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The Relationship Between Percent Deficit Ideal Body Weight and the Prevalence, Severity, and Healing of Pressure Ulcers

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THE RELATIONSHIP BETWEEN PERCENT DEFICIT IDEAL BODY WEIGHT AND THE PREVALENCE, SEVERITY, AND HEALING OF PRESSURE ULCERS

by

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DEDICATION

I would like to dedicate this thesis to my mother, Helen Rusyn, who has always provided me with the encouragement, love and support throughout my education and career in nutrition.
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GLOSSARY

Braden Skin Scale - a scale used to determine patients at risk for forming pressure ulcers. The scale is composed of six sub scales that reflect sensory perception, skin moisture, activity, mobility, friction, and nutrition status. Each sub scale is scored from one to four with the exception of friction which is scored one to three (Bergstrom, Braden, Laguzza & Holman, 1987).

Healing Rate - For the purpose of this study, healing rate is defined as the number of months to heal the pressure ulcer. This will be calculated as 100 divided by the number of months to heal the pressure ulcer that equals percent healing rate.


Incidence - measures the proportion of a group initially free of pressure ulcers that develop them over a given time (Frantz, 1997).

Percent Deficit of Ideal Body Weight - calculated as 100-[(actual weight divided by IBW) x 100]. Deficits are graded as mild (5-15%), moderate (15-30%), and severe (>30%) (Strauss & Margolis, 1996).

Pressure Ulcer - lesions caused by unrelieved pressure that results in underlying tissue damage (Bergstrom et al., 1994). Pressure ulcers are classified by the National Pressure Ulcer Advisory Panel (NPUAP) staging system which is as follows:

Stage I: Nonblanchable erythema of intact skin, the heralding lesion of skin ulceration. In individuals with darker skin, discoloration of the skin, warmth, edema, induration, or hardness may also be indicators.

Stage II: Partial-thickness skin loss involving epidermis, dermis, or both. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.

Stage III: Full-thickness skin loss involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue.

Stage IV: Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (e.g., tendon, joint capsule). Undermining and sinus tracts also may be associated with Stage IV pressure ulcers (Bergstrom et al., 1994).
Prevalence - measures the proportion of a group that has pressure ulcers at a given time. The time frame may be a single point in time for each person or a time period during which the proportion of cases that occur are counted (Frantz, 1997). For purposes of this study, prevalence will be referred to as the number of pressure ulcers per patient. Low prevalence of pressure ulcers were considered those patients who have two or less pressure ulcers and high prevalence were those patients who have three or more pressure ulcers.

Severity - For purposes of this study severity will be determined using pressure ulcer stages classified by the (NPUAP) staging system. Stage I or II pressure ulcers will be referred to as "superficial" and Stage III and IV pressure ulcers will be referred to as "severe".

Shearing – When outer layers of skin slide with rough or sticky surfaces, pulling and possibly tearing underlying tissues (Bergstrom et al., 1987).
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Abstract

THE RELATIONSHIP BETWEEN PERCENT DEFICIT IDEAL BODY WEIGHT AND THE PREVALENCE, SEVERITY, AND HEALING OF PRESSURE ULCERS

by Cheryl Anne Masters

This retrospective study determined whether percent deficit ideal body weight (IBW) was a risk factor for the prevalence, severity or healing of pressure ulcers. This study hypothesized that nursing home residents below their IBW with pressure ulcers have more severe, higher prevalence, and a slower healing rate than resident who are above their IBW. Medical record data were analyzed for all subjects that met the selection criteria from April, 1997 to April, 1998. Fifty-eight subjects aged 39 to 104 years were assigned to one of two groups (Group 1(n=28) were less than 99% of IBW; Group 2 (n=30) were greater than or equal to 99% of IBW). There was no correlation between percent deficit IBW and pressure ulcer prevalence ($r^2 = -0.017$) or between percent deficit IBW and healing rate ($r^2 = -0.010$). There was no association between percent deficit IBW and severity ($x^2 (N=58)=3.35; p=.06$). Black subjects (n=20) had a higher prevalence of pressure ulcers than white subjects ((n=38), $x^2 (N=58) = 4.634; p = .002$). Bedridden subjects (n=34) had slower healing rates than non-bedridden subjects (n=24),($F(58) = 2.38$, $p=.02$).
Chapter 1

Introduction

Pressure ulcers are a common occurrence and serious medical condition that affect all areas of health care. The Agency for Health Care Policy and Research (AHCPR) defined "pressure ulcers are any lesion caused by unrelieved pressure resulting in damage of underlying tissue" (Bergstrom et al., 1994). Enormous evidence is available regarding nutrition and its relationship to wound healing. In a review of the literature from 1943 to 1989, Breslow (1991) noted multiple prospective and cross-sectional studies that demonstrated people with pressure ulcers were malnourished. Also, Breslow noted several studies that demonstrated people who were malnourished and especially underweight were at risk for developing pressure ulcers. This study sought to determine whether percent deficit ideal body weight was related to the prevalence, severity, and healing of pressure ulcers.

Significance of the Problem

Pressure ulcers have been a significant problem for several reasons. First of all, pressure ulcers have occurred in every healthcare setting such as hospitals, nursing homes, spinal cord rehabilitation centers, hospice, home care, and in the community (Allman, 1997). Prevalence and incidence rates have been high. A national pressure ulcer prevalence survey of 177 hospitals showed that the prevalence of pressure ulcers was 11.1% (Meehan, 1994). In nursing home patients, incidence rates were between 17% to 35% and prevalence rates between 7% to 23% (Smith, 1995). According to the National
Hospital Discharge Survey, the prevalence of pressure ulcers have more than doubled over the past ten years (Stotts, 1997). Patients with pressure ulcers used more medical resources, required 50% more nursing time, had longer length of stays and had higher hospital costs (Krainski, 1992).

In order to prevent future pressure ulcers from occurring, the identification of risk factors associated with pressure ulcers have been developed. Such factors include immobility, incontinence, altered level of consciousness, and nutritional factors such as inadequate dietary intake and impaired nutrition status (Bergstrom et al., 1992). Patients who are bed or chair-bound or who are unable to reposition themselves are at greater risk when combined with the above risk factors.

Increased cost also has been associated with pressure ulcer treatment. In 1992, the estimated cost of pressure ulcer care in nursing homes were $355 million and $60 million in home care settings (Allman, 1997). Total cost for all settings was estimated at $1.3 billion (Allman, 1997). Additionally, patients with pressure ulcers were associated with longer lengths of stay and increased mortality and morbidity (Levine & Tortolos, 1995).

It is interesting to note that parallel problems exist with malnutrition as do pressure ulcers. First of all, the prevalence rate of malnutrition is high. According to Gallagher-Allred, Voss, Finn, and McCamish (1996), in studies involving more than 1,327 hospital patients, 40-55% were found to be malnourished or at risk for malnutrition, and up to 12% were severely malnourished. Consequently, one of the outcomes of malnutrition is poor wound healing and increased incidence of infection (Coats, Morgan, Bartolucci &
Malnutrition has been linked to increased length of hospital stay and increased costs (Chima et al., 1997). Also, malnourished patients have been associated with post-operative complications, and increased rates of morbidity and mortality (Velanovich, 1991). Therefore due to the existing similarities between pressure ulcers and malnutrition, their interrelationships are significant and worthy of further study.

**Objective**

The objective of this research was to determine whether a vital component of nutrition assessment, namely, percent deficit ideal body weight was an independent risk factor for the prevalence, severity, and healing of pressure ulcers.

**Statement of Purpose**

Overall concern for pressure ulcers has been evident in the health care field. The association of the relationship between nutrition and pressure ulcers has been clearly demonstrated (Breslow, 1991). Nutrition parameters used to evaluate nutrition status in persons with pressure ulcers have been identified. These often include albumin, total protein, transferrin, and total lymphocyte count that measure visceral protein stores. Somatic protein stores are measured by ideal body weight, total fat, body muscle stores, and creatinine height index. (Strauss & Margolis, 1996). Studies conducted (Allman et al., 1986; Berlowitz & Wilking, 1989; Breslow, 1991; Ek, Unosson, Larsson, Vonschenck, & Bjurulf, 1991; Moolten, 1972; Mulholland, Tui, Wright, Vinci, & Shafiroff, 1943) suggest that percent deficit ideal body weight could be a risk factor for the development of pressure ulcers and could have an impact on prevalence, severity, and
healing. Information sought in this study will provide insight into whether percent deficit ideal body weight is a risk factor for prevalence, severity, or healing of pressure ulcers.

**Hypothesis**

The hypotheses that were tested in this research were the following:

1. Subjects who are below their ideal body weight and have pressure ulcers will have a significantly higher prevalence of pressure ulcers than subjects who are above their ideal body weight and have pressure ulcers (p < .05).

2. The severity of pressure ulcers will be significantly higher in those subjects who are below their ideal body weight than those subjects who are above their ideal body weight (p < .05).

3. Subjects who are below their ideal body weight and have pressure ulcers will exhibit significantly slower healing rates than those subjects who are above their ideal body weight and have pressure ulcers (p < .05).

**Assumptions**

For purposes of this study it was assumed that:

1. Patients with pressure ulcers received the appropriate therapy and the most efficacious treatment for healing of the pressure ulcer, and if the pressure ulcer did not heal, therapy changes were made in order to aid healing.

2. Medical record data were accurate as related to number, stage, and size of the pressure ulcer, and body weight.
Limitations

The limitations relevant for interpreting this study were that:

1. Number of subjects was limited to those available within the study period.

2. Demographic distribution of subjects (gender, ethnicity, ages) were limited to those available within the study period.

3. Number and severity of pressure ulcers were not controlled and was limited to those present in subjects within the study period.

4. Actual number of pressure ulcers, number of months to heal, and severity were not measured by the researcher due to the retrospective nature of the study, rather the researcher relied on information present in the medical record. To ascertain the validity of the existing data, skin round notes in the medical record and nursing skin assessment sheets were cross-checked (See Methodology, p. 46).

To ascertain that the medical record data used for this retrospective study was accurate skin round notes were compared to the nursing skin assessment sheets. No discrepancies were identified in the records. If any medical data recording errors had been found, that medical record would have been eliminated.
Chapter 2

Review of the Literature

Conceptual Framework

Several conceptual frameworks exist in the literature as related to pressure ulcers. First, there is a brief discussion of two conceptual frameworks that are applied to pressure ulcer-related research but are not used here. Second, there is a discussion of the Braden and Bergstrom conceptual framework that was selected for this study.

The Web of Causation is a community-based framework for pressure ulcer development that includes socioeconomic factors and personal belief systems (Oot-Giromini, 1993). It included the following risk factors: mobility, activity, moisture, nutrition, friction and shear, and altered sensory perception.

The Web of Causation was first described by MacMahon and Pugh (1970). The Web of Causation used the concept in which the whole is greater than the sum of its parts. Socioeconomic factors included knowledge and ability of the caregivers, availability and affordability of equipment and services. Personal value factors were activities of daily living, medical conditions, coping abilities, attitudes, and desire to participate in health regimens. According to the Web of Causation, failure of any of these factors in the community would prevent the appropriate care from taking place and increase the risk of hospitalization (Oot-Giromini, 1993). The Web of Causation considered a holistic
approach, and was more specific for a community setting. Since the Web of Causation was designed for the community setting, it did not fit the framework of this study which is in a nursing home.

Another conceptual scheme that took a more systems approach is the Neuman Systems Model (Neuman, 1982). The Neuman Systems Model was a practical framework for terminally ill client care. This systems approach organized assessments and interventions that faced the client during a terminal illness. The aim of this model was to provide a structure that identifies the parts and their interrelationship for the whole client as a complete system.

The Neuman System Model was based on the conceptual framework of stress and the client's reaction to stressors. Stressors are forces that occur within the internal and external environment of the client (Lile, Pase, Hoffman, & Mace, 1994). These stressors can be identified or classified to possibilities. These stressors can be either intrapersonal, interpersonal or extrapersonal. As applied to the prevention of pressure ulcers, intrapersonal stressors are shearing force, limited mobility, age, dehydration, inadequate nutrition, altered thought process, and spiritual doubts. Interpersonal stressors consisted of cultural and family role conflicts, unskilled caregivers, and language barriers. Extrapersonal stressors were limited temporal, human and material resources and inability to meet financial obligations. It was the effects of these stressors which determined whether a pressure ulcer will develop. The goal was to prevent the invasion of these
stressors and to maintain stability of the client's system and reduce encounters with stressors and the degree of reaction.

In the Neuman System Model the patient was represented by a series of concentric circles surrounding the inner core (Neuman, 1989). The core structure was an inner circle that consisted of survival factors common to all organisms such as genetic makeup or strength of body organs. Next, was a series of concentric broken circles represented by internal lines of resistance. These were considered protective forces that encompass and protect the basic core structure such as an individual's immune system response or healing process. Next, a solid line that encircled the internal tones of resistance, represented the normal wellness level of the patient. This line adjusted over time to manage stressors so the inner basic core is protected. Next, a broken outer circle represented the flexible line of defense. This line was dynamic and was able to be altered over time. It protected the patient's normal state and prevented the influx of stressors. This flexible line of defense was strengthened by factors such as sleep, hydration, and nutrition that kept the system free from stressors (Lile et al., 1994).

According to Neuman (1982), each client's system consisted of a dynamic combination of five variables. These five variables were physiological, psychological, sociocultural, developmental and spiritual. These variables were considered concurrently in each circle. The relationship between these variables and the degree of the reaction of the client's system to these stressors was the foundation of this system.
Although the Neuman Systems Model was designed for the secondary prevention of pressure ulcers, its primary intrapersonal stressor was the advancement of terminal illness (Lile et al., 1994). Since the subjects of this study were not considered terminally ill, the Neuman Systems Model did not fit the framework of this study.

The Braden and Bergstrom (1987) conceptual schema was selected as the conceptual framework for this study. Although this schema had not been developed recently, it had been used as the conceptual framework for more recent studies (Jiricka, Ryan, Carvalo, & Bukvich, 1995; Prevost, 1992). Most importantly, it fit the conceptual framework of this study more closely than the others reviewed such as the Web of Causation (MacMahon & Pugh, 1970) and the Neuman Systems Model (Neuman, 1982).

The conceptual framework developed by Braden and Bergstrom (1987) was the theoretical framework for the organization of the critical components in the etiology of pressure ulcers. The following risk factors: age, nutrition, sensory perception, moisture, activity, mobility, friction and shear, arteriolar pressure, emotional stress, smoking, and skin temperature related to the development of pressure ulcers. According to Braden and Bergstrom (1987), the two prime determinants for the development of pressure ulcers were (1) intensity and duration of pressure, and (2) tissue tolerance of the skin tissue and its supporting structure. Tissue tolerance denoted the ability of the skin and its supporting structures to endure the effects of pressure with adverse consequences. Increased pressure on the surface of the skin was caused by immobility, decreased activity such as bed rest, and decreased ability to sense pain or pressure. Tissue tolerance of the skin
relates to extrinsic and intrinsic factors. Extrinsic factors are those factors that affected the surface of the skin and its ability to tolerate exposure. Exposure to moisture, friction, and shear were the three extrinsic factors. Intrinsic factors influenced the structure and integrity of the skin and its supporting structures that included nutrition, age, arteriolar pressure, and other hypothetical factors such as interstitial fluid flow, emotional stress, smoking, and skin temperature. Intrinsic factors included supporting structures which are the vascular and lymphatic systems that serve the skin and underlying structure.

**Terminology**

Skin ulceration due to pressure and shear are commonly referred to as decubitus ulcers, bed sores, ischemic ulcers, or pressure sores (Yarkony, 1994). These terms are used to describe a “pressure wound” (Margolis, 1995). A “pressure wound” was described in 1992 by the Wound Healing Society to denote the anatomic disruption of underlying subcutaneous fat, muscle, tendon, and/or bone or organ. This differed from the description of a “wound” which was described as a disruption of normal anatomic structure and function and did not include underlying tissue (Margolis, 1995). There is now consensus that the term “pressure ulcer” rather than decubitus ulcer or bed sore, pressure sore, or ischemic ulcer is the most appropriate term used to describe a pressure wound (Smith, 1995).

**Staging**

Pressure ulcer staging has been used to determine the severity of the ulcer. Over twenty years ago, Darrell Shea, an orthopedic surgeon at the University of Miami,
published a landmark paper describing a method of classifying and managing pressure ulcers (Shea, 1975). Shea's numeric classification system consisted of an orderly evolution of the pressure ulcer. Each pressure ulcer grade was defined by the anatomic limit of soft tissue damage that could be observed. Shea believed that all layers of soft tissue were involved in a Grade I pressure ulcer. However, in his pressure ulcer classification system, the clinical presentation of Grade I pressure damage was limited to the epidermis, the outer skin layer. This epidermal damage could range from soft tissue swelling, induration, heat and erythema of unbroken skin to moist, superficial ulceration. Grades II, III, and IV pressure ulcers gave anatomic limits of involved soft tissue based on Shea's understanding of the pathophysiology of soft tissue breakdown. Shea's original work had been modified (Eltorai & Chung, 1977; International Association for Enterostomal Therapy (IAET), 1987; National Pressure Ulcer Advisory Panel (NPUAP), 1989; Yarkony et al., 1990). Also, there has been a proliferation of many alternative pressure ulcer staging systems. In the literature some classification systems had from three (Blom, 1985; Morrison, 1984) to six (Jones & Millman, 1986; Lowthian, 1994; Yarkony et al., 1990) grades of pressure ulcers. Some systems began with a Grade 0 and others begin with a Grade I. One system used letters rather than numbers (Wallace, Sears, & Clark, 1980) to classify pressure ulcers and one used colors (Cuzzell, 1988). No one classification system has been universally accepted which has caused communication problems (Maklebust, 1995).
The Staging System developed by Shea (1975) was similar to those recommended by the National Pressure Ulcer Advisory Panel (NPUAP) Consensus Development Conference and has been cited more consistently than others (NPUAP, 1989). This staging system has been the one most commonly used and has been adopted by the Agency for Health Care Policy and Research (AHCPR) pressure ulcer guideline panels and has been published in both sets of clinical practice guidelines (Bergstrom et al., 1994).

**Reverse Staging**

Pressure ulcer staging is only appropriate for determining the maximum depth of tissue damage (NPUAP, 1995). When pressure ulcer staging systems are used to describe healing, it is assumed that full thickness pressure ulcers heal by replacing the same structural layers of body tissue that were lost. When Stage IV ulcers heal they do not replace lost muscle, fat and dermis before they re-epithelialize. Actually, the crater is replaced by granulation tissue composed of endothelial cells, fibroblasts, collagen, and extra cellular matrix. Therefore, Stage IV pressure ulcers cannot become Stage III, Stage II and/or Stage I ulcers.

The healing of a Stage IV pressure ulcer should be documented only by improvement in wound characteristics such as size, depth, amount of necrotic tissue, exudate, and amount of granulation tissue (NPUAP, 1995). Pressure ulcer staging should be used only to document the depth of tissue affected by the pressure ulcer and should never be used to describe the healing of an ulcer.
Indicators of Quality of Care

Pressure ulcers have come to indicate quality or non-quality health care. Regulatory agencies use pressure ulcers as an indicator of quality care given to patients. Pressure ulcers are affected by the quality of nursing care. Nursing homes with fewer staff and more rapid turnover have been shown to have a higher prevalence rate of pressure ulcers versus a nursing home with a more stable staff (Rudman, Slater, Richardson & Mattson, 1993). Consequently, quality improvement programs have included pressure ulcer staging as an indicator that needs to be measured and assessed. Furthermore, regulatory agencies can determine fines for licensed health care facilities based on the number of certain stages of pressure ulcers (Maklebust, 1995). In some settings, nursing home personnel have been rewarded for documenting that pressure ulcers are healing (Fairchild, 1992).

Functions of Staging Systems

Pressure ulcer staging has been used for several purposes. Topical treatment and pressure-reducing equipment are prescribed by standard protocols per pressure ulcers stage (Maklebust, 1995). Pressure ulcer staging has been marketed and used as a treatment guideline without consideration of other wound characteristics. As a result, some agencies use the same dressing protocol on every ulcer of the same stage. Medicare beneficiaries are provided specific levels of care based on the severity of the pressure ulcer (Maklebust, 1995).
Staging systems have also been used to determine reimbursement criteria (Maklebust, 1995). Third-party payers have used pressure ulcer staging systems as reimbursement criteria. Extended care facilities are paid a higher fee for patients with Stages III and IV pressure ulcers relative to patients with Stage I or Stage II ulcers.

Other purposes for pressure ulcer staging systems exist (Barr, 1993). These systems are used for inclusion criteria for research studies, as an assessment parameter, as reimbursement criteria, as a guide for product selection, as a marketing strategy for manufacturers, and as a method of evaluating response to pressure ulcer therapies.

Prevalence and Incidence

Data on the epidemiology of pressure ulcers showed the magnitude of the high prevalence of pressure ulcers. The Fourth National Pressure Ulcer Prevalence Survey was performed in 1995 that included 265 hospitals (Barczak, Barnett, Childs & Bosley, 1997). This study revealed that the prevalence rate among 39,874 patients ranged between 1.4% to 36.4%. Other studies revealed prevalence rates between 17% to 35% of patients had pressure ulcers upon admission to a nursing home (Smith, 1995). The sacrum and heels were the most common sites for occurrence in the above studies. Patients between the ages of 71 to 80 had 29% of the ulcers found. Female patients had 48% of the pressure ulcers and males comprised 47% with gender not being reported in 5% of the patients (Barczak et al., 1997). Seventy-four of the pressure ulcers were either Stage I or II. It has been reported that 65% of pressure ulcers are either Stage I or II (Smith, 1995). It was theorized that increased prevalence could be due to several reasons: (1) high patient
acuity, (2) longevity of the patient population, (3) reduction in clinical staff, (4) less attention to pressure ulcer prevention, (5) decreased quality of health care (Barczak et al., 1997). Lastly, Barczak et al. (1997) found the most frequently reported pressure ulcer stage among African Americans was Stage II (39%).

Prevalence rates among different settings are high. Prevalence rates in nursing homes ranged from 17.4-28%, 25-62% among spinal cord injury and rehabilitation centers, and 13.8-19% among persons in home care settings which included hospice patients (Allman, 1997). Prevalence rates ranged between .04% and .08% in the community.

Incidence rates suggested that 5.4% of all patients will develop a pressure ulcer during hospitalization (Allman, 1997). In nursing home populations, 28% of patients will develop a pressure ulcer within four weeks of admission and 10.9% will develop one within the first six months. Regional spinal cord rehabilitation centers reported that 40% of patients developed pressure ulcers during their initial hospitalizations. Incidence rates for community-based persons aged 55-75 was at least 1.8%.

Cost

Reported costs of treating pressure ulcers are staggering. A recent study was designed to assess the cost of managing pressure ulcers from their initial occurrence in long-term care through the entire course of treatment, including hospital treatment for complications (Xakellis & Frantz, 1996). The mean cost of treatment, including long-term care and hospital costs was $2,731 per ulcer. Excluding hospital costs, the mean cost of
treatments was $489 per ulcer. In a review of the literature, Day, Hayes, McAree-Kennedy, and Dierksen (1997) reported costs as high as $15,000 for the treatment of one ulcer. Less conservative estimates are between $30,000 to $86,000 to treat one pressure ulcer.

Pressure ulcer Medicare treatment costs were examined in different health care settings in 1992 (Allman, 1997). The mean hospital charge of patients with pressure ulcers as a primary diagnosis was $21,675 and $2,900 per case for physician charges. The total for 34,000 inpatients with a primary diagnosis of pressure ulcers was 836 million dollars. When pressure ulcers were a secondary diagnosis, an average of $10,986 in additional charges were attributed to pressure ulcers.

Other studies showed the more severe the ulcer, the higher the cost to treat. Frantz, Berquist and Specht (1995) showed the mean costs associated with ulcer treatment of a Stage I pressure ulcer was $1.55 per day and Stage II ulcer was $3.65 per day. Stage III and IV ulcers had higher average costs per day. The mean cost for treating Stage III ulcers was $4.46 per day and $6.03 for Stage IV ulcers.

Length of Stay

Pressure ulcers have been associated with extended lengths of stay. For example, patients who developed pressure ulcers were shown to have an average length of stay of two days longer than those who did not have pressure ulcers (Allman, 1997). The mean length of stay for hospitalized patients with pressure ulcers was nearly five times than that noted for other patients in another cross-sectional study (Allman et al., 1986).
Morbidity and Mortality

An increased risk of morbidity and mortality has been associated with pressure ulcers. Allman (1998) reported a mortality rate of 59.4% for those who developed pressure ulcers versus 38.1% for those who did not. Bergstrom and Braden (1992) found a 92% death rate in those patients who developed new pressure ulcers within six weeks after hospitalization and were three times likely to die than patients who did not develop pressure ulcers.

Co-morbidities associated with pressure ulcers included infections of the localized area which could lead to sepsis, cellulitis, and osteomyelitis (Yarkony, 1994). Thomas, Goode, Tarquine, and Allman (1996) hypothesized that pressure ulcers may not cause increased mortality directly, but rather death could be attributed to co-morbidities associated with the presence of pressure ulcers.

Pain

Pain was another common occurrence in people with pressure ulcers. One study reported that 59% of patients with pressure ulcers had some degree of pain (Dallam et al., 1995). One study reported 45.4% of patients had horrible pressure ulcer pain (Allman et al., 1987; Allman, 1997). The NPUAP (1995) stated, “pressure ulcers are a significant and increasing source of considerable human suffering.” In the Clinical Guideline for pressure ulcer treatment, pain was mentioned as an issue which needed further research (Dallam et al., 1995).
Risk Factors for the Development of Pressure Ulcers

The Agency for Health Care Policy and Research (AHCPR) assembled a multidisciplinary panel of experts to define early interventions for prevention and management of Stage I ulcers. The Clinical Practice Guidelines (Bergstrom et al., 1992) reflected the state of current knowledge regarding the effectiveness of policies designed to prevent pressure ulcers. It stated that "bed and chair-bound individuals or those with impaired ability to reposition should be assessed for additional factors that increased risk for developing pressure ulcers. These factors included immobility, incontinence, and nutritional factors such as inadequate dietary intake, impaired nutritional status, and altered level of consciousness."

Over 100 risk factors exist in the development for pressure ulcers (Day et al., 1997). Risk factors are needed to identify those at risk who would need timely intervention for prevention. According to Allman (1997), risk factors represented specific characteristics that predispose a person to the development of pressure ulcers. Immobility and inability to reposition oneself were the major factors related to pressure and friction on the skin. Being bedridden or confined to a chair increased the risk of developing pressure ulcers.

Factors associated with the development of pressure ulcers related to the susceptibility of the skin plus the presence of constant pressure or friction on the skin. Conditions which make the skin susceptible to pressure ulcers were urinary and fecal incontinence due to the presence of moisture which can cause skin maceration and edema.
The presence of feces was also an irritant. Skin susceptibility also was increased by diabetes. Diabetes could cause dehydration as well as circulation problems, both of which could increase the risk of pressure ulcers (Spector, 1994).

There were many other factors related to the risk of developing pressure ulcers. Some factors were identified in a review of the literature by Sparks (1993). More direct factors were sustained soft tissue pressure, shearing force, friction, and negative nitrogen balance. Indirect factors were incontinence, skin maceration, anemia, obesity, dehydration, altered mental status, and underlying disease. Reduced general resistance, decreased mobility, restraints, paralysis, loss of vascular tone, de-vitalization of deep tissue, steroids, radiation therapy, mattress type, and high friction bed coverings were other indirect factors.

Still other factors found were impaired nutrition status, decreased serum albumin, decreased blood pressure, age, increased temperature, sensory deficits, medications, smoking, activity, cultural influences, family support, length of stay, self-care abilities, moisture, and infections (Sparks, 1993). Additional factors included client beliefs, behavioral interventions, social competence, tissue stress and strain, and the relationship between nursing staff and the development of pressure ulcers. Dehydration, low body weight, Medicare payments, Black race, narcotic medications, insulin therapy, Parkinson's disease, and male gender were also found to be factors.
Demographics as Risk Factors for Pressure Ulcers

Age, sex, and race factors have been associated with pressure ulcers (Spector, 1994). Pressure ulcers have been a major problem among elderly persons. Older people lose subcutaneous tissue. As people age the epidermis thins and loses elasticity and there is a loss of subcutaneous tissue. Furthermore, the skin becomes drier in aging due to a reduction in sebaceous gland activity. Dry, scaling skin is a significant factor associated with the development of pressure ulcers (Allman, Goode, Patrick, Burst & Bartolucci, 1995). Being elderly is seen as a co-factor in impaired healing. According to Stotts and Wipke-Tevis (1996), healing is delayed not by age alone, but by many chronic diseases associated with impaired healing such as vascular disease.

Differences among males and females were also associated with the risk of pressure ulcers. Spector (1994) found males were at increased risk and hypothesized that they were more likely to develop pressure ulcers because they weighed more than women and therefore exerted greater pressure.

Although the data is inconsistent, Blacks and Latinos represent the fastest growing segment of the population with in the 85 years or more age group and are expected to increase in population within the next five decades (Lyder, 1996). Nonetheless, pressure ulcer prediction studies that include ethnic minority subjects are significantly lacking in the literature.

Race differences have been found in the literature. Previous studies found African-American patients to have the majority of Stage IV ulcers (Meehan, 1994). Spector
(1994) found being Black increased the likelihood of having pressure ulcers upon admission to a nursing home. Fuhrer, Garber and Rintala (1993) did find African-Americans had more severe ulcers than Caucasians. Bergstrom et al. (1992) stated that early stage sores may be more difficult to identify on people with dark skin. Carlson, King, Kirk, Temple and Heinemann (1992) did not find a relationship between race/ethnicity and occurrence of pressure ulcers during acute care, rehabilitation or early follow up. Fuhrer et al. (1993) also did not find a relationship between race/ethnicity and occurrence of an ulcer nor did they find a relationship between race/ethnicity and number of pressure ulcers on the day of exam.

**Risk Assessment Tools**

In order to prevent pressure ulcers from occurring it is important to identify those who are at risk. Risk assessment tools were developed as a way to efficiently identify those who may be at risk (Goodridge, 1993). The concept of a risk assessment tool for pressure ulcers was initially attributed to Norton, Exton-Smith, and McLaren (1962) with the publication of the Norton Scale. This tool assessed pressure ulcer risk using a numerical scoring system based on five criteria: physical condition, mental state, activity, mobility, and incontinence. Each criteria were assigned a numerical score from one to four based on the nurses’ clinical judgment. The higher the score the less likely the patient is to develop a pressure ulcer.

Although the Norton scale was developed specifically for the geriatric population, it soon became widely used for other populations (Goodridge, 1993). But, it did not
incorporate moisture, friction, shear, nutrition, and pain. Some criticized that these factors, which play a role in pressure ulcer development were not included (Jones & Millman, 1986).

In 1973, Gosnell developed a risk assessment scale. Basically, Gosnell modified the Norton scale to create the Gosnell Scale (Gosnell, 1989). The scale consisted of five categories. Each category was given a numerical score from one to five. The lower the score the higher the patient risk for developing pressure ulcers. The five categories were mental status, continence, mobility, activity, and nutrition. A nutrition category replaced Norton’s physical condition category and incontinence was renamed continence. Demographic data, medical diagnosis, admission and discharge data were added. Other clinical items included vital signs, height and weight, appearance of the skin, tone, sensation and medications. Research of this scale found that impaired mobility, impaired activity, altered nutrition status and altered mental status were strongly associated with pressure ulcer formation (Goodridge, 1993).

Other assessment scales were developed since the Norton and Gosnell Scales (Jones & Millman, 1986; Knoll, 1982; Lowthian, 1979; Pritchard, 1986). The components of these scales were very similar to the Norton and Gosnell Scales except for Lowthian (1979). All of these scales included a nutrition component. Pritchard (1986) included hemoglobin as part of the nutrition assessment. Knoll (1982) included oral fluid intake in addition to a nutrition component.
The Braden Scale was developed in 1987 (Bergstrom et al., 1987). It was developed from the conceptual framework for a study of pressure ulcers (Braden & Bergstrom, 1987). The Braden Scale consisted of six sub scales which include nutrition, mobility, activity, sensory perception, friction and shear, and skin moisture. Each area was scored on a scale of one to four, with a total possible score of 23 points. The higher the Braden Score, the lower the risk for pressure ulcer development. According to Goodridge (1993), the Braden Scale incorporated variables regarded as key contributors to pressure ulcer formation such as moisture, friction, and shear. Only the Braden and Norton Scales have been tested extensively for reliability (Bergman-Evans, Cuddigan & Bergstrom, 1994). The Braden Scale demonstrated fairly good reliability, although no data has been reported for the Norton Scale (Goodridge, 1993).

Physiology of Skin Breakdown

In order for skin to break down, pressure, friction, shear, and moisture are the four factors that must occur (Remsberg & Bennett, 1997). Pressure is the major force that results in the formation of pressure ulcers. Pressure applied directly against the skin can lead to tissue damage through a series of events. Hypoxia, acidosis, and hemorrhage into the interstitium can occur due to compromised blood flow. Cell death can occur as a result of accumulation of toxic cellular waste. Tissue necrosis causes an inflammatory response which can cause further damage. The skin over bony prominences are areas most vulnerable to damage. Pressure ulcers usually occur on the sacrum, trochanter, isheal spine, and heels.
Friction and shear are the other two factors which are necessary for the development of pressure ulcers (Remsberg & Bennett, 1997). Friction occurs when skin is moved across a surface such as sliding a patient across a mattress, has the head of the bed elevated, or is reclining in a chair. These opposing forces can also result in shear. Shearing can occur when outer layers of the skin slide with rough surfaces that can pull and tear underlying tissue (Bergstrom et al. 1987). The other factor necessary for the development of pressure ulcers is moisture.

Excess moisture leads to maceration and rashes, thereby weakening the natural barrier of the outer layer of the skin (Bergstrom et al. 1987). Diarrhea and incontinence, two risk factors for the development of pressure ulcers are related to moisture (Remsberg & Bennett, 1997). Another factor which can contribute to skin breakdown is age. In the elderly, anatomic skin changes such as decreased elasticity, collagen strength, and subcutaneous adiposity, as well as cellular deficits such as decreased fibroblast replication are assumed to be contributors (Remsberg & Bennett, 1997).

Pressure Relieving Strategies and Devices

Pressure relieving strategies have been used to treat and prevent pressure ulcers for centuries (Levine & Tortolos, 1995). The foundation for the prevention and treatment of pressure ulcers is turning and positioning (Remsberg & Bennett, 1997). The AHCPR guidelines provide a description to implement this strategy (Bergstrom et al. 1994).

The accepted standard for turning and repositioning immobile patients is every two hours (Remsberg & Bennett, 1997). Although turning and positioning have been
generally included in the pressure ulcer prevention and treatment, there are very few published reports suggesting this technique is effective.

All pressure-relieving strategies are based on reducing or eliminating tissue pressure (Remsberg & Bennett, 1997). Most pressure-relieving devices are designed to conform to the contours of the body so the pressure can be distributed over a large surface rather than concentrated in a specific area. These devices are constructed of foam and filled with gel, foam, air, water, or polyfill. Other products include pads, cushions, mattress overlays, special mattresses and specialty beds. Padding, cushions, and most mattress overlays are static devices so when the patient is not moving the tissue pressure is constant. Other products such as specialty beds, and some mattress overlays are dynamic. When the patient is not moving, the bed alternates pressure over the body surface.

Pressure Ulcer Care

Care of Stage I and II pressure ulcers. Local wound care included wound cleansing, debridement and dressings (Goode & Thomas, 1997). Stage I pressure ulcers appear as intact skin with discoloration, an induration or nonblanchable erythema. No treatments have been shown to improve the outcome of these lesions. Stage I pressure ulcers were a signal that the patient was at high risk for further skin breakdown.

Stage II pressure ulcers are shallow, usually not infected, and have very little necrotic tissue (Goode & Thomas, 1997). They typically heal in weeks with little or no scarring except for pigment changes. Debridement for Stage II pressure ulcers such as by sharp, mechanical, or enzymatic means are not necessary. Wound cleansing for Stage II
pressure ulcers is usually accomplished by pouring saline over the wound. Mechanical scrubbing should not be performed since it causes friction to the skin. Dressings are applied to keep the wound moist. Moisture has been shown to enhance healing, decrease scarring, and decrease wound pain. Dressings for Stage II pressure ulcers include hydrocolloid, semi-permeable foam dressings, and polyurethane film. Hydrocolloid dressings are adhesive, moldable, carbohydrate based materials that form a gel. These dressings provide a physiologic environment, reduce pain and improve healing (Goode & Thomas, 1997).

Foam dressings are made from sponge-like polymers and vary in drainage absorbing capacity (Goode & Thomas, 1997). Polyurethane film dressings are semi-permeable, transparent, and adhesive. They allow moisture to leave the wound but prevent bacteria and other contaminants from entering.

Care of Stage III and IV pressure ulcers. Debridement is necessary for Stage III and IV pressure ulcers (Goode & Thomas, 1997). Debridement is necessary to remove devitalized tissue. Devitalized tissue promotes the growth of bacteria and increases the risk of infection. Debridement methods can be classified as autolytic, mechanical, enzymatic, or sharp. Autolytic debridement consists of covering the pressure ulcer with a synthetic dressing such as transparent adhesive, hydrocolloid gel or semi-permeable foam. Tissue macrophages, neutrophils, and enzymes present in the tissue fluid remove bacteria and devitalized tissue (Goode & Thomas, 1997).
Mechanical debridement consists of several methods such as wound irrigation, scrubbing, and wet-to-dry-dressings (Goode & Thomas, 1997). Wet-to-dry dressings are the most popular. A moist dressing is applied to the pressure ulcer. As the dressing dries it adheres to the pressure ulcer. As the dressing is removed, it also removes both viable and necrotic tissue. This type of debridement can be painful and should be used when large amounts of necrotic tissue are present (Goode & Thomas, 1997).

Enzymatic debridement is the process of applying enzymes to remove devitalized tissue (Goode & Thomas, 1997). Dressings for Stage III and IV pressure ulcers depend on the characteristics of each wound. The condition of the wound bed, amount of necrotic tissue, and amount of drainage are factors to consider in selecting a dressing. The standard dressing for Stage III and IV is a saline-moistened gauze. The gauze should be kept continuously moist. Antiseptics and skin cleansers should not be used to moisten wound packing due to the presence of cytotoxic chemicals (Goode & Thomas, 1997).

Assessment of Healing

The assessment of wound healing is one of the most important principles of pressure ulcer management (Bergstrom et al. 1994). Wound healing is the natural repair of injured tissue through a series of molecular and cellular events (Ferrell, 1997). Assessment of this process is difficult in clinical settings. In order to assess the effectiveness of treatment and to maximize healing rates, accurate documentation of progress is essential. A designated system to monitor daily changes has not been established (NPUAP, 1995).
Few tissues in the human being have the capacity to regenerate (Ferrell, 1997). Pressure ulcers are replaced by scar tissue, contraction of the wounds, and epithelialization. Scar tissue, extracellular collagen, and epithelialization restores skin functions such as temperature loss, fluid loss, and barriers to infection. However, scar tissue does not replace tissue layers and anatomic structures. Scar formation often results in a decrease of skin elasticity and contractures. Decreasing inflammation, wound contraction, and maximum resistance to shear may take a long time to return. Therefore, even though complete healing is defined by the establishment of an epithelial covering, molecular and cellular processes continue for many months (Ferrell, 1997).

Methods for Estimating Wound Size

The most common method of evaluating wound healing is usually done by measuring wound size over time (Ferrell, 1997). Wound size can be an indicator of severity and prognosis. Linear measurements such as diameter or circumference are accomplished by a ruler estimating the size of the opening of a wound. Surface area can then be determined by estimating the product of the two diameters taken at right angles.

Other ways to assess healing over time are used by clinicians (Ferrell, 1997). Describing wound characteristics such as increasing granulation tissue, closure of sinus tract, and undermining are some observable signs. Serial photographs are used to capture qualitative changes over time.
Assessment Scales for Wound Healing

Assessment scales have been developed in order to assess wound healing. The Sessing Scale is an observational seven-point scale that describes wound healing (Ferrell, Artinian, & Sessing, 1995). A number is assigned to the description that most closely matches the pressure ulcer. Changes in pressure ulcers are calculated by the change in the numerical value over time. The Sessing Scale has been shown to be reliable and valid and even more predictive of healing rates than the initial size.

The pressure ulcer status tool is another scale used to evaluate healing. It consists of 13 separate domains which includes a description of size, depth, wound edges, undermining, necrotic tissue type and amount, exudate type and amount, surrounding skin color, peripheral tissue edema and induration, granulation tissue, and epithelialization (Bates-Jentsen, Vredevoe & Brecht, 1992). The content validity of this method has been reported, although is still under investigation (Ferrell, 1997).

Healing Rates

Very little data exists to validate expected healing rates (Ferrell, 1997). The average healing rate for Stage II pressure ulcers was 9 millimeters per day for those on a low air-loss bed and 3.2 millimeters per day for those on a foam mattress (Ferrell, Osterweil & Christenson, 1993). For Stage III and IV pressure ulcers, the healing rate was 10 millimeters per day on a low air-loss bed and less than 1 millimeter per day on a foam mattress. Generalized healing rates have not yet been developed (Ferrell, 1997).
The Process of Wound Healing

The wound healing process consists of three phases: (a) inflammatory phase, (b) proliferative phase, and (c) remodeling phase (Kiy, 1997; Utley, 1992). The inflammatory phase is a natural reaction to any acute trauma or surgical incision (Gogia, 1992; Kiy, 1997; Utley, 1992). It begins immediately after injury and continues up to six days. The first to arrive at the inflammatory site are neutrophils. Neutrophils engulf injurious agents and kill bacteria. Monocytes arrive after neutrophils, but they differ from neutrophils because they survive longer and ingest larger particles. Macrophages arrive last to ingest, neutralize, and destroy harmful substances and ingest cellular debris caused by death of host cells (Gogia, 1992; Kiy, 1997; Utley, 1992).

The proliferative phase is the second stage of wound healing. It occurs at the first week of injury up to three weeks (Gogia, 1992; Kiy, 1997; Utley, 1992). In this stage epithelialization and angiogenesis occurs. Epithelialization has been defined as a process in which epithelial cells proliferate and form a protective covering to cover the wound. Angiogenesis is the growth of new blood vessels which makes possible the growth of granulation tissue. Granulation tissue is the new tissue formed as soft tissue repairs. Granulation tissue is made of connective tissue cells, in growing young vessels, and collagen. The wound is strengthened by the cross-linking of granulation tissue and collagen which forms the scar (Gogia, 1991; Kiy, 1997; Utley, 1992).

The remodeling phase begins during the second or third week and can last up to two years (Gogia, 1992; Kiy, 1997). Scar tissue remolds by softening, flattening, and
strengthening its tissues. Contraction occurs by the action of myofibroblasts. Their function is to pull wound edges inward through a contractile motion into the wound’s interior.

**Nutrition and Wound Healing**

Several nutrients are factors in the wound healing process. Protein, albumin, carbohydrate, fat, vitamin A, C, B-complex vitamins, vitamin K, iron, zinc, and copper have all been shown to affect the wound healing process (Konstantinides & Lehmann, 1993).

Improved wound healing also has been shown in patients receiving total parenteral nutrition (TPN) (Albina, 1994). Wound healing is an anabolic process although if adequate calories are not provided, protein catabolism and cell deterioration can occur (Kiy, 1997). Furthermore, protein will be broken down into glucose (Utley, 1992). Underfeeding can cause medical problems and should be avoided. Barbul and Purtill (1994) reported that providing 50% of estimated calorie needs could result in decreased tissue granulation and protein deposition in rats. On the other hand obesity is more of a significant risk factor for wound-related complications than malnutrition (Albina, 1993).

Carbohydrates provide the energy required for white blood cell function (Utley, 1992) and are necessary for anti-inflammation to occur (Kiy, 1997). Glucose is required for fibroblastic migration, phagocytic activity, and cellular proliferation to occur (Brylinsky, 1995; Kiy, 1997). Therefore, if inadequate carbohydrates are provided muscle wasting, loss of subcutaneous adipose tissue, and poor wound healing could result.
Protein is vital to skin integrity. Low protein intakes can cause a decreased inflammatory response (Kiy, 1997). Decreased protein synthesis can cause proliferation, remodeling collagen synthesis, and lymphocyte formation to be impaired (Stotts & Wipke-Tevis, 1996; Utley, 1992). Both decreased calories and protein intakes were found to be correlated with decreased healing (Breslow, Hallfrisch, Guy, Crawley & Goldberg, 1993).

Several amino acids have been found to be crucial to the wound healing process (Brylinsky, 1995; Kiy, 1997). In animals, methionine reduces the inflammatory stage and accelerates the rate of fibroplasia. Methionine converts to cystine which is a co-factor for collagen synthesis. Histidine has been found to increase wound strength. Arginine has been shown to enhance immune activity especially T-cell function (Kirk et al., 1993). Cystine plays an important role as a co-factor in enzyme processes especially for collagen formation. Glutamine plays a role to decrease protein catabolism, although supplemental glutamine has not been shown to have noticeable effects on wound healing (Thomas, 1996). Arginine enhances collagen deposition in healthy volunteers, but no studies have been done on patients with pressure ulcers.

The impairment of wound healing has been associated with fat deficiency (Brylinsky, 1995). Fats are a concentrated energy source. They are also a crucial component of cell membranes and prostaglandins. Furthermore, essential fatty acid requirements are known to increase after injury. Deficiencies in essential fatty acids are shown to impair wound healing.
The omega-3 fatty acids play a role in wound healing by affecting the inflammatory response (Stotts, 1997). The primary omega-3 fatty acid, linoleic acid is metabolized to arachadonic acid which is a precursor of leukotrienes that modulate the inflammation and immune response.

Vitamins, Minerals and Wound Healing

Vitamin C is one of the vitamins most closely associated with wound healing. Vitamin C is required for collagen synthesis (Brylinsky, 1995). Levels of vitamin C rise in healing tissue, then return to normal after healing which is indicative of the healing process (Mazotta, 1994). A deficiency of vitamin C has been associated with delayed wound healing and breakdown of already healed wounds (Mazotta, 1994). The stress associated with wound healing requires an increased need for vitamin C. Wound healing is directly affected by the oxidation of ascorbic acid. Vitamin C also facilitates leukocyte formation which increases the resistance to infection. Megadoses (one to two grams per day) of vitamin C accelerated collagen formation even in those patients with normal levels of vitamin C (Barbul & Purtill, 1994; Mazotta, 1994; Thomas, 1996). According to Thomas (1996) these studies are controversial.

Vitamin A deficiency has been associated with delayed wound healing (Thomas, 1996). Roles of vitamin A in wound healing included facilitating wound debridement, stimulating fibroblast proliferation, increasing wound strength, and increasing susceptibility to infection. Vitamin A exhibits anti-steroid activity and has been shown to counteract delayed healing in patients on corticosteroids (Thomas, 1996). Vitamin A
deficiency could cause inadequate inflammatory response, while an excess has been shown to cause excess inflammatory response (Stotts & Wipke-Tevis, 1996). Vitamin A deficiency is correlated with decreased collagen synthesis, decreased epithelialization, and increased incidence of wound infection.

Zinc is the most well known mineral in wound healing (Barbul & Purtill, 1994). Zinc is a co-factor for more than 70 enzymes, many of which are critical to wound healing. Zinc deficiency has been associated with decreased fibroblast proliferation, collagen synthesis, impaired wound strength, and delayed epithelialization. Although zinc has been implicated in wound healing, no study has shown improved wound healing in patients who were not zinc deficient and supplemented with zinc (Thomas, 1996).

Iron is essential for wound healing (Mazotta, 1994). Iron is related to collagen formation and cellular respiration. Iron is essential for carrying hemoglobin. Both hemoglobin and iron have been shown to be related to the development of pressure ulcers (Breslow, 1991). Iron deficiency impairs the proliferation of cells involved with healing and wound debridement (Mazotta, 1994). Iron deficiency is related to decreased wound strength and increased incidence of infection.

Other components in wound healing are B-complex vitamins, copper, and vitamin K. All of these are necessary for collagen formation (Brylinsky, 1995). Deficiency of B-complex vitamins decreases resistance to infection. Pyridoxine, thiamine and riboflavin contribute to wound healing through antibody formation and to promote enzyme activity needed for the metabolism of protein, carbohydrate, and fat. Copper is needed for
collagen and erythrocyte formation. Vitamin K is necessary for normal coagulation and a
deficiency can lead to wound infection.

Vitamin E deficiency has not been shown to play a role in wound healing although
it does have inflammatory action. The role of vitamin E in wound healing is controversial
(Mazotta, 1994; Thomas, 1996).

Biochemical Markers and Wound Healing

Several studies have associated hypoalbuminemia with the development of
pressure ulcers (Breslow, 1991; Breslow & Bergstrom, 1994; Ek et al., 1991; Finucane,
1995). Hypoalbuminemia has also been linked to increased morbidity and mortality
(Brylinsky, 1995; Telfer & Moy, 1993). It has been theorized that albumin itself is not the
direct factor in wound healing, rather, hypoalbuminemia causes interstitial edema which
interferes with the exchange of nutrients at the cellular level (Brylinsky, 1995; Utley,

Another indicator of visceral protein stores is serum transferrin (Flanigan, 1997). Serum
transferrin responds sooner than albumin to indicate a change in protein status.
Serum transferrin has a shorter half-life of eight to ten days, compared to albumin which
has a half-life of approximately 21 days.

Total lymphocyte count is a visceral protein status indicator which has been shown
to be decreased in patients with pressure ulcers (Breslow, 1991). Total lymphocyte count
is also an indicator of humoral and cell-mediated immunity. Malnutrition is one factor that
contributes to the decreased responsiveness of the immune system.
Serum cholesterol can indicate malnutrition if below 150 milligrams per deciliter. Lower serum cholesterol has been associated with pressure ulcer severity (Liu, Spungen, Fink, Losada & Bauman, 1996) and pressure ulcer risk (Trumbore et al., 1990).

**Malnutrition and Pressure Ulcers**

Malnutrition is defined as a state of nutritional insufficiency attributable to either inadequate dietary intake or defective assimilation or use of food ingested (Bergstrom et al., 1994). Malnutrition is diagnosed if serum albumin level is less than 3.5 g/dl, total lymphocyte count is less than 1800 mm, or body weight has involuntarily decreased more than 15% (Bergstrom et al., 1994). These and other factors such as total protein, transferrin, ideal body weight, total body fat, body muscle stores, and creatinine height index can be used to diagnose malnutrition and have been used to evaluate nutrition status in patients with pressure ulcers (Strauss & Margolis, 1996).

Significant advances have been made in understanding the relationship between nutrition and wound healing. Malnutrition is frequently unrecognized. “The skeleton in the hospital closet” has been the term commonly used to describe unrecognized and untreated malnutrition in hospitals (Coats et al., 1993). Strauss and Margolis (1996) reported the prevalence of malnutrition is as high as 50% in some long-term care facilities. Further, protein malnutrition was detected in 25% of hospital patients and 50% of general surgical patients (Mazotta, 1994). Other studies reported rates of malnutrition between 40 to 59% (Chima et al., 1997). Gallagher-Allred et al. (1996) reported malnourished surgical patients were two to three times more likely to have minor and major
complications as well as increased mortality. Also, their length of stay can be extended up to 90% compared to well nourished patients. According to Osak (1993), increased postoperative morbidity and mortality have been shown in the malnourished patient. Furthermore, hospital costs can be 35% to 75% higher for malnourished patients.

**Malnutrition as a Risk Factor for Pressure Ulcers**

Malnutrition has been shown to be a risk factor for the development of pressure ulcers. Malnutrition delays wound healing, decreases immunocompetence, increases risk of infection, causes longer hospital stays, and increases morbidity and mortality (Mazotta, 1994). Poor nutritional status has been shown to significantly decrease healing time in patients with deep pressure ulcers (Brylinsky, 1995). In a review of the literature from 1943 to 1989, dietary intake of protein, calories, body weight, total protein, albumin, hemoglobin, total lymphocyte count, and iron have been associated with the development of pressure ulcers (Breslow, 1991). Difficulty feeding oneself was associated with the development of pressure ulcers by Brandeis, Ooi, Hossain, Morris, and Lipsitz (1994). Ek et al. (1991) found decreased albumin, decreased weight, and decreased triceps skinfold as risk factors for the development of pressure ulcers. Breslow & Bergstrom (1994) concluded that cross-sectional studies reveal that consuming inadequate energy and protein, being underweight, having low triceps skinfold measurement, and having low serum albumin levels were associated with having pressure ulcers. Prospective studies show consuming inadequate energy and protein, poor Braden Scale scores, and low
albumin levels to be associated with the development of pressure ulcers (Breslow & Bergstrom, 1994).

In spite of Breslow's (1991) study that found decreased albumin and decreased hemoglobin are associated with pressure ulcer risk some studies did not show albumin to be associated with pressure ulcers (Bergstrom & Braden, 1992; Thomas, 1997). Decreased total lymphocyte count and anemia have not been shown to be correlated with the development of pressure ulcers (Berlowitz & Wilking, 1989). Nor has total calories, vitamins C and A, zinc and iron been shown to predict the development of pressure ulcers (Bergstrom & Braden, 1992). Percent ideal body weight, body mass index, triceps skinfold thickness, and mid-arm circumference also have not been shown to predict pressure ulcers in patients (Bergstrom & Braden, 1992).

**Body Weight as a Risk Factor for the Development of Pressure Ulcers**

To identify risk factors for the development of pressure ulcers, Berlowitz and Wilking (1989) reviewed medical records of 301 admissions to a chronic care hospital over a 13-month period. It was found that the 100 patients who developed pressure ulcers weighed significantly less than patients without pressure ulcers. Others factors associated with the development of pressure ulcers included low hemoglobin level, lower serum protein level, lower serum albumin level, and impaired dietary intake. Non-nutritional factors included altered level of consciousness and being bed-ridden or chair bound. Weight index in percent was found to be a risk factor for the development of pressure ulcers by Ek et al. (1991). Five hundred one patients newly admitted to a long-
term care unit were observed. A significant difference was found in weight index in percent in patients who developed pressure ulcers from those who did not. Weight index in percent is determined from actual weight/reference weight x 100. Reference weight = Female (0.65 x ht - 40.4) and Male (0.80 x ht - 62.0). Therefore, these findings illustrate that weight and weight index in percent is associated with the development of pressure ulcers.

Bergstrom and Braden (1992) performed a prospective study of 200 nursing home patients at risk of developing pressure ulcers. The purpose of the study was to determine if dietary intake, nutritional status, or other physical markers are risk factors for the development of pressure ulcers. It was found that patients who developed Stage I pressure ulcers had lower percentage of ideal body weight, triceps skinfold, and body mass index than patients who did not develop pressure ulcers. Lower dietary protein and calorie intake were also significantly associated.

Allman et al. (1995), performed a prospective study to identify nutrition and other characteristics as risk factors for Stage II or greater pressure ulcers among patients whose activity was limited to bed or chair. Decreased body weight, depleted triceps skinfold, and decreased lymphocyte count were significantly associated with pressure ulcer development. Non-nutritional factors included previous history of pressure ulcers, immobility, and fecal incontinence. This study concluded that decreased body weight was an independent and significant risk factor for pressure ulcers in hospitalized patients whose activity was limited to a bed or chair.
Decreased body mass index was hypothesized to be a risk factor for the development of pressure ulcers in a Veterans hospital (Liu et al., 1996). Forty-eight male quadriplegics who developed pressure ulcers were compared to those quadriplegics without pressure ulcers and to non-quadriplegic, non-pressure ulcer controls. It was found that the pressure ulcer quadriplegic group had significantly lower body mass index than the control group and the non-pressure ulcer quadriplegic group. Metabolic rate measured as resting energy expenditure (REE) was significantly higher in the quadriplegics with pressure ulcers versus the quadriplegics without pressure ulcers or the healthy non-spinal cord injured controls. Therefore, this study illustrated that quadriplegics with pressure ulcers had lower body mass index and higher resting energy expenditures. In summary, cross-sectional studies have associated the risk of developing pressure ulcers with being underweight. Conversely, body weight has not been shown to be correlated with the risk of pressure ulcers (Bergstrom & Braden, 1992; Brandeis, Morris, Nash & Lipsitz, 1990; Inman, Sibbald, Rutledge & Clark, 1993).

Pressure Ulcer Prevalence and Percent Deficit Ideal Body Weight

Several cross-sectional studies have associated a relationship between the prevalence of pressure ulcers and percent deficit ideal body weight. Ideal body weight is one factor commonly used to diagnose malnutrition. Spector (1994) was the first to study the correlation of pressure ulcers for a nationally representative sample of 2,803 nursing home residents. He estimated the contribution of resident health characteristics to the probability of having had a pressure ulcer during a nursing home stay. Findings indicate
that being underweight increased the likelihood of having had a pressure ulcer during the stay. Other factors included inability to feed oneself, Parkinson's disease, paraplegia, diabetes, older age, male, unable to walk, cognitively impaired, frequent fecal and urinary incontinence, and being admitted from a hospital. Spector (1994) hypothesized that underweight persons are at risk due to the fact that they have susceptible bony prominences and underweight may be accompanied by malnutrition.

Another cross-sectional study included 232 nursing home patients who were surveyed to assess nutritional status and determine the prevalence of pressure ulcers (Pinchofsky-Devin & Kaminsky, 1986). Of the 17 patients who had pressure ulcers, all were considered severely malnourished. Of those patients, 70.5% had visceral protein deficits, 5.8% had somatic protein, and 23.5% had mixed protein deficits. Somatic protein deficit was determined by percent IBW (Metropolitan Life Insurance Co., 1983). This study showed that although the majority of patients had visceral protein deficits, some had somatic or mixed deficits.

The relationship between the prevalence of pressure ulcers and body weight was demonstrated by Allman et al. (1986). A cross-sectional survey with 634 patients was done to determine the prevalence of pressure ulcers. The study demonstrated that patients with pressure ulcers weighed significantly less than patients without pressure ulcers or those at risk of developing pressure ulcers. Hypoalbuminemia was also significantly correlated.
In a prospective study of wound dressing and nutritional support of 52 patients with pressure ulcers, 73% of the patients were severely nutritionally depleted (Gorse & Messner, 1987). The degree of nutritional depletion was determined using a point system equally weighing serum albumin, total lymphocyte count, and body weight. Body weight was positively correlated with the prevalence of pressure ulcers.

**Pressure Ulcer Severity and Percent Deficit Ideal Body Weight**

There were even fewer studies that demonstrated a relationship between severity of pressure ulcers and ideal body weight. In a retrospective study, Moolten (1972) reviewed the charts of 50 chronic hospital patients with pressure ulcers. Nutritional status of patients with deep pressure ulcers was worse than patients with superficial ulcers. Superficial ulcers were defined as those involving only the skin, and deep ulcers as extending more deeply and requiring extensive therapy. Fifty-two percent of patients with deep ulcers and 32% of patients with superficial ulcers were underweight. Other nutrition factors shown to be correlated with severity were hypoalbuminemia and lower hemoglobin levels.

Breslow, Halfrisch and Goldberg (1991) compared the nutritional status of 14 tube-fed nursing home patients with pressure ulcers to 12 tube-fed patient controls without pressure ulcers. Calorie and protein intakes were higher in patients than in patient controls. Despite higher levels of calorie and protein intake by patient subjects than patient controls, biochemical measures of nutrition status were worse in the study subjects. Pressure ulcer surface area was positively correlated with calorie intake per
kilogram of body weight per day and negatively correlated with body mass index. Serum albumin was lower in patients than controls as was hemoglobin level. Patients with Stage IV pressure ulcers had lower serum cholesterol levels than those patients with Stage II/III ulcers. Thus, studies associated severity of pressure ulcers with calorie intake, albumin, and hemoglobin levels, and negatively correlated with body weight as related to body mass index.

**Pressure Ulcer Healing and Percent Deficit Ideal Body Weight**

Evidence related to the correlation of healing of pressure ulcers is restricted to one study. Mulholland et al. (1943) hypothesized that hypoproteinemia caused pressure ulcers. It was found that pressure ulcer healing was accompanied by increased in body weight, serum albumin, and total protein. The relationship between healing of pressure ulcers and body weight was negatively associated by Henderson, Trumbore and Mobaran (1992). To date, there are no other published reports replicating these observations.

**Implications for the Present Study**

Pressure ulcers are a major concern in hospital patients and nursing homes. The role of nutrition in contributing to pressure ulcer risk has been examined in many studies. Nearly all sources of information reviewed indicate that malnutrition is a major factor contributing to the risk of pressure ulcers. Narrowing down the nutritional risk factors to identify malnutrition could provide clinicians with a quick and easy method to diagnose malnutrition and consequently the risk of developing pressure ulcers. Percent deficit ideal
body weight is an inexpensive nutritional marker readily available in clinical settings and could be used to identify patients at risk of developing pressure ulcers.

Overall, few studies have examined whether ideal body weight is related to the healing, prevalence, or severity of pressure ulcers. Since 1943, only nine studies made a correlation between patients who were underweight with pressure ulcer prevalence or the development of pressure ulcers (Allman et al., 1986; Allman et al., 1995; Bergstron & Braden, 1992; Berlowitz & Wilking, 1989; Ek et al., 1991; Gorse & Messner, 1987; Liu et al., 1996; Pinchofsky-Devin & Kaminsky, 1986; Spector, 1994). Moreover, only two studies revealed that weight is related to pressure ulcer severity (Breslow et al., 1991; Moolten, 1972). Equally important, only one study showed a healing response after weight started to increase (Mulholland et al., 1943). If percent deficit ideal body weight is identified as a risk factor for prevalence, severity, and healing of pressure ulcers, then measurement of percent deficit ideal body weight could be a valuable and inexpensive indicator of nutrition status and provide information about pressure ulcer prevalence, severity, and healing.
Chapter 3

Methodology

Setting

The setting for this study was the Veterans Affairs Medical Center Nursing Home in Lake City, Florida. This hospital-based facility consisted of a 180-bed extended care area, a 60-bed intermediate care area, and a 30-bed dementia special care unit. The average length of stay among patients admitted to the extended care area and the dementia special care unit was approximately six months and the intermediate care area was approximately 30 days.

Design

This research was retrospective and correlational, and utilized medical record data on all subjects who had pressure ulcers during the 12-month time frame from April 1997 to April 1998. Based on retrospective medical record review, all subjects who were included in the study were assigned to one of two groups. Group 1 were those who were less than 99% of IBW, and Group 2 were those who were greater than or equal to 99% of IBW.
Subject Selection

All patients who resided in the long-term care area and had pressure ulcers within the 12-month time frame and met the selection criteria were included in the study. Fifty-eight patients met the selection criteria and were included in the study.

Selection Criteria

In order to be selected for this study, the subjects must have met the following inclusion/exclusion criteria.

Inclusion Criteria.
1. Had a pressure ulcer within the 12-month time frame of the study.
2. Were considered an inpatient during the time of the study.

Exclusion Criteria.
1. Subjects who expired prior to the healing of the pressure ulcer.
2. Subjects who were discharged from the nursing home with pressure ulcers.
3. Subjects who had surgery which closed the wound or amputation.
4. Subjects whose medical records were unavailable.
5. Discrepancies in the medical record concerning weight, number of pressure ulcers, number of months to heal, or severity. (No discrepancies were identified.)

To ascertain that the data used for this retrospective study were correct, skin round notes and nursing skin assessment sheets were compared. No discrepancies concerning the number of pressure ulcers, number of months to heal and severity were found in records of the 58 subjects. No subjects were excluded based on criteria (5).

Protection of Human Rights

Permission to perform this study was granted by the Lake City VA Research Committee.

At the University of North Florida this study was categorized by IRB as exempt according to the Code of Federal Regulation Number (45 CRF 46.101(b)): and is a Category 4.
Instruments

Data was collected and recorded on the data collection tool. The form used to record the data was the VA Form 7051d Data Sheet published by the U.S. Government printing office 1987, 181-822/56909. It consisted of a wide column on the left followed by a series of numbered columns from 1-31. The tool allowed for 19 subjects per page.

Entered in the left column were the subjects' last name and the last four digits of their social security number. Entered in the following columns were: weight, ideal body weight, percent ideal body weight, number of pressure ulcers, number of superficial pressure ulcers, number of severe pressure ulcers, number of months to heal the superficial pressure ulcers, number of months to heal the severe pressure ulcers, diagnosis, treatment, nutrient supplementation, if bedridden (indicated by a yes or no), diabetes (indicated by yes or no), body mass index, age, race, sex, albumin, and cholesterol.

Data Collection Procedures

Medical records were requested and reviewed. All data was obtained from the medical record. The initial nutritional assessment at the onset of the pressure ulcer was used to obtain the weight, ideal body weight, percent ideal body weight, body mass index, nutrition supplementation, age, and sex. Albumin and cholesterol levels were obtained from the laboratory section of the chart. The number, stage, and number of months to heal the pressure ulcer was obtained from the nurses' skin assessment notes. The diagnosis, if diabetes was present, whether or not the patient was bedridden, and race were obtained from the history and physical performed by the medical doctor upon
admission. The type of treatment the subject was receiving for the pressure ulcers was obtained from the weekly skin round notes.

Prevalence was defined as the number of pressure ulcers per patient. "Low prevalence" was considered those patients with two or less pressure ulcers and "high prevalence" was considered those patients with three or more pressure ulcers. Severity of pressure ulcers was determined by using a pressure ulcer staging system (NPUAP, 1989). Severity was classified as either "superficial" or "severe". Two or less superficial and zero severe pressure ulcers were considered superficial for statistical purposes. Three or more superficial pressure ulcers and one or more severe pressure ulcers were considered severe for statistical purposes. Stage I and II pressure ulcers which involved only the skin were classified as superficial and pressure ulcers that were Stages III and IV and extended more deeply and involved the subcutaneous tissue were classified as severe. "Healing rate" was determined in order to compare whether it took longer for the superficial or severe pressure ulcers to heal.

Healing rate was calculated as the number of superficial or severe pressure ulcers divided by the number of months to heal. This was calculated as 100 divided by the number of months to heal the pressure ulcer which equaled the healing rate percentage. Therefore, the higher the healing rate percentage, the faster the pressure ulcers healed. If the subject had both a superficial and severe pressure ulcer then the higher of the two healing rates would be considered the healing rate for the subject. Superficial pressure ulcers which have a high prevalence (>3) were considered severe for statistical purposes.
Independent and Dependent Variables

The dependent variables were prevalence, severity, and healing rates. The dependent variables were compared between patients who were either above or below their ideal body weight. From this, we categorized the subjects into two groups: Group 1 were subjects less than 99% of IBW and Group 2 were subjects greater than or equal to 99% of IBW. Group 1 and Group 2 were the independent variables throughout the study.

Statistical Analysis

After the data was recorded in the data collection tool, it was statistically analyzed by the SAS System using the following:

Hypothesis 1. Subjects who are below their ideal body weight range and have pressure ulcers will have a significantly higher prevalence of pressure ulcers than subjects who are above their ideal body weight and have pressure ulcers. Hypothesis Number 1 was tested by:

1. A Spearman Correlation Coefficient test was used to determine whether there was a significant statistical correlation between prevalence of pressure ulcers and ideal body weight.

2. Prevalence was also measured by a standard Chi-square test that determined whether a measure of association existed between Group 1 or Group 2.

Hypothesis 2. The severity of pressure ulcers will be significantly higher in those subjects who are below their ideal body weight than those subjects who are above their ideal body weight and have pressure ulcers. Hypothesis Number 2 was tested by:
1. The relationship between severity and ideal body weight was analyzed by a standard Chi-square test to determine if a measure of association existed between Group 1 or Group 2.

**Hypothesis 3.** Subjects who are below their ideal body weight and have pressure ulcers will exhibit significantly slower healing rates than subjects who are above their ideal body weight and have pressure ulcers. Hypothesis Number 3 was tested by:

1. A Spearman correlation coefficient test was used to determine if there was a statistical significant correlation between healing rate of pressure ulcers and ideal body weight.

2. A $t$-test was used to determine whether or not a significant difference existed between the mean heal rates of subjects in Group 1 and Group 2.

The chi-square test was used to determine if any associations exist between the independent variable (ideal body weight) or dependent variables (prevalence, severity, healing rate) and the following: cholesterol, albumin, bedridden, diabetes, age, nutrition supplementation, body mass index, or race.
Chapter 4

Results

All subjects with pressure ulcers from April 1997 to April 1998 were screened for inclusion in the study. Of the 64 subjects screened, 58 of the charts reviewed were included in the study. Reasons for exclusion were: (a) one subject had a Stage IV pressure ulcer which resulted in a below the knee amputation; (b) one subject had surgery which closed the wound; (c) three subjects expired prior to the healing of the pressure ulcer, and (d) one subject’s medical record was unavailable for review.

Fifty-eight subjects met the selection criteria and were included in the study. Of the 58 subjects, there were 28 (48%) subjects in Group 1 and 30 (52%) in Group 2.

Prevalence

Table 1 shows the pressure ulcer prevalence between groups. There were a total of 123 pressure ulcers. There were 61 (49.5%) pressure ulcers in Group 1 and 62 (50.5%) in Group 2. Each subject in Group 1 had an average of 2.3 ($M=2.3$, $SD=2.3$), ($n=28$) pressure ulcers per subject, and Group 2 had an average of 1.9 ($M=1.9$, $SD=1.5$), ($n=30$) pressure ulcers per subject. Of the 58 subjects, 42 subjects had less than three pressure ulcers and 16 subjects had less than or equal to three pressure ulcers. Of those with less than three pressure ulcers, there were an average of 1.4 ($M=1.4$, $SD=0.5$), ($n=19$) pressure ulcers in Group 1 and 1.3 ($M=1.3$, $SD=0.4$), ($n=23$) pressure ulcers per
subject in Group 2. Of those subjects who had greater than or equal to three pressure ulcers, each subject in Group 1 had an average of 3.1 (M=3.1, SD=0.4), (n=9) pressure ulcers and Group 2 had an average of 4.1 (M=4.1, SD=1.8), (n=7) pressure ulcers per subject. Of the 123 pressure ulcers, one Black subject had 13 (10.5%) of the 123 pressure ulcers. Of the 12 subjects who had severe pressure ulcers, 3 (25%) had more than one severe pressure ulcer.

Severity

Table 1 shows the pressure ulcer severity between groups. Of the 123 pressure ulcers, 105 (85%) were considered superficial (Stages I or II), and 18 (15%) were considered severe (Stage III or IV). In Group 1 subjects had an average of 2.6 (M=2.6, SD=4.2), (n=28) superficial pressure ulcers. In Group 2 subjects had an average of 1.8 (M=1.8, SD=1.2), (n=30) superficial pressure ulcers. Subjects in Group 1 had an average of 0.5 (M=0.5, SD=0.8), (n=28) severe pressure ulcers. In Group 2 subjects had an average of 0.2 (M=0.2, SD=0.5), (n=28) severe pressure ulcers.

Demographic Data

Table 2 shows the demographic data between groups. Of the 58 subjects, 56 (97%) subjects were male and 2 (3%) were female. There were 27 (48%) males in Group 1 and 29 males in Group 2 (52%). There was 1 (50%) female subject in Group 1 and 1 (50%) female subject in Group 2.

Age was defined in three groups: (a) those less than 65, (b) between 65 and 75, and (c) those over 75 years old. Age range was between 39 and 104. Mean age for both
groups were 74 years old ($M=74$, $SD=11.14$), $(N=58)$. Twelve (21%) of the subjects were less than 65. Of those less than 65, 5 (42%) were in Group 1 and 7 (58%) were in Group 2. Seventeen (29%) subjects were between 65-75. Of those 65-75, 8 (47%) were in Group 1 and 9 (53%) were in Group 2. Twenty-nine (50%) subjects were over 75. Of those greater than 75, 15 (52%) were in Group 1 and 19 (66%) were in Group 2.

There were 37 White, 20 Black, and 1 Asian subject for the entire group. For statistical purposes the Asian subject was included in the White group. Of the 20 Black subjects, 10 (50%) were in Group 1 and 10 (50%) were in Group 2. Of the 38 White subjects, 18 (47%) were in Group 1 and 20 (53%) were in Group 2.

**Clinical Data**

Table 3 shows the characteristics of the study population between groups. Of the 58 subjects, 14 (24%) had dementia. Seven (50%) demented subjects were in Group 1 and 7 (50%) were in Group 2. Of the 58 subjects, 17 (29%) had diabetes. Five (29%) diabetics were in Group 1 and 12 (71%) were in Group 2. Of the 58 subjects, 34 (59%) were bedridden. Twenty-one (62%) bedridden subjects were in Group 1 and 13 (38%) were in Group 2. Eighteen (31%) subjects had cerebral vascular accidents (CVA) with hemiparesis. Of those who had CVA’s, 10 (56%) were in Group 1 and 8 (44%) were in Group 2. Of the 58 subjects, 9 (16%) had urinary tract infections (UTI). Six (67%) UTI subjects were in Group 1 and 3 (33%) were in Group 2. Of the 58 subjects, there were 5 (9%) quadriplegics. Five (100%) quadriplegics were in Group 2. Of the 58 subjects, 31 (53%) were receiving nutrition supplementation. Of those receiving nutrition
supplementation, 19 (61%) subjects were in Group 1 and 12 (39%) subjects were in Group 2. Of those who were receiving nutrition supplementation, 13 (42%) were tube-fed. Of those who were tube-fed, 9 (69%) were in Group 1 and 4 (31%) were in Group 2.

Biochemical Data

Table 3 shows the biochemical data between groups. Albumin was defined as either low (≤3.0 g/dl) or high (>3.0 g/dl). Albumin levels ranged from 1.8 to 4.0 dl. Mean albumin level for both groups were 3.25 g/dl (N=58), (M=3.25, SD=0.43). Of the 58 subjects, 18 (31%) subjects had albumin levels less than or equal to 3.0 g/dl. Ten (56%) subjects were in Group 1 and 8 (44%) subjects were in Group 2. Forty (69%) subjects had albumin levels over 3.0 g/dl. Eighteen (45%) subjects with albumin levels greater than 3.0 g/dl were in Group 1 and 22 (55%) were in Group 2.

Cholesterol was defined as either low (<170 mg/dl) or high (>170 mg/dl). Cholesterol levels ranged from 95 to 271 mg/dl. Mean cholesterol level for both groups was 159 mg/dl (N=58), (M=159, SD=38.81). Of the 58 subjects, 41 (71%) subjects had cholesterol levels less than 170 mg/dl. Of those with cholesterol levels less than 170 mg/dl, 22 (54%) were in Group 1 and 19 (46%) were in Group 2. Seventeen (29%) subjects had cholesterol levels greater than or equal to 170 mg/dl. Of those, 6 with cholesterol greater than or equal to 170 mg/dl (35%) were in Group 1 and 11 (65%) were in Group 2.
Anthropometric Data

Table 4 shows the anthropometric data between groups. Mean weight in pounds of subjects in Group 1 and Group 2 was 152.6 (N=58), (M=152.6, SD=33.51). Mean IBW for subjects in Group 1 and Group 2 was 100.3% (M=100.3%, SD=18.45), (N=58).

Mean weight for Group 1 was 133.9 pounds (M=133.9, SD=21.0), (n=28). Mean weight for Group 2 was 170.1 pounds (M=170.1, SD=33.8), (n=30). Mean IBW for Group 1 was 157.1 pounds (M=157.1, SD=19.1), (n=28). Mean IBW for Group 2 was 148.8 pounds (M=148.8, SD=24.0), (n=30).

Group 1 weighed an average of 23.2 pounds below their IBW. Group 2 weighed an average of 21.3 pounds above their IBW. Body Mass Index was defined as either low (≤25) or high (>25). Body Mass Index ranged from 13.9 to 34.4. Mean BMI for the entire group was 22 (M=22, SD=5.03), (N=58). Of the 58 subjects, 44 (76%) subjects had a BMI of less than or equal to 25 and 14 (24%) subjects had a BMI of over 25. The mean BMI for Group 1 was 19 (M=19, SD=2.6), (n=28) and the mean BMI for Group 2 was 25.3 (M=25.3, SD=4.9), (n=30).

Hypotheses Results

Table 5 shows the hypotheses results.

1. Ideal body weight and pressure ulcer prevalence. There was no association or correlation, respectively, between IBW and prevalence: $\chi^2 (N=58) = 0.563$, $p = .45$; $r = -0.017$, $p=0.89 (N=58)$. Therefore, this study did not show a relationship between prevalence or number of pressure ulcers and ideal body weight. Out of the 28 subjects who were in
Group 1, 19 (68%) had a low number of pressure ulcers (<2) and 9 (32%) had a high (≥3) number of pressure ulcers. In Group 2, 23 (77%) had a low (<2) number of pressure ulcers and 7 (23%) had a high (≥3) number of pressure ulcers. Table 6 shows the chi-square results. There was no correlation.

2. Ideal body weight and pressure ulcer severity. There was no association between IBW and severity: $\chi^2 (N=58) = 3.35$, $p = .06$ (Table 5). Of the 58 subjects, 22 (38%) had severe (>0 severe and/or >2 superficial) pressure ulcers. Fourteen (64%) were in Group 1 and 8 (36%) were in Group 2. Out of the 58 subjects, 36 (62%) had superficial (<2 superischials) pressure ulcers. Fourteen (39%) were in Group 1 and 22 (61%) were in Group 2. Although a higher percentage of those in the severe category were less than their IBW, the results were not statistically significant. Table 7 shows the chi-square results. These results suggest that there was no association between severity and percent deficit ideal body weight.

3. Ideal body weight and pressure ulcer healing rate. The combined mean healing rate of both groups were 1.06 pressure ulcers per month ($M=1.06$, $SD=0.78$), ($N=58$). There was no correlation or significant difference between IBW and healing rate, respectively ($r^2 = -0.010$, $p = .94$), ($N=58$); $t(58) = 0.209$, $p = .83$. The mean healing rate of Group 1 was ($M=1.08$, $SD = 0.91$), ($n=28$). The mean healing rate for Group 2 was ($M = 1.03$, $SD = 0.65$), ($n=30$). Table 8 shows the chi-square results. There was no correlation.

Significant Results of the Factors Associated with Independent or Dependent Variables
Table 9 shows the significant results of the factors associated with independent or dependent variables.

1. **Pressure ulcer prevalence and race.** There was an association between pressure ulcer prevalence and race: \( \chi^2 (N=58) = 4.634, p=.03 \). Of the 58 subjects, there were a total of 42 (72\%) subjects in the low prevalence group, and of those, 31 (74\%) subjects were White and 11 (26\%) subjects were Black. The high prevalence group was more evenly distributed. There were 16 (28\%) subjects in the high prevalence group, and of those, 9 (56\%) subjects were Black and 7 (44\%) were White. With regard to the low prevalence group, White subjects made up a much higher percentage (74\%) than Black subjects (26\%). Table 10 shows the chi-square results. Therefore, White subjects had a lower prevalence than Black subjects.

2. **Pressure ulcer severity and race.** An association existed between severity and race. There was a significant difference between the number of Black and White subjects in the severe compared to the non-severe group: \( \chi^2 (N=58) = 9.501, p=.002 \). Of the 58 subjects, 36 (62\%) subjects were in the non-severe group (0 severe; \( \leq 2 \) supercfls). Out of the 36 subjects, 29 (81\%) were White and 7 (19\%) were Black. In the severe group (>0 severe; \( \geq 3 \) supercfls), 13 (59\%) subjects were Black and 9 (41\%) were White. Table 11 shows the chi-square results. Therefore, Blacks comprised a greater percentage (59\%) of the severe cases than non-severe cases and are associated.

3. **Pressure ulcer healing rate and bedridden.** Mean healing rate was significantly higher in subjects non-bedridden than those who were bedridden (t(58)=2.38, p=.02). Of the
58 subjects, 24 (41%) were non-bedridden and 34 (59%) were bedridden. Non-bedridden subjects had a mean healing rate of 1.34 (M=1.34, SD = .928), (N=58) pressure ulcers per month which was greater than the bedridden subjects who had a mean healing rate of 0.86 (M=0.86, SD=.863), (N=58) pressure ulcers per month. Table 12 shows the chi-square results. This is a significant difference. Bedridden subjects had a slower healing rate.

4. Ideal body weight and bedridden subjects. There was an association between IBW and bedridden patients: $\chi^2 (N=58) = 8.883, p = .003$. Of the 58 subjects, 34 (59%) of the subjects were bedridden and 24 (41%) were non-bedridden. In Group 1, 22 (79%) were bedridden and 6 (21%) were non-bedridden. In Group 2, 12 (40%) were bedridden and 18 (60%) were non-bedridden. Table 13 shows the chi-square results. Therefore, a higher percentage of underweight subjects were bedridden. There was an association.

Non-Significant Associations

Table 14 shows the non-significant associations found to be present among various factors and the independent or dependent variables.
Acceptance or Rejection of Hypotheses

Hypothesis 1

Subjects who are below their ideal body weight and have pressure ulcers will have a significantly higher prevalence of pressure ulcers than subjects who are above their ideal body weight and have pressure ulcers.

Result: Subjects below their ideal body weight with pressure ulcers did not have a significantly higher prevalence of pressure ulcers than subjects above their ideal body weight. Therefore, the hypothesis is rejected.

Hypothesis 2

The severity of pressure ulcers will be significantly higher in those subjects who are below their ideal body weight than those subjects who are above their ideal body weight.

Result: Severity of pressure ulcers for subjects below their ideal body weight was not significantly higher than the severity of pressure ulcers in subjects above their ideal body weight. Therefore the hypothesis is rejected.

Hypothesis 3

Subjects who are below their ideal body weight and have pressure ulcers will exhibit significantly slower healing rates than those subjects at or above ideal body weight and have pressure ulcers.

Result: There was no significant difference between the mean healing rates of subjects who are below ideal body weight and have pressure ulcers than subjects at or above their ideal body weight and have pressure ulcers. Therefore the hypothesis is rejected.
Chapter 5

Discussion

This study sought to determine if percent deficit ideal body weight affected the prevalence, severity, or healing of pressure ulcers. The study hypotheses are:

1. Subjects who are below their ideal body weight and have pressure ulcers will have a significantly higher prevalence of pressure ulcers than subjects who are above their ideal body weight and have pressure ulcers.

2. The severity of pressure ulcers will be significantly higher in those subjects who are below their ideal body weight than those subjects who are above their ideal body weight.

3. Subjects who are below their ideal body weight and have pressure ulcers will exhibit significantly slower healing rates than those subjects who are above their ideal body weight and have pressure ulcers.

Although the relationship between nutrition status and prevalence, healing, or severity of pressure ulcers has been found in several studies, few studies have shown that percent deficit ideal body weight is related to the prevalence, severity, and healing of pressure ulcers. For that reason, this topic was chosen as the hypotheses for this study. Past research has associated a relationship between the prevalence of pressure ulcers and percent deficit ideal body weight (Allman et al., 1986; Pinchofsky-Devin & Kaminski,
ideal body weight has been associated with the severity of the development of pressure ulcers. This research has further suggested that malnutrition is a major risk factor which contributes to the development of pressure ulcers.

This study attempted to identify a specific indicator of malnutrition, that is, percent deficit ideal body weight as a risk factor for the development of pressure ulcers. Additional factors such as albumin, cholesterol, BMI, and nutrition supplementation have been shown to be risk factors for the development of pressure ulcers (Bergstrom & Braden, 1992; Ek et al., 1991; Liu, 1994). Due to these relationships, the above characteristics were compared to the independent and dependent variables to observe if any significant associations exist.

These study results suggest that there were no relationships that exist between percent deficit ideal body weight and pressure ulcer prevalence, severity or healing. These study outcomes may have been affected by several limitations. First, the sample size was small. Fifty-eight subjects were included in the study. The under-representation of female subjects was another limitation to this study. Ninety-eight percent of the study population was male. There were two women and fifty-six men.

Another limitation was that there were very few severe pressure ulcers. One hundred twenty-three pressure ulcers were tracked with 105 superficial and 18 severe. Since there were few severe pressure ulcers and insufficient diversity of healing rates in Groups 1 and 2 there was insufficient data to measure significance.

Due to lack of prior data concerning IBW and pressure ulcers, this study used <99% IBW for Group 1 and ≥99% IBW for Group 2. For example, patients who were
99% of IBW were classified in the below IBW group. Likewise patients who were
greater than or equal to 99% of IBW were in the above IBW group (new grouping
recommendations are discussed on page 70).

There was no association between IBW and prevalence. The association between
IBW and prevalence was measured by chi-square analysis and Spearman correlation
coefficient analysis \( (X^2 = 0.563; p = .45); (r^2 = -0.017; p = .89) \). These tests were
appropriate since there were two groups of body weight (<99% and ≥99%) and two
groups of prevalence (<3 and ≥3 pressure ulcers). There was no significant difference
between the number of subjects with a high number of pressure ulcers (≥3) in Group 1
\( (n=9) \) than Group 2 \( (n=7) \), therefore, no association or correlation exists.

Lack of association may have been influenced by the number of subjects with a
high number of pressure ulcers. Out of 58 subjects, only 16 had a high prevalence (≥3) of
pressure ulcers. Of those, nine subjects were in Group 1 and seven were in Group 2.
These results disagreed with several cross-sectional studies that documented an
association between prevalence of pressure ulcers and percent deficit IBW (Allman et al.,
1995; Berstrom et al., 1992; Spector, 1994).
There was no association between IBW and severity. The association between IBW and severity was measured by chi-square ($X^2 = 3.35; p = .06$). Chi-square was the test of choice since there were two groups of body weight and two groups of pressure ulcer severity. In Group 1, 63.6% had more severe pressure ulcers than superficial pressure ulcers (38.9%). Group 2 had only 26.7% severe pressure ulcers compared to 73% which were superficial. Very few studies have been conducted to address IBW and pressure ulcer severity. This study agreed with studies by Inman (1993) and Allman (1986) but disagreed with studies by Moolten, (1972) and Gorse and Messner (1987).

There was no correlation between IBW and healing rates. The association was measured by Spearman correlation coefficient analysis ($r_s = 0.010; p = .94$) and by t-test analysis ($t=0.209; p = .83$). Since mean healing rates could be calculated between the two groups, a t-test was applicable. Since healing rate was a continuous variable, the Spearman correlation coefficient analysis could also be utilized. The t-test and the Spearman correlation coefficient analysis showed no correlation or association, respectively. This could be due to the fact that the mean healing rates between the two groups were not very diversified. The mean healing rate of Group 1 was 1.08 pressure ulcers per month and Group 2 was 1.03 pressure ulcers per month. There has been only one study since 1943 that showed a correlation between IBW and healing rate (Mulholland et al. 1943), and conversely, the results of this current study disagree with the study by Mulholland et al. A possible reason why these study results disagree with
those of Mulholland's study could be due to the study design. Mulholland's study observed healing as an effect of nutrition intervention. This study did not use any interventions, nutrition or otherwise to measure healing.

Some significant associations were found between the independent and dependent variables and characteristics of the study subjects. Black subjects had higher prevalence rates than White subjects. Also, Black subjects had more severe pressure ulcers than White subjects. Bedridden subjects exhibited slower healing rates than non-bedridden subjects. Lastly, significantly more bedridden than non-bedridden subjects were below 99% of IBW.

There was an association between prevalence and race. Black subjects were found to have a higher prevalence of pressure ulcers than White subjects. The association was measured by a chi-square test ($\chi^2 = 4.634; p = .03$). Black subjects comprised 56% of the high prevalence group, but only 26% of the low prevalence group. Although prevalence and race were not hypotheses of this study, the two variables were analyzed due to the evidence in the literature that racial differences in severity exist (Meehan, 1994). This study is in agreement with other studies which found that nonwhites were more likely than whites to develop a pressure ulcer during rehabilitation and that nonwhites were more likely to present with a severe ulcer at follow-up (Rintala, 1995).

There was an association between severity and race. A significant difference between the number of Black and White subjects exist in the severe group compared to the superficial group. The association was measured by chi-square ($\chi^2 = 9.501; p = .002$).
Chi-square was the appropriate test for this analysis due to two groups of severity (0 severe or ≤2 supercials) and (>0 severe or ≥3 supercials) two racial groups, Black and White. In the superficial group, 29 (81%) out of the 36 subjects were White and only 7 (19%) were Black. Black subjects comprised 59% (n=13) of the severe cases, but only 19% (n=7) of the superficial cases. The results of this study agreed with previous studies that showed an association between severity and race (Fuhrer et al., 1993; Meehan, 1994).

There was an association between healing rates and bedridden subjects. The association was measured by t-test analysis (t = 2.38; p = .02) which showed if any significant differences existed between the mean healing rates between the two groups of bedridden subjects compared to those non bedridden subjects. The mean healing rate was significantly higher in the non-bedridden (1.34) group than in the bedridden group (0.86). Being bedridden was chosen as a variable of this study due to the evidence in the literature that bedridden subjects have an increased risk of developing pressure ulcers (Bergstrom et al., 1992). These results are consistent with other data that strongly correlate the relationship between bedridden subjects and increased risk of developing pressure ulcers (Allman et al., 1995). Allman (1997) states mobility and activity factors are the most consistently cited in the literature that predispose a person to developing pressure ulcers. Bed and chair bound individuals are at increased risk as cited by the Clinical Practice Guidelines (Bergstrom et al., 1992). It can be speculated that non-bedridden patients may have higher healing rates due to their ability to relieve the direct pressure of their sores.
An association was found between IBW and bedridden subjects. A higher percentage of bedridden subjects were below 99% of IBW. The association was measured by chi-square ($\chi^2 = 8.883; p = .003$). Seventy-nine percent ($n=22$) of bedridden subjects were underweight and 21% ($n=6$) were not bedridden. Forty percent ($n=12$) of patients greater than 99% of IBW were bedridden and 60% ($n=18$) were not. The literature does not substantiate the relationship between being bedridden and being malnourished, although it can be assumed that many bedridden patients need to be fed by nursing staff. Patients who need to be fed have been shown to be at increased risk for malnutrition and weight loss due to inadequate staff available to adequately feed patients (Poehlman, 1993).

A variable that was not shown to be associated with any of the independent or dependent variables was albumin. Albumin was chosen mainly because it has been strongly correlated with pressure ulcers (Breslow, 1991). Age and diabetes were two additional factors not shown to be associated with any variables. Age and diabetes were observed due to the multiple times they have been listed in the literature as predisposing factors for the development of pressure ulcers (Brandeis et al., 1994).

Body Mass Index was not correlated with any of the independent or dependent variables. Forty-four subjects had a BMI of less than or equal to 25 and only 8 subjects had a BMI of greater than 25. There may not have been enough subjects with BMI's over 25 to find a significant relationship. Body Mass Index was chosen as a variable due to the
presence of some studies that use BMI instead of ideal body weight (Brandeis et al., 1994).

Cholesterol was observed due to the evidence in the literature that low cholesterol is related to malnutrition (Morley & Silver, 1995) and pressure ulcers (Breslow, Halfrisch et al., 1991). No associations were present.

No significant observations were made among the various diagnoses. Only five quadriplegics were present for the entire group. Seventeen patients had CVA's with left or right hemiparesis. Eight patients had UTI's and 14 patients had dementia. This study did not attempt to identify diagnoses associated with pressure ulcers although there have been several identified. Immobility, incontinence, altered level of consciousness are a few (Bergstrom et al., 1992).

Overall, this research has agreed with other research results as stated here. This research did not show that IBW was a risk factor for prevalence, severity, or healing of pressure ulcers. Although associations were observed between prevalence and race, severity and race, healing rate and bedridden subjects, IBW and nutrition supplementation, and IBW and bedridden subjects. Results of this study did not show any significant associations between the independent or dependent variables and diabetes, BMI, cholesterol, albumin, or age.

As discussed earlier, there were several limitations to this study which may have contributed to the lack of support for the two hypotheses related to prevalence and healing rates. First is the small sample size. A larger sample size would have possibly provided a
larger number of severe pressure ulcers to compare against the superficial pressure ulcers. In addition, a larger sample size may have provided a larger diversification of healing rates which may have improved the chances of an association related to the hypotheses. Lastly, body weight had a very close association between the two groups. Mean IBW for Group 1 was 133.7 pounds and mean IBW for Group 2 was 169.4 pounds. Those subjects who were 100% of their ideal body weight were classified in the overweight group. Similarly, those subjects who were 98% of IBW were combined with those subjects who were truly underweight such as those below 90% of IBW. Lastly, the lack of female subjects was another limitation to this study. Lack of female subjects hindered the observation of whether or not gender is related to the occurrence of pressure ulcers as has been shown in other studies (Rintala, 1995).

**Application of Results**

This study has found relationships between race and severity, and race and pressure ulcer prevalence. These results should be applied to future studies which examine the relationship between race and pressure ulcers. If Blacks are found to be at higher risk for developing more severe pressure ulcers and having higher prevalence, then measures can be taken into account for these risk factors in the future. Also, relationships have been found between bedridden subjects and weight and healing rates. If future studies clarify these results, then specific guidelines can be incorporated into the care of bedridden patients to account for these risk factors.
Pressure ulcers are a major problem in all areas of health care. It has been shown that pressure ulcers are related to malnutrition (Breslow, 1991). Ideal body weight is one factor commonly used to diagnose malnutrition (Bergstrom, 1994). Therefore, if percent deficit IBW could be shown to be a risk factor for pressure ulcers, then clinicians would have a quick, easy and inexpensive tool to identify patients who may be at risk for developing pressure ulcers. At the very least, this study may lead clinicians to use percent deficit IBW as a risk factor for the development of pressure ulcers. Indeed, this would ultimately impact the timeliness of nutrition intervention for patients who may be at risk of developing pressure ulcers.

Future Research Directions

In the future, similar studies should provide a larger and more varied sample size. Another recommendation for future studies would be to include more women. This could also be accomplished by a larger sample size. Since men have been shown to be at higher risk for developing pressure ulcers (Rintala, 1995), it should be apparent that sex differences do exist and further research is needed in this area. In the VA, studies including several nursing homes could be done to provide information on pressure ulcers among the Veteran population. A larger sample size would also provide a larger number of severe pressure ulcers and possibly a higher diversification of healing rates. This study provided a small number of severe pressure ulcers and a close association between healing rates and body weight.
Future studies that observe body weight as being over or under IBW should classify body weight into two groups outside of the standard IBW range: Group 1: <90% of IBW, Group 2: <90% of IBW, Group 2: ≥110% if IBW. The subjects within the 10% IBW range could be the standard or comparison group.

Since this study has shown that Blacks were more likely to have a higher prevalence of pressure ulcers and more severe pressure ulcers that Whites, these should be the topics of further study. Also, future studies should include and compare data on Hispanics, Asians, and Native Americans. According to Rintala (1995), Caucasians and African-Americans have been studied explicitly, but no studies have been found that specifically examined the relationship of pressure ulcers among these groups.

Additionally, since this study has shown that bedridden subjects were more likely to be underweight and have slower healing rates, further research needs to be done to determine why bedridden subjects are more likely to be underweight. Also, slower healing rates in bedridden subjects needs further examination to determine the etiology and whether decreased healing rates are indirectly related to being underweight. Finally, it is acknowledged that future research is needed to determine if IBW is related to the prevalence, severity and healing of pressure ulcers.
Table 1

Pressure Ulcer Characteristics Between Groups 1 and 2

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th></th>
<th>Group 2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(&lt;99% of IBW)</td>
<td></td>
<td>(&gt;99% of IBW)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>Mean ± SD</td>
<td>n</td>
</tr>
<tr>
<td>No. pressure ulcers</td>
<td>61</td>
<td>49.5</td>
<td></td>
<td>62</td>
</tr>
<tr>
<td>Mean No. of pressure ulcers per subject</td>
<td>28</td>
<td>2.3 ± 2.3</td>
<td></td>
<td>30</td>
</tr>
<tr>
<td>Prevalence of pressure ulcers per subject</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;3</td>
<td>19</td>
<td>45.0</td>
<td>1.4 ± 0.5</td>
<td>23</td>
</tr>
<tr>
<td>≥3</td>
<td>9</td>
<td>56.0</td>
<td>3.1 ± 0.4</td>
<td>7</td>
</tr>
<tr>
<td>Mean No. Superficial pressure ulcers per subject</td>
<td>28</td>
<td>2.6 ± 4.2</td>
<td></td>
<td>30</td>
</tr>
<tr>
<td>Mean No. Severe pressure ulcers per subject</td>
<td>28</td>
<td>0.5 ± 0.8</td>
<td></td>
<td>30</td>
</tr>
</tbody>
</table>
Table 2

Demographics Between Groups 1 and 2

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(&lt; 99% of IBW)</td>
<td>(≥99% IBW)</td>
</tr>
<tr>
<td></td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>1</td>
<td>50</td>
</tr>
<tr>
<td>Male</td>
<td>27</td>
<td>48</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;65</td>
<td>5</td>
<td>42</td>
</tr>
<tr>
<td>65-75</td>
<td>8</td>
<td>47</td>
</tr>
<tr>
<td>&gt;75</td>
<td>15</td>
<td>52</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>10</td>
<td>50</td>
</tr>
<tr>
<td>White</td>
<td>18</td>
<td>47</td>
</tr>
</tbody>
</table>
Table 3

Characteristics of the Study Population

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(&lt;99% of IBW)</td>
<td>(≥99% of IBW)</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>%</td>
</tr>
<tr>
<td>Diagnosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dementia</td>
<td>7</td>
<td>50</td>
</tr>
<tr>
<td>Diabetes</td>
<td>5</td>
<td>29</td>
</tr>
<tr>
<td>Bedridden</td>
<td>21</td>
<td>62</td>
</tr>
<tr>
<td>CVAs w/hemiparesis</td>
<td>10</td>
<td>56</td>
</tr>
<tr>
<td>Urinary tract infections</td>
<td>6</td>
<td>67</td>
</tr>
<tr>
<td>Quadriplegics</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>Nutrition Supplementation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Receiving nutrition Supplements</td>
<td>19</td>
<td>61</td>
</tr>
<tr>
<td>Tube-fed</td>
<td>9</td>
<td>69</td>
</tr>
<tr>
<td>Laboratory Values</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;170 mg/dl</td>
<td>22</td>
<td>54</td>
</tr>
<tr>
<td>≥170 mg/dl</td>
<td>6</td>
<td>35</td>
</tr>
<tr>
<td>Albumin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;3.0 g/dl</td>
<td>18</td>
<td>45</td>
</tr>
<tr>
<td>≤3.0 g/dl</td>
<td>10</td>
<td>56</td>
</tr>
</tbody>
</table>
Table 4

Anthropometrics

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (&lt;99% of IBW)</th>
<th>Group 2 (≥99% of IBW)</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>%</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Body Weight</td>
<td>28  48</td>
<td>133.9 ± 21.0</td>
</tr>
<tr>
<td>Ideal Body Weight</td>
<td>28  48</td>
<td>157.1 ± 19.1</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>28  48</td>
<td>19.0 ± 2.6</td>
</tr>
</tbody>
</table>

Table 5

Hypotheses Results

<table>
<thead>
<tr>
<th></th>
<th>Chi-square Value</th>
<th>P</th>
<th>Spearman Coefficient Value</th>
<th>p</th>
<th>t-test Value</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>N* IBW &amp; Prevalence</td>
<td>0.563</td>
<td>.45</td>
<td>0.01703</td>
<td>.89</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S** IBW &amp; Severity</td>
<td>3.35</td>
<td>.06</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N IBW &amp; Heal Rate</td>
<td></td>
<td></td>
<td>0.01008</td>
<td>.94</td>
<td>0.2092</td>
<td>.83</td>
</tr>
</tbody>
</table>

* N = Non-significant (N = 58)  ** S = Significant (p < .05)  IBW = Ideal Body Weight
Table 6

Chi-square Analysis of Ideal Body Weight and Pressure Ulcer Prevalence

<table>
<thead>
<tr>
<th>Pressure ulcers (No.)</th>
<th>Group 1 (&lt;99% of IBW)</th>
<th>Group 2 (≥99% of IBW)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2</td>
<td>19 68%</td>
<td>23 77%</td>
<td>42</td>
</tr>
<tr>
<td>≥3</td>
<td>9 32%</td>
<td>7 23%</td>
<td>16</td>
</tr>
<tr>
<td>Total</td>
<td>28 48%</td>
<td>30 52%</td>
<td>58 100%</td>
</tr>
</tbody>
</table>

(N = 58)

Table 7

Chi-square Analysis of Ideal Body Weight and Pressure Ulcer Severity

<table>
<thead>
<tr>
<th></th>
<th>Superficial (≤2 superficial pressure ulcers)</th>
<th>Severe (&gt;0 severe &amp;/or &gt;2 superficial pressure ulcers)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;99% of IBW</td>
<td>14 39%</td>
<td>14 64%</td>
<td>28</td>
</tr>
<tr>
<td>Group 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥99% of IBW</td>
<td>22 61%</td>
<td>8 36%</td>
<td>30</td>
</tr>
<tr>
<td>Total</td>
<td>36 62%</td>
<td>22 38%</td>
<td>58 100%</td>
</tr>
</tbody>
</table>

(N = 58)
Table 8
Ideal Body Weight and Healing Rate

<table>
<thead>
<tr>
<th>Ideal Body Weight</th>
<th>Healing Rate in Months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
</tr>
<tr>
<td>Group 1 (&lt; 99% of IBW)</td>
<td>28</td>
</tr>
<tr>
<td>Group 2 (≥ 99% of IBW)</td>
<td>30</td>
</tr>
</tbody>
</table>

(p < .05) (N = 58)

Table 9
Significant Results of Factors Associated with the Independent or Dependent Variables

<table>
<thead>
<tr>
<th></th>
<th>Chi-square</th>
<th>Spearman Coefficient</th>
<th>t-test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Value</td>
<td>p</td>
<td>Value</td>
</tr>
<tr>
<td>Prevalence &amp; Race</td>
<td>4.634</td>
<td>.03</td>
<td></td>
</tr>
<tr>
<td>Severity &amp; Race</td>
<td>9.501</td>
<td>.002</td>
<td></td>
</tr>
<tr>
<td>Heal Rate &amp; Bedridden</td>
<td></td>
<td></td>
<td>2.38</td>
</tr>
<tr>
<td>IBW &amp; Bedridden</td>
<td>8.883</td>
<td>.003</td>
<td></td>
</tr>
</tbody>
</table>

(N = 58) (p < .05)
Table 10

Chi-square Analysis of Pressure Ulcer Prevalence and Race

<table>
<thead>
<tr>
<th>Pressure ulcers (No.)</th>
<th>Black</th>
<th>White</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤2</td>
<td>11</td>
<td>31</td>
<td>42</td>
</tr>
<tr>
<td></td>
<td>26%</td>
<td>74%</td>
<td></td>
</tr>
<tr>
<td>≥3</td>
<td>9</td>
<td>7</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>56%</td>
<td>44%</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>20</td>
<td>38</td>
<td>58</td>
</tr>
<tr>
<td></td>
<td>34%</td>
<td>66%</td>
<td>100%</td>
</tr>
</tbody>
</table>

(N=58)

Table 11

Chi-square Analysis of Pressure Ulcer Severity and Race

<table>
<thead>
<tr>
<th>Pressure ulcer severity</th>
<th>Black</th>
<th>White</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 severe ≤2 superficial pressure ulcers</td>
<td>7</td>
<td>29</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td>19%</td>
<td>81%</td>
<td></td>
</tr>
<tr>
<td>&gt;0 severe &amp;/or ≥3 superficial pressure ulcers</td>
<td>13</td>
<td>9</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>59%</td>
<td>41%</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>20</td>
<td>38</td>
<td>58</td>
</tr>
<tr>
<td></td>
<td>34%</td>
<td>66%</td>
<td>100%</td>
</tr>
</tbody>
</table>

(N=58)
Table 12

Pressure Ulcer Healing Rate and Bedridden

<table>
<thead>
<tr>
<th></th>
<th>Healing Rate in Months</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Mean ± SD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-bedridden</td>
<td>24</td>
<td>1.34 ± 0.928</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bedridden</td>
<td>34</td>
<td>0.863 ± 0.594</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 13

Chi-square Analysis of Ideal Body Weight and Bedridden Subjects

<table>
<thead>
<tr>
<th>Group</th>
<th>Non-bedridden subjects</th>
<th>Bedridden subjects</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>6</td>
<td>22</td>
<td>28</td>
</tr>
<tr>
<td>&lt;99% of IBW</td>
<td>21%</td>
<td>79%</td>
<td></td>
</tr>
<tr>
<td>Group 2</td>
<td>18</td>
<td>12</td>
<td>30</td>
</tr>
<tr>
<td>≥99% of IBW</td>
<td>60%</td>
<td>40%</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>24</td>
<td>34</td>
<td>58</td>
</tr>
<tr>
<td></td>
<td>41%</td>
<td>59%</td>
<td>100%</td>
</tr>
</tbody>
</table>
Table 14

Non-significant Results

<table>
<thead>
<tr>
<th></th>
<th>Chi-square</th>
<th>Spearman</th>
<th>t-test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Value</td>
<td>p</td>
<td>Value</td>
</tr>
<tr>
<td>Ideal Body Weight and</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>0.036</td>
<td>.84</td>
<td></td>
</tr>
<tr>
<td>Albumin</td>
<td>0.554</td>
<td>.45</td>
<td>0.22582</td>
</tr>
<tr>
<td>Cholesterol</td>
<td></td>
<td></td>
<td>0.18974</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td>-0.05145</td>
</tr>
<tr>
<td>Diabetes</td>
<td>3.427</td>
<td>.06</td>
<td></td>
</tr>
<tr>
<td>Pressure Ulcer Prevalence and</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>0.610</td>
<td>.43</td>
<td>0.05405</td>
</tr>
<tr>
<td>Age</td>
<td>2.302</td>
<td>.31</td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>1.027</td>
<td>.31</td>
<td></td>
</tr>
<tr>
<td>Albumin</td>
<td>0.376</td>
<td>.54</td>
<td></td>
</tr>
<tr>
<td>Nutrition Supplement</td>
<td>0.835</td>
<td>.36</td>
<td></td>
</tr>
<tr>
<td>Bedridden</td>
<td>0.051</td>
<td>.82</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>0.040</td>
<td>.84</td>
<td></td>
</tr>
<tr>
<td>Pressure Ulcer Severity and</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>0.039</td>
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(N = 58) (p < .05)
References


Vita

Cheryl Anne (Rusyn) Masters

Born

Education

Copley High School
Copley, Ohio

University of Akron
Akron, Ohio
Bachelor of Arts in Foods and Nutrition 1986
Bachelor of Science in Dietetics 1991

Tri City Dietetic Internship Consortium
Saginaw, Michigan
Completed fifty-one-week internship sponsored by six area hospitals 1991

University of North Florida
Jacksonville, Florida
Masters of Science in Health; Specialization: Nutrition (in progress) 1995-Present

Professional Experience

North Florida/South Georgia Veterans Health Care System
Lake City Division
Clinical Dietitian: Acute, long-term care, primary care, and quality management.
Clinical Preceptor for University of Florida’s Master level approved pre-professional practice program. 1994-Present

Kidney Dialysis Center
Jacksonville, Florida
Provided nutrition assessment and counseling for kidney dialysis and peritoneal dialysis patients.

Suburban General Hospital
Norristown, Pennsylvania
Clinical/Administrative Dietitian 1992-1993
Professional Affiliations

American Dietetic Association
Florida Dietetic Association
Jacksonville Dietetic Association
Institute of Food Technologists
ETA Sigma Gamma National Honor Society

Registration

Registered Dietitian (RD)
Licensed Dietitian (LD)