Exploring known risk factors for pressure injury with visual technology

by

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Submitted in fulfilment of the requirements for the degree of
Master of Nursing

Deakin University
April, 2012
I am the author of the thesis entitled Exploring Known Risk Factors for Pressure Injury with Visual Technology

submitted for the degree of Master of Nursing

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DEAKIN UNIVERSITY
CANDIDATE DECLARATION

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is the result of my own work and that where reference is made to the work of others, due acknowledgment is given.

I also certify that any material in the thesis which has been accepted for a degree or diploma by any university or institution is identified in the text.

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I have been inspired by many people close to me and who inevitably sustained me during this journey. It is only through the encouragement and wisdom from these many professional colleagues and friends that has led me to completion.

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ABSTRACT

Background and significance: Pressure injuries are a clinical indicator for health care quality. Interface pressure mapping systems have the potential to provide extensive numerical and visual data related to risk for these injuries. However, the application of these systems has been limited in the clinical setting. Under-explored areas include measurement of pressure gradients around a peak interface pressure point to understand deep tissue injury and identification of body shape as a pressure injury risk marker through varying muscle tone and tissue tolerance. These knowledge gaps are significant because improvements in risk assessment, including factors not currently included, may lead to better identification of people at risk of developing pressure injuries and more appropriate application of costly preventative interventions.

The aims of this study were to explore:

1. correlations between two interface pressure mapping indices and selected risk factors for pressure injury,
2. visual anatomical characteristics of patients through the use of interface pressure mapping and the correlation between shape and selected risk factors for pressure injury.

Methods: This nested exploratory study utilised a prospective correlational design. A convenience sample of 120 medical and surgical patients was enrolled. Patients were positioned on mapping equipment (Tekscan Cliniseat™), supine in bed with an elevation of 30% at the head. Data were collected from clinical records and through clinical assessment. Pressure injury risk was assessed using the Waterlow Risk Assessment Tool. Ten separate mapping measurements were taken from which mean peak interface pressure and pressure gradients were calculated. Data were analysed using both statistical and manufacturer-specific software. To assess correlation both Pearson’s r and Spearman’s rho were utilised because not all data were normally distributed.

Results: The first research aim investigated correlations between three selected risk factors for pressure injury and interface pressure mapping indices. No correlations were evident with the exception of peak interface pressure with pressure gradient at 1.5cm and 2.5cm; and gradient 1.5cm with 2.5cm. The second aim was addressed through identification of five shapes exhibited by patterns of skin surface area in contact with the interface pressure mapping surface. These five shapes were further reduced to two groups; namely round/square and other shapes. Round/square shapes were statistically significantly associated with higher weight, Body Mass Index and Waterlow Risk Assessment score.

Discussion and conclusions: The study findings have enabled refinement of Defloor’s conceptual framework for prediction and prevention of pressure injury. Documentation of the correlation between peak interface pressure and pressure gradients has contributed to the understanding of deep tissue injury. In addition, as a pilot study, visual assessment of buttock shape has demonstrated potential for identifying risk of ischial or sacral pressure injury. These findings have significance for policy, practice and research. The policy imperative to use available risk assessment tools needs to be reconsidered. Increased use of interface pressure mapping systems in the clinical setting has great educational promise through the potential visualisation of deep tissue injury as well as peak interface pressures at the skin surface. The potential for assessment of shape in the clinical setting requires further research and development. Finally further research informing understanding of the extent of deep tissue injury through calculation of gradients greater than 2.5cm from the point of peak interface pressure is needed.
<table>
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<td>2D</td>
<td>Two dimensional</td>
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<td>3D</td>
<td>Three dimensional</td>
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<tr>
<td>ABS</td>
<td>Australian Bureau of Statistics</td>
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<td>ACT</td>
<td>Australian Capital Territory</td>
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<td>AWMA</td>
<td>Australian Wound Management Association</td>
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<td>BMI</td>
<td>Body Mass Index</td>
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<td>CHC ACT</td>
<td>Calvary Health Care ACT</td>
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<td>EPUAP</td>
<td>European Pressure Ulcer Advisory Panel</td>
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<td>MIPPI</td>
<td>Mapping and Intervention for Prevention of Pressure Injury</td>
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<td>NPUAP</td>
<td>National Pressure Ulcer Advisory Panel</td>
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<tr>
<td>NSW</td>
<td>New South Wales</td>
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<tr>
<td>PUPPS</td>
<td>Pressure Ulcer Point Prevalence Survey</td>
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<td>RCNMP</td>
<td>Research Centre for Nursing and Midwifery Practice</td>
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<tr>
<td>SPSS</td>
<td>Statistical Package for the Social Sciences</td>
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<td>TCH</td>
<td>The Canberra Hospital</td>
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<td>USA</td>
<td>United States of America</td>
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<td>WHO</td>
<td>World Health Organisation</td>
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CHAPTER ONE - INTRODUCTION

Introduction to Pressure Injuries

Pressure injuries have been recognised as a patient safety problem and as a major challenge for healthcare professionals and healthcare systems (Collier & Moore, 2006; Maklebust, 2005; Ousey, 2005; Torra i Bou, Garcia-Fernandez, Pancorbo-Hidalgo & Furtado, 2006). They have been identified as a nursing specific clinical indicator and an indicator for the quality of care provided by health services (Baharestani et al., 2009; Harrison, Logan, Joseph & Graham, 1998; Ousey, 2010). Pressure injuries can be typically identified with frail, debilitated, elderly or neurologically impaired patients, those who experience periods of acute or prolonged illness and particularly those who are immobile for extended periods (PUPPS 2, 2005). Whilst the number of Australians living with a pressure injury is unknown, Hibbs (1988, p.32) describes this situation as an epidemic but “a silent one hidden under the sheets”, with many pressure injuries consequently remaining “not only unseen but also untreated, unrecorded and uncusted” (PUPPS 2, 2005, p.6). Moreover, a large percentage of pressure injuries are considered to be preventable (Boyle & Green, 2001; Maklebust & Sieggreen, 2001; NPUAP & EPUAP, 2009; Torra i Bou et al., 2006), thereby indicating that a large number of people are suffering. The identification of patients considered at risk of pressure injury development and the implementation of prevention strategies is therefore crucial.
The definition of a pressure injury as published in the Pressure Ulcer Prevention and Treatment Clinical Practice Guidelines by the National Pressure Ulcer Advisory Panel (NPUAP) and the European Pressure Ulcer Advisory Panel (EPUAP) is “a localised injury to the skin and/or underlying tissue, usually over a bony prominence, that results from pressure or in combination with shear” (2009, p.16). A number of other definitions have also been advanced. The Australian Wound Management Association (AWMA) (2001, p.4) and Maklebust (2005, p.365) have used “any lesion caused by unrelieved pressure that damages underlying tissue”, whilst Dealey (1994, as cited in Ousey, 2005, p.2) has suggested “ulceration of the skin following disruption of the blood supply due to pressure, friction or shear, or a combination of all of these factors”. The common factors are the presence of pressure on tissues, and on the underlying structures, resulting in tissue deformation, tissue injury and irreversible necrosis.

Pressure-induced tissue damage as described above has variously been referred to as a bed sore, pressure sore, decubitus ulcer or pressure ulcer in the literature. A bibliometric analysis of these terms conducted by Dunk and Arbon (2009; Appendix A) showed that pressure ulcer was the most commonly used term over the period 2001 to 2006. The NPUAP and EPUAP (2009) international guidelines use the word injury as the classification describes tissue loss rather than ulceration. This thesis will use the term pressure injury as a description of the condition as it is believed that the adoption of this terminology will have benefits by focussing clinicians on their role in early assessment and prevention (Dunk & Arbon, 2009). It also differs from all other terms as it draws attention to the causation rather than to the description of the wound.
Pressure injuries are classified to provide an indication of the severity of the skin and tissue damage (AWMA, 2001; Dealey & Lindholm, 2006) with the first classification system developed by Shea in 1975. Multiple and sometimes complex classification systems have since been developed but the most widely used system internationally is the one published by the NPUAP and EPUAP (Dealey & Lindholm, 2006), the latest version of which was issued in 2009. In Australia a pressure injury staging system currently uses the four classifications of pressure injuries, with an additional classification for necrotic tissue as ‘unstageable’. These have been published in Clinical Practice Guidelines for the Prediction and Prevention of Pressure Ulcers by the AWMA (2001), with an updated version expected in late 2011.

The importance of pressure injuries as a health problem stems from the number of people affected worldwide, and the associated costs incurred by health systems (Dealey, 2004; Lyder, 2006; Ousey, 2005). Pressure injuries have also been assessed as extending the length of hospital stay for affected patients, impacting on hospital bed availability and reducing overall hospital efficiencies. Graves, Birrell and Whitby (2005) have reported a study in Queensland that showed that the median extension of length of hospital stay due to pressure injury was 4.3 days. The authors also cite earlier studies that indicated that the length of stay could be extended by as much as 50 days due to pressure injury. In United States hospitals, pressure injury can increase a patient’s period in hospital by up to five times (Maklebust & Sieggreen, 2001; NPUAP & EPUAP, 2009).
Conservative estimates based on prevalence studies in Australia indicate that between 3% and 36.7% of hospital in-patients will have a pressure injury (Prentice, Stacey & Lewis, 2003). A range of other authors have reported the results of studies that show prevalence in United States of America (USA) and European settings is also within this range (Clark, Defloor & Bours, 2004; Walsh & Bennett, 2004). The cost of healing per pressure injury has been determined as between £1489 for a Category I injury up to £14,771 for Category IV in the United Kingdom (Franks, 2007). In addition, litigation in the USA for pressure injuries developed by hospital in-patients has resulted in monetary awards with a median value of $250,000 (Bennett, O’Sullivan, DeVito & Remsberg (2000) as cited in Lyder, 2006). Prentice and Stacey (2001) also report that the number of litigation cases in Australia related to pressure injury development is increasing. The financial cost to health care systems of pressure injuries is therefore large, and any reduction in pressure injuries would translate into direct financial benefits.

Apart from the financial impact on the health system, and the operational impact on the individual hospitals, pressure injuries have a very real and negative impact on both sufferers and care-givers. The patient’s quality of life has been reported as being significantly affected (Baharestani, 2008; Franks, 2007; Franks, Winterberg & Moffatt, 2002; Ousey, 2010) to such an extent that Hietanen (2006) has suggested that it is easy for patients with pressure injuries to feel that they are hostage to the injury. Pain has been identified as the greatest issue for sufferers (Gorecki et al., 2009; Hopkins, Dealey, Bale, Defloor & Worboys, 2006). Mobility, quality and quantity of sleep, the ability to work and social engagement have all been cited as negatively affected by pressure injury.
In addition to pain, odour and physical disfigurement have been reported as lowering the self-esteem of sufferers and heightening feelings of isolation and social rejection (Baharestani, 2008; Gorecki et al., 2009). Sibbald, Chapman & Contreras- Ruiz (2006) have suggested that issues such as control of odour, pain and sleep may be more important for the patient than the actual healing of the wound itself. In addition, there are likely to be financial concerns and associated stress regarding the impacts of reduced work, lost career opportunities and the costs of ongoing treatment (Baharestani, 2008; Franks, 2007). The impact of pressure injuries on elderly persons is sufficiently great as to be increasingly viewed as potential neglect or elder abuse and subject to potential litigation (Clarkson, 2007; Lyder, 2006; Walsh & Bennett, 2004). Prolonged healing times and often multiple injuries are also known to increase the risk of infection, with osteomyelitis as a result of pressure injury being a contributing cause of death in some patients. In a 2005 report prepared for the Victorian Quality Council, the Australian Bureau of Statistics (ABS) determined 923 people in had died with pressure injuries determined as the primary or secondary cause of death during the period 2001-2003 (ABS, 2005, as cited in PUPPS 2, 2005).

Care-givers are also known to be affected by the development and treatment of pressure injuries, particularly for care provided outside of a health care facility. These concerns include the fear of damaging the wound from inadequate knowledge, the disfigurement of the patient, their own reduced social interaction, and ongoing financial concerns (Baharestani, 2008; Franks, 2007). In a nursing context, clinicians have been physically affected due to the effort required in the
management of disabled patients (Scales, 1976) and mentally affected by feelings of responsibility and guilt over pressure injury development (Ousey, 2005). Whilst occupational health and safety guidelines have reduced the direct lifting that historically has been a feature of nursing, the current application of lifting devices brings administrative overheads for clinicians. In addition, if insufficient lifting devices or personnel are not provided by a healthcare facility, the necessary turning regimens and off-loading frequencies are unlikely to be achieved with direct implications on the potential for pressure injury development.

**Development of Pressure Injury Management**

Pressure injuries are an ongoing factor in the human condition and have been found on Egyptian mummies dating back over 4000 years (Anthony, 1996; Ousey, 2005). In 1593 Fabricus Hilanus in the Netherlands described the characteristics of pressure injuries (Defloor, 1999; Ousey, 2005). Around the same time Ambrose Pare in France was treating pressure injuries and suggested that good nutrition, addressing the underlying illness, relief of pressure, proper dressings and psychological support were all factors in prevention and management (Levine, 2005; Ousey, 2005). In 1815 William Heberdeen devised a bed frame that would aid the treatment of pressure injuries (Cherry, 2006) and the importance of changing position was recognised by 1848 by Robert Graves (Defloor, Vanderwee, Wilborn & Dassen, 2006). The contribution of shearing forces to pressure injury development was first noted by Riechel in 1958, although not acknowledged until 1970 (Defloor, 1999).
Nurses have traditionally played a pivotal role in pressure injury management. Dealey (2004) has suggested that until the late 20\textsuperscript{th} century pressure injuries were seen as almost exclusively a nursing problem and most doctors avoided any responsibilities in this area. This view, and the pre-eminent position of nurses in the field, has developed due to the combination of two separate but related arguments from the middle half of the 19\textsuperscript{th} century.

The first position was advanced by Florence Nightingale in 1860 when she promoted the responsibility of nurses in the prevention of pressure injuries. Nightingale maintained that pressure injuries were preventable with good nursing care, and the development of an injury reflected on the provision of that care rather than being associated with any disease process (Ousey, 2005). The second was the position adopted by the medical community as a result of arguments advanced by Charcot during the late 19\textsuperscript{th} century that pressure injuries were unavoidable in the case of spinal injury due to the perception at that time that pressure injury resulted from neurological deficits rather than ischaemic problems (Gebhardt, 2004; Levine, 2005; Prentice & Stacey, 2001). Gebhardt (2004) argues that the medical status of Charcot was such that his view was almost universally accepted and as a consequence pressure injuries became a nursing rather than a medical issue. In more recent times the prevention and treatment of pressure injuries been recognised as requiring an interdisciplinary approach (Ousey, 2005).

In an endeavour to sift through the available research and present the consensus results for use by clinicians a range of national and international bodies have developed and published consensus pressure injury guidelines (Cherry, 2006;
van Zelm, Clark & Haalboom, 2006). The NPUAP and EPUAP Guidelines (2009) aim to “provide evidence-based recommendations … that can be used by health care professionals” (p.7). The strength of the evidence underpinning each recommendation in the NPUAP & EPUAP Guidelines (2009) is noted by a score from A (supported by direct scientific evidence) through to C (supported by indirect evidence). The Guidelines are therefore a means through which the movement towards evidence-based practice can be facilitated.

In spite of these developments, and the increased focus on the need to address all aspects of pressure injury from aetiology to treatment, the challenges remain formidable and the level of evidence that underpins the practice recommendations is poor (Ankrom et al., 2005; Bader & Oomens, 2006; Clark, 2008; Colin, 2006; Collier & Moore, 2006; Gebhardt, 2004; Nelson et al., 2003; Papanikolaou, Clark & Lyne, 2002). This overall assessment is reflected in that the vast majority of the NPUAP & EPUAP (2009) Guidelines recommendations are rated at the lowest strength of evidence score.

The ability to predict the development of pressure injury remains low and there is a continuing reliance on clinical judgement and ongoing visual observations of the skin surface (Bader & Oomens, 2006; Gebhardt, 2004; Stekelenburg, Gawlitta, Bader & Oomens, 2008). One used approach to understanding pressure injury development is through a conceptual framework that models the known risk factors and interventions and the ways in which they interact. Such frameworks have been developed by Braden and Bergstrom (1987) and Defloor (1999). Whilst variously called a conceptual model, framework, or scheme I will standardise on
the term ‘framework’ as this best indicates the diverse and incomplete nature of the pressure injury problem.

**Introduction to the Study**

As noted previously in this chapter, the aetiology of pressure injuries is not fully understood (Bader & Oomens, 2006; Collier & Moore, 2006; Gebhardt, 2004; NPUAP & EPUAP, 2009; Papanikolaou et al., 2002). Potentially harmful pressures are developed within the skin and underlying tissue by the weight of the human body as it pushes down on the skeletal structure. Posture and the associated distribution of weight over the support surface are therefore of fundamental importance (Roaf, 1976). The propensity for pressure injury development is therefore increased when the weight becomes distributed through the relatively small amount of soft tissue that lies between bony prominences and the underlying support surface (Defloor, 2000). When these forces are exerted over time the blood flow to the underlying tissues is impacted and pressure injury can result. The forces of concern are pressure, shear and friction.

Pressure is widely considered to be the most important of these mechanical forces (NPUAP & EPUAP, 2009; Reger, Ranganathan, Orsted, Ohura & Gefen, 2010; Takahashi, Black, Dealey & Gefen, 2010). The pressure at the skin surface acts perpendicularly on the tissue and can be measured as interface pressure, although the value of interface pressure alone as a predictor of pressure injury development has been questioned (Bader & Oomens, 2006; Oomens, Loerakker & Bader, 2009; Swain & Bader, 2004).
With the exception of hydrostatic loading, the pressure distribution from a force applied to the body will be heterogeneous in nature resulting in different areas suffering differing amounts of interface pressure (Oomens et al., 2009). These differing pressures give rise to shear, a force that acts parallel to tissue. The areas with the highest rate of change of pressure over distance, or the highest pressure gradient, will experience the highest levels of shear strain, and hence be most susceptible to pressure injury development (McLane, Krouskop, McCord & Fraley, 2002; Oomens et al., 2009; Takahashi et al., 2010). Shear forces cannot exist separately from pressure and a number of authors have noted that the effects of pressure are magnified in the presence of shear (Ayello, Baranoski, Lyder & Cuddigan, 2008; Chow, Juvinall & Cockrell, 1976; Collier & Moore, 2006; Defloor, 1999; Exton-Smith, 1976).

Pressure, shear and friction are however difficult to measure in the clinical environment or to appreciate by visual inspection of the skin surface. NPUAP & EPUAP Guidelines (2009) indicate that pressure gradient is crucial in the development of pressure injury. An ability to determine where high pressure gradients exist may therefore be useful in assessing pressure injury risk with clinical practice.

Interface pressure mapping systems, typically comprised of multiple “sensels” across a measuring mat, enable the visualisation of the distribution of the pressure at the interface between the skin and the supporting surface. These systems therefore provide the means to determine areas of tissue that are under the
greatest stress by highlighting rates of change of pressure through visual means. Interface pressure mapping has been used in studies associated with patient positioning and when considering the efficacy of support surfaces (Hanson, Langemo, Anderson, Hunter & Thompson, 2006; Rithalia, 2005; Shelton & Lott, 2003).

Interface pressure mapping systems provide information on the shape at the interface surface. The importance of this shape information for pressure injury development is largely unknown, but body shape has been regarded as an important source of health and disease risk information (Wells, Treleaven & Cole, 2007). Swain and Bader (2004) have also suggested that the shape of at-risk patients may change due to loss of muscle tone, therefore increasing pressure injury development risk. The Defloor (1999) conceptual framework, used to describe the interaction of various factors associated with the development of pressure injury, suggests that body build plays a role in the ability of tissue to tolerate pressure. The ability to monitor and consider shape may therefore provide valuable clinical information for the assessment of pressure injury risk.

The primary approach for the prevention of pressure injury has been to identify patients considered to be at risk and to implement preventative strategies (Exton-Smith, 1976; Gebhardt, 2004; Papanikolaou et al., 2002). The current tools available to nurses are therefore those that support preventative and management strategies, but an ability to accurately predict development of a pressure injury is currently impossible in clinical practice (Kottner & Balzer, 2010). In addition to the forces of pressure, shear and friction acting on tissue, a wide range of personal-
centric, or intrinsic, risk factors have been identified as contributing to pressure injury development (Defloor, 1999; Kottner & Balzer, 2010). From these observed risk factors a variety of risk tools have been constructed and utilised by clinicians in the assessment of pressure injury risk (Gardner, Dunk, Eggert, Gardner & Wellman, 2006 [Appendix B]; Torra i Bou et al., 2006). There is however no current evidence that the use of risk assessment tools provide more effective prevention than clinical judgement alone (Anthony, Papanikolaou, Parbotecah & Saleh, 2010; Kottner & Balzer, 2010; Pancorbo-Hidalgo, Garcia-Fernandez, Lopez-Medina & Alvarez-Nieto, 2006; Schoonhoven et al., 2002). An improved understanding of risk factors and hence an improved ability to predict risk, whether through the use of risk assessment tools or some other measure, should lead to reduced levels of pressure injury development.

As a nurse I have been exposed to both the clinical and the social aspects of pressure injuries for many years. In my current and previous roles I have also been engaged in clinical research, and have become particularly interested in how pressure injury prediction may be improved. In 2004 I was involved as the Project Coordinator for the Mapping and Intervention for Prevention of Pressure Injury (MIPPI) project undertaken by the Research Centre for Nursing and Midwifery Practice (RCNMP) at The Canberra Hospital (TCH). The MIPPI study investigated the relationship between risk factor scores, interface pressure and capillary blood flow (Gardner et al., 2006) exposed me to the use of technology for the mapping of interface pressure, and introduced me to the potential use of interface shape and pressure gradient visualisation for pressure injury prediction.
This research focussed on pressure injury assessment and prevention and the correlation between a number of risk factors encountered by nurses in clinical practice and interface pressure mapping indices were explored. The risk factors are weight, Body Mass Index (BMI) and risk score as determined by the Waterlow Risk Assessment Tool. The interface pressure mapping indices are peak interface pressure, and pressure gradients around the peak interface pressure point. An interface pressure mapping system, a technology not frequently used by clinical nurses, was used in the study. The pressure mapping technology was important as it provided, not only the ability to record numerical data, but also to simultaneously provide a visual observation of the pressure distribution and the shape across the interface surface.

The aims of the study were twofold, namely:

1. To explore correlations between two interface pressure mapping indices and selected risk factors for pressure injury, and
2. To explore the visual anatomical characteristics of the buttock region of patients through the use of interface pressure mapping and the correlation between shape and selected risk factors for pressure injury.

In this thesis I will initially examine the current literature relating to pressure injury development and prevention. In Chapter Two I will consider the current state of pressure injury knowledge and the application of interface pressure mapping and visualisation techniques in particular. The importance of pressure gradients in the formation of pressure injuries and the potential for the utilisation of
shape to inform risk will also be considered. Pressure injury risk assessment will be examined. The methodology through which the study was developed, data obtained and analysis conducted is described in detail at Chapter Three. The results from the study are presented in Chapter Four, and discussed in detail in Chapter Five.

The study has showed that a high level of correlation exists between interface pressure and the pressure gradient at a distance 1.5cm from the point of peak interface pressure. Given the importance given to pressure gradient as an indicator of shear stress and tissue distortion, (Oomens et al., 2009; Rithalia, 2005) interface pressure mapping as described herein may provide an indication of pressure injury risk. The results of the study also show that the shape of the buttock area in contact with the support surface may be a useful indicator of risk of pressure injury development.

Most studies into pressure injury have either utilised healthy volunteers (Rithalia, 2005), or concentrated on groups known to have a high incidence of pressure injury development (Gebhardt, 2004). This study utilises a variety of patients in an acute hospital setting and therefore significantly adds to the knowledge base associated with pressure injury risk and the development of preventative strategies through the involvement of a more relevant population sample.
CHAPTER TWO - LITERATURE REVIEW

Introduction

Pressure injuries are widely considered to be a major health problem with large numbers of people affected worldwide (Baharestani et al., 2009; Lyder, 2006; Maklebust & Sieggreen, 2001; McClemont, 1984). Pressure injuries create wide-ranging effects on patients in terms of pain and social impact, on care-givers in terms of physiological and psychological stress, and on health care facilities in terms of cost, governance and legal responsibilities (Baharestani, 2008; Rithalia, 2004). The national cost of pressure injuries is subject to some conjecture. In Australia an estimate of the cost of pressure injuries of $350 million per annum was made by the then Minister for Health in 1997 (Woolridge, 1997, as cited in Prentice, 2002). This figure is supported by modelling conducted by Graves et al. (2005) that indicated that the overall cost of pressure injuries in Australia is approximately $300 million each year. Costs in the United Kingdom are variously reported as being approximately £600 million through to £2-3 billion each year (Cherry, 2006; Ousey, 2010).

The scale of the pressure injury problem, both in hospitals and in the community, is commonly assessed through the study of prevalence and incidence rates (Baharestani et al., 2009; Dealey, 2004; Prentice et al., 2003). Prevalence is the number of people with a pressure injury as a proportion of a population at a moment in time. Prevalence therefore includes both those patients who may have been admitted to a healthcare facility with an existing pressure injury as well as
those who may have developed a pressure injury during their treatment. Incidence is the number of new pressure injuries that have developed over time (Baharestani et al., 2009; Boyle & Green, 2001; Dassen, Tannen & Lahmann, 2006).

Throughout the world both of these rates remain unacceptably high with pressure injury prevalence in Australia reported as ranging from 3% to 36.7% between 1983 and 2002 (Prentice et al., 2003). The prevalence rate across the health care facilities in the Australian Capital Territory (ACT) has been reported as being between 18% and 29% (Gardner & Dunk, 2004). In Victoria PUPPS 3 (2006) has reported a prevalence rate of 17.6%. A prevalence pilot study conducted for the EPUAP by Clark et al. (2004) across five countries in Europe showed that 18.1% of patients surveyed had established pressure injuries. The pressure injury prevalence in USA hospitals has been estimated by the NPUAP to be 15% (Ayello & Braden, 2002; NPUAP & EPUAP, 2009), although other studies have shown rates as high as 65% (Moore, 2005). A study conducted in New South Wales (NSW) by Boyle and Green (2001) showed that incidence ranged between 6% and 21% for an intensive care unit. Direct comparison of both prevalence and incidence figures between health care settings has however been hampered by inconsistency in the structure and performance of the studies (Baharestani et al., 2009; Clark et al., 2004; Dassen et al., 2006; Gardner et al., 2009).

Over the recent past there has been a consolidated focus both in Australia and internationally on addressing the problem of pressure injuries. This work has included the establishment of advisory panels and committees, the development and update of a number of clinical practice guidelines for the prevention and
management of pressure injuries (AWMA, 2001; NPUAP & EPUAP, 2009), and focus on the benefits of multi-disciplinary research. One outcome of this work has been a heightened interest in the use of prevalence data to better understand the extent of the pressure injury problem (Dealey, 2004) and hence to assist in the development of more focussed strategies for its remediation (Dunk & Trevitt, 2005 [Appendix C], Whitehead & Arbon, 2007). Bader and Oomens (2006) have stated that reduction in prevalence rates can only be achieved through an improved understanding of the aetiology of pressure injury from both the basic science and clinical experience viewpoints.

Prevention is widely considered as the most efficient method to address the pressure injury problem (Defloor, 1999; Ousey, 2009; Walsh & Bennett, 2004). Thomas (2006) has divided preventative strategies into five categories, namely; (1) recognising the risk, (2) decreasing the effects of pressure, (3) assessing nutritional status, (4) avoiding excessive bed rest and prolonged sitting, and (5) preserving the integrity of the skin. This thesis will focus primarily on the area of pressure injury risk recognition through the application of interface pressure mapping technology, and on the potential use of visual assessment of the buttck region.

In this chapter I will initially examine the anatomy and physiology of skin and the normal skin responses to pressure. The importance of interface pressure and pressure gradients will be considered. I will then examine the causes of pressure injury, and the various factors that are involved. The importance of a conceptual framework for the holistic consideration of pressure injury risk factors will be introduced. The means of assessing the risk of developing a pressure injury
will be examined, together with an assessment of the efficacy of risk assessment tools. The chapter will conclude with a discussion on preventative strategies and determination of gaps in the current knowledge base.

Anatomy and Physiology

This section will concentrate on the anatomy and physiology of skin, and the way in which skin is affected by pressure. The various forces that can impact on the human body, and the means by which these can be transmitted, will be examined. I will introduce the concepts of interface pressure and tissue tolerance, the application of interface pressure measurement and mapping, and the utilisation of interface pressure mapping in a clinical environment.

Structure of Skin

Skin is the largest organ in the human body and contributes approximately 10% to overall body mass (Butcher & White, 2005). The function of skin is to protect the body by providing an interface to the external world and thereby shielding the inner tissues (Maklebust & Sieggreen, 2001). The skin also undertakes a number of vital bodily functions including (1) regulating body temperature, (2) transmitting sensations, (3) protecting against excessive fluid loss, and (4) acting as an excretory organ (Bale, Cameron & Meaume, 2006; Maklebust & Sieggreen, 2001).
Skin has a multi-layered structure with each layer having different cells and structures, and providing a separate function. These layers are the epidermis, the dermis, and the subcutaneous layer or hypodermis. The epidermis and dermis are both separated and attached by the basement membrane (Maklebust & Sieggreen, 2001; Ousey, 2005). This interlocking, semi-permeable membrane is comprised largely of collagens. It provides structural support to the epidermis and allows fluid and cell exchange between the layers (Butcher & White, 2005; Carville, 2005; Maklebust & Sieggreen, 2001).

The epidermis is the outermost layer. It is a thin, avascular layer that regenerates every four to six weeks and is itself composed of five functionally different strata. The outermost stratum is the *stratum corneum*, consisting of dead keratinocyte cells and providing a major chemical and mechanical defence for the body. The second stratum the *stratum lucidum*, only occurs on the palms and soles. This layer is transparent with no visible nuclei (Carville, 2005). The next stratum is the *stratum granulosum*, containing active keratinocytes and Langerhans cells. The latter cells originate in bone marrow and play an important role in immune responses (Maklebust & Sieggreen, 2001). The penultimate stratum of the epidermis is the *stratum spinosum*. This stratum also contains Langerhans cells but cannot regenerate (Maklebust & Sieggreen, 2001). The *stratum spinosum* provides protection against the forces of shear and friction (Bale et al., 2006). The innermost stratum is the *stratum germinativum*, a single layer of cells that is the only one that can regenerate or create new cells (Baranoski, Ayello & Tomic-Canic, 2008; Maklebust & Sieggreen, 2001).
In contrast to the multi-layered epidermis, the dermis is structured as a matrix of collagen and elastin that provides nutrients and mechanical strength to the skin (Bale et al., 2006; Baranoski, Ayello & Tomic-Canic, 2008; Maklebust & Sieggreen, 2001). The dermis is divided into two functionally different layers. The outermost layer is the papillary dermis. This layer contains collagen and reticular fibres important for healing, and capillaries that provide nourishment to the epidermis via the basement membrane (Carville, 2005; Maklebust & Sieggreen, 2001). The reticular dermis consists of networks of collagen bundles that provide elasticity to the skin and anchor the dermis to the subcutaneous tissue (Maklebust & Sieggreen, 2001). The elastic fibres in the dermis are important contributors to the way in which tissue recovers from deformation. In areas that have less densely packed capillaries and elastic fibres the tissue recovery will be delayed with resultant adverse effects to the tissue (Hagisawa, Shimada, Arao & Asada, 2004).

The hypodermis, or subcutaneous layer, lies beneath the dermis. This layer provides the main supporting framework for the skin and promotes the blood supply to the dermis that is required for that layer’s regenerative function (Baranoski, Ayello & Tomic-Canic, 2008; Carville, 2005). The hypodermis provides a cushion or shock absorber between the skin and the underlying muscles and bones (Baranoski, Ayello & Tomic-Canic, 2008; Maklebust & Sieggreen, 2001). These layers have a complex circulatory system that will now be described.

The blood vessels in skin comprise arteries for supply of blood, capillary beds to allow blood to flow from arteries to veins, and veins that provide drainage away from the skin. Blood pressure in the capillaries is significantly lower than in
the arteries, and drops between the arteriolar and venous ends from approximately 32 mmHg to approximately 12 mmHg (Maklebust and Sieggreen, 2001). The capillary loops in the skin run vertically to the surface and are coiled at their bases, thereby reducing the risk of occlusion as a result of direct pressure. In the subcutaneous tissue however, the blood vessels are substantial and lie predominantly parallel with the deep fascia. These vessels are therefore at much greater risk of distortion and occlusion as a result of pressure (Bliss, 1993, as cited in Collier & Moore, 2006; Butcher & White, 2005).

**Pressure, Interface Pressure and Tissue Response to Pressure**

Pressure as it relates to its effect on the human body can be described as a force exerted perpendicular to the tissue (Defloor, 1999; Gibson, Barbenel & Evans, 1976; Ousey, 2005). This gravitational force is also often referred to as compression (Collier & Moore; 2006; Gibson et al., 1976). Takahashi et al. (2010) define pressure as the amount of force exerted perpendicular to a surface per unit area. Collier and Moore (2006) calculate average pressure as body weight divided by skin contact area. Defloor (2000) and Rithalia and Kenney (2001) describe interface pressure as the pressure applied to the epidermis by the surface that is supporting it, calculated by the formula: Interface pressure equals patient weight divided by surface area support. This calculation implies that the greater the surface area of the body in contact with a supporting surface, the lower the average tissue interface pressure will be. This implication will be revisited later in this chapter when reviewing pressure injury prevention.
Two important points can be extrapolated from the definition of interface pressure above. The first is the impact of the area over which the pressure is measured, and illustrates the means by which a pressure map of the contact area can be constructed using technology. This can be achieved by taking multiple measurements over a grid of uniformly sized and distributed pressure sensors in a specialised mat and illustrating the measured pressure from each sensor location of the mat. The granularity of the resulting image will therefore depend on the sensitivity of the pressure sensors and the number and size of each individual measurement location. This description becomes important later in this thesis when discussing the utilisation of a particular interface pressure mapping system.

The second point is that as interface pressure involves both skin and a support surface, the nature of both the epidermis and the characteristics of the support surface may have an impact on the measured pressure. A number of authors have indeed commented on the complex nature of interface pressure and how it is influenced by the presence of a bony prominence at the point of pressure, the shape of that prominence, and the amount and nature of tissue covering the underlying structure (Defloor, 2000; Swain and Bader, 2004). Both Bader and Oomens (2006) and Collier and Moore (2006) have highlighted the different effects that body site can have on measured pressure due to the local bone, muscle and tissue structure. The mechanical characteristics of the supporting surface has also been stated as important for the observed interface pressure (Defloor, 2000) as noted later in this chapter in the discussion on pressure relieving devices.
These mechanical characteristics are also important to remember when considering circulation. Capillary closure depends on local pressure gradients across the blood vessel and not just on interface pressure at the skin surface. This relationship has been remarked upon by Swain and Bader (2004) who have noted that body tissues are able to withstand high levels of omni-directional hydrostatic pressure without any adverse effects. Bader and Oomens (2006) have also commented that interface pressures well above capillary pressures can be supported for some time by the soft tissues before blood flow is seriously impaired.

Measurements taken by Landis in 1930 determined that the pressure in the capillary bed in healthy medical student volunteers ranged between 12 and 32 mmHg. The figure of 32 mmHg has since been cited extensively in the literature as the threshold for capillary occlusion (Frantz and Xakellis, 1989; Maklebust & Sieggreen, 2001; McClemont, 1984; Mulder, Taro, Seeley & Andrews, 1994; Thompson-Bishop & Mottola, 1992). In 1941 Landis revised his work and identified that a more realistic figure for capillary closing pressure should be between 45-50 mmHg, after which damage was likely to occur (Collier & Moore, 2006). A number of authors have also suggested that 32mm Hg should not be used as a damage threshold figure as it represents a localised measure at an area not at risk of developing pressure injury (Bader & Oomens, 2006; Defloor, 1999; Shelton & Lott, 2003; Swain & Bader, 2004; Takahashi et al.; 2010; Thompson, 2005).

Studies to date have indicated that the actual figure for capillary closing will depend on the site of the pressure attack, the structure of the bone and tissue structure at that site, and the overall health status of the patient (Collier & Moore;
2006; Defloor, 1999). A pressure threshold as low as 12 mmHg has been suggested for severely compromised patients (Shelton & Lott, 2003). This is of great concern when interface pressures measured on a regular mattress have been reported to be as high as 100 – 150 mmHg over bony prominences (Barnett & Ablarde, 1994).

Any pressure measured at the skin will be transmitted from that interface to the anatomy below (Collier & Moore, 2006). Defloor (1999) cites studies conducted by Kosiak fifty years ago to argue that an average of 70-80% of external pressure is distributed within the internal tissue without development of pressure injury. Collagen and elastin are postulated to assist here. In areas where there is very little superficial covering, however, the majority of the pressure is transferred directly to the underlying tissue (Defloor, 1999).

The nature of the subcutaneous tissues, in terms of thickness, tone and mechanical integrity, coupled with the proximity of bony prominences, are key factors when considering the relationship between the application of interface pressure and the internal forces (Oomens et al., 2009; Swain & Bader, 2004). The term tissue tolerance has been coined to describe the variability of a patient’s reactions to pressure forces in the presence of the wide range of risk factors (Defloor, 1999). When tissue is compressed between a bony prominence and the surface where a patient is sitting or lying pressure is transmitted into the tissue from the surface and a counter pressure is exerted outwards from the bone. The positioning of the patient is therefore a key factor for the continuing blood supply to the tissue between the bony prominence and the skin (Defloor, 2000). A completely horizontal position has been shown to have the lowest interface pressure, with increasing elevation of the head resulting in both increasing pressure
and a shift in the peak interface pressure point. At 30° elevation, higher pressures are observed at the sacrum (Defloor, 2000; Krapfl & Gray, 2008). This pressure can currently only be measured at the interface but it is assumed there are pressure gradients occurring deeper within the tissues.

**Pressure Gradients**

The concept of pressure gradients is raised every so often in the clinical and bioengineering literature with the seminal writing being by McClemont in 1984 and taken up by others. McClemont (1984) described a situation where the opposing forces from the skin and the bone result in a cone-shaped pressure gradient (Maklebust & Sieggreen, 2001; McClemont, 1984). Within this cone (also called the McClemont cone) the external pressure can increase by a factor of between three and five if the pressure site sits over a bony surface, for example the sacrum. Fat and muscle have little tolerance for decreased blood flow and are more sensitive to pressure than skin (Ayello et al., 2008; Collier & Moore, 2006; Maklebust & Sieggreen, 2001). Internal deformation of the tissue will be affected by the different structure and mechanical nature of the bones and tissue layers. These differences result in a heterogeneous distribution of the deformation, areas of differing interface pressure within the tissue and the formation of pressure gradients (Oomens et al., 2009). The areas of differing pressure also give rise to shearing forces (Defloor, 1999; Gibson et al., 1976). The complex ways in which these shearing forces impact on pressure injury development will be examined in the following section addressing pathophysiology.
The ability to determine where these steep pressure gradients occur may also indicate areas subject to a higher risk of tissue distortion leading to capillary occlusion, and may therefore contribute to inform whether patients are at risk or not at risk of development of pressure injuries (Brienza, Geyer, Sprigle & Zułkowski, 2008; Rithalia, 2005). The application of pressure to tissue induces capillary collapse when the pressure is higher than the capillary pressure. The result of this applied pressure is that blood flow is interrupted and both the supply of oxygen and the ability to evacuate metabolic waste are reduced. Prolonged pressure can then lead to tissue necrosis which presents as a pressure injury (Allen, Ryan & Murray; 1994; Bader & Oomens, 2006; Defloor, 1999; Maklebust & Sieggreen, 2001; Roaf, 1976; Swain & Bader, 2004). Taking up the concept of a cone-shaped pressure gradient, destruction in the subcutaneous tissues may occur before damage to the skin surface is evident (Maklebust & Sieggreen, 2001).

**Interface Pressure Measurements**

In 2006 Bader and Oomens noted the important contribution that bioengineering can make in pressure injury research. This contribution has included the development of a range of interface pressure mapping systems. Many authors consider that interface pressure mapping has an important place in understanding pressure injury aetiology (Bader & Oomens, 2006; Swain & Bader, 2004). The visualisation provided by pressure maps has allowed the identification of areas at high pressure. It is also possible to use this mapping to calculate gradients and therefore identify potential areas of deep tissue injury. However, no empirical studies reporting exploration of this potential use of mapping technology
could be found in the published literature. High gradients are believed to suggest that the underlying tissue may be highly distorted and therefore more susceptible to pressure injury development (Krouskop, Noble, Brown & Marburger, 1986; Rithalia, 2005; Rithalia & Kenny, 2001; Shelton & Lott, 2003; Swain & Bader, 2004).

An improved appreciation of interface pressure distribution is also believed to be useful in obtaining a better understanding of the impact of support surfaces and the efficacy of pressure relieving devices (Rithalia, 2004; Shelton & Lott, 2003; Swain & Bader, 2004). The utility of interface pressure measurements is best summarised by Krouskop and Van Rijswijk (1995) who stated:

The only meaningful standard for pressures is to keep them as low as possible. This can be assessed using interface pressures. The lower the interface pressure, the lower the tissue pressure; and the lower the interface pressure gradient, the lower the pressure gradient in the tissue (p.35).

When using interface pressure mapping there are technical constraints such as a need for consistent positioning of study participants, better understanding of data acquisition and analysis, and easier display for interface pressure maps, particularly around the high risk body surface areas where the pressure gradient is high (Rithalia & Kenney, 2001; Shelton & Lott, 2003). In addition, interface pressure measurement systems require regular calibration to ensure that measurements are consistent and comparable (Allen, Ryan, Lomax & Murray, 1993).
The application of interface pressure mapping in the clinical setting is currently limited. As commented upon previously, interface mapping systems require regular calibration to ensure that the data acquired is accurate and hence comparable between scans for an individual, or between patients (Allen et al., 1993). Swain and Bader (2004) also note that significant time and effort is currently involved in data collection and analysis. It has been found in practice that these issues will impact upon nurses for the clinician utilisation of pressure mapping technology in three important areas namely:

1. A comprehensive set of guidelines would be required to be developed covering both the application and utilisation of the technology; and
2. Nursing workload and training would need to be augmented for use and interpretation of interface pressure mapping systems; or
3. Skilled technicians would need to be introduced into the clinical environment to correctly apply interface pressure mapping.

Gardner et al. (2006) have described pressure mapping systems as generally being too complicated, expensive, sensitive and labour intensive for clinical application. If these limitations could be overcome, or the benefits of interface pressure mapping be shown to outweigh these limitations, the clinical application of the technology should be further considered.
Pathophysiology

In this section the pathophysiology of pressure injuries will be addressed. The impact of pressure on tissue will be examined together with the main extrinsic and intrinsic factors that are known to affect the development of pressure injuries. Sites on the body where pressure injuries are most prevalent, and the means by which these injuries are classified, will also be described.

Development of Pressure Injury

The aetiology of pressure injuries is not fully understood (Bader & Oomens, 2006; Gebhardt, 2004; Rithalia, 2005; Swain & Bader, 2004). It is generally thought to be the result of a number of forces exerted on the tissues: these being pressure, shear and friction. Pressure injuries are generally believed to be the result of extended and/or repeated ischaemic attacks without adequate time for the tissue to recover thereby resulting in tissue necrosis (Gibson et al., 1976; Hagisawa et al., 2004; Swain & Bader, 2004).

There are two important factors in the causation of pressure injury, namely the level of the pressure applied and the duration over which the application occurs. An inverse relationship has been determined to exist between the level and duration of pressure, that is, low pressure for long periods as well as high pressure for short periods can cause pressure injury (Barnett & Ablarde, 1994; Brand, 1976; Collier & Moore, 2006; Defloor, 2000; Defloor, De Bacquer & Grypdonck, 2005; Oomens et al., 2009; Reswick & Rodgers, 1976; Stekelenburg et al., 2008).
The relationship between the external pressure applied to the patient’s skin and tissues and the effects of these pressures on the local microcirculation has been a key focus of research to date (Collier & Moore, 2006). Once the duration-pressure threshold is exceeded tissue damage continues even after the pressure is relieved. Repeated pressure is also important, particularly when repeated within a time period that is inadequate for the tissue to recover (Bader, 1990; Brand, 1976; Gibson et al., 1976; Hagisawa et al., 2004; Maklebust & Sieggreen, 2001).

Prolonged pressure is understood to cause ischaemic changes at and around the point of the pressure attack (Collier & Moore, 2006; Stekelenburg et al., 2008). Pressure affects all of the tissue between the external surface and the skeletal anatomy, but the greatest tissue destruction is at the bony interface. A prolonged localised pressure attack on tissue impairs the local blood supply and the lymphatic circulation thereby both limiting the supply of oxygen to the affected tissues and resulting in an accumulation of toxic materials in the same tissue (Bader, 1990; Defloor, 1999; Swain & Bader, 2004). Extended periods of these pressure-induced changes will result in cell necrosis, tissue breakdown and pressure injury development (Bader, 1990; Bader & Oomens, 2006; Collier & Moore, 2006; McClemont, 1984; Swain & Bader, 2004).

Once this pressure, or load as it is sometimes called, is removed there will be a large and sudden increase in blood flow through the tissue that has been ischaemic (Bader, 1990; Barnett & Ablarde, 1994). The reperfusion arises due to a reduction in the tissue resistance to blood flow, and has been reported by Collier and Moore (2006) as being as much as 30 times the resting value. The result can be
harmful effects and the establishment of what has been variously described as an ischaemic or reperfusion injury (Bader & Oomens, 2006; Swain & Bader, 2004) or a superficial pressure injury (Nixon, Cranny & Bond, 2005).

As noted above, while pressure is widely believed to be a major factor in pressure injury development, the exact mechanisms that cause the development are not understood. There is a developing view that a number of different aetiologies are at work and pressure injuries can initially develop both at the skin surface and deep within the tissue structure; the deep tissue injury (Baharestani et al., 2009; Maklebust & Sieggreen, 2001; Nixon et al., 2005; Oomens et al., 2009; Stekelenburg et al., 2008). It is not currently possible to delineate the point at which alterations to intact skin indicate irreversible damage within (Nixon et al., 2005). The concept of a deep tissue injury may be better understood in the context of cone-shaped pressure gradients described earlier and will now be explored in more detail.

In addition to pressure, extrinsic factors such as shear appear to be particularly damaging to tissue. Barton (1976) noted that pressure injuries were due to two concurring factors, namely pressure in excess of capillary closing pressure and damage to the blood vessels as a result of shear. High pressure gradients have been determined to generate large shear forces and hence contribute to breakdown of the skin (Mueller, Zou and Lott, 2005; Rithalia, 2005). Swain and Bader (2004) have also highlighted the effect on cells and subsequent cell breakdown are more pronounced at the edges of an area of compression where the pressure gradients are greatest.
A deep tissue injury can be considered as originating deep within the tissue rather than at the surface and assessment of pressure injury size must take into consideration the possibility of unseen necrosis in the area of the pressure gradient (Ankrom et al., 2005; Maklebust & Sieggreen, 2001). Whilst there is very limited empirical research conducted about deep tissue injuries, making them an important area for additional study, there is agreement about the propensity for injuries to develop in this way. This clinical consensus can be summarised as agreement that:

1. magnification of pressure probably occurs through the McClemont pressure cone (McClemont, 1984) or a similar mechanism;
2. blood vessels in the deep tissue run generally parallel to the skin surface and are therefore more easily occluded (Collier & Moore, 2006);
3. muscle and fat are also susceptible to pressure induced damage (Ayello et al., 2008; Bader & Oomens, 2006; Maklebust & Sieggreen, 2001; McLeod, 1997);
4. muscle and fat are closer to bony prominences and therefore more susceptible to distortion and deformation (McLeod, 1997); and
5. these effects listed above may be compounded by shear (Collier & Moore, 2006; McLeod, 1997).

Whilst the causes of tissue necrosis are not fully understood there is a general acceptance that the most common sites for pressure injury development are the bony prominences where maximum tissue compression occurs and skin blood flow is consequently decreased due to pressure (Bader & Oomens, 2006; Collier &
Hagisawa et al. (2004) noted that the most susceptible areas are the sacrum when in the supine position, and the ischial tuberosities when sitting. McClemont (1984) states that the most common sites for pressure injury development include “the sacrum, ischial tuberosities, the heels and the greater trochanters” (p.1). In data collected within European countries during a 2001/2002 study, the sacrum was determined as the most common site for pressure injuries, representing more than 25% of recorded injuries (Clark et al., 2004). The heel was the second most common site, also representing almost 25% of injuries. Clark et al. (2004) write that the typical site for the most severe pressure injuries is the sacrum. Severity is usually measured using the staging classification described earlier with ‘severe’ being applied to Category 3 and 4 injuries.

In summary this section has discussed the mechanisms through which pressure impacts upon tissue can result in the development of a pressure injury. The concept of a deep tissue injury, originating deep within the tissue rather than at the surface and thereby being largely unseen and difficult to detect, has also been introduced. The most common sites for the development of pressure injury have been reviewed. The following sections will discuss the extrinsic and intrinsic factors that are most widely believed to affect pressure injury development.
Extrinsic Factors Affecting Pressure Injury Development

As will be evident from the literature review so far, the development of pressure injury is a complex process and arises differently for different individuals, being affected by a multitude of factors both within the individual (intrinsic) and external to the individual (extrinsic). Apart from pressure, which has already been discussed, the most commonly recognised extrinsic factors are friction and shear (Bale & Jones, 2006; Ousey, 2005; Ousey, 2009). Moisture is another important factor that is included within the concept of skin microclimate, which is becoming more widely considered (Clark et al., 2010; Orsted, Ohura & Harding, 2010). Skin microclimate is primarily thought of as an extrinsic factor. The key extrinsic factors are now presented in more detail.

Shear

Shear is a mechanical stress that operates parallel to the skin surface (Ayello et al., 2008; McLeod, 1997; Reger et al., 2010; Takahashi et al., 2010) with the effect occurring predominantly in the deep tissues and resulting in obstructed, torn or stretched blood vessels (Defloor, 1999; Maklebust & Sieggreen, 2001). When a high level of shear is present the amount of external pressure necessary to produce vascular occlusion is about half the amount needed when shear is not present (Bennett, Kavner, Lee & Trainor, 1979). Shearing also decreases the amount of time that tissue can remain under pressure before ischemia occurs (Maklebust & Sieggreen, 2001).
Shear is greatly affected by patient position (Defloor, 1999) and a clinical example is commonly used to illustrate the effect. When the head of a bed is elevated a greater compressive force is placed on the sacral tissues than when the bed is in the flat position. The patient’s body weight pulls the tissues attached to the bone in a downward direction while the skin stays stationary on the bed linen (Brienza et al., 2008; Collier & Moore, 2006; Gibson et al., 1976; Maklebust & Sieggreen, 2001; Scales, 1976).

Shear can be differentiated from pressure in that the two forces act in different directions with respect to the skin surface. At the same time a number of authors have highlighted the fact that shear and pressure accompany one another (Defloor, 1999; Reger et al., 2010; Takahashi et al., 2010). Moreover, highly localised pressure will compress tissue and distort adjacent tissues thereby creating shearing forces. A recent consensus document (Reger et al., 2010) has highlighted how steep pressure gradients and shear can combine to increase or reduce the amount of distortion within the skin.

**Friction**

Friction occurs when one surface moves across another (Maklebust & Sieggreen, 2001; Ousey 2009; Oomens et al., 2009; Reger et al., 2010). Friction tends to keep the skin in place as the body moves thereby creating shear stresses in the soft tissues overlying bony prominences (Reger et al., 2010). Maklebust and Sieggreen (2001) have noted that friction is a precursor to shear and therefore increases the potential for deeper tissue damage.
While friction is not considered to be a primary factor in pressure injury development, it will exacerbate the stripping of broken epidermis or even result in an initial skin surface breakage (Collier & Moore, 2006). In skin that is dry, friction can exacerbate moisture loss from the skin cells (Ousey, 2009). If the supporting surface is moist, the impact of shear may be heightened due to an increased friction coefficient between the skin and the surface (Collier & Moore, 2006). The condition of this supporting surface and the adjoining skin is known collectively as the skin microclimate. Skin microclimate is a concept that was discussed in the 1970s (Roaf, 1976) but has been overlooked in the intervening decades (Clarke et al., 2010)

**Skin Microclimate**

For pressure injury development the microclimate refers to skin surface temperature and moisture at the body-surface interface (Clark et al., 2010; Rapp, Bergstrom & Padhye, 2009). Tissue moisture is a recognised factor in the development of pressure injury and is believed to change the resilience of the epidermis (AWMA, 2001). Moist skin has been shown to have a higher friction coefficient than dry skin (Bale, 2005; Collier & Moore, 2006) and, according to Maklebust and Sieggreen (2001), is five times more likely than dry skin to suffer from a pressure injury.

Increases in body temperature generally have been shown to increase tissue oxygen requirements and therefore increase pressure injury risk (Defloor, 1999).
Clark et al. (2010) highlight the need for additional research in order to fully understand how microclimate and related factors influence pressure injury development, and whether qualitative measures can be developed to indicate increased risk for pressure injury. The conclusion to be drawn is that microclimate management should aim to avoid extremes of both skin temperature and moisture.

**Intrinsic Factors Affecting Pressure Injury Development**

Apart from the extrinsic factors discussed in the preceding section, the predisposition of an individual to pressure injury development will also be affected by a large number of intrinsic factors which affect the load-bearing capacity or tolerance of the tissue (Defloor, 1999). The main, widely accepted, intrinsic risk factors are mobility, age and nutritional status (Collier & Moore, 2006; Defloor, 1999; Maklebust & Sieggreen, 2001; Swain & Bader, 2004).

**Mobility**

Immobility and diminished activity are the most commonly identified risk factors in studies and are considered primary risk factors in the development of pressure injuries (Collier & Moore, 2006; McClemont, 1984; Ousey, 2005; Takahashi et al., 2010). In non-impaired patients the effects of continuous pressure signal small changes in body movement and positioning to relieve the load and to restore normal blood flow and tissue perfusion (Maklebust & Sieggreen, 2001; McLeod, 1997; Takahashi et al., 2010). Impairment of the nervous system or a reduced ability to respond to discomfort or pain predisposes an individual to
prolonged and intense pressure. Hence mobility relates directly to an ability to achieve pressure relief (Ayello et al., 2008). The regular relief of pressure is therefore commonly considered to be the single most important factor in avoiding pressure injury development (Collier & Moore, 2006; Defloor, 1999; Ferguson-Pell, Bell & Evans, 1976; Krapfl & Gray, 2008; McClemont, 1984; Roaf, 1976). A study conducted on 838 patients in a geriatric setting by Defloor et al. (2005), with turning rates between 2 hours and 4 hours, determined that more frequent turning lowered the incidence of pressure injury in patients incapable of mobility. The use of pressure relieving and reducing devices have also been determined to be relevant in this regard and are discussed later in this chapter.

**Age**

Increasing age is a major risk factor for the development of pressure injury (Clark & Stephen-Haynes, 2005; Collier & Moore, 2006). During the ageing process the epidermis thins, sweat glands become fewer in number, dermal proteins reduce, blood vessels become thinner and more fragile, and pain sensitivity reduces (Baranoski, Ayello & Tomic-Canic.; 2008; Brienza et al., 2008; Maklebust & Sieggreen, 2001). The dermis also thins by up to 20% as collagen and elastin levels reduce and muscles lose their tone. Defloor (1999) has noted that as elastin decreases pressure is conducted more directly to the interstitial liquid and to the cells. As ageing occurs the blood supply reduces, skin elasticity is reduced, and the skin becomes stiffer and less pressure-resistant due to reduced mechanical properties (Alexander & Cook, 1976; Bale, 2005; Defloor, 1999; Morris, 2005; Reger et al., 2010).
Older age is also associated with increased risk of chronic illness, poor peripheral perfusion and loss of peripheral sensation. The sum of these factors means that the skin of the older person is more sensitive to pressure, friction and shear. Defloor (1999) has noted that the pressure distribution capacity of tissue correlates negatively with age, with the result that elderly persons are therefore at a higher risk of developing a pressure injury (Baranoski, Ayello & Tomic-Canic, 2008).

**Nutritional Status**

Nutritional status is an important contributor to both the risk of developing a pressure injury, and to the severity and extent of that injury (Collier and Moore, 2006; Cuddigan, 2008; McClemont, 1984; Ousey, 2010; Posthauer & Thomas, 2008; Schols et al., 2006). Considerable research has been undertaken into many aspects of nutritional status related to propensity for pressure injury and this is well summarised in recent guidelines. The NPUAP consensus conference in 2010 (Black et al., 2011) agreed that severe malnutrition alters tissue tolerance and therefore increases the risk of pressure injury development. Patients with poor nutritional status and corresponding loss of subcutaneous tissue, have been found to be more susceptible to pressure injuries as underlying bony structures become more prominent (McClemont, 1984; Swain & Bader, 2004). As an example, a study conducted in France by Meaume, Colin, Barrois, Bohbot and Allaert (2005) found that patients placed on nutritional supplements had a lower incidence of pressure injury development than those who were not provided with such supplements).
In summary, a wide range of extrinsic and intrinsic factors have been determined to have an impact on pressure injury development, although exactly how these factors are linked remains unclear (Clark, 2004; Collier & Moore, 2006; Defloor, 1999). Further research is therefore required on pressure injury factors, both individually and collectively, to better understand the impact on pressure injury aetiology. Development of a conceptual framework helps to identify where gaps still occur and two frameworks will now be reviewed. Before this review, the classification of pressure injury severity will be briefly described and appraised.

**Pressure Injury Severity and Classification**

Pressure injuries are classified to provide an indication of severity (Dealey & Lindholm, 2006; Nixon et al., 2005). Dealey and Lindholm (2006) also note that these measures of severity make an important contribution to prevalence studies and hence to assessing the effectiveness of preventative strategies. As already discussed, multiple classification systems, often multi-faceted and sophisticated, have been published since the system advanced by Shea in 1975 (Dealey & Lindholm, 2006). The most commonly used classification system is the one originally published by the NPUAP in 1992 (Dealey & Lindholm, 2006), and now included in the combined NPUAP-EPUAP Guidelines the latest version of which was updated in 2009. In Australia the AWMA (2001) published a classification system consistent with that advocated by the NPUAP in 1992.

The 2009 NPUAP-EPUAP Guidelines recognise that different terms to describe the stage or grade of a wound have been used by each body separately and
now classify pressure injuries as categories/stages that address the depth of the wound (Baranoski, Ayello & Langemo, 2008). In this study the term ‘category’ will be used to describe the classification of pressure injuries. The category descriptions are detailed in Table 1 below.

**Table 1. NPUAP & EPUAP (2009) Pressure Injury Category Descriptions**

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category I</td>
<td>Intact skin with non-blanchable erythema of a localised area, usually over a bony prominence. Discolouration of the skin, warmth, oedema, hardness, or pain may also be present. Darkly pigmented skin may not have visible blanching.</td>
</tr>
<tr>
<td>Category II</td>
<td>Partial thickness loss of dermis presenting as a shallow open ulcer with a red/pink wound bed, without slough. It may also present as an intact or open/ruptured serum-filled or sero-</td>
</tr>
<tr>
<td>Category III</td>
<td>Full thickness loss. Subcutaneous fat may be visible, but bone, tendon, or muscle are not exposed. Some slough may be present. The injury may include undermining and tunnelling.</td>
</tr>
<tr>
<td>Category IV</td>
<td>Full thickness loss with exposed bone, tendon or muscle. Slough or eschar may be present and undermining and tunnelling is often included.</td>
</tr>
</tbody>
</table>

Note. NPUAP & EPUAP Pressure Ulcer Prevention and Treatment Clinical Practice Guidelines 2009

Classification systems however have a number of reported weaknesses, including the lack of supporting evidence to justify the classifications made (Dealey & Lindholm, 2006; Nixon et al., 2005). Category I injuries are of particular current interest as these are seen as precursors to pressure injury development where damage to the underlying tissues has not yet occurred. Category I injuries represent the most easily reversible level of damage. The validity of this classification to people with darkly pigmented skin is the subject of significant debate.
In addition to the system as described above, there are a number of additional categories thereby demonstrating that the current four classification system does not cover all known types of pressure injury. For example, the NPUAP-EPUAP Guidelines (2009, p.17) include two additional categories for the USA of “Suspected Deep Tissue Injury- Depth Unknown” and “Unstageable: Full thickness skin or tissue loss - Depth Unknown”. Further, the classification of ‘Unavoidable’ has been discussed by the NPUAP and consensus reached that not all pressure injuries are avoidable (Black et al., 2011). This consensus supports a personal account by Dr Randall Duffield (1999) of his experiences with a pressure injury whilst hospitalised. His summary was that, on reflection, his pressure injury was within the small percentage that is believed to be largely unavoidable (Black et al., 2011; Hietanen, 2006; Torra i Bou et al., 2006).

Inter-rater reliability has been highlighted as particularly important in classifying wounds as the allocation of various descriptions of tissue damage requires the ability to recognise and assess the affected tissue. The application of a classification system therefore continues to require clinical experience and judgement (Dealey & Lindholm, 2006; Defloor & Schoonhoven, 2004). Accurate assessment is required for prevalence studies, for the identification of those at risk, and for the development of pressure injury preventative and management strategies (Moore, 2005).
Conceptual Framework

There is consensus that the extrinsic and intrinsic factors previously described are all associated with the development of a pressure injury. One method for bringing these factors together and exploring their inter-relationships is through the development of a conceptual framework, a limited number of which have been developed. In 1987 Braden and Bergstrom presented the framework (see Figure 1 below) that formed the basis of the Braden Scale for pressure injury risk assessment. The Braden and Bergstrom framework has been adopted by the AWMA and used in the AWMA Clinical Practice Guidelines (2001) to describe pressure injury development.
The other very widely used risk assessment tool, the Waterlow Risk Assessment Tool, does not have an explicit conceptual framework. Waterlow has however acknowledged the concept of the McClemont ‘cone’ in her explanation of pressure injury aetiology (Waterlow, 2005b).

Defloor (1999) presented an alternative conceptual framework (see Figure 2) that placed tissue tolerance as an intermediate variable in pressure injury development rather than a causal factor as is the case in the Braden-Bergstrom framework. This framework by Defloor (1999) also explicitly recognised that medical and nursing interventions are important contributing factors for the duration of pressure and shearing forces. It is argued by Defloor (1999) that a
conceptual framework assists with understanding the pathophysiology of pressure injuries resulting in improved understanding of overall risk, development of hypotheses about the effectiveness of preventative measures, testing of these measures and finally the associated implementation of management and prevention strategies.

**Figure 2.** Defloor’s Conceptual Scheme for Prediction and Prevention of Pressure Sores (1999)

**Prevention**

Defloor’s (1999) conceptual framework is focused on prevention as well as prediction and the focus of this review now turns to prevention. The approach taken to prevent the development of pressure injury has remained fundamentally unchanged for centuries, namely to identify patients deemed to be at risk and to
relieve pressure (Exton-Smith, 1976; Gebhardt, 2004). Prevention (rather than treatment once an injury is established) is the most efficient method to manage the pressure injury problem in both human and economic terms. Prevention is achieved either through the use of regular manual repositioning or through the provision of a pressure re-distributing support (Clark, et al., 2004; Defloor, 1999; Nixon, Cranny & Bond., 2007; Ousey, 2009; Roaf, 1976).

This section will cover the methods by which pressure injury prevention is addressed, beginning with the role of clinical practice guidelines. The section will include the assessment of pressure injury risk and the application of risk assessment tools, with particular emphasis on the Waterlow Risk Assessment Tool. Finally the role and importance of pressure relieving devices will be considered.

**Clinical Practice Guidelines**

Clinical practice guidelines have been progressively developed by national and international bodies since that of the NPUAP in 1992 to assist patient and practitioner decisions about healthcare (NPUAP & EPUAP, 2009). In Australia these pressure injury guidelines were first published in 2001 (AWMA, 2001). These are currently being revised and are due to be relaunched in late 2011. Clinical practice guidelines recognise the scope of the pressure injury problem, provide prevention and treatment recommendations, and provide indications about the strength of the evidence that is currently available to underpin these recommendations (AWMA, 2001; NPUAP & EPUAP, 2009). In developing the guidelines, the authors consider the available evidence and provide consensus
results for use by clinicians (Cherry, 2006; van Zelm et al., 2006). Clinical practice guidelines include one or more pressure injury classification systems as previously discussed.

For clinicians and managers guidelines provide a structured framework through which to address all facets of the pressure injury problem. Unfortunately, the level of evidence for the majority pressure injury prevention recommendations is noted as Level C; that is supported by indirect evidence only. This low evidence level status reinforces comments made by Gebhardt (2004) regarding the slow progress towards understanding pressure injury aetiology and preventive strategies. One result of this limited understanding is a continuing reliance on clinical judgement (Bader & Oomens, 2006; Stekelenburg et al., 2008).

**Pressure Injury Risk**

As the most effective means of addressing pressure injuries is to prevent their occurrence the identification of those patients at risk is critical. Collier and Moore (2006) simply define risk as the probability of a patient developing a pressure injury. The determination of risk is a key first step in prevention strategies, as different combinations of risk factors can be important for the development of pressure injuries for patients with compromised health (Ayello et al., 2008; Papanikolaou et al., 2002; Stekelenburg et al., 2008) and a variety of management strategies may be needed to prevent pressure injury development. Key extrinsic and intrinsic risk factors have been discussed in previous sections although Defloor (1999, p.206) makes the comment that “so many risk factors have been documented
that one easily loses track”. This section will address the means by which risk is typically assessed.

**Risk Assessment**

Risk assessment is a process by which the various factors affecting pressure injury development are considered and the patient’s predisposition to these injuries is determined (Defloor & Grypdonck, 2004; Kottner & Balzer, 2010; Pancorbo-Hidalgo et al., 2006). As previously indicated this process still relies extensively on clinical judgement but is typically undertaken in conjunction with the utilisation of a risk assessment tool. A variety of risk assessment tools have been developed and implemented in clinical practice since the early 1960s, typically with minimal research associated with their development (Dealey, 2004). The most commonly used risk assessment tools are by Braden, Norton and Waterlow (Torra i Bou et al., 2006). Waterlow (2005a) has however noted that the Waterlow Risk Assessment Tool and the Norton system have been designed to determine risk, whereas the Braden scale was designed to predict pressure injury development. These fundamental differences need to be considered when comparing the efficacy of the various systems.

Risk assessment tools consider the pressure injury risk factors within a documented structure. The tools are typically conceptually similar in structure as they include a similar range of factors considered to be important in pressure injury development. These factors differ however in assumed importance and hence in the weighting ascribed in the various assessment systems. Factors are assigned
scores and risk level is determined by summation (Kottner & Balzer, 2010; Papanikolaou et al., 2002; Papanikolaou, Lyne & Lycett, 2003). The tools are designed to aid a clinician’s judgement for the identification of patients at risk of developing a pressure injury and hence to apply preventative measures (Baharestani et al., 2009; Bell, 2005; Clark, 2004; Cuddigan, 2008; Jull & Griffiths, 2010; Kottner & Balzer, 2010; Ousey, 2010; Torra i Bou et al., 2006). Risk assessment tools are not an intervention although it is hoped that improved insight into risk factors and the way in which these affect each other provides better identification of those who are genuinely at risk.

Risk assessment tools have themselves been the subject of assessment, with the major assessed factors being sensitivity and specificity. Sensitivity is the ability of the tool to correctly identify a true characteristic. Specificity is the extent to which the tool correctly identifies the absence of a characteristic. An effective risk assessment tool should therefore have both high sensitivity and high specificity (Bell, 2005; Defloor & Grypdonck, 2004; Torra i Bou et al., 2006). Jull and Griffiths (2010), Defloor and Grypdonck (2004) and Smith et al. (1995) also argue that evaluation of pressure injury risk assessment tools based on sensitivity and specificity alone is compromised as nursing interventions are inevitably involved as a result of the outcome from the tool and these also need to be considered.

A problem with risk assessment tools in general is that they need to include all relevant risk factors, and not include those that are not considered relevant. That is, they need to be specific to the risk being assessed (Smith, Forster & Young, 2006). Criticisms of pressure injury risk assessment tools include both that (a) they...
do not account for all risk factors (Stekelenburg et al., 2008) and that (b) they can be too complex (Anthony et al., 2010; Torra i Bou et al., 2006). Smith et al. (1995) have stated that current risk assessment tools do not prevent the application of prevention measures to patients not in need of them or not applying them to vulnerable patients. These problems have also been demonstrated in other areas of health such as falls (Smith et al., 2006).

Anthony et al. (2010) determined that whilst the tools do measure risk, somewhat paradoxically, there is no evidence that their use actually reduces pressure injury. Risk assessment is simply a first step in a chain of actions to put in place appropriate preventative measures, to continue to evaluate skin condition and to modify clinical measures being taken.

A range of authors have suggested that identified weaknesses with the various tools can be mitigated with the exercise of professional judgement in parallel with their use (Bell, 2005; Gardner et al., 2006; Ousey, 2009). Gebhardt (2004) suggested that no risk assessment tool has proved itself more adept in identifying patients at risk of developing a pressure injury than professional judgement. Finally, Defloor and Grypdonck (2004) have suggested that the main benefit of these tools may be to simply act as a reminder to nurses that pressure injury development is possible.

Regardless of the debate around their efficacy, completed pressure injury risk assessment using existing tools is currently required both in acute and long stay
healthcare facilities both in Australia and internationally. The Waterlow Risk Assessment Tool is the preferred tool in the ACT and is now described briefly.

**Waterlow Risk Assessment Tool**

The Waterlow Risk Assessment Tool was developed by Judy Waterlow in the United Kingdom in 1985. It is a multi-variable tool that assesses the patient according to pre-defined demographic, health and behavioural factors to determine a risk score (Anthony et al., 2010; Kottner & Balzer, 2010). The variables used are weight and build, continence, skin type, mobility, gender and age, and appetite, and includes the consideration of special risks associated with tissue malnutrition, neurological deficits, surgery/trauma and special medications. These categories enable the scorer to complete a detailed clinical assessment of the patient. The operational details of this instrument are presented in more detail in the Methods Chapter. Its sensitivity, specificity and acceptance by nurses and other health professionals is appraised here.

The Waterlow Risk Assessment Tool has been described as complex, with a tendency to classify more patients at risk than those who might actually be at risk (Torra i Bou et al. 2006), and as a poor predictor of pressure injury occurrence (Boyle & Green, 2001). Thus, the assessed sensitivity of the Waterlow tool by Torra i Bou et al. (2006) is 89%, with specificity assessed at 29%. The low specificity value has been used to explain why preventative measures are often applied to patients who do not actually need them, with commensurate cost and nursing impacts (Pancorbo-Hidalgo et al., 2006). Judy Waterlow (2005a) noted the
importance of being systematic when undertaking the risk assessment, and suggested that the score is not the end point, but rather the precursor to a broader question of how the risk can be reduced. The Waterlow Risk Assessment Tool has been periodically upgraded, with the latest version being updated and published in 2005. Despite its lack of theoretical framework and low specificity it is still widely used internationally.

**Body Shape**

Body shape is of increasing interest to researchers as an indicator of risk, although the level of focussed research has been limited. Interest to date ranges from the potential to link posture to interface pressure in seated patients (Riley & Bader, 1988) through to general use as an indicator of overall health status and how changes in shape may influence disease risk (Wells et al., 2007). Swain and Bader (2004) also suggest that loss of muscle tone in at risk patients will result in a change of body shape. Defloor (1999) acknowledges body build as a pressure injury risk factor.

Body shape has been found to have an effect on interface pressure, as shape is affected by skeletal structure, the musculature structure including muscle tone, and the amount of subcutaneous fat (Swain & Bader, 2004). Lindan and Greenway (1965) showed that obese patients had greater areas of increased pressure but lower peak interface pressures. The same study showed higher peak interface pressures were recorded with cachectic patients in comparison to those of normal weight. Swain and Bader (2004) report however that no link has been discerned between
weight and interface pressure, or between Body Mass Index (BMI) and interface pressure. Further research into the area of interface pressure and body type is therefore required.

**Pressure Relieving Devices and Support Surfaces**

As already indicated, regular re-positioning and use of effective pressure relieving devices are considered to be most effective means for the prevention of pressure injury (Defloor et al., 2005; Defloor et al., 2006; Gunningberg, 2005; Rithalia, 2004). Although products are available from many health industry providers, the science associated with the evaluation of support surfaces is still developing and there are few guidelines to indicate the level of pressure reduction required for specific areas of the body (Gebhardt, 2004; Gunningberg, 2005; Rithalia & Kenney, 2001).

The properties of immersion and envelopment are important for all pressure distributing devices (Takahashi et al., 2010). Immersion is the degree to which a patient will sink into a surface, whilst envelopment refers to how well the surface moulds to body contours. Whilst high immersion and envelopment are associated with higher pressure redistribution, they also impact on patient mobility and independence.

One of two types of devices is typically employed in pressure injury prevention. Constant low pressure devices are designed to relieve pressure by moulding around the shape of the patient’s body to increase the support area and
thereby reduce tissue interface pressure. Examples of constant low pressure devices include foam or fibre-filled mattresses overlays, waterbeds and gels which are non-powered, non-mechanical and remain motionless except in response to patient’s movement (Fletcher, 2006; Rithalia, 2004).

Alternating pressure devices reduce the duration of the pressure by alternating high and low pressures between the patient’s body and support surface (Cullum, Deeks, Sheldon, Song & Fletcher, 2003; Fletcher, 2006; Gunningberg, 2005; Takahashi et al., 2010; Thompson-Bishop & Mottola, 1992). Alternating pressure devices such as overlays or mattresses provide cyclic inflation of air cells placed transversely across the mattress surface. They have moving parts and require an electric power source.

The structure of a mattress is a factor in the dispersion of pressure. Defloor (2000) estimates that a non-pressure reducing mattress only supports 10%-20% of the body, whereas many pressure-reducing mattresses spread the load of the body by conforming to the body shape. Defloor (2000) also notes that, for a person weighing 80kg, the average interface pressure is between 30 and 60 mmHg, with the pressures under bony prominences being much higher. Cullum et al. (2003) and Gunningberg (2005) both argue that there is insufficient evidence to definitively debate the relative merits of alternating pressure versus constant low pressure mattresses. Earlier studies conducted in the USA, however, have provided results that indicate that low-air-loss beds are superior to pressure reduction mattresses and overlays in achieving a reduction in interface pressures (Ferrell, Osterwell & Christenson, 1993; Mulder et al., 1994). The 2009 NPUAP & EPUAP Guidelines
indicate the there is no evidence of the superiority of any higher-specification foam mattress over another.

The evidence that is currently available shows that patients determined to be at risk of developing a pressure injury should not be placed on an ordinary foam mattress as higher specification pressure relieving mattresses have been shown to reduce the incidence of pressure injury in patients deemed at risk (Gunningberg, 2005; Legood & McInnes, 2005). The economic benefits to the health care organisation due to the reduction in pressure injuries from the use of higher specification foam mattresses have also been demonstrated (Legood & McInnes, 2005). The ability for a clinician to implement a choice of mattress as part of a prevention strategy is however often limited by organisational accessibility and processes for acquisition of devices.

**Gaps in Knowledge**

The preceding review of the literature has highlighted that, whilst progress is being made, significant knowledge gaps exist in all aspects of the understanding of pressure injury. These gaps exist across all areas including aetiology and pathophysiology, risk factors and the way that these alter and interact from person to person, as well as methods of risk assessment and approaches for prevention. Bader and Oomens (2006) speak for many researchers when they suggest that the limited knowledge available means that choice of preventative aids and risk assessment techniques are still dominated by subjective measures.
One area of particular interest that has been identified centres on interface pressure and in particular the role that may be played by pressure gradients around a peak interface pressure point. Whilst pressure gradients are known to be important in the development of a pressure injury (Brienza et al., 2008) the relationship of pressure with other effects such as shear has yet to be rigorously examined (Swain & Bader, 2004). It has been found that there is very little empirical research available exploring factors such as shear forces and associated tissue deformation. A related area where knowledge is limited is that of deep tissue injury. Suspected deep tissue injury has recently been recognised as a separate classification in the NPUAP and EPUAP (2009) Guidelines. The role of deformation in the formation of deep tissue injury is suspected (Stekelenburg et al., 2008) and as noted by Defloor (1999, p.211) “almost all pressure is passed to the underlying tissue” over bony prominences where the skin covering can be very thin.

Another area where knowledge is lacking is risk assessment, both in terms of the overall efficacy of the tools themselves and with respect to the interplay and completeness of the various risk factors. This issue has been commented upon by Defloor (1999) who has noted that developments in this field would provide better detection of patients who are genuinely at risk. In addition improved risk assessment may be achieved through the consideration of factors that have not been included in risk assessment tools to date. One of these is body shape. Swain and Bader (2004) and Wells et al. (2007) note that shape may be a marker of overall health and that shape is likely to change with varying muscle tone and tissue
tolerance. The relationship of both shape and changes in shape to pressure injury risk is unclear.

**Summary**

This chapter has reviewed the current literature pertaining to pressure injury development and has considered a range of relevant aspects. These include the nature of skin and how pressure injuries are believed to develop, risk factors that affect a person’s propensity to pressure injury, means of assessing risk, and preventative techniques. The review has shown that significant gaps continue to exist in the knowledge base.

The literature reviewed in this chapter has highlighted the importance of understanding how risk factors and the combination of these may affect each individual differently. Improvements in risk assessment, including those factors that may not be currently included in assessment tools, may lead to the better identification of those persons actually at risk and the improved tailoring of management strategies. Reductions in pressure injury prevalence can be seen to be fundamentally linked to improved prevention management, techniques and equipment. The ability to target limited healthcare resources to those actually requiring preventative assistance is therefore critically impacted by the ability to assess risk. Any improvement in risk assessment will therefore have a positive effect on patients, healthcare providers and health administration.
New technologies offer potential for improvement in the reduction of pressure injury prevalence rates. The application of interface pressure mapping technology, and the way in which this might be applied for pressure injury risk reduction will be explored in the remainder of this thesis.
CHAPTER THREE - METHODS

Introduction

The literature review reported in the previous chapter has confirmed that pressure injuries are a significant problem in terms of both human suffering and as a significant financial cost to health care facilities both nationally and internationally. Nurses currently conduct assessment for pressure injury risk through use of approved risk assessment tools in conjunction with clinical judgement. However, the fact that many patients still develop pressure injuries suggests that both the assessment of risk and the preventative interventions applied need further development.

A number of authors (Stekelenburg et al., 2008; Swain & Bader, 2004) have shown that there are differing skin responses to pressure. Bader and Oomens (2006) have suggested that whilst pressure measurements are important they cannot be used alone to predict vulnerable tissue areas or identify patients at risk. Current risk assessment tools consider a wide range of contributing factors but do not include pressure. The application of interface pressure mapping systems to provide a visual display of pressure at the skin surface may be one specific technology that can enhance pressure injury risk assessment.

This thesis will contribute to the body of knowledge regarding pressure injury risk by providing additional information. The use of interface pressure mapping provided both numerical measurements and visualisation of the pressure at
the interface of the skin and the mattress surface. Once obtained, these measurements allow for the exploration of relationships that may exist between the interface pressure and the pressure injury risk.

This chapter will begin with a statement of the study aims and research questions, and will then describe the methodology used in this study, including the research design, population and samples, data collection and management, the methods used for analysis, and ethical considerations. The results of the analysis will be presented in the following chapter.

**Relationship to the Mapping and Intervention for Prevention of Pressure Injury Project**

This study was conducted as a ‘follow-on’ to the MIPPI project undertaken by the RCNMP at The Canberra Hospital between July 2004 and April 2005. Data collection was undertaken as part of the MIPPI project aim to investigate the relationship between risk factor scores, interface pressure and capillary blood flow (Gardner et al., 2006). The main project was a controlled trial comparing interface pressures generated in the sacral region on a standard hospital mattress and two types of intervention mattress. The baseline measurements using the standard mattress were used for the study as described in this thesis. I was part of the MIPPI investigating team and fulfilled the role as project coordinator. Within this role I was responsible for the day-to-day activities within the MIPPI project, the training and management of the data collectors, and the acquisition and initial data reduction.
Study Aims and Questions

The baseline MIPPI data provided a rich source of information about interface pressures of patients when cared for on standard hospital mattresses. This study ‘Exploring known risk factors for pressure injury with visual technology’ further analysed this data as described herein to address the study aims, namely:

1. Exploration of correlations between two interface pressure mapping indices and selected risk factors for pressure injury, and
2. Exploration of the visual anatomical characteristics of the buttock region of patients through the use of interface pressure mapping and the correlation between shape and selected risk factors for pressure injury.

A series of questions were explored with regard to the pressure injury risk factors and two interface pressure mapping indices; namely weight, BMI, the Waterlow Risk Assessment score, the peak interface pressure and the pressure gradient. The questions investigated were grouped to accord with the Defloor (1999) conceptual framework presented in the literature review, and were as follows:

Pressure related questions

What is the relationship between Peak Interface Pressure and Weight?
What is the relationship between Peak Interface Pressure and BMI?
What is the relationship between Peak Interface Pressure and Risk Score as determined by the Waterlow Risk Assessment Tool?

What is the relationship between Peak Interface Pressure and Gradient 1.5cm?

What is the relationship between Peak Interface Pressure and Gradient 2.5cm?

Shear related questions

What is the relationship between Gradient 1.5cm and weight?

What is the relationship between Gradient 1.5cm and BMI?

What is the relationship between Gradient 1.5cm and Risk Score as determined by the Waterlow Risk Assessment Tool?

What is the relationship between Gradient 2.5cm and weight?

What is the relationship between Gradient 2.5cm and BMI?

What is the relationship between Gradient 2.5cm and Risk Score as determined by the Waterlow Risk Assessment Tool?

What is the relationship between Gradient 2.5cm and Gradient 1.5cm?

Tissue tolerance related questions

What is the relationship between BMI and Risk Score as determined by the Waterlow Risk Assessment Tool?

What is the relationship between weight and BMI?

What is the relationship between weight and Risk Score as determined by the Waterlow Risk Assessment Tool?
Design

This was an exploratory study utilising a prospective correlational design. Correlational studies enable examination of the relationship between pairs of variables, as well as comparison between groups. This design allows quantification of the strength of the relationship between the variables, as well as the direction of the relationship – that is, whether the association between variables is direct or inverse (Schneider, 2003).

The study investigated the correlation between two interface pressure mapping indices and selected risk factors for pressure injury, and explored the visual anatomical characteristics of patients and the correlation between shape and selected risk factors for pressure injury. The pressure injury risk factors were weight, BMI, and the risk scores as determined by the Waterlow Risk Assessment Tool. The interface pressure mapping indices were peak interface pressure and the pressure gradient, measured at 1.5cm and 2.5cm from the point of peak interface pressure.

Population and Sample

This section describes the population and sample characteristics for the study. The means by which patients were recruited to the study, together with inclusion and exclusion criteria are included.
Population

The population for the study was drawn from the two principal hospitals in the Australian Capital Territory (ACT), The Canberra Hospital (TCH) and Calvary Health Care ACT (CHC ACT). Whilst physically located within the ACT the hospitals serve both the ACT and the surrounding region of New South Wales (NSW). At the time of the study TCH was a 450 bed public tertiary referral hospital, whilst CHC ACT was a community hospital with an approximate capacity of 300 beds treating both public and private patients.

Medical and surgical clinical areas within both hospitals were utilised for this study. At TCH patients were recruited from the vascular/urology, renal, antenatal/gynaecology and cardiology departments. At CHC ACT patients were recruited to the study from coronary care unit, intensive care unit, high dependency unit, orthopaedic, convalescent / lower dependence, short stay, and the general medical and surgical clinical areas. This mix of clinical areas enabled recruitment of patients with a range of conditions including those who were acutely and chronically ill. However, this mix of diseases in the sample may have been a confounding variable.

Sample

The original sample size was determined by the requirements of the randomised controlled trial component of the MIPPI project that required a total of one hundred and forty completed patient datasets. The randomised controlled trial component was ceased prior to completion due to changes in the status of the
industry partner named in the AusIndustry Research and Developments grant. At that stage the completed sample size was 126. There were no applicable human data on which to base a sample size calculation for this study. A sample size calculation demonstrated that a study with a sample of 119 would have a 90% power at the 0.001 significance level to detect a Pearson’s correlation coefficient of 0.4. This meant that there was ample power in the study to detect larger correlation coefficients even with non-normally distributed data (Cohen, 1988).

**Patient Identification and Recruitment**

The MIPPI project was advertised within each hospital with posters describing the study (Appendix D) located in the corridors in clinical areas. The clinical nurses in each area made patients aware of the opportunity to participate in the study and on request provided patients with an information sheet describing the project. In the event that a patient expressed an interest to participate, the research nurses employed for the study were informed and the consent process as described in the ethical approval section of this chapter was followed.

Patients were recruited into the project at any time during their admission to the nominated clinical areas. In addition, any patients who transferred into clinical areas where the study was taking place were approached to determine their willingness to participate.
Inclusion and Exclusion Criteria

The main inclusion criteria for this study were patients who were (1) sixteen years and older, (2) identified as an inpatient and occupied a bed, and (3) who had either no pressure injuries or were identified as having a pressure injury on the sacral region which did not exceed Category 1 as defined by the AWMA 2001 Guidelines. A description of a Category 1 pressure injury was presented in Chapter Two, Table 1. To assist clinical nurses with decisions about potential patients who could be approached to participate in the study, a detailed set of criteria (Appendix E) were developed as a result of the pilot test conducted within the MIPPI project.

Patients less than 16 years of age were totally excluded from the study as the Waterlow Risk Assessment Tool (Appendix F) has not been designed for use in paediatrics. In addition, four other exclusion criteria were applied. The first of these were patients who did not consent to participate. The second factor for exclusion were those patients identified by nursing staff as requiring a high dependency of care with constant observation. The final two criteria concerned patients who could not tolerate lying supine with a 30% head elevation (two pillows) and those who could not lie completely still for the length of time required for data collection.

Interface Pressure Mapping Indices and Risk Factors

In order to investigate the study aims as previously described, the following independent and dependant variables were investigated from the recorded data:
• Interface shape – as determined from the ClinSeat™ mapping display (described later). Interface shape is an independent variable.
• Weight – as measured – independent variable.
• Body Mass Index (BMI). This is a function of weight and height, calculated as weight in kilograms divided by the square of height in metres, and was a dependent variable (BMI = weight/ (height)^2).
• Waterlow Risk Assessment Tool Score. This is determined from a variety of inputs as described in Chapter Two and was a dependent variable.
• Peak Interface Pressure – as measured. Peak interface pressure was a dependent variable.
• Gradient at 1.5 cm. The gradient was determined from the value of the peak interface pressure and the pressure at a distance 1.5cm from the site of the peak interface pressure and was a dependent variable.
• Gradient at 2.5 cm. As per the gradient at 1.5cm, but measured 2.5cm from the peak interface pressure point. PG_{2.5} was a dependent variable.

Weight and BMI and the Waterlow Risk Assessment Tool were selected for further study as these were quantitative factors that could have a relationship with peak interface pressure, pressure gradient and interface shape. The Waterlow Risk Assessment Tool was included in the study as this is the pressure injury risk assessment tool used in the ACT.
Study Equipment, Tools and Data

A combination of equipment, paper clinical tools, computer generated data and software analysis tools were utilised for this study. The following section will describe in detail the structure and application of these various data measurement and collection mechanisms.

Equipment

The major equipment employed during the study comprised the designated standard hospital mattress and hospital bed, and the Tekscan Clinseat™ pressure mapping system. A designated study mattress was used and ensured standardisation of the data collection processes and consistency of study equipment as it was impossible to accurately assess the age of mattresses already circulating within the hospital system. The potential for the project results to be influenced by mattress age and condition was therefore removed.

The mattress was the Comfort Plus™, the standard mattress used in the ACT hospitals at the time of the study. The mattress was developed by Australian Healthcare Industries and was made from high quality foam with convoluted foam ‘egg shell’ designed to reduce pressure and to lower shearing mechanisms. This mattress had been recommended by the company to be used for patients identified at low risk of pressure injury. The company did not provide any definition of low risk, nor any specific Waterlow score that equated with low risk of pressure injury development. The determination of low risk provided by Waterlow as being less than 10 was therefore used. Ethical considerations associated with the placement of
‘at risk’ patients on this mattress are covered in the later section addressing ethics approvals. Further technical specifications and measurements of the Comfort Plus™ foam mattress such as density, hardness, indentation factor and resilience are detailed in Appendix G.

The designated study mattress was transported on a dedicated hospital bed. Again, the utilisation of dedicated equipment ensured consistency of the surface on which the mattress was placed. In addition the use of a hospital bed ensured that the bed was easy to move within the hospital environment and to maintain compliance with TCH occupational health and safety requirements.

The other major piece of equipment utilised within the study was the Tekscan ClinSeat™ interface pressure mapping system (Tekscan User Manual, 2001). The ClinSeat™ system comprised the Microsoft Windows™ based ClinSeat™ software loaded onto to dedicated laptop computer, and other associated hardware as shown pictorially at Figure 3. The Tekscan ClinSeat™ system hardware is comprised of three components as described below; namely a sensor mat, parallel interface module and the Tekscan handle.
The ClinSeat™ sensor mat (53cm x 49cm) consisted of approximately 2,000 individual pressure sensing locations, referred to as ‘sensing elements’ or ‘sensels’. The sensels were arranged in rows and columns on the sensor mat and uniformly placed at 1cm intervals. Each sensel could be seen as an individual square on the computer screen when the 2-D Contours View as described below was selected. The digital output of each sensel was divided into 256 increments, and displayed as a value (raw sum) in the range of 0-255 by the software. For presentation purposes the ClinSeat™ software divided the mat into four quadrants.

Data from the sensor mat was collected via the Tekscan handle and provided to the ClinSeat™ software via the parallel interface module. Interface pressure information could be viewed as an image or as actual pressure values. A Dell laptop was used concurrently with the sensor mat to provide a visual display.
description of the software output is provided in the section addressing computer-generated data.

**Clinical Tools**

A paper form was used to record clinical information during the data collection process. This form comprised three components: demographic and clinical information, the Waterlow Risk Assessment Tool score, and later assessment of sacral pressure injury incidence as detailed in the ethical considerations.

A data collection tool (see Appendix H) was designed and utilised to record demographic data taken verbally from the patient and extracted from the clinical notes. This component was designed by the investigating team and validated in a pilot study as part of the MIPPI project. The design of the paper based form matched the computer screen of the electronic data base to minimise the potential for transcription errors. The clinical parameters used for this study were recorded on the tool and included weight from which BMI was calculated later.

The Waterlow Risk Assessment Tool is the standard risk assessment tool in use in ACT hospitals and has been utilised for this study. As discussed in Chapter Two, the purpose of a risk assessment tool is to identify individuals ‘at risk’ of developing a pressure injury through the systematic assessment of the patient for identified risk factors (Torra i Bou et al., 2006).
The Waterlow Risk Assessment Tool is a multi-variable tool that assesses the patient according to pre-defined demographic, health and behavioural factors to determine a risk score (Anthony et al., 2010; Kottner & Balzer, 2010). The variables used are weight and build, continence, skin type, mobility, gender and age, and appetite, and includes the consideration of special risks associated with tissue malnutrition, neurological deficits, surgery/trauma and special medications. These categories enable the scorer to complete a detailed clinical assessment of the patient. The Waterlow scale provides weighted scores in several categories, and also allows multiple scores in a number of categories. Several scores in each category can be awarded to accurately reflect the patient’s risk status with the total score reflecting the overall risk level. A patient with a score 10 or greater is considered at risk. A score greater than 15 assesses the patient at high risk and greater than 20 at very high risk. The minimum possible score is 1 and the maximum score is 64. For the purposes of this study the patients were identified at risk for scores of 10 and above or not at risk for scores less than 10.

**Computer Generated Data**

The Tekscan ClinSeal™ system generated data over a user-selected period at one minute intervals. The results of each measurement were a complete pressure map of the interface pressure across the sensor mat. The Clinseat™ system provided a variety of ways in which to view the results of measured interface pressure data. In this study the 2D (two dimensional) Contours View, the 3D (three dimensional) Wireframe View and the Peak Interface Pressure vs. Time Plot were
utilised (Tekscan User Manual, 2001). Examples of these displays are shown at Appendix I.

2D Contours View

The 2D Contours View showed interface pressure depicted according to a user-selected colour scale and interpolated the pressure between adjacent sencels. This interpolation provided a ‘smoothed’ rather than a ‘pixellated’ view of the pressure across the measurement area.

A cursor was available in the 2D Contours View that linked to both the numerical pressure readings and the visual pressure map pictures. By placing the cursor on the picture a numerical reading of the x-y coordinates of that location on the sensor mat was generated on the computer screen. The use of the cursor therefore allowed the numerical measurement of the pressure at any point across the sensor mat to be ascertained. A line function was also available on this display and provided the means to measure the distance between two selected points on the display. As the 2D Contours View displayed the pressure at all interfacing areas across the measurement surface, an assessment of the skin contact shapes could be made from this view.

3D Wireframe View

The 3D Wireframe View depicted pressure in both height and colour using the same colour scale as selected for the 2D Contours View. The 3D Wireframe
View allowed visualisation of both peak interface pressure and the pressure gradient. Whilst the 3D Wireframe View was useful in visualising the pressures across the measurement surface, and in preparation for the measurement of pressure gradients, no cursor was available and numerical pressure measurements could not therefore be retrieved from this view.

**Peak Interface Pressure vs. Time Plot**

The Tekscan ClinSeat™ Peak Interface Pressure vs. Time Plot provided a graphical depiction of the peak interface pressure recorded in each of the four quadrants on the sensor mat for each of the ten measurements taken over the measuring period. Each quadrant was shown in a different colour to aid readability of the graph.

The Peak Interface Pressure vs. Time Plot could be accessed from the 2D Contours View. When selected, the location of the peak interface pressure in each quadrant was shown on the 2D Contours View by the use of a coloured square corresponding to the quadrant colour on the Peak Interface Pressure vs. Time Plot. The Peak Interface Pressure vs. Time Plot was utilised during the study as a means to identify the level and the location of the peak interface pressure.

**Process of Data Collection and Management**

Data collection for the nested study reported here was undertaken within the MIPPI study, and the following sections refer to the organisation and collection
techniques for that study. The MIPPI data underwent additional data reduction and analysis within this study for the investigation of the aims as posed in Chapter One and the detailed questions as described later in Chapter Five. This section describes all aspects of the collection and management of the data, from inter-rater reliability through to initial data reduction in preparation for data analysis to answer the aims posed in this thesis.

**Training and Inter-rater Reliability**

The MIPPI data collectors were three Registered Nurses who had a minimum post registration clinical experience of five years. All research nurses were given extensive education in the management of the tools and equipment for the project. Research nurses worked in a team of three or more members, with each of the team members rotated within the designated roles in the data collection process each day; namely clinical history recording, operation of the pressure mapping system and overall assistance. This intensity of research staff ensured that the team could operate independently of the clinical ward activity and was needed to minimise interference to other patients and staff within busy clinical areas.

Inter-rater reliability was assessed for all research nurses to ensure consistent data collection practices. This assessment covered the use of the Waterlow Risk Assessment Tool, the calibration of the interface pressure mapping system and the set-up of the Tekscan ClinSeat™ software. The inter-rater reliability tests were conducted prior to the commencement of data collection.
To assess capability in the use of the Waterlow Risk Assessment Tool a written multiple choice test was given to all research nurses prior to commencement of the study requiring a pass rate of 100%. All data collectors achieved this requirement. During the data collection process random testing using the initial test was undertaken on all data collectors on a monthly basis by a member of the investigating team and a 100% pass rate was required and achieved.

A daily calibration of the Tekscan ClinSeat™ pressure mapping system was required in order that the raw digital output was converted to actual pressure units (mmHg) reliably over time. The calibration process generated a measurement that needed to be entered in the ClinSeat™ software demonstrating that the system was functioning correctly at the beginning of each day. The calibration procedure is described in detail in the ClinSeat™ User Manual (2001). A consistent approach to calibration was needed and therefore all research nurses were trained in this aspect.

Data Collection

At the beginning of each day of data collection the study bed and mattress were prepared by placing the sensor mat on the mattress and connecting the interface pressure mapping system hardware components to the sensor mat. Patients who had consented to participate in the study were approached by the research team and the patient was moved to the study bed and mattress. To ensure that the patient’s privacy was protected in shared accommodation areas bed curtains were drawn during the data collection process. Minimal disruption to other patients
and clinical activities was achieved by maintaining daily communication with the Clinical Nurse Consultants within each area.

Data were collected from both the clinical notes and verbally from the patient. Measurements of the patient’s height, weight, body temperature and blood pressure were collected first. The research nurse asked several other questions regarding diagnosis on admission, co-morbidities and medications that the patient was taking. Of these data, only height and weight were used for this study. The Waterlow risk assessment tool was used to provide a score to determine the patient’s level of risk for the development of a pressure injury. The patient was then asked to lie on their most comfortable side and a small marker was taped on the lowest vertebra of the spine where the anal cleft begins, identifying the sacral region. The patient then lay on their back on the interface pressure sensor mat. The marker was then removed once the visual picture was present on the computer screen and the research nurse identified the sacral region, by row and column on the computer software.

Patients were positioned supine with an elevation of 30% at the head. The patient was asked to lie as still as possible while measurements were taken by the Tekscan system and transcribed to a paper data collection tool. Patients were also requested not to cross their legs during the data collection period. Patients were able to wear their night gown or bed clothing and a sheet was placed over their body to keep the patient warm. At any time during the data collection process the patient could withdraw from the study.
Ten separate measurements were taken over a 10 minute period. As previously described, the visual display of the interface pressure readings were recorded in the form of a movie, with representation of a coloured visual display and also numerical data. At the end of the data collection process the study bed and mattress were replaced by the original bed and mattress. Patients were assisted back to bed, and the study bed was removed from the designated clinical area.

**Data Consolidation**

As described earlier, the data collection process incorporated information taken verbally from the patient, their clinical records and the software-generated visual and numerical data. Once data collection had been completed data were transferred to a purpose designed Access™ electronic data base in the RCNMP. All collected and recorded data were entered into the database at the end of each day by the research nurses.

For this nested study, data were transferred into an Excel data base specifically designed to hold the visual shapes, peak interface pressures, and interface pressure mapping gradients. Data in the Excel database were also cross checked with the original paper records and the MIPPI Access™ data base every ten data entries. Data were then transferred into SPSS version 15.0 where an additional process of checking that the data were complete was undertaken. Frequencies were generated and reviewed to identify errors and missing data.
**Initial Data Reduction**

The complex measurements generated by the Tekscan system provided the potential for many factors to be calculated. These raw data were reduced to meaningful study data, collated and prepared for statistical analysis. This preparation involved the determination of the value and site of the peak interface pressure, the calculation of gradients and the determination of shape from the visual images of the interface surface. These are discussed in more detail in the following paragraphs.

**Data Reduction for Peak Interface Pressure and Gradient**

**Correlations**

*Peak Interface Pressure*

The peak interface pressure was determined from the Tekscan ClinSeat™ software, and specifically from the Peak Interface Pressure vs. Time Plot. As noted previously this display provided a line graph view of the peak interface pressures recorded in each quadrant of the sensor mat for each of the ten measurements taken over the recording period.

The peak interface pressure point was identified by reference to the Peak Interface Pressure vs. Time Plot to determine the peak interface pressure itself and the relevant quadrant. By reference to the 2D Contours View the point at which the peak interface pressure occurred was then located. The Tekscan cursor function was utilised to mark the position of the peak interface pressure using the centre of
the selected sensel. This position was recorded directly from the cursor readout as 
\((x_p, y_p)\) for each of the ten measurements. The mean of the peak interface pressures 
in each quadrant over the measurement periods was determined with the highest of 
the four means recorded as the peak interface pressure on the mat.

**Pressure Gradient**

In this component of the study the gradients at two points from the point of 
peak interface pressure were compared; namely at 1.5 cm and at 2.5 cm. 
Measurements to determine gradients were taken for each of the ten recordings for 
each patient and averaged to determine a single pressure gradient for each patient at 
1.5cm and 2.5cm. The 2D Contours View digital display screen was used as 
described later to determine gradients. The 3D Wireframe View was utilised to 
visualise the overall pattern of the data for each measurement. As noted earlier the 
lack of a cursor in this view precluded its direct use in the determination of the 
gradients.

Measurements were taken at 1.5cm and 2.5cm from the \((x_p, y_p)\) point to 
ensure that the gradient recorded was considered across at least two sensels and 
three sensels respectively, each sensel being 1cm across. The Tekscan 2D Contours 
View was used to locate the point of the lowest pressure at the selected range. The 
Tekscan line function was used to determine the required 1.5cm and 2.5cm 
distances, and the pressure recorded as \(P_1\) and \(P_2\) at these respective points. The 
positions for the pressure measurements were recorded as \((x_1, y_1)\) and \((x_2, y_2)\) in the 
Excel database.
Readings at 1.5 cm and at 2.5 cm were not necessarily in same direction as the aim was to determine the greatest gradient around the peak interface pressure point \((x_p, y_p)\). In the event that doubt existed from the 2D Contours View regarding the site of lowest pressure at either the 1.5cm or 2.5cm distance reference was made to the 3-D Wireframe View and a series of measurements were taken to ensure that the lowest pressure reading was obtained.

An observation was made that the distance readout from the Tekscan ClinSeat™ line function, and the computed distance between the recorded points did not always provide an exact match. For this reason, the computed distance was utilised to determine gradient as this provided a more accurate and reliable computation of the resulting gradient.

The determination of gradients at 1.5cm and 2.5cm for the study was made using the following methodology:

1. The peak interface pressure and the \((x_p, y_p)\) position of the peak interface pressure point were recorded.
2. The difference in pressure between the point of peak interface pressure (PIP) and that at 1.5cm and 2.5cm \((\Delta P_n)\) was simply determined from:

\[
\Delta P_1 = P_{IP} - P_1. \quad (\text{Equation 1})
\]

\[
\Delta P_2 = P_{IP} - P_2. \quad (\text{Equation 2})
\]
3. The distance \((D_n)\) between the point of peak interface pressure and the 1.5cm and 2.5cm measurements was obtained from the following equations:

\[
D_1 = \sqrt{(x_1 - x_p)^2 + (y_1 - y_p)^2} \quad \text{(Equation 3)}
\]

\[
D_2 = \sqrt{(x_2 - x_p)^2 + (y_2 - y_p)^2} \quad \text{(Equation 4)}
\]

4. The pressure gradient \((G_n)\) in mmHg/cm was then obtained by

\[
G_1 = \frac{\Delta P_1}{D_1} \quad \text{(Equation 5)}
\]

\[
G_2 = \frac{\Delta P_2}{D_2} \quad \text{(Equation 6)}
\]

5. The gradient for each of the ten pressure measurements was individually determined at 1.5 cm and 2.5 cm from the peak interface pressure site, and averaged to arrive at the recorded pressure gradients for each patient.

**Data Reduction for Shape Correlation**

The determination of shapes was made post-factum from the recorded data using the 2D Contours View display screen. As discussed earlier, in this view the software presented ten pictures of the total skin contact area for each patient on the measurement surface. Each image was visually reviewed and coded as oval, round, rectangle, pear or square. The decision for allocating each sample to the particular shape was that (1) the pattern of the interfacing skin fitted this general description.
and (2) there was consistency in shape over the period of ten readings. In the event that the upper legs and/or lower back were also represented on the screen, the shape was determined without taking this information into account. The coded shapes were added to the Excel database.

**Data Analysis**

This section provides an overview of the data analysis undertaken for this thesis. A brief explanation of tests for normality and the outcomes of these tests is included. A summary of the investigations conducted into relationships with shape, and for correlation between other variables is included. The detailed results of these tests are contained in Chapter Four.

**Test for Normality**

The complete set of variables as previously described was individually explored for normality using the Kolmogorov-Smirnov Test (K-S Test). The significance value was >0.5 if normality was indicated. These initial tests indicated that none of the variables were normally distributed. The data were then transformed using a logarithmic transformation (base 10) and retested using the K-S Test. The logarithmically transformed weight, peak interface pressure, gradient 1.5cm and 2.5cm were found to be normally distributed. The transformed BMI and Waterlow Risk Assessment Tool were not normally distributed.
Tests for Correlation

A series of correlation tests were conducted on all variables to determine the strength and direction of the linear relationship between pairs of variables. The Pearson product moment correlation coefficient was used for variables that were normally distributed. As BMI and Waterlow risk scores were not normally distributed, Spearman’s rank-order correlation was employed to assess correlations involving these variables. The strength of the correlations observed were assessed from the coefficient of correlation \( r \) as being (1) small for \( r \) between 0.1 and 0.29, (2) medium for \( r \) between 0.3 and 0.49 and (3) large for \( r \) between 0.5 and 1.0 (Cohen, 1988).

Application of Mann-Whitney U test

The Mann-Whitney U test is a non-parametric counterpart to the t-test and can be applied to explore the differences between two independent groups where the continuous measures are not normally distributed. Interface pressure shapes were divided into groups with similar characteristics as described in the following chapter; namely round/square and other shapes. The differences between the two groups were explored with respect to all variables. To assess the size of the observed effect, the Mann-Whitney z value was transformed to a ‘\( r \)’ value through the relationship \( r = z / \sqrt{N} \) (where \( N \) is the sample size), and the resultant \( r \) value considered in the same small, medium, large format as for the correlations above (Cohen, 1988).
Ethics

Ethics approvals were gained from Deakin University Human Research Ethics Committee, from the ACT Human Research Ethics Committee, and from the Calvary Healthcare Human Research Ethics Committee. Copies of the approval letters are shown at Appendices J to L. The main ethical issues for the study were the potential for harm, consent, and privacy and confidentiality as described below.

Potential for Harm

Three areas of potential harm to patients recruited to this study were identified. These were the utilisation of sophisticated equipment not normally used in clinical practice; the movement of patients to and from the study equipment; and the potential risk of pressure injury for those patients identified at high risk. These risks are discussed separately below.

Equipment Risk

All mechanical equipment used in the study was inspected by ACT Health’s Biomedical Engineering Department and approved for use within the two hospitals. In addition, to ensure compliance with infection control and prevention practices, all equipment was wiped down with antiseptic spray between data collection processes. The mat was covered by a soft cleanable cover, and this mat cover was changed between each patient data collection set. The infection control processes were approved by the Infection Control Department.
**Movement Risk**

The majority of patients involved in the study were able to move independently from their hospital bed to the study hospital bed. Patients who could not move independently were transferred by research nurses using designated lifting equipment meeting ACT Health’s Occupational Health and Safety requirements. All data collectors underwent training from ACT Health Injury Prevention and Management Unit in the use of lifting equipment and other assistive patient handing devices as endorsed by ACT Health to ensure that the potential for harm in moving from one bed to the other was minimised.

**Pressure Injury Risk**

At the time of the study, it was standard clinical practice to place all patients on a regular hospital bed and mattress as part of the admission process and to then conduct a risk assessment using the Waterlow Risk Assessment Tool. If the patient was determined to be ‘at risk’ then an advanced pressure relieving mattress would be put in place.

As noted earlier in this chapter, for the purpose of the study, participants were required to be placed on a ‘Comfort Plus™’ mattress, the same mattress as used throughout the hospitals for patients identified as ‘not at risk’ or ‘at low risk’ by the Waterlow Risk Assessment Tool scoring system. Patients who were identified as being ‘at risk’ of pressure injury were therefore placed on a mattress not specifically designed to be pressure relieving for a period of ten minutes during
the study. Patients deemed by clinical staff to be at risk for pressure injury development over this short period were not recruited to the study.

The patient sacral area was visually reviewed for pressure injury seven to fourteen days post study. Alternatively, if the patient had been discharged, the clinical record was reviewed for any documentation of pressure injury. The staging systems used to classify pressure injuries have been described in the literature review. The AWMA (2001) classifications were used for this study. No follow up pressure injuries were identified.

**Consent**

All patients who participated in this study gave informed written consent. A patient information sheet was provided as part of this consent process in order to ensure that all participants were adequately informed about the research (Appendix M). Written consent was then sought following a full explanation of the data collection process and after the patient had been given an opportunity to ask questions. The patient consent forms for TCH and CHC ACT (see Appendix N and Appendix O) stated that the participants could withdraw from the study at any time without altering their medical management. Contact details of the investigator, supervisor and ethics committees were made available.

Experienced Registered Nurses were specifically employed as research nurses for the study. Due to the acuity of the clinical environment the consent process was undertaken the day prior to data collection to ensure that sufficient
time was available to explain the study to patients and to ensure the data collection procedure fitted with the patient’s therapeutic plan. The research nurses’ clinical experience and judgement enabled them to determine whether a patient was appropriate to be recruited into the study.

**Privacy and Confidentiality**

All data collected were de-identified upon computer data entry but remained re-identifiable until data analysis had been completed. Paper files carried study identifying numbers and were stored in a locked filing cabinet at the RCNMP. The key linking patients’ identifying data to the study identifying numbers was kept separate from the paper files in a lockable filing cabinet at the RCNMP. Computer access was password protected and was only accessible by the research staff and study investigators. Upon completion of the study, computer data were transferred to CD-ROM and stored in a locked filing cabinet for seven years in accordance with the National Health and Medical Research Council (NHMRC) guidelines.

**Summary**

This chapter has outlined the methodology by which the aims of this study to (1) explore correlations between two interface pressure mapping indices and selected risk factors for pressure injury, and (2) explore the visual anatomical characteristics of the buttock region of patients through the use of interface pressure mapping and the correlation between shape and selected risk factors for pressure injury have been addressed. The study design, population and sample, and the data

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collection, management, reduction and analysis processes have been discussed. The next chapter will present the findings of the study.
CHAPTER FOUR - RESULTS

Introduction

The previous chapter provided the methodology utilised to investigate the relationships that exist between the selected pressure injury risk factors, the pressure mapping indices and the anatomical patterns of the sacral region in hospitalised patients using a visual interface pressure mapping system. The conceptual framework within which these risk factors and indices are considered was introduced in the literature review. In this chapter the results are presented.

The pressure interface mapping system provided visual images displaying the skin surface area in contact with the sensor mat and these images were categorised into groups describing the resultant visual shapes. Three pressure injury risk factors and three interface pressure mapping indices were used in the analysis. The risk factors were weight, BMI and risk level as determined by the Waterlow Risk Assessment Tool. The pressure mapping indices were peak interface pressure and the pressure gradients at distances of 1.5 cm and 2.5 cm from the peak interface pressure point. Pearson’s product moment correlation coefficient (Pearson’s r) and Spearman’s rank order correlation (Spearman rho) were applied to the data to investigate the nature of the relationship between the variables. The Mann-Whitney U test was applied to the data to explore the differences between the visual shapes and six variables.
This chapter begins with a description of the demographic characteristics of the study and exploration of the sample against the Waterlow Risk Assessment Tool assessment criteria. Each research aim is then addressed in turn.

The study aims are:

1. To explore correlations between two interface pressure mapping indices, and selected risk factors for pressure injury.
2. To explore the visual anatomical shape of patients through the use of interface pressure mapping and the correlation between shape and selected risk factors for pressure injury.

Study Sample

A total of 126 patients consented to participate in the MIPPI study, from which five were subsequently excluded. Two exclusions were patients who could not tolerate the required period of immobility, and three were excluded with extremely high interface pressure readings (Gardner et al., 2006). For this nested study one patient was excluded as the visual shape was identified to be outside the parameters of the interface mat leaving a study sample comprising 120 patients within a hospital setting. The sample was representative of the population of both hospitals in medical and surgical areas excluding the most severely ill. Patients were enrolled into the study as previously described in the methods chapter. The final sample comprised 56.7% males (n = 68) and 43.3% females (n= 52) ranging in age from 18 to 88.
**Sample Characteristics**

Patients’ level of risk for pressure injury was assessed using the Waterlow Risk Assessment Tool. This instrument with its component sections and the calculation of the risk index level has been detailed in the literature review chapter. The utilisation of the Waterlow Risk Assessment Tool therefore resulted in patients being classified as either ‘not at risk’, with a Waterlow Risk Assessment Tool score of less than 10, or ‘at risk’ if the score was determined to be 10 or greater. More than half the sample was categorised at risk of pressure injury development using the Waterlow Risk Assessment Tool with 46% assessed as not at risk (n=55) and 54% at risk (n=65). The at risk group were older and comprised a higher percentage of males (see Table 2 below).
Table 2. Demographic Characteristics Compared According to Pressure Injury Risk Category

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No. Patients % of sample</th>
<th>Mean +/- (SD) or Median (min, max) (^a)</th>
<th>Mode</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk Category</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not At risk</td>
<td>55 (46%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>At Risk</td>
<td>65 (54%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not At Risk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>27 (49%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>28 (51%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>50.6 +/- (18.97)</td>
<td></td>
<td>8</td>
</tr>
<tr>
<td>Risk Score *</td>
<td>6 (2, 9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>At Risk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>25 (38%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>40 (62%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>68 +/- (12.7)</td>
<td></td>
<td>17</td>
</tr>
<tr>
<td>Risk Score *</td>
<td>14 (10, 28)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(^a\) Medians provided for Waterlow Risk Assessment Tool score because not normally distributed.
Waterlow Risk Assessment Tool Scores

This section of the chapter is designed to give the reader a more complete understanding of the characteristics of the patient sample as scored using the Waterlow Risk Assessment Tool. Table 3 presented below details the patient characteristics within each Waterlow Risk Assessment Tool subcategory. The items in the subcategory that comprised ‘skin type and visual areas’ within the Waterlow Risk Assessment Tool were not mutually exclusive. A number of participants were identified as being at risk within more than one skin type sub-category. Eighteen participants were identified being at risk in two sub-categories. Eight participants were identified being at risk in three sub-categories. Three participants were identified being at risk in more than four sub-categories. As a result the most frequent skin type was determined as dry (47.5%, n=57). However 37.5% of patients (n=45) had healthy skin.

In a similar manner a number of patients were scored in more than one sub-category in the tissue malnutrition category. The highest risk identified here was smoking (15.8%, n=19). Four participants were identified being at risk in more than one tissue malnutrition sub-category. Three participants were identified as being at risk in more than two sub-categories. One participant was identified at risk in four sub-categories. Only 37.5% (n=45) of the sample were at an average weight. Five patients (4.2%) were recorded as being below average weight.
<table>
<thead>
<tr>
<th>Waterlow Risk Tool Categories</th>
<th>Waterlow Risk Tool Subcategories</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>Female</td>
<td>52</td>
<td>(43.0)</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>68</td>
<td>(57.0)</td>
</tr>
<tr>
<td>Age</td>
<td>14-49</td>
<td>33</td>
<td>(27.5)</td>
</tr>
<tr>
<td></td>
<td>50-64</td>
<td>32</td>
<td>(26.6)</td>
</tr>
<tr>
<td></td>
<td>65-74</td>
<td>26</td>
<td>(21.6)</td>
</tr>
<tr>
<td></td>
<td>75-80</td>
<td>18</td>
<td>(15.0)</td>
</tr>
<tr>
<td></td>
<td>80+</td>
<td>11</td>
<td>(9.2)</td>
</tr>
<tr>
<td>Skin type &amp; visual areas</td>
<td>Healthy</td>
<td>45</td>
<td>(37.5)</td>
</tr>
<tr>
<td></td>
<td>Tissue paper</td>
<td>10</td>
<td>(8.3)</td>
</tr>
<tr>
<td></td>
<td>Dry</td>
<td>57</td>
<td>(47.5)</td>
</tr>
<tr>
<td></td>
<td>Oedematous</td>
<td>21</td>
<td>(17.5)</td>
</tr>
<tr>
<td></td>
<td>Clamy</td>
<td>0</td>
<td>(0.0)</td>
</tr>
<tr>
<td></td>
<td>Discoloured</td>
<td>19</td>
<td>(15.8)</td>
</tr>
<tr>
<td></td>
<td>Broken</td>
<td>11</td>
<td>(9.2)</td>
</tr>
<tr>
<td>Continence</td>
<td>Complete/ catheterised</td>
<td>113</td>
<td>(94.2)</td>
</tr>
<tr>
<td></td>
<td>Occasionally incontinent</td>
<td>0</td>
<td>(0.0)</td>
</tr>
<tr>
<td></td>
<td>Catheter/ incontinent of faeces</td>
<td>7</td>
<td>(5.8)</td>
</tr>
<tr>
<td></td>
<td>Doubly incontinent</td>
<td>0</td>
<td>(0.0)</td>
</tr>
<tr>
<td>Tissue</td>
<td>Terminal cachexia</td>
<td>1</td>
<td>(0.8)</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>Cardiac failure</td>
<td>17</td>
<td>(14.2)</td>
</tr>
<tr>
<td></td>
<td>Peripheral vascular disease</td>
<td>8</td>
<td>(6.7)</td>
</tr>
<tr>
<td></td>
<td>Anaemia</td>
<td>10</td>
<td>(8.3)</td>
</tr>
<tr>
<td></td>
<td>Smoking</td>
<td>19</td>
<td>(15.8)</td>
</tr>
<tr>
<td>Waterlow Risk Tool Categories</td>
<td>Waterlow Risk Tool Sub-categories</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>----------------------------------</td>
<td>----</td>
<td>------</td>
</tr>
<tr>
<td>Mobility</td>
<td>Fully</td>
<td>75</td>
<td>(62.5)</td>
</tr>
<tr>
<td></td>
<td>Restless/fidgety</td>
<td>2</td>
<td>(1.7)</td>
</tr>
<tr>
<td></td>
<td>Apathetic</td>
<td>8</td>
<td>(6.7)</td>
</tr>
<tr>
<td></td>
<td>Restricted</td>
<td>35</td>
<td>(29.2)</td>
</tr>
<tr>
<td></td>
<td>Inert/traction</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Chair-bound</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Neurological deficit</td>
<td>Moderate</td>
<td>11</td>
<td>(9.2)</td>
</tr>
<tr>
<td></td>
<td>Moderate to Severe</td>
<td>2</td>
<td>(1.7)</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Appetite</td>
<td>Average</td>
<td>77</td>
<td>(64.2)</td>
</tr>
<tr>
<td></td>
<td>Poor</td>
<td>33</td>
<td>(27.5)</td>
</tr>
<tr>
<td></td>
<td>Nasogastric tube/fluids only</td>
<td>9</td>
<td>(7.5)</td>
</tr>
<tr>
<td></td>
<td>Nil By Mouth /Anorexia</td>
<td>1</td>
<td>(0.8)</td>
</tr>
<tr>
<td>Build/Weight for Height</td>
<td>Average</td>
<td>45</td>
<td>(37.5)</td>
</tr>
<tr>
<td></td>
<td>Above average</td>
<td>48</td>
<td>(40)</td>
</tr>
<tr>
<td></td>
<td>Obese</td>
<td>22</td>
<td>(18.3)</td>
</tr>
<tr>
<td></td>
<td>Below average</td>
<td>5</td>
<td>(4.2)</td>
</tr>
<tr>
<td>Major Surgery/Trauma</td>
<td>Orthopaedic – below waist spinal</td>
<td>3</td>
<td>(2.5)</td>
</tr>
<tr>
<td></td>
<td>On table &gt; 2 hours</td>
<td>1</td>
<td>(0.8)</td>
</tr>
<tr>
<td>Medication</td>
<td>Cytotoxics</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>High dose steroids</td>
<td>27</td>
<td>(22.5)</td>
</tr>
<tr>
<td></td>
<td>Anti-inflammatory</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

*Note.* For more detail on the breakdown of Waterlow Risk scores for the study sample see Appendix P.
Exploration of Relationships between Pressure Injury Risk Factor Characteristics and Interface Pressure Measurements

A series of questions were explored with regard to the pressure injury risk factors and two interface pressure mapping indices; namely weight, BMI, the Waterlow Risk Assessment score, the peak interface pressure and the pressure gradient. The last variable was measured at two separate distances from the peak interface pressure point to explore the nature of the gradient. The mean peak interface pressures and gradients recorded at shown at Table 4 below. As can been seen from the table the mean pressure reduces as the distance from the peak interface pressure reduces.

<table>
<thead>
<tr>
<th>Position</th>
<th>Mean Pressure (mmHg)</th>
<th>Mean Gradient (mmHg/cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak interface pressure point</td>
<td>54.9</td>
<td></td>
</tr>
<tr>
<td>1.5cm</td>
<td>29.8</td>
<td>11.4</td>
</tr>
<tr>
<td>2.5cm</td>
<td>24.0</td>
<td>9.3</td>
</tr>
</tbody>
</table>

Initial tests for normality using the K-S test indicated that none of the variable distributions were normal (see Appendix Q). The logarithmically transformed data for weight, peak interface pressure, gradient 1.5cm and gradient 2.5cm variables were determined to be normally distributed. The logarithmically transformed BMI and pressure injury risk score (as determined by the Waterlow...
Risk Assessment Tool) data were not normally distributed. The degree of correlation between the variables was determined by the application of the Pearson’s product moment correlation coefficient (Pearson’s r) for normalised data, and the Spearman’s rank order correlation (Spearman rho) for non-normal data as described in the previous chapter. The Spearman’s rho test was therefore applied to all tests involving BMI and Waterlow Risk Assessment score as these data were not normally distributed even after logarithmic transformation. The Pearson’s r test has been applied to remainder of the data where the logarithmically transformed data was normally distributed.

As stated in Chapter Three the study questions were grouped to accord with the Defloor (1999) conceptual framework and were as follows:

**Pressure related questions**

- What is the relationship between Peak Interface Pressure and weight?
- What is the relationship between Peak Interface Pressure and BMI?
- What is the relationship between Peak Interface Pressure and Risk Score as determined by the Waterlow Risk Assessment Tool?
- What is the relationship between Peak Interface Pressure and Gradient 1.5cm?
- What is the relationship between Peak Interface Pressure and Gradient 2.5cm?

**Shear related questions**

- What is the relationship between Gradient 1.5cm and weight?
- What is the relationship between Gradient 1.5cm and BMI?
What is the relationship between Gradient 1.5cm and Risk Score as determined by the Waterlow Risk Assessment Tool?

What is the relationship between Gradient 2.5cm and weight? What is the relationship between Gradient 2.5cm and BMI?

What is the relationship between Gradient 2.5cm and Risk Score as determined by the Waterlow Risk Assessment Tool?

What is the relationship between Gradient 2.5cm and Gradient 1.5cm?

**Tissue tolerance related questions**

What is the relationship between BMI and Risk Score as determined by the Waterlow Risk Assessment Tool?

What is the relationship between weight and BMI?

What is the relationship between weight and Risk Score as determined by the Waterlow Risk Assessment Tool?

The results of the analysis, showing the coefficients of correlation for each of the relationships above, are provided in Table 5 below. Four large correlations, using the Cohen (1988) description for size of correlation as detailed in Chapter 3, were identified. Weight correlated strongly with BMI as expected given the direct relationship between these risk factors (correlation coefficient 0.82). Peak interface pressure correlated with gradient 1.5cm and gradient 2.5cm with correlation coefficients of 0.77 and 0.78 respectively. Given that peak interface pressure is used in the calculation of gradient some degree of correlation was anticipated. In addition gradient 2.5cm and gradient 1.5cm had a correlation coefficient of 0.90. Given that these gradients were both measured from the point of peak interface pressure again some degree of correlation was expected. All relationships had a
positive correlation thereby showing that as one value increases the second parameter also increases at some rate.

Table 5. Correlation Between Weight, BMI, Waterlow Risk Assessment Score, Peak Interface Pressure, Gradient 1.5cm and 2.5cm

<table>
<thead>
<tr>
<th>Variables</th>
<th>Weight</th>
<th>BMI</th>
<th>Waterlow Score</th>
<th>Peak Interface Pressure 1.5cm</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>0.82b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>p &lt; 0.01</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waterlow Score</td>
<td>0.15b</td>
<td>0.23b</td>
<td>0.15b</td>
<td>0.23b (p =0.01)</td>
</tr>
<tr>
<td></td>
<td>(p = 0.09)</td>
<td>(p =0.01)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak interface pressure</td>
<td>0.23a</td>
<td>0.16b</td>
<td>0.19b</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>(p=0.01)</td>
<td>(p=0.07)</td>
<td>(p=0.04)</td>
<td></td>
</tr>
<tr>
<td>Gradient 1.5cm</td>
<td>0.22a</td>
<td>0.17b</td>
<td>0.09b</td>
<td>0.77b (p&lt;0.01)</td>
</tr>
<tr>
<td></td>
<td>(p=0.01)</td>
<td>(p=0.06)</td>
<td>(p=0.31)</td>
<td>(p&lt;0.01)</td>
</tr>
<tr>
<td>Gradient 2.5cm</td>
<td>0.18a</td>
<td>0.09b</td>
<td>0.10b</td>
<td>0.78b</td>
</tr>
<tr>
<td></td>
<td>(p=0.06)</td>
<td>(p=0.34)</td>
<td>(p=0.26)</td>
<td>(p&lt;0.01)</td>
</tr>
</tbody>
</table>

aPearson's r test applied

bSpearman’s rho test applied

The pressure injury risk factors, weight, BMI and Waterlow risk assessment score, showed only small correlation with peak interface pressure, or with the two pressure gradients. This lack of correlation between potential risk factors and measurements of peak interface pressure is explored in the next chapter.
Visual Anatomical Patterns and Relationships with Study Variables

The ClinSeat™ interface pressure mapping system’s software displayed a visual image which represented the skin contact of the buttock area, inclusive of the sacral and ischial regions, onto the surface of the pressure mapping mat. From an investigation of the patterns, five distinctive shapes could be categorised. These shapes were described as round, oval, square, pear and rectangle. The frequency of the round shape was the highest of the five identified shapes; round 54.2% (n=65), oval 19.2% (n=23), square 15.8% (n=19), pear 5.8% (n=7) and rectangle 5% (n=6).

The shapes were divided into groups, namely round/square (n=84) and other shapes (n=36). The division into these two groups was based on the overall broad commonality between the various shapes for the characteristic BMI, Waterlow Risk Assessment Tool score, and peak interface pressure as described in detail at Table 6. The five original shapes identified and the subsequent classification into two groups was validated by my supervisor, not otherwise externally validated. These groupings were considered as being important to provide an insight into whether any effect due to shape could be discerned.
### Table 6. Detailed Breakdowns of Shape Specific Data for BMI, Waterlow Risk Assessment Score and Peak Interface Pressure

<table>
<thead>
<tr>
<th>Shapes</th>
<th>N</th>
<th>At Risk/</th>
<th>Male/</th>
<th>BMI</th>
<th>Waterlow Risk Score</th>
<th>Peak Interface Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Not At</td>
<td>Female</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Round</td>
<td>65</td>
<td>41/24</td>
<td>34/31</td>
<td>Mean</td>
<td>27.69</td>
<td>13.03</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Median</td>
<td>28</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mode</td>
<td>28</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SD</td>
<td>5.21</td>
<td>6.51</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Range</td>
<td>18 - 44</td>
<td>3 - 34</td>
</tr>
<tr>
<td>Oval</td>
<td>23</td>
<td>13/10</td>
<td>12/11</td>
<td>Mean</td>
<td>24.82</td>
<td>8.43</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Median</td>
<td>24</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mode</td>
<td>24</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SD</td>
<td>4.9</td>
<td>4.13</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Range</td>
<td>19 - 40</td>
<td>3 - 17</td>
</tr>
<tr>
<td>Square</td>
<td>19</td>
<td>13/6</td>
<td>14/5</td>
<td>Mean</td>
<td>29.68</td>
<td>13.36</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Median</td>
<td>28</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mode</td>
<td>28</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SD</td>
<td>6.95</td>
<td>6.88</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Range</td>
<td>16 - 41</td>
<td>5 - 26</td>
</tr>
<tr>
<td>Rectangular</td>
<td>6</td>
<td>3/3</td>
<td>4/2</td>
<td>Mean</td>
<td>24.83</td>
<td>12.17</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Median</td>
<td>25</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mode</td>
<td>25</td>
<td>17</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SD</td>
<td>3.19</td>
<td>5.34</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Range</td>
<td>20 - 30</td>
<td>4 - 17</td>
</tr>
<tr>
<td>Pear</td>
<td>7</td>
<td>3/4</td>
<td>4/3</td>
<td>Mean</td>
<td>21.42</td>
<td>10.57</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Median</td>
<td>20</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mode</td>
<td>18</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SD</td>
<td>4.12</td>
<td>4.03</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Range</td>
<td>18 - 30</td>
<td>5 - 18</td>
</tr>
</tbody>
</table>
The Mann-Whitney U test is a non-parametric counterpart to the t-test and can be applied to explore the differences between two independent groups where the continuous measures are not normally distributed. This statistical test was used to explore the differences between pressure injury risk factors and interface pressure measurements (namely the peak interface pressure and pressure gradients) for the two grouped body shapes, round/square and other. The results of the Mann-Whitney U test are shown at Table 7. The size of the effect due to shape was determined from the r value using the Cohen (1988) descriptions of small/medium/large as presented in Chapter 3.

Table 7. Pressure Injury Risk Factor Characteristics and Peak Interface Pressure Measurements: Differences Between Body Shapes

<table>
<thead>
<tr>
<th>Variables</th>
<th>Z value</th>
<th>Median</th>
<th>r</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Round &amp;</td>
<td>Other shapes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Square shape</td>
<td>n = 36</td>
<td>n = 84</td>
<td></td>
</tr>
<tr>
<td>Weight</td>
<td>-3.11</td>
<td>80</td>
<td>69.5</td>
<td>-0.28</td>
</tr>
<tr>
<td>BMI</td>
<td>-3.95</td>
<td>28</td>
<td>24</td>
<td>-0.36</td>
</tr>
<tr>
<td>Risk Score</td>
<td>-2.74</td>
<td>12</td>
<td>9.5</td>
<td>-0.25</td>
</tr>
<tr>
<td>Peak interface</td>
<td>-1.31</td>
<td>53</td>
<td>51</td>
<td>-0.12</td>
</tr>
<tr>
<td>pressure</td>
<td>Gradient 1.5</td>
<td>-1.25</td>
<td>10.3</td>
<td>9.4</td>
</tr>
<tr>
<td></td>
<td>Gradient 2.5</td>
<td>-0.34</td>
<td>8.65</td>
<td>8.75</td>
</tr>
</tbody>
</table>
The results of the Mann-Whitney U tests showed there is a statistically significant difference between the round/square and other shapes for BMI ($z = -3.95$, $p<0.01$), weight ($z = -3.11$, $p < 0.01$) and Waterlow Risk Assessment Tool score ($z = -2.74$, $p < 0.01$) although effect size is small with the exception of BMI and shape. There is no difference between the shapes with respect to peak interface pressure and gradients of 1.5 and 2.5cm. In summary having a round or square shape as identified by image produced by the interface pressure mapping system is associated with a higher Waterlow risk score, higher weight and higher BMI.

**Summary**

The results have been presented for the two research aims informing this study. After describing the characteristics of the study sample, the first research aim investigated correlations between the three pressure injury risk factors and the three interface pressure mapping indices through a series of fourteen questions. Both Pearson’s $r$ and Spearman’s rho tests were utilised as whilst some of the logarithmically transformed data were normal other data sets were determined as not normal. No correlation was evident with the exception of peak interface pressure with pressure gradient at 1.5cm and 2.5cm, and gradient 1.5cm and 2.5cm which exhibited strong correlations. The correlations associated with peak interface pressure and gradient were anticipated as these risk indices were all based on the position and value of the measured peak interface pressure.
The second research aim was addressed through consideration of the five shapes formed by skin surface area on the interface pressure mapping surface. Due to the overall similarity of the characteristics of the round and square shapes two groups were identified for statistical analysis (round/square and other shapes). The Mann-Whitney test was utilised to investigate the six study variables within these groups. A statistically significant difference was observed between the two groups for weight, BMI and risk score as determined by the Waterlow Risk Assessment Tool. The implications of these results will be discussed in the following chapter.
CHAPTER 5 - DISCUSSION

Introduction

The previous chapter presented the results obtained from the analysis of the data acquired during this study. In this chapter the findings are discussed in relation to the study aims. In order to present both a succinct and logical discussion the chapter is structured around the research questions that guided this study.

The study’s two aims explored the correlation between two interface pressure mapping indices and selected risk factors for pressure injury, and explored the visual anatomical shapes of patients using the same indices and risk factors. An interface pressure mapping system, a technology not frequently used by clinical nurses, was used in the investigation of the potential correlations. The pressure mapping technology was important as it provided not only an ability to record numerical data but to simultaneously provide a visual observation of the pressure distribution across the interface surface. The study results indicate that both assessment tools and technology have the potential to contribute in the preventative management of pressure injury risk.

One of the benefits of this study is that it has been conducted on vulnerable at-risk patients in a hospital setting. The study addresses the gap that Rithalia (2005) identified for the measurement of peak interface pressures on unhealthy rather than on healthy subjects within controlled conditions, while noting that the former are less able to tolerate the measurement procedures. Rithalia (2005) also
suggests that the peak interface pressure measurements for at-risk patients are likely to differ from healthy volunteers due to differences in soft tissue and intrinsic properties. This study therefore contributes to the overall body of knowledge regarding pressure injury development and management through the involvement of a more relevant population sample.

In the review of the literature in Chapter Two, the Defloor (1999) conceptual framework for pressure injury development was introduced. This conceptual framework has three major sections, namely compressive forces, shearing forces and tissue tolerance to both pressure and oxygen. In the previous chapter the questions with respect to correlation were grouped according to this framework. A revised conceptual framework, based on amendments to the Defloor (1999) framework as presented in Chapter Two, will be advanced as a result of the observed results and as a basis for the further consideration of pressure injury risk in a clinical setting.

Investigation of Correlations between Risk Factors for Pressure Injury and Pressure Mapping Indices

This section details the investigation of correlations between the selected risk factors for pressure injury and the pressure mapping indices. The relevance of the correlations to the Defloor (1999) conceptual framework, and hence to the development of a pressure injury, will also be discussed.
The first study question investigated the correlation between the study risk factors for pressure injury, namely weight, BMI and Waterlow Risk Assessment score, and the pressure mapping indices, namely peak interface pressure, and the gradients at 1.5cm and 2.5cm from the site of the peak interface pressure.

Correlations were observed between peak interface pressure and the gradients at 1.5cm and 2.5cm, and between the two gradients. No correlations were observed between the Waterlow Risk Assessment Tool scores and any other risk factors or the pressure mapping indices.

**Correlation between Peak Interface Pressure and Pressure Gradient**

The study analysis identified a large correlation coefficient (0.77) between peak interface pressure and the pressure gradient at a distance 1.5cm from the point of the peak interface pressure. Another large correlation (0.78) was determined to exist between the peak interface pressure and the gradient at 2.5cm. A third large correlation (0.91) was found between the gradient at 1.5cm and that at 2.5cm. This last correlation is to be expected as both gradients have been measured from the point of peak interface pressure.

The Defloor (1999) conceptual framework comprised two causal factors, namely compressive force (or pressure) and shearing force. Defloor (1999), Reger et al. (2010) and Takahashi et al. (2010) have all highlighted that pressure and shear accompany one another through localised pressure compressing tissue and thereby distorting adjacent tissues. Mueller et al. (2005), Rithalia (2004) and Rithalia (2005) have also highlighted that high pressure gradients are known to generate
large shear forces. The high level of correlation between peak interface pressure and pressure gradient therefore suggests that as peak interface pressure increases the area surrounding the site of the peak interface pressure becomes increasingly subject to higher gradients and hence to higher shearing forces.

As described in Equations 5 and 6 in Chapter Three, the pressure gradients at 1.5cm and 2.5cm are dependent on the difference in pressure between the point of peak interface pressure and the pressure reading at the 1.5cm and 2.5cm mark respectively. The large coefficient of correlation between peak interface pressure and the gradients at both 1.5cm and 2.5cm therefore demonstrates that there is a direct relationship between these variables. The difference in pressure between the peak interface pressure point and that at 1.5cm and 2.5cm distant increases as the peak interface pressure increases.

The mean pressures experienced at the various points together with the mean gradients as shown in Table 4 provide indications of the nature of the area surrounding the point of peak interface pressure as follows:

- The pressure is experienced to the 1.5cm distance in a ‘V’ shape rather than as a ‘U’ or bathtub shape. In the event that the latter description was correct the gradient to 1.5cm would be essentially flat, with a steep drop off after that point. This is not the case and the pressure experienced to the 1.5cm distance can be considered as analogous to an inverted classical mountain top (or a ‘V’ shape).
The area around the peak interface pressure point that is subject to the highest pressure gradient is restricted in size, and increases slowly with increasing pressure. The correlation between peak interface pressure and gradient at 1.5cm range indicates that these two indices increase together. In the event that the area subject to high pressure expanded with increasing interface pressure, the gradient to 1.5cm could be expected to remain static or reduce as either of these scenarios would have a lower correlation coefficient.

The impact of pressure reduces with distance from the point of peak interface pressure. The pressure gradient in the region between 1.5cm and 2.5cm from the point of peak interface pressure is less than that to 1.5cm. The distance from 1.5cm to 2.5cm can therefore be considered as a more gently inclined or flattened ring surrounding this conical region.

In this way this research supports the concept that pressure is transmitted into the tissue layers in a conical formation or V-shaped pressure gradient (Maklebust & Sieggreen, 2001; McClemont, 1984). McClemont (1984) also noted that whilst the point of the cone may provide visual indications, the whole of the cone needs to be considered in prevention management.

The high level of correlation between peak interface pressure and the pressure gradients at both 1.5cm and 2.5cm show that the area at the base of the ‘cone-like’ pressure damaged area remains essentially constant rather than
increasing as the peak interface pressure increases. Currently there is no empirical measurement of the dimensions of this underlying tissue damage. It may be that staggered measurement of pressure gradients can be used to provide demarcation of the area of deep tissue damage. This study has only investigated gradients to 1.5cm and 2.5cm but it would be interesting to calculate a wider range of gradients to see if there were clearly observable boundaries. From these measurements a mathematical description of the distribution of pressure across the affected area could be developed. In addition, from that description, and with a time dimension included, it would be possible to determine the total amount of pressure contained within that pressure intensity distribution and to more closely examine the nature of the inverse pressure – time relationship as cited by Stekelenburg et al. (2008).

As explored in the review of the literature the internal pressure may be many times the pressure recorded on the skin surface (Collier & Moore, 2006; Maklebust & Sieggreen, 2001; McClemont, 1984). Shearing is also understood to occur predominantly in the deep tissue and has the effect of reducing the amount of pressure necessary for vascular occlusion (Bennett et al., 1979; Defloor, 1999; Maklebust & Sieggreen, 2001). The potential formation of deep tissue injury is understood to be indicated by high pressures at the deep tissue level (Baharestani et al., 2009; Nixon et al., 2005; Oomens et al., 2009; Stekelenburg et al., 2008) and high pressure gradients across the interface surface (Ankrom et al., 2005).

Rithalia (2004) has stated that high pressure gradients occur around bony prominences and small variations in pressure sensor position can provide large variations in measurements. A strength of the current study is that this issue was avoided by developing an interface pressure map of the entire patient buttock.
region and interface area then measuring the gradient around the peak interface pressure point irrespective of where this occurred. Peak interface pressure variability was observable in both time and space through the use of the Tekscan 2D Contours View and the Peak Interface Pressure vs. Time Plot. The impact of variability was reduced through the use of the averaged peak interface pressure and the averaged gradients from the ten interface pressure measurements.

This thesis contributes to the understanding of deep tissue injury through the exploration of pressure gradients and the application of interface pressure technology. The measurement of pressure gradient however requires specialised and expensive equipment, and processes that are not currently available to the clinician nurse in ward areas. Whilst variations to current clinical practice would be required to take advantage of interface pressure mapping in the routine assessment of hospital patients, this technology will be significantly less expensive than reliance upon alternative measurement techniques such as Magnetic Resonance Imaging.

Implications of Lack of Other Correlations

As noted earlier no correlation was observed between the Waterlow Risk Assessment Tool risk score and any of the other risk factors or pressure mapping indices, thereby supporting comments made by Torra i Bou et al. (2006) and others that question the efficacy of risk assessment tools in general. Stekelenburg et al. (2008) suggest that limitations in the overall ability of risk assessment scores to accurately predict the subsequent occurrence of pressure injuries may be a result of
them not accounting for the complete array of factors involved in pressure injury development. Should this view be correct, the implications are that risk assessment scoring systems would become more complex, more difficult for clinical nurses to use, and hence reduce even further the likelihood of them being accurately employed in a clinical setting.

Swain and Bader (2004) have reported that no link has been discerned between weight and interface pressure, and between Body Mass Index (BMI) and interface pressure. Defloor (1999) has however indicated in his conceptual framework (see Figure 2) that body build, and by extension weight, is a contributing factor for the intensity of compressive force as a causal factor for pressure injury development. This study has shown no correlation between peak interface pressure and weight, nor between peak interface pressure and BMI, thereby suggesting that further research is required in this area.

**Investigation of Shape**

This section details the exploration of the observed two-dimensional shape of patients through the use of interface pressure mapping and the correlation between shape and the selected risk factors for pressure injury and the pressure mapping indices. The relevance of shape and the correlations to the Defloor (1999) conceptual framework, and hence to the development of a pressure injury, will also be discussed.
The second study question investigated the two dimensional anatomical shapes of the buttock region formed by a body lying 30 degrees supine upon an interface pressure measurement mat. The area of the shapes includes the sacral and ischial areas which are known as high risk regions for pressure injury development (Dassen et al., 2006; Hagisawa et al., 2004; McClemont, 1984). The means of obtaining interface measurements and determining shape was detailed in Chapter Three.

Statistically significant shape dependent effects were observed for BMI, weight and Waterlow risk assessment score. No shape dependent effects were observed for the pressure mapping indices, namely peak interface pressure and pressure gradient. These results will now be further discussed in more detail.

**Exploring the Effect of Shape on BMI**

In this study it was observed that there was an effect of shape on BMI and weight through the application of the Mann-Whitney U test. Higher BMI and higher weight correlated with the round/square shapes with this group having a BMI of 28 compared to 24 and weight approximately 10kg higher. Given that weight and BMI are linked as described in Chapter Three, further discussion will be limited to the effect of shape on BMI.

The measurement of BMI provides an indication of the nutritional status of the individual (Ousey, 2005; Posthauer & Thomas, 2008). Furthermore, BMI is considered as a more accurate tool for evaluating nutritional status than weight
alone as increased weight does not necessarily equate to a better nutritional status (Baranoski & Ayello, 2008). A link between poor nutritional status and the risk of pressure injury development has also been suggested (Black et al., 2011; Collier & Moore, 2006; NPUAP & EPUAP; 2009; Ousey, 2005). The relationship between BMI and shape may therefore be useful in this regard. Patients with a BMI in the range 18.5 to 25 are considered to have a healthy weight/height ratio. Individuals with a BMI greater than 25 are considered to be overweight, whilst those under 18.5 are considered underweight (WHO, 2010).

Wells et al. (2007) have also identified that the human body shape can be a source of information about health risks in patients. Whilst the Wells et al. research has focussed on the utility and application of three dimensional all-of-body shape information for disease risk assessment, the shapes investigated in this study are consistent with their approach as the shapes examined herein represent a two dimensional view of the three dimensional buttock region lying 30 degrees supine. Wells et al. (2007, p 419) have also suggested that body shape has utility as a “marker of health status” and that a “clinical focus on shape may achieve greater ‘connection’ with the patient than does BMI, which is difficult for the layperson to calculate and interpret”.

This study has shown that round/square shapes are associated with higher BMI, and by extension may be an indicator of higher pressure injury risk probably due to altered nutritional status. Whilst the implication is encouraging as a pilot study the relationship observed does not recognise the greater risk associated with underweight BMI measurements, nor malnutrition. Further research is therefore required in order to determine the impact of each shape individually and whether a
link to malnutrition can be established. Should such a link be shown, a simple catalogue of shape diversity may therefore provide information on weight distribution and hence on health risks, including the risk of pressure injury in immobile patients. The clinical applications of shape can be seen to be as follows:

- Patient shape information could be used as an adjunct to risk assessment tools, and
- Patient shape information could be used on admission as a replacement to a risk assessment using a formal risk tool.

Shape is not a feature of either the Defloor (1999) or the Braden- Bergstrom (1987) conceptual frameworks, although both frameworks acknowledge the importance of tissue tolerance in the development of pressure injury. The Defloor (1999) framework does specifically include tissue mass as a component of tissue tolerance. However, this concept is mentioned only in reference to paralysed patients losing muscle mass over bony prominences. A link between tissue tone and body shape has been suggested by Swain and Bader (2004) thereby providing tacit, although as yet unproven, acknowledgement that shape may be an important factor in pressure injury risk assessment.
Exploring the Effect of Shape on Waterlow Risk Assessment Score

The study identified a medium effect on risk score as a result of shape. Patients exhibiting a round/square shape were identified to have a median Waterlow risk score of 12 whereas the median risk score for the other shapes was 9.5. This difference indicates that those having a shape of round/square shape may be likely to be at risk of developing a pressure injury as determined by the Waterlow Risk Assessment Tool.

As previously discussed, risk assessment is a critical element in pressure injury management (Baranoski, Ayello & Langemo, 2008; Mastronicola & Romanelli, 2006). Risk assessment tools are commonly used to provide a means for standardisation and to overcome issues with inexperienced nurses making clinical judgements and the importance is also highlighted of documented risk assessment to address potential litigation (Pancorbo-Hidalgo et al., 2006; Lyder 2006). Despite this, the calculation of risk of development of a pressure injury is a somewhat inexact science as indicated by studies undertaken by Anthony et al. (2010), Papanikolaou et al., (2003) and Torra i Bou et al. (2006). Pressure injury risk calculations are a multi-variable problem as evidenced by the structure of assessment tools, including the Waterlow Risk Assessment Tool. The ability to accurately predict the development of pressure injury is therefore, currently, extremely limited (Defloor & Grégorie, 2004; Kottner & Balzer, 2010; Ousey, 2010; Papanikolaou et al., 2003; Torra i Bou et al.). The simplicity of these tools may be the reason for their popularity and that they should only be used as an adjunct, rather than a replacement, to clinical judgement (Jull and Griffiths, 2010).
Defloor (1999) has highlighted the link that should exist between risk assessment tools and conceptual frameworks, but has also noted that the structure of most risk tools are not research based. The Waterlow Risk Assessment tool, developed over 30 years ago through empirical study, is an example of this situation. An improved ability of risk assessment tools to accurately identify those at risk, coupled with increasingly effective preventative measures, can therefore be expected to lower the incidence of pressure injury development. The utilisation of shape may be an easy to implement addition to clinical judgement and could be used in conjunction with a more formal risk assessment using the Waterlow Risk Assessment Tool.

**Exploring the Implications of the Lack of Other Shape Effects**

Swain and Bader (2004, p.43) have noted that “the shape of a subject will have an effect on the interface pressure”. Swain and Bader (2004) further suggest that the effects on interface pressure due to anatomical characteristics are likely to be subtle, and that patients with similar body types can exhibit significantly different interface pressure. As noted previously no effect due to shape was observed for the pressure mapping indices, namely peak interface pressure and pressure gradient. The implication of this finding may be that variations of shape for an individual over time are important with respect to interface pressure, rather than the ability to correlate shape and interface pressure across a complete population at a single point in time. This issue therefore requires further research.
In a clinical environment shape can be visually measured for patients in potential risk groups by non-invasive methods. Swain and Bader (2004) suggest that, for clinical use, a graphical representation of the interface pressures and of the patient-surface interface area (that is, the shape) may be a more important representation that the absolute accuracy of the interface pressure measurements alone. They also suggest that at risk patients will demonstrate a change in shape due to loss of muscle tone (thus linking back to Defloor’s (1999) ideas). Whilst the findings within this study do not necessarily support the Swain and Bader (2004) statement, further longitudinal studies may indicate whether shape change may be precursor to increased risk of pressure injury development. Should the Swain and Bader (2004) postulation with respect to shape and risk be correct, shape may be a clearer conduit to achieve early indication of increased pressure injury risk than measurement of the interface pressure itself.

**Amended Conceptual Framework**

At Figure 2 the Defloor (1999) conceptual framework was presented. This framework acknowledged the important role played by medical and nursing interventions for patients deemed to be at risk, and Defloor (1999) also noted that preventive measures can only be considered successful if they contribute to a decrease in the incidence of pressure injury. The Defloor (1999) framework is however a static framework depicting what is essentially a dynamic process. The findings from this study, together with ideas proposed in the more recent literature and application of more contemporary terminology, have been used to amend the Defloor framework.
The amended conceptual framework presented at Figure 4 maintains the underlying basis of the Defloor (1999) framework and has the following broad structure, namely:

- The causal factors of pressure and shear are maintained.
- The location of the pressure attack is included. This inclusion is consistent with the Defloor (1999) statement regarding the transmission of the majority of interface pressure to underlying tissues when the superficial covering is thin. It is also consistent with the views of a range of authors who argue that tissue geometry and the location of bony prominences will influence the internal mechanical conditions (Bader & Oomens, 2006; Collier & Moore, 2006; Hagisawa et al., 2004; Swain & Bader, 2004).
- The causal factors are combined prior to the consideration of intrinsic factors. This is consistent with statements by a number of authors that pressure and shear occur together (Defloor, 1999; Reger et al., 2010; Takahashi et al., 2010).
- The ability of the tissue to tolerate a force is maintained from the Defloor (1999) framework. Tissue tolerance is influenced by a range of intrinsic factors that affect the predisposition of an individual to pressure injury development (Bader & Oomens, 2006; Collier & Moore, 2006; Defloor, 1999; Defloor & Grypdonck, 2004; Swain & Bader, 2004).
A ‘feedback’ loop is included to highlight the ongoing effects that medical/nursing interventions have on pressure and shear, and hence on pressure injury risk and development (Defloor, 1999; Defloor & Grypdonck, 2004). For example, the placement of a patient on a pressure-reducing support surface will influence the interface pressure and shear being experienced, and hence affect the manner in which pressure impacts on the tissue.

The framework focuses predominantly on pressure injury prevention rather than pressure injury development. The formation of a pressure injury may be a failure of the preventative processes.

Terminology is updated, namely ‘pressure injury’ has been adopted in lieu of ‘pressure ulcer’. The term ‘forces’ is now applied to describe the direction that the force acts upon the tissue.
Figure 4. Amended Conceptual Framework 2011 for Pressure Injury Prevention adapted from DeFloor Conceptual Scheme 1999
In addition, a clinical practice dimension is included within the framework. This additional dimension is designed to (1) reinforce the application and importance of the conceptual framework to the clinical setting, (2) provide guideline for clinicians regarding the assessment and support tools that are applicable in the pressure injury prevention process, and (3) provide a basis for ongoing pressure injury prevention education. The contribution to clinical practice that has been identified from this study is the potential application of visualisation technologies to augment, and potentially reduce, the reliance on risk assessment tools. As discussed in this thesis beneficial visualisations can be provided through (1) the interface pressure mapping output for the determination of areas subject to the highest pressure gradients and hence, in combination with the site of the pressure attack at the greatest risk of pressure injury development, and (2) the overall shape of the interface surface and how this might vary.

**Strengths and Limitations of the Study**

The great strength of the study is the utilisation of interface pressure mapping visualisation to inform aspects of clinical practice. The study also contributes to the understanding of deep tissue injury through the exploration of pressure gradients and the application of interface pressure mapping technology. A further strength is that development of an interface pressure map of the entire interface surface overcomes the issue of variability that has been associated with the use of a single pressure sensor.
The nature of the sample (convenience) limits the generalisability of the findings to populations with similar demographic and clinical characteristics. There are some limitations to the study related primarily to technical aspects of pressure mapping. In a technological sense a limitation arises from the 1 cm x 1 cm sensel structure of the Tekscan measurement mat. This structure involves a series of discrete rather than continuous measurements across the measurement area, with interpolation between measurements being provided by the Tekscan software. The interpolated values have been consistently used for the pressure readings and for the determination of pressure gradients in order to avoid the ‘stepped’ or ‘pixcellated’ nature of the raw data and to have available a finer granularity.

Maximum interface pressure readings were taken from wherever they occurred on the mat, rather than at the same site (such the sacral region) as the aim was to investigate the gradients around the peak interface pressure rather than the gradient associated with any particular site. The ability to translate the output of this study to any consideration of site-specific pressure is therefore limited.

Two limitations arise in the manner that pressure gradient information was determined. The first is that pressure measurements were taken at distances of 1.5cm and 2.5cm from the point of peak interface pressure. Although the pressure region has been shown in this study and by others to be conical in overall form (Maklebust & Sieggreen, 2001; McClemont, 1984 for example), it needs to be recognised that pressure does not reduce consistently in all directions with range. As the aim of the study was to utilise maximum gradients that existed around the peak interface pressure points, the 1.5cm and 2.5cm circumferences were examined.
for the lowest pressure at those ranges (the greatest pressure difference from the peak interface pressure). Whilst in the majority of cases the lowest 1.5cm pressure and the lowest 2.5cm pressure were in the same direction, further investigation into pressure mapping is required to determine the finer detailed structure of the conical form.

The second limitation arises in that the 1.5cm and 2.5cm gradients were both determined from the point of the peak interface pressure. Whilst the first measurement has provided valuable information into the nature of gradients around areas of high pressure, the second measurement would have been more useful if it had been measured either 1cm or 1.5cm outward from the 1.5cm point. This would have avoided the obvious high level of correlation between the gradient at 1.5cm and that at 2.5cm and would have provided an insight into the structure of the gradient as the range from the point of peak interface pressure increases. The area affected by large pressure gradients could therefore have been investigated. This is an area considered worthy of further study.

Determination of shape has been based solely on visual observation and interpretation and shapes have been amalgamated into two broad categories. Given that an effect due to shape has been determined for weight, BMI and the risk score (as determined by the Waterlow Risk Assessment Tool) as a pilot study, additional work on developing a more stringent series of shape determination guidelines could be useful in further examining this area. This further consideration should include each shape separately as this may provide additional information for the utilisation of shape in clinical application.
Summary

This study has shown that strong correlations exist between peak interface pressure and the pressure gradients measured at 1.5cm and 2.5cm from the site of the peak interface pressure. Whilst some correlation could have been expected, the strength of the correlation and the nature of gradients provide some insight into the distribution of pressure in the region surrounding the peak interface pressure point. The results of this study reinforce the conical pressure distribution as first postulated by McClemont in 1984. Moreover, the utilisation of interface pressure mapping as described herein can contribute to the identification of potential areas of deep tissue injury through the determination of areas subject to high pressure and high pressure gradients.

Round/square shapes have been indicated as a pilot study to have higher median weight, higher median BMI and a higher median Waterlow risk assessment score than the combination of other shapes. This association may provide a simple indicator for increased risk of pressure injury development, whether through poor nutritional status or some other factor. In clinical practice the ability to conduct rapid visual assessment of buttock shape would inform a similar rapid decision on the need for preventative and management strategies to be developed and implemented. Such simplified assessment techniques would assist clinical nurses in the provision of preventative management strategies. The final chapter of this thesis will explore the policy, clinical education and practice implications of the findings and make recommendations for further research.
CHAPTER 6 – CONCLUSION

Introduction

This chapter presents the conclusions to this study entitled ‘Exploring known risk factors for pressure injury with visual technology’. The study utilised interface pressure mapping technology to investigate the following aims:

1. Explore correlations between two interface pressure mapping indices and selected risk factors for pressure injury, and
2. Explore the visual anatomical characteristics of the buttock region of patients through the use of interface pressure mapping and the correlation between shape and selected risk factors for pressure injury.

The use of pressure mapping systems, and the associated visualisation of the distribution of pressure across the interface surface, has allowed the identification of areas of high pressure on tissue at the interface and the calculation of associated pressure gradients. The widely held view that high gradients give rise to high shear forces, and that high shear increases the impact of pressure, means that the ability to visualise gradients may provide an almost immediate indication of areas that may be prone to pressure injury development, including the development of deep tissue injury. In addition the shape of the area in contact with the support surface has been able to be viewed and categorised. While some researchers argue that there are limitations to pressure mapping due to measurement variability and difficulties in
clinical application, the ability to utilise this technology to view areas subject to high pressure gradients may see a resurgence in its use.

The value of a conceptual framework to describe pressure injury development has been re-examined within this thesis and an amended framework, based on that of Defloor in 1999, has been developed. The new framework includes consideration of the combined pressure and shear forces, places greater emphasis on the medical and nursing interventions, and importantly considers the overall pressure injury prevention process as a dynamic process and one that may be better understood through visualisation of key risk factors. The formation of a pressure injury is seen as a failure of the ability to assess pressure injury risk and manage the causal factors. In addition, the new conceptual framework updates the terminology to provide more contemporary descriptors.

This study has contributed to the knowledge base pertaining to pressure injury risk assessment through the investigation of sick patients in a busy clinical hospital environment. The study has suggested that the utilisation of visual interface pressure mapping technology, and an assessment of interface shape, may provide a valuable adjunct, or possibly an alternative, to the use of risk assessment tools.
Implications of the Study

The study has implications in a number of areas associated with pressure injury development. These implications are considered under the broad categories of policy, clinical practice and education, and further research.

Policy Implications

As noted at the commencement of this thesis, pressure injuries have been accepted as an indicator for the quality of care by health services. Jull and Griffiths (2010) note however that this acceptance assumes that health providers have the necessary tools through which to take action to address the pressure injury problem. One of the most important tools currently utilised by clinicians is a risk assessment tool. However this study did not however show any correlation between high Waterlow risk assessment scores and either peak interface pressure or pressure gradient. Therefore these findings add to an increasing body of evidence that suggests pressure injury risk assessment tools have significant limitation in the prediction of risk of developing pressure injury.

One means to redress this situation may be through clearly basing risk assessment tools on a conceptual framework that describes how the various risk factors impact on pressure injury development. In this thesis Defloor’s conceptual framework, developed over a decade ago, has been expanded and updated. This new framework may serve as a basis for consideration for the development of improved risk assessment tools.
Clinical Practice and Education Implications

The utilisation of pressure mapping technology as discussed in this thesis has implications for clinical practice. Interface pressure mapping technology has the potential to provide a real time view of the pressure intensity function; conceptually similar to the monitoring of heart rate and other vital signs. Real time peak interface pressure and pressure gradient display would provide a visual cue to alert clinicians to instigate preventative measures before pressure increases to destructive levels. As pressure injuries are often discovered after irreversible tissue damage has already taken place, the availability and use of interface pressure mapping technology in high risk points throughout hospitals, for example emergency departments, intensive care units and operating theatres, could be a valuable contribution to the monitoring of at-risk patients and the prevention of pressure injuries. The introduction of this technology may target efficiencies in cost from prevention and early identification of pressure injury development.

The ability to view peak interface pressure and pressure gradients through visual technology will provide educational benefits. This technology will help clinicians to have a better understanding of the causal factors for tissue damage and especially the risk of deep tissue injury. The education of clinicians, patients and external care-givers through the visualisation of interface pressure mapping will provide a better appreciation of how support surfaces and other factors impact on interface pressure and associated pressure gradients. An improved understanding of these factors should result in more focused preventative interventions and better overall pressure injury risk management.
Recommendations for Further Research

A large number of recommendations can be made for further research as a result of the outcomes of this study. The initial recommendation is for additional work to be done with respect to shapes to determine the extent to which this may be a clinical indicator of pressure injury development of risk. Further analysis of data for each shape individually could be undertaken to determine whether this claim can be supported. Additional studies are also required to confirm whether a relationship exists between patient’s shape and increasing risk of pressure injury. This study has identified a relationship between shape and weight, BMI and Waterlow risk assessment score. In addition the potential of the pressure mapping technology to be able to provide 2D and 3D images has not been fully explored.

A number of areas for further study are associated with the investigation of pressure gradients. It has been observed earlier in this study that measurement of both the 1.5cm gradient and the 2.5cm gradient from the peak interface pressure point resulted in an expected high correlation between these variables and failed to illuminate the true nature of the gradient to the 2.5cm distance. Further investigation to explore all points on the gradient between the peak interface pressure point and the 2.5cm would provide more information on the structure and extent of the pressure affected area (rather than the single 1.5cm point used in this study). In a similar manner extension of the measured area beyond the 2.5cm distance, and the determination whether gradients at these extended distances correlated with interface pressure could also be undertaken. This analysis could be useful in determining the typical extent of the pressure affected region around a pressure point, and whether this area increases with increasing pressure. Currently
this particular areas of study is currently somewhat restricted by the present limitations of interface pressure mapping technology.

Further research is recommended in the area of risk assessment tools, and particularly in the structuring of risk tools to a conceptual framework. Further study into the utilisation of visual techniques, and the utilisation of these techniques in risk assessment, is also believed to have potential to provide improved outcomes for the prevention of pressure injury.

**Conclusion**

The findings from this study have enabled refinement of Defloor’s conceptual framework for prediction and prevention of pressure injury. Investigation of the correlation between peak interface pressure and pressure gradients around the peak pressure point has contributed to the understanding of deep tissue injury. The study has also shown that the utilisation of visual interface pressure mapping technology, and an assessment of interface shape, may provide a valuable adjunct, or possibly an alternative, to the use of risk assessment tools. This study has greatly contributed to the knowledge of nurses pertaining to the care of patients through increased understanding of pressure injury risk assessment.


Ann Marie Dunk


Is it time for a new descriptor 'pressure injury': a bibliometric analysis

Dunk AM & Arbon P

Abstract
Prediction, prevention and management of pressure injuries are areas that require specific attention from nurses in clinical practice. Moreover, increased awareness that these injuries are preventable is an important precursor to changing nurses' practice and reducing the incidence of pressure injuries. The language and terminology that we use in daily practice can impact on the understanding and approach that nurses take to care delivery. In this arena of wound care practice commonly used terminology that emphasizes the nature of the wound, rather than its causation, may be a significant factor that limits the level of concern about prevention and responsibility taken by clinicians. This paper argues that the term 'pressure injury' promotes a better understanding of the fact that these wounds are preventable and may refocus the attention of nurses providing care to at-risk patients.

Introduction
A change in attitudes and belief may be influenced through the choice of terminology. This paper focuses on the role of nursing terminology as an influence on nursing care now practiced in the prevention of pressure injury.

The International Classification for Nursing Practice (ICNPs) emphasizes the need for unifying approaches to promote integration and harmonisation of nursing terminologies across countries and languages.

The primary motivation for a unified nursing language system is to be able to communicate and compare nursing practice furthermore underpinning research evidence across settings, countries and languages. This unification of nursing terminology supports the further development of the discipline in areas such as clinical decision-making, evaluation of nursing care, improvement of patient outcomes, development of health policy and generation of knowledge through research.

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Specifically, this paper contributes to the development of international nursing terminology and argues for a review of the terminology used to describe the tissue damage caused by unrelieved external pressure on tissues resulting in damage to the underlying tissues. Emphasis is placed on the definition of this condition by causation; that is as an injury, thus moving the focus of nursing care to prevention.

Common nursing terminology used to describe this injury includes pressure ulcer, pressure sore, decubitus ulcer and bed sore, with each term focusing on tissue damage rather than causation. Deliberate choice of language and focus on prevention has the potential to promote early intervention and to significantly improve patient outcomes. This paper reports on the results of a bibliometric analysis of nursing terms used to describe pressure injury in research papers in published journals sourced from CINAHL and MEDLINE databases covering a 5-year period in the English language.

Background
History has shown that pressure injuries are not a modern occurrence, with evidence of treatment as early as the XXI Dynasty. Whilst debate as to treatment has evolved over the centuries, in the later part of the 20th century the movement towards a focus on prevention of pressure injury has been evident.

The nursing role in pressure injury management is significant in that nurses are the prime deliverer of wound care and have the ability to determine and institute preventative practice measures for those considered to be at risk and coordinate multidisciplinary teams in the management of the wound. To be able to effectively prevent the development of a pressure injury, the clinician must understand the pathophysiology and causes that contribute to the development of the injury.
and this fact may be one of the influences that has resulted in the use of different terms to describe this common problem over time.

Current terminology

Tissue damage resulting from abnormally sustained pressure may be referred to as a bed sore, pressure sore, decubitus ulcer, pressure ulcer or pressure injury. All of these terms refer to the same problem encountered by many patients and all are caused by sustained pressure resulting in ischemia⁷. Lately, the term pressure ulcer has been promoted as it is thought to more accurately reflect the pathology of pressure-derived tissue degradation and the characteristics of the resulting lesion⁷.

Prevention and management of pressure injuries is now the focus of international debate⁸. The strategy for prevention includes recognising the level of risk, decreasing the effects of pressure, assessing and improving nutritional status, avoiding excessive bed rest and prolonged sitting and preserving the integrity of the skin. The principles of management include assessing severity, reducing pressure, friction and shear forces, optimizing local wound care and management, removing necrotic debris, managing bacterial contamination and correcting nutritional deficits. Scientific research addressing both prevention and management has been extensive and noted in the literature over the past 4 decades⁹.

Guidelines have been established by international professional bodies for the classification of severity of the injury according to characteristics of the wound and/or surrounding tissue. Both the European Pressure Ulcer Advisory Panel (EPUAP) and the American National Pressure Ulcer Advisory Panel (NPUAP) have published clinical practice guidelines over the past 20 years. In 2003 the Australian Wound Management Association published clinical practice guidelines for the prevention and treatment of pressure ulcers⁹.

These national and international bodies have focused on the prevention of pressure injuries in an attempt to curtail the incidence and prevalence of pressure injuries among at-risk patients. There is now a move in pressure injury management focus away from treatment and toward early risk assessment and preventive management. Consequently it is timely to consider the terminology employed by nurses and specialists in wound management in describing this form of injury and to encourage the use of terminology, which underlines the fact that the problem is preventable and should be understood as an injury, frequently associated with inadequate preventative care.

Method

This project utilised aspects of classic bibliometric analysis technique in a pilot investigation to determine patterns in the usage of common terminology for pressure injury. The study was unfunded and set out to trial the potential value of bibliometrics in informing our understanding of the nursing terminology used in this field of wound care. Terminology currently and consistently used throughout the international academic discourse to describe pressure injury includes the following terms: bed sore, decubitus ulcer, pressure sore, pressure ulcer, pressure necrosis, ischemic ulcer, pressure wound and pressure injury.

The bibliometric method uses empirical data and quantitative analysis to trace formal communications in published literature and to study the patterns of publications within a field. The pilot used several elements of the bibliometric approach. Publication counts, a basic tool in bibliometric analysis, provided a descriptive and quantitative indicator of the prevalence of common pressure injury terms. The study mapped the development of descriptive language used by health clinicians to describe pressure injury over time.

Bibliometric analyses use objective publication data and do not attempt to interpret or assess the content or quality of publications or the motivations of the researchers⁰.

Data

The bibliometric data was obtained by searching two international literature databases: the Cumulative Index to Nursing and Allied Health (CINAHL) and MEDLINE (OVID). The following search terms were utilised to gather the data:

- Pressure sore.
- Pressure injury.
- Bed sore.
- Pressure necrosis.
- Pressure ulcer.
- Ischemic ulcer.
- Decubitus ulcer.
- Pressure wounds.

Each database was utilised to search for the eight terms used to describe pressure injury. Findings of the two database searches were combined and duplicate journal articles were deleted from the search.

Papers published in the academic literature that met the following inclusion criteria were accepted:

- Published in the English language.
- Academic journal articles only.
- Published in the years between 2001 and 2006.
- Have one of the eight search terms in either the title, keywords listed for the article or in the abstract.

Papers excluded from the data set were:

- Commentary and editorial papers.
- Papers where none of the eight search terms were listed in the title, keywords or the abstract.
- Papers where the abstract indicated no relevance to the pressure injury topic.

The final search resulted in more than 3400 articles, which were then screened to remove any duplication and assessed against the inclusion and exclusion criteria. Once all articles were reviewed, a total of 1256 articles obtained from 398 journals remained in the data set and were retained for analysis. Database searches were undertaken in mid-2007. Author citation patterns and journal impact factors were evaluated using the Web of Science citation index.

Results
The results presented here arise from a relatively simple descriptive analysis of the data using several typical bibliometric techniques. The relative prevalence of each of the eight terms in the final (cleaned) data set was roughly equal across the two electronic literature databases. For example, the most commonly used (prevalent) term recovered from articles cited in MEDLINE was also the most commonly used term in articles listed in CINAHL.

Growth in the field
Over all terms the results showed an increase in publication volume until 2002 (Graph 1). However, 2006 showed a considerable decline in publication across all categories. It is unlikely that this result is due to a lag in recording papers within the databases because both databases are updated regularly. Results also show that authors seem to be using one of the eight key terms in the title more frequently than in the abstract or in both the abstract and title.

Graph 1. Growth in field.

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Journal patterns

The 1,756 articles included in this analysis were published in 398 different journals. Analysis of the journal patterns included comparison of the number of articles focused on pressure injury (and other terms) for each journal title. The top ten journals with the most publications are listed in Table 1.

Bradford’s Law provides a general guideline for describing the distribution of academic papers across journals in a field of study. The characteristic pattern of distribution of articles was first described by Samuel C. Bradford in 1934 and demonstrates the exponentially diminishing returns arising from extending a search for references in the scientific literature. According to Bradford’s Law, about one third of all journals publishing in a field will contain most articles, a second third of the journals publishing in the area (middle group) will contain a smaller number of articles and the remaining one third will contain only one or two articles in each journal. For librarians, students, and researchers, the underlying message is that extending a literature search beyond the top one third of journals that dominate publication in that field results in little additional return. The law can be expressed as a ratio of $1:n^2$. In this project the top eight journals with the highest number of articles published accounted for 39% (n=608) of all articles published over the 5-year period. The middle group accounting for 45 journals captured a further 32% (n=567) of all articles and, finally, the 345 journals with low publication rates for this field of study accounted for 33% (n=561) of articles. While Bradford’s Law is not intended to be a precise measure, it does provide a good estimate of the typical distribution of papers across scientific journals and fields of study. In this pilot study the best fit formula ratio is $1:n^2$ ($1:n^2$ – where n = the Bradford Multiplier).

Author patterns

Eleven key authors were identified from the 1,756 articles. Prolific authors were defined as authors with four or more articles published during the 5-year study period following the example of Eshbanka, Werthner & Dickson. Table 2 shows the top 11 authors and the number of articles published by each over the study period.

Table 1. Top ten journal details.

<table>
<thead>
<tr>
<th>Journal name</th>
<th>No of articles</th>
<th>Discipline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ostomy Wound Management</td>
<td>127</td>
<td>Multidisciplinary</td>
</tr>
<tr>
<td>Advances in Skin and Wound Management</td>
<td>113</td>
<td>Multidisciplinary</td>
</tr>
<tr>
<td>Journal of Wound Care</td>
<td>89</td>
<td>Multidisciplinary</td>
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<tr>
<td>British Journal of Nursing</td>
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<td>Journal of WOCN</td>
<td>67</td>
<td>Nursing</td>
</tr>
<tr>
<td>Journal of Trauma Visibility</td>
<td>53</td>
<td>Medicine</td>
</tr>
<tr>
<td>Nursing Times</td>
<td>46</td>
<td>Nursing</td>
</tr>
<tr>
<td>Nursing Standard</td>
<td>34</td>
<td>Nursing</td>
</tr>
<tr>
<td>Primary Intention (newer:</td>
<td>31</td>
<td>Multidisciplinary</td>
</tr>
<tr>
<td>Wound Practice and Research</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wounds: A Compendium of Clinical Research and Practice</td>
<td>30</td>
<td>Multidisciplinary</td>
</tr>
</tbody>
</table>

Table 2. Author patterns.

<table>
<thead>
<tr>
<th>Author name</th>
<th>Number of articles</th>
<th>Number of pressure injury-related citations in 2004-2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hampton S</td>
<td>22</td>
<td>2/3</td>
</tr>
<tr>
<td>Ayello EA</td>
<td>17</td>
<td>9/2</td>
</tr>
<tr>
<td>Collins F</td>
<td>17</td>
<td>1/0</td>
</tr>
<tr>
<td>Thomas D</td>
<td>11</td>
<td>0/5</td>
</tr>
<tr>
<td>Collins N</td>
<td>9</td>
<td>2/0</td>
</tr>
<tr>
<td>Clark M</td>
<td>8</td>
<td>3/2</td>
</tr>
<tr>
<td>DeFoor T</td>
<td>8</td>
<td>16/15</td>
</tr>
<tr>
<td>Lyder CH</td>
<td>8</td>
<td>0/0</td>
</tr>
<tr>
<td>Moore Z</td>
<td>8</td>
<td>2/7</td>
</tr>
<tr>
<td>Russell L</td>
<td>8</td>
<td>2/0</td>
</tr>
</tbody>
</table>

Lotka’s Law describes the typical frequency of publication of authors in a field of study. The number of authors making a contribution is about $1/n^2$ of those making one contribution, where ‘a’ is often nearly two. In other words, the number of authors publishing a set number of articles is a fixed ratio to the number of authors publishing a single article and as the number of articles published by an author increases, authors producing that many publications become less frequent. For example, there may be 1/4 as many authors publishing two articles within a specified time period as there are single-publication authors, 1/9 as many publishing three articles, 1/16 as many publishing four articles and so on.

Citation analysis provides a technique helpful in understanding the impact of an author on the field of study and involves assessment of the frequency and pattern of citations in articles. Table 2 shows the total number of citations accrued for papers by each of the top ten authors.
over a 2-year period. A limitation of this result is that it refers to citations for all of the authors' papers for that year and may include papers in other topic areas. While these authors are quite focused in their field of study, more detailed analysis may have altered the result reported here.

Rate of use of key words
The most common key word utilised across all three categories, including title, abstract and article title, was pressure ulcer (Graph 2). When individually assessed, pressure ulcer remained the most commonly used term for every year from 2001 to 2006 inclusive. Pressure sore was second in its frequency of use across all three categories, followed by decubitus ulcer then pressure wound.

Identifying the structure of the scientific field
Disciplinary basis
Analysis of the data set showed a strong nursing and medical disciplinary focus for journals publishing in this field of study.

Discussion
With the growth of knowledge on the cause and effects of pressure injury, it is timely to consider the potential influence of language and terminology on practice in the field of prevention and management of pressure injury.

Several terms are consistently used in the literature to describe a pressure injury. These include pressure ulcer,
pressure sore, decubitus ulcer, bed sore, pressure necrosis and ischaemic ulcer. These terms are used to describe any lesion caused by unrelieved pressure that results in damage to underlying tissue.

The most commonly used descriptor is pressure ulcer. The *Oxford Dictionary* defines ulcer as "the defect of continuity of the epithelium covering a surface, when forming a defined crater". More recently the BFUAP and NPUAP pressure ulcer prevention and treatment clinical practice guidelines in 2009 defined pressure ulcer as "an area of localised injury to skin and/or underlying tissue usually over a bony prominence, as a result of pressure or pressure associated with shear".

The second most prevalent term, pressure sore, is described in the *Oxford Dictionary* as a sore produced by continued pressure on a part of the body. We now know, however, that there are other contributing factors associated with the development of a pressure injury.

The third favoured term, decubitus ulcer, refers to wounds developed over bony prominences while in the recumbent position, especially the sacrum, heel or occiput. *Decumbere* means "to lie down" in Latin. The term *bed sore* means an ulceration of the buttocks or heels, developed by a constant pressure on a mattress on the invalid's skin.

Whilst the past 5 years have seen growth in the use of the term pressure ulcer in favour of other terms, there has also been a steady increase in the use of decubitus ulcer as a descriptive term.

Prevalence studies have shown that pressure injuries do develop in many other parts of the body, not only affecting invalids and it can be argued that these three most commonly used terms fail to provide a satisfactory "generic" term that adequately describes the injury on most occasions. This is especially relevant to the production of wound care texts and teaching materials.

The *Oxford Dictionary* describes injury as "a wrongful action or treatment especially to the body". The term pressure injury differs from all other common terms in that it draws attention to causation rather than to the description of the wound itself.
It may be useful to review the use of terminology in this field and to emphasise that these injuries are preventable. A focus on causation may influence clinical practice and broaden the range of clinicians involved in the prevention and management of the wound beyond wound care specialists.

This paper has reviewed the terminology most commonly used to describe the wounds arising from prolonged pressure on the tissues published in academic journals over a 5-year period. It identifies that there are numerous descriptors used in clinical practice to describe this singular antonym and argues that the prevalent terms in the literature have severe limitations. A move forward to a standard descriptor for pressure injury is considered to have long-term benefits in education, clinical decision-making and may help to focus the clinician’s attention on their role in early assessment and prevention of the injury commonly caused by prolonged pressure on the tissues, rather than the management of the “end point” injury itself.

Reference
APPENDIX B – GARDNER, DUNK, EGGERT, GARDNER & WELLMAN ARTICLE

Pressure injury: an exploration of the relationship between risk factors and interface pressure

Anne Gardner, Ann Marie Dunk, Marlene Eggert, Glenn Gardner & David Wellman

Abstract
Pressure injuries are a serious risk for patients admitted to hospital and are thought to result from a number of forces operating on skin tissue (pressure, shear and friction). Most research on interface pressure (IP) has taken place using healthy volunteers or mannequins. Little is currently known about the relationship between pressure injury risk and IP for hospital patients.

This relationship was investigated with a sample of 121 adult hospital patients. Pressure injury risk was evaluated using the Waterlow Risk Assessment Tool (WRAT) and IP was measured at the sacrum using a Tekscan Clinitron™ IP sensor mat. Other factors considered were body mass index (BMI), blood pressure, reason for hospital admission, comorbidities and admission route to hospital. Patients were classified according to WRAT categories (low risk, at risk, high risk, very high risk) and then remained still on a standard hospital mattress for 10 minutes while IP was measured.

Participants in the ‘low risk’ group were significantly younger than all other groups (p=0.001) and there were some group differences in BMI. IP readings were compared between the ‘low risk’ group and all of the participants at greater risk. The ‘low risk’ group had significantly lower IP at the sacrum on a standard hospital mattress than those at greater risk (p=0.002). Those at greater risk tended to have IP readings at the low end of the compromised IP range.

This study is significant because it describes a new, clinically relevant methodology and presents findings that challenge clinician assumptions about the relationships between pressure injury risk assessment and IP.


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Introduction

Development of pressure injuries is a serious risk for patients admitted to hospital. Pressure injuries are usually associated with prolonged bed rest but can also develop during short periods of immobility.

The aetiology of pressure injuries is not fully understood. Injuries result from a number of factors exerted on skin tissue, with the most important being pressure (as well as shear and friction). Prolonged pressure applied to a localized area of tissue can result in occlusion of capillary blood flow. This occlusion causes tissue ischaemia, cell destruction and tissue death. The resulting pressure injury not only causes great distress to patients but is also very expensive for the healthcare system. Part of the expense is due to the widespread occurrence of pressure injuries. Recent reports from Victoria indicate that the point prevalence of pressure injuries amongst public hospital patients was 25.8% in 2003, and fell to 17.6% in 2006. A similar study in the ACT reported rates ranging between 18% and 29% across the health sector.

Interface (or contact) pressure (IP) is one component related to pressure injury formation and refers to the pressure applied to a person’s body by the supporting surface. IP is usually measured over a small area using a sensor mat and provides scaled readings (milligrams of mercury – mmHg) that represent the level of forces between the body and the support surface. However, the sensor mat does not distinguish the direction in which forces operate and does not measure shear or friction components.

Interpretation of IP readings and the relationship between IP and pressure injuries is complex. IPs of 30 mmHg and higher exceed average capillary pressure in healthy adults and can lead to capillary occlusion. Indeed, 30 mmHg has been referred to as the critical value for pressure injury development. However, as discussed by Rüdel and others, there is evidence that pressure much lower than this may sometimes cause occlusion depending on individual differences such as length of time of pressure application, age, the collagen content of the dermis and amount of shear forces. Whilst IP is not the same as capillary closing pressure, it remains one of the excitatory factors that can be mitigated by efforts to reduce the likelihood of such patients developing a pressure injury.

Nurses use a range of clinical risk assessment tools to identify those patients at particular risk of developing pressure injuries so that appropriate interventions can be implemented. These tools are based on risk factors identified in clinical settings and investigations. Risk factors can include personal characteristics such as age and sex, as well as various health indicators such as continence status and neurological impairment.

The validity of a risk assessment tool is the degree to which the tool is correctly predicted. Establishing the validity of pressure injury risk assessment tools is difficult because there is no single test to predict the occurrence of pressure injuries. The most common assessment tools usually have good sensitivity but comparatively low specificity, classifying many people at risk who do not go on to develop pressure injuries.

The major difficulty in reconciling acceptable sensitivity (how good the test is at predicting who will go on to develop pressure injuries) and specificity (how good the test is at predicting those who are not at risk) is that the two are usually at odds. The sensitivity of the tools is increased by the use of clinical judgment, but this is not always available.

Pressure reducing surfaces are designed to reduce IP on areas of the body, usually bony prominences, most at risk of pressure injury. Evaluation of the usefulness of these devices is often conducted using equipment to directly measure the amount of pressure exerted at the support surface/body interface. However, the measurement equipment is currently too complicated, costly, sensitive and laborious for routine use in the clinical setting. Therefore, recommendations for use of pressure relieving equipment are based primarily on laboratory research conducted on mannequins, laboratory animals or, at best, healthy volunteers. However, healthy volunteers have patterns of tissue structure and blood flow that are quite different from the tissue structure and blood flow of the frail, ill and elderly patients who require interventions in the clinical setting. It is therefore important to conduct empirical research with the groups towards whom interventions are targeted, so that the relationships between IP, level of pressure injury risk and other individual characteristics can be more clearly understood.

Methodology

As the research partner for an AusIndustry Research and Development Grant, we designed a multisite study to
investigate the relationship between pressure injury risk and the IP with hospital mattresses as part of a larger clinical trial of intervention mattresses. The study population comprised patients admitted to two acute hospitals.

A pilot study with 11 healthy adult volunteers was conducted to develop protocols for the main study and to evaluate the suitability of several mattresses for use as IP measurement surfaces. For the pilot, measurements were taken on both sacrum and trochanter on each of the mattresses. Trochanter measurements were found to be widely variable and measurement at this site was discontinued. The convoluted foam (egg shell) Comfort Plus™ mattress was chosen to represent the standard hospital mattress for the study because IP readings were less dispersed and there was greater test-retest reliability for IP measured on this mattress when compared to another test mattress (cubed foam mattress). Adding to the external validity of using this mattress was that it was widely used in NSW and ACT hospitals at the time of the study, including the participating hospital sites. The final study protocols are briefly described in the methods section below. Full details of study protocols and training manual are available from the author on request.

Method

Design

A prospective multi-site design was used for the study, with a convenience sample of patients. Participants were assessed for pressure injury risk and then had IP measured lying on a standard hospital mattress.

Participants

After gaining approval from relevant Human Research Ethics Committees, participants were recruited from two Australian teaching hospitals. A total of 126 patients consented to participate in the study from a variety of medical, surgical and midwifery wards. Inclusion criteria were that patients were at least 16 years of age and could move or be moved from one bed to another. Only patients thought capable of lying in one position for two 20 minute periods were invited to participate. Patients were excluded if they were clinically too unwell, were being discharged within 3 hours or were expecting surgery within 6 hours.

Two consenting patients were later excluded because they were unable to tolerate the extended period lying motionless required for a component of the larger study. Both patients had a body mass index (BMI) greater than 35 (classified as obese). Three participants had extremely high IP readings (>1.5 interquartiles above the IP mean of participants with similar pressure injury risk), and were subsequently excluded from data analyses; the final sample size was 112. Participants ranged in age from 18-88 years (M=60.10, SD=18.02), and 42% were female (n=50). Eight participants (6.6%) were recruited from obstetric wards, 59 from medical wards (48.8%) and 54 from surgical wards (44.6%).

Survey Instrument

Demographic data, patient characteristics and a pressure injury risk assessment tool were combined into a single survey instrument. Demographic data included age, sex, weight and height. Data collected on patient characteristics included blood pressure, hospital ward (medical/surgical/obstetric), the reason for admission, current comorbidities and admission route to hospital.

Comorbidities were evaluated using the Deyo, Charlson & Ciol™ adaptation of the Charlson Comorbidity Index (CCI), a tool used widely in Australia and internationally. The CCI is designed to measure the level and intensity of comorbid health conditions and contains 19 categories based on the ninth revision of the International Classification of Diseases diagnoses codes (ICD-9-CM). For the current study, selected CCI scales were used to form three comorbid condition categories: neoplastic comorbidity, cardiac comorbidity and endocrine comorbidity. The neoplastic comorbid condition category used the CCI scales for any tumour, leukaemia, lymphoma, and metastatic solid tumour. Participants with a myocardial infarction and/or congestive heart failure on the CCI were re-coded as having a cardiac comorbidity. Participants with diabetes and/or diabetes with end organ damage were re-coded as having an endocrine comorbidity.

Pressure injury risk was assessed with the Waterlow Risk Assessment Tool (WRAT), a tool that identifies pressure injury risk on 11 separate risk domains. Some domains are measured with mutually exclusive categories (i.e. age group) while others are additive and participants can score along more than one dimension of the domain (i.e. skin type and visual areas). Scores from the 11 domains are added to provide a total pressure injury risk figure which then places a participant into one of four risk groups. These are 'low risk' (<10), 'at risk' (10-14), 'high risk' (15-19) and 'very high risk' (20+). Clinical interventions are usually directed at patients scoring 10 or greater on the WRAT scale. While any person can develop pressure injuries, the low risk group represents participants not targeted for any specific pressure injury
interventions and, for the purpose of this study, this group will be referred to as the 'no risk' group.

Study equipment

The study mattress [ComfortPlus™] was placed on a non-electric standard hospital bed with metal base, and bedding was prepared so that the sheet was placed on the mattress without tension and there were no piles or folds in the area of the pressure sensor mat.

All IP measurements were taken using the Tekscan Advanced Clinical Seating Pressure Assessment System (Clinical™), v5.25. A pressure sensor mat (55x85cm) was placed on the mattress and connected to a notebook computer with dedicated software. Following procedures recommended by the supplier, each sensor mat was used for 50 measurements before being exchanged for a new mat to guard against creep. The pressure sensor mat was calibrated daily and prior to the first use of a new mat.

Data collection

Data collection took place in the hospital wards and units, with equipment being transported to each clinical area daily.

After gaining participant consent, demographic information, patient characteristics and WRAT data were gathered from a mixture of participant interviews and hospital records. All participants were assisted to transfer onto the study mattress and wear hospital pyjama pants over their underwear for each IP measurement cycle. A paper marker was placed on the sacrum to identify this site by row and column on the IP measurement computer. The marker was carefully removed after site identification and participants were instructed to remain supine with one pillow supporting their head at a measured 30° angle. Participants were requested to remain still and not to cross their legs during the data collection period. IP was measured continuously for 10 minutes as a movie, with a snapshot taken every minute. The colour image snapshots were converted to measures of pressure calculated as mmHg. For each snapshot, the peak or maximum reading at the sacrum site was entered into the database, making a total of 10 readings for each participant.

WRAT data collection procedures were standardized among the research team to provide consistency of scoring. Standardisation involved education sessions for research staff and multiple scoring of some participants to evaluate consistency of scores amongst research staff. Inter-rater reliability was calculated for the WRAT based on a sample of six participants who were examined independently by the three nurse research assistants during the course of the study. There was 100% agreement on sub-scale and total WRAT scores.

Data analysis

All data were managed using the SPSS V11.5 computer software package. Demographic and patient characteristics were summed and WRAT scores were calculated for each patient and grouped according to WRAT risk level. Blood pressure was measured and classified according to World Health Organization recommendations (low, normal or high).

Hospital wards were coded to represent either medical, surgical or obstetric wards. The reason for admission provided by patients was recoded into eight categories, seven of which represented reasons specific to different anatomical systems (i.e. musculo-skeletal, respiratory, cardiovascular) and the eighth was a generic 'other' category. Comorbidities were examined using the categories of neoplasm comorbidity, cardiac comorbidity and endocrine comorbidity. There were two options available for route of admission to hospital. These were emergency/ambulance admission and scheduled admission from home.

IP scores were examined for consistency. There were some individual IP readings of zero that were not included in further analysis because they represented suspect readings. There were also some IP readings that were not similar to other IP readings for individual participants. The decision was made to delete any IP readings that were greater or less than 5mmHg different from any other IP readings for a participant. As a result, four participants had nine IP measurements available, one participant had eight IP readings and another participant had seven IP readings available. The remaining 115 participants had all 10 IP readings.

IP means were calculated for each participant and for each of the WRAT categories. ANOVAs with planned comparisons were used to compare WRAT categories along scaled demographic variables and IP means. Planned comparisons involved comparing the WRAT no risk group to each of the other WRAT groups. Chi-square was used to compare sex distributions, and descriptive statistics were used for patient characteristics.

Results

To investigate whether participants differed along demographic variables according to level of pressure injury risk (WRAT risk group categories), mean age and WRAT scores were calculated for each group and ANOVAs conducted.
with planned comparisons calculated between the no risk WRAT group and each of the other groups. Significant group differences were found for age (F(3,117)=12.48, p<0.001) and BMI (F(3,117)=3.73, p=0.01). Sex frequencies were calculated for each risk group. Means, frequencies and the results of the planned comparisons for age and BMI are presented in Table 1.

The results of the planned comparisons indicated that the no risk WRAT group were significantly younger (F=22.55, p<0.001) and had a lower BMI (F=6.35, p=0.02) than the very high risk group. Other significant comparisons indicated that the no risk group were younger than both the at risk (F=15.54, p<0.001) and high risk (F=18.82, p<0.001) groups but did not significantly differ from these groups in terms of BMI. WRAT risk groups did not differ significantly in their sex balance. The overall BMI mean was 27.01 (SD=5.67).

**Patient clinical characteristics and WRAT risk categories**

To describe the breadth of clinical characteristics of participants, frequencies of clinical characteristics were examined according to WRAT risk group categories. The percentage of participants at no risk of pressure injuries was calculated for each clinical characteristic. The remainder of the participants with that specific characteristic were therefore categorized into one of the WRAT groups at greater risk of developing pressure injuries. Results are presented in Table 2 and describe the distribution across the four WRAT groups. Because of the small group sizes for many of the clinical characteristic groups, no inferential analyses were conducted. Therefore, generalizability of these results is limited.

Less than one third of participants in the high blood pressure group were in the no risk WRAT group, compared to approximately one half of the participants with normal blood pressure and three quarters of those with low blood pressure. Less than 30% of participants admitted to hospital for respiratory, cardiovascular and skin reasons were in the no risk WRAT group. This compares to 100% of the gynaecological participants and almost 70% of the gynaecological and urinary participants graded in the no risk WRAT group.

Only 14% of participants with cardiac comorbidities (n=4) and 15% of those with endocrine comorbidities (n=3) were classified in the no risk WRAT category. For participants with a neoplastic comorbidity, this figure was almost 40% (n=11).

Additionally, 13 participants had two comorbid conditions. Four of these had both a neoplastic and a cardiac comorbidity, eight had a cardiac and an endocrine comorbidity, and one had a neoplastic and an endocrine comorbidity. It is of note that all nine participants in the very high risk WRAT category presented with dual comorbidities.

**IP and WRAT risk categories**

IP means were calculated according to WRAT risk categories. Means are presented in Table 3.

Participants in the no risk group had an IP mean that was approximately 4.2mmHg lower than the at risk group and more than 7mmHg lower than the other two groups. Because group sample sizes were so variable, participants in the at risk, high risk, and very high risk WRAT categories were combined into a single group (risk). The IP mean for the risk group (n=46) was 32.4mmHg (SD=11.56). A two-tailed t-test was then conducted comparing the means for the risk and no risk groups. Levene’s test for equality of variance suggested that the two IP means did not have equal variance and the test was therefore conducted without assuming equal variance. The result indicated that the risk group had a significantly higher IP mean than the no risk group (t=3.13, df=116.63, p=0.002).

**Table 1. Demographic means and frequencies by WRAT risk group categories with planned comparisons.**

<table>
<thead>
<tr>
<th>WRAT risk group</th>
<th>No risk (n=56)</th>
<th>At risk (n=35)</th>
<th>High (n=22)</th>
<th>Very high (n=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>59.61</td>
<td>15.97</td>
<td>60.15</td>
<td>12.78</td>
</tr>
<tr>
<td>BMI</td>
<td>25.67</td>
<td>4.78</td>
<td>27.43</td>
<td>5.20</td>
</tr>
<tr>
<td>Sex (F/M) n=121</td>
<td>77/46</td>
<td>12/23</td>
<td>9/13</td>
<td>4/5</td>
</tr>
</tbody>
</table>

BMI calculated as weight divided by height
Significant planned comparisons with the "no risk" WRAT group:

* F=19.54, p<0.001
  a F=18.82, p<0.001
  b F=29.56, p<0.001
  c F=6.35, p<0.001

**PRIMARY INTENTION**
Discussion

This study presented data about the relationship between pressure injury risk, as measured by the WRAT, and IP measured at the sacrum for hospital patients lying on a standard hospital mattress. Whereas previous studies have investigated pressure injury risk and IP separately, the relationship between the two has not previously been evaluated. Previous research has measured IP on various support surfaces using healthy volunteers or mannequins. This study is therefore unique because participants represented a broad cross-section of adult inpatients across a wide range of ages and health conditions.

Participants who were not considered to be at risk of developing pressure injuries were found to have IP readings within the acceptable IP range. In contrast, those at risk of developing pressure injuries recorded average IP readings generally at the lower end of the compromised IP range based on the average capillary pressure found by Landis and colleagues (IP<32mmHg).

Table 2. Participant clinical characteristics by WRAT risk group categories.

<table>
<thead>
<tr>
<th>WRAT risk group characteristics</th>
<th>No risk (n=56)</th>
<th>At risk (n=39)</th>
<th>High (n=22)</th>
<th>Very High (n=0)</th>
<th>Total (n=121)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>7 (17%)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Normal</td>
<td>36 (69%)</td>
<td>21</td>
<td>14</td>
<td>7</td>
<td>60</td>
</tr>
<tr>
<td>High</td>
<td>13 (23%)</td>
<td>7</td>
<td>2</td>
<td>2</td>
<td>32</td>
</tr>
<tr>
<td>Admission reason</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>7 (32%)</td>
<td>7</td>
<td>5</td>
<td>2</td>
<td>22</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>12 (57%)</td>
<td>6</td>
<td>3</td>
<td>0</td>
<td>21</td>
</tr>
<tr>
<td>Genito-urinary</td>
<td>10 (57%)</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>5 (22%)</td>
<td>8</td>
<td>4</td>
<td>0</td>
<td>18</td>
</tr>
<tr>
<td>Gynaecological</td>
<td>11 (50%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>11</td>
</tr>
<tr>
<td>Respiratory</td>
<td>3 (22%)</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Skin</td>
<td>2 (9%)</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>Other</td>
<td>6 (25%)</td>
<td>5</td>
<td>4</td>
<td>3</td>
<td>18</td>
</tr>
<tr>
<td>Comorbidities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neoplasm</td>
<td>11 (53%)</td>
<td>11</td>
<td>7</td>
<td>0</td>
<td>20</td>
</tr>
<tr>
<td>Cardiac</td>
<td>4 (14%)</td>
<td>14</td>
<td>5</td>
<td>5</td>
<td>20</td>
</tr>
<tr>
<td>Endocrine</td>
<td>3 (15%)</td>
<td>7</td>
<td>8</td>
<td>0</td>
<td>20</td>
</tr>
<tr>
<td>Admission route</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ambulance/emergency</td>
<td>26 (69%)</td>
<td>23</td>
<td>15</td>
<td>6</td>
<td>72</td>
</tr>
<tr>
<td>Home</td>
<td>27 (55%)</td>
<td>12</td>
<td>7</td>
<td>3</td>
<td>48</td>
</tr>
</tbody>
</table>

* Percentages represent the percent of participants in each patient characteristic category in the 'no risk' group.
A Thirteen participants had more than one comorbidity.

Table 3. Interface pressure means by WRAT risk categories.

<table>
<thead>
<tr>
<th>Risk category</th>
<th>n</th>
<th>M</th>
<th>SD</th>
<th>Range</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>No risk</td>
<td>55</td>
<td>26.54</td>
<td>8.45</td>
<td>9.3 - 47.2</td>
<td>23.36 - 28.90</td>
</tr>
<tr>
<td>At risk</td>
<td>35</td>
<td>30.30</td>
<td>11.17</td>
<td>14.5 - 60.8</td>
<td>25.86 - 34.84</td>
</tr>
<tr>
<td>High</td>
<td>22</td>
<td>34.42</td>
<td>13.76</td>
<td>16.3 - 62.6</td>
<td>28.33 - 40.51</td>
</tr>
<tr>
<td>Very high</td>
<td>9</td>
<td>33.97</td>
<td>10.54</td>
<td>24.1 - 55.4</td>
<td>25.86 - 42.07</td>
</tr>
</tbody>
</table>

PRIMARY INTENTION 147 VOL. 14 NO. 4 NOVEMBER 2036
From a clinical perspective, this finding adds weight to the claim that the WRAT like many other risk assessment tools, over-estimates the risk of pressure injury. Over-estimation of risk is likely to lead to complacency in clinical practice. As Gebhard argues, clinicians realize that few patients identified as being at risk actually go on to develop a pressure injury. This results in complacency when there are many competing claims on clinicians’ time and intervention can be delayed, possibly resulting in greater prevalence. More work is therefore needed to reduce the over-estimation of risk.

In this study, although there were significant differences in IP readings depending on WRAT risk category, the statistical comparisons between each WRAT category were not possible given the variety of WRAT group sizes. Future research could further investigate whether there are any other significant differences in IP across the WRAT risk categories. It is also unclear how IP varies across WRAT risk categories for people with specific health concerns and across specific ages. For example, do people in their 60s with heart conditions have different IP readings if they are in different WRAT categories based on other demographic or clinical characteristics.

There were patterns of clinical characteristics that need to be tested in future studies. For example, a greater percentage of participants with high blood pressure were in one of the groups at risk of developing a pressure injury compared to those with low or normal blood pressure. Participants in medical wards were more likely to be classified as at risk of pressure injury than participants in surgical or obetric wards. Not surprisingly, all participants admitted for obstetric reasons were classified in the no risk WRAT category. This contrasts with the four admission reasons with the smallest number of participants respiratory, cardiovascular, skin and other. Participants admitted to hospital for these reasons were less likely to be classified in the no risk WRAT group than one of the three at risk groups. However, sample size in these groups restricts generalisability to other populations.

Admission route to hospital results suggest that patients admitted from home were more likely to be in the no risk WRAT group than those admitted as accident and emergency patients. There is some indication in the literature that these transferred from other hospitals are more likely to be at risk of pressure injury. One possible explanation for this increased risk is that inter-hospital transfer may be a surrogate for increased severity of illness as patients are transferred from smaller hospitals to larger tertiary centres. Increased pressure injury risk may also apply for patients requiring emergency admission to hospital.

It is of note that the BMI mean values for the no risk, at risk and high risk groups were all in the clinically overweight category, and the very high risk group mean was in the obese category. The relationships between BMI, IP and pressure injury risk are not straightforward. For instance, the WRAT scores more risk to people who are underweight than people who are obese. In contrast, a recent study of hospitalised patients found that being 95kg or heavier was more strongly associated with the development of pressure ulcers than being 50kg or lighter.

There are some limitations to the study. We have used IP as a surrogate measure for changes in skin viability at the sacrum. It is one of the three most important extrinsic parameters to examine when evaluating intervention mattresses used to prevent pressure injury (the other two being bloodflow and microclimate). While it cannot be assumed that IP and bloodflow correspond exactly, IP is much more easily measured non-invasively. There is an established clinical literature on the use of IP for development of devices for individual orthopaedic and paraplegic patients, so the strengths and limitations of the technology are well understood.

Conclusion
This research has made important contributions to the field on two levels. First, the findings are meaningful for clinicians in bringing new knowledge to our assumptions about risk factors, risk assessment and pressure injury. Second, this study has established a methodological breakthrough in moving this approach to pressure injury research out of the laboratory into the clinical environment and therefore producing more clinically relevant results. As such, we have instituted a new approach to researching pressure injury risk assessment strategies.

Acknowledgements
The study was funded through an Australian Grant (GRA 02/697) in partnership with Australian Healthcare Industries (formerly Bensor Medical). Thanks to staff of the Canberra Hospital and Calvary Health Care ACT for assistance in this research project. Thanks to Ms Donna Mowbray for her valuable input in the early stages of the project. We would also like to acknowledge the dedication of the research nurses who collected the data - Mel Symons, Cecelia Waelbling, Stephen Bederick, Vicki Brown and Sam Weight.
Disclaimer
Australian Healthcare Industries, whilst providing support to the project, has not influenced the design or analysis of the study in any way.

References
APPENDIX C - DUNK & TREVIT ARTICLE

How the AWMA Clinical Practice Guidelines for Prediction & Prevention of Pressure Ulcers are being used across the ACT

Ann Marie Dunk • Corinne Trevit

Primary Intentions 2005 3/33: 121.

In 2002, the Canberra Hospital and University of Canberra Research Centre for Nursing Practice embarked on a 2 year quality project. One part of this project was aimed to develop and establish consistent and sustainable processes for pressure injury prevention management in the ACT. This project was funded by Quality and Safety Forum, ACT Health.

Part of the project-specific objectives was to establish and implement a clinical practice improvement plan (CPIP) within the public health system i.e. the Canberra Hospital, Calvary Health Care ACT and Community Health. The CPIP was based on the Australian Wound Management Association’s (AWMA) clinical practice guidelines for pressure ulcers and formulated a strategy for implementation of the CPIP.

This project comprised of four phases. The first and fourth phases were data collection phases (baseline and post-intervention respectively). The second phase was the development of CPIP and other strategies to reduce pressure injury prevalence and the third phase was the implementation of the strategies.

This third phase allowed for the implementation, primarily using tiered education and clinical support strategies.

The fourth phase facilitated comparative analysis of the implementation of the CPIP across both acute and community health care settings in the ACT.

The CPIP based on the AWMA guidelines provided consistent evidence-based recommendations for best practice in predicting and preventing pressure ulcers. This provided a strategy to assist clinicians to identify patients at risk of pressure injury and make decisions on prevention strategies in pressure management.

This multifaceted project across the ACT has heightened awareness of the importance of pressure injury prevention and has provided ACT healthcare clinicians and health care providers with strategic directions in the prediction and prevention of pressure ulcers.

Since the completion of this project in 2004, the ACT Quality and Safety Forum appointed a Pressure Injury Prevention Reference Group, ACT Health. This reference group consists of nurses, clinicians, educators and researchers from all health areas within the ACT, including allied health, aged care representatives, medical and general practitioners, products and supply representatives and consumers. The ACT Chief Nurse, Jennifer Beaulder, chairs this group.

As part of a strategy to implement research into clinical current best practice, this reference group will develop an evidence-based framework for preventing pressure injury. The framework will promote consistency across the Territory. It will also provide leadership, promote evidence-based practice, support research and education regarding prevention of pressure injury and quality indicators, which are relevant monitors.

Ann Marie Dunk
Corinne Trevitt
ACT Wound Management Association Inc.
April 2004
APPENDIX D- POSTER DESCRIBING THE MIPPI STUDY

This ward is a study site for a study to prevent pressure injuries (bed sores)

The Research Centre for Nursing Practice is looking for patients to participate in a study to prevent pressure injuries by investigating the efficacy of pressure relieving mattresses (MIPPI Phase III)

What is involved?

Research staff will measure:
- The pressure that the buttock or hip exerts on the mattress (interface pressure) and;
- The skin blood flow in these areas with laser Doppler sensor
- Measurements are taken while patients lie on the sensors.
- Taking part will take approximately 1 1/2 hour of your time.

MIPPI Phase III is approved the ACT Health Human Research Ethics Committee.

Interested and want to know more? Ask the nurse looking after you to give you a MIPPI study patient information sheet or ring Marlene Eggert or Ann Marie Dunk on 62442396
APPENDIX E - INCLUSION AND EXCLUSION CRITERIA

Inclusion criteria

- A patient 16 years and older admitted as inpatient to The Canberra Hospital or Calvary Health Care ACT and who occupies a hospital bed.
- A patient with no pressure injury or with a pressure injury on the sacrum that does not exceed Category 1 in accordance with the AWMA (2001) guidelines.

Exclusion criteria

- A patient who did not consent to participate in the MIPPI project.
- Patients who had previously participated in MIPPI project.
- A patient identified by Clinical Nurse Consultant as high dependency/requiring constant observation.
- Patients who were expected to be discharge within the next 3 hours.
- A patient who is unable to tolerate or medically unfit to lie flat with two pillows for the length of time required for data collection process.
- A patient who was extremely emaciated.
- Patients who was extremely restless.
- Patient unable to tolerate or medically unfit to lie at 30 degree angle head elevation with two pillows for length of time required for data collection.
- A patient who was placed on the surgery emergency list for the day and if their theatre time was not knownPatients who had pre-existing pressure injury Category 2 or above on the sacrum.
• A patient who was in post-surgery or post-investigation period and not allowed to be moved.

• A patient with an amputation large limb or who had plaster or splint reaching above the knee on the leg.

• Patients who were medically unsafe to move as identified by Clinical Nurse Consultant, ie patients moved only by log rolls, chest drains, incubated or raised intracranial pressure.

• Patients whose treatment would be compromised if moved ie intravenous chemotherapy in progress, multiple drains and infusions.

• A patient who was too unwell to be moved ie nausea, frequently vomiting, diarrhoea or pain.

• A patient who was a woman pregnant 12 weeks and more.

• A patient who was a woman pregnant with twins or triplets

• A patient that required medication isolation ie MRSA, VRE, neutropenia, radioactive isotopes as identified in ward care procedure.

• A patient that was cognitively impaired.

• Patients that required a low sensory stimulant environment

• Patients considered by the Clinical Nurse Consultant or senior nurse in charge of the shift to be unsafe to move or be moved onto the study mattress.
APPENDIX F - WATERLOW RISK ASSESSMENT TOOL

Circle applicable scores and add circled scores for total score. Several scores per category may be selected.

<table>
<thead>
<tr>
<th>Sex &amp; Age</th>
<th>Skin Type &amp; Visual Areas</th>
<th>Continence</th>
<th>Tissue Malnutrition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>Healthy</td>
<td>0</td>
<td>Eg. Terminal cachexia 8</td>
</tr>
<tr>
<td></td>
<td>Tissue paper</td>
<td>1</td>
<td>Cardiac failure 5</td>
</tr>
<tr>
<td>Female</td>
<td>Dry</td>
<td>1</td>
<td>Periph vascular disease 5</td>
</tr>
<tr>
<td>14--49</td>
<td>Oedematous</td>
<td>1</td>
<td>Anaemia 2</td>
</tr>
<tr>
<td>50-64</td>
<td>Claymy</td>
<td>1</td>
<td>Smoking 1</td>
</tr>
<tr>
<td>65-74</td>
<td>Discoloured</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>75-80</td>
<td>Broken</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>80+</td>
<td></td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mobility</th>
<th>Neurological Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fully</td>
<td>Eg Diabetes, MS, CVA</td>
</tr>
<tr>
<td></td>
<td>Motor / sensory paraplegia</td>
</tr>
<tr>
<td>Restless / fidgety</td>
<td>moderate or</td>
</tr>
<tr>
<td>Apathetic</td>
<td>4</td>
</tr>
<tr>
<td>Restricted</td>
<td>moderate to severe or</td>
</tr>
<tr>
<td>Inert / traction</td>
<td>severe</td>
</tr>
<tr>
<td>Chair-bound</td>
<td>(circle one score only)</td>
</tr>
</tbody>
</table>

Adapted from Judy Waterlow Risk Assessment Tool with kind permission. www.judyswaterlow.com

NAME: __________________
APPENDIX G- MATTRESS INFORMATION/

TECHNICAL SPECIFICATIONS 'COMFORT PLUS'

COMFORT PLUS MATTRESS

Description:
The 'Comfort Plus Mattress' was developed by Bosshard Medical and is made of high quality Australian foam. The convoluted foam ('egg shell') is pressure reducing and low shearing mattress.

Recommendations:
Bosshard Medical recommendations are that this pressure care mattress can be used for the low risk patients.

Specifications:

Foam:
- Foam thickness is 135mm
- Top layer: 75mm
  - Density 35Kg/m3
  - Tear resistance 250N/m minimum
  - Tensile strength 60 KPa minimum
  - Hardness, L.F.D 115N (Newton) at 40%.
- Base: 60mm
  - Density 35 Kg/m3
  - Tear resistance 300N/m minimum
  - Tensile strength 80 KPa minimum
  - Hardness, L.F.D 200N (Newton) at 40%.
- Treated with Ultrafresh. Ultrafresh is antifungal and antibacterial compound.

Covers:
- Nylon, fabric with flame retardant polyurethane coating
- High strength durable fabric, soft, very flexible.
- Impermeable
- Smooth surface produces very low friction force
- Fibre Composition 100% Nylon
- Hydrostatic Waterhead 250 Kpa
- Breathability <200g/m2/24hrs
- FR properties AS 3744.1 non-ignition, AS 2281
APPENDIX H - DATA COLLECTION TOOL MIPPI 3

MIPPI DATA SHEET

Study No: ____________
Randomisation No: ____________
Date: ________________

Facility: (1) TCH □ (2) CALVARY □ (3) TBA □
Location/Ward: ________________
Data Collector: ________________

PARTICIPANT DATA

UR Number: ________________
D.O.B: ___________(dd)/_________(mm)/__________(yyyy)
Admission Date: ___________(dd)/_________(mm)/__________(yyyy)
Reason for Admission: ________________
Withdraw Reason: ________________

CLINICAL DETAILS

BP: ___________ mmHg
Height: ___________ cm  Weight: ___________ kg  BMI: ___________%
Chart/notes □  Chart/notes □
Estimation □  Estimation □
Ward scales □  Ward scales □
Patient □  Patient □
Hb: ___________ g/L  O2 Therapy □
Sample date: ________________

PATIENT DRESS

Dress: No. of pillows:
Underpants □  1 pillow □
Gown □  2 pillows □
Pyjama/nightie □
Other □

ADMISSION ROUTE:

From Home □
A&E □
Ambulance Transport □
Emergency Theatre □
### WATERLOW RISK ASSESSMENT TOOL

**Sex:**
- Male: 1
- Female: 2

**Age:**
- 1: 14-49
- 2: 50-64
- 3: 65-74
- 4: 75-89
- 5: 90+

**Medication Cytotoxics:**
- High dose sterols / Anti infl am
- None

**Catheterisation:**
- Complete/ Catheterised: 0
- Occasional incontinence: 1
- Catheter / Incontinent: 2
- Double incontinence: 3

**Appetite:**
- Average: 0
- Poor: 1
- NG Tube / Flatus only: 2
- NSM / Anaesthetic: 3

**Build/Weight for Height:**
- Average: 1
- Above Average: 2
- Below Average: 3

**Major Surgery:**
- Orthopaedic: 5
- Below waist: 6
- On table > 2hr: 7
- None: 8

**Neurological Deficit:**
- (eg. Diabetes, MS, CVA)
- Malignancy: 1
- Paraplegia: 2
- Moderate: 4
- Severe: 6
- None: 8

### WATERLOW RISK SCORE
- <10 Low Risk
- 10 - 14 At Risk
- 15 - 19 High Risk
- 20+ Very High Risk

### MEDICATIONS

**Cytotoxics**
- Name: __________________________  Dose: ____________  Last Taken: __________________________
- Name: __________________________  Dose: ____________  Last Taken: __________________________
- Name: __________________________  Dose: ____________  Last Taken: __________________________

**Steroids**
- Name: __________________________  Dose: ____________  Last Taken: __________________________
- Name: __________________________  Dose: ____________  Last Taken: __________________________
- Name: __________________________  Dose: ____________  Last Taken: __________________________

**Anti-Inflammatory**
- Name: __________________________  Dose: ____________  Last Taken: __________________________
- Name: __________________________  Dose: ____________  Last Taken: __________________________
- Name: __________________________  Dose: ____________  Last Taken: __________________________

**Nutrition:**
- Parenteral nutrition: [ ]
- Liquid supplements: [ ]

**Analgesic:** (taken in the last four hours only)
- Name: __________________________  Dose: ____________  Last Taken: __________________________
- Name: __________________________  Dose: ____________  Last Taken: __________________________
- Name: __________________________  Dose: ____________  Last Taken: __________________________

---

This document is confidential. If found, please return to THE RESEARCH CENTRE FOR NURSING PRACTICE, OR ANN MARIE DUNK OR MARLENE EGGERT, PO BOX 11, WODUN, ACT 2606.

MPPI PHASE II DATA COLLECTION FORM. THIS VERSION 21.01.05
<table>
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**SUPPORT SURFACE INFORMATION**

UR NO:______________

Recorded by:__________________

**READING ONE**

1. **MATTRESS**
   - Comfort Plus [ ]
   - Foam Air [ ]
   - Ruby [ ]
   - (Standard Hospital)
   - (Constant Low Pressure)
   - (Alternating Pressure)

2. **LASER DOPPLER**
   - SACRUM [ ]

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<tr>
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3. **INTERFACE PRESSURE READINGS**
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   - Equilibrium loaded [ ]

Sensor Mat no:__________________

Sensor Mat use:__________________

File Name:__________________

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READING TWO

1. MATTRESS

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<td>(Alternating Pressure)</td>
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2. LASER DOPPLER

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3. INTERFACE PRESSURE READINGS

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Stamp Notes

(Did you remember the finish time?)

Ann Marie Dunk
FOLLOW-UP SURVEY UP TO 7 DAYS POST
DATA COLLECTION

UR NO:________________________

Survey
To be completed within 7 days of data collection.
Survey Date: ___________  
Visual Patient records

Results

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</table>

Study No: __________________
Date: ________________

THIS DOCUMENT IS CONFIDENTIAL. IF FOUND, PLEASE RETURN TO THE RESEARCH CENTRE FOR NURSING PRACTICE, C/- ANN MARIE DUNK or MARLENE FOGERTY, PO BOX 11, WODEN, ACT 2606.

MIFPI PHASE 11 DATA COLLECTION FORM. THIS VERSION 21.01.98

Ann Marie Dunk

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Figure I-1. Tekscan ClinSeat™ 2D Contours View with corresponding pixelated view
Figure I-2. Tekscan ClinSeat™ 3Dimensional Wireframe View

Figure I-3. Tekscan Clinseat™ Peak Interface Pressure vs. Time plot with corresponding 2D Contours view in colour-coded quadrants
APPENDIX J- ETHICS LETTER OF APPROVAL -

DEAKIN UNIVERSITY HUMAN RESEARCH ETHICS COMMITTEE

Research Services
Office of the Deputy Vice-Chancellor (Research) (Melbourne Campus)

MEMORANDUM

TO: Dr Judy Currey
    School of Nursing, Burwood

FROM: Secretary, Deakin University Human Research Ethics Committee (DU-HREC)

DATE: 19 February 2008

SUBJECT: Project EC 30-2008  
          (Please quote this project number in future communication.)
          Mapping and intervention for the prevention of pressure injury (MIPPI)

Interim approval for this project was ratified at the DU-HREC meeting held on 18 February 2008.

Approval has been given for Ann Marie Dunk, under the supervision of Dr Judy Currey, School of Nursing, to undertake this project for a period of three years from 7 February 2008.

The approval given by the Deakin University Human Research Ethics Committee is given only for the project and for the period as stated in the approval. It is your responsibility to contact the Executive Officer immediately should any of the following occur:

• Serious or unexpected adverse effects on the participants
• Any proposed changes in the protocol, including extensions of time.
• Any events which might affect the continuing ethical acceptability of the project.
• The project is discontinued before the expected date of completion.
• Modifications are requested by other HREC's.

In addition you will be required to report on the progress of your project at least once every year and at the conclusion of the project. Failure to report as required will result in suspension of your approval to proceed with the project.

DU-HREC may need to audit this project as part of the requirements for monitoring set out in the National Statement on Ethical Conduct in Research Involving Humans (1999).

Vicky Bates
On behalf of DU-HREC
(03) 9251 7123

Signature Redacted by Library
Ms Marlene Eggert  
Research Centre for Nursing Practice  
The Canberra Hospital

Dear Ms Eggert

The ACT Health and Community Care Human Research Ethics Committee considered the proposed study 'Mapping and Intervention for Prevention of Pressure Injury Phase III' at the meeting held on 16 November 2004. Ethics Committee Submission No ETH.10/04.534 refers.

I confirm that Professor Arbon did not participate while the members considered the study.

The Committee approved the study including the Patient Information Sheet (Version 1: 2 November 2004), Consent Form (Version 1: 2 November 2004) with an amendment to the poster.

The Committee agreed that the 'Poster heading should include the word 'involved' after the word 'is' and the wording 'site for a study' should be deleted and second last paragraph, the word 'by' should be included after the word 'approved'.

Please forward an amended poster to the Ethics Committee.

I attach for your records an Outcome of Consideration of Protocol form.

You may recall that the ACT Health and Community Care Guidelines for Submission of Application require you to complete payment of the levy when approved by the Ethics Committee.

Please forward $27.50 levy fee to the Secretariat, ACT Health and Community Care Human Research Ethics Committee, GPO Box 825, Canberra ACT 2601 as soon as possible. An invoice is attached for your attention.

Yours sincerely,

Elizabeth Grant AM  
Chair  
Ethics Committee  
16 November 2004

[Signature Redacted by Library]
Thursday 9 March 2005

Ms Marlene Eggert  
MIPPI Study Manager  
Research Center for Nursing Practice  
The Canberra Hospital  
PO Box 11  
Woden ACT 2606

Dear Ms Eggert

On the recommendation of the Human Research and Ethics Committee, which met on Wednesday 16 February 2005, the Executive Management Group has approved the second range of amendments to your Research Proposal "Mapping and Intervention for Prevention Injury (MIPPI)"

The committee would appreciate a regular update and a report on completion of the study. Should you wish to publish your project and Calvary Health Care is in any way identified, the Committee wishes to approve the paper prior to publication.

Yours Sincerely

Dr Elizabeth O’Leary  
Chairperson  
Human Research & Ethics Committee  
Calvary Health Care ACT

Signature Redacted by Library

Ann Marie Dunk  
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MIPPI study patient information sheet

The Mapping and Intervention for Prevention of Pressure Injury (MIPPI) study is a two year research project. It investigates the efficacy of pressure relieving mattresses in the prevention of pressure injuries (pressure sores) by measuring interface pressure and skin blood flow. The study is being conducted by the Research Centre for Nursing Practice at The Canberra Hospital & University of Canberra.

The study is conducted in three phases:
- MIPPI phase I is a pilot study on healthy volunteers that is now completed.
- MIPPI phase II is a pilot study on patients.
- MIPPI phase III - the main trial. You are invited to participate in this phase.

MIPPI phase III is a multicentre trial which is conducted in hospitals in the ACT and interstate.

What is involved if I participate in MIPPI phase III?

The MIPPI research nurse will take your height, weight, body temperature and blood pressure. The research nurse will also ask you about any medical conditions you may have apart from the one that brought you to hospital and about the medications you take. Your hospital notes may also be looked at by the MIPPI research nurse to gather this information.

The MIPPI research nurse will assess your risk of developing a pressure injury. If your risk is low, you fall into the observation group.
If you fall into the “At risk”, “High risk” or “Very high risk” category, you fall into the intervention group.

If you fall into the observation group you will be assigned to the study of a standard hospital mattress. For this study your interface pressure and skin blood flow will be measured on the standard hospital mattress twice.

If you fall into the intervention group your interface pressure and skin blood flow will be taken once on the standard hospital mattress and once again on either a constant low pressure mattress or an alternating pressure mattress.

A constant low pressure mattress is a specialist mattress designed to prevent pressure injuries by moulding itself around the body shape.
An alternating pressure mattress is again a special mattress which distributes pressure with air cells which blow up and deflate.

The MIPPI study nurse will randomly allocate you to one of the special mattresses.
Skin blood flow measurement

The buttoc will be the body area studied as a measurement site. You will need to lie flat and with your head on one or two pillows while this measurement is taken. The skin blood flow on your measurement site will be taken with a laser Doppler capillary blood flow meter. A very small, flat sensor probe, about the size of your fingernail will be fixed to your skin with tape. Readings will be taken with you not resting on the sensor for 5 minutes and then resting on the sensor for 10 minutes. You will need to lie still while the skin blood flow is being measured. If you are allergic to tape you should not participate in this study.

Interface pressure measurements

Interface pressure is the pressure that builds up between a person’s body and the mattress they rest on. In general, the harder the mattress, the higher the interface pressure.

To measure the interface pressure on the trial mattresses we put a soft sensor mat on the trial mattress and ask you to lie on it. To explore how to trace the buttoc (sacrum) on the computer monitor, a folded paper towel will be placed with tape over the lowest vertebra of your back as a marker. This will be removed after a minute or so. The interface pressure measurement takes 10 minutes. We ask you to lie still while this measurement is taken.

Is there any possibility that I will be harmed by my participation?

The planned investigations are not expected to harm you. The trial mattresses are being used for their intended purpose in this study. The pressure mapping equipment consists of a soft sensor mat (53cm x 49cm) in a soft material, cleanable cover. There is no known danger associated with being placed on the interface pressure sensor mat. These mats are commonly used to measure the pressure under the buttocks of people who are wheelchair bound.

The laser beam used for measuring the skin blood flow is harmless if applied to body tissue, even if for a long time. However, a laser beam aimed at the eye can cause damage to the retina at the back of your eye. The laser sensor is safe to view from a distance of more than 20 cm. As the laser sensor will be applied to the lower half of your body it is well away from your eyes.

If you are unable to move onto the trial mattress on your own, then the MIPPI research nurses and a warden, if needed, will help you.

Your privacy during your participation in the trial will be protected as the measurements will be either taken in your hospital room or a treatment room in the ward.
If you want more information about this study, please contact the study investigators Ms. Marlene Eggert or Ms. Ann Marie Dunk on ph 6244 2396 at the Research Centre for Nursing Practice. The chief investigator, Dr. Anne Gardner, can be contacted at the Cabrini-Deakin Centre for Nursing Research in Melbourne on ph (03) 9508 1905. You can also ask the nurse who looks after you on the ward to contact the study investigators on ext. 2396.

Should you have any problems or queries about the way in which this study was conducted, and you do not feel comfortable contacting the research staff, you can contact the ACT Department of Health Ethics Committee Secretary on Second Floor, North Building, London Circuit, Canberra City, ACT 2601 or on phone number 02-62050846.

Version Two, 5 January 2005
Consent Form to Participate in a Research Project

I, ____________________________
(name of participant)

being the ____________________________ of ____________________________
(state relationship eg son/wife/parent) (name of patient)

of ____________________________
(street) (suburb/town) (state & postcode)

have been asked to consent to [my relative/child’s] participation in a research project entitled:

Mapping and Intervention for the Prevention of Pressure Injury (MIPPI) phase II

In relation to this project I have read the Patient Information Sheet and have been informed of the following points:

1. Approval has been given by the ACT Department of Health Ethics Committee.

2. The aims of the project are firstly to test the data collection processes for MIPPI in the clinical setting and secondly to check the suitability of the study mattresses in the ward environment.

3. The results obtained from the study may or may not be of direct benefit to my medical management.

4. The research nurses will be determining my pressure injury risk with a Waterlow risk assessment tool. If I fall into the low risk category for pressure injury, I will have two sets of measurements on a standard hospital mattress taken. If I am not well enough to move onto the study mattress by myself, then the nurses will help me.

Each set of measurements is made up with an interface pressure measurement and skin blood flow measurement. Interface pressure is the pressure which builds up between the body and the study mattress. It is measured with a sensor mat. The skin blood flow is measured with a Laser Doppler device which has a sensor ½ centimetre wide and is attached to the skin with tape.

The body site where the measurements are taken are either under my buttock or under my hip. I can choose which position is most comfortable for me. I have to lie flat on one pillow while the measurements will be taken. The skin blood flow will first be taken while I lie in my side for 5 minutes and again while I lie on my back or hip for 10 minutes. After that the interface pressure will be measured with a sensor mat that is put under my buttock or hip. This

The Canberra Hospital & University of Canberra Research Centre for Nursing Practice

Ann Marie Dunk
measurement will take 10 minutes. Having the two sets of measurements taken will take 50 minutes to 1 hour. I will be asked to wear nightwear while the measurements are taken.

5. If I fall into the "At risk", "High risk" or "Very high risk" category the first set of measurements that will be taken is the same as described above. The difference is that the second set will be taken on either a constant low pressure mattress or an alternating pressure mattress. The research nurse will tell me which mattress is going to be used. If I cannot move onto the study mattress by myself, the nurses will help me.

6. These are some possible adverse effects or risks related to this project which include:
   - A small risk of a skin allergic reaction to the tape used to fix the laser Doppler sensor to the tape.

7. My involvement in this project may be terminated if any of the following circumstances develop:
   - I feel too breathless to continue lying flat with my head on one pillow
   - Lying still in one position gets too painful for me
   - If I feel too uncomfortable to continue with my participation in the study

8. Should I develop a problem which I suspect may have resulted from my involvement in this project, I am aware that I may contact Dr. Anne Gardner, Assistant Director of Nursing (Research), Research Centre for Nursing Practice, The Canberra Hospital & University of Canberra, ph 6244 2396 or Marlene Eggert, MIPPI Study Manager on ph 6244 2396.

9. Should I have any problems or queries about the way in which the study was conducted, and I do not feel comfortable contacting the research staff, I am aware that I may contact the ACT Department of Health Ethics Committee Secretary on Second Floor, North Building, London Circuit, Canberra City, ACT 2601 or on phone number 02-62050846.

10. I can refuse to take part in this project or withdraw from it at any time without affecting my medical care.

11. Participation in this project will not result in any extra medical and hospital costs to me.

12. I understand that the results of the research will be made accessible and that my involvement and my identity will not be revealed.

13. In giving my consent, I acknowledge that the Research Centre for Nursing Practice staff directly involved in the study, may examine my medical records only as they relate to this project.

14. I have read the MIPPI phase II study patient information sheet.

15. Compensation for any injury or illness suffered as a result of participation in this study is covered through the hospital insurance.
After considering all these points, I accept the invitation to participate in this project.

I also state that I have/have not participated in any other research project in the past 3 months. If I have, the details are as follows:

Date: ____________ Witness: ____________________________ (Please print name)

Signature: ____________________________ (of participant/volunteer) ____________ (of witness)

Investigator's Signature: ____________________________

Page 3 of 3, Version 1 13/07/04
APPENDIX O - CONSENT MIPPI 3 (CHC ACT)
Consent Form to Participate in a Research Project

I, ____________________________
(name of participant)

of ____________________________
(street) (suburb/town) (state) (postcode)

have been asked to consent to my participation in a research project entitled:

**Mapping and Intervention for Prevention of Pressure Injury**

In relation to this project I have read the Patient Information Sheet and have been informed of the following points:

1. Approval has been given by the Medico Moral, Human Research and Ethics Committee (MMHREC).

2. This project has 3 aims:
   - To measure the pressure between a person and a mattress (interface pressure) and their skin blood flow and to measure the relationship between the two.
   - To measure the relationship of interface pressure and skin blood flow to a pressure injury risk assessment score (Waterlow score)
   - To compare the efficacy of pressure relieving mattresses

3. The results obtained by the study may or may not be of direct benefit to my medical management.

4. The research nurses will be determining my pressure injury risk with a Waterlow risk assessment tool. If I fall into the low risk category for pressure injury, I will have two sets of measurements on a standard hospital mattress taken. If I am not well enough to move onto the study mattress by myself, then the nurses will help me. Each set of measurements is made up with an interface pressure measurement and skin blood flow measurement. Interface pressure is the pressure which builds up between the body and the study mattress. It is measured with a sensor mat. The skin blood flow is measured with a Laser Doppler device. This has a sensor ½ centimetre wide and is attached to the skin with tape.

The body site where the measurements are taken is the buttock area. I have to lie flat with my head on one or two pillows while the measurements will be taken. The skin blood flow will first be taken while I lie in my side for 5 minutes and again while I lie on my back for 10 minutes. After that the interface pressure will be measured with a sensor mat that is put under my buttock. This measurement will take 10 minutes. Having the two sets of measurements taken will take 1 ½ hours.

Ann Marie Dunk
I will be asked to wear nightwear while the measurements are taken.

If I fall into the “At risk”, “High risk” or “Very high risk” category the first set of measurements that will be taken is the same as described above. The difference is that the second set will be taken on either a constant low pressure mattress or an alternating pressure mattress. The research nurse will allocate by random the mattress which is going to be used. If I cannot move onto the study mattress by myself, the nurses will help me.

5. These are some possible adverse effects or risks related to this project which include:
   • A small risk of a skin allergic reaction to the tape used to fix the laser Doppler sensor to the tape.

6. My involvement in this project may be terminated if any of the following circumstances develop:
   • I feel too breathless to continue lying with my head elevated on one or two pillows
   • Lying still in one position gets too painful for me
   • If I feel too uncomfortable to continue with my participation in the study

7. Should I develop a problem which I suspect may have resulted from my involvement in this project, I am aware that I may contact the Chief Investigator Dr. Anne Gardner, Associate Professor, Cabrini-Deakin Centre for Nursing Research, Cabrini Health Malvern, 183 Wattletree Road, Malvern Vic, ph (03) 9508 1905 or Marlene Eggert and Ann Marie Dunk, MIPPI Study Managers, Research center for Nursing Practice (The Canberra Hospital & University of Canberra) on ph (02) 6244 2396.

8. Should I have any problems or queries about the way in which the study was conducted, and I do not feel comfortable contacting the research staff, I am aware that I may contact Raeleigh Mooney, secretariat of the MMHREC on (02) 6201 6104.

9. I can refuse to take part in this project or withdraw from it at any time without affecting my medical care.

10. Participation in the project will not result in any extra medical and hospital costs to me.

11. I understand that the results of the research will be made accessible and that my involvement and my identity will not be revealed.

12. In giving my consent, I acknowledge that the relevant Chief Investigator and Co-Investigators directly involved in the study may examine my medical records only as they relate to their project.

13. Compensation for any injury or illness suffered as a result of participation in this study is covered through the hospital insurance.
After considering all of these points, I accept the invitation to participate in this project.

I also state that I have/have not participated in any other research project in the past three (3) months. If I have the details are as follows:

Date: __________  Witness: ____________________________  (Please print name)

Signature: ____________________________  
(of participant/volunteer)  (of witness)

Investigator’s Signature: _____________________________________

I agree that the data collected for this study may be used to answer study questions related to the MIPPI study.

Date: __________  Witness: ____________________________  (Please print name)

Signature: ____________________________  
(of participant/volunteer)  (of witness)

Investigator’s Signature: _____________________________________

Version 2, 5 January 2005
APPENDIX P - DETAILED BREAKDOWN NOTES FOR
WATERLOW RISK SCORES

Table 3 presented a breakdown of the Waterlow scores assigned in the
assessment of risk for the patients in the study sample. Explanatory notes from Table 3
are presented below.

\(^a\) The Waterlow Risk Assessment Tool includes ages down to 14. As noted previously no patient
under 16 was enrolled into the study.

\(^b\) The choices in this category are not mutually exclusive and the figures therefore include patients who
were scored against multiple skin types/visual selections (eg. dry and discoloured, tissue paper and dry
and broken skin, etc). The breakdown of this multiple scoring is as follows:
- Of the ten patients recorded as having tissue paper skin, seven were also recorded in at
  least one other category
- Of the 57 patients recorded as having dry skin, 22 were also recorded in at least one other
category
- Of the 21 patients recorded as having oedematous skin, 16 were also recorded in at least
  one other category
- Of the 19 patients recorded as having discoloured skin, 17 were also recorded in at least
  one other category
- Of the 11 patients recorded as having broken skin, 10 were also recorded in at least one
  other category

\(^c\) The choices in this category are not mutually exclusive and the figures therefore include patients who
were scored against multiple tissue malnutrition selections. The breakdown of this multiple
scoring is as follows:
- Of the 17 patients recorded as cardiac failure, four were also recorded in at least one
  other category
- Of the eight patients recorded as having peripheral vascular disease, two were also
  recorded in at least one other category
- Of the ten patients recorded as having anaemia, three were also recorded in at least
  one other category
- Of the 19 patients recorded as smoking, four were also recorded in at least one other
category
APPENDIX Q – RESULTS OF TESTS FOR NORMALITY

Table M-1 below shows the results of the Kolmogorov-Smirov Test (K-S test) for each variable under investigation. The initial analysis of the data for normality showed that none of the variables were normally distributed. The results of the K-S test after the data was transformed using a Logarithmic transformation base 10 is shown at Table M-2.

Table Q-1. Initial analysis for normality of study parameters

<table>
<thead>
<tr>
<th>Variables</th>
<th>n</th>
<th>M</th>
<th>SD</th>
<th>Skew</th>
<th>Std Err</th>
<th>Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td>120</td>
<td>78.83</td>
<td>18.597</td>
<td>0.967</td>
<td>1.698</td>
<td>2.407</td>
</tr>
<tr>
<td>BMI</td>
<td>120</td>
<td>26.95</td>
<td>5.616</td>
<td>0.856</td>
<td>0.517</td>
<td>0.755</td>
</tr>
<tr>
<td>Waterlow risk level</td>
<td>120</td>
<td>10.93</td>
<td>5.693</td>
<td>0.816</td>
<td>0.520</td>
<td>0.396</td>
</tr>
<tr>
<td>Peak interface</td>
<td>120</td>
<td>54.90</td>
<td>12.629</td>
<td>0.701</td>
<td>1.152</td>
<td>0.120</td>
</tr>
<tr>
<td>Gradient 1.5cm</td>
<td>120</td>
<td>11.45</td>
<td>5.012</td>
<td>1.291</td>
<td>0.458</td>
<td>1.564</td>
</tr>
<tr>
<td>Gradient 2.5cm</td>
<td>120</td>
<td>9.32</td>
<td>3.486</td>
<td>1.446</td>
<td>0.318</td>
<td>3.036</td>
</tr>
</tbody>
</table>
Table Q-2. Analysis of study parameters after Logarithmic transformation

<table>
<thead>
<tr>
<th>Variables</th>
<th>n</th>
<th>M</th>
<th>SD</th>
<th>Skew</th>
<th>Std err</th>
<th>Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td>120</td>
<td>1.8852</td>
<td>1.1001</td>
<td>0.012</td>
<td>0.00913</td>
<td>0.692</td>
</tr>
<tr>
<td>BMI</td>
<td>120</td>
<td>1.4216</td>
<td>0.08803</td>
<td>0.267</td>
<td>0.00804</td>
<td>0.053</td>
</tr>
<tr>
<td>Waterlow risk level</td>
<td>120</td>
<td>1.9767</td>
<td>0.24200</td>
<td>-0.373</td>
<td>0.02200</td>
<td>-0.349</td>
</tr>
<tr>
<td>Peak interface pressure</td>
<td>120</td>
<td>1.7286</td>
<td>0.09739</td>
<td>0.167</td>
<td>0.00889</td>
<td>-0.339</td>
</tr>
<tr>
<td>Gradient 1.5cm</td>
<td>120</td>
<td>1.0228</td>
<td>0.17438</td>
<td>0.372</td>
<td>0.1592</td>
<td>-0.391</td>
</tr>
<tr>
<td>Gradient 2.5cm</td>
<td>120</td>
<td>0.9429</td>
<td>0.14933</td>
<td>0.331</td>
<td>0.01363</td>
<td>0.124</td>
</tr>
</tbody>
</table>