method for heat loss, as well as for postoperative rewarming in recovery rooms. There have been many studies on this product finding similar results. Ereth, Lennon, and Sessler (1992) showed that with the Bair Hugger®,

convective rewarming techniques are effective in significantly increasing mean skin temperature, but not in significantly increasing core temperature in post-operative patients.[83] These experimenters also found that all the patients in the study were significantly vasoconstricted. Ereth, Lennon, and Sessler (1992) proposed that the thermoregulatory vasoconstriction (in post-operative patients) limits heat transfer from peripheral to central thermal compartments and impedes skin surface warming of the body core.[83]

Sessler and Moayeri (1990) studied four postoperative warming devices consisting of two different types of radiant heaters, a water blanket, and different settings on the Bair Hugger, measuring heat flux in five healthy, unanesthetized volunteers.[84] Although the water blanket transferred considerable heat, the Bair Hugger on 'high' transferred the most heat to the subjects as measured by heat flux.[84] This is intuitive when considering the Bair Hugger uses convective rewarming which covers a large amount of the anterior aspect of the body, while the warm water blanket uses conductive heat transfer. For the water blanket to be effective, it would need to be in close contact with the body, essentially like a second skin. A limitation to radiant heat is that the natural curved areas of the body remain unexposed. The radiant heaters in the studies were positioned directly over the subjects, thereby reducing the irradiated body surface area. For the radiant heaters to be effective, they need to be angled to the sides of the body to increase the incidence of irradiation.

Mort, Rintel and Altman (1990) evaluated the Bair Hugger with hypothermic post-operative CABG patients measuring shivering, core and skin temperatures, and the peripheral- to- central temperature difference.[82] They found a significant reduction in

shivering, measured by the visual observation shivering scale (VOSS) and verified with EMG recordings from pectoral, biceps, and quadricep muscle groups.[82] It is curious that the researchers did not record EMG from a muscle that is initiated early in shivering, such as the masseter. The pectorals are initiated late in thermoregulatory shivering. They found a significant increase in skin temperatures, but no difference in core temperatures and the peripheral- to- central temperature difference between groups.[82]

Studies by Sharkey, Lipton, Murphy and Giesecke (1987,) and Sharkey, Gulden, Lipton and Giesecke (1993,) used radiant heat to determine its effects on shivering in hypothermic post-operative patients.[48,47] Both studies found that VO_2 significantly decreased and that shivering was significantly inhibited.[48,47] They also found that patients requested the radiant heat be turned back on to increase their comfort when the lamps were switched off. Radiant heat has been shown to inhibit shivering in non-clinical[45,46] and clinical situations.[47,48] None of these experimenters measured heat gain or rewarming capabilities of radiant heat in their studies.

Joachimsson, Nystrom and Tyden (1987) studied heat balance after cardiac surgery with hypothermic cardiopulmonary bypass (CPB).[85] Measuring shivering, core temperatures, mean skin and fingertip skin temperatures, they examined groups consisting of no active rewarming (control), heated and humidified inspired gases (inhalational rewarming), radiant heat over the whole body less the head, and the combination of this radiant heat with heated inspired gases.[85] Joachimsson, Nystrom and Tyden (1987) found that the inspired gases group gave little improvement, while the radiant heat groups significantly increased the measured temperatures, which were restored to normal earlier than those in the controls.[85] Also, with this radiant heat postoperative shivering was

almost abolished.[85] In a later study these same researchers, Joachimsson, Nystrom and

Tyden (1987), compared a control group against a radiant heat with heated gases group

while including hypertension and peripheral vasoconstriction in their measures.[86] They

found in the radiant heat group the postoperative rewarming was accomplished earlier,

shivering, oxygen uptake, CO_2 production and ventilation volumes were significantly

reduced, and postoperative hypertension and vasoconstriction were greatly decreased.[86]

Unfortunately, the researchers did not specify if their measure for peripheral

vasoconstriction was protected from local heating effects from the radiant heater. If not, it

is unsure how accurate their results were for measuring peripheral vasoconstriction.

To summarize, there have been a significant number of studies investigating

various methods of post-operative rewarming but with varying results concerning their

efficacy. It has been commonly found that blankets and head coverings have no

significant value in post-operative rewarming or with the inhibition of shivering. Also,

inhalational rewarming does not have a significant effect in rewarming or the inhibition of

shivering. It has been suggested that warmed inspired gases is ineffective due to the fact

that the specific heat of the gas (air and oxygen) is extremely low. The circulating warm

water blanket has proven that it is effective in raising the mean skin temperature, but has

'hot spots' which may present a danger of burning. It did not significantly inhibit post-

operative shivering. The circulating warm water blanket also was not significant in raising

the core temperature, which like the convective rewarmers, is probably due to

thermoregulatory peripheral vasoconstriction. The convective rewarmers, such as the

Bair Hugger, have had promising results. The convective rewarmers have had success in

significantly increasing the mean skin temperature, but were not significant in increasing

the core temperature. There have been mixed results with inhibition of shivering, therefore, the literature is inconclusive. Also, convective rewarmers have not significantly decreased the thermoregulatory peripheral vasoconstriction. The studies using radiant heaters seem to be inconsistent. There has been a wide range of heat lamp output used as well as the area of the body irradiated. This makes it difficult to assess the different studies using radiant heat. Some studies found that radiant heat significantly increased the mean skin temperature, but not as efficiently as convective heat. Also, some studies found that radiant heat significantly increased core temperature, while others did not. The most consistent finding in the literature is that radiant heat significantly decreases shivering. There are studies that suggest that radiant heat also significantly reduces thermoregulatory peripheral vasoconstriction, although the number of studies researching this hypothesis is limited. The studies utilizing radiant heat are sometimes inconsistent in their methods, which has lead to inconclusive results. It seems that none of the post-operative rewarming devices or techniques are successful in significantly increasing core temperature, significantly decreasing the occurrence of shivering, and significantly decreasing thermoregulatory peripheral vasoconstriction.

2.0 RATIONALE

Researchers have shown that the current methods used to help rewarm post-operative patients and protect them from the potential dangers of shivering are inadequate.[18,82,80,23] A possible solution is to maximize the benefits of convective rewarming and radiant heat by combining them. Ereth, Lennon, and Sessler (1992) showed that convective rewarming techniques are effective in increasing mean skin temperature in post-operatively rewarming patients.[83] Radiant heat has been shown to inhibit shivering [45,47,48,86] and has been suggested that it may also inhibit peripheral vasoconstriction.[86] The convective rewarming technique may be maximized by increasing the peripheral blood flow if it is used in combination with radiant heat. The objective of this experiment was to see if applying radiant heat on the blush area inhibits shivering, as well as inhibit peripheral vasoconstriction, thereby increasing the effectiveness of the convective rewarming technique.

Triggering heat sensors in the blush area to vasodilate the peripheral capillary beds of the patient increases the effectiveness of convective rewarming. By increasing the volume of blood to the periphery, there is an increase in the heat capacitance of venous return to the core. The heat added to the body by the convective rewarming device and the radiant heater is more efficiently absorbed. The hypothesis was based on the findings that one of the cold thermoregulatory effectors, shivering, is inhibited by the application of radiant heat to the blush area.[45,46,47,48,86] Since both shivering and vasoconstriction are thermoregulatory responses to cold, vasoconstriction is also inhibited by the application of radiant heat.[86]

3.0 HYPOTHESES

The hypotheses of this study were that applying radiant heat on the blush area of post-operative cardiac and non-cardiac surgical patients would:

- decrease the number and severity of shivering epochs,

- inhibit peripheral vasoconstriction,

- decrease the rewarming time required to reach the core temperature of $37^{\circ}C$,

- increase the core temperature rewarming rate,

- increase the mean skin temperature.

4.0 METHODS

4.1 Subjects

Subjects were selected from patients scheduled for long duration elective surgeries and cardiac surgeries: for example, radical cystectomies, thoracic aneurysms, abdominal aortic aneurysms, cardiac valve replacements and coronary artery bypass grafts (CABG). Subjects were approached to participate in the study prior to surgery at Vancouver Hospital. Each patient was informed of the procedures, risks and benefits involved by the investigators. No subjects were identified in any reports of the completed study. Subjects who participated were required to sign a letter of consent according to the ethics guidelines of Vancouver Hospital, the University of British Columbia, and Simon Fraser University. The study was conducted in the Special Post-Anaesthetic Recovery room (SPAR) and the Cardiac Surgical Intensive Care Unit (CSICU) at Vancouver Hospital. Patients admitted into the study had pulmonary artery or esophageal temperatures within the range of 33.0- 35.5°C, upon admission to post-operative recovery. Patients were screened and eliminated for drug use, communicable conditions such as hepatitis or HIV, recent fever, infection, malignant hyperthermia, weight outside of the range of $\pm 25\%$ ideal body weight, liver disease, metabolic disorders, thyroid disease, and age greater than eighty-five years old. All relevant medical conditions or drugs introduced to the patients, were noted.

4.2 Procedures

There were two experimental groups:

Group #1: convective rewarming technique alone (BH) (n=16).

Group #2: combination of convective rewarming technique and radiant heat on the blush area (RH) (n=17).

Group #1 was the standard to which group #2 was compared. The term 'control group' was avoided due to the inherent lack of control involved in clinical research. The two experimental groups proceeded identically with exception of the active rewarming by radiant heat in group #2. The assignment of patients to the trials were randomized. Shivering was evaluated by the VOSS (Maximum Visual Observation Shivering Scale)[80, 23] and electromyography (EMG) activity. Core and skin temperatures, and peripheral vasoconstriction were also measured.

Subjects were manually monitored immediately upon arrival in post-operative care. Electronic monitoring and rewarming intervention began shortly thereafter. The electronic hook-up and rewarming intervention followed the post-operative nursing team's primary care. The manual recording was therefore reported for a number of minutes before the electronic recording was hooked-up and initiated. These manual recordings were indexed as negative time until the electronic monitoring system was initiated. Patients arriving from surgery were monitored continuously until their core temperature (by pulmonary artery or esophageal temperature sites) reached 37.0°C and was maintained for thirty minutes.

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Real time electronic monitoring of EMG data, skin temperature and peripheral vasoconstriction was achieved by connecting sensors to a National Instruments DAQCard-700® A-D card converter which fed into an AST Ascentia 810N® laptop computer. The data were treated and stored with National Instruments Labview® software written specifically for this study. Analog to digital data sampling occurred at 1 KHz for the first 25 of every 30 seconds. The final five seconds were required for the treatment and storage of data. The EMG data were treated on-line by calculating the root mean square over 1 second intervals. The skin temperature and peripheral vasoconstriction digital data were averaged over 30 second intervals.[49] The experimenter manually recorded the subjective VOSS measure, and the core temperature measures from the Marquette heart monitor, or the esophageal temperature unit, every 1 minute for the first hour, and every 5 minutes after that on the data collection sheets. Ambient temperature was periodically recorded. All drug injections were recorded with regard to type and dosage.

The experiment was approved by both the University of British Columbia Medical Ethics Committee and the Simon Fraser University Ethics Committee. This study was in part supported by a grant from the Vancouver Foundation.

All equipment used in this research was safety tested and approved by the department of Biomedical Engineering at Vancouver Hospital.

4.2.a Rewarming Devices

The convective rewarming device used in both groups was Augustine Medical Incorporated's Bair Hugger®. The Bair Hugger®'s output level was set on 'high'. The Bair Hugger® was used in accordance with its specified directions. The Bair Hugger® blanket covered the anterior aspect of the body to just above the patient's nipple-line. A cotton blanket covered the Bair Hugger® blanket as directed in its user manual. The patient's head and neck remained exposed. The patient's arms were not under the Bair Hugger,® to avoid any local heating effects[87,88] in the forearm. Local heating of the tissues would contaminate the forearm-fingertip measure used as an indicator of peripheral vasomotor tone, which is described below. The forearm with the thermistors measuring peripheral vasoconstriction were shielded from any external heat sources.

An overhead radiant heater constructed by Innovative Industries Incorporated® was used for experimental group #2. The heating unit consisted of two banks of infra-red heatlamps lying perpendicular to the body. The heating elements were housed in a casing with a curved reflector behind them. These cases were mounted on a swivel to have the ability to maximize the angle of incidence of heat energy delivered to the patient. The radiant heater was located over the patient's blush area while lying supine. The distance between the radiant heater's elements and the top of the patient's ears was measured approximately 75 centimeters. This was a convenient distance between patient and heater to allow medical personnel access to the patient, while maintaining the ability to sustain the desired skin temperature. There were two temperature thermistors secured bilaterally

on the patient's forehead for proportional temperature autofeedback control to the radiant heater. Temperature output of the lamps were controlled by these thermistors to maintain the skin temperature at a maximum 41°C. This temperature was chosen for a number of reasons. A blush area skin temperature between 39-43°C is common to the literature in similar studies.[4,45,47,48,85,86] Skin temperatures greater than 43°C may burn.[47] Also, since the warm thermosensors' maximal activation is in this temperature range, peaking around 45°C, (see 1.1.d.ii Cutaneous Thermoreception), a skin temperature of 41°C is close to optimal activation while maintaining a degree of safety.

4.2.b Data Collection

Shivering was evaluated by the VOSS (maximum Visual Observation Shivering Scale)[80,23] and root mean square electromyography (EMG) activity (discussed below); while core and skin temperatures (as defined below), and peripheral vasoconstriction were also measured.

4.2.b.i Shivering

In the VOSS measure, the patients were observed by the experimenters every 1 minute for the first hour, and every five minutes thereafter. Patient's shivering was rated according to the scale in Table 1 [80,23] and documented manually on a data sheet. The observation was carried out exclusively by the primary experimenter for uniformity throughout the research.

Table 1. Documentation of Shivering: VOSS

0 = no visible or palpable shivering	
1 = palpable mandible vibration or ECG artifact	
2 = visible fasciculations of the head or neck	
3 = visible fasciculations of pectorals or trunk	
4 = generalized shaking of the entire body and teeth chattering	

The primary site for recording EMG activity was the muscle belly of the masseter. The sternocleidomastoid muscle in the neck was used as a secondary site, and lastly the trapezius, when there were difficulties at the primary site. Possible difficulties of the area included beards and local tissue damage. Shielded bipolar surface electrodes were spaced approximately 1- 1.5 cm apart placed in parallel over the muscle belly. The electromyographic signal was then amplified by the microelectrode preamplifier with a high-pass filter at 3 Hz and a low-pass filter at 1 KHz. The signal then ran through the Hum Bug (Quest Scientific®), to remove narrow-band noise, such as 50 and 60 Hz noise. The electromyographic signal then ran to an oscilloscope, where the signal was observed in real time, as well as to the A-D board, where the signal was digitized and recorded by the computer, as described above. The EMG data was treated on-line by root mean square averaging (RMS) over 1 second intervals.[89] Using root mean square treated data was appropriate since the study was attempting to look at the power of the shivering epochs and not individual spikes in the electromyographic record.[90]

4.2.b.ii Temperature

In the majority of cardiac patients the core temperature was monitored with a thermistor in the tip of a Swan catheter probe inserted into the patient's pulmonary artery. The Swan catheter was inserted immediately prior to surgery by the anaesthetist, where its digital output was displayed on the overhead Marquette monitor. The core temperature probes were labelled according to the subjects' code and then calibrated once removed from the subject. The patients without Swan catheters had their core temperatures measured at the esophageal temperature site with thermistors. Esophageal thermistors were also calibrated post-extubation from the subject.

All skin temperatures were taken using YSI 700 series thermistors, with an accuracy of $\pm 0.1^\circ$C. The thermistors were attached tightly to the skin with reflective tape.

The weighted mean skin temperature was derived from 4 sites located at the shoulder, the chest, the anterior thigh, and the lateral calf. This calculation was weighted according to the Ramanathan formula (Mean Skin Temperature = (.30)shoulder + (.30)chest + (.20)thigh + (.20)calf).[91,62,92]

The skin temperature gradient between the forearm and the index finger was measured. This temperature gradient was used as an indicator of active peripheral vasoconstriction. The measure compares the temperature gradient between the skin of the forearm and the fingertip, which has anatomical and physiological differences. The fingertip has arteriovenous anastomoses, which appear to have a role as heat loss thermoregulatory effectors, while the forearm does not. Since blood flow is the chief

internal factor in determining skin temperature[92], a modification in vasomotor tone

changes the skin temperature.[92] Significant vasoconstriction was defined as skin

temperature gradients greater than or equal to 4ºC, as defined by Sessler.[59,60,63,58,93] If the

temperature gradient is less than 4 ºC, the patient is peripherally vasodilated.[59,60,63,58,93]

The thermistors were attached to the patient's left arm, and positioned at the height of the

heart. The forearm thermistor was placed on the radial side of the arm midway between

the elbow and the wrist; the fingertip thermistor was positioned on the tip of the index

finger opposite the nailbed. The forearm was shielded from the heating sources so that

both temperature sites had similar conditions and did not experience local heating

effects.[87,88] The arm was free of IV catheters and blood pressure cuffs.

Anthropometric measures such as height and weight were recorded by the

preoperative nursing staff at Vancouver Hospital. The body surface area was calculated

from the height and weight using the formula (Body Surface Area = $\text{height}^{0.725}$ *

$\text{weight}^{0.425}$ * 0.007184).[94,72,95]

5.0 DATA PROCESSING AND ANALYSIS

The variables (1) time required to reach normothermia (37°C), and (2) rate of core temperature rewarming (°C/min), were analyzed from the nadir of each subject's core temperature. When afterdrop occurred, the nadir closest to the initiation of rewarming was chosen as the onset to best reflect the rewarming period from the coldest core temperature. The BH and RH groups were compared for differences in (1) CSICU entry core temperature, and (2) lowest core temperature, to ensure similar groups. The rate of core temperature rewarming compared the slope between the BH and RH groups.

The mean skin temperature and skin forearm-fingertip temperature gradient measures were analyzed at specific time intervals over the postoperative rewarming time course. Between group differences were analyzed at minute 1 to ensure that the groups started from similar temperature profiles.

Shivering was measured by the VOSS and analyzed by averaging each subject's VOSS score over the postoperative rewarming time course. The average VOSS shivering scores were transformed by square root to improve statistical properties. Subject VOSS scores were also analyzed between BH and RH groups by averaging the number of shivering observations at each particular VOSS score (1, 2, 3, 4).

The EMG data collected were not used in the analysis. It became clear during the data acquisition and analysis that the EMG data were not representative of shivering in postoperative patients. During many occurrences of shivering there was little to no EMG activity on the oscilloscope or on the digital RMS EMG record.

6.0 STATISTICAL ANALYSIS

All variables were compared between the convective heat rewarming group (BH) and the radiant heat rewarming group (RH). For each of the variables, mean skin temperature and forearm minus fingertip temperature gradient, the post-operative time course was analyzed by mixed design univariate analysis of variance (ANOVA). The independent variables include the two study groups (BH vs. RH) on one factor, with each dependent variable (Mean Skin Temperature and Forearm- Fingertip Temperature Gradient) measured at three distinct time intervals. The time intervals for mean skin temperature were minutes 26, 52, and 72, while the temperature gradient time intervals were at minutes 25, 50, and 70. Tukey's post-hoc tests were conducted. The tests established the 5% level of significance.

The variables (1) time required to reach 37°C, (2) rate of core temperature rewarming, and (3) average VOSS shivering score, were compared between BH and RH groups with two-tailed unpaired t-tests. The tests established the 5% level of significance. A difference between groups of 25% or greater was considered to be clinically significant.

7.0 RESULTS

All the patients involved in the study were unconscious when transferred from the operating room (OR) to the Cardiac Surgical Intensive Care Unit (CSICU) or post-anaesthetic recovery unit (PAR). While the nursing staff completed their initial assessment and monitoring hook-up, the experimenter proceeded with the study's monitoring and hook-up. The assignment of patients to subject groups was random, therefore, no differences between the groups with regard to demographic factors were expected. Analysis of many demographic factors, including body composition, surgical times and temperatures, core temperature upon entry to the CSICU, as well as inotropes, analgesics, and sedatives used, found there were no differences between the groups, as seen in Table 2. Also, there were similar ratios of men: women between the groups, and the surgical procedures were similar. (BH= 13 coronary artery bypass grafts (CABG X 3-6), 3 aortic aneurysms (thoracic and / or abdominal); while RH= 12 coronary artery bypass grafts (CABG X 3-6), 4 aortic aneurysms (thoracic and / or abdominal), 1 radical cystectomy.) All results are reported as the mean ± standard error of the mean (S.E.) response of the subject group.

Table 2. Demographics

	BH (n=16)	RH (n=17)
Age (yrs)	68.4 + 2.7	64.6 + 2.0
Height (cm)	167.7 + 2.3	169.8 + 3.3
Weight (kg)	77.6 + 3.3	76.8 + 3.0
BSA (m2)	1.87 + 0.1	1.88 + 0.1
Sex	14 M : 2 F	14 M : 3 F
Surgical Procedures	13 CABG: 3 aneurysm	12 CABG: 4 aneurysm: 1 rad cyst
Surgical Time (min)	285.9 + 23.4	302.7 + 25.6
Cold CPB (min)	162.6 + 18.9	150.6 + 15.0
OR coldest Myocardial Temp (ºC)	12.3 + 0.6	14.1 + 1.5
CSICU ambient temp (ºC)	22.4 + 0.2	22.0 + 0.3
Patient's entry to CSICU (Tcore ºC)	35.0 + 0.1	34.8 + 0.2
Patient's coldest temp OR (Tcore ºC)	31.2 + 0.6	30.7 + 0.7
Nitroglycerin (ug/kg/min)	0.91 + 0.1	0.88 + 0.3
Dopamine (ug/kg/min)	2.2 + 0.3	2.6 + 1.2
Morphine (mg IV)	6.4 + 1.1	5.7 + 0.8
Versed (mg IV)	3.9 + 0.7	3.0 + 0.4
Epinephrine (ug/min)	2.5 + 0.3	2.2 + 0.6

7.1 Core Temperature

Time for T_{core} to reach normothermia (T_{core}, minutes). Firstly, the groups were compared with regard to the lowest core temperature achieved in the O.R. (BH= 31.2 $\pm$ 0.6ºC vs. RH= 30.7 $\pm$ 0.7ºC), as well as their initial T_{core} upon admission to the CSICU (BH= 35.0 $\pm$ 0.1ºC vs. RH= 34.8 $\pm$ 0.2ºC), and no difference was found. The time required for core temperature to reach normothermia (37ºC) was found to be significantly different (two tailed p=0.0016) between groups. In one case, the patient's normothermic

temperature was found to be 36.8ºC, not 37ºC. In this case the patient reached 36.8ºC at the normal rate and reached a plateau for a period of time. Since heat was continuously added according to protocol, the patient did reach 37ºC after a very long period. During the plateau at T_{core} 36.8ºC, the patient started sweating. When the rewarming ceased at 37ºC, the patient's core temperature rapidly dropped back down to 36.8ºC while continuing to sweat. This seemed to be the patient's normal core temperature, as (s)he soon stopped sweating after reaching this plateau. This case was unique within the study.

The convective heat group took considerably longer (BH= 146.9 ± 11.0 min) to rewarm than the radiant heat group (RH= 103.3 ± 7.7 min). The difference in rewarming times is statistically significant (p=0.0016) as well as clinically significant (>25% improvement), as demonstrated in Figures 2 and 3. Figure 2 is a graph which includes all of the patient rewarming profiles. The BH group is represented by triangles(▲), and the RH group is represented by circles(O). Figure 2 may be somewhat confusing in distinguishing between individual subjects, but its primary purpose is to present the core temperature rewarming profiles for all of the subjects in the study. Figure 3 is a compilation of four randomly chosen T_{core} rewarming profiles from both BH and RH groups. The representation of study groups seen in Figure 3 shows a more discrete comparison of rewarming profiles than Figure 2.

Figure 2. All Subjects' Core Temperature Rewarming Profiles, BH and RH

BH vs. RH Core Temperature Rewarming Profiles
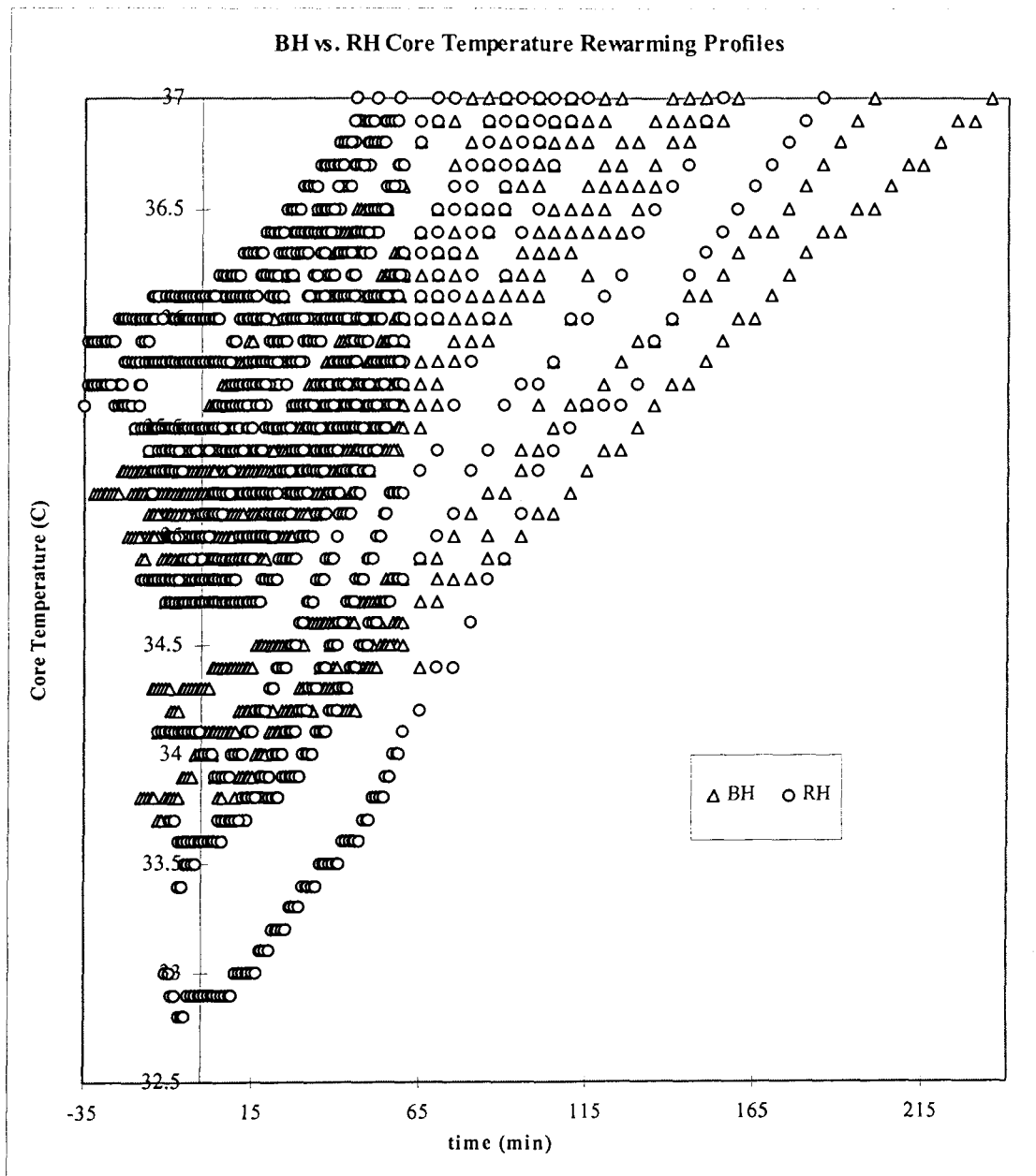
Upon examining Figure 3, it can be seen that both subject groups followed similar

rewarming patterns, in that there was a slow increase in T_{core} initially, which rapidly

increased in slope. Once the patient was introduced to the postoperative rewarming

conditions and became adjusted to the environment, the body was able to rewarm more

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homologously. This is represented by the more linear section in the T_{core} rewarming profile.

Figure 3. Core Temperature Rewarming Profiles (Random Sample)
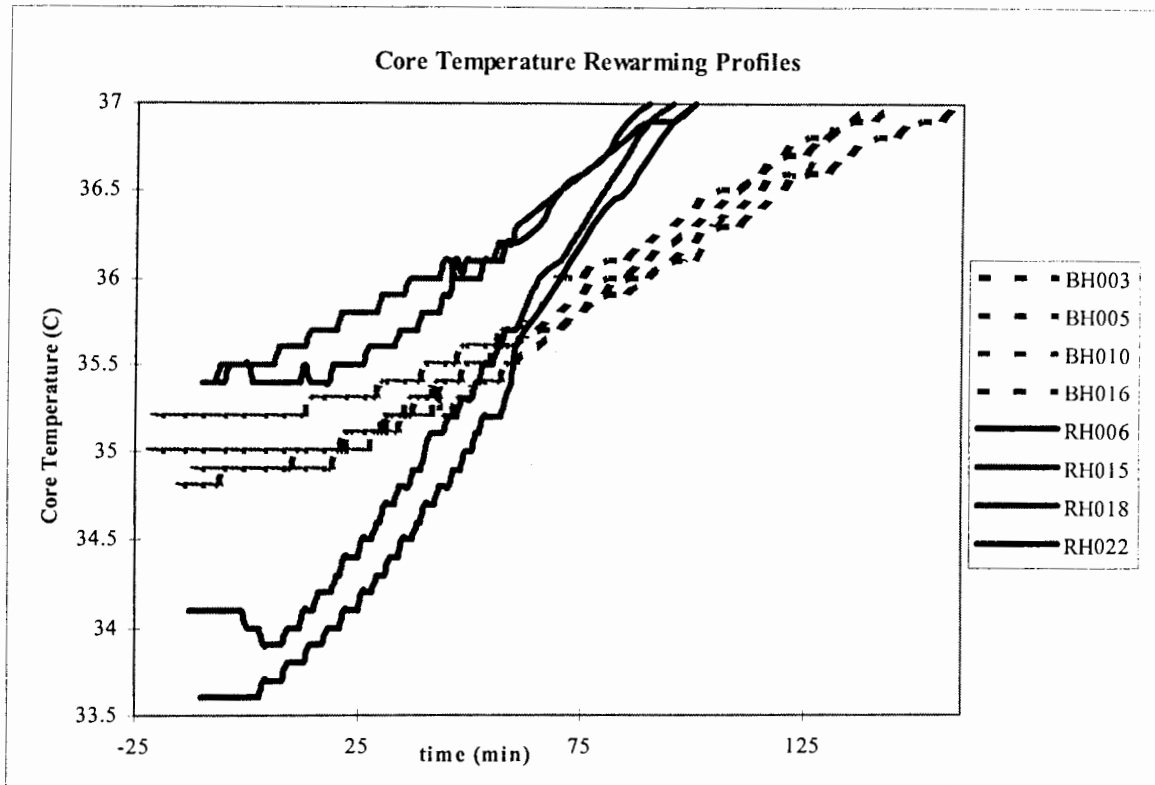
T_{core} **Rewarming Rate (°C/min).** The rate of rewarming was analyzed between groups for the entire rewarming period where a significant difference (two tailed $p=0.004$) was found. The radiant heat group had a significantly higher rewarming rate (RH = 0.022 $\pm$ 0.002°C/min) compared to the convective heat group (BH = 0.015 $\pm$ 0.001°C/min). The difference in rewarming rates is statistically significant ($p=0.004$) as well as clinically significant (>25% improvement). These rewarming rates can be viewed in Figure 3, by observing that the RH slopes are considerably steeper than in the BH group.

7.2 Mean Skin Temperature

The mean skin temperature for the groups was compared at minute 1, and no difference was found (BH = 30.2 ± 0.3ºC vs. RH = 30.0 ± 0.3ºC). The two groups started at similar temperatures, but quickly diverged. The radiant heat group took a much steeper increase in temperature to a higher plateau; while the convective heat group's rise was not as dramatic and plateaued lower. These results can be observed in Table 3, and Figures 4 and 5.

Table 3. Mean Skin Temperature

	Minute 26	Minute 52	Minute 72
BH	32.6 ± 0.2	33.6 ± 0.2	34.2 ± 0.2
RH	34.2 ± 0.2 *	35.6 ± 0.2 *	36.2 ± 0.2 *

* indicates significant difference

Post-hoc tests indicated there was a significant difference between groups at each of the time intervals, as designated in Table 3. Figure 4 graphs the mean skin temperature mean at the appropriate values of Table 3. The significant difference between groups is obvious when observing Figure 4. Figure 5 is an example Mean Skin Temperature Rewarming Profile between groups.

Figure 4. Mean Skin Temperature

Averaged Mean Skin Temperature over Rewarming Period
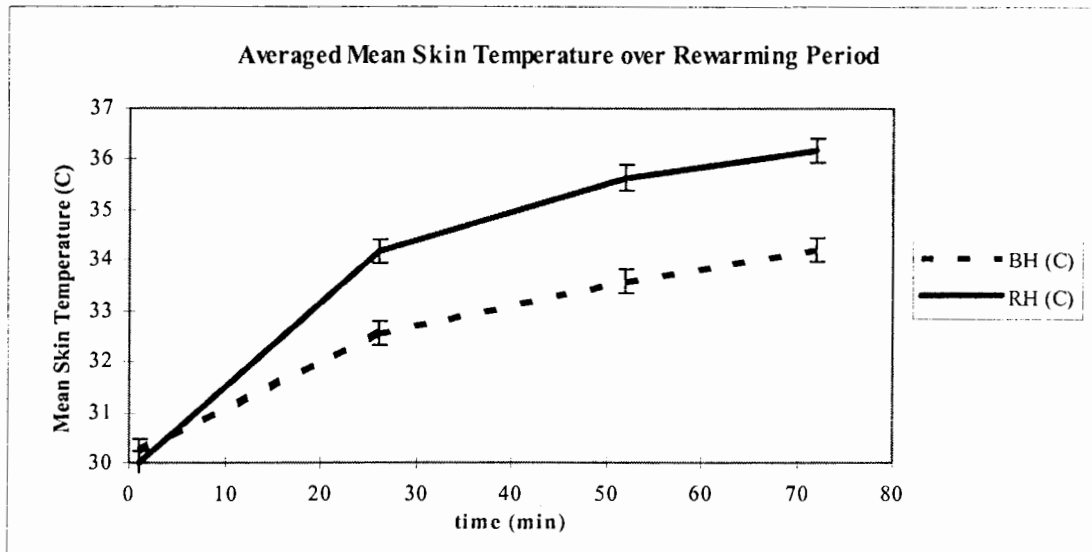
Figure 5. Example BH vs. RH Mean Skin Temperature Profile

BH vs. RH Mean Skin Temperatures over time
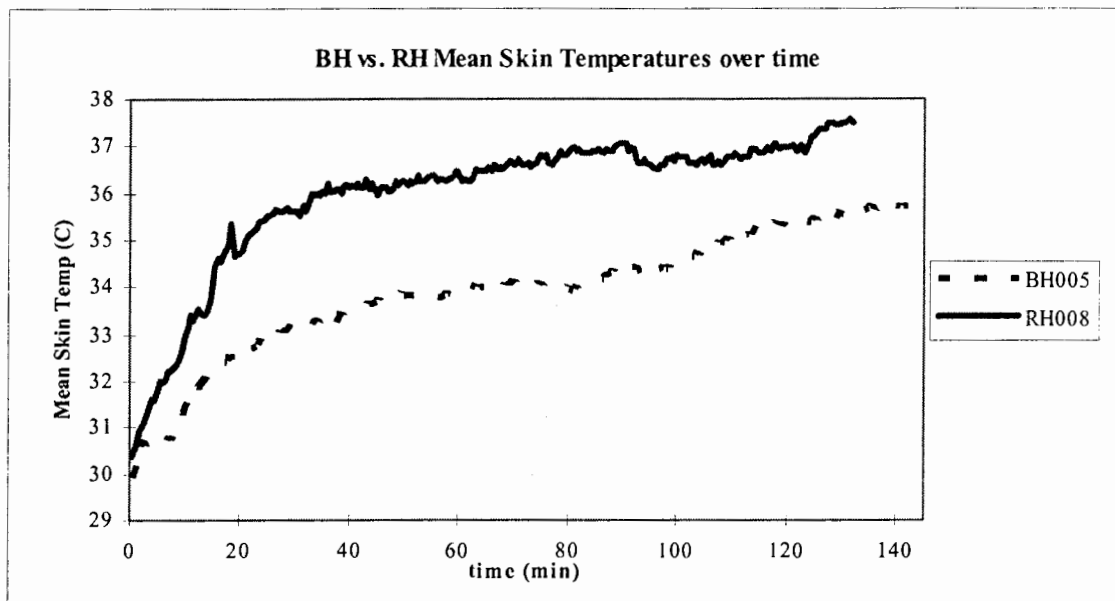
Analysis between the groups (BH vs. RH) of mean skin temperature at minutes 26, 52,

and 72, found a significant difference. Mixed design univariate ANOVA found that the

radiant heat group was significantly warmer than the convective heat group ($F(1, 31)$ = 67.88; p< 0.0001). (see Table 4.)

Table 4. Mean Skin Temperature ANOVA Table

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***************************
* Between Subjects Effects *
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Source	SS	DF	MS	F	P
Hypothesis	87.7383	1	87.7383	67.8810	0.0000
Error	40.0685	31	1.2925		

Examining the results of polynomial contrasts conducted *a priori* show that there is largely a linear trend ($F(1, 31)$ = 126.0; p<0.0001) in the mean skin temperature rewarming profile.(as seen in Table 5.)

Table 5. Mean Skin Temperature ANOVA Table. Single Degree-of-Freedom Polynomial Contrasts

Degree	SS	DF	MS	F	P
1	56.4341	1	56.4341	26.0155	0.0000
Error	13.8829	31	0.4478		
2	0.9950	1	0.9950	6.6914	0.0146
Error	4.6095	31	0.1487		

The means increase significantly in a linear fashion as the values of Mean Skin Temperature increase. This can be seen in Figures 4 and 5, in the steep initial segment, as well as the plateau portion of the graph. There is also a quadratic trend to the means'

increase ($F(1,31) = 6.7$; $p=0.0146$) as the values of Mean Skin Temperature increase (as seen in Table 5.). The quadratic trend is observed as the change in shaped ordering of the means between the initial steep slope and the plateau.

7.3 Forearm-Fingertip Temperature Gradient

The Forearm-Fingertip Temperature Gradient for the groups was compared at minute 1, and no difference was found (BH = $5.0 \pm 0.2^{\circ}$C vs. RH = $4.6 \pm 0.4^{\circ}$C). Another concern that may have affected the results was inotrope effects. Analysis of the inotropes used found that their was no difference between the groups, as seen in Table 2. Results can be observed in Table 6, and Figures 6 and 7. The BH and RH groups started at similar temperature gradients, but diverged, as did the Mean Skin Temperature (MST) profiles. (as seen above.)

Table 6. Forearm-Fingertip Temperature Gradient

	Minute 25	Minute 50	Minute 70
BH	5.4 ± 0.3	5.8 ± 0.4	6.0 ± 0.5
RH	4.5 ± 0.3	3.9 ± 0.4 *	4.1 ± 0.5 *

* indicates significant difference

While the divergence between the groups is not as great as with MST, the groups do proceed in opposite directions (as seen in Figure 6.) The convective group increased its temperature gradient over the rewarming period, and the radiant heat group decreased its

temperature gradient. Figure 6 graphs the Forearm-Fingertip Temperature Gradient at the appropriate values of Table 6. Post-hoc tests indicated there was a significant difference between groups at minutes 50 and 70, as designated in Table 6. This difference at each time interval can be observed in Figure 6.

Figure 6. Averaged Forearm-Fingertip Temperature Gradient Profile
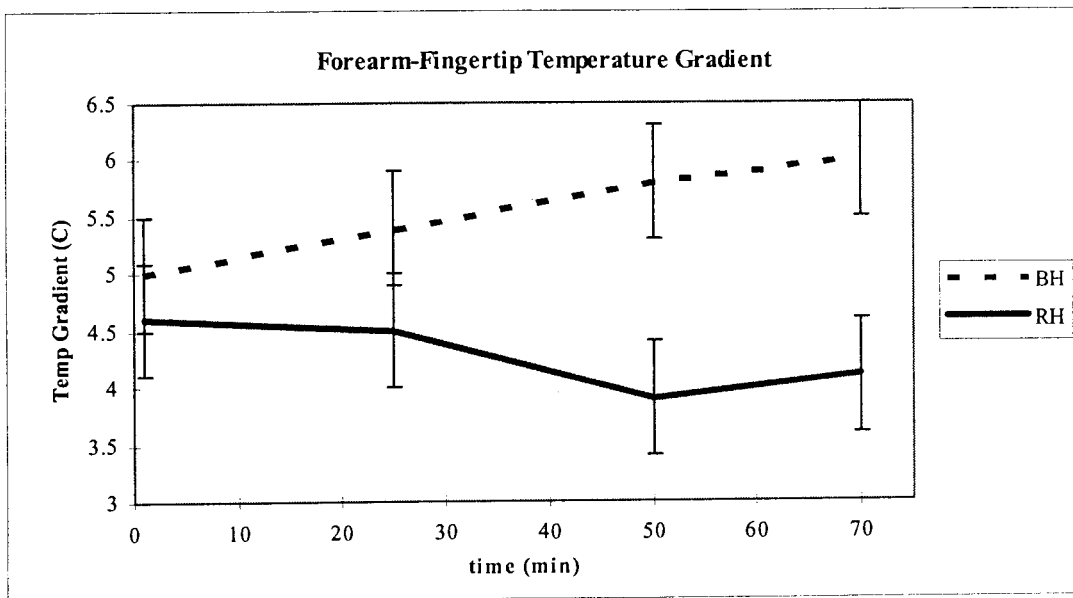
Figure 7 is an example Forearm-Fingertip Temperature Gradient Rewarming Profile between groups. This example shows two randomly sampled subject profiles.

Figure 7. Example Forearm-Fingertip Temperature Gradient Profile
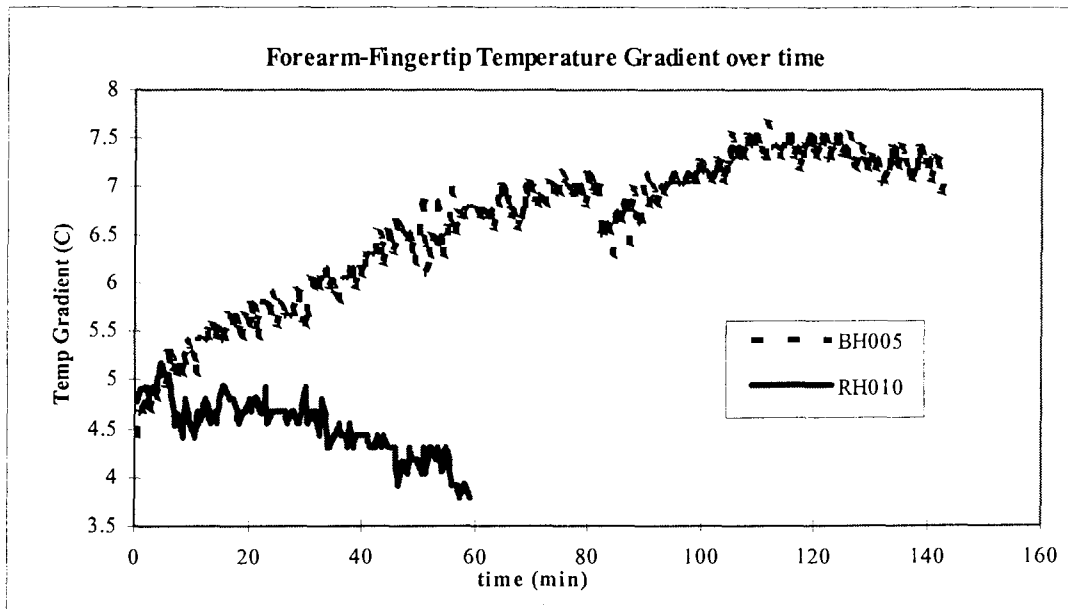
Table 7. Forearm-Fingertip Temperature Gradient ANOVA Table

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**************************
* Between Subjects Effects *
**************************
        Source      SS      DF    MS           F        P

Hypothesis    56.6909   1    56.6909     8.3184   0.0072
Error        204.4528   30    6.8151
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Analysis between the groups (BH vs. RH) of the forearm-fingertip temperature gradient at

minutes 25, 50, and 70, found a significant difference. Mixed design univariate ANOVA

found that the radiant heat group had a significantly smaller forearm-fingertip temperature

gradient than the convective heat group (F(1, 30) = 8.32; p= 0.0072).(Table 7.) The

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difference between groups increase slowly over the rewarming profile, as seen in Figure 6.

7.4 Shivering: VOSS measure

All patients were given neuromuscular block reversal upon entry to the CSICU and PAR. All patients' shivering was measured according to the VOSS scale, Table 1. Each patient's VOSS score was analyzed and averaged over his / her rewarming period. The average VOSS scores were transformed by square root, to give a normal distribution. The analysis of the transformed VOSS scores found a statistical difference between the groups (two tailed p= 0.000023). The BH group (BH = 0.70 ± 0.10) shivered significantly more than the RH group (RH = 0.09 ± 0.05). The BH group almost maintained a VOSS of 1, while the RH group was almost at VOSS zero, no shivering. The VOSS data were also analyzed with respect to the average number of times each patient reached the different levels of the VOSS scale, Table 8 and Figure 8.

Table 8. Average Number of Shivering Observations per Subject at Each VOSS Level

VOSS	BH	RH
1	13.9 ± 2.1	2.8 ± 1.7
2	8.4 ± 2. 5	0.6 ± 0.6
3	5.4 ± 2.4	0
4	1.9 ± 0.8	0

Examining the results of Table 8, it is clear that the BH group shivered not only more often, but also more severely than the RH group. The difference between the groups becomes even more obvious when looking at Figure 8.

Figure 8. VOSS Score Representation Average per Subject

Patient Ave of Number of Times Shivered at each VOSS score

(Y-axis: Ave # of Shivering Observations at VOSS X; X-axis: VOSS score; legend: BH, RH)
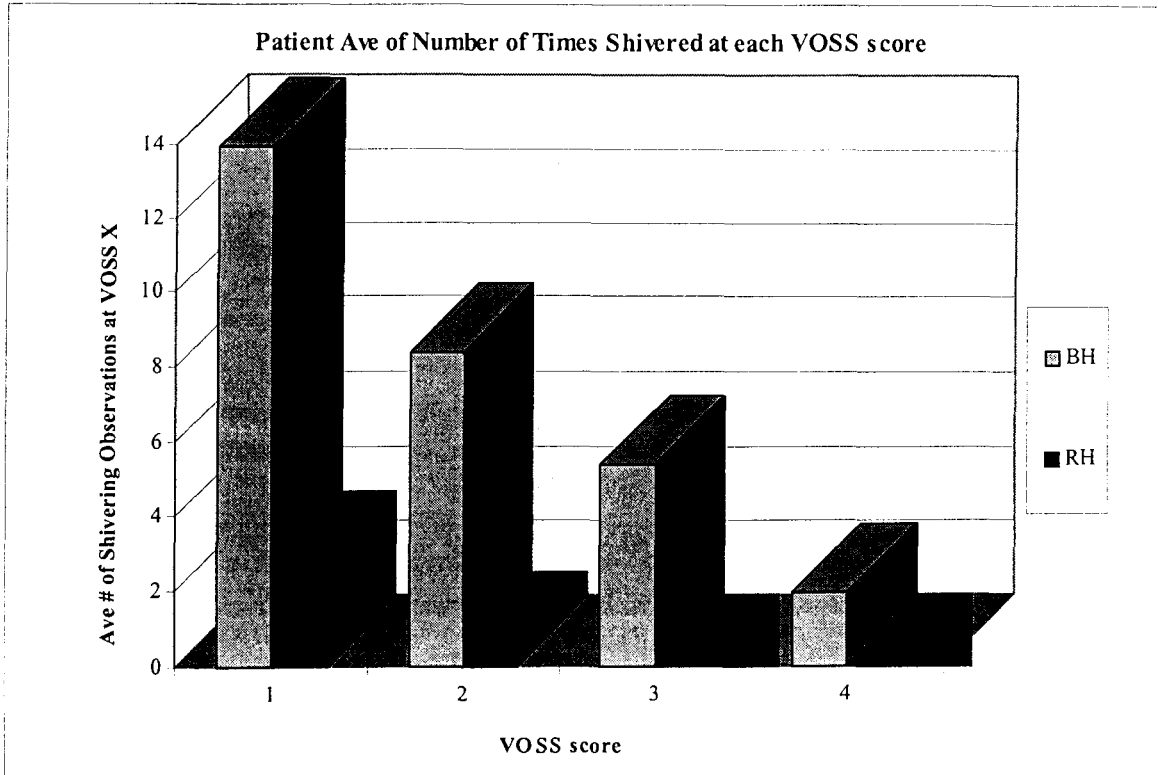
In Figure 8, the X-axis indicates the ascending levels of VOSS representing increasing severity of shivering. The Y-axis represents the average number of shivering observations at each level of VOSS per rewarming period. Figure 8 exemplifies the difference between the BH and RH groups, that the RH group shivered less severely and less often. The BH group experienced shivering at all levels of VOSS, including generalized shaking of the entire body; while the RH group sporadically reached a mere maximum shivering of VOSS 2.

8.0 DISCUSSION

There were many positive physiological and clinical results found in this study. The physiological results will be discussed followed by their clinical ramifications.

8.1 *Physiological findings:*

8.1.a Shivering

It has been shown that heating the pre-optic area in guinea pigs inhibits normal thermoregulatory responses to cold, such as shivering.[8] Also found was that the blush area in monkeys,[46] cats and human volunteers was sensitive to temperature and changes in temperature.[4,42,44] The blush area is innervated by the trigeminal nerve to the hypothalamus, and has been found to have a powerful influence on shivering.[45] By stimulating the blush area with radiant heat, researchers were able to inhibit shivering in hypothermic volunteers[45] and hypothermic postoperative patients[47,48,85,86] The results found in this study support the hypothesis that radiant heat on the blush area inhibits postoperative shivering. The frequency and severity of shivering in the radiant heat group was nearly zero, while the convective heat group consistently experienced shivering, with extreme epochs being observed. This can be seen in Figure 8. Clinically, these findings are significant to postoperative care for not only cardiac surgical patients, but all surgeries. By using radiant heat postoperatively, caregivers have a non-medicinal option to help control shivering. By inhibiting shivering with radiant heat, the demand and stress upon the body will be reduced. Patients will experience a more stable and unremarkable postoperative recovery, seen by decreased oxygen demand, myocardial work, respiratory

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demand, and mean arterial pressures.[18,48,74,75,80,81] A beneficial aspect of avoiding drugs

to control shivering is a simpler pharmacological profile for the patient.

Mekjavic and Eiken (1985) hypothesized that the mechanism inhibiting shivering

is that radiant heat on the innervated area causes a withdrawal of cold-receptor

stimulation.[45] The radiant heat on the blush area activates the warm-thermoreceptors and

overrides cold- receptor stimulation. By overriding the cold-receptor stimulation, the

afferent information processed at the hypothalamus represents the warm input, and the

cold thermoregulatory mechanism of shivering is discontinued. In this study the thermal

stimulation by radiant heat on the blush area was fairly intense at a skin temperature of

41ºC. This hyperstimulation of warm-receptors in the blush area feasibly caused the

withdrawal of cold thermoregulatory stimulation and therefore, inhibited shivering. Other

studies that have not found an inhibition of shivering with radiant heat may have followed

different methods.

8.1.b Peripheral Vascular Tone

The peripheral vasculature that contains arteriovenous anastomoses (A-V shunts),

such as the fingertips, have unique and specialized thermoregulatory functions.[41] The A-

V shunts respond to indirect heating and cooling, by vasodilating and vasoconstricting,

respectively.[41] When patients enter the CSICU from the O.R., they are peripherally

vasoconstricted. Their bodies cooled enough during surgery, despite the shift in the

thermoneutral zone (TNZ) due to the anaesthesia,[3,18,55,57,58,59,60,61,64,58] to reach threshold

and trigger peripheral vasoconstriction. The constricted A-V shunts in the hands decrease

blood flow to the tissues, and therefore, heat capacitance. Comparing temperatures

between the forearm (tissue without A-V shunts), and the fingertip (tissue with A-V shunts), reflects blood flow. The measure of forearm-fingertip temperature gradient is not used to quantitatively measure blood flow in the peripheral tissues, but to qualitatively indicate vasomotor activity. The forearm-fingertip temperature gradient has been positively correlated with the laser Doppler method[59] of blood flow measurement.

While inhibiting shivering by rewarming the patient with radiant heat on the blush area, this study aimed to use the same mechanism to inhibit peripheral vasoconstriction seen in postoperative patients. Heating the blush area to 41°C activated warm-thermoreceptors to override the cold- receptor stimulation, thus inhibiting peripheral vasoconstriction. It was expected that it may be difficult to trigger this mechanism since the peripheral vasoconstriction threshold is higher than the shivering threshold. This means that vasoconstriction occurs before shivering when a person is cooled. To inhibit peripheral vasoconstriction by overriding the cold stimuli in a hypothermic postoperative patient, an intense stimuli was required. Maintaining a skin temperature in the blush area of 41°C may have been sufficient to trigger the response, but the researchers included convective rewarming in the group to maximize the warm stimuli. Maximizing the warm stimuli to inhibit peripheral vasoconstriction was necessary to see if it could be accomplished. Examining the results, it seemed to be more difficult to trigger the mechanism to inhibit peripheral vasoconstriction. This is seen by looking at the results in Table 6, and Figure 6. The table indicates that minutes 50 and 70 were significant different, while minute 25 was not. This is also represented in Figure 6, which is seen as a slower change in difference between the groups. This may be simply due to at minute 25 the patients were still too cool to trigger the mechanism. In comparing the difference

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between groups for Mean Skin Temperature (Figures 4 and 5) and Forearm-Fingertip Temperature Gradient (Figures 6 and 7) over the rewarming profiles, the vasomotor mechanism was slower in its response. The slope at which these variables changed over time is much greater in Mean Skin Temperature than Forearm-Fingertip Temperature Gradient.

The results in this study indicate that the peripheral vascular tissues are less vasoconstricted when cardiac surgical patients are rewarmed postoperatively with radiant heat on the blush area. By causing the patient to be less vasoconstricted peripherally, there was effectively a greater volume of blood with which to transfer heat energy back to the core. By increasing the heat energy carrying capacity of blood returning to the core, patient rewarming was more efficient. The clinical significance of these results will be discussed in the next section. For this method to be effective in rewarming patients, a positive heat flux to the patient is required. This study combined the convective rewarming method with the intense radiant heat on the blush area, to have a positive heat flux. A common radiant heater on its own may not be able to provide intense enough heat energy, over a large enough area to create a positive heat flux. The radiant heater used in this study was constructed by design with curved reflectors behind the elements to increase the angles of incidence to the curves of the human body, and to maintain quite a high skin temperature (41°C) over a large surface area.

8.2 Clinical Findings

As discussed above, the triggering of physiological mechanisms improves the efficiency of postoperative rewarming. The core temperature rewarming profiles seen in Figure 3 elucidate the difference in methods in returning postoperative patients to normothermia. The radiant heat group was found to decrease the time to normothermia (by about 30%) by increasing the rate of rewarming (by about 50%). By triggering peripheral vasodilation and increasing heat capacitance of venous return to the core, the rewarming rate drastically improved. The higher mean skin temperatures in the radiant heat group suggest that there was more heat being supplied to the skin than in the convective heat group. With higher peripheral perfusion, more heat energy was available for transfer back to the cold core. A higher mean skin temperature is clinically relevant only when it is able to contribute to the rewarming of the core. Since the skin is an interface between the core and the environment, a positive heat flux through the skin to the core is required if non-invasive rewarming is advocated. A peripherally vasodilated patient has a greater potential to rewarm faster in this situation. These factors, peripheral vasodilation and a high mean skin temperature, contribute to the ultimate goal of rewarming the patient's core to normothermia.

Clinically, bringing the patient to normothermia in less time means removing the patient from a vulnerable and dangerous situation. By avoiding hypothermia the patient's functioning improves systemically, with regards to the cardiovascular, respiratory, hematologic, and immunologic systems, and specifically in myocardial and cerebral functioning."[74] Also improved is the function of the coagulation cascade,[77] as well as drug metabolism.[50,54,3,78]

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A concern was raised by medical staff that if patients were rewarmed too fast, patients might become peripherally vasodilated and have a sudden decrease in blood pressure. This was an issue addressed by the researchers before initiating the study. This would only be a concern for patients who were hemodynamically unstable, such as cardiac surgical patients, and these are normally monitored quite closely. The CSICU nursing staff were aware of the concern, and responded by adding volume (for example, blood and albumin) to the patient to stabilize them.

9.0 CONCLUSIONS

The results of this study strongly suggest a number of conclusions, both physiological and clinical. By applying radiant heat (41°C) on the blush area of postoperative cardiac surgical patients in combination with convective rewarming: shivering decreases in frequency and intensity, peripheral vasomotor tone is decreased, mean skin temperature is increased, normothermia is achieved faster, and the core rewarming rate is higher. These results suggest an improved rewarming method that may be utilized for all postoperative hypothermic patients. Hopefully the conclusions found in this study can be adopted in postoperative care, and patients will benefit with healthier recoveries.

Now that the researchers have shown that combined convective and radiant heat is more efficient than convective heat alone, further study is necessary to determine radiant heat's potential alone, and to clarify the best way to apply it.

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