6 Susceptibility of Spinal Cord-Injured Individuals to Pressure Ulcers

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Introduction

The development of pressure ulcers due to tissue breakdown and cell necrosis is one of the most significant secondary complications of spinal cord injury (SCI). There are many factors that lead to the occurrence of tissue breakdown. Similarly, SCI affects multiple systems of the body, primarily below the level of the lesion but also with systemic effect. The most readily obvious change in an individual with SCI is altered motor function, particularly that which affects limb movement. In addition, sensory dysfunction can alter proprioception and reaction to environmental stimuli such as pain and temperature. Over time, the body will change, as muscle bulk is lost due to disuse muscle atrophy, leading to a higher proportion of fatty tissue and poor vascularity. Other systems affected include cardiovascular, respiratory, bowel and bladder function, and digestion. The extent of systemic involvement and functional ability directly affects the individuals' susceptibility to development of pressure ulcers.

In order to clinically describe individuals with SCI, the American Spinal Injuries Association (ASIA) has developed a standardized method for determining the neurological status of a patient and for classifying SCI. The ASIA impairment score has two components: neurological level of injury and neurological impairment. The first component describes the motor and sensory level of injury. The second describes the functional ability (Table 6.1). The ASIA impairment score is widely accepted as a clinical measure and as such it can be used as a broad indicator of a sub-population's risk for pressure ulcer development. The neurological level and extent of impairment are often very different from the anatomical level of injury, and the ASIA score provides a more reliable indicator of risk status than level of injury.

Just as the ASIA impairment score contains more than one component, so the development of pressure ulcers is a multi-factorial process. These factors can be classified as extrinsic factors primarily related to the interface between the individual and external environment and intrinsic factors related to the clinical and physiological profile of the individual. Changes in clinical status following injury will alter intrinsic factors that increase the risk of tissue breakdown leading to pressure ulcer development. For example, urinary incontinence will alter the micro-environment of the skin surface and make it more susceptible to maceration and breakdown.

Table 6.1. ASIA Impairment Score

ASIA grade	Neurological function		Neurological impairment
	Motor injury	Sensory injury	
A	Complete	Complete	No motor or sensory function in the sacral segments S4–S5
B	Complete	Incomplete	No motor function below the level of injury. Sensory function may be normal or impaired but is present.
C	Incomplete	Incomplete	Some motor function is preserved below the level of injury but more than half the key muscles show significant weakness
D	Incomplete	Incomplete	Motor function is preserved below the level of injury and less than half the key muscles show significant weakness
E	None	None	Normal motor and sensory function

Changes in environmental factors, such as the seating system used, will alter external factors that affect pressure ulcer risk status, as summarized in Table 6.2.

The interactions between risk factors and clinical status provide guidelines for susceptibility to pressure ulcer development both generally, e.g. for patient sub-groups such those with complete quadriplegia (ASIA grade A), and specifically for individuals.

Intrinsic risk factors in pressure ulcer development arise as a direct consequence of the SCI. Motor paralysis below the level of a spinal cord lesion reduces muscular activity and leads to loss of muscle bulk, thus reducing soft tissue coverage over the bony prominences of the pelvic region. The proportion of avascular fatty tissue increases, leading to decreased regional vascularity. Loss of normal muscle tone leads to abnormal responses to environmental stimuli, such as applied pressure, thus increasing the risk of blood flow becoming compromised. Furthermore, individuals with higher levels of SCI are likely to experience dysfunctional central control of circulation, which can lead to autonomic dysreflexia.

Table 6.2. Factors in pressure ulcer development

Factor	Classification	Cause
Disuse muscle atrophy	Intrinsic	Motor paralysis
Reduced vascularity and blood flow	Intrinsic	Motor paralysis
Impaired/absent sensation	Intrinsic	Sensory paralysis
Reduced mobility (including loss)	Intrinsic	Motor paralysis
Poor nutritional status	Intrinsic	Poor diet
Applied pressure	Extrinsic	External loading of soft tissues
Shear at the support interface	Extrinsic	Poor posture and/or poor support materials
Adverse micro-environment at support interface	Extrinsic	Numerous, including raised temperature, sweating, incontinence, infection

Motor paralysis will also directly affect a person's ability to respond unconsciously to potential noxious stimuli, e.g. fidgeting while sitting or turning while asleep. Reduced mobility also profoundly alters the individual's ability to consciously perform postural manoeuvres necessary to relieve prolonged applied pressure, from weight-shifting while sitting to walking. The loss or reduction of mobility is further compromised by sensory paralysis, leading to the absence or alteration of normal perception of environmental stimuli such as pain or temperature. Individuals with complete sensory paralysis can no longer sense where their limbs are without visual cues, i.e. they experience a loss of proprioception. These changes affect the risk of pressure ulcer development because the individual cannot sense the warning signals that prompt action to prevent tissue damage.

Thus it can be seen that many of the primary factors that increase the susceptibility of individuals with SCI to pressure ulcer development are the inter-related intrinsic changes in body characteristics and functional abilities which occur following SCI. To a large extent, these changes have been considered to be immutable. Nutritional status can be altered by adequate diet, but a complete SCI remains an irreversible lesion that does not exhibit spontaneous recovery. There is much research currently in progress to develop clinical treatments to cure SCI. This remains exploratory, and thus the current clinically applicable techniques to prevent pressure ulcers largely address extrinsic factors that can be changed, such as applied pressure and the micro-environment of the user/support interface. These approaches to pressure ulcer prevention can be classified as education focused or device focused. Educational prevention techniques include programmes that address patient and/or carer education. Device-oriented methods focus on

the provision of appropriate equipment for postural support and pressure relief, such as mattresses and wheelchair seating cushions. These approaches to pressure ulcer prevention are complementary and should be reviewed periodically throughout the lifetime of the individual with SCI.

Variation in Pressure Ulcer Risk Factors Following SCI

The clinical profile of an individual with an SCI will vary both among individuals, due to different levels of lesion, and for one person over time. The individual with an SCI will undergo the ageing process with chronic motor and/or sensory dysfunction. Moreover, it has been shown that an SCI can increase the effective rate of ageing [1, 2]. As more people live longer with SCI, research has been carried out to determine the longer-term risk status for clinical complications. With specific regard to the risk of pressure ulcer development, the individual with SCI remains at increased risk at all times post injury; however, the relative risk status and the most critical risk factors will vary over the course of time. The first stage at which pressure ulcer risk is heightened is in the acute SCI phase, immediately following injury.

Risk Factors During SCI

Following traumatic injury to the spinal cord there is an immediate decrease or loss of reflex activity below the level of the lesion. This condition is known as 'spinal shock' and although it is transient, there is considerable variation in the period required for the restoration of this activity, ranging from a few days to several months. Reflex return may be particularly delayed in patients with severe but incomplete SCI [3]. In addition, there is frequently concurrent trauma to multiple systems, which must be addressed with urgency if the patient is to survive the initial insult. Thus, the treatment received in the period immediately following a traumatic SCI is critical both to initially stabilize the patient and to ensure an optimal prognosis once the acute phase is past. It is important that skin care is rigorously monitored, for the development of pressure ulcers during the immediate post-injury phase will severely impede subsequent rehabilitation.

Patients who sustain an SCI should be admitted as soon as possible to a specialized spinal injuries unit, because secondary complications will start very rapidly following SCI. Even among specialized units, the standards of care for acute SCI vary from conservative treatment to rapid spinal stabilization and remobilization. The original protocol pioneered by Guttmann takes a very conservative approach [4]. The patient is immobilized for up to 12 weeks, to allow the traumatic fracture to stabilize, before gradually commencing a rehabilitation programme. Tissue health status should continue to be monitored to avoid development of pressure ulcers. In addition to prolonging the acute phase of the hospital admission, the early develop-

Table 6.3. Demographics of acute SCI study population

Gender	Age	Level of injury	Extent of injury
Male: 73%	16–32 years	Above T6: 33%	Complete: 40%
Female: 27%	Mean: 22 years	Below T6: 67%	Incomplete: 60%

ment of pressure ulcers can negatively affect the individual's psychological adjustment to life with an SCI. The pressure-relieving characteristics of the user support surfaces, at this stage specifically the mattress and any other support pillows, should therefore be evaluated together with the positioning of the individual in the bed.

Conservative management of acute SCI, such as employed at the National Spinal Injuries Centre, Stoke Mandeville NIH Trust, is for the patient to be immobilized until the fracture is stable, as seen on radiographic assessment of the site. The patient is positioned in a supine position with the spine in alignment and lies on a foam mattress with pillows placed under the head, buttocks and ankles to relieve pressure on the high-risk pressure points of the occiput, sacrum and heels. A turning regime is employed where the patient is turned from side to side using a turning bed at 2- to 4-h intervals, with the spine maintained in alignment at all times.

A study was carried out to evaluate the efficacy of this clinical treatment protocol for maintenance of tissue health. Interface pressures and transcutaneous oxygen levels were measured at the sacrum of subjects who had sustained a traumatic SCI [5]. Fifteen individuals were admitted to the study within 9 weeks of injury. The clinical demographics of the study population are summarized in Table 6.3.

Transcutaneous oxygen levels were measured using a Radiometer TCM3 blood gas monitor (Copenhagen, Denmark). The sensor electrode was placed over the sacrum along the midline. Concurrently, interface pressures were measured on either side of the tissue gas electrode using an Oxford Pressure Monitoring system (Talley Medical, Romsey, Hants, UK). Measurements were made for a period of 25±5 min at intervals of 1–2 weeks until the subject began to remobilize in a wheelchair.

It was found that median interface pressures at the sacral region were around 30 mmHg. This implies that the practice of "gapping" patients on pillows to relieve pressures over the bony prominences is often ineffective because an adequate gap is not achieved. The simplest approach to remedy this situation is to employ clinical guidelines that indicate the minimum gap necessary around a bony region. This would generally be around 10 cm, but this may not always be feasible if there is significant spinal instability, and in such cases it would be more effective to use an active pressure relief mattress, such as a Clinitron bed, (Hill-Rom, Ashby de la Zouch, Leicestershire).

No significant relationship between transcutaneous oxygen level and time post injury was found, indicating that the risk of tissue breakdown did not alter during the period of acute immobilization for this subject group. This implies that rapid remobilization and reduced frequency of turning when in bed may be inappropriate for many individuals with acute SCI due to the prolonged effects of spinal shock. As the proportion of cases of incomplete spinal cord trauma increases and acute bed rest continues to become briefer, this has significant implications for the clinical care of acute SCI patients.

Risk Factors During Initial Rehabilitation

The initial rehabilitation (sub-acute) phase for an individual with SCI can vary in both time post injury (from a few weeks to several months), depending on the course of the acute phase, and duration (also from a few weeks to several months). The goal of initial rehabilitation post SCI is to equip the individual with the skills and equipment necessary for them to maximize their potential abilities so that they can become reintegrated in society. This goal requires much hard work by the affected individual, his or her caregivers and the whole clinical team. It is of primary importance that during initial rehabilitation every patient and carer is thoroughly educated in the aetiology of pressure ulcers and their prophylaxis. Critical skills to be learned include the ability to carry out a pressure relief regime, both through postural changes where possible and through the provision of appropriate equipment, e.g. cushions, wheelchairs, mattresses. The need for routine skin inspection and care must also be emphasized, with particular regard to pressure areas such as the ischia, sacrum and greater trochanters.

The selection of a wheelchair seating system for the person who has recently sustained an SCI must involve consideration of many diverse criteria. In some ways it is the most important part of rehabilitation. The right combination of wheelchair and support cushion will allow the user to maximize their functional potential and interact fully with their environment. In addition, it will take full account of the user's requirements with regard to particular needs and appearance. Conversely, an inappropriate seating system can lead to poor posture, reduced functional abilities and isolate the user from their environment. All these factors can, in turn, exacerbate the risk of pressure ulcer development in the rehabilitating individual.

Thus the seating requirements of each patient must be thoroughly assessed at this time so that appropriate seating and other support surfaces can be recommended. The prescription of wheelchair seating systems must be based on a comprehensive assessment of user function (actual and potential) and seated posture. The clinical profile should be considered but of greater relevance are the actual individual characteristics at the seating/support interface for a specific user.

A study was carried out to determine changes in transcutaneous oxygen response to applied pressure during the initial rehabilitation of SCI sub-

jects [6]. All study participants had suffered traumatic SCI less than 1 year previously and were assessed while sitting on their prescribed support cushions. The initial guidelines for cushion prescription were for all patients to receive a 4-in. foam cushion, except those with complete quadriplegia who more frequently received a Sumed gel cushion. Subjects with a history of pressure ulcer development during the acute phase were prescribed a Jay Medical or Jay Active foam/gel cushion. Subjects were classified according to their level of injury as paraplegic (below T6) or quadriplegic (above T6). Transcutaneous oxygen levels were measured over the bony prominence of the ischial tuberosity using a Radiometer TCM3 blood gas monitor. Initial assessment was made once the patient was sitting up for more than 4 h a day and was repeated at intervals of 2–4 weeks until discharge. The sensors were attached with the subject in a side-lying position with hips and knees flexed to approximate their relative posture in sitting. After a 10-min equilibration period the subject was carefully transferred to the sitting posture on their support cushion. Tissue status was then monitored for a continuous period of 25±5 min with appropriate pressure relief as required.

Tissue oxygen levels under applied load tended to improve during initial rehabilitation for quadriplegic subjects and to deteriorate for those with paraplegia. These counter-intuitive findings support the reports of others, such as Noble [7], that quadriplegics develop pressure ulcers less frequently than paraplegics, particularly than those with flaccid paraplegia. This may be because the spasticity experienced by individuals with higher level lesions means that loss of muscle bulk is slightly less than in those with no tone, i.e. with flaccid paralysis. The higher activity levels of paraplegic individuals may also be a factor since they may be more likely to neglect regular pressure relief manoeuvres due to their other activities.

Risk Factors for the Chronic SCI Population

Following initial rehabilitation, the SCI patient must maintain a high level of skin care at all times in order to prevent the occurrence of pressure ulcers. However, this economically and psychologically costly secondary complication remains one of the most common reasons for re-admission to hospital [7]. The patient with a major pressure ulcer requires an average of 180 days nursing time [8]. Allman et al. [9] found that development of a nosocomial pressure ulcer was associated with significant and substantial increases in both hospital costs and length of stay in a group of patients admitted to hospital with reduced mobility due to a primary diagnosis of hip fracture. Xakellis and Frantz [10] found that the cost of treating pressure ulcers was greatly increased when a patient required hospitalization. These studies did not focus on individuals with SCI, but it can reasonably be predicted that the outcomes would be poorer for those individuals with greater initial impairment. The most recent comprehensive figures available

indicate that the cost to the health service in the UK is in excess of £ 250 million per annum in 1990 [11]. In the USA, the cost of treating pressure ulcers was estimated to be in excess of $ 1.33 billion per annum in 1994 [12]. When adjusted for inflation, this implies that current costs are around £ 416 million per annum in the UK and around $ 1.89 billion per annum in the USA.

Thus it can be seen that the prevention of pressure ulcers is a highly cost-effective goal. Pragmatically, it is also important to have clear treatment guidelines for the efficacious clinical management of pressure ulcers when they do develop. Both the European Pressure Ulcer Advisory Panel (EPUAP) and the National Pressure Ulcer Advisory Panel (NPUAP) have issued clinical guidelines for the prevention and treatment of pressure ulcers [13, 14, 15]. It is recommended that conservative treatment options, such as topical dressings, be employed whenever possible for the individual with chronic SCI who develops a pressure ulcer. In most cases early identification of a Grade I or II pressure ulcer with superficial breakdown involving only the dermal layers can be treated successfully by complete bed rest with total pressure relief over the affected area combined with appropriate dressings, as the healing period is relatively short. There are many types of topical dressings and antibiotics that can be employed to promote wound healing [12]. The common goal is to produce a moist wound environment that will promote cell proliferation.

In some cases, tissue breakdown is so extensive that conservative treatment alone is not appropriate. Grade III or IV pressure ulcers involve total breakdown of the dermal and epidermal layers, sometimes extending to the underlying muscle, that requires a prolonged period of bedrest to heal. This cannot be considered acceptable. The presence of necrotic tissue (eschar) will impede wound healing and such tissue may be removed by surgical intervention, specifically sharp debridement. Excision of sloughy tissue and cleansing of the ulcer to stop infection should permit the development of granulating tissue.

Split skin grafts may be employed to promote healing in the early stages for pressure ulcers with limited muscular involvement. However if the pressure ulcer exhibits areas of deep tissue breakdown, e.g. extending to the bone with surrounding undermining and fibrotic margins (Grade IV and some Grade III ulcers), more radical surgical treatment is required. The overall goal of surgical procedures is to excise and close the ulcer. A number of tissue-flap procedures have been developed to achieve wound coverage. Myocutaneous flaps and island fasciocutaneous flaps are most widely used [16, 17], with fasciocutaneous flaps also being found to be successful on some non-healing pressure ulcers [18].

The principal procedure employed at the National Spinal Injuries Centre is simple excision of the ulcer and bony prominence followed by direct closure. The ulcer is excised in toto with the bony prominence underneath. The wound is then closed in layers with a primary skin closure. A retrospective review of surgical patients with pressure ulcers treated at the National Spinal Injuries Centre by excision and closure was carried out. All

patients admitted during a twelve year period (1980–1992) and treated by the same surgeon (IN) were studied and evaluated. A total of 400 operational procedures involving 218 patients were performed. There was a 2.25% incidence of multiple ulcers, leading to an overall total of 409 ulcers repaired surgically over the twelve year period.

The retrospective review of surgical cases showed that some patients experienced more than one surgical procedure during the period. It was important to differentiate between possible causes of repeated tissue breakdown and therefore two classes of repeated procedure were defined. Revision of a pressure ulcer was defined as a repeated surgical procedure at the same site within 1 year of the original procedure. This was considered to indicate that the original wound had failed to heal adequately. In contrast, recurrence of tissue breakdown was defined as a surgical procedure at the same site between 1 and 5 years after the original procedure.

During the 12-year review period 73 patients (33.5%) were treated on two or more occasions. In 37 of these cases this was due to bilateral and/or multiple ulcers being repaired by a series of surgical procedures. Surgical revision was found to have been required in 24 cases (6.0%). Recurrence of tissue breakdown was found to have occurred in 15 cases. One patient had two episodes of tissue breakdown at the same site 3 and 6 years after initial surgery. One other patient had recurrent breakdown over bilateral trochanteric regions after a 4.5-year interval. Thus a total of 18 ulcers (4.5%) recurred during the 12-year review period.

The relative prevalence of the 409 pressure ulcers treated by surgical excision and closure is summarized in Table 6.4, together with revision and recurrence rates. When these cases were classified according to the site of the initial pressure ulcer, revision rates appeared to be higher for the sacrum and greater trochanter than for the ischium. The reverse situation was seen with recurrence rates. Ischial ulcers were twice as likely to exhibit repeated breakdown requiring surgical repair as those occurring at the sacrum or greater trochanter.

Twenty-one (9.6%) patients had two or more episodes of tissue breakdown at different sites separated by periods greater than 1 year, e.g. initial breakdown of the ischial region with a second breakdown of the sacral region 4 years later. However, it was noted that 10 of these patients also had a history of concurrent multiple pressure ulcers at some time during the

Table 6.4. Revision and recurrence rates classified by pressure ulcer site

Pressure ulcer site	Revisions	Recurrences
Sacrum	7 (7.0%)	3 (3.0%)
Ischium	9 (5.5%)	10 (6.1%)
Greater trochanter	8 (6.6%)	4 (3.3%)

review period. Seven patients had a history of recurrent ischial ulcers where the left or right side was specified for all ulcers. In these cases, recurrent breakdown was contralateral in four patients and on the same side in two patients. One patient exhibited recurrent ischial breakdown both contralaterally and ipsilaterally. This finding implies that prophylactic ischiectomies may be effective at preventing recurrent breakdown over the same ischial tuberosity but they may increase the risk of contralateral breakdown due to postural asymmetry.

In this review we were able to assess the operational procedures by one surgeon over a 12-year period from 1980 to 1992, thus providing longer-term follow-up information. The recurrence rate of 5.3% for all ulcers represents an overall success rate of 94.7%, with 5 years' follow-up, for sacral, ischial and trochanteric ulcers closed by direct excision and closure. This compares favourably to other surgical techniques for the repair of pressure ulcers.

A prospective study was carried out to evaluate any changes in transcutaneous gas levels pre-operatively and to determine whether tissue health status is altered in unloaded and loaded soft tissues post-operatively. Transcutaneous gas levels were monitored in subjects who underwent surgical excision and closure of pressure ulcers during the period June 1989 to December 1990. Ethical approval for this study was obtained from the Aylesbury Vale Authority Research Ethical Committee. Twenty-one subjects were included in this study on meeting the selection criteria, i.e. normal haemoglobin levels pre-operatively, absence of systemic degenerative conditions and provision of informed consent. Three cases in this group had bilateral ulcers, leading to a total of 24 ulcers. The clinical demographics of this study population are shown in Table 6.5.

Tissue health was assessed pre-operatively once the area of tissue breakdown was free of slough and necrotic tissue. The sensor electrode of the Radiometer TCM3 blood gas monitor was located 20–50 mm from the margin of the wound over superficially healthy skin. Transcutaneous gas levels were monitored for a period of 25±5 min in order to determine a stable unloaded tissue response.

The pressure ulcer was then repaired by total excision of the necrotic tissue, ulcer and underlying bony prominence followed by primary closure. An elliptical incision was made around the ulcer, followed by excision of the

Table 6.5. Clinical demographics of surgical study population: summary of subject profiles

Gender	Age	Level of injury	Duration of injury	Location of ulcer(s)
Male: 76% Female: 24%	16–80 yrs Mean: 42 years	Above T6: 33% Below T6: 67%	Acute: 14% Chronic: 86%	Ischium = 58% Sacrum = 13% Trochanters = 29%

whole ulcer, making sure that the pseudo-epithelial lining of the ulcer was excised in toto. The underlying bony prominence was exposed and excised. In ulcers with undermined cavities, the boundaries of the cavity are easily identified by packing it with ribbon gauze and thus changing it into a pseudotumour. Dissection was then carried out to remove the whole lining.

Table 6.6. Clinical demographics of surgical study population: individual subject profiles

No.	Gender	Age	Level	Duration [a]	Location of ulcers	Previous surgical repair
1	M	50	A	Chronic	R ischium	N
2	M	27	B	Chronic	L ischium	N
3	M	42	B	Chronic	Sacrum	N
4	F	80	A	Chronic	L ischium	Y
5	M	59	A	Chronic	R ischium	Y
6	M	43	A	Acute	Sacrum	N
7	M	19	A	Chronic	L posterior trochanter	N
8	F	71	B	Chronic	L ischium	N
9	M	57	B	Chronic	R ischium	N
10	M	37	A	Chronic	B. posterior trochanters	N
11	M	19	B	Chronic	L ischium	Y [b, c]
12	F	36	B	Chronic	L ischium	N
13	F	22	B	Chronic	Bilateral ischia	– [c]
14	F	22	B	Chronic	R ischium and perineum	– [c]
15	M	16	B	Acute	Bilateral trochanters	N
16	M	38	B	Chronic	L posterior trochanter	N
17	M	19	B	Acute	Sacrum	N
18	M	50	B	Chronic	R ischium	N
19	M	72	A	Chronic	L trochanter	N
20	M	43	B	Chronic	L ischium	Y [b]
21	M	58	B	Chronic	L ischium	N

[a] Acute, less than 2 years post-injury; Chronic, more than 2 years post-injury.
[b] Previously repaired by rotation flap.
[c] Full surgical history not available.

The wound was then closed in as many layers as possible. This makes closure of the ulcer achievable without undue tension. A suction drain was always left for a few days in order to drain the deep area.

The sutures were usually removed in two stages at 10 and 11 days post-operatively. Twenty-four hours following removal of all sutures, transcutaneous gas levels in unloaded tissues were again assessed. The monitoring site was 20–50 mm medial to the mid-point of the suture line.

A further assessment of tissue response under load was carried out following remobilization in the wheelchair for 19 subjects. Transcutaneous gas levels were monitored at the same site as for the post-operative assessment. Regional interface pressures at the subject support interface were monitored simultaneously using the Oxford Pressure Monitoring system. The sensors were located using the same experimental protocol followed for the study of initially rehabilitating SCI subjects (see above). Sensors were attached over the region of the surgical repair with the subject in a side-lying position with hips and knees flexed to approximate their relative posture in sitting. The tissue gas electrode was located 20–50 mm medial to the mid-point of the suture line and the pressure sensors were placed either side. After a 10-min equilibration period the subject was carefully transferred to the sitting posture on their standard support cushion. Tissue status was then monitored for a continuous period of 25±5 min with the appropriate pressure relief as required.

The distribution of pressure ulcer locations found in the long-term review was reflected in the study of transcutaneous gas levels in surgical subjects, with ischial ulcers representing 58% of cases.

The unloaded transcutaneous oxygen pressure ($TcPO_2$) in normal healthy subjects is considered to be in the region of 80 mmHg [19]. The risk of tissue necrosis increases as the blood supply becomes inadequate and $TcPO_2$ falls. Unloaded $TcPCO_2$ is around 35 mmHg. If tissue health becomes compromised due to inadequate blood supply, $TcPCO_2$ will start to increase due to the accumulation of noxious by-products from tissue respiration. Pre-operatively, $TcPO_2$ was generally found to be in excess of 30 mmHg, and $TcPCO_2$ levels were abnormally high in a number of cases. Thus soft tissues surrounding regions of necrotic tissue may have slightly compromised tissue gas levels due to a reduced clearance of tissue waste, even though they may appear visually normal. Post-operatively $TcPO_2$ levels in unloaded tissues were generally observed to be in excess of 30 mmHg and $TcPCO_2$ was within the normal range for an increased number of subjects. Thus transcutaneous gas levels in unloaded tissue surrounding a repaired wound were similar to those found in other healthy soft tissues. This may be due to the operation itself stimulating blood flow, or it may simply be that regional blood flow improves as a result of the removal of necrotic tissue.

Future Developments in Pressure Ulcer Research: Decrease in the Susceptibility of the Individual with SCI

Despite the development of many support devices and the application of many training programs the incidence of pressure ulcers remains unacceptably high, particularly in the SCI population. Furthermore, there remain a significant number of individuals with SCI who exhibit chronic recurrence of tissue breakdown despite the use of high-performance support cushions.

Device-orientated prevention techniques continue to be developed and refined. Active pressure-relief mattresses often incorporate temperature sensors to control the micro-environment, and this type of technology is now starting to be applied in wheelchair cushions. In addition, 'smart' cushions have been developed that monitor the duration of applied pressure, i.e. static sitting time, and issue an alarm when the user should perform a pressure-relief manoeuvre.

Advanced technologies and new pharmacological approaches are being explored that can affect the intrinsic clinical status of the individual with a SCI. The long-term application of implanted electrical stimulation devices offers a unique means to alter the intrinsic characteristics of paralysed muscle, leading to sustained improvements in regional tissue health. These changes can reduce the risk of pressure ulcer development by increasing regional blood flow and improving interface pressure distribution [20]. In addition, electrical stimulation can be applied to dynamically alter conditions at the seating support interface through stimulated muscular contractions, thus facilitating periodic changes in interface pressure.

The use of anabolic steroids has also been investigated for both the treatment and prevention of pressure ulcers in the SCI population. Any individuals with chronic 'non-healing' pressure ulcers exhibit concurrent malnutrition and weight loss. The anabolic steroid, oxandrolone, has been found to be effective on pilot studies of wound treatment. A significant majority of individuals exhibited healing of pressure ulcers after treatment for up to 6 months [21, 22]. Demling and De Santi found that optimizing nutrition alone was ineffective. However, when this was supplemented with oxandrolone therapy there was an increase in weight gain by around 1.8 kg/week (4 lb/week), which was significantly correlated with wound closure. Weight gain due to oxandrolone is primarily lean body mass, i.e. muscle tissue. No side effects have been noted with oxandrolone and this approach may therefore be applicable for pressure ulcer prevention in malnourished individuals at high risk for pressure ulcer development. Further study is necessary to determine the safety and efficacy of oxandrolone for long-term therapy.

In addition to altering the intrinsic susceptibility of individuals with SCI the incidence of pressure ulcers may be decreased by more effective delivery of care. Current clinical management is predominantly based on the ethos that pressure ulcers are avoidable given adequate preventative care, and the fact that they continue to occur at high rates is seen to imply that

the patient is to some degree negligent. However, the validity of the underlying assumptions of this care model warrants further investigation.

Adequate preventative care implies that individuals at risk of pressure ulcer development are receiving both appropriate education and appropriate equipment. A recent survey of the prevalence of pressure ulcers in 5,000 hospitalized patients throughout Europe, carried out by the EPUAP, indicates that the first criterion is frequently not met [23]. The survey included all hospital inpatients, and it could be argued that individuals with SCI are not typical and will receive adequate prophylactic care. On the other hand, it can be seen from the studies presented in this paper that clinical expertise and standard treatment guidelines are not in themselves sufficient. They should be considered the starting point for effective prevention of pressure ulcers, rather than the end point.

The majority of specialized SCI rehabilitation units will provide educational programs for in-patients with acute SCI or when people are re-admitted for continuing care. However, the fact that an individual requires inpatient hospital care for treatment of a complication such as a pressure ulcer generally implies a failure in the educational process, since the problem has not been managed at an early stage. The incongruity is illustrated by the findings of a recent survey by Walter et al. [24], who found that 38% of participants reported having current problems with a pressure ulcer but only 21% of these individuals wanted to discuss their problem with a therapist. A satisfaction rate of around 80% among this group would appear somewhat unexpected.

Various approaches to improving educational awareness have been proposed. Some are based on refining the existing models of care through the development of more accurate scales for predicting pressure ulcers [25]. Others seek to modify the model through the incorporation of new technology and increased patient involvement with their care.

Initial studies have shown that the use of telehealth interventions may improve the tracking and management of pressure ulcers [26]. It was found that video monitoring combined with access to a telephone helpline will increase the number of reported pressure ulcers. However, the majority of this increased incidence is due to the reporting of Grade I and II pressure ulcers that are rarely reported in standard care models. Thus, the telehealth intervention produced an increased rate of health care utilization but this was generally to deal with less severe complications and could therefore be more cost effective. It was also found that people who employed telehealth were more likely to return to work. This has led to the hypothesis that telehealth may promote self-efficacy among users with SCI. Future work is required to investigate the role of telehealth on both physiological and psychological variables affecting the risk of SCI individuals for pressure ulcer development.

Changing the medical model to include the patient in his or her own care is another component in the future development of approaches to decreasing susceptibility to pressure ulcer development. Contingency management is a behavioural methodology that has been widely used in the treatment of substance abuse. The general protocol is reinforcement of positive patterns of be-

haviour through a reward system, such as financial compensation or vouchers, with the overall objective of the individual internalizing the behaviour patterns so that they no longer need the rewards in order to carry them out. Contingency management procedures have been designed for patients with high rates of non-compliance in skin care [27]; however, there is some controversy over the use of monetary rewards [28]. Further work is necessary to determine the long-term efficacy of this approach to behavioural modification.

Even as the incidence of and prognosis for SCI changes with improved prevention and the possibility for a cure, so the susceptibility to pressure ulcer development in the current SCI population is changing with new developments in many fields. Behavioural techniques and technological developments can alter the risk status of individuals by changing environmental factors. Implanted technologies and pharmacological agents have the potential to alter clinical risk factors, in particular by reversal of disuse muscle atrophy. The development of these multi-factorial preventative approaches expands the possibilities for reducing the future incidence of pressure ulcers in the spinal cord-injured population.

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