Intra-operative pressure sores

Bridel, Jane Elizabeth

Intra-operative Pressure Sores

Abstract

As the extent of the pressure sore problem within hospitalized populations has been realized, their costs estimated and changes in the management of the NHS occurred, interest in pressure sores has increased and focused toward prevention. Research has served to underline the complexities of the issues involved and it is now acknowledged that the development of a pressure sore is dependent upon a complicated interplay of many variables, including the intensity and duration of pressure and the tolerance of the skin.

The focus of this study was to generate data relating to the research question 'what is the extent of intra-operative pressure damage to the skin within a UK hospital setting?'. However, also pertinent was literature relating to the use of risk assessment tools - their predictive validity and value as descriptors of research patient populations. A quantitative descriptive study design was developed to determine post-operative incidence of pressure sore development and the reliability and predictive validity of the Braden Scale. Patients were recruited to the study according to age (=>55 years), surgery type (elective major general and vascular) and intra-operative position (supine and lithotomy). Descriptive data relating to risk, skin and peri-operative time intervals were recorded.

Skin assessments amongst a sample of 26 patients identified a pre-operative prevalence of 36% - an unexpected finding, a post-operative incidence of 12.5% consistent with results from North American research and a period prevalence of 56%. The Braden Scale demonstrated good reliability and the most sensitive and specific Braden Score was determined as 19 not at risk/<19 at risk. A discussion of results identified limitations relating to the methodology adopted and issues, such as, inclusion and exclusion criteria of blanching and non blanching reactive hyperaemia and the application of the definition of incidence which lacked clarity within the literature.

Intra-operative Pressure Sores

Jane Elizabeth Bridel

Master of Arts

University of Durham

Department of Sociology

Year of Submission: 1995

Table of Contents

List of Figures

List of Tables

List of Abbreviations

AHCPR	Agency for Health Care Policy and Research
CSAG	Clinical Standards Advisory Group
DHSS	Department of Health and Social Security
DoH	Department of Health
HMSO	Her Majesty's Stationary Office
IAET	International Association of Enterostomal Therapy
Jr	Junior
NHS	National Health Service
NPRU	Nursing Practice Research Unit
ODA	Operating Department Assistance

Section I: A Review of the Literature

Chapter 1 - Introduction

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' (Parish et al 1983). Their occurrence has been a challenging phenomenon throughout the centuries, the problems dating back at least to the time of the Pharaohs, as evidenced on the mummified body of an Egyptian priestess (Thompson-Rowling 1961). Recent studies exploring the extent of the problem within the National Health Service (NHS) have revealed hospital prevalence rates ranging from 4-18.6% (Barbenel et al 1977, David et al 1983, Girvin and Griffiths-Jones 1989 and O'Dea 1993).

Pressure sores have both cost and quality implications for health services. Patients suffer discomfort and pain, and in the extreme pressure sores cause death. Health authorities face increased expenditure for the individual patient with a pressure sore, as well as reduced availability of hospital beds and the through-put potential of other patients (Hibbs 1988).

The need to establish targets for pressure sore reduction has been recommended by Government (DoH 1993) and many Purchasing Authorities have included targets in Quality Contracting for 1993/94 and 1994/95. They are increasingly being seen as preventable sequelae rather than a tolerable complication of illness, and the emphasis is on identifying risk factors and implementing appropriate preventative interventions. This is reflected in the development of a plethora of local pressure sore prevention policies in recent years (Hibbs 1982, Starling 1990, DoH 1993), which advocate risk assessment, skin care, repositioning, equipment provision and planned monitoring of the problem.

Increased interest and research has served to underline the complexity of the issues involved more than providing simple solutions. It is now acknowledged that the development of a pressure sore is likely to be dependent upon a complicated interplay of many variables, both intrinsic and extrinsic in nature (Cullum and Clark 1992). 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 to the pressure applied (Braden and Bergstrom 1987). 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 amongst bioengineers. Criticisms of research in this field relate to the use of animal skin for experiments, the omission of loading shear in calculations, a failure to account for autoregulation processes and differences in tissue tolerance.

Despite the limitations of such research and little clinical data in support, general aims of equipment and practice have been to provide support surfaces that generate interface pressures of less than 32mmHg (that is, mean capillary pressure) and/or intermittent relief of pressure on a given area of skin after a period not exceeding two hours (CSAG 1993). The individual nature of the skin's response to pressure and the complicated interplay of factors involved in tissue damage challenge the use of these general threshold values and question their applicability to practice.

Factors affecting tissue tolerance can be subdivided into those that are extrinsic and intrinsic in nature. Their exact contribution 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. In particular, biological factors, such as measures of plasma proteins and blood pressure require further examination to establish their role as aetiological factors.

With specific reference to theatre, a comprehensive review of the literature reveals little information relating to the genesis of intra-operative pressure sores, and the contribution of operating room practice on aetiology is undefined. The following chapters provide an overview of pressure sore pathophysiology, detail the recent interest in and trend to prevent pressure sores, review causes established by both epidemiological and pathophysiological research, discuss the value of pressure sore risk assessment tools within the context of practice and research, and review the literature relating specifically to pressure sore development and the operating department.

Chapter 2 - The Pathophysiology of Pressure Sores

2.1 Introduction

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' (Parish et al 1983). They are complex lesions of the skin and underlying structures and vary considerably in size and severity. This Chapter clarifies the anatomical structures affected and structural elements which play a role in pressure sore pathology, as well as providing an overview of the descriptive pressure sore classifications found within the literature.

2.2 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 (Krouskop 1983) and is a dynamic structure in which cellular replacement and modification in response to local need is a continual process throughout life (Barton and Barton 1981). It is relatively resistant to water, chemicals and bacteria and provides some protection for the body against mechanical damage. Structurally, it consists of 2 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. Three of the layers are worthy of mention. 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 (Torrance 1983). 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 (Torrance 1983 and Barton and Barton 1981). 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, 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 which help protect against mechanical damage. Indeed the physical characteristics of the dermis are essentially determined by the collagen/elastin matrix.

Collagen is synthesized in connective tissue fibroblasts, secreted from the cells and stabilized by the formation of cross-linkages which vary in permanence. It constitutes 99% dry weight dermis (Hall 1984). 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 (Hall 1984). 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 (Krouskop 1983).

A subcutaneous layer separates the dermis from the deeper structures of deep

fascia, muscle and bone. Containing similar structures to the dermis 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 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 it is the last line of protection of 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-epithelial 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. 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.

2.3 The Vascular System and Capillary Blood Flow

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 is supported by a network of vascular and lymph vessels. 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.

In relation to pressure sore aetiology 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 (Torrance 1983). The arterioles branch into a network of metarterioles (throughfare vessels), capillaries and venules. These structures are known collectively as the micro-circulation (Lamb et al 1980). Muscle cells at the origin of the capillaries act as pre-capillary sphincters and are important in the control of blood flow.

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 the peripheral resistance. It alters the tone of smooth muscle in the walls of the arterioles which under normal conditions maintain a continuous vasoconstrictor tone (Lippold and Winton 1979). 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 their so called name of throughfare vessel. 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 precapillary sphincters - a phenomenon known as 'active vasomotion' (Kosiak 1961). The precapillary sphincters determine flow independent of the action of the arterioles and are controlled by the release of vasodilator substance and/or oxygen demand (Guyton 1992).

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 (Kosiak 1961 and Lamb et al 1980) since capillaries remain closed for 60-95% of the time. 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.

Difficulties occur in the determination of capillary (hydrostatic) pressure since measurement renders the vessel abnormal (Lippold and Winton 1979). Values adopted are from the work of Landis (1930) 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.

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. In relation to pressure sore aetiology, the fragile nature of the structure of the capillary wall and their low intravascular pressure render them particularly vulnerable to occlusion and/or damage by external loads. Indeed, pressure sores do develop mainly as a result of disruption to the vascular network. The next section details the pathophysiological processes involved.

2.4 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 (Barton and Barton 1981). 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. The following paragraphs detail both the processes individually and then classifies the progression of tissue damage in terms of its clinical appearance.

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 precapillary sphincters and metarterioles. The increase in blood flow may reach 30 times its resting value (Lamb et al 1980) and the bright red flush so produced is known as reactive hyperaemia.

The hyperaemic reaction is proportional to the duration of the occlusion and generally lasts 1/2 - 3/4 of the occlusion time (Lewis and Grant 1925 and Goldblatt 1925). 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 (Krouskop et al 1978). 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 (see Figure 1). Squeeze out of interstitial fluid is important in two ways. Firstly, 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 (Krouskop 1983).

Secondly, upon 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. Evidence of this first stage of skin necrosis is non blanching reactive hyperaemia, swelling, induration or loss of the epidermis by blistering or ulceration.

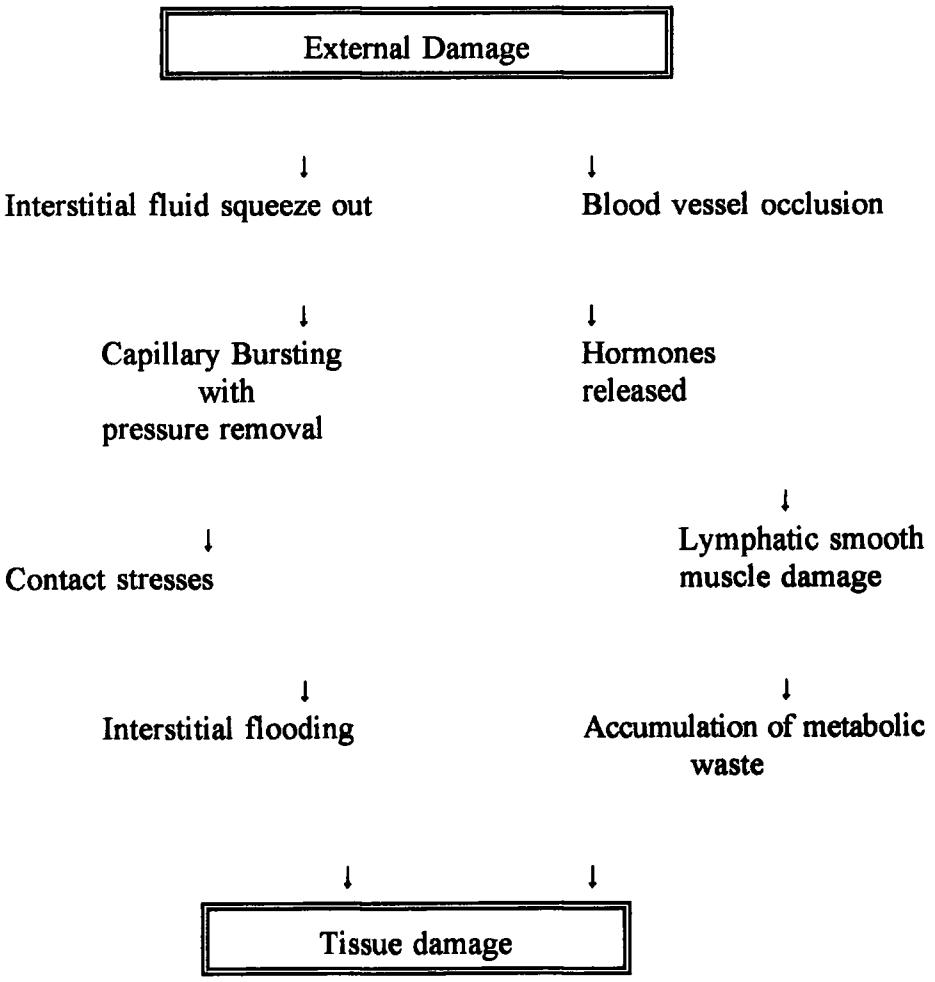
The interstitial oedema interferes with metabolite exchange, causes distortion and thickening of tissues compressed between bone and the support surface, and further increases the vulnerability of the skin (Torrance 1983). Progressive loss of tissue occurs if the application of pressure is not relieved, the wound extending inward. This is described as a Type 1 pressure sore by Barton and Barton (1981).

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. Distortion of the blood vessels disrupt endothelial cells and activate 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 extent of the tissue damage beneath (Barton and Barton 1981).

Figure 1

Integrated Model of Tissue Damage



Krouskop (1983 p.264)

This is described as a Type 2 pressure sore by Barton and Barton (1981) and upon presentation such a pressure sore would be classified as a full thickness

sore (see below). Particularly vulnerable to this type of damage are arterioles and the microcirculation of the subcutaneous layer.

Having briefly reviewed the two main pathophysiological processes which result in tissue necrosis it is valuable to analyze and classify the events which occur since tissue changes can range from reactive hyperaemia to the appearance of a large cavity.

2.5 The Classification of Pressure Sores

Pressure sores have been classified by a number of authors on the basis of their pathology (Barton and Barton 1981) and clinical appearance (Shea 1975, David et al 1983, Torrance 1983, IAET 1987, Lowthian 1987, Lyder 1991, AHCPR 1992, DoH 1993 and Reid and Morison 1994).

A review of the classification systems reveals that with the exceptions of Barton and Barton (1981) and Forrest (1980) who base their classifications upon pressure sore pathology, a number of general trends. The words 'Grade' and 'Stage' are interchangeable, and wound characteristics graded from 2-4/5 are similarly categorised by different authors, based upon clinical appearance. Overlap, however, is apparent depending upon the starting point of Stage 1 and whether 4 or 5 categories are defined.

For example: Stage 1 pressure sores are described as reactive hyperaemia and/or other skin changes where the skin remains intact and/or loss of the epidermis; Stage 2 pressure sore as a superficial break in the skin involving the epidermis and/or dermis; Stage 3 as involving the dermis and/or subcutaneous fat, Stage 4 sores as including subcutaneous fat and/or muscle and bone and; Stage 5 as muscle and bone involvement.

Categories including the descriptors superficial, partial thickness and full thickness wounds are also found within these classifications (IAET 1987,

Lowthian 1987, AHCPR 1992, DoH 1993 and Reid and Morison 1994). These generally classify breaks in the epidermis/dermis as superficial or partial thickness wounds and those involving the subcutaneous fat and any tissue beneath as full thickness wounds.

The differences in the classification systems used, although indistinct, do complicate the review of the literature particularly where comparison of trends from subsections of the literature require discussion (for example, prevalence and incidence - see Chapter 4). To enable simple presentation, comparison and contrasting of the literature in proceeding chapters, the results of pressure sore research shall be broadly categorised as detailed in Table 1. These categories represent, in the broadest terms, aspects relating to severity and morbidity.

Table 1

Pressure Sore Descriptor Definitions

Reactive Hyperaemia	either non-blanching or blanching and may be of variable duration
Partial Thickness	loss of epidermis and/or dermis.
Full Thickness	involvement of sub-cutaneous fat and/or muscle and bone.

2.6 Summary

Pressure sores are complex lesions of the skin which develop as a result of two processes - occlusion of blood vessels by external pressure and endothelial damage of arterioles and the micro-circulation due to the application of disruptive and shearing forces (Barton and Barton 1981). 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.

Their severity varies from reactive hyperaemia to tissue destruction involving

skin, subcutaneous fat, muscle and bone - hence a number of classification systems have been designed. In broad terms they can be described as reactive hyperaemia (either non-blanching or blanching), partial thickness wounds (loss of epidermis and/or dermis) and full thickness wounds (sub-cutaneous fat, muscle and bone).

Chapter 3 - The Trend Toward Prevention

3.1 Introduction

As the extent of the pressure sore problem within hospitalized populations has been realized, their costs estimated and changes in the management of the NHS occurred, interest in pressure sores has increased and focused toward prevention of pressure sores. This Chapter briefly details the historical development of current interest in the prevention of pressure sores, the component parts of the many district level pressure sore prevention strategies and difficulties which arise in determining the effectiveness of recommended practice. Within this context the issue of pressure sore prevention in the operating department is explored providing some background with regard to the need to investigate this area of practice.

3.2 Historical Perspective

The past thirty years has seen an abundance of epidemiological research highlighting the extent of the pressure sore problem. Early milestones including the work of Norton et al (1962), Barbenel et al (1977), Hibbs (1982) and David et al (1983) are still frequently referenced and compared to more recent results. These studies reported prevalence rates ranging from 6.6-8.8% (Barbenel et al 1977, David et al 1983 and Hibbs 1982) and illustrated high incidence/prevalence rates amongst specific patient populations, such as elderly (Norton et al 1962 and Barbenel et al 1977) and orthopaedic patients (Hibbs 1982).

During the same period speculation and calculations with regard to the cost of pressure sores to the NHS were made by several authors. For example, Fernie (1973) using prevalence figures estimated an annual National cost of £60 million pounds (60,000 pressure sores at £1,000/sore). This figure was later used as the basis for further calculations and 'inflation adjusted' by Scales et

al (1982) - £150 million, Waterlow (1985) - £200 million and Exton-Smith (1987) - £420 million. These figures are frequently cited within the pressure area care literature (Barbenel et al 1977, Dealey 1991, Livesley 1989, Simpson and Livesley 1993, Hunt 1993, Cubbin and Jackson 1991, Smith 1993, Clough 1994, Preston 1991 and Young and Dobrzanski 1992) despite the limitations of the original calculation.

Also reported are the results of a case study which calculated the opportunity costs of hospital care for one patient with a large gangrenous sacral pressure sore to be £25,905 (Hibbs 1988). Although criticised due to the inclusion of fixed and marginal costs (NPRU 1992) which, if excluded, leave a residual cost of £5,116 specific to the pressure sore (of which £4,085 are attributed to bed hire), the study illustrated the extent of lost opportunity in relation to lost patient days, standard admissions and standard cases.

In response to concerns regarding the extent of the problem, avoidable human suffering and financial waste, Professor Brian Livesley convened and led the Working Party of the King's Fund Pressure Sore Study Group in 1987 (Livesley 1989). Following an exploratory workshop and two National symposia the Working Party recommended that each District Health Authority develop a strategy for the prevention and management of pressure sores. The component parts in the development of such a strategy were published in 1989 (Livesley 1989), by which time The King's Fund Group had assisted some 20% of Health Districts in the United Kingdom to develop strategies for the prevention and management of pressure sores.

The momentum in interest has been further maintained by changes in the management and organization of health services and increasing emphasis upon cost effectiveness and efficiency in health care delivery, monitoring of indicators of the quality of care delivered (DHSS 1983) and a National directive toward ill-health prevention and health promotion within both acute and community settings (DoH 1991). Indeed, 'The Health of the Nation, A

Consultative Document for Health in England' (DoH 1991) made specific reference to the pressure sore problem and stated "It is currently considered that pressure sores are largely preventable by a district-level multi-disciplinary programme of intervention ..... The Government's view is that an annual reduction of at least 5%-10% in their incidence would be a reasonable target." (p.98).

The discussions that followed the release of this consultative document highlighted the paucity of incidence data upon which to base targets and little evidence of the effectiveness of preventative strategies (NPRU 1992). This lack of baseline knowledge precluded the setting of National targets within the Government White Paper, 'The Health of the Nation, A Strategy for Health in England' released later (DoH 1992).

However, further discussion and attention followed and led to the publication of 'Prevention and Treatment of Pressure Sores: Guidelines for Good Practice' by the Clinical Standards Advisory Group (CSAG 1993), questioning of the Government's "programme of action on the prevention of pressure sores" by Baroness Masham of Ilton in the House of Lords (Hansard 1993) and the publication of 'Pressure Sores A Key Quality Indicator' by the Department of Health (DoH 1993). These events have firmly placed the responsibility of the prevention of pressure sores within the remit of acute and community Trusts with recommendations to develop a policy base (DoH 1993) and identified a monitoring role for the Health Authorities with responsibility for purchasing services (Hansard 1993).

So, despite the paucity of incidence data upon which to base targets and little evidence of the effectiveness of preventative strategies (NPRU 1992), purchaser/provider contracts during the past two years have included local agreements to establish base-line measurements and annual targets of 5-10% reduction in incidence (Leeds Health Care 1993-1994 and 1994-1995). Also in evidence are the publication of a plethora of local pressure sore

prevention/management policies, guidelines and protocols. The guidelines for the development of these policies, guidelines and protocols are described and component parts and limitations explored in the following sections.

3.3 Policy Development

The process of developing a policy base to pressure sore prevention and management is described by numerous authors within the literature (for example, Hibbs 1982, Millward 1990, Richardson 1990, Starling 1990, Barker 1992, Blaber 1993, Candler et al 1993, CSAG 1993, Livesley 1989, Simpson and Livesley 1993 and DoH 1993), with those published in latter years utilising the recommendations of the King's Fund Pressure Sore Study Group (Livesley 1989 and Simpson and Livesley 1993). Key components of the process described include:-

(a) formation of a multidisciplinary pressure sore group,
(b) collecting baseline information on the extent of the pressure sore problem, the risk profile of the surveyed population, available resources (equipment, staffing levels, skill mix and budgets) and existing policies and knowledge,
(c) interpretation of baseline information to identify need,
(d) development of multidisciplinary standards/protocols/policies for prevention/treatment,
(e) implementation of the standards/protocols.policies by securing endorsement of the proposals at all levels and addressing education and resource needs,
(f) evaluation of the elements of the implementation (Simpson and Livesley 1993 and DoH 1993).

This activity has resulted in the organization and reporting of point prevalence surveys as a method to establish baseline measures (O'Dea 1993, Starling 1990, Candler et al 1993, Hibbs 1982, Blaber 1993, Dealey 1991, Richardson

1990 and Millward 1990) and the publication at a local level of pressure area care policies/guidelines/standards for prevention and/or management. Examples are numerous but a number of common factors prevail. Typically, the policies/guidelines/standards recommend assessment of risk using a 'recognized' risk assessment tool, assessment of skin using a classification system, attention to the nutritional state of the patient, instigation of an individualised repositioning schedule, provision of a suitable support surface, 'regular' reassessment of risk/skin and evaluation of care.

The majority of recommendations relate to the care of patients at ward level but many of the policies also advocate the provision of pressure sore prevention from pre-admission to discharge and make specific reference to assessment and equipment provision within X-ray, Accident and Emergency Departments and the Operating Theatre (for example, City and Hackney Health Authority 1989, Hibbs 1988a, United Leeds Teaching Hospitals NHS Trust, Barker 1992, CSAG 1993, St. James's University Hospital NHS Trust 1993, Gloucester Health Authority 1990 and North Lincolnshire Health Authority 1991).

The implementation stage is less frequently reported. A programme of repeat point prevalence surveys are reported by some (Cullum and Clark 1992, Richardson 1990, Candler et al 1993, Dealey 1991 and 1994) with results leading to questions as to the appropriateness of such a measurement for evaluation (Cullum and Clark 1992). The implementation of the recommendations has identified a number of limitations associated with key elements of the prevention policies and problems associated with the interpretation of trends observed.

3.4 Limitations of Prevention Policies

Limitations to the key elements of the prevention policies are four fold. Firstly, an assumption is made that pressure sore prevention is cheaper than treatment

although the component procedures, guidelines and equipment products remain untested and the costs unaccounted (Clark et al 1991). Indeed, a recent study examining the costs of a prevention strategy compared to treatment only, using hypothetical figures, concluded that:-

"The cost of 'prevention' is not, in our estimates, substantially cheaper than 'treatment' and may be more expensive because:-

* in our preventative regime, the cost of preventative care per patient is a significant proportion of the cost of treatment (excluding opportunity costs of longer stay). Prevention is not a low cost or 'one shot' solution.

* the number of patients at risk is substantially larger than the number developing a pressure sore in the estimates we have used."

(Touche Ross & Co. 1993, p 5)

Secondly, limitations are associated with the accuracy of risk assessment tools and a paucity of data indicating the conversion of 'at risk' individuals to pressure sores (that is, the validity) or indeed margins of error in reliability and their effect upon validity. A review of the literature challenges their role and function and dependence upon the score in determining the care planned at an individual level (see Chapter 6, Bridel 1994, Shakespeare 1994 and Clark and Farrar 1992).

Thirdly, the specific contribution of many factors associated with pressure sore development (such as, nutritional status and incontinence) are unclear (see Chapter 5). Despite the importance attributed to these factors within prevention strategies the effectiveness of many interventions are undetermined.

Finally, there is a paucity of data which demonstrate the benefits of the wide

range of pressure relieving/reducing equipment available for use on beds, chairs, trolleys and X-ray and operating tables. It has been previously noted by Young (1992) that information regarding the effectiveness of the many pressure sore prevention aids is poor. In his survey forty-eight products were reviewed and manufacturers asked to provide information describing their effectiveness. Of the forty-eight products no evidence of effectiveness was available for twenty-four (50%), anecdotal evidence was provided for ten (21%), results of laboratory testing were provided for a further ten (21%) with only four (8%) being studied by clinical trial. Of these four only two had been subjected to a randomized clinical trial.

In relation to the Operating Department, products available for use on the operating table are limited in the U.K. to a dry polymer gel pad (Central Medical Supplies) and a liquid displacement cell mattress (Charnwood). These products both demonstrate reduced interface pressure measurements at key anatomical sites or total body areas in comparison to conventional operating table mattresses (Moore et al 1992 and Neander and Birkenfeld 1991). However, neither have been subjected to randomized clinical trial and their effectiveness in reducing or preventing pressure sore development is unknown (see Chapter 7 and Moore et al 1992).

Recommendation for and justification of their use is essentially by default and reports of the high interface measurements recorded on conventional operating table mattresses (Moore et al 1992, Neander and Birkenfeld 1991). The selection and purchase is based upon subjective appraisal and consideration of aspects such as, ease of use/cleaning/laundering, acceptability to staff and patients and cost.

With regard to monitoring methods, limitations are related to the definition of the term pressure sore and its clinical application (that is, the reliability of the measurement tools used), methodologies employed in data collection (see Chapter 4) and interpretation of results within the context of other changes in

the NHS (Cullum and Clark 1992). Incidence studies illustrate that pressure sores are essentially a transient reversible event and, therefore, question the usefulness of prevalence surveys - since their main role and purpose is as a measure of chronic, long-lived disease (see Chapter 4).

Problems in using prevalence measures as an indicator of improvements over time are illustrated by Clark and Cullum (1992) who reported an increase in prevalence from 6.8% in a 1986 survey to 14.2% in a 1989 survey despite the introduction of a prevention policy at the site (Hibbs 1988) and an increase in the availability of pressure relieving equipment from sixty-nine to one hundred and eighty-six items. The authors suggest a number of explanations could account for the results and conclude that "in the absence of data detailing prevalence measured at regular intervals (for example, weekly), it may be premature to infer that changes in the prevalence of pressure sores mark variations in the quality of nursing care" (Clark and Cullum 1992).

With regard to the use of incidence monitoring, published data to date refer mainly to specific patient groups and are of a descriptive nature. Only one study (Clark and Watts 1994) reports the incidence of pressure sores within a NHS Trust Hospital (for a one year period) and no published incidence data relating to the benefits of introducing a strategy of prevention are available.

In relation to the impact of specific interventions in an area such as the Operating Department which represents a small component of hospital stay, measurements of hospital prevalence and incidence are further limited as a method of evaluation of practice change. Benefits from the assessment and allocation of equipment during the intra-operative period require pre- and immediate post-operative skin assessments to determine the precise time of the pressure sore event. Such measures have not been employed to date by those evaluating hospital prevention strategies.

3.5 Summary

The literature illustrates the increasing interest at a National level in pressure sore prevention resulting from increasing evidence of the extent of the problem, speculation and concern regarding the cost of treatment, changes in the management culture within the Health Service and political pressure. At local (hospital) level, the increasing emphasis upon prevention has led to the publication of a plethora of policies, guidelines and protocols.

Perusal of such policies, guidelines and protocols and the literature relating to their implementation does, however, challenge a number of assumptions made and identifies a number of limitations associated with key elements of prevention policies. Little evidence exists to demonstrate that such strategies are effective in reducing pressure sore incidence and with regard to the theatre environment, many of the policies make specific reference to assessment and equipment provision despite limited information regarding likely benefits of such strategies and monitoring methods which lack sensitivity in demonstrating any effect.

Chapter 4 - The Epidemiology of Pressure Sores

4.1 Introduction

The word epidemiology is derived from the Greek meaning "studies upon people" (Farmer and Miller 1983) and is comprehensively defined by Morris as:

> The study of health and disease of populations and groups in relation to their environment and ways of living. (Morris 1975).

Specific aims of the science are to: give perspective to the range, pattern and proportion of health and disease in human populations; 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 (Morris 1975, Alderson 1983, Holland and Karhausen 1978).

Generally health service personnel have a limited picture of the natural history of disease and the health and disease status of the population they serve, since impressions gained are based upon experiences of day-to-day contacts with a succession of individual patients and their families (Donaldson and Donaldson 1985). The various uses of epidemiology all derive from the principle that whole populations or representative samples are studied, not individuals or patients.

This chapter presents details of epidemiological studies undertaken to assess the scope of the pressure sore problem. They are divided into two types - prevalence studies and incidence studies - each one detailing results in relation to the extent of the problem and information gleaned with regard to predisposing factors. First, however, the reader is introduced to the statistical terms used by epidemiologists and general considerations which must be made

in the interpretation of epidemiological studies.

4.2 Epidemiology - A Brief Overview

The science of epidemiology has traditionally been the responsibility of the medical profession (DHSS 1972), though not restricted to an identifiable group of trained individuals since many sources of data are readily available and accessible and the tools are simple and relatively cheap (Holland and Karhausen 1978). Nursing's involvement with epidemiology is gradually increasing as demands for objective data are made from the NHS management structure (DHSS 1983) and advocates of good practice management (DHSS 1986).

Epidemiological data have a basic unit of measurement - the rate. This has three components: the numerator (the total number of people who experience the event), the denominator (the total number of people in the population potentially at risk), and a specified time period during which events take place (Donaldson and Donaldson 1985).

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 structure of a population are not taken into account. Since pressure sores affect mainly hospitalised and/or severely disabled people, the crude rate is not a useful indicator and is rarely used (Barbenel et al 1977).

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 between groups of different populations and may give indicators about the natural history of the disease and the causes or predisposing factors (Donaldson and Donaldson 1985). 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.

Other common terms found are prevalence and incidence rates. 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) (Donaldson and Donaldson 1985). It is a useful indicator of the extent of chronic, long-lived disease and disability.

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 (Minotti 1978). It is a useful measure of the extent of burden created by short-lived or quickly recoverable diseases/problems.

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 (McCarthy 1982). Errors can arise in the number of identified cases (numerator error) or when the defined population size is inaccurate (denominator error). It is acknowledged that in epidemiological study "optimum conditions rarely apply, and there is always some degree of error" (Holland and Karhausen 1978). Problems encountered by epidemiologists are often out of their control since completeness of data depends on three factors: first, that every individual with a given condition actually presents with it to the health service; second, that, upon presentation, the health care professional/data recorder recognises the condition; and, third, that the condition is accurately documented/recorded.

In the epidemiological study of pressure sores it is the latter two factors which are most limiting in the determination of the true extent of the problem. Recognition problems 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 (Barbenel et al 1977, Gould

1986, Gendron 1980, Torrance 1983 and David et al 1983).

Similarly, inaccurate documentation/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 (Barbenel et al 1977, Gould 1986, Torrance 1983, Richardson 1990). In the past, inadequate understanding of the aetiology of sores and a lack of appreciation that the complicated process is exacerbated by events occurring during hospitalisation, meant that the development of a sore was taken as an indication of poor nursing care. This, in turn, produced feelings of guilt and denial amongst nurses who were then reluctant to discuss and explore the subject (Torrance 1983, Hibbs 1982 and Richardson 1990).

These aspects require consideration when appraisal of pressure sore epidemiological study is undertaken. Each study must be taken on individual merit since methods vary, as do sample sizes, time and resources. Many published studies are written by clinical nurses who have undertaken the work in the course of their normal duties, whereas other accounts are of commissioned work with teams 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 that informed interpretation precedes application to practice, planning or comparison with other research results. Within the context of the issues highlighted, the results of prevalence and incidence studies are now discussed.

4.3 Prevalence Studies

A number of point prevalence studies have been undertaken in the past 25 years (Barbenel et al 1977, David et al 1983, Barbenel et al 1980, Nyquist and Hawthorn 1987, Girvin and Griffiths-Jones 1989 and O'Dea 1993) and results are very similar despite differences in methodology, pressure sore definition, and the size of surveyed populations. A summary is provided (see Table 2).

The rates ranging from 5.3% to 18.6% all reflect the prevalence within health regions, health authorities and hospitals without or prior to the development of a pressure sore prevention policy.

Attempts to test the reliability of methods and determine the accuracy of reporting of pressure sores were made by Barbenel et al (1977) and David et al (1983). They sampled a small number of wards and researchers independently surveyed all patients individually, and compared results to the main study method of using the ward nurse in charge to provide details. The results of Barbenel et al (1977) suggested that the number of hospitalised patients with pressure sores would be underestimated in the main survey by 2%-3.5% (Barbenel et al 1977). Indeed, similar conclusions were also made by David et al (1983) who estimated an overall shortfall in reporting of approximately 2.4%.

Barbenel et al (1977) also reported major disagreements in the reporting of Grade 1 pressure sores (that is, reactive hyperaemia) amongst the seven nurses who were involved in the check survey, and as a result this grade was omitted from their analysis. O'Dea (1993) recognised the limitations of including reactive hyperaemia within her prevalence and presented results both with and without. It is unfortunate that no other reference is made to this problem by other authors. The point prevalence rates reported then provide a general guide to the occurrence of the problem within the hospital setting, but it is not a precise measure, since the extent of numerator error is not fully explored by many authors.

A period prevalence is also reported in the literature by Waterlow (1985 and 1988). She surveyed 649 patients from medical, surgical, orthopaedic, geriatric, trauma, coronary and intensive care wards whether already in-patients when the study commenced or admitted during the study period. A period prevalence of 17.1% was recorded. However, three important aspects limit the validity and reliability of these results and clearly illustrate some of the pitfalls involved in

Table 2 Summary of Point Prevalence Studies

Study	Prevalence rate %	Sample size	Grades of sore	Method	Exclusions
Barbenel et al 1977	8.8	10,751	2-4	Questionnaire	Maternity Mental def Psychiatry
Barbenel et al 1980	9.4	999	2-4	Questionnaire	Maternity Neonates Psychiatry
David et al 1983	6.6	13,409	1-4	Interview	Maternity Psychiatry
Nyquist and Hawthorn 1987	5.3	2,513	1-4	Questionnaire	Maternity Psychiatry
Girvin and Griffiths-Jones 1989	10.2	1,010	1-4	Interview and records	Maternity Paediatric Psychiatry
O'Dea 1993	18.6	3213	1-4	Observation and records	Maternity Paediatric Psychiatry

the critical review of epidemiological study. Firstly, Waterlow excluded short-stay patients (two days and under) from the survey, and in so doing changed the denominator population. Secondly, the main researcher (namely Waterlow) examined more than 90% of patients herself, and could well have introduced bias. Thirdly, the results of the check survey by Barbenel et al (1977) cast serious 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.

Other information obtained from prevalence 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. In relation to patient characteristics, various factors have been established. The majority of patients with pressure sores are over 65 years; they are more commonly seen on women than men; and many patients are immobile (bed- or chair-fast) and/or incontinent.

Interpretation of many results are limited, however, since with the exception of Barbenel et al (1977) and Waterlow (1988), the characteristics of the pressure sore positive groups are analyzed in isolation of the main (pressure sore negative) population; that is, the specific rates are not analyzed. The limitations of these methodologies are clearly explained if gender is examined. 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 crude results) about individual patient risk particularly in respect of incontinence and gender (Girvin and Griffiths-Jones 1989).

The three studies which actually detail specific rates do, however, contribute greatly to the growing body of knowledge relating to pressure sore aetiology. Barbenel et al (1977) and Waterlow (1988) presented age-specific analyses which illustrated that prevalence increases with age.

Perhaps of more interest, though, are mobility- and continence-specific rates. For example, of patients who are totally helpless and chair-fast, pressure sore prevalence is reported as 25% (Barbenel et al 1977) and 40% (Barbenel et al 1980) respectively, whereas in semi-ambulant patients only 7.1% and 6.5% have pressure sores. Similar results are detailed by Waterlow (1988) for continence/incontinence. These results also illustrate that many factors may be involved in pressure sore aetiology, and no one single cause is identifiable. For example, although 40% of totally helpless chair-fast patients had pressure sores, 60% did not (Barbenel et al 1980).

The prevalence studies do, then, 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.

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%, 97.6% and 97.1% reported by Nyquist and Hawthorn (1987), Girvin and Griffiths-Jones (1989) and David et al (1983) respectively.

More specifically sores are found on the sacrum, heels and buttocks. These areas are not adapted to weight bearing (Braden and Bergstrom 1987) and are not normally exposed to unrelieved pressure (Exton-Smith and Sherwin 1961), adding further evidence to the body of knowledge linking pressure sore development to impaired mobility.

The burden upon the health service of the severity of grade of pressure sore is not easily assessed due to the 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 (1983) superficial pressure sores account for slightly more than half of all sores reported, and full thickness sores account

for less than 20%, indicating that most pressure sores are superficial in nature.

To summarise, then, prevalence studies indicate that a large number of hospital patients have pressure sores, results ranging from 5.3% to 18.6%. The prevalence rates reported provide a general guide to the occurrence of the problem within the hospital setting but are not a precise measure, since numerator errors are apparent and denominators have different terms of reference.

Factors which may predispose to pressure sore development, such as age, mobility and incontinence are identified when pressure sore positive and pressure sore negative groups are compared. They also suggest that no one single causative factor exists. The prevalence studies have also revealed that almost all pressure sores occur below the waist (with particularly vulnerable areas being the sacrum, buttocks and heels), and slightly more than half are superficial. Having reviewed the small number of prevalence studies, incidence studies are now detailed and similarities and differences in relation to reliability and validity issues and results are explored.

4.4 Incidence Studies

Results of incidence studies vary considerably, reported rates ranging from 12% to 66% (see Table 3). Close scrutiny reveals both similarities and wide differences in incidence depending on the population samples of individual studies. For example, the three studies which explored the incidence of pressure sores amongst surgical patients of all ages record similar rates despite variations in the method used (Hicks 1970, Stotts 1988 and Kemp et al 1990). The other studies which recorded extremely high incidence rates (with the exception of Gosnell (1973), whose sample was small) sampled patients who were elderly and/or had very limited mobility (Norton et al 1962, Clarke and Kadhom 1988, Versluysen 1986 and Gebhardt 1992).

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 (Hicks 1970, Stotts 1988, Clarke and Kadhom 1988 and Versluysen 1986) but provide no further evidence of the possible link to incontinence. Attention to the composition of the denominator population of incidence studies is then important, and incidence rates reported only with reference to them.

In respect of the reliability of the data collected and the accurate determination of the numeracy population, pre-study preparation of data collectors (Stotts 1988), skin assessment verification by researchers (Kemp et al 1990) and the use of a working definition of the term pressure sore as "a break in the skin" (Norton et al 1962 and Clarke and Kadhom 1988) are described in the methodologies. Little further reference is made to the issue.

The likelihood of under case ascertainment was mentioned only by Hicks (1970), who reviewed patient records and suggested that some Grade 1 (or reactive hyperaemia) 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 (Gosnell 1973, Versluysen 1986 and Gebhardt 1992). In view of the findings of Barbenel et al (1977), where major disagreements in the reporting of reactive hyperaemia 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.

The two incidence studies which sampled a broad range of age groups reported that reactive hyperaemia and superficial breaks in the skin accounted for 97.1% (Stotts 1988) and 95.5% (Kemp et al 1990) of all sores. Prevalence studies, on the other hand, reported reactive hyperaemia and superficial breaks as

accounting for 45.4% (David et al 1983), 63.4% (Nyquist and Hawthorn 1987) and 79.5% (Girvin and Griffiths-Jones 1989) of all sores.

It is an expected find that the continued monitoring of skin undertaken to determine incidence will more accurately detect the short episodes of persistent redness experienced by patients than a once-only point prevalence inspection. In view of the magnitude of the difference observed, results suggest that for the majority, pressure sores are a short-lived event and, therefore, raise questions as to the usefulness of prevalence as a measure of the extent of the problem.

However, the absence of reports of full thickness pressure sores in all but one incidence study (Versluysen 1986) is also worthy of comment, since prevalence studies record their proportions as 13.4% (Nyquist and Hawthorn 1987), 17% (Hibbs 1982) and 22.8% (David et al 1983) 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 fully realised, and their value in determining the high cost-incurring full thickness pressure sores is limited.

In respect of the onset of pressure sores, two important aspects are realised by incidence monitoring. That is, the majority of pressure sores develop in the first two weeks following admission to hospital (Stotts 1988, Norton et al 1962 and Versluysen 1986) and the likelihood of a patient developing a pressure sore increases with length of stay (Stotts 1988 and Norton et al 1962).

In respect of the former, Norton et al (1962) provides the strongest evidence of this since all patients were followed from admission to discharge/death. In their sample of 248 patients a total of 59 patients developed pressure sores, 41 (69.5%) within the first two weeks. Other figures are also reported by Versluysen (1986), who observed that by the fifth day in hospital 83% of all patients affected by pressure sores had developed at least one lesion.

Table 3 Summary of Incidence Studies

Study	Sample size	Time scale	Speciality group	Age range	Grades of sore	Incidence %
Hicks 1970	100	?Admission-14 days	Surgery over 2 hours	10 days-85 years	1-3	13
Stotts 1988	387	Admission-3 weeks	Surgery	22-81 years	1-4	17.3*
Kemp et al 1990	125	Admission-10 days post-op	Surgery	23-84 years	1-4	12
Norton et al 1962	248	Admission-discharge	Elderly care	>65 years	2	24
Gosnell 1973	30	Admission-4 weeks	Elderly care	>65 years	2	13.3
Clark and Kadhom 1988	88	Admission-6 weeks	Orthopaedic Geriatric ITU	Chair/bed-fast	2	29.5
Versluysen 1986	100	Admission-15 days	Femoral fracture	>70 years	1-5	66*

* includes pressure sores observed on admission

In respect of length of stay, the likelihood of a patient developing a pressure sore is related to their relative risk. Stotts (1988), for example, found a linear relationship between increased length of stay and increasing pressure sore incidence, but also reported that the proportion of patients designated as high risk on a modified Norton scale (see Chapter 5) increased with length of stay. Similarly, Norton et al (1962) 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.

To summarise, then, incidence studies report wide variations in rates which are accounted for by differences in the denominator population. 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.

4.5 Summary

A review of the literature describing the epidemiology of pressure sores provides information relating to the classic uses of epidemiology: that is, 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. Comparison of incidence and prevalence studies indicates that pressure sores are both reversible and progressive in nature and valuable information is provided with regard to how many and which patients may or may not develop pressure sores. However, a clear picture of the causes is not determined and questions such as how they might be prevented remain unanswered.

Chapter 5 - The Aetiology of Pressure Sores

5.1 Introduction

That epidemiology studies provide evidence of predisposing factors but not a clear cut picture of the causes of pressure sores is consistent with results of research examining aspects of the aetiological process. Increased interest and research in the past 20 years has served to underline the complexity of the issues involved more than providing simple solutions.

Critical determinants of pressure sore development have been described as being the intensity and duration of pressure and the tolerance of the skin in its supporting structures to pressure (Braden and Bergstrom 1987) - these are inextricably linked. This chapter explores these aspects in detail and discuss and debate the common assumptions made in the pressure sore literature relating to the use of 32mmHg and 2 hour movement regime as intensity and duration threshold values.

5.2 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 due to variations in the tolerance of the skin as well as providing evidence that the nature of the applied force will have great bearing upon outcome.

The capillaries have little resistance against direct pressure and great emphasis has been placed upon the establishment of external pressure threshold levels (Bennett and Lee 1988). It is widely quoted that if external pressure is greater than mean capillary pressure (of 32mmHg) then capillary occlusion occurs and damage ensues (Barton and Barton 1981, Daniel et al 1981, Morison 1989 and

Crow 1988). Great reliance has been placed upon this hypothesis and has governed the development of pressure sore prevention equipment and policies (Bridel 1992). This hypothesis does, however, have major pitfalls.

Firstly, 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 (Claus-Walker et al 1973) and treatment with steroids for rheumatoid arthritis (Hall et al 1974). Also reported are age related changes triphasic in nature (Hall et al 1981).

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. A model of the aetiological events which probably occur when the collagen content of the skin is reduced has been developed by Krouskop (1983) - see Figure 1 (p.10). It is known that as collagen is removed from tissue a larger fraction of an externally applied load is transmitted to interstitial fluids which leaves the pressurised area (Reddy et al 1975) and, if sufficient, allows cell to cell contact and capillary bursting.

That collagen plays a key role in pressure sore aetiology is a relatively recent concept (Krouskop 1983) but it does interrelate other known variables such as the increased pressure sore risk with increasing age as noted by epidemiological study (Barbenel et al 1977) and may also explain why patients exposed to similar conditions- such as prolonged immobility -have differing outcomes (Barbenel et al 1977 and 1980). The use of the model also provides a framework which interrelates many other predisposing factors such a diet, physiological and psychological stress, steroid administration, poor oxygen saturation, lymphatic drainage and interstitial flow as discussed in Section 5.3.2.

Examination of the role of collagen does then challenge 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.

Secondly when external pressure is applied to the skin an autoregulation process allows internal capillary pressures to rise correspondingly. Landis (1930) noted that within one minute from the time of external pressure application (60mmHg) a rise in the capillary pressure occurred and stabilized at approximately 10mmHg higher than the external pressure. Similarly, Collins and Ludbrook (1967) and Bennett and Lee (1985) found that the application of an external pressure of 60mmHg did not inhibit blood flow in healthy subjects.

It appears that this autoregulation process only breaks down in those with normal circulation when external pressure exceeds diastolic pressure (Holstein et al 1979) indicating that the use of 32mmHg is conservative. Conversely, in patients with increased susceptibility, such as the elderly or severely ill where the autoregulatory mechanism is not apparent, occlusion has been reported when pressures of less than 20mmHg are applied (Bennett and Lee 1985) indicating that the use of 32mmHg is again inappropriate.

Thirdly, the situation is further complicated by variations in the application of a given load. It has become apparent that an external pressure applied in a uniform or enveloping manner has little if any long term effect upon tissue. For example, a deep sea diver may be subject to extreme (but uniform) 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 (Bliss 1993).

This was first observed and discussed by a pioneer in the field of the

biomechanical aspects of pressure sore formation - Husain (1953) - who experimented with rats. A tourniquet applied to rats' tails produced no permanent changes with the exception of those exposed to 800mmHg for six hours and the author emphasized the need to distinguish between evenly distributed pressure and localized or point pressure (Husain 1953).

Further evidence of this was later reported by Branemark (1976) in studies of the microcirculation of human skin. He found that following controlled occlusion of blood flow (using a tourniquet) for up to three hours, circulation was re-established with few signs of damage and even when occlusion was maintained for seven hours it was observed that the 'majority' of the microcirculation was re-established and maintained.

It is the effect of the application of a local or point pressure upon the skin which is then of interest in pressure sore aetiology. Present knowledge stems largely from animal testing and actual values vary enormously due to the differing animals, tissues and methods used. Despite wide variations in threshold values reported general trends emerge and are discussed in the following paragraphs.

It is widely quoted that prolonged low pressure is as hazardous as short term high pressure (Morison 1989, Braden and Bergstrom 1987, Scales 1976) and that an inverse relationship exists between the amount and duration of pressure (Kosiak 1959). However, a closer look at the experiments undertaken whilst supporting these statements also reveals that inappropriate conclusions and oversights in the interpretation of results have been made.

It is important in the review of this 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 who have failed to clearly report the clinical significance of their results.

Husain (1953), for example, whilst 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 4 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.

Table 4

Tabulated Results of Husain 1953

Pressure Intensity mmHg	Pressure Duration hours	Tissue Changes
100	2	Patchy congestion
100	6	Severe changes
600	1	Patchy congestion
600	6	Severe changes

However, the most interesting aspect of the results were that the low and high pressures over a similar time span (of one-two and six hours) produced similar tissue changes. This was completely overlooked by the author despite similar findings reported by Brooks and Duncan (1940) who concluded that the duration of pressure application was of greater importance than the degree of pressure.

A similar omission was made by Kosiak (1961) who applied pressures ranging from 35-240mmHg to the muscle of rats for periods of one, two, three and four hours and examined the tissue microscopically. 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.

So an important though essentially unrecognized 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.

The 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 pressure for short periods both cause ulceration (Kosiak 1959, Groth 1942 and Dinsdale 1973).

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 pressures of any value and time periods of any duration. Despite the limitations of the results a parabolic intensity duration curve was developed in the 1970s by Reswick and Rogers (1976) who appear to base the lower threshold value upon the main capillary pressure 32mmHg. Its use in the 1990s is not supported by the literature 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 and complicate the pressure/time/tissue damage equation altering the threshold values of the parabolic intensity duration curve (Bennett and Lee 1985 and 1988 and Gibson et al 1976). Pressure consists of the load perpendicular to the tissue's surface and shear the load parallel to the tissue's surface. It is difficult to create pressure without shear and shear without pressure.

The effect of varying the amount of shear upon human skin was reported by Bennett and Lee (1985). Using a sensor head incorporating four sensors (two pressure, one shear and one blood flow plethysmograph) they were able to determine the relationship between pressure and shear in producing blood flow occlusion. Using the palm of the hand of four healthy subjects the authors found that low shear caused occlusion within the 100-120mmHg pressure range and high shear in the 60-80mmHg pressure range. They concluded that the primary force generating mechanical occlusion is pressure but that shear plays an important contributory role and its presence cannot be ignored.

In relation to pressure sore aetiology the authors also reported other interesting data. 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 normal, elderly and paraplegic subjects. Results indicated that elderly and paraplegic subjects experienced greater shear and reduced blood flow than normal subjects at the same pressure values, providing further evidence of the individual nature of the load/response relationship.

In summary, then, research relating to the intensity and duration of pressure required for pressure sore development highlights the individual nature of the skin's response to pressure and that the problem of tissue breakdown is a multi-dimensional 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.

5.3 Tolerance of the Skin

Factors affecting tissue tolerance can be sub-divided into extrinsic and intrinsic factors (Braden and Bergstom 1987). Extrinsic factors affect tissue tolerance by impinging upon the surface of the skin and include exposure to moisture, irritants and friction. They have received little research interest and their relationship with pressure sore aetiology is not clear. 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 alter the structural constituents and perfusion of tissue. The influence of these factors are discussed.

5.3.1 Extrinsic Factors

The contribution of moisture is linked to pressure sore development in numerous aetiological accounts with particular reference to incontinence (Braden and Bergstrom 1987). The link was initially identified by early epidemiological studies such as those conducted by Barbenel et al (1977 and 1988) and Norton et al (1962) and since then reinforced by Waterlow (1988).

However, moisture whether in the form of urine, perspiration or wound drainage, does not in itself cause pressure sores (Torrance 1983). It is hypothesized that it enhances the frictional component of shearing force (Torrance 1983) or combines with by-products of laundering processes and incites chemical attack upon the skin (Alberman 1992).

In respect of incontinent patients it is suggested that other characteristics associated with patients who suffer incontinence, such as old age and reduced mobility, are the link between high pressure sore occurrence and incontinence (Torrance 1983). Despite the unclear relationship incontinence and/or skin moisture is included in the various risk assessment scales developed in the past thirty years (Norton et al 1962, Waterlow 1985 and Braden and Bergstrom 1987) which does reflect the importance attributed to this factor by 'experts' 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 60's (Bettly 1960, Lowthian 1982 and Torrance 1983) 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 (Bettly 1960 and Lowthian 1982). 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 do cause skin rash without the compounding problem of pressure (Alberman 1992 and Gunnell 1992). It is suggested that the use of plastic under sheets and skin dampness provide a combination which simulates the closed patch test technique used by dermatologists and so potentiates the irritant material present (Alberman 1992). The clinical significance of this is not, however, understood.

The evidence that friction increases the susceptibility of the skin to pressure ulceration was provided by experimentation on pigs by Dinsdale (1974). He compared the results of pressure only with pressure plus friction which were applied over 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 amongst the normal pigs. Pressure alone required a level of 290mmHg to produce ulceration, whereas pressure with friction produced ulcers at 45mmHg.

In a further experiment using an isotope clearance technique Dinsdale established that friction did not produce ulcers by an ischemic mechanism involving the generation of shear (Dinsdale 1974). 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 were observed (Dinsdale 1973). Despite no further study to validate the work of Dinsdale (1974) the importance attributed to its role is being increasingly recognized and is included in the risk assessment tool developed by Braden and Bergstrom (1987).

An interaction model involving all the extrinsic factors impinging upon the skin's surface and reducing tolerance to pressure has been developed by Alberman (1992), who hypothesized that the factors are inter-related in the hospital environment (see Figure 2). 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.

5.3.2 Intrinsic Factors

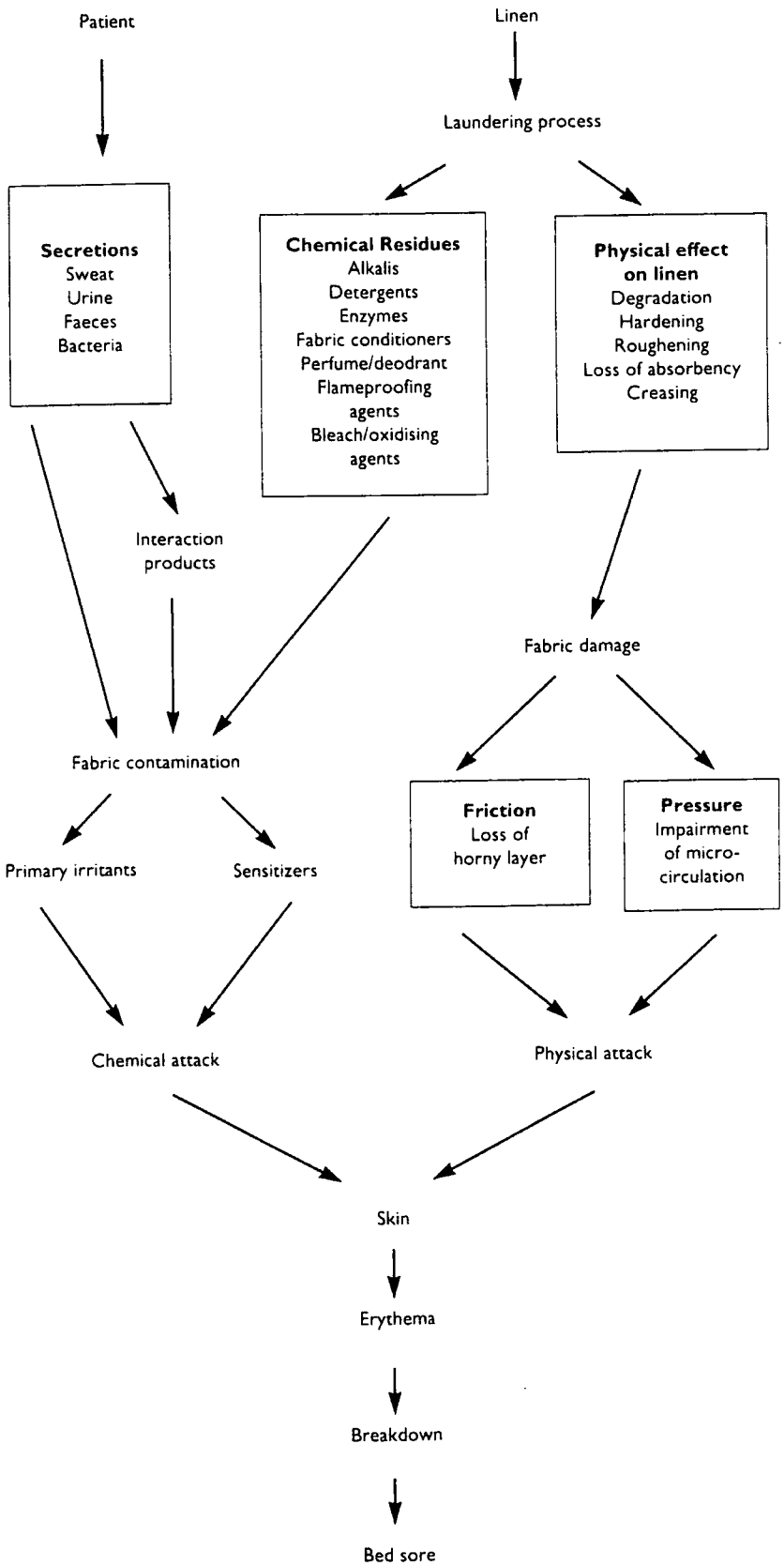
Intrinsic factors - affecting the ability of the skin and supporting structures to respond to pressure and shear forces are numerous. For the purpose of this review they are classified as factors affecting the collagen component of the skin and tissue perfusion.

As highlighted in the previous section in the review of the use of 32mmHg as a threshold value for external pressure, the role of collagen plays a key role in determining the skin's ability to tolerate pressure. 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 and their influence is explored.

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 (Hall et al 1974). It appears that such changes occur as a result of a gradual reduction in the synthesis of collagen from 20-60 years and a dramatic degradation of collagen in the 60 plus age group (Hall et al 1981). These changes have direct application to pressure risk assessment and interrelate with results from epidemiological study indicating increased risk of pressure sore development with increasing age (Barbenel et al 1977 and Waterlow 1988). It has also been shown that the administration of steroids mimics and

Figure 2

Extrinsic Factors - An Interaction Model



(Alberman 1992)

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 predominant cause is unknown but it has been observed that withdrawal results in a reversal of the changes (Hall et al 1974).

The effect of nutritional state upon the collagen content of the skin is not documented in the literature. Much research has been undertaken and shown that protein, carbohydrates, fats, vitamins and trace elements are necessary for the synthesis and maturation of collagen in wound healing (Bergstrom et al 1986, Brown 1991 and Keighley 1982) but the effect of an absence of essential nutrients upon the total collagen content of the skin has received little attention.

Nutritional research has concentrated upon nutrient profiles of patients with existing pressure sores and revealed deficiencies in albumin, vitamin C and zinc (Breslow 1991). Values are often similar, however, to deficiencies observed in pressure sore free hospitalized elderly patients (Morgan et al 1975 and Goode et al 1992) and, therefore, no conclusions can be made. It may be hypothesized that if essential nutrients are required for collagen synthesis and stability during healing then the general metabolism of collagen which undergoes a continual process of synthesis, maturation and degradation (Hall 1984) 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 et al (1973) observed an associated breakdown of collagen (by examining excretion of electrolytes in urine). Indeed, this observation was the basis of the theory later developed by Krouskop (1983) that collagen was a likely key factor in pressure sore development. The theory is supported by results reported by Bennett and Lee (1985) who determined that paraplegic subjects had a reduced resistance to external load when compared to normal

subjects and links in epidemiological data suggesting spinal cord injured patients have increased pressure sore risk (Richardson and Meyer 1981).

Tissue perfusion is affected by a number of intrinsic factors including 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. Pertinent literature is reviewed in the following paragraphs.

In relation to the effect of variations/changes of the systemic blood pressure a number of issues require clarification particularly in respect of the effect of blood pressure upon capillary blood pressure and flow. Three studies have reported that the mean systolic blood pressure (sBP) of patients who develop sores are lower than patients who do not (Mawson et al 1988, Schubert 1992 and Cullum and Clark 1992) and have suggested that reduced systolic blood pressure results in reduced tissue perfusion. Similar suggestions are made about links between diastolic pressures of less than 60mmHg and pressure sore development (Kemp et al 1990).

However, differences in recorded systolic blood pressures are not wide and overlap - with values of 120+/-21mmHg and 130+/-17mmHg sBP for patients who develop sores and 132+/-32mmHg and 140+/-20mmHg for patients who do not (Cullum and Clark 1992 and Schubert 1992 respectively). In view of problems of reliability associated with BP recordings and the overlap of recorded BP values the clinical application of these results are severely limited.

Furthermore, the suggestion that a lower sBP (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 (Guyton 1992) and it is hypothesized that capillary pressure is controlled not by systemic blood

pressure but by active vasomotion (Nicol and Webb 1955).

Local blood flow is controlled by acute and long term mechanisms. A sudden change in arterial pressure does lead to a surge or reduction in blood flow through a tissue but within minutes an autoregulatory mechanism readjusts local flow to values of approximately 3/4 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 50-250mmHg have very little effect on the rate of capillary blood pressure flow which is determined, in the main, by the release of a vasodilator substance and/or oxygen demand (Guyton 1992).

The 'critical closing pressure' is the pressure within a vessel at which it collapses completely and blood flow ceases (Lippold and Winton 1979). 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 (Lippold and Winton 1979 and Burton and Jamada 1951). In the skin and subcutaneous tissues the interplay of forces is further complicated by the presence of shear forces (Bennett and Lee 1985).

That four variables are involved explains why no individual patient 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 amongst pressure sore positive patients but the individual patient regardless of a high systolic blood pressure also develops a pressure sore 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 due to the many individual internal variables involved.

The other intrinsic factors affecting tissue perfusion further complicate the picture. Pathology which alters the oxygen exchange/demand/supply at tissue level increase 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 are somewhat hypothetical (Braden and Bergstrom 1987). However, their potential to exacerbate other pathophysiological processes justify a brief overview.

There is evidence that low serum protein concentrations (particularly hypoalbuminaemia) are associated with pressure sore development (Cullum and Clark 1992 and Holmes et al 1987) although other studies do not demonstrate the link (Kemp et al 1990 and Goode et al 1992). It is suggested that decreased serum protein affects 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 (Cullum and Clark 1992). 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 (Krouskop 1983).

In respect of oxygen demand, changes in skin and body temperature are thought to alter tissue susceptibility to ischaemic injury (Fisher et al 1978). An increase in skin temperature of one degree centigrade 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 (Holmes et al 1987 and Cullum and Clark 1992) 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 (Lamid and El Ghatit 1983) the suggestion being that short and long term side effects of smoking ultimately affect tissue oxygenation and reduce tissue tolerance (Braden and Bergstrom 1987).

The presence of factors which potentiate endothelial cell damage and thrombosis and curtail the nutrient supply completely also require consideration. Barton and Barton (1981) listed a number of potentiating factors which include among others, endotoxins, metabolic acidosis, dehydration, burns, thromboplastins (released during surgery), bacteraemia, hypoxia and blood stasis. However, the exact nature of the relationship between these factors and pressure sore development has not been established and again highlights that a complicated interplay of factors are likely to determine eventual outcome.

Many factors can affect tissue perfusion and complicate the pathophysiology of pressure sore development. The importance of the intrinsic factors in pressure sore development are not clearly defined and aspects pertaining to the critical closing pressure of blood vessels as well as the variety of other suggested potentiating factors illustrate the complicated processes involved.

In conclusion an overview of the literature relating to factors affecting skin tolerance further underlies the complex nature of the physiological processes involved in pressure sore aetiology. Key issues are summarised within the context of preceding sections and important elements of the aetiological processes highlighted.

5.4 Summary

A review of the aetiology of pressure sores highlights the individual nature of the skin's response to pressure and that the problem of tissue breakdown is a multi-dimensional process. Critical determinants are classified as: the intensity and duration of pressure, including capillary pressure and pressure application and; the tolerance of the skin and it's supporting structures to pressure, including extrinsic and extrinsic factors.

The use of mean capillary pressure (32mmHg) as a threshold value in terms of external load is challenged and disregarded. The response of the skin's capillary network to external load appears to be largely determined by the collagen content of the dermis and the autoregulatory response allowing internal capillary pressure to rise. Wide variations at an individual level are apparent.

If the external pressure is uniform then little (if any) long term effect will be noted. 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 due to the differing animals, tissues and methods used. Despite wide variations in threshold values reported general trends emerge. Firstly, 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. Secondly, an inverse relationship between the intensity and duration of pressure, that is low pressure for long periods and high pressure for short periods both cause ulceration, although, direct application in terms of threshold values is not possible since there is evidence that shear stress complicates the pressure/response relationship at an individual level.

Extrinsic factors including skin moisture, irritants and friction are inter-related. 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.

The exploration of intrinsic factors including the role of collagen and determinants of capillary closing pressure and tissue perfusion illustrates that a large number of variables are involved in determining response to external pressure and highlights the individual nature of tissue tolerance and the complex nature of pressure sore aetiology. The exact contribution of the many other factors in determining pressure sore development are largely undetermined and research provides contradictory results and/or a limited number of studies which require validation by further exploration.

Chapter 6 - Assessing Patient Risk

6.1 Introduction

A key component of all pressure sore prevention policies is the utilisation of a 'recognised' assessment tool for predicting patients most likely to develop sores. Such tools attempt to provide a basic structure to and simplify assessment of the risk of pressure sore development to the individual patient. They are also used by researchers to allow comparison of groups of patients.

Assessment tools generally examine various characteristics of a population and single out those individuals who exhibit enough of the characteristics associated with the condition so that preventative measures may be directed toward those at risk (Taylor 1988). Two basic requirements of such a tool are that measures derived from it are both reliable and valid within the context of it's application (that is, clinical practice and/or research).

Numerous risk assessment scales are reported in the literature and this chapter provides a critical review of these tools in relation to their reliability and validity and conclusions made regarding their usefulness in clinical practice and research.

6.2 Issues of Reliability

The reliability of such a measurement tool refers to the degree of consistency and accuracy with which it measures the attribute it is supposed to be measuring (Polit and Hungler 1989 and Treece and Treece 1986). There are various aspects of reliability which include stability, internal homogeneity and equivalence, which are described within the context of pressure sore risk assessment tools.

Stability is the degree to which repeated measures give the same results

(Treece and Treece 1986), but this is difficult to test in pressure sore risk assessment of hospital populations since changes in patients' at risk status are expected to occur over time as they either become well or deteriorate. It is, however, worthy of consideration within research design and validity testing with respect to the timing of the administration of risk assessment tools. For example, day and night time assessments of the same patient may yield different scores due to differences in the interpretation of elements such as mobility or incontinence. Exploration of this and the effect upon validity has not been explored or reported within the pressure sore risk calculator literature.

The internal homogeneity is the extent to which all of the subparts of a tool measure the same characteristic (Seaman 1987). However, differing scores from the subparts of pressure sore risk assessment tools are an expected outcome, and it is the total score which is of interest. Internal homogeneity testing, does therefore, not feature in the literature.

Since risk assessment tools are a 'current status' measure and a number of different personnel (either clinical or research) may employ the tool it is the equivalence of the measure which requires exploration and review. Equivalence testing determines the consistency of the tool in producing the same results when applied to the same subjects by different assessors (inter-rater) or when two parallel tools are used to assess a given subject at the same time.

Most commonly such tests are used to compute levels of agreement between raters and are presented as percent agreements and/or correlations (Taylor 1988, Polit and Hungler 1991, Bergstrom et al 1987a and b). Interpretation of such calculations is difficult, though, since there is no established standard for what the reliability should be for pressure sore risk assessment tools. Indeed, in a review of medical clinical prediction rules by Wasson et al (1985) which discussed methodological standards they merely stated that 'a report must include precise definitions of the predictive findings' and 'a report met the standard for an adequate definition of predictive findings if the authors

described a method, usually a protocol, and a standard reporting form for recording the findings'. Actual reliability testing, then, was not included in the methodological standard devised by the authors for medical clinical prediction tools (Wasson et al 1985).

The interpretation of the literature is further complicated by criticisms of the use of correlation coefficients as a measure of inter-rater reliability, since a correlation measures the strength of the relation between two scores and not the agreement (Goodwin and Prescott 1981, Bland and Altman 1986). For example, if two raters consistently scored with a one point difference the correlation would be 1 (or -1). It is suggested, therefore, that a measure of agreement such as the Kappa statistic be used (Bland and Altman 1986, Streiner and Norman 1989) but such calculations are not reported within the pressure sore risk calculator literature.

6.3 Issues of Validity

Validity refers to the degree to which an instrument measures what it is supposed to be measuring and there are a number of elements including content, face, construct, and criterion-related validity (Polit and Hungler 1989 and Treece and Treece 1986).

Content validity is the judgement by experts of the adequacy or representative nature of the content of a tool in relation to the subject under scrutiny (Treece and Treece 1986). Face validity is the judgement by experts of whether a given measurement tool appears to measure what it is supposed to measure (Seaman 1987). Both feature within the pressure sore risk assessment literature, although their use is questionable due to the high degree of subjectivity involved. Neither test allows accurate, objective determination of validity or conclusions to be drawn that a given tool is valid. Despite such limitations many pressure sore risk calculators have been introduced to clinical practice on the basis of content and face validity (see below).

Construct validity determines the extent to which a tool measures the concept or variable that it is designed to measure (Seaman 1987). The term construct frequently refers to the phenomenon of concepts that are not directly observable and testing involves either the application of two instruments which measure the same construct (and correlation of results), or, application of the known-groups technique where groups that are expected to differ on the attribute under scrutiny are administered the tool (Seaman 1987 and Polit and Hungler 1991). Determining construct validity is particularly difficult (Polit and Hungler 1991) and does not feature in the pressure sore literature which focuses upon elements of criterion related validity (including predictive and concurrent).

Criterion related validity attempts to establish the relationship between a given measurement tool and another criterion whereby the instrument is said to be valid if scores correlate highly with the criterion (Polit and Hungler 1991). Such an approach to the measurement of validity requires a reliable and valid criterion for comparison. Within the field of pressure sores the definitions of the criterion 'a pressure sore' do vary (as detailed in Chapter 2) and this complicates the external validity of results, that is, the generalisability of findings from a given study sample to larger populations (Seaman 1987).

Within criterion-related validity the distinction is made between predictive criterion-related validity and concurrent criterion-related validity. The former refers to the ability of an assessment to differentiate between those subjects who will or will not develop the defined criterion at a future date and the latter the ability to distinguish those who do or do not exhibit the given criterion in the present (Polit and Hungler 1991).

In general predictive validity of pressure sore risk assessment tools are reported in the literature where measures of pressure sore incidence are undertaken, and concurrent validity where either prevalence data is collected, or retrospective analysis is undertaken utilising the worst pressure sore risk

score (either before or after the pressure sore event).

Determination of predictive and concurrent criterion-related validity is made by a variety of calculations including correlations, sensitivity, specificity and predictive values of the positive and negative tests (Polit and Hungler 1991, Larsen 1986 and Taylor 1988). The calculations of sensitivity, specificity and predictive values of the positive and negative test all derive from a simple 9 figure table (see Table 5).

Table 5

Validity Calculation Table

	Pressure Sore Positive	Pressure Sore Negative	Total
At risk	TP	FP	TP + FP
Not at risk	FN	TN	FN + TN
Total	TP + FN	FP + TN	Total All

Key TP=True Positive FP=False Positive TN=True Negative FP=False Negative

The sensitivity of a tool is the accuracy in predicting those who develop the condition and within the context of this study may be defined as 'the percentage of those who develop pressure sores, and were so predicted by the scale' (Lilienfeld and Lilienfeld 1980, p.151) - that is, TP/TP+FN x 100.

The specificity is the accuracy in predicting those who do not develop the condition and may be defined as 'the percentage of those who do not develop pressure sores, and were so predicted by the scale' (Lilienfeld and Lilienfeld 1980, p. 151) - that is, TN/TN+FP x 100.

The predictive values of positive and negative tests have a slightly different method of calculation. The former is said to be the percentage of those at risk of pressure sore development who actually develop a pressure sore (TP/TP+FP

x 100), and the latter, the percentage of those not at risk of pressure sore development who do not develop a pressure sore (TN/FN+TN x 100).

These aspects of validity are important when an assessment tool is applied to the practical situation since over and/or under prediction of cases have implications at individual patient and budgetary levels. The purpose of assessment is that 'preventative measures be applied only to those who would develop the problem and stop resources from being wasted on those who would not' (Warner and Hall 1986).

The focus taken within the pressure sore literature is upon measures of sensitivity and specificity (Taylor 1988, Bergstrom et al 1987a), Shakespeare 1994 and Clark and Farrar 1992). However, interpretation of results is complicated by a lack of differentiation between predictive and concurrent validity and provision of preventative activities by clinical nurses within research settings which would be ethically unacceptable to withdraw (Clark and Farrar 1992).

Furthermore, the research setting and/or method may in itself generate erroneous data, for example, due to increased knowledge of observers. Validation is, therefore, not of the tool itself but the application of the tool within a specific context and validity is supported to a lesser or greater extent by evidence rather than proven (Polit and Hungler 1991). The more evidence gathered the better the support for the degree of validity.

Other considerations regarding validity relate to the reliability of a tool. Reliability and validity are not independent qualities and a tool that is unreliable cannot be valid although a tool can be reliable without being valid (Polit and Hungler 1991). Both aspects of a tool must, therefore, be considered during development and testing procedures prior to use within either clinical or research environments. It is apparent, however, within the context of pressure sore risk assessment tools that many currently in use have been

introduced to clinical practice prior to adequate validity and reliability testing.

6.4 Pressure Sore Risk Assessment Tools

The following sections detail the historical development of pressure sore risk assessment scales, describe the main tools currently in use, provide explanations as to why they have been introduced to the clinical environment prior to adequate validity and reliability testing and the recent developments within the field which are attempting to rectify the problems.

6.5 The Norton Score

The first risk assessment tool was designed thirty years ago during a study of geriatric nursing problems, and is referred to as the Norton Score (Norton et al 1962). It was devised primarily for the purpose of research in an attempt to provide a simple way of evaluating patients' general physical and mental condition so that a comparison of treatments in relation to patient outcomes could be made in conjunction with their potential risk of developing pressure sores.

The tool they created 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 physically good condition, mentally alert, ambulant, capable of full mobility and not incontinent, to a minimum of 5 for a patient who is in very bad physical state, stuporous, confined to bed, immobile and doubly incontinent.

In their survey of 250 patients they found an almost linear relationship between the initial scores (i.e., the assessment score on admission to hospital) and the incidence of pressure sores - the definition given to a pressure sore being a break in the skin surface. Following the research Norton et al stated

that the scoring system 'proved to be a reliable way of evaluating a patient's general condition and his liability to develop pressures sore' (p.224). They then stated that:-

> 'Patients with a total score of 14 or less are liable to develop pressure sores and when the score is lower than 12 the risk is very great indeed'
> (Norton et al 1962 p. 225)

It was then recommended by the authors that the scoring system was used as the basis for assessment of pressure sore risk and that calculated scores determined the frequency of nursing attention for individual patients.

At this stage in the development of nursing research design, theories and practice little attention was paid to the issue of validity and reliability. The linear relationship between scores and pressure sore development found was strong enough evidence to recommend use of the tool, in an environment which had a pressure sore incidence of 24%. The limitations of the tool were balanced by the extent of the clinical problem.

It is possible, however, to calculate measures of criterion related predictive validity using data published in the original text. Results are as follows:- sensitivity 63%, specificity 70%, positive test 39% and negative test 86%, indicating that some patients classified as not at risk did develop pressure sores, whilst others classified as at risk did not develop sores. 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' (Norton et al 1962). No actual data were published allowing conclusions to be drawn about the tool's reliability.

So, despite the occurrence of over and under prediction of patients the tool was recommended for use within pressure sore prevention initiatives (Hibbs 1982,

Torrance 1983), and was reported in the late 1980's to be the most predominant tool in use at ward level (Anthony 1987, Girvin and Griffiths-Jones 1989 and Spencely 1988).

During the same period a plethora of articles were published referring to the assessment tool -some attempting to validate it's predictive ability (Gaston 1984, Lincoln et al 1986, Goldstone and Goldstone 1982, Roberts and Goldstone 1979 and Newman and West 1981), and others detailing modifications to the scale and professing improved predictive ability (Warner and Hall 1986, Goldstone and Roberts 1980, Gosnell 1973 and Pritchard 1986).

Again, using data published within texts, validity calculations of the Norton Score can be made and are summarised in Table 6. Caution must be used in comparison of results since definitions of the term pressure sore, methods of data collection and the time Norton scores recorded (in relation to time of sore development) differ amongst the studies.

A general picture of under and over prediction of pressure sore development emerges. Sensitivity and the predictive value of the negative test illustrate under case ascertainment. Results range from 0-93% and 80-98% respectively and indicate under prediction of 7-100% of those developing sores and incorrect allocation of 2-20% of patients to the not at risk category.

Conversely, the specificity and predictive values of the positive test results which range from 36-94% and 0-53% respectively, indicate the over prediction of 6-64% of those not developing sores and incorrect allocation of 47-100% of patients to the at risk category.

Little mention is made in the literature of the reliability of the score. Of the studies detailed in Table 6 two failed to make any reference to the reliability of the scale (Newman and West 1981 and Goldstone and Goldstone 1982), one

Table 6

Validity Calculations - Norton Score (at risk score <15)

Study	Concurrent or Predictive Validity	Sample Size	Sens %	Spec %	Pred Value of +ve lest %	Pred Value of -ve lest %
Norton et al 1962	predictive	250	63	70	39	86
Roberts and Goldstone 1979	predictive	59	92	56	37	96
Newman and West 1981	predictive	88	83	63	14	98
Goldstone and Goldstone 1982	predictive	40	89	36	53	80
Gaston 1984	concurrent	262	73	69	49	87
Lincoln et al 1986	predictive	36	0	94	0	85

reported that the ward nurses involved were trained in the use of the scale but did not detail reliability (Roberts and Goldstone 1979), and another that the interrater reliability of the investigators "was tested and verified", though specific methods and results were not reported (Gaston 1984).

Only Lincoln et al (1986) explored and reported the interrater reliability of the tool in a systematic manner. On four occasions each subject (n=73) was independently assessed by two investigators by means of the Norton tool and the resulting paired scores were analyzed for absolute, 1-point and risk versus non risk percent agreement. The results ranged from 10-70%, 58-80% and 60-100% (respectively). To facilitate reaching greater 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 as the study progressed up to and including the third assessment. During the fourth assessment, however, percent agreement fell - indeed overall agreement was lower on the fourth assessment than it had been on the first.

The discussions attempting to reach consensus raised specific problems. Difficulties arose in the interpretations of the internal ratings such as fair versus poor and limited versus slightly limited, scoring of the subsection 'physical condition', and differences in opinion between the medical and surgical nurses. Even after developing standardised definitions the investigators continued to have difficulty agreeing (Lincoln et al 1986). These results do then suggest that the Norton Score is not a reliable tool.

A review of the validity and reliability of the Norton Score reveals severe limitations in its ability to assess the individual risk of developing pressure sores. At the time of its development the magnitude of the clinical problem in the original research setting balanced the limitations of the tool and it was recommended for use.

However, as further work has illustrated its inability to accurately predict patients who may or may not be at risk and in view of the potential cost of misplaced resources the use of this tool as a single indicator for the implementation of pressure sore prevention strategies is not supported by the literature, nor is its use as a tool to compare different groups of patients in relation to differences in treatment and resulting outcomes. In attempts to overcome limitations, adaptations of the scoring system have been reported and discussion of these now follows.

6.6 Adapted Versions of the Norton Score

A number of authors have attempted to adapt the Norton Score (Gosnell 1973, Goldstone and Roberts 1980, Pritchard 1986, Williams 1992, Stotts 1988,

Warner and Hall 1986 and Towey and Erland 1988). Similar problems arise in the interpretation of results in terms of validity and reliability calculations. Some are merely anecdotal descriptions of the development of a 'new and better' scoring system (Pritchard 1986 - Douglas Score, and Williams 1992 - Smedley Score) with some comparison made to the Norton Score as way of evaluation.

Other studies have reduced, increased and/or exchanged subsections within the score and reported predictive ability are summarised in Table 7 (calculations being made using data from published text). As can be seen, sample sizes vary enormously as do predictive abilities. Those that have accurately predicted patients who developed sores have seemingly done so by a general over prediction within the total sample (Gosnell 1973 and Goldstone and Roberts 1980).

Similarly, those that have good prediction of patients not at risk have done so by gross under prediction of those who actually were at risk and developed pressure sores (Warner and Hall 1986 and Stotts 1988). Another shows poor predictive ability on all aspects (Towey and Erland 1988).

In respect of the reliability of the tools used two studies made no reference to testing (Warner and Hall 1986 and Goldstone and Roberts 1980) and two studies developed mutually exclusive operational definitions for each item within the score but did not document further details of reliability testing (Gosnell 1973 and Stotts 1988).

Towey and Erland (1988) detailed calculations of the internal consistency of each subsection and described this as a reliability analysis. Reliability of the tool in terms of agreement between data collectors were not detailed, indeed no reference was made to person(s) responsible for patient assessment.

A brief review of assessment tools based upon the pioneering work of Norton

Table 7

Validity Calculations - Adapted Versions of the Norton Score

Study	Concurrent or Predictive Validity	Sample Size	Sens %	Spec %	Pred Value of +ve lest %	Pred Value of -ve lest %
Gosnell 1973	Predictive	30	100	73	36	100
Goldstone and Roberts 1980	Predictive	60	100	49	51	100
Warner and Hall 1986	unknown	334	65	89	15	96
Stotts 1986	Predictive	387	16	94	38	84
Towey and Erland 1988	Predictive	60	17	44	18	37

et al (1962) reveals inadequate testing of the reliability of the tools and errors in the over and under prediction of patients who may or may not develop pressure sores. Their use in the practical and/or research setting is, then, not supported by a review of the literature.

During the same period, however, whilst some authors concentrated upon the Norton Score and tried unsuccessfully to provide an acceptable alternative, concurrent developments emerged in 1985 with the publication of the Waterlow Card - this assessment tool is now described and discussed.

6.7 The Waterlow Score

The Waterlow Card was designed to be a practical 'aide-memoir' of the preventive aids available and treatment guide, as well as, promoting awareness of the causative factors of pressure sore development and determining risk (Waterlow 1985). The card was developed after a review of 'pressure sore' research and discussions by the author with her medical colleagues. Following the piloting of the tool after which minor adjustments were made, a survey involving 650 patients was undertaken and the revised design published (Waterlow 1985).

The scoring system incorporates six main areas of risk, including 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. In the extreme then a total score of 64 is possible (Waterlow 1985).

The author then identified different degrees of risk according to scores obtained - 10+ = at risk, 15+ = high risk and 20+ = very high risk, having determined the score of 10 as at risk during the survey and reporting that no patient with a score under 12 developed a pressure sore. However, no data were published which allow the reader to explore the validity or reliability of the tool. Details pertaining to the number of patients identified as at risk were not made, nor the actual incidence or prevalence of pressure sores noted.

In a later publication describing the same survey Waterlow reports a 17.1% period prevalence and highlights that all patients with sores were identified by the scoring system as at risk (that is, 100% sensitivity). However, a histogram of analysis by score indicates that large numbers of patients identified as at risk did not develop pressure sores (that is, poor specificity) and that 100% sensitivity was achieved by over prediction of risk (Waterlow 1988). The data

are further limited since the score was not used prospectively to predict patients who developed sores, but rather identified patients with existing damage as at risk (that is concurrent criterion related validity).

In respect of the reliability of the tool no comparison of scores was undertaken nor was accuracy of skin assessments evaluated (Waterlow 1985 and 1988). Further investigations involving the Waterlow Score have been undertaken by Dealey (1989) and Wardman (1991) who independently compared Waterlow and Norton scores. The studies are limited because both used prevalence not incidence data, but are the only available published work to date.

Using data published within the text, concurrent validity has been calculated for both Waterlow and Norton results and are detailed in Table 8. It is interesting to note that although the Waterlow Score correctly categorises 98 and 100% of patients with sores as at risk, the results suggest that many patients classified as at risk do not develop pressure sores (that is poor specificity).

In respect of the reliability of the tool, Dealey did not detail any measures taken in the collection of data from the 175 sampled patients, but did undertake a separate study involving student nurses. The learners on four wards independently assessed the same five patients using the Waterlow and Norton Scores and percent agreement of scores +/- 1 point were calculated. Norton was reported to have 70% agreement Waterlow only 60%.

In the study by Wardman (1991) 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.

Table 8

Validity Calculations - Waterlow and Norton

Study	Predictive or Concurrent Validity	Sample Size	Sens % W N	Spec % W N	Pred Value of +ve lest % W N	Pred Value of -ve lest % W N
Dealey 1989	Concurrent	175	98 88	14 26	36 37	94 81
Wardman 1991	Concurrent	32	100 80	14 82	34 66	100 69

A review of the Waterlow score card reveals that although the tool has good sensitivity this appears to result from over prediction of those at risk (that is, poor specificity). Furthermore, the tool has poor reliability, though investigation of this has not been adequately undertaken. Despite these criticisms nurses throughout the country adopted the tool (Richardson 1990, Millward 1990, Dealey 1989 and Girvin and Griffiths-Jones 1989). It's use as the basis of pressure sore prevention strategies at ward/unit level can be justified on the basis of 100% sensitivity and identification of different levels of risk and provision of guidelines for resource allocation.

However, the limitations of the tool prevent it's use in the assessment of the relative risk of pressure sore development amongst control and treatment groups in relation to and comparison of patient outcomes. In an attempt to find such a tool further exploration of the literature was undertaken and revealed an American development - the Braden Scale which is described and discussed in the next section.

6.8 The Braden Scale

The Braden Scale composed of six subscales, was developed from a conceptualisation of the aetiological factors involved in pressure sore formation

following an extensive review of the literature. Critical determinants of pressure sore development were classified as the intensity and duration of pressure and the tolerance of tissues to pressure (Braden and Bergstrom 1987).

The six subscales are derived from the critical determinants involved-mobility, activity and sensory perception reflecting the intensity and duration of pressure and skin moisture, nutritional status and friction and shear 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 critical cut off point at which patients are deemed to be at risk for developing pressure sores has been set at 16 points or less following clinical validation of the tool (Bergstrom et al 1987a). Publication of the tool followed validity and reliability studies (Bergstrom et al 1987a and Bergstrom et al 1987b).

Initial work focused upon interrater reliability of the tool amongst different grades of staff, with three comparisons reported within one article. The first compared scores obtained from a graduate student and registered nurse in a rehabilitation setting. 22 subjects were assessed and 86 pairs of observations obtained. Absolute percent agreement was 88% and +/- 1 point agreement 100%. Other 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 - problems noted relating to the literacy level and interpretation difficulties amongst both grades of staff together with a poor knowledge of the patients. The conclusion drawn was that the tool should be used by Registered Nurse care givers (Bergstrom et al 1987a).

In a further study published later in 1987, inter-rater reliability amongst adult intensive care staff was reported as being at the r=0.89 level (Pearson's product moment correlation) - details of the number of assessments made or percent agreements were not given (Bergstrom et al 1987b). As previously highlighted,

the use of correlations to assess the reliability of a tool has been criticised (Bland and Altman 1986) and interpretation limited. However, the a comparison of these results with those of the Norton and Waterlow scores is favourable toward the Braden Scale (Lincoln et al 1986 and Dealey 1989).

Following the reliability studies, the validity of the tool was explored using patients admitted to medical and surgical units. 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% 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% and specificity 90% for one unit and 100% and 64% (respectively) for the other (Bergstrom et al 1987a).

In the later publication 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 by the investigators every 48 hours for 2 weeks or until discharge from the hospital. A total of 60 patients were studied and a pressure sore incidence of 40% established. A score of 16 was 83% sensitive and 64% specific. The predictive value of the positive score was 61% and the predictive value of the negative score 85% (Bergstrom et al 1987b).

The first two published reports of the validity of the Braden Scale indicate some under and over prediction of pressure sore risk. Results range from 0-15% for under case ascertainment and 0-37% for over prediction of those at risk although these compare favourably with the Norton Score in all aspects (that is, sensitivity, specificity, and the value of both positive and negative

tests). Direct comparisons to the Waterlow Score are inappropriate since all published data reports concurrent validity. However, the clinical implications of the differences in sensitivity and specificity between Waterlow and Braden (i.e., whether it is better to over predict by many cases or under predict a few) requires further debate and investigation.

In conclusion, despite the limitations of the statistical methods used, the Braden Scale is the most reliable tool described in the nursing literature. The validity of the tool compares favourably in comparisons with the Norton and Waterlow Scores. The use of the tool in clinical practice is supported by the literature, though the implications of error in the sensitivity of the tool require further investigation.

In view of the limitations of both Norton and Waterlow in reliability of their scoring systems, and results documented by Bergstrom et al (1987a & b) for both reliability and validity of the Braden Scale, the latter is determined as the most appropriate tool for use in descriptive research as well as in the comparison of control and treatment groups in relation to risk and outcomes.

6.9 Summary

There are two basic requirements of a good assessment tool - validity and reliability.That is the tool must identify those persons it claims to identify and it must identify the same person regardless of who uses it. A critical review of pressure sore risk assessment scales has been undertaken using available validity and reliability data and illustrates the limitations of testing methodologies.

A distinction is made between their usefulness in clinical practice and research and it is suggested that the implications of error (within the clinical enviroment) in sensitivity and specificity require further investigation. Within a research context the Braden Scale is determined as the most appropriate tool

for descriptive use as well as in the comparison of control and treatment groups in relation to risk and outcomes.

Chapter 7 - Intra-operative Risk

7.1 Introduction

A review of the available literature shows that few studies on the genesis of intra-operative pressure sores exist, and the contribution of operating room practice as an aetiological factor is largely undefined. 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 aspects will be discussed in the following Chapter.

7.2 Interface Pressures and the Operating Table

That patients are exposed to external pressures far in excess of main capillary pressures is evidenced in a number of studies which have measured the interface pressures on standard hospital tables. Studies using a Gaymer mercury manometer reported sacral readings all above 56mmHg (Campbell 1989) and mean sacral readings of 46+/- 16mmHg (Stewart and Magnano 1988) in conscious subjects. Similar results were also reported by Moore et al (1992) who used an Oxford Pressure Manometer and recorded mean values of 45.7mmHg at the sacrum of volunteers lying on a conventional hospital operating table mattress.

Neander and Birkenfeld (1991) used twenty 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, in terms of age and body weight).

Perhaps of most interest are the increase in interface pressures noted by Campbell (1989) 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% higher than the pre-anaesthetic induction measures, and suggest that interface measurements using healthy conscious volunteers provide a conservative picture of pressures patients are exposed to whilst undergoing surgery.

The importance of these measurements, however, are unknown in terms of their potential to generate skin damage. The widespread use of 32mmHg as a threshold value has been challenged in Chapter 5 since it fails to account for autoregulation mechanisms and the wide variations in individual capacity to resist pressure. It is too simplistic, then, to merely 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 subsequent development of pressure sores requires further review and exploration of the literature relating to pressure sores as a post-operative complication. This now follows. The literature pertaining to events in the operating department and pressure sore development has been generated by United States researchers, with one exception - namely a study of surgically acute fractured neck of femur patients (Versluysen 1986).

7.3 Post-operative Complication of Pressure Sores

The possible association between pressure damage and events in the operating department was first suggested by Hicks in 1970 who undertook a retrospective review of medical and nursing notes and found a 13% incidence of pressure sores amongst 100 patients who had undergone surgery lasting two hours or longer. Of further note is 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 due to 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 (for example rigid corrugated surface or foam cushion), and differences in ward nursing practices. However, the possible link was made.

Further evidence began to suggest that skin damage could result from pressure exposure in the operating theatre, as reports of occipital alopecia were documented (Stewart and Magnano 1988). For example, Lawson et al (1976) found a 14% incidence of occipital alopecia in patients following cardiopulmonary by-pass, which was reduced to 1% by changing the position of the patients head every thirty minutes. Despite this, the association between events in the operating department remained of little consequence and severe cases have been reported as surgical burns (Gendron 1980 and Dobbie 1976).

Gendron (1980) noted that severe burn-like injuries were reported, but remained unexplained by either faulty electrosurgical equipment or a lapse in safe operating room practice. He began to speculate that many unexplained burns were not burns at all, and concluded that it was a lack of recognition of their true nature that guaranteed their continuance. An interest in the concept of pressure damage emerged, and an overview of the incidents revealed a number of common factors were involved. Amongst others 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 was always an area exposed to sustained pressure (for example, the sacrum).

Further reports have followed citing case studies amongst vascular surgical patients (Vermillion 1990) and high 'day of surgery' incidence rates amongst surgical acute fractured neck of femur patients (Versluysen 1986). However,

many questions remained unanswered and, in an attempt to address the limitations of the few reported studies, Kemp and associates (1990) conducted a study in the late 80's which 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 patients (12%) 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 intraoperative diastolic hypotension, and had lower preoperative Braden scores than patients who did not, none of these were statistically significant. Of particular note, however, is that 70% of pressure sores were first observed as patients were being transferred from the operating table, and Kemp et al (1990) called for further study to enable the development of a multivariate model for use as an accurate predictor of patients at risk during surgery.

The literature relating to surgical patients and the operating department does then suggest a causal relationship between events during surgery and the subsequent development of pressure sores. However, studies are scanty and with the exception of Kemp et al (1990) those available lack detail in the documentation of research design and degree of patient risk due to other factors such as mobility, activity and nutritional status. Also, with the exception of one study which reported high day of surgery incidence rates amongst surgical acute fractured neck of femur patients, the extent of the problem within the UK National Health Service is unknown. Despite a lack of data a number of preventative strategies refer to the theatre environment. These are now detailed in the following section.

7.4 Intra-operative Pressure Sore Prevention

A review of pressure sore prevention policies, guidelines and protocols identifies a number of strategies recommended for the theatre environment. These include: attention to hydration; lifting and handling (to minimise friction and shear); 'safe' positioning; pre-operative risk assessment; trolley mattress replacement or silicore fibre overlays; continuity in the use of preventative ward equipment; immediate post-operative skin assessment (allowing initiation of post-operative plan of care) and; provision of intra-operative preventative aids (City and Hackney Health Authority 1989, Hibbs 1988a, United Leeds Teaching Hospitals NHS Trust, Barker 1992, CSAG 1993, St. James's University Hospital NHS Trust 1993, Gloucester Health Authority 1990 and North Lincolnshire Health Authority 1991).

Recommendations for the intra-operative period are limited to provision of equipment designed specifically for the operating table which do not impinge upon the stability of the patient's position (and hence safety) or anaesthetic and surgical needs. The products available for use on the operating table in the U.K. include a dry polymer gel pad, replacement foam mattresses, a liquid displacement cell mattress and silicore fibre overlays.

Of the four types of product available, only two have been evaluated under laboratory conditions, one has been evaluated in the clinical environment but none have been subjected to clinical evaluation by randomized control trial. The two products tested (dry polymer gel pad and liquid displacement cell mattress) under laboratory conditions using non-anaesthetized volunteers both demonstrated reduced interface pressure measurements at key anatomical sites or total body areas in comparison to the conventional operating table mattresses used as the 'control' (Moore et al 1992 and Neander and Birkenfeld 1991). Indeed sacral readings reported for both products were below the previously disputed critical threshold value of 32mmHg.

Clinical evaluation of the gel pad was undertaken in the late 70's by Gendron (1980). He reported a prospective study involving 89 patients (age range approximately 10-89 years) undergoing various surgical procedures lasting from two to eight hours and utilising the gel pad. Of the eighty-nine patients positioned on the gel pad thirty (34%) were reported as having blanching erythema and three (3.3%) as Stage 2 - defined as 'redness, edema and induration at times with epidermal blistering or desquamation' (p 239). It was also noted that a further patient reported as having a necrotic area post-operatively did not receive the gel pad during surgery.

Interpretation of these results is difficult due to the number of limitations in the reporting of the study. Details regarding method of data collection, skin assessment schedule, actual surgical times and patient characteristics including degree of risk due to other factors such as age, blood pressure, mobility and nutritional status are not reported. Furthermore, the absence of data referring to a comparative group prevents any conclusion regarding their effectiveness in reducing or preventing pressure sore development.

It appears, therefore, that justification for the use of equipment in the prevention of intra-operative pressure sores is essentially by default and reports of high interface measurements recorded upon conventional operating table mattresses. Of the equipment reviewed, two have undergone laboratory testing but none have been subjected to randomized clinical trials and their effectiveness in reducing or preventing pressure sore development is unknown.

7.5 Summary

A review of the literature pertaining specifically to intra-operative pressure sore development does suggest a causal relationship between events during surgery and the subsequent development of pressure sores. However, studies are scanty and, with the exception of Kemp et al (1990) those available lack detail in the documentation of research design and fail to explore the

contribution of specific factors in pressure sore aetiology.

The extent of the problem with the UK National Health Service is unknown, but despite this lack of data many hospital pressure sore prevention policies recommend various strategies for the threatre environment. Preventative strategies for the intra-operative period are limited to equipment which has been demonstrated under laboratory conditions to reduce interface pressures in comparison to standard operating table mattresses but none have been subjected to randomized clinical trials and their effectiveness in reducing or preventing pressure sore development is unknown.

Section II: The Research Study

Chapter 8 - The Research Method

8.1 Introduction

This chapter provides details of the study aims, a brief outline of the main study design and a full description of the research site. The review of the literature pertaining specifically to intra-operative pressure sore development (Chapter 7) does suggest a causal relationship between events during surgery and pressure sore development, although the contribution of specific factors in pressure sore aetiology is unknown. The extent of the problem within the UK National Health Service is unknown and the benefits of specific interventions in the reduction or prevention of the problem are untested. Three general research questions are then generated following the literature review. These include:

1. What is the extent of intra-operative pressure damage to the skin within a UK National Health Service hospital setting?

2. Which key variables are associated with intra-operative pressure sore development?

3. What are the benefits of using a pressure reducing mattress on the operating table in relation to post-operative pressure sore incidence?

The focus of this study was to generate data relating to the first question detailed as this was identified as the important 'first step' in research investigating this subject area. However, also pertinent was literature relating to the use of risk assessment tools as predictors of pressure sore occurrence and their value as descriptors of research patient populations. In particular, the Braden Scale (Bergstrom et al 1987a) which reports good reliability and

validity within American settings - but has not been tested within a British hospital setting (as discussed previously in Chapter 6).

8.2 Aims of the Study

The aims of the study were then to:-

1. Assess the post-operative incidence of skin damage within a UK hospital setting.
2. Explore the reliability and validity of the Braden Scale in a British hospital setting.
3. Provide data which may justify the implementation of preventative strategies and/or further research in the field.

8.3 Design

A quantitative descriptive study design was developed to determine pre-operative prevalence and assess post-operative incidence of pressure sore development amongst a purposive sample of patients admitted for elective surgical procedures. Patients were recruited to the study according to age (=>55 years), surgery type (elective major general and vascular) and intra-operative position (supine and lithotomy). Descriptive data relating to risk, skin and peri-operative time intervals were recorded. The study design was limited by funding restrictions which dictated a 2 week preparation and 10 week data collection period within one hospital setting (enabling the researcher to combine part-time work within the operating theatre as an anaesthetic nurse and research time).

8.4 Research Setting

The research site was a 600 bedded North Eastern District General Hospital with a seven suite operating theatre which undertakes Genito-urinary,

Gynaecology, General, Orthopaedic, Vascular, Ear Nose and Throat, Plastics, and Obstetric surgery.

A traditional organisation of staff predominates and the general pattern of patient/theatre staff interaction is as follows:-

* Pre-operative visits are undertaken by Recovery Nurses. There is no weekend service, day cases are not seen and the number of patients visited depends upon afternoon and evening workload and staffing levels.

* The pre-waiting area (where patients are received from ward staff into the care (or responsibility) of theatre personnel is managed by a reception clerk and/or auxiliary.

* Anaesthetic room escort from pre-waiting is undertaken by qualified, student or auxiliary nurse (the two latter predominate). The theatre checklist is completed and usually this nurse remains with the patient until anaesthetic induction.

* Anaesthetic room personnel include the Operating Department Assistant (ODA), Anaesthetist and escort (as above). Pre-operative monitoring is commenced, intra-venous access established and anaesthetic induction undertaken.

* Qualified nursing staff within theatre provide assistance to the surgeon during operative procedures and manage the supply of necessary equipment, instruments and materials.

* Post-operative care is then managed by recovery nurses who follow up patients at ward level for postoperative pain control and support of Patient Controlled Analgesia.

As a result of this method of staff organisation patients are under the care of a group of qualified nurses whose contribution is almost entirely given over to the environment, equipment, meeting the surgeons wants and procedures such as scrubbing up and counting swabs. Indeed it is possible (and frequently happened) that patients have no interaction with a qualified nurse until entering the recovery area. Documentation of care was limited to the 'care delivered' section of the care record and would merely note that the theatre checklist is in order, venflon site, anaesthetist and type of anaesthetic, surgeon, operation performed, and sutures used. The literature reviewed suggests that this method of staff organisation is not untypical (Bevan 1989, Audit Commission 1991, Wellings 1991, and Johnson 1991).

In relation to equipment and positioning with few exceptions (orthopaedic trauma patients with unstable fractures) patients are transferred to theatre on standard trolleys with 4 inch foam mattresses, laying on a carrying canvas and folded drawer sheet. Patients are transferred onto and from the operating table using the canvas which usually remains in situ intra-operatively.

The operating tables have 4 inch foam rubber covered mattresses. A warming mattress is placed between the canvas and foam mattress when patients undergo major surgery. Intra-operative positioning is the responsibility of the ODA who with the help of auxiliary staff transfer and position the patient on the operating table. If necessary sandbags, gamgee pads and foam wedges are used to maintain the intra-operative position (as determined by the ODA and/or Anaesthetist). Such equipment is used to support access to the operative area and to protect the patient from physical trauma and/or nerve compression. To prevent the occurrence of deep vein thrombosis of the lower limbs a corrugated rubber rest is placed beneath the heels of the patients in the supine position.

Post-operatively 'major cases' are transferred immediately from the operating table to their ward bed which is ordered by the Anaesthetist, Surgeon, ODA

or qualified Theatre Nurse. Pressure sore prevention mattress provision is dependent upon the identification of need by the ward staff. Patients would usually remain on the canvas in a supine position until recovery staff have checked observations and administered analgesia. No pressure reducing aides are available to theatre or recovery personnel and no attention was given to this area of patient care.

During the study patients were positioned on the operating table according to routine practice within the research setting. The layers between the patient and the operating table were, therefore, the folded drawer sheet, carrying canvas, warming blanket and anti-static 4 inch foam mattress. All patients included in the study were transferred to their ward bed immediately post-operatively and care determined by recovery and ward nurses.

8.5 The Method

During a period of ten weeks (May - July 1992) patients were recruited to the study using the inclusion criteria as detailed in table 9. The inclusion criteria were determined by practicalities of obtaining pre-operative consent, throughput of patients within the surgical unit and literature pertaining to the characteristics of patients likely to develop pressure sores and the intensity and duration of pressure.

The study focused upon planned surgery, due to perceived problems in obtaining informed consent from acute admissions and an inability to co-ordinate data collection on a 24 hour basis (for example, recovery service Mon-Fri 8am - 9pm). Also, local infection control policy dictated minimal movement of staff between general and orthopaedic theatres. A review of the throughput of patients in orthopaedics and general theatres indicated a larger proportion of general and vascular surgery in comparison to orthopaedics - hence the decision to study the former. This was supported by the literature review pertaining to intra-operative risk (Chapter 7) which indicates that

pressure sores are found amongst patients undergoing general and vascular surgery.

Table 9

Inclusion and Exclusion Criteria

Inclusion Criteria
a. scheduled for elective general and vascular surgery;
b. aged 55 years or over on day of surgery;
c. scheduled to undergo major surgery (that is, expected time on the operating table of greater than 90 minutes);
d. the surgical procedure warrants supine or lithotomy position intra-operatively.
Exclusion criteria
a. pre-operative skin assessment reveals pressure damage.
b. patients scheduled to undergo surgery for varicose veins.

In order to sample patients with a reasonable degree of risk of pressure sore development patients aged 55 or over were included on the basis of the literature which indicates that the likelihood of pressure sore development increases with age (Barbenel et al 1977 and Waterlow 1988). Similarly, the pressure/time component, although difficult to determine at an individual level would indicate that on the standard operating table mattress (Neander and Birkenfeld 1991 and Campbell 1989) the likelihood of a patient developing a sore whilst under-going 'minor' surgery would be negligible. On the basis of theatre experience a distinction between major and minor surgery was made using an arbitrary time of under or over 90 minutes (the exception being surgery for varicose veins which is minimally invasive and considered to be minor but has lengthy operating time). The inclusion of patients undergoing surgery warranting supine and lithotomy positions was determined by literature

detailing the most common site of sores, namely, sacrum, heels and buttock areas (Barbenel et al 1977, Nyquist and Hawthorn 1987, Girvin and Griffiths-Jones 1989 and David et al 1983).

Patients eligible for the study were visited pre-operatively given a full explanation of the study and written consent obtained (as required by the Health Authority Ethics Committee).

With regard to the reliability of the Braden scale qualified nurses from 3 wards were volunteered by their respective Ward Sisters and approached by the researcher and asked to participate as data recorders for the study. Following agreement (no refusals) the researcher provided a full explanation of the study and the Braden Scale on a one to one or small group discussion basis. The nurses and the researcher were then paired and required to independently assess two patients. The timing of the assessments and selection of patients was determined by the ward nurses and undertaken during the two week preparation period.

8.6 Data collection

Data collection was co-ordinated by the researcher and undertaken by all recovery area nursing staff (qualified), designated qualified ward staff and the researcher (see below). The information collected from the patients included in the study are summarised in Table 11.

Descriptive data were collected pertaining to the general characteristics of patients with pressure sores and aetiological factors related to their development relevant to the theatre environment. These include, age and gender (Barbenel et al 1977 and Waterlow 1988), date of admission (Stotts 1988 and Norton et al 1962) and type of surgery (Gendron 1980 and Vermillion 1990) which have been identified by previous researchers as factors associated with pressure sore positive patients. Similarly aspects relating to

periods of immobility were recorded (Barbenel et al 1988, Kemp et al 1990, Brooks and Duncan 1940, Husain 1953 and Kosiak 1961) - pre-medication time (patients are confined to bed following administration of premedication), pre and post-operative mattress and cushion provision, time lifted onto and from the operating table, and pre and post-operative mobility and activity (within Braden Scale).

A skin assessment scale sensitive to reactive hyperaemia (Lewis and Grant 1925, Goldblatt 1925 and Lamb et al 1980) was chosen from the variety of classification systems reported in the literature (see Chapter 2). The Torrance assessment scale was selected and adapted to include 'no discolouration of skin' (see Table 10) - to clearly distinguish between assessment of normal skin and missing data. Skin areas assessed were limited to the sacrum, buttocks and heels (that is, those areas most commonly affected and related to supine and lithotomy positioning).

Skin assessments were recorded prior to surgery in the anaesthetic room and post-operatively in the recovery area (when the transfer canvas was removed) and daily from day one to eight. Follow up assessments continued up to 8 days post-operatively to determine the incidence of Barton and Barton Type 2 pressure sores which have a delayed presentation (Barton and Barton 1981, Gendron 1980 and Vermillion 1990).

During the two week preparation period explanations and demonstrations of the tools used in the research proforma were given to the staff involved and particular attention was given to measuring the inter-rater reliability (or equivalence) of the skin assessment tool prior to the study. Nurses involved in data collection were paired and required to assess 2 patients independently using the skin assessment tool. The nurses received a full explanation of the tool and provided with a physical demonstration of blanching reactive hyperaemia and photographic examples of all grades. A total of 18 qualified nurses (9 pairs)- including the researcher - undertook skin assessments on 5

Table 10

Skin Assessment Scale

Grade	Descriptor
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 sub-cutaneous fat
Grade 5	necrosis penetrates the deep fascia and extends to muscle

Adapted from Torrance 1983

anatomical sites of 14 patients generating a total of 70 paired assessment scores. There was 98.6% absolute agreement of grades (that is, 69/70), which equates to a Kappa statistic of 0.96 (strength of agreement = 'very good' Altman 1991) and a weighted Kappa of 0.93.

Specific responsibility for data recording was as follows. Pre-operative data including demographic information and anaesthetic room skin assessment, together with intra-operative details were recorded by the research co-ordinator. Skin areas inspected were the buttocks, sacrum and heels. Post-operative skin assessments were undertaken by recovery unit staff who graded buttock, sacrum and heel areas when the carrying canvas was removed (usually within 15 minutes of being lifted from the operating table). Follow up was continued by designated (qualified) ward staff who inspected the skin (time of day not specified) and documented mattress and seating provision daily from post-operative day 1 to 8.

In relation to the validity of the Braden Scale - calculations for the sampled population were undertaken pre-operatively by the researcher and daily by the designated ward staff so that scores were sensitive to variability of risk

Table 11

Summary of Data Recorded

Demographic Details	Date of Birth Gender Date of surgery. Ward Planned Surgery
Descriptive Data relating to Risk	Braden Score - 1 day pre-op Post-operatively - 1st-8th day Date of Admission Pre-operative Starvation Period Pre-medication Time Pre and post operative mattress and seating provision.
Skin assessment of sacrum, buttocks, and heels	Anaesthetic room Immediately post-op 1/2-1 hour post-op Post-operatively - immediately 1st-8th day
Peri-operative Time Intervals	Time lifted onto operating table Time lifted from operating table Position on operating table

resulting from major surgery and subsequent recovery. These data were then linked to skin assessment data to determine criterion-related validity.

8.6 Summary

A quantitative descriptive design was developed to assess the post-operative incidence of skin damage and to explore the reliability and validity of the

Braden Scale within a British hospital setting. Patients were recruited to the study according to age (=> 55 years), surgery type (elective major general and vascular) and intra-operative position (supine and lithotomy). Descriptive data relating to risk, skin and peri-operative time intervals were collected during a 2 week preparation and 10 week study period.

Chapter 9 - Results

9.1 Introduction

A total of 28 patients were recruited during the ten week study period. Surgery was cancelled in two cases (following anaesthetic screening) leaving a residual sample of 26 for analysis of pressure sore incidence and the validity of the Braden Scale. In relation to the reliability of the scale 10 nurses were involved in generating 18 Braden Scale assessments of 9 patients. For the purposes of the study the term pressure sore is defined as 'superficial loss of the epidermis and/or reactive hyperaemia at the same site for two consecutive days or more' (adapted from Kemp et al 1990, Bergstrom et al 1987a, Lyder 1991 and Versluysen 1986). Data analysis was performed using Arcus Pro-Stat version 3.25 for Dos.

9.2 Results - General

The sample of 26 patients comprised ten men and sixteen women with an age range of 56-87 years (mean 71.4, median 69.5). 80.7% (21/26) of patients were noted as having skin changes, on a total of 45 different skin areas during their hospital stay for varying periods of time. Using the definition of a pressure sore as 'superficial loss of the epidermis and/or reactive hyperaemia at the same site for two consecutive days or more' a pressure sore period prevalence of 56% (14/25) was determined. Of these patients 4 had superficial loss of the epidermis (period prevalence 16%-4/25) and 8 suffered persistent reactive hyperaemia at the same site for 4 or more consecutive days.

Complete data were obtained for 24 patients (two patients excluded following anaesthetic room skin assessment-see below). During the study period 19/24 patients were nursed on standard hospital 'King's Fund' mattresses. Of the five patients provided with a pressure relieving mattress or overlay, two were provided within 24 hours post-operatively and no skin changes noted. The

other three patients were provided mattresses on days three, four and five post-operatively after the development of a pressure sore. Seven of the patients were noted to sit on pillows during the post-operative period and two patients were provided with a superdown (silicore fibre 'low risk') cushion.

9.3 Pre-operative Results

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 prior to surgery (visual skin assessments were not undertaken). Actual skin assessments in the anaesthetic room, however, revealed that 46% of patients (12/26) had skin changes on a total of 22/130 skin areas observed (see Table 12). Of these, 9 were noted to have one or more areas of blanching reactive hyperaemia and three a superficial break in the skin at the sacrum. (One patient was also observed to have a general red discolouration of the skin on her foot which was attributed to vascular disease following discussion with the vascular surgeon.)

Table 12

Pre-operative Skin Changes by Description and Area

	Number of Patients	Number of Affected Areas	Heels	Buttocks	Sacrum
Blanching Hyperaemia	12	19	12	6	1
Superficial Break in Skin	3	3	-	-	3
TOTAL	12	22	12	6	4

Continued follow-up of 24 patients in the post-operative period identified that of 9 with reactive hyperaemia pre-operatively 6 had persistent reactive hyperaemia for 1-8 days. Using the definition of a pressure sore as superficial

loss of the epidermis and/or reactive hyperaemia at the same site for two consecutive days or more a pre-operative prevalence of 36% (9/25) is determined.

Table 13

Summary of Pre-operative Variables

Variable		All Patients n=26	Pre-operative pressure sore negative n=16	Pre-operative pressure sore positive n=9
Age (years)	Range Mean Median Standard Deviation	56-87 71.4 69.5 1.96	56-87 69.5 68 8.35	60-82ns* 74 76 7.69
Gender	Male Female	10 14	5 11	5ns** 4
Pre-operative Admission Period (days)	Range Mean Median Standard Deviation	1-34 5.03 1	1-7 1.75 1 1.8	1-34***** 8.55 4 11.2
Preoperative Braden Score	Range Median	14-23 23	17-23 23	14-23ns*** 21
Pre-medication Time (minutes)	Range Mean Median Standard Deviation	0-440 181.6 173.5	0-440 183.2 183 98	66-275ns* 164 145 69
Starvation Time (minutes)	Range Mean Median Standard Deviation	547-1390 804 798	547-1390 777.7 707.5 217	555-1210ns* 848.4 875 192.8

* t test **Chi-square *** Mann Whitney (two-tailed)

*****significant p=0.012 Chi-square

The extent of the pre-operative problem was an unexpected find of the study and is discussed more fully in the following chapter. However, due to the high proportion of patients affected, further analysis was undertaken to determine differences between pressure sore positive and negative groups in relation to factors such as age, gender, premedication times, starvation periods, pre-operative Braden Scores and admission dates. These are detailed in Table 13. Only pre-operative admission period demonstrated differences between groups, with a significantly longer pre-operative hospital stay amongst pressure sore positive patients (p=0.012).

Further consideration of the skin response was explored by the development of a pre-operative skin severity scale. The total number of areas affected pre-operatively were multiplied by the grade and a severity scale calculated for each patient.

For example: grade 1 x 1 area = 1
grade 1 x 2 areas = 2
grade 2 x 1 area = 2

This was then correlated (using Spearman Rho correlation) with pre-operative variables to explore relationships. The variables of age and admission were significantly associated with pre-operative skin changes (see Table 14).

Table 14

Pre-operative Skin Severity Scale Correlated to Variables

Variable	Correlation Coefficient	
Age	0.43	p=0.028
Admission	0.73	p=0.0001
Pre-operative Braden	-0.22	ns
Starvation	0.28	ns
Premedication	0.128	ns

9.4 Post-operative Results

Following pre-operative skin assessments two patients were excluded from the study (as three or more areas of skin changes were noted), leaving a residual sample of 24. The 24 comprised ten men and fourteen women with an age range of 56-87 years (mean 70.4 years, median 69 years, standard deviation 8.01). It is noteworthy that 2 patients with observed superficial skin loss during pre-operative assessments were included in the post-operative follow-up. Patients spent between 65 and 303 minutes on the operating table (mean 131 minutes, median 110 minutes, standard deviation 58.8). Surgery performed on these patients included cholecystectomy (n=12), bowel resection (n=9), arterial grafting (n=2) and miscellaneous (n=1).

Areas of skin noted during pre-operative assessment as either blanching hyperaemia and/or superficial skin loss were excluded in the post-operative analysis which refers, then, only to 'new' skin changes. A pattern of skin changes emerged from the data collected by recovery and ward staff. 71% of patients (17/24) were noted to have at least one new area of blanching hyperaemia when assessed by recovery staff. Continued follow up revealed a 12.5% pressure sore incidence (3/24) on day one post surgery which persisted for 1-5 days. None of the reactive hyperaemia (either blanching or non-blanching) first noted during the immediate post-operative skin assessment progressed to superficial loss of the epidermis. Indeed, it is of particular note that 3 patients with non-blanching skin areas immediately post-operatively had resumed normal skin colour by post-operative day 1 and suffered no subsequent superficial skin loss.

Differences between post-operative pressure sore positive and negative groups were explored for variables including age (t-test), gender (Chi-square), admission period (Mann-Whitney), starvation time (t-test), premedication time (t-test) and time on the operating table (t-test). None demonstrated significant trends. Further consideration of the skin response was explored by the

development of a post-operative skin severity scale, as above. The total number of areas affected (including those with pre-operative changes) were multiplied by the grade and a severity scale calculated for each patient. This was then correlated (using Spearman Rho correlation) with variables to explore relationships. The variables of admission, pre-operative Braden and time on the table were significantly associated with post-operative skin changes (see Table 15).

Table 15

Post-operative Skin Severity Scale Correlated to Variables

Variable	Correlation Coefficient	
Age	0.385	ns
Admission	0.53	p=0.0072
Pre-operative Braden	-0.4	p=0.047
Starvation	0.03	ns
Premedication	-0.19	ns
Time on Operating Table	0.41	p=0.043

9.5 The Reliability of the Braden Scale

A total of 10 qualified nurses (including the researcher) undertook paired Braden assessments on 9 patients. The raters undertook assessments simultaneously but 'blind' to each others scoring. Scores recorded ranged from 15-22 with 44.4% (4/9) absolute agreement of scores which equates to a Kappa statistic of 0.34 (strength of agreement = Fair, Altman 1991) and a weighted Kappa of 0.75 indicating differences in paired scores to be small.

Indeed, agreement to point 1 was 88.8% (8/9) with areas of disagreement associated with the sensory perception (4 different scores) and friction and shear (1 different score) categories. Also, using Bergstrom et al's (1987a) definition of 'at risk' as 16 or less, classification of the recorded scores to 'at

risk' and 'not at risk' illustrates 100% agreement between raters.

9.6 The Validity of the Braden Scale

Validity was determined utilising the definition of the term pressure sore as 'superficial loss of the epidermis and/or reactive hyperaemia at the same site for two consecutive days or more'. In order to calculate predictive validity analysis was undertaken only on patients who entered the study as 'pressure sore free', therefore, patients with pre-operative reactive hyperaemia persistent to day 1 post-operative were excluded. Predictive validity calculations are made, then, using a sample of n=16 of whom 5 developed a pressure sore.

Sensitivity and specificity are calculated for pre-operative Braden scores, day 1 post-operative Braden scores and the Braden score recorded on the day preceding the first observed skin changes. The results are detailed in Table 16. Sensitivity and specificity demonstrate an inverse relationship, and pre-operative scores are less informative than either post-operative day 1 and worst/pre sore scores. Both the latter demonstrate best sensitivity and specificity combinations at 19 not at risk/<19 at risk.

9.7 Summary.

Pre and post-operative assessment of skin amongst a sample of 26 surgical patients with an age range 56-87 years identified a period prevalence of 56% and post-operative incidence of 12.5%. A total of 4 patients (16%) experienced superficial skin loss and 8 patients suffered persistent reactive hyperaemia at the same site for 4 or more consecutive days. The reliability of the Braden scale was tested using 10 qualified nursing staff who assessed 9 patients and demonstrated fair agreement. The validity of the scale was calculated from a sample of 16 pressure sore free patients and the most sensitive and specific score determined as <19 at risk.

Table 16

Sensitivity and Specificity of Braden Scores

	Pre-operative Braden Score		Day 1 Post-operative Braden Score		Pre Sore and Worst Recorded Braden Score	
Threshold Score	Sens	Spec	Sens	Spec	Sens	Spec
23 not at risk <23 at risk	60%	73%	100%	0%	80%	0%
22 not at risk <22 at risk	20%	91%	100%	0%	80%	0%
21 not at risk <21 at risk	20%	91%	100%	9%	80%	9%
20 not at risk <20 at risk	20%	91%	100%	18%	80%	18%
19 not at risk <19 at risk	20%	100%	100%	36%	80%	45%
18 not at risk <18 at risk	20%	100%	60%	64%	40%	64%
17 not at risk <17 at risk	0%	100%	60%	73%	40%	73%
16 not at risk <16 at risk	-		40%	82%	20%	82%
15 not at risk <15 at risk	-		20%	82%	0%	82%
14 not at risk <14 at risk	-		0%	91%	0%	91%
13 not at risk <13 at risk	-		0%	100%	0%	100%

KEY: Sens = Sensitivity
Spec = Specificity

Chapter 10 - Discussion of Results

10.1 Introduction

The results are discussed within the context of published research, identified limitations and the aims of the study. The aims were to: assess the post-operative incidence of skin damage within a UK hospital setting; explore the reliability and validity of the Braden Scale in a British hospital setting and provide data which may justify the implementation of preventative strategies and/or further research in the field. Limitations include: absence of a pilot to test the method; inclusion of non-blanching hyperaemia and a superficial break in the skin within the same Grade definition; poorly defined exclusion criteria; the application of the definition of incidence; small sample size and; limited reliability testing.

A pilot of the study design was not planned or undertaken due to time constraints resulting from difficulties in obtaining funding. The absence of a pilot led to the researcher proceeding with the study within the confines of limitations identified relating to inclusion and exclusion criteria (see section 10.2), the skin assessment tool employed which failed to distinguish between intact and broken skin (see section 10.4) and a failure to record the duration of post-operative reactive hyperaemia in the immediate post-operative period (see section 10.4).

Unexpected difficulties also arose due to over estimation of throughput of eligible patients by the researcher due to non systematic review of operating lists. The pre-study estimate was that approximately 5 patients per week would be eligible but the reality was an average of 3 with an attrition rate of 7.5% due to surgery cancellation. The main problem identified following completion of the study and a retrospective systematic review of operating lists for a six month period was that throughput varied from week to week from none to seven eligible patients. Such issues require consideration and accurate

prediction of throughput in future studies.

10.2 General Results

Comparison of the overall pressure sore period prevalence of 56% with other results must be undertaken with caution. There is no direct comparison within the literature, with other studies of surgical patients reporting incidence. However, it is also apparent that there is confusion in the application of the term incidence, with two studies including 'on admission' pressure sores and presenting these data as incidence rather than period prevalence. These two studies report incidence rates of 17.3% and 66% (Stotts 1988 and Versluysen 1986). Differences, however, are clearly apparent in relation to the age of the research populations and the methodologies employed.

Stotts (1988) sampled 387 surgical patients with an age range 22-81 years, recorded skin assessments on every third day and did not clearly define the term pressure sore. Versluysen (1986) recruited 100 acute admission patients suffering femoral fracture with a minimum age of 70 years. The reported incidence of 66% as a crude rate does not reflect the extent of tissue damage amongst the 100 patients suffering femoral fracture. The 66 patients identified as having pressure sores shared a total of 225 lesions (Versluysen 1986). Results of this study can, therefore, be described as within expected boundaries but no direct comparison is available within the literature.

The allocation of a pressure relieving mattress or overlay to only five patients, of whom two were 'pressure sore free', whilst 9 patients with pressure damage to the skin (observed for periods in excess of 4 days) were provided with no specialist equipment is consistent with results of previous studies. For example, Girvin and Griffiths-Jones 1989 and David et al 1983 report equipment utilisation rates of only 17% and 42% for patients with pressure sores. Whether their limited use is due to poor recognition and knowledge or limited availability of equipment requires further investigation.

Of particular note, however, was a general observation during the examination of nursing records that when pressure sore development was identified as a potential problem the aim of planned care within the nursing record stated 'for skin to remain intact' and entries to the progress notes were commonly 'skin remains intact'. The majority of patients did then achieve the stated aim and reactive hyperaemia was not recognised as a noteworthy occurrence.

10.3 Pre-operative Results

The observation of a pre-operative prevalence of 36% is an unexpected finding of the study. Only Versluysen (1986) has previously reported pre-operative skin damage. She found an incidence of 27% amongst the study sample of 100 patients who were over 70 years and admitted for femoral fracture. The nature of this condition meant exposure to high risk events pre-operatively such as, time left laying following fall, time on casualty trolleys, periods of bed rest in traction and the presence of urinary incontinence amongst 71% of the study group. In view of the relatively low risk of these elective surgical patients the results compare unfavourably and require validation by further study.

Statistical analysis of variables in relation to pressure sore occurrence are, however, consistent with the literature. A relationship between length of stay (pre-operative admission period) and pressure sore occurrence has previously been demonstrated (Norton et al 1962 and Stotts 1988). The variable age did not demonstrate statistical differences between pressure sore positive and negative groups, but the skin severity scale indicated an association between age and extent of skin changes - including reactive hyperaemia. This is consistent with research relating age to pressure sore occurrence (Barbenel et al 1977, Waterlow 1988 and Kemp et al 1990) and skin collagen (Hall et al 1974 and Hall et al 1981).

Of further interest are the descriptive data relating to the variables starvation and pre-medication periods. These show no significant differences between

groups but do illustrate aspects of the general experience of patients admitted for surgery. For example, patients are confined to bed following pre-medication administration and such periods ranged from 0 to in excess of 6 hours (mean 181.6 minutes). Indeed the mean pre-medication time (and confinement to bed) was greater than the mean time on the operating table. In relation to starvation periods results are similar to those reported by Thomas (1987). A full review of the implications of the effects of starvation upon physiological processes is outside the bounds of this study but results do indicate the need for further review.

A limitation of the study design, highlighted by the unexpected observation of such extensive pre-operative skin changes amongst the study sample, was the lack of specificity of the exclusion criterion relating to skin damage. The criterion merely stated 'pre-operative skin assessment reveals pressure damage'. In order to maintain a reasonable throughput of patients despite the observation of blanching hyperaemia amongst patients the researcher then altered this to 'pre-operative skin assessment revealed pressure damage on three or more areas of skin'. This too was flawed since specification of grade for exclusion should have been determined. As a consequence continued follow-up in the post-operative period of two patients with a superficial break in the skin was undertaken.

This raised questions during the analysis of results in relation to the inclusion of patients with persistent blanching hyperaemia and the interpretation and application of the term incidence. The literature review undertaken prior to the study did not provide guidance on the inclusion or exclusion of blanching or non-blanching hyperaemia. For example, of those studies reporting incidence four studies did not specify exclusion criteria (Gebhardt 1992, Versluysen 1986, Hicks 1970 and Stotts 1988) and as previously mentioned two studies included damage present on admission as 'incidence' (Versluysen 1986 and Stotts 1988).

Clarke and Kadhom (1988) included patients 'without an existing pressure sore on admission' and defined a pressure sore as a break in the skin. The issue of blanching and non-blanching hyperaemia was not addressed or reported. Gosnell (1973) included patients with 'no pressure sores on admission' but did not provide an operational definition of the term and Kemp et al (1990) included patients who were 'pressure sore free'. Their definition for post-operative analysis included erythema which did not disappear within 30 minutes of pressure relief.

No common protocol, was then, identifiable and it is interesting to note that previous researchers have not made reference to the issue. A review of skin classification tools (Reid and Morison 1994) and publications available to the researcher after the data collection for this study was undertaken does little to clarify the issue (Bergstrom and Braden 1992, Berlowitz and Wilking 1989 and Ek et al 1991). Future study would then require careful consideration of the pathophysiological evidence to determine inclusion and/or exclusion of blanching and non-blanching hyperaemia. Also it is acknowledged that patients with an actual break in the skin (at any site) during anaesthetic room skin assessment should have been excluded from post-operative follow-up.

The extent of pre-operative skin changes and limitations of the study design with regard to the specificity of the exclusion criteria raises questions as to the calculation of incidence. Incidence is defined as the proportion of subjects who first present with a given problem during a defined period of time, in relation to the population at risk (Minotti 1978). It could be argued, therefore, that all patients with any skin changes pre-operatively should have been excluded from the post-operative calculation of incidence.

However, the study results reflect previous methodologies employed by researchers in the field who include patients with pressure sores on admission within the reported pressure sore incidence of the study populations (Stotts 1988 and Versluysen 1986). Also, the original aim of the study was to assess

post-operative incidence of skin damage in order to explore the extent of intra-operative pressure damage and the researcher has clearly stated the method of calculation and presented results as 'new areas affected'.

The unexpected finding of a pre-operative prevalence of 36% indicates the need for further investigation of the pre-operative period, highlighted a limitation of the study design in the lack of specificity of the exclusion criteria relating to skin damage and identified insufficient debate or consideration within the literature of the inclusion of reactive hyperaemia.

10.4 Post-operative Results

Post-operative results illustrate a pattern of skin change which is supported by the literature relating to tissue tolerance, interface pressures and post-operative incidence rates.

The extent of immediate post-operative skin changes observed in this study have no direct comparison, since other studies have not reported immediate post-operative reactive hyperaemia unless it has persisted to day one post surgery (Versluysen 1986 and Kemp et al 1990). A total of 17 (71%) patients were noted to have at least one new area of reactive hyperaemia during the immediate post-operative period. The results have limited application due to the small sample size but are supported by evidence relating to the intensity and duration of pressure and interface pressure generated by standard operating table mattresses (Neander and Birkenfeld 1991 and Campbell 1989). In view of the large number of patients affected the results indicate and justify further exploration.

The 'theatre generated' 12.5% incidence of new skin changes observed is similar to those reported by Hicks 1970 (13%), Lawson et al 1976 (14%), Versluysen 1986 (39%) and Kemp et al 1990 (12%). Differences between the studies do exist, for example, Versluysen's elderly acute population, Kemp et

al 1990 and Stotts 1988 in relation to age range (23-84 years and 22-81 years respectively) and elements of methodologies (for example, skin assessments performed every third day), but, post-operative incidence of pressure sores lies within expected boundaries.

No significant differences between pressure sore positive and negative groups were determined, a result echoed by previous research (Kemp et al 1990) indicating perhaps the complexity of the aetiological processes (that is, no single prognostic factor). A more sensitive measure, the post-operative skin severity scale does suggest relationships between skin changes observed during the immediate post-operative period and the variables admission date, pre-operative Braden scores and time on the table.

Length of stay has previously been discussed (see section 10.3), and the association with pre-operative Braden scores and time on the table are consistent with the results of Kemp et al (1990) who found no statistical differences between pressure sore positive and negative groups but did determine, by discriminant analysis of results, a predictive model which included the variables: extracorporeal circulation; age; time on the table; pre-operative Braden scores; serum albumin and; total protein. The results of this study, then, have limited application due to sample size and pressure sore definition but do reflect previous research in the field.

A limitation of the skin assessment tool was identified in a review of the immediate post-operative data and raised questions regarding the assumption generated by a review of the pathophysiology literature (in Chapter 5), that non-blanching hyperaemia indicates micro-circulatory disruption and a precursor to superficial skin loss. Of the seventeen patients who were recorded by recovery staff as exhibiting an area of reactive hyperaemia in the immediate post-operative period three were rated as a Grade 2. All of these areas had resumed normal colour by post-operative day one and no subsequent delayed superficial skin loss observed. These observations support literature more

recently reviewed which indicates the need to distinguish between intact and broken skin within a staging classification (Lyder 1991).

Also, general observations of patients during the post-operative period and reconsideration of the literature identified a further limitation relating to the skin assessment schedule. It is important to distinguish between a 'normal' reactive hyperaemic response of short duration (5-10 minutes) and evidence of prolonged capillary occlusion. The reactive hyperaemic response has been shown to be proportional to the duration of the occlusion and generally lasts 1/2 to 3/4 of the occlusion time (Lewis and Grant 1925, Goldblatt 1925 and Lamb et al 1980). A second post-operative skin assessment in the recovery area 1/2-1 hour following the initial assessment would have improved validity in the determination of post-operative incidence where the definition of the term pressure sore included persistent reactive hyperaemia.

The results of the study, although limited, generate knowledge relating to the natural history of pressure sore development. The theatre generated incidence (12.5%) accounted for less than one quarter of the period prevalence (56%) and neither the blanching or non-blanching areas noted post-operatively led to superficial skin loss. It could be hypothesised, therefore, that a one off exposure to pressure is unlikely to generate an actual break in the skin. This view is supported by the results of Versluysen (1986) and Gebhardt (1992) which suggest that pressure sore development results from repetitive and/or continuous exposure to pressure and friction.

10.5 The Reliability of the Braden Scale

Analysis of the 9 paired assessments of patients using the Braden Score indicates only 'fair' agreement (as defined by Altman 1991) of actual total scores (Kappa= 0.34), although, only one pair of raters were more than one point different as reflected by the weighted Kappa of 0.75. In terms of percent agreement, these were 44.4% absolute agreement and 88.8% 1 point

agreement. Of particular importance, however, within the clinical environment are the implications of differences in scores to the identification of 'at risk'. In this instance, using Bergstrom et al's (1987a) definition of 'at risk' as 16 or less, classification of the recorded scores to 'at risk' and 'not at risk' achieved 100% agreement between raters. Clearly, then, if the validity of the tool is good rater differences are not clinically important.

In comparison results reported by Bergstrom et al (1987a) are similar for qualified and graduate students. They reported 88% absolute agreement, and 100% agreement to 1 point. In relation previous work exploring the reliability of the Norton Score and Waterlow Card results are favourable. Dealey (1989) reported 1 point agreement of 70% for the Norton Score and 60% for the Waterlow Scale amongst student nurses. In a study by Lincoln et al (1986) inter-rater reliability was measured on four occasions for each subject (n=73) and results indicated wide variation in agreement. Absolute agreement ranged from 10-70%, 1 point agreement 58-80% and risk versus no risk 60-100%.

However, limitations regarding sample size and methodology require consideration in the interpretation of results. Guidance within the literature regarding sample size for reliability testing of clinical prediction tools is not provided within key references (Altman 1991, Wasson et al 1985, Goodwin and Prescott 1981, Donner and Eliasziw 1987, Bland and Altman 1986 and Fielding and Foster 1987). Two published accounts of reliability (Donner and Eliasziw 1987 and Streiner and Norman 1989) do detail calculation methods in relation to reliability coefficients by analysis of variance techniques which are measures of association rather than measures of agreement. However, none cite methods or discuss sample size for tests of agreement. As a consequence interpretation of results lacks guidance.

The Kappa statistic involves calculating the number of agreements expected by chance and compares this to actual agreement. It is suggested that any value below 0.5 will indicate poor agreement but 'the degree of acceptable agreement

must depend upon circumstance' (Altman 1991 p.409), 'it is important to show the raw data' (Altman 1991 p.409) and 'statistics cannot provide a simple substitute for clinical judgement'(Altman 1991 p.409). Given that both the raw data and weighted kappa indicate good agreement, and the classification to 'at risk' and 'not at risk' demonstrated 100% agreement then it can be concluded that reliability was good within the qualified nurses sampled but that results are not generalisable.

Also assumptions cannot be made with regard to changes in the reliability over time. All paired assessments using the Braden Scale were undertaken within seven days following one to one and/or small group education by the researcher, highlighting issues relating to key prognostic factors and interpretation of components of the scale. In considering the use of the scale within either a research or practice setting further testing would have been valuable to detect changes in reliability over time.

Of further consideration in reliability are sampling methods and patient characteristics. For example, if all patients are clearly at no pressure sore risk and all raters classify patients with 'top' scores then the reliability of the tool may appear good. It is essential, therefore, to gain a representative sample. This aspect of reliability testing was not considered during study design and nurses involved merely instructed to select any patients from their ward. Despite this Braden scores recorded ranged from 15 to 22 (median 17).

10.6 The Validity of the Braden Scale

The results of validity calculations of the Braden Scale within this small sample, provide limited clinical application. Interpretation of the results is difficult due to the small sample size (n=16), but results reflect those of Clark and Farrer who determined that an inverse relationship exists between sensitivity and specificity (Clark and Farrer 1992) and recommendations by Bergstrom et al (1987a) that each centre test validity within their own patient

population.

The results indicate that of least value were pre-operative Braden Scores despite previous results describing a statistically significant correlation between post-operative skin severity scale and pre-operative Braden scores for all patients (n=24) (see section 9.4 and 10.4). Calculations, however, are distinctly different with the correlation calculation incorporating all reactive hyperaemia (including transient) and representing concurrent validity as opposed to predictive validity.

The post-operative day 1 and day pre sore/worst recorded indicate the same optimum threshold score of 19 (sensitivity 100% and 80% - specificity 36% and 45%). The clinical application of these results is not, however, supported due to the small sample size and implications of the poor specificity (for example, unnecessary allocation of resources). Furthermore, the observation of skin changes amongst the study sample challenge and question the clinical application of such tools within the practice setting.

It has been suggested by Crow and Clark (1990) that a distinction is made between predicting risk and assessing the nature of the risk. The former they suggest requires indicators predicting their development and the latter evaluation of the response of the skin to load. This is supported by the results of this study whereby 8 patients suffered persistent reactive hyperaemia for 4 or more consecutive with Braden scores ranging from 14 to 18. This raises the question 'if the patient has a red area does it matter what their score is?'

Additional review of the literature further challenges the role and function of risk calculators which identifies that their components have been determined by either a review of the literature relating to pressure sores and/or on the basis of clinical experience and judgement (Bridel 1994). Key prognostic factors have not yet been determined and the role of the component parts of the various assessment tools in relation to pressure sore aetiology and relative

risk are not quantified (Bridel 1994).

Research exploring such basic questions are few in number (Bergstrom and Braden 1992, Ek et al 1991 and Batson et al 1993) and the sampling of specific patient groups precludes generalisation of results. A reflection of the need to identify key prognostic factors are the variety of diversity of the component parts within risk assessment tools. A tabulation of 5 calculators listed 13 different variables of which only one (mobility) was common to all (Flanagan 1993). Limitations are also apparent in a failure to include experiential variables such as nursing practice (for example, frequency of turns) within research methodologies which may in fact impact as the most important variable determining the likelihood of a patient developing a pressure sore.

Furthermore, there is no scientific basis to the scores allocated to either elements within individual risk factors or between different risk factors. The weighting given to elements within each risk factor are simple ordinal scales (with the exception of the Waterlow Card). Differences between factors are not identified and the use of simple ordinal scales within individual categories is transposed to all categories (Bridel 1994). There are then fundamental limitations to the risk calculators detailed in the current literature (including the Braden Scale) - identification of key prognostic factors and the weighting both within and between each factor. This prompted review of their role and usefulness in relation to both clinical practice and research.

Potential clinical functions include:-

a) raising awareness of risk factors at a clinical level;
b) providing structure to patient assessment;
c) aiding rationale allocation of limited resources (such as mattresses);
d) initiating preventative care and;
e) interpreting audit data within a clinical setting (Cullum et al 1995).

Indeed, risk assessment using a 'recognised' tool is the cornerstone of pressure area care policies/guidelines/standards, despite no published evidence that their utilisation reduces pressure sore incidence (Cullum et al 1995).

Potential research functions include:-

a) descriptors of the sample population;
b) indicators of risk allowing comparison of patients randomised to different arms of trials;
c) comparison of results with other studies and;
d) informed/meaningful application of the results within clinical practice;

Given their potential role, future research should explore the development of tools utilising regression models to choose and weight the factors which best predict the development of a sore (Bridel 1994, Bridel 1995, Cullum et al 1995 and Wasson et al 1985), as opposed to continuation of validity testing of existing tools. That is, the study results and further review of the literature published after study design and data collection does not support further exploration of the Braden Scale.

Summary

A discussion of the results within the context of published research, identified limitations and the aims of the study, highlights a number of issues relating to the methodology adopted and lack of clarity within the literature. The extent of pre-operative skin damage was an unexpected finding and indicates the need for further investigation of the pre-operative period.

The results and the wider literature suggest that a one-off exposure to pressure is unlikely to generate an actual break in the skin and that the study of one particular aspect of the patient's experience may be limited. This is also born out in considering general issues relating to the validity of risk calculators and

the need to identify key prognostic factors and inclusion of experiential variables in prognostic models.

The Braden Scale demonstrated good reliability within a clinical setting but testing was limited and results not generalisable. Of particular note is the lack of direction within the literature in relation to sample size calculations for the Kappa Statistic. Similarly, the validity of the Scale was tested on a small sample and results not generalisable. However, the limitations of such tools were highlighted, led to further review of the literature and the debate may inform future research.

Chapter 11 - Conclusions and Recommendations

11.1 Introduction

A review of the literature pertaining to pressure sore pathophysiology, epidemiology and risk assessment tools, and this research study involving pre and post-operative skin and risk assessment of surgical patients has challenged a number of commonly used assumptions, generated interesting data relating to the general experience of patients and identified limitations and lack of clarity within the literature. A number of the issues raised are applicable to both practice and future research.

11.2 Implications for Practice

The results of the research study, although small, identified a large proportion of patients (12/26) with either a break in the skin or persistent redness at the same site for 4 or more days. The extent of the problem justifies a review of pressure sore prevention practice, documentation of care and equipment provision within the research setting.

The review of the pathophysiology, epidemiology literature and aetiology literature of pressure sores indicates particular patient characteristics associated with pressure sore development (for example, increasing age and immobility), but highlights the individual nature of the skin's response to pressure and that the problem of tissue breakdown is a multi-dimensional process. General threshold values for either intensity or duration of pressure are not definable, but reactive hyperaemia is the usual physiological response observed where capillary occlusion has occurred. This response is proportional to the duration of capillary occlusion and generally lasts half to three quarters of the occlusion time. It is recommended, therefore, that traditional practice and language, such as, '2 hourly turns' and 'skin intact' is inappropriate and that individualised preventative care is delivered and evaluated by using the skin response

(reactive hyperaemia) as an objective measure.

The review of the risk assessment tool literature and discussion of results highlights limitations of the tools available resulting from their development methodologies. A number of advantages are associated with their use at a clinical level. These include: raising awareness of risk factors; providing structure to patient assessment; aide to allocation of limited resources and; prompt the initiation of preventative care. Indeed the perceived clinical advantages reflect their widespread use within pressure sore prevention strategies, where often the risk assessment tool total score is linked to a specific prescription for equipment or intervention. In view of the limitations of these tools, however, it is recommended that risk assessment tools are used only in association with skin assessment and evaluation, that practitioners are provided with an understanding of the limitations of such tools and the total score informs but does not prescribe care.

11.3 Implications for Research

This study has raised four aspects of pressure sore development requiring academic debate and research. These include: further exploration of pressure sore occurrence within surgical populations to determine the true extent of the problem; consideration of nursing practice and ritual in relation to pre-operative and pressure area care; prognostic research and development of risk assessment tools using regression models; and differentiation between blanching and non-blanching reactive hyperaemia within inclusion and exclusion criteria and the practical application of the definition of incidence.

List of References

AHCPR (1992) Pressure Ulcers in Adults: Prediction and Prevention. Clinical Practice Guideline No.3. AHCPR Publication No.92-0047. Rockville MD.

Alberman, K. (1992) Is there any connection between laundering and the development of pressure sores? Journal of Tissue Viability, Vol. 2 (2) 55-56.

Alderson, M. (1983) An Introduction to Epidemiology. (2nd Edition) Macmillan Press, London.

Altman, D. (1991) Practical Statistics for Medical Research. Chapman and Hall, London.

Anthony, D. (1987) Are you in the dark? Nursing Times, Vol. 83 (34) 27-30.

Audit Commission (1991) NHS Occasional Paper No. 1 - Value for money on the NHS - Sterile Services. HMSO, London.

Barbenel, J.C., Jordan, M.M., Nicol, S.M. and Clark, M.O. (1977) Incidence of pressure sores in the Greater Glasgow Health Board Area. The Lancet, Vol. 2, 548-550.

Barbenel, J.C., Jordan, M.M. and Nicol, S.M. (1980) Major pressure of sores. Health and Social Services Journal, Vol. 90 (2) 1344-1345.

Barker, Z. (1992) Pressure sore prevention - making a strategy work: a multidisciplinary, holistic approach. In: Harding, K.G., Leaper, D.L. and Turner, T.D. (Eds.) 1st European Conference on Advances in Wound Management. Macmillan Mags., London. p.168-169.

Barton, A. and Barton, M. (1981) The Management and Prevention of Pressure

Sores. Faber, London.

Batson S., Adam S., Hall G. and Quirke S. (1993) The development of a pressure area scoring system for critically ill patients: a pilot study. Intensive and Critical Care Nursing Vol. 9 (3) 146-151

Bennett, L. and Lee, B.Y. (1985) Pressure versus Shear in Pressure Sore Causation. Chap.3 In: Lee, B.Y. Chronic Ulcers of the Skin. McGraw Hill, New York.

Bennett, L. and Lee, B.K. (1988) Vertical shear existence in animal pressure threshold experiments. Decubitus, Vol. 1 (1) 18-24.

Bergstrom, N. and Braden, B. J. (1992) A prospective study of pressure sore risk among institutionalised elderly. Journal of the American Geriatrics Society, Vol. 40 (8) 747-758.

Bergstrom, N., Braden, B.J., Lagussa, A. & Holman, V. (1987a) The Braden Scale for predicting pressure sore risk. Nursing Research, Vol. 36, 205-210.

Bergstrom, N., Denuth, P.J. and Braden, B.J. (1987b) A clinical trial of the Braden Scale for predicting pressure sore risk. Nursing Clinics of North America, Vol 22 (2) 417-428.

Bergstrom, N., Braden, B., Krall, K. and Brandt, G. (1986) Adequacy of descriptive scales for reporting diet intake in institutionalized elderly. Journal of Nutrition for the Elderly, Vol. 6 (1) 3-16.

Berlowitz, D.R. and Wilking, S. Van B. (1989) Risk factors for pressure sores: a comparison of cross-sectional and cohort-derived data. Journal of American Geriatrics Society Vol. 37 (11) 1043-1050.

Bettly, F.R. (1960) Some effects of soap on the skin. British Medical Journal, Vol. 1, 1675-1679.

Bevan, P.G. (1989) The Management and Utilizations of Operating Departments. NHS Management Executive VFM Unit, HMSO, London.

Blaber, C. (1993) Centred on excellence. Nursing Times, Vol. 89 (49) 61, 64 & 66.

Bland, J.M. and Altman, D.G. (1986) Statistical methods for assessing agreement between two methods of clinical measurement. The Lancet, Vol. 1, 307-310.

Bliss, M. (1993) Aetiology of pressure sores. Reviews in Clinical Gerontology, Vol. 3, 379-397.

Braden, B.J. and Bergstrom, N. (1987) A conceptual schema for the study of the etiology of pressure sores. Rehabilitation Nurse, Vol. 12 (1) 8-16.

Braden, B.J. and Bergstrom, N. (1989) Clinical utility of the Braden Scale for predicting pressure sore risk. Decubitus, Vol. 2 (3) 44-46 & 50-51.

Branemark, P.I. (1976) Microvascular Function at Reduced Flow Rates. In: Kenedi, R.M., Cowden, J.M. and Scales, J.T. Bedsore Biomechanics. University Park Press, Maryland. p.63-68

Breslow, R. (1991) Nutritional status and dietary intake of patients with pressure ulcers: review of research literature 1943-1989. Decubitus, Vol. 4 (1) 16-21.

Bridel, J. (1992) Pressure sores and intra-operative risk. Nursing Standard, Vol. 7 (5) 28-31.

Bridel, J. (1994) Risk assessment. Journal of Tissue Viability, Vol. 4 (3) 84-85.

Bridel, J. (1995) Interpreting pressure sore data. Nursing Standard, Vol. 9 (19) 52-54.

Brooks, B. and Duncan, G.W. (1940) Effects of pressure on tissues. Archives of Surgery, Vol. 40, 696-709.

Brown, K. (1991) The role of nutrition in pressure area care. Journal of Tissue Viability Vol. 1 (3) 63-64.

Burton, A.C. and Jamada, S. (1951) Relation between blood pressure and flow in the human forearm. Journal of Applied Physiology, Vol. 4, 329.

Campbell, K. (1989) Pressure point measures in the operating room. Journal of Enterostomal Therapy, Vol. 16 (3) 119-124.

Candler, S., Clifford, I. and Starling, M. (1993) A new policy of prevention. Nursing Times, Vol. 89, (42) 76-80.

City and Hackney Health Authority (1989) Strategy for Fractured Neck of Femur Patients. City and Hackney Health Authority, London.

Clark, M. and Cullum, N. (1992) Matching patient need for pressure sore prevention with the supply of pressure redistributing mattresses. Journal of Advanced Nursing, Vol. 17, 310-316.

Clark, M., Cullum, N., Crow, R.A., Chapman, R.G., Farrer, S. and Rabland, L.B. (1991) Prevention of Pressure Sores: Aspects of the identification of patient vulnerability and the effectiveness of preventative interventions. Final Report to the Department of Health: Nursing Practice Research Unit.

Clark, M. and Farrer, S. (1992) Comparison of pressure sore risk calculators. In: Harding, K.G., Leaper, D.L. and Turner, T.D. (Eds.) 1st European Conference on Advances in Wound Management. Macmillan Mags., London. p.158-162.

Clark, M. and Watts, S. (1994) The Incidence of Pressure Sores within a National Health Service Trust Hospital during 1991. Journal of Advanced Nursing, Vol. 20, 33-36.

Clarke, M. and Kadhom, H.M. (1988) The nursing prevention of pressure sores in hospital and community patients. Journal of Advanced Nursing, Vol. 13, 365-373.

Claus-Walker, J. Campos, R.J., Carter, R.E. and Chapman, M. (1973) Electrolytes in urinary calculi and urine of patients with spinal cord injuries. Archives of Physical Medicine and Rehabilitation, Vol. 54, 109-114.

Clough, N.P. (1994) The cost of pressure area management in an intensive care unit. Journal of Wound Care, Vol. 3 (1) 33-35.

Collins, G.H. and Ludbrook, J. (1967) Behaviour of vascular beds in the human upper limb at low perfusion pressure. Circulation Research, Vol. 21, 319-325.

Crow, R. (1988) The challenge of pressure sores. Nursing Times, Vol. 84 (38) 68, 71 & 73.

Crow R. A. and Clark M. (1990) Current Management for the Prevention of Pressure Sores. Chapter 5 in Bader D. L. (Ed.) Pressure Sores - Clinical Practice and Scientific Approach
Macmillan Press, London.

CSAG (1993) Prevention and Treatment of Pressure Sores: Guidelines for Good Practice. Clinical Standards Advisory Group (Chairman: June Clark), Health Research Centre. Middlesex University.

Cubbin, B. and Jackson, C. (1991) Trial of a pressure area risk calculator for intensive therapy patients. Intensive Care Nursing, Vol. 7 (1) 40-44.

Cullum, N. and Clark, M. (1992) Intrinsic factors associated with pressure sores in elderly people. Journal of Advanced Nursing, Vol. 17, 427-431.

Cullum, N., Deeks, J., Fletcher, A. et al (1995) 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 Vol.2 (1)

Daniel, R.K., Priest, D.L. and Wheatley, D.C. (1981) Etiologic factors in pressure sores; an experimental model. Archives of Physical Medicine and Rehabilitation, Vol. 62, 492-498.

David, J.A., Chapman, R.G., Chapman, E.J. and Lockett, B. (1983) An Investigation of the Current Methods used in Nursing for the Care of Patients with Established Pressure Sores. Northwick Park: Nursing Research Unit, U.K.

Dealey, C. (1989) Risk assessment of pressure sores: a comparative study of Norton and Waterlow scores. Nursing Standard, Vol.3 (28) Supplement, 11-12.

Dealey, C. (1991) The size of the pressure sore problem in a teaching hospital. Journal of Advanced Nursing, Vol. 16, 663-670.

Dealey, C. (1994) Monitoring the pressure sore problem in a teaching hospital. Journal of Advanced Nursing, Vol. 20, 652-659.

DHSS (1972) Management Arrangements for the Reorganized NHS HMSO, London.

DHSS (1983) NHS. Management Enquiry Report (Chairman: Griffiths). HMSO, London.

DHSS (1986) Neighbourhood Nursing: A Focus for Care: Report of the Community Nursing Review Team (Chairman: Cumberlege). HMSO, London.

Dinsdale, S.M. (1973) Decubitus ulcers in swine: light and electron microscopic study of pathogenesis. Archives of Physical Medicine and Rehabilitation, Vol. 54, 51-56.

Dinsdale, S.M. (1974) Decubitus ulcers: role of pressure and friction in causation. Archives of Physical Medicine and Rehabilitation, Vol. 55, 147-152.

Dobbie, A.K. (1976) Session D Discussion. In: Kenedi, R.M., Cowden, J.M. and Scales, J.T. (Eds.) Bedsore Biomechanics. University Park Press, Maryland. p.249-258

DoH (1991) The Health of the Nation: A Consultative Document for Health in England. HMSO, London.

DoH (1992) The Health of the Nation: A Strategy for Health in England. HMSO, London.

DoH (1993) Pressure Sores - A Key Quality Indicator: A Guide for NHS Purchasers and Providers. BAPS Health Publications Unit, Lancashire.

Donaldson, R. and Donaldson, R. (1985) Essential Community Medicine (including relevant social sciences). MTP Press, Lancaster.

Donner, A. and Eliasziw, M. (1987) Sample size requirements for reliability studies. Statistics in Medicine. Vol. 6, 441-448.

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

Exton-Smith, A.N. and Sherwin, R.W. (1961) The Prevention of Pressure Sores: Significance of Spontaneous Bodily Movements. The Lancet, Vol. 2 1124-1126.

Exton-Smith, A.N. (1987) The patient's not for turning. Nursing Times Vol.83 (42) 42-44.

Farmer, R. and Miller, D.L. (1983) Lecture Notes on Epidemiology and Community Medicine. (2nd Edition) Blackwell Scientific Publications, Oxford.

Fernie, G.R. (1973) Biomechanical Aspects of the Aetiology of Decubitus Ulcers on Human Patients. PhD Thesis, University of Strathclyde, Glasgow.

Fielding, P. and Foster, J. (1987) Reliability and validity of an in-patient rating scale. Chap.7 in Fielding, P. (1987) Research in the Nursing Care of Elderly People. John Wiley & Sons, Chichester.

Fisher, S., Szymke, T.E., Apte, S.Y. and Kosiak, M. (1978) Wheelchair cushion effect on skin temperature. Archives of Physical Medicine and Rehabilitation, Vol. 59, 68-72.

Flanagan M. (1993) Predicting pressure sore risk. Journal of Wound Care Vol.2 (4) 215-218

Gaston, S.F. (1984) Evaluation of the Norton Scale of Risk for the

Development of Pressure Ulcers (unpublished study). Sacramento, California. cited by Taylor, K.J. (1988) Assessment Tools for the Identification of Patients at Risk for the Development of Pressure Sores: a review. Journal of Enterostomal Therapy, Vol. 15 (5) 201-205.

Gebhardt, K. (1992) Preventing pressure sores in orthopaedics. Nursing Standard, Vol. 6 (23) 4-6.

Gendron, F. (1980) Burns occurring during lengthy surgical procedures. Journal of Clinical Engineering, Vol. 51 (1) 19-26.

Gibson, T., Barbenel, J.C. and Evans, J.H. (1976) Biomechanical Concepts and Effects. In: Kenedi, R.M., Cowden, J.M. and Scales, J.T. (Eds.) Bedsore Biomechanics, University Park Press, Maryland. p.25-30.

Girvin, J. and Griffiths-Jones, A. (1989) Towards Prevention. Nursing Times, Vol. 85, (12) 64-66.

Gloucester Health Authority (1990) Strategy for Fractured Neck of Femur Patients. Gloucester Health Authority.

Goldblatt, H. (1925) Observations upon reactive hyperaemia. Heart, Vol. 12, 281-294.

Goldstone, L.A. and Goldstone, J. (1982) The Norton Score: an early warning of pressure sores? Journal of Advanced Nursing, Vol. 7, 419-426.

Goldstone, L.A. and Roberts, B.V. (1980) A preliminary discriminant function analysis of elderly orthopaedic patients who will or will not contract a pressure sore. International Journal of Nursing Studies, Vol. 17 (1) 17-23.

Goode, H.F., Burns, E. and Walker, B.E. (1992) Vitamin C depletion and

pressure sores in elderly patients with femoral neck fracture. British Medical Journal, Vol. 305, 925-926.

Goodwin, L.D. and Prescott, P.A. (1981) Issues and approaches to estimating inter-rater reliability in nursing research. Research in Nursing and Health, Vol. 4, 323-337.

Gosnell, D.J. (1973) An assessment tool to identify pressure sores. Nursing Research, Vol. 22, 55-59.

Gould, D. (1986) Pressure sore prevention and treatment: an example of nurses' failure to implement research findings. Journal of Advanced Nursing, Vol. 11, 389-394.

Groth, K.E. (1942) Klinische beobachtungen und experimentelle studien uber die enstehung des dekubitus. Acta Chir. Scandinavica, Vol. 87, Supplement 76, 198 (English Summary).

Gunnell, D.J. (1992) Mysterious slapped face rash at holiday centre. British Medical Journal, Vol. 304, 477-479.

Guyton, A.C. (1992) Human Physiology and Mechanisms of Disease. W.B. Saunders Co., U.S.A.

Hall, D.A. (1984) The Biomedical Basis of Gerontology. John Wright & Sons Ltd., G.B.

Hall, D.A., Blackett, A.D., Zajoc, A.R., Switala, S. and Airey, C.M. (1981) Changes in skinfold thickness with increasing age. Age and Ageing, Vol. 10 (1) 19-23.

Hall, D.A., Read, F.B., Nuki, G, ET AL (1974) The relative effects of age and

cortico steroid therapy on the collagen profiles of dermis from subjects with R.A. Age and Ageing, Vol. 3 (1) 15.

Hansard (1993) 8th June, 1993. HMSO, London.

Hibbs, P. (1982) Pressure sores: a system of prevention. Nursing Mirror, Vol. 155 (5) 25-29.

Hibbs, P. (1988) The economics of pressure ulcer prevention. Decubitus, Vol. 1 (3) 32-38.

Hibbs, P.J. (1988a) Pressure Area Care for The City & Hackney Health Authority. City & Hackney Health Authority, London.

Hicks, D.J. (1970) An incidence study of pressure sores following surgery. In: American Nurses' Association (1970) ANA Clinical Sessions, Appleton-Century-Crofts, New York. p.49-54.

Holland, W. and Karhausen, L. (Eds.) (1978) Health Care and Epidemiology. Henry Kimpton, London.

Holmes, R., Macchino, K., Jhangiani, S.S., Agarwal, N.R. and Savino, J.A. (1987) Combating pressure sores - nutritionally. American Journal of Nursing, Vol. 87 (10) 1301-1303.

Holstein, P., Neilson, P.E. and Barras, J.R. (1979) Blood flow cessation at external pressure in the skin of normal limbs. Microvascular Research, Vol. 17, 71-79.

Hunt, J. (1993) Application of a pressure area risk calculator in an intensive care unit. Intensive and Critical Care Nursing, Vol. 9, 226-231.

Husain, T. (1953) An experimental study of some pressure effects on tissues with reference to the bed sores problem. Journal of Pathology Bacteriology, Vol. 66, 347-358.

IAET (1987) Standards of Care for Dermal Wounds: Pressure Sores. International Association of Enterostomal Therapy, Irvine Ca.

Johnson, G. (1991) Non-nursing duties in theatre. Part 2. British Journal of Theatre Nurses, Vol. 1 (4) 20-22.

Keighley, J.K. (1982) Wound Healing in Malnourished Patients. AORN Journal, Vol. 35, (6) 1094-1099.

Kemp, M.G., Keighley, J.K., Smith, D.W. and Morreale, B. (1990) Factors which contribute to pressure sores in surgical patients. Research in Nursing and Health, vol. 13, 293-301.

Kosiak, M. (1959) Etiology and pathology of ischaemic ulcers. Archives of Physical Medicine and Rehabilitation, Vol. 40, 62-69.

Kosiak, M. (1961) Etiology of decubitus ulcers. Archives of Physical Medicine and Rehabilitation, Vol. 42, 19-29.

Krouskop, T.A. (1983) A synthesis of the factors which contribute to pressure sore formation. Medical Hypothesis, Vol. 11, 255-267.

Krouskop, T.A., Reddy, N.P., Spencer, W.A. and Secor, J.W. (1978) Mechanisms of decubitus ulcer formation - a hypothesis. Medical Hypothesis, Vol. 4, 37-39.

Lamb, J.F., Ingram, C.G., Johnston, I.A. and Pitman, R.M. (1980) Essentials of Physiology. Blackwell Scientific Pubs., G.B.

Lamid, S. and EL Ghatit, A.Z. (1983) Smoking, spasticity and pressure sores in spinal cord injured patients. American Journal of Physical Medicine, Vol. 62 (6) 300-306.

Landis, E.M. (1930) Micro-injection studies of capillary blood pressure in human skin. Heart, Vol. 15, 209-228.

Larsen, E. (1986) Evaluating validity of screening tests. Nursing Research, Vol. 35, 186.

Lawson, N.W., Mills, N.L. and Ochner, J.L. (1976) Occipital alopecia following cardiopulmonary bypass. Journal of Thoracic and Cardiovascular Surgery, Vol. 71, 342-347.

Leeds Health Care (1993-94) Schedule A: Quality Contracts. Leeds Health Care.

Leeds Health Care (1994-95) Schedule A: Quality Contracts. Leeds Health Care.

Lewis, T. and Grant, R. (1925) Observations upon reactive hyperaemia. Heart, Vol. 12, 73-120.

Lilienfeld, A.M. and Lilienfeld, D.E. (1980) Foundations of Epidemiology. (2nd Edition) Oxford University Press, New York.

Lincoln, R., Roberts, R., Maddox, A., Levine, S. and Patterson, C. (1986) Use of the Norton pressure sore risk assessment scoring system with elderly patients in acute area. Journal of Enterostomal Therapy, Vol. 13 (4) 132-138.

Lippold, O.C.J. and Winton, F.R. (1979) Human Physiology. (7th Edition) Churchill Livingstone, G.B.

Livesley, B (Ed.) (1989) The Prevention and Management of Pressure Sores within Health Districts The Working Party of the Pressure Sore Study Group at The King's Fund Centre for Health Services Development, London.

Lowthian, P. (1982) Prevention of heel sores. Lancet Vol. 1, 1089

Lowthian, P. (1987) Classification and Grading of Pressure Sores. Care Science and Practice, Vol. 5 (1) 5-9.

Lyder, C. (1991) Conceptualisation of the stage I pressure ulcer. Journal of Enterostomal Nursing, Vol. 18, (5) 162-165.

Mawson, A.R., Biundo, J.J., Neville, P., Linares, H.A., Winchester, Y. and Lopez, A. (1988) Risk factors for early occurring pressure ulcers following spinal cord injury. American Journal of Physical Medicine and Rehabilitation, Vol. 67, 123-127.

McCarthy, M. (1982) Epidemiology and Strategies for Health Planning. King Edward's England Hospital Fund, London.

Millward, J. (1990) Relieving the pressure. Nursing the Elderly, Vol. 2 (4) 14-16.

Minotti, A. (1978) Incidence Studies and Registers. In: Holland, W. and Karhausen, L. (Eds.) Health Care and Epidemiology. Kimpton, London.

Moore, E., Rithalia, S. and Gonsal Korale, M. (1992) Assessment of the Charnwood operating table and hospital trolley mattresses. Journal of Tissue Viability, Vol. 2 (2) 71-72.

Morgan, A.G., Kelleher, J., Walker, B.E., Lososwky, M.S., Droller, H. and Middleton, R.S.W. (1975) A nutritional survey in the elderly: blood and urine

vitamin levels. International Journal of Vitamin and Nutritional Research, Vol. 45, 448-462.

Morison, M.J. (1989) Early assessment of pressure sore risk. The Professional Nurse, Vol. 4 (9) 428-431.

Morris, J.N. (1975) Uses of Epidemiology. (3rd Edition) Churchill Livingstone, London.

Neander, K-D. and Birkenfeld, R. (1991) Decubitus prophylaxis in the operating theatre? Journal of Tissue Viability, Vol. 1 (3) 71-74.

Newman. P. and West, P. (1981) Pressure sores 2 - the value of the Norton Score. Nursing Times Occasional Paper 77. Vol. 29 (21) 15-21.

Nichol, P.A. and Webb, R.L. (1955) Vascular Patterns and Active Vasomotion as Determiners of Flow Through Minute Vessels. Angiology, Vol. 6, p 291.

North Lincolnshire Health Authority (1991) Prevention and Management of Pressure Damage within North Lincolnshire Health Authority. North Lincolnshire Health Authority.

Norton, D., McLaren, R. and Exton-Smith, A.N. (1962) An Investigation of Geriatric Nursing Problems in Hospital. Churchill Livingstone, Edinburgh.

NPRU (1992) The Financial Costs of Pressure Sores to the National Health Service: A Case Study. Nursing Practice Research Unit; University of Surrey.

Nyquist, R. and Hawthorn, P.J. (1987) The prevalence of pressure sores within an Area Health Authority. Journal of Advanced Nursing, Vol. 12, 183-187.

O'Dea, K. (1993) Prevalence of pressure damage in hospital patients in the

U.K. Journal of Wound Care, Vol. 2 (4) 21-225.

Parish, L.C., Witkowski, J.A. and Chrissey, J.T. (1983) The Decubitus Ulcer. Masson Pub., New York.

Polit, D.F. and Hungler, B.P. (1989) Essentials of Nursing Research Methods, Appraisal and Utilisation. (2nd Edition) J.B. Lippincott Company, Philadelphia.

Polit, D.F. and Hungler, B.P. (1991) Nursing Research: Principles and Methods. (4th Edition) J.B. Lippincott Company, Philadelphia.

Preston, K. (1991) Counting the cost of pressure sores. Community Outlook, September, 1991, 19-24.

Pritchard, V. (1986) Calculating the risk. Nursing Times, Vol. 82 (8) 59-61.

Reddy, N.P., Krouskop, T.A. and Newell, P.H. (1975) Biomechanics of a lymphatic vessel. Blood Vessels, Vol. 12, 261-278.

Reid, J. and Morison, M. (1994) Towards a consensus: classification of pressure sores. Journal of Wound Care, Vol. 3 (3) 157-160.

Reswick, J.B. and Rogers, J. (1976) Experience at Ranchos Los Amigos Hospital with devices and techniques to prevent pressure sores. In: Kenedi, R.M., Cowden, J.M. and Scales, J.T. (Eds.) Bedsore Biomechanics. Baltimore University Park Press. p.301-310.

Richardson, B. (1990) Pressure sores - a manager's perspective. Nursing Standard, Vol. 5 (12) 11-13.

Richardson, R.R. and Meyer, P.R. (1981) Prevalence and incidence of pressure sores in acute spinal cord injuries. Paraplegia, Vol. 19, 235-247.

Roberts, B.V. and Goldstone, L.A. (1979) A survey of pressure sores in the over sixties on two orthopaedic wards. International Journal of Nursing Studies, Vol. 16, 355-264.

Scales, J.T. (1976) Air Support Systems for the Prevention of Bed Sores. In: Kenedi, R.M., Cowden, J.M. and Scales, J.T. (Eds.) Bedsore Biomechanics. University Park Press, Maryland. p.259-268.

Scales, J.T., Lowthian, P.T., Poole, A.G. and Ludman, W.R. (1982) "Vaperm" patient support system: a new general purpose hospital mattress. The Lancet, Vol. 2 1150-1152.

Schubert, V. (1992) Hypotension as a risk factor for the development of pressure sores in elderly subjects. Journal of Tissue Viability, Vol. 2 (1) 5-8.

Seaman, C.H.C. (1987) Research Methods Principles, Practice and Theory for Nursing. (3rd Edition) Prentice Hall, London.

Shakespeare, P. (1994) Scoring the risk scores. Journal of Tissue Viability, Vol. 4 (1) 21-22.

Shea, J.D. (1975) Pressure sores. classification and management. Clinical Orthopaedics, Vol. 112, 89-100.

Simpson, G. and Livesley, B. (Eds.) (1993) The Prevention and Management of Pressure Sores within Hospital and Community Settings, The Research for Ageing Trust, London.

Smith, M. (1993) Decubitus ulcers: a multidisciplinary view. Nursing Standard, Vol. 7 (15/16) 25-28.

Spenceley, P. (1988) Norton -v- Waterlow. Nursing Times, Vol. 84 (32) 52-53.

St. James's University Hospital NHS Trust (1993) Pressure Area Care Policy. St. James's University Hospital NHS Trust, Leeds.

Starling, M. (1990) Project improves practice. Nursing Times, Vol. 86 (6) 40-41.

Stewart, T.P. and Magnano, S.J. (1988) Burns or pressure ulcers in the surgical patient. Decubitus, Vol. 1 (1) 36-40.

Stotts, N.A. (1988) Predicting pressure ulcer development in surgical patients. Heart Lung, Vol. 17 (6) Part 1, 641-647.

Streiner, D.L. and Norman, G.R. (1989) Reliability. In: Health Measurement Scale: A Practical Guide to their Development and Use. Oxford University Press, Oxford. Chapter 8, 79-96.

Taylor, K.J. (1988) Assessment tools for the identification of patients at risk for the development of pressure sores: a review. Journal of Enterostomal Therapy, Vol. 15 (5) 201-205.

Thomas, A. (1987) Pre-operative fasting - a question of routine? Nursing Times, Vol. 83 (49) 46-47.

Thompson-Rowling, J. (1961) Pathological changes in mummies. Proceedings of the Royal Society of Medicine, Vol. 54, p. 409.

Torrance, C. (1983) Pressure Sores: Aetiology, Treatment and Prevention. Croom Helm, London.

Touche Ross & CO. (1993) The Costs of Pressure Sores. Touche Ross & Co. London.

Towey, A.P. and Erland, S.M. (1988) Validity and reliability of an assessment tool for pressure ulcer risk. Decubitus, Vol. 1 (2) 40-48.

Treece, E.W. and Treece, Jr., J.W. (1986) Elements of Research in Nursing. (4th Edition) The C.V. Mosby Company, St. Louis.

United Leeds Teaching Hospitals NHS Trust (undated) The Prevention and Management of Pressure Sores within The United Leeds Teaching Hospitals NHS Trust.

Vermillion, C. (1990) Operating room acquired pressure ulcers. Decubitus, Vol 3 (1) 26-30.

Versluysen, M. (1986) How elderly patients with femoral fracture develop pressure sores in hospital. British Medical Journal, Vol. 292, 1311-1313.

Wardman, C. (1991) Norton -v- Waterlow. Nursing Times, Vol. 87 (13) 74, 76 & 78.

Warner, V. and Hall, D.J. (1986) Pressure sores: a policy for prevention. Nursing Times, Vol. 82 (16) 59-61.

Wasson, J.H., Sox, H.C., Neff, R.K. and Goldman, L. (1985) Clinical prediction rules: applications and methodological standards. New England Journal of Medicine, Vol. 313 (13) 793-799.

Waterlow, J. (1985) A risk assessment card. Nursing Times, Vol. 81 (48) 24-27.

Waterlow, J. (1988) The Waterlow Card for the prevention and management of pressure sores: towards a pocket policy. Care Science and Practice, Vol. 6 (1) 8-12.

Wellings, P. (1991) Where are we today? British Journal of Theatre Nurses, Vol. 1 (7) 21-22.

Williams, C. (1992) A comparative study of pressure sore prevention scores. Journal of Tissue Viability, Vol. 2 (2) 64-66.

Young, J. (1992) Preventing pressure sores: does the mattress work? Journal of Tissue Viability, Vol, 2 (1) p. 17.

Young, J.B. and Dobrzanski, S. (1992) Pressure sores, epidemiology and current management concepts. Drugs and Aging, Vol. 2 (1) 42-57.