Title

The relationship of heel skin oxygen tension, heel skin temperature, and external pressure in adults undergoing hip surgery

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Publication Date

2005

Peer reviewed|Thesis/dissertation

THE RELATIONSHIP OF HEEL SKIN OXYGEN TENSION, HEEL SKIN TEMPERATURE, AND EXTERNAL PRESSURE IN ADULTS UNDERGOING HIP SURGERY

by

VIVIAN K. WONG

DISSERTATION

Submitted in partial satisfaction of the requirements for the degree of

DOCTOR OF PHILOSOPHY

in

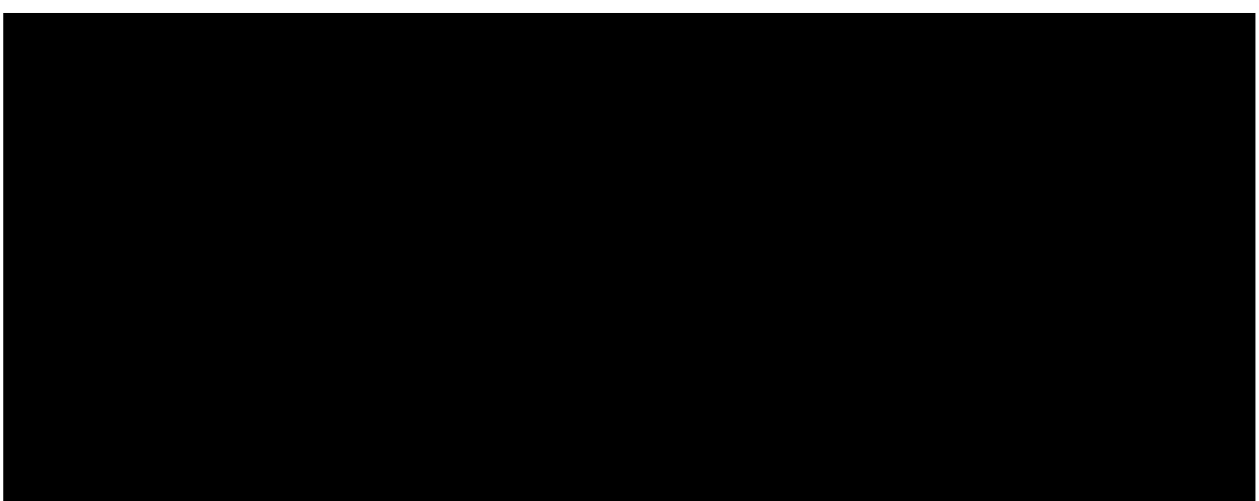
NURSING

in the

GRADUATE DIVISION

of the

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO



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Abstract

THE RELATIONSHIP OF HEEL SKIN OXYGEN TENSION, HEEL SKIN TEMPERATURE, AND EXTERNAL PRESSURE IN ADULTS UNDERGOING HIP SURGERY

by

Vivian K. Wong, RN, CNS, PhD(C)

University of California, San Francisco, 2005

The purpose of this one-group, prospective, repeated measures design study was to examine how the external pressure of the bed surface affects heel oxygen delivery (transcutaneous oxygen [$PtcO_2$]) and temperature in adults in the first 3 days after hip surgery. Oxygen and temperature sensors were placed on the plantar surface of each foot, close to the heels. Pain intensity was measured with a Visual Analog Scale. Measures were taken on room air and with supplemental oxygen when the heels were 1) suspended above the bed surface for 20 minutes (preload), 2) on the bed surface for 15 minutes (loading), and 3) again suspended above the bed surface for 15 minutes (unloading).

Eighteen subjects (9 men and 9 women) from two acute care hospitals participated in the study. Their mean age was 58.3 years (SD 16.08). Findings showed that when compared with preload on room air, both loading and unloading in all three days resulted in a reduction in heel $PtcO_2$ bilaterally ($p = 0.000$). The heel $PtcO_2$ responses were not significant in both legs during the loading and unloading conditions on supplemental oxygen. On room air, heel skin temperature increased during loading and unloading in both legs on post-op days 1 ($p = 0.003$) and 3 ($p = 0.042$) but did not

change on post-op day 2. With supplemental oxygen, heel skin temperature increased during loading and unloading in both legs on all three post-op days ($p = 0.01$). Pain score decreased on days 2 and 3 ($p = 0.015$) with supplemental oxygen. Pain intensity was not correlated with heel $PtcO_2$ or heel skin temperature. These findings show that heel $PtcO_2$ falls with external pressure and does not rise when pressure is removed. There is an increased pressure ulcer risk during loading and after pressure relief. Heels on both legs are at risk of low oxygenation and pressure ulcer development after surgery. Further work is needed to understand why even this short period of external pressure results in decreased oxygen and why the oxygen does not return to baseline when pressure is removed.

Nancy A. Stotts, RN, EdD, FAAN – Chair

6/13/05

Date

ACKNOWLEDGEMENTS

Writing this dissertation has given me a new meaning to the acknowledgments I have read in other publications. The limited vocabulary in the written language would not have adequately expressed my heartfelt gratitude to all the people who have helped me to arrive at this point of my life journey. These good people stood by my side to share with me their heart and brain, to pull me out of the pool of despair and trouble, and to light up my academic path.

First and foremost, I am forever indebted to Dr. Nancy Stotts, the dissertation Chairperson, my advisor, role model, mentor, and friend. I have learned a great deal beyond the research training through her vision and enthusiasm. Dr. Stotts has taught me how to think with a critical mind and then to put the thoughts in paper. She has provided me opportunities for professional growth and has assisted me in securing my future career. She is an inspiring star to me in the darkness of uncertainty, ignorance, and struggle throughout the four years of doctoral study.

I would like to express my deepest gratitude to other members of my dissertation committee. Dr. Erika Froelicher, Chairperson for my qualifying examination committee, has provided expert guidance in research design and perpetual support throughout my doctoral study. She has been very kind, patient, and caring throughout the qualifying examination and dissertation process. Dr. Harriet Hopf, the subject matter expert in tissue oxygenation, has spent endless hours in teaching me the concept of tissue oxygenation. She has provided me in-depth knowledge, dependable guidance, and invaluable clinical support in the research process. Dr. Glenna Dowling always offered me critical insights and encouragement throughout the dissertation process. I am

immensely grateful to another great teacher, Dr. Steven Paul, who provided extensive support related to data management and data analysis.

I am very grateful for the support of the orthopedic surgeons, Dr. Harry Jergesen and Dr. Michael Ries, who facilitated my access to patients at the UCSF Medical Center. I would like to extend my thanks to the staff at the UCSF orthopedic clinic, especially Patricia Nichols for her support with subject recruitment. My sincere thanks go to the staff at 12 Long Hospital, and especially to Janet Oatis, nurse manager, for the continual support during data collection. Thanks are extended to all the personnel at the General Clinical Research Center at UCSF.

Many individuals at El Camino Hospital helped with my research process. Dr. Michael Greenfield facilitated the hospital-based IRB approval. Judy Holt, the orthopedic nurse educator, has offered tremendous assistance with subject recruitment. Diana Russell, Vice President of Patient Care Services, has gone beyond her duty to facilitate my access to the facility. Bonnie Trottier helped me to identify potential subjects. My heartfelt thanks go to the following orthopedic surgeons who supported me with patient recruitment: Dr. Richard Gayle, Dr. Bernardo Ferrari, Dr. James Page, Dr. Eric Test, Dr. Jeffrey Kliman, and Dr. John Parker. Thanks are extended to the staff at Pre-admission Unit and 5 East, especially to Diane Allen and Chris Tarver, nurse managers of the respective units, for the continual support during data collection.

A special debt of gratitude is owed to all the subjects at both hospitals who participated in the study.

I also greatly appreciated the support from many of my friends who are always there to listen, to help and to share. Nancy Woodward, a long-time colleague and friend,

helped me to get into and then get out of the doctoral program by her letters of recommendation. She assisted me in gaining access into the El Camino Hospital research system. Dr. Sylvia Lee, a close friend for many years, provided valuable advice throughout my doctoral study. I must acknowledge my many other fellow students, whom I can converse freely about the joy and sorrow of doctoral study.

Last but not least, I owe my utmost gratitude to my parents, Mr. Wong Cheuk Hung and Mrs. Wong-Lai Hang Kuen. Throughout the early school years, they never failed to comfort me whenever I failed a test. They only ask me to try my best in everything I do. Their love and support throughout the years have helped me to complete this milestone. To my husband, Thomas, who continues to give himself, his time, and his energy, in supporting all aspects of my doctoral study. To my daughters, Chermaine, Karmay, and Sherlene, who still love me despite the limited attention I can give. To my three brothers and all my in-laws who cheer me along the way.

This study was funded by the Individual National Research Service Award, the 2004 American Nurses Foundation Grant, the UCSF General Clinical Research Center, the UCSF Graduate Student Research Award, and the Wound, Ostomy, and Continence Nursing Scholarship.

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CHAPTER ONE

THE STUDY PROBLEM

A pressure ulcer is primarily caused by pressure that exceeds capillary closing pressure and results in ischemia (Kosiak, 1959), leading to various levels of skin and tissue destruction. Pressure ulcers cause pain and distress and interfere with rehabilitation. A pressure ulcer can aggravate or result in other complications including infection (Colsky, Kirsner, & Kerdel, 1998; Ellis, Finn, Noone, & Leaper, 2003) and death (Allman, 1998; McGregor, 1995). Managing pressure ulcers imposes financial and social constraints on the individual and society (Allman, Goode, Burst, Bartolucci, & Thomas, 1999; Fox, 2002).

The incidence of pressure ulcers in the United States is increasing (Cuddigan, Berlowitz, & Ayello, 2001). Heels are the second common site of pressure ulcer development in the United States (Cuddigan et al., 2001) and in other developed countries (Gunningberg, Lindholm, Carlsson, & Sjoden, 1999; Young, Nikoletti, McCaul, Twigg, & Morey, 2002). Heel ulcers occur in critical care (Burdette-Taylor & Kass, 2002), long-term care (Horn et al., 2002), and during the post-operative period (Nixon, Brown, McElvenny, Mason, & Bond, 2000).

Pressure ulcers are a frequent complication of a hip fracture and its surgical repair (Lichtblau, 2000; Margolis, Knauss, Bilker, & Baumgarten, 2003). According to the American Academy of Orthopedic Surgeons, there are 172,000 total hip replacements performed in the United States annually (Fallon, 2005). In California alone, nearly 18,000 hip replacements are performed in the older adults annually (Stotts, 1999). The

most common sites of pressure ulcers in people with hip surgeries are the sacrum and heels (Gunningberg et al., 1999; Versluysen, 1985).

Sacral pressure ulcers have received much attention in recent years. Pressure-reduction and pressure-relief mattresses and beds have been developed to diminish the risk of pressure ulcers on the torso. These support surfaces are found to be quite effective in lowering sacral pressure but less effective on lowering heel pressure (Hardin, Cronin, & Cahill, 2000). A study measuring the interface pressures (pressure exerted on the skin by the mattress) on a variety of devices including standard hospital mattresses, foams, and low-air loss beds showed that heel interface pressures ranged from 35 to 85 mmHg (Hedrick-Thompson, 1992). This means that the heels are subjected to a pressure much higher than the acceptable capillary closing pressure of 32 mmHg, a threshold beyond which skin damage may occur (Landis, 1930). Recent studies on alternating pressure air mattresses also showed that low air-cell pressures did not produce low interface pressure under the heels (Rithalia, 2004).

Patients undergoing hip surgery are at high risk for heel ulcers on both legs due to the combination of increased external pressure from immobility, friction, shear, and decreased blood flow due to pain and positioning during surgery. The mechanism of heel ulcer formation in relation to tissue perfusion and external pressure in the adults undergoing hip surgery requires further exploration.

Tissue Oxygenation

Pressure ulcer development is caused by external pressure compressing soft tissue on/over a bony prominence, reducing skin and tissue tolerance, and blocking capillary blood flow and tissue oxygenation. Oxygen diffuses along gradients of decreasing partial

pressure through the alveoli and blood into the tissues. The rate of diffusion of oxygen is directly proportional to the partial pressure of the oxygen (oxygen tension) (Guyton & Hall, 1997).

Transcutaneous oxygen tension ($PtcO_2$) is a measure of the oxygen diffusion from the dermal capillaries to the skin surface (Talbot, Neuman, Saidel, & Jacobsen, 1996). Transcutaneous oxygen levels are dependent on systemic blood flow and arterial oxygen content (Kram, Appel, & Shoemaker, 1989). The level of $PtcO_2$ is dependent on the balance between the arterial oxygen partial pressure and metabolic oxygen consumption.

External pressure causes a reduction in skin oxygen tension (Colin & Saumet, 1996; Knight, Taylor, Polliack, & Bader, 2001). Any interference with blood flow or oxygen delivery and transport will impede tissue and skin oxygenation, subsequently increasing the risk of tissue ischemia and/or skin breakdown. After hip surgery, an immobilizer (abduction pillow) is often used to keep the hip in alignment and to prevent prosthetic dislocation (Rao & Bronstein, 1991; Yuan & Shih, 1999). The heels are often confined to the surface of the bed. The non-operative leg is used by the patient for turning and repositioning in bed and the heel of this leg may be used as pivot point, thus making it susceptible to constant friction and pressure. The less mobile heel on the operative side may also be subjected to external pressure from the bed surface leading to heel ulcers (Kosiak, 1966). Both heels are at risk for development of pressure ulcers. Whether heel skin oxygen tension in the operative leg is different from the non-operative leg is not known.

Reactive Hyperemia

When external pressure is applied to skin, flow is decreased or ceases. As pressure is released, reactive hyperemia occurs where there is an increase in blood flow above baseline when circulation is re-established. Some researchers theorize that pressure ulcer development may be related to a lack of physiologic increase in blood flow after pressure is relieved (Herrman, Knapp, Donofrio, & Salcido, 1999; Xakellis, Frantz, Arteaga, & Meletiou, 1993); that is, the lack of hyperemic response. The relationship between changes in heel skin oxygen tension and hyperemic response needs further exploration.

Skin Temperature

Reactive hyperemia may be related to an increase in tissue temperature when there is an inflammatory response, increased perfusion (Sprigle, Linden, McKenna, Davis, & Riordan, 2001), and damaged tissue cannot dissipate heat efficiently (Sae-Sia, Wipke-Tevis, & Williams, 2005). When the heels are subjected to external pressure, there may be a decrease in skin blood flow and tissue conduction of heat leading to a decrease in temperature in the heel skin. Consequently, the application and removal of external pressure may induce a different local thermal response in the skin. Whether the rise or fall in local skin temperature can indicate tissue damage is controversial (Newman & Davis, 1981; Schubert, 1994; Sprigle et al., 2001).

Pain

Pain is a significant part of a patient's experience after hip surgery (Bierma-Zeinstra et al., 2001). Postoperative pain can interfere with mobility (Parker & Rajan, 2001) and physical function, despite the use of pain medication (Strassels, Chen, & Carr,

2002). Pain stimulates the sympathetic nervous system resulting in arteriolar vasoconstriction leading to reduced blood flow and lowered skin oxygen tension. Pain thus may compromise skin perfusion (Akca et al., 1999) and increase the risk of pressure ulcers. Some data show that the majority of pressure ulcers are diagnosed between admission and the fourth day after hip surgery (Gunningberg, Lindholm, Carlsson, & Sjoden, 2000). Coincidentally, patients usually have high pain levels during the first few postoperative days, especially on the first day after total hip surgery (Strassels et al., 2002). Pain thus may be a confounding variable when examining blood flow, if not controlled for or measured.

Vasoconstriction

Besides pain, vasoconstriction can also be caused by hypovolemia (Gottrup, Firmin, Rabkin, Halliday, & Hunt, 1987), epinephrine infusion (Jensen, Jonsson, Goodson, Hunt, & Roizen, 1985), cigarette smoking (Jensen, Goodson, Hopf, & Hunt, 1991), and a decrease in temperature (Hopf et al., 2000). Therefore, patients undergoing hip surgery are at high risk for heel ulcers due to the combination of increased external pressure from immobility, friction, shear, and decreased blood flow due to pain, blood loss and reduced body temperature.

Thus, the purpose of this study is to examine the relationship among external pressure, heel skin oxygen tension ($PtcO_2$), and heel skin temperature in the non-operative and operative leg after hip surgery in adults. External pressure refers to loading, the subjection of the heels to pressure exerted by the bed surface. Preload is the time period before loading when heels are kept off the bed surface. Loading is when the legs are placed on the bed surface with heels touching the bed. Unloading is when the

heels are kept off the bed surface after a period of loading. The specific aims are to determine whether:

1. there is a difference in heel skin oxygen tension and heel skin temperature in the first three days after surgery in the *non-operative leg* when compared to the *operative leg*,
2. heel skin oxygen tension and heel skin temperature change during the immediate unloading period,
3. changes in heel skin temperature are correlated with changes in heel skin oxygen tension in the loading unloading response,
4. pain affects heel skin oxygen tension and heel skin temperature during the loading and unloading period.

The study hypotheses are:

1. There will be no overall change in heel skin oxygen tension ($PtcO_2$) response in either leg during the loading and unloading conditions in the first three days after surgery on room air.
2. There will be no overall change in heel skin temperature in either leg during the loading and unloading conditions in the first three days after surgery on room air.
3. There will be no difference in heel skin oxygen tension in either leg in the first three minutes during unloading on post-op days 1-3 on room air.
4. There will be no difference in heel skin temperature in either leg in the first three minutes during unloading on room air.
5. There will be no correlation between changes in heel skin oxygen tension and changes in heel skin temperature between preload and end of unloading in either leg on post-op days 1-3 on room air.

6. There will be no correlation between changes in heel skin oxygen tension in each leg and the development of heel skin breakdown in the first three days after surgery on room air.
7. There will no overall change in pain scores at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery on room air.
8. There will be no correlation between heel skin oxygen tension in either leg and pain scores at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery on room air.
9. There will be no correlation between heel skin temperature in either leg and pain scores at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery on room air.
10. There will be no overall change in heel skin oxygen tension response in either leg during the loading and unloading conditions in the first three days after surgery with an oxygen challenge.
11. There will be no overall change in heel skin temperature during the loading and unloading conditions in either leg in the first three days after surgery with an oxygen challenge.
12. There will be no difference in heel skin oxygen tension in either leg in the first three minutes during unloading on post-op days 1-3 with an oxygen challenge.
13. There will be no difference in heel skin temperature in either leg in the first three minutes during unloading on the first three post-op days with an oxygen challenge.

14. There will be no correlation between changes in heel skin oxygen tension and changes in heel skin temperature on either leg between preload and end of unloading on post-op days 1-3 with an oxygen challenge.
15. There will be no correlation between changes in heel skin oxygen tension in each leg and the development of heel skin breakdown in the first three days after surgery with an oxygen challenge.
16. There will be no overall change in pain score at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery with an oxygen challenge.
17. There will be no correlation between heel skin oxygen tension in either leg and pain scores at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery with an oxygen challenge.
18. There will be no correlation between heel skin temperature in either leg and pain scores at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery with an oxygen challenge.

CHAPTER II

REVIEW OF LITERATURE

The literature review will begin with a description of normal skin and skin blood flow and then address pathophysiology of pressure ulcer development, hyperemic response, tissue oxygenation and measurement of transcutaneous oxygen tension, skin temperature, and hip surgery.

Normal Skin and Blood Flow Mechanism

Normal Skin Structure

Normal skin consists of three layers: epidermis, dermis, and subcutaneous tissue. The epidermis consists of stratified squamous epithelium in the outermost layer and a single row of columnar basal cells in the inner layer. The dermis is composed of three types of connective tissue: collagen, elastic tissue, and reticular fibers. Elastin fibers are interspersed in the reticular layer and give skin its elasticity (Hill, 1998). This fibrous and elastic matrix provides a basis for the branching of blood vessels, nerves, lymphatics, epidermal appendages, and sweat glands (Parker, 2000). The autonomic system supplies the motor innervation of the skin. Adrenergic fibers innervate the blood vessels, hair erector muscles, and apocrine glands (Habif, 1996).

The subcutaneous tissue is the deepest layer of the skin and mainly consists of adipose tissue. It varies in thickness in all areas of the body. It insulates the underlying fascia, muscle and bone and acts as a mechanical shock absorber (Hill, 1998). Arteries from the deep fascia form a network of arterioles in the subcutaneous tissues. The arterioles further branch out into metarterioles and capillaries supplying the dermis (Nixon, 2001).

Microcirculation/Skin Perfusion/Skin Blood Flow

Microcirculation includes arterioles, capillaries, and venules. The arterioles in the skin branch into metarterioles. Metarterioles further divide into capillaries. Smooth muscle cells at the origin of the capillaries act as precapillary sphincters, monitoring capillary blood flow (Ganong, 2001). From the capillaries, blood enters the venules and eventually returns to the heart. Microcirculation is controlled partly by sympathetic vasoconstrictor impulses from the brain and partly by vasoactive substances secreted locally by the endothelial system (Bliss, 1998).

Blood Flow in Tissue

Blood flow through the skin is 10 to 20 times greater than is required to supply necessary metabolites and oxygen. A larger volume of blood flows in the deeper subpapillary layers and a smaller volume of nutritive capillary blood flows in the more superficial layers (Schubert, 1994). Under basal conditions about 8.5% of the total blood flow passes through the skin, controlled primarily by the sympathetic nervous system (Parker, 2000).

Active vasoconstrictor nerves are adrenergic and secrete norepinephrine. Painful stimuli cause diffuse production of epinephrine and norepinephrine, leading to cutaneous vasoconstriction (Ganong, 2001) and subsequent reduced blood flow. On the contrary, a decrease in vasoconstriction and the local production of bradykinin in sweat glands and vasodilator metabolites will result in cutaneous vasodilatation (Ganong, 2001).

Besides the central nervous system, cutaneous blood flow is also affected by the thermoregulatory response. Blood flow in response to hot and cold stimuli can vary from 1 to 150 mL/100g of skin per minute (Ganong, 2001). During exercise, body temperature

rises and cutaneous vessels dilate. Cold causes vasoconstriction but with severe cold, there is superficial vasodilatation (Ganong, 2001). Autoregulation of blood flow occurs when there is an acute change in arterial blood pressure or an increase in venous pressure. Blood flow to the capillaries is readjusted accordingly.

Pathophysiology of Pressure Ulcer Development

Pressure Ulcer Development

The primary cause of pressure ulcers is pressure that exceeds capillary closing pressure and results in ischemia (Kosiak, 1959). Even though capillary closure depends on local pressure gradients within the vessel wall, a capillary closing pressure of 32 mmHg has been used as a threshold beyond which skin damage may occur. This is the average pressure required to occlude blood flow in the arteriolar limb of capillaries supplying the fingernail bed (Landis, 1930). Yet, tissue ischemia may occur even when external pressure is lower than capillary closing pressure. Capillary flow is not blocked but it may not deliver enough oxygen to meet tissue oxygen utilization (Xakellis, Frantz, Arteaga, & Meletiou, 1991).

Both high external pressure applied for a short duration and low external pressure applied for a long duration over bony prominences will cause localized areas of cellular necrosis (Kosiak, 1959). Also, superficial tissue ulceration can be caused by the effect of mechanical forces acting on localized areas of skin and subcutaneous tissue (Kosiak, 1961). When patients are in bed and immobile, the source of external pressure can be a bed surface, tight bed-covers, or pressure and friction generated when the person becomes restless. External pressure reduces or blocks blood supply, affecting the delivery of nutrients and oxygen to the tissue. If external pressure is maintained or

repeated, tissue oxygenation and perfusion will be compromised and tissue damage and eventual cell necrosis will result (Bader, Barnhill, & Ryan, 1986).

Tissue Tolerance to External Pressure

Tissue tolerance is the extent to which tissue can endure the effects of physiological insult without showing damage. Tissue tolerance decreases when tissue is exposed to external forces such as shear, friction, and pressure. Tissue tolerance varies depending on the difference in anatomical make-up under the skin. The responses of skin over bone and skin over muscle to local compression were studied in normal volunteers (n=12). Transcutaneous partial pressure of oxygen ($PtcO_2$), subcutaneous pressure, and displacement (indentation of skin) were measured on normal skin in humans over the tibialis anterior muscle and over the tibia (Sangeorzan, Harrington, Wyss, Czerniecki, & Matsen, 1989). For $PtcO_2$ to reach zero, it required significantly greater external pressure acting on skin over muscle (71±16 mmHg) than on skin over bone (42±8 mmHg) ($p<0.001$). The displacement was greater in skin over muscle than over bone when $PtcO_2$ equals zero ($p<0.001$). Subcutaneous pressure at which $PtcO_2$ reached zero did not differ in the two sites (Sangeorzan et al., 1989). This unique study shows that skin over muscle tolerates greater external pressure than that of skin over bone before tissue oxygenation ceases. Tissue over bony prominences, therefore, is more susceptible to pressure related damage than soft tissues.

Shear is a force exerted parallel to the tissue. It happens when center of gravity pulls down the body but the skin and the upper fascia remain in the original position (Defloor, 1999). For instance, when a person is semi-reclining in bed or chair, the body slides down but the upper skin layers are still affixed to the chair or bed. This shear

between two layers of tissue leads to stretching, kinking and possibly tearing of the perforating vessels in the subcutaneous tissues (Defloor, 1999). Some studies have shown that skin and tissue can adapt to the usual, normal mechanical load and shear forces (Goldstein & Sanders, 1998). However, as shear forces increase, skin breakdown occurs (Goldstein & Sanders, 1998). Shearing can cause endothelial damage to the micro circulation. With a high level of shear, the degree of pressure needed to produce vessel occlusion is reduced by almost half (Dinsdale, 1974). Shear, working simultaneously with pressure, promotes vessel occlusion (Bennett, Kavner, Lee, & Trainor, 1979).

Friction occurs when a moving surface is brought into contact with another surface. Friction enables normal mechanical functions of the body such as holding a cup and walking on a wet floor. However, as the magnitude of frictional forces increases, separation of the epidermal cells at level of the stratum spinosum occurs (Knapik, Reynolds, Duplantis, & Jones, 1995). For instance, a restless person may sustain heel skin damage when the heels are repeatedly rubbed against the bed surface. The effects of the force of friction depend on several factors such as age, anatomical site, and skin hydration (Sivamani, Goodman, Gitis, & Maibach, 2003). Friction and shear both reduce tissue tolerance and have been shown to be predictive of pressure ulcer occurrence (Perneger et al., 2002; Schue & Langemo, 1998).

Microstructural Changes under External Pressure

The effect of external loading pressure on local tissue damage has been examined in a rat model (Bosboom et al., 2001). Tissue damage was visible 24 hours after pressure loading of the muscle. The damage extended from superficial to deep muscle tissue in a zone never exceeding the diameter of the pressure load indentor. Undamaged tissue had

the typical cross-striated appearance of skeletal muscle, while the cross-striation disappeared and mononuclear cells had infiltrated the damaged muscle tissue (Bosboom et al., 2001). Other research using histological methods showed that deep tissue damage is evident through intact skin (Daniel, Priest, & Wheatley, 1981; Salcido et al., 1994).

Human tissue also suffers structural damage from external pressure. Microstructural changes in human skin exposed to static versus cyclic pressures were investigated. The anatomical human heel was simulated by a model of watch glass, agar layer, and foreskin (Edsberg, Natiella, Baier, & Earle, 2001). Static pressures between 50 and 170 mmHg and dynamic pressures between 110 to 170 mmHg were applied. Dynamic (cyclic-relief) pressure induced parallel alignments of connective tissue collagen bundles, which were oriented perpendicular to the original tissue surface. Constant static pressure produced alignment of the collagen bundles of the connective tissue parallel both to one another and to the compressed tissue surface, indicating the beginning of matrix breakdown (Edsberg et al., 2001). This study showed that constant pressure without relief leads to microstructural breakdown. Since the foreskin used in this study is avascular, micro-structural damage may be different in living, vascular skin. However, this study lays the foundation for future in vivo work to examine tissue damage.

Skin tissues were excised from the sacrum of a deceased subject, where a superficial pressure ulcer had developed. Microscopic exam of the human pressure ulcer showed that there was a relatively dense network of collagen fibers in the papillary layer of the boundary area of damaged tissue when compared with the healthy area (Arao, Obata, Shimada, & Hagisawa, 1998). The authors proposed that morphological changes

of the papillae observed in the boundary area affect microcirculation, impairing tissue viability by inhibiting nutritive blood supply and by accumulating metabolic byproducts that predispose to tissue damage (Arao et al., 1998). Knight and colleagues (2001) measured sweat lactate production, the by-product of anaerobic respiration that occurred due to local occlusion of blood supply. Sweat lactate concentration was higher in pressure loaded tissue (after 30-60 minutes of loading) as compared to unloaded tissue, indicating impairment of blood supply (Knight et al., 2001).

The above pathological findings indicate a relationship between waste production accumulation and skin blood supply. It has been proposed that external pressure is partially transferred from the skin to the interstitial fluid (Dodd & Gross, 1991). The pressure may occlude blood supply and interfere with venous and lymphatic drainage, therefore contributing to the building up of waste products of metabolism in the interstitial spaces and cells (Gottrup, 1994). The disruption of interstitial fluid may also allow cell-to-cell contact, leading to cell membrane rupture, and thereby losing the cushioning effect of interstitial fluid and causing tissue necrosis (Krouskop, 1983).

Microvascular Response to External Pressure

Apart from microstructural changes, microvascular changes also occur in response to external pressure. The adverse effect of microvascular changes depend on the magnitude and duration of external pressure. The microvascular response is usually presented as skin perfusion and/or tissue oxygenation.

Skin perfusion/microcirculation/blood flow is a measure of the movement of red blood cells within the surface capillaries of the skin (Fromy et al., 1997). Tissue oxygenation is a combined process of oxygen delivery, tissue oxygen transport, and oxygen consumption of the cells (Gottrup, 1994). Perfusion is important because it

controls oxygen tension (Chang & Mathes, 1982) and tissue oxygenation, and determines tissue viability. High arterial oxygen tension in the vasculature accelerates the rate and depth of oxygen penetration into tissues (Drucker et al., 1996). Cell death eventually occurs if tissue perfusion is compromised, usually due to external pressure or internal blood vessel constriction. Tissue oxygenation is measured via subcutaneous oxygen tension and oxygen delivery to the skin is measured by transcutaneous oxygen tension.

Skin Microcirculation and Magnitude of External Pressure

Fromy and colleagues (1997) observed that a significant reduction of microcirculation of the human forefoot occurred with 10 mmHg of external pressure ($p<0.001$) when measured with laser Doppler. As external pressure increased up to 60 mmHg, skin blood flow continued to decrease (Fromy et al., 1997). The effect of pressure loading on the sacrum was studied in female non-smokers ($n = 9$) (age = 22 years ± 8.1) and smokers ($n = 9$) (age = 22.3 years ± 19.6) using laser Doppler (Noble, Voegeli, & Clough, 2003). Loading resulted in a significant reduction in blood flow in all subjects ($p < 0.05$). A limitation of the laser Doppler method is that it measures flow in comparison to baseline and that baseline is not quantified in any way. Nonetheless, a number of studies showed that with an increase in external pressure, transcutaneous oxygen and perfusion also decreased (Abu-Own, Sommerville, Scurr, & Coleridge-Smith, 1995; Colin & Saumet, 1996; Schubert & Fagrell, 1989; Xakellis et al., 1991).

However, a study of external pressure on rats showed that with an increase in pressure, skin perfusion initially increased until reaching a maximum and then decreased further with an increase in pressure, eventually reaching zero perfusion (Herrman et al., 1999). In another study, the effect of surface pressure and skin temperature on skin

perfusion was measured in hairless fuzzy rats. Skin surface pressure was applied by a computer-controlled plunger while a laser Doppler flowmeter measured skin perfusion (Patel, Knapp, Donofrio, & Salcido, 1999). For unheated skin, perfusion increased as skin surface pressure increased from 3.7 to 18 mmHg. Further increases in surface pressure caused a decrease in perfusion (Patel et al., 1999).

Data differ depending on whether external pressure leads to an increase or a reduction in skin blood flow. Most studies do not measure the increase in flow since it happens fairly quickly. It seems like there is an increase in skin blood flow when pressure is applied below a certain magnitude. The increased skin blood flow may be explained by the vasodilation of arterioles and increased vessel diameters in response to increased skin surface pressure (Herrman et al., 1999). Another explanation for the transient increase in blood flow is the pressure-induced vasodilation (PIV) response. PIV is a protective reflex response that depends on mechanoreceptors, namely, the capsaicin-sensitive fibers. The reflex causes vasodilation in response to a progressive increase in local pressure (Koitka et al., 2004). PIV works at skin temperature of 34 – 35 °C (Koitka et al., 2004). With a continual increase or prolonged duration of pressure, there will be more micro-structural changes with less vasodilation, and ultimately, blood flow stops.

Duration and Magnitude of External Pressure on Pressure Ulcer Development

Many studies utilize skin blood flow, tissue perfusion, and/or histological findings to measure the relationship between external pressure and pressure ulcer development. Duration of external pressure has some effects on tissue blood flow and subsequent pressure ulcer development. Studies in this area are quite scarce and the exact duration of external pressure on pressure ulcer development has not yet been defined.

Classic Animal Studies

In an early experiment of pressure applications (n=62) over dogs' femoral trochanter and ischial tuberosity, Kosiak (1959) discovered that both high external pressure (510 mmHg) applied for short periods (2 hours) or low external pressure (140 mmHg) applied for a long time (11 hours) caused localized areas of cell necrosis. Pressure applications ranged from 100 mmHg to 550 mmHg for periods of 1 to 12 hours. Histological changes occurred after 60 mmHg pressure was applied for 1 hour (Kosiak, 1959).

Daniel and colleagues (1981) examined the effect of subjecting swine (n=30) to localized pressures ranging from 30 mmHg to 1000 mmHg for periods of 2 to 18 hours (Daniel et al., 1981). Pressure application sites were observed daily and histological exam of the involved tissue was performed on day 7. It was found that with high pressure - short duration (500mmHg, 4 hours) and low pressure - long duration (100mmHg, 10 hours), deep muscle destruction occurred even though skin was intact. Tissue damage occurred beginning at the muscle layer and extended upward to the lower dermis when there was high pressure - long duration (800 mmHg, 10 hours) or low pressure with prolonged duration (200 mmHg, 15 hours). Both skin and muscle damage took place during long duration of low to high pressures (600 mmHg, 11 hours; 200 mmHg, 16 hours) (Daniel et al., 1981). This study showed that the initial pathological damage occurred in the muscle and then progressed toward the skin with increasing pressure and / or duration. Since skin capillary pressures range from 13 to 32 mmHg, external pressure applied might have been transmitted to the bone (Kosiak, 1959).

Muscle necrosis was observed after 4 hours of applied pressure while skin damage occurred after 8 hours in this swine model. The authors speculated that the primary cause of skin destruction was due to mechanical damage whereas the primary cause of muscle necrosis was due to tissue ischemia (Daniel et al., 1981).

Computer-controlled pressure was applied for six hours for a maximum of five sequential sessions, to skin over the greater trochanter of anesthetized fuzzy rats (Salcido et al., 1994). Histopathological changes that occur in the development of pressure ulcers were similar, but more pronounced after the third, fourth, and fifth sessions as compared to the first or second sessions. The damage developed first in the muscle rather than the dermis or epidermis. Recurrent pressure results in increasingly severe damage to the vascular system and parenchyma (Salcido et al., 1994). Salcido and colleagues (1995) then applied 145 mmHg pressure for 5 consecutive 6-hour sessions in another experiment with fuzzy rats and showed a greater than 90% incidence of pressure ulcers (Salcido et al., 1995).

These animal experiments all support the inverse relationship between pressure and duration in tissue damage. Prolonged pressure damages all tissue layers and damage occurs first in deep muscle and progresses upward to the fascia, subcutaneous tissue, and finally the skin.

Clinical Settings

In clinical settings, the adverse effect of prolonged subjection to external pressure happens in immobilization. The relation between pressure ulcer incidence and length of immobilization in the supine position without pressure-relieving devices was explored in spinal cord injured patients (n=49) (Curry & Casady, 1992). Immobilization exceeding 6

hours was associated with subsequent development of a sacral or occipital pressure ulcer ($p=0.0094$) (Curry & Casady, 1992). In a multi-site survey across 33 states on the incidence of intraoperatively acquired pressure ulcers, it was noted that as the length of surgery increased, the incidence of pressure ulcers also increased (Aronovitch, 1999). However, it should be noted that both the sympathetic and parasympathetic nervous system innervations are ablated in those with spinal cord injury and under anesthesia. The ability to generalize the findings to people with intact autonomic nervous system is limited.

Blood Flow Changes During and After Pressure Application

Sanada and colleagues (1997) explored the relationship of skin blood flow and post-operative pressure ulcer development in 24 patients who underwent lengthy surgeries (surgery time = 360-460 minutes). Skin blood flow was measured using laser Doppler flowmetry at the sacrum in patients placed in the supine position and at the iliac bony prominence in patients placed in the prone position (Sanada et al., 1997). Pressure ulcer development was assessed immediately after surgery and 24 hours later. Skin blood flow in the 9 patients who developed pressure ulcers (36%) decreased within 1 hour after pressure application and did not return to the pre-pressure application level during the surgery duration. Skin blood flow gradually increased during pressure application and remained increase for the duration of surgery in patients who did not develop pressure ulcers. Other factors such as obesity, blood volume loss, and blood pressure were not significant determinants of pressure ulcer incidence in these patients. Pressure ulcer development seemed to be influenced mainly by the lack of physiologic increase in skin blood flow at the pressure site rather than by the length of imposed pressure (Sanada et

al., 1997). This finding agrees with the few animal studies that showed an increase in skin blood flow during pressure application. However, the study contradicts many other studies that supported the inverse relationship between pressure duration and skin blood flow (Daniel et al., 1981; Kosiak, 1959) (Table 1). This is the only study to-date that indicated a physiological increase in skin blood flow during pressure loading reduces pressure ulcer incidence. The replacement of blood and fluid during surgery could be a confounding factor in the relationship between pressure and skin blood flow in this study. Also, interface pressure (pressure exerted on skin by the mattress) between the operative table surface and the body was not measured.

The relationship between skin blood flow at the greater trochanter and one hour of low levels of compressive pressure was examined in healthy older (n=22) and healthy younger adults (n=19) using laser-Doppler velocitometer (Xakellis et al., 1993). After one hour of being subjected to pressure from an inflatable mattress overlay, blood flow was significantly higher than baseline levels for healthy persons regardless of their age. The authors postulated that the increased skin blood flow could be caused by reflex vasodilation of the dermal blood vessels in response to their being partially occluded (Xakellis et al., 1993). One of the confounding factors could be external pressure (interface pressure) exerted from the mattress overlay. The interface pressure is less than 32 mmHg and still allows microcirculation. Furthermore, this study is conducted on healthy subjects, which did not represent the at-risk population. A similar study examined at-risk elderly patients (n=16) showed an inconsistent pattern of skin blood flow during compression but with an increased skin blood flow after release of compression (Frantz, Xakellis, & Arteaga, 1993).

Table 1.Major Studies Showing Relationship between the Magnitude and Duration of External Pressure and Pathological Changes in Blood Flow and Tissue

Author / Year	**Species and study site**	**Pressure / duration**	**Perfusion/skin blood flow**	**Histological changes**
Kosiak, 1959	Trochanter and ischium of dogs	60 mmHg for 1 hr	Not reported	Pathologic changes in tissue
Daniel et al., 1981	Swine	- 500 mmHg, 4 hrs 100 mmHg, 10 hrs - 800 mmHg, 10hrs 200 mmHg, 15 hrs - 600 mmHg, 11hrs 200 mmHg,16 hrs	Not reported	- Skin intact, deep muscle destruction - Damage occurred from muscle to skin - Skin and muscle damage
Bader et al., 1986	Anterior surface of human forearm	1.33 N/mm force 10% strain	Obliteration of blood flow to capillaries	Not reported
Xakellis et al., 1993	Human trochanter	Lying on air mattress with interface pressure of <32 mmHg for 60 mins.	Dermal blood flow higher than baseline after 60 minutes of compression	Not reported
Abu-Own et al.,1995	Human heels	50 mmHg	Cessation of blood flow	Not reported
Colin et al., 1996	Human sacral skin	10 mmHg at 2 min intervals up to 180 mmHg	Decrease in skin blood flow at 20 mmHg; skin blood flow at its lowest at 90 mmHg	Not reported
Meinders et al.,1996	Human footsole	> 300 mmHg	Stop skin microvascular flow	Not reported
Fromy et al., 1997	Forefoot of humans	10 mmHg	Reduction of microcirculation	Not reported
Herrman et al., 1999	Hips of fuzzy rats	58 mmHg	Complete restriction of blood flow	Not reported
Patel et al., 1999	Hairless fuzzy rats	3.7 - 18 mmHg 19 - 55 mmHg > 55 mmHg	Skin perfusion increases Decrease in perfusion Zero perfusion	Not reported
Bosboom et al., 2001	Tibialis anterior muscle in rats	Various loads at 75, 525, & 1875 mmHg for 24 hrs	Not reported	Tissue damage
Mayrovitz et al.,2003	Human heels	20 mmHg for 5 mins	Skin blood flow occluded	Not reported

Risk Factors for Pressure Ulcers

Many risk factors contributed to the development of pressure ulcers. The most commonly identified risk factors are immobility, external pressure over bony prominence, moisture, inadequate nutrition, and co-morbidities (van Marum et al., 2001). There is a huge volume of literature identifying specific risk factors associated with pressure ulcer development in different clinical settings. Most studies from 1994 to 2004 agreed that pressure ulcer incidence in hospitals is associated with fecal or urinary incontinence, impaired mobility, malnutrition, and decreased mental status (Maklebust & Magnan, 1994; Reed, Hepburn, Adelson, Center, & McKnight, 2003). In nursing homes residents (n = 43,881), the common risk factors associated with pressure ulcer development were decrease in mobility, nutritional problems, diabetes, and urinary or fecal incontinence (Berlowitz et al., 2001; Brandeis, Ooi, Hossain, Morris, & Lipsitz, 1994). In non-hospice home care patients (n=1711), predictors of pressure ulcers included confinement to bed, dependence in dressing, a current fracture, oxygen use, needing assistance with transferring, and urinary incontinence ($p < 0.001$) (Bergquist, 2003).

Across all settings in all of the above studies, impaired mobility, incontinence, and inadequate nutrition are factors associated with pressure ulcer development. Reduced mobility is related to pressure ulcer development and the risk increased with time (Boyle & Green, 2001). Sustained skin wetness increased vulnerability to pressure-induced blood flow reduction, rendering the skin at risk for pressure ulcer development (Mayrovitz & Sims, 2001) in incontinent patients. Underweight (Krause, Vines, Farley, Sniezek, & Coker, 2001), low body mass index (Kernozek, Wilder, Amundson, &

Hummer, 2002), low prealbumin level (Guenter et al., 2000), low serum albumin level (Bourdel-Marchasson et al., 2000) and poor nutrition (Casimiro, Garcia-de-Lorenzo, & Usan, 2002) are associated with pressure ulcer development.

Underlying severe physiological conditions contribute to a greater incidence of pressure ulcers. In critical care, the effects of specific risk factors for the development of pressure ulcers were examined (Theaker, Mannan, Ives, & Soni, 2000). Five of the specific risk factors identified as being independently significant ($p < 0.05$) in pressure ulcer development were norepinephrine infusion, APACHE II score, fecal incontinence, anemia, and length of stay (Theaker et al., 2000). The existence of co-morbidities has been associated with pressure ulcer development in all clinical settings. The presence of cardiovascular disease, cerebrovascular accident, high serum urea showed a significant relationship with an impaired blood flow response (van Marum et al., 2001). Commonalities among these physical conditions all referenced back to the identified risk factors, namely, inadequate blood supply, wetness of skin, and immobility.

Reactive Hyperemia/Hyperemic Responses

Hyperemia occurs when blood initially flows through previously occluded vessels. In the context of pressure ulcer, occlusion has been created by external pressure. When external pressure is released, dilation of vessels results in increased blood flow beyond baseline. The increase in flow is visible as erythema or reactive hyperemia.

A study on isolated rat skeletal muscle arterioles found that mechanosensitive mechanisms are likely to contribute significantly to the in vivo development of reactive hyperemia. With changes in hemodynamic forces (intraluminal pressure and flow/shear stress), endothelial and smooth muscle cell stretch receptors are activated. Increased

levels of endothelial nitric oxide (NO) is synthesized which contributes to vasodilation of arterioles and subsequently, the development of the peak reactive dilation (Koller & Bagi, 2002).

An increase in skin blood flow, the normal hyperemic response to external pressure, could be the result of a compensatory vasodilatation of dermal circulation that serves to minimize the damage from the compressive pressure (Xakellis et al., 1993). Some researchers hypothesized that pressure ulcer development may be related to the velocity of blood flow after a period of ischemia (Meijer, Germs, Schneider, & Ribbe, 1994) or a lack of physiologic increase in blood flow after pressure is relieved (Herrman et al., 1999; Xakellis et al., 1993).

Reduced hyperemic response may result from capillary damage, endothelial dysfunction, capillaries being plugged by leukocytes, or failure of the capillaries to dilate (Herrman et al., 1999). Reduced hyperemic response means reduced perfusion. Lack of the hyperemic response or nonblanchable erythema is a sign of impaired blood supply and tissue destruction (Bergstrom, Bennett, & Carlson, 1994).

Heel Skin Blood Flow

The hyperemic response of the heel skin to external pressure has been examined in several studies. Meinders and colleagues (1996) measured skin microvascular responses of the human footsole to changes in externally applied pressure. Skin microvascular blood flow was measured in healthy volunteers (n=11), during and after external mechanical pressure of different magnitudes. Pressures above 40kPa (300 mmHg) stopped skin microvascular blood flow. Releasing the applied pressure resulted in a hyperemic response that increased when the applied pressure increased from 40 to 80

kPa (300 to 600 mmHg). Higher pressures from 80 kPa to 160 kPa (600 mmHg to 1200 mmHg) did not influence the amplitude in skin microvascular response, but resulted in a longer delay to maximal hyperemia (Meinders, de Lange, Netten, Wollesheim, & Lutterman, 1996). The external pressure used in many of the experiments was much higher than that experienced in the average clinical settings.

Mayrovitz and colleagues (1997) explored the hyperemic response of heel skin in 11 healthy women using laser-Doppler imaging scan. Heel perfusion was significantly reduced upon pressure loading and during loading while a significant hyperemic response was seen for up to 10 minutes after pressure release ($p<0.01$) (Mayrovitz, Smith, Delgado, & Regan, 1997). This study gives some insight into heel skin blood flow in response to pressure, even though important data such as vascular status of the subjects and magnitude of the external load were not addressed.

Mayrovitz and Smith (1998) went on to examine the effect of loading and unloading on heel perfusion using laser Doppler Imaging to examine heel blood perfusion in 11 persons with normal vascularity before pressure loading (10 minutes), during pressure loading (40 minutes) and after off-loading (20 minutes). Subjects had to lie supine. Subject's one heel was placed on a transparent plate (pressure loading) through which heel blood perfusion data were obtained (Mayrovitz & Smith, 1998). Heel perfusion was rapidly and significantly reduced on loading ($P < 0.01$) with the greatest reduction within the central heel area; perfusion remained uniformly depressed throughout the loading interval while off-loading was associated with a rapid onset hyperemia which exceeded baseline ($P < 0.01$) for 10 minutes.

Mayrovitz et al.(1999) then studied the effect of load magnitude and duration on hyperemic response of the heel in 14 women with normal vascularity (Mayrovitz, Macdonald, & Smith, 1999). Heels were subjected to sequential external loads with graded magnitudes (30-140 mmHg) and durations (2.5-20 minutes). All heel loads and durations resulted in hyperemic responses, with the largest increase in peak response occurring between heel loads of 60 and 120 mmHg. Recovery times (time taken for blood flow to return to baseline) also increased with both load duration and magnitude. The maximum recovery time was 7.5 minutes with 140 mmHg pressure applied for 20 minutes (Mayrovitz et al., 1999).

Mayrovitz and colleagues recently (2003) examined heel skin blood flow after pressure relief in young, healthy subjects (n=12). The heel was subjected to a cyclic pressure of 20 mmHg (loading) and 10, 5, and 0 mmHg (progressive unloading) at 5-minute intervals. Skin blood flow, measured by laser Doppler, decreased to an ischemic level at 20 mmHg of loading pressure. Skin blood flow was greater than the baseline level when pressure was released to 0 mmHg ($p<0.001$) but was less than baseline level when pressure was only released to 10 mmHg ($p<0.001$). The authors suggested that partial relief of pressure might cause a partial blunting of hyperemia, resulting in skin blood flow lower than baseline level (Mayrovitz, Sims, Taylor, & Dribin, 2003). The 5-minute interval may be too short to fully test hyperemic response.

In all Mayrovitz's studies on the heels of healthy adults, skin blood flow was reduced with application of external pressure and increased after relief of pressure. Microcirculation in relation to pressure in the heel is consistent with other bony

prominences of the body. Further work is needed in older persons and in those who are at risk for pressure ulcer development.

Ischemia and Reperfusion

An extended cycle of ischemia and reperfusion may produce adverse effects on tissue. Cycles of ischemia and reperfusion at different frequency and duration were induced on the dorsal skin of un-anesthetized rats (n=5) using an external pressure of 50 mmHg (Peirce, Skalak, & Rodeheaver, 2000). Cycles varied from 1 hour of ischemia followed by 0.5 hours of reperfusion to a maximum of 10 hours of ischemia with no reperfusion. Cycles per day and number of days were also manipulated. Tissue injury increased with increased cycles, duration of ischemia, and frequency of cycles. The increase in tissue damage was marked by increased tissue necrosis, leukocyte extravasation, decreased skin blood flow, and skin thickness. The authors postulated that increase in tissue damage happens in the reperfusion phase with the presence of toxic oxygen-free radicals, neutrophils and macrophages, and capillary plugging by leukocytes (Peirce et al., 2000). Consequently, the transport of nutrients and waste products within tissue may be affected. The ischemic phase in this model is produced by compressing an external magnet over the skin under which an implanted steel plate was tunneled. The steel plate was held in place by the surrounding fascia. The effect of reperfusion injury on tissue is worth further exploration since a large number of pressure-relief beds and mattresses simulate the cyclic pressure relief mode.

The above studies showed that hyperemic response occurs after a period of external pressure with vascular occlusion. Time taken for the returning of previous skin blood flow depends on duration and magnitude of external pressure. Hyperemic

responses occur with relief of pressure. Higher external pressure produces a longer hyperemic response time. The relationship between ischemia-reperfusion and skin blood flow requires further investigation.

Microvascular Response and Skin Temperature

Reactive hyperemia may be related to an increase in tissue temperature when there is an inflammatory response or increased perfusion (Sprigle et al., 2001). Likewise, microvascular damage may cause skin temperature in the compromised area to drop (Sprigle et al., 2001). Changes in local skin temperature may be indicative of local reactive hyperemia or early stage of tissue damage (Sprigle et al., 2001).

Existing pressure ulcers do not present an increase in skin temperature. When examining skin perfusion and skin temperature in 12 elderly patients (89 ± 7 years) with an early stage pressure ulcer (involving epidermis and dermis) and 10 elderly (75 ± 5 years) without a pressure ulcer, there was increased skin perfusion in the early stages of pressure ulcer but not an increase in skin temperature (Schubert, 1994).

The response of skin perfusion to pressure-induced ischemia was examined in rats (Herrman et al., 1999). There was a gradual increase in skin temperature with an increase in pressure. Herrman and colleagues (1999) suspected a compromised vasodilator mechanism in these animals. The slight increase in temperature probably reflected increased perfusion (Herrman et al., 1999). A recent study on sacral skin temperature in neurologically impaired patients showed that local skin temperature has a predictive value on pressure ulcer development (Sae-Sia et al., 2005).

Physiology of Tissue Oxygenation

One of the pivotal factors in wound healing is tissue oxygenation. Tissue oxygenation depends on the function of the respiratory and circulatory systems. Tissue oxygenation involves oxygen delivery to the tissues (tissue oxygen transport) and oxygen consumption of the cells (Gottrup, 1994). Reduced tissue oxygen tension is related to pressure ulcer development. This section will address the tissue oxygenation, factors affecting tissue oxygenation, and measurement of transcutaneous oxygen tension. Tissue oxygenation in pressure ulcer development also will be examined.

Tissue Oxygenation

Oxygen diffuses from the alveoli of the lungs into the pulmonary capillaries and diffuses into the blood. In the blood, most of it is bound to red blood cells with a small amount dissolved in the plasma. Oxygen then diffuses out of the red blood cell through the capillary wall, interstitial fluid, crosses the cell membranes, and goes into the mitochondria of peripheral tissues (Wagner, 2000). The delivery of oxygen to the soft tissue depends on the balance between the central cardiovascular mechanisms to deliver blood flow and the tissue metabolic demand for oxygen (Gottrup, 1994). A series of events including adequate oxygenation in the lungs, enough blood flow, and a good oxygen-carrying capacity of blood ensure delivery of oxygen to tissues.

Systemic Circulation

Blood flows through vessels by the pumping action of the heart, diastolic recoil of the walls of the arteries, compression of the veins by skeletal muscles, and the negative thoracic pressure during inspiration (Ganong, 2001). Most of the arteries contain elastic tissues which can stretch and recoil with the pumping of the heart. In periods of low flow

and low oxygenation, central circulatory mechanisms are mobilized by receptors in the aortic arch and carotid bodies, resulting in release of epinephrine with subsequent peripheral vasoconstriction. This infusion of volume maintains flow to the heart and lungs (Schlichtig, Kramer, & Pinsky, 1991). When the need for blood flow is high, tissues such as the muscle are able to increase the number of active capillaries. The skin, subcutaneous tissue and the gastrointestinal tract, however, are regulated by autonomic nerve reflexes and cannot increase blood flow (Gottrup, 1994). These tissues are among the first to be affected during a decline in oxygen delivery (Gottrup, 1994).

The central nervous system has important roles in controlling cardiac output and systemic blood flow. At rest, the heart rate is under the control of the parasympathetic nervous system that secretes acetylcholine to slow down conduction in the atrioventricular node. In response to stress, the sympathetic nervous system releases norepinephrine at the nerve endings throughout the circulation. Epinephrine also is released from the adrenal gland. Both norepinephrine and epinephrine act through the alpha-adrenergic vasoconstricting mechanisms in the periphery, and both increase contractility by stimulating alpha- and beta-adrenergic receptors in the heart (Burkhoff & Weisfeldt, 2000). Consequently, heart rate and myocardial contractility increase while the vascular tone in the various organs is being regulated (Burkhoff & Weisfeldt, 2000). The diameter of the arterioles is regulated systemically by circulating vasoactive substances and vasomotor nerves. Noradrenergic fibers on vessels are responsible for vasoconstriction. The sympathetic nervous system also has a vasoconstrictive effect on the venous system.

Complementary to the sympathetic nervous system is the kidney's renin-angiotensin system. In response to reduced renal perfusion, renin is released to produce angiotensin II, a very potent peripheral vasoconstrictor and coronary arterial constrictor. Angiotensin II stimulates the production of aldosterone to retain sodium in the circulation and increase arterial blood pressure (Ganong, 2001).

Systemic circulation is important to peripheral blood flow but is not representative of peripheral tissue perfusion. Distribution of blood flow to the peripheral soft tissue is mediated by mechanisms that are altered due to the different metabolic needs of the tissues (Gottrup, 1994). Central blood volume falls only when homeostatic mechanisms are exceeded (Hopf et al., 2000). Therefore, measurements of blood pressure, central venous pressure, urine output, cardiac output, lactic acid and base deficit are not reliable indices of tissue oxygenation (Drucker et al., 1996; Gottrup et al., 1987; Powell, Schultz, Burris, Drucker, & Malcom, 1995). Measuring oxygen tension in the subcutaneous tissue ($PsqO_2$) is a more reliable method of evaluating tissue oxygenation (Gottrup, 1994). Tissue oxygen tension drops long before any hemodynamic changes occur and it corresponds with hemodynamic changes only when the systemic circulatory system is severely compromised (Hopf & Hunt, 1994).

The Arterioles and Capillary Bed

The arterioles divide into metarterioles, which may connect directly with venules by thoroughfare vessels. Capillaries branch out from these thoroughfare vessels. In some areas of the body, arteriovenous shunts connect arterioles to venules, by-passing the capillaries and blood flow through thoroughfare vessels to the venules (Ganong, 2001).

At any given time, about 5% of the blood is circulating in the capillaries for oxygenation, nutrient supply, and waste product exchange between interstitial fluid and blood.

Regulation of the circulatory system depends on changes in resistance to flow (Gottrup, 1994). Resistance is dependent on the viscosity of the blood and the length and radius of the blood vessels. The relationship between the flow in a long narrow tube, the viscosity of the fluid, and the radius of the tube is expressed by the Poiseuille-Hagen formula (Ganong, 2001):

$$F = (P_A\text{-}P_B) \times \pi/8 \times 1/\eta \times r^4/L$$

Where F denotes flow, $(P_A\text{-}P_B)$ is the pressure difference between 2 ends of the tube, η is the viscosity of the fluid, r is the radius of the tube, and L is the length of the tube.

Specifically, the radius of the arterioles is primarily responsible for the control of both speed and volume of blood flow. When the radius of the blood vessel is increased, resistance is reduced resulting in an increase in blood flow (Ganong, 2001). Vascoconstriction may either decrease or increase flow velocity. Viscosity of the blood depends mostly on the percentage of the volume of blood occupied by red blood cells (hematocrit) and the variations in plasma proteins. In larger vessels, increases in hematocrit cause an increase in viscosity. In arterioles, capillaries, and venules, hematocrit has relatively less effect on the peripheral resistance (Ganong, 2001). Besides the diameter of the arterioles and blood viscosity, the density of capillaries, the presence of non-capillary pathways such as shunts also contributes to capillary perfusion and delivery of oxygen to tissue (Gottrup, 1994).

Total Body Oxygen Delivery

Normally, approximately 1000 ml/min of oxygen is delivered to the body and only about 250ml/min is needed for metabolism (Nathan & Singer, 1999). The quantity of oxygen transferred in one minute can be computed as follows (Nunn, 1989):

Cardiac output x arterial oxygen content

At rest: 5000 ml/min x 20 ml O_2/100 ml blood = 1000 ml/min

The excess in oxygen supply ensures that the body is able to cope with variations in oxygen delivery without compromising aerobic respiration and oxygen consumption. However, when oxygen delivery falls below a critical point (about 550 ml/min) or oxygen demands increase beyond the oxygen supply and/or tissue oxygen extraction, anaerobic respiration occurs and a rise in lactate from anaerobic metabolism results (Nathan & Singer, 1999). Systemic oxygen delivery does not determine whether oxygen is distributed to organs in proportion to their needs.

About 35-45 % of the total blood volume is made up of red blood cells. Oxygen is carried largely by hemoglobin with a small amount present in the plasma. One gram of adult hemoglobin (calculated molecular weight) can potentially bind 1.39 ml oxygen (Nunn, 1989). A small fraction of the circulating hemoglobin consists of methemoglobin and carboxyhemoglobin that do not bind oxygen (Lavoisier, 1998). The lower binding capacity of 1.34 ml/gm is the measured hemoglobin oxygen binding that accounts for the methemoglobin and carboxyhemoglobin.

Arterial oxygen content (CaO_2) is the sum of the oxygen bound by hemoglobin (Hb) and the oxygen dissolved in the plasma. It is derived from the equation

$CaO_2 = (Hb \times 1.34 \times SaO_2) + (PaO_2 \times 0.003)$

where 1.34 estimates the amount of oxygen that can be bound by 1 gm of hemoglobin when it is fully saturated (SaO_2 = 1.0). The solubility coefficient of O_2 in human plasma with a PaO_2 of 100 mmHg is 0.003 (Snyder & Carroll, 1982). That is, the dissolved oxygen in the plasma is about 0.3 ml/dl, or less than 2% of the total oxygen carried in the blood (Nathan & Singer, 1999). For an arterial blood with 15 mg/dl of hemoglobin and SaO_2 of 1.0, the CaO_2 equals about 20 ml of oxygen per dl of blood.

$$CaO_2 = (15 \times 1.34 \times 1.0) + (100 \times 0.003)$$
$$= 20.1 \text{ ml/dl} + 0.3 \text{ ml/dl}$$
$$= 20.4 \text{ ml/d}$$

When blood passes through the tissue capillaries, the amount of oxygen bound with hemoglobin is reduced, on the average, to 14.4 ml (Guyton & Hall, 1997). Under normal conditions about 5 ml of oxygen are transported to the tissues by each 100 ml of blood passing through the tissue capillaries (Guyton & Hall, 1997). When the partial pressure of oxygen falls to 40 mmHg in the tissue capillaries, only about 0.12 ml of oxygen remains in the dissolved state. That is, 0.12 ml of oxygen is normally transported to the tissue by each 100 ml of plasma (Guyton & Hall, 2000). The dissolved oxygen, however, is important as it is used by the tissues.

Oxygen Transport to Tissue

Oxygen goes from the microcirculation through the blood vessel walls to the interstitial fluid mainly by diffusion (Gottrup, 1994). The movement of gases depends on the partial pressure gradient. Net diffusion of gas goes from an area of high pressure to an area of low pressure and is proportional to the gas pressure difference between the 2 areas (Guyton & Hall, 1997). Oxygen diffuses along gradients of decreasing partial pressure from the air through the alveoli and blood into the tissues. Similarly, carbon

dioxide also diffuses down the partial pressure gradient from the tissues and blood to the alveoli (Ganong, 2001). The rate of diffusion of each gas is directly proportional to the pressure caused by this alone, which is called the partial pressure of the gas (Guyton & Hall, 1997). The pressure exerted by oxygen in the tissue is called tissue oxygen partial pressure (PO_2) or tissue oxygen tension.

Tissue oxygen tension is determined by arterial oxygen tension, arterial oxygen content, oxygen consumption, and perfusion (Hopf et al., 1997). The arterial oxygen content (CaO_2) is the total amount of oxygen dissolved in the plasma and oxygen bound to hemoglobin and is determined by arterial oxygen tension (PaO_2) and hemoglobin concentration (Ganong, 2001). Diffusion of oxygen into the tissue will be affected if there is inadequate dissolved oxygen in the plasma, insufficient oxy-hemoglobin in the arterial blood for different tissues, or a small arterial cellular gradient. Normally, subcutaneous tissue oxygen tension ($PsqO_2$) increases with increase in PaO_2 (Gottrup et al., 1987; Hopf et al., 1997; Jonsson, Jensen, Goodson, West, & Hunt, 1987).

When arterial blood reaches the peripheral tissue such as the muscle, PO_2 is 95 mm Hg at the capillary, about 40 mmHg in interstitial fluid, and 23 mmHg inside the cells. Large initial pressure difference causes oxygen to diffuse very rapidly from blood into tissue. As a result, the capillary PO_2 falls to about 40 mmHg, which is similar to the pressure in the interstitial fluid (Guyton & Hall, 1997). The PO_2 of blood entering veins from high oxygen consumption tissue capillaries is about 40 mmHg. The PO_2 is much higher coming from highly perfused skin that has lower oxygen consumption than that of tissue. As oxygen diffuses away from the capillary, it is consumed by the tissue and the tension of oxygen falls (Gottrup, 1994). Less oxygen diffuses to tissue from the venous

end of the capillary than from the arterial end of the capillary. Eventually, oxygen tension in the tissue is also expected to be similar to that in the venule. Subcutaneous tissue oxygen tension is fairly similar to the oxygen tension in blood from subcutaneous veins, if not contaminated by muscle venous outflow (Gottrup et al., 1987; Gottrup, Gellett, Kirkegaard, Hansen, & Johannsen, 1988).

Fick's Principle

The amount of oxygen delivered to the tissue is determined by a few measurable factors, namely, arterial oxygen tension (PaO_2), hemoglobin concentration (Hb), the percent of hemoglobin saturated with oxygen in arterial blood (SaO_2), cardiac output, oxygen consumption (VO_2), and the affinity of hemoglobin for O_2 (Snyder & Carroll, 1982). Fick's principle states that cardiac output (Qt) is a quotient of oxygen consumption (VO_2) and the arterial venous oxygen content difference ($CaO_2 - CvO_2$).

Fick equation: $Qt = VO_2 / (CaO_2 - CvO_2)$

The total oxygen content is the sum of the arterial oxygen content (CaO_2) and venous oxygen content (CvO_2). The difference in arterial venous oxygen content (Ca-vO2) is the amount of oxygen that is delivered to the tissue. Oxygen consumption (VO_2) is the amount of oxygen that diffuses from the capillaries into all tissues and is about 250 ml/min (Snyder & Carroll, 1982). Since total oxygen delivery is about 1,000 ml/min, the remaining 750 ml/min of oxygen carried in 5 L/min is going back to the heart as venous flow. Applying Fick's principle, the normal venous oxygen content can be calculated. For cardiac output at 5 L/min (50 dl/min), Hb at 15 gm, CaO_2 at 20 ml/dl, and VO_2 at 250 ml/min, CvO_2 equals 15 ml/dl.

$Qt = VO_2 / (CaO_2 - CvO_2)$
$50 = 250 / (20 - CvO_2)$
$CvO_2 = 15$ ml/dl

The normal arterial-venous oxygen content difference is 20-15 = 5 ml/dl. In other words, about 5 ml of oxygen are transported to tissues per 100 ml of blood.

However, oxygen transported to tissues may change depending on variations in cardiac output, fraction of oxygen delivery/arterial PO_2, and hemoglobin level. The total amount of oxygen delivery to tissue remains unchanged even when hemoglobin levels are low, provided that the cardiac output is sufficient. Total oxygen content in blood will be reduced when hemoglobin, arterial oxygen tension, or oxygen saturation levels are abnormal. Table 2 gives some examples of the changes in CaO_2 and oxygen delivery with variations in cardiac output, hemoglobin levels, and oxygen saturation.

Table 2 Examples of Variations in Tissue Oxygen Delivery

Cardiac output CO (L/min)	**Oxygen satura-tion SaO_2 (%)**	**Arterial oxygen tension PaO2 In mmHg**	**Hemo-globin Hb (gm/dl)**	**Arterial oxygen content CaO_2 computed (ml/dl)**	**Oxygen delivered to tissue VO2 (ml/min)**	**Venous blood flow to the heart (ml/min)**	**CvO_2 Venous oxygen content (ml/dl)**	**$C_{a-v}O_2$ Arterio-venous delivery of O_2 (ml/dl)**
5	1.0 (100%)	100	15	20.00	250	1000 - 250=750	750/50 = 15	20-15 = 5.0
5	0.5 (50%)	27	15	10.13	250	506.5 - 250 = 256.5	256/50 = 5.13	10.13 - 5.13 = 5.0
10	1.0 (100%)	100	7	9.68	250	968 - 250 = 718	718/10 0 = 7.18	9.68 - 7.18 = 2.5
5	1.0 (100%)	100	7	9.68	250	484-250 = 234	234/50 = 4.68	9.68 - 4.68 = 5.0
5	0.5 (50%)	27	7	4.77	235			

Oxyhemoglobin Dissociation Curve

The other factors that affect oxygen transport to the tissue are hemoglobin affinity for oxygen and oxygen saturation level. The relationship between the extent of oxygen binding to hemoglobin and PO_2 can be shown in the oxyhemoglobin dissociation curve. The amount of oxygen bound to hemoglobin increases in a sigmoid shaped fashion as the PO_2 increases at 37 °C with a pH of 7.4 and normal 2,3-DPG levels (Nunn, 1989; Schauf, Moffett, & Moffett, 1990). The P_{50} for human hemoglobin oxygen binding is 26.3 mmHg. This is the level of PO_2 required when the hemoglobin is 50% saturated with oxygen (Nunn, 1989). Hemoglobin consists of 4 oxygen molecule binding sites. The binding of the first oxygen molecule to the hemoglobin has a relatively lower affinity. With increase in oxygen saturation, subsequent bindings of the second, third, and fourth oxygen molecule occur more readily (Lavoisier, 1998). Likewise, with low oxygen saturation or low hemoglobin, oxygen content in venous blood is reduced. The first oxygen molecule comes off easily but the subsequent oxygen molecules are harder to unload from the hemoglobin since they are more tightly bound than the first two.

The hemoglobin affinity for oxygen is affected by 4 factors: carbon dioxide (CO_2), pH, temperature, and 2,3 diphosphoglycerate (2,3 DPG) (Schauf et al., 1990), all of which are interrelated. These factors can displace the oxyhemoglobin curve to the right or the left as reflected by changes in P_{50}.

The shift of the oxyhemoglobin curve to the right in response to changes in the blood CO_2 and hydrogen ions is referred to as the Bohr effect (Guyton & Hall, 2000). In the lungs, CO_2 diffuses from the blood into the alveoli, reducing the hydrogen ion concentration and the formation of carbonic acid in blood. This shifts the oxyhemoglobin

curve to the left. The hemoglobin affinity for oxygen increases and percentage of hemoglobin saturation goes up (Guyton & Hall, 2000). A lower PO_2 is required to bind a given amount of oxygen with the left-shift of the curve (Ganong, 2001). The left shift of the curve indicates easier hemoglobin oxygen binding in the lungs but less oxygen is delivered to the tissue. When the blood reaches the tissue capillaries, CO_2 enters the blood from the tissue and displaces oxygen from the hemoglobin. Some of the CO_2 forms carbonic acid and dissociates to give out hydrogen ion, thus lowering the pH. The hydrogen ions may also bind to deoxyhemoglobin and reduce the affinity of hemoglobin for oxygen. The oxyhemoglobin curve shifts to the right, indicating increase release of oxygen to the tissue and a reduction in oxygen saturation (Guyton & Hall, 2000; Schauf et al., 1990). A higher PO_2 is required for hemoglobin to bind a given amount of oxygen in the right-shift curve (Ganong, 2001). In other words, at a given oxygen saturation, the PO2 goes up and more oxygen is unloaded and delivered to the tissue.

Changes in pH values have different implications for the arterial blood flow and venous blood flow. The curve flattens at the right, top where it represents the arterial blood values. (Ganong, 2001). The arterial PO_2 and arterial oxygen saturation is slightly decreased by a reduction in pH. At the venous point where the curve has a steep slope, large amounts of oxygen are given out per unit drop in PO_2 when pH is low. Since tissue PO_2 is closer to the venous PO_2 than the arterial PO_2, the right-shift of the curve will raise the venous PO_2 (Nunn, 1989).

The increase in blood temperature from fever and active skeletal muscles also increases the dissociation of oxygen from hemoglobin (Schauf et al., 1990). Temperature affects the solubility coefficient of oxygen in blood (Nunn, 1989). The effect of changing

temperature on blood PO_2 varies from 7.4% / °C at low saturation, to 1.3% / °C at high PO_2 (Severinghaus, 1979). As temperature increases, blood PO_2 increases in order for the hemoglobin to bind a given amount of oxygen. The production of the metabolite, 2,3-DPG from erythrocyte is increased at low PO_2. The 2,3-DPG reduces hemoglobin affinity for oxygen and therefore shifts the curve slightly to the right (Nunn, 1989; Schauf et al., 1990). With the increases in temperature, hydrogen ion, and 2,3-DPG, the oxyhemoglobin curve shifts to the right and a higher oxygen partial pressure is needed to bind a given amount of oxygen. At the same time, the right-shift curve favors the unloading of oxygen from hemoglobin in the tissue.

If blood flow through the tissue increases, a larger quantity of oxygen is transported into the tissue in a given period of time and tissue PO_2 increases. Most tissues limit tissue PO_2 increase by reducing blood flow in response to increase in PaO_2. The maximum PO_2 increase is up to about 95 mmHg, which is the PO_2 in the arterial blood. As the blood passes through the tissue capillaries, it loses several ml of oxygen to the tissue, which reduces the PO_2 of the capillary blood (Guyton & Hall, 2000). On the contrary, when cells use more oxygen for metabolism, interstitial fluid PO_2 falls. Tissue PO_2 is therefore determined by a balance between the rate of oxygen transport to the tissues and the rate of oxygen utilization by the cells (Guyton & Hall, 1997).

Oxygenation at the Cellular Level

Oxygen is needed in the mitochondria for the generation of adenosine triphosphate (ATP). During aerobic respiration, the enzyme cytochrome oxidase consumes 90% of the body's oxygen and generates ATP by the process of oxidative phosphorylation (Nathan & Singer, 1999). ATP is the principal energy source for all

energy-requiring reactions in all body cells (Nathan & Singer, 1999). When ATP is utilized in the cells to provide energy, it is converted to adenosine diphosphate (ADP) and an inorganic phosphate ion (Guyton & Hall, 1997). As the concentration of ADP increases, it in turn increases the metabolic combination of both oxygen and various nutrients to release energy. This energy, together with an inorganic phosphate ion, is used to reconvert ADP back to ATP (Nunn, 1989).

ADP + inorganic phosphate ion + energy $\leftrightarrow$ ATP

Oxidative phosphorylation is carried out only when the PO_2 within the mitochondrion is above a critical level of 1 mmHg. When the PO_2 falls below this level, anaerobic pathway takes place (Nunn, 1989). Under normal conditions, the rate of oxygen utilization by the cells is controlled by the rate of energy expenditure within the cells, that is, by the rate at which ADP is formed from ATP (Guyton & Hall, 1997). Enzymes in the mitochondria convert the products of carbohydrate, protein, and fat metabolism to carbon dioxide and water via the citric acid cycle (Ganong, 2001). Glucose is degraded to pyruvate in the cells after a series of chemical reactions. When oxygen is adequate, pyruvate enters the citric acid cycle and is metabolized to carbon dioxide and water through the process of aerobic glycolysis. During this process, large quantities of ATP are liberated. If the oxygen supply is not sufficient, most of the pyruvate will not enter the citric cycle but will be reduced to lactate via the process of anaerobic glycolysis. Of interest, aerobic glycolysis is always present in wounded tissue. Oxygen consumption as well as glucose and pyruvate oxidation are unaltered in wounded tissue. Wounding increases glucose uptake and lactate production (Caldwell et al., 1984). Lactate from wound cells, leukocytes, macrophages, endothelial cells and fibroblasts all

acquire their energy by aerobic glycolysis (Guyton & Hall, 2000). Anaerobic glycolysis generates much smaller quantities of energy-rich phosphate bonds than in aerobic respiration (Ganong, 2001). Lungs, heart, and blood vessels ensure cellular integrity and function by maintaining a continuous and adequate supply of oxygen to the cells.

Cellular Oxygenation and Wound Healing

Tissue oxygen consumption is affected by the activation status of the cells, hormones, temperature of tissue, and drugs (Gottrup, 1994). All cells consume oxygen but leukocytes use more oxygen than fibroblasts (Gottrup et al., 1987). Oxygen is needed for bacterial killing, angiogenesis, epithelialization, and collagen deposition (Hopf et al., 1997; Whitney & Heitkemper, 1999). Collagen deposition is proportional to oxygen tension and perfusion (Jonsson, 1991).

With normal perfusion, collagen deposition is enhanced when PaO_2 is increased. Fibroblasts in wounds produce lactate and lactate production increases as oxygen concentration rises in the phagocytic environment (Trabold et al., 2003). The accumulation of lactate enhances the enzymatic activity of dioxygenase prolyl hydroxylase (Zabel, Hunt, Mueller, & Goodson III, 2003). This dioxygenase enzyme removes an oxygen atom from the dissolved oxygen and uses it for collagen peptide synthesis. Collagen synthesis is essential to wound healing.

The major dioxygenase enzymes that aid in the production of collagen use oxygen as a substrate. These oxygenase reactions proceed at a rate based on the local concentration of oxygen (PaO_2) and need a km of 20 mmHg (Zabel et al., 2003). The Km is the substrate concentration that reacts at ½ of its maximum rate (½ Vmax). The rate of oxygenase reactions is at half-maximal when PaO_2 is at 20 mmHg. The rate is maximal

when PO_2 is at about 200 mmHg (Zabel et al., 2003). The PaO_2 of wound fluid in human wounds is about 30-40 mmHg (Zabel et al., 2003), which means that these enzymes function just beyond half capacity and the rate of collagen synthesis is much reduced.

The oxygen consumption and bacteriocidal production of oxidant by neutrophils in wounds also depend on local PO_2. Wound neutrophils are stimulated to produce bacteriocidal superoxide (Allen et al., 1997). A PO_2 ranges from 45 to 80 mmHg is needed for oxidant production at half maximal rate. The maximal rate of oxidant production requires a $PO_2 > 300$ mmHg (Allen et al., 1997). The low PO_2 in wound fluid interferes with oxygen consumption and superoxide production.

Clinically, the probability of ulcer healing in patients with diabetic foot ulcers is low when transcutaneous oxygen tension on the dorsum of the foot is < 25 mmHg (Kalani, Brismar, Fagrell, Ostergren, & Jorneskog, 1999). Low subcutaneous oxygen tension is directly correlated with development of postoperative wound infection (Hopf et al., 1997).

Factors Affecting Tissue Oxygenation

Tissue oxygenation is affected by many factors including oxygen supply, vasoconstriction, temperature, and nitric oxide. Tissue perfusion is considered adequate only when tissue oxygen demand is satisfied (Gottrup, Firmin, Chang, Goodson III, & Hunt, 1983).

Tissue oxygen tension varies during hypoxic and hyperoxic conditions. Gottrup and colleagues (1988) examined oxygen transport when dogs were subjected to hypoxia (fraction of oxygen (FIO_2) decreasing from 0.21 to 0.10) and hyperoxia (FIO_2 increasing from 0.21 to 1.0). Tissue oxygen tension fell during hypoxia. Hypoxia induces

catecholamine production leading to vasoconstriction and subsequent reduction in oxygen tension. Local vasodilatation may occur in some tissues when there is an increased metabolism and a reduced oxygen supply to local blood vessels (Guyton & Hall, 1997). Venous pooling may provide a local oxygen reservoir to increase tissue oxygen tension (Sair, Etherington, Winlove, & Evans, 2001).

Only minor changes were found in oxygen transport measurements during hyperoxia (Gottrup et al., 1988). Supplemental oxygen has been shown to increase PaO_2 which in turn increases tissue oxygen tension (Gottrup et al., 1987; Gottrup et al., 1988; Hopf & Hunt, 1994; Jonsson, 1991). Greif and colleagues (2000) studied the effect of supplemental oxygen administration during the peri-operative period on the incidence of wound infection on surgical patients (n=500). Supplemental inspired oxygen was given at either 80% or 30% during the operation and for 2 hours afterwards. Patients (n=250) who received 80% inspired oxygen had a higher subcutaneous oxygen tension and a lower incidence of infection than those (n=250) who received 30% oxygen (p=0.01). The incidence of surgical wound infections in patients who received 80% oxygen was lower than in patients receiving 30% oxygen (Greif, Akca, Horn, Kurz, & Sessler, 2000).

Hyperbaric oxygen therapy has been shown to increase the delivery of oxygen to ischemic tissues and reduce edema (Bouachour et al., 1996). The large amount of dissolved oxygen increases tissue oxygen tension, lengthens the diffusion distance of oxygen in the arterial end of the capillary (Gottrup, 2002), and delivers more oxygen to tissue.

Hemoglobin and Oxygen Delivery

Hemodilution has little effect on oxygen delivery to the tissue so long as the volume of flow is maintained. Due to compensatory increase in cardiac output, increase in subcutaneous blood flow, and decrease in blood viscosity, subcutaneous oxygen tension remains close to normal in isovolemic anemia (Hopf et al., 2000). Even in severe isovoloemic anemia with a hemoglobin concentration of 50 g/L, the level of subcutaneous oxygen tension was maintained at baseline levels in healthy volunteers (Hopf et al., 2000). Since subcutaneous oxygen delivery depends on diffusion across a long intercapillary distance, the oxygen carrying capacity of hemoglobin contributes a relatively minor fraction of subcutaneous tissue oxygen (Hopf et al., 2000). Anemia seems to have little effect on tissue oxygen tension, provided that the heart pumps sufficient volume and the tissue is normally perfused (Gottrup, 2002).

Vasoconstrictive Effects

Microcirculation supplying the skin and subcutaneous tissue is under neural and humoral adrenergic control. The sympathetic system can be stimulated by circulating catecholamines such as epinephrine and norepinephrine leading to vasoconstriction, which in turn may reduce PO_2 in subcutaneous tissue. Vasoconstriction may be caused by catecholamine excess due to hypovolemia (Gottrup et al., 1987), epinephrine infusion (Jensen et al., 1985), pain (Akca et al., 1999), cigarette smoking (Jensen et al., 1991), and changes in temperature (Hopf et al., 2000).

A mild reduction (5%) in blood volume due to blood or fluid loss will produce vasoconstriction (Gottrup et al., 1987). Poorly controlled surgical pain reduces tissue oxygen tension (Akca et al., 1999). The effects of epidural anesthesia on $PsqO_2$ was

tested in healthy volunteers (n=15)(Treschan et al., 2003). Subjects underwent epidural, general, and combined epidural and general anesthesia while PsqO2 was measured in the arm and thigh. General anesthesia influences peripheral perfusion by reducing the threshold for thermoregulatory vasoconstriction by 2°– 4°C and dilating peripheral vessels in both arms and legs (Treschan et al., 2003), resulting in an increase in subcutaneous oxygenation in extremities. When epidural anesthesia is used instead, it causes a lower-body sympathetic block that produces arteriovenous shunt vasodilation in the legs and a slight compensatory constriction in the arms. Consequently, epidural anesthesia in this study significantly increases subcutaneous oxygen partial pressure in the thighs by 9 ± 2 mmHg (from 54 ± 8 mmHg to 63 ± 7 mmHg) without altering oxygen tension in the arms ($p<0.001$) (Treschan et al., 2003).

Adequate pain control has been shown to increase wound tissue oxygen tension postoperatively. Buggy and colleagues (2002) examined the effect of pain control on wound oxygen tension in patients after major abdominal surgery. Patients were randomized in either the epidural group (combined general-epidural anesthesia followed by epidural post-op analgesia) (n = 16) or the intravenous analgesia group (general anesthesia followed by intravenous morphine analgesia) (n = 16) (Buggy, Doherty, Hart, & Pallett, 2002). The subcutaneous oxygen sensor and temperature sensor were placed subcutaneously in the wound before closure and removed after 24 hours. The mean subcutaneous oxygen tension decreased rapidly within the first three hours and then continued to decrease in a linear fashion. The epidural group showed significant higher mean tissue oxygen tension ($p = 0.002$) and lower pain score ($p < 0.02$) than the intravenous group. Mean post-op tissue temperature started at 35 °C and gradually

increased to 38.5 – 39 °C in both groups. Buggy and colleagues also found a higher subcutaneous oxygen tension after breast reconstruction in women receiving paravertebral analgesia than in women receiving intravenous morphine analgesia (Buggy & Kerin, 2004). The author suggested that higher subcutaneous oxygen tension is associated with superior analgesia. Adequate pain control promotes systemic vasodilatation, which in turn improves tissue perfusion and tissue oxygen tension.

Temperature

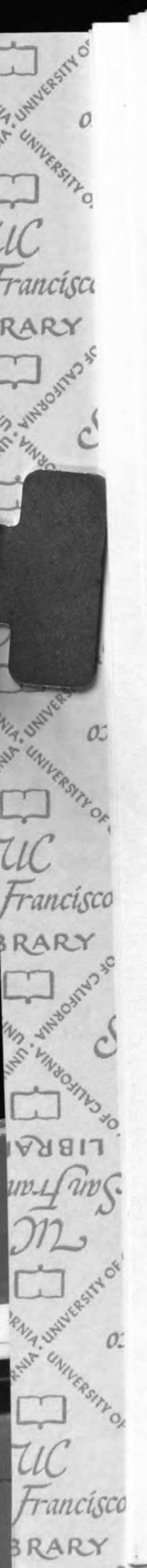
Thermoregulation affects local subcutaneous tissue blood flow. Heat loss in the subcutaneous tissue and skin is thermoregulated by vasoconstriction and vasodilation (Hopf et al., 2000). Subcutaneous temperature correlates with subcutaneous perfusion. In normothermic people, a subcutaneous temperature of at least 34.5 °C indicates normal perfusion (Hopf et al., 2000). Vasoconstriction occurs when skin temperature is reduced by 0.2 °C from an average of 36.7 °C via central infusion of cold fluid in healthy people (n=16) (Lopez, Sessler, Walter, Emerick, & Ozaki, 1994). In volunteers who were awake, prolonged shivering induced by cold exposure decreased subcutaneous oxygen tension while heat exposure increased subcutaneous oxygen tension (Sheffield et al., 1996). The application of local heat using a subcutaneously implanted oxygen tonometer increased both subcutaneous tissue temperature and PsqO2 (Rabkin & Hunt, 1987). Mean subcutaneous tissue temperature increased by 4 °C while PsqO2 increased by 80% from baseline (Rabkin & Hunt, 1987). Ikeda and colleagues (1998) examined the effect of external skin warming on subcutaneous oxygen tension by placing a radiant-heat bandage on the anterior thighs of healthy volunteers (n=8) (Ikeda et al., 1998). Skin was indirectly heated at 3 different temperatures for 2 hours. Subcutaneous oxygen tension

was significantly increased (50%) when heating was provided at 38 °C with skin temperature at 36 °C (33°C before heating)($p<0.05$) (Ikeda et al., 1998).

Nitric Oxide

Tissue oxygenation is regulated in part by the release of nitric oxide from the vascular endothelium. Endothelium-derived nitric oxide (NO) is one of the factors that plays a role in the control of vascular smooth muscle tone. It is formed by the enzyme NO-synthase which is an arterial vasodilator and an inhibitor of platelet activation (Pohl, Wagner, & de Wit, 1993).

It is generally agreed that an increase in blood flow imposes an increase in shear stress on the endothelial cells of the blood vessel, stimulating NO production (Pohl et al., 1993). In response to NO, the vascular diameter increases, allowing an increase in volume flow. This "flow-dependent dilation" helps to maintain optimal vascular flow in certain tissues such as the muscle when demand for flow is high, such as in exercise or reactive hyperemia. Local warming at 42 °C also caused cutaneous vasodilation involving NO-synthase production (Kellogg, Liu, Kosiba, & O'Donnell, 1999). Reactive hyperemic flow response in the forearm is significantly reduced ($p<0.03$) when endothelium-derived nitric oxide is blocked (Meredith et al., 1996). A reduction in NO may contribute to an increase in oxidant production, increase in neutrophil adhesion, and endothelial dysfunction in ischemia/reperfusion injury (Andonegui & Kubes, 2001). The production of NO thus helps to match tissue oxygen supply and demand (Pohl et al., 1993). The relationship between subcutaneous PO_2 and NO has not been established.



Measuring Tissue Oxygenation

Since tissue oxygen utilization in subcutaneous tissue is constant and the volume of consumption is quite small (Evans & Naylor, 1966), measurement of tissue oxygen partial pressure (PO_2) is a sensitive index of small changes in local blood flow.

Transcutaneous oxygen tension ($PtcO_2$) is a measure of the oxygen present in the skin (Benscoter, Gerber, & Friedberg, 1984). Transcutaneous measurements are dependent on systemic blood flow and arterial oxygen content (Kram et al., 1989). The level of $PtcO_2$ is dependent on the balance between the arterial oxygen partial pressure and metabolic oxygen consumption. Transcutaneous oxygen tension usually follows the trend of the partial arterial oxygen tension and has its own range of normal values. When there is severely reduced cardiac output and peripheral perfusion, $PtcO_2$ is flow dependent (Tremper, 1984), reflecting the capacity of the local circulatory system to carry oxygen to the skin (Benscoter et al., 1984). Measuring $PtcO_2$ is non-invasive. $PtcO_2$ can be a good indicator to measure reduction in capillary flow when skin is compromised.

The transcutaneous oximeter contains a polarographic electrode embedded on a flat sensing surface (Tremper, 1984). An electrode is applied to the skin using an adhesive ring and contact solution. A heating element in the surface electrode controlled by a thermistor warms the skin to a preset temperature (Lusiani, Visona, Nicolin, Papesso, & Pagnan, 1988). The usual selected skin temperature is 42-44 °C. However, the optimal skin temperature for the sensor to detect $PtcO_2$ is 44 °C at which the cutaneous vascular smooth muscle will be completely relaxed with maximum

vasodilation (Schubert, 2000). The electrode detects diffusion of oxygen from the skin and heating of the stratum corneum enhances the diffusion (Soini & Takala, 1991).

Heating the skin dilates local capillaries and thereby increases local blood flow. It causes changes in the skin lipid structure which facilitates a faster diffusion of oxygen through the skin (Rabkin, Alena, Morse, Goodson III, & Hunt, 1988). The temperature mediated increase in local perfusion has been criticized as destroying normal homeostasis as it results in an overestimation of perfusion (Rabkin et al., 1988). However, it precludes misdiagnosis of ischemia.

Oxygen Challenge

An oxygen challenge is recommended for $PtcO_2$ measurements. During normal perfusion, subcutaneous oxygen tension increases proportionally with increase in PaO_2 whereas during vasoconstriction or low blood flow, $PsqO_2$ changes little with variations in PaO_2 (Gottrup et al., 1987; Hopf et al., 1997; Jonsson et al., 1987). In well perfused animals, doubling the fraction of inspired oxygen (FiO_2) from 0.21 to 5.0 increased PaO_2 2 to 3 times and induced a 100% increase in $PsqO_2$ (Gottrup et al., 1987). In humans, administering 40-60% (0.4 – 0.6 FiO_2) of oxygen via a simple facemask with oxygen flow set at 7-10 Lpm is considered an oxygen challenge. Giving inspired oxygen magnifies the changes in oxygen tension with changes in flow.

$PtcO_2$ Measurement

Trancutaneous oxygen tension is a sensitive indicator of oxygen delivery to the skin. $PtcO_2$ values vary in different areas of the body. "Normal" values of both measurements are evaluated on the basis of their relationship with perfusion and oxygenation at a given value of the fraction of inspired oxygen.

Problems pertaining to the instrument include artifacts from miscalibration and variability from cellular damage (tubing insertion or heating of skin). Although these technical issues may affect tissue oxygen tension, $PtcO_2$ measurement seems to have high accuracy across subject populations in almost all studies. $PtcO_2$ is a reliable and valid measure of the changes in oxygen delivery to the skin respectively. The accuracy and precision of $PtcO_2$ measurement are shown in Table 3.

Table 3. Measurement of $PtcO_2$

Construct of the measure	**Transcutaneous oxygen tension ($PtcO_2$)**
Instrument	An Electrode Oximeter
Accuracy / Validity	The accuracy of $PtcO_2$ at readings ≤150 mmHg is ± 1% of the value ± 2 mmHg, at readings ≥ 150 mmHg is ± 1% of the value ± 4 mmHg (Novametrix Medical Systems, 1991) Construct validity has been established in the context of its use in peripheral vascular diseases (de Groote et al., 1995), amputation and revascularization (Kram et al., 1989; Liu, 1999). The value of $PtcO_2$ varies in subjects over different parts of the body surface (Schubert, 2000). There are no established criterion validity.
Sensitivity	A more sensitive instrument when skin is warmed to 44°C than when lower temperatures are used. Measurement of tissue oxygen levels during oxygen administration improves the ability to assess wound perfusion (Gottrup et al., 1987) and $PtcO_2$ is an accurate predictor in wound healing. Reported instability due to electrode drift occurs at a rate of ≤ 1mm Hg per hour. Calibration is needed prior to measurement (Wipke-Tevis, Stotts, Williams, Froelicher, & Hunt, 2001).
Specificity	Individual data specificity and sensitivity were established for assessment of PO_2 changes. An established value predicts wound healing in below the knee amputations (Kram et al., 1989).
Precision / Reliability	The percent error associated with intrasensor reliability was 4.8% and 7.2% for intersensor reliability (Wipke-Tevis et al., 2001).
Consistency	Using a heated electrode to increase local perfusion may destroy normal homeostasis. Measurement may change natural phenomenon (Rabkin et al., 1988). Yet, $PtcO_2$ has been consistent in showing trends of changes in skin perfusion (Baldwin, 2001).
Reproducibility	$PtcO_2$ has a wide range of normal values in different disease conditions (Lusiani et al., 1988). It offers a baseline for appraising wound healing (Kalani et al., 1999) and has the potential to record consistent changes over a long-term period in patients with COPD (Melillo, Catapano, Ferrari, & Pedrinelli, 1994).
Repeatability	Some variability on repeated measurements.
Advantages	It is non-invasive. Electrode can be placed on most parts of the body (except thick skin areas such as the plantar aspect of the foot, open areas, bony prominences, or veins) to measure local skin perfusion.
Limitations	Manipulation of local perfusion by heat may not reveal the true oxygen diffusion to the skin. It is possible but not precise enough for gas exchange calculations (Planes, Foray, & Raffestin, 2001). Values may be affected by pressure from the sensor.

Tissue Oxygenation and Pressure Ulcer Development

Subcutaneous tissue oxygenation is controlled mainly by vascular flow, oxygen delivery, and oxygen consumption. Any interference with blood flow or oxygen delivery and transport will impede tissue oxygenation, subsequently leading to tissue ischemia. Consistent, prolonged, unrelieved external pressure deprives the tissue of its oxygen supply. Tissue oxygenation indicates whether tissue receives adequate blood flow. Poor tissue oxygenation is a precursor to pressure ulcer development.

Transcutaneous oxygen tension ($PtcO_2$) was measured in healthy adults (n=28) over the trochanters. Compressive weights from 0 to 1200g in 200g increments were applied by a loading apparatus (Xakellis et al., 1991). As compressive weight increased, $PtcO_2$ decreased (p=0.0001). A compressive weight that ranged from 400-1000g caused $PtcO_2$ to drop to zero. The amount of compressive weight needed for each subject to reach a zero in $PtcO_2$ was different. The authors suggested that variations in compressive weight to reduce $PtcO_2$ to zero could be due to the differences in skin structure that protected it from external pressure (Xakellis et al., 1991). In addition, the ability to deliver oxygen to tissues varies from person to person.

The relationship of pressure and $PtcO_2$ over the sacrum has been examined in several studies. Knight and colleagues (2001) applied pressure to the sacrum of healthy adults (n=14) and measured $PtcO_2$ response. Transcutaneous oxygen tension at the sacrum decreased to 44% and 77% with applied loading pressure at 40 and 120 mmHg respectively (Knight et al., 2001). This study did not describe the actual mean values of $PtcO_2$ at baseline or during loading. In a one group pre-test/post-test study, transcutaneous oxygen tension at the sacrum was monitored in patients prone to tissue

breakdown (n=20) (Bader & Gant, 1988). External pressure was applied for a 10 minute period through an indenter. Pressure was increased until $PtcO_2$ was reduced to below 20mmHg, at which time pressure was removed. Applying external pressures from 22 to 92 mmHg reduced $PtcO_2$ to half of its value as compared to $PtcO_2$ before pressure application (Bader & Gant, 1988). A lesser amount of pressure is needed for $PtcO_2$ reduction in the at-risk sample as in the Bader (1998) study as compared to the aforementioned studies.

In another study on pressure and $PtcO_2$, external pressure was applied on the sacral area using a special device on 16 healthy people (Colin & Saumet, 1996). A significant decrease in $PtcO_2$ occurred when external pressure of 40 mmHg was applied to healthy adults ($p<0.05$) (Colin & Saumet, 1996). As external pressure increased between 65mmHg and 110 mmHg, sacral $PtcO_2$ was reduced to zero (Colin & Saumet, 1996). When $PtcO_2$ reaches zero, all the oxygen supplied to the tissue is consumed by skin metabolism, leaving no excess to reach the surface. It is therefore a reflection of the tolerance of cutaneous circulation to external load (Sangeorzan et al., 1989). The amount of external pressure needed to reduce $PtcO_2$ to zero over muscle is greater than that over bone ($p<0.001$) (Sangeorzan et al., 1989). In particular, skin over bony prominences such as the sacrum is more subjected to reduction in tissue oxygenation than skin over muscle.

All the above studies showed that external pressure is related to reductions in $PtcO_2$. The amount of reduction in tissue oxygen tension varies with different pressure magnitudes and location of pressure application.

Pressure Ulcer and Hip Surgery

Age and Pressure Ulcer

Pressure ulcers occur in all ages, even though there is a positive correlation between age and pressure ulcer development. A national study on pressure ulcer incidence and prevalence in 116 acute care hospitals (n= 17,560 patients) found that pressure ulcers developed in 7% of the patients (n = 383); 73% of which occurred in patients older than 65 years (Whittington, Patrick, & Roberts, 2000). An analysis from a European database on the relationship of medical conditions and pressure ulcer incidence in the ambulatory elderly (n=75,168) also confirmed that pressure ulcer incidence increased with advancing age (Margolis et al., 2003). Medical conditions such as diabetes, vascular disease, hip fracture, Alzheimer's disease, and malnutrition are more likely to develop as a person gets older. All these underlying medical conditions contribute to the risk of pressure ulcer development (Margolis et al., 2003).

Across cultures in the Spanish, Swiss, and German populations, pressure ulcer prevalence also increases with advancing age (over 80 years old) (Casimiro et al., 2002; Perneger et al., 2002; Tsokos, Heinemann, & Puschel, 2000). Since most women outlive men, it is not surprising that subjects who develop pressure ulcers are not only older but are more likely to be female than those who do not develop ulcers (Bergstrom, Braden, Kemp, Champagne, & Ruby, 1998; Tsokos et al., 2000).

Occurrence of Pressure Ulcers

The presence of a pressure ulcer is a significant problem in various settings. The prevalence of pressure ulcers in hospitalized patients is high, ranging between 10% and 18% (Cuddigan et al., 2001). These patients may have existing pressure ulcers or may

have acquired the ulcers during their hospital stay. Patients may also be discharged from the hospital with non-healing pressure ulcers.

A retrospective cohort study of 109 long-term care facilities nationwide showed a pressure ulcer prevalence of 22% (Horn et al., 2002). The mean age of these long-term care residents was 79.9 years. During the 12-week study period, 19% of them developed a new pressure ulcer and 6% who had existing pressure ulcers also developed a new ulcer. Those who developed new ulcers (n=457) were more likely to be female, older, cognitively impaired, and immobile than those who had an existing pressure ulcer (n=534) (Horn et al., 2002).

Pressure ulcers are also present in non-institutionalized people. A survey was conducted to assess the prevalence of pressure ulcers in 177 home health agencies in 19 states with a total of 21,529 patients (Meehan, O'Hara, & Morrison, 1999). The pressure ulcer prevalence was 6.8%. The total number of ulcers reported was 2,526 (an average of 1.7 pressure ulcers per patient) and having a pressure ulcer was the most frequently reported reason for admission to the agency's caseload. The prevalence of pressure ulcers seems to be lower in home care than in hospitals. A community-based home health agency reported a pressure ulcer incidence of 3.6% in patients 60 years and older (Bergquist & Frantz, 1999). Another cross-sectional survey of patients on admission to home care agencies (41 home care agencies in 14 states) was reported (Ferrell, Josephson, Norvid, & Alcorn, 2002). The mean age of the patients was 75 years (n=3048) and 30% were at risk for ulcer development. Pressure ulcers occurred in 9.1%, among whom 37.4% had more than one ulcer and 14% had 3 or more ulcers (Ferrell et al., 2002). Pressure ulcer prevalence in both hospital and long-term care is higher than in

home care. Perhaps, due to the fact that some hospitalized patients die, others go to long-term care facilities, and other with ulcers are discharged with referral for home care. However, pressure ulcers are prevalent in all 3 settings at a high rate.

Aging Skin

Age is an important factor in pressure ulcer development in both the healthcare institution and the home. How the skin changes in response to aging are worth exploring. As a person ages, the elastin content of the soft tissue decreases, making the tissue most susceptible to mechanical stress and subsequently affecting transport of nutrients and lymphatic drainage (Krouskop, 1983). Reduction in collagen synthesis results in lower mechanical strength in tissue. Increased stiffness in tissue may decrease resistance to interstitial fluid flow, reducing the cushioning effect of the interstitial fluids on cells (Krouskop, 1983). There is also reduced total vasculature of the skin, including a reduced number of capillaries per unit skin area (Ryan, 1966). In addition, the natural aging of the skin has been associated with connective tissue damage, decline in the ability to repair damaged DNA, and increased trans-epidermal water loss (Gilchrest, 2003). Thus, the protective function of the skin is impaired in the elderly.

Skin in the elderly responds to thermal stimulus differently than that of younger persons. An early study done to measure skin blood flow using laser-Doppler in long-term care patients indicated that younger individuals (< 60 years) had a greater increase (more than 100%) in skin blood flow over the lateral part of the hip in response to thermal stimulus at 40 °C than those older persons over age 60 ($p < 0.01$) (Ek, Lewis, Zetterqvist, & Svensson, 1984).

External forces such as pressure and shear also affect the aging skin in a different way. When pressure was applied to the heel for 10 minutes in 14 volunteers, there was an increase in water content of epidermis and dermis in young persons, but less so in the elderly as detected by ultrasound scanner (Ryan, Thoolen, & Yang, 2001). The authors postulated that the anatomical structure of the vascular bed of the upper dermis helps to maintain the resilience of the skin in the young but less so in the elderly (Ryan et al., 2001).

Bennett and colleagues (1981) compared ischial arteriolar blood flow in hospitalized geriatric patients (n=14) and healthy young men (n=9) in the sitting position. A hard seat was equipped with devices sensing arteriolar pulsatile blood flow. Average shear forces developed by the geriatric hospitalized group were 3 times higher than that of the young healthy group. Tipping the seat backwards through 20 degrees increased blood flow, reduced pressure, and lowered shear in the geriatric group only (Bennett, Kavner, Lee, Trainor, & Lewis, 1981). Pressure and shear caused a more pronounced effect on skin and blood flow in the elderly than in the young.

Location of Pressure Ulcers

Most pressure ulcers that occurred in hospitalized patients are primarily located in the sacrum, buttocks, and heels (Gunningberg et al., 1999; Young et al., 2002). Across all settings from a post-mortem exam (n=10,222) in Germany, the sacrum was the most frequent location (69.6%) for ulcers that involved the subcutaneous tissue and beyond (Tsokos et al., 2000). Although the nursing care and sleep surfaces may not be the same in patients in every country, location of pressure ulcers in a patient is quite universal.

Variations in microscopic skin structure may contribute to the occurrence of pressure ulcers in certain body locations. External pressures have different effects on skin microcirculation in different areas of the body (Schubert & Fagrell, 1989). A study was undertaken to identify the morphological features of the capillary and elastic fiber distribution of the human skin in terms of susceptibility to pressure sore development. Post mortem microscopic examination revealed densely distributed elastic fibers in the ischial skin but less so in the sacral skin. The sacral skin was found to have the most numerous blood capillaries as compared to the ischial and gluteal skin (Hagisawa, Shimada, Arao, & Asada, 2001). Skin with the most capillary supply may suffer the most when external pressure interferes with flow. The less dense distribution of elastic fibers combined with the greater capillary supply make the sacral skin more vulnerable to pressure ulcer development than the ischial or gluteal skin when exposed to external pressure.

Heels are susceptible to pressure because only a relatively thin heel pad covers the calcaneus bone. The heel is subjected to repetitive trauma during normal walking and high pressures when patients lie supine or use the heel as a pivot point when changing position in bed. Data are not available on the morphological features of capillary and elastic fiber in heels as related to pressure ulcer development.

Hip Surgeries

Pressure ulcers may develop in any patient population when risk factors are present. Among those at highest risk for pressure ulcers are patients who have hip surgery (Stotts, 1988; Versluysen, 1986). Surgery in general has been related to pressure ulcer development. A post-operative pressure ulcer incidence of 15.6% was recorded in

elective surgical patients (n=440) whose surgery lasted at least 90 minutes (Nixon et al., 2000). Pressure ulcer incidence in this study was associated with increased number of hypotensive episodes, high mean core temperature (<35.8 °C) during surgery, and reduced mobility the first day after surgery (Nixon et al., 2000). These contributing factors are also prevalent in hip surgeries.

Most of the hip surgeries are performed for hip fractures or osteoarthritis. Total hip replacement is a procedure performed for both hip fractures and osteoarthritis, while open reduction and internal fixation with nailing may be done for hip fractures only.

Total hip replacement (total hip arthroplasty) is a surgical procedure in which the diseased hip joint is resected and replaced with a new artificial weight bearing surface (Tate & Scrulo, 1998). It is indicated for progressive osteoarthritis, inflammatory arthritis, or aseptic necrosis, or following fractures of the acetabulum or the neck of the femur. Total joint arthroplasty may be remarkably effective in relieving pain and increasing mobility (Harrison Principles of Internal Medicine, 2001).

Hip Fractures

About 350,000 hip fractures occur each year in the United States. By the year 2050, it is estimated that there will be 1,800 hip fractures a day. The cost of a hip fracture care averages $33,000 per patient (*American Academy of Orthopaedic Surgeons*, 2001). The risk of hip fractures is higher in women than in men. The risk increases with age (Baudoin, Fardellone, Bean, Ostertag-Ezembe, & Hervy, 1996), low bone density, and history of a previous fracture (Cummings & Melton, 2002).

Over decades, the incidence of pressure ulcer is higher in those with hip fractures as compared to those with osteoarthritis (Versluysen, 1985). Jensen and Juncker (1987)

reported that pressure ulcers occurred in 30% of patients with hip fractures and 4% of patients receiving total hip surgeries due to other causes (Jensen & Juncker, 1987). This may be explained by the fact that patients with hip fractures are generally older and have associated underlying systemic diseases (Versluysen, 1986).

Having hip fracture or hip surgery is associated with increased risk of new pressure ulcer development in people (n=75,168) aged 65 years and older who are not institutionalized (Margolis et al., 2003). The incidence of hospital-acquired pressure ulcer among elderly, hip fracture patients (n=9400) has been recorded as 8.8% (Baumgarten et al., 2003), where pressure ulcer incidence was associated with a minimal surgery duration of 2 hours, long interval between admission and surgery, general anesthesia, and intensive care unit stay (Baumgarten et al., 2003). These factors are all related to extended periods of immobility. One study showed that by optimizing preoperative pain relief and reducing the time from admission to operation, pressure ulcer development was significantly reduced ($p<0.01$) (Hommel, Ulander, & Thorngren, 2003).

Heel Pressure Ulcers in Hip Surgery

Pressure ulcer development was monitored following general surgery that lasted more than 4 hours (Schoonhoven, Defloor, & Grypdonck, 2002). Forty-four patients (21.2%) developed 70 pressure ulcers in the first 2 days following surgery (Schoonhoven et al., 2002). More than half (52.9%) of the pressure ulcers developed on the heels, and 15.7% developed in the sacral area (Schoonhoven et al., 2002). Data compiled by the National Pressure Ulcer Advisory Panel show the heel is the second most frequently affected location for pressure ulcers in the United States (Cuddigan et al., 2001). Yet,

only a few studies reported the prevalence of heel ulcers in the elderly undergoing hip surgery.

Kosiak (1966) in an often cited study reported that heel ulcers were noted in patients with hip fractures (35.2%). A classic study by Versluysen (1985) showed that 32% of the patients (n=283) with either hip fracture or elective hip surgery developed a pressure ulcer. Nearly a quarter (23%) of the ulcers was located on the heel (Versluysen, 1985). Another study on hip surgery patients (n=124) over the age of 65 found that the heel was one of the most common locations of pressure ulcer occurrence (19%) (Gunningberg et al., 1999). The majority of pressure ulcers were diagnosed on the day of surgery. Heel ulcers are prevalent in patients with hip fractures both before and after surgery.

During hip surgery, the elevation of lower extremities may reduce perfusion in the afflicted leg (Martin, 2000). Factors affecting vascular status of the contralateral limb include increased pressure at the groin intraoperatively, the lateral decubitus positioning of the patient in the operating room table, the use of hypotensive anesthesia, and existing vascular diseases (Smith, Pellicci, Sharrock, Mineo, & Wilson, 1989). The proximal femoral blood flow in the operative leg was shown to be reduced during total hip arthroplasty (Hupel, Schemitsch, Aksenov, & Waddell, 2000). Arterial insufficiency was reported after total hip replacement on the contralateral leg (Smith et al., 1989). The compromised vascular status peri-operatively makes both legs more prone to pressure ulcer development.

Postoperatively, an abductor pillow is strapped between the legs while the patient is in bed, securing the legs' position and body alignment. Mobility is limited by the

abductor pillow and may be limited due to fear of prosthesis dislocation (Young, Haughton, & Williams, 1998). Meanwhile, the heels are left in direct contact with the mattress, unprotected from the pressure of the mattress. In addition, subtle movement of the leg causes friction to the heels. Patients (n=291) who score high on risk factors such as friction, shear, and moisture have shown to be at high risk of developing heel ulcers in the hospital (Tourtual et al., 1997).

Furthermore, the non-operative leg is used by the patient for turning and repositioning in bed and the heel of the non-operative leg may be used as a pivot point, thus making the non-operative leg more susceptible to constant friction and pressure. The less mobile heel on the operative side may also be subjected to external pressure from the bed surface leading to heel ulcers (Kosiak, 1966). Patients undergoing hip surgery are at high risk for heel ulcers on both legs due to the combination of increased external pressure from immobility, friction, shear, and decreased blood flow due to pain and positioning during surgery. The mechanism of heel ulcer formation in relation to tissue perfusion and external pressure in the elderly undergoing hip surgery requires further exploration.

Healing of heel ulcers is often impaired due to systemic factors such as vascular problems, renal insufficiency, and diabetes. In addition, local factors like small amount of subcutaneous fat with limited muscle coverage, and difficulty in avoiding external pressure also affect healing of heel ulcers (Treiman, Oderich, Ashrafi, & Schneider, 2000). Consequently, these ulcers usually progress and enlarge, leading to soft tissue infection, bone infection, or gangrene at times (Reyzelman, Lipsky, Hadi, Harkless, & Armstrong, 1999; Treiman et al., 2000). Pressure ulcers on the heels may last for

months, become unresponsive to conservative management, and eventually may require extensive foot debridement, and revascularization (Gentile et al., 1998). When infection persists or the ulcer fails to heal, partial calcanectomy or amputation may become necessary (Bollinger & Thordarson, 2002; Treiman et al., 2000).

Study Model

Pressure ulcers are prevalent in all clinical settings and the prevalence increases as people age. The pathophysiology of pressure ulcer development is complex. It is believed that prolonged or high levels of external pressure acting on bony prominences will cause occlusion of blood flow to the skin and tissue, leading to ischemia and necrosis of cells. The minimal amount of external pressure causing tissue damage is not known. And whether local skin temperature is indicative of pressure ulcer development is still controversial. Etiologies proposed in pressure ulcer development include localized tissue ischemia, changes in hyperemic response, ischemic-reperfusion injury, and microstuctural damage.

Adequate oxygen delivery to the tissue depends on sufficient blood supply from the heart, normal arterial oxygen tension, adequate arterial oxygen content, the amount of oxygen consumption, and adequacy of blood flow. Factors that reduce tissue oxygenation include impaired oxygen supply, vasoconstriction, and reduced nitric oxide production. In addition, external pressure can hinder oxygen delivery, leading to inadequate tissue oxygenation. Tissue necrosis and pressure ulcers may develop.

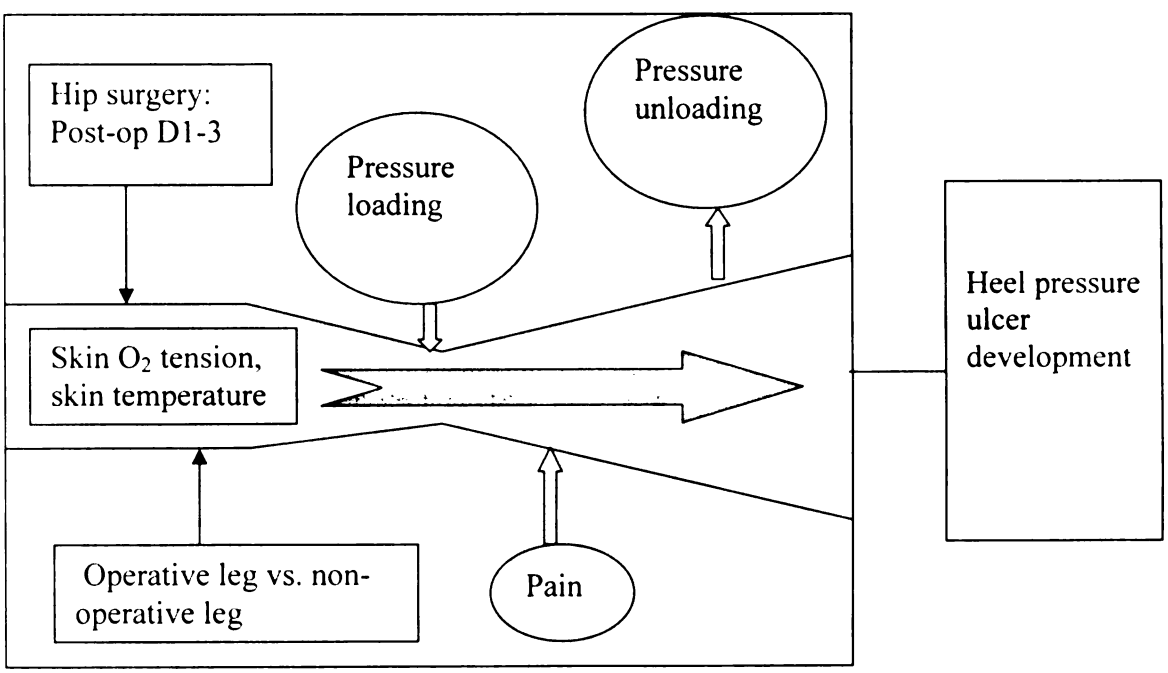
Patients with hip fractures or hip surgeries are at higher risk of developing pressure ulcers than those without hip problems. The heels of both the operative and

non-operative legs are subjected to external pressure. Preventing heel tissue breakdown involves reducing external pressure, and improving heel perfusion and oxygenation.

The study model (Figure 1) thus examines the relationship among heel transcutaneous tissue oxygen tension, heel skin temperature, pain, and external pressure in adults in the first three days after hip surgery.

Figure 1. Study model of the effect of external pressure on skin oxygen tension and temperature in the heels of adults with hip surgery.

The study model illustrates the independent variable of pressure loading/unloading and the dependent variables of skin oxygen tension, skin temperature, and pressure ulcer development. The effect of pain is also examined. The leg with hip surgery versus the leg without surgery is a control/comparison condition.



CHAPTER III

METHODOLOGY

Design

A one-group, prospective, repeated measures design was used.

Sample

The inclusion criteria for the study were as follows: patients 21 years or older undergoing hip surgery, both legs must be present, a score ≤ 5 on the Mental Status Questionnaire (MSQ), an ankle-brachial index (ABI) ≥ 0.9 in both legs, intact sensory perception on the plantar surface of the feet of the operative and non-operative legs measured with a 5.07 Semmes-Weinstein (S-W) monofilament, and if diabetic, a fasting plasma glucose < 140 mg/dL or random plasma glucose range from 120-180 mg/dL before surgery. Exclusion criteria were existing chronic foot ulcers and medical conditions that result in carbon dioxide retention, including documented chronic obstructive airway diseases.

A score of > 5 errors on the MSQ implies moderate or severe mental impairment. An ankle-brachial index (ABI) of < 0.9 indicates the presence of lower extremity arterial disease (Bonham & Flemister, 2002; Whiteley, Fox, & Horrocks, 1998; Zink, Rousseau, & Holloway Jr, 1992). Tissue blood flow and oxygenation to the foot may be affected. Diabetic neuropathy is caused by the nerve damage in the sensory, motor, and autonomic nervous systems. Diabetic neuropathy mainly affects unmyelinated afferent fibers and impairs the vasodilation function in cutaneous blood flow (Stansberry et al., 1999). Therefore, subjects with a loss of sensory perception on the foot were excluded. Subjects with diabetes would be included if blood sugar was within the acceptable values before

surgery. The desirable perioperative control of blood glucose ranged from 120-180 mg/dL or a fasting blood glucose < 140 mg/dL (Gavin, 1992; Marks, 2003). Subjects with existing chronic foot ulcers were excluded as there is potential for tissue inflammation, affecting oxygen delivery to the foot. Since subjects receive an oxygen challenge during the study, those with carbon dioxide retention conditions were excluded as oxygen would reduce the hypoxic stimulus to breathe.

Sample Size Calculation

Studies comparing $PtcO_2$ values between an operative leg and a non-operative leg have not been found. $PtcO_2$ on the trochanter of one leg was measured against the other leg in normal subjects as part of a study (Liu, 1999). The effect size was 0.89. If this effect size was used, estimated sample size would be close to 22. Planned sample size was 34 to allow for drop-outs and death. A preliminary power analysis on 9 subjects showed that there were significant differences in heel $PtcO_2$ in both legs during loading and unloading as compared to the preload when subjects were measured on room air, using a 0.05 α level with 80% power. The sample size requirement varied greatly depending on the pair-wise comparisons made on the non-operative and operative leg, the time of measurement, the day of measurement, and whether the subject was having an oxygen challenge. After discussion with the biostatistician on clinical relevancy, a sample size of 20 was recommended for this study with its repeated measures analysis.

Settings and Recruitment of the Sample

Subjects for this study were recruited from the UCSF Medical Center and El Camino Hospital. Both hospitals have a diverse population and culture. According to the California State Census Data Center, 53% of the population in California consists of

minorities. This minority population includes Black or African American (6.4 %), Asian or Pacific Islander (11.1 %), Hispanics (32.4%), and others (3.1%) (*Population by Race/Ethnicity, Incorporated Cities by County*, 2000). While there are some differences in the distribution of minority patients at UCSF and El Camino Hospital, in general, the patient population resembles the population of the State. There has been no data indicating a difference in $PtcO_2$ values by gender, race, or ethnicity. Therefore, no attempts were made to include or exclude any population by gender, race, or ethnicity.

The Procedure for Recruitment

Approval for recruiting subjects, reviewing medical records, and conducting the study were obtained from the UCSF Committee on Human Research (IRB) and the El Camino Hospital Institutional Review Board. Information fliers (Appendix C) describing the study were posted in the orthopedic clinics. So patients attending the clinics might view the flier and contact the researcher directly if interested to learn more about the study.

Initial information about the study was provided to potential subjects by staff of each institution, although the recruitment procedures were different at the two clinic sites. At UCSF, the staff in the orthopedic clinic mailed the recruitment letter (Appendix C) and informational flier as part of the standard mailing to the subjects. The researcher obtained the potential subjects' phone numbers from the clinic staff. The recruitment letter indicated subjects who did not want to participate were to call the researcher, otherwise, the researcher would call them after a week.

At El Camino Hospital, staff working in the admission unit and the orthopedic nurse educator made the initial contact with prospective subjects to determine a subject's

interest in learning about the study, and to introduce the researcher to the potential subject. If a patient was interested in the study and agreed to have the researcher call for further discussion, the phone number of the subject was given to the researcher. The researcher then called the subject and explained the study protocol. Eligibility was determined by chart review, interview with potential subjects, and performing MSQ, ABI, and S-W monofilament tests.

The researcher described and explained the study to the potential subject. All questions were answered. Written informed consent was obtained by the researcher in person. Medical charts were reviewed to screen for exclusion criteria.

Instruments/Measures

Demographic Variables

Demographic variables gathered from the medical record/interview included the subject's age, gender, ethnicity and race, smoking history and current smoking status, medical conditions, type of anesthesia, length of surgery, and estimated blood loss.

The Mental Status Questionnaire (MSQ)

The MSQ is a 10-item questionnaire that was derived from a 31-item Mental Status Questionnaire to screen for cognitive functioning in the older adult (Kahn, Goldfarb, Pollack, & Peck, 1960). It provides a brief, objective, and quantitative assessment that covers orientation in time and place, remote memory, and general knowledge (McDowell & Newell, 1996). The number of wrong answers and unanswered items are counted as errors. Cut-points are determined by the number of errors. A scoring point of 6 -10 generally indicates moderate to severe mental impairment (Zarit, Miller, & Kahn, 1978). In fact, the increasing number of errors is associated with more

severe chronic brain syndrome rating (Kahn et al., 1960). The test-retest reliability was 0.87 at 2-4 weeks (Lesher & Whelihan, 1986). Sensitivity (45% - 94%) and specificity (96% - 99%) varied according to different cut-point (McDowell & Newell, 1996). Correlations reported when comparing with other mental status assessment tools ranged from 0.57 to 0.88 (McDowell & Newell, 1996). A cut-point of ≤ 5 was used in this study.

Ankle-brachial Index (ABI)

ABI is an indirect method of assessing arterial blood flow in the leg. Since ankle/foot pressure varies with central aortic pressure, the values are commonly compared to pressures in the brachial arteries, which are used as a reference and are assumed to be normal (Zink et al., 1992). ABI is obtained using a hand-held Doppler (Park Electronics Model 840, Aloha, Oregon) and sphygmomanometer to measure the systolic pressures in the brachial, dorsalis pedis and/or posterior tibial arteries (Bonham & Flemister, 2002). If arterial blood flow is normal, the pressure in the foot or ankle should be equal or only slightly higher than that in the arm. An index of 1.0 to 1.1 is considered normal, whereas below 0.9 indicates the presence of lower extremity arterial disease (Bonham & Flemister, 2002; Whiteley et al., 1998; Zink et al., 1992). ABI less than 0.9 in either leg was used as one of the exclusion criteria in this study.

Pulse Oximeter

Arterial oxygen saturation (SaO_2) was measured by a handheld pulse oximeter (Novametrix, model 512). The oximeter probe determined SaO_2 by sensing differences in the absorption of 2 wavelengths of light: red light and infrared light. Hemoglobin

saturated with oxygen absorbed more infrared light and less red light. The accuracy of this oximeter is 80-100% ± 2% SpO2 ± 1 standard deviation.

Heel Skin Oxygen Tension

Transcutaneous oxygen tension ($PtcO_2$) is a measure of the oxygen diffusion from the dermal capillaries to the skin surface (Talbot et al., 1996). The measurements are dependent on systemic blood flow and arterial oxygen content. A change in heel $PtcO_2$ is indicative of a change in oxygenation in the heel skin. It is measured noninvasively with a Novametrix Transcutaneous Oximeter (Model 840) (Novametrix Medical Systems, 1991). A modified Clark electrode is applied to the skin using a double-sided adhesive ring and contact solution. The local skin is warmed by the sensor to 44°C, enhancing diffusion of oxygen through the skin to the sensing electrode (Soini & Takala, 1991).

The accuracy of $PtcO_2$ oximetry at readings ≤ 150 mmHg is ±1% of the value ± 2 mmHg, at readings > 150 mmHg is ± 1% of the value ± 4 mmHg (Novametrix Medical Systems, 1991). Construct validity of using $PtcO_2$ as an indicator of skin blood flow has been established (Baldwin, 2001; de Groote et al., 1995; Kram et al., 1989; Liu, 1999; Planes et al., 2001). The value of $PtcO_2$ correlates appropriately with specific subject condition. Sensitivity is greater when skin is warmed to 44°C than when lower temperatures are used. Reported instability due to electrode drift occurs at a rate of 1 mmHg per hour. Calibration of the machine is required before starting the measurement (Wipke-Tevis et al., 2001).

Skin Temperature

Heel skin temperature was measured with the RSP Temperature Monitor with the 400 series Thermistors. Skin temperatures are measured from temperature sensors in contact with the skin surface. There is no known correlation between skinfold thickness and temperature variations at a given site (Frim, Livingstone, Reed, Nolan, & Limmer, 1990). Sensors were self-adhered to the skin surface without tape in this study.

Pain

Pain was assessed using the Visual Analog Scale (VAS) and a picture of a body outline. VAS measures pain intensity on a 10 cm scale (0 mm = no pain, 100 mm = worst pain you can imagine). The person places a mark through the line at the point that best describes how much pain he/she is experiencing at that particular moment (McGuire, 1997). Many studies have demonstrated the reliability of using VAS to assess subjective pain in the clinical setting (Bergh, Sjostrom, Oden, & Steen, 2000; McCormack, Horne, & Sheather, 1988; McGuire, 1997; Salo et al., 2003). Location of pain in any part of the body is identified by the use of a body outline (Melzack, 1975). The drawings of the body are used to assess the spatial distribution of pain.

Assessing Neuropathy

Sensory neuropathy of the feet was assessed by the Semmes-Weinstein (SW) monofilament test (Zangaro & Hull, 1999). The SW monofilaments are calibrated nylon monofilaments attached to a handle. They are used to measure a patient's ability to feel a point of pressure over the metatarsal heads and toes, the medial and lateral midfoot on the plantar surface of the foot (four sites on the sole of each foot) (*Diabetic Foot Screen for Loss of Protective Sensation*, 1998). The monofilament is applied perpendicular to the

skin with enough force to cause the monofilament to buckle for approximately 1 second. These monofilaments generate a reproducible buckling pressure and have sizes from 1.65 to 6.65. The higher the number of the monofilament, the more difficult it is to bend, and a greater force must be applied to bend the monofilament for the patient to feel the pressure (Zangaro & Hull, 1999). Most studies use the 5.07 SW monofilament (Barber, Conolley, Spaulding, & Dellon, 2001; Jeng, Michelson, & Mizel, 2000). The inability to feel a Semmes-Weinstein monofilament of 5.07 (as in diabetic neuropathy) represents a sensory loss of about 98% (Jeng et al., 2000). Subjects with a loss of protective sensation at any one of the eight sites (both feet) were excluded from the study.

Study Variables

The independent variables include *leg* (operative and non-operative), *day* (the 3 post-operative days), *time* (preload, loading, and unloading), and phase (room air and oxygen challenge). The dependent variables are *heel skin oxygen tension*, *heel skin temperature*, and *frequency of pressure ulcer development.*

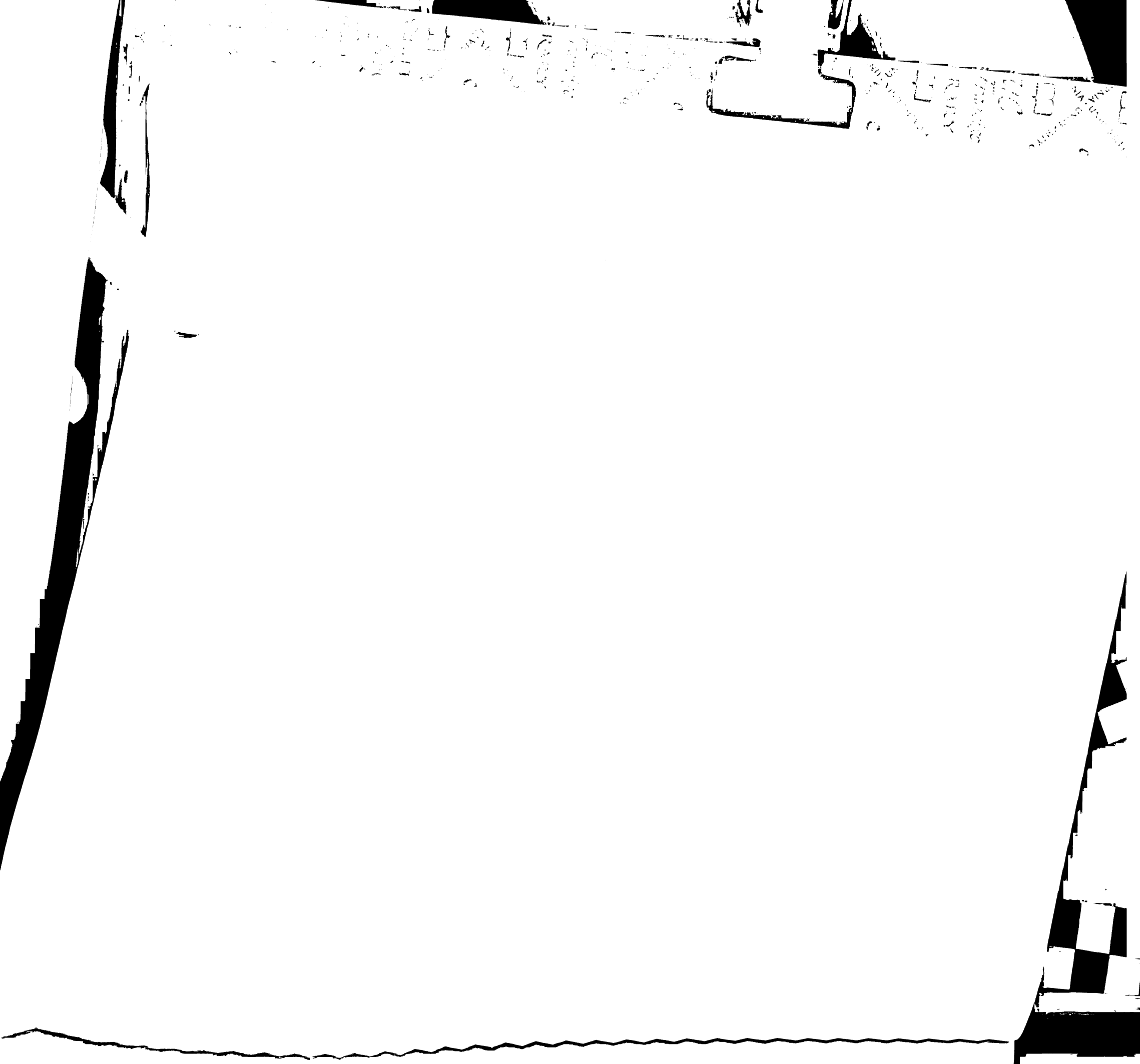
Independent Variables

Leg

Both the operative leg (the leg with total hip replacement) and the non-operative leg were studied.

Day

Days were post-operative day 1 (the day after the operation), post-op day 2, and post-op day 3.



Time

For each of the 3 post-operative day, time was recorded at preload (right before pressure loading after the heels were suspended above the bed surface for 20 minutes), during pressure loading (the 3rd, 6th, 9th, 12th, and 15th minute when heels were placed on the bed surface) and during pressure unloading (the 3rd, 6th, 9th, 12th, and 15th minute when heels were suspended above the bed surface). In addition, time was recorded at the 1st, 2nd, and 3rd minute during pressure unloading to reflect the hyperemic response period.

Phase

The data collection procedure was done in two phases: room air condition (breathing room air) and with an oxygen challenge (breathing 40 – 60% of oxygen via a simple face mask with oxygen flow set at 7-10 Lpm).

Dependent Variables

Heel Skin Oxygen Tension and Heel Skin Temperature

Heel skin oxygen tension ($PtcO_2$) is defined as oxygen delivery to the heel skin. Heel skin temperature is the skin temperature recorded on both heels. Both heel $PtcO_2$ and heel skin temperature were measured at preload, during loading, and during unloading on each day of the measurement period. $PtcO_2$ level was considered as a baseline value after 20 minutes when there was no more fluctuations in reading for one minute. Measurements were recorded every three minutes during the 15-minute loading time, every minute during the first three minutes of unloading, and every three minutes during the remaining 15-minute unloading time.

Pressure Ulcer Frequency

Heel pressure ulcer is defined as any of the following changes on heel skin: a defined area of persistent redness, mottled area, blister, bluish area, open wounds, or blackened area. Frequency is how often per subject if occurs.

Effect Modifier

Pain

Pain is assessed at preload, the 15th minute of loading, and the 15th minute of unloading using the VAS. Subjects are reminded to use the patient controlled analgesia (PCA) and to request for additional pain medicine during the data collection period.

Procedure

Data collection procedure on each of the three post-operative day:

1. With the patient supine in bed, the heels were suspended at about 15° from the bed surface by placing a pillow under the calf for 15 minutes.
2. Pain intensity and location were assessed using Visual Analog Scale (VAS) and a body outline.
3. Oxygen saturation was measured using the Pulse Oximeter. If O_2 saturation was < 95%, oxygen was given until saturation reached 95%.
4. Transcutaneous oxygen ($PtcO_2$) sensors were applied to the heels using double sided adhesive.
5. The temperature sensors were applied to the heel skin.
6. The legs were covered with a single bed-sheet throughout the procedure.
7. Preload $PtcO_2$ and heel skin temperature were recorded.

8. After preload measurements were obtained, loading pressure was applied by placing the heels onto the bed surface.
9. Heel $PtcO_2$ and heel skin temperature were recorded every three minutes during the loading duration of 15 minutes and at the end of loading.
10. At the 14^{th} minute of the loading duration, pain was assessed using VAS and the body outline.
11. After the 15^{th} minute of loading, external pressure was relieved (pressure unload) by lifting the heels and replacing the pillow under the calf to keep pressure off the heels.
12. Heel $PtcO_2$ and heel skin temperature were measured every three minutes for the next 15 minutes during unloading.
13. At the 14^{th} minute of unloading, pain was assessed using VAS and the body outline; and
14. After the 15^{th} minute of unloading, all sensors were removed and the heels were suspended unless otherwise indicated.

The above procedure was carried out twice: when subject was breathing room air and when the subject was breathing oxygen at 7-10 Lpm via a simple face mask.

Data Analysis

The Statistical Package for the Social Sciences, version 13.0 was used to manage and analyze data. Descriptive statistics were used to examine each variable. The overall design was a 3-way repeated measures analysis of variance (RMANOVA) with three within subjects factors: leg, day, and time. The three within subjects factors were analyzed using 1) leg with two levels: operative leg and non-operative leg; 2) day with

three levels: post-operative day 1, day 2, and day 3; 3) time with 11 levels: preload, during loading (loading time at 3rd, 6th, 9th, 12th, and 15th minute), and during unloading (unloading time at 3rd, 6th, 9th, 12th, and 15th minute); and 4) time with four levels: pressure loading at 15th minute and during unloading (unloading at the 1st, 2nd, and 3rd minute).

The analysis allowed trending of $PtcO_2$ and temperature for the overall study period. Then further analysis was conducted to explore the effect of loading, hyperemia, and unloading and also in comparison with preload. The 3-way RMANOVA design allowed for testing of 1) the main effect of day, 2) main effect of leg, 3) main effect of pressure loading time, 4) interaction of day x leg, 5) interaction of day x pressure loading time, 6) interaction of leg x pressure loading time, and 7) interaction of day x leg x pressure loading time. If any of the main effects were significant, a follow-up contrast testing was done. If the interaction was significant, tests of simple effects or tests of trends were performed. For these post-hoc tests, the overall family of contrasts (alpha) was kept at 0.05. The criteria for significance for any one contrast equaled 0.05 divided by the total number of contrasts. Consequently, an alpha of < 0.0167 was the criterion for significance of three contrasts, < 0.01 was the criterion for significance of five contrasts, and < 0.01 was the criterion for significance of 10 contrasts. Correlation analysis was used to determine if 1) heel skin temperature was correlated to heel skin oxygen tension and if 2) demographic variables were correlated to the dependent variables. Data on room air and data on oxygen challenge were analyzed separately but using the same analysis plan. The incidence of heel pressure ulcer and demographic data were described.

CHAPTER IV

RESULTS

Characteristics of the Study Sample

Demographic Data

Forty people were approached for participation in the study. Twenty-two people refused to participate because they did not want to do it (n = 14), their surgery was cancelled or postponed (n = 6), were unable to contact (n = 1), did not meet the age requirement (n = 1). Eighteen subjects consented to participate in the study. Half of the sample was recruited from El Camino Hospital and the other half was from the UCSF Medical Center.

The mean age of the study sample (n = 18) was 58.3 years (SD 16.08), ranging from 26 years to 93 years. There were 50 % males and 50 % females. The majority of the subjects were White (61.1 %), the rest were Asian (22.2 %), Black or African American (11.1 %), and Hispanic (5.6%). One person reported being a smoker. Mean ankle-brachial index (ABI) of the non-operative leg and operative leg was 1.0 (SD 0.08) and 1.0 (SD 0.14) respectively, indicating that having lower extremity arterial disease was unlikely. ABI was not measured in five subjects due to subject refusal or pain on moving of legs. These five subjects had no documented lower extremity vascular disease. No subjects had sensory loss of the feet when tested with the 5.07 S-W monofilament. None of the subjects had diabetes. All of the subjects scored ≤ 5 on the Mental Status Questionnaire (mean 0.39, SD 1.24), indicating that they were cognitively competent.

Subjects in this sample underwent total hip arthroplasty (THA) for the first time or as a revision for the following reasons: osteoarthritis (61.1 %), degenerative joint disease (27.8 %), and hip fracture (11.1 %). Other health conditions included past hip surgeries (44.4 %), hypertension (27.8 %), coronary artery disease (11.1 %), and foot surgeries (5.6 %). Demographic data was shown in Table 4.

Table 4. Demographics of the Study Subjects

Mean age in years	58.3 (SD 16.08)
	Percentage
Gender	
Male	50
Female	50
Race/Ethnicity	
White	61.1
Black or African American	11.1
Asian	22.2
Hispanic	5.6
Primary Diagnosis	
Osteoarthritis	61.1
Degenerative joint disease	27.8
Fracture	11.1
History of disease and treatment	
Hypertension	27.8
Deep vein thrombosis	5.6
Foot surgery	5.6
Coronary artery disease	11.1
Other health conditions	66.7

Peri-Operative Data

On the average, the duration of surgery was 185.7 minutes (SD 96.45). Estimated blood loss during surgery was 655.6 ml (SD 959.92). Most of the subjects (90%) received autologous blood transfusion within the first 2 days of surgery. Fifteen subjects (83.8 %) had general anesthesia only while 3 subjects (16.7 %) had spinal anesthesia. Most subjects' ASA levels (sicker with a higher score) were II (58.8%) and III (29.4%)

while a small percentage was levels I and IV (11.8%). Most subjects (77.8%) denied pain or had a pain score of < 4 while resting in bed. Four subjects (22.2%) had pain scores of 7 – 10 due to muscle spasms. None of the subjects has heel pain. Subjects complained of pain in the operative hip and/or leg. After surgery, all subjects were placed on Hill-Rom pressure-reduction mattresses (Model Advanta) with built-in heel-relief function (set at either comfort mode or pressure-relief mode). The surgery related information is shown on Table 5.

The subjects'room temperature during data collection ranged from 21-23 °C, humidity at 60%. Subjects wore either the thigh-hi or knee-hi compression stockings (66.7 – 88.9 %) during the 3 postoperative days. Less than half of the subjects were treated with the Intermittent Pneumatic Compression Device (IPD) over the 3 days. The IPD delivered intermittent compression at 45 mmHg during the 2-hour data collection visit in 7 subjects (38.9 %) during the 1st post-op day, in 5 subjects (27.8 %) during the 2nd post-op day, and in 4 subjects (22.2 %) during the 3rd post-op day (Table 6).

Table 5. Surgery Related Information

	Percentage	n
Previous hip surgery	44.4	8
Type of anesthesia		
General	83.3	15
Epidural/spinal	16.7	3
ASA classification		
I	5.9	1
II	58.8	10
III	29.4	5
IV	5.9	1
Mean duration of surgery in minutes	185.7 (SD 96.5)	
Mean estimated blood loss in ml	655.6 (SD 959.9)	

Table 6. Leg Conditions and Compression Management during the Study

	Post-op	Non-operative leg		Operative leg	
		%	n	%	n
Swelling	Day 1	38.9	7	38.9	7
	Day 2	55.6	10	61.1	11
	Day 3	38.9	7	44.4	8
Compression stocking	Day 1	88.9	16	77.8	14
	Day 2	88.9	16	77.8	14
	Day 3	77.8	14	66.7	12
Intermittent pneumatic compression device	Day 1	38.9 % (n = 7)			
	Day 2	27.8 % (n = 5)			
	Day 3	22.2 % (n = 4)			

Transcutaneous Oxygen Tension ($PtcO_2$) and Skin Temperature

Descriptive statistics were performed on the study variables. Mean $PtcO_2$ of each heel was examined as was the mean heel skin temperature for each of the loading and unloading periods on each day on room air (Table 7) and with an oxygen challenge (Table 8).

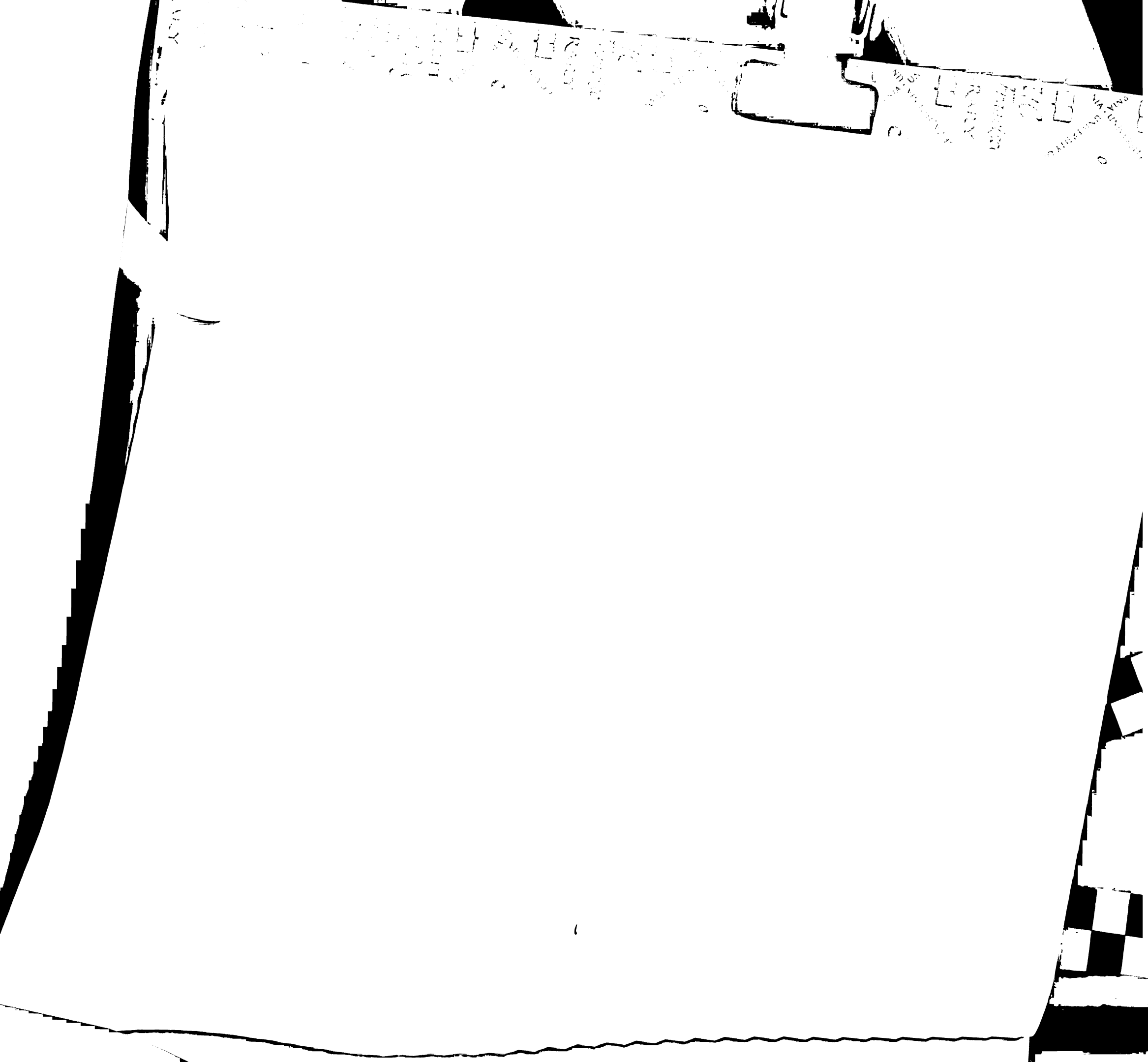


Table 7. Mean Heel $PtcO_2$ and Mean Heel Skin Temperature for Each Leg on Room Air

Postop	Conditions	Non-operative leg		Operative leg	
	Loading/unloading periods	Mean heel $PtcO_2$ (mmHg) (SD)	Mean skin temperature (° F) (SD)	Mean heel $PtcO_2$ (mmHg) (SD)	Mean skin temperature (° F) (SD)
Day 1	Baseline	70.6 (21.16)	90.8 (4.69)	70.2 (25.33)	91.2 (4.78)
	Loading for 15 minutes	60.3 (21.74)	91.5 (4.77)	58.8 (26.37)	91.6 (4.53)
	Hyperemia (unloading for the first 3 minutes)	56.5 (24.23)	90.0 (5.86)	51.8 (27.21)	89.9 (5.86)
	Unloading for 15 minutes	51 (23.59)	91.9 (4.50)	47.6 (27.27)	92.3 (4.36)
Day 2	Baseline	70.8 (22.16)	91.4 (6.01)	74.8 (24.72)	90.8 (6.05)
	Loading for 15 minutes	57.7 (20.32)	91.6 (4.64)	62.5 (22.67)	91.8 (6.01)
	Hyperemia (unloading for the first 3 minutes)	49.0 (18.35)	90.1 (5.66)	53.0 (21.69)	90.7 (5.33)
	Unloading for 15 minutes	43.7 (17.02)	91.9 (4.41)	48.9 (24.31)	91.5 (5.80)
Day 3	Baseline	80.0 (21.41)	89.5 (6.12)	69.2 (17.53)	89.8 (5.96)
	Loading for 15 minutes	68.1 (19.83)	91.6 (6.07)	56.8 (16.21)	91.6 (5.85)
	Hyperemia (unloading for the first 3 minutes)	61.4 (18.90)	90.0 (5.78)	50.0 (16.13)	90.5 (5.45)
	Unloading for 15 minutes	56.1 (18.31)	92.0 (5.67)	46.2 (16.81)	92.1 (5.71)

Table 8. Mean Heel $PtcO_2$ and Mean Heel Skin Temperature for Each Leg with an Oxygen Challenge

Postop	Conditions	Non-operative leg		Operative leg	
	Loading/unloading periods	Mean heel $PtcO_2$ (mmHg)	Mean skin temperature (° F)	Mean heel $PtcO_2$ (mmHg)	Mean skin temperature (° F)
Day 1	Baseline	58.5 (24.41)	91.6 (4.78)	55.8 (29.90)	91.8 (4.67)
	Loading for 15 minutes	52.7 (20.83)	92.0 (4.66)	58.3 (34.23)	92.3 (4.30)
	Hyperemia (unloading for the first 3 minutes)	50.6 (20.34)	92.1 (4.60)	59.0 (35.81)	92.2 (4.36)
	Unloading for 15 minutes	51 (22.65)	92.2 (4.59)	58.9 (33.55)	92.3 (4.46)
Day 2	Baseline	46.6 (22.41)	91.3 (5.68)	50.6 (25.34)	91.9 (5.64)
	Loading for 15 minutes	46.6 (22.42)	91.4 (5.78)	50.6 (27.19)	92.2 (5.68)
	Hyperemia (unloading for the first 3 minutes)	48.1 (25.90)	91.2 (5.66)	51.9 (28.97)	92.1 (5.57)
	Unloading for 15 minutes	48.3 (27.39)	91.3 (5.77)	53.2 (31.58)	92.2 (5.64)
Day 3	Baseline	68.1 (18.80)	90.5 (5.24)	51.0 (21.29)	91.2 (4.07)
	Loading for 15 minutes	63.5 (20.12)	90.9 (4.91)	50.4 (22.42)	91.7 (3.73)
	Hyperemia (unloading for the first 3 minutes)	63.5 (24.25)	90.8 (4.83)	51.8 (28.03)	91.9 (3.57)
	Unloading for 15 minutes	63.3 (27.39)	90.9 (4.77)	54.0 (30.54)	92.2 (3.51)

Hypotheses

1. There will be no overall change in heel skin oxygen tension ($PtcO_2$) response in either leg during the loading and unloading conditions in the first three days after surgery on room air.

The null hypothesis was rejected. Heel $PtcO_2$ in both legs decreased during loading and unloading as compared to preload across all 3 post-operative days. The non-operative and the operative leg did not differ in the heel $PtcO_2$ response to external loading. Repeated measures analysis of variance was used in which the within subjects factors were time (preload, loading, and unloading), leg (non-operative and operative), and day (post-op days 1, 2, & 3).

There was a main effect of time. Heel $PtcO_2$ decreased significantly in loading and unloading as compared to preload baseline ($p = 0.000$) (Figures 2 & 3). Post-hoc tests showed that the decrease was significant at all the five loading and five unloading times ($p = 0.000$). There were no main effects of leg ($p = 0.49$) or day ($p = 0.555$). The change over time did not depend on leg (no leg x time interaction) ($p = 0.389$) or day (no day x time interaction) ($p = 0.401$).

Figure 2. Heel PtcO2 in the non-operative leg on room air during preload, loading, and unloading

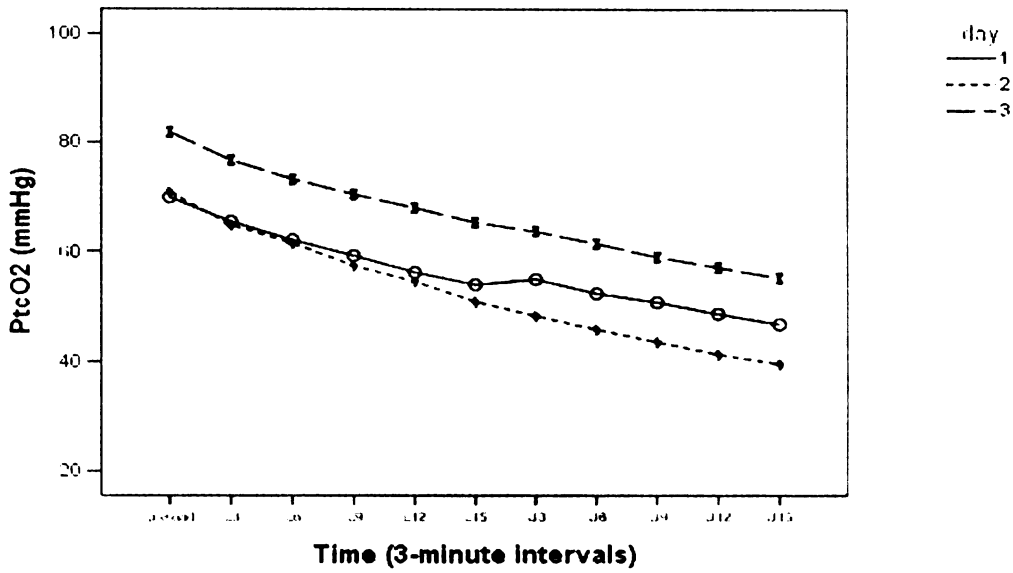
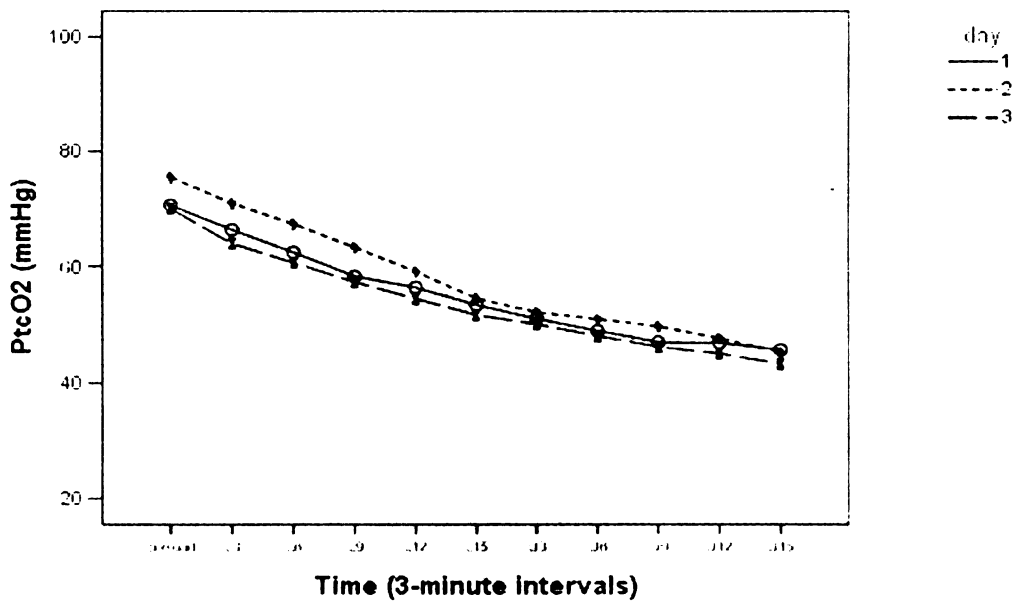


Figure 3. Heel PtcO2 in the operative leg on room air during preload, loading, and unloading.



To further describe the trends of changes over time of both legs on each post-op day, RMANOVA was used in which the within subjects factors were time and leg (Table 9).

Table 9. Overall Trends of Changes in Heel $PtcO_2$ in the 3 post-op Days on Room Air

Dependent variables	Post-op day 1	Post-op day 2	Post-op day 3
Heel $PtcO_2$	n = 16 There was a main effect of time (p = 0.000). There were significant decreasing linear (p = 0.000) and quadratic trends (p = 0.000). There was no main effect of leg (p = 0.861). The change over time did not depend on the leg (no leg x time interaction) (p = 0.335).	n = 16 There was a main effect of time (p = 0.000). There were significant decreasing linear (p = 0.000) and quadratic trends (p = 0.002). There was no main effect of leg (0.239). The change over time did not depend on the leg (no leg x time interaction) (p = 0.715).	n = 16 There was a main effect of time (p = 0.000). There were significant decreasing linear (p=0.000), quadratic (p = 0.000), and cubic (p = 0.004) trends in both legs. The main effect of leg showed that there was a difference in $PtcO_2$ between the legs (p = 0.036). The change over time did not depend on the leg (no leg x time interaction) (p = 0.607).

There was a decrease in $PtcO_2$ in the first 3 days after hip surgery; and yet, there was no difference in heel $PtcO_2$ when the non-operative leg was compared to the operative leg on post-op day 1 (Figure 4) and day 2 (Figure 5). However, the change in $PtcO_2$ was not the same on each leg on post-op day 3 (Figure 6) with operative leg being lower.

Figure 4. Trends of changes in PtcO2 in both legs on room air over preload, loading, and unloading on post-op day 1.

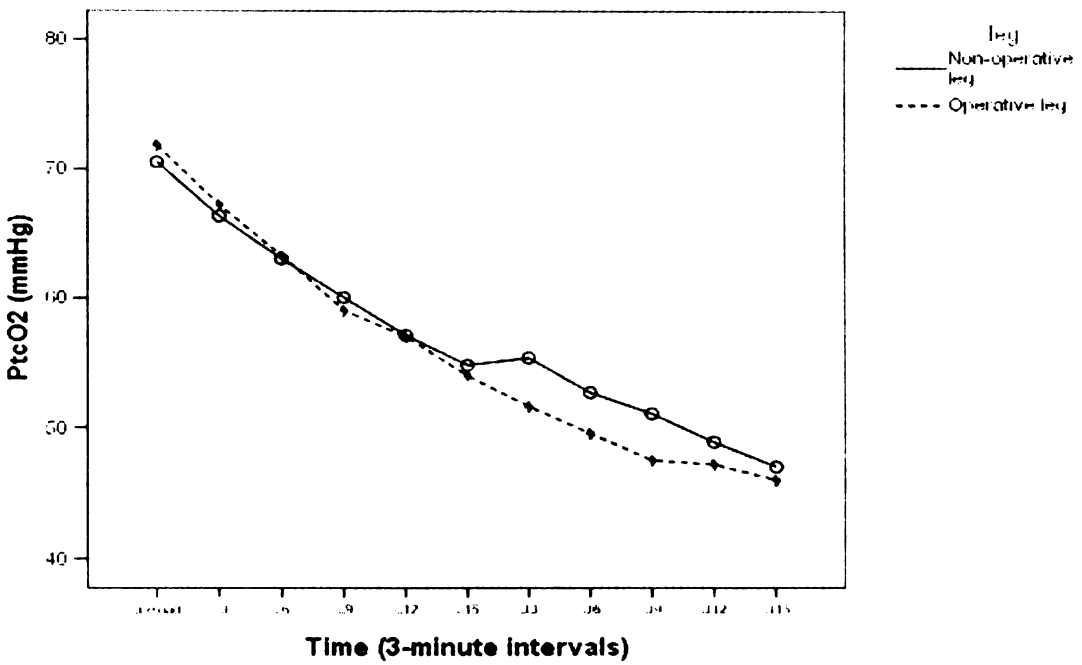


Figure 5. Trends of changes in PtcO2 in both legs on room air during preload, loading, and unloading on post-op day 2.

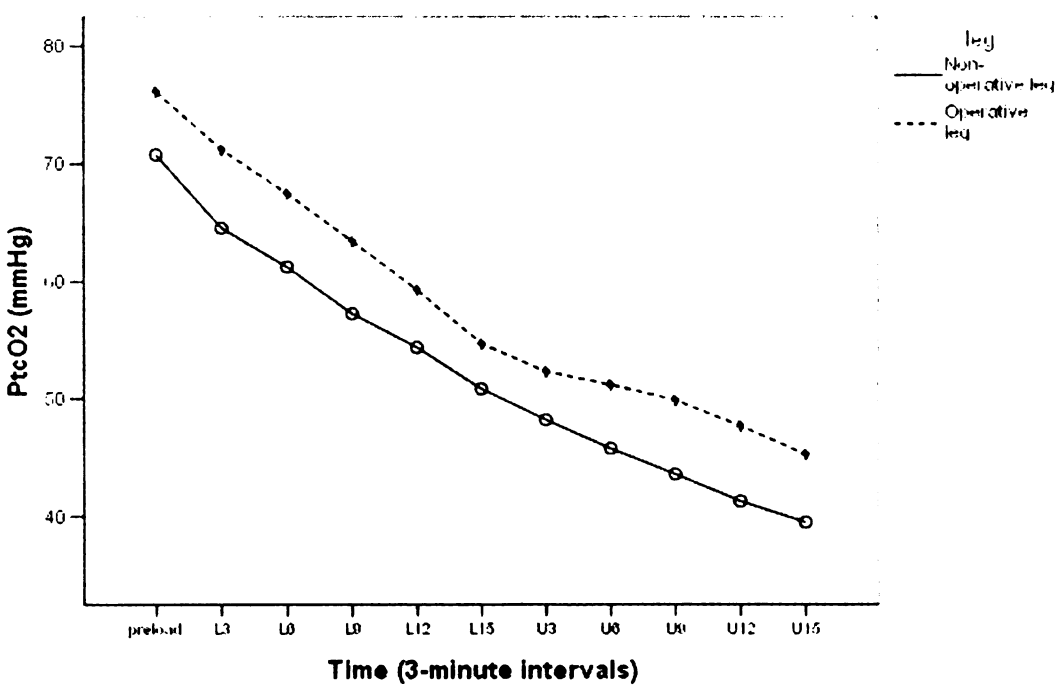
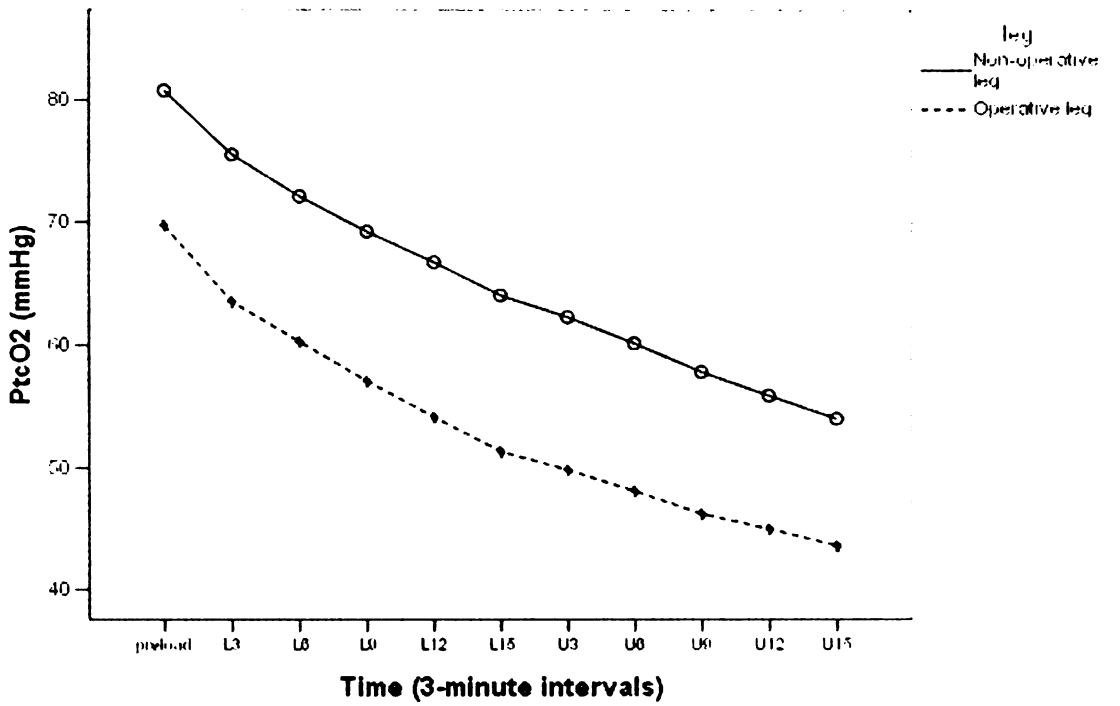


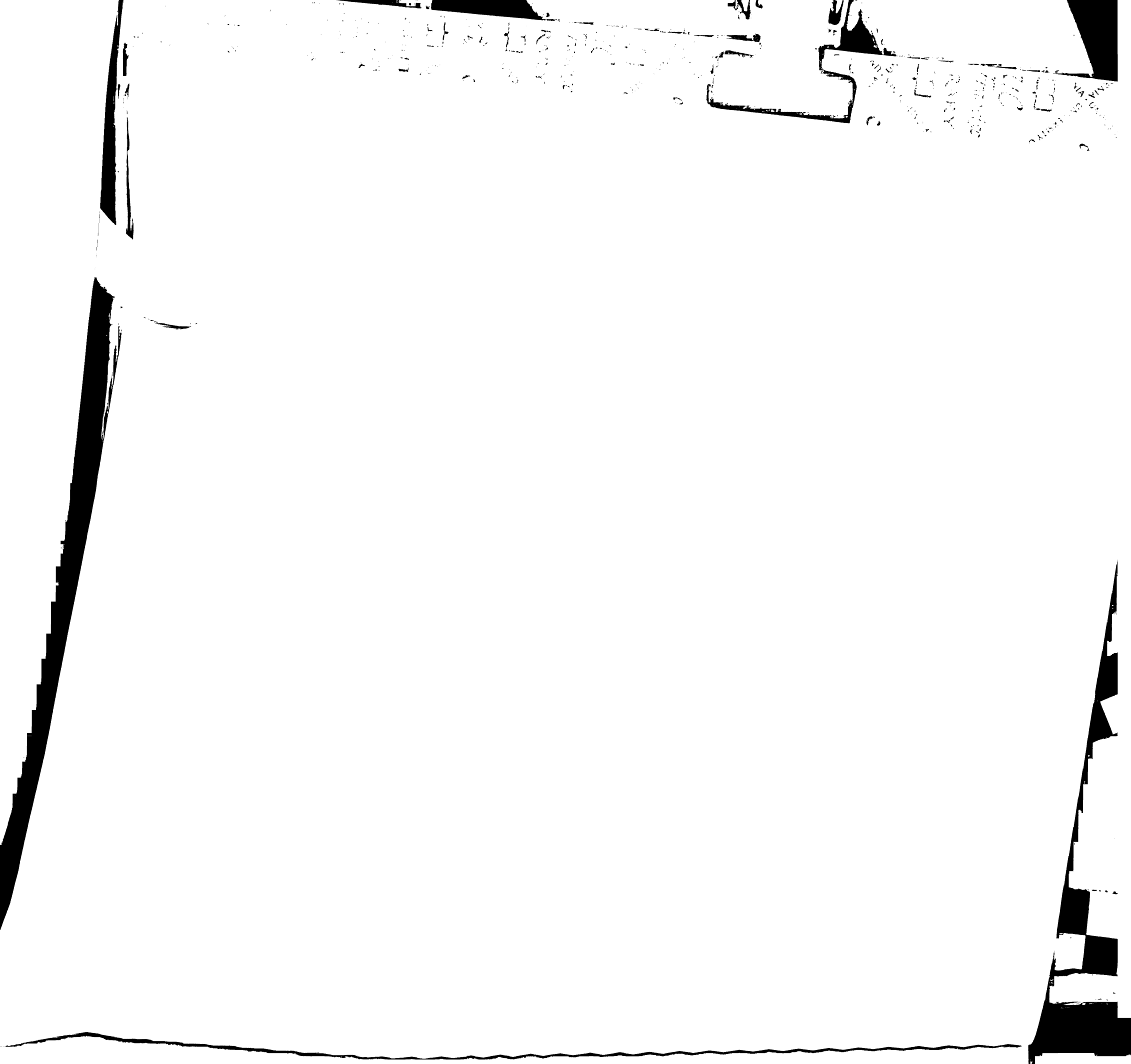
Figure 6. Trend of changes in PtcO2 in both legs on room air during preload, loading, and unloading on post-op day 3.



The above analyses showed that $PtcO_2$ decreased during loading and unloading on each post-op day. To determine if the magnitude of change in $PtcO_2$ is the same on each post-op day, a change score (the difference in $PtcO_2$ between preload and the 15^{th} minute of unloading) was computed. RMANVOA was used in which the within subjects factors were day (days 1, 2, & 3) and leg (non-operative and operative). There were no main effects of day ($p = 0.434$), leg (0.892), or day x leg interaction ($p = 0.874$). The decrease in heel $PtcO_2$ was the same, not dependent on whether is was post-op day 1, 2 or 3.

2. There will be no overall change in heel skin temperature in either leg during the loading and unloading conditions in the first three days after surgery on room air.

The null hypothesis was rejected. There was an overall change in heel skin temperature during loading and unloading on both legs. Repeated measures analysis of



variance was used in which the within subjects factors were time (preload, loading, and unloading), leg (non-operative and operative), and day (post-op days 1, 2, & 3).

There was the main effect of time and leg by time interaction. Heel skin temperature increased over time during loading and unloading ($p = 0.001$) (Figures 7 & 8). Post-hoc tests revealed significance at all 5 loading and 5 unloading times ($p < 0.007$). The change over time depended on whether it was the non-operative or operative leg (leg x time interaction) ($p = 0.031$). The change over time did not depend on the day (no day x time interaction) ($p = 0.289$).

Figure 7. Heel skin temperature in the non-operative leg on room air during preload, loading, and unloading.

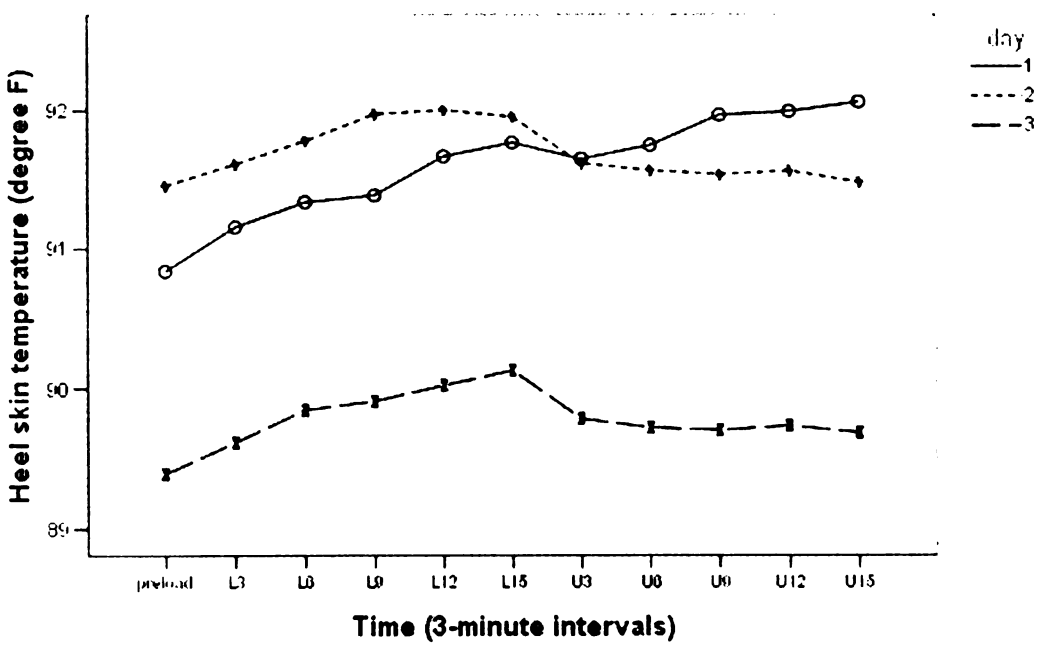
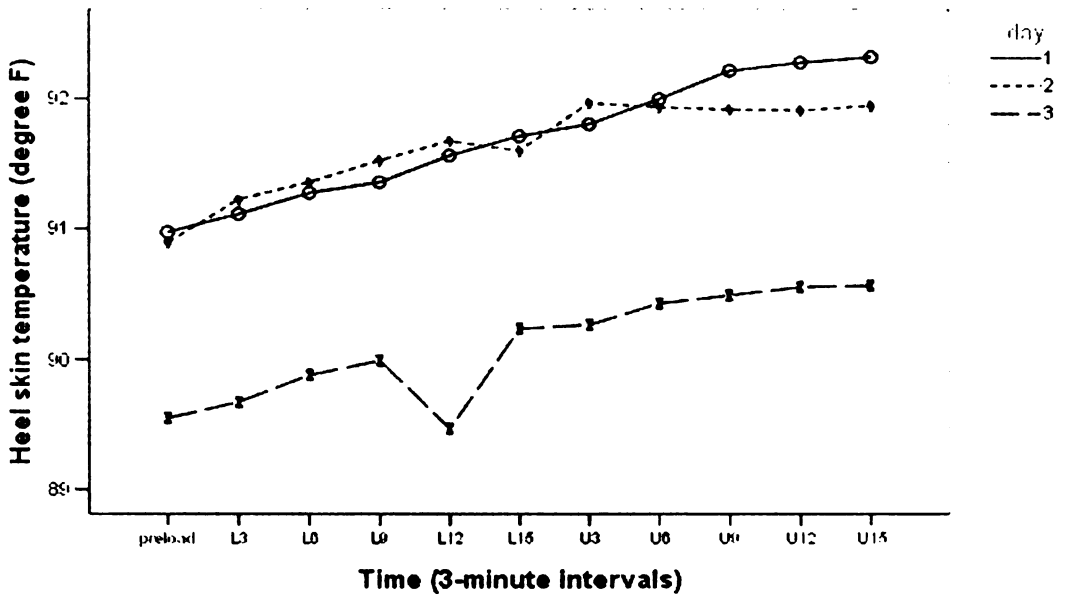


Figure 8. Heel skin termperature in the operative leg on room air during preload, loading, and unloading.



To further describe the trends of changes over time of both legs on each post-op day, RMANOVA was used in which the within subjects factors were time and leg (Table 10).

Table 10. Overall Trends of Changes in Heel Skin Temperature in the 3 Post-op Days on Room Air

	Post-op Day 1	Post-op Day 2	Post-op Day 3
Heel skin temperature	n = 16 There was a main effect of time (p = 0.003). There was significant increasing linear trend (p = 0.004). There was no main effect of leg (p = 0.903) or time x leg interaction (p = 0.055).	n = 17 There were no main effect of time (p = 0.235), leg (0.966), or time x leg interaction (p = 0.273).	n = 17 There was a main effect of time (p = 0.042). There was a significant increasing quadratic trend (p = 0.003). There was no main effect of leg (p = 0.737). The change over time did not depend on the leg (no leg x time interaction) (0.075).

Heel skin temperature on both legs increased during loading and unloading on post-op day 1 and day 3. The increase in skin temperature did not differ in the 2 legs. There was no heel skin temperature change on day 2.

The above analyses showed that heel skin temperature increased on two of the post-op days. To determine if the magnitude of heel skin temperature increase is the same on each post-op day, a change score (the difference in heel skin temperature between preload and the 15^{th} minute of unloading) was computed. RMANVOA was used in which the within subjects factors were day (days 1, 2, & 3) and leg (non-operative and operative). There were no main effects of day ($p = 0.167$), leg ($p = 0.765$), or day x leg interaction ($p = 0.241$). The change in heel skin temperature was the same, not dependent on whether it was post-op day 1, 2, or 3.

3. There will be no difference in heel skin oxygen tension in either leg in the first three minutes during unloading on post-op days 1-3 on room air.

The null hypothesis was rejected. Heel $PtcO_2$ was not the same during the immediate unloading period. The hyperemic response (first 3 minutes of unloading) was not seen in either leg in any of the days. RMANOVA was used in which the within subject factors were time (loading at 15-minute and unloading in the first 3 minutes). Testing of simple effects was done on each leg on each post-op day (Table 11).

Table 11. Changes in $PtcO_2$ in Each Leg from the 15th Minute of Loading to the First 3 Minutes of Unloading on the First 3 Post-op Days on Room Air

Legs	Post-op Day 1	Post-op Day 2	Post-op Day 3
Non-operative leg	N = 16 There was no main effect of time (p = 0.467).	N = 16 There was a main effect of time (p = 0.000). Post-hoc test revealed that $PtcO_2$ decreased significantly at the 2nd min. (p = 0.003) and 3rd min. (p = 0.000) of unloading when compared with loading at the 15th minute.	N = 17 There a main effect of time (p = 0.008). Post-hoc test revealed that $PtcO_2$ decreased significantly at the 3rd minute (p = 0.012) of unloading when compared with loading at the 15th minute.
Operative leg	N = 17 There was a main effect of time (p = 0.010). Post-hoc tests revealed that $PtcO_2$ decreased significantly at the 3rd min. (p = 0.000) of unloading when compared with loading at the 15th minute.	N = 18 There was a main effect of time (p = 0.000). Post-hoc test revealed that $PtcO_2$ decreased significantly at the 2nd min. (p = 0.012) and 3rd min.(p = 0.0001) of unloading when compared with loading at the 15th minute.	N = 18 There was a main effect of time (p = 0.006). Post-hoc test revealed that $PtcO_2$ decreased significantly at the 3rd min. (p = 0.009) of unloading when compared with loading at the 15th minutes.

There was a change in $PtcO_2$ when loading at 15 minutes was compared to the first 3 minutes of unloading (Figures 9, 10, & 11). $PtcO_2$ in the non-operative leg decreased during the first 3 minutes of unloading on post-op day 2 and day 3. $PtcO_2$ in the operative leg decreased in the first 3 minutes of unloading on all 3 post-op days.

Figure 9. Heel PtcO2 in both legs on room air during the first 3 minutes of unloading on day 1.

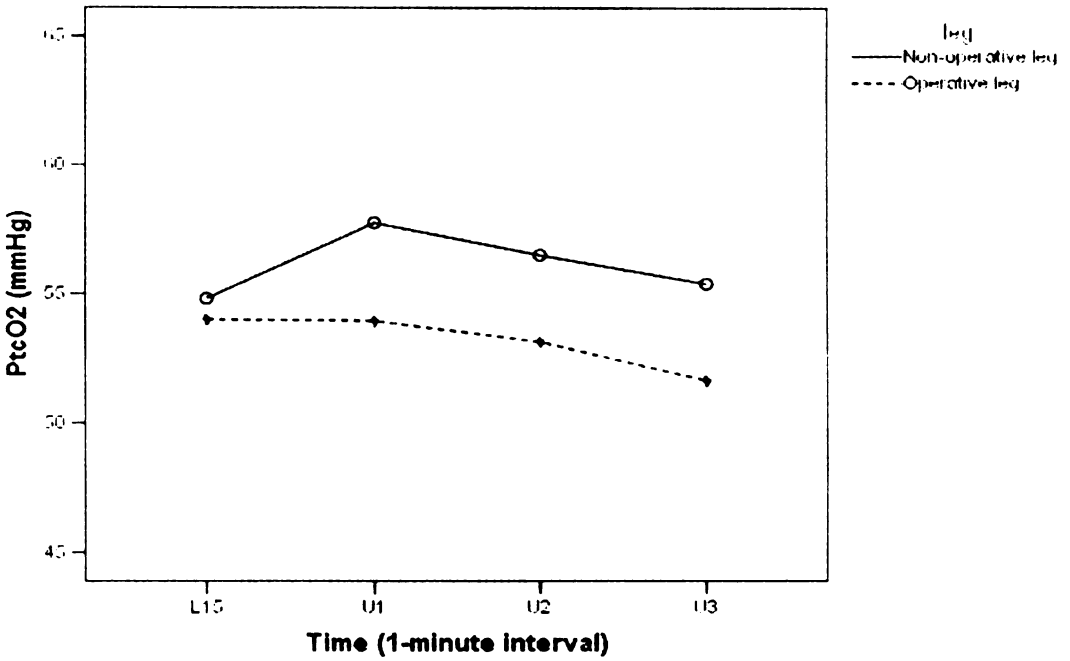


Figure 10. Heel PtcO2 in both legs on room air during the first 3 minutes of unloading on day 2.

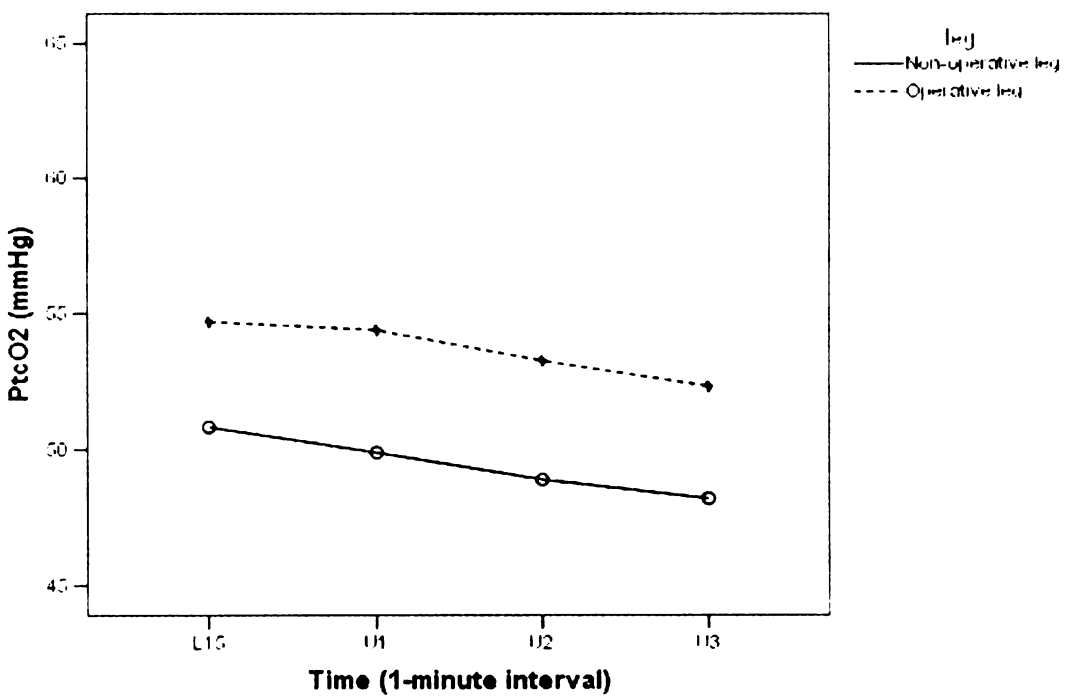
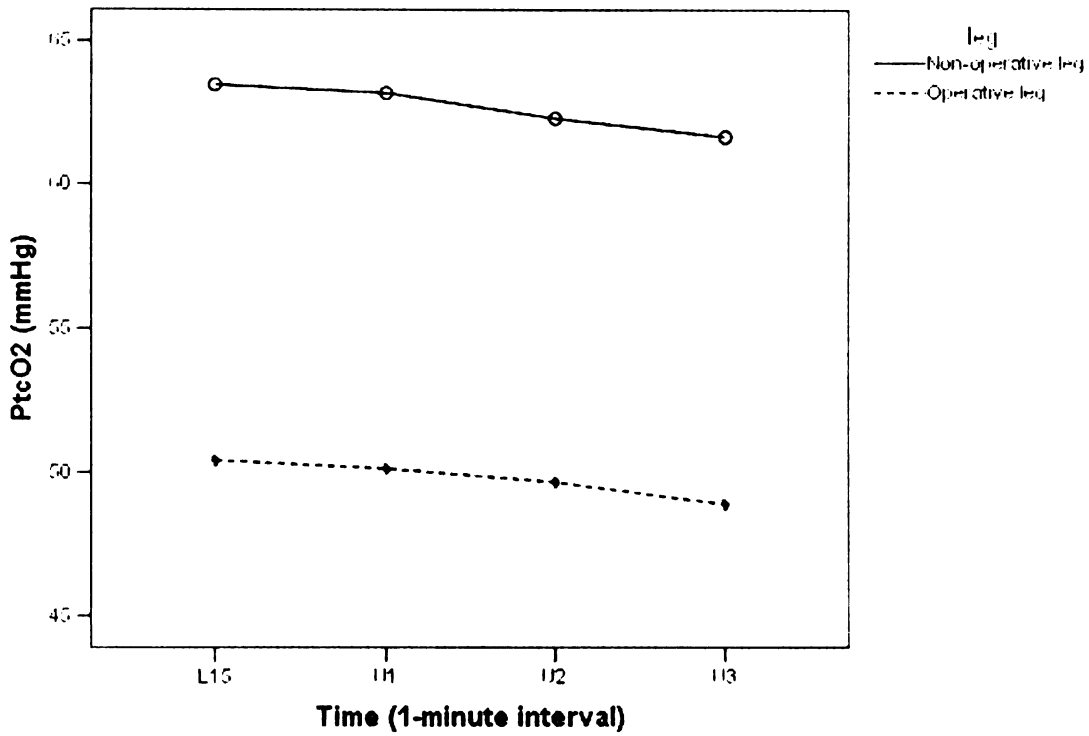


Figure 11. Heel PtcO2 in both legs on room air during the first 3 minutes of unloading on day 3.



4. There will be no difference in heel skin temperature in either leg in the first three minutes during unloading for the first three post-op days on room air.

Heel skin temperature response was different in the two legs. RMANOVA was used in which the within subject factors were time (loading at 15-minute and unloading in the first 3 minutes). Testing of simple effects was done on each leg on each post-op day (Table 12).

The null hypothesis was rejected for the non-operative leg. There was no difference in heel skin temperature from the 15th minute of loading to the first 3 minutes of unloading in the non-operative leg in post-op day 1. On post-op day 2 and 3, heel skin

temperature in the non-operative leg decreased in the first 3 minutes of unloading (Figures 12, 13, & 14).

The null hypothesis was not rejected for the operative leg. Heel skin temperature in the operative leg did not differ in the first 3 minutes of unloading from loading at 15th minute in post-op days 1, 2, and 3 (Figures 12, 13, & 14).

Table 12. Changes in Heel Skin Temperature from the 15th Minute of Loading to the First 3 Minutes of Unloading in Each Leg on the First 3 Post-op Days on Room Air

Legs	Post-op Day 1	Post-op Day 2	Post-op Day 3
Non-operative leg	N = 16 There was no main effect of time (p = 0.107).	N = 17 There was a main effect of time. Heel skin temperature decreased significantly during the first 3 minutes of unloading as compared to the 15th minute of loading (p = 0.021). Post-hoc tests revealed no significance at each contrast (p > 0.02).	N = 17 There was a main effect of time (p = 0.002). Post-hoc tests revealed that heel temperature decreased significantly at the 1st minute (p = 0.002), 2nd minute (p = 0.003) and 3rd minute (p = 0.006) of unloading when compared with loading at the 15th minute.
Operative leg	N = 17 There was no main effect of time (p = 0.259).	N = 18 There was no main effect of time (p = 0.467).	N = 18 There was no main effect of time (p = 0.779).

Figure 12. Heel skin temperature in both legs on room air during the first 3 minutes of unloading on day 1.

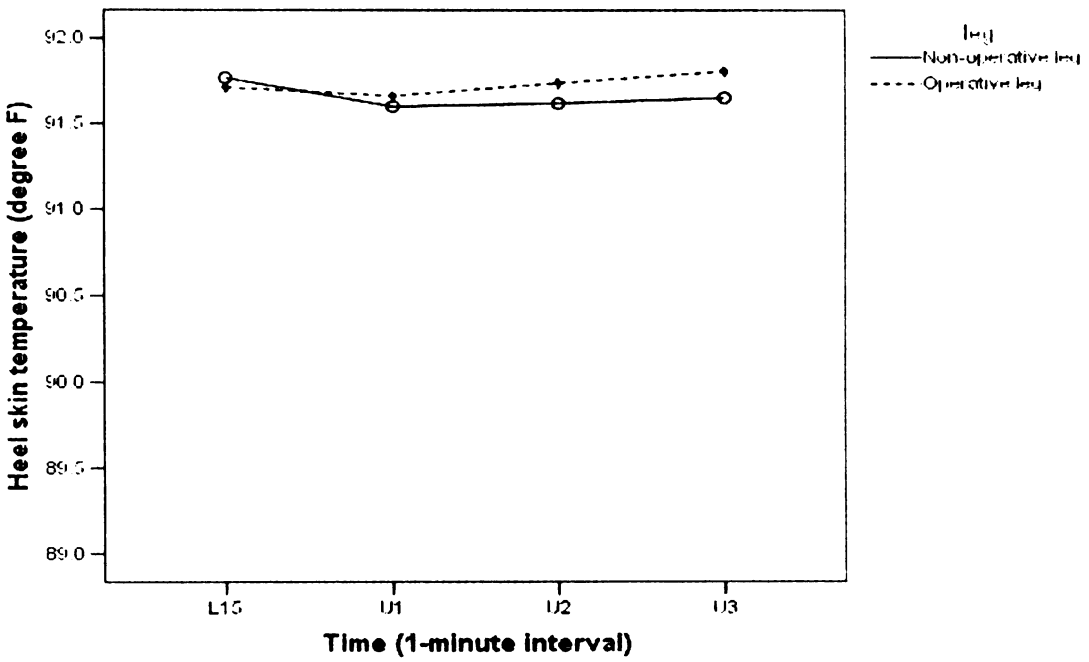


Figure 13. Heel skin temperature in both legs on room air during the first 3 minutes of unloading on day 2.

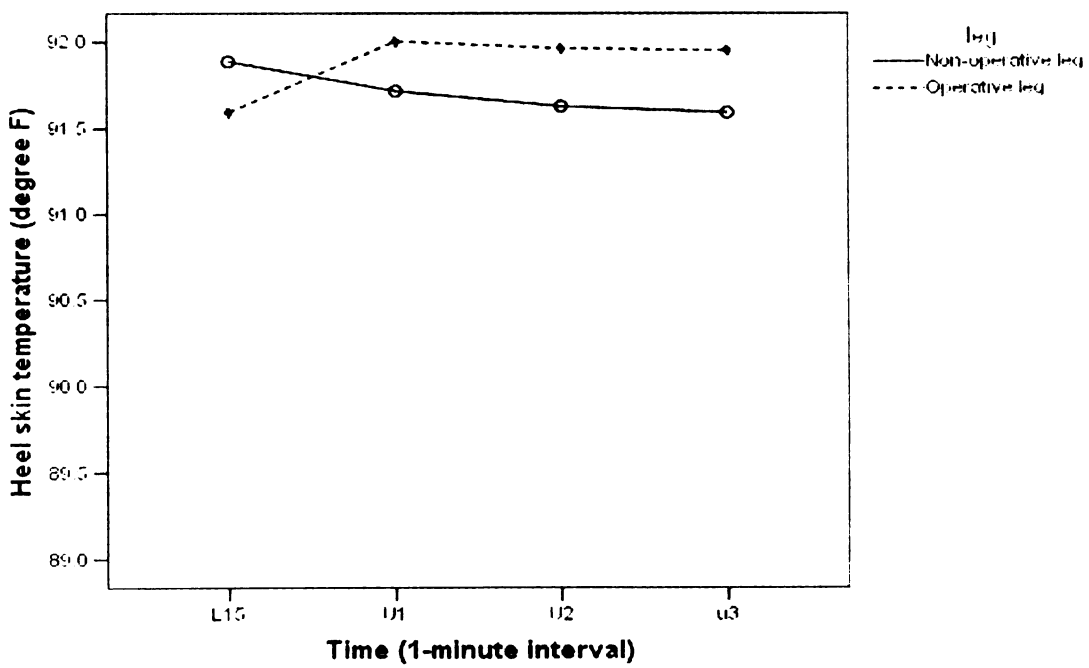
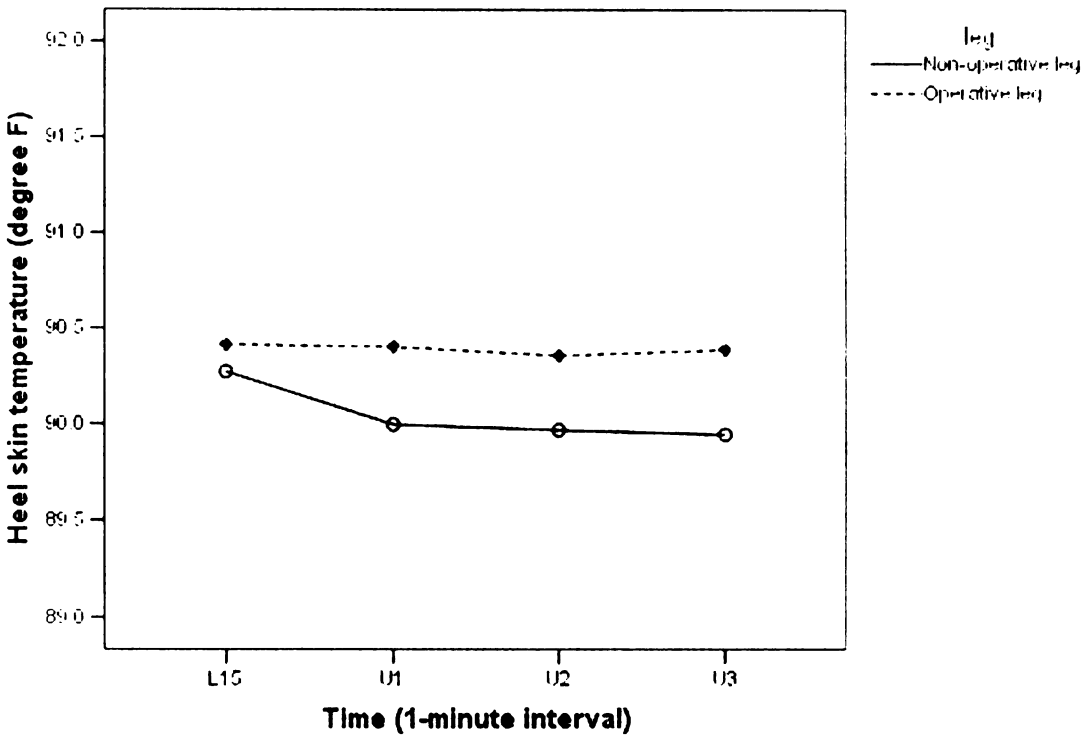


Figure 14. Heel skin temperature in both legs on room air during the first 3 minutes of unloading on day 3.



5. There will be no correlation between changes in heel skin oxygen tension and changes in heel skin temperature between preload and end of unloading in either leg on post-op days 1-3 on room air.

Comparisons were made on the changes in $PtcO_2$ and changes in heel skin temperature from preload to unloading (Table 13) on each post-op day using Pearson's Product Moment correlation.

Table 13. Correlation of the Changes in $PtcO_2$ and Changes in Heel Skin Temperature on Post-op Days 1, 2, and 3 on Room Air

Post-op days	Non-operative leg	Operative leg
Day 1	There was no significant correlation between $PtcO_2$ and heel skin temperature ($p = 0.553$, $r = 0.16$).	There was no significant correlation between $PtcO_2$ and heel skin temperature ($p = 0.815$, $r = -0.061$).
Day 2	There was no significant correlation between $PtcO_2$ and heel skin temperature ($p = 0.928$, $r = -0.025$).	There was no significant correlation between $PtcO_2$ and heel skin temperature ($p = 0.668$, $r = 0.108$).
Day 3	There was no significant correlation between $PtcO_2$ and heel skin temperature ($p = 0.702$, $r = -0.1$).	There was no significant correlation between $PtcO_2$ and heel skin temperature ($p = 0.067$, $r = -0.441$).

The null hypothesis was not rejected. There was no correlation between changes in $PtcO_2$ and changes in heel skin temperature during pressure loading and unloading when compared to preload baseline.

6. There will be no correlation between changes in heel skin oxygen tension in each leg and the development of heel skin breakdown in the first 3 days after surgery on room air.

The null hypothesis was not rejected. The decrease in heel skin $PtcO_2$ over time in both legs in the first 3 post-op days was not related to the development of heel skin breakdown in the first 3 days after surgery. None of the 18 subjects developed any heel skin breakdowns.

7. There will be no overall change in pain scores at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery on room air.

The null hypothesis was not rejected. There was no change in the pain score when the last minutes of loading and unloading were compared with preload on the

first three post-operative days. RMANOVA was used in which the within subjects factors were day (post-op days 1, 2, and 3) and time (preload, loading, and unloading). There were no main effects of day ($p = 0.073$), time ($p = 0.638$), or day x time interaction ($p = 0.453$).

8. There will be no correlation between heel skin oxygen tension in either leg and pain scores at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery on room air.

Comparisons were made between heel $PtcO_2$ on each leg and pain score at preload, the last minutes of loading and unloading on each post-op day using Pearson's Product Moment Correlation (Table 14). Heel $PtcO_2$ in the non-operative leg was inversely correlated with pain score at the last minute of unloading on post-op day 3 only.

Table 14. Correlation of Heel $PtcO_2$ and Pain Score on Post-op Days 1, 2, and 3 on Room Air

Post-op days	Times	Correlation of $PtcO_2$ and pain			
		Non-operative leg		Operative leg	
		r	p	r	p
Day 1	Preload	0.351	0.182	0.155	0.553
	Loading	0.323	0.241	0.117	0.666
	Unloading	-0.187	0.521	-0.117	0.678
Day 2	Preload	-0.142	0.599	-0.343	0.164
	Loading	-0.312	0.239	-0.157	0.533
	Unloading	-0.145	0.592	0.000	0.999
Day 3	Preload	-0.186	0.475	0.142	0.574
	Loading	-0.143	0.598	0.293	0.255
	unloading	-0.556	0.021 *	0.088	0.728

* Significant correlation, $p < 0.05$

9. There will be no correlation between heel skin temperature in either leg and pain scores at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery on room air.

Comparisons were made between heel skin temperature on each leg and pain score at preload, the last minutes of loading and unloading on each post-op day using Pearson's Product Moment Correlation (Table 15). On post-op day 2, heel skin temperature was inversely correlated with pain score in both legs at preload and the last minute of loading.

Table 15. Correlation of Heel Skin Temperature and Pain Score on Post-op Days 1, 2, and 3 on Room Air

Post-op days	Times	Correlation of heel skin temperature and pain score			
		Non-operative leg		Operative leg	
		r	p	r	p
Day 1	Preload	-0.439	0.089	0.337	0.185
	Loading	-0.372	0.172	0.245	0.360
	Unloading	-0.279	0.335	0.086	0.760
Day 2	Preload	-0.749	0.001 *	0.678	0.002 *
	Loading	-0.759	0.000 *	0.709	0.001 *
	Unloading	-0.461	0.063	0.449	0.062
Day 3	Preload	0.148	0.571	0.161	0.523
	Loading	0.179	0.506	0.086	0.744
	unloading	-0.018	0.943	-0.018	0.945

* Significant correlation, $p < 0.05$

10. There will be no overall change in heel skin oxygen tension response in either leg during the loading and unloading conditions in the first three days after surgery with an oxygen challenge.

The null hypothesis was not rejected. When the subjects were receiving oxygen at 7-10 lmp via a simple face mask, there was no overall change in heel $PtcO_2$ in either leg during loading and unloading as compared to preload in all 3 days. Repeated

measures analysis of variance was used in which the within subjects factors were time (preload, loading, and unloading), leg (non-operative and operative), and day (post-op days 1, 2, & 3).

There were no main effects of time (p =0.09), leg (p = 0.794), or day (0.777). There was no leg x time (p = 0.179) or day x time (p = 0.581) interactions (Figures 15 & 16).

Figure 15. Heel PtcO2 in the non-operative leg with an oxygen challenge during preload, loading, and unloading

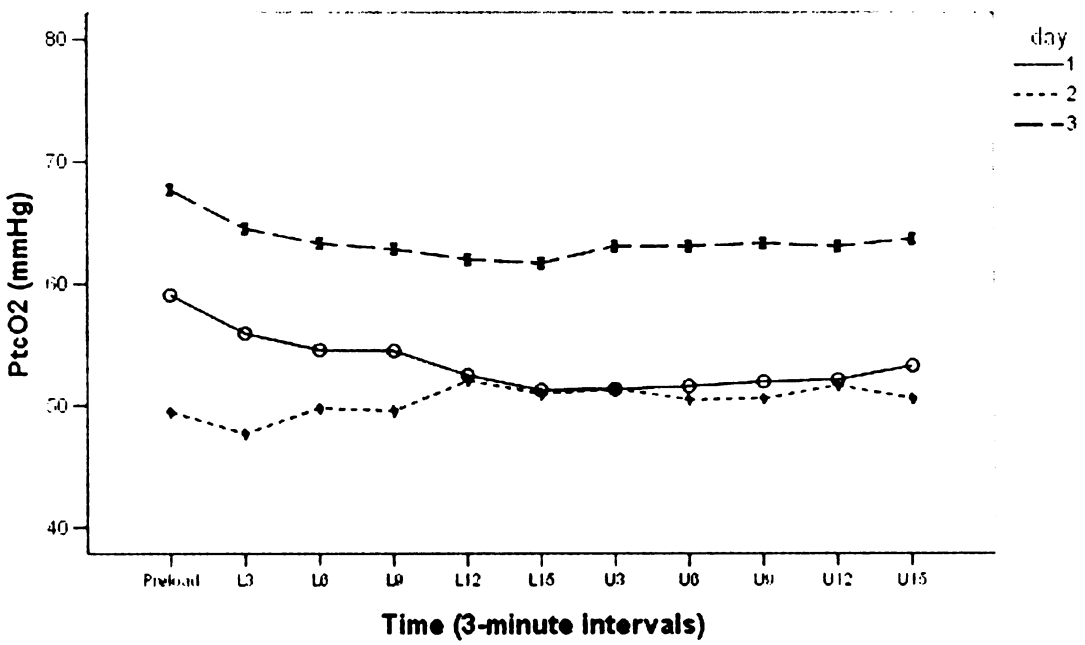
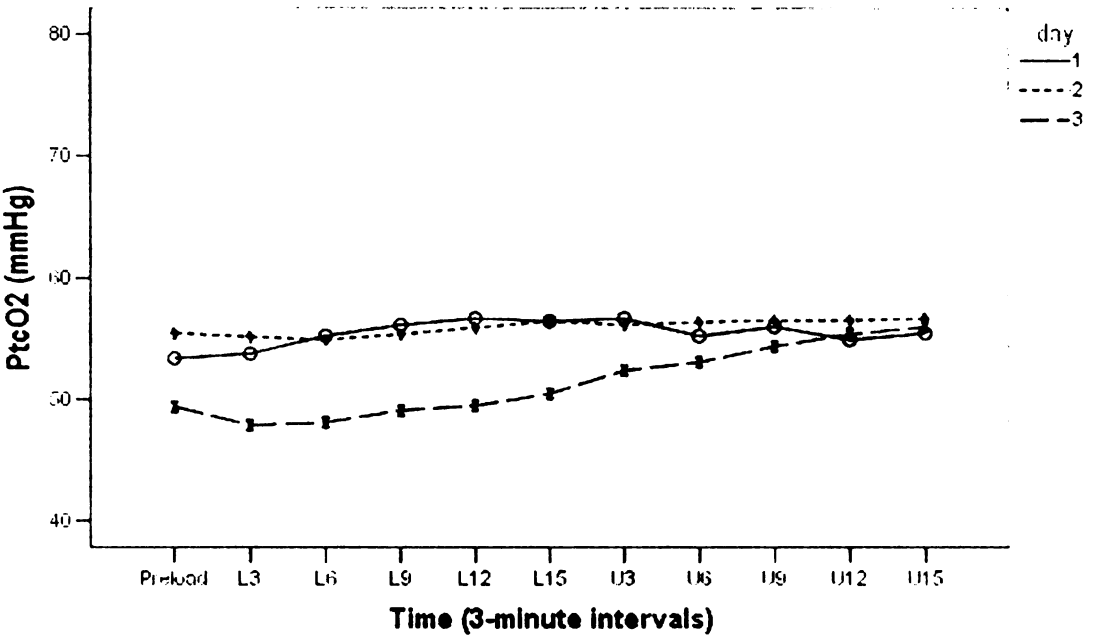


Figure 16. Heel PtcO2 in the operative leg with an oxygen challenge during preload, loading, and unloading.



To further describe the trends of changes over time of both legs on each post-op day, RMANOVA was used in which the within subjects factors were time and leg (Table 16).

Table 16. Trends of Changes in $PtcO_2$ on Both Legs on Different Post-op Days with an Oxygen Challenge

Dependent variables	Post-op day 1	Post-op day 2	Post-op day 3
Heel $PtcO_2$	N = 17 There were no main effects of time (p = 0.582), leg (p = 0.408), or time x leg interaction (p = 0.058).	N = 16 There were no main effects of time (p = 0.606), leg (p = 0.301), or time x leg interaction (p = 0.810).	N = 14 There was a main effect of leg (p = 0.037). $PtcO_2$ in the non-operative leg was different from $PtcO_2$ in the operative leg. Simple effects tests on each leg showed no main effects of time (p = 0.589).

There was no change in $PtcO_2$ in both legs over time in the first 3 post-op days. On post-op day 3, the change in $PtcO_2$ was different in one leg from the other with the non-operative leg being higher.

11. There will be no overall change in heel skin temperature during the loading and unloading conditions in either leg in the first three days after surgery with an oxygen challenge.

The null hypothesis was rejected. There was an overall change in heel skin temperature over time when compared with preload in all 3 days. Repeated measures analysis of variance was used in which the within subjects factors were time (preload, loading, and unloading), leg (non-operative and operative), and day (post-op days 1, 2, & 3). There was the main effect of time ($p = 0.01$). Post-hoc tests revealed that heel skin temperature increased significantly at the 6^{th} min. ($p = 0.003$), the 9^{th} min. ($p = 0.005$), the 12^{th} min. ($p = 0.002$), and the 15^{th} min. ($p = 0.001$) of loading and at the 9^{th} min. ($p = 0.005$) and 12^{th} min ($p = 0.005$) of unloading when compared with preload (Figures 17 & 18). There were no main effects of leg ($p = 0.233$) or day ($p = 0.573$). The change over time did not depend on the leg (no leg x time interaction) ($p = 0.322$) or day (no day x time interaction) ($p = 0.457$).

Figure 17. Heel skin temperature in the non-operative leg with an oxygen challenge during preload, loading, and unloading.

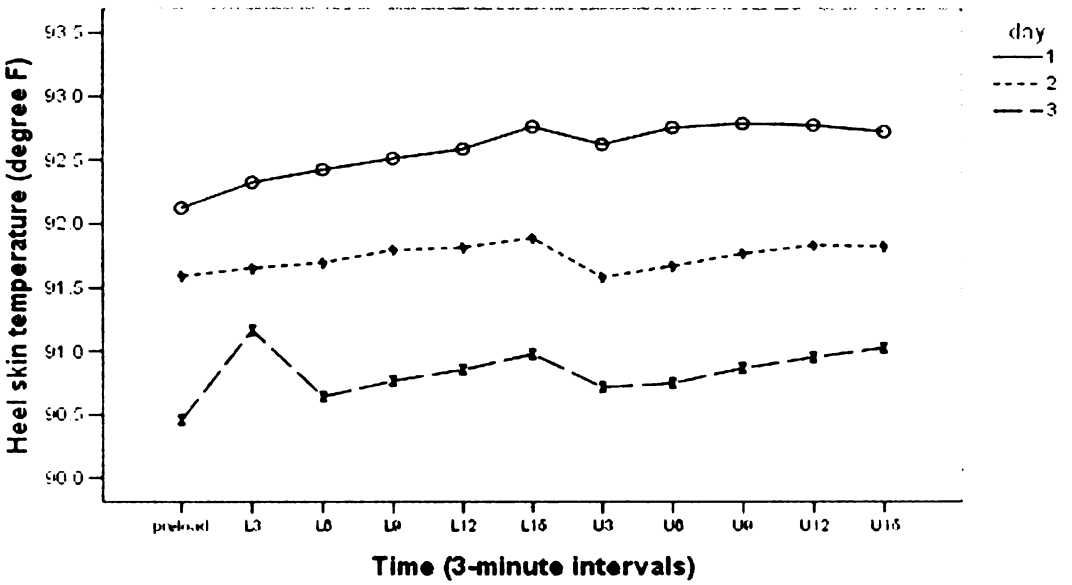
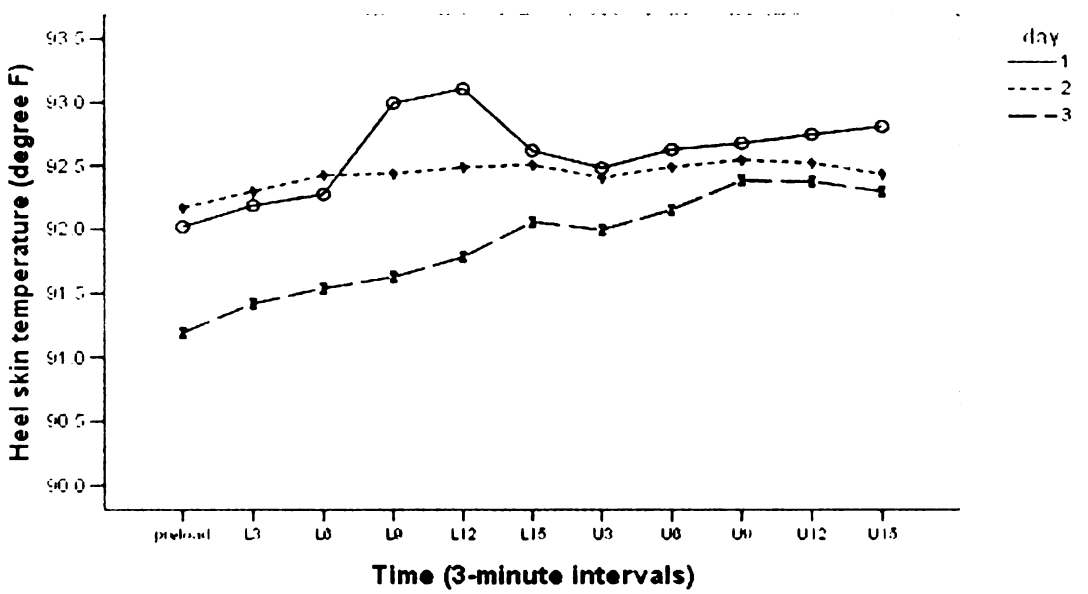


Figure 18. Heel skin temperature in the operative leg with an oxygen challenge during preload, loading, and unloading.



To further describe the trends of changes over time of both legs on each post-op day, RMANOVA was used in which the within subjects factors were time and leg (Table 15). There was no significant trend in changes of heel skin temperature on both legs when each post-op day was examined.

Table 17. Trends of Changes in Heel Skin Temperature on Both Legs on Different Post-op Days with an Oxygen Challenge

Dependent variables	Post-op day 1	Post-op day 2	Post-op day 3
Heel skin temperature	N = 17 There were no main effects of time (p = 0.058), leg (p = 0.859), or time x leg interaction (p = 0.412).	N = 17 There were no main effects of time (p = 0.427), leg (p = 0.182), or time x leg interaction (p = 0.584).	N = 16 There were no main effects of time (p = 0.081), leg (p = 0.207), or time x leg interaction (p = 0.206).

12. There will be no difference in heel skin oxygen tension in either leg in the first three minutes during unloading on post-op days 1-3 with an oxygen challenge.

The null hypothesis was not rejected. There was no difference in heel skin oxygen tension during the first 3 minutes of unloading as compared to the 15th minute of loading in both legs over the 3 post-op days. RMANOVA was used in which the within subject factors were time (loading at 15-minute and unloading in the first 3 minutes). Testing of simple effects was done on each leg on each post-op day (Table 18).

Table 18 Changes in $PtcO_2$ in Each Leg from the 15th Minute of Loading to the First 3 Minutes of Unloading on the First 3 Post-op Days with an Oxygen Challenge

Legs	Post-op Day 1	Post-op Day 2	Post-op Day 3
Non-operative leg	N = 17 There was no main effect of time (p = 0.25).	N = 16 There was no main effect of time (p = 0.445).	N = 16 There was no main effect of time (p = 0.075).
Operative leg	N = 18 There was no main effect of time (p = 0.704).	N = 18 There was no main effect of time (p = 0.828).	N = 16 There was no main effect of time (p = 0.076).

13. There will be no difference in heel skin temperature in either leg in the first three minutes during unloading on post-op days 1-3 with an oxygen challenge.

The null hypothesis was rejected. During the first 3 minutes of unloading, heel skin temperature change was inconsistent in the legs over the 3 post-operative days. RMANOVA was used in which the within subject factors were time (loading at 15-minute and unloading in the first 3 minutes). Testing of simple effects was done on each leg on each post-op day (Table 19).

Table 19. Changes in Heel Skin Temperature from the 15th Minute of Loading to the First 3 Minute of Unloading in Each Leg on the First 3 Post-op Days with an Oxygen Challenge

Legs	Post-op Day 1	Post-op Day 2	Post-op Day 3
Non-operative leg	N = 17 There was no main effect of time (p = 0.116).	N = 17 There was a main effect of time (p = 0.014). Post-hoc tests revealed that skin temperature decreased significantly at the 2nd minute of unloading (p = 0.015).	N = 16 There was no main effect of time (p = 0.09).
Operative leg	N = 18 There was the main effect of time (p = 0.031). Post-hoc tests revealed that skin temperature decreased significantly at the 3rd minute of unloading (p = 0.015).	N = 18 There was no main effect of time (p = 0.174).	N = 17 There was no main effect of time (p = 0.432).

There was a decrease in skin temperature during the first 3 minutes of unloading in the non-operative leg on post-op day 2 and in the operative leg on post-op day 1.

14. There will be no correlation between changes in heel skin oxygen tension and changes in heel skin temperature on either leg between preload and the end of unloading on post-op days 1-3 with an oxygen challenge.

Comparisons were made on the changes in $PtcO_2$ and changes in heel skin temperature from preload to unloading (Table 20) on each post-op day using Pearson's Product Moment correlation.

Table 20. Correlation of the Changes in $PtcO_2$ and Changes in Heel Skin Temperature on Post-op Days 1, 2, and 3 with an Oxygen Challenge

Postop days	Non-operative leg	Operative leg
Day 1	There was no significant correlation between $PtcO_2$ and heel skin temperature ($r = -0.408$, $p = 0.104$)	There was no significant correlation between $PtcO_2$ and heel skin temperature ($r = -0.083$, $p = 0.744$).
Day 2	There was no significant correlation between $PtcO_2$ and heel skin temperature ($r = 0.264$, $p = 0.324$).	There was no significant correlation between $PtcO_2$ and heel skin temperature ($r = 0.062$, $p = 0.807$).
Day 3	There was no significant correlation between $PtcO_2$ and heel skin temperature ($r = 0.239$, $p = 0.372$).	There was no significant correlation between $PtcO_2$ and heel skin temperature ($r = 0.118$, $p = 0.676$).

The null hypothesis was not rejected. There was no correlation between changes in $PtcO_2$ and changes in heel skin temperature during loading and unloading when compared to preload baseline in either leg on all three days.

15. There will be no correlation between changes in heel skin oxygen tension in each leg and the development of heel skin breakdown in the first three days after surgery with an oxygen challenge.

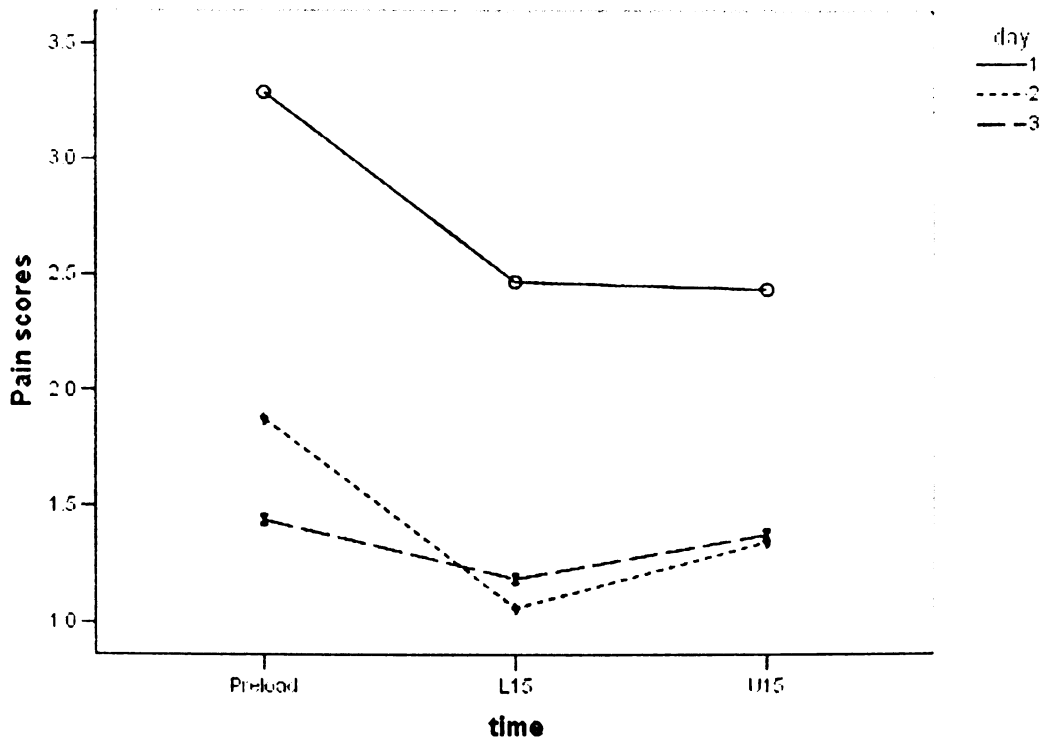
The null hypothesis was not rejected. There was no change in heel $PtcO_2$ and there was no development of heel skin breakdown in the first three days after surgery. None of the 18 subjects developed any heel skin breakdowns in the first three post-op days.

16. There will be no overall change in pain score at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery with an oxygen challenge.

The null hypothesis was rejected. The pain score decreased on post-op days 2 and 3 as compared to post-op day 1 (Figure 19). There was less pain at loading as compared to preload in all three days. RMANOVA was used in which the within

subjects factors were day (post-op days 1, 2, and 3) and time (preload, loading, and unloading). There was a main effect of day ($p = 0.015$). There was a significant decreasing linear trend ($p = 0.013$) in all 3 post-op days. There was a main effect of time ($p = 0.02$). Post-hoc test revealed that pain score decreased significantly at the last minute of loading ($p = 0.017$) when compared with preload. The change over loading and unloading times did not depend on the day (no day x time interaction) ($p = 0.386$).

Figure 19. Trends of changes in pain score with an oxygen challenge at preload, the last minutes of loading and unloading on the first three post-operative days.



17. There will be no correlation between heel skin oxygen tension in either leg and pain scores at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery with an oxygen challenge.

Comparisons were made between heel $PtcO_2$ on each leg and pain score at preload, the last minutes of loading and unloading on each post-op day using Pearson's Product Moment Correlation (Table 21). Heel $PtcO_2$ and pain score were inversely correlated in the non-operative leg at the last minute of loading on post-op day 2.

Table 21. Correlation of Heel $PtcO_2$ and Pain Score on Post-op Days 1, 2, and 3 with an Oxygen Challenge

Post-op days	Times	Correlation of $PtcO_2$ and pain			
		Non-operative leg		Operative leg	
		r	p	r	p
Day 1	Preload	-0.267	0.300	-0.170	0.500
	Loading	-0.238	0.357	-0.011	0.966
	Unloading	-0.414	0.111	-0.028	0.916
Day 2	Preload	0.123	0.650	-0.051	0.839
	Loading	-0.573	0.020 *	-0.128	0.613
	Unloading	-0.304	0.252	-0.187	0.457
Day 3	Preload	0.185	0.494	0.226	0.383
	Loading	-0.413	0.126	0.007	0.980
	Unloading	0.077	0.776	0.140	0.618

* Significant correlation, $p < 0.05$

18. There will be no correlation between heel skin temperature in either leg and pain scores at preload, the last minute of loading, and the last minute of unloading in the first three days after surgery with an oxygen challenge.

Comparisons were made between heel skin temperature on each leg and pain score at preload, the last minutes of loading and unloading on each post-op day using Pearson's Product Moment Correlation (Table 22). On post-op day 2, heel skin

temperature was inversely correlated to pain score in both legs at the last minute of unloading.

Table 22. Correlation of Heel Skin Temperature and Pain Score on Post-op Days 1, 2, and 3 with an Oxygen Challenge

Post-op days	Times	Correlation of heel skin temperature and pain score			
		Non-operative leg		Operative leg	
		r	p	r	p
Day 1	Preload	-0.327	0.200	-0.103	0.683
	Loading	-0.412	0.100	-0.186	0.460
	Unloading	-0.291	0.274	-0.106	0.687
Day 2	Preload	-0.298	0.245	-0.316	0.201
	Loading	-0.395	0.117	-0.272	0.274
	Unloading	-0.611	0.009 *	-0.469	0.049 *
Day 3	Preload	-0.130	0.631	-0.182	0.484
	Loading	0.214	0.443	0.230	0.392
	Unloading	-0.280	0.294	-0.131	0.616

* Significant correlation, $p < 0.05$

Correlation Analysis

Additional correlation analyses were done among dependent variables and demographic variables. The age of the subject was positively correlated with the number of previous hip surgeries ($r = 0.535$, $p = 0.022$) and ASA assessment score (sicker when scores higher) ($r = 0.483$, $p = 0.05$). The longer the duration of hip surgery, the more was the estimated blood loss ($r = 0.674$, $p = 0.003$). Swelling of the non-operative leg on the first day was related to swelling of the non-operative leg on the 2nd day ($r = 0.484$, $p = 0.042$) and the 3rd day ($r = 0.532$, $p = 0.023$). Swelling of the operative leg on day 2 and day 3 were positively correlated ($r = 0.714$, $p = 0.007$). Significant correlations specific to room air and oxygen challenge conditions were as follow.

On Room Air

There were fewer changes in $PtcO_2$ in both legs in the following conditions: when the legs were swollen, the intermittent pneumatic compression device was on, and the ankle brachial index was high. Changes of heel skin temperature were less when there was a higher ASA score. Wearing of compression stocking on the operative leg on day 1 was related to greater change in heel skin temperature during loading (Table 23).

Table 23. Correlations of Variables on Room Air

Variables	Variables	r	p	n
Changes in $PtcO_2$ in the non-operative leg on day 1	Swelling of non-operative leg on day 3	-0.595	0.015	16
Changes in $PtcO_2$ in the operative leg on day 1	Swelling of the operative leg on day 3	-0.659	0.004	17
Changes in heel temperature in the operative leg on day 1	ASA assessment	-0.577	0.019	16
Changes in $PtcO_2$ in the non-operative leg on day 2	Changes in $PtcO_2$ in the non-operative leg on day 3 Intermittent pneumatic compression device on day 2	0.532 -0.530	0.034 0.035	16 16
Changes in $PtcO_2$ in the operative leg on day 2	Ankle-brachial index on operative leg	-0.562	0.045	13
Changes in heel temperature in the operative leg on day 2	Estimated blood loss Duration of surgery	-0.813 -0.564	0.000 0.018	18 17
Changes in $PtcO_2$ in the operative leg on day 3	Intermittent pneumatic compression device on day 3	-0.519	0.027	18
Changes in heel temperature during loading in the operative leg on day 1	Compression stocking on operative leg on day 1	0.516	0.034	17
Changes in $PtcO_2$ in the non-operative leg during loading on day 2	Intermittent pneumatic compression device on day 2	-0.504	0.047	16
Swelling of non-operative leg on day 3	Ankle-brachial index on non-operative leg	0.708	0.007	13

With an Oxygen Challenge

There was a greater change in heel skin temperature in the non-operative leg when the ankle brachial index was higher or when there were less estimated blood loss.

The changes in $PtcO_2$ in the operative leg on day 2 and day 3 were positively correlated (Table 24). Heel $PtcO_2$ of both legs in response to unloading was examined first by subjecting the heels to loading pressure (bed surface) for 15 minutes and then by taking the pressure off the heels for 15 minutes. Hyperemic response times increase with both load duration and magnitude in healthy young adults (Mayrovitz et al., 1999). Mayrovitz et al (1999) examined the hyperemic response on heels of healthy, young adults with an external pressure of various magnitudes and durations and found that the longest response time was about 7.5 minutes. Measuring heel $PtcO_2$ and temperature for 15 minutes after pressure was relieved in this study might allow a better recording of hyperemic response.

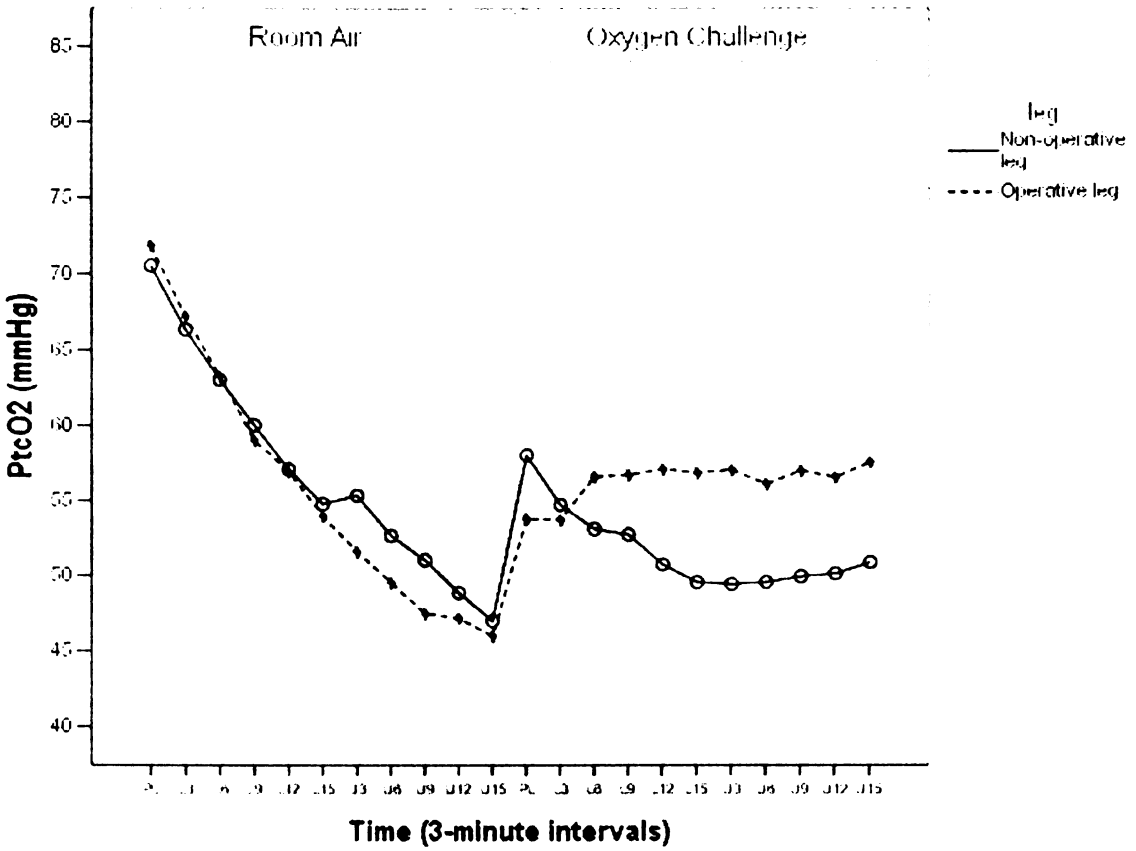
Table 24. Correlations of Variables with an Oxygen Challenge

Variables	Variables	r	p	n
Changes in heel skin temperature in the non-operative leg on day 2	Estimated blood loss	-0.637	0.006	17
Changes in $PtcO_2$ in the operative leg on day 2	Changes in $PtcO_2$ in the operative leg on day 3	0.888	0.000	15
Changes in heel skin temperature in the non-operative leg on day 3	Ankle brachial index of the non-operative leg	0.617	0.033	12
Ankle brachial index on non-operative leg	Swelling of non-operative leg on day 2	0.600	0.030	13
	Swelling of non-operative leg on day3	0.708	0.007	13

Heel $PtcO_2$ Response on Room Air and with an Oxygen Challenge

When examining the heel $PtcO_2$ response during the loading and unloading conditions on room air and with an oxygen challenge, heel $PtcO_2$ was different when an oxygen challenge was given. RMANOVA was used in which the within subjects factors were time (preload, loading, and unloading on room air and preload, loading, and unloading with an oxygen challenge) and leg (non-operative and operative).

Figure 20. Heel PtcO2 response on room air and with an oxygen challenge on day 1.



On post-op day 1, there was the main effect of time and time by leg interaction (Figure 20). Heel $PtcO_2$ decreased over time ($p = 0.034$). Post-hoc tests revealed significance at all times during room air ($p < 0.002$) but not with oxygen challenge. The change over time depended on whether it was the non-operative or operative leg (time x leg interaction) ($p = 0.029$). There was no main effect of leg ($p = 0.807$).

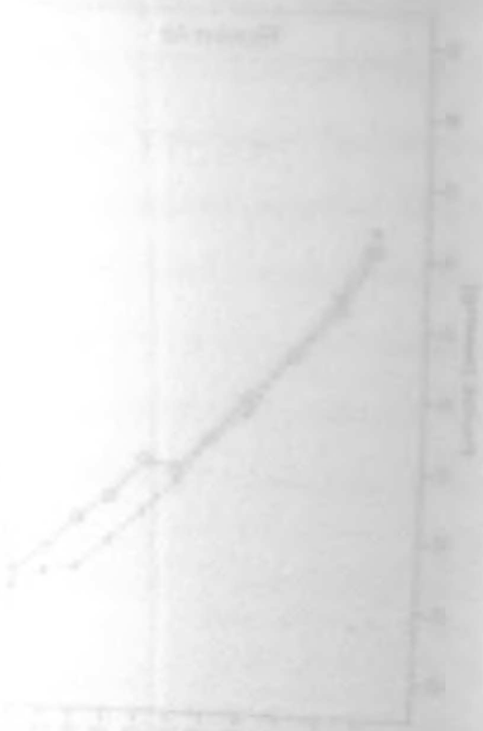
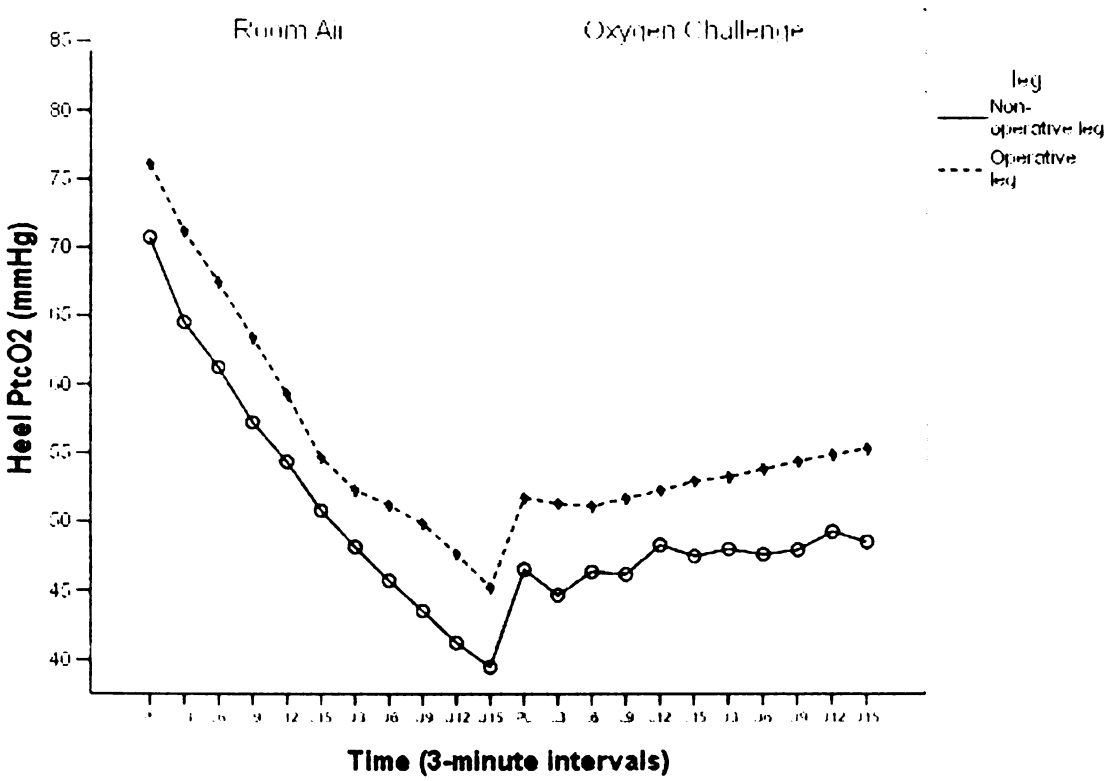
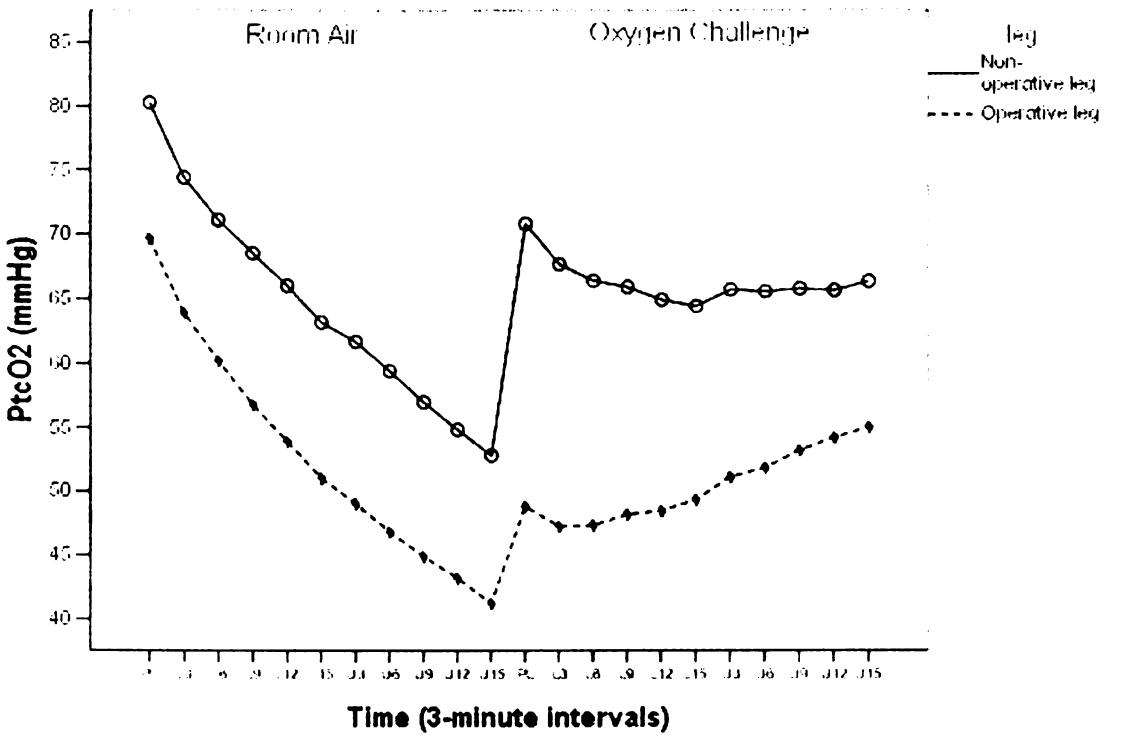


Figure 21. Heel PtcO2 response on room air and with an oxygen challenge on day 2.

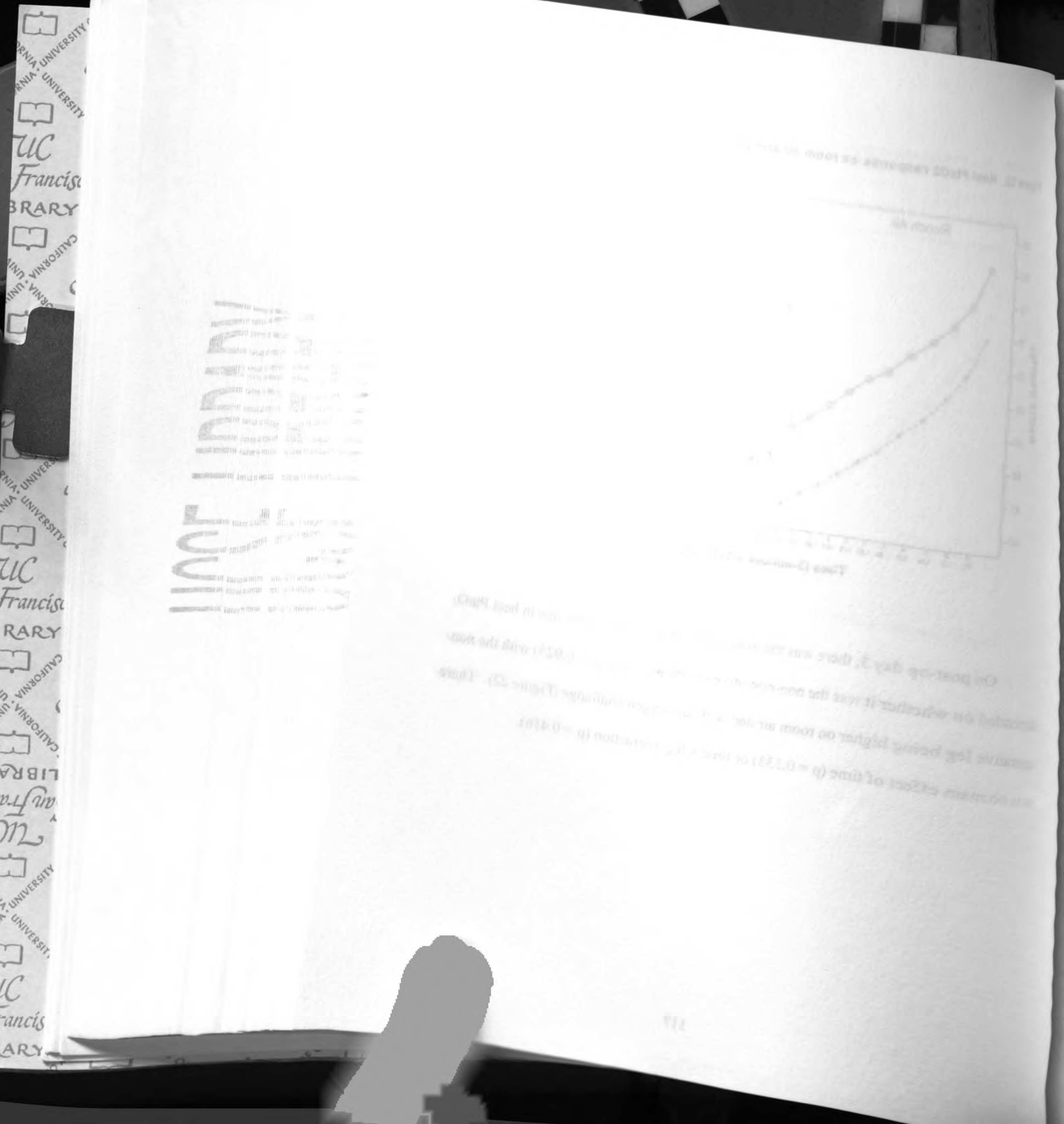


On post-op day 2, there was the main effect of time. Heel $PtcO_2$ decreased significantly over time ($p = 0.002$) on room air and with oxygen challenge (Figure 21). Post-hoc tests revealed significance at all times ($p < 0.002$) except at preload and at the 6th minute of loading with an oxygen challenge. There was no main effect of leg ($p = 0.084$) or leg by time interaction ($p = 0.006$).

Figure 22. Heel PtcO2 response on room air and with an oxygen challenge on day 3.



On post-op day 3, there was the main effect of leg. The difference in heel $PtcO_2$ depended on whether it was the non-operative or operative leg ($p = 0.025$) with the non-operative leg being higher on room air and with an oxygen challenge (Figure 22). There was no main effect of time ($p = 0.133$) or time x leg interaction ($p = 0.416$).



CHAPTER V

DISCUSSION

Patients undergoing hip surgery are at risk of low heel skin oxygenation. The purpose of this study was to examine the relationship among external pressure, heel skin oxygen tension ($PtcO_2$), and heel skin temperature in the operative and non-operative leg after hip surgery in adults. Eighteen adult subjects undergoing total hip replacement surgery were followed during the first three post-operative days. The responses of heel $PtcO_2$ and heel skin temperature to pressure loading and unloading were recorded when subjects were breathing room air and when subjects were receiving an oxygen challenge. Hip surgery, pressure loading, the hyperemic response, oxygen challenge, pain, the study limitations, implications, and recommendations for future research will be discussed.

Study findings show that when subjects were breathing room air, heel $PtcO_2$ decreased during loading and unloading in both legs across all three post-operative days. The hyperemic response was not apparent in either leg during the immediate pressure unloading period. Heel $PtcO_2$ in the non-operative leg did not change on post-op day 1 but $PtcO_2$ decreased on post-op days 2 and 3. For the operative leg, heel $PtcO_2$ decreased during the immediate unloading period on all three days.

Heel skin temperature increased during loading and unloading in both legs on post-op days 1 and 3. Heel skin temperature did not change on post-op day 2. During the immediate unloading period, heel skin temperature in the non-operative leg decreased on post-op days 2 and 3 but did not change on post-op day 1. For the operative leg, heel skin temperature did not change during the immediate unloading period on any of the three post-op days.

When giving an oxygen challenge, heel $PtcO_2$ did not rise as expected nor was the hyperemic response visible. In fact, there was no difference in heel $PtcO_2$ in both legs during the immediate unloading period on all three post-op days.

Heel skin temperature increased during loading and unloading in both legs across all three post-op days with an oxygen challenge. During the immediate unloading period, heel skin temperature decreased in the non-operative leg on post-op day 2. Also, skin temperature decreased in the operative leg on post-op day 1. Heel skin temperature did not change on any other day in both legs during the immediate post-op period.

Hip Surgery

There has been no documentation of measuring heel $PtcO_2$ or heel skin temperature in response to pressure loading in hip surgery patients. A recent study on hip and knee surgery patients examined $PtcO_2$ at the incision site, the contralateral site, and the chest site of patients (n = 24) (McPhail, Cooper, Hodge, Cabanel, & Rooke, 2004). Measurements were taken pre-operatively, on the second post-op day and two months after surgery. $PtcO_2$ decreased at all sites on post-op day 2 as compared to pre-op and 2-month post-op (McPhail et al., 2004). Going through surgery predisposes a person to many external factors such as hemodynamic changes and hypoxia, which may affect $PtcO_2$ (Gottrup, 2004). In this present study, the pre-operative preload heel $PtcO_2$ was not measured and could not be compared to the post-operative preload $PtcO_2$.

About half of the subjects in this study were on intermittent pneumatic compression device (IPC) and most of the subjects had compression stockings with the percentage depending on day (67 – 89%). The IPC is intended to prevent venous stasis by emptying deep veins in the legs while compression stockings are used to prevent

distension of the veins (Morris & Woodcock, 2004). The IPC device delivers sequential compression pressure from ankle to below the knee at 45 mmHg for 11 seconds and then decompresses every 60 seconds. Compression inflation and deflation cycles did not seem to affect $PtcO_2$ in the lower legs (Rithalia, Edwards, & Sayegh, 1988). In fact, $PtcO_2$ decreased with each compression but returned to pre-compression values during the deflation time in both healthy adults (n = 14) and older patients (n = 14) (Rithalia et al., 1988). In a study on patients with post-thrombotic leg ulcers (n = 10), the use of IPC was positively correlated with reduction of leg edema ($r = 0.912$, $p < 0.002$) (Kolari, Pekanmaki, & Pohjola, 1988). The authors suggested that reduction in edema might increase $PtcO_2$. However, the reduction of leg edema in subjects with leg ulcers (n = 8) had not been shown to raise $PtcO_2$ at the ulcer site in another study (Nemeth, Falanga, Alstadt, & Eaglstein, 1989). These studies showed that edema may not affect oxygen diffusion to the skin.

In the present study, edema of the legs was present in about 40% of subjects on both day 1 and day 3. On post-op day 2, about 60% of the subjects had leg edema. Coincidentally, heel skin temperature increased during the loading unloading response across all three post-op days, regardless whether subjects were breathing room air or receiving an oxygen challenge. The relationship between edema and skin temperature needs further investigation.

When comparing both legs, heel $PtcO_2$ in the non-operative leg was higher than that in the operative leg during both room air and with an oxygen challenge on post-op day 3. In other words, the heel skin of the non-operative side is better oxygenated than that of the operative side on the third post-operative day only. However, both heels were

tally at risk of low oxygenation when subjected to external pressure on all three post-erative days.

Pressure Loading

The measurement of $PtcO_2$ varies over different parts of the body. Heel $PtcO_2$ easured at 44 °C for 16-20 minutes in the undamaged skin of the heel (n = 6) was 71 mHg (median) with an interquartile range of 58 – 81 mmHg in one study (Schubert, 000). Preload mean heel $PtcO_2$ in the non-operative leg measured at 44 °C for 20-25 inutes on room air in this study was 70.6 mmHg (SD 21.16), 70.8 mmHg (SD 22.16), nd 80.0 mmHg (SD 21.41) on post-op days 1, 2 and 3 respectively. Preload mean heel $PtcO_2$ in the operative leg was 70.2 mmHg (SD 25.33), 74.8 mmHg (SD 24.74), and 69.2 mmHg (SD 17.53) on post-op days 1, 2 and 3 respectively.

Application of external pressure reduced $PtcO_2$ in the trochanter (Xakellis et al., 1991), sacrum (Bader & Gant, 1988; Knight et al., 2001) and foot (Fromy et al., 2002). The decrease in $PtcO_2$ with increasing loading pressure was described as "sigmoidal" in the sacral site (Colin & Saumet, 1996) and quadratic (2^{nd} degree polynomial) in the trochanteric site (Xakellis et al., 1991). The findings of this present study agreed with other studies that heel $PtcO_2$ decreased during loading in the room air phase. However, heel $PtcO_2$ did not change during loading on all three post-op days when subjects were receiving 7-10 Lpm of oxygen.

Skin blood flow, as measured by laser Doppler, also decreased with pressure loading. Mayrovitz and colleagues have done a series of heel perfusion studies in healthy subjects. All the studies showed that heel skin blood flow, as measured by laser Doppler, decreased during pressure loading (Mayrovitz et al., 1999; Mayrovitz et al., 2003;

Mayrovitz & Smith, 1998; Mayrovitz et al., 1997). However, as laser Doppler technology measures relative values of skin blood flow in skin capillaries (Kragelj, Jarm, Erjavec, Presern-Strukelj, & Miklavcic, 2001), the response to pressure loading in skin blood flow cannot be quantified as it can be with transcutaneous oxygen tension. A lower loading pressure (20 mmHg) was needed to produce a decrease in skin blood flow while a higher loading pressure (40 mmHg) was needed to produce a decrease in $PtcO_2$ (Colin & Saumet, 1996). An external pressure of above 300 mmHg actually stopped skin mircovascular blood flow in the sole of the foot in humans (Meinders et al., 1996).

Since skin blood flow plays an important part in transporting heat from the skin surface (Jaszczak, 1988), external pressure impeding blood flow may lead to an increase in skin temperature. Heel skin temperature in this study increased during loading under both room air and oxygen challenge conditions.

Hyperemic Response

$PtcO_2$/Skin Blood Flow

An increase in skin blood flow is considered to be the normal hyperemic response when external pressure is relieved. The increased skin blood flow could be caused by reflex vasodiation of the blood vessels in response to their being partially occluded (Xakellis et al., 1993). Application of pressure to the skin leads to deformation of underlying arterioles (Bader et al., 1986). Subsequent alterations in vasoactive metabolites and contractility of the arteriolar wall may induce vasodilation (Capp et al., 2004). Vasodilation accommodates a bigger volume of blood flow. In all Mayrovitz's studies on the heels of healthy adults, skin blood flow increased after relief of pressure

(Mayrovitz et al., 1999; Mayrovitz et al., 2003; Mayrovitz & Smith, 1998; Mayrovitz et al., 1997).

The amount of loading pressure seems to have no influence on hyperemia. In a Swedish study on sitting pressure and perfusion of buttock skin in paraplegic and tetraplegic patients (n = 8) and healthy controls (n = 10), hyperemia occurred when loading pressure was greater than 2 N/cm2 (0.015 mmHg/cm2) (Thorfinn, Sjoberg, & Lidman, 2002). Thorfinn and colleagues (2002) did not find a correlation between the amount of reactive hyperemia (as measured by laser Dopper imaging) and absolute values of sitting pressures. Even though the interface pressure in the present study was not measured, in the current study enough loading pressure should have been delivered when the bed surface was switched to comfort mode during loading as evidenced by the decrease in $PtcO_2$. Yet, a hyperemic response was not seen in either heel in the post hip surgery subjects in the present study.

A reduced hyperemic response may result from capillary damage (Herrman et al., 1999). Also, an increase in local metabolism may increase the oxygen consumption and therefore lower the $PtcO_2$ (Schubert, 2000; Soini & Takala, 1991). Any endothelial swelling may affect the entrance of erythrocytes into the capillaries and hamper the delivery of oxygen (Schubert, 2000), thus reducing $PtcO_2$ even after pressure relief.

Whether the endothelium-derived nitric oxide (NO) plays a role in the local blood flow during reactive hyperemia is still controversial. Increases in blood flow leads to increases in shear on the endothelial cells, which in turn stimulates NO production (Bruckdorfer, 2005; Pohl et al., 1993). Nitric oxide signals the vascular endothelium to dilate. Nitric oxide has been found to mediate reactive hyperemia (Koller & Bagi, 2002).

More recent studies, however, found that NO concentrations do not increase during hyperemia and may not play a part in vasodilation response (Wong, Wilkins, Holowatz, & Minson, 2003; Zhao, Pergola, Roman, & Kellogg, 2004).

The hyperemic response in this study might not have happened or might have been very brief. In a study comparing the cutaneous vascular response to transient loading in female smokers (n = 9) and female non-smokers (n = 9), a rapid hyperemic response peaked at 20 seconds of unloading (Noble et al., 2003). In Noble and colleagues study (2003), loading was applied to the sacrum and laser Doppler was used to measure the mean skin blood flow. The increase in skin blood flux dropped back to preload baseline within two minutes of unloading in all subjects (Noble et al., 2003). When studying the sole of the foot using laser Doppler, hyperemic response peaked at about 10 seconds after external pressure was removed (Meinders et al., 1996). Measuring heel $PtcO_2$ every second for the first three minutes of unloading in this study might have detected a hyperemic response, should there be one.

The hyperemic response has been shown to be smaller in people with diabetes (Mayrovitz & Sims, 2004), in smokers (Noble et al., 2003), and in subjects with peripheral vascular diseases (Kragelj et al., 2001) as compared with healthy subjects. The present sample included only one smoker. None of the subjects had diabetes or peripheral vascular diseases. The decrease in heel $PtcO_2$ during unloading in both legs of post hip surgery patients has not previously been documented in the literature.

Skin Temperature

An increase in skin temperature has been related to an increase in external pressure in rats (Herrman et al., 1999). Upon removal of pressure, the increase in tissue

temperature may be due to increased perfusion or an inflammatory response (Sprigle et al., 2001). A clinical study on humans also showed that after external pressure was removed, skin temperature increased in the sacrum and gluteus maximus muscle (Schubert & Fagrell, 1991).

During the immediate pressure unloading period, the change of skin temperature is inconsistent. In an often cited study done by Mahanty and Roemer (1979) on the thermal response of the trochanter skin to pressure application, skin temperature rose quickly and then decreased. Temperature peak increases occurred at 3 to 5 minutes upon unloading (Mahanty & Roemer, 1979). Thermography taken on the forearm after loading showed that there was a 1-3 minutes of time lapse before temperature increased to a maximum (Goller, Lewis, McLaughlin, & Verhonick, 1976). The thermal response of cyclic pressure on the lower leg of human subjects (n = 3) was studied (Sanders, 2000). Skin temperature was taken every 30 seconds at the pressure unloading site and a contralateral site using infrared temperature sensor. Temperature decreased immediately after unloading and then increased. The author suggested that reperfusion might have occurred (Sanders, 2000). Skin temperature generally increased during unloading after a brief period of decrease. Likewise, the present study showed some decrease in heel skin temperature, however, the decrease was not consistent with the leg or the post-op day. There was no change in heel skin temperature in most of the immediate unloading period. Afterwards, there was an increase in heel skin temperature during the rest of the 12 minute unloading time in both legs under both room air and oxygen challenge conditions. This study was different from the above studies in that heel skin temperature increased during both loading and unloading.

Pressure ulcer development within two weeks of admission and sacral skin temperature were examined in hospitalized neurologically impaired Thai patients (n = 14) (Sae-Sia et al., 2005). Subjects were positioned supine (loading) and then were positioned on the lateral side (unloading) for 15 minutes. Sacral temperature was measured initially within two to four days of admission and again in two to three days. Mean sacral skin temperature in subjects who developed a pressure ulcer (stage 1 and 11) was higher than those who did not develop a pressure ulcer during both loading ($p = 0.001$) and unloading ($p = 0.002$) at both the initial and subsequent assessments. The inconsistency of data collection time may be a confounding factor in the Sae-Sia study (2005). In addition, these subjects were either bed- or chair-bound before data collection and deep tissue damage might have already occurred. Yet, the increased sacral skin temperature during loading and unloading in patients who eventually developed pressure ulcers indicated that increased skin temperature is associated with compromised tissue.

Subcutaneous temperature has been shown to correlate with subcutaneous perfusion (Hopf et al., 2000). However, there is no literature showing the relationship between $PtcO_2$ and skin temperature. Heel skin oxygen tension decreased while heel skin temperature increased over loading and unloading under room air condition. The reduced blood flow and the increased tissue oxygen consumption may be a possible explanation for the increased heel skin temperature.

Oxygen Challenge

Giving an oxygen challenge ($FiO_2 = 0.4 - 0.6$) increases PaO_2, $PsqO_2$, and $PtcO_2$ when there is normal subcutaneous tissue perfusion (Hopf et al., 1997). In subcutaneous tissue with normal peripheral tissue perfusion, partial pressure of oxygen is similar in

both the tissue and the blood (Gottrup, 2004). If perfusion decreases, oxygen extraction from the subcutaneous tissues increases, which in turn lowers the subcutaneous tissue tension (Gottrup et al., 1987) and subsequently affecting oxygen delivery to the skin. An increase in partial oxygen pressure after increased FiO_2 indicates normal tissue perfusion (Hopf et al., 1997).

In this study, oxygen saturation was about 98 - 99% when subjects were receiving an oxygen challenge. All subjects had ABI >0.9 in both legs. Loading and unloading did not affect heel $PtcO_2$ while breathing 7-10 Lmp oxygen. During loading, flow is inadequate but perhaps, arterial oxygen is still normal and so $PtcO_2$ does not change. It is possible that subjects were relatively under-hydrated.

When examining heel $PtcO_2$ across both room air and oxygen challenge phases, $PtcO_2$ at preload with an oxygen challenge was higher than $PtcO_2$ at the last minute of unloading on room air in all three days. However, $PtcO_2$ at preload with an oxygen challenge was still lower than the preload value on room air. The result is not conclusive since some of the subjects were given an oxygen challenge prior to breathing room air.

Pain

In this study, the pain score did not change under loading/unloading conditions on room air but decreased with an oxygen challenge. Most subjects complained of pain in the operative hip only when trying to get out of bed. None of the subjects reported pain in the heels. Since subjects stayed in bed for the entire two hours of data collection and were receiving patient controlled analgesia, pain was under control. An exception was four subjects with muscle spasms who reported pain intensity of 7 – 10 on the VAS.

The effect of pain on subcutaneous oxygen tension has been documented (Akca et

al., 1999; Buggy et al., 2002; Buggy & Kerin, 2004). In general, adequate pain control during the immediate post-operative period attenuates vasoconstriction, improves perfusion, and increases tissue oxygen tension. In this study, the inverse correlation between pain and heel $PtcO_2$ was only seen in the non-operative leg once under room air and once with an oxygen challenge. Pain score has minimal effect on heel $PtcO_2$ overall.

The effect of pain on heel skin temperature is more apparent in this study. On post-op day 2 in both legs, heel skin temperature was higher when there was less pain, specifically at loading time on room air and at unloading time with an oxygen challenge. Literature on the relationship between pain and skin temperature is scarce. Likewise, the effect of skin temperature on pain also needs further investigation. A few clinical studies showed that local warming reduces pain in patients with cholelithiasis ($p < 0.01$) (Kober, Scheck et al., 2003), renal colic (Kober, Dobrovits et al., 2003), and low back pain (Nuhr et al., 2004) during emergency transport.

Limitations of the Study

This study was done as true to the clinical setting as possible, not controlling for covariates such as leg edema, wearing of compression stockings, and/or intermittent pneumatic compression device, or the interface pressure of the mattress. Heel $PtcO_2$ and heel skin temperature were measured in the clinical environment.

There is a large variation in heel $PtcO_2$ within each individual subject among the 3 days. Most of the subjects were wearing compression stockings and removal of the stockings was not possible. To place the sensors, the foot part of the stocking was pulled back through the opening at the posterior aspect of the toes. Sensors were slipped in and placed on the plantar aspect of the foot as close to the heel and as consistently as possible.

The same location of sensor placement could not be guaranteed each day. Placing the sensors on the same location may decrease the intra-subject variability.

Wearing of the compression stockings may affect skin temperature. An elevated hyperemic response was shown to be affected by local heating (Capp et al., 2004). However, in this study, the hyperemic response was not apparent. The maximum heel skin temperature measured was 36.6 °C (97.9 °F).

The hyperemic response is not seen in this study. Since hyperemic response can be very short upon unloading, minute changes in $PtcO_2$ and heel skin temperature might not have been captured with the measurements being recorded every minute for the first 3 minutes of unloading.

The other main factors affecting $PtcO_2$ response i.e., pain (Akca et al., 1999) was not controlled in the present study, although it was measured and its effect on $PtcO_2$ evaluated. Subjects were encouraged to use patient-controlled-analgesia (PCA) and oral analgesic for maximum pain control. Pain assessment during preload, loading, and unloading yielded a range of scores from 0 to 8. Many subjects denied having pain while resting in bed. Pain level did not fluctuate during the 2 hours of data collection.

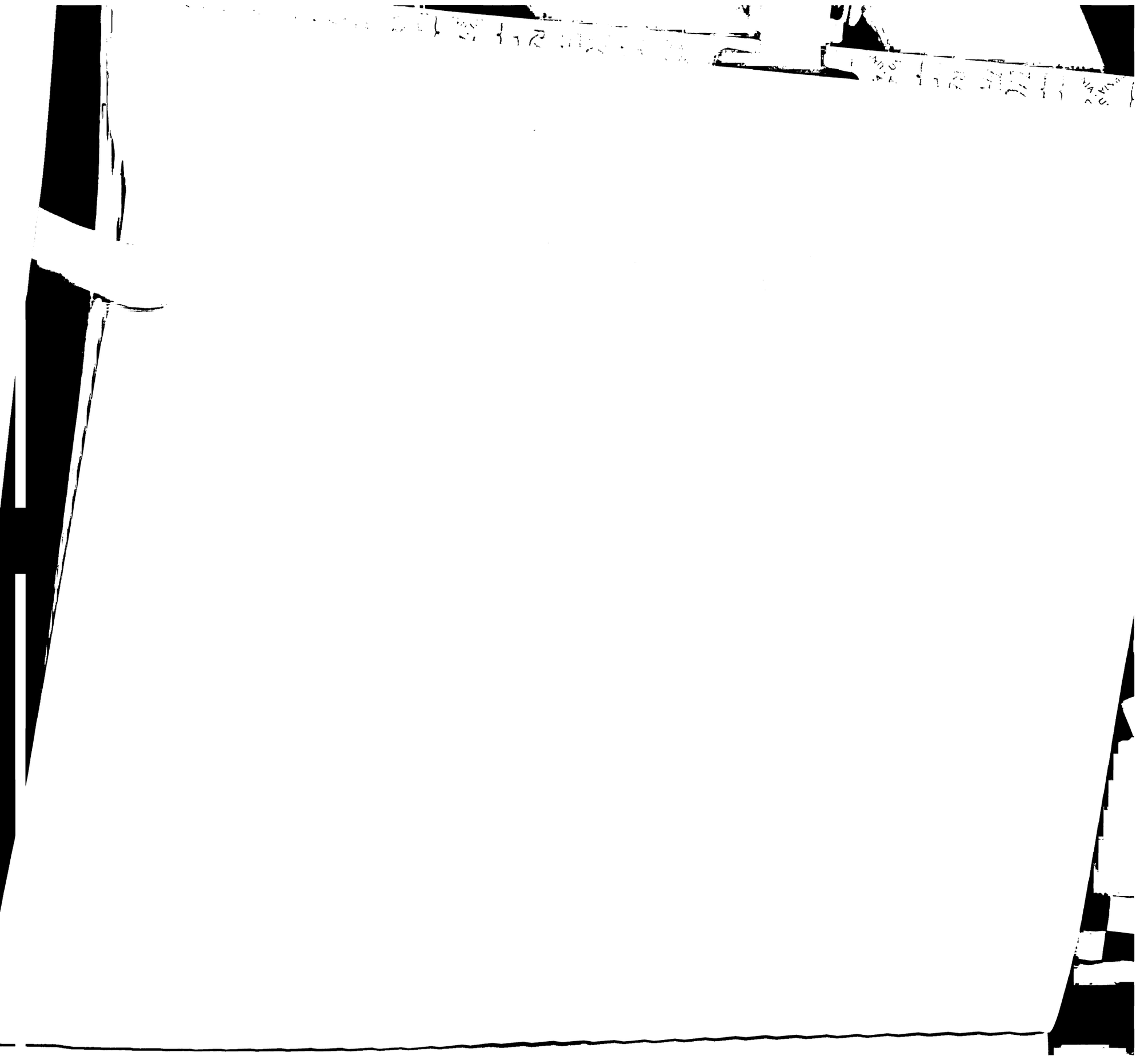
Volume status also was not controlled (Greif et al., 2000). All subjects in the study had crystalloid replacement at least on the first post-op day and they all resumed their normal drinking and eating pattern by day 2. The hematocrit in this study was not recorded. However, almost all subjects received an autologous blood transfusion within the first 2 days after surgery. The mean hematocrit in another study on post hip surgery patients (n = 58) was between 29.8 – 30.8 % in all 3 post-op days (Whitney & Parkman, 2004).

The focus of this study was the loading unloading response of the heels after hip surgery. There are no comparable data on the loading response of the heels before surgery. Also, since heel $PtcO_2$ had a decreasing trend in the room air phase, giving an oxygen challenge right after the room air phase may explain the relatively low preload values during the oxygen challenge phase. The room air phase and oxygen challenge phase can be done at two separate times. Since some subjects were given an oxygen challenge first while majority were on room air first, there was no control for the order effect. The order of the two phases needs to be standardized in future research.

Due to the limited sample size, it was not meaningful to analyze the data using multiple regression. Otherwise, it will be helpful to know the unique contribution of each factor aforementioned in accounting for the changes in heel $PtcO_2$ and heel skin temperature.

Implications and Recommendations

The major finding of this study is that heel $PtcO_2$ decreased after placing the heels on the bed surface for 15 minutes. The decrease in heel $PtcO_2$ continued after the heels were removed from the bed surface. The hyperemic response that was documented in other studies with unloading was blunted in this group of patients after hip surgery. The heels of both the non-operative and operative leg responded the same way to external pressure. Movement of the lower legs is limited since the legs are strapped onto either side of the abductor foam. In other words, heels on both the non-operative and operative legs are at risk of low skin oxygenation and pressure ulcer development after hip surgery. It is postulated that in order to ensure adequate heel skin oxygenation and blood flow, the heels should be kept off pressure at all times in the post hip surgery population. Even



though all the subjects were placed on a pressure-reduction bed with a built-in heel pressure relief mode, few beds had the heel pressure relief mode turned on. When the researcher went to collect data, she found most beds were set on the comfort mode (without pressure relief). Also, subjects' heels did not lie on the corresponding section of the heel-relief feature. It is important to keep heels off the bed surface by utilizing the bed's heel pressure relief function or placing the calf on a pillow.

Although heel $PtcO_2$ decreased over loading and unloading while heel skin temperature increased over loading and unloading on room air, these two variables were not correlated. The physiological phenomenon of changes in each variable requires further exploration.

Future studies should explore the following questions: 1) How do heel skin oxygenation and skin temperature respond to pressure loading and unloading in both legs in the age and gender matched population across three consecutive days? 2) Why tissue oxygen continues to fall after pressure is relieved and the hyperemic response is blunted? 3) Are these findings consistent with those of healthy (non-surgical) people? 4) Will edema and compression stockings affect the temperature or skin oxygenation? and 5) Are the findings the same in the sacral area?

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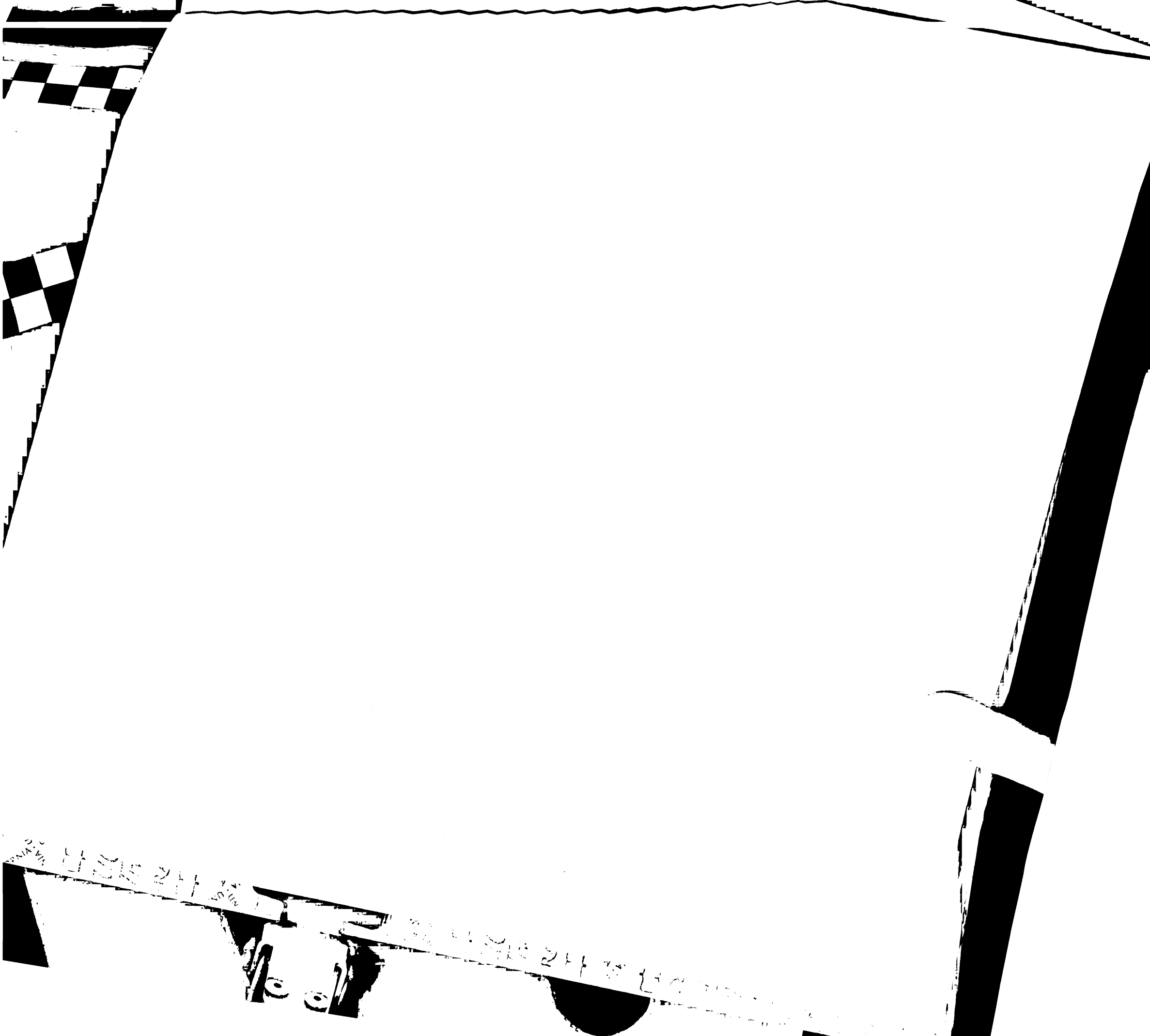

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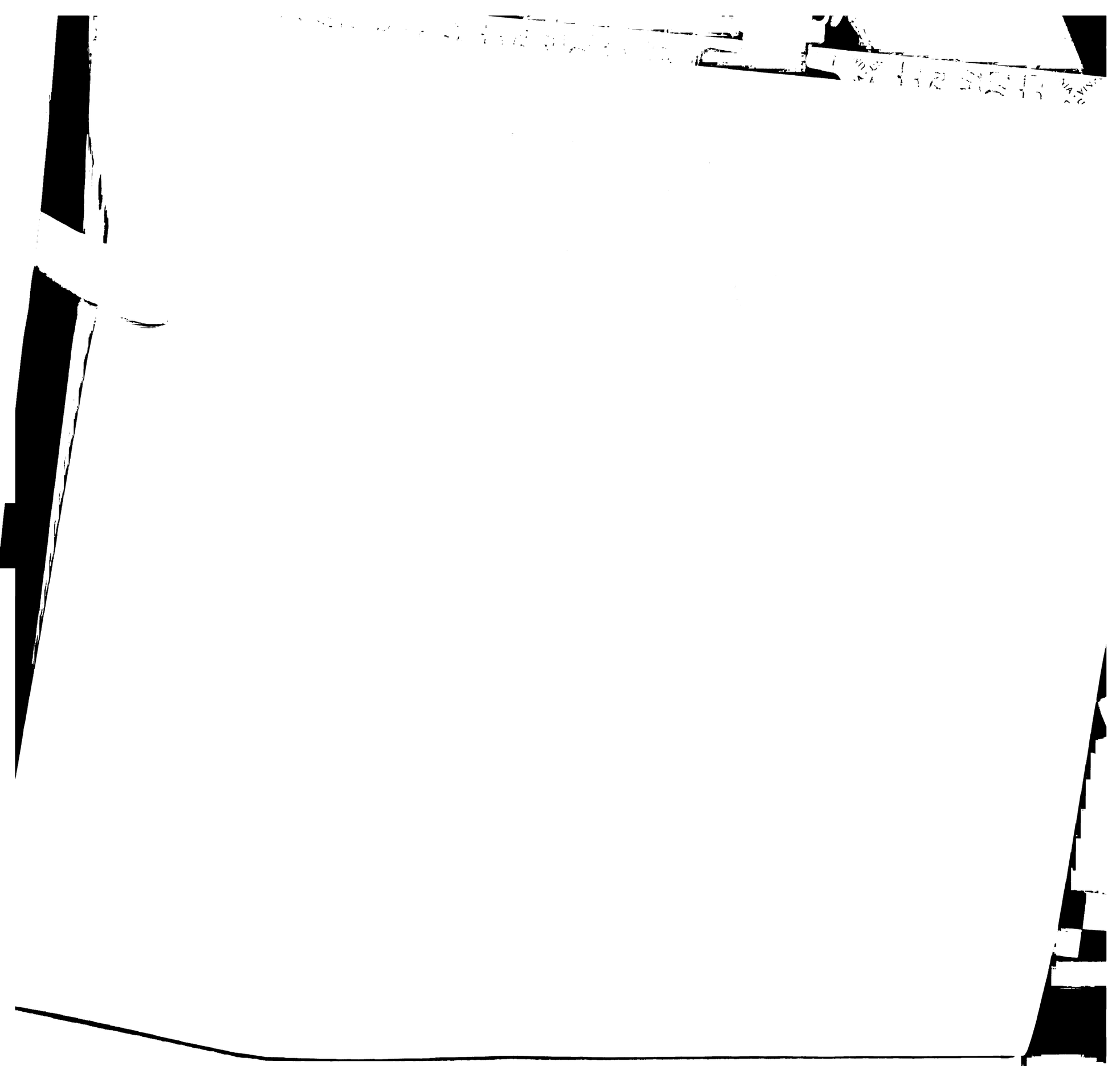

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APPENDIX A

COMMITTEE ON HUMAN RESEARCH APPROVAL LETTER

COMMITTEE ON HUMAN RESEARCH
OFFICE OF RESEARCH, Box 0962
UNIVERSITY OF CALIFORNIA, SAN FRANCISCO
www.research.ucsf.edu/chr/AppliyforApproval.asp
chr@ucsf.edu
(415) 476-1814

CHR APPROVAL LETTER

TO Nancy Stotts, R.N., Ed.D.
Box 0610

Vivian Wong, RN, MSN
Box 0610,

RE: Heel Pressure Ulcer Risk in Hip Surgery Patients

The Committee on Human Research (CHR) has reviewed and approved this application to involve humans as research subjects. This included a review of all documents attached to the original copy of this letter.

Specifically, the review included but was not limited to the following documents:
Consent Form, Dated 6/7/04

The CHR is the Institutional Review Board (IRB) for UCSF and its affiliates. UCSF holds Office of Human Research Protections Federalwide Assurance number FWA00000068. See the CHR website for a list of other applicable FWA's.

Comment: The approval of this modification includes the removal of the Condition in our letter of May 28, 2004.

APPROVAL NUMBER H1156-24097-01A. This number is a UCSF CHR number and should be used on all correspondence, consent forms and patient charts as appropriate.

APPROVAL DATE: June 24, 2004 **EXPIRATION DATE:** May 28, 2005 **Expedited Review**

GENERAL CONDITIONS OF APPROVAL: Please refer to www.research.ucsf.edu/chr/ApplyforCHRApprovalCond.asp for a description of the general conditions of CHR approval. In particular, the study must be renewed by the expiration date if work is to continue. Also, prior CHR approval must be obtained before implementing any changes to the consent document(s) or any changes in the protocol unless these changes are required urgently for the safety of the subjects.

HIPAA "Privacy Rule" (45CFR164): This study requires individual consent/authorization for use and/or disclosure of Protected Health Information (PHI).

Sincerely,

Reese T. Jones, M.D.
Chair, Committee on Human Research

APPENDIX B

HUMAN SUBJECTS CONSENT FORM

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO
CONSENT TO BE A RESEARCH SUBJECT

Heel Pressure Ulcer Risk in Hip Surgery Patients

A. PURPOSE AND BACKGROUND

Nancy Stotts, RN, EdD and Vivian Wong, RN, MSN from the Department of Physiological Nursing are conducting a study to learn more about how to prevent heel damage in adults who have hip surgery. Specifically we are interested in heel skin oxygen and temperature, and how they are affected when the heels are placed on the surface of the bed. This study is funded by the National Institute of Nursing Research.

You are being asked to participate in this study because you are having hip surgery.

B. PROCEDURES

If you agree to be in this study, we will first ask you 10 short questions about your memory and knowledge of the surroundings and current events. We will then use a thin, plastic-like filament to touch a few spots on the sole of each foot to see if you can feel the touch sensation. Next, we will take blood pressure on your arm and lower legs, which will give us an idea of how well blood flows in the small blood vessels on your legs. These procedures help us to determine if you can be enrolled in our study.

Data will be collected 3 times: on post-operative days 1, 2, and 3. The data collection procedure will be done in your hospital room with you in your hospital bed. We will use the following procedures:

1. Baseline
   1.1. While lying on your back, a small pillow will be placed under your calf to suspend your heels at about 15° for about 15 minutes.
   1.2. You will be asked to rate the amount of pain you have and asked to show us where the pain is located, using a body diagram.
   1.3. The amount of oxygen carried in your blood will be measured with a soft sensor on your finger.
   1.4. A warm oxygen sensor and a temperature sensor will be taped to each heel.
   1.5. Heel oxygen tension and heel skin temperature will be continuously recorded.
2. Heel on Bed Phase
   2.1. The pillow underneath your calf will then be removed and your heels will be placed on the bed surface for 15 minutes.
   2.2. Before the end of the 15 minutes, you will be asked about your pain intensity and location.
3. Recovery Phase
   3.1. At the end of 15 minutes, the pillow again will be placed under your calf and your heels will be kept off the bed surface for the 15 minutes.
   3.2. Before the end of the 15 minutes, you will be asked about pain intensity and pain location.
4. Oxygen Challenge

4.1. You will be given 7-10 liters of oxygen per minute (an oxygen challenge) using a face mask. Oxygen carried by blood will be recorded using the same soft sensor around your finger.

4.2. The procedures 1, 2, and 3 above will be repeated while you are breathing oxygen.

5. Post operative visit

5.1 At your first doctor's appointment after surgery, we will meet you at the office and observe your heel skin condition. This will take less then 5 minutes.

Participation in the study will take a total of about 6.0 hours. All study procedures will be done in the hospital and at the doctor's office.

C. RISKS/DISCOMFORTS

1. Monitoring oxygen level in your blood using the finger sensor may cause discomfort to your finger, but you may ask the researcher to re-adjust the finger sensor or decline the monitoring.
2. The sensors for measuring heel skin oxygen tension and temperature may cause discomfort or irritation. If this happens, you may ask the researcher to place the sensors on different locations on the heels or decline the measurements and withdraw from the study.
3. Lifting your legs and placing a pillow under your calf or removing the pillow and putting the heels on the bed surface may cause discomfort to your legs and/or heels. However, this is not different from usual care in the hospital. If this happens, you may ask the researcher to place your legs in the most comfortable position or decline participation in the procedure.
4. Confidentiality: Participation in research may involve a loss of privacy, but information about you will be handled as confidentially as possible. If you sign this consent form, you are allowing Nancy Stotts and Vivian Wong to review your medical records. Your name will not be used in any published reports about this study.

Treatment and Compensation for Injury:

If you are injured as a result of being in this study, treatment will be available. The costs of such treatment may be covered by the University of California, depending on a number of factors. The University does not normally provide any other form of compensation for injury. For further information about this, you may call the office of the Committee on Human Research at (415) 476-1814

D. BENEFITS

There will be no direct benefit to you from participating in this study. However, the information gained from the study will help the researchers learn more about how skin oxygenation and skin temperature in the heel of the operative or non-operative leg are related to external pressure, such as a bed surface. It may help nurses and other health care providers understand how pressure ulcers on the heel are formed.

E. ALTERNATIVES

The alternative to participation is not to participate. If you choose not to participate in this study, you will still receive the same level of medical management from your doctor and the same level of nursing care.

F. COSTS

You will not be charged for any of the study procedures. The costs of all tests associated with this study will be covered by the study.

G. PAYMENT

You will not be paid for participating in this study.

H. QUESTIONS

This study has been explained to you by Vivian Wong who signed below and your questions were answered. If you have any other questions about the study, you may call Vivian Wong or Nancy Stotts at (415) 476-4412.

I. CONSENT

You have been given copies of this consent form and the Experimental Subject's Bill of Rights to keep.

You will be asked to sign a separate form authorizing access, use, creation, or disclosure of health information about you.

PARTICIPATION IN RESEARCH IS VOLUNTARY. You have the right to decline to participate or to withdraw at any point in this study without penalty or loss of benefits to which you are otherwise entitled.

If you wish to participate in this study, you should sign below.

Date	Subject's Signature for Consent
Date	Person Obtaining Consent

APPENDIX C

RECRUITMENT FLIER

Heel Pressure Ulcer Risk in Hip Surgery Patients *

Are you 21 years and older?
Are you going to have hip surgery?

If your answers are "YES" to the above questions, you are invited to participate in a study.

Purpose of the study: To help us understand more about how pressure (such as the bed surface) affects the oxygen level and temperature in the heel skin after hip surgery.

What is involved in the study?

- Your oxygen level will be checked with a finger sensor.
- The oxygen and temperature of your heel skin will be measured with sensors placed on your heels.
- Your heels will be suspended above the bed with a pillow placed under your calf.
- Your heels will be returned to the bed surface.
- You will be asked to breathe oxygen and the heel suspension on bed surface process will be repeated.

How many study visits will there be?

- 3 visits while you are in the hospital
- Follow up in your surgeon's office or phone call

If interested in the study, please let your doctor or the office personnel know or call (415) 476-4412. The nurse researcher will call you, explain the study, and determine if you would like to participate.

Thank you in advance for your interest in this study!

* A study conducted by Vivian Wong & Nancy Stotts from the University of California, San Francisco

APPENDIX D

DATA COLLECTION FORMS

Demographic Data Collection

Subject ID # _____

Age: ____
Gender: 0 – male
1 – female
Diagnosis: _____

Ethnicity & race:
1 – American Indian or Alaska Native
2 - Asian
3 – Black or African American
4 – Native Hawaiian or other
5 – White

1. Hispanic
2. Non-Hispanic

Current smoker:
0 – No
1 – Yes

Smoking history:
# of cigarettes/cigars/pipes per day: ___
Smoking since (year)____
Quit smoking in _____ month/year
Relapse smoker ____

Major health conditions:
__ DM
__ Peripheral vascular disease
__ Stroke
__ Femoral-popliteal bypass
__ Deep vein thrombosis
__ Hip surgeries
__ Foot surgeries
__ Other

Estimated blood loss during surgery: _____ c.c. Duration of surgery (minutes)_____
Type of anesthesia:
_____

ASA_____ Surgical risk _____

Level of sedation in PACU: _____

Current Medications: _____
_____
_____

Type of mattress: _____

Data collection Post-op Day 1

Breathing Room air **Date:** _____ **Subject ID#** _____

PaO_2 = _____% Heel conditions: Rt._____ Sensation: +/- Lt.___Sensation: +/- Operative leg: 1 – Right 2 – Left

Time series	Preload		Pressure loading for 15 minutes										Pressure relief for 15 minutes													
Time mins			3		6		9		12		15		1		2		3		6		9		12		15	
heel	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
$PtcCO_2$																										
Heel temp																										
$PtcO_2$																										
Pain scores and location																										
VAS																										
location																										

With Oxygen Challenge PaO_2 = _____%

Time series	Preload		Pressure loading for 15 minutes										Pressure relief for 15 minutes													
Time mins			3		6		9		12		15		1		2		3		6		9		12		15	
heel	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
$PtcCO_2$																										
Heel temp																										
$PtcO_2$																										
Pain scores and location																										
VAS																										
location																										

Data collection Post-op Day 2

Breathing Room air **Date:** _____ **Subject ID#** _____

PaO_2 = _____% Heel conditions: Rt._____ Sensation: +/- Lt.___Sensation: +/- Operative leg: 1 – Right 2 – Left

Time series	Preload		Pressure loading for 15 minutes										Pressure relief for 15 minutes													
Time mins			3		6		9		12		15		1		2		3		6		9		12		15	
heel	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
$PtcCO_2$																										
Heel temp																										
$PtcO_2$																										
Pain scores and location																										
VAS																										
location																										

With Oxygen Challenge PaO_2 = _____%

Time series	Preload		Pressure loading for 15 minutes										Pressure relief for 15 minutes													
Time mins			3		6		9		12		15		1		2		3		6		9		12		15	
heel	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
$PtcCO_2$																										
Heel temp																										
$PtcO_2$																										
Pain scores and location																										
VAS																										
location																										

Data collection Post-op Day 3

Breathing Room air **Date:** _____ **Subject ID#** _____

PaO_2 = _____% Heel conditions: Rt._____ Sensation: +/- Lt.___Sensation: +/- Operative leg: 1 – Right 2 – Left

Time series	Preload		Pressure loading for 15 minutes										Pressure relief for 15 minutes													
Time mins			3		6		9		12		15		1		2		3		6		9		12		15	
heel	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
$PtcCO_2$																										
Heel temp																										
$PtcO_2$																										
Pain scores and location																										
VAS																										
location																										

With Oxygen Challenge PaO_2 = _____%

Time series	Preload		Pressure loading for 15 minutes										Pressure relief for 15 minutes													
Time mins			3		6		9		12		15		1		2		3		6		9		12		15	
heel	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
$PtcCO_2$																										
Heel temp																										
$PtcO_2$																										
Pain scores and location																										
VAS																										
location																										

APPENDIX E

MENTAL STATUS QUESTIONNAIRE

Subject ID # _____

Mental Status Questionnaire

1. What is the name of this place? _____
2. Where is it located (address)? _____
3. What is today's date? _____
4. What is the month now? _____
5. What is the year? _____
6. How old are you? _____
7. When were you born (month)? _____
8. When were you born (year)? _____
9. Who is the president of the United States? _____
10. Who was the president before him? _____

Adapted from Kahn, R. L., Goldfarb, A. I., Pollack, M., & Peck, A. (1960). Brief objective measures for the determination of mental status in the aged. *Am J Psychiatry, 117*, 326-328.

Number of correct items = _____