PERIPHERAL SKIN TEMPERATURE CHANGES OF BURN WOUNDS

A THESIS

SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS

FOR THE DEGREE OF MASTER OF SCIENCE

IN THE GRADUATE SCHOOL OF THE

TEXAS WOMAN'S UNIVERSITY

COLLEGE OF NURSING

BY

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DENTON, TEXAS

MAY 1981

ACKNOWLEDGEMENTS

I want to thank my husband, Dr. Tom Staats, for his patience, guidance, and assistance in the editing of this thesis.

My committee members, Geri Goosen, Dr. Barbara Carper, Dr. Helen Bush, and Dr. Margie Johnson, have helped me immensely in the construction of this thesis.

I want to thank my mother, Anne R. Barnett, for her needed support. Thanks also go to all of my friends and family for comforting while I was away from my husband and developing this thesis.

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CHAPTER 1

INTRODUCTION

Annually, there are 200,000 individuals who thermally injure their skin, which is the largest and one of the most vital organs of the body. Thermal destruction of the skin causes altered anatomy and physiology in these individuals.

When skin is thermally injured, a major consequence occurs; that is, impaired vascular flow of the burned area. This occlusion of microvasculature will affect the healing process. An effective method of assessing the vascular flow to normal skin is by measuring the peripheral skin temperature.

Currently, the only accurate method of assessing the temperature variations of a burned wound is by infrared thermography. Infrared thermography has been utilized as an accurate method of evaluating the severity of a burn and of prognosticating the fate of the burned tissue, by measuring the temperature variations (Birch, Branemark, Nilsson, & Lundskog, 1968). The patency of the vascular supply to the skin appears to be the deciding factor in whether a deep

partial-thickness burn eventually heals or becomes an area of full-thickness loss.

Birch et al. (1968) emphasized the need for further investigations into the effects of burning on the burn vasculature and for further efforts directed toward maintaining circulation in these burned vessels. The peripheral skin temperature of normal skin is an accurate measure of vascular flow and/or metabolism. The peripheral skin temperature is elevated as a consequence of increased vascular flow and/or metabolism. With a decreased peripheral skin temperature there is a decrease in vascular flow and/or metabolism.

Problem of Study

The central problem formulated for this investigation was to describe the nature and course of temperature changes over time in the healing of deep partial-thickness and full-thickness burns.

Justification of Problem

The circulatory system supplies the body organs with the needed nutrients essential for metabolism and survival of the organs. The largest organ of the body is the skin, which has many functions. One of the

most important functions of the skin is the body's first line of defense against infection. Bacteria and other organisms are kept out of the body by the physical barrier of the skin. Loss of body fluids is prevented by the anatomy of the skin and balance of the delicate fluid is maintained. Body temperature is controlled by the decreasing or increasing evaporation of sweat glands located in the skin. The most extensive sensory organ of the body is the skin. Sensations are initiated at the surface of the skin and relayed to the central nervous system. The central nervous system interprets these sensations as light touch, pain pressure, hot, or cold; thus, allowing modification of the immediate environment to avoid damage or destruction.

Located within the skin are sebaceous glands which soften and lubricate the skin. Sunlight reacts with cholestrol compounds within the skin to make vitamin D. The skin has a cosmetic effect that varies from individual to individual which serves to identify the individual as a certain race but also by identifying individual textures such as the fingerprint patterns (Feller & Archambeault, 1974).

When the skin is burned, all of these functions are altered minimally in deep partial-thickness burns or entirely in full thickness burns. The circulation of the burned area is affected initially and some delayed reactions occur which could affect the depth of the burn and the healing process (Baxter, 1977; Buwalda, 1969; Chao, Eisley, & Yang, 1977; Hinshaw, 1968; Loebl, Marvin, Curreri, & Baxter, 1974; Order & Moncrief, 1964).

This investigation proposes to determine the circulatory changes of deep partial-thickness and full-thickness burns with the aid of a peripheral skin temperature monitor. The patency of the vascular supply to the skin appears to be the deciding factor in whether a deep partial-thickness burn eventually heals or becomes an area of full-thickness skin loss. There is a need for further investigation into the effects of burning on the burn vasculature and for further efforts directed toward maintaining circulation in these burned vessels (Birch et al., 1968). With the use of a biofeedback peripheral skin temperature monitor, a burned patient may be able to learn to increase the vascular flow to the injured area at will.

If a standardized method to determine the vascularity of a burn wound was established, then it may be feasible to diagnostically differentiate between deep partial-thickness and full-thickness burns. Being able to differentiate between the deep partial-thickness and full-thickness burns is a major problem in burn wound management.

Burn injury is known to cause complex changes in blood flow and in the coagulation process (Chao et al., 1977). Generally, the local tissue temperature is elevated by increased arterial vascularity or increased metabolism. With decreased circulation or decreased metabolism there is a decreased emissivity of heat from the skin (Winsor, 1971).

The presence and quality of the vascularization in the injured tissue determines the healing process. Thermography can be utilized in differentiating the various degrees of burns (Birch et al., 1968; Buwalda, 1969). There are several disadvantages of thermographic examination. A thermogram is very expensive. The patient has to be taken to a special room for thermographic examination; it cannot be done at the bedside. Also, the patient has to be exposed for

20 minutes to adjust to an ambient temperature of 70 degrees Fahrenheit or less prior to thermographic examination (Clark, Mullan, & Pugh, 1977).

The biofeedback peripheral skin temperature monitor is 1/30 of the cost of a thermogram, it is light-weight, and can be transported in a suitcase. The skin temperature of a burn wound can be taken at the bedside and there is no undue exposure of the burn wound area. Most important is that nurses can monitor burn patients' peripheral skin temperatures with the biofeedback instrument with short-term training. Burned patients may be able to increase the vascular flow through biofeedback training with a peripheral skin temperature monitor.

Theoretical Framework

Hunt and Van Winkle's (1976) theoretical framework of wound healing is comprised of five phases: (a) coagulation, (b) complement activity, (c) chemotaxis, (d) circulation, and (e) collagen. When a burn injury occurs, the open wound has collagen exposed, causing thrombogenesis. The platelets aggregate and there is a release of thromboplastin in injured tissue which stimulates clotting. This

comprises the coagulation phase of wound healing (Hunt & Van Winkle, 1976).

Baxter (1977) alluded to the coagulation phase by examining the systemic and local responses of thermal injury. The investigator feels as though it is germane to elucidate on the systemic and local responses of a burn injury. Baxter (1977) specified that the final amount of cell death and extracellular destruction is dependent on local chemical responses to the initial insult and to systemic factors set in motion by the systemic response to burn injury. Immediately following thermal injury, intense vasoconstriction results from histamine released and catecholamines. Within a few hours local vasodilatation occurs, as a result of kinins released from damaged cells. This vasodilatation in the burned wound facilitates increased capillary permeability and plasma goes into the tissues. This increased capillary permeability causes an increased hemoconcentration which enhances sludging of blood flow. With 24 hours of injury, platelets and leukocytes aggregate and stick firmly to the vessel walls, producing thrombosis, further occluding vascular flow.

Also, the edema formed by plasma going into the tissues (extracellular fluid diffuses into the extravascular compartment due to the increased capillary permeability) puts pressure on the vessels inhibiting vascular flow. The transfer of oxygen and nutrients to the damaged cells is impaired by all of the above factors (Baxter, 1977). There is also an increase in fibrin split products following thermal injury which supports the hypercoagulation of burn patients' blood (Curreri, Wilterdink, & Baxter, 1975).

Polymorphonucleocytes and macrophages migrate through the endothelial cells into the extravascular space (Hunt & Van Winkle, 1976). The complement activity ensues, which is bacteriolysis and phagocytosis of bacteria in the wound.

Chemotaxis is the ability of a chemical stimulus to attract the polymorphonucleocytes and macrophages to the bacteria. When complement is being utilized, this will facilitate chemotaxis and is believed to be the chemical stimulus for chemotaxis (Hunt & Van Winkle, 1976). These first three phases of wound healing (coagulation, complement activity, and chemotaxis) occur within minutes after injury. The

circulation phase of wound healing occurs 5 to 7 days postburn. During this phase the clotted vessels become patent and clots dissolve. In the periphery of the burn wound neovascularization occurs, which is proliferation of new capillaries. These new capillaries bring more blood supply, more oxygen to the injured tissues, and the new capillaries clear the tissues of metabolic by-products. This capillary proliferation precipitates re-epithelialization (Hunt & Van Winkle, 1976). Deep partial-thickness burns re-epithelialize not only from the wound edge but from remaining hair follicles and other deep dermal appendages. Full-thickness burns can heal only from the edge of the injury.

As a better nutrient supply to the injured area is enhanced by capillary proliferation, the fibroblasts make more collagen. Fibroblasts are cells from which connective tissue is developed. These fibroblasts are stimulated by inflammation to produce collagen (Hunt & Van Winkle, 1976). Collagen is a protein substance of the body and is the major constituent of skin, tendons, ligaments, bones, cartilage, fasia, and the septa of various organs. The principal

component of scar tissue is collagen, which gives the healed area strength.

Assumptions

The assumptions of this investigation were:

1. Coagulation of the vasculature does occur after a thermal injury.

2. Sludging of vascular flow occurs after a thermal injury.

3. Neovascularization does occur as burn wounds are healing.

4. All of the above will affect the peripheral skin temperature and be detectable by peripheral skin temperature monitoring.

5. Peripheral skin temperature is a reflection of vascular and/or metabolic alterations.

Research Question

What is the nature and course of temperature changes over time in the healing of deep partial-thickness and full-thickness burns as measured by a peripheral skin temperature monitor?

Definition of Terms

For the purposes of this investigation, the ensuing terms are given the following specific definitions:

1. Deep partial-thickness burn--a second degree burn with destruction of varying depths of epidermis with hair follicles, sebaceous glands, and sweat glands remaining viable.

2. Full-thickness burn--a third degree burn having no surviving epithelial elements and complete coagulation of subdermal vessels.

3. Microvasculature--small blood vessels within the body that supply the skin with adequate circulation and nutrients.

4. Peripheral skin temperature--an external correlate of blood flow in the vasculature.

5. Healing process--the restoration of the burn wound to a normal condition.

6. Neovascularization--the proliferation of new blood vessels.

7. Control--an analogous non-burned area on each subject.

Limitations

The limitations of this investigation are as follows:

1. The sample consisting of seven subjects did not allow for generalization to the population.

2. A purposive sample drawn from one geographic area was utilized in this study. This limited generalization to other population groups.

Summary

A major consequence of a burn injury is impaired vascular flow to the burned area. This occlusion of microvasculature will affect the healing process. Nurses can measure the vascular flow to a burned area with the aid of a Biofeedback Peripheral Skin Temperature Monitor. The research question of this study is: What are the peripheral skin temperature changes of healing full-thickness and deep partial-thickness burns? Hunt and Van Winkle's (1976) theory of wound healing is utilized as the theoretical framework for this study. Definition of terms and limitations of the study have been presented.

CHAPTER 2

REVIEW OF THE LITERATURE

The following review of literature is organized along the lines of Hunt and Van Winkle's (1976) theory of wound healing. This is followed by a discussion of methods for measuring temperature variations of burn wounds.

Theory of Wound Healing

Coagulation

In the coagulation phase of wound healing, thrombogenesis occurs due to an open burn wound having collagen exposed. The platelets aggregate and there is a release of thromboplastin in injured tissue which stimulates clotting (Hunt & Van Winkle, 1976).

Sevitt (1957) investigated the alterations in vascularity of rabbit ears that had been emersed in hot water. The first effect of heat was immediate blanching due to contraction of skin capillaries. Blanching passes off within a minute then erythema develops. This erythematous area is due to dilatation of capillaries and arterioles. Blood flow

through the affected area is increased and local skin temperature rises 1 to 2 degrees centigrade. Later, capillary blood flow decreases, the erythema may become slightly cyanotic from the reduction of hemoglobin, diapedsis of red cells taking place, and pavementing of capillary wall by leukocytes occurs. This process is associated with edema. One of the most important effects of burning is local development of an abnormal capillary permeability which allows protein-rich fluid to escape into the tissue spaces. Loss of fluid from heat-affected capillaries may continue up to 24-48 hours or sometimes longer (Sevitt, 1957). This loss of capillary fluid causes hemoconcentration and enhances slowing of blood flow.

Sevitt (1957) found that in severe burns the capillary endothelium is severely affected. Capillaries exude fluid rapidly and erythrocytes flowing along minute vessels become more and more concentrated. As a consequence, capillary resistance increases and blood flow becomes stagnant and ends in cessation or stasis of blood flow associated with blockage of minute vessels by tightly packed masses of red cells. Histological examination shows capillaries dilated and

tightly packed with blood corpuscles. These corpuscles are not true thrombi but are blood cell clumps undergoing necrosis, which is part of the general necrobiotic change occurring in the area affected by stasis. Stasis develops hours, minutes, or seconds after burning; the interval decreasing as the severity of burn increases until stasis develops within the burning period. A high temperature of 68 degrees centigrade caused stasis at 10 seconds (Sevitt, 1957). Stasis is important in burns because:

1. It is irreversible--the affected area will die and slough (i.e., a deep partial-thickness burn will convert into a full-thickness burn if stasis is through the whole depth of the skin. If stasis is not through the whole depth of skin the flow in deep dermis may return to normal and epithelialization will take place).

2. In extensive burns a significant part of red cell volume is trapped in engorged capillaries and is permanently lost to the circulation. Rapid leakage of fluid-causing edema also results in concentration of capillary blood and hence stagnation of the flow (Sevitt, 1957).

Extent of tissue death following burn injury can change for varying periods of time. These periods of time may be influenced by slowing of the circulation, thromboses, edema, and infection (Goulian & Conway, 1968).

Hinshaw (1968) and Dobrkovsky, Malek, and Zastava (1968) supported the idea of a deep-partial thickness burn converting to a full-thickness burn due to stasis, which was acclaimed in Sevitt's (1957) study. Hinshaw (1968) added that edema formation will further occlude vascular flow and convert a deep partial-thickness burn to a full-thickness burn.

Moylan, Inge, and Pruitt (1971) checked the circulation of 60 limbs that had sustained circumferential burns. The circulation was evaluated with an ultrasonic flowmeter. All of the limbs had impaired circulation below the level of the burn. Escharotomies were performed and a return of arterial flow occurred immediately. This study indicated the deleterious affect of edema on vascular flow.

A study by Order and Moncrief (1964) demonstrated the vascular alterations produced by second and third degree experimental burns and those produced

by burn wound injuries occurring in clinical fatalities. After producing 20% second and third degree burns in rats, sequential injection studies were performed immediately, 24 to 48 hours, 1, 2, and 3 weeks following injury, Immediately following experimental third degree burn injury, most of the subpapillary plexus is occluded. Twenty-four hours postburn wound injury, the entire vascular architecture lying beneath the burn wound is occluded and devitalization is complete. Forty-eight hours postburn the vascular architecture remains unchanged and the inflammatory reaction present is limited to the fascia. Second degree experimental burn wound injury does not have the associated marked vascular loss seen in the immediate postburn period. Twenty-four and 48 hours postinjury, more of the subpapillary plexus was intact and there was no period of complete devitalization of the entire wound.

Knisely (1968) denoted that blood passing through small vessels in a burned area changed to a sludge. In burned animals and humans, Knisely (1968) directly observed large masses of agglutinated blood cells plug small vessels immediately. The forcible reduction of

flow all over the body caused by the change in the physical consistency of the blood led to hypoxia of vessel walls, loss of body fluids into the tissues, progressive hemoconcentration of the passing blood, and final stuffing and impaction of small vessels with sludge.

Schoen, Wells, and Kolmen (1971) examined the microcirculatory flow following flame injury. An increased blood viscosity after severe flame burns has been attributed to contributing to the decreased microcirculatory flow and tissue ischemia. Ten mongrel dogs were subjected to an open-flame injury. The burn was over 50% total body surface area and third degree. From preburn values the viscosity of whole blood increased 19% immediately after the thermal injury, with a maximum of 49% increase in blood viscosity 90 minutes after injury. Changes in the blood viscosity correlated with the increased hematocrit. The concentration of fibrinogen increased by 5% immediately after thermal injury, with a maximum increase of 14% from preburn values 60 minutes after thermal injury. Changes in blood viscosity correlated with changes in plasma fibrinogen. Blood viscosity

is increased due to the effects of increased hematocrit and fibrinogen. The increased blood viscosity contributed to the microcirculatory impairment by an increased resistance to blood flow through the microvasculature.

Jelenko, Jennings, O'Kelley, and Byrd (1971) attempted to define the threshold of body surface area which must be burned in order that distant microvasculature exhibited irreversible alterations. Contact burns of 15 seconds duration at 730 degrees centigrade were placed on the flanks of rabbits. Photomicrographs of the omental microvessels were obtained and the internal cross-sectional diameter of the vessels was measured. Transudation of plasma, sludging, granular flow, leukocyte pavementing, platelet thrombi, and arteriolar and venular plugging were noted in one-half of the subjects with 1.7% total body surface area burns, and in all of the subjects with 5% total body surface area burns (TBSA). The data in this study indicated that marked microvascular changes are associated with relatively small burns.

In 1973, Jelenko, Jennings, O'Kelley, and Byrd repeated the above study, but subjected the rabbits

to larger percentage of burns. The rabbits' omental microvasculature exhibited arteriolar dilatation associated with distant burns of 10% TBSA or less. Arteriolar construction was a constant finding if burn trauma exceeded 8% TBSA. As the burn increased in magnitude, the magnitude of arteriolar dilation decreased. Distant arterioles respond to burn injury by varying their diameters by an amount inversely proportional to the magnitude of injury. Intravascular sludging, thrombus formation, and plasma exudate was observed frequently and associated with burns of all sizes but was more profound and frequent following larger burns (Jelenko et al., 1973).

These results were supported by Zarem and Pandya's (1974) exploratory study on the effects of burns on vascular flow. Chao et al.'s (1977) study affirmed the results of impaired vascular flow in distal arterioles to the burn injury, as stated in Jelenko et al.'s (1973) study.

Curreri et al. (1975) revealed that elevated fibrin split products occur following thermal injury. Fifty-five burned patients' serum was assayed serially during the first 2 postburn weeks for fibrin split products by the staphylococcal clumping test (SCT).

The SCT results were increased above normal in all patients at some period during the first 2 postburn weeks. The ilbrin split products were elevated consistently during the fourth and eighth days postburn (Curreri et al., 1975). This study supports the coagulation phase of Hunt and Van Winkle's (1976) wound healing theory.

In addition to the previously mentioned studies, Peacock and Van Winkle (1976) indicated that histamine, serotonin, and kinins cause the increased vascular permeability in injured tissues. The venules leak fibrin into the nearby lymphatics and the lymphatics become plugged. The stoppage of lymphatic drainage, local vasodilatation, and leakage of fluid into extravascular space produce the classic signs of inflammation which are: redness, swelling, and heat.

Baxter (1977) supported the physiological reactions of burn injury described by Peacock and Van Winkle (1976). In addition, Baxter (1977) stated that the progressive vascular impairment may produce an area of ischemia 3 to 7 times greater in depth than the initial cellular damage initiated by the heat. The thrombotic tendency in the burn wound is present for a minimum of 5 days (Baxter, 1977).

Fox and Lasker (1962) studied the fluid and electrolyte alterations in burned monkeys. One group of monkeys was subjected to flash burns and the other group of monkeys was subjected to scald burns. In flash burns there was an increase in venous pressure which indicated intense vasoconstriction. The range of increase was from 3 to 28 ml. with an average of 12 ml. above the preburn values. Fox and Lasker (1962) implied that the rise in venous pressure postburn suggests the early onset of circulatory depression.

Jelenko, Jennings, Byrd, and O'Kelley (1974) checked for the presence of an arteriolar constrictor substance in blood of burned patients. There were seven albino rabbits in the control group and in the burned group. When whole blood or plasma from burned subjects was injected into unburned subjects the arteriolar constriction paralleled the changes in burned subjects. Even when dialysis of the plasma was performed it still was associated with arteriolar constriction after infusion. This suggests that the blood-borne arteriolar constrictor substance had a large molecular weight or a smaller molecule which is

protein bound. An arteriolar constrictor substance was identified in the study by Robson, Del Beccaro, and Heggers (1979).

Robson et al. (1979) demonstrated the effect of prostaglandins on the dermal microcirculation after a burn injury. The zone of stasis of a burn wound would convert into a zone of coagulation because prostaglandins decreased the internal diameter of the microvasculature (Robson et al., 1979). This study revealed the arteriolar constrictor substance, prostaglandins, that Jelenko et al. (1974) proposed existed.

Barr and Arrowsmith (1970) examined the thermal damage to red blood cells of a burn patient's blood and on whole blood in vitro that was heated. A scanning electron microscope was utilized to enable the direct examination of a cell surface and photographs were taken. There were two mechanisms of cellular damage evident in the burned patient's blood. The first mechanism was that of immediate severe damage. The second mechanism was that the transfused blood cells were also severely damaged. Heat damage in erythrocytes was accompanied by the development of

a general coarseness of the membrane and areas of pitting. It was suggested that one mechanism responsible for the reduced viability of red cells in burned patients was due to lipid and phospholipid loss. Thermal damage might therefore cause premature aging of erythrocytes.

Loebl et al. (1974) believed that decreased erythrocyte survival does not result from intrinsic damage to the red cells, but to extracorpuscular factors present in the circulation of the burned individuals. This study used two groups: Group I had 9 nonburned individuals and 13 burned patients; Group II had 14 burned patients. Rachromate is a radioactive material that combines with erythrocytes and was utilized to determine the half-life of the erythrocytes. In this study it was found that the half-life or normal erythrocytes from nonburned donors was markedly decreased when administered to burned patients. The autologous half-life of burned patients' own erythrocytes was similarly decreased. When the burned patients' cells were studied in non-burned volunteers, red cell survival was found to be normal. This study characterized but did not

precisely identify a humoral mechanism which results in increased erythrocyte destruction following thermal injury.

Harris, Cottman, and Baxter (1980) examined the pathogenesis of abnormal erythrocyte morphology in burns. In 30 burn patients, plasma and cell membrane lipids were analyzed bi-weekly. Plasma-free fatty acids (FFA) averaged 2 times normal. All patients exhibited abnormal erythrocytes called echinocytes. Normal red blood cell morphology was restored by incubation with fat-free albumin or normal plasma, but not by dialysis. Echinocytes were reformed by adding free fatty acid. These results suggest a primary role of increased FFA in the sustained abnormal red blood cell morphology in burns (Harris et al., 1980).

All of the previously mentioned studies signified the coagulation phase of Hunt and Van Winkle's (1976) theory of wound healing. The next phase in the theory of wound healing is the complement phase.

Complement Phase

Polymorphonucleocytes and macrophages migrate through the endothelial cells into the extravascular space. Then complement activity ensues, which is

bacteriolysis and phagocytosis of bacteria in the wound (Hunt & Van Winkle, 1976).

Cuthbertson and Tilston (1970) stated that shortly after the initial changes in a burn injury there was an increase in polymorphonuclear leukocytes to aid in the defense against infection. Peacock and Van Winkle (1976) further supported the complement phase of wound healing by asserting that diapedesis occurs after burn injury. Diapedesis is when leukocytes move through the vessels to the extravascular space. Peacock and Van Winkle (1976) also, acclaimed that polymorphonuclear leukocytes concentrate at the site of injury.

The chief function of leukocytes is to protect the body against microorganisms which cause diseases or infections. Foreign particles such as bacteria may be engulfed or phagocytosed by the leukocytes (Miller & Keane, 1972).

In this section, works of authors have been presented which support the complement phase of the Theory of Wound Healing (Hunt & Van Winkle, 1976). The next phase to be discussed is the chemotaxis phase.

Chemotaxis Phase

Chemotaxis is the ability of a chemical stimulus to attract the polymorphonucleocytes and macrophages to the bacteria. When complement activity is occurring, this will facilitate chemotaxis and is believed to be the chemical stimulus for chemotaxis (Hunt & Van Winkle, 1976).

Peacock and Van Winkle (1976) further supported the chemotaxis phase. These authors denoted that leukotaxine, a chemotactic agent, attracts leukocytes into the injured area.

The first three phases of wound healing (coagulation, complement activity, and chemotaxis) occur immediately after injury. The circulation phase of wound healing occurs 5 to 7 days postburn and is the next phase that will be reviewed.

Circulation Phase

During the circulation phase the clotted vessels become patent and the clots dissolve. In the periphery of the burn wound neovascularization occurs. Neovascularization is the proliferation of new capillaries. More blood supply and more oxygen are brought to the injured tissues, and the tissues are cleared of

metabolic by-products by the new capillaries. Re-epithelialization is precipitated by this capillary proliferation (Hunt & Van Winkle, 1976).

Order and Moncrief's (1964) study disclosed that full-thickness burns exhibited revascularization by granulation tissue, 1 week postburn, but most of the burn wound remained avascular. Two and 3 weeks post-burn, a progressive increase in the neovasculature was seen and was limited primarily to the fascia. Deep partial-thickness burns, 1 week postburn, exhibited revascularization accomplished by the reestablishment of patency in the arterial plexus, as well as small foci of granulation tissue.

Order and Moncrief (1964) demonstrated that in third degree burns there is a slow process of revascularization and revascularization is incomplete as late as 3 weeks postburn injury, which emphasizes the vascular destructive effect of third degree burns. In direct contrast, Order and Moncrief (1964) revealed that the rapid revascularization and lack of complete devitalization that is associated with second degree burn wound injury explains the regenerative capacity often clinically associated with this type of injury.

In the circulatory phase, more nutrients are supplied to the injured area by capillary proliferation. Then collagen is produced by fibroblasts which marks the onset of the collagen phase of Hunt and Van Winkle's (1976) theory of wound healing.

Collagen Phase

Fibroblasts make collagen. Fibroblasts are cells from which connective tissue is developed. Inflammation stimulates fibroblasts to produce collagen (Hunt & Van Winkle, 1976). Collagen is the principal component of scar tissue and gives the healed area strength.

Chao et al. (1977) avowed that full-thickness burns have no surviving epithelial elements and the destroyed area becomes dry, hard, and black. In the viable tissue below the slough, cellular and capillary activity produce a layer of granulation tissue, and the enzymatic activity of this layer loosens the slough which finally flakes off and exposes the red surface of the granulations. Granulations are made up of loops of newly formed capillaries and fibroblasts. In full-thickness burns the granulations contain no epithelial cells; therefore, healing can occur only

by ingrowth of cells from the surviving epithelial edge around the burn site. Then there is a reduction in vascularity in granulation tissue, and the tissue finally becomes scar tissue primarily made up of collagen. Wound healing ceases when scar tissue is formed in the collagen phase.

The preceding discussion of research on vascularity and burn wound healing, organized according to the theoretical framework of Hunt and Van Winkle's (1976) theory of wound healing, provides the foundation of knowledge about the hypothesized relationship of severity of burn and skin vascularity in the wound itself and at distal points. The following section will explore the relationship of vascular flow, peripheral skin temperature, and methodologies for their measurement.

Methods of Measuring Skin Temperature of Burn Wounds

Wilmore, Orcutt, Mason, and Pruitt (1975) implied that burn patients have a readjustment in central temperature setpoint of the hypothalamus. Readjustment in hypothalamis temperature setpoint following burn injury was suggested by burn patients choosing

an elevated ambient temperature for comfort. The control group consisting of five nonburned individuals felt comfortable at an ambient temperature of 27.8 degrees centigrade, while the nine burned patients had an increased ambient temperature of 30.4 degrees centigrade for comfort. This study indicated that burned patients do have a differential temperature preference from normal individuals, but the actual skin temperatures were not measured.

Chao et al. (1977) presented that the exudate in deep partial-thickness burns had a heat-dissipating barrier. This resulted in a higher skin temperature at the burn site. In full-thickness burns the eschar causes a certain diffusion resistance. When the injured tissues were resuming their proper function, the fluid leakage is reduced during the process of healing. The healing surfaces are becoming dry and the temperatures are beginning to drop to normal values. Chao et al. (1977) did not specify the method of measuring the skin temperature but did indicate that burned skin temperature is higher than nonburned skin temperature.

Aulick, Wilmore, Mason, and Pruitt (1977) studied the influence of the burned wound on peripheral circulation in thermally injured patients. Twenty-eight thermally injured patients with a mean burn size of 39.5% total body surface and nine normal individuals were studied. Copper-constantan thermo-couples were attached to the burn wounds and nonburned areas of all subjects. A rectal probe was utilized to monitor the body core temperature of the subjects. The subjects' legs were placed in a full-length plethyomograph to monitor the leg blood flow of burned and nonburned legs. The results of this study were that leg blood flow and mean leg skin temperatures were significantly increased by the local presence of the burn wound. Leg blood flow in the uninjured limbs of burned patients was similar to that of the normal controls. In all subjects studied, leg blood flow increased with the extent of the leg burn.

Aulick et al. (1977) mentioned that in third degree wounds, which are associated with superficial vascular thrombosis, leg blood flow is near control levels shortly after injury. Leg blood flow increases

by the end of the first week, which is associated with formation of a richly vascularized wound bed. Conversely, deep partial-thickness injury does not destroy the superficial vascular bed, and blood flow is elevated in these legs as soon as circulatory volume is restored.

Currently, one accurate but extremely expensive method of measuring skin temperature of burn wounds is thermography. Another method utilized to measure normal skin temperature is a Biofeedback Peripheral Skin Temperature Monitor. The following section is a review of literature of these two methods of measuring skin temperature of burn wounds.

Thermography

Thermography in the measurement of infrared waves emitted by all objects which are above absolute zero (Mladick, Georgiade, & Thorne, 1966). In Mladick et al.'s (1966) study, burn patients were placed in a 72 degree Fahrenheit room for 20 minutes, then thermography was taken. Full-thickness burns were colder than normal skin and appeared as black areas on thermograms. Deep partial-thickness burns were 2 degrees centigrade cooler than normal skin.

After the acute stage (1 week) the deep partial-thickness burn areas gradually warmed up as healing progressed until eventually only the full-thickness burns remained as cold spots.

Birch et al. (1968) evaluated the severity of burn wounds with infrared thermography. Twelve healthy rabbits' ears were shaved and burned with 82 degrees centigrade circulating water. Infrared thermograms showed cold areas over most burns revealing that circulation underlying the skin had been destroyed. Infrared thermography appeared to be able to detect heat from underlying vessels through the eschar and, thus, gave a fairly accurate prognosis of the burn. A few of the large central vessels which had remained open following the burn thrombosed; an event which only the infrared thermogram was able to record.

From the fifth day until the third week, infrared thermography showed a slight reduction in the size of the central cold area of the burn as the microvasculature around the periphery of the burn became more profuse and as preexisting vessels increased in size. Infrared thermography was the most accurate method of

determining whether the burn was deep partial or full-thickness because thermograms showed whether or not the large important subdermal vessels were occluded. Birch et al. (1968) stated that infrared thermography is an accurate method of evaluating the severity of burn and of prognosticating the fate of the burned tissue.

In addition to the previously mentioned studies, Buwalda (1969) found that the best thermographic diagnosis of a burn was made within 12 hours after the trauma. Buwalda (1969) stated that with thermography the beginning of disturbance in circulation can be detected and treatment can be instituted accordingly.

Winsor (1971) disclosed that the thermographic evaluation of a burned patient produces a pictorial record of the degree, the extent, and geographic distribution of burns with a high degree of accuracy. Deep partial-thickness burns were on the average 2 degrees centigrade cooler than the normal skin, and full-thickness burns were greater than 2 degrees centigrade cooler than normal skin. Thermography yields information about soft tissue abnormalities, such as burns and trauma, not obtainable in any other way (Winsor, 1971).

Hackett (1974) compared the accuracy of diagnosing the depth of burn with chemography against assessments made by plastic surgeons. In 314 burned areas involved, the surgeons were incorrect 74 times and in 28 areas the surgeons did not know. Thermographic diagnosis was incorrect in 34 instances. Thermography is considered a superior method of diagnosing the depth of a burn than simple clinical examination (Hackett, 1974).

The utilization of thermography in diagnosing the degree of burn and prognosticating the fate of burned tissue has been upheld by the previously mentioned studies. The thermography studies (Birch et al., 1968; Buwalda, 1969) emphasized the importance of detecting vascular flow in the injured area to determine an accurate diagnosis and prognosis of the burned area. One current cost-effective method of assessing vascular flow to normal skin is the Biofeedback Peripheral Skin Temperature Monitor. Following is a review of literature of the Biofeedback Peripheral Skin Temperature Monitor and vascular changes.

Biofeedback Peripheral Skin Temperature Monitor

Roberts, Kewman, and MacDonald (1973) investigated the voluntary control of skin temperature in seven hypnotic subjects. Skin temperatures were recorded on a Grass Model-7 Polygraph with two 10,000 ohm Fenwall Uni-curve Interchangeable curve-matched Thermistors taped to the pad of the subject's middle finger on the left and right hands. The first four subjects were able to voluntarily raise and lower the peripheral skin temperatures of each hand, while under a hypnotic trance.

During a separate special session, the peripheral skin temperature of Subject 1 was monitored while blood flow was recorded over the volar digital artery using an ultrasonic flow meter and transcutaneous probe. When the subject increased the peripheral skin temperature in the monitored hand, an accelerated pulse and blood flow were detected. When the subject decreased the peripheral skin temperature in the monitored hand, blood flow was almost completely cut off except in the suppressed arterial pulse. Roberts et al. (1973) suggested that voluntary control in regulating the peripheral skin temperature appears to be of sufficient

magnitude to make possible the therapeutic management of disorders that might be helped by localized changes in blood flow (e.g., burns, arthritis).

Lynch and Kohn (cited in Miller, 1974) also showed that the learned change in peripheral skin temperature involves abrupt changes in peripheral circulation. Lynch and Kohn (cited in Miller, 1974) utilized the same procedure as in Roberts et al.'s (1973) study. Miller, Barber, DiCara, Kamiya, Shapiro, and Stoyva (1974) stated that the control of temperature (or of peripheral circulation) is likely to be an especially good model situation for discovering and studying the various factors that have significant effects on the learning of a visceral response.

The cardiovascular mechanisms which regulate skin temperature in the hands are closely tied in with the activity of the sympathetic division of the autonomic nervous system. When sympathetic activation occurs, this results in vasoconstriction. As a result the flow of blood to the area will decrease. As decreased quantities of blood flow through the capillaries to the tissues near the skin surface, the skin temperature will drop (Autogenic Systems, 1976).

Conversely, an increase in hand temperature is brought about by vasodilatation. Skin temperature may be influenced by several other factors. These factors include climate, blood viscosity, skin condition, and the presence or absence of certain chemicals in the blood (Autogenic Systems, 1976).

Summary

The review of literature has outlined the theory of wound healing (Hunt & Van Winkle, 1976) and methods of measuring temperature variations of burns. The theory of wound healing (Hunt & Van Winkle, 1976) has five phases: (a) coagulation, (b) complement activity, (c) chemotaxis, (d) circulation, and (e) collagen. Literature has been cited to support all of the phases of wound healing. The two methods of measuring temperature variations of burns (Thermography and Biofeedback Peripheral Skin Temperature Monitor) have been upheld by literature cited.

CHAPTER 3

PROCEDURE FOR COLLECTION AND TREATMENT OF DATA

The type of study in this investigation was a descriptive, exploratory study. Polit and Hungler (1978) defined a descriptive study as an investigation attempting to describe some type of phenomenon and a careful analysis of a situation that may reveal relevant factors or relationships which are undetected. A descriptive investigation begins with the identification of a problem and the researcher observes, describes, and perhaps classifies some type of phenomenon (Polit & Hungler, 1978).

In this study, the researcher attempted to describe the peripheral skin temperature changes that occur during the healing process of deep partial-thickness and full-thickness burns. A repeated measures design was utilized in this investigation. Polit and Hungler (1978) indicated that a technique which utilizes the subjects as their own controls and the subjects being exposed to more than one

condition as a repeated measures design. The burned subjects in this investigation did have peripheral skin temperatures of a nonburned area as their control. Peripheral skin temperatures were measured on deep partial-thickness burns, full-thickness burns, and analogous nonburned areas, every 12 hours times 3 days and 1 time a day thereafter until the final grafting procedure (coverage of the wound) or discharge.

The treatment of data in this study was simple linear regression analysis of the peripheral skin temperature difference scores between burned areas (deep partial-thickness and full-thickness) and analogous nonburned areas. A natural log linear regression analysis of the temperature difference scores between deep partial-thickness burns and analogous nonburned areas was performed on the first four subjects. A *t*-test was performed to compare the temperature difference scores of a deep partial-thickness burn and analogous nonburned area, to a full-thickness burn and analogous nonburned area.

Setting

An adult burn center at a large county hospital located in a metropolitan area was utilized as the setting in this study. This county hospital has a total bed capacity of 850 beds. The adult burn center has a 4-bed acute burn intensive care unit and a 14-bed burn intensive care unit.

Population and Sample

The study had an accessible population defined by Polit and Hungler (1978) as a conglomerate of cases that conform to specific criteria and is accessible to the researcher. The accessible population of this investigation did meet the criteria of burn victims with deep partial-thickness burn and/or full-thickness burn with an analogous nonburned area, at the adult burn center.

The type of sample appropriate for this investigation was a purposive sample. This study did have a purposive sample specified by Polit and Hungler (1978) as the researcher having knowledge about the population and its elements so that subjects can be specifically selected to be included in the sample. This purposive sample was comprised of burned patients having the

following delimitations: (a) 60% or less than total body surface area burns, (b) deep partial and/or full-thickness burns, (c) between the ages of 16 and 65 years, (d) no concomitant peripheral vascular or cardiovascular diseases, injuries, or chronic illnesses.

Protection of Human Subjects

Permission to conduct the study was obtained from the Texas Woman's University Human Subjects Review Committee (Appendix A) and from the graduate school (Appendix B). Written permission was also obtained from the participating agency (Appendix C).

The subjects participating in this investigation remained anonymous and their names do not appear in the written report of this study. A code was utilized to indicate identification of subjects. All information obtained was kept by the investigator in a locked desk and will be destroyed 1 year after the study. Subjects' names were not used except on consent forms. The consent forms (Appendix D) were kept separate from the data sheet (Appendix E), temperature chart (Appendix F), and body mapping chart (Appendix G).

The investigator applied Betadine solution to the thermistors to prevent cross contamination of the burned wound. If cross contamination occurred, which was indicated by the wound culture reports, monitoring was discontinued.

During dressing changes all burn patients experienced some discomfort. If the subjects exhibited undue discomfort during the measuring of their peripheral skin temperature readings, monitoring was discontinued.

Instruments

The instrument that was utilized in this investigation was the Autogen 1000 Peripheral Skin Temperature Biofeedback Monitor. This instrument is a battery powered electronic multiprob thermometer with integrated circuits for auditory and visual display of absolute external body temperature. Three thermistors were utilized in this study, which are three yellow-springs research grade thermistors.

The validity of the Autogen 1000 Peripheral Skin Temperature Monitor is that it registers the temperature measure accurately to a 1/20 of a degree in Fahrenheit (Autogneic Systems, 1976). The reliability

is .05 degree Fahrenheit. The Autogen 2000 Peripheral Skin Temperature Monitor has battery checks to ensure proper operations (Autogenic Systems, 1976).

A body mapping chart was utilized in this study to designate where the burns were located on the body and the sites of temperature recordings. On the body mapping chart, an "X" mark was used for full-thickness burns, a circle for deep partial-thickness burns, and a square designated the placement of the thermistor on the nonburned area.

A data base sheet utilized in this study contained demographic data, percentage of burn, date of admittance, cause of burn, date of discharge, medications, and date of grafting. A chart for recording the temperature was utilized in this investigation. On the Y axis of the chart was the days postburn and on the X axis of the chart was the temperature of burns (deep partial-thickness and full-thickness), the nonburned area, the culture reports, the ambient room temperature, and body temperature (oral or rectal).

Data Collection

Each patient admitted to the adult burn center was assessed for meeting the criteria of subjects in

this investigation. The subjects met the specified delimitations. The demographic data, percentage of burn, cause of burn, and date of admittance was recorded on each subject. Demographic data such as age, sex, and race were collected to determine if there was a differentiation in peripheral skin temperatures according to sex, age, and race.

The first peripheral skin temperature readings were obtained within 48 hours postburn. The peripheral skin temperature readings consisted of absolute temperatures of a burned area (deep partial-thickness and/or full-thickness burns) and an analogous nonburned area of the body. The peripheral skin temperature readings on the nonburned area were the controls for this study.

Betadine solution was applied to the thermistors before and after readings had been taken. Betadine is an antiseptic, germicidal solution that is effective against gram positive and negative microorganisms (Baker, 1979). This solution was applied to thermistors as a sterilization procedure to prevent contamination of the burn wounds. Gauze was utilized to keep the thermistor in place while the temperatures were

being registered. This procedure takes approximately 30 seconds.

After the first peripheral skin temperature readings had been obtained, the investigator obtained peripheral skin temperature readings every 12 hours for 3 days. The peripheral skin temperature readings were obtained every 12 hours for 3 days because so many physiological reactions are occurring during this time period (e.g., erythrocytes aggregate, sludging due to sequestration of extracellular fluid, leukocytes adhering to intima of vessels, and the forming of thrombi).

After the third day of recording peripheral skin temperatures every 12 hours, the researcher obtained readings once per day at the same time every day. These daily peripheral skin temperature recordings were collected until the patient's wound was adequately covered with grafts or the patient was discharged from the hospital.

The ambient room temperature was recorded prior to the peripheral skin temperature readings. This ambient room temperature was measured by placing one of the thermistors above the patient and a recording

was collected. To measure this extraneous variable of ambient room temperature, the recordings were obtained prior to collection of the peripheral skin temperature data.

Wound culture reports are done every other day in the adult burn center. The wound culture reports were recorded to identify the presence of microorganisms in the burned wound. A burn wound culture of 10^5 or above will indicate burn wound sepsis.

Treatment of Data

There were three statistical tests appropriate for this study. First, a simple linear regression analysis of the peripheral skin temperature difference scores of deep partial-thickness and full-thickness burns and analogous nonburned area within subjects was performed to study the relationship of the independent and dependent variables. The independent variable was the days postburn and the dependent variable was the skin temperature difference score between the burned area (deep partial-thickness or full-thickness) and an analogous nonburned area on each subject.

Linear regression analysis is a statistical procedure for determining a straight line to fit the data in such a way that the deviations from the line are minimized (Polit & Hungler, 1978). This technique lent itself well to examining for an underlying relationship between temperature difference of scores and time (days postburn).

The second statistical test was a natural log linear regression analysis of the temperature difference scores between deep partial-thickness burns and analogous nonburned areas, which was performed on the first four subjects with deep partial-thickness burns. Simple linear regression analysis assumes an underlying relationship among variables which is linear and additive. When examination of the scatterplot suggests deviation from linearity as in this study, then the relationship can be restated in a linear form by transforming the original variables. The most commonly used is the natural log transformation (Nie, Hull, Jenkins, Steinbrenner, & Bent, 1975). Because of the curvilinearity of data in this study, such a natural log regression analysis was performed.

To compare the temperature difference scores between a deep partial-thickness burn and an analogous nonburned area to a full-thickness burn and an analogous nonburned area, a *t*-test was performed. The basic parametric procedure for testing significant differences of group means is the *t*-test (Polit & Hungler, 1978). The *t*-test was used in this study to examine differences in mean scores.

CHAPTER 4

ANALYSIS OF DATA

In this chapter a description of the sample is given. Figures are utilized to indicate the temperature differences of deep partial-thickness burns and analogous nonburned areas in each subject and temperature differences in full-thickness burns and analogous nonburned areas in each subject. A figure is also utilized to demonstrate the regression curve. Analysis of data is presented with the temperature difference between deep partial-thickness burns and analogous nonburned areas in each subject, and the temperature difference between full-thickness burns and analogous nonburned areas of each subject.

Description of Sample

In this investigation the sample consisted of seven Caucasian males who were admitted to the adult burn unit in a large county hospital. The percentage of total body surface area burns (TBSA) was from 57% TBSA burns to 12% TBSA burns. Each subject had deep partial-thickness and/or full-thickness burns and

analogous nonburned areas. The age range was from 21 to 51 years of age. The cause of burns was due to flame. The subjects had no concomitant peripheral vascular or cardiovascular diseases, injuries, or chronic illnesses. Following is a description of each subject.

Subject #1 was a 27-year-old, Caucasian male with deep partial-thickness burns to the face, neck, back, and extremities totaling 53% TBSA burns. This subject was hospitalized for 15 days and was discharged on day 16 postburn without grafting.

Subject #2 was a 51-year-old, Caucasian male with 38% TBSA deep partial-thickness burns to the face, back, right upper extremity, left forearm, left hand, and right upper aspect of chest. Subject #2 was discharged from the hospital, without grafting, 15 days postburn.

Subject #3 was a 49-year-old, Caucasian male with 17% TBSA burns. The third subject had full-thickness burns to the chest, left lateral neck, left upper arm, and right forearm, which were grafted 8 days postburn. This subject sustained deep partial-thickness burns to the hands and right anterior thigh.

Subject #4 was a 37-year-old, Caucasian male with 15% TBSA deep partial-thickness burns to the face, left shoulder, and hands. This subject was discharged, without grafting, on day 5 postburn.

Subject #5 was a 23-year-old, Caucasian male with 22% TBSA burns, which were deep partial-thickness on the chest and face, and full-thickness on the upper extremities and right shoulder. Grafting was done on this subject's upper extremities and right shoulder, 3 days postburn.

Subject #6 was a 21-year-old, Caucasian male with 20% TBSA burns. This subject sustained full-thickness burns to the right axilla and right upper arm and deep partial-thickness burns to the right lateral chest, right forearm, and left hand. Grafts were placed on this subject's right axilla and right upper arm 5 days postburn.

Subject #7 was a 26-year-old, Caucasian male who had 12% TBSA deep partial-thickness burns to the right lateral abdominal area, right upper extremity, and left hand and forearm. This subject was discharged, without grafting, 9 days postburn.

Findings

Temperature differences between burned areas and analogous nonburned areas were derived by subtracting the peripheral skin temperatures (in degrees Fahrenheit) of the nonburned areas from the peripheral skin temperatures (in degrees Fahrenheit) of the burned areas of each patient. Table 1 shows the temperature difference between deep partial-thickness burns and analogous nonburned areas of each subject, and the corresponding days postburn. There was an unequal number of table entries for each subject due to early discharges or because the sites at which temperature readings were taken and covered with the surgical dressing after grafting full-thickness burns in close proximity. The greatest temperature difference was recorded around day 3.5 to day 4. In several subjects, the temperature difference became noticeably smaller after the greatest temperature difference recorded. This temperature difference drop was especially noticeable in Subjects 1, 2, and 3.

Figures 1, 2, and 3 indicate the temperature difference (in degrees Fahrenheit) between deep partial-thickness burns and analogous nonburned areas

Table 1

Temperature Difference of Deep-Partial Thickness Burns and Nonburned Area Subjects

Days Postburn	Subjects						
	#1	#2	#3	#4	#5	#6	#7
1.5			6.2	4.4	.4	3.0	1.7
2			3.8	4.7	6.3	1.4	2.7
2.5	.6	8.2	5.2	1.8	6.0		1.7
3	3.4	.8	6.5	5.1	-1.7		2.8
3.5	20.6	11.1	1.3	.6			2.0
4	10.3	7.8	11.3	6.1			3.8
4.5	7.7	04.4	1.2				
5	3.6	-.2	7.8				3.0
6	12.5	1.5	7.1				2.9
6.5	3.0	-1.5					
7	0	2.4	4.6				1.4
8	-2.3	-3.0					
9	.4	.1					

Table 1--(Continued)

Days Postburn	Subjects						
	#1	#2	#3	#4	#5	#6	#7
10	1.2	1.6					
11	.8	.1					
12	-.3	8.4					
13	1.5	2.5					
14	.6	1.0					
15	.5						

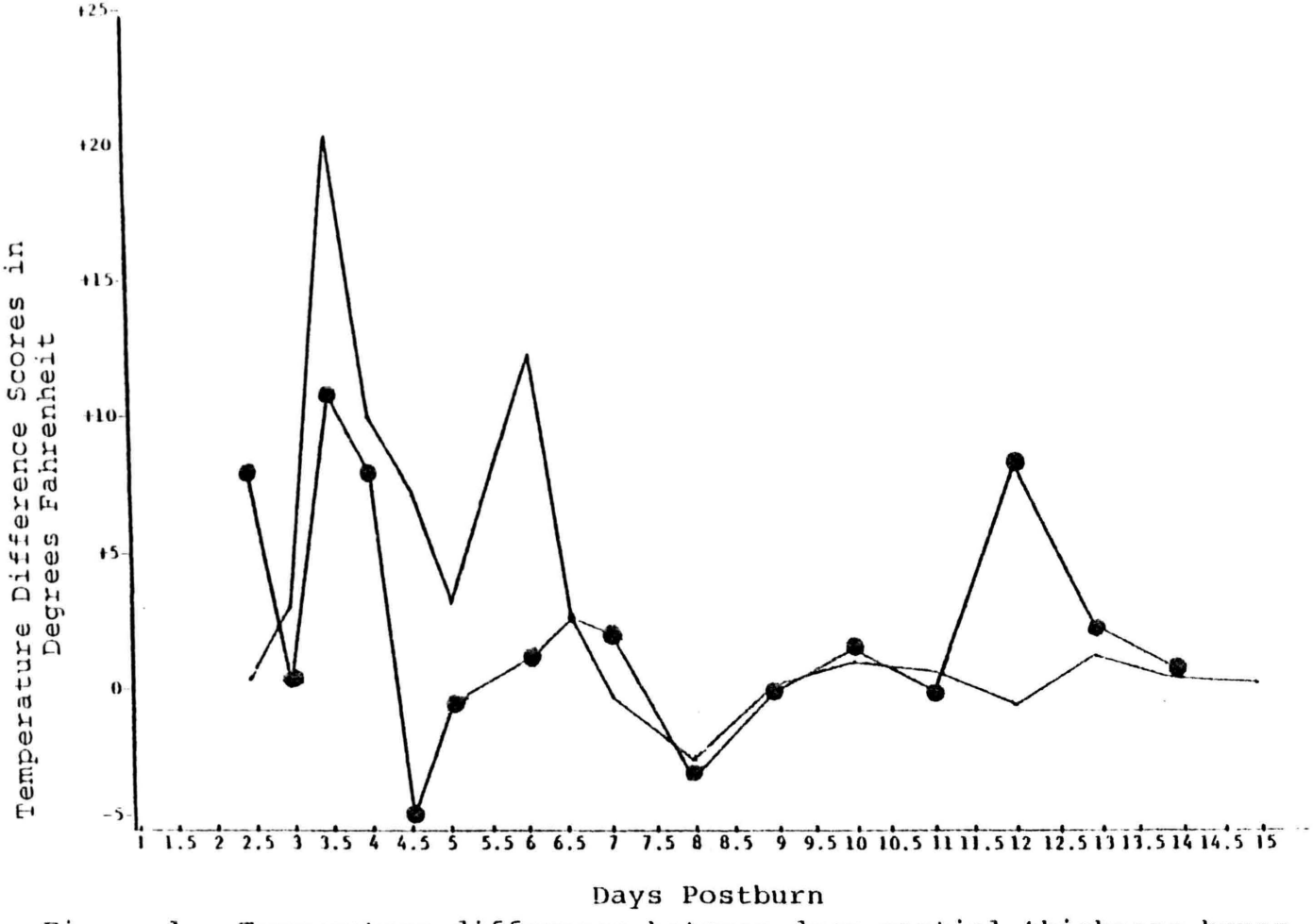


Figure 1. Temperature difference between deep partial-thickness burns and nonburn areas.

Subject #1 _____ Subject #2 —●—●—●—

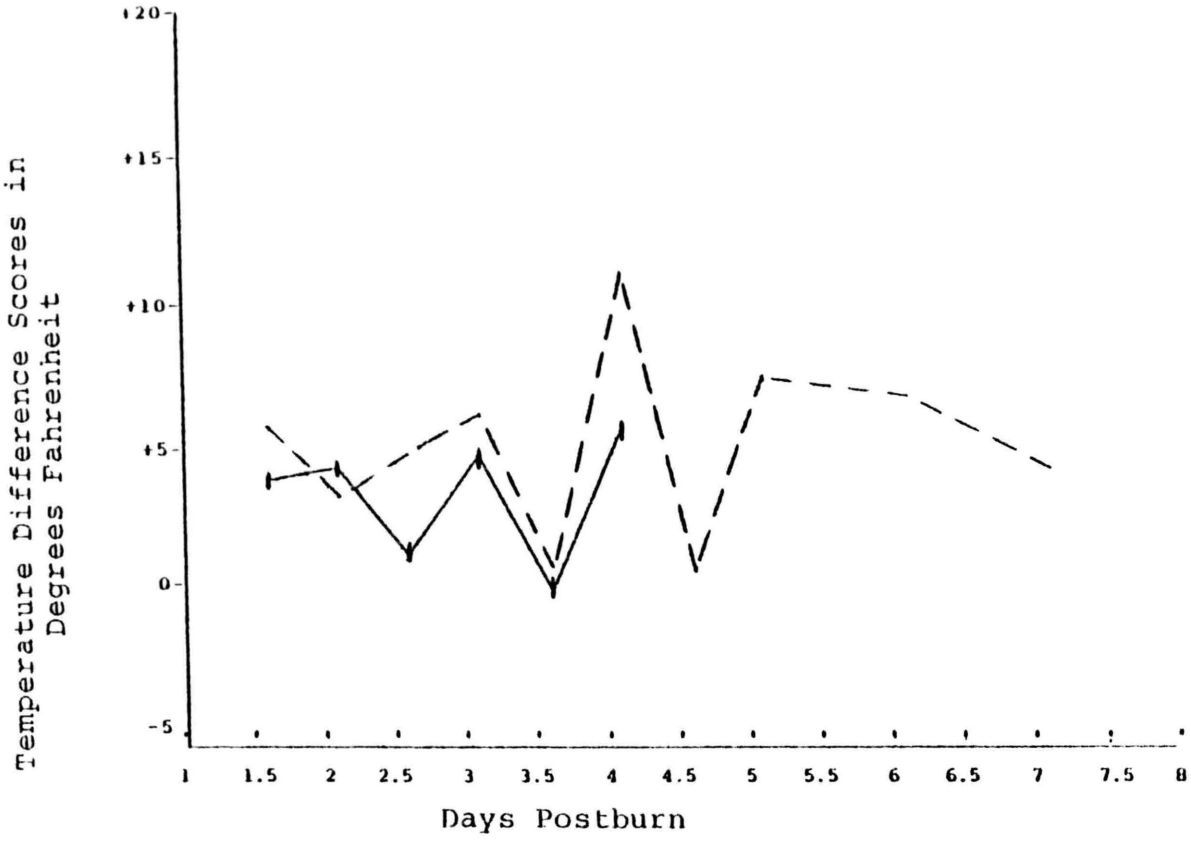


Figure 2. Temperature difference between deep partial-thickness burns and nonburned areas.

Subject #3 ----------

Subject #4 —/—/—/—

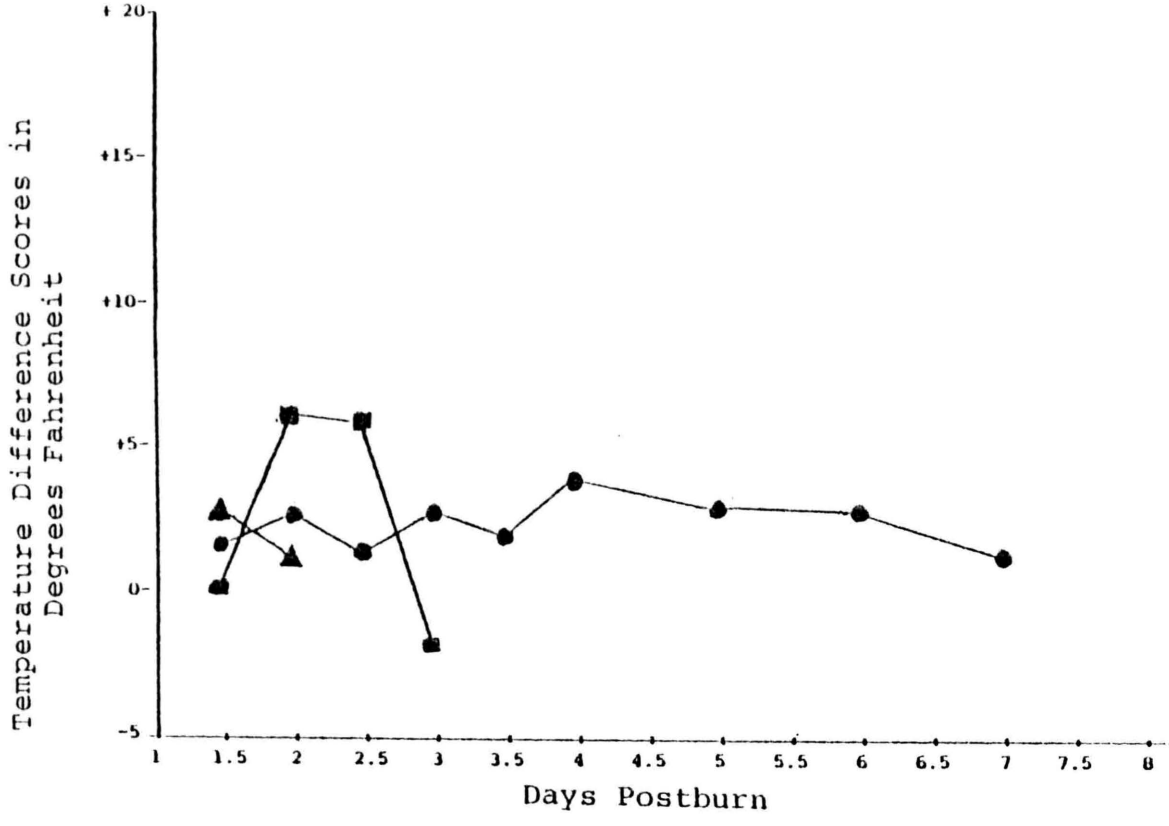


Figure 3. Temperature difference between deep partial-thickness burns and nonburned areas.

Subject #5

Subject #6

Subject #7

of each subject over time. There was a similarity of Subjects 1 and 2 and of Subjects 3 and 4. The temperature difference of Subjects 1 and 2 sharply increased on day 3.5, but by day 4, there was a drop in temperature difference. Subjects 3 and 4 had a rise in temperature difference on day 4. Subjects 5, 6, and 7 were eliminated from further analysis because of too few data points.

Table 2 presents the means and standard deviations of temperature difference scores between deep partial-thickness burns and analogous nonburned areas for the first four subjects over time. The highest mean temperature difference score was on days 3.5 and 4. There was a 7.4 degree mean drop in temperature difference from day 4 to day 4.5. There was a rise of the mean temperature difference in day 6 and a 6.2 mean drop from day 6 to day 6.5.

Temperature difference scores over time for deep partial-thickness and full-thickness burns were subjected to simple linear regression analysis, the results which can be seen in Table 3 and Table 4, respectively. Only one of the generated regression equations reached statistical significance, suggesting

Table 2

Means and Standard Deviations of Skin Temperature Difference Scores between Deep Partial-Thickness Burns and Analogous Nonburned Areas Over Time

Days Postburn	Mean	SD
1.5	5.3	1.27
2	4.3	3.13
2.5	3.5	2.40
3	4.0	2.45
3.5	8.4	9.44
4	8.9	2.40
4.5	1.5	6.06
5	3.7	4.00
6	7.0	5.50
6.5	.8	3.18
7	2.3	2.30
8	-2.7	.50
9	.3	.21
10	1.4	.28
11	.5	.50
12	4.1	6.15
13	2.0	.71
14	.8	.28

Table 3

Simple Linear Regression Analysis of Deep Partial-Thickness Burns

Subject #	Pearson *r*	Level of Significance
1	-0.508	$p < .0375$
2	-0.314	$p < .2370$
3	0.048	$p < .8953$
4	-0.038	$p < .9431$
5	-0.046	$p < .9539$
6	-1.000	$p < .0$*
7	-0.000	$p < .9992$

*Spurious due to two data points.

Table 4

Simple Linear Regression Analysis of Full-Thickness Burns

Subject #	Pearson *r*	Level of Significance
3	0.507	$p < .1349$
5	-0.320	$p < .5997$
6	-0.170	$p < .7477$

a linear relationship. Consequently, the next step in data analysis, the test for equality of lines (a statistical comparison of the slopes and intercepts of significant regression lines) proved to be unnecessary. A closer look at the data, however, suggested curvilinearity. Consequently, a natural log transformation of grouped temperature difference scores was completed and regression analysis of this transformed data was done. The log of mean temperature difference scores constituted the dependent variable and time (in days) the independent variable.

The group mean temperature difference in the first four subjects with deep partial-thickness burns is plotted in Figure 4. Also, in Figure 4 is the calculated regression curve for the data. This regression curve was obtained by taking the log of the mean temperature difference as the dependent variable and the day as the independent variable. Note that in the first days after the burn the temperature difference drops by one-half degree per day. According to the curve, there would be no temperature difference after day 15. This regression curve (Table 3) had a Pearson $r = -0.295$, $p < .0394$, thereby achieving statistical

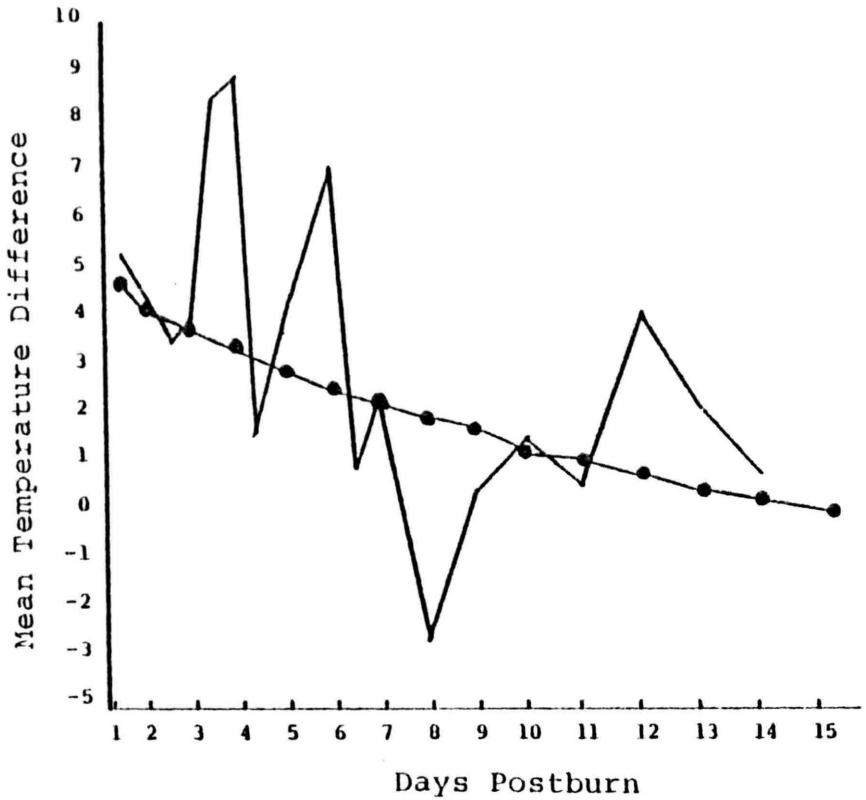


Figure 4. Average of temperature difference of Subjects 1, 2, 3, and 4 and regression curve.

Regression Curve —●—●—●—●—

Mean Temperature Difference _____

significance, but it indicates a weak and inverse relationship.

Table 5 represents the subjects with full-thickness burns, indicating temperature difference between full-thickness burns and analogous nonburned areas, and the corresponding days. The temperatures of full-thickness burns were not statistically significant from the temperatures of the analogous nonburned area. The grafting schedule was a confounding variable which limited the number of temperature readings obtained. Some of the subjects that had suspected full-thickness burns were not grafted so, therefore, the burns were deep partial-thickness. This eliminated a few subjects and caused a lack of sufficient number of subjects with full-thickness burns for additional regression analysis. Figure 5 has the temperature difference of full-thickness burns and analogous nonburned areas and the corresponding days plotted.

A *t*-test between sequential temperature difference scores over time of a full-thickness burn to an analogous nonburned area and temperature differences of a deep partial-thickness burn to an analogous nonburned area in the one subject whose grafting schedule did not

Table 5

Temperature Difference of Full-Thickness Burns and Nonburned Areas

Days Postburn	Subject # 3	5	6
1.5	-1.8	+ .8	- .5
2	- .7	+2.8	+ .4
2.5	+1.0	+6.8	-2.1
3	- .4	-1.5	- .5
3.5	- .8		-1.6
4	- .3		
4.5	- .3		
5	- .9		
6	+4.2		
7	+ .1		

prevent this comparison, was significant at the .001 level ($t = 5.915(9)$, $p < .001$). This highly significant finding would suggest that within-subject temperature difference score comparisons may be a fruitful area of study in the future, to diagnose the degree of burn.

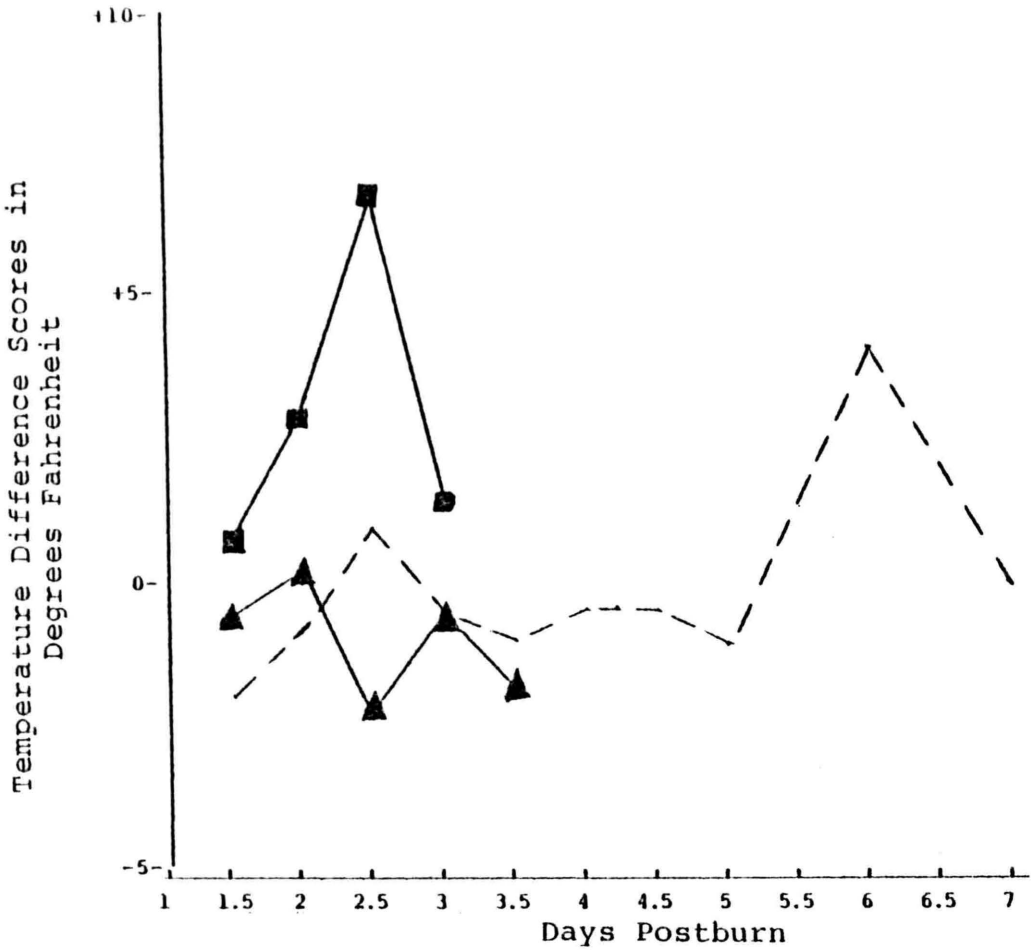


Figure 5. Temperature difference of full-thickness burns and nonburned areas.

Subject #3----------
Subject #5
Subject #6

Summary of Findings

The major findings of this study were as follows:

1. The temperature difference between deep partial-thickness burns and analogous nonburned areas in the same subject peaked at around day 3.5 to day 4 postburn and then decreased approximately 7 degrees Fahrenheit.

2. A regression curve indicated that the temperature difference between deep partial-thickness burns and analogous nonburned areas in the same subject, after reaching a maximum temperature, dropped an average of one-third degree Fahrenheit each day.

3. Full-thickness burns had a temperature lower than or equal to the temperature of analogous nonburned areas in the same subject.

4. There was a greater temperature difference in a deep partial-thickness than in a full-thickness burn when compared to analogous nonburned areas in the same subject.

CHAPTER 5

SUMMARY OF THE STUDY

In this study the following research question was investigated: What is the nature and course of temperature changes over time in the healing of deep partial-thickness and full-thickness burns as measured by a peripheral skin temperature monitor? The peripheral skin temperature is a reflection of vascular and/or metabolic alterations.

Summary

Seven burn patients, at a burn center, with the diagnosis of deep partial-thickness and/or full-thickness burns due to flame comprised the sample in this study. The first peripheral skin temperature readings were obtained within 48 hours postburn. The peripheral skin temperature readings consisted of absolute temperatures of burned areas (deep partial-thickness and/or full-thickness burns) and analogous nonburned areas. The peripheral skin temperature readings on the nonburned areas were the controls for this study.

Betadine solution was applied to the thermistors before and after readings had been taken to prevent contamination of the burn wounds. Gauze was utilized to keep the thermistors in place while the temperatures were being registered, which took 30 seconds.

After the first peripheral skin temperature readings were obtained, the investigator obtained peripheral skin temperature readings every 12 hours for 3 days. After the third day of recording peripheral skin temperatures every 12 hours, this researcher obtained readings once per day at the same time every day, until the subjects' wounds were grafted or subjects' were discharged from the hospital.

The ambient room temperatures and the subjects' body core temperatures were recorded, along with the peripheral skin temperature readings. Wound culture reports were recorded to identify the presence of microorganisms in the burn wounds.

Discussion of Findings

At the onset it is important to underscore that the following interpretations of the results of this investigation are limited by the small sample size and

small number of data points common to many clinical exploratory studies.

When a regression analysis was done on each subject's deep partial-thickness and full-thickness burn temperature difference scores, there were no significant results. But when a regression analysis was performed on the natural log transformation of the first four subjects' deep partial-thickness burn temperature difference scores, it was statistically significant with a Pearson $r = -0.295$ at the .04 level of significance, which is a weak inverse relationship. The raw data indicated that deep partial-thickness burns had the highest temperature the first day postburn. The temperature dropped an average of one-third of a degree in Fahrenheit each consecutive day, until day 15. On day 15, the temperatures of the deep partial-thickness burns were the same as the analogous nonburned areas. Here the statistical findings may indicate that deep partial-thickness burns had increased vascularity and this vascularity decreased daily until day 15, when deep partial-thickness burns were healed and had normal vascularity. These results support the findings of Aulick et al. (1977).

The initial increase in peripheral skin temperature of deep partial-thickness burns indicated that the burns were not severe enough to produce pronounced coagulation and sludging of blood flow to the burned area. Sevitt (1957) stated that erythema developed in burn wounds within a few minutes after injury. This erythematous area is due to dilatation of capillaries and arterioles. Blood flow through the burned area is increased and local skin temperature rises 1 to 2 degrees centigrade (Sevitt, 1957).

In partial-thickness burns, Sevitt (1957) avowed that stasis was not through the whole depth of skin and the blood flow in deep dermis will return to normal and epithelialization will take place. Order and Moncrief (1964) declared that deep partial-thickness burn wound injury does not have the marked vascular loss in the immediate postburn period. Twenty-four and 48 hours postinjury of deep partial-thickness burns, more of the subpapillary plexus was intact and there was no period of complete devitalization of the entire wound (Order & Moncrief, 1964).

Order and Moncrief (1964) further showed that deep partial-thickness burns, 1 week postburn, exhibited

revascularization accomplished by reestablishment of patency in the arterial plexus. Peacock and Van Winkle (1976) claimed that in injured tissues there is a stoppage of lymphatic drainage, local vasodilatation, and leakage of fluid into extravascular space producing the classic signs of inflammation which are: redness, swelling, and heat. This data support the increased peripheral skin temperatures of deep partial-thickness burns found in this investigation.

Chao et al. (1977) affirmed that the exudate in deep partial-thickness burns had a heat-dissipating barrier. This exudate resulted in a higher skin temperature at the deep partial-thickness burn site. As the healing surfaces became dry, the temperatures were beginning to drop to normal values (Chao et al., 1977). This supported the findings in this investigation.

Birch et al. (1968) utilized infrared thermography to diagnose burns. In deep partial-thickness burns, infrared thermography was able to detect heat from underlying patent vessels (Birch et al., 1968).

In the thermogram studies investigated by Mladick et al. (1966), and Winsor (1971), deep partial-thickness

burns were 2 degrees centigrade cooler than normal skin. For an accurate temperature recording with thermography, the subjects have to be placed in a 72 degree Fahrenheit room for 20 minutes prior to thermographic examination. This cooling of burn subjects may be the reason for the decreased temperature in deep partial-thickness burns, which was contrary to the findings in the present investigation.

Unfortunately, there was a small sample of full-thickness burns. There were too few data points to be grouped together to obtain a regression analysis of the log transformations of mean scores. The number of temperature recordings of full-thickness burns was limited by the grafting schedule and several suspected full-thickness burns were not grafted, therefore, these burns were post hoc diagnosed as deep partial-thickness burns.

Most of the peripheral skin temperature readings of full-thickness burns were cooler than analogous nonburned areas in the same subject, as shown in Figure 5 of Chapter 4, but lack of data points precludes any conclusions other than suggestive consideration. These cooler peripheral skin temperature readings of

full-thickness burns are supported by the following research.

Decreased peripheral skin temperature is due to decreased vascular flow to the area (Autogenic Systems, 1976). Sevitt (1957) claimed that in severe burns there is stasis of blood flow, due to increased capillary resistance by concentration of erythrocytes and exudation of fluid from capillaries.

Order and Moncrief (1964) affirmed that immediately following a full-thickness burn, most of the subpapillary plexus is occluded. Twenty-four hours postburn, the entire vascular architecture lying beneath the burn wound is occluded and devitalization is complete. The vascular architecture remains unchanged 48 hours postburn. One week postburn, the full-thickness burns remained avascular (Order & Moncrief, 1964).

Schoen et al. (1971) further showed that full-thickness burns, caused by flame, had increased blood viscosity due to the effects of increased hematocrit and fibrinogen. This increased blood viscosity contributed to the mircocirculatory impairment by an increased resistance to blood flow through the microvasculature (Schoen et al., 1971).

The loss of capillary fluids into the tissues, the homoconcentration of the passing blood, and final stuffing and impaction of small vessels with sludge of full-thickness burns caused a lower peripheral skin temperature of the burns than the analogous nonburned areas, that was evident in the present investigation. The thermographic studies, also, asserted that full-thickness burns are colder than normal skin and deep partial-thickness burns.

Mladick et al. (1966) affirmed that full-thickness burns were colder than normal skin and appeared as black areas on thermograms. The full-thickness burns remained as cold spots 1 week postburn (Mladick et al., 1966).

Birch et al. (1968) revealed that infrared thermography was able to detect thrombosing of the large central vessels of full-thickness burns. Buwalda (1969) fostered that with thermography the beginning of disturbance in circulation can be detected and treatment can be instituted accordingly.

Winsor (1971) claimed that deep partial-thickness burns were 2 degrees centigrade cooler than normal skin. Full-thickness burns were even colder by being

greater than 2 degrees centigrade colder than normal skin, as thown by thermography examiantion (Winsor, 1971). Aulick et al. (1977) also mentioned that third degree wounds, which are associated with superficial vascular thrombosis, leg blood flow is near control levels shortly after injury.

The t-test comparing the temperature difference scores of a deep partial-thickness burn and an analogous nonburned area to a full-thickness burn and an analogous nonburned area was significant with a $T = 5.915(9)$ at the .001 level. This suggests that the full-thickness burn has less of a temperature difference from an analogous nonburned area than the deep partial-thickness burn. A full-thickness burn has pronounced coagulation and a deep partial-thickness burn does not, which is supported by the findings of this t-test and previously mentioned literature.

Conclusions and Implications

As shown by the curvilinear regression line in Figure 4 of Chapter 4, the temperature difference between deep partial-thickness burns and analogous nonburned areas was greatest on day 1. This temperature difference decreased by approximately one-third of a degree

Fahrenheit daily until day 15 when there was no temperature difference. This data suggest that deep partial-thickness burns do not have vascular impairment. In fact, there was probably an initial increase in vascularity (dilated capillaries and arterioles) in the deep partial-thickness burns, which accounts for the greatest temperature difference in comparison to analogous nonburned areas on day 1 postburn. The data suggest that by day 15 postburn, the deep partial-thickness burns were healed and the vascularity in the healed burn wounds was the same as in normal skin. Generally speaking, the observed results are consistent with previous literature regarding the physiology of burn wounds. The investigator contends that the curvilinear regression line (Figure 4, Chapter 4) probably suggests increased vascularity in deep partial-thickness burns, then progressive lessening of vascularity to normal.

A similar regression analysis could not be done on full-thickness burns because there were too few data points. But most of the peripheral skin temperature readings of full-thickness burns were cooler than analogous nonburned areas (Figure 5, Chapter 4). These

cooler temperatures of full-thickness burns were possibly due to the sludging and stasis of vascular flow to the wounds. In full-thickness burns there is increased blood viscosity by increased hematocrit and fibrinogen leukocytes adhering to the intima of vessels, clumping of erythrocytes, and sequestration of fluids, all of which cause stasis of blood flow. The suggested decreased vascularity to full-thickness burns appeared to cause lower peripheral skin temperature readings than in analogous nonburned areas in the same subjects, a phenomenon cited by previous research about the physiology of burn wounds.

The coagulation phase of the theory of wound healing (Hunt & Van Winkle, 1976) was consistent with the theory suggested in full-thickness burns of this investigation. Coagulation of the blood vessels was indicated in this investigation by lower peripheral skin temperatures of full-thickness burns than analogous nonburned areas.

The first three phases (coagulation, complement activity, and chemotaxis) of Hunt and Van Winkle's (1976) theory of wound healing were not evident in the deep partial-thickness burns. The circulation and

collagen phases of the wound healing theory (Hunt & Van Winkle, 1976) were supported in this investigation. In the deep partial-thickness burns, there were patent vasculature indicated by higher peripheral skin temperature differences (Figure 5, Chapter 4) initially. In the collagen phase, granulation tissue has a reduction in vascularity. As the deep partial-thickness burns were healing more granulation tissue is produced and this was supported by the decreasing peripheral skin temperature difference each day (Figure 5, Chapter 4). Hunt and Van Winkle's (1976) theory of wound healing was appropriate for this investigation.

This investigator recorded the wound culture reports of each subject during this study. There was no wound infection produced by taking the peripheral skin temperatures of the wounds. Betadine was an effective antiseptic and did not damage the thermistors. The subjects of this investigation did not complain of pain, while the temperature readings were being taken. Hence, thermistors were an appropriate, painless, and sterile mode of temperature measurement of burn wounds.

This investigation had a small sample size, therefore limiting the generalizability of this study. The

implications of this study are that the Biofeedback Peripheral Skin Temperature Monitor can be used to measure the vascularity of burn wounds without pain to the subject and without contamination of the wound. The Biofeedback Peripheral Skin Temperature Monitor may be used to measure the vascularity of burn wounds instead of thermography. Another implication of this study was that the circulation and collagen phases of Hunt and Van Winkle's (1976) theory of wound healing was supported by the peripheral skin temperature changes of deep partial-thickness burns. The coagulation phase of the wound healing theory (Hunt & Van Winkle, 1976) was supported by the majority of the full-thickness burns' peripheral skin temperatures being cooler than analogous nonburned areas in the same subjects. This suggests that the characteristic of full-thickness burns being cooler than analogous nonburned area may prove in later studies to assist in the diagnosis of degree of burns, to prognosticate the fate of the burn wound and serve as an aid in instituting appropriate treatment and therapy accordingly. This is particularly evident when one compares the temperature difference scores of a deep partial-thickness

burn and analogous nonburned area to a full-thickness burn and analogous nonburned area.

The Biofeedback Peripheral Skin Temperature Monitor (Autogen 1000) can be utilized to assess the vascularity of burn wounds, as thermography has done. But the Biofeedback Peripheral Skin Temperature Monitor is 1/30 of the cost of a thermograph. There was no undue exposure of the subjects of this investigation, while taking the peripheral skin temperature readings with the Biofeedback Peripheral Skin Monitor. Prior to the thermographic examinations the subjects have to be unclothed in a 72 degree Fahrenheit room for 20 minutes. Thermographic examination cannot be done at the bedside. The Biofeedback Peripheral Skin Temperature Monitor is light-weight, can be transported in a suitcase, and the peripheral skin temperatures were taken at the bedside in this investigation. Nurses can monitor burn patients' peripheral skin temperatures with the biofeedback instrument. Additionally, burned patients may be able to increase the vascular flow to an area through biofeedback training with a Biofeedback Peripheral Skin Temperature Monitor.

A study by Bird and Colburne (1980) demonstrated enhanced rehabilitation of an electrical burn patient through thermal biofeedback. The subject was a 22-year-old male who had received severe electrical burns to the left wrist, resulting in coagulation of several flexor tendons and total sensory loss and motor loss of the median and ulnar nerves. Seven months after the injury the subject's skin temperature of the left hand, as compared to the right, was depressed to between 77 and 80 degrees Fahrenheit, resulting in pain and restriction of movement in the injured arm. This subject had 21 sessions of 30-minute peripheral skin temperature biofeedback training utilizing the Autogenic 2000b Feedback Thermometer with visual display. In the last two sessions, the subject performed the same procedure without biofeedback to determine the effectiveness of training. During the ninth training session, the temperature of the injured hand rose above that of the normal hand, a skill that was retrained for the remainder of the sessions. The temperature in the injured hand rose from 77.6 degrees Fahrenheit to 98.8 degrees Fahrenheit, in the twelfth session. The pain had decreased markedly during the

ninth session when the injured hand temperature rose to the 90-93 degrees Fahrenheit range. The subject has been able to recall at will the warming procedure to decrease pain.

Six weeks after Bird and Colburne's (1980) study the subject was to undergo tenolysis of the extensor tendons at the wrist and an abdominal flap skin graft. The attending physician noted a rate regrowth of the nerves, especially the motor components to the deep muscles of the palm of the hand, and found it unnecessary to do a skin graft as the skin had matured sufficiently. There was motor and sensory recovery in the middle, ring, and little fingers but no index finger recovery and hypesthesia in the thumb.

Recommendations for Further Study

For further investigations, the researcher replicating this study should take more frequent peripheral skin temperature readings and/or take peripheral skin temperature readings at more sites on the burn wound, especially full-thickness burns. This would increase the number of readings and data points for statistical analysis. This is especially important for full-thickness burns because these burns are grafted early

postburn. The researcher should, also, have a larger sample size.

Utilizing the Biofeedback Peripheral Skin Temperature Monitor, burned patients may be trained to increase the vascular flow to the injured area, in the circulatory and collagen phases, to enhance healing and decrease pain as it was done in Bird and Colburne's (1980) study. Measurement of peripheral skin temperature of burn wounds may hold promise for appraising vascularity in the healing process, the early differentiation of deep partial-thickness and full-thickness burns, and prognosticating the fate of burn wounds.

Another area of future study would be to establish the difference between a peripheral skin temperature monitor and a thermograph in assessing the vascularity of burn wounds. Also, a determination should be made if there would be any discrepancies in infection rate and reported discomfort level in the use of the two procedures.

APPENDIX A

TEXAS WOMAN'S UNIVERSITY
Box 23717, TWU Station
Denton, Texas 76204

1810 Inwood Road
Dallas Inwood Campus

HUMAN SUBJECTS REVIEW COMMITTEE

Name of Investigator: M'Lou Barnett Staats Center: Dallas

Address: 1810 Inwood Road #426 Date: 6/17/80

Dallas, Texas 75235

Dear Ms. Staats:

Your study entitled Peripheral Skin Temperature Changes of Burn Wounds

has been reviewed by a committee of the Human Subjects Review Committee and it appears to meet our requirements in regard to protection of the individual's rights.

Please be reminded that both the University and the Department of Health, Education, and Welfare regulations typically require that signatures indicating informed consent be obtained from all human subjects in your studies. These are to be filed with the Human Subjects Review Committee. Any exception to this requirement is noted below. Furthermore, according to DHEW regulations, another review by the Committee is required if your project changes.

Any special provisions pertaining to your study are noted below:

____ Add to informed consent form: No medical service or compensation is provided to subjects by the University as a result of injury from participation in research.

____ Add to informed consent form: I UNDERSTAND THAT THE RETURN OF MY QUESTIONNAIRE CONSTITUTES MY INFORMED CONSENT TO ACT AS A SUBJECT IN THIS RESEARCH.

____The filing of signatures of subjects with the Human Subjects Review Committee is not required.

__XX__Other: See below.

____No special provisions apply.

Sincerely,

Estelle D. Kurtz

Chairman, Human Subjects
Review Committee

at Dallas

Please submit a single page to be used as an addendum to your application the answers to the following questions?

1. What steps will you take if contamination occurs and is traced back to the thermistors?

2. What grade are the thermistors - industrial, hobby, mil spec or designed for human environment?

PK/smu/3/7/80

APPENDIX B

TEXAS WOMAN'S UNIVERSITY

DENTON, TEXAS 76204

THE GRADUATE SCHOOL

September 10, 1980

Ms. M'Lou Barnett Staats
1810 Inwood Road
Room 426
Dallas, Texas 75235

Dear Ms. Staats:

I have received and approved the Prospectus for your research project. Best wishes to you in the research and writing of your project.

Sincerely yours,

Robert S. Pawlowski
Provost

RP:dl

cc Dr. Barbara Carper
Dr. Anne Gudmundsen
Graduate Office

APPENDIX C

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING

AGENCY PERMISSION FOR CONDUCTING STUDY*

THE Parkland Memorial Hospital

GRANTS TO M'Lou Barnett Staats
a student enrolled in a program of nursing leading to a Master's Degree at Texas Woman's University, the privilege of its facilities in order to study the following problem.
The peripheral skin temperature changes of burn wounds as they are healing. The burn wounds will be deep partial-thickness and full-thickness burns. The peripheral skin temperatures will be measured by a Autogen 2000 Peripheral Skin Temperature Biofeedback Monitor. A non-burned area will be utilized as a control for each subject.

The conditions mutually agreed upon are as follows:

1. The agency (may) ([redacted]) be identified in the final report.
2. The names of consultative or administrative personnel in the agency (may) ([redacted]) be identified in the final report.
3. The agency ([redacted]) (does not want) a conference with the student when the report is completed.
4. The agency is (willing) ([redacted]) to allow the completed report to be circulated through interlibrary loan.
5. Other [redacted]

Date: June 19, 1980

[redacted]
Signature of Agency Personnel

M'Lou B. Staats
Signature of Student

Beth C Vaughan-Woodall
Signature of Faculty Advisor

*Fill out & sign three copies to be distributed as follows: Original - Student; First copy - Agency; Second copy - TWU College of Nursing.

APPENDIX D

Oral Form B

Hello, I am M'Lou Staats, a graduate student in nursing at Texas Woman's University in Dallas and am conducting research for a thesis. I would like to take the skin temperature of burned wound(s) and a nonburned area as you progressively heal. The temperature readings will be taken every 12 hours for 3 days, then every day thereafter until your wound(s) are covered with grafts.

The health care that you receive will not in any way be affected by your participation, nonparticipation, or withdrawal from the study. An antiseptic, Betadine, will be applied on the thermistors to kill any bacteria and to prevent contamination of your wound(s). If at any time there is undue discomfort while applying the thermistors, I will discontinue the monitoring. No names will be used in the report of this research study. All of the data for this research study will be presented as group data, so you will remain anonymous.

The benefits to be expected are the opportunity to get to participate in the advancement of science for burn patients and to enhance the nursing care of burn patients. Also, we may be able to detect infection at an early stage.

No medical service or compensation will be provided to you by Texas Woman's University as a result of injury from participation in research. I will answer any questions you have concerning the procedures. You are free to withdraw your consent and to discontinue participation in the project at any time.

Consent Form
TEXAS WOMAN'S UNIVERSITY
HUMAN SUBJECTS REVIEW COMMITTEE

(Form B)

Title of Project: Peripheral Skin Temperature Changes of Burn Wounds

Consent to Act as a Subject for Research and Investigation:

I have received an oral description of this study, including a fair explanation of the procedures and their purposes, any associated discomforts or risks, and a description of the possible benefits. An offer has been made to me to answer all questions about the study. I understand that my name will not be used in any release of the data and that I am free to withdraw at any time. I further understand that no medical service or compensation is provided to subjects by the university as a result of injury from participation in research.

_____ _____
Signature Date

_____ _____
Witness Date

Certification by Person Explaining the Study:

This is to certify that I have fully informed and explained to the above named person a description of the listed elements of informed consent.

_____ _____
Signature Date

_____
Position

_____ _____
Witness Date

(Form B) -- Continued

One copy of this form, signed and witnessed, must be given to each subject. A second copy must be retained by the investigator for filing with the Chairman of the Human Subjects Review Committee.

APPENDIX E

Data Base Sheet

Name:

Age:

Sex:

Race:

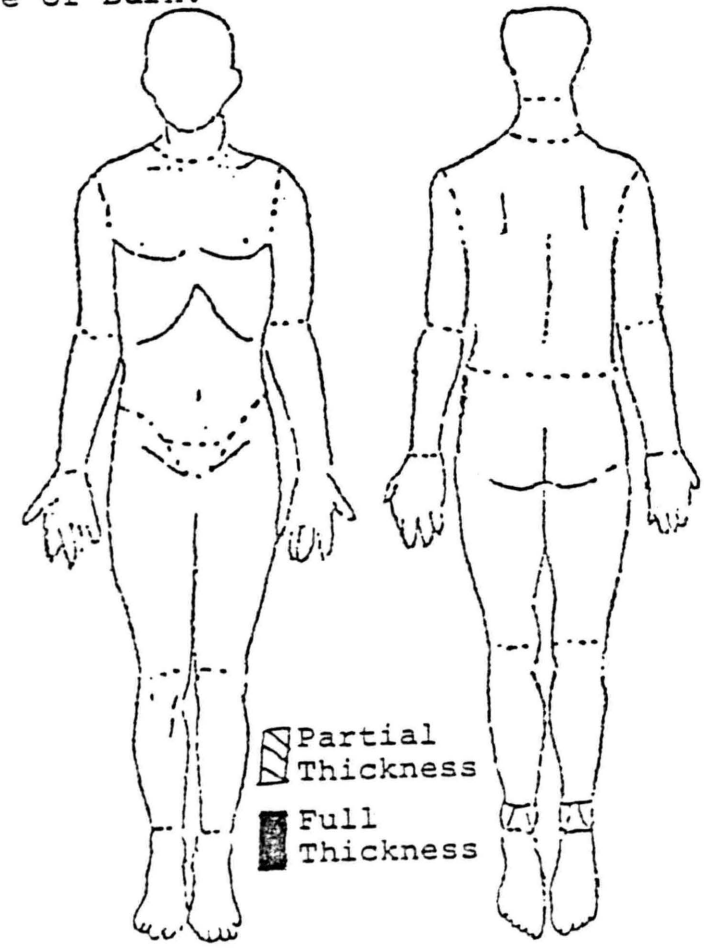
Date of Admittance:

Medications:

Cause of Burn:

Date of Final Grafting:

Percentage of Burn:



APPENDIX F

Temperature Chart

Post-Burn	Post-Grafting	DPT	FT	NBA	Body Temp.	Ambient Temp.	Culture Reports

DPT = Deep partial-thickness burn

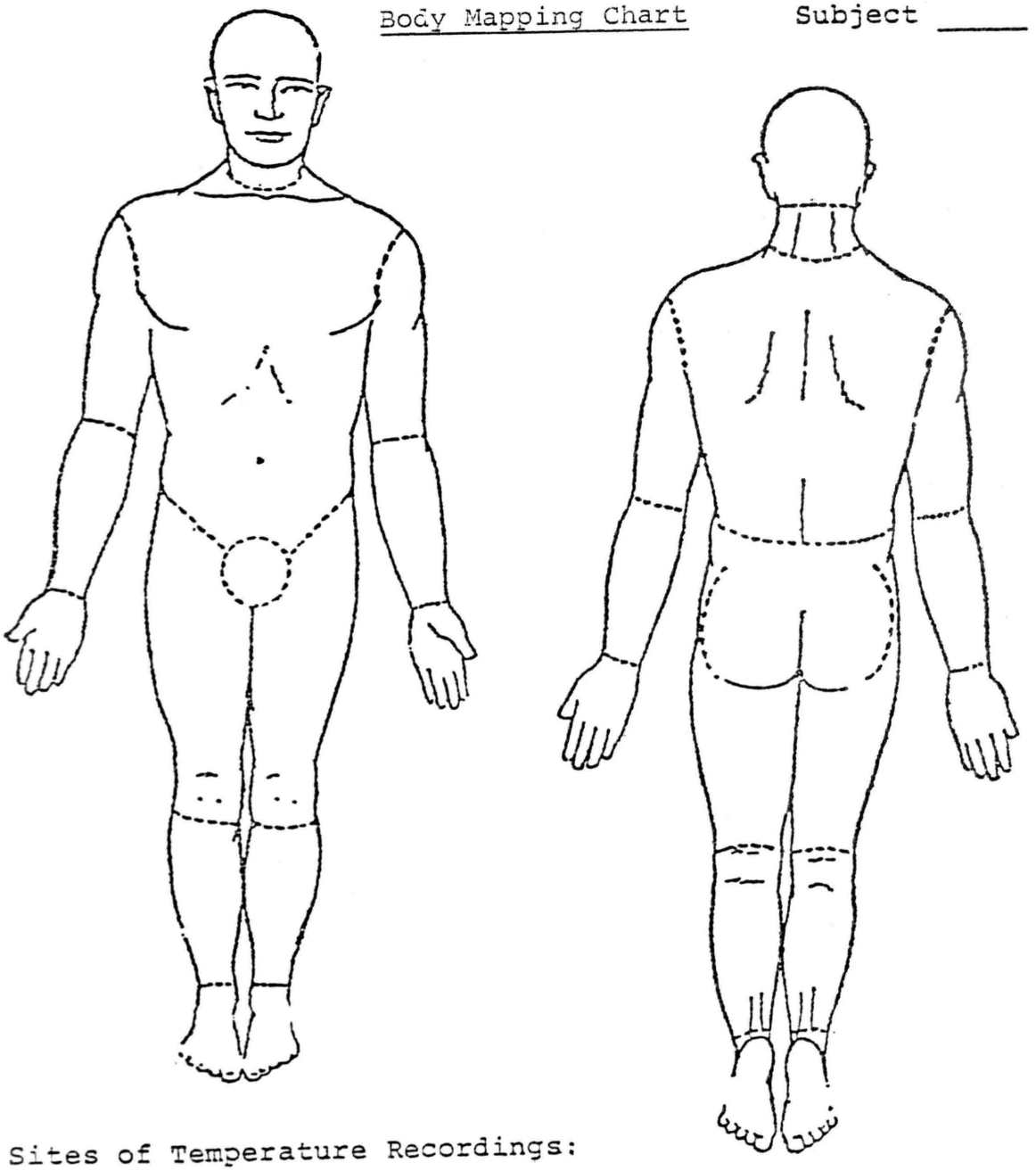
FT = Full-thickness burn

NAA = Nonburned area

Subject _____

APPENDIX G

Body Mapping Chart Subject _____



Sites of Temperature Recordings:

x = full-thickness burns

0 = Deep partial-thickness burns

■ = Nonburned area

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