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# A Study of Sacral Tissue Tolerance to Pressure Using Transcutaneous Measurements of Oxygen and Carbon Dioxide

by

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# ABSTRACT

FACULTY OF MEDICINE, HEALTH AND LIFE SCIENCES
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Doctor of Philosophy

## A STUDY OF SACRAL TISSUE TOLERANCE TO PRESSURE USING TRANSCUTANEOUS MEASUREMENTS OF OXYGEN AND CARBON DIOXIDE

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Pressure ulcers are a serious problem facing patients with restricted mobility or inability to change position. Recognised complications include pain, discomfort, hampered rehabilitation and reduced quality of life. The prevalence of pressure ulcers in hospitals within the UK ranges from 10% to 33% of the patient population and the prevention and treatment of pressure ulcers has been identified as costing the National Health Service 4% of the annual budget.

Pressure ulcers are primarily caused by pressure distorting the capillaries and cutting off the blood supply for a critical length of time, leading to tissue necrosis. The pressure is the result of the individual's own body mass, through gravity, causing compression of the tissues between the supporting surface and the underlying bone. Many intrinsic factors have been identified as influencing the ability of tissues to tolerate pressure. These are fundamentally factors that influence tissue perfusion, the availability of oxygen to tissues and the removal of metabolites.

Transcutaneous partial pressure monitoring of oxygen ($tcPO_2$) is widely used to assess tissue perfusion, and interest in the value of transcutaneous partial pressure monitoring of carbon dioxide ($tcPCO_2$) has been growing. The objective of this work was to examine the relationship between individuals' peak sacral interface pressure, changes in $tcPO_2$ & $tcPCO_2$ levels and capillary blood flow to assess individuals' tolerance to pressure exerted through their own body mass.

Measurements were undertaken on healthy volunteers and identified four patterns of response of $tcPO_2$ and three of $tcPCO_2$ reflecting the varying degrees to which tissue perfusion was compromised and the ability of the tissue to tolerate and respond to pressure. In order to compare the response of individuals' sacral tissue to pressure the maximum level of $tcPO_2$ and $tcPCO_2$ achieved when subjected to pressure and the total area under the curve (AUC) were used to reflect the loss of oxygen and accumulation of carbon dioxide. As well as inter-subject variation, intra-subject variation was observed with a significant difference identified between the responses of $tcPO_2$ & $tcPCO_2$ for individual's left and right sacral sites. This supports the need for sites to be treated as independent sites.

No association was found between body mass index (BMI) and peak sacral pressure, or between BMI, peak sacral pressure and response type of tcPO2 & $tcPCO_2$.

The study indicates that $tcPCO_2$ is a more significant indicator of sacral tissues' tolerance to pressure than $tcPO_2$, and that the response type (Type A(iii)) of $tcPCO_2$ is representative of total capillary closure with a significant accumulation of carbon dioxide (12-22.4kPa). This may play a role in identifying individual's level of tolerance to pressure and susceptibility to tissue damage, which would prove invaluable to patients and the National Health Services alike. Further work is required to understand the point of tissue death.

## Table of Contents

## List of Figures

### Chapter 1



### Chapter 2



### Chapter 3

## List of Tables

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# Chapter 1

## Introduction

This thesis examines the risks associated with pressure ulcer development and undertakes research to assess, through physiological measurements, individuals' tolerance to pressure exerted through their own body mass. In order to appreciate the significance of pressure ulcers this chapter provides a general introduction to the importance of preventing pressure ulcers, the anatomy and physiology of healthy skin, the aetiology of pressure ulcers and the risk assessment tools that have been developed and used to date.

### 1.1 Why Prevent Pressure Ulcers?

Pressure ulcers are a serious problem in modern healthcare, and have long been one of the major problems facing patients with restricted mobility or inability to change position (Kosiac, 1959). Recognised complications include causing tremendous pain, discomfort, hampered rehabilitation and a reduced quality of life (Franks et al, 1999) for those patients unfortunate enough to be affected.

The severity of the pressure damage can prolong the length of stay in hospital and be a potential source of serious systemic complications, which can add to the complexity of the patient's condition. The extended length of stay in a hospital is not only difficult for the patient concerned but also delays another patient from being admitted for treatment. Hibbs (1988) identified that for a patient with the most severe grade of pressure ulcer the length of stay was increased to 180 days, compared with an average stay of 10.9 days. This is the equivalent of treating sixteen standard patients on the basis of time.

The prevalence of pressure ulcers was found to be 4% - 10% in a UK District General Hospital (Clark & Watts, 1994, Cullum et at 2001). In October 2002, the prevalence of pressure ulcers for patients in a large teaching hospital was found to be 9.6%, and so at any one time of the 1300 patient population approximately 125 people would be suffering from pressure ulcers. By 2005 the prevalence had risen to 21%, and with a patient population of 800, one hundred and sixty eight patients would be suffering from pressure ulcers.

Pressure ulcers are a financial burden to the National Health Service through the cost of treatment, the time taken to care for patients and potential litigation costs. Hibbs (1988) calculated that the treatment of a full thickness pressure ulcer with bone involvement to be £26,000. This was equivalent to treating twenty-one orthopaedic patients on the basis of cost. Collier (1999) applied a similar formula to that of Hibbs (1988) and calculated the cost to be £40,000. Within Collier's calculations the patient's average length of stay was 10.5 days. If a pressure reducing surface, used to assist in the prevention of a pressure ulcer costs £5-10 per day, then the additional cost for prevention would be £52 to £105.

The cost of treating and preventing pressure ulcers in a 600 bedded general hospital was identified by Touche Ross (1993) to be between £600,000 to £3 million per year. The annual cost to Southampton University Hospitals NHS Trust for pressure reducing surfaces was £650,000 for 2001/02. When the cost of dressings is included the expenditure escalates further. In 2004 the cost to the National Health Service for the treatment of pressure ulcers was identified as being 4% (£1.4 - 2.1 Billion) of the Health Service Budget (Bennet et al, 2004). As the majority of pressure ulcers are preventable the costs awarded through litigation are also increasing with awards upwards of £100,000 (Robertson, 1987). The prevention of pressure ulcers is therefore important for patients and the National Health Service.

## 1.2 Structure and Function of Skin, Subcutaneous Tissue and Muscle

The health of skin, subcutaneous tissue and muscle is dependant on the adequate provision of oxygen and nutrients, and the removal of waste products produced as a result of cell respiration. Tissue damage and necrosis occurs when tissue perfusion is compromised sufficiently to prevent cell respiration and the removal of metabolic waste products. When pressure is the primary cause for tissue perfusion being compromised sufficiently to cause tissue damage and necrosis, the damage is referred to as a pressure ulcer.

To understand why tissue is susceptible to pressure damage a brief description of the anatomy and physiology is given.

### 1.2.1 Skin

The skin is one of the largest organs of the body, with the surface area of an average adult being approximately 19,354cm$^2$. It acts as a barrier to environmental hazards such as mechanical damage, invasion by micro-organisms, desiccation, ultra violet radiation, and is involved in body temperature regulation.

The skin is also a sensory organ and collects information via an extensive neuronal network regarding pressure, vibration, pain and temperature. All of these are potential hazards if undetected.

The thickness of skin varies from 0.57 mm to 4.4mm, and is thicker on the extensor, ventral surfaces, i.e. palms of hands and soles of feet (Hole, 1990). There are two main layers to the skin, the outer, thinner, epidermis and the thicker underlying dermis. Then beneath the dermis lies the subcutaneous layer/superficial fascia, consisting of areolar and adipose tissue (see Figure 1.1). The epidermis is mainly avascular receiving it's nutrition from vessels reaching up through the papillae of the connective tissue.

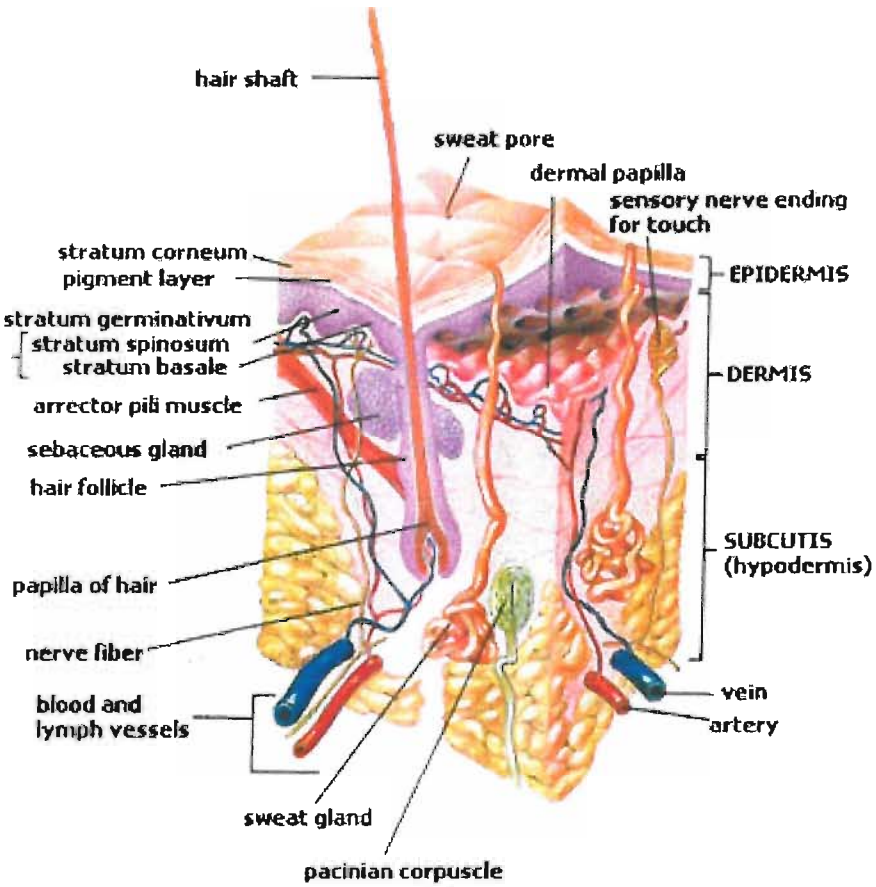



Figure 1.1 Diagram of the structure of the skin and underlying subcutaneous layer

(Ref: http://training.seer.cancer.gov/ss_module14_melanoma/unit02_sec01_anatomy.html)

The epidermis consists of stratified squamous epithelium organised into 4-5 layers, with 5 layers found where exposure of the skin to friction is greatest i.e. soles of feet, palms of hands. Most of the cells in the layers of the epidermis arise from the continually dividing germinating cells of the basal cell layer (stratum basale). As the cells progress up through the layers the nuclei degenerate and the cells die. The cells are shed from the final layer, stratum corneum. The daughter cells are pushed up from the stratum basal to form the stratum spinosum, which is characterised by flattened cells with short spines/processes. The older cells of the next layer, stratum granulosum, have granules of keratohyalin, which is involved in the first steps of forming the protein, keratin. Stratum Lucidum is a layer that only occurs in the thick skin of palms and soles. It consists of flat, dead cells that contain a translucent substance called eleidin. Eleidin is formed from keratohyalin and is eventually transformed to keratin. It's translucent properties lead to its name. The outer thick layer, stratum corneum, consists of layers of flat, dead cells where the cytoplasm and nucleus have been replaced by keratin. Keratination causes the cells to become hardened and die, giving the epidermis it's characteristics. These cells are continually shed and replaced. The variation in thickness of the stratum corneum accounts for differences in skin thickness. The older cells are displaced towards the surface where they mature, die and are eventually shed.

The dermis is a vascular layer, lying beneath the epidermis, with a rich capillary and lymphatic network. The dermis surface area is greatly increased through small, finger-like projections called dermal papilla. These project into the epidermis and contain loops of capillaries supplying oxygen and nutrition. Some papillae contain Meissner's corpuscles. The papillae increase the mechanical strength of the skin by binding the epidermis to the underlying tissues. The dermis mainly consists of a network of fibrous connective tissue containing collagen and elastin fibres. The elastin provides the elasticity and collagen provides the tensile strength of the skin.

Sweat glands and hair follicles with their sebaceous glands and arrector pili muscles are also found in the dermis. The sweat glands help to stabilise body temperature by excreting in response to excessive heat. It is the evaporation of the secretion that cools the surface of the epidermis. Sebatious glands secrete sebum to help protect the skin from dehydration. Within the hair follicle bulb, concentric layers of keratinised, pigmented cells form the hair shaft.

The lower layer of the dermis is called the reticular layer and consists of dense, irregular collagenous connective tissue. The irregular arrangement enables flexibility and strength in all directions. This section contains many blood vessels. Adipose tissue, hair follicles, nerves, blood vessels and sweat glands occupy the space between the interlacing fibres. The reticular layer is attached to organs or underlying bone by the subcutaneous layer. The arrector pili muscles attached to the hair follicle elevates the hair shaft upon contraction and facilitates the secretion of sebum. The dermis supports the epidermis. Dermal thickness and therefore strength varies over the surface of the body from a thickness of 0.5mm in the eyelids to 3.0mm on the soles of feet, the average being 1-2mm.

The blood vessels within the dermis commence as small arterioles branching off arteries within the subcutaneous layer, lead into capillaries and then return via venules which in turn feed into veins within the subcutaneous layer. The capillaries are the site of oxygen exchange and the amount of blood flow through the capillaries is controlled by smooth muscle bands, precapillary sphincters, which constrict proportional to the level of stimuli by the sympathetic nervous system. When the sphincters constrict, pressure inside the capillaries reduces and the vessels collapse. In resting tissues most capillaries are collapsed. Some tissues contain arteriovenous shunts, which allow arteriolar blood to completely bypass capillaries, and these are used to help control body temperature.

### 1.2.2 Subcutaneous Layer

The subcutaneous layer, also referred to as the subcutis, lies immediately below the dermis and consists mainly of loose connective and adipose tissue (fat). Cells, called adipocytes, specialise in the storage of triglycerides and the cytoplasm and nucleus are pushed to the side of the cell. Adipose tissue is a good insulator and helps to reduce heat loss through the skin. It is also a good energy reserve and helps to support and protect various organs.

The amount of fat varies between body regions between individuals. Women generally have more body fat and it is more evenly distributed. Adipose tissue helps distribute pressure applied to the skin and is relatively well vascularised, containing the main blood vessels that supply the skin.

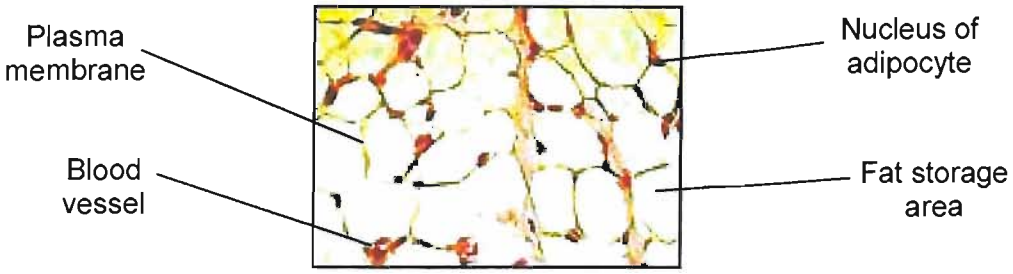



Figure 1.2 Sectional view of adipose tissue

(Ref: http://training.seer.cancer.gov/module_anatomy/images/illu_connective_tissues_1.jpg)

### 1.2.3 Muscle

Muscle underlines the subcutaneous layer and consists of elongated cells called muscle fibres, which utilise adenosine triphosphate (ATP) to generate force to enable body movements, maintain posture, and generate heat. There are three types of muscle: skeletal, cardiac and smooth. The muscle referred to in pressure ulcers is skeletal muscle.

Skeletal muscle is usually attached to bone and is a striated voluntary muscle as it can be made to contract or relax by conscious control. A muscle fibre is roughly cylindrical, long and has several nuclei located at the periphery along it's length. Muscle is surrounded and penetrated by layers of fibrous, avascular connective tissue called deep fascia. Blood flow through muscle at rest is approximately 1 l/min, whilst under strenuous exercise it increases significantly to 25l/min. The blood flow is determined by arterial pressure and vascular resistance.

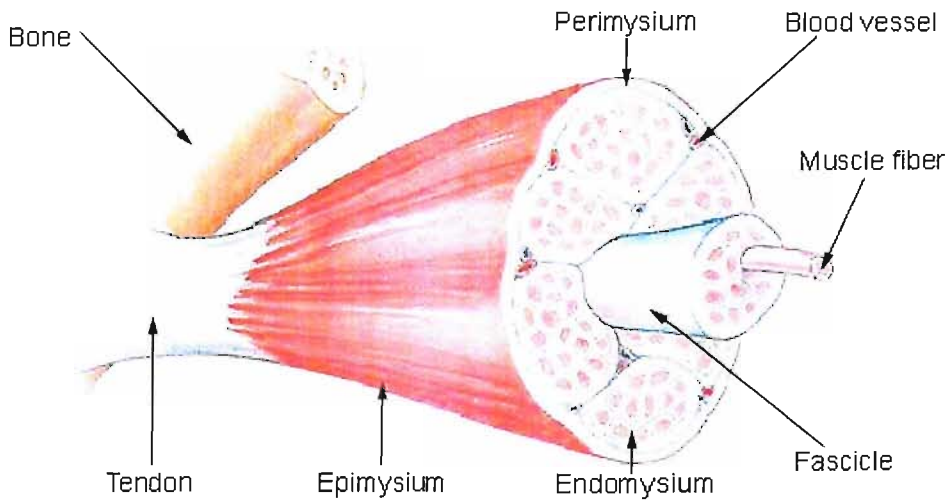



Figure 1.3 Skeletal muscle

(Ref: http://training.seer.cancer.gov/module_anatomy/images/illu_muscle_structure.jpg)

## 1.3 Pressure Ulcers

### 1.3.1 What is a Pressure Ulcer ?

Pressure ulcers are multifactorial in aetiology. Many definitions have been collated over the years, with no one definition appearing to incorporate all factors succinctly. Bliss (1990) suggests that a pressure ulcer is an area of tissue death caused by pressure distorting the capillaries and cutting off the blood supply for a critical length of time. Within the definitions of Braden & Bergstorm (1987) and Cullum & Clark (1992) the critical determinants of pressure ulcers are identified as the intensity and duration of applied pressure, and extrinsic and intrinsic factors also affect tissue tolerance. Extrinsic factors include magnitude, duration and direction of pressure, incontinence, level of mobility, microclimate of the skin, sensory/motor loss, pain, and smoking. Intrinsic factors include conditions affecting the cardiovascular systems (such as hypotension, anaemia and peripheral vascular disease), age, body type, nutritional status and infection.

The extent of damage ranges from non-blanchable erythema of intact skin to extensive destruction with necrosis of subcutaneous tissue and muscle (see Figure 1.4).

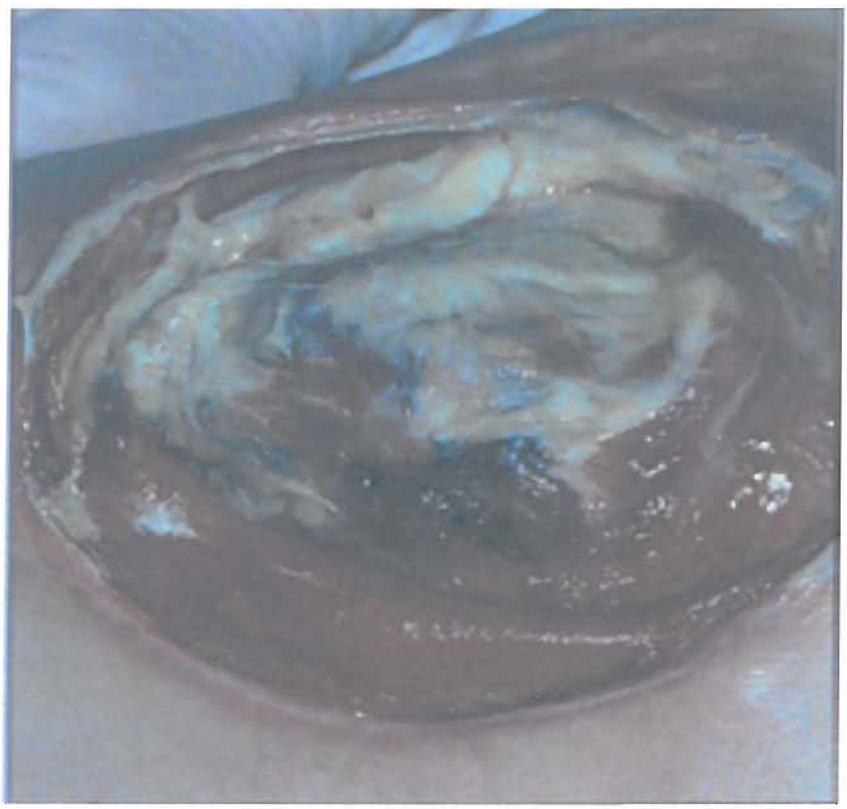
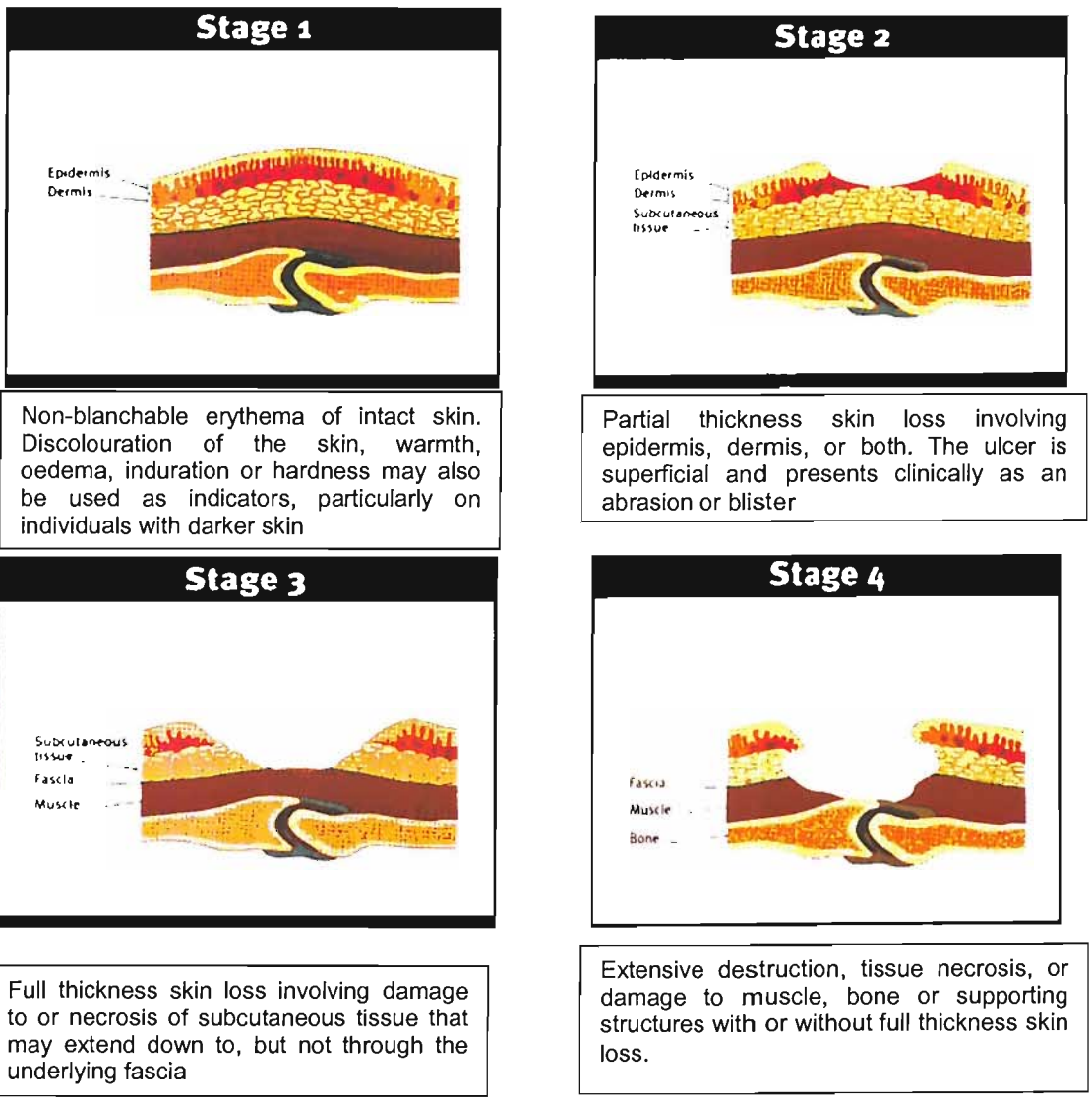


Figure 1.4 Photograph of one of the most severe grades of pressure ulcer

The tissues involved in pressure damage defines the grade/severity of damage. Shea (1975) classified four stages; Stage one being reversible and stage 4 the most severe.

- Stage one is defined as acute inflammation in all layers, with irregular, ill defined area of soft tissue swelling, induration and heat. The effect is limited to the epidermis of the skin and resembles an abrasion. It is reversible.
- Stage two is defined as inflammation and fibroblastic response extending through the dermis to subcutaneous fat. This is also reversible
- Stage three the ulcer extends through into the subcutaneous fat with extensive undermining, essentially full thickness skin defect.
- Stage four there is penetration to deep fascia with involvement of muscle and bone. This identifies the four layers of tissue involved as the skin, subcutaneous fat, muscle and bone.

Figure 1.5 Four stages of pressure ulcers, European Pressure Ulcer Advisory Panel, 1999



The European Pressure Ulcer Advisory Panel (EPUAP), 1999, described the four stages of pressure ulcers as shown in figure 1.5, ranging from stage 1 with non-blanchable erythema, to stage 4 with extensive tissue necrosis including muscle.

### 1.3.2 Aetiology of Pressure Ulcers

There are several factors that cause and affect the development and treatment of pressure ulcers. Some of these factors will be extrinsic to the patient and be associated with environmental and physical parameters. Other factors will relate to the intrinsic well being of the patient and monitored by physiological measurement. This section will identify those factors and discuss the background to current knowledge of pressure ulcer development.

#### 1.3.2.1 External Factors Influencing the Development of Pressure Ulcers

The development of a pressure ulcer can be influenced by a number of factors, which are extrinsic to some aspect of the physiology of the patient, but nevertheless contribute to changes in the environment and subsequent physiological response of the patient.

When tissue is in contact with a surface, physical factors contribute to the risk of damage: pressure, friction, shear forces, temperature, and moisture.

**Magnitude of pressure**

The primary cause of pressure ulcers arises from gravitational forces acting on the human body, in circumstances whereby relatively high pressures occur for prolonged periods of time in specific anatomical areas. The pressure at the interface between the soft tissues and the surface (e.g. mattress, or seat of chair) may not be uniform temporally or spatially. When the external pressure is sufficient to impair the integrity of the blood supply and lymphatic drainage for prolonged periods of time, tissue ischaemia leads to necrosis (Bader & Gant 1988, Bader & White, 1998, Bennett & Lee, 1990). The external pressure, involved in the development of pressure ulcers, is the result of the individuals own body mass, through gravity, causing compression of the tissues between the supporting surface and the underlying bone. Importantly, without pressure there can also be no friction or shear forces on the tissues.

In general the body can be subjected to pressures in addition to those arising from gravitational forces. Atmospheric pressure is evenly distributed over the surface of the

body and provided that pressure variation within gaseous cavities e.g. inner ear and lungs is accounted for then there is no discomfort or damage to the body. The body can also tolerate hydrostatic pressures, as experienced in sub-aqua diving and compression chambers. At 16 atmospheres of hydrostatic pressure, the blood pressure was found not to alter as long as air has free access to frontal sinuses, ethmoid and mastoid air cells (Husain 1953).

When an object is placed upon a surface a pressure will result. The magnitude of the pressure is related to the surface area in contact with the object, the mass of the object and the acceleration due to gravity.

Pressure is defined as the force divided by the area and the systeme international units (S.I) are Newtons per metre squared. Force is defined as the product of the mass of the patient in kilograms and the acceleration due to gravity. The acceleration due to gravity is 9.81 metres per second squared. For example, if one considers the pressure required to push a drawing pin into a board the force applied by your thumb (4N) to push the drawing pin creates a higher pressure at the pin point than that experienced by your thumb, this is because the surface area of the pin point (0.5mm$^2$ or 5x10$^{-6}$ m$^2$) is considerably smaller than the surface area of your thumb (2x10$^{-4}$ m$^2$).

Pressure on thumb = $\frac{4N}{2 \times 10^{-4}\ m^2}$ = $2 \times 10^4\ N/m^2$

Pressure on board at pin point = $\frac{4N}{5 \times 10^{-6}\ m^2}$ = $8 \times 10^6\ N/m^2$

Blood pressure is more commonly measured using the imperial measurement of mmHg and interface pressure is equally most often referred to as a measurement of mmHg. Therefore to convert N/m$^2$ to mmHg the following information is required:

$$pressure(N/m^2) = \frac{pressure(mmHg)}{7.501x10^{-3}}$$

$$pressure(mmHg) = pressure(N/m^2)x7.501x10^{-3}$$

By contrast transcutanous partial pressure of oxygen and carbon dioxide used to be measured in mmHg, but is more commonly measured in kiloPascals (kPa). 1 Pascal is the equivalent to 7.501 x 10$^{-3}$mmHg therefore 1 kPa is equivalent to 7.5mmHg.

Extending the above analogy to the human body the pressure through sitting and lying is not uniformly applied because bony prominences form localised areas of higher pressure due to reduced surface area. The measurement of interface pressure has been used to indicate the level of pressure at the surface/body contact point, which varies according to the nature of the surfaces at different anatomical sites, commonly the sacrum, trochanter and the heels.

Swain et al (1993) identified the interface pressures for the NHS pink marble-effect mattress. The average sacral pressure was 99mmHg after the mattress had 6 months of clinical use, the average heel pressure was 173 mmHg, and the average pressure at the trochanter was 117mmHg. In comparison a range of foam mattresses with improved pressure reducing properties reduced the sacral pressure to between 9mmHg to 32mmHg, pressure at the trochanter to between 14mmHg to 35mmHg and pressure at the heels to between 32mmHg to 49mmHg. It was concluded that the pressure reducing properties of the foam mattress are improved by greater conformity to the contours of the body's surface therefore increasing the surface area over which the forces are applied.

Pressure to tissue over any bony prominence is transmitted from the surface of the skin to the underlying dense bone, compressing all the underlying tissue to varying degrees. The elasticity of the tissues results in the force being distributed in a hammock or sling affect with the greatest pressure over the bone (Shea 1975). Experiments by Le et al (1984) identified that although the surface pressure may be 25 – 35mmHg the internal pressure may be three to five times higher, which supports the views of Shea (1975).

The posture of an individual influences the magnitude of the pressure by changing the surface area over which the force is distributed (Barbenel, 1991; Defloor, 2000) and alters which bony prominences are involved. To explore the impact of postural changes on the magnitude of the pressure researchers have used interface pressure measurements, which are explored further in chapter 2, section 2.2. The lowest interface pressures were thought to occur when an individual is in the horizontal position because the pressure is distributed over a larger surface area. However, Defloor (2000) undertook studies to examine the affect of ten positions on interface pressure. He used a flexible measuring sheet with 684 sensors enabling him to identify peak pressures. The maximum interface pressure was identified when the subjects were in the lateral position, 90 degrees, not lying on the shoulder (see Figure 1.6). The pressure was 58.6 ± 11.9 mmHg for a standard hospital mattress and 46.2 mmHg ±

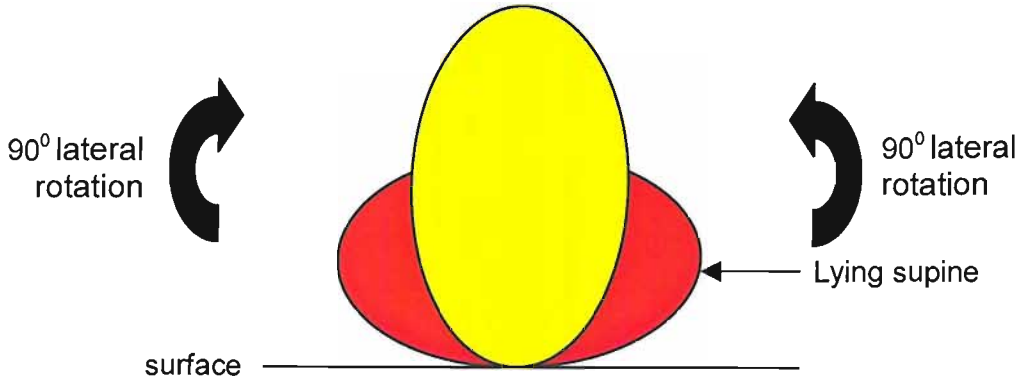



Figure 1.6 Lateral position, 90 degrees rotation from the supine position

9.6mmHg on a polyethylene-urethane mattress. The lowest interface pressure occurred when the patient was in the 30 degree semi-Fowler position, where the pressure was 30.3 ± 5.5mmHg for the standard hospital mattress and 24.4 ± 3.9mmHg for the polyethylene-urethane mattress. Defloor believed that shear would be prevented in this position because the head and foot end were elevated 30 degrees thus inhibiting the person sliding down the bed. The study was undertaken on 62 healthy volunteers but did not include an even distribution of age, sex or body mass index. These findings also contradict the findings of Siderank et al (1992), who found a significantly higher pressure in the semi-Fowler position than the supine. Sideranko et al undertook the study on intensive care patients, whereas Defloor used healthy subjects and therefore the differences may be due to changes in tissue tone, depth and structure due to the medical conditions. In addition the semi-Fowler position was at 45 degrees rather than 30 degrees.

As pressure ulcers are caused as a result of pressure causing occlusion of the blood and lymph vessels for a critical period of time, researchers have been particularly interested in capillary pressure. This is because it is commonly considered that when the applied pressure to tissue exceeds that of capillary pressure, capillary occlusion will occur causing ischaemia of the surrounding tissue (Scales, 1980, Sugama et al 2002). This suggests that measuring the interface pressure should assist in helping to indicate whether the pressures were potentially sufficient to cause tissue damage. Currently, the primary method of relieving pressure at the interface is through pressure relieving mattresses. Manufacturers have therefore striven to reduce the interface pressure to a minimum, for any given patient according to weight.

Although Kosiac and Dinsdale demonstrated the positive benefit that pressure relief had on the tissue's ability to tolerate pressure, there is still a need for guidance on the level of pressure relief required to avoid tissue breakdown by ischaemia. To develop such guidance there is a need to identify and accurately measure the interface pressure between the skin and the surface upon which the patient is in contact. Equally by measuring capillary pressure, steps can be taken to reduce the interface pressure below that of the capillary pressure. However, this is traditionally an invasive procedure (Landis 1930 & Sugama et al 2002) and so alternative methods need to be investigated and have led to work exploring tissue perfusion.

Bader (1990a) was interested in exploring the nature of tissue recovery to repeated loading and he postulated that the nature of the recovery would be determined by the resilience of the tissues and structures affected. The effect of the pressure on tissue viability would need to be measured to identify potential areas of tissue damage clinically. As tissue viability is dependent on an adequate supply of oxygen and nutrients (and the removal of metabolites) provided by the blood supply, Bader (1990a) chose transcutaneous gas monitoring, previously demonstrated to be an accurate and repeatable method of measuring tissue perfusion (Newson & Rolfe 1982, and Steinacher & Wodick, 1983.). Previously, Bader & Gant (1988) had identified that a range of pressures ranging from 22mmHg (3kPa) to 92mmHg (12.2kPa) were required to generate a 50% reduction in resting transcutaneous oxygen tension ($T_cPO_2$). The transcutaneous electrode was incorporated in an indentor that was used to apply a load employing a pressure of 30mmHg (4kPa). No clear explanation was given as to why 30mmHg was chosen as the load, however, it was only described as being a moderate load. The load was applied perpendicular to the sacral skin to avoid significant friction and shear. The sacral tissue was loaded for 10-15 minutes and then unloaded for 2 to 5 minutes. This was then repeated at least twice more. Measurements were undertaken on eight healthy subjects and six debilitated subjects (4 with multiple sclerosis and 2 with spinal cord injury) with no recent history of sacral sores, to ensure that tissue was intact at the time of the experiments. Interestingly for the young healthy subjects there was a significant reduction in $T_cPO_2$ initially in response to the application of the load. The level then partially recovered during the loaded period. The tissue recovery to pre-loaded $T_cPO_2$ levels was achieved rapidly when the load was removed. Subsequent applications of the same load had less effect on the $T_cPO_2$ level. In contrast some of the debilitated subjects showed no recovery during the loaded period and a full recovery of $T_cPO_2$ levels was not achieved. Subsequent loading diminished the $T_cPO_2$ levels further.

Arterial gas tensions have been used by some researchers to represent the normal range of $tcPO_2$ and $tcPCO_2$ (Bader 1990a & b, Knight et al 2001) with $tcPO_2$ ranging from 6.8 to 12kPa and $tcPCO_2$ from 4.8 to 5.9kPa. Transcutaneous partial pressure for oxygen and carbon dioxide however, would be expected to differ to arterial levels because it represents the local skin oxygenation and reflects the local balance of oxygen supply and demand. Other studies have identified a wide variation in $tcPO_2$ values of unloaded tissue, both at different sites of the same individual and between individuals.

Dowd et al (1983), identified the normal range of transcutaneous gas tensions for oxygen to be 6-12.9kPa, Rodrigues et al (2001) 5.54 – 13.67kPa and Takiwaki et al (1991), 4.79 – 11.27kPa (2 standard deviations from the mean). On comparing hard and soft sites Seiler Stahelin (1979), identified baseline $tcPO_2$ to be similar, with a range of 9.28 – 13.76kPa.

In relation to $tcPCO_2$ Takiwaki et al (1991) and Rodrigues et al (2001) identified a baseline range of 3.6 – 6.3kPa and 2.74 – 7.44kPa respectively for $tcPCO_2$.

The variation in range between studies may be explained by the following differences between the studies, which have been summarised in Table 1.1. Briefly these are:

- sites of measurement
- postural position which could cause haemodynamical changes,
- selection of volunteers and what constitutes as normal, healthy
- sex
- age
- temperature of electrode for transcutaneous partial pressure of oxygen and carbon dioxide.

The anatomical site selected for measuring transcutaneous partial pressure of oxygen and carbon dioxide varies between the studies undertaken. Takiwaki et al, (1991) and Seiler and Stahelin(1979) have examined the variation between different anatomical sites, Takiwaki et al (1991) undertook simultaneous measurements of the cheek, palm, anterior aspect of the forearm, abdomen, back and tibia and posterior aspect of the leg, and Seiler et al compared the trochanteric area (hard site) and quadriceps (soft site). Other than the face (forehead and cheek) and palm of hand, no significant differences were found between the sites.

The variation in sites between the studies may also be linked to postural position, as can be seen from Table 1.1, the positions adopted by the studies included lying prone, supine, lateral and sitting.

The variation between selection criteria for volunteers across previous studies may have contributed to the variation in results. Some previous studies included smokers (Dowd et al, 1983 & Takiwaki et al, 1991) and nicotine is known to be a vasoactive substance, capable of influencing cutaneous circulation (Workman & Sceffield, 1983). Although the results of Dowd et al (1983) did not demonstrate the lowest range of partial pressure of oxygen, which would be expected if tissue perfusion was compromised, other studies have indicated that smoking compromises the hyperaemic response (Noble et al, 2003). Although age has been considered to influence peripheral perfusion Dowd et al (1983) found no significant relationship between age and levels of transcutaneous oxygen and carbon dioxide.

The measurement of transcutaneous gas tensions involves the heating of the underlying skin to values in excess of normal physiological temperatures to ensure maximum vasodilatation and reliable gas detection. The recommended temperature ranges from 42-44°C and the range of temperatures used across the studies may help to explain some of the variation in the results. Knight el al (2001) and Bader (1990) used 44.5 °C, Takiwaki et al (1991) and Seiler and Stahelin(1979) used 44 °C, and Rodrigues et al (2001), whose selection criteria for volunteers most closely resembled this study, used 43 °C. Interestingly work has not been undertaken to explore if there is a significant influence of the results with the use of different temperatures.

The reproducibility of $tcPO_2$ and $tcPCO_2$ measurements was examined by Coleman et al (1986) and the coefficient of variation was found to be 10% when measurements were repeated daily for 3 weeks.

**Table 1.1. Transcutaneous Partial Pressure of Oxygen and Carbon Dioxide Levels Identified in Previous Work**

| | Arterial (Bader, 1990a & Knight et al, 2001) | Dowd et al (1983) | Seiler & Stahelin (1979) | Rodrigues et al (2001) | Takiwaki et al (1991) | Coleman et al (1986) |
|---|---|---|---|---|---|---|
| Transcutaneous partial pressure of oxygen (kPa) | 6.8 –12 | 6 – 12.9 | 9.28 – 13 | 5.54 -13.4 | a) mean across all sites 4.79 – 11.27<br><br>b) 10cm above iliac crest 6.4 – 9.65 | a) Chest wall, 2nd rib, mid-clavicular 5.5 – 12.5<br><br>b) 10cm below knee joint, lateral to anterior tibial border 6.3 – 12.2 |
| Transcutaneous partial pressure of carbon dioxide (kPa) | 4.8 – 5.9 | | | 2.7 - 7.4 | a) 3.6 – 6.3<br><br>b) 8.1 – 8.9 | |
| Sex | male & female | male & female | male & female | male & female | male | male & female |
| Age | 24 - 78 | 12-84 yrs | 17-52, mean age 28 | under 25yrs | 24-46yrs (mean 29yrs) | 18-61Yrs (mean 32yrs) |
| Health | Bader (1990a) 6 volunteers with spinal cord injuries; 3 with amputations; 2 with cerebral palsy; 2 with GuillainBarre.<br><br>Knight et al (2001) healthy volunteers | Generally healthy no cardiovascular disease, no talking or smoking during expt. | No definition | No skin or cardiovascular disease No systemic disease No medication No smoking or alcohol addiction Alcohol and caffeine restricted for 6 hrs pre. | Healthy volunteers, forbidden to smoke 2hrs prior to study | Healthy and free of cardiovascular disease Not allowed to smoke or talk during expt. |
| Position during measurement | Prone | Supine | Hard site volunteer in lateral position, soft site volunteer prone | Sitting position | Prone or supine | Lying supine |
| Site of measurement | Sacral | 10cm below knee joint, lateral to anterior tibial border | Hard site trochanteric area Soft site quadriceps | Left ventral forearm & 3rd finger | Cheek, palm, anterior aspect of forearm, abdomen, back, tibia and posterior aspect of leg | Mid-clavicular and below knee |
| Temperature of electrode | 44°C | | 44°C | 43°C | 44°C | 44°C |

Tissue viability is equally dependent on the adequate removal of carbon dioxide, a waste product of metabolism.

The tension of $CO_2$ in the lungs is 5.6 kPa (40mm Hg) and in tissues 6.1 kPa (46 mm Hg). This is because $CO_2$ and water ($H_2O$) are formed by aerobic cell metabolism and therefore the local partial pressure of $CO_2$ within tissues rises above that of the arterial blood tension. Therefore as the blood flows through tissue capillaries, carbon dioxide will diffuse across into the plasma of the capillaries increasing the tension of carbon dioxide from 5.6 to 6.1 kPa. The blood returns via the veins to the right side of the heart and then to the lungs via the pulmonary artery. Because the $CO_2$ tension is now higher in the venous blood than the alveoli, as the venous blood passes through the lungs the $CO_2$ diffuses from the blood into the alveoli and the carbon dioxide tension gradually falls again to 5.6 kPa. The blood is then recirculated to the left side of the heart via the pulmonary artery, and pumped on to the arteries of the body.

The production of $CO_2$ effects acid/base balance, and by diffusing into the capillaries enables the transportation of $CO_2$ from the site of formation (tissues) to the site of elimination (the lungs). Normally the level of $CO_2$ is maintained at 6.1 kPa because an adequate level is required to maintain a normal blood pH of 7.4. The pH scale is an acid/alkaline indicator which measures the level of hydrogen ions. The range extends from 0-14 with 7 being neutral. As the pH falls below 7 there is an increase in acidity, and as it increases above there is an increase in alkalinity. The blood must be kept at a pH of between 7 and 7.8 to sustain life.

When the $CO_2$ diffuses into the capillaries it can remain part of the blood plasma in different physical forms or diffuse into the red blood cells (see Figure 1.7). Within the blood plasma $CO_2$ dissolves in $H_2O$ to form carbonic acid ($H_2CO_3$), which has a pH of about 4. This is represented by the following chemical equation:

$$CO_2 + H_2O = H_2CO_3$$

The carbonic acid is buffered by sodium bicarbonate, which is produced by the red blood cells. This forms part of the acid/base balance which is essential in order to maintain a pH of 7.4. The ratio between sodium bicarbonate and carbonic acid needs to be 20:1.

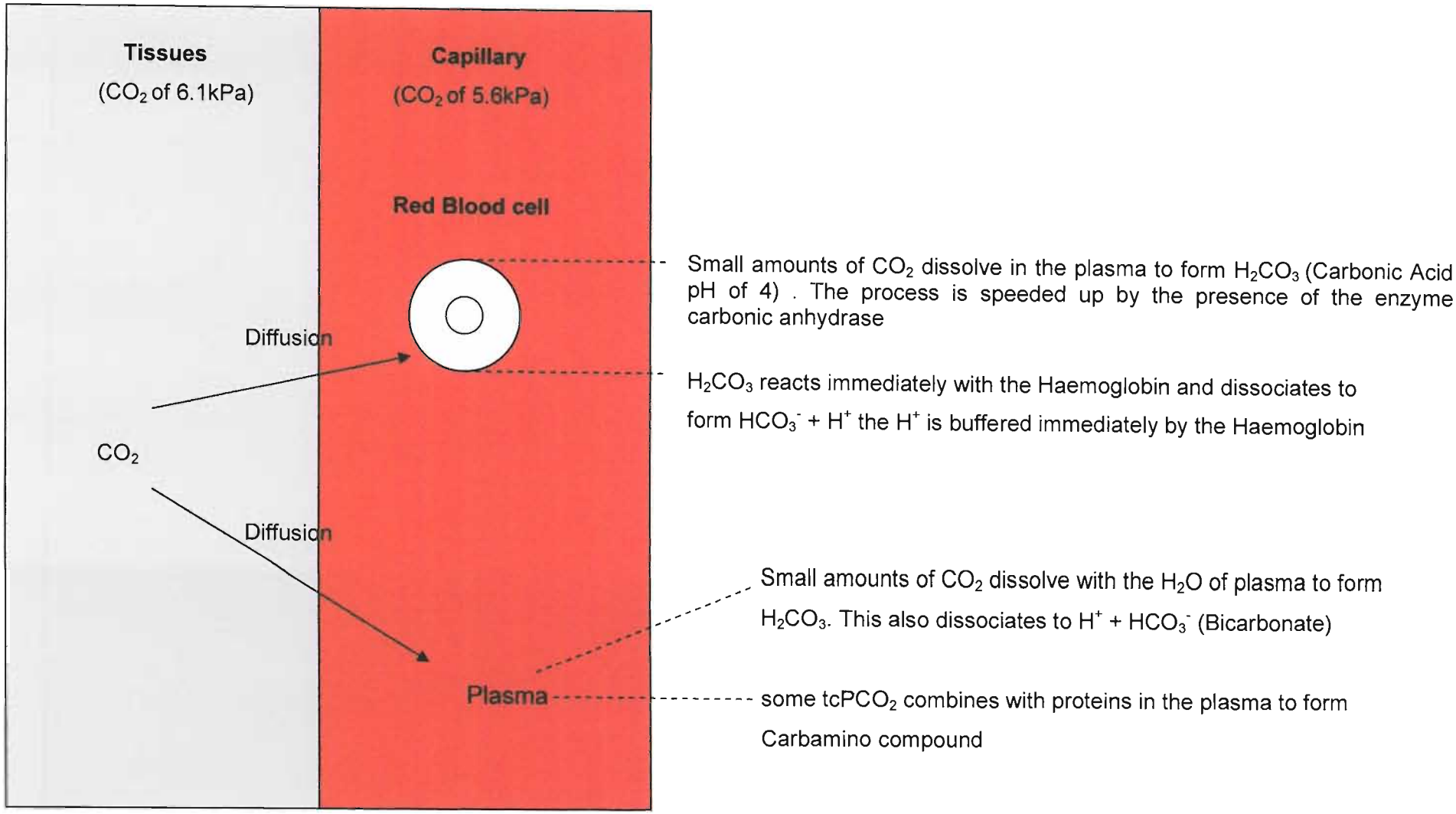



**Figure 1.7 Diagrammatic representation of the processes involved in the removal of CO2 from tissues to the capillaries.**

Bicarbonate is produced in the red blood cells in the following way. As the blood passes through the tissue capillaries, most of the $CO_2$ passes through the plasma to the red blood cells where it is rapidly converted to carbonic acid by an enzyme called carbonic anhydrase. The carbonic acid reacts with the haemoglobin which gives up its oxygen and forms bicarbonate.

$CO_2$ + carbonic anhydrase ⟶ $H_2CO_3$

(Released from tissue cells) (within red blood cell) (Carbonic Acid)

$H_2CO_3$ + Haemoglobin ⟶ $H^+$ + $HCO_3^-$

(Carbonic Acid) (Bicarbonate)

pH is a balance between $CO_2$ in solution in the blood plasma and plasma bicarbonate levels. Changes occurring primarily in the bicarbonate plasma levels indicate metabolic changes. Changes occurring to the $CO_2$ in solution are termed respiratory. This is because the sodium bicarbonate in the plasma is determined by diet and formation or excretion of sodium bicarbonate via the kidneys.

The $CO_2$ in solution is directly proportional to the $PCO_2$ in the lungs, alveoli (arterial blood) and locally through the production of $CO_2$ as a waste product of metabolism. The greater the alveolar ventilation the lower the $CO_2$. However, blood rich in $CO_2$, pumped through the blood vessels of limbs does not stimulate respiration, nor does total ischaemia of one of four limbs even though the accumulation of $CO_2$ and $H^+$ is significant (Comroe, 1974). Therefore local capillary occlusion, and subsequent ischaemia will certainly not trigger a respiratory response. Insufficient or lack of removal of $CO_2$ causes increasing local acidity through the accumulation of $CO_2$ levels and this can be seen through the transcutaneous measurement of the partial pressure of carbon dioxide ($tcPCO_2$), when pressure is applied to sacral tissue (Knight et al 2001).

$CO_2$ is produced as part of aerobic and anaerobic cell metabolism. Hypercapnia occurs within tissues as a result of tissue perfusion being compromised and increases in the partial pressure of $CO_2$ are correlated to the degree that tissue perfusion is compromised (Johnson, 1991).

Increases in partial pressure of $CO_2$ has been identified through animal studies to vary between tissues during periods occlusion of blood flow. Johnson (1991) compared the results of several animal studies demonstrating the range of differences between tissues. For example occlusion of the left anterior ascending coronary artery led to $CO_2$ levels rising to 55.5kPa within the heart of dogs, arterial occlusion of the kidney led to levels of 16.3kPa within the medulla of the renal tissue, and arterial occlusion of rabbits intestine led to an increase from 3.3 to 13.2kPa.

When the partial pressure of oxygen is maintained within the normal range, cells have been found to tolerate extreme acidosis as a consequence of high levels of $CO_2$. However, in the event of ischemia the same levels of $CO_2$ result in tissue damage (Katsura et al, 1994). When the blood supply to tissues is blocked or compromised, as occurs in the development of pressure ulcers, tissue hypoxia is always accompanied by tissue hypercapnia (Johnson, 1991). Interest has grown in the role that hypercapnia plays in cell apoptosis and Hotter el al (2004) found that in the combined presence of renal cell hypoxia and hypercapnia apoptosis occurred. The role of $CO_2$ in tissue apoptosis is still not fully understood in that is it an indirect or direct effect of the raised intracellular pH due to the accumulating $CO_2$. Therefore further work is still required to understand the mechanism and role of $CO_2$.

The effect of tissue relief through the use of a dynamic support cushion was described by Bader (1990a) using $tcPO_2$ and $tcPCO_2$. Dynamic refers to an active system where the air pressure in groups of cells within the cushion are alternately increased and decreased. Bader (1990a) found that the response of a healthy male demonstrated the ability of tissue to improve tolerance to pressure upon re-exposure. The initial drop in $tcPO_2$ was greater than that using the indenter. Although the $tcPO_2$ levels improved, a full recovery to pre-experiment levels had not occurred after 3 complete cycles of the alternating cushion, but did occur following removal of pressure. For the subject with multiple sclerosis, the initial $tcPO_2$ level was lower than that of the healthy subject, however, although the recovery was slower it appears to have recovered to the pre-experimental level. For the subject with a spinal cord injury recovery exceeded the pre-experimental $tcPO_2$ level. The $tcPCO_2$ level, however, rose following an increase in pressure and then plateaued. $tcPO_2$ level did not respond to the cyclical inflation and deflation of the air sacs. For one of the subjects with spinal cord injury the $tcPO_2$ levels declined in response to the pressure but showed no evidence of tissue recovery after 1.5 cycles. The subject therefore lifted his own body weight using his arm strength to relieve pressure. This resulted in full recovery of $tcPO_2$ level following the second lift.

Importantly, this demonstrated that the individual's lift technique was more effective than the cyclical inflation deflation of the seat cushion i.e. total pressure relief is more effective than alternating pressure. Although the $tcPCO_2$ level also responded to the individual's lift technique it didn't completely return to the pre-experimental level. There were no results available in the paper demonstrating the response of $tcPCO_2$ levels to pressure in the healthy subjects. The changes in levels were much slower than that of the $tcPO_2$ levels and from the examples given did not fully recover. Bader suggests that this may be a biochemical marker that could potentially be used to determine the susceptibility to tissue breakdown, as the removal of metabolites is slower than the re-oxygenation.

The results of the above experiments demonstrate that even between subjects with spinal cord injury there are significant differences in the effect that pressure has on their tissue perfusion. This raises the question as to what changes in tissue perfusion are significant to the development of pressure ulcers? Although research was undertaken on a limited number of subjects it identifies that there is an active vasomotor response to applying and releasing pressure, which diminishes the effect of subsequent loading. Bader postulated that if the cycles were continued that the $tcPO_2$ levels would remain constant even during the periods of loading. This response could be explained by the effect of the mechanical stress and anoxic state occurring in the tissues, stimulating the release of histamine and prostaglandin, which are known vasodilators.

Bader (1990a) also postulated that part of the partial recovery of $tcPO_2$ level during the loading period may be due to reactive hyperaemia to the tissues having some visco-elastic properties. The tissues therefore exhibit stress relaxation with time under constant deformation.

The use of an indenter to apply the pressure, in the above studies, also requires further examination. The indenter was positioned and used when the healthy subjects were lying prone, and would not be tolerated by many patients' due to their medical condition. Therefore the development of a different method of applying and assessing the effects of pressure that is both accurate and tolerated by subjects, is important in order that patients in the more acute setting can be involved in studies.

Patterns of changes in tissue perfusion to pressure were only examined for a limited number of subjects, and didn't include a reasonable range of age, sex and body

mass index. This study will investigate the peak pressures sacral tissue is subjected to through individuals' own body mass and examine the influence on local tissue perfusion and diffusion of $tcPO_2$ and $tcPCO_2$ to local tissue. Once normal patterns of response have been identified future work will be able to explore if certain trends or trigger levels are associated with changes preceding tissue damage.

**Duration of pressure**

It is not just the magnitude of pressure that is of interest, but also the duration that the pressure is applied. Duration in the development of pressure ulcers is influenced by an individual's ability to change their position so reducing or relieving the pressure. The individual must therefore be able to move and respond to discomfort felt in association with not changing their position and therefore alleviating the affects of pressure. Anything that influences an individual's ability to be able to move and/or respond to discomfort is susceptible to the development of pressure ulcers. For example;

Loss of motor control – stroke, spinal cord injury
Loss of sensation – neuropathy, spinal cord injury
Restricted ability to move due to body weight (Obesity)
Lowered level of consciousness –analgesia, sedation, medical condition
Depression – loss of will to want to move
Pain - restricting movement due to fear of pain

The nerve supply for the skin is found in the epidermal layer and consists of free nerve endings, which are sensitive to pain, and Merkel's discs, sensitive to touch. Meissner's corpuscles and root hair plexuses also have free nerve endings sensitive to touch and are found in the dermis and superficial fascia respectively. The Pacinian corpuscles are found in the superficial fascia and consist of nerve endings surrounded by many layers of flattened cells. Deformation of these causes the nerve terminal to fire resulting in an awareness of pressure above a certain threshold, and this in turn stimulates an individual to move, reducing or removing the stimuli.

People move whether they are asleep or awake in response to sensory stimuli received by the brain. This is a protective physiological phenomenon to avoid excessive pressure on various parts of the body. Exton-Smith & Sherwin (1961) studied the number of movements a group of fifty elderly patients made during the night and related it to the development of pressure ulcers. Of ten patients with an

average nightly movement of between 0-20 nine developed pressure ulcers. Of the group with an average number of movements of between 51-100 per night, none developed pressure ulcers. Movement is important because it is the factor affecting duration and if the individual is unable to change their position assistance is required to relieve the pressure. Unfortunately only the frequency of movement was noted, not the duration of time between movements, it would also have been interesting to understand what caused the differences in frequency of movement, was it always linked with their medical condition or did other factors need to be considered.

Studies have been undertaken to examine the relationship between duration and magnitude of pressure and the effects on tissue viability. Brooks & Duncan (1940) undertook experiments on the tails of albino rats to determine the effect of known amounts of pressure applied for measured lengths of time on various specific tissues. The pressure was applied uniformly to the tail of the rat by means of plethysmograph. One hundred and fifty experiments were performed with pressures ranging from 20 to 1419 rnmHg. The periods that the pressure was applied for varied from 3 to 48hrs. The rats' tails were observed for periods up to up 3 months and microscopic sections were taken at various stages to observe pathological changes.

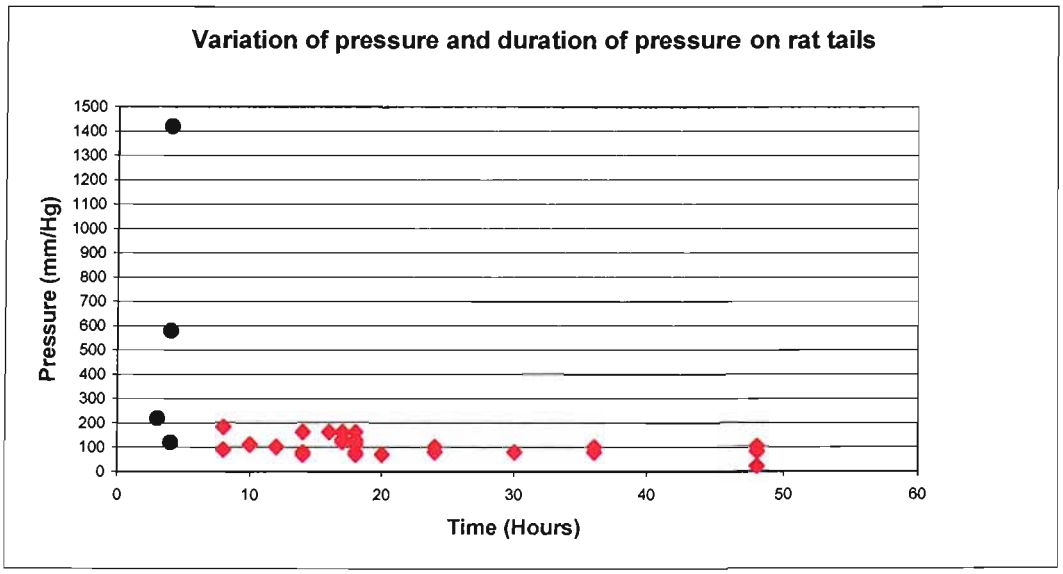
The results presented graphically in Figure 1.8 indicate that there would seem to be a critical time period, between 5-8hours, where the tolerance to pressure becomes insufficient to prevent tissue necrosis. Following this period of time the lower pressures were not tolerated without damage occurring. The range of pressures applied did not cover an even range of pressures from 0mmHg upwards and therefore it can only be postulated that the pressure applied exceeded the critical closing pressure for the capillaries within the rats tails. The results also demonstrate that higher pressures can be tolerated for limited periods of time, without tissue necrosis occurring.

Husain (1953) concluded, from work undertaken on guinea pigs, that evenly distributed pressure over a wide area of the body is much less damaging to tissue than localised/point pressure. Low pressure maintained over a long period of time produced more tissue damage than high pressure for short periods. Therefore Husain concluded that duration of pressure is more important than the actual pressure, which supports the findings of Brooks & Duncan (1940). It was also found that tissue injury occurred through an increase in the permeability of the capillaries. This was particularly noticeable after the release of the pressure as interstitial

oedema occurred leading to the congestion of lymphatic and venous channels. Cellular damage followed. Husain explained that the release of persistent pressure is followed by reactive hyperaemia, oedema and often capillary and venous haemorrhage.

Kosiac (1959) demonstrated an inverse relationship between the amount and duration of pressure that tissue tolerated before pathological changes occured. Furthermore, Kosiac (1961) undertook a series of experiments involving forty albino rats and demonstrated that microscopic pathological changes were absent or less prominent when the alternating pressure was applied in contrast to constant pressure. The hamstring muscle of the albino rat was exposed to pressures ranging from 35 to 240mmHg for periods of one to four hours. Constant pressures were applied to the hamstring group of muscles of twelve normal rats for a predetermined length of time. Eight normal rats were exposed to alternating five minute periods of pressure and no pressure. No changes were noted to the muscle when subjected to pressures of 35mmHg. The application of intermittent pressure demonstrated that the muscle would tolerate intermittent pressures of 70mmHg for two hours with no sign of damage. In contrast the application of 70mmHg of constant pressure produced moderate changes after 2 hours.

**Figure 1.8 Graphical representation of Brooks' work (1940) showing the effect of varying pressure and duration on rat tails**




◆ necrosis of the tail occurred ● no necrosis to the tail occurred

Figure 1.9 and Figure 1.10 show graphically the response of the muscle to constant and intermittent pressure. Figure 1.9 readily illustrates the inverse relationship between time and pressure, which supports Husain's work described above. Even though it identifies an overall relationship it is apparent that different rats have different levels of tolerance to duration and magnitude of pressure. For example it would be expected that if more than 10% damage to muscle occurs when 200mmHg was applied for 3 hours, the same or more severe damage would be expected to occur when 250mmHg pressure applied for the same period or longer. This was not the case and requires further work to understand why.

The influence of intermittent pressure supports the need to regularly change a patient's position to relieve pressure. This work also stimulated the early work into the development of alternating pressure reducing surfaces.

The potential effect of Kosiac (1961) using a 20 gauge needle inserted into the muscle to measure tissue pressure must be considered because this may have influenced the response of the muscle to pressure. Fluid was also infused through the needle at a rate of 4.6cc per hour that may have increased the internal pressure to the muscle causing a different response by the tissue.

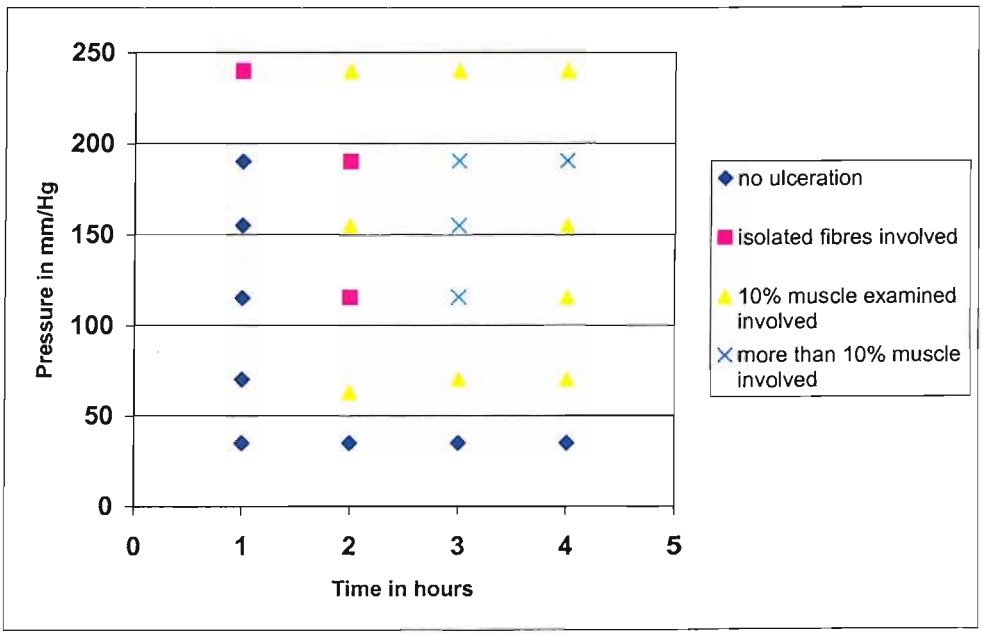



**Figure 1.9** **The time pressure relationship when constant pressure is applied to the hamstring group of muscles of normal rats (Kosiac, 1961)**

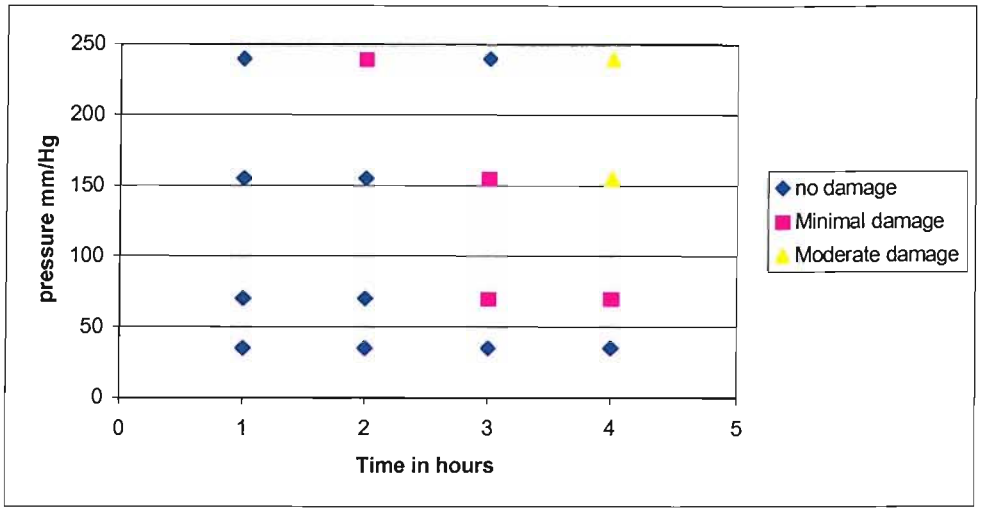



**Figure 1.10 The time pressure relationship with pressure applied for 5 minutes and then released for 5 minutes to the hamstring group of muscles of normal rats (Kosiac, 1961)**

### Shear force

Shearing is the asynchronous movement of adjacent surfaces of tissue leading to displacement and damage. This can be caused by posterior sacral skin being fixed due to friction with the bed whilst the patient slides down. This causes shearing in the deep fascia resulting in stretching and angulations of vessels causing thrombosis. Due to the introduction of electric profiling beds the occurrence of shear may be reduced as the positions required for patient treatment and comfort can be better maintained through the positioning of the frame so preventing the patient from sliding down the bed. Improved manual handling techniques i.e. glide sheets and the use of hoists also helps to prevent the traditional primary causes of shear.

### Friction

A friction force is caused by two surfaces being in contact with each other and one moving over the other e.g. the patient being dragged over the bed sheets. The frictional force is in the opposite direction to the applied force. The movement is superficial and friction removes the outer protective stratum corneum, which accelerates the onset of ulceration. A friction force will be affected by the applied pressure, but the damage caused is usually superficial and should therefore be referred to as friction damage rather than a pressure ulcer.

Dinsdale (1974), was interested in the effects of friction in addition to the magnitude and duration of pressure and undertook a series of experiments with normal and paraplegic pigs to examine the effects of pressure with friction over time. The friction was applied using an electro-mechanical device. The first experiment applied a constant pressure, both with and without friction, for 3 hours over the posterior superior iliac spine. The pressure was then removed and the pig observed for seven days. No ulceration was found when pressure without friction was less than 480mmHg. Animals receiving both pressure and friction were more susceptible to ulceration, however, with pressures of greater than 500mmHg friction did not increase the frequency of ulceration.

The second experiment applied intermittent pressure for three 1.5hour periods with a 1hour rest between each application of pressure. This was repeated for five days. During the 1.5hour period, pressure and friction were applied on one side of the posterior superior iliac spine for the first week and then pressure alone on the other side the following week. The areas were observed for seven days. When ulceration occurred it presented within 18 hours of the first day's pressure application. In those areas that received both pressure and friction ulcers occurred with pressures as low as 45mmHg. In the areas that received no friction a pressure of 290mmHg was required to cause ulceration. The second experiment is felt to closer resemble a patients' experience as it explored the effect of being intermittently exposed to pressure and friction. The friction is an important element due to the additional mechanical damage caused to tissue.

A third experiment was designed to measure the significance of friction. A pressure of 159mmHg was applied to the right and left posterior iliac spines for one day, in three 1.5hour periods with 1hour rest periods between each pressure application. A coin was flipped to identify whether it was the right or left side that received friction every 15minutes. Twenty-four hours after the experiment the sites were assessed. Two of the thirteen pigs, who did not experience the friction, developed partial thickness decubitus ulcer. Eleven of the fourteen pigs that had friction combined with pressure of 159mmHg experienced partial thickness debicutus ulcers. The question remains though, what was different about the pigs that weren't exposed to friction and developed a partial thickness debicutus ulcer and those that were exposed to friction and didn't develop a partial thickness debicutus ulcer? No reference is made and this would be of interest in identifying a true risk group. Finally a fourth experiment

was undertaken to examine the blood flow in skin and subcutaneous fat with external pressure or friction, but was inconclusive.

Dinsdale (1974) concluded that as the pressure was reduced the length of time required to cause tissue damage increased. This supports the work of Kosiac (1961) and Brooks & Duncan (1940) in identifying an inverse relationship between the magnitude and duration of pressure in the production of decubitus ulcers. Importantly, the work also indicated the significance of friction. Dinsdale also tested the hypothesis that friction produced a shear force that occludes the blood vessels and therefore decreases tissue perfusion. The question as to whether friction can be created without exposing an area to the effects of shear requires further consideration outside the scope of this study.

**Excessive moisture**

When moisture is uncontrolled it waterlogs and softens the epidermal tissues making them more susceptible to maceration by decreasing the tensile strength. Moist skin is more susceptible to damage as a consequence of friction and shear because of changes to the drag coefficient (Pfeffer, 1991). It is thought to increase the risk of pressure damage fivefold. (Reuler & Cooney, 1981). Moisture in the form of urine and faecal incontinence pose the greatest risk to epidermal tissue damage due to the pH of the moisture, bacteria and toxins (Norton et al, 1962 & Allman et al, 1986). However, with good skin care and continence management this factor can be controlled. It is important to note that urine and faeces cause irritation and inflammation of the epidermis without the influence of pressure and can been seen at ileostomy and colostomy sites. Therefore the primary cause of damage is due to moisture this must be identified and not confused with damage resulting primarily from pressure.

### 1.3.2.2 Internal Factors Influencing Tissue Tolerance of Pressure

Many intrinsic factors have been considered to influence the ability of tissue to tolerate pressure. Sufficient pressure can cause complete occlusion of the blood supply and tissue death. Temperature, exercise, nutritional status and medical conditions that reduce the oxygenation of the tissues, such as respiratory complications; cardiac failure leading to insufficient circulation of oxygenated blood; narrowing of the blood vessels; low haemoglobin levels therefore restricting the ability of the blood to carry oxygen; loss or reduced motor and sensory responses affecting the individuals response to pain and tissue damage are all considered contributory

factors. Fundamentally they are factors that a) influence tissue perfusion and the availability and/or ability of oxygen to be delivered to the tissues for cell respiration and for metabolites to be removed, and b) influence the ability of tissues to accommodate the pressure, without detrimental effect. The availability of oxygen is influenced by respiration, central and peripheral circulation, tissue blood flow and metabolism. The ability of tissue to accommodate the pressure is influenced by skeletal shape, body mass, tissue thickness, tone and distribution.

**Factors influencing tissue perfusion and the availability of oxygen**

**(i) Respiratory influence**

The transport of oxygen and metabolites via the energy dependant transport system require intact membranes if osmotic and fluid haemostasis are to be preserved between the intracellular and extracellular environments. Aerobic energy production through oxidative phosphorylation requires adequate oxygenation. Hypoxia is the commonest cause of lethal and sub-lethal injury.

Oxygen is needed by cells for cell respiration and the production of energy. A constant supply is required, and is provided through respiration into the alveoli of the lung. The oxygen is transported through diffusion into the alveolar capillary blood. The oxygen binds to haemoglobin, which is then circulated by the heart and circulatory system to the capillaries of organs and tissues where gas exchange takes place through diffusion. Therefore any medical condition hampering; i) the effectiveness of an individuals respiration and diffusion of oxygen into the alveoli capillary blood i.e. chronic obstructive airways disease or asthma; or ii) the availability or ability of haemoglobin to combine with the oxygen i.e. anaemia or carbon monoxide poisoning; or iii) the effectiveness of the heart and circulatory system for example due to heart failure or peripheral vascular disease, will compromise the delivery of oxygen to organs and or tissues (Lubbers, 1983; Lund, 1983). The following parameters influence the oxygen pressure field in the tissue:

- Blood flow
- Blood haemoglobin content
- Position of the oxygen dissociation curve
- Arterial $PO_2$
- Tissue oxygen consumption
- Capillary radius
- Radius of the cylinder supplied with oxygen

Tissue metabolism influences tissue $PO_2$ in two ways. Firstly the more active the tissue the higher the demand for oxygen and local level of $PCO_2$, hydrogen ($H^+$), and the lower the level of $PO_2$. Secondly, the greater the use of oxygen the lower the level of tissue $PO_2$ and the steeper the concentration gradient for diffusion. The net effect of these changes on tissue depends on how much blood flow changes and the haemoglobin oxygenation curve shift s in relation to the increased use of oxygen. Tissue metabolism is decreased by the cold and rises with increases in temperature. Slight increases in temperature have a marked effect on increasing the metabolic demands of the skin. A rise by 1°C causes a 10% increase in tissue metabolism and oxygen demand (Fisher et al, 1978).

**(ii) Blood supply and capillary pressure**

The cardiovascular system is responsible for the transport of oxygen, nutrients and hormones to tissues and for the removal of carbon dioxide and metabolites for excretion by expiration and the kidneys. This is a requirement for all tissues.
It is achieved by an exchange between the blood flow within the capillary network and interstitial fluid across the capillary wall. The skin has an extensive network of capillaries, which are fed by arterioles. The density of capillaries and blood supply varies in different regions of the body. For example the face and scalp have a rich blood supply whereas the lower leg, particularly over the tibia has a poorer supply.

Blood flow to organs is related to their needs, and local mechanisms exist to enable blood flow to be increased when required. Muscles need a higher level of oxygen during exercise, which is brought about by an increased blood flow. During exercise the accumulation of metabolites, which consist of carbon dioxide ($CO_2$), potassium ($K^+$) and other substances act on the arterioles and override the sympathetic vasoconstrictor tone causing vasodilatation. The accumulation of metabolites also occurs if the blood flow has been occluded. When the circulation is restored the vasodilatation stimulated by the metabolites can be seen as the skin becomes warm and flushed. This is known as reactive hyperaemia.

The accumulation of metabolites causes pain known as ischaemic pain and stimulates the body to take action to try and increase or re-establish blood flow for the removal of the metabolites and re-oxygenation of the tissues. For example during angina the pain due to ischaemia of cardiac muscle forces an individual to slow their activity to a level that the circulation can support.

As pressure ulcers are caused by a pressure level sufficient to occlude the blood and lymph vessels for a critical period of time researchers have been interested in the capillary pressure. This is because it is commonly considered that tissue pressure exceeding that of capillary pressure will occlude the capillaries causing ischaemia of the surrounding tissue. (Scales, 1980, Sugama et al, 2002). Work to identify capillary pressure was undertaken by Landis (1930). He identified that arteriolar capillary pressure ranged from 21-48mm/Hg, with an average of 32mmHg. The venous pressure ranged from 6-18mmHg with an average of 12mmHg. Landis(1930) undertook studies on the skin capillaries in the nail folds of healthy human individuals and patients with no cardiovascular disease. Single capillary loops in the finger of the subjects were cannulated with a micropipette. This in turn was connected to a manometer, the height of which was adjusted to balance the blood pressure in the vessel under observation. The individuals were in the recumbent position with the finger precisely at the level of the manubrium sterni. Landis (1930) also examined the effect of the position of the hand in relation to capillary pressure and findings demonstrated that when the hand is above the base of the heart the average pressure in the venous limb remained approximately constant. If the hand was dropped below the suprasternal notch the increase of capillary pressure was almost identical with the theoretical hydrostatic increment of pressure due to the column of blood in the veins. The arteriolar pressures increased in line with the venous capillary pressure when the finger fell below the suprasternal notch.

Following the work of Landis (1930), capillary pressure is commonly taken to be 32mmHg. Although Landis (1930) identified the average arteriolar pressure as 32mmHg his work also demonstrated that there is a range of pressures for healthy people and that these are influenced by postural change and temperature. Patients may experience many pathophysiological changes which can influence capillary pressure e.g. hypotension, vascular disease, medication such as inotropes. A patient's capillary pressure could therefore be significantly lower than that of a healthy individual, thus increasing their risk of developing pressure ulcers, or higher potentially increasing tissue tolerance.

Due to the work of Landis (1930) many concluded that if the interface pressure exceeded 32mmHg, tissue ischemia would occur. This, however, is now being challenged as the stiffness, distribution and composition of body tissue are thought to influence the interface pressure and differ between individuals. As such a definitive

figure such as 32mmHg whilst providing an indication of pressure tolerance it is unlikely to offer clear guidance for risk assessment purposes. More recent studies have demonstrated an increase in skin blood cell flux, measured using the laser Doppler technique, when pressures of 25 to 50mmHg were applied to sacral tissue for healthy volunteers (Schubert and Fagrell, 1989). Histopathological examination of tissue from rats' trochanteric region, following the application of 145.3mmHg (250g) of pressure for 6hours, identified changes due to hyperaemia, primarily in the subcutis, but no tissue necrosis (Salcido et al, 1994).

The blood supply to the skin is regulated by body temperature. When the body is hot the blood vessels vasodilate and when the body is cold the blood vessels constrict, which takes the blood vessels away from the surface of the skin to minimise heat loss. This reaction is brought about partly by the direct action of the local temperature and partly by the temperature regulation centre in the hypothalamus of the brain. The hypothalamus alters the sympathetic tone of the blood vessels within the skin. With heat the sympathetic tone is reduced and the vessels dilate, and in cold the sympathetic tone is increased and the blood vessels constrict. Therefore controlling the room temperature is important for any study examining the response of tissue perfusion to a stimulus. The blood vessels are also sensitive to mechanical and chemical stimulation. For instance a white line effect occurs if a blunt end of a pin is stroked across the skin. As the pin is moved a white line appears corresponding to where the blood has been forced out of the underlying capillaries. This is then followed by the hyperaemic response, and increased reddening due to the increased blood flow.

Injury to the skin caused by drawing the sharp end of the pin across the skin, or damage caused by heat or acid/alkali produces the triple response. Here the firm pressure of the pinpoint stimulates vasodilatation, resulting in a visible red line. The surrounding area to the line is a red flare, with an irregular margin caused by arteriolar dilatation. The dilatation of the arterioles can only occur however if the sensory nerves are intact because the sensory nerves from the skin run via the dorsal nerve roots of the spinal cord and axon reflex arc. Medical conditions affecting this response include spinal cord injuries and multiple sclerosis. The area also becomes raised due to an increase in tissue fluid as a result of increased permeability of the capillaries. The elevated area is termed the wheal.

In terms of the level of oxygen carried by the blood, a shortage of oxygen alone is termed anoxia and if less severe, hypoxia. There are four main types of anoxia. Anoxic anoxia or hypoxic hypoxia is anoxia due to a shortage of oxygen in the air, or lung disease limiting access of oxygen to the blood and leads to a low oxygen tension in the arterial blood. The second type of anoxia is anoxic anaemia due to a deficiency of haemoglobin. The oxygen tension will be normal but there is insufficient oxygen being carried. The third type is stagnant anoxia where the blood flow is too slow and therefore insufficient oxygen is supplied to meet the demand of the tissues, this occurs during heart failure. The fourth type is histotoxic anoxia and is due to failure of the cells extracting the oxygen from the blood for example through cyanide poisoning.

Therefore pathophysiological changes reducing the effectiveness of the cardiac output, respiration or causing narrowing/constriction of the blood vessels will influence tissue perfusion.

Factors reducing perfusion of the skin and tissues will influence the susceptibility of tissues to damage occurring due to ischaemia. Sufficient pressure can cause complete occlusion of the blood supply and tissue death (Kosiac, 1961, Dinsdale 1974). Oxygenation of tissues can be reduced by temperature, exercise and medical conditions i.e. respiratory complications, cardiac failure leading to insufficient circulation of oxygenated blood, narrowing of the blood vessels, low haemoglobin levels therefore restricting the ability of the blood to carry oxygen, loss or reduced motor and sensory responses affecting the individuals response to pain and tissue damage.

Previous studies have identified (Rathscheck & Schroeder, 1974) that despite the increase in blood flow following the administration of acetylcholine, which stimulates the cholingeric sympathetic nervous system therefore increasing the local blood flow, tissue hypoxia still occurred. It was found that the blood flowed through the capillaries without reaching the nutritive capillaries. The blood flow that is important for gas exchange is that through the small, thin-walled vessels. The blood flowing through the artery to vein and arteriole to venule shunts does not supply the oxygen to tissue cells. This indicates that blood flow measurements are insufficient to use as an indicator of oxygen levels within the tissue, which is why the interest over the years has gravitated towards $tcPO_2$ measurements as a possible indicator of tissue viability.

### (iii) Temperature

Skin temperature is related to core body temperature and environmental temperature, which will influence capillary pressure. Landis (1930) explored the effect of temperature and found that capillary pressure rose in response to raising the temperature. Raising the temperature to between 41-43°C the capillary arteriolar pressure rose by 22-36mmHg. When raising the temperature to between 43-47°C the venous capillary pressure rose by 22-33mmHg. From Landis' research, it appears that the experiment involved three subjects only for the arteriolar and venous capillary response, but the results in each case demonstrated as temperature rose pressure rose, and as temperature reduced pressure reduced. On lowering the external temperature to 9.8-10.2°C the arteriolar and venous pressure initially fell by 6-11mmHg below the preceding normal values. However after 5-8mins the pressure rose to 2-14mmHg above the normal values.

Although there is concern that the invasive technique used by Landis (1930)influenced the pressure, the work does demonstrate that capillary pressure is readily influenced by external factors such as temperature.

### (iv) Quality and quantity of subcutaneous tissues and musculature

The structure and compressive nature of the tissues can influence the level of pressure that can be tolerated tissue before damage occurs. Nola and Vistnes (1980) undertook animal studies and demonstrated that a pressure-time regime consistently produced cutaneous ulceration over a bony pressure point but no damage in a location where muscle separated the skin and bone.

Some conditions cause loss of muscle bulk and tone, weight loss, reduced peripheral perfusion of tissues e.g. diabetes, cardiac failure (Swain & Bader 2002). Since pressure is force per unit area and so the shape and weight of an individual will affect the interface pressure. The shape of an individual is dependant on the skeletal size, the quantity, tone and shape of musculature and the amount of subcutaneous fat. The compressive characteristics of the tissues and structures will influence their susceptibility to breakdown in response to pressure (Swain & Bader 2002, Bader 1990a &b).

Medical conditions influence the internal factor characteristics, and the degree of influence depends upon the individual and the level to which the medical condition

has progressed. People with cachexia have higher interface pressures because there is less tissue over the bony prominences (Defloor 2000) and there is usually an elevated metabolic rate increasing the demand for oxygen. The Waterlow score (Waterlow, 1985) and other risk assessment tools have incorporated build/weight for height or body mass index as a risk factor. Allman et al (1995) found a significant association between lower body weight, <58kg, and pressure ulcer development. Obese people show greater areas of increased pressure, but the peak pressures are lower (Lindan & Greenway, 1965).

No trend is evident between a subject's weight and interface pressure, or the subject's build, characterised by body mass index, and interface pressure. The effect of an individual's anatomy on the interface pressure is "more subtle" (Swain & Bader 2002) and the effect of pressure on the physiology is also very individual, making it difficult for current risk assessment tools to be effective at identifying true risk.

### 1.3.3 Risk Assessment Tools

In order to identify patients at risk of developing pressure ulcers and assist in the management and prevention of pressure ulcers, many risk assessment tools have been developed over the years. Risk assessment tools have been used to assist in the management and prevention of pressure ulcers. This section will examine, first the development and role of the assessment tools used to indicate the level of susceptibility of patient to developing pressure ulcers, and second, the validity and reliability of the tools.

#### 1.3.3.1 History of Risk Assessment Tools

The review is limited to the English Language and covers a period from 1940 through to the submission of this work. The first reference to risk assessment in relation to the risk of an individual patient developing a pressure ulcer is the work of Norton et al (1962) who explored geriatric nursing problems in hospitals and highlighted the importance of identifying those patients at risk of developing pressure ulcers.

Prior to the work of Norton et al. (1962) certain diseases had been identified as significantly increasing the risk of developing pressure ulcers, but no method of identifying an individual's level of risk within a group. For example, if the incidence of pressure ulcers amongst patients with spinal cord injuries is 10%, there is no method

of identifying which ten patients out of a group of 100 will go on to develop pressure ulcers.

The aetiology of pressure ulcers is multifactorial and assessment tools attempt to incorporate contributory factors and weight the score according to the degree of influence on the development of pressure ulcers.

The work of Norton et al (1962) acknowledged the multifactorial nature of the aetiology of pressure ulcers and by incorporating their clinical experience, acknowledged that the patient's general condition was of great importance in determining whether or not a pressure ulcer would develop. The value of assessment tools emerged as a consequence of their work.

The Norton Scale was used to assess the patient's general condition by means of a simple scoring system, which took into account five aspects of the patient's condition. Those aspects are physical condition, mental condition, activity, mobility and incontinence and the scores were weighted according to the level of impact perceived in causing pressure ulcers, see Table 1.2. Thus the aetiology of pressure ulcers was established as playing a significant role in the design of an assessment tool. Norton et al (1962) used the assessment tool to assist in identifying the significance of the patients' general condition in the development of pressure ulcers and the impact of nursing care. Each of the five aspects had four components with a descending rating scale of four to one, so the maximum score of twenty represented the optimum general condition of a patient and therefore minimum risk of developing pressure ulcers. The minimum score of five, however, represented the worst physical condition and therefore greatest susceptibility to developing pressure ulcers. Forty eight per cent of the 250 patients involved in the study with an initial score of less than twelve went on to develop pressure ulcers and 32% of patients with an initial score of 12-14. In contrast only 5% went on to develop pressure ulcers if the initial score was 18-20. Norton et al (1962) identified an almost linear relationship between the initial score and the incidence of pressure ulcers. The average initial score of all patients developing pressure ulcers was 12.9. Following the development of pressure ulcers the score average had decreased slightly to 12.3. In comparison the average score for patients who didn't develop pressure ulcers was to 15.7 on admission. The study therefore also highlighted the importance of on going assessment in response to change in the patients' condition.

**Table 1.2 Patient Assessment Form (Scoring System) Norton et al., 1962**

| | | Physical Condition | Mental Condition | Activity | Mobility | Incontinence | Total Score |
|---|---|---|---|---|---|---|---|
| Patient Name | Date | Good 4<br>Fair 3<br>Poor 2<br>Very Bad 1 | Alert 4<br>Apathetic 3<br>Confused 2<br>Stupor 1 | Ambulant 4<br>Walk/Help 3<br>Chair bound 2<br>Bed 1 | Full 4<br>Slightly limited 3<br>Very Limited 2<br>Immobile 1 | Not 4<br>Occasional 3<br>Usually 2<br>Doubly 1 | |
| | | | | | | | |
| | | | | | | | |

*Patients with a total score of 14 or less are liable to develop pressure ulcers and when the score is lower than 12 the risk is very great*

It is unclear why Norton et al (1962) selected the five aspects discussed and the evidence to support the weighting of the score e.g. if someone is confused why are they more likely to develop pressure ulcers because confused patients can be very mobile or quite sedentary.

Bliss et al (1966) adopted a modified version of the clinical score devised by Norton et al (1962) in examining the effectiveness of various surfaces in the treatment of pressure ulcers. The clinical score being used as part of the selection criteria for subjects meant the study was in-part dependant on the validity, specificity and sensitivity of the clinical score in predicting those patients that would have normally, without the intervention of the surface, have gone on to develop pressure ulcers. The modification to the scoring system consisted of inverting the rating scale of the four components using a score of 0-3, and interchanging the positions of grades "confused" and "apathetic".

A score of fifteen would indicate a patient at maximum risk of developing a pressure ulcer and a score of zero would indicate a minimum risk of developing a pressure ulcer, see Table 1.3. There would not appear to be any benefits or rationale for the modifications, only that of convenience.

Applying the modified clinical score Bliss et al (1966) undertook further analysis of the original records from the work of Norton et al (1962) and found that fifty one per cent of patients admitted with a clinical score of 9-15 developed pressure ulcers in the following two weeks. In comparison four per cent of patients with a clinical score

of 0-2 developed pressure ulcers. For patients who had been admitted for a considerable period of time a rising score over a period of time i.e., onset of double incontinence from occasional incontinence of urine frequently resulted in the development of pressure ulcers. This reinforced the views of Norton et al. (1962) that reassessment in response to changes in clinical condition was crucial.

**Table 1.3 The Clinical Score (Bliss et al., 1966)**

| | | Physical Condition | Mental Condition | Activity | Mobility | Incontinence | Total Score |
|---|---|---|---|---|---|---|---|
| Patient Name | Date | 0 Good<br>1 Fair<br>2 Poor<br>3 Bad | 0 Alert<br>1 Confused<br>2 Apathetic<br>3 Stupor | 0 Ambulant<br>1 Walk with help<br>2 Chair bound<br>3 In Bed all day | 0 Full<br>1 Slightly limited<br>2 Very limited<br>3 Immobile | 0 Not<br>1 Occasional<br>2 Usually of urine<br>3 Doubly incontinent | |
| | | | | | | | |
| | | | | | | | |

Bliss et al (1966) identified a score of seven or more as part of the selection criteria for patients admitted into the trial. However, there is no explanation as to why this score was selected. Critically the scoring system would also not identify someone at risk if they were chair bound or bed bound and immobile as the score would only be five to six. In comparison the scoring system devised by Norton et al (1962) would have identified the risk as very great. It is therefore suggested that the modifications to the scoring system may have detrimentally affected the tools reliability and validity. Use of sensitivity analysis would improve validation as any modification to the tool could be comprehensively tested.

Only in 1979 did the clinical scale of Norton et al (1962) become known as the Norton Scale, being made available as a pocket size card to be used by ward staff. A score of fourteen or below indicated that a patient was at risk of developing pressure ulcers.

The five aspects of the patient's condition selected by Norton et al (1962) within the assessment tool are not independent of each other. For example, a patient's level of activity will be directly influenced by their ability or inability to mobilise independently. Similarly a patient may be capable of independent mobilisation but if their mental condition is such that they lack the incentive or motivation to move the effect is equivalent to that of a patient whose mobility is partially or totally compromised. Thus mental condition, activity and mobility are all closely inter-related. The equality of the

weighted scoring system to the five contributory factors identified in the Norton Scale was questioned by Goldstone & Roberts (1980), as was the lack of mutual exclusivity between the contributory factors. For example, deterioration in a patient's physical condition is likely to be reflected in their level of activity and mobility. Goldstone & Roberts (1980) studied the factors that had contributed to the final Norton Score in 39 patients, discriminate analysis was undertaken on the results. The patients were divided into Group A (no signs of erythema or development of pressure ulcers) and Group B (the presence of erythema or pressure ulcers). It was identified that there was a significant difference in the mean score of the patients activity, Group A's mean score being 2 and Group B's score being 1, and mobility, Group A's mean score being 2.39 and Group B's mean score being 1.95. Goldstone & Roberts (1980) concluded that a very simple assessment tool could be used based on the sum of the activity and mobility. It was acknowledged within the study that the sample size was too small and that various specialities needed to be involved in addition to orthopaedics. Later, Goldstone & Goldstone (1982) examined the predictability of routine admission information i.e. blood pressure, pulse, age, sex, temperature, Norton Score and some variants of the Norton Score based on varying the weighting of contributory factors for a random sample of elderly orthopaedic, trauma patients. The sample consisted of 40 patients, age 60 plus, randomly selected, over a 15-month period and found that the Norton score was statistically significant ($P<0.01$).

Goldstone & Goldstone (1982) also explored the weighting of the contributory factors. Significant correlation occurred between physical and mental score, physical and activity, and mental and incontinence. An adaptation of the Norton Score was developed using only the physical condition and incontinence factor with the addition of age. Age was added because it was identified as the only other significant factor, other than the Norton Score, that varied between the group that developed pressure ulcers and the group that were free of pressure ulcers. A scoring system was devised, with the risk rising with age, Table 1.4, but no rationale given for the weighting selected.

The development of many other assessment tools has since followed, but all are adaptations of, or an upgrading of the Norton Scale. Gosnell (1973) attempted to identify specific variables demonstrated to be important in the development of pressure ulcers, devising and evaluating an assessment tool for identifying high risk patients, see Table 1.5.

**Table 1.4 Age Group Scoring (Goldstone & Goldstone, 1982)**

| Age Group | Score |
|---|---|
| 60-64 | 3.5 |
| 65-69 | 3 |
| 70-74 | 2.5 |
| 75-79 | 2 |
| 80-84 | 1.5 |
| 85-89 | 1 |
| 90-94 | 0.5 |
| >95 | 0 |

**Table 1.5 Data Collection Sheets for the Assessment of Patients' Potential for Developing Pressure Ulcers, (Gosnell, 1973).**

Sheet 1

| Name | | Diagnosis | | | |
|---|---|---|---|---|---|
| Age | Height | Date of Admission | | | |
| Sex | Weight | Date of Discharge | | | |
| Rating Scale | | | | | |
| | Mental Status | Continence | Mobility | Activity | Nutrition |
| | 5 Alert | 4 Fully controlled | 4 Full | 4 Ambulatory | 3 Good |
| | 4 Apathetic | 3 Usually controlled | 3 Slightly limited | 3 Walks with assistance | 2 Fair |
| | 3 Confused | 2 Minimally controlled | 2 Very limited | 2 Chair fast | 1 Poor |
| | 2 Stuporous | 1 Absence of control | 1 Immobile | 1 Bedfast | |
| | 1 Unconscious | | | | |
| Date | | | | | Total Score |

Sheet 2

| Date | Vital Signs | | | Skin Appearance | Skin Tone | Skin Sensation | Medications | Comments |
|---|---|---|---|---|---|---|---|---|
| | T | P | R | | | | | |
| | | | | | | | | |

Thirty patients, with no pressure ulcer at the time of admission, aged 65 years and over, were studied from extended care facilities. Gosnell (1973) revised the general physical condition to nutritional status, incontinence changed to continence and additional categories were added, namely: demographic details, medical diagnosis, skin appearance, height, weight, vital signs and medication. Following the inclusion of these categories, additional contributory factors were identified as influencing the development of pressure ulcers: diastolic blood pressure of below 60mm/Hg and raised body temperature. Unfortunately no raw data was presented within the paper and the method adopted utilised an adaptation of the Norton Score and therefore did not independently identify contributory factors. Gosnell (1973) did define the meaning of the subcategories for each group heading, but gave no indication as to how the weighting had been derived. For example, within the mental score it was not clear why confusion weighted more than apathy. There was also no explanation to support why Gosnell (1973) included the other parameters selected. Unlike Goldstone & Goldstone (1982) Gosnell (1973) found neither age or sex to be a differentiating factor, nor race. Three of the four patients who developed pressure ulcers had a primary diagnosis within the circulatory disturbance category. Seventeen of the thirty patients, however, had circulatory disorder thus suggesting that the significance as a contributory factor is limited. Medication was also identified by Gosnell (1973) as being an important contributory factor as all four patients developing pressure ulcers were found to be receiving analgesics, cardiotonics and tranquillisers. Two of the patients were receiving two different types of tranquillisers. However Gosnell (1973) went on to identify that twenty six of the remaining patients who did not develop pressure ulcers received these three types of medication and therefore arguably this questions the validity of this as a critical variable.

Waterlow (1985) was still concerned about the apparent lack of knowledge of preventative aids and up-to date treatment relating to the different stages of pressure ulcers. A card was devised as an aide-memoire, designed to raise awareness of causative factors, determine the risk, and give guidance on pressure ulcer grading and whether preventative or active treatment was required (Figure 1.11). It became more than an aide memoire and was utilised across the National Health Service as part of many hospitals' prevention and treatment policies for pressure ulcers.

Assessments were recommended to be undertaken at the time of admission and then repeated at any point when the patient's condition changed. The Waterlow score

is more explicit, incorporating additional factors considered to increase the risk of developing pressure ulcers, such as age, sex and special risk factors i.e. sensory deprivation, high dose anti-inflammatory or steroids, smoking, orthopaedic surgery or factures below waist. The higher the score the higher the risk, with 10-14 indicating 'at risk', 15-19 'high risk' and 20 plus 'very high risk'. The additional factors identified were not evidence based and the weighting, like that of the Norton scale is subjective.

Pritchard (1986) felt that patients on her male medical ward were at risk of developing pressure ulcers, but were not being identified as at risk through the Norton Score. Questioning the sensitivity of the Norton Score, a ward staff meeting was arranged to discuss risk factors that the ward staff felt were important and not sufficiently covered. The ward was called Douglas Ward and therefore the tool designed became known as the Douglas tool (Pritchard, 1986). Three additional aspects were identified by the ward staff and added: nutritional state and low haemoglobin; pain; and skin condition. The scores were weighted according to the perceived level of impact in causing pressure ulcers. Similar to the Norton Scale no substantive evidence was referred to, to support the weighting (Papanikolaou et al, 2007). Following the assessment of 28 patients the trial the staff felt that perhaps each speciality could have varying risk factors affecting the level of risk for each patient. For example steroid therapy, diabetes, cytotoxic therapy and an additional two points would be deducted for each extra risk factor. Pritchard (1986) did not go on to identify the specificity and sensitivity of the tool.

## WATERLOW PRESSURE ULCER PREVENTION/TREATMENT POLICY

RING SCORES IN TABLE, ADD TOTAL. MORE THAN 1 SCORE/CATEGORY CAN BE USED

| BUILD/WEIGHT FOR HEIGHT | ♦ | SKIN TYPE VISUAL RISK AREAS | ♦ | SEX AGE | ♦ |
|---|---|---|---|---|---|
| AVERAGE | | HEALTHY | 0 | MALE | 1 |
| BMI = 20-24.9 | 0 | TISSUE PAPER | 1 | FEMALE | 2 |
| ABOVE AVERAGE | | DRY | 1 | 14 - 49 | 1 |
| BMI = 25-29.9 | 1 | OEDEMATOUS | 1 | 50 - 64 | 2 |
| OBESE | | CLAMMY, PYREXIA | 1 | 65 - 74 | 3 |
| BMI > 30 | 2 | DISCOLOURED GRADE 1 | 2 | 75 - 80 | 4 |
| BELOW AVERAGE | | BROKEN/SPOTS | | 81 + | 5 |
| BMI < 20 | 3 | GRADE 2-4 | 3 | | |
| BMI=Wt(Kg)/Ht (m)$^2$ | | | | | |

**MALNUTRITION SCREENING TOOL (MST)** (Nutrition Vol.15, No.6 1999 - Australia)

A - HAS PATIENT LOST WEIGHT RECENTLY
- YES - GO TO B
- NO - GO TO C
- UNSURE - GO TO C AND SCORE 2

B - WEIGHT LOSS SCORE
- 0.5 - 5kg = 1
- 5 - 10kg = 2
- 10 - 15kg = 3
- > 15kg = 4
- unsure = 2

C - PATIENT EATING POORLY OR LACK OF APPETITE
'NO' = 0; 'YES' SCORE = 1

NUTRITION SCORE
If > 2 refer for nutrition assessment / intervention

| CONTINENCE | ♦ | MOBILITY | ♦ |
|---|---|---|---|
| COMPLETE/ CATHETERISED | 0 | FULLY | 0 |
| URINE INCONT. | 1 | RESTLESS/FIDGETY | 1 |
| FAECAL INCONT. | 2 | APATHETIC | 2 |
| URINARY + FAECAL INCONTINENCE | 3 | RESTRICTED | 3 |
| | | BEDBOUND e.g. TRACTION | 4 |
| | | CHAIRBOUND e.g. WHEELCHAIR | 5 |

**SPECIAL RISKS**

| TISSUE MALNUTRITION | ♦ | NEUROLOGICAL DEFICIT | ♦ |
|---|---|---|---|
| TERMINAL CACHEXIA | 8 | DIABETES, MS, CVA | 4-6 |
| MULTIPLE ORGAN FAILURE | 8 | MOTOR/SENSORY | 4-6 |
| SINGLE ORGAN FAILURE (RESP, RENAL, CARDIAC,) | 5 | PARAPLEGIA (MAX OF 6) | 4-6 |
| PERIPHERAL VASCULAR DISEASE | 5 | **MAJOR SURGERY or TRAUMA** | |
| ANAEMIA (Hb < 8) | 2 | ORTHOPAEDIC/SPINAL | 5 |
| SMOKING | 1 | ON TABLE > 2 HR# | 5 |
| | | ON TABLE > 6 HR# | 8 |

MEDICATION - CYTOTOXICS, LONG TERM/HIGH DOSE STEROIDS, ANTI-INFLAMMATORY MAX OF 4

| SCORE |
|---|
| 10+ AT RISK |
| 15+ HIGH RISK |
| 20+ VERY HIGH RISK |

# Scores can be discounted after 48 hours provided patient is recovering normally


Obtainable from the Nook, Stoke Road, Henlade TAUNTON TA3 5LX
* The 2005 revision incorporates the research undertaken by Queensland Health.

www.judy-waterlow.co.uk

**REMEMBER TISSUE DAMAGE MAY START PRIOR TO ADMISSION, IN CASUALTY. A SEATED PATIENT IS AT RISK**
ASSESSMENT (See Over) IF THE PATIENT FALLS INTO ANY OF THE RISK CATEGORIES, THEN PREVENTATIVE NURSING IS REQUIRED A COMBINATION OF GOOD NURSING TECHNIQUES AND PREVENTATIVE AIDS WILL BE NECESSARY
ALL ACTIONS MUST BE DOCUMENTED

### PREVENTION

**PRESSURE REDUCING AIDS**

**Special Mattress/beds:**
- 10+ Overlays or specialist foam mattresses.
- 15+ Alternating pressure overlays, mattresses and bed systems
- 20+ Bed systems: Fluidised bead, low air loss and alternating pressure mattresses

Note: Preventative aids cover a wide spectrum of specialist features. Efficacy should be judged, if possible, on the basis of independent evidence.

**Cushions:** No person should sit in a wheelchair without some form of cushioning. If nothing else is available - use the person's own pillow. (Consider infection risk)
- 10+ 100mm foam cushion
- 15+ Specialist Gell and/or foam cushion
- 20+ Specialised cushion, adjustable to individual person.

**Bed clothing:** Avoid plastic draw sheets, inco pads and tightly tucked in sheet/sheet covers, especially when using specialist bed and mattress overlay systems
Use duvet - plus vapour permeable membrane.

### NURSING CARE

| | |
|---|---|
| General | HAND WASHING, frequent changes of position, lying, sitting. Use of pillows |
| Pain | Appropriate pain control |
| Nutrition | High protein, vitamins and minerals |
| Patient Handling | Correct lifting technique - hoists - monkey poles Transfer devices |
| Patient Comfort Aids | Real Sheepskin - bed cradle |
| Operating Table Theatre/A&E Trolley | 100mm(4ins) cover plus adequate protection |
| Skin Care | General hygene, NO rubbing, cover with an appropriate dressing |

### WOUND GUIDELINES

| | |
|---|---|
| Assessment | odour, exudate, measure/photograph position |

### WOUND CLASSIFICATION - EPUAP

| | |
|---|---|
| GRADE 1 | Discolouration of intact skin not affected by light finger pressure (non-blanching erythema) This may be difficult to identify in darkly pigmented skin |
| GRADE 2 | Partial thickness skin loss or damage involving epidermis and/or dermis The pressure ulcer is superficial and presents clinically as an abrasion, blister or shallow crater |
| GRADE 3 | Full thickness skin loss involving damage of subcutaneous tissue but not extending to the underlying fascia The pressure ulcer presents clinically as a deep crater with or without undermining of adjacent tissue |
| GRADE 4 | Full thickness skin loss with extensive destruction and necrosis extending to underlying tissue. |
| Dressing Guide | Use Local dressings formulary and/or www.worldwidewounds |

IF TREATMENT IS REQUIRED, FIRST REMOVE PRESSURE

**Figure 1.11** Pressure ulcer risk assessment card produced by Judy Waterlow 1985, revised 2005. Designed as a pocket guide for nurses identifying the scores to be allocated according to various aspects of the patient's condition, what the total score represents and guidance for the prevention and treatment of pressure ulcers (ref.www.judy-waterlow.co.uk).

Bergstrom et al (1987a) were concerned about the subjectivity of the interpretation of the scores and developed the Braden Scale for predicting pressure ulcer risk. The scale was based on a conceptual schema identifying the intensity and duration of pressure and tissue tolerance as the two critical determinants of pressure sores. The intensity and duration of pressure was related to mobility, activity and sensory perception and tissue tolerance to extrinsic and intrinsic factors. The scale consists of six subscales; sensory perception, activity, mobility, moisture, friction and nutrition. Bergstrom et al (1987a) tried to overcome the reliability and validity problems associated with the Norton and Waterlow scores by describing the subscales of the Braden score in more detail. For example, within sensory perception the 4 ratings are completely limited, very limited, slightly limited and no impairment. Completely limited is defined as "unresponsive to painful stimuli, either because of state of unconsciousness or severe sensory impairment, which limits ability to feel pain over most of body surface." With the exception of friction each subscale is rated 1(least favourable) to 4 (most favourable). Friction is rated from 1 to 3, and the Braden scale was the first scale to include friction. A total score of 23 points is possible and a score of equal to or less than 16 was judged to identify the patient as being at risk of developing a pressure ulcer.

The most commonly used risk assessment tools are the Norton scale, Waterlow score and Braden scale (Edwards, 1994). However, many indices for assessing the patient's risk of developing a pressure ulcer have been developed. More than seventeen indices have been identified from the literature, and they have played a significant role in pressure ulcer prevention policies and treatment plans within the National Health Service. These indices have predominantly been developed using subjective judgement of the relative importance of possible risk factors rather than evidence based (Ryan et al, 1989; NHS Centre of Reviews and Dissemination, 1995, Papanikolaou et al, 2007).

#### 1.3.3.2 Validity – Inter-rator Reliability, Sensitivity and Specificity

The validity of assessment tools are concerned with inter-rator reliability, sensitivity, specificity, positive predictive value and negative predictive value. Sensitivity is the proportion of positive tests obtained in subjects who developed pressure ulcers. Specificity is the proportion of the negative tests obtained in patients that did not develop pressure ulcers. The predictive value of a positive test is the proportion of patients with pressure ulcers who were predicted to have them. Finally the predictive

value of a negative test is the proportion of those patients who did not develop pressure ulcers and were predicted not to.

Inter-rator reliability is influenced by staff having different levels of knowledge, skills and experience, which influence their interpretation of both the tool and presentation of patient condition. Lincoln et al (1986) carried out a pilot study to determine inter-rator agreement, face validity, and predictive value of the Norton Score in assessing elderly patients in acute settings. The selection criteria included patients that were 65 years plus, and had an absence of pressure ulcers. The patients were selected by trained nursing assistants. Sample I consisted of seventy three patients and was used to determine inter-rator agreement. Sample II consisted of fifty patients and was used to determine predictive validity. Patients were assessed and scored using the Norton Scale independently by two investigators. Definitions for each of the category were identified, discussed and agreed to facilitate greater agreement. At the end of each data collection the number of same scores were totalled and divided by the total number of patients. The absolute agreement was poor with 28% in week one, 46% in week two, 70% in week three and 10% in week four. Lincoln et al. (1986) compared ratings ± 1 which improved the level of agreement to between 58% to 80%. If the rators scores were then examined to identify whether the patients were simply identified as being at risk or not e.g. score less than 14 or greater than 14 respectively the inter-rator agreement rose to 88%-100% during week 1 - 3, and fell to 60% in week 4. The rators usually agreed on extremes in each category e.g., good or very bad, but often had difficulty discriminating between the inter-ratings e.g., limited verses slightly limited. Each rator found the physical category too broad. The nursing care may have prevented the development of pressure ulcers although the scores were not shared with the staff caring for the patients. Of the five patients who developed pressure ulcers four were surgical patients and the score dropped dramatically after surgery at which time they developed pressure ulcers. This supports the need for continuing assessment in response to patients' condition.

Norton (1989) and Waterlow (1985) acknowledged that assessment tools should be updated in response to advances in technology and medicine because since the tools were originally designed patients are surviving illnesses that once they would not. Norton (1989) increased the level of risk from 15 to 16 and although Waterlow was questioned for not including pain as part of the assessment (Flanagan, 1993) Waterlow has resisted incorporating it within the scale for assessment but has identified is as part of nursing care within the recommendations for pressure sore

prevention (Waterlow, 2005). The rationale for these changes however appear arbitrary and again questions the predictive value of the tools.

Edwards (1995) explored the validity of the Waterlow pressure ulcer risk calculator and found that the inter-rater agreement demonstrated scores, which were both positive and negative, indicating an absence of persistent bias. The gross differences in scoring also occurred through out the study suggesting that the discrepancies were not due to a learning process. Disagreement occurred most within the 'skin-type, build/weight for height and mobility' category. Edwards (1995) concluded that the Waterlow Score should not be used in isolation to decide the distribution of preventative resources. In terms of validity Edwards (1995) found that the initial Waterlow score obtained a sensitivity of 100% and specificity of 10.34%. The predictive value of the positive test was 7.14% and negative test 100%.

The sensitivity of an assessment tool refers to the proportion of actual cases of pressure ulcers in comparison to those who score positively using the risk assessment tool, and the ability of the scale's score to reflect changes. The Norton Scale has been challenged many times. Lincoln et al (1986) demonstrated that the predictive validity of the Norton Score was poor because the two patients identified on admission as being at risk did not go on to develop pressure ulcers. The specificity was also identified to be poor with five of the thirty-four patients, identified not at risk, developing pressure ulcers. The study therefore did not support the direct linear relationship found between pressure ulcer development and Norton Score on admission, identified by Norton et al (1962).

Goldstone and Goldstone (1982) claimed that the Norton Scale had a tendency to over predict, see Table 1.6. Norton (1989), however, felt that once patient's were identified 'at risk' using the assessment tool the intervention of nursing care reduced the number of patients going on to develop pressure ulcers. Goldstone & Goldstone (1982) tried to account for this by having the Norton Score completed by an experienced nurse who was neither a member of the ward staff nor did they discuss the score or it's significance with other staff. No special treatment was given to patients in response to the Norton Score. However, some patients predicted to develop pressure ulcers did not go on to suggesting that the tool is insufficiently sensitive. Norton (1989) stated that the simplicity of the Norton Scale was deliberate because she felt that increasing the sensitivity would influence the reliability of the tool. Norton, however, had been criticised for not including nutrition which she went

on to explain was included in the category for the patient's general physical condition (Edwards, 1995; Flanagan, 1993). This again highlights the importance placed on the individual nurses basic knowledge, understanding and interpretation of each category of the patient's condition in enhancing the validity and reliability of the assessment tool.

**Table 1.6 Classification Correctness using Norton Score <14 as a Predictor (Goldstone & Goldstone, 1982)**

| | | |
|---|---|---|
| | Test Positive (Prone to develop Pressure ulcers) | Test Negative (Free of Pressure ulcers) |
| Developed Pressure ulcers | 16 (A) | 2 (B) |
| Free of Pressure ulcers | 14 (C) | 8 (D) |

The sensitivity, specificity, predictive value of a positive test and predictive value of a negative test for the Norton score can be calculated from the data in Table 1.6. The formula for each are as follows and the values for A,B,C & D are identified in Table 1.6:

Sensitivity (%) = $\frac{A}{A+B}100$ Specificity (%) = $\frac{D}{C+D}100$

Predictive value of a positive test (%) = $\frac{A}{A+C}100$

Predictive value of a negative test (%) = $\frac{D}{B+D}100$

The sensitivity was calculated to be 88.9%, specificity 36.4%, predictive value for a positive test 53.3% and predictive value for a negative test 80%.

The Braden scale has been examined more than other scales for sensitivity, specificity and predictive value of a positive and negative test. The initial studies were undertaken within a rehabilitation unit and institutionalised elderly unit (Bergstrom et al, 1987a). When tested in an intensive care setting the sensitivity and specificity was adversely affected with sensitivity falling from 100% to 83% and specificity from 90% to 64% (Bergstrom et al, 1987b). The percent predictive value of a positive and

negative test was 61 and 85% respectively and it has been recommended that clinical areas should assess the Braden score before implementing clinically and identify the appropriate threshold score for "no risk" and "at risk" status (Edwards, 1994; Braden, 1989; Flanagan, 1993). This suggests that the predictive tool is not sufficiently objective and that the subscales and allocation of scores not sufficiently evidence based.

Possibly it should have been considered that the aim of the risk assessment scales was to reduce the risk by intervention and the intervention should be considered when scoring the patient to reflect whether the intervention is considered to be successfully reducing the risk presented.

Using a sensitive form of assessment tool is an essential part of pressure ulcer prevention (Defloor & Grypdonck, 2005). The majority of risk indices are unvalidated as they have not been subject to rigorous scrutiny in terms of inter-rator reliability, specificity, sensitivity (Edwards, 1994, 1995; NHS Centre of Reviews & Dissemination, 1995; Patterson & Bennett, 1995; Royal College of Nursing, 2001; National Institute Clinical Excellence, 2001). A lack of clarity with some of the clinical assessment definitions within the risk indices are open to broad interpretation by the rator, which will vary according to their knowledge base and clinical experience, so potentially leading to an inaccurate assessment. This indicates that many risk assessment indices should only be used as an aide memoire or educational tool (Royal College of Nursing, 2001).

Perceived risk to developing pressure ulcers affects patient management. The contributory factors identified through clinical assessment, and therefore level of risk identified, directly influence the type of care i.e. positioning, frequency of change in position, and resources utilised e.g. type of pressure reducing surface.

As current risk assessment tools generally lack objectivity, sensitivity and/or specificity a more accurate and reliable assessment tool is key to ensuring the successful care for the patient, and the effective and efficient utilisation of resources in the prevention of pressure ulcers. It is also essential that a true risk assessment tool be identified in order to permit evaluations of the effectiveness of pressure reducing surfaces and other treatments for the prevention of pressure ulcers.

## 1.4 Discussion and Hypothesis

### Discussion

The need to prevent pressure ulcers is a necessity as a large majority of pressure ulcers are preventable (Hibbs 1982) and therefore the pain, discomfort and distress to patients and their family/carers, and costs to the health service could be significantly reduced.

New assessment tools continue to be developed, but are still adaptations of existing assessment tools. Therefore they continue to be more subjective than objective with poor inter-rator reliability. An ideal measure of risk should be both sensitive and specific. Vagueness of the factors allows broad interpretation by the user (Carlton, 1990). Norton (1989), however felt that regardless of the sophistication, no scale could be more than an indicator. The importance of evaluating the appropriateness of the tool to each clinical setting has been emphasised by various studies (Williams, 1991). Bergstorm and Morison, as sited by Flanagan (1993), also agreed that tools are not a substitute for sound clinical judgement, they are merely a guideline and that having a good foundation of knowledge on the aetiology of pressure ulcers and the contributory factors to the various categories in the multitude of assessment tools available is vital if they are to be used effectively. So fundamentally they are only as good as the nurse using them.

The current recommendation of the Royal College of Nursing (RCN, 2001) and the National Institute for Clinical Excellence (NICE 2001) are that clinical judgement is as good as if not better than assessment tools. This is a relative, not absolute assessment and could be better or equal to the poor performance of current assessment tools. The use of clinical judgement raises concerns due to the subjectivity and poor inter-rator reliability, especially in today's climate of staffing pressures, sometimes poor skill mix and different levels of knowledge and experience. It is evident that there are currently no evidence-based indices for assessing true risk of developing pressure ulcers, and more objective methods for assessing individual patient risk need to be developed. The key is to prevent pressure ulcers from occurring, by identifying those truly at risk and intervening to reduce/remove the risk.

To try and overcome the difficulties with the subjectivity of current assessment tools researchers have been exploring the use of transcutaneous monitoring of oxygen ($tcPO_2$) as a retrospective indicator of tissue viability. The variability of $tcPO_2$ levels between anatomical sites, and hard and soft tissues has been explored (Dowd et al (1983), Rodrigues et al (2001), Seiler & Stahelin (1979)) and the use of $tcPO_2$ monitoring to assess the success of replanted limb parts (Matsen et al, 1980). Transcutanous partial pressure of oxygen monitoring has also successfully been used to identify the wound healing potential for above or below knee amputations (Pinzur et al, 1992).

The response of $tcPO_2$ in local tissue to applied pressure has been studied and found that the level of pressure required to reduce $tcPO_2$ levels and the pattern of response varies considerably between individuals (Bader & Gant, 1988; Bader, 1990a &1990b; Knight et al, 2001), indicating the varying degrees of tolerance individuals have to pressure. However, to date the sample size of studies has been small and pressures used were believed to be representative of the physiological range for interface pressures. The studies have therefore not been based upon an understanding of how sacral tissue responds when subjected to pressure exerted through own body mass. Finally the responses observed have not been correlated to clinical outcome.

**Hypothesis**

From the preceding review it is apparent that an objective tool should be useful in patient management. To avoid inter and intra-rator variation the objective tool must be based on physiological measurements which are sensitive to changes in tissue perfusion. The role of the current assessment tools can then purely be as an aide memoir in relation to reminding staff of some of the contributory factors to be considered when considering interventions to reduce an individuals' level of risk. The key is to prevent rather than react once damage has occurred. The aim of this research is to develop a method for identifying the peak sacral pressure for each individual and monitor the effect of the pressure on $tcPO_2$ and $tcPCO_2$. The monitoring of the transcutaneous oxygen and carbon dioxide will be used as an indicator of tissue tolerance and prospective indicator of the risk of tissue damage as a consequence of pressure. The methods available for measuring interface pressure and tissue perfusion are discussed in Chapter 2.

The responses will be analysed to identify:

i) $tcPO_2$ and $tcPCO_2$ changes for normal tissue subjected to physiologically relevant interface pressure;

ii) relative changes and associated relationships;

iii) $tcPO_2$ and $tcPCO_2$ changes for normal tissue subjected to varying interface pressure.

The hypothesis being tested throughout the work is that pressures exerted by an individuals' body mass on sacral tissue in the supine position are close to a critical perfusion threshold, and because the capillary blood flow is influenced by interface pressure, when blood flow is sufficiently reduced it results in a loss of oxygen and an excess accumulation of carbon dioxide in the tissue. These parameters may be measured and used to assess individuals' tolerance to pressure exerted through their own body mass, and used as a predictor of risk of pressure ulcer formation.

# Chapter 2 Measurement Techniques for Tissue Perfusion

The primary cause of pressure ulcers is when tissue is exposed to a magnitude and duration of pressure which occludes local blood flow sufficiently to cause tissue ischaemia and necrosis. In order to identify tissue viability, measurement techniques are required to identify the pressures individuals' are exposed to when sitting and/or lying on surfaces. The effect the pressure has on tissue perfusion and the ability of oxygen to diffuse to and carbon dioxide to diffuse from tissue cells requires further examination. This chapter will explore methods available for identifying tissue perfusion and interface pressure.

## 2.1 Non-invasive measurement techniques for tissue perfusion

Laser Doppler fluximetry and transcutaneous partial pressure monitoring of oxygen are both methods widely used to evaluate tissue perfusion and will be explained and discussed in relation to the strengths and weaknesses of each technique.

### 2.1.2 Laser Doppler Fluximetry (LDF)

LDF provides a non-invasive measure of blood flow in the very small blood vessels of the microvasculature. The depth of tissue sampled is typically 1mm, with the capillary diameters of 10 microns and the velocity spectrum measurement typically 0.01 to 10mm/s. Therefore LDF can measure blood flow in the capillaries close to the skin surface associated with the nutritional blood flow, and the flow in the underlying arterioles and venules involved in regulation of skin temperature. The deep horizontal plexus at the subcutaneous dermal junction is not measured.

The technique depends on the Doppler principle whereby low power light from a monochromatic stable laser (see Figure 2.1) is scattered by moving red blood cells and as a consequence the frequency of light is broadened. If the light is reflected from objects moving towards the probe the wave form bunches together resulting in a higher frequency. If the object is moving away the wave spreads out and the frequency reduces (see figure 2.2). The frequency broadened light, together with laser light scattered from static tissue, is photodetected and the resulting photocurrent processed to provide a blood flow measurement (Bongard & Bounameaux, 1993).

LDF is used to detect velocity and volume through the number of moving particles. However, it is sensitive to all movement and therefore is prone to movement artefacts. The probe is mounted using double sided tape to avoid problems of pressure loading.

The environment temperature needs to be controlled to reduce the changes in perfusion as a consequence of temperature.

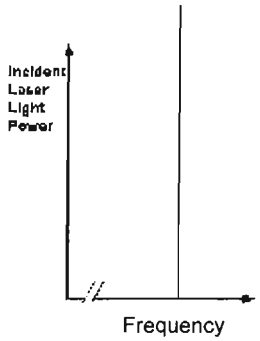



Figure 2.1 A representation of the spectral frequency of laser light transmitted (Monochromatic light)

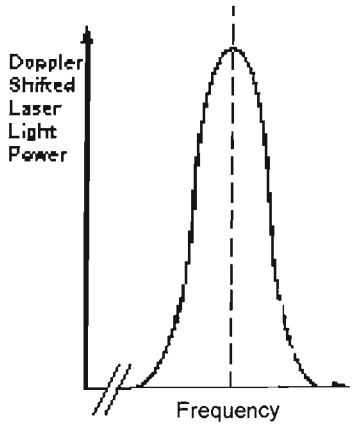



Figure 2.2 The spectral frequency of laser light detected is broadened as a result of scatter from moving red blood cells

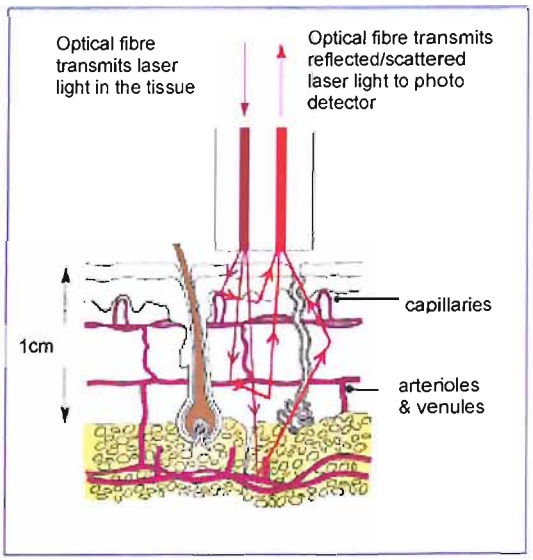



Figure 2.3 Diagram showing laser light being emitted and detected

(Reference for Figures 2.1 – 2.3
http://www.moor.co.uk/files/Theory/Moor_Laser_doppler_theory_Issue_1.pdf)

A laser Doppler signal may still be observed when blood circulation has stopped. Although flow has ceased cell movement may still be occurring and this signal is detected and termed biological zero. However it is effective at studying fast changes of skin blood flow e.g. flux motion activity and flow variations during provocative tests. The velocity and flow is relative rather than absolute because the light strikes the red blood cells at many different angles due to the scatter as it penetrates the upper non-vascular layers, and the complex geometry of the capillaries in the dermis (Holloway G.A, 1983).

The basic structure of the skin is quite variable in different areas of the body, with age and pathology. Tissue perfusion exhibits a considerable degree of heterogeneity therefore measurements only millimetres apart may present very different results (Harrison et al, 1993). This may be the result of variability in the distribution of the microcirculation (Braverman et al, 1990), or physiological function (Harrison et al, 1988). This limits the application of LDF because the interpretation of one area can not be considered representative of other local areas for the same individual. In addition variation in flux has also been noted for the same individual and site at intervals of hours, days and weeks. Therefore the technique has been used to observe changes in flux in response to physiological challenges and has previously been used to examine the effect of temperature and pressure on local sacral skin blood flow (Shubert & Fagrell, 1989; Brienza et al, 2005).

### 2.1.2 Transcutaneous Partial Pressure of Oxygen and Carbon Dioxide Monitoring

Transcutanous partial pressure monitoring of oxygen is widely used to assess tissue perfusion. Human skin releases small amounts of oxygen and carbon dioxide, which Baumberger and Goodfriend(1951) demonstrated by sealing a finger in phosphate buffer solution at 45°C. They found that the oxygen tension approached that of arterial oxygen tension. Oxygen tension is also referred to as partial pressure. However it wasn't until the 1970's that transcutaneous blood gas measurements were taken by heated miniaturised oxygen and carbon dioxide sensors (Huch and Huch, 1983, Eberhard et el, 1976). The measurement is polarographic, based on an electro-chemical electrode chain. A platinum cathode acts as the sensor electrode and the silver reference electrode the anode. The electrode is covered by a membrane to stabilise the conditions of diffusion.

The oxygen electrodes are based on the Clark-type polarographic $PO_2$ electrode, where oxygen that diffuses through the skin is electrochemically reduced by the cathode as a result of a current-generating process:

$$O_2 + 2H_2O + 4e^- \leftrightarrows 4\ OH^-$$

The electrolyte solution usually contains potassium chloride, the cathode of platinum or pure gold, and the anode is silver ring coated with silver chloride. At the anode the following reaction occurs:

$$4\ Ag + 4Cl^- \rightarrow 4\ AgCl + 4e^-$$

The reduction of oxygen generates a current which is fed via the cathode into the $PO_2$ channel, where it is converted into voltage and digitalized. The flow of current generated by the polarising voltage(mV) is linearly proportional to the oxygen tension at the cathode surface. The voltage is conveyed to a microcomputer and reconverted to display $PO_2$ in mmHg or kPa.

The carbon dioxide ($CO_2$) electrode is based on the Stow-Severinghaus type electrode (Stow et al, 1957: Severinghaus & Bradley, 1958). The sensor consists of a pH sensitive glass electrode which is a concentric silver/silver chloride (Ag/AgCl) reference electrode. As $CO_2$ diffuses from the skin and through the membrane into the electrolyte solution the $CO_2$ reacts with the water forming carbonic acid ($H_2CO3$) which immediately dissociates into bicarbonate($HCO_3^-$) and hydrogen ($H^+$).

$$H_2O + CO_2 \leftrightarrows H_2CO_3 \leftrightarrows H^+ + HCO_3^-$$

The changes in hydrogen ions in the electrolyte imply changes in the pH. As the pH changes, the voltage between the glass and reference electrode changes. The pH change is conveyed to a $CO_2$ reading on the basis of the linear relationship between pH and the negative logarithm of transcutaneous partial pressure of carbon dioxide.

The combined sensor consists of a heating element, 2 temperature sensors, a Clark type oxygen electrode and Severinghaus-type carbon dioxide electrode. There is a common diffusion membrane and electrolyte. The hydroxyl ($OH^-$) ions created at the cathode are buffered by the electroyte.

The electrode is attached to the skin and the heat is transferred from the heating element to the skin via the silver body. The heat produces local vasodilatation, increasing permeability of the skin to oxygen and carbon dioxide.

Measurement of the transcutaneous oxygen tension ($TcPO_2$) was successfully used to monitor the oxygenation of arterial blood in neonates (Huch et al, 1976). However the results for adults did not correlate as well with arterial oxygenation. Evans and Taylor (1967) showed an increase in partial pressure of oxygen of the skin following drug induced hyperaemia leading them to conclude that there was a large gradient between the arterial blood compartment and the surface of the skin. This may be due to the resistance of the skin to oxygen diffusion by the stratum corneum, use by the dermis for metabolism, and the effective rate of cutaneous blood flow.

The electrode therefore has a thermistor-controlled heating element. The temperature of the electrode can be regulated from 37°C to 45°C. This enables the electrode to heat the skin creating local hyperaemia and an increased blood flow. The increased availability of oxygen locally enables the oxygen to pass through the diffusional resistance offered by the extravascular dermo-epidermal spaces in order to reach the sensors. The current in the sensor is linearly proportional to the $PO_2$ signal.

The temperature of the sensor has been widely debated. Mani et al (1986) were able to differentiate between ulcers of different aetiology using a sensor temperature of $43^0$C. However when a temperature of $37^0$C was used within the same study no changes in TcPO2 were noted. Mani et al (1986) also found that there was no significant difference when the TcPO2 was measured at 43, 44 or $45^0$C. So although the electrode falsely raises the local temperature this factor is consistent and is required for accuracy and consistency of the readings.

$TcPO_2$ measurements have since been used in clinical practice to select the level of amputation in patients with critical limb ischaemia (Ratliff et al 1984), to understand the efficacy of vasoactive drugs (Romanelli et al 1991), to investigate the degree of peripheral arterial occlusion (White et al, 1982) and for the evaluation and management of chronic wounds (Mani, 1985). Measuring tissue oxygenation using this technique is non-invasive. The European Consensus Document on critical limb ischaemia (Tooke, 1990) recommended that $TcPO_2$ measurements be used to define ischaemic skin. A $TcPO_2$ value of $\leq$ 10mmHg (1.33kPa), which does not increase on inhalation is accepted as evidence of ischaemia.

The stratum corneum offers resistance to oxygen diffusion and therefore it has been suggested that this is reduced by using a tape stripping technique Jaszczak & Sejrsen, (1987) and Takiwaki et al (1991). However, the overall condition of the patient must be considered, as the skin may be very friable and susceptible to damage.

A control site is used as well as the site of interest to enable the identification of variations in the TcPO2 due to variations in the systemic oxygen delivery. In studies of leg ulcers the subclavicular area is used as the control site and is utilised to calculate the regional perfusion index (RPI). This eliminates the variability due to systemic changes in oxygen delivery (Hauser & Shoemaker,1983).

Romanelli and Falanga (1999) describe how $T_cPO_2$ monitoring has successfully been used to detect the post occlusive reactive hyperaemia response. This is used to determine early diagnosis of microvascular disease and changes in local oxygen availability around a wound. Slagsvold et al (1988), caused temporary ischaemia for 3 minutes by using a pneumatic blood pressure cuff during which the TcPO2 decreased to 0mmHg. Upon release of the cuff the time taken for the TcPO2 levels to start rising is recorded and termed the oxygen reappearance time (ORT). The oxygen recovery index (ORI) refers to the oxygen diffusion rate (mmHg/min) at the steepest part of the recovery curve. The ORT indicates the rate of reperfusion, which is slower in patients with peripheral vascular disease and the ORT is felt to be related to the functional state of the microcirculation and is dependant on the number of functional capillaries.

More latterly interest has grown in exploring the viability of tissue and variation in the tolerence to pressure, between individuals Bader & Gant (1988), found that for a 62year old subject with multiple sclerosis, when the pressure applied to the sacral area was increased to 46mmHg (6.1kPa), the blood supply was impaired sufficiently to reduce oxygen levels to 2.7kPa (20mmHg). In contrast, a pressure of 100mmHg (13.3kPa) was required to reduce oxygen levels below 2.7kPa (20mmHg) for a 61 year old subject following a cerebral vascular accident. This could possibly be linked with the patient being hypertensive, therefore affecting the capillary closing pressure. Bader & Gant (1988), noted that there was a biphasic response. In the first phase there is compression of the soft tissues but the integrity of the blood circulation is maintained, and in the second phase the protective mechanism is minimal and relatively small changes in applied pressure produce a significant reduction in $tcPO_2$ levels. No obvious trends were identified from the results of the 20 subjects, however interestingly the 3 subjects that

had had a history of pressure sores had relatively low values of applied pressure required to produce arterial occlusion. Arterial occlusion was identified by $tcPO_2$ levels falling to 0kPa and the pressures required were 40, 45 & 47mmHg (5.3, 6.0 & 6.3kPa).

Bader (1990a), demonstrated that with successive cycles of loading applied to the sacral tissue of healthy individuals the effect is diminished implying a vasomotor control mechanism. This would invalidate any results gained through repeating loading of sacral tissue within a short time period.

When sufficient prolonged pressure is applied to the skin, the partial or total occlusion of underlying vessels not only impairs the supply of oxygen and nutrients to satisfy the metabolic demands of the tissue, but also the removal of waste products via lymphatic and venous drainage causing an accumulation. As already discussed carbon dioxide is one of these metabolites, and clear relationships between depressed levels of $tcPO_2$ and elevated levels of $tcPCO_2$ have been identified by Bader (1990a), and Knight et al (2001).

### 2.1.3 Summary

Laser Doppler fluximetry is helpful in the quantification of flow in the microvascular bed and has been used effectively to explore fast changes of skin blood flow such as changes in blood flux as a consequence of applied pressure, effect of temperature and the hyperaemic response (Brienza et al, 2005; Noble et al, 2003; Schubert & Fagrell, 1989). However the nature of the technology makes it very sensitive to movement, which is more difficult to control for the patient population in comparison with healthy volunteers. It also examines the velocity and volume of blood flow, but not the quality of the blood in relation to oxygenation or the effectiveness of the oxygenation of surrounding tissues, and the removal of metabolites.

$TcPO_2$ monitoring however does reflect the relative oxygenation of tissue, and carbon dioxide monitoring will also act as an indicator of the level of effectiveness of tissue perfusion, as an increase in levels will represent less efficient clearance. This technique has already successfully been used by Bader & Gant (1988), Bader (1990a), Knight et al (2001) and many others as an indicator of tissue oxygenation. This research is intended to extend and understand the effects of pressure on tissue viability further through studying the response of oxygen and carbon dioxide levels in response to the force created through individuals' own body mass.

## 2.2 Measurement Techniques for Interface Pressure

### Introduction

The accurate measurement of interface pressure is important in being able to identify the levels of pressure tissues are naturally exposed to and the effectiveness of the design and use of pressure reducing surfaces. It is also used in understanding the differences between individuals due to their body weight, size, tissue distribution and posture. Many attempts have been made to determine the minimal degree and duration of pressure required to consistently cause tissue damage with the aim that by reducing the pressures below this level and controlling the duration of time pressure ulcers could be prevented (Kosiac, 1961, Husain 1953 & Dinsdale, 1974). Measurement of interface pressure is subject to great variability with differences between anatomical sites. Even on the same individual differences are caused by small changes in posture. There are also differences between the type of pressure monitoring system and interpretation of result e.g. whether the maximum or average is quoted. Any technique designed to measure interface pressure will have an effect on the very parameter it is trying to measure (Swain & Bader, 2002).

The sensors need to comply with several criteria to successfully measure the interface pressure over bony prominence. They should be thin, soft and flexible so they can conform to the shape of the body and surface, and be of small diameter in relation to the bony prominence. It has been suggested that the diameter to thickness ratio should not be less than 10:1 (Barbenel J. 1991).

### 2.2.1 Electro Pneumatic Sensors

Electro pneumatic sensors are available as individual sensors or as sheets. The advantage of individual sensors is that if they are smaller than the area of interest therefore reducing the error. It is also less likely to compromise how the surface that an individual is sitting or lying on conforms to an individuals' body shape and weight, which in itself would influence the interface pressure.

The equipment is a closed system and the pneumatic sensors have thin flexible walls with electrical contacts on opposing internal surfaces. The sensor is placed at the patient- support surface interface and inflated with air to separate the electronic contacts on the walls of the cells. The cells are then allowed to deflate and the pressure at which the electrical contacts form an electrical connection is interpreted as the interface

pressure. Due to the limited number of cells, identifying the point of maximum pressure has been hampered using this system because the pressure distribution is influenced by subtle body movements and changes in the support surface influencing the pressure distribution.

Allen et al (1993) examined the repeatability and accuracy of 3 different systems and the Talley Pressure Evaluator SA500, was found to be the most accurate with a system error of 12% ± 1 and repeatability of ±0.07kPa. When several systems were compared by Ferguson-Pell& Cardi (1993) the Talley pressure monitor (96 sensors, Talley Medical, Romsey, Hants) was again found to be the most accurate, stable and reproducible. However, application is limited by ease of use, speed and data presentation. Gyi et al (1998) also found that if only 75% of the sensor face was covered then the reading was only 82% of the correct value.

### 2.2.2 Force Sensing Array

The large array systems have the advantage of many sensors therefore enabling the identification of the point of maximum pressure. As the readings are taken in real-time changes occurring due to slight postural changes or changes in the support surface can be monitored. Therefore changes in position or surface can also be assessed to identify the impact of changing position or surface because although one pressure point may have been reduced, another location may have increased.

Force sensing array systems generally rely on pressure-induced changes in either resistance or capacitance materials within the sensors which produce an electrical output. The pressure sensitive material has to be sufficiently soft and compliant to obtain sufficient sensitivity. If the flexibility of the surface is insufficient to deform to the same extent as the surface that the individual is sitting or lying the accuracy of the measurements will be compromised (Swain & Bader 2002). Most soft materials are time-dependant, therefore showing signs of creep and hysteresis (Barbenel J. 1991). Over time the effect of creep and hysteresis have been reduced by the development and application of algorithms.

When the Tekscan and Force Sensing Array (FSA, Vista Medical) were compared by Ferguson-Pell& Cardi, 1993, the FSA was rated well in clinical application but demonstrated hysteresis (± 19%) and creep (4%). The Tekscan showed more substantial hysteresis (± 20%) and creep (19%), but was preferred for the real time display.

Creases in the sensor array mat can give falsely high readings therefore care must be taken when positioning the mat.

### 2.2.3 Summary

Although electro pneumatic sensors are more free from artefact than force sensing arrays it is not possible to predict the point of highest pressure or the overall changes in pressure distribution that a change in position or surface will have. The pressure sensing array is intrinsically less accurate, but ensure that the sampling in terms of the impact of pressure on tissue perfusion is undertaken at the point of highest pressure (Bain et al, 2003). Real time is helpful to assess changes that may be occurring due to slight changes in posture and the maximum pressure is of interest rather than the average because the tissues likely to be most vulnerable are those exposed to the highest pressure.

In order that the point of peak pressure can be identified for the proposed research the force sensing array will be the interface pressure monitoring system of choice. The levels of hysteresis will need to be measured and taken into account in the research methodology, but from the work of Ferguson-Pell& Cardi (1993) the force sensing array (FSA) system manufactured by Vista is more reliable.

# Chapter 3 Validation of Measurement Techniques

This chapter will discuss a series of experiments to test the accuracy and reliability of the technology selected and methodologies developed for:

i) identifying the level and location of peak pressure exerted sacrally through an individuals' own body mass;
ii) identifying an acceptable technique for the reapplication of peak sacral pressure;
iii) identifying the baseline level of oxygen and carbon dioxide perfusion in sacral tissue;
iv) measuring the variation of transcutaneous partial pressure of oxygen and carbon dioxide changes associated with the reapplication of peak sacral pressure.

## 3.1 Accuracy of the Force Sensing Array Selected to Identify the Level and Location of Peak Sacral Pressure

The ultra thin Force Sensing Array (FSA) torso mat was selected to measure the interface pressure. It is manufactured by Vista, and is 0.92m by 0.52m in size giving a mat area of 0.49$m^2$ and sensing area of 0.42$m^2$. There are 1024 sensors, with each sensor measuring 1.5 x 1cm, and arranged 32 by 32 with a calibrated force range of 0-300mmHg.

The FSA uses a piezo resistive, semi conductive, polymer which is sandwiched between two layers of highly conductive rip stop nylon fabric. This sandwich is then encased in a protective cover of polyurethane, followed by lycra. The changes in resistance which result from different pressures on the piezo resistive, semi-conductors are interpreted by an interface module and relayed to the computer where they are displayed as pressure values.

To ensure conformity of the mat to the surface of the sacrum, when sitting or lying, the layers are allowed to move freely. This combined with the lycra cover is to minimise an effect known as hammocking. Hammocking occurs when there are restrictions to the surface conforming to the shape of the sacrum, resulting in the possible increase in interface pressure. The mat is 0.36cm thick and the technology measures the highest forces exerted anywhere on the sensor in contrast to other technologies that average the force. This enables extremely localised areas of peak pressure to be identified.

The mat is latex free and is supplied with disposable isolation bags to protect against, contamination by infection or urine, and reduce shear forces. The accuracy stated by the company is ± 10 per cent with 5 per cent attributable to creep and 5 per cent to hysteresis. Creep is a process whereby a signal may change due to an equilibration process. Hysteresis occurs when the value of a signal varies according to the increase or decrease of another parameter. The FSA mat is surface conforming. Ninety two percent of the maximum sensor output is achieved at 0.1 seconds, ninety four per cent at 20 seconds and ninety nine per cent at 3 minutes.

### 3.1.1 Calibration

Calibration of any item of equipment is essential to ensure the accuracy of equipment is maintained, and should be undertaken in accordance with manufactures guidelines. The accuracy in relation to the claims of the manufacturer is tested to establish any additional errors that need to be considered.

The ultra thin FSA torso mat was calibrated in accordance with the manufacturer's guidelines using the calibration jig provided by the manufacturer. The mat is positioned on the bottom platen with the top side facing up and without the isolation bag. Then the inflator bag is positioned centrally over the mat to ensure equal pressure exerted over the mat's surface. The second platen is then positioned on top forming a sandwich which then slides into the calibration rig (Figures 3.1a to 3.1f).

The auto calibration system ensures a constant pressure is applied for calibration, which is preferential to the manual calibration system where consistency is difficult to maintain. The auto calibration programme exerts a known pressure to the FSA mat by inflating the inflator bag to a set pressure. Initially 300mmHg is applied and held to allow for creep. Then the inflator bag deflates to the minimum pressure and the sensors allowed to equilibrate. 150mm/Hg is then applied and held to calibrate for creep. The inflator bag then deflates again to the minimum pressure and the sensors allowed to rest. The inflator bag is then re-inflated to exert 300mmHg and deflated to minimum pressure. The sensors are then left to equilibrate before the process is repeated once more. During the calibration process sample pressure readings are measured as the pressure is increased to the maximum pressure, and then as the pressure is decreased from the maximum pressure down to zero. This captures the extent of hysteresis, and the FSA software uses a hysteresis correction algorithm to correct the pressure accordingly.

Figure 3.1a Calibration jig showing lower platen

Figure 3.1b Force sensing mat positioned on lower calibration jig platen

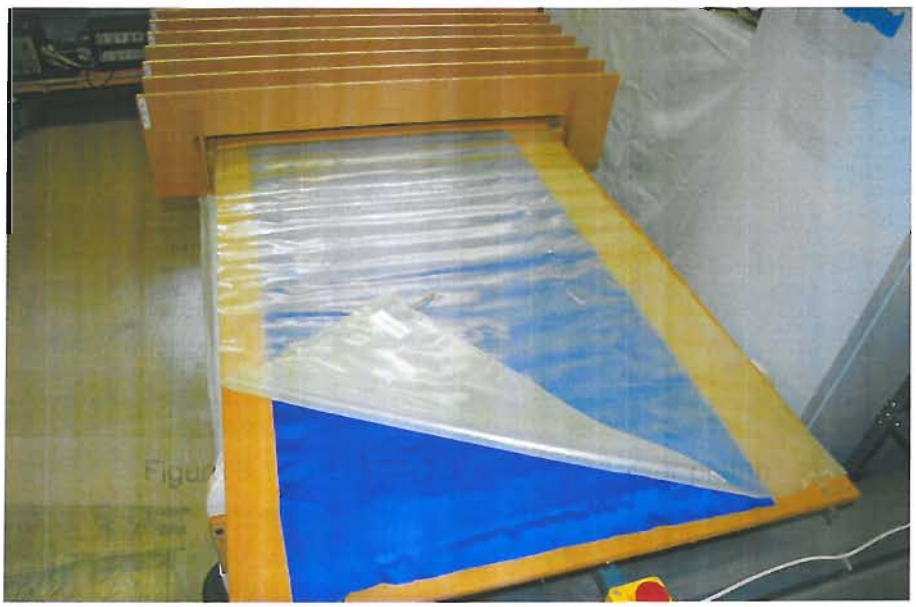


Figure 3.1c Force sensing mat with inflator bag in position

Figure 3.1d Calibration jig showing upper platen in position

Figure 3.1e Calibration "sandwich" inserted and connected to auto-calibration pump


Figure 3.1f Calibration "sandwich" inflated

### 3.1.2 Validation of Calibration

In order to validate the accuracy of the auto-calibration programme the ultra thin FSA mat was left in the calibration jig following auto-calibration. Known pressures were applied using a hand air pump, which was attached to one of the two pieces of tubing connected to the inflator mat and a sphygmomanometer, accurate to ±1mmHg (see Figure 3.2). Pressure was increased by increments of 20mmHg, from 20mmHg up to 300mmHg. The inflator mat was then deflated by 20mmHg decrements from 300mHg down to zero. This enabled a comparison of applied pressure verses measured pressure. The sample rate was set to take one frame every 0.3 seconds (3.33Hz) and readings taken for a period of 30 seconds once the required pressure was reached. Therefore, for each level of pressure 100 frames were taken and the mean of the frames was used as the FSA reading at that pressure. This approach also enabled an assessment of the effects of hysteresis.

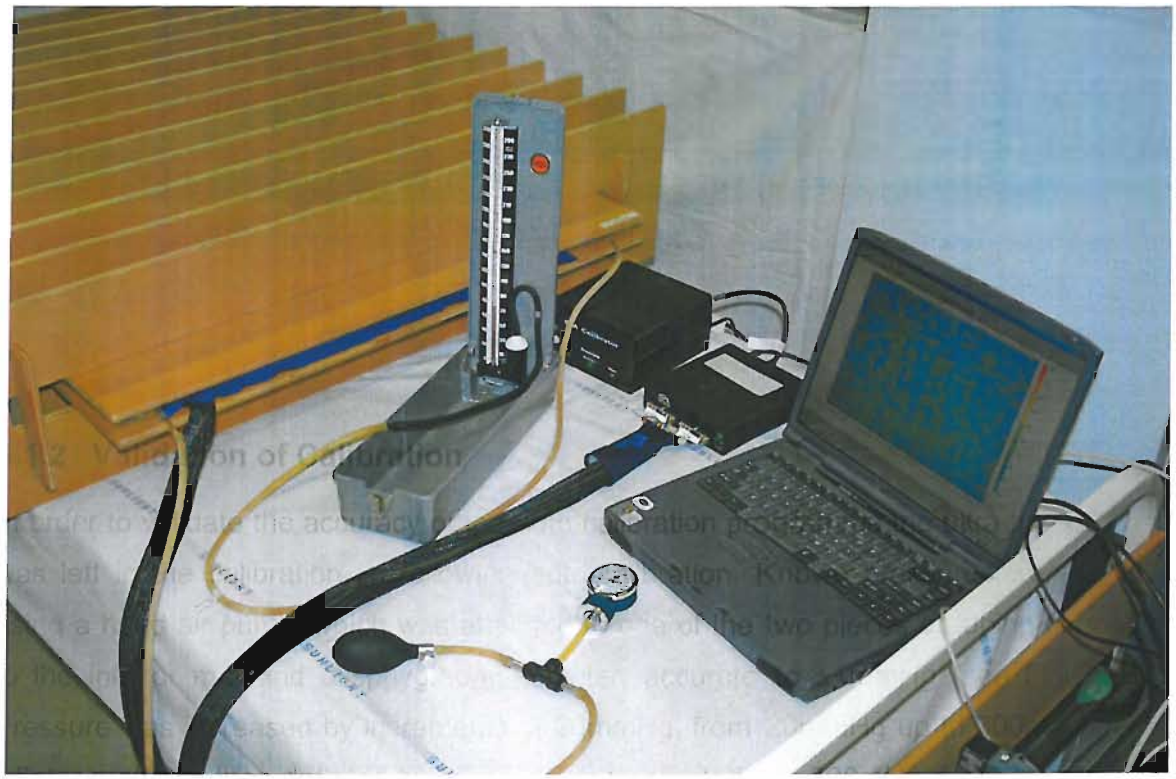


**Figure 3.2 Equipment setup for validation technique**

### 3.1.3 Calibration Results

Table 3.1 indicates that the percentage error ranged from 27.6% at 20mmHg to 5.5% at 160mmHg and the sensors reached saturation at 280mmHg. This exceeds the margin of error stated by the company of ±10%. Through the method of least squares the line of best fit was found ($y = x + 5$) and is shown in Figure 3.3, where y represents the measured pressure and $x$ represents pressure applied. The equation indicates an acceptable association between the actual pressure and the measured pressure. 5 mmHg

is the value with no applied pressure and represents a small offset to the values measured. The manufacturer's identified the threshold noise to be set at 3mmHg which supports this result.

The % error from the pressure is calculated using the equation ((B-A)/A) x100 with A representing the Actual pressure applied and B representing the mean pressure reading using the FSA mat. The coefficient of variation (%) for each of the actual pressure values was calculated using the equation SD/$\overline{X}$ x 100.

The coefficient of variation demonstrates that below 60mmHg the measurements may be subject to between 3-9% variation, and is consistently higher between 20 to 60 mmHg than for other pressures tested.

With 280mmHg applied to the FSA mat the sensors had reached saturation with all sensors reading a maximum measurement of 300mmHg.

When pressure was decreased percentage error increased. Table 3.2 indicates that 19.9 is the lowest percentage error at 220mmHg and 37.9 the largest percentage error at 40 mmHg. From Figure 3.4 the relationship can be described such that $y = 1.18x + 9.4$ and $R^2 = 0.9987$.

### 3.1.4 Conclusion and Discussion

The agreement, particularly on the increasing pressure was encouraging. However, the percentage error was greater than anticipated over the total range of pressures. The range of pressures anticipated to arise as a consequence of an individuals' own body weight are between 30-80mmHg over the sacral area. The error for this range is between 9.9% -12.4% when the pressure was being increased, and is in the range where individual measurements are subject to the greatest variation. This is much closer to the manufacturer's statement of ±10%. However, the percentage error when the pressures were being decreased fell significantly outside of this at 28.9% - 37.9%. Two potential sources for error are identified:

i) Hysteresis Correction - The software supplied by the manufacturer is designed to correct for hysteresis. If the pressures are increasing, the calculated correction factor is taken from the increasing pressure curve of the calibration file. If the pressure is decreasing the calculated calibration factor is taken from the decreasing pressure curve.

**Table 3.1 FSA Mat Measured Pressure Against Actual Pressure - Pressure Increasing**

| (A) Actual pressure (mmHg) | (B) Mean FSA reading (mmHg) | SD of the reading (mmHg) | Error (%) | Coefficient of Variation (%) |
|---|---|---|---|---|
| 20 | 25.5 | 1.7 | 27.5 | 6.8 |
| 40 | 43.9 | 1.5 | 9.8 | 3.5 |
| 60 | 67.4 | 1.8 | 12.4 | 2.7 |
| 80 | 93.0 | 2.0 | 12.1 | 2.1 |
| 100 | 109.2 | 2.1 | 9.2 | 2.0 |
| 120 | 129.9 | 2.7 | 8.3 | 2.1 |
| 140 | 149.4 | 2.8 | 6.7 | 1.9 |
| 160 | 168.9 | 3.2 | 5.5 | 1.9 |
| 180 | 190.3 | 3.7 | 5.7 | 1.9 |
| 200 | 208.3 | 4.6 | 4.2 | 2.2 |
| 220 | 231.8 | 4.4 | 5.3 | 1.9 |
| 240 | 251 | 5.5 | 4.6 | 2.2 |
| 260 | 276.4 | 4.6 | 6.3 | 1.7 |
| 280 | 300 | 0 | 7.1 | 0 |

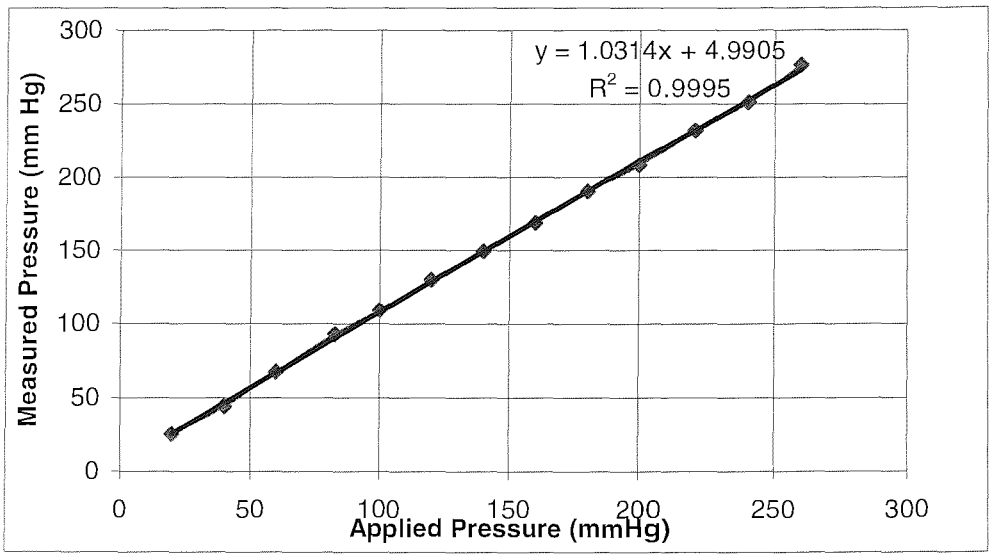



**Figure 3.3 FSA Readings Taken Using Calibration Rig - Increasing Pressure**

**Table 3.2 FSA Mat Measured Pressure Against Actual Pressure - Pressure Decreasing**

| Actual pressure (mm/Hg) | FSA reading - decreasing pressure (mm/Hg) | Average SD (mm/Hg) | Error (%) | Coefficient of variation (%) |
|---|---|---|---|---|
| 240 | 293.6 | 4.8 | 22.3 | 1.6 |
| 220 | 263.7 | 5.5 | 19.9 | 2.1 |
| 200 | 242.8 | 5.0 | 21.4 | 2.1 |
| 180 | 223.9 | 4.6 | 24.4 | 2.0 |
| 160 | 198.7 | 4.3 | 24.2 | 2.2 |
| 140 | 175.1 | 3.8 | 25.0 | 2.2 |
| 120 | 153.9 | 3.4 | 28.2 | 2.2 |
| 100 | 128.9 | 3.4 | 28.9 | 2.6 |
| 80 | 106.9 | 2.7 | 33.7 | 2.5 |
| 60 | 82.7 | 2.6 | 37.9 | 3.2 |
| 40 | 55.1 | 2.1 | 37.8 | 3.7 |
| 20 | 27.0 | 2.4 | 35.2 | 9.0 |

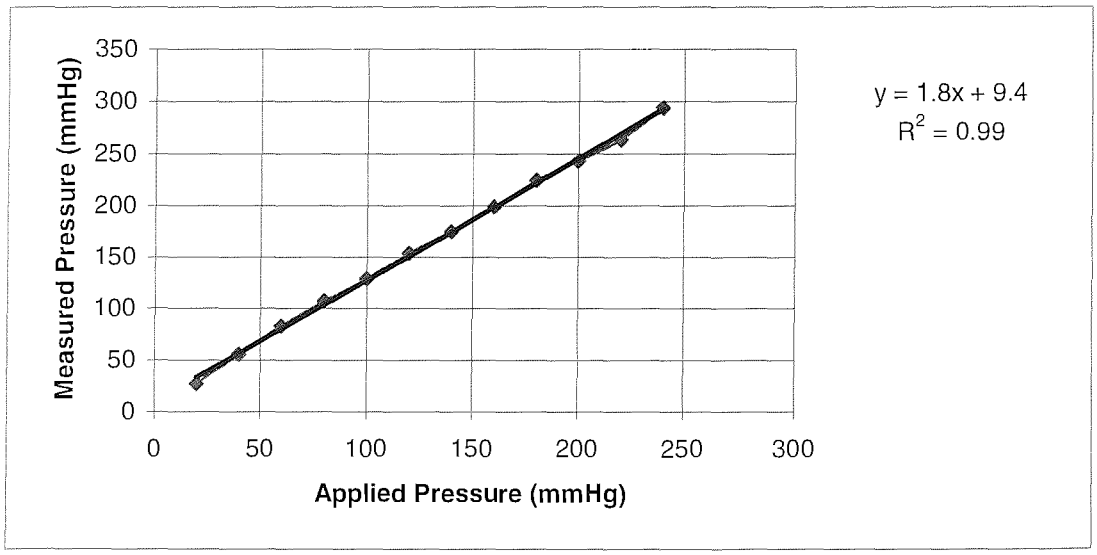



**Figure 3.4 FSA Mat Readings Taken Using Calibration Rig - Decreasing Pressure**

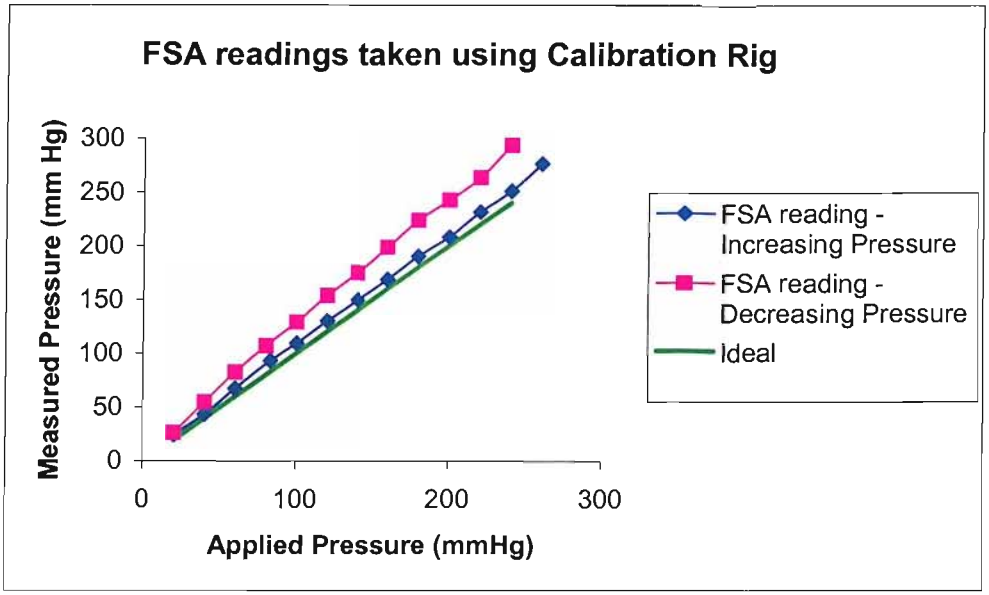



**Figure 3.5 Comparison of FSA Readings - Increasing and Decreasing Pressure**

For the purposes of this study rather than increasing and decreasing the level of pressure being applied, a constant interface pressure is applied as a result of the volunteer lying supine. Therefore it is expected that the influence of hysteresis would be minimised.

ii) Sensor Response Time - The second factor is sensor sample rate which was set at 0.3 frames per second and once the required pressure was reached readings were taken for 30 seconds. As only 92% of the maximum output of the sensors is achieved in 0.1 seconds, 94% in 15 seconds and 99% in 3 minutes this again may account for some of the inaccuracy identified and therefore measurements using the FSA mat should be taken after a minimum of 3 minutes in order to ensure the sensors had achieved maximum output. Tests were repeated and measurements taken after 3 minutes. The level of accuracy remained the same.

### 3.1.5 Testing for Creep

To enable the analysis of results to be adjusted for creep, the degree of creep in the FSA mat had to be identified before experimentation.

In order to test the FSA mat for creep it was placed in the calibration rig and 100mmHg applied to the mat for a period of 1 hour. Readings were taken every 15 seconds. As the maximum output of the sensors is not achieved for an initial period the results were only compared to a known pressure following a 5 minute stabilisation period.

Figure 3.6 illustrates that there is a steady increase in pressure readings, the reading plateaus at 112mmHg after approximately 32minutes. Therefore the percentage error for the equilibrated reading is 12%.

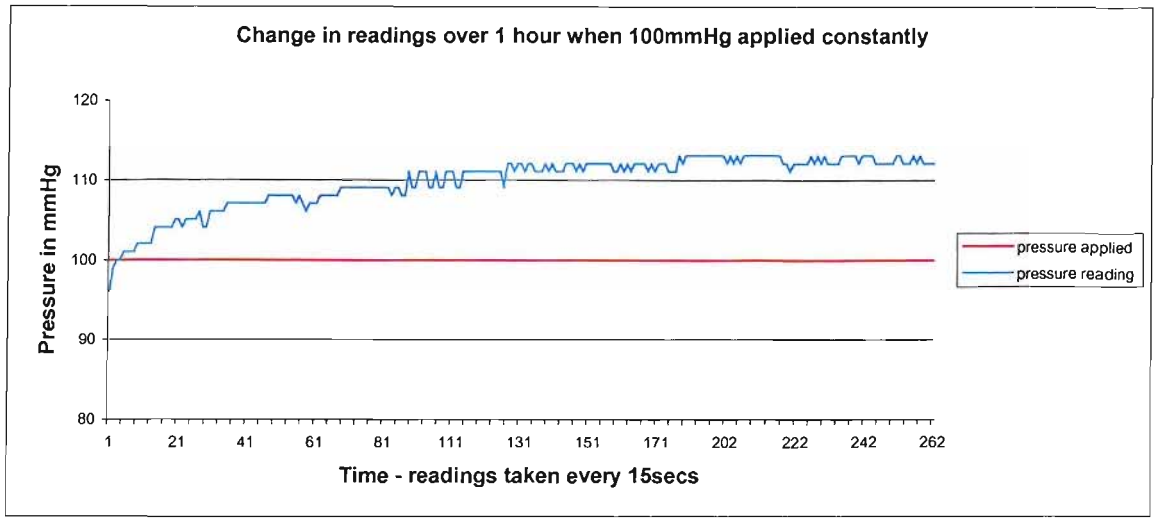



**Figure 3.6 Graph showing the extent of creep of FSA mat when100mmHg was applied for a period of 1 hour - readings taken every 15seconds**

From this experiment the degree of creep appears to be a maximum of 12mmHg over a period of one hour, but remains within 10mmHg within the first 20minutes. As the FSA mat will be used for a maximum of 10 minutes for the main study a 10% error should be accounted for. However, as the margin of error for accuracy is 12% for interface pressures between 40 to 100mmHg the greater margin of error will be taken into account for the methodology of the main study.

### 3.1.6 Identification of Peak Pressure on Sacrum

The Force Sensing Array (FSA) is a satisfactory method of measuring pressure, and the method for identifying the point of peak pressure on the sacrum of the subject can be tested. Further assessment was undertaken to demonstrate the accuracy and repeatability for the technology and methodology to be used in the main study.

The location of the point of peak pressure on the sacrum is required so that the transducers used to measure the partial pressure of oxygen and carbon dioxide can be applied to the point of peak pressure corresponding to the subject's own body weight. This is then the point to which the peak pressure identified is reapplied, and the effects of the pressure on tissue perfusion monitored.

### 3.1.6.1 Method

The design of the volunteer study was subject to peer review and ethic committee approval. Experiments were undertaken in a side room and measures taken to ensure that there were no interruptions to maintain the volunteer's privacy and dignity at all times. The temperature of the room was monitored and remained constant at 20°C. Volunteers were recruited by distribution of the study details via email and posters throughout Southampton General Hospital. The study was explained verbally to all volunteers by the researcher and an information sheet given explaining the procedure (see Appendix 1). Any questions were answered and if they were satisfied a consent form signed. The volunteers were informed that they could withdraw from the study at any time. A total of ten volunteers were used for this part of the study.

The calibrated Force Sensing Array (FSA) was positioned over the middle third of a standard hospital bedframe and pentaflex mattress. The same pentaflex mattress was used on all occasions. The volunteer then lay supine on the mattress, with their sacral area positioned on the FSA. The readings identified by the FSA are viewed in real time enabling the researcher to locate the point of maximum pressure. This was achieved by rolling the volunteer sufficiently to place an object on the point of highest pressure. The volunteer was then rolled back with the object still in position to check that the correct position has been identified. As the FSA consists of 1024 sensors which are all identified, and coded on the visual display it is possible to confirm that the correct position has been identified. Once the correct position has been confirmed the point is marked on the sacrum with a marker pen and the object removed. This was repeated three times to confirm the repeatability of the technique.

### 3.1.6.2 Results

The first solid marker produced to identify and mark the highest point of pressure on the volunteer was a circle of plastic, the diameter of which matched the size of the partial pressure of oxygen and carbon dioxide transducer, and the depth was 1cm. However, the mattress deformed sufficiently in response to the solid marker such that the interface pressure was not increased sufficiently to be identified by the FSA mat. To overcome this problem a squash ball was used, which was identified by the mat, as seen in Figure 3.7, 3.8, 3.9 & 3.10. Figure 3.7 & 3.9 indicated that the point of highest pressure was on the right side of the volunteer, and once the marker had been positioned it was visible on points N-K16. Figures 3.8 & 3.10 demonstrate that the technique is repeatable.

In Figure 3.8 the areas of lower pressure in the cells immediately surrounding the four cells of highest pressure marked in red (N to K16) is the result of reduced contact with the pressure sensing mat as the ball is compressed by the volunteer's body weight.

#### 3.1.6.3 Conclusion

The method was found to be accurate, repeatable and accepted by the subjects who volunteered.

**Figure 3.7 Readings of the FSA mat showing point of highest pressure**

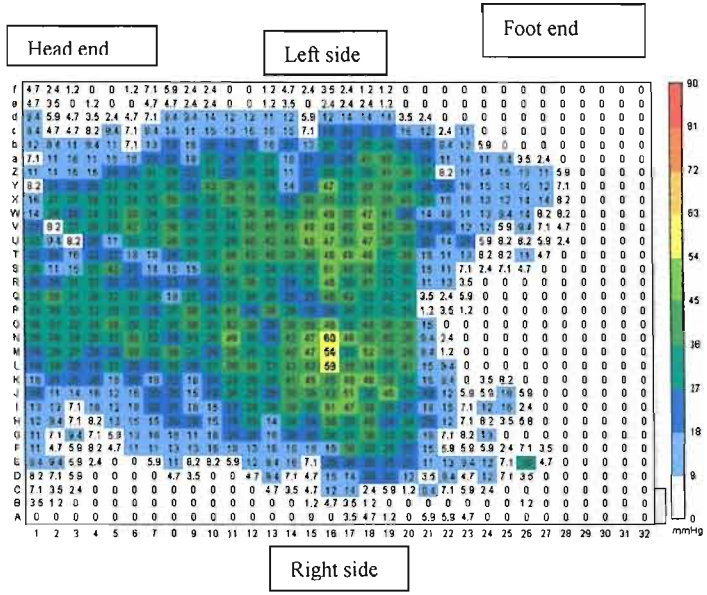



**Figure 3.8 Readings of FSA mat with marker in situ**

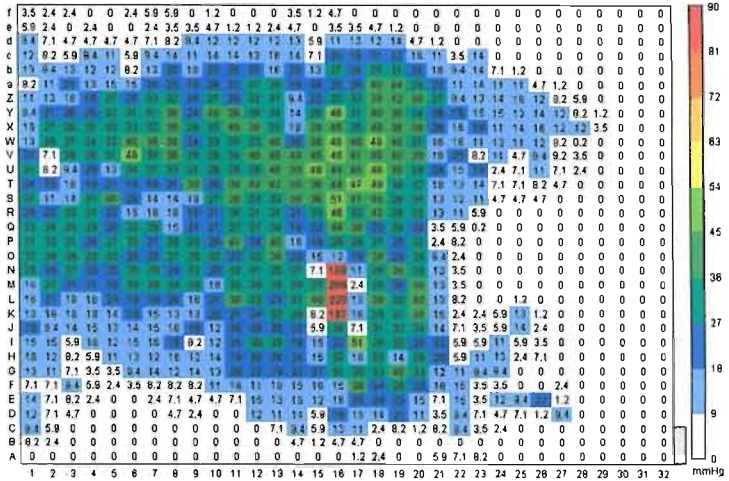



**Figure 3.9 Readings of the FSA mat showing point of highest pressure**

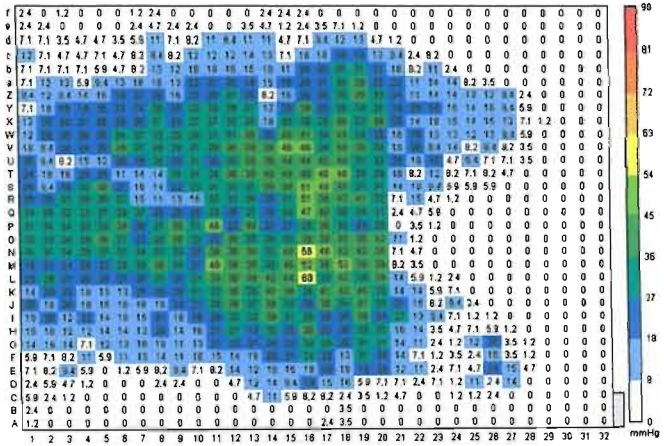
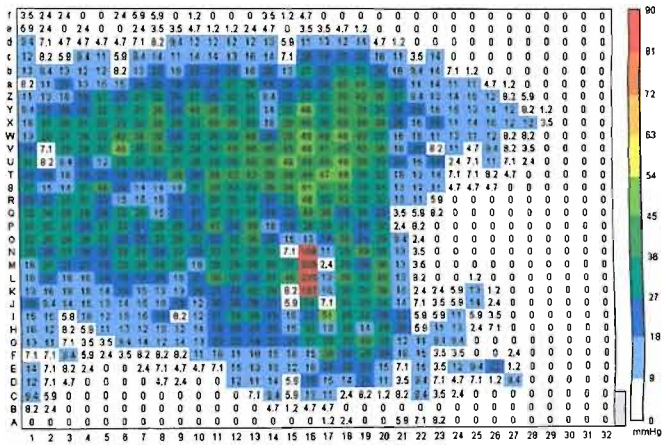



**Figure 3.10 Readings of FSA mat with marker in situ**



## 3.2 Application of Peak Pressure

As described in Chapter 2 previous experimental studies to assess tissue perfusion have required the subject to lie in a prone position whilst pressure was applied. As the majority of patients are unable to lie prone due to their medical condition(s) it was an objective of this study to design and develop an experimental approach that would be readily tolerated by volunteers and, importantly, was transferable to the clinical environment for patient assessments. Therefore a new methodology was required, enabling pressure to be applied to the sacral area when the volunteer was lying in a lateral position.

This section describes the methodology and a series of tests undertaken to identify the accuracy and repeatability of the method. Development of a robust method was considered essential in order that any changes to individuals' oxygen and carbon dioxide perfusion within sacral tissue, in response to the pressure being applied, can be studied and compared with confidence. It was also important to assess the acceptability of the methodology by the volunteers and its transferability to clinical areas.

### 3.2.1 Method

The method of applying the peak pressure identified to the sacral area consists of applying a flexible bladder, which is used within the cuff of a sphygmomanometer's cuff, over the two transcutaneous oxygen and carbon dioxide monitoring electrodes positioned on the two sacral points of highest pressure. A padded belt is then positioned around the individual, to hold the flexible bladder in position. In the same manner as normal blood pressure measurement, one of the tubes from the flexible cuff is attached to a sphygmomanometer and the other to a small hand pump. The cuff is then inflated and the pressure measured using the sphygmomanometer. The flexibility of the bladder enables it to envelope the electrodes, therefore applying an evenly distributed force sacrally (see Figures 3.11a - 3.11d).

In order to test the accuracy and repeatability of the methodology used to reapply the interface pressures identified, a Kikuhime resonant pressure sensor, (Advancis Medical), was used. Unlike the FSA mat the Kikuhime consists of a small hand held system which uses a maximum of three individual sensors. The sensors consist of two resonators located on the surface of a diaphragm, and the applied pressure is measured from the difference of two the resonant frequencies. In order to stabilise the resonators and protect them from surrounding fluid they are encapsulated in a micro-vacuum cavity. The accuracy is ± 1mmHg. (Harada et al, 1999). The size of each individual sensor is 1cm x 1cm wide and 0.5mm thick. The sensors were located under the transcutaeous oxygen

and carbon dioxide electrodes, located at the two sacral points of highest pressure. The flexible bladder was then applied over the electrodes and held in position with the padded belt. The bladder was then attached to a calibrated sphygmomanometer and 10 to 100mmHg was applied in increments of 5 mmHg. By comparing the pressure measured by the Kikuhime pressure sensor with the pressure applied, the accuracy of the technique could be identified. This test was repeated 4 times further to check repeatability.

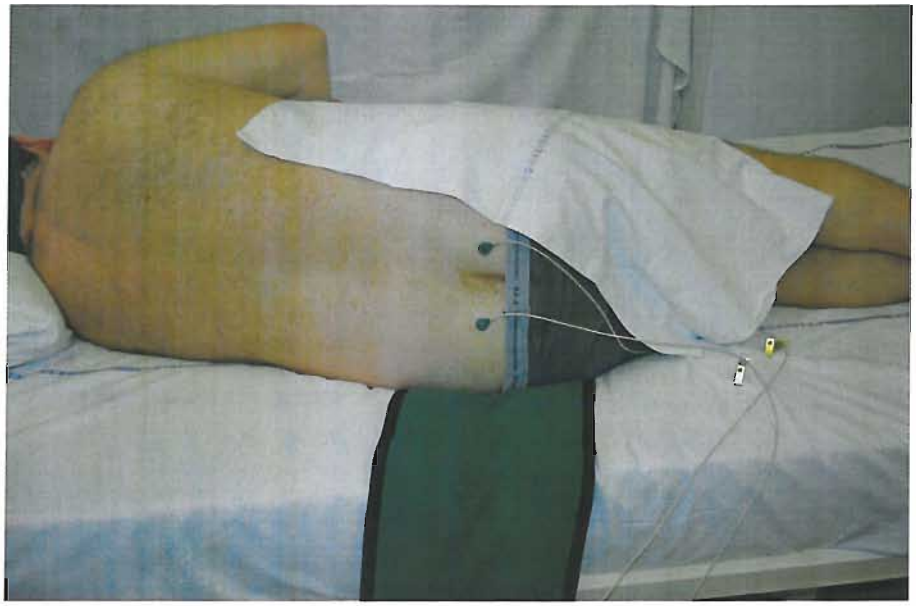


**Figure 3.11a Transcutaneous electrodes in situ on peak pressure sacral points**

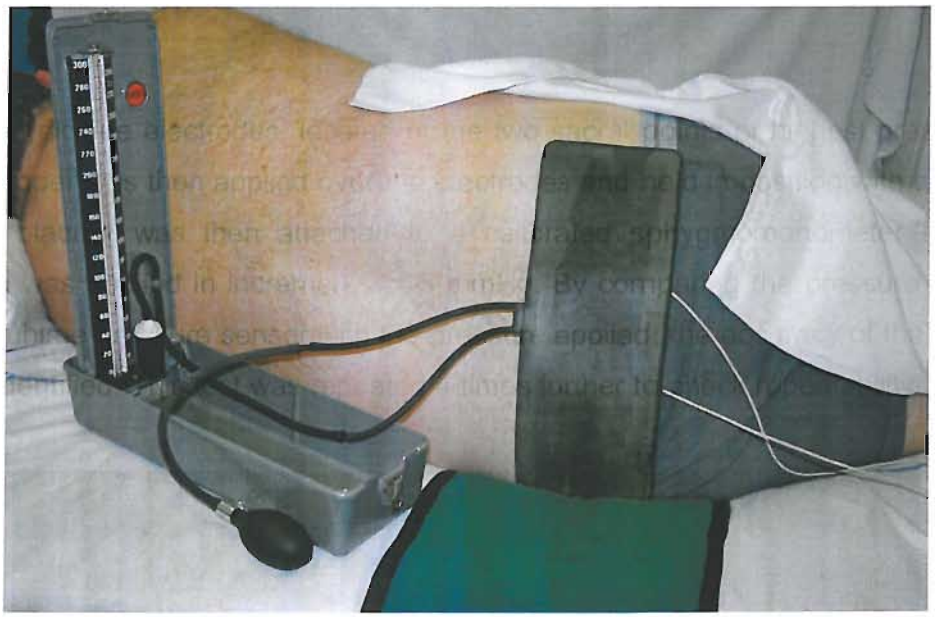


**Figure 3.11b Flexible bladder positioned over electrodes with calibrated sphygmomanometer attached**

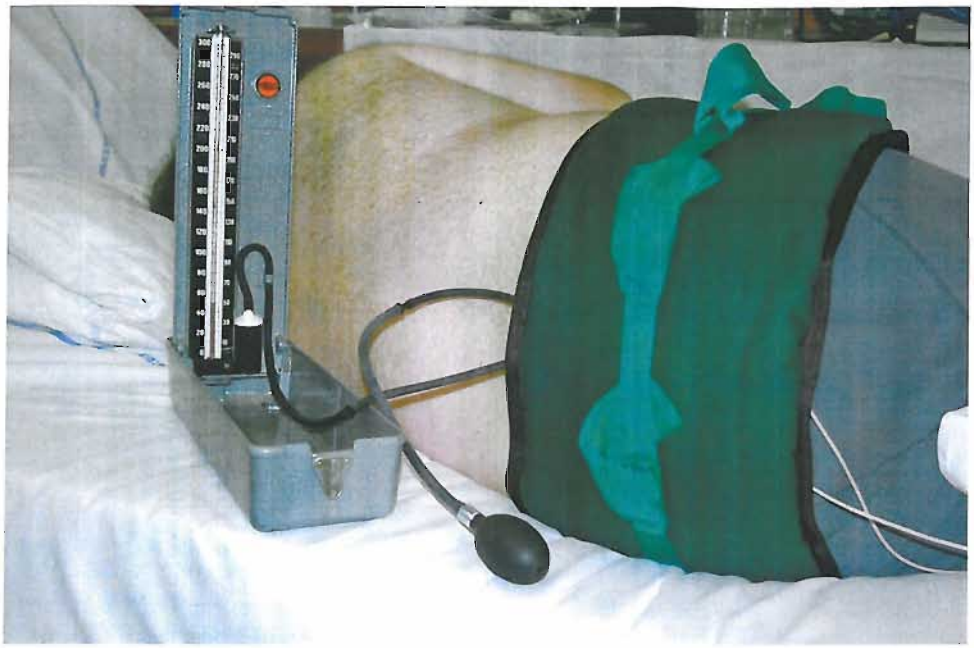


**Figure 3.11c Flexible bladder and padded belt in situ with calibrated sphygmomanometer attached**

**Figure 3.11d Flexible bladder and padded belt in situ showing bladder inflated and electrodes concealed**

### 3.2.2 Results

The results (Table 3.3) show that the percentage error of the method for applying pressure to the sacral tissue ranged from –1.2 to +4%, with a maximum standard deviation from each mean of 1.1mmHg. Through the method of least squares the line of best fit was found and is shown in Figure 3.12. From this it can be calculated that the measured pressure (mmHg) equals 0.99 of the applied pressure plus 0.07 (y=0.99 + 0.07). The equation indicates an acceptable association between the pressure applied and pressure measured.

The coefficient of variation ranged from 3.5 to 5.7% below 30mmHg and from 0.5 to 2.2% for pressures from 30mmHg to 100mmHg.

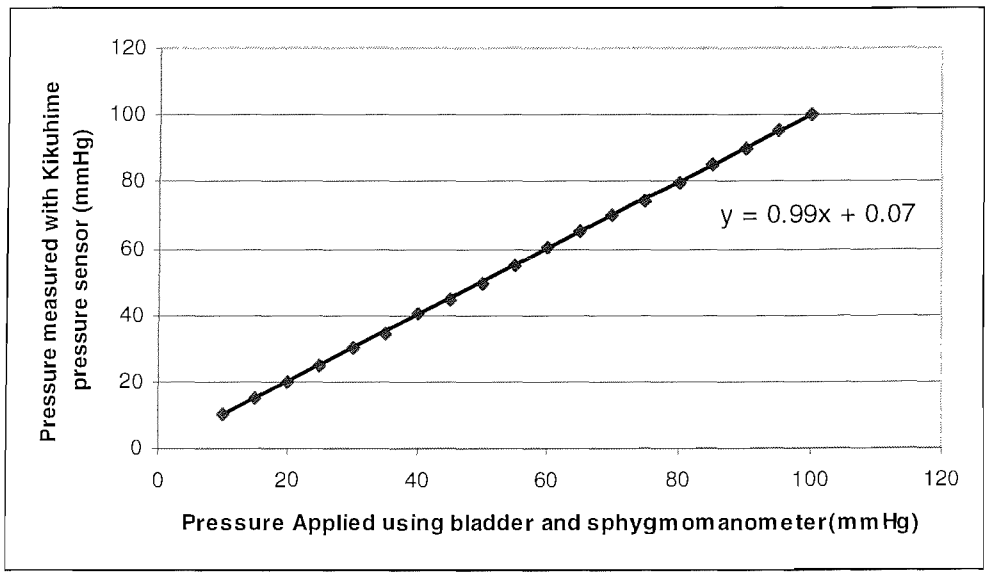



**Figure 3.12 Pressure applied using bladder and sphygmomanometer compared with the pressure measured by the Kikuhime pressure sensor.**

### 3.2.3 Conclusion

The agreement between the pressure applied and pressure measured was very strong with an $R^2$ value of 0.99. For the range of pressures anticipated to arise as a consequence of individuals' own body mass (30 to 80mmHg) the range of 0.7 to 2.2% variation for the repeated measurements of pressures applied was considered an acceptable for this study.

Table 3.3 Comparison of Pressure Applied Bladder and Sphygmomanometer with Pressure Recorded using Kikuhime Pressure Sensor

| Pressure applied with bladder and sphygmomanometer (mmHg) | Pressure (mmHg) recorded using the Kikuhime pressure sensor | | | |
|---|---|---|---|---|
| | Mean | Standard Deviation | % Error | Coefficient of variation (%) |
| 10 | 10.4 | 0.5 | 4.0 | 5.3 |
| 15 | 14.8 | 0.8 | -1.3 | 5.7 |
| 20 | 20 | 0.7 | 0.0 | 3.5 |
| 25 | 25 | 1.0 | 0.0 | 4.0 |
| 30 | 30.4 | 0.5 | 1.3 | 1.8 |
| 35 | 34.6 | 0.5 | -1.1 | 1.6 |
| 40 | 40.4 | 0.9 | 1.0 | 2.2 |
| 45 | 44.8 | 0.8 | -0.4 | 1.9 |
| 50 | 49.4 | 0.5 | -1.2 | 1.1 |
| 55 | 55 | 1.0 | 0.0 | 1.8 |
| 60 | 60.6 | 0.5 | 1.0 | 0.9 |
| 65 | 65 | 1.0 | 0.0 | 1.5 |
| 70 | 70 | 1.0 | 0.0 | 1.4 |
| 75 | 74.4 | 0.5 | -0.8 | 0.7 |
| 80 | 79.6 | 0.5 | -0.5 | 0.7 |
| 85 | 85.2 | 1.1 | 0.2 | 1.3 |
| 90 | 89.8 | 0.4 | -0.2 | 0.5 |
| 95 | 95 | 1.0 | 0.0 | 1.1 |
| 100 | 100.2 | 0.8 | 0.2 | 0.8 |

## 3.3 Accuracy of Electrodes Used to Measure Transcutaneous Partial Pressure of Oxygen ($tcPO_2$) and Transcutaneous Partial Pressure of Carbon Dioxide ($tcPCO_2$)

Tension is the driving force that drives oxygen and carbon dioxide from one region to another, and gases always move from a region of high tension to low. The transcutaneous measurement of oxygen and carbon dioxide is not just an indicator of the tension of

oxygen and carbon dioxide transported by the local circulatory system, but also the ability of gases to move between cells and tissues.

For cells to survive it is not just important that there is adequate blood flow, or that the quality of the blood being carried by the circulatory system is sufficient to sustain cell metabolism, but that the oxygen and carbon dioxide can diffuse effectively to and from the local tissue cells respectively. The technique used to measure transcutaneous partial pressure of oxygen ($tcPO_2$) and transcutaneous partial pressure of carbon dioxide ($tcPCO_2$) has been validated through previous studies (see Chapter 2, Section 2.1) and therefore the aim of this experiment is to identify the accuracy of each of the four electrodes to be used in the study.

### 3.3.1 Method

The Perimed Periflux 5000 system was used because four Radiometer $tcPO_2$ and $tcPCO_2$ electrodes can be used simultaneously. This enables measurements of a control site and two or three sacral sites to be measured simultaneously. For this study only three electrodes were required. Therefore the accuracy will be defined for the three electrodes and corresponding ports used for this study. The electrodes are calibrated prior to every reading, in accordance with manufacturers guidance. In order to test the accuracy and repeatability of the electrodes they were exposed to a known mix and concentration of gases. The gas used for calibration is a mix of 5% carbon dioxide and 21% oxygen and the same gas mix was used to check the accuracy of the electrodes following calibration. The gas was simultaneously passed across the face of the electrodes using the same method as that used when calibrating the electrodes. This was repeated four times to check repeatability of the results. The time taken for the electrode to respond to a change in carbon dioxide levels was also observed by exposing the electrodes to air at the end of the test.

### 3.3.2 Results

The same three electrodes and ports of the Perimed Periflux 5000 system were used throughout the research and the results are illustrated in Figures 3.13.

The accuracy of the electrodes in relation to oxygen is relatively good with the lowest reading being 21.2 kPa and highest being 22.7kPa (see Table 3.4). The average mean for electrode 1 was 21.9kPa, electrode 3 was 22.5kPa and electrode 4 was 21.4kPa. The percentage error ranged from +1% to +8%. The accuracy of the electrodes for carbon dioxide measurements ranged from 4.4kPa to 5.3 kPa and the average mean for each

electrode was as follows: electrode 1 was 5kPa, electrode 3 was 4.9kPa and electrode 4 was 5.3. The percentage error ranged from -12% to +6%. The mean response time for each electrode to respond to a change in carbon dioxide from 5 down to 0mmHg was 20 seconds. No check of linearity was undertaken as it was assumed.

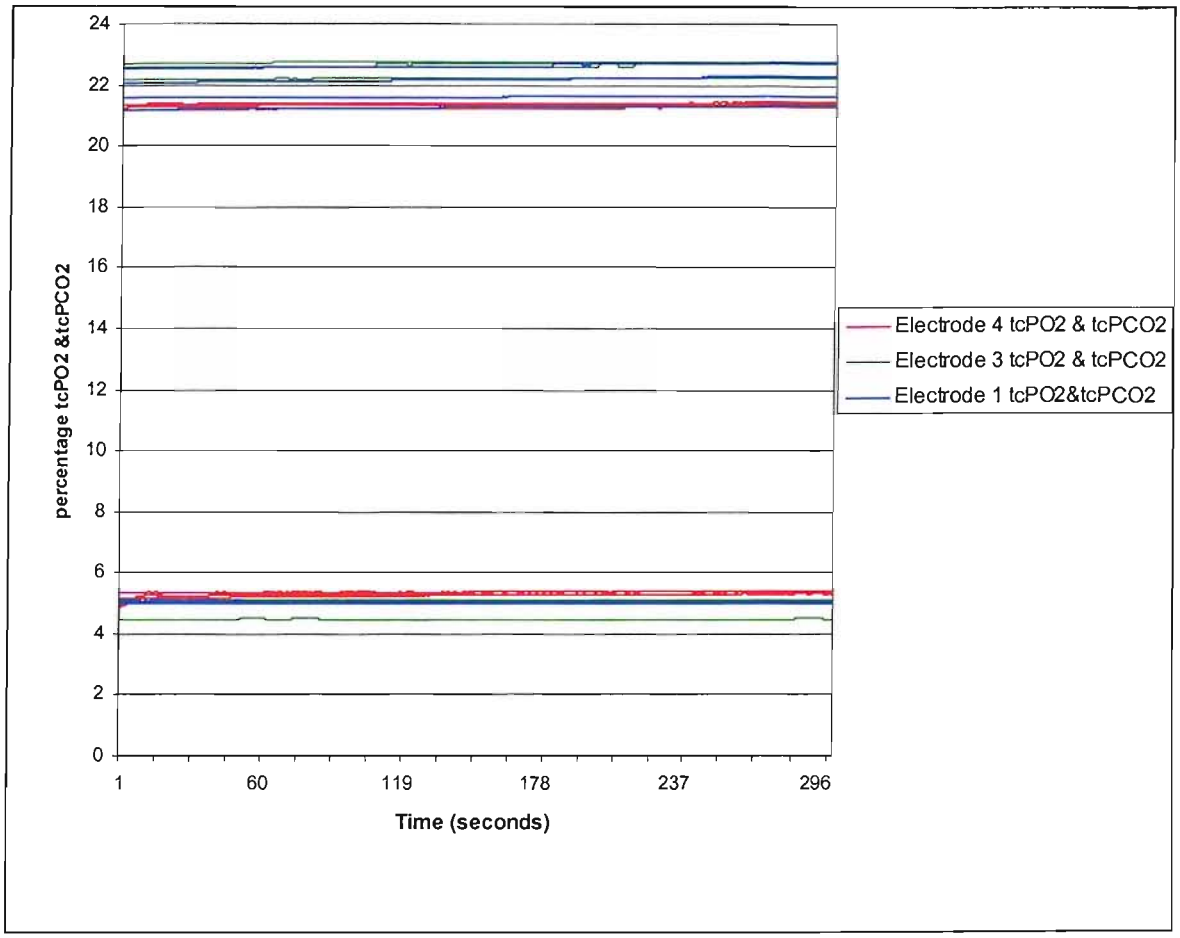



**Figure 3.13 Illustration of the accuracy of the three electrodes when simultaneously exposed to 21 % oxygen and 5 % carbon dioxide**

### 3.3.3 Conclusions

The results identify the differences between the electrodes with electrode 4 showing the greatest degree of accuracy for oxygen and electrode 1 showing the greatest degree of accuracy for carbon dioxide. To ensure consistency throughout the research the same electrodes and ports on the Perimed Periflux 5000 system were used for the same anatomical sites throughout the each study.

Although the range of percentage error for the carbon dioxide fell outside of 10% it was representative of an error of -0.6kPa and + 0.3kPa which was considered acceptable for

this study. The rate of which electrodes respond to a change in level of oxygen or carbon dioxide must be taken into consideration when examining the results of the study.

**Table 3.4 Summary of Results from the 4 Accuracy Tests for the Three Electrodes**

| | Partial Pressure Oxygen (21% control) | | | | Partial Pressure Carbon Dioxide (5% control) | | | |
|---|---|---|---|---|---|---|---|---|
| | Test | Mean | Max | Min | Test | Mean | Max | Min |
| Electrode 1 | 1 | 22.2 | 22.3 | 22.0 | 1 | 5.1 | 5.1 | 5.0 |
| | 2 | 22.6 | 22.7 | 22.5 | 2 | 5.0 | 5.0 | 4.9 |
| | 3 | 21.3 | 21.4 | 21.2 | 3 | 5.0 | 5.1 | 5.0 |
| | 4 | 21.6 | 21.6 | 21.5 | 4 | 4.9 | 4.9 | 4.9 |
| Electrode 3 | 1 | 22.6 | 22.7 | 22.6 | 1 | 5.1 | 5.1 | 4.9 |
| | 2 | 22.6 | 22.6 | 22.5 | 2 | 5.1 | 5.1 | 5.0 |
| | 3 | 22.7 | 22.7 | 22.6 | 3 | 5.0 | 5.0 | 5.0 |
| | 4 | 22.2 | 22.2 | 22.1 | 4 | 4.5 | 4.5 | 4.4 |
| Electrode 4 | 1 | 21.3 | 21.3 | 21.2 | 1 | 5.2 | 5.3 | 5.1 |
| | 2 | 21.4 | 21.4 | 21.4 | 2 | 5.4 | 5.4 | 5.4 |
| | 3 | 21.4 | 21.5 | 21.5 | 3 | 5.3 | 5.3 | 5.0 |
| | 4 | 21.4 | 21.4 | 21.2 | 4 | 5.2 | 5.3 | 4.9 |

# Chapter 4 Examination of the Relationship Between Sacral Tissue Perfusion and Pressure Applied as a Consequence of Healthy Individuals' Own Body Mass

The presence of pressure, primarily occurring at the interface between the soft tissues and a support surface, for example a mattress, is considered the initiating factor in the development of pressure ulcers (Barbenel, 1991). Individual characteristics, such as body mass and muscle tone effect the distribution of the pressure exerted as a consequence of an individual's own body mass, and is expected to vary between individuals.

Previous studies have applied a range of nominal pressures that were considered to be representative of the physiological range for interface pressures (Bader, 1990a; Knight et al, 2001). However, the following experiment was designed to assess the level of tolerance of individuals' sacral tissue to pressure, exerted through their own body mass, by examining the relationship between changes in transcutaneous partial pressure of oxygen ($tcPO_2$) and carbon dioxide ($tcPCO_2$) levels in response to the pressure applied. Hence, the objectives of this experiment were to determine the normal range of pressures exerted on sacral tissue by body mass, and the changes in $tcPO_2$ and $tcPCO_2$ that this pressure generates.

An individual's pressure resulting from their own body mass was identified using the Force Sensing Array (FSA) pressure sensing mat. The methodology for this measurement is described in Section 3.1. The cutaneous perfusion response was measured by monitoring changes in transcutaneous levels of partial pressure of oxygen and carbon dioxide. The methodology for measurement is described in Sections 3.3 and 3.4.

## 4.1 Method

In order to provide an acceptable spread of age, sex and body mass index an experimental population of forty volunteers was considered to be sufficient for the pilot study. The volunteers were healthy, defined as not under the care of a physician, no known cardiovascular or respiratory disease, normo-tensive, non-smokers and not taking any medication. The purpose of the study was explained orally to each volunteer and an information sheet provided explaining the procedure. Any questions were answered and volunteers were requested to sign a consent form if they were happy to proceed. The volunteers were informed that following consent they could withdraw from the study at any time. The Local Research Ethics Committee approved the study (LREC No. 026/04/t).

For each of the volunteers, the following demographic details were taken: blood pressure, age, sex, height and mass to calculate body mass index, past medical history, time of day, and point in menstrual cycle for female volunteers. Each volunteer was required to refrain from exercising excessively for 2 hours prior to their experimental study session, and from consuming caffeine-containing drinks and alcohol for 8 hours before the study.

Experiments were undertaken in a treatment room, with a constant ambient temperature of 20°C.

The calibrated FSA pressure sensing mat was positioned over the middle third of a hospital bed with a standard hospital mattress (Pentaflex, Huntleigh Healthcare). The volunteer was asked to lie supine on the mat, with one pillow, no head-rest elevation and their sacral area positioned on the pressure sensing mat. The point of peak pressure exerted over the sacrum through the individuals own body mass was identified as described in Section 3.2. The peak and the second highest points of pressure were marked using a non-permanent marker pen.

Having identified the highest points of pressure on both left and right sacral sites, the pressure sensing mat was removed and a padded belt, approximately 30cm wide, positioned under the volunteer with the volunteer lying supine, in a left lateral position (90° rotation), that is on their left hand side. It was assumed that as a consequence of the pressure sensing exercise taking approximately 10 to 15 minutes, the volunteer had adequately rested and their body temperature acclimatised to the room temperature.

Three calibrated electrodes[1] for transcutaneous oxygen and carbon dioxide were then applied to the volunteer. These are the same electrodes as described in Section 3.4. The first, control, electrode was positioned in the left sub-clavicular area, this location being readily accepted as a suitable control site when undertaking vascular studies (Hauser & Shoemaker, 1983). The control electrode would permit naturally occurring physiological changes to be identified and taken into consideration when examining the response of sacral tissue to pressure. The second and third electrodes were located on the two sacral points identified as having the highest interface pressure as a consequence of the individual's own body mass. The same electrode was always used for the control site, and left and right sacral sites. This was to ensure that the margin of error associated with each electrode was constant for each site and did not then result in erroneous responses or changes observed.

[1] Radiometer electrodes model number: E5280-tc$PO_2$ / tc$PCO_2$

To ensure maximum vasodilatation of cutaneous tissue under the electrode the temperature of the electrode was set to 43°C. Once in position, manufacturer's instructions for the electrodes stipulate that a 10 minute rest period was required to ensure physiological stabilisation. A further 10 minutes enabled the baseline readings of transcutaneous oxygen and carbon dioxide to be determined.

The peak sacral pressure identified using the pressure sensing mat was then applied to the sacral electrodes by positioning an inflatable bladder over the sacral electrodes, and held in position by the padded belt (see Figure 3.9). The bladder, attached to a calibrated sphygmomanometer, was inflated to achieve the peak sacral pressure. The subsequent response of the sacral tissue to the application of pressure was measured using $tcPO_2$ and $tcPCO_2$ electrodes and recorded using a Periflux 5000 system, attached to a laptop computer. Data is collected until either the electrode readings are considered to have stabilised or the volunteer indicates that they are unable to tolerate the pressure.

Once the readings had stabilised, to remove the pressure instantaneously the padded belt was released and the bladder removed. The $tcPO_2$ and $tcPCO_2$ levels were monitored and recorded until they returned to levels measured prior to the application of pressure, or to a point where they appear to have stabilised. Repeating experiments for six of the volunteers assessed repeatability and variability of results.

The data collected were analysed to investigate the following:

i) identification of the range of body mass index and interface pressure and whether there is any relationship between these two variables;

ii) identification of the range, mean and standard deviation of baseline $tcPO_2$ and $tcPCO_2$ readings for the control site and the two sacral sites and investigation of any differences between these groups. This was important because if a significant difference existed between the control site and the two sacral sites the measurements would need to be treated individually. Significantly this would be different from the conventional experimental approach to date which take an average of the two sites;

iii) the range of $tcPO_2$ and $tcPCO_2$ levels in response to pressure exerted through individuals' own body mass; the significance of any difference between the left and right sacral site; the relationship identified between the changes in $tcPO_2$ and $tcPCO_2$ responses: the significance of differences observed when compared to baseline levels;

iv) the patterns of the response of $tcPO_2$ and $tcPCO_2$ to pressure and identify any delineable trends;

v) the relationship between interface pressure and response of sacral tissue to pressure.

## 4.2 Results

### 4.2.1 Demographic Details

Forty healthy volunteers were recruited, twenty three male and seventeen female, with a mean age of forty one and mean systolic blood pressure of 113mmHg and diastolic of 70mmHg. A complete summary of the demographic details for the volunteers can be found in Table 4.1

**Table 4.1 Summary of the Volunteers' Demographic Details**

| | Age (Years) | Sex | Systolic Blood Pressure (mmHg) | Diastolic Blood Pressure (mmHg) | Peak pressure exerted through own body mass measured by FSA (mmHg) | Body Mass Index ($Kg/m^2$) |
|---|---|---|---|---|---|---|
| Range | 25 - 60 | 17 Female 23 Male | 100 -140 | 50 - 85 | 38 - 74 | 16.3 - 40.9 |
| Mean | 41 | | 113 | 68 | 58 | 27.1 |
| SD | 7.5 | | 9.62 | 9.3 | 7.5 | 6.4 |

### 4.2.2 Relationship Between Interface Pressure and Body Mass Index (BMI)

For the study population the interface pressures ranged from 38-74mmHg. Current assessment tools used to assess risk of patients developing pressure ulcers specifically identify both under mass and over mass individuals as being at greater risk of tissue damage due to pressure. Clinically the body mass index is commonly used to identify whether patients are under mass, normal, over mass or obese. However, when the body mass indexes for the study population are plotted against the corresponding interface pressure, as illustrated in Figure 4.1, no clear relationship is apparent. Using Spearman's rank correlation, the correlation between body mass index and interface pressure is 0.37, indicating a poor correlation, significant at the p=0.05 level (1-tailed). A perfect correlation has a value of +1 or -1 and no correlation gives a value of 0. Any value below 0.5 is considered a weak correlation. Therefore these findings supports the non-experimental

views of others (Swain & Bader, 2002) that body mass index is insufficiently sensitive to indicate the likely interface pressure that individuals' are exposed to as a consequence of their own body mass.

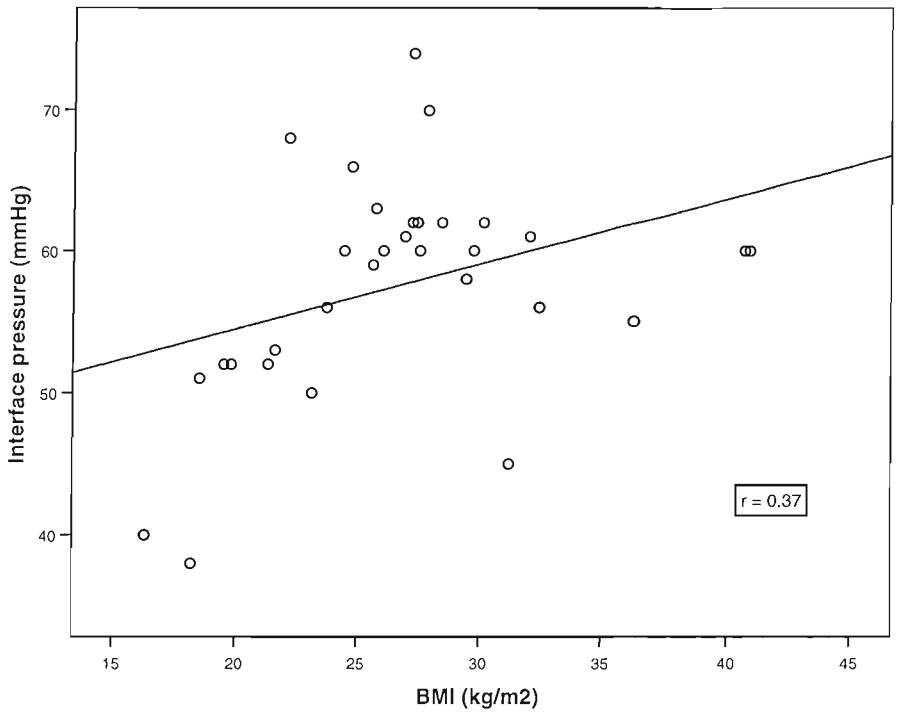



**Figure 4.1 Scatter Diagram of the Body Mass Index and Interface Pressure**

### 4.2.3 Normal Baseline Values of $tcPO_2$ and $tcPCO_2$ for the Control Site and the Two Sacral Sites Investigated

The baseline measurements of $tcPO_2$ and $tcPCO_2$ for the control site, left sacral site and right sacral site were identified for the study population by calculating the mean reading from the ten minute period of measurements prior to the application of pressure. The levels ranged from 3 to 14.6kPa for $tcPO_2$ and from 3.6 to 6.9kPa for $tcPCO_2$. Table 4.2 and 4.3 summarise the baseline mean, median, standard deviation, skewness, minimum and maximum level of $tcPO_2$ and $tcPCO_2$ respectively for the three sites.

**Table 4.2 Summary of $tcPO_2$ Levels Without Pressure**

| | Control site $tcPO_2$ (kPa) (n=40) | Right Sacral site $tcPO_2$ (kPa) (n=40) | Left Sacral site $tcPO_2$ (kPa) (n=40) |
|---|---|---|---|
| Mean | 8.7 | 9.3 | 9.2 |
| Median | 8.6 | 9.5 | 9.4 |
| Standard Deviation | 2.4 | 2 | 2 |
| Skewness | 0.01 | -0.4 | -0.7 |
| Minimum | 3 | 4 | 3.7 |
| Maximum | 14.6 | 12.6 | 6.9 |

**Table 4.3 Summary of $tcPCO_2$ Levels Without Pressure**

| | Control site $tcPCO_2$ (kPa) (n=40) | Right Sacral site $tcPCO_2$ (kPa) (n=40) | Left Sacral site $tcPCO_2$ (kPa) (n=40) |
|---|---|---|---|
| Mean | 5.2 | 5.3 | 5.3 |
| Median | 5.3 | 5.5 | 5.5 |
| Standard Deviation | 0.64 | 0.7 | -0.02 |
| Skewness | -0.49 | -0.7 | 0.1 |
| Minimum | 3.6 | 3.7 | 3.8 |
| Maximum | 6.4 | 6.4 | 6.9 |

Figure 4.2 illustrates the range of baseline readings for $tcPO_2$ and $tcPCO_2$ levels for all three sites indicating that the greatest range for $tcPO_2$ was observed at the control site, with a maximum level of 14.6kPa and minimum level of 3kPa. The greatest range for $tcPCO_2$ was observed at the left sacral site with a range of 3.6 to 6.9kPa.

The distribution of data for the two sacral sites and control site are slightly skewed for the baseline $tcPO_2$ and $tcPCO_2$ results. Therefore, non-parametric tests were applied to examine the degree of differences existing between the sites. Using the Wilcoxan signed rank test no significant difference was identified between the three sites. The p-values ranged from 0.1 to 0.9, and the results were only considered significant if the p-value is $\leq$ 0.05.

The range of baseline readings for both $tcPO_2$ and $tcPCO_2$ support the findings of previous studies (Seiler & Stahelin (1979), Coleman et al (1986), Takiwaki et al (1991) and Rodrigues et al (2001).

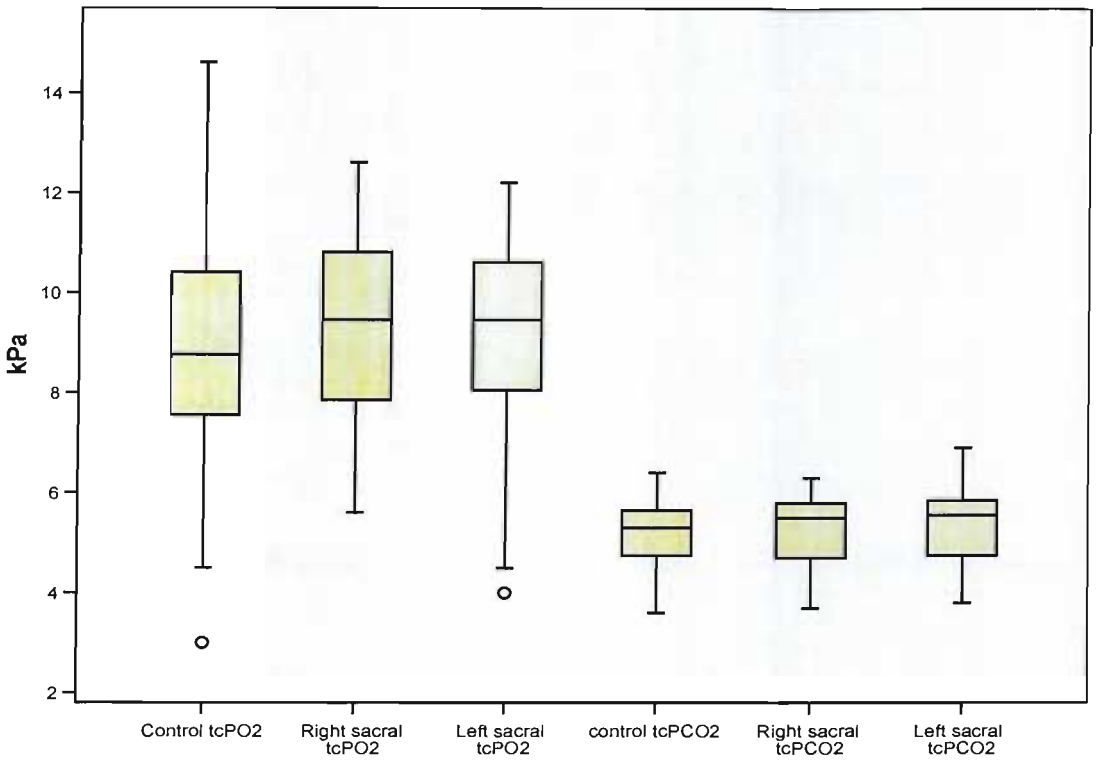



**Figure 4.2 Box and whisker plot of baseline levels of $tcPO_2$ and $tcPCO_2$. The upper and lower bars represent the upper and lower quartile respectively. The line across the centre box is the median.**

The reproducibility of the results was tested for the left and right sacral site by repeating the $tcPO_2$ and $tcPCO_2$ measurements for six of the original 40 volunteers. The second set of measurements for both sacral sites was made on a separate day, under the same conditions as described in section 4.1. The mean and the coefficient of variation was calculated. This was done by calculating the standard deviation (SD) of the differences between each measurement, and its corresponding mean, and by identifying the mean ($x$) of the mean for the six volunteers.

The equation for calculating the coefficient of variation is:

$$\text{coefficient of variation} = \frac{SD}{x}100$$

The coefficient of variation for baseline levels of $tcPO_2$ and $tcPCO_2$ for sacral tissue was identified as 8.7% and 6.1% respectively. This falls within the findings of Coleman et al (1986), who identified a coefficient of variation of 10% for $tcPO_2$.

Figure 4.3 shows the differences of the $tcPO_2$ measurements from each volunteer's mean measurement, plotted against the average of the two measurements for each volunteer. This is a Bland and Altman plot and provides a good illustration of differences between the repeated measurements. The data points predominantly fall within ±2kPa along the zero line. The variation attributed to natural physiological changes. The average difference from the mean was identified to be zero with a standard deviation of 0.9kPa and the mean of the means identified to be 10.1kPa.

Figure 4.4 shows the Bland and Altman plot for the differences between of the repeated baseline sacral $tcPCO_2$ measurements. The variation around the zero line is tighter with the data points lying within ±1kPa. The average difference from the mean was identified to be zero with a standard deviation of 0.3kPa and the mean of the means identified to be 5kPa. As for $tcPO_2$ the variation is attributed to natural physiological changes.

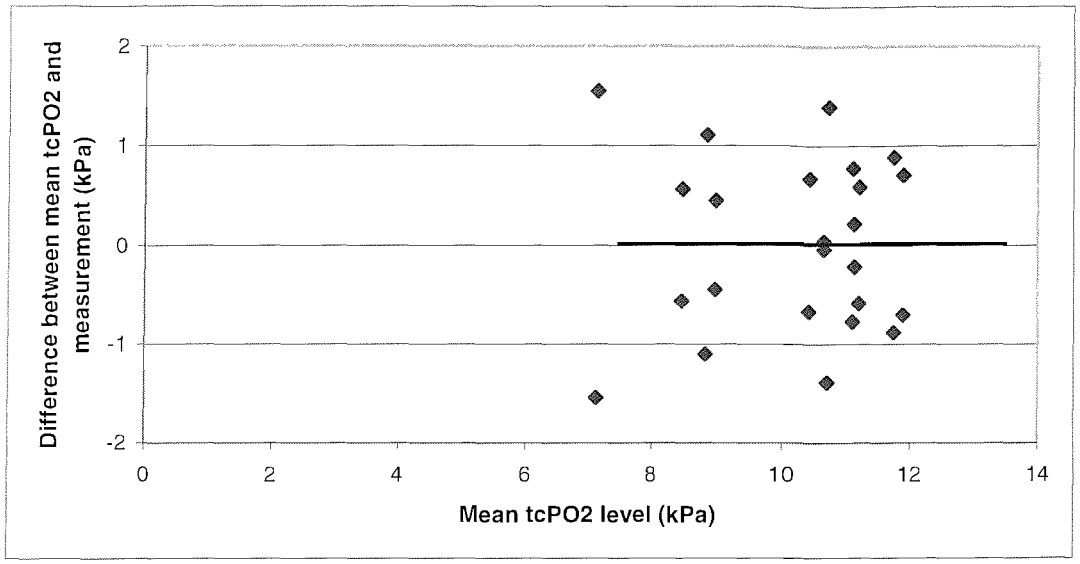



**Figure 4.3** Bland and Altman plot comparing repeated baseline $tcPO_2$ measurements of six volunteers

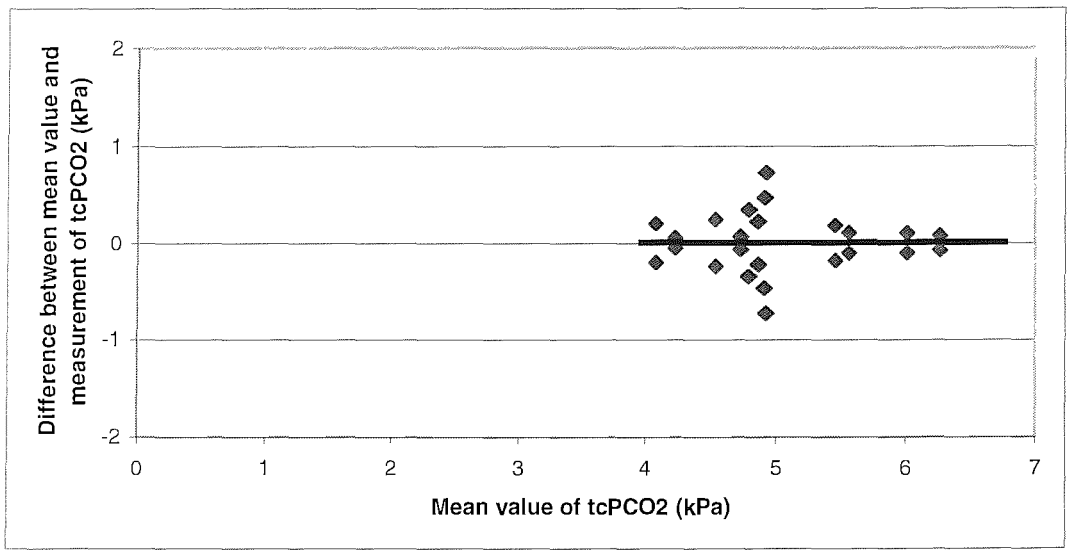



**Figure 4.4** Bland and Altman plot comparing repeated baseline $tcPCO_2$ measurements of six volunteers

### 4.2.4 Changes in Sacral Tissue $tcPO_2$ and $tcPCO_2$ Levels as a Consequence of Pressure Applied Equivalent to that Exerted Through Individuals' Own Body Mass, and When Compared with Baseline Levels

The measured peak pressure was applied to the corresponding volunteer. Following application of the pressure, the oxygen and carbon dioxide levels in the tissue were measured.

Once the peak pressure for each individual had been achieved, it was maintained at this level for a minimum of 15 minutes. During this 15-minute period both the level of $tcPO_2$ and $tcPCO_2$ was independently recorded. The initial 2 minutes under constant pressure was excluded from the results to ensure stabilisation and equalisation was achieved.

Table 4.4 provides a summary of the results and shows that tcPO2 levels declined in response to the application of pressure applied as a result of the volunteers' own body mass. The mean value was reduced to 4.1kPa (± 3.1) for the right sacral site, and to 4.5kPa (±2.9) for the left sacral site. Levels of $tcPCO_2$ increased a mean value of 7.3kPa (±2.8) for the right sacral site and 6.9kPa (±2.3) for the left sacral site.

The results are illustrated in Figure 4.5 and show a difference between the baseline readings and readings when sacral tissue is subjected to pressure. The significance of the difference between the baseline readings and measurements when pressure was applied was calculated using the Wilcoxan signed rank test, and found to be strongly significant for both $tcPO_2$ and $tcPCO_2$, with a p-value of 0.000.

**Table 4.4 Descriptive Statistics for $tcPO_2$ and $tcPCO_2$ Levels when Sacral Tissue Loaded and Unloaded**

| (n=40) | Baseline $tcPO_2$ levels for sacral sites (kPa) | | Maximum $tcPO_2$ levels for sacral sites under pressure (kPa) | | Baseline $tcPCO_2$ levels for sacral sites (kPa) | | Maximum $tcPCO_2$ levels for sacral sites under pressure (kPa) | |
|---|---|---|---|---|---|---|---|---|
| | Right | Left | Right | Left | Right | Left | Right | Left |
| **Mean** | 9.2 | 9.2 | 4.2 | 4.5 | 5.3 | 5.3 | 7.3 | 6.9 |
| **Standard deviation** | 1.95 | 2.0 | 3.1 | 2.9 | 0.7 | 0.8 | 2.8 | 2.3 |
| **Minimum** | 5.6 | 4 | 0.1 | 0.4 | 3.7 | 3.8 | 3.7 | 4.2 |
| **Maximum** | 12.6 | 12.2 | 11.1 | 9.4 | 6.4 | 6.9 | 16.1 | 15.2 |

Figure 4.5 also illustrates the difference in range of measured levels of $tcPO_2$ and $tcPCO_2$ for the left and right sacral sites when subjected to pressure.

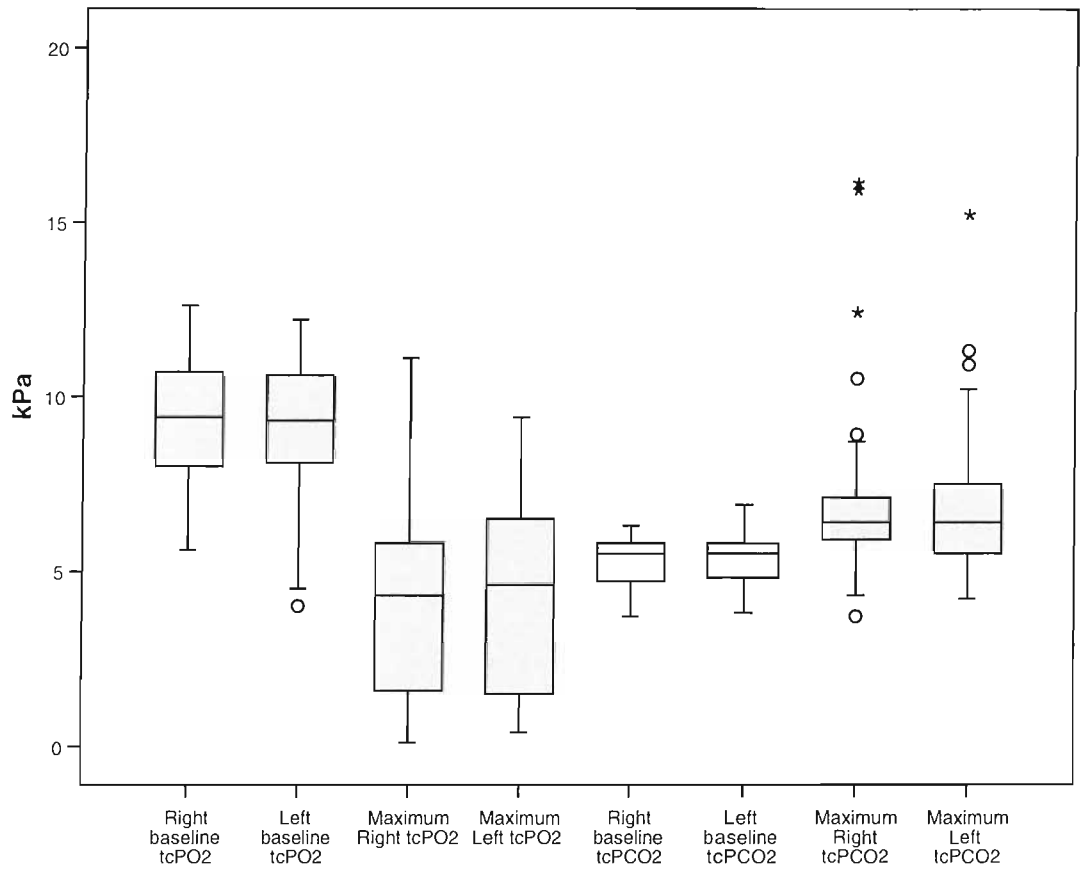



**Figure 4.5** **Box and whisker plot illustrating the differences in the range of $tcPO_2$ and tcPCO2 between baseline levels and sacral tissue subjected to pressure exerted through their own body mass.**

To examine the significance of the difference observed the correlation coefficient was calculated using the Spearmen's rank correlation, and found to be 0.39, significant at the 0.05 level (2-tailed) for $tcPO_2$ and 0.41, significant at the 0.05 level (2-tailed), for $tcPCO_2$. Figures 4.6 and 4.7 show the correlation between the responses of the left and right sacral sites and indicate a poor correlation. A value of less than 0.5 is considered a weak correlation and therefore the left and right sacral sites were analysed independently.

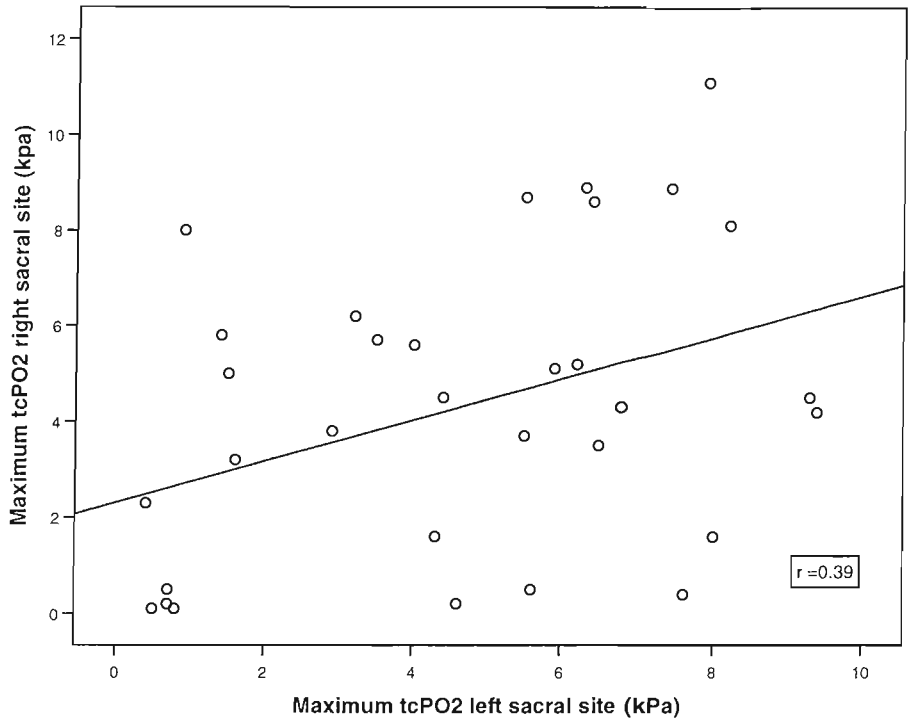



**Figure 4.6** Scatter diagram showing the correlation between the maximum levels of $tcPO_2$ achieved under pressure for the left and right sacral sites

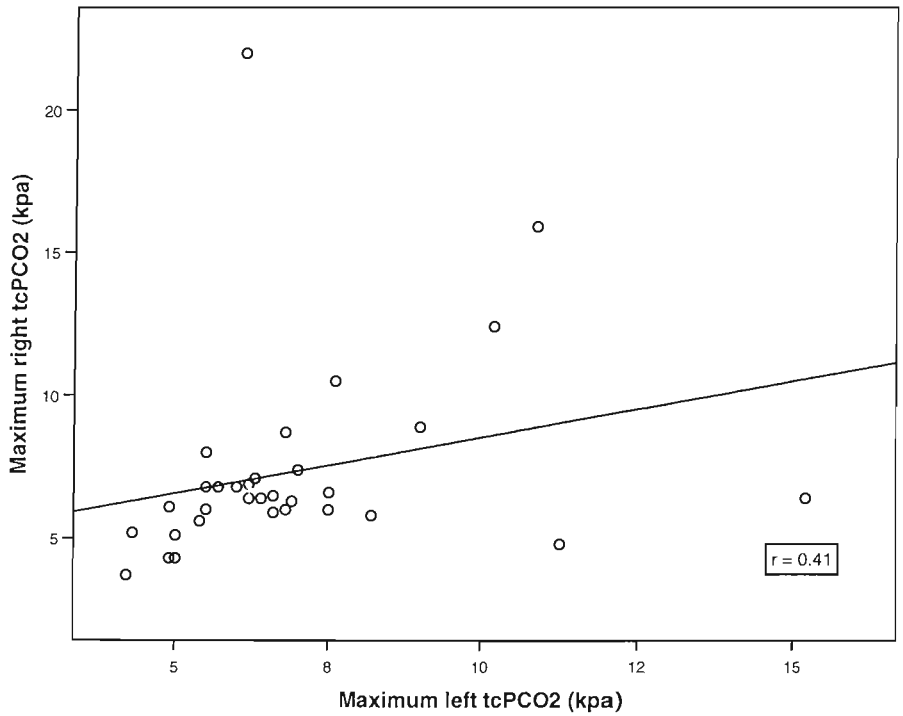



**Figure 4.7** Scatter diagram showing the correlation between the maximum levels of $tcPCO_2$ achieved under pressure for the left and right sacral sites

On examining the range of responses of sacral tissue when subjected to pressure exerted through individuals' own body mass, two distinct clusters fall outside the upper quartile range for the maximum level of $tcPCO_2$ achieved. The significance of the difference between these 3 groups was examined using the Friedman test for non-parametric data and found to be very significant with a p value of 0.002. Figure 4.8 illustrates the three groups of $tcPCO_2$ responses and this will be examined further in section 4.2.5 and 4.2.6.

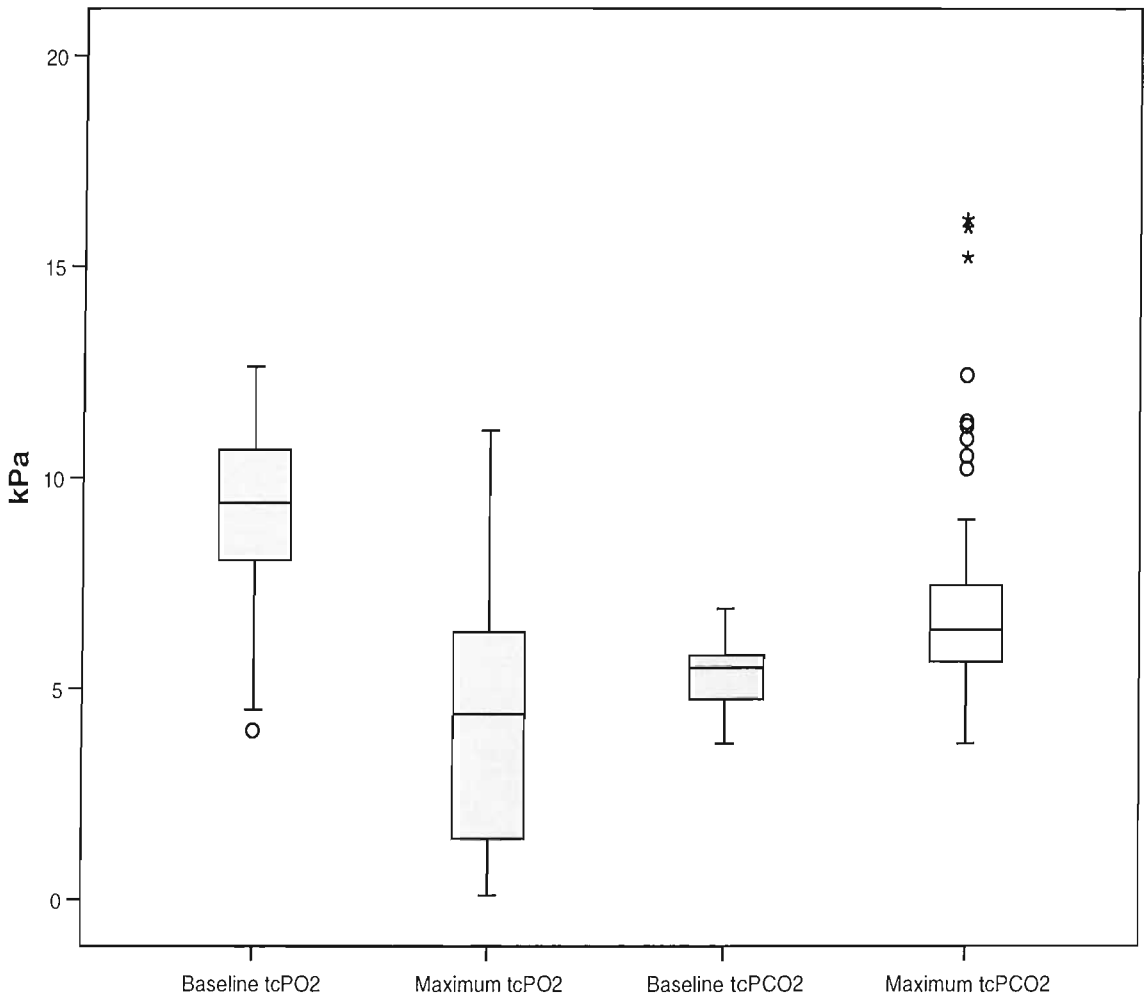



**Figure 4.8** **Box and Whisker plot illustrating the range of responses of sacral tissue to pressure exerted through individuals' own body mass. The two groups of outliers for $tcPCO_2$ are indicated by ° and ***

The degree of association between changes in levels $tcPO_2$ and $tcPCO_2$ when sacral tissue was subjected to pressure was identified using the Pearson's P correlation, and was found to have a correlation value of r = -0.53, significant at the 0.01 level (see Figure 4.8). A correlation of 0.5 and above is considered acceptable. Figure 4.9 also indicates that $tcPCO_2$ levels predominantly do not start rising above the upper limit of the normal range (7.5kPa) until $tcPO_2$ levels have fallen below 1kPa.

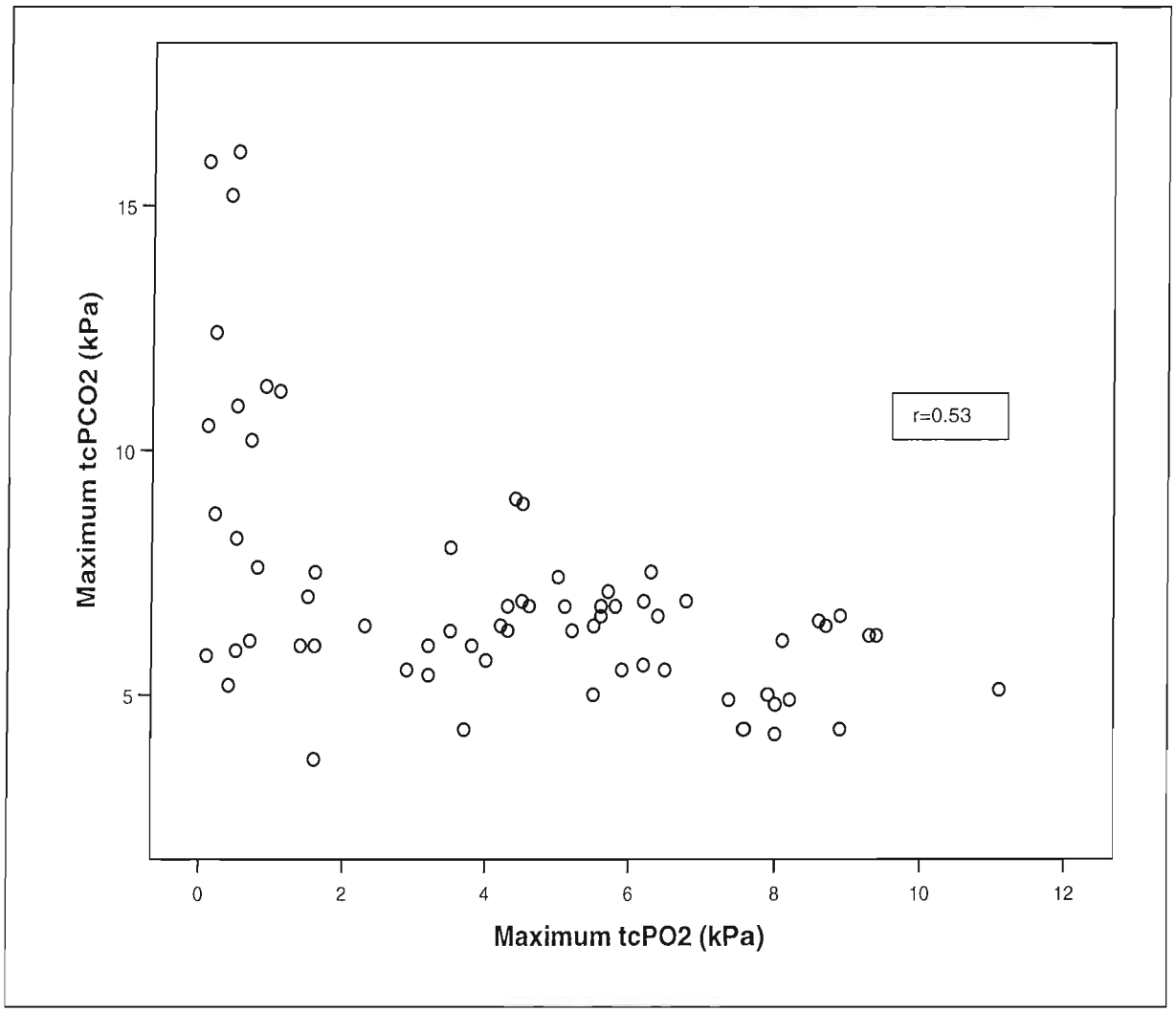



**Figure 4.9 Correlation between $tcPO_2$ and $tcPCO_2$ levels when sacral tissue is subjected to pressure exerted through their own body mass**

### 4.2.5 Physiological Trends in Measurement of Transcutaneous Partial Pressure for Oxygen and Carbon Dioxide, in Response to Externally Applied Pressure to Healthy Sacral Tissue: Patterns of Response

Section 4.2.4 identified the full range of $tcPO_2$ levels in response to the applied pressure as between 0.4 to 11.1kPa and between 3.7 to 15.2 kPa for $tcPCO_2$. Due to the large variation in the degree to which oxygen and carbon dioxide levels responded to pressure the patterns of response were examined in order further understand the nature of the responses.

The requirement to treat each sacral site as an independent site was identified in section 4.2.4 and therefore eighty responses have been analysed from the forty volunteers.

Following the application of the individual's peak sacral pressure to their sacral tissue, $tcPO_2$ levels rapidly decreased for all subjects over the initial two minutes. By visual inspection the following four responses were identified:

A. decreases to between 0 to 0.9kPa and fluctuates around that value (Figure 4.10);
B. decreases to below 1kPa, and doesn't recover above 2kPa (Figure 4.11);
C. decreases to <1kPa and shows varying degrees of recovery (Figure 4.12);
D. decreases, but not below 1kPa and recovers to varying degrees (Figure 4.13).

The corresponding response of $tcPCO_2$ to the four $tcPO_2$ responses described above are described below. The ranges for each response type were identified from Figure 4.8

A. for the first response there are three variations:
   i) minimal increase in $tcPCO_2$, but within normal range (2.7-7.4kPa) (see figure 4.14);
   ii) some elevation in $tcPCO_2$ exceeding the normal range (>7.5 - ≤12.4kPa) (see figure 4.15);
   iii) steady elevation in $tcPCO_2$ significantly exceeding normal range, (>12.4kPa) (see figure 4.16);
B. for the second response a type A (i) or Type A(ii) $tcPCO_2$ response was observed(see figure 4.14 & 4.15);
C. some elevation in $tcPCO_2$ , but within normal range (2.7-7.4kPa) (Figure 4.10);
D. None or minimal change in $tcPCO_2$ from the baseline prior to pressure being applied (Figure 4.13).

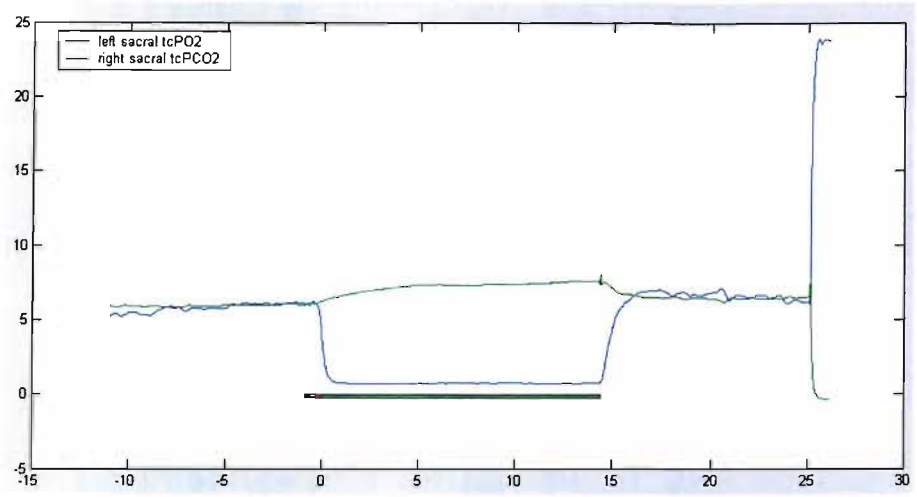



**Figure 4.10 Type A response for $tcPO_2$**

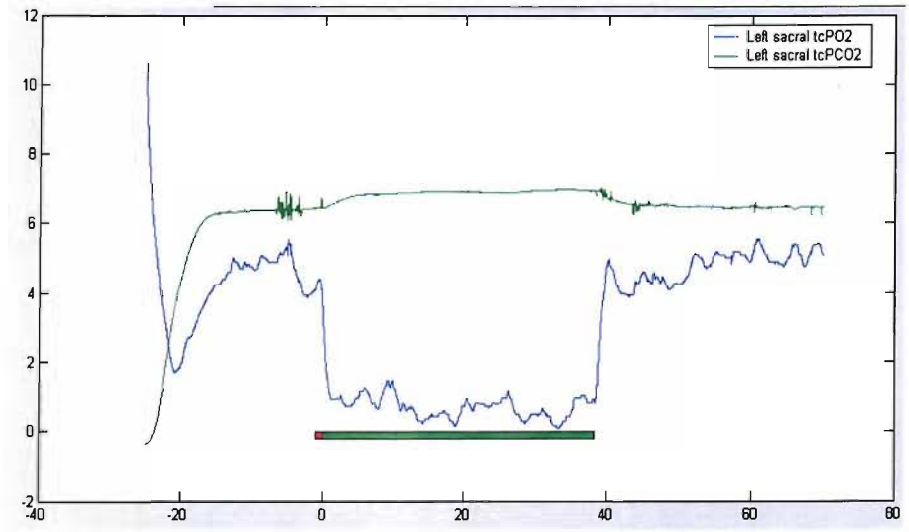



**Figure 4.11 Type B response for $tcPO_2$**

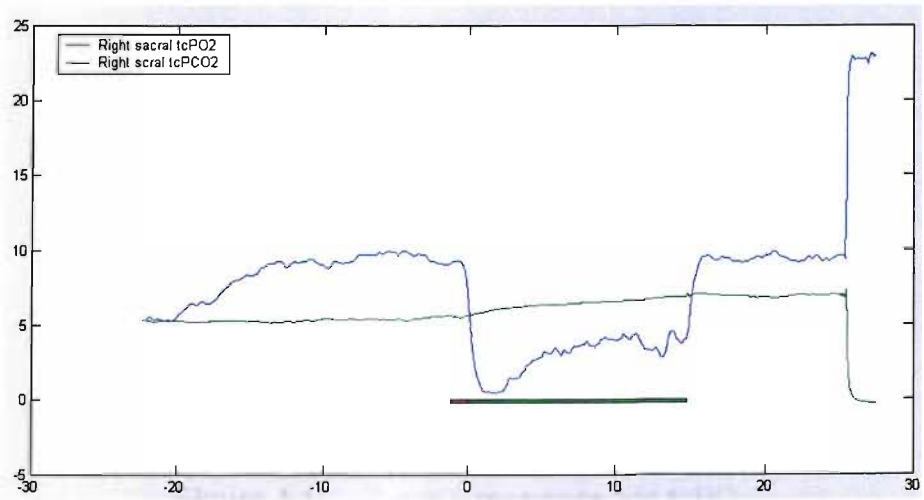



**Figure 4.12 Type C response for $tcPO_2$**

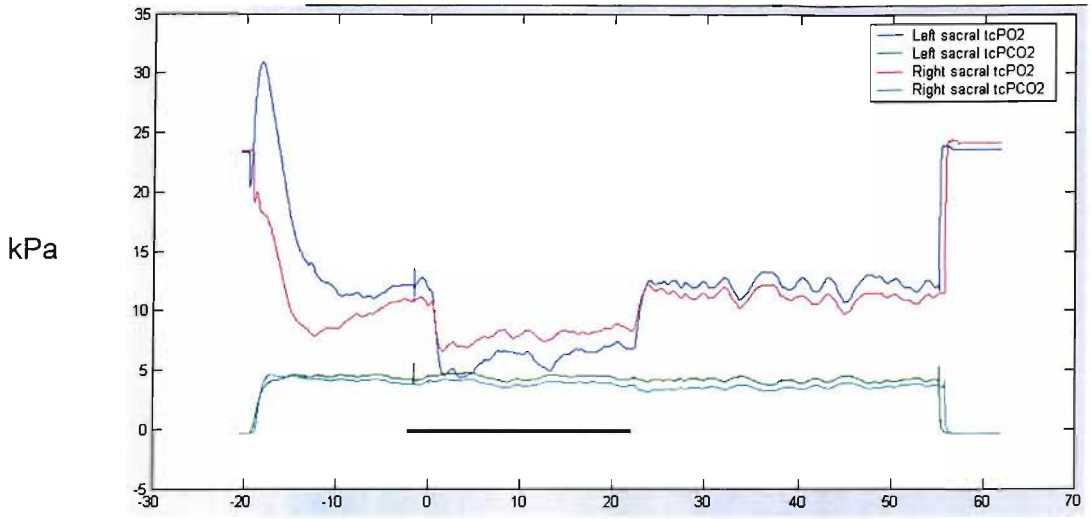



**Figure 4.13 Type D response for $tcPO_2$**

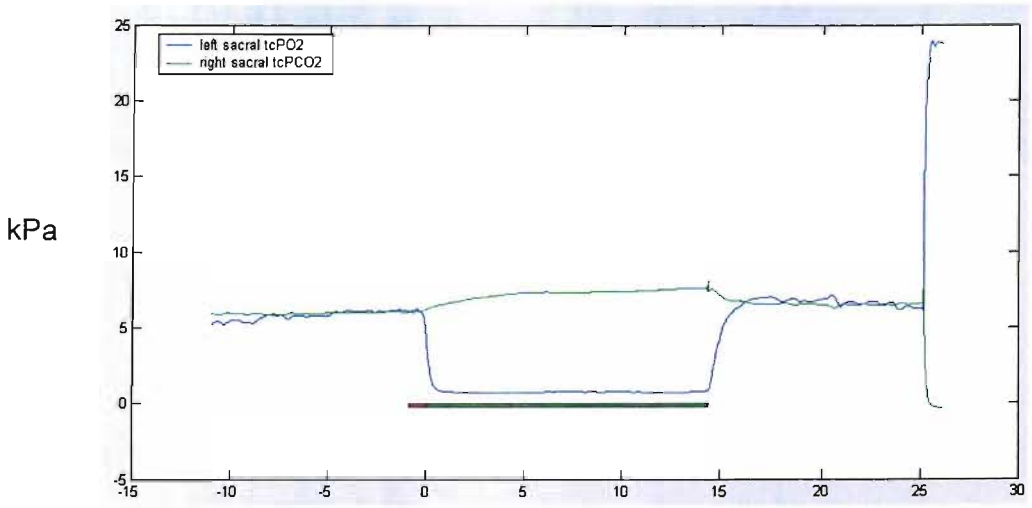



**Figure 4.14 Type A(i) response for $tcPCO_2$**

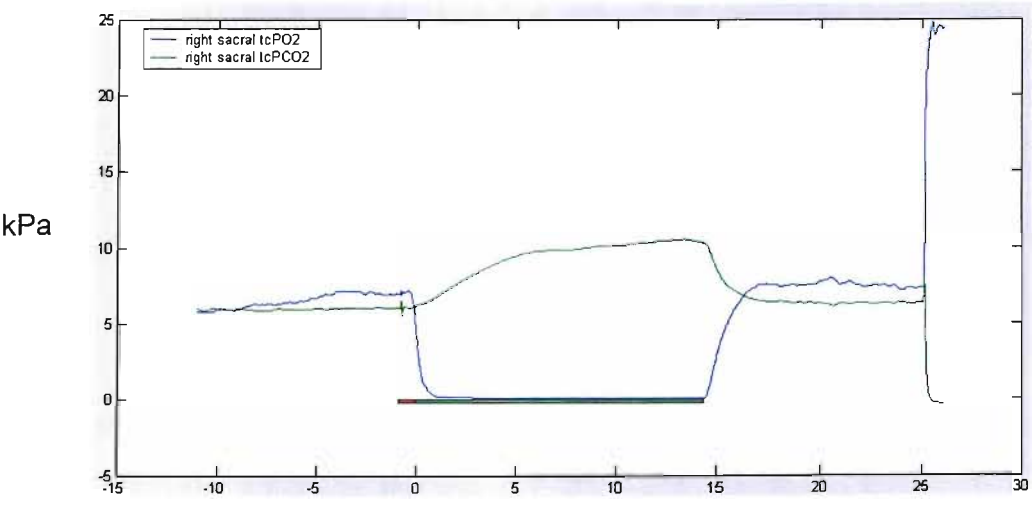



**Figure 4.15 Type A(ii) response for $tcPCO_2$**

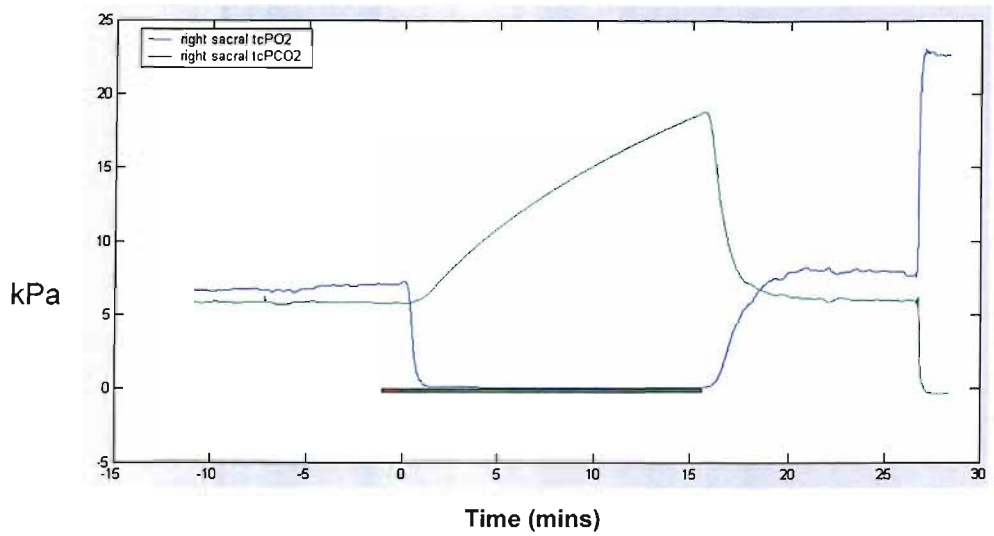



**Figure 4.16 Type A(iii) response for $tcPCO_2$**

Further analysis of the response types identified was undertaken to assess whether the categories had statistical significance. The nature of response type A(iii) for $tcPCO_2$ was very distinctive, and will be analysed further in section 4.3.2. The values of $tcPO_2$, defining the upper and lower threshold for each response type was based on the distribution of maximum $tcPO_2$ and $tcPCO_2$ levels achieved for each response type as illustrated in Table 4.5 and 4.6, and Figure 4.17 and 4.18, where very distinct breaks between the data sets can be observed.

**Table 4.5 Frequency Statistics for Maximum Level of $tcPO_2$ Achieved for each Response Type Identified**

| | | Type_D | Type_C | Type_B | Type_Ai | Type_Aii | Type_Aiii |
|---|---|---|---|---|---|---|---|
| Median | | 5.8 | 4.1 | 1.6 | .5 | .5 | .4 |
| Minimum | | 1.4 | 2.3 | 1.5 | .1 | .1 | .1 |
| Maximum | | 11.1 | 7.9 | 1.6 | .8 | 1.1 | .5 |
| Percentiles | 25 | 4.4 | 3.5 | 1.5 | .3 | .2 | .1 |
| | 50 | 5.8 | 4.1 | 1.6 | .5 | .5 | .4 |
| | 75 | 8.1 | 5.7 | 1.6 | .8 | .9 | .5 |

**Table 4.6 Frequency Statistics for Maximum Level of $tcPCO_2$ Achieved for each Response Type Identified**

| | | TypeD (n=43) | TypeC (n=16) | Type B (n=5) | Type_Ai (n=7) | Type_Aii (n=5) | TypeAiii (n=4) |
|---|---|---|---|---|---|---|---|
| Median | | 6.3 | 6.2 | 6.8 | 5.9 | 10.7 | 15.9 |
| Minimum | | 3.7 | 5.0 | 6.0 | 5.2 | 8.2 | 15.2 |
| Maximum | | 9.0 | 8.0 | 7.4 | 7.6 | 12.4 | 16.1 |
| Percentiles | 25 | 5.1 | 5.5 | 6.0 | 5.5 | 9.0 | 15.2 |
| | 50 | 6.3 | 6.2 | 6.5 | 5.9 | 10.7 | 15.9 |
| | 75 | 6.8 | 6.8 | 6.9 | 6.8 | 11.2 | 16.1 |

Type A responses were further subdivided for $tcPCO_2$ measurements and the corresponding values for $tcPO_2$ are presented in Table 4.5.

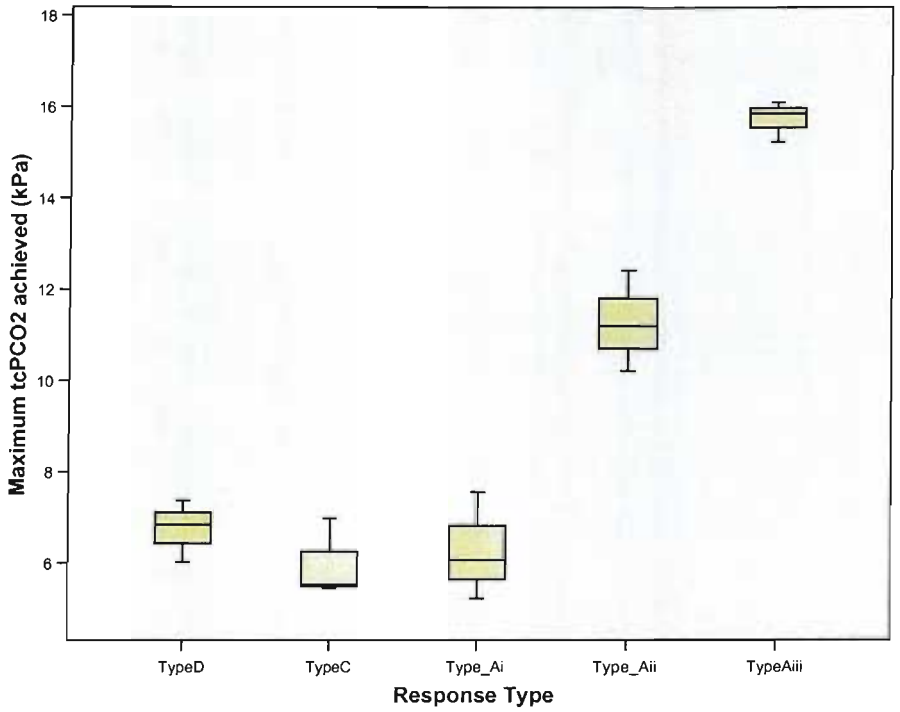



**Figure 4.17** **Box and Whisker plot illustrating the range of $tcPO_2$ levels associated with each response type**

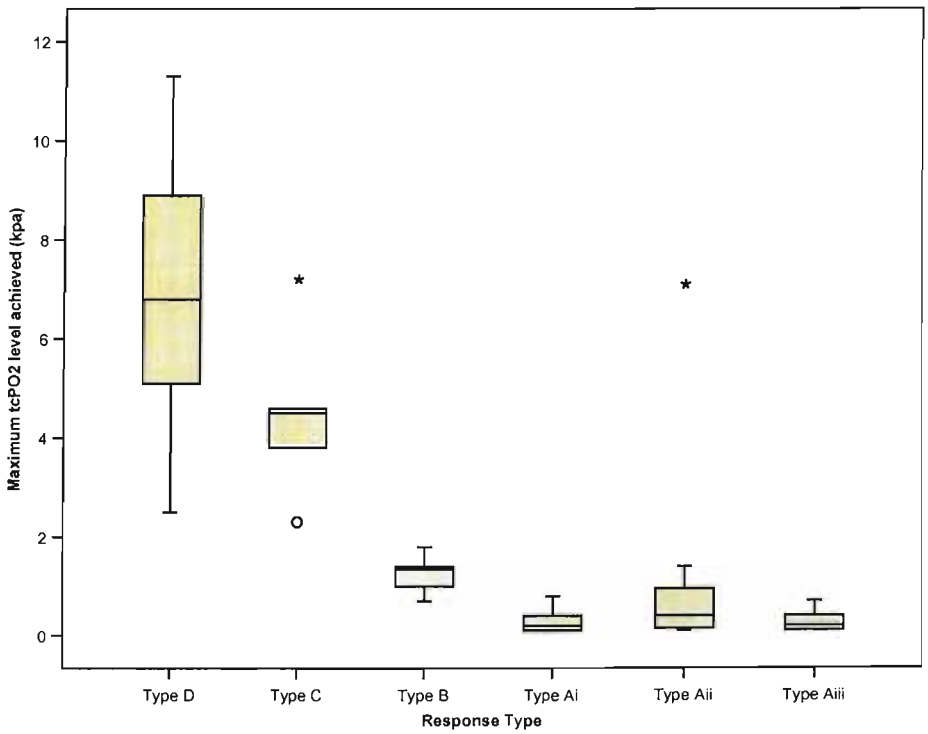



**Figure 4.18** **Box and Whisker plot illustrating the range of $tcPCO_2$ levels associated with each response type**

From figure 4.17 and 4.18, it is apparent that only after $tcPO_2$ levels fall below 1kPa that changes in response type for $tcPCO_2$ occur. The significance of the difference between the response types D, C, B & A for $tcPO_2$ and A(i),(ii) & (iii) for $tcPCO_2$ were analyzed using the Friedman test for non-parametric data. No significant difference was identified between type C & D for $tcPO_2$. Therefore these two groups were combined. The significance of the difference between response type A, B and C/D was found to be significant (p=0.05).The difference between response type A(i), (ii) and (iii) was found to be very significant, p=0.002.

The number of responses for each response type is presented in Table 4.7 and shows that 74% of individual's sacral sites appear to have a degree of tolerance to pressure exerted through their own body mass, with $tcCO_2$ levels remaining within the normal range and tcPO2 showing signs of recovery when loaded.

**Table 4.7 Frequency of Sacral Tissue Responses for 40 Volunteers**

| (n=80) | | Type of $tcPO_2$ response | | | |
|---|---|---|---|---|---|
| | | **A** (decreases to between 0 - 0.6kPa) | **B** (decrease to below 1kPa, but does not recover above 2kpa) | **C** (decreases to 0-0.8 kPa and partially recovers) | D (decreases, but not below 1kPa, followed by significant recovery) |
| **Type of $tcPCO_2$ response** | **A(i)** (minimal increase in $tcPCO_2$) | 7(9%) | 5(6%) | 16(20%) | 43(54%) |
| | **A(ii)** (some elevation in $tcPCO_2$ above normal range) | 5(6%) | | | |
| | **A(iii)** (steady elevation in $tcPCO_2$) | 4(5%) | | | |

### 4.2.6 Relationship Between the Gradient of the Slope of the Response and the Maximum Level of Oxygen and Carbon Dioxide Achieved when Sacral Tissue is Exposed to Pressure

Each response type identified in section 4.2.5 (see figures 4.10 to 4.16) presented a different slope. The slope represented the rate of change in $tcPO_2$ following the application of pressure, and the rate of increase in $tcPCO_2$ in response to pressure. The gradient of the slope of response was therefore calculated in order to examine whether there was a sufficiently significant relationship between the gradient of the slope and response type that could be used to confirm and/or predict the response type.

The gradient of the slope of the response to pressure was calculated for all subjects. This was calculated from a linear regression of all data points, starting 2 minutes after the pressure was applied at the correct level up to the time of release (see figure 4.19). The maximum level of $tcPO_2$ and $tcPCO_2$ was identified in section 4.2.5 as the main defining parameter for the response types and therefore it was the relationship between the gradient of the slope and maximum level of $tcPO_2$ and $tcPCO_2$ achieved under pressure which was explored using the Spearman's rank correlation.

No correlation was found between the gradient of the slope and maximum level of $tcPO_2$. This is because the gradient of the $tcPO_2$ slope is not sensitive to the difference in levels that $tcPO_2$ may fall to, and recover to e.g. if the $tcPO_2$ falls to and remains between 1-2 kPa of oxygen the gradient will be similar to that of tissue that shows minimal loss of oxygen in response to the pressure, which is then maintained at a relatively constant level for the duration of the period that pressure is applied.

The correlation between the maximum level of $tcPCO_2$ and gradient of the slope of the response was found to be higher level of correlation with a correlation of 0.59, significant at the 0.01 level, with $R^2 = 0.73$ (see Figure 4.20). Unlike the changes in $tcPO_2$, $tcPCO_2$ levels predominantly only increase if there is a response to the pressure being applied. Therefore the gradient of the response is not repeated through the three types of responses (Type A(i), (ii),(iii)), and could be considered to assist in defining the response type.

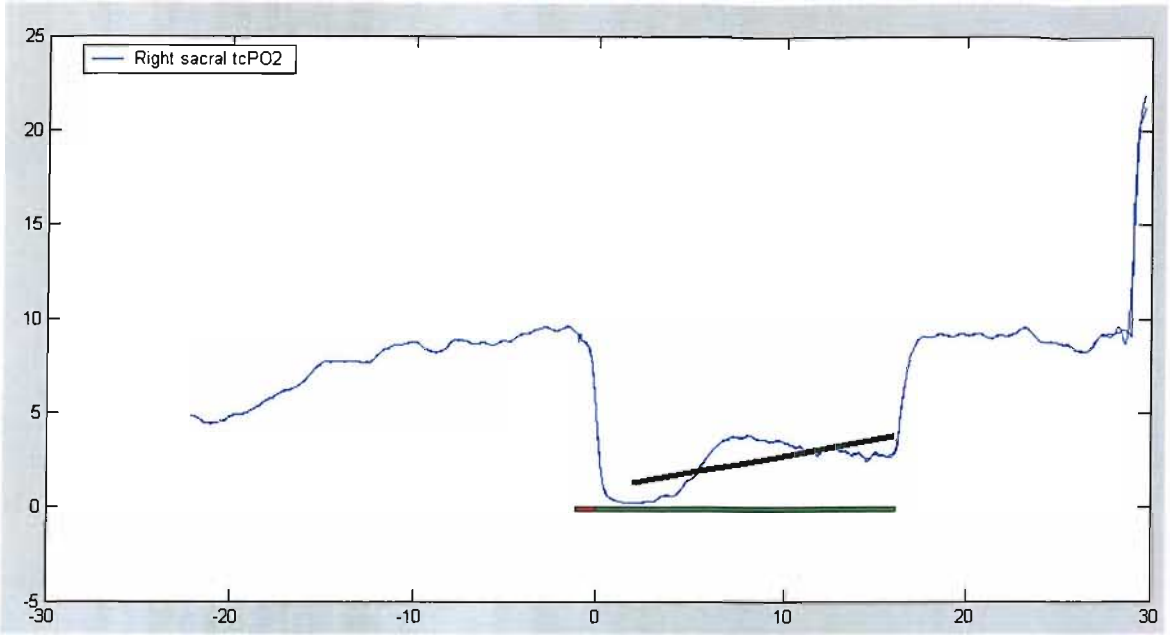



**Figure 4.19** **An illustration of the slope of response for $tcPO_2$ when sacral tissue subjected to pressure from which the gradient is calculated**

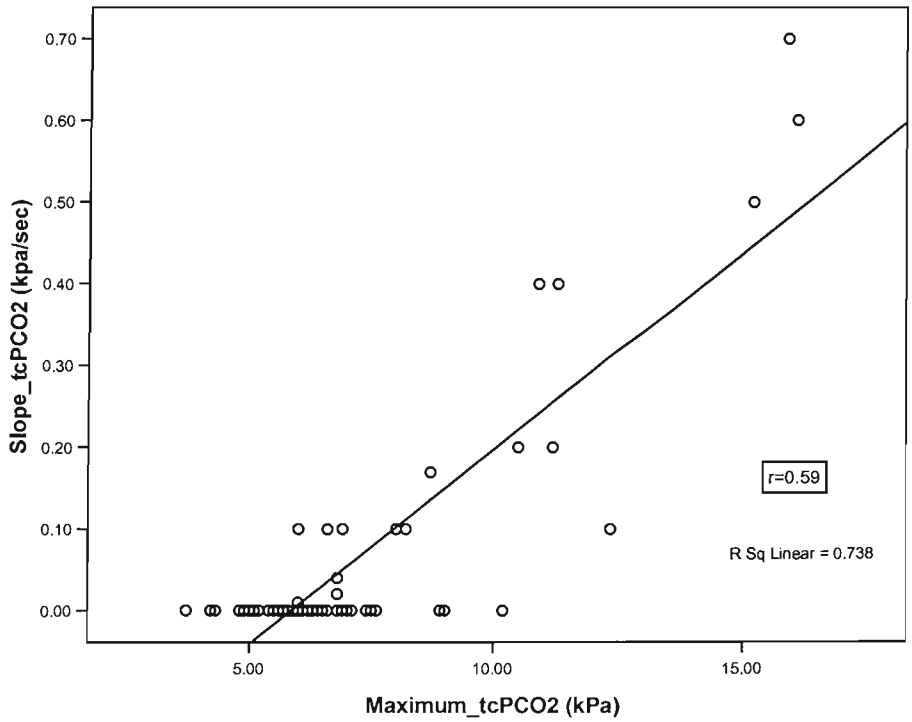



**Figure 4.20** **Correlation between the slope of the response of $tcPCO_2$ and maximum level of $tcPCO_2$ achieved under pressure**

### 4.2.7 Relationship Between Interface Pressure Exerted through Individuals' Own Body Mass and Response Type of $tcPO_2$ and $tcPCO_2$

The interface pressure identified for the 40 volunteers ranged from 38mmHg to 74mmHg (see Table 4.1). If a strong correlation exists between the interface pressure and response of sacral tissue tcPO2 and $tcPCO_2$ this would help to explain the range of responses observed between individuals to pressure. However, plotting the maximum level of tcPO2 and $tcPCO_2$ achieved under pressure against interface pressure applied, see Figures 4.21 and 4.22 respectively, and demonstrates no association. This was confirmed by calculating the correlation coefficient, using the Spearman's rank correlation. The correlation for $tcPO_2$ and interface pressure was found to be 0.042 and the correlation of $tcPCO_2$ and interface pressure was –0.12. Zero represents no correlation, therefore no association was found to exist between the interface pressure and the response of $tcPO_2$ and $tcPCO_2$ for the healthy volunteers. This supports the view that the compressive characteristics of soft tissues influence individuals' susceptibility to pressure (Swain and Bader, 2002). Therefore interface pressure is not a predictor of tissue tolerance.

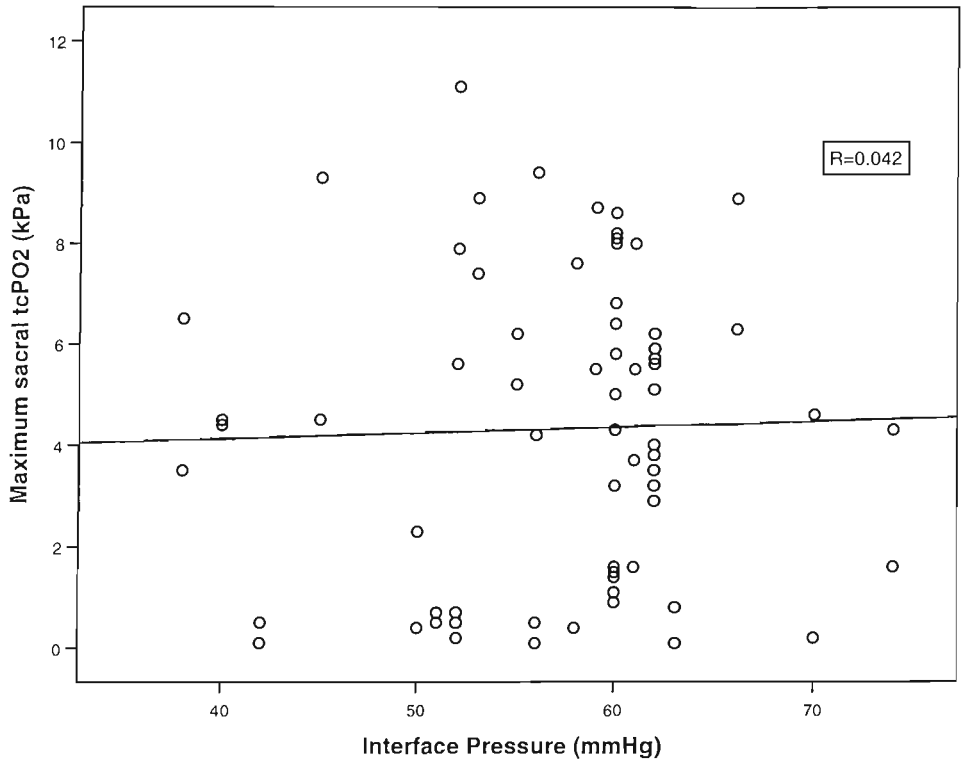



**Figure 4.21 Scatter diagram of $tcPO_2$ against interface pressure applied**

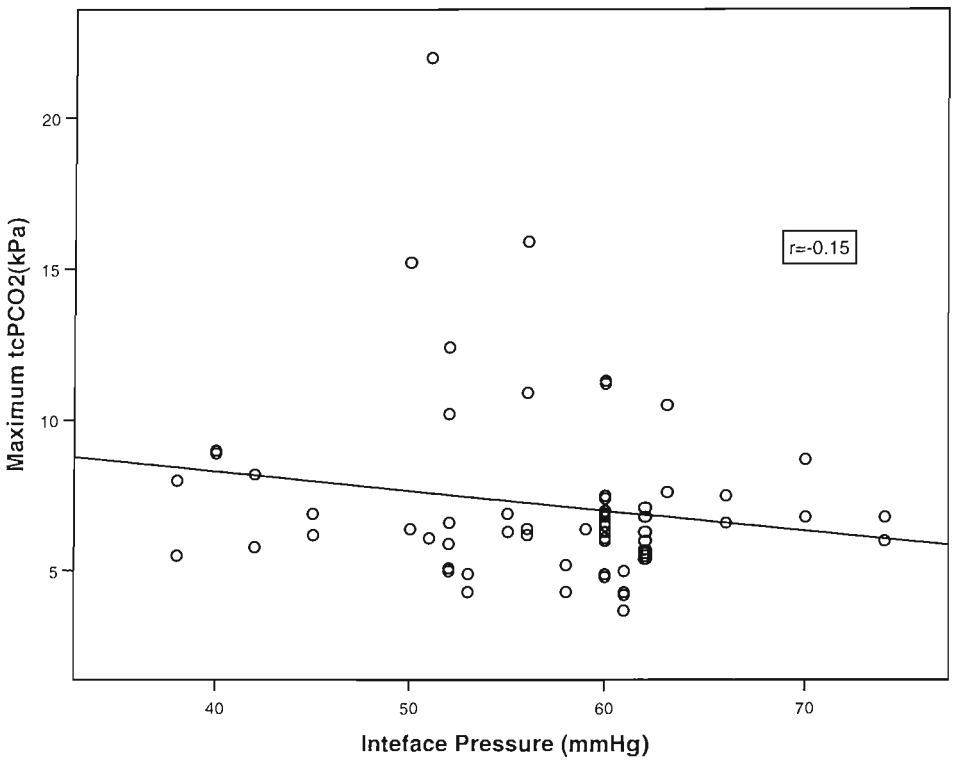



**Figure 4.22 Scatter diagram of $tcPCO_2$ against interface pressure applied**

## 4.3 Response of $tcPO_2$ and $tcPCO_2$ when Blood Flow Occluded

Pressure sufficient to cause capillary occlusion and cessation of blood flow has been defined by many as the key factor causing tissue damage in the development of pressure ulcers (Bliss, 1990 & Braden & Bergstorm, 1987 & Cullum & Clark, 1992). In section 4.2.6 it was postulated that the response type A(iii), indicated a pattern of response that represented local capillary blood flow occlusion due to the steady and significant increase in $tcPCO_2$.

The following experiment was designed to examine the response of $tcPO_2$ and $tcPCO_2$ following the application of pressure known to cause arterial blood vessel closure. The response derived from this experiment could then be compared with response type A(iii) to establish whether this response is representative of local capillary closure, and a reflection of the local tissues inability to tolerate the pressure being applied. It was anticipated that following the occlusion of blood flow via the brachial artery the $tcPO_2$ level will fall to zero and $tcPCO_2$ levels will rise significantly above normal levels.

### 4.3.1 Method

When measuring individual's systolic blood pressure the brachial arterial pressure is exceeded temporarily, causing cessation of blood flow due to occlusion of the brachial artery. This is achieved by applying an inflatable cuff around the upper arm and applying pressure until the radial pulse is no longer palpable. The pressure is measured using a sphygmomanometer. This technique was used to occlude the brachial artery, whilst simultaneous measurements of $tcPO_2$ and $tcPCO_2$ were recorded.

Four healthy volunteers were considered a sufficient number to explore the nature of response of $tcPO_2$ and $tcPCO_2$ to pressure known to cause arterial occlusion. The pressure sufficient to cause brachial artery closure was identified for each volunteer by palpating the radial artery whilst inflating the blood pressure cuff, the point at which the brachial artery is no longer palpable denotes the systolic pressure. This was also confirmed by gradually releasing the pressure and using a stethoscope over the brachial artery, in the cubical fossa, to audibly confirm the pressure at which the sounds first appear, denoting systolic pressure. The cuff was then released and the volunteer rested for a period of 15minutes.

The $tcPO_2$ and $tcPCO_2$ electrode was then positioned over the ulna, approximately 5cm below the elbow. A site over bone was selected because the sacral points of highest

pressure lie over the bony prominences of the sacrum. Measurements were taken for 10 minutes prior to baseline readings being recorded to ensure physiological stabilisation.

Following the initial baseline readings, pressure was applied directly over the electrode using a blood pressure cuff inflated to 200mmHg systolic pressure, so as to overcome individuals' systemic physiological response to arterial occlusion. Simultaneously a blood pressure cuff was applied to the upper arm, again to a pressure of 200mmHg. 200mmHg was applied to ensure arterial occlusion because natural fluctuations in blood pressure are caused by breathing and physiological compensatory mechanisms. The pressure was held for a maximum of 10 minutes, which was the maximum period of time tolerated by the volunteers, and measurements of $tcPO_2$ and $tcPCO_2$ recorded. The technique was repeated with 4 different volunteers.

The data collected was analysed to investigate:

- the baseline levels of $tcPO_2$ and $tcPCO_2$;
- change in levels of $tcPO_2$ and $tcPCO_2$ in response to arterial occlusion;
- the pattern of response.

### 4.3.2 Results

Baseline levels of $tcPO_2$ and $tcPCO_2$ were calculated from the ten minute period when no pressure was applied and the readings had stabilised. They were found to remain within the normal range. These data were slightly skewed and therefore non-parametric tests were applied (see Table 4.8).

Following the application of sufficient pressure to occlude the brachial artery, $tcPO_2$ levels decreased rapidly to mean value of 0.4 (±0.1) kPa, and $tcPCO_2$ level rose significantly and achieved a mean level of 13.4 kPa (±0.8). The overall results are presented in Table 4.9.

**Table 4.8 Baseline levels of $tcPO_2$ and $tcPCO_2$**

| (n = 4 ) | | Baseline Arterial tcPO2 | Baseline Arterial tcPCO2 |
|---|---|---|---|
| Mean | | 9.8 | 5.2 |
| Std. Error of Mean | | 0.3 | 0.1 |
| Median | | 9.6 | 5.1 |
| Standard Deviation | | 0.6 | 0.2 |
| Skewness | | 1.3 | 0.9 |
| Std. Error of Skewness | | 1.0 | 1.0 |
| Maximum | | 10.6 | 5.4 |
| Percentiles | 25 | 9.3 | 5.0 |
| | 50 | 9.6 | 5.1 |
| | 75 | 10.4 | 5.4 |

**Table 4.9 Maximum $tcPO_2$ and $tcPCO_2$ Levels Achieved when Sufficient Pressure Applied to Occlude the Brachial Artery**

| (n = 4 ) | | Maximum Arterial tcPO2 | Maximum Arterial tcPCO2 |
|---|---|---|---|
| Mean | | 0.4 | 13.4 |
| Std. Error of Mean | | 0.1 | 0.4 |
| Median | | 0.4 | 13.4 |
| Standard Deviation | | 0.1 | 0.8 |
| Skewness | | 0.8 | -0.1 |
| Std. Error of Skewness | | 1.0 | 1.0 |
| Maximum | | 0.6 | 14.1 |
| Percentiles | 25 | 0.3 | 12.6 |
| | 50 | 0.4 | 13.4 |
| | 75 | 0.6 | 14.1 |

The degree of difference between the baseline levels $tcPO_2$ and $tcPCO_2$ with those where pressure was applied are illustrated in Figure 4.23. The significance of the change in levels was examined using Wilcoxan signed rank test. The change in $tcPO_2$ and $tcPCO_2$ levels was significant for both with a p value of 0.06.

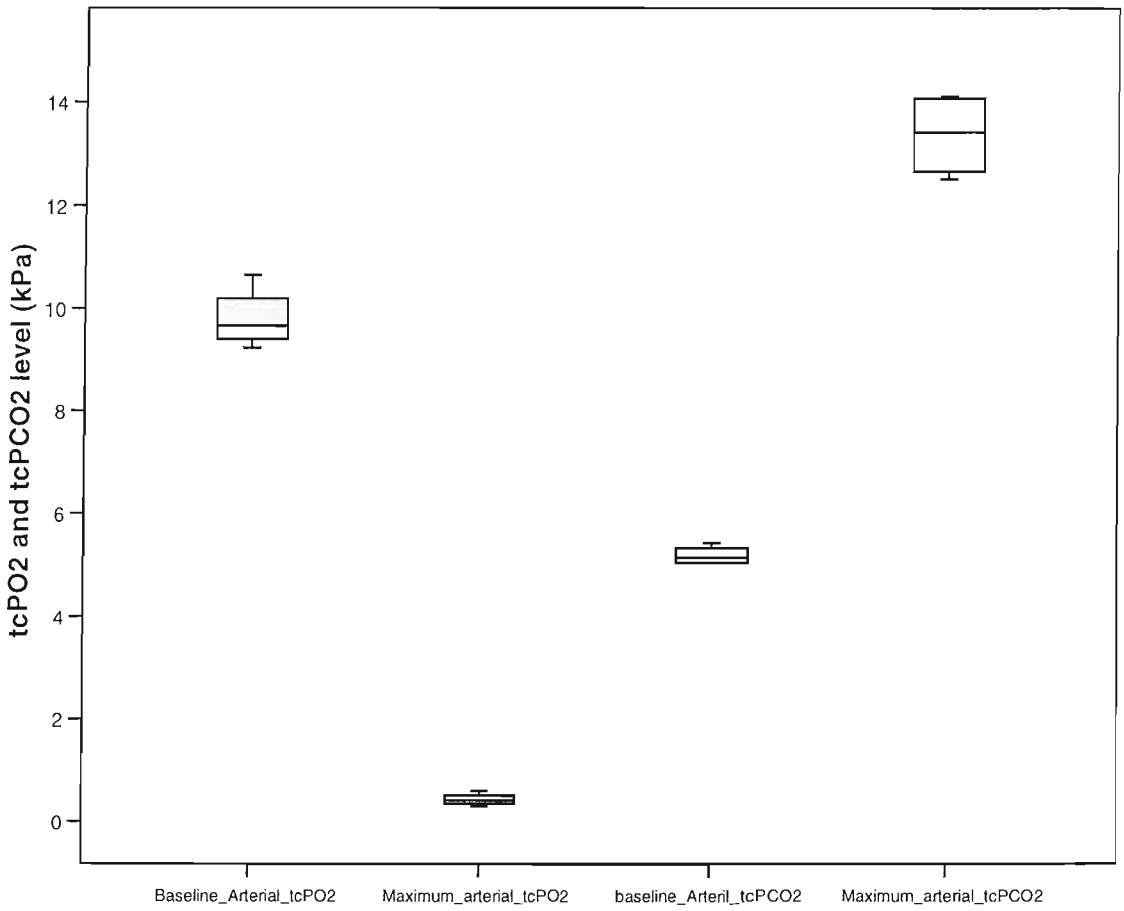



**Figure 4.23 Box and Whisker plot illustrating the range of baseline levels and maximum levels of $tcPO_2$ and $tcPCO_2$ achieved when sufficient pressure applied to cause brachial artery occlusion**

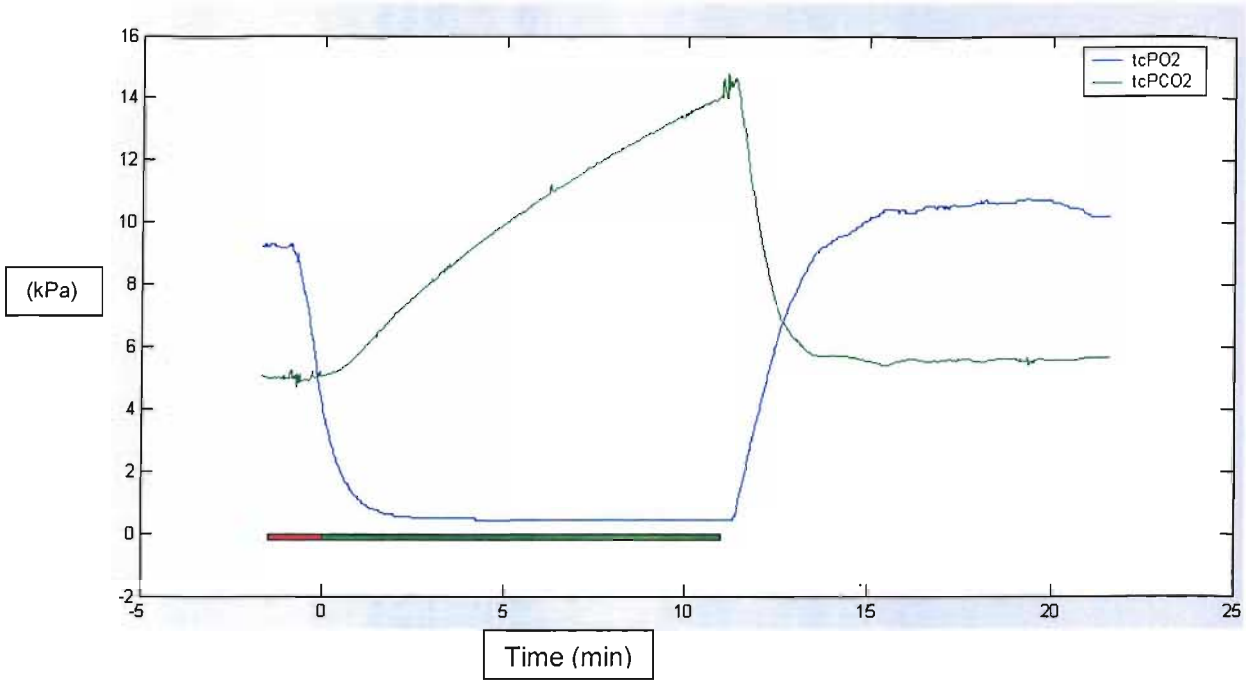



**Figure 4.24 Illustration of the pattern of response of changes in $tcPO_2$ and $tcPCO_2$ associated with arterial occlusion**

The pattern of response illustrated in Figure 4.24 demonstrates the rapid loss of oxygen and significant rise in carbon dioxide in response to the application of pressure sufficient to cause arterial occlusion. The increase in $tcPCO_2$ levels only ceased upon the release of the pressure.

On comparing the pattern of response with the severest type of response observed when pressure was applied to sacral tissue (type A(iii)response) the results appeared very similar and the correlation was identified using the Pearson's P correlation and r = 0.9, which is highly significant (illustrated in figure 4.25). Therefore the responses can be described as sufficiently associated that the response seen in sacral tissue can be described as representing capillary closure.

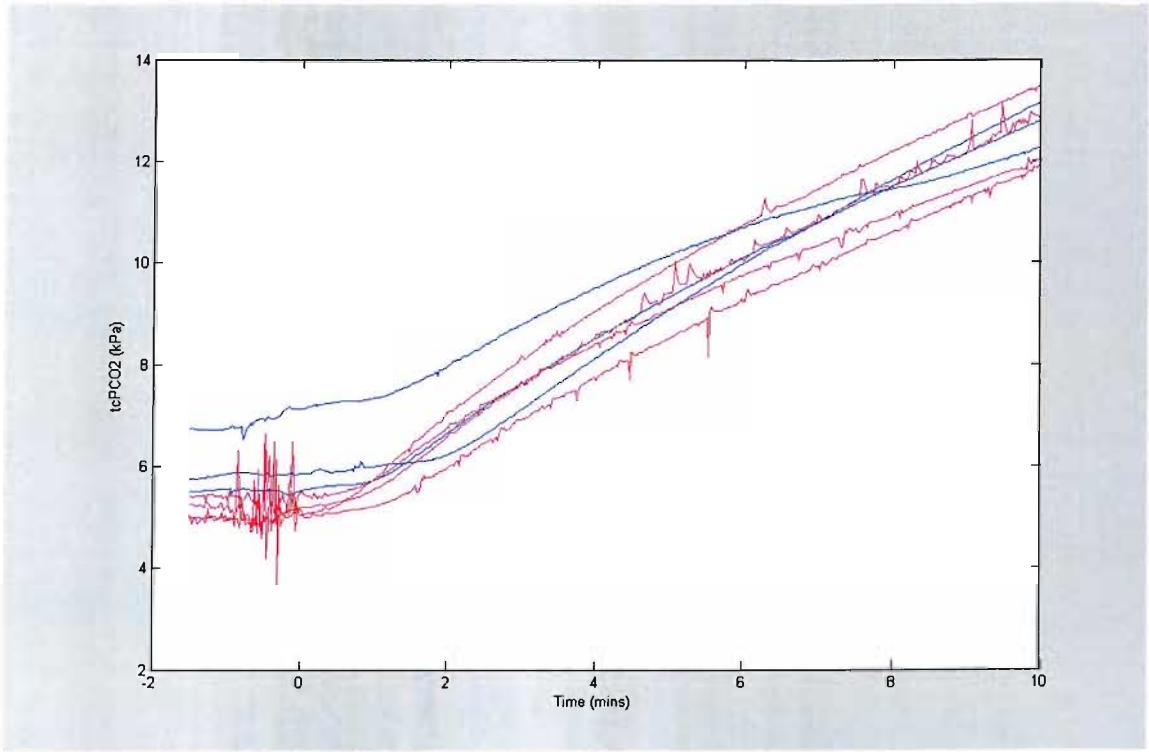



**Figure 4.25** **The response of $tcPCO_2$ to arterial closure (red lines) and type A(iii) response in sacral tissue (blue lines).**

## 4.4 Assessment of a Range of Pressures Applied to Sacral Tissue in Relation to the Type of Response of $tcPO_2$ and $tcPCO_2$

The level of pressure required to effect the levels of $tcPO_2$ and $tcPCO_2$ needs exploring further to better understand the relationship. If the pressure applied is identified as being close to a critical threshold level then one of two responses could result, dependant on local physiological changes and systemic physiological parameters e.g. blood pressure. If this is found to be the case it may explain the intra-subject variability between left and right sacral tissue responses to the same pressure, and inter-subject variability identified in section 4.1. Therefore more needs to be understood in relation to the range over which pressure can be increased or decreased before a change in response type results.

This experiment was designed to explore whether the response individuals' sacral tissue moves through the different response types of $tcPO_2$ and $tcPCO_2$, identified in section 4.2.6, when the pressure applied is decreased or increased sufficiently.

### 4.4.1 Method

A similar methodology described in experiment 4.1 was used to examine the relationship between sacral tissue perfusion and pressure applied as a consequence their own body mass, was utilised.

The aspects that differ in this experiment are that the two sacral sites identified had a range of pressures applied from 0 to 100mmHg. The pressure applied was increased in increments of 10mmHg and the individuals' sacral tissue was allowed to rest between the applications of pressure for a minimum of four hours to allow the sacral tissue to recover and avoid stimulating an active vasomotor response which would diminish the effect on subsequent loading (Bader, 1998, & Bader 1990b). Ten volunteers were considered to be a sufficient number to explore whether changes occur in the response of $tcPO_2$ and $tcPCO_2$ to a range of pressures. However, from twelve volunteers recruited, six withdrew leaving six participating in the study to completion.

As for the methodology in experiment 4.1 the $tcPO_2$ and $tcPCO_2$ electrodes were allowed 10minutes for physiological stabilisation, followed by 10 minutes of baseline measurement recording. The pressure was then applied for a period of 15minutes, after which the pressure was released.

The higher pressures applied (>70mmHg) were checked using the Kikuhime Resonant Pressure Sensor (described in section 3.3.1), which was positioned next to the $tcPO_2$ and $tcPCO_2$ electrodes. This was because as the bladder, used to apply the pressure to the sacral area, was inflated the contact surface area over the sacral site was reduced, due to the increased curvature of the bladder as the pressure applied was increased. Therefore it was essential that the pressure achieved was checked at the interface and degree of accuracy confirmed.

The changes in $tcPO_2$ and $tcPCO_2$ were quantified by the maximum levels of $tcPO_2$ and $tcPCO_2$ achieved under pressure, the Area Under the Curve (AUC) during the period of pressure and the 3 response types of $tcPO_2$ and $tcPCO_2$, identified in section 4.1. The range of responses as the pressure is increased will be examined to identify whether individuals moves through all response types identified in 4.2.6, and the range of pressures that each response type extends over. The AUC and maximum level of $tcPO_2$ and $tcPCO_2$ achieved under pressure is illustrated in figure 4.26

The changes in $tcPO_2$ and $tcPCO_2$ were examined by:

- identifying the type of response, as identified in section 4.2.6, that each sacral site presents over the range of pressures;
- the pattern of response types in relation to the steady increase in pressure applied;
- the relationship between the maximum $tcPO_2$ and $tcPCO_2$ achieved in sacral tissue when exposed to increases in the pressure applied and the maximum levels achieved;
- the total level of oxygen remaining in the sacral tissue and carbon dioxide accumulated and how the levels change as the pressure was increased. This will be demonstrated by calculating the area under the curve for the period that pressure is applied.

## 4.4.2 Results

### 4.4.2.1 Demographics of the Six Volunteers

With the exception of one of the volunteers the ages ranged between 40 and 44yrs of age. The final volunteer was 60 years of age. The body mass indices ranged from being underweight to obese. The demographics for all volunteers are summarised in Table 4.10.

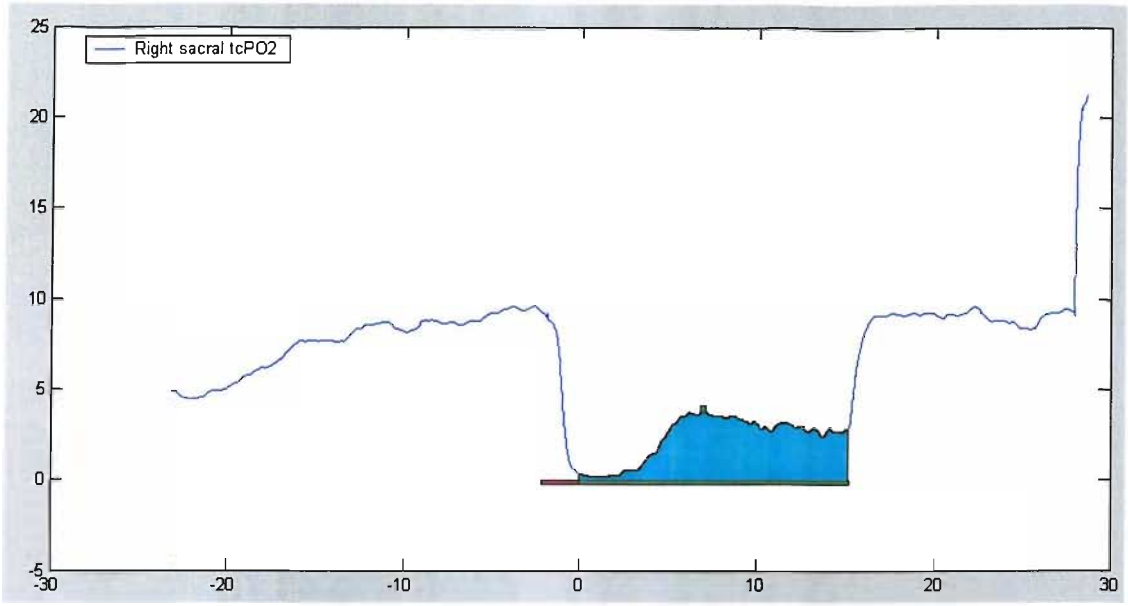



**Figure 4.26 Illustration of the AUC identified by the shaded turquoise area and the maximum level of $tcPO_2$ identified by the light green square.**

**Table 4.10 Summary of Demographic Details for the Six Volunteers**

| Sex | Age (yrs) | Weight (kg) | Height (cms) | Body Mass Index ($kg/m^2$) | Systolic Blood pressure (mmHg) | Diastolic Blood Pressure (mmHg) | Peak Sacral Pressure (mmHg) |
|---|---|---|---|---|---|---|---|
| M | 40 | 131.3 | 179 | 41 | 110-140 | 80 | 60 |
| F | 43 | 83 | 163 | 31.2 | 110 | 70 | 45 |
| M | 43 | 77.2 | 173 | 25.8 | 110 | 70 | 63 |
| F | 60 | 63.5 | 161 | 24.5 | 140-145 | 70 | 66 |
| M | 43 | 96 | 188 | 27.2 | 115-130 | 80 | 74 |
| M | 44 | 64 | 180 | 19.8 | 100-115 | 70 | 52 |
| **Mean** | 45.5 | 85.5 | 174 | 28.3 | | | 60 |
| **Min** | 40 | 63.5 | 161 | 19.3 | 100 | 70 | 45 |
| **Max** | 60 | 131.3 | 188 | 41 | 145 | 80 | 74 |

### 4.4.2.2 Range of Responses of $tcPO_2$ and $tcPCO_2$ Following the Application of a Range of Pressures, Increased in Increments of 10mmHg from 0 to 100mmHg.

Responses for eleven of the twelve sacral sites demonstrated changes of $tcPO_2$ and $tcPCO_2$ from levels indicating minimal changes in oxygen and carbon dioxide levels (response Type D) when 10mmHg was applied, to the severest type of response being exhibited with $tcPO_2$ levels falling and remaining below 1kPa and $tcPCO_2$ accumulating

steadily and significantly to levels in excess of 12kPa (response Type A(iii)), by the time 100 mmHg was applied. The responses of each sacral site at each pressure are presented in Table 4.11.

Table 4.11 indicates that $tcPO_2$ is more sensitive to initial changes in pressure and $tcPCO_2$ relatively insensitive to changes in pressure until the pressure has been sufficient to cause the loss of $tcPO_2$ below 1kPa. The transition range between Type D response to Type A(ii) response for $tcPO_2$ ranges from 10mmHg for six of the sacral sites, to up to 30mmHg for one sacral site. The remaining four sites demonstrated response type D to A within 19mmHg. Fluxuations in the type of response existed with increasing pressure between type D and C, but once the response type changed from Type D to Type B the response did not revert back to a Type D response. The fluxations between response types of $tcPO_2$ in four of the sacral sites (Volunteer 2, right sacral site; Volunteer 5, right sacral site; Volunteer 6 right sacral site; Volunteer 5, left sacral site) to increasing levels of pressure would indicate a transitional phase representing the critical nature of pressure and response of local tissue.

Changes in the response type of $tcPCO_2$ were only observed once $tcPO_2$ levels changed to a type A or B response. The severest response type of $tcPCO_2$ (response type A(iii) was only observed when a type A response for $tcPO_2$ occurred, therefore indicating that $tcPCO_2$ is a stronger indicator for the latter stages of ischaemia.
The left and right sacral sites for the same volunteer responded differently to the same pressures with only volunteer one's left and right sacral site responding in the same order to pressures. This supports the findings of 4.2 and substantiates the need for each sacral site to be treated independently.

**Key explaining colour coding for Table 4.11 below.**

| Response Type | Description of response Types |
|---|---|
| D | $tcPO_2$ level does not fall below 1kPa and shows varying degrees of recovery |
| C | $tcPO_2$ level falls below 1kPa, but shows varying degrees of recovery |
| B | $tcPO_2$ level fall below 1kPa, and doesn't recover above 2kPa |
| A | $tcPO_2$ level fall below 1kPa and remain below 1kPa |
| Ai | $tcPCO_2$ levels remain within normal range, no significant increase observed |
| Aii | $tcPCO_2$ levels increase (exceeding the normal range) and plateau >8 ≤ 12kPa |
| Aiii | $tcPCO_2$ levels significantly increase to levels >12kPa |

**Table 4.11 Response Types of Sacral Tissue to Pressures Ranging from 10 to 100mmHg (key shown on the previous page)**

| | Volunteer 1 | | Volunteer 2 | | Volunteer 3 | | Volunteer 4 | | Volunteer 5 | | Volunteer 6 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Right sacral site | | Right sacral site | | Right sacral site | | Right sacral site | | Right sacral site | | Right sacral site | |
| Pressure applied (mmHg) | $tcPO_2$ | $tcPCO_2$ | $tcPO_2$ | $tcPCO_2$ | $tcPO_2$ | $tcPCO_2$ | $tcPO_2$ | $tcPCO_2$ | $tcPO_2$ | $tcPCO_2$ | $tcPO_2$ | $tcPCO_2$ |
| 10 | D | Ai | D | Ai | D | Ai | D | Ai | D | Ai | D | Ai |
| 20 | D | Ai | D | Ai | D | Ai | D | Ai | D | Ai | D | Ai |
| 30 | D | Ai | D | Ai | D | Ai | D | Ai | B | Ai | D | Ai |
| 40 | D | Ai | C | Ai | D | Ai | D | Ai | A | Ai | B | Ai |
| 50 | D | Ai | D | Ai | C | Ai | D | Ai | C | Ai | A | Ai |
| 60 | D | Ai | A | Aii | C | Ai | D | Ai | A | Aiii | B | Ai |
| 70 | D | Ai | B | Ai | A | Aii | D | Ai | A | Aiii | A | Aii |
| 80 | D | Ai | A | Aiii | A | Aiii | A | Aiii | A | Aiii | A | Aii |
| 90 | B | Aii | A | Aiii | A | Aiii | A | Aiii | A | Aiii | A | Aiii |
| 100 | A | Aiii | A | Aiii | A | Aiii | A | Aiii | A | Aiii | A | Aiii |
| | **Volunteer 1** | | **Volunteer 2** | | **Volunteer 3** | | **Volunteer 4** | | **Volunteer 5** | | **Volunteer 6** | |
| | Left sacral site | | Left sacral site | | Left sacral site | | Left sacral site | | Left sacral site | | Left sacral site | |
| Pressure applied (mmHg) | $tcPO_2$ | $tcPCO_2$ | $tcPO_2$ | $tcPCO_2$ | $tcPO_2$ | $tcPCO_2$ | $tcPO_2$ | $tcPCO_2$ | $tcPO_2$ | $tcPCO_2$ | $tcPO_2$ | $tcPCO_2$ |
| 10 | D | Ai | D | Ai | D | Ai | D | Ai | D | Ai | D | Ai |
| 20 | D | Ai | D | Ai | D | Ai | D | Ai | C | Ai | D | Ai |
| 30 | D | Ai | D | Ai | D | Ai | D | Ai | D | Ai | D | Ai |
| 40 | D | Ai | D | Ai | D | Ai | D | Ai | D | Ai | D | Ai |
| 50 | D | Ai | D | Ai | D | Ai | D | Ai | A | Aii | D | Ai |
| 60 | D | Ai | A | Ai | D | Ai | D | Ai | A | Aii | D | Ai |
| 70 | D | Ai | A | Aiii | D | Ai | D | Ai | A | Aiii | D | Ai |
| 80 | D | Ai | A | Aiii | D | Ai | A | Aiii | A | Aiii | D | Ai |
| 90 | B | Aii | A | Aiii | D | Ai | A | Aii | A | Aiii | A | Aiii |
| 100 | A | Aiii | A | Aiii | D | Ai | A | Aiii | A | Aiii | A | Aiii |

Four of the twelve sacral sites exhibited all response types of $tcPCO_2$ in order of severity as the pressure was increased. The critical threshold of pressure for three of the type A(ii) responses was < 10mmHg and one between 10 to 19mmHg. Four of the sacral sites changed from type A (i) to A(iii) with increasing levels of pressure. A type A(ii) may have occurred if the pressure was increased in smaller increments, for example 5mmHg. Eleven of the twelve sacral sites changed from a type A(i) response to type A(iii) response as the pressure applied was increased from 10 to 100mmHg, with both sacral sites for volunteer 5 presenting with a type A(iii) response with 50mmHg. The amount of pressure required to result in a change in response type from a Type A(i) to Type A(iii) response ranged from less than 9mmHg (six sacral sites) to 29mmHg (two sacral sites). Response type A(ii) and A(iii) were observed at pressures as low as 50mmHg, which is lower than Knight et al (2001) where $tcPCO_2$ levels were observed to exceed 10.5kPa only when pressure applied exceeded 80mmHg. It was observed that once a type A(iii) response occurred, in response to the pressure being applied, fluxuations between a type A(iii) and A(ii) occurred, but not type A(i) and A(iii).

To ensure that the technique for applying the pressure to the sacral sites remained accurate when the higher pressures were applied the interface pressure was checked between the inflated bladder and sacral tissue for pressures from 70 to 100mmHg using the Kikuhime Resonant Pressure Sensor. The results demonstrated that the pressure applied was accurate to within ±1mmHg. This was particularly reassuring for volunteer 3, whose results showed the greatest tolerance to pressure with the left sacral site's response type for $tcPO_2$ not deviating from Type D and $tcPCO_2$ response type not deviating from response type A(i), even with 100mmHg.

A typical example of the changes in $tcPO_2$ and $tcPCO_2$ as the pressure applied is increased from 10 to 100mmHg can be seen in Figure 4.27 and 4.28. Figure 4.27 indicates that as the pressure increases the oxygen levels gradually falls. Using the response types identified in section 4.2.5 a type D response is shown to occur for this volunteer from 10 to 80mmHg. The critical threshold is identified at 90mmHg when a type B response is observed followed by a response type A(i) at 100mmHg. The corresponding changes in $tcPCO_2$ are illustrated in Figure 4.28 with the critical threshold for the type A(ii) response observed at 90mmHg and 100mmHg for a type A(iii) response.

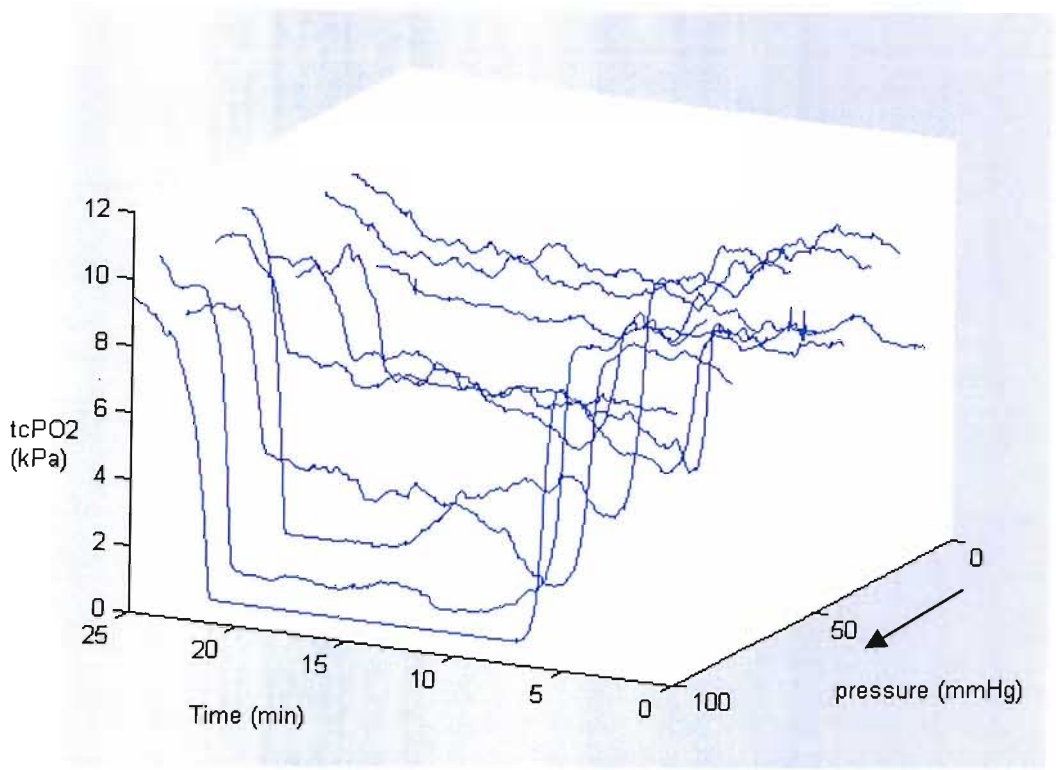



**Figure 4.27 Illustrating the changes in response of $tcPO_2$ in sacral tissue as the pressure is increased from 10 to 100mmHg in Volunteer 1, Right sacral site**

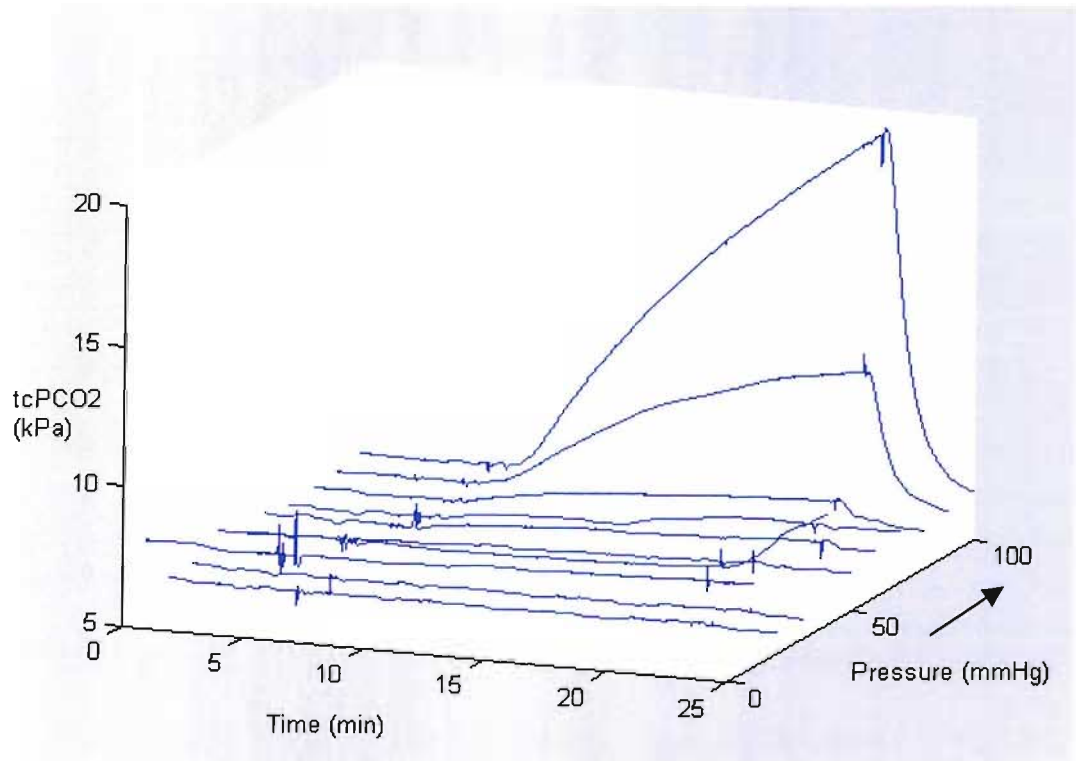



**Figure 4.28 Illustrating the changes in response of $tcPCO_2$ in sacral tissue as the pressure is increased from 10 to 100mmHg in Volunteer 1, right sacral site**

#### 4.4.2.3 Defining the Response Type for $tcPCO_2$ to Pressure in Relation to Gradient of Response.

It has already been identified that the slope of the response of $tcPO_2$ to pressure, has a weak correlation with the type of response to pressure. (see section 4.2.6, Figure 4.19). In contrast the correlation between response type and slope for $tcPCO_2$ was found to be sufficiently significant with an r value of 0.6. Therefore only the relationship between the response type and gradient for $tcPCO_2$ will be examined across the 120 data sets collected through this part of the study. The slope was calculated from the linear regression of all data points starting two minutes after pressure applied was achieved, until the point of pressure release.

Using the Spearman's rank correlation of coefficient the significance of the relationship between $tcPCO_2$ and slope was found to be highly significant with a value of R=0.81, significant at the 0.01 level. Figure 4.29 illustrates the correlation and also has the boundaries for three $tcPCO_2$ response types identified.

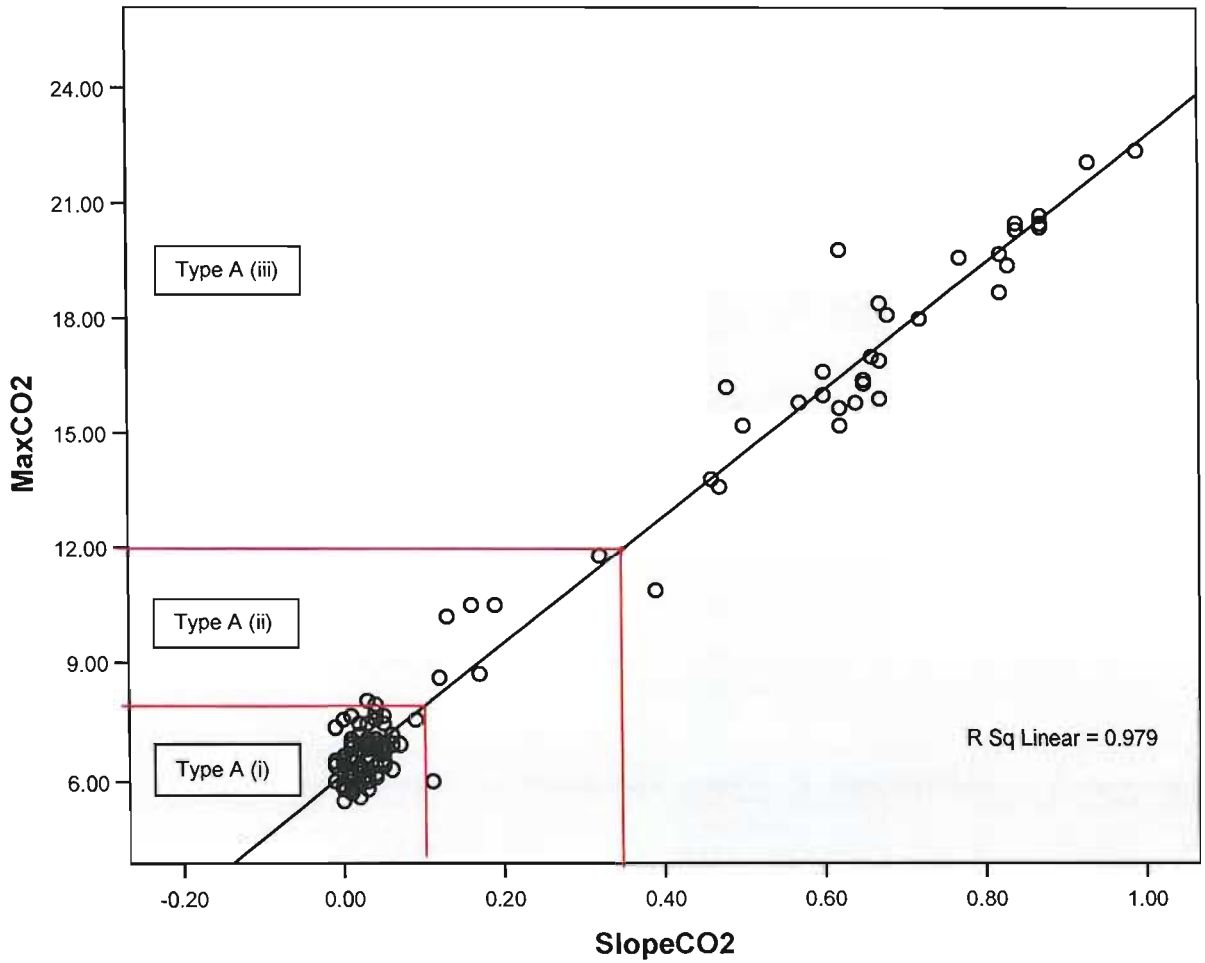



**Figure 4.29** **Scatter diagram of gradient for slope of $tcPCO_2$ plotted against the maximum level of $tcPCO_2$ achieved at the sacral sites under pressure. The red lines identify the upper limit of response for Type A(i), and the upper limit Type A(ii) response as defined in Section 4.2.5**

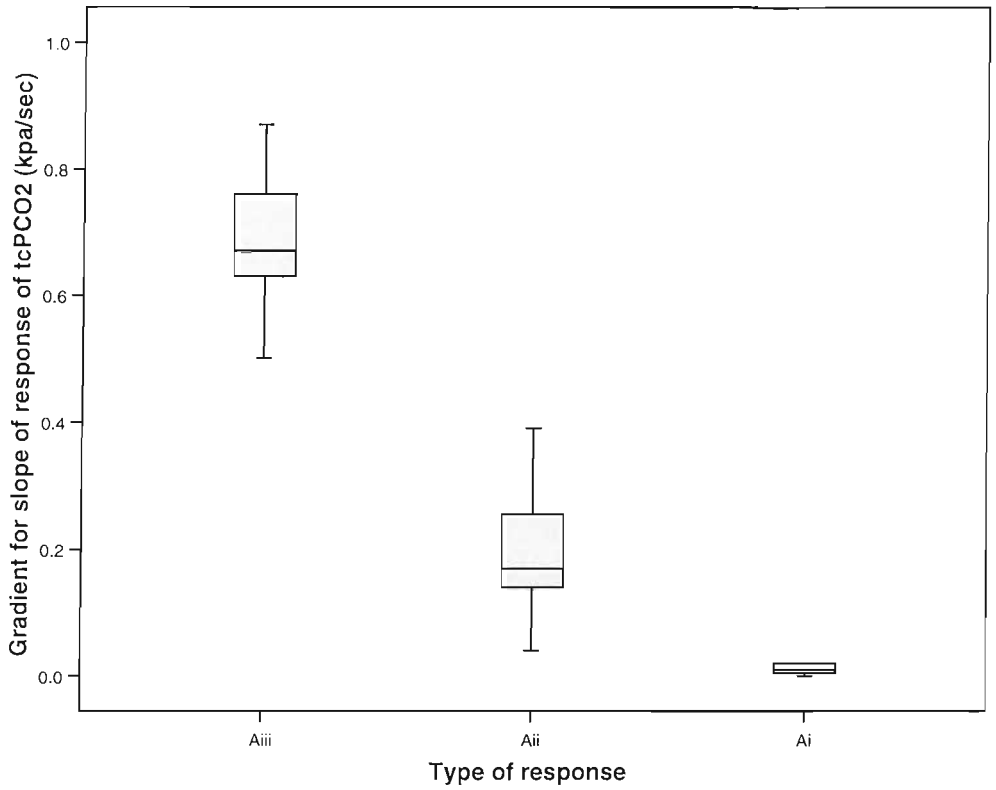



**Figure 4.30 Box and whisker plot illustrating the range of the gradient of the slope for each type of $tcPCO_2$ response**

Figure 4.30 illustrates the increase in gradient with the three response types (type A(i),(ii) & (iii)) of $tcPCO_2$ to pressure. The difference between the gradients of each response type was found to be significant using the Friedman test, with a P value of 0.001.

Type A(iii) response presented with a distinct shape to the curve following the application of pressure. The shape of the curve can be seen in Figure 4.28, when the sacral tissue was subjected to 100mmHg. In order to examine the nature of the response these data were examined using generalised linear modelling to investigate both linear and quadratic interactions. A significant linear and quadratic interaction was identified. The line of best fit through the quadratic curve of all type A(iii) responses had an $R^2$ value of 0.86, highly significant at a level of 0.000. Figure 4.30 illustrates the curves for all the type A(iii) responses. The data from 2 minutes after the pressure was applied to the point of pressure release was plotted for each response in order to see the full range of variation between the responses. The red dotted line though the centre of the responses represents the line of best fit.

The quadratic curve equation is: $y = at^2 + bt + c$. The 95% confidence interval for all responses shown in Figure 4.30 was identified and the range for the time coefficient ($b$), remained constant, the time squared coefficient ($a$) ranged from $-8.04 \times 10^{-6}$ to $-7.49 \times 10^{-6}$, and the constant ( $c$ ) ranged from 5.7 to 5.8.

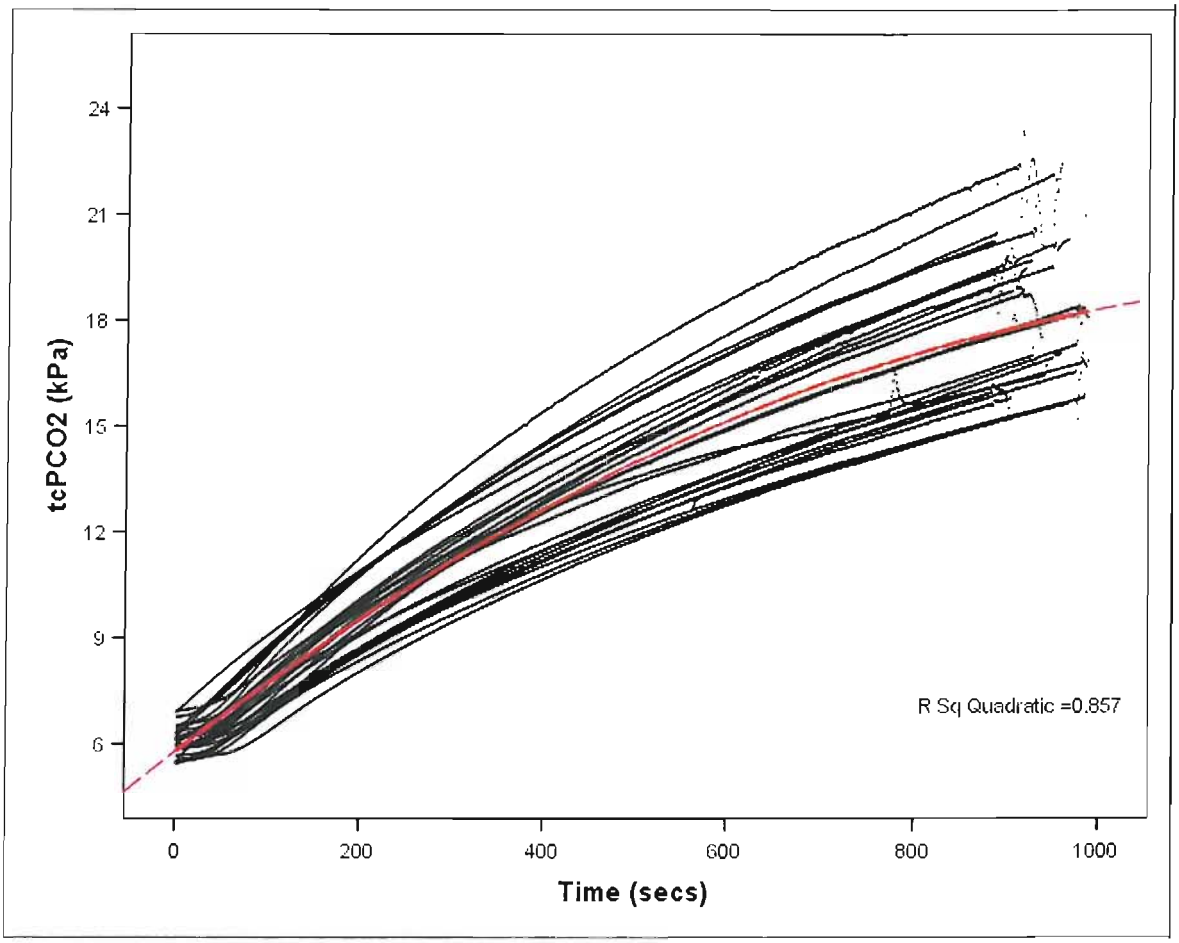



**Figure 4.31 Response curves of all type A(iii) responses**

A significant difference has been identified between the slopes relating to the three $tcPCO_2$ response types, A(i), (ii) and (iii), and are sufficiently independent groups to be considered as a means of defining the response type of sacral tissue to pressure. The nature of type A(iii) response presented has been identified as having a specific type of response curve, illustrated in Figure 4.27 and 4.30, and was defined as a quadratic curve. Response type A(iii) for $tcPCO_2$ maybe definable in future studies through the gradient of the slope of response and by the quadratic response of the curve. This result differs from the finding of Hotter et al (2004) who identified a linear exponential response of the renal tissue $tcPCO_2$ with levels plateauing after ten minutes renal artery occlusion in rats. However, other animal

studies did not describe a plateaued effect after the significant increase (Johnson et al, 1991) and further work is required to examine what may happen after a longer period of time.

#### 4.4.2.4 Maximum Level of $tcPO_2$ and $tcPCO_2$ Achieved Under Pressure and Relationship Between Both Gases

In order to further explore the range of pressure resulting in the different types of response observed the maximum level of $tcPO_2$ and $tcPCO_2$ maintained when pressure was applied to the sacral tissue was examined. The maximum level of $tcPO_2$ identified whilst the tissue was exposed to pressure indicates the degree of tolerance of local sacral tissue to the pressure being applied. As identified in section 4.2.6 $tcPO_2$ levels initially fall in response to pressure, but then either increase to varying degrees representing different levels of recovery, or remain below 1kPa. The first two of the fifteen minutes that the sacral tissue was subjected to pressure, were excluded because the initial period of decline could lead to the identification of a higher level of recovery than was actually achieved.

The two sacral sites for each volunteer were treated as independent sites, following the findings in section 4.2.4. Therefore twelve sets of results were analysed from the six volunteers.

Table 4.12 identifies the range of the maximum levels of $tcPO_2$ and $tcPCO_2$ achieved within the local sacral tissue in response to each level of pressure applied. Even at 20mmHg of pressure was sufficient for some sacral sites to respond with $tcPO_2$ levels falling below the normal range of 5.1-11.8kPa, and $tcPCO_2$ exceeding the upper limit of the normal range (4 to 7.4kPa). Although the minimum level of $tcPO_2$ falls to 0.2 at 40mmHg there isn't a significant change in $tcPCO_2$ levels until 50mmHg. This supports the findings of section 4.2.6 which identified that changes in $tcPCO_2$ only appear to occur once $tcPO_2$ levels stabilised below 1kPa. The range of responses of the twelve sacral sites are also presented in Table 4.10 and indicate the variability between the sites in response to pressure. Even with 100mmHg applied to the sacral sites the $tcPO_2$ levels ranged from 0.1 to 3kPa and $tcPCO_2$ from 7 to 22.4kPa. The range of responses is even more apparent in Figure 4.33, which shows the overall trend in changes of $tcPO_2$ and $tcPCO_2$, with the upper and lower quartiles identified by the outer markers on the lines. The inter-quartile range excludes outliers, and shows that as the pressure applied increases the sacral tissue demonstrates a

steady reduction in $tcPO_2$ levels, with all but two sacral sites falling below 1kPa at 90mmHg. In contrast as the pressure applied increases there are minimal changes in $tcPCO_2$ levels observed for the majority of sacral sites, until 70mmHg is achieved. At this point a significant change is apparent, followed by a steady increase in $tcPCO_2$ levels as the pressure is increased. When 100mmHg is applied the $tcPCO_2$ levels in all but one sacral site, exceed 15kPa with the maximum level achieved reaching 22.4kPa.

The relationship between changes in levels of sacral tissue $tcPO_2$ and $tcPCO_2$ when exposed to pressure were examined using Spearman's Rank correlation due to the non-parametric nature of the results. The correlation was found to be –0.75, significant at the 0.01 level (2-tailed), which indicates a strong correlation.

**Table 4.12 Summary of Maximum Level of $tcPO_2$ and $tcPCO_2$ Achieved at the Twelve Sacral Sites of Six Volunteers when Pressure Applied from 10 to 100mmHg.**

| Pressure Applied (mmHg) | | Maximum $tcPO_2$ achieved (kPa) | Maximum level of $tcPCO_2$ achieved (kPa) |
|---|---|---|---|
| 0 (n=12) | Minimum | 4.5 | 4 |
| | Maximum | 11.8 | 7.4 |
| | Median | 8.9 | 5.8 |
| | Inter-quartile Range | 7.8 – 9.7 | 5.6 – 6.1 |
| 10 (n=12) | Minimum | 4.1 | 5.8 |
| | Maximum | 11.2 | 8 |
| | Median | 7.9 | 6.5 |
| | Inter-quartile Range | 6.6 – 10.2 | 6.5 – 6.9 |
| 20 (n=12) | Minimum | 3 | 5.5 |
| | Maximum | 11.3 | 7 |
| | Median | 7.8 | 6.1 |
| | Inter-quartile Range | 6.2 – 10.4 | 5.9 – 6.5 |
| 30 (n=12) | Minimum | 1.3 | 5.6 |
| | Maximum | 10.1 | 7.5 |
| | Median | 7.3 | 6.2 |
| | Inter-quartile Range | 5.1 – 9.1 | 5.9 – 6.5 |
| 40 (n=12) | Minimum | 0.2 | 5.8 |
| | Maximum | 9.8 | 7.6 |
| | Median | 5.5 | 6.6 |
| | Inter-quartile Range | 3.3 – 8.5 | 5.9 – 7.2 |
| 50 (n=12) | Minimum | 0.1 | 6.2 |
| | Maximum | 9.3 | 15.2 |
| | Median | 4.9 | 6.7 |
| | Inter-quartile Range | 2.7 – 7.9 | 6.4 – 7.3 |
| 60 (n=12) | Minimum | 0.1 | 5.7 |
| | Maximum | 9.4 | 15.9 |
| | Median | 4.0 | 6.5 |
| | Inter-quartile Range | 0.7 – 7.1 | 6 – 8.3 |
| 70 (n=12) | Minimum | 0.1 | 6.3 |
| | Maximum | 8.9 | 20.7 |
| | Median | 2.8 | 7.4 |
| | Inter-quartile Range | 0.2 – 6 | 6.6 – 14.2 |
| 80 (n=12) | Minimum | 0.1 | 6.4 |
| | Maximum | 7.4 | 20.5 |
| | Median | 0.4 | 13.7 |
| | Inter-quartile Range | 0.2 – 3.7 | 7.2 – 16.7 |
| 90 (n=12) | Minimum | 0.1 | 7.4 |
| | Maximum | 7.1 | 20.5 |
| | Median | 0.4 | 15.9 |
| | Inter-quartile Range | 0.2 – 0.6 | 10.8 – 20.2 |
| 100 (n=12) | Minimum | 0.1 | 7 |
| | Maximum | 3.1 | 22.4 |
| | Median | 0.3 | 18.2 |
| | Inter-quartile Range | 0.1 – 0.6 | 16.3 - 20 |

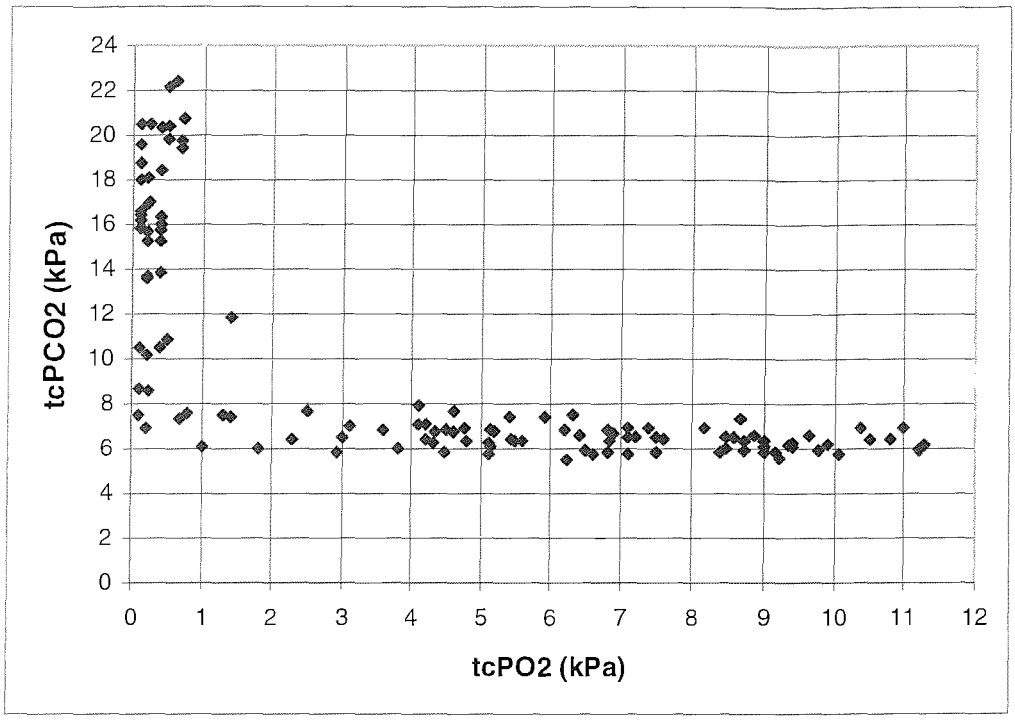



**Figure 4.32 Relationship between changes in sacral tissue $tcPO_2$ and $tcPCO_2$ in response to increases in pressure**

Figure 4.32 shows that once the $tcPO_2$ levels fall below 1kPa, the increase in $tcPCO_2$ levels were noticeable and ranged from remaining within normal levels, to rising significantly to levels exceeding 22kPa. This supports the findings of section 4.2.6. In addition, as the $tcPCO_2$ levels rise there is an apparent break between data points at 12kPa, which also supports the upper limit of the second type of $tcPCO_2$ response (Type A ii), identified in section 4.2.6. The relationship between tcPO2 and $tcPCO_2$ shown in Figure 4.32 supports the work of Tonnessen (1997) when examining changes in organ $PCO_2$ levels between aerobic and anaerobic metabolism.

The normal range of $tcPCO_2$ was identified from the baseline readings of the sacral site and was identified as ranging from 4 to 7.4kPa. This is close to that found for the forty volunteers that participated in the research examining the response of individuals' sacral tissue perfusion to their own body mass (see section 4.2.3) where the normal range was identified as ranging from 3.6 to 6.9mmHg. Figure 4.33 illustrates that $tcPO_2$ and $tcPCO_2$ levels can remain within the normal range when pressure is applied. The $tcPO_2$ levels start decreasing in response to increasing pressure before increases in $tcPCO_2$ levels are observed, and $tcPCO_2$ levels continue to increase after $tcPO_2$ levels have 'bottomed-out'.

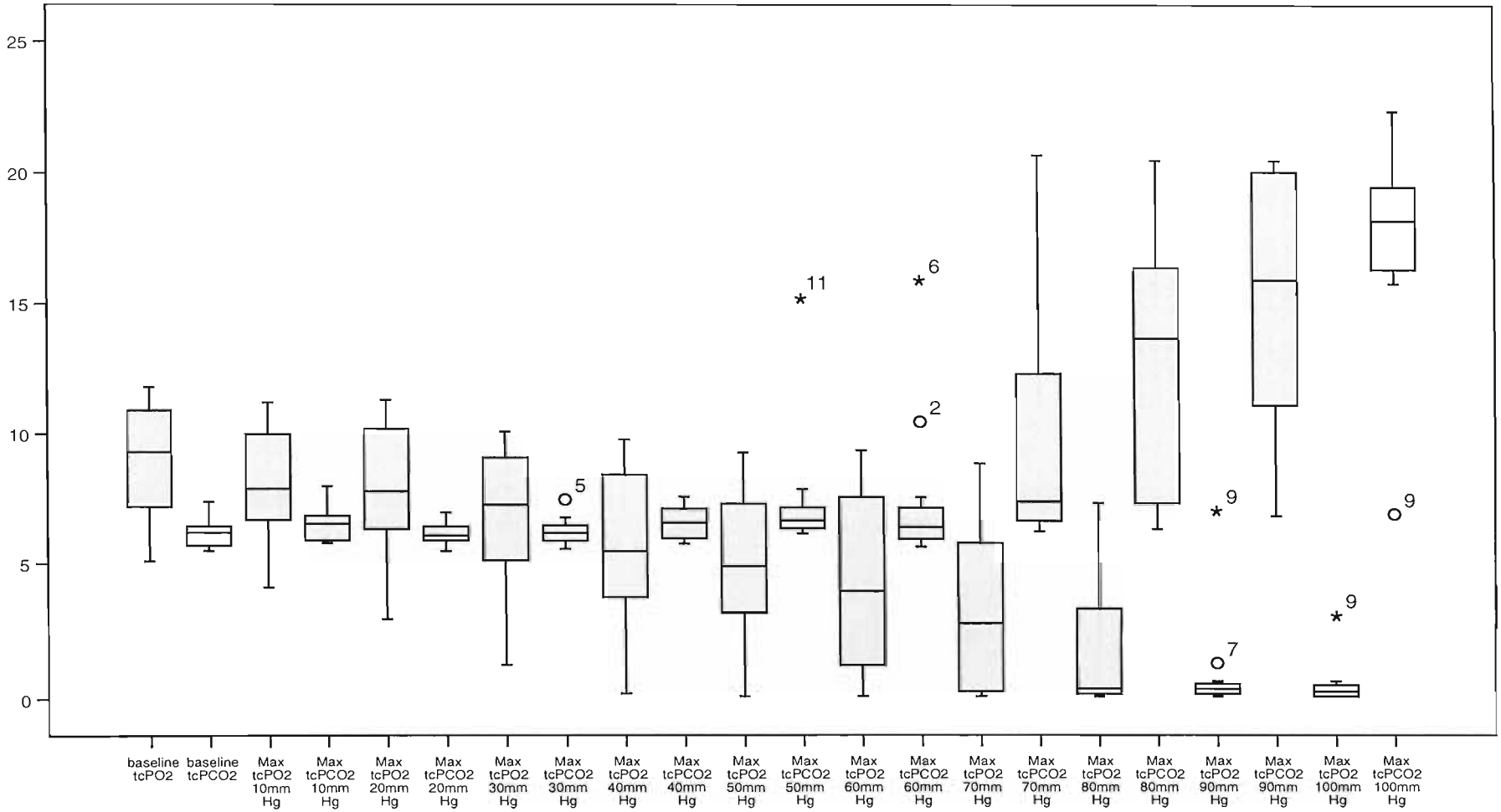



**Figure 4.33 Box and Whisker plot of maximum level of $tcPO_2$ and $tcPCO_2$ achieved when sacral tissue exposed to pressure increased in increments of 10mmHg, ranging from 0-100mmHg**

| | Volunteer 1 | | Volunteer 2 | | Volunteer 3 | | Volunteer 4 | | Volunteer 5 | | Volunteer 6 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Right sacral site | | Right sacral site | | Right sacral site | | Right sacral site | | Right sacral site | | Right sacral site | |
| Pressure applied (mmHg) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) |
| 10 | 4.1 | 7.1 | 8.7 | 5.9 | 6.6 | 5.8 | 6.9 | 6.7 | 7.1 | 5.8 | 9.0 | 6.1 |
| 20 | 6.8 | 5.9 | 6.5 | 6.0 | 6.2 | 5.5 | 11.3 | 6.2 | 3.0 | 6.5 | 5.1 | 5.8 |
| 30 | 4.5 | 5.9 | 5.1 | 6.1 | 6.8 | 6.3 | 7.5 | 6.5 | 1.3 | 7.5 | 9.2 | 5.6 |
| 40 | 2.9 | 5.9 | 4.6 | 7.6 | 9.8 | 5.9 | 8.7 | 7.3 | 0.2 | 7.0 | 1.0 | 6.1 |
| 50 | 5.1 | 6.3 | 8.7 | 6.4 | 4.5 | 6.9 | 8.5 | 6.5 | 2.3 | 6.4 | 0.1 | 7.5 |
| 60 | 6.6 | 5.7 | 0.1 | 10.5 | 3.8 | 6.0 | 8.6 | 6.5 | 0.1 | 15.9 | 1.8 | 6.0 |
| 70 | 4.3 | 6.3 | 1.4 | 7.4 | 0.2 | 8.6 | 8.9 | 6.6 | 0.2 | 18.1 | 0.1 | 8.7 |
| 80 | 4.8 | 6.4 | 0.1 | 20.5 | 0.2 | 17.0 | 0.2 | 13.6 | 0.2 | 15.2 | 0.2 | 10.2 |
| 90 | 0.7 | 7.4 | 0.2 | 20.5 | 0.1 | 16.6 | 0.2 | 13.6 | 0.1 | 16.2 | 0.2 | 15.66 |
| 100 | 0.1 | 19.6 | 0.1 | 18.7 | 0.1 | 16.4 | 0.1 | 15.8 | 0.2 | 16.9 | 0.1 | 18.0 |
| | **Volunteer 1** | | **Volunteer 2** | | **Volunteer 3** | | **Volunteer 4** | | **Volunteer 5** | | **Volunteer 6** | |
| | Left sacral site | | Left sacral site | | Left sacral site | | Left sacral site | | Left sacral site | | Left sacral site | |
| Pressure applied (mmHg) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) | Maximum tcPO2 (kPa) | Maximum tcPCO2 (kPa) |
| 10 | 5.9 | 7.4 | 9.6 | 6.6 | 10.4 | 6.9 | 6.8 | 6.8 | 11.2 | 5.9 | 10.8 | 6.5 |
| 20 | 8.4 | 5.9 | 9.9 | 6.2 | 11.0 | 7.0 | 10.5 | 6.4 | 7.2 | 6.5 | 8.5 | 6.0 |
| 30 | 7.5 | 5.9 | 9.0 | 6.3 | 10.1 | 5.7 | 7.1 | 6.5 | 5.2 | 6.8 | 9.4 | 9.4 |
| 40 | 5.4 | 7.4 | 8.2 | 6.9 | 9.2 | 5.8 | 5.1 | 6.8 | 5.6 | 6.3 | 7.6 | 6.4 |
| 50 | 6.2 | 6.9 | 5.5 | 6.4 | 9.3 | 6.2 | 4.8 | 6.9 | 0.4 | 15.2 | 4.1 | 7.9 |
| 60 | 3.6 | 6.8 | 0.8 | 7.6 | 9.0 | 5.9 | 6.4 | 6.6 | 0.5 | 10.9 | 4.3 | 6.8 |
| 70 | 6.8 | 6.9 | 0.7 | 20.7 | 5.4 | 6.5 | 6.3 | 7.5 | 0.4 | 16.0 | 4.6 | 6.8 |
| 80 | 4.2 | 7.1 | 0.7 | 19.7 | 7.4 | 6.9 | 0.4 | 13.8 | 0.4 | 15.8 | 2.5 | 7.7 |
| 90 | 1.4 | 11.8 | 0.5 | 20.4 | 7.1 | 6.9 | 0.4 | 10.5 | 0.5 | 19.8 | 0.4 | 20.33 |
| 100 | 0.7 | 19.4 | 0.0 | 22.4 | 3.1 | 7.0 | 0.4 | 18.4 | 0.5 | 22.1 | 0.4 | 16.3 |

Indicates $tcPCO_2$ ≤7.4 kPa
Indicates $tcPCO_2$ > 7.4 - 12 kPa
Indicates $tcPCO_2$ >12kpa
Indicates $tcPO_2$ < 1kPa
Indicates $tcPO_2$ ≥ 1kPa to <5.0 kPa
Indicates $tcPO_2$ ≥5 kPa

**Table 4.13 Range of Sacral Tissue $tcPO_2$ and $tcPCO_2$ Responses for Each Sacral Site when Subjected to Pressures from 10 to 100mmHg**

In order to represent the differences in responses to pressure observed between individuals and individual's sacral site, Table 4.13 identifies the maximum level of $tcPO_2$ and $tcPCO_2$ achieved under pressure, for each sacral site at each pressure from 10 to 100mmHg. The chart is colour coded to reflect the range of $tcPO_2$ and $tcPCO_2$ levels identified in each type of response identified in section 4.2.6, with the maximum level of $tcPO_2$ and $tcPCO_2$ achieved used as the indicator to define the response type. Eleven of the twelve sites changed from maintaining levels of $tcPO_2$ and $tcPCO_2$ within the normal range at the lower levels of pressure, to significant increases in $tcPCO_2$ levels and corresponding falls in $tcPO_2$ by the time 100mmHg is applied. The one exception was volunteer 3's left sacral site, which maintained $tcPCO_2$ levels within the identified normal range for the full range of pressures. The table closely resembles that of Table 4.11 which identified the nature of the response of each sacral site to the range of pressures.

Six out of twelve trends identified within Table 4.13 changed from green to red without fluxuations between response types, representing gradual loss of oxygen and accumulation carbon dioxide as the pressure increases. The fluxuations seen between the colours however is always between the green and orange, or orange and green, not green and red. Table 4.13 also identifies that the critical range of pressure between the green and red response was within 9 mmHg for five sacral sites, within 19mmHg for five sacral sites and 29mmHg for one sacral site. The fluxuations between green and orange and orange and green require further examination and will be explored in the discussion.

The limitation of examining the maximum level of $tcPO_2$ and $tcPCO_2$ achieved under pressure is that the figure it not necessarily representative of the final level of $tcPO_2$ and $tcPCO_2$ within the local tissue, but the peak measurement achieved during the application of pressure. This could misrepresent the degree of overall recovery or intolerance of the sacral tissue to pressure. The area under the curve has been examined to identify the total loss of oxygen and total gain in carbon dioxide within the tissue as a result of the application of pressure. The fluxuations in levels of $tcPO_2$ and $tcPCO_2$ in response to pressure has been explored further in order to identify whether corresponding fluxuations in capillary blood flow are occurring. This is presented in section 4.5

#### 4.4.2.5 Effect of Different Levels of Pressure on Total Loss and Total Gain of $tcPO_2$ and $tcPCO_2$ in Sacral Tissue, Demonstrated Through Calculating Area Under the Curve

To address the possible limitations of measuring the nature of the response of sacral tissue to pressure by identifying the peak level of $tcPO_2$ and $tcPCO_2$ achieved under pressure the area under the curve was calculated. The area under the curve represents the total level of oxygen remaining in local tissue and total accumulation of carbon dioxide during the period that the sacral tissue was exposed to pressure.

For $tcPO_2$ and $tcPCO_2$ the area under the curve was calculated from 2 minutes after the pressure required was achieved, to the point that the pressure was released. This resulted in a time period of 13minutes for each sacral site. The first two minutes were excluded as this included the transient change of the initial response to pressure. The transient change of the initial response of $tcPO_2$ to pressure was excluded because the volunteers' normal level of $tcPO_2$ prior to pressure being applied ranged from 5.1 to 11.8 kPa. Therefore if the initial two minute decline had been included the maximum level of $tcPO_2$ identified during the period of pressure would have been artificially high and could inappropriately skew the area under the curve calculations. This could incorrectly indicate a greater tolerance to the pressure than had actually occurred.

The area under the curve (AUC) was calculated using the following equation:

$$AUC=\sum_{T1}^{T2} x$$

T equals time and indicates the start and end point of the period identified for the area under the curve to be calculated. For this study readings were taken every second from the transcutaneous electrodes and therefore $x$ is the value of $tcPO_2$ or $tcPCO_2$ recorded at each second.

The normal range for $tcPO_2$ and $tcPCO_2$ in terms of area under the curve is calculated by multiplying the normal range limits by the time that the sacral tissue was exposed to pressure (780seconds). With an upper limit of the normal range for $tcPCO_2$ having already been identified 7.4kPa the upper level for the area under the curve is 5772kPa/seconds.

The lower limit of $tcPO_2$ of 5.1kPa for the lower limit of the normal range results in an area under the curve of 3978 kPa/second. In Figure 4.33 the AUC for $tcPO_2$ is plotted against that of $tcPCO_2$ in order to explore the relationship between the two.

**Table 4.14 Frequency Distribution Data for the Range of AUC in Response to Pressure Ranging from 10 to 100 mmHg**

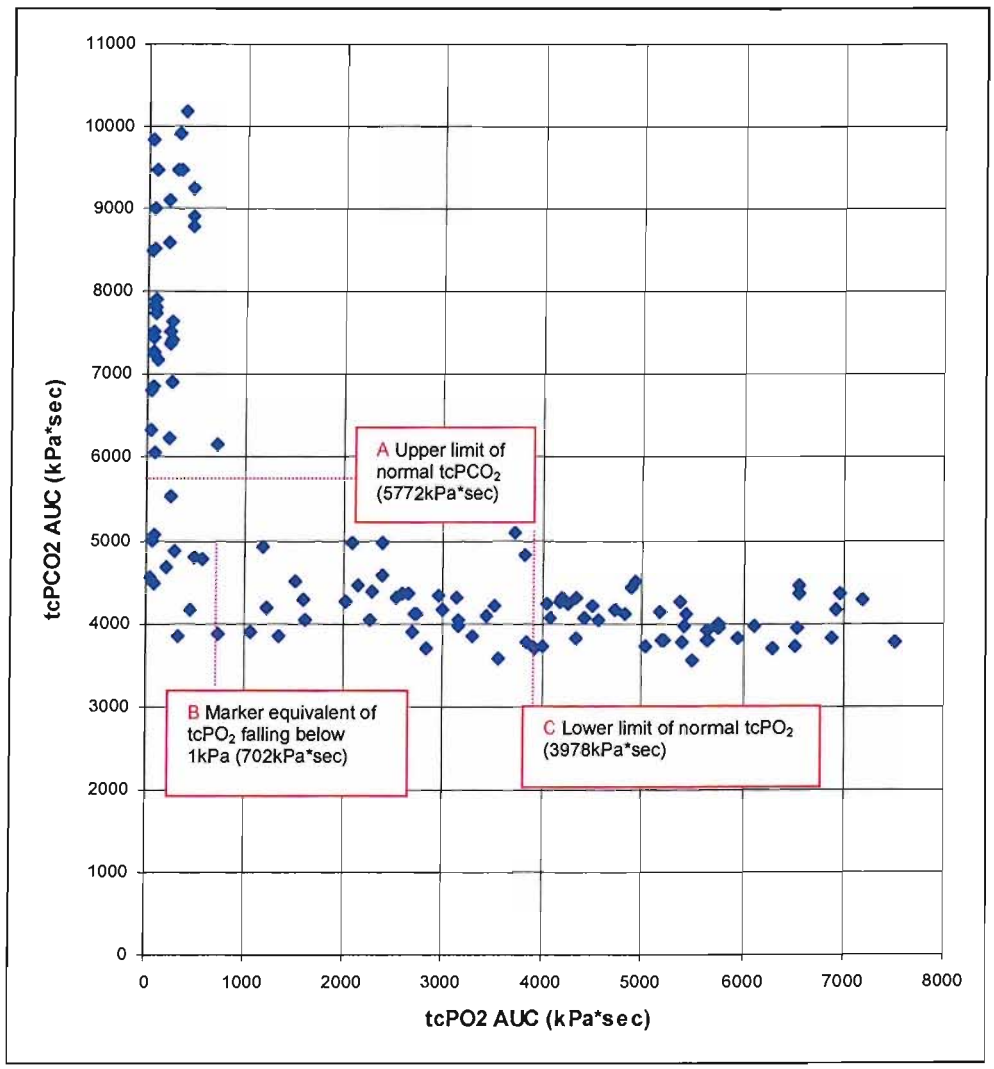
| | | Area Under the Curve $tcPO_2$ (kPa/sec) | Area Under the Curve $tcPCO_2$ (kPa/sec) |
|---|---|---|---|
| Median | | 2454 | 4327 |
| Skewness | | 0.37 | 1.2 |
| Std. Error of Skewness | | 0.22 | 0.22 |
| Minimum | | 44 | 3551 |
| Maximum | | 7511 | 10169 |
| Percentiles | 25 | 247 | 3988 |
| | 50 | 2454 | 4327 |
| | 75 | 4540 | 6820 |

As the data is non parametric the correlation between AUC for $tcPO_2$ and $tcPCO_2$ in response to pressure was calculated using the Spearman's rank correlation and found to be -0.75 significant at the 0.01 level (2-tailed). The results of each sacral site's response to the range of pressures are tabulated in Table 4.15 and colour coded according the category as identified in Figure 4.34.

The merits of using the type of response, maximum level of $tcPO_2$ and $tcPCO_2$ achieved under pressure, and area under the curve to describe the nature of response and degree of tissue tolerance to pressure will be explored further in the discussion, Chapter 5.

**Figure 4.34 Relationship between changes in AUC for $tcPO_2$ and $tcPCO_2$ when sacral tissue exposed to pressures ranging from 10 to 100mmHg**

In the diagram marker A represents the upper limit of normal $tcPCO_2$; marker B is a marker representing the equivalent of 1kPa for $tcPO_2$; and marker C is the lower limit for normal $tcPO_2$



## Table 4.15 Identifying the Changes in AUC for $tcPO_2$ and $tcPCO_2$ in Response to Pressures from 10 to 100 mmHg

| | Volunteer 1 | | Volunteer 2 | | Volunteer 3 | | Volunteer 4 | | Volunteer 5 | | Volunteer 6 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Right sacral site | | Right sacral site | | Right sacral site | | Right sacral site | | Right sacral site | | Right sacral site | |
| Pressure applied (mmHg) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) |
| 10 | 2383.0 | 4571.0 | 5450.0 | 4104.0 | 3846.0 | 3779.0 | 4281.0 | 4230.0 | 4003.0 | 3715.0 | 5654.0 | 3916.0 |
| 20 | 4362.0 | 3819.0 | 3301.0 | 3851.0 | 3567.0 | 3578.0 | 6887.0 | 3822.0 | 1227.0 | 4186.0 | 2828.0 | 3700.0 |
| 30 | 4356.0 | 3817.0 | 2687.0 | 3902.0 | 4097.0 | 4076.0 | 4528.0 | 4220.0 | [illegible] | 4865.0 | 5507.0 | 3551.0 |
| 40 | 1334.0 | 3852.0 | 2071.0 | 4954.0 | 5660.0 | 3786.0 | 4940.0 | 4493.0 | [illegible] | 4481 0 | [illegible] | 3836.0 |
| 50 | 3156.0 | 4036.0 | 5189.0 | 4130.0 | 2020.0 | 4248.0 | 4846.0 | 4120.0 | [illegible] | 4164.0 | [illegible] | 4553.0 |
| 60 | 3925.0 | 3702.0 | [illegible] | 6321.0 | 1054.0 | 3886.0 | 4748.0 | 4169.0 | [illegible] | 7258.0 | 723.0 | 3869.0 |
| 70 | 2263.0 | 4034.0 | 565.0 | 4779.0 | [illegible] | 5071.0 | 5386.0 | 4257.0 | [illegible] | 7841.0 | [illegible] | 4999.0 |
| 80 | 2735.0 | 4102.0 | [illegible] | 9830.0 | [illegible] | 7807.0 | [illegible] | 6868.0 | [illegible] | 7178.0 | [illegible] | 6061.0 |
| 90 | [illegible] | 4675.0 | [illegible] | 9466.0 | [illegible] | 7901.0 | [illegible] | 6804.0 | [illegible] | 7840.0 | [illegible] | 7278.00 |
| 100 | [illegible] | 9005.0 | [illegible] | 8488.0 | [illegible] | 7528.0 | [illegible] | 7454.0 | [illegible] | 7730.0 | [illegible] | 8528.0 |
| | Volunteer 1 | | Volunteer 2 | | Volunteer 3 | | Volunteer 4 | | Volunteer 5 | | Volunteer 6 | |
| | Left sacral site | | Left sacral site | | Left sacral site | | Left sacral site | | Left sacral site | | Left sacral site | |
| Pressure applied (mmHg) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) | AUC tcPO2 (kPa*sec) | AUC tcPCO2 (kPa*sec) |
| 10 | 3725.0 | 5096.0 | 6563.0 | 4354.0 | 6547.0 | 4463.0 | 7190.0 | 4278.0 | 7511.0 | 3760.0 | 6927.0 | 4151.0 |
| 20 | 5224.0 | 3790.0 | 6108.0 | 3974.0 | 6965.0 | 4349.0 | 6530.0 | 3949.0 | 4430.0 | 4076.0 | 5416.0 | 3766.0 |
| 30 | 5219.0 | 3787.0 | 5751.0 | 3993.0 | 6290.0 | 3696.0 | 4197.0 | 4259.0 | 2584.0 | 4366.0 | 5936.0 | 3809.0 |
| 40 | 3125.0 | 4303.0 | 4909.0 | 4430.0 | 5048.0 | 3716.0 | 2953.0 | 4341.0 | 3153.0 | 3974.0 | 4578.0 | 4034.0 |
| 50 | 3445.0 | 4094.0 | 2712.0 | 4107.0 | 5760.0 | 3944.0 | 2637.0 | 4350.0 | [illegible] | 7374.0 | 2381.0 | 4975.0 |
| 60 | 1585.0 | 4282.0 | [illegible] | 4802.0 | 6512.0 | 3725.0 | 3513.0 | 4210.0 | [illegible] | 5526.0 | 2287.0 | 4369.0 |
| 70 | 4046.0 | 4232.0 | [illegible] | 8776.0 | 2998.0 | 4154.0 | 3836.0 | 4830.0 | [illegible] | 6904.0 | 2526.0 | 4300.0 |
| 80 | 2132.0 | 4448.0 | [illegible] | 9239.0 | 4216.0 | 4314.0 | [illegible] | 7524.0 | [illegible] | 7417.0 | 1178.0 | 4907.0 |
| 90 | 721.0 | 6147.0 | [illegible] | 9468.0 | 4361.0 | 4311.0 | [illegible] | 6224.0 | [illegible] | 9472.0 | [illegible] | 9091.00 |
| 100 | [illegible] | 8903.0 | [illegible] | 10169.0 | 1499.0 | 4367.0 | [illegible] | 8588.0 | [illegible] | 9917.0 | [illegible] | 7636.0 |

indicates tcPCO2 >5772<8250

indicates tcPCO2 >8250

indicates tcPO2 <702

indicates tcPCO2 levels remain within normal range <5772

indicates tcPO2 levels ≥702 <3978

## 4.5 Relationship Between Changes in Sacral $tcPO_2$ and $tcPCO_2$ and Local Changes in Blood Flow in Response to Pressures Ranging from 0 to 100 mmHg

Changes observed in sacral tissue $tcPO_2$ and $tcPCO_2$ levels, in response to pressure, are thought to be associated with changes in local capillary blood flow. It is postulated that the reduction in $tcPO_2$ and steady increase in $tcPCO_2$ in response to pressure, is a reflection of the degree to which local capillaries have been compressed or distorted sufficiently to compromise and occlude local capillary blood flow. Laser Doppler Fluximetry (LDF) is a non-invasive, real-time measurement of blood flow suited to measuring relative flow changes in microvasculature to a depth of approximately 1mm from the epidermis (Braverman et al, 1990). The output from LDF is expressed as arbitrary units (AU) as it represents the level of change rather than actual flow values. By simultaneously measuring changes in $tcPO_2$, $tcPCO_2$ levels and LDF flux in response to the application of different levels of pressure to the sacral tissue the association between the two changes can be examined further to understand the physiological response to pressure.

### 4.5.1 Method

The method described in section 4.4, used to examine the critical nature of pressure applied to sacral tissue, was utilised for this part of the study. The same six volunteers participated, and the same range of pressures was applied, namely 10 to 100mmHg in increments of 10mmHg. Therefore each volunteer had ten data sets measured.

The only difference in methodology was that a Moor Instruments DRT4 laser Doppler monitor was used with two DP1T-V2 skin probes, see Figure 4.34. The probes were calibrated according to the manufacturer's instructions, and probe one always used for measurements of the right sacral site and probe 2 for the left. This is because each probe has slightly different sensitivity and hence output, therefore using the same probe for each site minimises potential differences in flux due to inter-probe variations. The point of application of the laser Doppler probe was marked to ensure consistency in it's placement over the ten separate measurement sessions.

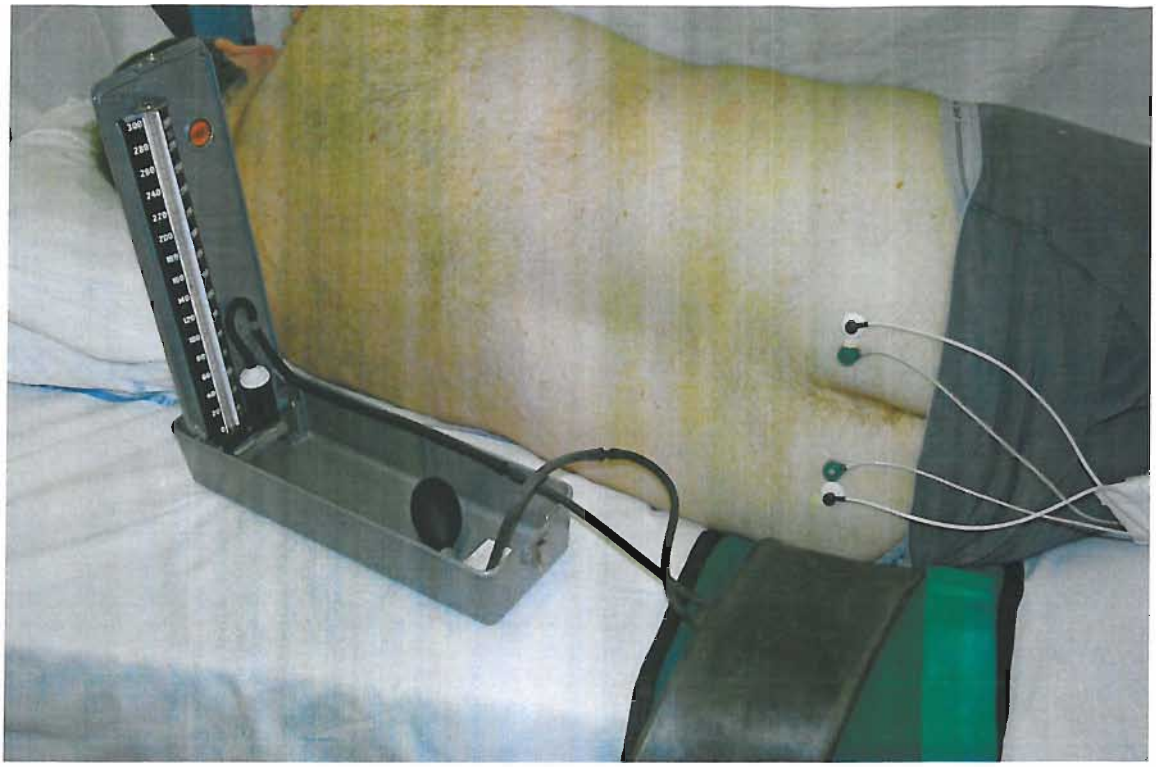


**Figure 4.35 Laser Doppler skin probes in situ adjacent to transcutaneous electrodes**

The laser Doppler probes are very sensitive to movement and therefore it was noted when volunteers moved their legs inadvertently, coughed or sneezed, in order that false responses could be extrapolated from the results.

The parameters measured and analysed were:

- mean baseline flux measured over a ten minute period prior to the application of pressure;
- mean flux measured over a ten minute period during the application of pressure. The first two minutes response following the initial application of pressure was excluded to ensure stabilisation of the readings. This value was then used to calculate the percentage change in flux;
- the percentage change in flux was calculated from the difference between the baseline flux and flux measured when sacral tissue is subjected to pressure;
- the percentage change in flux was then compared with maximum $tcPO_2$ and max $tcPCO_2$ level achieved when the sacral tissue was exposed to pressures ranging from 10 to 100 mmHg.

## 4.5.2 Results

### 4.5.2.1 Demographic Details

As volunteer group was the same as for previous experiments, for demographic details refer to section 4.4.1, Table 4.10.

### 4.5.2.2 Changes in Sacral Tissue Flux as a Consequence of Pressure.

All six volunteers had ten separate sets of readings taken in order that the effect of the full range of pressures applied could be observed. Therefore the baseline mean flux was calculated from all 60 sets of data, the results of which are shown in Table 4.16.

As the skewness (identified in Table 4.16) was more than double the standard error of skewness, the degree to which the data is skewed was considered significant. Therefore non-parametric tests were used for the analysis of these data. The difference between the left and right mean flux was found to be 13.2 AU, but interestingly the 25% quartile were very close with the left a mean flux of 19.4 AU for the right sacral site and 19.5 AU for the left.

**Table 4.16 Summary of the Frequency Statistics for the Mean Baseline Flux Readings for Left and Right Sacral Sites**

| (n=60) | | Mean Baseline Flux right sacral site (AU) | Mean Baseline Flux left sacral site (AU) |
|---|---|---|---|
| Mean | | 39.1 | 28.0 |
| Std. Error of Mean | | 3.3 | 1.6 |
| Median | | 37.1 | 23.9 |
| Std. Deviation | | 25.4 | 12.6 |
| Skewness | | 1.5 | 1.1 |
| Std. Error of Skewness | | .3 | .3 |
| Range | | 115.0 | 56.6 |
| Minimum | | 9.8 | 11.5 |
| Maximum | | 124.8 | 68.1 |
| Percentiles | 25 | 19.4 | 19.5 |
| | 50 | 37.1 | 23.9 |
| | 75 | 49.4 | 33.6 |

**Table 4.17 Summary of the Frequency Statistics for Mean Percentage Change in LDF Flux Readings for Sacral Sites when Subjected to Pressures of 10 to 100 mmHg**

| | | 10mmHg | 20mmHg | 30mmHg | 40mmHg | 50mmg | 60mmHg | 70mmHg | 80mmHg | 90mmHg | 100mmHg |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Mean | | 33.5 | 26.1 | 12.7 | -2.7 | 14.5 | -22.5 | 3.4 | -18.6 | -65.8 | -53.0 |
| Median | | 45.7 | 16.0 | 2.6 | -14.5 | 5.0 | -31.2 | -16.2 | -12.4 | -82.6 | -68.7 |
| Minimum | | -27.9 | -13.3 | -42.5 | -46.5 | -57.8 | -88.4 | -83.9 | -90.6 | -94.1 | -91.3 |
| Maximum | | 91.9 | 119.0 | 87.2 | 70.0 | 84.7 | 45.1 | 188.7 | 73.6 | 14.5 | 8.0 |
| Percentile | 25 | -4.4 | 9.4 | -21.5 | -25.7 | -19.5 | -53.1 | -54.7 | -83.4 | -92.2 | -87.1 |
| | 50 | 45.7 | 16.0 | 2.6 | -14.5 | 5.0 | -31.2 | -16.2 | -12.4 | -82.6 | -68.7 |
| | 75 | 57.1 | 42.2 | 54.3 | 16.8 | 57.7 | 18.3 | 35.7 | 35.1 | -51.2 | -10.0 |

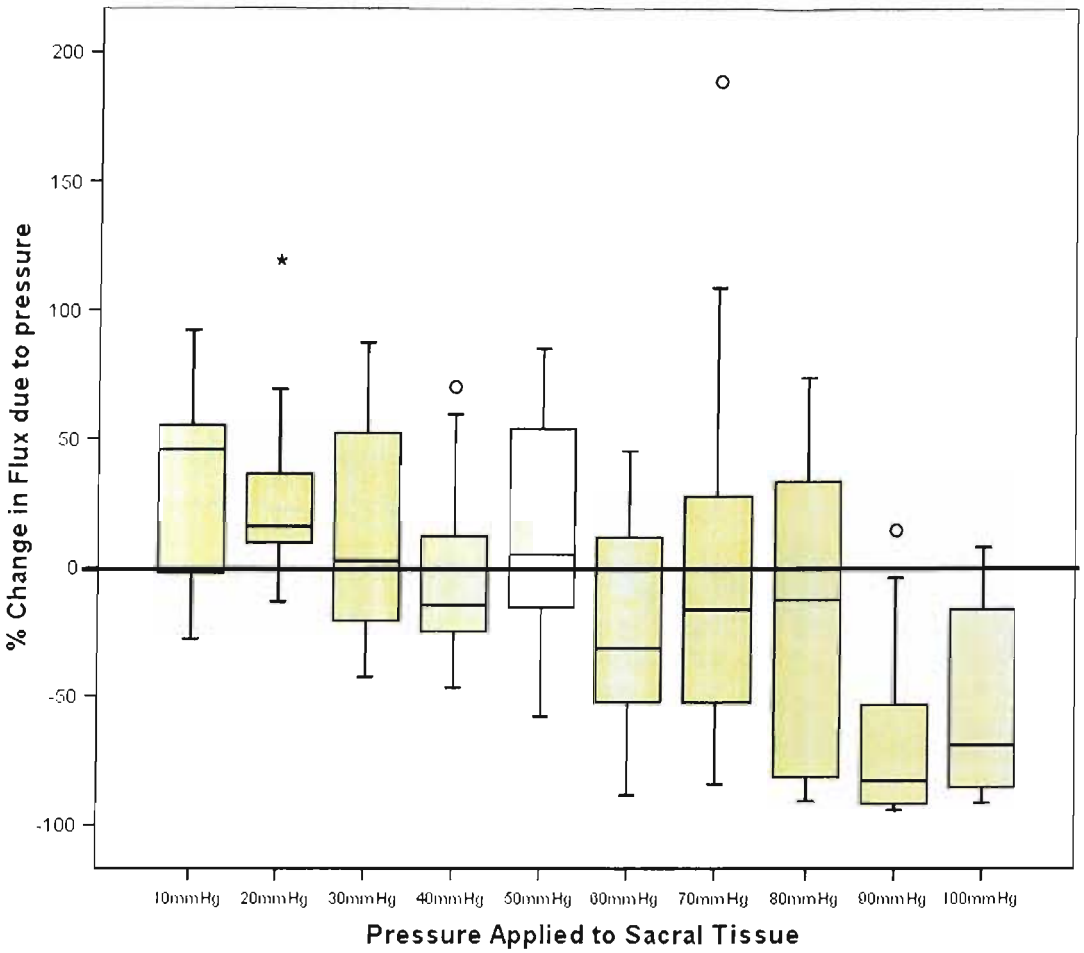



**Figure 4.36 Medians and inter-quartile ranges for mean percentage change in flux as pressure applied is increased from 10 to 100 mmHg**

Figure 4.36 illustrates the effect of pressure on the mean percentage change in flux for sacral tissue and shows a gradual overall reduction in percentage change flux as the pressure is increased. A full summary for each pressure is presented in Table 4.17. The fluxuations in velocity between 50 to 80mmHg are due to an increase in velocity of blood flow due to partial occlusion of the capillary due to pressure.

The significance of the differences between the percentage change in flux from 10 to 100mmHg was examined using the Wilcoxan signed rank test. The difference reached a point of significance when sacral tissue was subjected to 40mmHg (P = 0.03). The significance continued to increase with an increase in pressure, with the difference between percentage change in mean flux having a P value of 0.002 at 100mmHg, which is strongly significant. The P values for all pressures are presented in Table 4.18, and of note the significance of change at 70mmg plummets to 15% (P=0.15).

**Table 4.18 Significance of the Difference in Percentage Change of LDF Flux Between 10 mmHg and Incremental Increases in Pressure Up to and Including 100 mmHg**

| Pressure applied to sacral sites (mmHg) | Significance of difference in percentage change in flux between 10mmHg and next level of pressure applied (P value) (n-12) |
|---|---|
| 20 | 0.4 |
| 30 | 0.4 |
| 40 | 0.03 |
| 50 | 0.08 |
| 60 | 0.005 |
| 70 | 0.158 |
| 80 | 0.008 |
| 90 | 0.003 |
| 100 | 0.002 |

### 4.5.2.3 Comparison of Measurements Recorded Simultaneously of Sacral Tissue's Percentage Change in LDF Due to Pressure and Maximum Levels of $tcPO_2$ and $tcPCO_2$ Achieved

The baseline and maximum levels of $tcPO_2$ and $tcPCO_2$ when sacral sites were subjected to pressure ranging from 10 to 100mmHg are presented in section 4.4.2.4, Table 4.12 and Figure 4.33. In order to examine the relationship between local capillary blood flow and changes in $tcPO_2$ and $cPCO_2$ the data recorded simultaneously for percentage flux change in Arbitrary Units (AU) and maximum $tcPO_2$ levels achieved have be plotted against each other, and the same for $tcPCO_2$ and are illustrated in figures 4.37 and 4.38 respectively. Using the Spearman's rank correlation the correlation was identified as $r = 0.58$ (significant at the 0.01 level) and $r = -0.56$ (significant at he 0.01 level) respectively, demonstrating a significant correlation between changes observed in $tcPO_2$. $tcPCO_2$ and blood flux, used to represent flow.

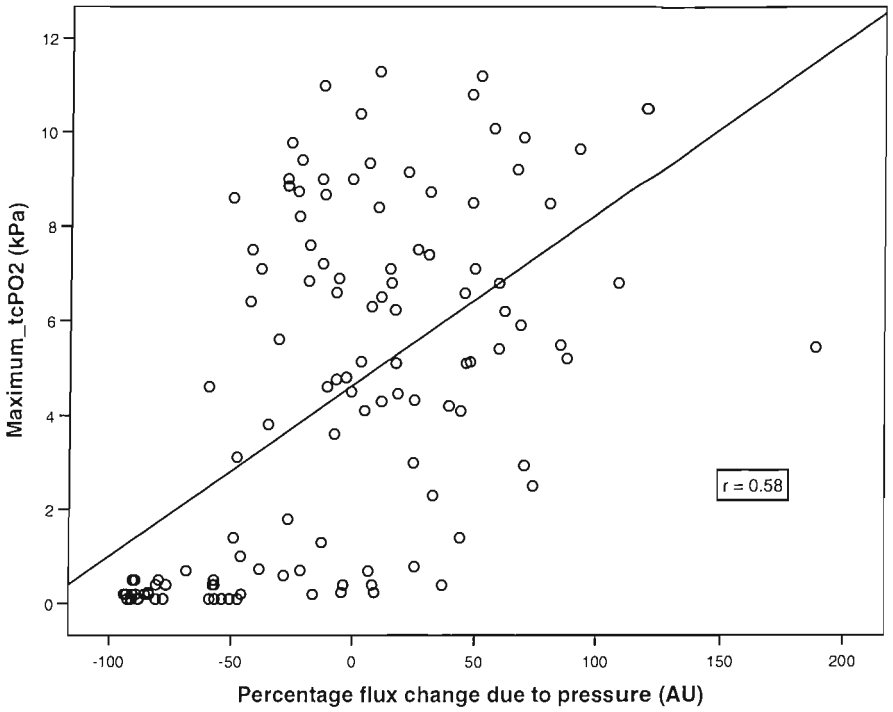



**Figure 4.37** **Percentage change in LDF flux in arbitrary units (AUC) plotted against the maximum level of $tcPO_2$ achieved in sacral tissue when simultaneously subjected to the same pressures**

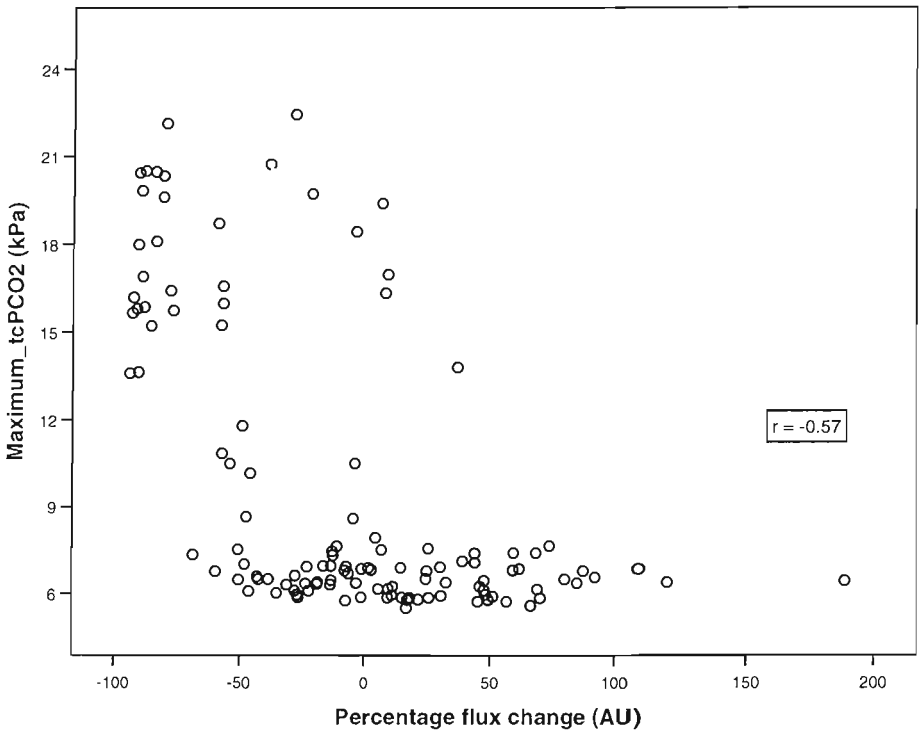


**Figure 4.38 Percentage change in LDF flux in arbitrary units (AU) plotted against the maximum level of $tcPCO_2$ achieved in sacral tissue when simultaneously subjected to the same pressures**

### 4.5.3 Summary

The percentage change in LDF flux shows a significant reduction in local capillary blood flow as the level of pressure sacral tissue is subjected to increases. When the relationship between changes in blood flux and maximum levels of $tcPO_2$ and $tcPCO_2$ achieved when sacral tissue was subjected to pressures were examined a significant correlation was identified. This supports the theory that deterioration in $tcPO_2$ levels with increasing pressures and increasing levels of $tcPCO_2$ are associated with changes in local capillary blood flow.

The relationship identified between $tcPCO_2$ and percentage change in blood flux in response to pressure (illustrated in Figure 4.36) is similar to the correlation identified between maximum levels of $tcPO_2$ and maximum levels of $tcPCO_2$. This supports the significance of the association identified between percentage change in flux and maximum $tcPO_2$ when subjected to pressure, and supports the theory that increases in $tcPCO_2$ are predominantly observed following sufficient capillary closure as a consequence of the pressure.

Careful placement of the bladder used to apply the pressure was required to ensure total coverage of the $tcPO_2$ and $tcPCO_2$ electrodes and two LDF probes. For future work a wider bladder and remodelled belt to incorporate two tightening straps, is required. This is because as the higher pressures were applied the smaller the surface area of sacral contact. This could account for some of the outliers illustrated in Figure 4.34. In addition the angle of the LDF probe may have been influenced by the application of pressure and may again help explain some of the outliers of the over all trend.

# Chapter 5 Discussion and Way Forward

## 5.1 Introduction

Tissue damage caused by pressure is not a new phenomenon. Pressure ulcers were identified in Egyptian mummies and remain a common and concerning issue today, with prevalence's of 18.1% being identified within the UK (Clark, 2002).

Pressure ulcers have a tremendous impact for both the patient and National Health Service. Patients suffer additional pain, reduced quality of life, extended length of stay and increased morbidity and mortality with pressure ulcer development (Franks et al., 1999; Allman et al, 1986 & 1995; Hibbs, 1988). The cost to the National Health Service for the treatment of pressure ulcers has been identified as 4% of the Health Service Budget (Bennet et al, 2004). Therefore the prevention of pressure ulcers is an important area of care to be addressed within the National Health Service (Essence of Care Promoting Health (2006); National Health Service, National Institute for Health and Clinical Excellence (2005)).

In order to prevent the development of pressure ulcers the level of risk of individuals to developing tissue damage as a consequence of pressure needs to be identified. As discussed in Chapter 1 many risk assessment tools have evolved since the work of Norton et al (1962), but they remain more subjective tools than objective with poor inter-rator reliability. Any improved measure of risk should be both sensitive and specific.

Vagueness of the factors used to determine risk permits broad interpretation by the user and so, regardless of the sophistication of the assessment tool, no scale could be more than an indicator (Norton, 1989; Carlton, 1990; Flanagan, 1993; Royal College Nursing 2001; National Institute for Health and Clinical Excellence, 2005). As such clinical judgement has been considered to be as good as if not better than risk assessment tools (Gould et al, 2004). It is possible that the use of clinical judgement and risk assessment tools increase the risk of pressure ulcers developing due to the subjectivity and poor inter-rator reliability of the assessment tools used and, especially if insufficient time, skills and experience are available clinically. It is likely that the assessment tools used are only as good as the person using them. There is therefore a requirement for an objective assessment tool, based on physiological measurements, to identify the tolerance of sacral tissue to pressure.

To overcome the difficulties with the subjectivity of current assessment tools researchers have been investigating the use of transcutaneous monitoring of oxygen ($tcPO_2$) as an indicator of tissue viability. The use of $tcPO_2$ monitoring to assess the success of replanted limb parts (Matsen et al, 1980), and to identify the wound healing potential for above or below knee amputations (Pinzur et al, 1992) has also been investigated.

The variability of $tcPO_2$ levels between anatomical sites, and hard and soft tissues has been investigated (Dowd et al (1983), Rodrigues et al (2001), Seiler & Stahelin (1979)). The response of $tcPO_2$ in local tissue to the application of constant and intermittent pressure has been studied and found that the level of pressure required to reduce $tcPO_2$ levels and the pattern of response varies considerably between individuals (Bader and Gant, 1988; Bader, 1990a; Knight et al, 2001), indicating the varying degrees of tolerance that individuals have to pressure. However, to date, the pressures identified within the studies were believed to be representative of the physiological range for interface pressures rather than understanding how individuals' sacral tissue responds when subjected to pressure exerted through their own body mass.

As the primary cause of pressure ulcer development is due to the pressure being sufficient to cause capillary occlusion and tissue death due to ischaemia, it is noticeable that only more recent studies have included the examination of changes in local levels of $tcPCO_2$ in response to pressure (Bogie et al ,1995; Bader, 1998; Knight et al 2001). The relationship between oxygen ($O_2$) and carbon dioxide ($CO_2$) is intrinsically linked through cell metabolism. When the blood supply to either an organ or tissue is occluded ischaemia presents as a dual phenomenon with tissue hypoxia always being accompanied by hypercapnia (Johnson & Weid, 1991; Tonnessen, 1997; Hotter et al, 2004). Studies with renal tubular cells showed that signs of apoptosis were only exhibited when hypoxia and hypercapnia were present jointly. Hypoxia or hypercapnia alone was not sufficient to cause cell death (Hotter el al, 2004).

In effect the oxygen is utilised for cell metabolism and a metabolite is $CO_2$. Sufficient blood flow for clearance of $CO_2$ is essential otherwise the accumulating $CO_2$ quickly dissolves to form carbolic acid, elevating the local pH. $CO_2$ also results from the bicarbonate buffering of anaerobic acid generation. Therefore for this study it was postulated that the measurement of carbon dioxide may be a stronger indicator of the risk of cell death as levels continue to accumulate during anaerobic metabolism. This is supported by the fact that once blood flow has sufficiently inhibited the supply of $O_2$ those levels remain unchanged until blood flow is reinstated. In contrast, when blood flow is inhibited the levels

of $CO_2$ do not remain constant and so would suggest that this would provide a more significant indicator of risk of tissue ischaemia occurring.

Further, previous hypoxic models have submitted renal cells to hypoxia alone and found $CO_2$ levels maintained at the normal level (Hotter et al 2004). When hypercapnia was superimposed to hypoxia in a renal tubular epithelial LLC-PK1 cell culture model, the appearance of apoptotic (programmed cell death) cell features were induced suggesting that $CO_2$ levels play a significant role in determining the mechanisms of cell death associated with ischaemia.

Hence, the hypothesis tested through this study is that pressures exerted by an individuals' body mass on sacral tissue in the supine position are close to a critical perfusion threshold, and because the capillary blood flow is influenced by interface pressure, when blood flow is sufficiently reduced it results in a loss of oxygen and an excess accumulation of carbon dioxide in the tissue. These parameters may be measured and used to assess individuals' tolerance to pressure exerted through their own body mass, and used as a predictor or risk indicator of pressure ulcer formation.

Experiments were designed to demonstrate the relationship between pressure, changes in $tcPO_2$ and $tcPCO_2$ and capillary blood flow, and have been described in Chapters 3 and 4.

The main findings were:

- No relationship was identified between interface pressure and body mass index;
- There was no significant difference in baseline levels of $tcPO_2$ and $tcPCO_2$ between either control or sacral sites in response to pressure;
- Four types of $tcPO_2$ responses (Type A, B, C, D) and three types of $tcPCO_2$ responses (Type A (i) to A (iii)) to pressure were identified[3] ;
- Significant differences were identified in $tcPO_2$ and $tcPCO_2$ levels between left and right sacral sites when exposed to equal pressure;
- The rapid rise of $tcPCO_2$ associated with the loss of $tcPO_2$, observed in response Type A(iii), is representative of complete capillary closure;
- The variability of critical pressure threshold at which individuals' and the individual's left and right sacral sites present each response type of $tcPO_2$ and $tcPCO_2$ .

[3] Refer to Appendix 2 for response types of $tcPO_2$ and $tcPCO_2$

## 5.2 Discussion

### 5.2.1 Relationship Between Interface Pressure and Body Mass Index

Current risk assessment tools, used to identify the risk of tissue damage as a consequence of pressure, identify body mass index as being linked to an increased risk of developing pressure ulcers. For example, the Waterlow Score (see figure 1.11, Chapter 1) allocates one point if BMI is above average, two points if patient is obese and three points if the patient is below average.

The size, shape and weight of the volunteer will vary considerably according their skeletal size and shape, quantity, shape and tone of muscle and amount of subcutaneous fat. In this study, the range of body mass index, from underweight to obese, resulted in a range of peak pressures (38-74mmHg). It was expected that the shape and size of an individual would be represented by their body mass index (BMI) and that this would be related directly to the interface pressure identified. That is, the interface pressure would be higher for obese and below average BMI.

However, from this study there was no observable association between body mass index and interface pressure, which was confirmed using the Spearman's rank correlation, identifying a very poor correlation of 0.37, with a p-value of 0.05. This supports the conclusion of Swain and Bader (2002), that it is not possible to predict the interface pressure from an individual's body type. Indeed earlier, Swain and Peters (1997) had noted that the differences must be subtler than body mass index as individuals with similar weight and height can have significantly different interface pressures. Body mass index does not indicate the distribution of weight around the body, or the quality of underlying tissues such as muscle bulk or tone, blood flow or compressive stiffness of tissues or skeletal shape (Swain and Bader, 2002).

However, these findings disagree with those of Lindan and Greenway (1965) who observed that cachexic underweight persons had higher peak pressures, whilst obese persons had greater areas of increased pressure but lower peaks. The differences in the findings may be due to the advances that have been made in the manufacturing of mattress foam, since 1965. The design and grade of foam now used for hospital mattresses has far greater ability to conform to the surface shape of individuals, thereby reducing interface pressures (Medical Devices Agency report PS1, 1993).

### 5.2.2 Baseline Measurements of Transcutaneous Partial Pressure of Oxygen and Carbon Dioxide

Many studies have examined $tcPO_2$ values for unloaded tissue, at different anatomical sites, for the same individual and different individuals. Dowd et al. (1983) identified a baseline range of transcutaneous gas tensions for oxygen as being 6 -12.9kPa, Rodrigues et al. (2001) 5.54 – 13.67kPa and Takiwaki et al. (1991) 4.79 – 11.27kPa (± 2 SD). Further, on comparing hard (tissue over a bony prominence) and soft sites (tissue over muscle) Seiler and Stahelin (1979) identified baseline $tcPO_2$ to be similar, with a range of 9.28 – 13.76kPa.

The baseline levels for $tcPO_2$ identified from this study (see Section 4.2.3, Table 4.2 and Section 4.4, Table 4.12) support these findings with a range of 5.2 to 13.3kPa and 4.5 to 11.8kPa for the sacral sites. The baseline readings for the control site (sub-clavicular) were comparable with a range of 3 to 14.6kPa.

The measurement of transcutaneous gas tensions involves the heating of the underlying skin to values in excess of normal physiological temperatures to ensure maximum vasodilatation and reliable gas detection. The recommended temperature ranges from 42-44°C and the range of temperatures used across the studies may help to explain some of the variation in the results. Knight el al. (2001) and Bader (1990) used 44.5°C, Takiwaki et al. and Seiler et al. used 44°C, and Rodrigues et al. (2001) used 43 °C. This study used 43 °C and the results most closely align with those of Rodrigues et al. (2001).

Fewer studies have included $tcPCO_2$ monitoring as part of their investigations. Takiwaki et al. (1991) and Rodrigues et al. (2001) identified a baseline range of 3.6 – 6.3kPa and 2.7 – 7.4kPa respectively for $tcPCO_2$. Again this study supports these findings with a range of 3.7 to 6.9kPa identified for the experiment examining the response of individuals' sacral tissue to their own body mass (see section 4.2, Table 4.3) and 4 to 7.4kPa for the experiment assessing the effect of a range of pressures on sacral tissue perfusion (see section 4.4,Table 4.12). No significant difference was identified between the control site and left or right sacral sites, which supports the findings of Seiler and Stahelin (1979).

Although the ranges differed slightly between the two experiments, examining the response of individuals' sacral tissue to their own body mass and assessing the effect of a range of pressures on sacral tissue perfusion, the six volunteers involved in the latter study (section 4.4) also participated in the first experiment examining individual's

responses to their own body mass (section 4.2). The baseline measurements relating to the six volunteers in section 4.4 were repeated on ten different occasions for each of the volunteers, which spanned a period of several weeks for each individual. Maximum aerobic venous $PCO_2$ is known to vary under different conditions, for example with changes in haemoglobin concentration and diet (Tonnessen, 1997). The metabolism of carbohydrate is known to consume six molecules of oxygen and releases six molecules of carbon dioxide. In contrast the metabolism of saturated fatty acids requires twenty three molecules of oxygen and produces sixteen molecules of carbon dioxide (Tortora, 1981). From section 4.4, Figure 4.31, the $tcPCO_2$ readings that correspond with $tcPO_2$ which remain within the normal range even when the tissue is exposed to pressure should perhaps be considered as forming part of the normal range that is physiologically acceptable.

The variation of baseline measurements was examined by repeating the measurements for the same individual, at the same time of day on two different days for six of the volunteers. This was to determine the reproducibility of the measurements. The mean coefficient variation for $tcPO_2$ and $tcPCO_2$ was identified to be 8.7% and 6.1% respectively (see section 4.2.3). This falls within the 10% identified by Coleman et al (1986). The difference may be linked to the site, as the measurement site used by Coleman et al (1986) was 10cm below the knee joint, and this study involved sacral tissue. The temperature of the transcutaneous electrodes also differed with the electrodes being heated to 44°C for the work undertaken by Coleman et al (1986) and 43°C for the study.

### 5.2.3 Response of Sacral Tissue to Pressure Exerted Through the Individual's Own Body Mass

The study identified four response types (A, B, C, and D) for oxygen ($tcPO_2$) as a consequence of pressure exerted through an individual's own body mass (see section 4.2.5 and figures 4.9 to 4.12). Similarly three response types (A(i), A(ii) and A(iii)) for carbon dioxide ($tcPCO_2$) were identified. The response types for A, B, C and D are illustrated in Figures 4.9 to 4.12 respectively. The response types for A(i), A(ii), and A(iii) are illustrated in Figures 4.13 to 4.15 respectively.

All of the four responses for oxygen indicated an initial rapid decrease in $tcPO_2$ levels and this is considered to be representative of the rate of metabolism. The initial application of pressure is sufficient to alter the local blood supply and destabilise the supply and demand balance for oxygen to cause the decline in $tcPO_2$. The degree of capillary closure affects the degree of initial loss of oxygen. The pattern of response of $tcPO_2$ following the initial

decline is representative of the degree to which the local tissue can compensate for and therefore tolerate the pressure applied and supports the findings of Seiler and Stahelin(1979), Bader and Gant (1988), Bader (1990a), and Knight et al. (2001).

The type D response, where $tcPO_2$ levels declined but not below 1 kPa, and demonstrate varying degrees of recovery, indicates the greatest tolerance to pressure and, it is suggested, is a consequence of the thickness, tone and mechanical integrity of the subcutaneous tissues and the proximity of the bony prominences. The partial recovery may be explained by autonomic adjustment, which is principally stimulated in response to low oxygen supplies to local tissue. The cells release vasodilatory substances such as potassium, hydrogen ions, carbon dioxide and lactic acid, which stimulate local vasodilatation (Tortora, 1981).

Once the pressure was sufficient to cause $tcPO_2$ levels to fall below 1kPa only then were all three types of $tcPCO_2$ responses observed (see table 4.12 and Figure 4.32). This has not been noted in other studies relating to sacral tissues response to pressure, and would suggest that $tcPO_2$ is sensitive to initial changes in tissue as a consequence of pressure, but once levels fall to below 1 kPa, $tcPCO_2$ becomes the stronger indicator of changes within local tissue, monitoring the severity of local tissue ischaemia.

Type A(i) response is where $tcPO_2$ levels fall below 1kPa, but $tcPCO_2$ levels remain within the normal range (Figure 4.14). This response was considered to be the point at which oxygen was still being provided to local tissues, but the level of oxygen supply was compromised sufficiently by the pressure such that the rate of consumption of oxygen by local tissue matched the rate of demand. Therefore an insufficient pressure gradient would exist for the diffusion of oxygen to the epidermis for measurement by the transcutaneous electrode. The $tcPCO_2$ levels remained within the normal range indicating that the local capillary flow was maintained sufficiently for the removal of carbon dioxide. Previously it has been identified that the mitochondrial partial pressure of oxygen required to generate high energy phosphate bonds (Adenosine triphosphate) necessary to maintain aerobic cellular biochemical functions is only 0.13 to 0.4kPa (Leach and Treacher, 1998). This may explain the finding of a Type A(i) response where the $tcPO_2$ levels have fallen below 1kPa, but with $tcPCO_2$ levels remaining within the normal range. The lack of increase in $tcPCO_2$ would suggest that the pressure applied has not been insufficient to cause total capillary closure, and the work of Leach and Treacher (1998) would suggest that sufficient $O_2$ is available to support aerobic cell metabolism.

The Type A(ii) $tcPCO_2$ response was also noted by Knight et al (2001), where $tcPCO_2$ levels steadily rose above the upper limit of the normal range to levels of up to 12kPa. The response is identifiable by the trend and shape of the response as there is an initial steady increase followed by a slight reduction in the rate of diffusion of $tcPCO_2$ resulting in the levels of $tcPCO_2$ starting to plateau (Figure 4.15). The upper limit was identified from the three clusters identified in figure 4.8. The upper level of this category, i.e., 12kPa, although referred to in Knight et al. (2001) was not explained. This level of response is thought to indicate that local capillary blood flow has been sufficiently reduced by the pressure to a point where the rate of removal of carbon dioxide does not match the rate of production resulting in an accumulation within local tissue.

Previously tissue has been defined as hypoxic when aerobic cellular biochemical function is only 0.13 – 0.4kPa (Leach and Treacher, 1998). Other studies have also interpreted a zero level of $tcPO_2$ to represent a state of anoxia and be indicative of capillary occlusion (Bader, 1990a & b; Knight et al, 2001; Seiler and Stahelin, 1979). The findings of this study suggest that total capillary closure occurs only when there is a significant and relatively rapid increase of $tcPCO_2$ above 12 kPa corresponding with $tcPO_2$ levels below 1kPa. This is the third type of $tcPCO_2$ response.

As such, the third type of response, Type A(iii), represents the severest response of local sacral tissue to pressure (Figure 4.16). The $tcPO_2$ levels fall, and remain below 1 kPa, and $tcPCO_2$ levels rose significantly resulting in levels of between 13.6 to 22.4kPa, with a median value of 18.3 kPa being measured. The most likely explanation is that the pressure applied has been sufficient to cause capillary closure. The $tcPCO_2$ levels continue to accumulate as a consequence of anaerobic cell metabolism, whereby the hydrogen ions generated as a by-product of lactic acid and the hydrolysis of adenosine tri-phosphate and adenosine 5'-diphosphate, are buffered by bicarbonate and produce carbon dioxide. This response has not been observed previously in sacral tissue, but is supported by the findings of Hotter et al. (2004) who observed changes in cortical $PCO_2$ levels calculated from tissue pH measurements when the renal artery was occluded for thirty minutes. Hotter et al (2003) found that renal vascular occlusion induced an immediate rapid rise in tissue $tcPCO_2$ reaching mean values over 15kPa at 5 minutes of ischaemia, and a mean value of 30kPa by 15minutes. However, after 15 minutes $tcPCO_2$ levels where found to plateau. This was not the finding of this study as the level of $tcPCO_2$ only ceased to increase following the removal of pressure. It was not possible to identify the maximum level of tcPCO2 as this would have required extending the length of time

that pressure was applied for thus risking sacral tissue death in the volunteers, which for ethical reasons was not an objective of this study.

The Type A(iii) response of $tcPCO_2$ observed in this study identified that the nature of the accumulation of $tcPCO_2$ followed the line of a quadratic curve, with a medial $r^2$ value of 0.86 and p value of 0.000 (see Figure 4.30).The upper $R^2$ value was 0.999 with a p value of 0.000, which is very highly significant. The $tcPCO_2$ levels only started to decline upon removal of the pressure. It is considered that the $tcPCO_2$ levels are the precursor to cell death due to the rapid change in local pH as a consequence of the accumulation of carbon dioxide, which readily dissolves to from carbolic acid. As discussed above in Section 5.1, previous in vivo studies have identified that apoptotic indicators (DNA fragmentation and significant increase in Caspase-3 activity) occur only when cells are exposed to hypoxia and hypercapnia simultaneously. Carbon dioxide levels need to exceed 18% gas atmospheres for caspase-3 activity and 30% for DNA fragmentation (Hotter et al., 2004). 1% dry gas is equivalent to 0.95kPa, therefore 18% is equivalent to 17.1kPa. This perhaps explains why tissues have different survival times when exposed to hypoxia, because different tissues have different metabolic rates and therefore different time periods are required before a critical level of carbon dioxide is achieved.

The three types of $tcPCO_2$ responses (Type A(i), A(ii) and A(iii)) observed, and relationship observed between $tcPO_2$ and $tcPCO_2$ (see Figure 4.32) are supported by the findings of Tonnessen and Kvarstein (1995), who examined changes in renal tissue $tcPCO_2$ between aerobic and anaerobic metabolism, and the mathematical modelling of ischaemic hypoxia undertaken by Gutierrez (2004). The changes in $tcPO_2$ and $tcPCO_2$ observed during this study were demonstrated as being associated with changes in local capillary blood flow following the experiment described in section 4.4 involving laser Doppler measurements to indicate changes in the velocity of local capillary blood flow. Laser Doppler readings were measured simultaneously to the readings of $tcPO_2$ and $tcPCO_2$ when the range of pressures applied, from 10 to 100mmHg, increased in increments of 10mmHg. The difference in mean flux from baseline readings compared with that observed during the application of pressure increased as the level of pressure applied increased.

The range of responses identified through this study are therefore considered to represent the varying degrees to which the pressure has compromised sacral tissue perfusion and diffusion of oxygen and carbon dioxide to and from the local capillary blood supply. The response can be described in terms of either maximum levels of $tcPO_2$ and $tcPCO_2$ achieved under pressure or the total area under the curve. The maximum level identified

for $tcPO_2$ represents the level to which local tissue managed to recover to whilst. The maximum level of $tcPCO_2$ represents the maximum level that $CO_2$ levels rose to when the tissue was subjected to pressure. Finally the total area under the curve represents the total level of $tcPO_2$ remaining in the tissue and total level of $tcPCO_2$ that has accumulated during the application of pressure. Each approach has advantages and disadvantages. Using the maximum level of $tcPO_2$ and $tcPCO_2$ recorded may not represent the overall trend as the maximum level may be achieved 5minutes into the 15 minute period during which pressure was applied, and may be followed by a further decline. Equally, if the maximum level of $tcPCO_2$ achieved is identified to be the cause or trigger for tissue necrosis then calculating the total accumulation of $tcPCO_2$ for the duration of exposure to pressure could be misleading. Therefore until it is understood as to whether cell death is triggered by either a critical value of carbon dioxide being achieved or the total accumulation of carbon dioxide over a period of time, a decision can not be made as to which method is most representative of the risk of tissue death occurring as a consequence of pressure.

However, the types of responses could be used to describe the adaptive and non-adaptive responses of individual's to pressure. Hence, if following future work a type A(iii) response is associated with tissue death as soon as the response of $tcPCO_2$ is identified as following a quadratic curve the period of time that the pressure is applied for could be reduced, thereby minimising the risk of tissue damage as a consequence of the test itself.

It was interesting that some of the volunteers were identified as presenting a response Type A(iii), associated with the tissue demonstrating signs of non-tolerance, to pressure exerted through their own body mass. This raises the question as to whether some individuals are innately more susceptible to tissue damage due to pressure than others and, importantly, would support the requirement for individual assessment. One of these volunteers, presented with the same response type to relatively low pressures when the range of pressures (10 to 100mmHg) were applied to their sacral tissue. This supports the consistency with which individuals' sacral tissue responds to pressures. This study also identifies $tcPCO_2$ as the stronger indicator of the degree/severity of sacral tissue ischaemia. However, further work is required to observe the levels of $tcPCO_2$ associated with sacral tissue cell death to understand the significance of peak level of carbon dioxide achieved in the tissue, the accumulated total quantity of carbon dioxide in the tissue, or duration of time that local tissues are exposed to elevated carbon dioxide levels. A research protocol was submitted for ethics committee approval to examine $tcPCO_2$ levels in the patient population, but in the current form has been declined. Such a study is the

next step within the overall research. This will enable the examination of patients' responses to pressure exerted through their own body mass to be compared with those identified for the normal healthy population. In addition differences between patients considered who do and do not go on to develop a pressure ulcer during their hospital admission.

### 5.2.4 A Rapid Rise of $tcPCO_2$ with the Associated Reduction of $tcPO_2$, Observed in Response Type A(iii) is Representative of Capillary Closure

As discussed in 5.2.3 the severest response to pressure observed was the Type A(iii) response. This response was considered to represent total capillary occlusion as a consequence of the pressure applied to the sacral tissue. The accumulation of carbon dioxide is explained by the continual production of carbon dioxide following capillary closure as a consequence of anaerobic cell metabolism. Further it was observed that the accumulation of carbon dioxide continued until the pressure was released and capillary blood flow reinstated. This is because the local blood supply is responsible for the removal of carbon dioxide from local tissues by diffusion and transports the carbon dioxide to the lungs where it is expired from the body via ventilation. Pressure as low as 50mmHg was identified as sufficient to cause this response for some individuals (see section 4.4, Table 4.11). This is lower than pressures identified by Knight et al. (2001) who identified that pressures of 80mmHg or more were required to stimulate a similar type of response.

In order to test whether the Type A(iii) response was representative of a response associated the total occlusion of blood flow pressure was applied to occlude the brachial artery and simultaneous measurements taken of changes in $tcPO_2$ and $tcPCO_2$. Results were then compared with those of the response Type A(iii). The responses were observed to be very similar with the mean maximum $tcPCO_2$ levels achieved under pressure for 10 minutes being 13.4kPa for pressure sufficient to cause arterial occlusion and 13kPa for the Type A(iii) responses. The standard deviation was 0.8kPa for each group of responses. As Type A(iii) response for $tcPCO_2$ also supported the findings of Hotter et al. (2004), when the renal artery was occluded for 30minutes, it is concluded that response Type A(iii) is representative of capillary closure, with consequent tissue ischaemia.

### 5.2.5 Significant Differences Between Sacral Sites when Exposed to pressure

As identified in section 4.2.4, each volunteer's two sacral sites responded differently to pressure. Indeed, the difference was such that for some individuals' each sacral site presented a different response type to the same level of pressure. The correlation coefficient between the left and right sacral sites for tcPO2 and $tcPCO_2$ was found to be very poor with r values of 0.39 and 0.41 respectively. Therefore each sacral site was treated as an independent site. This has not been noted in previous studies and supports the premise that different sites should be assessed separately rather than taking one measurement to be representative of the whole individual.

The work examining individuals' sacral tissue response to their own body mass raised the question as to whether the differences in the response of the sacral sites was posturally related. This was because all volunteers adopted a left lateral, supine position for the measurements. However, the results of the experiment described in section 4.4 excluded the influence of posture as the side with the higher measurements fluxuated between sessions. The differences may be due to differences in local tissue structure possibly due to the dominant side having better muscle bulk and established capillary network, or that as the main artery divides from the common abdominal aorta and each sacral site is served by arterioles branching off the right and left common iliac artery.

### 5.2.6 Variability in Pressure at which Individuals, and the Individual's Left and Right Sacral Sites, Present Each Response Type of $tcPO_2$ and $tcPCO_2$ – Critical Pressure Threshold

For the six volunteers studied in section 4.4.2 the pressure, at which the different types of responses changed, varied between individuals and between individuals' sacral sites. Response Type A(iii) was observed for one volunteer at a pressure as low as 50mmHg. In contrast when 100mmHg of pressure was applied to another volunteer they had one sacral site that maintained $tcPO_2$ levels above 3kPa and $tcPCO_2$ was observed as being within the normal range thus indicating a high tolerance to pressure (see Table 4.11).

Changes were also examined in terms of area under the curve which represents the accumulated loss of oxygen and the accumulated gain of carbon dioxide. Similar results

were obtained using this approach of analysis, in that, a wide variation in tolerance to pressure was observed between individuals and individual's sacral sites.

Further the range of pressures over which each individual sacral site remained within one response type also varied. For example, Type A(i) responses were observed in some individuals at up to pressures of 40mmHg only. In others this response type was maintained when pressure was increased to 100mmHg. For Type A(ii) responses, in some individuals it was found that pressure only needed to be increased by an additional 9mmHg for the response to change to a Type A(iii). In others up to 19mmHg was required for the response to change. These findings suggest that using a pre-determined pressure is an inappropriate approach to adopt when seeking to identify an individual's tolerance to pressure. Such a finding is critical when developing a risk assessment tool based on physiological measurements.

Seven of the sacral sites translated from a Type D and Type A(i) response where, following the application of pressure, $tcPO_2$ levels showed signs of recovery following the initial decline in levels and $tcPCO_2$ levels remained within the normal range, to a Type A(iii) response with $tcPO_2$ levels falling and remaining below 1 kPa and $tcPCO_2$ levels rising significantly to levels in excess of 12kPa. There is some fluxuation between Type C and D response, but once pressure has been sufficient to result in a Type B response for $tcPO_2$ if a fluxuation in response type occurs it is between response Type A and B.

Similarly if the pressure has been sufficient to result in a Type A(ii) response it may fluxuate with a Type A(i) response. But if a Type A(iii) response has resulted as a consequence of the pressure applied then it only fluxuates with a Type A(ii) response.

These findings may support the view that the critical threshold of the response type and changes between the response types at certain levels, are the result of systemic changes in blood pressure that are sufficient to either improve or reduce the local tolerance pressure. Volunteer's blood pressures were taken at the end of each measurement, however, this was insufficient to examine whether there was a direct relationship between blood pressure and type of response and should be addressed in future work.

Whether the nature of the response was examined by response type (see Table 4.11), maximum level of $tcPO_2$ and $tcPCO_2$ (see table 4.13) or area under the curve (see Table 4.15) the critical pressure thresholds varied. As suggested above individuals tolerance to pressure can not be measured by applying a pre-determined pressure as was the case in

previous studies (Seiler (1979); Bader (1990); Knight et al. (2001)). Rather there is a need to identify the nature of the response associated with the pressure exerted as a consequence of the individual's own body mass, and then identify by how much the pressure applied needs to be reduced until the response is consistently within a Type D/C response type for $tcPO_2$ combined with a Type A(i) response for $tcPCO_2$. Such a response would indicate that an apparent tolerance to the pressure being applied was being maintained.

## 5.3 Conclusion and Way Forward

The above discussion leads to the following conclusions and recommendations.

There was no significant relationship identified between body mass index (BMI) and interface pressure. Whilst this finding is supported by the work of others, body mass index is considered too crude a measurement as it does not take into account the thickness, tone or mechanical integrity of an individual's subcutaneous tissues. Similarly there was no significant relationship identified between the type of response of $tcPO_2$ and $tcPCO_2$, and BMI or interface pressure. It is recommended therefore that the use of BMI as part of current risk assessment tools be reviewed with the aim of replacing it with a more sophisticated approach to appraising the effect of an individual's body mass on their ability to tolerate pressure ulcers.

No significant difference was identified between baseline levels of $tcPO_2$ and $tcPCO_2$, for the left and right sacral site and control site (sub-clavicular). This suggests that the control site chosen for this study was appropriate.

A significant difference exists between the response of individuals' right and left sacral sites to pressure: this was an unexpected finding. The difference was such that an individuals' left and right sacral site could present a different type of response to the same level of pressure. This has not been noted by previous studies and may support the anecdotal evidence that clinically a patient can be assisted to change their position at regular and even time intervals, but a pressure ulcer may develop on one sacral side, but not the other. This supports the view that tissue tolerance varies not only inter-subject, but also intra-subject. Therefore it is recommended that the two sacral sites be considered independently of each other when determining individual's tolerance to pressure. Further work is required to explore this finding and what factors lead to the differences in response, such as differences in the quality and depth of epidermis, subcutaneous and

muscle layers and how they are altered by pressure. This could possibly be explored using of MRI.

The sacral tissue of normal healthy volunteers presents a range of responses when subjected to pressure. Changes in response types of $tcPO_2$ are less significant as they are only sensitive to the early changes in tissue as a consequence of pressure. The responses for $tcPCO_2$ reflect the findings of other studies. In addition this study has identified a further response type - Type A(iii) - for sacral tissue. This response, where $tcPO_2$ falls and remains below 1kPa and $tcPCO_2$ levels significantly rise to levels exceeding 12kPa with a maximum of 22kPa identified, is representative of the response associated with capillary/arterial closure, and is representative of a non-adaptive response. The response type exhibits similar changes in $tcPO_2$ and $tcPCO_2$ as that observed by others in renal tissue and cardiac tissue when vessel closure and ischaemia present. This finding is important as it indicates that carbon dioxide is a stronger indicator of capillary occlusion and tissue ischaemia, the most likely key controlling factors in pressure ulcer development.

The study identified that changes in $tcPCO_2$ response types were not observed until $tcPO_2$ remained below 1 kPa. This has not been described previously for sacral tissue in humans, although animal studies have identified a similar relationship between changes in oxygen and carbon dioxide levels under ischaemic conditions.

The study has identified the significant variation in tolerance that individuals have to different levels of pressure. Such variation can be represented by the range of responses observed both to the pressure exerted through individuals' own body mass and the variation both inter-subject and intra-subject in the pressure required to stimulate the different response types in individuals. The identification of the type of response through the pattern of response of $tcPO_2$ and $tcPCO_2$, maximum level of $tcPO_2$ and $tcPCO_2$ achieved under pressure and Area Under the Curve (see Table 4.11, 4.13 and 4.15 respectively) could be used to identify individuals' tolerance of tissue to pressure. This finding has significant implications for the development of risk assessment tools based on physiological measurements.

Exploratory work has identified that individuals have different levels of pressure for critical thresholds between response types, and that the range of pressures that each response type covers also varies. The sample population was small for this part of the study and so further work is required to substantiate the significance of the findings. Continuous blood

pressure monitoring should also be considered in order to identify the influences of systemic changes on observed fluxuations in tolerance at the critical threshold.

All the responses observed are associated with changes in local capillary flux as a consequence of pressure applied to tissue. This confirms that the responses observed are related to local capillary blood flow. As such changes in $tcPO_2$ and $tcPCO_2$ are directly attributable to changes in blood flow.

The nature of response Type A(iii) was found to be a quadratic curve and may be used for future modelling. This differs from the exponential curve identified in previous animal renal studies. It is recommended that future work examines further the nature of the response, and whether this can be used as an early indicator of response type. This would enable the period of time that pressure is applied for to be reduced minimising further any risk of tissue damage as a consequence of the measurement methodology.

The gradient of the response of $tcPCO_2$ was significantly different between the response types (Type A i, ii, and iii), and as with the quadratic curve could be considered as a factor in future work to identify the response type within a shorter time period for the application of pressure.

The research has shown that once $tcPO_2$ level has fallen below 1 kPa the change in $tcPCO_2$ level is the stronger indicator of local tissue response to pressure, and is able to indicate the severity of local tissue ischaemia. Further work is required to understand the relationship between cell death and the following parameters:

- peak level of $tcPCO_2$ achieved;
- the total accumulated quantity of carbon dioxide; and
- the duration of exposure to a certain level of $tcPCO_2$ .

This current study has investigated responses in healthy individuals. It is recommended that future work should include the monitoring of patients who naturally, due to their medical condition, go on to develop tissue damage as a consequence of pressure exerted through their own body mass. The work to date suggests that tissue damage is associated with a certain response type, for example Type A(iii), and as such it could be used as a physiological predictive tool to prevent the development of pressure ulcers by intervening to either improve local tissue tolerance to pressure or by reducing the interface pressure sufficiently to a level identified as being safely tolerated by the individual. Such a tool

would prove invaluable to the health and welfare of patients and to the staff caring for them.

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# Volunteer Information Sheet

**Study Title:**
A study exploring the relationship between the blood supply to the tissue overlying the bottom and pressure exerted through the individuals own body weight.

**Invitation Paragraph:**
You are being invited to take part in a research study. Before you take part it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

Consumers for Ethics in Research (CERES) publish a leaflet entitled 'Medial Research and You'. This leaflet gives more information about medical research and looks at some questions you may want to ask. You may obtain a copy from CERES,PO Box 1365, London N16 OBW.

Thank you for reading this.

**What is the purpose of the study?**
Pressure sores are a significant problem causing tremendous pain, discomfort and reduced quality of life. The severity of pressure damage can prolong length of stay in hospital and be a potential source of serious systemic complications. This is detrimental to the individual and can also delay admission to hospital for others. The most common site that pressure sores develop is the bottom.

Pressure sores are described as an area of tissue that has died due to pressure distorting the blood vessels so cutting off the blood supply for a critical length of time. Pressure is influenced by factors such as position, tone and condition of tissue, and body weight. Duration of pressure is influenced by an individual's ability to sense discomfort and change position to relieve the pressure. Many additional factors have been identified as reducing an individual's tolerance to pressure, for example, medical conditions affecting the effectiveness of the circulation of blood, or the level of oxygen transported by the circulating blood supply to tissues, which is essential for the survival of tissue cells.

Pressure sores are considered to be preventable in the majority of cases with the correct intervention and care. The key to prevention is identifying those patients at risk. Risk assessment tools used to identify those high risk patients, have up to now been developed on opinions of relative importance of possible risk factors, rather than evidence based. By examining the relationship between pressure applied to an individual's bottom and the effect on the blood supply, it will contribute to the development of an evidence based risk assessment tool, so enabling appropriate care to be given. This study will help to identify if the proposed method for undertaking the measurements is accurate, repeatable and acceptable. The study will take place over a period of 3 months.

**Why have I been chosen?**
We are looking for healthy volunteers to identify if the proposed method for undertaking measurements is accurate, repeatable and acceptable. The results will also help to explore the normal relationship between pressure applied to the bottom of a healthy individual, and the effect that applying the pressure has on the blood supply to the area on the bottom being pressed. You have kindly expressed interest in participating in the study. If after reading this information sheet you are still interested, and meet the requirements for the experiments described below, you can volunteer to take part.

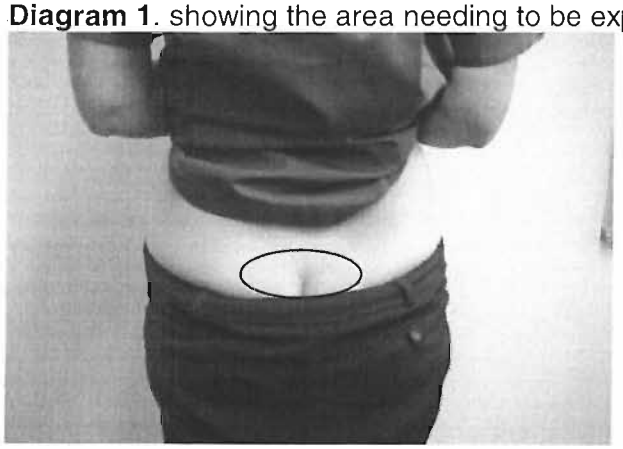
**Do I have to take part?**
It is up to you to decide whether or not you wish to take part. If you decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time and without giving a reason.

**What will happen to me if I take part?**
If you agree to take part in the study you will have the choice of participating in 1, 2 or all 3 of the experiments. Each experiment takes approximately 1 hour and for experiment 3 you will need to visit for two, separate, identical sessions.

***Experiment 1***
This experiment is to test the accuracy and repeatability of identifying the area of highest pressure on your bottom when you are lying on a bed. You will be asked to lie on a bed, and your bottom will be positioned on a thin (1mm) pressure sensing mat (PSM). The PSM measures the pressure resulting from your own body weight. The readings will take up to 5 minutes. You will then be asked to roll slightly to one side to enable a small solid marker to be placed on the skin of your bottom where the PSM has identified the highest pressure point to be. In order to position the solid marker your trouser's/skirt and undergarments will need to be loosened and moved down just enough to expose your lower back/cocyx. This will enable the identified area only, to be exposed and marker positioned (see diagram 1 below). This again should take up to 5 minutes.

You will then be asked to roll back and the marker checked for correct positioning. If correct, the solid marker is removed and the position identified with a marker pen, which will wash off. The experiment will then be repeated three times.

**Diagram 1**. showing the area needing to be exposed for the experiments 1-3.



**Experiment 2**
This experiment will examine the effect of incremental increases in pressure on the blood supply to an area of your bottom.

You will not be able to participate in this experiment if you are currently a smoker, under the care of your GP or Hospital Doctor, taking any medication, or suffer from any known heart or

lung disease. Your blood pressure, height and weight will be measured, and you will be asked your age, medical history and for women, what point you are in your menstrual cycle because this influences the blood circulation. The pressure points will be identified as described in Experiment 1. Following this an unfastened broad belt will be positioned around you, covering the site of highest pressure. You will then be asked to lie on the bed, on your left side, and the belt released. You will need to undress sufficiently to expose the area of skin that has been marked to identify the point of highest pressure. This will be achievable by loosening your trousers/skirt and undergarments sufficiently to enable them to be rolled down just enough to expose your lower back/cocyx area.(see diagram 1).
For the first 4 volunteers only, the method for applying the pressure will be checked by laying the pressure sensing mat over your bottom and applying a transducer to the mat. The transducer, including the fixation ring, is approximately 30mm in diameter. Pressure is then applied by inflating a bladder positioned over the transducer and held in place by fastening the broad belt. The belt and PSM will then be removed.

All volunteers, including the first 4, will then be rested for 10 minutes and the transducer, and fixation ring (approximately the size of a £2 coin) applied to the skin of the area identified on your bottom. The transducer is solid state and non invasive. It warms the skin and is able to measure the level of oxygen and carbon dioxide in that area, indicating the effectiveness of the blood supply. A baseline measurement will be taken of the effectiveness of the local blood supply when no pressure is applied followed by the application of 10mmHg for 15minutes. The application of pressure is achieved by positioning an inflatable bladder over the transducers and the belt reapplied to hold the bladder in position. The bladder will then be inflated to the pressure required to be applied. After 15minutes the pressure will be released by releasing the belt and deflating the bladder, and the blood flow monitored for a further 10 minutes. Once the measurement is complete the transducer is removed and the experiment concluded.

On nine subsequent visits the pressure applied will be increased by increments of 10mmhg until 100mmHg is achieved. Therefore in total ten visits, lasting up to 45 minutes are required for the completion of this experiment.

***Experiment 3.***
This experiment will examine the response of the blood supply over the surface of the bottom in response to pressure being applied that is equal to that exerted through the individual's weight.
You will not be able to participate in this experiment if you are currently a smoker, under the care of your GP or Hospital Doctor, taking any medication, or suffer from any known heart or lung disease. Your blood pressure, height and weight will be measured, and you will be asked your age, medical history and for women, what point you are in your menstrual cycle because this influences the blood circulation. You will be asked to lie flat on the bed and the three highest pressure points will be identified, using the procedure described in Experiment 1. Your head will then be elevated to a 30 degree angle and three highest points of pressure identified. You will then be asked to roll onto your side with an unfastened broad belt positioned around bottom. After resting for 10 minutes three transducers will be applied to the three highest pressure points identified. In order to position the transducers you will need to undress sufficiently to expose the area of skin that has been marked to identify the point of highest pressure. This will be achievable by loosening your trousers/skirt and undergarments sufficiently to enable them to be moved down just enough to expose your lower back/cocyx area.(see diagram 1). A fourth transducer will be positioned on the upper chest. The transducers will take approximately 15 minutes to stabalise after which the pressures identified will be applied by laying an inflatable bladder over the transducers all of which will be held in place by fastening the broad belt. After the readings have stabilised the pressure will be released and the broad belt unfastened. The transducer measurements will continue until they have returned to normal. The transducer will then be removed and the experiment

concluded. Arrangements for a follow-up appointment to repeat the measurements will be made or confirmed.

For all three experiments if you are male, as the researcher is female, a chaperone will be present.

**What do I have to do?**
Experiment 1: You will not have to do anything in preparation.

Experiment 2 & 3: You will need to avoid caffeine containing drinks and alcohol for 8 hours before the study and excessive exercise 2 hours before.

**What are the benefits of taking part?**
There are no benefits for taking part and the measurements will not affect you at all. The results will be used to identify if any refinement to the method is required, and the data will indicate reference ranges which will be invaluable for future research.

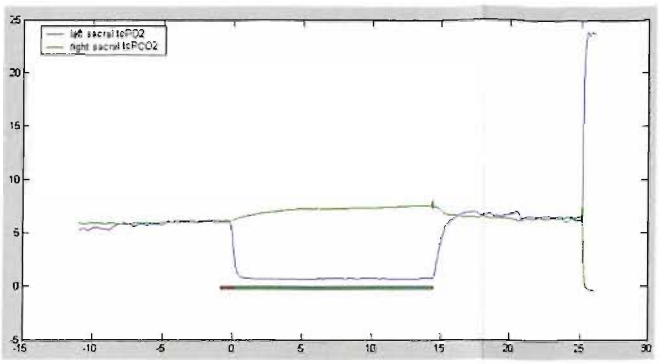
**Will taking part in this study be kept confidential?**
All information collected about you, during the course of this research, will be kept strictly confidential. Any information will be identified by a number and no record of your name or address with be kept with the information.

**Who is organising the research?**
This study is organised and funded entirely by Southampton University Hospitals NHS Trust.

**Who has reviewed the study?**
This study has been approved by Southampton and South West Hampshire Local Research Ethics Committee. Submission number: 026/04/t

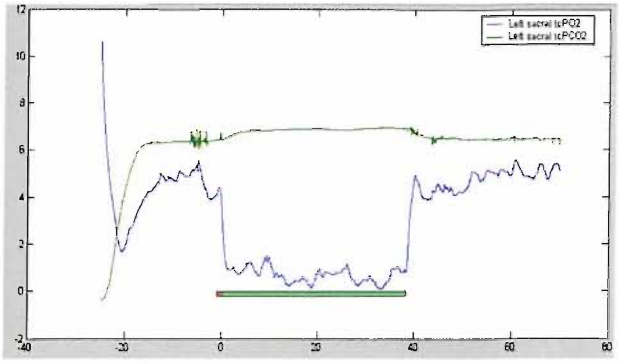
**Contact for further information.**
Judith Sillitoe, Department of Medical Physics & Bioengineering, Southampton General Hospital, Tremona Road, Southampton, SO16 6YD.
Tel: 02380796794, Email: Judith.sillitoe@suht.swest.nhs.uk

Thank you for considering taking part in this study.



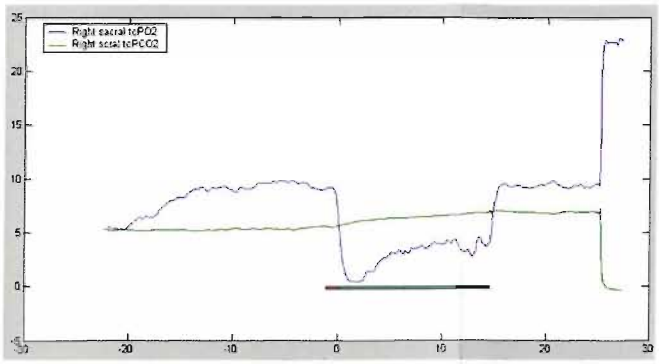

Time (mins)

**Figure 4.9 Type A response for $tcPO_2$**



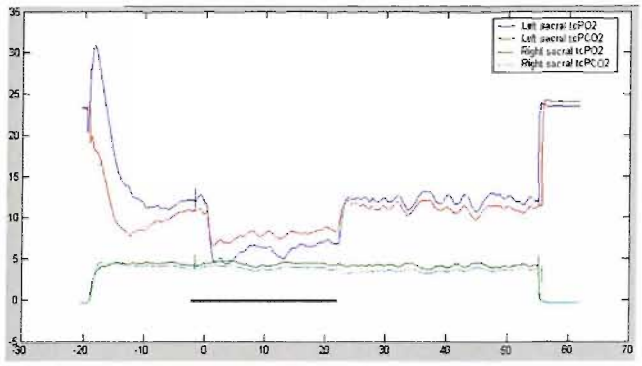

Time (mins)

**Figure 4.10 Type B response for $tcPO_2$**



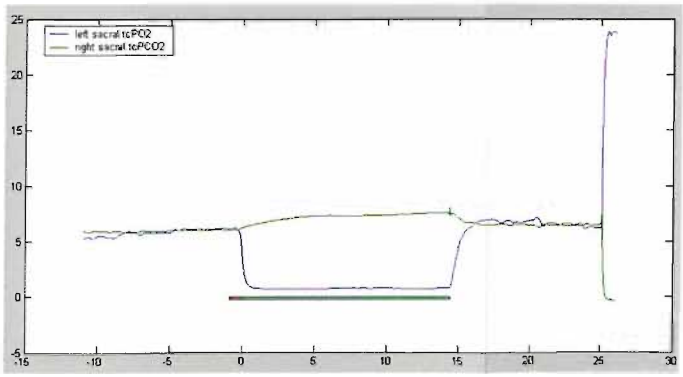

Time (mins)

**Figure 4.11 Type C response for $tcPO_2$**



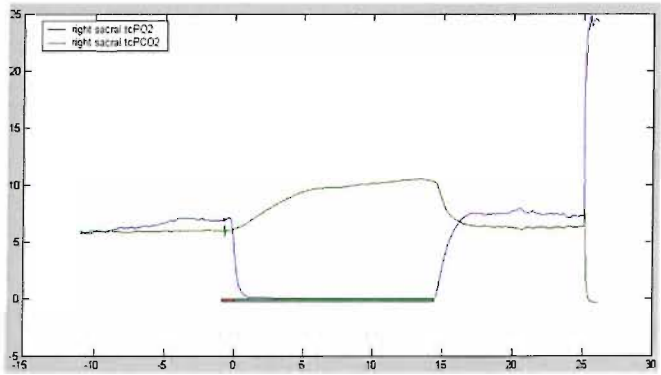

Time (mins)

**Figure 4.12 Type D response for $tcPO_2$**




Time (mins)

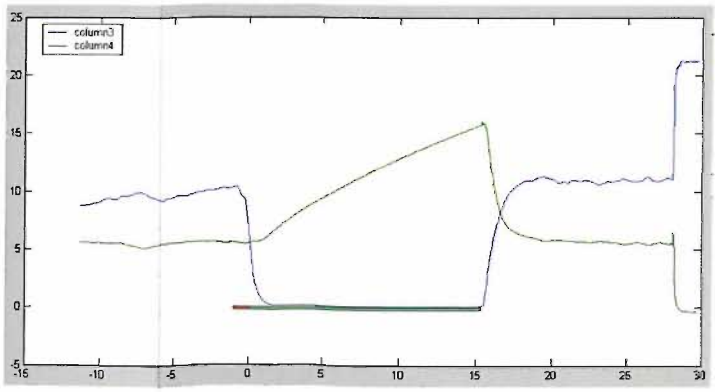
**Figure 4.13 Type A(i) response for $tcPCO_2$**




Time (mins)

**Figure 4.14 Type A(ii) response for $tcPCO_2$**

kPa




Time (mins)

**Figure 4.15 Type A(iii) response for $tcPCO_2$**