# Predicting and preventing pressure sores in surgical patients

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# Predicting and Preventing Pressure Sores in Surgical Patients


## Abstract

The thesis comprises literature reviews which present arguments novel to the field and two discrete but related studies, which in combination make a contribution to the classification, assessment of risk and prevention of pressure sores.

The first study, a randomised controlled trial involving 446 patients undergoing vascular, general and gynaecology surgery, the use of a dry visco-elastic polymer pad intra-operatively reduced the probability of pressure sore development by half. Pressure sore incidence was 11% (22/205) for patients allocated to the dry polymer pad and 20% (43/211) for patients allocated to the standard operating table mattress.

Both studies explored key prognostic factors using multi-variate methods. Analysis of data derived from the randomised controlled trial found four factors to be independently associated with post-operative pressure sore development including intra-operative hypotensive episodes, Day 1 Braden mobility scale and intra-operative mean core temperature. The second study, a prospective cohort study involving 101 patients identified non-blanching erythema, pre-operative albumin, weight loss preceding admission and intra-operative minimum diastolic blood pressure. Results are consistent with findings from the literature review which identified key factors in the prediction of pressure sore development (reduced mobility, nutrition, perfusion, age and skin condition).

The second study also explored the clinical significance of erythema in defining and classifying the term 'pressure sore'. Using laser Doppler imaging it was determined that blanching and non-blanching erythema are characterised by high blood flow of differing intensity. Discriminant analysis identified three general patterns in skin blood flow, which enabled scan classification with good agreement between clinical and predicted classifications. The results confirm data derived from the prospective observations of skin suggesting that non-blanching erythema is not indicative of irreversible ischaemic damage and resolves in approximately two thirds of cases. The point at which non-blanching erythema becomes irreversible remains unknown.

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## Statement of Research Collaboration

The thesis comprises two discrete research studies. The first study, a two centre randomised controlled trial involving 446 surgical patients was undertaken in collaboration with the Northern and Yorkshire Clinical Trials and Research Unit (NYCTRU),University of Leeds who offer a specialised Trials service. The roles and responsibilities of myself (Principal Investigator) and the NYCTRU were as follows.

I instigated the research, determined the main research question and made the original funding application to Yorkshire Regional Health Authority to undertake a randomised controlled trial comparing the standard operating table mattress with the dry visco-elastic polymer pad. Funding was agreed in principle but the original proposal was then further developed with methodological advice from the NYCTRU which included the double triangular sequential design. Funding was subsequently agreed by the Yorkshire Regional Health Authority (which later became NHSE Northern and Yorkshire) and included the trial co-ordination services of NYCTRU.

During the trial set-up my role was clearly defined as principal investigator and clinical co-ordinator, working with the NYCTRU in protocol development and data collection forms, securing ethical approval, obtaining manager/ward/theatre/ Consultant approval, Clinical Research Nurse(CRN) appointments and training, budgetary and management arrangements, inter-rater reliability study and equipment purchase. When data collection commenced I remained responsible for clinical site management, CRN support and development, the research budget and where necessary participated in recruitment and data collection.

The NYCTRU co-ordinated protocol development and data collection forms, established the database and associated data validation processes, set-up the telephone randomisation facility and the Data Monitoring Committee. During the trial the NYCTRU had responsibility for all aspects of data management including validation, monitoring and storage. Sequential analyses were undertaken and reported to the Independent Data Monitoring Committee.

All statistical methodologies and analyses were determined and undertaken by the NYCTRU. The prognostic factor analysis was refined and developed with my input

providing clinical expertise and interpretation. I analysed the inter-rater reliability study, non-randomised patients and skin data collected beyond the primary endpoint.

Following the main analyses, I co-ordinated the study write-up working closely with the NYCTRU statisticians to ensure accuracy in the presentation of the methodology and results.

In the second study, an exploratory study involving laser Doppler imaging of skin and a prospective cohort, research collaborations were established at the proposal development stage and during data analyses as follows.

I was principal investigator on all research applications, with co-applicants including Mr Julian Scott (Consultant Vascular Surgeon) and Dr Stephen Smye (Director of R&D/Head of Medical Physics) for the application to the NHSE Northern and Yorkshire Research Capacity Committee. I instigated the research, determined the main research question and study design for the prospective cohort study, but sought advice and support in the use and application of physiological measurement technologies.

During the study, as principle investigator I developed the research protocol, set up the study obtaining relevant approval (ethics, manager, ward sister, Consultant) and maintained overall responsibility for the conduct of the study, data entry including validation and associated budgetary management.

In relation to statistical techniques I determined the statistical design and undertook all analyses for the prospective cohort study with supervision provided by Mr Dawood Dassu (NYCTRU). Methodological advice was sought from Dr Robert Ackroyd (University of Leeds) for the laser Doppler imaging and the discriminant analysis was undertaken by Dr Michael Hutchinson (University of Newcastle). During the study write-up statistical supervision was maintained to ensure accuracy in the presentation of the methodology and results.

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## List of Abbreviations

| | |
|---|---|
| AHCPR | Agency for Health Care Policy and Research |
| ATP | Adenosine triphosphate |
| BP | Blood Pressure |
| CRN | Clinical Research Nurse |
| DMC | Data Monitoring Committee |
| DOH | Department of Health |
| ICU | Intensive Care Unit |
| LOS | Length of Stay |
| Max | Maximum |
| Min | Minimum |
| NPS | No pressure sore |
| PACU | Post Anaesthetic Care Unit |
| PEST | Planning and Evaluation of Sequential Trial Software |
| Pre-op | Pre-operative |
| PS | Pressure sore |
| PU | Perfusion Unit |
| SAS | Statistical Analysis Software, SAS Institute Inc |
| UK | United Kingdom |

# Predicting and Preventing Pressure Sores in Surgical Patients

## Preface

This thesis is part of a programme of research, which aims to improve assessment, skin classification and prevention of pressure sores. The work builds upon published reviews of the literature and small exploratory study previously accredited. This work, together with a NHS programme to promote research based practice identified priorities for research.

Original publications including reviews of the literature presented in Appendix 2 are summarised in Chapter 1, with all further work being original to this thesis. Reviews presented in Chapters 2 and 6 challenge much of the rhetoric within the current pressure area care literature and present arguments which are novel to the field. In particular the application of the existing research base to the daily practice of nursing has been considered in order to clarify key issues for research. For example, evidence regarding the pathology of pressure sore development is linked to clinical observations of intact skin and identified the need to undertake basic physiological research.

The thesis comprises two discrete but related studies, which in combination make a contribution to the classification, assessment of risk and prevention of pressure sores. The two studies are presented in Sections 1 (Chapter 1-5) and 2 (Chapters 6-10). Section 1 outlines a randomised controlled trial of an intra-operative mattress intervention for the prevention of pressure sores and prognostic factor analysis to identify peri-operative risk factors and this informed further investigation. Section 2 details an exploration of skin changes preceding pressure sore development and further investigation of prognostic factors.

The findings from both studies, summarised in Section 3 (Chapter 11) have important implications for practice and research in relation to intra-operative pressure sore prevention, prediction of pressure sores and classification and definition of pressure sores.

The literature base supporting the work has evolved over a ten-year period. Manual searching commenced in 1991 using the International Nursing Index (1975 onwards)

and Index Medicus. Relevant citations, such as books and articles were sought following review of the initial literature obtained. The literature was subsequently searched on an annual basis using the International Nursing Index, Index Medicus and later CINHAL and MEDLINE, with manual searching of the journal of Tissue Viability and conference proceedings such as European Wound Management Association.

In 1998 MEDLINE was searched from 1976 to 1997 using the MESH term DECUBITUS combined with the text words erythema, reactive hyperaemia, ischaemia, skin blood flow, foot ulcer, diabetic foot, transcutaneous oxygen, laser Doppler in various combinations and the names of known researchers in the field (including, Fagrell and Bader). The literature obtained was then systematically assessed and further citations retrieved. This pathophysiology literature was further supplemented by three systematic reviews of the literature relating to pressure sore risk assessment scales and the effectiveness of preventive interventions [1-3].

# Intra-operative Pressure Sore Prevention and Prediction

## Chapter 1 Background and Exploratory Study

### 1.1 Introduction

Pressure sores are described as 'a lesion on any skin surface that occurs as a result of pressure and includes reactive hyperaemia as well as blistered, broken or necrotic skin'[4]. They are complex lesions of the skin and underlying structures and vary considerably in size and severity. The majority of pressure sores occur below the waist with particularly vulnerable areas being the sacrum, buttocks and heels[5].

The principal causative factor is the application of localised pressure to an area of skin not adapted to the magnitude of such external forces. They are associated with increased mortality rates and are a marker for underlying disease severity and other comorbidities[6-9].

Pressure sores have both cost and quality implications for health services and they are increasingly seen as preventable sequela rather than a tolerable complication of illness. The emphasis is on identifying risk factors and implementing appropriate interventions to prevent pressure sore occurrence[10].

This is reflected in the development of a plethora of local pressure sore prevention policies which advocate risk assessment, skin care, repositioning, equipment provision and planned monitoring of the problem[11]. The majority of recommendations relate to the care of patients within hospital wards but many of the policies also advocate pressure sore prevention from pre-admission to discharge and make specific reference to assessment and equipment provision within Radiology and Accident and Emergency Departments and Operating Theatres.

However, many practice recommendations are not based on good research evidence. In particular, the evidence base associated with assessment of risk is limited and the effectiveness of preventative interventions have not been demonstrated using robust research methodologies[1].

This chapter provides a brief summary of key areas of the literature including the definition and classification of pressure sores, aetiology and a brief summary of previous research involving surgical patients and peri-operative care.

## 1.2 Definition and Classification

There is no agreed definition of the term pressure sore and researchers utilise a range of definitions including blanching erythema, blanching erythema of a specified duration (minutes or consecutive days), non-blanching erythema and skin break in order to determine the prevalence or incidence[12-15].

The severity of pressure sores varies from reactive hyperaemia to tissue destruction involving skin, subcutaneous fat, muscle and bone - hence a number of classification systems have been developed[10;16-24].

With the exception of Barton and Barton[16], classification systems 'grade' or 'stage' 4 or 5 categories to describe pressure sores reflecting the tissue layers affected (epidermis, dermis, sub-cutaneous fat, muscle and bone). Most include a category for skin changes of intact skin which vary from a hyperaemic response of short duration to persistent non-blanching discolouration with localised swelling, heat and induration[21;22]. Pressure sore rates are usually reported in terms of total numbers and associated grade or stage.

Barton and Barton[16]classify pressure sores on the basis of pathological study and determined two categories of sore. They observed pressure sores that initially developed as superficial loss of the epidermis with gradual destruction of deeper tissues when pressure remained unrelieved. These they categorised as Type I pressure sores. Also observed were pressure sores initiated by ischaemia of muscle and subcutaneous fat resulting in widespread deep tissue damage before destruction of the dependent superficial layers and the appearance of a deep sore. These they categorised as Type II pressure sores. Clinical reports suggest that manifestation of these pressure sores occurs six to eight days following the pressure assault[25;26].

## 1.3 Aetiology

A review of the mechanisms that protect the skin microvasculature from ischaemic assault and restore local tissue perfusion following occlusion illustrates clearly that there is an interaction between the pressure assault and the capacity of the skin to maintain and effectively restore skin blood flow. A number of auto-regulatory mechanisms exist to protect the skin from pressure assault and these processes break down at pressure values that are highly variable. Pressure sore development is multidimensional and complex[27].

A conceptual schema for the study of the aetiology of pressure sores was developed by Braden and Bergstrom[28] and provides a useful framework. They identified the critical determinants of pressure sore development as the intensity and duration of pressure and the tolerance of the skin and it's supporting structure to pressure. At an individual level pressure sores develop as a result of the interaction between these two elements.

### *1.3.1 Intensity and Duration of Pressure*

The primary cause of pressure sores is the application of pressure in areas of skin and tissue not adapted to external pressure assault. Whilst no critical threshold values can be determined in relation to intensity and duration of pressure previous review has established important principles[27].

***Local verses uniform pressure.*** The nature of the pressure assault is important in the development of pressure sores. It is the effect of the application of a local or point pressure upon the skin that is of interest in pressure sore aetiology. Such localised pressure is complicated by shear forces, contact area, underlying bone, bone depth, pressure distribution, contact surface conditions and associated tissue distortion[29-31].

***Critical pressure thresholds.*** It appears that the autoregulation processes that maintain skin blood flow during pressure assault break down at pressure values that are highly variable and there is no universal capillary occlusion threshold level. The 'critical closing pressure' is the pressure within a vessel at which it collapses completely and blood flow ceases. It is determined by an interplay of forces including intravascular pressure, muscle contraction and elastic forces of the blood vessel wall

and externally applied pressure[32]. That at least four variables are involved explains why no individual response is the same, although trends are apparent.

***Parabolic intensity-duration curve.*** Studies which examine the relationship between pressure and time ulcer/no ulcer all report an inverse relationship between the amount and duration of pressure, that is, low pressure for long periods and high pressure for short periods both cause ulceration[33-36].

***Critical time threshold.*** Reappraisal of early studies[37-39] suggests that once a critical pressure threshold and critical time value is exceeded then tissue damage will proceed at a similar rate regardless of the magnitude of the pressure applied [27].

### *1.3.2 Tolerance of the Skin to Pressure*

Braden and Bergstrom[28] use the term tissue tolerance to 'denote the ability of both the skin and it's supporting structures to endure the effects of pressure without adverse sequela'. They distinguish between extrinsic and intrinsic factors affecting tissue tolerance and describe intrinsic factors as 'those that influence the architecture and integrity of the skin's supporting structures and/or the vascular and lymphatic system that serves the skin and underlying structures.'

***Extrinsic factors.*** The main extrinsic factor affecting skin tolerance to pressure is the application of frictional forces, which exacerbates the pressure assault by causing mechanical disruption of the epidermis[35;40]. Other extrinsic factors commonly cited in the literature include moisture and skin irritants. It is noteworthy that urinary incontinence is not identified as a risk factor by studies utilising multivariate statistical analyses (see Chapter 2), suggesting that incontinence/moisture is not a primary factor but a symptom or indicator of poor physical condition, particularly in elderly populations. Skin irritants have been the subject of little research and their contribution is unknown.

***Architecture of the skin.*** Many intrinsic variables associated with pressure sore development directly affect collagen an important element within the structure of the skin and underlying tissues. Attention to this important structure has developed following observations, which revealed that the collagen content of the dermis is reduced following spinal cord injury[41]. It appears that collagen prevents disruption to the microcirculation by buffering the interstitial fluid from external load, thereby

maintaining the balance of hydrostatic and osmotic pressures[42]. The collagen theory interrelates with other predisposing factors such as age, nutrition and spinal cord injury, which affect the synthesis, maturation and degradation of the connective tissue[27].

***Perfusion.*** A large number of intrinsic perfusion related factors are associated with pressure sore development including systemic blood pressure, extracorporeal circulation, serum protein, smoking, serum haemoglobin, diseases of the vascular system, vasoactive drug administration and increased body temperature[27]. The literature suggests an overall trend. That is, the tolerance of the skin is affected by perfusion related variables but there is no single cause-effect factor. This can be linked to the physiology of blood flow and the interplay of factors which determine capillary blood pressure, exchange mechanisms between capillaries and interstitial fluid[32] and factors affecting the availability of essential nutrients (particularly oxygen) to the local tissue.

The development of pressure sores is determined by various aetiological factors particular to individual patients that determine the ability of the skin to respond to external pressure and maintain skin integrity.

## 1.4 Intra-operative Pressure Sores and Exploratory Study

A literature review of intra-operative pressure sore development suggested a causal relationship between events occurring during surgery and the subsequent development of pressure sores[43;44]. However, there are few prospective studies[12;25;45] and with the exception of Kemp et al[12] published studies fail to specify research design or include specific factors on pressure sore aetiology. Research and case study reports of Barton and Barton Type II pressure sores suggest an increased risk amongst vascular and cardio-vascular surgical patients due to prolonged periods on the operating table and perfusion related variables[12;25;26].

The incidence of intra-operative pressure sores had not been explored in an UK hospital setting, yet many hospital pressure sore prevention policies included practice recommendations for operating theatres. Recommendations for the peri-operative period generally included pre-operative risk assessment and provision of equipment from a limited range designed for use on operating tables.

Products available for use on operating tables included a dry visco-elastic polymer pad, replacement foam mattresses, a liquid displacement cell mattress and silicone fibre overlays[46]. In 1994, during protocol development none of these product types had been subjected to clinical evaluation by randomised control trial. Two had been evaluated under laboratory conditions (dry visco-elastic polymer pad and liquid displacement cell mattress) using non-anaesthetised volunteers and both demonstrated reduced interface pressure measurements at key anatomical sites or total body areas compared with the conventional operating table mattresses[47;48].

The dry visco-elastic polymer pad had also been evaluated in a small prospective study[25]. Of the 89 patients positioned on the dry polymer pad 34% were reported as having blanching erythema and 3.3% Stage 2 pressure sores, defined as 'redness, oedema and induration at times with epidermal blistering or desquamation'. Interpretation of these results is difficult due to numerous limitations in the reporting of the study. Furthermore, the absence of a control group prevents any conclusion regarding their effectiveness in reducing or preventing pressure sores.

An exploratory study was undertaken in Hartlepool General Hospital in 1992[44;49]. The aims of the study were to: -

1. Assess the post-operative incidence of skin damage within an UK hospital.
2. Explore the reliability and validity of the Braden Scale in an UK hospital.
3. Provide data that may justify the implementation of preventative strategies.

The study sample comprised 24 elective surgical patients. All were positioned on a standard operating theatre mattress and using a definition of the term pressure sore as persistent discolouration at the same site for two consecutive days or more, a 'theatre' pressure sore incidence of 12.5% was determined. The skin changes persisted for 1-5 days post-operatively. The extent of the problem in this small sample of relatively 'low risk' elective surgical patients suggested the need or further work.

Findings informed subsequent research as follows:

A limitation of the Torrance skin classification was identified, whereby Grade 2 includes both non-blanching erythema and superficial epidermal damage. Prior to the study it was assumed that non-blanching erythema was a sign of irreversible damage leading to skin loss[19;27] and therefore acceptable to combine both elements within

one stage. However, skin loss was not found to be an inevitable consequence of non-blanching erythema indicating the need to distinguish between intact and broken skin.

Observations of patients during the immediate post-operative period identified reactive hyperaemia responses ranging from less than 2 minutes to greater than 30 minutes and highlighted the need for an assessment schedule to distinguish between a normal and abnormal hyperaemic response.

An unexpected finding was a pre-operative pressure sore prevalence of 33.3% amongst a relatively low risk sample of patients. No comparative data was available and highlighted the need to take account of pre-operative pressure sores in further research.

The Braden Scale was found to have 44.4% absolute agreement between assessors, 88.8% within 1 point agreement and 100% agreement for risk/not at risk categorisation. These results were very favourable compared with the reliability of other risk assessment scales[50]. On this basis the Braden Scale was recommended for use in further research as a reliable framework for the recording and exploration of prognostic factors (see Chapter 2).

## 1.5 Summary

A brief overview of the literature and an exploratory study involving surgical patients highlights the multi-dimensional nature of pressure sore development, the individual nature of the skins response to external forces including pressure and suggests a causal relationship between events during surgery and subsequent development of pressure sores. The review and exploratory study determines a need for further exploration of pressure sore occurrence in surgical populations, evaluation of the relative effectiveness of intra-operative interventions in reducing or preventing pressure sores and highlights the need to refine the classification of pressure sores in order to distinguish between intact and broken skin and a normal and abnormal hyperaemic response.

# Chapter 2 Assessment of Pressure Sore Risk

## 2.1 Introduction

In the literature concerning the prevention and management of pressure sores baseline assessment is commonly associated with the term 'risk assessment' and there has been a focus toward the development and use of risk assessment scales to facilitate the identification of 'at risk' patients[1;23].

However, there is increasing debate within the literature about how to undertake risk assessment and in particular the role of risk assessment scales[51]. It was recommended by the United States Agency for Health Care Policy and Research (AHCPR) that, 'A systematic risk assessment can be accomplished by using a validated risk assessment tool such as the Braden Scale or Norton Score.'[23].

In contrast the Effective Health Care Bulletin on the prevention and treatment of pressure sores concluded that, 'The evidence on the accuracy of pressure sore risk scales is confusing, and it is not clear that these scales are better than clinical judgement or that they improve outcomes'[1].

The research literature serves to underline the complexity of the processes involved in both nursing assessment of patient need and factors associated with pressure sore development. In order to inform the debate the following sections review evidence relating to the cognitive processes involved in general nursing assessment, issues relating to the construction, limitations and validity of risk assessment scales and prospective cohort studies which identify prognostic factors associated with pressure sore development.

## 2.2 Cognitive Process in Risk Assessment

The purpose of baseline nursing assessment is to identify both actual and potential problems, which then inform individual planning and delivery of effective care. In

practice, then, baseline assessment aims to answer the following two clinically important questions:

- Has the patient an existing pressure sore?
- What is the patients risk of pressure sore development?

Practice recommendations which focus entirely upon risk assessment of pressure sore free patients such as the AHCPR guidelines[23] do not address the first clinical question and the practising nurse is, then, faced with three clinical management issues:

- Who to assess to determine the presence or absence of an existing pressure sore?
- Who to assess for potential risk of pressure sore development?
- How to assess for potential risk of pressure sore development?

In order to determine whom to assess for the presence or absence of existing pressure sores and potential risk, high risk groups and broad characteristics of patients most likely to present with or develop pressure sores within health care settings can be identified from the epidemiological literature.

High prevalence and incidence of pressure sores are reported amongst patient groups which reflect these characteristics including: elderly medical[11;52], nursing home[6;53;54], cardiovascular and vascular surgical[12;25], acute orthopaedic/hip fracture[11;14;54;55], intensive care[8;56], spinal cord injured[57], the young disabled[58] and the terminally ill[7;9;59].

The main characteristic associated with both the presence of and development of pressure sores is reduced mobility and increased age[15;18;54;60]. It is suggested that baseline skin inspection to determine the presence or absence of a pressure sore and to identify patients who require assessment of potential risk is required if one or more of the following criteria apply:

- mobility/activity restrictions - confined to bed or chair
- aged over 75 years
- high risk group [61]

When baseline skin assessment identifies that a patient has an existing pressure sore the patient has an actual problem which requires treatment and that treatment will depend upon the severity of the sore and the individual patient circumstance. Patients with existing pressure sores are also at risk of further pressure sore development and require active prevention.

When baseline skin assessment determines that a patient is pressure sore free, the purpose of further assessment is to identify any potential risk of pressure sore development in order that preventative measures can be adopted.

## 2.3 Nursing Assessment - An Overview

Nursing assessment is a dynamic decision making process[62]. Its purpose is to provide an accurate picture of the patient including both their capacity to perform activities of daily living and the stability of their condition. Judgements are then made regarding the nursing care required and frequency of monitoring[62;63].

Various elements have been identified which are important in assimilation and interpretation of information by nurses whilst assessing patients. There is evidence that nurses develop knowledge structures for gathering and organising information, which enables them to select, weight and combine important factors. Nurses also distinguish between relevant and irrelevant information and cues, link past experiences and knowledge to the current situation and grasp the whole situation rather than distinguishing a series of sub-tasks[64-66].

The quality of assessments and associated decisions about the nursing care that is required are related to underpinning theoretical knowledge, previous experience, specialty based knowledge relating to usual patterns of recovery, perceptual awareness, recognition skills and knowing the patient[62;63;66;67].

Knowing the patient is increasingly understood to be a key component of excellent nursing practice[67;68]. Through a finely tuned knowledge of their patients, experienced nurses are able to notice opportunities for action, to understand the meaningfulness and importance of ordinary occurrences and to act upon them, promoting patient recovery. The process of knowing the patient shapes caring activities and is integrally linked with patient outcomes[67].

Crow et al[62] in their critical review illustrate the dynamic and continuous nature of nursing assessment. Nurses continually update assessments of patients as their condition changes. They recognise their usual pattern of responses and use both their current status and judgements of the predicted future course of the patient to inform decisions about nursing care needs[67;69-72]. It follows that any shortcomings in the judgements nurses formulate will lead to decision errors[62].

A review of the components involved in nursing assessment illustrates the complexity of the processes involved. The process is dynamic and continuous with the nurse making judgements about immediate and future care needs including the frequency of monitoring. In relation to pressure sore prevention it raises questions with regard the appropriateness of summarising with a single score something which is multi-faceted[73], the development of clinical assessment skills (from novice to expert) and the role of risk assessment scales in nursing assessment of potential risk.

## 2.4 Construction and Limitations of Risk Assessment Scales

The most valid method of constructing predictive scales involves the use of statistical regression models to identify, rank and weight the factors which together best predict the development of a pressure sore[1;74]. Such studies are known as prognostic factor studies and they are generating increased methodological scrutiny and critique as statistical techniques advance within the medical research arena[75].

Simon and Altman[75] describe three types of prognostic studies including:
1. Early exploratory studies. Such investigations commonly examine issues such as the association of a factor with diagnosis and disease characteristics or the development of reproducible assays.
2. Studies to determine whether prognostic factors provide improved means of identifying patients at particularly high or low risk of disease progression or death.
3. Studies to determine which subsets of patients benefit from a given therapy.

They suggest that type 1 studies might be called Phase 1 prognostic factor studies and type 2 and 3 each include what might be called Phase 11 and Phase 111 factor studies. The Phase 11 studies are exploratory and generate hypotheses from extensive analysis of data. Phase 111 studies are large, confirmatory studies of pre-stated hypotheses and allow for more precise quantification of the effect[75]. Also

detailed are methodological recommendations for the conduct of prognostic factor studies including the use of inception cohorts, <15% patients lost to follow up, reproducible measures, blinding of outcome, standardised or randomised treatment, pre-stated hypotheses and sample size calculations.

Although there is a considerable pressure sore literature only 12 cohort studies have been found which determine key prognostic factors associated with pressure sore development (Table 2.1). These can only be considered as Phase 1 prognostic factor studies. Whilst themes can be identified, the small number of prospective studies world-wide and the diversity of health care settings and associated risks, highlights that pressure sore prognostic factor research is very much in its infancy. Questions remain outstanding in relation to the magnitude of effect of generic and specialty specific risk factors in different patient populations.

In a recent systematic review more than 40 pressure sore risk assessment scales were found detailed in the literature[2]. The review determined that the majority of risk assessment scales have been developed on the basis of expert opinion, literature review and/or adaptation of an existing scale with only seven original scales. Of the seven original scales only one had selected variables identified through a regression analysis of an inception cohort study[76]. Indeed, 61% were developed by modification of an existing scale and one scale - the Dutch Consensus Scale[77] is a 4th generation modification of the original Norton Score[2].

There is then no statistical basis to the majority of risk assessment scales either in relation to the selection of risk factors or the scores allocated to elements within the scales. In most risk assessment scales using a simple ordinal scoring system, the weighting within the scale is equally allocated between risk factors. Thus any potential differences in the contribution or importance of one factor over another or the cumulative importance of two or more factors are not identified.

The absence of a statistical base is evident in the large number of variables found in risk assessment scales. McGough[2] identified 23 different variables in 38 modified risk assessment scales with most frequent inclusion of continence/moisture (36 scales), nutrition/appetite (32 scales) and mobility (30 scales). Given that only 2/12 studies using multi-variate analyses identify continence variables as important (both identified faecal incontinence), whilst 10/12 identify mobility related factors the validity of these scales in defining 'at risk' patients is immediately questionable.

Limitations in the construction of risk assessment scales are reflected in results of testing of their predictive validity. McGough's[2] systematic review found only 6 of 43 risk assessment scales have been tested for their predictive validity. Of these six scales, the Braden Scale has been subjected to the most testing across the greatest variety of clinical settings, both in the hospital and in the community. Although Bergstrom and colleagues have demonstrated that the predictive validity estimates for the Braden Scale have been high, other researchers have failed to replicate these findings[77-81]. The different patient populations studied may partially account for the variation, as the incidence of pressure sores within each setting will vary. However, this cannot explain the difference in predictive validity values obtained in the same care setting such as a medical-surgical acute care units[77-80;82].

There also remain many outstanding questions in relation to the validity and effectiveness of risk assessment scales. Edwards[51] doubts the appropriateness of applying measures of sensitivity and specificity to scales which from their conception were never based or tested on mathematical models. Difficulties in comparing the validity of different scales are highlighted by Deeks[73] who points out that nursing care in the research environment will effect both sensitivity and specificity and that risk assessment scales will appear to be performing most poorly in the settings where preventative care is most effective. The testing of these scales is further compounded by the incipient nature of pressure sore development. Risk assessment scales are only a snapshot view and in acute or critical illness the score may not reflect the patient's condition at the time of pressure sore development.

Furthermore, there is no evidence that risk assessment scales are better than nurse's judgement in identifying patients at risk[1;2] and insufficient evidence to determine whether risk assessment scales are effective in reducing the incidence of pressure sores[2].

## 2.5 Clinical Utility of Risk Assessment Scales

A review of the evidence challenges a central role for risk assessment scales as a valid method of screening for risk of pressure sore development as recommended by the AHCPR[23]. However, the literature highlights the complex nature of pressure sore development and the numerous factors potentially involved. Within a practice setting

awareness and assessment may be variable and risk assessment scales may be of use in providing a framework for assessment, highlighting key risk factors.

Risk assessment scales have been developed in an attempt to provide a structure and consistency to patient assessment. In a review of 138 pressure sore prevention policies 90% recommended their use[83]. Their widespread utilisation would suggest clinical nurses' value them and that their limitations are not necessarily important within the clinical environment. Evidence suggests that their introduction in conjunction with the establishment of skin care teams, education programmes and care protocols may reduce the incidence of pressure sores[2]. They can provide a number of important advantages which can be applied to practice[61]. However, the use of such scales as a single instrument to assess patient risk of pressure sore development cannot be supported on the basis of current evidence.

## 2.6 Key Prognostic Factors

A search of the literature (see Preface) identifies 12 cohort studies, which have undertaken multi-variate analyses to identify characteristics of patients who develop pressure sores (Table 2.1). The studies cited have used a variety of possible risk factors, measured similar variables in different ways, included various population groups and do not utilise a common pressure sore definition. Despite these differences, five key themes emerge including mobility, nutrition, perfusion, age and skin condition. These can be directly related to the aetiology of pressure sore development where the interaction between the intensity and duration of pressure (mobility) and the tolerance of the skin (nutrition, perfusion and age) determines the skin response (skin condition).

**Table 2. 1 Cohort Studies Utilising Multi-variate Statistical Analyses**

| Study | Sample | Incidence | Prognostic Factors | Statistical Method |
|---|---|---|---|---|
| Clarke and Kadhom 1988 [84] | 88 Hospitalised (orthopaedic, elderly+ICU) bedfast/chairfast | 29.5% | 1.change in condition of skin<br>2.time on pressure area care<br>3.appetite<br>4.Norton Score<br>5.diagnosis<br>6.method of manual pressure relief<br>7.observed skin condition<br>8.age | Discriminant Analysis |
| | 30 Community bedfast/chairfast | 20% | 1.appetite<br>2.condition skin<br>3.frequency of care<br>4.Norton Score<br>5.age<br>6.diagnosis | |
| Guralnik et al 1988 [85] | 5193 US nation-wide cohort 55-75years 10 year follow-up | 2.2% | 1.heart disease (negative association)<br>2.activity level<br>3.self assessed health<br>4.smoking<br>5.neurologic abnormality<br>6.dry or scaling skin<br>7.anaemia (Hb<12) | Multiple Logistic Regression |
| Berlowitz and Wilking 1989 [60] (Record review) | 185 chronic medical | 10.8% | 1. cerebrovascular accident<br>2. bed or chair bound<br>3. impaired nutritional intake | Multiple Logistic Regression |
| Kemp et al 1990 [12] | 125 Surgical >20 years stratified by operating time | 12% | 1.age<br>2.time on operating table<br>3.extracorporeal circulation | Discriminant analysis |

| | | | | |
|---|---|---|---|---|
| Ek et al 1991 [13] | 495 long term medical<br>LOS >3weeks | 10.1% | 1.albumin<br>2.mobility<br>3.activity<br>4.food intake | Multiple Regression |
| Marchette et al 1991 [86]<br>(Record review) | 161 postoperative ICU<br>>59 years | 39.1% | 1. skin redness<br>2. days static air mattress for prevention<br>3. fecal incontinence<br>4. diarrhoea<br>5. preoperative albumin | Discriminant Analysis |
| Bergstrom and Braden 1992 [6] | 200 nursing home<br>> 65 years<br>LOS>10days<br>Braden <18 | 73.5% | 1.Braden Scale<br>2.diastolic blood pressure<br>3.temperature<br>4.dietary protein intake<br>5.age | Logistic Regression |
| Hoshowsky and Schramm 1994 [87] | 505 surgical | 16.8% | 1.time on operating table<br>2.vascular disease<br>3.age over 40 years<br>4.pre-operative Hemphill scale | Logistic Regression |
| Brandeis et al 1994 [53] | 1322 nursing home | 12.9% | 1.ambulation difficulty<br>2.fecal incontinence<br>3.diabetes mellitus<br>4.difficulty feeding oneself | Logistic Regression |
| Allman et al 1995 [88] | 286 hospitalised<br>>55 years<br>bed/chair >5 days<br>hip fracture<br>length of stay >5days | 12.9:% | 1. nonblanchable erythema of intact sacral skin<br>2. lymphopenia<br>3. immobility<br>4. dry sacral skin<br>5. decreased body weight | Multivariate Cox Regression |
| Bergstrom et al 1996[89] | 843 nursing home and acute | 12.8% | Model 1<br>1.Braden Scale<br>2.age<br>3.race<br>Model 2<br>1.mobility<br>2.activity<br>3.Cardiovascular disease | Logistic Regression |
| Schnelle et al 1997[90] | 100 nursing home incontinent | 21% | 1.blanchable erythema severity | Stepwise multiple regression |

### *2.6.1 Mobility*

The important relationship between reduced mobility and pressure sore occurrence suggested by early prevalence surveys are confirmed by cohort studies which identify mobility related factors to be significant and independent predictors of pressure sore development (Table 2.1). Of the 12 studies detailed in Table 2.1, 10 identify mobility related factors as important determinants of pressure sore development. Interpretation broadly identifies mobility related factors as all of the following: method of manual pressure relief; activity level; neurological abnormality; bed or chair bound; time on operating table; mobility; days static air mattress for prevention; ambulation difficulty and immobility.

### *2.6.2 Factors Affecting Skin Tolerance*

Studies utilising univariate analyses have identified a large number of factors affecting skin tolerance which are significantly associated with pressure sore development[27]. However, the three themes emerging from multi-variate analyses both challenge and support some common assumptions made with regard the relative importance of pressure sore risk factors.

Nutrition related factors including: appetite; anaemia; impaired nutritional intake; albumin; dietary protein intake; difficulty feeding oneself; lymphopenia and decreased body weight, are identified by 8 of the 12 studies although the exact relationship remains unclear. It is likely that reduced dietary intake is a general indicator of morbidity, as well as directly affecting tissue perfusion and skin structures which reduce tolerance to pressure[27]. The wide range of factors identified within such a small number of research studies reflects the absence of recognised indicators of nutritional status.

Factors affecting tissue perfusion including: smoking; anaemia; cerebrovascular accident; extracorporeal circulation; blood pressure; vascular disease and; diabetes mellitus are identified by 7 studies. This is the most diverse area of the literature reflecting the large number of variables that affect tissue perfusion. The existing research base cannot identify key factors more specific than 'perfusion related' and there is a clear need for further exploration of these variables in specialty specific patient populations.

Four of the cohort studies determine age as associated with pressure sore development. Other studies suggest that in high risk groups age is less important

than associated morbidity [60;88]. The relationship is likely to be multi-factoral and related to both increased morbidity and disease, which affect mobility and age related changes of the skin and these in turn reduce tissue tolerance[91].

It is noteworthy that urinary incontinence is not identified by any of the studies as associated with pressure sore development using multi-variate statistical techniques. This challenges the common assumption that incontinence is an important risk factor in pressure sore development. It is likely that incontinence is strongly associated with both immobility and age and these factors emerge as more important in a multi-variate model.

### *2.6.3 Skin Response to Pressure*

Finally, skin condition is identified as a risk factor associated with subsequent skin loss by all 5 studies which included this variable as a potential risk factor. It is an aspect of risk of increasing interest and is discussed more fully in Chapter 6 (Section 6.6).

## 2.7 Summary

A review of the evidence suggests that nursing assessment is a dynamic and continuous process involving synthesis of information from a variety of sources including underpinning knowledge, previous experience, specialty based knowledge, recognition of important indicators and knowledge of the patient. Nursing skill is required in order to select, weight and combine important factors in order to notice, understand and act.

It is essential from the outset to identify which patients require baseline skin assessment in order to determine the presence or absence of existing pressure sores. Subsequent assessment of individual patient risk is an ongoing and dynamic process supported by knowledge of key prognostic factors (in particular mobility) the patients individual skin response to pressure and prognosis, specialty based knowledge and clinical experience.

Risk assessment scales whilst limited in construction methods and validity may provide a framework and appropriate prompts for assessment of pressure sore risk but their use as a single instrument to assess risk is not supported on the basis of

current evidence. The research need is not validation of existing risk assessment scales, but identification of key prognostic factors using statistical modeling to develop predictive tools or frameworks which support assessment processes.

A review of prognostic factor research identifies important themes and key factors in the prediction of pressure sore development including reduced mobility, nutrition, perfusion, age and skin condition. These can be directly related to the aetiology of pressure sore development where the interaction between the intensity and duration of pressure (mobility) and the tolerance of the skin (nutrition, perfusion and age) determines the skin response (skin condition) and provide a framework for further investigation.

# Chapter 3 Research Design and Statistical Method

## 3.1 Introduction

Following the literature review and exploratory study (Chapters 1 and 2) outstanding practice issues remained in relation to pre-operative screening for pressure sores, peri-operative risk assessment and provision of effective intra-operative interventions. This chapter outlines the research methodology adopted in order to address the following research questions:

What are the benefits of using an intra-operative intervention in relation to post-operative pressure sore incidence?
Which variables are associated with post-operative pressure sore development?
What is the extent of pre-operative damage to the skin?

## 3.2 Treatment Intervention

The dry visco-elastic polymer pad (Action Inc.) was chosen as the intervention on the basis of previous laboratory evidence suggesting reduced interface pressures[48], ease of practical application in direct contact with the skin, intra-operative stability, ease of cleaning and cost.

Patients assigned to the intervention group were allocated a dry visco-elastic polymer pad for the torso (sacral and buttock areas) and heels which were positioned in direct contact with the patient's skin. Patients assigned to the control group were positioned in the normal way on the operating table with sacral and buttock areas in direct contact with the carrying canvas. Heel supports were standardised to a gamgee pad across the research sites

Intra-operative warming mattress provision (JMW Systems Ltd) was standardised for both treatment and control groups across both research centres.

## 3.3 Research Objectives

### *3.3.1 Primary Objective.*

a. to compare the post-operative pressure sore incidence in patients positioned on a standard operating table mattress with those positioned on a dry visco-elastic polymer pad.

### *3.3.2 Secondary Objectives*

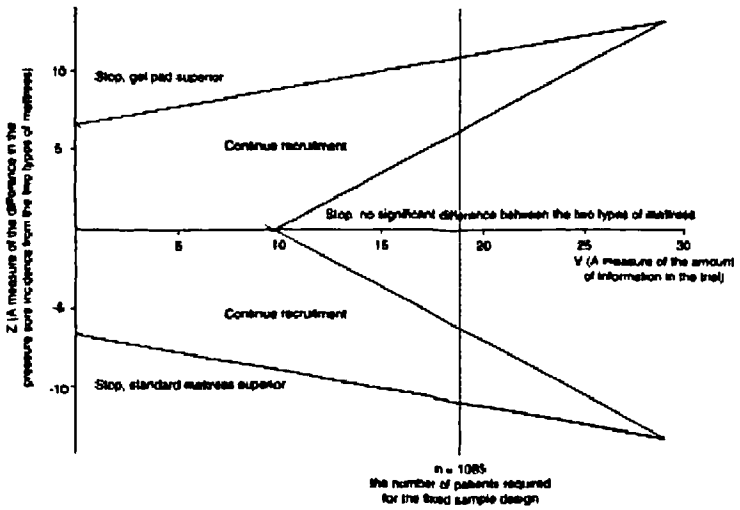
b. to investigate the variables which most significantly contribute to post-operative pressure sore development

c. establish the pre-operative pressure sore prevalence.

## 3.4 Study Design

A sequential double triangular design[92] was chosen for this randomised, double blind, controlled trial of the intra-operative use of a dry visco-elastic polymer pad (Figure 3.1). The sequential design provides a mechanism for stopping the clinical trial early if the superiority of the dry visco-elastic polymer pad or the standard operating table mattress become apparent as the data accumulates.

**Figure 3. 1 Illustration of the Double Triangular Sequential Design**



The trial was designed to detect an absolute difference in the incidence of theatre-generated pressure sores from 10% on the standard mattress to 5% on the dry polymer pad with 90% power at the 5% significance level. Two important issues required consideration in statistical design and informed the choice of the sequential design[92] as follows:

1. There was a difficulty in determining sample size in advance of the study due to the large degree of uncertainty in the expected incidence of pressure sores in both arms of the trial[12;25;45;49].
2. There was a need to determine the relative effectiveness of the dry polymer pad as quickly as possible because it was already in use as a preventative intervention.

### *3.4.1 Sample*

The sample comprised surgical in-patients from St. James's University Hospital, Leeds and The General Hospital, Hartlepool.

Based on an estimated difference in incidence 10% (standard) to 5% (dry polymer) simulated results indicated that between 500 and 1,000 patients would be required. A fixed sample design with the same assumptions would have required a total of 1085 patients to be recruited. Also the double triangular sequential design was specifically chosen so that:

1. Inferiority of the dry polymer visco-elastic polymer pad would be distinguishable from a lack of difference between the two mattress types.

2. There would be early stopping under the null hypothesis of no difference between the treatments. If no difference exists between the two mattress types, then simulated sample size calculations indicated that the stopping boundary would be reached between 500 and 750 patients.

### *3.4.2 Inclusion Criteria*

a. scheduled for elective major general, gynaecological or vascular surgery
b. aged 55 years or over on day of surgery
c. scheduled to undergo 'major' surgery
d. intra-operative position to be supine or lithotomy

Major surgery was defined as a planned surgical procedure with an average surgical time of 90 minutes or more.

### *3.4.3 Exclusion Criteria*

a. general surgery sub-specialities including liver, urology and breast surgery

b. pressure damage of Grade 2a or above observed pre-operatively.

c. ward staff provision of pre-operative alternating pressure mattress

d. dark skin pigmentation which preclude reliable identification of Grade 1 and grade 2a skin assessments.

e. skin conditions over the sacrum, buttocks or heels which preclude reliable identification of Grade 1 and Grade 2a skin assessments.

### *3.4.4 Randomisation*

Following inclusion to the study patients were randomised to either the standard operating table mattress or the dry visco-elastic polymer pad. Randomisation was stratified by hospital (Hartlepool, Leeds) and age (55-69 and 70 or over). A telephone randomisation schedule was developed within random permuted blocks of 6, with a run in of 8, and managed by the Yorkshire Clinical Trials and Research Unit.

### *3.4.5 Blinding*

A research nurse recorded all pre and intra-operative data and removed the dry visco-elastic polymer pad prior to transfer to the Post Anaesthetic Care Unit (PACU). The record pertaining to the intra-operative randomised mattress allocation remained separate from the main data collection proforma to ensure that post-operative skin assessments were blind to the mattress allocation.

The PACU and ward staff recorded all post-operative skin assessments and were blind to the intra-operative mattress allocation.

### *3.4.6 Data Monitoring*

Independent interim analyses were conducted after recruitment of the first 200 patients and subsequently after every 100 patients recruited. Results were presented to an independent Data Monitoring Committee (DMC) which was responsible for recommending when the trial should be stopped. The DMC and statistician were blind to treatment allocation during the course of this trial.

## 3.5 Ethical Considerations

Ethical considerations required consultation and compromise in the clinical research sites. The need for informed pre-operative consent and the prospective nature of the data collection dictated the inclusion of elective surgical patients.

Also of importance was that dry visco-elastic polymer pads were in use within the St. James's site on an ad hoc basis and this influenced design elements in two ways. Firstly, it was one of the criteria which determined the selection of the double triangular sequential design, as detailed below. Secondly, patients were excluded if pre-operative ward care included the provision of an alternating pressure mattress.

## 3.6 Outcome Criteria

Skin was assessed using an adapted version of the Torrance scale[19] whereby Stage 2 was sub-divided to enable the distinction between intact and broken skin as detailed in Table 3.1. Also included was 'Grade 0' (no discolouration of the skin) to clearly distinguish between assessment of normal skin and missing data.

**Table 3. 1 Skin Classification Scale (Adapted from Torrance 1983[19])**

| Skin Grade | Description of skin |
|---|---|
| 0 | No skin discolouration |
| 1 | Redness to the skin - blanching occurs |
| 2a | Redness to the skin - non-blanching area |
| 2b | Superficial damage to epidermis |
| 3 | Ulceration progressed through the dermis |
| 4 | Ulceration extended into subcutaneous fat |
| 5 | Necrosis penetrating the deep fascia and extending to muscle |

The main endpoint for the trial was determined as a success - no pressure sore, or failure - pressure sore (Table 3.2) at any of the five skin sites most likely to incur skin damage (sacrum, left and right buttocks, and left and right heels). The primary endpoint was established using the definition of a pressure sore as 'a persistent

discolouration of the same skin site on two or more successive days', a definition adapted from those used by previous researchers[12-14;22]. A particular feature of the end point definition was the specificity of persistent worsening of the skin condition from its pre-operative condition to that post-operatively for three successive assessments.

Supplementary skin assessment data was also collected up to post-operative Day 8 (or discharge) to exclude the occurrence of Barton and Barton[16]Type II pressure sores (see Section 2.2).

**Table 3. 2 Primary Endpoint Definition**

| Assessment time | | | | |
|---|---|---|---|---|
| Immediate preanaesthetic<br><br>Skin grade | Immediate (up to 1/2 hour) post operation<br><br>Skin grade | 1/2- 1 hour following immediate assessment<br>Skin grade | Day 1 post operation (8 am - 8 pm)<br><br>Skin grade | Site-specific outcome |
| 0 | 0 | any grade | any grade | Success |
| 0 | >1 | 1 | >1 | Failure |
| 0 | >1 | >1 | 0 | Success |
| 0 | >1 | 0 | any grade | Success |
| 1 | 0 | any grade | any grade | Success |
| 1 | 1 | any grade | any grade | Success |
| 1 | >2a | >2a | >2a | Failure |
| 1 | >2a | >2a | 0 or 1 | Success |
| 1 | >2a | 0 or 1 | any grade | Success |

## 3.7 Statistical Method: Randomised Controlled Trial

Sequential analysis was performed using the odds ratio formulation of the double triangular design with the binary outcome of success and failure as defined in Table 3.2. The null hypothesis was no difference in post-operative pressure sore incidence whether the patients were assigned a dry polymer pad during their operation or standard foam operating table mattress.

Figure 3.1 illustrates the trial design where Z measures the cumulated evidence of the difference in the incidence of pressure sores on the two treatment arms of the trial, and V indicates the amount of information contained in the data about the treatment effect. The sample statistics, denoted by Z and V were computed and plotted at each interim analysis. $\theta$ is the odds of developing a pressure sore on a dry polymer pad compared with a standard operating table mattress and is the measure of the difference between the treatments.

Mathematically the effect of the dry visco-elastic polymer pad is given by

$$\theta = \frac{P_G (1 - P_S)}{P_S (1 - P_G)}$$

where $P_S$ is the proportion of patients allocated a standard mattress developing a pressure sore and $P_G$ is the proportion of patients allocated a dry visco-elastic polymer pad developing a pressure sore, such that

$\theta > 1$ if the dry polymer pad is inferior,

$\theta = 1$ if they are equivalent, and

$\theta < 1$ if the dry polymer pad is superior.

The statistics Z and V are given by:

$$Z = (n_S F_G - n_G F_S) / n$$

$$V = \frac{n_G n_S SF}{n^3}$$

where $n_S$ = the number of patients allocated to the standard mattress with endpoint recorded

$n_G$ = the number of patients allocated to the dry polymer pad with endpoint recorded
$F_S$ = the number of patients developing a pressure sore on the standard mattress
$F_G$ = the number of patients developing a pressure sore on the dry polymer pad
$F = F_S + F_G$
$n = n_S + n_G$
and $S = n - F$.

It was expected that the mattress effect at each of these interim analyses would be adjusted for the strata used in the randomisation allocation rule. The four other *a priori* important variables to be accounted for in the event of crossing a stopping boundary were type of operation (vascular, non-vascular), length of operation, proportion of time hypotensive and pre-operative length of stay[12;25;45;49]. The boundaries used for an inspection at an interim analysis were adjusted for the limited number of analyses occurring at discrete time points. This adjustment over a series of interim analyses resulted in narrower stopping boundaries which take the form of a 'Christmas tree' shape. The theory of the double triangular sequential design provided a method of calculating adjusted unbiased estimates of the probability of developing a pressure sore on the two types of mattress when a stopping boundary had been crossed.

All statistical analyses for the trial were carried out on 'intention to treat' basis using Planning and Evaluation of Sequential Trial Software (University of Reading), Version 3 (PEST3) and Statistical Analysis Software, SAS Institute Inc (SAS) software packages. The stratified analysis of treatment effect in this trial was carried out using the overrunning analysis option. The covariate adjustment analysis was carried out using the logistic regression and interactive matrix language procedures in SAS and the overrunning analysis option in PEST3. Terms were only included in the logistic model if their associated chi-square p-value was less than 0.05. Models were chosen by forward stepwise regression and by backward elimination to see if the same statistical model could be established using both methods. These methods involve respectively including the most, and excluding the least, statistically significant terms in the model in a step by step manner. Interactions with treatment were examined as well as main effects.

## 3.8 Reliability

Previous research has demonstrated poor inter-rater reliability between clinical and research staff in the assessment of blanching erythema[15] and in the classification of skin changes and pressure sores[93].

During the 3 month study set-up time training was provided to ward, recovery and intensive care staff involved in the study and the inter-rater reliability of the skin assessment tool was assessed between clinical staff and the research nurses and between the research nurses at the different hospitals prior to the study start date.

Inter-rater reliability and its effect on the validity of the main endpoint was also assessed during the course of the study by independent co-assessment. Completeness of data was subject to nursing staff availability.

## 3.9 Statistical Method: Prognostic Factor Analysis

***Univariate Analysis.*** Variables were entered into a linear logistic regression model with a binary response of pressure sore or no pressure sore. Variables were included in the analysis if >75% of data were present. Since type of mattress was found to significantly effect pressure sore development (section 4.3) mattress effect was first fitted into the logistic model and then the remaining variables were entered into the logistic regression model using forward and backward elimination. Variables which resulted in a p value of <0.01 were included for further analysis.

***Correlation of Variables.*** Correlations between variables with >75% of data present, were examined using Pearson's correlation coefficient for continuous data or Spearmans rank correlation for ordered categorical data. Where variables were correlated with a correlation coefficient of >0.7 and an associated p-value of <0.01[94] one was eliminated from further consideration on the basis of clinical experience.

***Multivariate Modeling***. The remaining variables were then entered into a logistic regression model with adjustment for type of mattress. The model was derived both by forward selection and backward elimination. A significance level of 0.05 was used for the chi-squared statistic associated with selection or dropping a term in the model.

The model was determined only from patients with complete data for all candidate variables. Therefore, when the final set of variables was obtained the model was refitted with only those final variables in the model statement. This ensures a maximum data set for the variables within the model.

The forward and backward regression model fitting procedures were repeated just using patients assigned to the standard arm of the trial to see if the same model was obtained. The robustness of the final model was further examined by excluding all observations with an associated deviance residual of greater than 2.58 ($p = 0.01$) (that is, outlying subjects). The model fitting procedure was repeated to determine whether the same set of selected variables was obtained.

Variables included in the analysis were calculated as follows: pre-operative immobility time was the time from pre-medication or arrival in pre-waiting to start time of anaesthetic; length of operation was from the start of anaesthesia to the time lifted from the operating table; total immobility time was the sum of pre-operative immobility time and length of operation; blood pressure was recorded intra-operatively at 10 minute intervals and a hypotensive episode was determined when a diastolic blood pressure of less than 60 mm Hg was recorded on two successive occasions.

All statistical analyses were carried out using the LOGISTIC procedure of the SAS statistical software package.

# Chapter 4 Results

## 4.1 Introduction

Patients were recruited from November 1994 to June 1996 when results reached a stopping boundary for the primary research question (Figure 4.1). This chapter outlines the main findings of the study.

## 4.2 Sample

Of the 720 patients potentially eligible for inclusion in the trial 274 (38%) were excluded. Reasons for the high attrition rate are detailed in Table 4.1.

446 patients were randomised into the trial, 222 to the dry visco-elastic polymer pad group and 224 to the standard mattress (Table 4.1). The main endpoint was determined for 416 patients, with incomplete data for 30 patients resulting from lost forms (3) and incomplete post-operative skin assessment records (27).

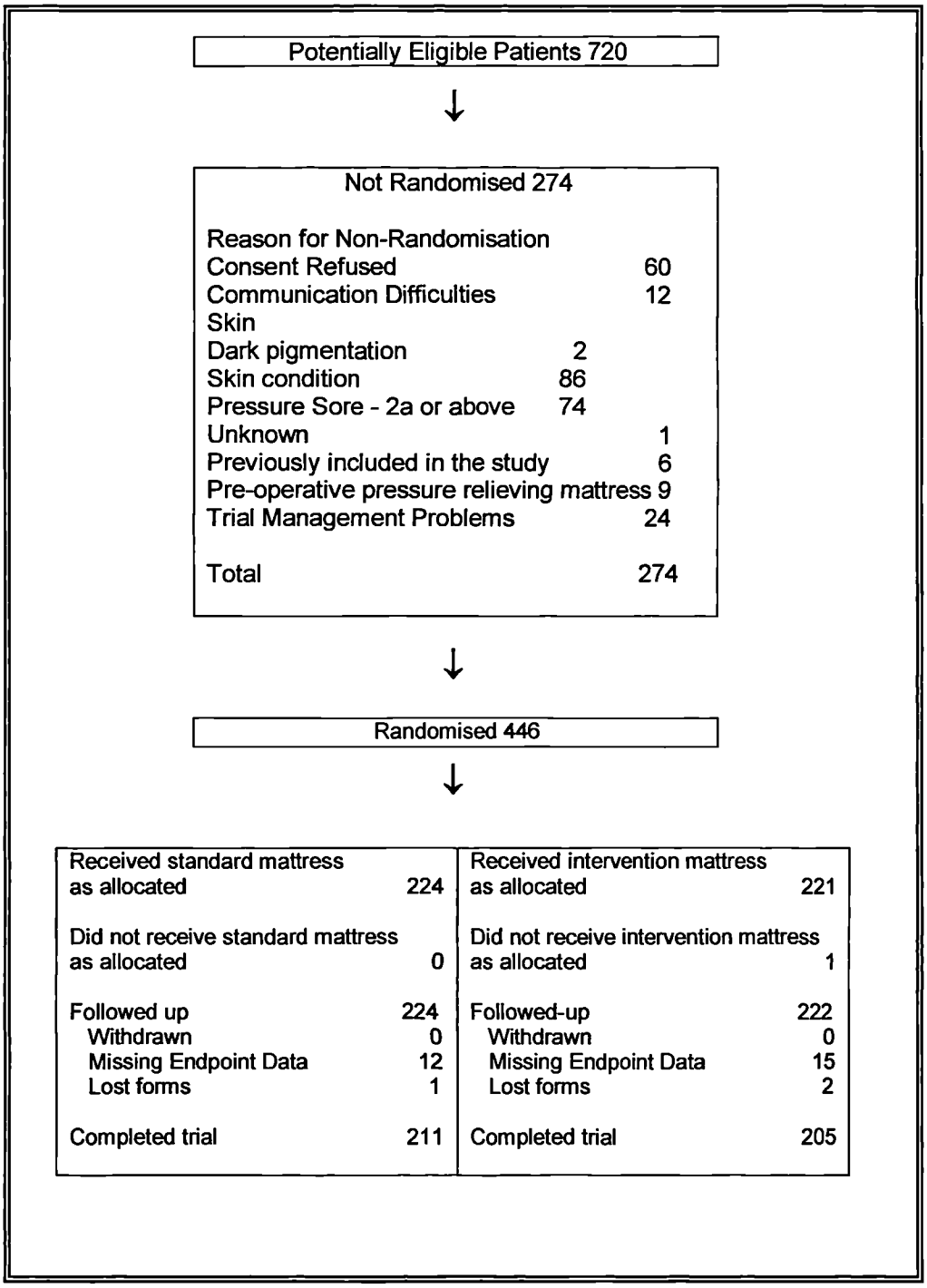
## 4.3 Outcome

Using the outcome criteria detailed in Table 3.2, a post-operative pressure sore incidence of 15.6% (65/416) was determined. The majority of endpoint failures were skin changes from Grade 0 pre-operatively to Grade 1 post-operatively (56/65) with 4 patients having Grade 0 - 2a pressure sores and 4 patients Grade 0 - 2b pressure sores and 1 patient a Grade 1 – 2b pressure sore. Pressure sores were observed on a total of 95 skin sites on sacral (39), buttock (40) and heel (16) areas.

Of the 56 Grade 0 - 1 pressure sores, only 20 had resolved by post-operative day 2. Of the remaining 36, 33 continued to have persistent blanching hyperaemia for varying periods up to Day 8 post-operatively, 2 persisted and subsequently deteriorated to 2a and 1 persisted and deteriorated to 2b. Of the 4 Grade 2a pressure sores all remained as either blanching or non-blanching up to Day 8. Of the 5 Grade 2b pressure sores, 2 resolved within 24 hours to Grade 1 which then persisted for

varying periods up to Day 8, 1 resolved to 2a within 24 hours which persisted to Day 8, and 2 persisted as 2b up to Day 8.

Table 4. 1 Trial Profile



Post-operative skin assessments up to Day 8 identified a total of 35 patients with Grade 2a and 21 patients with Grade 2b pressure sores (Table 4.2), an incidence of 12.6% (56/443), of which 16% (9/56) were related to the trial end-point.

**Table 4. 2 Grade 2 Pressure Sores up to Day 8 Post-operatively**

| | Hartlepool n=133 | | St.James's n=310 | | Total | |
|---|---|---|---|---|---|---|
| Grade 2a | 13 | 9.7% | 22 | 7% | 35 | 7.9% |
| Grade 2b | 4 | 3% | 17 | 5.5% | 21 | 4.7% |
| Total | 17 | 12.8% | 39 | 12.6% | 56 | 12.5% |

## 4.4 Randomised Controlled Trial

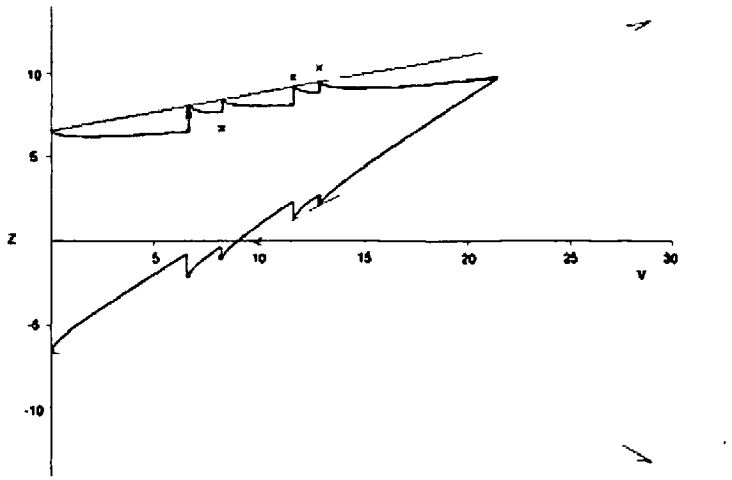
Pressure sore incidence was 11% (22/205) for patients allocated to the dry polymer pad and 20% (43/211) for patients allocated to the standard operating table mattress (Table 4.3). The sample statistics, denoted by Z and V were computed and plotted at each interim analysis (Figure 4.1). The median unbiased estimate of treatment effect, based on the odds of developing a pressure sore after being placed on the dry polymer pad during surgery compared with being placed on a standard operating table mattress, was estimated after adjustment for centre and age group. There was a significant reduction of pressure sores on the dry polymer pad as compared with the standard mattress, $\theta = 0.46$ with 95 per cent confidence interval of (0.26, 0.82), $p = 0.010$. The adjusted point estimates of the probability of developing a pressure sore on the dry polymer gel pad and the standard operating table mattress were 0.11 and 0.21 respectively.

**Table 4. 3 Trial Endpoint by Centre and Mattress Allocation**

| | Hartlepool | | Leeds | | |
|---|---|---|---|---|---|
| Trial endpoint | Polymer Pad | Standard | Polymer Pad | Standard | Total |
| Failure | 3 | 0 | 19 | 43 | 65 |
| Success | 59 | 67 | 124 | 101 | 351 |
| Undetermined | 4 | 0 | 13 | 13 | 30 |
| Total | 66 | 67 | 156 | 157 | 446 |

Table 4.4. reports baseline characteristics and variables thought *a priori* to affect outcome. At each centre and for both age groups, the treatment allocation was evenly balanced and the proportion of patients undergoing vascular surgery on the two types of mattress was similar. There was a tendency for patients assigned to the standard operating table mattress to have slightly longer length of operation, longer pre-operative stay and proportionally more time in a hypotensive state than patients assigned to the dry polymer pad.

**Figure 4. 1 The Difference in Pressure Sore Incidence for the two Mattresses**




A sensitivity analysis was carried out assuming that the patients with missing endpoints had developed pressure sores. The odds of pressure sore development on the dry polymer pad compared with the standard operating table mattress was $\theta$ = 0.62, 95% confidence interval (0.39, 1.00), p = 0.048, which is consistent in the sense that the difference in the effects of the two types of mattress is in the same direction as that in the main analysis.

**Table 4. 4 Baseline Variables of Treatment and Control Groups**

| Variable | Levels | Polymer Pad (total = 222) | Standard mattress (total = 224) |
|---|---|---|---|
| Centre | Hartlepool<br>Leeds | 66 (30%)<br>156 (70%) | 67 (30%)<br>157 (70%) |
| Age group | 55-69<br>70+ | 124 (56%)<br>98 (44%) | 128 (57%)<br>96 (43%) |
| Gender | Male<br>Female<br>Not known (forms lost) | 119 (54%)<br>101 (45%)<br>2 (1%) | 116 (52%)<br>107 (48%)<br>1 (0%) |
| Preoperative Braden Scale (Braden and Bergstrom 1988) | 10-14<br>15-19<br>20-23<br>Not known (forms lost) | 1 (0%)<br>17 (8%)<br>202 (91%)<br>2 (1%) | 0 (0%)<br>23 (10%)<br>200 (89%)<br>1 (0%) |
| Type of surgery | Vascular<br>Non-vascular<br>Unknown (forms lost) | 69 (31%)<br>151 (68%)<br>2 (1%) | 70 (31%)<br>153 (68%)<br>1 (0%) |
| Length of operation | Less than 90 minutes<br>90-179 minutes<br>180 minutes or longer<br>Unknown (forms lost) | 50 (23%)<br>108 (49%)<br>62 (28%)<br>2 (1%) | 40 (18%)<br>110 (49%)<br>73 (33%)<br>1 (0%) |
| Length of preoperative hospital stay | 0-1 days<br>2-4 days<br>5 days or more<br>Unknown (forms lost) | 107 (48%)<br>62 (28%)<br>51 (23%)<br>2 (1%) | 89 (40%)<br>74 (33%)<br>60 (27%)<br>1 (0%) |
| Proportion of time hypotensive during operation | None<br>1% - 24%<br>25% - 49%<br>50% - 74%<br>75% - 100%<br>Unknown (forms lost) | 107 (48%)<br>48 (22%)<br>26 (12%)<br>24 (11%)<br>15 (7%)<br>2 (1%) | 94 (42%)<br>56 (25%)<br>35 (16%)<br>25 (11%)<br>13 (6%)<br>1 (0%) |

The effect of the variables thought *a priori* to be important including centre, age, type of surgery, length of operation, length of pre-operative stay in centre, and proportion of time the patient was in a hypotensive state during surgery, were examined using logistic regression to see if they modified the estimate of the difference in the incidence of pressure sores between the two mattress types. The same model was obtained by using forward selection and backward elimination.

In order of descending importance, the following variables were significant in modifying the estimate of the difference in the probability of developing a pressure sore on the two mattress types:

- CENT - Centre (Hartlepool = 1, Leeds = 0)
- OPLN - Length of operation in minutes
- HYPO - Proportion of time the patient was in a hypotensive state
- STAY - Length of preoperative stay in centre in days

Age and type of surgery were found not to be important in the presence of the other variables. Age was not found to be important even when included in the model on its own. The model for response with the standard errors of the parameter estimates in parentheses, based on 416 patients with determined endpoints, was given by

$$\eta = \underset{}{-2.50} \underset{(0.34)}{-2.26\,CENT} + \underset{(0.62)}{1.26\,HYPO} + \underset{(0.49)}{0.00415\,OPLN} + \underset{(0.0016)}{0.0309\,STAY} \underset{(0.015)}{}$$

where the probability that a patient will develop a pressure sore is given by

$$p = 1 / (1 + e^{-\eta})$$

Thus, the probability that a patient will develop a pressure sore, according to the definition of this trial, was higher at Leeds than Hartlepool, increased with the proportion of time in a hypotensive state during their operation, longer length of operation and longer preoperative centre stay.

The mean proportion of time in a hypotensive state for patients in this trial was 19.2%, mean length of operation was 155.2 minutes, and mean length of preoperative stay in centre was 4.7 days. Hence at Hartlepool, the probability of a patient who had these characteristics of developing a pressure sore was p= 0.02 and for a similar patient at St. James's the probability was p= 0.19.

The median unbiased estimate of treatment effect after adjustment for centre and the covariates proportion of time hypotensive, length of operation and length of preoperative stay, was $\theta = 0.5$ with a 95% confidence interval (0.27, 0.89), $p = 0.020$. The effect of the dry polymer pad was slightly modified but outcomes remained statistically significantly different. The covariate adjusted value of the mattress effect

can be interpreted by imagining a patient with the probability of developing a pressure sore after being placed on a standard mattress for their operation of p = 0.20 (the observed incidence). The estimated probability of developing a pressure sore on the dry polymer pad for such a patient is p = 0.11 with a 95% confidence interval of (0.07, 0.19).

The covariates which were important in modifying the treatment effect were examined for interaction effect with the mattress. Treatment centre was found to be significant, p = 0.012.

## 4.5 Reliability and Validity

A total of 133 paired assessments were undertaken by 94 nurses for the pre-study inter-rater reliability assessments generating data for 664 skin sites. There was disagreement for 15/664 (2.2%) skin sites, affecting 12/133 (9%) patients. Disagreements were mainly '0-1' (13) with 2 disagreements for '1-2a'. A majority of the disagreements were associated with assessment of heels (10/12 patients).

A total of 171 co-assessments were undertaken in the recovery area (105) and ward (65), generating a total of 851 site comparisons between the main trial assessment and co-assessment. Of these there was discrepancy of 72/851 (8.5%). All discrepant co-assessments were only one grade on the skin assessment scale with 68 0/1 and 4 1/2a disagreements.

Despite the overall number of discrepancies the number of misclassifications of success or failure which would have resulted had the co-assessments been used for determination of the main endpoint rather than the main trial assessments would have been 5 (3 successes would have been failures, 1 failure would have been a success and 1 success would have been undetermined). Incorporating these altered endpoints into a sensitivity analysis of the treatment effect results in a median unbiased estimate for $\theta = 0.50$ with 95% confidence interval (0.29, 0.88), p = 0.016. Therefore the significant difference in the effect of the two mattress types remains.

## 4.6 Prognostic Factor Analysis

***Univariate analysis.*** The 49 variables explored by univariate analysis are detailed in Table 4.5. Other variables with >25% missing data were not investigated in the analysis (neutrophils, monocytes, leucocytes and blood loss) nor were blood measurements which displayed a majority of readings at the limit of detection (basophils and eosinophils). The variables with an associated p value of <0.01 are detailed in Table 4.6.

**Table 4. 5 Variables Explored by Univariate Analysis**

| | |
|---|---|
| General -<br>Age<br>Gender<br>Type of surgery<br>Type of anaesthetic<br>Position during surgery<br><br>Mobility -<br>Length of pre-operative stay<br>Pre-operative Immobility Time<br>Length of Operation<br>Total immobility time<br>Post-operative immobility<br><br>Braden Scale -<br>Pre-operative Braden Total<br>Pre-operative Braden subscales (6)<br>Post-operative Braden Total<br>Post-operative Braden subscales (6)<br><br>Equipment -<br>Pre-operative mattress<br>Recovery room mattress<br>Ward mattress | Pre-operative Physiological Measures -<br>Haemoglobin<br>Total White Cell Count<br>Lymphocytes<br>Platelets<br>Albumin<br>Total Plasma Proteins<br>Sodium<br>Potassium<br>Urea<br>Creatinine<br>History of Weight Loss<br>Body Mass Index<br>Temperature<br>Starvation Time<br>Pre-operative systolic BP<br><br>Pre-operative systolic BP<br>Intra-operative Physiological Measures -<br>Core and peripheral Temperature (minimum and mean)<br>Number of Hypotensive episodes<br>Proportion in hypotensive state (%) |

**Correlations**. Correlation between variables detailed in Table 4.6 are described in Table 4.7. The number of hypotensive episodes was selected because it is easier to obtain in clinical practice. Of the 4 Braden Scale variables, Day 1 Braden Mobility Scale was selected as this can be more easily predicted peri-operatively than either the Day 1 Total score or other subscales. With respect to intra-operative core temperature the mean value was selected as it is a better measure of intra-operative

temperature compared with minimum temperatures which relate to the start and end of surgery.

**Table 4. 6 Variables Associated with Pressure Sore Occurrence.**

| Variable | Chi-squared value | p-value |
|---|---|---|
| Pre-operative Immobility time | 24.65 | <0.001 |
| Number of hypotensive episodes | 23.05 | <0.001 |
| Day 1 Braden Scale Activity | 22.26 | <0.001 |
| Proportion of time hypotensive | 21.43 | <0.001 |
| Day 1 Braden Scale Mobility | 20.81 | <0.001 |
| Day 1 Braden Scale Total | 15.48 | <0.001 |
| Type of surgery (vascular, non-vascular) | 12.61 | <0.001 |
| Day 1 Braden Scale Friction and Shear | 12.07 | 0.001 |
| Mean core temperature | 10.87 | 0.001 |
| Length of operation | 8.08 | 0.004 |
| Minimum peripheral temperature | 7.74 | 0.005 |
| Minimum core temperature | 7.73 | 0.005 |
| Length of preoperative hospital stay in days | 6.68 | 0.010 |

**Table 4. 7 Significantly Correlated Variables**

| Correlated variables | | Correlation coefficient | p-value |
|---|---|---|---|
| Day 1 Braden Scale | Mobility v Total | 0.87 | <0.001 |
| Hypotension | No episodes v Proportion time | 0.84 | <0.001 |
| Day 1 Braden Scale | Total v Activity | 0.84 | <0.001 |
| Day 1 Braden Scale | Total v Friction and Shear | 0.83 | <0.001 |
| Day 1 Braden Scale | Mobility v Friction and Shear | 0.74 | <0.001 |
| Day 1 Braden Scale | Mobility v Activity | 0.71 | <0.001 |
| Core Temperature | Mean v minimum | 0.73 | <0.001 |

**Multi-variate Modeling.** The final set of candidate variables for multivariate analysis of prognostic factors with adjustment for type of intra-operative mattress (MATT) are detailed in Tables 4.8. Multivariate examination identified 4 prognostic variables including, the number of hypotensive episodes (HYPO), pre-operative immobility time (IMMT), Day 1 Braden Mobility Scale (MOB1) and mean core temperature (MECT). The same 4 variables were obtained by using both forward and backward elimination methods.

The model chosen with parameter estimates shown along with their standard errors in parentheses was:

$\eta$ = - 19.71 - 0.84 MATT + 0.06 HYPO - 0.01 IMMT - 0.67 MOB1 + 0.55 MECT

(8.81) (0.33) (0.02) (0.004) (0.23) (0.24)

where the probability of developing a pressure sore was given by:

$p = 1 / ( 1 + e^{-\eta})$

**Table 4. 8 Final Candidate Variables for Multivariate Examination - Variable Parameters**

| Variable | Median | Range | Mean |
|---|---|---|---|
| Pre-operative immobility time (IMMT) | 40mins | 0 - 270 | 66.2mins |
| Type of Surgery (SURG) | 2 | 1-2 | |
| Number of hypotensive episodes (HYPO) | 1 | 0 - 46 | 3.6 |
| Day 1 Braden Mobility Scale (MOB1) | 3 | 1-4 | |
| Mean core temperature (MECT) | 35.8°C | 33.0-37.9 | 35.8°C |
| Length of operation (OPLN) | 140 mins | 35-552 | 157.8 mins. |
| Minimum peripheral temperature (MNPT) | 30.5°C | 21-35 | 30.2°C |
| Length of pre-operative stay (STAY) | 2 | 0 - 119 | 4.7 |

Thus the probability of a patient developing a pressure sore arising from elective major surgery was associated with: increased number of hypotensive episodes and mean core temperature during surgery; reduced mobility Day 1 post-operatively and; shorter length of pre-operative immobility.

Repeating the model selection procedure just for patients assigned to a standard mattress during surgery, produced the following model:

$\eta$ = -27.15 + 0.08 HYPO - 0.02 IMMT - 0.71 MOB1 + 0.76 MECT

(11.54) (0.03) (0.006) (0.29) (0.32)

Since the final model showed the probability of developing a pressure sore increased with decreasing length of pre-operative immobility which is not as would be clinically expected *a priori* the above analyses were repeated without pre-operative immobility time in the multi-variate procedures to ascertain whether other variables might be identified as important. No additional variables in the final model were identified as important predictors in this analysis and the values of the coefficients for the factors remained very similar.

$\eta$ = - 25.3 - 0.77 MATT + 0.07 HYPO - 0.68 MOB1 + 0.70 MECT

(8.34) (0.32) (0.02) (0.23) (0.23)

Similarly, repeating the model selection procedure just for patients assigned to a standard mattress during surgery, produced the following model:

$\eta$ = -31.07 + 0.08 HYPO - 0.72 MOB1 + 0.84 MECT

(10.81) (0.03) (0.28) (0.30)

Sample calculations using hypothetical patient scenarios illustrate differences in the probability of post-operative pressure sore development (see Table 4.9).

**Table 4. 9 Illustrative Examples: Probability of Post-operative Pressure Sore Development**

Probability Equation

$\eta = -25.3 - 0.77\ MATT + 0.07\ HYPO - 0.68\ MOB1 + 0.7\ MECT$

$p = 1 / (1 + e^{-\eta})$

| | | |
|---|---|---|
| Patient 1: | Dry visco-elastic polymer mattress | 1 |
| | Number of hypotensive episodes | 10 |
| | Day 1 Braden Mobility Scale | 1 |
| | Mean core temperature | 36.8°C |

$\eta = -0.29$

$p = 0.43$ or 43% probability of pressure sore development

| | | |
|---|---|---|
| Patient 2: | Standard mattress assigned | 0 |
| | Number of hypotensive episodes | 10 |
| | Day 1 Braden Mobility Scale | 1 |
| | Mean core temperature | 36.8°C |

$\eta = 0.48$

$p = 0.62$ or 62% probability of pressure sore development

| | | |
|---|---|---|
| Patient 3: | Dry visco-elastic polymer mattress | 1 |
| | Number of hypotensive episodes | 2 |
| | Day 1 Braden Mobility Scale | 3 |
| | Mean core temperature | 35.5°C |

$\eta = -3.12$

$p = 0.0422$ or 4.22% probability of pressure sore development

| | | |
|---|---|---|
| Patient 4: | Standard mattress assigned | 0 |
| | Number of hypotensive episodes | 2 |
| | Day 1 Braden Mobility Scale | 3 |
| | Mean core temperature | 35.5°C |

$\eta = -2.35$

$p = 0.087$ or 8.7% probability of pressure sore development

The model fitting procedures were repeated with substitution of highly correlated variables. Day 1 Braden Scale Activity rather than Mobility resulted in the same other variables selected for inclusion the final model. Similarly, repeating the model fitting procedure with proportion of time hypotensive rather than number of hypotensive episodes, resulted in selection of the same variables other than mean core temperature. Making both of these changes simultaneously resulted in the same other variables (including mean core temperature) being included in the model. Re-examination of the model without outliers (only two observations had large deviance residuals) resulted in the same set of variables selected in the model.

## 4.7 Pre-operative Prevalence

Of the 720 patients potentially eligible for the study 74 were excluded due to existing pressure damage, a pre-operative prevalence of 10.3%. Grades of pressure sore observed and whether recorded in the nursing or medical notes by ward staff are detailed in Table 4.10.

**Table 4. 10 Pre-operative Pressure Sores by Grade and Accuracy of Records**

| Recorded in notes | Grade 2a | Grade 2b | Grade 3 | Grade 4 | Grade 6 |
|---|---|---|---|---|---|
| Yes | 5 | 12 | 4 | 4 | 1 |
| No | 26 | 21 | 3 | - | - |
| Total | 31 | 33 | 7 | 4 | 1 |

## 4.8 Summary of Results

In this randomised controlled study of 446 patients having vascular, general and gynaecology surgery the use of a dry visco-elastic polymer pad intra-operatively reduced the probability of pressure sore development by half. Although the effect was modified by the variables centre, proportion of time hypotensive, length of surgery and pre-operative length of stay, the effect of the dry visco-elastic polymer pad remained statistically significant. Similarly, in sensitivity analyses accounting for skin assessment variation and undetermined endpoints the effect of the dry visco-elastic pad in reducing post-operative pressure sore incidence remained statistically significant.

Twelve factors were found to be significantly associated with post-operative pressure sore development at the p value of <0.01 and prognostic factors identified by logistic regression modeling included intra-operative hypotensive episodes, Day 1 Braden mobility scale and intra-operative mean core temperature. When Day 1 Braden mobility was replaced by Day 1 Braden activity in the modeling process the same other variables were selected for inclusion in the final model. Similarly, repeating the model selection procedure for patients assigned to the standard arm of the trial identified the same variables.

Of the 720 patients potentially eligible for inclusion in the trial 74 were excluded due to pre-operative pressure damage of Grade 2a or worse representing a pre-operative prevalence of 10.3%. Over half of the observed pressure sores were not documented in nursing records.

# Chapter 5 Discussion

## 5.1 Introduction

The consistency of the research findings compared to previous research, together with the main limitations of the research methodology are discussed in the following sections. There is particular reference to the overall incidence of pressure sores, limitations in the outcome definition of the study, the effectiveness of pressure reducing support surfaces and a discussion of the variables found to be important in determining outcome. Issues pertaining to patient risk assessment are also discussed and the implications of the results to practice and further research determined.

## 5.2 Incidence of Pressure Sores

The overall endpoint failure rate of 15.6% is consistent with findings from other studies of elective surgical patients which report pressure sore incidence rates ranging from 5% - 57.4%[12;25;45;86;87;95;96]. The apparently wide variation in incidence can be accounted for by differences in pressure sore definition, assessment schedules, sampling and exclusion criteria.

Skin grades observed were also consistent with findings from other studies of the post-operative period. There was a predominance of persistent blanching hyperaemia, a small number of Grade 2a and 2b sores and a complete absence of severe progressive sores[12;25;45;86;87;95]. Continued follow up to Day 8 did not identify the delayed appearance of Barton and Barton Type II pressure sores[16].

An absence of severe progressive sores reflects the specificity of the exclusion criteria and the short post-operative follow-up[45]. The paucity of post-operative evidence of Barton and Barton Type II pressure sores that have previously been identified by case study reports [25 26] suggests these are very rare and their occurrence should prompt local investigation.

However, it is noteworthy that patients with existing skin damage, including signs of potentially irreversible changes (non-blanching hyperaemia) were excluded from the study. There is evidence that patients with existing pressure sores are at greater risk of developing further sores than pressure sore free patients [9 6 11]. The sample is further affected by the exclusion of patients with other skin damage, including vascular ulcers, to ensure validity of the skin classification.

## 5.3 Pressure Sore Definition

One criticism, which could be levelled at the present study, is the inclusion of blanching hyperaemia within the endpoint definition of a post-operative pressure sore. Criticism may be waged on two points -firstly, the reliability of skin assessment and it's affect upon the validity of results and secondly the importance of blanching erythema.

With respect to the first point, co-assessments quantified the level of disagreement between assessors as 8.5%. However, the sensitivity analysis determined that disagreement affected classification of only 5 endpoints (2.9%) and did not impact upon the overall difference observed between the two mattresses.

The importance of hyperaemia and other skin changes observed on intact skin has been the source of recent debate[97]. Central to this are questions regarding the relationship between the observation of reactive hyperaemia and subsequent skin/tissue loss. Erythema and other skin changes are included in pressure sore classification systems and definitions for the purposes of research, audit and clinical practice, and are considered to be clinically important[98]. From a physiological perspective erythema whether blanching or non-blanching in a localised area following a pressure assault is a clear indicator that capillary occlusion/partial occlusion has occurred resulting in a reactive hyperaemic response. The endpoint criteria used in this study attempts to distinguish between a normal reactive hyperaemia (transient) response and an abnormal response (persistent skin changes). The validity of the outcome criteria is further supported by the large percentage (45/65, 69%) of patients whose skin was observed to have changes persistent beyond the endpoint period of Day 1 post-operatively. However clinical interpretation of these results must be undertaken with caution until the significance of erythema is established.

Physiological differences between the clinical observations of blanching hyperaemia, non-blanching hyperaemia and non-blanching hyperaemia with associated induration, heat and pain have not been determined in relation to measures of skin perfusion or subsequent skin loss (see Chapter 6). Cumulative research evidence is emerging which suggests that hyperaemia is an identifiable risk factor associated with subsequent skin loss and provides an indicator of the patients individual response to pressure assault and care interventions[86;88;90]. However, questions regarding the validity of the outcome definition and the inclusion of blanching hyperaemia are a limitation of this study.

## 5.4 Randomised Controlled Trial

With respect to the endpoint failure rate for the control and experimental groups, two recent studies include the dry polymer pad within a prescribed treatment regime. Hoshowsky and Schramm[87] reported the results of a trial, comparing the use of standard, foam/gel and dry polymer gel mattresses in the prevention of heel pressure sores. The randomisation method involved each patient receiving a standard mattress for one heel and experimental mattress for the other, each patient providing his/her own control. Interpretation is difficult since incidence is calculated using only one immediate post-operative skin assessment, there is no comparison between pre and post-operative assessments and incidence for each support surface is not detailed, although a statistically significant difference in incidence was reported in favour of the dry polymer pad.

Papantonio et al[95] in a study of 136 patients utilised the dry polymer gel intra-operatively for all patients (elective cardiac surgery). They reported a pressure sore incidence of 27.2% but did not identify exclusion criteria nor distinguish between pre, peri-operative and post-operative incidence.

Results are also consistent with the broader conclusions derived from a systematic review of pressure sore prevention equipment[1]. Using only randomised controlled trials the review concluded that low technology constant pressure supports reduce pressure sore incidence when compared with standard mattress provision. Whilst the standard operating table mattress is not directly comparable to the standard bed mattress the principle that a pressure reducing surface reduces patient pressure sore risk is established.

Four of the six variables considered of *a priori* importance reduced the magnitude of the treatment effect. These included hospital, length of operation, proportion of time hypotensive and length of pre-operative stay.

Unexplained are the differences between hospitals and the low failure rate at Hartlepool (3/129). The differences observed in the immediate post-operative period (trial endpoint) are not mirrored in the continued post-operative follow up to Day 8, where a similar number of Grade 2 pressure sores were observed across both centres (Table 4.2). It is unclear whether patient characteristics (hypotensive episodes, length of surgery and Braden Scores), measurable practices (pre-operative length of stay, post-operative mattress) and/or unmeasured practices (positioning/repositioning) account for the variation within the immediate post-operative period.

The relationship between proportion of time hypotensive and treatment effect is consistent with other research that reports an association between occlusion pressures and systemic blood pressure[99-102]. The maintenance of capillary flow and the 'critical closing pressure' is determined on an individual basis by an interplay of forces including arterial blood pressure[27].

Also, that increased length of surgery modified the mattress treatment effect is consistent with evidence relating to the duration of pressure and pressure sore development[27]. Time is important in two ways. Firstly, there is evidence of low/reduced blood flow in response to external pressure and associated diminished tissue oxygenation[103-105], with suggested risk of gradual development of ischaemic conditions. Secondly, if complete occlusion or ischaemia occurs then time becomes important in determining the extent of tissue damage[27].

The results illustrate that where factors increase the risk of capillary occlusion, the potential benefit of reducing external pressure is modified (or reduced).

A relationship between length of hospital stay and pressure sore incidence has been reported[45;106] and some debate exists as to whether length of stay reflects morbidity and intrinsic risk or hospitalisation and continuous exposure to extrinsic factors such as high pressures[14] or both. If the latter, it suggests that prevention in one patient care area affect the potential benefits in another and results support a prevention policy from admission to discharge.

That age and type of surgery did not modify the treatment effect indicates that the mattress is effective in the prevention of pressure sores during major surgery regardless of the age of the patient and in all three types of surgery.

## 5.5 Prognostic Factor Analysis

With the exception of duration of pre-operative immobility, the 9 final candidate variables identified (Table 4.8) and final logistic regression model are consistent with findings from other studies which have utilised multi-variate analyses (Table 2.1) and reflect the aetiology of pressure sores where the interaction between the intensity and duration of pressure (mobility and length of operation) and the tolerance of the skin (number of hypotensive episodes, mean core temperature and minimum peripheral core temperature) determine pressure sore outcome. The two other variables identified (type of surgery and pre-operative length of stay) are likely to reflect a combination of pressure variables and factors affecting skin tolerance.

With specific reference to surgical studies the variables identified by logistic regression are similar to those identified by Kemp et al[12] and Hoshowsky and Schramm[87]in their prognostic factor analyses (see Table 2.1). Despite differences in the study design and the limited number of variables considered by Kemp et al[12] (6 variables) and Hoshowsky and Schramm[87] (13 variables) the common factors identified provide validity to the results obtained.

Differences in the data collected, in particular temperature variables raise questions about the importance of intra-operative body and skin temperature and confirmatory studies are required in other populations. In this study univariate analysis determined that pressure sore incidence was associated with both increased core temperature and reduced peripheral temperatures. However, multivariate analysis identified core temperature as more important. Results suggest both metabolic and perfusion causality.

The temperature of tissue affects its metabolic demands and early studies report the effects of increased skin temperature on the magnitude of the hyperaemic response[107] and the protection offered by tissue cooling[108]. However, skin temperature is also an indicator of skin perfusion[32], although the relationship is not linear[109]. In the intra-operative period reduced peripheral temperatures reflect vasoconstriction and reduced peripheral perfusion.

Results are limited by local variation in theatre practice and the use of a warm air overblanket (Bairer Hugger) for some major surgical patients. The inclusion of temperature variables were of an exploratory nature and their identification within the prognostic model indicates the need for further research separating the effects of metabolic and perfusion elements.

The important relationship between reduced mobility and pressure sore occurrence suggested by early prevalence surveys are confirmed by cohort studies which identify mobility related factors to be significant and independent predictors of pressure sore development (Table 2.1). Of the 12 studies detailed in Table 2.1, 10 identify mobility related factors as important determinants of pressure sore development. In this study variables identified by univariate analysis included length of operation and Day 1 post-operative Braden Scale subscales of mobility and activity. Model fitting procedures determined mobility and activity as predictors of pressure sore development.

The unexpected finding that increased pre-operative immobility time reduced risk of post-operative pressure damage requires further consideration. Possible explanations for this finding are pre-operative exclusion of patients with existing pressure damage; speciality related differences in pre-medication practice and theatre organisation (effecting trolley wait in theatre waiting area). For example, differences are apparent between type of surgery and median pre-operative immobility times - vascular 25 minutes (5-260), general 50 minutes (range 0-270) and gynaecology 85 minutes (10-265). However, this could just be a chance or spurious finding in this hypothesis generating study. Future studies which include pre-operative immobility time require data relating to patients both with and without existing pressure damage to examine the significance of this finding.

Variables showing little or no relationship with pressure sore development including age, albumin, total plasma protein, body mass index, haematology factors and post-operative mattress allocation require discussion.

Age is one of five themes which emerge from studies utilising multi-variate analyses (Table 2.1) with four of the cohort studies identifying age as associated with pressure sore development. Other studies suggest that in high risk groups age is less important than associated morbidity[60;88]. In the present study age was found to be not significant. Sampling was purposive in relation to age (55 years and over) and

surgery (major) and results concur that potential age related differences in risk are less important in such a homogenous group than the morbidity associated with individual circumstance. The relationship is likely to be multi-factoral and related to both increased morbidity and disease, which affect mobility and age related changes of the skin, which reduce tissue tolerance[91]. In the application of results to practice the wider body of evidence supports the use of age as a screening measure.

With regard to nutritional status as measured by serum albumin and body mass index, results concur with other research of elective surgical patients. Whilst, nutrition related factors are identified by 8 of the 12 studies using multi-variate statistical analyses (Table 2.1), they are not identified by the two studies of surgical patients[12;87].

Little previous work has been undertaken exploring haematological factors such as haemoglobin, platelets, white blood cells, urea and electrolytes levels[110;111]. The variables were included on an exploratory basis since data were readily retrieved from pre-operative screening tests. The majority of values were within the normal range and results suggest that in pre-operative patients with no existing skin damage such factors are of little prognostic value. Again, given the specificity of the exclusion criteria validation of these findings is required.

Post-operatively 57% of patients were allocated a variety of pressure sore prevention equipment including silicore fibre overlays, foam mattresses and dynamic systems. Given that such allocation was not random and four mattress categories were in use (standard, overlay, mattress replacement and dynamic) one would not expect to find a statistically significant difference in post-operative mattress allocation and outcome.

The homogeneity found within the Braden Scale subscales raises issues regarding the limitations of risk assessment scales in terms of development methodologies and validity. The three subscales – Mobility, Activity and Friction and Shear – are all correlated with the total score and Mobility is correlated with both Activity and Friction and Shear. Substitution of Mobility for Activity in the logistic regression modeling resulted in selection of the same variables in the final model. It would appear, therefore, that three of the subscales within the Braden Scale explain much of the same variability and are measuring the same risk factor expressed in different ways.

The prognostic model derived from this study is novel, hypothesis generating and requires confirmation by further study. Results are limited by study design since the hypothesis and sample size were not determined by the prognostic factor study but by the primary research question related to the effectiveness of intra-operative pressure relief. The model is further limited by the outcome definition in relation to the inclusion of blanching erythema and the limited post-operative follow-up period included (up to Day 1 post-operative).

Also, nursing care variables (for example, turning and positioning) which have been the subject of little research[1] were not included within the data set since assessing the effect of variation in practice was not required within the randomised design. The model derived, therefore, is context specific and requires further confirmatory study for each of the prognostic factors identified. Examples are provided in Table 4.10 to illustrate the potential application of prognostic factor research to assessment of risk.

This type of study is described by Simon and Altman[75] as a Phase 1 prognostic factor study, that is, early exploratory study. They also present guidelines for the design of Phase 3 prognostic studies, that is large confirmatory studies of pre-stated hypotheses and discuss issues of sample size, statistical analysis, interpretation and application of results within clinical practice.

The results from this and other studies of surgical patients provide a baseline for future prognostic factor research. The exploratory nature of the study precludes generalisation of results and direct application to practice. The development of a probability equation does illustrate the future potential application of prognostic factor research within the clinical setting in topics of relevance to nursing and further highlights the limitations of current assessment tools.

## 5.6 Pre-operative Prevalence

Pre-operative prevalence of pressure sores are unreported by previous researchers. It is noteworthy that all severe sores (Grade 3 and above) in the combined cross sectional and prospective cohorts were reported only on patients in the pre-operative period and that more patients were excluded due to pre-operative pressure damage than subsequently developed pressure sores in the post-operative period.

The results highlight further the limitation of the randomised controlled trial that considered only the prevention of new pressure damage. Given that Allman and colleagues[88] determined a seven fold increase in risk associated with the presence of a non-blanching erythema the extent of the problem pre-operatively highlights the need for further research to explore peri-operative risk factors in patients with existing damage, the effectiveness of intra-operative interventions in reducing the conversion of non-blanching areas to skin breaks and where skin loss has occurred the prevention of further damage.

It is also noteworthy that approximately two thirds of the pre-operative pressure damage was not recorded in the ward nursing or medical notes. It is feasible that non-blanching areas observed in the anaesthetic room were incipient in nature and therefore not observed by ward staff. However, that two thirds of superficial skin breaks and three of seven Grade 3 pressure sores were not recorded in the notes raises serious issues of responsibility and accountability for theatre staff and highlights the need to undertake and document pre-operative skin assessments in order to establish a baseline for care.

## 5.7 Summary

In this randomised controlled study of 446 patients undergoing vascular, general and gynaecology surgery the use of a dry visco-elastic polymer pad intra-operatively reduced the probability of pressure sore development by half. It is noteworthy that the majority of endpoint failures were persistent blanching hyperaemia, but that 69% of these patients were observed to have persistent skin changes beyond the peri-operative period and this furthers the debate regarding the clinical importance of this outcome.

The prognostic model derived from this study is exploratory in nature and provides a baseline for future prognostic factor research. The prognostic factors identified are consistent with findings from other studies that have utilised multi-variate analyses. The small number of studies world-wide and the limitations of study design preclude generalisation of results and direct application to practice.
Multi-variate analysis also highlights further the limitations in the development methodologies of risk assessment tools and associated poor validity, since three of the subscales within the Braden Scale explain much of the same variability and are measuring the same risk factor expressed in different ways.

The pre-operative prevalence of pressure sores and poor documentation raises serious issues for theatre teams and indicates the need for pre-operative screening of 'high risk' groups and documentation of pre-operative skin condition. It also highlights the need for further research to establish the effectiveness of intra-operative interventions in the prevention of further pressure damage.

## 5.8 Recommendations

### *5.8.1 Implications for Practice*

This study, together with a wider body of knowledge derived from a systematic review of pressure sore prevention equipment[1] provide evidence that low technology constant pressure supports are effective in reducing pressure sore incidence when compared with standard mattress provision. The minimal cost and ease of use supports their general use within theatre practice.

The results of the prognostic factor analysis and pre-operative prevalence of pressure sores highlights the limitations of risk assessment practices which are based upon poorly constructed risk assessment scales and current theatre practices. It is necessary to have further debate with regard the role of risk assessment scales and how best the evidence regarding prognostic factors can be incorporated into nursing assessment processes [61].

Pre-operative skin assessment is not common practice and risk assessments are made using a 'recognised' scale or on the basis of type of surgery. Results indicate the need for pre-operative screening of high risk groups and documentation of pre-operative skin condition. High risk groups should be determined locally, combining knowledge of the broad pressure sore literature, surgical procedures undertaken (duration of surgery, anaesthetic techniques) and post anaesthetic care facilities[61;112].

### *5.8.2 Implications for Research*

The results of this study raise three areas for further research.

Of most importance is that the validity of outcome definitions which include erythema are determined. Associated with this central issue is the need to establish whether there are differences between blanching and non-blanching erythema, and clinical signs that may indicate irreversible damage and subsequent skin loss.

The pre-operative prevalence of 10.3% highlights the need for further research to establish the effectiveness of intra-operative interventions in reducing the conversion of non-blanching areas to skin breaks and where skin loss has occurred the prevention of further pressure damage. However, recruitment of the appropriate patient group would pose research management and design problems, requiring the selection of a large number of patients and high attrition in the immediate pre-operative period.

Prognostic factor research in this field is limited to Phase 1 studies and requires further development and exploration. However, such work will be limited in its application to practice until an evidence based definition of the term pressure sore is established.

# Chapter 6 Pathology of Pressure Sore Development

## 6. 1 Introduction

Pathology is, 'the sequence of events that occurs from the time of first injury to the time when the disease expresses itself in functional and structural terms' (Woolf 1998[113]p.4). This Chapter outlines both normal and abnormal physiological processes which protect the skin and underlying structures from pressure-induced damage, the pathological mechanisms which lead to skin breakdown and links the pathological stages to the clinical manifestations of responses to external pressure.

## 6.2 Anatomy of the Skin

The tissues involved in pressure sore development are the skin, subcutaneous fat, deep fascia, muscle and bone. Skin in particular plays an important role. It is described as the largest organ of the body[113] and is a dynamic structure in which cellular replacement and modification in response to local need is a continual process throughout life[16]. It is relatively resistant to water, chemicals and bacteria and provides some protection for the body against mechanical damage. Structurally, it consists of 3 layers - the epidermis, the dermis and subcutaneous tissue.

The epidermis consists mainly of stratified squamous epithelium (keratinocytes) and a small number of melanocytes (for melanin synthesis), Langerhans cells (antigen-presenting cells) and Merkel cells (neuroendocrine function). The squamous epithelium cells are arranged in four layers including stratum corneum, granular layer, stratum spinosum and stratum germinativum (or basal layer). The stratum corneum consists of cells that have no nuclei or cytoplasmic organelles, contain little water, are tightly packed and provide a physical barrier against water, bacteria and chemicals. These cells are constantly being shed and replaced by cells from the deeper layers.

A basement membrane separates the basal layer from the underlying dermis and the basal cells are attached to the membrane by structures known as hemidesmosomes. This basal lamina region consists of four zones including:

- the plasma membrane of the epidermal cells which contain hemidesmosomes
- an electron-lucent area (lamina lucida), which contains the protein laminin
- an electron-dense area (lamina densa), consisting of type IV collagen
- extensions of the lamina densa providing attachments to the underlying dermis[113].

The dermis consists of two layers, the papillary dermis and reticular dermis. The former is configured in a series of papillae that are separated by projections of the epidermis, known as rete pegs[114]. The collagen/elastin matrix of the papillary dermis is 'loose', and orientated at right angles to the epidermis. It supports loops of blood vessels, known as the papillary (nutritional) capillaries and nerve fibres responsive to touch, pain and temperature[113;115].

The reticular dermis is beneath an imaginary line joining the tips of the rete pegs and consists of thick collagen bundles orientated parallel to the overlying epidermis[113]. It supports blood vessels referred to as the subpapillary (or non-nutritional) vascular bed[115] as well as sweat glands, sebaceous glands and hair follicles.

It is the collagen and elastin connective tissues that provide the skin with its characteristic recovery following stretching[114]. Collagen is synthesised in connective tissue fibroblasts, secreted from the cells and stabilised by the formation of cross-linkages that vary in permanence. It constitutes 99% dry weight dermis[116]. The collagen fibres form a series of layers with fibres in adjacent layers aligned at a fixed angle. When external pressure is applied the fibres, which are inextensible, rotate relative to one another until they approach a parallel alignment. As the fibres move nearer to a parallel alignment tension increases. When external pressure is removed the collagen is restored to its former open structure by elastic fibres which are intertwined around the collagen bundles[116]. The process of extension and recoil by rotation and alignment is an important aspect of the property of the collagen/elastin matrix because as well as buffering internal structures of the body it also protects the interstitial fluids and cells of the dermis from external pressure[42].

A subcutaneous layer separates the dermis from the deeper structures of deep fascia, muscle and bone. It varies in thickness (depending upon body type, gender and the location on the body) due to the presence of a large number of fat cells, which provide mobility to skin and padding to dissipate pressure. The fat cells are arranged in lobules, which are separated by bands of connective tissue known as interlobular septa[113].

The deep fascia beneath is a dense essentially avascular, inelastic membrane that covers muscle and muscle groups and over bony prominences may merge with the outer layer of the bone. It is resistant to pressure and it is the last line of protection of vulnerable muscle tissue.

In summary the skin is characterised by a number of structures which afford protection from mechanical disruption. Tissues beneath, including the layers of subcutaneous fat and deep fascia, also contribute toward protection of the skin's underlying structures. Despite these characteristics, pressure sores develop mainly as a result of disruption to the vascular network of arteries, arterioles and capillaries. With continued reference to the anatomical structures described, the following section provides a detailed account of the vascular system and capillary blood flow and briefly highlights vulnerable aspects.

## 6.3 The Vascular System

A network of vascular and lymph vessels ensures the supply of necessary nutrients and oxygen to support cell metabolism and epidermal mitosis, blood flow to facilitate temperature regulation, and the removal of waste products from the skin.

The arteries supporting the skin pierce the deep fascia and form a network of arterioles in the subcutaneous tissues with capillary branches supplying the hair follicles and sebaceous and sweat glands within the dermis.

The arterioles, which are highly muscular enabling changes to their diameter, branch into a network of metarterioles that have a structure midway between arterioles and capillaries. They do not have a continuous muscle coat, but smooth muscle fibres encircle the blood vessel at intermediate points[32].

The metarterioles further sub-divide into capillaries, some of which are large and are called preferential channels and others which are small and are known as true capillaries[32]. Smooth muscle cells at the origin of the capillaries act as pre-capillary sphincters and are important in the control of blood flow.

The capillaries are composed of a single layer of highly permeable endothelial cells surrounded by a basement membrane. Between each endothelial cell is a small channel referred to as an intercellular cleft and within the endothelial cells are plasmalemmal vesicles. These structures are important in the exchange of nutrients and other substances between the blood and interstitial fluid[32].

After passing through the capillaries blood enters the venule and returns to the general circulation. The structures including the metarteriole, capillaries and venules are known collectively as the microcirculation[117].

### *6.3.1 Blood Flow Control Mechanisms*

An important characteristic of the vascular system is the ability of each tissue to control local blood flow in proportion to need and various acute and long-term auto-regulatory mechanisms are evident in order that blood flow is directly related to local tissue demand[32].

Direct observation of the microcirculation by microscope reveals that there is an intermittent ebb and flow through the capillary network controlled by the opening and closing of the metarterioles and precapillary sphincters - a phenomenon known as 'vasomotion'[32;39]. Flow through the metarterioles and capillaries is controlled by local metabolic needs by either the release of a vasodilator substance(s) or oxygen demand, however the exact mechanism is not known[118]. An interplay of osmotic and hydrostatic pressures of plasma and interstitial fluid determine capillary permeability and reabsorption as well as directly affecting the use of lymph vessels in removing proteins, large waste particles and excess fluid[32].

An increase in blood flow through the capillary bed requires an increase in supply from the feeding artery. The local mechanisms that determine capillary blood flow also involve a feedback mechanism, which can initiate dilatation of the larger arterial vessels. Rapid flow of blood through the arteries and arterioles causes 'sheer stress' on the endothelial cells of the artery wall resulting in the release of endothelium-derived relaxing factor (EDRF). The EDRF then relaxes the arterial muscle and the

artery dilates, thus increasing the blood supply. In the long-term, if blood flow continues excessively for days/weeks/months the arterial vessels enlarge[32]. Indeed, the size of arterial vessels appear to be readjusted throughout life so that blood flow velocity is never great enough to cause an inordinate amount of blood flow resistance[32].

An acute increase or decrease in arterial blood pressure will result in a surge or reduction in blood flow through a tissue but within minutes an auto-regulatory mechanism readjusts flow to values of approximately 3/4 of the previous level. The mechanism involved is not clearly understood (metabolic/myogenic), but the resulting autoregulation of blood flow ensures protection of capillaries from excessive pressure and maintains blood flow despite changes in arterial pressure. Over a period of hours/days/weeks a long term regulatory mechanism is apparent, with control established by changes in the vascularity of the tissue[32;118].

Similarly, an autoregulatory mechanism, known as the veni-arteriolar response, protects the microcirculation from increases in venous pressure (for example, during standing or venous occlusion). An increase in venous pressure triggers an axon reflex of sympathetic nerve fibres causing contraction of arterioles and a reduction of flow[119].

Other mechanisms involved in the control of blood flow include nervous and humoral mechanisms whereby various vasoconstrictor agents (for example, norepinephrine, epinephrine, angiotensin and vasopressin) and vasodilator agents (for example, bradykinin, histamine, prostaglandins and various ions) are released. Some result in systemic effects and others in localised changes to tissue/organ blood flow[32].

### *6.3.2 Factors Affecting Skin Blood Flow*

Skin blood flow varies from individual to individual, is site dependent and is affected by a combination of systemic, local and disease related factors. These require consideration in the use and interpretation of various technologies in assessing skin blood flow and in the identification of possible risk factors in pressure sore development.

Individual differences, site specific variation in skin blood flow and the positive correlation between skin temperature and blood flow are well documented[109;120;121]. As a consequence, measures of skin blood flow are most commonly used to explore

stimulus response, where relative changes rather than absolute values are examined and the effects of the stimuli outweigh other factors influencing blood flow in the test area[122].

An increase in blood flow is observed following localised skin trauma and/or infection. The classic response to local trauma is referred to as 'the triple response', which includes the red reaction, wheal and flare. The red reaction is due to capillary dilatation, the wheal results from oedema of the local area and the redness spreading out from the injury (the flare) is due to arteriolar dilatation[123]. Similarly local inflammation of skin or underlying tissue due to infection, chemical trauma, sunburn, radiation damage and so on, results in increased localised skin blood flow with associated heat, pain and swelling[113;124;125].

The wider literature illustrates disease pathologies which may increase baseline skin blood flow including diabetes mellitus[126], liposclerotic skin resulting from venous insufficiency[127] and spinal cord injury[128]. Of particular note is that the increase to baseline skin blood flow effects the capacity of the skin to respond to thermal stimuli and localised trauma and a reduced hyperaemia response is observed. This increases the risk of skin damage[126-128].

The interplay of factors in the control and autoregulation of skin blood flow are clearly illustrated by Tooke and Brash[126] who reviewed microcirculatory function of 'the diabetic foot'. Early increased microvascular pressure and flow cause an endothelial 'injury response' leading to microvascular sclerosis. With increasing duration of diabetes the sclerotic process results in limitation of vasodilatation with reduced maximal hyperaemia and loss of autoregulation. The increased baseline skin blood flow results in higher skin temperature and it is unclear whether the blood flow is adequate to meet the increased metabolic tissue demand. The pathophysiology is further complicated by peripheral neuropathy, which affects the sympathetic nerve fibres and reduces both the veni-arteriolar and axon flare response. The consequence of these pathophysiological changes is a high prevalence of minor trauma induced foot ulcers within the diabetic population.

### *6.3.3 Protective Auto-regulatory Blood Flow Mechanisms in Response to Pressure*

Of particular interest in pressure sore aetiology are autoregulatory mechanisms which affect blood flow during and following pressure assault including raising of

capillary pressure to maintain flow, intermittent flow at sub-critical pressures, response to repetitive loading and the reactive hyperaemia response following full/partial occlusion.

When external pressure is applied to the skin an autoregulatory process allows internal capillary pressure to rise correspondingly. Landis[129] noted that within one minute from the time of external pressure application (60mmHg) a rise in capillary pressure occurred and stabilised at approximately 10mmHg higher than the external pressure.

Reduced blood flow is maintained at sub-critical external pressures[108;130;131]. Romanus[108] demonstrated that sub-critical pressure application resulted in temporary circulatory arrest followed by variable periods of recirculation. Using intravital microscopy he determined that recirculation was characterised by an unevenly distributed, slow and jerky blood flow.

Also of importance is the effect of repetitive loading on skin tissue as demonstrated by Bader[103]. In two studies involving healthy volunteers the application of external load resulted in a reduction of transcutaneous oxygen tension which partially recovered during the load period. Following load removal, tissue recovery to unloaded oxygen levels was rapid. With each further load application the effect on transcutaneous oxygen tension diminished demonstrating an active vasomotor response mechanism.

### *6.3.4 Reactive Hyperaemia*

Partial or full arterial/capillary occlusion results in anoxia and a build up of metabolites. Release of pressure produces a large and sudden increase in blood flow through the deprived tissue, a response known as reactive hyperaemia. This was first described by Lewis and Grant[132] who reported that during occlusion the supplying blood vessels became dilated, providing a reservoir of blood and a rapid high flow following pressure release.

It is thought that there are two mechanisms involved in the post-occlusive reactive hyperaemia response - the immediate post-occlusive blood flow is determined mainly by myogenic mechanisms, and the recovery of blood flow to baseline levels is influenced by metabolic factors[133]. The dominant metabolic driver is undetermined and it is generally believed to be either oxygen deficit or metabolite release from

anoxic tissue[118]. It is known that the reactive hyperaemia response is independent of vasomotor control[118].

The reactive hyperaemia response following occlusion of skin blood flow has been studied by researchers using various techniques including transcutaneous oxygen tension, laser Doppler flow, tissue reflectance spectrophotometry and skin temperature. The hyperaemia response can be quantified in various ways and parameters compared within and between groups.

In normal healthy individuals the magnitude (maximum value), total hyperaemia and duration of the reactive hyperaemia response is related to the duration of the occlusion[132;134-136]. The duration of the hyperaemia response is approximately 1/2 - 3/4 of occlusion time although occlusion times studied are short[132;133]. The maximum peak value is inversely proportional to the lowest values during occlusion and related to baseline skin blood flow and skin temperature[136-139]. Other factors affecting the reactive hyperaemia response are also associated with groups at high risk of pressure sore development including age and vascular disease. Evidence relating to spinal cord injury is inconclusive. Comparison of the reactive hyperaemia response following short periods of occlusion in elderly and young subjects has identified lower peak perfusion values and faster times to peak in elderly groups resulting in a much reduced total hyperaemia[140;141]. Indeed mean total hyperaemia values reported by Hagisawa et al[140] clearly illustrate the reduced hyperaemia response - younger subjects 7236 perfusion units, older subjects 1825 perfusion units.

Patients with symptomatic vascular disease have a reduced or absent hyperaemia response. Reactive hyperaemia is delayed, diminished and prolonged in patients with intermittent claudication when compared with young and elderly controls and completely absent in many patients with critical ischaemia[142-144]. Smokers have a reduced hyperaemia response[140], as do patients with medical conditions that result in vascular changes including diabetes mellitus[126], systemic sclerosis[145] and end stage renal failure[146].

Reactive hyperaemia in the spinal cord injured requires consideration given their high risk of pressure sore development. Studies exploring reactive hyperaemia in spinal cord injured subjects have found no differences in the response when compared with able-bodied controls, supporting other evidence that reactive hyperaemia is independent of vasomotor control[140;147;148].

However, Schubert and Fagrell[128] reported significant differences in the percentage rise in skin blood flow over the sacrum and faster time to peak, lower peak and reduced percentage rise in skin blood flow over the gluteus muscle area for spinal injured patients compared with healthy subjects. These are similar to the effects of ageing on the post-occlusive reactive hyperaemia response. Furthermore, Barbenel and Cui[122] assessed the skin response of paraplegic subjects to thermal insulation. They found that in able-bodied subjects thermal insulation resulted in a significant rise in both skin temperature and skin blood flow, whereas, paraplegic subjects only demonstrated a significant rise in skin temperature. The lack of thermally induced hyperaemia may have clinical consequences and reduce tissue tolerance during pressure assault.

## 6.4 Pathological Mechanisms Leading to Skin Breakdown

A review of the literature suggests three types of pressure sore with possibly three mechanisms that lead to tissue breakdown. The three different types of pressure sore described by researchers include: necrosis of the epidermis or dermis which may or may not progress to a deep sore[16;34;149]; deep or 'malignant' pressure sores where necrosis is first observed in the subcutaneous tissue (muscle or fat) and tracks outwards[16;150] and; full thickness wounds of dry black eschar[149].

The mechanisms leading to tissue breakdown are not entirely clear from the limited research undertaken to date but at least three pathophysiological processes are evident including:

- occlusion of skin blood flow and subsequent injury due to abrupt reperfusion of the ischaemic vascular bed
- endothelial damage of arterioles and the microcirculation due to the application of disruptive and shearing forces
- direct occlusion of blood vessels by external pressure for a prolonged period resulting in cell death.

A limitation of research in the area of pressure sore pathophysiology is the difficulty in replicating the clinical situation. The majority of pathology research details the microvasculature response to a single pressure assault, whereas, in the clinical environment, patients are exposed to repetitive pressure complicated by friction and

shearing forces. It is not possible to determine a dominant pathological mechanism. Indeed it is possible that all three mechanisms play a role in the development of pressure induced skin lesions.

There is also difficulty in determining the point at which the ischaemic assault becomes critical and results in tissue breakdown. Ischaemic conditions can develop even when partial flow is maintained[105] and both clinical and pathological signs of localised trauma including non-blanching erythema with associated induration and swelling can resolve without superficial skin loss[37;98]. The current research base cannot address this issue.

### *6.4.1 Occlusion of Skin Blood Flow and Tissue Reperfusion*

The majority of the pathophysiology literature outlines events following a period of critical ischaemia and subsequent reperfusion of skin leading to pressure sore development. Visible changes/loss of skin are followed by deeper tissue destruction.

The application of local pressure causing complete occlusion of blood flow causes squeeze out of blood from the micro-circulation and a decrease in both glucose and Adenosine triphosphate (ATP) indicating anaerobic cell metabolism[151].

Following pressure release the microvasculature rapidly refills with blood from both the arterioles and venules and a reactive hyperaemia response is observed[35;37;38;151]. The endothelial cells of the capillary wall swell[16;35;152], are infiltrated with leucocytes[38] and increase in permeability resulting in extravasion of red blood cells, white blood cells and plasma leakage into the interstitial space[35;108;149;151-153]. White blood cells are observed to adhere to the endothelial wall[108;151], red blood cells form rouleaux and thrombi but do not necessarily occlude the vascular lumen[108;149;151;152].

The consequence of these events include localised oedema[37;38;108;149;151] and a significant decrease in blood flow through the microvasculature to either low or no flow[151;152], resulting from a combination of increased blood viscosity, obstruction within the vasculature by red and white cells and the reduced vascular lumen[35;152]. This response known as the 'no-flow phenomenon' extends the ischaemic assault[151;152;154;155].

Much of the research pertaining to the pathophysiology of pressure sore development was undertaken over 20 years ago. However, the similarities between

these early studies of pressure sore related pathological events and the recent literature describing reperfusion injury identifies a common sequence of events suggesting pressure sore development may result from reperfusion injury, a phenomenon which provokes tissue injury due to the process of abrupt reperfusion of the ischaemic vascular bed[118;155;156]. Reperfusion injury is characterised by two distinct but related events - endothelial dysfunction and neutrophil adhesion[155].

Altered microvascular permeability, cytoskeletal changes, endothelial cell swelling and neutrophil adhesion are characteristic of endothelial injury during reperfusion, resulting in tissue oedema, capillary plugging by neutrophil adhesion to the endothelium and reduced or no blood flow[155-157]. The no-reflow is a consistent feature of reperfusion injury[156]. Important similarities between pressure sore pathophysiology and reperfusion injury is that the no-flow phenomenon is not related to thrombus formation but a combination of factors including endothelial disruption and swelling, red blood cell rouleaux formation and neutrophil adhesion[108;152;154;156].

The metabolic events that initiate the cellular response of reperfusion injury have been investigated in the major organs of the body in attempts to develop preventative treatments. There are some differences in response between organs dependent upon their basic cellular structure and adaptation, but a number of common metabolic features are evident. Biochemical events are complex, but described briefly. During the ischaemic assault oxygen debt leads to anaerobic metabolism of ATP and a build up of metabolites. When perfusion is restored biochemical events generate oxygen free radicals which cause cell membrane damage and cellular dysfunction. High intracellular calcium ions, which increase during both ischaemia and reperfusion also, have a pivotal role in cell damage[123;156;157].

Whilst the cellular events of pressure sore development and reperfusion injury pathophysiology indicate broad similarities, biochemical events have not been explored in relation to the former and firm conclusions cannot, therefore, be drawn. However, from a clinical perspective three pathophysiological features are of potential importance including the initial reactive hyperaemia response, subsequent development of tissue oedema and the low or no-flow phenomenon.

### *6.4.2 Subcutaneous Tissue Necrosis*

Two research studies describe pressure induced lesions which first develop in the muscle, subcutaneous fat or deep dermis and lead to eventual death of the

dependant skin area which sloughs off to reveal a cavity beneath[16;150]. Barton and Barton[16] describe these as Type 11 pressure sores and clinical experience suggests that they are most frequently seen over the trochanter and the sacral areas.

The experimental models describe two processes - repetitive disruptive and shearing forces causing endothelial cell damage and activation of intrinsic clotting mechanisms particularly in the dermis[16] and a single pressure assault of relatively long duration resulting primarily in muscle necrosis[150]. These clearly link to the body sites affected - the sacrum to repetitive disruptive forces (for example, repeatedly slipping down in sitting/semi-recumbent position) and the trochanter can be exposed to long periods of high external pressure.

Barton and Barton[16] describe in detail the effect of disruptive and shearing forces in pressure sore development by inducing a gait disorder on the foot of the mouse. The external repetitive forces cause distortion of blood vessels, disruption to endothelial cells and activation of intrinsic clotting mechanisms. Platelets aggregate and occlude the affected vessels causing ischaemic necrosis of dependent tissues. The epidermis was observed to remain intact for a number of days before sloughing off to reveal the extent of the tissue damage beneath. Some similarities exist in the pathological events described by Barton and Barton[16] when compared with reperfusion injury but an important distinction is that platelet aggregation was observed to cause blood vessel occlusion and ischaemia of dependent tissue. It is not clear, however, from the existing research whether two different mechanisms exist nor the applicability of Barton and Barton's work given the severity of repetitive forces induced by the gait disorder.

With respect to muscle damage there is also difficulty in determining the pathological events that lead to necrosis and whether reperfusion exacerbates the primary ischaemic assault. There is also difficulty in determining the point at which the ischaemic assault becomes critical resulting in pressure sore development. Muscle necrosis has been observed even when skin ulceration does not occur[38;150;158]. It would seem, however, that two critical components in the development of such sores are underlying bone which magnify the pressure by increasing interstitial pressure particularly in the muscle[159] and that the duration of pressure required is far in excess of clinically accepted periods of immobility.

### *6.4.3 Prolonged Ischaemia Resulting Directly in Tissue Necrosis*

Whilst areas of dry black eschar are observed clinically there is little evidence within the literature as to the pathological mechanisms involved. However, findings of Witkowski and Parish[149] who describe histological changes characteristic of eschar/gangrene suggest that the mechanism is distinct from those previously described.

Witkowski and Parish[149] reported histological changes as follows:
'Black eschar. This phase in the decubitus ulcer spectrum represents full-thickness destruction of the skin. The tissue appears basophilic. Although the general architecture of the dermis is preserved, the cellular details are obliterated. The epidermis is not present in the black eschar. Red blood cells and inflammatory cells are not evident; neither are the other changes previously described in blanchable and nonblanchable erythema and decubitus dermatitis.' (p.1017).

'The black eschar usually occurs in areas where the skin is thin and bony prominences and tendons are close to the surface. It may occur on normal-appearing skin or be preceded by blanchable erythema, or decubitus dermatitis. In either event, it is usually surrounded by an inner zone of nonblanchable erythema and outer zone of blanchable erythema. The black tissue is grossly dehydrated and compressed. Its acellular, dry nature suggests that necrosis occurred without reperfusion of the skin, unlike what is thought to occur in the other phases of the decubitus spectrum.' (p. 1020).

Similar in description to the 'dry gangrene' most frequently seen in the lower limb in patients with severe atherosclerosis where arterial narrowing has progressed slowly over a long period[113], it suggests that pressure sores may in some instances arise from prolonged ischaemic assault and direct tissue necrosis. It is unclear which tissue layer has the primary ischaemic injury.

## 6.5 The Definition and Classification of Pressure Sores

Pressure sores have been defined mainly for the purposes of research. There is no agreed definition of the term, with researchers defining a pressure sore to reflect the population under study. Severity ranges from definitions which include blanching

erythema (usually for defined time periods such as 30 minutes or >1 consecutive day), non-blanching erythema and skin break.

Pressure sore classification systems have been developed in order to categorise the severity of pressure sores. A large number have been developed[24] and the majority grade or stage pressure sores according to the tissue layer affected. In clinical practice the purpose of a classification system is to provide a common descriptor of sore severity, a benchmark measure to evaluate interventions which promote healing and in relation to 'early' pressure sores (such as non-blanching erythema) prompt active interventions which may prevent tissue loss.

The recent debate regarding the definition of a Grade 1 pressure sore with descriptors ranging from blanching erythema, non-blanching erythema and skin loss[22;97] is flawed in two ways. Firstly, it has not recognised the difference between defining a pressure sore to determine the incidence and prevalence (for audit and research) and a classification system which includes a range of clinical observations of relevance to planning and evaluating care delivery.

Secondly, the definitions and underlying assumptions of a Grade 1 pressure sore are not evidence based. Clinical observations of blanching and non-blanching erythema have not been validated against physiological measures of skin perfusion or in relation to subsequent skin loss and their clinical significance is not fully understood.

Non-blanching erythema is recognised as reversible by some[22], but indicative of irreversible damage by others[19]. Only recently has research evidence been available which determines that a break in the skin is not an inevitable consequence of non-blanching erythema[88].

Expert opinion in the United States of America attributes no importance to the observation of blanching erythema. The AHCPR clinical guidelines relating to pressure sores state 'Stage 1 pressure ulcers are defined as nonblanchable erythema of intact skin - the heralding lesion of skin ulceration. Note: Reactive hyperaemia can normally be expected to be present for one-half to three fourths as long as the pressure occluded blood flow to the area (Lewis and Grant 1925). This should not be confused with a Stage 1 pressure ulcer).' [23].

However, there are pathological differences between 'normal' skin and areas of blanching erythema. Witkowski and Parish[149] examined biopsies of skin and describe in pathological terms the changes observed from blanching erythema through to black eschar. They noted a number of pathological changes in skin samples obtained from 18 areas of blanching erythema, which are summarised in Table 6.1. Their findings suggest that blanching erythema may be clinically important.

**Table 6. 1 The Pathology of Early Pressure Sores[149]**

| | |
|---|---|
| Blanchable erythema<br><br>Main changes: papillary dermis | a. the capillaries and venules are greatly dilated with prominent endothelial cells<br>b. mild lymphocytic peri-vascular infiltrate and mild to moderate edema in upper dermis<br>c. occasionally observed:<br>i. fibrin thrombus in the deep dermis<br>ii. degenerative changes in the eccrine sweat gland secretory coils and ducts<br>iii. focal necrosis of the subcutaneous fat with polymorphonuclear leucocytes<br>d. epidermis and reticular dermis appear normal |
| Nonblanchable erythema<br><br>Main changes: papillary dermis | a. red blood cell engorgement of the capillaries and venules<br>b. vascular ectasia, the peri-vascular infiltrate and edema of the papillary dermis<br>c. platelet aggregates in some sections<br>d. vascular engorgement followed by perivascular and later diffuse hemorrhage<br>e. degeneration of the eccrine sweat glands, sebaceous glands and subcutaneous fat<br>f. loss of cell membranes and inflammatory infiltrate<br>g. occasionally observed:<br>i.fibrosis and engorgment with red blood cells in reticular dermis<br>ii. fibrin thrombi and (rarely) organised thrombus<br>h. epidermis still appears normal |
| Decubitus dermatitis<br><br>Main changes: papillary dermis and epidermis | a. changes seen in non blanching erythema are more frequent and pronounced<br>b.epidermis:<br>i. diffuse eosinophilia with erosions and crust formation<br>ii. focal eosinophilia and necrosis with or without subepidermal separation<br>iii. epidermal atrophy with a subdermal blister<br>iv. subepidermal bulla with a relatively normal appearing epidermis<br>v. necrosis of folicular structures and degeneration of internal and external root sheaths |

Similarities and transitional phases were noted between blanching erythema, non-blanching erythema and decubitus dermatitis, with a spectrum of pathological changes within each clinical grade and overlap between grades. These pathological findings have not been linked to the classification of pressure sores and outstanding questions remain. When is damage irreversible and do the pathological manifestations reflect a spectrum of reactive hyperaemia responses (that is, high blood flow) or reperfusion injury (that is, low blood flow)?

## 6.6 Erythema and Skin Loss

Skin condition is one of five themes, which emerge from studies utilising multi-variate analyses to identify key prognostic factors (Table 2.1). It is identified as a risk factor associated with subsequent skin loss by all 5 studies that included skin condition as a variable.

Clarke and Kadhom[84] in a prospective study involving 88 hospitalised and 30 community bedfast or chair fast patients reported that 'skin changes are apparent before an actual skin break occurs'. Skin condition variables were identified by discriminant analysis as factors associated with pressure sore outcome. The study involved nursing staff and carers completing diary sheets on each occasion that pressure area care was given. It is not clear from the research report how 'the state of the skin at the site' was recorded - that is, whether descriptions or category options were used and there is no descriptive data reporting the skin conditions observed. This study is, however, important in providing research evidence linking visible changes in skin condition to subsequent skin loss.

Further evidence is provided by the US National Health and Nutrition Survey 10 year follow up which found individuals with physician diagnosed dry or scaling skin at baseline were 2.5 times more likely to develop pressure sores during the 10 year follow up period than those individuals with normal skin[85]. Results cannot be applied directly to practice due to the limitations associated with the survey methodology, but together with other research are further evidence of an association between skin condition and pressure sore occurrence.

Marchette et al[86] in a retrospective record review of 161 surgical intensive care patients reported 'a significant relationship between the incidence of redness and

skin ulcers (p=0.00001)' although actual conversion rates were not reported. Discriminant analysis also identified skin redness as one of a combination of five factors that predicted 93% of the patients who developed pressure sores. The study methodology of record review is limited and there is no definition of the term redness (whether blanching or non-blanching or both).

Allman and colleagues[88] in a prospective cohort study of hospitalised patients with activity limitations and aged over 55 years, identified non-blanching erythema of the sacrum at baseline assessment as one of five predictors of pressure sore development using Kaplan-Meir survival analysis and Cox regression analysis. A pressure sore was defined as epithelial loss or skin breakdown over a bony prominence. The risk ratio of pressure sore development associated with the presence of non-blanching erythema at baseline assessment was 7.52 ($p = 0.05$). Non-blanching erythema observed during hospital follow up (not included in the primary analysis) was also determined as significantly associated with pressure sore development with conversion of 11/19 (57.9%) hospital acquired non-blanching skin areas to a pressure sore ($p<0.001$). The presence of blanching erythema was not recorded, nor was detail regarding other skin changes such as induration, swelling, heat or pain at the non-blanching sites.

Finally, Schnelle and colleagues[90] in a study of 100 incontinent nursing home residents determined that blanching erythema severity was the only variable predictive of 'nonblanchable erythema plus Stage 2'. They also reported that 29% of subjects with a non-blanchable erythema on first observation subsequently developed a pressure sore at the same site. Study results are difficult to interpret clearly due to the various skin descriptors used and it is not clear whether the multiple regression analysis was performed using the pressure sore free cohort. Furthermore the relationship between non-blanching erythema and pressure sore development does not appear to have been explored.

In summary, the evidence base is limited by the following. Firstly, it is not possible to determine whether blanching or non-blanching erythema are indicators of risk, although evidence from two studies suggest a relationship between non-blanching erythema and pressure sore occurrence. Secondly, it is unclear whether non-blanching erythema with or without other skin changes reflects post-occlusive reactive hyperaemia or irreversible ischaemic damage.

## 6.7 Analysis of Secondary Data

In the study of intra-operative pressure sore prevention and prediction (Chapters 2-5) skin assessment data was collected up to Day 8 post-operatively but not included in the primary endpoint. Secondary analysis was performed using this data in order to clarify the emerging research questions. In this secondary analysis Grade 2a and 2b pressure sores recorded for 1 day with no other skin changes on preceding or proceeding days were included, resulting in 38 patients with one or more Grade 2a skin changes and 24 patients with a Grade 2b.

For the purpose of the analysis a pressure sore is defined as a skin break or blister and classified as a Grade 2b. Skin changes preceding pressure sore development are detailed in Table 6.2, illustrating that pressure sores are both incipient and preceded by skin changes including blanching and non-blanching erythema.

The skin assessments undertaken on the five skin sites were summarised as a single grade using the worst skin area observed. Transient blanching erythema was excluded with categorisation as follows:

Group 0 Grade 0/Grade 1a observed for one day

Group 1 Grade 1a observed for two or more consecutive days

Group 2 Grade 2a observed for one or more days

The analysis, therefore included only patients who developed post-operative pressure sores from Day 2-8 and excluded 7 patients who developed pressure sores up to Day 1, for whom data was incomplete (only 1 day skin data available). The relationship between skin changes and subsequent pressure sore development were examined using the chi-square statistic and linear logistic regression.

**Table 6.2 Skin Changes and Post-operative Pressure Sores**

| Post-operative Pressure Sore | Skin Assessment Group 0 | 1 | 2 | Total |
|---|---|---|---|---|
| No | 182 | 195 | 38 | 415 |
| Yes | 4 | 8 | 5 | 17 |
| Total | 186 | 203 | 43 | 432 |

Chi-squared value=8.2984 p=0.016

Differences were observed between Group 0 compared with Group 2 (odds ratio 5.98, p=0.010) and Group 1 with Group 2 (chi-squared = 4.1891, p= 0.041 and odds ratio 3.20, p=0.051). Whilst an increased risk of pressure sore development was also associated with Group 0 compared with Group 1 (odds ratio 1.86) this was not significant (p=0.315).

The analysis suggests then, that it is the presence of non-blanching erythema, which is of importance in pressure sore development and indicates the need for further exploration to determine the relationship between non-blanching erythema and pressure sore development.

Further analysis was undertaken in order to determine the significance of blanching erythema of any duration but there are difficulties in quantifying risk due to the large number of patients assessed as having a blanching area for 1 day (Table 6.3). Further exploration is required in order to determine the risk associated with blanching erythema, both transient and persistent.

**Table 6. 3 Skin Changes of any Duration and Pressure Sore Development (including peri-operative sores)**

| Pressure Sore | Skin Grade 0 | 1a1day | 1a>1day | 2a | Total |
|---|---|---|---|---|---|
| No | 21 | 161 | 195 | 38 | 415 |
| Yes | 3 | 8 | 8 | 5 | 24 |
| Total | 24 | 169 | 203 | 43 | 439 |

Chi-squared value=6.5458 p=0.088

Finally, there were various patterns in the skin changes observed prior to pressure sore development. These included observation of a blanching area on a site other than the subsequent pressure sore site; periods of persistent blanching erythema followed by assessments of 'normal' skin immediately preceding the appearance of the pressure sore and; periods of persistent erythema preceding pressure sore development. It is unclear, therefore, what aspects of blanching erythema may be important and whether other factors (such as mobility) in combination provide a better understanding of associated risk.

## 6.7 Summary

The poor knowledge base associated with skin changes and the pathophysiology of pressure sore development at capillary level poses problems from both a practice and research perspective. At a practice level difficulties are encountered in interpretation of clinical signs and symptoms and determining their clinical importance (risk assessment). Whilst best practice might advocate the prevention of skin redness the scientific base has not been tested clinically. Difficulties are also encountered in the assessment of patients with darkly pigmented skin. The proposed research will inform the application of health technologies to this clinical problem.

From a research perspective there is difficulty in determining exclusion criteria and endpoint definitions. The validity of pressure sore outcome definitions which include skin changes have not been determined and yet are crucial to research exploring the effectiveness of equipment/nursing care interventions and prognostic factors. Sample size requirements to demonstrate a difference in support surface using an outcome of skin break would render many studies non-viable.

There is a clinical and research need, therefore, to validate clinical signs and symptoms of pressure assault of the skin against physiological measures of skin perfusion and subsequent skin/tissue loss.

# Chapter 7 Pilot Study

## 7.1 Introduction

The study of intra-operative pressure sore prevention and prediction (Section 1) and further review and synthesis of the pathology literature (Chapter 6) highlight the need for further research to validate pressure sore definition, establish whether there are differences between blanching and non-blanching erythema, and the importance of erythema as a risk factor. In order to inform main study design and sample size calculations a small prospective pilot study was undertaken as detailed in the following sections.

## 7.2 Research Questions and Aims

### *7.2 1 Research Questions and Aims*

1. What are the physiological differences between normal skin, blanching erythema, non-blanching erythema with or without other skin changes (such as local induration, oedema, pain and discolouration)?
2. Which clinical signs and symptoms of the skin response are predictive of skin loss?
3. Which variables are independently predictive of pressure sore development?

### *7.2.2 Pilot Study Aims*

A pilot study was undertaken to assess the practical utility of the physiological measurement technology of choice, inform main study design and provide data to calculate sample size. The main study aims were to:

a. Assess the validity of the clinical grading of erythema by comparison with a measure of skin blood flow
b. Assess the validity of clinical signs of erythema as predictors of pressure sore development
c. To identify variables which independently are predictive of subsequent pressure sore development

## 7.3 Physiological Measurement Technologies

There are tools available that quantify tissue perfusion in various ways. Some can be used easily in the clinical environment and others require controlled laboratory conditions. The main issue under investigation is whether there are physiological differences between normal skin, blanching erythema, non-blanching erythema with or without other skin changes (such as local induration, oedema, pain and discolouration). Also important is whether the erythema observed is a manifestation of reactive hyperaemia (characterised by high blood flow) or ischaemic reperfusion injury (characterised by low blood flow).

A tool reliable for measuring both high and low blood flow across an area of pressure induced erythema was sought. It was also essential that the measurement tool did not alter the localised response and a non-touch technique was preferred.

From the range of potential technologies available[160-162] laser Doppler imaging was considered as the most applicable measure of skin blood flow.

Reasons for exclusion of other technologies are detailed as follows:
***plethysmography*** is an effective pulse detector used to assess changes in amplitude following occlusion but does not measure capillary flow.
***transcutaneous oxygen and carbon dioxide tensions*** require manipulation of the local environment (heating) and stabilisation time of approximately 20 minutes. They are not direct measures of skin blood flow.
***skin thermometry*** is an indirect measure of skin blood flow and a rise in temperature due to reactive hyperaemia is delayed compared with laser Doppler flux[137]. Further, the relationship between blood flow and skin temperature is non-linear[163].
***thermography*** is an indirect measure of skin blood flow, the equipment is cumbersome and impractical in the clinical environment.
***isotope clearance techniques*** are invasive procedures and the assessment area is small.
***dynamic videomicroscopy*** used to measure size and density of capillaries and red blood cell velocity. Not suitable for measurements in the post occlusive reactive hyperaemia period as flow rate changes too quickly[160], the assessment area is small and movement affects the capillaries in view.

***laser Doppler perfusion moniter*** provides relative measure of skin blood flow. Can be used on any body site but requires direct skin contact and assessment area is small ($1mm^3$). Other problems include lack of calibration capability, measurement reproducibility and motion sensitivity.

## 7.4 Laser Doppler Perfusion Imager

The Laser Doppler Perfusion Imager (Moor Instruments Ltd) provides high resolution imaging of Doppler flux (a measure proportional to blood flow) and for the skin provides assessment of the full dermal thickness. It generates a colour image of blood flow made up of individual blood flow values for areas of $2mm^2$, known as pixels (Figure 7.1).

The Laser Doppler Perfusion Imager has a number of advantages including non-contact measurement, assessment of large areas, speed and the ability to function in ambient lighting conditions.

Low power red light (wavelength = 6.32*n*m) is directed to the skin. The incident light is scattered by static tissue and moving blood cells. The Doppler shifted light from moving blood and non-shifted light from tissue is then detected and processed to yield flux. This is an arbitrary value and not a measure of absolute flow - the signal is a product of the number of red blood cells moving in the sample volume and the mean velocity of the moving red blood cells. Because it is neither velocity nor flow the term flux has been adopted [162]. The algorithm used to compute flux is:

$$\text{flux} = K \int_{\omega 1}^{\omega 2} \omega . \frac{P(\omega)\, d\omega}{dc} - \text{noise}$$

where

| | |
|---|---|
| $\omega$ | is the frequency of the Doppler shift |
| $P(\omega)$ | is the power of signal at frequency $\omega$ |
| dc | is the intensity of all detected light |
| $\omega 1$ | is the high pass filter frequency |
| $\omega 2$ | is the low pass filter frequency |
| K | is a scaling constant |
| noise | is the shot and dark noise of the detector |

## 7.5 Pilot Study Method

A prospective cohort study and laser Doppler imaging was undertaken of general and vascular surgical patients admitted to St. James's University Hospital, Leeds between April and July 1998.

### *7.5.1 Inclusion Criteria*

Patients were recruited and written informed consent obtained if they met the following criteria:

a. scheduled for elective major general or vascular surgery.

b. aged 55 years or over on day of surgery.

c. intra-operative position to be supine or lithotomy.

Major surgery was defined as procedures with an average surgical time of 120 minutes or more.

### *7.5.2 Exclusion Criteria*

Three sub-specialities within general surgery were not included (liver, urology and breast surgery). Other exclusion criteria included:

a. planned ICU admission following surgery.

b. laproscopic surgery.

c. dark skin pigmentation which precludes reliable identification of Grade 1a and Grade 1b skin assessments.

d. skin conditions over the sacrum, buttocks or heels, which preclude reliable identification of Grade 1a and Grade 1b skin assessments.

### *7.5.3 Data Collection*

Clinical skin assessments of three sites (sacrum and buttocks) were performed by a research nurse pre-operatively, immediately post-operatively (canvas removal, 0.5-1 hour and 1-1.5 hours) and daily for 8 days (or discharge) using guidelines described by Lowthian (1994) and classified as detailed in Table 7.1.

Laser Doppler imaging of the sacrum and buttocks was performed during the immediate post-operative period (0.5 - 1 hour and 1 – 1.5 hours) subject to post-anaesthetic needs.

***Imaging Technique.*** Laser Doppler imaging was performed by a research nurse. For initial set up various aspects were standardised including the bed height (100 cm), scan head angle (67.5°), scan head height (125 cm) and distance of the scan head from skin (80 cm).

Patients were placed in a lateral position, and the buttock and sacral areas outlined using a laser beam area marker facility. Minor adjustments were made to the scan head angle so that imaging commenced at the bedsheet/skin interface. Imaging then commenced, the laser moving in a raster motion across the skin from the bedsheet upwards.

The single image function on the Moor Instruments LDI (Version 3.01) was utilised and the scan speed standardised to 4 milliseconds/pixel.

### *7.5.4 Outcome Definition*

For the purposes of data analysis Grade 1 skin changes were classified as detailed in Table 7.2. For comparison with laser Doppler imaging, the skin classification as determined at the time of scanning was used. A pressure sore was defined as a skin area assessed as Grade 2 or above.

**Table 7. 1 Clinical Skin Assessment Scale**

| | |
|---|---|
| Grade 0 – No skin discolouration | |
| Grade 1 | a - Erythema – blanching |
| | b - Erythema – non-blanching |
| | c - Pain |
| | d - Induration |
| | e - Heat |
| | f - Oedema |
| | g - Skin discolouration (specify) |
| Grade 2 - | Superficial skin break/blister or partial thickness wound involving epidermis/dermis only |
| Grade 3 - | Full thickness wound involving subcutaneous tissue |
| Grade 4 - | Full thickness wound through subcutaneous tissue to muscle or bone |
| Grade 5 - | Black eschar |

**Table 7. 2 Classification of Grade 1**

| | |
|---|---|
| Grade 1a | Erythema - blanching |
| Grade 1b | Erythema - non blanching |
| Grade 1b+ | Erythema - non blanching plus at least one other physical sign 1c-1g |

## 7.6 Pilot Study Results

A total of 34 patients were recruited during the three month pilot study, with laser Doppler imaging data complete for 25 patients and skin assessment data for 31 patients (2 lost forms and 1 cancelled surgery). Difficulties were encountered in undertaking laser Doppler imaging due to patient factors (post-operative pain and haemodynamic status) and study management factors (such as availability of patient bed and extended theatre time).

Skin classification and mean value for perfusion units for the 25 laser Doppler imaging scans are detailed in Table 7.3. The daily post-operative skin assessments for 31 patients were classified as detailed in Table 7.4. One patient developed a pressure sore.

**Table 7.3 Mean Perfusion Units by Skin Grade**

| Mean Perfusion Units | Skin Grade 0 (n=10) | Skin Grade 1a (n=11) | Skin Grade 1b (n=4) |
|---|---|---|---|
| Minimum | 78.4 | 72.1 | 135 |
| Maximum | 168.5 | 274.3 | 350.3 |
| Mean | 119.2 | 169.1 | 277.7 |
| Standard Deviation | 35.81 | 83.09 | 97.51 |

**Table 7. 4 Post-operative Skin Observations by Grade**

| Skin Grade 0 | Skin Grade 1a | Skin Grade 1b | Skin Grade 1b+ | Skin Grade 2 |
|---|---|---|---|---|
| 10 | 12 | 6 | 2 | 1 |

## 7.7 Variable Identification for Image Analysis

The Moor LDI image processing software package (Version 3.01) was used to quantify various characteristics of the laser Doppler images (or scans). The main scan area was defined using the 'Region of Interest' facility. Where necessary edges were cut to remove bedsheets (if appropriate), areas of leg and perineum (if included within the scan image) and 'interference lines' resulting from movement during imaging. The main scan area was then outlined using the 'box' function and saved,

thus enabling repeated processing using identical image dimensions.

The images were displayed in perfusion units using 'Colour Palette 2', (6 colours at 2,2,3,3,3,3 parts on a 16 increment range: range set from 0 to 1000 perfusion units – values above 1000 are also presented in the top increment colour). Perfusion units (as opposed to relative units) were used to generate all summary values for the image. Perfusion units adjust for distance and normalise for the gain (signal strength).

Summary values were generated for both unsmoothed and smoothed images. Smoothing modifies each pixel according to the 8 neighbouring pixels, by calculation of a weighted average. Various options were explored in order to identify key characteristics or variables to summarise the image and enable discrimination between skin grade.

A common approach is to identify a central and surrounding annular area and compare these to the outer or background area. However, difficulties were encountered due to the variation in size, shape and spread of peak blood flow areas and in some cases complicated by the presence of more than one peak area.

Histograms showed that the distribution of pixels were skewed, with peaks at 0-99 and 100-199 perfusion units, in all cases, suggesting little variability in background values (Figure 7.2). Some histograms were noted to have a second histogram peak and this was a particular characteristic associated with 4 of 4 Grade 1b areas (Figure 7.3).

**Figure 7. 1 Histogram of Pixel Values Showing Typical Distribution**

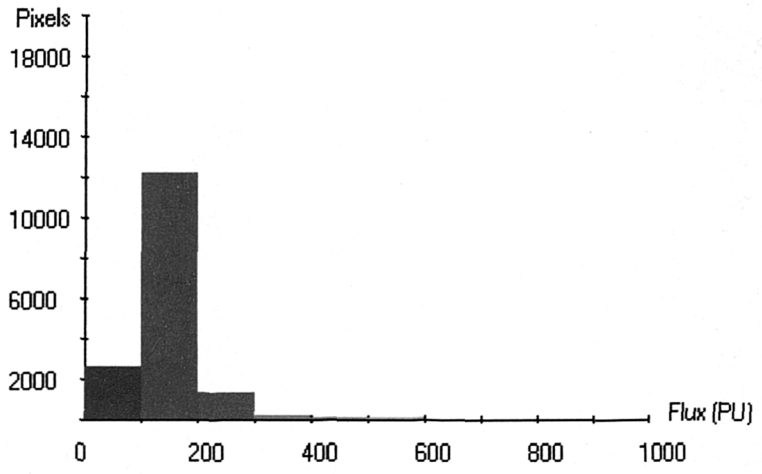
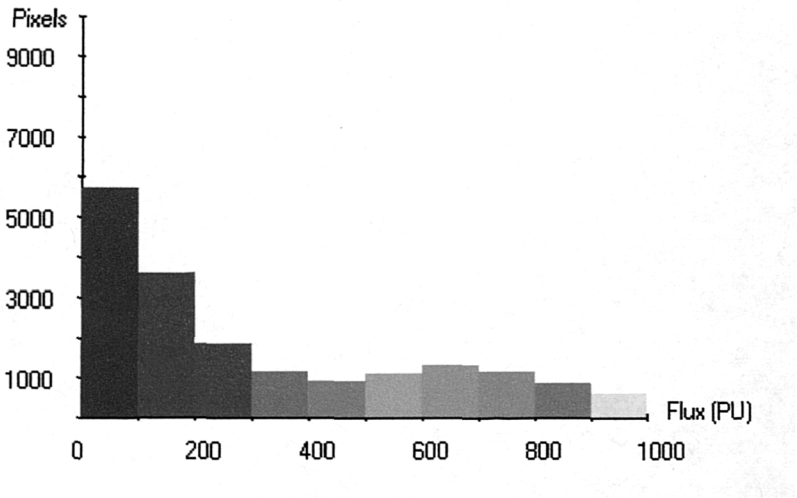



**Figure 7. 2 Histogram of Pixel Values for a Grade 1b Skin Area Showing a Two Peak Distribution**




Customising each scan and cutting ¾ and ½ maximum values did not generate meaningful data particularly in relation to ratio values comparing peak with background. Cutting perfusion units with a value of 500 or less from each scan revealed a clearly defined peak flow area for all those graded clinically as a 1b with variation in 1a and 0 skin areas.

The preliminary processing identified a number of potentially useful summary variables for inclusion in statistical analysis. These included, mean, minimum, maximum, standard deviation and median perfusion unit values for both unsmoothed

and smoothed images, an 11 category histogram (10 equal categories between 0-1000 and 1 category >1000 using smoothed images) and summary histogram variables including 'medium' (proportion of pixels with perfusion unit between 300 and 600) and 'high' (proportion of pixels with perfusion unit greater than 600).

## 7.8 Implications for Main Study

As well as providing data for sample size calculation, the pilot study informed amendments to the study method as follows:

- No patients were observed to have a Grade 1b+ during the immediate post-operative period. Account was taken of this in the main study method by incorporating laser Doppler imaging after patients returned to the ward.
- Patient inclusion criteria were changed to ensure a higher incidence of pressure sores.
- Skin assessments were conducted until discharge from hospital to accurately determine skin changes and pressure sore incidence.

Since the laser Doppler imaging set-up method was unchanged the images obtained during the pilot phase were retained for inclusion in the main analysis.

# Chapter 8 Research Design and Statistical Method

## 8.1 Introduction

Literature review and analysis of secondary data (Chapter 6) identifies gaps in the knowledge base, which are clinically important.

Firstly, existing evidence does suggest that the observation of non-blanching erythema is an important risk factor in pressure sore development but this requires confirmation by further research. There is little evidence pertaining to the role of blanching erythema and its' importance as a risk factor is unknown. In general, prognostic factor research in relation to pressure sore development is limited to Phase 1 studies and requires further development and exploration.

Secondly, in relation to the pathology of pressure sore development it is unclear whether clinically defined erythema - blanching and non-blanching, are physiologically different and whether non-blanching erythema with or without other skin changes (such as local induration, oedema, pain and discolouration) reflect post-occlusive reactive hyperaemia (high blood flow) or irreversible ischaemic damage (low blood flow).

These related issues were investigated by undertaking a prospective cohort study to determine variables predictive of pressure sore development and an exploratory study using laser Doppler imaging of skin changes to identify physiological differences between clinical skin grades. This chapter clarifies the research questions and aims and outlines the main research and statistical methods.

## 8.2 Research Questions and Aims

### *8.2.1 Research Questions*

1. What are the physiological differences between normal skin, blanching erythema, non-blanching erythema with or without other skin changes (such as local induration, oedema, pain and discolouration)?

2. Which clinical signs and symptoms of the skin response are predictive of skin loss?
3. Which variables are independently predictive of pressure sore development?

### *8.2.2 Research Aims*

a. Assess the validity of the clinical grading of erythema by comparison with measurement of skin blood flow using Laser Doppler Imaging
b. Assess the validity of clinical signs of erythema as predictors of pressure sore development
c. Identify variables which independently are predictive of subsequent pressure sore development

## 8.3 Study Design

A prospective cohort study and exploratory laser Doppler imaging was undertaken of patients admitted to St. James's University Hospital, Leeds between September 1998 and May 1999.

### *8.3.1 Inclusion Criteria*

Surgical in-patients at St. James's University Hospital were recruited and written informed consent obtained if they met the following criteria:
a. scheduled for elective major general or vascular surgery OR
   acute orthopaedic, vascular and general surgical admission.
b. aged 55 years or over on day of surgery.
c. expected length of stay of 5 or more days.

Elective major surgery was defined as a planned surgical procedure with an average surgical time of 90 minutes or more.

### *8.3.2 Exclusion Criteria*

a. general surgery sub-specialities including liver, urology and breast surgery
b. dark skin pigmentation which preclude reliable identification of Grade 1a and 1b skin assessments.
c. skin conditions over the sacrum, buttocks or heels which preclude reliable identification of Grade 1a and 1b skin assessments.

### *8.3.3 Data Collection*

***Elective Admissions.*** Elective general and vascular patients were recruited pre-operatively and informed written consent obtained. Skin was assessed pre-operatively, immediately post-operatively and daily until discharge using a combination of clinical and physiological measures as follows:

a. clinical skin assessment - pre-operatively, post-operatively and daily until discharge
b. laser Doppler imaging - 1/2 hour post-operatively, 1 hour post-operatively
c. laser Doppler imaging - purposive ward based imaging on Grade 1b and Grade 1b+ skin areas at any time during hospital stay.

In addition to informed written consent for participation in the study, verbal consent was also obtained immediately prior to laser Doppler imaging.

***Acute Admissions.*** Acute general, vascular and orthopaedic surgical patients were recruited up to 72 hours following admission and informed written consent obtained. Skin was assessed clinically on a daily basis until discharge. If a skin area was assessed as non-blanching, where feasible the area was scanned using the laser Doppler imager (subject to verbal consent as above).

Assessments were undertaken by a research nurse in consultation with the patient and theatre, PACU and ward staff. Skin assessments were performed using guidelines described by Lowthian (1994) and classified as detailed in Table 7.1. Other risk factors which may be predictive of pressure sore development were obtained by clinical assessment and from medical and nursing records.

***Imaging Technique.*** Laser Doppler imaging assessments were undertaken by a research nurse in consultation with the patient and theatre, PACU and ward staff.

The feasibility of ward based laser Doppler imaging was dependant upon:

| | |
|---|---|
| staff availability | porters to assist with inter-departmental transfer of the imager |
| | nursing staff to assist patient repositioning) |
| patient suitability | general status |
| | positioning difficulties |
| | patient agreement |
| staff agreement | ward nursing staff |

Laser Doppler imaging was performed by a research nurse. For initial set up various aspects were standardised including the bed height (100 cm), scan head angle (67.5°), scan head height (125 cm) and distance of the scan head from skin (80 cm).

Patients were placed in a lateral position, and the buttock and sacral areas outlined using a laser beam area marker facility. It was not possible to standardise the position adopted by the patient and therefore the angle of the skin area in relation to the scan head was variable.

Minor adjustments were made to the scan head angle so that imaging commenced at the bedsheet/skin interface. Imaging then commenced, the laser moving in a raster motion across the skin from the bedsheet upwards.

The single image function on the Moor Instruments LDI (Version 3.01) was utilised and the scan speed standardised to 4 milliseconds/pixel.

### *8.3.4 Sample Size*

***Laser Doppler Imaging.*** To detect differences in mean blood flow (perfusion units) between clinical skin grades – Grade 0, Grade 1a, Grade 1b and Grade 1b+, a minimum sample size of 42 scans was estimated using pilot study data (Table 7.3). This was based on an Analysis of Variance with 95% power at the 5% significance level.

***Prognostic Factor Analysis.*** To assess the validity of clinical signs of hyperaemia as predictors of pressure sore development a sample of 300 patients was estimated for the comparison of the proportion of patients classified as having Grade 0, Grade 1a, Grade 1b and Grade 1b+ skin areas who subsequently developed a pressure sore. This was based on a Chi-square test with 80% power at the 5% significance level (two-sided). Data used to calculate sample size included analysis of secondary data in the study of intra-operative pressure sores (Chapter 6, part 6.7) and orthopaedic pressure sore audit data.

Sample size for multi-factoral analyses are difficult to estimate because parameters such as correlations between variables and effect sizes are difficult to obtain. Therefore, to identify variables which are independently predictive of subsequent pressure sore development using regression methods an accepted 'rule of thumb' was applied, that is analysis would include no more than *n*/10 variables, where *n* is the sample size[164].

## 8.4 Outcome Definition

For the purposes of data analysis skin changes preceding pressure sore development were categorised by Grade as detailed in Table 7.2. A pressure sore was defined as a skin area assessed as Grade 2 or above (Table 7.1), that is, a superficial skin break/blister or worse.

For comparison with laser Doppler imaging, the skin classification as assessed at the time of scanning was used.

## 8.5 Statistical Method: Discriminant Analysis of Laser Doppler Imaging

The Moor LDI image processing software package (Version 3.01) was used to

quantify various characteristics of the laser Doppler images (or scans) for both original and smoothed images as detailed in Chapter 7 (part 7.7). All statistical analyses were undertaken using SPSS.

To delineate the factors that would predict the classification scans by skin classification group (Grade 0, 1a and 1b) discriminant analysis was used with independent variables including the mean, minimum, maximum and the standard deviation for perfusion units for both unsmoothed and smoothed scans and summary histogram variables medium (proportion of perfusion units in the range 300-600) and high (proportion of perfusion units greater than 600). The dependent or criterion variable was the clinically assessed skin grade.

Discriminant analysis generates two new variables (discriminant functions) that are a linear combination of the original independent variables, which maximise separation between the skin classification groups. The discriminant scores for each scan were computed by applying the discriminant function formulae. A territorial map was constructed to identify the boundaries used for classifying scans into groups based on the discriminant scores.

To assess how well the discriminant function works, the misclassification rate was calculated for each skin classification group. This was done using the classification rules created to classify the original scans.

Discriminant analysis was repeated using different combinations of independent variables in order to identify those variables which together best predict the clinical classification group.

## 8 6 Statistical Method: Prognostic Factor Analysis

### *8.6.1 Erythema and Pressure Sore Development*

A Chi-square test was used to compare the proportion of pressure sore free patients classified as having Grade 0, Grade 1a, Grade 1b and Grade 1b+ on any skin site who subsequently developed a pressure sore. To identify which clinical signs and symptoms of the skin response were predictive of skin loss, the odds of pressure sore development for Grade 0, Grade 1a, 1b and 1b+ were compared using single factor logistic regression.

Skin changes preceding pressure sore development were classified by Grade, independently for each site, and the difference in frequency of pressure sore between Grades examined using Fisher's exact test. Finally, the natural history of skin changes preceding each pressure sore were mapped for descriptive analysis.

Statistical analyses were carried out using the Stata Statistical Software, Stata Corporation, Release 6.0 (Stata).

### *8.6.2 Multi-factor Analysis*

***Univariate Analysis.*** An association between variables and pressure sore occurrence was explored using single factor logistic regression with a binary response of pressure sore or no pressure sore. Also, *n* by 2 frequency tables and simple summary statistics were used to explore the relationship between the binary response and explanatory variables. Variables were included in the multi-factoral analysis if ≥75% of data was present and where single factor models resulted in a p value of $<0.2$[164].

***Correlation of Variables.*** In a regression model it is not possible to separate the effect of one prognostic variable on outcome from the effect of another where they are both highly correlated. Correlation of explanatory variables is known as colinearity and to avoid this, correlations between variables were examined using Pearson's correlation coefficient for continuous data or Spearmans rank correlation for ordered categorical data. Where variables were correlated with a correlation coefficient of >0.7 and an associated p-value of $<0.01$[94], one was eliminated from further consideration.

***Multi-factor Modeling.*** The final candidate variables were entered into a logistic regression model using forward stepwise selection. The p value determined entry (<0.25) and removal (>0.9). The variables identified by backward stepwise selection were then used as the basic model for further logistic regression analysis. Correlated variables were dropped and added systematically in order to determine the final model in which each variable independently predicted subsequent pressure sore development as assessed by the size of the odds ratio and p value.

The model was determined only from patients with complete data for all candidate variables. Therefore, when the final set of variables was obtained the model was

refitted with only those final variables in the model statement. This ensures a maximum data set for the variables within the model.

Statistical analyses were carried out using the Stata Statistical Software package.

# Chapter 9 Results

## 9.1 Introduction

Patients were recruited to the study from September 1998 to May 1999 as determined by the funding provision for the data collection period. This chapter outlines the main findings of the study.

## 9.2 Laser Doppler Imaging

### *9.2.1 Sample*

Including the patients recruited to the pilot study, a total of 56 laser Doppler images of the sacral and buttock areas were obtained from 40 patients. Six of the laser Doppler images obtained were excluded from analysis due to the presence of an existing pressure sore (5) and psoriasis (1).

A residual sample of 50 laser Doppler images of sacral and buttock areas from 37 patients were included for discriminant analysis. A selection of laser Doppler images and associated clinical Grades` are illustrated in Appendix 3.

The sample comprised 21 women and 16 men admitted for vascular (n=19), general (n=13) and orthopaedic (n=5) surgery. The majority of patients were planned admissions (n=32) and the mean age of the sample was 72.4 years (range 55– 88 years). Prospective follow-up identified that 2 patients developed pressure sores.

Difficulties were encountered in undertaking laser Doppler imaging post-operatively as found in the pilot study due to patient factors (post-operative pain and haemodynamic status) and study management factors (such as availability of patient bed and extended theatre time). Ward based imaging was limited by availability of portering assistance to transfer the imager to the ward, nursing assistance on the ward to position the patient and the patients general condition, ability to maintain a lateral position and willingness to participate.

These problems exacerbated difficulties in obtaining images of 1b and 1b+ skin changes. Eleven of 13 1b+ and 25 of 44 1b skin changes were observed on heels, therefore they were not suitable for laser Doppler imaging. Where imaging of 1b areas were planned in some cases the 1b area had subsequently resolved to a 1a at the time of imaging and in others pressure sore development had occurred.

### *9.2.2 Discriminant Analysis*

Clinical skin classification and associated variables for the 50 scans, after smoothing are detailed in Table 9.1 suggesting differences between skin Grades.

**Table 9. 1 Skin Classification Grade and Variable Parameters for Smoothed Scans**

| Perfusion Units (PU) | Grade 0 n=16 | Grade 1a n=26 | Grade 1b n=8 |
|---|---|---|---|
| Maximum PU | | | |
| Minimum | 264 | 217 | 836 |
| Maximum | 1293 | 1311 | 2115 |
| Median | 484.5 | 972 | 1421 |
| Standard deviation | 262.20 | 365.89 | 468.5 |
| Minimum PU | | | |
| Minimum | 5 | 3 | 1 |
| Maximum | 39 | 43 | 41 |
| Median | 22.5 | 18.5 | 15 |
| Standard deviation | 9.80 | 10.01 | 15.04 |
| Mean PU | | | |
| Minimum | 73.2 | 61.7 | 79.6 |
| Maximum | 168.3 | 273.9 | 339.5 |
| Median | 101.95 | 144.85 | 283.45 |
| Standard deviation | 34.76 | 73.13 | 104.39 |
| Standard deviationPU | | | |
| Minimum | 20.79 | 20.62 | 68.96 |
| Maximum | 80.51 | 252.6 | 371.53 |
| Median | 43.52 | 101.96 | 259.01 |
| Standard deviation | 15.32 | 69.41 | 107.19 |
| Medium | | | |
| Minimum | | | |
| Maximum | 0 | 0 | 0.02 |
| Median | 0.04 | 0.26 | 0.18 |
| Standard deviation | <0.01 | 0.08 | 0.10 |
| | 0.01 | 0.09 | 0.07 |
| High | | | |
| Minimum | | | |
| Maximum | 0 | 0 | <0.01 |
| Median | <0.01 | 0.15 | 0.23 |
| Standard deviation | 0 | 0.01 | 0.14 |
| | <0.01 | 0.04 | 0.09 |

Using discriminant analysis, the independent variables which in combination correctly classified 72% of the scans included smoothed medium (proportion of pixels with PU value between 300 and 600) and smoothed high (proportion of pixels with PU value greater than 600) and are referred to as the 'basic model'.

The pooled within-groups correlations between discriminating variables and discriminant functions are detailed in Table 9.2. and illustrate that Function 1 is correlated with the proportion of high pixels and Function 2 with the proportion of medium pixels.

**Table 9. 2 Basic Model: Correlation of the variables high and medium with discriminant functions 1 and 2**

| Variables | Discriminant Functions | |
|---|---|---|
| | 1 | 2 |
| High | 0.977* | 0.213 |
| Medium | 0.377 | 0.926* |

*largest absolute correlation between each variable and any discriminant function

Results suggest that Functions 1 and 2 can be clearly labelled as follows:

Function 1 larger proportion of high pixels – 'high intensity scans'

Function 2 larger proportion of medium pixels – 'medium intensity scans'

The scans were then plotted using the discriminant scores (Figure 9.1) and classified by group (Table 9.3). Classification compares the clinically assessed grade against the predicted grade based upon the discriminant function scores, for each scan.

Results suggest that 'high intensity scans (large Function 1 or large proportion of high pixels) discriminates between Grade 1b and the others. Those scans that are not high intensity (smaller Function 1) can be classified into Grade 0 and 1a by examining the proportion of medium pixels as follows:

Large Function 2 Grade 1a

Smaller Function 2 Grade 0

**Figure 9. 1 Basic Model: Laser Doppler Images Plotted by Discriminant Functions**

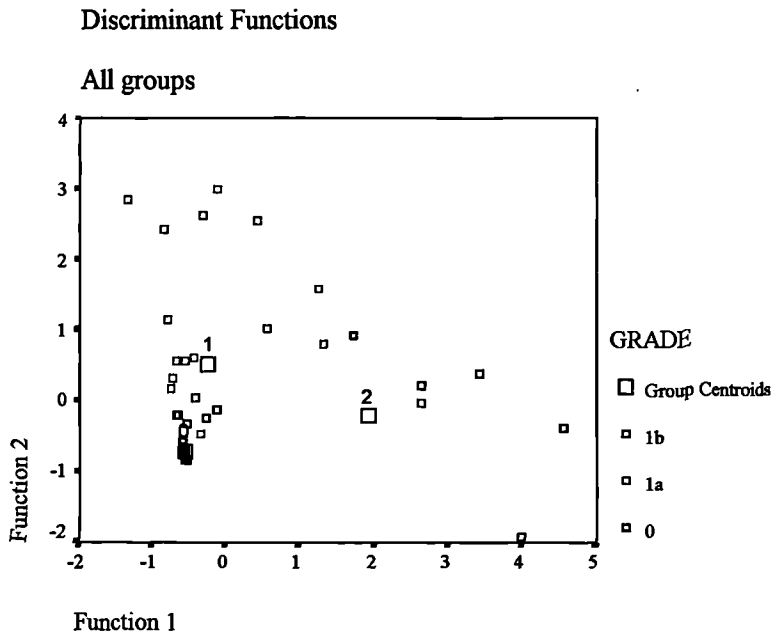



**Table 9. 3 Basic Model: Clinical Skin Classification and Predicted Laser Doppler Image Classification**

| Clinical Classification | Predicted Group Membership | | | Total |
|---|---|---|---|---|
| | 0 | 1a | 1b | |
| 0 | 14 | 2 | 0 | 16 |
| 1a | 8 | 17 | 1 | 26 |
| 1b | 1 | 2 | 5 | 8 |
| Total | 23 | 21 | 6 | 50 |

Addition of further variables improved this basic model and enabled 82% of scans to be correctly classified as illustrated in Figure 9.2 and Tables 9.4 and 9.5 and is referred to as the 'complex model'.

**Table 9. 4 Complex Model: Correlations between Discriminating Variables and Discriminant Functions**

| Discriminant Variables | Discriminant Functions | |
|---|---|---|
| | 1 | 2 |
| Standard Deviation PU | 0.868* | 0.058 |
| Maximum PU | 0.784* | - 0.012 |
| High | 0.767* | 0.360 |
| Mean PU | 0.544* | 0.007 |
| Medium | 0.472 | - 0.480* |
| Minimum PU | - 0.161 | 0.167* |

*largest absolute correlation between each variable and any discriminant function

**Figure 9. 2 Complex Model: Laser Doppler Images Plotted by Discriminant Functions**

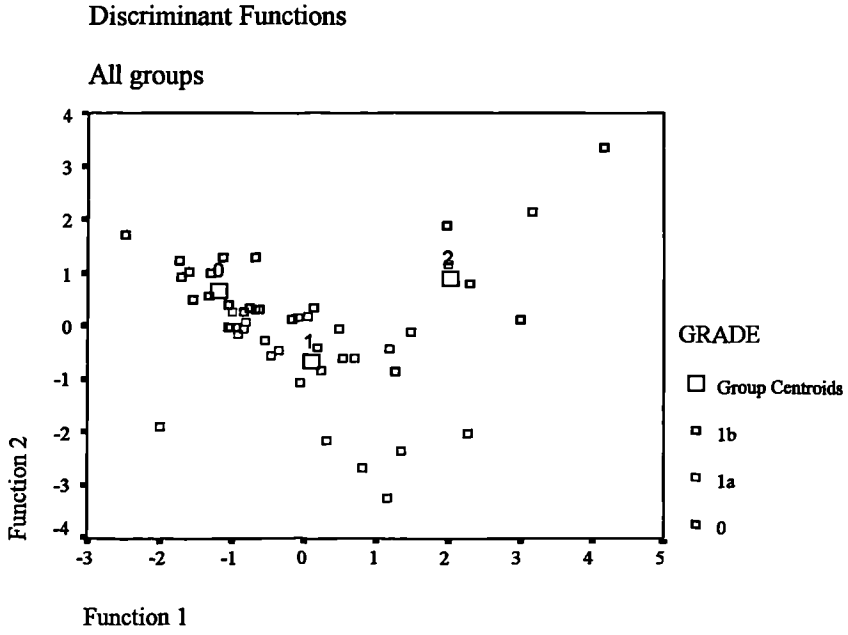



**Table 9. 5 Complex Model: Clinical Skin Classification and Predicted Laser Doppler Image Classification**

| Clinical Classification | Predicted Group Membership | | | Total |
|---|---|---|---|---|
| | 0 | 1a | 1b | |
| 0 | 15 | 1 | 0 | 16 |
| 1a | 4 | 21 | 1 | 26 |
| 1b | 0 | 3 | 5 | 8 |
| Total | 19 | 25 | 6 | 50 |

Interpretation of the discriminant functions are difficult although some comparison can be made to the basic model suggesting that Function 1, which is dominated by the variables standard deviation, maximum and high, represents 'high intensity scans' and Function 2 which is dominated by the variable medium represents 'medium intensity scans'. It is noteworthy that in the complex model there is complete discrimination between Grade 0 and Grade 1b.

## 9.3 Prognostic Factor Analysis

### *9.3.1 Sample*

One hundred and nine patients were recruited to the prospective cohort study and follow-up was complete for 101. Incomplete follow-up resulted from cancelled elective surgery and discharge (2) and patient request to discontinue (4). Patients for whom only one skin assessment was performed due to early death or discharge were also classified as lost to follow-up (2).

The sample comprised 60 women and 41 men admitted for vascular (39), general (22) and orthopaedic (40) surgery. Fifty five patients were planned admissions and 46 acute. The mean age of the sample was 75 years (range 55 – 95 years).

A total of 34 pressure sores were observed on 19 patients during their hospital stay. Four patients had one pressure sore on first assessment and of these three developed further sore(s). The majority of pressure sores observed were superficial sores, with 29 classified as Grade 2, 4 as Grade 4 and 1 as Grade 5. Body sites affected included 14 heels, 11 sacral areas and 9 buttock areas, with all severe sores observed on heels. Duration of superficial sore varied considerably from 1-25 days with resolution of 13 prior to discharge (Table 9.6).

**Table 9. 6 Duration of Grade 2 Pressure Sores**

| Duration of Grade 2 pressure sore | Pressure sores resolved before discharge | Pressure sores present on death | Pressure sores present on discharge |
|---|---|---|---|
| 1 day | 2 | - | 2 |
| 2-3 days | 6 | - | 1 |
| >4 days | 5 | 2 | 11 |
| Total | 13 | 2 | 14 |

### *9.3.2 Erythema and Pressure Sore Development*

Analysis was initially undertaken using only patients who were pressure sore free on entry to the study (n=97). Of these 15 patients (15.5%) developed a pressure sore(s). Chi-square analysis identified significant differences across Grades and subsequent pressure sore development (Table 9.7). Due to the small number of Grade 0 observations single factor logistic regression was undertaken using Grade 1a as the baseline comparison. This procedure identified significantly increased odds

of pressure sore development associated with the observation of Grade 1b and 1b+ (Table 9.8). It also identified increased odds of pressure sore development associated with Grade 0 compared to Grade 1a but this was not statistically significant.

Due to the small number of observations in both the Grade 0 and 1b+ groups the odds of pressure sore development was re-examined after combining Grade 0 with 1a and Grade 1b with 1b+. This identified a significantly increased odds of pressure sore development associated with the observation of Grade ≥1b (Table 9.9).

**Table 9. 7 Skin Changes Preceding Pressure Sore Occurrence**

| Pressure Sore | Grade 0 | Grade 1a | Grade 1b | Grade 1b+ | Total |
|---|---|---|---|---|---|
| No | 6 | 55 | 17 | 4 | 82 |
| Yes | 1 | 3 | 9 | 2 | 15 |
| Total | 7 | 58 | 26 | 6 | 97 |

Chi-square 13.02, p = 0.005

**Table 9. 8 Single Factor Logistic Regression Model of Skin Changes Preceding Pressure Sore Occurrence Compared to Grade 1a**

| | Odds Ratio | p-value | 95% Conf. Interval |
|---|---|---|---|
| Grade 0 | 3.06 | 0.365 | 0.27 to 34.19 |
| Grade 1b | 9.71 | 0.002 | 2.36 to 39.97 |
| Grade 1b+ | 9.17 | 0.035 | 1.17 to 71.71 |

Number of Cases n=97

**Table 9. 9 Single Factor Logistic Regression Model of Skin Changes Preceding Pressure Sore Occurrence Compared to Grade ≤1a**

| | Odds Ratio | p-value | 95% Conf. Interval |
|---|---|---|---|
| Grade ≥1b | 7.98 | 0.001 | 2.29 to 27.80 |

Number of Cases n=97

Due to limitations in the sample size further analysis was undertaken in order to explore the clinically important differences in the incidence of pressure sores between skin Grade. Skin assessment data for all patients (n=101) was classified by

Grade, independently for each site, and the difference in frequency of pressure sore between Grades examined using Fisher's exact test. From a total of 505 skin areas (5 per patient) 10 sites were excluded from analysis due to the presence of an existing pressure sore (n=4) or amputated/bandaged limb (n=6). Four of the five skin sites demonstrated statistically significant differences between the skin Grade and conversion to pressure sore (Table 9.10), with high incidence rates noted for skin areas observed to be non-blanching with or without other signs and symptoms (≥1b).

**Table 9. 10 Skin Changes and Conversion to Pressure Sores**

| Worst Grade | Left Buttock | | Right Buttock | | Sacrum | | Left Heel | | Right Heel | | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | PS | NPS | PS | NPS | PS | NPS | PS | NPS | PS | NPS | PS rate |
| 0 | 0 | 25 | 0 | 28 | 2 | 24 | 1 | 20 | 1 | 21 | 4:118<br>3.28% |
| 1a | 2 | 56 | 2 | 54 | 4 | 57 | 1 | 55 | 1 | 59 | 10:281<br>3.44% |
| 1b | 1 | 15 | 2 | 14 | 4 | 9 | 1 | 13 | 3 | 7 | 11:58<br>15.94% |
| 1b+ | 0 | 1 | 1 | 0 | 0 | 0 | 2 | 4 | 2 | 3 | 5.8<br>38.46% |
| Fishers Exact | p= 0.554 | | p= 0.010 | | p= 0.049 | | p= 0.023 | | p= 0.004 | | |

PS = pressure sore
NPS = no pressure sore

Finally, summary of all skin assessments undertaken on patients who developed pressure sores were mapped (Appendix 4) illustrating important features and variation in the skin response prior to and following pressure sore development. Four skin areas converted directly from Grade 0 to a pressure sore, 3 went through phases of Grade 1a or 1b and resolved to Grade 0 prior to the appearance of a pressure sore and 23 progressed from a Grade 1a, 1b or 1b+ to a pressure sore. Also, the 4 severe sores were preceded by assessments of Grade 1b+ skin changes and Grade 2 pressure sores.

Statistical analyses were carried out using the Stata statistical software package.

### *9.3.3 Multi-factor Analysis*

Multi-factor analysis was undertaken using only patients who were pressure sore free on entry to the study (n=97). Of these 15 patients (15.5%) developed a total of 26 pressure sores including 2 patients who developed 3 severe (Grade 3 and above) pressure sores.

***Univariate analysis.*** The 33 variables explored by univariate analysis are detailed in Table 9.11 and Appendix 5 (Section 5.1). Variables with ≥25% missing data were not included in further analysis (intra-operative temperature, proportion of time hypotensive, number of hypotensive episodes). Other variables with missing data associated with only one type of surgery was also excluded (intra-operative mattress, intra-operative warming mattress and critical ischaemia).

The worst recorded Braden Score and best mattress allocated during hospital stay, preceding pressure sore development were included in the analysis. In ascending order worst to best mattress was defined as high density foam, alternating pressure overlay mattress and alternating pressure replacement mattress[165]. In relation to weight loss, patients were asked whether they had suffered weight loss of greater than 6 Kilograms in the 6 months preceding surgery.

Single factor logistic regression (Table 9.8) using the variable Grade was limited by the small number of patients classified as Grade 0 and 1b+. Analysis indicated no statistical differences between Grades 0/1a or Grades 1b/1b+; therefore the variable Grade was reclassified as two groups combining Grade 0 with 1a and Grade 1b with 1b+.

Variables with an associated p value of <0.2 using single factor logistic regression are detailed in Table 9.12.

**Table 9. 11 Variables Explored by Univariate Analysis**

| |
|---|
| **Patient variables:**<br>Age (years), gender, Braden subscales and total score, existing wound (yes/no), diabetes (yes/no), pre and post-operative serum albumin(g/l), pre and post-operative haemoglobin (g/dl), body mass index, history of weight loss (yes/no), pre-operative temperature (°C), pre-operative blood pressure (mmHg) |
| **Admission variables:**<br>Type of admission (elective/acute), type of surgery (vascular/general/orthopaedic) |
| **Intervention variables:**<br>Ward mattress (foam/alternating overlay/alternating replacement) |
| **Intra-operative variables:**<br>Diastolic and systolic blood pressure - minimum, maximum, final (mmHg), type of anaesthetic (general/spinal and epidural), length of surgery (minutes) |
| **Skin variable:**<br>Grade ≤1a/≥1b |

**Table 9. 12 Variables Associated with Pressure Sore Occurrence (p<0.2).**

| Variable | Odds Ratio | p-value |
|---|---|---|
| Grade ≥1b | 7.99 | 0.001 |
| Type Anaesthetic | 6.91 | 0.013 |
| Weight Loss | 0.26 | 0.047 |
| Acute/Elective Surgery | 2.82 | 0.079 |
| Wounds | 0.34 | 0.113 |
| Pre-operative albumin | 0.85 | 0.011 |
| Pre-operative haemoglobin | 0.60 | 0.016 |
| Post-operative haemoglobin | 0.66 | 0.035 |
| Post-operative albumin | 0.90 | 0.040 |
| Diastolic BP min | 0.95 | 0.084 |
| Diastolic BP max | 0.96 | 0.086 |
| Age at surgery | 1.05 | 0.182 |
| Diastolic BP final | 0.98 | 0.308 |

**Correlations**. Correlations of variables are summarised in Table 9.13 indicating association between many of the variables. Only intra-operative diastolic blood pressure minimum verses final had a correlation coefficient of >0.7 and p-value <0.001, the latter eliminated from further analysis.

However, there remained a large number of variables measuring the same physiological indicators at different points in time and overlap of information in the surgery-related variables. Whilst the use of a stepwise procedure in the multi-factor modeling (as detailed below) does not require exclusion of all variables[164] a pragmatic approach was taken to reduce the number of variables which did not have high correlation coefficients but were significant at p-values of <0.05. The variable with the highest (or lowest) odds ratio and lowest p-value was selected for inclusion in the preliminary modeling process.

The diastolic blood pressure values were correlated and the intra-operative diastolic blood pressure minimum was determined as having the lowest odds ratio and p-value from single factor logistic regression. Similarly, pre-operative and post-operative albumin and haemoglobin were correlated and both pre-operative albumin and pre-operative haemoglobin were selected for initial modeling.

**Table 9. 13 Significantly Correlated Variables**

| Variable | Correlation coefficient | p-value |
|---|---|---|
| Diastolic blood pressure intra-operative min V final | 0.74 | <0.001 |
| Pre-operative albumin V post-operative albumin | 0.58 | <0.001 |
| Diastolic blood pressure intra-operative min V max | 0.57 | <0.001 |
| Pre-operative haemoglobin V post-operative haemoglobin | 0.55 | <0.001 |
| Pre-operative albumin V pre-operative haemoglobin | 0.45 | <0.001 |
| Wounds V pre-operative albumin | 0.40 | <0.001 |
| Pre-operative albumin V age | -0.35 | 0.001 |
| Weight loss V pre-operative albumin | 0.29 | 0.013 |
| Pre-operative haemoglobin V age | -0.27 | 0.010 |
| Weight loss V pre-operative haemoglobin | 0.25 | 0.027 |

Types of anaesthetic and acute/elective surgery were correlated. Type of anaesthetic was determined by single factor logistic regression as being most associated with pressure sore development and this was included in initial modeling.

Finally, although there was an association between the three variables wounds, weight loss and age with serum albumin and haemoglobin all three were included in the initial modeling process because the relationship is known to be multi-factorial[166;167].

***Multi-factor Modeling.*** The final 8 candidate variables detailed in Table 9.14 were analysed using forward stepwise logistic regression. However, difficulties were encountered due to the amount of missing data in relation to the sample size for the variables pre-operative albumin, pre-operative haemoglobin and weight loss. Missing values were replaced by imputed data. Regression models were used to 'predict' the value of the missing items and the predicted values imputed. For example, missing values for pre-operative haemoglobin were predicted by modeling of the variables post-operative haemoglobin, pre-operative albumin, post-operative albumin, pressure sore, body mass index and age. The statistical technique enables prediction of a missing variable on the basis of the value of other known variables for a given patient compared to the remaining sample (Appendix 5, Section 5.2).

Forward stepwise logistic regression using a p-value of <0.25 to determine entry and >0.9 for removal identified the variables grade, pre-operative albumin, weight loss and intra-operative minimum diastolic blood pressure as the variables most associated with pressure sore development (Table 9.15). Details of the multi-factor

analysis and modeling process (using the imputed data for completeness) are detailed in Appendix 5 (Section 5.3).

The addition and substitution of the correlated variables including post-operative haemoglobin, post-operative albumin, wounds, age and acute/elective surgery did not significantly alter the model. Intra-operative maximum diastolic blood pressure was found to have a similar prognostic value as intra-operative minimum diastolic blood pressure (Table 9.16).

Repeating the final model with the original missing data for pre-operative albumin and weight loss resulted in similar odds associated with pressure sore development but reduced levels of significance (Table 9.17).

**Table 9. 14 Final Candidate Variables for Multi-factor Examination - Variable Parameters**

| | Range | Mean | Median |
|---|---|---|---|
| Age | 55.57 – 95.21 years | 75.14 years | 75.89 |
| Type anaesthetic | General / other | | General |
| Grade | ≤1a / ≥1b | | ≤1a |
| Pre-op haemoglobin | 8.9-16.3g/dl | 12.61 | 12.9 |
| Pre-op albumin | 24-48g/l | 37.40 | 39 |
| Weight loss | Yes / No | | No |
| Wounds | Yes / No | | No |
| Diastolic BP min | 15-90 mmHg | 47.60 | 47 |

**Table 9. 15 Prognostic Factors Identified by Forward Stepwise Logistic Regression**

| | Odds Ratio | p-value | 95% Conf. Interval |
|---|---|---|---|
| Pre-op albumin | 0.81 | 0.009 | 0.70 to 0.95 |
| Grade ≥1b | 7.02 | 0.008 | 1.67 to 29.49 |
| Weight loss | 0.29 | 0.092 | 0.07 to 1.22 |
| DiastolicBPmin | 0.96 | 0.205 | 0.90 to 1.02 |

Number of Cases n=90

**Table 9. 16 Logistic Regression Model Substituting Maximum Intra-operative Diastolic Blood Pressure**

| | Odds Ratio | p-value | 95% Conf. Interval |
|---|---|---|---|
| Pre-op albumin | 0.79 | 0.004 | 0.68 to 0.93 |
| Grade ≥1b | 6.12 | 0.013 | 1.46 to 25.75 |
| Weight loss | 0.30 | 0.099 | 0.07 to 1.25 |
| DiastolicBPmax | 0.96 | 0.161 | 0.91 to 1.02 |

Number of Cases n=90

**Table 9. 17 Logistic Regression Model using Original Data with Missing Values**

| | Odds Ratio | p-value | 95% Conf. Interval |
|---|---|---|---|
| Pre-op albumin | 0.85 | 0.074 | 0.71 to 1.02 |
| Grade ≥1b | 6.35 | 0.036 | 1.13 to 35.83 |
| Weight loss | 0.32 | 0.199 | 0.06 to 1.81 |
| DiastolicBPmax | 0.95 | 0.211 | 0.88 to 1.03 |

Number of Cases n=68

Repeating the prognostic factor analysis including the 4 patients with existing pressure sores on admission to the study resulted in similar variable selection (Table 9.18). The existing pressure sore was recorded as an existing wound and pressure sore occurrence defined as new pressure sore development (1 patient developed no further pressure sore, 3 patients did develop further pressure sores). Three of the 4 variables identified were the same as those identified in the primary analysis.

**Table 9. 18 Logistic Regression Model – Including Patients with Existing Pressure Sores**

| | Odds Ratio | p-value | 95% Conf. Interval |
|---|---|---|---|
| Pre-op albumin | 0.85 | 0.025 | 0.74 to .98 |
| Grade ≥1b | 7.44 | 0.004 | 1.91 to 28.97 |
| Type anaesthetic | 8.11 | 0.023 | 1.33 to 49.53 |
| Weight loss | 0.22 | 0.025 | 0.06 to 0.83 |

Number of Cases n=98

## 9.5 Summary of Results

In this study of general, vascular and orthopaedic surgical patients aged over 55 years laser Doppler Imaging of 50 skin areas identified clear differences in blood flow between normal and non-blanching erythema. Discriminant analysis correctly classified 72% of the scans using a basic model including the variables medium and high. Addition of further variables improved this basic model and enabled 82% of scans to be correctly classified.

In the prognostic factor analysis, of 15 factors associated with pressure sore development ($p=<0.2$), logistic regression modeling identified non-blanching erythema as an independent predictor of pressure sore development. Other variables identified by the modeling process included pre-operative albumin, weight loss preceding admission and intra-operative minimum diastolic blood pressure. No difference in the odds of pressure sore development was found to be associated with blanching erythema compared to normal skin.

# Chapter 10 Discussion

## 10.1 Introduction

The results of the study are discussed in relation to the study aims, the consistency of the results compared to previous research, the limitations of the research and methodological issues, which require consideration in further research. Overall results from the laser Doppler imaging and prospective observation of skin changes which preceded pressure sore development concur with respect to establishing that non-blanching erythema is an abnormal physiological response (that is, distinct from normal) and a clinically measurable valid predictor of pressure sore development.

## 10.2 Laser Doppler Imaging

The aim of this exploratory study was to assess the validity of the clinical grading of erythema by comparison with measurement of skin blood flow using Laser Doppler Imaging. Despite variability in scan quality due to patient movement during imaging and the small sample size, good discrimination between clinically assessed Grades was found using summary image data in discriminant analysis. There was 72% agreement between clinically assessed skin Grades and the predicted skin grade in the basic discriminant model comprising the variables medium and high. Indeed the territorial map (Figure 9.1) identifies three general patterns in skin blood flow, which are translated into Grades 0, 1a and 1b as follows:

| | |
|---|---|
| 'high intensity scans' (large Function 1) | → Grade 1b |
| 'medium intensity scans' (large Function 2) | → Grade 1a |
| 'low intensity scans' (small Function 2) | → Grade 0 |

Addition of further variables improved this basic model and enabled 82% of scans to be correctly classified. Interpretation of this complex model is difficult (it is not clear what Functions 1 and 2 represent) and results are presented cautiously due to the small sample size. However, it is noteworthy that all misclassifications were +/- one Grade and in particular no Grade 0 areas were classified as a Grade 1b and vice versa when classification was predicted using the discriminant model.

In terms of misclassification by Grade, differences between high intensity blanching and non-blanching erythema would appear to be difficult to differentiate clinically by applying light finger pressure and this reflects experience in practice and evidence from the pathological examination of skin biopsies which identified similarities and transitional phases and overlap between Grades[149]. Sample size calculations in future research need to consider inclusion of possible independent variables that affect the intensity of reactive hyperaemia (such as age and disease).

The levels of agreement between the clinically assessed skin areas and predicted skin classification using the discriminant models are not as consistent as inter-rater reliability assessments in the study of intra-operative pressure sores (Chapter 4) where all discrepant co-assessments were +/- one grade and 91.5% agreement was observed.

Laser Doppler imaging of the sacral and buttock areas provides a general picture of the physiological range in blood flow values for normal skin and areas of reactive hyperaemia following localised pressure assault in an uncontrolled physical environment. Blood flow ranged from 1 to 2115 perfusion units across skin areas, but the distribution of mean blood flows, ranging from 73 to 339 perfusion units for all scans illustrates little variability in 'normal' skin blood flow despite the absence of any environmental controls (such as ambient temperature) or patient grouping (such as vascular).

Imaging of clinically assessed Grade 1a and 1b areas illustrates that both blanching and non-blanching erythema are characterised by high skin blood flow suggesting that the responses observed are not pathologically different but reflect the capacity of the skin to increase blood flow locally up to 10 fold compared to baseline. No evidence was observed of the 'no-flow phenomenon' associated with endothelial swelling, leukocyte infiltration, red blood cell rouleaux and thrombi formation and localised oedema previously reported by Romanus[151] and Brånemark[152].

The research has important limitations that prevent generalisation of results and leaves one aspect of the research question unanswered. The sample size calculation undertaken assumed that a univariate analysis would discriminate between the three groups. However, image analysis requires consideration of a number of variables simultaneously in order to identify discriminating features. It is suggested that the sample should be at least five times as many subjects per group as the number of

variables to be examined[164], therefore a sample of at least 90 skin sites should have been sought.

Overall, practical difficulties in performing the laser Doppler imaging led to a shortfall in sample size and the need to include all scans (including those obtained in the pilot phase) in the main analysis and the sample size was not sufficient to test the discriminants on an independent sample of images.

There were also difficulties in summarising the images due to the number of different combinations for clinical skin assessment and blood flow patterns. Three skin areas were assessed clinically – sacrum, left buttock and right buttock but image analysis involved summarising each scan as one single area. The image analysis compared the summary statistics for this combined single area to the worst of three Grades allocated clinically. Various patterns emerged for example, in some cases a clinical assessment of three Grade 1a areas was recorded, but the corresponding scan was characterised by only one area of high blood flow. In other cases the corresponding scan was characterised by two areas of high blood flow. Future research will need to develop reliable image processing methods and accurate mapping of clinical assessments to enable separate analysis of multiple high blood flow areas from the same image.

The practical difficulties in performing laser Doppler imaging of critically ill, elderly and immobile patients and the observation of the majority of Grade 1b+ skin areas on heels reduced the potential sample size and resulted in an absence of Grade 1b+ skin areas for analysis. Questions remain about skin blood flow patterns associated with non-blanching erythema with local induration, oedema, pain and/or discolouration and whether this is characterised by high blood flow (post-occlusive reactive hyperaemia) or low blood flow (irreversible ischaemic damage).

Despite the limitations and exploratory nature of the research, discriminant analysis suggests that both blanching and non-blanching erythema are physiologically distinct from 'normal' skin, and clinically, these can be assessed with reasonable accuracy. Also, the good level of agreement found between clinical assessment and a measure of skin blood flow suggests that despite some of the difficulties encountered, this technology may be clinically useful in the assessment of patients with darkly pigmented skin.

## 10.3 Prognostic Factor Analysis

Prognostic factor analysis was undertaken in order to assess the validity of clinical signs of erythema and identify other variables, which are independently predictive of subsequent pressure sore development using a prospective cohort of patients. There was a considerable short-fall in the required sample estimated to assess the validity of clinical signs of erythema in predicting subsequent pressure sore development, but findings remain important and relevant to nursing practice and assessment of patient risk.

### *10.3.1 Sample*

The overall incidence of new pressure sores (Grade 2 or above) of 15.5% is consistent with other studies of major surgical and acute orthopaedic patients, which include the number of patients with superficial skin loss or blister[88;95;96]. The incidence rate was much greater than that reported in the study of intra-operative pressure sores involving general, vascular and gynaecology surgical patients (Table 4.2) reflecting greater specificity in the inclusion criteria for vascular and general surgical patients (expected length of stay of 5 or more days), inclusion of acute orthopaedic patients[96;168] and those with pre-existing Grade 1b/1b+ skin changes[88].

The development of 4 severe sores (Grade 3 and above) in three patients (3/97=3.09%) contrasts the results found in the study of intra-operative pressure sores (Chapter 4) but is consistent with other research which reports pressure sore incidence by Grade for surgical and orthopaedic patient populations. The reported incidence of severe pressure sores range from 0.19% to 9%[11;12;25;86;168 88].

In relation to the Grade distribution from 0 to 1b+ the results provide a comprehensive picture of skin changes which both resolve and/or proceed to pressure sore development. Of particular note is the overall combined incidence of erythema (Grade 1a-1b+). 92.78% (90/97) of patients had erythema which either resolved or preceded pressure sore development during their hospital stay. The results concur with the secondary analysis of the study of intra-operative pressure sores (Chapter 6, Section 6.7) where 94.5% of patients were observed to have a blanching or non-blanching skin area.

The extent of such skin changes was an unexpected finding given the relatively low incidence of erythema ranging from 11.76%-33.71%, reported by other researchers

who include blanching and non-blanching erythema within their classification of pressure sores[12;25;95] Only Marchette et al[86] in their study of Intensive Care patients report a high incidence (60.25%) of 'skin redness'. Similarly, the incidence of non-blanching erythema is much higher than that previously reported by Allman and colleagues[88] and the secondary analysis of the study of intra-operative pressure sores. However, Allman and colleagues[88] recorded skin assessments weekly and reported only the incidence of non-blanching erythema of sacral skin and in the study of intra-operative pressure sores (Chapter 4) the sample included a lower risk population with data collection only to post-operative day 8. These differences mean that results are not directly comparable.

### *10.3.2 Erythema and Pressure Sore Development*

The small sample size was a limitation in establishing the predictive value of clinical signs of erythema in relation to subsequent pressure sore development. This was further limited by the large denominator population of patients who were observed to have a blanching erythema on at least one site during their hospital stay. It is noteworthy that the logistic regression identifies the predictive value of non-blanching erythema as a general risk factor - patients were classified by worst grade for any skin site during hospital stay or preceding the first pressure sore. Differences between Grade 0 (that is, 'normal' skin) and Grade 1a could not be established, nor could differences between Grade 1b and 1b+.

However, results clearly indicate that non-blanching erythema with or without other clinical signs and symptoms are distinct from blanching erythema and predictive of subsequent pressure sore development. This was confirmed by the prognostic factor analysis, which determined non-blanching erythema as an independent risk factor.

Attempts to quantify blanching erythema to reflect differences in the duration of erythema observed, such as, once only verses consecutive days, was unsatisfactory due to the incipient nature of some pressure sores, the small sample size and the variation in recruitment date to the study. For example, if persistent erythema was defined as a Grade 1a for 2 consecutive days, two patients with a blanching erythema on first day assessment and a pressure sore on second day assessment could not be classified as having either transient or persistent erythema, thus reducing the sample size. Also, in some cases the duration of erythema was incomplete because acute patients were recruited to the study up to 3 days following admission and elective patients pre-operatively regardless of admission date. Future

research will need to consider sampling issues and baseline inclusion criteria (such as risk profile and recruitment time in relation to date of admission) and the use of modeling techniques in order to explore the association between risk and duration of blanching erythema.

Results of the single factor logistic regression are reflected in the site specific conversion rates (Table 9.10) which indicate similarities between Grade 0 and Grade 1a skin areas and a much higher incidence of pressure sores associated with non-blanching erythema. Whilst limited because the sites are not independent, the results yield clinically useful and previously unreported similarities in the conversion of skin grade to pressure sore across all sites.

Mapping of skin changes which precede pressure sore development yields important descriptive data from a clinical perspective and highlights the difficulty in assessing the risk associated with skin changes at an individual patient level. Nine pressure sores developed by the second day assessment from a baseline of Grade 0 (n=3), Grade 1a (n=2) and Grade 1b (n=4). Whilst results suggest that the observation of Grade 1b for any duration can be considered a predictor of risk there is difficulty in translating the large number of Grade 1a areas observed into something clinically meaningful due to the high denominator population.

Also, three anomalies are observed, whereby skin changes including Grade 1a and 1b are observed but appear to resolve to a Grade 0 before subsequent skin breakdown. The data are limited by missing skin assessments in two of these cases. However, the observation of the resolution to Grade 0 raises questions regarding the appropriateness of the classification of skin changes by worst grade at any time preceding the first pressure sore and the duration of the 'at risk' period following the observation of skin changes. In the three cases a time lapse of 6-8 days was observed between the recording of a Grade 1a/1b and subsequent skin loss. It is unclear whether such observations are actually important in determination of outcome. Future research will need to consider the implications of missing data and defining the period preceding pressure sore development for sample size and analysis.

The very high pressure sore incidence following the observation of Grade 1b+ does suggest that this has greater predictive validity than Grade 1b but in three of the five cases where pressure sores were preceded by Grade 1b+, this was secondary to the

development of a pressure sore at a different site. Further exploration is inappropriate due to the small sample size and it remains unclear then, whether it is the presence of another pressure sore, which is important or the observation of a Grade 1b+.

A review of other skin changes which preceded pressure sore development indicates that for the majority of pressure sores there is an obvious pattern of progressive change observed over a period of days and that pressure sore development occurs due to prolonged exposure to pressure assault. Eight pressure sores were preceded by 2-5 assessments of erythema and twelve were preceded by 8 or more assessments of erythema ranging from Grade 1a to 1b+. However, sample size and missing data precludes further exploration of the relationship between duration of erythema and subsequent pressure sore development.

It is also noteworthy that the 4 severe sores were preceded by assessments of Grade 1b+ skin changes and Grade 2 pressure sores, that is, clear evidence of damage to the area preceded the development of a severe sore.

### *10.3.3 Multi-factor Analysis*

The overall sample size and distribution of characteristics within the sample were limitations in establishing variables whose presence are independently predictive of pressure sore development. Whilst the sample size did appear to be sufficiently large using the accepted 'rule of thumb' of *n*/10 variables, where *n* is the sample size[164], a number of problems were encountered and it is possible that the data are not representative and important effects have been missed.

The distributions of some variable parameters were skewed by type of surgery. There were insufficient data within some categorical variables to undertake meaningful analysis and categories were combined (for example, Grade). Also, missing data reduced the sample available within the multi-factoral modeling process, which requires complete data for all candidate variables. As a result, difficulties were encountered in determining risk associated with mobility, type of anaesthetic and nutritional indicators including serum albumin, serum haemoglobin and weight loss.

Furthermore, difficulties were encountered in collecting reliable and valid data for the variable critical ischaemia as well as variables found to be predictive of outcome in

the study of intra-operative pressure sore prevention and prediction (including proportion of time hypotensive and intra-operative temperature). In relation to critical ischaemia, diagnosis was determined first by patient history. If patients had a history of claudication and pain at rest for greater than 2 weeks then diagnosis of critical ischaemia required an ankle brachial pressure of less than 50mmHg. These were not recorded in a large number of vascular patients with the clinical symptoms and therefore a diagnosis was not possible for the purposes of this research.

With respect to intra-operative blood pressure, data quality was determined by the normal anaesthetic monitoring and recording methods for the speciality/surgical procedure and these were variable. Monitoring techniques included the use of invasive arterial manometers which provide output on a minute by minute basis and electronic sphygnomanometers which are programmed by the anaesthetist to record blood pressure at 10-15 minute intervals. Recording techniques also varied with general and vascular surgical patients monitoring computerised and stored providing a detailed post-operative record, either on disc or paper print-out. However, it was common practice in acute orthopaedics for blood pressure readings to be manually recorded at 15 minute intervals and the values were frequently rounded to the nearest 10mmHg. It was not possible, therefore, to accurately calculate a hypotensive episode using a diastolic of 60mmHg and a time period of 10 minutes.

In relation to intra-operative temperature, this was not routinely monitored and recorded in any specialty and relied on the time and good will of theatre staff to set up monitoring. Whilst this was achieved in the elective surgical population further problems were encountered in the computerised storage and retrieval of the data. Without any paper record of the monitoring, data sets were lost due to down loading errors. In the acute surgical population setting up intra-operative monitoring arrangements was impractical due to the variation in pre-operative admission period and frequent changes in the theatre schedules. So, despite the potential importance of this variable in determining pressure sore outcome (Chapter 4), missing data prevented inclusion of this variable in the analysis. The difficulties highlight research design issues associated with prognostic factor research involving both acute and elective surgical populations where variables include detailed intra-operative data.

The 8 candidate variables for multi-factoral examination (Table 9.14) are consistent with findings from other studies, which have utilised multi-variate analyses (Table 2.1) although no mobility related factors were found to be associated with pressure

sore development. With the exception of type of anaesthetic, the candidate variables including age, skin grade, haemoglobin, albumin, weight loss, wounds and intra-operative diastolic blood pressure (minimum) are either direct or indirect measures associated with the key themes identified as risk factors in pressure sore development including poor nutrition, factors affecting perfusion, increased age and skin condition (Chapter 2, section 2.6).

In relation to activity and mobility, the patient population was relatively homogenous with bedfast and chairfast patients (activity score 1 or 2) accounting for 86 of 99 patients and 83 of 97 patients with a mobility score of either 2 or 3. Further analysis and addition of the Braden activity and mobility scores did identify a trend between immobility and pressure sore development. 20% (12/48) patients with a combined Braden activity and mobility score of 2-4 developed sores, whereas 12.82% (5/39) patients with a combined score of 5-8 developed sores, but this association was not statistically significant.

As reported in other studies[77-81] the Braden Scale did not discriminate well between patients who did or did not develop pressure sores when analysed as a continuous variable or categorical variable (≤16, >17). Again the scores reflected the homogenous nature of the patient population with a majority of patients (76/99) scoring from between 13 and 18.

The relationship between type of anaesthetic (general verses spinal/epidural) and pressure sore development is likely to reflect existing co-morbidity. Spinal and epidural anaesthesia are administered for various purposes including induction and maintenance of a controlled hypotension (for example, during vascular surgery) and due to associated co-morbidity (for example, in acute surgery). It is suggested that spinal/epidural anaesthesia increases risk by reducing both blood pressure and mobility[169]. Further exploration determined that administration of spinal or epidural anaesthesia was limited to a very small number of acute orthopaedic and vascular surgical patients. Sample size and clinical indication for administration preclude any meaningful exploration of relationships to intra-operative hypotension and mobility and no conclusions can be made regarding its importance as an independent risk factor.

As reported in the results due to the amount of missing data in relation to the sample size for the variables pre-operative albumin, pre-operative haemoglobin and weight

loss missing values were replaced by imputed data. Regression models were used to 'predict' the value of the missing items and these were used in the subsequent modeling process. This assumes that missing data is random and the calculation error is unknown.

Despite limitations of data size the final logistic regression model is consistent with findings from other studies which have utilised multi-factoral analyses (Table 2.1) and validates previous research by Allman and colleagues[88]. Non-blanching erythema is clearly identified as an independent predictor of pressure sore development and a key prognostic factor. Together with the literature review (Chapter 2) which identifies prognostic factor themes the work sets out a framework for further Phase 2 and 3 prognostic factor research.

## 10.4 Summary

In this study involving 50 laser Doppler images of skin and a prospective cohort study of 101 acute and elective orthopaedic, vascular and general surgery patients non-blanching erythema was found to be distinct from 'normal' skin and an independent predictor of pressure sore development.

Laser Doppler imaging provides a general picture of the physiological range in blood flow values for normal skin and areas of reactive hyperaemia following localised pressure assault in an uncontrolled physical environment. The distribution of mean blood flows for all scans illustrates little variability in 'normal' skin blood flow. Imaging also determined that both blanching and non-blanching erythema are characterised by high blood flow, of differing intensity suggesting that the responses observed are not pathologically different but reflect the capacity of the skin to increase blood flow locally up to 10 fold compared to baseline. There was no evidence of the 'no flow' phenomenon, and the study is limited by the inability to image Grade 1b+ skin areas.

Discriminant analysis identified three general patterns in skin blood flow, which enabled scan classification with good agreement between clinical and predicted classifications. In terms of misclassification by Grade, differences between high intensity blanching and non-blanching erythema would appear to be difficult to differentiate clinically by applying light finger pressure and this reflects experience in practice.

The prognostic model derived from the study validates previous research, which identifies non-blanching erythema as an independent predictor of pressure sore development. Other prognostic factors identified including pre-operative serum albumin, minimum intra-operative diastolic blood pressure and history of weight loss are consistent with findings from other studies which have utilised multi-variate analyses and the prognostic factor themes identified in Chapter 2. The findings of the work provide a framework for further Phase 2 and 3 prognostic factor research and further challenge the use of poorly constructed risk assessment scales in practice.

# Chapter 11 Summary and Recommendations

## 11.1 Introduction

This chapter provides a summary of key elements of the literature which informed the programme of research and presents a summary of the results and recommendations for the programme as a whole in relation to intra-operative pressure sore prevention, predicting pressure sore development and the classification and definition of pressure sores. The literature review and research results together provide new evidence in relation to the debates regarding predicting pressure sore development and the classification and definition of pressure sores, provide direction for further research and have implications for nursing practice.

## 11.2 Summary of Literature Review

### *11.2.1 Intra-operative pressure sore prevention*

A literature review of intra-operative pressure sore development suggested a causal relationship between events occurring during surgery and the subsequent development of pressure sores[43;44]. However, with one exception published prospective studies fail to specify research design or include specific factors on pressure sore aetiology.

Many hospital pressure sore prevention policies include practice recommendations for operating theatres such as pre-operative risk assessment and provision of equipment from a limited range designed for use on operating tables. However, there is no evaluation of the relative effectiveness of intra-operative interventions in reducing or preventing pressure sores.

### *11.2.2 Prediction of Pressure Sores*

In the literature concerning the prevention and management of pressure sores, baseline assessment is commonly associated with the term 'risk assessment' and there has been a focus toward the development and use of risk assessment scales to facilitate the identification of 'at risk' patients.

Risk assessment scales, whilst limited in construction methods and validity, may provide a framework and appropriate prompts for assessment of pressure sore risk but their use as a single instrument to assess risk is not supported by current evidence. The research need is not validation of existing risk assessment scales, but identification of key prognostic factors using statistical modeling to develop the content of predictive tools or frameworks that support assessment processes.

A review of prognostic factor research identifies important themes and key factors in the prediction of pressure sore development including reduced mobility, nutrition, perfusion, age and skin condition. These can be directly related to the aetiology of pressure sore development where the interaction between the intensity and duration of pressure (mobility) and the tolerance of the skin (nutrition, perfusion and age) determines the skin response (skin condition) and provide a framework for further investigation.

### *11.2.3 Classification and definition of pressure sores*

A review of the mechanisms that protect the skin microvasculature from ischaemic assault and restore local tissue perfusion following occlusion illustrates clearly that there is an interaction between the pressure assault and the capacity of the skin to maintain and effectively restore skin blood flow. A number of auto-regulatory mechanisms exist to protect the skin from pressure assault and these processes break down at pressure values that are highly variable. Pressure sore development is multidimensional and complex.

A detailed review of the pathology of pressure sore development suggests three types of pressure sore with possibly three mechanisms that lead to tissue breakdown. A limitation of current research is the difficulty in replicating the clinical situation and in determining the point at which the ischaemic assault becomes irreversible and results in tissue breakdown. This means that classifying and defining the term 'pressure sore' has a poor evidence base, particularly in relation to the classification of erythema.

Whilst pathological differences between 'normal' skin, blanching erythema and non-blanching erythema are reported by Witkowski and Parish[149] clinical observations of blanching and non-blanching erythema have not been validated against physiological measures of skin perfusion or in relation to subsequent skin loss, and their clinical significance is not fully understood. It is not possible to determine whether blanching

and/or non-blanching erythema are indicators of risk and it is unclear whether non-blanching erythema with or without other skin changes reflects post-occlusive reactive hyperaemia or irreversible ischaemic damage.

## 11.3 Summary of Research Findings

### *11.3.1 Intra-operative pressure sore prevention*

In the randomised controlled study of 446 patients undergoing vascular, general and gynaecology surgery the use of a dry visco-elastic polymer pad intra-operatively reduced the probability of pressure sore development by half. Pressure sore incidence was 11% (22/205) for patients allocated to the dry polymer pad and 20% (43/211) for patients allocated to the standard operating table mattress. Although the effect was modified by the variables centre, proportion of time hypotensive, length of surgery and pre-operative length of stay, the effect of the dry visco-elastic polymer pad remained statistically significant.

Inter-rater reliability was measured during the study and 72/851 (8.5%) discrepant co-assessments were observed. Sensitivity analyses accounting for skin assessment variation determined that the effect of the dry visco-elastic pad in reducing post-operative pressure sore incidence remained statistically significant.

It is noteworthy that the majority of endpoint failures were persistent blanching hyperaemia and it remains unclear whether this is a valid endpoint for intervention studies (see 11.3.3). Sample size precludes secondary endpoint analysis using non-blanching erythema or skin break.

Of the 720 patients potentially eligible for the study 74 were excluded due to existing pressure damage, a pre-operative prevalence of 10.3%. The pre-operative prevalence of pressure sores and poor documentation raises serious issues for theatre teams and indicates the need for pre-operative screening of 'high risk' groups and documentation of pre-operative skin condition. It also highlights the need for further research to establish the effectiveness of intra-operative interventions in the prevention of further pressure damage.

### *11.3.2 Prediction of pressure sores*

Both studies explored key prognostic factors using multi-variate methods.

Analysis of data derived from the randomised controlled trial involving 446 patients aged over 55 years and undergoing vascular, general and gynaecology surgery found twelve factors to be significantly associated with post-operative pressure sore development at the p value of <0.01. Prognostic factors identified by logistic regression modeling included intra-operative hypotensive episodes, Day 1 Braden mobility scale and intra-operative mean core temperature. The prognostic model derived from this study was exploratory in nature and limited to the peri-operative period – the primary endpoint only up to day 1 post-operatively.

The results, whilst consistent with the research literature relating to prognostic factors, are not generalisable due to the inclusion of persistent blanching erythema in the endpoint definition (see 11.3.3). However results were important in informing further research priorities.

In relation to risk assessment, the logistic regression modeling found three of the sub-scales of the Braden Scale were highly correlated and explained much of the same variability, measuring the same risk factor expressed in different ways. Together with the development of a probability model, results highlight further the limitations of previous methods employed to develop risk assessment scales.

The second study was a prospective cohort study involving 101 general, vascular and orthopaedic surgical patients aged over 55 years. Prognostic factor analysis of 15 factors associated with pressure sore development (p=<0.2) was conducted and logistic regression modeling identified non-blanching erythema as an independent predictor of pressure sore development. Other variables identified by the modeling process included pre-operative albumin, weight loss preceding admission and intra-operative minimum diastolic blood pressure. No difference in the odds of pressure sore development was found to be associated with blanching erythema compared to normal skin and Braden Scale scores were found not to be associated with subsequent skin loss.

Limitations of the study include the small sample size and missing data for some key variables. However, results are consistent with findings from other studies which have utilised multi-variate analyses.

### *11.3.3 Classification and definition of pressure sores*

Fifty laser Doppler images of sacral and buttock areas from 37 general, vascular and orthopaedic surgical patients aged over 55 years were collected. Discriminant analysis correctly classified 72% of the scans using a basic discriminating model including the variables medium (proportion of pixels with perfusion unit between 300 and 600) and high (proportion of pixels with perfusion unit greater than 600). Addition of further variables improved this basic model and enabled 82% of scans to be correctly classified.

Discriminant analysis identified three general patterns of skin blood flow, which enabled scan classification with good agreement between clinical and predicted classifications. In terms of misclassification by skin assessment scale, differences between high intensity blanching and non-blanching erythema would appear to be difficult to differentiate clinically by applying light finger pressure. This reflects experience in practice.

Despite the limitations and exploratory nature of the research laser Doppler imaging provided a general picture of the physiological range in blood flow values for normal skin and areas of reactive hyperaemia following localised pressure assault in an uncontrolled physical environment. The distribution of mean blood flows for all scans illustrates little variability in 'normal' skin blood flow. Imaging also determined that both blanching and non-blanching erythema are characterised by high blood flow of differing intensity, suggesting that the responses observed are not pathologically different but reflect the capacity of the skin to increase blood flow locally up to 10 fold compared to baseline. There was no evidence of the 'no flow' phenomenon, but the study was limited by the inability to image Grade 1b+ skin areas.

The prospective cohort study was limited by the small sample size in establishing the predictive value of clinical signs of erythema in relation to subsequent pressure sore development. This was further limited by the large denominator population of patients who were observed to have a blanching erythema on at least one site during their hospital stay. Attempts to quantify blanching erythema, to reflect differences in the duration of erythema observed, was unsatisfactory due to the incipient nature of some pressure sores and the variation in recruitment date to the study. Differences between Grade 0 (that is, 'normal' skin) and Grade 1a could not be established, nor could differences between Grade 1b and 1b+. However, results clearly indicate that non-blanching erythema with or without other clinical signs and symptoms are distinct

from blanching erythema and predictive of subsequent pressure sore development. This was confirmed by the prognostic factor analysis, which determined non-blanching erythema as an independent risk factor.

The results confirm the analysis of secondary data (Chapter 6, Section 6.7) suggesting that non-blanching erythema is not indicative of irreversible ischaemic damage and resolves in approximately two thirds of cases. They also concur with the laser Doppler imaging of skin blood flow which suggests that blanching and non-blanching erythema are not pathologically different. The point at which non-blanching erythema becomes irreversible remains unknown.

## 11.4 Recommendations

### *11.4.1 Intra-operative pressure sore prevention*

This study, together with a wider body of knowledge derived from a systematic review of pressure sore prevention equipment[1] provides evidence that low technology constant pressure supports are effective in reducing pressure sore incidence when compared with standard mattress provision in theatre.

Pre-operative skin assessment is not common practice and risk assessments are made using a 'recognised' scale or on the basis of type of surgery.
**It is recommended that theatre staff undertake pre-operative skin assessment of known high risk groups and provide low technology constant pressure mattresses for the prevention of erythema in 'at risk' patients (see 11.4.2).**

The pre-operative prevalence of 10.3% highlights the need for further research to establish the effectiveness of intra-operative interventions in reducing the conversion of non-blanching areas to skin breaks and, where skin loss has occurred, the prevention of further pressure damage.

***It is recommended that further trials, which aim to establish the relative effectiveness of intra-operative interventions, define broader inclusion criteria, allowing entry of patients with non-blanching erythema and existing pressure sores.***

### *11.4.2 Prediction of Pressure Sores*

The research findings and results of other studies, which identify key prognostic factors using multi-variate methods, highlight the limitations of current risk assessment scales. The components within these scales do not match key prognostic factors identified, rarely include skin variables and their predictive validity is inconsistent. This raises issues for research and practice.

**It is recommended that future research include:**

a) **a systematic review and meta-analysis of prognostic factor research to validate and refine prognostic factor themes and identify a minimum data set of variables for further prognostic factor research**
b) **phase 2 prognostic factor studies of sufficient size to determine which factors provide improved means of identifying patients at particularly high or low risk of pressure sore development**
c) **prognostic factor research uses the outcome definition for the term pressure sore as 'superficial skin break/blister or partial thickness wound involving epidermis/dermis'.**

In relation to practice, the programme of work requires a review of guidelines for the assessment of risk such as those published in the USA[23] and UK[170] which do not link skin assessment to the risk assessment process.

**It is recommended that key prognostic factors and skin assessment are linked directly to the risk assessment processes.**

### *11.4.3 Definition and classification of pressure sores*

A distinction is required between the clinical assessment and classification of skin changes for practice and research purposes and the definition of the term pressure sore.

***Blanching erythema*** In relation to blanching erythema, the need to include such observations in a skin classification system and record this information in practice requires some debate. The skin response of patients preceding pressure sore development (Appendix 4) and high incidence of blanching erythema in the sample, illustrate the difficulties in translating the observation of blanching erythema into a meaningful risk factor.

Overall the evidence relating to blanching erythema as a risk factor is limited. This cohort study did not identify blanching erythema as predictive of subsequent

pressure sore development. In the study of intra-operative pressure sore prevention and prediction, analysis of secondary data identified an increased odds of pressure sore development associated with persistent blanching erythema (2 consecutive days or more) but this was not significant (Chapter 6, Section 6.7). Other research has identified blanching erythema as a predictor of 'non-blanching erythema and Grade 2 pressure ulcers' using multi-factoral methods but the study was limited by sample size, outcome definition and the population studied was atypical, that is, all patients were incontinent[90].

There is evidence that there are pathological differences between normal skin and blanching erythema[149] and laser Doppler imaging identified blood flow patterns distinct from 'normal'.

The mapping of skin changes which preceded pressure sore development (Appendix 4) indicates that for the majority of pressure sores there is an obvious pattern of progressive change observed over a period of days and that pressure sore development occurs due to prolonged exposure to pressure assault. Fourteen skin areas had 4 or more assessments of blanching erythema recorded prior to non-blanching erythema/pressure sore development suggesting that persistent erythema may be an important indicator of risk factor.

**On balance it is recommended that:**

**a) blanching erythema is included in skin classification systems and recorded in practice**

**b) blanching erythema is not included within the definition of the term pressure sore or as an outcome measure for research and audit.**

In relation to research, it remains unclear whether blanching erythema is predictive of subsequent pressure sore development.

**It is recommended that further research is undertaken to explore the association between duration of blanching erythema and pressure sore development, addressing design issues such as sample size, inclusion criteria (such as risk profile and recruitment time in relation to date of admission) and the use of modeling techniques.**

***Non-blanching erythema*** Non-blanching erythema is an independent predictor of pressure sore development. However, it is not indicative of irreversible ischaemic damage and resolves in approximately two thirds of cases. The point at which it

becomes irreversible remains unknown. The use of current clinical assessment techniques (light finger pressure) do not discriminate well between high intensity blanching and non-blanching erythema.

**It is recommended, therefore, that:**

a) **non-blanching erythema is not included within the definition of the term pressure sore for the purposes of prognostic factor research**
b) **judgement is required in order to assess the appropriateness of including non-blanching erythema within outcome definitions for the purposes of intervention studies and audit**
c) **non-blanching erythema be recorded as an independent risk variable in research which aims to identify factors predictive of pressure sore development**
d) **practical technical applications are sought to improve assessment of non-blanching erythema in practice across all body sites and skin pigmentations.**

Non-blanching erythema is an important predictor of subsequent pressure sore development and should, therefore be recorded in practice to assess risk and moniter nursing interventions.

**It is recommended therefore, that skin classification systems used in practice include non-blanching erythema.**

***Non-blanching erythema plus other clinical signs and symptoms*** Whilst predictive of subsequent pressure sore development, the underlying pathology associated with non-blanching erythema plus other clinical signs and symptoms such as localised induration, pain, oedema and discoloration remains unclear.

**It is recommended that:**

a) **skin blood flow patterns associated with Grade 1b+ are assessed**
b) **the relationship between biochemical markers of reperfusion injury and the observation of Grade 1b+ are explored.**

## Reference List


1. Cullum N, Deeks J, Fletcher A, Long A, Mouneimne H. Sheldon T *et al.* The prevention and treatment of pressure sores: How effective are pressure-relieving interventions and risk assessment for the prevention and treatment of pressure sores? *Effective Health Care Bulletin* 1995;**2**:pp 1-16.

2. McGough AJ. A systematic review of the effectiveness of risk assessment scales used in the prevention and management of pressure sores. 1999. University of York. *Unpublished MSc Thesis*

3. Cullum N, Nelson EA, and Nixon J. Pressure Sores. Clinical Evidence A compendium of the best available evidence for effective health care Issue 1(June 1999), pp 211-217. 1999. London, BMJ Publishing Group.

4. Parish LC, Witkowski JA, Chrissey JT. The decubitus ulcer. New York: Masson Publications, 1983.

5. Bridel J. The epidemiology of pressure sores. *Nursing Standard* 1993;**7**:pp 25-30.

6. Bergstrom N, Braden B. A prospective study of pressure sore risk among institutionalized elderly. *Journal of the American Geriatrics Society* 1992;**40**:pp 747-758.

7. Thomas DR, Goode PS, Tarquine PH, Allman RM. Hospital-acquired pressure ulcers and risk of death. *Journal of the American Geriatrics Society* 1996;**44**:pp 1435-1440.

8. Clough NP. The cost of pressure area management in an intensive care unit. *Journal of Wound Care* 1994; **3**:pp 33-35.

9. Berlowitz DR, Wilking SVB. The short-term outcome of pressure sores. *Journal of the American Geriatrics Society* 1990;**38**:pp 748-752.

10. DOH. Pressure sores - a key quality indicator: a guide for NHS purchasers and providers. London: DOH, 1993.

11. Clark M, Watts S. The incidence of pressure sores within a National Health Service Trust hospital during 1991. *Journal of Advanced Nursing* 1994;**20**:pp 33-36.

12. Kemp MG, Keithley JK, Smith DW, Morreale B. Factors that contribute to pressure sores in surgical patients. *Research in Nursing and Health* 1990;**13**:pp 293-301.

13. Ek A-C, Unosson M, Larsson J, Von Schenck H, Bjurulf P. The development and healing of pressure sores related to the nutritional state. *Clinical Nutrition* 1991;**10**:pp 245-250.

14. Versluysen M. How elderly patients with femoral fracture develop pressure sores in hospital. *British Medical Journal* 1986;**292**:pp 1311-1313.

15. Barbenel JC, Jordan MM, Nicol SM, Clark MO. Incidence of pressure sores in the Greater Glasgow Health Board. *Lancet* 1977;**ii**:pp 548-550.

16. Barton AA, Barton M. The Pressure Sore. *The management and prevention of pressure sores*, pp 26-38. London: Faber, 1981.

17. Shea JD. Pressure sores: classification and management. *Clinical Orthopaedics* 1975;**112**:pp 89-100.

18. David JA, Chapman RG, Chapman EJ, Lockett B. An investigation of the current methods used in nursing for the care of patients with established pressure sores. UK: Northwick Park: Nursing Research Unit, 1983.

19. Torrance C. Pressure sores: Aetiology, treatment and prevention. London: Croom Helm, 1983.

20. International Association of Enterostomal Therapy. Dermal wards: pressure sores. Philosophy of the IAET. *Journal of Enterostomal Therapy* 1988;**15**:pp 4-17.

21. Lowthian P. Classification and grading of pressure sores. *Care Science and Practice* 1987;**5**:pp 5-9.

22. Lyder CH. Conceptualization of the stage I pressure ulcer. *Journal of Enterostomal Nursing* 1991;**18**:pp 162-165.

23. AHCPR (Agency for Health Care Policy and Research). Pressure ulcers in adults: prediction and prevention: quick reference guide for clinicians. Rockville: U.S. Department of Health and Human Services, 1992.

24. Reid J, Morison M. Towards a consensus: classification of pressure sores. *Journal of Wound Care* 1994; **3**:pp 157-160.

25. Gendron F. Burns occurring during lengthy surgical procedures. *Journal of Clinical Engineering* 1980;**5**:pp 19-26.

26. Vermillion C. Operating room acquired pressure ulcers. *Decubitus* 1990;**3**:pp 26-30.

27. Bridel J. The pathophysiology of pressure sores. *Journal of Wound Care* 1993;**2**:pp 230-238.

28. Braden B, Bergstrom N. A conceptual schema for the study of the etiology of pressure sores. *Rehabilitation Nursing* 1987;**12**:pp 8-16.

29. Bennett L, Lee BY. Pressure versus shear in pressure sore causation. In Lee BY, ed. *Chronic ulcers of the skin*, New York, USA: McGraw Hill, 1985.

30. Bliss MR. Aetiology of pressure sores. *Reviews of Clinical Gerontology* 1993;**3**:pp 379-397.

31. Sacks AH. Theoretical prediction of a time-at-pressure curve for avoiding pressure sores. *Journal of Rehabilitation and Development* 1989;**26**:pp 27-34.

32. Guyton AC. Local control of blood flow by the tissues, and humoral regulation. In Guyton AC, ed. *Human Physiology and Mechanisms of Disease*, pp 136-142. USA: W.B. Saunders Co. Ltd, 1992.

33. Kosiak M. Etiology and pathology of ischaemic ulcers. *Archives of Physical Medicine and Rehabilitation* 1959;**40**:pp 62-69.

34. Groth KE. Klinische beobachtungen und experimentelle studien uber die enstehung des dekubitus. *Acta Chirurgia Scandinavica* 1942;**87**:p 198 (English Summary).

35. Dinsdale SM. Decubitus ulcers in swine: light and election microscopic study of pathogenesis. *Archives of Physical Medicine and Rehabilitation* 1973;**54**:pp 51-56, 74.

36. Reswick JB, Rogers J. Experience at Ranchos Los Amigos Hospital with devices and techniques to prevent pressure sores. In Kenedi RM, Cowden JM, Scales JT, eds. *Bedsore biomechanics*, pp 301-313. London: Macmillan Press, 1976.

37. Brooks B, Duncan GW. Effects of pressure on tissues. *Archives of Surgery* 1940;**40**:pp 696-709.

38. Husain T. An experimental study of some pressure effects on tissues with reference to the bed-sore problem. *Journal of Pathology Bacteriology* 1953;**66**:pp 347-358.

39. Kosiak M. Etiology of decubitus ulcers. *Archives of Physical Medicine and Rehabilitation* 1961;**42**:pp 19-29.

40. Dinsdale SM. Decubitus ulcers: role of pressure and friction in causation. *Archives of Physical Medicine and Rehabilitation* 1974;**55**:pp 147-152.

41. Claus-Walker J, Campos RJ, Carter RE, Chapman M. Electrolytes in urinary calculi and urine of patients with spinal cord injuries. *Archives of Physical Medicine and Rehabilitation* 1973;**54**:pp 109-114.

42. Krouskop TA. A synthesis of the factors which contribute to pressure sore formation. *Medical Hypotheses* 1983;**11**:pp 255-267.

43. Bridel J. Pressure sores and intra-operative risk. *Nursing Standard* 1992;**7**:pp 28-31.

44. Bridel, J. Intra-operative Pressure Sores. 1995. University of Durham. *Unpublished MA Thesis*

45. Stotts NA, Paul SM. Pressure ulcer development in surgical patients. *Decubitus* 1988;**1**:pp 24-30.

46. Bridel J. Assessing trolley and table products. *Nursing Standard* 1993;**7** **(Tissue Viability Supplement)**:p 11.

47. Moore E, Rithalia S, Gonsakorale M. Assessment of the Charnwood operating table and hospital trolley mattresses. *Journal of Tissue Viability* 1992;**2**:pp 71-72.

48. Neander K-D, Birkenfeld R. Decubitus prophylaxis in the operating theatre? *Journal of Tissue Viability* 1991;**1**:pp 71-74.

49. Bridel J. Pressure sore risk in operating theatre. *Nursing Standard* 1993;**7 (Tissue Viability Supplement)**:pp 4-10.

50. Bridel J. Assessing the risk of pressure sores. *Nursing Standard* 1993;**7**:pp 32-35.

51. Edwards M. Pressure sore risk calculators: some methodological issues. *Journal of Clinical Nursing* 1996;**5**:pp 307-312.

52. Bridel J, Banks S, Mitton C. The admission prevalence and hospital-acquired incidence of pressure sores within a large teaching hospital during April 1994 to March 1995. In Cherry GW, et al, eds. *Proceedings of the 5th European Conference on Advances in Wound Management*, pp 83-85. London: Macmillan Magazines, 1996.

53. Brandeis GH, Ooi WL, Hossain M, Morris JN, Lipsitz LA. A longitudinal study of risk factors associated with the formation of pressure ulcers in nursing homes. *Journal of the American Geriatrics Society* 1994;**42**:pp 388-393.

54. Zulkowski K. MDS+ RAP items associated with pressure ulcer prevalence in newly institutionalised elderly. *Ostomy and Wound Management* 1998;**44**:pp 40-53.

55. Allman RM, Laprade CA, Noel LB, Walker JM, Dear MR, Smith CR *et al.* Pressure sores among hospitalized patients. *Annals of Internal Medicine* 1986;**105**:pp 337-342.

56. Bergstrom N, Demuth PJ, Braden BJ. A clinical trial of the Braden scale for predicting pressure sore risk. *Nursing Clinics of North America* 1987;**22**:pp 417-428.

57. Richardson RR, Meyer PR. Prevalence and incidence of pressure sores in acute spinal cord injuries. *Paraplegia* 1981;**19**:pp 235-247.

58. Clark M, Cullum N. Matching patient need for pressure sore prevention with the supply of pressure redistributing mattresses. *Journal of Advanced Nursing* 1992;**17**:pp 310-316.

59. Bale S, Finaly I, Harding KG. Pressure sore prevention in a hospice. *Journal of Wound Care* 1995;**4**:pp 465-468.

60. Berlowitz DR, Wilking SVB. Risk factors for pressure sores: a comparison of cross-sectional and cohort-derived data. *Journal of the American Geriatrics Society* 1989;**37**:pp 1043-1050.

61. Nixon J, McGough A. Principles of Patient Assessment: Screening for Pressure Ulcers and Potential Risk. In Morison M, ed. *The Prevention and Treatment of Pressure Ulcers*, pp 55-74. Edinburgh: Mosby, 2001.

62. Crow RA, Chase J, Lamond D. The cognitive component of nursing assessment: an analysis. *Journal of Advanced Nursing* 1995;**22**:pp 206-212.

63. Benner P. From novice to expert: excellence and power in clinical nursing. Menlo Park, California: Addison-Wesley, 1984.

64. Pyles SH, Stern PN. Discovery of nursing gestalt in critical care nursing: the importance of the gray gorilla syndrome. *Image: The Journal of Nursing Scholarship* 1983;**15** :pp 51-57.

65. Prescott PA, Soeken KL, Ryan JW. Measuring patient intensity: a reliability study. *Evaluation and the Health Professions* 1989;**12**:pp 128-134.

66. Benner P, Wrubal J. Skilled clinical knowledge: the value of perceptual awareness. *Nurse Educator* 1982;**7**:pp 11-17.

67. MacLeod M. 'It's the little things that count': the hidden complexity of everyday clinical nursing practice. *Journal of Clinical Nursing* 1994;**3**:pp 361-368.

68. Jenny J, Logan J. Knowing the patient: one aspect of clinical knowledge. *Image: The Journal of Nursing Scholarship* 1992;**24**:pp 254-258.

69. Jocavone J, Dostal M. A descriptive study of nursing judgement in the assessment and management of cardiac pain. *Advances in Nursing Science* 1992;**15**:pp 54-63.

70. Corcoran-Perry S, Graves J. Supplemental information seekin behaviour of cardio-vascular nurses. *Research in Nursing and Health* 1990;**13**:pp 119-127.

71. Tanner CA, Benner P, Chelsa C, Gordon DR. The phenomenonolgy of knowing the patient. *Image: The Journal of Nursing Scholarship* 2000;**25**:pp 273-280.

72. Yates JF. Judgements in decision making. London: Prentice-Hall, 1990.

73. Deeks JJ. Pressure sore prevention: using and evaluating risk assessment tools. *British Journal of Nursing* 1996;**5**:pp 313-320.

74. Bridel J. Risk assessment. *Journal of Tissue Viability* 1994;**4**:pp 84-85.

75. Simon R, Altman DG. Statistical aspects of prognostic factor studies in oncology. *British Journal of Cancer* 1994;**69**:pp 979-985.

76. Salzberg CA, Byrne DW, Cayton CG, et al. A new pressure ulcer risk assessment scale for individuals with spinal cord injury. *American Journal of Physical Medicine and Rehabilitation* 1996;**75**:pp 96-104.

77. Halfens RJ. The reliability and validity of the Braden scale. Harding KG, Leaper DL, and Turner TD. Proceedings of the 7th European Conference on Advances in Wound Management. 1997. London, Macmillan.

78. Barnes D, Payton RG. Clinical application of the Braden scale in the acute-care setting. *Dermatology Nursing* 1993;**5**:pp 386-388.

79. Capobianco ML, McDonald DD. Factors affecting the predictive validity of the Braden Scale. *Advances in Wound Care* 1996;**9**:pp 32-36.

80. Langemo DK, Olson B, Hunter S, Hanson D, Burd C, Cathcart-Silberberg T. Incidence and prediction of pressure ulcers in five patient care settings. *Decubitus* 1991;**4**:pp 25-36.

81. Ramundo JM. Reliability and validity of the Braden scale in the home care setting. *Journal of Wound Ostomy and Continence Nurses* 1995;**22**:pp 128-134.

82. Bergstrom N, Braden BJ, Laguzza A, Holman V. The Braden scale for predicting pressure sore risk. *Nursing Research* 1987;**36**:pp 205-210.

83. Watts S, and Clark M. Pressure sore prevention: a review of policy documents. 1993. University of Surrey, Nursing Practice Research Unit. Ref Type: Report

84. Clarke M, Kadhom HM. The nursing prevention of pressure sores in hospital and community patients. *Journal of Advanced Nursing* 1988;**13**:pp 365-373.

85. Guralnik JM, Harris TB, White LR, Cornoni-Huntley JC. Occurrence and predictors of pressure sores in the National Health and nutrition examination survey follow-up. *Journal of the American Geriatrics Society* 1988;**36**:pp 807-812.

86. Marchette L, Arnell I, Redick E. Skin ulcers of elderly surgical patients in critical care units. *Dimensions of Critical Care Nursing* 1991;**10**:pp 321-329.

87. Hoshowsky VM, Schramm CA. Intraoperative pressure prevention: an analysis of bedding materials. *Research in Nursing and Health* 1994;**17**:pp 333-339.

88. Allman RM, Goode PS, Patrick MM, Burst N, Bartolucci AA. Pressure ulcer risk factors among hospitalized patients with activity limitation. *Journal of the American Medical Association* 1995;**273**:pp 865-870.

89. Bergstrom N, Braden B, Kemp M, Champagne M, Ruby E. Multi-site study of incidence of pressure ulcers and the relationship between risk level, demographic characteristics, diagnosis and prescription of preventive interventions. *Journal of American Geriatrics Society* 1996;**44**:pp 22-30.

90. Schnelle JF, Adamson GM, Cruise PA, Al-Samarrai N, Sarbaugh FC, Uman G *et al.* Skin disorders and moisture in incontinent nursing home residents: intervention implications. *Journal of the American Geriatrics Society* 1997;**45**:pp 1182-1188.

91. Allman RM. Pressure ulcer prevalence, incidence, risk factors and impact. *Clinics in Geriatric Medicine* 1997;**13**:pp 421-436.

92. Whitehead J. The design and analysis of sequential clinical trials. Chichester: Ellis Horwood, 1992.

93. Healey F. The reliability and utility of pressure sore grading scales. *Journal of Tissue Viability* 1995;**5**:pp 111-114.

94. Fielding LP, Fenoglio-Preiser CM, Freedman LL. The future of prognostic factors in outcome prediction for patients with cancer. *Cancer* 1992;**70**:pp 2367-2377.

95. Papantonio CT, Wallop JM, Kolodner KB. Sacral ulcers following cardiac surgery: incidence and risks. *Advances in Wound Care* 1994;**7**:pp 24-36.

96. Scott EM. Hospital-acquired pressure sores in surgical patients. *Journal of Wound Care* 1998;**7**:pp 76-79.

97. Hitch S. NHS Executive Nursing Directorate: strategy for major clinical guidelines, prevention and management of pressure sores. A literature review. *Journal of Tissue Viability* 1995;**5**:pp 3-24.

98. Lowthian P. Pressure sores: a search for definition. *Nursing Standard* 1994;**9**:pp 30-32.

99. Holstein P, Neilson PE, Barras JR. Blood flow cessation at external pressure in the skin of normal limbs. *Microvascular Research* 1979;**17**:pp 71-79.

100. Larsen B, Holstein P, Lassen NA. On the pathogenesis of bedsores: skin blood flow cessation by external pressure on the back. *Scandinavian Journal of Plastic and Reconstructive Surgery* 1979;**13**:pp 347-350.

101. Holloway GA, Tolentino G, De Lateur BJ. Cutaneous blood flow responses to wheelchair cushion pressure loading measured by Laser Doppler flowmetry. In Lee BY, ed. *Chronic ulcers of the skin*, pp 57-68. New York: McGraw Hill, 1985.

102. Bennett L, Kavner D, Lee BY, Trainor FS. Skin blood flow in seated geriatric patients. *Archives of Physical Medicine and Rehabilitation* 1981;**62**:pp 392-398.

103. Bader DL. The recovery characteristics of soft tissue following repeated loading. *Journal of Rehabilitation Research and Development* 1990;**27**:pp 141-150.

104. Frantz R, Xakellis GC, Arteaga M. The effects of prolonged pressure on skin blood flow in elderly patients at risk for pressure ulcers. *Decubitus* 1993;**6**:pp 16-20.

105. Xakellis GC, Frantz RA, Arteaga M, Meletiou S. A comparison of changes in the transcutaneous oxygen tension and capillary blood flow in the skin with increasing compressive weights. *American Journal of Physical Medicine and Rehabilitation* 1991;**70**:pp 172-177.

106. Norton D, McLaren R, Exton-Smith AN. An investigation of geriatric nursing problems in hospital. Edinburgh: Churchill Livingstone, 1962.

107. Catchpole BN, Jepson RP. Hand and finger blood flow. *Clinical Science* 1955;**12**:pp 33-40.

108. Romanus EM. Microcirculatory reactions to controlled tissue ischaemia and temperature: a vital microscopic study on the Hamster's Cheek Pouch. In Kenedi RM, Cowden JM, Scales JT, eds. *Bedsore biomechanics*, pp 79-82. London: Macmillan Press, 1976.

109. Felder D, Russ E, Montgomery H, Horwitz O. Relationship in the toe of skin surface temperature to mean blood flow measured with a plethysmograph. *Clinical Scientist* 1954;**13**:pp 251-257.

110. Holmes R, Macchiano K, Jhangiani SS, Agarwal NR, Savino JA. Combating pressure sores: nutritionally. *American Journal of Nursing* 1987;**87**:pp 1301-1303.

111. Cullum N, Clark M. Intrinsic factors associated with pressure sores in elderly people. *Journal of Advanced Nursing* 1992;**17**:pp 427-431.

112. Parkinson H, Coleman S, Nixon J, and Scott J. Peri-operative pressure area care - implementation of a research based assessment and management flow chart. 1999. 35th Annual Congress and Exhibition National Association of Theatre Nurses. 12-10-1999.

113. Woolf N. Pathology basic and systemic. London: WB Saunders Co. Ltd, 1998.

114. Scales JT. Pathogenesis of pressure sores. In Bader DL, ed. *Pressure sores clinical practice and scientific approach*, pp 15-26. London: Macmillan Press, 1990.

115. Fagrell B. Advances in microcirculation network evaluation: an update. *International Journal of Microcirculation: Clinical and Experimental* 1995;**15 (S1)**:pp 34-40.

116. Hall DA. The Biomedical Basis of Gerontology. G.B.: John Wright & Sons Ltd, 1984.

117. Lamb JF, Ingram CG, Johnston IA, Pitman RM. Essentials of physiology. G.B.: Blackwell Scientific Publications, 1980.

118. Michel CC, Gillott H. Microvascular mechanisms in stasis and ischaemia. In Bader DL, ed. *Pressure sores clinical practice and scientific approach*, pp 153-164. London: Macmillan Press, 1990.

119. Henriksen O. Local sympathetic reflex mechanism in the regulation of blood flow in the human subcutaneous adipose tissue. *Acta Physiologica Scandinavica* 1977;**450 (Suppl)**:pp 7-48.

120. Schubert V, Fagrell B. Local skin pressure and its effects on skin microcirculation as evaluated by Laser-Doppler fluxmetry. *Clinical Physiology* 1989;**9**:pp 535-545.

121. Ek A-C, Lewis DL, Zetterqvist H, Svensson P-G. Skin blood flow in an area at risk for pressure sore. *Scandinavian Journal of Rehabilitation Medicine* 1984;**16**:pp 85-89.

122. Barbenel JC, Cui ZF. Abnormal thermal hyperaemia in the skin in paraplegia. *Physiological Measurement* 1993;**14**:pp 231-239.

123. Ganong WF. Review of Medical Physiology. Stamford, Connecticut: Appleton and Lange, 1999.

124. Soter NA. Acute effects of ultraviolet radiation on the skin. *Seminars in Dermatology* 1990;**9**:pp 11-15.

125. Russell NS, Knaken H, Bruinvis IAD, Hart AAM, Begg AC, Lebesque JV. Quantification of patient to patient variation of skin erythema developing as a response to radiotherapy. *Radiotherapy and Oncology* 1994;**30**:pp 213-221.

126. Tooke JE, Brash PD. Microvascular aspects of diabetic foot disease. *Diabetic Medicine* 1996;**13**:p S26-S29.

127. Cheatle TR, Stibe ECL, Shami SK, Scurr JH, Coleridge-Smith PD. Vasodilatory capacity of the skin in venous disease and its relationship to transcutaneous oxygen tension. *British Journal of Surgery* 1991;**78**:pp 607-610.

128. Schubert V, Fagrell B. Postocclusive reactive hyperaemia and thermal response in the skin microcirculation of subjects with spinal cord injury. *Scandinavian Journal of Rehabilitation Medicine* 1991;**23**:pp 33-40.

129. Landis EM. Micro-injection studies of capillary blood pressure in human skin. *Heart* 1930;**15**:pp 209-228.

130. Daly CH, Chimoskey JE, Holloway GA, Kennedy D. The effect of pressure loading on the blood flow rate in human skin. In Kenedi RM, Cowden JM, Scales JT, eds. *Bedsore biomechanics*, pp 69-78. London: Macmillan Press, 1976.

131. Bader DL, Gant CA. Changes in transcutaneous oxygen tension as a result of prolonged pressures at the sacrum. *Clinical Physics Physiology Measurements* 1988;**9**:pp 33-40.

132. Lewis T, Grant R. Observations upon reactive hyperaemia in man. *Heart* 1925;**12**:pp 73-120.

133. Khan F, Carnochan FMT, Abbot NC, Wilson SB. The effect of oxygen supplemention of post-occlusive reactive hyperaemia in human forearm skin. *International Journal of Microcirculation: Clinical and Experimental* 1991;**10**:pp 43-53.

134. Hagisawa S, Ferguson-Pell M, Cardi M, Miller D. Assessment of skin blood content and oxygenation in spinal cord injured subjects during reactive hyperaemia. *Journal of Rehabilitation and Development* 1994;**31**:pp 1-14.

135. Mahanty SD, Roemer RB. Thermal response of skin to application of localized pressure. *Archives of Physical Medicine and Rehabilitation* 1979;**60**:pp 584-590.

136. Walmsley D, Wiles PG. Reactive hyperaemia in the skin of the human foot measured by laser Doppler flowmetry: effects of duration of ischaemia and local heating. *International Journal of Microcirculation: Clinical and Experimental* 1990;**9**:pp 345-355.

137. Ninet J, Fronek A. Cutaneous postocclusive reactive hyperemia monitored by Laser Doppler flux metering and skin temperature. *Microvascular Research* 1985;**30**:pp 125-132.

138. Mayrovitz HN, Smith J, Delgado M, Regan MB. Heel blood perfusion responses to pressure loading and unloading in women. *Ostomy and Wound Management* 1997;**43**:pp 16-26.

139. Johnson JM, O'Leary DS, Taylor WF, Kosiba W. Effect of local warming on forearm reactive hyperaemia. *Clinical Physiology* 1986;**6**:pp 337-346.

140. Hagisawa S, Barbenel JC, Kenedi RM. Influence of age on postischaemic reactive hyperaemia. *Clinical Physics Physiology Measurements* 1991;**12**:pp 227-237.

141. Schubert V, Fagrell B. Evaluation of the dynamic cutaneous post-ischemic hyperaemia and thermal response in elderly subjects and in an area at risk for pressure sores. *Clinical Physiology* 1991;**11**:pp 169-182.

142. Kvernebo K, Slagsvold CE, Gjolberg T. Laser Doppler Flux Reappearance Time (FRT) in patients with lower limb atherosclerosis and healthy controls. *European Journal of Vascular and Endovascular Surgery* 1988;**2**:pp 171-176.

143. Slagsvold C-E, Stranden E, Rosen L, Kroese AJ. The role of blood perfusion and tissue oxygenation in the postischemic transcutaneous $PO_2$ response. *Angiology: The Journal of Vascular Diseases* 1992;**43**:pp 155-162.

144. Slagsvold C-E, Rosen L, Stranden E. The relation between changes in capillary morphology induced by ischemia and the postischemic transcutaneous $PO_2$ response. *International Journal of Microcirculation: Clinical and Experimental* 1991;**10**:pp 117-125.

145. Valentini G, Leonardo G, Moles DA, Apaia MR, Maselli R, Tirri G *et al.* Transcutaneous oxygen pressure in systemic sclerosis: evaluation at different sensor temperatures and relationship to skin perfusion. *Archives of Dermatological Research* 1991;**283**:pp 285-288.

146. Wilkinson SP, Spence VA, Stewart WK. Arterial stiffening and reduced cutaneous hyperaemic response in patients with end-stage renal failure. *Nephron* 1989;**52**:pp 149-153.

147. Mahanty SD, Roemer RB, Meisel H. Thermal response of paraplegic skin to the application of localized pressure. *Archives of Physical Medicine and Rehabilitation* 1981;**62**:pp 608-611.

148. Bidart Y, Maury M. The circulatory behaviour in complete chronic paraplegia. *Paraplegia* 1973;**11**:pp 1-24.

149. Witkowski JA, Parish LC. Histopathology of the decubitus ulcer. *Journal of the American Academy of Dermatology* 1982;**6**:pp 1014-1021.

150. Daniel RK, Priest DL, Wheatley DC. Etiologic factors in pressure sores: an experimental model. *Archives of Physical Medicine and Rehabilitation* 1981;**62**:pp 492-498.

151. Romanus M. Microcirculatory reactions of local pressure induced ischaemia: a vital microscopic study in Hamster Cheek Pouch and a pilot study in man. *Acta Chirurgica Scandinavica* 1977;**479 (Suppl)**:pp 1-30.

152. Brånemark P-I. Microvascular function at reduced flow rates. In Kenedi RM, Cowden JM, Scales JT, eds. *Bedsore Biomechanics*, pp 63-68. London: Macmillan Press, 1976.

153. Romanus M, Svensjo E, Ehira T. Microvascular changes due to repeated pressure-induced ischemia: intravital microscopic study on Hamster Cheek Pouch. *Archives of Physical Medicine and Rehabilitation* 1983;**64**:pp 553-555.

154. Ames III A, Wright L, Kowada M, Thurston JM, Majno G. Cerebral ischaemia: II the no-reflow phenomenon. *American Journal of Pathology* 1968;**52**:pp 437-453.

155. Lefer AM, Lefer DJ. The role of nitric oxide and cell adhesion molecules on the microcirculation in ischaemia-reperfusion. *Cardiovascular Research* 1996;**32**:pp 743-751.

156. Homer-Vanniasinkam S, Crinnion JN, Gogh MJ. Post-ischaemic organ dysfunction: a review. *European Journal of Vascular and Endovascular Surgery* 1997;**14**:pp 195-203.

157. Kerrigan CL, Stotland MA. Ischaemia reperfusion injury: a review. *Microsurgery* 1993;**14**:pp 165-175.

158. Nola GT, Vistnes LM. Differential response of skin and muscle in the experimental production of pressure sores. *Plastic and Reconstructive Surgery* 1980;**66**:pp 728-735.

159. Guttmann L. The prevention and treatment of pressure sores. In Kenedi RM, Cowden JM, Scales JT, eds. *Bedsore biomechanics*, pp 153-159. London: Macmillan Press, 1976.

160. Franzeck UK, Rayman GA. Assessment of microvascular function and tissue viability. In Tooke JE, Lowe GDO, eds. *A Textbook of Vascular Medicine*, pp 111-140. London: Arnold, 1996.

161. Coleridge-Smith PD. Methods of vascular investigation. In Tooke JE, Lowe GDO, eds. *A Textbook of Vascular Medicine* , pp 100-110. London: Arnold, 1996.

162. Fronek A. Clinical value of Laser Doppler fluxmetering. In Bernstein EF, ed. *Vascular diagnosis*, pp 626-629. USA: Mosby, 1993.

163. Fronek A. Clinical experience with transcutaneous $PO_2$ and $POC_2$ measurements. In Bernstein EF, ed. *Vascular diagnosis*, pp 620-625. USA: Mosby, 1993.

164. Altman DG. Practical Statistics for Medical Research. London: Chapman & Hall, 1991.

165. Cullum N, Deeks J, Sheldon TA, Song F, and Fletcher J. Beds, mattresses and cushions for pressure sore prevention and treatment. The Cochrane Library (Issue 2, Update Software). 2000.

166. Dybkaer R, Lauretzen M, Krakauer R. Relative reference values for clinical chemical and haematological quantities in "healthy" elderly people. *Acta Med Scandinavica* 1981;**209**:pp 1-9.

167. Phillips A, Shaper GA, Whincup PH. Association between serum albumin and morality from cardiovascular disease, cancer and other causes. *Lancet* 1989;**ii**:pp 1434-1436.

168. Gebhardt K. Preventing pressure sores in orthopaedics. *Nursing Standard* 1992;**6**:pp 4-6.

169. Bliss M, Simini B. When are the seeds of postoperative pressure sores sown? *British Medical Journal* 1999;**319**:pp 863-864.

170. RCN. Pressure ulcer risk assessment and prevention. 2000. London, RCN.

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# Appendix 2
# Publications

# Pressure sores and intra-operative risk

Jane Bridel RGN, BSc (Hons), Cert Health Ed, is a part-time postgraduate student, Health Care Research Unit, University of Durham.

RCN NURSING *Update*

This article provides complementary reading to RCN Nursing Update Learning Unit 16. A repeat of the television component will be broadcast on BBC2 on Friday morning, October 23, from 3am to 4am. Copies of the RCN Nursing Update supplement are available from *Nursing Standard*. Contact Olive Healey, tel 081-423 1066, or see the special advertisement in the classified section.

*The enormous cost of pressure sore care is well documented (1, 2), but a review of the available literature shows few studies on the genesis of intra-operative pressure sores exist and the contribution of operating room exposure as an aetiological factor is largely undefined. This article provides a brief overview of the aetiology of pressure sores, details interface pressures reported on operating tables, and critically reviews the literature suggesting a link between events during the intra-operative period and post-operative pressure sore formation.*

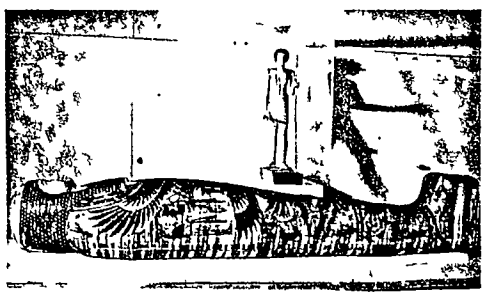
The occurrence of pressure sores has been a challenging phenomenon throughout the centuries. Evidence from the mummified body of an Egyptian priestess suggests the problem dates back at least to the time of the Pharaohs (3).

It persists today, and studies have revealed hospital prevalence rates ranging from 4 to 10.1 per cent (4-7). Because of the escalating costs of the treatment of pressure sores, they are now seen as preventable sequelae rather than a tolerable complication of illness, and the emphasis is on identifying risk factors and implementing appropriate interventions.

A review of the available literature shows that few studies on the genesis of intra-operative pressure sores exist, and that the contribution of operating room practice as an aetiological factor is largely undefined.

The evidence to date can be divided into two main areas: research reviewing the post-operative complications of surgical patients and articles relating to interface pressure measurements of current theatre equipment. These will be discussed, but a brief overview of the aetiology of pressure sore development will be provided first.

**Aetiology of pressure sores** Pressure sores are defined as areas of necrosis caused by excessive and prolonged pressure (8). They include reactive hyperaemia as well as blistered, broken or necrotic skin (9).



Evidence from an Egyptian mummy suggests the problem of pressure sores dates back at least to the time of the Pharaohs.

Damage to the skin from pressure is the result of two concurring processes that commonly coexist. These are the exclusion of blood from the skin by the application of sustained superficial pressure in excess of the mean capillary pressure, and thrombosis of arterioles and the microcirculation caused by the application of disruptive and shearing forces. These damage the endothelial cells locally in dependent areas of skin and subcutaneous tissues (10).

The critical determinants of pressure damage and sore development are the intensity and duration of pressure, and the tolerance of the skin and its supporting structures.

Research has attempted to identify threshold levels in relation to intensity and duration of pressure, but results vary considerably and uncertainties concerning accuracy have precluded universal acceptance of critical threshold values. Criticisms of research in this field relate to the use of animal skin for experiments, the omission of loading shear in calculations (11, 12) and a failure to account for autoregulation processes and differences in tissue tolerance.

The conclusions, however, that prolonged low pressure causes more damage than short-term high pressure (13, 14) and that mean arteriolar pressure in humans is 32 mmHg (15), with evidence provided by nursing research in relation to movement and activity (16, 17), has provided a baseline rationale for the development of pressure sore prevention equipment and policies.

Exploring these controversial issues would require considerable time and space, but the general aims of equipment and practice have been to provide support surfaces that generate interface pressures of less than 32mmHg and/or intermittent relief of pressure on a given area of skin after a period not exceeding two hours (18).

The factors affecting tissue tolerance can be sub-divided into extrinsic and intrinsic factors. The extrinsic factors include increased moisture and the presence of frictional forces. The intrinsic factors include nutritional status, decreased arteriolar pressure and increased age. Also

**The contribution of operating room practice as a factor in the formation of pressure sores is largely undefined.**

References
1. Livesey B. An expensive epidemic. *Nursing Times* 1987. 83, 7, 79.
2. Hibbs P. Prevention of pressure sores. Unpublished paper. 1987.
3. Thompson-Rowling J. Pathological changes in mummies. *Proceedings of the Royal Society of Medicine.* 1961. 54, 409.
4. Barbenel J C *et al.* Incidence of pressure sores in the Greater Glasgow Health Board Area. *The Lancet.* 1977. 2, 548-550.
5. David J A *et al.* An investigation of the current methods used in nursing for the care of patients with established pressure sores. London, Northwick Park Nursing Research Unit. 1983.
6. Ek A C, Bowman G. A descriptive study of pressure sores: the prevalence of pressure sores and the characteristics of patients. *Journal of Advanced Nursing.* 1982. 7, 1, 51-57.
7. Girvin J, Griffiths-Jones A. Towards prevention. *Nursing Times.* 1989. 85, 12, 64-66.
8. Beland J, Passos J. *Clinical Nursing: Pathophysiological and Psychosocial Approaches.* Fourth edition. London, Collier MacMillan.1981.
9. Parish L C *et al. The Decubitus Ulcer.* New York NY, Masson. 1983.
10. Barton A, Barton M. *The Management and Prevention of Pressure Sores.* London, Faber. 1981.
11. Clark M. Preventing pressure sores: matching patient and mattress using interface pressure measurements. *Decubitus.* 1989. 2, 1, 34-39.
12. Bennett L, Lee B Y. Vertical shear existence in animal pressure threshold experiments. *Decubitus.* 1988. 1, 1, 18-24.
13. Kosiak M. Etiology and pathology of ischaemic ulcers. *Arch Phys Med Rehabil.* 1959. 40, 62-69.
14. Scales J T. Air support systems for the prevention of bedsores. In Kenedi R M *et al* (Eds). *Bedsore Biomechanics.* Maryland MD, UniPark Press. 1976.
15. Landis E N. Micro-injection studies of capillary blood pressure in human skin. *Heart.* 1930. 15, 209-228.
16. Exton-Smith A N, Sherwin R. The prevention of pressure sores. Significance of spontaneous bodily movements. *The Lancet.* 1961. 2, 7212, 1124-1126.

involved are interstitial fluid flow, emotional stress, smoking and skin temperature (17, 19, 20). Again, a full review of these factors is not possible, but the multifactoral nature of tissue tolerance has had great bearing on the interpretation of research and complicates the process of defining those patients at risk (21, 22).

Having highlighted the multi-factoral nature of pressure sore development, interface pressures recorded on operating tables will now be detailed to explain why patients may be at risk during surgery.

**Interface pressures** A number of studies that have measured the interface pressures on standard hospital tables show that patients are exposed to external pressures far in excess of mean capillary pressures. Studies using a Gaymer mercury manometer reported sacral readings all of which were above 56mmHg (23), and mean sacral readings of 46 plus or minus 16mmHg (24) in conscious subjects.

Neander and Birkenfeld (25) used 20 healthy volunteers and measured the interface pressures on a standard operating table surface using a full-length sensory mat linked to a micro-computer. The results showed that surface pressures of up to 70mmHg frequently occurred over large areas of the body.

Differences in results can be accounted for by the limitations of the manometers used, variations in operating tables and sampled volunteers, for example, as regards age and body weight.

Perhaps of most interest is the increase in interface pressures noted by Campbell (23), who compared pre-anaesthetic induction, post-anaesthetic induction and post-surgical recordings. The post-surgical measures of patients on the operating table for more than 2.5 hours were 35 per cent higher than the pre-anaesthetic induction measures. This suggests that interface measurements using healthy conscious volunteers provide a conservative picture of the pressures to which patients are exposed while undergoing surgery. The importance of these measurements, however, is unknown in terms of their potential to generate skin damage.

The widespread use of 32mmHg as a threshold value is in dispute because it fails to account

for autoregulation mechanisms that enable capillaries to maintain blood flow until external pressures reach diastolic magnitude (26). Other factors involved include the differences in the application of localised and uniform pressure and the influence of shear.

It is too simplistic, then, to state that patients are exposed to external pressures in excess of the mean capillary value of 32mmHg and are therefore at risk of developing pressure sores during surgery. To establish whether there is a causal relationship between patients' exposure to pressure during surgery and the subsequent development of pressure sores requires further review of the literature.

## Post-operative complication

The possible association between pressure damage and events in the operating department was first suggested by Hicks (27), who found a 13 *per cent incidence of pressure sores on 100* patients who had undergone surgery lasting two hours or longer. Hicks found that the incidence of pressure sores in patients whose surgery lasted four hours or more was twice that of patients whose surgery lasted less than four hours.

Problems arise in the application of his work to current working practices because of the lack of information on when the sores developed in relation to the day of surgery, which type of operating table pad was in use during that period and differences in ward nursing practices.

Further evidence began to suggest that skin damage could result from pressure exposure in the operating theatre, as reports of occipital alopecia were documented (24). For example, Lawson *et al* (28) found a 14 per cent incidence of occipital alopecia in patients following cardiopulmonary bypass, which was reduced to 1 per cent by changing the position of the patient's head every 30 minutes.

Despite this, the association between events in the operating department remains of little consequence and severe cases have been reported as surgical burns (29, 30). Gendron (29) noted that severe burn-like injuries were reported but could not be explained either by faulty electrosurgical equipment or a lapse in safe operating room practice. He began to speculate that many unexplained burns were not in fact burns, concluding that the lack of recognition of their true nature guaranteed their continuance.

An overview of the incidents revealed a number of common factors. These included the time of surgery (procedures longer than four hours), the type of surgery (vascular surgery had been performed) and the site of trauma, which was always an area exposed to sustained pressure (for example, the sacrum).

Gendron then detailed two prospective studies involving 89 patients undergoing various surgical procedures lasting between two and eight hours who rested on silicone gel pads, and 184 patients not resting on gel pads whose surgery lasted between half an hour and two hours.

The interpretation and application of the results to clinical practice is not possible, however, because details of the method used are not given, patients' exposure to standard operating conditions is not compared with results from silicone gel pad use, and patients' degree of risk due to other factors, such as mobility, is not analysed.

Further reports have followed, citing case studies (31) and high 'day of surgery' incidence rates (32). Many questions remain unanswered, however, and in an attempt to address the limitations of the few reported studies, Kemp *et al* (33) *conducted a study that* aimed to determine a relationship between a number of factors, including time on the operating table, age, hypotensive episodes during surgery, pre-operative Braden scores and the development of pressure sores.

The study involved 125 patients who were admitted for elective in-patient surgery. Fifteen (12 per cent) developed a total of 23 pressure sores. Although patients who developed pressure sores were older, spent more time on the operating table, experienced a greater proportion of episodes of intra-operative diastolic hypotension and had lower pre-operative Braden scores than patients who did not, none of these was statistically significant.

Seventy per cent of pressure sores were, however, first observed as patients were being transferred from the operating table, and the authors called for further study to enable the development of a multivariate model for use as an accurate predictor of patients at risk during surgery.

A review of the literature relating to surgical patients and the operating department suggests that there is a causal relationship between events during surgery and the subsequent development of pressure sores. With the exception of Kemp *et al* (33), the available studies lack detail in the documentation of research design and the degree of patient risk due to other factors, such as mobility, activity and nutritional status.

To define the contribution of specific factors in the aetiology of pressure sore development in the operating department requires further research, and the development of a multivariate prediction tool is necessary to identify patients at high risk ●

17. Braden B J, Bergstrom N. A conceptual scheme for the study of the etiology of pressure sores. *Rehab Nurs.* 1987. 12, 1, 8-12,16.
18. Exton-Smith A N. The patient's not for turning. *Nursing Times.* 1987. 83, 42, 42-44.
19. Copeland-Fields L D, Hoshiko B R. Clinical validation of Braden and Bergstrom's conceptual schema of pressure sore risk factors. *Rehab Nurs.* 1989. 14, 5, 257-260.
20. Cullum N, Clark M. Intrinsic factors associated with pressure sores in elderly people. *Journal of Advanced Nursing.* 1992. 17, 4, 427-431.
21. Crow R. The challenge of pressure sores. *Nursing Times.* 1988. 84, 38, 68-73.
22. Barratt E. Pressure sores: putting risk calculators in their place. *Nursing Times.* 1987. 83, 7, 65-70.
23. Campbell K. Pressure point measures in the operating room. *Journ Enterostomal Ther.* 1989. 16, 3, 119-124.
24. Stewart T P, Magnano S J. Burns or pressure ulcers in the surgical patient. *Decubitus.* 1988. 1, 1, 36-40.
25. Neander K D, Birkenfeld R. Decubitus prophylaxis in the operating theatre. *Journal of Tissue Viability.* 1991. 1, 3, 71-74.
26. Bennett I, Lee B Y. Pressure versus shear in pressure sore causation. In Lee B Y (Ed). *Chronic Ulcers of the Skin.* New York NY, McGraw Hill. 1985.
27. Hicks D. An incidence study of pressure sores following surgery. In *ANA Clin. Sessions.* Norwalk VA, Appleton Century Crofts. 1971.
28. Lawson N W *et al.* Occipital alopecia following cardiopulmonary bypass. *Journal of Thoracic and Cardiovascular Surgery.* 1976. 71, 342-347.
29. Gendron F. Burns occurring during lengthy surgical procedures. *Journal of Clinical Engineering.* 1980. 51, 1, 19-26.
30. Dobbie A K. Section D discussion. In Kenedi R M *et al* (Eds). *Bedsore Biomechanics.* Baltimore MD, UniPark Press. 1976.
31. Vermillion C. Operating room acquired pressure ulcers. *Decubitus.* 1990. 3, 1, 26-30.
32. Versluysen M. How elderly patients with femoral fracture develop pressure ulcers in hospital. *British Medical Journal.* 1986. 292, 1311-1313.
33. Kemp M G *et al.* Factors which contribute to pressure sores in surgical patients. *Res Nurs Health.* 1990. 13, 5, 293-301.

# Assessing the risk of pressure sores

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*This article provides an overview of the basic requirements of a good assessment tool and details the development of three pressure sore risk assessment scales – the Norton score (1), the Waterlow score (2) and the Braden scale (3). The tools described are critically reviewed in relation to their reliability and validity, and conclusions made regarding their usefulness in clinical practice and research.*

There are two basic requirements of a good assessment tool – validity and reliability.

**Validity** This can be evaluated by calculating the sensitivity, specificity and predictive values of positive and negative tests:

- Sensitivity is the accuracy in predicting those who develop the condition (4)
- *Specificity is the accuracy in* predicting those who do not develop the condition eg the percentage of those who do not develop pressure sores, as predicted by the scale (5)
- The predictive value of positive tests is the percentage of those at risk of pressure sore development who actually develop a pressure sore
- The predictive value of negative tests is the percentage of those not at risk of pressure sore development who do not develop a pressure sore.

These aspects of validity are important when an assessment tool is applied to the practical situation since over- and/or under-prediction of cases has implications for wrongly assessed patients and allocation of resources.

**Reliability** of an assessment tool can be calculated in a variety of ways but is usually expressed as per cent agreement or correlation:

- Percentage agreement is calculated by determining the number of rater agreements and dividing this by the number of rater agreements plus disagreements.
- Correlation measures of reliability can be used to quantify the magnitude and direction of a relation, and scores range from -1.00 to +1.00. The closer to 1.00, the better the reliability of the tool.

The validity and reliability of assessment tools are important in the comparison of control and treatment groups so that degree of risk can be matched and patient outcomes attributed to the intervention. Many of the assessment tools currently in use, however, have not been tested prior to their implementation.

**Norton score** Norton *et al* (1) designed the first risk assessment tool 30 years ago as a way of simply evaluating patients' physical and mental condition with their liability of developing pressure sores.

The tool assessed five areas: physical condition, level of consciousness, activity, mobility, and incontinence. Each area was scored on a scale of 1 to 4, with overall scores ranging from a maximum of 20 for the patient who is in good condition in all areas to a minimum of 5 for a patient who is in very poor condition.

In their survey of 250 patients, they found an almost linear relationship between the initial score (the assessment score on admission to hospital) and the incidence of pressure sores – defined as a break in the skin surface.

The authors recommended that the scoring system be used as the basis for assessment of pressure sore risk and to determine the frequency of nursing attention for patients.

Retrospective calculation of validity measures using the original data reveals the validity is poor: sensitivity 63 per cent, specificity 70 per cent, positive test 39 per cent and negative test 86 per cent. Reliability was considered in the study design which stated that closely similar scores could be obtained by different observers. To eliminate possible error, however, recordings were made by the same observer on every patient at weekly intervals. No data were published allowing conclusions to be drawn on reliability.

**Table 1. Validity calculations- Norton score.**

| Study | Sample Size | Sens % | Spec % | Pred value of positive test % | Pred value of negative test % |
|---|---|---|---|---|---|
| Norton et al 1962 | 250 | 63 | 70 | 39 | 86 |
| Robert and Goldstone 1979 | 64 | 93 | 43 | 37 | 96 |
| Newman and West 1981 | 88 | 83 | 63 | 14 | 98 |
| Goldstone and Goldstone 1982 | 40 | 89 | 36 | 53 | 80 |
| Gaston 1984 | 262 | 73 | 69 | 49 | 87 |
| Lincoln et al 1986 | 36 | 0 | 94 | 0 | 85 |

**Table 2. Validity calculations- adapted versions of the Norton score.**

| Study | Sample Size | Sens % | Spec % | Pred value of positive test % | Pred value of negative test % |
|---|---|---|---|---|---|
| Gosnell 1973 | 30 | 100 | 73 | 36 | 100 |
| Goldstone and Roberts 1980 | 60 | 100 | 49 | 51 | 100 |
| Warner and Hall 1986 | 334 | 65 | 89 | 15 | 96 |
| Stotts 1988 | 387 | 16 | 94 | 38 | 84 |
| Towey and Erland 1988 | 60 | 17 | 44 | 18 | 37 |

Despite the occurrence of over- and under-prediction of patients, the tool was recommended for use in pressure sore prevention (6, 7) and was reported in the late Eighties to be the most predominant tool used on wards (8, 9, 10).

During the same period, a plethora of articles on the tool was published - some attempting to validate its predictive ability (11-15) and others detailing modifications to the scale and claiming improved predictive ability (5, 16-18).

Validity calculations can also be done with these studies (Table 1). Caution is necessary in comparing the results as definitions of the term pressure sore, methods of data collection and time when the scores are recorded in relation to sore development, differ between the studies.

Sensitivity and the predictive value of the negative test illustrate under-prediction. Results range from 0-93 per cent and 80-98 per cent respectively and indicate under-prediction of 7-100 per cent of those developing sores and incorrect allocation of 2-20 per cent of patients to the 'not at risk' category.

Conversely, the specificity and predictive value of the positive test results, which range from 36-94 per cent and 0-53 per cent respectively, indicate the over prediction of 6-64 per cent of those not developing sores and the incorrect allocation of 47-100 per cent of patients to the 'at risk' category.

Reliability testing of the score is infrequently mentioned in the literature. Of the studies in Table 1, two failed to make any reference to the reliability of the scale (13, 15), one reported that the ward nurses were trained in the use of the scale but did not detail reliability (14), and another that the interrater reliability of the investigators 'was tested and verified', though specific methods and results were not reported (11).

Only Lincoln *et al* (12) explored the interrater reliability of the tool in a systematic manner. On four occasions, each of the 73 subjects was independently assessed by two investigators and the resulting paired scores were analysed for absolute, 1-point and risk versus non-risk per cent agreement. The results ranged from 10-70 per cent, 58-80 per cent, and 60-100 per cent respectively.

To improve agreement, the investigators discussed the ratings after each data collection session and attempted to reach consensus on the meaning of the individual items of the Norton score. This appeared to improve interrater agreement up to and including the third assessment. During the fourth, however, the overall agreement was lower than it had been on the first assessment.

Difficulties arose in the interpretations of ratings such as 'fair' versus 'poor' and 'limited' versus 'slightly limited', scoring of the subsection 'physical condition', and differences of opinion between medical and surgical nurses. Even after developing standardised definitions, the investigators continued to have difficulty agreeing (12).

The poor validity and reliability of the tool

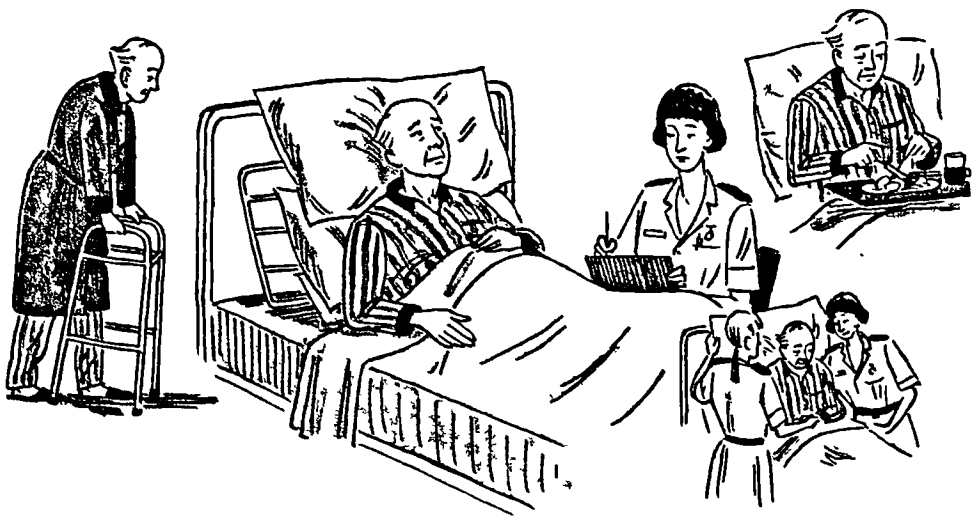

**Table 3. Validity calculations- Waterlow (W) and Norton (N).**

| Study | Sample Size | Sens % | | Spec % | | Pred value of positive test % | | Pred value of negative test % | |
|---|---|---|---|---|---|---|---|---|---|
| | | W | N | W | N | W | N | W | N |
| Dealey 1989 | 175 | 98 | 88 | 14 | 26 | 36 | 37 | 94 | 81 |
| Wardman 1991 | 32 | 100 | 80 | 14 | 82 | 34 | 66 | 100 | 69 |

References
1 Norton D *et al. An Investigation of Geriatric Nursing Problems in Hospital.* London, The National Corporation for the Care of Old People. 1962.
2. Waterlow J. A risk assessment *card. Nursing Times. 1985. 81, 48, 24-27.*
3 Braden B J, Bergstrom N. A conceptual schema for the study of the etiology of pressure sores. *Rehabilitation Nursing.* 1987. 12, 1, 8-12, 16.
4. Warner V, Hall D J. Pressure sores: a policy for prevention. *Nursing Times.* 1986. 82, 16, 59-61.
5. Lilienfeld A M, Lilienfeld D R. *Foundations of Epidemiology.* Second edition. New York NY, Oxford University Press. 1980.
6. Hibbs P. Pressure sores: a system of prevention. *Nursing Mirror.* 1982. 155, 5, 25-29.
7. Torrance C. *Pressure Sores: Aetiology, Treatment, and Prevention.* London, Croom Helm. 1983.
8. Anthony D. Are you in the dark? *Nursing Times.* 1987. 83, 34, 27-30.
9. Girvin J, Griffiths-Jones A. Towards prevention. *Nursing Times.* 1989. 85, 12, 64-66.
10. Spenceley P. Norton v Waterlow. *Nursing Times.* 1988. 84, 32, 52-53.
11. Gaston S F. Evaluation of the Norton scale of risk for the development of pressure ulcers. Unpublished study. Sacramento, California. Cited by Taylor K J. Assessment tools for the identification of patients at risk for the development of pressure sores: a review. *Journal of Enterostomal Therapy.* 1988. 15, 5, 201-205.
12. Lincoln R *et al.* Use of the Norton pressure sore risk assessment scoring system with elderly patients in an acute area. *Journal of Esterostomal Therapy.* 1986. 13, 4, 132-138.
13. Goldstone L A, Goldstone J. The Norton Score: an early warning of pressure sores? *Journal of Advanced Nursing.* 1982. 7, 5, 419-426.
14. Roberts B V, Goldstone L A. A survey of pressure sores in the over sixties on two orthopaedic wards. *International Journal of Nursing Studies.* 1979. 16, 355-364.

and its inability to predict accurately which patients are or are not at risk does not support the use of this tool as a single indicator for pressure sore prevention or for research.

A number of authors have attempted to adapt the Norton score to reduce the errors (5, 16-21), but similar problems have arisen with the calculation of validity and reliability.

Some are merely anecdotal descriptions of the development of a 'new and better' scoring system such as the Douglas score and Smedley score (18, 19) with some comparison made with the Norton score by way of evaluation. Other studies which have reduced, increased and/or exchanged subsections within the score and reported predictive ability are summarised in Table 2 (calculations being made using data from the text). Sample sizes vary enormously as do predictive abilities. Those that have accurately predicted patients who developed sores appear to have done so by over-prediction (16, 17). Similarly, those that have good prediction of patients not at risk have done so by gross under-prediction of those at risk and who developed pressure sores (5, 20). Another shows poor predictive ability on all aspects (21).

Two studies made no reference to reliability (5,16) and two studies developed mutually exclusive operational definitions for each item within the score but did not document further details of reliability testing (17, 20). Towey and Erland (21) detailed calculations of the internal consistency of each subsection and described this as a reliability analysis. Reliability in terms of agreement between data collectors was not recorded and no reference was made to anyone being responsible for patient assessment.

A brief review of adapted assessment tools reveals inadequate testing of reliability, and errors in the prediction of which patients will or will not develop pressure sores. The tools' practical and research-based uses are thus not supported by the literature.

In 1985, an alternative tool, the Waterlow score, was launched following the results of a 650 patient survey (2).

**Waterlow score** The Waterlow card was designed as a practical 'aide-memoire' of the preventive aids and treatments available which also promoted awareness of the causes of pressure sores and determined risk (2).

The scoring system incorporates six main areas of risk; build/weight, continence, skin type, mobility, sex/age, and appetite, with a special risk section alerting the user to tissue malnutrition, neurological deficit, surgery/trauma, and specific medication. The normal risk section scores can range from 1 to 32, and the special risk from 0 to 22, resulting in a maximum score of 64. From the survey data, different degrees of risk according to scores were obtained: 10+ = at risk, 15+ = high risk, and 20+ = very high risk.

No data were published, however, which allowed full exploration of the validity or reliability of the tool. Details of the number of patients at risk were not recorded, nor was the incidence of pressure sores.

In a later publication referring to the same survey (22), Waterlow reported a 17.1 per cent (period) prevalence. Analysis of scores indicated that large numbers of patients considered 'at risk' did not develop pressure sores. The validity is further questioned by possible researcher bias as she stated that she 'observed more than 90 per cent of all patients herself'.

Problems also arise with the reliability. The tool was introduced following a full explanation of the survey to all nurses working in each ward area. No comparison of scores was undertaken nor was accuracy of skin assessments evaluated. The tool was not supported by operational definitions for each item, and in view of the large number of items and problems of meaning reported with the Norton score, the tool probably has poor reliability.

## Independently assessed

It can be concluded that the 100 per cent sensitivity was achieved by over-prediction of patients at risk, and that reliability is likely to be poor. These conclusions are supported by Dealey (23) and Wardman (24) who independently compared Waterlow and Norton scores. Both studies are limited because they use prevalence not incidence data, but are currently the only studies available. Using data published within the text, reliability data were calculated for both Waterlow and Norton results (Table 3). Although the Waterlow correctly categorises 98 and 100 per cent of patients with sores as at risk, the specificity is extremely poor, indicating gross over-prediction of patients at risk.

No reliability measures were recorded during the data collection from the 175 sampled patients in Dealey's study, but a separate study involving

student nurses was undertaken. The learners on four wards independently assessed the same five patients using the Waterlow and Norton scores and per cent agreement of scores +/- 1 point were calculated. Norton was reported to have 70 per cent reliability and Waterlow only 60 per cent.

In the study by Wardman (24), the scores reported represented the consensus view of a cross-section of qualified and unqualified staff involved in the assessment process. Any difficulties in the scoring of patients were not detailed.

Despite these criticisms, the tool was adopted nationwide (9, 23, 25, 26). The limitations of the tool, however, prevent its use in the assessment of the relative risk of pressure sore development.

On further exploration of the literature, an American development called the Braden scale was revealed.

**The Braden scale** The Braden scale, composed of six subscales, was developed from a literature review of the aetiological factors involved in pressure sore formation. The critical factors were found to be the intensity and duration of pressure and tissue tolerance to pressure (3). From these, the six subscales are derived - mobility, activity and sensory perception reflecting the intensity and duration of pressure, and skin moisture, nutritional status and friction reflecting tissue tolerance.

Each subscale has 3/4 levels which all have an operational definition. They are rated from 1 (least favourable) to 3/4 (most favourable) and total scores range from 6 to 23. The point at which patients are deemed to be at risk for developing pressure sores is 16 points.

Initial work focused upon the reliability of the tool and three studies were conducted to establish interrater reliability among different grades of staff. One study of 22 subjects compared scores obtained from a graduate student and registered nurse in a rehabilitation setting. Absolute agreement was 88 per cent and +/-1 point agreement was 100 per cent (27).

Further comparisons, involving licensed practical nurses and nursing assistants in a long-term elderly care institution, did not produce such good results. Many ratings were close but few were identical as a result of literacy level differences, interpretation difficulties among both staff grades and poor knowledge of the patients. The conclusion drawn was that the tool should be used by registered nurses (27). In a later study, interrater reliability was reported as r=0.89 (Pearson's product moment correlation). Details of per cent agreements were not given (28).

Following the reliability studies, the validity of the tool was explored in medical and surgical units (27). Primary nurses recorded skin assessments and primary or associate nurses rated the patient using the Braden scale at weekly intervals until discharge, transfer or death.

One hundred consecutive patients were studied on each unit and a pressure sore incidence of 7 and 9 per cent recorded (where the pressure sore definition included persistent erythema for 24 hours or more). Sensitivity and specificity calculated for each score indicated that 16 points or less was the most accurate in predicting risk. At this level sensitivity was 100 per cent and specificity 90 per cent for one unit and 100 per cent and 64 per cent (respectively) for the other.

In the later study (28) results were not as favourable, although the method of data collection was markedly different. In this study patients admitted to an adult intensive care unit were rated using the Braden scale within 24-72 hours of admission by the primary nurse. No further Braden score was calculated. Skin assessments were then performed every 48 hours for two weeks or until discharge from the hospital.

A total of 60 patients were studied and a pressure sore incidence of 40 per cent established. A score of 16 was 83 per cent sensitive and 64 per cent specific. The predictive value of the positive score was 61 per cent and the predictive value of the negative score 85 per cent (28).

## Good design

Results may vary because only one Braden score was recorded in the later publication, and the skin assessments, apparently undertaken by the investigators, may have resulted in more accurate documentation of persistent erythema than the former publication which relied upon primary nurses.

Regardless of differences, the results of the validity analyses are good with margins of error ranging from 0-15 per cent for under-prediction and 0-37 per cent for over-prediction of those at risk. Methods employed to test the tool have been rigorously planned and researcher bias minimised by good design.

**Conclusion** The Braden scale is reliable when assessment is undertaken by registered nurses and is the most reliable tool described in the nursing literature. Validity of the tool is generally good and compares favourably in comparisons with the Norton and Waterlow scores.

The clinical implications of the differences in sensitivity and specificity between Waterlow and Braden (ie.whether it is better to over-predict by many cases or under-predict a few) requires further debate and investigation ●

15. Newman P, West J. Pressure sores 2 - the value of the Norton score. *Nursing Times*. 1981. 29, 21, 15-21.
16. Goldstone L A, Roberts B V. A preliminary discriminant function analysis of elderly orthopaedic patients who will or will not contract a pressure sore. *International Journal of Nursing Studies*. 1980. 17, 1, 17-23.
17. Gosnell D J. An assessment tool to identify pressure sores. *Nursing Research*. 1973. 22, 1, 55-59.
18. Pritchard V. Calculating the risk. *Nursing Times*. 1986. 82, 8, 59-61.
19. Williams C. A comparative study of pressure sore prevention scores. *Journal of Tissue Viability*. 1992. 2, 2, 64-66.
20. Stotts N A. Predicting pressure ulcer development in surgical patients. *Heart & Lung*. 1988. 17, 641-647.
21. Towey A P, Erland S M. Validity and reliability of an assessment tool for pressure ulcer risk. *Decubitus*. 1988. 1, 2, 40-48.
22. Waterlow J. The Waterlow card for the prevention and management of pressure sores: towards a pocket policy. *Care Science and Practice*. 1988. 6, 1, 8-12.
23. Dealey C. Risk assessment of pressure sores: a comparative study of Norton and Waterlow scores. *Nursing Standard*. 1989. 3, 27, supp. 11-12.
24. Wardman C. Norton v Waterlow. *Nursing Times*. 1991. 87, 13, 74-78.
25. Richardson B. Pressure sores - a manager's perspective. *Nursing Standard*. 1990. 5, 12, suppl. 11-13.
26. Millward J. Relieving the pressure. *Nursing the Elderly*. 1990. 2, 4, 14-16.
27. Bergstrom N *et al*. The Braden Scale for predicting pressure sore risk. *Nursing Research*. 1987. 36, 4, 205-210.
28. Bergstrom N *et al*. A clinical trial of the Braden Scale for predicting pressure sore risk. *Nursing Clinics North America*. 1987. 22, 2, 417-428.

# Pressure sore risk in operating theatres

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*Little information is available on the genesis of intra-operative pressure sores. A pilot study was set up to investigate whether it is necessary, to prevent pressure sores developing in the operating department. Skin assessments and Braden score readings were taken pre-operatively and post-operatively. Half of the 26 patients had skin changes pre-operatively and there was a 'theatre generated' incidence of 12.5 per cent. The author concludes that further investigation should be commenced.*

*This study was made possible by a £5,000 grant from the Nuffield Hospital Theatre Nurse Award and undertaken at Hartlepool Hospital.*

Pressure sores are defined as 'a lesion on any skin surface that occurs as a result of pressure and includes reactive hyperaemia as well as blistered, broken or necrotic skin' (1). Studies have revealed hospital prevalence rates ranging from 6.7 to 10.1 per cent (2, 3, 4, 5). Pressure sores are increasingly being viewed as preventable sequelae rather than a tolerable complication of illness and the emphasis is on identifying risk factors and implementing appropriate interventions.

A comprehensive review of the literature finds few studies on the genesis of intra-operative pressure sores, and the contribution of operating room practice on aetiology is undefined (6).

In an attempt to answer the question of whether pressure sore prevention in the operating department is necessary or possible, a research method was developed. A ten-week pilot study of the research design has recently been completed and the results do suggest that the main study will identify a causal relationship between time on the operating table and post-operative pressure sore development.

The aims of the pilot study were to:

- Identify unforeseen problems in the practical application of the research method
- Allow the research co-ordinator to review objectively the number of staff and level of support required for the research team
- Explore the reliability and validity of the Braden Scale in a British hospital setting
- Provide data that may validate the control group data of the main study.

Patients over 55 years of age undergoing major surgery were selected from general and vascular surgical lists. Data collection was co-ordinated by the author and undertaken by designated qualified staff within the surgical unit.

The information collected from the patients included age, sex, type of surgery and operating table position. Key elements of the data were skin assessments using an adapted version of Torrance's pressure sore classification (7) (Table 1), and determination of the risk of pressure sore development by calculation of Braden scores (8).

The Braden Scale, composed of six subscales, was developed from a literature review of the factors involved in pressure sore formation (8). Critical determinants included the intensity and duration of pressure and the tolerance of the skin to pressure. From these, the six subscales are derived – mobility, activity, sensory perception, skin moisture, nutritional status, and friction and shear.

Each subscale has three or four levels, which all have an operational definition. They are rated from 1 (least favourable) to 3/4 (most favourable) and total scores range from 6-23. The cut-off point at which patients are judged to be at risk of developing pressure sores has been set, following clinical validation, at 16 points or lower (9).

| **Table 1. Skin assessment scale (7).** |
|---|
| **Grade 0** No discolouration of skin |
| **Grade 1** Redness to skin - blanching occurs |
| **Grade 2** Redness to skin - non-blanching occurs and/ or superficial damage to epidermis |
| **Grade 3** Ulceration progressed through to dermis |
| **Grade 4** Ulceration extends into subcutaneous fat |
| **Grade 5** Necrosis penetrates the deep fascia and extends to muscle |

## Reliability checks

Results of both reliability and validity studies (9, 10) suggest the Braden Scale is the most appropriate tool for use in research. It is the most reliable tool described in the nursing literature and the validity is generally good and compares favourably with the Norton and Waterlow scores.

Explanations and demonstrations of the tools used in the research proforma were given to the staff involved before the pilot study, and inter-rater reliability of the skin and Braden score assessments was examined before the study.

During the study, reliability checks were made by the researcher, who visited each patient two or three times during the period of data collection, conducted unstructured interviews and consulted nursing notes before independently scoring patients on the Braden Scale. Results were compared with the ward nurse assessment and discrepancies were noted.

**Table 2. Pre-operative skin change by grade and area.**

| | Number of patients | Number of skin areas | Heels | Buttocks | Sacrum |
|---|---|---|---|---|---|
| Grade 1 | 13 | 20 | 13 | 6 | 1 |
| Grade 2 | 3 | 3 | - | - | 3 |
| Total | 16 | 23 | 13 | 6 | 4 |

**Table 3. Pre-operative skin change/no change in relation to age, pre-operative Braden score, premedication time and starvation period.**

| | Number of patients | Average Braden | Average age | Average pre-med | Average starvation |
|---|---|---|---|---|---|
| Skin change | 13 | 20.75 | 73.5 | 2h5min | 13h33min |
| No change | 13 | 22.07 | 69.3 | 3h6min | 13h16min |
| All patients | 26 | 21.4 | 71.4 | 2h58min | 13h24min |

**Table 4. Pre-operative skin damage/no damage in relation to age, Braden score, and time on the operating table.**

| Skin damage | per cent of patients | Average time on table | Average age | Average Braden score Day 1 PO |
|---|---|---|---|---|
| None | 29% | 2h2min | 69.1 years | 16.7 |
| Immed | 35% | 1h54min | 71.7 years | 17.3 |
| 1/2 -1h | 31.5% | 2h38min | 70 years | 16 |
| Day 1+ | 12.5% | 2h5min | 76 years | 15.3 |

**Results** For the purposes of data analysis, results were divided into three sections; pre-operative data, theatre data and, pre/post-operative combined data.

A pressure sore was defined as persistent skin discolouration at the same site for two consecutive days or more (11). This renders much of the reactive hyperaemia noted post-operatively as obsolete. To provide details of the pattern of skin damage that emerges, however, such data is reported as 'skin changes'.

A total of 26 patients were recruited comprising ten men and 16 women with an age range of 56 - 87 years (mean 71.4 years, median 70 years).

**Pre-operative data** Braden scores ranged from 14 to 23, (mean 21.4, median 23). The majority of patients (n=25) were assessed as 'not at risk' (score of more than 16).

Pre-operative questioning of patients on the ward and a review of nursing notes revealed no pre-operative skin damage for any patient on the day before surgery. Skin assessments made in the anaesthetic room, however, revealed that 50 per cent of patients (n=13) had skin changes on a total of 23 skin areas observed (Table 2). Of these, three patients were noted to have a Grade 2 sacral sore.

Because of the high proportion of patients affected, further analysis was undertaken to try to link factors such as age, pre-operative Braden scores, premedication times, starvation periods and admission dates, with pre-operative skin changes. All factors showed similar results between no change/skin change patients (Table 3), with the exception of the pre-surgery admission period.

## Observed skin changes

Seventy-nine per cent of patients (7/8) who were in hospital for four or more days before surgery had observed skin changes. This compares with 31.25 per cent of patients hospitalised only one day pre-operatively (5/16). Furthermore, the three Grade 2 sores observed pre-operatively were all noted on patients who were hospitalised for four or more days.

Follow-up of 24 patients in the post-operative period revealed that 8/11 patients had pre-operative skin changes which persisted for one to eight days. Using the working definition of a pressure sore as described earlier, a pre-operative incidence of 33.3 per cent is determined.

**Theatre data** Following pre-operative skin assessments, two patients were excluded from the study (as three or more areas of skin damage were noted).

The 24 remaining patients comprised ten men and 14 women with an age range of 56-87 years (mean 70.4, median 69).

'Areas of skin damage' refers only to 'new' skin changes as those noted pre-operatively were excluded in the post-operative analysis.

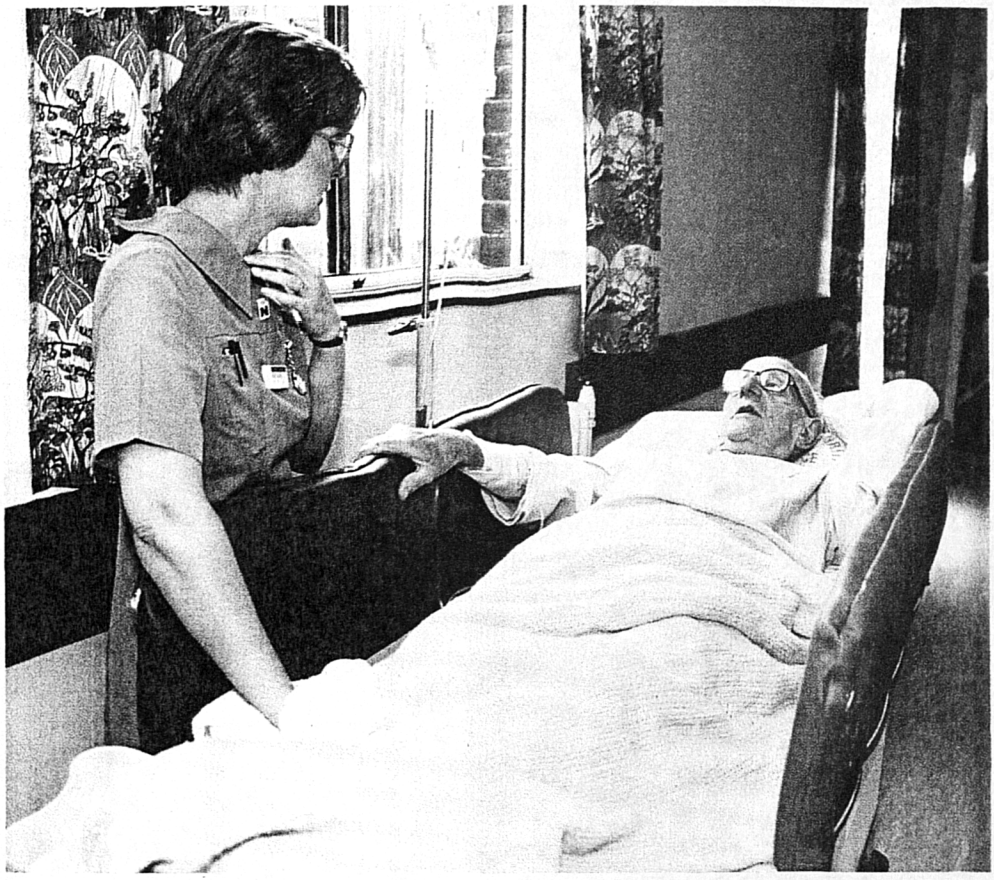
Patients spent between 1 hour 5 minutes and 5 hours 3 minutes on the operating table (mean two hours 11 minutes, median 1 hour 50 minutes). Eleven patients were on the operating table for 2 or more hours (mean age 70), and 13 under 2 hours (mean age 71.23).

Surgery performed on these patients included cholecystectomy (n=13), bowel resection (n=9), arterial grafting (n=2), and miscellaneous (n=3).

Seventy-one per cent of patients were noted to have at least one new area of blanching hyperaemia when first assessed by recovery staff, and 44 per cent had skin areas that remained red up to one hour post-operatively.

Using the working definition of a pressure sore, follow-up revealed a 12.5 per cent incidence of Grade 1 pressure sores on the first day post-surgery that persisted for one to five days. None of this Grade 1 skin damage, however, progressed to a Grade 2 pressure sore.

**Post-operative data** Further analysis of the pattern of skin damage was undertaken in relation to age, time on the operating table and post-operative Braden scores (Table 4).



MARK PINDER

**Off to theatre: Time spent on the operating table could be linked to pressure sore development.**

**Pre and post-operative combined data** The aim of combining all the data collected pre and post-operatively was to provide a general picture of the study group during hospitalisation and assess the validity of the Braden Scale.

Seventy-nine per cent of patients (n=19) were noted as having skin changes, on a total of 38 different skin areas during their hospital stay for varying periods of time. Using the working definition of a pressure sore, an incidence of 54 per cent (n=13) was determined – this included a 12.5 per cent (n=3) incidence of Grade 2 sore.

A total of 13 patients were assessed on the Braden score as being 'at risk' of pressure sore development on at least one day during hospitalisation with periods of risk ranging from one to nine days, (mean 3.5, median 3.5). By post-operative Day 8, only two patients remained 'at risk'.

Comparison of Braden scores with recordings of skin damage determined post-operative Day 1 as the most sensitive in accurately predicting those patients who may or may not be 'at risk'.

The validity of the scale was examined by calculating the sensitivity and specificity of the scores. The sensitivity – defined as the percentage of patients who developed pressure sores and were so predicted by the scale (12) – was 77 per cent. The specificity of the scale – defined as the percentage of patients who did not develop pressure sores and were so predicted by the scale (12) – was 73 per cent.

In respect of reliability, results were very good. Of the 24 patients scored, ward and researcher scores were within +/- 1 and classification of 'at risk/not at risk' was the same for 20 patients. The main areas of disagreement were the nutrition and friction and shear subsections.

**Summary** Documentation of the skin condition of a group of surgical patients revealed a number of interesting results. Pre-operative skin changes were observed on 50 per cent of those sampled and post-operative changes occurred on 71 per cent – a pattern emerging with continued follow-up and a 12.5 per cent 'theatre incidence'. During the nine- day study, there was an overall pressure sore incidence of 54 per cent.

**Discussion** The observation of skin changes among 50 per cent of the sample in the pre-operative period is an unexpected finding of the pilot study.

Only Versluysen (11) has previously reported pre-operative skin damage. She found an incidence of 27 per cent among 100 patients over 70 years

References

1. Parish L C *et al. The Decubitus Ulcer.* New York, Masson Publishers. 1983.
2. Barbenel J C *et al.* Incidence of pressure sores in the Greater Glasgow Health Board Area. *Lancet.* 1977, 2, 54.
3. David J A *et al. An Investigation Of The Current Methods Used In Nursing For The Care Of Patients With Established Pressure Sores.* Northwick Park, Nursing Research Unit. 1983.
4. Girvin J, Griffiths-Jones A. Towards prevention. *Nursing Times.* 1989. 85, 12, 64-66.
5. Dealey C. The size of the pressure sore problem in a teaching hospital. *Journal of Advanced Nursing.* 1991. 1, 3-7.
6. Bridel J. Pressure sore development and intra-operative risk. *Nursing Standard.* 7, 5, 28-30.
7. Torrance C. Pressure sores. *Aetiology, Treatment And Prevention.* London, Croom Helm. 1983.
8. Braden B J, Bergstrom N. A conceptual schema for the study of the etiology of pressure sores. *Rehabilitation Nursing.* 1987. 12, 1, 8-12/16.
9. Bergstrom N *et al.* A clinical trial of the Braden Scale for predicting pressure sore risk. *Nursing Clinics of North America.* 1987. 22, 2, 417-428.
10. Bergstrom N *et al.* The Braden Scale for predicting pressure sore risk. *Nursing Research.* 1987. 36, 4, 205-210.

old admitted for femoral fracture who were exposed to high risk pre-operative factors such as lengths of time spent on casualty trolleys, periods of bed rest in traction and the presence of urinary incontinence among 71 per cent of the study group.

In view of the relatively low risk of the pilot sample the results compare unfavourably.

Speculation of the likely causes of such extensive skin changes is limited since actual skin assessments were not performed until patients entered the anaesthetic room. The results do, however, highlight the need for further investigation to determine whether aspects of theatre preparation are detrimental to skin integrity.

The pattern of post-operative skin changes that emerged in this study have no direct comparison, since other studies using a similar working definition of a pressure sore have effectively ignored immediate post-operative reactive hyperaemia unless it has persisted to Day 1 post-surgery (11, 13). The skin change patterns that emerge in relation to age, Braden score, and time on the operating table are consistent, however, with knowledge of pressure sore development (2, 10).

For example, a greater proportion of patients over 70 years exhibited post-operative reactive hyperaemia than those under 70 years. Also, on average, periods of time on the operating table in excess of two hours produced more reactive hyperaemia than periods of less than two hours.

Age and time, however, as single factors are not predictive and this is consistent with results from the study by Kemp *et al* (13), as well as the literature relating to tissue tolerance and pressure sore aetiology (10, 14).

## Anticipation useful

A similar pattern emerges with Braden scores although it is interesting to note the slower recovery time of skin on patients determined as at risk. Of further note is that immediate post-operative reactive hyperaemia is persistent to post-operative Day 1 only on those patients who are identified as at risk by their post-operative Braden score. It suggests that anticipation of the post-operative Braden score may be useful in predicting the progression of reactive hyperaemia post-operatively and this will be considered in the design of the main study.

The actual percentage of patients noted to have reactive hyperaemia during the immediate post-operative period (71 per cent) is a reflection of the high pressures known to be generated at the operating table/patient interface, though previously unquantified in terms of skin damage (6, 15,16). In view of the large percentage of patients affected, the pilot illustrates the need to continue the line of investigation and validate results.

The 'theatre generated' 12.5 per cent incidence observed is similar to that reported by Versluysen (11) and Kemp *et al* (13). Although direct comparison of the results is not possible because of differences in sampling, the post-operative incidence of pressure sores lies within expected boundaries.

## Further investigation

The theatre specific data has provided a pattern of skin change that is supported by the literature relating to tissue tolerance, interface pressures and post-operative incidence rates. In view of the large number of patients affected and a lack of comparable data the results indicate the need to proceed with further investigation.

**Pre and post-operative data combined** Comparison of the overall pressure sore incidence of 54 per cent with other results must be done with caution. Studies have reported incidence rates of 12 per cent and 17 per cent but the respective age range of the patients reviewed were 23-84 years and 17-86 years, and sample sizes 125 and 387 (13, 17).

Comparison with the Versluysen study, which reported an incidence of 66 per cent, must also be made cautiously since the incidence rate alone does not truly reflect the overall extent of tissue damage. The 66 patients identified as having pressure sores shared a total of 225 lesions, most of which were classified as Grade 2 or more (11).

**Braden Scale** The validity of the Braden Scale in a British hospital setting compares favourably with results of the Norton Score (18) and Waterlow Card (19), and results support its use in future research.

Problems with the reliability of the tool were observed during the course of the pilot study and highlighted the need for preparation before the study. A review of the literature reveals poor reliability for the Norton Score and Waterlow Card (18, 19), so despite the acknowledged discrepancies noted, results support the use of the Braden Scale in the main study.

**Summary** That 50 per cent of the sample were observed as having skin changes pre-operatively was an unexpected finding of the study. This, together with the observation of skin changes in the immediate post-operative period and a resulting 12.5 per cent 'theatre generated' incidence, indicate the need to proceed with further investigation

11. Versluysen M. How elderly patients with femoral fracture develop pressure sores in hospital. *British Medical Journal*. 1986. 292, 1311-1313.
12. Lilienfeld A M, Lilienfeld D E. *Foundations of Epidemiology*. 2nd Edition. New York, Oxford University Press. 1980.
13. Kemp M G *et al*. Factors which contribute to pressure sores in surgical patients. *Research in Nursing Health*. 1990. 13, 5, 293-301.
14. Krousop T A. A synthesis of factors which contribute to pressure sore formation. *Medical Hypothesis* 1983. 11, 255-267.
15. Neander K D, Birkenfeld R. Decubitus prophylaxis in the operating theatre. *Journal of Tissue Viability*. 1991. 1, 3, 71-74.
16. Campbell K. Pressure point measures in the operating room. *Journal of Enterostomal Therapy*. 1989. 1, 3, 119-124.
17. Stotts N A. Predicting pressure ulcer development in surgical patients. *Heart and Lung*. 1988. 17, 1-7.
18. Taylor K J. Assessment tools for the identification of patients at risk of development of pressure sores: a review. *Journal of Enterostomal Therapy*. 1988. 15, 5, 201-205.
19. Waterlow J. The Waterlow card for the prevention and management of pressure sores: towards a pocket policy. *Care Science and Practice*. 1988. 1, 8-12.

# The epidemiology of pressure sores

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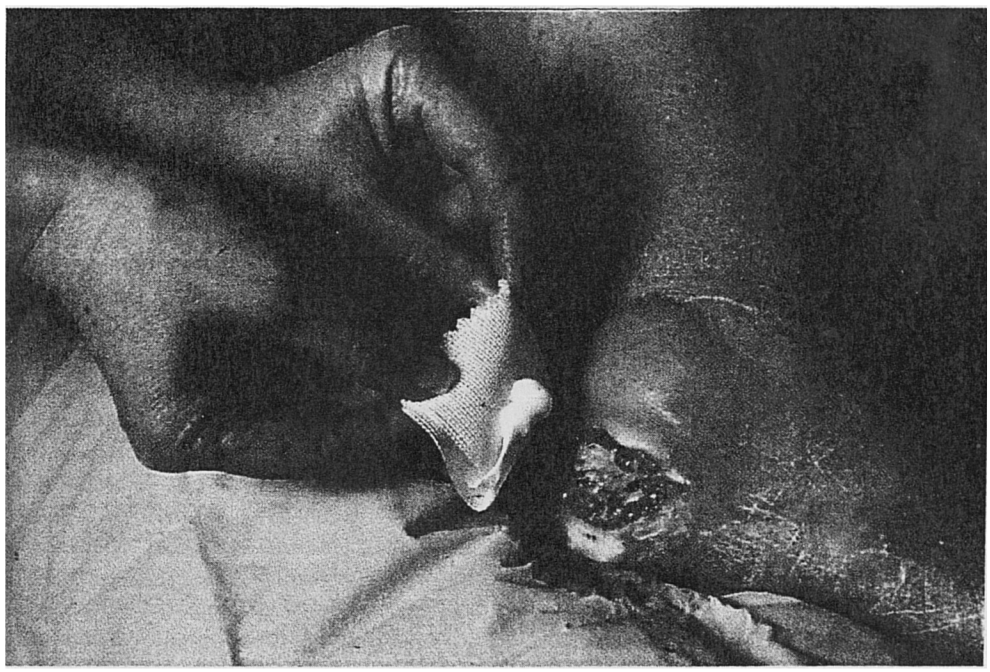

Areas not adapted to weight-bearing such as heels are a common site for pressure sores, supporting the link with impaired mobilty.

*This article presents details of epidemiological studies undertaken to assess the extent of pressure sores in hospital. Both prevalence and incidence studies, which reveal the full extent of the problem and its predisposing factors, are discussed. The author also outlines the statistical terms used by epidemiologists as well as general considerations which must be made in the interpretation of epidemiological studies.*

Health service personnel tend to have a limited picture of the disease and the health status of their local population, as their impressions are based upon everyday contacts with a succession of individual patients and their families (1). Studies of epidemiology which are based on whole populations or representative samples rather than individuals or patients can, therefore, present a perspective of the range and pattern of health and disease in human populations which is not otherwise available. Such studies also add a greater breadth and understanding to the causes, predisposing factors and natural history of diseases and provide data necessary for the management, planning and evaluation of services for the promotion of health and prevention and treatment of disease (2, 3, 4).

**Understanding epidemiology** The science of epidemiology has traditionally been the responsibility of the medical profession (5), though it is not restricted to an identifiable group of trained individuals since many sources of data are readily available and the tools are simple and relatively cheap (4). Nursing's involvement with epidemiology is gradually increasing as demands for objective data are made from managers within the National Health Service (6) and advocates of good practice management (7).

The results of epidemiological studies are usually expressed in statistical terms and have a basic unit of measurement: the rate. This has three components (1):

The numerator (the total number of people who experience the event)

The denominator (the total number of people in the population potentially at risk)

A specified time period during which events take place.

**Crude rates** A crude rate uses the entire population as the denominator and, though it has the ability to convey an impression in a single figure, it is somewhat limited in its application since aspects such as the age and sex of a popu-

lation are not considered. As pressure sores affect mainly hospitalised and/or severely disabled people, the crude rate is not a useful indicator and is rarely used (8).

**Specific rates** Specific rates look beyond the crude rate and describe the number of events occurring within a subgroup of the population. They allow comparison between groups within, and from, different populations and may give indications about the natural history of the disease and the cause or predisposing factors (1). Age and sex are perhaps the most commonly expressed specific rates, but others include race, social class and occupation – or, in the case of pressure sores, mobility and continence.

**Prevalence and incidence rates** Prevalence and incidence rates are both commonly used. Prevalence is a measure of the number of persons with a disease in a defined population either within a certain time (period prevalence) or at a specific point in time (point prevalence) (1). It is a useful indicator of the extent of chronic disease and disability and is useful in terms of pressure sores for accurately portraying the burden upon services of Grade 4 and 5 sores.

Incidence is the proportion of subjects who first present with a given problem during a defined period of time, in relation to the local population at risk (9). It is a useful measure of the extent of burden created by short-lived or quickly recoverable diseases/problems and is a sensitive measure of Grade 1 and 2 pressure sores.

The reliability and validity of the results of epidemiological study ultimately reflect the accuracy and completeness of the data and the method of data collection (10). Errors can arise in the number of identified cases (numeracy error) or when the defined population size is inaccurate (denominator error).

**Problems with epidemiological studies** It is acknowledged that in epidemiological study 'optimum conditions rarely apply, and there is always some degree of error' (4). Problems encountered by epidemiologists are often out of their control since completeness of data depends on three factors:

- Every individual with a given condition actually presents with it to the health service
- Upon presentation, the health care professional or data recorder recognises the condition
- The condition is accurately documented and recorded.

In the epidemiological study of pressure sores it is the second and third factors which are most limiting in determining the true extent of the problem. Difficulties in recognising the problem arise from lack of knowledge, differences in the interpretation of the term pressure sore, attributing skin damage to other causes (such as burns), and simply failing to observe (8, 11-14).

Similarly, inaccurate documentation and reporting of pressure sores arises from lack of knowledge and differences in the interpretation of the term, as well as attitudes of the institution and priority given to them (15, 16).

Previously, inadequate understanding of the aetiology of sores and a lack of appreciation that the process is exacerbated by events in hospital meant that the development of sores was considered an indication of poor nursing care.


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**Arrival in hospital: Incidence studies show that most sores develop in the first two weeks after admission.**

**Table 1. Summary of point prevalence results.**

| Study | Prevalence rate % | Sample size | Grades of sore | Method | Exclusions |
|---|---|---|---|---|---|
| Barbanel *et al* [8] | 8.8 | 10,751 | 2-4 | Questionnaire | Maternity Learning difficulties Psychiatry |
| Barbanel *et al* [17] | 9.4 | 999 | 2-4 | Questionnaire | Maternity Neonates Psychiatry |
| David *et al* [14] | 6.6 | 13,409 | 1-4 | Interview | Maternity Psychiatry |
| Nyquist and Hawthorn [18] | 5.3 | 2,513 | 1-4 | Questionnaire | Maternity Psychiatry |
| Girvin and Griffiths-Jones [19] | 10.2 | 1,010 | 1-4 | Interview and records | Maternity Paediatric Psychiatry |

## SUMMARY BOX 1 Prevalence studies

- Prevalence studies indicate that a large number of hospital patients have pressure sores, ranging from 5.3 per cent to 10.2 per cent. The prevalence rates reported provide a general guide to the occurrence of the problem within the hospital setting but are not a precise measure.
- Factors which may predispose to pressure sore development, such as age, mobility and incontinence are identified by comparing pressure sore positive and negative groups. The studies suggest that no one single causative factor exists.
- Almost all pressure sores are shown to occur below the waist (with particularly vulnerable areas being the sacrum, buttocks and heels), and slightly more than half are the Grade 2 classification.

This resulted in feelings of guilt and denial among nurses who were then reluctant to discuss the subject (13).

Evidence of this is found in studies by Hibbs (16), who reported no significant reduction in the incidence of new sores over a three-year period, and Richardson (15) who noted a two-fold increase in prevalence rates, following the introduction of a policy of prevention, investment in resources and education of staff. Both authors attributed these results not only to improved recognition of pressure sores, but also to a reduction in the guilt and denial associated with pressure sores.

***Appraisal of studies*** *Each study must be taken on individual merit as methods, sample sizes, time and resources all vary. Many published studies are written by clinical nurses who have undertaken the work in the course of their normal duties, whereas other accounts are specially commissioned and have a team employed solely for the purpose of research. Techniques, attention to detail, statistical analysis and written reports vary immensely in the field, and it is essential to gain a basic knowledge of research appraisal so that informed interpretation precedes application to practice or planning. In the context of the issues highlighted, the results of prevalence and incidence studies are now discussed.*

**Prevalence studies** A number of point prevalence studies have been undertaken in the past 25 years (8, 14, 17-19) and results are very similar despite differences in methodology, pressure sore definition, and the size of surveyed populations (Table 1). The rates, ranging from 5.3 per cent to 10.2 per cent, all reflect the prevalence within health regions, health authorities and hospitals without a pressure sore prevention policy.

**Reliability** Attempts to test the reliability of methods and determine the accuracy of pressure sore reporting were made by Barbanel *et al* (8) and David *et al* (14). They sampled a small number of wards and the researchers independently surveyed all patients individually, as opposed to the main study method of using the ward nurse in charge to provide details.

Barbanel *et al* (8) found there were more disagreements in the reporting of Grade 1 pressure sores among the seven nurses who were involved in the check survey, and as a result this grade was omitted from their analysis. It is unfortunate that no other reference is made to this problem by other authors. In addition, reliability checks suggested that the number of hospitalised patients with pressure sores would be underestimated in the main survey by 2-3.5 per cent. Indeed, similar conclusions were also made by David *et al* (14) who estimated an overall shortfall in reporting of approximately 2.4 per cent.

At first glance, the underestimation of cases appears to be supported by the work of Waterlow (20, 21) who published details of a period prevalence study. A total of 649 patients from medical, surgical, orthopaedic, geriatric, trauma, coronary and intensive care wards were included in the study, whether they were already inpatients when the study commenced or were admitted during the study period.

A period prevalence of 17.1 per cent was recorded suggesting that the results of earlier point prevalence studies may be extremely inaccurate. Three important aspects limit the validity and reliability of these results, however, and clearly illustrate some of the pitfalls involved in critically reviewing epidemiological study.

First, Waterlow excluded short-stay patients (two days and under) from the survey and, in so doing, changed the denominator population, thus preventing direct comparison with other point prevalence results. Second, the main researcher (Waterlow) examined more than 90 per cent of patients herself, and could well have introduced bias. Third, the results of the check survey by Barbanel *et al* (8) cast doubts on the value of Waterlow's work since the Grade 1 classification of pressure sore was included without consideration or testing of the reliability.

The prevalence rates reported, therefore, provide a guide to the occurrence of the problem in the hospital setting, but it is not a precise measure, since numerator errors are apparent and denominators have different terms of reference.

Other information obtained from these preva-

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**Prevalence studies reveal that women over 65 years of age with impaired mobility are most commonly reported as having pressure sores.**

lence studies relates to patient characteristics – for example age, gender, state of continence and mobility – and also pressure sore characteristics, such as the anatomical sites affected and the severity of grades.

**Age and sex** In relation to patient characteristics, various factors have been established. The majority of patients with pressure sores are over 65 years old; they are more commonly seen in women than men; and many patients are immobile (bed- or chair-fast) and/or incontinent.

**Table 2. Summary of incidence studies.**

| Study | Sample size | Time scale | Speciality group | Age range | Grades of sore | Incidence % |
|---|---|---|---|---|---|---|
| Hicks[24] | 100 | ? Admission-14 days | Surgery over 2 hours | 10 days-85 years | 1-3 | 13 |
| Stotts[25] | 387 | Admission-3 weeks | Surgery | 22-81 years | 1-4 | 17.3* |
| Kemp *et al*[26] | 125 | Admission-10 days post-op | Surgery | 23-84 years | 1-4 | 12 |
| Norton *et al*[28] | 248 | Admission-discharge | Elderly care | >65 years | 2 | 24 |
| Gosnell[29] | 30 | Admission-4 weeks | Elderly care | >65 years | 2 | 13.3 |
| Clark and Kadhom[29] | 88 | Admission-6 weeks | Orthopaedic Elderly ITU | Chair-/bed-fast | 2 | 29.5 |
| Versluysen[30] | 100 | Admission-15 days | Femoral fracture | >70 years | 1-5 | 66* |
| Gebhardt[31] | 74 | Admission-15 days | Femoral fracture | 62-99 years | 1-5 | 43 |

* includes patients admitted with pressure sores

Interpretation of many results are limited, however, since with the exception of Barbanel *et al* (8) and Waterlow (21), the characteristics of the pressure sore positive groups are analysed in isolation of the main (pressure sore negative) population. The specific rates are not analysed.

The limitations of this data can be shown by studying gender data. The fact that more female than male patients suffer pressure sores can be simply attributed to the fact that they constitute a higher proportion of the over-65 population and occupy more hospital beds. Unfortunately, false assumptions have been made (using the crude results) about individual patient risk, particularly in respect of age and sex.

The two studies which actually detail specific rates do, however, contribute greatly to the growing body of knowledge relating to pressure sore aetiology. Barbanel *et al* (8) and Waterlow (21) presented age-specific analyses which illustrated that prevalence increases with age.

**Mobility and continence rates** Perhaps of more interest, though, are mobility specific rates and continence specific rates. For example, patients who are totally helpless and chair-fast have a reported pressure sore prevalence of 25 per cent (8) and 40 per cent (17), whereas with semi-ambulant patients only 7.1 per cent and 6.5 per cent respectively have pressure sores. Similar results are detailed by Waterlow (21) for continence/incontinence. The correlation between pressure sore prevalence and age, therefore, may well be an artefact of the relationship between age and such factors as these.

These results also illustrate that many factors

**SUMMARY BOX 2**
**Incidence studies**

- Incidence studies report wide variations in rates which are accounted for by differences in the denominator population. Such wide variations are expected and consistent with results of prevalence studies. Despite the limitations of the incidence studies for determining Grade 1 pressure sores, comparisons to prevalence results reveal important trends.
- Differences in grade distribution suggest that pressure sores are both reversible and progressive in nature, and for the majority are a short-lived event.
- Further comparisons and specific documented evidence also indicate that a high proportion of pressure sores develop in the first two weeks following admission to hospital, but that increasing length of stay increases the likelihood of sore development.

may be involved in pressure sore aetiology, with no single cause being identifiable. For example, although 40 per cent of totally helpless chair-fast patients had pressure sores, 60 per cent did not (8).

The prevalence studies thus indicate factors which may predispose to pressure sore development when pressure sore positive and pressure sore negative groups are compared. They also suggest that no one single causative factor exists.

**Sites and severity of sores** In respect of the anatomical sites and severity of grade, the prevalence studies also provide valuable information and present similar results.

It is clear that nearly all pressure sores are found below the waist with figures of 96.5 per cent, 97.6 per cent and 97.1 per cent reported by Nyquist and Hawthorn (18), Girvin and Griffiths-Jones (19), and David *et al* (14) respectively. Most sores are found on the sacrum, heels and buttocks. These areas are not adapted to weight bearing (22) and are not normally exposed to unrelieved pressure (23), adding further evidence to the link between pressure sore development and impaired mobility.

The burden upon the health service of the severity of pressure sore is not easily assessed due to differences in definition of the term and resulting distortion of figures. Two trends are, however, worthy of comment. With the exception of David *et al* (14), Grade 2 pressure sores account for slightly more than half of all sores reported, and full thickness Grade 4 sores account for less than 20 per cent, indicating that most pressure sores are superficial in nature.

**Incidence studies** Results of incidence studies vary considerably, reported rates ranging from 12 per cent to 66 per cent (*Table 2*). Close scrutiny reveals similarities as well as wide differences in incidence depending on the population samples.

The three studies which explored the incidence of pressure sores among surgical patients of all ages record similar rates despite variations in the method used (24-26).

The other studies which recorded extremely high incidence rates (with the exception of Gosnell (27), whose sample was small) sampled patients who were elderly and/or had very limited mobility (28-31). Indeed, the wide variations in incidence are expected and consistent with results of prevalence studies. They reinforce the data indicating that increasing age and immobility are predisposing factors (24, 25, 29, 30) but provide no further evidence of the possible link to incontinence.

Attention to the composition of the denominator population of incidence studies is thus important.

**Reliability** In terms of the reliability of the data collected and the accurate determination of the numeracy population, pre-study preparation of data collectors (25), skin assessment verification by researchers (26) and the use of a working definition of the term pressure sore as 'a break in the skin' (28, 29) are described in the methodologies. Little further reference is made.

The likelihood of under-case ascertainment was mentioned only by Hicks (24), who reviewed patient records and suggested that some Grade 1 pressure sores which did not progress were probably not recorded. She acknowledged the limitations inherent in the use of existing data.

The three remaining studies make no reference to the reliability of the data recording process (27, 30, 31). In view of the findings of Barbanel *et al* (8), where major disagreements in the reporting of Grade 1 pressure sores were noted, the results of studies must be interpreted with caution.

Despite the limitations of the results of incidence studies and the wide variations in the incidence rates reported, when compared to prevalence results a number of important trends emerge in relation to the rates, grade distribution and onset of pressure damage.

**Grade distribution** All the incidence rates reported are higher than the prevalence rates, the differences accounted for by the much greater proportion of Grade 1 and 2 pressure sores recorded.

The two incidence studies which sampled a broad range of age groups reported that Grade 1 and 2 pressure sores accounted for 97.1 per cent (25) and 95.5 per cent (26) of all sores. Prevalence studies, on the other hand, reported Grade 1 and 2 sores as accounting for 45.4 per cent (14), 63.4 per cent (18) and 79.5 per cent (19) of all sores.

As expected, the continued monitoring of skin to determine incidence will detect the short episodes of persistent redness experienced by patients more accurately than a once-only point prevalence inspection. The magnitude of the difference observed suggests that for the majority, pressure sores are a short-lived event.

The absence of reports of Grade 4 pressure sores in all but one incidence study (30) is also worthy of comment, since prevalence studies record their proportions as 13.4 per cent (18), 17 per cent (8) and 22.8 per cent (14) of all sores.

Results suggest that the time scales of the incidence studies do not allow the potential effect of the slow progressive nature of sores to be realised, and their value in determining the high

cost-incurring Grade 4 pressure sores is limited.

Comparison of the results of incidence and prevalence studies and the differences in the grade distribution do, therefore, provide valuable information indicating that pressure sores are both reversible and progressive in nature.

**Onset of sores** Two important aspects of the onset of pressure sores are revealed by incidence monitoring. That is, the majority of pressure sores develop in the first two weeks following admission to hospital (25, 28, 30) and the likelihood of a patient developing a pressure sore increases with length of stay (25, 28).

Norton *et al* (28) provides the strongest evidence of the early development of pressure sores since all patients were followed from admission to discharge/death. In their sample of 248 patients, 59 developed pressure sores, 41 (69.5 per cent) within the first two weeks. Other figures are also reported by Versluysen (30), who observed that by the fifth day in hospital, 83 per cent of all patients affected by pressure sores had developed at least one lesion.

**Length of stay** In terms of length of stay, the likelihood of a patient developing a pressure sore is related to his or her relative risk. Stotts (25), for example, found a linear relationship between increased length of stay and increasing pressure sore incidence, but also noted the proportion of patients designated as high risk on a modified Norton scale increased with length of stay.

Similarly, Norton *et al* (27) found that most patients who developed pressure sores later than two weeks following admission were noted as having a deterioration in general condition, reflected by a reduced Norton score.

**Summary** A review of the literature describing the epidemiology of pressure sores provides information on the extent, predisposing factors and natural history of the problem. The review also illustrates common problems associated with the reliability of data collected and inappropriate interpretation of results.

Prevalence studies provide a general guide to the extent of the problem but are not a precise measure. Between 5-10 per cent of patients may be affected at any one time. Factors which may predispose to pressure sore development such as increasing age, reduced mobility and incontinence are identified, though results suggest that no single cause exists.

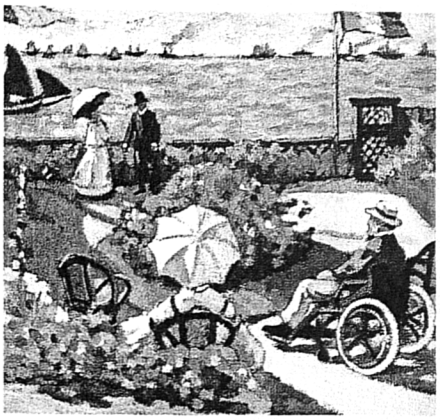
Incidence studies are consistent with the results of prevalence studies and reinforce the evidence about predisposing factors. They also suggest that pressure sores are both reversible and progressive in nature, and for the majority are a short-lived event experienced during the first two weeks following admission to hospital.

The epidemiology of pressure sores does, then, provide valuable information about how many and which patients may or may not develop pressure sores. A clear picture of the causes, however, is not provided, and questions on how sores might be prevented remain unanswered ●

References

1. Donaldson R, Donaldson R. *Essential Community Medicine (Including Relevant Social Sciences).* Lancaster, MTP Press. 1985.
2. Morris J N. *Uses of Epidemiology.* Third edition. Edinburgh, Churchill Livingstone. 1975.
3. Alderson M. *An Introduction to Epidemiology.* Second edition. London, Macmillan Press. 1983.
4. Holland W, Karhausen L. *Health Care and Epidemiology.* London, Henry Kimpton. 1978.
5. DHSS. *Management Arrangements for the Reorganised NHS.* London, HMSO. 1972.
6. DHSS. *NHS Management Enquiry Report.* London, HMSO. 1983.
7. DHSS. *Neighbourhood Nursing: A Focus for Care: Report of the Community Nursing Review Team.* London, HMSO. 1983.
8. Barbanel J C *et al.* Incidence of pressure sores in the Greater Glasgow Health Board Area. *The Lancet.* 1977. 2, 584-550.
9. Minotti A. Incidence studies and registers. In Holland W, Karhausen L (Eds). *Health Care and Epidemiology.* London, Kimpton. 1978.
10. McCarthy M. *Epidemiology and Strategies for Health Planning.* London, King Edward's Hospital Fund. 1982.
11. Gould D. Pressure sore prevention and treatment: an example of nurses' failure to implement research findings. *Journal of Advanced Nursing.* 1986. 11, 4, 389-394.
12. Gendron F. Burns occurring during lengthy surgical procedures. *Journal of Clinical Engineering.* 1980. 51,1,19-26.
13. Torrance C. *Pressure Sores: Aetiology, Treatment and Prevention.* London, Croom Helm. 1983.
14. David J A *et al. An Investigation of the Current Methods Used in Nursing for the Care of Patients with Established Pressure Sores.* London, Northwick Park Hospital, Nursing Practice Research Unit. 1983.
15. Richardson B. Pressure sores: a manager's perspective. *Nursing Standard.* 1990. 5, 12, suppl.11-13.
16. Hibbs P. Pressure sores: a system of prevention. *Nursing Mirror.* 1982. 155, 5, 25-29.
17. Barbanel J C *et al.* Major pressure of sores. *Health and Social Services Journal.* 1980. 90, 2, 1344-1345.
18. Nyquist R, Hawthorn P J. The prevalence of pressure sores within an area health authority. *Journal of Advanced Nursing.* 1987. 12, 2, 183-187.
19. Girvin J, Griffiths-Jones A. Towards prevention. *Nursing Times.* 1989. 85, 12, 64-66.
20. Waterlow J. A risk assessment card. *Nursing Times.* 1985. 81, 48, 24-27.
21. Waterlow J. The Waterlow card for the prevention and management of pressure sores: towards a pocket policy. *Care Science and Practice.* 1988. 6, 1, 8-12.
22. Braden B J, Bergstrom N. A conceptual schema for the study of the etiology of pressure sores. *Rehabilitation Nursing.* 1987. 12, 1, 8-16.
23. Exton-Smith A N, Sherwin R W. The prevention of pressure sores: Significance of spontaneous bodily movements. *The Lancet.* 1961. 2, 7212, 1124-1126.
24. Hicks D J. *An Incidence Study of Pressure Sores Following Surgery: ANA Clinical Sessions 1970.* New York, Appleton-Century Crofts. 1970.
25. Stotts N A. Predicting pressure ulcer development in surgical patients. *Heart and Lung.* 1988. 17, 641-647.
26. Kemp M G *et al.* Factors which contribute to pressure sores in surgical patients. *Research in Nursing and Health.* 1990. 13, 5, 293-301.
27. Gosnell D J. An assessment tool to identify pressure sores. *Nursing Research.* 1973. 22, 1, 55-59.
28. Norton D *et al. An Investigation of Geriatric Nursing Problems in Hospital.* Hong Kong, National Corporation for the Care of Old People. 1962.
29. Clarke M, Kadhom H M. The nursing prevention of pressure sores in hospital and community patients. *Journal of Advanced Nursing.* 1988. 13, 3, 365-373.
30. Versluysen M. How elderly patients with femoral fracture develop pressure sores in hospital. *British Medical Journal.* 1986. 292, 1311-1313.
31. Gebhardt K. Preventing pressure sores in orthopaedics. *Nursing Standard.* 1992. 6, 23, suppl. 4-6.

# The aetiology of pressure sores



A review of the anatomy of the skin and underlying structures and of the literature concerning processes involved in pressure sore development

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Epidemiological studies provide evidence of predisposing factors but not a clear-cut picture of the causes of pressure sores. Indeed, increased interest and research in the past 20 years has served to underline the complexity of the issues involved rather than to provide simple solutions.

It is now acknowledged that the development of a pressure sore is likely to be dependent on a complicated interplay of many variables, both intrinsic and extrinsic in nature[1].

In an attempt to provide an overview of the complex nature of pressure sore development the following article describes the anatomy of the skin, the blood supply of the skin and underlying structures and the pathophysiological process of tissue breakdown. These sections are followed by a review of the literature concerned with the critical determinants of pressure sore development — the intensity and duration of pressure and the tolerance of the skin and its supporting structures to pressure.

## Pressure sore physiology

### Anatomy of the skin

The tissues involved in pressure sore development are the skin, subcutaneous fat, deep fascia, muscle and bone.

Skin, described as the largest organ of the body[2], is a dynamic structure in which cellular replacement and modification in response to local need is a continual process throughout life[3]. It is relatively resistant to water, chemicals and bacteria and provides some protection for the body against mechanical damage.

Structurally, it consists of two layers — the epidermis, an outer avascular layer, and an inner layer known as the dermis.

The epidermis consists of stratified squamous epithelium which in turn has five distinct layers. The cells of the outermost layer (stratum corneum) contain little water, are tightly packed and provide a physical barrier against water, bacteria and chemicals. These cells are constantly being shed and replaced from the deeper layers.

The stratum spinosum contains two structures that contribute to the relative resistance of the skin to mechanical disruption — desmosomes and tonofibrils. Desmosomes are intercellular bodies formed by plasma membranes which link adjacent cells of the spinosum and tonofibrils are intracellular filaments found in bundles and link to the desmosomes[4].

Stratum germinativum is the deepest layer of the epidermis and consists of cells which continually undergo mitotic division and enable the skin to regenerate.

The dermis beneath contains a network of blood vessels, lymph vessels, nerves, gland and hair follicles. These structures, stabilised within the dermis by a tough flexible matrix of connective tissues (collagen and elastin), contribute to the regulation of body temperature, excretion of waste and sensory perception, and buffer internal organs from physical damage.

Interdigitation between the dermis and epidermis by dermal papillae and the flexible matrix of the dermis are both particularly important features that help protect against mechanical damage. Indeed the physical characteristics of the dermis are essentially determined by the collagen/elastin matrix.

Collagen is synthesised in connective tissue fibroblasts, secreted from the cells and stabilised by the formation of cross-

linkages which vary in permanence. It constitutes 99g/100 dry weight dermis[5]. The collagen fibres form a series of layers with fibres in adjacent layers aligned at a fixed angle.

When external pressure is applied the fibres, which are inextensible, rotate relative to one another until they approach a parallel alignment. As the fibres move nearer to a parallel alignment tension increases. When external pressure is removed the collagen is restored to its former open structure by elastic fibres that are intertwined around the collagen bundles[5].

The process of extension and recoil by rotation and alignment is an important aspect of the property of the collagen/elastin matrix because, as well as buffering internal structures of the body, it also protects the interstitial fluids and cells of the dermis from external pressure[2].

*A subcutaneous layer* separates the dermis from the deeper structures of deep fascia, muscle and bone. Containing similar structures as the dermis, it varies in thickness (depending on body type, gender and the location on the body) because of the presence of a large number of fat cells that provide mobility to skin and padding to dissipate pressure.

The deep fascia beneath is a dense, essentially avascular, inelastic membrane which covers muscle and muscle groups *and, over bony* prominences, may merge with the outer layer of the bone. It is resistant to pressure and is the last line of protection for vulnerable muscle tissue.

The skin, then, is characterised by a number of structures which allow protection from mechanical disruption. These include the desmosomes and tonofibrils of the epidermis, interdigitation of the dermo-epidermal junction and the collagen/elastin matrix of the dermis. Tissues beneath, including the layers of subcutaneous fat and deep fascia, also contribute toward protection of the skin's underlying structures.

Despite these characteristics, pressure sores do develop, mainly as a result of disruption to the vascular network of arteries, arterioles and capillaries.

### The vascular system and capillary blood flow

A network of vascular and lymph vessels carry the necessary nutrients and oxygen to support cell metabolism and epidermal mitosis, and enable the blood to facilitate temperature regulation and the removal of waste products from the skin.

The arteries supporting the skin pierce the deep facia and form a network of arterioles in the subcutaneous tissues with branches supplying the hair follicles and sebaceous and sweat glands. Arteries are vulnerable and prone to angulation where they pierce the deep fascia, and subcutaneous fat has poor tolerance to shearing forces and offers little protection to the arterioles from such disruption[4].

The arterioles branch into a network of metarterioles (throughfare vessels), capillaries and venules. These structures are known collectively as the micro-circulation[6]. Muscle cells at the origin of the capillaries act as pre-capillary sphincters and are important in the control of blood flow (Fig 1).

Perfusion and function of the capillaries are regulated and affected by both central and local control mechanisms which aim to fulfil two functions — nutrient and metabolite exchange and control of peripheral resistance.

The sympathetic nervous system, by the release of noradrenaline, controls peripheral resistance. It alters the tone of smooth muscle in the walls of the arterioles which, under normal conditions, maintain a continuous vasoconstrictor tone[7]. There is no parasympathetic antagonism — an increase in flow results from decreased sympathetic tone.

Within the microcirculation, blood tends to flow regularly only in the metarterioles between the arterioles and venules, hence the name 'throughfare vessels.'

Direct observation of the microcirculation by microscope has revealed that there is an intermittent ebb and flow through the capillary network controlled by the opening and closing of the pre-capillary sphincters — a phenomenon known as 'active vasomotion'[8]. The pre-capillary sphincters determine flow independent of the action of the arterioles and are controlled by the release of vasodilator substance and/or oxygen demand[9].

It is thought that the intermittent arrangement of blood flow means that much of the exchange and equilibrium between tissue fluids and blood takes place when blood flow is stopped since capillaries remain closed for 60 to 95% of the time[6,8].

An interplay of oncotic and hydrostatic pressures of plasma and interstitial fluid determine capillary permeability and reabsorption as well as directly affecting the use of lymph vessels in removing proteins, large waste particles and excess fluid. Difficulties occur in the determination of capillary (hydrostatic) pressure since measurement renders the vessel abnormal[7]. Values adopted are from the work of Landis, who developed a microinjection method for determining blood pressure in single capillaries, and reported average pressures at the arterial limb as 32mmHg and the venous limb as 12mmHg[10].

Blood components (mainly water and solutes) filter from the capillaries into the interstitial space of the tissue at the arterial end and return all but 10% at the venous end.

The fragile nature of the structure of the capillary walls and the capillaries' intravascular pressure render them particularly vulnerable to occlusion and/or damage by external loads. Pressure sores develop mainly as a result of disruption to the vascular network; the next section details the pathophysiological processes involved.

### Pathophysiology of pressure sore development

Pressure sores develop as a result of two processes — occlusion of blood vessels by external pressure and endothelial damage of arterioles and the microcirculation due to the application of disruptive and shearing forces[3]. The two processes, which are often concurrent, initiate a series of pathophysiological events which may or may not result in tissue damage and the appearance of a pressure sore.

Occlusion of blood vessels results in anoxia and a build up of metabolites. Release of pressure produces a large and sudden increase in blood flow as the anoxia and metabolites act upon pre-capillary sphincters and metarterioles. The increase in blood flow may reach 30 times its resting value and the bright red flush so produced is known as reactive hyperaemia[6].

The hyperaemic reaction is proportional to the duration of the occlusion and generally lasts half to three-quarters of the occlusion time[11]. If the lymphatic vessels of the dependent tissue are intact, and excess interstitial fluid resulting from the acute rise in capillary flow is removed, then permanent tissue changes will not progress[12].

Tissue changes do progress, however, when occlusion is prolonged and external load causes damage to lymphatic vessels and/or significant squeeze out of interstitial

fluid (Fig 2). Squeeze out of interstitial fluid is important for two reasons. First, if sufficient volume leaves the interstitial space, cell-to-cell contact can occur, resulting in cell membrane rupture and the release of toxic intracellular materials.

Second, on removal of the external pressure the sudden reduction in interstitial fluid pressure results in capillary bursting and interstitial flooding. If lymphatic vessels have been damaged by prolonged pressure and anoxia then the toxic intracellular materials and excess fluid remain in the area and necrosis ensues[2].

Evidence of this first stage of skin necrosis is non-blanching reactive hyperaemia, swelling, induration or loss of the epidermis by blistering or ulceration. The interstitial oedema interferes with metabolite exchange, causes distortion and thickening of tissues compressed between bone and the support surface and further increases the vulnerability of the skin[4]. Progressive loss of tissue occurs if the application of pressure is not relieved, and the wound will extend inward.

Endothelial damage of arterioles and the microcirculation occurs as a result of the application of disruptive and shearing forces to the skin and subcutaneous tissues on areas of the body not normally exposed to such forces[3].

Distortion of the blood vessels disrupts endothelial cells and activates intrinsic clotting mechanisms. Platelets aggregate and occlude the affected vessels causing ischaemic necrosis of dependent tissues. The epidermis may remain intact for a number of days before it sloughs off to reveal the tissue damage beneath[3].

Such a pressure sore would be classified as a stage 3, 4 or 5, depending on the initial tissue layer affected and the progression allowed before exposure. Particularly vulnerable to this type of damage are arterioles and the microcirculation of the subcutaneous layer.

Pressure sores have been classified by a number of authors and based upon the cause[3] and macroscopic appearance[13]. They were most comprehensively detailed by Torrance[4] who described the clinical appearance of pressure sores in five developmental stages (superseding the less specific grade 1 to 4 classifications initially proposed in 1977[14]).

Torrance described the five developmental stages as: blanching hyperaemia; non-blanching hyperaemia; progression through the dermis; progression into underlying subcutaneous fat and infective necrosis penetrating the deep fascia[4] (Table 1).

The description incorporated pathophysiological complications associated with each stage, leading to further tissue damage, and the clinical appearance of sores at each developmental stage.

An important aspect of this developmental classification is that it allows both the underlying pathophysiological process to be determined as well as the extent of the tissue damage present.

The reason why some patients develop pressure sores while other do not, despite exposure to similar circumstances, requires further review of the literature.

Critical determinants of pressure sore development have been described as being the intensity and duration of pressure and the tolerance of the skin and its supporting structures to pressure[15] — both are inextricably linked. The following section explores these aspects in more detail.

### The intensity and duration of pressure

Research relating to the intensity and duration of pressure are broadly divided into studies concerned with capillary pressure, the application of uniform pressure and the application of localised pressure. Key references are discussed below and highlight the individual nature of the response to external pressure owing to variations in the tolerance of the skin and provide evidence that the nature of the applied force will have great bearing upon outcome.

The capillaries have little resistance to direct pressure, and great emphasis has been placed on the establishment of external pressure threshold levels[16]. It is widely quoted that if external pressure is greater than mean capillary pressure (of 32mmHg) then capillary occlusion occurs and damage ensues[3,17]. Great reliance has been placed upon this hypothesis and has governed the development of pressure sore prevention equipment and policies. The hypothesis does, however, have major pitfalls.

First, it does not account for the protective function of collagen. Attention to this important structure has developed following observations which revealed that the collagen content of the dermis is reduced following spinal cord injury[18] treatment with steroids for rheumatoid arthritis[19] and age-related triphasic changes[20].

It appears that collagen prevents disruption to the microcirculation by buffering the interstitial fluid from external load, thereby maintaining the balance of hydrostatic and oncotic pressures. A model of the aetiological events that probably occur when the collagen content of the skin is reduced has been developed (Fig 2). It is known that, as collagen is removed from tissue, a larger fraction of an externally applied load is transmitted to interstitial fluid, which leaves the pressurised area and, if sufficient, allows cell-to-cell contact and capillary bursting[21].

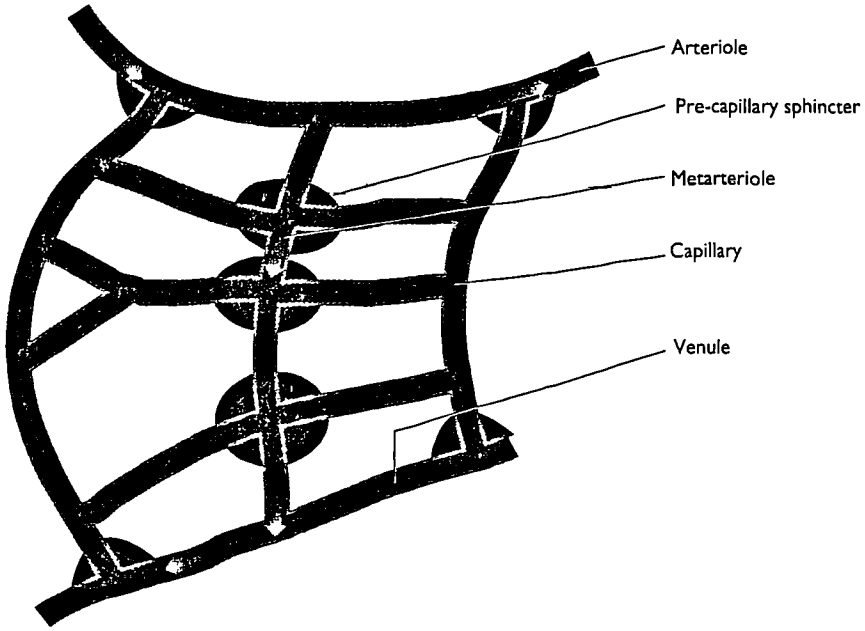
The key role played by collagen in pressure sore aetiology is a relatively recent discovery but the theory does interrelate with other known variables, such as the increased pressure sore risk with advancing age, as noted by epidemiological study[14], and may also explain why patients exposed to similar conditions — such as prolonged immobility — have differing outcomes[22].

Krouskop's model also provides a framework which interrelates many other predisposing factors such as diet,

**Table 1. Developmental classification of pressure sores (Torrance)4**

| | |
|---|---|
| **Stage 1.** | Blanching hyperaemia: reactive hyperaemia blanches on light finger pressure, indicating that the microcirculation is intact |
| **Stage 2.** | Non-blanching hyperaemia: erythema does not blanch upon light finger pressure and local oedema, induration, blistering or superficial ulceration may be present |
| **Stage 3.** | Ulceration progresses through the dermis to the interface with the subcutaneous tissue. Distinct ulcer edges are apparent though surrounded by erythema |
| **Stage 4.** | Ulceration extends into the subcutaneous fat and tissue necrosis is compounded by thrombosis and infection of the fatty tissue. The deep fascia temporarily arrests downward progression but results in lateral progression with a distinct ulcer margin. The deeper areas of the sore are characterised by inflammation, fibrosis and retraction |
| **Stage 5.** | Infective necrosis penetrates the deep fascia. Muscle destruction occurs rapidly and bone and body cavities can become involved |

**Fig 1. The microcirculation[6,7]**




physiological and psychological stress, steroid administration, poor oxygen saturation, lymphatic drainage and interstitial flow[2].

Examination of the role of collagen challenges the commonly held belief that if external pressure exceeds the internal mean capillary pressure of 32mmHg then damage ensues. Evidence indicates that the collagen content of the dermis, which alters with disease and/or age, will affect the capacity of the dermis to buffer external pressure and so the threshold pressure will vary from individual to individual.

Another factor that challenges the hypothesis is that when external pressure is applied to the skin an autoregulation process allows internal capillary pressures to rise correspondingly.

It has been shown that within one minute from the time of external pressure application of 60mmHg, a rise in the capillary pressure occurs and stabilises at approximately 10mmHg higher than the external pressure[10]. Other experiments revealed that the application of an external pressure of 60mmHg did not inhibit blood flow in healthy subjects[23,24].

It appears that this autoregulation process breaks down only in those with normal circulation when external pressure exceeds diastolic pressure[25], indicating that the use of 32mmHg is conservative. Conversely, in patients with increased susceptibility, such as elderly or severely ill people where the autoregulatory mechanism is not apparent, occlusion has been reported when pressures of less than 20mmHg are applied[24] indicating that the use of 32mmHg is again inappropriate.

A review of the literature with specific reference to the application of external load to capillaries provides no simple load-response equation. The response of the skin's capillary network to external load is determined by the collagen content of the dermis and the autoregulatory mechanism, allowing internal pressure to rise. It is apparent that there are wide variations in individual capacity to resist pressure and the use of 32mmHg as a universal threshold is inappropriate.

The situation is further complicated by variations in the manner in which a given load is applied. It has become apparent that an external pressure applied in a uniform or enveloping manner has little if any long-term effect on tissue. For example, a deep-sea diver may be subject to extreme (but even) external pressure without suffering tissue damage. Similarly, a limb deprived of its blood supply by the application of a tourniquet will not develop a 'pressure sore' as a consequence. This was first observed and discussed by Husain, a pioneer in the field of the biochemical aspects of pressure sore formation, who experimented with rats[26]. A tourniquet applied to rats' tails produced no permanent changes with the exception of those exposed to 800mmHg for six hours, and the author emphasised the need to distinguish between evenly distributed pressure and localised or point pressure.

Further evidence of this was reported in studies of the microcirculation of human skin; following controlled occlusion of blood flow for up to three hours, circulation was re-established with few signs of damage. Even when occlusion was maintained for seven hours it was observed that the 'majority' of the microcirculation was re-established and maintained[27].

It is the effect of the application of a local or point pressure upon the skin which is of interest in pressure sore aetiology. Present knowledge stems largely from animal testing and actual values vary enormously owing to the differing animals, tissues and methods used. Despite wide variations in threshold values, general trends emerge.

It is widely quoted that prolonged low pressure is as hazardous as short-term high pressure[15, 28] and that an inverse relationship exists between the amount and duration of pressure[29]. However, a closer look at the experiments undertaken reveals that, although these statements can be supported, inappropriate conclusions and oversights in the interpretation of results have been made.

It is important in a review of the literature to differentiate between studies which examine the pressure/time and extent of tissue damage relationship and those which examine the simpler pressure/time ulcer/no ulcer relationship. It

is authors of the former type of study who have failed to report clearly the clinical significance of their results.

Husain[26], for example, while contributing to the overall body of knowledge in respect of the pathophysiology of pressure sores and the importance in distinguishing between uniform and local pressure, makes serious errors in the interpretation of data relating to local pressure application. A summary of his reported findings are detailed in Table 2 and from these results Husain concluded that low pressures maintained for long periods seem to induce more tissue damage than high pressures for short periods.

However, the most interesting aspect of the results was that the low and high pressures over a similar time span (of one to two and six hours) produced similar tissue changes; this was completely overlooked by the author despite similar findings by Brooks and Duncan[30], who concluded that the duration of pressure application was of greater importance than the degree of pressure.

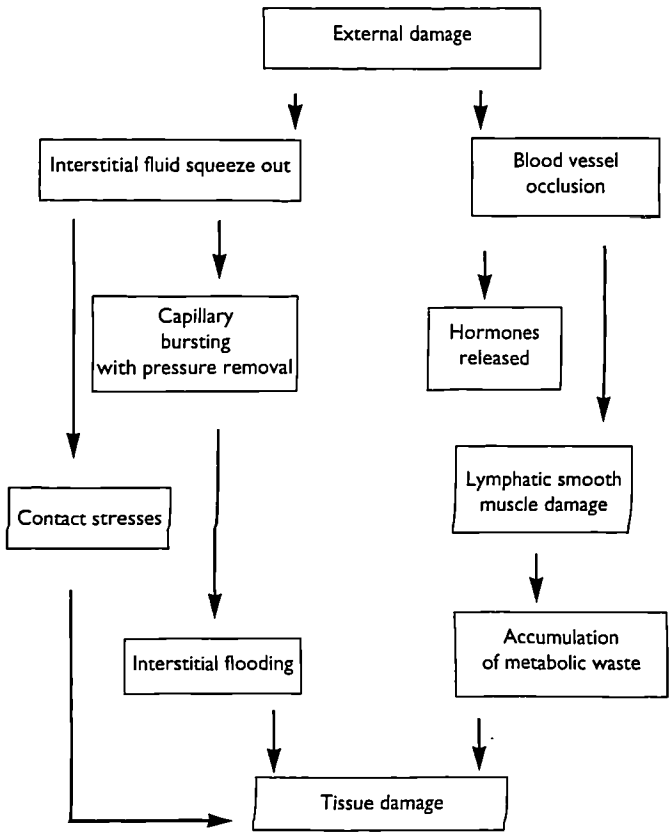
A similar omission was made by Kosiak who applied pressures ranging from 35 to 240mmHg to the muscle of rats for periods of one, two, three and four hours and examined the tissue microscopically[8]. Results indicated that once above a critical pressure (35mmHg), and critical time value (one hour), as the time of applied pressure increased so did tissue damage. The extent of the tissue damage was the same regardless of the pressure applied. These findings were not highlighted by the author or discussed in any way.

Therefore an important though essentially unrecognised finding of the studies examining the pressure/time extent of tissue damage relationship is that once a critical pressure threshold value and critical time value is exceeded then tissue damage will proceed at a similar rate regardless of the magnitude of the pressure applied.

Studies which examined the pressure/time ulcer/no ulcer relationship all reported an inverse relationship between the amount and duration of pressure, that is, low pressure for long periods and high pressures for short periods both cause ulceration[29,31,32].

Direct application of the results in terms of threshold values is limited since the studies used differing techniques and animal tissues and reported results varied. The most important aspect of the results is that they highlight the need to consider

**Fig 2. Integrated model of tissue damage (Krouskop)[2]**




pressures of any value and time periods of any duration.

Despite the limitations of the results, a pressure/time curve was developed basing the lower threshold value on the mean capillary pressure of 32mmHg[33]. (Fig 3).

Its use is no longer supported by the evidence, which highlights the individual nature of pressure/load response and disregards the use of mean capillary pressure as a threshold value.

Furthermore, a re-examination of the working assumptions of the early researchers has revealed that shear forces are involved, which complicate the pressure/time/tissue damage equation and

**Table 2 Tabulated results of Husain[26]**

| Pressure intensity (mmHg) | Pressure duration (hrs) | Tissue changes |
|---|---|---|
| 100 | 2 | Patchy congestion |
| 100 | 6 | Severe changes |
| 600 | 1 | Patchy congestion |
| 600 | 6 | Severe changes |

are likely to account for the wide variations in the results and alter the threshold values of the parabolic intensity duration curve[16,24,34].

The differences in pressure and shear forces were defined by Bennett and Lee Pressure consists of the load perpendicular to the tissue's surface and shear the load parallel to the tissue' surface[24]. It is difficult to create pressure without shear and shear without pressure

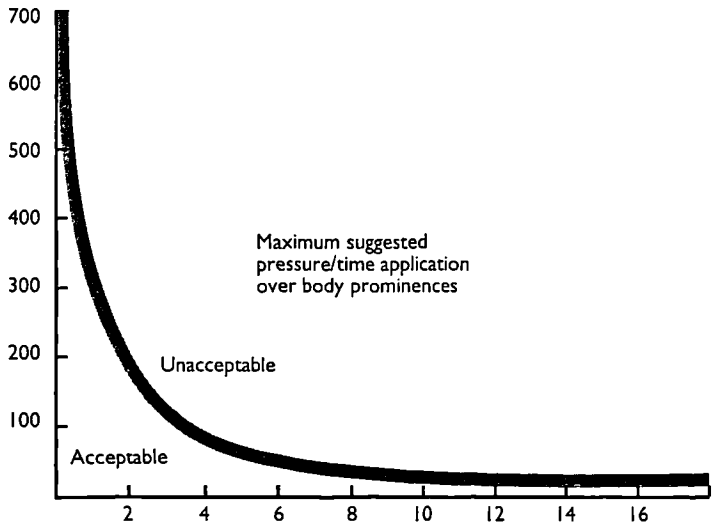
The effect of varying the amount o shear on human skin has been reported[24] Using a sensor head incorporating fou sensors (two pressure, one shear and one blood flow plethysmograph) researcher were able to determine the relationship between pressure and shear in producing blood flow occlusion. Using the palm o the hand of four healthy subjects, the authors found low shear caused occlusion within the 100 to 120mmHg pressure range and high shear in the 60 to 80mmHg pressure range. They concluded that the primary force generating mechanical occlusion is pressure but that shear plays an important contributary role and its presence cannot be ignored[24].

In relation to pressure sore aetiology the authors also reported other interesting data[24]. Using the same sensor head, they measured pressure and shear forces generated at the interface between a hard wheelchair seat and the ischial tuberosities of various subjects. They reported that elderly people and those with paraplegia had reduced blood flow and experienced greater shear than normal subjects at the same pressure values, providing further evidence of the individual nature of the load response relationship.

This review of the literature examining the pressure/time, ulcer/no ulcer relationship, has highlighted the individual nature of the skin and underlying tissues to pressure and emphasised the need to consider pressures of any value and time periods of any duration.

The review highlights the individual nature of the skin's response to pressure and that the problem of tissue breakdown is a multidimensional process. It is increasingly apparent that individual factors determining the tolerance of the skin to pressure affect the load response relationship. The variables involved are discussed in the following section.

**Fig 3 Pressure/time curve**




### Tolerance of the skin

Factors affecting tissue tolerance can be subdivided into those that are extrinsic and those that are intrinsic. Extrinsic factors affect tissue tolerance by impinging upon the surface of the skin and include exposure to friction, irritants and moisture. Intrinsic factors affect the ability of the skin and supporting structures to respond to pressure and shear forces by influencing the sensation/perception/ response mechanism and/or altering the structural constituents and perfusion of tissues.

Extrinsic factors have received little research interest and their relationship with pressure sore aetiology is not clear. The contribution of moisture, for example, is linked to pressure sore development in numerous accounts, with particular reference to incontinence[14,15,35,36].

However, moisture, whether in the form of urine, perspiration or wound drainage, does not in itself cause pressure sores[4]. It has been suggested that it enhances the frictional component of shearing force[4] or combines with by-products of laundering processes and incites chemical attack on the skin[37].

Other characteristics associated with patients who suffer incontinence, such as those of advancing age and reduced mobility may be the link between high pressure sore occurrence and incontinence[4].

Despite this unclear relationship, incontinence and/or skin moisture is included in the various risk-assessment scales developed in the past 30 years[15,35,36] which does reflect the importance attributed to this factor on the basis of epidemiological study and clinical observation.

Similarly, the role of skin irritants such as starch, altered pH by excessive use of soap and detergent residues in hospital sheets is not clearly determined. A link has been established since at least the 1960s[38], with most references made to the dangers of excessive use of soap; it appears that surface lipids and sebum removed by soap allows dehydration, exposes the skin to water-soluble irritants and bacteria and increases frictional forces[38,39]. These factors then reduce the tolerance of the skin to pressure.

Other accounts refer to the effects of detergent and enzyme residues in linen which can cause skin rashes without the compounding problem of pressure[37,40]. It may be that the combination of plastic under sheets and skin dampness simulates the closed patch test technique used by dermatologists and so potentiates the irritant material present. The clinical significance of this is not yet clear[37].

The evidence that friction increases the susceptibility of the skin to pressure ulceration was provided by experimentation on pigs[41]. This compared the application of 'pressure only' with 'pressure plus friction', applied to tissues covering the iliac spines of paraplegic and normal pigs. In both instances, more ulcers developed on those exposed to pressure plus friction, with particularly startling results among the normal pigs. Pressure alone required a level of 290mmHg to produce ulceration, whereas pressure with friction produced ulcers at 45mmHg.

A further experiment using an isotope clearance technique established that friction did not produce ulcers by an ischaemic mechanism involving the generation of shear[41]. This reinforced results of a previous study whereby tissue was examined by electron microscopy and disruption to the avascular epidermis by the mechanical forces generated by friction was observed[32].

To date no further study has validated this work, although its importance is being increasingly recognised and is included in the risk assessment tool developed by Braden and Bergstrom[15].

An interaction model involving all the extrinsic factors impinging on the skin's surface and reducing tolerance to pressure has been developed (Fig 4)[37].

A criticism of the model is that it does not link moisture to friction but on the whole it provides a comprehensive yet simple picture of the likely processes involved.

A review of the literature relating to the the effect of extrinsic factors on the skin has revealed that aspects including skin moisture, irritants and friction are interrelated; they cannot in themselves cause pressure sores but appear to potentiate the damaging effects of pressure. Skin

moisture, incontinence and friction are viewed as being particularly important in the aetiological process by clinical experts and are included as risk factors on risk assessment scales. However, the exact nature of their relationship to pressure sore development is not clearly defined.

Numerous intrinsic factors affect the ability of the skin and support structures to respond to pressure and shear forces. For the purpose of this review they are classified as factors affecting the collagen component of the skin and tissue perfusion.

The content of collagen in the dermis is determined by a number of factors including age, steroid administration and availability of nutrients as well as spinal cord injury. These factors affect the synthesis, maturation and degradation of the connective tissue

Age-related changes in the collagen content of the skin are particularly interesting. It has been shown that the total collagen content of the skin of 'normal' subjects falls at a steady rate over the age range of 30–80 years[19]. It appears that such changes occur as a result of a gradual reduction in the synthesis of collagen from 20 to 60 years with a dramatic degradation of collagen in the 60-plus age group[20].

These changes have direct application to pressure risk assessment and interrelate with results from epidemiological studies indicating increased risk of pressure sore development with increasing age [14,36].

It has been shown that the administration of steroids mimics and exacerbates the ageing process and leads to a reduction in the collagen content of the skin. Whether reduced synthesis, instability or increased degradation of collagen is the main cause is unknown, but it has been observed that withdrawal results in a reversal of the changes[19].

The effect of nutritional state upon the collagen content of the skin is not documented in the literature. Much research has been undertaken showing that protein, carbohydrates, fats, vitamins and trace elements are necessary for the synthesis and maturation of collagen in wound healing[42,43,44], but the effect of an absence of essential nutrients on the total collagen content of the skin has received scant attention.

Nutritional research has concentrated on nutrient profiles of patients with existing pressure sores and revealed deficiencies in albumin, vitamin C and zinc[45]. However, values are often similar

**Fig 4. Extrinsic factors: an interaction model[37]**

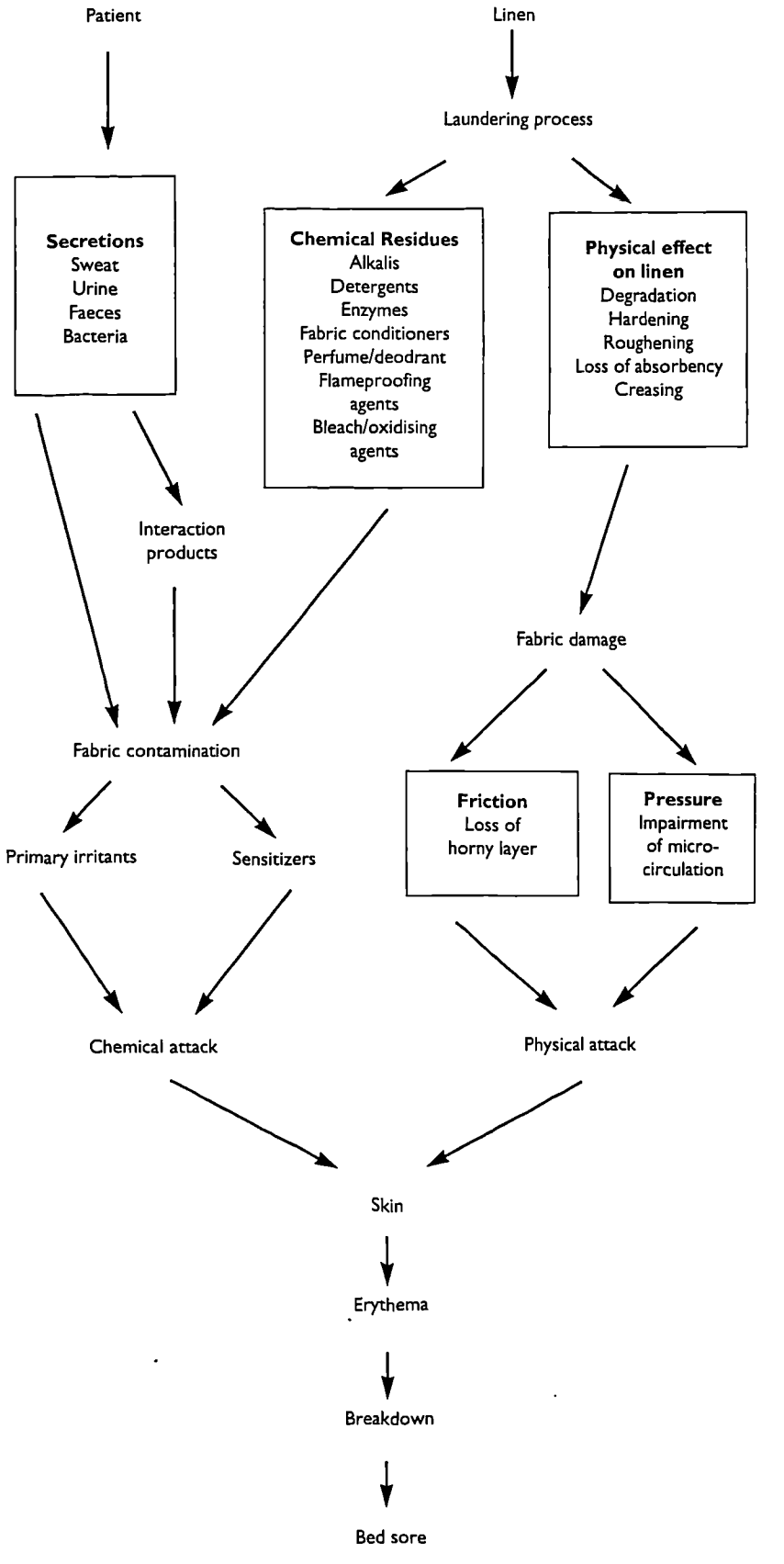
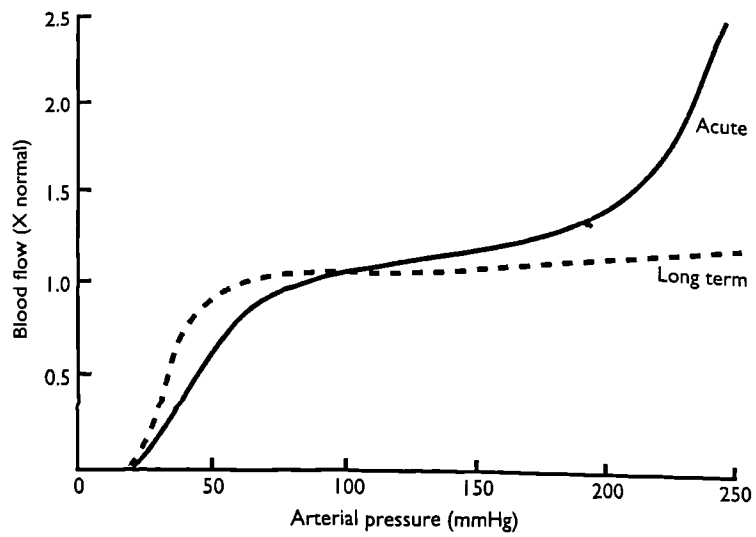


**Fig 5. Arterial blood flow through a muscle[9]**




to deficiencies observed in pressure sore-free hospitalised elderly patients[46, 47], and results are inconclusive.

It may be hypothesised that, if essential nutrients are required for collagen synthesis and stability during healing, the general metabolism of collagen, which undergoes a continual process of synthesis, maturation and degradation, will also be affected by nutrient deficiencies. Investigation of this, and possible implications for pressure sore risk assessment, is necessary.

In respect of the effect of changes in the spinal cord-injured patient, Claus-Walker observed an associated breakdown of collagen by examining excretion of electrolytes in urine[18]. Indeed, this observation was the basis of the theory later developed by Krouskop that collagen was a likely key factor in pressure sore development[2].

The fact that collagen degradation occurs following injury interrelates with a later study which determined that people with paraplegia had a reduced resistance to external load when compared to normal subjects[24] and links with epidemiological data suggesting spinal cord-injured patients have an increased pressure sore risk[48].

Tissue perfusion is affected by a number of intrinsic factors: systemic blood pressure, serum protein, lymphatic drainage, body temperature, smoking and serum haemoglobin as well as factors which potentiate endothelial cell damage and increase platelet thrombosis.

Three studies have reported that the main systolic blood pressure (sBP) of pressure sore positive groups is lower than that of pressure sore negative groups[1,49,50] and have suggested that reduced systolic blood pressure results in reduced tissue pressures of less than 60mmHg and pressure sore development[51].

However, differences in recorded systolic blood pressures are not wide, and overlap — with values of 120± 21mmHg and 130± 17mmHg sBP for pressure sore positive patients and 132± 32mmHg and 140± 20mmHg for pressure sore negative patients[1,50]. In view of problems of reliability associated with blood pressure recordings and the overlap of recorded blood pressure values, the clinical application of these results is severely limited. Furthermore, the suggestion that a lower systolic blood pressure (of the magnitudes reported) results in reduced tissue perfusion contradicts research by physiologists relating to the control of blood flow through the microcirculation.

An important characteristic of the circulation is the ability of each tissue to control its own capillary blood flow in proportion to its own need[9] and it is hypothesised that capillary pressure is controlled not by systemic blood pressure but by active vasomotion[52].

Local blood flow is controlled by short and long-term mechanisms. A sudden change in arterial pressure does lead to a surge or reduction in blood flow through a tissue, but in minutes an autoregulatory mechanism readjusts flow by approximately 25% of the previous level.

Over a period of hours/days/weeks a long-term regulatory mechanism is apparent, with control established by changes in the vascularity of the tissue. Changes in arterial pressure between 50 and 250mmHg have very little effect on the rate of capillary blood flow (Fig 5) which is determined in the main by the release of a vasodilator substance and/or oxygen demand[9].

The 'critical closing pressure' is the pressure within a vessel at which it collapses completely and blood flow ceases[7]. It is determined by an interplay of forces between intravascular pressure, muscle contraction and elastic forces of the blood vessel wall and externally applied pressure. In the skin and subcutaneous tissues the interplay of forces is further complicated by the presence of shear forces[24].

The fact that four variables are involved explains why no individual response is the same, although trends are apparent. The effect of severe hypotension resulting in a prolonged period of low intravascular pressure easily fits the equation that an average lower mean systolic blood pressure is found among pressure sore-positive patients but the fact that patients with high systolic blood pressure also develop pressure sores also fits the equation if a high external load is applied and/or blood vessel walls are weak and/or shear forces are present, and so on.

There is, then, no simple relationship between systemic blood pressure tissue perfusion and pressure sore development. A number of variables are involved in determining capillary pressure, capillary flow and blood supply to a given tissue and this explains why general trends may be apparent in pressure sore incidence, but direct application to practice is limited owing to the many individual variables.

The other intrinsic factors affecting tissue perfusion further complicate the picture. Pathology which alters the oxygen exchange/demand/supply at tissue level increases the vulnerability of the skin and underlying structures to damage from external load.

The relationships between the factors affecting oxygen exchange/demand/supply and pressure sore development have not been adequately tested and theories are hypothetical[15]. However, their potential to exacerbate other pathophysiological processes justifies a brief overview.

There is evidence that low-serum protein concentrations (particularly hypoalbuminaemia) are associated with pressure sores[1,53] although other studies

do not demonstrate the link[47,51].

Decreased serum protein may affect the filtration and absorption forces at capillary level, resulting in interstitial oedema which interferes with interstitial nutrient exchange and increases the vulnerability of dependent structures to damage[1].

Similarly, other factors which lead to an increase in interstitial fluid, such as impaired lymphatic drainage, are also likely to increase the vulnerability of the skin and underlying tissues to pressure damage by altering the nutrient exchange and exacerbating tissue hypoxia[2].

In respect of oxygen demand, changes in skin and body temperature are thought to alter tissue susceptibility to ischaemic injury[54]. An increase in skin temperature of 1°C causes a 10% increase in tissue metabolism, and it is suggested that the increase in nutrient demand exacerbates other pathophysiological factors causing pressure sore development.

Reduction in the oxygen-carrying capabilities of the blood are also linked to pressure sore development. Decreased haemoglobin levels have been associated with pressure sore occurrence[1,52], although differences between pressure sore positive and pressure sore negative subjects were not significant.

Cigarette smoking has been positively correlated to the presence of sores in a study of spinal cord-injured patients[15].

The presence of factors which potentiate endothelial cell damage and thrombosis and curtail the nutrient supply completely also require consideration. A number of potentiating factors include (among others) endotoxins, metabolic acidosis, dehydration, burns, thromboplastins (released during surgery), bacteraemia, hypoxia and blood stasis[3]. However, the exact nature of the relationship between these factors and pressure sore development is unknown.

A review of the factors affecting the tolerance of the skin and supporting structures to pressure and shear forces underlines the complex nature of the physiological processes involved in pressure sore aetiology. The exact contribution of intrinsic and extrinsic factors to pressure sore development is largely undetermined, and research provides contradictory results and/or a limited number of studies which require validation by further exploration.

## Conclusion

In this review of the aetiology of pressure sores the anatomy and physiology of the skin and underlying structures have been described and details of this complicated pathophysiological processess discussed.

The review has highlighted the individual nature of the skin's response to pressure and emphasised the fact that the problem of tissue breakdown is a multidimensional process. ■

REFERENCES


1 Cullum, N., Clark, M. Intrinsic factors associated with pressure sores in elderly people. *J Adv Nurs* 1992; **17:** 4, 427–431.

2 Krouskop, M. A synthesis of the factors which contribute to pressure sore formation. *Med Hypothesis* 1983; **11:** 255–267.

3 Barton, A., Barton, M. *The Management and Prevention of Pressure Sores.* London: Faber, 1981.

4 Torrance, C. *Pressure Sores: Aetiology, treatment and prevention.* London: Croom Helm, 1983.

5 Hall, D. A., *The Biomedical Basis of Gerentology.* Guildford: John Wright, 1984.

6 Lamb, J. F., Ingram, C.G., Johnson, T.A., Pitman, R.M. *Essentials of Physiology.* London: Blackwell Scientific, 1980.

7 Lippold, O.C.J., Winton, F.R. *Human Physiology* (7th edn). Edinburgh: Churchill Livingstone, 1979.

8 Kosiak, M. Etiology of decubitus ulcers. *Arch Phys Med Rehab* 1961; **42:** 19–29.

9 Guyton, A.C. *Human Physiology and Mechanisms of Disease.* London: W.B. Saunders, 1992.

10 Landis, E.M. Microcirculation studies of capillary blood pressure in human skin. *Heart* 1930; 15: 209–228.

11 Lewis, T., Grant, R. Observations upon reactive hyperaemia in man. *Heart* 1925; **12:** 73–120.

12 Krouskop, T.A., Reddy, N.P., Spencer, W.A., Secor, J.W. Mechanisms of decubitus ulcer formation: a hypothesis. *Med Hypothesis* 1978; **4:** 1, 37–39.

13 Forrest, R.D. The treatment of pressure sores. *J Int Med Res* 1980; **8:** 430–435.

14 Barbanel, J.C., Jordan, M.M., Nicol, S. M., Clark M.O. Incidence of pressure sores in the Greater Glasgow Health Board Area. *Lancet* 1977; 2: 548–550.

15 Braden, B.J., Bergstrom, N. A conceptual schema for the study of the etiology of pressure sores. *Rehab Nurs* 1987; **12:** 1, 8–16.

16 Bennett, L., Lee, B.Y. Vertical shear existence in animal pressure threshold experiments. *Decubitus* 1988; **1:** 1, 18–34.

17 Daniel, R.K., Priest, D.L., Wheatley, D.C. Etiologic factors in pressure sores: an experimental model. *Arch Phys Med Rehab* 1981; **62:** 492–498.

18 Claus-Walker, J., Campos, R.J., Carter, R.E., Chapman, M. Electrolytes in urinary calculi and urine of patients with spinal cord injuries. *Arch Phys Med Rehab* 1973; **54:** 109–114.

19 Hall, D.A., Reed, F.B., Nuki, G et al. The relative effects of age and corticosteroid therapy on the collagen profiles of dermis from subjects with rheumatoid arthritis. *Age Ageing* 1974; **3:** 15.

20 Hall, D.A., Blackett, A.D., Zajac, A.R. *et al.* Changes in skin fold thickness with increasing age. *Age Ageing* 1981; **10:** 19.

21 Reddy, N.P., Krouskop, T.A., Newall, P.H. Biomechanics of a lymphatic vessel. *Blood Vessels* 1975; 12: 261–278.

22 Barbanel, J.C., Jordan, M.M., Nicol, S.M. Major pressure of sores. *Health Soc Serv J* 1980; **90:** 2, 4715, 1344–1345.

23 Collins, G.M., Ludbrook, J. Behaviour of vascular beds in the human upper limb at low perfusion pressure. *Circ Res* 1967 **21:** 319–325.

24 Bennett, L., Lee, B.Y. Pressure verses shear in pressure sore causation. In: Lee, B.Y. *Chronic Ulcers of the Skin.* New York: McGraw Hill, 1985.

25 Holstein, P., Neilson, P.E., Barras, J.R. Blood flow cessation at external pressure in the skin of normal limbs. *Microvas Res* 1979; **17:** 71–79.

26 Husain, T. An experimental study of some pressure effects on tissues with reference to the bed sore problem. *J Path Bact* 1953; **66:** 347–358.

27 Braenmark, P.I. Microvascular function at reduced flow rates. *In:* Kenedi, R.M., Cowden, J.M., Scales, J.T. *(eds). Bedsore Biomechanics.* Maryland: University Park Press, 1976.

28 Morison, M. Early assessment of pressure sore risk. *Prof Nurse* 1989; **4:** 9, 428–431.

29 Kosiak, M. Etiology and pathology of ischaemic ulcers. *Arch Phys Med Rehab* 1959; **40:** 62–69.

30 Brooks, B., Duncan, G.W. Effects of pressure on tissues. *Arch Surg 1940;* **40:** 696–709.

31 Groth, K.E. Klinishe Beobachtungern und experimentelle studien uber die enstehumg des dekubitus. *Acta Chir Scand* 1942; **87:** (supp 76), 198.

32 Dinsdale, S.M. Decubitus ulcers in swine: light and electorn microscopic study of pathogenesis. *Arch Phys Med Rehab* 1973; **54:** 51–56.

33 Reswick, J.B., Rogers, J. Experience at Rancho Los Amigos Hospital with devices and techniques to prevent pressure sores. In: Kennedi, R.M., Cowden, J.M., Scales, J.T. (eds). *Bedsore Biomechanics.* Baltimore: University Park Press, 1976.

34 Gibson, T., Barbanel, J.C., Evans, J.H. Biomechanical *concepts and effects.* In: Kenedi, R.M., Cowden, J.M., Scales, J.T. (eds). *Bedsore Biomechanics.* Maryland: University Park Press, 1976.

35 Norton, D., McLaren, R. Exton-Smith, A.N. *An Investigation of Geriatric Nursing Problems in Hospital.* Hong Kong: National Corporation for the Care of Old People, 1962.

36 Waterlow, J. The Waterlow card for the prevention and management of pressure sores: towards a pocket policy. *Care Sci Prac* 1988; **6:** 1, 8–12.

37 Alberman, K. Is there any connection between laundering and the development of pressure sores? *J Tissue Viability* 1992; **2:** 2, 55–56.

38 Bettly, F.R. Some effects of soap on the skin. *Br Med J* 1960; **1:** 1675–1679.

39 Lowthian, P. Prevention of heel pressure sore (Letter). *Lancet* 1982; **1:** 8177, 1089.

40 Gunnell, D.J. Mysterious slapped face rash at holiday centre. *Br Med J* 1992; 304: 477–479.

41 Dinsdale, S.M. Decubitus ulcers: role of pressure and friction in causation. *Arch Phys Med Rehab* 1974; **55:** 147–152

42 Bergstrom, N., Braden, B., Krall, K., Brandt, G. Adequacy of descriptive scales for reporting diet intake in institutionalised elderly. *J Nutr Elderly* 1986; **6:** 1, 3–16.

43 Brown, K. The role of nutrition in pressure area care. *J Tissue Viability* 1991; **1:** 3, 63–64.

44 Keighley, J.K. Wound healing in malnourished patients. *AORN J* 1982; **35:** 6, 1094–1099.

45 Breslow, R. Nutritional status and dietary intake of patients with pressure ulcers: review of research literature 1943–1989. *Decubitus* 1991; **4:** 1, 16–21.

46 Morgan, A.G., Kelleher, J., Walker, E.E. et al. A nutritional survey in the elderly: blood and urine vitamin levels. *Int J Vit Nutr Res* 1975; **45:** 448–462.

47 Goode, H.F., Burns, E., Walker, E.E. Vitamin C depletion and pressure sores in elderly patients with femoral neck fracture. *Br Med J* 1992; **305:** 925–926.

48 Richardson, R.R., Meyer, P.R. Prevalence and incidence of pressure sores in acute spinal cord injuries. *Paraplegia* 1981; **19:** 235–247.

49 Mawson, A.R., Biundo, J.J., Nevill, P. et al. Risk factors for the early occurring pressure ulcers following spinal cord injury. *Am J Phys Med Rehab* 1988; **67:** 123–127.

50 Schubert, V. Hypotension as a risk factor for the development of pressure sores in elderly subjects. *J Tissue Viability* 1992, **2:** 1, 5–8.

51 Kemp, M.G., Kiethley, J.K., Smith, D.W., Morreale, B. Factors which contribute to pressure sores in surgical patients. *Res Nurs Health* 1990; **13:** 5, 293–301.

52 Nicol, P.A., Webb, R.L. Vascular patterns and active vasomotion as determinants of flow through minute vessels. *Angiology* 1955; **6:** 291.

53 Holmes, R., Macchiano, K., Jhangiani, S.S. et al. Combating pressure sores: nutritionally. *Am J Nurs* 1987; **87:** 10, 1301–1303.

54 Fisher, S., Szymke, T.E., Apte, S.Y., Kosiak, M. Wheelchair cushion effect on skin temperature. *Arch Phys Med Rehab* 1978; **59:** 68–72.

# Appendix 3
# Laser Doppler Images

# Appendix 3

## Laser Doppler Images

**Image 1 Skin area clinically assessed as Grade 0**

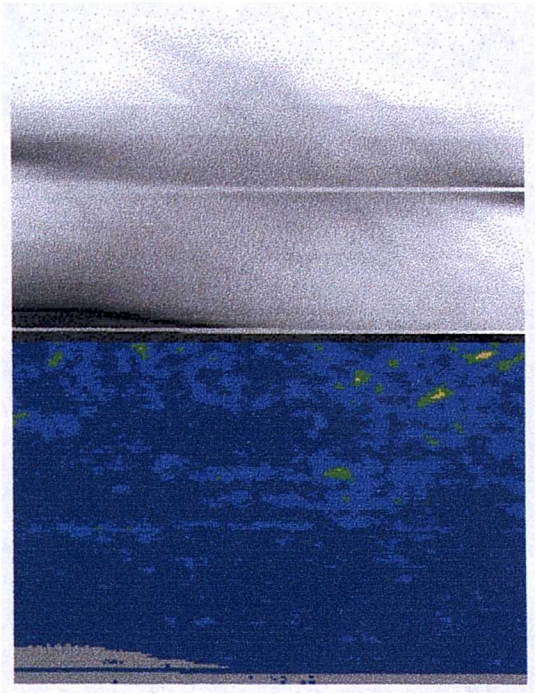


**Image 2 Skin area clinically assessed as Grade 0**

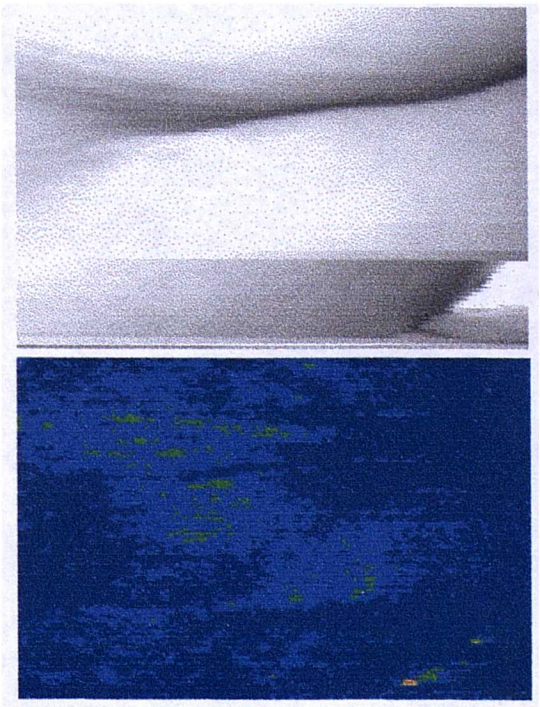


**Image 3 Skin area clinically assessed as Grade 1a**

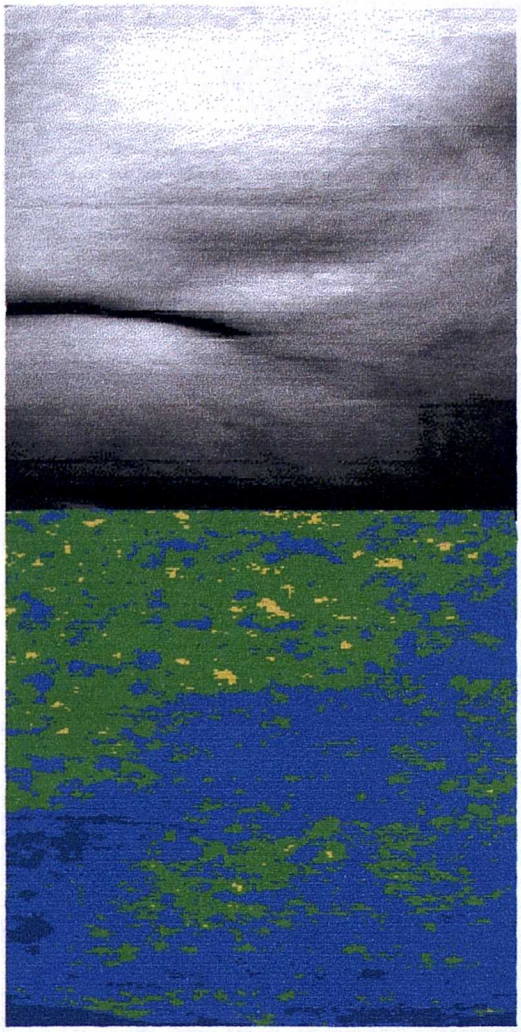


**Image 4 Skin area clinically assessed as Grade 1a**

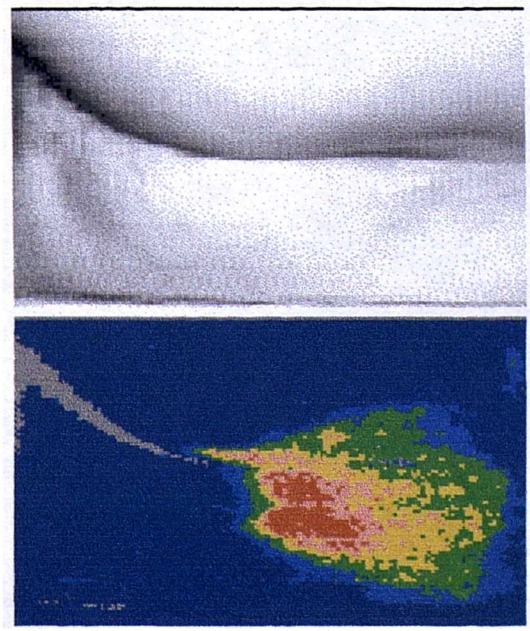
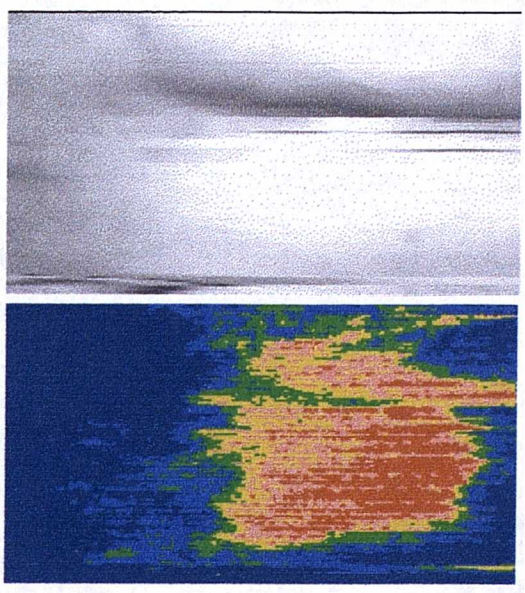


**Image 5 Skin area clinically assessed as Grade 1b**



**Colour Key in Perfusion Units**

# Appendix 4
# Skin Assessments Preceeding Pressure Sore Development

# Appendix 4

## Skin Assessments Preceding Pressure Sore Development

Skin assessment schedules for all skin sites with an existing pressure sore and preceding pressure sore development are detailed in Tables A4.1 to A4.6 using the clinical skin assessment scale defining Grades 0 to 5 (Table 7.1) and classification of Grade 1 skin changes (Table 7.2). Other data codes also include:

$^{i}$ indicating intra-operative pressure sore development

- indicating missing data

x indicating dressing insitu at time of assessment

**Table A4.1 Pressure Sore on First Assessment**

| Patient Number | Skin Site | Skin assessment Schedule | Destination |
|---|---|---|---|
| 1 | Heel | 4 x – x - - x x x – x - - x | Discharged |
| 2 | Sacrum | 2 – 2 2 – 2 2 2 2 - - - 2 2 1b 1b 1b 1b 1b 1b 1b | Discharged |
| 3 | Buttock | 2 2 2 2 2 – 2 2 2 2 - - 2 2 – 2 2 2 2 2 – 2 | Discharged |
| 4 | Heel | 2 2 2 2 – 2 2 2 2 x | Discharged |

**Table A4.2 Grade 0 Preceding Pressure Sore Development**

| Patient Number | Skin Site | Skin assessment Schedule | Destination |
|---|---|---|---|
| 5 | Sacrum | 0$^{i}$ 2 1a 1b 1a – 1a 1a 1a - - | Discharged |
| 6 | Heel | 0 – 2 2 x x x x - - x – x – x – x x x 5 4 4 – x | Discharged |
| 6 | Heel | 0 – 2 2 x x x x - - x – x – x – x x x 5 4 4 – x | Discharged |
| 7 | Sacrum | - - - 0 0 0 0 0 – 0 - - 0 - - - - 0 - - - - - - - 2 2 2 2 – 1b$^{+}$ - 2 2 2 – 2 2 | Discharged |

**Table A4.3 Grade1a/1b and Resolution to Grade 0 Preceding Pressure Sore Development**

| Patient Number | Skin Site | Skin assessment Schedule | Destination |
|---|---|---|---|
| 8 | Sacrum | 1b - - 1b 0 0 - - - - 0 2 2 2 - 2 - - 2 - - - - 2 - x - - - - - 2 x - x - - x 2 - 2 | Discharged |
| 9 | Sacrum | 1a 1a 1a 0 0 – 0 0 1a – 1a 1a 1a – 1a 1a 1a 1a - - 1a - - - 0 0 - - - 2 2 2 2 - - 1a | Discharged |
| 9 | Buttock | 1a 1a 1a 0 0 – 0 0 1b 0 0 1a 0 0 1a – 1a 1a 1a 1a 1a - - 1a - - - 0 0 - - - 2 2 1a 1a - - 1a | Discharged |

**Table A4.4 Grade 1a Preceding Pressure Sore Development**

| Patient Number | Skin Site | Skin assessment Schedule | Destination |
|---|---|---|---|
| 10 | Sacrum | 1a' 2 2 2 - - 1a 1a 1a – 1a | Discharged |
| 2 | Heel | 1a – 2 2 - - 2 2 2 - - - 1b 2 2 1b 2 2 x x x | Discharged |
| 11 | Sacrum | 0 1a - - 1a 1a 1a 2 2 – 1a – 1a 1a x x 1a | Discharged |
| 12 | Sacrum | 0 0 0 0 0 0 0 0 0 0 - - - 0 - - 1a - - 1a 1a 1a 1a 2 2 2 | Transferred |
| 12 | Buttock | 0 0 0 0 0 0 0 0 0 0 - - - 0 - - 1a - - 1a 1a 1a 1a 2 2 1b | Transferred |
| 12 | Buttock | 0 0 0 0 0 0 0 0 0 0 - - - 0 - - 1a - - 1a 1a 1a 1a 2 2 1b | Transferred |
| 12 | Heel | 0 1a 1a 1a 1a x 1a x 1a - - - x - - 1a 2 x x x x | Transferred |
| 7 | Buttock | - - - 0 1a 1a 0 0 - 0 - - 0 - - - - 1a - - - - - - - 2 2 2 2 - 2 - 2 1b 1b - 1b 1b | Discharged |
| 3 | Buttock | 0 0 0 0 1a 1a - 1a 1a 1a 1a - - 1a 1a - 1a 1a - 1a 2 2 1a 1a - 1a | Discharged |

**Table A4.5 Grade 1b Preceding Pressure Sore Development**

| Patient Number | Skin Site | Skin assessment Schedule | Destination |
|---|---|---|---|
| 13 | Sacrum | 1b' 2 – 2 – 2 2 2 2 2 2 – 2 2 2 – 1a 1a - - 1a 1a 1a 0 0 | Discharged |
| 13 | Buttock | 1b - 1b - 1b 1b 1b 1b 1b 1b - 1b 1b 1b - 1b 1b - - 1b 2 2 2 2 | Discharged |
| 14 | Sacrum | 0 1a - - 1a 1a 1b 1b 1b 0 1a – 1a 1a 1a 1a 1a 1a 1a – 1a 1a 1a 1b 2 - 2 2 - 2 2 2 2 2 2 2 2 2 | Died |
| 15 | Heel | 0 1a 1a 1a - - 1a 1a 1b 1b 1a 2 - - 2 – x – x – 2 2 x x x x x | Discharged |
| 6 | Sacrum | 0 - - - 0 0 0 0 - - 0 - 0 - 0 0 0 0 0 0 1b - - 1a - - - - - - 0 0 0 0 - 1a 1a 1a - 1a 1a - - - - - 1a - - - 1a - 1a - - 1b - 1b - 1b 2 | Discharged |
| 16 | Heel | 1b 2 2 2 - - - - 2 - - | Discharged |
| 16 | Heel | 1b 2 2 2 - - - - 2 - - | Discharged |
| 17 | Buttock | 1b 1b 1b 1b – 2 2 2 2 – 2 – 2 2 2 2 2 2 – 2 2 2 – 1b | Discharged |
| 4 | Heel | 1b 2 2 2 2 – 2 2 2 2 x | Discharged |

**Table A4.6 Grade $1b^+$ Preceding Pressure Sore Development**

| Patient Number | Skin Site | Skin assessment Schedule | Destination |
|---|---|---|---|
| 2 | Heel | $1b^+$ - $1b^+$ $1b^+$ - x $1b^+$ $1b^+$ $1b^+$ - - - $1b^+$ $1b^+$ 5 5 5 5 x x x | Discharged |
| 14 | Heel | 0 1a - - 1a 1a 1a 1a 1a 0 0 - 1a 1a 1b 1b 1b 1a 1a - 1a 1a 1a 1a 1b - 1b 1b - $1b^+$ 2 2 2 2 2 2 2 2 | Died |
| 18 | Heel | $1b^+$ $1b^+$ x 4 x - - x - x - x | Discharged |
| 7 | Buttock | - - - 0 1a 1a 1a 1a - 0 - - 0 - - - - 1a - - - - - - - 1b 1b 1b 1b - $1b^+$ - 1b 2 2 - 2 2 | Discharged |
| 19 | Heel | 1a - - - - 1a 1a 1a 1a 1a – 1b 1a 1a 1a - 1b - $1b^+$ - $1b^+$ $1b^+$ - $1b^+$ - - $1b^+$ - 2 1b 1a - - 1a 1a 1a - 1a - 1a 1b | Discharged |

# Appendix 5
# Prognostic Factor Analysis

# Appendix 5

# Prognostic Factor Analysis

## 5.1 Univariate Analysis

### 5.1.1 Logistic linear regression

| Variable | Odds Ratio | 95% CI | p | Number of patients |
|---|---|---|---|---|
| age | 1.05 | 0.98, 1.12 | 0.182 | 97 |
| typesurg general | 2.52 | 0.51, 12.50 | 0.259 | 97 |
| orthopaedic | 3.02 | 0.73, 12.44 | 0.125 | |
| A/E surg acute | 2.82 | 0.89, 9.00 | 0.079 | 97 |
| wounds | 0.34 | 0.09, 1.30 | 0.113 | 97 |
| diabetic | 0.68 | 0.17, 2.80 | 0.599 | 97 |
| BMI | 0.94 | 0.83, 1.08 | 0.427 | 92 |
| weight loss | 0.26 | 0.07, 0.98 | 0.047 | 84 |
| Pre-op temp | 1.62 | 0.46, 5.77 | 0.451 | 96 |
| pre-op Hb | 0.60 | 0.39, 0.91 | 0.016 | 87 |
| Pre-op alb | 0.85 | 0.75, 0.96 | 0.011 | 82 |
| Post-op Hb | 0.67 | 0.46, 0.97 | 0.035 | 97 |
| Post-op alb | 0.90 | 0.81, 0.99 | 0.040 | 88 |
| Pre-op lostay | 1.00 | 0.97, 1.04 | 0.834 | 95 |
| Pre-op losurg | 1.00 | 0.99, 1.01 | 0.744 | 95 |
| Type anaesthetic | 6.91 | 1.51, 31.69 | 0.013 | 95 |
| Worst Braden | 1.05 | 0.84, 1.31 | 0.681 | 97 |
| Braden cat | 0.73 | 0.24, 2.21 | 0.580 | 97 |
| Braden moisture 3 | 1 | 0.10, 10.07 | 1.000 | 97 |
| 4 | 0.48 | 0.044, 5.14 | 0.541 | |
| Braden activity | 0.56 | 0.133, 2.32 | 0.420 | 97 |
| Braden mobility | 0.70 | 0.20, 2.40 | 0.572 | 97 |
| Braden nutrition | 0.73 | 0.23, 2.39 | 0.606 | 97 |
| Mattress 3 | 0.6 | 0.065, 5.58 | 0.653 | 96 |
| 4 | 1.17 | 0.36, 3.81 | 0.799 | |
| Systolic BP min | 1.00 | 0.97, 1.03 | 0.957 | 91 |
| Systolic BP max | 0.99 | 0.98, 1.02 | 0.843 | 91 |
| Systolic BP final | 0.99 | 0.97, 1.02 | 0.849 | 90 |
| Pre-op systolic BP | 1.00 | 0.98, 1.02 | 0.947 | 95 |
| Diastolic BP min | 0.95 | 0.90, 1.01 | 0.084 | 91 |
| Diastolic BP max | 0.96 | 0.93, 1.01 | 0.086 | 91 |
| Diastolic BP final | 0.98 | 0.94, 1.02 | 0.308 | 89 |
| Pre-op diastolicBP | 0.97 | 0.93, 1.02 | 0.226 | 95 |
| Grade 1b | 9.71 | 2.36, 39.97 | 0.002 | 97 |
| 1b+ | 9.17 | 1.17, 71.71 | 0.035 | |
| 0 | 3.06 | 0.27, 34.19 | 0.365 | |

## 5.1.2 ttest and Chi-square

**agecat**

| ps | agecat 55-75 | 75+ | Total |
|---|---|---|---|
| 0 | 36 | 46 | 82 |
| | 81.82 | 86.79 | 84.54 |
| 1 | 8 | 7 | 15 |
| | 18.18 | 13.21 | 15.46 |
| Total | 44 | 53 | 97 |
| | 100.00 | 100.00 | 100.00 |

**agesurg**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 82 | 74.58143 | .9983083 | 9.040066 | 72.59511 | 76.56775 |
| 1 | 15 | 77.95793 | 2.107623 | 8.162788 | 73.43753 | 82.47833 |
| combined | 97 | 75.10357 | .9091548 | 8.954136 | 73.29891 | 76.90822 |
| diff | | -3.376502 | 2.50388 | | -8.347332 | 1.594328 |

**tsurg**

| ps | tsurg Vascular | General | Ortho | Total |
|---|---|---|---|---|
| 0 | 34 | 18 | 30 | 82 |
| | 91.89 | 81.82 | 78.95 | 84.54 |
| 1 | 3 | 4 | 8 | 15 |
| | 8.11 | 18.18 | 21.05 | 15.46 |
| Total | 37 | 22 | 38 | 97 |
| | 100.00 | 100.00 | 100.00 | 100.00 |

**aesurg**

| ps | aesurg 1 | 2 | Total |
|---|---|---|---|
| 0 | 48 | 34 | 82 |
| | 90.57 | 77.27 | 84.54 |
| 1 | 5 | 10 | 15 |
| | 9.43 | 22.73 | 15.46 |
| Total | 53 | 44 | 97 |
| | 100.00 | 100.00 | 100.00 |

**wounds**

| ps | wounds Present | Absent | Total |
|---|---|---|---|
| 0 | 9 | 73 | 82 |
| | 69.23 | 86.90 | 84.54 |
| 1 | 4 | 11 | 15 |
| | 30.77 | 13.10 | 15.46 |
| Total | 13 | 84 | 97 |
| | 100.00 | 100.00 | 100.00 |

**diabet**

| ps | diabet Present | Absent | Total |
|---|---|---|---|
| 0 | 12 | 70 | 82 |
| | 80.00 | 85.37 | 84.54 |
| 1 | 3 | 12 | 15 |
| | 20.00 | 14.63 | 15.46 |
| Total | 15 | 82 | 97 |
| | 100.00 | 100.00 | 100.00 |

**bmi**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 77 | 24.04573 | .5162811 | 4.530348 | 23.01747 | 25.07399 |
| 1 | 15 | 23.04811 | 1.076071 | 4.167606 | 20.74016 | 25.35605 |
| combined | 92 | 23.88307 | .4656741 | 4.466589 | 22.95807 | 24.80808 |
| diff | | .9976245 | 1.263219 | | -1.511981 | 3.50723 |

**weight loss**

| ps | wl Present | Absent | Total |
|---|---|---|---|
| 0 | 13 | 60 | 73 |
| | 72.22 | 90.91 | 86.90 |
| 1 | 5 | 6 | 11 |
| | 27.78 | 9.09 | 13.10 |
| Total | 18 | 66 | 84 |
| | 100.00 | 100.00 | 100.00 |

**pretemp**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 81 | 36.58889 | .0487498 | .4387481 | 36.49187 | 36.6859 |
| 1 | 15 | 36.68 | .1000951 | .3876668 | 36.46532 | 36.89468 |
| combined | 96 | 36.60313 | .0439411 | .4305327 | 36.51589 | 36.69036 |
| diff | | -.0911108 | .1212976 | | -.3319499 | .1497283 |

**prehb**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 75 | 12.79467 | .1947777 | 1.686824 | 12.40656 | 13.18277 |
| 1 | 12 | 11.475 | .3455617 | 1.197061 | 10.71442 | 12.23558 |
| combined | 87 | 12.61264 | .1807112 | 1.685561 | 12.2534 | 12.97189 |
| diff | | 1.319667 | .5073302 | | .3109584 | 2.328375 |

**pohb**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 82 | 11.58659 | .1737584 | 1.57345 | 11.24086 | 11.93231 |
| 1 | 15 | 10.62667 | .3620664 | 1.402277 | 9.850112 | 11.40322 |
| combined | 97 | 11.43814 | .1604563 | 1.580311 | 11.11964 | 11.75665 |
| diff | | .9599186 | .4351114 | | .0961134 | 1.823724 |

**poalb**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 74 | 34.37838 | .653842 | 5.624561 | 33.07527 | 35.68148 |
| 1 | 14 | 30.85714 | 1.474535 | 5.517206 | 27.6716 | 34.04268 |
| combined | 88 | 33.81818 | .6102455 | 5.72461 | 32.60525 | 35.03111 |
| diff | | 3.521236 | 1.634577 | | .2718034 | 6.770668 |

**prelos**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 80 | 4.3125 | 1.527449 | 13.66192 | 1.27219 | 7.35281 |
| 1 | 15 | 5.066667 | 1.634353 | 6.329824 | 1.561327 | 8.572006 |
| combined | 95 | 4.431579 | 1.30951 | 12.76352 | 1.831517 | 7.031641 |
| diff | | -.7541667 | 3.609627 | | -7.922169 | 6.413836 |

**losurg**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 80 | 142.625 | 10.02093 | 89.62995 | 122.6788 | 162.5712 |
| 1 | 15 | 150.9333 | 25.72223 | 99.62176 | 95.76464 | 206.102 |
| combined | 95 | 143.9368 | 9.312682 | 90.7688 | 125.4463 | 162.4274 |
| diff | | -8.308333 | 25.66171 | | -59.2674 | 42.65074 |

**anaes**

| ps | anaes: General | anaes: Other | Total |
|---|---|---|---|
| 0 | 76 | 4 | 80 |
| | 87.36 | 50.00 | 84.21 |
| 1 | 11 | 4 | 15 |
| | 12.64 | 50.00 | 15.79 |
| Total | 87 | 8 | 95 |
| | 100.00 | 100.00 | 100.00 |

**worst Braden**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 82 | 15.91463 | .2672539 | 2.420087 | 15.38288 | 16.44639 |
| 1 | 15 | 16.2 | .7380799 | 2.858571 | 14.61698 | 17.78302 |
| combined | 97 | 15.95876 | .2516772 | 2.478733 | 15.45919 | 16.45834 |
| diff | | -.2853659 | .6991274 | | -1.673309 | 1.102577 |

**Braden category , lrchi col**

| ps | brad NAR | AR | Total |
|---|---|---|---|
| 0 | 32 | 50 | 82 |
| | 82.05 | 86.21 | 84.54 |
| 1 | 7 | 8 | 15 |
| | 17.95 | 13.79 | 15.46 |
| Total | 39 | 58 | 97 |
| | 100.00 | 100.00 | 100.00 |

**Braden sensory perception**

| ps | wsp 3 | 4 | Total |
|---|---|---|---|
| 0 | 7 | 75 | 82 |
| | 100.00 | 83.33 | 84.54 |
| 1 | 0 | 15 | 15 |
| | 0.00 | 16.67 | 15.46 |
| Total | 7 | 90 | 97 |
| | 100.00 | 100.00 | 100.00 |

**Braden moisture**

| ps | wmoist 2 | 3 | 4 | Total |
|---|---|---|---|---|
| 0 | 4 | 36 | 42 | 82 |
| | 80.00 | 80.00 | 89.36 | 84.54 |
| 1 | 1 | 9 | 5 | 15 |
| | 20.00 | 20.00 | 10.64 | 15.46 |
| Total | 5 | 45 | 47 | 97 |
| | 100.00 | 100.00 | 100.00 | 100.00 |

**Braden activity**

| ps | wact normal | ar | Total |
|---|---|---|---|
| 0 | 10 | 72 | 82 |
| | 76.92 | 85.71 | 84.54 |
| 1 | 3 | 12 | 15 |
| | 23.08 | 14.29 | 15.46 |
| Total | 13 | 84 | 97 |
| | 100.00 | 100.00 | 100.00 |

**Braden mobility**

| ps | wmob: normal | wmob: ar | Total |
|---|---|---|---|
| 0 | 54 | 28 | 82 |
| | 83.08 | 87.50 | 84.54 |
| 1 | 11 | 4 | 15 |
| | 16.92 | 12.50 | 15.46 |
| Total | 65 | 32 | 97 |
| | 100.00 | 100.00 | 100.00 |

**Braden nutrition**

| ps | wnut: normal | wnut: ar | Total |
|---|---|---|---|
| 0 | 22 | 60 | 82 |
| | 81.48 | 85.71 | 84.54 |
| 1 | 5 | 10 | 15 |
| | 18.52 | 14.29 | 15.46 |
| Total | 27 | 70 | 97 |
| | 100.00 | 100.00 | 100.00 |

likelihood-ratio chi2(1) = 0.2592 Pr = 0.611

**Braden friction and shear**

| ps | wfs: 1 | wfs: 2 | wfs: 3 | Total |
|---|---|---|---|---|
| 0 | 5 | 43 | 34 | 82 |
| | 100.00 | 82.69 | 85.00 | 84.54 |
| 1 | 0 | 9 | 6 | 15 |
| | 0.00 | 17.31 | 15.00 | 15.46 |
| Total | 5 | 52 | 40 | 97 |
| | 100.00 | 100.00 | 100.00 | 100.00 |

**Mattress**

| ps | matt: 2 | matt: 3 | matt: 4 | Total |
|---|---|---|---|---|
| 0 | 36 | 10 | 36 | 82 |
| | 85.71 | 90.91 | 83.72 | 85.42 |
| 1 | 6 | 1 | 7 | 14 |
| | 14.29 | 9.09 | 16.28 | 14.58 |
| Total | 42 | 11 | 43 | 96 |
| | 100.00 | 100.00 | 100.00 | 100.00 |

**sbpmin**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 76 | 95.34211 | 2.528889 | 22.04635 | 90.3043 | 100.3799 |
| 1 | 15 | 95.66667 | 4.427906 | 17.14921 | 86.16975 | 105.1636 |
| combined | 91 | 95.3956 | 2.225717 | 21.23199 | 90.97383 | 99.81738 |
| diff | | -.3245614 | 6.032233 | | -12.31048 | 11.66136 |

**sbpmax**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 76 | 138.4737 | 3.021936 | 26.34463 | 132.4537 | 144.4937 |
| 1 | 15 | 137 | 7.166058 | 27.75402 | 121.6303 | 152.3697 |
| combined | 91 | 138.2308 | 2.770509 | 26.42897 | 132.7267 | 143.7349 |
| diff | | 1.473684 | 7.507249 | | -13.44306 | 16.39043 |

**sbpfinal**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 75 | 118.4933 | 2.709501 | 23.46497 | 113.0945 | 123.8921 |
| 1 | 15 | 117.2 | 7.328483 | 28.38309 | 101.482 | 132.918 |
| combined | 90 | 118.2778 | 2.548997 | 24.18191 | 113.213 | 123.3426 |
| diff | | 1.293333 | 6.877047 | | -12.37335 | 14.96002 |

**preopsbp**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 80 | 137.4375 | 3.02813 | 27.08442 | 131.4102 | 143.4648 |
| 1 | 15 | 137.9333 | 6.194826 | 23.99246 | 124.6468 | 151.2199 |
| combined | 95 | 137.5158 | 2.718888 | 26.50045 | 132.1174 | 142.9142 |
| diff | | -.4958333 | 7.496121 | | -15.38164 | 14.38998 |

**dbpmin**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 76 | 48.67105 | 1.417731 | 12.35949 | 45.84679 | 51.49532 |
| 1 | 15 | 42.73333 | 2.222111 | 8.606199 | 37.96738 | 47.49929 |
| combined | 91 | 47.69231 | 1.256747 | 11.9886 | 45.19556 | 50.18905 |
| diff | | 5.937719 | 3.347488 | | -.7136669 | 12.58911 |

**dbpmax**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 76 | 74.05263 | 1.756981 | 15.317 | 70.55255 | 77.55272 |
| 1 | 15 | 66.6 | 3.313465 | 12.83299 | 59.49333 | 73.70667 |
| combined | 91 | 72.82418 | 1.58585 | 15.12804 | 69.67361 | 75.97474 |
| diff | | 7.452632 | 4.224886 | | -.942126 | 15.84739 |

**dbpfinal**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 75 | 59.69333 | 1.669436 | 14.45774 | 56.36691 | 63.01976 |
| 1 | 14 | 55.42857 | 3.710268 | 13.88255 | 47.41302 | 63.44412 |
| combined | 89 | 59.02247 | 1.523897 | 14.37641 | 55.99405 | 62.0509 |
| diff | | 4.264762 | 4.184615 | | -4.052614 | 12.58214 |

**preopdbp**

| Group | Obs | Mean | Std. Err. | Std. Dev. | [95% Conf. | Interval] |
|---|---|---|---|---|---|---|
| 0 | 80 | 72.3125 | 1.505643 | 13.46688 | 69.31559 | 75.30941 |
| 1 | 15 | 67.66667 | 3.626183 | 14.04415 | 59.88928 | 75.44406 |
| combined | 95 | 71.57895 | 1.394324 | 13.59019 | 68.81049 | 74.34741 |
| diff | | 4.645833 | 3.814011 | | -2.928036 | 12.2197 |

**grade**

| | grade | | | | |
|---|---|---|---|---|---|
| ps | 1a | 1b | 1b+ | 0 | Total |
| 0 | 55 | 17 | 4 | 6 | 82 |
| | 94.83 | 65.38 | 66.67 | 85.71 | 84.54 |
| 1 | 3 | 9 | 2 | 1 | 15 |
| | 5.17 | 34.62 | 33.33 | 14.29 | 15.46 |
| Total | 58 | 26 | 6 | 7 | 97 |
| | 100.00 | 100.00 | 100.00 | 100.00 | 100.00 |

## 5.2 Imputation commands used to model missing data

**Prehb**

```
 impute prehb   pohb prealb poalb ps bmi agesurg , gen(prehb_i)
 10.31% (10) observations imputed
```

**Prealb**

```
 impute prealb  pohb prehb  poalb ps bmi agesurg , gen(prealb_i)
 15.46% (15) observations imputed
```

**wl**

```
 gen xwl=wl
 (13 missing values generated)

 xi:logistic  xwl bmi

Logit estimates                                   Number of obs   =         80
                                                  LR chi2(1)      =       6.85
                                                  Prob > chi2     =     0.0089
Log likelihood = -39.228405                       Pseudo R2       =     0.0803

------------------------------------------------------------------------------
         xwl | Odds Ratio   Std. Err.       z     P>|z|       [95% Conf. Interval]
-------------+----------------------------------------------------------------
         bmi |   .8388533   .0614055    -2.400   0.016       .7267358    .9682678
------------------------------------------------------------------------------

. predict pwl
(option p assumed; Pr(xwl))
(5 missing values generated)

. gen     wl_i = wl
(13 missing values generated)

. replace wl_i=1 if bmi<27  & wl==. & bmi~=.
(9 real changes made)

. replace wl_i=2 if bmi>=27 & wl==. & bmi~=.
(3 real changes made)

.
. l studyno wl pwl bmi wl_i if wl==.

        studyno        wl         pwl         bmi       wl_i
 10.       035a         .    .2204292    23.16524          1
 11.       012a         .    .2475073    22.30469          1
 13.      049a1         .    .3242235    20.15625          1
 14.       036a         .    .4044564    18.17867          1
 18.       047a         .           .           .          .
 34.       024a         .     .102612    28.31758          2
 53.       042a         .    .3070284    20.60935          1
 55.       045e         .    .0513406    32.57457          2
 59.       033e         .     .300469    20.78587          1
 65.       034e         .    .1772088    24.71433          1
 71.       010a         .    .1749058    24.80469          1
 83.       023e         .    .2392745    22.55912          1
 88.       047e         .      .11174    27.77442          2

.
```

## 5.3 Multi-factoral modelling

### 5.3.1 Forward stepwise logistic regression

```
sw logistic  ps     agesurg  i.anaes  i.grade  prehb_i  prealb_i  i.wl_i i.wounds
dbpmin,    forward pe(0.25) pr(0.9)

i.anaes               Ianaes_1-2   (naturally coded; Ianaes_1 omitted)
i.grade               Igrade_1-2   (naturally coded; Igrade_1 omitted)
i.wl_i                Iwl_i_1-2    (naturally coded; Iwl_i_1 omitted)
i.wounds              Iwound_1-2   (naturally coded; Iwound_1 omitted)
                      begin with empty model
p = 0.0006 <  0.2500  adding   prealb_i
p = 0.0045 <  0.2500  adding   Igrade_2
p = 0.1231 <  0.2500  adding   Iwl_i_2
p = 0.2050 <  0.2500  adding   dbpmin

Logit estimates                                   Number of obs   =         90
                                                  LR chi2(4)      =      28.34
                                                  Prob > chi2     =     0.0000
Log likelihood =  -26.37836                       Pseudo R2       =     0.3495
------------------------------------------------------------------------------
      ps | Odds Ratio   Std. Err.       z     P>|z|       [95% Conf. Interval]
---------+--------------------------------------------------------------------
prealb_i |   .8129145   .0641146    -2.626   0.009       .6964831    .9488097
Igrade_2 |   7.018493   5.140427     2.660   0.008       1.670375    29.48994
 Iwl_i_2 |   .2979539   .2142419    -1.684   0.092       .0727944    1.219552
  dbpmin |   .9603676   .0306417    -1.267   0.205       .9021503    1.022342
------------------------------------------------------------------------------
```

### 5.3.2 Final model selection

```
.
. xi: logistic ps                 i.grade i.wl_i dbpmin         pohb
i.grade               Igrade_1-2   (naturally coded; Igrade_1 omitted)
i.wl_i                Iwl_i_1-2    (naturally coded; Iwl_i_1 omitted)

Logit estimates                                   Number of obs   =         90
                                                  LR chi2(4)      =      22.04
                                                  Prob > chi2     =     0.0002
Log likelihood =  -29.53023                       Pseudo R2       =     0.2718

------------------------------------------------------------------------------
      ps | Odds Ratio   Std. Err.       z     P>|z|       [95% Conf. Interval]
---------+--------------------------------------------------------------------
Igrade_2 |   6.899318   4.780219     2.788   0.005       1.774385    26.82653
 Iwl_i_2 |   .2648163   .1807045    -1.947   0.052       .0695191    1.008753
  dbpmin |   .9439873   .0302887    -1.797   0.072       .8864505    1.005258
    pohb |   .7402889    .159406    -1.397   0.163       .4854142     1.12899
------------------------------------------------------------------------------
NB pohb associated with pressure sore development <0.25, therefore substitue prehb for
pohb in stepwise model


. xi: logistic ps                 i.grade i.wl_i dbpmin         poalb
i.grade               Igrade_1-2   (naturally coded; Igrade_1 omitted)
i.wl_i                Iwl_i_1-2    (naturally coded; Iwl_i_1 omitted)

Logit estimates                                   Number of obs   =         81
                                                  LR chi2(4)      =      22.45
                                                  Prob > chi2     =     0.0002
Log likelihood = -26.063233                       Pseudo R2       =     0.3011

------------------------------------------------------------------------------
      ps | Odds Ratio   Std. Err.       z     P>|z|       [95% Conf. Interval]
---------+--------------------------------------------------------------------
Igrade_2 |   11.23297   8.778142     3.095   0.002       2.428356    51.96089
 Iwl_i_2 |   .3053555   .2275324    -1.592   0.111        .070883    1.315435
  dbpmin |   .9519405   .0302931    -1.548   0.122       .8943808    1.013205
   poalb |   .9594919   .0671623    -0.591   0.555       .8364869    1.100585
------------------------------------------------------------------------------
NB poalb not associated with presure sore development when substituted for prealb
therefore prealb remains most approriate variable for inclusion.
```

```
. xi: logistic ps          i.grade i.wl_i dbpmin        i.wounds
i.grade                Igrade_1-2    (naturally coded; Igrade_1 omitted)
i.wl_i                 Iwl_i_1-2     (naturally coded; Iwl_i_1 omitted)
i.wounds               Iwound_1-2    (naturally coded; Iwound_1 omitted)

Logit estimates                                    Number of obs   =         90
                                                   LR chi2(4)      =      20.06
                                                   Prob > chi2     =     0.0005
Log likelihood = -30.521438                        Pseudo R2       =     0.2473

------------------------------------------------------------------------------
      ps | Odds Ratio   Std. Err.       z     P>|z|       [95% Conf. Interval]
---------+--------------------------------------------------------------------
Igrade_2 |   7.992186   5.638323      2.946   0.003       2.005212     31.8545
 Iwl_i_2 |   .2697125   .1799479     -1.964   0.050       .0729445     .997262
  dbpmin |   .9482685   .0294605     -1.710   0.087       .8922499    1.007804
Iwound_2 |   .7918889   .6888438     -0.268   0.789       .1439547    4.356148
------------------------------------------------------------------------------
```

**NB wounds not associated with presure sore development when prealb removed from model therefore prealb remains most approriate variable for inclusion.**

```
. xi: logistic ps          i.grade i.wl_i dbpmin        agesurg

i.grade                Igrade_1-2    (naturally coded; Igrade_1 omitted)
i.wl_i                 Iwl_i_1-2     (naturally coded; Iwl_i_1 omitted)

Logit estimates                                    Number of obs   =         90
                                                   LR chi2(4)      =      20.17
                                                   Prob > chi2     =     0.0005
Log likelihood = -30.467456                        Pseudo R2       =     0.2487

------------------------------------------------------------------------------
      ps | Odds Ratio   Std. Err.       z     P>|z|       [95% Conf. Interval]
---------+--------------------------------------------------------------------
Igrade_2 |   7.630197   5.455709      2.842   0.004       1.878942     30.98547
 Iwl_i_2 |   .2734031   .1822628     -1.945   0.052       .0740209      1.00984
  dbpmin |   .9463357   .0289002     -1.806   0.071       .8913544     1.004709
 agesurg |   1.019574   .0468922      0.421   0.673       .9316881     1.115751
------------------------------------------------------------------------------
```

**NB agesurg not associated with presure sore development when prealb removed from model therefore prealb remains most approriate variable for inclusion.**

```
. xi: logistic ps prealb_i i.grade i.wl_i                        dbpmax

i.grade                Igrade_1-2    (naturally coded; Igrade_1 omitted)
i.wl_i                 Iwl_i_1-2     (naturally coded; Iwl_i_1 omitted)

Logit estimates                                    Number of obs   =         90
                                                   LR chi2(4)      =      28.76
                                                   Prob > chi2     =     0.0000
Log likelihood = -26.169593                        Pseudo R2       =     0.3546

------------------------------------------------------------------------------
      ps | Odds Ratio   Std. Err.       z     P>|z|       [95% Conf. Interval]
---------+--------------------------------------------------------------------
prealb_i |   .7949572   .0630491     -2.893   0.004        .680509     .9286534
Igrade_2 |   6.123963   4.487125      2.473   0.013       1.456612     25.74669
 Iwl_i_2 |   .3019519   .2193059     -1.649   0.099       .0727302     1.253605
  dbpmax |   .9630256   .0258809     -1.402   0.161       .9136127     1.015111
------------------------------------------------------------------------------
```

**NB dbpmax has smaller p value and similar odds ratio to dbpmin therefore substitue dbpmax for dbpmin in model**

```
. xi: logistic ps prealb_i i.grade i.wl_i dbpmin        i.aesurg

i.grade               Igrade_1-2   (naturally coded; Igrade_1 omitted)
i.wl_i                Iwl_i_1-2    (naturally coded; Iwl_i_1 omitted)
i.aesurg              Iaesur_1-2   (naturally coded; Iaesur_1 omitted)

Logit estimates                                   Number of obs   =         90
                                                  LR chi2(5)      =      28.93
                                                  Prob > chi2     =     0.0000
Log likelihood = -26.086843                       Pseudo R2       =     0.3567

------------------------------------------------------------------------------
      ps | Odds Ratio   Std. Err.       z     P>|z|       [95% Conf. Interval]
---------+--------------------------------------------------------------------
prealb_i |   .8386749   .0730508    -2.020   0.043       .7070526    .9947994
Igrade_2 |   6.495016   4.784121     2.540   0.011       1.533202    27.51447
 Iwl_i_2 |   .2563404   .1944182    -1.795   0.073       .0579741    1.133445
  dbpmin |   .9539565   .0316577    -1.420   0.155       .8938834    1.018067
Iaesur_2 |   1.901073   1.612478     0.757   0.449       .3605912    10.02265
------------------------------------------------------------------------------
```

**NB aesurg not associated with presure sore development (p>0.25) in the presence of other variables therefore not approriate substitute for anaes.**

```
. xi: sw logistic  ps      i.grade
>                          pohb
>                          prealb_i
>                          i.wl_i
>                          dbpmax,
>                          forward pe(0.25) pr(0.9)
>                          ;
i.grade               Igrade_1-2   (naturally coded; Igrade_1 omitted)
i.wl_i                Iwl_i_1-2    (naturally coded; Iwl_i_1 omitted)
                      begin with empty model
p = 0.0006 <  0.2500  adding   prealb_i
p = 0.0045 <  0.2500  adding   Igrade_2
p = 0.1231 <  0.2500  adding   Iwl_i_2
p = 0.1609 <  0.2500  adding   dbpmax

Logit estimates                                   Number of obs   =         90
                                                  LR chi2(4)      =      28.76
                                                  Prob > chi2     =     0.0000
Log likelihood = -26.169593                       Pseudo R2       =     0.3546

------------------------------------------------------------------------------
      ps | Odds Ratio   Std. Err.       z     P>|z|       [95% Conf. Interval]
---------+--------------------------------------------------------------------
prealb_i |   .7949572   .0630491    -2.893   0.004        .680509    .9286534
Igrade_2 |   6.123963   4.487125     2.473   0.013       1.456612    25.74669
 Iwl_i_2 |   .3019519   .2193059    -1.649   0.099       .0727302    1.253605
  dbpmax |   .9630256   .0258809    -1.402   0.161       .9136127    1.015111
------------------------------------------------------------------------------
```

**NB pohb in the presence of prealb is not associated with pressure sore development at the significance level p<0.25 therefore not required in the final model.**

### .5.3.3 Final Core Model

```
.
. xi: logistic ps prealb_i i.grade i.wl_i dbpmin
i.grade                Igrade_1-2    (naturally coded; Igrade_1 omitted)
i.wl_i                 Iwl_i_1-2     (naturally coded; Iwl_i_1 omitted)

Logit estimates                                    Number of obs   =         90
                                                   LR chi2(4)      =      28.34
                                                   Prob > chi2     =     0.0000
Log likelihood =  -26.37836                        Pseudo R2       =     0.3495

------------------------------------------------------------------------------
      ps | Odds Ratio   Std. Err.       z     P>|z|     [95% Conf. Interval]
---------+--------------------------------------------------------------------
prealb_i |   .8129145   .0641146    -2.626   0.009      .6964831    .9488097
Igrade_2 |   7.018493   5.140427     2.660   0.008      1.670375    29.48994
 Iwl_i_2 |   .2979539   .2142419    -1.684   0.092      .0727944    1.219552
  dbpmin |   .9603676   .0306417    -1.267   0.205      .9021503    1.022342
------------------------------------------------------------------------------
OR.
. xi: logistic ps prealb_i i.grade i.wl_i dbpmax
i.grade                Igrade_1-2    (naturally coded; Igrade_1 omitted)
i.wl_i                 Iwl_i_1-2     (naturally coded; Iwl_i_1 omitted)

Logit estimates                                    Number of obs   =         90
                                                   LR chi2(4)      =      28.76
                                                   Prob > chi2     =     0.0000
Log likelihood = -26.169593                        Pseudo R2       =     0.3546

------------------------------------------------------------------------------
      ps | Odds Ratio   Std. Err.       z     P>|z|     [95% Conf. Interval]
---------+--------------------------------------------------------------------
prealb_i |   .7949572   .0630491    -2.893   0.004       .680509    .9286534
Igrade_2 |   6.123963   4.487125     2.473   0.013      1.456612    25.74669
 Iwl_i_2 |   .3019519   .2193059    -1.649   0.099      .0727302    1.253605
  dbpmax |   .9630256   .0258809    -1.402   0.161      .9136127    1.015111
------------------------------------------------------------------------------
```