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Predictors of Pressure Ulcer Development in Adult Critical Care Patients

by

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A Dissertation submitted to the

Graduate School-Newark

Rutgers, The State University of New Jersey

In partial fulfillment of the requirements

for the degree of

Doctor of Philosophy

Graduate Program in Nursing

Written under the direction of

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Newark, New Jersey

May 2010

## ABSTRACT OF THE DISSERTATION

### Predictors of Pressure Ulcer Development in Adult Critical Care Patients

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The purpose of this study was to examine the relationships between theoretically- and empirically derived risk factors and pressure ulcer development in adult critical care patients. Theoretically-derived risk factors under investigation included mobility, activity, sensory perception, moisture, friction/shear, nutrition, age and arteriolar pressure. Empirically-derived risk factors under investigation included length of intensive care unit stay, severity of illness, vasopressor administration and comorbid conditions.

The sample was comprised of 347 patients admitted into a medical surgical intensive care unit from October 2008 through May 2009. Data was abstracted from various sources within the patient's computerized medical record. Hypotheses testing consisted of both correlational and logistic regression analysis.

Significant correlations were found between the following theoretically-derived risk factors: total Braden scale score, representing cumulative risk, mobility, sensory perception, friction/shear, nutrition, age and arteriolar pressure. Empirically-derived risk factors significantly associated with pressure ulcer development were length of ICU stay, severity of illness, norepinephrine, vasopressin and the comorbid conditions of cardiovascular disease and infection. In logistic regression analysis, the variables mobility, age, intensive care unit length of stay and cardiovascular disease explained a significant portion of the variance in pressure ulcer development in this study sample.

This study contributed to the body of knowledge regarding pressure ulcer risk factors that confront the critically ill, however, more empirical evidence is needed to further validate these risk factors in the ICU population. Development of an ICU pressure ulcer risk assessment model or refinement of the Braden and Bergstrom conceptual framework is warranted in order to appropriately and more fully explain pressure ulcer development in this population. This risk assessment model may then serve as the basis for the development of a risk assessment tool designed specifically to measure pressure ulcer risk in adult critical care patients.



## Acknowledgements

First, I would like to offer my sincere thanks to Dr. Charlotte Thomas-Hawkins for her countless hours of mentoring during the creation of this dissertation. I am truly grateful to you for all of your knowledge, guidance and patience. To my dissertation committee members, Dr. Lucille Eller, Dr. Nancy Gardner and Dr. John Kolassa, thank you all for your willingness to share your expertise and feedback with me during the creation of this dissertation.

To my colleagues at Englewood Hospital and Medical Center, including the critical care team, the nursing executive team and my fellow advanced practice nurses, who have been with me throughout this entire journey-thank you for sharing your knowledge, support, and kind words. To my long-time colleague, Dr. Sharon Roche, a special thank you for your unwavering willingness to share your clinical expertise and more importantly for your true gestures of friendship. To Lia Sabbagh, research librarian at the medical center, I am eternally grateful for your kind manner and assistance in obtaining my many reference requests.

This dissertation is dedicated to my family, my sons, Greg and Glenn and my husband Les, who have seen me through this entire journey, and always supported and encouraged me along the way. To my parents, my mom and my late dad, who have been steadfast supports in all of my life's endeavors and to my sister Nancy, who has since birth been my constant companion in life's journey.

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## CHAPTER 1

### The Problem

Pressure ulcers are observed in patients across the care continuum including the intensive care unit (ICU). In the critically ill, the presence of a pressure ulcer poses an additional comorbid threat in an already physically compromised patient. Theaker and colleagues (2000) cite pressure ulcers as one of the most under-rated medical problems that plague the critically ill. Despite advances in medical technology and the use of formalized prevention programs, the numbers of pressure ulcers acquired during hospitalizations continue to grow. The Healthcare Cost and Utilization Project (2006) found that pressure ulcer occurrence increased 63% in hospitalized patients in the decade between 1993 and 2003, with only an 11% increase in the total number of hospitalizations during that same time period. By 2006, an 80% increase in pressure ulcer occurrence was documented by the Healthcare Cost and Utilization Project between 1993 and 2006 (Russo et. al, 2008). In the ICU setting, prevalence rates of acquired pressure ulcers are cited as the highest among hospitalized patients ranging from 14% to 42% as compared to acquired pressure ulcer prevalence rates of 14 % to 17% among patients in the general hospital setting, (Keller et al, 2002; Whittington & Briones, 2004). Incidence rates of pressure ulcers acquired in the ICU are cited between 8% and 33% (Eachempati, 2001; Wolverson et al., 2004).

The human consequences associated with the development of a pressure ulcer can not be overestimated. Diminished quality of life, pain and body image disturbances have all been reported as sequelae of pressure ulcer development (Langemo et al. 2000; Meehan, 2000; Quarino et al., 2003; Rastinehead, 2006). In addition, the presence of a

pressure ulcer is associated with prolonged hospital lengths of stay, increased risk of nosocomial infection and increased mortality (Allman et al., 1999; Bo et. al, 2003; Graves et. al., 2005; Redelings, Lee & Sorvillo, 2005).

The financial impact of pressure ulcer development can be staggering. Estimated costs associated with the management of pressure ulcers in the United States (U.S.) are cited between 5 and 11 billion annually (Beckrich and Aronovitch, 1999; Reddy et. al., 2006). In 2008, the Center for Medicare and Medicaid Services (CMS) imposed reimbursement restrictions for care associated with hospital acquired pressure ulcers adding to the financial burden of acute care facilities (Center for Medicare and Medicaid Services, 2008). These restrictions are based on the premise that a hospital acquired pressure ulcer is largely a preventable condition if adequate prevention programs are implemented. According to CMS (2006), pressure ulcer occurrence in hospitalized patients is considered an adverse patient safety event that is associated with high volume, high cost and could reasonably be prevented through the use of evidenced based guidelines. In an attempt to control costs for health care events that are considered preventable, CMS ceased reimbursement to hospitals for care associated with hospital acquired Stage III and Stage IV pressure ulcers beginning in 2008. Thus, there is an urgent need to identify modifiable risk factors for pressure ulcer development in patients admitted to ICU settings.

ICUs provide highly complex care to the sickest patients in our health care system. Approximately 10% of all acute care hospital beds in the U.S. can be found in ICUs (Agency for Health Care Research & Quality (AHRQ), 2001). Annually, 4.4 million patients are admitted into the intensive care setting at a cost of approximately 60 billion

dollars. The aging ICU population coupled with increases in the acuity of the ICU patient projected by AHRQ (2001) will also contribute significantly to a growth in hospital acquired pressure ulcers. Moreover, high disease burden, coupled with the need for multiple life saving technologies place ICU patients at the greatest risk for pressure ulcer development (Krapfl & Mackey, 2008). Thus, there is a need to examine modifiable factors that contribute to pressure ulcer development in the critical care population.

Clinical practice guidelines for pressure ulcer prevention have been developed by the Agency for Healthcare Policy and Research (now the Agency for Healthcare, Research and Quality) (1992), the Wound, Ostomy and Continence Nursing Society (2003) and the Registered Nurses Association of Ontario (2005). All of these guidelines recommend the assessment of predisposing factors that put patients at risk for pressure ulcer development using a validated risk assessment tool. The purpose of risk assessment is to assist the clinician in determining patients at risk for pressure ulcer development, thus leading to the implementation of appropriate pressure ulcer prevention strategies in patient care. In the U.S., the Braden scale (Bergstrom et al., 1987) is the most widely used tool for identifying patients at risk for pressure ulcer development and is recommended for use in all three of these clinical practice guidelines. The Braden scale is based on the conceptual framework for pressure ulcer development developed by Braden and Bergstrom (1987).

Braden and Bergstrom (1987) and other theorists posit that the relationship between intensity and duration of pressure is a considerable factor in the development of pressure ulcers (Husain, 1953; Kosiak, 1959). Both Husain (1953) and Kosiak (1959) found an inverse relationship between duration and intensity of pressure in creating tissue ischemia



and pressure ulcer formation. Low intensity pressure for a prolonged period or high intensity pressure for a short period of time can both lead to tissue ischemia and pressure ulcer formation especially over a bony prominence. Braden and Bergstrom (1987) identified that activity, mobility and sensory perception (the patient's ability to perceive and respond to prolonged pressure) all play a role in the body's ability to compensate for intense and prolonged pressure. All theorists agree that the relationship between altered sensory perception and pressure ulcer development is significant in pressure ulcer development.

Tissue tolerance for pressure is the second factor purported to be related to pressure ulcer development (Braden & Bergstrom, 1987; Kosiak 1959). Kosiak (1959) found that metabolic factors including poor nutrition, anemia and edema render body tissues less tolerant for pressure. Braden and Bergstrom (1987) postulate that certain intrinsic and extrinsic factors restrict the ability of the patient to tolerate pressure and increase the patient's risk of pressure ulcer development. These factors include friction and shear, moisture, poor nutritional intake, age and low arteriolar pressure.

Empirical evidence is limited supporting the individual risk factors theoretically purported to be related to pressure ulcer development in the ICU population as measured using the Braden scale. While evidence supports the total Braden score as a predictor of pressure ulcer development in the ICU population (Bours et al., 2001; Carlson et al., 1999; Fife et al, 2001; Jiricka et al., 1995; Wolverson et al., 2005), the measurement of the contributions of the individual risk factors has not garnered strong empirical support, and has been subject to limited investigation. Current clinical practice guidelines recommend that patients be screened for the risk of pressure ulcer development using a

validated risk assessment tool such as the Braden scale (AHRQ, 1992; Ontario Nurses Association, 2005; Wound, Ostomy and Continence Nursing Society, 2003) however, the level of evidence supporting this risk assessment is largely based on Level IV or “C” level evidence, defined within these guidelines as evidence obtained from one of the following: expert committee reports, expert opinion, one supporting controlled trial, two case studies or clinical experiences of authorities in the field (AHRQ, 1992; Ontario Nurses Association, 2005; Wound, Ostomy and Continence Nursing Society, 2005). Clearly, guidelines for assessment of risk factors for pressure ulcer development should be informed by a body of large, rigorous outcome studies that collect and analyze data related to risk factors that contribute to pressure ulcer development in ICU patients.

There are also varying degrees of empirical support for additional risk factors not measured by the Braden scale that may potentially alter tissue tolerance and contribute to pressure ulcer development in ICU patients. These factors include advancing age (Bours et al, 2001; Frankel et al., 2007), low arteriolar pressure (Batson, 1993), prolonged intensive care unit length of stay (Bours et al., 2001; Eachampeti et al., 2001; Theaker et al., 2001), severity of illness, (Theaker et al., 2000), comorbid conditions such as diabetes, sepsis, and vascular disease (Batson et al., 1993; Bours et al., 2001; Frankel, Sperry & Kaplan, 2007) and iatrogenic factors such as the use of vasopressor agents (Batson et al., 1993; Frankel et al., 2007; Theaker et al., 2000). Thus, while the Braden scale is the most widely used tool for assessment of modifiable pressure ulcer risk factors, findings from the empirical literature suggest that additional risk factors not assessed via this instrument may also be important determinants of pressure ulcer development in adult critical care patients.

Studies in the critical care population yield a high degree of variability regarding the significant risk factors that confront this population. Consequently, there is a lack of consensus among researchers and practitioners regarding the factors that afford the most significant risks for pressure ulcer development in ICU patients, making it difficult to determine which prevention strategies would be the most beneficial to patient care. The multiplicity of factors represented in the literature also illustrates the multifactorial nature of pressure ulcer development. In fact, a systematic review of empirical research that focused on pressure ulcer epidemiology, risk factors and prevention in critical care patients found no conclusive evidence across studies to support specific risk factors that consistently confront critically ill patients (de Laat, et al., 2006). In fact, AHRQ has pointed to the need for research that explicates the essential risk factors for pressure ulcer development in hospitalized patients (Ayello & Lyder, 2008).

The purpose of this study was to identify which risk factors derived from both the theoretical and empirical literature best predict pressure ulcer development in adult critical care patients. The intention is that this study will contribute to the body of knowledge regarding the full range of risk factors that significantly influence pressure ulcer development in this population. Findings from this study will provide evidence regarding the essential risk factors that pose the greatest risk for pressure ulcer development in ICU patients.

#### Statement of the Problem

1) What risk factors significantly predict pressure ulcer development in adult critical care patients?

### Subproblems

- 1) What are the relationships between the Braden scale risk factors (mobility, activity, sensory perception, moisture, nutrition, friction and shear) and other theoretically-derived risk factors (age and arteriolar pressure) to pressure ulcer development in adult critical care patients?
- 2) What Braden scale risk factors predict pressure ulcer development in adult critical care patients?
- 3) What are the relationships between the empirically-derived risk factors (ICU length of stay, severity of illness, vasopressor administration, comorbid conditions) and pressure ulcer development in adult critical care patients?
- 4) What empirically-derived risk factors predict pressure ulcer development in adult critical care patients?
- 5) Which theoretically- and empirically-derived risk factors, taken together pose the greatest threat for pressure ulcer development in terms of variance accounted for in adult critical care patients?

### Definition of Terms

A pressure ulcer is conceptually defined as a localized injury to the skin, and/or underlying tissue usually over a bony prominence that develops as a result of pressure or pressure in combination with shear and/or friction and is staged according to the degree of damage clinically observed (National Pressure Ulcer Advisory Panel, 2007). A Stage I ulcer is as defined as intact skin with non-blanchable redness of a localized area. A Stage II ulcer is defined as partial thickness skin loss presenting as a shallow open ulcer with a red/pink wound base. A Stage III ulcer is defined as full thickness tissue loss with visible

subcutaneous tissue. A Stage IV ulcer defined as full thickness tissue loss with exposed muscle, bone or tendon. Suspected deep tissue injury is defined as a purple or maroon localized area of discolored intact skin or a blood filled blister. Unstageable is defined as an ulcer with full thickness tissue loss in which the base of the ulcer is covered with slough or eschar. Operationally, a pressure ulcer is defined as the presence of a pressure ulcer of any stage after admission to the ICU.

Mobility is theoretically defined as the ability to change and control body positions (Braden & Bergstrom, 1987). Altered mobility increases the chance that a person will be exposed to prolonged and intense pressure. Operationally, mobility is defined as the patient's score on the Braden scale mobility subscale (Bergstrom & Braden, 1987).

. Activity is theoretically defined as the overall degree of physical activity (Braden & Bergstrom, 1987). Activity is operationally defined as the patient's score on the Braden scale activity subscale (Bergstrom & Braden, 1987).

. Sensory perception is theoretically defined as the ability of the individual to perceive and respond to discomfort as a result of exposure to pressure (Braden & Bergstrom, 1987). Operationally, sensory perception is defined based on the patient's score on the Braden scale sensory perception subscale (Bergstrom & Braden, 1987).

Friction is defined by Braden and Bergstrom (1987) as the force that results when two surfaces move across each other such as occurs from dragging a patient to change position. Shear is theoretically defined as two opposing surfaces sliding over each other causing destruction and deformation of vascular bed (Braden & Bergstrom, 1987). Operationally, friction and shear are defined as the patient's score on the Braden scale friction/shear subscale (Bergstrom & Braden, 1987).

Moisture is theoretically defined as exposure of the skin to perspiration, urine, stool and drainage from wounds or fistulae (Braden & Bergstrom, 1987). Moisture is operationally defined as the patient's score on the Braden scale moisture subscale (Bergstrom & Braden, 1987).

Nutrition is theoretically defined as the usual nutritional intake of the patient inclusive of oral, parenteral or enteral feeding routes (Braden & Bergstrom, 1987). Nutrition is operationally defined as the patient's score on the Braden scale nutrition subscale (Bergstrom & Braden, 1987).

Low arteriolar pressure is theoretically defined as a diastolic blood pressure below 60 mm Hg (Braden & Bergstrom, 1987). Operationally, low arteriolar pressure is defined as the total number of hours in the first 48 hours of the ICU admission that the patient experiences one or more of the following: mean arterial pressure below 60mm Hg; systolic blood pressure below 90; diastolic blood pressure below 60 (National Institute of Health, 2008; Society for Critical Care Medicine, 1999).

Age is theoretically defined as the chronologic age of the patient, that is the number of years elapsed from birth to a given time (McPherson, 2008). Operationally, age is defined as the chronologic age of the patient at the time of admission into the intensive care unit.

Intensive care unit length of stay is theoretically defined as the total amount of time of the intensive care unit admission. Intensive care unit length of stay is operationally defined as the total number of hours the patient spent in an intensive care unit bed.

Severity of illness is theoretically defined as the factors that influence the outcome of a severe illness based on a degree of acute illness and chronic health conditions (Knaus

et al., 1985). The patient's APACHE II (Acute Physiologic and Chronic Health Evaluation) score operationally defines severity of illness (Knaus et al., 1985).

Vasopressor agents are theoretically defined as a class of drugs that induce vasoconstriction for the purpose of elevating mean arterial pressure (Gooneratne & Manker, 2007). Vasopressor agents are operationally defined as the total number of hours of administration of one or more of the following vasopressor agents: norepinephrine, epinephrine, phenylephrine, vasopressin or dopamine during the ICU admission.

Comorbidity is theoretically defined as a concomitant but unrelated pathologic or disease process (Stedman, 2008). Comorbidity is operationally defined as the presence of any of the following conditions prior to or present during the intensive care unit admission: diabetes mellitus, vascular disease, and infection/sepsis.

Adult critical care patients are defined as those individuals over the age of eighteen that meet the admission criteria for the intensive care unit. Criteria for admission to an intensive care unit include but are not limited to the following conditions: respiratory failure, hemodynamic instability, patients requiring vasoactive medications, invasive hemodynamic monitoring, acute neurological dysfunction, treatment of hypotension or shock, severe metabolic/electrolyte derangement, acute renal failure requiring continuous venovenous hemofiltration (CVVH), and patients undergoing high risk or extensive surgical procedures (Society for Critical Care Medicine, 1999).

### Delimitations

The empirical literature suggests a multiplicity of pressure ulcer risk factors that confront critically ill adults. Critically ill patients represent a unique subset of hospitalized patients. Due to a high burden of illness, among hospitalized patients, these

patients may be at greatest risk for pressure ulcer development. Since this study seeks to determine risk factors that predict pressure ulcer development in critically ill adults, this sample will be delimited to adult patients that are admitted into the medical/surgical intensive care unit for greater than 24 hours without an existing pressure ulcer.

### Significance of the Study

Despite the development of clinical practice guidelines aimed at pressure ulcer prediction and prevention by the AHRQ (formerly the Agency for Health Care Policy and Research) in 1992, pressure ulcer rates in hospitalized patients have continued to increase. The Healthcare Cost and Utilization Project (2006) found that the occurrence of pressure ulcers of all stages increased 63% in hospitalized patients in the decade between 1993 and 2003, with only an 11% increase in the total number of hospitalizations during that same time period (Russo & Elixhauser, 2003). By 2006, an 80% increase in pressure ulcer occurrence was documented by the Healthcare Cost and Utilization Project between 1993 and 2006 (Russo et. al, 2008). Both Cuddigan and colleagues (2001) and Maklebust (2005) concur that a sustained nationwide reduction in pressure ulcer prevalence has not been realized despite the introduction of clinical practice guidelines and advances in available prevention technologies.

Negative patient outcomes have been associated with pressure ulcer development. In hospitalized patients, the presence of a pressure ulcer has been found to significantly increase the risk of nosocomial infection including the development of fatal septicemia (Allman et al, 1999, Redelings et al., 2005). Increased mortality rates have also been associated with pressure ulcer development. Brown (2003) found one-year mortality rates to be 78% in patients with hospital acquired Stage III and Stage IV pressure ulcers. In



intensive care patients, the presence of a pressure ulcer has been significantly associated with in-hospital mortality (Bo et al., 2003). Empirical evidence also supports that pressure ulcers have a profound impact on the overall quality of patient's lives including pain, social concerns, body image disturbances as well as a loss of independence and control (Langemo et. al, 2000; Meehan, 2000; Quarino, et. al, 2003; Rastinehad, 2006; Spilsbury et al., 2007).

Costs associated with pressure ulcer development and treatment continue to spiral. Overall health care expenditures associated with pressure ulcers in the U.S. are estimated to be as high as \$11 billion annually (Reddy et al, 2006). In 2007, CMS reported 257,412 cases of hospital acquired Stage III and Stage IV pressure ulcers with a mean cost of treatment reported at \$43,180 per pressure ulcer. According to the HealthGrades Patient Safety in American Hospitals study (2008) \$2.47 billion in excess health care costs were required for the treatment of hospital acquired pressure ulcers of all stages between the years 2004 and 2006. In addition, empirical studies have found that pressure ulcer development is associated with prolonged lengths of hospital stay further contributing to increased health care costs (Allman et. al., 1999; Beckrich & Aronovitch, 1999; Graves et. al., 2005).

According to CMS (2006), Stage III and Stage IV pressure ulcer occurrence in hospitalized patients is an adverse patient safety event that is associated with high volume, high cost and could be reasonably prevented through the use of evidence based guidelines. In the HealthGrades Patient Safety in American Hospitals Study (2008), pressure ulcers of any stage were identified as one of the top three most commonly occurring patient safety events in U.S. hospitals along with failure to rescue and post-

operative respiratory failure. According to the report, 455,305 pressure ulcers occurred in U.S. hospitals between 2004 and 2006, accounting for 40% of all patient safety events. In an attempt to control costs for health care events that are considered preventable, CMS imposed reimbursement restrictions to hospitals for care associated with hospital acquired Stage III and Stage IV pressure ulcers beginning in 2008. The financial, safety and reimbursement implications possess the power to ignite a greater urgency to the need to identify significant risk factors in an effort to thwart pressure ulcer development.

The shift of financial responsibility for pressure ulcer development by CMS to acute care institutions reflects a pervasive belief that pressure ulcer development is largely a preventable condition. The debate over the preventability of pressure ulcers has been ongoing for the past decade with experts seated on both sides of the argument. Whether all pressure ulcers are truly avoidable is debatable (Glover, 2005); however a paucity of evidence exists to refute this claim.

Despite quality care and best practice, pressure ulcers do develop in hospitalized patients. For patients admitted to an intensive care unit, the risk of pressure ulcer occurrence is even greater. Empirical evidence supports the hypothesis that admission to an intensive care unit significantly increases a patient's risk of pressure ulcer development (Baumgarten et. al, 2003; Baumgarten et. al., 2008). Even with consistent and ongoing skin assessment, early identification of skin changes and the implementation of appropriate strategies to minimize damage, skin and tissue damage may be unavoidable in critically ill patients (Langemo & Brown, 2006). In addition, current clinical practice guidelines encompassing pressure ulcer risk assessment have been largely informed by expert opinion. There is an urgent need for rigorous empirical

investigation of modifiable pressure ulcer risk factors to inform evidence-based policies and practices for pressure ulcer prevention.

A gap also exists in the current knowledge regarding the most significant risk factors for pressure ulcer development that confront critically ill patients. In a systematic review of the empirical research focused on pressure ulcer epidemiology, risk and prevention in critical care patients, de Laat and colleagues (2006) found that findings across studies did not yield conclusive evidence to support specific risk factors that consistently confront critically ill patients. The authors cited that differences in methodologies, variables under study, outcomes and populations precluded the ability to make any meaningful comparisons between studies. These findings corroborate the results of Keller and colleagues (2002) in an earlier review of the empirical literature of pressure ulcer risk and prevention in critical care patients, who also found weak evidence supporting specific significant pressure ulcer risk factors. The lack of a risk assessment tool developed exclusively for critical care patients was also found in both reviews to be an impediment to accurately determining pressure ulcer risk in this population. Both Keller and colleagues and de Laat and colleagues cite the need for well designed research focused on risk factors and risk assessment in the critically ill to gain more insight into this clinical problem. Moreover, Ayello and Lyder (2008) in an AHRQ publication on patient safety and quality note that while many pressure ulcer risk factors have been identified in the literature, a hierarchy of risk factors has not yet been determined. The need for research to determine the essential risk factors is also recommended by these authors.

There is a paucity of recent studies that have examined risk factors for pressure ulcer development in the critical care population. In the past five years, only three studies have been identified that specifically investigated pressure ulcer risk factors that confront the critically ill (Frankel et.al, 2007; Pender & Frazier, 2005; Wolverton et. al, 2005). Current research that identifies risk factors for pressure ulcer development is crucial to guide prevention policies and practices as new advances in technology in the intensive care environment have the potential to alter the relevant risk factors that confront critically ill patients.

The first step toward prevention of pressure ulcers is to determine what constitutes appropriate pressure ulcer risk. Once risk factors are accurately identified, policy makers, practitioners and other key stakeholders will be able to establish the foundation for the identification and implementation of focused prevention strategies for this population. According to Langemo and colleagues (2008), a gap exists in the current knowledge regarding the pressure ulcer prevention strategies that would best meet the needs of the critical care population. In this era of fiscal responsibility and accountability, the provision of risk appropriate prevention interventions that will afford the best patient outcomes is both clinically and economically prudent. Without prevention strategies targeted at the most significant risk factors, health care dollars have the potential to be spent on interventions that could provide little or no effect on patient outcomes and may contribute to inefficiency in the use of caregiver time.

The intent of this study is to fill a gap in knowledge regarding the factors that pose the greatest risk for pressure ulcer development in adults in critical care units. Both theoretically-derived and empirically-derived risk factors will be examined in this study

since a rigorous identification of pressure ulcer risk factors, based on both the theoretical and empirical literature is an important prerequisite for the reduction of incidence of hospital acquired pressure ulcers and the associated negative sequelae in this population. Ultimately, the accurate identification of risk factors that pose the greatest threat can lead to the testing and implementation of evidence-based pressure ulcer prevention strategies that can translate into reductions in pressure ulcer occurrence and health care costs and promote positive health outcomes in critical care patients.

## CHAPTER 2

### Review of the Literature

This research will examine the relationships between risk factors gleaned from the theoretical and empirical literature and the development of pressure ulcers in hospitalized critically ill adult patients. In this chapter, the theoretical underpinnings of the etiology of pressure ulcer development are discussed followed by a review of empirical support for the theoretical propositions to be tested in this study. The review of the theoretical literature of pressure ulcer development encompasses three conceptual viewpoints and is presented in chronological order. Since the development of a pressure ulcer is a pathophysiologic phenomenon, all theoretical perspectives are based on a synthesis of the available physiologic and empirical literature of the time. A review of risk factors for pressure ulcer development in the critical care population gleaned from the empirical literature is also presented, followed by the theoretical rationale for the study and the hypotheses to be tested.

### Theoretical Framework

The works of early theorists (Husain, 1953; Kosiak, 1959) emphasized the significance of the relationships between 1) intense and prolonged pressure and pressure ulcer development and 2) tissue tolerance and pressure ulcer development. Current theoretical perspectives of pressure ulcer development continue to support these relationships based on the research of these theorists.

Husain's (1953) work stressed the relationship between intense and prolonged pressure and pressure ulcer development. In his experiments with rats, Husain found that 100 mmHg of pressure applied for two hours produced only microscopic changes in the

muscle. When 100 mmHg was applied for six hours, severe changes occurred in the muscle. Therefore, identical levels of pressure are capable of causing different degrees of tissue injury based on the amount of time the pressure is sustained.

Husain's (1953) work also supported the relationship between tissue tolerance and pressure ulcer development. Husain found that rat muscle sensitized to 100 mm Hg of pressure for two hours was less able to tolerate subsequently lower applied pressures (50 mmHg). This resulted in the destruction of muscle due to the inability of the muscle to tolerate pressure.

Kosiak (1959), in his work on the etiology and pathology of ischemic ulcers found that ischemic ulcers occurred more commonly in debilitated patients who were unable to change position independently due to either a loss of strength or a loss of sensation. The inability of the patient to sense pain from pressure over a bony prominence was a key factor in pressure ulcer development. Kosiak found a high incidence (85%) of ischemic ulcers in paralyzed World War II veterans. He purported three contributing factors to pressure ulcer development and classified these as ischemic factors, neurotrophic factors and metabolic factors.

According to Kosiak (1959), prolonged tissue ischemia caused by pressure exceeding tissue capillary closing pressure, especially over a bony prominence, is a significant contributor to pressure ulcer development. Similar to the work of Husain (1953), Kosiak found a relationship between the intensity and duration of pressure and pressure ulcer development. In his experiments with dogs, Kosiak (1961) concluded that high levels of pressure for short periods of time or low levels of pressure for long durations of time were both equally capable of yielding ischemic changes and pressure

ulcer formation. Kosiak postulated that time and intensity of pressure creates an inverse relationship in the development of pressure ulcers.

Neurotrophic factors include disturbances in neurological pathways from spinal cord or peripheral nerve injuries. Kosiak (1959) hypothesized that factors such as a loss of sensation which prevents the patient from experiencing pressure and pain over a bony prominence, and a loss of motor function interfere with the patient's ability to change position, thereby leading to pressure ulcer formation. Kosiak also posited that metabolic factors such as nutritional deficits, peripheral edema and anemia can all influence the ability of the tissues to tolerate pressure thus leading to pressure ulcer development. According to Kosiak, poor nutrition makes the patient less tolerant of the forces of pressure; edema hinders the flow of oxygen and nutrients to cells; and anemia affects the ability of ischemic tissue to survive due to a deficit of oxygen. Moreover, Kosiak notes that the occurrence of an ulcer even after minimal pressures of short duration in a severely malnourished patient underscores the proposition that well nourished tissue is better capable of tolerating the destructive force of pressure when it occurs.

#### *Braden and Bergstrom Conceptual Schema*

The most widely known and studied conceptualization of pressure ulcer development in the contemporary literature is the Braden and Bergstrom conceptual schema for the etiology of pressure sores, now termed pressure ulcers (Braden & Bergstrom, 1987). This conceptual framework will provide the theoretical basis for this research. The Braden and Bergstrom framework supports the multivariate nature of the cause of pressure ulcer development and is derived from a synthesis of the empirical literature. In concert with the works of earlier researchers including Husain (1953) and



Kosiak (1959), Braden and Bergstrom postulate that intensity and duration of pressure and tissue tolerance are the two critical determinants for pressure ulcer development. According to Braden and Bergstrom, intensity of pressure describes the amount of pressure needed to cause capillary collapse. Once capillary collapse occurs, tissue anoxia and cell death ensue. Duration of pressure refers to the amount of time the patient is subjected to the forces of pressure. Exposure of the skin to intense pressure for short periods of time can lead to development of a pressure ulcer. Conversely, low intensity pressure for prolonged periods of time is just as capable of causing a pressure ulcer.

Braden and Bergstrom (1987) also posit that several clinical factors can have a direct influence on the intensity and duration of pressure experienced by the patient. Level of mobility, activity and sensory perception are identified as antecedents to prolonged and intense pressure in the Braden and Bergstrom model. Specifically, decreased levels of mobility (ability to change and control body positions) and decreased levels of activity (being bed-bound, chair-bound or non-ambulatory) have been identified in the model as factors that influence intensity and duration of pressure that, in turn, lead to pressure ulcer development. Moreover, consistent with Kosiak (1959), altered sensory perception in patients is also identified by Braden and Bergstrom as a risk factor for pressure ulcers. The inability to perceive or respond to discomfort places a patient at greater risk for the negative effects of pressure that is pressure ulcer formation.

Braden and Bergstrom (1987) also postulate that other clinical factors can also increase one's risk for pressure ulcer development by contributing directly to the ability of the body tissues to tolerate pressure. Tissue tolerance is defined by Braden and Bergstrom as "the ability of both the skin and its supporting structures to endure the

effects of pressure without adverse sequelae” (p.8). Intrinsic and extrinsic factors can lead to low tissue tolerance that in turn influence pressure ulcer development in response to lower pressure and shorter durations of time. Moisture, friction and shear are extrinsic factors that can alter the skin surface and increase one’s risk for pressure ulcer development (Braden & Bergstrom, 1987). Increased exposure of the skin’s surface to moisture (i.e. diaphoresis, incontinence or other drainage) alters skin integrity, decreases tissue tolerance and leads to the potential for pressure ulcer development. Friction alters the epidermal-dermal junction of the skin, thus decreasing tissue tolerance (Braden and Bergstrom). Lastly, shear, defined as the interaction of gravity and friction, exerts a force parallel to the skin which can destroy the vascular bed in the deep portion of the superficial fascia, with subsequent tissue ischemia, cell death and the potential for pressure ulcer development (Reichel, 1958).

Intrinsic factors are described as those factors internal to patients that can place them at risk for pressure ulcer development. Braden and Bergstrom (1987) theorize that these factors can alter the normal physiologic functioning of body systems and lead to a diminished ability of soft tissue to tolerate and endure pressure. These factors include advancing age (due to decreased elastin in the skin), poor nutrition (poor intake, hypoproteinemia, vitamin/mineral deficiencies) and low arteriolar pressure (diastolic blood pressure below 60 mmHg).

In summary, all theorists agree that risk factors for pressure ulcer development influence the intensity and duration of pressure and the ability of the tissues to tolerate pressure. These risk factors include mobility, activity, sensory perception, nutrition, friction, shear, moisture, age and low arteriolar pressure (Braden & Bergstrom, 1987;

Husain, 1953; Kosiak, 1959). These theorists postulate that the presence or absence of these risk factors influences the development of a pressure ulcer.

### Empirical Literature: Pressure Ulcer Risk Factors

Research supports the multifactorial etiology of pressure ulcer development in the critical care population. This section presents empirical support for the relationships between risk factors postulated by Braden and Bergstrom (1987) and pressure ulcer development in intensive care unit (ICU) patients, followed by a discussion of the relationships between risk factors gleaned from the empirical literature and pressure ulcer development in this population. Lastly, a summary of the current state of knowledge regarding pressure ulcer risk factors in the critical care population is presented, gaps in the empirical literature are identified and study hypotheses are outlined.

#### *Empirical Support: Braden and Bergstrom Risk Factors and Pressure Ulcer*

##### *Development*

The Braden Scale (Bergstrom, Braden, Laguzza & Holman, 1987) is the tool most frequently used to measure pressure ulcer risk in the U. S. in patients in a variety of health care settings, and it is based on the conceptual schema identified by Braden and Bergstrom (1987). The Braden scale measures the cumulative risk of pressure ulcer development for seven theoretical concepts: activity, mobility, sensory perception, moisture, friction, shear and nutrition. According to Braden and Bergstrom (1987), these Braden scale concepts are considered the clinical proxies for the two primary determinants of pressure ulcer development, that is, intensity and duration of pressure and tissue tolerance for pressure. Age and arteriolar pressure are two additional clinical factors posited in the Braden and Bergstrom model as risk factors for pressure ulcer

development. However, these concepts are not included as measures in the Braden scale. Thus, while Braden and Bergstrom postulate age and arteriolar pressure as intrinsic risks for pressure ulcers, they are not factors that contribute to cumulative risk in the Braden scale measure. Despite this, the relationships of age and arteriolar pressure to pressure ulcer development in critical care patients have been examined.

In the critical care population, six studies have been identified that tested the proposition that risk factors purported by Braden and Bergstrom (1987) and measured by the Braden scale are significantly related to pressure ulcer development. In one study, Jiricka and colleagues (1995) used the Braden scale to determine the relative contributions of risk factors to pressure ulcer development in a prospective study of 85 ICU patients. A statistically significant difference in total Braden scale scores, i.e., cumulative risk, was found between patients who developed pressure ulcers and those who did not ( $t(83) = 4.22, p < .01$ ). The patients who developed pressure ulcers had lower Braden scale scores indicating increased cumulative risk as compared to those who did not develop pressure ulcers. In this study, there were also statistically significant differences, that is lower scores in the sensory perception ( $t(35) = 3.98, p < 0.01$ ), moisture ( $t(35) = 4.3, p < 0.01$ ) and friction/shear ( $t(35) = 2.97, p = 0.004$ ) subscales in patients who developed pressure ulcers compared to patients who did not develop pressure ulcers. In addition, logistic regression analysis revealed that sensory perception and moisture risks were significant predictors of pressure ulcer development (OR = 2.01, (1.14-3.56); OR = 4.61, (1.70-12.52) respectively (no p values reported). Specifically, the risk for pressure ulcer development for patients with low moisture scores was more than four and one-half times higher compared to patients with higher moisture subscale scores.

Patients with sensory perception deficits were two times more likely to experience pressure ulcer development than patients with higher scores on the sensory perception subscale. Mobility, nutrition and activity risk factors were not related to pressure ulcer development in this study.

Carlson and colleagues (1999) used the Braden scale to examine the extent to which intrinsic and extrinsic risk factors predicted pressure ulcer development in a prospective study of 136 medical ICU patients. Findings revealed that a low mean total Braden score significantly predicted the risk of pressure ulcer development (Cox regression coefficient, -0.29,  $p = .046$ ). On further examination, decreased sensory perception was found to be the only risk factor significantly related to pressure ulcer development (Cox regression coefficient, -0.86,  $p=0.011$ ). Activity, moisture, mobility, nutrition and friction/shear were not found to be significant predictors of pressure ulcer development in this study. Carlson et al. found only slight variation in activity risk scores for patients with and without pressure ulcer development and concluded that, since most patients were confined to bed in the critical care setting, activity may not a useful predictor of pressure ulcer development in ICU patients.

Bours and colleagues (2001), in a secondary analysis of data, examined pressure ulcer prevalence, pressure ulcer risk factors and the use of pressure ulcer prevention interventions in 850 Dutch ICU patients. The relationships between cumulative risk as reflected by the total Braden scale score, individual risks, measured as Braden subscale scores, and pressure ulcer development were examined. Age in years was also measured as a continuous variable. In logistic regression analysis, the total Braden scale score, i.e. cumulative risk, (OR=1.24, (1.15-1.34),  $p < 0.05$ ) and age (OR= 2.42, (1.43-4.08,  $p <$

0.05) emerged as significant predictors of pressure ulcer development. Patients with lower total Braden scores had a 24% higher risk of developing a pressure ulcer compared to patients with higher total Braden scores. In addition, patients over 60 years of age were two times more likely to develop a pressure ulcer compared to patients in this sample under age 60. In a second logistic regression model that only included the Braden subscale scores, only moisture and mobility risks were found to be significant predictors of pressure ulcer development. Specifically, patients who developed pressure ulcers had lower mobility and moisture subscale scores compared to patients who did not develop pressure ulcers (OR 1.82, (1.41-2.34),  $p < 0.05$ ; OR 1.35, 95% CI 1.06-1.71,  $p < 0.05$  respectively). No empirical support was found for the predictive ability of activity, sensory perception, nutrition, and friction/shear risks for pressure ulcer development.

Fife and colleagues (2001), in a prospective study of risk factors for pressure ulcer development in 186 neurological ICU patients, found that a total Braden scale score of  $\leq 15$ , i.e. cumulative risk predicted pressure ulcer development. For the 117 patients in this sample with a total Braden scale score of 15 or less, the overall incidence of pressure ulcer development was 19.7%, whereas no incidence of pressure ulcer development was found for total Braden scores of 16 or higher. In multivariate analysis, the total Braden score and body mass index accounted for 36 % of the variance in pressure ulcer development ( $r^2 = .364$ ,  $p = .0002$ ,  $p = .0430$  respectively).

In a similar study, Wolverton and colleagues (2005), in a performance improvement study of nosocomial pressure ulcer rates in 422 ICU patients, found low total Braden scale scores in patients who developed pressure ulcers. Ninety three percent of patients in the sample who developed pressure ulcers had Braden scores of 16 or less and no patients

who had a Braden score of 19 or better developed a pressure ulcer. Data presented in this study was limited to descriptive statistics. No analysis of the predictive ability of Braden total score or subscales was undertaken in this study.

On the other hand, Pender and Frazier (2005) examined the relationship of 11 variables including the total Braden score and pressure ulcer development in a sample of 40 mechanically ventilated ICU patients and their study findings revealed no significant difference in Braden scale score between patients that developed a pressure ulcer and those that did not ( $t = -1.176, p = .260$ ). The authors concluded that in this sample of critically ill patients, the Braden scale was a poor discriminator of pressure ulcer risk. Sample size may have been a limitation in this study.

While six studies were identified that examined mobility, activity, sensory perception, moisture, nutrition, and friction/shear risks for pressure ulcer development in ICU patients as measured by the Braden scale, advancing age and low arteriolar pressure, not measured by the Braden scale, have been measured as continuous variables in empirical investigations. Age over 60 was found to be predictive of pressure ulcer development in the critical care population in three studies (Bours et al, 2001; Frankel, et al., 2007; Theaker et al, 2000) and in one study (Eachempati et al., 2001), age over 70 was found to be predictive of pressure ulcer development. Several studies however did not find advanced age to be predictive of pressure ulcer development in the critical care population (Batson, et al, 1993; Carlson et al. 1999; Jiricka et al, 1995; Wolverson et al., 2005). Only two studies measured arteriolar pressure in critically ill patients and found no relationship between low arteriolar pressure and pressure ulcer development (Batson, et al., 1993; Pender & Frazier, 2005).

In summary, in five of the six studies that measured risks for pressure ulcer development using the Braden scale, the total Braden score, i.e., cumulative risk was found to be a significant predictor of pressure ulcer development in ICU patients. However, only three studies examined the contributions of each of the individual risk factors to pressure ulcer development in the critical care population, and results revealed inconsistent findings. In two of the three studies, sensory perception was found to be a significant predictor of pressure ulcer development (Carlson et al., 1999; Jiricka et al., 1995). Two of the three studies found that moisture was a significant predictor of pressure ulcer development (Bours et al, 2001; Jiricka et al., 1995), and the predictive ability of mobility for pressure ulcer development was supported in only one study (Bours, 2001). Similarly, the contribution of friction/shear as a risk factor was supported in only one study (Jiricka, 1995). Activity and nutrition were not found to be significant risk factors for pressure ulcer development in any of these three studies.

Studies that examined the relationship between advancing age and pressure ulcer development also revealed inconsistent findings. This finding is surprising since the relationship between advancing age and pressure ulcer development is supported in non-critical care patients in the general hospital setting (Allman et al., 1995; Fischer et al., 2004; Russo et al., 2008). Perhaps in the critical care population, the burden of illness experienced by the patient plays a larger role in the development of pressure ulcers regardless of the age of the patient. More research surrounding the contribution of advanced age to pressure ulcer development in ICU patients is needed to determine if this is a significant risk factor in this population.



Moreover, the theoretically purported relationship between low arteriolar pressure and pressure ulcer development is not supported in the two studies that tested this proposition. However, there is empirical support for this relationship in other care settings. For example, in the long term care setting, Bergstrom and Braden(1992) found in a study of 200 newly admitted nursing home patients, that those patients who developed pressure ulcers had lower systolic and diastolic blood pressures ( $p < 0.001$ ). Similarly, Schubert (1991) found in a study of 130 hospitalized geriatric patients, that patients who developed pressure ulcers had significantly lower systolic blood pressure ( $130 \pm 17$  mmHg,  $p < 0.05$ ) compared to patients without pressure ulcers ( $140 \pm 20$  mmHg).

The small body of studies that have tested the Braden and Bergstrom (1987) risk assessment approach for pressure ulcer development using the Braden scale reveals inconsistent and inconclusive findings in the ICU population. While the sparse empirical literature lends preliminary support for the cumulative effect, i.e., total Braden scale score, of seven risk factors on pressure ulcer development as postulated by Braden and Bergstrom(1987), clearly more research is needed. The risk assessment approach used in these studies, i.e., cumulative risk as measured by the Braden scale, does not include the full range of factors that pose risks for pressure ulcer development in ICU patients. Thus, there is a need for a large body of rigorous investigations that explain the relative contributions of a more expansive range of risk factors, both individually and combined for pressure ulcer development in the hospitalized critical care population.

### *Empirically-Derived Risk Factors and Pressure Ulcer Development*

In addition to the risk factors for pressure ulcer development postulated by the Braden and Bergstrom (1987) framework, there is some empirical support for a more expansive pressure ulcer risk assessment approach for ICU patients and potential refinement of the Braden and Bergstrom framework. Four risk factors gleaned from the empirical literature have also been described as potential risks for pressure ulcer development in ICU patients and will be examined in this study. These risks include length of ICU admission, severity of illness, use of vasopressor agents, and comorbid conditions (i.e. diagnoses). Five studies were found that examined these empirically-derived risk factors in critical care patients.

Batson and colleagues (1993), in a pilot study of 50 critically ill patients examined risk factors for pressure ulcer development in an attempt to develop a valid tool aimed at weighting risk factors for pressure ulcer development. Comorbid conditions (diabetes), use of vasopressor agents (epinephrine and norepinephrine) and other risk factors presumed to contribute to pressure ulcer development were examined in this study. In multiple regression analysis, comorbid conditions (diabetes) ( $B = 14.9$ ,  $p < 0.001$ ), and use of vasopressor agents: norepinephrine ( $B = 14.9$ ,  $p < 0.001$ ), and epinephrine ( $B = 14.9$ ,  $p < 0.003$ ), emerged as significant predictors of pressure ulcer development. The researchers concluded that a replication of this study with a larger sample was needed and also recommended the development of a risk assessment tool specifically designed for critical care patients.

In a prospective study of risk factors for pressure ulcer development in 286 patients admitted into the ICU setting, Theaker and colleagues (2000) examined twenty-two

variables of which severity of illness as measured by the APACHE II scores, comorbid conditions (diabetes), length of ICU stay, and use of vasopressor agents (dobutamine, dopamine, epinephrine, norepinephrine) were included in the risk assessment model. In multivariate logistic analysis, five factors emerged as significant predictors including three empirically-derived risk factors that will be examined in this study. These include: use of vasopressor agents (norepinephrine) (OR = 8.11, (3.64-18.0),  $p < 0.001$ ), severity of illness OR = 3.4(1.4-7.92),  $p = 0.004$ , and length of stay  $\geq$  three days (OR= 2.76 (1.08-7.05),  $p = 0.034$ ). Notably, patients who received norepinephrine for greater than 60% of their ICU stay were eight times more likely to develop a pressure ulcer as compared to those patients who either did not receive norepinephrine or who received the infusion for shorter periods of time. Similarly, patients in this sample with a severity of illness represented by an APACHE II score of 13 or greater were three times more likely to develop a pressure ulcer as compared to patients with an APACHE II score of less than 13. In addition, patients with an ICU length of stay greater than three days were three times more likely to develop a pressure ulcer as compared to patients with an ICU length of stay less than three days. Comorbid conditions were not significant predictors of pressure ulcer development in this study. The researchers concluded that pressure ulcer development is the result of a multitude of factors, and proposed that current risk assessment tools do not capture the range of risk factors that confront the critically ill, resulting in potentially unreliable methods for determining patients at risk for pressure ulcer development in the critical care setting.

Bours and colleagues (2001) examined several variables presumed to be related to pressure ulcer development including length of ICU admission and comorbid conditions

(infection) in a secondary analysis of data of 850 ICU patients. In multivariate logistic analysis, length of ICU admission ( $OR=4.64(2.71-7.95, p < 0.05)$ ) and comorbid conditions (infection) ( $OR = 3.43 (1.61-7.32), p < 0.050$ ) were found to be significant predictors of pressure ulcer development. The authors concluded that pressure ulcers are the result of complex inter-relationships between multiple risk factors, making prediction of pressure ulcer risk in ICU patients difficult and in need of further investigation.

Eachempati and colleagues (2001) investigated risk factors for pressure ulcer development in a prospective study of 412 surgical intensive care patients. Several variables were measured in this study of which illness severity as measured by the APACHE III, vasopressor agents and intensive care unit length of stay were included. Of all patients in the study that developed pressure ulcers, 97% of the ulcers were noted in patients who remained in the ICU for seven days or longer. Study findings revealed that patients in the ICU for greater than seven days who were older ( $> 70$  years of age) had an 8% increased risk for pressure ulcer development, suggesting an important combined effect of age and length of ICU admission on pressure ulcer development in ICU patients. Severity of illness and vasopressor agents were not found to be significant predictors of pressure ulcer development in this study.

Finally, Frankel and colleagues (2007) examined risk factors for pressure ulcer development in a retrospective study of 820 patients admitted to a surgical ICU. Risk factors examined in this study included severity of illness as measured by APACHE II scores, comorbid conditions (diabetes, vascular disease, spinal cord injury) and vasopressor use, among other variables. In a univariate analysis, comorbid conditions (diabetes, vascular disease),  $p < 0.01$ ) and vasopressor use ( $p < 0.02$ ) were found to be

significantly related to pressure ulcer development. In multivariate analysis, comorbid conditions, that is a history of diabetes ( $OR = 2.7(1.1-6.4)$ ,  $p = 0.023$ ), was found to be an independent predictor of pressure ulcer development. Logistic regression analysis revealed that patients with diabetes were nearly three times more likely to develop a pressure ulcer, compared to other patients in this sample. Use of vasopressor agents and severity of illness did not predict pressure ulcer development in this study.

In summary, a small number of studies in the critical care population examined the effects of risk factors gleaned from the empirical literature on pressure ulcer development in intensive care unit patients. ICU length of stay was examined in three studies and was found to be a significant predictor for pressure ulcer development in all three studies (Bours et al., 2001; Eachempeti et al., 2001; Theaker et al. 2001). Severity of illness was examined in three studies and was found to be a significant predictor of pressure ulcer development in only one study (Theaker et al., 2000). Use of vasopressor agents was examined in four studies and was found to be predictive of pressure ulcer development in three out of four studies (Batson et al., 1993; Frankel et al., 2007; Theaker et al., 2000). Finally, comorbid conditions including diabetes, infection, and vascular disease were each found to be predictive of pressure ulcer development in ICU patients in studies that included one or more of these variables in the risk assessment model (Batson, et al., 1993; Bours et al., 2001; Frankel, et al., 2007).

These findings suggest that length of stay, use of vasopressor agents, comorbid conditions, and severity of illness may be important risk determinants of pressure ulcer development in intensive care unit patients. Clearly, is it difficult to draw any meaningful conclusions regarding a range of factors, both theoretically- and empirically-derived, that

pose a risk for pressure ulcer development in critical care patients from a small body of studies, and more research is needed to gain a clear understanding of the full range of factors that pose the greatest risk in the critical care population.

#### *Current State of Knowledge, Gaps and Limitations*

Theorists and findings from the empirical literature suggest that risks for pressure ulcer development in ICU patients are multifactorial. While Braden and Bergstrom (1987) contend that mobility, activity, sensory perception, moisture, friction/shear, advancing age and low arteriolar pressure are important risks for pressure ulcer development, findings from the empirical literature indicate that these factors may not represent the full spectrum of pressure ulcer risks in critical care patients.

The literature, while small provides preliminary evidence that several risk factors postulated by Braden and Bergstrom (1987) pose a significant risk for pressure ulcer development in critical care patients. Sensory perception, moisture, friction/shear, and age were found to be significantly related to pressure ulcer development in ICU patients, but these relationships were not consistent in all studies that tested these relationships. Clearly, the sparse body of work in this area yields limited knowledge regarding the contributions of these risk factors to pressure ulcer development in critical care patients, and further empirical exploration is needed.

On the other hand, this small body of literature provides preliminary evidence that other risk factors for pressure ulcer development postulated by Braden and Bergstrom (1987) may not be relevant in ICU patients. In three studies that examined mobility and activity risks, only one study found mobility to be a significant predictor, while activity did not predict pressure ulcer development in ICU patients in any study. It is plausible

that, among ICU patients, mobility and activity are non-discriminating concepts since all critically ill patients experience diminished or absent levels of both activity and mobility due to the nature of critical illness and treatment interventions that typically render ICU patients immobile and bed-bound. This notion is supported by Carlson and colleagues (1999) who found that activity was not a useful predictor of pressure ulcer development in ICU patients as only small variations in Braden activity subscale scores were evident among all subjects. Similarly, deLaat and colleagues (2006) noted that activity and mobility do not discriminate between critically ill patients that are at risk for pressure ulcer development and those that are not at risk. Variables, however that capture the typical levels of movement experienced by ICU patients were measured in one study and found to be predictive of pressure ulcer development. In Batson's and colleagues' (1993) investigation, the variables "restricted movement" ( $B=7.7$ ,  $p < .001$ ) and "too unstable to turn" ( $B= 17.4$ ,  $p < .001$ ) predicted pressure ulcer development in a sample of ICU patients. Findings from this study underscore the premise that mobility and activity risks postulated by Braden and Bergstrom and measured in the Braden scale may not discriminate risk in the critical care population. Since the body of work that has examined mobility and activity as risk factors for ICU patients is sparse, more research is needed in this area.

Similarly, the emergence of both nutrition and low arteriolar pressure as non-significant risks for pressure ulcer development in all studies that measured these variables is noteworthy. It is plausible that the Braden nutritional subscale does not fully capture the spectrum of nutritional status in critically ill patients. For example, Eachempati and colleagues(2001) operationalized nutrition risk as "the number of days

without nutrition” in their sample of ICU patients, and this variable significantly predicted pressure ulcer development. More evidence is needed to determine the ability of the Braden nutrition subscale to capture nutritional risk for pressure ulcer development in critically ill adults. In addition, a plausible explanation for the lack of empirical support for the relationship between low arteriolar blood pressure and pressure ulcer development may be that constant monitoring of blood pressure through the use of arterial lines and automatic cuff pressure in intensive care settings result in quicker implementation of interventions aimed at elevating blood pressure. The limited number of studies that have measured the effects of these risk factors on pressure ulcer development in the critical care population precludes the ability to draw conclusions about these relationships and more research in this area is needed.

The studies that examined the relationship between empirically-derived risk factors gleaned from the empirical literature, while small, provides preliminary evidence that length of ICU stay, use of vasopressor agents, comorbid conditions, and severity of illness may also pose significant risks for pressure ulcer development in critical care patients. Although these factors were found to be significantly related to pressure ulcer development in ICU patients, these findings were not consistent in all studies that tested these relationships. Clearly, the sparse body of work in this area yields limited knowledge regarding the contributions of these risk factors to pressure ulcer development in ICU patients, and further empirical exploration is warranted.

In summary, there is a dearth of studies that have tested the proposition that intrinsic and extrinsic clinical factors put ICU patients at increased risk for pressure ulcers. The Braden and Bergstrom (1987) framework may provide a theoretical basis for



explaining pressure ulcer development in ICU patients, but the risks for pressure ulcer development in this framework may not represent the full range of risks in critical care patients. Thus, there is a need for studies designed to evaluate the extent to which a more expansive range of risk factors gleaned from the theoretical and empirical literature predict pressure development in ICU patients (deLaat et al, 2006; Doughty, 2008, Keller et al., 2008). Moreover, the identification of risk factors derived from both the Braden and Bergstrom conceptual schema and the empirical literature can serve as the basis for an appropriate risk assessment approach for this population.

The purpose of this study is to examine the extent to which risk factors postulated by Braden and Bergstrom (1987) and those factors gleaned from the empirical literature predict pressure ulcer development in a sample of ICU patients.

#### Theoretical Rationale

Theory postulates pressure ulcer development to be a multifactorial phenomenon. The two critical factors that influence pressure ulcer development are the intensity and duration of pressure and the tissue's ability to tolerate pressure (Braden & Bergstrom, 1987, Husain, 1953, Kosiak, 1959).

Factors purported by theory that contribute to prolonged and intense pressure and lead to pressure ulcer development include the individual's degree of mobility, activity and sensory perception. Theory posits that diminished mobility, diminished activity and diminished sensory perception all influence the intensity and duration of pressure experienced by an individual and can lead to pressure ulcer development.

Theoretically purported factors that influence tissue tolerance are categorized as extrinsic and intrinsic factors (Braden & Bergstrom, 1987). Exposure to extrinsic factors

such as moisture, friction and shear diminish the ability of body tissues to tolerate pressure and lead to pressure ulcer development. Intrinsic factors such as nutrition, advancing age and low arteriolar pressure adversely affect the architecture of the skin and body tissues, decreasing tissue tolerance, thus leading to pressure ulcer development (Braden & Bergstrom, 1987). Theory posits that the presence of moisture, friction and shear each can influence tissue tolerance and lead to pressure ulcer development. In addition, theory posits that decreased nutrition, advancing age and low arteriolar pressure each can independently influence tissue tolerance and lead to pressure ulcer development.

The relationships between length of ICU admission, severity of illness, vasopressor administration and comorbid conditions and pressure ulcer development in critical care patients are not theoretically defined as risk factors for pressure ulcer development but are supported in varying degrees in the empirical literature. Empirical studies purport that length of ICU admission, vasopressor administration, severity of illness and comorbid conditions are each positively related to pressure ulcer development.

### Hypotheses

The following hypotheses were investigated in adult critical care patients admitted into the intensive care setting:

1. Total Braden scale score is inversely related to pressure ulcer development in adult critical care patients.
2. Mobility is inversely related to pressure ulcer development in adult critical care patients.
3. Activity is inversely related to pressure ulcer development in adult critical care patients.

4. Sensory perception is inversely related to pressure ulcer development in adult critical care patients.
5. Moisture is positively related to pressure ulcer development in adult critical care patients.
6. Friction/Shear is positively related to pressure ulcer development in adult critical care patients.
7. Nutrition is inversely related to pressure ulcer development in adult critical care patients.
8. Age is positively related to pressure ulcer development in adult critical care patients.
9. Arteriolar pressure is inversely related to pressure ulcer development in adult critical care patients.
10. Length of intensive care unit stay is positively related to pressure ulcer development in adult critical care patients.
11. Severity of illness is positively related to pressure ulcer development in adult critical care patients.
12. Vasopressor administration is positively related to pressure ulcer development in adult critical care patients.
13. Comorbid conditions are positively related to pressure ulcer development in adult critical care patients.
14. There will be significant combined effects in terms of variance accounted for, of theoretically- and empirically-derived risk factors on pressure ulcer development in adult critical care patients.

Figure 1  
Relationships between the Theoretically-Derived Risk Factors and Pressure Ulcer Development

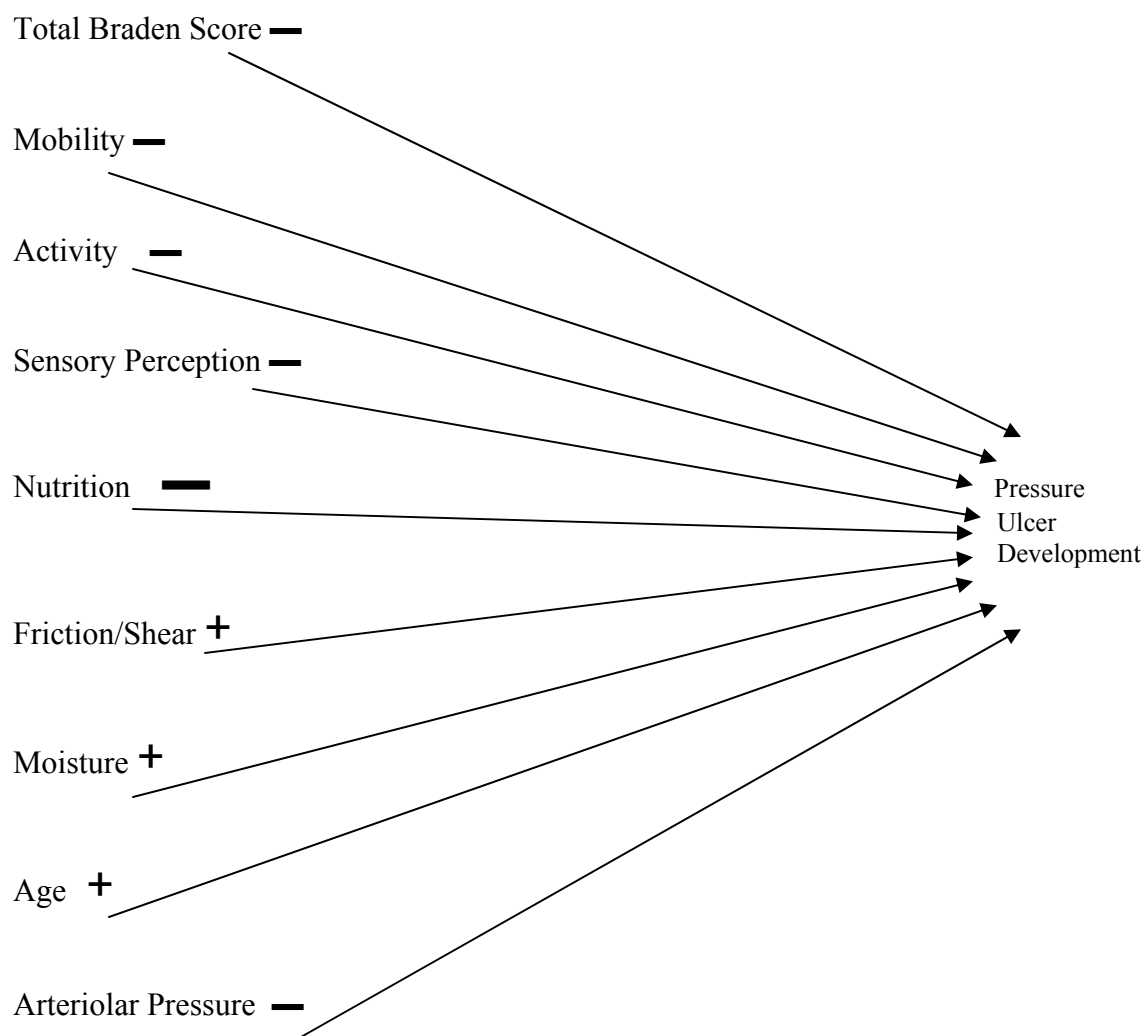
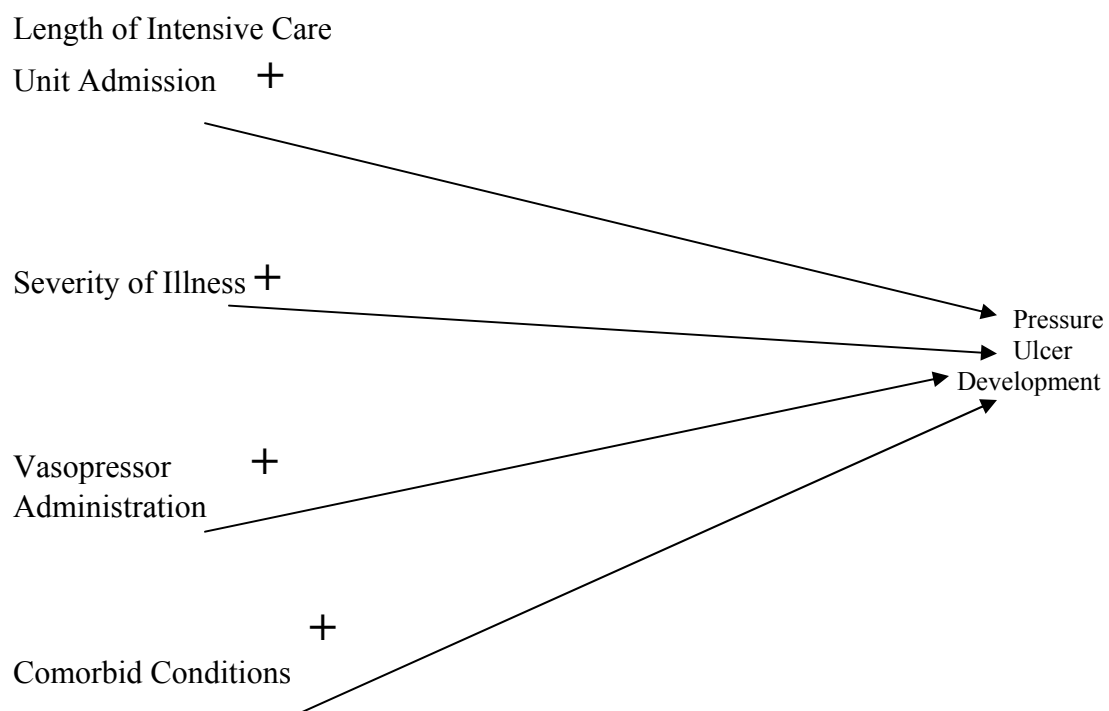


Figure 2  
Relationships between Empirically-Derived Risk Factors and Pressure Ulcer Development



## CHAPTER 3

### Methods

This chapter describes the research design inclusive of the research setting, sample and sampling methodology, the instruments, the data collection procedures and analysis for this study. A descriptive, correlational design was used in this study to investigate the relationships between theoretically- and empirically-derived risk factors and pressure ulcer development in order to identify significant predictors of pressure ulcer development in adult patients admitted to the intensive care unit (ICU) setting. A retrospective analysis using existing patient data was conducted.

### Research Setting

The setting for this study was a 12 bed medical/surgical ICU in a 500 bed suburban Magnet teaching hospital located in northeastern New Jersey. The medical/surgical ICU setting was selected to control for an ICU confounding effect that multiple ICU types could potentially pose. This ICU admits approximately 700 patients per year with an average length of ICU admission of three days. Admission to this unit follows the criteria outlined by the Society for Critical Care Medicine (1999) and is based on the medical decision of the attending critical care physician (intensivist) and critical care fellow. Major diagnoses for patients admitted into the medical/surgical intensive care unit typically include septic shock, respiratory failure, multisystem organ failure, postoperative neurosurgery, vascular surgery, and hemodynamic instability.

### Sampling Methods

All adult patients admitted into the medical/surgical ICU during the eight-month time period from October 2008 through May 2009 that met the inclusion criteria were

included in this sample. Inclusion criteria for this study were as follows: any adult patient eighteen years of age or older admitted into the medical/surgical ICU for greater than 24 hours duration. Exclusion criteria included: age of less than eighteen upon admission to the ICU, or a preexisting pressure ulcer of any stage. Patients admitted into the ICU for less than 24 hours were excluded as insufficient data precluded measurement of the study variables. Patients with an existing pressure ulcer were excluded as this study sought to identify factors that lead to pressure ulcer development in this population.

Power analysis for correlational and regression analysis were calculated to determine the appropriate sample size. For correlational analysis, using a moderate effect size ( $r = .30$ ) based on the literature (Fife et al., 2001), a sample size of 85 was needed to obtain a power of .80 at a .05 significance level (Cohen, 1988). Using a moderate effect size ( $f^2 = .15$ ), based on a review of the literature (Batson et al., 1993; Fife et al., 2001) and a significance level of .05 and 24 predictor variables, a minimum sample size of 169 was needed to obtain a power of .80 (Cohen, 1988) for regression analysis. The total sample size for this study was 347, which exceeded the minimum number of patients required to achieve statistical power for correlational and regression analyses.

Of the 579 patient admissions into the medical/surgical ICU from October 2008 through May 2009, 347 patients met the inclusion criteria and were included in the final sample. Two hundred and thirty two patients were eliminated from the sample due to the following: age under 18 ( $n=2$ ), admission of less than 24 hours ( $n=190$ ), admission with an existing pressure ulcer ( $n=38$ ) or incomplete data ( $n=2$ ).

The age range for patients in this sample was 20 to 97 years ( $M = 68.69$ ;  $SD = 17.1$ ). Forty nine percent were male and fifty-one percent female. Ethnic make-up included

Caucasian (73.5%), Black/African American (14%), Asian/Pacific Islander (7.5%), and Hispanic (5%) and other (.3%), closely approximating the ethnic make-up of the population in northeastern New Jersey (U.S. Census Bureau, 2005-2007).

The majority of patients were admitted into the intensive care for the following diagnoses: respiratory failure/distress (20.7%), sepsis/septic shock (17.3%), neuro-medicine(15%), general medicine (12.7%), neuro-surgery (6.9%), GI bleed (6.9%), GI surgery (6.6%), vascular surgery (4.9%), cardiac-medicine(3.5%), cardiac arrest (1.7%), other surgical procedures (4%). At discharge from the intensive care unit, 69.5% were transferred to a medical/surgical unit, 16% were transferred to a progressive care unit, 10 % expired, and the remaining 4% were discharged or transferred to another facility.

Demographic characteristics of the sample can be found in Table 1.

Table 1

Frequency Distribution of Selected Demographic Subject Variables

Variable	n	Percentage
<b>Gender</b>		
Male	171	49%
Female	176	51%
<b>Race</b>		
Caucasian	255	73.5%
Black/African American	48	13.8%
Asian/Pacific Islander	26	7.5%
Hispanic	17	4.9%
Other	1	.3%
<b>ICU Admitting Diagnosis</b>		
Respiratory Arrest/Distress	72	20.7 %
Sepsis/Septic Shock	60	17.3 %
Neuro-medicine	52	15.0 %
General Medicine	44	12.7 %
GI Bleed	24	6.9%
Neuro-surgery	24	6.9%
GI surgery	23	6.6%
Vascular surgery	17	4.9%
Other-surgery	13	3.7%
Cardiac-medicine	12	3.5%



Cardiac arrest	6	1.7%
<b>Disposition at Discharge</b>		
Medical/Surgical Unit	241	69.5%
Progressive Care Unit	55	15.9%
Dead	36	10.4%
Other Facility(Rehab, SNF, other)	12	3.5%
Discharge	3	.9%

### Instruments

#### Pressure Ulcer Development

During the patient's ICU admission, pressure ulcer assessment (if present) is recorded every 12 hours by the medical/surgical ICU staff RN in a computerized patient record termed Quantitative Sentinel™ (QS). This documentation consists of pressure ulcer location, stage, size and assessment of the ulcer appearance. In 2008, all staff RNs in this medical center received a mandatory education program on pressure ulcer staging developed by the PI in order to ensure that documentation in the patient record accurately reflects the patient assessment. In addition, RN staff participates in on-going pressure ulcer education programs in order to maintain competence in pressure ulcer assessment. For this study, pressure ulcer occurrence as documented in the patient record was recorded as either present or absent after admission into the ICU.

#### Braden Scale for Predicting Pressure Ulcer Risk

The Braden Scale for Predicting Pressure Ulcer Risk is used in the medical/surgical ICU to assess patients' pressure ulcer risk (Bergstrom et al., 1987). The scale is composed of six subscales that measure the theoretically-derived concepts of activity, mobility, sensory perception, moisture, nutrition, and friction/shear. Subscale scores range from 1 to 4, with the exception of the friction/shear subscale which ranges from 1 to 3. The activity subscale contains four levels of risk and includes the following

assessment parameters: 1= bedfast 2= chairfast 3= walks occasionally and 4= walks frequently. The mobility subscale contains four levels of risk and includes the following assessment parameters: 1= completely immobile 2= very limited 3= slightly limited and 4= no limitations. The sensory perception subscale also contains four levels of risk and includes the following assessment parameters: 1= completely limited 2= very limited 3= slightly limited and 4= no impairment. The moisture subscale contains four levels of risk and includes the following assessment parameters: 1= constantly moist 2= moist 3= occasionally moist and 4= rarely moist. The nutrition subscale contains four levels of risk and includes the following assessment parameters: 1= very poor 2= probably inadequate 3= adequate and 4= excellent and the friction/shear subscale has three levels of risk and includes the following assessment parameters: 1= problem 2= potential problem and 3= no apparent problem. All subscale levels contain narrative descriptors that define these assessment parameters in order to assist the practitioner in the appropriate selection of risk level. Pressure ulcer risk is based on a summated score ranging from 6 to 23. Lower scores on the Braden scale indicate increased risk of pressure ulcer development. According to Bergstrom and Braden (2002), a cut-off score of 18 or less on the Braden Scale establishes pressure ulcer risk. This is the cut-off score currently recommended for clinical practice.

The Braden scale is the most widely used tool in clinical practice in the United States across the care continuum and has been subject to the most extensive psychometric testing of all the pressure ulcer risk tools currently in use (Bolton, 2007; Seonsook et al., 2003). Additionally, the Braden scale is one of the two scales recommended by the Agency for Health Care Policy and Research (AHCPR) (1992), now the Agency for

Healthcare Quality and Research and the Wound, Ostomy and Continence Nursing Society (2003) for use in measuring pressure ulcer risk.

Three initial reliability studies (Bergstrom et al., 1987) for the Braden scale were conducted on patients in skilled nursing facilities. Interrater reliability coefficients in these three studies were reported to range from  $r = .83$  to  $r = .99$ ,  $p < .001$ . In the intensive care population, interrater reliability of RN staff using the Braden scale was calculated at  $r = .89$   $p < .001$  (Bergstrom et al., 1987). In another study in the intensive care population, high interrater reliability was found using percentage of agreement, reported to range between 88% and 93% (Jiricka et. al., 1995).

Predictive validity was measured in the initial two validity studies of the Braden scale, using sensitivity and specificity ratings (Bergstrom et al., 1987). Studies were conducted simultaneously on two medical surgical units with samples of 100 patients admitted to each unit. The scale was found to be 100% sensitive in both units but the specificity varied from 64% to 90%. In the intensive care setting, Bergstrom et al. (1987) found the predictive validity of the scale to be 83% sensitive and 64% specific, similar to values obtained in one of their initial studies. The authors reported that the predictive value of a negative (PVN) test using a cut-off score of 16 was 85% while the predictive value of a positive test (PVP) was 61%. In further studies of the Braden scale in the critical care population, Jiricka and colleagues (1995) found that at an initial Braden score of 11, the scale was 75% sensitive and 65% specific with a PVP of 73% and PVN of 67%. In studies, comparing the Braden scale to other risk scales currently available, the Braden scale performed similarly or superior to the other risk scales across various care settings (Bolton, 2007; Pancorbo-Hildago et al., 2006).

Braden scale and subscale scores are recorded daily in the computerized medical record (QS) for each patient in the medical/surgical ICU setting. All RN staff are trained on the proper use and scoring of the Braden scale in order to ensure that the risk level assessed accurately reflects the patient condition. This education is provided to staff members on an on-going basis and in 2008, all RN staff were required by the medical center to complete a mandatory computer based self learning module developed by the PI on the proper use of the Braden scale. For this study, the patient's total Braden scale score and Braden subscale scores documented in the first 24 hours of admission to the ICU were used in analyses.

#### *Age*

For every patient in the ICU, age was recorded on the patients' medical record as their chronological age when they are admitted into the intensive care unit setting.

#### *Arteriolar Pressure*

Arteriolar pressure was measured on all patients in the ICU using invasive arterial monitoring and/or non-invasive automatic cuff blood pressures and is recorded at a minimum of every two hours in the computerized patient record. For patients with arterial lines in this medical/surgical ICU, invasive (direct) blood pressure readings are correlated with cuff blood pressures every 12 hours and as necessary in order to validate consistency among readings. In addition, the Square Waveform test is performed every shift in order to confirm that the arterial line accurately reflects the patient's arterial pressure. Accuracy is also confirmed by leveling the transducer of the arterial line at the patient's phlebostatic axis (level of the right atrium) at insertion and at every patient position change thereafter (Weigard & Carlson, 2005). The Marquette Solar 8000 monitoring

system is used to record and display both arterial and automatic cuff blood pressures and mean arterial blood pressures. Performance verification for the monitoring system is conducted on an annual basis by the biomedical engineering department in accordance with the manufacturer guidelines and the National Institute of Standards criteria (Marquette, 1992). For this study, the total number of hours during the first 48 hours of the ICU admission that the patient's mean arterial pressure was below 60, and/or systolic blood pressure was below 90 mmHg and/or the diastolic blood pressure was below 60 mmHg was computed separately.

#### *Intensive Care Unit Length of Stay*

Intensive Care Unit length of stay was calculated as the total number of hours the patient spent in the ICU.

#### *Severity of Illness*

##### *APACHE II*

The APACHE II scale was used as a measure of severity of illness. The APACHE II scale is a prognostic scoring system that provides a measure of the severity of illness (Knaus et al., 1985). The APACHE II scale score is computed based points assigned in the three areas: 1) acute physiological parameters (temperature, mean arterial pressure, heart rate, respiratory rate, oxygenation, arterial pH, serum sodium, serum potassium, serum creatinine, hematocrit, white blood count, and Glasgow Coma Scale score), 2) chronic health conditions including a history of severe organ insufficiency including liver disease, cardiovascular disease, chronic renal disease, chronic respiratory disease or immuno-compromised states and 3) age. The score is computed in the first 24 hours of the intensive care unit admission. Total score ranges from 0 to 71 with higher scores

indicating more severe illness and higher risk of mortality. An interrater reliability coefficient of .90 has been cited for the APACHE tool used in medical-surgical intensive care units (Kho et al. 2007). According to Knaus and colleagues (1985), at a .50 predicted risk of death, the APACHE II yielded a sensitivity of 47%, specificity of 94.4% and an 85.5% correct classification rate.

Since the APACHE II score is not routinely computed in this intensive care unit, the APACHE II score was calculated by the principal investigator (PI) at the time of medical record data abstraction.

#### *Vasopressor agent*

Use of vasopressor agents was operationalized as the administration of one or more of the following vasopressor agents as documented in patient's medical record during the ICU admission: norepinephrine, epinephrine, phenylephrine, vasopressin or dopamine. In addition, the total length of time (in hours) for each vasopressor agent administered was abstracted from the patient's medical record.

#### *Comorbidity*

Comorbidity was assessed as the presence or the absence (yes/no) of current or past medical history of any of the following conditions as noted in the patient's medical record: diabetes mellitus, vascular disease (cardiovascular and/or peripheral vascular), infection/sepsis.

#### *Demographic Data*

The following demographic data and patient characteristics were abstracted to describe the study sample: ethnicity, gender, admitting ICU diagnosis, pressure ulcer

stage/anatomic location at the time of discharge from the ICU if present and the number of hours into the admission the pressure ulcer developed.

### Procedure for Data Collection and Analysis

This study received exempt status from the Institutional Review Boards (IRB) of Englewood Hospital and Medical Center, Englewood, New Jersey and Rutgers, The State University of New Jersey (Appendices A and B).

Data were abstracted from the hospital's existing Eclipsys™ and Quantitative Sentinel™ (QS), computerized documentation systems and other computerized portions of the medical record. Quantitative Sentinel provided the patient data for the following variables under study: pressure ulcer presence or absence, admission Braden scale score, admission Braden subscale scores (activity, mobility, sensory perception, friction/shear, moisture, nutrition), age, mean arterial pressure (MAP), systolic blood pressure, diastolic blood pressure, length of ICU admission and use and duration of vasopressor agent administration. APACHE II scores were computed by the PI at the time of data abstraction, and presence or absence of comorbid conditions (diabetes mellitus, vascular disease, infection/sepsis) was abstracted from the computerized version of the patient's medical record.

### *Data Abstraction Record*

Data were recorded on a data abstraction form developed for this study (Appendix C). This form is two-sided. On side one all measurements of the study variables were recorded in addition to other aforementioned descriptive characteristics of the study sample. Side two contained the operational definitions of all variables. Data for the APACHE II scale were collected on a separate data abstraction record (Appendix D). On

side one of this form all APACHE parameters were recorded. Side two provided definitions of selected parameters.

The data abstraction record was stripped of all identifying information including name, birth date, addresses, phone numbers, medical record number, insurance information and any other information that could identify the patient. No identifiers were used to link subjects to abstracted data.

#### Human Subjects Protection

This study posed no risk to human subjects as variables abstracted reflect care parameters utilized and recorded during routine patient care. All patient information recorded on the data abstraction record was de-identified in order to ensure patient anonymity.

Data collected from this study was entered in a Statistical Package for the Social Sciences (SPSS) Version 16.0 computer database. All subject data was de-identified in this database. All files were password protected and accessible only by the PI. Data files were backed up on a CD and stored in a locked desk in the researcher's office. The PI has sole access to this locked data.

Data collected from this study that is either published or presented will be reported as aggregate data only, and no subject identifiers will be used. Computer files and data collection tools will be discarded after completion of the study and the three year IRB data maintenance timeframe.



## CHAPTER 4

### Analysis of the Data

The purpose of this study was to identify risk factors derived from both the theoretical and empirical literature that best predict pressure ulcer development in adult critical care patients. The pressure ulcer risk factors under investigation included the total Braden scale score representing cumulative risk, mobility, activity, sensory perception, moisture, friction/shear, nutrition, age, arteriolar pressure, length of ICU admission, severity of illness, vasopressor administration and comorbid conditions. Data were collected from 347 adult patients admitted into the medical/surgical intensive care unit of a Magnet teaching hospital in northeastern New Jersey. This chapter presents findings from the analysis of the data.

### Statistical Description of the Variables

#### *Dependent Variable*

##### *Pressure Ulcer*

Of the 347 patients in the sample, 65 patients developed a pressure ulcer, representing 18.7% of the total sample. Of these pressure ulcers, 31% were identified at discharge from the ICU as Stage I; 35% Stage II; 1.5% Stage III; 1.5% Stage IV; 23% suspected Deep Tissue Injury and 8% Unstageable. Anatomical locations included sacrum (58%), buttocks (34%), heels (5%), and other anatomic locations (3%).

#### *Independent Variables*

##### *Braden Scale*

The mean score for the total Braden scale for this sample was 14.28 (SD = 2.68, range 6-23). The mean score on the sensory perception subscale was 2.85 (SD = .939,

range 1-4). The mean score on the moisture subscale was 3.4 (SD = .756, range 1-4). The mean score on the activity subscale was 1.08 (SD = .442, range 1-4). The mean score on the mobility subscale was 2.53 (SD = .829, range 1-4). On the nutrition subscale, the mean score was 2.29 (SD = .655, range 1-4) and on the friction/shear subscale the mean score was 2.10 (SD = .655, range 1-3).

#### *Age*

The mean age of the sample was 68.9 years (SD = 17.51, range 20-97).

#### *Arteriolar pressure*

The mean number of hours that the mean arterial pressure (MAP) was below 60 was 2.41 hours (SD = 4.96, range 0-38). The mean number of hours that the systolic blood pressure was below 90 was 3.65 hours (SD = 6.74, range 0-48). The mean number of hours that the diastolic blood pressure was below 60 was 23.1 hours (SD = 15.39, range 0-48).

#### *Length of ICU admission*

The mean length of stay for the ICU admission was 118.84 hours (SD = 155.58, range 24-1104).

#### *Severity of Illness*

The mean score on the APACHE II scale was 17.26 (SD = 7.72, range 0-39).

#### *Vasopressor Administration*

The mean number of hours of norepinephrine administration during the ICU stay was 13.8 hours (SD = 50.5, range 0 – 48). The mean number of hours of vasopressin administration during the ICU stay was 3.76 (SD = 22.25, range 0-305). The mean number of hours of dopamine administration during the ICU stay was 1.7 (SD = 9.44,

range 0-104). The mean number of hours of epinephrine administration during the ICU stay was 0.29 (SD = 3.69, range 0 – 56.48) and the mean number of hours of phenylephrine administration during the ICU stay was 1.29 (SD = 8.80, range 0-137,).

### *Comorbid Conditions*

The following comorbid conditions were investigated: vascular disease (cardiovascular and peripheral vascular), diabetes mellitus and infection. Fifty-five percent (191/347) had no previous history of vascular disease. By etiology, 91% (317/347) had no history of peripheral vascular disease, while 9 % (30/347) did have a past history. Forty-two percent of patients had a past history of cardiovascular disease; 28% (97/347) had a past medical history of diabetes mellitus; and 35% (120/347) of the sample was found to have had infection. These findings are summarized in Table 2.

**Table 2**  
**Descriptive Statistics of Study Variables**

<b>Dependent Variable</b>	<b>n</b>	<b>Percentage</b>
Pressure Ulcer	65	18.7%
Pressure Ulcer Stage		
Stage I	20	30.8%
Stage II	23	35.4%
Stage III	1	1.5%
Stage IV	1	1.5%
DTI	15	23.1%
Unstageable	5	7.7%
Pressure Ulcer Location		
Sacrum	38	58.5%
Buttocks	22	33.8%
Heels	3	4.6%
Other	2	3.1%

<b>Independent Variables (n=347)</b>	<b>M(SD)</b>	<b>Range</b>
Total Braden Score	14.28(2.68)	6-23
• Sensory Perception Subscale	2.85(.936)	1-4

• Moisture Subscale	3.40(.756)	1-4
• Activity Subscale	1.08(.442)	1-4
• Mobility Subscale	2.53(.829)	1-4
• Nutrition Subscale	2.29(.655)	1-4
• Friction/Shear Subscale	2.10(.473)	1-3
Age	68.69(17.51)	20-97
Arteriolar Pressure (hours)		
• MAP < 60	2.41(4.96)	0-38
• Systolic < 90	3.65(6.74)	0-48
• Diastolic < 60	23.12(15.39)	0-48
Length of ICU admission (hours)	118.84(155.58)	24-1108
Severity of Illness (APACHE score)	17.268(7.72)	0-39

<b>Vasopressor Administration (Hours)</b>	<b>M(SD)</b>	<b>Range</b>
Norepinephrine	13.87(50.05)	0-481
Epinephrine	0.29 (3.69)	0-56
Phenylephrine	1.29 (8.80)	0-137
Dopamine	1.70 (9.44)	0-104
Vasopressin	3.76 (22.24)	0-305

<b>Comorbid Conditions</b>	<b>n</b>	<b>Percentage</b>
• Vascular Disease	156	45%
PVD	30	9 %
Cardiovascular	145	42%
• Diabetes	97	28%
• Infection	120	35%

### Psychometric Properties of the Instrument

Reliability for the Braden Scale is best measured using a measure of interrater reliability. Due to the retrospective design of this study, the ability to measure interrater reliability was not possible and was recognized as a limitation of this study design.

Predictive validity focuses on the relationship between current performance on a measure and future performance on some related variable (Waltz et al., 2004). Since the

purpose of the Braden scale is to predict pressure ulcer risk, predictive validity is both essential and appropriate. Predictive validity was measured using sensitivity and specificity ratings in addition to negative and positive predictive values. The sensitivity measure refers to the number of patients in the sample that were correctly identified as being at risk for pressure ulcers and developed a pressure ulcer. The specificity measure identifies those patients that were correctly identified as not at risk for pressure ulcer development and remained pressure ulcer free. The positive predictive value describes the percentage or probability of patients that developed pressure ulcers who were identified as being at risk for pressure ulcer development. The predictive value of a negative rating predicted to be at risk for pressure describes how accurately the Braden scale predicted which patients would remain pressure ulcer free. For this sample, the sensitivity was 100%, specificity was 7%, predictive value of a positive test was 20% and the predictive value of a negative test was 100% at a cut-off score of 18.

### Hypotheses

Hypotheses 1-13 were tested using the Pearson product moment correlation coefficient. Alternative methods available for measuring relationships when using dichotomous, ranked or continuous level variables include PHI, point-biserial and Spearman rho. However, according to Munro (2005), these methods are considered shortcut versions of  $r$  and will provide the same result as Pearson  $r$  in most circumstances. Therefore, Pearson  $r$  was selected to measure the relationships between the variables under study. Hypothesis 14 was tested using direct logistic regression. In logistic regression, no assumptions regarding distributions of the independent or predictor variables need to met, therefore predictor variables do not need to be normally

distributed, linearly related, exhibit homoscedasticity or have equal variance within each group (Tabachnick & Fidell, 2001). As such, no data transformations were attempted on skewed variables. SPSS version 16.0 for Windows was used for the statistical analyses.

### *Hypothesis 1*

Hypothesis 1 stated that there would be an inverse relationship between total Braden scale score and pressure ulcer development in adult critical care patients. The Pearson product moment correlation testing this relationship was  $r = -.276$ ,  $p = .000$ . In direct logistic regression, the total Braden scale score was not found to be a significant predictor of pressure ulcer development in this sample of adult critical care patients ( $B = .102$ , 95% CI = .692-1.774,  $p = .670$ ). Based on this finding, Hypothesis 1 was supported only in bivariate analysis.

### *Hypothesis 2*

Hypothesis 2 stated that mobility would be inversely related to pressure ulcer development in adult critical care patients. The Pearson product moment correlation for this relationship was  $r = -.275$ ,  $p = .000$ . In direct logistic regression, mobility was found to be a significant predictor of pressure ulcer development in adult critical care patients ( $B = -.823$ , 95% CI = .201-.959, OR = .439,  $p = .039$ ). Based on these findings, Hypothesis 2 was supported.

### *Hypothesis 3*

Hypothesis 3 stated that activity would be inversely related to pressure ulcer development in adult critical care patients. The Pearson product moment correlation for this relationship was  $r = -.088$ ,  $p = .103$ . Based on this finding, Hypothesis 3 was not supported.

#### *Hypothesis 4*

Hypothesis 4 stated that sensory perception would be inversely related to pressure ulcer development in adult critical care patients. The Pearson product moment correlation for this relationship was  $r = -.208$ ,  $p = .000$ . In direct logistic regression, sensory perception was not found to be a significant predictor of pressure ulcer development in adult critical care patients ( $B = .035$ , 95% CI = .496- 2.162,  $p = .926$ ). Hypothesis 4 was supported only in bivariate analysis.

#### *Hypothesis 5*

Hypothesis 5 stated that there would be a positive relationship between moisture and pressure ulcer development in adult critical care patients. Moisture was reverse coded in order to provide for easier interpretation of this relationship. The Pearson product moment correlation was  $r = .104$ ,  $p = .054$ . Based on this finding, Hypothesis 5 was not supported.

#### *Hypothesis 6*

Hypothesis 6 stated that friction/shear would be positively related to pressure ulcer development in adult critical care patients. The friction/shear subscale was reverse coded to facilitate interpretation as lower scores on the friction/shear subscale indicate higher levels of friction/shear. The Pearson product moment correlation was  $r = .196$ ,  $p = .000$ . In multivariate analysis of all patients, friction/shear was not found to be a significant predictor of pressure ulcer development in adult critical care patients ( $B = .867$ , 95% CI = .755- 7.510,  $p = .139$ ). Hypothesis 6 was supported in bivariate analysis.

#### *Hypothesis 7*

Hypothesis 7 stated that nutrition would be inversely related to pressure ulcer development in adult critical care patients. The Pearson product moment correlation was  $r = -.175$ ,  $p = .001$ . In multivariate analysis, nutrition was not found to be a significant predictor of pressure ulcer development ( $B = -.411$ , 95% CI = .277-1.586,  $p = .355$ ). Based on these findings, Hypothesis 7 was supported in bivariate analysis, but not supported in multivariate analysis.

#### *Hypothesis 8*

Hypothesis 8 stated that age was positively related to pressure ulcer development in adult critical care patients. The Pearson product moment correlation was  $r = .130$ ,  $p = .015$ . In direct logistic regression, age emerged as a significant predictor of pressure ulcer development in adult critical care patients ( $B = .033$ , 95% CI = 1.00-1.06, OR = 1.03,  $p = .030$ ). Based on these findings, Hypothesis 8 was supported in both bivariate and multivariate analysis.

#### *Hypothesis 9*

Hypothesis 9 stated that arteriolar pressure was inversely related to pressure ulcer development in adult critical care patients. The Pearson product moment correlation between mean arteriolar pressure below 60 and pressure ulcer development was  $r = .122$ ,  $p = .023$ . In direct logistic regression, mean arteriolar pressure below 60 did not emerge as a significant predictor of pressure ulcer development in adult critical care patients ( $B = -.075$ , 95% CI = .857-1.00,  $p = .067$ ). The Pearson product moment correlation between systolic blood pressure below 90 mmHg and pressure ulcer development was  $r = .140$ ,  $p = .009$ . In multivariate analysis, systolic blood pressure below 90 did not emerge as a significant predictor of pressure ulcer development in this sample ( $B = .038$ , 95% CI =



.976-1.10,  $p = .229$ ) The Pearson product-moment correlation for diastolic blood pressure less than 60 and pressure ulcer development was  $r = .228$ ,  $p = .000$ . In direct logistic regression, diastolic blood pressure below 60 mmHg, did not emerge as a significant predictor ( $B = .018$ , 95% CI = .991- 1.04,  $p = .183$ ) Based on these findings, Hypothesis 9 was supported in bivariate analysis, but not in multivariate analysis.

#### *Hypothesis 10*

Hypothesis 10 stated that the length of the intensive care unit stay was positively related to pressure ulcer development in adult critical care patients. The Pearson product moment correlation for this relationship was  $r = .502$ ,  $p = .000$ . In multivariate analysis, ICU length of stay emerged as a significant predictor ( $B = .008$ , 95% CI = 1.00=1.01, OR = 1.008,  $p = .000$ ). Based on these findings, Hypothesis 10 was supported in bivariate and multivariate analysis.

#### *Hypothesis 11*

Hypothesis 11 stated that severity of illness, measured using the APACHE II score, was positively related to pressure ulcer development in adult critical care patients. The Pearson product moment correlation for this relationship was  $r = .288$ ,  $p = .000$ . In multivariate analysis, the total APACHE II score was not a significant predictor of pressure ulcer development in this sample ( $B = .019$ , 95% CI = .958-1.08,  $p = .547$ ). Hypothesis 11 was supported in bivariate analysis, but not in multivariate analysis.

#### *Hypothesis 12*

Hypothesis 12 stated that vasopressor administration would be positively related to pressure ulcer development in adult critical care patients. The Pearson product moment correlations for the relationships between vasopressor agents and pressure ulcer

development were as follows: norepinephrine ( $r = .395$ ,  $p = .000$ ), phenylephrine ( $r = .041$ ,  $p = .442$ ), epinephrine ( $r = .078$ ,  $p = .146$ ), dopamine ( $r = .087$ ,  $p = .105$ ) and vasopressin ( $r = .268$ ,  $p = .000$ ). Based on the significant bivariate correlations, norepinephrine and vasopressin were included in multivariate analysis. In multivariate analysis including all patients, norepinephrine and vasopressin did not emerge as significant predictors of pressure ulcer development in this sample ( $B = .011$ , 95% CI = .996- 1.02,  $p = .145$ ;  $B = .021$ , 95% CI = .996-1.04,  $p = .095$  respectively). Based on these findings, Hypothesis 12 is supported in bivariate analysis by the vasopressor agents norepinephrine and vasopressin.

### *Hypothesis 13*

Hypothesis 13 stated that comorbid conditions were positively related to pressure ulcer development in adult critical care patients. The Pearson product moment correlations testing these relationship were vascular disease ( $r = .115$ ,  $p = .031$ ) peripheral vascular disease ( $r = .043$ ,  $p = .429$ ) cardiovascular disease ( $r = .147$ ,  $p = .006$ ), diabetes mellitus ( $r = .014$ ,  $p = .800$ ) and infection ( $r = .210$ ,  $p = .000$ ). In multivariate analysis, cardiovascular disease was found to be a significant predictor of pressure ulcer development ( $B = 1.08$ , 95% CI = 1.34- 6.47, OR = 2.95,  $p = .007$ ), while infection was not found to be a significant predictor ( $B = .190$ , 95% CI = .534- 2.73,  $p = .649$ ). Based on these findings, Hypothesis 13 is partially supported in both bivariate (vascular disease, cardiovascular disease, infection) and multivariate analysis (cardiovascular disease). Table 3 summarizes the bivariate correlations between the independent variables and the dependent variable of pressure ulcer development.

Table 3

**Bivariate Correlations: Independent Variables and Dependent Variable (Pressure Ulcer Development)**

<b><u>Independent Variable</u></b>	<b><u>r</u></b>
Total Braden Score	-.147**
• Mobility	-.275**
• Activity	-.103
• Sensory Perception	-.208**
• Moisture	.104
• Friction/Shear	.196**
• Nutrition	-.175**
Age	.130*
Arteriolar Pressure	
• MAP < 60	.122*
• Systolic<90	.140**
• Diastolic<60	.288**
Length of ICU Admission	.502**
Severity of Illness (APACHE II)	.288**
Vasopressor Administration	
• Norepinephrine	.395**
• Phenylephrine	.041
• Epinephrine	.178
• Dopamine	.105
• Vasopressin	.268**
Comorbid Conditions	
• Vascular Disease	.115*
Cardiovascular Disease	.147**
PVD	-.043
• Diabetes Mellitus	.014
• Infection	.210**

\*Correlation significant  $\leq 0.05$  level\*\* Correlation significant  $\leq 0.01$  level*Hypothesis 14*

Hypothesis 14 stated that there would be significant combined effects in terms of variance accounted for of theoretically- and empirically- derived risk factors on pressure ulcer development in adult critical care patients. Independent variables found to be significantly associated with the dependent variable of pressure ulcer development were

included in a logistic regression analysis. These variables included total Braden scale score, mobility, sensory perception, friction/shear, nutrition, age, arteriolar pressure (mean arterial pressure < 60, systolic blood pressure < 90, diastolic blood pressure < 60), length of intensive care unit admission, severity of illness, vasopressor administration (norepinephrine, vasopressin) and comorbid conditions (cardiovascular disease, infection). Vascular disease was eliminated from the regression as the correlation coefficient between vascular disease and cardiovascular indicated a high degree of multicollinearity ( $r = .937$ ,  $p = .000$ ). According to Munro (2005), correlations between variables of greater than 0.85 indicate the presence of multicollinearity. All variables were entered into the regression simultaneously. According to Tabachnick and Fidell (2001), this is the preferred method if predictor variables do not differ from each other in terms of order or importance.

The test of model coefficients indicated that the model was significant ( $\chi^2=132.135$ ,  $df = 8$ ,  $p = .000$ ). The goodness of fit statistic using the Hosmer-Lemeshow Test indicated that the model was a good fit for the data ( $\chi^2= 11.67$ ,  $df=8$ ,  $p= .167$ ) (Table 4). The overall accuracy of the model to predict patients developing a pressure ulcer was 88 %. The sensitivity of the model was 51% and the specificity was 97%. The positive predictive value of the model was 77% and the negative predictive power was 89%. Tables 4 and 5 summarize the Hosmer-Lemeshow Test and the model discrimination.

Table 4

Hosmer and Lemeshow Test			
Step	Chi-square	df	Sig.
1	6.993	8	.537

Table 5

Observed			Predicted		
			PU		Percentage Correct
			no	yes	
Step 1	PU	No	272	10	96.5
		Yes	32	33	50.8
		Overall Percentage			87.9

a. The cut value is .500

According to the Cox & Snell and Nagelkerke R Squares tests, 32% to 51% of the variance in pressure ulcer development is explained by the model (Table 6). Table 7 depicts the regression coefficients, Wald statistics, odds ratios, significance levels and 95% confidence intervals for the independent variables that were included in the regression equation. Odds ratio revealed that patients in the sample who were more mobile were 60% less likely to develop a pressure ulcer as compared to patients who were less mobile ( $B = -.823$ ,  $p = .039$ ,  $OR = .439$ , 95% CI 20% to 96%). Older patients as compared to younger patients were 3% more likely to develop a pressure ulcer ( $B = .003$ ,  $p = .030$ ,  $OR = 1.033$ , 95% CI 3% to 6.4%) and patients with longer ICU stays as compared to shorter ICU stays were 1% more likely to develop a pressure ulcer ( $B = .009$ ,  $p = .000$ ,  $OR = 1.008$ , 95% CI 5% to 1.1%). Patients with cardiovascular disease were 2.9 times more likely to develop a pressure ulcer than those without a history of cardiovascular disease ( $B = 1.082$ ,  $p = .007$ ,  $OR = 2.952$ , 95% CI 1.3-6.4).

Table 6 Model Summary

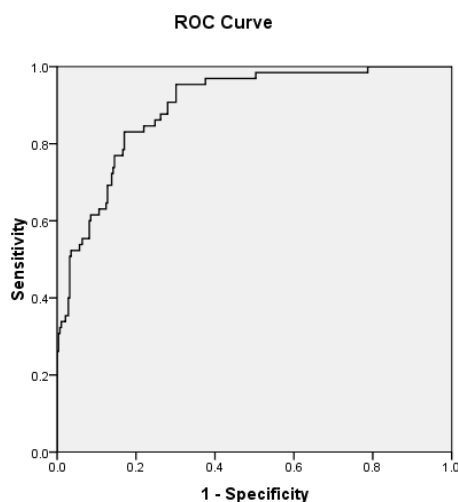
Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	202.591 <sup>a</sup>	.317	.512

Table 7. Variables in the Equation

	B	S.E.	Wald	Sig.	Exp(B)
Step 1 <sup>a</sup>					
age	.033	.015	4.725	.030	1.033
totalhrsICU	.008	.002	21.996	.000	1.008
Cardiovas(1)	1.082	.401	7.288	.007	2.952
coinfection(1)	.190	.416	.208	.649	1.209
MAPbelow60	-.075	.041	3.363	.067	.928
sysbelow90	.038	.032	1.450	.229	1.039
diasbelow60	.018	.014	1.771	.183	1.019
Bradentot	.102	.240	.181	.670	1.108
SensPer	.035	.376	.009	.926	1.035
Mobility	-.823	.398	4.262	.039	.439
Nutrition	-.411	.445	.854	.355	.663
Fricshear	.867	.586	2.190	.139	2.381
TotNorepi	.011	.007	2.126	.145	1.011
totvasop	.021	.013	2.794	.095	1.021
totalapache	.019	.032	.363	.547	1.019
Constant	-7.049	2.857	6.087	.014	.001

The area under the ROC (receiver operating characteristics) curve was also analyzed. The area under the curve can be used to assess model discrimination. The value of the area under the curve ranges from 0.5 to 1.0. The closer this value is to 1.0, the better the model is at predicting pressure ulcer development. For this model, the area under the curve was calculated to be .89. Figure 1 graphically depicts the ROC curve.

Figure 3



Area under the Curve = .897

Based on the findings, Hypothesis 14 was partially supported. Significant theoretically-derived independent predictors included age and mobility. Significant empirically-derived risk factors included ICU length of stay and cardiovascular disease.

In summary, Hypothesis 1, 2, 4, 6, 7, 8, 9, 10 and 11 were supported only in bivariate analysis. Hypotheses 2, 8 and 10 were also supported in multivariable analysis. Hypotheses 3 and 5 were not supported. Hypothesis 12 was partially supported by two vasopressor agents- norepinephrine and vasopressin. Hypothesis 13 was partially supported by the comorbid conditions of cardiovascular disease and infection. In multivariate analyses, cardiovascular disease was found to be a significant predictor of pressure ulcer development. Hypothesis 14 was partially supported. The variables mobility, age, length of ICU admission and cardiovascular disease were found to be significant predictors of pressure ulcer development and explained 32% to 51% of the variance in pressure ulcer development in adult critical care patients.

### Additional Findings

Additional analyses were undertaken in order to better understand the relationships between the independent variables, demographic variables and pressure ulcer development. Specifically, the following additional analyses were conducted: 1) an examination of the relationship between race and gender, and pressure ulcer development; 2) an analysis of the extent to which the independent variables predicted pressure ulcer development in a subsample of patients with Stage 1 ulcers excluded; 3) a comparison of the extent to which theoretically- and empirically-derived risk factors differed in patients who developed pressure ulcers and patients that did not and 4) an examination of select independent variables on the subset of patients who developed a pressure ulcer ( n = 65).

#### *Race, Gender and Pressure Ulcer Development*

Data analyses were undertaken in order to determine if there were associations between the demographic variables of race and gender and pressure ulcer development. No statistically significant correlations were found with respect to both variables and the development of pressure ulcers( $r = .029$ ,  $p = .589$  and  $r = -.010$ ,  $p = .847$  respectively).

#### *Predictive Ability of Independent Variables for Development of Pressure Ulcers Defined as Stage II-IV, Unstageable and Deep Tissue Injury*

Since a Stage I ulcer is not defined as an actual break in skin integrity, an additional analysis of the extent to which the independent variables [total Braden scale score, mobility, sensory perception, friction/shear, nutrition, age, arteriolar pressure( mean arterial pressure, systolic blood pressure, diastolic blood pressure), length of intensive care unit admission, severity of illness, vasopressor administration( norepinephrine,



vasopressin) and comorbid conditions(cardiovascular disease, infection)] independently predicted the development of pressure ulcers defined as Stage II, Stage III, Stage IV, Unstageable and Suspected Deep Tissue Injury was conducted on the sample excluding the 20 patients that developed a Stage 1 pressure ulcer. The test of model coefficients indicated that the model was significant ( $\chi^2=122.964$ ,  $p=.000$ ). The goodness of fit statistic using the Hosmer-Lemeshow test revealed a nonsignificant result, indicating that the model was a good fit for the data ( $\chi^2= 11.67$ ,  $df=8$ ,  $p=.666$ ) (Table 8). The overall accuracy of the model to predict patients having a pressure ulcer was 90.5%. The sensitivity of the model was 51% and the specificity was 97%. The positive predictive value of the model was 72 % and the negative predictive value was 93% (Table 9).

Table 8

Hosmer and Lemeshow Test			
Step	Chi-square	df	Sig.
1	5.836	8	.666

Table 9

Classification Table					
Observed			Predicted		
			PU		Percentage Correct
			no	yes	
Step 1	PU	No	273	9	96.8
		Yes	22	23	51.1
		Overall Percentage			90.5

According to the Cox & Snell and Nagelkerke R Squares tests, 31% to 57% of the variance in pressure ulcer development was explained by this model (Table 10). Table 11 depicts the regression coefficients, Wald statistics, odds ratios, significance levels and

95% confidence intervals for the independent variables that were entered into the logistic regression analysis. Odds ratios revealed that patients who experienced greater amounts of friction/shear were almost six times more likely to develop a pressure ulcer of Stage II, III, IV, Unstageable or suspected Deep Tissue Injury than those with less or absent friction/shear ( $B=1.74$ ,  $p = .014$ ,  $OR = 5.715$ , 95% CI 1.423-22.95). Patients with longer lengths of ICU stay were 1% more likely to develop a pressure ulcer as compared to patients with shorter ICU stays ( $B= .008$ ,  $p = .000$ ,  $OR = 1.008$ , CI 4% to 12%). Patients who received more hours of norepinephrine were almost 2% more likely to develop a pressure ulcer as compared to those patients who received no norepinephrine or shorter durations of norepinephrine ( $B= .017$ ,  $p = .040$ ,  $OR = 1.017$ , 95% CI 1% to 33%) Patients with cardiovascular disease were almost 3.4 times more likely to have a pressure ulcer than those without a history of cardiovascular disease ( $B= 1.218$ ,  $p = .019$ ,  $OR = 3.380$ , 95% CI 1.22 to 9.34).

Table 10

Model Summary			
Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	139.035 <sup>a</sup>	.313	.569

Table 11

Variables in the Equation

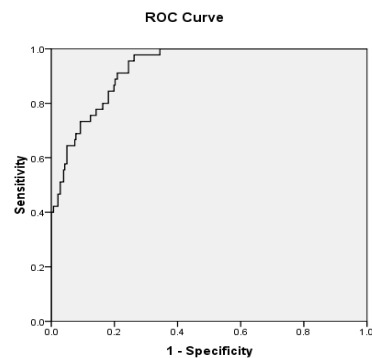
	B	S.E.	Wald	Sig.	Exp(B)
Step 1 <sup>a</sup> age	.030	.019	2.562	.109	1.031
totalhrsICU	.008	.002	18.063	.000	1.008
Cardiovas(1)	1.218	.519	5.510	.019	3.380
coinfection(1)	.180	.517	.121	.728	1.197
MAPbelow60	-.077	.046	2.792	.095	.926

sysbelow90	.011	.040	.067	.795	1.011
diasbelow60	.031	.017	3.213	.073	1.031
Bradentot	.033	.310	.011	.915	1.034
SensPer	.286	.487	.344	.557	1.331
Fricshear	1.743	.709	6.039	.014	5.715
TotNorepi	.017	.008	4.223	.040	1.017
Mobility	-.976	.508	3.687	.055	.377
Nutrition	.296	.581	.259	.611	1.344
totvasop	.026	.014	3.333	.068	1.026
totalapache	.015	.043	.122	.727	1.015
Constant	-10.512	3.779	7.737	.005	.000

a. Variable(s) entered on step 1: age, totalhrsICU, Cardiovas, coinfection, MAPbelow60, sysbelow90, diasbelow60, Bradentot, SensPer, Fricshear, TotNorepi, Mobility, Nutrition, totvasop, totalapache.

The area under the ROC (receiver operating characteristics) curve was also analyzed. For this model, the area under the curve was calculated to be .929. Figure 2 graphically depicts the ROC curve.

Figure 4



Area under the curve = .929

In summary, additional analysis of all patients excluding 20 patients that developed Stage I ulcers and including patients with Stage II, III, IV, suspected Deep Tissue Injury or Unstageable ulcers, four variables, friction/shear, length of ICU admission, norepinephrine infusion and cardiovascular disease explained 31%-57% of the variance in pressure ulcer development in adult critical care patients.

#### *Between Group Comparisons*

Additionally, patients who developed pressure ulcers were compared to patients who did not on the following variables: total Braden scale score, sensory perception, mobility, nutrition, friction/shear, age, length of ICU stay, norepinephrine infusions, vasopressin infusions, arteriolar pressure, total APACHE score, ICU admitting diagnosis, disposition at discharge and comorbid conditions (cardiovascular disease, infection).

Independent T-test analyses revealed significant differences in mean scores between patients who developed pressure ulcers of any stage and patients who did not develop pressure ulcers for the following variables: total Braden scale score(  $t = 5.9$ ,  $p = .000$ ), sensory perception (  $t = 3.95$ ,  $p = .000$ ), mobility( $t = 5.31$ ,  $p = .000$ ), nutrition (  $t = 3.68$ ,  $p = .000$ ), friction/shear (  $t = 3.71$ ,  $p = .000$ ), age ( $-2.72$ ,  $p = .007$ ), ICU length of stay ( $t = -6.20$ ,  $p = .000$ ), norepinephrine infusion (  $t = -.4.00$ ,  $p = .000$ ), vasopressin infusion (  $t = -2.57$ ,  $p = .012$ ), mean arterial pressure below 60 ( $t = -2.00$ ,  $p = .049$ ), systolic blood pressure below 90(  $t = -2.33$ ,  $p = .022$ ), diastolic blood pressure below 60 ( $t = -4.35$ ,  $p = .000$ ), and APACHE score ( $t = -5.58$ ,  $p = .000$ ).

Chi square revealed statistically significant differences between patients who developed pressure ulcers and those that did not for the following variables: ICU diagnosis [ $\chi^2 = 36.93(11, N = 347)$ ,  $p = .000$ ], disposition at discharge [ $\chi^2 = 36.33(5, N =$

347),  $p = .000$ ], cardiovascular disease [ $\chi^2 = 7.53(2, N = 347)$ ,  $p = .006$ ], and infection [ $\chi^2 = 15.29(2, N = 347)$ ,  $p = .000$ ]. Table 13 summarizes the comparison between patients that developed pressure ulcers and those that did not.

**Table 12**  
**Comparison of Patients with Acquired Pressure Ulcers and Patients without Pressure Ulcers on Select Independent Variables**

Variable	Pressure Ulcer M(SD)	No Pressure Ulcer M(SD)	Statistic
Total Braden Score	12.73(2.65)	14.63(2.65)	$t = 5.9^{**}$
Sensory Perception	2.40(.884)	2.94(.928)	$t = 3.95^{**}$
Mobility	2.06(.788)	2.64(.801)	$t = 5.31^{**}$
Nutrition	2.06(.555)	2.35 (.665)	$t = 6.38^{**}$
Friction/Shear	1.90(.491)	2.14(.458)	$t = 3.71^{**}$
Age	73.44(15.01)	67.50(17.88)	$t = -2.72^{**}$
ICU LOS	281.21(256.14)	81.41(85.78)	$t = -6.20^{**}$
Norepinephrine Infusion (hours)	54.98(101.50)	4.39(16.05)	$t = -4.00^{**}$
Vasopressin Infusion (hours)	16.15(47.59)	.909(7.09)	$t = -2.57^*$
MAP < 60 (hours)	3.67(5.83)	2.12(4.71)	$t = -2.00^*$
Systolic BP < 90 (hours)	5.61(7.72)	3.20(6.42)	$t = 2.33^*$
Diastolic BP < 60 (hours)	30.43(15.26)	21.43(14.95)	$t = -4.35^{**}$
APACHE II Score	21.89(6.71)	14.63(2.65)	$t = -5.58^{**}$

ICU Diagnosis	N (%)	N (%)	
-Respiratory Failure/Distress	24(37%)	48(17%)	$\chi^2(11, N = 347) = 36.93^{**}$
-Sepsis/Septic Shock	19 (29%)	41(18%)	
-GI bleed	2 (3%)	22 (4%)	
-GI Surgery	5 (8%)	18 (6%)	
-Neuro-med.	5 (8%)	47 (17%)	
-Neuro-surgery	1 (1%)	23 (8%)	
-Vascular Surg.	1(1%)	16 (6%)	
-Med-general	4(6%)	40 (14%)	
-Med-cardiac	1(1%)	11 (4%)	
-Cardiac arrest	3(4%)	3 (1%)	
-Surgery-other	0 (0%)	13 (5%)	
ICU Disposition			
Med-Surg	27 (42%)	214 (76%)	$\chi^2(5, N = 347) = 36.33^{**}$
Step-Down	17 (26%)	38 (14%)	
Rehab/SNF	1 (2%)	1 (0.4%)	
Dead	17 (26%)	19 (7%)	
Discharge	0 (0%)	3 (1%)	
Other	3 (5%)	7 (3%)	
Cardiovascular Disease	Yes 37(57%) No 28 (43%)	Yes 108(38%) No 174(62%)	$\chi^2(1, N = 347) = 7.53^{**}$
Infection	Yes 36(55%) No 29(45%)	Yes 84(30%) No 198(70%)	$\chi^2(1, N = 347) = 15.29^{**}$

\* Significance at or below 0.05

\*\*Significance at or below 0.01

#### *Additional Analysis of Patients with Pressure Ulcers*

In the subsample of patients who developed pressure ulcers (n=65), select independent variables were examined including: hours to pressure ulcer development, age, norepinephrine administration time and total Braden scale scores. Variables were recoded into categorical variables in order to provide more insight into the development of pressure ulcers and for ease of interpretation.

In analysis of all stages of pressure ulcers, the mean time to pressure ulcer development was 133.61 hours (range 5-573, SD 120.13). When time to pressure ulcer development was recoded into a categorical variable, 32% (21/65) of patients developed pressure ulcers in the first 48 hours of the admission, 11% (7/65) from 49 to 72 hours and 23%(15/65) between 73 hours and 145 hours. Therefore, 43% of the sample developed a pressure ulcer in the first 3 days and 66% of patients who developed a pressure ulcer did so in the first six days of the ICU admission. In patients with Stage II or greater pressure ulcers (n=45), 40% (18/45) of these patients developed the ulcer in the first three days of the ICU admission and 67% (30/45) developed the ulcer in the first six days of the ICU admission. No statistically significant relationship was found between pressure ulcer stage and time to pressure ulcer development( $r = -0.47$ ,  $p = .711$ ) in this subsample of patients.

Age was recoded into a categorical variable and revealed that 19% (12/65) of the patients that developed pressure ulcers were between 20-60, 29% were between the ages of 61-75, 35% were between 76 and 85 and 17% were 86 or older. Eighty-one percent of the patients with pressure ulcers were 61 years of age or older. A non-significant correlation was found between age and pressure ulcer stage ( $r = -.074$ ,  $p = .556$ ) in this subsample of patients.

Norepinephrine was recoded into a categorical variable for this analysis. Forty-nine percent (32/65) of patients who developed pressure ulcers of any stage also received norepinephrine. Of these, 41% (13/32) received norepinephrine for 48 hours or less. A total of 53% (17/32) of patients who developed a pressure ulcer received norepinephrine for 72 hours or less. Of the 34% (11/32) who developed pressure ulcers and received

norepinephrine infusions for greater than 121 hours, 90% (10/11) of the pressure ulcers that developed were Stage II or greater. No statistically significant association was found between norepinephrine infusion and pressure ulcer stage ( $r = .219$ ,  $p = .149$ ) in this subsample of patients.

The total Braden scale score was recoded into a categorical variable for this analysis. In patients who developed a pressure ulcer, 72% (47/65) had a Braden scale score of 14 or less as compared to 50% (142/282) of patients that did not develop a pressure ulcer. Forty-five percent (29/65) of patients with pressure ulcers were deemed at high risk for pressure ulceration (Braden scale score of 10-12) or very high risk (Braden scale score of 6-9). A statistically significant association between the total Braden score and pressure ulcer stage was not found ( $r = -.106$ ,  $p = .400$ ). Table 14 summarizes the additional analysis for patients with pressure ulcers.

**Table 13**  
**Analysis of Patients with Pressure Ulcers (n=65)**

<b>Time to Pressure Ulcer Development</b>	<b>n (%)</b>
0-48 hours	21 (32%)
49-72 hours	7 (11%)
73- 144 hours	15 (23%)
145 hours or more	22 (34%)

**Pressure Ulcers by Stage and Hours to Development**

	Stage I	Stage II	Stage III	Stage IV	DTI	Unstageable	Total
1-48 hours	8	7	0	0	5	1	21
49-72 hours	2	2	0	0	2	1	7
73- 144 hours	3	6	1	0	3	2	15
145 hours or greater	7	8	0	1	5	1	22
<b>Age and Pressure Ulcer (n=65)</b>		<b>n (%)</b>					
20-45 years		3(5%)					
46-60 years		9(14%)					



61-75 years	19(29%)
76-85 years	23(35%)
86 or older	11(17%)
<b>Norepinephrine and Pressure Ulcers (n=32)</b>	<b>n (%)</b>
0.5-24 hours	7(22%)
25-48 hours	6(19%)
49-72 hours	4(14%)
73-121 hours	4(14%)
122 or more hours	11(34%)
<b>Total Braden Score and Pressure Ulcers (n=65)</b>	<b>n(%)</b>
19-23 (no risk)	0
15-18 (at risk)	18(28%)
13-14 (moderate risk)	18(28%)
10-12 (high risk)	23(35%)
9 or less (very high risk)	6(9%)

## Chapter 5

### Discussion of the Findings

The purpose of this study was to examine the relationships between theoretically- and empirically-derived risk factors and pressure ulcer development in adult critical care patients. Risk factors derived from the Braden and Bergstrom Framework (i.e., theoretically-derived factors) under investigation included mobility, activity, sensory perception, moisture, friction/shear, nutrition, age and arteriolar pressure. Empirically-derived risk factors under investigation included length of intensive care unit stay, severity of illness, vasopressor administration and comorbid conditions. This chapter includes an interpretation of the findings of these hypothesized relationships in relation to the theory and empirical findings from which these hypotheses were derived.

### Theoretically-Derived Risk Factors

Hypotheses 1-7 tested the propositions that risk factors purported by the Braden and Bergstrom (1987) conceptual framework were significantly related to pressure ulcer development. The Braden scale measures cumulative risk of pressure ulcer development based on seven theoretically postulated risk factors including mobility, activity, sensory perception, moisture, nutrition and friction/shear. Hypothesis 1 tested the relationship between the total Braden scale score, representing cumulative risk and Hypotheses 2-7 tested the relationships between each of the individual risk factors and pressure ulcer development.

#### *Cumulative Risk and Pressure Ulcer Development*

Hypothesis 1 stated that there would be an inverse relationship between the total Braden scale score and pressure ulcer development in adult critical care patients. This

hypothesis was supported in correlational analysis which found a significant inverse correlation ( $r = -.276$ ). In multivariate analysis, the total Braden scale score was not found to be independently related to, that is, a significant predictor of pressure ulcer development in this population. In contrast to the findings in this study, cumulative risk as measured by the total Braden score in three previous studies in the critical care population significantly predicted pressure ulcer development in multivariate regression analysis (Bours et al., 2001; Carlson et al. 1999; Fife et al, 2001).

Upon examination of the Braden scale's predictive validity in the present study, the scale exhibited 100% sensitivity, 7% specificity, with a 20% predictive value of a positive test (PVP) and a 100% predictive value of a negative test (PVN) at a total score of 18. In a previous examination of the Braden scale's sensitivity and specificity in the critical care population, Bergstrom et al. (1987) found the predictive validity of the Braden scale to be 83% sensitive and 64% specific at a score of 18, with a PVP of 61% and PVN of 85%, while Jiricka and colleagues (1995) reported that at a score of 15, the sensitivity of the scale was 100%; the specificity 10.8%; the PVP 59.3%; and the PVN 100%. Findings in this study were consistent with the findings of Jiricka and colleagues, suggesting that in the ICU population, the predictive validity of the Braden scale may be suboptimal as evidenced by the extremely low specificity values. In contrast, research has shown that the Braden scale exhibits better specificity in non-critical care populations, indicating less false positive values. For example, in Capobianco and McDonald's (1996) study of 50 medical/surgical patients, they found that a Braden score of 18 resulted in 71% sensitivity and 83% specificity. Similarly, in the long term care setting, Braden and

Bergstrom (1994) found that at a score of 18, sensitivity and specificity were 75% and 79% respectively.

Overprediction of pressure ulcer cumulative risk is reflected by low specificity and low PVP values as demonstrated in the present study. According to Bolton (2007), overprediction typically is the weakest metric of predictive validity for any pressure ulcer risk assessment scale. Two explanations of overprediction are possible in the clinical environment. First, the risk assessment tool may have successfully identified patients at risk, subsequently mobilizing the clinician to implement appropriate pressure ulcer prevention strategies, thus successfully averting pressure ulcer occurrence. Or secondly, potentially unnecessary pressure ulcer prevention strategies were implemented, resulting in excessive healthcare costs and potential inefficient use of caregiver time.

In the current study, ancillary independent t-test analysis revealed a significant difference in mean Braden scale scores between patients in the sample who developed pressure ulcers and those that did not, however both groups scored well below a score of 18, indicating a cumulative pressure ulcer risk for both groups [ $M = 12.73 (SD = 2.65)$  and  $M = 14.63 (SD = 2.65)$  respectively]. This statistically significant finding may be of little clinical significance. Since 94% of patients in this sample were found to be at risk for pressure ulcer development, but only 18.7% of the entire sample developed a pressure ulcer, the Braden scale overpredicted pressure ulcer risk. This overprediction makes it difficult to draw any clinically significant conclusions regarding the scale's ability to predict pressure ulcer development in this population. According to Defloor and Grypdonck (2004), interpretation of the Braden scale's ability to predict risk must also be made in conjunction with the pressure ulcer prevention strategies in place. However, the

authors note that the larger the numbers of false positives, the greater likelihood that prevention strategies were needlessly implemented. In the present study, 75% (n = 261) of the patients were classified at risk for pressure ulcer development but remained pressure ulcer free.

While the Braden scale in the present study exhibited very low specificity and PVP, the sensitivity and PVN were extremely high and likely occurred because only 6% of the sample (n = 21) were not deemed to be at risk for pressure ulcer development based on their total Braden Scale scores. All of these patients who were not at risk remained pressure ulcer free.

The lack of significance of cumulative risk for pressure ulcer development in the current study may be the result of the timing of the pressure ulcer risk assessment used for analysis. In the ICU in which this study was conducted, pressure ulcer risk assessments are conducted in the first 24 hours of the patient's ICU admission and then repeated every 24 hours as recommended in current clinical practice guidelines (WOCN, 2003). The Braden scale score that was recorded in the first 24 hours of admission to the ICU was chosen as the pressure ulcer risk measure in this study as this represents a consistent time period for determining each patient's initial risk for pressure ulcer development. Similarly, the Braden scale score recorded upon admission to the ICU were used as the pressure ulcer risk measure in two other studies in the critical care population (Fife et al., 1999; Jiricka et al, 1995). In contrast, the timing of pressure ulcer risk measurements used in other studies in the ICU population was inconsistent and ranged from the use of cross-sectional measurement (risk assessment done at the time of data collection), mean Braden scale scores recorded over time and the lowest Braden scale

score recorded over a period of time (Bours et al., 2001, Carlson et al., 1999; Pender & Frazier, 2005). It is plausible that variations in the timing of the Braden scale score used for analysis may influence the scale's predictive ability. Thus, for future research, there is a need to standardize the timing of the Braden scale measurement used for data analysis.

### *Mobility and Pressure Ulcer Development*

Hypothesis 2 stated that mobility would be inversely related to pressure ulcer development in adult critical care patients. Correlational analysis revealed a relationship that was significant and in the theoretically expected direction, that is, a significant inverse association ( $r = -.275$ ) between mobility and pressure ulcer development. In multivariate analysis, mobility was significantly related to, that is a significant predictor of pressure ulcer development, and the hypothesis was supported. This finding is consistent with previous research that examined this relationship in ICU patients (Batson et al., 1993; Bours et al., 2001). To gain a further understanding of the influence of mobility on pressure ulcer development in ICU patients, differences in mean mobility scores between patients who developed pressure ulcers and those who did not were examined. Independent t-test analysis revealed that the mobility score was significantly lower ( $M = 2.06$ ,  $SD = .788$ ) in patients who developed pressure ulcers as compared to patients that did not develop pressure ulcers ( $M = 2.64$ ,  $SD = .665$ ). Theoretically, mobility is defined by Braden and Bergstrom (1987) as the ability to move independently regardless of the patient's activity level. In the ICU population, the activity level for the majority of ICU patients is considered bed-bound, therefore it is plausible that immobility (the inability of the patient to independently move in bed) and not level of activity would represent a significant pressure ulcer risk as findings from the present study suggest.

### *Activity and Pressure Ulcer Development*

Hypothesis 3 stated that activity would be inversely related to pressure ulcer development in adult critical care patients. This hypothesis was not supported and is consistent with other empirical studies that also found activity to be a non-significant predictor of pressure ulcer development in ICU patients (Bours et al, 2001; Carlson et al, 1999; Jiricka et al, 1995).

Activity, though similar conceptually to mobility is theoretically defined by Braden and Bergstrom (1987) as the overall degree of physical activity of the patient, ranging from bed bound to ambulatory. Since most ICU patients were bed-bound in this sample, there was little to no variability in the level of activity among all patients in this sample. Thus, for ICU patients, the concept of activity may not be useful for the prediction of pressure ulcer risk in patients who are bed-bound and inactive.

### *Sensory Perception and Pressure Ulcers*

Hypothesis 4 stated that sensory perception would be inversely related to pressure ulcer development in adult critical care patients. This hypothesis was supported in correlational analysis, but sensory perception was not independently related to, that is a significant predictor of pressure ulcer development in multivariate analysis. This finding is inconsistent with two studies that showed that sensory perception was a significant predictor of pressure ulcer development in ICU patients (Carlson et al., 1999; Jiricka et al., 1995). To further examine the influence of sensory perception on pressure ulcer development in the study sample, an ancillary analysis was done to examine differences in mean sensory perception scores between patients who developed pressure ulcers and those who did not. Independent t-test analysis revealed that the sensory perception scores

differed significantly between patients who developed pressure ulcers ( $M = 2.4$ ,  $SD = .886$ ) and those that remained pressure ulcer free ( $M = 2.94$ ,  $SD = .928$ ). Although, statistically significant, there is little variability evident between these scores. The lack of support for sensory perception as a predictor of pressure ulcer development in the present study may be attributed to the diminished levels of sensory perception of all ICU patients in this sample, rendering this risk factor non-significant when analyzed with other risk factors.

#### *Moisture and Pressure Ulcer Development*

Hypothesis 5 stated that moisture would be a positively related to pressure ulcer development in adult critical care patients. This hypothesis was not supported, however, the relationship did approach significance in correlational analysis ( $p = .054$ ). In two previous studies in the ICU population, moisture was found to be a significant predictor of pressure ulcer development (Bours et al., 2001; Jiricka et al., 1995). One plausible explanation for the non-significant finding in this study may be the frequent use of indwelling devices in the ICU setting that minimize the patient's skin exposure to moisture from two primary sources, urine (indwelling urinary catheters) and liquid stool (fecal containment devices). Bowel management systems, also called fecal containment devices, were introduced to the clinical market in 2004 after both studies identified above were published. In fact, the use of these devices in the ICU setting, in combination with a pressure ulcer prevention program, was found to decrease pressure ulcer development for patients exposed to high levels of moisture from liquid stool incontinence (Benoit & Watts, 2007).



### *Friction/Shear and Pressure Ulcer Development*

Hypothesis 6 stated that friction/shear would be positively related to pressure ulcer development in adult critical care patients, and this hypothesis was supported in correlational analysis. However, friction/shear was not found to be independently related to that is a significant predictor of pressure ulcer development in the study sample.

Since a Stage I pressure ulcer is not defined as a break in skin integrity, a logistic regression analysis was undertaken to examine the independent relationship between friction/shear and pressure ulcers that are characterized by actual alterations in skin integrity. This ancillary analysis was conducted on the subsample of patients (n= 327) who developed Stage II , Stage III, Stage IV, suspected Deep Tissue Injury and Unstageable pressure ulcers and those that remained pressure ulcer free. Findings revealed that friction/shear was a significant predictor of Stage II and greater pressure ulcer development ( $B= 1.74$ ,  $p = .014$ ,  $OR = 5.715$ ,  $95\% CI = 1.423-22.95$ ). This finding is consistent with Jiricka and colleagues' (1995) study of ICU patients who found friction/shear to be a significant predictor of pressure ulcer development. In the present study, patients with higher exposure to friction/shear were almost six times more likely to develop a Stage II or greater pressure ulcer. Since friction alone causes skin damage confined to the epidermal and dermal layers, and in combination with shear causes damage at deeper levels due to the angulation of the vessels in the deep superficial fascia (Pieper, 2000), these combined forces likely result in pressure ulcers at Stage II or greater and may explain the predictive ability of this risk factor for Stage II or greater pressure ulcers in this study.

Repositioning of patients and patient transfers are two common activities that subject patients to the forces of friction and shear. Conceptually, friction and shear diminish tissue tolerance for pressure and lead to pressure ulcer development (Braden and Bergstrom, 1987). It is plausible that, in the critically ill, diminished levels of mobility lead to a patient's total dependence on caregivers for both repositioning and transfers, thereby subjecting the patient to greater levels of friction and shear, which can result in pressure ulcer development (Sibbald et al., 2009).

#### *Nutrition and Pressure Ulcer Development*

Hypothesis 7 stated that nutrition would be inversely related to pressure ulcer development in adult critical care patients. A significant correlation was found between nutrition and pressure ulcer development ( $r = -.175$ ), however in multivariate analysis, nutrition was not found to be independently related to pressure ulcer development. This finding is consistent with other studies in the critical care population that found that nutrition was not a significant predictor of pressure ulcer development (Bours et al., 2001; Carlson et al., 1999; Jiricka et al., 1995). Eachempati and colleagues (2001) did not use the Braden scale to measure nutrition, and measured nutrition as the "number of days without nutrition". This measure significantly predicted pressure ulcer development in critical care patients. One explanation for the non-significant finding in this study may be an incongruence between the conceptualization of nutrition and how it is operationalized by the Braden nutritional subscale in ICU patients. Theoretically, nutrition is defined as the patient's usual nutritional intake pattern (Braden and Bergstrom, 1987). A critically ill patient's inability to articulate a diet history may render the Braden nutritional score useless in the initial days of the ICU admission. Additionally, many biological markers

for nutrition such as body weight, albumin and, in some cases, prealbumin may produce erroneous results due to fluid shifts that occur in critical illness, thus creating greater challenges in determining appropriate objective nutritional markers. According to Doughty (2008), nutrition is a dimension of most pressure ulcer risk assessment scales including the Braden scale; however there still remains lack of agreement among researchers and clinicians regarding the best metric of nutritional status.

#### *Age and Pressure Ulcer Development*

Hypothesis 8 stated that age was positively related to pressure ulcer development in adult critical care patients. This hypothesis was supported in both bivariate and multivariate analyses. Additionally, the mean age of patients in the study who developed pressure ulcers was 73 years compared to a mean age of 67 years in patients who did not develop pressure ulcers. Age, as hypothesized by Braden and Bergstrom (1987), is an intrinsic factor that influences tissue tolerance to pressure; however it is not included as a measure in the Braden scale. Support for advancing age as a risk factor for pressure ulcer development in ICU patients is also evident in the empirical literature (Bours et al., 2001; Eachempati et al., 2001; Frankel et al., 2007; Theaker et al., 2000). The findings from this study add to a small but growing body of evidence that supports advancing age as a risk factor for pressure ulcer development in ICU patients.

#### *Arteriolar Pressure and Pressure Ulcers*

Hypothesis 9 stated that arteriolar pressure was inversely related to pressure ulcer development in adult critical care patients. This hypothesis was based on the theoretical proposition purported by Braden and Bergstrom (1987) that low arteriolar pressure diminishes tissue tolerance for pressure leading to pressure ulcer development.

Significant correlations were found between mean arterial pressure (MAP) below 60 ( $r = .122$ ), systolic blood pressure less than 90 ( $r = .140$ ) and diastolic blood pressure less than 60 ( $r = .228$ ) and pressure ulcer development. Additionally, independent t- test analysis revealed that patients who developed pressure ulcers experienced a MAP less than 60 for significantly longer periods of time (mean hours = 3.67,  $p = .049$ ), a systolic BP less than 90 for significantly longer periods of time (mean hours = 5.61,  $p = .022$ ) and a diastolic BP less than 60 for significantly longer periods of time (mean hours = 30.43,  $p = .000$ ) than patients who did not develop pressure ulcers (mean hours = 2.12, 3.2, and 21.43 respectively). In multivariate analysis, however, low arteriolar pressure variables were not independently related to pressure ulcer development. These results were similar to findings in previous studies in the ICU population that found non-significant relationships between low arteriolar pressure and pressure ulcer development (Batson, et al., 1993; Pender & Frazier, 2005). One explanation for this non-significant finding may be that, in the ICU setting, the frequent monitoring of blood pressure results in quicker implementation of interventions aimed at raising arterial pressure, thus the effect of lower arteriolar pressure on pressure ulcer development may be minimized.

#### Empirically-Derived Risk Factors

Hypotheses 10 to 13 tested the relationships between four empirically-derived risk factors and pressure ulcer development in adult critical care patients. These risk factors included length of intensive care unit stay, severity of illness, vasopressor administration and comorbid conditions.

### *Length of Stay and Pressure Ulcers*

Hypothesis 10 stated that there would be a positive relationship between intensive care unit length of stay and pressure ulcer development in adult critical care patients. This hypothesis was supported in bivariate and multivariate analysis. Patients with longer lengths of stay in the intensive care unit were more likely to develop pressure ulcers in this study. For patients that developed pressure ulcers, the mean length of stay was 281 hours (11.7 days). This compares to a mean length of stay of 81 hours (3.3 days) for patients who remained pressure ulcer free. This finding is consistent with other empirical literature in the critical care population that length of stay in the ICU was a significant predictor of pressure ulcer development (Bours, et al., 2001; Eachempati et al., 2001; Theaker et al., 2000). Findings from this study and previous research support the proposition that patients who stay for longer periods of time in an ICU setting have a greater risk of pressure ulcer development. The time to pressure ulcer development is also of clinical value. For patients in this study who developed a pressure ulcer, 66% of patients developed the ulcer in the first six days of the ICU admission, suggesting that this time period in the admission should be marked with hypervigilance with regard to pressure ulcer risk assessment and prevention strategies.

### *Severity of Illness and Pressure Ulcer Development*

Hypothesis 11 stated that severity of illness would be positively related to pressure ulcer development in adult critical care patients. The APACHE II scale was used to measure severity of illness and is a reliable and valid prognostic indicator of illness severity. This hypothesis was supported in correlational analysis. A significant positive correlation was found between severity of illness and pressure ulcer development ( $r =$

.288). In multivariate analysis, severity of illness was not found to be independently related to, that is a significant predictor of pressure ulcer development. While not a significant predictor, differences in mean illness severity scores revealed that patients who developed pressure ulcers had significantly higher mean levels of illness severity as measured by the APACHE II (  $M = 21.89$ ,  $SD 6.71$ ) compared to patients who remained pressure ulcer free ( $M = 14.63$ ,  $SD 2.65$ ).

Strong empirical support is not evident for illness severity as a predictor of pressure ulcer development in the critical care population. For three studies that measured the relationship between illness severity and pressure ulcer development, only one of these studies (Theaker et. al., 2000) found that illness severity was a significant predictor. Findings from the present study are consistent with previous findings and suggest that, while the APACHE II scale provides a valid measure of severity of illness and mortality risk, the APACHE II scale may not be a reliable empirical indicator for severity of illness as a pressure ulcer risk.

#### *Vasopressor Administration and Pressure Ulcer Development*

Hypothesis 12 stated that vasopressor administration would be positively related to pressure ulcer development in adult critical care patients. Vasopressor agents examined in this investigation were norepinephrine, epinephrine, phenylephrine, dopamine and vasopressin. This hypothesis was partially supported in correlational analysis. Of these five agents, only norepinephrine and vasopressin were found to be significantly associated with pressure ulcer development ( $r = .395$ ,  $p = .000$ ; and  $r = .268$ ,  $p = .000$ , respectively). In multivariate analysis, neither norepinephrine nor vasopressin was independently related to pressure ulcer development. However, in an ancillary

multivariate analysis of a subsample of patients ( $n = 327$ ) that excluded those patients who developed a Stage I ulcer, norepinephrine significantly predicted Stage II or greater pressure ulcer development. Of note, 32 (49%) of the 65 patients in the present study that developed a pressure ulcer received norepinephrine. Moreover, the mean number of hours of norepinephrine infusions that patients who developed Stage II or higher pressure ulcers received during the ICU stay was significantly higher ( $M = 55$  hours) compared to a mean number of hours of norepinephrine infusion for those patients who remained pressure ulcer free ( $M = 4$  hours).

These findings are similar to those in previous studies (Batson, et al, 1993; Theaker et al, 2000). One complication of norepinephrine administration is hypoperfusion caused by excessive vasoconstriction in response to hypotension (Gooneratne & Manaker, 2008). This hypoperfusion produces inadequate perfusion of the extremities, most notably in the fingers and toes, mesenteric organs and kidneys. It is plausible that this potent vasoconstriction can also result in hypoperfusion of the skin, resulting in ulcer formation over bony prominences. There is a small, but growing body of evidence to support norepinephrine as a predictor of pressure ulcer development in ICU patients, however more research is needed to validate this relationship.

The lack of significance of epinephrine and phenylephrine as risk factors for pressure ulcer development may represent a methodological limitation of this study. The number of patients in the sample who received epinephrine and phenylephrine may have been too small to yield statistical significance ( $n = 4$  and  $n = 16$  respectively).

### *Comorbid Conditions and Pressure Ulcer Development*

Hypothesis 13 stated that comorbid conditions would be positively related to pressure ulcer development in adult critical care patients. Vascular disease (cardiovascular and peripheral), diabetes mellitus and infection were the comorbid conditions examined. This hypothesis was partially supported. Significant correlations between vascular disease ( $r = .115$ ,  $p = .031$ ), cardiovascular disease ( $r = .147$ ,  $p = .006$ ), and infection ( $r = .210$ ,  $p = .000$ ) and pressure ulcer development were found. In multivariate analyses, only cardiovascular disease was independently related to pressure ulcer development.

Findings from the empirical literature reveal conflict regarding the relationship between comorbidity and pressure ulcer development in ICU patients. Vascular disease was significant in univariate analysis in one study (Frankel et al., 2007), while peripheral vascular disease was found to be a significant, independent predictor in a second study (Theaker et al., 2000) and non-significant in a third study (Batson et al., 1993). The lack of significance of peripheral vascular disease as a predictor in this study may also have occurred because of a methodological limitation; only 9% ( $n = 30$ ) of patients in the sample had a history of peripheral vascular disease.

On the other hand, cardiovascular disease was found to be a significant predictor of pressure ulcer development. The association between cardiovascular disease and pressure ulcer development has been supported in non-ICU populations including inpatients in general hospitals (Bergstrom et al., 1996; Lindgren et al., 2004) and cardiac surgery patients (Lewicki et al., 1997; Pokorny, et al. 2003). According to Lewicki and colleagues (2000), increased prevalence of pressure ulcers among patients undergoing



cardiac surgery may be due to the pathophysiological changes that occur with cardiovascular disease including underlying vessel disease, poor ventricular function, and heart failure, all which diminish the cardiovascular system's ability to respond to pressure. It is plausible that these pathophysiological mechanisms underlying the development of pressure ulcers in cardiac surgery patients are also a mechanism for pressure ulcer development in non-cardiac surgery ICU patients with cardiovascular disease and further research is warranted.

*A Multivariate Model Explaining Pressure Ulcer Development in Adult Critical Care Patients*

Hypothesis 14 stated that there would be a significant combined effects in terms of variance accounted for, of theoretically- and empirically-derived risk factors on pressure ulcer development in adult critical care patients. Hypothesis 14 was partially supported. Two theoretically-derived risk factors, mobility and age and two empirically-derived risk factors, ICU length of stay and cardiovascular disease accounted for 32% to 51% of the variance in pressure ulcer development in this sample of adult critical care patients.

Pressure ulcer risk is described as a multivariate phenomenon in Braden and Bergstrom's (1987) conceptual model. In their pressure ulcer risk model, initially developed and tested in patients in skilled nursing facilities and the long term care setting, factors are postulated as risks for pressure ulcer development (mobility, activity, sensory perception, moisture, nutrition, friction/shear, age and arteriolar pressure). In the current study, significant bivariate relationships were found between all of these risk factors and pressure ulcer development with the exception of moisture and activity. However in multivariate analyses, only three of these factors (mobility, friction/shear and

age) were independently related to pressure ulcer development. While a small body of studies in the critical care population have found sensory perception, mobility, moisture, friction/shear (Bours et al., 2001; Carlson et al., 1999; Jiricka et al., 1995) and age (Bours et al., 2001; Eachempati et al. 2001; Frankel, et al, 2007; Theaker et al., 2000) to be significant risk factors for pressure ulcer development, no studies in this population have found nutrition as measured by the Braden scale, or activity to be significantly related to pressure ulcer development.

Other risk factors, not included in the Braden and Bergstrom framework accounted for some variance in pressure ulcer development in this study. Length of ICU stay, norepinephrine administration, and cardiovascular disease were independently related to pressure ulcer development in multivariate analysis, suggesting that these factors should be considered important risks for pressure ulcer development in critically ill adults. In fact, findings in this study related to length of ICU stay and norepinephrine administration support a small but growing body of empirical evidence that these factors pose a significant risk for pressure ulcer development in the critical care population (Batson, et. al, 1993; Bours et al., 2001; Eachempati et al., 2001; Theaker et al., 2000)

While the multifactorial etiology of pressure ulcer development is evident, findings from this study suggest that the pressure ulcer risks stipulated in Braden and Bergstrom's conceptual framework alone do not fully explain pressure ulcer development in the critical care population. The combined effects of some, but not all theoretically- and empirically-derived risk factors accounted for significant variance in pressure ulcer development, suggesting that refinement and testing of a risk assessment model for

critical care patients is necessary in order to accurately assess pressure ulcer risks and provide a basis for explaining pressure ulcer development in this population.

## Chapter 6

### Summary, Conclusions, Limitations, Implications and Recommendations

This study was designed to gain a greater understanding of the risk factors for pressure ulcer development in adult critical care patients. This study examined the relationships between nine theoretically-derived risk factors and pressure ulcer development (activity, mobility, sensory perception, moisture, nutrition, friction/shear, age and arteriolar pressure) and four empirically-derived risk factors and pressure ulcer development (length of ICU stay, severity of illness, vasopressor administration and comorbid conditions).

Theoretically-derived risk factors under investigations were based on the Braden and Bergstrom (1987) conceptual framework for the etiology of pressure ulcers. In this framework, positive relationships were posited between friction/shear, age, moisture and pressure ulcer development, while inverse relationships were posited between activity, mobility, sensory perception, nutrition, arteriolar pressure and pressure ulcer development.

Empirically-derived risk factors examined that were posited to have positive relationships with pressure ulcer development included length of ICU stay, severity of illness, vasopressor administration and comorbid conditions.

Since pressure ulcer risk is considered to be a multifactorial phenomenon, the combined effect of the theoretically- and empirically-derived risk factors was examined in order to determine the cluster of risk factors, or risk factor model that pose the greatest threat for pressure ulcer development in hospitalized critically ill patients.

The following hypotheses were tested:

1. Total Braden scale score, or cumulative risk, is inversely related to pressure ulcer development in adult critical care patients.
2. Mobility is inversely related to pressure ulcer development in adult critical care patients.
3. Activity is inversely related to pressure ulcer development in adult critical care patients.
4. Sensory perception is inversely related to pressure ulcer development in adult critical care patients.
5. Moisture is positively related to pressure ulcer development in adult critical care patients.
6. Friction/Shear is positively related to pressure ulcer development in adult critical care patients.
7. Nutrition is inversely related to pressure ulcer development in adult critical care patients.
8. Age is positively related to pressure ulcer development in adult critical care patients.
9. Arteriolar pressure is inversely related to pressure ulcer development in adult critical care patients.
10. Length of intensive care unit stay is positively related to pressure ulcer development in adult critical care patients.
11. Severity of illness is positively related to pressure ulcer development in adult critical care patients.
12. Vasopressor administration is positively related to pressure ulcer development in adult critical care patients.

13. Comorbid conditions are positively related to pressure ulcer development in adult critical care patients.

14. There will be significant combined effects in terms of variance accounted for, of theoretically- and empirically-derived risk factors on pressure ulcer development in adult critical care patients.

The study sample consisted of 347 patients that were admitted into an adult critical care unit at a Magnet teaching hospital in the northeast. Patients were included if they met the inclusion criteria of 18 years of age or greater and in the ICU for  $\geq 24$  hours. Patients were excluded if they were under 18 years of age, had been in the ICU for  $< 24$  hours or had a pre-existing pressure ulcer. The sample consisted of 49% male and 51% female. Ages of the patients ranged from 20 to 97. Seventy-four percent of the sample was Caucasian and thirteen percent were Black/African American. The most frequent reason for admission into the ICU was respiratory distress/respiratory failure. Of the 347 patients, 65 developed a pressure ulcer during the ICU stay, yielding a total pressure ulcer incidence of 18.7%.

All data were abstracted from various parts of a computerized patient record. The Braden scale scores for seven of the theoretically-derived risk factors (activity, mobility, sensory perception, moisture nutrition and friction/shear) were abstracted from the patient record. Age, arteriolar pressure, length of ICU stay, vasopressor administration and comorbid conditions were also obtained from data recorded in the patient record. Severity of illness, measured using the APACHE II clinical data, was abstracted from the patient record and used to calculate the severity of illness score.

Hypotheses 1-13 were tested using Pearson's product moment correlations and direct logistic regression. Risk factors significantly related to pressure ulcer development in correlational analysis were subjected to logistic regression analysis and included the following: cumulative risk as measured by the total Braden scale score, mobility, sensory perception, nutrition, friction/shear, age, arteriolar pressure, length of ICU stay, norepinephrine and vasopressin administration, APACHE II severity of illness score and the comorbid conditions of cardiovascular disease and infection. In multivariate analysis, mobility, age, length of ICU stay and cardiovascular disease explained 32% to 51% of the variance in pressure ulcer development in this study sample of adult ICU patients.

In summary, while the majority of risk factors were found to be significantly associated with pressure ulcer development in bivariate correlational analyses in this sample, multivariate analysis revealed that only two theoretically-derived risk factors (mobility and age), and two empirically-derived risk factors (ICU length of stay and cardiovascular disease) were independently related to pressure ulcer development.

#### Limitations

1) Due to the retrospective design of this study, the following limitations are recognized:

a) The researcher's lack of control over the risk assessment measurements conducted by staff nurses. Risk assessment using the Braden scale is conducted on a daily basis in the ICU setting where this research occurred by nurses that have been trained to conduct these assessments.

b) The researcher's inability to assess and stage developing pressure ulcers.

Pressure ulcers were staged and recorded in the patient record by staff nurses caring for the patient. The nursing staff receives annual education on pressure ulcer assessment and staging.

c) Lack of control over accuracy of the data recorded in the medical record. Most of the data abstracted for this study, however represents objective clinical data that would not vary based on the study design.

2) Use of a single intensive care unit in one hospital decreases the generalizability of these study findings.

### Conclusions

Conclusions that may be drawn from this study of 347 adult critical care patients include the following:

- 1) Theoretically-derived risk factors significantly associated with pressure ulcer development were mobility, sensory perception, nutrition, friction/shear, age and arteriolar pressure.
- 2) Empirically-derived risk factors significantly associated with pressure ulcer development were length of ICU stay, severity of illness, vasopressor administration (norepinephrine/vasopressin) and the comorbid conditions of cardiovascular disease and infection.
- 3) The risk factors mobility, age, ICU length of stay and cardiovascular disease explained a significant portion of the variance in pressure ulcer development.



- 4) Refinement of the Braden and Bergstrom conceptual model or development of a critical care risk assessment model is needed in order to more accurately explain pressure ulcer development in this population.

#### Implications for Nursing

Pressure ulcer risk has been described as complex and multifactorial (WOCN, 2009). The Braden and Bergstrom (1987) framework for the etiology of pressure ulcers postulates a cluster of risk factors that seek to explain the development of a pressure ulcer. However, risk factors not described in this framework may also explain pressure ulcer development in the ICU population as suggested by the findings of this study and previous research.

The Braden scale, based on the Braden and Bergstrom framework, is the most widely used tool to measure pressure ulcer risk in the United States today (Lyder & Ayello, 2008). In the critical care population, most patients are deemed at risk for pressure ulcer development based on the Braden scale risk assessment; however, cumulative risk as measured by the Braden scale was not predictive in this sample of ICU patients. According to Pancorbo-Hildago and colleagues (2006), there is a paucity of evidence regarding a decrease in pressure ulcer occurrence based on current risk assessment procedures. In the critical care population, both de Laat and colleagues (2006) and Keller and colleagues (2002) concur that no risk assessment tool currently exists that adequately measures pressure ulcer risk in this population. Development and testing of a risk assessment model that explains pressure ulcer development in critically ill adults can provide an appropriate foundation for the development of a risk assessment tool designed exclusively for use in the critical care population.

Two risk factors theorized by Braden and Bergstrom, mobility and friction/shear significantly predicted pressure ulcer development in this study sample. Turning and repositioning an immobile patient is a basic tenet of nursing care. Regular repositioning of patients with diminished levels of mobility is recommended in current pressure ulcer prevention guidelines. While evidence describing the optimal frequency for repositioning immobile patient is lacking (Krapfl & Gray, 2008), there is consensus that regular repositioning is vital especially in patients with diminished mobility. In a Cochrane review of support surfaces, a small body of evidence also supports the use of low air loss pressure redistribution mattresses in the ICU population (McInnes et al., 2008). The use of low air loss pressure redistribution mattresses in combination with regular turning and repositioning of immobile patients may be two pressure ulcer prevention strategies essential in this population to reduce pressure ulcer occurrence.

The very act of repositioning an immobile patient predisposes the patient to increased exposure to friction/shear, which may alter the skin's integrity. Advocates of safe patient handling procedures recommend the use of glide sheets and patient transfer devices to reduce friction and shear, while simultaneously protecting staff from musculoskeletal injuries (Sibbald et al., 2009). As friction/shear has been shown to be independently related to pressure ulcer development in this population in this study sample, incorporation of these devices in the critical care setting may prove to be advantageous in diminishing the deleterious effects of friction/shear on the skin of critically ill patients.

Patients in this study who experienced the longest lengths of stay were more likely to develop a pressure ulcer. The most vulnerable time for pressure ulcer development was

found to be the first week of the ICU stay. This finding was consistent with other studies in the ICU population (Carlson, et al, 1999; Fife et al., 2001). Based on this finding, the first week of the ICU admission should be the point at which pressure ulcer prevention strategies should be initiated, in combination with increased vigilance by the staff for pressure ulcer occurrence. The first week of the ICU admission, however is also the most likely time period in which the patient experiences the greatest physiologic instability, requiring nursing and other members of the health care team to manage multiple life-saving technologies while simultaneously preventing pressure ulcers. During this time period, communication of the potential for pressure ulcer development among all members of the health care team is crucial. Daily multidisciplinary rounds may be an appropriate forum for the discussion of pressure ulcer prevention strategies. Moreover, a multidisciplinary forum can also serve to underscore the premise that pressure ulcer prevention is the responsibility of all health care team members and not an aspect of patient care exclusively in the domain of nursing.

Findings from this study also suggest that other intrinsic and extrinsic factors play a significant role in the development of pressure ulcers in this population. These factors include advanced age, cardiovascular disease and norepinephrine administration. As suggested by these findings, older patients, those patients with a history of cardiovascular disease and those patients receiving norepinephrine should be prime targets for early implementation of pressure ulcer prevention strategies.

Even with proper implementation of the best prevention strategies, pressure ulcers do occur. In a position statement by the Wound, Ostomy, and Continence Nurses Society (2009), unavoidable pressure ulcer occurrence is recognized as a phenomenon that can

occur in certain clinical circumstances in which all pressure ulcer risk factors can be not modified or removed. Certain prevention strategies may be medically contraindicated such as turning of a hemodynamically unstable patient or the presence of multiple risk factors can make it increasingly difficult for the health care team to adequately prevent pressure ulcer development. Critically ill patients represent a key patient population in which an unavoidable pressure ulcer may occur. The paradox is that pressure ulcer occurrence is defined by the National Quality Forum (CMS, 2006) as a “never event”, leaving caregivers in a challenging situation of trying to prevent a pressure ulcer that may not be realistically prevented. Continued research on pressure ulcer risk factors is imperative in this population, not only to decrease the incidence, but to validate the phenomenon of the unavoidable pressure ulcer in an effort to potentially influence health care policy.

This study contributed to the body of knowledge regarding pressure ulcer risk factors that confront the critically ill, however, more empirical evidence is needed to further validate these risk factors in the ICU population. Development of an ICU pressure ulcer risk assessment model or refinement of the Braden and Bergstrom model is warranted in order to appropriately and more fully explain pressure ulcer development in this population. This risk assessment model may then serve as the basis for the development of a risk assessment tool designed specifically to measure pressure ulcer risk in critical care patients.

### Recommendations

Based on the findings of this study, the following recommendations for future research are proposed:

1. Replication of the current study using multiple sites in order to improve the generalizability of the study findings.
2. Prospective study of the theoretical and empirical variables under investigation in this study to strengthen the validity of these risk factors.
3. Intervention research that tests the effects of various prevention strategies such as support surfaces, fecal containment devices, repositioning frequency, use of glide sheets and patient transfer equipment on pressure ulcer development in ICU patients.
4. Development and testing of a pressure ulcer risk prediction model for the critical care population.

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Englewood Hospital & Medical Center  
Institutional Review Board

**Request for Institutional Review Board Exemption From Review  
for Retrospective Review of Existing Records**

I request that the project listed below be granted exemption from IRB approval because:

- 1) The project is limited to already obtained documents, records, and data;
- 2) all records for the project will be kept in accordance with requirements for confidentiality so that participants (those whose data are being reviewed) cannot be identified directly or through identifiers linked to them.

09-

**EHMC Project # XR-350**

**Project Title: Predictors of Pressure Ulcer Development in Adult Critical Care Patients.**

**Project Objective(s):**

*No patient identifiers or facility identifiers will be recorded, collected or released.  
(See attached summary)*

**Site: Englewood Hospital and Medical Center**

**Principal Investigator: Jill Cox MS, RN, APRN-BC, CWOCN**

**Advisor: Charlotte Thomas-Hawkins PhD, RN, Associate Professor, Rutgers,  
College of Nursing, Rutgers University**

*[Signature]*

Signature Principal Investigator

*5/7/09*  
Date

**PROJECT GRANTED EXEMPTION**

Date:

*5-12-2009*

*[Signature]*

**Lawrence R. Krakoff, M.D.** *Lewis Attus, MD*

✓ **Chairman, Institutional Review Board**

Englewood Hospital & Medical Center  
Institutional Review Board

**SUMMARY FORM  
NEW PROJECT**

DATE: 5/09

EHMC Protocol # ~~09-350~~ <sup>XR</sup>

**Protocol Title:** Predictors of pressure ulcer development in adult critical care patients

Date of Protocol: 5/09

Sponsor: N/A

Food & Drug Administration Status: N/A

Remuneration: N/A

Financial/Proprietary Interest: N/A

**Principal Investigator:** Jill Cox, MS, RN, APRN-BC, CWOCN

**Advisor:** Charlotte Thomas-Hawkins PhD, RN, Associate Professor, College of Nursing, Rutgers, the State University of NJ

**Study Coordinator:** N/A

**SITE:** Englewood Hospital and Medical Center

**BRIEF DESCRIPTION OF PROTOCOL:** The intent of this study is to fill a gap in knowledge regarding the factors that pose the greatest risk for pressure ulcer development in adults in critical care units. Both theoretically-derived and empirically-derived risk factors will be examined in this study. Records will be reviewed of all patients admitted into the medical/surgical ICU between October 2008-March 2009. All data abstracted from the medical record will be de-identified on a data abstraction form in such a manner that subjects cannot be linked to information.

**AIM:** The purpose of this study is to identify which risk factors best predict pressure ulcer development in adult critical care patients. The intention is that this study will contribute to the body of knowledge regarding the full range of risk factors that significantly influence pressure ulcer development in this population.

**DESIGN:** Retrospective chart review using a descriptive, correlational design

Signature: Principal Investigator

Date: 5/7/09



**RUTGERS UNIVERSITY**  
**Office of Research and Sponsored Programs**  
**ASB III, 3 Rutgers Plaza, Cook Campus**  
**New Brunswick, NJ 08901**

June 4, 2009

P.I. Name: Cox  
 Protocol #: E09-546

Jill Cox  
 869 River Vale Road  
 River Vale NJ 07675

Dear Jill Cox:

**Notice of Exemption from IRB Review**

**Protocol Title:** "Predictors of Pressure Ulcer Development in Adult Critical Care Patients"

The project identified above has been approved for exemption under one of the six categories noted in 45 CFR 46, and as noted below:

**Exemption Date:** 5/28/2009                      **Exempt Category:** 4

This exemption is based on the following assumptions:

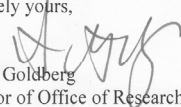
- **This Approval** - The research will be conducted according to the most recent version of the protocol that was submitted.
- **Reporting** - ORSP must be immediately informed of any injuries to subjects that occur and/or problems that arise, in the course of your research;
- **Modifications** - Any proposed changes MUST be submitted to the IRB as an amendment for review and approval prior to implementation;
- **Consent Form (s)** - Each person who signs a consent document will be given a copy of that document, if you are using such documents in your research. The Principal Investigator must retain all signed documents for at least three years after the conclusion of the research;

**Additional Notes:**                      **None**

**Failure to comply with these conditions will result in withdrawal of this approval.**

The Federalwide Assurance (FWA) number for Rutgers University IRB is FWA00003913; this number may be requested on funding applications or by collaborators.

Sincerely yours,

  
 Sheryl Goldberg  
 Director of Office of Research and Sponsored Programs  
[graser@orsp.rutgers.edu](mailto:graser@orsp.rutgers.edu)

cc: Charlotte Thomas-Hawkins



## Appendix C

## DATA ABSTRACTION RECORD

Age: \_\_\_\_\_ Gender: Male Female  
 Ethnicity: Caucasian Black/African American  
 Asian Hispanic Indian Other: \_\_\_\_\_

Date/Time of ICU adm. \_\_\_\_\_ Date/Time Discharge \_\_\_\_\_

Total # hours of ICU admission \_\_\_\_\_

Disposition at D/C: Med/Surg PCU SNF/Rehab Dead  
 Discharge

## ICU Admitting Diagnosis:

Respiratory Failure Hemodynamic Instability  
 Sepsis/Septic Shock Neuro-surgery Neuro-medicine  
 GI bleed GI surgery Vascular surgery  
 Multisystem Organ Failure Other: \_\_\_\_\_

## Comorbid Conditions

Vascular Disease (Cardiac or peripheral) YES NO

Diabetes Mellitus YES NO

Sepsis/Infection YES NO

APACHE SCORE ☐ (Record on attached page)

# hours MAP < 60 in 1<sup>st</sup> 48 hours of admission: \_\_\_\_\_

# hours systolic BP < 90 in 1<sup>st</sup> 48 hours of admission: \_\_\_\_\_

# hours diastolic BP < 60 in 1<sup>st</sup> 48 hours of admission: \_\_\_\_\_

## Braden Scale

Admission Braden Score: \_\_\_\_\_ Admission Subscale Scores:

Sensory Perception: \_\_\_\_\_

Moisture: \_\_\_\_\_

Activity: \_\_\_\_\_

Mobility: \_\_\_\_\_

Nutrition: \_\_\_\_\_

Friction/Shear: \_\_\_\_\_

## Vasopressor Use (If yes, see attached page)

YES NO Norepinephrine YES No Vasopressin YES No

Neosynephrine YES NO Epinephrine YES No Dopamine

Acquired Pressure  
 Ulcer: YES NO  
 Stage: I II III  
 IV DTI  
 Unstageable  
 Location: Sacrum  
 (CIRCLE)  
 Buttocks  
 Ischium  
 Trochanter  
 Heel Other: \_\_\_\_\_

# of Hours  
 Into  
 admission PU  
 developed:  
 \_\_\_\_\_

Specialty Bed:  
 Yes No  
 Fecal  
 Containment  
 Device:  
 Yes No

## Side Two: Operational Definitions

**Age:** Age of the patient on admission to the ICU

**Gender:** Male or Female

**Ethnicity:** Found in physician portal

**ICU LOS:** total number of hours the patient was in the ICU

**Disposition at discharge:** Circle the patient's disposition at d/c from MSICU

**ICU Admitting Diagnosis:** Circle the primary diagnosis of the pt at the time of admission to the ICU.

**Comorbid Conditions:** Identify if the patient has a PMH or current history of vascular disease (cardiac or peripheral), diabetes mellitus, sepsis/infection. Located in physician portal.

**APACHE:** see attached to record the data points for the APACHE Score. Clinical information found in the physician's portal and QS.

**Blood Pressure:** Record total # of hours MAP under 60, Systolic BP under 90 or diastolic BP under 60 for the first 48 hours of the ICU admission

**Braden Scale:**

Record the admission and each admission subscale score from QS

**Pressure Ulcer:**

Document no, if no PU developed after admission to the ICU

Document yes if the patient acquired a PU anytime after admission to the ICU

Circle Stage and location as recorded in QS on the day of patient discharge from the ICU

Document the number of hours into the admission that PU was discovered

**Specialty Bed:** indicate if a specialty bed was used for this patient during the ICU stay.

**Fecal Containment Device:** indicate if a fecal containment device was used during the ICU admission

**Vasopressor Use:**

If no vasopressor administered, circle no

If norepinephrine, epinephrine, neosynephrine, vasopressin or dopamine administer, circle YES and record on the following pages the amount of time of administration for each of the agents administered during the ICU stay.

## Norepinephrine

119

[illegible]

**Epinephrine or Phenylephrine (circle)**

[illegible]

## Vasopressin

[illegible]

## Dopamine

[illegible]

## Appendix D

Table 2. The APACHE II Severity

Physiologic Variable	of Disease Classification Systems									
	High Abnormal Range					Low Abnormal Range				
	+4	+3	+2	+1	0	+1	+2	+3	+4	Points
Temperature - rectal (°C)	≥105.8F°	102.1 to 105.7°		101.3 to 102°	96.8 to 101.2°			85.9 to 89.4°	≤85.8°	
Mean Arterial Pressure - mm Hg	≥160	130 to 159	110 to 129		70 to 109				≤49	
Heart Rate (ventricular response)	≥180	140 to 179	110 to 139		70 to 109			40 to 54	≤39	
Respiratory Rate (non-ventilated or ventilated)	≥50	35 to 49		25 to 34	12 to 24		6 to 9		≤5	
Oxygenation: A-aDO <sub>2</sub> or PaO <sub>2</sub> (mm Hg)	≥500	350 to 499	200 to 349		<200					
a. FIO <sub>2</sub> >0.5 record A-aDO <sub>2</sub> FIO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>								
b. FIO <sub>2</sub> <0.5 record PaO <sub>2</sub> FIO <sub>2</sub>					PO <sub>2</sub> >70			PO <sub>2</sub> 55 to 60	PO <sub>2</sub> <55	
Arterial pH (preferred)	≥7.7	7.6 to 7.69		7.5 to 7.59	7.33 to 7.49			7.25 to 7.32	7.15 to 7.24	<7.15
Serum HCO <sub>3</sub> (venous mEq/l)	≥52	41 to 51.9		32 to 40.9	22 to 31.9			18 to 21.9	15 to 17.9	<15
(not preferred, but may use if no ABGs)										
Serum Sodium (mEq/l)	≥180	160 to 179	155 to 159	150 to 154	130 to 149			120 to 129	111 to 119	≤110
Serum Potassium (mEq/l)	≥7	6 to 6.9		5.5 to 5.9	3.5 to 5.4			2.5 to 2.9		<2.5
Serum Creatinine (mg/dl) Double point score for acute renal failure	≥3.5	2 to 3.4	1.5 to 1.9		0.6 to 1.4			<0.6		
Hematocrit (%)	≥60		50 to 59.9	46 to 49.9	30 to 45.9			20 to 29.9		<20
White Blood Count (total/mm <sup>3</sup> ) (in 1000s)	≥40		20 to 39.9	15 to 19.9	3 to 14.9			1 to 2.9		<1
Glasgow Coma Score (GCS)										
Score = 15 minus actual GCS										
A. Total Acute Physiology Score (sum of 12 above points)										
B. Age points (years)	≤44 = 0;	45 to 54 = 2;	55 to 64 = 3;	65 to 74 = 5;	≥75 = 6					
C. Chronic Health Points (see below and reverse for definitions)										
<b>Total APACHE II Score</b> (add together the points from A+B+C)										

**Chronic Health Points:** If the patient has a history of severe organ system insufficiency or is immunocompromised as defined below, assign points as follows:  
 5 points for nonoperative or emergency postoperative patients  
 2 points for elective postoperative patients

**Definitions:** organ insufficiency or immunocompromised state must have been evident **prior** to this hospital admission and conform to the following criteria: Liver - biopsy proven cirrhosis and documented portal hypertension; episodes of past upper GI bleeding attributed to portal hypertension; or prior episodes of hepatic failure/encephalopathy/coma. **Cardiovascular** - New York Heart Association Class IV. **Respiratory** - Chronic restrictive, obstructive, or vascular disease resulting in severe exercise restriction (i.e., unable to climb stairs or perform household duties; or documented chronic hypoxia, hypercapnia, secondary polycythemia, severe pulmonary hypertension (>40 mmHg), or respirator dependency. **Renal** - receiving chronic dialysis. **Immunocompromised** - the patient has received therapy that suppresses resistance to infection (e.g., immunosuppression, chemotherapy, radiation, long term or recent high dose steroids, or has a disease that is sufficiently advanced to suppress resistance to infection, e.g., leukemia, lymphoma, AIDS).

Interpretation of Score

0 to 4 = ~4% death rate	10 to 14 = ~15% death rate	30 to 34 = ~75% death rate
5 to 9 = ~8% death rate	15 to 19 = ~25% death rate	> 34 = ~85% death rate

Temperature Conversion:  
4 points:  $\geq 105.8^{\circ}\text{F}$  ( $\geq 41.0^{\circ}\text{C}$ )

3 points: 102.1-105.7 (39-40.9 C)

1 point: 101.3-102 (38.5-38.9 C)

0 points: 96.8-101.2 (36-38.4 C)

1 point: 93.1-96.7 (34-35.9 C)

2 points: 89.5-93 (32-33.9 C)

3 points: 85.9-89.4 (30-31.9C)

4 points:  $\leq 85.8$  ( $\leq 29.9$ )

§ Adapted from Crit Care Med 1985; 13:818-829

## Appendix E

BRADEN SCALE FOR PREDICTING PRESSURE SORE RISK					
Patient's Name:	Evaluator's Name:	DATE OF ASSESSMENT:			
<b>Sensory perception</b> Patient's ability to feel pain and pressure-related discomfort	<b>1. Completely limited:</b> Unresponsive to painful stimuli, due to diminished level of consciousness or sedation. OR <b>2. Limited:</b> Limited ability to feel pain over most of body surface	<b>3. Slightly limited:</b> Responds to discomfort by moaning or turning. OR <b>4. No impairment:</b> Responds to discomfort by verbalizing. Has no sensory deficit which would limit ability to feel or voice pain or discomfort.			
<b>Moisture</b> Skin is exposed to moisture	<b>1. Constantly moist:</b> Skin is kept moist by incontinence, sweat, etc. Dampness is detected every time patient is moved or turned. <b>1. Bedfast:</b> Confined to bed.	<b>2. Moist:</b> Skin is often but not always moist. Must be changed at least once a shift. <b>2. Chairfast:</b> Ability to walk severely limited. Must be assisted into chair or wheelchair.	<b>4. Rarely moist:</b> Skin is usually dry. Linen requires changing only at routine intervals.		
<b>Activity</b> Level of physical activity	<b>1. Completely immobile:</b> Does not make any slight changes in body or extremity position without assistance.	<b>2. Very limited:</b> Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently.	<b>4. Walks frequently:</b> Walks independently at least once every day and inside room at least once every 2 hours during waking hours.		
<b>Mobility</b> Ability to change and control body position	<b>1. Very poor:</b> Never eats a complete meal. Eats 2 servings or less of protein (meat or dairy products) per day. OR <b>2. Poor:</b> Eats 3 or more servings of protein (meat or dairy products) per day. OR <b>3. Fair:</b> Eats 4 or more servings of protein (meat or dairy products) per day. OR <b>4. Good:</b> Eats 5 or more servings of protein (meat or dairy products) per day.	<b>2. Probably inadequate:</b> Rarely eats a complete meal. Eats 2 servings or less of protein (meat or dairy products) per day. OR <b>3. Adequate:</b> Eats 3 or more servings of protein (meat or dairy products) per day. OR <b>4. Excellent:</b> Eats 4 or more servings of protein (meat or dairy products) per day.	<b>4. No limitations:</b> Makes major and frequent changes in position without assistance.		
<b>Nutrition</b> Food intake pattern	<b>1. Very poor:</b> Never eats a complete meal. Eats 2 servings or less of protein (meat or dairy products) per day. OR <b>2. Poor:</b> Eats 3 or more servings of protein (meat or dairy products) per day. OR <b>3. Fair:</b> Eats 4 or more servings of protein (meat or dairy products) per day. OR <b>4. Good:</b> Eats 5 or more servings of protein (meat or dairy products) per day.	<b>2. Probably inadequate:</b> Rarely eats a complete meal. Eats 2 servings or less of protein (meat or dairy products) per day. OR <b>3. Adequate:</b> Eats 3 or more servings of protein (meat or dairy products) per day. OR <b>4. Excellent:</b> Eats 4 or more servings of protein (meat or dairy products) per day.	<b>4. Excellent:</b> Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and dairy products per day. Does not require supplementation.		
<b>Friction and Shear</b>	<b>1. Problem:</b> Requires moderate to maximum assistance in moving. OR <b>2. No problem:</b> Requires minimal assistance in moving. OR <b>3. No problem:</b> Requires no assistance in moving.	<b>2. Potential problem:</b> Moves feebly or requires minimum assistance. During a move, requires assistance to get up, down, or against sheets, chair, restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	<b>3. No apparent problem:</b> Moves in bed and in chair independently and completely during move. Maintains good position in bed or chair at all times.		
ADOPTED 1997			TOTAL SCORE:		

<sup>1</sup> Nothing by mouth

<sup>2</sup> Intravenous

<sup>3</sup> Total Parenteral Nutrition

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### Curriculum Vitae

1960: Born August 15, Englewood, New Jersey

1978: Graduated Bergenfield High School, Bergenfield, New Jersey

1978-1979: Attended Montclair State University

1982: Graduated, Englewood Hospital School of Nursing, Diploma Nursing

1988: Graduated, William Paterson University, BSN, 1988

1992: Graduated, Rutgers, The State University of NJ, MS in Nursing 1992

#### Principal Positions/Occupations

1982-1988: Staff Nurse, Englewood Hospital and Medical Center Englewood, NJ

1988-1992: Nurse Educator Staff Development, Englewood Hospital and Medical Center, Englewood, NJ

1992-present: Advanced Practice Nurse: Medical/Surgical and Wound Ostomy and Continence Nursing, Englewood Hospital and Medical Center, Englewood NJ

1992-1994: Adjunct clinical faculty: Bergen Community College, Paramus, NJ

1999-present: Wound/Ostomy Clinician, Valley Home Care, Ridgewood NJ

#### Publications

1995. *Med-Surg Nurse*, "Inflammatory Bowel Disease: Implications for the Medical Surgical Nurse." (CE feature article)

2007: Mosby's Nurses CE, Standards-Based Nursing Program "Wound Care: Assessment of Surgical Wounds"